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EAU Guidelines on Male Infertility

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1. Definition

"Infertility is the inability of a sexually active, non-contracepting couple to achieve pregnancy in one year" [WHO, 2000].

2. Epidemiology and aetiology

About 25% of couples do not achieve pregnancy within 1 year, 15% of the couples seek medical treatment for infertility and ultimately less than 5% remain childless. Infertility affects both men and women. Male causes for infertility are found in 50% of these couples. In many couples, however, male and female factors are present. In case of a single factor the fertile partner may compensate for the less fertile partner. Infertility then usually becomes manifest if both partners are subfertile. This explains why in infertile couples there is often a coincidence of male and female factors.

Reduced male fertility can be the result of congenital and acquired urogenital abnormalities, infections of the male accessory glands, increased scrotal temperature (varicocele), endocrine disturbances, genetic abnormalities and immunological factors [1]. In 40–60% of cases the only abnormality is the semen analysis and there is no relevant history or abnormality on physical examination and endocrine laboratory testing (idiopathic male infertility). Semen analysis reveals a decreased number of spermatozoa (oligozoospermia), decreased motility (asthenozoospermia) and many abnormal forms on morphological examination (teratozoospermia). Usually, these abnormalities come together and are described as the OAT-syndrome (oligo-astheno-teratozoospermia) (Table 1).

3. Prognostic factors

The main factors influencing the prognosis in infertility are:

- Duration of infertility
- Age and fertility status of the female partner
- Primary or secondary infertility
- Results of semen analysis

When the duration of infertility exceeds four years of unprotected intercourse, the conception rate per month is only 1.5%. At present, in many western

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Table 1
Aetiology and distribution(%) of male infertility among 7057 men (1)

No demonstrable cause	48.5
Sexual factors	1.7
Urogenital infection	6.6
Congenital anomalies	2.1
Acquired factors	2.6
Varicocele	12.3
Endocrine disturbances	0.6
Immunological factors	3.1
Idiopathic abnormal semen (OAT syndrome)	26.4
Other abnormalities	3.0

countries women postpone their first pregnancy until they have finished their education and have started a professional career. However, the fertility of a woman of 35 years is only 50% of the fertility potential of a woman aged 25 years; by the age of 38 years this has reduced to only 25%, and over the age of 40 years it is less than 5%. Female age is the most important single variable influencing outcome in assisted reproduction. In the diagnosis and management of male infertility it is essential to consider the fertility chances of the female partner since this might determine the final outcome [2].

4. Medical history and physical examination

Investigation of the male partner should include full medical history and physical examination according to the standardised scheme published by WHO (1) so that any causative factor can be diagnosed and, if possible, treated. Also this helps implement evidence-based medicine in this interdisciplinary field of reproductive medicine.

5. Investigations

Semen analysis should follow the guidelines of the World Health Organisation (WHO) Laboratory Manual for Human Semen and Sperm-Cervical Mucus Interaction, which is in its fourth edition [3].

5.1. Hormonal investigation

Endocrine malfunctions as the primary cause of male infertility is rare. Hormonal screening can be limited to determining follicle-stimulating hormone (FSH), luteinizing hormone (LH) and testosterone levels. In men diagnosed with azoospermia or extreme OAT, it is important to distinguish between obstructive and non-obstructive causes. A criterion with reasonable predictive value for obstruction is a normal FSH with bilaterally a normal testicular volume. However, 29% of men with a normal FSH appear to have defective spermatogenesis.

5.2. Microbiological assessment

Indications for microbiological assessment include abnormal urine samples, urinary tract infections, prostatitis, epididymitis, silent ejaculate infections (MAGI) and sexually transmitted diseases (STDs). The clinical implications of white blood cells detected in a semen sample is as yet undetermined. However, in combination with a small ejaculate volume, this may point to a (partial) obstruction of the ejaculatory ducts caused by a (chronic) infection of the prostate or seminal vesicles. Genital infections may instigate the production of spermatotoxic free oxygen radicals. Gonorrhoea and chlamydia trachomatis can also cause obstruction of the epididymis and the vas deferens [4].

5.3. Ultrasonography

Scrotal ultrasound can be helpful in the assessment of testicular size, in finding signs of obstruction, such as dilatation of the rete testis, enlarged epididymis with cystic lesions and absence of the vas deferens, to exclude signs of testicular dysgenesis like inhomogeneous testicular architecture and microcalcifications and in the assessment of reflux of blood in men with a varicocele [5].

TRUS can be performed on patients with a low seminal volume and in whom distal obstruction is suspected, if possible with high resolution and high frequency (7 MHz) biplane transducers. Seminal vesicle enlargement (anterior-posterior diameter $\geq$15 mm) and seminal vesicle roundish anechoic areas are TRUS anomalies more often associated with ejaculatory duct obstruction, especially when the semen volume is $\leq$1.5 ml. Other known anomalies in cases of obstructive azoospermia are Müllerian duct cysts or urogenital sinus/ejaculatory duct cysts and ejaculatory duct calcifications [6].

5.4. Testicular biopsy

A diagnostic testicular biopsy may be performed in men with azoospermia, normal testicular volume and normal FSH to differentiate between obstructive and non-obstructive azoospermia. Testicular biopsy can also be performed as part of a therapeutic process in patients with clinical evidence of non-obstructive azoospermia who decide to undergo ICSI. About 50–60% of men with non-obstructive azoospermia have some seminiferous tubules with spermatozoa that can be used for ICSI [7].

Table 2
Causes of spermatogenic failure

Congenital factors	
1.	Anorchia
2.	Testicular dysgenesis/cryptorchidism
3.	Genetic abnormalities (Klinefelter's syndrome, Y chromosome deletions)
4.	Germ cell aplasia (sertoli cell only syndrome)
5.	Spermatogenic arrest (maturation arrest)
Acquired factors	
6.	Trauma
7.	Testicular torsion
8.	Post-inflammatory (orchitis) forms
9.	Exogenous factors (medications, cytotoxic drugs, irradiation, heat)
10.	Systemic diseases (liver cirrhosis, renal failure)
11.	Testicular tumour
12.	Varicocele
13.	Surgeries that can damage vascularisation of the testes
Idiopathic forms	
14.	OAT-syndrome

6. Primary spermatogenic failure

6.1. Definition

Primary spermatogenic failure is defined as impaired spermatogenesis originating from causes different than hypothalamic-pituitary diseases. The severe forms of primary spermatogenic failure have a clinical presentation as non-obstructive azoospermia [1] (Table 2).

6.2. Aetiology

Typical findings from the physical examination of a patient with spermatogenic failure may be abnormal secondary sexual characteristics, gynaecomastia and low testicular volume (<15 cc per gonad) and/or consistency. FSH may be elevated (*Hyper*gonadotrophic hypogonadism) or normal [1].

7. Obstructive azoospermia

7.1. Definition

Obstructive azoospermia means the absence of both spermatozoa and spermatogenic cells in semen and post-ejaculate urine due to bilateral obstruction of the seminal ducts.

7.2. Classification

Intratesticular obstruction has been reported in 15% of obstructive azoospermia [8] and is usually caused by post-inflammatory obstruction of the rete testis.

Epididymal obstruction is the most common cause of obstructive azoospermia, affecting 30–67% of azoospermic men with a serum FSH less than twice the upper limit of normal [9]. Congenital forms of obstruction (disjunction between efferent ductless and corpus epididymidis, agenesis/atresia of a short part of the epididymis) are rare. Young's syndrome, characterised by proximal epididymal obstruction and chronic sinopulmonary infections [10], results from a mechanical blockage due to debris within the proximal epididymal lumen.

Among the acquired forms, those secondary to acute (gonococcal) and subclinical (e.g. chlamydial) epididymitis are considered to be most frequent [11]. Azoospermia caused by surgery may occur after bilateral epididymal cyst removal.

Vas deferens obstruction following vasectomy is the most frequent cause of acquired obstruction. About 2–6% of these men request vasectomy reversal. Of those undergoing vasovasostomy, 5–10% will also have an epididymal blockage due to tubule rupture, making vasoepididymostomy mandatory [12].

Congenital bilateral absence of the vas deferens (CBAVD) is found in 1:1600 men and in all men with cystic fibrosis. Men with CBAVD appear to have mutations of the cystic fibrosis gene in 85% of the cases. CBAVD can therefore be considered a genital form of cystic fibrosis [13].

Ejaculatory duct obstruction is found in about 1–3% of obstructive azoospermia [14]. These obstructions can be classified as cystic or post-inflammatory. Cystic obstructions are usually congenital (Müllerian duct cyst or urogenital sinus/ejaculatory duct cysts) and are medially located in the prostate between the ejaculatory ducts. In urogenital sinus anomalies, one or both ejaculatory ducts empty into the cyst, while in Müllerian duct anomalies, ejaculatory ducts are laterally displaced and compressed by the cyst [15]. Post-inflammatory obstructions of the ejaculatory duct are usually secondary to urethroprostatitis [16].

Congenital or acquired complete obstructions of the ejaculatory ducts are commonly associated with low semen volume, decreased or absent seminal fructose and acid pH. The seminal vesicles are usually dilated (anterior-posterior diameter >15 mm) on transrectal ultrasound [6].

Typical clinical findings in men with obstructive azoospermia are a normal testicular volume (>15 cc per testis), enlarged and hardened epididymidis, nodules in the epididymis or vas deferens, absence or partial atresia of the vas deferens, signs of urethritis or prostatitis and Prostatic abnormalities on rectal examination. Obstructive lesions of the seminal tract should be suspected in azoospermic or severely

oligozoospermic patients with normal-sized testes and normal endocrine parameters [1].

8. Genetic disorders in infertility

Knowledge of genetic abnormalities in infertility is mandatory for every urologist working in andrology.

8.1. Chromosomal abnormalities

In a survey of pooled data from 11 publications, including a total of 9,766 infertile men, the incidence of chromosomal abnormalities was found to be 5.8% [17]. Of these, sex chromosome abnormalities accounted for 4.2% and autosomal abnormalities for 1.5%. In comparison, the incidence of abnormalities in pooled data from three series totalling 94,465 newborn male infants was only 0.38%, of which 131 (0.14%) were sex chromosome abnormalities and 232 (0.25%) were autosomal abnormalities [18]. The possibility of abnormalities increases with the severity of impaired spermatogenesis [19]. Standard karyotype analysis should therefore be offered to all men with damaged spermatogenesis who are seeking fertility treatment by IVF/ICSI.

Klinefelter's syndrome is the most frequent sex chromosome abnormality. Adult men with Klinefelter's syndrome have small firm testicles that are devoid of germ cells. The phenotype can vary from a normally virilized man to one with stigmata of androgen deficiency, including female hair distribution, scanty body hair and long arms and legs because of late epiphyseal closure.

Leydig cell function is commonly impaired in men with Klinefelter's syndrome [20]. Testosterone levels may be normal or low, oestradiol levels normal or elevated and FSH levels are increased. Surprisingly, libido is often normal despite low testosterone levels, but androgen replacement may be needed with ageing.

Patients with Klinefelter's syndrome have an increased chance of producing 47, XXY spermatozoa. When IVF/ICSI is performed, pre-implantation diagnosis should be used or, if not available, amniocentesis and karyotype analysis [21].

8.2. Y chromosome microdeletions

A large number of case series have been published on microdeletions of the long arm of the Y chromosome involving areas where the genes are to do with spermatogenesis. Although some microdeletions may even occur in the fertile population, they are more prevalent in the infertile population. The most commonly reported abnormality is a microdeletion in the region which encompasses the DAZ gene. Men with microdeletions of the Y chromosome do not have any phenotypic abnormalities other than abnormal spermatogenesis [22].

8.3. Cystic fibrosis mutations and male infertility

Cystic fibrosis (CF), a fatal autosomal-recessive disorder, is the most common genetic disease of Caucasians. Men with CF are azoospermic due to congenital bilateral absence of the vas deferens (CBAVD). Men with isolated CBAVD often carry mutations of the cystic fibrosis transmembrane conductance regulator (CFTR) gene. This gene is located on the short arm of chromosome 7 and encodes a membrane protein that functions as an ion channel and also influences the formation of the ejaculatory duct, seminal vesicle, vas deferens and distal two thirds of the epididymis (Wolffian duct structures) [23].

When a man has CBAVD, it is important to test him and his partner for cystic fibrosis mutations. If she is also found to be a carrier, the couple must very carefully consider whether to proceed with ICSI using the husband's sperm, as the chance of a baby with cystic fibrosis will be 25% if he is heterozygous or 50% if he is homozygous [24]. If the female partner is negative for known mutations, her chance of being a carrier of unknown mutations is about 0.4%. In these circumstances, the possibility of her heterozygous partner fathering a child with cystic fibrosis is approximately 1:400 [25].

Genetic counselling is mandatory in couples with a genetic abnormality found in clinical or genetic investigation and in patients who carry an (potential) inheritable disease.

9. Varicocele

Varicocele is a common abnormality with the following andrological implications:

- Failure of ipsilateral testicular growth and development
- Symptoms of pain and discomfort
- Reduced fertility

9.1. Classification

The following classification of varicocele [1] is useful in clinical practice:

Subclinical: Not palpable or visible at rest or during Valsalva manoeuvre, but demonstrable by scrotal ultrasound and colour Doppler examination).

Grade 1: Palpable during Valsalva manoeuvre but not otherwise
Grade 2: Palpable at rest, but not visible
Grade 3: Visible and palpable at rest

9.2. Varicocele and fertility

Varicocele is a physical abnormality present in 2–22% of the adult male population [1]. It is more common in men of infertile marriages, affecting 25–40% of those with abnormal semen analysis. The exact association between reduced male fertility and varicocele is unknown, but analysis of the WHO data clearly indicates that varicocele is related to semen abnormalities, decreased testicular volume and decline in Leydig cell function.

Two large prospective randomized studies of varicocele treatment in adults gave conflicting results [26,27], the largest of them indicating benefit [27]. However, a recent review of the literature indicated no benefit (the common odds ratio was 0.85% (95% CI 0.49–1.45) [28].

Treatment of varicocele to achieve pregnancy in infertile partnerships remains controversial and all investigations to date have been subject to criticism. Further investigations are needed, particularly for younger couples.

Current information fits with the hypothesis that in some men the presence of varicocele is associated with progressive testicular damage from adolescence onwards and consequent reduction in fertility. However, in infertile couples this impaired fertility potential will only be manifest if female fertility is also reduced.

While treatment of varicocele in adolescents may be effective, there is a significant risk of overtreatment. Data from ongoing studies should provide more information in this respect.

10. Hypogonadotrophic hypogonadism

Primary hypogonadotrophic hypogonadism is caused either by hypothalamic or pituitary diseases. The failure of hormonal regulation can easily be determined [29]. Endocrine deficiency leads to a lack of spermatogenesis and testosterone secretion due to decreased secretion patterns of LH and FSH. Standard treatment is human chorionic gonadotrophin (hCG) treatment, with the later addition of human menopausal globulin (hMG), dependent on initial testicular volume. If hypogonadotrophic hypogonadism is hypothalamic in origin, a 1-year therapy with pulsatile gonadotrophin releasing hormone (GnRH) is as effective as gonadotrophins in stimulating spermatogenesis. Once pregnancy has been induced, patients can return to testosterone substitution.

Secondary hypogonadotrophic hypogonadism can be caused by obesity, some drugs, hormones and anabolic steroids.

11. Cryptorchidism and testicular tumours

Cryptorchidism is the most frequent congenital abnormality of the male genitalia with a 2–5% incidence at birth. At the age of 3 months the incidence is reduced spontaneously to 1–2%. Approximately 20% of undescended testes are nonpalpable and may be located within the abdominal cavity.

The aetiology of cryptorchidism is multifactorial and both disrupted endocrine regulation and several gene defects may be involved. For a normal descent of the testes, a normal hypothalamo-pituitary-gonadal axis is needed. Although the majority of boys with maldescended testes show no endocrine abnormalities after birth endocrine disruption in early pregnancy can potentially affect gonadal development and normal descent. It has been postulated that cryptorchidism can be the consequence of testicular dysgenesis, a developmental disorder of the gonads due to environmental and/or genetic influences early in pregnancy. This testicular dysgenesis syndrome (TDS) can result in maldescent, reduced fertility and an increased risk for malignant development [30].

11.1. Relationship with fertility

Semen parameters in men with a history of cryptorchidism are often impaired: in 2–9% of infertile patients, a history of cryptorchidism is present [31]. It has been suggested that surgical treatment performed before the age of 3 years has a positive effect on semen quality. However, paternity in men with a history of unilateral cryptorchidism is almost equal (89.7%) to paternity in men without cryptorchidism (93.7%). Also, in men with unilateral cryptorchidism paternity seems independent of the age of orchidopexy, preoperative testicular location and testicular size [32]. In men with bilateral cryptorchidism, however, oligozoospermia can be found in 31% and azoospermia in 42%. In cases of bilateral cryptorchidism paternity is only 35–53% [33].

11.2. Germ cell tumours

Cryptorchidism is a risk factor for testicular cancer development and is associated with testicular microcalcification and carcinoma in situ of the testis. In about 5–10% of testicular cancers a history of cryptorchidism can be found [34]. The risk of a germ cell

tumour is higher in men with cryptorchidism and impaired fertility: 2–6% of men with a history of cryptorchidism and 0.5–1% of infertile men will develop a testicular tumour [34].

Dysgenic testes have an increased risk of developing testicular cancer in adulthood. These cancers seem to arise from pre–malignant gonocytes or carcinoma-in-situ (CIS) cells [35]. Testicular microlithiasis can be associated with both germ cell tumours and carcinoma in situ of the testis.

11.3. Testicular microlithiasis

Microcalcifications inside the testicular parenchyma can be found in 0.6–9% of men referred for testicular ultrasound [36]. Although the true incidence in the general population is unknown it probably is a rare condition. However, the ultrasound findings are prevalent in men with germ cell tumours, cryptorchidism, testicular dysgenesis, male infertility, testicular torsion and atrophy, Klinefelter's syndrome, hypogonadism, male pseudohermaphroditism, varicocele, epididymal cysts, pulmonary microlithiasis and non-Hodgkin lymphoma. The incidence seems to increase with the use of high frequency ultrasound machines.

The relationship between testicular microlitiasis (TM) and infertility is unclear, but probably relates to dysgenesis of the testis, with slough of degenerated cells inside a obstructed seminiferous tubule and failure of the Sertoli cells to phagocytize the debris. Secondarily, calcification occurs.

Testicular microcalcifications is a condition found in testis at risk for malignant development: the reported incidence of TM in men with germ cell malignancy is 6–46%. This has resulted in the hypothesis that TM is to be considered a premalignant condition. It remains, however, to be established whether TM is present before development of the invasive testicular germ cell tumours (TGCT's), and is therefore a putative parameter for the pre-invasive stage of TGCTs, known as carcinoma in situ (CIS). On testicular biopsies in men with TM carcinoma in situ is more prevalent, especially in men with bilateral microlithiasis [36]. On the other hand TM is more often found in men with a benign testicular condition and the microcalcifications itself are not malignant.

More studies are needed to calculate the actual risk for developing a germ cell tumour and the need for ultrasonographic follow-up. It is also important to encourage and educate patients about self-examination, since this may result in early detection of germ cell tumours. The routine use of biochemical tumour markers, abdominal and pelvic CT or testicular biopsy does not seem to be justified for patients with isolated TM.

12. Male accessory gland infection

Infections of the male accessory glands are potentially correctable causes of male infertility [4,37]. In this context, urethritis and prostatitis, orchitis and epididymitis have been mentioned as male accessory gland infection by the WHO [1]. However, concrete data are lacking to confirm a negative influence of these diseases on sperm quality.

Urethritis and prostatitis are not always associated with male sub- or infertility [4]. In many cases, basic ejaculate analysis does not reveal a link between accessory sex gland infection and impaired sperm characteristics. Furthermore, antibiotic treatment often only eradicates micro-organisms; it has no positive effect on inflammatory alterations and/or cannot reverse functional deficits and anatomical dysfunctions.

As the aetiology of acute urethritis is unknown in most cases at the time of diagnosis, empiric therapy is suggested, with one single dose of a fluoroquinolone, followed by a 2-week regimen of doxycycline. Treatment is effective both for gonococcal and (co-existing) chlamydial/ureaplasmal infections. Only antibiotic therapy of (chronic) bacterial Prostatitis has proved to be efficacious in providing symptomatic relief, eradication of micro-organisms and a decrease in cellular and humoral inflammatory parameters in urogenital secretions [38].

Although antibiotic procedures for male accessory gland infection may provide improvement in sperm quality, therapy does not always enhance the probability of conception.

13. Idiopathic male infertility

Many men presenting with infertility are found to have idiopathic oligo-astheno-teratozoospermia (OAT). No demonstrable cause of male infertility, except for OAT, is found in 40–75% of infertile men [1]. The unexplained forms of male infertility may be caused by several factors, such as chronic stress, endocrine disruption due to environmental pollution, reactive oxygen species and genetic abnormalities.

14. Treatment

14.1. Counselling

Sometimes certain 'lifestyle' factors may be responsible for poor semen quality: for example obesity, alcohol abuse, use of anabolic steroids, extreme sports (marathon training, excessive strength sports), and

increase in scrotal temperature through thermal underwear, sauna or hot tub use or occupational exposure to heat sources. A considerable number of drugs can affect the spermatogenesis.

14.2. Medical (hormonal) treatment

There is no evidence that empiracle hormonal therapies, such as human menopausal gonadotrophin (HMG)/human chorionic gonadotrophin (HCG), androgen, anti-oestrogens (clomiphene and tamoxifen), prolactin inhibitors (bromocriptine) and steroids, improve pregnancy rates in men with idiopathic OAT. However, some primarily endocrinological pathologies can be treated medically [39].

- Low testosterone - testosterone substitution is indicated; substitution exceeding normal physiological values has a negative effect on the spermatogenesis
- Hypogonadotrophic hypogonadism - pulsatile GnRH, i.v. or sc; the usual starting dose is 5 mg, increased if necessary to 10–20 mg, every 90 minutes. Alternatively, HCG 1500 IE and HMG 150 IE (FSH) i.m. twice weekly can be applied.
- Hyperprolactinaemia - dopamine agonists

In patients with sperm auto-antibodies, corticosteroids are not recommended because of serious side effects and unproven lack of efficacy.

A wide variety of empiric drug approaches have been performed (Table 3). The scientific evidence for empirical approaches is low. Criteria for the analysis of all therapeutic trials have been re-evaluated. There is consensus that only randomised-controlled trials, with 'pregnancy' as the outcome parameter, can be accepted for efficacy analysis. Use of recombinant human follicle-stimulating hormone in patients with idiopathic oligozoospermia with normal FSH and inhibin B may be a debatable choice for the future to improve spermatogenesis. Further studies are necessary.

Tamoxifen and testosterone undeconate appear to increases the natural conception rate in a selection of men with idiopathic oligozoospermia [40].

14.3. Surgical treatment

14.3.1. Varicocele

A range of surgical and radiological techniques can be used to treat varicocele. Successful treatment will lead to a significant improvement in semen quality in at least 44% of men treated.

14.3.2. Microsurgery/epididymovasostomy

Only urologists with experience in microsurgery should undertake this procedure [41]. Considering its limited effect on pregnancy rates (20–30%), it is advisable to combine epididymovasostomy with microsurgical epididymal sperm aspiration (MESA), and cryopreserve the harvested spermatozoa for ICSI.

The indications for epididymovasostomy include congenital and acquired obstructions at the level of the epididymis, in the presence of a normal spermatogenesis (testicular biopsy).

Table 3
Empiric therapy of idiopathic oligoasthenoteratozoospermia (OAT)[‡]

Therapeutic approaches	EAU recommendations
Hormonal	
GnRH	Contradictory results. Not controlled trials. Not recommended
HCG/hMG	Lack of efficacy. Not recommended
FSH	Lack of efficacy. Further trials needed
Androgens	Lack of efficacy. Not recommended
Anti-oestrogens (clomiphene citrate, tamoxifen-testosterone undeconate)	Potentially effective. Use must be counterbalanced to possible side-effects
Non-hormonal	
Kinin-enhancing drugs	Unproven efficacy. Use in clinical trials only
Bromocriptine	Lack of efficacy. Not recommended
Antioxidants	May benefit selected patients. Use in clinical trials only
Mast cell blockers	Some efficacy shown. Further evaluation needed. Use in clinical trials only
Alpha-blockers	Lack of efficacy. Not recommended
Systemic corticosteroids	Lack of efficacy. Patients with high levels of antisperm antibodies should enter an ART program
Magnesium supplementation	Unproven efficacy. Not recommended

FSH = follicle-stimulating hormone; GnRH = gonadotrophin-releasing hormone; hCG = human chorionic gonadotrophin; hMG = human menopausal gonadotrophin; ICSI = intracytoplasmic sperm injection; IVF = in-vitro fertilization.
[‡] Also based in parts on the recommendations of the "Infertility Guidelines Group" of the Royal College of Obstetricians and Gynaecologists, London 1998.

14.3.3. Vasovasostomy

Vasovasostomy can be performed either macroscopically or microscopically, though the latter is more effective in improving pregnancy rates. The likelihood of initiating pregnancy is inversely proportional to the obstruction interval and becomes less than 50% after 8 years. Important prognostic factors are the development of antisperm antibodies, the quality of the semen and the partner's age. In approximately 20% of men who have undergone a vasovasostomy, sperm quality deteriorates to the level of azoospermia or extreme oligospermia within 1 year. Poor sperm quality and autoantibodies frequently prevent a spontaneous pregnancy and assisted reproduction is indicated [12].

14.3.4. MESA

MESA in combination with ICSI is indicated when reconstruction (vasovasostomy, epididymovasostomy) cannot be performed or is unsuccessful. An alternative would be percutaneous aspiration of sperm from the caput epididymis (PESA). If a MESA or PESA procedure does not produce spermatozoa or very low numbers of motile spermatozoa, a testicular biopsy can be performed with testicular sperm extraction (TESE) to be used for ICSI [42].

14.3.5. Transurethral incision of ejaculatory ducts or midline prostatic cysts.

Distal obstructions of the genital tract are commonly caused by infections of the prostatic urethra and the accessory glands, or by a cyst in the midline of the prostate. Treatment of the obstruction by transurethral incision of the cyst or the ejaculatory ducts may lead to an increase in semen quality and, occasionally, spontaneous pregnancy [16].

For further information consult the extensive guidelines on Male Infertility (ISBN 90-806179-8-9), available to all members of the European Association of Urology at their website - www.uroweb.org.

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