

Maria Rosaria Maluccio, MD
 Gianfranco Cafforio, MD
 Alberto Chiti, MD
 Giovanni Orlandi, MD, PhD
 Simone Gallerini, MD
 Luigi Murri, MD, Prof.

University Centre for Adaptive Disorders and Headache
 – Department of Neurosciences, University of Pisa, Italy

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Headache Education for the Medical Students: Wolff's Postulates

The initiative of the article entitled “Square One: Headache Education for the Medical Student,” in the March 2007 issue of *Headache* to introduce and disseminate a list of core competencies for medical students, is long overdue, and is to be applauded. There is, however, one astounding suggestion that flies in the face of established fact – that is, that Wolff's vascular theory of migraine should *not* be taught to medical students. The authors state that the (Education) Committee is of the opinion that Wolff's theory is “invalid and impedes more up-to-date understanding of the pathophysiology of migraine.” With respect, the Committee is totally misguided – opinions which are not supported by the facts have no place in science! The available hard scientific evidence has repeatedly vindicated Wolff's stance.

It is important to understand exactly what Wolff was saying. He wrote “The pain of vascular headache results from increased tension within or about pain sensitive cranial artery walls.”¹ He neither stated nor implied that the pathophysiological process in migraine originated from the extracranial arteries – what he showed was that the *pain* of migraine originated from the extracranial arteries. As was clearly stated in one of his articles, “these results have no bearing on preheadache phenomena. They concern only the origin of migraine pain.”² These findings have never been disproven. Blau tried to, but his figures actually confirmed that the extracranial arteries are in fact involved (in 43% of cases in his flawed study).³ On the contrary, Wolff's findings have been confirmed repeatedly.⁴⁻¹⁰

Current understanding of migraine pathophysiology implicates changes in the trigeminovascular system, which by definition is composed of the trigeminal subnucleus caudalis, the trigeminal nerve, and the intracranial arteries.¹¹ This definition has become almost universally accepted, in spite of the complete absence of hard scientific evidence that the intracranial arteries are the source of migraine pain. Indeed, it is this definition, from which the extracranial arteries are arbitrarily excluded, and not Wolff's theory, which impedes “more up-to-date understanding of migraine!” Redefinition of the trigeminovascular system to include the extracranial branches of the external carotid artery would comprise a real advance in the understanding of migraine.

Elliot Shevel, BDS, DipMFOS, MB, BCh
 The Headache Clinic—Research and Development,
 Houghton, South Africa

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Headache Education for the Medical Students – Wolff's Postulates: A Response

As the authors of the article, "Square One," we did not intend to attack Dr. Harold Wolff's reputation, rather to bring our understanding of migraine up to date, partially based on Dr. Olesen's work on regional cerebral blood flow changes during aura.¹ Wolff stated in his textbook that "the preheadache phenomena of scotomas results from cranial vasoconstriction,"² which is refuted by Dr. Olesen's and many subsequent studies. Wolff's theory states that the pain of migraine results from vasodilatation.² The timing of migraine pain is not linked to vasodilation.¹ The fact that vasodilatation in the absence of neurogenic inflammation is not painful makes it clear that vasodilatation is not sufficient, and may not be necessary, for migraine pain. Incidentally, what is the cause of nonthrobbing migraine pain?

Relying on vasodilatation to explain migraine pain ignores the pain of one-third of migraine sufferers.

That pain relief is obtained when an extracranial vessel is compressed is not evidence that that vessel is the source of pain. Other mechanisms may account for this. Diffuse noxious inhibitory control and other neuromodulatory reflexes are alternative explanations for this observation.

The core curriculum for medical students was written by the authors, but was reviewed, edited, and accepted by the Education Committee and the Executive Committee of the American Headache Society. We all felt that the Wolff theory – that aura is caused by vasoconstriction and pain is caused by vasodilatation – without reference to spreading depression and oligemia, neurogenic inflammation, is untrue, and leads to bad medicine.

Harold Wolff was a great scientist and clinician. He brought scientific inquiry to headache medicine. We certainly did not intend to besmirch his reputation.

William B. Young, MD

Thomas Jefferson University Hospital – Neurology,
Philadelphia, PA, USA

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Concomitant Triptan and SSRI or SNRI Use: What is the Risk for Serotonin Syndrome?

Sclar et al estimate that, during 2003-2004, an annualized mean of 694,276 patients were simultaneously prescribed or continued use of a triptan along with a selective serotonin reuptake inhibitor (SSRI) or a selective serotonin/norepinephrine reuptake inhibitor (SNRI).¹ Several times in the article they reiterate the US Food and Drug Administration (FDA) alert of 2006 and caution that the combination of medications is potentially fatal. They conclude, "Based on the available empirical evidence, we suggest that physicians avoid prescribing this combination of medications if possible, and closely monitor patients who must utilize this combination for signs and symptoms of serotonin syndrome." However, their estimate of the widespread coprescription does not add to the empirical