Detection of Middle Cerebral Emboli During Coronary Artery Bypass Surgery Using Transcranial Doppler Sonography

To the Editor:

Spencer et al recently described the use of transcranial Doppler sonography to record the passage of emboli through the middle cerebral artery (MCA) during coronary endarterectomy. They recorded transient signals at the time of releasing common carotid cross clamps and inferred that these were due to microbubbles. Similar signals at other times were assumed also to be indicative of emboli, although of unproven constitution. They suggested that the usefulness of this technique might be extended to cardiac sources of embolism.

We have recently concluded such a study. Transcranial Doppler (TC2-64 EME) was used to record middle cerebral artery flow velocity before, during, and immediately after bypass during coronary artery surgery. Continuously recorded velocities from the right MCA were averaged over 30 seconds and stored on an Apple 2e microcomputer. The software also detected high-amplitude transients (more than twice the amplitude under steady-flow conditions) and recorded their number over each 30-second period. High-amplitude signals were common at the time of aortic cannulation and at the inception of bypass, but were also recorded during stable bypass. In vitro studies by ourselves and others using model extracorporeal circulations show that the signal characteristic recorded during surgery can be attributed to microemboli. This interpretation is strengthened by the results of a randomized study of the effect of an additional arterial line filter on the incidence of "emboli" and on the neuropsychological sequelae of coronary artery bypass surgery.

<table>
<thead>
<tr>
<th>Inception of bypass</th>
<th>Filtered</th>
<th>Nonfiltered</th>
<th>p*</th>
</tr>
</thead>
<tbody>
<tr>
<td>median &amp; range/2.5 mins</td>
<td>36 (5–82)</td>
<td>45 (5–100)</td>
<td>0.1</td>
</tr>
<tr>
<td>During bypass</td>
<td>6 (0–10)</td>
<td>243 (30–768)</td>
<td>0.001</td>
</tr>
</tbody>
</table>

*Values obtained by Wilcoxon test.

We randomized 40 patients to filtered and nonfiltered bypass. There was no significant difference in age, pre-bypass Paco2 or length of time on bypass. There was also no significant difference in the incidence of microemboli counted at the onset of bypass (Table 1), but a major difference during stable bypass (p<0.001). This became increasingly striking as bypass progressed. Patients in the group with an extra filter and less recordable microemboli were less likely to show postoperative deterioration in performance on a neuropsychological test battery, the difference being significant for the Rey Auditory Verbal Learning test (p<0.01, Wilcoxon test). This strongly supports the view that the consistently detectable neuropsychological deterioration in a proportion of patients after coronary artery bypass surgery is due at least in part to cerebral embolism.

We would agree with Spencer and colleagues that this ability to record the passage of emboli is very promising.

M. J. G. Harrison
W. Pugsley
S. Newman
C. Paschalis
L. Klinger
T. Treasure
B. Aspy
University College and Middlesex School of Medicine
London, UK

References

Effect of Intra-aortic Balloon Pumps on Cerebral Circulation

To the Editor:

We read with great interest the article by Brass in which he discusses the effect of intra-aortic balloon pumps upon the cerebral circulation as studied by transcranial Doppler (TCD) sonography. We were disappointed, however, that the discussion did not include some specific points that we feel need to be addressed. The first is the fact that in the spectral waveform shown in the article, the author does not comment as to the specific ratio between the rate of the spontaneous cardiac cycle and that of the intra-aortic balloon pump assist. The importance of this lies not only in the appearance of retrograde flow, as was very astutely pointed out by Dr. Brass, but also in the strange waveform that preceded it.

We have had the opportunity of studying several patients treated with intra-aortic balloon pumps. The waveforms we have found do not show such a prominent dicrotic notch and, therefore, do not have as much diastolic flow drop as in Dr. Brass's patient, particularly at the point marked in the cardiac cycle (Figure 1). This point corresponds to the period during which the aortic balloon is being inflated, and it seems paradoxical that, in Dr. Brass's patient, the diastolic flow velocity drops. One plausible explanation is that the subject in question develops functional aortic valve insufficiency upon inflation of the balloon, which would result in a temporary loss of pressure in the proximal aorta resulting in a drop in cerebral blood flow velocity. Upon reaching a critical point of balloon inflation, however, the volume and pressure would stabilize across the aortic valve, leading to a progressive increase in cerebral blood flow velocity. This would then be followed by the drop caused by deflation of the balloon, which could result in reversal of the direction of cerebral blood flow, as noted by the author in two of his patients.

The second point of interest was the fact that the mean velocity calculated for the patient shown in the article, while the balloon was functioning, was higher than that found while the patient was in sinus rhythm. Our experience is to the contrary; that is, during intra-aortic balloon pump assist, the mean velocity in the brain is usually lower than that found during spontaneous cardiac function. We think these points are important because, in fact, they validate Dr. Brass's position about the importance of TCD monitoring in the study of these individuals. In the future, TCD may not only help regulate the functioning of the pump, but also identify individuals at increased risk for cerebral ischemia/hypoxia during treatment with this device. We wonder whether Dr. Brass would like to elaborate upon these points.

Camil R. Gomez, MD, FACA
James R. McLaughlin, DO
Philip C. Njemanze, MD
Department of Neurology

Camilo R. Gomez, MD, FACA
James R. McLaughlin, DO
Philip C. Njemanze, MD
Department of Neurology
Letters to the Editor

The following is in response:

To the Editor:

Dr. Gomez brings out several important points about intra-aortic balloon pumps (IABPs). I did not specifically state the ratio between the spontaneous cardiac cycle and the IABP shown in my figure, which was 1:1. A similar velocity profile was seen in other pumping ratios, but only during assisted systoles.

The significance of the “strange waveform” can be understood by examining the waveform recorded in the radial artery. The first peak in the cycle corresponds to blood flow from the spontaneous left ventricular systole. The next peak is the systole produced by the balloon (diastolic augmented peak).

I do not think the trough between these two peaks represents a dicrotic notch. In general, balloon inflation is timed to occur just before the dicrotic notch. The middle cerebral artery velocity envelope in my figure very closely matches the radial artery pressure tracings. The reversal of flow occurs late in diastole, coincident with balloon deflation. Waveforms were identified based on the well-recognized patterns in the radial artery.

Dr. Gomez’s hypothesis for the effects of cerebral blood flow with aortic-valve insufficiency with an IABP is certainly an interesting one. It should be easy to investigate this point, and the effects of aortic insufficiency without an IABP, with transcranial Doppler (TCD).

Dr. Gomez’s second point brings up a crucial question: What effect does an IABP have on the cerebral circulation? The answers may be different for cardiogenic shock and myocardial ischemia without failure. I do not believe the minor differences noted in the mean velocity are significant. There is variability in the internally computed values for velocity (and pulsatility) on the TCD machines. It is probably even greater with the highly pulsatile velocity profiles seen with an IABP. I strongly encourage other TCD investigators and manufacturers to develop, and validate, better quantitative methods for these types of investigations to help address these important issues.

The IABP and other left ventricular assist devices are used to reduce cardiac oxygen demands while augmenting coronary perfusion. This technique of diastolic pressure augmentation and afterload reduction has an important role in the management of patients with refractory angina, cardiogenic shock, or in association with cardiothoracic procedures. The success of these devices is often gauged by the disappearance of anginal pain, improved cardiac output and blood pressure, and improved renal function. These are important measures for survival, but ignore the critical question of brain perfusion.

Our goal in reporting these findings was to point out that with the TCD, issues of cerebral perfusion can be addressed for left ventricular assist devices. Given the flexibility of the triggering of balloon inflation on most of these devices, it may be possible to adjust to the timing to maximize cerebral perfusion. Perhaps future models will use a combination of cardiac and cerebral measures to adjust the timing and inflation pulses of these and other left ventricular assist devices.

Lawrence M. Brass, MD
Yale Stroke Program, Department of Neurology
Yale Medical School
New Haven, CT

References


Infusion of Recombinant Tissue Plasminogen Activator for Treatment of Basilar Artery Occlusion

To the Editor:

Multiterritorial brain stem ischemic syndromes due to vertebrobasilar artery occlusion are associated with mortality rates of over 70%.1,2 It has been shown that local intra-arterial application of streptokinase and urokinase in patients with angiographically
Effect of intra-aortic balloon pumps on cerebral circulation.
C R Gomez, J R McLaughlin and P C Njemanze

Stroke. 1990;21:1512-1513
doi: 10.1161/01.STR.21.10.1512.b

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/21/10/1512.2.citation

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org/subscriptions/