

Diagnosis, Evaluation and Treatment of Adolescent Varicocele

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INTRODUCTION

A varicocele can be defined as an abnormal tortuosity and dilation of the veins of the pampiniform plexus. Idiopathic varicocele is usually asymptomatic. It is noticed as an asymmetry in scrotal size, and presents as heaviness in the scrotum, or rarely with testicular pain. In most cases the adolescent is unaware of the varicocele and it is discovered during a regular physical examination or during examination for military service.¹⁻⁴

The incidence of high-grade varicocele is approximately 5 % throughout the world.⁵ Varicocele is associated with a time dependant growth arrest in adolescents and adult males.⁶ There is a clear association between varicocele, infertility and testicular growth arrest.⁷⁻⁹ It is also known that varicocelectomy can reverse testicular growth arrest in adolescents.¹⁰⁻¹³ These facts have raised the question of how to best manage the adolescents with a varicocele.

Adolescents do not present with infertility and thus should prophylactic repair be performed to prevent infertility in the future? Who would benefit the most by varicocelectomy: the adolescents with testicular growth arrest or any adolescent with a varicocele? Is it better to wait for a semen analysis or offer earlier treatment based on testicular growth arrests? These questions can be only answered when we have better understanding of the pathophysiology of varicocele.

The purpose of this review is to present the current literature on adolescent varicocele and provide guidelines to the clinician how to manage the adolescents with a varicocele.

EPIDEMIOLOGY

In the general population of healthy males the overall incidence of varicocele (all grades) is 10% to 15%.^{4,5,14,15} Approximately 30-50 % of males with primary infertility have a varicocele.¹⁶⁻¹⁹ Varicocele is most common on the left side. Varicocele appears at early puberty however it can occasionally be found in preadolescent boys.^{2,20} The incidence in older adolescence varies from 12.4 % to 17.8 % with an average of 14.7 %, similar to the incidence in adult males. (Table 1.)

TABLE 1
Incidence of varicocele in general population of healthy adolescents.

Reference:	No of patients:	Age:	Incidence: (total)
Oster, 1971 ¹⁰¹	837	10-19	16.2%
Steeno, 1976 ¹⁰³	4067	12-25	14.7%
Yerokhin, 1979 ¹⁴⁶	10000	10-17	12.4%
Belloli, 1993 ⁹	9861	10-16	16.0%
Niedzielski, 1997 ⁵	2478	10-20	17.8%

ANATOMY

The arterial blood supply to the testis comes from the testicular artery, vasal artery and the cremasteric artery. At the level of testis all three arteries anastomose to allow adequate blood supply even with division of the testicular artery.^{21,22}

The venous drainage (Figure 1) is more complicated with many individual variations. Above the testis is a network of communicated veins called the pampiniform plexus (PP), the drainage from PP is via the testicular vein trunci, pudendal veins and cremasteric veins.^{23,24} In most cases the testicular vein trunci form a single testicular vein entering the renal vein on the left and the inferior vena cava on the right. Venographic studies have demonstrated that the left testicular vein can rarely enter the inferior vena cava, and there are communications between the testicular vein and the inferior vena cava below the level of the renal veins.²⁵⁻²⁷

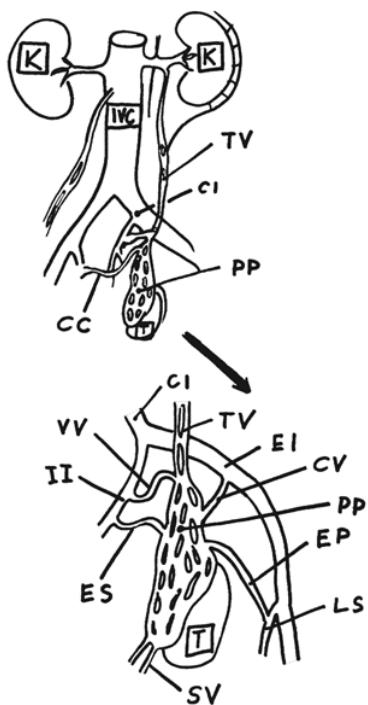


FIGURE 1. Anatomy of venous drainage from left testis.

There are also cross-communications between the left and right testicular venous systems (Figure 2).^{26,28-30}

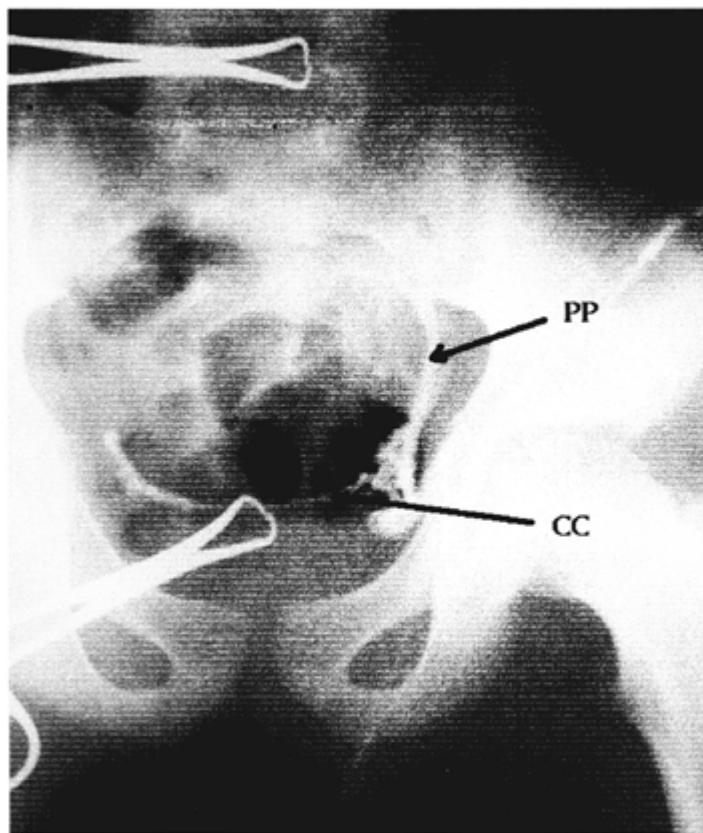


FIGURE 2. Intraoperative venogram showing left to right cross-communicating veins CC-cross-communications, PP-pampiniform plexus veins

ETIOLOGY

There are several theories attempting to explain the etiology of varicocele. The predominance of the left side varicocele and the unique anatomy of the left testicular vein are the basis for several theories explaining the etiology of varicocele.

The presence of venous valves was long believed to be a guarding mechanism against developing a varicocele and incompetence of the venous valve system was thought to be responsible for varicocele development. However, it was shown that there are males without varicocele who have incompetent venous valve system and males with varicocele who had competent venous valves.²⁹

Second, because the left testicular vein is longer than the right, the hydrostatic pressure difference could be a factor causing a left varicocele. Although the left testicular vein is longer than the right, the simple difference in hydrostatic pressure of a standing column of blood can not be the only reason for development of a varicocele because all males would be affected.

A third theory known as a "nutcracker effect" is speculated to occur when the testicular vein is compressed between the superior mesenteric artery and aorta. The increase in hydrostatic pressure results in varicocele formation. However although high left renal vein to vena cava pressure gradients are noted in patients with varicocele, it is not a consistent feature.^{31,32}

More recently it has been hypothesized that increased arterial blood flow to the testis at puberty exceeds the venous capacity resulting in venous dilatation and a varicocele.^{33,34} This is consistent with the findings noted in all animal models studied to date, however confirmation in humans will be necessary.

PATOPHYSIOLOGY

The pathophysiology of varicocele can be studied in animal models by partial ligation of the left renal vein.³⁵ Many features of the human condition, like increased temperature of the affected testis, increased arterial blood flow and histopathological changes can be replicated in animal models.

The following theories attempt to explain the deleterious effect of varicocele on testicular function.

Hyperthermia

The presence of a varicocele is associated with elevated scrotal and testicular temperature and altered spermatogenesis. Experimental studies have shown that spermatogenesis occurs optimally at temperatures lower than body temperature. Many of the enzymes responsible for optimal DNA synthesis in the testis are temperature dependent.^{36,37} The scrotal position of the testis and the cooling system provided by the pampiniform plexus surrounding the testicular artery allows for heat exchange and is responsible for regulating optimal temperature for spermatogenesis.³⁸ Stasis of blood in the varicocele with resultant increased temperature may be responsible for the deleterious effect of varicocele on spermatogenesis.³⁹ Increased temperature is associated with decreased number of spermatogonia and increased apoptosis of germinal epithelium cells.⁴⁰

Hypoxia and "adrenal reflux"

Stasis of blood in pampiniform plexus could affect partial oxygen pressure and change aerobic metabolism in the testis. However hypoxia has not been demonstrated in testicular venous blood sampling in humans or experimental animals.^{41,42} Reflux of blood down the testicular vein has been demonstrated in patients with varicocele.^{43,44} Therefore exposure of the testis to adrenal or renal metabolites is hypothesized as cause for testicular damage. However adrenal or renal metabolites at the level of the testis have not been documented.^{35,45} Adrenalectomy done on rats with experimental varicocele did not diminish the effects of the varicocele.^{46,47} Thus the adrenal/renal reflux theory does not appear to be responsible for the testicular damage associated with varicocele.^{46,48}

Abnormal blood flow

A current hypothesis assumes that increased blood flow through the testis can affect spermatogenesis.^{49,50} An increase in hydrostatic pressure with a change in filtration pressure could considerably alter the composition of the interstitial fluid.⁵¹ This alteration conceivably could alter the intimate paracrine communications between the Leydig cells, peritubular myoid cells and Sertoli cells ultimately affecting spermatogenesis.¹ The myoid cells and capillary epithelium undergo pathological changes in association with varicocele that may effect transmembrane transport of substrates to the germinal epithelium.⁵²

Endocrine imbalance

Puberty, spermatogenesis and testicular development are regulated by the hypothalamic-pituitary-testicular axis. There is a wide array of endocrine abnormalities associated with varicocele.

Leydig cells are under the control of luteinizing hormone (LH) and responsible for testosterone production. Some studies have shown that the serum testosterone level may be affected by varicocele, however it is intratesticular testosterone that is important in regulation of spermatogenesis.^{53,54} In experimental animal models a varicocele can result in decreased intratesticular testosterone level.⁵⁵ The results of human studies are mixed. Ando et al. found reduced serum testosterone level in males with varicocele and increase in serum testosterone level after repair of varicocele.^{56–60} Swerdloff and Walsh however showed that there was no difference in testosterone level between males with and without varicocele.⁶¹

Increased LH serum levels and an abnormal response to gonadotropin releasing hormone (GnRH) could implicate a compromise of the hypothalamic-pituitary-gonadal axis involved in the control of testosterone level and spermatogenesis; a pattern similar to hypergonadotropic hypogonadism.^{62,63} Increased LH level results in Leydig cells hyperplasia; a known histologic finding in varicocele testicular biopsies.^{64–66}

Sertoli cell responsiveness to FSH may be diminished in varicocele patients. Stimulation of Sertoli cells with FSH reversed spermatogenesis arrest in experimental animal models.⁶⁷ Altered levels of serum inhibin found in patients with varicocele may reflect altered function of Sertoli cells.⁶⁸ Cameron et al. noticed that the Sertoli-germ cell junctional complexes appeared to be structurally abnormal in patients with varicocele. They concluded that testicular disruption associated with a varicocele is a phenomenon of the adluminal compartment, and that the Sertoli cell is more sensitive to perturbation of the testicular environment than are germ cells. The Sertoli cell may be the primary intratubular site of alteration leading secondarily to spermatogenic disruption.⁶⁹ Histologic studies of the testis from patients with varicocele showed absent germ cells or altered spermatogonia to Sertoli cell ratio.⁷⁰

Paracrine regulations of the testis

Insight into the detailed mechanism of spermatogenesis is even more complicated as spermatogenesis is also regulated by complex interactions and signals at the cellular level in the testis.¹ (Figure 3)

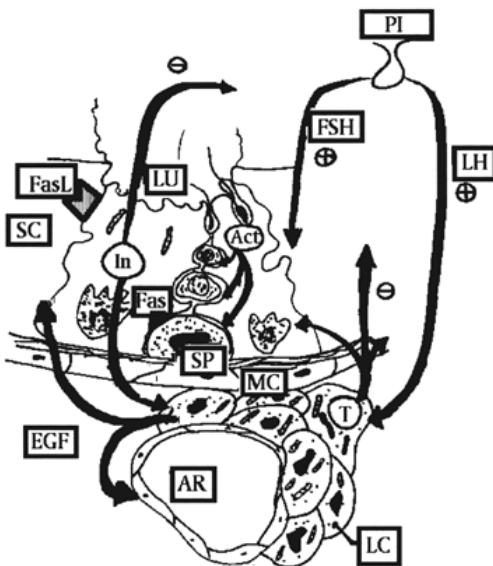


FIGURE 3. Endocrine regulation of spermatogenesis.

Both Sertoli cells (SC) and Leydig cells (LC) regulate spermatogenesis by steroidogenesis and growth factors production.^{71,72}

Sertoli cells, tightly regulate germ cell proliferation and differentiation and are implicated in the control of germ cell apoptosis. Fas (APO-1, CD95), a transmembrane receptor protein expressed by germ cells, transmits an apoptotic signal within cells when bound by Fas ligand (FasL) produced by Sertoli cells. The Fas system has been implicated in immune regulation, including cytotoxic T cell-mediated cytotoxicity, activation-induced suicide of T cells, and control of immune-privileged sites.⁷³ SC stimulated by FSH produce inhibin (In) and activin (Ac).⁷⁴ Inhibin has negative feedback control on pituitary and FSH secretion. Inhibin also binds to Leydig cells (LC) regulating testosterone (T) production. Activin binds to round spermatids and spermatogonia (SP), effecting spermatogenesis. Spermatogonia are known to stimulate transferrin production by SC by an unidentified protein substance.⁷⁵

Leydig cells control spermatogenesis not only by steroids production but also by epidermal growth factors (EGF) which binds to spermatogonia and spermatids regulating cell divisions.⁷⁶ Receptors for transforming growth factor (TGF), one of the EGFs produced by LC, were found in peritubular myoid cells.⁷⁷ Peritubular myoid cells (PC) secrete peritubular myoid cell substance (PmodS) which stimulates SC. LC control adluminal tubular compartment and transport of nutrients from the vascular space to germinal epithelium by vascular endothelial growth factor (VEGF). VEGF is of particular interest in varicocele since VEGF regulates endothelial permeability and is an angioproliferative factor.⁷⁸

Locally produced neurotropins play their distinct role in spermatogenesis regulation.⁷⁹ Opioids receptors are found on LC. During stress, release of endorphins stimulate opioid receptors and decreases testosterone production. Blocking the opioid receptors by naloxone restores testosterone production to normal.⁸⁰

With each discovery of a new paracrine substance, and a better understanding of molecular mechanisms controlling spermatogenesis we come closer to the time when we will accurately predict which adolescent will require surgical or medical interventions for testicular dysfunction.

PATHOLOGY

Testicular hypotrophy

Testicular function most effected by the varicocele is spermatogenesis.⁸¹ The most common findings on semen analysis are: increased number of pathological sperm forms, decreased motility and decreased sperm density.^{8,82,83} Sperm analysis in adolescents with varicocele shows decreased sperm density, increased number of pathological forms and decreased motility however there are no established norms for adolescent semen analysis.⁸² Varicocele is also associated with testicular growth arrest in adolescents.^{10,12,84} Testicular growth arrest may be considered the hallmark of testicular damage in adolescent varicocele. Significant volume loss in adolescents with varicocele has been noted in 77 % of boys, 10% of whom had a left testis one-fourth the size of the right testis.⁸⁵ Testicular hypotrophy is time dependent.^{86,87}

Testicular volume during preadolescents is constant and at the onset of puberty the testis suddenly increases in size even prior to other pubertal changes. In adolescents with a varicocele the rapid growth of the testis between the ages of 11 and 16 is effected by the varicocele and results in a volume discrepancy between the right and left testis.

HISTOPATHOLOGY

Testicular biopsy in males with varicocele shows a wide array of abnormalities. The most common findings are Leydig cells hyperplasia, decreased number of spermatogonia per tubule, spermatogenesis arrest and sloughing of germinal epithelium.⁸⁸⁻⁹² A thickened basement membrane of seminiferous tubules

and proliferative lesions of endoepithelium are often demonstrated and may affect transportation of oxygen and glucose through these structures.^{93,94}

DIAGNOSIS

Since the adolescent with a varicocele is often asymptomatic it is usually found on routine physical exam. The patient should be examined standing in a warm room to relax the scrotum and allow easier examination of the spermatic cord. The scrotum is first visually inspected for any obvious distention around the spermatic cord; a visible varicocele is considered a large or Grade 3 varicocele. The scrotum, testes, and cord structures are then gently palpated. A palpable varicocele has been described as feeling like a bag of worms or a squishy tube. More subtle varicoceles may feel like a thickened or asymmetric cord. The nonvisible, but palpable varicocele, is considered to be moderate in size (Grade 2). If a varicocele is not palpable but the patient performs a Valsalva maneuver which distends the pampiniform plexus of veins, then a small (Grade 1) varicocele is present. After examining the patient in the standing position, the patient should be examined supine. A thickened cord due to a varicocele should resolve in the supine position, whereas a thickened cord due to a lipoma will not change when the patient is supine. Secondary varicocele especially on the right side should always be excluded since it can be caused by serious conditions like retroperitoneal tumors, kidney tumors or lymphadenopathy.⁹⁵ Idiopathic varicocele is more prominent in the upright position and disappears in the supine position. A secondary varicocele does not change its size so dramatically in the supine position.

Testicular size needs to be measured to determine if the varicocele is adversely affecting the growth of the testis. The volume of a normal testis measures 1 to 2 ml in the prepubertal male. Due to extensive individual variation in normal growth and development, testicular size is correlated with Tanner Stage, growth velocity, and bone age rather than chronological age.¹

A number of methods have been used to measure the size of the testis. These include visual comparison, rulers, calipers, comparative ovoids (Prader Orchidometer), punched-out elliptical rings (Takahara Orchidometer), and ultrasound. A high correlation ($r = .992$) between ultrasound and actual volume was noted and was shown to be highly reproducible.⁹⁶ The Prader Orchidometer was shown to correlate with ultrasound measurement in 256 patients ($r = .91$), though the degree of correlation was dependent upon the investigator's clinical experience. In a clinical study of 22 male adolescents with a varicocele, 24% of patients with growth arrest would have been missed if measured by Prader Orchidometer alone, and three patients felt to have a significant size discrepancy (>2 ml) by Prader Orchidometer measurements were found to be normal by ultrasound volume estimate. These findings indicate that clinical estimates of testicular size by the Prader Orchidometer are not as accurate or reproducible as those determined by ultrasound. Accurate measurement is important because operative decisions rest in the balance.⁹⁷

There is significant disagreement as to what constitutes a significant size discrepancy justifying surgical intervention. Testicular ultrasound is the most accurate and reproducible method to assess testicular volume and significant testicular size variations. A volume difference of less than 2 ml can be due to the measurement technique alone. Therefore, size variation of greater than 2 ml by ultrasound is currently the best indicator of testicular damage and should serve as the minimal requirement for surgical repair of the adolescent varicocele.¹ Surgical intervention reverses testicular growth arrest and assessment of testicular volume postoperatively predicts resolution of the varicocele.⁹⁸

MANAGEMENT

There are some cardinal questions to be answered regarding management of adolescent varicocele.

Is it justified to promote the awareness about varicocele among pediatricians and primary care providers and to look for varicocele in asymptomatic adolescents?

There is sufficient evidence that varicocele is associated with testicular growth arrest in adolescents and varicocelectomy results in testis "catch-up" growth.^{8,10} Lenzi et al. showed that early varicocele repair in adolescents resulted in better semen analysis results than in untreated adolescents after 2-8 years of follow up.¹³ Based on these studies it seems justified to encourage non-urologists to look for varicocele in the adolescents during physical examination. Examination of the genitalia at puberty also allows the clinician to find other urologic abnormalities like cryptorchidism, hernia, curvature of penis and improve the health of adolescents.⁹⁹⁻¹⁰²

Once a varicocele has been found, what information needs to be given to the patient and his parents?

A number of psychological reactions (anxiety, depressed mood) were experienced in approximately 30% of boys who were informed about varicocele.¹⁰³ Since the word "infertility" is often associated with sexual impairment we believe that during discussion with the patient and his parents the only fact which should be stressed is that the varicocele may result in a decrease in testicular volume that can be reversed by surgical treatment. However it is hard to discount the association between varicocele and infertility in this era of common accessibility to the medical literature on the Internet (we found more than 50 World Wide Web pages searching for the term "varicocele", all had the word "infertility" in the text). Since there are studies which demonstrate an abnormal semen analysis in adolescents it seems advisable to discuss all the findings first with the parents who can be helpful in presentation of the problem to the child.^{104,105}

Once diagnosed, who should we treat? Varicocele is the most common correctable cause of male infertility.¹⁰⁶ A recent metaanalysis of the literature done by Pryor and Howards showed that two thirds of patients will have improvement in semen analysis after varicocele repair, and 40 % of partners will become pregnant.¹⁶ Historically adolescent varicocele was left untreated since its relation to infertility was not well established. Subsequently Kass and Belman showed that testicular growth arrest could be reversed by varicocelectomy in adolescents.¹⁰⁷ Repair of varicocele reverses not only the growth arrest but also improved semen analysis in adolescents and young males.^{105,108,109}

There is good evidence that with time, if left untreated, the varicocele will continue to effect testicular growth with progressive loss of volume and progressive deterioration in semen analysis.^{6, 84} Goldstein suggests that varicocele causes a progressive decline in fertility and that prior fertility in men with varicocele does not predict resistance to varicocele induced impairment of spermatogenesis in the future so called "secondary infertility".⁶

The association between varicocele and infertility is well established.⁸ The most difficult question is which clinical test should we use to establish the indications for surgical treatment in an adolescent with a varicocele.

Currently the clinical tests used to establish indications for varicocele repair are:

- Grade of varicocele
- Measurement of testicular volume to assess testicular growth arrest.
- Gonadotropin releasing hormone (GnRH) stimulation test
- Measurement of pampiniform veins diameter
- Serum luteinizing hormone (LH), follicle-stimulating hormone (FSH) and inhibin levels

Varicocele grade

Varicocele grade does not correlate well with abnormal spermograms or infertility in adults.¹¹⁰ There are different opinions regarding correlation between grade of varicocele and degree of testicular hypotrophy in adolescents. Lyon and associates found no correlation of varicocele grade and testicular size in 30 adolescents.⁸⁵ In contrast Skoog, Steeno, and Paduch all independently noticed that boys with severe varicocele have a smaller ipsilateral testicle.^{10,97,103} It was also noticed that the smaller the testis the worse the semen analysis results.^{82,8} However grade of varicocele by itself should not be the sole indication for treatment.

Testicular volume

There is an abundance of literature confirming that varicocele is associated with testicular growth arrest in adolescents and varicocele repair results in testicular "catch-up" growth.^{10,105,108,109} Testicular growth arrest with volume difference of more than 2 ml assessed by ultrasonography is the most common indication for treatment.¹⁰ The development of secondary infertility is another strong argument for early varicocele repair as if left untreated the varicocele will not only effect testicular volume but also effects spermatogenesis.⁶ A decrease in testicular volume is the best indicator for surgical correction of a varicocele. However, not every boy with a varicocele and testicular growth arrest will be infertile and there is still a need to search for a test which would better distinguish between adolescents with varicocele who will develop infertility and those who will remain fertile. In adult males the situation is a little simpler because the indications for surgery are usually established after 12 months of infertility confirmed by abnormal semen analysis and the presence of a varicocele. Obtaining a semen sample in adolescents is possible but difficult.

Gonadotropin releasing hormone (GnRH) stimulation test

Damage to germinal epithelium results in compensatory stimulation of the pituitary gland and subsequent increase in FSH and LH production by gonadotrophs.¹¹¹ Intravenous administration of GnRH stimulates the pituitary gland to release FSH and LH. FSH levels are elevated in any condition (like varicocele) effecting the integrity of germinal epithelium.¹¹² In theory GnRH stimulation test could distinguish between adolescents with a varicocele who have abnormal testicular functions and those who have normal spermatogenesis.¹¹ However clinical practice showed that the GnRH stimulating test is expensive, requires multiple serum samples and lacks the association between abnormal results, growth arrest and infertility.^{113, 114} An abnormal GnRH stimulating test was found in 30 % of adolescents with varicocele and was not correlated with atrophy or infertility.¹¹⁵ Currently it seems that GnRH stimulation test has limited role in clinical evaluation of adolescent with a varicocele.

Pampiniform plexus veins diameter

Ultrasonographic measurement of pampiniform plexus veins diameter (PPVD) has been used to look for subclinical varicocele in adult infertile population when physical exam is inconclusive and to follow persistent varicocele but PPVD measurements are not useful in adolescents.^{116,117}

Inhibin level

The serum inhibin levels reflect the integrity of the seminiferous tubule. However, there is not enough data to use serum inhibin levels in clinical decisions at this time.^{68,74,118-120}

Currently, prophylactic surgery for every adolescent with varicocele is not advised since it would result in treatment of 15 % of adolescents.

In summary it seems that treatment should be offered to:

- adolescents with testicular growth arrest (2 SD from normal testicular growth curves, more than 2 cc of difference between left and right testicle)
- adolescents with abnormal semen analysis with high-grade varicocele
- adolescents with symptoms: pain, heaviness, swelling
- adolescents with bilateral varicoceles

TREATMENT OPTIONS

Treatment options in the management of adolescent with a varicocele originated from the practice of adult male infertility and subsequently were applied to the treatment of adolescents.

Varicocele repair can be done by surgical ligation and division of testicular veins or intravenous embolization of testicular veins.

Three open surgical approaches are currently used: subinguinal approach (Marmar), inguinal approach (Ivanissevich) and retroperitoneal approach (Palomo). Laparoscopic varicocele ligation has often been used in adults. Embolization techniques, regardless of embolizing material, can be divided into: antegrade (infusion through scrotal part of pampiniform plexus veins) and retrograde (catheter placed through femoral vein puncture) infusion.

The failure rate, frequency of complications, cost and outcome are important factors which need to be evaluated in choosing preferable treatment option in adolescent repair.(Table 2) It is important to remember that the majority of studies on varicocele repair relates to adult infertile males with varicocele and not adolescents.

TABLE 2
Surgical approach, complications and relative costs of varicocele repair.

Technique	Hydrocele rate	Failure rate	Cost	References
Retroperitoneal	7-10 %	9-11 % (artery sparing) <3 % (artery taking)	Low	121 10 12
Inguinal	3-7 %	9-12%	Low	123 129 147
Inguinal microscopic	< 1%	2.1 % 0.6%	Moderate	124 125
Laparoscopic	1.25 %	9 % 1.25 % in adolescents	High	148 149
Embolization		19 %	High	135

FAILURE RATE

Failure of treatment is defined here as a persistent or (rarely) recurrence of the varicocele after the repair and can occur in 9 % to 16 % of adolescents.¹²¹ Persistence of the varicocele results in lack of "catch-up" testicular growth.¹² Most authors attribute the high recurrence rate to missed venous collaterals which run parallel to the main testicular vein. The collaterals can be quite difficult to identify and ligate separately from the testicular artery. Reported persistence rate using artery sparing retroperitoneal approach ranges from 3% to 11%.^{12,121-123} Ligation of both testicular vein trunci and the artery has the advantage of a decreased persistence rate and does not result in testicular atrophy since the testis has collateral arterial blood supply from the cremasteric and deferential artery.^{12,21} Atassi and colleagues achieved a persistence rate below 2 % in adolescents treated by high retroperitoneal ligation with testicular artery ligation.¹²

There is, however, some objection to simultaneous ligation of the testicular artery in men with previous inguinal surgery since there may be compromised blood supply from the cremasteric and deferential arteries. Interruption of the testicular artery in these patients has a high probability of

developing testicular atrophy. Also, subsequent vasectomy in patients with testicular artery division should be avoided since ligation of the vasal artery could result in testicular atrophy in the absence of the testicular artery.

The high persistence rate and postoperative hydrocele rate resulted in the development of microsurgical inguinal approaches for correction of the varicocele. Both subinguinal and inguinal microsurgical repair are used quite often in adults with varicocele and indeed offer lower persistence rates and a low incidence of postoperative hydrocele.^{124–126} The low persistence rate using the microsurgical inguinal repair is attributed to ligation of all distended vein trunci and collaterals at the level of the deep inguinal ring.^{124–126} Experience in microsurgical inguinal varicocele repair in adolescent is limited. Reports by Minevitch and Goldstein demonstrated a significantly lower rate of persistence and postoperative hydrocele.^{127,128} The microscopic approach allows one to ligate only the veins leaving the lymphatic vessels intact what decreased the postoperative hydrocele rate to less than 1%.^{125,126}

Laparoscopic varicocele repair with, and without artery sparing modifications seems to be suitable treatment technique especially since recent reports in adults showed a lower rate of persistence.^{129–131} Laparoscopic surgery in the pediatric population is gaining acceptance but it bares the risk of significant complications like bowel perforation, major vascular injury, pneumothorax, and incisional hernia. There is not much experience with laparoscopic varicocele ligation in the adolescent population.^{132–134}

Retrograde embolization, unfortunately is associated with an unacceptable high rate of persistence and is the most expensive of treatment techniques.¹ Possible explanation of such a high persistence rate of the varicocele after embolization can be attributed to the highly variable anatomy of the testicular venous drainage and technical difficulties.¹³⁵ Antegrade embolization is more often used to treat persistent varicoceles than as an initial treatment.^{136–139}

Other options to decrease the rate of varicocele persistence are intraoperative venography and methylene blue injections.^{27,123,140,141} Intraoperative venography in theory should facilitate ligation of all testicular vein trunci and decrease the rate of persistence. Hart recommends routine use of intraoperative venography because 16% of their 62 patients had missed venous vessels after initial venous ligation.¹⁴² Similar conclusions, based on a decreased persistence rate, were also made by Levitt, Zaontz and Gill.^{143–145} However Palmer and Kass reported no difference in their rate of varicocele persistence after repair with and without intraoperative venography.^{121,123,141} Based on these studies intraoperative venography offer little benefit in the repair of adolescents with a varicocele.

Based on our review of the literature and the results of a survey of pediatric urologists in the United States high retroperitoneal ligation of the testicular artery and veins offers the best results in adolescents. Although currently the high retroperitoneal approach is a treatment of choice in adolescent varicocele, pediatric urologists should consider using the microscopic inguinal or subinguinal approach with arterial preservation.

CONCLUSIONS

The adolescent with a varicocele presents the clinician with an interesting and challenging problem. There is a great need for further basic research to help better select the patient who needs surgical correction of his varicocele.

We have outlined recommendations which can be used in everyday practice. Each clinician needs to make his/her own decisions regarding who, when and how to treat the adolescent with varicocele.

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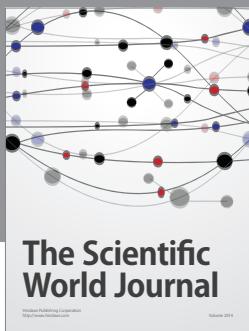
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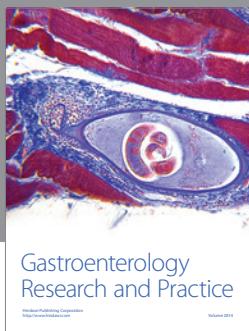
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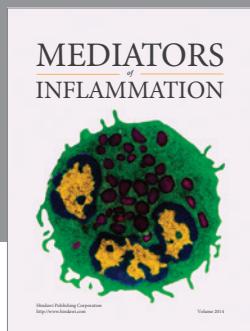
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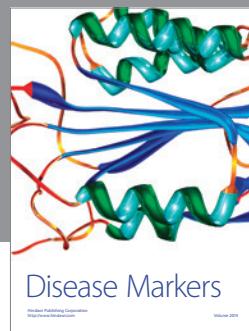
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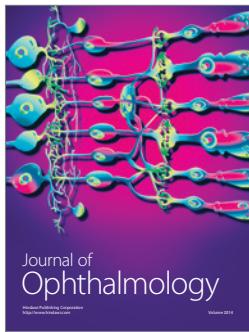
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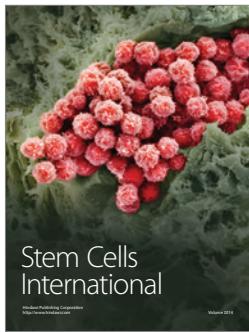
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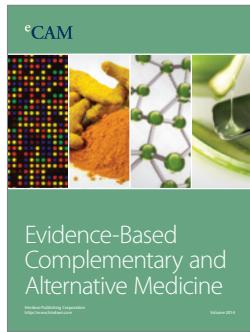
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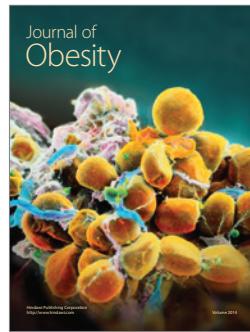
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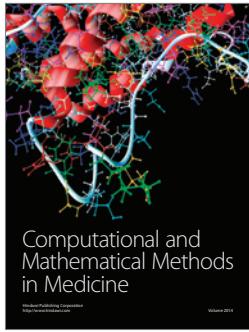
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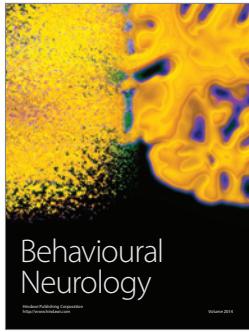
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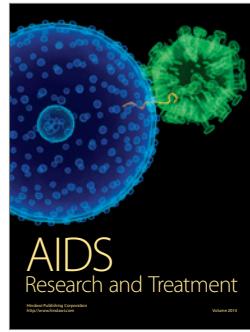
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