Approximately 8%–15% of couples are unable to conceive after 1 year of unprotected intercourse (1). A male factor is solely responsible in ~20% of infertile couples and contributes in another 30%–40% of couples (2). A male infertility factor is often defined by abnormal semen parameters but may be present even when the semen analysis is normal. The purpose of this document is to provide clinicians with principles and strategies for the evaluation of couples with male infertility problems.

**GOALS OF EVALUATION**

Male infertility can be due to a variety of conditions, many, but not all, of which can be identified and treated. When the cause of abnormal semen parameters cannot be identified, as is true in many patients, the condition is termed idiopathic. Rarely, patients with normal semen quality may have sperm that either are incapable of oocyte fertilization or harbor genetic abnormalities that prevent normal fetal development.

Ideally, the identification and treatment of correctable conditions will improve the male partner’s fertility and allow conception to be achieved naturally. Detection of certain genetic causes of male infertility provides the opportunity to inform affected couples about the risk of transmitting genetic abnormalities that may affect the health of offspring, may affect the chance for successful treatment, and can help to guide treatment options. Evaluation of the infertile man also is aimed at identifying any underlying medical conditions that may present as infertility. Some, such as testicular cancer and pituitary tumors, can have serious health consequences if not properly diagnosed and treated (3).

**INDICATIONS FOR EVALUATION**

Evaluation for infertility is indicated for couples who fail to achieve a successful pregnancy after ≥12 months of regular unprotected intercourse. Earlier evaluation and treatment may be justified, based on medical history and physical findings and is warranted after 6 months for couples in which the female partner is >35 years old (4). Men having concerns about their future fertility also merit evaluation.

At a minimum, the initial screening evaluation of the male partner of an infertile couple should include a reproductive history and analysis of at least one semen sample. If the initial evaluation is abnormal, then referral to someone experienced in male reproduction is recommended.

**Reproductive History**

The reproductive history should include: 1) coital frequency and timing; 2) duration of infertility and previous fertility; 3) childhood illnesses and developmental history; 4) systemic medical illnesses (such as diabetes mellitus and upper respiratory diseases); 5) previous surgery; 6) medications and allergies; 7) sexual history (including sexually transmitted infections); and 8) exposures to gonadotoxins (including environmental and chemical toxins and heat). Previous fertility does not exclude the possibility of a newly acquired, secondary, male infertility factor. Evaluation is the same for men with primary infertility (never having fathered a pregnancy) and secondary infertility (having previously fathered a pregnancy).

**Semen Analysis**

Semen analysis is the cornerstone of the laboratory evaluation of the
infertile man and helps to define the severity of the male factor. Physicians should provide patients with standardized instructions for semen collection, including a defined pre-test abstinence interval of 2–5 days. Although a standard duration of abstinence is important for evaluation of semen parameters, some men with severe oligozoospermia can have equal or better sperm concentration with a short (hours) period of abstinence, supporting the potential use of multiple semen analyses during assisted reproductive technology treatment cycles (5–7). Semen can be collected by means of masturbation into a specimen cup or by intercourse with the use of special semen collection condoms that do not contain substances toxic to sperm. Ideally, the specimen should be collected at the laboratory. If collected at home, the specimen should be kept at room or body temperature during transport and examined in the laboratory within 1 hour of collection. To ensure accurate results, the laboratory should have a quality control program for semen analysis that conforms to the standards outlined in the Clinical Laboratory Improvement Amendments (CLIA); additional information including proficiency testing can be found on the CLIA website [8].

The semen analysis provides information on semen volume as well as sperm concentration, motility, and morphology (Table 1) [9]. Methods for semen analysis are discussed in many textbooks, and detailed laboratory protocols have been published by the World Health Organization (WHO) [10]. The diagnosis of azoospermia can be established only after the specimen is centrifuged (preferably at 3,000g) for 15 minutes and the pellet is examined. The current WHO criteria for evaluating sperm morphology [10] are similar to the “strict criteria” described by Kruger (Tygerberg) [11, 12], in that relatively few sperm are classified as having normal morphology, even in semen obtained from fertile men. Strict sperm morphology has been used to identify couples at risk for poor or failed fertilization with the use of standard in vitro fertilization (IVF) techniques [11] and thus to identify those who may be candidates for intracytoplasmic sperm injection (ICSI) [13]. However, the value and necessity for ICSI in those having isolated abnormalities in strict morphology has been questioned [14].

Clinical reference ranges have been established for sperm concentration, motility, and morphology to help classify men as fertile or subfertile [15]. The semen parameters of men with documented fertility have been compared with those of infertile men among couples participating in a clinical trial of superovulation and intrauterine insemination (IUI). Sperm parameters that predicted male fertility were sperm concentration >48 million/mL, sperm motility >63%, and sperm morphology >12% normal (strict criteria). Parameters that predicted male subfertility were sperm concentration <13.5 million sperm/mL, sperm motility <32%, and sperm morphology <9% normal. Values between the fertile and subfertile thresholds were considered to be “indeterminate” [16]. Although each sperm parameter could predict fertility and subfertility, none was a powerful discriminator. It is important to emphasize that normal reference values for semen parameters do not reflect normal sperm concentration in the general population, nor do they equate with the minimum values required for conception; men with semen variables outside the reference ranges may be fertile, and conversely, men having values within the reference range still may be infertile.

**COMPONENTS OF A COMPLETE EVALUATION FOR MALE INFERTILITY**

When the initial screening evaluation reveals an abnormal male reproductive history or demonstrates abnormal semen parameters, a thorough evaluation by a urologist or other specialist in male reproduction is indicated. More detailed evaluation of the male partner should be considered also in couples with unexplained infertility and those who remain infertile after successful treatment of identified female infertility factors.

The more thorough evaluation for male infertility should expand on the screening evaluation by including a complete medical history and physical examination performed by a urologist or other specialist in male reproduction. Based on the results obtained, additional tests and procedures may be recommended, including serial semen analyses, endocrine evaluation, post-ejaculatory urinalysis, ultrasonography, specialized tests on semen and sperm, and genetic screening.

**Medical History**

The patient’s medical history can identify risk factors and behaviors or lifestyles that could have significant impact on male infertility. In addition to all of the elements of the reproductive history described above, the medical history should be expanded to include: 1) a complete review of systems; 2) family reproductive history; and 3) a detailed social history, including any past or current use of anabolic steroids, recreational drugs, tobacco, and alcohol.

**Physical Examination**

A general physical examination is an integral part of the evaluation of infertile men. Particular attention should be directed to the genitalia, including: 1) examination of the penis, noting the location of the urethral meatus; 2) palpation and

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**TABLE 1**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Reference value</th>
</tr>
</thead>
<tbody>
<tr>
<td>On at least two occasions</td>
<td></td>
</tr>
<tr>
<td>Ejaculate volume</td>
<td>1.5 mL</td>
</tr>
<tr>
<td>pH</td>
<td>7.2</td>
</tr>
<tr>
<td>Sperm concentration</td>
<td>$15 \times 10^6$ spermatozoa/mL</td>
</tr>
<tr>
<td>Total sperm number</td>
<td>$39 \times 10^6$ spermatozoa/ejaculate</td>
</tr>
<tr>
<td>Percentage motility</td>
<td>40%</td>
</tr>
<tr>
<td>Forward progression</td>
<td>32%</td>
</tr>
<tr>
<td>Normal morphology</td>
<td>4% normal</td>
</tr>
<tr>
<td>And</td>
<td></td>
</tr>
<tr>
<td>Sperm agglutination</td>
<td>Absent</td>
</tr>
<tr>
<td>Viscosity</td>
<td>$\leq 2$ cm thread after liquefaction</td>
</tr>
</tbody>
</table>

Note: Data from World Health Organization, 2010 [10].
 measurement of the testes; 3) the presence and consistency of both vasa and epididymides; 4) the presence or absence of a varicocele; 5) secondary sex characteristics, including body habitus, hair distribution, and breast development; and 6) digital rectal examination where indicated. The diagnosis of congenital bilateral absence of the vasa deferentia (CBAVD) is established by physical examination; scrotal exploration is unnecessary.

**OTHER PROCEDURES AND TESTS FOR ASSESSING MALE INFERTILITY**

**Endocrine Evaluation**

Hormonal abnormalities of the hypothalamic-pituitary-testicular axis are well recognized, but uncommon, causes of male infertility. Endocrine disorders are extremely uncommon in men with normal semen parameters.

An endocrine evaluation is indicated for men having: 1) abnormal semen parameters, particularly when the sperm concentration is <10 million/mL; 2) impaired sexual function; or 3) other clinical findings that suggest a specific endocrinopathy. Some experts think that all infertile men merit an endocrine evaluation, but there is no established consensus of opinion. The minimum initial hormonal evaluation should include measurements of serum FSH and total testosterone (T) concentrations. When the total T level is low (<300 ng/mL), more extensive evaluation is indicated and should include a second early morning measurement of total T and measurements of free testosterone (Tf), LH, and prolactin (PRL). Although serum gonadotropin concentrations vary because they are secreted in a pulsatile manner, a single measurement usually is sufficient to determine the clinical endocrine status. The relationships among serum T, LH, follicle-stimulating hormone (FSH), and PRL concentrations help to provide an understanding of the source of abnormal total T levels (Table 2). Whereas many men with abnormal spermatogenesis have a normal serum FSH level, a markedly elevated serum FSH concentration clearly indicates an abnormality in spermatogenesis. In individuals with an FSH level in the upper normal range, there may be impaired spermatogenesis as well. Measurement of the thyroid-stimulating hormone (TSH) concentration also should be obtained in men who require a more thorough endocrine evaluation.

Serum inhibin B concentration has been proposed as a marker for spermatogenesis. Inhibin B levels are significantly lower in infertile men than in fertile men and correlate better than FSH levels with sperm parameters [17]. Given the significantly greater cost of measuring inhibin B, FSH currently remains the preferred test for screening purposes.

**Post-ejaculatory Urinalysis**

A low-volume or absent antegrade ejaculate suggests incomplete semen collection, retrograde ejaculation, lack of emission, ejaculatory duct obstruction, hypogonadism, or CBAVD. To exclude retrograde ejaculation, a post-ejaculatory urinalysis should be performed in men having an ejaculate volume <1.0 mL, except in those diagnosed with hypogonadism or CBAVD. It is important also to determine whether an improper or incomplete collection or a very short abstinence interval (<1 day) might be the cause.

The post-ejaculatory urinalysis is performed by centrifuging the urine specimen for 10 minutes at 300g, followed by microscopic examination of the pellet at ×400 magnification. In men with azoospermia or asperma, the presence of any sperm in the post-ejaculatory urinalysis suggests retrograde ejaculation. In men with low ejaculate volume and oligozoospermia, “significant numbers” of sperm must be observed to support the diagnosis of retrograde ejaculation; there is no consensus of expert opinion on the minimum number required [18].

**Ultrasonography**

Because nearly the entire male genital tract can be imaged easily and accurately, ultrasonography is a useful tool for detecting abnormalities of the male genital tract that may adversely affect fertility. However, ultrasonography is indicated for only a minority of infertile male patients.

**Transrectal ultrasonography.** Normal seminal vesicles are usually <1.5 cm in anteroposterior diameter [19]. Transrectal ultrasonography (TRUS) revealing dilated seminal vesicles or ejaculatory ducts and/or midline cystic prostatic structures suggests, but does not by itself establish, the diagnosis of complete or partial ejaculatory duct obstruction [20]. Affected men typically produce a low-volume acidic ejaculate containing no sperm or fructose. Men with CBAVD may exhibit similar findings because they often have absent or atrophic seminal vesicles. Men with partial ejaculatory duct obstruction often, but not always, exhibit low semen volume, oligoasthenospermia, and poor progressive motility. Some experts recommend routine TRUS for oligozoospermic men having low-volume ejaculates, palpable vasa, and normal physical findings that suggest a specific endocrinopathy.

### TABLE 2

<table>
<thead>
<tr>
<th>Basal hormone levels in various clinical states.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Clinical condition</strong></td>
</tr>
<tr>
<td>Normal spermatogenesis</td>
</tr>
<tr>
<td>Hypogonadotropic hypogonadism</td>
</tr>
<tr>
<td>Abnormal spermatogenesis</td>
</tr>
<tr>
<td>Complete testicular failure/hypogonadotropic hypogonadism</td>
</tr>
<tr>
<td>PRL-secreting pituitary tumor</td>
</tr>
</tbody>
</table>

| a Many men with abnormal spermatogenesis have a normal serum FSH, but a marked elevation of serum FSH is clearly indicative of an abnormality in spermatogenesis. |

Sperm DNA integrity is maintained in part by the effect of di- 
de cross-links between protamines that allow for the 
sperm DNA damage can occur as a result of intrinsic factors, such as protamine 
DNA fragmentation tests have been developed to measure sperm DNA 
assays, specifically analyze the number of breaks in the 
DNA. Indirect tests, such as the sperm chromatin structure 
assay (SCSA), define abnormal chromatin structure as an 
after vasectomy. Risk factors for ASA formation include trauma, torsion, biopsy, orchitis, testicular cancer, and 
Sperm viability can be assessed by mixing fresh semen with 
method, such as eosin Y or trypan blue, or by the use of 
Sperm viability tests can also be identified by means of incubation in pentoxifylline. Viable sperm will develop motility after exposure to 
Sperm DNA damage is more common in infertile men and 
Sperm DNA damage is also associated with sponta-
neous recurrent miscarriage. However, existing data relating to the relationship between abnormal DNA integrity and 
reproductive outcomes are too limited to routinely 
block penetration of the cervical mucus, and prevent fertili-

testicular size with normal serum T, although there is no consensus on this.

Scrotal ultrasonography. Careful physical examination can identify most scrotal pathology, including varicoceles, sper-
matoceles, absent vasa, epididymal induration, and testicular masses. Scrotal ultrasonography can identify occult varico-
cele that are not palpable, but such lesions have no demonstrat-
ed clinical significance (21). Scrotal ultrasonography can be helpful for better defining vague or ambiguous physical 
examination findings or abnormalities (including apparent masses) and can be performed in men having tests located in 
the upper scrotum, a small scrotal sac, or other anatomy 
that hinders physical examination. Scrotal ultrasonography 
should also be considered for men presenting with infertility and 
and ASA are likely to have reproductive tract obstruction. 
Otherwise, routine testing for ASA is not indicated.

Sperm Viability Tests

Sperm viability can be assessed by mixing fresh semen with 
supravital dye, such as eosin Y or trypan blue, or by the use of 
the hypoosmotic swelling (HOS) test (10). These assays 
determine whether nonmotile sperm are viable by identi-
fying which sperm have intact cell membranes. In dye tests, 
viable sperm actively exclude the dye and remain colorless whereas nonviable sperm readily take up the stain. Unfortu-
nately, sperm judged to be viable by means of dye tests can 
not be used for IVF. In the HOS test, viable nonmotile sperm, 
which swell when incubated in a hypoosmotic solution, can 
be used successfully for ICSI (26). Viable nonmotile sperm 
can also be identified by means of incubation in pentoxifylline. Viable sperm will develop motility after exposure to 
Sperm viability tests can also be identified by means of incubation in pentoxifylline (27).

Sperm Deoxyribonucleic Acid (DNA) 
Fragmentation Tests

DNA integrity is important for normal embryo development. Sperm DNA integrity is maintained in part by the effect of di-
sulfide cross-links between protamines that allow for the 
compaction of chromatin in the nucleus. Sperm DNA damage 
can occur as a result of intrinsic factors, such as protamine 
deficiency and mutations affecting DNA compaction, or 
from extrinsic factors, such as heat, radiation, and gonado-
toxins. The term “DNA fragmentation” refers to denatured or damaged sperm DNA that can not be repaired. A number of 
clinical tests have been developed to measure sperm DNA 
fragmentation rates. Direct methods, such as the single-cell gel electrophoresis assay (Comet) and terminal deoxynucleo-
tide transferase-mediated dUTP nick-end labeling (TUNEL) 
assays, specifically analyze the number of breaks in the 
DNA. Indirect tests, such as the sperm chromatin structure 
assay (SCSA), define abnormal chromatin structure as an 
after vasectomy. Risk factors for ASA formation include trauma, torsion, biopsy, orchitis, testicular cancer, and 
Sperm viability tests can also be identified by means of incubation in pentoxifylline (27).

Sperm viability tests can also be identified by means of incubation in pentoxifylline (27).
recommend any of these tests for the male partner in an infertile couple, but the effect of abnormal sperm DNA fragmentation on the value of IUI or IVF and ICSI results may be clinically informative [31]. Although no treatment for abnormal DNA integrity has been proven to have clinical value, varicocele repair and antioxidant use may affect sperm DNA integrity. Sperm retrieved from the tests tend to have better sperm DNA quality in men with abnormal ejaculated sperm DNA integrity [32]. Because the prognostic clinical value of DNA integrity testing may not affect the treatment of couples, the routine use of DNA integrity tests in the clinical evaluation of male-factor infertility is controversial [33].

Less Commonly Used Specialized Tests
Numerous other tests of sperm function have been used predominantly in research studies. Sperm penetration assays may detect defects in sperm fertilizing capacity and could identify patients who would benefit from application of ICSI. However, because ICSI is routinely used during IVF for male-factor infertility couples, this test is rarely of any clinical value. The acrosome reaction of human sperm can be detected with the use of specialized staining techniques. Rates of spontaneous acrosome reactions and acrosome reactions induced by agents such as calcium ionophore and progesterone have been measured. Sperm from infertile men tend to demonstrate higher acrosome levels spontaneously but lower levels in the presence of inducers [34]. A number of biochemical tests of sperm function have been studied, including measurements of sperm creatine kinase [35] and reactive oxygen species (ROS). ROS appear to be generated by both seminal leukocytes and sperm cells and can interfere with sperm function by peroxidation of sperm lipid membranes and creation of toxic fatty acid peroxides [36]. Other tests and procedures have been used to select sperm for ICSI and may identify gametes with better quality, including hyaluronic acid binding, membrane maturity testing, apoptotic evaluation, and magnified sperm examination [37]. However, these tests have a very limited role in the evaluation of male infertility because they have limited clinical utility and typically do not affect treatment.

Genetic Screening
Genetic abnormalities can cause infertility by affecting sperm production or sperm transport. Men with nonobstructive azoospermia or severe oligozoospermia (<5 million/mL) are at increased risk for having a genetic abnormality compared to fertile men [38]. The most common genetic abnormalities found in such men are numeric and structural chromosomal aberrations that impair testicular function and Y-chromosome microdeletions that are associated with isolated defects in spermatogenesis. In addition, most men with CBAVD can be assumed to have an abnormality of the cystic fibrosis transmembrane conductance regulator (CFTR) gene. When indicated, efforts to identify genetic causes for infertility can have a major impact on the choice and outcome of treatment.

Cystic fibrosis gene mutations. There is a strong association between CBAVD and mutations of the CFTR gene, which is located on chromosome 7 [39]. Almost all men with clinical cystic fibrosis exhibit CBAVD. Additionally, as many as 80% of men with CBAVD have documented mutations of the CFTR gene. Failure to detect a CFTR abnormality in men with CBAVD does not exclude the presence of a mutation that cannot be identified with currently available methods. Therefore, most men with CBAVD should be assumed to have a CFTR gene mutation unless they have renal anomalies. To determine the risk of conceiving a child affected with cystic fibrosis, it is important to test the female partner of an affected man. Even if the female partner is negative according to currently available testing, the couple remains at some risk because some of the less common mutations may be missed unless the entire gene is sequenced.

The prevalence of CFTR mutations is also increased among men with azoospermia related to congenital bilateral obstruction of the epididymides and those with unilateral vasa agenesis. Consequently, genetic evaluation should be considered for those having either abnormality. Some men presenting with either unilateral or bilateral vasa agenesis and unilateral renal agenesis have the mesonephric duct abnormalities associated with hereditary renal dysplasia, which has an autosomal dominant form of inheritance with incomplete penetrance and variable expression. These patients do not have CFTR mutations and require genetic counseling before IVF [40, 41].

Karyotypic chromosomal abnormalities. The prevalence of chromosomal abnormalities is increased in infertile men and inversely proportional to sperm count; the prevalence is 10%–15% in azoospermic men [42], ~5% in men with severe oligozoospermia (<5 million/mL), and <1% in men with normal sperm concentrations [43]. Sex chromosomal aneuploidy (Klinefelter syndrome; 47,XXY) accounts for about two thirds of all chromosomal abnormalities observed in infertile men [44]. The prevalence of structural autosomal abnormalities, such as inversions and balanced translocations, also is higher in infertile men than in the general population [45]. Rare azoospermic men may be found to have the 46,XX disorder of sexual development resulting from translocation of sex-determining region Y (SRY) to one of their X chromosomes. Couples in which the male partner has a gross karyotypic abnormality are at increased risk for miscarriages and for having children with chromosomal and congenital defects. Therefore, men with nonobstructive azoospermia or severe oligozoospermia should be evaluated with a high-resolution karyotype before using their sperm to perform ICSI.

Y-chromosome microdeletions. Microdeletions of clinically relevant regions of the Y chromosome have been found in 7% of infertile men with severely impaired spermatogenesis, compared with 2% of normal men. However, the percentage of men with Y-chromosome microdeletions increases to 16% in men with azoospermia or severe oligozoospermia [46]. Such microdeletions are too small to be detected by standard karyotyping, but they can be identified with the use of polymerase chain reaction techniques to analyze sequence-tagged sites that have been mapped along the entire length of the Y chromosome.

Most deletions causing azoospermia or oligozoospermia occur in regions of the long arm of the Y chromosome.
(Yq11) known as the azoospermia factor (AZF) regions, designated as AZFa (proximal), AZFb (central), and AZFc (distal). It appears that these regions, and possibly other regions of the Y chromosome, contain multiple genes necessary for spermatogenesis. For example, the DAZ (deleted in azoospermia) gene, which encodes a transcription factor usually present in men with normal fertility, is located in the AZFc region.

The specific location of the deletion along the Y chromosome influences its effect on spermatogenesis. Many men with a microdeletion in the AZFc region of the Y chromosome have severe oligozoospermia. Others with AZFc region deletions are azoospermic but may still produce sufficient numbers of sperm to allow testicular sperm extraction. Sperm production in such men appears to be stable over time, and the results of ICSI are not affected adversely by the AZFc deletion (47). In contrast, deletions involving the entire AZFb region appear to predict a very poor prognosis for sperm retrieval (48). The same may be true for men having deletions involving the entire AZFa region of the Y chromosome (49).

Sons of individuals with Y-chromosome microdeletions will inherit the abnormality and, therefore, may also be infertile (50). Although a microdeletion of the Y chromosome is not known to be associated with other health problems, few data exist regarding the phenotypes of the sons of fathers with such genetic abnormalities. A recent report showed that some men with Y-chromosome microdeletions had abnormalities of the pseudoautosomal regions (PARs) of the Y chromosome. Although most of these men had some sperm production, 16% of men had genetic aberrations of the short-stature-homeo-box (SHOX) gene, the best known production, 16% of men had genetic abnormality, because there may be other, currently unknown, gene sequences on the Y or other chromosomes that also might be required for normal spermatogenesis. Conversely, some Y-chromosome microdeletions are rarely found in fertile or subfertile males who have fathered children (46, 52). Y-Chromosome analysis should be offered to men who have nonobstructive azoospermia or severe oligozoospermia before performing ICSI with their sperm.

**SPERM CHROMOSOME ANEUPLOIDY**

Sperm DNA aneuploidy can be assessed by fluorescent in situ hybridization technology (53). One study has reported that up to 6% of men presenting with infertility and a normal karyotype had an increased frequency of meiotic alterations detectable in their sperm (54). Men with the highest risk of sperm aneuploidy are those with karyotypic abnormalities, severely abnormal sperm morphology, and nonobstructive azoospermia (53). Patients with recurrent pregnancy loss and recurrent IVF failure also may benefit from sperm aneuploidy testing (55, 56). Currently, limitations to the routine use of this technology include cost, inability to screen the actual sperm used in ICSI, and difficulty of assigning a meaningful risk assessment to couples based on the test results (57).

**SUMMARY**

Men with nonobstructive azoospermia or severe oligozoospermia (<5 million/mL) are at increased risk for having a definable genetic abnormality and should be offered karyotype and Y-chromosome analysis before performing ICSI with their sperm. Genetic counseling may be offered when a genetic abnormality is suspected in either the male or the female partner and should be provided whenever a genetic abnormality is detected.

**CONCLUSION**

An initial screening evaluation of the male partner of an infertile couple is indicated when pregnancy has not occurred after 12 months of unprotected intercourse or after 6 months of failure to conceive when the female partner is >35 years old. Earlier evaluation may be warranted when medical history and physical findings indicate or suggest specific male or female infertility risk factors and for men who question their reproductive potential.

A thorough evaluation by a urologist or other specialist in male reproduction, including a complete medical and reproductive history and physical examination, should be performed if the initial screening evaluation reveals an abnormal male reproductive history or demonstrates abnormal semen parameters. Additional tests aimed at defining the cause may be required.

**ACKNOWLEDGMENTS**

This report was developed under the direction of the Practice Committee of the American Society for Reproductive Medicine (ASRM) as a service to its members and other practicing clinicians. Although this document reflects appropriate management of a problem encountered in the practice of reproductive medicine, it is not intended to be the only approved standard of practice or to dictate an exclusive course of treatment. Other plans of management may be appropriate, taking into account the needs of the individual patient, available resources, and institutional or clinical practice limitations. The Practice Committee and the Board of Directors of the American Society for Reproductive Medicine have approved this report.

The following members of the ASRM Practice Committee participated in the development of this document. All Committee members disclosed commercial and financial relationships with manufacturers or distributors of goods or services used to treat patients. Members of the Committee who were found to have conflicts of interest based on the relationships disclosed did not participate in the discussion.

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**REFERENCES**


