Urol Res (1989) 17:213–217

Urological Research

Original articles

Neurophysiology of penile erection

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Accepted: January 3, 1989

Summary. In 6 dogs and 6 monkeys electrical stimulation of the cavernous, pudendal and hypogastric nerve was performed to gain better understanding of the erectile neurophysiology. Arterial flow, intracorporeal pressure and venous restriction studies during single and combined neurostimulation demonstrated that initiation and maintenance of erection is a parasympathetic phenomenon. Penile rigidity however, could only be achieved with additional pudendal nerve stimulation resulting in muscular compression of the blood distended cavernous bodies. Detumescence or subsidence of erection is primarily under sympathetic control, due to inhibition of sinusoidal smooth muscle relaxation. On the basis of our observations we conclude that penile erection is dependent upon three neurophysiological mechanisms: 1. the parasympathetic "vascular mechanism", the somatomotor "muscular mechanism" and the sympathetic "inhibitory mechanism".

Key words: Penile erection – Neurophysiology – Rigidity – Detumescence

Introduction

According to our recently presented results on erectile anatomy and physiology penile erection is the result of: 1. arterial dilatation, 2. cavernous relaxation, and 3. venous restriction [3, 4, 10]. The role of the parasympathetic nervous system in initiation and maintenance of erection has been determined, whereas the exact neurological understanding of erectile control remains undefined.

Part of this paper was presented at the 9th Symposium of the Association for Experimental Urology of the German Urological Society, June 17–18, 1988, Aachen, Federal Republic of Germany

Previous studies of the neurophysiology of the erectile mechanism have resulted in more detailed understanding [9, 5].

Material and methods

A total of 6 dogs (16 to 30 kg BW) and 6 monkeys (8 to 10 kg BW) underwent electrode implantation (Avery Laboratories, NY) for selective and combined neurostimulation of the cavernous, pudendal and proximal hypogastric nerve as described elsewhere [5].

Arterial flow changes during electrostimulation-induced erection and/or detumescence were measured using an ultrasonic flow probe (Transonic Systems, Inc.; Ithaca, NY) placed around the internal pudendal artery in the dogs. Intracorporeal pressure, arterial blood flow to the penis and systemic blood pressure (16-GA cannula placed in the femoral artery) were measured simultaneously and recorded on a Grass polygraph.

Venous capability was tested by clamping the infrarenal aorta temporarily while perfusing the corpus cavernosum with normal saline at a constant flow rate (Harvard perfusion pump; 0.9-7 ml/min) [4].

The student's-t-test was used for statistical analysis.

Cavernous nerve stimulation

After all monitoring devices were in place baseline readings for arterial penile flow, corpus cavernosum pressure and systemic blood pressure were taken from each animal.

All dogs and all monkeys underwent electrostimulation of the cavernous nerve twice: with the individual threshold parameters to induce an erection, and subsequently with a higher amplitude. Additionally, all dogs underwent a penile perfusion trial in order to study the venous outflow system of the corporeal body before and during cavernous nerve stimulation.

Pudendal nerve stimulation

In 4 primates single pudendal nerve stimulation was performed in addition to the cavernous stimulation, followed by a combined neurostimulation of the cavernous and pudendal nerve to see its effect on the erectile tissue.

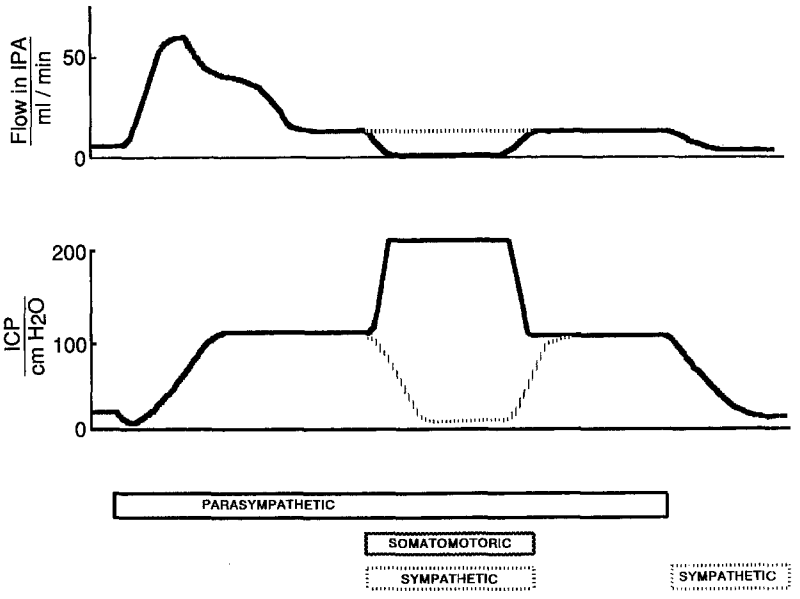

Fig. 1. Parasympathetic stimulation resulted in an initial intracorporeal pressure drop (cavernous smooth muscle relaxation) followed by an arterial inflow (upper chart) and intracorporeal volume and pressure increase up to full tumescence. Stimulation of the somatomotor pudendal nerve fibers at the fully erect state culminated in a tremendous intracorporeal pressure rise above systemic blood pressure and penile rigidity. Additional hypogastric nerve stimualtion resulted in subsidence of the parasympathetic-induced (cavernous nerve) erection. Erection recurred after sympathetic activation was turned off. IPA = internal pudendal artery; ICP = intracorporeal pressure

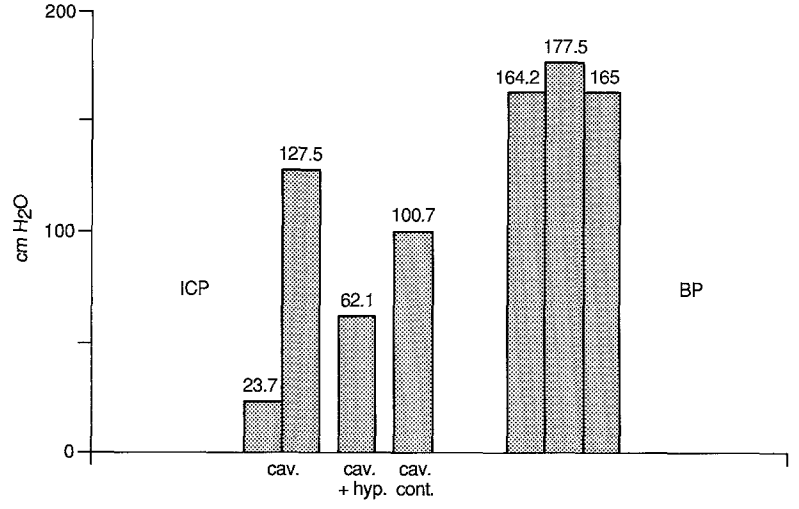

Fig. 2. Intracorporeal pressure (ICP) changes after cavernous (cav.) and hypogastric (hyp.) nerve stimulation in dogs. Single cavernous nerve did result in a significant ICP rise and full erection ($P<0.005$). Additional hypogastric (sympathetic) nerve activation caused a significant pressure decrease and detumescence occurred ($P<0.005$). Furthermore, the systemic blood pressure was elevated unter the sympathetic nerve stimulation. The erectile response was almost completely renewed after cessation of the sympathetic excitation. BP = systemic blood pressure

Table 1. Stimulation parameters

Cavernous nerve stimualtion	
- voltage:	0.1 to 6 volts (dog) 6 to 9 volts (monkey)
- frequency:	20 Hz (dog) 7 to 10 Hz (monkey)
Pudendal nerve stimulation	
- voltage:	6 to 9 volts (monkeys only)
- frequency:	33 Hz
Hypogastric nerve stimulation	
- voltage:	14 volts (dogs only)
- frequency:	20 Hz

Hypogastric nerve stimulation

The neurostimulation-induced effect of the sympathetic nerve fibers of the proximal hypogastric nerve, alone and in combination with cavernous nerve stimulation was tested in 6 dogs.

Each arterial flow and intracorporeal pressure trial was followed by a saline perfusion study under single and combined neurostimulation patterns as described in Table 1.

Results

Cavernous nerve stimulation

The penile resting pressure in both corpora cavernosa was approximately 11 to 25 cm H_2O. With a latency of about 10 s after stimulation of the cavernous nerve the

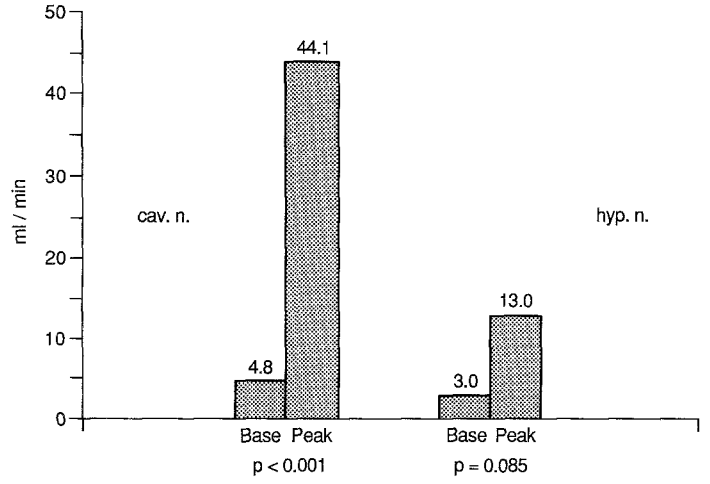

Fig. 3. Arterial flow increase after single cavernous and hypogastric nerve stimulation

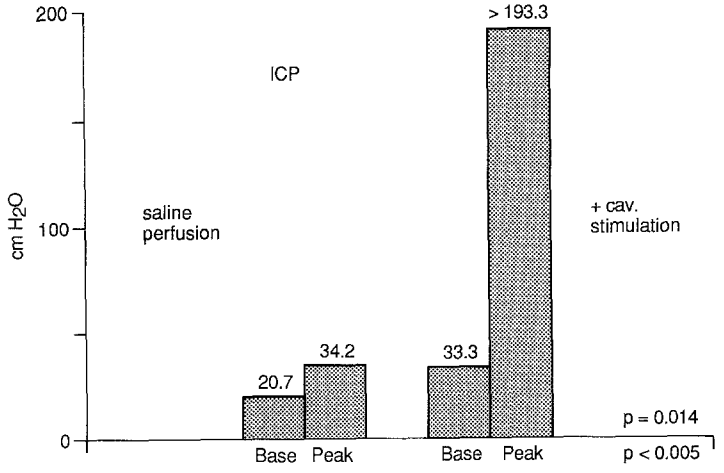

Fig. 4. Saline perfusion alone did not show any significant ICP rise. However, additional cavernous nerve stimulation did result in a significant rise in ICP (same perfusion rate). ICP = intracorporeal pressure

penis lengthered and engorged, before it became tumescent. An initial drop in intracorporeal pressure was followed by a pressure increase up to a mean peak pressure of 127.5 cm H_2O to approximate the systolic blood pressure (Figs. 1 and 2). The high intracorporeal pressure plateau was maintained as long as stimulation was continued.

In the monkeys a slight difference in the erectile response was observed. During the tumescence phase the penis showed a pulsatile increase in length and diameter in accordance with the monkey's own pulse, and engorgement of the glans penis and deep dorsal vein. At the time of full erection the mean intracorporeal pressure reached 130 cm H_2O (dogs 127.5 cm H_2O).

Electrostimulation of the cavernous nerve bundle was immediately followed by a tremendous arterial flow to the corpora cavernosa (4.8 to 44.1 ml/min; Fig. 3) and an initial drop in intracorporeal pressure (Fig. 1). During the tumescence phase the penile blood flow declined to almost baseline levels as soon as the intracorporeal pressure reached a plateau (127.5 cm H_2O). Detumescence occurred shortly after cavernous stimulation was terminated. During the entire study the systemic blood pressure remained stable. Saline perfusion of the dog's corpus cavernosum with the arterial supply cut off temporarily did not result in penile erection. Yet, additional cavernous nerve stimulation caused a rapid pressure increase in the corpus cavernosum above the systemic blood pressure (>193.3 cm H_2O; Fig. 4)

Pudendal nerve stimulation

Electrical activation of the pudendal nerve fibers in the monkeys did not show any significant change in intracorporeal pressure (Tab. 4). Once erection or tumescence was established by cavernous stimulation, additional pudendal nerve activation induced contraction of the striated muscle surrounding the proximal

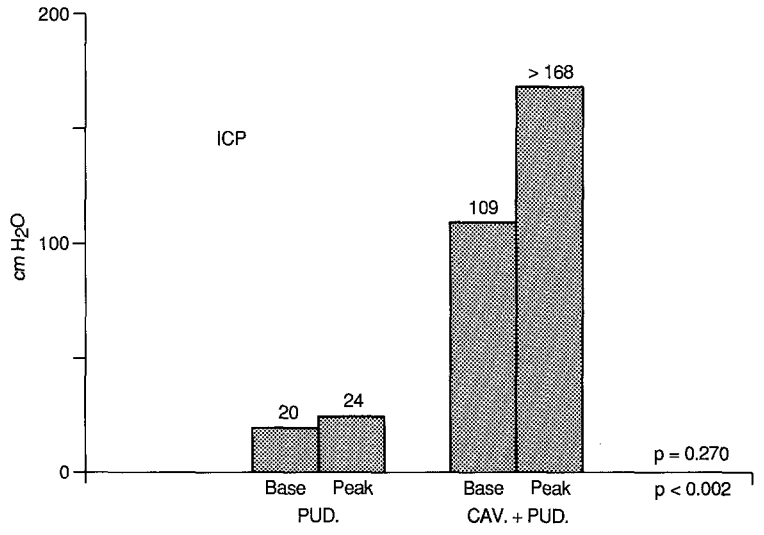

Fig. 5. Pudendal nerve stimulation alone did not show any significant effect on the erectile tissue ($P = 0.270$). Additional pudendal nerve stimulation, however resulted in penile rigidity with a tremendous intracorporeal pressure rise ($P < 0.002$; monkeys)

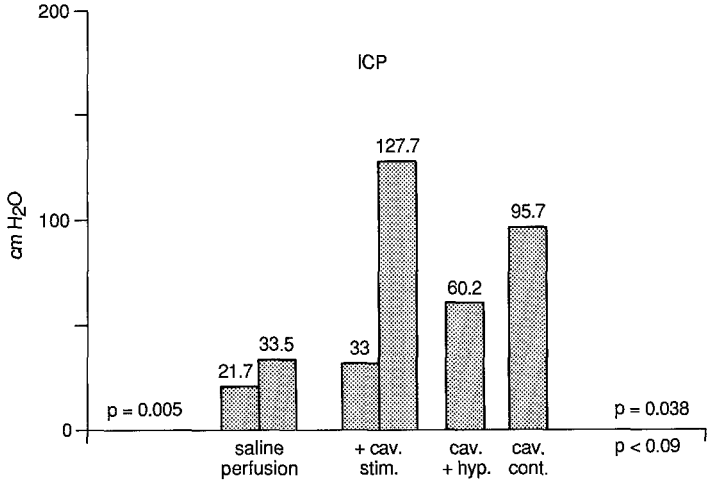

Fig. 6. Combined cavernous and hypogastric nerve stimulation during saline perfusion did result in an initial ICP rise followed by a significant ICP drop after sympathetic (hypogastric) nerve stimulation was initiated ($P = 0.038$). ICP = intracorporeal pressure

penile shaft (ischiocavernous muscles), culminated in an intracorporeal pressure rise above 168 cm H_2O (Figs. 1 and 5) and the penile shaft became rigid. With termination of pudendal stimulation or fatiguing of the striated muscles the pressure returned to the erection plateau (Fig. 1).

Hypogastric nerve stimulation

Neither a difference in penile length or engorgement was observed nor did the intracorporeal pressure change significantly ($P = 0.44$) with single hypogastric stimulation. The arterial flow increase was small, however the systemic blood pressure went up to a mean value of 181 cm H_2O.

Activation of the hypogastric nerve during cavernous stimulation-induced erection resulted in penile detumescence. The intracorporeal pressure declined by more than 50% (127.5 to 62.1 cm H_2O; $P < 0.005$) after initiation of the hypogastric nerve stimulation (Fig. 2). Erection recurred within 30 seconds after cessation of the sympathetic excitation (Figs. 1 and 2). Similar findings occurred with hypogastric stimulation in the saline perfusion or venous studies. Additional hypogastric nerve activation inhibited further cavernous stimulation-induced intracorporeal pressure rise, and the pressure declined, resulting in penile detumescence (Fig. 6). Once the sympathetic excitation was cut off, a renewed pressure increase up to full erection or rigidity was observed.

Discussion

The nervi erigentes were named by Eckhardt (1863) [2] after he discovered that erection could be induced by electrostimulation of parasympathetic nerve fibers

running along with the pelvic pelxus. In 1938 Semans and Langworthy [12] demonstrated the crucial role of the sympathetic nervous system in the erectile mechanism, however later findings were contradictory and the hemodynamic changes on the erectile tissue during sympathetic nerve stimulation were never assessed [1, 6, 11].

In our animals models, penile erection was initiated and maintained by electrostimulation of the cavernous nerves. Hemodynamicly, the parasympathetic excitation resulted in an increased arterial flow to the penis due to arterial dilatation, followed by an intracorporeal pressure increase up to the fully erect state, which is about 20 to 30 cm H_2O below systemic blood pressure. The initial drop and subsequent rise in corporeal pressure indicates that smooth muscle relaxation must have taken place with cavernous stimulation. With increased intracorporeal volume and pressure the subtunical venous plexus is compressed between the extended sinusoidal spaces and corporeal smooth muscle and the rigid tunica albuginea and venous restriction takes places, as recently shown [3, 4].

By virtue of the combined cavernous and pudendal neurostimulation studies we were able to demonstrate that penile rigidity is related to the tone of the striated muscle surrounding the proximal part of the penile shaft (ischiocavernous muscles) which are innervated by somatomotor nerve fibers deriving from the pudendal nerve. Extracorporeal muscular compression of the blood distended corpora cavernosa culminated in intracorporeal pressure increase, resulting in a fully rigid penis. Our result correspond with the findings of Lavoisier et al. [8] on human volunteers, who found a correlation between reflex contractions of the ischiocavernous muscles and intracorporeal pressure increase.

According to our hypogastric nerve stimulation studies sympathetic excitation can subside or suppress penile erection. Similar findings were made by Langley and Anderson in 1895 [7] and Semans and Langworthy in 1938 [12] however, the hemodynamic changes on the erectile tissue during sympathetic stimulation remained unknown. The arterial flow, penile pressure and venous restriction studies in our animal model demonstrated that the inhibiting effect of sympathetic nerve fiber activation is due to contraction or blocking of cavernous stimulation-induced corporeal smooth muscle relaxation resulting in subsidence of erection.

Summarizing our data we postulate that complete penile erection and rigidity is dependent upon the integration of three neurological control centers: parasympathetic, somatomotor and sympathetic. Initiation and continuation of erection is a *parasympathetic* phenomenon, based upon vascular and corporeal smooth muscle relaxation determining the flaccid, latent, tumescence and erection phase and should be understood as the *vascular mechanism* of erection. Additional *somatomotor*-controlled extracorporeal striated muscle contraction leads to compression of the blood distended cavernous bodies with full rigidity, and therefore is defined as the *muscular mechanism.*

Detumescence and subsidence of erection is primarily a *sympathetic* phenomenon, due to corporeal smooth muscle contraction and should be described as the *inhibitory mechanism* of penile erection. Furthermore, it may play an important role in the pathogenesis of psychogenic impotence.

References

1. Domer FR, Wessler G, Brown RL, Charles C (1978) Involvement of the sympathetic nervous system in the urinary bladder, internal sphincter and in penile erection in the anesthetized cat. Invest Urol 15:404–407
2. Eckhardt C (1863) Untersuchungen über die Erektion des Penis beim Hunde. Beitr Anat Physiol 3:123–150
3. Fournier Jr GR, Jünemann K-P, Lue TF, Tanagho EA (1987) Mechanism of venous occlusion during canine penile erection: an anatomic demonstration. J Urol 137:163–167
4. Jünemann K-P, Luo JA, Lue TF, Tanagho EA (1986) Further evidence of venous outflow restriction during erection. Br J Urol 58:320–324
5. Jünemann K-P, Lue TF, Melchior H (1987) Die Physiologie der penilen Erektion. II. Neurophysiologie der penilen Erektion. Urologe [A] 26:289–293
6. Klinge E, Sjøstrand NO (1977) Comparative study of some isolated mammalian smooth muscle effectors of penile erection. Acta Physiol Scand 100:354–367
7. Langley JN, Anderson HR (1895) The innervation of the pelvic and adjoining viscera. J Physiol 19:71–130
8. Lavoisier P, Courtois F, Barres D, Blanchard M (1986) Correlation between intracavernous pressure and contraction of the ischiocavernosus muscle in man. J Urol 136:936–939
9. Lue TF, Takamura T, Schmidt RA, Palubinskas AJ, Tanagho EA (1983) Hemodynamincs of erection in the monkey. J Urol 130:1237–1241
10. Lue TF, Müller SC, Jünemann K-P, Fournier Jr GR, Tanagho EA (1987) Hämodynamische Veränderungen während der Erektion und funktionelle klinische Diagnostik der penilen Gefäße mittels Ultraschall und gepulsten Doppler. Aktuel Urol 18:115–123
11. Melman A, Henry D (1979) The possible role of the catecholamines of the corpora in peniel erection. J Urol 121:419–421
12. Semans JH, Langworthy OR (1938) Observations on the neurophysiology of sexual function in the male cat. J Urol 40:836–846

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