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## TEACHING NEUROIMAGES: MICROVASCULAR DECOMPRESSION OF THE OPTIC NERVE

Mandeep S. Ghuman, Chandigarh, India: We were interested to read the Neuro*Image* by Woodall et al.<sup>1</sup> In addition to ectatic internal cerebral artery abutting the optic nerve, we also noted the tortuous course of intra-orbital optic nerves with dilated perineural subarachnoid space, features consistent with idiopathic intracranial hypertension (IIH). IIH also results in vision diminution and even though the cause-and-effect relationship of vascular loop compression is unclear, it is important to rule out other causes and provide conservative management before resorting to surgery.

Compression over cranial nerves by redundant vessels and the correlation between compression and neurologic symptoms have not been shown. Vascular loop compression of the 5th cranial nerve resulting in trigeminal neuralgia was first suggested by Dandy<sup>2</sup> in 1934. The same concept was expanded to explain various cranial nerve disorders including hemifacial spasms, glossopharyngeal neuralgia, and geniculate neuralgia. Janetta<sup>3</sup> was the first to perform microvascular decompression and proposed that the redundant arterial loops compress the 8th cranial nerve at the cerebellopontine angle leading to symptoms of vertigo, tinnitus, and auditory loss.

The pathophysiology of vascular compression syndromes is controversial. A redundant vascular loop abutting, indenting, or even compressing any nerve is frequently seen on imaging in asymptomatic subjects, and demonstration of vascular compression should not be the sole diagnostic criterion when considering surgical decompression. 4–6

Author Response: M. Neil Woodall, Cargill H. Alleyne, Jr., Augusta, GA: We appreciate Dr. Ghuman's comments regarding our article. Dr. Ghuman correctly argued that vascular compression of the optic nerve is not the sole diagnostic

consideration in the workup of monocular visual loss. Based on MRI findings, he argued that our patient should have been diagnosed with, and treated for, IIH rather than taken to surgery. Our patient presented with painless, progressive, monocular visual loss. He specifically denied any history of headaches and had no other signs or symptoms of increased intracranial pressure. On formal funduscopic examination, no papilledema was appreciated to support a diagnosis of IIH.7 The diagnosis of IIH is clinical.8 While neuroimaging features such as posterior globe flattening, optic nerve sheath distension, nerve tortuosity, and an empty sella are associated with IIH, only posterior globe flattening reliably predicts a diagnosis of IIH.9 Our diagnosis is further supported by the patient's improvement in vision following microvascular decompression. While vascular compression of the optic nerve is not the only diagnostic consideration in cases of visual loss, it is vital when another explanation is not apparent, not only for our patient but for other reported patients. 10

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## Teaching Neuro Images: Microvascular decompression of the optic nerve

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