5.1: Modern Review:

**Derivation:** Oligospermia is made up of two words - Oligo and Spermia. Oligo means small or few and Spermia means spermatozoa.

**Definition:** Oligospermia is one of the causative factors of male infertility where sperm count is less in semen than the normal (i.e. <20 million/ml). In Modern medical science, it is described under Male Infertility.

Infertility is defined as the inability to achieve pregnancy after one year of unprotected intercourse. An estimated 15% of couples meet this criterion and are considered infertile. Conditions of the male that affect fertility are still generally under diagnosed and undertreated. Infertility may be (a) primary where conception has never occurred and (b) secondary where conception has failed to occurred after a period of fertility.

**Incidence:** 80% of couples achieve conception if they so desire; within one year of having regular intercourse with adequate frequency (4-5 times a
Another 10% will achieve the objective by the end of second year. As such, 10% remain infertile by the end of second year.

**Causes of infertility:** 1) Physiological  2) Pathological  3) Psychological

1. **Physiological:** - Due to anovulation infertility is the rule prior to puberty and after menopause. But it should be remembered that the girl may be pregnant even before menarche and pregnancy is possible within few months of menopause. Fertility is lower until 16-17 yrs and after 35 yrs. Conception is not possible during pregnancy as the pituitary gonadal axis is suppressed by hCG and hence no ovulation. During lactation infertility is said to be relative. Despite the fact that the patient is amenorrheic during lactation, ovulation and conception can occur. However, in fully lactating women (breastfeeding 5-6 times a day and spending 60 minutes in 24 hrs), pregnancy is unlikely up to 10 weeks postpartum.

2. **Pathological:** - Conception depends on the fertility of both the male and female partner. An estimated approximately 40-55% infertility are due to female factors alone, 30- 40% due to male factors alone, 10% due to a combination of female and male factors, and 10% remain unexplained in spite of thorough investigation with modern technical knowledge. It is also strange that 4 out of 10 patients of unexplained category become pregnant within 3 years without having any specific treatment.

3. **Psychological:** - Infertility is invariably associated with Psychological stress related, not only to the diagnostic and therapeutic procedures themselves but also to repeated cycles of hope and loss associated with each new procedure or cycle of treatment. These feelings are often combined with a sense of isolation from friends and family. Counseling and stress management techniques should be introduced early in the evaluation of infertility. In addition to the psychological benefits of stress management, it is possible that stress contributes to infertility in some couples.

**Factor responsible for male fertility are:**

- Healthy spermatozoa should be deposited high in the vagina at or near the cervix.
The spermatozoa should undergo changes (Capacitation, acrosome reaction) and acquire motility.

The motile spermatozoa should ascend through the cervix into the uterine cavity and the fallopian tubes.

The spermatozoa should fertilise the oocyte at the ampulla of the tube.

**Causes of male infertility:**

The following causes/factors may be found to be responsible.

1. Defective spermatogenesis.
2. Obstruction of the efferent duct system.
3. Failure to deposit sperm high in the vagina.
4. Errors in the seminal fluid.

1. **Defective Spermatogenesis:** - FSH stimulates spermatogenesis from basal cells of the seminiferous tubules. Sertoli cells envelope the germ cells and support spermatogenesis. Sertoli cell function is controlled by FSH and testosterone. Scrotal temperature should be $1^\circ-2^\circ$ F less than the body temperature. LH is required for the synthesis of testosterone from the Leydig cells. FSH also stimulates the Sertoli cells to produce androgen binding proteins (ABP) and inhibin B. ABP binds to testosterone and dihydrotestosterone to maintain the local high concentration of androgens. Spermatogenesis and sperm maturation need a high androgenic environment. Inhibin B inhibits FSH secretion. Spermatogenesis is controlled predominantly by the genes on Y chromosome. Approximately 74 days are required to complete the process of spermatogenesis. Additional 12-20 days are needed for spermatozoa to travel the epididymis. The causes of defective spermatogenesis are:

- **Congenital**
  - Undescended testes - the hormone secretion remains unaffected, but the spermatogenesis is depressed. Vas deferens is absent (bilateral) in about 1-2% of infertile males.
  - Kartagener syndrome (autosomal disease): - There is loss of ciliary function and sperm motility.
  - Hypospadias: - It causes failure to deposit sperm high in vagina.
• **Thermal factor**: - The scrotal temperature is raised in conditions such as varicocele, big hydrocele or filariasis. Varicocele probably interferes with the cooling mechanism or increases catecholamine concentration. Other causes are using tight undergarment or working in hot atmosphere. In all these conditions, the depressed spermatogenesis may be temporary or reversible.

• **Infection**: - (a) Mumps orchitis after puberty may permanently damage spermatogenesis. (b) The quality of the sperm is adversely affected by chronic systemic illness like bronchiectasis. Bacterial or viral infection of the seminal vesicle or prostate depresses the sperm count. (c) T. Mycoplasma or Chlamydia trachomatis infection is also implicated.

• **General factors**: - Chronic debilitating diseases, malnutrition or heavy smoking reduce spermatogenesis. Alcohol inhibits spermatogenesis either by suppressing Leydig cell synthesis of testosterone or possibly by suppressing gonadotrophin levels.

• **Endocrine**: - Testicular failure due to gonadotrophin deficiency (Kallmann’s syndrome) is rare. FSH level is raised in idiopathic testicular failure with germ cell hypoplasia (Sertoli-cell-only-syndrome). Hyperprolactinaemia is associated with impotence. Other endocrine disorders are Diabetes Mellitus (D.M), Hypopituitarism, Thyroid dysfunction or Adrenohyperplasia.

• **Genetic**: - Common chromosomal abnormality in azoospermic male is Klinefelter’s syndrome (47 XXY). Gene deletions have been detected in the long arm of Y chromosome for patients with severe oligospermia.

• **Iatrogenic**: - Radiation, Cytotoxic drugs, Nitrofurantoin, Cimetidine, β blockers, Antihypertensive, Anticonvulsant and Antidepressant drugs are likely to hinder spermatogenesis.

• **Immunological factor**: - Antibodies against spermatozoal surface antigens may be the cause of infertility. This result in clumping of the spermatozoa after ejaculation.

2. **Obstruction of the efferent ducts**: - The efferent ducts may be obstructed by infection like tubercular, gonococcal or by surgical trauma
(herniorrhaphy) following vasectomy. In Young’s syndrome there is epididymal obstruction and bronchiectasis.

3. Failure to deposit sperm high in vagina (coital problems): -
   - Erectile dysfunction
   - Ejaculatory defect - Premature, retrograde and absence of ejaculation
   - Hypospadias.

4. Errors in seminal fluid: -
   - Unusually high or low volume of ejaculate
   - Low fructose content
   - High prostaglandin content
   - Undue viscosity
   - Sperm abnormality: - Loss of sperm motility (asthenozoospermia), abnormal sperm morphology (round headed sperm, teratozoospermia) is the important factors.

<table>
<thead>
<tr>
<th>Table 5.1.1: Common cause of male infertility</th>
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<tbody>
<tr>
<td><strong>Pre-testicular</strong></td>
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<tr>
<td><strong>Endocrine</strong></td>
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<tr>
<td>• Gonadotrophin deficiency</td>
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<td>• Thyroid dysfunction</td>
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<tr>
<td>• Hyperprolactinaemia</td>
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<tr>
<td><strong>Psychosexual</strong></td>
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<tr>
<td>• Erectile dysfunction</td>
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<tr>
<td>• Impotence</td>
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<tr>
<td><strong>Drugs</strong></td>
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<tr>
<td>• Antihypertensives</td>
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<td>• Antipsychotics</td>
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<tr>
<td><strong>Genetic</strong></td>
</tr>
<tr>
<td>• Klinefelter’s syndrome (47 XXY)</td>
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<tr>
<td>• Y chromosome deletions</td>
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Pathophysiology:

Gonadal and sexual functions are mediated by the hypothalamic-pituitary-gonadal axis, a closed-loop system with feedback control from the testicles (see image below). The hypothalamus, the primary integration center, responds to various signals from the CNS, pituitary gland, and
testicles to secrete gonadotropin-releasing hormone (GnRH) in a pulsatile pattern approximately every 70-90 minutes. The half-life of GnRH is 2-5 minutes. Release of GnRH is stimulated by melatonin from the pineal gland and inhibited by testosterone, inhibin, corticotropin-releasing hormone, opiates, illness, and stress. GnRH travels down the portal system to the anterior pituitary, located on a stalk in the sella turcica, to stimulate the release of the gonadotropins, luteinizing hormone (LH), and follicle-stimulating hormone (FSH).

Fig.5.1.2: Hypothalamic-pituitary-gonadal axis stimulatory and inhibitory signals

Gonadotropin-releasing hormone (GnRH) from the hypothalamus stimulates the release of follicle-stimulating hormone (FSH) and luteinizing hormone (LH) from the pituitary. FSH stimulates the Sertoli cells to facilitate sperm production, while LH stimulates testosterone release from the Leydig cells. Feedback inhibition is from testosterone and inhibin.

FSH and LH, glycopeptides with a molecular weight of 10,000 daltons, are each composed of an alpha chain that is identical to that of human chorionic gonadotropin (HCG) and thyroid-stimulating hormone (TSH), but with a beta chain that is unique for each. FSH has a lower plasma concentration and longer half-life than LH, and it has less obvious pulsatile changes. The pulsatile nature of GnRH is essential to normal gonadotropin release; a continuous stimulation inhibits their secretion.

The hypothalamus also produces thyrotropin-releasing hormone (TRH) and vasoactive intestinal peptide (VIP), both of which stimulate prolactin
release from the anterior pituitary, and dopamine, which inhibits prolactin release. Men with elevated prolactin levels present with gynecomastia, diminished libido, erectile dysfunction, and occasionally galactorrhea. Prolactin inhibits the production of GnRH from the hypothalamus and LH and FSH from the pituitary. Gonadotropin release is modulated by various other signals, such as estradiol (a potent inhibitor of both LH and FSH release), and inhibin from the Sertoli cell, which causes a selective decrease in FSH release.

FSH and LH are released into system circulation and exert their effect by binding to plasma membrane receptors of the target cells. LH mainly functions to stimulate testosterone secretion from the Leydig cells of the testicle, while FSH stimulates Sertoli cells to facilitate germ cell differentiation.

Testosterone is secreted in a diurnal pattern, peaking early in the morning. In the body, testosterone circulates 2% in the free form, 44% bound to sex hormone–binding globulin (SHBG), and 54% bound to albumin. Testosterone is converted to dihydrotestosterone (DHT) by the action of 5-alpha reductase, both locally and in the periphery, and to estrogen in the periphery. Testosterone and estradiol function as feedback inhibitors of gonadotropin release. The testicle contains the Leydig cells and the Sertoli cells and is covered by the tunica albuginea, which also provides septae that divide it into approximately 200-350 pyramids (see image below). These pyramids are filled with the seminiferous tubules. A normal testicle contains 600-1200 seminiferous tubules with a total length of approximately 250 meters. The interstitium between the seminiferous tubules contains the Leydig cells, fibroblasts, lymphatics, blood vessels, and macrophages. Histologically, Leydig cells are polygonal with eosinophilic cytoplasm. Occasionally, the cytoplasm contains crystalloids of Reinke after puberty.
Fig.5.1.3: Testicular histology magnified 500 times

Leydig cells reside in the interstitium. Spermatogonia and Sertoli cells lie on the basement membrane of the seminiferous tubules. Germ cells interdigitate with the Sertoli cells and undergo ordered maturation, migrating toward the lumen as they mature.

Seminiferous tubules are made up of Sertoli cells and germ cells and are surrounded by peritubular and myoid cells.

Sertoli cells are columnar, with irregular basal nuclei that have prominent nucleoli and fine chromatin. They rest on the basement membrane and serve mainly to support, nourish, and protect the developing germ cells and to provide a blood-testis barrier to provide a microenvironment that facilitates spermatogenesis and maintains the germ cells in an immunologically privileged location. Sertoli cells also secrete inhibin, which provides negative feedback on the hypothalamus, and androgen-binding protein, which helps modulate androgen activity in the seminiferous tubules. In addition to FSH, Sertoli cell function is modulated by intratesticular testosterone and signals from peritubular myoid cells.

Germ cells (precursors to spermatozoa) are derived from the gonadal ridge and migrate as gonadocytes to the testicle before testicular descent. In response to FSH stimulation at puberty, germ cells become spermatogonia and undergo an ordered maturation to become spermatozoa. The entire process of development from spermatogonium to spermatid takes 74 days and is described in 14 steps; as they mature the developing spermatids progress closer to the lumen of the seminiferous tubule.
Spermatogonia rest on the basement membrane and contain dense nuclei and prominent nucleoli. Three types are described: A dark (Ad), A pale (Ap), and B cells. Ad cells (stem cells) divide to create more Ad cells (stem cell renewal) or differentiate into daughter Ap cells every 16 days. Ap cells mature into B spermatogonia, which then undergo mitotic division to become primary spermatocytes, which are recognized by their large centrally located nuclei and beaded chromatin. The mitotic division does not result in complete separation; rather, daughter cells maintain intracellular bridges, which have functional significance in cell signaling and maturation.

Primary spermatocytes undergo meiosis as the cells successively pass through the preleptotene, leptotene, zygotene, and pachytene stages to become secondary spermatocytes. During this time, the cells cross from the basal to the adluminal compartments. Secondary spermatocytes contain smaller nuclei with fine chromatin. The secondary spermatocytes undergo a second meiosis and become spermatids. This reduction division (ie, meiosis) results in a haploid chromosome number. Therefore, a total of 4 spermatids are made from each spermatocyte.

Next, the spermatids undergo the process of spermiogenesis (through stages named Sb1, Sb2, Sc, Sd1, and Sd2), which involves the casting of excess cytoplasm away as a residual body, the formation of the acrosome and flagella, and the migration of cytoplasmic organelles to their final cellular location. The acrosome, a derivative of the Golgi process, surrounds the nucleus anteriorly and contains enzymes necessary to penetrate the ovum. The mature spermatid is then located adjacent to the tubule lumen and contains dark chromatin with an oval-shaped nucleus.

After their release from the Sertoli cells into the lumen of the seminiferous tubules, the spermatids successively pass through the tubuli recti, rete testis, ductuli efferentes, and, finally, the epididymis (see image below). The epididymis is a 3- to 4-cm long structure with a tubular length of 4-5 m. As sperm move from the head to the tail, they mature and acquire fertilization capacity. Sperm from the head move with immature wide arcs and are generally unable to penetrate the egg, while those from the tail propel forward and have better penetration capacity. The transit time varies with age.
and sexual activity but is usually from 1-12 days. The epididymis additionally secretes substances for sperm nutrition and protection such as glycerophosphorylcholine, carnitine, and sialic acid.

Sperm next enter the vas deferens, a 30- to 35-cm muscular conduit of Wolffian duct origin. The vas is divided into the convoluted, scrotal, inguinal, retroperitoneal, and ampullary regions and receives its blood supply from the inferior vesicle artery. In addition to functioning as a conduit, the vas also has absorptive and secretory properties. During emission, sperm are propelled forward by peristalsis. After reaching its ampullary portion behind the bladder, the vas joins with the seminal vesicles, at the ejaculatory duct, which empties next to the verumontanum of the prostate. During ejaculation, the ejaculate is propelled forward by the rhythmic contractions of the smooth muscle that surrounds the ducts and by the bulbourethral muscles and other pelvic muscles. Bladder neck closure during ejaculation is vital to ensure antegrade ejaculation.

Fig.5.1.4: Normal male ductal anatomy

Normal ejaculate volume ranges from 1.5 to 5 ml and has a pH level of 7.05-7.8. The seminal vesicles provide 40-80% of the semen volume, which includes fructose for sperm nutrition, prostaglandins and other coagulating substances, and bicarbonate to buffer the acidic vaginal vault. Normal seminal fructose concentration is 120-450 mg/dL, with lower levels suggesting ejaculatory duct obstruction or absence of the seminal vesicles. The prostate
gland contributes approximately 10-30% (0.5 ml) of the ejaculate. Products include enzymes and proteases to liquefy the seminal coagulum. This usually occurs within 20-25 minutes. The prostate also secretes zinc, phospholipids, phosphatase, and spermine. The testicular-epididymal component includes sperm and comprises about 5% of the ejaculate volume.

In addition to the components already listed, semen is also composed of secretions from the bulbourethral (Cowper) glands and the periurethral glands of Litre, each producing 2-5% of the ejaculate volume, serving mainly to lubricate the urethra and to buffer the acidity of the residual urine. The ordered sequence of release is important for appropriate functioning.

For conception, sperm must reach the cervix, penetrate the cervical mucus, migrate up the uterus to the fallopian tube, undergo capacitation and the acrosome reaction to digest the zona pellucida of the oocyte, attach to the inner membrane, and release its genetic contents within the egg. The cervical mucus changes consistency during the ovulatory cycle, being most hospitable and easily penetrated at mid cycle. After fertilization, implantation may then take place in the uterus. Problems with any of these steps may lead to infertility.

Clinical approach of male infertility to investigation:

Objective of investigation

- To detect the aetiological factors
- To rectify the abnormality in an attempt to improve the fertility
- To give assurance with explanation to the couple if no abnormality is detected.

History: Age, duration of marriage, history of previous marriage and proven fertility if any, are to be noted.

A general medical history should be taken with special reference to sexual transmitted diseases, mumps orchitis after puberty, diabetes, recurrent chest infection or bronchiectasis. Relevant surgery such as herniorrhaphy, operation on testes or other surgeries in the genital area are to be enquired. Occupational history should be directed towards exposure to excessive heat or radiation. Enquiry about the sexual history includes frequency of intercourse, full penetration of penis inside the vagina or orgasm in right time,
whether either partner experiencing discomfort or lack of satisfaction. Social habits, particularly heavy smoking and alcohol are of importance.

**Examination:** A full physical examination is performed to determine the general state of health. Examination of the reproductive system includes inspection and palpation of the genitalia. Attention should be paid to the size and consistency of the testicles. Testicular volume (measured by an orchidometer) should be at least 20 cm$^3$. Presence of varicocele should be elicited in the upright position.

**Investigations:**

(a) Routine investigations include urine and blood examination including postprandial sugar.

(b) Seminal fluid analysis: This should be the first step in investigation because, if some gross abnormalities are detected (Example being absence of sperm- azoospermia), the couple should be counseled for the need of ART (Assisted Reproductive Technology).

<table>
<thead>
<tr>
<th>Table 5.1.5: Normal semen values as suggested by WHO (2002)</th>
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<tbody>
<tr>
<td><strong>Volume</strong></td>
</tr>
<tr>
<td><strong>pH</strong></td>
</tr>
<tr>
<td><strong>Sperm concentration</strong></td>
</tr>
<tr>
<td><strong>Total sperm count</strong></td>
</tr>
<tr>
<td><strong>Motility</strong></td>
</tr>
<tr>
<td><strong>Morphology</strong></td>
</tr>
<tr>
<td><strong>Viability</strong></td>
</tr>
<tr>
<td><strong>Leucocytes</strong></td>
</tr>
<tr>
<td><strong>Sperm agglutination</strong></td>
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Collection – The collection is best done by masturbation failing which by coitus interruptus. The semen is collected in a clean wide mouthed dry glass jar. The sample so collected should be sent to the laboratory as early as possible so that the examination can be performed
within two hours. The coitus should be avoided for 2-3 days prior to the test (abstinence).

Normal values: There is at present no standard of normalcy for semen but the given mentions are an average. Considerable variation occurs from person to person and at different times in any one individual. The sperm count gives reflection of the man’s health status 74 days back, when actually spermatozoa were produced. There is wide range of normalcy and it is not wise to interpret the analysis as abnormal, if the values are found lower than mentioned. However, one should repeat the test at least twice at about two and half month’s interval, if the report is abnormal.

In selected cases, biochemical tests of creatinine phosphokinase and reactive oxygen species are done as sperm function tests. Creatinine phosphokinase helps sperm transport while reactive oxygen species and the peroxides interfere with sperm function.

<table>
<thead>
<tr>
<th>Nomenclature</th>
<th>Description</th>
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<tbody>
<tr>
<td>Aspermia</td>
<td>Failure of emission of semen (no ejaculation).</td>
</tr>
<tr>
<td>Oligospermia/ Oligozoospermia</td>
<td>Sperm count is less than 20 million per ml.</td>
</tr>
<tr>
<td>Polyzoospermia</td>
<td>Count is more than 350 million per ml.</td>
</tr>
<tr>
<td>Azoospermia</td>
<td>No spermatozoosperm in the semen.</td>
</tr>
<tr>
<td>Asthenozoospermia</td>
<td>Reduce sperm motility.</td>
</tr>
<tr>
<td>Leucocytospermia</td>
<td>Increase white cells in semen.</td>
</tr>
<tr>
<td>Necrozoospermia</td>
<td>Spermatozoa are dead or motionless.</td>
</tr>
<tr>
<td>Teratozoospermia</td>
<td>70 % spermatozoa with abnormal morphology.</td>
</tr>
<tr>
<td>Oligoasthenoteratozoospermia</td>
<td>Disturbance of all 3 variables.</td>
</tr>
</tbody>
</table>

Normal male fertility requires a count of over 20 million spermatozoa per ml and a progressive motility of over 25 percent.
However, it is not rare to have pregnancy with a sperm count of 5 million per ml. If the semen analysis is normal and the wife confirms that ejaculation takes place in her vagina, no further investigations are needed in the male. Semen values normally vary widely. Two properly performed semen analysis at least 4 weeks apart should be done when one report is abnormal.

In case of azoospermia, oligospermia, low volume ejaculate, problem in sexual potency; the following diagnostic procedure are essential.

- **Serum FSH, LH, testosterone, prolactin and TSH:** Testicular dysfunction causes rise in FSH and LH. Low level of FSH and LH suggest hypogonadotrophic hypogonadism. Leyding cell dysfunction causes low testosterone and high LH level. Elevated prolactin due to pituitary adenoma may cause impotency.
- **Fructose content in the seminal fluid:** Its absence suggests congenital absence of seminal vesicle or portion of the ductal system or both.
- **Testicular biopsy:** It is done to differentiate primary testicular failure from obstruction as a cause of azoospermia or severe oligospermia. The biopsy material is to be sent in Bouin’s solution and not in formal saline.
- **Transrectal Ultrasound (TRUS):** It is done to visualise the seminal vesicles, prostate and ejaculatory ducts obstruction. Indications of TRUS are: (i) Azoospermia or severe oligospermia with a normal testicular volume. (ii) Abnormal digital rectal examination. (iii) Ejaculatory duct abnormality (cysts, dilatation or calcification). (iv)Genital abnormality (hypospadius).
- **Vasogram:** It is a radiographic study done to evaluate the ejaculatory duct obstruction. It is mostly replaced by TRUS.
- **Karyotype analysis:** This is to be done in cases with azoospermia or severe oligospermia and raised FSH. Klinefelter’s syndrome (XXY) is the commonest. Micro deletions of the long arm of Y chromosome can also cause severe seminal abnormalities.
- **Immunological tests:** Two types of antibodies have been described – sperm agglutinating and sperm immobilizing; the latter is probably related to infertility. The antibodies are produced following infection (orchitis),
trauma or vasectomy. These antibodies can be detected from the serum by the sperm immobilizing test. Presence of sperm antibodies in the cervical mucousa is demonstrated by post-coital test.

- Presence of plenty of pus cells requires prostatic massage. The collected fluid is to be examined by staining and culture to detect the organisms and appropriate antibiotic sensitivity.

**Treatment:**

The treatment of male is indicated in (i) extreme oligospermia, (ii) azoospermia, (iii) low volume ejaculate and (iv) impotency.

To improve spermatogenesis the following measures may be helpful.

1. **Assurance:** The infertile couple remains psychologically disturbed right from the beginning, more so as the investigation progresses. The couple in such cases should be tactfully handled to minimize psychologic upset. When minor defects are detected in both the husband and the wife, each of which alone could not cause infertility but in combination, they significantly decrease the fertility potential. As such, the faults should be treated simultaneously and not one after the other. Even when a gross abnormality is detected and the prospect of pregnancy is bleak, an optimistic discussion is worth rewarding.

2. **Body weight:** Overweight or underweight of any partner should be adequately dealt with to obtain an optimum weight. Body mass index of 20-24 is optimum.

3. **Smoking and alcohol:** Excess smoking or alcohol consumption is to be avoided.

4. **Coital problems:** The coital problems should be carefully evaluated by intelligent interrogation. Advice to have intercourse during the midcycle too often gives the result early enough even prior to investigation. Using LH test kit, one can detect LH surge in urine by getting a deep blue colour of dipstick. The test should be performed daily between day 12 to day 16 of a regular cycle. Timed intercourse over 24-36 hours after the colour change reasonably succeeds in conception. Minor psychosexual problems should be corrected accordingly.
5. General care: Improvement of general health, reduction of weight in obese, avoidance of alcohol and heavy smoking are of help. Avoidance of tight and warm undergarments or occupation that may elevate testicular temperature is helpful.

6. Use of vitamins E, C, D, B₁₂ and folic acid as antioxidants to improve spermatogenesis is of doubtful value.

7. Medications that interfere spermatogenesis should be avoided.

8. In hypogonadotrophic-hypogonadism, the disorders of spermatogenesis can be treated with the following therapy with varying success.
   - hCG 5000 IU intramuscularly once or twice a week is given to stimulate endogenous testosterone production.
   - hMG is added to hCG when there is no sperm in the ejaculate with hCG alone.
   - Dopamine agonist (cabergoline) is given in hyperprolactinaemia to restore normal prolactin and testosterone level. This improves libido, potency and fertility.

9. Pulsatile GnRH therapy in infertile male with GnRH deficiency (Kallmann’s syndrome) is effective. It is administered by minipump infusion. Cases with hypogonadotrophic hypogonadism may also respond with GnRH therapy.

10. In hypergonadotrophic-hypogonadism, no form of medical treatment can improve fertility in men.

11. Clomiphene citrate 25-50 mg orally daily for 25 days with rest for 5 days for 3 cycles is given. It increases serum level of FSH, LH and testosterone.

12. In presence of antisperm antibodies in the male, its significance is unclear and the benefits of systemic corticosteroids are uncertain. Currently intrauterine insemination (IUI) with washed spermatozoa is the choice of treatment for such cases.

13. Genital tract infection needs prolonged course of antibiotics. Generally doxycycline or erythromycin for a period of 4-6 weeks depending on the response.

14. In retrograde ejaculation – phenylephrine (α-adrenergic agonist) is used to improve the tone of internal urethral sphincter. Sperm may be recovered from the neutralized urine. Processed spermatozoa could be used for IUI.
15. In teratospermia, asthenospermia specific causes are unknown. No treatment is available. Donor insemination (AID) is the option.

16. In genetic abnormality – artificial insemination with donor sperm (AID) is the option as no other treatment is available.

17. Surgical
   - When the patient is found to be azoospermic and yet testicular biopsy shows normal spermatogenesis, obstruction of vas must be suspected. This should be corrected by microsurgery – vasoepididymostomy or vasovasostomy. After vasovasostomy patency is obtained in about 80% of cases and pregnancy rate is about 50%.
   - Surgery for varicocele for improvement of fertility is not helpful. Hydrocele is corrected by surgery.
   - Orchidopexy in undescended testes should be done between 2-3 years of age to have adequate spermatogenesis in later life.

18. For impotency psychosexual treatment may be of help. Hyperprolactinaemia needs further investigation and treatment. For erectile dysfunction sildenafil (25-100 mg) or tadalafil (10-20 mg) is currently advised. A single dose (depending on response) is given orally one hour before sexual activity. In unresponsive cases, artificial insemination is to be thought of.

19. Assisted reproductive technology (ART) for male infertility – Prospect of male infertility has improved significantly with the advent of ART. IUI, TESE, PESA, MESA and intracytoplasmic sperm injection (ICSI) are now the treatment available for infertile males.

5.2: Ayurvedic Review:

Based on the available references it is clear that śukra term is used for whole testicular secretions, which contains sperms and androgens. Term Retas for total ejaculate means semen and Virya for only androgens.

Hence the term śukra Kshaya or Ksheena śukra can be taken as any decrease in the śukra either quantitatively or qualitatively. Thus the term śukra Kshaya or Ksheena śukra seems to be analogue with the term Oligospermia.
Nidana of Ksheena śukra: In the classics the etiology of Ksheena śukra is described in detail. For better understanding, these causative factors can be divided into the factors pertaining to Aharaja, Vihara, Manasika, Nidanarthakara and Vaidya Krit, Avasthajanya hetu, Anyahetu. Accordingly they are being described here in detail under separate headings.

1. Aharaja Hetu:
   a. Asatmya ahara
   b. Rooksha, Laghu, Ushna ahara
   c. Excessive intake of Teekta, Kashaya, Amla, Lavana ahara
   d. Anashana
2. Viharaja Hetu
   a. Ati Vyayama
   b. Atimaithuna
   c. Akalamaithuna
   d. Ayonimaithuna
3. Manasika Hetu
   a. Chinta
   b. Soka
   c. Bhaya
   d. Krodha
4. Vaidya krit Hetu
   a. Shastrakarma Vibramsha
   b. Ksharakarma Vibramsha
   c. Agnikarma Vibramsha
5. Avasthajanya Hetu
   a. Vriddha avastha
   b. Vyadhi avastha
6. Kshataja Hetu

1. Asatmya Ahara Sevana: It means the substances or practice, which is incompatible to the body. Here we can list alcohol, tobacco etc. Excessive consumption of alcohol (more than 60 gm/day), tobacco and narcotics leads to infertility. Alcohol consumption lowers plasma testosterone synthesis. Ethanol increases the metabolic clearance rate of testosterone concomitants
with an increase in hepatic 5 alpha reductose activities and increased conversion of androgen into estrogen. Thus testicular dysfunction may occur in patients prior to alcohol liver cirrhosis.\textsuperscript{41}

Excessive consumption of Ruksha, Tikta, Kashaya, Katu, Lavana and Amla Rasa leads to šukra Dushti in terms of Ksheenašukra.

Excessive intake of Lavana and Katu Rasa leads to Pumsatva Upghata\textsuperscript{42}. In the same manner excessive intake of Tikta Rasa leads to šukra Shoshana and srotokharatya\textsuperscript{43}, consumption of excessive Kashaya Rasa leads to šukravarodha and Srotovibandha. On the basis of above, it may be said that excessive usage of the above said Rasa might be in any form i.e. Ahara or Aushahdi will lead to the impairment of šukra either qualitatively or quantitatively.

Anashana - Ati Ananashana or fasting which is one of the main reasons for Ksheena šukra is the Vata Prakopaka Nidana. Sport star, High proportion of trained athlete, ballet dancers and gymnasts, who practice regular light diet i.e. stringent weight control are reported to have infertility. Thus there must be an optimal body weight for successful reproductive function. Restoration of body weight near to ideal body weight (IBW) results in restoration of normal gonadotrophin secretary pattern. Serum testosterone level decreases during fasting and intense exercise in healthy individual.

\textbf{2.Viharaja Hetu}\textsuperscript{44}: Over indulgence exercises leads to Vata Prakopa, this in turn causes \textit{depletion of successive tissue elements and finally šukra Kshaya}. \textit{The physical effects of strenuous exercise on hypothalamic, pituitary gonadal function in male have been established, decreased testicular androgens (testosterone and dihydrotestosterone) and increased adrenal androgen have been noted in the plasma of highly trained male athlete. This reference clearly supports the view of Charaka that Ati Vyayama is a causative factor of Ksheena shukra. The causative factors such as Ati Vyavaya (excessive intercourse), Akala Yonigamana (intercourse in improper time), Ayoni Gamana (intercourse in other than vagina / perverted sexual activities) and Atišukra Sravana directly lead to the Pratiloma Kshaya of šukraDhatu. Now it is a fact that the silent infection (asymptomatic) of semen also leads to male infertility because of poor semen quality.}
Ati Ushna Sevana: Ati Ushna Sevana means exposure to excessive hot zones as well as intake of hot articles. The temperature within the scrotum is normally 1 to 2 degree lower than ambient body temperature. It is generally believed that this lower temperature is necessary for normal testicular function. Therefore thermal exposure or working near hot furnaces, radiation (X-ray exposure) and routine use of hot tubs is the cause where the elevated temperature directly applied to the testis can impair spermatogenesis.

The testes are sensitive to radiation damage; decreased secretion of testosterone appears to be a consequence of diminished testicular blood flow.

3. Manasika Hetu:\(^{45}\): The main culprit involved here may be taken as Vyana Vata because Manas and Vyana Vata are interrelated, by the virtue of their common site i.e. Hridaya and the inter-site disturbances or disequilibrium can lead to the dysfunction of both factors. So any disturbances in Vyana Vata and Manas may lead to disturbance in \(\text{sukra Vaha Srotas}\), as it is present all over the body, where Vyana Vayu moves around.

Among the above said factors Chinta, Shoka, Bhaya and Krodha are the factors, which are having root cause in depression/anxiety. Recent research categorized losses in adulthood, which are of greatest importance or etiological factors in depression.

These include losses of relationships, health status or prestige, self-esteem, self-confidence, security, a fantasy or hope of fulfilling an important fantasy and something and someone of great symbolic value. Any one of these losses may precipitate a depressive reaction in adult \(^{46}\).

4. Vaidyakrita Nidana (iatrogenic Causes): Under this we can consider Shastra, Kshara and Agni Karma Vibhrama which are mentioned in Charaka Samhita as the Nidana for \(\text{sukra Dushti}\).

The patients, who have had a bladder neck surgery along with urethral re-implantation during childhood, may be infertile. Because the bladder neck surgery has ablated the internal sphincter. These individuals often experience retrograde ejaculation and their ejaculate will be less than 1ml in volume and Oligospermic are Azoospermic in nature.
Apart from surgery, many drugs may interfere with spermatogenesis either directly or through alterations in the endocrine system. Medication such as sulphasalazine cimetidine, nitrofurantoin or ingestants such as caffeine, nicotine, alcohol, marijuana and cocaine has also been implicated as gonadotoxic agents. Withdrawal from these substances enables the return of normal spermatogenesis. The use of anabolic steroids, usually by athletes may also interfere with normal spermatogenesis.

Hence different surgical, para-surgical measures, medications may lead to abnormality of semen. It has been carefully analyzed and mentioned in our classics that while doing Shastra karma especially in the pelvic region Vitapa marma should be avoided. If any injury to Vitapa-marma it may lead to Shukra-dusti. While dealing with Kshara, it has been directly labeled as the cause for Pumsatwa Nasha.

5. Avasthajanya Hetu:

a) Vriddha avastha/Jara (Ageing): During old age the Kshaya of all the Dhatu takes place. Hence Shukra Kshaya occurs as a physiological process. Effect of age on Spermatogenesis - Human testicular parenchyma is significantly reduced in aged men. Other age related changes include decreased volume of seminiferous epithelium, increased thickness of the tubular wall and decreased length of the tubule.

Lower seminiferous epithelial volume in older men is associated with lower daily sperm production per man. Age related reduction in daily sperm production in humans is significantly correlated with loss of sertoli cells. Furthermore old men have additional germ cell degeneration during the prophase of meiosis, in addition to the physiological germ cell loss at the end of meiosis in the normal cycle.

b) Vyadhi avastha/Karshana Janya (Disease Induced): Vyadhi-Karshana is told as one of the cause for the Sukradusti. But there is no exact reference for śukraDushti being one of the Upadrava in any one of the diseases in Charaka except Sthaulya. Atisthulata is considered as undesirable and the person with overweight will have krichra Vyavayata (difficult in copulation) due to the less quantity of śukra. Sushruta while dealing with Sahaja Arsha
mentions that the person suffering from Sahaja Arsha will have Ksheena Retas. In Madhava Nidana Parishishta, Atisara is mentioned as one of the disease, which will cause śukra dushti.

The review of andrological / Urological literature as well as other literature pertaining to medicine gives many diseases that will ultimately lead to poor semen quality.

6. Kshataja Hetu (Trauma): It can be taken as any external injuries due to trauma, accident, surgery etc. Vitapa Marmabhigata may lead to śukravaha Sroto Dushti. Vitapa Marma is Vaikalyakara Marma and Saumya; therefore it may show its effect after certain time and not immediately. So the external injuries to vital organs Vrishana, Shepha and the surrounding Marma when get injured will develop grave disturbances in śukravaha Sroto Moola, to vitiate śukra, hence these are to be handled carefully. Injury to testis, testicular torsion may lead to atrophy of the testis and impaired fertility.

Poorvaroopa: Poorvaroopa are the premonitory features occurring before exhibiting of the main symptoms indicating a disease are known as poorvaroopa. No specific poorvaroopa have been mentioned for Ksheena śukra in the classics, the lakshna in the avyakta stage can be taken as poorvaroopa of ksheena śukra.

Roopa and:

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<th>Sl. No.</th>
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Samprapti: According to modern view emotional stress may interfere with the GnRH (Gonadotrophin releasing hormone), which initiates the release of LH (Lutinizing Hormone) and FSH (Follicle Stimulating Hormone) from anterior Pituitary, which are responsible for spermatogenesis. Thus disturbance in GnRH secretion ultimately results in defective spermatogenesis. Dosha Prakopa here may be interpreted as hormone imbalance.

Pitta Prakopa may be caused increased temperature due to thermal exposure, radiation, hot tubs, where elevated temperature directly applied to testis can impair spermatogenesis.

Khavaigunya Karaka Nidana like Abhigata, Shastra, Kshara, Agni Karma Vibrama mentioned in the classics directly affects the Shukravaha Srotas especially testis and resultant Shukra Kshaya. Any surgery or drugs (Sulphasalazine, Citeratedin, Alcohol), which are gonadotoxic agents, that affects spermatogenesis, can be considered here.

Samprapti of Ksheena śukra Based on Kriyakala:
Sanchaya and Prakopa: In this stage the causative factors vitiate Sharirika and Manasika Dosha initially. The Manasa and Sharira Hetu are interacting i.e. they disturb each other depending on the indulgence in Hetu and/or severity and susceptibility of the site. The vitiated Sharirika Dosha in turn vitiates Agni to resulting in the production of Ama. The whole process might be mediated by Manas and Manasa Dosha at any level to produce Ama. Further Ama vitiates the functioning capacity of Dosha. The site of this manifestation has its base at Hridaya, Vyana Vata. At this stage the vitiation of Dosha can also be produced by external factors like Kshata, improper use of Shastra, Kshara and Agni Karma. Hence this state of Vaishamya of Dosha
might be at the level of Hridaya being able to be named as Sanchaya and Prakopavastha, the first and second stages of disease manifestation.

**Prasaravastha:** In the extension of Prakopavastha, the enough strong Doshas will move from this site to the next place for further vitiation of Dhatu, as the whole of sentient body is both the vehicle and the field of operation. The Rasadi Dhatus are vitiated and further provoked by Manas, Manasa Vikara and Hetu along with morbid Dosh at Rasadi Srotas. The role of Manas cannot be neglected in this context. Manas vitiated by its own Nidana, moves through Dashadhamani to the level of śukra Vaha Sira to vitiate the same, as śukra is also traversed in the whole body and Manas is also Sukshma and pervading in whole body. The vitiation of Rasa Dhatu and the extension of the same further to Raktadi Dhatu, which come later to Rasa in the ladder of metabolic discreet, are named as Anuloma type of Dushti. Here another process of manifestation is also possible when the enough potent Hetu directly vitiate Shukra and Shukra Vaha Srotas, there the below upward type of Dusti starts from Shukra Dhatu to Rasa Dhatu. The same thing is termed also as Pratioma type of Dusti e. g, Ativyavaya without consuming Vajikarana Yoga. This complete mechanism can be named as Prasara Stage of pathogenesis.

**Sthana Samshraya:** The vitiated Sharirika and Manasika Doshas vitiate the Shukra Vaha Srotas, thereby disturbing the functioning balance of Sarva Sharirastha śukra (Endocrine function) and Svasthanastha śukra (Retas) at the level of Moolla of śukra Vaha Srotas (Vrishana and Sepha). This is termed as Dosha Dushya Sammurchana. Here if due to any reason either due to Bija dosha or any other external factors like Kshata to śukravaha Srotas (scrotal injury), the Srotas is having any susceptibility that becomes the best site accordingly for the symptom presentation and may be termed as Khavaigunya. This stage is the endogenous presentation of Sthana Samshraya of Ksheena śukra. In other way, some external causes at the level of Sarva Sharira or Vrushana and Sepha can lead to direct Sthana Samshraya for Ksheena śukra viz., Sephabhighata, Shashtra karma Vibrama. This is the 4th stage of Samprapti i.e. Sthana Samshraya.
This is also the stage when Poorvarupa are noticed. Though specific prodromal symptoms are not explained in the classics, the Avyakta or Ishat Vyakta Lakshana present all over the body or locally can be anticipated according to the severity of Hetu as well as Doshic vitiation.

**Vyakta:** The Dosha Dushya Sammurchana at śukravaha Srotomoola further extends and lands up in śukra Dusti in general and in Ksheena śukra in particular producing the Lakshana pertaining to Sarva Shareera and also Vrushana and Medra.

**Bhedā:** It is the 6th stage, wherein if the disease progresses due to non-availability or failure of treatment the Upadrava appear and makes the disease impossible to cure.

**Samprapti Ghataka:**

<table>
<thead>
<tr>
<th>Dosha</th>
<th>Tridosha with dominate Vata and Pitta dosha</th>
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<tr>
<td>Dushya</td>
<td>Rasa, Majja, Shukra</td>
</tr>
<tr>
<td>Agni</td>
<td>Jatharagni, Dhatwagni</td>
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<td>Srotas</td>
<td>Rasavaha, Sukravaha, Manovaha</td>
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<td>Amashaya</td>
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<td>Sanchara Sthana</td>
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<tr>
<td>Adhisthana</td>
<td>Vrushana and Medra</td>
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<tr>
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<td>Madhyama</td>
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<td>Vyakta Sthana</td>
<td>Shukra Dhatu</td>
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**Dosha:** The disturbance of equilibrium of Dhatu cannot occur without the vitiation of Vata, Pitta and Kapha. So considering the location, signs and symptoms and cause of vitiation of Vata, Pitta and Kapha, all the diseases by them are diagnosed on the basis of vitiation of respective Dosha.

**Vata:** It is considered to be the vital cellular force for movement or Gati in the body having Sukshma (subtle) property to pierce minute channels in the body and to carry other morbid Dosha into different parts of the body to cause the diseases according to specific Samprapti.\(^{58}\)

Further Sushruta has referred another authentic thing that śukra Dosha are specifically caused by the vitiation of Vyana and Apana Vayu.\(^{59}\) Ksheena Shukra being one of the major varieties of śukra Dosha, the affliction of Vyana and Apana Vata cannot be denied. Here Vyana seated in Hridaya which
circulates in the whole body act as central controlling system and Apana seated in Basti which circulate in Medra acts as peripheral controller of physiology of ejaculation of šukra. Moreover the qualities of Vata like Ruksha, Laghu etc., are quite opposite to the qualities of šukra. So increased Ruksha Guna of Vata, decreases the Snehamsha of šukra leading to šukra Shoshana.

Therefore it can be concluded that Vyana and Apana Vata dusti causes šukravaha Sroto Dusti and further leads to šukra Kshaya i.e. disturbance in the functioning of Vyana Vata leads to the defect in the production of šukra by affecting Sarvadaihika Shukra (Androgens) whereas in Apana Vata Dusti production of šukra will be normal but there will be difficulty in ejaculation due to loss of Snigdhamsha in šukra.

Pitta: Aggravated Pitta due to its Ushna Guna dries up the Somatmaka šukra leading to šukra Shoshana and intern Ksheena šukra.

Vitiated Agni due to Anashana etc., Nidana leads to Mandagni and production of Ama. This Ama is responsible for improper production of Rasa Dhatu which finally results in improper nourishment of šukra Dhatu present in whole body as well as in šukra Vaha Srotomoola i.e. Vrishana and Sepha. Over exposure to Ushnata also increases Pitta, which may be taken as increased temperature in the Vrushana Pradesha leading to defective spermatogenesis.

Kapha: Though šukra Kshaya is a disease in which Vata and Pitta are the main morbid Dosha but the role of Kapha Dosha cannot be ruled out. Kapha and šukra have the Ashraya Ashrayi Bhava. Kapha is sheltered in šukra and both have almost similar Guna. So by seeing the šukra Kshaya Lakshana Kapha Kshaya can be inferred.

In the Samprapti Kapha gets vitiated by the Ama i.e., the Picchila Guna of Kapha is increased by Ama and this causes the obstruction in the micro circulatory channels of Shukra Vaha Srotas leading to Ksheena šukra.

Dooshya: It includes Rasa and šukra. Though from Rasa to šukra all the Dhatu are involved in the pathogenesis, but apart from Rasa and šukra the involvement of other Raktadi Dushya are negligible.

In Ksheena šukra as explained earlier due to Agnimandya and Ama the formation of Rasa Dhatu is hampered. As Khalekapota Nyaya refers to the
need for specific ingredients for generation of each tissue form, this mechanism will be interrupted by the improper formation of Rasa i.e., the further Dhatu especially śukra will be devoid of its nourishing material.

**Agni:** As Dhatvagni always depends on Jatharagni the role of Jatharagni is of utmost importance. Any impairment in Jatharagni can cause impairment in śukradhatvagni. So the disturbances in Agni disturb the functions of śukra. Hypo functioning of Dhatvagni especially Rasagni occurs due to diminished Jatharagni and results in the production of improperly metabolized Rasa Dhatu. This results in depletion of further Dhatu, along śukra.

**Ama:** Ama is produced in the Koshta due to hypo-functioning of Jatharagni later due to hypo-functioning of Dhatvagni. Ama is the prime factor for the increase of abnormal Picchila Guna of Kapha, which causes the obstruction in the pathway of śukra.

The five Bhutagni are responsible for metabolism at molecular level. The deficiency or diminution of this Agni may result into the production of unwanted and incompatible products of finer metabolism. Free radicals that may be identified as Ama at the first level considered being responsible for tissue damage. This has been proved fact that the primary suspects in the link between environmental assaults and infertility are oxygen free radicals, also called oxidants. These are unstable particles that are released as a by-product of many natural chemical processes in the body. Infections and environmental assaults can produce high level of oxidants. Evidence now strongly indicates they can also damage sperm.

**Srotas:** The pathogenesis confirms at the level of Srotas in any of the disease and hence also in Ksheena śukra. The chief Srotas involved in the pathogenesis of Shukra Dushti viz., Ksheena śukra are Rasavaha Srotas, śukravaha Srotas and Manovaha Srotas.

Shukravaha Dhamani\(^{60}\) is categorized under Adhogami Dhamani amongst which two acts for the production of śukra and other two work to ejaculate the same. Adhogami Dhamani in general is intended to pass/expel the substances viz., Mutra, śukra, from above downwards. Any of the disturbances at these dhamani due to their respective Hetu will land up in disturbances either in śukra production process or śukra Visarga (ejaculatory)
properties. Hence it seems that the production and ejaculation of šukra (Semen and Androgen) is controlled by Neuro-endocrinal-vascular system, the intactness of which aids to the proper production and ejaculation of šukra and any disturbance in the same may lead to the pathological manifestations of production or ejaculation.

**Lakshana of Ksheena-šukra:** Lakshana of Ksheena šukra can be divided into two groups viz. related to šukra Vaha Srotas and those symptoms related to Sharira.

Lakshana of Ksheena šukra can manifest due to dysfunction or hypo-functioning of one or other properties of šukra (Semen and Androgen).

**The symptoms related to šukra Vaha Srotas –**

**Shighrapraseka, Chiradvapraseka and šukraAvisarga:** Snigdha, Sara and Anupravana Bhava are the biophysical or physicochemical properties of šukra as mentioned earlier. The affliction of these properties by any exogenous or endogenous factor leads to its dysfunction. For example lack of Dravatva, Saratva and Anupravanatvatva, due to increased Ruksha Guna of Vata leads to delayed ejaculation (Chiradvapraseka). Defect in Madhurata, Avisrata and Madhugandhi means lack of nutrient substances in ejaculate material and any extra smell is indicative of pathological process, which may lead to ejaculatory problems such as early ejaculation, delayed ejaculatory and anejaculatory (Shighrapraseka, Chiradvapraseka and šukra Avisarga).

**Toda or Vedana of Vrushana and Medra:** If Avidahi and Snigdha properties are hampered due to Ushnam, Teeksha Guna of Pitta and Ruksha Guna of Vata, the effect will be burning sensation (Vrushana and Medrayodhomayateeva), pain in genital organs (Ati Toda, Vrushana Medra Vedana). Dalhana opines that the various Vedana seen in Ksheena šukra are caused due to vitiated Vayu.

**Shonita Alpa Shukra, Mishrita Nissarana:** When other Dosha is associated as Pitta with Vata, it causes Shoshana of Ap Bhaga of Shukra due to Ruksha property of Vata. According to this the Teekshna and Ushna Guna of Pitta does more Shoshana. The Ruksha and Laghu Guna of Vata cause the reduction of Ghanata of Shukra, so Tanuta is seen. Phenila is the resultant of Vata Dosha. Apart from spermatozoa, semen contains pus cells,
precursor cells, macrophages, mucus threads, crystals gelatinous bodies, bacterial matter and RBC, which are all considered as Anyadhatu, and the semen containing excessive above substances may be called Anyadhatu Samsrsta Retas (Mishrita Nissarana).

Presence of RBC can be seen in Vataja Retas as well as Raktaja Retas. To differentiate, in Vataja Retas, only few RBC will be present which gives Arunavarna and there may not be any change in volume of ejaculate, where as in case of Raktaja śukra, hemorrhage will be severe which gives reddish colour and the volume of ejaculate will be high.

The symptoms related to whole Shareera –

Dourdhyaa, Mukhashosha, Panduta, Sadana, Shrama, Timiradarshana and Bhrama: Ksheena śukra will be caused due to the Kshaya in preceding Dhatu. So when Kshaya of almost all Dhatus are involved, the symptoms like Dourdhyaa, Mukhashosha, Panduta, Sadana, Shrama, Timiradarshana and Bhrama can be seen. Also as śukra is pervading all over the Sharira, its Kshaya will show the Lakshana all over the body. Here we can consider the function of testosterone. Testosterone plays important role in protein formation and muscle development also in bone growth and calcium retention. Testosterone increases basal metabolism and it is seen that normal quantities of testosterone are injected into a castrated adult the number of red cells per cubic millimeter of blood increases 15 to 20 percent so it can be consider that it is helpful in production of RBC. Steroids hormones can increases the re-absorption of sodium in the distal tubules of the kidneys.

Upadrava of śukra Kshaya: No direct Upadrava has been mentioned for Ksheena śukra. But while dealing with Shukra Kshayajanya Klaibya, it is said that, as śukra is the end product of Dhatu Parinama, it is said to be the essence (Paramadhama) of food. Its wastage leads to number of serious diseases or even death.

Sadhya Sadhyata: Assessment of the Sadhyasadhyata is mandatory before the commencement of treatment. It determines whether the disease is curable or not. Ksheena śukra being Dwidoshaja is said to be Kriccha Sadhaya, Pitta Prakruti Purusha has less śukra and if he is affected with śukra Kshaya then the prognosis is still more Kasthasadhya.

Chikitsa: Charaka, Sushruta, Vagbhata have dealt with the Chikitsa aspect of śukra Dosha. The Samanya Chikitsa for śukra Dosha are Snehana, Svedana,
Vamana, Virechana, Niruha Basti and Anuvasana Basti followed by Uttarabasthi which can be adopted for all types of śukra Dosha including śukra Kshaya.

The śukra Dosha can be treated with proper diet of Madhura and Tikta Rasa, proper Vyavaya and Vyayama, timely elimination of Dosha in proper quantity.

Coming to the Vishesha Chikitsa of Ksheena śukra, after Panchakarma procedures (Snehana, Vamana, Virechana and Basti) the Shamanoushadhi are to be administered. Basti is considered as the main line of treatment in Ksheena śukra (Ksheena śukrasya chativa snehabastir balapradah).

The principle treatment in any Dhatu Kshaya is to administer the Dravya, which are having the same qualities of that Dhatu Eg. Mamsa in Mamsakshaya, śukra in śukra Kshaya. In śukra Kshaya, Nakra Retah is admissible. In Sushruta Samhita Vajikara Dravyas are particularly recommended for śukra Kshaya in Ksheena Baleeya chapter some of them are Vajikara Utharika, Amalaka Yoga, MashaYoga, Swayamguptadi Yoga.

Dravya having the properties of Madhura, Sheeta, Snigdha, Picchila such as Kshira, Ghrita, Mushali etc. are very good śukra Dravya. Charaka has mentioned śukrajanaaka Gana, 10 Dravya namely Mashaparni, Meda, Shatavari, Kulinga etc.

Uttara Basti: Administration of medicine through urethra of males has been emphasized in the disorders of śukra. Various śukra Shodhana as well as śukra Pravartaka drugs bring about the desired therapeutic effect and helps in palliating the Apana Vata.

CHAPTER 6
DRUG REVIEW

GHRITA

Vernacular name

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<tr>
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<td>Clarified butter</td>
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