Now that 15 years has elapsed since Viagra came on the market, and the majority of men with ED have hope for good rigid erections, attention has somewhat shifted toward ejaculatory issues. More and more men see me about problems with the “finish” of the sexual act, including “too soon”; “too late or not at all”; or inadequate amount or change in color (often bloody) or consistency.

Ejaculation is a complex function that involves emotions (feeling of intense affection for the partner); neurophysiological reflexes based on adequate sensory input from the genitals to the spinal cord and higher brain centers; hormones; anatomy and alterations thereof; and learned behaviors.

Although ejaculation is one word, the act is comprised of separable physiological events, including (1) emission, during which the semen containing sperm wells up in the part of the urethra passing through the prostate and causes the man’s feeling of “the point of no return”; (2) propulsion, in which the bladder neck, just above the prostatic urethra, closes like the iris of a camera, and muscles in the perineum (area between scrotum and anus) contract rhythmically, forcing semen out through the penis; and (3) orgasm, which is the brain’s interpretation of utter satisfaction in the climax of the sexual act.

The above mentioned components of ejaculation are under identifiable nerve control (for example, sympathetic and parasympathetic pelvic nerves), which differs for each part—partly explaining how a man can
experience one or more aspects of “the finish” without one or two of the others.

Adding complexity is the fact that erection and ejaculation can occur independently. Some men after prostate cancer surgery cannot erect well--but can have an orgasm, at the same time not experiencing emission or propulsion of semen. A rigid erection is certainly helpful to effect ejaculation. It is felt that the penis is more pleasantly sensitive when the skin is tight over the shaft (i.e., penis hard); and recruitment of more and more sensitive nerves leads to the spinal reflex translating into emission and propulsion of semen. This explains why Viagra and its relatives can improve (and perhaps even delay) ejaculation, even though these drugs are indicated for poor erections. Men who lose their erection soon after penetration learn to compensate by rushing coitus and thereby ejaculating prematurely.

In certain disease states involving nerve function, there can be impairment of erection and/or the components of ejaculation. Occasionally, one of the 1st signs of diabetes mellitus in a man is diminution of these sexual functions and over time, many diabetics will eventually have problems erecting and ejaculating properly. In spinal cord injuries, depending on the level at which the cord is injured, there may be erections that are not sustained and which the man cannot feel (“reflex erections”), less likely ejaculation, and not infrequently ejaculation-- if it occurs--with few sperms.

Premature or early ejaculation, if present, may be lifelong or acquired. My own observation is that many men who, when younger, have had premature ejaculation, “overcompensate” with behavioral changes as
they age, and they control ejaculation “too well” leading to delayed ejaculation. Ejaculation can be looked at as a spectrum from too early to too late [or not at all]. Some investigators have stated the average time between penile penetration and ejaculation is seven minutes. Premature ejaculation has no strict definition, but is in the eyes of the beholders, that is, the man and his partner. If a man climaxes less than one minute after vaginal penetration that could be disappointing for him and/or his partner, but in some cases it’s just fine.

The sex therapists Masters and Johnson in the mid-part of the 20th century popularized behavioral methods to reduce premature ejaculation. This was mainly having the man take himself to the brink of emission (the “inevitability” discussed above) and withdrawing, often squeezing the penis at the end of the shaft to damper the reaction--only to restart the act soon thereafter. Doing this and other mechanical maneuvers, (e.g. withdrawal and gently “pulling down” testes), do tend to retrain and retard the ejaculatory reflex. A corollary is to find a way to desensitize the penis using 1 or 2 condoms; or applying a cream such as EMLA or a spray such as Promescent) to slow down nerve transmission from the penis to the spinal cord. Promescent, worked on by my esteemed colleague Dr. Ron Gilbert [taken from us unexpectedly and tragically earlier this year] may be advantageous as regards absorption BELOW the skin and therefore having less of a tendency to numb the sexual partner.

Many antidepressants (AD), especially selective serotonin reuptake inhibitors in the family of Prozac (SSRI’s), do variably delay ejaculation, and are now a mainstay of treatment of premature ejaculation. A non-SSRI antidepressant, Anapranil, works well and if taken in low doses, by some either “on demand” (1-4 hours before sex) or every other day,
has a low side effect profile. Of the SSRI drugs currently marketed, paroxetine (Paxil) has a strong inhibitory effect (for some too strong), whereas the related drug sertraline (Zoloft) seems to have “just the right effect” and is currently my preference for patients seeking a pharmacological solution. I myself have not seen behavioral side effects in men using these drugs to help ejaculation—but low dose and on demand use may be the reason why.

Dapoxetin, a newer SSRI drug around for more than 10 years and already used in many other countries, is going through its final stage of FDA clearance for US release. Its advantage is a quicker both onset of action and elimination from the body—perhaps making it more ideal than its predecessors for improving ejaculation only and minimally behavior/depression.

Prolactin, a hormone secreted by our pituitary, causes milk development in pregnancy— but has a broad role in male sexual dysfunction. Most doctors know that overproduction of prolactin by the pituitary can lead to sexual dysfunction and low sperm counts/infertility. So-called “hyperprolactinemia” can be caused by a benign pituitary tumor producing this substance. A current question is whether prolactin levels can be manipulated within the “normal range”, since it seems that prolactin by its nature tends to retard ejaculation and increase the latency time (time during which man cannot get another erection after orgasm). Studies of a drug called cabergoline (Dostinex), used to treat excesses of prolactin, shows some promise (studies are preliminary—and safety issues need to be addressed) in promoting earlier ejaculation in those who “have trouble pulling the trigger”--and perhaps even the ability for a man to have multiple orgasms.
Of course testosterone plays a role in ejaculation, with too little of it contributing to delay or no ejaculation. Some studies with Dostinex suggest the latter works best when used in conjunction with testosterone supplementation. Thyroxin, an important hormone produced in a familiar gland in the neck, has a definite in sexual function. New onset of delayed ejaculation might warrant a lab check of thyroid function, e.g., to exclude hypothyroidism.

Finally, my tangential departure to discussion of hematospermia, or blood in the ejaculate. The most common (and expected) cause, these days, are prostate biopsies. This bleeding will stop on its own. Most studies suggest hematospermia is benign, i.e., not related to any serious diseases including genitourinary tract cancer; and it often goes away without treatment (is self-limiting). I myself do not feel that hematospermia alone should be treated as a “prostate infection” with antibiotics--but some of my colleagues disagree. I might consider antibiotics if the man also has painful ejaculation, painful urination, urgency with poor flow--and more so, if there are corroborating lab data such as abnormal urinalysis and/or urine (or sometimes semen) culture. In man over 50 (? over 40) with persistent blood the semen, I might go a bit further beyond a GU exam: including prostate ultrasound done transrectally and sometimes a prostate MRI with contrast. When I have ordered these tests, and assuming a normal prostate exam and PSA, I have rarely, if ever, identified a cause that—if treated—will permanently eradicate the bloody semen. For example, on ultrasound, one can see things like calcifications in the prostate or ejaculatory ducts (joining the seminal vesicles and vasa to prostate)--or prostatic cysts or seminal vesicle (semen containers above prostate)
dilations. However, these types of abnormalities are seen just as often in men who have never had blood in the semen as opposed to those who have experienced this symptom. I myself believe some men just have very fragile plexuses of veins lining the prostatic ducts (which sometimes are a bit brittle, perhaps related to calcium deposits), through which semen is emitted before propulsion. “Tearing” of such small vessels is the prostate’s equivalent of a benign nosebleed.

From a pragmatic viewpoint, we as urologists are getting better at translating the obscure and complex physiological concepts regarding hormonal and neurobiology of the sexual act into tangible solutions to improve a man’s (and couples') pleasure during coitus.

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