Introduction
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The Saga of the Vanishing sperm published in the journal Biologist (1996) by Jenny Hall suggested that a world-wide decline in semen quality have received much attention, but have also been the cause of much controversy. However a decline in the health of the male reproductive system seems to be true. **Is the human testis at risk or is the evidence flawed?**

Infertility is commonly defined as the failure of conception after at least 12 months of unprotected intercourse (Rowe et al., 1993). It is clear that it is a common problem affecting young couples, and equally clear that it results in considerable distress for those couples affected. The feelings experienced by infertile couples encompass anger, depression, anguish, denial, guilt, shame, inadequacy, shock, isolation and embarrassment (Wright et al., 1991).

Infertility is a problem that affects men and women everywhere in the world. Although estimates of its prevalence are not very accurate and vary from region to region, according to WHO about 8% of couples experience some form of infertility problem during their reproductive lives (WHO 1993). When extrapolated to
the global population this means that 50 to 80 million people probably have a problem with fertility a condition that causes personal suffering and disruption to family life. It is estimated that there are about 2 million new infertile couples per year and the number are increasing. This should be compared to an estimated 5.9 million new cancer cases per year and 100 million new clinical malaria cases, but nevertheless represents a significant burden on national health resources.

The changes in demographic patterns over the past 50 years in the developed countries, and in particular over the past 20 years in some developing countries, make infertility a more urgent problem, the awareness of which is rapidly increasing. Several studies conducted over the past 20 years have suggested a worldwide decline in sperm quality, arousing concern about male infertility in the future. The possibility of a decline in the quality of semen is not the only indicator that the human testis is at risk. Recent increase in the incidence of congenital malformation of the male reproductive tract, and in the incidence of testicular cancer give genuine cause for concern. Cancer of the testis mainly affects young men and is clearly associated with a high rate of other abnormalities, such as undescended testes and dysgenic testes, but the cause remain unknown. The incidence of hypospadius and
undescended testes are reported to be on the increase in the
general population. The evidence of declining semen quality should
be considered in association with those other trends in male
reproductive disorders.

Such observation have resulted in much speculation about
the cause of the decline in male reproductive function. It has been
suggested that environmental factors, acting in foetal and early
neonatal life may adversely affect subsequent spermatogenic
capacity. If this is the case, then the semen quality of an individual
must be determined very early in life, possibly before birth, and not
related to the time of one ejaculation. Oestrogens or compounds
with oestrogen-like activity, taken by pregnant woman have
suggested to affect the testicular function of male offspring
adversely. Many manmade toxins in the environment may act as
oestrogens and these pose a theoretical risk to the male foetus if
ingested by the mother before or during pregnancy.

I. ENVIRONMENTAL FACTORS

The World Health Organization estimates that 8% of couples
or 50 to 80 million people world wide, experience some form of
infertility. In industrialized nations, approximately 10-15% of couples
experience either primary or secondary infertility. Investigators
hypothesize that environmental factors have the potential to alter
male and female reproductive tissues and thus affect the ability of couples to conceive healthy offspring. Individuals may be exposed to these chemicals in the work place and through soil, air, water and food.

**Cigarette Smoke**

To date, most studies of environmental exposure and reproductive function have focused on the association between cigarette smoking and infertility. [Sofikitis et al., 1995; Vine, 1996; Zavos et al., 1998]. Individuals may be exposed to the toxicants in cigarette smoke through both active and passive smoking. At least 43 carcinogens and more than 300 polycyclic aromatic hydrocarbons have been identified among the 4,000 chemical constituents of cigarette smoke. In addition, tobacco smoke contains several other known toxicants, such as methylisocyanate, benzene, acetone, ammonia, arsenic, butane, carbonmonoxide, cyanide, dichlorodiphenyltrichloromethane (DDT), formaldehyde, lead, methanol, polonium 210 and naphthalene.

Chemical agents or mutagens may affect male reproduction by direct effect on the testes and their ability to produce sperm through the process of spermatogenesis. Those mechanisms may involve the hormonal control of spermatogenesis or directly affect the germ and sertoli cells within the seminiferous tubules. Various
investigators have shown that smoking is associated with detrimental effect on sperm concentration, sperm motility and the percentage of morphologically normal spermatozoa.

**Alcohol**

Only a few studies have evaluated the effect of alcohol consumption on male fertility. Heavy alcohol use in men is associated with testicular atrophy. (Close et al., 1990).

**Occupational Exposures**

Although most studies of environmental exposures and infertility have focused on cigarette smoke, a few studies indicate that occupational exposures may also be associated with an increased risk for infertility.

**Heat Exposure**

Heat exposure seems to have a deleterious effect on male fertility and must be considered a significant risk factor for male infertility. In men under normal healthy environmental conditions the testicular thermoregulation system is certainly able to maintain the normal scrotal hypothermy. In men repeatedly subjected to abnormal situations (such as drivers or workers exposed to high temperature) and perhaps also in men with impaired arterio-venous testicular systems, there may be chronic-thermo-dysregulation.
which may, in time, result in substantial changes in sperm characteristics such as motility and morphology. Radiant heat also can affect testicular function such as decreased or altered sperm production. (Thonneau et al., 1996; Figa-Talamanca et al., 1996; Bonde, 1992). This exposure is potentially encountered by welders, fire fighters, ceramic and foundry workers and food preparers. Several studies have established that heat can affect the seminiferous epithelium and subsequently the quality of the sperm.

**Organic Solvents**

Organic Solvents are thought to affect fertility in men employed as painters. Ethylene glycol ethers, which have wide spread applications in paints, varnishes, thinners and printing inks have been linked to an increased prevalence of oligospermia and azoospermia. (Veulemans et al., 1993). Carbondisulfide mainly used in the manufacture of viscose rayon fibres and production of carbon tetrachloride, has been linked to decreased libido and loss of potency.

**Heavy Metals**

Heavy metals such as manganese and lead present in a number of industries may also affect male fertility. Occupational exposure to manganese may occur in steel manufacturing, mining and dry - alkaline battery production plants. Notably manganese
exposure has been associated with impotence, decreased libido, reduced secretion of 17-ketosteroids, and reduced fertility among male workers. Lead exposure has been associated with oligospermia, increased percentage of abnormal sperm, decreased function of the prostate and seminal vesicles and subfertility. (Tas et al., 1996).

**Environmental Contaminants**

In one of the studies made by Irvine et al. (1996) in United Kingdom, over a period of 11 years had observed a deterioration in semen quality, based on studies in 577 men who visited laboratories in Scotland. They estimated 2.1% decrease in the sperm concentration per year. A similar observation is also made by Auger et al. (1995) who found annual decrease of 2.6% in the sperm count of fertile men, 0.3% in motility and 0.7% in normal sperm morphology. Studies made by Younglai et al. (1998) also observed an annual decrease of 1.44% in sperm density in men attending 11 fertility centers across Canada over a period of 12 years from 1984. In addition to decrease in semen quality evidences were also available for increasing abnormalities associated with male genitourinary tract. These abnormalities included testicular cancer, hypospadius and cryptorchidism.
A wide range of environmental pollutants are known to threaten male reproductive ability, most of them are oestrogenic compounds, are found in detergents and plastics. New research suggests that the insecticide dicophane, (DDT) may be a culprit. Although DDT has been banned or restricted for two decades in the developed world, it is still in widespread use in many malarial zones, from where it can be exported via food or the atmosphere. A persistent metabolite of DDT P, P'-dichlorodiphenyltrichloro ethylene (P, P'-DDE) has the traits of a potent androgen (Kelce et al., 1995). Additionally dicophane is known to attack androgen receptors. The possible consequences of inhibition of androgen receptor binding and of subsequent transcriptional activity are similar to the dangers posed by environmental oestrogens.

Environmental agents can adversely affect germ cell development at many different stages from proliferating spermatogonia to mature spermatozoa. Possible different toxic effects include cell death, sublethal damage or genetic change. Cell dying within the epithelium may do so by necrosis or apoptosis. Recent evidence suggest that apoptosis is the major mechanism of action of some testicular toxins. Adverse physiological conditions such as gonadotropins deprivation, nonlethal germ cell damage will
be repaired or leave permanent effect on structure and function of mature spermatozoa, including possibility of genetic defects.

Occupational exposure to sex steroids such as oestrogens, can exert negative biofeedback on FSH secretion and result in decreased sperm production, sexual dysfunction, gynecomastia and hypogonadotropic hypogonadism, and with a potential to cryptorchidism and testicular cancer. Prenatal exposure to oestrogens could potentially inhibit foetal gonadotropin secretion and reduce sertoli cell proliferation. Several compounds are known to have antiandrogenic activity such as 9,10 Dihydrophenanthrene, Linuron, Vincozolin, DDT/DDE, and flutamide (Chang and Liao, 1987; Kelce et al., 1995). Direct testicular toxicity may potentially affect separate cell types within testis with subsequent effect on spermatogenesis and steroidogenesis in general. No specific human sertoli cell toxins have yet been established. Ionization radiation and alkylating agents such as nitrogen mustard, vincristine, procarbazine, prednisone were found to have most profound toxicity on human germ cells. The most sensitive cells are spermatogonia. Non proliferating spermatogonia if destructed, leads to irreversible spermatogenic damage, while proliferating spermatogonia can be replaced from stem cell reserves.
Environmental oestrogens constitute a class of chemical agents that structurally resemble 17β oestradiol and which possess oestrogenic activity. The environmental oestrogens can be divided into two main categories. 1. phyto-oestrogens, which are plant derived oestrogenic molecules and have co-evolved with humans 2.xeno-estrogens, which are chemically synthesized manmade molecules such as phenols, pesticides, plastics and polychlorinated biphenyls released into the environment (Toppari et al., 1996). Because oestrogens play a key role in regulating several reproductive processes, it is feasible that exposure of humans to environmental oestrogens in an modern environment may influence the reproductive system. In this regard, ample evidence has accumulated during the past decade to support the notion that environmental oestrogens may cause reproductive disorders. However, less clear (i) their pathophysiological effects on the reproductive process (ii) whether the influence of phyto-oestrogens and xeno-oestrogens on the reproductive system are similar or different and (iii) how some environmental oestrogens induce oestrogenic effects, whereas some are anti-oestrogenic.

II. IATROGENIC FACTORS

Infertility could be a result of complications from certain operations such as prostatectomy, and from childhood operations
on the male genital tract. Repair of inguinal hernias or hydrocoeleles, and other genital or inguinal operations may damage the vasa deferentia leading to their partial or complete blockage and possibility of antisperm antibody formation.

**Herniorrhaphy**

Bilateral inguinal herniorrhaphy particularly in infancy carries a high risk for causing iatrogenic obstruction of the vas deferens. Such patients generally report infertility in young adulthood (Britton, 1975).

**Radiation**

Germ cells are particularly sensitive to radiation, while Leydig cells are relatively resistant. At single exposure below 600 rads, the germcell damage is reversible, but above this damage is likely to be permanent.

**Chemotherapy**

Anti neoplastic agents are unable to differentiate between normal and abnormal tissue and exert their cyto-toxic effect on the exquisitely on sensitive germinal epithelium of testes. Biopsies of these testes demonstrate complete early spermatogenic arrest often with germinal aplasia, with normal appearing Leydig and sertoli cells. Hormonal studies have shown however, that Leydig cell
function is disturbed following chemotherapy, with reduced free testosterone levels detected in the serum. In addition, decreased libido and erectile dysfunction have been observed in a large number of chemotherapy patients.

Drugs

Production of spermatozoa in the testes may be depressed by drugs used for the treatment of certain ailments. Such drugs include hormones, sulphasalazine, cimetidine, nitrogurantoin, niridazole, spirinolactone and colchicira (Rowe et al., 1993).

III. ACQUIRED DEFECTS OF THE TESTIS, PROSTATE AND SPERM

Orchitis

Approximately 15-20% of adult men who contract mumps (epidemic parotiditis) develop orchitis which is commonly unilateral. Bilateral involvement occurs in only 10% of affected men. Testicular atrophy can develop within 1 to 6 months, but it may take years. With the advent of mumps vaccine the incidence of mumps and associated orchitis is becoming increasingly rare. Less than one third of men with bilateral orchitis recover normal semen parameters. Acute viral orchitis seen in 30% of post pubertal males with mumps parotitis causes permanent injury to the geminal epithelium of the seminiferous tubules.
Syphilis

This can similarly affect the testicles causing interstitial edema and endarteritis, often resulting in seminiferous tubules damage and subsequent infertility.

Epididymal infections (epididymitis)

Epididymal infections may result in ductal obstruction and post testicular azoospermia. Severe epididymal infection may also involve the testicle with a resultant orchitis leading to testicular atrophy. Azoospermia will occur if the process is severe and bilateral. Obstruction is suggested by induration and nodularity of the epididymis. Causative organism includes Mycobacterium tuberculosis, Neisseria gonorrhoea, and the more common gram-negative pathogens of the urinary tract. Gram-positive organisms are rarely responsible for epididymal infections. (Hendry et al., 1983).

Immunologic causes: Antisperm antibodies

Many studies have sought to determine whether antibodies directed against sperm antigens play a role in male factor infertility. Sperm antibodies are proposed to impede sperm transport in the female genital tract and to interfere with sperm-egg interaction. However all antibodies detected are not known to influence sperm motility or pregnancy rate. Most current investigation focus on
immunoglobulin (IgA) antibodies in semen or bound to sperm membranes. Estimates of the prevalence of sperm antibodies among infertile men have ranged from 3 to 20%. The cause of immune infertility is unknown, one hypothesis is that infection and inflammation of the testes and epididymis render sperm antigens immunogenic.

Wolff and Anderson (1988) made quantitative analysis of the leucocytes in the semen for immunological characterization. They observed that the leucocytes contained 50-60% granulocytes, 20-30% macrophages, 2-5% T Lymphocytes, but B-Lymphocytes were rarely present in the semen. A comparative study of the seminal leucocytes of fertile and infertile patients made by Auraux and others (1985) revealed a higher percentage of granulocytes in the semen of sub fertile men.

**Varicocele**

Varicocele is a vascular abnormality of the scrotum that is defined as dilated veins of the pampiniform plexus (Tulloch et al., 1952). The exact pathophysiology of varicocele is not known. Varicose are thought to impair normal testicular function by elevating scrotal temperature via reflux of warm abdominal blood through incompetent valves of the veins (Moore and Quick, 1923; Lerchl et al., 1993). Varicocele are currently the most common
abnormality identified in men being evaluated for infertility (Sigman and Howard, 1998). Varicoceles are normally diagnosed by physical examination through palpation of the spermatic cord before and during a valsalva manoeuvre with the patient in a standing position. The diagnosis is based upon the clinician's subjective impression of either venous dilation or reflex of blood. These vascular lesions have been arbitrarily divided into three grades based upon physical finding, large varicoceles are visible, medium varicoceles are palpable and small varicoceles are only palpable during a valsalva manoeuvre. The vast majority of physicians who manage male infertility patients believe that repair of varicocele will improve fertility (WHO 1992).

IV. DEVELOPMENTAL AND STRUCTURAL DEFECTS OF THE TESTIS AND SPERM

Cryptorchidism

Cryptorchidism was found in 6.4% of infertile men. Although patients with sex chromosomal abnormalities, hypogonadotropic hypogonadism, or androgen resistance may also have cryptorchidism, there is no clear explanation why the testis fails to descend into the scrotum. The extend of damage to the seminiferous tubules with cryptorchidism is quite variable. A high rate of infertility is found among men with bilateral cryptorchidism.
Men with one undescended testis are much more likely to be fertile. (Chiwers et al., 1986)

**Genetic causes**

The causes of male infertility still remain largely unknown, however an increasing list of genetic defects leads to reproductive failure. Survey showed in somatic karyotypes a higher incidence of numerical and structural chromosomal aberrations in infertile and sub fertile men than in the neonatal population that is 50 times more 47XXY karyotypes, 4 times more 47XYY karyotypes, 60 times more 46XX karyotype, 20 times more 46X, der Y karyotype were found. Robertsonian translocation, reciprocal translocation, inversions and additional marker chromosomes were respectively 8.5, 5.8 and 3 times more frequent in subfertile men than in new borns. (Chandley et al., 1979; Krauszo and Forti 2000). Microdeletion of the q11 region of the Y chromosome related to the dysfunction of deleted in azoospermia (DAZ) and RNA binding motif genes (RBM genes) have been reported in azoospermic as well as in oligoasthenoteratozoospermic men (Silber et al., 1998). Other mutations on the X chromosome such as those present in Kallman gene, the androgen-receptor gene and mutation in autosomal gene such as those causing myotonic dystrophy and CFTR gene are known to interfere with normal spermatogenesis (Huynh et al.,
Certainly more genes are involved, some acting on their own, some acting in collaboration with other genes and also interacting with external factors. Finally mitochondrial DNA may also play a role in male infertility.

**Sertolicell only syndrome**

Sertoli cell only syndrome may have several causes including congenital absence of germ cells, genetic defects, and androgen resistance. Clinical findings include azoospermia in association with normal virilization, testis of normal consistency but slightly smaller size, and no gynaecomastia. Plasma testosterone and serum LH levels are normal but plasma FSH is usually elevated. With other testicular disorders such as mumps, cryptorchidism and damage due to radiation or toxins. The seminiferous tubules may also contain sertoli cells, but in these men testes are very small, the histologic pattern is not uniform, and severe sclerosis and hyalinization are prominent features. (De Kretser et al., 1972).

**Spermatogenic arrest**

This is observed in 4-30% of azoospermic patients. Interruption of germ cell differentiation resulting in oligospermia (partial arrest) or azoospermia (complete arrest). It generally occurs in normal patients with normal testicular volume and gonadotropin levels. Most cases are due to genetic abnormalities occurring in the
prophase of the first meiotic division. Acquired cases can be due to hormonal, thermic or toxic factors. (Martin Du Pan, 1997).

**Anomalies of sperm structure (Immotile cilia syndrome or Kartagener syndrome)**

Immotile cilia syndrome is a rare disorder caused by a defect in the axoneme of the cilia in the respiratory tract and sperm tail. (Afzelius, 1975) Men with this disorder produce sperm that are immotile but normal in density and morphology. These men are uniformly sterile. Additional clinical findings include situs inversus, chronic sinusitis and bronchiectasis.

**Globozoospermia**

It is an uncommon condition probably occurring in 0.1% of all andrology patients, and it is characterized by the complete absence of the acrosome vesicle and the disorganisation of the mid piece and the tail. These abnormalities are found in all sperms in the patient ejaculate and lead to the inability to bind the human oocyte zona pellucida and to produce the sperm oocyte fusion. The hereditary background to this condition has not yet been established (Carrell et al., 1999). A polygenic and polymorphic mode, a monogenic mode, a dominant inheritance and a homozygous autosomal gene defect have also been suggested (Trokoudes et al., 1995).
V. HORMONAL CAUSES AND ANDROGEN RESISTANCE

Lack of gonadotropin stimulation of the testes may be the cause of infertility in some men. This can be caused by a broad range of disorders that prevent the secretion of FSH and LH by the pituitary. At times the problem may be higher up, affecting the secretion of gonadotropin - releasing hormone by the hypothalamus. The disorders are either congenital or acquired. The incidence of primary endocrine defects in infertile men is less than 2%.

**Hypogonadotropic hypogonadism**

Testicular dysfunction in men with hypogonadotropic hypogonadism results from inadequate gonadotropin stimulation of the testis. Abnormalities within the pituitary gland or a disturbance in gonadotropin releasing hormone (GnRH) secretion by the hypothalamus can result in gonadotropin deficiency. Serum levels of testosterone are reduced and LH levels are low or in the low normal range. (Lieblich et al., 1982).

**Hyper prolactinaemia**

Prolactin has a significant effect on male reproductive function. By acting on the hypothalamo-pituitary-gonadal axis, prolactin affects gonadal function, seminal plasma, accessory reproductive gland function and even sexual behaviour. Hyperprolactemia affects the pulsatile release of LH by the anterior
pituitary which in turn leads to decreased testosterone synthesis. Elevated prolactin levels also inhibits 5 alpha reductase activity and adversely affects spermatogenesis, apart from causing structural changes in the testis. (Katovich et al., 1985).

VI. SYSTEMIC DISEASES

Testicular function is frequently abnormal in men with acute and chronic systemic illness. (Semple et al., 1987) Weight loss, malnutrition and stress contribute to these changes. Chronic liver disease is often associated with infertility sexual dysfunction and reduced androgenisation (Boyden et al., 1983). Serum FSH and LH levels are often increased but they may be normal. Gonadal dysfunction is common in men with chronic renal failure, reduction in libido, sexual dysfunction, reduced muscle mass and impaired androgenisation all contribute to a poor quality of life for these patients. Testosterone levels are often low and LH and FSH levels are either normal or elevated. Prolactin level may increase. In men serum testosterone levels decline transiently after intense exercise and during fasting because these stress decrease gonadotropin secretion libido may also decline.
VII. IMPAIRMENT OF SPERM TRANSPORT (OBSTRUCTIVE AZOOSPERMIA)

Genital duct obstruction is found in 5-7% of infertile patients. Obstruction may occur at any level of the genital tract. It can be congenital or acquired or secondary due to infection, or vasectomy. Most of the patients with azoospermia maintain normal size of testis and normal gonadotropin levels. In congenital absence of the vas deferans there is usually an associated absence of seminal vesicles and ampulla. The semen volume is low, acidic and fructose negative. (Jequier et al., 1985).

VIII. PROBLEMS OF EJACULATION

Retrograde ejaculation

The normal ejaculation process is dependent upon the anatomical and functional integrity of the urinary bladder neck and posterior urethra. It involves emission of semen into the urethra, closure of the bladder neck and propulsion of the content from the urethra. Any interference, anatomical, traumatic, neurogenic or drug-induced, with this integrity may result in abnormal function of the internal sphincter of the urethra, and cause retrograde ejaculation (RE). RE was first described by De Albuquerque (1939). He termed this phenomenon 'retropermia' a nomenclature which was rapidly dispensed with. Since 1950, it has been specifically
referred to as retrograde ejaculation. Any alteration in the neurological stimulus involving semen emission and bladder neck competence may affect a man's normal anterograde ejaculation, leading to RE. Traumatic or surgical injury to the sympathetic nerves may also cause this. In addition to ejaculatory dysfunction due to neurogenic origin, RE can be of myogenic or neuromuscular origin, mainly after surgical procedures in the bladder neck and urethra. Other aetiological factors among urological patients are neurogenic, i.e., after spinal cord injury (Dahlberg and Hovatta, 1989) and diabetic neuropathy (Ellenberg and Weber, 1966) or congenital factors such as meatal stenosis and posterior urethral valve (Strachan et al., 1988). Among infertile patients, the distribution of the aetiologies is rather different, due mainly to the difference in the population age of the patients.

**Anejaculation**

Anejaculation and ejaculatory dysfunction are the terms used to describe the inability of a man to have an ejaculation. (Glander, 1998). It is a relatively uncommon disorder that can occur as a result of spinal cord injury, retroperitoneal lymphnode dissection or other retroperitoneal surgery, diabetic mellitus, transverse myelitis or multiple sclerosis. In addition, the etiology may be psychogenic or idiopathic. The nerves that are responsible for carrying the signal for
injured after spinal trauma resulting in paraplegia or quadriplegia, major bowel or vascular surgery or surgery for testicular cancer.

IX. SEXUAL DYSFUNCTION

Male erectile dysfunction

Male erectile dysfunction defined as “the inability to achieve or maintain erection sufficient for sexual intercourse” - is one of the most common sexual dysfunctions in men (Korenman, 1995). Although erectile dysfunction can be primarily psychogenic in origin, most patients have an organic disorder, commonly with some psychogenic overlay. Some men assume that erectile failure is a natural part of the ageing process and tolerate it; for others it is devastating. Withdrawal from sexual intimacy because of fear of failure can damage relationships and have a profound effect on overall well being for the couple (Krane, 1989). Erectile dysfunction often accompanies chronic illnesses, such as diabetes mellitus, heart disease, hypertension and a variety of neurological diseases. Normal erectile function requires the coordination of psychological, hormonal, neurological and vascular cavernosal factors. Alteration in any one of these factors is sufficient to cause erectile dysfunction. Not uncommonly, a combination of factors is also involved.
INTRAUTERINE INSEMINATION

Intrauterine insemination (IUI) is an assisted conception treatment method that can be used for alleviation of infertility in certain groups of patients. It involves the deposition of washed semen sample in the uterine cavity around the predicted time of ovulation. Usually the woman’s ovaries are also stimulated to produce a few oocytes. These manipulations have certain advantages over the natural situation. Firstly, more sperm are spared from destruction in the vagina. Secondly the distance which sperm have to travel to reach the site of fertilization in the fallopian tube is greatly shortened. Thirdly, more oocytes are available in the fallopian tubes and this increase the chances of at least one of them being fertilized. Finally, the presence of more than one embryo likewise improves the chances of one of them implanting successfully in the uterus. IUI is one of the simpler and less expensive assisted conception treatment methods. (Loy et al.,1990).

Therapeutic insemination has had been in practice for last 200 years. The first therapeutic insemination was performed in London in 1770’s by Dr. John Hunter in a patient whose husband had hypospadius and this resulted in pregnancy. Intrauterine insemination (IUI) is a commonly used treatment for a wide range of infertility problems which are seen in about 15 percent of couples.
The rationale of IUI performed using husband’s spermatoza is to overcome the problems of vaginal acidity, cervical mucus hostility and deposits good number of highly motile and morphologically normal sperms in the uterus near the fundus at the anticipated time of ovulation. In case of oligospermia deposition of sperms directly in the uterine cavity prevents intravaginal and intracervical loss of sperms, resulting in increasing the number of sperms available for fertilization. (Brasch et al., 1994).