Transcranial Doppler Measurements of the Middle Cerebral Artery

Effect of Hematocrit

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We evaluated the effects of hematocrit on blood velocity in the middle cerebral artery measured by transcranial Doppler ultrasonography in 45 patients. Hematocrits ranged from 16% to 46%, and systolic blood velocities ranged from 70 to 190 (mean 40 to 140) cm/sec. Decreases in hematocrit correlated with increases in blood velocity in the middle cerebral artery. These results suggest that transcranial Doppler measurements made in the presence of anemia may need correction. (Stroke 1988;19:1466-1469)

In 1982, Aaslid et al1 introduced the transcranial Doppler (TCD), an ultrasonic device capable of measuring blood velocity in the basal cerebral arteries through an intact cranium. Because noninvasive TCD measurements offer the advantages of safe, easy, and repeatable studies of the intracranial circulation, many efforts are underway to determine the full potential of this new technique. The two characteristics of the velocity waveform that have found clinical use are velocity and pulsatility (usually expressed as the Gosling pulsatility index2). Decreases in pulsatility index are usually seen with damping of the velocity waveform. The damping is caused by energy lost through a high-resistance pathway due to a variety of causes, such as a highly stenotic vessel, a vessel carrying collateral blood flow (because of a longer pathway that the blood must traverse),3 or a vessel with altered compliance (e.g., arteriovenous malformation–feeding vessels).4

The major factors that determine blood velocity are the pressure gradient along a vessel, the length and cross-sectional area of the vessel, and viscosity of the blood. Increased velocity from reduced cross-sectional area occurs in vessels with narrowing due to high-grade stenosis from arteriosclerosis, with vasospasm, or with a functional narrowing (e.g., collateral blood flow with increased flow in the anterior communicating artery in unilateral carotid occlusion).3

Although a clear correlation has been found between blood velocity and cross-sectional area, little attention heretofore has been paid to the role of blood viscosity in blood velocity. Hematocrit is the major determinant of blood viscosity. Cerebral blood flow measured by other methods has been shown to be inversely proportional to hematocrit.5

Our study was undertaken to investigate the influence of hematocrit on blood velocity in the middle cerebral artery as measured by TCD.

Subjects and Methods

Forty-five patients (21 men, 24 women) were examined using TCD. Their mean age was 37 (range 5-80) years. The patients were selected from all TCD studies performed at the Non-Invasive Vascular Laboratory of the Neurological Institute of New York based on the following criteria: technically adequate TCD study, hematocrit determined as percent within 24 hours of TCD examination, no transfusion or hemodilution therapy within the past 2 months, no evidence of hemorrhage on computed tomogram, normal level of alertness, no evidence of intracranial stenosis by TCD, and no evidence of congestive heart failure or chronic obstructive pulmonary disease by history or physical examination. Stroke was the reason for TCD examination in 14 patients and transient ischemic attack the reason in...
seven (normal patients); 24 patients were studied as part of an ongoing prospective investigation into sickle cell disease.

We used Model TC2-64 (EME, Überlingen, FRG), a microcomputer-controlled directional TCD device operating at 2 mHz pulsed ultrasound, using a 64-point fast Fourier transform spectrum analyzer. The transcranial probe is 22 mm in diameter and uses a focused beam operating at depths of 25–155 mm electronically adjustable in 5-mm steps. The pulse repetition frequency was 5–10 kHz, with a sound intensity range of 10–100 mW/cm², a pulse length of 13 μsec, a gate width of 13 μsec, a vessel wall filter of 150 Hz, and a low-pass filter of 10 kHz. The output is displayed as a time vs. Doppler shift plot on a cathode ray tube. A hard copy was made on an Epson RX-80 dot matrix printer (Torrance, California) using a program written by one of us for use on the Tandy Model 100 laptop computer (Fort Worth, Texas).

All studies were performed with the patient in bed, with the head elevated no more than 30°. All patients were at rest during the study. TCD recordings were made at 45, 50, and 55 mm along the courses of both of the middle cerebral arteries. The arithmetic mean of these six readings (three from each side) were used in all calculations. At each depth, the systolic and the mean blood velocities as centimeters per second and the systolic:diastolic (S/D) ratio were recorded. From these data, the Gosling pulsatility index was calculated using the method of Gosling and King as the difference between the systolic and diastolic blood velocities divided by the mean blood velocity.

**Results**

A high correlation was found between hematocrit and systolic blood velocity (Figure 1) and mean blood velocity. There was a wide range of hematocrits (10–46%) and a correspondingly large range of systolic blood velocities (60–289 cm/sec), with the greatest effect on velocity found at lower hematocrits. Hematocrit was slightly higher in older patients (Figure 2). This finding can be accounted for, in part, by the predominance of younger patients with sickle cell anemia.

The correlation between hematocrit and blood velocity was unrelated to the diagnosis of sickle cell anemia, being found among patients with and without this condition (Figure 1). The regression curves for normal patients and those with sickle cell disease were within 1% of each other at a hematocrit of 45% and within 15% at a hematocrit of 25%. Pulsatility index, which has been correlated with resistance to blood flow, was not significantly influenced by hematocrit (Figure 3).

The relation between systolic blood velocity ($V_{systolic}$) and hematocrit ($Hct$) can be expressed as $V_{systolic}=326 e^{-0.033 Hct}$, with a Pearson correlation coefficient of 0.79 ($p<0.001$). The correlation between the systolic and mean ($V_{mean}$) blood velocities is described by the equation $V_{systolic}=1.26V_{mean}+20$ ($r=0.98$, $p<0.0005$), indicating that either velocity can be used for such measurements. The regression equation for the normal patients is $V_{systolic}=228 e^{-0.025 Hct}$.

**Discussion**

Our study demonstrates a clear correlation between hematocrit and blood velocity and indicates that hematocrit must be included in the small list of factors that can affect blood velocity during TCD examination. The effect of hematocrit on
blood velocity measured by TCD must be taken into account in determining the clinical significance of a given finding. This effect on blood velocity is most evident at lower hematocrits.

Hematocrit is the major determinant of blood viscosity. We suspect that the changes observed are directly related to changes in viscosity, but we have not ruled out other possible explanations, including lowered oxygen carrying capacity, a repartitioning of inertial and viscous resistance losses in the basal cerebral arteries, or an increase in the cardiac output, although no indications of such changes were evident among our patients.

Since abnormal elevation in blood velocity is presently the major criterion for diagnosing intracranial stenosis and for following the clinical course of vasospasm and its therapy, the range of changes that can be attributed solely to hematocrit are of obvious importance. In vasospasm or collateral blood flow, the blood velocity is usually increased along the entire length of the vessel. The effects of hematocrit on blood velocity are also seen diffusely. In focal intracranial stenosis, the change in blood velocity is likely to be limited to the 10–15 mm region near the stenosis, which should help prevent misdiagnosis based on altered hematocrit.

For vasospasm and its therapy, widespread increased blood velocities do not allow a simple means of estimating the clinical significance of the TCD measurement. Aaslid has correlated mean blood velocities of >120 cm/sec with moderate to severe spasm. Harders and Gilsbach used mean frequency shifts of 2 kHz (78 cm/sec) for subcritical and 3 kHz (117 cm/sec) for critical spasm. Our results indicate that blood velocities in this range may be due solely to the increased blood velocity seen with anemia. Several ongoing trials have been organized to investigate if early hemodilution can improve the clinical outcome from ischemic stroke. There have been several studies that, using hemodilution, have documented increased cerebral blood flow, reduced infarct size, and qualitatively improved electrocardiogram. Our findings may mean that patients being treated with hypervolemic hemodilution therapy could develop changes in TCD blood velocities that might be misinterpreted as a sign that vasospasm was increasing in response to therapy when spasm was actually unchanged or improved.

In addition to its use as a monitoring device, TCD may be helpful in addressing some of the basic questions about hemodilution, among them does increased blood flow depend on the percent reduction in hematocrit?, is there a single optimum hematocrit?, is pretreatment hematocrit an important variable?, and should the effects of hemodilution be based on some objective measurement of blood flow or simply on peripheral hematocrit? We are using current protocols from our laboratory, attempting to study the correlation between TCD and xenon determinations of regional cerebral blood flow in a variety of clinical settings to better define the role of this innovative ultrasonographic technique.

References


KEY WORDS • blood flow velocity • hematocrit • ultrasonics
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