The human sexual response - similarities and differences in the anatomy and function of the male and female genitalia: are they a trivial pursuit or a treasure trove?

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Abstract

The review examines a number of points of similarity and difference between males and females in relation to the human sexual response and the anatomy and functions of their respective genitalia. A variety of topics including foetal sexual differentiation, innervation of the genitalia, penis in erection and flaccidity and corresponding neurotransmitters, pelvic striated musculature, vaginal and clitoral function and respective neurotransmitters, enigma of TRH’s actions, the orgasm, orgasmic pelvic striated and smooth muscle activity, genital secretory activities and the corpus spongiosum in men and women have been examined and specific areas of difference that deserve further study and investigation highlighted. The reward should be a better understanding of how the sexual response mechanisms can differ between the two sexes and allow focus on better gender-based treatments for dysfunctions.
Historical introduction

i) Foetal sexual differentiation

There has always been a fascination to compare and contrast the human male and female genitals that at first sight appear so very different. Despite the obvious external differences, an early anatomical portrayal of the female vagina was surprisingly drawn as being like the male’s elongated penis but turned inside out! However, it was not until quite recent times with the discoveries of the human X and Y chromosomes and the study of the development of the fertilised mammalian egg that the mammalian male and female genitalia differentiation from the same primitive tissue (the genital anlagen) was driven differently by the Y chromosome which encodes a gene referred to as TDF (testis determining factor) which initiates the conversion of the indifferent foetal gonad (the ovo-testis) into a functional testis locally secreting the Antimullerian Factor to prevent the development of the female Mullerian duct system and systemic testosterone to maintain and develop the male Wollfian duct system and the external genitalia (Jost 1973, Wilson 1978). In the absence of the Y chromosome the testis determining pathway is not initiated and the ovotestis develops as an ovary and the foetal differentiation takes the female route. Female development can be regarded as the default model, it is the basic pattern onto which the male differentiation is impressed, even the presence of oestrogen is not thought of as necessary for the female genitalia to develop (Jost 1973, Wilson 1978).

ii) External foetal genital differentiation

Externally the structures of the urogenital tubercle formed the labia and clitoris. The foetal secretion of large amounts of testosterone (and its conversion in the prostate
and penis to the active 5- dihydrotestosterone by the enzyme 5-alpha reductase) however
converts them into the scrotum and penis respectively. The fact that a number of the
underlying structures of the genitals are developed from the same primitive anlagen may
well be useful in understanding the functional control of the male and female genitals so
that rather than being merely an interesting academic exercise in comparing and
contrasting there may be a reward in their study.

Kinsey, Pomeroy, Martin & Gebhard (1953) listed similarities and differences in
the male and female anatomical structures involved in the sexual response. They
summarised their discussion with “In brief we conclude that the anatomic structures
which are most essential to sexual response and orgasm are nearly identical in the human
male and female. The differences are relatively few. They are associated with the
different functions of the sexes in reproductive processes, but (my italics) they are of no
great significance on the origins and development of sexual response and orgasm.”

The Hebrew word “chiddush” has one translation as “finding the new in the old”; this review is an attempt to do just that!

**Innervation of the male and female genitalia**

In both sexes genital innervation was essentially thought to be through the
autonomic nervous system via the parasympathetic and sympathetic supplying the former
with its single neurotransmitter of acetylcholine (blocked by either atropine or
tubocurare) and the latter with nor-adrenaline (blocked by alpha blockers viz
phentolamine and beta blockers viz propranolol). It was simplistically thought that the
two systems worked approximately in opposition, the excitatory parasympathetic being
opposed by the inhibitory sympathetic. Such simplicity had to be abandoned with
discovery of the non-adrenergic, non-cholinergic (NANC) peptidergic and nitrergic
neural systems and when it became apparent that nerves could contain and release more
than one neurotransmitter at their endings. Many putative neurotransmitters were found
to be quite large peptides (VIP, Substance P, Calcitonin Gene Regulatory Peptide
(CGRP), endothelin). The full list would contain near 40 neuropeptides. An even greater
revolution in the concept of peripheral innervation came from the discovery that the gas
Nitric Oxide (NO) was a neurotransmitter. Another complexity that substantially altered
our thinking about how the genitals were controlled came from the concept of
neuromodulation- a substance released by the nerve endings that did not specifically
activate a function but modulated the release or activity of another neurotransmitter.

The rapid application of some of these findings to the innervation of the genitalia
has not been without its problems. Classical neurophysiology demanded a number of
crucial pieces of evidence before the investigator was allowed to conclude that the
candidate neurotransmitter under investigation was the true neurotransmitter of the
organ’s function. These criteria are listed in Table 1. In the rush to characterise the neural
control of the genitals a number of these evidential steps have been bypassed and are
probably now abandoned. A further problem with them now is that gaseous
neurotransmitters such as NO (and carbon monoxide CO) do not fulfil a number of the
criteria viz they are not stored at the site (being manufactured as needed), they are not
enzymatically catabolised and there is no transmitter receptor site (/neuromuscular
junction) to block for anti-NO (CO) agents.
i) presence at the site
ii) synthesis at the site
iii) physiological action when administered at the site
iv) metabolising enzyme(s) at the site
v) blockade by specific antagonists at the site.

Table 1. Classical neurophysiological evidence needed to conclude that a candidate neurotransmitter was a true neurotransmitter at the site. Discovery of gaseous transmitters has made some criteria obsolete.

Penis - erection and flaccidity

i) the ischiocavernosus and bulbocavernosus muscles

One of the earliest suggested anatomic mechanisms for erection was proposed by Varolius in 1573 and involved the contraction of the two striated muscles ischiocavernosus (IC) and bulbospongiosus (BS) at the base of the penis (Gerstenberg, Levin & Wagner 1991). They were supposed to shut off the penile venous drainage and thus cause an erection due to the filling, without emptying, of the cavernosal chambers by the incoming arterial blood. Remarkably, this mechanism stayed quoted but unsupported by laboratory research in the literature for nearly 500 years! Numerous authors and textbooks repeated the mantra that the contraction of the two muscles were the basis for erection. However, when recordings of the emg’s of both muscles during erection to visual sex stimulation were actually monitored it was clear that if no voluntary effort was
made the muscles did not initiate or contract during the erection process (Gerstenberg, Levin & Wagner 1990). Shafik (1995) stimulated separately each muscle electrically while recording the pressures in the urethra and corpora cavernosa. Contraction of the IC created a rise in pressure in the corpora cavernosa while BS contraction only caused pressure in the corpus spongiosum but not in the corpora cavernosa. This confirmed that the BS muscle plays no role in the erection process (but may play a part in ejaculation) while the IC muscle can aid in increasing the corpora cavernosa pressure only when voluntarily contracted. Some men find that by voluntarily contracting the BS and IC muscles they can facilitate their attainment of an erection and transiently increase its rigidity. In sleep the BS and IC muscles appear to become active for nocturnal erections unlike conscious erections. The mechanism for the creation of the former is thus not identical to that for the latter and some care has to be taken in using them as a model for normal conscious erections.

ii) the neurotransmitter for erection

The hunt for the mechanism that change the flaccid urinary penis into the erect sexual one soon dismissed involving acetylcholine as human erections are atropine insensitive. The finding of VIP in genitals led to testing VIP as the neurotransmitter but in fact while VIP satisfied criteria i) and ii) (Table 1) it did not create a true erection when injected into the human corpora cavernosa, it caused a tumescent penis but not a rigid one (Aidaikin, Kottegoda & Ratnam 1986, Wagner & Gerstenberg 1987). Thus it could not be the sole neurotransmitter. After much investigation and a good dose of serendipity the remarkable discovery of the nitric oxidase (NOS)-arginine-NO-cyclic GMP pathway that caused the relaxation of the smooth muscles of the corpora cavernosa
evolved (Burnett, Lowenstein, Bredt, Chang & Snyder 1992) and the complexity of penile tumescence and rigidity became apparent, especially aided by the action of sildenafil in inhibiting phosphodiesterase V the enzyme that catabolised cyclic GMP. The cyclic GMP phosphorylates protein kinase G (PKG) that then phosphorylates numerous ion channels activating Ca\(^{2+}\) channels that hyperpolarize the arterial and cavernosal membranes causing relaxation of the muscles. Most workers now agree that neurogenic NO is the principle agent for penile erection Cartledge, Minhas & Eardley 2001). Subsequent investigation however, has revealed a number other vasoactive agents present in the cavernosal tissues but their roles in creating the erectile and subsequently the flaccid penis are far from being fully understood (Hedlund, Alm & Andersson 2000). We do not know how the long-term mechanisms maintain a flaccid urinary penis. It is accepted wisdom that a high sympathetic (adrenergic) tone keeps the cavernosal smooth muscle contracted because of the n-adrenaline released at the nerve endings and acting on the alpha1 adrenoreceptors. Part of the arousal/erection process is in reducing this tone but this cannot be the whole answer because rare cases are known of males who cannot form n-adrenaline because of lack of the enzyme dopamine-betahydroxylase. These males have a delayed or absent ejaculation and while they have erections they do not suffer from priapism (Mathias, Bannister, Cortelli et al 1990). Clearly other vasoconstrictor mechanisms than n-adrenaline must be utilised in long-term flaccidity possibly involving endothelin 1, angiotensin, thromboxane A\(_2\) and some prostaglandins (PGF\(_2\)).
Vaginal and Clitoral sexual function

Vagina

There is no organ in the male comparable to the vagina, the penis is the homologous organ of the clitoris yet strangely all the early studies on the blood flow of the female genitalia focussed on the vagina and practically completely ignored the clitoris. This may have been due to the relative ease by which the vaginal blood flow could be studied once the photoplethysmograph had been redeveloped (Levin 1997). It wasn’t until the advent of Doppler ultrasound that measured blood velocity (units of cm/sec rather than a true flow of ml/sec) making the monitoring of the clitoral “blood flow” relatively facile. Indeed investigators are still publishing papers advocating the use of such techniques (Khalife & Binik 2003). Its major difficulty at the moment is that the probe has to be hand held at the right angle onto the clitoris creating possible arousal.

i) the neurotransmitter for increasing vaginal blood flow

The suggested role of acetylcholine as the major transmitter for the vasodilation of the vagina in sexual arousal was refuted when Wagner & Levin (1990) showed that atropine (the major antimuscarinic cholinergic antagonist) could not block the increased blood flow during arousal nor stop the orgasm from occurring. Early immuno-histological studies identified the presence of VIP in nerves innervating the smooth muscle and blood vessels of the vagina (Levin 1991) (Table 1, criterion i). Subsequent functional studies in the conscious human female indicated that VIP increased the blood flow to the vagina and could induce the formation of a neurogenic vaginal transudate (lubrication) two features that operated in the vagina of a sexually aroused woman satisfying Table 1, criterion (iii). The strong inference was that VIP was the likely
vaginal candidate neurotransmitter producing the genital arousal by increasing the arterial blood supply creating conditions that would facilitate the formation of tissue fluid (increased arterial hydrostatic pressure) that would enter the interstitial space and ultimately trickle through the vaginal epithelium onto its luminal surface to become the vaginal lubricating transudate (Levin 1999). There have been no human (or even animal) studies to show that antagonists of VIP can alone blockade the increase in vaginal blood flow on arousal and only recently has there been investigations in rabbit vagina of an inhibitor(s) of a neutral endopeptidase (NEP, EC 3.4.24.11) that catabolises VIP (Wayman, Morren, Turner, Naylor & van der Graaf 2002). Its possible therapeutic use in humans to enhance vaginal blood flow is being investigated. Because, as described above, NO was found to be the key activator of the increased blood flow to the penis during sexual arousal it was thought that an identical mechanism of similar importance would be found controlling the vaginal sexual blood flow response. This was proposed despite immuno-histological evidence in human vaginas that there was very little NOS in pre-menopausal vaginal tissues and practically none in postmenopausal (Hoyle, Stones, Robson, Whitely & Burnstock 1996) unlike the rich concentration in the clitoris (Burnett et al 1995). Like the penis the clitoris is an androgen-dependent tissue while the vagina is basically oestrogen-dependent.

The best controlled investigation on the possible influence of the NOS-arginine-NO-guanylate cyclase- cyclicGMP pathway in influencing vaginal blood flow is indirect from the study of Laan, et al (199) who investigated the action of sildenafil (the inhibitor of PDE5 that catabolises cyclic GMP) on vaginal blood flow measured by photoplethysmography in the basal and aroused states. Sildenafil treatment did not
influence the basal flow and only gave rise to a modest 20% increase in the amplitude of the photoplethysmographic signal (vaginal pulse amplitude or VPA). In fact as the VPA is a completely arbitrary index of vaginal blood volume change it cannot be assumed that a 20% increase in VPA means a 20% increase in blood flow *per se*! A more robust measure of vaginal blood flow is needed to quantify the change brought about by sildenafil (and thus indirectly by inference the NOS-NO-cyclic GMP mechanism). Clearly the more modest role of cyclicGMP (and by inference NO?) in influencing vaginal blood flow does not match its basic importance in influencing penile blood flow but that is not unexpected because, as was stated at the beginning of this section, the vagina is not homologous with the penis that honour rests with the clitoris!

One obvious fact that arises from reviewing the innervation of the vaginal peripheral circulation is the lack of follow-up functional studies. While Hoyle et al’s (1996) excellent immunohistochemical study of a host of neuropeptides revealed their location amongst the various blood vessels of the organ the surprising fact is that after seven years we still do not know whether the exposed innervations are of motor nerves or of sensory nerves nor what the various putative neurotransmitters actually do at their respective sites; function lags far behind location!

**ii) the enigma of the action of TRH in women**

One fascinating, and as yet unexplained, difference between men and women is their responses to a central neurotransmitter the tripeptide TRH (Thyrotropin Releasing Hormone) when it is injected intravenously. In both males and females it can give rise to various transient side effects such as facial and body warmth, nausea, urinary urgency and a metallic taste on the tongue. It had no effect on penile erection, the organ remaining
completely flaccid and no activation of any sexual arousal feelings (Levin & Wagner unpublished communication). However, when injected into women the same side effects as in men can occur but in 44% of women (7 out of 16 subjects) transient vaginal warmth, lubrication and pressure similar to that of mild sexual arousal is induced (Blum & Pulini 1980). Levin & Wagner (1986) examined the effects of an intravenous (iv) 200 microgram dose of TRH against a saline placebo injection in 9 female subjects measuring their vaginal blood flow by both photoplethysmography and the heated oxygen electrode. Vaginal warmth was experienced in 7 of the 9 (78%) while 2 of the 9 (22%) had facial but not vaginal warmth. Small increases in power consumption of the heated electrode (an index of increased vaginal blood flow) were noted in 6 of 8 subjects (75%) while saline either caused no effect or a decrease. Unfortunately movement artefacts made interpretation of the photoplethysmographic records difficult, 3 out of 9 (33%) showed clear-cut increases in vaginal pulse amplitude (VPA) while 4 out of 9 (44%) showed increases but these were not significant. The conclusion was that iv TRH can induce small increases in both the blood flow and the feeling of its activation in the human vagina. Acute experiments in anaesthetised sheep also showed that close intra-arterial injection of TRH to the vagina (via the femoral artery) increased vaginal blood flow before circulating around the systemic circulation indicating a direct action at the vaginal level. Thus TRH has a unique action in causing a mild genital and central arousal in women but not in men. No other study of this action has yet been undertaken. Many fascinating questions arise - Is TRH another genital neurotransmitter in the female? Why has it no action in the male? Is the mild sexual arousal caused by the increased activation
of vaginal blood flow or is it because it activates some area of the brain? Is TRH involved in normal sexual arousal in women? There is a harvest here yet to be reaped!

**Clitoris**

The clitoris has been known as a major focus for women’s sexual enjoyment since antiquity (Levin 2001). Despite this long history it is only recently that serious scientific study of the organ has been undertaken. Even its anatomy and histological structure has been poorly researched. Van Turnhout, Hage and van Diest (1995) confirmed by dissection in fresh cadavers that the bilateral vestibular bulbs on either of the vagina terminated into the glans of the clitoris while Toesca, Stolfi and Cocchias (1996) reported that the corpora cavernosa of the clitoris is essentially similar to that of the penis except that there is no subalbugineal layer interposed between the tunica albuginea and the erectile tissue. In the penis this tissue engorges with blood during sexual arousal and becomes compressed against the unyielding tunica creating penile rigidity – a true erection. The lack of this plexus in the clitoris indicates that while the organ can become tumescent or engorged it cannot, like the penis, become stiffly erect. The clitoris thus does not really become erect with sexual excitement but engorged. Occasional papers are published describing clitoral priapism and claiming that the organ has a prolonged erection but it would be more accurate to say a prolonged engorgement (Medina 2002). Another difference between the clitoris and the penis is in the shape, extent and orientation of the suspensory ligaments supporting the two structures (Rees, O’Connell, Plenter & Hutson 2000). O’Connell, Hutson, Anderson & Plenter (1998) published a re-evaluation of the gross clitoral structure based on cadaveric dissections of ten females. They described it as a triplanar complex with a midline corpora (1-2cm wide and 2-4 cm
long) lying in the median sagittal plane that gave rise to the paired crura (5-9 cms long)
lying parallel to the ischiopubic rami and separate bulbs (3-7 cm long, crescentic or
triangular) sitting posterior to the corpora. This complex of erectile tissue surrounds the
urethra. It was claimed that the so-called urethral bulbs did not form the core of the labia
minora as usually portrayed but are part of the clitoral tissue. The actual size of the
clitoral tissue complex was also much larger than usually portrayed but unfortunately the
volume of the tissue was not quantified using modern stereological techniques.

The smooth muscle of the human penile corpora cavernosa is contracted during
flaccidity under the influence of a high sympathetic tone. On sexual arousal the tone is
reduced and the muscle relaxes allowing blood to flow into the erectile chambers. This
change in the function of the smooth muscle (contraction to relaxation) can be monitored
by measuring the electrical myographic activity of the muscle inside the penis using a
concentric electrode, contraction creates spontaneous electrical activity relaxation
produces none. Gerstenberg, Levin & Wagner (1989) were the first to record these
changes in the muscular electrical activity of the human cavernosal smooth muscle in situ
and since their publication a large literature has developed on the subject (Vardi,
have made similar electrical measurements in the clitoris and recorded spontaneous
electrical myographic activity of similar sympathetic tonus as observed in the penis. It
thus appears that both the penis and the clitoris are kept flaccid by a high sympathetic
tone.
The orgasm

Both sexes experience orgasms at the peak of their sexual arousal with similar changes in their respiratory, circulatory and muscular systems and as far as written descriptions with any gender references removed can be held to characterise the activity male and female assessors cannot distinguish whether such descriptions are written by a male or a female (Vance & Wagner 1976). This suggests that the mental experience of the orgasm is essentially similar for males and females. Typologies of orgasms only appear to have been created for females (Levin 1980, Meston, Hall, Levin & Sipski 2003, Mah & Binik 2002) perhaps because there are a number of separate sites that can produce orgasm in women but in men most sexual attention is nearly always focussed on the penis. No comparative descriptions of orgasm produced by stimulation of men’s prostates (per rectum) compared to that produced by penile stimulation (like the previous mentioned study on male and female orgasms) have ever been published. It should always be remembered that absence of evidence for a men’s orgasm typology is not evidence of an absence for a men’s orgasm typology!

There appears, however, to be significant differences in male and female orgastic activity namely: -

i) the female, unlike the male, can have repeated (multiple) orgasms separated by very short intervals (Masters & Johnson 1966),

ii) the female can have an extended orgasm (status orgasmus) lasting for a long time (Masters & Johnson 1966),

iii) with the recorded pattern of pelvic muscular contractions males have a divided rhythmic pattern not seen in women (Bohlen, Held, Sanderson & Alhgren 1982)
iv) if the male orgasm is initiated its expression continues automatically even if the sexual stimuli eliciting it is stopped: with females it is claimed that if the stimuli eliciting the orgasm is stopped the orgasm ceases (Sherfey 1973).

What are the possible reasons/mechanisms for these differences?

In the case of the first, that women can have multiple orgasms while men do not, the explanation lies in the fact that men ejaculate while women normally do not (apart from the claims of those who have urethral discharges). Levin (2003b) has recently discussed critically and in some detail the proposed physiological mechanism of this behaviour which involves the hormone prolactin claimed to be released only at orgasm and not by sexual arousal per se.

At present there does not appear to be any obvious explanation for women having longer orgasms than men and there are no known reasons for the differences in striated muscular activity between men and women in relation to their contractile activity.

i) pelvic striated muscular contractions at orgasm in men and women

In both men and women the pelvic striated musculature is normally activated at orgasm to give rhythmic contractions (Masters & Johnson 1966). In men, these contractions (especially of the BC muscle (Shafik 1995)) power the forceful ejaculatory spurts of semen (Gerstenberg, Levin & Wagner 1990) and are always present concomitant with the orgasm despite having separate mechanisms (Levin 2003). In the case of women however, while many express pelvic contractions of their circumvaginal striated muscles (IC and BC muscles) at orgasm, surveys and individual reports have revealed that a significant number do not experience (perceive?) or have these contractions but yet claim to have orgasms (Bohlen, Held, Sanderson & Alhgren 1982).
Why there is this clear difference between the sexes has been ignored and remains unexplained? It may have something to do with the fact that women can have different types of smooth/striated muscular activity at orgasm as shown in the record by John Perry published in Levin (2001). In this subject, if the anterior vaginal wall is the focus of the sexual stimulation then the contractile activity is expressed in both uterine and PC striated muscles but if the clitoris is the focus much greater activity was observed in the PC striated muscle with little activity from the uterine smooth muscle. Clearly much investigative work in this area needs to be undertaken.

ii) genital smooth muscle contractions during orgasm in men and women

At orgasm in males ejaculation usually takes place at the same time although the mechanisms sub-serving both are different (Levin 2003b). The ejaculatory muscular mechanism consists of adrenergic activated contractions of the smooth muscle in the capsules of the genital accessory organs and peristalsis in the vas deferens and urethra.

These are co-ordinated with the striated muscle contractions that force the ejaculate out under pressure, the smooth muscle contractions are to load up the urethra with the semen for the former. If the striated muscles are paralysed only a dribbling ejaculation occurs.

While there is no female ejaculatory phenomena directly comparable to that of the male (apart from the controversial urethral expulsions - see section on Skene’s glands) some authors have suggested that the equivalent activity are the uterine contractions that occurs at orgasm in women. Moreover, as it is known that the ejaculation mechanism is the cause of the refractory period in males (Masters & Johnson 1966, Levin 2003b) it has been proposed that when these uterine contractions occur in women they occur at their
terminal orgasm switching the female arousal off and preventing further orgasmic activity they thus generate a female “refractory period”.

It is interesting to note that Kinsey et al (1953) and Masters & Johnson (1966) disagreed on the appearance of uterine contractions. According to the former authors “the upper end of the uterus goes into rhythmic contractions of considerable frequency whenever there is sexual arousal” (my italics) but the latter claimed “that specific uterine patterns do not develop unless the individual subject undergoes an orgasmic experience” (my italics). Unfortunately there are simply too few published recordings/data to allow a definitive answer to these interesting speculations and comments.

**Genital secretory activities during arousal**

The genital secretions/fluids of the male involve those from the prostate, seminal vesicles, glands of Littré and Cowper’s or the bulbourethral gland while those of the female are from the uterine (endometrial) glands, the infolded cervical epithelial crypts (not true glandular tissue), the vaginal neurogenic transudate, Skene’s (paraurethral) and Bartholin’s (paired vulvovaginal) glands.

1) **uterine (endometrial) glands**

These glands are obviously unique to women. There is only one study that has investigated the possible effect of sexual arousal on the secretion of the uterine glands. This utilised the insertion into the uterus of a conscious subject of a radiotelemetering pH-measuring capsule (Fox, Colson & Watson 1982). On 10 occasions of coitus there was a significant rise in the intraluminal pH of 0.5 to 0.95 with any form of sexual stimulation and increased to a peak some 2-3 minutes after orgasm and it usually
remained elevated for 30 minutes. Unfortunately, as Levin (1992) pointed out, while these changes may be highly relevant to reproductive processes influencing sperm function by changing the ionic make-up of the uterine fluid, the capsule intra-uterine capsule used was very large, had to be kept in place by an IUD, and could well have created foreign body damage to the endometrial lining during motility caused by the arousal allowing plasma to enter the uterus. Further studies with miniaturised and less damaging electrodes are essential.

ii) Bartholin’s glands

Kinsey et al (1953) claimed “During sexual activity an increase in Bartholin secretions provide one of the best indications of erotic response. Of this fact many observant participants in sexual activities are aware”; they quoted 16 references to support the statement! Yet, Masters and Johnson (1966), who can hardly be called poor observers of the arousal process having studied some thousands of orgasms, reported on the changes in Bartholin’s glands and all they could say was “It is true that Bartholin’s glands do respond to sexual stimulation by secretory activity. However this…. develops only in the late excitement phase or early-plateau phase levels of sexual tension. The nulliparous study subjects rarely produce more than a drop of the mucoid material from each duct. The multiparous occasionally develops 2 or even 3 drops of the material. Under observation, however, there never has been sufficient secretory material produced to accomplish more than minimal lubrication of the vaginal introitus”. The actual fluid that lubricates the vagina, the neurogenic transudate (Levin 1999), is produced practically immediately arousal is induced while that produced by the glands at the stage of arousal just before orgasm would hardly be of much use to lubricate the vagina for coitus!
Masters and Johnson thus completely dismissed the importance of the secretion of the glands in relation to either lubrication or neutralisation of vaginal acidity.

So whose assessment of Bartholin’s glandular function during arousal are we to accept?

Apart from a near ten year-old French review by Chretien & Berthou (1994) there has been no published investigative study of Bartholin’s glands in arousal since that of Masters & Johnson some 37 years ago! The only new interesting fact about the glands that has come to my attention was a personal report from an experimental investigation of sexual arousal in a female subject; no overt Bartholin’s gland secretion was seen but when the observer pressed into the glandular area a significant secretion was expressed! Could it be that Masters & Johnson missed the fact that the glandular secretion is normally expressed during coitus by the pressure of the thrusting penis at the introitus during coitus? We obviously need new observations and experimentation before we completely discount the introital lubricative function of these glands.

**iii) Skene’s (paraurethral) glands**

Kinsey et al (1953) remarked that the prostate and seminal vesicles of the male have embryonic equivalents in the female embryo but that “they never develop in the adult female and do not produce any secretions equivalent to the male”. This unfortunately is a piece of misinformation because about 90% of females do have some developed (vestigial) prostatic tissue which is known as Skene’s glands (Tepper, Jagirda, Heath & Geller 1984) found localised in the urethra, approximately 10% of the glandular tissue being around the area of the bladder sphincter but the main part of the tissue is in the more distal urethra in 66% of women (Levin 2003a). Masters & Johnson did not
mention the glands or their activity in their studies of female sexual arousal but in some women they can produce a small urethral discharge during sexual arousal and especially at orgasm, the so-called “female ejaculation”. The glands have also been linked to the controversial G-spot (Levin 2003a), an area of the anterior vaginal wall said to be highly sensitive to strong digital/penile stimulation (Grafenberg 1950). Zaviacic (1999) has recently published a monograph on the glands and their possible function.

A fascinating pilot study by Santamaria (1997) investigated whether the vestigial paraurethral glands actually secreted in all women during orgasm but that the secretion of many (most?) back-fluxed retrogradely into the bladder and urine rather than becoming expressed through the urethra to the outside. Urine samples were estimated before and after orgasm with an microparticle enzyme immunoassay for the one of the gland’s secretory product Prostate Specific Antigen (PSA). It was found in the post-orgasmic urine in 75% of women after orgasm but not in their pre-orgasmic sample. If this result can be confirmed it may be a possible forensic test/objective marker for orgasm in women. The study needs repeating with a more sensitive immunoradiometric test of the PSA and to see whether orgasm is essential to produce the PSA in the urine or whether just sexual arousal is the cause.

Corpus spongiosum in men and women

In men the softer erectile tissue of the corpus spongiosum surrounds the penile urethra protecting it from being occluded by the hardness of the erect corpora cavernosa and constitutes the erectile tissue of the glans its softness preventing damage to the
female genitalia. The corpus spongiosum system in the penis is a low pressure erectile tissue being engorged with only one third to one half of that in the corpus cavernosum.

In women the corpus spongiosum appears not to be as localised as in the male and has a controversial dispersed distribution. The erectile tissue invested around the urethra has been regarded as spongiosum, also the periurethral mucous membrane (the area surrounding the urethral meatus) as the equivalent of the penile glans (van Turnhout, Hage, & van Diest 1995) and the vaginal bulbs that unite ventrally to the urethral meatus ending in the glans of the clitoris (van Turnhout, Hage, & van Diest 1995).

The penile glans (corpus cavernosum erectile tissue) is acknowledged as the most sensitive part of the human penis while the clitoral glans (of similar erectile tissue make-up) is also thought of as the most sensitive part of the female’s genitalia but the “periurethral glans” has generally been ignored as an important part of the sensitive erotic tissue of the female. It has been suggested, from direct observations of erotic videos of coitus, that in-and-out coital penile thrusting stimulates the periurethral glans (Levin 1991) and, moreover, this may be an important factor in the inducing of orgasm by coitus in some women. No investigation, whether laboratory or questionnaire-based, has furthered the topic.

**Genital reflexes.**

One obvious difference between the male and female genitalia is the number of reflexes that can be elicited from the female’s as compared with the male’s. The various female reflexes are listed in Table 2 in historical order with their name, method of activation by their respective stimuli and their possible sexual roles. They all involve
either changes in pelvic/ or genital muscle activity or changes in genital blood flow. The stimuli used to activate most (but not all) of the reflexes to a very large extent mimic the activity of a thrusting penis during coitus. The fundamental importance of “vaginal tenting” (vagino-levator activity) to delay sperm transport in the reproductive process has been proposed by Levin (2002). All of the reflexes have been described previously in some detail in a review by Levin (2003). The rationale of the review was to examine whether women gained anything from coitus apart from pregnancy? The conclusion, after the characterisation of all the reflexes and their possible effects on the muscles and pelvic/genital blood flow, was that the reflexes gained and maintained vaginal/clitoral and pelvic functionality.

Stimulating the glans clitoris also creates a reflex, the bulbocavernosus reflex (Vodusek & Fowler 1999). It will presumably tighten the vaginal introitus around the inserted penis and create more pleasurable feelings for both coital participants. What of the reflexes in the male? Like the female the bulbocavernosus reflex can be activated by stimulation (tapping) of the glans but it can also be elicited from a number of sites in the male genitalia by electrical means (Vodusek & Fowler 1999). The actual function of the reflex is unlikely to be involved in facilitating/enhancing the rigidity the erection because Shafik(1995) has clearly demonstrated that its contraction does not cause any increase in the corpus cavernosal pressure but only in the urethral pressure (it is a muscle for ejection not erection). Remarkably, there does not appear to be any other fully reported reflex in the male.
<table>
<thead>
<tr>
<th>Reflex</th>
<th>Stimulus/activation</th>
<th>Possible sexual roles</th>
</tr>
</thead>
<tbody>
<tr>
<td>clitoro-pelvic</td>
<td>clitoral vibration</td>
<td>?</td>
</tr>
<tr>
<td>Gillan &amp; Brindley</td>
<td>sustained tonic contraction of pelvic floor muscles</td>
<td></td>
</tr>
<tr>
<td>(1979)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>vagino-cavernosus</td>
<td>vaginal balloon rapid distention transient contraction of bulbocavernosus and ischiocavernosus</td>
<td>Increase tumescence of clitoris</td>
</tr>
<tr>
<td>Shaffik (1993)</td>
<td></td>
<td>“milking” penile urethra</td>
</tr>
<tr>
<td>vagino-levator</td>
<td>vaginal balloon rapid distention levator contracts</td>
<td>creates “vaginal tenting” lifts cervix away from posterior floor</td>
</tr>
<tr>
<td>Shafik (1995)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Reflex</td>
<td>Stimulus/activation</td>
<td>Possible sexual roles</td>
</tr>
<tr>
<td>-------------------------------</td>
<td>--------------------------------------------</td>
<td>-------------------------------------</td>
</tr>
<tr>
<td>Vagino-puborectalis</td>
<td>vaginal balloon rapid distention</td>
<td>?</td>
</tr>
<tr>
<td>Shafik (1995)</td>
<td>contraction of puborectalis</td>
<td></td>
</tr>
<tr>
<td>Vagino-clitoral</td>
<td>vaginal balloon distention insertion and withdrawal</td>
<td>enhanced blood flow to clitoris</td>
</tr>
<tr>
<td>Lavoisier et al (1995)</td>
<td>increase in velocity clitoral blood flow</td>
<td></td>
</tr>
<tr>
<td>Vagino-vesicourethral</td>
<td>vaginal balloon distention</td>
<td>prevents urine leakage during coitus</td>
</tr>
<tr>
<td>Shafik &amp; El-Sibai (2001)</td>
<td>bladder relaxation urethral sphincter contracts</td>
<td>preserving sperm</td>
</tr>
</tbody>
</table>

Table 2. Female genital reflexes showing activation stimuli and possible functional sexual roles. See text for details.
References


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