SHORT COMMUNICATION

ROLE OF TESTOSTERONE ON PAIN THRESHOLD IN RATS

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Abstract: Pain thresholds were recorded in rats by progressive increase in electrical stimulation to induce tail withdrawal, vocalisation and vocalisation after discharge. It was observed that castration resulted in significant reduction of pain threshold which however returned to normal level on substitution with testosterone therapy.

Key words: pain threshold
rat
castration
testosterone

INTRODUCTION

Perception of pain in animals is known to be modulated by variety of factors such as immobilization, restriction of food intake and exposures to extremes of environmental temperature (1-3).

The present work is aimed to study effect of castration and testosterone replacement on threshold for nociceptive responses.

METHODS

The study was carried out in 16 male albino rats (100 to 150 days old, b.w. 200 to 250 g) housed individually in separate plastic cages with food and water available ad libitum. The experimental protocol consisted of restraining the animal for 30 min in a medium size talcum powder tin cut into two halves longitudinally to accommodate the animal. The head and the tail of the animal were kept outside the tin on either side by modifications at both ends of the tin (4). Two stainless steel needles (gauge 30) were inserted intracutaneously in the middle of the tail. Electrical stimulation consisted of a train of one sec duration of 100 Hz and pulse width 1 msec (5). The voltage was progressively increased in steps of 0.1 volt. An interval of 5 min was kept between two successive electrical shocks in the same animal.

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RESULTS

Castration significantly decreased the threshold of the three responses studied (i.e. tail withdrawal, vocalization and vocalization after discharge). Replacement therapy with testosterone in three castrated animals showed no significant deviation of response in relation to precastrated values (Table I).

DISCUSSION

Perception of pain is modulated in part by endogenous opioids located in brain and spinal cord. Beta-endorphin (an endogenous opioid peptide) appears to influence nociception at spinal and supraspinal levels (6). It has been shown by Tseng et al that injection of beta-endorphin intrathecally and/or into the ventricles increased the threshold to nociceptive stimulus (6). It is, therefore, suggested that steroids modulate nociceptive response by affecting beta-endorphin levels.

Gonadal steroids may also modulate the activity of neurotransmitters and other neuropeptides participating in analgesic response.

TABLE I: Nociceptive stimulus in volts (mean ± SE) for eliciting nociceptive responses (A, B, C) in control (I) and experimental groups (II & III).

<table>
<thead>
<tr>
<th></th>
<th>Tail withdrawal</th>
<th>Vocalisation</th>
<th>Vocalisation after discharge</th>
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<tbody>
<tr>
<td>(a)</td>
<td></td>
<td>(b)</td>
<td>(c)</td>
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<tr>
<td>I</td>
<td></td>
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<tr>
<td>Control group (n=16)</td>
<td>0.57 ± 0.06</td>
<td>0.8 ± 0.09</td>
<td>1.29 ± 0.11</td>
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<td>II</td>
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<td>Castrated group (n=8)</td>
<td>0.3 ± 0.05*</td>
<td>0.37 ± 0.07*</td>
<td>0.49 ± 0.05*</td>
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<td>III</td>
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<tr>
<td>Castrated group with testosterone replacement (n=8)</td>
<td>A 0.55 ± 0.128</td>
<td>NS</td>
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14 days (A), 21d (B) & 28d (C) after testosterone replacement.

*P<0.05; NS = Not significant

Statistical analysis was done by paired/unpaired test.

REFERENCES