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# Comment on the Editorial by Goadsby and Akerman 'The trigeminovascular system does not require a peripheral sensory input to be activated—Migraine is a central disorder', in *Cephalalgia* January 2012

The authors present the results of the elegant experiments of Lambert et al. as evidence supporting their claim that the trigeminovascular system does not require a peripheral sensory input to be activated (1).

Goadsby and Akerman make a reasonable case that it is unlikely that cortical spreading depression (CSD) initiates migraine. This is not, however, the crux of the matter. What Lambert et al. in fact showed is that even if CSD does not initiate migraine attacks, it most certainly does lead to increased discharge rates in trigeminal second order neurons (2). Lambert et al. state, 'At a variable time after the successful initiation of CSD, the basal discharge rate of responsive trigeminovascular neurons began to increase and continued to increase for upwards of 20 minutes in some animals'. The increased discharge rates following CSD initiation were unaffected by injection of lidocaine into the trigeminal ganglion, whether it was administered before or after the initiation of CSD.

What was effectively demonstrated is that (a) CSD is indeed followed by increased discharge rates in trigeminal second order neurons and (b) that this increased discharge does not involve activation of first order trigeminovascular nociceptors that innervate the dura and meningeal blood vessels, but that there may instead be subcortical mechanisms involved.

It is certainly not proof that 'trigeminovascular activation can be generated within the brain without a peripheral sensory input'. The evidence from Lambert's study can surely be interpreted only to mean the exact opposite. The discharge rate of trigeminal neurons only increased *after* the initiation of CSD. That Goadsby and Akerman have misinterpreted Lambert et al.'s results is evident from their claim that Lambert et al. demonstrated that lignocaine does not prevent 'CSD-induced spontaneous discharge'. It is either CSD-induced or it is spontaneous—it cannot be both. In this case, Lambert et al. showed that it was indeed CSD-induced. In order for Goadsby and Akerman to prove that migraine is a central disorder, they will have to show that the discharge rate of trigeminal neurons increases spontaneously without the prior initiation of CSD, or indeed of any other peripheral changes.

## References

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2. Lambert GA, Truong L and Zagami AS. Effect of cortical spreading depression on basal and evoked traffic in the trigeminovascular sensory system. *Cephalalgia* 2011; 31: 1439–1451.

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