Hyperoxia of internal jugular venous blood in brain death

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The oxygen tension and acid-base values of internal jugular venous blood were compared to those of arterial and systemic mixed venous blood in 40 patients suffering from severe head injury. The results were divided into "brain death" and "coma groups" according to the clinical status when the sample was obtained. Hyperoxia of the jugular venous blood was a common finding in both groups, especially in the brain death group. The mean value of the arteriojugular venous oxygen content difference was 1.86 vol % in the brain death group, and 4.41 vol % in the coma group. Although most of the oxygen content differences in the brain death group were below 3 vol %, there was an overlap with the coma group at the low level of arteriojugular O₂ difference. For definitive assessment of brain death, the fact that jugular pO₂ was higher than that in systemic mixed venous pO₂ was more valuable than the decrease of arteriojugular oxygen difference. The extreme hyperoxia of the jugular venous blood is possibly a sign of brain death, but should not be interpreted as the result of decreased oxygen consumption in the brain, because most of the jugular venous blood is returned from the extracerebral tissues. The causes of jugular hyperoxia in the state of brain death are discussed.

KEY WORDS: head injury · brain death · cerebral blood flow · brain metabolism · oxygen tension · internal jugular venous blood

Our previous studies showed that the difference in the oxygen content of arterial and internal jugular venous blood was remarkably reduced in patients with severe head injury. Recently, the extreme reduction of the arteriojugular venous oxygen difference has been suggested as a sign of brain death. The purpose of our present study is to clarify whether the measurement of internal jugular venous blood gases and pH offered a definitive indicator of brain death and reflected the extent of impaired cerebral metabolism.

Materials and Methods
This series includes 40 patients unconscious after severe head injury treated in our trauma unit during the last 2 years. There were 21 fatalities in patients who showed a brain death syndrome in the terminal stage. The remaining 19 patients survived and were discharged from our special unit. In all patients, blood samples were frequently drawn from three sources at the same time: the femoral artery, superior bulb of the internal jugular vein by percutaneous punc-
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ture, and right atrium through an indwelling catheter. Arterial and jugular venous blood specimens (150 total) were obtained from all 40 patients; a total of 34 mixed venous blood specimens were obtained from 12 patients. The \( \text{pO}_2 \), \( \text{pCO}_2 \), and \( \text{pH} \) were determined with an electrode system.* Hemoglobin concentration was determined as cyanmethoglobin spectrophotometrically at 540\( \mu \). Oxygen content (vol %) was calculated from \( \text{pO}_2 \), \( \text{pCO}_2 \), \( \text{pH} \), and \( \text{Hb} \) concentration using Kelman-Nunn's computer nomogram.# The data were divided into two groups according to the clinical state at the time the blood samples were obtained:

**Brain Death Group.** The patients were deeply comatose; they had no spontaneous respiration, severe hypotension, fixed and dilated pupils, an isolectric-electroencephalogram (EEG), and cerebral circulatory arrest as represented by a nonfilling phenomenon in the cerebral angiogram. All patients in this group required mechanical ventilation with a high concentration of oxygen, and received a continuous drip of norepinephrine to maintain their blood pressure above 80 mm Hg.

**Coma Group.** These patients, although comatose with or without spontaneous respiration, did not show all the criteria of brain death.

**Results**

Figure 1 illustrates the internal jugular venous \( \text{pO}_2 \) of each group when plotted against arterial \( \text{pO}_2 \). Arterial \( \text{pO}_2 \) varied widely from 30 to 600 mm Hg, according to the pulmonary function and oxygen fraction of inspired gas in each patient. In the coma group, the jugular \( \text{pO}_2 \) was increasing only slightly with the arterial \( \text{pO}_2 \), whereas the comparable values in the brain death group were extremely high.

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*Electrode system II. meter, Model 113, manufactured by Instrumentation Laboratory Inc., Boston, Massachusetts.
The oxygen content of arterial and jugular venous blood is compared in Fig. 2. The fact that the arterial oxygen content was markedly reduced in both groups was due to depletion of Hb concentration rather than lowered pO₂. The oxygen content of the jugular venous blood (CjvO₂) was not so reduced when compared with arterial oxygen content (CaO₂), and, consequently, arteriojugular venous differences invariably decreased in both groups. The mean value of the oxygen content difference was 1.86 vol % in the brain death group, while in the coma group it was 4.41 vol %. Except for a few cases, the oxygen difference in the brain death group was below 3 vol %. However, there was a considerable overlap in arteriojugular oxygen difference between those two groups. Some of the coma patients with an arteriojugular oxygen difference below 3 vol % survived, although most low differences resulted in brain death within a few days.

Figure 3 shows the jugular pO₂ of both groups contrasted to that of the mixed venous blood (PvO₂) obtained from the right atrium at the same time. Although most of the jugular venous pO₂ values in the coma group were significantly high, they were lower than the pO₂ of systemic venous blood. In the brain death group the jugular venous pO₂ values were all higher than the mixed venous pO₂.
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TABLE 1
Summary of mean values (with standard errors) of pO₂, CO₂, pCO₂, and pH in the arterial and jugular blood in each group

<table>
<thead>
<tr>
<th>Mean Values</th>
<th>Coma Group (106)</th>
<th>Brain Death Group (46)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Femoral Artery</td>
<td>Jugular Vein</td>
</tr>
<tr>
<td>pO₂ (mm Hg)</td>
<td>220.4 ± 15.6</td>
<td>39.1 ± 0.1</td>
</tr>
<tr>
<td>CO₂ (vol %)</td>
<td>16.32 ± 0.28</td>
<td>11.91 ± 0.27</td>
</tr>
<tr>
<td>Cₐ–jO₂ (vol %)</td>
<td>4.41 ± 0.17</td>
<td>38.5 ± 0.5</td>
</tr>
<tr>
<td>pCO₂ (mm Hg)</td>
<td>30.6 ± 0.6</td>
<td>7.9 ± 0.4</td>
</tr>
<tr>
<td>pJv–CO₂ (mm Hg)</td>
<td>0.065 ± 0.003</td>
<td>0.051 ± 0.005</td>
</tr>
<tr>
<td>pH</td>
<td>7.519 ± 0.007</td>
<td>7.45 ± 0.007</td>
</tr>
</tbody>
</table>

The mean values and standard errors of pO₂, pCO₂, CO₂, and pH in both groups of arterial and jugular blood are summarized in Table 1. To a lesser degree arteriojugular venous difference in pCO₂, and pH were also reduced in both groups, more prominently in the brain death group.

Discussion

It is known that the pO₂ of the internal jugular venous blood increases strikingly in the state of brain death. In fact, it has been proposed that the measurement of cerebral arteriovenous oxygen difference could be a biological test to determine brain death.¹,²,⁴,⁶

The findings in our present study support this idea. Most of the arteriojugular oxygen content differences in the brain death group were below 3 vol %, with a mean value of 1.86 vol %. These values were in sharp contrast to the coma group, the mean of which was 4.46 vol %. Because of the considerable overlap in arteriojugular O₂ difference between the coma and brain death groups, it is doubtful that any level of arteriojugular oxygen difference could be relied upon as a definite dividing line.

In normal conditions, the pO₂ of the internal jugular venous blood is lower than...
that of the systemic mixed venous blood obtained from the right atrium. However, the internal jugular \( pO_2 \) was elevated in patients with severe head injury, and in brain death always showed higher values than the mixed venous \( pO_2 \). Thus, in determining brain death, a comparison of \( pO_2 \) is possibly more accurate and reliable as a metabolic parameter than reduced arteriojugular oxygen content difference.

Recently much attention has been directed to cerebral circulatory arrest in the state of brain death, as shown by the nonfilling phenomenon in cerebral angiography. In our present study, there was definitive evidence of total cerebral circulatory arrest in all the patients with brain death caused by head injury. Blood flow was only detected in the distribution of the external carotid system including the meningeal vessels. The cessation of sinus flow was also proved angiographically in these patients, as shown in Fig. 4. It may be inferred from these observations that the internal jugular blood in the state of brain death is mainly composed of the venous blood that returns from the dura, cranium, and extracranial soft tissues passing through the cavernous sinus and other venous channels. In such situations, the internal jugular blood is not representative of the mixed cerebral venous blood, although 97% of it normally returns from the brain tissue. This means that jugular hyperoxia in brain death cannot be attributed to the reduction of cerebral oxygen consumption. The arteriovenous method of obtaining \( CMRO_2 \) is not valid in brain death, when the jugular venous blood is no longer the mixed cerebral venous blood.

To clarify this problem, an additional study was performed on a patient in the brain death group. The changes in \( pO_2 \) in both the jugular and systemic mixed venous blood before and after temporary clamping of the external carotid artery on the same side as shown in Fig. 5. As soon as the external carotid artery was clamped, jugular venous \( pO_2 \), which had been at a high level, was lowered almost to that of the mixed venous \( pO_2 \). When the vessel was once again unclamped the jugular venous \( pO_2 \) increased to the previous level, while that of the mixed venous blood did not show an appreciable change throughout the procedure. This means that the majority of the blood in the superior jugular bulb is coming from the external carotid artery on the ipsilateral side.

Why is the internal jugular venous blood hyperoxygenated? Possible mechanisms are:
1. Since extracerebral tissues originally consume less oxygen than the cerebral tissue, the \( pO_2 \) of the external jugular venous blood is much higher than that of the internal jugular venous blood.
2. Because of the arrest of the internal carotid blood flow in brain death, most of the common carotid flow is "stolen" by the external carotid artery.
3. The fact that the external carotid regions are more "luxury perfused" results in the hyperoxia of the venous blood returned from the extracerebral tissues.
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References


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