Title: EFFECT OF ELECTROCONVULSIVE THERAPY ON PLASMA BETA ENDORPHIN

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Introduction. Electroconvulsive therapy (ECT) has been reported to produce an opiate-like catalepsy and analgesia in rats that can be attenuated by naltrexone pre-injection. Naloxone also attenuates the cardiovascular response to ECT in rats. These studies suggest a functional release of endogenous opioids during ECT. Moreover, they suggest that endorphins might play a role in the mechanism of action of ECT in man. The purpose of this study was to assess the effect of ECT on plasma endorphin and cardiovascular response in man.

Methods. After approval of the study protocol by the Human Subjects Committee and informed consent for study participants, patients scheduled for ECT were analyzed. All patients were pre-medicated with 0.5 mg atropine IM. Patients were induced with methohexital 40-60 mg and succinylcholine 30-40 mg IV. ECT was administered with 120 volts AC administered over 0.5 sec. Blood for plasma beta endorphin was drawn following the following procedures: (1) After placement of an intravenous line, (2) after induction of anesthesia, (3) five minutes after ECT, and (4) at discharge from the recovery room. Each time blood was drawn for plasma beta endorphin, heart rate (HR), systolic blood pressure (SBP), and diastolic blood pressure (DBP) were recorded. Rate-pressure product (RPP) was calculated as the product of HR and SBP. Plasma beta endorphin was quantitated with a New England Nuclear radioimmunoassay kit. Analysis of variance with Duncan's Multiple Range test was used to look for differences between observations of the dependent variables, and significance was defined as p<0.05.

Results. Eight ECT treatments were studied in 3 patients, average age 65 years, average weight 72.4 kg. Results are summarized in Table. No significant changes in HR, SBP, DBP, RPP or plasma beta endorphin occurred as a result of induction of anesthesia (p>0.05). After application of ECT, systolic blood pressure increased from 111±6 (SEM) torr to 156±13 torr (p<0.05), RPP increased from 9423±935 torr/min to 15200±1558 torr/min (p<0.05), and plasma beta endorphin increased from 29.2±2.1 pg/ml to 48.6±5.7 pg/ml (p<0.05, see figure). Both HR and DBP increased after ECT, but the change was not statistically significant (p>0.05). At discharge from the recovery room, HR had increased from 96.5±3.8 after ECT to 104.7±10.8, which was significantly higher than both pre-ECT values (p<0.05). SBP and RPP were significantly decreased after recovery from the post-ECT values. Plasma beta endorphin levels remained significantly elevated from pre-ECT values at discharge from the recovery room (p<0.05, see figure). Over the study time course, plasma beta endorphin level was significantly correlated with HR (R=0.43, p=0.01), SBP (R=0.70, p=0.001), and RPP (R=0.75, p=0.001).

Discussion. This study demonstrates a release of plasma beta endorphin during ECT in humans. A correlation of plasma beta endorphin to cardiovascular stress indicators has also been demonstrated. Since naloxone attenuates the cardiovascular response to ECT in rats, plasma beta endorphin or other endogenous opioids may be involved in the regulation of cardiovascular function after activation by stress. Since the post-ECT opiate-like catalepsy and analgesia in rats is also blocked by naloxone, a possible function for beta endorphin in the action of ECT in man is suggested. Long-term studies examining the effect of a series of ECT treatments on plasma beta endorphin are in progress.

### Table: Effect of ECT on Plasma Beta Endorphin, Blood Pressure, and Heart Rate

<table>
<thead>
<tr>
<th></th>
<th>1/1min</th>
<th>3/3.8</th>
<th>83.8±4.4</th>
<th>96.5±3.8</th>
<th>104.7±10.8</th>
</tr>
</thead>
<tbody>
<tr>
<td>SBP (torr)</td>
<td>114±6</td>
<td>111±6</td>
<td>156±13b</td>
<td>117±13</td>
<td>156±13</td>
</tr>
<tr>
<td>DBP (torr)</td>
<td>76±5</td>
<td>76±5</td>
<td>89±6</td>
<td>90±1</td>
<td>90±1</td>
</tr>
<tr>
<td>RPP* (torr/min)</td>
<td>9928±837</td>
<td>9426±935</td>
<td>15200±1558</td>
<td>12140±161</td>
<td>12140±161</td>
</tr>
<tr>
<td>β-END (pg/ml)</td>
<td>27.2±3.7</td>
<td>29.2±1.2</td>
<td>48.6±5.7a</td>
<td>44.7±5.7a</td>
<td>44.7±5.7a</td>
</tr>
</tbody>
</table>

* RPP = Rate pressure product = HR x SBP
a Significantly different from before induction and before ECT (p<0.05)
b Significantly different from before induction, before ECT, and after recovery (p<0.05)

Effect of Electroconvulsive Therapy on Plasma β-Endorphin

![Graph showing the effect of ECT on plasma beta endorphin levels](image)

*Significantly different from before induction and before ECT, p<0.05

References:
2. Belenky GL and Holaday JW: The opiate antagonist naloxone modifies the effects of electroconvulsive shock (ECS) on respiration, blood pressure, and heart rate. Brain Res 177:414-417, 1979