Survival of Rabbits After Prolonged Cerebral Ischemia

RONALD J. KOLATA, DVM

SUMMARY Cerebral ischemia was produced by a combination of vascular occlusion and mild systemic hypotension in 2 groups of rabbits. Arterial blood pressure, arterial pH, arterial blood gases, blood glucose and PCV were monitored and recorded before, during and for 3 hours after reperfusion. Return of EEG activity, vasomotor control, spontaneous ventilation and corneal reflex were also recorded. At 4, 8, 12, 24 and 48 hours after reperfusion, the rabbits' neurologic status was assessed according to an arbitrary scale based on motor function. The 2 groups differed in return of reflexes and motor function. Eighty percent of the rabbits ischemic for 20 minutes and 75% of the rabbits ischemic for 30 minutes survived. The graduated response of motor function to cerebral ischemia is attributed to the ventilatory and circulatory support given the rabbits for the first 3 hours after reperfusion. The graduated response of motor function to ischemia supports the suggestion that motor function can be used as an index of neurologic damage.

A VARIETY OF METHODS to control the cerebral circulation of rabbits has been used to study the effects of cerebral ischemia (CI) in these animals. The methods vary in their complexity. Simple clamping of the aorta just distal to the coronary sinus can cause CI. If a left ventricular outflow reservoir is also used, fatal cardiac dilatation is prevented and reversible cerebral ischemia is achieved. Most other methods of achieving CI involve arterial ligation and one or more other maneuvers. One method involves permanent basilar artery ligation, temporary bilateral carotid occlusion, and inflation of a cervical pressure cuff to cause CI. A simplified method employs bilateral temporary carotid artery occlusion and drug-induced profound systemic hypotension. A similar and more simplified method uses a cervical pressure cuff inflated to 1.5 atm pressure to cause CI. A method which does not employ arterial occlusion but achieves CI by using drug-induced profound hypotension, tilting the rabbit head up and ventilating it with a mixture of 96% N₂ and 4% O₂ has been described. These methods have been classified and commented on. Some have to be criticized because they require extensive or traumatic surgery to gain control of the cerebral circulation. Some of them, also, cause hypoxemia and ischemia of organs in addition to the brain, which could result in artifactual responses during recovery.

The method described herein overcomes many of these criticisms as it requires minor surgical intervention, allows venous return from the brain, maintains adequate blood oxygenation, and preserves adequate systemic blood pressure.

Methods

Surgical Preparation

Each rabbit is given 0.2 mg of atropine kg/body weight. Anesthesia is induced with a gas mixture of halothane and oxygen. After intubation, anesthesia is maintained with the same mixture. The rabbit is prepared and draped for aseptic surgery. A 3 cm incision is made in the lateral surface of the neck beginning at the caudal edge of the 1st cervical vertebra. The subcutaneous tissues are incised, and the splenius muscle is identified and reflected dorsally to expose the intertransversarius cervicus dorsalis muscle. The intertransversarius cervicus dorsalis and the intertransversarius intermedius muscles are separated to expose the vertebral artery as it passes out of the transverse foramen of the second cervical vertebra. The artery is identified and ligated...
with tantalum clips. Next the carotid artery is isolated from the nerve trunks that are adjacent to it within the carotid sheath, and a ligature is placed loosely around it so that it can be immobilized for clamping. Two-thirds of a 12-inch length of umbilical tape is placed into a space created between the cervical vertebrae dorsally and the trachea ventrally. The remaining 1/3 is left protruding from the incision. The wound is covered with a sterile gauze pad, the rabbit rolled over, and the surgical procedure repeated on the previously unoperated side. Once the vertebral arteries are ligated and the carotid arteries isolated, the previously buried length of umbilical tape is brought out of the skin incision.

After control measurements are made, anesthesia is stopped, and trimethaphan camphor sulfonate* is infused intravenously until peak systolic pressure is < 80 mm Hg. Cerebral ischemia is immediately induced by clamping the common carotid arteries with non-crushing vascular clamps and by firmly tightening the umbilical tape ligature by tying it around the perivertebral muscles at the level of the third cervical vertebra, thereby occluding muscular branches anastomosing with the occipital branch of the vertebral artery. During CI, the peak systolic pressure is kept between 70–80 mm Hg by infusion of trimetaphan or norepinephrine as necessary.

Method of Procedure

To investigate the efficacy of this method of producing reversible cerebral ischemia, New Zealand white rabbits of either sex and weighing between 2–2.5 kg were used. Twenty-four rabbits were surgically prepared as described. Five were used to determine the completeness of the cerebral ischemia achieved by this method. Five were allowed to recover from surgery without undergoing a period of ischemia to assess whether or not grossly detectable neurological signs were caused by the surgical procedure. Fourteen rabbits had selected physiological and neurological parameters monitored before, during, and after CI to determine the degree of neurological impairment caused by prolonged ischemia.

Physiological Measurements

The skin overlying the cranium was incised on the dorsal midline and reflected laterally.

Stainless steel screws, to serve as dural electrodes, were threaded into the skull. A screw was positioned 1 cm to either side of the midline of the skull at the frontoparietal suture. A third screw, to serve as a reference electrode, was threaded into the sagittal crest. The skin incision was sutured closed leaving the screws protruding.

A catheter was inserted into the femoral artery to enable blood pressure recording and for obtaining blood samples. Catheters were also inserted percutaneously into both saphenous veins to facilitate injection of drugs. Five hundred units of heparin per kg were administered. The rabbits were placed in sternal recumbency, and needle electrodes were placed into contralateral sides of the chest to allow recording of the electrocardiogram.

The endotracheal tube was connected to a Harvard Model 601 ventilator. Anesthesia was maintained by connecting a Drager halothane vaporizer to the inlet of the ventilator so that room air was drawn through the vaporizer. The ventilator was adjusted to provide an arterial oxygen tension (Pao2) of at least 80 torr and an arterial carbon dioxide tension (Pco2) of 28–34 torr. Control readings of EEG and ECG activity and control values for blood glucose and packed cell volume (PCV) were obtained.

Arterial pressure was monitored with an E and M P1000 pressure transducer and ECG activity with an E and M cardiac preamplifier MK IV. Both variables were recorded using a Physiograph Four recorder (E and M Instrument Co.). Arterial pressure was monitored and recorded continuously during the 3 hour observation period. ECG activity was also monitored and recorded in conjunction with blood pressure to identify any persistent arrhythmia.

EEG activity was monitored and recorded using a Grass model III D Electroencephalograph (Grass Instrument Co.). EEG activity was recorded during the control period, during the onset of CI until a silent recording was obtained, and then intermittently during and after the ischemia period to detect the reappearance of EEG activity. The sensitivity of the machine was set to record a 1 cm deflection for each 50 microvolt change in potential. A silent EEG recording was considered to be activity 5 μV peak to peak or less.7 ECG activity was monitored using the ECG preamplifier of the encephalograph so that ECG artifacts appearing in the EEG tracing could be identified.

Arterial gases and pH were determined using an IL 213 Digital Blood Gas Analyzer (Instrumentation Laboratory, Inc.). All measurements were made in duplicate. Blood gases and pH were monitored intermittently after the ventilator was adjusted for each rabbit. Values of samples taken at the midpoint of the ischemia period, at 10 minutes after reperfusion, and at 1, 2, and 3 hours after reperfusion were recorded. When spontaneous respiratory activity became strong and regular, the rabbit was disconnected from the ventilator and its blood gases and pH monitored. When it could maintain a Pco2 < 40 torr and a Paco2 of at least 80 torr, mechanical ventilatory assistance was discontinued. During the post-ischemia period, rabbits not maintaining a Paco2 of at least 80 torr were considered to have abnormal pulmonary function and were eliminated from the study. Blood glucose determinations were made using a YSI model 23A Glucose Analyzer (Yellow Springs Instrument Co.). PCV was determined using an Adams Autocrit Centrifuge (Clay-Adams, Inc.).

Once the control samples were obtained, anesthesia was stopped and trimethaphan was infused until peak systolic pressure was < 80 mm Hg. Cerebral ischemia was immediately induced by clamping the common

*Arfonad, Roche.
carotid arteries with noncrushing vascular clamps and by firmly tightening the umbilical tape ligature. During CI, the peak systolic pressure was kept between 70-80 mm Hg by infusion of Arfonad or norepinephrine.

In a group of 10 rabbits (Group I), after 20 minutes, the carotid arteries were undamped and the cervical umbilical tape ligature was removed. In a group of 4 rabbits (Group II), cerebral ischemia was maintained for 30 minutes and was terminated as in Group I. As soon as ischemia was ended, peak systolic pressure was raised to 100 mm Hg by infusion of norepinephrine until the rabbit gained vasomotor control and could maintain that pressure unassisted. After removal of the umbilical tape ligature, the wound was sutured.

At the time of return of vasomotor control, respiratory activity, corneal reflex, and EEG activity were recorded. Three hours post ischemia, the catheters and recording electrodes were removed and each rabbit was moved to an individual cage.

In this study, neurological recovery was defined as return of gross motor functions, i.e., ability to assume and maintain normal posture and ability to coordinate movement and survival during the 48 hours observation period. Each rabbit's functional status was assessed and given a numerical value according to a scale (table 5). Each rabbit was evaluated at 4, 8, 24, and 48 hours post-ischemia. After the final evaluation, each rabbit was killed with an overdose of barbiturate. Brains of 4 rabbits of Group I and 2 of Group II were removed as soon as possible (about 5 minutes) after the rabbit died. These were immediately placed in full strength formalin (37%) with just enough tap water added to allow the brain to sink. The brains were refrigerated and fixed in this solution for 24 hours. After this period, they were placed in 10% buffered formalin for at least one week. The brains were cut transversely into slices which were subsequently imbedded in paraffin, sectioned, and stained with hematoxalin and eosin.

The data from both groups were analyzed statistically using dependent and independent t-tests as appropriate. A value for \( P \) equal to or less than 0.05 was considered to indicate a significant difference between the data analyzed.

Results

Completeness of Interruption of Cerebral Blood Flow

The completeness of the interruption of cerebral blood flow was investigated in 5 rabbits. One rabbit was decapitated at the atlantooccipital joint after induction of cerebral ischemia and the disappearance of EEG activity. No arterial blood flow was seen while the rabbit was being ventilated and its heart was beating. One rabbit was killed and barium suspension was injected into the left ventricle at a pressure of 150–200 mm Hg while all clamps and ligatures were in place. No barium was seen in the vessels of the brain by radiographic or direct visual examination. In the remaining 3 rabbits, cerebral blood flow was measured by autoradiographic technique using \(^{14}\)C labelled antipyrine. No flow was detected.

Neurological Changes Due to Surgical Procedure

An additional five rabbits, allowed to recover from surgery without undergoing a period of CI, were observed intermittently for 48 hours. No difference in behavior or motor abilities was seen between the surgically prepared rabbits or normal rabbits housed in the same environment.

Physiological Data

The control values of blood gases, pH, blood glucose, and PCV were similar in Group I and Group

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**Table 1** Effect of 20 Minutes of Cerebral Ischemia on Some Blood Parameters in 10 Rabbits

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Baseline</th>
<th>Mid isch.</th>
<th>End isch.</th>
<th>1 Hr.</th>
<th>2 Hr.</th>
<th>3 Hr.</th>
</tr>
</thead>
<tbody>
<tr>
<td>PaO₂ (torr)</td>
<td>96 ± 11</td>
<td>98 ± 8</td>
<td>105 ± 8</td>
<td>105 ± 13</td>
<td>106 ± 11</td>
<td>105 ± 8</td>
</tr>
<tr>
<td>PaCO₂ (torr)</td>
<td>32 ± 3</td>
<td>29 ± 4</td>
<td>30 ± 4</td>
<td>28 ± 2</td>
<td>30 ± 3</td>
<td>30 ± 5</td>
</tr>
<tr>
<td>ApH</td>
<td>7.45 ± .03</td>
<td>7.46 ± .03</td>
<td>7.45 ± .03</td>
<td>7.47 ± .04</td>
<td>7.45 ± .05</td>
<td>7.41 ± .06</td>
</tr>
<tr>
<td>Glucose (mg/dL)</td>
<td>106 ± 20</td>
<td>104 ± 23</td>
<td>96 ± 31</td>
<td>104 ± 46</td>
<td>107 ± 66</td>
<td>110 ± 57</td>
</tr>
<tr>
<td>PCV (%)</td>
<td>37 ± 2</td>
<td>34 ± 2</td>
<td>36 ± 2</td>
<td>38 ± 2</td>
<td>39 ± 2</td>
<td>38 ± 2</td>
</tr>
</tbody>
</table>

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**Table 2** Effect of 30 Minutes of Cerebral Ischemia on Some Blood-Parameters in 4 Rabbits

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Baseline</th>
<th>Mid isch.</th>
<th>End isch.</th>
<th>1 Hr.</th>
<th>2 Hr.</th>
<th>3 Hr.</th>
</tr>
</thead>
<tbody>
<tr>
<td>PaO₂ (torr)</td>
<td>106 ± 7</td>
<td>110 ± 8</td>
<td>114 ± 6</td>
<td>112 ± 9</td>
<td>106 ± 10</td>
<td>106 ± 15</td>
</tr>
<tr>
<td>PaCO₂ (torr)</td>
<td>31 ± 2</td>
<td>29 ± 1</td>
<td>25 ± 1</td>
<td>28 ± 5</td>
<td>30 ± 1</td>
<td>30 ± 7</td>
</tr>
<tr>
<td>ApH</td>
<td>7.45 ± .04</td>
<td>7.46 ± .02</td>
<td>7.44 ± .02</td>
<td>7.47 ± 1.0</td>
<td>7.38 ± .04</td>
<td>7.40 ± .04</td>
</tr>
<tr>
<td>Glucose (mg/dL)</td>
<td>110 ± 24</td>
<td>100 ± 17</td>
<td>84 ± 11</td>
<td>81 ± 23</td>
<td>98 ± 37</td>
<td>97 ± 40</td>
</tr>
<tr>
<td>PCV (%)</td>
<td>36 ± 1</td>
<td>34 ± 1</td>
<td>35 ± 1</td>
<td>37 ± 3</td>
<td>37 ± 3</td>
<td>36 ± 2</td>
</tr>
</tbody>
</table>
less stimulated vigorously. The rabbits ischemic for 30 minutes were initially more depressed and less responsive than the rabbits ischemic for 20 minutes (table 5). At 48 hours, the rabbits having a score of 1 were upright and would eat and drink, but were not as responsive as the normal rabbits living in the same environment. They would remain sitting for long periods without moving, and tremors of the head and limbs were present when they moved about. Rabbits having a score of 2 would move only when vigorously stimulated, were very uncoordinated on moving, and would frequently fall. They did not eat or drink spontaneously. Those with a score of 3 were recumbent, appeared blind, and could not control their rear limbs.

The 3 rabbits that died did so within 18 hours post-ischemia. At 8–10 hours after ischemia the following signs appeared in these rabbits: lethargy, progressive extensor rigidity, irregular panting respiration, opisthotonus, and periodic running movements.

Although there was no statistically significant difference in neurological scores between the rabbits ischemic for 20 to 30 minutes, the mean scores of Group I were consistently higher than the scores of Group II (table 5).

**Histopathology**

Subdural hemorrhages were found on the brains of all rabbits ischemic for 30 minutes. No hemorrhages were found within the substance of these brains. The histopathological lesions consisted of loss of neurons, ischemic cell changes, increased vascularity, and status spongiosus. These lesions were found in the hippocampus and the cerebellum. Many dark neurons were seen throughout the cortex and brain stem. There was no clear distinction in the severity of the lesions between the two groups.

**Discussion**

The cerebral blood supply of the rabbit flows through the internal carotid and vertebral arteries. There is a small collateral supply which consists of anastomoses between arteries supplying the muscles of the neck and the carotid and vertebral arteries. These vessels were controlled by the surgical preparation used. Although the surgical preparation does not preclude blood flow through the ventral spinal artery, flow does not occur probably because of the trimethaphan-induced hypotension. However, Sainio\(^6\) found that blood flow to the brain of rabbits, in which he inflated a cervical pressure cuff to induce cerebral ischemia, had no collateral flow through the ventral spinal artery, and Hossmann\(^9\) has shown that

### Table 3. Effect of 20 and 30 Minutes of Cerebral Ischemia on Recovery of EEG Activity and Some Reflexes in Rabbits

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Group I (10 rabbits)</th>
<th>Group II (4 rabbits)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Return of vasomotor control</td>
<td>8 ± 2 Min.</td>
<td>7 ± 1 Min.</td>
</tr>
<tr>
<td>Return of resp. activity</td>
<td>8 ± 4 Min.</td>
<td>7 ± 1 Min.</td>
</tr>
<tr>
<td>Return of corneal reflex</td>
<td>26 ± 3 Min.</td>
<td>37 ± 6 Min.</td>
</tr>
<tr>
<td>Return of EEG activity</td>
<td>26 ± 3 Min.</td>
<td>34 ± 7 Min.</td>
</tr>
<tr>
<td>Ventilatory support discontinued</td>
<td>23 ± 7 Min.</td>
<td>39 ± 7 Min.</td>
</tr>
</tbody>
</table>

### Table 4. Arterial Pressure Changes During Post-Ischemic Period

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Group I (10 rabbits)</th>
<th>Group II (4 rabbits)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peak mean pressure</td>
<td>124 ± 2 mm Hg</td>
<td>123 ± 5</td>
</tr>
<tr>
<td>Interval from end of ischemia</td>
<td>17 ± 3 Min.</td>
<td>18 ± 7</td>
</tr>
<tr>
<td>Stable mean pressure</td>
<td>95 ± 10 mm Hg</td>
<td>101 ± 2</td>
</tr>
<tr>
<td>Interval from end of ischemia</td>
<td>34 ± 5 Min.</td>
<td>34 ± 6</td>
</tr>
</tbody>
</table>

II (tables 1 & 2). The duration from arterial clamping to the onset of EEG silence was between 35 and 70 sec in both groups. Times to recovery of reflexes are seen in table 3. There were significant differences in recovery of corneal reflex and EEG activity between the groups, and the times at which ventilatory assistance could be discontinued was also different between groups. Respiratory movements (gasping) were noted at about the same time as vasomotor control returned.

In both groups the mean arterial pressure rose steadily once vasomotor control returned. Arterial pressure reached its peak 17–20 minutes post-ischemia and then returned to a stable but lower pressure by 30–44 minutes post-ischemia (table 4). It remained at this level to the end of the monitoring period.

Paco₂ decreased in both groups during the period of ischemia. It remained at less than the control value during the recovery period because the rabbits tended to hyperventilate spontaneously. Changes in PCV and blood glucose concentration were insignificant.

**Neurological Status**

At 4 hours post-ischemia the rabbits of both Group I and Group II were recumbent and unresponsive un-

### Table 5. Mean Neurologic Scores* of Rabbits Subjected to 20 and 30 Minutes of Cerebral Ischemia

<table>
<thead>
<tr>
<th>Hours post ischemia</th>
<th>Group I (10 rabbits)</th>
<th>Group II (4 rabbits)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>2.7 ± 0.48</td>
<td>2.5 ± 0.71</td>
</tr>
<tr>
<td></td>
<td>2.6 ± 1.17</td>
<td>2.5 ± 1.27</td>
</tr>
</tbody>
</table>

*Criteria for scoring: 1 = Alert, normal upright posture. Minor motor dysfunction. 2 = Poorly responsive to stimuli, can attain normal posture but has severe incoordination. 3 = Responsive only to vigorous stimulation, cannot maintain normal posture. 4 = Dead.
The rabbits that died in this study had signs of increasing historically accepted limits. However, it has long been known that the vegetative functions of the brain can be eliminated by vagotomy and sympathectomy. Routine use of atropine and trimethaphan prior to induction of cerebral ischemia abolished these vasomotor events in the rabbits and prevented the problem of pulmonary edema. This observation may explain the occurrence of pulmonary edema noted in other such studies and may bear some relation to the pulmonary damage seen with hypoxemia.  

An additional vasomotor event that was seen in this study was a transient but pronounced rise in mean arterial blood pressure after cerebral ischemia. The same sort of change has been seen in dogs. Systemic pressure rose, once spontaneous vasomotor control was regained. The rise was slow, reaching its peak 15-20 minutes after reperfusion, and then slowly declining to a stable pressure 30-40 minutes after reperfusion (table 4). This response was probably a manifestation of cerebral hyperperfusion that is known to follow prolonged cerebral ischemia. This hyperperfusion is recognized to cause cerebral edema. The rabbits that died in this study had signs of increasing intracranial pressure (vide supra) most likely caused by cerebral edema.

Over 70% of the rabbits (11/14) survived through the 48 hour observation period. This is a greater number than would be expected to survive such prolonged cerebral ischemia on the basis of historically accepted limits. However, it has long been known that the vegetative functions of the brain will return after prolonged ischemia, and recent evidence suggests that extracranial factors are of great importance in limiting post-ischemia recovery. Monkeys have survived and recovered from 20 minutes of cerebral ischemia, and evidence for return of neuronal activity has been obtained after 30 and 60 minutes of cerebral ischemia. It is believed, on the basis of these findings, that adequate support of cardiovascular and respiratory functions in the post-ischemia period is critical to survival. This evidence encourages the belief that the cardipulmonary support afforded these rabbits allowed a prompt return of adequate cerebral blood flow and that this accounts for the greater than expected survival rate.

Results of the tests for completeness of cerebral ischemia indicate that complete ischemia was achieved. The physiological data obtained from, and the neurological scores assigned to, the rabbits indicate a reproducible degree of cerebral ischemic injury was achieved. The difference in the results between the 2 groups indicates that the severity of the insult varied with duration. Return of corneal reflex, EEG activity, and adequate spontaneous ventilation were significantly prolonged in the 30 minute ischemia group and motor deficits were more profound. This finding supports the suggestion that motor disability can be used as an index of neuronal damage and be a more useful end point for the evaluation of treatment regimens than survival alone. These findings, combined with the fact that this method of producing cerebral ischemia in rabbits does not have the disadvantage of causing ischemia or hypoxia of other vital organs, and the fact that the ischemia is reversible and that animals can be recovered for prolonged study, seems to make this model suitable for use in investigation of cerebral ischemia in lower animals.

Acknowledgments

The author wishes to acknowledge the dedicated technical assistance of Mrs. M.E. Kolata, the encouragement and support of the late Dr. D.B. Polis of the Naval Air Development Center, Warminster, PA, and the help of Drs. M. Reivich and J. Greenberg of the Cerebral-Vascular studies unit of the University of Pennsylvania Medical School.

References

models of brain ischemia. Stroke 7: 14–17, 1976

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**Occlusion of the Vertebral or Basilar Artery**

**Follow Up Analysis of Some Patients with Benign Outcome**

**LOUIS R. CAPLAN, M.D.**

**SUMMARY** Ten patients with angiographically verified occlusion of the basilar or vertebral artery have been followed for an average of 2.75 years. None has developed further ischemia after the initial stroke, and 4 patients survived without any clinical deficit. In occlusive disease of the posterior circulation, the critical period for deficit acquisition is at the time of occlusion. Extent of the deficit depends on the rapidity of development of adequate collateral circulation, and the presence of distal embolization at the time of occlusion. Some patients survive basilar occlusion without permanent deficit.

**OCCLUSION of the basilar artery is generally considered a very serious event incompatible with normal survival. Kubik and Adams** described 18 patients with brainstem infarction due to occlusion of the basilar artery discovered at postmortem examination and emphasized the abrupt onset and frequent fatal outcome. Marshall subsequently pointed out that many untreated patients with the clinical picture of posterior circulation vascular disease do not develop serious deficits; however, the underlying vascular pathology in this group of clinical patients was usually a major factor limiting recovery from cerebral ischemia. J Surg Res 9: 525–529, 1969

**No reference could be found on follow up of patients with angiographically documented vertebrobasilar occlusive disease.**

**The author’s files, and those of the Harvard Stroke Registry,** were searched for patients meeting the following criteria: 1) patients who were examined and followed carefully by the author during hospitalization for acute stroke; 2) technically satisfactory angiograms taken during the stroke which documented an occlusion of either a vertebral or the basilar artery; 3) patients who survived the acute stroke and had been followed by the author more than 6 months. The temporal profile of illness and clinical outcome in this group of patients gave insights into the pathogenesis of the clinical deficit and mechanisms of compensation.

**Results**

**Case Material**

Ten patients (8 male, 2 female) from 26–71 years of age (average 52.2 years) were studied clinically and angiographically. Six had occlusion of the basilar artery (2 proximal and 4 midportion beyond the
Survival of rabbits after prolonged cerebral ischemia.
R J Kolata

Stroke. 1979;10:272-277
doi: 10.1161/01.STR.10.3.272

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