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Letter to Editor

A Methodological Remark on In Vivo Models of Experimental Subarachnoid Hemorrhage

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İn Vivo Deneysel Subaraknoid Kanama Modelleri Hakkında Metodolojik bir Hatırlatma

Anahtar Kelimeler: Subaraknoid kanama, serebral vazospazm, arteryal kan, deneysel modeller

Dear Editor,

We have read with great interest the article 'Vagal Nerve Degeneration and Pulmonary Artery Vasospasm After Subarachnoid Hemorrhage' which was published in the last issue of this journal[1]. Despite high numbers of experimental investigations focusing on subarachnoid hemorrhage (SAH) induced cerebral vasospasm, the studies concerned with systemic effects of SAH have rarely been performed. Authors present such an experimental study with interesting and valuable findings. They showed the existence of vagal nerve degeneration and pulmonary artery spasm following experimental SAH. Vagal nerve ischemia at brainstem due to spasm of 'vagal nerve root supplying arteries' has been speculated to be the possible mechanism leading to vagal nerve degeneration and subsequent pulmonary artery spasm.

Since cerebral vasospasm have long been a popular subject of intensive research interest especially for neurosurgeons, the variety of experimental models with their own advantages and disadvantages have been performed to mimic actual SAH[3]. The experimental model (autologous blood injection to cisterna magna) used by authors in this study is one of the most commonly used technique due to its simplicity and low cost. However, a particular point should be discussed about this study. The authors have preferred to apply venous blood injection instead of routine arterial blood injection into cisterna magna. Although, many etiological factors including trauma, arteriovenous malformations, arteriovenous fistulas, cavernomas, neoplastic disorders and many others, can be reasons for development of SAH, the term 'SAH' without any additional description is specifically understood as a clinical state caused by

dissemination of arterial blood into cisterns due to a rupture of an intracranial aneurysm. Therefore, the SAH models performed by cisternal blood injection are traditionally applied by using autologous arterial blood to constitute not only the most resembling clinical state seen after aneurysm rupture but also evident vasospatic changes in cerebral arteries.

Although, cerebral vasospasm is not primary concern of this study, spasm of 'vagal nerve root supplying arteries' has been speculated to be the reason leading to vagal nerve ischemia, degeneration and pulmonary artery spasm. In this manner, we believe that using arterial blood for cisternal injection should have been preferred in this study to constitute the most similar clinical state with actual SAH. Additionally, vasospasm of 'vagal nerve root supplying arteries' which has been speculated to be the reason of pulmonary artery spasm would have appeared to be a more convincing mechanism in that way, since the superiority of arterial blood injection to venous blood injection to develop vasospasm in this experimental model has been well documented[2].

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