Cerebral Hemispheric "Counter-Steal" Phenomenon During Hyperventilation in Cerebrovascular Diseases

BY G. RAIVONDO PISTOLESE, M.D.,* VITTORIO FARAGLIA, M.D.,* ALESSANDRO AGNOI, M.D.,† MASSIMILIANO PRENCIPE, M.D.,† ENRICA PASTORE, M.D.,* CARLO SPARTERA, M.D.,* AND PAOLO FIORANI, M.D.*

Abstract:
Cerebral Hemispheric "Counter-Steal" Phenomenon During Hyperventilation in Cerebrovascular Diseases

Use of hyperventilation (HV) was recently suggested for the treatment of acute cerebrovascular insufficiency. There is indeed no general agreement on the effectiveness of hyperventilation even though clinical and experimental findings could support its value in clinical use.

During carotid surgery, hyperventilation was used in order to attenuate cerebral ischemia effects during carotid clamping, and a counter-steal phenomenon affecting a whole cerebral hemisphere was demonstrated. This suggests the role that hyperventilation may play in the treatment of cerebral ischemia.

Methods
A 70-year-old man affected by aorto-iliac atherosclerosis underwent left aorto-femoral dacron bypass surgery. In April, 1971, he had a sudden loss of consciousness for a few minutes following subjective vertigo, transient left-arm monoparesis, and speech disorders. In May, 1971, he suffered from sudden right amaurosis for a few minutes and was hospitalized.

At physical examination, the patient was hypertensive (180/100 mm Hg) and exhibited a vascular bruise on the right carotid artery and a left...
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mild hemiparesis. The EEG and brain scan were normal. Angiography of the four cervical vessels showed severe stenosis at the origin of the internal right carotid artery (fig. 1).

An internal right carotid endarterectomy with dacron patch was performed in June, 1971. During surgery rCBF from eight regions was studied in basal conditions and during hyperventilation.

Xenon 133 was injected into the common carotid artery after clamping of the external carotid artery. For the measurement of CBF during clamping, the common carotid artery was occluded as soon as the curve reached the maximal value.10

The rCBF was calculated from the two-minute slope by automatic analysis.22 Mean arterial blood pressure (MABP) was recorded by means of an arterial catheter and continuously controlled. The same arterial catheter was used to take arterial blood samples for pH, P\textsubscript{O\textsubscript{2}}, and P\textsubscript{CO\textsubscript{2}} measurement. Anesthesia was induced by 500 mg sodium thiopental and maintained with oxygen, 50% nitrous oxide and 0.4% methoxyflurane (Penthrane). Relaxation was maintained by means of d-tubocurarine chloride.

Respiration was controlled with the aid of an Engstrom respirator. Normocapnia was reached following the Engstrom-Herzog nomogram, and hyperventilation was obtained by increasing the amount of oxygen and nitrous oxide 50%, while the percentage of methoxyflurane was unchanged.10

During hyperventilation arterial blood pressure was maintained at basic levels by the angiotensin infusion (Hypertensine, Ciba).

All the techniques and methods employed have been described elsewhere.10

Results

The results are reported in table 1 and figure 2.

REGIONAL CEREBRAL BLOOD FLOW (rCBF) UNDER BASIC CONDITIONS

rCBF values for the hemisphere which has been studied ranged between 29.5 ml/100 gm/min and 41.6 ml/100 gm/min with an average of 35.3 ml/100 gm/min. P\textsubscript{CO\textsubscript{2}} was 40 mm Hg and MABP was 136 mm Hg.

*FIGURE 1*

The right and left carotid artery angiography shows a severe stenosis of the right internal carotid artery at the origin.
TABLE 1

rCBF Values During Normoventilation and Hyperventilation

<table>
<thead>
<tr>
<th>Channel</th>
<th>rCBF (ml/100 gm/min)</th>
<th>Base</th>
<th>Clamping</th>
<th>Hyperventilation (HV)</th>
<th>HV + hypotension</th>
<th>HV + clamping</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td></td>
<td>36.3</td>
<td>37.6</td>
<td>45.4</td>
<td>25.8</td>
<td>27.9</td>
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<tr>
<td>2</td>
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<td>36.5</td>
<td>40.2</td>
<td>40.7</td>
<td>24.2</td>
<td>27.2</td>
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<tr>
<td>3</td>
<td></td>
<td>41.6</td>
<td>41.7</td>
<td>61.6</td>
<td>25.8</td>
<td>32.4</td>
</tr>
<tr>
<td>4</td>
<td></td>
<td>31.8</td>
<td>31.6</td>
<td>76.6</td>
<td>20.6</td>
<td>23.5</td>
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<td></td>
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<td>35</td>
<td>45.5</td>
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<td>26</td>
</tr>
<tr>
<td>6</td>
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<td>34.5</td>
<td>41.7</td>
<td>69.9</td>
<td>22</td>
<td>29.4</td>
</tr>
<tr>
<td>7</td>
<td></td>
<td>29.5</td>
<td>37.5</td>
<td>56.2</td>
<td>20.3</td>
<td>26</td>
</tr>
<tr>
<td>8</td>
<td></td>
<td>38.8</td>
<td>38.7</td>
<td>51.1</td>
<td>25.2</td>
<td>29</td>
</tr>
</tbody>
</table>

MABP (mm Hg) | 136 | 130 | 140 | 96 | 143 |
Paco2 (mm Hg) | 40 | 40 | 25 | 25 | 24 |
Pao2 (mm Hg) | 180 | 200 | 140 | 170 |
\(\text{apH} \) | 7.39 | 7.39 | 7.55 | 7.58 | 7.56 |
Time | 0 | 13' | 45' | 75' | 95' |

rCBF DURING CLAMPING

rCBF was not significantly affected by clamping. Its average value was 38 ml/100 gm/min, with a range between 31.6 ml/100 gm/min and 41.7 ml/100 gm/min. Paco2 was 40 mm Hg and MABP was 130 mm Hg. Clamping determined no reduction in rCBF since the internal carotid artery was affected by severe stenosis.

rCBF DURING HYPERVENTILATION

After 20 minutes of hyperventilation, Paco2 values of 25 mm Hg were obtained and kept constant while PaO2 values were 200 mm Hg and pH was 7.55. MABP was 140 mm Hg. rCBF was investigated ten minutes after such values were reached.

CBF increased in all regions and its average value was 55.7 ml/100 gm/min, with a range between 40.7 ml/100 gm/min and 76.6 ml/100 gm/min (fig. 3). The mean percentage increase from basic values was 57%. To study autoregulation during hyperventilation, hypotension was then induced by stopping angiotensin infusion.

rCBF studies were performed again after reaching an MABP value of 96 mm Hg while PaCO2 was 25 mm Hg. Under such conditions cerebral blood flow decreased in all regions. Its average value was 23.4 ml/100 gm/min, with a range between 20.3 ml/100 gm/min and 25.8 ml/100 gm/min (fig. 2).

CLAMPING DURING HYPERVENTILATION

rCBF studies were carried out during clamping after the MABP was raised to 143 mm Hg. In such conditions the average CBF value was 25.1 ml/100 gm/min, with a range between 23.5 ml/100 gm/min and 32.4 ml/100 gm/min (fig. 2).

Surgery was performed during normocapnia and normotension. Carotid clamping was maintained for 20 minutes. The patient woke normally and had a normal postoperative course.

Discussion

rCBF values under basic conditions ranged between 29.5 ml/100 gm/min and 41.6 ml/100 gm/min, with an average of 35.3 ml/100 gm/min, somewhat lower than the mean normal value (fig. 2). Such a reduction in CBF is probably related to the older age of the patient and to the presence of cerebral arteriosclerosis, in addition to a diminished metabolic demand during general anesthesia.

rCBF during two minutes of carotid clamping showed modification between -0.2 ml/100 gm/min and +8 ml/100 gm/min. We do not consider these variations significant with the two-minute slope method. Furthermore, the carotid clamping showed no modification of CBF since severe stenosis was already present, and perhaps this vessel did not participate to cerebral circulation. The study of rCBF during hyperventilation pointed out a homogeneous increase throughout the affected hemisphere.

This phenomenon may have occurred because the almost maximal vasodilatation and loss of responsiveness to CO2 were present in the affected hemisphere supplied by the right stenotic carotid artery.
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FIGURE 2

rCBF modifications during normoventilation and hyperventilation. The broken line indicates the mean value.

FIGURE 3

rCBF modifications during normoventilation and hyperventilation show a "counter-steal" phenomenon in the right cerebral hemisphere.

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In such conditions the hyperventilation could induce a blood shifting from the contralateral hemisphere, which reacts by vasoconstriction to hypocapnia, and a paradoxical reaction may result. The MABP drop confirmed the supposed loss of autoregulation in all the regions where “counter-steal” took place; then it confirmed the occurrence of this redistribution phenomenon.

On the contrary, hyperventilation during clamping has not again determined a “counter-steal” phenomenon even though MABP was constant. Therefore, this different response to hyperventilation may be secondary to the reduction of CBF during the hypotension test. Then the absence of blood shifting toward the right affected hemisphere, resulting from hyperventilation after hypotension, may be explained by reduced response to CO₂ which the vessels of the contralateral hemisphere could develop.

In conclusion, our results show a “counter-steal” phenomenon of a whole hemisphere and suggest the role that hyperventilation may play in the treatment of cerebral ischemia.

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