Is female sexual dysfunction receiving proper clinical attention? : a clinical review on female sexual dysfunction

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Table of Contents

Introduction..........................................................................................................................4
Background Information......................................................................................................7
Etiology/Pathogenesis........................................................................................................11
Epidemiology and Risk Factors.........................................................................................16
Manifestations....................................................................................................................18
History and Physical Exam................................................................................................21
Treatment ...........................................................................................................................23
Conclusion .........................................................................................................................30
References..........................................................................................................................32
Abstract..............................................................................................................................37
Is Female Sexual Dysfunction Receiving Proper Clinical Attention?

A Clinical Review on Female Sexual Dysfunction

Sexuality is a crucial part of every person’s life (Berman, Shuker, and Goldstein, 1999). It is not only a means by which humans procreate, but it is also a means of pleasure. Unfortunately, not everyone is finding pleasure in sexual experiences. It was estimated that approximately one out of every four patients consulting a primary care provider for any illness suffers from a sexual dysfunction (Spera, Pili, Gnessi, Spera, and Mariani, 2003). In 1999, the National Health and Social Life survey found that 43% of females and 31% of males aged 18 to 59 years reported sexual difficulties (Hwang, 1999). Moreover, another recent study showed that the percentage of women reporting sexual problems exceeded men in almost every aspect of sexual function and performance (Earle, 2003).

These sexual difficulties that women are having are collectively known as female sexual dysfunctions. Female sexual dysfunction (FSD) is a broad classification of disorders that is succinctly defined by the Diagnostic and Statistic Manual of Mental Disorders (2000) as “a disturbance in sexual desire and in the psychophysiological changes that characterize the sexual response cycle and cause marked distress and interpersonal difficulty.” In other words, sexual dysfunction in women is defined as disorders of sexual desire, arousal, orgasm and/or sexual pain, which results in significant personal distress and may have an impact on the quality of life (Munarriz, Kim, Pauls, Traish, and Goldstein, 2004). From this definition, FSD has been further classified by the Report of the International Consensus Development Conference on Female Sexual
Dysfunction (Basson, Berman, Burnett, Derogatis, Ferguson, Fourcroy, et al., 2000) into four subgroups consisting of sexual desire disorders, sexual arousal disorders, orgasmic disorder and sexual pain disorders.

This classification system can be further divided into subtypes including lifelong versus acquired, generalized versus situational and etiology. In addition, a physiological condition that causes a female sexual dysfunction can exacerbate a psychological condition, further complicating the clinical scenario (Berman, Shuker, and Goldstein, 1999). This creates an overlap of the problems at hand, making diagnosis and treatment much more complex.

Therefore, to make an accurate diagnosis of FSD, the clinician must use a careful thought process. Unfortunately, most clinicians have only a small amount of proper training on how to elicit a thorough sexual history and little exposure to the current treatments (Polonsky, 2001). A recent survey of North American medical schools found that the majority (61%) provided 10 hours or less of human sexual education, 17% provided 11-19 hours of instruction while a scant 15% mandated 20 hours or more (Leiblum, 2001). A more recent study done in 2003 showed similar results to Leiblum’s study, stating that only 54.1% of the medical schools provided three to ten hours of sexual education and pointed out that only 44.6% of medical schools offered courses for continuing medical education (Solursh, Ernst, Lewis, Michael-Prisant, Mills and Solursh, 2003).

A survey done on a patient’s comfort level when speaking with physicians about sexual issues survey showed that 71% of patients thought their doctor would dismiss any of their concerns over sexual issues (Marwick, 1999). In addition, 68% of patients stated
that they feared embarrassing their physician by bringing up sexual problems (Marwick, 1999). This survey shows inadequacies in the clinician-patient relationship.

It is clear that there is a public desire for professional help concerning sexual issues, which means there needs to be some expanded efforts made by clinicians to further their knowledge on sexual education (Solursh et al., 2003). The purpose of this clinical review is to outline the current information on female sexual dysfunction. This review will go over the normal sexual response cycle and the four different types of FSD. It will also cover the many etiological causes and pathogenesis of FSD including medical and psychological issues. In addition, this article will focus on specific clinical manifestations. This paper will further explain the work-up a patient would go through to rule out a physiological cause of FSD, along with giving guidance on how to illicit a thorough sexual history from the patient. This review will guide the clinician in therapeutic management of the FSD patient, whether it is pharmacological (traditional medicine and herbal supplements) or non-pharmacological (vaginal dilators, individual and marital therapy). Overall, this review’s purpose is to ease the primary care provider’s decision-making process on the issue of female sexual dysfunction.
**Background Information**

In order to understand the causes of FSD, it is essential to know the female anatomy involved and how this anatomy responds to a sexual stimulus. The female anatomy involved for sexual intercourse predominately focuses on the external genitalia including the clitoris and vaginal vault.

The clitoris and vestibular bulbs are the erectile tissue for the female. The clitoris is located immediately behind the anterior labial commissure, while the vestibular bulbs are coupled structures that lie along the vaginal opening under the skin of the labia (Naughton, 2003). Innervation for the clitoris comes from the pelvic and hypogastric plexuses, along with the uterovaginal plexus which carries sympathetic (T1-L3) and parasympathetic (S2-S4) fibers (Naughton, 2003). Somatic sensory innervation of the clitoris arises in the skin and proceeds through the dorsal nerve of the clitoris, where it continues within the pudendal nerve to arrive at the sacral spinal cord (Moore, 1992). The iliohypogastric pudendal artery provides the inflow of blood to the clitoris (Naughton, 2003).

The vaginal wall consists of three layers: the mucous layer, the muscular layer and the fibrous layer. The aglandular mucosa is made up of stratified squamous cell epithelium that is affected by hormonal changes (Berman, Shuker and Goldstein, 1999). The muscular layer consists of smooth muscle and a vast vascular bed, which may swell during sexual activity (Berman, Shuker and Goldstein, 1999). Finally, the fibrous layer encompasses the mucosal and muscular layers to provide structural support to the vagina; this layer consists of elastin and collagen fibers that allow for enlargement of the vaginal vault during sexual arousal or childbirth (Berman, Shuker and Goldstein, 1999).
The vaginal innervation is similar to that of the clitoris in that autonomic innervation comes from the hypogastric and sacral plexus, along with the uterovaginal plexus containing both the parasympathetic and sympathetic fibers (Naughton, 2003). The pudendal nerve is noted to be responsible for somatic sensory innervation (Naughton, 2003). There are also more nerve fibers in the anterior, distal aspect of the vagina (Hilliges, Falconer, Ekman-Ordenberg and Johansson, 1995). In addition to the nerve supply, the arterial blood supply to the vagina comes from an extensive network of vessels consisting of the uterine, hypogastric, middle hemorrhoidal and clitoral arteries (Naughton, 2003).

To initiate the physiology of female sexual response, there must be some sort of sexual stimulus or arousal. During the resting state, clitoral corporal and vaginal smooth muscles are contracting (Munarriz et al., 2004). After sexual stimulation, neurogenic and endothelial release of nitric oxide (NO) plays a prominent role in clitoral cavernosal artery and helicine arteriolar smooth muscle relaxation (Munarriz et al., 2004). Following this event, there is an increase in clitoral cavernosal artery inflow, an increase in clitoral intracavernosal pressure, and clitoral engorgement (Munarriz et al., 2004). The effect is extrusion of the glans clitoris and enhanced sensitivity (Munarriz et al., 2004).

In continuation of the response, the vaginal epithelium reabsorbs sodium from the submucosal capillary plasma transudate (Munarriz et al., 2004). Several neurotransmitters, including NO and vasoactive intestinal peptide (VIP), are released regulating vaginal vascular and non-vascular smooth muscle relaxation (Munarriz et al., 2004). Substantial increase in capillary inflow in the submucosa surmounts sodium
reabsorption leading to three to five milliliters of vaginal transudate, enhancing lubrication (Munarriz et al., 2004). Relaxation of the vaginal smooth muscle results in increased vaginal length and luminal diameter specifically in the distal two-thirds of the vagina (Munarriz et al., 2004).

Although the intricate details of how each neurotransmitter operates during the sexual response cycle are not known, there is a basic understanding (Brackett, Bloch, and Abae, 1994). Nitric oxide is thought to be a key player in the sequence of events because as sexual stimulation occurs, nitric oxide is released from the endothelial cells in the pelvic arteries (Ahmed, 2001). Nitric oxide is thought to aid in smooth muscle relaxation and therefore vasodilatation (Ahmed, 2001), which allows the clitoral and vaginal muscles to become engorged with blood causing hypersensitivity (Munarriz et al., 2004). VIP is also thought to be an important neurotransmitter as it is responsible for vaginal vasodilatation and the formation of lubricating fluid during sexual arousal (Berman, Shuker and Goldstein, 1999). In addition, it is thought that sexual arousal is repressed by the sympathetic nervous system, whereas the parasympathetic nervous system elicits and maintains sexual arousal (Berman et al, 1999). It also appears that dopamine is an activating neurotransmitter for sexual activity, while serotonin is an inhibitor (Fourcroy, 2003).

Hormones must also be noted in the sexual response cycle, specifically estrogen and testosterone since there are numerous estrogen and androgen receptors in the clitoral and vaginal tissues (Berman and Bassuk, 2002). Estrogen appears to play a role in vaginal wall thickness and lubrication (Berman, Shuker and Goldstein, 1999). Androgens, specifically testosterone, are thought to have a positive effect on libido,
although there have been no studies that have shown a clear association with testosterone levels and a satisfactory sexual life (Shifren, 2004).

Masters and Johnson (1966) organized all of this physiology into a four-step model that consists of excitement phase, plateau phase, orgasmic phase and resolution. They stated that any sort of somatogenic or psychogenic stimulation initiates the first phase. If an adequate amount of sexual tension existed from this stimulation, then the cycle would progress to the plateau phase (Masters and Johnson, 1966). Here sexual tensions intensify and later reach the threshold from which the individual may have an orgasm. Keeping in mind that the main response to sexual stimuli is widespread vasocongestion, and the secondary response is a generalized increase in muscle tension, the orgasmic phase lasts only a few seconds during which the vasocongestion and muscle tension are released (Masters and Johnson, 1966). The last phase, or resolution, is a reverse reaction where the individual returns through the plateau and excitement phase to an unstimulated state (Masters and Johnson, 1966).
Etiology/Pathogenesis

Hormonal/Endocrine Issues
Some of the most common causes of hormone-related female sexual dysfunction are dysfunction of the hypothalamic-pituitary axis, surgical or medical castration, natural menopause, premature ovarian failure, and chronic birth control pill use (Berman, Berman and Goldstein, 1999). Estrogen deficiency alone can cause “loss of collagen and adipose tissue in the vulva, attenuated maturation of vaginal epithelial cells, thinning and loss of premenopausal ridges, bleeding and ulceration of the vaginal epithelium after minor trauma, delayed onset of lubrication with sexual stimulation, and an increase in vaginal pH leading to heightened vulnerability to urogenital pathogens and flora” (Bachmann, Ebert and Burd, 1999, p.199). In addition, androgens seem to have an effect on sexual desire, sexual arousal, and the overall sense of well being (Newman, 1999). Unfortunately, androgen levels appear to decline as a result of the aging process (Shifren, 2004). Also, oral contraceptives and estrogen replacement therapy diminish testosterone levels because exogenous estrogen therapy increases the levels of many hepatic proteins, including sex hormone-binding globulin (SHBG), the primary binder for testosterone (Shifren, 2004).

Physical Problems
Dyspareunia, or pain during sexual intercourse, can develop due to medical problems. By describing dyspareunia as superficial or deep, it helps to narrow down the possible cause. Pain that appears to be more superficial can be caused by a number of things including vulvitis, vulvovaginitis, vulvovestibulitis, genital herpes, urethritis, atrophic vulvitis, irritants (spermicides and latex), gynecological and obstetric interventions (episiotomies), local radiotherapy, and sexual traumas (Berman and Bassuk,
On the other hand, pain that appears to be more deep in nature can be caused by pelvic inflammatory disease, fibromyalgia, gynecological, pelvic or abdominal surgery, postoperative adhesions, endometriosis, genital or pelvic tumors, urinary tract infections, and ovarian cysts (Berman and Bassuk, 2002).

In addition, chronic illnesses can have an effect on sexual function. “Issues pertaining to the type of disability or chronic illness include the overall consequences of the disability or specific illness, whether there is an effect on genital function, secondary complications (such as fatigue), whether the disorder is static or progressive, iatrogenic concerns, and concomitant medical problems associated with the disability or illness” (Sipski and Alexander, 1997, p.3). Diseases and disabilities that are specifically liable in deterring sexual function include spinal cord injury, cerebrovascular accidents, multiple sclerosis, traumatic brain injury, cardiac and pulmonary diseases, cancer, diabetes, and arthritis (Sipski and Alexander, 1997).

**Neurogenic Disorders**

Neurological disorders that cause female sexual dysfunctions are the same disorders that cause erectile dysfunction (Berman, Berman and Goldstein, 1999). “These include spinal cord injury, diseases of the central or peripheral nervous system including diabetes, and complete upper motor neuron injuries affecting sacral spinal ligaments” (Berman and Bassuk, 2002, p.112). Other diseases that can have an impact on sexual functioning are multiple sclerosis, polymyositis, amyotrophic lateral sclerosis, myasthenia gravis and many others (Sipski and Alexander, 1997). The detrimental effect these diseases may have on sexual functioning is usually due to non-genital causes (Bach and Bardach, 1997).
**Vasogenic Disorders**

Hypertension, hypercholesteremia, smoking, and diabetes mellitus all lead to arteriosclerosis of the vasculature, including the vasculature of the genitals. Goldstein and Berman (1998) wrote that diminished blood flow secondary to atherosclerosis of the iliohypogastric/pudendal arterial bed is called the clitoral and vaginal vascular insufficiency syndromes. They further state that due to diminished pelvic blood flow, vaginal and clitoral smooth muscle can become fibrotic. This can result in symptoms such as vaginal dryness and dyspareunia (Goldstein and Berman, 1998). Atherosclerotic changes are also being linked to interrupting normal relaxation and dilation responses of the clitoral smooth muscle when engaged in sexual activity (Berman and Bassuk, 2002).

**Previous Pelvic Surgeries**

Pelvic surgeries can have a variety of effects on a women’s sexual functioning. Not only are there postoperative effects to be concerned about, but there can also be long-lasting effects. Hysterectomies cause concern because due to the direct anatomical disruption of genital organs and innervation, there is potential for a sexual dysfunction (Naughton, 2003). While removing the uterus, the arterial supply must be ligated which can result in ovarian atrophy and vaginal wall and clitoral smooth muscle fibrosis (Berman and Bassuk, 2002). If the hysterectomy is coupled with an oophorectomy, then this raises concern over inducing surgical menopause and therefore becoming estrogen deficient (Carlson, 2003).

There are also more surgical procedures which cause concern when focusing on female sexual dysfunction. For instance, repairing an episiotomy can make the vaginal
opening too tight, (Carlson, 2003) which can cause pain during sexual activity. In
addition, vulvectomy can cause dyspareunia, hyperesthesia, pruritus, and persistent
significant numbness making penile penetration indistinguishable (Andersen and Hacker,
1983). Furthermore, vulvectomy, mastectomy and colostomy are disfiguring procedures
which can diminish the patient’s self-esteem (Carlson, 2003). Pelvic procedures can
threaten sexual dysfunction.

Pharmacological Causes

Pharmaceuticals are a part of everyday life for a large percentage of the population. As with any drug, there is the possibility of having a side effect to the medication. Sexual dysfunctions are a common side effect to many drugs. Sexual side effects can alter a patient’s quality of life and could even affect compliance with the medication (Weiner and Rosen, 1997).

There is a gender bias concerning sexual side effects of medications on women (Weiner and Rosen, 1997). However, it is generally thought that anti-hypertensives such as diuretics, beta-blockers, calcium channel blockers, and anti-adrenergics cause sexual side effects that include diminished libido and anorgasmic (Carlson, 2003). Anticholinergics appear to decrease lubrication (Carlson, 2003). Benzodiazepines and antidepressants can also make it difficult to achieve orgasm (Carlson, 2003). In addition, chemotherapy (specifically cyclophosphamide and anti-estrogens) can cause an array of sexual problems including vaginal dryness, diminished libido and anorgasmic (Carlson, 2003). Opiates also tend to lower libido (Carlson, 2003). These prescription medicines listed above are widely used, which increases the possibility of developing a sexual side effect.
**Psychological Issues**

Whether or not a pathophysiological condition is present, women will be significantly affected by their emotional and relational issues (Berman, Berman and Goldstein, 1999). Problems with self-esteem, body image and the relationship with the partner can all affect sexual functioning (Berman, Berman and Goldstein, 1999). Stress, as indicated by fatigue and sleep disturbance, was the most common psychological factor leading to a sexual dysfunction (Newman, 1999). Individuals who struggle with gender identity, sexual knowledge and attitudes may also experience problems (Newman, 1999). Also, psychological disorders such as depression, dysthymia, obsessive compulsive disorder and anxiety disorder are associated with female sexual dysfunction (Berman, Berman and Goldstein, 1999). Other factors such as physical, sexual or verbal abuse place individuals at a greater risk for a sexual dysfunction (Naughton, 2003). For example, victims of childhood sexual abuse involving penetration had a 95% rate of having a sexual dysfunction as an adult (Sarwer and Durlack, 1996). Altogether there are many psychological aspects that can alter sexual function, but all must be considered in order to effectively treat the patient.
Epidemiology and Risk Factors

Regardless of patient demand for increase in clinical services for sexual problems and the potential effect this could place on their interpersonal relationships, epidemiological studies on sexual dysfunctions are relatively scarce (Laumann, Paik, and Rosen, 1999). In 1999, an analysis was published on a study done by the National Health and Social Life Survey (NHSLS) which assessed the prevalence and risk of developing a sexual dysfunction (Laumann et al., 1999). This study used a national probability sample of 1749 women and latent class analysis was used to evaluate the sexual symptoms.

This study concluded that approximately 43% of women experience a sexual dysfunction (Laumann et al., 1999). It was estimated that 22% of women experience low sexual desire, 14% experience problems with sexual arousal including lubrication difficulties, and seven percent experience sexual pain (Laumann et al., 1999). This landmark study showed how prevalent sexual dysfunction is in the United States.

The NHSLS study was also able to identify various social groups who are at risk for developing a sexual dysfunction. Married women were least likely to develop a sexual problem, where as non-married women were one and a half times more likely to have sexual climax issues and sexual anxiety. Women who graduated from college were approximately half as likely to have any sexual dysfunctions as compared to women who had not graduated from high school. The study found that black women were more likely to have problems with sexual desire, white women were more likely to have sexual pain and Hispanic women had consistently lower complaints of any sexual dysfunction. Other risk factors included women who experience emotional or stress-related problems,
financial setbacks, a current health condition, women with little sexual experience, and women who have been victims of childhood sexual abuse and/or rape.

On the contrary, women with a sexual history of more than five lifetime partners did not increase the risk of a sexual dysfunction (Laumann et al., 1999). Masturbation and having same-sex activity also did not increase the risk of sexual problems (Laumann et al., 1999). Having a sexually transmitted disease did not affect sexual functioning for women, and neither did moderate to high alcohol consumption.
Manifestations

According to the *Diagnostic and Statistical Manual of Mental Disorders* (2000), the definition for female sexual dysfunction states there must be a disturbance in the sexual response cycle that causes marked distress and interpersonal difficulty. The manual further divides the classifications into groups consisting of sexual desire disorders, sexual arousal disorders, orgasmic disorders, sexual pain disorders, sexual dysfunction due to a general medical condition, substance-induced sexual dysfunction and sexual dysfunction not otherwise specified (Diagnostic and Statistical Manual, 2000).

There are three diagnostic criteria for hypoactive sexual desire disorder. The first criterion is that the patient has a recurrence of persistent lack of sexual interest or fantasies for sexual activity (DSM, 2000). The second is that this lack of interest causes marked distress for the patient, and the last criterion is that there is no other Axis I disorder that can better account for the lack of desire (DSM, 2000). Another condition known as sexual aversion disorder is classified under hypoactive sexual desire. Sexual aversion disorder is defined as the deliberate avoidance of any genital sexual contact with another individual, which causes marked interpersonal distress (DSM, 2000). Typical behavior traits of individuals with a hypoactive sexual desire disorder include going to sleep early, traveling, neglecting personal appearance, using substances and being overly involved in work or social activities (DSM, 2000). Interestingly, a decreased female libido is very rarely the only manifestation of other diseases (Newman, 1999). The largest single reason is lack of satisfaction with their mate’s behavior. Depression is another major reason for decreased libido (Newman, 1997).
Female sexual arousal disorder is the next classification group with three diagnostic criteria. Female sexual arousal disorder is described as recurrent or persistent inability to initiate or maintain an adequate lubrication or swelling in response to sexual activity (DSM, 2000). In order for this diagnosis to be established, the patient must also be experiencing marked distress and there can be no other medical condition accountable for the sexual disorder (DSM, 2000). The most common compliant for this disorder is lack of lubrication (Newman, 1999). The arousal phase is more likely to be affected by chronic health conditions such as diabetes mellitus or pelvic cancer (DSM, 2000).

The next category is female orgasmic disorder, which consists of a recurrent delay or absence of sexual orgasm after sufficient sexual contact (DSM, 2000). This disorder also causes the patient marked distress and can not be caused by another medical condition (DSM, 2000). For patients who describe a lifelong problem with anorgasmy, sexual inexperience should be considered because lifelong anorgasmy rarely is the result of an underlying pathology (Newman, 1999).

Next are the sexual pain disorders. Sexual pain disorders can be split into dyspareunia and vaginismus. Dyspareunia is explained as genital pain before, during, or after sexual intercourse (DSM, 2000). This disorder also must cause marked distress in the patient: it also can not be caused by another medical condition or vaginismus or insufficient lubrication (DSM, 2000). Vaginismus is described as involuntary contraction of the perineal muscles upon insertion of anything into the vaginal opening (DSM, 2000). In some patients, even anticipation of anything being inserted into the vagina can cause muscle spasm (DSM, 2000). This condition is more commonly found in younger females, in females with negative views towards sex, and in sexually abused females.
(DSM, 2000). As with all of the other disorders, vaginismus must also cause marked personal distress and can not be better accounted for by another medical condition (DSM, 2000).

Finally, the last two classifications are sexual dysfunction due to a general medical condition and substance-induced sexual dysfunction. Sexual dysfunction due to a general medical condition is defined as any sexual dysfunction (hypoactive desire, sexual arousal disorder, orgasmic disorder, or sexual pain) that is due to some pathophysiological process (DSM, 2000). Alternatively, substance-induced sexual dysfunction is described as any sexual dysfunction as the result of any exogenous substance (DSM, 2000). It is a key point that substance-induced sexual dysfunctions arise only at the time of intoxication (DSM, 2000). As with the other sexual dysfunctions, there must be marked distress noted in the patient and there can be no other reason for the dysfunction (DSM, 2000).

The Diagnostic and Statistical Manual of Mental Disorders classification system is used as an aid in the diagnosis of female sexual dysfunction. However, if none of the diagnoses above can explain a present condition, there is the diagnosis of sexual dysfunction not otherwise specified. Otherwise, a sound diagnosis should be made after meeting the criteria.
**History and Physical Exam**

Anytime a patient presents to the office, a thorough medical history should always be done. A clinician needs to keep in mind that women are usually not forthcoming with their sexual problems. A complete sexual history needs to be actively sought (Lightner, 2002). A sexual history might begin with the questions, “Are you sexually active, and if you are, are you satisfied with your sex life?” (Polonsky, 2001) Other questions include, how long has this been bothering you and does this problem only happen with your current partner, or has it happened before? (Polonsky, 2001) Another question that may be helpful is asking the patient if she has any idea of why this problem is happening (Polonsky, 2001). Questions should be focused on whether the dysfunction is based on a desire, arousal, orgasm or pain disorder, and questions should also be aimed in narrowing down a possible cause for the dysfunction. This includes a past medical history looking for medical and psychological issues. There are also validated questionnaires, such as the Female Sexual Function Index, which help to discriminate between depression and a primary sexual dysfunction (Naughton, 2003).

The physical exam should look for general medical conditions, and the exam should always include a pelvic exam. In the case of a sexual pain problem, the pelvic exam is utilized to determine the origin of the pain. This is best determined by doing the manual exam first (Naughton, 2003). If vaginitis, sexually transmitted diseases, or cervical cancer is the main concern, then the speculum exam should be done first to obtain cultures and a Papanicolaou smear (Naughton, 2003). The pelvic exam should also be used to assess vaginal dryness, vaginal atrophy, neuromas, redundant labia, urethral prolapse and other untypical physical findings (Goldstein, Traish, Kim, and
The pH of the vagina can also be taken and used as an indirect indicator of vaginal lubrication (Berman, Berman and Goldstein, 1999).

Laboratory studies can also be performed. An endocrine evaluation is appropriate, and includes a serum follicle-stimulating hormone level, lutenizing hormone level, serum estradiol, dehydroepiandrosterone, total testosterone, free testosterone and prolactin levels (Lightner, 2002). This will help determine if the FSD could be secondary to menopause, testosterone insufficiency or a hypothalamic-pituitary axis lesion. Also, a complete blood count and a chemistry panel are helpful in excluding medical conditions that disrupt the hypothalamic-pituitary axis such as human immunodeficiency virus infection, end-stage renal disease, chronic obstructive lung disease and unexplained menopause (Shalender, Berman, Berman and Hellstrom, 2003).

Typically a diagnosis of FSD is made through a complete medical history. However, there are more objective ways to physiologically measure FSD. Female genital blood flow is measured using vaginal photoplethysmography, which is a tampon-shaped device that emits infrared light (Naughton, 2003). The device has a sensor that detects light reflected back from the mucosa, and the numerical value is representative of the vaginal blood flow (Naughton, 2003). Duplex Doppler ultrasound has also been used to measure female genital blood flow (Berman, Berman and Goldstein, 1999). Genital vibratory perception thresholds can also be measured using a standard biothesiometer (Berman, Berman and Goldstein, 1999). Typically, these measurements are done before and after sexual stimulation in a controlled setting (Berman, Berman, and Goldstein, 1999).
Treatment

Pharmacological Treatment

Since the release of Viagra in 1998, pharmaceutical companies have been vigorously searching for the same type of remedy that would ameliorate female sexual dysfunction. Unfortunately, there are still no pharmaceuticals approved by the Food and Drug Administration, but there are several being pursued (Fourcroy 2003). These pharmaceutical include apomorphine, bupropion, melanocortins, phentolamine, alprostadil, androgens, sildenafil and other phosphodiesterase inhibitors (Fourcroy, 2003). In addition, there are many herbal remedies that claim to alleviate FSD. In this section, each of these will be further examined.

Apomorphine, a dopamine agonist, has been sought after as a potential treatment because dopamine appears to be an activating neurotransmitter for sexual activity (Fourcroy, 2003). Apomorphine does appear to increase sexual activity in animals and it has already been approved for erectile dysfunction in Europe (Fourcroy, 2003). A placebo-controlled study was done to see if premenopausal women with either hypoactive sexual desire disorder or sexual arousal disorder were affected by oral apomorphine that was administered daily (Caruso, Agnello, Intelisano, Farina, Di Mari, and Cianci, 2003). The results suggested that 69% reported improvement with sexual desire and arousal (p< 0.05), and few women had problems with side effects. Unfortunately, due to the small sample size (n=44), additional studies are needed. Another study, which has already been through phase II of clinical trials, is assessing if intranasal apomorphine can benefit women with sexual arousal and/or sexual desire disorder (Kendirci and Hellstrom, 2004). There were 75 females who enrolled in the
study and the p values ranged from 0.0007 to 0.01. This study found that after nasal application of apomorphine, 55% of women noticed improvement with sexual satisfaction and 23% reported increased genital sensation. Due to the small number of subjects, these figures could not be deemed statistically significant. Both of these studies have shown promising potential, and therefore apomorphine needs to be further studied.

Bupropion, another dopamine agonist, is also being studied as a possible remedy for female sexual dysfunction. A study done in 2001 evaluated bupropion sustained release to see if it had an effect on women with idiopathic hypoactive sexual desire disorder (Segraves, Croft, Kavoussi, Ascher, Batey, Foster, Bolden-Watson, and Metz, 2001). This study established that 29% of women were treatment responders in two weeks, and 38% were treatment responders by the end of the 12 week study (p< 0.0001). Again, this study had a small sample size of 51 subjects, and therefore it would need to be done on a larger scale. Another study done in 2004 by Segraves, Clayton, Croft, Wolf and Warnock looked at the effects of bupropion SR on premenopausal women who had idiopathic hypoactive sexual desire disorder. This study found that bupropion had a statistically significant effect on increasing sexual responsivity (n=41, p=0.0002), but that it appeared to have a stronger effect on measures of orgasm than desire. Bupropion SR is still currently being studied as a treatment for FSD.

Another potential pharmaceutical for FSD is a melanocyte-stimulating hormone called PT-141 (Fourcroy, 2003). PT-141 also acts on the central nervous system and has been found to increase vaginal blood volume during erotic stimulation more consistently than Viagra (Morris, 2003). Melanocyte-stimulating hormone PT-141 is currently undergoing phase I clinical trials (Morris, 2003).
In addition to the centrally acting agents, phentolamine (an alpha adrenergic antagonist) has also been studied as a possible way to manage FSD (Fourcroy, 2003). A pilot study was conducted in 1999 observing the effects of oral phentolamine on women with female sexual arousal disorder (Rosen, Phillips, Gendrano, and Ferguson, 1999). This study found that phentolamine was well tolerated and was associated with mild improvements in sexual functioning. The study also had statistically significant measures (p <0.05) of improvement in sexual arousal consisting of lubrication and tingling sensations. Due to this study’s small amount of subjects (n=6), more research would have to be done on phentolamine, although none is currently being done (Fourcroy, 2003).

Alprostadil is another pharmaceutical that is being looked at for alleviating FSD. Alprostadil is a prostaglandin that is indirectly involved in smooth muscle relaxation and vasodilatation (Padma-Nathan, Brown, Fendl, Salem, Yeager and Harning, 2003). A recent randomized, double-blinded, placebo-controlled study was done on topical alprostadil to see how effective it was at alleviating female sexual arousal disorder in premenopausal women (Padma-Nathan et al., 2003). The study showed that women did have improvement in sexual arousal, but this finding was not statistically significant when compared to the high placebo response (n=94, p=0.05). Phase II clinical trials are currently being conducted (Fourcroy, 2003).

Sildenafil, or Viagra, has received much attention for treating erectile dysfunction, which would only bring about the question of can it help women with FSD? A randomized, double-blinded, placebo-controlled study assessed the effects of sildenafil on pre- and postmenopausal women. This study could not demonstrate any improvement
in subjective sexual arousal (Basson, McInnes and Smoith, 2002). Another double-blind, placebo controlled study was done to see if sildenafil had an effect on post-menopausal women with sexual arousal disorder (Berman, Berman, Toler, Gill and Haughie, 2003). This study showed that women with sexual arousal disorder did have a significant improvement in sexual arousal (n=202, p<0.02), but women with sexual arousal disorder and concomitant hypoactive sexual desire disorder did not benefit from treatment. Sildenafil has also been studied thoroughly to see if it had any effect on female sexual dysfunction induced by selective serotonin reuptake inhibitors. A literature review done on all articles published from 1989 to 1996 found sildenafil to be beneficial in reversing induced sexual dysfunction (Shen, Urosevich, and Clayton, 1999). In addition, sildenafil has been examined for treatment of FSD due to multiple sclerosis. A study using 30 subjects concluded that sildenafil did produce limited benefit with lubrication (p<0.01) however there was no improvement in orgasm (p>0.441) and it did not help with desire (p>0.196) (DasGupta, Wiseman, Kanabar and Fowler, 2004).

Androgen and estrogen replacement therapy has also been used to treat FSD. Androgen replacement in women with FSD and testosterone insufficiency can improve sexual desire, arousal and orgasm in 50% to 70% of women (Goldstein, Traish, Kim and Munarizz, 2004). However, the diagnosis of androgen insufficiency is controversial because androgen levels vary throughout the month and there are no set guidelines of what constitutes below normal range (Goldstein, Traish, Kim and Munarizz, 2004). Androgen insufficiency is currently defined as diminished libido, arousal and orgasmic capabilities with an adequate level of estrogen and below the physiologic range of androgen (Goldstein, Traish, Kim and Munarizz, 2004). Although androgen
supplementation is not approved by the Food and Drug Administration for women, it is currently being used in clinical practice (Shifren, 2004). Estrogen therapy is also currently being used clinically for dypareunia secondary to vaginal dryness (Goldstein, Traish, Kim and Munarizz, 2004). Unfortunately, estrogen therapy has not consistently increased sexual desire or activity, and many women with FSD who are taking estrogen therapy are unresponsive (Goldstein, Traish, Kim and Munarizz, 2004). Both estrogen and androgen replacement come with significant safety concerns and side effects, and therefore should be used only for a selective group of patients.

There has been research done on dyspareunia, specifically vulvar vestibulitis. A study done by Zolnoun, Hartmann and Steege (2003) assessed the effectiveness of nightly use of five-percent lidocaine on women with vulvar vestibulitis for 17 months. The study found that long-term nightly use of topical, five-percent lidocaine cream helped improve the symptoms (dyspareunia and vestibular touch) of the patients (n=61, p=0.002). A randomized, double-blinded, controlled clinical trial is still needed.

Finally, herbal remedies have also been popular in treating FSD. A literature review done in 2003 by Rowland and Tai evaluated common herbal agents used to treat sexual dysfunctions. They found that yohimbine, an alpha adrenergic antagonist, has had several double-blinded, placebo-controlled studies done which show 20% to 40% improvement in male sexual functioning, but little research has been done with female sexual dysfunction. They also stated that ginseng and ginkgo biloba hold promise for FSD because these herbs have shown a prosexual potential through preliminary studies. Other herbs, such as damiana, muira puama, saw palmetto and maca, have claimed to improve sexual functioning, but lack scientific background.
Non-pharmacological Treatment

“Most successful treatments for sexual dysfunction are psychophysiological, in that physiological change circularly interacts with a psychological change,” (Heiman, 2002, p.445). Psychological treatment has been a mainstay in the treatment of FSD, and it will likely continue to be. In general, sex therapy is not to improve genital performance, but to improve sexual comfort and pleasure (Leiblum and Wiegel, 2002). Psychological treatments are especially efficacious in treating orgasmic disorders and vaginismus. Data are too limited to conclude if it helps with hypoactive sexual desire disorder or inhibited sexual arousal (Heiman, 2002).

There exists one more non-pharmacological treatment option, mechanical devices. Two devices exist, one that works by vibratory stimulation and the other by causing vascular engorgement through using a vacuum system (Billups, 2002). The clitoral vacuum device, called Eros Therapy, is the only treatment that has been approved by the Food and Drug Administration for FSD and can be obtained through a prescription (Billups, 2002). It is thought that the Eros Therapy has an advantage over the common vibrator because vibratory stimulation induces clitoral engorgement as long as the dorsal nerves are still well vascularized. However, Eros Therapy will engorge the clitoris even if vascular disease is present (Billups, 2002). A study (n=20, p<0.001) was done during a three month period which showed women with FSD improve in areas of sensation (90%), vaginal lubrication (80%), orgasm (55%), and sexual satisfaction (80%) after using the Eros Therapy (Billups, Berman, Berman, Metz, Glennon, and Goldstein, 2001). Other findings suggest that Eros Therapy might have long term benefits by conditioning clitoral and vaginal smooth muscle (Billups, 2002).
Overall, there are many areas being explored in order to alleviate female sexual dysfunction. Pharmacologically speaking, there is hope especially with the centrally acting agents such as apomorphine, bupropion and melanocortin. Also, long-term, topical lidocaine therapy seems to help dyspareunia associated with vulvar vestibulitis. In addition, the herbal remedies offer popular choices such as yohimbine, ginseng and gingko biloba. Unfortunately, all of these treatments need further research to monitor safety and efficacy.

Traditionally, psychotherapy has also been used and seems to be especially effective in orgasmic disorders. The clitoral vacuum device is the first treatment option available that is recommended by the Food and Drug Administration. With FSD, becoming a growing field of interest, there is hope that treatment options will expand.
Conclusion

Sexuality is an important part of every individual’s life (Berman, Shuker, and Goldstein, 1999). Unfortunately, many people are suffering from some type of sexual dysfunction, and a majority is women. Even more unfortunate is that clinicians only have a small amount of training on how to elicit a sexual history (Polonsky, 2001), little education on human sexuality (Leiblum, 2001), and very few resources to seek continuing medical education on sexual topics (Solursh, Ernst, Lewis, Michael-Prisant, Mills and Solursh, 2003). By patient demand, there should be more mandatory hours allocated to human sexuality and dysfunctions in medical school, and there should be more resources for continuing medical education on sexual topics in order to bring practicing clinicians up to speed, especially on the topic of female sexual dysfunction.

The intention of this paper was to update primary care clinicians on female sexual dysfunction. This review showed that there are not only psychological causes for FSD, but physiological causes too. This paper also showed the high prevalence of FSD among women, and the associated risk factors. Manifestations of the dysfunctions were discussed, with specific insight on how to distinguish a psychological dysfunction from a physiological dysfunction. Previous studies were also scrutinized in search of a future treatment for FSD, including pharmacological and non-pharmaceutical approaches. From these studies, the only treatment approved by the Food and Drug Administration is the Eros Therapy vacuum device. There is also promise for the future on pharmaceuticals acting specifically on the central nervous system. Psychotherapy is another treatment alternative, although it appears to be more beneficial with hypoactive sexual desire disorder and vaginismus.
In conclusion, female sexual dysfunction can have a strong, negative impact on an individual’s quality of life. There is a need for continued research on the efficacy and safety of possible treatments including pharmacological and non-pharmacological approaches. In addition, future research should be done to reassess how well clinicians are being trained to handle sexual problems. Fortunately, a new interest has developed in the area of sexual dysfunctions, with increased attention on female sexual issues. Hopefully, this interest will continue among researchers and an efficacious treatment will be developed.


Is female sexual dysfunction receiving proper clinical attention? A clinical review on female sexual dysfunction
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Objective. The purpose of this review was to determine if female sexual dysfunction is receiving proper clinical attention. This review focuses on updated information regarding the etiology, risk factors, manifestations, and treatment for female sexual dysfunction. Method. Several databases were searched including MEDLINE, EBSCO, CINAHL, OhioLINK and PsychINFO. Results. The results show this is a vast amount of current research being done on female sexual dysfunction specifically in the area of pharmaceutical treatment. Many pharmaceuticals and herbs that act on the central nervous system are promising, but clinical trials are still pending. Conclusion. This clinical review established that there is a large prevalence of women who suffer from female sexual dysfunction, and researchers are actively seeking treatment options. However, women’s sexual health concerns are often unanswered, and therefore clinicians need to spend more time with women addressing sexual health issues.