

ORGASMIC GUSHING: WHERE DOES THE FLUID COME FROM AND HOW IS IT PRODUCED?

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Abstract

There are three sexual fluids from women: lubrication (e.g. transudation of fluid across the mucosa of the vagina, and mucus from the greater vestibular glands); female ejaculation (from paraurethral glands); and gushing. Orthodox western medicine and physiology does not yet have a standardized description or explanation for the third, gushing. The present paper proposes that the gushing fluid is a filtrate of plasma, produced by the mechanism known as transudation. This is an additional application for the transudation mechanism, after the well accepted role in lubrication of the vagina. The present model proposes that the fluid released in a gush arises from the ventral wall of the vagina due to the presence there of increased surface area of mucosa, dilated arterioles during the sex flush that sweeps over the vagina, pressurized venous and lymphatic plexuses, and compression provided by muscle contraction during sexual climax and orgasm.

Key Words : Orgasm, Vagina, Gushing, Female Ejaculation, Sex Fluids

INTRODUCTION

Some cultures recognize three sexual fluids from women including, for example, Tao Zen writers Chia and Chia (1986:51-52), or Trukese and other Micronesian cultures described in works cited by Sevely (1987:68). Orthodox western medicine and physiology recognize two of these sexual fluids. The first, lubrication, consists of mucous secretions from the greater vestibular (formerly Bartholin's) glands and transudation of fluid across the mucosa of the vagina. The second, termed female ejaculation, consists of secretions from the female prostate; the female prostate, also known as the paraurethral (formerly Skene's) glands, releases its secretion through the paraurethral ducts and urethra to the vulva at sexual climax in some women. The third is known in some circles as gushing.

Since Grafenberg's description of the "erotic zone ... on the anterior wall of the vagina" (Grafenberg, 1950), and Ladas, Whipple and Perry's (1982) presentation of the broader biology of the G-spot, a reasonable understanding has been developed of the anatomy and physiology of the paraurethral glands and female ejaculation. Significant advances have included detailed histochemical analyses of the glands and their biochemical machinery for production of such components as the signal chemical, prostate specific antigen (Zaviačič, 1999). Confusion still arises however when the paraurethral glands and female ejaculation are used in attempts to explain all expulsions during female sexual climax, as in Sevely's (1987) detailed review. Gushing is a separate phenomenon. When the two events, female ejaculation

and gushing, are sufficiently understood it should be possible to re-examine many published accounts of female orgasmic expulsions, classify each as either female ejaculation or gushing, and thereby derive much more clear-cut data for each than is currently available.

Gushing is described as thin, watery fluid, with little or no colour, taste, smell or residue. The volume released seems to vary between a few drops, to tens of millilitres to a seemingly limitless volume (e.g. 126 mL, Sevely, 1987:92). The release may occur just once during a coital session, or may occur repeatedly with a series of climax events. When large quantities are released repeatedly some women report indications of dehydration symptoms. The fluid may be released in a gush, that may be projected away from the body in a spurting stream, or may flow, seep or trickle without force. As with female ejaculation, gushing does not occur in all women, and in those who do report the phenomenon it does not occur during all sessions of intercourse i.e. it is an irregular and unpredictable biological event.

THE MODEL

The present paper proposes that the gushing fluid is a filtrate of plasma, produced by the mechanism known as transudation. It is proposed that the fluid released in a gush arises from the ventral wall of the vagina due to the presence there of rich vascular supply, a dense venous plexus, loose connective tissue, and a permeable epithelial membrane. The pattern of flow, described as various forms of gushing, results from a sudden increase in tissue fluid content or pressure that probably results from a wave of vasodilation, reinforced by muscle contractions.

BACKGROUND

This model resolves some of the problems that have hampered acceptance and understanding of the phenomenon of gushing. There has been no organ in the external genital tract known to be capable of producing fluid, on demand, in such quantity. Glands as small as the paraurethral glands cannot. If instead of 'on demand' it was produced over a period of time it would need to be stored somewhere in a vesicle or bladder, but no such structures have been reported by anatomists. No ducts have been found draining into the vagina and yet most descriptions indicate the fluid seems to arise from somewhere on the ventral wall of the vagina, apparently associated with stimulation of the G-spot (female prostate). Some observers however have interpreted the fluid as arriving from the urethra, or being delivered directly into the vestibule. The model resolves the problem of absence of a storage vessel, by proposing that fluid is produced 'on demand'. The problem of 'what organ would be capable of such fluid production' is resolved in the description below, of arteriolar, capillary and epithelial exchange mechanisms of the vagina.

THE MODEL DEVELOPED: SOURCES, PRODUCTION, AND EXPULSION OF FLUID

The model of gushing deriving by transudation is expanded under three headings:

1. source of fluid (anatomy)
2. production of fluid (physiology)
 - a. surface area
 - b. driving force (i. inflow, ii. outflow, iii. compression)

- c. permeability
- 3. expulsion of fluid (sexual climax)

1. SOURCE OF FLUID

The vaginal wall has three main layers: adventitia, muscularis, and mucosa. The mucosa consists of a lamina propria of loose connective tissue and a very permeable, non-keratinized stratified squamous epithelium (Fig. 1). The epithelium is 150-200µm thick, and both the lamina propria and adventitia contain significant amounts of elastic fibre. The mucosa contains few nerve endings, but the adventitia contains many; the latter would be consistent with roles in vascular regulation and sensory monitoring of pressure and stretch.

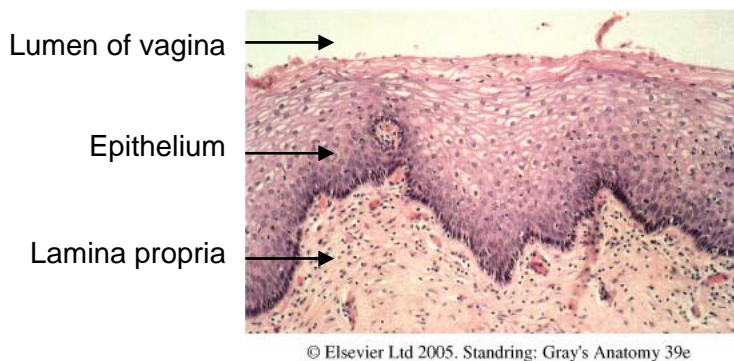


Figure 1: Innermost Layers of the Vaginal Wall

This section through the mucosa of the vagina, shows the non-keratinized stratified squamous epithelium, and the lamina propria beneath, containing numerous blood vessels. Note the papillated interface between the epithelium and underlying connective tissue, providing for stretching of the wall during arousal. Haematoxylin and eosin. (Standring, 2005)

The mucosa has "two median longitudinal ridges on its epithelial surface, one anterior and the other posterior. Numerous transverse bilateral rugae extend from these *vaginal columns*. They are divided by sulci of variable depth" (Standring, 2005:1353-1354). Many anecdotal reports regarding gushing refer to these rugae (Fig. 2) on the ventral vaginal column.

The sulci were previously described as ducts opening onto the wall of the vagina (de Graaf, 1672, translated by Jocelyn & Setchell, 1972). These apparent ducts were individual lacunae, or pit-like recesses in the mucosa. The lacunae add to the surface area draining into the lumen. They are not thought to be connected to the paraurethral glands which are ducts, surrounded with secretory cells, that drain into the urethra (Zavaičić, 1999). De Graaf (1672) pointed out that the ventral wall was so wrinkled that it was effectively two to three times as thick as the dorsal wall, and there were many more rugae in the distal compared with the proximal vagina. He described many tiny pores that lubricate, and discharge copiously during sex, like semen. He was not confusing this with female ejaculate, which he correctly attributed to the female prostate, being expelled via the urethra.

The lamina propria consists of loose connective tissue (also called areolar connective tissue): "like a collapsed sponge this tissue contains innumerable potential spaces ... capable of becoming enlarged and distended with fluid" (Bloom & Fawcett, 1968:131). The wall of the vagina is richly supplied by arteries (Fig. 1), which dilate during arousal; as a result, the lamina propria becomes oedematous. The veins and lymph vessels draining the vaginal wall

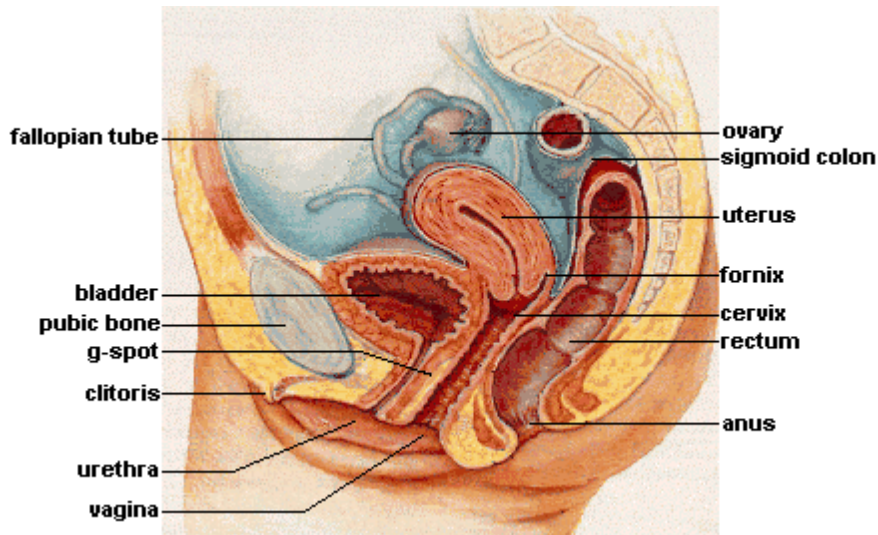


Figure 2: Anatomical Relations of Pelvic Organs in the Human Female

The pubic bone is a feature of the ventral wall (anterior). Behind it (posterior) are the bladder and urethra, G-spot, and vagina with walls that are characterized by horizontal folds called rugae. The proximal vagina receives the cervix; the distal vagina opens to the vestibule or vulva.

are organized as networks i.e. a venous plexus and a lymphatic plexus. The resulting thickening of the vaginal wall (Fig. 3) produces the “vaginal cuff” (or “orgasmic platform”), which provides additional stimulation to the penis during coitus.

The blood supplying the vaginal walls arrives from the internal iliac artery in a number of branches, notably the vaginal branches from the uterine artery, and the internal pudendal artery. The vaginal arteries anastomose to form a longitudinal median azygos artery on the ventral surface and another on the dorsal surface of the vagina, feeding the mucosa (Standring, 2005:1333).

Engorgement of the vaginal arteries during arousal and filling of the venous plexus causes oedema of the mucosa and transudation for lubrication. The present paper posits that further changes in the transudation accompanying sexual climax cause gushing.

TRANSUDATION

Transudation is filtration of water, electrolytes, and some proteins from plasma, across a membrane - usually a serous membrane. The vaginal mucosa is unusual in exhibiting transudation without being a serous membrane. Serous membranes are found in the pericardium, pleura, and peritoneum, where a fine film of serous fluid acts as a surfactant-type lubricant between two apposed membranes. The fluid that diffuses onto the vaginal mucosa during vasocongestion also provides this type of friction modification during sexual intercourse, especially in combination with mucus secreted from the cervical and greater vestibular glands. Glycogen from the epithelial cells would provide further modification to the fluid characteristics. At rest a small quantity of fluid diffuses onto the mucosal surface to maintain hydration. During arousal the lamina propria becomes oedematous and substantial quantities diffuse onto the surface, providing sufficient lubrication for intercourse. At climax,

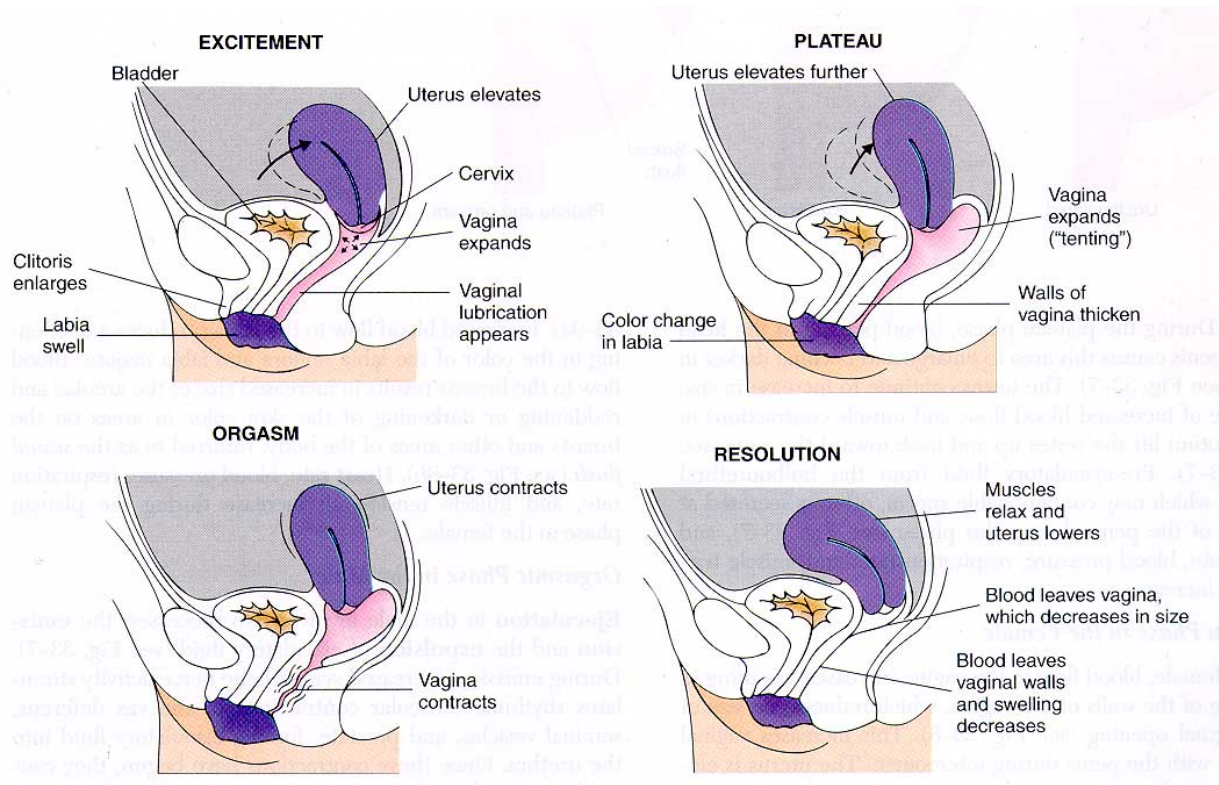


Figure 3: Changes in the External and Internal Genitalia of the Female during the Sexual Response Cycle

These sketches illustrate vaginal lubrication, thickening of vaginal walls, and the vaginal cuff, produced by engorgement of vaginal mucosa; increased surface area created by tenting, contractions during orgasm etc. (Rhoades & Pflanzler, 2003:985 Fig. 33-8)

an additional increase in diffusion can result in free liquid flowing into the vagina or even gushing out of the vagina.

When the feeder arteries and arterioles dilate, the tissue spaces in the lamina propria fill with fluid. When capillary pressure is increased around serous membranes, the rate of transudation across the membrane increases in direct proportion to the increase in pressure (e.g. Stewart et al., 1997). From this it can be predicted that an increase in interstitial hydrostatic pressure at climax would lead to a predictable and measurable increase in transudation into the vagina during the orgasm. The speed with which fluid flux can alter, and the significant quantities of fluid involved, are demonstrated in acute pulmonary oedema: such events as blockage of pleural lymphatic drainage, back pressure from reduced cardiac pumping, or a severe drop in plasma colloid pressure, can result in transudation of one to two litres of fluid into the lungs in 30 minutes, leading to death.

2. PRODUCTION OF FLUID

The chemical composition of gushing fluid needs to be compared with that of serum, plasma, and transudate collected from the same subjects under other conditions. Vaginal fluid produced by transudation at rest is a modified plasma solution. Compared with plasma, vaginal transudate contains: high potassium, low sodium, and high urea (Moghissi, 1979; Wagner & Levin, 1980a); typical plasma proteins - albumins and globulins; and also acetic

acid, lactic acid, and various other volatile fatty acids (Moghissi, 1979). Under conditions of sexual arousal, the rate of fluid production increases and the composition is less modified: the sodium and water reabsorption mechanisms are exceeded (Moghissi, 1979). Similar changes are seen when sweat or serous saliva secretions increase from basal toward maximum flow rates, and mechanisms that would usually reabsorb sodium, secrete potassium, or reabsorb solvent, have little time to act; thus as flow rates increase the proportion of sweat or saliva that is essentially a filtrate of plasma constitutes the major portion of the secretion.

On the basis of the chemical composition of transudate and its change with increased flow during arousal (Moghissi, 1979), the present model predicts that the fluid produced during gushing would exhibit greater change. As the fluid production rate reached maximum it would overwhelm the mucosa’s capacity to modify its composition and consequently it would approximate that of serum. The model needs to describe a mechanism that can deliver a large quantity of fluid across a membrane in a short time. In biological systems, increases in diffusion are achieved by (a) an increase in surface area across which the flux is occurring; (b) an increase in the driving force which, in this case, is a gradient of hydrostatic pressure; or by (c) increasing the diffusion coefficient by, for example, altering the permeability of the membrane.

(a) an increase in surface area across which the flux is occurring

There are three features of the vaginal wall that have the potential to increase the functional surface area. Figure 1 shows the deep corrugations at the interface between lamina propria and epithelium. This feature is shared with urothelium which, as illustrated in Figure 4, allows the mucosa to stretch, transferring the larger area to the luminal surface of the epithelium, and concomitantly thinning the distance across which the fluid must diffuse (discussed further at (c) below). Secondly, the rugae of the ventral wall (Fig. 2) effectively store surplus surface area that can become available by flattening of the folds as a result of movements, muscle contractions, or development of localised oedema. Thirdly, the proximal vagina “tents”, or “balloons” (Fig. 3) as the region toward the cervix expands its surface and flattens the mucosa.

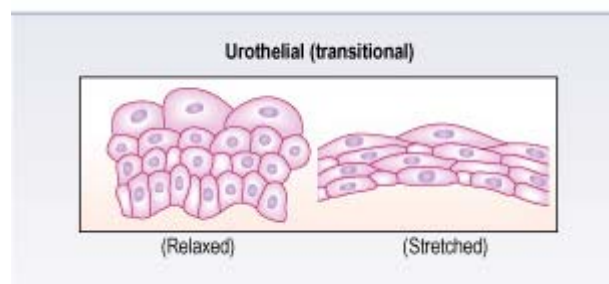


Figure 4: Urothelium Becomes Thinner When It Is Stretched

Stretching urothelium increases its surface area and reduces the thickness of the membrane, increasing the rate of diffusion across the membrane. (Standing, 2005)

(b) an increase in the driving force

Transudation occurs across a permeable membrane. It basically consists of the water content of plasma, or an electrolyte solution, which moves by bulk flow-type flux when vasocongestion occurs. The pressure gradient can be increased by increased arterial pressure

or rate of inflow of blood; increased venous pressure or disruption to outflow of blood or lymph; or squeezing of the tissue spaces as would occur during contractions of skeletal muscle, smooth muscle, or myoepithelial cells dispersed through the lamina propria.

Increased venous pressure increases transudation at serosal membranes such as the pleura or pericardium, so it can be predicted to be a major contributor in the vagina also. Certainly venous pressure increases during the vasocongestion that causes erection. Thus transudation sufficient for lubrication accompanies erection. What would cause the additional flow called gushing? There would need to be sudden increases in tissue interstitial pressure. Hydrostatic pressure results from an imbalance between inflow and outflow. Hence any sudden increase in inflow that was not matched with an increased outflow would significantly elevate hydrostatic pressure in the tissue spaces, increasing flux across the mucosal wall. In the pericardium this mechanism is tested systematically by elevating myocardial venous pressure and measuring increases in epicardial transudation /pericardial effusion (Stewart et al., 1997).

i) What would cause a sudden increase in pressure or rate of inflow? The basic hydrostatic pressure of the feeder arteries is established by systemic systolic blood pressure. In humans, blood pressure rises dramatically at the time of orgasm: Figure 5 shows an example in which systolic blood pressure of a woman doubled during each orgasm (Fig. 5). This elevated blood pressure significantly increases the driving force for the transudation mechanism.

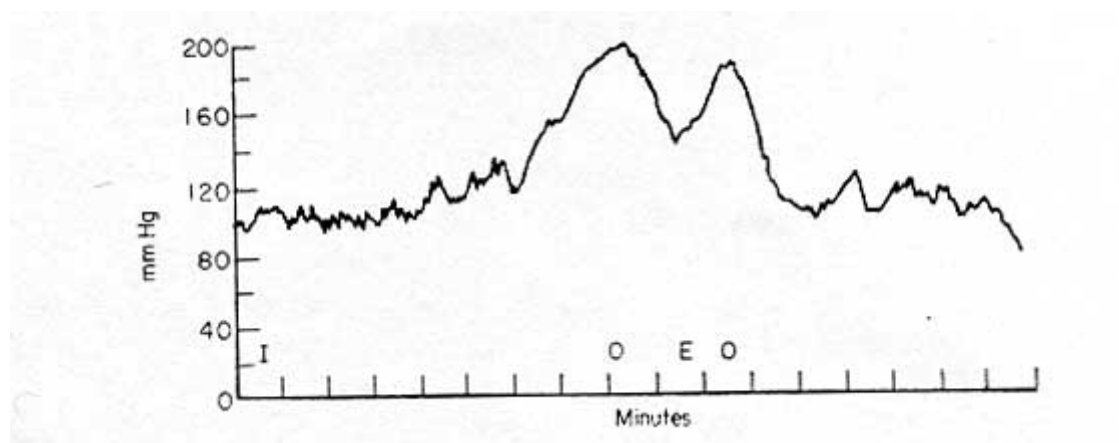


Figure 5: A Recording of Systolic Blood Pressure During Coitus in One Human Female Subject.

I = intromission achieved; O = female orgasm; E = male ejaculation. (Fox & Fox, 1971:323 Fig. 10)

Inflow is provided via the longitudinal median unpaired arteries of the vagina and thence arterioles of the mucosa. A wave of hyperpolarization such as that causing release of nitric oxide would temporarily decrease arteriolar tone and increase flow into the capillaries feeding the interstitial fluid of the vaginal wall. Nitric oxide has a characteristically brief action before being metabolized, thus a spurt or gush of fluid would be released as a discrete event. Nitroergic neurons have been identified in human vagina associated with blood vessels (Hoyle et al., 1996).

The skin flush that often characterizes sexual climax is achieved by a sudden wave of peripheral vasodilation (reminiscent of the hot flushes of menopause). Is gushing a localized

outcome of "vaginal flushing"? A sex flush of the vagina? Such vasodilator effects are possibly mediated via cholinergic sympathetic outflow from the anterior hypothalamus (sympathetic vasodilator system, Guyton & Hall, 2006:207-208) as occurs to cause the sudden "flush" and sweating when a fever reaches its "crisis" (Guyton & Hall, 2006:899), or inhibition of sympathetic vasoconstrictor output from the posterior hypothalamus (Guyton & Hall, 2006:895). Parasympathetic mechanisms are also possible: rapid and copious fluid flow occurs in the stomach in response to a long vagovagal reflex. In this gastric reflex (Guyton & Hall, 2006:798-799) sensory information is transmitted in the vagus nerve, to the brainstem, and back to the stomach also in the vagus nerves. The reflexes are initiated by distension of the stomach mucosa, and also by tactile stimulation of the surface of the stomach mucosa. This gastric secretion can also be initiated by signals from the brain, especially from the limbic system. These reflex components bear remarkable similarity with the phenomena of interest in the vagina except that vaginal reflexes would act via sacral pathways. Although the central origin of the innervation has not been clarified, the peripheral synapses have been found to be not atropine sensitive (Wagner & Levin, 1980b), so the responses are presumed to be mediated by VIP¹ (Levin, 2002:4008).

ii) Reduced outflow also increases interstitial pressure and thus transudation. Tonic contractions of the vaginal wall could easily close the venous plexus and prevent the egress of blood, as occurs during penile erection. In a study of oxytocin secretion during orgasm, Carmichael et al. (1987) observed large fluctuations in blood flow with each phasic contraction of genital musculature. The blood flow was monitored using anal photoplethysmography (Carmichael et al., 1987). The mechanism being referred to here is observed readily in the lung where, if venous or lymphatic drainage fails causing even slight increase in interstitial pressure, tissue fluid crosses into the alveolar air space causing pulmonary oedema. The serous membrane of the pleura demonstrates similar high flux of fluid if challenged by significant back-pressure (Guyton & Hall, 2006:489-490). In erectile tissues venous outflow can be blocked by compression of veins, or by vasoconstriction of vessels draining the tissue. Hoyle et al. (1996) identified NPY¹ innervation of the vaginal blood vessels that could regulate appropriate vasoconstriction (Levin, 2002).

iii) In addition to shutting down the veins and lymphatics, contractions of the vaginal wall would squeeze the mucosal "sponge"; this would raise interstitial pressure further during climax. It would squeeze fluid out, and into the vaginal lumen, as can be achieved by manual compression in an aroused subject. During orgasm it could be achieved by contractions of the smooth muscle of the fibromuscular walls of the vagina, or by contractions of the surrounding skeletal muscles such as the bulbospongiosus. Vaginal plethysmography, used to monitor vasocongestion of the vagina, show slight decreases in vasocongestion associated with contractions of pubococcygeus muscles whether they occur during climax (see Fig. 6 below, from Perry, 1999a) or during Kegel exercises (Perry, 1999b) and this is consistent with fluid being squeezed out of the tissue during the contractions. The substantial drop in vasocongestion once orgasm had been reported by the subject recorded in Figure 6 (Perry, 1999a) also implies that substantial fluid was squeezed out of the tissue by the strong pelvic muscle contractions of the sexual climax, leading to a decrease in the oedema of the tissue.

(c) Increasing the permeability of the membrane

Rapidity of fluid flux across a membrane is greatly enhanced if the membrane becomes thinner (see Fig. 4) or more permeable. Hoyle et al. (1996) identified CGRP¹-containing neurons in the vaginal mucosa, and speculated a role for them in regulating capillary

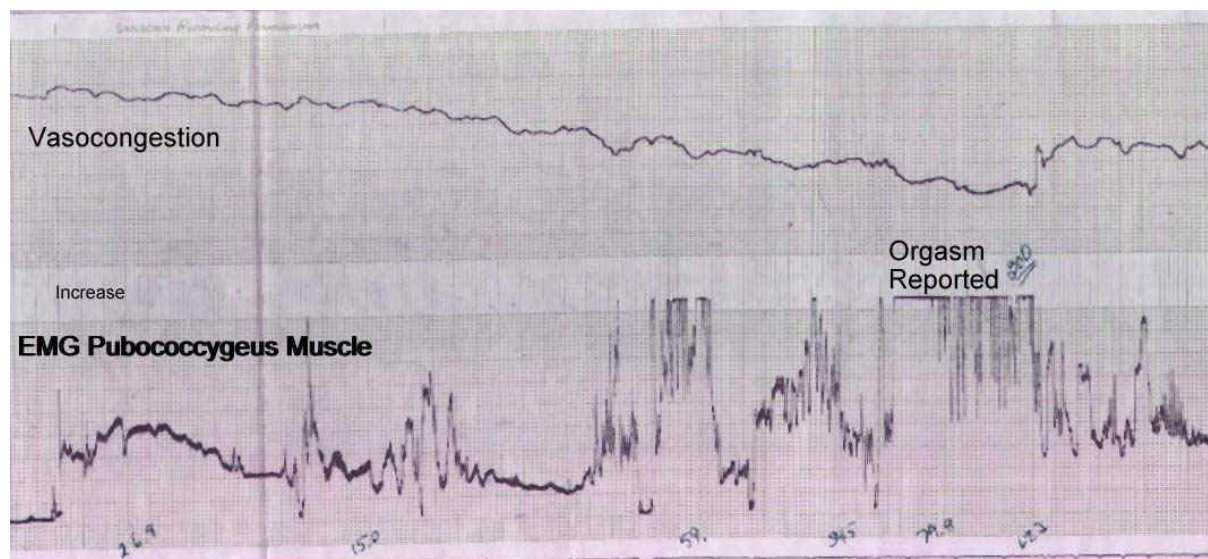


Figure 6: Vasocongestion and EMG Activity During Masturbation

In the recording above, vasocongestive and electromyographic (EMG) activity are shown as detected by a combination VPG/EMG vaginal sensor. Higher readings are towards center of the chart. Vaginal blood flow increases during masturbation until they reach a peak during reported orgasm and both drop off. Note that short EMG peaks (as over the "M" of Muscle) result in a momentary measured or apparent decrease in bloodflow. This subject had very strong pelvic muscles. Note a peak (momentary) reading of 200 microvolts (RMS) that was manually recorded. Ten-second averages shown are 26.9, 15.2, x, 59, 34.5, 79.8, and 69.2. (April, 1979) (Perry, 1999a)

permeability. Capillary permeability can be altered neurally by inducing slight changes in electrical membrane potential of the capillary endothelial cells. Water and electrolyte secretion by glands of the gastrointestinal tract occurs by parasympathetic activation, causing hyperpolarization of cell membranes and chloride influx, ultimately driving water and electrolyte flux on the luminal side of the epithelial cells (Guyton & Hall, 2006:793). This mechanism is used to increase flow through cholinergically innervated gastric and salivary glands and can induce 20-fold changes in rates of fluid flow (Guyton & Hall, 2006: 793-794). Levin and Wagner (1978) used the membrane potential and electrolyte concentrations in the vagina to confirm the significant potassium transport mechanism there i.e. electrolyte flux across the vaginal mucosa is regulated, not passive (at least, not at rest). The question of how the electrolyte flux is regulated, appears to have been answered in the immunohistochemistry study by Hoyle et al. (1986), who found nerves containing CGRP, as well as Nitric Oxide (identified via NOS), VIP and NPY associated with blood vessels in the human vagina.

Levin (2002) referred to this electrolyte-involved mechanism as the neurogenic transudate, which provides fluid beyond that due to the hydrostatic pressure changes described above. "The ultrafiltrate trickles through the intercellular spaces of the vaginal epithelium saturating the limited reabsorptive Na^+ transfer capacity of the cells ..." (Levin, 2002:408-409). The thinning of the epithelium illustrated above (Fig. 4) would facilitate this diffusion of fluid.

Thus, the increased surface area, increased imbalance between inflow and outflow of blood, and increased permeability of the mucosa, would each add to the delivery of a pulse of fluid at orgasm.

3. EXPULSION OF FLUID

Expulsion of the gushing fluid can occur with or without significant momentum. The mechanisms that *expel* the fluid are probably separate from the mechanisms that *produce* the fluid. The most likely source of propulsion is contraction of vaginal musculature and associated perineal muscles especially the pubococcygeus muscles that are typically involved at climax. This would be assisted by general bearing down movements, and by contraction of myoepithelial cells through the loose connective tissue. Any pooling of fluid that occurred in the vagina during coitus would add to the volume expelled at climax.

As described in the preceding section though, these contractions would also be contributing to production of the fluid by pressurizing the venous plexus and squeezing the mucosa.

WHY THE VENTRAL WALL?

There are several explanations for the ventral vaginal column being more involved in gushing than the dorsal column. As de Graaf (1672) pointed out, it can be several times thicker than the dorsal wall. The urethra is embedded in the ventral wall (Fig. 7). Erection of the corpus spongiosum around the urethra can cause the urethral tissue to bulge into the vagina, enhancing the visibility and palpability of the ventral wall. The ventral wall also contributes to anchoring the external urethral sphincter. This anchoring would tend to stabilize the ventral column, such that contractions of adjacent muscles would squeeze it rather than passively drag it. The female prostatic tissue in the wall of the urethra is distributed dorsally, toward the vagina (Zaviačič, 1999 and Fig. 7B) and the region of special sensitivity known as the G-Spot is associated with it (Fig. 2). This sensitivity probably encourages humans to select coital positions that enhance direct stimulation to the ventral wall of the vagina, further increasing the likelihood that gushing mechanisms will be activated.

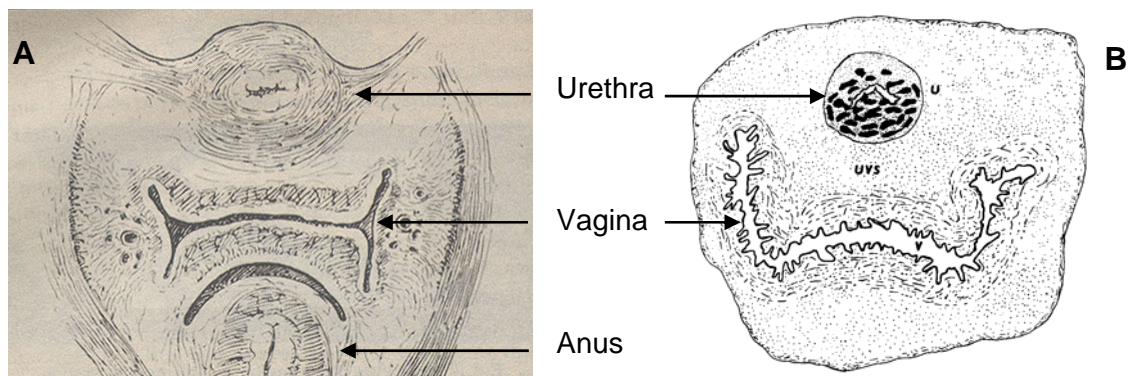


Figure 7: The Urethra Is Embedded in the Ventral Wall of the Vagina

A. Section through the mid-level vagina (Sevely 1987). **B.** Section through the distal vagina; prostatic tissue is distributed around the dorsal and dorso-lateral urethra, toward the vagina. U urethra; UVS urethro-vaginal septum; V vaginal canal (Zaviačič 1999).

EXTRAPOLATION TO LARGE VOLUME FEMALE EJACULATE

Development of the present model for orgasmic gushing was inspired by the attempts of people to explain the very large volumes of fluid expelled by some women during orgasm. The female prostate (paraurethral) glands could not produce such large volumes on demand, since they use merocrine and apocrine secretory mechanisms (Zaviačič, 1999:42). Prior production of secretions would require storage. It is unlikely that an organ large enough to store such quantities has been overlooked by anatomists for thousands of years. The only structure with suitable capacity and access to the exterior would be the urinary bladder; descriptions of the experiences (anecdotal evidence) precludes the urinary bladder as the source, due to the regularly reported capacity of women to produce normal urine immediately before and after expulsions of orgasm, and the high PSA (prostate specific antigen) and low urea concentrations of the fluid (Zaviačič, 1999:80). The only solution would be for an organ with the capacity to produce large quantities of fluid *on demand*. The present paper describes this well understood mechanism in the vagina.

Despite the capacity of the vagina to produce large quantities of fluid on demand, most anecdotal evidence indicates that the fluid is most often (but not always) observed being expelled via the urethra, not the vagina. Could the same mechanism exist in the urethra? Possibly. The transitional epithelium at the neck of the bladder, in the region known as the trigone, is commonly replaced by vaginal-type epithelium in women but not in men (Standring, 2005:1292). It remains for the histological anatomy and physiology of the urethra, including lacunae, to be investigated. The newly developed model will provide a simple framework to use when investigating questions of whether the mechanism described herein for orgasmic gushing from the vagina could also explain expulsion of large volumes of sexual fluid from the urethra during sexual climax. If appropriate microstructures are found, it will not be necessary to postulate a new mechanism for production of the thin, watery secretion that has been reported – the mechanism is already understood (this paper) – all that is needed is to check whether this mechanism can occur in the urethra. Investigation should explore the mucosa that lines the lacunae along the urethral walls, in regions that are not occupied by prostatic tissue.

CONCLUDING STATEMENT

The model proposed here, in summary, is that gushing probably results from a sex flush washing through the vagina, increasing blood input to the mucosa, accompanied by contractions of the vagina that increase the pressure in the venous plexus; these factors combine to increase transudation across the mucosa. Transudation is probably re-inforced or enhanced by the contractions effectively squeezing fluid out of the lamina propria and lacunae, and sometimes forcing the fluids out of the vagina under pressure.

LE JAILLISSEMENT ORGASMIQUE: D'OÙ VIENT LE FLUIDE ET COMMENT EST-IL PRODUIT?

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Résumé

La femme produit trois fluides sexuels: un lubrifiant (p.ex. transsudation de fluide à travers la muqueuse du vagin, et mucus des glandes du grand vestibule); l'éjaculas de la femme (des glandes paraurétrale); et un fluide jaillissant. La médecine et la physiologie orthodoxe occidentale n'a pas encore établi une description ou une explication standardisée pour le troisième, le fluide jaillissant. Ce manuscrit propose que le fluide jaillissant soit un filtrat du plasma produit par un mécanisme connu sous le nom de transsudation. Ceci est une application supplémentaire du mécanisme de transsudation, après les rôles bien établis de lubrification du vagin, et la production de fluides séreux. Le modèle présenté suggère que le fluide libéré lors du jaillissement provienne de la paroi ventrale du vagin suite à la présence à cet endroit d'une superficie de muqueuse plus importante, d'artérioles vaginale dilatées durant l'excitation sexuelle, de l'augmentation de pression au sein des plexus veineux et lymphatiques et de la compression résultant de la contraction musculaire au cours de la jouissance et de l'orgasme sexuel.

Mots clés : Orgasme, Vagin, Jaillissement, l'éjaculas de la femme, fluides sexuels

ORGASMIC 'GUSHING': WOHER KOMMT DIESE VAGINALFLÜSSIGKEIT UND WIE WIRD SIE PRODUZIERT?

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Zusammenfassung:

Frauen produzieren während des Koitus und Orgasmus drei verschiedene Flüssigkeiten: Vaginalsekret zur Lubrikation (durch Absonderung oder 'transudation' von Flüssigkeit über die vaginale Schleimhaut und Schleim von den Vestibulardrüsen oder Bartholin-Drüsen), weibliches Ejakulat der paraurethralen Drüsen, und 'gushing'. Diese dritte Flüssigkeit 'gushing' ist bisher in der orthodoxen, westlichen Medizin und Physiologie noch nicht beschrieben oder erklärt worden. Die vorliegende Abhandlung schlägt vor, dass die 'gushing' Flüssigkeit ein Filtrat des Blutplasmas ist und durch 'transudation' in der Vagina entsteht. Die Rolle der Vagina in der Erzeugung von Vaginalsekret und seröser Flüssigkeit zur Lubrikation ist vielfach beschrieben und erwiesen. Die Entstehung der 'gushing' Flüssigkeit

durch 'transudation' wäre eine zusätzliche Applikation für diesen Prozess. Das vorliegende Modell schlägt vor, dass die im 'gushing' freigesetzte Vaginalflüssigkeit von der ventralen Scheidewand entspringt. Gründe dafür sind die größere vaginale Schleimhautoberfläche, die erhöhte Durchblutung der Vagina während des sex flushes, die vermehrte 'vasocongestion' in den venösen und lymphatischen Plexen, und die Muskelkontraktionen während des sexuellen Klimax und Orgasmus.

Schlüsselwörter: Orgasmus, Vagina, gushing, weibliches Ejakulat, Vaginalflüssigkeiten

IL "GUSHING" DURANTE L'ORGASMO: DA DOVE VIENE IL LIQUIDO E COME VIENE PRODOTTO?

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Abstract

Nelle donne sono presenti tre liquidi sessuali: la lubrificazione (per es. la congestione del liquido attraverso la mucosa della vagina e il muco dalle ghiandole vestibolari più grandi); la eiaculazione femminile (dalle ghiandole parauretrali); e il "gushing". La medicina e la fisiologia tradizionali occidentali non hanno ancora trovato una definizione standard per il terzo. Il presente articolo propone che il liquido generato durante il "gushing" sia un filtrato del plasma, prodotto attraverso il meccanismo di congestione. Questa diventa allora un'ulteriore applicazione del meccanismo di congestione, oltre al ruolo ben accettato nella lubrificazione della vagina e nella generazione dei liquidi sierosi. Il presente modello propone che il liquido rilasciato durante il "gushing" venga prodotto dalle aumentate pareti della mucosa, dalle arteriole dilatate durante il rossore che investe il corpo e anche la vagina, dai compressi plessi venosi e linfatici, e dalla pressione apportata dal muscolo contratto durante l'eccitamento sessuale e l'orgasmo.

Key Words : Orgasmo, Vagina, Gushing, Eiaculazione femminile, Liquidi sessuali

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FOOTNOTES

¹ Abbreviations: CGRP calcitonin gene-related peptide; NOS nitric oxide synthase; NPY neuropeptide Y; VIP vasoactive intestinal peptide.

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