Variations in the Anatomy of the Rabbit Cervical Carotid Artery

Jai S. Lee, MD; Mark G. Hamilton, MDCM, FRCS(C); Joseph M. Zabramski, MD

Background and Purpose A model for cerebral ischemia that requires injection of emboli into the internal carotid artery of the rabbit is commonly used. However, in our experience we have found the anatomy of the cervical carotid to be highly variable. If not appreciated, this may result in unexpectedly high variability in the severity of ischemic injury. We undertook this experimental protocol to determine whether it was possible to characterize the anatomy of the rabbit cervical carotid artery.

Methods We examined and recorded the architecture of the cervical carotid arteries of 105 consecutive rabbits involved in experimental protocols to evaluate the role of tissue-type plasminogen activator during embolic stroke.

Results Two basic patterns of origin of the internal carotid artery were identified: lateral origin, classified as type I, and dorsomedial origin, classified as type II. In addition, there were three subsequent variations in the origin and morphology of the occipital artery in relation to the internal carotid artery: origin from the external carotid artery (subtype A); origin proximal on the internal carotid artery (subtype B); and origin distal on the internal carotid artery (subtype C).

Conclusions The classification of the anatomy of the cervical carotid artery of the rabbit into these easily recognized types will assist those attempting to use this embolization model. Failure to recognize the origin of the occipital artery from the internal carotid artery can result in the misdirection of embolic material into the occipital artery and significantly reduce the effectiveness of this stroke model. (Stroke. 1994; 25:501-503.)

Key Words • animal models • carotid arteries • embolism • rabbits

Cerebral embolism remains a major component of the problem of patients with cerebrovascular disease. To investigate the pathophysiology and evaluate new management strategies for human stroke, models of embolic stroke that use emboli injected directly into the internal carotid artery of the rabbit are commonly employed.1-6 However, in our initial experience with such a model we appreciated that the anatomy of the cervical carotid artery in the rabbit was highly variable, with the occipital artery frequently originating from the internal carotid artery. If these anatomic variations are not recognized, they may create an unexpectedly high variability in the incidence and severity of ischemic injury. We undertook this protocol to determine whether it was possible to characterize the anatomy of the rabbit cervical carotid artery.

Materials and Methods

This study was conducted in accordance with all published guidelines of the US Department of Health and Human Services and the National Institutes of Health. The protocols were approved by the Animal Welfare Committee of the Barrow Neurological Institute. This facility is approved by the American Association of Accreditation of Laboratory Animal Care.

New Zealand White rabbits (Western Oregon Rabbit Company, Plymouth, Ore), all weighing approximately 3 kg, were being used in experimental protocols examining the role of tissue-type plasminogen activator in the treatment of embolic stroke. Both male and female rabbits were used. We examined and recorded the architecture of the left cervical carotid artery in 105 consecutive rabbits.

The details of our rabbit model of embolic stroke have been described.1,3 The rabbits were anesthetized with an intramuscular cocktail of ketamine (10 mL, 100 mg/mL), acepromazine (2 mL, 10 mg/mL), and xylazine (1.5 mL, 100 mg/mL) (KAX) in a dose of 0.6 mL/kg. The rabbits were then placed in the supine position, and a tracheostomy was performed. Ventilation was maintained with a volume-cycled ventilator with a tidal volume of 20 mL/kg and an FiO2 of 21%. The ventilator rate was adjusted to maintain the Paco2 between 35 and 45 mm Hg. The rabbits were paralyzed with 0.5 mg/kg tubocurarine followed by an infusion of 0.75 mg/h. Anesthesia was maintained with hourly intramuscular injections of the KAX cocktail (0.6 mL/kg). A rectal probe was placed for monitoring core temperature; normothermia was maintained by adjusting a heating blanket.

The left common, internal, and external carotid arteries were exposed and isolated using standard microsurgical techniques. The internal carotid artery was exposed up to the point of entry into the skull base. The anatomy of the cervical carotid artery was then recorded. At the end of the treatment protocol, the rabbits were killed with an intravenous injection of KAX cocktail.

Results

One hundred five left cervical carotid arteries were examined. Two basic patterns of origin of the internal carotid artery were identified: lateral origin, which we classified as type I (Fig 1), and dorsomedial origin, which we classified as type II (Fig 2). The internal carotid artery travels behind the external carotid artery in the type II cervical carotid arteries to head laterally and deep toward the skull base. In addition, there were

Received April 21, 1993; final revision received August 30, 1993; accepted October 5, 1993.

From the Division of Neurological Surgery, Barrow Neurological Institute, Phoenix, Ariz.

Reprint requests to Joseph M. Zabramski, MD, c/o Editorial Office, Barrow Neurological Institute, 350 W Thomas Rd, Phoenix, AZ 85013.
Three subtypes are identified based on the location of the occipital artery (OA): subtype A is with the origin of the OA from the external carotid artery (ECA); subtype B has the OA originating from the proximal ICA; and subtype C is with the OA originating distally on the ICA. The relative sizes of the vessels have been drawn to scale (see text).

The type IA variation was the most common, occurring in 48.6% of the animals (Table). The occipital artery originated from the internal common artery in 25.7% of all animals: in 29.2% of type I animals and 18.2% of type II animals. A distal origin of the occipital artery from the internal carotid artery occurred in 13.3% of all animals. In this latter situation, the occipital artery may originate very close to the point of entrance of the internal carotid artery into the skull base.

The cervical carotid artery and its branches were measured in 20 consecutive rabbits. The mean ± SD size of the common carotid artery was 2.00 ± 0.31 mm, the main trunk of the external carotid artery 1.90 ± 0.36 mm, the internal carotid artery 1.13 ± 0.25 mm, and the occipital artery 1.00 ± 0.25 mm.

We prefer to visualize the passage of the autologous arterial thrombus embolic material directly into the internal carotid artery. This can be quite difficult in the type II vessels because the internal carotid artery travels behind the external carotid artery. However, the external carotid artery can usually be manipulated to permit adequate visualization of the internal carotid artery origin.

More important, however, is the location of the occipital artery origin. We found that the occipital artery originated from the internal carotid artery 25.7% of the time, with a distal origin occurring in 13.3% of animals. Scremin et al provided the only other description of the rabbit cervical carotid anatomy. Based on only nine animals, they recognized that the occipital artery originated from the internal carotid artery in approximately one third of the animals. However, they did not provide any details concerning the location of the vessel origin. This information is crucial when using the rabbit stroke model.

Positive identification of the internal carotid and occipital arteries can be difficult, particularly when the vessels rise from a common trunk (subtype B and subtype C). The similar size of the occipital and internal carotid arteries in the rabbit further adds to the confusion. The only sure method of identifying the internal carotid artery is to follow it to the skull base, where it penetrates the foramen lacerum. Alternatively, if the occipital artery is followed distally it is found to course posteriorly, giving off branches.

When the occipital artery rises from the internal carotid artery, it must be occluded from the internal carotid artery during embolization procedures. If this is not accomplished, the emboli can travel into the occipital artery and lodge in the extracranial rather than intracranial vessels. Embolization of extracranial branches leads to a decrease in the incidence and severity of the ischemic insult to the brain. Although not formally assessed, our experimental experience suggests that the variability of stroke in the rabbit emboli model is increased through failure to deal with the occipital artery. In preliminary studies done at our institution.

### Table: Frequency of Cervical Carotid Artery Variations in 105 Rabbits

<table>
<thead>
<tr>
<th>Anatomic Variation*</th>
<th>No.</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>IA</td>
<td>51</td>
<td>48.6</td>
</tr>
<tr>
<td>IB</td>
<td>10</td>
<td>9.5</td>
</tr>
<tr>
<td>IC</td>
<td>11</td>
<td>10.5</td>
</tr>
<tr>
<td>IIA</td>
<td>27</td>
<td>25.7</td>
</tr>
<tr>
<td>IIB</td>
<td>3</td>
<td>2.9</td>
</tr>
<tr>
<td>IIC</td>
<td>3</td>
<td>2.9</td>
</tr>
</tbody>
</table>

*See text.
before recognizing the variability of the occipital artery origin, the incidence of embolic infarction was unac-
ceptably low (approximately 50%) in control animals
(J.M.Z., unpublished data, 1989). Failure to identify
intracranial emboli in many of these early animals
stimulated our interest in anatomy of the cervical
vessels. After we began following the internal carotid
artery to the skull base and occluding the occipital
artery when it rose from the internal carotid, the
incidence of ischemic injury in control animals in-
creased to 85%. We prefer to use temporary vessel
clips to exclude the occipital artery from the experimen-
tal field. When this is accomplished, the embolic mate-
rial will be consistently directed into the cerebral circu-
lation, thereby minimizing experimental variability.

References
1. Rigamonti D, Uede T, Johnson PC, Bojanowski WM, Awad IA,
   Michael KT, Carter LP, Spetzler RF. A new model of cerebral
   embolic ischemia using autologous arterial thrombus. Barrow Neu-
2. Benes V, Zabramski JM, Boston M, Puca A, Spetzler RF. Effect of
   intra-arterial antifibrinolytic agents on autologous arterial emboli
3. Carter LP, Guthkelch AN, Orocio J, Temeltas O. Influence of
   tissue plasminogen activator and heparin on cerebral ischemia in a
4. Agnelli G, Buchanan MR, Fernandez F, Boneu B, Van Ryan J,
   Hinch J, Collen D. A comparison of the thrombolytic and hemor-
   rhagic effects of tissue-type plasminogen activator and strep-
5. Lyden PD, Zivin JA, Clark WA, Madden K, Sasse KC, Mazzarella
   VA, Terry RD, Presa GA. Tissue plasminogen activator-mediated
   thrombolysis of cerebral emboli and its effect on hemorrhagic
   Hemerway CC, Johnston P. Tissue plasminogen activator:
   reduction of neurologic damage after experimental embolic stroke.
7. Scremin OU, Sonnenschein RR, Rubinstein EH. Cerebrovascular
   anatomy and blood flow measurements in the rabbit. J Cereb Blood

Editorial Comment

It is a well-established principle that meticulous
attention to detail is necessary for the success of
biologic experiments. The accompanying article by
Lee et al provides an excellent illustration of this
principle in stroke research. These authors found that
there is considerable anatomic variation in the carotid
artery in the neck of the rabbit. Injection of emboli into
the carotid artery in the rabbit is used frequently in the
studies of embolic stroke. When the occipital artery
originates from the internal carotid artery, some of the
emboli are misdirected to extracranial structures with

Hermes A. Kontos, MD, Associate Editor
for Basic Science
Department of Medicine
Medical College of Virginia
Richmond, Va
Variations in the anatomy of the rabbit cervical carotid artery.
J S Lee, M G Hamilton and J M Zabramski

Stroke. 1994;25:501-503
doi: 10.1161/01.STR.25.2.501

Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1994 American Heart Association, Inc. All rights reserved.
Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/25/2/501

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org//subscriptions/