Spinal proerectile effect of oxytocin in anesthetized rats

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Spinal proerectile effect of oxytocin in anesthetized rats. Am J Physiol Regulatory Integrative Comp Physiol 280: R1870–R1877, 2001.—The spinal cord contains the neural network that controls penile erection. This network is activated by information from peripheral and supraspinal origin. We tested the hypothesis that oxytocin (OT), released at the lumbosacral spinal cord level by descending projections from the paraventricular nucleus, regulated penile erection. In anesthetized male rats, blood pressure and intracavernous pressure (ICP) were monitored. Intrathecal (it) injection of cumulative doses of OT and the selective OT agonist [Thr4,Gly7]OT at the lumbosacral level elicited ICP rises whose number, amplitude, and area were dose dependent. Thirty nanograms of OT and one-hundred nanograms of the agonist displayed the greatest proerectile effects. Single injections of OT also elicited ICP rises. Preliminary injection of a specific OT-receptor antagonist, hexamethonium, or bilateral pelvic nerve section impaired the effects of OT injected it. NaCl and vasopressin injected it at the lumbosacral level and OT injected it at the thoracolumbar level or intravenously had no effect on ICP. The results demonstrate that OT, acting at the lumbosacral spinal cord, elicits ICP rises in anesthetized rats. They suggest that OT, released on physiological activation of the PVN in a sexually relevant context, is a potent activator of spinal proerectile neurons.

urogenital; sexual reflexes; paraventricular nucleus

PHARMACOLOGICAL STIMULATION of the paraventricular nucleus of the hypothalamus (PVN), through a variety of neuroactive compounds in conscious rats and electrical or pharmacological stimulation of the PVN in anesthetized rats, elicits penile erection and intracavernous pressure (ICP) increases (3, 8). The PVN contributes descending oxytocinergic fibers to the spinal cord (7), and the paraventriculospinal tract originates in the paravascular part of the PVN (15). In male rats, the lumbosacral spinal cord contains oxytocinergic fibers (29), some of which synapse onto spinal preganglionic neurons (30). Furthermore, specific oxytocin (OT) binding sites are present in the sacral parasympathetic nucleus and the dorsal grey commissure of the L6-S1 spinal cord (34). Finally, PVN neurons are transsynaptically activated by information from peripheral and supraspinal origins (24). We tested the hypothesis that OT released by paraventriculospinal pathways could elicit ICP rises in anesthetized rats. They suggest that OT, released on physiological activation of the PVN in a sexually relevant context, is a potent activator of spinal proerectile neurons.

MATERIAL AND METHODS

Animals. Adult male Sprague-Dawley rats, sexually naïve and weighing 200–250 g, were purchased from Charles River (Saint-Aubin les Elbeufs, France). Rats were housed in groups of four in plastic cages containing wood-chip bedding. They had free access to commercial pelleted rodent chow (Piètement, Provins, France) and tap water. Cages were placed in an animal facility maintained at 20°C and kept in a 12:12-h light-dark cycle (lights on at 8 AM). All animal experiments were carried out in accordance with the European Economical Community Directive of November 24, 1986 (86/609/EEC) on the use of laboratory animals. All efforts were made to minimize animal suffering and to reduce the number of animals used.

Experimental procedure. Rats were anesthetized with an intraperitoneal injection of urethane (1.2 g/kg in sterile water), and their temperature was maintained at 37°C using a heating pad and monitored by an electronic thermometer. They had free access to commercial pelleted rodent chow (Piètement, Provins, France) and tap water.
intravenous (iv) overdose of urethane. Ten microliters of
mm proximal to the major pelvic ganglion.
lateral aspect of the prostate, and nerves were sectioned 2–3
perform pelvic nerve section (PNx rats), a suprapubic inci-
consecutive injections were separated by a 15-min period. To
0.9%. When we used cumulative injections of drugs, two
compounds dissolved in 10
the different groups of rats that we used. For it injections,
pressure transducer (Elcomatic 750, Glasgow, UK) and inject
washed with heparinized saline (25 U/ml) to record blood pressure (BP) via a
were catheterized with polyethylene tubings filled with hep-
fluid leakage. Rats were tracheotomized to prevent aspira-
saliva and to perform artificial ventilation when muscle
relaxant was used. The carotid artery and jugular vein
were catheterized with polyethylene tubings filled with hep-
arized saline (25 U/ml) to record blood pressure (BP) via a
pressure transducer (Elcomatic 750, Glasgow, UK) and inject
drugs intravenously, respectively. ICP recording was per-
fomed as described previously (12). Tables 1 and 3 display
a Hamilton syringe filled with saline to prevent cerebrospinal
neck muscles and skin layers. The catheter was connected to
rat’s head was placed in a stereotaxic frame and was rotated
ose downwards to facilitate catheter insertion. The cathete-
, a polyethylene tubing (PE-10) stretched to 150% of its
original length in hot water, was cut to the required length so
that its distal opening reached the L4–L6 or T12-T13 levels of
the spinal cord. The skin and neck muscles were incised and
retracted. The atlantooccipital membrane was opened, and the
catheter, flushed with sterile NaCl 0.9%, was carefully
advanced in the caudal direction. Finally, the rostral free end
of the catheter was secured with the ligatures that closed the
neck muscles and skin layers. The catheter was connected to
a Hamilton syringe filled with saline to prevent cerebrospinal
fluid leakage. Rats were tracheotomized to prevent aspira-
tion of saliva and to perform artificial ventilation when muscle
relaxant was used. The carotid artery and jugular vein
were catheterized with polyethylene tubings filled with hep-
arinized saline (25 U/ml) to record blood pressure (BP) via a
pressure transducer (Elcomatic 750, Glasgow, UK) and inject

RESULTS
Cumulative injections of OT. Injection (it) of OT elicited ICP rises (Fig. 1A). Table 2 displays the number
of rats, named responders, that had at least one ICP rise during the recording. Kruskal-Wallis one-way
ANOVA on ranks demonstrated that there was a treatment effect on the number of responders (H = 41.8,
df = 9, P = 3.6 × 10^{-6}). At least one ICP rise was recorded after injection of NaCl, OT at the L4-L6 and
at the T12-T3 levels, OT iv, OT agonist (Fig. 1B), and OT at the L4-L6 level after curarization. In contrast,
the groups that received injection of the OT antagonist
followed by OT at the L4-L6 level, AVP, OT at the
L4-L6 level after PNx, or HXM (P < 0.05 for each)
cluded significantly fewer or no responders. There-
ence, the effects of OT were impaired by the OT antag-
Fig. 1C) by lesioning preganglionic fibers con-
veyed by the pelvic nerve and by blocking the transmission between pre- and postganglionic neu-
rons. AVP had no effect on ICP.

To further search for differences between groups of
responders, we analyzed the number of ICP rises per

Table 1. Effects of drugs, injected either it or iv, on intracavernous pressure of anesthetized male rats

<table>
<thead>
<tr>
<th>Drug</th>
<th>Route/Site</th>
<th>Dose</th>
<th>Number of Rats</th>
</tr>
</thead>
<tbody>
<tr>
<td>NaCl (0.9%)</td>
<td>it L4–L6</td>
<td>8 injections</td>
<td>10</td>
</tr>
<tr>
<td>OT</td>
<td>it L4–L6</td>
<td>0.3, 1, 3, 10, 30, 100, and 300 ng</td>
<td>10</td>
</tr>
<tr>
<td>OT</td>
<td>it T12–T13</td>
<td>0.3, 1, 3, 10, 30, 100, and 300 ng</td>
<td>10</td>
</tr>
<tr>
<td>OT</td>
<td>iv</td>
<td>0.3, 1, 3, 10, 30, 100, and 300 ng</td>
<td>8</td>
</tr>
<tr>
<td>[Thr^{4}, Gly^{7}]OT</td>
<td>it L4–L6</td>
<td>0.5, 1, 3, 10, 30, 100, and 300 ng</td>
<td>8</td>
</tr>
<tr>
<td>AVP</td>
<td>it L4–L6</td>
<td>0.5, 1, 3, 10, 30, 100, and 300 ng</td>
<td>8</td>
</tr>
<tr>
<td>[(S)PMP^{1}, D-Trp^{2}, Pen^{6}, Arg^{8}]OT</td>
<td>it L4–L6</td>
<td>100 ng*</td>
<td>8</td>
</tr>
<tr>
<td>PNx</td>
<td>iv</td>
<td>60 mg/kg*</td>
<td>5</td>
</tr>
<tr>
<td>HMX</td>
<td>iv</td>
<td>30 mg/kg*</td>
<td>5</td>
</tr>
</tbody>
</table>

NaCl (0.9%), sodium chloride; OT, oxytocin; [Thr^{4}, Gly^{7}]OT, oxytocin agonist; [(S)PMP^{1}, D-Trp^{2}, Pen^{6}, Arg^{8}]OT, oxytocin antagonist; AVP, [Arg^{8}]vasopressin; HMX, hexamethonium, nicotinic receptors antagonist; gallamine triethiodide, striated muscle relaxant; PNx, bilateral section of the pelvic nerve; iv, intravenously. *Before cumulative doses of OT intrathecally (it) delivered at the L4–L6 level.
group (Table 2). Kruskal-Wallis one-way ANOVA on ranks demonstrated that there was a treatment effect on the number of ICP rises ($H = 41.5$, df = 9, $P = 4.1 \times 10^{-6}$). Rats that received an injection of NaCl at the L4-L6 level, OT iv, and OT it at the T12-T13 level significantly displayed fewer ICP rises than rats treated with OT it at the L4-L6 level, the OT agonist administered it at the L4-L6 level, or OT it at the L4-L6 level after curarization ($P < 0.05$ for each). In contrast, there was no significant difference among the last three groups.

We also searched for a dose effect of OT or its agonist on the total number of ICP rises (Fig. 2). The response curve of the it OT L4-L6 group was bell shaped (Fig. 2). In this group, there was a statistically significant effect of the dose of OT injected on the number of ICP rises (Friedman repeated-measures ANOVA on ranks, $\chi^2 = 38.8$, df = 7, $P = 2.0 \times 10^{-6}$). Ten and thirty nanograms of OT elicited significantly more ICP rises than the other doses ($P < 0.05$ for each). In the group treated with the OT agonist delivered it at the L4-L6 level, the dose effect was also present (Friedman repeated-measures ANOVA on ranks, $\chi^2 = 27.8$, df = 7, $P = 2.0 \times 10^{-4}$), but the greatest doses used elicited the greatest number of ICP rises. One-hundred nanograms of the OT agonist elicited significantly more ICP rises than vehicle 0.3, 1, 3, and 10 ng ($P < 0.05$ for each).

Fig. 1. Three recordings of blood pressure (BP; top traces) and intracavernous pressure (ICP; bottom traces; in mmHg) in 3 different anesthetized male rats. A: intrathecal injection of 0.9% NaCl (at $t = 0$ min), then each 15 min, increasing doses of oxytocin (OT; 0.3, 1, 3, 10, 30, 100, and 300 ng) were injected by the same route. B: same as A, but injection of the OT agonist [Thr$^4$,Gly$^7$]OT was performed. C: at $t = 15$ min, injection of the OT antagonist [(S)PMP$^1$,D-Trp$^2$,Pen$^6$,Arg$^8$]OT was performed, then same doses of OT as in A.
Table 2. Effects of it and iv injections of OT and other drugs 1) on the number of rats that displayed at least one ICP rise during the experiment (responders) relative to N and 2) on the number of ICP rises per rat in each group

<table>
<thead>
<tr>
<th>Drug</th>
<th>Route/Site</th>
<th>Responders/N</th>
<th>Number of ICP Rises/Rat</th>
</tr>
</thead>
<tbody>
<tr>
<td>NaCl (0.9%)</td>
<td>it L4–L6</td>
<td>8/10</td>
<td>2 ± 2</td>
</tr>
<tr>
<td>OT</td>
<td>it L4–L6</td>
<td>10/10</td>
<td>7 ± 4</td>
</tr>
<tr>
<td>OT</td>
<td>it T12–T13</td>
<td>6/10</td>
<td>2 ± 2*</td>
</tr>
<tr>
<td>OT</td>
<td>iv</td>
<td>6/8</td>
<td>3 ± 3*</td>
</tr>
<tr>
<td>[Thr4, Gly7]OT</td>
<td>it L4–L6</td>
<td>8/8</td>
<td>6 ± 3</td>
</tr>
<tr>
<td>[(S)PMP1, n-2, Trp2, Pen6, Arg8]OT</td>
<td>it L4–L6</td>
<td>1/8*</td>
<td>1 ± 2*</td>
</tr>
<tr>
<td>AVP</td>
<td>it L4–L6</td>
<td>1/8*</td>
<td>1 ± 1*</td>
</tr>
<tr>
<td>PNx</td>
<td>iv</td>
<td>1/8*</td>
<td>1 ± 1*</td>
</tr>
<tr>
<td>Gallamine</td>
<td>iv</td>
<td>0/5*</td>
<td>0*</td>
</tr>
</tbody>
</table>

N, no. of rats in each group; ICP, intracavernous pressure. *Statistically different from OT delivered it at the L4–L6 level (P < 0.05). No. of ICP rises/rat are means ± SE.

significantly more ICP rises than vehicle 0.3, 1, and 3 ng (P < 0.05 for each).

Comparing the effects of OT and its agonist, 10 ng of OT elicited a greater number of ICP rises than 10 ng of the agonist (P = 0.0321). No difference in the number of ICP rises occurred at 30 and 100 ng between OT and its agonist (P = 0.0548 and 0.1584, respectively). Finally, 300 ng of the OT agonist elicited more ICP rises than 300 ng of OT (P = 0.0084).

We further measured the effects of OT and its agonist on the amplitude of the ICP rises, expressed as the ICP/BP ratio. OT (10, 30, and 100 ng) elicited the greatest ICP rises as measured by the ratio ICP/BP (P < 0.05; 1). OT agonist (100 and 300 ng) elicited the greatest ICP rises (P < 0.05; 2).

Furthermore, there was a dose effect on the AUC of the ICP rise, expressed by the AUC/BP ratio (Fig. 4; χ² = 34.0, df = 7, P = 1.6 × 10⁻⁵). OT (10 and 30 ng) elicited significantly greater AUC/BP than the other doses (P < 0.05 for each). In contrast, there was no difference between 10 and 30 ng OT. The OT agonist also yielded a significant dose effect on AUC/BP [F(7,63) = 7.81, P = 2.5 × 10⁻⁶]. One-hundred and three-hundred nanograms of the OT agonist yielded a significantly greater AUC/BP (P < 0.05 for each), although there was no dif-
DISCUSSION

Results of the present study provide evidence for a proerectile effect of OT at the lumbosacral level in anesthetized rats. Delivering OT intravenously had no effect on ICP. OT receptors are present in the reproductive tract of males (11). OT contracts smooth muscles of the genital tract in vivo (20) and contracts the corpus cavernosum in vitro (32). Therefore, one cannot expect a proerectile (relaxant) effect of OT through an effect on a peripheral target.

Delivering OT at the T12-T13 level had no reliable effects on ICP. The thoracolumbar spinal cord contains sympathetic neurons destined to the penis (18). These sympathetic pathways are classically considered antierectile. Therefore, even if OT activated these neurons, the consequence could not be an erection. Proerectile effects should therefore be attributed to a specific targeting of OT on the lumbosacral spinal cord.

This hypothesis was confirmed by the fact that only rats that received OT at its specific agonist, [Thr⁴, Gly⁷]OT, reliably displayed ICP rises. Vasopressin did not elicit such a response. Therefore, ICP rises were elicited by OT and not vasopressin. The presence of ICP rises occurring on it NaCl are likely spontaneous events. Their number is not constant between rats or the rank of injection. However, it suggests that erection may occur spontaneously under anesthesia in rats. After injections of the OT antagonist [(S)PMP⁴, d-Trp², Pen⁶, Arg⁸]OT, followed by OT, ICP rises were only recorded in one rat. It is unclear whether the ICP rises in this animal could be considered spontaneous. In this rat, we numbered two and three ICP rises in response to 100 and 300 ng OT, respectively. We hypothesize that in this animal, the dose of OT antagonist was not great enough to prevent responses to high doses of OT. We also consider that in the one rat that displayed ICP rises after vasopressin injection, such ICP rises occurred spontaneously. Indeed, they were recorded after an injection of 0.3 ng vasopressin. When compared with other groups, neither 0.3 ng OT nor its agonist elicited ICP rises.

After it injections of 30 ng OT, the number of ICP rises, their amplitude, and area were greater than those recorded with the other OT doses. After injection of the OT agonist, 100 and 300 ng elicited the greatest numbers of ICP rises, and these reached the greatest amplitudes. The bell-shaped curve was evident when using OT, and rare or no ICP rises were recorded after 300 ng OT. In contrast, 300 ng of the OT agonist still elicited many responses. OT and [Thr⁴, Gly⁷]OT display the same affinity for the OT receptors (11).

Table 3. Effect of a single i.t. injection of OT at the L4–L6 spinal cord on 1) the number of rats that displayed at least one ICP rise during the experiment (responders) relative to N, 2) the number of ICP rises per rat in each group, 3) the latency of the first ICP rise (s), 4) the duration of ICP rises (s), 5) the amplitude of ICP rises (ICP/BP ratio), and 6) the AUC of ICP rises (arbitrary units)

<table>
<thead>
<tr>
<th>Doses, ng</th>
<th>Responders/N</th>
<th>Number of ICP</th>
<th>Latency (s)</th>
<th>Duration (s)</th>
<th>ICP/BP</th>
<th>AUC/BP</th>
</tr>
</thead>
<tbody>
<tr>
<td>10</td>
<td>9/12</td>
<td>4.4 ± 1.3</td>
<td>597 ± 149</td>
<td>75 ± 8.0</td>
<td>0.70 ± 0.050</td>
<td>26.9 ± 3.3</td>
</tr>
<tr>
<td>30</td>
<td>6/7</td>
<td>4.9 ± 1.4</td>
<td>696 ± 202</td>
<td>65 ± 7.0</td>
<td>0.68 ± 0.10</td>
<td>22.6 ± 3.4</td>
</tr>
<tr>
<td>100</td>
<td>6/7</td>
<td>4.7 ± 1.0</td>
<td>422 ± 129</td>
<td>90 ± 9.0</td>
<td>0.75 ± 0.1</td>
<td>36.5 ± 6.9</td>
</tr>
<tr>
<td>300</td>
<td>2/8*</td>
<td>1.0 ± 0.7</td>
<td>507 ± 216</td>
<td>52 ± 2.0</td>
<td>0.44 ± 0.03</td>
<td>9.1 ± 0.9</td>
</tr>
</tbody>
</table>

Values are means ± SE. BP, blood pressure; AUC, area under the curve. *Statistically different from the other doses.
receptor present in uterine smooth muscle (2); a difference between spinal and peripheral OT receptors could explain why 300 ng OT does not elicit any erectile response in our experiment. It is unlikely that the saturability of the OT receptor accounts for the lack of ICP rises on 300 ng OT, because this dose should elicit at least as many ICP rises as 100 ng OT. It is interesting to note that 300 ng OT elicited no more ICP rises when injected as either cumulative or single doses. Could the desensitization of the OT receptor explain this decrease of ICP response? In cultured astrocytes, OT applications elicited calcium rises in which amplitude decreased if the application was repeated. The authors observed a 20-min wash period between two applications before they could record a full recovery of the calcium response (10). In our experiment, a period of 15 min separated two consecutive injections, which suggests that no recovery could occur in this condition. OT (10, 30, and 100 ng) elicited fewer ICPs on single doses compared with cumulative doses. Furthermore, the ICP/BP ratio displayed a bell-shaped response curve for cumulative treatments, but no such profile was noted on single-dose treatments. It remains unclear to us whether such differences rely on time of exposure of the receptor to OT or to an interaction between time of exposure and dose.

At high doses, OT may bind vasopressin receptors. The latter are present in the lumbosacral spinal cord of rats (33). Although 10 times less potent than vasopressin, OT could act on the V1 receptor of sympathetic preganglionic neurones of the neonate rat spinal cord (27). In our experiment, OT acting at vasopressin receptors could display inhibitory effects on spinal proerectile neurones. We tested the effects of it vasopressin. Neither OT (10, 30, and 100 ng) elicited any ICP rises on single doses compared with cumulative doses. Furthermore, the ICP/BP ratio displayed a bell-shaped response curve for cumulative treatments, but no such profile was noted on single-dose treatments. It remains unclear to us whether such differences rely on time of exposure of the receptor to OT or to an interaction between time of exposure and dose.

Penile erection in conscious mammals recruits autonomic pathways to the penis and somatic pathways to the perineal striated muscles (25). In conscious mammals, contraction of striated muscles on the erect penis elicits peaks of penile pressure rises that largely over-ride BP (5, 23, 26). According to some authors, bulbospongiosus (BS) and ischiocavernosus (IC) motoneurons receive descending projections from the PVN (35). Furthermore, transsynaptic retrograde labeling from the IC or BS muscles using PRV or rabies virus labels some neurones in the PVN (19, 31). According to these data, OT could also control the somatic outflow to the perineal striated muscles. However, although rare OT-immunoreactive fibers have been demonstrated in the ventral horn of the rat spinal cord (30, 34), this area does not contain OT receptors (34). After OT injection, we never recorded ICP rises over BP, and the injection of the striated muscle blocking agent gallamine triethiodide did not affect the ICP rises elicited by OT. Therefore, our data demonstrate an effect of OT on penile pressure, independent of striated muscle, and suggest a lack of excitatory effect of OT onto IC and BS motoneurons.

OT may activate lumbosacral parasympathetic neurones and interneurones destined to pelvic organs other than the penis. Indeed, it was demonstrated that in conscious female rats, it OT increased micturition pressure and decreased bladder capacity and micturition volume (22). Interestingly, the most efficient dose in this model was 30 ng OT. We also identified 30 ng as the dose of OT that yielded the greatest probability of eliciting ICP rises when injected in cumulative doses, and only the number of responders when OT was injected as single doses. If comparable doses of OT activate different parasympathetic outflows, then it remains to be determined how the spinal network integrates this increase of OT, because all pelvic viscera are not active at the same time. It may be suggested that in a sexually relevant context, it is the convergence of information from the periphery and from supraspinal structures that elicits the specific activation of proerectile pathways at the spinal cord level.

In conscious rats, noncontact erections and drug-induced erections (e.g., apomorphine-induced erections) reflect the activity of supraspinal nuclei. These erections are transient and repetitive, and recording ICP during noncontact and apomorphine-induced erections revealed transient rises of ICP (6). In our experiments, injections of OT elicited phasic ICP rises. It suggests that the spinal cord translates the tonic excitation by supraspinal nuclei or by OT into the phasic activation of parasympathetic pathways leading to phasic ICP rises.

In our experiment, OT would be a potent activator of the spinal generator of penile erection. Once this rhythmic generator is activated, OT could not further regulate the number, the amplitude, and the area of the ICP rises, as evidenced by lack of dose effect of OT injection on the ICP/BP and AUC/BP ratios.
Pharmacological stimulation of the PVN in conscious rats and its electrical stimulation in anesthetized rats elicit penile erection (4, 8). Lesions of the PVN suppress apomorphine-induced erections (4) and impair noncontact erections (16). In rats, fibers issued from the paraventricular part of the PVN reach the lumbosacral spinal cord (7). The SPN contains OT-immunoreactive fibers and OT receptors (30, 34). Our results suggest that by delivering OT at the lumbosacral level, we mimicked the release of OT by PVN-spinal fibers in rats. The present results demonstrate that OT exerts proerectile effects, as measured through increases in ICP, when it is delivered at the L4-L6 spinal cord in anesthetized rats. They demonstrate that the effects are specific, being mimicked by a specific agonist but not by arginine-vasopressin, and are blocked by a specific OT antagonist. Proerectile effects of OT are due to the activation of autonomic efferent pathways running in the pelvic nerves.

Perspectives

Our experiments suggest that the lumbosacral spinal cord is the final target of a proerectile, oxytocinergic pathway in which perikarya are in the paraventricular part of the PVN. This pathway represents a very efficient and direct proerectile link between supraspinal nuclei and the spinal cord. To better understand the contribution of peripheral and supraspinal information to the generation of erection, it is tempting to test the effects of OT in rats after a complete section of the spinal cord at the thoracic level, i.e., the interruption of the proerectile PVN-spinal pathway. Also, the comparison of the responses of the spinal cord to OT after a section that would be performed either immediately or several days before the test would provide an understanding of the strategies that some spinal networks can use to compensate for the lack of supraspinal information.

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