FACTORS ASSOCIATED WITH
MALE INFERTILITY
Hear, sat ore, hears dear goddess, hear!
Suspend thy purpose, if thou didst intend
To make this creature fruitful!
Into her womb convey sterility!
Dry up in her the organs of increase;
and from her derogate body never spring
A baby to honour her!

- Shakespeare (King Lear)

Wives alone were treated as responsible for
fruitlessness, since olden times. The enlightenment in
the study of male and female genital systems present us
the causality of infertility as husband, wife or both.
Some of the factors responsible for male infertility are
briefed here (Walsh, 1977).

Endocrine abnormality, including hypothalamus,
pituitary, testis, adrenal or thyroid remained as a cause
for infertility in at least 20% of the patients (Dubin and
Amelar, 1971).

HYPOTHALAMIC DISORDERS:

Kallaman's syndrome

In 1944 when Kallaman described this syndrome many
facts related to it were unknown. Today more details about
this syndrome are known. It is caused by the lack of
secretion and release of gonadotropin releasing hormone
(Gn RH) from hypothalamus. Symptoms are hypogonadotropic
hypogonadism and anosmia. Kallaman described about total loss of smell in these patients, however, more recently it has been recognised that in some patients it is partial. The role of smell in reproduction is well known. In those who lose the sensation of smell, before puberty, maturity is delayed. This lead to the suggestion that pheromone, a chemical substance, which has been shown in animals might also be present in human. Evidences are available to support its existence (Skandhan et al. 1979).

The most common disorder associated with Kallaman's syndrome are cryptorchism, cleft lip or palate and congenital deafness. Rarely patients may be associated with colour blindness, synkinesis, mental retardation, microphallus or short fourth metacarpal.

Histologically, the testes show immature seminiferous tubules. Germinal cells are mostly undifferentiated. But occasionally spermatogonia of early type are seen. Leydig cells are absent.

Plasma levels of LH, FSH and testosterone are in the prepubertal range. Following treatment with HCG, plasma testosterone levels usually rise into the normal range. TSH and ACTH level is normal in these patients. However, in some, lack in GH is reported.

Recently, Mortimer et al. (1974) have treated their patients with Gn RH and in them the sexual maturation was completed and spermatogenesis was also initiated.
Fertile eunuch syndrome

In the patients with fertile eunuch syndrome, spermatogenesis is found to be normal, but Leydig cells are absent. Serum FSH level is normal, whereas LH and testosterone are low. The mechanism by which the spermatogenesis occurs is not clear. It is possible that a small amount of circulating LH is sufficient for synthesis of testosterone and the latter is responsible for spermatogenesis. But, the amount of testosterone is not sufficient to androgenize the patient and so he remains as eunuch.

Other hypothalamic disorders

Neoplasms, inflammatory lesions, degenerative disorders, pituitary stalk section and injury to the hypothalamus arising from various vascular lesions can give rise to hypogonadotropism associated with a deficiency of other pituitary hormones.

PITUITARY DISORDERS:

If pituitary insufficiency takes place before puberty, it leads to growth retardation associated with thyroid and adrenal deficiency. After puberty, profound hypogonadism is one of the earliest signs. It is followed by thyroid and adrenal deficiency symptoms. Histologically, the testes show a prepubertal state. Maturation arrest, reduction in diameter of the tubules and progressive thickening and hyalinization of the tunica propria are observed. Serum LH, FSH and testosterone levels are low. Urinary and plasma...
corticoids may be lowered. Plasma TSH is low with reduced $^{131}$I thyroïd uptake.

Prader-Willi Syndrome, Laurence-Moon-Bardet-Biedl syndrome and familial cerebellar ataxia are three rare disorders reported due to defect in hypothalamic - pituitary axis.

**TESTICULAR DISORDERS:**

**Chromosomal abnormalities**

Many of the male infertility cases are because of the somatic chromosomal abnormalities. When the total sperm count was less than 20 million per ml, the incidence of chromosomal abnormality was 1.9 percent, but the value rose to 11 when the sperm count was less than 10 million. De Krester et al. (1972) have shown high incidence of chromosomal defect in oligozoospermic and azoospermic patients. List of chromosomal abnormalities associated with male infertility is given below:

<table>
<thead>
<tr>
<th>Sex chromosomal</th>
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<tbody>
<tr>
<td>XXY</td>
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<tr>
<td>XY/XXY</td>
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<td>XXXY</td>
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<td>XY/XO</td>
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<td>XY/XX</td>
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<tr>
<td>XY q+/q-</td>
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1. Klinefelter's syndrome

Klinefelter's syndrome is the most common form of hypogonadism affecting 0.2 percentage of the human population. The disorder is characterized by gynecomastia, azoospermia, atrophic testes, increased urinary excretion of pituitary gonadotropins and various degrees of under androgenization. The fundamental defect in these patients is the presence of two or more X chromosomes. All the cases with two or more X chromosomes plus at least one Y chromosome in all or part of his body tissue are included under Klinefelter's syndrome. All patients of this syndrome have firm testes of small size (usually less than 2.0 cm and always less than 3.5 cm in length) with defect in spermatogenesis. Klinefelter's syndrome is very much similar to decreased Leydig cell function except for the gynecomastia. Gynecomastia usually occurs bilaterally first during adolescence. It remains painless and slowly disfigure the patient.

The histological structure of testes shows the hyalinization and fibrosis of seminiferous tubules and adenomatous clumping of the Leydig cells. Plasma and urinary LH and FSH levels are high and on the average plasma testosterone levels are only 50% of normal. However,
the level of testosterone shoots up when LH level is high. So it is advisable to estimate LH rather than testosterone in Klinefelter's syndrome.

Approximately 30 other karyotypic varieties of Klinefelter's syndrome have been described other than the typical ones. The most common clinical variants is the 46XY/47XXX mosaics. In these cases, testes may be normal in size and the endocrinal abnormalities are less prominent.

2. XXY syndrome

A true picture of clinical and endocrinological abnormalities in this condition is not known well. These patients are taller in size and have had pustular acne. Their spermatogenesis may be normal, minimally affected or severely damaged. Whenever the count was affected, the spermatogenic arrest remained as a cause.

3. Sex reversal syndrome

Men with karyotype 46XX are included here. These patients resemble those of Klinefelter's syndrome with small testes, gynecomastia and azoospermia. Histologically testes show aspermatogenesis, hyalinization of the tubules and Leydig cell hyperplasia.

4. Sex chromosomal abnormalities associated with intersexuality

The patients included under this title are either with (1) mixed gonadal dysgenesis or with (2) true hermaphroditism. In many cases, at the time of birth, the disease is detected.
However, till they reach the adulthood no symptoms are visible, and it is impossible to detect the disease.

a. Mixed gonadal dysgenesis:

This is a common disorder. This remains as a frequent cause of ambiguous genitalia in the neonate. These patients are mostly not virilized and many of them have been reared as females. All patients have a uterus, a vagina, and at least one fallopian tube; typically there is a testis on one side and a streak gonad on the other. The most common karyotype is 45X0/46XY. After puberty, the virilization is seen in many. But the seminiferous tubules contain no germinal components and only Sertoli cells are present.

b. True hermaphroditism:

In this condition both testicular and ovarian tissue are present. These patients are well masculinized. The most common karyotype is 46XY, recent evidence suggests that these patients have an H-Y antigen.

5. Y Chromosomal abnormalities

In male infertility studies, many variants of Y chromosomes have been reported. 46 XYq+ and 46 XYq- are the commonly seen karyotypic patterns, in this.

6. Autosomal abnormalities

Many autosomal abnormalities are associated with male infertility. D-D translocations, ring chromosomal abnormalities.
reciprocal translocations and Robertsonian aberrations are included under this.

Sertoli cell - only syndrome

The patient with slightly smaller testes than normal complains with azoospermia. Histological examination of testis shows seminiferous tubules slightly smaller in diameter and there is no germinal elements but, there is no hyalinization or peritubular fibrosis. Sertoli cells are present. Leydig cells are normal. Serum LH and testosterone are usually normal. But plasma FSH level is increased.

Congenital anorchia

The patients with congenital anorchia are phenotypic males with 46XY karyotype and non palpable testes. They have complete development of wolffian system, but absence of the mullerian duct structures and gonads. Prepubertally they are like cryptorchids. Usually at the time of surgical exploration it is detected that no testicular tissue is present. However, virilization in these patients are normal, full development of urogenital sinus, external genitalia and normal phallic development. From this we can conclude that the testicular tissue remains absent from 2nd or probably from 3rd trimester.

Myotonia dystrophica

This is a rare condition. In this disorder patient suffers from progressive muscular atrophy, frontal baldness and cataract. Testicular atrophy leads to hypogonadism. The sectional study of testes shows the hyalinization or fibrosis
of the seminiferous tubules to moderate derangement of spermatogenesis. As a reflection of injury to the seminiferous tubules, serum FSH level is elevated. This disorder is inherited as an autosomal dominant trait.

Male Turner's syndrome (Noonan's syndrome; Ulrich's syndrome; XX or XY Turner's phenotype)

This is an inherited autosomal dominant mutation. It affects both male and female with normal karyotype. The signs and symptoms of the disease are webbed neck, short stature, congenital heart disease, cubitus valgus and other congenital defects. This order is very much similar to mixed gonadal dysgenesis, where the karyotype is XO/XY. In male Turner's syndrome the most common clinical finding is pulmonic stenosis and atrial septal defects. Plasma testosterone level may be low, and serum LH and FSH levels may be elevated postpubertally. Cryptorchism with hypoplastic and germinal aplasia of testes is very common. However, some patients may have normally placed testes with normal function and are fertile.

Defective androgen synthesis

The normal spermatogenesis requires presence of high amount of testosterone in semiferous tubule for the stages beyond the pachytene spermatocyte. Consequently, any defect in the synthesis of testosterone inhibits the spermatogenesis. Many of such disorders are hereditary and few are acquired.

In the normal cases, for the conversion of cholesterol to testosterone five enzymes are required. They are
1. 20, 21 - desmolase
2. 3β - hydroxysteroid dehydrogenase and isomerase
3. 17 - hydroxylase
4. 17, 20 - desmolase and
5. 17β - hydroxysteroid dehydrogenase

A defect in any one of these enzymes lead to the defect in testosterone either in utero or in later life. The first three of the enzymes are common with adrenal glands. So the defect in any one of these enzymes occurs in uterine life leads to congenital adrenal hyperplasia incomplete virilization and defective genitalia. The deficiency of one of the last two enzymes will lead to inadequate virilization and ambiguous genitalia.

The phenotypic expression of the disease varies as many defects in enzyme synthesis are known. Thus the clinical picture also extended from hypspadias to those with female external genitalia or with cryptorchism. All patients have regression of mullerian ducts, but virilization of the wolffian ducts, urogenital sinus, and urogenital tubercle varies. At puberty, the degree of sexual maturation varies from florid virilization with or without gynecomastia to complete failure. So far, in all reported cases the common observation is the abnormality of the external genitalia. The diagnosis of this condition is possible by estimation of testosterone and gonadotropin. The level of testosterone is low and that of gonadotropin is elevated.
The testosterone synthesis in adult may be caused by other means like drugs, renal failure or alcoholic cirrhosis.

Certain drugs as the following inhibit spermatogenesis:

1. Aminoglutethimide inhibits the side chain cleavage of cholesterol and subsequent hydroxylation
2. Spironolactone affects the enzyme 17, 20 desmolase
3. Cyproterone acetate acts in the similar way spironolactone affects
4. Cyproketone
5. 17β - estradiol
6. Hydroxymethylene and
7. Medrogestone inhibit the 3β-hydroxysteroid dehydrogenase
8. Testosterone suppress testosterone synthesis.

The high level of testosterone feeds back the hypothalamus and hypophysis to inhibit synthesis and release of gonadotropins and in turn the testosterone (Figure 2). This is an accepted reason for deterioration of infertility cases where the patient is on testosterone therapy. Testosterone selectively suppresses the LH synthesis, thereby lowering the testosterone secretion by Leydig cells. FSH inhibition is not required to inhibit the spermatogenesis. Testosterone synthesis is restored in such cases by HCG administration. HCG is known to behave like LH.

The patients with uremia often complain of libido, reduced potential, sterility, testicular atrophy and gynecomastia.
Testicular biopsies reveal spermatogenic arrest at the level of primary spermatocyte. Leydig cells are normal, but it is reported that testosterone level in blood is abnormal in these patients. Since the LH level was above normal, it is concluded that the Leydig cells are not responding to LH in the presence of high level of urea. It is also supported by the fact that the testosterone synthesis in these patients is doubled after dialysis treatment. A lesion at the level of hypothalamo hypophysis is also postulated by some as these patients respond to cyclopteronc acetate by releasing Gn RH. However, the increased level of Gn RH had no effect on hypophysis.

A proper explanation to the hypogonadism and gynecomastia in patients with alcoholic cirrhosis is not available. The conclusion can be drawn from the various studies that the regular alcoholic consumption lead to the suppression of testosterone synthesis in Leydig cells. In chronic alcoholics the LH secreting cells of adenohypophysis are also damaged.

Defective androgen action

The mechanism of action of androgen in different tissues is known well. Plasma testosterone crosses the cell membrane by simple diffusion and is converted to dihydrotestosterone (DHT). This combines with the cytoplasmic receptor to form dihydrotestosterone-receptor complex. This complex is transferred to the nucleus where it combines with acceptors (A) located on the chromatin. These events
stimulate the synthesis of messenger RNA and then protein synthesis. Defect in this mechanism, at any level, lead to abnormal spermatogenesis and no virilization. Since it occurs, in utero also, the people with this defect is always infertile with ambiguous genitalia.

Defective androgen action can lead to anyone of the four conditions like,

1. complete testicular feminization,
2. incomplete testicular feminization,
3. familial incomplete male pseudohermaphroditism, type 1, and
4. familial incomplete male pseudohermaphroditism, type 2.

ABNORMALITIES OF THYROID FUNCTION:

Dysfunction of thyroid as a cause of sterility is very rare. Since the condition is curable easily, it is advisable to check the patients in this line also.

The testicular biopsies in patients with hyperthyroidism reveal spermatogenic arrest at primary spermatocytic level. The serum levels of FSH and testosterone are elevated.

The interplay of the two glands namely, thyroid and testes is not known. However, the changes may be due to the non specific factors such as stress or increased temperature.
Abnormalities of Adrenal Function:

Impotence and decreased libido are relatively common in patients with Addison's disease. Many showed the serum levels of gonadotropins and testosterone as normal, so the impairment of potency and libido might be due to general weakness of the body which occurs in adrenal insufficiency.

Congenital adrenal hyperplasia is one of the commonest disorder of adrenal function which leads to abnormal spermatogenesis. The cause of the disease is due to the deficiency of two enzymes, 11-hydroxylase or 21-hydroxylase. Normally, the condition is not noticed unless the child reaches few years of age, when the insufficiency of corticosteroid will be recognised. This includes early maturation of genitalia and early development of secondary sexual characteristics. Acne, coarsening of the voice, frequent penile erection and excessive muscular development are highly noticeable within the first few years of life. The testes usually remain infantile in size though the acceleration of masculinization continues. Sterility, small stature and small testes are remarkable features of an adult without getting any treatment in childhood. The hypogonadism is the result of excess of androgen from adrenal gland which suppresses the gonadotropin secretion. Treatment with cortisone cures the patient. It suppresses the production of androgen synthesised by the adrenal gland and restores the pulsatile release of LH. Fertility can be regained by chronic treatment.
A mild form of adrenal hyperplasia is reported by few in which the precocious puberty and masculinization before puberty are commonly observed. Semen shows oligozoospermia, poor sperm motility and an increase in immature forms of spermatozoa. However, empirical treatment with cortisone in those oligozoospermic patients will have to be encouraged. Hormonal study is important before starting the treatment.

**VARICOCELE:**

The varicocele as a cause of infertility was noticed first time in 1930. Since 1929, surgery was considered as a line of treatment. Ligation of varicocele turned to be real successful for the relief of infertility. A summary on the condition follows (Dubin and Amelar, 1977).

The increased temperature in the scrotum was considered as responsible for the condition. The heat might be constant as the blood is stasis. Many reports have shown the increased scrotal temperature in those patients. Their status of gonadotropin and testosterone was found to be normal.

Varicocele usually occurs on the left side owing to the insertion of the left internal spermatic vein, into the renal vein at a right angle; the right internal spermatic vein usually enters the inferior vena cava at an oblique angle. The increased pressure in the venous system may be due to the erect posture. Moreover, the superior mesentric artery and aorta may squeeze the left renal vein while beating in synchrony leading to increased pressure on the valves at
the junction of the internal spermatic and left renal veins.

Radiographically it is shown that the blood from the renal vein flow in a retrograde manner along the left internal spermatic vein. Some believe that the blood from the left adrenal carried more catecholamines and exposure of the testes to this lead to the decreased production of spermatozoa. Some others believe that the highly concentrated toxic metabolic substances such as steroids, which are potential spermatogenic inhibitors, enter the left spermatic vein in undetoxicated form and impair the spermatogenesis.

Considering the above aspects the best treatment to the disease is ligation of the internal spermatic vein at a point above the cross over anastomosis with the venous drainage from the right testis. This technique is also tried in patients of severely impaired fertility without varicocele and found to be success (Anderson et al. 1975).

Report of a study shows that 71% of the patients who underwent ligation of spermatic vein had a marked improvement in semen quality and 55% of their wives became pregnant (Dubin and Amelar, 1977). Mehan (1974) observed 32% of success. Pregnancy was achieved by many after 3 months of the operation in their husbands. Statistically it is shown that the pregnancy achieved after 5.5 mean months of the treatment.
Other treatments include spermatic ligation with medicinal therapy. Treatment with clomiphene citrate alone shows good results according to Check (1973).

ANATOMICAL DISORDERS:

Cryptorchism

The development of male gonads is under the control of hormones, during embryonic life. Well established mechanism helps the gonads to get transferred to its adult position, in scrotal sac, during the last trimester of pregnancy. A defect in this maintenance mechanism, can fail to do so. This condition is called cryptorchidism, it is unilateral or bilateral. The best time for treatment of this condition is between the years of 5 and 10. The HCG administration is accepted as the choice. Thirty five percentage of patients respond to the same. However, LH-RH to children did not effect the undescended testicle except producing testosterone. Provided it fails, surgical intervention is possible. Before puberty, if the testicle(s) is not descended, the sperm production is very much affected leading to azoospermia or oligozoospermia. This condition is explained on the basis of increased temperature inside abdomen which is not favourable for spermatogenesis (Bergada, 1974)

Azoospermia

The cause of this condition is any one of the following two,

1. aspermatogenesis and
2. blockage of the tract.
1. **Aspermatogenesis**

This is due to hormonal (Heller, Moore and Paulsen, 1960) or mental imbalance (Belaisch, 1974), lack of stimulatory or presence of inhibitory enzymes, absence of certain known or unknown chemicals, presence of inhibitory drugs and chemicals (Heller, Moore and Paulsen, 1960; Potts, Beyler and Schane, 1974; Prior and Ferguson, 1950; Stadtler and Horn, 1974), lack of proper nutrition (Joel, 1971), in conditions like febrile illness, diabetes and various infections including mumps, tuberculosis, gonorrhea, prostatitis, acute non specific epididymis, small pox, leprosy (Amelar and Dubin, 1977a) and T-mycoplasma (Fowlkes, Mac Leod and O'Leary, 1975) and in external conditions like exposure to radiation, heat (Kanwar, Bawa and Singal, 1971) high altitude and changed climate (Amelar and Dubin, 1977a). Allergic reactions, paraplegia and renal chronic failure also can lead to aspermatogenesis. Age as well as frequent ejaculation (Mitchell, Nelson and Hafez, 1976) will affect the quality of semen. When all these aforesaid conditions responsible for aspermatogenesis remains as mild, the conditions remains as oligozoospermia or asthenozoospermia or combination of these two - oligoasthenozoospermia.

2. **Blockage of the tract**

A congenital defect which is experienced in the routine investigation is the absence of vas deferens. Since vas and seminal vesicles are originated from the same source, the seminal vesicles also remain absent. The condition can
be detected by the routine semen examination. Fructose is released in semen from seminal vesicle. Its absence in semen may indicate this condition.

Congenitally, in some people, the duct of the male genital system, is not in continuity. This leads to azoospermia, though the production is normal. Condition is generally termed, azoospermia due to occlusion. No chemical method is in use to detect the condition. Testicular biopsy alone help in these cases (Agarwal et al. 1974; Rajan, 1977d).

IMMUNOLOGICAL:

Once organic lesions are ruled out in both partners, after proper investigations, the chances of infertility are due to immunological causes.

The antigenicity of male reproductive secretions is a well established fact. Landsteiner (1899) was the first to demonstrate the antigenicity of spermatozoa (Rao, Sadri and Sheth, 1961). The knowledge about the species specificity of antigen possessed by the mammalian semen was the result of experiments conducted by Mudd and Mudd in 1929 and by many others at a later age (Edwards, 1960). In man, the antigenicity of testes (Kesh, 1962), spermatozoa and seminal plasma (Rao, 1964; Rao and Sadri, 1958; 1960) is well established. There are 17 antigens in seminal plasma (Rao, 1964; Rao and Sadri, 1960).

A well organised immunological mechanism is present in female genital system, especially in the uterus, but not
in vagina (Vaerman, 1975). Thus the introduction of semen into this system turns to be a provocation for the production of antibodies against different semen antigens.

Semen antigens available in the vagina may be taken up by the cells of outer layer of vagina or cervix (Isojima and Ashitaka, 1964). The presence of antigens in reticulo endothelial system lead to isoimmunization of the host. It is demonstrated by Waldmen et al. (1972) that in women the antibodies are also secreted locally. Jones (1980) believe that sometime the antigen is not sufficient enough to produce antibody in general system, but can be detected locally in such cases.

Specific antibodies to the absorbed antigens are produced. They are capable to agglutinate, immobilize, precipitate, destroy or otherwise affect the spermatozoa with antigen source during the following exposures. With further availability of antigen the production of antibodies will continue. This antigen antibody reaction remain as a cause hypofertility and not for sterility (Cohen, 1975).

Many tests are introduced for the detection of immunogenic infertility. They are reviewed recently (Skandhan, 1979). The percentage of sperm agglutinating antibodies reported by different authors as a cause of infertility is given in table 6.

The incidences of sperm immobilizing antibodies are not less. It was seen in 2.0% of infertile women in the
<table>
<thead>
<tr>
<th>Author</th>
<th>Percentage of positive cases</th>
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<tbody>
<tr>
<td>Franklin and Dukes (1964)</td>
<td>78.9</td>
</tr>
<tr>
<td>Schwimmer et al. (1967)</td>
<td>43.7</td>
</tr>
<tr>
<td>Boettcher and Hay (1963)</td>
<td>19.0</td>
</tr>
<tr>
<td>Isojima (1969)</td>
<td>26.5</td>
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<tr>
<td>Glass and Vaidya (1970)</td>
<td>20.5</td>
</tr>
<tr>
<td>Rao and Rangnkar (1970)</td>
<td>9.3</td>
</tr>
<tr>
<td>Vaidya and Glass (1971)</td>
<td>15.0</td>
</tr>
<tr>
<td>Hanafiah et al. (1972)</td>
<td>8.0</td>
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<tr>
<td>Anasbacher et al. (1973)</td>
<td>10.1</td>
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<td>Schiedel et al. (1974)</td>
<td>7.0</td>
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<tr>
<td>Gupta and Garg (1975)</td>
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<tr>
<td>Shulman et al. (1976)</td>
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<td>Skandhan et al. (1976)</td>
<td>6.2</td>
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<td>Shulman et al. (1977)</td>
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<tr>
<td>Sudo et al. (1979)</td>
<td>24.5</td>
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<tr>
<td>Skandhan et al. (1981)</td>
<td>8.0</td>
</tr>
</tbody>
</table>
first study conducted by Isojima and Tien Shun Li (1967). The percentage of positive cases reported by different workers were 2.5 (Vaidya and Glass, 1971), 2.7 (Schiedel et al. 1974), 3.7 (Skandhan et al. 1976), 9.4 (Sudo et al. 1979) and 4.0 (Skandhan, Skandhan and Bhatt, 1981).

The question, why all women are not immunised against spermatozoa, had come up along with the concept of immunological infertility. Results of elaborative immunoelectrophoretic study of Fazio and Katchell (1972) shows that the antigens of seminal plasma are not foreign bodies to women. Das and Padma (1973) put forward a new theory explaining the mechanism as PG present in seminal fluid, secretions of cervix and vagina which could suppress the lymphocyte response to the sperm antigens, resulting in the suppression of antibody production to sperm antigen in fertile women. The hypothesis explains that in some men the amount of PG in semen is less and thus remain as the cause for antibody production in their wives.

Immunoreaction continues to develop when antigen remains in contact. Avoidance of antigen would lead to the fall of antibody titer. This basic principle of immunology was considered as an excellent measure by Franklin and Dukes (1964) for lowering isoantibodies level. Many workers advocated their patients for regular use of condom and intermittent estimation of antibody titer.

When the antibody is no more present, the couple is allowed to discontinue condom therapy (Anasbacher, Yeung and Behrman, 1973). It would be advisable to arrange the
first unprotected intercourse on the day of ovulation, so that fertilization could be expected to happen more quickly instead of its action as a booster antigenic dose.

Autoimmune infertility was first suggested by Rumke and Hellinga (1959). Brokowski (1976) explained that spermatostasis resulting from an occlusion in the vas deference or the epididymis could lead to the injury of epithelial cells of epididymis or seminiferous tubule and entry of antigens and the formation of autoantibodies. Rumke and Hellinga (1959) reported the sperm agglutinating autoantibody as 3%. Many others reported the presence of it in infertility cases (Schwimmer et al. 1967; Skandhan et al. 1976).

Autoimmobilizing antibodies are reported by few workers (Anasbacher, Yeung and Behrman, 1973; Skandhan et al. 1976; Skandhan, Skandhan and Bhatt, 1981).

Antigen against spermatogenesis is reported by Katsah (1967).

The cause for unsuccessful infertility after reunion of vas deferens in post vasectomised patients is mainly immunogenic. Desensitizing therapy, including antihistamines and occasional autohemotherapy is claimed to have attained goal in the treatment for the males (Zaitsev and Pashinyan, 1974).