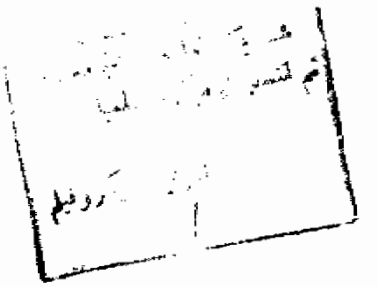
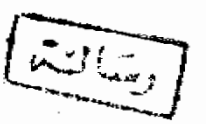


NON SURGICAL TREATMENT OF IMPOTENCE

THESIS

Submitted for partial fulfilment of Master Degree in

DERMATOLOGY AND VENEREOLOGY



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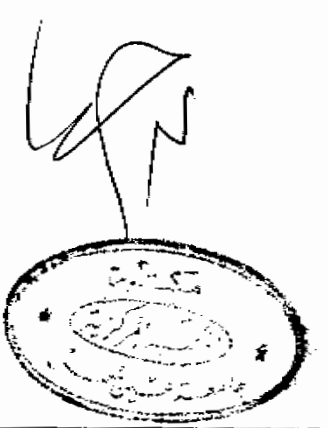

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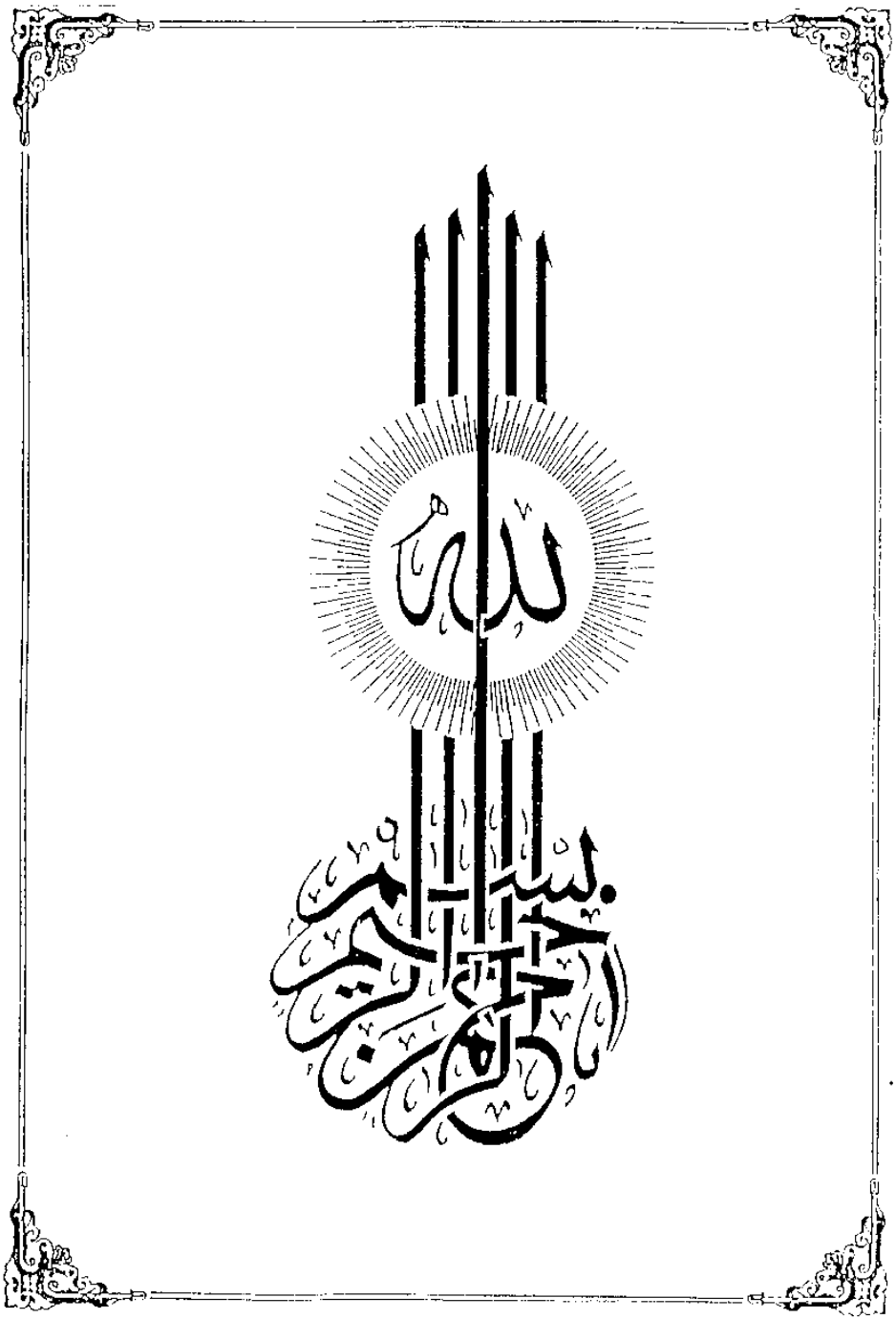

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INTRODUCTION

Penile erection is a complex physiologic response that is dependent upon the integration of vascular, endocrine, and neurologic mechanisms. Although many aspects of the neurohormonal control of erection are still uncertain, it is clear that erectile responses can be triggered by a variety of psychogenic and reflexogenic stimuli and that they are produced by a neurally mediated vasodilation of the penile blood vessels leading to an increase in blood flow and distension of the cavernous tissue **(Lue et al., 1983).**

As regards psychogenic erectile failure, good therapeutic results can be obtained with sophisticated psychotherapy that addresses cognitive and systemic elements **(Lopiccolo, 1988).**

As regards neurogenically impotent patients surgical therapy was the only treatment option offered in the past, but it is anticipated that in the future there will be established alternative therapies to the penile prosthesis such as more specific intracavernosal pharmacotherapy, oral pharmacotherapy and external devices **(Morales, 1982; Nadig, 1986).**

In cases of focal arterial vasculogenic impotence the treatment by revascularization procedures appears established, but a more logical therapy is combination of intracavernosal pharmacotherapy and if necessary a vascular reconstructive procedure to enable the medical therapy to become more effective **(Padma and Goldstein, 1988).**

In 1988 Tanagho stated that many endocrinally impotent patients are highly amenable to treatment by hormonal, surgical or other treatment modalities, however some cases for which all the contributing factors are not yet clear, proves more difficult to treat.

As we see in some instances, especially in neurogenic impotence and in partial arterial insufficiency, pharmacologic management might be a possibility. However,Broad-scale use of the pharmacologic approach is not yet recommended because of its potential danger, yet new drugs that might be safer and new delivery techniques might be on the horizon for the near future **(Tanagho, 1988).**

AIM OF THE WORK

Review of the literature as regards verifying the role of non-surgical methods in treatment of impotence and to present an up to date source not only to practicing doctors, but also to those who wish to achieve an overview of the current and evolving concepts of penile erection, impotence diagnosis and management.

MECHANISM OF ERECTION

MECHANISM OF ERECTION

The mechanism and hemodynamics of penile erection have long been a matter of controversy and assumption **(Aboseif et al., 1988)**. But over the last few years, there have been appreciable advances in the understanding of the physiology of penile erection **(De Tejada et al., 1988)**.

Considered from a purely physiologic stand point, erection is a neurologically mediated vascular event resulting in engorgement of the corpora cavernosa with blood **(Klein, 1988)**.

In the flaccid state the arterioles are constricted and the sinusoids are contracted. Together they exert maximal resistance against arterial flow, allowing only a small amount of blood to enter the corpora for nutritional purposes.During erection the smooth muscles of the sinusoids and arterioles relax, increasing the sinusoidal compliance and causing the peripheral resistance to decrease to a minimum. This results in an instantaneous increase in arterial flow, dilatation of the arterial tree and filling of the sinusoids. This dilatation not only allows blood to rush in but it also permits transmission of about 80 percent of the systolic blood pressure to the sinusoidal spaces with

little dampening. This high intracavernous pressure converts a soft flaccid organ to a blood-distended erect penis **(Lue and Tanagho, 1987).**

While corporeal filling occurs continuously, dynamic changes in the penis occur in two phases:

(A) Initial filling of the cavernous spaces causes corpora cavernosa to straighten, elongate and dilate and results in an overall increase in penile length and circumference (Tumescence). Intracorporeal pressure stays relatively constant during this initial phase, and maximal changes in circumference may occur before any increase in penile rigidity **(Metz and Wagner,1981).**

(B) With further filling, intracorporeal pressure rises and imparts axial rigidity to the penis, allowing vaginal penetration. An average fully rigid erection requires 80 to 115 ml. of blood for a 7.5 cm. increase in penile length and may take from several seconds to several minutes to develop **(Newman et al., 1981).**

The maintenance of an erection requires a balance between arterial inflow and venous outflow, tumescence will be maintained as long as arterial inflow meets or exceeds venous drainage **(Eric, 1988).**

In the nineteenth century, venous occlusion was thought to be the main factor in maintaining erection. Later investigations supported a greater dependence on increased arterial flow **(Aboseif et al., 1988).**

Detumescence results when the signals for shunting blood into the cavernosal arteries cease or diminish and venous outflow of the corpora cavernosa exceeds arterial inflow. Like tumescence, detumescence occurs in two phases, with a rapid initial fall in the intracorporeal pressure, followed by a slower decrease in penile circumference **(Eric, 1988).**

Stimuli for erection:

Penile erection is a reflex phenomenon over which man has a little direct voluntary control, that is man cannot will or demand an erection **(Masters and Johnson, 1970).** The forms of stimulation that can reflexly elicit erection have been classified as either: Reflexogenic or psychogenic.

(a) Reflexogenic erection:

Stimulation or excitation of the genital organs, for example light stroking of the glans penis [the sensibility of the glans penis is mainly subserved by the second sacral nerve **(Gunterberg and Petersen,1976)**] or vague interoceptive stimuli arising in the bladder,urethra or rectum may produce

the so called reflexogenic erection via activation of the spinal reflex arc, these stimuli are transmitted through the pudendal nerve to the sacral erection centre in S 2,3,4 segments that complete the reflex are by sending parasympathetic efferents through the pelvic nerve to the penis "nervi erigentes" leading to erection.
The nervi erigentes send impulses via the pelvic nerves and hypogastric plexus to the arteries for circulation in the erectile tissues,the resulting arterial dilatation increases circulation in these tissues and simultaneous venous compression impedes drainage, hence tumescence occurs **(Fitzpatrik, 1975).**

(b) Psychogenic erection:

This type of erection results from stimulation or excitation of the erotic centres of the brain: the frontal cortex, hypothalamus and area for emotions.
This excitation may occur through visual, tactile, auditory or imaginary stimuli **(Fitzpatrik, 1974, Rivard, 1982).**

Psychogenic erection can certainly use the sympathetic erectile pathway. The proof is that many men with complete lesions of the cauda equina or conus medullaris have intact psychogenic erection, despite complete loss of penile sensation and reflex erection.

Almost certainly psychogenic erection can also use the parasympathetic pathway.The evidence is that some men retain good psychogenic erection after radical sympathectomy or after lesions of the hypogastric plexus that appear to be complete.

It is noteworthy that though a complete para-sympathetic lesion or a probably complete sympathetic lesion may leave psychogenic erection intact, more commonly such lesions impair it **(Brindley, 1991).**

Also, psychogenic stimuli such as guilt or hostility often acting at a subconscious level can inhibit the erection reflex **(Farag, 1982).**

It has to be noted that psychogenic and reflexogenic stimuli act synergistically in producing erection and that sacral erection centre receives input impulses from local reflexogenic stimuli as well as from psychic stimuli in higher cortical centres.

The Neurotransmitter:

The neurotransmitters controlling penile erection and detumescence are still under investigation. Recent studies have suggested that vasoactive intestinal polypeptide (VIP)

(which is a smooth muscle relaxant found in the intestine and male genital tract) either alone or in combination with alpha adrenergic blockade or acetylcholine, may be responsible for erection **(Lue and Tanagho, 1987).**