Female sexual dysfunction (FSD) is a common complaint in menopausal women. It occurs when the quality of the sexual relationship is associated with personal or couple distress. Current research shows that many postmenopausal women have an increased sexual responsiveness as a result of reduced fear of pregnancy, absence of the need for contraceptives and the end of menstrual distress.

Aetiology

It is important not to label the normal age related changes in sexual response as pathology. Sexual problems, however, may be the first sign of an underlying illness, a sign of a deteriorating marital relationship, or a symptom caused by hormone deficiency. FSD has a multifactorial aetiology and even if there is co-existing psychosocial or psychological morbidity, ensuring a normal hormonal milieu is vital for effective management.

Hormones

Estrogen

The sharp decline in estrogen is the biochemical hallmark of the menopause. The resulting vaginal atrophy often leads to dyspareunia with a subsequent decline in libido. Estrogens play an essential role in sexual function. These hormones are responsible for the alleviation of hot flushes leading to better sleep patterns, less fatigue and the prevention and treatment of atrophic vulvovaginitis. Appropriate estrogen replacement may be enough to reverse a negative sexual cascade, although estrogen therapy only does not appear to increase libido.

Androgens

All androgens decrease with age. In premenopausal woman, the ovaries and adrenals together account for 50% of the total testosterone production. The remaining 50% comes from the conversion in the skin, liver and adipose tissue of androgenic precursors derived from the adrenal gland and ovary.

Despite the continued production of testosterone by the ovaries, total testosterone levels decline due to decreased production of adrenal androgen precursors which are available for peripheral conversion. In surgical menopause, the loss of ovarian production results in dramatically reduced levels.

Only 1 to 2% of testosterone is free and physiologically active due to sex hormone binding globulin (SHBG) binding strongly to testosterone. The bound fraction includes 66% tightly bound to SHBG and 33% weakly bound to albumin.

This whole topic is complicated by the difficulty in measuring testosterone, especially in females. Weakly bound testosterone can easily dissociate from albumin at tissue level. Therefore, serum levels may not accurately define dynamics at a cellular level.

The dramatic fall in testosterone levels with a surgical menopause may add to atrophy of the female genitalia. Despite the continued production of testosterone by the ovaries, total testosterone levels decline due to decreased production of adrenal androgen precursors which are available for peripheral conversion. In surgical menopause, the loss of ovarian production results in dramatically reduced levels.

Vulvo-vaginal Atrophy (VVA)

Atrophic vaginitis is under-reported by women, under-recognised by health care providers and therefore under-treated. VVA is a progressive symptom in the climacteric as opposed to other menopausal symptoms which may be self limiting. It is important to also remember that one third of women with VVA suffer vaginal symptoms despite systemic hormone treatment.

The International Menopause Society (IMS) statement on postmenopausal vaginal atrophy issued on World Menopause Day October 18th 2010 recommends the following.

- Treatment should be started early before irrevocable atrophic changes have occurred
- Treatment needs to be continued to maintain the benefits (noting chronicity of condition)
- All local estrogen preparations are effective and patient preference will often dictate choice of treatment
- Delay in starting local treatment will reduce the degree of response
- An initial loading dose to stimulate receptors followed...
by a maintenance dose once or twice a week
• Additional progesterone is not required as the low amounts of estrogen absorbed when administered correctly does not stimulate the endometrium

Alternatives to local estrogen therapy include intravaginal DHEA (phase 3 clinical trials), ospemifene tablets (a new selective estrogen receptor modulator), TSEC (bazodixifene plus conjugated equine estrogens) and vaginal moisturizers.

Vaginal moisturizers hydrate the vaginal mucosa and have to be used several times a week. They improve the balance of intracellular fluids in the vaginal mucosa and some may restore the acidic vaginal pH.

Vaginal lubricants provide comfort during sexual activity. They should ideally be silicone or water-based. Lubricants prevent irritation and potentially avoid mucosal tears, thus preventing pain with coitus and leading to an improvement in all domains of sexual activity.

Breast cancer survivors, especially those on aromatase inhibitors, have severe VVA. Sexual activity if desired is an important part of the rehabilitation process. The above modalities used appropriately will optimise sexual function and lessen emotional distress and improve psychosexual adjustment.

**Hypoactive Sexual Desire Disorder (HSDD)**

The commonest sexual dysfunction in the climacteric is HSDD. The hypothesis to consider is what role the lowered testosterone in the climacteric contributes to the HSDD. The reduced levels of testosterone in postmenopausal women are associated with a loss of libido, decreased sexual activity, diminished feelings of wellbeing and fatigue. If the latter causes personal or interpersonal distress, testosterone therapy is an option. This applies especially to the scenario of a patient who has had a bilateral oophorectomy where there is a sudden precipitous drop in hormone levels.

Before we ascribe HSDD to be purely hormonal in aetiology, we have to exclude other causes. They include psychosocial issues, psychological disorders, mental conditions and pharmacological agents. This involves taking a detailed history and allowing the patient time to express herself to care givers with a non-judgmental attitude towards sexual issues.

Testosterone therapy for women is a complex and ongoing debate. The European Union approved the use of the Tesosterone Transdermal Patch (TTP) (intrinsa) in 2007. In clinical practice, testosterone therapy can be safely used after excluding other causes of HSDD and informing patients that it is still off-label therapy in South Africa. Owing to the unavailability of TTP in South Africa, testosterone implants could be used at a dose of 40 milligrams. The dose is repeated at 4 to 6 months if there has been a definite response. Response can only be assessed 2 weeks after insertion of implant. There is no point continuing androgen therapy in the absence of a change in HSDD. In patients who request implants at intervals of less than 4 months, it is advisable to measure the free testosterone index, and only repeat the dose if the level is in the lower quartile of the normal range. Unfortunately, the implants have recently also been removed from the market and are no longer available in South Africa.

Testosterone therapy is usually given to patients after ensuring that they are well estrogenised. However, the recent ADORE study confirmed the efficacy of TTP in treating HSDD in naturally menopausal women with or without concomitant estrogen therapy use. Thus, testosterone therapy may have a place even in patients in whom estrogen therapy is contraindicated.

Appropriate candidates for testosterone therapy include patients with premature ovarian insufficiency, surgical menopause, adrenal insufficiency and hypopituitarism. Androgens heighten response to psychosexual stimulation. They also cause external genitalia to become more sensitive leading to more consistent sexual gratification. Overall, it induces a greater sense of well being.

Alternatives to testosterone therapy include tibolone (livifem) and DHEA. Tibolone does have weak androgenic activity and does not increase SHBG (sex hormone binding globulin). Dehydroepiandrosterone (DHEA) is a neurosteroid with a wide range of functions. The role of DHEA in the improvement of sexual function is controversial and we await more studies.

The sexual function of the partner is vital and the appropriate questions have to be asked. With many therapies being available for male sexual dysfunction, early referral of the male sexual partner may be indicated. It is also important to emphasise intimacy even if penetrative sex is not possible. Couples need to be informed of accessories that can aid the sexual response to enable them to gain access if so desired.

Relationship duration also influences sexual satisfaction. Sexual activity and satisfaction decline with increased duration of the relationship.

**Conclusion**

Sexual health is a fundamental human right. Age must not be a barrier to sexuality. Skill is needed to identify the subset of patients who will benefit with hormonal enhancement. Practitioners entrusted with the care of patients in the climacteric have to develop their abilities in sexual medicine in order to provide comprehensive care.
References

1. Bouman WP. Keeping sex alive in later years Chapter 1 Sexual Health and the Menopause 2005:1-7
2. Ramage M. Female Sexual Dysfunction and Menopause Chapter 2 Sexual Health and the Menopause, 2005:11-17
4. Udoff LC. Androgen Production and Therapy in women; Up to date 31/08/2012
5. Nappi RE & Palacios S. Impact of vulvovaginal atrophy on Sexual Health; Climacteric 2014; 17:3-9
13. Davis SR. Should women receive androgen replacement therapy and if so how? Clini Endocrinol (2010)72,1449-1554
15. Traish AMetal. DHEA – A precursor steroid or an active hormone in human physiology. J.Sex Med 2011;8:2960-2982