Quantitative magnetic resonance angiography

In the past, clinical decisions regarding treatment of neurovascular disorders leading to ischemia have been guided by the percentage of stenosis of the vessel in question. However, such an approach assumes a predictable and stable relationship between the percentage of stenosis and the degree of flow reduction it causes. Historically, this type of relationship has been difficult to document. Thus, a method for noninvasively measuring the absolute flow of specific cerebral arteries is of potential practical value.

Methods: We set to quantify the mean blood flow (Qm, in mL/min) in the cerebral arteries using quantitative magnetic resonance angiography (QMRA), and to compare the findings in normal vessels with those found in vessels considered pathologically narrowed. Specific vascular segments were identified, studied, and the results entered into a database. Statistical analyses of the measurements were carried out using StatPlus for Microsoft Excel. It involved comparing of the Qm found in specific vessels, as well as those found in normal and abnormal vessels, using analysis of variance (ANOVA). The abnormal vessels were selected from specifically identified magnetic resonance angiography (MRA) studies.

Results: A total of 57 patients, 26 men and 31 women, with ages ranging from 19 to 86 years (mean = 64.5), underwent MRA with subsequent QMRA of 157 arteries. The latter included 72 internal carotid (ICA), 45 vertebral (VA), 18 common carotid (CCA), 13 middle cerebral (MCA), and nine basilar (BA) arteries. The mean Qm obtained were CCA = 313.9 (+/-115.4), ICA = 231.1 (+/-83.7), VA = 90.5 (+/-45.8), MCA = 92.5 (+/-62.3) and BA = 120.1 (+/-64.5). ANOVA showed significant differences between individual vessels ($P < .00001$). The values obtained were consistent with those predicted mathematically, as derivatives of their proportional contributions to overall cerebral arterial flow (Qbrain) as a product of the normal cardiac output (CO) [ie, Qbrain = CO*.2]. Further ANOVA of the normal and abnormal vessels showed statistically significant differences between the two groups (186.5 +/- 108.6 vs. 117.9 +/- 76.1, respectively; $P= .000514$).

Conclusions: The use of QMRA provides the mean for direct measurement of absolute blood flow within the cerebral arteries in physiologic and pathologic states. This technique may be of future importance in characterizing absolute flow compromise in the cerebral arteries under a variety of clinically relevant circumstances [1)].

Intracranial atherosclerotic disease (ICAD) is an important cause of ischemic stroke. The etiology of stroke in patients with ICAD could be due to several mechanisms including hypoperfusion, artery-to-artery embolism, and plaque extension over small penetrating artery ostia. Management of symptomatic ICAD includes medical and endovascular management. Quantitative magnetic resonance angiography (MRA) is a technique that allows for non-invasive measurement of large vessel blood flow in the head and neck. Here, we describe procedural and clinical outcomes on three patients who presented with symptomatic ICAD and were treated with angioplasty and stenting. Quantitative MRA was used pre- and post- procedurally to assess the effects of stenting on the intracranial blood flow. Quantitative measures of intracranial blood flow may serve as an additional triage tool in the evaluation of patients with symptomatic ICAD [2)].

Quantitative magnetic resonance angiography measured large-vessel vertebrobasilar territory flow, and patients were designated as low or normal flow based on a prespecified empiric algorithm considering distal territory regional flow and collateral capacity. For the present study, post hoc analysis was performed to generate additional predictive models using age-specific normalized flow measurements. Sensitivity, specificity, and time-to-event analyses were compared between the algorithms. The original prespecified algorithm had 50% sensitivity and 79% specificity for future stroke risk prediction; using a predictive model based on age-normalized flows in the basilar and posterior cerebral arteries, standardized to vascular anatomy, optimized flow status thresholds were identified. The optimized algorithm maintained sensitivity and increased specificity to 84%, while demonstrating a larger and more significant hazard ratio for stroke on time-to-event analysis. Conclusions These results indicate that flow remains a strong predictor of stroke across different predictive models, and suggest that prediction of future stroke risk can be optimized by use of vascular anatomy and age-specific normalized flows [3].

1)

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2)

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3)

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