Resumption of Anticoagulation During Hypertensive Cerebral Hemorrhage With Prosthetic Heart Valve

To the Editor:

We would like to bring to your attention our recent dilemma of when to re-anticoagulate a patient with a hypertensive cerebral hemorrhage who concurrently has a prosthetic heart valve.

The patient, a 63-year-old woman, had had her aortic heart valve replaced in 1978 with a modified Bjork-Shiley prosthetic valve and had been taking warfarin without bleeding complications. She was also hypertensive and treated with enalapril. She was admitted to a local community hospital after having been found unconscious and was later noted to have a right hemiparesis with attention, language, and memory abnormalities. Computed tomography (CT scan) showed a left thalamic hemorrhage that had extended into the third ventricle, the internal capsule, and the caudate nucleus. The anticoagulant was stopped after admission, and she was transferred to our service a few days later.

We faced the difficult decision of whether to restart the warfarin and risk the possibility of rebleeding or to wait an indefinite period of time during which the risk of embolization would be great. An extensive literature search directed at guiding our strategy was fruitless. We decided to treat her with platelet antiaggregants for 2 weeks (empirically chosen) and then restart the warfarin. On Day 10, however, she became completely unresponsive to verbal stimuli, inattentive to her surroundings, and showed complete motor deficit. Repeat CT scan failed to show any new areas of bleeding, and she was therefore started on heparin i.v. drip at a rate of 800 units/hr without any preceding loading dose. In <24 hours she began to improve and eventually was switched from heparin to warfarin. No rebleeding occurred and her neurologic condition has remained stable.

Literature supporting a rationale for re-anticoagulating patients in this setting is not available. Although the natural history of intracerebral hemorrhage suggests that this is a monophasic event, it is unknown whether the addition of anticoagulant drugs increases the risk of rebleeding. We suggest that reinstitution of anticoagulant therapy after hemorrhage suggests that this is a monophasic event, it is unknown whether the addition of anticoagulant drugs increases the risk of rebleeding. We suggest that reinstitution of anticoagulant therapy after hemorrhage is safe.

We conclude that the timing of re-anticoagulation in this setting should be individualized and based on an assessment of both the specific risk factors related to the valve and potential causes leading to the hemorrhage. An average waiting period of 19 days in our patients proved to be safe.

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References

Atrial Septal Aneurysm as a Cause of Cerebral Embolism in Young Patients

To the Editor:

The article by Belkin and associates,1 in which they reported a high prevalence of embolic events in a series of 36 consecutive patients with atrial septal aneurysm, has several additional important clinical implications besides those put forth by the authors. First, atrial septal aneurysm should always be considered among the cardiac causes of cerebral embolism that can be detected by echocardiography in young patients.1

Second, the consideration should be even more serious if the patient develops simultaneous embolic events in both the systemic and pulmonary circulation since biatrial myxoma and paradoxical embolism are the only two other conditions that can cause 'bilateral' embolization.

Third, the frequent association of paradoxical embolism with right-to-left atrial shunting with atrial septal aneurysm found by Belkin and associates1 results from their aggressive use of contrast echocardiography.4 The sensitivity of contrast echocardiography in the detection of paradoxical embolism, of course, might be further enhanced had they used it in conjunction with the Valsalva maneuver.2

As a matter of fact, when requesting echocardiography of a stroke patient in the evaluation for cardiogenic embolism, the referring physician should not only request contrast study but also specify the application of the Valsalva maneuver as routine echocardiography does not employ either of these maneuvers.2

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References


The following is in reply:

Dr. Cheng is certainly correct. We always employ the Valsalva maneuver when contrast echocardiographic studies are negative at rest.

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Does Transcranial Doppler Ultrasonography Provide Information About Cerebral Microcirculatory Flow?

To the Editor:

In his editorial, J.C. Grotta1 regrets that "easily administered bedside studies such as ... transcranial Doppler do not provide information about microcirculatory flow". According to our experience with transcranial Doppler ultrasonography, this statement cannot be made unmodified. We use transcranial Doppler to detect changes of median artery flow velocity during breathing tests or Vasalva maneuvers.2,3 These transcranial Doppler examinations can be performed as simple bedside tests. In patients with lacunar infarctions or white matter lucencies due to Binswanger's disease, the expected changes of median cerebral artery blood flow velocity during breathing or the Valsalva maneuver are often diminished or absent (unpublished observations). Similar abnormalities of autonomic regulation are detectable by transcranial Doppler measurements in patients with longstanding arterial hypertension but without lacunar infarctions or white matter lucencies on computed tomography examinations. A study to describe the specificity and sensitivity of these transcranial Doppler tests in detecting cerebral microvascular disease is under way at our department.

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Resumption of anticoagulation after intracranial bleeding in patients with prosthetic heart valves.
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