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Ejaculation: the Process and Characteristics From Start to Finish

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Abstract

Purpose of Review—Semen analysis serves as the initial step in the evaluation of male infertility. However, given the difficulty in interpreting abnormal findings, physicians and patients often struggle with understanding the results. In this review, we aim to review the normal physiology of ejaculation and create an accessible resource for interpreting abnormal semen volume, viscosity, liquefaction, pH, appearance, and color.

Recent Findings—Emerging evidence has revealed that men with genitourinary tract infections have a greater number of seminal leukocytes, which may result in clumping of motile sperm and altered morphology. Hence, these patients may have abnormal sperm parameters secondary to their health status. Recent findings have further characterized the semen liquefaction process, suggesting that increased levels of semenogelin and decreased levels of proteases and plasminogen activators (e.g., urokinase and chymotrypsin) may be associated with the failure of semen to convert to a watery consistency.

Summary—This article creates a resource which may be referenced when abnormalities in semen analysis are encountered. We offer a comprehensive overview of normal ejaculation physiology and abnormal variants in male ejaculate volume—including aspermia, anejaculation, retrograde ejaculation, and hypo- and hyperspermia—and their potential etiologies. Additionally, we discuss several processes (infection, inflammation, and dysfunction of male sex glands) which may affect semen viscosity, liquefaction, and pH. Finally, our discussion of the potential colors of male ejaculate is meant to reduce the anxiety of both patient and provider. Through a better understanding of the process and varying characteristics of ejaculation, physicians may adequately counsel their patients on abnormal findings and concerns regarding infertility.

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Keywords

Ejaculation; Semen analysis; Male infertility; Spermatozoa

Introduction

Human male ejaculate, or semen, is an organic mixture composed of spermatozoa and fluid from the seminal vesicles, prostate, and bulbourethral glands. Semen analysis (SA) has long been the first investigative tool used when assessing the male factor in couples with infertility, which is a contributing factor in approximately 50% of cases [1, 2]. This test includes an assessment of macroscopic and microscopic parameters through a series of tests as described by the World Health Organization (WHO) [3••]. Beyond fertility implications, an abnormal SA has been associated with higher rates of testicular cancer and multiple non-malignant chronic diseases [4, 5]. Although SA is considered the gold standard of male fertility assessment, this test has been shown to be a poor indicator of reproductive outcomes. Results are also widely variable depending on sample collection methods, length of abstinence, and other factors [6, 7].

Many components of SA have been thoroughly explored, including a modern review of conventional methods and clinical interpretation of variables [8]. However, a paucity of literature exists regarding abnormal macroscopic features of SA, including semen volume, viscosity, liquefaction, pH, and appearance or color. A better understanding of these variables has important clinical implications for medical providers but would also empower patients with a knowledge of normal and abnormal semen variances.

In this article, we will briefly review the normal physiology of semen formation and ejaculation. This will be followed by a detailed literature review exploring the etiologies, significance, and management, when available, of variances in the macroscopic characteristics of SA. In doing so, we aim to create a resource that providers and patients may reference when abnormalities in these characteristics are encountered.

Physiology of Ejaculation

Male ejaculate is a complex, non-homogenous, multi-role mixture of immune and glandular cells, salts, peptide and steroid hormones, carbohydrates, organic acids, lipids, mucus, nucleic acids, vitamins, minerals, urogenital microbial flora, and over 2000 unique proteins [9–12]. Although some components of semen are fully soluble, others bind directly to spermatozoa or are packaged into cargo-bearing vesicles that can then fuse with spermatozoa. Once inside the female reproductive tract, these non-spermatic components may serve to further stimulate ovulation, alter reproductive-tract pH, modulate immune activity, nourish spermatozoa, or provide other functions that are yet to be determined [11–13]. Table 1 describes normal macroscopic semen parameters as defined by current WHO guidelines [3••].

The composition of post-ejaculatory semen is highly variable between men and within individual men over the course of their lifetimes. However, approximately 90% of the

semen is composed of fluid from the seminal vesicles, prostate, and bulbourethral gland, in descending order of percent contribution.[3••] As such, only a small fraction of the semen is composed of spermatozoa. Numerous factors that determine the fraction of spermatozoa in semen have been studied, including metabolic syndromes (e.g., diabetes mellitus), diet, drug use, varicoceles, outflow tract obstructing pathologies, and various genetic pathologies such as Klinefelter syndrome.[3••, 14–16]

The development of spermatozoa, a process known as spermatogenesis, begins at puberty with mature spermatozoa originating from germ cells found within the basement membrane of the seminiferous tubules of the testes. Sertoli cells, stimulated by follicle-stimulating hormone (FSH), and Leydig Cells, stimulated by luteinizing hormone (LH), both help to regulate spermatogenesis. Once puberty has begun, one cycle of spermatogenesis within the Sertoli cell epithelia begins every 13–16 days [14, 17, 18]; however, spermatogenesis is not consistently synchronous throughout all seminiferous tubules. Furthermore, the exact number of stages of spermatogenesis remains unclear and debated [14, 18]. After immotile spermatozoa are released into the tubules, they travel to the epididymis for further maturation and the development of functional motility. A fully mature sperm cell consists of a head, midpiece, and tail. The head contains the nucleus and is covered by the acrosome, which is filled with lysosomes that assist in fusion with an ovum. The mid-piece of the spermatozoa contains abundant mitochondria to produce ATP production that provides energy for the sperm cell's flagellum, or tail, which allows for propulsion [18]. The entire process of human spermatogenesis can vary in length, but previous studies estimate it at 42–76 days [14].

Mature spermatozoa are stored in the tail of the epididymis until the pelvic plexus receives sufficient sensory input from the hypogastric and pelvic nerves, in addition to the psychologically driven caudal paravertebral sympathetic chain, to initiate ejaculation [19]. The emission stage of ejaculation begins when the bladder neck is closed to prevent retrograde flow of the imminently passing ejaculate. This action coincides with the propulsion of sperm from the epididymis into the ductus (vas) deferens by smooth muscle contractions. From here, the sperm are carried through the spermatic cord towards the ejaculatory duct, where they mix with a clear fructose-rich solution from the seminal vesicle. As this new mixture passes through the prostate, an alkalinizing fluid is secreted which further thickens the fluid, now called semen, and protects the sperm from acidic conditions within the female reproductive tract. Finally, the semen passes through the bulbourethral glands which provide one final addition by releasing a fluid that both lubricates the urethral opening and clears it of any residual urine [11, 12, 14]. The final physiologic composition of the semen has now been produced and will be released in the next stage of ejaculation, termed expulsion. This refers to the ejection of semen through the urethral meatus, propelled by cyclic contractions of the striated pelvic muscles alongside those of the bulbospongiosus and ischiocavernosus muscles [20].

The stage of puberty during which spermatogenesis begins varies greatly between individuals. While some children begin producing motile sperm in Tanner stage 1, others have not been shown to produce viable sperm until as late as Tanner stage 4 or 5 based on longitudinal collection studies of spermatouria [21–23]. The median stage however sits

somewhere between Tanner stages 2 and 3 [21–23]. This variability has resulted in debate as to the developmental stage in which electroejaculation should be considered as a method of fertility preservation in children and adolescents undergoing cytotoxic and sterilizing treatments. Studies originating from several fertility centers have determined that while sperm can be found in semen as early as Tanner stage 1, they are too few and/or immotile to be viable for cryopreservation. Tanner stage 2 was the first stage in which viable sperm samples for cryopreservation were found; however, this was not a universal finding. In fact, it was only in Tanner stage 3 that electroejaculation was able to reliably produce acceptable sperm samples from adolescent boys [24, 25]. Interestingly, one paper found that levels of FSH, LH, inhibin B, and testosterone do not differ significantly between patients with acceptable sperm samples and those without [24].

Volume

Aspermia is defined as the complete lack of semen expulsed from the urethral meatus. This occurs when semen is not produced or the fluid cannot be propelled in an antegrade direction [26]. However, during aspermia, the ability to orgasm remains intact. Aspermia can be classified as anejaculation, when there is a complete lack of semen emission and expulsion, or retrograde ejaculation, when semen enters the bladder instead of emerging from the penis [26, 27].

Anejaculation is diagnosed by lack of antegrade ejaculation in addition to a non-viscous, fructose-negative, and sperm-negative post-orgasmic urinalysis [28]. Spinal cord injury (SCI) is the most common cause of neurogenic anejaculation, although many men with SCI will continue to have reflex erections and respond to oral erectogenic agents or penile injections of vasoactive substances [29]. Additional etiologies for neurologic anejaculation are related to congenital anomalies, such as spina bifida, and other neurologic problems, such as dysplasia of the lower spinal cord secondary to Parkinson’s disease, multiple sclerosis, or diabetes [26, 29]. Psychogenic issues, including stress and anxiety, as well as obstruction in the ejaculatory duct, pelvic surgery, and pelvic trauma may also be responsible for anejaculation [26, 29]. For example, retroperitoneal lymph node dissection in the treatment of testicular cancer involves the removal of postganglionic sympathetic nerves exiting the sympathetic chain and the hypogastric plexus, resulting in anejaculation [29]. However, nerve-sparing alternatives have been developed to preserve fertility in these patients [28, 29]. Alpha agonists, electroejaculation, and electrovibration stimulation have been used to treat patients with anejaculation who desire fertility [28].

Retrograde ejaculation can be further described as complete, with no antegrade fraction, or incomplete, with minimal antegrade fraction [28]. Patients with retrograde ejaculation will have a post-orgasmic urinalysis that is positive for fructose and sperm [28]. Similar to anejaculation, retrograde ejaculation can be due to SCI and other neurologic disorders, although it can also be a side effect of certain medications or surgical procedures that relax the bladder neck [26]. As bladder neck closure and seminal emission are under the control of alpha-adrenergic receptors, antagonists of these receptors, such as tamsulosin or alfuzosin, may result in retrograde ejaculation [29]. Additionally, many antipsychotic medications, such asthioridazine, risperidone, iloperidone, and clozapine, have been associated with

retrograde ejaculation due to their broad and diffuse receptor antagonizing ability [30]. Surgeries, including transurethral prostatectomy and prostatic ablative procedures, are other common causes of anatomic retrograde ejaculation [30]. Various medications have been used to treat patients' retrograde ejaculation and improve fertility, including midodrine, imipramine, and a combination of chlorpheniramine and phenylpropanolamine [28]. Electrovibration stimulation, sperm recovery from the urine, and surgical sperm retrieval can also be considered if pharmacologic therapy fails [28].

Hypospermia is defined as a semen volume less than 1.4 mL on at least two separate semen analyses [3••]. Hypospermia can be an isolated finding but is also associated with other sperm abnormalities, such as oligospermia (low sperm count), asthenozospermia (reduced sperm motility), or teratozospermia (abnormal morphology of sperm) [31]. Lack of sexual abstinence prior to specimen collection commonly results in hypospermia [31]. As previously discussed, incomplete retrograde ejaculation can cause partial antegrade ejaculation and thus hypospermia [31]. Hormonal irregularities may also be a contributing factor, including hypogonadism, which decreases circulating levels of testicular androgens and leads to quantitative and qualitative alterations of multiple components of male ejaculate, resulting in hypospermia [31]. Similarly, hyperprolactinemia caused by prolactinomas or drugs, such as spironolactone, cimetidine, antipsychotics, and ketoconazole, can decrease androgen levels and result in hypospermia as a result of inhibitory feedback from elevated prolactin levels on the hypothalamic–pituitary–gonadal axis [31]. The bilateral or unilateral absence of the vas deferens, as can be seen in patients with cystic fibrosis, as well as obstructive abnormalities, such as prostatic cysts or post-surgical stenosis of the surgical tract, can also be associated with hypospermia [31]. Treatment of hypospermia is variable and depends on the specific etiology (Table 2).

Hyperspermia has been previously defined as semen volume greater than 6.3 mL [32]. The pathological significance of hyperspermia is still under debate, although associations have been made between hyperspermia and genitourinary infections [31]. Previous literature suggests that the production of excessive seminal fluid during coitus may decrease the chances of successful fertilization by limiting the availability of spermatozoa to the female reproductive tract due to the dilution of the semen [32].

Viscosity

After ejaculation, semen coagulates and then gradually liquefies. However, in 12–29% of men, in a process likely related to infection, inflammation, dysfunction of male sex glands, or in diseases directly affecting male fertility, semen may be hyperviscous [33]. Semen hyperviscosity can be diagnosed by using a wide-bore pipette to dispense a drop of semen by gravity and measuring a thread length of more than 2 cm long [33]. The causes of semen hyperviscosity may contribute to infertility, and it has been postulated that hyperviscosity itself may be a contributing factor due to the impairment of normal sperm movement [33]. Esfandiari et al. [34] investigated the effect of increased seminal viscosity on patients undergoing in vitro fertilization and found that couples with seminal hyperviscosity experienced significantly lower fertilization rates, clinical pregnancy rates, and implantation rates. Hyperviscous semen can be treated by in vitro dilution of semen

and directly by overhydration, prostatic massage, and parenteral hyaluronidase with some success [33]. Additionally, mucolytic agents, anti-inflammatory agents, and antibiotics have been used for the management of hyperviscous sperm depending on the etiology [33].

Currently, there exists very limited literature related to hypoviscosity, or over-liquefaction, of semen. In a study using physical analysis of ejaculate to estimate the diagnostic potential and to predict the function of the seminal vesicles and prostate, 3.6% of semen samples analyzed had hypoviscous semen and were associated with biochemical markers that suggested impaired secretory activity of the seminal vesicles [35].

Large gelatinous clumps of semen have been observed in association with infertility [36]. In one study, manual manipulation of these clumps to separate the bound sperm failed, as the clumps would reform their shape, and only small numbers of free-moving sperm were observed [36]. Smaller gelatinous clumps are occasionally seen in fertile men, although in most cases these samples are normal and it is believed that the clumps are composed of large numbers of free-moving sperm [36]. During infection or inflammation, greater numbers of seminal leukocytes may result in clumping of motile sperm, decreasing acrosomal functionality and resulting in altered morphology [37••].

Liquefaction

Semen liquefaction refers to the ability of coagulated or gel-like semen to convert to a watery consistency due to the action of proteases originating from the seminal and prostatic fluid [38]. This process is of importance within the female reproductive tract, where liquefaction allows for the motility and transport of sperm to the fallopian tubes for egg fertilization [38]. Liquefaction is considered abnormal if semen does not begin to liquify after 30 min, referred to as delayed liquefaction, or if liquefaction is not complete 60 min after ejaculation, referred to as non-liquefaction [39]. Although the exact pathogenesis of non-liquefaction is unknown, semenogelin and liquefaction factor, found inside the seminal vesicles and the prostate respectively, appear to influence liquefaction [39]. Increased levels of semenogelin and decreased levels of proteases and plasminogen activators, such as urokinase and chymotrypsin, are associated with non-liquefaction, along with seminal vesiculitis, a lack of trace elements, and congenital prostatic deficiencies [39, 40].

pH

The normal pH values of liquified semen range from 7.2 to 7.8 according to current WHO guidelines, and are influenced by a complex buffer system including inorganic ions, organic acids, and proteins [3••, 41]. The pH is primarily determined by the acidic secretions of the prostate and the alkaline secretions of the seminal vesicles which provide the semen with a high buffering capacity [42, 43]. The exact reference range for semen pH has been disputed [42, 44]. In fact, a previous study comparing several hundred semen samples found that both patients with normal and abnormal sperm concentration and motility values had an average semen pH of 8.2 [44]. This is also complicated by the fact that, once ejaculated, semen pH increases with time as natural buffers are lost to the effect of environmental carbon dioxide [45••]. Nevertheless, semen pH offers a variety of clinically relevant information. Semen that

is acidic (pH < 7.2) can be indicative of a congenital bilateral absence of the vas deferens or blockage of the seminal vesicles [42, 45••]. Alkaline semen (pH > 8.0), on the other hand, has been associated with infections, inflammatory conditions, and increasing age [42, 45••]. Although some studies have suggested an association between pH and fertility parameters, such as sperm motility and count, these correlations are weak and require further validation [45••]. Interestingly, resuspending healthy sperm in acidic solutions has been shown to significantly reduce sperm motility and capacitation compared to non-acidic samples, which may indicate a role of the vaginal microenvironment on sperm activity [43].

Appearance and Color

Normal semen appearance typically appears cream/gray, opalescent, or off-white. This is due to the secretions of the prostate and seminal vesicles rather than the sperm component. [3••, 14] A mild gradual yellowing of the semen is normal with age and/or prolonged abstinence from ejaculation. This yellowing is secondary to the accumulation of lipofuscin granules from dead epithelial cells that are shed in the secretions of the seminal vesicles.[3••, 14, 46, 47]

The opacity of the semen is dependent on several factors that are inherent to the individual (e.g., age) or contextual (e.g., diet, abstinence from ejaculation, medication, or infection). Less opaque semen has been proposed to be due to a lower sperm concentration [3••]. However, the validity of using opacity and color of semen as proxies for concentration and reproductive viability of the ejaculate is not agreed upon [48]. Prior literature has shown that opacity and sperm concentration of the semen will progressively decrease if the ejaculation occurs shortly after a prior ejaculation and every additional recent ejaculation before that [3••]. It should also be noted that colorless mucus-like ejaculate is likely to be pre-ejaculatory fluid from the bulbourethral glands.

Yellow to Orange Coloration

As noted prior, some yellowing of the semen can occur naturally with any accumulation of lipofuscin in seminal fluid, whether due to the individual's age, time spent abstinent from ejaculation, or other benign etiologies, including retained urine in the urethra [14]. While not clearly supported in research studies, it is intuitive and anecdotally reported that foods, drugs, or environmental exposures that can seep into and tint the urogenital tract tissues can also cause benign yellowing of the semen. Orange-tinted semen, for instance, has been noted in those patients receiving rifampin [49]. As such, a slight yellowish tint to the semen is not a cause for concern in and of itself. However, there are occasionally serious pathologies that can also cause the semen to appear yellow [3••, 48]. These include infections such as orchitis or prostatitis which can lead to pyospermia or leukospermia, in which white blood cells accumulate in the semen. These conditions not only cause the semen to discolor but also impair its reproductive viability [3••, 50, 51]. This occurs from a number of potential mechanisms, ranging from cytokines impairing sertoli cell function to polymorphonuclear neutrophils' reactive oxygen species causing DNA fragmentation or damage to the sperm's motility, acrosome, or cell membrane [50, 52–56].

Jaundice, secondary to excessive bilirubin levels, can also result in a yellow appearance of the semen [3••]. While alcohol has been hypothesized to be associated with yellow semen, no primary literature has yet come to this conclusion. On the contrary, one study looking at the semen of one hundred men, some consuming on average seven alcoholic drink equivalents a day, found that the semen of men who regularly consumed alcohol was still white in color. However, changes in other semen parameters were observed [57]. This association may be attributable to alcohol indirectly by dehydration or urine contamination acutely or due to mineral and vitamin deficiencies, leukospermia, and/or hyperbilirubinemia in individuals with alcoholic liver disease [58].

Pink to Red Coloration

A pink to red color of semen is one of the most frequently cited atypical colors of semen. Foods, such as beets, and certain drugs are sometimes cited as causing color changes when consumed in large quantities. However, the most common cause of red discoloration in semen is hematospermia (hemospermia), or blood in the semen, which is often painless and self resolves. Hematospermia, in fact, makes up 1% of all urologic symptoms [59]. Fresh blood within the semen will show a diffuse or streaked light red to brownish color, while dark brown or black clots usually indicate that some time has passed since the initial bleeding occurred [3••, 60].

Fortunately, most causes of hematospermia are benign, particularly in younger men under thirty, and may even be idiopathic in approximately 20% of patients [60]. The most commonly known causes are infection, such as prostatitis, urethritis, or epididymo-orchitis, medical intervention such as a prostate biopsy, and genitourinary trauma. However, a workup is occasionally indicated to evaluate for the severity of infection, presence of malignancy, or other pathology that requires treatment [61]. This includes a patient history to rule out pseudohematospermia, such as blood leftover from a partner, or to elicit a probable cause of hematospermia, such as trauma or recent surgery/procedure. For example, a prostate biopsy has a post-procedure hematospermia rate of nearly 80% [62]. On clinical examination, high blood pressure or fever may point to a systemic or infectious etiology, respectively. With any concern of hematospermia, a complete physical exam of the urogenital area is warranted, alongside a digital rectal examination in older men to elucidate potential prostatic pathology. Finally, a urinalysis is a commonly agreed upon initial diagnostic tool to use in the determination of different potential etiologies, such as infection. However, as previously noted, hematospermia is not an uncommon symptom and is usually benign, self-resolving, or manageable in an outpatient setting. Extensive diagnostic workup of hematospermia is not commonly encouraged without strong clinical reasoning elicited by the patient's presentation [63, 64].

Brown to Black Coloration

Darkly colored semen, of which old blood is a common cause, potentially warrants further workup. This is especially true when found in combination with other atypical characteristics of semen or bodily symptoms. There are some causes of dark and black semen that coincide with medical conditions requiring attention. For instance, multiple case reports have indicated schistosomiasis as a cause of brown or black semen, although

ejaculates can also appear red, yellow, and even white [65–67]. One recent report notably analyzed the semen of a man who had abstained from ejaculation for 7 days and produced a "9.8 mL sample of non-viscous brown-colored semen with a bad odor." Microscopic semen evaluation showed slight sperm agglutination, a large number of Schistosoma haematobium ova, extensive debris, and many amorphous cells under a high power field (× 200) [66]. The patient had failed to father any children that he was aware of and his semen viability was deemed approximately 13% of normal.

Other noteworthy non-infectious and non-bleeding etiologies of dark semen include SCI, heavy metal toxicity, and uric acid crystal deposition. SCI remains poorly understood in regard to its capacity to darken semen. It is not, however, a ubiquitous symptom and while blood is occasionally found in the semen, it is not required for the semen to be dark [68]. A number of patients with SCI have also been described with dark semen containing platinum alongside elevated levels of lead, manganese, and nickel [69]. Black-reddish semen with a large volume of colored precipitates (1.0 mL of the total 2.4 mL after centrifugation) from a patient with symptoms of chronic prostatitis has also been reported [70]. Despite initial warranted suspicion, red blood cells were not found under direct microscopy. However, large quantities of red-brownish crystals with a morphology compatible to uric acid crystals were detected and found to be responsible for the abnormal color. Serum and urine uric acid levels were normal, indicating an unknown mechanism of uric acid accumulation in the prostatic and seminal fluid, although urinary reflux was proposed. A low purine diet was found to successfully treat the symptoms of the suspected prostatitis and reduced semen uric acid levels to reference range limits, resulting in a normal return of semen parameters (Table 3).

Conclusions

Semen analysis (SA) is the first and most fundamental step in the evaluation of male fertility potential. Variances in the macroscopic characteristics of SA, including semen volume, viscosity, liquefaction, pH, and appearance or color, have wide-ranging etiologies and clinical implications. A better understanding of these variances has the potential to guide counseling for patients with certain variances while also potentially allowing for intervention to improve fertility and additional medical outcomes. Continued robust methodological research on this topic is necessary to better determine the origins of macroscopic abnormalities in SA and guide recommendations regarding clinical significance and management.

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Papers of particular interest, published recently, have been highlighted as:

• Of importance

•• Of major importance

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Table 1

Reference values for macroscopic semen parameters

Semen parameter	Reference value
Volume (mL)	1.4–6.3
Viscosity	Forms small discrete drops (thread < 2 cm long)
Liquefaction	Within 60 min
pH	7.2–7.8
Appearance	Gray-opalescent

Table 2

Definitions and known etiologies of abnormal variants in male ejaculate volume

Variant	Definition	Etiologies
Aspermia	Lack of ejaculated semen	
Anejaculation	Lack of seminal emission and expulsion *Post-orgasmic urinalysis*: non-viscous, fructose-negative, and sperm-negative	*Neurologic*: SCI, secondary spinal cord dysplasia (e.g., diabetes, Parkinson's, multiple sclerosis), congenital disorders *Psychogenic*: anxiety, stress *Other*: pelvic obstruction, surgery, and trauma
Retrograde ejaculation	Lack of forward propulsion of semen *Post-orgasmic urinalysis*: positive fructose and sperm	*Medications*: alpha-adrenergic antagonists, antipsychotics *Surgeries*: transurethral prostatectomy and prostatic ablative *Neurologic*: SCI
Hypospermia	Ejaculated semen less than 1.4 mL on two separate measurements	*Hormonal*: hypogonadism, hyperprolactinemia, androgen-reducing medications *Obstructive*: prostatic cysts, post-surgical stenosis, absence of vas deferens *Other*: lack of sexual abstinence prior to collection, incomplete retrograde ejaculation
Hyperspermia	Ejaculated semen greater than 6.3 mL	No known etiologies

Table 3

Potential colors of male ejaculate with known etiologies

Color	White-gray	Yellow-orange	Pink-red	Brown-black
Causes	• Normal color • Minerals and vitamins inherent to prostatic and seminal fluid	• Normal accumulation of lipofuscin with age or time spent abstinent • Contamination of semen by urine • Pharmaceuticals and supplements (e.g., rifampin) • Pyospermia/Leukocytospermia caused by STI, orchitis, or prostatitis • Bilirubin accumulation (e.g., jaundice) • Dyes from food and/or drink/ contaminants	• Hematospermia with unoxidized blood • Secondary to prostatitis, UTI, orchitis, stones, iatrogenic injury, trauma-induced tearing of the testiculo-urethral tract, benign or malignant tumors of the testicles, prostate and/or urethra • Idiopathic (20–30% of cases) • Uric acid accumulation • Schistosomiasis • Food and drugs	• Hematospermia with oxidized blood • Spinal cord injury • Uric acid accumulation • Schistosomiasis • Heavy metal toxicity (platinum, manganese, lead, nickel)