Subarachnoid hemorrhage (SAH) is defined as the presence of blood within the subarachnoid space due to a pathologic process, typically **ruptured aneurysm or arteriovenous malformation**. While the incidence is 9 per 100,000, it is one of the life-threatening causes of headache and should be on the differential in any undifferentiated patient. A number of medical therapies have been studied in the treatment of SAH including steroids, antiepileptic medications, antifibrinolytics, and calcium channel blockers. While current studies have not shown significant benefit nor harm with the use of antiepileptics, steroids or antifibrinolytics, there is a statistically significant benefit in the use of calcium channel blockers. This discussion will focus on the effectiveness of **calcium channel blockers in the setting of SAH**.

Patients with SAH can present with the classic "thunderclap headache" or sudden onset of the worst headache ever. They can also present with low-grade fever, nausea/vomiting, neck pain, meningismus, seizure, focal neurologic deficit, syncope, stroke-like symptoms, confusion, delirium, or cognitive impairment. I encountered a patient during my Neurocritical Care rotation who presented with fever and low back pain concerning for renal colic or pyelonephritis. She did not have an initial complaint of headache or neurologic deficit and only developed changes in mental status on hospital day three at which point a CT head was done confirming the presence of a SAH. What makes the diagnosis tricky is that patients can have a small bleed that presents as a headache that is maximal but then subsides referred to as a **sentinel bleed**. These patients can then return to the emergency department with similar or worse symptoms because they are at risk for re-bleed and cerebral vasospasm. These patients benefit greatly from early diagnosis and management of the underlying pathology resulting in the bleed.

Cerebral vasospasm occurs a **few days after the bleed, typically peaks in day four to ten, and lasts up to two weeks post-bleed**. Cerebral vasospasm can cause significant morbidity to the patient due to delayed cerebral ischemia which can manifest with a myriad of stroke-like symptoms. In a Cochrane review of 16 trials involving 3,361 patients, authors concluded that there were improved outcomes after subarachnoid hemorrhage in patients treated with calcium channel blockers. Calcium channel blockers overall had a number needed to treat of 19 and relative risk of 0.81 while **oral nimodipine** alone had a relative risk of 0.67. All other calcium channel blockers included in the study had results that were not statistically significant. There was no benefit noted in giving intravenous calcium channel blockers and in fact the hypotension that occurs with IV administration was associated with increased harm. **It is interesting to note that there was some heterogeneity in the studies and when a single large trial was excluded, the benefit of calcium channel blockers was not statistically significant**. However, given the potential benefits, the authors concluded that oral nimodipine 60 mg every four hours for 3 weeks post-bleed is indicated in the setting of SAH.

From what I learned during my Neurocritical Care rotation and review of the literature, it is important to consider SAH on the differential in the patient presenting with a **wide variety of neurologic and psychiatric complaints**. As an ED provider, our role is to accurately diagnose and admit the patient to a neurosurgical service for further evaluation of potential aneurysms or

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other pathology causing the bleed that may require surgical intervention. Management in the ED should include symptomatic support and identification of the potential complications of SAH including re-bleed, delayed ischemia, and/or hydrocephalus.

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