Sexuality in chronic illness: no longer ignored

Even though many diseases and their treatments impair sexual function, medical publications often omit sexual issues. However, national probability samples and clinical studies from many countries confirm that most men and women regard sexual wellbeing as centrally important. The majority of 480 men made paraplegic by spinal-cord injury confirmed that regaining sexual function was their major priority.1 That sexual function is a legitimate aspect of medicine is shown in the draft working definition of the WHO declaration of sexual rights in 2002. “Sexual rights...include the right of all individuals...to (achieve) the highest attainable standard of sexual health...and to pursue a satisfying, safe and pleasurable sexual life.”

Being invited to edit and coauthor a series on sexual medicine is a welcome honour. I had a long-term dream of submitting six papers (the ultimate sextet) to the Lancet. Instead, today’s Lancet sees the first of three papers: sexual sequelae of common chronic diseases will be followed by reviews on sexual repercussions of endocrine disorders1 and neurological disorders.4 All three papers give examples of increased prevalence of sexual dysfunction with comorbid depression. Interdisciplinary fields such as psychoneuroendocrinology confirm that matters of the mind greatly modulate immunological, neurological, and endocrinological systems. Improved mental state from regained satisfactory sexual life might well ameliorate the conditions we seek to treat.

Sexual dysfunction may herald serious underlying disease. Increasing data indicate that generalised erectile dysfunction—in sleep, during self-stimulation, or during partner interaction—could signify generalised endothelial dysfunction and be regarded as a marker of asymptomatic coronary-artery disease.5

The series emphasises the need to address iatrogenic sexual dysfunctions, including those from non-nerve-

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sparing pelvic surgeries, chemotherapy, and other treatments. Understanding sexual response cycles in men and women allows us as physicians to recognise and assess the interruption of sexual response caused by therapeutic interventions or by the disease, and to provide the needed assistance. Assessment and therapy can be guided by current sex research. Psychophysiological measures of genital congestion, functional brain imaging, and corresponding data of subjective arousal from sexual stimulation have given some understanding to the complexities of sexual arousal (and its absence), and to important differences between the sexes. Examples of treatment to medically augment a damaged sexual response include phosphodiesterase inhibition to reverse male sexual dysfunctions of erection, desire, and ejaculation induced by selective serotonergic reuptake inhibitors; and local oestrogen treatment to lessen vulvar vaginal atrophy from severe oestrogen depletion associated with aromatase antagonism in survivors of breast cancer.

Some forms of sexual dysfunction are common. Pain on every occasion of intercourse is experienced by 14–40% of women and is thus more prevalent than erectile dysfunction in individuals younger than 50 years. However, the prevalence of chronic dyspareunia in sub-Saharan African countries and parts of the Middle East and south Asia due to genital mutilation is unknown: the clinical impression is that it affects the majority. Although the most common cause of dyspareunia in North America and Europe (ie, vulvar vestibulitis) is being investigated for its genetic, neurological, psychological, and endocrinological components, our ability to change the attitudes and beliefs underlying the practice of genital mutilation seems to be a daunting challenge. Moreover, these women are not free to choose non-penetrative sex.

Areas of uncertainty include the safety and efficacy of supplemental testosterone and oestrogen. Improvements in sexual desire, nocturnal erections, and ejaculation from testosterone supplementation are unquestionable in younger hypogonadal men. However, the physiological reduction in testosterone in ageing men is not associated with any precise sexual syndrome or with clear evidence of sexual benefit from supplementation. All women cease to make ovarian oestrogen by midlife, and yet dyspareunia from vulval atrophy is by no means universal. Genetic polymorphisms of steroidogenic enzymes might clarify subgroups of women at risk for various sexual dysfunctions on the basis of low activity of oestrogen and testosterone. These sex hormones continue to be produced intracellularly from precursor hormones, including androstenedione, dehydroepiandrosterone, and dehydroepiandrosterone sulphate. Long-term safety of oestrogen started at menopause in sexually symptomatic women remains unclear, but observational, cohort, and case-controlled studies that do not focus on asymptomatic women are encouraging. By contrast, no data exist for the long-term safety of concomitant treatment with transdermal testosterone, soon to be approved for women with past bilateral oophorectomy; however, the reduced androgen activity is permanent. Off-label use will lead to many women with natural menopause being supplemented with testosterone before adequate safety and efficacy data warrant such use. In view of the non-correlation between sexual function and serum androgen in women, who should receive supplementation is unclear. Although androgen deficiency for women cannot be defined by serum concentrations of androgens or their precursors, measurement of serum androgen metabolites might yet uncover a deficiency. However, matters could be more complex: genetic polymorphisms of the genes encoding the androgen receptor and various androgen coregulators might have to be investigated to truly define any androgen deficiency state. Moreover, these various markers of androgen activity should be investigated in women with and without diagnosed sexual dysfunction. To control for the many psychosocial factors known to greatly affect sexual function will be challenging.

In the foreseeable future, we could have new, sexually neutral antidepressants and selective modulators for oestrogen receptors that possess needed genital oestrogen action. More widespread use of nerve-sparing surgeries for pelvic cancer might be possible. Using plastination methods to preserve dissections, pelvic surgeons at the Leiden University Medical Centre are being taught autonomic nerve-sparing techniques by one of the series authors.

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Can lethal injection for execution really be “fixed”?!

After months of intensive investigation, US District Court Judge Jeremy Fogel recently concluded that California’s “implementation of lethal injection is broken”,1 creating “an undue and unnecessary risk that an inmate will suffer pain so extreme that it offends the Eighth Amendment”. Fogel identified “critical deficiencies”, including unreliable screening of members of the execution team, lack of meaningful training and supervision, unreliable record-keeping, improper preparation and administration of the anaesthetic thiopental, and inadequate and poorly designed facilities. He suggested that such procedural deficiencies could have been responsible for the botched California execution of Robert Lee Massie, who the state’s expert witness admits “may well have been awake when he was injected with potassium chloride”.

California is not alone in either reconsidering the death penalty or discovering problems with its implementation. The death penalty is currently on hold in 11 US states. Illinois and New Jersey are debating the death penalty itself. The others, Arkansas, California, Delaware, Florida, Maryland, Missouri, North Carolina, Ohio, and South Dakota, are evaluating the method of lethal injection.3 Missouri executions have been halted partly because the individual in charge admitted to possessing no written protocol and to halving the dose of anaesthesia.3,4 Recently, in Florida, technical ineptitude prolonged the execution of Angel Diaz.5 Diaz was awake and apparently speaking 24 min after the first injection, and was finally declared dead after 34 min and two sets of injections. 30 cm chemical burns in both antecubital fossae were found at autopsy, which prompted the medical examiner to conclude that the intravenous lines were misplaced and the drugs were delivered subcutaneously.4 Although state officials did not comment on whether he suffered, Diaz probably experienced extreme pain and progressive paralysis, ultimately succumbing to suffocation. Executions continue in other states, although similar deficiencies in training and implementation have been identified. For example, Alabama executioners indicated their intent to establish intravenous access in non-existent vessels, citing the “external carotid vein” and the “saphenous vein in the arm”.3

Such examples of bungled executions and ill-trained execution teams are evidence that unrecongnised suffering in lethal injection can occur due to inadequate anaesthesia, as previously reported.7 Rather than the

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