Committee 13

Male Orgasmic and Ejaculatory Disorders

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Orgasm and ejaculation constitute the final phase of the sexual response cycle. Although erection and ejaculation are co-ordinated, the mechanisms that produce them are different. We must, therefore, commence by defining our terms, before considering the physiology of normal ejaculation. Disorders of male sexual function affect erection and ejaculation quite differently.

DEFINITIONS

Orgasm: A pleasurable feeling (a cerebral event) usually associated with emission and/or ejaculation.

Emission: deposition of seminal fluid components from the ampullary parts of the vasa deferentia, seminal vesicles, and prostate gland into the posterior urethra.

Ejaculation: passage of seminal fluid through the urethra and its expulsion from the urethral meatus.

Rapid or premature ejaculation: Inability to delay ejaculation sufficiently to enjoy lovemaking. Persistent or recurrent occurrence of ejaculation with minimal sexual stimulation before, on, or shortly after penetration and before the person wishes it.

Delayed ejaculation: undue delay in reaching a climax during sexual activity

Anorgasmia: Inability to achieve an orgasm during conscious sexual activity, although nocturnal emission may occur.

Anejaculation: Absence of ejaculation during orgasm.

Retrograde ejaculation: Backward passage of semen into the bladder after emission usually due to failure of closure of the bladder neck mechanism, demonstrated by presence of spermatozoa in the urine after orgasm.

I. PHYSIOLOGY OF NORMAL EJACULATION

1. PRODUCTION OF SEMEN

The spermatozoa are stored in the tails of the epididymides and the ampullary parts of the vasa, and they normally constitute less than 0.1 % of the semen volume. The ejaculate is produced by combining the secretions of the prostate with the contents of the ampullary parts of the vasa deferentia, followed by their expulsion from the urethra washed out by fluid from the seminal vesicles [1]. The normal ejaculate can be split into four to six fractions [2]. Serial biochemical analysis indicates that the first part contains the maximum number of spermatozoa, and subsequent fractions contain sequentially less. Acid phosphatase, citric acid and zinc, emanating from the prostate, are in highest concentration in the first part of the ejaculate, whereas fructose, coming from the seminal vesicles, increases in concentration towards the end of the ejaculatory process. Alteration of the pH values in successive parts of the split ejaculate indicates how the acid component provided by the prostate is serially mixed with the more alkaline contribution of the fructose rich fluid from the seminal vesicles. Approximately 15 - 30% of the entire ejaculate is contributed by the prostate, and
50 - 80% by the seminal vesicular secretion; there is, in addition, a small contribution to the first part of the ejaculate from the bulbo-urethral (Cowper’s) glands which is rich in enzymes and plasminogen activator [3].

2. PROPULSION OF EJACULATE

Propulsion of the ejaculate is obtained by contraction of the containing chamber (proximal urethra) combined with proximal closure of the bladder neck to prevent retrograde flow and distal urethral patency. Expulsion is achieved by rhythmic contractions of the bulbospongiosus and bulbocavernosus muscles, which forces the contents through the distal urethra. Intermittent contraction of the urethral sphincter prevents retrograde flow into the proximal urethra [4,5].

3. ORGASM

The orgasmic pleasure comprises 2 phases: the increase in tension in the prostatic urethra wall, and sensory stimuli arising in the area of the verumontanum. From that point the ejaculatory reflex cannot be blocked or delayed anymore. This is followed by the expulsive phase, when stimuli and information are sent to the central nervous system from the glans penis and the whole length of the urethra. In summary, a normal sensation of orgasm needs a rigid erection, the discharge of the tension from the orgasmic center and good coordination between the 2 phases of ejaculation (emission and expulsion). The exact mechanism of nocturnal ejaculation with or without orgasm during sleep is unknown. Several hypotheses have been suggested: autonomic activity of the spinal centers (ejaculation without orgasm), lowering of the neurosensory control of the brain, or autonomic discharge of the «condensers» - see below.

II. NERVOUS PATHWAYS AND AREA CONTROLLING EJACULATION AND ORGASM

The ejaculatory reflex comprises sensory receptors and areas, afferent pathways, cerebral sensory areas, cerebral motor centers, spinal motor centers and efferent pathways [6].

1. SENSORY RECEPTORS AND AREAS

The glans penis constitutes the primary erogenic area, where Krause-Finger corpuscles are located in the mucosa. These corpuscles act as sensory receptors and seem to function as condensers when repetitive and cumulative stimulation is applied to the glans penis during sexual intercourse. These corpuscles discharge as soon as a certain level of excitation is achieved. The sensory information is transmitted to the spine and the brain.

The external genital organs (penis and testicles) should be distinguished from the extragenital erogenous organs (these areas are highly variable from one subject to another). The stimulation of these secondary erogenous areas contributes to maintaining the erection and provides sensory information enhancing the information from the Krause-Finger corpuscles.

2. AFFERENT PATHWAYS

Sensory information from the glans penis travels along two different pathways: first, via the sensory fibres of the pudendal nerve (dorsal nerve of penis) up to S4. The afferent «volley» then travels into the spine. It has been demonstrated in the monkey that destroying the dorsal nerves of penis will abolish or delay ejaculation. Secondly, via the hypogastric plexus that transmits information to the ganglia of the paravertebral lumbo-sacral sympathetic chain. It seems that there is a close contact between these autonomic and cerebrospinal nervous pathways.

3. CEREBRAL RECEPTOR AREAS

Seminal emission and ejaculation are integrated into the complex pattern of copulatory behavior by forebrain structures that include the medial preoptic area (MPOA) and the paraventricular nucleus of the hypothalamus (PVN). The MPOA, immediately rostral to the anterior hypothalamus, is essential for male copulatory behavior in all vertebrate species that have been tested (reviewed in Meisel & Sachs, [7]). Electrical stimulation of the MPOA can elicit ex copula seminal emission or ejaculation in monkeys [8] and rats [9]. Moderate doses of a mixed D1/D2 dopamine agonist (apomorphine) [10] or of a D1 agonist (thienopyridine) [11], microinjected into the MPOA, promote
erections and facilitate copulation of male rats, apparently by increasing parasympathetic tone. Higher doses of a mixed D1/D2 agonist, or of a selective D2 agonist, shift the autonomic balance to favour seminal emission and ejaculation [11].

Dopamine is released in the MPOA of male rats in the presence of an estrous female, and increases more during copulation [12]. Thus, the levels of extracellular dopamine in the MPOA may regulate the phases of copulation, with high levels triggering ejaculation. Electrical stimulation of the MPOA also elicits the urethrogenital reflex in rats, which may mimic orgasm in humans [13]. This reflex is usually elicited in anesthetized, spinally transected rats by distending the urethra with saline and then suddenly releasing the pressure. This results in rhythmic firing of the hypogastric, pelvic, and motor pudendal nerves and rhythmic contractions of the perineal muscles, similar to those seen during orgasm in humans. However, stimulation of the MPOA elicited the reflex, even without genital stimulation.

There are no neurons that extend from the MPOA to the lumbosacral spinal cord; therefore, its facilitative effects must be mediated via other structures. One possible mediator is the periaqueductal gray (PAG) of the midbrain, which receives input from the MPOA and sends efferents to the lumbosacral spinal cord. Another possible mediator is the PVN. There are reciprocal connections between the MPOA and the PVN. Stimulation of mixed D1 and D2 receptors [10,14] or specifically of D2 receptors [15] in the PVN also increases the number of ex copula erections and seminal emissions. Neurons that contain a marker (neurophysin) associated with oxytocin descend from the PVN to the lumbosacral spinal cord [16], where they may elicit seminal emission/ejaculation.

Whereas dopamine, via D2 receptors, promotes seminal emission/ejaculation, serotonin is inhibitory. Serotonin is released in the anterior lateral hypothalamus (LHA) of male rats at the time of ejaculation [17]. Microinjection of a selective serotonin reuptake inhibitor (SSRI) into the LHA delayed both the onset of copulation (as though the male had just ejaculated) and also delayed ejaculation after copulation had begun [17]. This effect is similar to the reported side effects of SSRI antidepressant drugs, which include decreased libido and difficulty achieving ejaculation or orgasm. The postejaculatory decrease in libido may result in part from decreased dopamine release in the nucleus accumbens, a major terminal of the mesolimbic dopamine tract [18]. Dopamine in the nucleus accumbens has been related to motivation and/or reward related to numerous behaviors, including eating, drinking, copulation, and drug addiction. Therefore, one site at which SSRI drugs may inhibit both libido and ejaculation is the LHA. While the N. accumbens probably mediates the SSRI-induced decrease in libido, it probably does not influence ejaculation directly. The structure mediating that effect is not known; however, neurons from the LHA do descend to the lumbar spinal cord, where the neurons controlling genital reflexes reside.

Another major inhibitory influence on both erections and ejaculation is the nucleus paragiganto- cellularis (nPGi) in the ventral medulla, which tonically inhibits the spinal nuclei that program the motor output to the genitals and the pelvic musculature. Lesions of the nPGi facilitate the elicitation of the urethrogenital reflex and also reflexive penile erections and anteroflexions [19]. Approximately 78% of the descending neurons from nPGi are serotonergic [13]. Selective serotonin neurotoxin lesions depleted serotonin in the lumbosacral spinal cord and released the urethrogenital reflex from its tonic inhibition [20]. Spinal transection releases the spinal neurons from inhibition and allows the reflex to be elicited by urethral distension. Therefore, either a lesion of the nPGi (the site of serotonergic cell bodies) or spinal transection (cutting the descending axons) will release the spinal neurons from inhibition. It is interesting that stimulation of the MPOA can elicit the reflex, even if the nPGi and spinal cord are intact. This suggests that the MPOA may inhibit the nPGi, as well as stimulating an excitatory site.

The control of copulation and ejaculation is, in many ways, similar in humans and other mammals. The evolutionarily older brain areas that subserve these functions are highly conserved across species. Dopaminergic drugs enhance, and serotonergic drugs impair several measures of sexual behavior and ejaculation in humans, monkeys, and rats. Seminal emission is elicited by sympathetic innervation in all species that have been studied. One difference, however, is that rats
typically have much shorter postejaculatory intervals than do men. Indeed, one to three prior ejaculations actually increase the number of erections and shorten the interval before the next ejaculation in rats [8]. Possible treatments for ejaculatory dysfunction, due either to a primary disorder or secondary to SSRI antidepressants, may include administration of either D2 agonists or selective serotonin receptor antagonists. Ferrari and Giuliana [21] reported that a selective D2 agonist, systemically administered, produced "premature ejaculation," which was counteracted by a D2 antagonist. On the other hand, administration of serotonin 5-HT1B or 5-HT2 antagonists may reverse the effects of serotonergic inhibition from either the LHA or the nPGI.

4. SPINAL MOTOR CENTERS

A "secretory center" is located at the Th12-L1-L2 spinal level. It is controlled by the sympathetic nervous system and is responsible for emission. A "mechanical center" is located at the S2-S4 level. It is controlled by the somatic nervous system and is responsible for expulsion.

5. EFFERENT PATHWAYS

The efferent sympathetic nerves emerge from the spinal column at Th12-L2 to form the lumbar sympathetic ganglia from which the descending nerves encircle the aorta on each side before coming together in the midline to form the hypogastric plexus just below the bifurcation of the aorta. From there the preganglionic sympathetic fibres conveyed by hypogastric nerves pass through the pelvis to synapse with postganglionic neurons in the pelvic plexus which terminate on the bladder neck, prostate, seminal vesicles and vasa deferentia [22]. The efferent somatic fibres emerge from the anterior horn of the S2-S4 spinal segments (Onuf's nucleus), they travel in the motor branch of the pudendal nerve to innervate the pelvic floor muscles including the bulbospongiosus and bulbocavernosus muscles.

a) Anatomy

The abdominal and pelvic sympathetic nervous system fundamentally shows a common structure in mammals including humans (Fig. 1). The cell bodies of the preganglionic sympathetic neurons are located in the lateral columns of the gray matter in the thoracolumbar segments of the spinal cord. Sympathetic nerve fibers exit the spinal cord via the ventral roots and reach the sympathetic chains bilaterally. The nerves proceed via the thoracic sympathetic chain to the caudal (inferior) enteric plexus, the major/minor splanchnic nerves, the celiac/cranial mesenteric plexuses, and the intermesenteric nerves. The nerves proceed via the lumbar sympathetic chain and the lumbar splanchnic nerves to the caudal mesenteric plexus.

In animals, the intermesenteric nerves and all lumbar splanchnic nerves merge into one plexus, the caudal mesenteric plexus, from which the colonic nerve and paired hypogastric nerves exit to the colon and the pelvic plexus, respectively (Fig. 2A). The caudal mesenteric plexus in animals corresponds to two plexuses, whereas in humans, the inferior mesenteric and superior hypogastric plexuses are separate [22] (Fig. 2B). The former plexus mainly innervates the colon and from the latter spring paired hypogastric nerves. The junction of the hypogastric nerve and the pelvic nerve constitutes the pelvic plexus in the pelvis, which is an integration of sympathetic and parasympathetic nervous systems. The branches from this plexus innervate the epididymis, vas deferens, seminal vesicle, prostate, bladder neck and urethra (Figs. 1 and 2). The pudendal nerve emanates from the sacral spinal cord and does not enter the pelvic plexus, but exits the pelvis through the greater sciatic foramen, reenters it through the lesser sciatic foramen, and innervates the perineal striated muscles (Fig. 1).

b) Neurophysiology

Application of retrograde axonal tracing methods to the vas deferens [23] revealed that the majority of postganglionic neurons distributed in the vas deferens originate from the pelvic plexus. The pelvic plexus receives neural input from both the hypogastric and pelvic nerves (Fig. 2). Electrical stimulation of the hypogastric nerve elicited contraction of the vas deferens in many mammals [24, 25], while stimulation of the pelvic nerve caused no detectable motor responses [25, 26]. Histochemical studies of the vas deferens have also shown that the adrenergic fibers mainly innervate the smooth muscle layers, whereas cholinergic ones chiefly innervate the subepithelial layer [4]. Stimulation of the hypogastric nerve has also elicited contraction of the bladder neck and
Figure 1: Diagram showing the nervous system controlling ejaculation. Emission from both the ejaculatory orifice and prostate, and bladder neck closure are controlled by the signals from the pelvic plexus. The pudendal nerve principally controls propulsion out of the urethra. The sacral splanchnic nerve (SSN) is found in about 30-50% of humans. BCM: bulbocavernosus muscle, Epi: epididymis, EUS: external urethral sphincter, HGN: hypogastric nerve, LSN: lumbar splanchnic nerve, PuN: pudendal nerve, PN: pelvic nerve, SC: spinal cord, SHP/CMP: superior hypogastric plexus in humans/caudal mesenteric plexus in animals, SV: seminal vesicle, SyC: Sympathetic chain.

Figure 2: Diagram showing the common sympathetic pathways controlling ejaculation (black) in mammals (A). The caudal mesenteric plexus in animals is divided into two plexuses in humans, inferior mesenteric plexus (IMP) and superior hypogastric plexus (SHP) (B). The pathways in blue is of minor importance in motor activity of the seminal tract. CM: cranial mesenteric, CMA: caudal mesenteric artery, CoN: colonic nerve, InMP: intermesenteric plexus. See Fig. 1 legend for additional abbreviations.
prostate as well as the vas deferens. On the proximal pathway to the hypogastric nerve, stimulation of the lumbar splanchnic nerve caused contraction of the vas deferens, prostate and bladder neck, whereas that of the intermesenteric nerve elicited no observable responses in any of those organs in many mammals [27].

The levels of the lumbar splanchnic nerves which elicited motor activity of the seminal tract were 2nd-5th in rats [27] and dogs, and 1st-3rd in humans [28,29]. Recent human anatomical study has revealed that almost all the lumbar splanchnic nerves originate from L2 and/or L3 lumbar sympathetic ganglia (corresponding to L1-2 spinal levels) [22]. Clinically, preservation of the L2 and/or L3 lumbar splanchnic nerve in retroperitoneal lymph node dissection of testicular cancer demonstrated restoration of ejaculatory function [29]. Partial and complete interruption of the pathway from the spinal cord to the seminal tract might cause retrograde ejaculation and emission loss, respectively. Partial inhibition might cause insufficient closure of bladder neck that permits the partially emitted seminal fluid to flow back into the bladder.

The cross-innervation of the peripheral sympathetic nervous system, which has been suggested from its architecture (Fig. 2), has been revealed in the dog and rat [27]. On the way of the common pathway from the lumbar splanchnic nerve to the seminal tract, some signals cross to the other side of the body at the level of the caudal mesenteric plexus and/or the pelvic plexus (Fig. 3). The pre-ganglionic axons passing through the hypogastric nerve very likely provide a bilateral innervation to postganglionic neurons in the pelvic plexuses, which also exhibit crossing to the bilateral vasa deferentia [27]. A similar pattern of multiple cross-innervation has also been identified in the rodent bladder neck.

When the sympathetic signals passing through the above pathway reach the seminal tract, norepinephrine is released from the terminal of the postganglionic neuron. Norepinephrine induces activation of alpha 1-adrenergic receptors on the smooth muscle cells, which elicits a rise in cytosolic calcium and results in the actin-myosin interaction. A combination of adrenergic and purinergic (ATP) mechanisms are necessary for contraction of the animal vas deferens, while norepinephrine is most probably the only significant neurotransmitter for contraction of human vas deferens. Transmitter release, or the basal tone of the smooth muscle of the vas deferens, might be variably modulated by many substances, i.e. acetylcholine, neuropeptide-Y. Once contraction of the smooth muscle of the seminal tract occurs, marked elevation of intraluminal pressure might occur at the cauda epididymis/proximal vas, which might push the spermatozoa out to the ampulla. Both nerve signal and distention of the wall of the ampulla might trigger contraction of the ampulla to emit the content into the posterior urethra.

When the common pathways described above are interrupted, the occurrence of compensatory mechanisms such as enhancement of the remaining sympathetic pathways and reorganization of synaptic connection in the pelvic plexus has been reported. After transection of the canine hypogastric nerve, surgical reconstruction is possible and cross-innervation through the hypogastric nerve described above can also be preserved [30].

The sympathetic nerves reaching the adrenal medulla via the thoracic sympathetic chain and the major/minor splanchnic nerves may have a possibility of affecting ejaculation through hormonal system. Catecholamines secreted from the adrenal medulla can elicit similar systemic reactions as those accompanying ejaculation, such as marked elevation of blood pressure, tachycardia, tachypnea and perspiration as well as local ejaculatory reactions.

Propulsion of the seminal fluid is caused by rhythmic contractions of the perineal striated musculature including the bulbocavernosus and ischiocavernosus muscles. Such muscles are innervated by the pudendal nerve and show excitement during ejaculation. The patients with sacral cord injuries usually show dribbling ejaculation due to the lack of contribution of the musculature. The peripheral nervous system controlling ejaculation has elaborate mechanisms for preserving its function against various injuries.
Figure 3: Diagram showing bilateral sympathetic efferent pathways projecting from a lumbar splanchnic nerve to the vasa deferentia on both sides. Four routes and two points of crossing to the other side are indicated. Similar cross-innervation is present in bladder neck and prostate. See Fig. 1 legend for abbreviations.
Neurophysiological tests allow objective evaluation of the nervous pathways controlling ejaculation. Four tests are routinely used.

1. **Pudendal Somatosensory Evoked Potentials (Pudendal SEPs)**

Somatosensory evoked potentials (SEPs) are defined as a transient alteration of the electroencephalogram (EEG) following peripheral nerve stimulation. They provide objective information concerning the afferent volley from the dorsal nerve of penis to the cortex. The technique consists of electrical stimulation of the dorsal nerve of penis with recording of the evoked responses over the spine and the scalp (2 cm behind the central vertex) (Fig. 4). First the sensibility threshold is measured. By definition, the sensibility threshold is the lowest perceivable sensation of the electrical current at the point of stimulation. The latency of the response is measured both at the onset of the response and the peak of the first reproducible deflection. By recording the response at 2 different levels, 3 different transit times are obtained: a total transit time (from penis to brain), a peripheral transit time (from penis to spine), and a central transit time (which is obtained by subtracting the peripheral from the total transit time). The peripheral transit time is approximately 13.5 ms. The total transit time is approximately 34 msec (onset) and 43 msec (top of P1 deflection) [31,32].

2. **Pudendal Motor Evoked Potentials (Pudendal MEPs)**

Motor Evoked Potentials (MEPs) explore the efferent pathways (pyramidal tracts) from brain to target muscle (bulbocavernous muscles). The technique consists of stimulating the motor cortex and sacral roots by means of a magneto-electric stimulator. For brain stimulation, the coil is applied 2 cm behind the vertex (Fig. 5). For sacral root stimulation, the coil is applied laterally to the spine. The response is picked up from the bulbocavernous muscles with co-axial EMG needle electrodes. Brain stimulation is performed, first at rest, and then during a voluntary contraction of the pelvic floor (facilitation procedure). Sacral root stimulation is performed only at rest. The response is measured at the onset of the first reliable deflection. By stimulating the central nervous system at 2 levels, 3 different transit times will be obtained: a total transit time (from brain to target muscle), a peripheral transit time (from sacral roots to target muscle) and a central transit time (obtained by subtracting the peripheral from the total transit time) (Fig 6). The total transit time measured in the bulbocavernous muscles is respectively 28 msec (brain stimulation patient at rest) and 23 msec (brain stimulation patient contracting the pelvic floor). The peripheral transit time is 7 msec (sacral root stimulation) [32].

3. **Sacral Reflex Arc Testing: The Somato-Somatic Reflex Arc**

The test allows the investigation of the sensory and motor branch of the pudendal nerve and of the sacral segments S2, S3, S4. The technique consists in stimulating the dorsal nerve of the penis and recording the response from the bulbocavernous muscles. The response consists usually of 2 deflections. The mean latency of the first deflection is 35 msec, although a late deflection is often observed at 80 msec [32,33].

4. **Sympathetic Skin Responses (SSRs)**

Electrical activity from the sympathetic nerve terminals controlling the sweat glands of the skin can be recorded following electrical stimulation of any peripheral nerve trunk. The test allows evaluation of the sympathetic efferent outflow to the skin of the genital organs. The dorsal nerve of the penis is stimulated using 2 ring electrodes wrapped around the penile shaft, the cathode being proximal. The stimulation consists of single electrical pulses applied at a rate of 0.05 Hz. Sympathetic skin responses are recorded from hand, foot, and perineum using disc electrodes affixed to the skin. Two tracings are superimposed to check the reproducibility of the response. The right median nerve is then stimulated, and SSRs are recorded from the hand, foot, perineum, and penis. The mean latency of hand, foot, and perineum SSRs following dorsal nerve of the penis stimulation are, respectively, 1.40 sec, 2 sec, and 1.4 sec. Following median nerve stimulation, the latency of penile SSRs is 1.50 sec [34,35].
Figure 4: Pudendal Somatosensory Evoked Potentials (Pudendal SEPs). The response is recorded 2 cm behind the central vertex. The latency of the first positive deflection (P1) is 38 msec.

Figure 5: Pudendal Motor Evoked Potentials (Pudendal MEPs): sites of stimulation and position of the coil.
A: Transcranial magnetic stimulation: the posterior edge of the coil is applied 2 cm behind the frontal vertex
B: Sacral root magnetic stimulation: the coil is applied laterally to the spine at the level of the iliac crest (from R.J. Opsomer et al, 1992, with permission for reproduction of Peeters Publishers [94])

Figure 6: Pudendal Motor Evoked Potentials (Pudendal MEPs) recorded from the periurethral sphincter in a normal subject. A: Transcranial magnetic stimulation. Subject at rest. Latency of the response is 21.5 msec. B: Transcranial magnetic stimulation. Latency of the response in 30 msec. Notice the increase in amplitude and shortening of the latency of the response when the patient contracts the pelvic floor: Facilitation procedure. C: Sacral root magnetic stimulation. Latency of the response is 7.7 msec (from R.J. Opsomer et al, 1992, with permission for reproduction of Peeters Publishers [94])
1. EMBRYOLOGY AND CONGENITAL ANOMALIES

As the male foetus develops, the Mullerian ducts normally disappear from above downwards under the influence of Mullerian inhibitory factor (MIF) which is produced by the Sertoli cells in the primitive testis. Failure of complete absorption may leave a small Mullerian duct remnant at the lower end that lies between the ejaculatory ducts. The Wolffian (mesonephric) ducts are composed of three distinct areas. The upper part forms the epididymis and distal vas deferens, while the proximal vas deferens, seminal vesicle and ejaculatory duct are derived from the middle area. The most caudal part is the common mesonephric duct, from which the ureteric bud springs at approximately 4 weeks of development: this becomes the ureter, and will induce the metanephric blastema to form the kidney. The urogenital sinus reabsorbs the lower end of this structure, and the ureteric orifices are thus separated from the vasa deferentia, seminal vesicles and ejaculatory ducts. Several complex anomalies may occur in this area leading to ectopic opening of the vas deferens and sometimes associated with anorectal anomalies [36]. If too much of the proximal vas precursor is absorbed, a variable amount of the proximal vas, seminal vesicle and/or ejaculatory duct may be absent. There may also be coexisting abnormalities in the ipsilateral kidney or ureter.

a) Mullerian duct cyst

Persistence of a small remnant of the Mullerian duct may lead to a cyst forming between the ejaculatory ducts which can become obstructed and cause diminution of the volume of the ejaculate and infertility. Haemospermia is not uncommon in these patients. Seminal analysis shows the changes characteristic of ejaculatory duct obstruction with a small volume (less than 1.5 mL), acid pH and little or no fructose. Both vasa are palpable and the epididymes usually feel distended. The diagnosis is established by transrectal ultrasound scan (TRUS), and the lesion can be delineated by percutaneous puncture of the cyst with instillation of radio-opaque medium (figure 7). The cyst can be incised or deroofed endoscopically after deline-
ting its extent by injection of blue dye (see below). Improvement in ejaculate volume and seminal quality follows in most cases [37].

b) Wolffian duct abnormalities

Congenital anomalies may be either sporadic, with a localized defect in the proximal part of the vas deferens or there may be a generalized maldevelopment due to a systemic genetic abnormality. Local Wolffian duct abnormality involves loss of a variable amount of the vas deferens, seminal vesicle and/or ejaculatory duct, and sometimes part of the ipsilateral urinary system as well. This may be associated with maldevelopment of the bladder neck and trigone, which fails to close effectively producing retrograde ejaculation.

Bilateral abnormalities are often associated with carriage of the cystic fibrosis gene [38]. Unilateral absence of the vas deferens was observed in 5%, and bilateral absence in 18% of 370 azoospermic males with normal serum FSH levels investigated by the author [39].

c) Prune belly syndrome

Patients with Prune Belly syndrome have normal libido, erections, and orgasms. Most have abnormal ejaculation and probably emission. In a study involving nine patients, seven had retrograde ejaculation and two produced ejaculae [40]. Five patients provided semen or urine passed after masturbation. Two produced ejaculated semen. One of the ejaculated specimens consisted of 4.5 cc of fluid indistinguishable from urine and one was 2.5 cc of fluid with the appearance of watery semen. Post masturbation urine specimens were of normal urinary appearance. None of the specimens contained sperm: no mention was made of the fructose content. Abnormal ejaculation thus appears to be present in the vast majority of patients with Prune Belly syndrome. Whether the primary abnormality is retrograde ejaculation or lack of emission is not clear.

2. TRAUMATIC DAMAGE

a) Imperforate anus

Ejaculatory duct obstruction may follow correction of imperforate anus. The pull through procedure passes close to the posterior aspect of the prostate, and damage is most likely if there has been closure of a recto-urethral fistula. Analysis of 20 subfertile males who had repair of imperforate anus in infancy indicated that 7 had no ejaculate, 11 were azoospermic, 1 was severely oligozoospermic and only 1 had a normal sperm concentration in a very small volume ejaculate [41]. Investigation revealed that both vasa were blocked in 5 men and one vas in a further 8 patients, apparently as a result of the original operative procedure.

b) Operations on the prostate

Antegrade ejaculation requires a closed bladder neck (and proximal urethra). Surgical procedures that compromise the bladder neck closure mechanism may result in retrograde ejaculation. Transurethral incision of the prostate (TUIP) results in retrograde ejaculation in 5% [42] to 45% [43] of patients and is probably related to whether one or two incisions are made and whether or not the incision includes primarily the bladder neck or extends to the level of the verumontanum.

The importance of contraction of the urethral smooth muscle at the level of the verumontanum has been hypothesized to be important in preventing retrograde ejaculation [42]. Transurethral resection of the prostate (TURP) carries a higher incidence of retrograde ejaculation than does TUIP. The reported incidence of retrograde ejaculation following TURP ranges from 42% [44] to 100% [45]. It occurs less frequently following open prostatectomy (either suprapubic or retropubic) then after TURP. In one series, the incidence of retrograde ejaculation following open prostatectomy was zero [44]. TURP is thought to disrupt the closure mechanism of the vesical neck, whereas open enucleation is less apt to produce this alteration.

After radical prostatectomy, ejaculation is bound to be lost since the seminal vesicles are removed with the prostate gland. Erectile impotence was the rule until detailed anatomical studies showed where the parasympathetic nerves ran on the surface of the prostate gland, and a nerve sparing operative technique was developed [46]. A sensation of orgasm can sometimes be preserved despite loss of ejaculation.

3. INFECTIVE DISORDERS

Genital infection such as gonorrhoea or non-specific urethritis can produce cicatrisation and obs-
striction anywhere in the male reproductive tract, especially if treatment is delayed. Urinary infection, especially if complicated by epididymitis, can also produce obstruction that may be situated at ejaculatory duct level. Routine vasography in subfertile men with azoospermia and normal serum FSH levels revealed post-infective vasal blocks in 8% and acquired ejaculatory duct obstruction in 4% [39].

Schistosomiasis is endemic in large parts of Africa, and is seen with increasing frequency in tourists returning from Africa who have contracted the disease whilst enjoying water sports: Lake Malawi has acquired an evil reputation in this respect. The disease may present with haematospermia [47] and fibrosis and calcification may lead to genital obstruction.

Genito-urinary tuberculosis can cause great damage to the male reproductive tracts, and since healing occurs with calcification, the lesions may be irreparable. Plain X-ray will often show the extent of the disease.

Haematospermia is seldom as ominous a symptom as haematuria, but this complaint should not be ignored. Analysis of the findings in 81 patients revealed that an inflammatory cause could be defined in most men under 30 years of age; however, there were a few (8%) with more serious disease including carcinoma of prostate and bladder [48]. It should be remembered, also, that schistosomiasis and tuberculosis could present in this way. Routine investigation of haematospermia by TRUS not uncommonly reveals the presence of small stones in the ejaculatory ducts, which may be associated with obstruction and dilatation of the seminal vesicles. Such stones usually pass spontaneously.

4. Neurological Disorders

a) Spinal cord injury

Damage to the spinal cord at the level of T12 to L2 may affect central reflex pathways and lead to permanent loss of ejaculation. Injury above T11 may allow reflex erection and ejaculation, although this can provoke autonomic dysreflexia with marked rise in blood pressure. In some paraplegic patients, application of a vibrator to the penis will lead to ejaculation; in others, electroejaculation may be necessary to produce spermatozoa that can be used for insemination. If the spinal reflex arc is intact, a hypogastric plexus stimulator can provide ejaculation in the comfort and security of the patients' home [49]. Alternatively, direct electroejaculation by rectal probe may be effective, but this may require a general anaesthetic and is done in hospital [50]. In a recent collective analysis of 40 paraplegic patients, 22 successfully produced pregnancies by natural insemination or assisted reproductive techniques [51].

Orgasm has been noted to occur in men with spinal cord injuries. Via self-report 42% to 61% of men reported the ability to achieve orgasm. Orgasms were also noted to occur in men with complete spinal cord injuries; however, overall orgasms were described as different than prior to their injuries. No laboratory-based analysis has been performed of the physiologic events occurring during orgasms in the male with spinal cord injury [52].

b) Para-aortic lymphadenectomy

This operation is usually done to clear lymph node metastases from testicular tumours, when the sympathetic nerves and ganglia may also be removed leading to loss of ejaculation. Early studies showed that up to three-quarters of patients lost antegrade ejaculation after full bilateral retroperitoneal lymph node dissection. As a result of careful anatomical studies, the technique of retroperitoneal lymph node dissection has been modified with nerve sparing so that antegrade ejaculation is now maintained in 70-90% of patients.

One quarter of the patients who complete chemotherapy for advanced testicular tumour have residual masses in the para-aortic region [53]. Amongst 231 consecutive patients undergoing para-aortic lymphadenectomy after chemotherapy at the Royal Marsden Hospital, there was persistent undifferentiated tumour in 21% [54]. In our experience of 186 patients, a nerve sparing operative technique introduced in 1984 led to a significant reduction in ejaculatory dysfunction from 37% to 19% [55]. Loss of ejaculation occurred significantly more often after bilateral (46%) compared to unilateral (14%) dissection, and was related to the size of the excised mass (<4 cm 4%; 4-8 cm 19%; >8 cm 60%).
It is important to anticipate this complication in young men with testicular tumours who may need chemotherapy or node dissection, and arrangements should be made for sperm storage before treatment commences. Excellent results can be obtained with artificial insemination using cryopreserved spermatozoa [56].

5. THE EFFECTS OF DRUGS ON ORGASM AND EJACULATION

a) Animal studies

Ever since the late sixties, serotonin (5-HT) has been known for its involvement in male rat sexual behaviour. It is generally assumed that central 5-HT has an inhibitory role in the neural control of masculine sexual behaviour in the rat. A decrease in 5-HT neurotransmission decreases the number of intromissions preceding ejaculation and shortens the time to ejaculation, whereas an increase in central 5-HT neurotransmission produces the opposite effect [57]. Administration of the selective 5-HT1A receptor agonist 8-OH-DPAT lowers 5-HT levels in some parts of the brain, and causes male rats to ejaculate at the first or second intromission and within seconds after being put in the vicinity of an estrous female rat. Therefore, it could be stated that 8-OH-DPAT renders male rats to be premature ejaculators [58]. Administration of different selective serotonin reuptake inhibitors (SSRIs), which results in higher levels of 5-HT, suppresses sexual behavior in male rats [59].

b) Human studies

In humans, the side effects of antidepressants on sexual function have been known for more than 25 years. In general, these substances increase the 5-HT concentration in the synapses, usually by uptake inhibition (e.g., SSRIs) [95]. The most commonly reported side effects are delay or absence of orgasm/ejaculation. In 1973, the English psychiatrist Eaton was the first to report on the beneficial aspects of this side effect. He administered the tricyclic antidepressant clomipramine to men with premature ejaculation without psychiatric disorders [60]. To date, many studies have been performed to investigate the effects of fluoxetine, paroxetine, sertraline (SSRIs) and clomipramine [61]. Table 1 shows the results of the most relevant studies [62-75]. Although no studies with long-term treatment with SSRIs have been published, it has become an accepted treatment for premature ejaculation.

Since psychiatric drugs and physical conditions like erectile dysfunction can cause secondary premature ejaculation, it is important to exclude and/or treat these causes before treating the symptom of premature ejaculation. For example, when a man is able to achieve a rigid erection, but not to maintain this erection for a certain amount of time, he could condition himself to ejaculate rapidly. It is very likely that his secondary premature ejaculation will be successfully treated by oral sildenafil or local vasoactive drugs, and not by SSRIs.

c) Side effects of specific drugs on ejaculation

1) Dopamine

The centrally acting neurotransmitter dopamine is known for its involvement in control of male rat sexual behavior. Taking the parameters of mounts and intromission frequencies and latency to ejaculation as measures of copulatory activity, most reports indicate that dopamine has a stimulatory effect that is exerted via D2 receptors. Enhancement of the ejaculatory behavior and the decrease in intromission frequency stimulated some authors to call this altered behavior a rat model for "premature ejaculation".

2) Morphine

Several studies have shown that systemic and central administration of morphine inhibits male rat sexual behavior. However, in one study [76], a small proportion of male rats reacted differently on a low dose of systemic morphine: there was a decrease of ejaculation latency, and in the number of intromissions prior to ejaculation. These conflicting results indicate that at least there is a role for the enkephalines in the modulation of sexual behavior in the male rat.

3) Ecstasy

The amphetamine analog MDMA, better known as the recreational drug ecstasy, is known and feared for its neurotoxic properties. It reduces brain concentrations of serotonin by inhibition of the metabolism and by long-lasting degeneration of 5-HT nerve terminals, as well as by decreasing the number of 5-HT uptake sites. In an experiment with male rats, Dornan and collaborators [77] found that a chronic administration of MDMA, caused less rats to display mounting behavior, and an increase in ejaculation latency in the responders. These results are conflicting with the above-described studies with serotonin receptor agonists.
<table>
<thead>
<tr>
<th>AUTHOR(S) &amp; REFERENCE</th>
<th>N</th>
<th>DOSE</th>
<th>STUDY DESIGN*</th>
<th>EFFECT/REMARKS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>CLOMIPRAMINE</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Segraves et al [62]</td>
<td>20</td>
<td>25-50 mg/day</td>
<td>DB, PC, crossover</td>
<td>placebo: 51 sec; clomipramine: 366 sec</td>
</tr>
<tr>
<td>Althof et al [63]</td>
<td>15</td>
<td>25-50 mg/day</td>
<td>DB, PC, crossover</td>
<td>baseline: 81 sec; clomipramine: 25 sec; 50 mg: 202 sec; 100 mg: 416 sec partner’s satisfaction included</td>
</tr>
<tr>
<td>Haensel et al [64]</td>
<td>22</td>
<td>25 mg 12-24 h prior to sexual activity</td>
<td>DB, PC, crossover</td>
<td>placebo: 2 min; clomipramine: 8 min; include controls: 9-11 min; placebo: 52 sec; clomipramine: 229 sec include controls: 8-11 min</td>
</tr>
<tr>
<td>Stassberg et al [65]</td>
<td>34</td>
<td>25 mg 4-6 h prior to sexual activity</td>
<td>DB, PC, crossover</td>
<td>placebo: 30-60 sec; fluoxetine: 15-180 sec; baseline: 0.9 min; fluoxetine: 9.6 min</td>
</tr>
<tr>
<td><strong>FLUOXETINE (SSRI)</strong></td>
<td></td>
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<tr>
<td>Kara et al [66]</td>
<td>14</td>
<td>20-40 mg/day</td>
<td>DB, PC</td>
<td>placebo: 30-60 sec; fluoxetine: 25-180 sec; baseline: 0.9 min; fluoxetine: 9.6 min</td>
</tr>
<tr>
<td>Lee et al [67]</td>
<td>11</td>
<td>20-60 mg/day</td>
<td>Open label</td>
<td>increase in ejaculation latency (p=0.007); controls: no effect</td>
</tr>
<tr>
<td>Haensel et al [68]</td>
<td>40</td>
<td>5-10 mg/day</td>
<td>DB, PC, crossover</td>
<td>placebo: 0.3 min; placebo: 0.45-0.6 min; paroxetine: 3.2-3.5 min</td>
</tr>
<tr>
<td><strong>PAROXETINE (SSRI)</strong></td>
<td></td>
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<td></td>
<td></td>
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<tr>
<td>Waldinger et al [69]</td>
<td>17</td>
<td>20-40 mg/day</td>
<td>DB, PC</td>
<td>baseline: 0.5 min; 20 mg: 7.5 min; 40 mg: 10 min; placebo no effect</td>
</tr>
<tr>
<td>Waldinger et al [70]</td>
<td>27</td>
<td>20-40 mg/day</td>
<td>DB, dose response</td>
<td>20 mg/day: 13-300 sec; 40 mg/day: 10-540 sec</td>
</tr>
<tr>
<td>McMahon and Touma [71]</td>
<td>26</td>
<td>20 mg 3-4 h prior to sexual activity</td>
<td>SB, crossover</td>
<td>baseline: 0.3 min; placebo: 0.45-0.6 min; paroxetine: 3.2-3.5 min</td>
</tr>
<tr>
<td><strong>SERTRALINE (SSRI)</strong></td>
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<td></td>
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<tr>
<td>McMahon [72]</td>
<td>37</td>
<td>50 mg/day</td>
<td>SB, PC, crossover</td>
<td>baseline: 0.3 min; placebo: 0.5 min; sertraline: 3.2 min; baseline: 1 min, 25 mg: 7.6 min; 50 mg: 13.1 min, 100 mg: 16.4 min</td>
</tr>
<tr>
<td>McMahon [73]</td>
<td>46</td>
<td>25-100 mg/day</td>
<td>Open label</td>
<td></td>
</tr>
<tr>
<td><strong>COMBINED</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Waldinger et al [74]</td>
<td>51</td>
<td>Fluoxetine: 20 mg/day</td>
<td>DB, PC</td>
<td>baseline: 18 sec; placebo: 29 sec; fluoxetine: 55 sec; sertraline: 117 sec; fluoxetine: 211 sec; paroxetine: 476 sec</td>
</tr>
<tr>
<td>Kim et al [75]</td>
<td>36</td>
<td>Fluoxetine: 40 mg/day</td>
<td>DB, PC, crossover</td>
<td>baseline: 46 sec; placebo: 2.3 min; fluoxetine: 2.3 min; sertraline: 4.3 min; clomipramine: 5.8 min</td>
</tr>
</tbody>
</table>

**KEY:** DB - double blind; SB - single blind; PC - placebo controlled

Derived in part from: Rowland et al. [61] with permission of the author

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and antagonists, because a decrease in central 5-HT would cause an increase in male rats' sexual behaviors. Probably, since MDMA has such dramatic effects in the brain, other factors may have played an important role in this experiment.

GABA. The neurotransmitter gamma-aminobutyric acid (GABA) occurs in the brain tissue. Two distinct types of GABA receptors are recognized: GABAA and GABAB. There is some evidence that the GABAB receptor agonists (like baclofen) inhibit sexual behavior in male rats, independently from the effects on motor systems. Efforts to discover a role for GABAA in the modulation of sexual behavior in the rat have failed so far.

4) Yohimbine.
The alpha2-adrenoceptor blocking agent yohimbine has been known for its aphrodisiac properties in rats and humans. In male rat studies, it increased mounting behavior without the need for physiological levels of serum testosterone. When looking at the effects on ejaculation, a decrease in ejaculation latency, intercopulatory interval, and post-ejaculatory interval is found.

d) Specific drug effects in human studies
1. Monoamine oxidase inhibitors.
The monoamine oxidase inhibitors (MAOIs) are mainly used in the treatment of neurotic or atypical depression. These drugs increase the levels of epinephrine, norepinephrine, dopamine and serotonin. The MAOIs have been known for their sexual side effects, with an incidence up to 20-40%. Delayed or inhibited ejaculation is reported for isocarbazid, phenelzine and tranylcypromine.

2. Cyproheptadine
It is an antihistaminic, formerly used in Cushing's disease and anorexia nervosa. It also increases serotonin levels in the brain. Several reports indicate that cyproheptadine is able to convert drug-induced orgasmic failure in both men and women.

3. Benzodiazepines
A number of benzodiazepines effective in treating generalized anxiety and panic attacks are also known to inhibit ejaculation in some men, presumably by enhancing gamma-aminobutyric acid (GABA). These drugs include diazepam, lorazepam, lorazepam, temazepam, flunitrazepam, flurazepam, nitrazepam, chlordiazepoxide, and alprazolam.

4. Stimulants
Amphetamine is a stimulating drug with affinity for different receptors in the central nervous system. It stimulates release of dopamine, inhibits monoamine oxidase and blocks the reuptake of both catecholamines and serotonin. It is reported to delay ejaculation in subjects without ejaculatory dysfunction.

Cocaine is an addictive "recreational" drug and stimulates the central nervous system through blocking of monoamine transporters. Different reports confirm that delayed ejaculation appears to be the most common sexual side effect. The influences of different drugs on ejaculation are delineated in table 2 [78].

6. Functional Disorders
• Seminal megavesicles
Adult polycystic kidney disease has been found in association with pathological dilatation of the seminal vesicles in 6 patients [79]. TRUS and percutaneous puncture of the seminal vesicles before and after resection of the ejaculatory ducts revealed that the gross dilatation of the seminal vesicles was not caused by obstruction, but appeared to be due to atonicity (megavesicles). These ultrasonic appearances, when described previously, were incorrectly thought to be due to seminal vesicle cysts. Pathological dilatation of the seminal vesicles in the absence of obstruction has been described previously, although the aetiology remains obscure [80].

V. INVESTIGATION

1. Evaluation of Patients with Rapid/Premature Ejaculation
a) Diagnostic criteria
The diagnostic criteria for rapid or premature ejaculation have been defined as follows:

• DSM-IV- Premature Ejaculation
  1) Persistent or recurrent ejaculation with minimal sexual stimulation before, on, or shortly after penetration and before the person wishes it. The clinician must take into account factors that
Table 2: Side effects of different drugs on ejaculation

<table>
<thead>
<tr>
<th>GROUP OF DRUGS</th>
<th>AGENT</th>
<th>EFFECT ON EJACULATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>ANALGESICS</td>
<td></td>
<td></td>
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<tr>
<td>NSAID'S</td>
<td>Naproxen</td>
<td>Decrease/absence of ejaculation/orgasm</td>
</tr>
<tr>
<td>Opioids</td>
<td>Methadone</td>
<td>Decrease/absence of ejaculation/orgasm</td>
</tr>
<tr>
<td></td>
<td>Morphine</td>
<td>Decrease/absence of ejaculation/orgasm</td>
</tr>
<tr>
<td>ANTIHYPERTENSIVES</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alpha blockers</td>
<td>Phenoxybenzamine</td>
<td>Decrease/absence of ejaculation/orgasm Retrograde ejaculation</td>
</tr>
<tr>
<td>Alpha and beta blockers</td>
<td>Labetolol</td>
<td>Decrease/absence of ejaculation/orgasm</td>
</tr>
<tr>
<td>Centrally acting</td>
<td>Clonidine</td>
<td>Decrease/absence of ejaculation/orgasm Retrograde ejaculation</td>
</tr>
<tr>
<td></td>
<td>Guanethidine</td>
<td>Decrease/absence of ejaculation/orgasm Retrograde ejaculation</td>
</tr>
<tr>
<td></td>
<td>Methyl/dopa</td>
<td>Decrease/absence of ejaculation/orgasm</td>
</tr>
<tr>
<td></td>
<td>Reserpine</td>
<td>Decrease/absence of ejaculation/orgasm</td>
</tr>
<tr>
<td>ANTI-PARKINSONISM AGENTS</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Bromocriptine</td>
<td>Decrease/absence of ejaculation/orgasm Decrease &amp; increase reported Spontaneous ejaculation</td>
</tr>
<tr>
<td></td>
<td>L-DOPA</td>
<td></td>
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<tr>
<td></td>
<td>Pergolide</td>
<td></td>
</tr>
<tr>
<td>APPETITE SUPPRESSANTS</td>
<td>Mazindol</td>
<td>Spontaneous ejaculation</td>
</tr>
<tr>
<td>CYTOTOXICS</td>
<td>Methotrexate</td>
<td>Decrease/absence of ejaculation/orgasm</td>
</tr>
<tr>
<td></td>
<td>Vincristine</td>
<td>Decrease/absence of ejaculation/orgasm</td>
</tr>
<tr>
<td>HORMONE ANTAGONISTS</td>
<td>Cyproterone acetate</td>
<td>Decrease/absence of ejaculation/orgasm</td>
</tr>
<tr>
<td>HYPNOTICS AND SEDATIVES</td>
<td>Barbiturates (all)</td>
<td>Decrease/absence of ejaculation/orgasm</td>
</tr>
<tr>
<td>LITHIUM</td>
<td></td>
<td>Decrease/absence of ejaculation/orgasm</td>
</tr>
</tbody>
</table>
| PSYCHOPHARMACEUTICALS   | (S)SRI's         | See table X.1
|                         | Trazodone        | Decrease/absence of ejaculation/orgasm Retrograde ejaculation |
|                         | MAOI's           | Decrease/absence of ejaculation/orgasm |
|                         | Irocumarboxazid | Decrease/absence of ejaculation/orgasm |
|                         | Phenelzine       | Decrease/absence of ejaculation/orgasm |
|                         | Tranylcypromine  | Decrease/absence of ejaculation/orgasm |
| Anxiolytics             | Benzodiazepines  | Decrease/absence of ejaculation/orgasm |
| Neuroleptics            | Chlorpromazine   | Decrease/absence of ejaculation/orgasm Retrograde ejaculation |
| Phenothiazines          | Fluphenazine     | Decrease/absence of ejaculation/orgasm Retrograde ejaculation |
|                         | Levomepromazine  | Decrease/absence of ejaculation/orgasm Retrograde ejaculation |
|                         | Perphenazine     | Decrease/absence of ejaculation/orgasm |
|                         | Pipethiazine     | Decrease/absence of ejaculation/orgasm |
|                         | Thioridazine     | Decrease/absence of ejaculation/orgasm Retrograde ejaculation |
|                         | Trifluoperazine  | Decrease & increase reported |
| Thioxanthenes           | Chlorprothixene  | Decrease/absence of ejaculation/orgasm Retrograde ejaculation |
|                         | Thiothixene      | Decrease/absence of ejaculation/orgasm Spontaneous ejaculation Retrograde ejaculation |
| Butyrophenones          | Haloperidol      | Painful ejaculation |
| Diphenylbutylinpiperidines | Pimozide      | Decrease/absence of ejaculation/orgasm |
Table 2: Side effects of different drugs on ejaculation (ctd)

<table>
<thead>
<tr>
<th>GROUP OF DRUGS</th>
<th>AGENT</th>
<th>EFFECT ON EJACULATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>MUSCLE RELAXANTS</td>
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<tr>
<td></td>
<td>Baclofen</td>
<td>Decrease/absence of ejaculation/orgasm</td>
</tr>
<tr>
<td>URINARY TRACT</td>
<td></td>
<td></td>
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<tr>
<td>Alpha blockers</td>
<td>Alfuzosin</td>
<td>Retrograde ejaculation</td>
</tr>
<tr>
<td></td>
<td>Phenoxymezamine</td>
<td>Retrograde ejaculation</td>
</tr>
<tr>
<td></td>
<td>Prazosin</td>
<td>Retrograde ejaculation</td>
</tr>
<tr>
<td></td>
<td>Tamsulosin</td>
<td>Retrograde ejaculation</td>
</tr>
<tr>
<td></td>
<td>Terazosin</td>
<td>Retrograde ejaculation</td>
</tr>
<tr>
<td>Alpha reductase inhibitors</td>
<td>Finasteride</td>
<td>Less ejaculatory volume</td>
</tr>
<tr>
<td>RECREATIONAL DRUGS</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alcohol</td>
<td></td>
<td>Decrease/absence of ejaculation/orgasm</td>
</tr>
<tr>
<td>Amphetamines</td>
<td></td>
<td>Decrease &amp; increase reported</td>
</tr>
<tr>
<td>Amylnitrile (poppers)</td>
<td></td>
<td>Increase in ejaculatory latency</td>
</tr>
<tr>
<td>Cocaine</td>
<td></td>
<td>Decrease/absence of ejaculation/orgasm</td>
</tr>
<tr>
<td>Heroin</td>
<td></td>
<td>Decrease &amp; increase reported</td>
</tr>
<tr>
<td>Marijuana</td>
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<tr>
<td>Methadone</td>
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</tr>
<tr>
<td>Tobacco</td>
<td></td>
<td>Decrease/absence of ejaculation/orgasm</td>
</tr>
</tbody>
</table>

After: Moors-Mommers, [78]

affect duration of the excitement phase, such as age, novelty of the sexual partner or situation, and recent frequency of sexual activity.

2) The disturbance causes marked distress or interpersonal difficulty.

3) The premature ejaculation is not due exclusively to the direct effects of a substance (e.g., withdrawal from opioids).

**ICD-10- PREMATURE EJACULATION**
*ICD= INTERNATIONAL CLASSIFICATION OF DISEASES*

1) The general criteria for sexual dysfunction (F52) must be met.

2) There is an inability to delay ejaculation sufficiently to enjoy lovemaking, manifest as either of the following:

- Occurrence of ejaculation before or very soon after the beginning of intercourse (if a time limit is required: before or within 15 seconds of the beginning of intercourse);
- Ejaculation occurs in the absence of sufficient erection to make intercourse possible.

3) The problem is not the result of prolonged abstinence from sexual activity.

**b) Assessment (Decision Tree A)**

Rapid ejaculation is assessed along two major dimensions:

- ejaculatory latency post-vaginal penetration and
- degree of voluntary control.

*The simplest dimension to assess is ejaculatory latency or duration of intercourse.* The clinician inquires how long it takes for the man to reach orgasm under each of the following circumstances: with masturbation, partners hand and/or mouth stimulation, and intercourse in varying positions. As the patient answers these questions, the doctor listens for various factors that may enable reassurance to be the primary treatment. These include men with unusually high and unrealistic expectations: for instance, that intercourse should last 45 minutes! The doctor sometimes can quickly educate these men to no longer consider themselves rapid ejaculators. Reassurance can sometimes be helpful to men who are young, inexperienced, or trying too hard to please a new partner.

*The second major dimension to be assessed when men complain of rapid ejaculation includes the factors that affect the man's degree of voluntary control.* These are generally psychosocial conditions. The doctor inquires whether the rapid ejaculation occurs under all circumstances or only with a specific partner or a specific circumstance. Selective rapid ejaculation, either specific to a partner or specific to a setting is usually acquired rapid ejaculation. In these cases, the clinician turns his attention to the life events that preceded the onset of the problem. For instance, acquired rapid ejaculation may follow a myocardial infarction or the discovery of his wife's infidelity. Acquired
**Decision Tree A:**
Decision tree of investigation and treatment of premature ejaculation (PE)

**History Taking: Patient and Partner**
- Psychosocial history
- Somatic history
- (Intravaginal) ejaculatory latency time
- Degree of voluntary control
- Duration of PE

**Symptom of PE?**
- **YES**
- **NO**

**PE secondary to erectile dysfunction or other sexual dysfunction?**
- **YES**
- **NO**

**Drug dependent PE?**
- **YES**
- **NO**

**Psychosocial/situational PE?**
- **YES**
- **NO**

**Meet all DSM IV criteria?**
- **YES**
- **NO**

**Consider laboratory assessment e.g., psychophysiological examination**
- Electrophysiological evaluation

**Concomitant ED or libido dysfunction?**
- **YES**
- **NO**

**Treatment**
- Behavioral techniques
  - Stop/start
  - Sensate focus
  - Quiet vagina
- Drug treatment (see text)
- Integrated psychodynamic approach
- Desensitization
  - Anaesthetic ointments
  - Double condoms

Or a combination of above

Follow-up

Change diagnosis/follow-up
rapid ejaculation may also mask an underlying erectile disorder. In these cases the rapid ejaculation is an adaptive response to prevent losing erections. The doctor should first proceed with the work-up and treatment for the erectile dysfunction and only treat the ejaculatory dysfunction if the symptom remains. Even when the rapid ejaculation is lifelong, the doctor needs to wonder why the man seeks help for the condition at this time. The answer is often that the stability of the marital relationship is being threatened and the man looks to the clinician for a simple face saving explanation and treatment for a more complex problem: marital deterioration.

Rapid ejaculation may also be a disguise for partner sexual dysfunction. For example, a woman may have an intense sexual aversion that has kept her partner quickly ejaculating to minimize her distaste for and distress during sex. Thus clinicians need to hear about the quality of the sexual adjustment over time to ascertain the social factors that may suggest how to plan treatment.

In summary, the assessment of rapid or premature ejaculation is done by sexual history taking. An attempt is made to ascertain the man’s social circumstances, his motivations for pharmacological or psychological treatment and his partner’s receptivity to such treatment. In some situations, it is best to not attempt a pharmacological therapy until social circumstances are further clarified or resolved because the patient who may ultimately have been helped by medications loses the opportunity because he or his partner could not surmount the psychosocial obstacles related to the sexual dysfunction.

2. ASSESSMENT OF DELAYED RETARDED EJACULATION

Assessment begins by reviewing the conditions under which the man is able to ejaculate, e.g. during sleep, with masturbation, with partner’s hand or mouth stimulation or infrequently with varying coital positions. The course of the problem is documented, and variables that improve or worsen performance are noted, such as the need for unconventional fantasies or a lifelong need to suppress spontaneous emotional expression. Questions concerning the man’s ability to relax, sustain and heighten arousal and the degree to which he can concentrate on sensations are posed. If orgasmic attainment had been possible previously, the life events/circumstances temporarily related to orgasmic cessation are reviewed, e.g. following his wife’s mastectomy: the man is afraid of hurting her and therefore only partially aroused. Societal/religious attitudes that may interfere with excitement are noted, such as the spilling of seed as a sin. Finally, questions concerning the quality of the nonsexual relationship are posed and problems explored.

3. ASSESSMENT OF HAEMOSPERMIA

Haemospermia requires full investigation. Culture of expressed prostatic secretion and urine will define the nature of an infective process such as prostatitis [81] and urine cytology and serum prostate specific antigen should be assayed to exclude bladder or prostatic cancer. Ultrasound scan of the testicles and epididymes should define any local disease. TRUS will demonstrate structural abnormality in the prostate or seminal vesicles, or may show up a stone in the ejaculatory duct or even a Mullerian duct cyst. Cystoscopy is seldom helpful.

4. ASSESSMENT OF SMALL VOLUME EJACULATE

(Decision Tree B)

If a man has difficulty with ejaculation, or has a small volume or absent ejaculate, it must first be established whether the problem is congenital or acquired. A careful clinical history should be taken, and physical examination will establish whether the testicles and epididymes are normal, and whether the vasa are present or absent, on each side. Next, it is essential to establish whether there is retrograde or completely absent ejaculation, by examination of a deposit of urine after centrifugation. The presence of spermatozoa indicates retrograde ejaculation. These facts will allow the patient to be placed into one of several broad categories, after which more detailed evaluation can take place.

Patients with ejaculatory duct obstruction usually (Decision Tree C) present with infertility. Seminal analysis may simply be reported a showing azoospermia or oligozoospermia, but the characteristic biochemical changes should be sought. There should be absence of part or the entire component of the ejaculate that comes from the vasa and seminal vesicles via the ejaculatory ducts. The volume is low (usually less than 1.5 ml), the pH is low (less than 7) and the fructose content is either low (less than 120 mg/100ml) or absent. If both vasa are palpable, a diagnosis of ejaculatory duct obstruction is very likely.

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PRESENTING COMPLAINT: - little or no ejaculate

Is there orgasm?  
NO  
Anorgasmia  
? nocturnal emission  
⇒ psychologist

YES

Is there any ejaculate?  
NO  
Seminal analysis  
? low volume  
⇒ Decision tree C

YES

? Are there sperm present in urine after orgasm  
NO  
Retrograde ejaculation  
⇒ urologist

YES

Aspermia  
⇒ Decision Tree C

DECISION TREE C:  
INVESTIGATION OF DIMINISHED EJACULATE

HISTORY
- little or no ejaculate?
- infertility?
- epididymitis?
- haemospermia?

SEMINAL ANALYSIS
- low volume?
- acid pH?
- reduced fructose?
  No  ⇒ ? functional

TRUS
- distended vesicles?
- abnormal cyst?
  No  ⇒ ? Diabetes mellitus

SEMINAL VESICULOGRAPHY
Percutaneous puncture or vasography
- confirms obstruction?
  No  ⇒ ? Polycystic kidney disease  ⇒ ? megavesicles

TRANSURETHRAL RESECTION
- volume increased?
  No  ⇒ Redo TUR

SEMINAL ANALYSIS
- sperm count improved?
  No  ⇒ Epididymo-vasostomies

Treatment success
When there is absence of the vasa, it is important to establish whether the condition is unilateral or bilateral. With unilateral absence of the vas deferens, the urinary system must also be checked by ultrasound scanning, as coexisting renal anomalies may be present [82]. With bilateral absence or malformation of the vasa, it is essential to consider whether the anomaly may be part of a genetic defect associated with carriage of the potentially harmful cystic fibrosis chromosome anomaly [38].

5. ASSESSMENT OF EJACULATORY DUCT OBSTRUCTION ON IMAGING

The lesion may be suspected by finding distended seminal vesicles on transrectal ultrasound scanning. However, the exact site of obstruction should be defined radiologically by vasography or percutaneous puncture of the seminal vesicles (figure 8). Subsequently, methylene blue dye may be instilled to outline the ejaculatory system so that it can be recognized after it has been entered at transurethral resection [83].

VI. TREATMENT

I. PSYCHOLOGICAL TREATMENT FOR RAPID EJACULATION

Since the early 1970's, an array of individual, conjoint, and group therapy approaches employing behavioral strategies, such as the stop-start, squeeze technique, progressive sensate focus exercises, masturbatory exercises and "quiet vagina" with the female astride, have evolved as the treatments of choice for rapid ejaculation. Behavioral treatment often begins with the man alone, having him repeatedly stimulate himself to midrange levels of excitement before pausing. After several repetitions he is permitted to ejaculate. The aim of this exercise is to help him learn intermediate levels of excitement and begin to slow down his arousal.

After mastering the self-stimulation exercise the partner is asked to repeatedly bring the man to

Figure 8: Vasograms showing a) Normal seminal vesicles and ejaculatory ducts, b) Mullerian duct cyst, c) Wolffian duct abnormality with absence of seminal vesicle, the duct terminating in a cystic malformation close to the prostatic urethra, d) seminal megavesicles (reproduced from the British Journal of Urology with permission).
high levels of excitement, initially through stimulation by her hand or mouth and later by vaginal thrusting, but stopping prior to ejaculation. This stop-start procedure allows the man’s arousal to decrease and thereby delays orgasm. This behavioral sequence is repeated several times after which the man is permitted to ejaculate. Masters and Johnson [84] subsequently developed a modification of this procedure known as the squeeze technique. At the point at which stimulation is stopped, the man’s glans penis is squeezed firmly but quickly by the partner which lowers arousal. Often however, this technique results in a partial loss of erection.

Sensate focus exercises are designed to allow the man to develop an awareness of his arousal level by lessening the demand characteristics of the sexual experience. In a slow, graduated fashion the man and his partner take turns giving and receiving pleasure. Initially the touching is restricted to nongenital/non breast stimulation; upon achieving ejaculatory control these areas are also pleasured.

"Quiet vagina" is an extension of the stop-start maneuver to include intercourse. After successful hand stimulation the woman sits astride or lies on top of the man and, without any thrusting or rhythmic movement envelops his penis in her vagina. The aim of this exercise is to desensitize the man to the wet, warm sensations of the vagina. After the man masters the "quiet vagina" for a prolonged period of time, movement by the woman is slowly introduced. The man directs her to stop when his excitement has increased. The couple sit/tie quietly until his arousal decreases whereupon they resume the exercise. This is repeated several times before the man eventually is allowed to ejaculate.

It is crucial for the therapist to monitor the partners’ needs and responses during therapy. The female partner may feel used and unimportant. This must be acknowledged while helping her to focus on the ultimate goal of pleasurable sex for both partners. Also, the therapist must monitor both patient and partner for the emergence of any resistance that will sabotage treatment.

Cognitive-behavioral treatment for this dysfunction focuses on challenging self-defeating ideas about sexuality or women while replacing them with facilitating thoughts about ejaculatory control, sexuality and intimacy. In addition, the behavioral skill of identifying the point of ejaculatory inevitability is learnt through the use of the stop-start technique and alternating intercourse positions or thrusting movements. Finally, a cooperative, intimate and satisfying relationship is established.

An integrated psychodynamic approach seeks to have the man or couple understand the hidden meaning of the rapid ejaculation, appreciate the interference of performance anxiety and, when ready, embark on a series of behavioral tasks. Clinicians need to be aware of the man or couple’s need for a symptom and how rare it is to find "simple cases" of rapid ejaculation.

It has been found that the impressive treatment success rates of 60% to 95% reported by Masters and Johnson [84] can not be replicated and are not sustainable. Three years after behavioral treatment, success rates dwindle to 25%. This data suggest that behavioral clinicians may have failed to recognize psychodynamic causes of the disorder or to develop long-term strategies that allow patients to maintain their initial therapeutic gains.

2. PSYCHOLOGICAL TREATMENT FOR DELAYED EJACULATION

Treatment efforts are guided by the assumptions underlying the contrary theoretical models of causation. Proponents of the inhibition model understand the symptom as the man’s muting of his excitement and prescribe techniques to increase excitement through prolonged, intense, rough stimulation or by interpreting the man’s unconscious aggressive impulses. Diametrically opposed to the inhibition model is the paradigm that considers delayed ejaculation as a failure of the man to be excited enough to achieve orgasm. Treatment efforts are aimed at having the men acknowledge their lack of both desire to have intercourse and arousal during intercourse. This model mirrors the conventional therapy for female anorgasmia focusing on decreasing demand and helping the patient focus on heightening erotic sensations.

Masters and Johnson [84] reported a low failure rate of 17.6% using a treatment combination of sensate focus, vigorous non-coital penile stimulation and modifications of intercourse technique. In another study 81% of men who were anorgasmic prior to treatment were successful in reaching orgasm through vibrator stimulation. No outcome statistics are known for treatment model that considers men insufficiently aroused.
3. DRUG TREATMENT FOR RAPID EJACULATION

Reducing penile skin sensitivity with the application of local anaesthetic gel can treat premature ejaculation. By keeping the cream in contact with the skin with a condom for thirty minutes, significant improvement was obtained [85]. It was, however, important to wash off the local anaesthetic prior to intercourse if diminution of vaginal sensitivity in the female partner was to be avoided [86]. Clomipramine, a tricyclic antidepressant, has been shown to produce significant delay in time to orgasm with increased satisfaction with sex life in prospective controlled trial, given in a dose of 25 mg 12 to 24 hours before inter course [64]. Fluoxetine (Prozac) given in a dose of 20 mg daily for 1 week and 40 mg daily thereafter has also been used, and produced significant benefit after 4 weeks treatment [87]. The female partners involved in the latter study subjected the effects to careful scrutiny including verification of intravaginal latency time.

Retrograde ejaculation can be treated with adrenergic drugs such as ephedrine, 30 - 60 mg, or a tricyclic antidepressant with anticholinergic effects such as desipramine, 50 mg, taken 1 - 2 hours before sexual activity. One patient with azoospermia and small volume ejaculate associated with an open bladder neck and unilateral absence of the vas deferens responded well to ephedrine with normalization of the seminal analysis, and subsequently a pregnancy was produced. Alternatively, spermatozoa can be retrieved from post-orgasmic urine by centrifugation after retrograde ejaculation, resuspended and used for artificial insemination with success: a cumulative pregnancy rate as high as 72% at 6 months has been achieved [88].

4. SPINAL INJURIES

In some paraplegic patients, application of a vibrator to the penis will lead to ejaculation; in others, electroejaculation may be necessary to produce spermatozoa that can be used for insemination. If the spinal reflex arc is intact, a hypogastric plexus stimulator will provoke ejaculation [49]. This method has the advantage that it can be used in the security of the patients’ home, and repeated ejaculation can improve the quality of the semen. Alternatively, direct electroejaculation by rectal probe may be effective, but this generally requires a general anaesthetic and is done in hospital [50]. In a recent analysis of 40 paraplegic patients, 22 successfully produced pregnancies by natural insemination or assisted reproductive techniques [51].

5. LOSS OF EJACULATION AFTER RETROPERITONEAL LYMPH NODE DISSECTION

Drug treatment for loss of ejaculation after paraaortic lymphadenectomy is not very successful [89] but electroejaculation can produce spermatozoa for insemination [90]. It is important to anticipate this complication in young men with testicular tumours who may need chemotherapy or node dissection, and arrangements should be made for sperm storage at the earliest opportunity before treatment commences. Excellent results can be obtained with artificial insemination using cryopreserved spermatozoa [56].

6. SURGICAL TREATMENT OF EJACULATORY DUCT OBSTRUCTION

It is very helpful if the lesion is accurately defined preoperatively by TRUS, so that all necessary arrangements can be made in advance. The obstruction should then be defined radiologically by vasography or percutaneous puncture of the seminal vesicles, and 5 - 10 mls of 1% methylene blue dye are instilled to indicate when the ejaculatory system has been entered. The patient should be placed in the lithotomy position and suitable drapes applied to allow access to rectal examination during the procedure. After preliminary cystoscopy, the resectoscope or optical urethroscope is inserted. A Mullerian duct cyst may simply be incised, releasing a gush of fluid, but there is a tendency for the incision to heal over and it may be preferable to resect the edges or make a cruciate incision. If the ejaculatory ducts are blocked at their lower ends, it may be simpler to resect the verumontanum, commencing just above it in the prostatic urethra and drawing the loop carefully downward. The appearance of the ejaculatory ducts is characteristic and easily recognized, resembling a horse's nostrils. Pressure on the seminal vesicles will produce abundant efflux once the obstruction has been relieved.

Analysis of results obtained with 87 patients with ejaculatory duct obstruction is summarized in Table 3 [91]. It may be seen that incision of Mullerian duct cyst was much the most successful procedure, but satisfactory results have been obtained in other patients, and have continued to be seen
Table 3: Number of patients with ejaculatory duct obstruction by group, number successfully treated/number with adequate follow up in each group (from Pryor and Hendry (91)).

<table>
<thead>
<tr>
<th>Group</th>
<th>Total Number</th>
<th>Number with follow up</th>
<th>Post-operative potency</th>
<th>Pregnancies produced</th>
</tr>
</thead>
<tbody>
<tr>
<td>CONGENITAL</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mullerian</td>
<td>17</td>
<td>12</td>
<td>10</td>
<td>5</td>
</tr>
<tr>
<td>Wolffian</td>
<td>19</td>
<td>6</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Traumatic</td>
<td>15</td>
<td>6</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Infective</td>
<td>19</td>
<td>6</td>
<td>4</td>
<td>2</td>
</tr>
<tr>
<td>Tuberculous</td>
<td>8</td>
<td>1</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Megavesicles</td>
<td>8</td>
<td>1</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Neoplastic</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TOTAL</td>
<td>87</td>
<td>31</td>
<td>18</td>
<td>10</td>
</tr>
</tbody>
</table>

since this study was completed. If reconstruction is not possible, sperm can be withdrawn by microscopic epididymal sperm aspiration (MESA) or percutaneously (PESA) and used for in-vitro fertilization [92]. Attempts to insert a permanent sperm reservoir gave only limited success and this treatment has now been abandoned [93].

VII. CONCLUSIONS

It is clear that there is much to study and understand in disorders of orgasm and ejaculation. Experimental evidence has shone light onto the biochemical function of the brain, especially in the limbic system and hypothalamus. The side effects of drug therapy have provided insight into functional disorders, and indicated effective methods of treatment. Congenital malformations and their relationship to genetic disorders are now more clearly understood: these are matters of importance in the present era of assisted reproduction, if perpetuation of serious anomalies such as fibrocystic disease is to be avoided. Surgically induced injuries that impact upon male reproductive function are now recognized and largely preventable by careful attention to preservation of normal structures during extirpative surgery.

It seems likely that in the future, these will be areas of much fruitful research.

VIII. RECOMMENDATIONS

1. Care should be taken to distinguish erectile dysfunction from difficulties with orgasm and ejaculation.
2. The difference between emission (deposition of semen into the posterior urethra) and ejaculation (from the urethral meatus) should be recognized.
3. The presence of retrograde ejaculation should be established early in the diagnostic work-up of patients with loss of ejaculation by appropriate examination of centrifuged urine after orgasm.
4. The afferent nervous pathways, cerebral receptor and motor areas, and efferent spinal and sympathetic nerves controlling orgasm and ejaculation should be clearly understood by physicians dealing with sexual dysfunction.
5. The facilitative role of dopamine and the inhibitory role of serotonin (5-HT) in the production of orgasm and ejaculation must be understood.
6. The effects of antidepressant and other drugs on the normal cerebral biochemical transmitters (See above : 5) should be appreciated.
7. Neurological tests to study the connections between the genitalia and the central nervous system should be more widely understood.
8. The embryology of the male genital tract, and congenital anomalies leading to ejaculatory malfunction should be familiar to all urologists.
9. Damage caused to the ejaculatory system by rectal pull-through procedures for imperforate anus should be more widely recognized by paediatric surgeons dealing with this condition.
10. The effects of bladder neck incision and prostatectomy on ejaculation must be explained to patients prior to surgery.
11. The possibility of ejaculatory duct obstruction should be considered in men with infertility who have had genital infection including sexually transmitted diseases, Schistosomiasis, tuberculosis and unexplained urinary infection.
12. The effects of neurological disease and spinal cord injury on sexual function should be widely understood (see section 14).

13. Nerve sparing techniques of retroperitoneal lymph node dissection should be used whenever possible.

14. Arrangements for sperm storage should be made prior to administration of chemotherapy likely to interfere with spermatogenesis or surgery that may interfere with ejaculation.

15. Rapid or premature ejaculation may be defined as inability to delay ejaculation sufficiently to enjoy lovemaking, or, alternatively, persistent or recurrent occurrence of ejaculation with minimal sexual stimulation before, on, or shortly after penetration and before the person wishes it.

16. Patients with rapid ejaculation should be fully evaluated with objective data. It is useful to measure ejaculatory latency time by stopwatch.

17. Psychosocial background to rapid ejaculation should be investigated, with involvement of the partner.

18. Societal and religious attitudes to sex should be recognized.

19. Treatment of rapid ejaculation should include behavioural therapy including the female partner.

20. Results of treatment of rapid ejaculation should be evaluated in both short and long term.

21. Drug therapy for rapid ejaculation may be given immediately prior to sexual activity (e.g. Clomipramine) or more chronically (e.g. Paroxetine). The dose schedule, effects and unwanted side effects of such therapy must be carefully monitored.

22. The volume, pH and fructose content of semen in subfertile men with oligozoospermia or azoospermia should be measured and the result noted by doctors caring for infertile couples.

23. Abnormalities in seminal volume or biochemical characteristics should be evaluated by a urologist. Transrectal ultrasound scanning is recommended as the first investigation, which may be supplemented by vasography or percutaneous puncture of the seminal vesicles to establish the presence of ejaculatory duct obstruction.

24. Urologists should be familiar with the anatomy of the ejaculatory ducts in relation to surrounding structures and be able to relieve ejaculatory duct obstruction safely by appropriate resection.

25. Electroejaculation should be available at selected fertility centres for patients with spinal injuries or loss of ejaculation after abdominal surgery.

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Some of the members of the International Committees
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