A comparison of effects of bipolar and monopolar electrocoagulation in brain

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Controlled bipolar and monopolar coagulation lesions were generated in the cerebral cortex of cats. Higher output powers were associated with larger lesions, while the lesion size was independent of the mode of coagulation. When cortical vessels were mobilized and coagulated for hemostasis, bipolar mode was associated with more rapid coagulation and less damage to the underlying brain. Higher output powers were not associated with larger lesions, probably because coagulation was more rapid. The neural damage resulting from radiofrequency current appears to be of thermal origin, and the blood-brain barrier dysfunction is a more sensitive measure of this damage than the stainable cellular changes.

Key Words - electrocoagulation, bipolar and monopolar • surgical hemostasis • blood-brain barrier

Use of electrocoagulation in surgery owes much to the early demonstration by d’Arsonval of the effect of electrical stimulation on muscle. He demonstrated that with alternating current the contraction produced would slowly decrease as the frequency of the stimulus was increased, until at about 10 kHz contractions disappeared altogether. As the frequency was increased further, only tissue heating occurred. Ward noted that the “damped wave” stimulation, that is, output with an exponentially decaying curve, produced better coagulation than “sine wave” stimulation. However, Sigel and Hatke have suggested that the waveform is not the critical determinant of coagulation bond strength. Using a current generator with interrupted sine wave pulses, they estimated that the amount of energy required for vascular coagulation in a living animal is about 40 watt-seconds per cubic millimeter of tissue.

Greenwood, who introduced bipolar coagulation to neurosurgery, estimated that the current necessary for this mode of coagulation is only one-third that required for monopolar coagulation. He postulated that the brain damage was minimized by the smaller volume of tissue in the electrical field and by decreased heat production. To further decrease damage from conduction of heat, he recommended irrigation with saline. Self-irrigating bipolar diathermy forceps and forceps with automatic thermocontrol have since been introduced.

It has become a widely held view that bipolar coagulation causes less focal damage to nervous tissue than the monopolar coagulation, despite the lack of quantitative comparison of lesions produced by these devices. The present study was directed toward quantification and comparison of the tissue damage produced by bipolar and monopolar modes of radiofrequency electrocoagulation.

Materials and Methods

Experimental Technique

A Malis bipolar coagulator was used as the radiofrequency generator unit in this study. This is a spark-gap generator with a power range of 0.5 to 38 watts and a current range of 0.05 to 0.72 amps. Output can be varied by a dial which operates by changing the ratio between the primary and the secondary turns of a solenoid coil. For this study, we used dial positions of 15, 25, 35, 45, and 60 only. The output...
was measured across a 50-ohm non-inductive resistor placed between the forceps tips, and consisted of interrupted pulses of 7- to 10-μsec duration with exponential decay and with an average interval of 400 ± 100 μsec between adjacent pulses. Each pulse contained a burst of 7 to 10 damped oscillations with frequency in the range of 1 mHz. Maximum peak-to-peak amplitudes of 300 to 2000 volts were measured for dial positions of 15 to 60. Output power for different dial positions was measured by passing the current from the coagulator through a 50-ohm non-inductive resistor. The temperature of the resistor was measured with a thermistor which was in close contact with the surface of the resistor. A graph showing thermistor resistance versus dial setting was obtained using the steady-state resistances of the thermistor. The resistor was then connected to a variable DC power supply, with the current through the resistor and the voltage across it monitored by an ammeter and a voltmeter, respectively. With the thermistor resistance being monitored again, a relationship between applied power and thermistor resistance was established and plotted. Using these two graphs, it was possible to determine the output power of the coagulator at the various positions. Dial positions of 15, 25, 35, 45, and 60 produced 1.0, 2.0, 5.2, 8.8, and 12.1 watts, respectively. Power associated with an individual pulse was then calculated and divided by the ratio of the area of the pulse to the area under the first one-half oscillation for a measure of power associated with the largest phase. This figure was then divided by the corresponding base-to-peak voltage for measure of the maximum charge per phase at each dial setting. The same generator was used for the production of all lesions.

The same microbipolar forceps were used for all coagulations, and tips were cleansed after each application. For monopolar coagulation, both leads from the bipolar forceps were connected to the same outlet on the coagulator, and the other outlet was connected to the animal by a low-resistance wire attached to the temporalis muscle. Coagulations were performed simulating techniques commonly accepted in neurosurgical practice. Thus, the bipolar coagulation was done under a thin layer of saline, and the monopolar coagulation in a relatively dry field.

The study consisted of two parts. First, controlled bipolar and monopolar lesions were produced in the cerebral cortex of the cat, and the cross-sectional areas of damage were measured. In the second part, pial veins of equal size were mobilized and coagulated with each mode to compare the coagulation effectiveness.

An oscilloscope and a radiofrequency ammeter were used to monitor the waveform and output during several preliminary coagulations in each part of the study. At the same levels of output, no difference was noted between the bipolar and monopolar setups, as demonstrated in Fig. 1.

**Placement of Cortical Lesions**

Twenty-four adult cats, weighing 2 to 4 kg, were anesthetized with 30 mg/kg of intraperitoneal diabutal. Femoral venous and arterial lines were placed, and blood pressure was monitored continually. Tracheotomy was performed, and the animal was paralyzed and placed on a small-animal respirator. The expiratory CO2 was maintained between 2% and 4%. Core body temperature was kept at 36° to 38°C. The animal's head was fixed in a stereotaxic apparatus, and bilateral craniectomies were fashioned. The dura and the arachnoid membranes were reflected. Then 2 ml/kg of 5 gm% solution of Evans blue in normal saline was injected intravenously, and 10 minutes were allowed for protein binding. During bipolar coagulation, the forceps tips were held 2 mm apart by a removable plug of dental cement, while, in monopolar coagulation, the tips were held together. Under X 40 magnification of a Zeiss operating microscope, the forceps tips were gently placed on the surface of the brain so that they did not alter brain contour; therefore, although the brain surface contact of the forceps in both settings was equal in area, it was of different configuration. Using the dial positions of 15, 25, 35, 45, and 60, bipolar and monopolar lesions of 2-second duration were placed on the crown of the parasagittal gyri in a random fashion. Five lesions were placed on each hemisphere. Three such lesions are seen in Fig. 2. Lesions that were associated with visible sparking or mechanical disruption of brain surface were discarded. The animals were monitored for 1 hour after completion of coagulation, and then the brains were removed and fixed in formalin.

Subsequently, coronal sections were made through
Comparison of bipolar and monopolar electrocoagulation

Fig. 2. Evans blue staining of cortex 1 hour after placement of 2-second lesions. Three lesions corresponding to dial positions of 35, 45, and 60 can be seen, from left to right.

Coagulation of Isolated Vessels

Twenty-three cats were prepared in the same way as for cortical lesioning. After exposure of the brain, cortical veins 0.2 to 0.4 mm in diameter were selected under magnification. The pia was incised and the veins were mobilized for a length of 5 to 6 mm, as demonstrated in Fig. 3. During coagulation, vessels were grasped with forceps and were lifted slightly off the surface of the brain. The same forceps were used in both modes, so the surface area and configurations of forceps tips were identical. Again, monopolar coagulation was done in a dry field, and bipolar under a layer of saline. The end point for coagulation was established by the minimum amount of current necessary to produce cessation of blood flow under X 40 magnification (Fig. 4). Bipolar and monopolar modes at dial positions of 15, 25, 35, 45, and 60 were selected in a random fashion to coagulate five to seven veins in each animal. Cats were then infused with 2 ml/kg of 5 gm% Evans blue, and monitored for 1 hour prior to sacrifice.

Following sacrifice, brains were removed and analyzed as described above. Control lesions were made...
B. Chehrazi and W. F. Collins, Jr.

FIG. 4. An isolated pial vein immediately after coagulation. No Evans blue staining of the parenchyma has developed yet.


FIG. 6. Evans blue discoloration of parenchyma in cross section through the center of the lesion after direct cortical coagulation. Dial position 35.

FIG. 7. Graph showing the cross-sectional area of lesions plotted against output and corresponding dial positions for direct cortical coagulation. The solid circles represent the monopolar and the open circles the bipolar modes. Lesion areas are shown in solid lines for Evans blue and in broken lines for histological stains.

Results

Cortical Coagulation

For the study of direct cortical coagulation, 170 lesions were examined. Lesions were clearly demarcated as a wedge-shaped structure with their base on the cortex. Microscopically, they were characterized by ground substance depigmentation and vacuolization and neuronal damage with pyknotic, angulated nuclei and loss of detail (Fig. 5). Disruption and discoloration of myelin was present when lesions extended into the white matter. In sections stained with Evans blue, the lesions were demarcated, with blue-green discoloration of the parenchyma and superficial cortical necrosis at the site of coagulation (Fig. 6). In control sections, no parenchymal staining by the...
Comparison of bipolar and monopolar electrocoagulation

**TABLE 1**

Comparison of bipolar and monopolar cortical electrocoagulation in generation of brain lesions*

<table>
<thead>
<tr>
<th>Mallis Dial Settings</th>
<th>Monopolar Mode</th>
<th>Bipolar Mode</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>EB Stain</td>
<td>H &amp; E &amp; KB Stain</td>
</tr>
<tr>
<td>15</td>
<td>0.4 ± 0.1</td>
<td>0.2 ± 0.0</td>
</tr>
<tr>
<td>25</td>
<td>0.9 ± 0.2</td>
<td>0.3 ± 0.0</td>
</tr>
<tr>
<td>35</td>
<td>9.1 ± 1.5</td>
<td>1.1 ± 0.2</td>
</tr>
<tr>
<td>45</td>
<td>16.7 ± 0.8</td>
<td>2.1 ± 0.2</td>
</tr>
<tr>
<td>60</td>
<td>19.0 ± 0.8</td>
<td>4.0 ± 0.2</td>
</tr>
</tbody>
</table>

*Number of lesions in each group are in parentheses. Each value is the mean area in sq mm ± standard error. EB = Evans blue; H & E = hematoxylin and eosin; KB = Kluver-Barrera luxol fast blue.

**TABLE 2**

Comparison of brain lesions associated with bipolar and monopolar vascular electrocoagulation*

<table>
<thead>
<tr>
<th>Mallis Dial Settings</th>
<th>Monopolar Mode</th>
<th>Bipolar Mode</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>EB Stain</td>
<td>H &amp; E &amp; KB Stain</td>
</tr>
<tr>
<td>25</td>
<td>6.5 ± 0.3</td>
<td>0.4 ± 0.0</td>
</tr>
<tr>
<td>35</td>
<td>5.2 ± 0.7</td>
<td>0.6 ± 0.1</td>
</tr>
<tr>
<td>45</td>
<td>1.5 ± 0.1</td>
<td>0.4 ± 0.1</td>
</tr>
</tbody>
</table>

*Number of lesions in each group are in parentheses. Each value is the mean area in sq mm ± standard error. EB = Evans blue; H & E = hematoxylin and eosin; KB = Kluver-Barrera luxol fast blue.

Mean cross-sectional areas for monopolar and bipolar lesions at selected dial positions were measured by Evans blue, H & E, and KB stains (Table 1). Lesions measured 0.2 to 3.0 sq mm using histological stains and 0.4 to 19.5 sq mm using Evans blue. With direct cortical lesioning, corresponding bipolar lesions were in general larger than monopolar ones (Fig. 7). The differences, however, were not statistically significant as measured by Evans blue or H & E and KB. The lesion areas determined by staining with Evans blue were, in general, seven times larger than those by H & E and KB stains. This relationship was consistent in both parts of this study, regardless of the mode of coagulation.

**Vascular Coagulation**

Coagulation of isolated vessels at dial position 15 resulted in failure of effective coagulation, and at dial position 60 resulted in frequent sparking and hemorrhage. These positions were, therefore, excluded from analysis. Dial position 25 was slow and relatively inefficient for coagulation in monopolar mode, but resulted in rapid and effective coagulation in bipolar mode. Eighty-five lesions were included for analysis. The mean cross-sectional areas for these lesions are shown in Table 2.

In contrast to direct brain lesioning during coagulation of isolated vessels, higher dial settings were associated with a shorter period of coagulation and generally smaller parenchymal lesions as compared to lower dial settings. This was especially pronounced with Evans blue staining, as seen in Fig. 8. Relative to the monopolar mode, the bipolar mode resulted in more rapid coagulation and smaller parenchymal spread of lesions at all dial positions tested. The differences were significant at 0.01 level for Evans blue and at 0.05 level for histological stains. Control brain sections, with dissected but not coagulated vessels, were free of lesions.

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Discussion

In 1961, Lilly stated that injury due to electrical stimulation of the brain could be either electrolytic or thermal in origin. The electrolytic injury could be related to the amount of unidirectional current passed through the tissue; and the thermal injury was essentially proportional to the average power dissipated in the tissue. Reporting on the production of brain lesions with electric currents, Rowland, et al., observed that, with biphasic current, the lesion was proportional to the number of microcoulombs (μC) per pulse and the total number of coulombs, but was independent of the frequency, pulse duration, current, or voltage amplitudes. They applied biphasic stimulation to brain with pulses containing fewer than 20 μC, without producing histological alteration. Using frequencies of 10 to 250 pulses per second, Pudenz, et al., demonstrated that blood-brain barrier (BBB) breakdown and neuronal damage are most closely related to the number of microcoulombs in each phase of a biphasic waveform. They were able to stimulate continuously for 36 hours without causing a BBB lesion when charge per phase was 0.3 μC or less. Considering that the charge per phase in radiofrequency waveforms is very small (estimated to be in the order of 1.0 μC for dial position 60 and 0.6 μC for 35), it is unlikely that any significant electrolytic injury takes place. It appears that large amounts of radiofrequency current can be passed through tissue with minimal physiological effect other than that of induction of heat.

Utilizing a symmetrical, low-frequency, biphasic current, Lilly noted that the thermal injury was proportional to the average power dissipated in the tissue. In radiofrequency currents, the physical principles involved are, however, complicated since the currents used change their direction of flow from 0.5 to 3 million times per second and distribute themselves in a manner very different from that of direct or alternating currents of low frequency. Thus, the relationship between voltage and current is upset by what is termed “phase shift.” Reporting their clinical experience with radiofrequency electroosurgical generators, Ward and McLean noted that the tissue coagulation was produced by the localized heating effect on tissue.

Mitchell and Lumb stated that the temperature gradient in the tissue depends on the power output of the generator, the specific resistance of the tissue, and the circulatory efficiency in dissipating the heat produced. Sigel and Hatke commented that high-frequency electric current changes remarkably during vessel coagulation. The constantly changing waveform, current density, and tissue impedance due to heating and progressive dessication complicate measurements of electrical energy at the tissue level.

Most radiofrequency coagulators generate frequencies of 0.5 to 3 mHz, have a tuned circuit, and are relatively unaffected by small changes in impedance; they therefore produce satisfactory coagulation without requiring adjustment to circuitry from one patient to the next. In this study, when an oscilloscope and a radiofrequency ammeter were used to monitor the waveform and output, no difference was noted between the bipolar and monopolar settings, indicating that the change in conductance between the two settings was not enough to cause a change in the output of this tuned generator. Thus, the same amount of radiofrequency energy was imparted to the brain during both bipolar and monopolar coagulation.

In this study, lesions measured by Evans blue stain were significantly larger than those measured by H & E and KB stains; the Evans blue lesions were on average six times larger than the corresponding stainable cellular changes. This relationship held in both parts of this study and for both modes of electrocoagulation. Discoloration of parenchyma by extravasated Evans blue in a measure of vascular permeability related to BBB dysfunction, and correlates well with the resultant focal edema. Pudenz, et al., and Mortimer, et al., have also noted the more extensive damage to BBB due to electrical stimulation. Agnew, et al., using horseradish peroxidase and electron microscopy, failed to confirm altered permeability in the face of widespread Evans blue staining. This suggests that the vital dye penetration may be a more sensitive indicator of the effects of electrical stimulation than the changes verifiable with electron microscopy.

In this study, when the current was applied directly to the cortex, the bipolar mode provided no protection against tissue damage relative to the monopolar mode. In fact, although the difference was not statistically significant, monopolar lesions were on the average smaller. However, when coagulating isolated vessels, the bipolar mode provided more rapid coagulation at settings tested and caused significantly less damage to the underlying brain. Lower levels of output not only did not protect against damage, but also were associated with larger lesions probably due to the longer period of application necessary for coagulation. It appears that the spread of the thermal lesion can be minimized by selecting an output level that results in the shortest duration of application but without sparking and secondary hemorrhage.

These studies fail to demonstrate that bipolar coagulation, even when used under a layer of saline, causes less neural damage than monopolar when coagulating brain. While coagulating an isolated vessel, however, the increased efficiency of bipolar coagulation results in a shorter period of current flow and less damage to the adjacent brain. We could not confirm Greenwood’s claim that in the bipolar mode coagulation is limited to the vessel held, without extension to adjacent brain tissue.

Neurosurgeons using bipolar coagulation cannot expect the technique in itself to limit the area of damage or to allow any decrease in care in protecting the brain.
Comparison of bipolar and monopolar electrocoagulation

adjacent tissue; rather, these studies reinforce the concept that all electrical coagulation causes damage that is best controlled by a careful surgical technique.

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