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Comment on "Pathophysiology of migraine" by professor PJ Goadsby in August 2012 edition of Ann Indian Acad Neurol

Sir,

In his article "Pathophysiology of migraine," in the Ann Indian Acad Neurol 2012 August; 15(Suppl 1):S15-S22,[1] Following points need serious consideration:

1. The author stated "Vascular changes are unrelated to the phase of the attack; indeed blood flow could be reduced or normal during the pain phase." To support this statement, he cited the research carried out by Olesen and the "Copenhagen Group." It is true that the Copenhagen Group found that changes in intracranial regional cerebral blood-flow (rCBF) were unrelated to the severity and timing of migraine pain[2] but this study of the Copenhagen Group had no relevance to extracranial vasodilatation. Olesen's group studied rCBF, not the blood-flow of the extracranial arteries. The use of the Copenhagen Group's observations in this context is misleading.
2. The author has previously claimed, and has now repeated, that migraine occurs without any change in the extracranial vessels. To support this claim, he referred to the work of Schoonman *et al*., who were able for the first time to accurately measure the diameters of most of the major intracranial and some of the extracranial vessels, both during migraine and interictally, and on left and right sides during unilateral migraine. They showed that none of the vessels measured dilated during migraine.[3] The only part of the extracranial vasculature that Schoonman *et al*., measured, however, was the last 1 cm of the main trunk of the external carotid artery, which has never been implicated in migraine. Schoonman *et al*., made no mention of the terminal branches of the external carotid artery as these vessels were not included in their study. The use of Schoonman's study to justify the claim that the terminal branches of the extracranial vessels are not involved in migraine is to misrepresent the results of Schoonman's study.
3. The third serious misrepresentation concerns the author's claim, that the gepant BIBN4096BS is "without vascular effects." Yes, BIBN4096BS does not actively constrict arteries in healthy volunteers,[4] but that is completely different from being without vascular effects in the context of migraine. To substantiate this claim, the author cited Petersen *et al*.,[4] Petersen's article is unambiguous – it clearly states that BIBN4096BS is

"very effective in preventing calcitonin gene-related peptide (CGRP)-induced vasodilatation." BIBN4096BS was in fact developed specifically to reverse the vasodilatation caused by the increased CGRP levels during migraine.[5] To cite Petersen's study to justify the claim that BIBN4096BS is without vascular effects is a contradiction.

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