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EXCESSIVE ACUTE MIGRAINE MEDICATION USE AND MIGRAINE PROGRESSION

To the Editor: We read with interest the article by Bigal et al.[1] commenting on migraine progression due to excessive use of acute migraine medication.

Transformed migraine is common, invariably disabling, and frequently hard to treat. The authors mention that among acute migraine treatments, opiates and barbiturates are associated with progression of migraine to chronic migraine. The critical dose of exposure for opiates is only 8 days per month and 5 days per month for barbiturates, which demonstrates that these medications are best avoided in the acute treatment of migraine.

Bigal et al. found nonsteroidal anti-inflammatory drugs (NSAIDs) protective in those with <10 days of headache. Our experience with migraine patients has been that they frequently underestimate and understate their frequency and duration of NSAID use. NSAIDs are widely available without prescription. They are the most frequently and widely used medications by patients and doctors for acute migraine treatment. In our experience, transformation of episodic migraine to chronic migraine frequently occurs in the setting of excessive NSAID use.

We would stress that migraine pathophysiology is complex and further investigation is needed. In addition, haphazard use of ibuprofen for headache may cause more harm than good.

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Editor's Note: The authors of the article were offered the opportunity to respond but declined.

1. Bigal ME, Lipton RB. Excessive acute migraine medication use and migraine progression. Neurology 2008;71:1821–1828.

CORRECTION

Fatal congenital myopathy and gastrointestinal pseudo-obstruction due to *POLG1* mutations

In the article "Fatal congenital myopathy and gastrointestinal pseudo-obstruction due to *POLG1* mutations" by C. Giordano et al. (*Neurology*® 2009;72:1103–1105), there is an error in the capitalization of an author's name. The author should have been listed as M. De Curtis. The authors regret the error.

Fatal congenital myopathy and gastrointestinal pseudo-obstruction due to *POLG1* mutations

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