Occlusion of the Blood Supply to the Brain of the Goat

Protective Effect of Deep Hypothermia*

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Because the brain of the goat receives its entire blood supply from the carotid arteries with no contribution from the vertebral arteries, this animal is unusually suited for studies involving complete circulatory arrest to the brain. Our initial objective was to determine, if possible, the relation of the temperature of the brain to the length of time its blood supply could be occluded safely. As the work progressed, it became apparent that conclusions concerning the blood supply of different portions of the brain could be drawn from the rate of their cooling and warming. This information can be used to demonstrate the pathways for the development of collateral blood flow during progressive occlusions of the arterial inflow to the brain.

Blood destined for the head and upper extremities leaves the aorta via the single common brachiocephalic artery. This vessel, after giving off both subclavian arteries, bifurcates into two external carotid arteries. As they proceed through the goat's long neck, each external carotid artery enters its respective rete; these retia lie close together in the posterior pituitary fossa and are connected by numerous vessels which cross the midline and form a continuous plexiform vascular bed. From the rete of each side arises an intracranial internal carotid artery, which immediately gives off its posterior communicating branch. These two vessels unite to form the basilar artery. The caliber of the basilar artery diminishes as it progresses caudally (Fig. 1), and it has been demonstrated by injections of latex and neoprene that, under normal circumstances, blood flows caudally in the basilar artery.

The vertebral plexus supplies the vertebrae and muscles of the neck. From this plexus arise small perforating branches which

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Note continuation of basilar artery into anterior spinal artery.

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unite with the anterior spinal artery. The vertebral plexus is supplied by the two vertebral arteries (branches of the subclavian arteries) and the two occipital arteries (branches of the external carotid arteries proximal to the origin of the mandibular artery), as shown in Fig. 2. This occipital-vertebral anastomosis accounts for the ability of the goat to tolerate ligation of both external carotid arteries caudal to the origin of the occipital arteries, whereas simultaneous ligation of both vertebral and carotid arteries results in unconsciousness.8

Ligation of both carotid arteries cephalad to the point of origin of the occipital and mandibular branches cannot be tolerated either, as shown in the following experiment. The left external carotid artery was ligated cephalad to the point of origin of the occipital and mandibular branches. Electrocorticograms demonstrated bilateral decreased voltages for 2 min., but after 3 min. the tracings became normal. The animal made a complete recovery. One day later, the opposite carotid artery was ligated at the same point. The electrocorticogram became isoelectric within 20 sec. and the animal did not regain consciousness or breathe spontaneously. Since the carotid arteries were ligated cephalad to the occipital branches, blood could not return to the carotid from the vertebral plexus, and retrograde flow in the anterior spinal artery was not sufficient to maintain viability of the brain stem.

Previous work from this laboratory has shown that the brain can be cooled selectively by a carotid-to-carotid extracorporeal cooling shunt.1 Our first experiments were designed, therefore, to study the effect of deep cerebral hypothermia in animals whose carotid arteries had been ligated above the mandibular branches.

Method

Angora wethers weighing 25 to 40 kg. and aged 2 to 4 years were used. The animals were fasted for 3 to 4 days before the experiment and were premedicated with 0.6 mg. of atropine intramuscularly 1 hour before induction of anesthesia with Sodium Pentothal (thiopental sodium) 500 mg. and Anectine (succinyl choline) 100 mg. Anesthesia was

Fig. 2. Vertebral plexus of the goat. Note major vertebral-occipital arterial anastomosis as well as numerous carotid contributions to vertebral plexus.
maintained with nitrous oxide and oxygen inhalation and succinyl choline by intravenous drip. Tracheostomy was performed just above the sternum, and respiration was controlled with a Harvard respirator.

The left femoral artery was cannulated to record systemic pressure with a Statham strain gauge* while the vein was cannulated to provide an intravenous route for drugs.

Bilateral frontal and parietal burr holes were made for taking electrocorticograms and cerebral hemispherical temperature. In 5 goats the posterior fossa was exposed through an occipital craniectomy. Needle thermistors were placed in the brain stem at the level of the obex and in each cerebellar hemisphere. Mediastinal temperature was recorded from an esophageal lead.

In all animals the vessels of the neck were dissected bilaterally from below the thyroid artery to above the occipital and mandibular branches. Ligatures were placed around the vessel above and below the thyroid artery and above the occipital and mandibular branches.

In the animals that were to have total cerebral occlusion, the aorta and common brachiocephalic arteries were isolated via a left thoracotomy in the 3rd interspace. The right internal mammary artery was clipped at its origin. In the earlier experiments the left internal mammary artery was clipped in the 3rd interspace while in later experiments it was clipped at its origin from the subclavian artery.

The extracorporeal cooling system used (Fig. 3) consisted of an 1/8-in. tygon outflow catheter from the proximal carotid emptying into a disposable mayon reservoir. A Pemco micropump using latex tubing, with a 1/8-in. internal diameter and a 1/8-in. wall, delivered the blood to a Brown-Harrison heat exchanger. Tygon tubing of 1/8-in. inside diameter carried the blood from the heat exchanger to the microfilter-bubble trap. The latter consisted of the autoclavable portion of an Abbott† blood-transfusion filter, the distal tubing serving as the inflow arterial catheter inserted into the distal carotid. All connectors were made of highly polished stainless steel; one connector, placed just proximal to the bubble trap, was constructed with a right-angle attachment for continuous monitoring of perfusion pressure. Both inflow and outflow catheters were beveled and highly polished to prevent eddying currents. The entire extracorporeal system was assembled under sterile conditions and rinsed with 500 cc. of saline containing 50 mg. of heparin‡ before arterial catheterization.

After the surgical procedures, heparin (7 mg./kg.) was given intravenously, and 20 min. later the proximal carotid artery was cannulated. When the reservoir was completely filled systemic pressure usually dropped to 60 or 70 mm. Hg. The distal artery then was cannulated and perfusion was started. The inflow catheter into the distal artery was tied not only at its insertion but

* Statham Instruments, Inc., 254 Carpenter Road, Hato Rey, Puerto Rico, U.S.A.

† Abbott Laboratories, North Chicago, Illinois.

‡ Acknowledgment is made of the use of heparin through the kindness of Dr. Henry A. Strade, Organon Inc., 20 Main Street, West Orange, New Jersey.
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Fig. 4. Graph of experiment in extracorporeal carotid-to-carotid cooling to illustrate relation between systemic pressure and brain cooling. (See text.)

The scale in the ordinate represents in this and all other graphs: 1. Temperature in °C. 2. Ml./min. 3. Mm./Hg pressure. Thus the ordinate 50 represents 5°C, 50 ml./min., and 50 mm. Hg.

The blood was cooled in the extracorporeal shunt, the rates of cooling of each cerebral hemisphere demonstrate that the amount of mixing in the retia depends on the relationship of the pressure in the perfusing shunt to the systemic blood pressure in the opposite carotid artery. Thus, when a large difference between these two pressures exists, the artery carrying the highest pressure is the chief supply for both retia. This relationship is demonstrated in Fig. 4.

At the beginning of cooling, a right carotid-to-carotid cooling shunt was used (Fig. 4). Systemic hypotension resulted from the immediate loss of blood as the reservoir was filled. Uniform and rapid cooling of both hemispheres and the cerebellum occurred. At B a drop in perfusion pressure was accompanied by a rise in systemic pressure; this produced a uniform rise in temperature. Hexamethonium, 25 mg., administered intravenously at C once again produced a systemic hypotension followed by a uniform drop in the temperature. This entire sequence was repeated at D. At E the left external carotid artery was occluded cephalad to the point of origin of the mandibular and occipital arteries and the perfusion pump was stopped. An immediate rise in the temperature of the cerebellum occurred and rose at a more rapid rate than the temperature of the cerebral hemispheres. At F the clamps on the left external carotid artery were released. A rapid
rise in temperature in both hemispheres and acceleration of the rate of warming of the cerebellum followed. With warm blood flowing in one carotid artery and cold in the other carotid artery, fluctuations in the differential pressure between the two carotid arteries allow one or the other to perfuse both hemispheres.

We have observed repeatedly that when the cerebral hemispheres have been cooled by this technique and the arterial inflow has been occluded, there is a constant upward drift of the temperature of both cerebral hemispheres. This gradual warming of the cerebral hemispheres could result either from warm blood entering the brain because of an inadequate arterial occlusion or from a temperature gradient from surrounding tissues. To investigate this, the following experiment was performed.

When the brain was cooled to 5°C. (Fig. 5), the perfusion pump was stopped and the goat was decapitated. The temperature of each cerebral hemisphere was then recorded every 2 min. for 1 hr. The room temperature was 27°C. The slope of warming in the cerebral hemispheres was $\frac{1}{4}$ of a degree per min. This obviously was caused by conduction of heat from surrounding tissues. This slope was identical to that obtained in experiments in which arterial inflow was occluded completely, and it is therefore unlikely that the upward drift of the temperature of the cerebral hemispheres in these experiments was produced by the entrance of warm blood into the brain. Consequently, this experimental preparation can be used to test the completeness of occlusion of the arterial blood supply to the brain or its component parts, as illustrated in the next experiment.

A right carotid-to-carotid cooling shunt again was used. The outflow catheter supplying the reservoir was placed just cephalad to the origin of the thyroid artery and the inflow catheter from the reservoir was placed cephalad to the mandibular artery. The external carotid artery was tied securely around the inflow catheter, so that the blood from the occipital artery could not enter the cerebral circulation. With the temperature of the brain at 5°C. (Fig. 6) at $A$, the left external carotid was clamped just cephalad to the thyroid artery and the left occipital artery was clamped. The perfusion pump was then
stopped. The cerebral hemispheres warmed faster than they would have done from conduction alone. The clamps were removed and the brain was recooled. At B the clamps were replaced and an additional clamp was placed on the left carotid caudad to the thyroid artery. When the pump was stopped, the rate of warming was the same. After recooling, a third clamp was placed on the right external carotid caudad to the thyroid artery (C). The rate of warming was unchanged and the brain was recooled again. At D, the left external carotid was clamped cephalad to the mandibular artery and the perfusion pump was stopped once again. The resultant warming curve was the same as that obtained in the isolated head.

Even though the occipital arteries were occluded effectively at A, B, and C, the rate of warming of the cerebral hemispheres suggests communication between the vertebral plexus and the external carotid system. It was not until the left external carotid was clamped cephalad to the mandibular artery that the rate of warming was identical with that seen in the isolated head, indicating that collateral supply from the vertebral plexus had been eliminated.

In these experiments, when occlusion of arterial inflow to the brain was attempted, the temperature of the brain stem and cerebellum rose more rapidly during occlusion than did the temperature of the cerebral hemispheres. This was true even when the rate of warming of the cerebral hemispheres indicated a total arterial occlusion. Occlusion of both carotid arteries below the thyroid artery and above the mandibular artery, accompanied by clamping of all intervening branches, effectively stops the flow of blood into the retia. However, because the brain stem and cerebellum warm more rapidly during this type of occlusion, we hypothesized that the anterior spinal artery, fed by the vertebral plexus, allowed a retrograde flow of blood into the basilar artery.

To determine whether arterial inflow to the entire brain could be arrested by occluding the common brachiocephalic artery, we occluded this vessel after cooling the brain. The change in temperature of the cerebral hemispheres was faster than would be expected from conduction alone (Fig. 7). The brain then was recooled. In addition to the brachiocephalic artery, both internal mammary arteries were ligated cephalad to their anastomosis with each 1st intercostal artery. The resulting drift of temperature upon occlusion of these additional vessels (Fig. 7) indicated a complete arterial occlusion to the cerebral hemispheres, the brain stem and cerebellum.

![Fig. 6. Cooling experiment to illustrate warming curves under various conditions of occlusions of arterial inflow. (See text.)](image-url)
The anatomic relationships of the intercostal arteries to the internal mammary and the common brachiocephalic system may be seen in Fig. 8. Presumably, occlusion of the blood supply to the entire brain, including the brain stem and cerebellum, could be obtained by clipping both internal mammary arteries as well as the common brachiocephalic artery and, in addition, clamping one external carotid artery distal to the mandibular artery while perfusing through the other carotid system. In this situation, when the outflow catheter into the reservoir is left open, no blood flows into the reservoir, thus indicating that occlusion is complete. Furthermore, the rate of warming of the cerebral hemispheres and the brain stem and cerebellum is identical and consistent with the expected drift from conduction alone.

These experiments developed for us two preparations with which we could study the relationship between the safe period of vascular occlusion and the temperature of the brain. In the first preparation, which we have termed a “neck occlusion,” the blood supply to the cerebral hemispheres is effectively stopped but the brain stem and cerebellum are still being supplied partially, presumably via a retrograde flow of blood in the anterior spinal artery. In the second preparation, which we have termed “chest occlusion,” there is an effective occlusion of the blood flow to the entire brain. Therefore, a series of experiments was performed to determine safe times of occlusion. The temperature of the brain was lowered selectively to 5–8°C by a right carotid-to-carotid shunt maintaining
the body-core temperature above 30°C. One of the two types of occlusion was made for varying periods of time. At the termination of the desired occlusion the clamps on the left external carotid artery were removed. After the electrocorticographic tracings had resumed their initial appearance, the catheters were removed from the right carotid artery, which was then ligated permanently. Polybrine* was given intravenously (1 mg. /mg. heparin). Postoperatively, the animal was kept propped on its knees. The tracheostomy was maintained 3 to 4 days. The animals that recovered were turned into the field 1 week after operation.

Since heparinization is necessary for extracorporeal cooling and since heparin prolongs the safe time of occlusion in dogs under normothermic conditions, 4, 5 the tolerance of goats to total (chest occlusion) cerebral vascular occlusion while heparinized at normal temperatures was also studied for purposes of control.

Results

There were 52 successful experiments, divided into the following groups: (1) occlusion of the chest at normal temperature, (2) occlusion of the neck under hypothermia, and (3) occlusion of the chest under hypothermia.

Chest Occlusion at Normal Temperature. The common brachiocephalic artery and the internal mammary arteries were occluded in 15 goats at normal temperatures after heparinization. The vessels of the neck also were dissected and clamped simultaneously, exactly as if they were to be perfused. The preparation was identical to the hypothermic occlusion of the chest except that perfusion was not done.

Clamping the common brachiocephalic artery produced a 300 to 350 mm. Hg rise in systemic blood pressure, which then subsided gradually. The electrocorticogram became isoelectric after 20 sec. (Fig. 9), but normal tracings appeared shortly after removal of the clamps.

* Polybrine-Poly-(1,5-dimethyl-1,5-diazaundecamethylene methobromide).

The occlusion was maintained for 15, 10, 7.5 and 5 min. in different animals. In the experiments in which the times of occlusion were 15 and 10 min., the internal mammary arteries on the right were clipped at the origin; but on the left they were clipped in the 3rd intercostal space. In the later experiments (7.5- and 5 min.- occlusions), both the internal mammary vessels were occluded at their origin.

The results are seen in Table 1. Only goats that were normal in every respect were considered to have recovered. In the group of goats in which the time of occlusion was 15 min., 1 of 4 animals survived. Two out of 5 survived in the group of 10-min. occlusion. Neither of the 2 goats in the group of 7.5-min. occlusion survived whereas 4 out of 5 animals in the group of 5-min. occlusion survived. All the deaths were caused by spontaneous respiratory arrest and occurred when the respirator was removed or on the evening of operation from spontaneous respiratory arrest.

Neck Occlusion under Hypothermia. “Neck occlusions” were made in 17 animals when the brain temperature was 5°C. to 8°C. One animal was occluded for 1 hr., 1 for 45 min., 8 for 30 min., 3 for 20 min., and 4 for 15 min.
TABLE 1

<table>
<thead>
<tr>
<th>Occlusion Time (min.)</th>
<th>Chest Occlusions</th>
<th>Neck Occlusions</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. of Animals</td>
<td>Survivors</td>
</tr>
<tr>
<td>Normothermic</td>
<td></td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
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<td>Hypothermic</td>
<td></td>
<td></td>
</tr>
<tr>
<td>60</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>45</td>
<td>10</td>
<td>1</td>
</tr>
<tr>
<td>30</td>
<td>5</td>
<td>4</td>
</tr>
<tr>
<td>20</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>15</td>
<td>2</td>
<td>4</td>
</tr>
</tbody>
</table>

One of the animals having a 20-min. occlusion, 6 of the 8 animals having 30-min. occlusions, and both the animals having 45-min. and 1-hr. occlusions died. In all of the animals that died the clinical features and pathologic findings were strikingly similar. All of them awakened and breathed spontaneously but none of them ever stood. Some of the animals were able to stay on their knees for a short while when propped up but their heads would sway from side to side with very little control. They all had lateral nystagmus and some had vertical nystagmus. Their pupillary reactions were normal. Power as well as sensation appeared normal. Since goats are unable to tolerate lying on their left side, it was not uncommon to find them dead in this position. The goats that recovered, on the other hand, displayed no such ataxia and were capable of righting themselves a few hours after cessation of anesthesia.

Histologic examinations of the brains of the goats that died showed focal lesions in the brain stem (Fig. 10) and loss of Purkinje cells (Fig. 11) in the cerebellum.

All of the goats with 15-min. occlusions survived. Two of the 3 with 20-min. occlusions and 2 of the 8 with 30-min. occlusions survived. In the 2 animals that survived

![Fig. 10. Photomicrograph of brain stem of goat that failed to survive a 30-min. occlusion of the neck under profound regional hypothermia. Focal necrosis is evident. Hematoxylin and eosin, ×34.](image-url)
after 30-min. occlusions, spontaneous hypertension occurred during the period of occlusion when the cerebral hemispheres were cold (Fig. 12). We have shown previously that profound systemic hypotension occurs when the temperature of the brain stem is less than $20^\circ$C.\(^4\) We believe, therefore, that in these 2 animals that survived 30 min. of occlusion, the blood flow to the brain stem was not only sufficient to warm it but also was sufficient to maintain its functional integrity.

*Chest Occlusions under Hypothermia.* Of the 20 goats in this group, occlusions were maintained for 1 hr. in 3 goats, 45 min. in 10, 30 min. in 5, and 20 min. in 2. All 3 of the animals in which 1-hr. occlusions were done and all but 1 of the 10 animals in which 45-min. occlusions were done died. Both of the animals having 20-min. occlusions and 4 of the 5 animals having 30-min. occlusions recovered and appeared normal in all respects (Table 1). Two of the 3 animals in which 1-hr. occlusions were done failed to breathe spontaneously and suffered a steady decline in blood pressure despite pressor agents and mechanical maintenance of ventilation. The remaining goat in which the occlusion time was 1 hr. was able to breathe spontaneously, but respiratory arrest occurred 12 hrs. postoperatively.

The 9 goats undergoing 45-min. occlusions and the 1 goat with a 30-min. occlusion which failed to survive breathed spontaneously and could remain on their knees with their head erect but could not stand. They died from 12 to 36 hrs. postoperatively from respiratory arrest. This is in marked contrast to the "neck occlusions" in which swaying of the head, truncal ataxia, and nystagmus were predominant features.

Histologic sections of the brains of goats dying after occlusions of the chest showed that the most marked damage was in the hippocampus with sparing of the brain stem,
Profound regional brain hypothermia in a goat with a 30-min. occlusion of the neck that survived. Note spontaneous and sudden rise in systemic blood pressure during period of occlusion.

Graph illustrating changes in temperature during a 1-hr. occlusion of the chest. Note superimposition of temperature of the stem upon that of the cerebral hemispheres.
a finding that is to be expected with total cerebral occlusion. In the 2 goats of this
group in which brain-stem temperature was monitored, the rate of rise of the temperature of
the stem after occlusion was identical with that of the cerebral hemispheres, indicating a
total occlusion of arterial inflow (Fig. 13).

Electrocorticograms showed diminished activity on cooling to 28°C., with some activity persisting as low as 21°C. After vascu-
ar occlusion the return of the electrocorticograms to normal did not parallel the return of
temperature but was related more closely to the duration of the occlusion. The longer the occlusions, the longer the time required for
a return to normal electrical activity. An example is shown in Fig. 14. The earliest ac-
tivity recorded usually was a series of high-voltage bursts followed by a long period of
electrical silence.

The hypotension reported previously from this laboratory, which occurred when the
temperature of the brain reached 24°C. to 20°C.,4 was observed in all goats but was al-
tered to a varying degree upon clamping of the common brachiocephalic artery. The usual response was a doubling of the systemic
pressure followed by a gradual decline over the period of the occlusion.

**Discussion**

In the hypothermic animals whose left int-
ernal mammary arteries were clamped in the 3rd interspace, it was possible to measure
the amount of blood draining from the com-
mon brachiocephalic system into the reservoir
during the period of occlusion. This never amounted to more than a few cc. How-
ever, it is entirely possible that under the hypertensive conditions consequent to clamping of the common brachiocephalic
artery at normal temperature, some blood may have entered this system via the aortic
internal mammary anastomosis in the 1st and 2nd interspace on the left. With the final
common path of cerebral blood flow to the retia clamped in the neck (external carotid above the mandibular), the only route this
blood could follow to the brain was by way of the anterior spinal artery. This may ex-
plain the 3 normothermic animals that survived after 10- and 15-min. occlusions. It
would seem that the anticoagulated normothermic safe time of occlusion in the goat is
not greater than 5 min. This is a surprising finding in view of the ability of anticoagu-
lated normothermic dogs to withstand total circulatory arrest for as long as 10 min. That heparin prolongs the safe period of total
cerebral ischemia has been demonstrated previously.5,7 During periods of circulatory
arrest, the concentration of lactic acid and of inorganic phosphate in tissue increases, while
that of phosphocreatine decreases, and the pH is lowered.10 Under such conditions, hypercoagulability of the blood develops and
persists 1 to 2 min. after re-establishment of the circulation.5 Multiple emboli form and
may impede the circulation in the capillaries. Experimental circulatory arrest in dogs re-
sulted in only 3 survivors out of 13 when the
period of arrest was 5 min. and 1 survivor with damage out of 12 animals when the period of arrest was 10 min. When Varidase* activated by human plasma was given, 6 of 7 dogs survived after 15 min. of circulatory arrest. A species difference in metabolic substrates between the goat and the dog may explain the discrepancy in safe times of occlusion.

When occlusions are made in the neck, profound regional hypothermia appears to increase the safe time of occlusion from 5 min. to 15 min.; longer occlusions are unsafe. Ischemic lesions appear in the brain stem, while the more common sites for ischemic damage, such as the hippocampus, remain unaffected. Fig. 4 shows the rise in temperature of brain stem and cerebellum during occlusion. It is postulated that enough blood enters the brain stem via the anterior spinal-basilar system to warm these structures, which increases their metabolic demand beyond that which can be provided by this flow of blood. If the anterior spinal-basilar retrograde flow was adequate to allow preservation of the cells, then it presumably would also allow for their function. Since the profound hypotension under regional hypothermia is entirely a result of a nonfunctioning stem, one would expect a return of normal systemic pressure under such circumstances. This actually occurred in 2 goats with 90-min. occlusions of the neck and both animals survived. The assumption that this retrograde blood flow was sufficient to warm the stem and also to maintain its viability and function at the increased temperature appears tenable.

Profound regional hypothermia prolongs the safe time of occlusion from 5 to 30 min. when occlusion is made in the chest. Under these circumstances the stem remains at the same temperature as the cerebral hemispheres (Fig. 13). If selective warming of the stem in the animals undergoing occlusions of the neck was responsible for the shortened safe time of occlusion of 15 min., one might expect a difference in the rate of warming of the cerebral hemispheres during the two types of vascular occlusion. This was measured and was found to be 3.4°C. in 15 min. for occlusions of the neck and 2.5°C. for occlusions of the chest. After 30 min. of occlusion, the temperature after occlusion of the neck had drifted 7.4°C., whereas the drift was only 5.4°C. after occlusion of the chest (Table 2). The drift of the temperature of the cerebrum after the occlusions of the chest was identical with the drift in the isolated goat’s head.

Since “total cerebral occlusions” in current clinical use do not interrupt the flow of blood to the brain stem by way of the anterior spinal artery, it is suggested that safe times of occlusion for humans undergoing regional hypothermia cannot be based on temperature of the cerebral hemispheres alone but will depend on the rate of warming of the brain stem. In the goat cerebral blood flow can be occluded completely by clamping the common brachiocephalic artery at its junction with the aorta after occluding both internal mammary arteries at their origin.

* Varidase—Streptokinase-streptodornase.

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**TABLE 2**

<table>
<thead>
<tr>
<th>Time</th>
<th>Neck</th>
<th>Chest</th>
<th>Difference</th>
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</thead>
<tbody>
<tr>
<td>15 min.</td>
<td>3.40 ± 0.313*</td>
<td>2.56 ± 0.247</td>
<td>0.98$p+&lt;.05$</td>
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<td></td>
<td>n=20</td>
<td>n=17</td>
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<tr>
<td>30 min.</td>
<td>7.40 ± 0.642</td>
<td>5.44 ± 0.599</td>
<td>1.96$p&lt;.02$</td>
</tr>
<tr>
<td></td>
<td>n=10</td>
<td>n=18</td>
<td></td>
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</tbody>
</table>

* Standard error of the mean.
† $p$ value.

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Conclusions

1. The degree of mixing of blood in the carotid retia depends on the relationship of the pressure in each carotid artery.
2. One external carotid artery can maintain an adequate blood supply to the brain of the goat.
3. Ligation of both external carotid arteries cephalad to the mandibular branches is not compatible with life.
4. Complete bilateral occlusion of the external carotid system reverses the normal flow in the basilar anterior spinal artery from a caudad to cephalad direction.
5. The rate of rise in temperature of the brain can be utilized to determine completeness of vascular occlusion when this rate is compared with that of the decapitated and previously cooled brain or head.
6. Occlusion of both external carotid arteries cephalad to the mandibular and occipital branches and below the thyroid artery, and occlusion of all intervening branches, totally occludes the blood supply to the cerebral hemispheres.
7. For total cerebral occlusion, including the supply to the brain stem and cerebellum, it also is necessary to occlude both the internal mammary and the common brachiocephalic arteries.
8. The safe time of occlusion in anticoagulated normothermic goats is not more than 5 min.
9. Profound regional hypothermia extends the safe period of occlusion in the goat to 30 min. when total (chest) arterial occlusions are made.
10. The “safe period of occlusion” under profound regional hypothermia in the goat is 15 min. when the occlusions are made in the neck. In this type of arterial occlusion, similar to that now used clinically, there is a retrograde flow in the anterior spinal artery.

References

9. Smith, M. C., and Adams, J. E. Prolonged anesthesia in the goat. (Unpublished)