Internal Jugular Oxygen Saturation and Arteriovenous Oxygen Difference During Artificial Embolization of Arteriovenous Malformations*

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Serial cerebral angiography has been used during extracranial carotid embolization of cerebral arteriovenous malformations (AVM) to observe the behavior of the emboli and to evaluate the extent of obliteration of their arterial supply. When the flow to the malformation is greatly reduced, a “critical point” is reached, with lessening of the sump effect, whereby there is no longer a preference for the emboli to enter the AVM. We consider serial cerebral angiography a useful method but not totally reliable for predicting the approaching danger point when the emboli are apt to lodge in normal vessels.

Hemodynamic studies in AVM indicate that, in bypassing the capillary barrier, the cerebrovascular resistance (CVR) is reduced, therefore the cerebral blood flow (CBF) is increased, and with it the jugular oxygen saturation (JSO). The arteriovenous oxygen difference ((A-V)O₂) is reduced, but the cerebral oxygen consumption (CMRO₂) remains normal. Surgical removal of the AVM is accompanied by reduction in cerebral blood flow and jugular oxygen saturation, as well as elevation in the arteriovenous oxygen difference. Embolization of the AVM produces progressive reduction of the abnormally increased cerebral blood flow accompanied by reduction in JSO₂ and elevation in (A-V)O₂, just as is seen with direct surgical removal. The CBF reductions can be calculated from the cerebral (A-V)O₂ if the cerebral oxygen consumption remains constant.

We have evaluated the changes in (A-V)O₂ and JSO₂ in blood from the ipsilateral jugular bulb and carotid arteries under general anesthesia during the process of embolization of arteriovenous malformations which were considered not amenable to direct surgical attack.

Procedure
Seven patients with cerebral arteriovenous malformations were studied during arterial embolization. Intratracheal anesthesia was used, supplemented by 50% oxygen; the level of anesthesia being approximately equivalent to stage III, plane 2, for ether anesthesia.

Arterial oxygen saturation, pO₂, pCO₂, and pH determinations from the ipsilateral carotid artery, were done throughout the procedure. A polyethylene catheter (P.E. 160) was inserted into the ipsilateral internal jugular vein to the jugular bulb. JSO₂ and (A-V)O₂ differences in volume percent correlations were done routinely every 10 minutes, as well as immediately following the instillation of emboli into the internal carotid artery. The oxygen content in arterial and venous blood was determined by the sum of each patient’s hemoglobin oxygen capacity in relation to the measured oxygen saturation, and the dissolved O₂. Dissolved oxygen was obtained as the product of the measured pO₂ and the appropriate O₂ solubility coefficient (Henry’s law). A photoelectric Oxymeter, model 10840, American Optical Company, was used to measure oxygen saturation. Serial cerebral angiography was also carried out repeatedly during the procedure.

Teflon beads from 1 to 4 mm in diameter were used for progressive embolization, numbered from 1 to 4, according to their size. The technique for embolization is similar to that reported by Luessenhop, et al.

Our patient group fell into malformations type C, D and E of Luessenhop, defined as

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follows: In type C the AVM is supplied by the middle cerebral artery, with some contribution from enlarged lenticulostriate arteries; in type D, the AVM is supplied equally by the anterior and the middle cerebral arteries; and in type E the entire contribution to the AVM comes from the anterior cerebral arteries.

Progressive embolization of the AVM was carried out on the basis of angiographic studies. JSO₂ and (A-V)O₂ were used to calculate relative cerebral blood flow, on the basis of the formula: \( \text{CBF} = \frac{\text{CMRO}_2}{(A-V)O_2} \times 100. \) Because our patients were operated on under general anesthesia, for practical purposes we assumed a CMRO₂ of 2.4 cc/100 gm/min.

Embolization was terminated when: 1) an (A-V)O₂ of 3.6 vol % or above was obtained; 2) the middle cerebral artery contribution in type D of AVM was occluded; or 3) beads went to a vessel not contributing to the malformation.

**Results**

Satisfactory angiographic evidence of significant obliteration of arteriovenous malformations was obtained in five of the seven cases. In these patients, the reductions in JSO₂ ranged from 3 to 19 %, and the elevations in (A-V)O₂ from 1.48 to 4.11 vol % (Table 1 and Fig. 1).

Type C malformations gave two types of response: 1) fast, significant elevations in (A-V)O₂, of 1.5 vol % or more, above the baseline figures, which grossly corresponded to 90% to 100% reductions in abnormal ce-

**TABLE 1**

<table>
<thead>
<tr>
<th>Patient</th>
<th>AVM Type</th>
<th>Hb Gm %</th>
<th>Arterial Blood (Carotid)</th>
<th>Internal Jugular O₂</th>
<th>(A-V)O₂</th>
<th>Abnormal CBF Reduction</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Oxygen</td>
<td>pH</td>
<td>I</td>
<td>F</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Sat % Tension Vol %</td>
<td>pCO₂</td>
<td>I* F*</td>
<td>I % F %</td>
</tr>
<tr>
<td>1</td>
<td>C</td>
<td>13.3</td>
<td>99.2 190 17.55 36</td>
<td>7.37 7.37</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>C</td>
<td>11.8</td>
<td>99.0 170 15.60 36</td>
<td>7.36 7.37</td>
<td>0.52</td>
<td>1.02</td>
</tr>
<tr>
<td>3</td>
<td>C</td>
<td>15.0</td>
<td>96.0 88 18.98 37</td>
<td>7.37 7.37</td>
<td>0.44</td>
<td>3.54</td>
</tr>
<tr>
<td>4</td>
<td>D</td>
<td>13.0</td>
<td>95.0 80 16.29 41</td>
<td>7.39 7.40</td>
<td>0.84</td>
<td>2.05</td>
</tr>
<tr>
<td>5</td>
<td>D</td>
<td>13.4</td>
<td>99.4 215 17.95 34</td>
<td>7.45 7.48</td>
<td>0.52</td>
<td>1.02</td>
</tr>
<tr>
<td>6</td>
<td>E</td>
<td>12.0</td>
<td>99.4 215 16.15 35</td>
<td>7.44 7.55</td>
<td>0.90</td>
<td>1.95</td>
</tr>
<tr>
<td>7</td>
<td>C</td>
<td>15.0</td>
<td>99.2 175 19.84 46</td>
<td>7.38 7.42</td>
<td>2.11</td>
<td>4.11</td>
</tr>
</tbody>
</table>

*I* = Initial.  
F = Final.
rebral blood flow (Cases 2 and 7, Fig. 2), and 2) slow, small (A-V)O$_2$ elevations from 0.5 to 1.21 vol % (Cases 1 and 3), which corresponded to 39% to 75% reductions in abnormal cerebral blood flow. Although preoperative angiograms in this type of AVM did not visualize the anterior cerebral artery, this vessel was well demonstrated in postembo
lization angiograms. A small remnant of the AVM could be seen in Case 7.

Type D malformation gave fast, significant elevations in (A-V)O$_2$, approximately 1 vol % or more, above baseline figures. These corresponded to the obliteration of the middle cerebral contribution to the AVM, and approximated reduction of the abnormal cerebral blood flow from 56 to 63% (Cases 4 and 5).

Beads went to a non-AVM vessel in Cases 3 and 6 with no observable neurological deficit. In Case 3, a type C AVM, a 1.5 mm bead occluded one of the normal branches of the middle cerebral artery when the (A-V)O$_2$ had increased from 0.84 vol % to 2.05 vol %, corresponding to 75% obliteration of the shunted blood flow through the lesion. In Case 6, a type E AVM, the first 2 mm bead occluded one branch of the middle cerebral distribution; the (A-V)O$_2$ was unchanged.

A late complication appeared in Case 5; 20 hours after the operation a central retinal artery occlusion occurred. The neurological status in the remainder of the patients was unchanged from the preoperative conditions.

**Discussion**

Gibbs, *et al.*, suggested that a low arteriovenous oxygen difference might indicate the presence of an intracranial arteriovenous fistula. Logan, *et al.*, demonstrated increased jugular oxygen saturation in cases of AVM. Shenkin, *et al.*, in their flow studies of cerebral AVM indicated that the cerebral blood flow is increased (average—$\frac{3}{2}$ times normal), the JSO$_2$ is also elevated, the (A-V)O$_2$ is reduced, and the CMRO$_2$ remained normal. Lennox and Gibbs (1932) and many others have suggested the possibility of calculating rapid changes in cerebral blood flow from observation of variations of the cerebral arteriovenous oxygen difference, assuming CMRO$_2$ to be constant. Studies in cerebral blood flow following extirpation of an AVM showed reduction of CBF of about 50% to normal and return to a normal (A-V)O$_2$.

We can conclude, therefore, that CMRO$_2$ remains constant, conditions which reduce the cerebral blood flow (including embolization) are accompanied by increase in (A-V)O$_2$ and decrease in the JSO$_2$ as well as the reciprocal 1/(A-V)O$_2$.

The blood sampled from the superior jugular bulb is considered free of contamination from extracerebral blood. Unilateral studies give CBF and CMRO$_2$ values representative of the whole brain. In cases of AVM, while there is a difference from side to side, both values are markedly elevated.

The calculation of CBF from the (A-V)O$_2$ difference is accurate in the unanesthetized patient, but not so under general anesthesia because of the associated changes in CMRO$_2$ and pCO$_2$. The cerebral oxygen consumption is decreased by general anesthesia depending on the agent and the depth
of the patient's temperature. Pentothal reduces CMRO$_2$ from 34% to 50%,$^{10,23,28}$ Halothane from 9% to 25%.$^{25,29,30}$ The deeper the anesthesia, the lower the CMRO$_2$. Each centigrade degree drop is accompanied by 6% reduction in CMRO$_2$.$^{16}$ However, once a steady level of anesthesia has been achieved, the cerebral oxygen consumption remains constant.$^{22}$ The reductions of CMRO$_2$ induced by surgical anesthesia further increase the JSO$_2$ and reduce the (A-V)O$_2$, accentuating the alterations already produced by the arteriovenous malformation.

The pCO$_2$ is proportional to the cerebral blood flow, under Halothane anesthesia and normocarbia there is slight elevation in CBF. With nitrous oxide anesthesia and normocarbia, the CBF remains normal.$^8$ The use of 50% oxygen in the inspired air has negligible effects on (A-V)O$_2$ and CBF.$^9$ Therefore, cerebral blood flow can be calculated from the arteriovenous oxygen difference under steady level of general anesthesia, if there are not important changes in pCO$_2$. For practical purposes we have assumed value for CMRO$_2$ of 2.4 cc/100 gm/min based upon published data for the effects of Halothane$^6$ anesthesia on CMRO$_2$.

As can be seen from the slope in the graph in Fig. 2, small changes at low (A-V)O$_2$ levels reflect significant reductions in cerebral blood flow, while at values greater than a 3 vol % (A-V)O$_2$ the CBF changes are less pronounced. An (A-V)O$_2$ of 4.5 vol % at 2.4 cc/100 gm/min CMRO$_2$ under general anesthesia will be equivalent to 6.3 vol % in the unanesthetized individual at 3.3 cc/100 gm/min of CMRO$_2$.

Lyons, et al.$^{19}$ in the assumption of 100% arterial oxygen saturation, established 60% JSO$_2$ as reflecting adequate cerebral blood flow and brain oxygenation in the unanesthetized patient. Their calculations are based on the work of Kety and Schmidt.$^{11}$ This value corresponds to 8 vol % (A-V)O$_2$ in a patient with 15 gm of hemoglobin; in the same patient under anesthesia with a lower CMRO$_2$, however, the (A-V)O$_2$ will be higher.

In patients with AVM subjected to cerebral embolization, the acceptable values of JSO$_2$ and (A-V)O$_2$ determined in conscious or anesthetized individuals and denoting adequate cerebral blood flow, cannot be entirely relied upon since persistence of even a small portion of the A-V shunt would produce an admixture of blood which would give persistently high values for JSO$_2$ and low values of (A-V)O$_2$. We have, therefore, arbitrarily assumed an (A-V)O$_2$ of 3.6 vol % or higher as an acceptable limit up to which embolization can be safely continued.

Embolization of the AVM was carried out on the basis of serial cerebral angiography, repeated during the procedure. JSO$_2$ and (A-V)O$_2$ were monitored at the same time, but they were not used to guide the embolization, unless values of 3.6 vol % or higher were obtained, at which point embolization was discontinued on the basis of the above reasoning.

Middle cerebral AVM (type C) when effectively obliterated, showed fast elevations

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**Fig. 3.** Relationship between cerebral blood flow and (A-V)O$_2$ at cerebral oxygen consumption rates frequently found under general anesthesia with 15 gm of hemoglobin per 100 cc of blood. The initial and final (A-V)O$_2$ values are represented by the horizontal arrows in each particular patient. This graph is made on the basis of constant CMRO$_2$ by the formula:

$$\text{CBF} = \frac{1}{(A-V)O_2} \cdot (A-V)O_2 \text{ being shown proportionally to } \frac{1}{(A-V)O_2}.$$  
(Patient 6 in this graph corresponds to No. 7 in the study.)

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in (A-V)O₂ to 1.5 vol % or more above the original figure. When the (A-V)O₂ elevations were slow and small, it was thought probably safe, if angiographically not contraindicated, to continue the embolization at least until a gain of 1 vol % (A-V)O₂ above original figures had been obtained. At this point pursuit to complete obliteration of the AVM based on angiography, JSO₂ and (A-V)O₂ was not considered safe, since they cannot accurately reflect the effective cerebral blood flow in the presence of even a small persistent AVM, nor the hemodynamic conditions which would suggest that additional emboli will lodge at appropriate sites. It is suggested that the use of radioisotopic transit time flow studies might be valuable.

Type D AVM should be considered satisfactorily embolized when fast elevations to 1 or more vol % (A-V)O₂ above the original figures are obtained; this reflects obliteration of the middle cerebral contribution to the AVM. To continue embolization beyond this point is not advisable, because the likelihood of the emboli entering the anterior cerebral artery is considerably less than is the dangerous possibility that the bead will occlude normal middle cerebral vessels.

Summary

We have felt that it is not safe to rely on angiography as the sole guide to embolization. Therefore, in seven cases, we have also evaluated changes in the arteriovenous oxygen difference and jugular oxygen saturation in blood from the ipsilateral jugular bulb and carotid arteries during the process of embolization of arteriovenous malformations. The values were found to be useful adjuncts to the technique of embolization but became less so as the point of total obliteration was reached.

References

18. Luessenhop, A. J., Kachmann, R. Jr., Shevlin, W., and Ferrero, A. A. Clinical evaluation of artificial embolization in the


