MODIFICATION OF THE CIRCULATORY RESPONSE TO ELECTROSHOCK THERAPY BY THIOPENTAL

LT. LAWRENCE D. EGBERT, USN (MC), PETER A. DUMAS, M.D.,
GEORGE C. GINTER, M.D., JAMES E. ECKENHOFF, M.D.

Electroshock therapy profoundly affects the circulation. With the onset of the convulsion, arterial blood pressure and heart rate suddenly decrease for approximately one second. This is followed by a period when blood pressure and heart rate increase to about 150 per cent of normal and the circulation time is reduced. Venous and intrathoracic pressures are elevated to over 100 mm. of mercury throughout the tonic phase of the convulsion and then gradually return to normal during the clonic phase. Variations in heart rhythm are common. Sinus tachycardia occurs in most patients and occasionally ventricular arrhythmias appear during the tonic convulsion. Later, in the clonic phase of the convulsion, the heart rate decreases rapidly, sometimes with the appearance of nodal rhythm. The return to normal is usually complete within ten minutes.

These alterations in circulatory dynamics may cause permanent damage to the cardiovascular system although Holmberg et al. believe that ordinarily no harm results because of the short duration of the disturbance. Nevertheless, Will, Rehfelt, and Neumann collected 33 reports of death following shock therapy, 10 of which were clearly due to circulatory failure.

Barbiturates may modify the circulatory response to electroshock. Hoff noted a pressor response to electroshock in dogs anesthetized with ether or paralyzed with curare but was unable to elicit such an effect in dogs under pentobarbital hypnosis. Griswold reported little or no rise in blood levels of epinephrin and norepinephrin when a barbiturate had been administered prior to shock. Wayne wrote that less hypotension was observed in his patients after he began administering thiobarbiturates before treatment, but he presented no confirmatory data. Also, Nowill, Wilson and Borders demonstrated a decreased incidence of cardiac arrhythmias when thiopental had been given prior to electroshock. We have investigated the effect of varying doses of thiopental upon blood pressure and heart rate following electroshock therapy.

Method

Eight patients were studied comparing two dose levels of thiopental. No preanesthetic medication was given. The order of treatment was randomized. Either 100 mg. or 500 mg. of thiopental were administered rapidly intravenously and immediately were followed by 80 mg. of succinylcholine. Exactly one minute after the thiopental had been injected, a direct current of 110 volts was applied through the temporal area for five seconds. During the period of apnea all patients' lungs were ventilated with bag and mask twelve times a minute using a ten liter flow of oxygen. An electrocardiogram was taken throughout the procedure except during the shock. Arterial blood pressure was monitored with an aneroid manometer. Six determinations per minute were made by this method. The duration of apnea and the time of awakening after the treatment were measured with a stopwatch.

Using the “t” ratio for statistical significance, comparisons were made between the two treatments on: the maximum change in blood pressure, the time during which the blood pressure was 20 per cent or more above the control blood pressure taken before the start of anesthesia, the greatest heart rate changes, and the duration of apnea and unconsciousness.
TABLE 1

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Sex</th>
<th>Before 500 mg. Thiopental</th>
<th>Change after Shock</th>
<th>Change after Shock</th>
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<tr>
<td></td>
<td></td>
<td>Maximum</td>
<td>Duration</td>
<td>Maximum</td>
</tr>
<tr>
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<td>130/82</td>
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<td>2</td>
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<td>130/90</td>
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</tr>
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<td>M</td>
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</table>

Mean        | 137/86 | +34/+24  | 38      | 136/89  | +78/+45  | 160    |

*Duration, in seconds, of hypertension greater than 120% of control.

RESULTS

After 500 mg. of thiopental, electroshock caused the blood pressure to rise from a control mean of 137/86 mm. of mercury to 171/110, an average increase of 34/24, while after 100 mg. of thiopental, the blood pressure rose from 138/89 to 214/134, an increase of 78/45 (table 1). Thus, following the smaller dose of thiopental, shock caused an average rise in systolic pressure of 44 mm. of mercury greater than following the larger dose. This is statistically significant ($P < .01$). The mean rise in diastolic pressure was 21 mm. of mercury greater after the smaller dose of hypnotic ($P < .01$). Following shock, blood pressure remained above 120 per cent of the control levels for an average of 38 seconds after 500 mg. of thiopental while hypertension lasted a mean of 160 seconds after 100 mg. thiopental. The mean difference of 122 seconds duration of hypertension is probably significant ($P < .05$). Hypotension did not occur after the treatment with either dose of thiopental.

Electroshock caused the heart rate to rise an average of 21 beats per minute after 500 mg. of thiopental, while after 100 mg. of thiopental it caused an average rise of 68 beats (table 2). This is statistically significant ($P < .01$). About one minute after the shock the pulse suddenly slowed in most patients. Variations in heart rhythm were observed in several patients. The most notable of these, a patient with severe cardiovascular disease (patient no. 4), had numerous ventricular extrasystoles during the period of tachycardia (heart rate 152) and variations in the contour of the P wave and duration of the P-R interval during the slowing of the heart after anesthesia with 100 mg. of thiopental. There were no changes in the electrocardiogram except a slight increase in heart rate (89) after 500 mg. of thiopental. Although the pulse rate during recovery from shock was significantly lower than in the control period, there was no difference in the slowing observed with the two methods of anesthetization.

The duration of apnea was significantly longer after the large dose of thiopental (mean of 361 seconds from the time of shock to return of respiration) than after the small dose (mean

TABLE 2

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Before 500 mg. Thiopental</th>
<th>Change after Shock</th>
<th>Before 100 mg. Thiopental</th>
<th>Change after Shock</th>
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</table>

Mean        | 88      | +21     | -15     | 85      | +68     | -16     |
242 seconds). Also, the duration of sleep after treatment was longer when the large dose was used (mean 12.9 minutes) than after the small dose (mean 6.9 minutes).

**Comment**

Our data show that the circulatory changes due to electroshock therapy can be lessened by increasing the dose of thiopental. This concurs with the data of Nowill, Wilson and Borders, who reported a decrease in the incidence of arrhythmias following shock treatment if thiopental had been given. They concluded that thiopental blocks the circulatory response to electroshock. They also found, as we have, that bradycardia follows the period of tachycardia whether or not thiopental was administered before treatment. They were able to prevent this with atropine.

There are several possible causes of the change in circulatory dynamics. Cortical impulses created by electroshock may excite central autonomic nervous system centers. Stimulation of the cerebral cortex with electrical current may result in either hypertension or hypotension in cats depending upon the area stimulated. The adrenal medulla is indirectly stimulated and blood levels of catechol amines are elevated after shock therapy. Also, tetraethylammonium chloride and hexamethonium prevent the hypertension associated with the convulsion by blocking the sympathetic ganglia. Muscular activity may account for some of the hypertension and tachycardia by overloading the circulation with blood squeezed partly from the muscles and partly from the lungs in a Valsalva maneuver.

This is probably not a major factor since a similar circulatory response follows subconvulsive electroshock.

Furthermore, blood pressure and heart rate rise significantly after a paralyzing dose of d-tubocurare, and in our patients, after succinylcholine. Holmberg et al. reported a decrease in the degree of hypertension and tachycardia when succinylcholine was administered with thiopental. They injected variable amounts of thiopental, however, and it is possible that their results were due to different levels of anesthesia.

Elevations of arterial carbon dioxide and depression of oxygen content in blood during shock therapy may affect the circulation but oxygenation of patients before treatment does not appreciably alter the circulatory response.

Dundee concluded from data obtained by Prime and Gray that thiopental probably depresses the vasomotor center. Thiopental reduces the vasomotor response to hypotension caused by increased intrapulmonary pressure. It will inhibit atrial and ventricular arrhythmias produced by direct electrical stimulation of the diencephalon and brain stem in cats paralyzed by succinylcholine. Thiopental may block the circulatory reaction to electroshock by inhibiting cortical impulses which stimulate medullary or hypothalamic centers, or perhaps by depressing these centers directly. Probably the bradycardia which follows tachycardia and hypertension is due to stimulation of the carotid baroreceptors. Brown, Brown and Hines believe that arrhythmias are due to simultaneous sympathetic and parasympathetic stimulation.

The degree of modification of the circulatory response to electroshock is most likely a function of the depth of narcosis. Kiersey, Bickford and Faulconer have shown that the electroencephalogram may be depressed to a flat line if enough thiopental is given. At this level of anesthesia cortical influences upon the circulation are probably minimal. The circulatory response to electroshock will depend upon the relative strength of the electrical stimulus versus the level of anesthesia as well as on the condition of the circulatory system prior to treatment.

The short duration of electroshock makes it possible to treat a large number of patients without mishap. However, modification of the circulatory response by deeper anesthesia might be useful in the patient with cardiovascular disease. The longer period of apnea and of unconsciousness necessitating greater posttreatment care as well as other hazards of thiopental, as outlined by Dundee, must be considered before planning to use a larger dose of thiopental. In practice we have been able to adjust the dose and timing of the thiopental to suit the needs of the individual patient.

**Summary**

The modification by thiopental of circulatory changes following electroshock therapy was studied in 8 patients. Comparisons were made...
of the degree of hypertension and tachycardia resulting from electroshock after 100 mg. or 500 mg. of thiopental had been injected.

Increasing the dose of thiopental significantly reduced the severity of the circulatory reaction stimulated by electroshock therapy. We believe that thiopental blocks this response by interfering with the central sympathetic outflow.

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REFERENCES


