

# **A Comparative Study of Three Diagnostic Techniques in the Evaluation of Ejaculatory Duct Obstruction**

**Thesis Submitted For Partial Fulfilment of the Doctorate Degree in  
Andrology**

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## **ABSTRACT**

The diagnosis of ejaculatory duct obstruction (EDO) is of importance since it is a surgically correctable cause of male infertility. In this study, TRUS guided seminal vesicle aspiration, seminal vesiculography, and cyst aspiration and dye injection were used to confirm TRUS findings of EDO. Out of 35 patients diagnosed by TRUS as having EDO, only 23 were confirmed to have EDO using these techniques. In conclusion, TRUS is a useful screening tool for EDO; however a TRUS diagnosis of EDO needs further confirmation by TRUS guided techniques. Furthermore, these techniques may have a therapeutic potential.

### **Key words:**

Male infertility, Ejaculatory duct obstruction (EDO), Transrectal ultrasonography (TRUS), Seminal vesicle aspiration, Seminal vesiculography, Cyst aspiration, Cyst dye injection.

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## **LIST OF ABBREVIATIONS**

<b>Abbreviation</b>	
EPS	Expressed prostatic secretion
TRUS	Transrectal ultrasonography
ED	Ejaculatory duct
EDO	Ejaculatory duct obstruction
NOA	Non-obstructive azoospermia
SV	Seminal vesicle
SVA	Seminal vesicle aspiration
SVG	Seminal vesiculography
STW	Seminal tract washout
CDU	Cystic dilatation of the prostatic utricle
TURED	Trans-urethral resection of the ejaculatory ducts
MLC	Midline cyst

***INTRODUCTION***

***&***

***AIM OF THE WORK***



## **INTRODUCTION**

Infertility secondary to ejaculatory duct obstruction (EDO) has been previously thought uncommon; however the ability to make this diagnosis on the basis of transrectal ultrasonography (TRUS) has resulted in an increase in the incidence of diagnosis (*Belker and Steinbock, 1990; Jarow, 1993 and Fisch et al., 2006*).

EDO is a cause of infertility in up to 5% of infertile men (*Jarow, 1994; Schlegel, 1997; Kadioglu et al., 2001 and Onur et al., 2007*).

The causes of EDO are well described including prostatic cysts, duct calcification, stones and blockage due to post-infectious or postoperative scar tissue (*Pryor and Hendry, 1991 and Fisch et al., 2006*).

The diagnosis of EDO is of importance since it is a surgically correctable cause of male infertility (*Meacham et al., 1993; Paick, 2000; Nagler et al., 2002 and Fisch et al., 2006*).

Diagnosis of EDO requires a high degree of suspicion because this disorder is associated with a lack of clear etiologic events and an absence of significant symptoms or signs in the majority of patients (*Paick et al, 2000 and Fisch et al, 2006*).

Complete bilateral EDO may be suspected on clinical grounds from the characteristic semen analysis of low volume (<2ml) of acid semen, azoospermia and absence of seminal fructose in a patient with

papable vasa (*Pryor and Hendry, 1991; Paick, 2000 and Fisch et al., 2006*).

Alternatively in partial or incomplete EDO, there is no pathognomonic semen analysis as in complete EDO. Patients with partial ejaculatory duct obstruction may have normal to low ejaculate volume, normal or reduced sperm count, or abnormal motility (*Pryor and Hendry, 1991; Hellerstein et al., 1992; Meacham et al., 1993; Netto et al., 1998 and Paick et al., 2000*).

For the affected patients, transurethral resection of the obstructed ejaculatory ducts (TURED) is the standard treatment. However, TURED is associated with potential complications including urine ejaculate, recurrent infection, scarring and rarely urinary incontinence. Such risks emphasize the important role of careful diagnosis and patient selection in this condition (*Fisch et al., 2006*).

The optimal method to evaluate EDO has not yet been defined. TRUS and MRI, although less invasive than other methods, provide only anatomical data regarding the static dimensions and positions of the distal genital tract which may not correlate with physical obstruction of the ejaculatory ducts. Trans-scrotal vasography on the other hand provides both static and dynamic information about the reproductive tract yet it is the most invasive technique (*Paick et al., 2000 and Fisch et al., 2006*).

Seminal vesicle aspiration and seminal vesiculography have also been used to diagnose EDO. The presence of sperm in the aspirated seminal vesicle fluid indicates EDO. Ante-grade injection of contrast medium in the seminal vesicles through TRUS guided seminal

vesiculography provides both static anatomical and dynamic information about the reproductive tract (*Jarow, 1996; Paick et al., 2000 and Fisch et al., 2006*).

The relationship between and the value of these diagnostic techniques in EDO management are undefined. Moreover, the most accurate diagnostic technique, which is defined as the one that best predicts a successful outcome after TURED is unclear (*Purohit et al., 2004*).

## **AIM OF THE WORK**

### **Primary Aim**

The aim of this study was to evaluate the accuracy of TRUS, which only provides static anatomical data, in the diagnosis of EDO. Patients with a TRUS diagnosis of EDO went on to have seminal vesicle aspiration (or cyst aspiration) and seminal vesiculography (or cyst dye injection), which provide both static and dynamic data, to determine if these tests confirm or exclude obstruction. This was done in an attempt to define an accurate diagnostic technique for EDO before transurethral resection of the ejaculatory ducts (TURED).

### **Secondary Aim**

This study also investigated the therapeutic potential of TRUS guided techniques in relieving EDO.

***REVIEW OF  
LITERATURE***

# **ANATOMY & EMBRYOLOGY OF THE MALE**

## **DUCTAL SYSTEM**

### **Embryological development of the male ductal system** (Figs.1-3)

By the eighth week of gestation, the ductal system is the same in either sex. It is formed of the mesonephric (*Wolffian*) duct on the lateral aspect of each gonadal ridge and the paramesonephric (*Mullerian*) duct on the lateral aspect of each mesonephric duct. The Wolffian duct ends by opening into the *urogenital sinus* dividing it into an upper part that will give rise to the bladder and a lower part that will give rise to the urethra, prostate and the external genitalia (*Hensle and Seaman, 1995*).

By the 6<sup>th</sup> week of gestation, the Mullerian duct begins to develop in both sexes as a funnel-shaped invagination in the coelomic (peritoneal) epithelium just lateral to and adjacent to the Wolffian duct (*Lawrence, 1992*).

The first event in the development of the male ductal system is Mullerian duct regression which occurs between seven and eight weeks of gestation under the influence of the Mullerian inhibitory factor (MIF) which is secreted by foetal Sertoli cells, and testosterone which is secreted by foetal Leydig cells (*George and Wilson, 1992*).

After local absorption of the polypeptide MIF, the mesenchyme alters the extracellular matrix by increased hyaluronidase activity or fibronectin lysis. These alterations facilitate the breakdown of the Mullerian basement membrane. The steroids: testosterone,

medroxyprogesterone and progesterone where found to augment the effect of MIF in vitro (*Ikawa et al., 1982*).

The Mullerian ducts undergo regression except for two small rudimentary portions: the appendix testis from the cranial end and the prostatic utricle from the caudal end. The prostatic utricle is a blind pouch that opens into the urogenital sinus in the midline, adjacent to the orifices of the ejaculatory ducts (*Maizels, 1998 and Sadler, 2004*).

By the 22<sup>nd</sup> week, the utricle and the ejaculatory ducts expand under the floor of the urethra and elevate the posterior wall of the urethra to form the verumontanum, which causes the lumen of the prostatic urethra to appear semilunar. Later, the utricle contracts to appear as a small structure on the tip of the verumontanum (*Maizels, 1998*).

The Wolffian ducts develop into the efferent ductules, the epididymis, the vas deferens, the seminal vesicle, the ejaculatory ducts, the posterior urethra above the verumontanum, the trigone of the bladder, the ureters, the pelvicalyceal system and collecting tubules. Developmental growth of these structures is stimulated by fetal testosterone which acts directly by local diffusion from the adjacent gonad (*Coffey, 1988*).

Initially, the cloaca is divided by the urorectal septum; into the posterior anal canal and the anterior urogenital sinus. The division is completed around the 7<sup>th</sup> week. The Wolffian ducts are included in the vesicourethral canal within the urogenital sinus. The cranial portion of the Wolffian duct in both sexes is used as the secretory element

(mesonephros), while its caudal part becomes associated with the developing gonad (*Tanagho, 1992*).

The transformation of the Wolffian ducts into the male genital tract begins after the Mullerian duct regression has commenced. The mesonephric tubules of the Wolffian duct establish contact with the developing rete and spermatogenic tubules to form the efferent ductules of the epididymis. Some of the degenerating tubules persist as rudimentary structures known as the paradidymis (appendix epididymis) on the epididymal head (*Tanagho, 1992*).

The portion of the Wolffian duct immediately distal to the efferent ducts becomes elongated, coiled and convoluted to form the rest of the epididymis. The central portion of the Wolffian duct develops concentric layer of mesenchyme (12 weeks), thick muscular wall (28 weeks) to become the vas deferens. At about the 12-13 week of gestation the seminal vesicles begin to develop as buds from the lower portions of the vasa. By the fourth month of gestation, it develops into a highly convoluted, glandular sac which measures about 5cm long, and with simple mucosal epithelium. By the sixth month of gestation, sacculations occur with the appearance of pseudostratified columnar epithelium and musculosa derived from that of the vas deferens (*Maizels, 1998 and Collins, 2005*).

Since the Mullerian and Wolffian ducts are separate it is unlikely that a Mullerian duct cyst would incorporate the EDs or contain sperm. On the other hand it is possible for a Wolffian duct cyst to be devoid of sperm which caused by secondary epididymal obstruction on top of long standing distal obstruction (*Sharlip, 1984*). However, there are reports of