Medication adaptation headache

Miriam Solomon, Stephanie J Nahas, Judy Z Segal and William B Young

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Editorial

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Miriam Solomon[1], Stephanie J Nahas[2], Judy Z Segal[3] and William B Young[2]

The purpose of this editorial is to challenge the choice of the term 'medication overuse headache' (MOH). MOH is not a new concept, but the name remains controversial. Although it is an improvement on previous labels such as 'drug abuse headache' and 'rebound headache', there is still more work to be done. Our criticisms of the portrayal of MOH are scientific and, broadly speaking, moral. We survey possible terms and their implications and make a recommendation.

A recent review article states that MOH is 'an avoidable disorder' (1). The current diagnostic criteria (International Classification of Headache Disorders, second edition, 2004 [ICHD-2]) are:

Medication-overuse headache (8.2)

A. Headache present on ≥15 days/month fulfilling criteria C and D.
B. Regular overuse for ≥3 months of one or more drugs that can be taken for acute and/or symptomatic treatment of headache.
C. Headache has developed or markedly worsened during medication overuse.
D. Headache resolves or reverts to its previous pattern within 2 months after discontinuation of overused medication.

It follows from this definition that MOH is avoidable by preventing overuse of acute medications for headache. What counts as 'overuse', for acute prescribed medications, is intake on ≥10 days per month for ≥3 months (for simple over-the-counter analgesics 'overuse' is defined as ≥15 days per month for ≥3 months).

MOH thus results from something that patients do, that they should not be doing, namely taking the drug more often than recommended. MOH develops when the patient does not comply with the conscientious physician's instructions (some physicians may of course fail to give patients proper instructions and, if so, the physician is not being conscientious). MOH used to be called 'medication abuse headache,' a term that brings to mind the idea that those who abuse medication (a medical violation) are akin to those who abuse recreational drugs (a social violation). It is good that this term, which unnecessarily impugns patients, is no longer in the headache vocabulary. However, MOH still blames the patient, suggesting that the secondary headache could have been avoided by following physician instructions about limiting the frequency of doses of acute medication. It suggests that MOH results from a lack of patient compliance. In our view, this is an incorrect moral judgment that does an injustice to the patient while distracting us from one of the important questions: how is this kind of headache caused?

Note that MOH is defined solely by diagnostic criteria (rather than, say, by an underlying mechanism). These diagnostic criteria are rarely, if ever, *known* to be present. Criterion D is not even present at the time that the patient presents for diagnosis. Technically, in the absence of criterion D, the physician diagnoses 'probable MOH'.

We want to suggest looking at criterion B in a similar way. Patients do not, often cannot, give reliable histories of their use of acute medication. The recommendations '≥10 days per month for ≥3 months' for prescription medications and '≥15 days per month for ≥3 months' for simple analgesics come from, at best, retrospective studies identifying 'risk factors' for the development of MOH. The only prospective study, published after the ICHD-2 criteria, groups abortive medications crudely, and does not use a no-abortive-treatment group as the comparator (2). Current recommendations do not come from the highest quality of evidence, and the basis for future recommendations remains scant. Moreover, 'risk factors' are not

[1]Temple University, USA.
[2]Thomas Jefferson University, USA.
[3]University of British Columbia, Canada.

Corresponding author:
Miriam Solomon, PhD, Department of Philosophy, Anderson Hall 750, Temple University 022-32, Philadelphia PA 19122, USA
Email: msolomon@temple.edu

Table 1. Options for nomenclature

Blame the patient	Blame the doctor	Mechanism-based
Medication abuse headache	Medication overtreatment headache	Medication induced headache (maybe with type 1, type 2, *etc.*)
Drug abuse headache	Iatrogenic headache	Feed-forward headache
Medication misuse headache		Drug-transformed (or amplified) headache
Medication overuse headache		Medication adaptation headache

necessary or sufficient conditions for the development of MOH; some frequent medication users will not develop MOH and some infrequent users will. A Clinical Therapeutics article in the July 1 issue of *The New England Journal of Medicine* acknowledges that 'good evidence is lacking with regard to individual susceptibility of medication thresholds for the development of medication-overuse headache' (3). Criterion B is a guide for prescribing physicians that represents a trade-off between avoiding MOH and treating acute headache (it does not represent the lowest frequency of use of acute medication that will produce MOH in the most susceptible individuals).

Is MOH 'an avoidable disorder', as Evers and Marziniak (1) claim? The ICHD-2 definition acknowledges that MOH does not happen with every patient who exceeds the guidelines, but only with 'susceptible' patients. It is likely, we think, that there is individual variability in the frequency of usage that results in MOH. Some individuals probably develop MOH after only 2 months of use of acute medication for $\geq$10 days per month. Others probably develop MOH after 3 months of use of acute medication for $\geq$8 days per month. If these especially susceptible individuals follow their physician's orders about how often they may take medication, is it really their fault if they develop MOH? Have these patients 'overused' medication? Is MOH realistically preventable in such cases (without depriving a large number of non-susceptible people who will benefit from the medication)?

The reality is that we do not know in advance what it will take for a particular individual to develop MOH. Physicians can still caution patients about 'overuse', but this message is conflicting for a patient who is also told to treat migraine early and aggressively.

Is MOH the result of improper prescribing or poor physician supervision? Blaming the doctor is no more (or less) justified than blaming the patient. Sometimes, physicians prescribe improperly and communicate poorly with their patients. But even with the best prescribing and communication practices, MOH can develop in vulnerable patients.

Recent data reveal that rats given repeated or continuous administration of triptans develop reversible tactile allodynia, increased CGRP labelling in trigeminal neurons long after triptan discontinuation, and increased CGRP in blood after nitroglycerin challenge (4). This supports the notion that the basic culprit in MOH could actually be the interaction between regular use of a medication and a person's individual neurochemistry. It is difficult to acknowledge this, since in medicine we are most comfortable with blaming the pathology (and, when that fails, with blaming the patient). Acute medications for migraine have relieved much suffering, although they may or may not have reduced the amount of overall suffering, since MOH is a common complication of treatment. We recommend a change in terminology that shifts the responsibility from the patient to the mechanisms involved, for example, 'medication adaptation headache' (MAH). Patients can still be advised that the best way to avoid developing MAH is to stay within stated guidelines for frequency of use of acute medication, and cautioned that MAH can still develop in susceptible individuals who stay within stated guidelines.

MAH is a moderately precise term. It distinguishes between headaches that are immediate medication side effects (such as nitroglycerin headaches) and headaches that take a while to evolve (these can be medication withdrawal headaches or MAH, and the two may turn out to have similar mechanisms). It is hoped that we can be more precise when we discover more about the mechanism of MAH, which may be a drug withdrawal, feed-forward, or drug adaptation syndrome.

We present in Table 1 a list of names that we considered. We recommend avoiding the assumption of blame for either patient or physician, and using a term instead that refers to the biochemical pathway(s) involved. As we learn more, such a term will become more precise, but at the present time we recommend the placeholder, 'Medication Adaptation Headache'.

References

1. Evers S and Marziniak M. Clinical features, pathophysiology, and treatment of medication-overuse headache. *Lancet Neurol* 2010; 9: 391–401.
2. Bigal ME, Serrano D, Buse D, Scher A, Stewart WF and Lipton RB. Acute migraine medications and evolution

from episodic to chronic migraine: A longitudinal population-based study. *Headache* 2008; 48: 1157–1168.
3. Loder E. Triptan therapy in migraine. *N Engl J Med* 2010; 363: 63–70.
4. De Felice M, Ossipov MH, Wang R, Lai J, Chichorro J, Meng I, et al. Triptan-induced latent sensitization a possible basis for medication overuse headache. *Ann Neurol* 2010; 67: 325–337.