Angiographic Features in Chinese Patients With Occlusive Cerebrovascular Disease

To the Editor:

Caplan et al in their recent review article 1 mentioned, based on personal communications, that angiography of Chinese patients with ischemic stroke frequently reveals middle cerebral artery (MCA) occlusive disease while internal carotid artery (ICA) disease is unusual. Results of our previous angiographic study on 47 Chinese patients with ischemic stroke frequently revealed middle cerebral artery (MCA) occlusion. 


References


Bed Rest After Ischemic Stroke Is Not a Main Reason for the Decline in Arterial Blood Pressure

To the Editor:

I should like to comment on the interesting paper by Grotta et al, "Baseline hemodynamic state and response to hemodilution in patients with acute cerebral ischemia," published in Stroke. 1 This is a pilot study of 9 consecutive patients at the acute stage of cerebral infarction (CI). We agree with the authors that despite the interest in hemodilution for acute stroke and its basis in cardiovascular physiology, there are no well known published data up to 1985 on the baseline systemic (or general) hemodynamic status of patients entering the hospital with acute CI. Such data are necessary for designing protocols that will optimize the desired effects on viscosity and cardiac pump function and that will be safe in patients who often have impaired cardiac function. 1-4 However, we do not agree with the authors that the gradual decline in mean arterial blood pressure, which is often seen in patients after stroke, is due to bed rest. Grotta et al have measured main parameters of systemic hemodynamics during the first 3 days in 9 patients after CI. We have repeatedly measured main parameters of systemic hemodynamics during the first 2 weeks with 76 patients after CI. Some data are now available, 1,3 and another part is in press (in Cor et Vasa). Some information we can give here.

A clinical series of 76 patients with CI was repeatedly investigated, using noninvasive techniques, i.e., integral rheography of the whole body. This method makes it possible to evaluate main parameters of general hemodynamics (left ventricular stroke volume, heart rate, cardiac output, and some others). It was shown that the heart rate, the cardiac output, and all components of the arterial pressure decrease considerably during the acute stage of the disease. These patients were divided into 2 groups: 1) patients with the ability to walk (including with assistance, in = 60) after 2 weeks, and 2) patients without the ability to walk (n = 16), who also have the lowest activities of daily living (ADL) score. The treatment in both groups was similar. There were no comatose patients in either group, no clinically diagnosed myocardial infarctions, and no deaths. The left ventricular stroke volume, heart rate, and cardiac output, and diastolic blood flow did not differ significantly between the 2 groups. There was a significant difference between the systolic blood pressure values (mean ± SEM): 157 ± 5 and 178 ± 4 mm Hg (p < 0.05) on the first day after CI, but no difference on the 14th day (142 ± 4 and 144 ± 7 mm Hg, respectively, for Groups 1 and 2). The present data show that the patients with poor prognosis (Group 2) have a higher initial systolic blood pressure than the others; however, in both groups a significant (p < 0.05) decrease in blood pressure takes place. We conclude that bed rest or the inability to walk (low ADL score) 2 weeks after an ischemic stroke is not a main reason for the...
lowering of blood pressure or other parameters of systemic (general) hemodynamics. The decline in arterial blood pressure after CI is a well-known phenomenon. A less known pathophysiologic mechanism is hemodynamics. The decline in arterial blood pressure after CI is a well-known phenomenon. We suppose that changes in general hemodynamics are secondary after the initial compensatory increase beyond their preinfarction levels and represent a natural course of changes in general hemodynamics after CI. Systemic hemodynamic monitoring and colloid osmotic pressure measurements are obligatory parts of this study to ensure the safety of patients with cerebrovascular disease, but we believe that noninvasive monitoring for routine investigation is less harmful than invasive monitoring.

We await with interest Dr. Grotta’s report on his experiences concerning hemodilution after stroke.

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References

The following letter is in response to Dr. Kauba:

To the Editor:

We appreciate Dr. Kauba’s remarks and interesting data pertaining to the decline in blood pressure after stroke. We did not mean to imply in our paper1 that this decline is caused by bed rest, only that it is often seen in patients who are at bed rest. The exact mechanism is unknown but we suggest two possibilities: 1) normalization after an initial transient rise in blood pressure, or 2) reduced cardiac output due to volume depletion.

There is often a transient increase in blood pressure after stroke (probably due to a release of catecholamines) and this finding is supported by Dr. Kauba’s data showing that the greatest elevation in blood pressure is seen in patients with the worst strokes. A subsequent decline in blood pressure during the first 72 hours after stroke may reflect normalization back to baseline levels. A recent study demonstrated a 10% decrease in mean arterial blood pressure in all stroke patients within the first 24 hours of hospitalization, but the decline was greatest in patients with the highest admission blood pressure. A transient rise in blood pressure followed by normalization over the next several days is further suggested by our experience administering a calcium blocker after stroke. When the drug, a vasodilator which lowers blood pressure, is titrated against blood pressure, frequent dose adjustments must be made in the first 24 hours since the transient rise in blood pressure is probably blunted by the drug. Within a day or so, dose adjustments are less necessary since the patient’s blood pressure has probably returned to baseline levels.

Dr. Kauba concludes from his data that the decline in blood pressure after stroke is “mostly due to slowing of the heart rate. No significant changes in the left ventricular stroke volume occur.” Under normal circumstances, however, a decline in heart rate without accompanying decline in stroke volume should not result in decreased blood pressure. However, many stroke patients are volume depleted, and their cardiovascular system may already be maximally compensated to maintain blood pressure. A further drop in cardiac output due to decreased heart rate might result in hypotension. One explanation why a decline in blood pressure was not seen in our hemodiluted patients might be that any volume depletion was rapidly corrected.

Finally, we agree that noninvasive monitoring would be preferable to Swan-Ganz catheters, but we have adopted invasive monitoring for the present protocol to 1) guide therapy aimed at optimizing cardiac output, and 2) determine a safe and effective therapeutic algorithm that could then be applied universally without invasive or detailed monitoring.

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References
Bed rest after ischemic stroke is not a main reason for the decline in arterial blood pressure.

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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/18/3/686.2.citation