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Brief Communication

Middle Meningeal Artery Dilatation in Migraine

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Objective.—To show that migraine pain is not related to dilatation of the dural meningeal arteries.

Background.—The origin of the pain in migraine has not yet been adequately explained and remains the subject of vigorous debate. Current theories implicate changes in the trigeminovascular system, which is defined as comprising the large intracranial vessels, and in particular, the dural meningeal vessels, the dura mater, and their neural connections.

Methods.—The anatomical relationships of the dural meningeal arteries to the dura mater and the inner surface of the calvarium are described.

Results.—The dural meningeal arteries lie in grooves in the inner table of the calvarium, are encased in the unyielding fibrous dura mater, and are consequently unable to dilate.

Conclusion.—The pain of migraine is not related to dilatation of the dural meningeal arteries.

Key words: meningeal arteries, migraine, vasodilatation

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The trigeminovascular system is composed of the large intracranial vessels, the dura mater, and their neural connections.[1,2] It is currently considered that the dural blood vessels and their neural connectivity to the trigeminal subnucleus caudalis, as part of the trigeminovascular system, play a fundamental role in the perception of migraine pain,[3,4] and that the key pathway for the pain is trigeminal input from the meningeal vessels.[5] While there is still debate over the initiating events in migraine, it is widely believed that "headache pain arises when the trigeminovascular system becomes activated causing vasodilatation. . . in pain producing meningeal tissues."[6] In addition, the triptan antimigraine agents selectively target, among other sites, serotonin 5-HT_{1B} receptors,[7,8] which are found on the smooth muscle of meningeal blood vessels,[9-13] and administration of calcitonin gene-related peptide (CGRP) causes a significant increase in dural blood flow in rats.[14]

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MATERIALS

Specimens were taken from the cranial vault of a cadaver being dissected for teaching purposes by medical students at the Anatomy Department at the University of the Witwatersrand Medical School. The tissue was donated in accordance with the South African Human Tissues Act. The cadaver had undergone an embalming process using the following embalming fluid perfused via the brachial artery: Methylated spirits 85%, Glycerine 9%, Formalin 4.5%, Phenol 1.1%, Thymol 0.1%. This embalming process, used by the Anatomy Department at the Witwatersrand University Medical School, does not cause swelling or distortion of the tissues.

Conflict of Interest: None

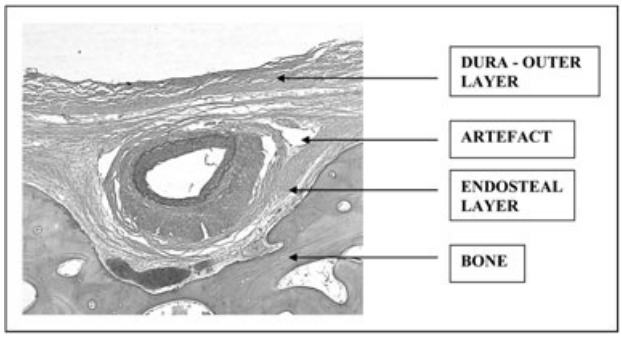

Figure.—Photomicrograph of a section of the human middle meningeal artery, encased in bone and dura mater. The dura mater is firmly adherent to the inner table of the skull.

ANATOMICAL RELATIONSHIPS OF THE DURAL ARTERIES

The cerebral dura mater (Latin for "hard mother") is a very tough membrane composed of 2 closely adherent layers of predominantly collagenous connective tissue. These layers tend to be at right angles to one another, so that the dura is rigid and resistant to stretching. The dura mater is closely applied to the interior of the skull without any intervening fat, and is fused with the periosteum.[15] An indication of the inelasticity of the dura mater is that the strain-independent elastic fractions for tendon, dura mater, and pericardium are similar.[16] The principal blood supply to the dura is via the middle meningeal artery, which lies mainly in the outer or endosteal layer of the dura.[17] As shown in the photomicrograph, in humans the artery is firmly encased between the layers of the unyielding dura mater (Fig.). Photomicrographs from different locations showed that this relationship is constant. With regard to the bony relationships of the dural meningeal arteries, in humans they run in grooves in the inner table of the calvarium, so that they are surrounded almost on 3 sides by bone, to which the dura is firmly adherent (Fig.).

DISCUSSION

The notion that dural vasodilatation plays an important role in the pathophysiology of migraine is based on animal experimental evidence only[18] – the administration of subtance P, neurokinin A, and CGRP causes dural vasodilatation in experimental rats[19] – but there is a *caveat*. Human anatomy is not identical to rat anatomy, and although animal experimentation often provides useful information, it cannot be assumed that data obtained from animal models accurately reflect the changes that occur in humans.[20] The dural meningeal vessels in rats may indeed be able to dilate, but in humans their dilatation is effectively prevented by their anatomical constraints. The contention that the dural meningeal vessels do not dilate during migraine is given further credence by recent research[21] that has shown conclusively that in migraine the diameter of the extracranial portion of the middle meningeal, the main trunk of the external carotid, the internal carotid, middle cerebral, basilar, and posterior cerebral arteries are no different from baseline, nor between headache and non-headache sides. Although the diameters of the intracranial dural portions of the middle meningeal arteries were not measured in this study, it would seem most improbable that the dural middle meningeal artery would react differently, particularly given its anatomic constraints as described above.

CONCLUSION

In humans the anatomical relationships of the dural meningeal arteries are such that their physical constraints within the inelastic dura mater and the bony channels in the inner table of the calvarium prevent them from dilating. The pain of migraine can therefore not be related to dilatation of the dural meningeal arteries.

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