Profound Hypothermia for Intracranial Surgery Using a Disposable Bubble Oxygenator*

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Surgeryons have long thought that better results might be achieved in the treatment of intracranial aneurysm if surgical repair could be performed without the danger of hemorrhage. Temporary clips placed on the proximal artery are helpful and have been widely employed but their application requires dissection of the cerebral arteries which may contribute to arterial spasm and thrombosis and result in cerebral infarction.15

An advantage of performing aneurysmorrhaphy with hemostasis achieved by temporary arrest of the circulation under deep hypothermia is that extensive dissection of the cerebral vessels is unnecessary. Intracranial operations have been performed in several clinics under these conditions and experience has shown that patients tolerate 30 or more minutes of cerebral ischemia if the temperature of the brain is reduced to about 15°C.5,7,9,11,13,18

In 1962 we reported our laboratory and clinical experiences with the surgery of aneurysms performed at low body temperatures attained by an extracorporeal circuit incorporating a rotating disc oxygenator.12 In many respects the results were gratifying but in others improvement was needed. The disc oxygenator required large amounts of blood for priming and considerable maintenance. Surgery often had to be delayed several days while donors for 4500 to 6000 cc. of fresh blood of the appropriate type were being located. By substituting a disposable bubble oxygenator which requires only saline and dextrose solutions as a prime, maintenance has been simplified and the blood bank relieved of a heavy burden.4 Possibly as a result of diluting the blood, time spent on cardiopulmonary bypass has been almost halved and troublesome venous bleeding from the craniotomy reduced. Laboratory and clinical experiences with this modified technique are described in the following account.

Laboratory Studies

Experiments were performed on 25 mongrel dogs weighing 22 to 45 kg., averaging 26 kg. Anesthesia was induced with thiopentonal sodium, the trachea intubated and methoxyflurane† then administered from a Heidbrink no. 8 ether vaporizer in the efferent line of a Bird Mark VIII respirator. Respirations were controlled at a rate of about 16 per minute except when cardiac asystole or ventricular fibrillation occurred. Anesthetic agent was not usually administered after the esophageal temperature fell below 25°C, unless the dog awakened during the rewarming period. The respiratory gas in all cases was 100 per cent oxygen.

The animals were connected to the extracorporeal circuit by venous cannulas inserted through the right innominate vein into the right atrium and through the femoral vein into the inferior vena cava. The blood drained by gravity into a Travenol disposable bubble oxygenator which was ventilated with 100 per cent oxygen at esophageal temperatures greater than 29°C and 95 per cent oxygen + 5 per cent carbon dioxide at temperatures lower than 29°C.

A totally occlusive pump propelled the blood from the oxygenator through a Brown-Harrison heat exchanger containing nylon rods and back to the animal by a femoral artery. In later experiments a second heat exchanger was added parallel with the first in order to obtain more efficient cooling and to reduce resistance to the flow of blood.

The extracorporeal circuit was primed with isotonic saline and 5 per cent dextrose in water. Low molecular weight dextran was not employed because of the suspicion that it might contribute to excessive bleeding from the craniotomy during

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the period of rewarming.\textsuperscript{6} Drake and Lewis have shown that low molecular weight dextran administered to hypothermic dogs significantly increases the volume of plasma whereas saline and plasma do not.\textsuperscript{5} The effect of dextran on the venous pressure of dogs was compared with that of plasma, whole blood, mannitol and isotonic saline (Table 1). The dextran solution resulted in a higher and more sustained elevation of venous pressure than any of the other solutions in the doses employed. Therefore, it was omitted from the priming medium of the extracorporeal circuit when the disposable bubble oxygenator was adapted, and 1000 cc. of isotonic saline and 500 cc. of 5 per cent dextrose in water substituted. More saline was used than might be suitable for a patient with cardiac disease, in order to minimize the risk of inducing cerebral edema by the administration of a solution, such as dextrose in water, which may become hypotonic when the solute is metabolized.

The pH and pCO\textsubscript{2} of arterial blood were determined at intervals during 15 of the experiments. Samples collected from the aorta in heparinized syringes were stored in ice water for 1–3 hours and then agitated at room temperature for several minutes prior to being introduced into two separate pH and pCO\textsubscript{2} meters.\textsuperscript{*} The water bath of one meter was maintained at 37\textdegree C, and that of the other at 20\textdegree C. Assuming an inverse relationship to temperature, the pH of arterial blood at body temperature, BT (taken as the arithmetic mean of the esophageal and rectal temperatures) was obtained by the formula:

\[
pH\textsubscript{BT} = \frac{pH\textsubscript{37} - pH\textsubscript{20}}{17} (37 - BT) + pH\textsubscript{37}
\]

The pCO\textsubscript{2} of arterial blood at body temperature was calculated from a similar formula assuming that the log pCO\textsubscript{2} varies directly with temperature.

\[
\text{log pCO}_2\textsubscript{BT} = \frac{\text{log pCO}_2\textsubscript{37} - \text{log pCO}_2\textsubscript{20}}{17} (37 - BT) + \text{log pCO}_2\textsubscript{37}
\]

\* Instrumentation Laboratory model 113.

### TABLE 1

**Effect of various solutions on venous pressure (intra-venous administration over 15 minutes to dogs)**

<table>
<thead>
<tr>
<th>No. of Animals</th>
<th>Mean Rise: Pressure mm./Saline</th>
<th>Median Duration in Minutes</th>
</tr>
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<tbody>
<tr>
<td>10 per cent low molecular weight dextran in saline 20 cc./kg.</td>
<td>4</td>
<td>77</td>
</tr>
<tr>
<td>Plasma 90 cc./kg.</td>
<td>3</td>
<td>41</td>
</tr>
<tr>
<td>Whole blood 90 cc./kg.</td>
<td>4</td>
<td>30</td>
</tr>
<tr>
<td>20 per cent solution mannitol 3 gm./kg.</td>
<td>3</td>
<td>34</td>
</tr>
<tr>
<td>Isotonic saline 90 cc./kg.</td>
<td>2</td>
<td>1</td>
</tr>
</tbody>
</table>

**Fig. 1.** A shift of the pH-pCO\textsubscript{2} line to the left is shown on 2 blood samples taken from a dog before (A) and after (B) a metabolic acidosis has developed. Base excess has fallen from 0 to −5. The pH and pCO\textsubscript{2} on each sample were measured at both 37\textdegree C, and 20\textdegree C, to construct the lines.

An alternative way of determining pCO\textsubscript{2} and also some other parameters of acid-base metabolism was employed. The pH and pCO\textsubscript{2} at 37\textdegree C, and 20\textdegree C, were plotted on the Sigggaard Andersen graph and connected by a straight line (Fig. 1).\textsuperscript{11} The pH at body temperature (which is quickly calculated) was located on the line and the value for pCO\textsubscript{2} read directly from the graph. A shift of the line to the left as determined from successive samples of blood during an experiment is evidence that a metabolic acidosis has developed. A value for base excess may also be read from the graph. Base excess is a measurement of the non-respiratory component of acid-base metabolism introduced by Astrup and his co-workers;\textsuperscript{2} it expresses in mEq/L the amount of strong base present in the blood. The normal value for base excess is arbitrarily fixed at zero while metabolic acidosis (a lack of base) is reported in negative values and metabolic alkalosis in positive values.\textsuperscript{2}

**Management of Perfusion.** Partial cardiopulmonary bypass was induced using about 100 cm. of venous drainage by gravity. The return arterial flow was balanced to keep the amount of blood in the extracorporeal circuit constant. Rates of flow initially ranged between 75 and 100 cc./kg./min. and fell somewhat during cooling but usually remained greater than 50 cc./kg./min. Low rates of flow or a fall of the arterial blood pressure much below 50 mm./Hg were corrected by adding 100–300 cc. of 5 per cent dextrose in water to the extracorporeal circuit.

The perfusion was usually continued until the electrocardiogram became iso-electric whether or