Table 2.4: Major aspect of history taking in male infertility

1. Infertility history
• Age of partner, time attempting to conceive. • Contraceptive method and duration. • Previous pregnancies (same partner/other partner). • Previous treatment. • Treatment evaluation of female partner.
2. Sexual history
• Potency, libido, lubricant use • Ejaculation, timed intercourse
3. Childhood and development cryptorchidism, hernia, testicular trauma/torsion/infection (mumps)
• Sexual development, puberty onset
4. Personal history
• Systemic diseases (diabetes, cirrhosis, hypertension), sexually transmitted diseases, tuberculosis, viral infections
5. Previous surgeries
• Orchidopexy, herniorrhaphy, orchiectomy (testicular cancer, torsion). • Retroperitoneal and pelvic surgery. • Other inguinal, scrotal and perineal surgery. • Bariatric surgery, bladder neck surgery, transurethral resection of prostrate.
6. Gonadotoxin exposure
• Pesticides, alcohol, cocaine, marijuana abuse-medication (chemotherapy agents, cimetidine, sulfasalazine, finestaride, calcium blockers, alpha blockers). • Organic solvents, heavy metals. • Anabolic steroids, tobacco use. • High temperature, electromagnetic energy. • Radiation.
7. Family history
• Cystic Fibrosis, endocrine disease and infertility in the family
8. Current health status
• Respiratory infection • Anosmia • Galactorrhea • Visual disturbances • Obesity

Table 2.5: World Health Organization criteria for a normal semen analysis

Lower limits of the accepted reference values for semen analysis	
Parameter	Reference values
On at least two occasions	
Ejaculate volume	1.5 ml
pH	7.2
Sperm concentration	15×10^6 spermatozoa/ml
Total sperm number	39×10^6 spermatozoa/ml
Percentage motility	40%
Forward progression	32%
Normal morphology	4%
Sperm agglutination	Absent
Viscosity	<2 cm thread after liquefication

Note: Data from WHO 2010[10] Practice Committee. Evaluation of the infertility male. Fertil Steri 2015.

2. Men with impaired sexual function
3. Men with other clinical findings that suggest a specific endocrinopathy.

There is no consensus on whether all infertile men should undergo a routine endocrine evaluation. Measurement of serum FSH and total testosterone (T) concentrations should be considered where indicated, and a more extensive evaluation should be considered if total T level is low (<300 ng/dL), which includes total testosterone levels, LH levels, FSH levels and prolactin with TSH (Table 2.6).

ULTRASOUND

Transrectal Ultrasound

Seminal vesicles are normally less than 1.5 cm in AP diameter. Transrectal ultrasonography can identify the presence of dilated seminal vesicles/ejaculatory ducts and/or midline prostrate cystic structures, which are indicative of complete or partial ejaculatory duct obstruction. In patients with complete ejaculatory duct obstruction, low volume, fructose negative, acidic and azoospermic features are often observed. Patients with CBAVD have

Because this chapter has limited space it is wise to outline only areas where medical management works or is reported to work and to open readers' minds to the principles involved in arriving at diagnosis and subsequent therapy and its results. Knowing the difference between curable/treatable and irreversible pathologic aetiologies saves time and costs for the infertile couple. The place of empirical treatment will be touched upon when necessary. Some drug usage can be off-label.[7] So issues involved in off-label drug usage are to be understood by the andrologist and the client should be counselled accordingly.

There are some obvious areas where medical interventions may help. Such examples are:

1. Hypogonadotrophic Hypogonadism (HH) especially post-pubertal HH
2. Excessive Reactive Oxygen species which cause sperm plasma membrane damage and sperm DNA fragmentation
3. Factors inducing apoptotic pathways in germ cells and spermatozoa
4. Androgen deficiency states
5. Male accessory gland infections (MAGI)
6. Erectile and ejaculation dysfunctions
7. Pre- and Post-chemo radiation to brain, abdomen, scrotal malignancies
8. Obesity and metabolic syndrome
9. Varicocele
10. Non-Obstructive Azoospermia (NOA)
11. Idiopathic oligo asthenospermia
12. Hyperprolactinemia, etc.

The easiest to understand but uncommon to find in practice is the treatable cause, viz. HH or hypogonadotrophic hypogonadism where S. FSH, S. LH, S. Testosterone are low or near lower normal level and semen parameters are deranged with symptoms of hypoandrogenism. And while the aetiology of HH may be treatable or not treatable, semen production and androgen output can improve with medications when testicular size is post-pubertal.[8] HH can be secondary to pathology involving hypothalamus and pituitary like Kallman's syndrome, Prader-Willi syndrome, Prolactinomas, other tumors, infections or sometimes it is idiopathic. In cases of HH, clinicians have to be alert about any history of exogenous testosterone or anabolic steroid intake by patient, with or without prescription, e.g. in body builders and athletes. In these cases stopping androgens and waiting to recover from azoospermia or oligospermia is advisable.[9] To hasten the recovery of spermatogenesis inj. hCG or oral clomiphene citrate (CC) have been used. In classic HH one needs to replace the gonadotropins which are low.[10] Main constraints are monetary as the treatment with HCG, HMG, rFSH can take months or years for restoring spermatogenesis and sperms to appear in the ejaculate. In some cases it may not resume if testes are small or undescended (cryptorchidism). In HH usually the androgen levels rise quicker than spermatogenesis resumption. One has to guard against supraphysiologic testosterone levels during HCG therapy. If HCG alone in doses 2500 units twice a week is working, more costly HMG injections can be avoided. But after a few months of hCG alone, if no sperms appear,[11] HMG can be added in doses of 150 units IM on alternate days (3 times per week) in addition to HCG. Married life should continue and pregnancies do occur with sperm counts as low as 5 million/mL in young couples with regular coital activity and healthy wife.

Is there a role for gonadotropins when testes themselves are not 'normal' like in hypergonadotrophic situations (raised FSH)? It is noted that in hypergonadotrophic situations,[12] 50% cases show hypoandrogenism (T <300 ng/dL). Any exogenous testosterone supplement simply suppresses the spermatogenic potential and acts rather like male contraceptive. So can we give hCG or HMG or clomiphene in these hypoandrogenic cases? Yes, it may help by correcting hypoandrogenism and more importantly improving the intra-testicular testosterone (ITT) by stimulating the Leydig cells. Normally ITT

tension free direct anastomosis impossible. The epididymis can be dissected free from the testis, being attached only by the head and brought up to the distal end of the vas, thus making anastomosis possible without traction.

It is important to note that the mean time between vasectomy reversal and conception is more than twelve months and, more important, that the fertility rate of the reversal group is the same as in the normal control group.

VASOEPIDIDYMOSTOMY

If obstruction is present at the level of the epididymis in the presence of a normal vas, the first-choice therapy is vasoepididymostomy. Vasoepididymostomy shall also be performed in case of unsuccessful vasectomy (Fig. 7.7A and B).

Technique consists of lateral opening of epididymis proximal to the level of obstruction and isolating a single tubule which, later on, is incised, but not transacted, and anastomosed side to end.

The results of this procedure show a patency rate of 85% with a pregnancy rate of only 44%. Vasoepididymostomy has a higher pregnancy rate than IVF with ICSI and should be preferred in any case of obstructive sterility at the epididymal level.

ORCHIOPEXY IN ADULTS

This is well known that cryptorchidism is associated with a high incidence of infertility even when unilateral. Spermatogenesis is delicately temperature sensitive. It will also preserve testicular hormonal function. The technique of orchiopexy in adults is similar to that employed for children. Even with a normal contralateral testis, orchiopexy is worthwhile to bring down a unilateral undescended testis to, if possible, a scrotal location where it can be examined. Leydig cell function in undescended testis can be retained. Orchiopexy in adults with bilateral undescended testes can induce spermatogenesis and allow pregnancy. Even a solitary cryptorchid testis, when properly placed in the scrotum, can provide enough testosterone to obviate the need for hormone replacement. When orchiopexy is performed, regular self-examination and yearly sonography are mandatory in adults.

The fate of persistently retractile testis in adults is unknown. These testes may suffer from impaired temperature regulation and impaired spermatogenesis. Scrotal orchiopexy can improve the semen quality and fertility of some of these men. Some men have ectopic testis, in which one testis is behind the other almost in the perineum. This also elevates testis temperature. When scrotal orchiopexy

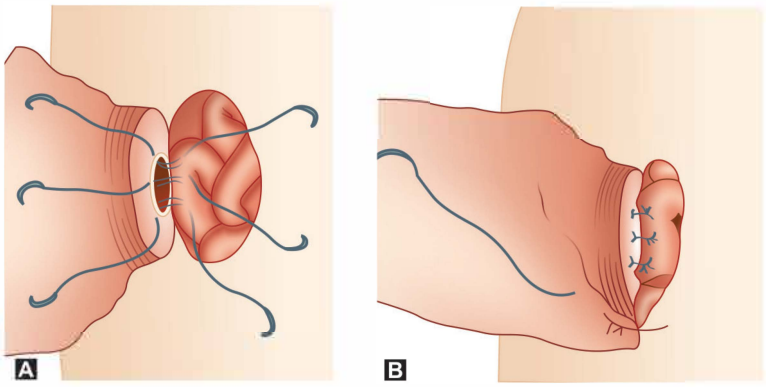

Fig. 7.7 A and B: The outer muscularis and adventitia of the vas are then approximated to the cut edge of the epididymal tunica with 6 to 10 additional interrupted sutures of 9–0 nylon double-armed with 100-micron diameter needles.

fragments which should be extemporaneously examined in order to find a sperm producing zone in the testis. Percutaneous biopsy has not given good results. More recently it has been suggested to press a slide to the opened testis and look for spermatozoa, under microscope. This technique avoids useless damaging of already small testis. One single spermatozoa can also be picked up from a small testis and can be used for ICSI. This procedure has a comparable fertilization and pregnancy rate.

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