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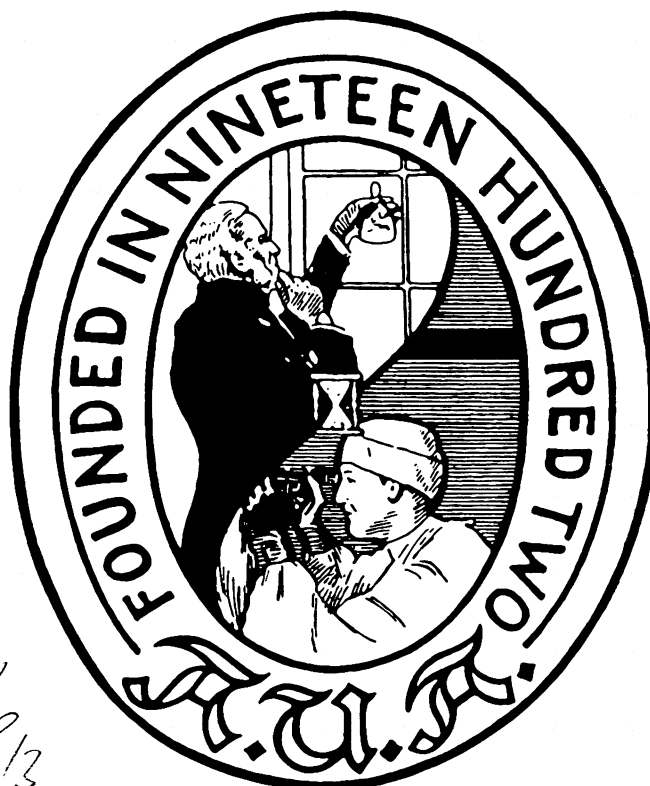
Volume 146

July 1991

Number 1

The Journal of UROLOGY®

Official Journal of the American Urological Association, Inc.
Founded In 1917 By Hugh Hampton Young



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Annual Meeting, American Urological Association, Inc., Washington, D. C., May 10-14, 1992

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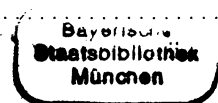
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SYMPATHETIC INHIBITION OF PAPAVERINE INDUCED ERECTION

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ABSTRACT

We studied the effect of neurostimulation of the lumbar sympathetic trunks on papaverine-induced penile erection in dogs and monkeys. The mean of 124 cm. H₂O intracavernous pressure after papaverine injection was reduced under sympathetic trunk stimulation to 40.6 cm. H₂O within a mean of 39 sec. In addition the flow of the internal pudendal artery was reduced indicating a decrease of the inflow to the penis. After the sympathetic stimulation was terminated, the intracavernous pressure increased again to the same level as before. This pressure recovery time was delayed after several sympathetic stimulations from 134 sec. (5 min. after papaverine injection) to at least 362 sec. (45 minutes after papaverine injection). Stimulation of the inferior hypogastric plexus had no effect on the intracavernous pressure.

The neurostimulation range (3-4V; 20 Hz) was most effective in reducing the papaverine action on penile smooth muscles.

The effect of sympathetic trunk stimulation seems to antagonize the relaxing action of papaverine on the cavernous smooth muscles and the cavernous arterioles. Our results imply that high sympathetic tone in some patients with psychogenic impotence may explain their poor response to intracavernous injection of papaverine.

KEY WORDS: papaverine, penis, penile erection

In 1982, Virag¹ reported the use of intracavernous papaverine injection in the treatment of erectile failure. During the following years papaverine has been used in therapy^{2,3} and in differential diagnosis⁴⁻⁶ of erectile dysfunction. It was proposed that papaverine can be used to substitute for the neurotransmitter to test arterial, venous and sinusoidal mechanisms. Papaverine was thought to be useful in differentiating organic from psychogenic impotence.² But based on further investigations it was suggested that this pharmacologic testing was not sufficient to provide a clear cut discrimination between patients with organic and psychogenic impotence.^{7,3}

Papaverine, the main benzyloquinoline alkaloid of opium, relaxes smooth muscles and reduces the contractile responses to stimulant agents by a wide variety of stimulants *in vitro*.⁸ However Beausang-Linder⁹ showed that papaverine-induced dilatation of cranial arteries in rabbits could be abolished by electrical stimulation of the cervical sympathetic trunk. In our recent canine studies¹⁰ we demonstrated that stimulation of the lumbar sympathetic trunks (STS) inhibited cavernous nerve-induced erection.

The purpose of this animal study is to determine the effect of STS on papaverine-induced penile erection.

MATERIALS AND METHODS

Six mongrel dogs (22-29 kg.) and five pigtail monkeys (6.7-8.3 kg.) were used in these experiments.

Animal preparation. The dogs were premedicated subcutaneously with azepromazine (0.1 mg./kg.) and anesthetized with pentobarbital intravenously (100-130 mg. as a bolus; maintenance dose of pentobarbital was given as required). Hydration was maintained by saline perfusion (two ml./kg./hour). The dogs were placed in the supine position. The abdominal cavity was entered by a lower midline incision and bipolar cuff electrodes (Avery Lab.) were placed around both lumbar sympathetic trunks at the level of L₅. In four of six dogs cuff electrodes were also implanted around the inferior hypogastric plexus. The midshaft of the penis was exposed by incising skin and

subcutaneous layers. Both cavernous bodies were punctured with a 21-gauge scalp vein needle for pressure recording. The right femoral artery was cannulated with a 16 gauge angiocath needle to record the systemic blood pressure. An ultrasound flow probe (1.5 mm.; Transonic system) was placed around the right internal pudendal artery. The monkeys were premedicated with ketamine (10 mg./kg.). Anesthesia was started with inhalation (closed anesthesia system; Foregger Rotameter model) of halothane (4%) and maintained at 0.6-1.0% after intubation. A Harvard Pump (Apparatus No. 607) was used for assisted respiration. Continuous intravenous saline infusion was given for fluid maintenance (3-5 ml./kg./hour). The monkeys were placed in the supine position, and a lower midline incision was made to enter the peritoneal cavity. The infrarenal aorta was exposed and cuff electrodes were placed on both sympathetic trunks (L₅). A corpus cavernosum was punctured with a 21 gauge scalp vein needle for pressure recording. The systemic blood pressure and pulse rates were measured externally every five minutes and recorded. All animals were placed on a 37°C controlled waterbath mattress (aquamatic model K 20) to prevent hypothermia. After the surgical set up the animals were left to equilibrate for 30 min.

Recording. The needles from the corpora and the femoral artery were connected to Statham transducers. A Polygraph Model 7 or a Beckmann Dynograph (type R 411) were used for the recordings.

Neurostimulation. The cuff electrodes were connected to subcutaneously placed receivers (Avery Lab.). A hand-held transmitter with antenna (Avery Lab.) was used to deliver radiofrequency signals to the receiver for neurostimulation (parameters: 1.2-6 V; 20 Hz). In a first protocol 10 mg. papaverine hydrochloride (Lilly Pharma) were injected to the cavernous bodies via one of the scalp vein needles to all animals. Sympathetic trunks were stimulated (3 V in dogs; 4 V in monkeys) for 60 seconds each five, 15, 30, and 45 minutes after papaverine injection. In a second protocol 10 mg. of papaverine were injected again to the cavernous bodies in four dogs and two monkeys 60 minutes after cessation of the first induced erection. The hypogastric nerve was stimulated in all four dogs after three and ten minutes by increasing the stimulation voltages from zero to 6 V. Five and fifteen minutes after the

Accepted for publication January 23, 1991.

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second papaverine injection sympathetic trunks were stimulated with 1.2 V for one minute in four dogs and two monkeys. Afterwards the stimulation voltage was increased during one minute to 6 V and stimulation was stopped after additional 60 seconds.

The statistical significance of correlations among different responses on STS during papaverine induced erections in dogs and monkeys (first protocol) was calculated with the wilcoxon signed rank test. Values were considered significantly when $p < 0.05$.

RESULTS

In the following section, data presented are mean values of the experiments unless indicated otherwise.

The intracavernous pressure increased after the first (second) papaverine injection from 17.9 (20.2) to 141 (137) cm. H₂O in dogs and from 15.6 (17.9) to 102 (105) cm. H₂O in monkeys. A small additional pressure hump of 5-10 cm. H₂O was always recorded 8-10 sec. after initiating STS.

The intracavernous pressure dropped (table 1), during series of nerve stimulations. Figure 1 shows a computer tracing of an original recording from a dog five min. after papaverine injection. Papaverine-induced erections lasted at least 15, and sometimes more than 60 minutes after intracavernous injection. Erections for more than one hour were abolished by removing several drops of blood from the corpora and flushing the corpora with saline. After the first neurostimulation was terminated the intracavernous pressure increased to its pre-neurostimulation level in all animals (figure 1; table 1).

Similar pressure decreases were elicited by neurostimulation fifteen, thirty and forty-five minutes after papaverine injection (table 1). Variability in the time interval from start of STS to the final reduction of intracavernous pressure was only about 15% (7 sec; table 1). The recovery time, (table 1) to regain the tumescence pressure increased after repeated sympathetic stimulations (figures 2 and 3). In one dog there was only one response to neurostimulation (five minutes after papaverine injection). The erection lasted for more than one hour. In two monkeys only the additional pressure hump was observed but no subsequent pressure reduction. In all experiments STS caused a brief systemic blood pressure drop (5-15 sec. after initiation of STS) of 5-20 cm. H₂O (figure 1). The internal pudendal artery flow rate temporarily increased after papaverine injection to a maximum of 16.5 ml./min. The maintenance flow during papaverine erection was four to 6.5 ml./min. (mean 5.6 ml./min.), it fell under the influence of the first sympathetic trunk stimulation to a mean of four ml./min. No arterial flow increase was observed after cessation of STS and additional neurostimulations after 15, 30 and 45 minutes.

Nine times in different dogs reduced voltages of 1.2-2 V was used in the second protocol. After the initial pressure hump a

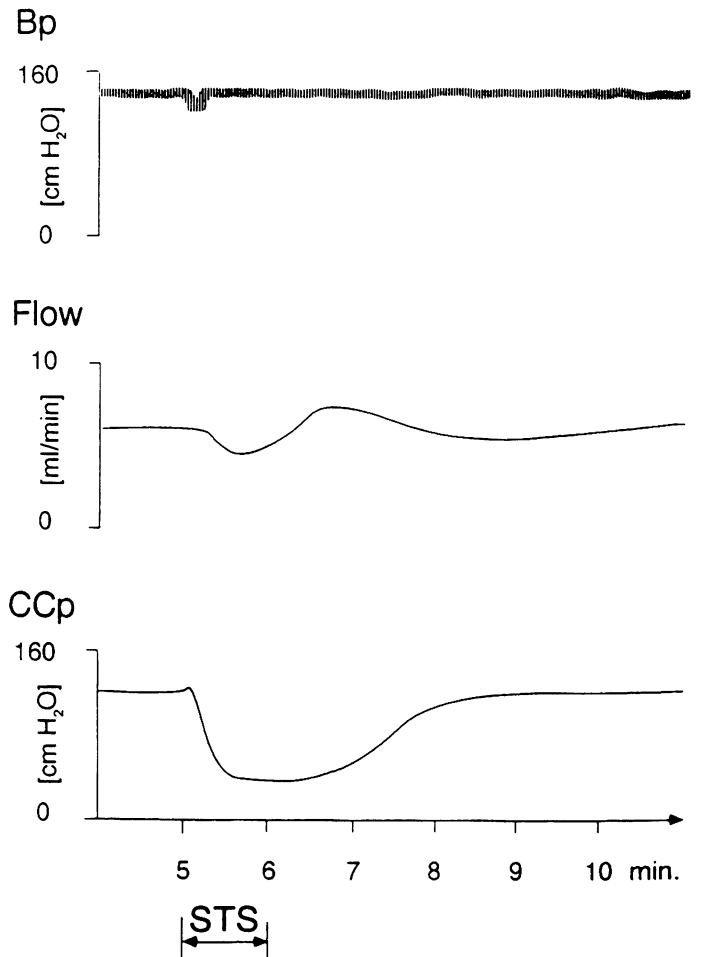


FIG. 1. Computer tracing of original recording of corpus cavernous pressure (CCp), arterial flow (Flow) of internal pudendal artery and systemic blood pressure (Bp) in dog; 5 min. after intracavernous injection of papaverine, lumbar sympathetic trunks (STS) were stimulated.

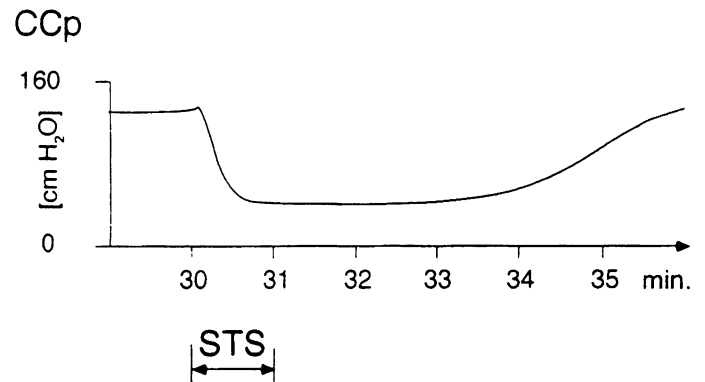


FIG. 2. Computer tracing of original recording of corpus cavernous pressure (CCp) in monkey; lumbar sympathetic trunks (STS) were stimulated 15 min. after intracavernous injection of papaverine.

TABLE 1. Effect of sympathetic trunk stimulation (STS) on intracavernous pressure (ICP) during papaverine-induced erections (first protocol)

Time of STS After Injection of Papaverine [min.]	Trials	ICP Under STS [cm. H ₂ O]	Time Required to Reduce ICP Under STS [sec.]	Recovery Time After STS [sec.]
A				
5	6	40.6 (30-53)	38.0 (30-45)	134.3 (90-180)
15	5	42.5 (31-49)	39.8 (32-46)	158.8 (100-220)
30	5	40.9 (26-54)	45.0 (36-48)	256.4 (130-400)
45	4	40.0 (29-57)	40.0 (30-46)	362.0 (260-570)
B				
5	3	33.8 (21-39)	36.5 (30-41)	128.1 (90-160)
15	3	33.5 (22-36)	39.1 (36-42)	133.0 (90-170)
30	3	37.0 (24-53)	39.2 (33-49)	185.3 (120-260)

A) Six dogs.

B) Three of five monkeys (two monkeys did not respond to neurostimulation).

All values are mean values (variability in brackets; only one trial per animal).

small pressure reduction of 10-25 cm. H₂O was recorded in four, and no reduction in five animals. Increasing the stimulation voltages of 6 V showed no additional effect compared to 3-4 V of the first protocol.

Hypogastric nerve stimulation (up to 6 V) did not influence the intracavernous (figure 4) or systemic blood pressure.

Statistical analysis: The wilcoxon signed rank test was applied to the correlation of the first protocol among intracavernous pressure (ICP) under STS, maintenance flow of the

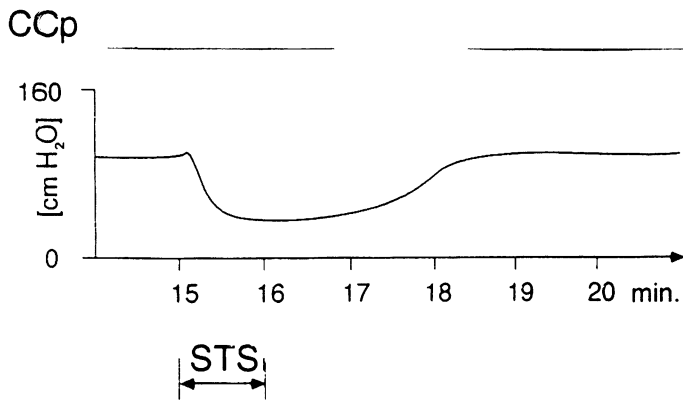


FIG. 3. Computer tracing of original recording of corpus cavernous pressure (CCp) in dog; lumbar sympathetic trunks (STS) were stimulated 30 min. after intracavernous injection of papaverine.

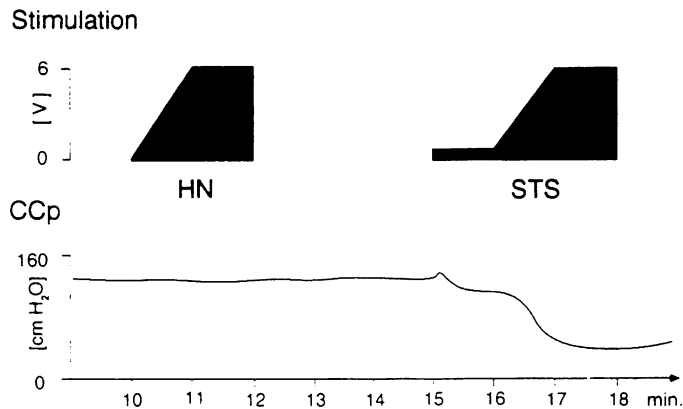


FIG. 4. Computer tracing of original recording of corpus cavernous pressure (CCp) in dog (second protocol). Hypogastric nerve (HN) was stimulated 10 min. and lumbar sympathetic trunks (STS) 15 min. after intracavernous injection of papaverine.

internal pudendal artery under STS, time required to reduce ICP under STS and recovery time after STS. ICP was reduced highly significant by STS ($p < 0.009$). The p value between maintenance flow of the internal pudendal artery before and after STS was 0.014. There was no significant difference of time required to reduce ICP by STS. The recovery time to regain the tumescence pressure increased significantly after repeated STS ($p < 0.02$) in dogs and monkeys (correlation between dogs versus monkeys $p < 0.05$).

DISCUSSION

These results demonstrate that the papaverine effect on penile smooth muscle is reduced by lumbar sympathetic trunks stimulation. STS decreased intracavernous pressure and flow of the internal pudendal artery.

A number of different animal studies have contributed to the understanding of the physiological and drug-induced penile erection. The smooth muscle relaxing properties of papaverine seem to result in two distinct hemodynamic changes¹¹: increase of arterial flow and venous resistance. These observations were consistent with the hemodynamic changes we found in dogs and monkeys.

We observed an intracavernous pressure reduction and a flow reduction in the internal pudendal artery during STS on papaverine induced erection. Both of these effects may be due to the contraction of smooth muscles, as suggested by in vitro experiments.¹² We believe that the reduction of intracavernous pressure by STS is due to a high venous outflow as we observed in our pharmaco-cavernosometry experiments (13). Fournier et al.¹⁴ mentioned a subalbugineal venular plexus which is compressed between distended sinusoids and the tunica albuginea

during erection. Contractions of cavernous smooth muscles open these venular plexus. We suggest that STS mediates a release of catecholamines on the corporeal tissue.¹⁵ These transmitters might be responsible for reopening of the compressed venules.

A rich adrenergic innervation has been demonstrated in the mammalian penis.¹⁶ An alpha receptor mediated contractile response to norepinephrine stimulation was clearly shown in cavernous smooth muscles of monkeys.¹⁶ Carati et al.¹⁷ proposed that a noradrenergic alpha adrenoreceptor system maintains penile flaccidity. Norepinephrine—the neurotransmitter of most sympathetic postganglionic endings—is probably released in penile tissue by STS.¹⁵ This excitatory action of norepinephrine is due to an increase of calcium influx, release of calcium from intracellular stores and suppression of calcium extrusion or sequestration in smooth muscle.¹⁸ This leads to an increase of the ionized intracellular calcium concentration. The intracellular coupling mechanism has not yet been fully analyzed. Phosphatidylinositol may be involved in the contractile response.¹⁸ Papaverine is believed to reduce this ionized intracellular calcium concentration by different mechanisms. There is evidence that papaverine is a very potent inhibitor of phosphodiesterase. This relaxant property of papaverine seems to be mediated by an accumulation of cyclic 3', 5' AMP.¹⁹ In contrast Huddart et al.²⁰ considered the main site of papaverine action to be on the calcium influx control mechanism. However the exact mechanism of adrenergic and papaverine action on the intracellular ionized calcium concentration awaits further investigations.

In all experiments the intracavernous pressure after papaverine injection was not totally reduced to its flaccid state on STS. Even stimulations for more than one minute or stimulations using higher voltages did not enhance the response. We suggest that a certain amount of norepinephrine is released by STS. This could explain the small variability in the time interval from start of STS to the final reduction of intracavernous pressure. However this quantity of the released neurotransmitter was not able to abolish the papaverine response on cavernous smooth muscles completely. The increased recovery time to regain the tumescence pressure after repeated STS is probably due to a decrease of papaverine action on cavernous smooth muscles. Finally we found a different response of STS on papaverine action in dogs and monkeys to regain the tumescence pressure. This might depend on different kinds of anesthesia we used or on differences of neurotransmitter release and smooth muscle activation in dogs and monkeys.

Previously Carati et al.¹⁷ observed that in dogs the response to pelvic nerve stimulation was not significantly altered by either stimulation or section of the hypogastric nerves. These results correspond to our findings of hypogastric nerve stimulation on papaverine induced erections. However we have not used higher voltages (14 V) which can reduce a pelvic nerve induced erection.²¹ Taking into account these neurophysiological observations we suggest that a current spread to the sympathetic trunks is responsible for intracavernous pressure changes by hypogastric nerve stimulation (14 V).

Recently Buvat et al.⁷ reported no response to papaverine in 33% (9/26 patients) of well documented psychogenic patients. The result of our study supports their conclusion that overactivity of the sympathetic nervous system can exert an inhibitory effect on papaverine induced penile erection.

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