The Effects of Short-acting Barbiturates on Arterial Pressure, Preganglionic Sympathetic Activity and Barostatic Reflexes

Per Skovsted, M.D.,* M. L. Price, A.B., Henry L. Price, M.D.†

The effects of two short-acting barbiturates (thiopental and methohexital) on arterial blood pressure, cervical preganglionic sympathetic activity, and barostatic reflexes have been studied in cats anesthetized with nitrous oxide. Both barbiturates reduced sympathetic nervous activity while failing to extinguish the reflex responses to electrical stimulation of the aortic depressor nerve. In decerebrated animals the same response was seen. Baroreceptor-denervated animals had a similar but exaggerated reaction. Spinal animals developed arterial hypertension without depression of sympathetic activity. It is concluded that the barbiturates inhibit sympathetic nervous activity by inhibiting “pressor” neurons in the medulla oblongata. The medullary “depressor” neurons are relatively unaffected and the barostatic reflexes consequently remain, although they are weakened. The preservation of these reflexes accounts, in part, for the circulatory stability observed during barbiturate anesthesia. (Key words: Barbiturates; Arterial pressure; Sympathetic nervous activity; Barostatic reflexes.)

There is evidence that the decreases in arterial pressure and myocardial contractile force following injection of thiopental (Pentothal) are enhanced by sympathetic block and counteracted by hypoxia, suggesting the presence of sympathetic tone and active barostatic and chemoreceptor reflexes during thiopental anesthesia.

On the other hand, the cardiovascular response to positive-pressure ventilation under thiopental anesthesia has been taken to indicate an absence of normal barostatic reflexes and cardiovascular depression after local injection of thiopental into the posterior hypothalamus offers evidence that central sympathetic centers are depressed by the drug. Also, the hypertension ordinarily produced by electroshock therapy (ECT) is diminished by thiopental.

Methohexital has been studied less extensively than thiopental but is considered to have similar cardiovascular effects. In this study direct measurements of sympathetic nervous activity were made in order to investigate the effects of methohexital and thiopental on sympathetic nervous activity and barostatic reflexes.

Methods

Subjects of the experiments were 33 cats weighing 1.4 to 3.9 kg. They were initially anesthetized with halothane (Fluothane) in oxygen by mask while a femoral artery, a vein and the trachea were cannulated. Halothane was then discontinued, and 20 mg gallamine (Flaxedil) given intravenously at once and again every half hour throughout the experiment. Respiration was controlled with a Harvard animal pump delivering 50 per cent nitrous oxide and 50 per cent oxygen. Total flow to the respirator was 4 l/min. A five-liter reservoir bag with pop-off valve was interposed in front of the Harvard respirator to permit escape of excess gas.

Sympathetic activity was recorded from teased strands of preganglionic fibers of the left cervical sympathetic trunk (divided just below the superior cervical ganglion). Details of the method have been described. The left aortic depressor nerve was divided low in the neck and the central end placed on plati-
<table>
<thead>
<tr>
<th>Sympathetic frequency (imp/sec)</th>
<th>Initial</th>
<th>Minimum</th>
<th>Per cent Depression</th>
<th>5-minute</th>
<th>10-minute</th>
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<th>Per cent Depression</th>
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num electrodes and stimulated at intervals with an AEL stimulator and a Grass isolation transformer. Stimulus characteristics were 0.02 to 1.0 volts, 5-msec pulse duration, 200 cps, and 15-second train length. The method for selecting the voltage used has been described. When the left aortic depressor nerve was unresponsive to stimulation the right was used instead.

To test the baroreflex reflex initially an intravenous injection of ephinephrine (2-5 μg) was used. Only fibers showing inhibition during the increase in blood pressure were studied further; unresponsive fibers were laid aside and new ones selected.

In all cats considered “normal” both vagi, the sinus nerves and, usually, the right aortic depressor nerve were left intact. In all cats described as “baroreceptor-denervated” both vagi and the aortic depressor nerves were divided; either the carotid sinus nerves were divided or ligatures were tied above and below the sinus bulbs. In either case baroreflex reflexes were abolished. Decerebrated cats were prepared by dividing the brain stem between the colliculi under halothane anesthesia with the animals’ heads in a stereotaxic frame, under direct vision. When typical decerebrate rigidity occurred, halothane was discontinued and gallamine given intravenously as described previously. “Spinal” cats underwent cord section at C1-2 under direct vision, after laminectomy. Blood pressure in the spinal cats was maintained above 70 mm Hg with the aid of a continuous infusion of norepinephrine (5 μg/ml 5 per cent glucose solution) delivered by a Harvard infusion pump at a rate of 0.08 to 1.9 ml/min. The spinal cats also received a basal anesthetic of 50 per cent nitrous oxide and gallamine.

Arterial pressure was sensed by a Statham P-23D transducer and recorded on a Grass...
recorder. Mean pressures were obtained by electrical damping. End-tidal $P_{CO_2}$ measured continuously by a Goddard capnograph (corrections were made for the spectral absorption caused by $N_2O$), was kept as close as possible to 5.4 per cent. Arterial blood samples (3 ml) were drawn before the administration of methohexital or thiopental and analyzed in an Instrumentation Laboratory for $pH$, $P_{CO_2}$ and $P_{O_2}$. Blood withdrawn was replaced with an equal volume of physiologic saline solution. Measured $P_{O_2}$ exceeded 100 mm Hg in all samples. Metabolic acidosis, if present, was corrected with NaHCO$_3$ on the basis of base deficit x kg body weight x 0.3 mEq.

Rectal temperature was measured with a Yellow Springs thermistor and maintained at 37–38°C with the aid of a “k-pad” (Gorman-Rupp).

Prior to the administration of the barbiturate each cat was exposed briefly to an inhalational anesthetic (halothane, methoxyflurane, cyclopropane or fluroxene). The following times were allowed for elimination of the anesthetics before giving barbiturates: methoxyflurane, 90 minutes; halothane, 45 minutes; fluroxene and cyclopropane, 30 minutes. Cats 1 to 5 were given both thiopental and methohexital; methohexital was given 30 minutes after the thiopental. The other 28 cats received either thiopental or methohexital. Methohexital was given intravenously in a dose of 2.5 mg/kg, thiopental in a dose of 5.0 mg/kg. These doses were selected because each produced moderate, but not excessive, depression of arterial pressure.

The circulatory response to depressor nerve stimulation has been expressed as the maximal per cent depression in mean arterial pressure during the 15-second stimulation period. The response of sympathetic activity was expressed differently, as the percentage of impulses missing during the time of stimulation: that is, the area of the prevailing sympathetic activity-time curve deleted by stimulation. This, we feel, is more representative than the peak response, since sympathetic activity often failed to remain inhibited during the entire stimulation, and measuring only the peak change would give undue weigh to the first few sec-
BARBITURATES AND SYMPATHETIC ACTIVITY

TABLE 2. Effects of Methohexital on Sympathetic Frequency and Mean Arterial Blood Pressure in Denervated Cats

<table>
<thead>
<tr>
<th>Sympathetic frequency</th>
<th>Initial</th>
<th>Minimum</th>
<th>Percent Depression</th>
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<th>10-minute</th>
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Mean arterial blood pressure (mABP) (mm Hg)

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<tr>
<td>Cat 11</td>
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Results

Methohexital (24 Cats)

The responses of sympathetic impulse frequency and mean arterial pressure in "normal" cats are shown in Table 1. Within a minute after the injection of methohexital, sympathetic frequency fell from 39.8 ± 10.6 imp/s to 10.8 ± 1.9 imp/s (P < 0.001) and mean arterial blood pressure from 120.8 ± 7.4 mm Hg to 91.9 ± 7.8 mm Hg (P < 0.001). Blood pressure invariably reached a minimum at the time of maximal depression of sympathetic activity; in no cat studied with methohexital or thiopental did the blood pressure decline precede that in sympathetic activity. After five minutes arterial pressure returned to the initial level while sympathetic activity, although slightly depressed, was not significantly different from the initial level. This was true also for the ten-minute values; although we extended the observation period to 15 minutes, no further change was seen after the tenth minute. Figure 1 shows a typical response.

The responses of sympathetic frequency and mean arterial blood pressure in baroreceptor-denervated cats are shown in Table 2. Sympathetic frequency fell initially from 66.8 ± 12.4 imp/s to 8.8 ± 1.5 imp/s (P < 0.01) and mean arterial pressure, from 125.0 ± 7.4 mm Hg to 67.8 ± 7.4 mm Hg. After five minutes mean arterial pressure returned to a level not significantly different from the initial level (118.1 ± 12.1 mm Hg), but sympathetic frequency, although increased (8.8 ± 1.5 to 41.8 ± 11.0), was still significantly below the ini-
The effect of methohexital on sympathetic frequency in spinal cats was tested on ten occasions in each of three animals (cats 22–24). When the infusion of norepinephrine was rapid enough to prevent any major fall in arterial pressure following the injection of methohexital no effect on sympathetic activity was seen. On the other hand, a greater decrease in blood pressure evoked an increase in sympathetic activity (see fig. 2). No depression of sympathetic activity similar to that seen in normal, decerebrated or baroreceptor-denervated animals was observed in any spinal cats.

**THIOPENTAL (14 CATS)**

The responses of sympathetic frequency and mean arterial blood pressure to thiopental in normal cats are shown in table 5.

<table>
<thead>
<tr>
<th>Table 3. Effects of Methohexitlon on Sympathetic Frequency and Mean Arterial Blood Pressure in Decerebrate Cats</th>
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<tbody>
<tr>
<td><strong>Sympathetic frequency (imp/sec)</strong></td>
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<tr>
<td>Cat 20</td>
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<tr>
<td>Cat 21</td>
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<td><strong>SE</strong></td>
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<table>
<thead>
<tr>
<th><strong>Mean arterial blood pressure (MAP) (mm Hg)</strong></th>
<th><strong>Initial</strong></th>
<th><strong>21-minute</strong></th>
<th><strong>5-minute</strong></th>
<th><strong>10-minute</strong></th>
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tial level ($P < 0.02$). After ten minutes sympathetic activity was 50.6 ± 11.0, barely but still significantly different ($P < 0.05$) from the initial level.

In order to compare the decreases in sympathetic activity and arterial blood pressure in normal cats with those in baroreceptor-denervated cats we have expressed the decreases as per cent depression from the initial level. In the denervated cats the decreases in both sympathetic activity and blood pressure were significantly greater ($P < 0.02$) than in normal animals.

Table 3 shows the effects of methohexital on sympathetic frequency and mean arterial blood pressure in three decerebrated cats. Although the number of observations is too small for statistical analysis, it appears that decerebrated cats had the same reaction to methohexital as normal cats.

Table 4 shows the effects on sympathetic activity and blood pressure of a 15-second period of electrical stimulation of the aortic depressor nerve, and the modification of these responses by methohexital. Neither response was significantly affected by the barbiturate.

| Table 4. Effects of Methohexital on the Responses of Mean Arterial Blood Pressure and Sympathetic Frequency to Aortic Depressor-nerve Stimulation |
|-----------------------------------------------|------------|-------------|--------------|
| **Sympathetic frequency response (per cent depression)** | **Initial** | **21-minute** | **5-minute** | **10-minute** |
| Cat 10 | 20.6 | 50.0 | 30.0 | 60.0 |
| Cat 11 | 33.6 | 15.4 | 44.0 | 50.0 |
| Cat 12 | 62.9 | 9.1 | 61.9 | 70.5 |
| Cat 13 | 27.8 | 45.5 | 34.5 | 50.0 |
| Cat 15 | 44.0 | 65.2 | 67.5 | 64.3 |
| Cat 18 | 27.9 | 18.8 | 25.0 | 20.0 |
| **Mean** | 36.1 | 34.0 | 43.8 | 52.6 |
| **SE** | 6.2 | 9.2 | 7.1 | 7.3 |

<table>
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<tr>
<th><strong>Mean arterial blood pressure response (per cent depression)</strong></th>
<th><strong>Initial</strong></th>
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Barbiturates and Sympathetic Activity

Sympathetic activity decreased from $46.7 \pm 14.9$ imps/sec to $9.8 \pm 2.7$ imps/sec ($P < 0.05$), and mean arterial blood pressure from $134.8 \pm 9.0$ mm Hg to $92.0 \pm 6.8$ mm Hg. In contrast to methohexital, thiopental produced an initial (5–7 sec) increase in arterial pressure before the decline began; this increase was not preceded by or associated with any increase in sympathetic nervous activity. The magnitude of the arterial pressure rise was roughly 20 mm Hg.

The responses of baroreceptor-denervated cats are shown in Table 6. Sympathetic activity fell from $95.3 \pm 32.3$ imps/sec to $14.2 \pm 8.3$ imps/sec ($P < 0.05$) and, as with methohexital, it returned to a level somewhat lower than the initial level ($61.3 \pm 30.0$ imps/sec at 15 min). Mean arterial pressure showed a small initial increase in half the cats and then a decrease to $106.0 \pm 13.1$ mm Hg ($P < 0.01$), with return to $153.3 \pm 16.5$ mm Hg at five minutes. As with methohexital, the fall in sympathetic activity was significantly higher in the “baroreceptor-denervated” than in the normal group, and this was true for the blood pressure decline as well.

The effect of thiopental on spinal cats was tested on three occasions in each of two animals. Here, also, there was an initial rise in arterial pressure which sometimes was associated with a slight and very brief (5–7 sec) decline in sympathetic activity, after which sympathetic activity returned to normal or increased, depending on the magnitude of blood pressure decline, as with methohexital.

The effects of a 15-second period of baroreceptor-nerve stimulation in five cats which responded well to such stimulation are shown in Table 7. Although it was present in five of six animals, the response after 2½ minutes was significantly depressed.

Discussion

Since methohexital and thiopental affected sympathetic activity and mean arterial pressure similarly (except for the brief thiopental-induced increase in blood pressure) they are discussed together as “barbiturates.” Since no effect of the barbiturates on the isolated spinalcord preparation could be shown, and since decerebrate cats reacted like normal animals, we assume that the main locus for barbiturate action on the sympathetic nervous system is within the brain stem, probably in the vasomotor center of the medulla oblongata. Traditionally, this has been considered to be com-
Table 5. Effects of Thiopental on Sympathetic Frequency and Mean Arterial Blood Pressure in Normal Cats

<table>
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<td>33.5</td>
<td>39.3</td>
<td>42.2</td>
<td></td>
</tr>
<tr>
<td><strong>SE</strong></td>
<td>14.9</td>
<td>2.7</td>
<td>12.2</td>
<td>13.6</td>
<td>14.8</td>
<td></td>
</tr>
</tbody>
</table>

|                  |         |         |         |          |           |           |
| **Mean arterial blood pressure (MABP) (mm Hg)** |         |         |         |          |           |           |
| Cat 1            | 112     | 154     | 80      | 130      | 95        | 94        |
| Cat 2            | 132     | 142     | 94      | 132      | 130       | 132       |
| Cat 3            | 130     | 135     | 96      | 135      | 130       | 130       |
| Cat 4            | 150     | 170     | 112     | 145      | 150       | 150       |
| Cat 5            | 115     | 140     | 104     | 115      | 115       | 115       |
| Cat 25           | 170     | 182     | 66      | 115      | 125       | 130       |
| **Mean**         | 134.8   | 153.8   | 92.0    | 128.7    | 125.2     | 125.2     |
| **SE**           | 9.0     | 7.6     | 6.8     | 4.8      | 7.0       | 7.7       |

posed of tonically active "pressor" neurons and "depressor" neurons active only upon receipt of impulses from peripheral stretch receptors located in major blood vessels. The "depressor" neurons function to hold the "pressor" neurons in check, thus providing the negative feedback which enables the reflex to operate.

In baroreceptor-denervated animals the medullary "pressor" neurons are uninhibited and fire at an elevated rate. From our data we conclude that the medullary pressor center is depressed by barbiturates. The depressor center may be depressed also, since the 2½-minute response to baroreceptor-nerve stimulation was reduced after thiopental; that found after methohexital showed a return toward normal, but it was still depressed in half the cats; if the depressor center were unaffected by methohexital an enhanced response to baroreceptor-nerve stimulation could have been expected. However, we did not find this.

That the barostatic reflexes are active even at the time of maximal depression of sympathetic activity and blood pressure (about 1½ min before the first baroreceptor-nerve stimulation) can be concluded from the finding that reductions in both sympathetic activity and blood pressure were significantly greater in baroreceptor-denervated cats than in normal cats. This suggests that normally some compensatory increase in sympathetic activity is induced by the hypotension. Obviously, however, this was not enough to compensate fully for the marked depression of the medullary vasomotor center caused by the direct action of the barbiturates. These findings complement and extend those of Bendixen and Laver.¹

That the depression in sympathetic activity is causative, not a coincidental, factor in the blood pressure decline is concluded from three findings: 1) the sympathetic decline always preceded or mimicked the fall in arterial pressure; 2) baroreceptor-denervated cats showed greater reductions in sympathetic activity and blood pressure than normal cats; 3) even when sympathetic activity and blood pressure were depressed baroreceptor-nerve stimulation pro-
### Table 6. Effects of Thiopental on Sympathetic Frequency and Mean Arterial Blood Pressure in Baroreceptor-denervated Cats

<table>
<thead>
<tr>
<th>Sympathetic frequency (imps/sec)</th>
<th>Initial</th>
<th>Maximum</th>
<th>Minimum</th>
<th>5-minute</th>
<th>10-minute</th>
<th>15-minute</th>
</tr>
</thead>
<tbody>
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<td>80</td>
<td>2</td>
<td>14</td>
<td>20</td>
<td>24</td>
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</tr>
<tr>
<td>Cat 27</td>
<td>240</td>
<td>55</td>
<td>150</td>
<td>150</td>
<td>150</td>
<td>150</td>
</tr>
<tr>
<td>Cat 28</td>
<td>120</td>
<td>1</td>
<td>20</td>
<td>40</td>
<td>42</td>
<td></td>
</tr>
<tr>
<td>Cat 29</td>
<td>75</td>
<td>7</td>
<td>21</td>
<td>40</td>
<td>40</td>
<td></td>
</tr>
<tr>
<td>Cat 30</td>
<td>36</td>
<td>12</td>
<td>35</td>
<td>40</td>
<td>40</td>
<td></td>
</tr>
<tr>
<td>Cat 31</td>
<td>21</td>
<td>8</td>
<td>11</td>
<td>14</td>
<td>15</td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>95.3</td>
<td>14.2</td>
<td>42.0</td>
<td>55.7</td>
<td>61.3</td>
<td></td>
</tr>
<tr>
<td>SE</td>
<td>32.3</td>
<td>8.3</td>
<td>21.9</td>
<td>25.3</td>
<td>30.0</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Mean arterial blood pressure (MAP) (mm Hg)</th>
<th>Initial</th>
<th>Maximum</th>
<th>Minimum</th>
<th>5-minute</th>
<th>10-minute</th>
<th>15-minute</th>
</tr>
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<td>190</td>
<td>122</td>
<td>200</td>
<td>193</td>
<td>180</td>
</tr>
<tr>
<td>Cat 27</td>
<td>170</td>
<td>170</td>
<td>136</td>
<td>165</td>
<td>155</td>
<td>155</td>
</tr>
<tr>
<td>Cat 28</td>
<td>155</td>
<td>200</td>
<td>100</td>
<td>140</td>
<td>174</td>
<td>174</td>
</tr>
<tr>
<td>Cat 29</td>
<td>200</td>
<td>200</td>
<td>116</td>
<td>190</td>
<td>180</td>
<td>174</td>
</tr>
<tr>
<td>Cat 30</td>
<td>148</td>
<td>149</td>
<td>117</td>
<td>136</td>
<td>150</td>
<td>156</td>
</tr>
<tr>
<td>Cat 31</td>
<td>95</td>
<td>100</td>
<td>45</td>
<td>90</td>
<td>121</td>
<td>115</td>
</tr>
<tr>
<td>Mean</td>
<td>163.0</td>
<td>168.2</td>
<td>106.0</td>
<td>153.5</td>
<td>162.2</td>
<td>158.0</td>
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<td>15.8</td>
<td>13.1</td>
<td>16.5</td>
<td>10.5</td>
<td>9.5</td>
</tr>
</tbody>
</table>

### Table 7. Effects of Thiopental on the Response of Mean Arterial Blood Pressure and Sympathetic Frequency to Aortic Depressor-nerve Stimulation

<table>
<thead>
<tr>
<th>Sympathetic frequency response (per cent depression)</th>
<th>Initial</th>
<th>21-minute</th>
<th>5-minute</th>
<th>10-minute</th>
<th>15-minute</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cat 26</td>
<td>34.6</td>
<td>0.0</td>
<td>15.0</td>
<td>18.8</td>
<td>30.4</td>
</tr>
<tr>
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<td>8.3</td>
<td>13.3</td>
<td>16.7</td>
<td>16.7</td>
</tr>
<tr>
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<td>20.0</td>
<td>80.0</td>
<td>83.3</td>
<td>85.7</td>
</tr>
<tr>
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<td>18.8</td>
<td>25.0</td>
<td>20.0</td>
<td>25.0</td>
</tr>
<tr>
<td>Cat 31</td>
<td>22.6</td>
<td>14.3</td>
<td>17.1</td>
<td>33.3</td>
<td>33.3</td>
</tr>
<tr>
<td>Cat 32</td>
<td>12.9</td>
<td>4.1</td>
<td>11.1</td>
<td>12.8</td>
<td>12.8</td>
</tr>
<tr>
<td>Mean</td>
<td>32.8</td>
<td>10.9</td>
<td>26.9</td>
<td>30.8</td>
<td>34.0</td>
</tr>
<tr>
<td>SE</td>
<td>10.1</td>
<td>3.3</td>
<td>10.8</td>
<td>10.9</td>
<td>10.8</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Mean arterial blood pressure response (per cent depression)</th>
<th>Initial</th>
<th>21-minute</th>
<th>5-minute</th>
<th>10-minute</th>
<th>15-minute</th>
</tr>
</thead>
<tbody>
<tr>
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<td>0.0</td>
<td>9.5</td>
<td>13.5</td>
<td>13.5</td>
</tr>
<tr>
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<td>1.8</td>
<td>3.7</td>
<td>4.5</td>
<td>6.4</td>
</tr>
<tr>
<td>Cat 28</td>
<td>28.1</td>
<td>7.5</td>
<td>21.4</td>
<td>26.5</td>
<td>27.3</td>
</tr>
<tr>
<td>Cat 29</td>
<td>11.3</td>
<td>4.0</td>
<td>5.0</td>
<td>7.9</td>
<td>7.4</td>
</tr>
<tr>
<td>Cat 31</td>
<td>12.3</td>
<td>3.6</td>
<td>7.4</td>
<td>9.6</td>
<td>10.5</td>
</tr>
<tr>
<td>Cat 32</td>
<td>25.7</td>
<td>10.9</td>
<td>30.0</td>
<td>28.6</td>
<td>30.9</td>
</tr>
<tr>
<td>Mean</td>
<td>16.8</td>
<td>4.6</td>
<td>12.8</td>
<td>15.1</td>
<td>16.0</td>
</tr>
<tr>
<td>SE</td>
<td>3.6</td>
<td>1.6</td>
<td>4.3</td>
<td>4.1</td>
<td>4.3</td>
</tr>
</tbody>
</table>
duced an additional decrease in sympathetic activity, which was followed also by a decline in arterial pressure. This last observation indicates that the vascular smooth muscle is reactive to sympathetic impulses under barbiturate anesthesia, and that sympathetic activation could have affected peripheral resistance, had it occurred.

The clinical implication of our findings is that an extraordinary degree of hypotension is to be expected in individuals relying on high sympathetic tone to maintain sufficient cardiovascular function (e.g., hypovolemic shock and heart failure), as suggested in 1943 by Halford. This should also be true for individuals with weak barostatic reflexes, as in orthostatic hypotension. Our findings fail to provide a basis for the warning of Marc-Aurele et al., who cautioned against using short-acting barbiturates where reflexly induced hyperscretion of catecholamines could occur, as in patients with pheochromocytomata. Presumably, the effect of thiopental in such cases is not reflex but is exerted upon the adrenal medulla by some direct action of the barbiturate.

References


Drugs

LIVER DAMAGE An increase in the serum level of the enzyme ornithine carbamoyl transferase (S-OCT) is a specific and sensitive sign of hepatic damage. Increased protein catabolism leads to increased deamination, which requires greater production of this enzyme by the liver. When the catabolic process subsides, the surplus enzymes are discharged into the serum, where the level increases. S-OCT has been shown to increase following administration of chloroform, carbon tetrachloride, and alcohol. Patient studies demonstrated that: 1) halothane anesthesia for angiography produced no change in enzyme level; 2) halothane anesthesia for surgical operation was followed by an increase in S-OCT for several days; 3) surgical operation with the patient under spinal anesthesia, without a decrease in blood pressure, was followed by a slight increase in S-OCT, but no change in SGOT or SGPT; 4) spinal anesthesia with a decrease in blood pressure was followed by a marked rise in S-OCT. It is concluded that halothane does not affect hepatic function. When surgical operation is carried out with the patient under halothane or spinal anesthesia, S-OCT increases, probably because of increased breakdown of protein. A reduction in blood pressure, leading to reduced perfusion of the liver, also causes a rise in S-OCT, particularly in patients more than 50 years of age. (Brohult, J., and Gillquist, J.: Serum Ornithine Carbamoyl Transferase Activity in Man After Halothane Anaesthesia and Spinal Anaesthesia with and without Systolic Blood Pressure Fall, Acta Chir. Scand. 135: 113 (No. 2) 1969.)