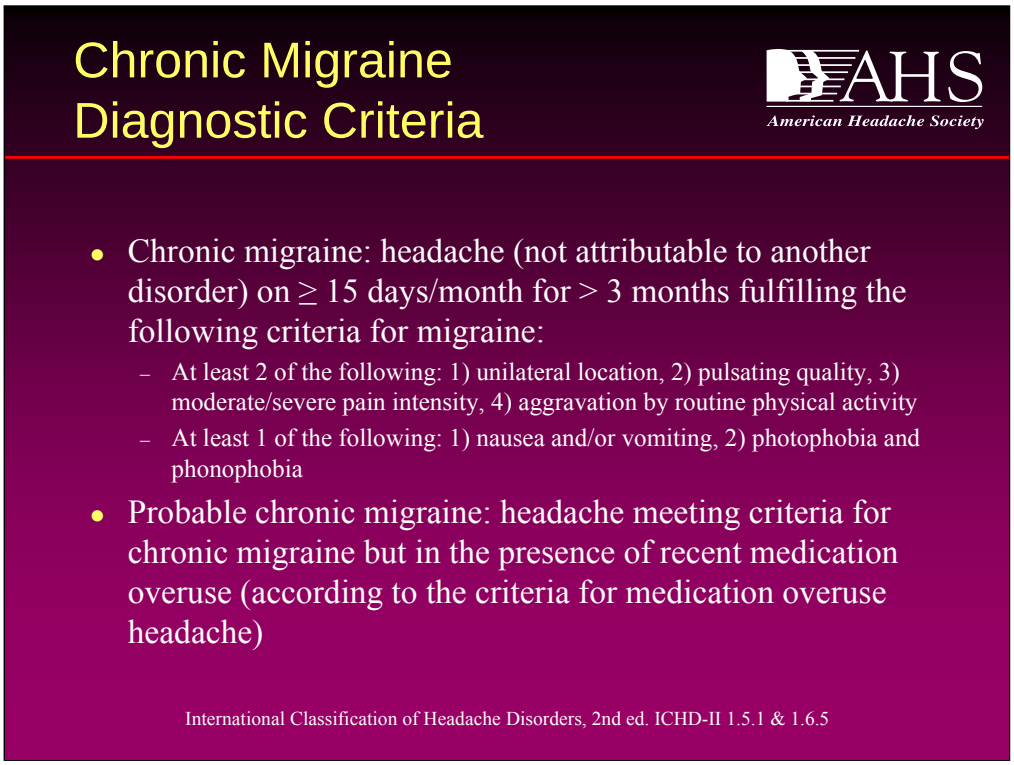
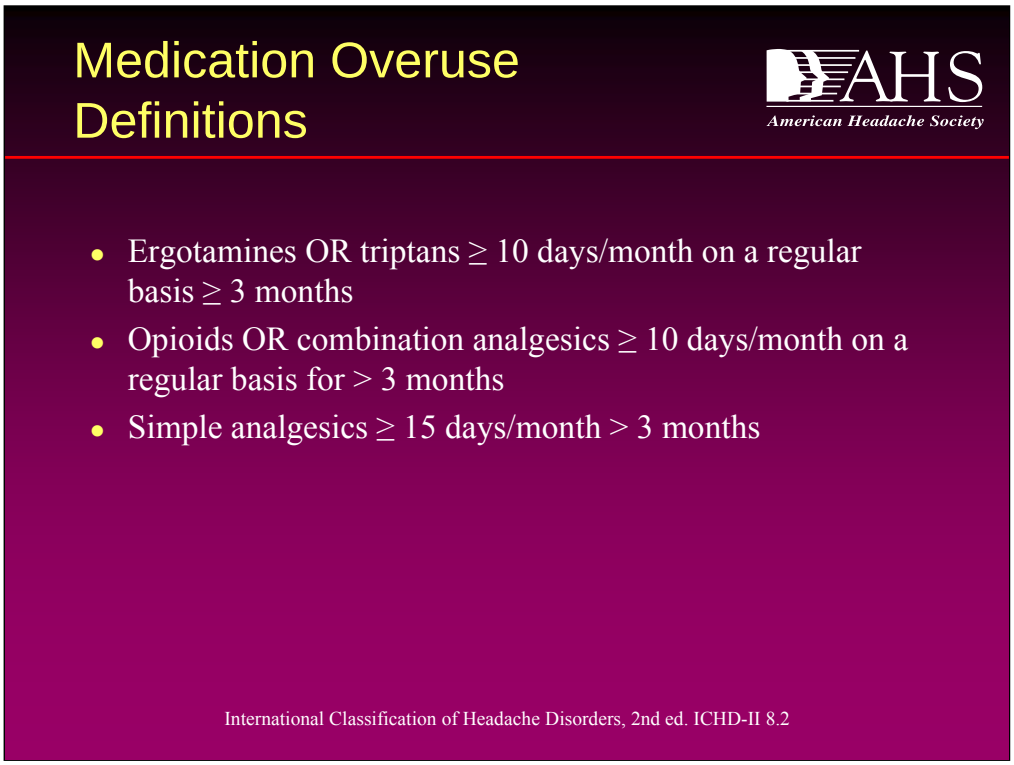
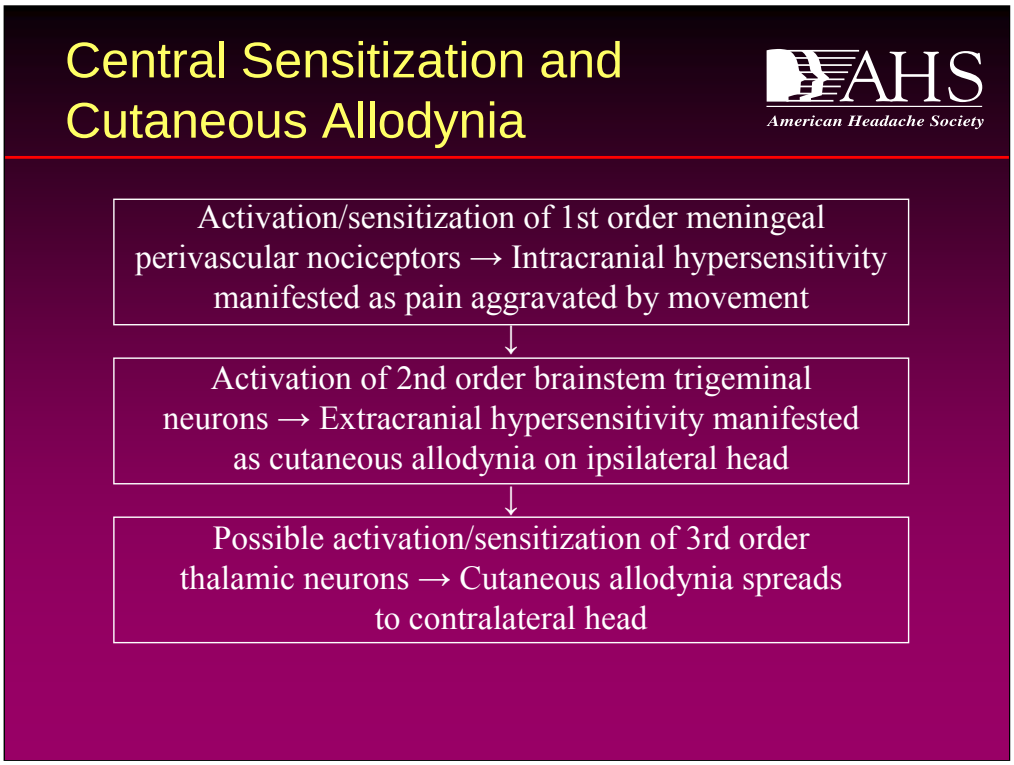


Most cases of chronic migraine evolve over time from migraine without aura. As chronicity develops, the headaches tend to lose their episodic character. There may be a pattern of mixed headache: occasional severe migraines and a more frequent headache that may lack the typical migrainous features such as throbbing pain, nausea/vomiting or light/sound sensitivity.

Since overuse of symptomatic medications has been implicated inthe evolution to near-daily headaches, if medication overuse is present, the headaches should be categorized as “probable chronic migraine,” and the suspected medication should be withdrawn. If near-daily headaches persist 2 months after cessation of the overused medication, then the headaches are redefined as “chronic migraine.” The distinction exists primarily for research purpose. Nonetheless, it serves to remind clinicians to monitor medication use of chronic headache patients very carefully.



While a cause-and-effect has not been firmly established, overuse of symptomatic migraine drugs, opioid or butalbital compounds, or analgesics is implicated in the development of chronic daily headaches with either a migraine-like or a mixed migraine-like and tension-type-like presentation. While overuse is defined in terms of treatment days (not doses) per month, the stipulation “on a regular basis” is significant—i.e., 2-3 days per week on an ongoing basis. Taking symptomatic medications on several successive days with long periods without medication use does not seem to be associated with medication overuse headache. This strategy, in fact, is often used to prevent severe menstrual migraine attacks.



During the process of central sensitization, first-order peripheral nociceptors sensitize second-order nociceptive dorsal horn neurons that, in turn, appear to sensitize third-order thalamic neurons. Burstein and colleagues have reported that 79% of patients studied experience increased skin sensitivity within the referred pain area on the ipsilateral head, which is a sign of sensitization of second order neurons within the brainstem. In two-thirds (67%) of patients, increased skin sensitivity later extended to the other side of the head and/or the forearms. They hypothesized that this hypersensitivity outside the distribution of the ipsilateral trigeminal nerve (i.e., the forearm) might result from sensitization of thalamic neurons that receive convergent input from the trigeminal nerve and nociceptors of spinal dorsal horn. Brain 2000;123: 1703-1709.