Ejaculatory duct obstruction

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Purpose of review

We surveyed the growing literature on ejaculatory duct obstruction and provide suggestions regarding its diagnosis and management.

Recent findings

Ejaculatory duct obstruction is a rare cause of male infertility. With the advent of the high resolution transurethral ultrasound (TRUS) technology, there has been an increase in diagnosis of this disorder. As for the treatment, it appears that central cystic lesions and partial obstructions respond best to transurethral resection of the ejaculatory ducts (TURED).

Summary

Ejaculatory duct obstruction is a rare but surgically correctable cause of male infertility. Although there are no pathognomonic findings associated with ejaculatory duct obstruction, the diagnosis should be suspected in an infertile male with oligospermia or azoospermia with low ejaculate volume, normal secondary sex characteristics, testes, and hormonal profile, and dilated seminal vesicles, midline cyst, or calcifications on TRUS. In select cases, TURED has resulted in marked improvement in semen parameters, and pregnancies have been achieved. More studies are needed in the areas of diagnosis and long-term surgical outcome.

Keywords

male infertility, ejaculatory duct obstruction, transrectal ultrasound (TRUS), transurethral resection of the ejaculatory ducts (TURED)

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Abbreviations

TRUS	transrectal ultrasound
TURED	transurethral resection of the ejaculatory ducts

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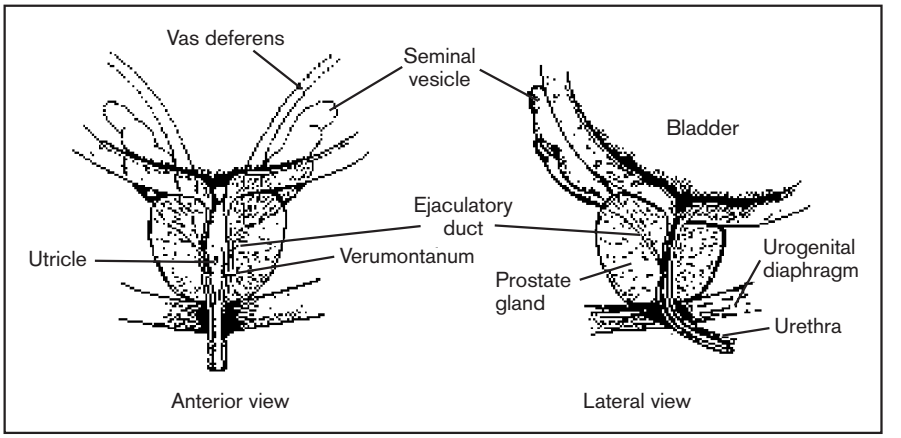
Introduction

Although obstruction of the epididymis and proximal vas have become well recognized and readily treated causes of male infertility [1], more distal obstructions have only relatively recently been recognized and treated [2–9]. Ejaculatory duct obstruction, although rare, is a surgically correctable cause of male infertility [2,5–15]. Use of high-resolution transrectal ultrasound (TRUS) has resulted in an increased incidence of diagnosis of this disorder [3,14,16,17]. Treatment of ejaculatory duct obstruction by transurethral resection of the ejaculatory ducts (TURED) has also become more common; there have been several reports of pregnancies following relief of ejaculatory duct obstruction using this technique [2,4,5,13,15,18–20]. Although various symptoms, signs, TRUS, radiographic, and cystoscopic findings have been associated with ejaculatory duct obstruction, none is pathognomonic for this disorder [2]. Moreover, the pathogenesis of ejaculatory duct obstruction in association with these findings, and how this obstruction impacts on male fertility, is not well understood. By examining the anatomy of the ejaculatory ductal system, and correlating it with symptomatology, semen analyses, TRUS, and pathologic findings in patients with a presumptive diagnosis of ejaculatory duct obstruction, a better understanding of ejaculatory duct obstruction and its impact on male infertility can be gained.

Anatomy

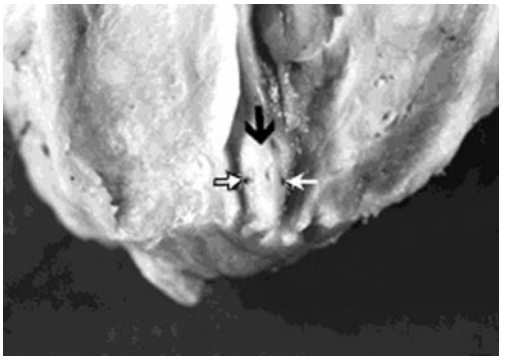
The ejaculatory ducts develop from the distal-most vas, from the wolffian duct system. The seminal vesicles develop as a blind diverticulum at the most terminal end of the vas [21]. The ejaculatory ducts are a direct continuation of the seminal vesicles and, anatomically, begin after the ampulla of the vas joins the seminal vesicle duct on its medial aspect at an acute angle [10,22,23] (Fig. 1). The ducts are approximately 1–2 cm long and enter the prostate obliquely posteriorly at its base, course medially and anteriorly through the prostatic glandular tissue, and enter the prostatic urethra at the verumontanum [10,13,22,23]. Between the two ejaculatory ducts at the verumontanum sits the prostatic utricle, a müllerian tubercle remnant of endodermal origin [23]. The ejaculatory ducts open in the majority of cases anterolateral to the orifice of the utricle [23]. In most men, the utricle is less than 6 mm in size but, in up to 10% of men, can exceed 10 mm [24]. The utricle does not communicate with any other structures [10,22,23,25]. Injection of methyl methacrylate into the vas deferens of intact autopsy prostate/seminal vesicles/vasa specimens reveals the ejaculatory ducts exiting close to one another

Figure 1. Schematic representation of the distal ejaculatory duct anatomy



at the verumontanum, with a small utricle lying between them. No methyl methacrylate can be seen exiting the utricle [26] (Fig. 2). In sagittal sections, the ejaculatory duct forms an almost straight course from the prostatic base to the verumontanum (Fig. 3). The close relationship of the ejaculatory ducts to the utricle can be seen in transverse section at the verumontanum of a radical retropubic prostatectomy specimen [26] (Fig. 4). The anatomic structures of the ejaculatory ductal system and their relationships can also be demonstrated using rectal coil magnetic resonance imaging [5,27]. In sagittal image, the relationships between the bladder, bladder neck, seminal vesicles, prostate, and ejaculatory ducts are demonstrated. In addition, this patient has a midline cyst that divides the ejaculatory ducts laterally (Fig. 5). Also note that the distal ejaculatory duct and cyst are distal and inferior to the bladder neck. Each duct is surrounded by circular lamellar tissue and, in turn, both ducts are surrounded by a communal muscular envelope [22,28]. The existence of a 'sphincter spermaticus' has been described, but its role in the pathophysiology of partial or functional ejaculatory duct obstruction remains poorly understood [4,11]. The ejaculatory ducts are lined by a yellow pigmented cuboidal to pseudostratified columnar epithelium [22,28] (Fig. 6).

Figure 2. Coronal section of the prostate from an autopsy specimen after injection of methyl methacrylate into the vasa deferentia



Note methyl methacrylate exiting from distal ejaculatory ducts (small white arrows) which sit lateral to the midline utricle (large black arrow).

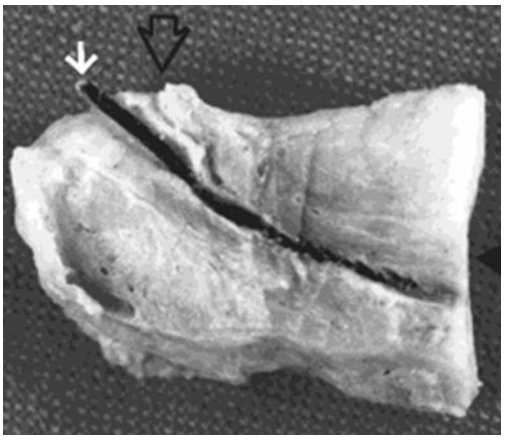
Etiologies of obstruction

Ejaculatory duct obstruction can be either congenital or acquired [13,15]. Congenital causes include congenital atresia or stenosis of the ejaculatory ducts and utricular, müllerian, and wolffian duct cysts. Acquired causes may be secondary to trauma, either iatrogenic or otherwise, or infectious or inflammatory etiologies [13,15]. Calculus formation secondary to infection may also cause obstruction [4]. Cyst formation from prior instrumentation or infection may also occur [25]. In many cases, patients with ejaculatory duct obstruction have no significant antecedent history [10]. Several authors have found that patients with congenital or noninfectious causes of ejaculatory duct obstruction do better after treatment than those with infectious causes [8,9,13,15,20]. Other authors, however, have not been able to support this [10,29,30]. In addition, there is a report of patients with partial ejaculatory duct obstructions having better improvements in their semen parameters than those with complete obstruction [20].

Symptoms

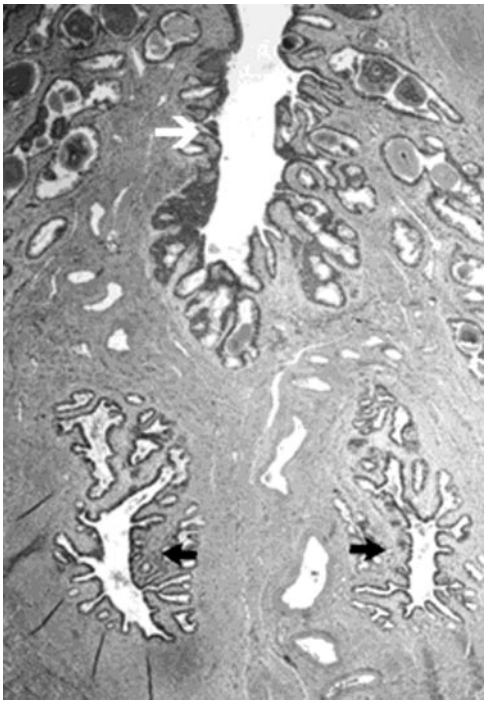
Patient complaints associated with ejaculatory duct obstruction can be quite variable but include infertility, decreased force of ejaculate, pain on or after ejaculation, decreased ejaculate volume, hematospermia, perineal or testicular pain, history of prostatitis or epididymitis, low back pain, urinary obstruction, dysuria, or no symptoms

Figure 3. Sagittal section of the prostate from an autopsy specimen after injection of methyl methacrylate into the vasa deferentia



Note ejaculatory duct (white arrow) exiting distal and inferior to the utricle (open arrowhead) and the almost straight course of the ejaculatory duct from the prostate base (black arrow) to the verumontanum.

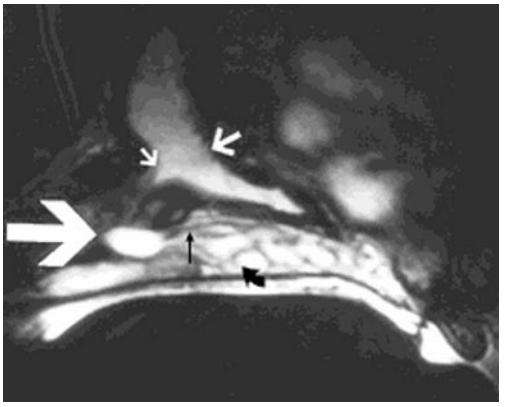
Figure 4. Transverse section through the verumontanum from a radical retropubic prostatectomy specimen



The close relationship of the ejaculatory ducts (black arrows) to each other and to the utricle (white arrow) is shown.

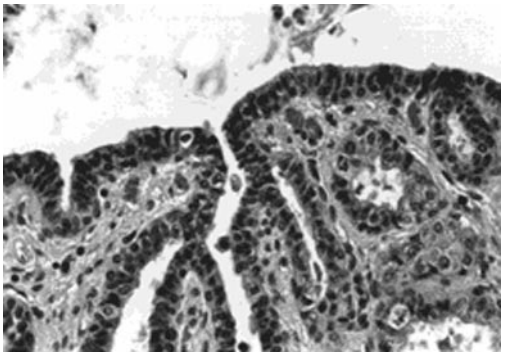
[2,5,9–11,13,29]. Symptoms are generally less pronounced or absent in patients with partial obstructions; however, partial obstructions can progress to complete

Figure 5. Sagittal image from rectal coil magnetic resonance imaging



The relationships between the bladder (medium white arrow), bladder neck (small white arrow), seminal vesicles (curved black arrow), and ejaculatory ducts (small black arrow), with a midline cyst (large white arrow) are shown. Note that the distal ejaculatory duct and midline cyst are quite distal and inferior to the bladder neck.

Figure 6. Histologic section of the distal ejaculatory duct epithelium from transurethral resection of the ejaculatory duct specimen



The figure shows cuboidal to pseudostratified columnar epithelium (hematoxylin and eosin).

obstruction [4–6]. No one symptom or constellation of symptoms can make a definitive diagnosis of ejaculatory duct obstruction.

Signs

Patients with suspected ejaculatory duct obstruction classically have normal physical examinations, including normal testes, absence of varicoceles, palpable vasa, normal rectal examinations, normal secondary sexual characteristics, and normal hormonal profiles. Occasionally, there will be a palpable seminal vesicle or mass on rectal examination, or prostatic or epididymal tenderness [2,5,10,13,29]. These patients, however, can, of course, have more than one disorder at the same time. That is, a

patient with ejaculatory duct obstruction might also have a varicocele or a patient with testicular failure might also have ejaculatory duct obstruction [6]. Although a patient might seem to demonstrate findings only of ejaculatory duct obstruction, complete evaluation for other concomitant, possibly treatable, disorders is necessary.

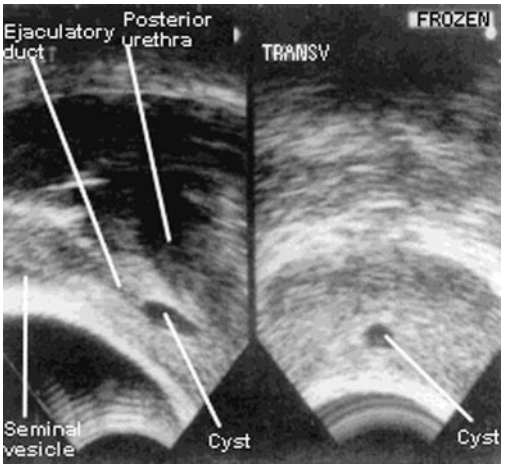
Semen analysis findings in men with partial ejaculatory duct obstruction include oligospermia or azoospermia, decreased motility, and decreased ejaculate volume [2,6,10]. In some men with only mild partial obstructions, semen analyses can approach normal parameters, although motility may remain low [4,6,29]. Decreased ejaculate volume, that is, volumes of less than 1 cm^3 (normal 1.5–5 cm^3), may be suggestive of ejaculatory duct obstruction, but it is by no means pathognomonic [2,4,5,10,16]. Although theoretically, with complete ejaculatory obstruction, seminal fluid should be fructose negative, often fructose is present, implying the presence of only partial obstruction [5,6]. Pryor and Hendry [13] have stated that the finding of a small volume of acid semen, which does not contain fructose, in a patient with palpable vasa is pathognomonic for ejaculatory duct obstruction.

Historically, vasography was the gold standard for diagnosis of proximal and distal ejaculatory duct obstruction [5,13,14,16]. Its invasive nature, however, with risks of iatrogenic stricture and vasal occlusion, and relative risks of general anesthesia and radiation exposure, have made TRUS a more attractive diagnostic technique [2,9,14,16–18,31]. TRUS is much less invasive and can demonstrate the anatomic relationships of the prostate, seminal vesicles, and ejaculatory ducts with exquisite detail [3,5,14,16,17,23,32,33]. Katz *et al.* [34] reported the use of ultrasound-guided transrectal seminal vesiculography under local anesthesia. Under TRUS guidance, a 22 G needle is advanced into the seminal vesicle and, after its position is confirmed with aspiration, contrast medium is injected. Although not generally accepted as yet, this technique eliminates the risks associated with vasography while preserving excellent radiographic visualization of the ejaculatory ducts. Jarow [35] has also shown that TRUS-guided seminal vesicle aspiration was useful in the diagnosis of partial ejaculatory duct obstruction when motile sperm are found in the aspirate. Orhan *et al.* [36] describe the use of TRUS-guided seminal vesicle aspiration to both diagnose ejaculatory duct obstruction and to collect sperm for assisted reproduction techniques.

TRUS findings in suspected ejaculatory duct obstruction include midline cysts (Fig. 7), dilated seminal vesicles (Fig. 8A) or ejaculatory ducts, and hyperechoic regions suggestive of calcifications (Fig. 8B) [2-5,10,17,32]. Although seminal vesicle dilation has been frequently associated with ejaculatory duct obstruction, it is not always present; conversely, normal fertile men can, at times, have dilated seminal vesicles [23,33,37,38]. Jarow [16] showed that seminal vesicle width, length, and area did not differ between fertile and infertile men on TRUS; he also stated, however, that cystic dilation of the seminal vesicles in association with abnormally low ejaculate volume is pathognomonic for ejaculatory duct obstruction. Recent literature proposes that seminal vesicles larger than 15 mm in transverse diameter are abnormal, suggesting ejaculatory duct obstruction [1–3,16]; however, this has not been universally accepted.

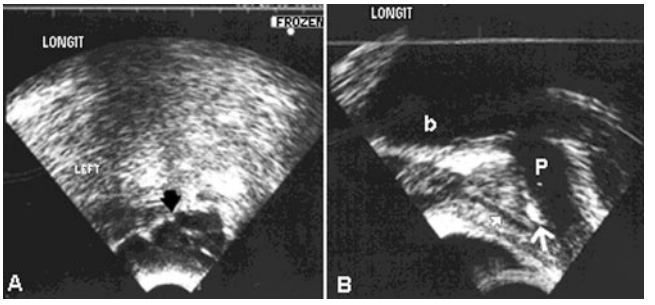
Midline cysts can be classified into two general categories, those that contain sperm and those that do not [18,25,39,40]. These can often be difficult to

Figure 7. Transrectal ultrasound images



Transverse image on the right and longitudinal on the left, showing a midline cyst at the distal ejaculatory duct.

Figure 8. Transrectal ultrasound longitudinal images



(A) Image depicts a dilated fluid-filled seminal vesicle (arrow) measuring 21.8 mm. (B) Image shows highly echogenic areas (large white arrow) in the region of the distal ejaculatory duct (small white arrow). b, bladder; P, posterior urethra.

distinguish [25,31,39]. The latter are generally called utricles or müllerian duct cysts. The differences between utricular and müllerian duct cysts include embryologic origin, with the utricular cysts being of endodermal and müllerian duct cysts being of mesodermal origin; location, with utricular cysts being midline near the verumontanum and müllerian duct cysts nearer the prostate base; and association of enlarged utricles with intersex disorders [16,17,41]. In any case, both cause ejaculatory duct obstruction by compressing the ducts, and both can be treated by TURED, albeit müllerian duct cysts may be more difficult to resect because of their more posterior location [41]. Cysts that contain sperm have been called wolffian or ejaculatory duct cysts or diverticula and are less common than the müllerian duct cysts [16,25,31,39,40]. Confusion as to whether a cyst is müllerian or wolffian in origin can be compounded by the fact that secondary epididymal obstruction can occur after long-term ejaculatory duct obstruction, resulting in the possible absence of sperm in a wolffian structure [30,32]. Midline cysts cause obstruction of the ejaculatory ducts by deviating them laterally or compressing them [10]. Jarow [16] showed, in comparing TRUS findings between fertile and infertile men, that infertile men had a significantly greater incidence of midline müllerian duct cysts than fertile men (11 versus 0%), but he could not draw any conclusions concerning the functional significance of this finding. As was true for seminal vesicle dilation, the presence of a midline cyst does not assure the diagnosis of ejaculatory duct obstruction, but certainly suggests it is in the correct clinical setting.

Calcifications along the course of ejaculatory ducts might be directly involved in obstruction, but those in the prostate itself are associated with prior inflammation, although not necessarily with symptomatic prostatitis [3,4,10]. How prostate inflammation leads to ejaculatory duct obstruction has not been well characterized; inflammatory involvement of the ducts themselves leading to stenosis or obstruction could play a role, whereas changes in compliance of the ejaculatory duct walls or of the adjacent prostatic tissue could cause a functional obstruction [13,15,17]. Calcifications at the junction of the ejaculatory ducts and the urethra have been described in normal individuals on TRUS [3,16]. Prostate or ejaculatory duct calcifications are associated with ejaculatory duct obstruction, but are not a reliable indicator of it. Jarow [16] found that hyperechoic lesions on TRUS were present in a similar proportion of fertile and infertile men.

Treatment

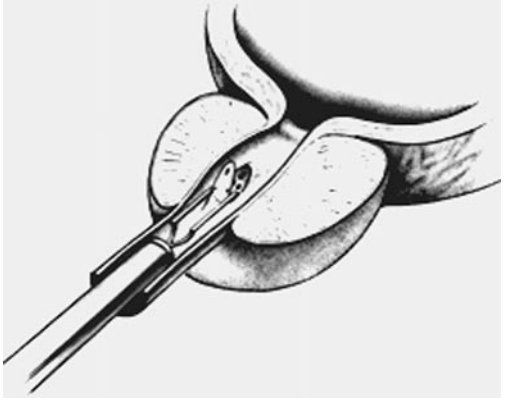
In patients with suspected ejaculatory duct obstruction, the standard procedure has become TURED [2,4,8–10,17]. Originally described by Farley and Barnes in 1973 [11], several reports have documented its efficacy [2–13,15,19].

TURED requires a setup similar to that of transurethral resection of the prostate [10]. An O'Connor drape is used. Cystourethroscopy is performed to rule out strictures in the anterior and bulbar urethra, as well as for evaluation of the posterior urethra. Cystoscopic findings include distorted verumontanum anatomy, splaying of the ejaculatory ducts, bulbous or bi-lobed verumontana, midline cysts, and inflammatory calcifications [10]. Once this is done, the resectoscope is inserted. The proximal verumontanum, which may be enlarged, is resected in the midline (Fig. 9). TURED is performed using pure cutting current without coagulation. Commonly, one or two chips are resected, removing the proximal verumontanum only. Although, historically, lateral Colling's knife incisions were made [17], resection lateral to the verumontanum is not necessary because the ejaculatory ducts are midline structures in this region [10].

With the bladder filled with irrigation fluid, palpation of the seminal vesicles is made easier. Mild pressure is exerted on the seminal vesicles, resulting in fluid expressed from the respective ejaculatory ducts. If no fluid is expressed, another small bite can be taken from the verumontanum, and seminal vesicle pressure applied again. In our experience, operative success for TURED is defined as fluid expression from both ejaculatory ducts at the termination of the procedure. If bleeding is encountered, gentle coagulation is recommended, taking care to avoid the ejaculatory ducts. A catheter is inserted into the bladder and may be left in place for 24–48 h. Postoperative urinary retention can occur after catheter removal, particularly in patients with prior voiding dysfunction. In these cases, reinsertion of the catheter for an additional 24–48 h may be necessary [10].

Complications due to TURED are rare if the procedure is done carefully and with expertise. Obviously, if resection is performed too proximally, damage to the bladder neck can result in retrograde ejaculation postoperatively. Resection too distally can cause damage to the external sphincter with subsequent urinary incontinence. Excessive postoperative fibrosis may result in scarring and subsequent azoospermia, implying reocclusion of the ejaculatory ducts. If this occurs, a repeat TURED may be necessary [10]. Contamination of the ejaculate with urine and seminal vesicle reflux of urine have also been reported [42,43], although the clinical significance of this has not been elucidated. We reported on a patient with seminal vesicle urinary reflux following TURED, causing significant post-void dribbling [44]. Secondary epididymal obstruction can occur after long-term ejaculatory duct obstruction, necessitating scrotal

Figure 9. Schematic diagram of resection of the proximal verumontanum



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exploration and vasoepididymostomy for patients who fail to improve after TURED and in whom this is suspected [30–32].

The patient is asked to refrain from sexual activity for 7–10 days. When sexual activity is resumed, hematospermia may be evident but is self-limited; the patient should be warned of this occurrence and reassured. A semen analysis is obtained 1 month following the resection.

Weintraub *et al.* [5] reported on eight patients with ejaculatory duct obstruction diagnosed by TRUS, rectal coil magnetic resonance imaging, and vasography. Eighty percent of patients were improved symptomatically after TURED, the majority had improvements in sperm density or volume, or both, and 25% were able to impregnate their wives [5]. Hellerstein *et al.* [4] reported on two patients with infertility, one with a large midline cyst and one with dilated seminal vesicles, who underwent TURED for presumed ejaculatory duct obstruction; both had significant improvements in semen parameters and both were able to impregnate their wives.

Meacham *et al.* [2] reported on 24 patients with clinical profiles consistent with ejaculatory duct obstruction, all of who underwent TURED. Fifty percent had an increase in sperm density or motility and 29% had an increase in ejaculate volume only. Seven of 24 (29%) were able to impregnate their wives [2]. Again, none of these studies report on the long-term effects of this procedure. Turek *et al.* [45] showed a greater than 50% improvement in semen parameters in 65% of patients after TURED. Twenty percent were able to initiate a pregnancy; there was a 20% overall complication rate with the most common being a watery ejaculate.

Netto *et al.* [46] showed that the etiology of the ejaculatory duct obstruction was a significant predictor of success after TURED. In those patients with a congenital cause to the obstruction, success rates were excellent with 100% improvement in semen parameters (motility, volume), 83% improvement in sperm count, and 66% pregnancy rate. In those patients with an acquired cause to the obstruction, only 37.5% had improved semen parameters and 12.5% improved pregnancy rate. Furthermore, although 33% of each group had complications, those in the congenital group were more minor in nature.

This notion of central cystic lesions responding best to TURED was confirmed by Kadioglu *et al.* [20] and Schroeder-Printzen *et al.* [8]. In addition, Kadioglu *et al.* [20] demonstrated that response to TURED depended on the degree of obstruction; improvements in semen parameters were significantly better in patients with partial obstruction (94%) than those with complete obstruction (59%).

Aside from TURED, Colpi *et al.* [47] describe antegrade seminal tract washout to relieve ejaculatory obstruction. The vasa were exposed scrotally and saline was injected antegrade to the seminal vesicles until the obstruction was relieved. Fertility was restored in this patient.

Conclusion

With the advent and increased use of high-resolution TRUS, abnormalities of the distal ejaculatory ducts related to infertility have been well documented [2–5,10,16]. Although there are no pathognomonic findings associated with ejaculatory duct obstruction, several clinical findings are highly suggestive. In an infertile male with oligospermia or azoospermia with low ejaculate volume, normal secondary sex characteristics, testes, and hormonal profile, and dilated seminal vesicles, midline cyst, or calcifications on TRUS, ejaculatory duct obstruction is suggested [2,9,10,16,17]. Of course, other causes of infertility may be concomitantly present and need to be searched for and treated as well. In select cases, TURED has resulted in marked improvement in semen parameters, and pregnancies have been achieved [2–5,10,16]. As is the case with all surgical procedures, proper patient selection and surgical experience are necessary to obtain optimal results. In patients with evidence of testicular dysfunction, chances of success are minimal. In addition, extended follow-up periods are needed after TURED to examine the long-term effects of this procedure. Better understanding of the anatomy and pathology of the ejaculatory ducts will help refine diagnostic and therapeutic procedures for this disorder.

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