Transient Ischemic Attacks Associated with Hypotension in Hypertensive Patients with Carotid Artery Stenosis

ROBERT L. RUFF, M.D., PH.D., WILLIAM T. TALMAN, M.D., AND FRANK PETITO, M.D.

SUMMARY In a group of 132 patients with transient ischemic attacks (TIAs) 7 patients (4 men, 3 women, ages 64 to 81) had TIAs preceded by hypotension. The average fall of mean blood pressure during an attack was 26.4 ± 5.5 mm Hg (SD). Only one of these patients had a TIA which was not preceded by hypotension. This episode occurred during a paroxysm of coughing. All 7 of these patients had hypertension, and cerebral arteriography performed in 4 of the 7 revealed hemodynamically significant carotid artery stenosis. Each of the 4 patients developed hypotension and a TIA after the procedure. Twenty of the other patients had hypotensive episodes but did not develop focal neurological deficits. None of these patients had carotid artery stenosis. This study suggests that hypertensive patients with carotid artery stenosis may be at risk to develop focal cerebral ischemia during acute hypotensive episodes.

THE ROLE of hypotension in the etiology of transient ischemic attacks (TIAs) and stroke is controversial. Denny-Brown and colleagues suggested that hypotension could induce focal neurological deficits in patients with carotid artery stenosis. More recently, several authors have stated that hypotension rarely, if ever, causes TIAs or stroke.

We sought to determine if a sub-population of patients with TIAs is susceptible to hypotensive episodes. We have found that the acute lowering of blood pressure resulting from antihypertensive medications, postural change, or cerebral arteriography was associated with transient focal neurological deficits in hypertensive patients with hemodynamically significant carotid artery stenosis.

Methods

The study group consisted of 132 patients admitted to the New York Hospital-Cornell Medical Center between 1975 and 1980 with a clinical diagnosis of TIA which met the following requirements: 1) the clinical syndrome in each case satisfied established criteria for TIA; 2) each patient was evaluated by a staff neurologist who formulated or agreed with the diagnosis; and 3) each was studied with a lumbar puncture and computed tomography which showed no evidence of a mass lesion, cerebral infarction, or hemorrhage. Eighty-nine of these patients had aortic arch studies (87 of these were believed to have ischemia in those parts of the brain supplied by the carotid artery). Carotid artery stenosis with greater than 85% reduction of the minimal common or internal carotid artery diameter was considered hemodynamically significant. Blood pressure, assessed by sphygmomanometry, and heart rate, determined by palpation of the peripheral pulse, were determined every 2–4 hours. The measurement interval was decreased to 15–30 minutes if a previously hypertensive blood pressure fell below the normal range (140/90 mm Hg) or if provocative measures such as postural changes, antihypertensive drug treatment, or cerebral arteriography were planned. Hypertension was defined by persistent elevation of the diastolic blood pressure above 90 mm Hg and of the mean blood pressure (systolic + diastolic blood pressure/2) above 130 mm Hg. A hypotensive episode consisted of a decrease in the mean blood pressure of at least 20 mm Hg which occurred in less than 4 hours.

The evaluation period was the acute hospitalization which lasted from 18–48 days. Probabilities were calculated using the two tailed Fisher Exact Test. Values were recorded as mean ± SD.

Results

All of the patients had TIAs during hospitalization, but only seven had TIAs associated with hypotension (table). These patients would develop neurologic signs which replicated their previous TIAs within 15 minutes of the onset of hypotension. The average fall in mean blood pressure was 26.4 ± 5.5 mm Hg. Patient 2 had a TIA which was not preceded by hypotension. This occurred during a 10 minute coughing paroxysm. The TIAs usually resolved within 2 hours of restoring the patient's blood pressure to its basal level.

Hypotension was associated with antihypertensive therapy (patients 1, 3–7), with cerebral arteriography (patients 1–4), and with postural change (patients 2–7). Antihypertensive treatment consisted of diuretics and vasodilators in 5 patients. Patient 7 received alpha-methyldopa in addition to a diuretic. In the study group there were 20 other patients who had hypotensive episodes unaccompanied by a focal neurological deficit. The average fall of mean arterial pressure during the hypotensive episodes in these patients was 25.8 ± 4.6 mm Hg. The patients with hypotension-related TIAs had a greater incidence of hypertension (7/7 vs 45/125,
interpreted as an electrical phenomenon secondary to developed focal electroencephalographic (EEG) slowing periods of hypotension. The EEG changes were not accompanied by TIAs and thus could have been occurring in the vascular distribution of previous TIAs during periods of hypotension. The EEG changes were not accompanied by TIAs and thus could have been interpreted as an electrical phenomenon secondary to previous structural damage. McCutchen reported that a patient with hypertension and left middle cerebral artery occlusion had TIA's which could be reproduced by hypotension. This patient developed hypotension and a TIA during examination by cerebral arteriography.

Kendall and Marshall demonstrated that TIA's could be replicated in only one of 37 patients when their blood pressure was acutely lowered. Though the subjects of their study were selected only for having previously had TIAs, the implication drawn from their results was that hypotensive episodes rarely caused symptomatic transient ischemia in any patients. Although TIAs were temporally related to hypotension in only 5.3% of patients in the present study, the incidence rose significantly to 25% in patients with hemodynamically significant carotid artery stenosis and to 44% in patients with carotid artery stenosis and hypertension. Therefore, in a select group of patients, hypotension may be an important etiologic factor in the genesis of TIAs.

It is unlikely that focal cerebral ischemia in our patients caused the hypotension. In every patient the hypotension preceded symptoms and thus seemed premonitory to the TIA. In addition, Gross and Marshall have reported that patients who developed strokes secondary to carotid artery stenosis did not demonstrate alterations in blood pressure.

The pathophysiological basis for the relationship between hypotension and TIAs cannot be determined from the current work. However, it seemed necessary for patients to have both hypertension and hemodynamically significant carotid artery stenosis. The possible mechanisms are a focal decrease in cerebral blood flow, impaired autoregulation, and altered baroreceptor function. The stenotic carotid artery may "require" the abnormally elevated arterial pressure in order to maintain flow within the brain. Thus, without implying any compromise of intrinsic autoregulation of cerebral blood flow, a sharp decrease in the pressure head in the artery would proportionately decrease the flow distal to the stenosis. Such focal symptoms from transient decreases in flow are implied by reports of patients developing TIAs with bouts of coughing which is known to decrease cerebral blood flow.

<table>
<thead>
<tr>
<th>Number of Episode</th>
<th>Mean Blood Pressure (MBP) (mm Hg)</th>
<th>Fall in MBP (mm Hg)</th>
<th>Change in heart rate (beats/min)</th>
<th>Patient</th>
<th>Age</th>
<th>Sex</th>
<th>TIA Distribution</th>
<th>% Stenosis Artery</th>
<th>Baseline Mean Blood Pressure (MBP) (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>4</td>
<td>140-150</td>
<td>25-35</td>
<td>-5 to +6</td>
<td>1</td>
<td>69</td>
<td>M</td>
<td>L Carotid</td>
<td>90%/L Carotid</td>
<td>145-150</td>
</tr>
<tr>
<td>3</td>
<td>135</td>
<td>20-25</td>
<td>-2 to 0</td>
<td>2</td>
<td>64</td>
<td>F</td>
<td>L Carotid</td>
<td>90%/L Carotid</td>
<td>135</td>
</tr>
<tr>
<td>3</td>
<td>135-145</td>
<td>30-35</td>
<td>0 to +6</td>
<td>3</td>
<td>72</td>
<td>F</td>
<td>R Carotid</td>
<td>60%/L Carotid</td>
<td>135-145</td>
</tr>
<tr>
<td>3</td>
<td>135-140</td>
<td>20-25</td>
<td>-8 to +4</td>
<td>4</td>
<td>69</td>
<td>M</td>
<td>R Carotid</td>
<td>60%/L Carotid</td>
<td>135-140</td>
</tr>
<tr>
<td>2</td>
<td>145-150</td>
<td>30-35</td>
<td>0 to +6</td>
<td>5</td>
<td>81</td>
<td>F</td>
<td>L Carotid</td>
<td>-</td>
<td>145-150</td>
</tr>
<tr>
<td>1</td>
<td>135</td>
<td>25</td>
<td>+6</td>
<td>6</td>
<td>69</td>
<td>M</td>
<td>L Carotid</td>
<td>-</td>
<td>135</td>
</tr>
<tr>
<td>1</td>
<td>135</td>
<td>35</td>
<td>0</td>
<td>7</td>
<td>73</td>
<td>M</td>
<td>Vert-Bas</td>
<td>-</td>
<td>135</td>
</tr>
</tbody>
</table>

Discussion

This study suggests that patients with hemodynamically significant carotid artery stenosis and hypertension are predisposed to focal ischemic neurological deficits when their blood pressure is rapidly lowered. This may also apply to patients with vertebrobasilar stenosis and hypertension but the one patient in the study with hypotension-related TIAs in the vertebrobasilar area did not have arteriography.

The results in this clinical study are similar to those of Meyer et al., who reported that hypertensive patients with extensive extracranial atherosclerosis developed focal electroencephalographic (EEG) slowing in the vascular distribution of previous TIAs during periods of hypotension. The EEG changes were not accompanied by TIAs and thus could have been interpreted as an electrical phenomenon secondary to
Cerebral blood flow autoregulation could have been compromised either a) locally distal to the stenosis or b) diffusely due to a shift of the autoregulatory curve with hypertension. In addition, atherosclerotic carotid disease may impair baroreceptor reflexes. In our patients with hypotension-related TIAs, the lack of a tachycardic response to the hypotensive episodes suggests that the baroreflex may have been compromised. Thus the presumed fall in cardiac output (decreased arterial pressure with stable heart rate) could have contributed to the development of focal cerebral ischemia.

This study suggests that patients with hypertension and carotid stenosis have a predilection for the development of hypotension and TIAs after arteriography. Each patient with hypotension-related TIAs suffered a TIA during a hypotensive episode following arteriography while no other patient did. Patients with carotid artery stenosis, in general, have a greater risk of neurological complications from cerebral angiography, and it is possible that the high incidence of TIAs in these patients simply reflected this risk. However, the association of hypotension and TIAs in precisely the same distribution as before the arteriogram speaks for a common etiology. Arteriography may have provoked symptoms by 2 known mechanisms. First, the radiographic contrast material can impair effective autoregulation of cerebral blood flow and, by altering the viscosity of the blood, may produce capillary sludging. Second, the contrast material, as a potent osmotic diuretic, can precipitate hypotension.

Based on the results of this study we conclude: Hypertensive patients with hemodynamically significant carotid (and perhaps vertebrobasilar) artery stenosis may be at a risk for developing symptomatic focal ischemia if their blood pressure is acutely lowered. In these patients blood pressure should be carefully maintained during and after cerebral arteriography.

References

Transient ischemic attacks associated with hypotension in hypertensive patients with carotid artery stenosis.

R L Ruff, W T Talman and F Petito

doi: 10.1161/01.STR.12.3.353

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/12/3/353