The subject of adolescent varicocele has gained increasing attention by pediatric urologists over the past decade. Through prior work by our adult colleagues and clinical studies in adolescents much has been learned. But the adolescent varicocele remains a mysterious abnormality in many respects. Among the unsolved mysteries are whether or not adolescent varicocele represents a truly “progressive” lesion, why growth is impaired in only some involved testes, and the uncertainty as to which boys truly benefit from surgical correction of their varicocele.

In this issue of the Dialogues we have assembled the writings of five experts in the field to address some of the principles behind our current understanding and management of the adolescent varicocele, including its clinical significance, pathophysiology, optimal imaging, indications for surgery and surgical techniques.

We hope that you find this issue useful in the care of your patients and perhaps even an inspiration to help contribute to our understanding of adolescent varicocele.

As a resident in the research laboratory, I was assigned the project of trying to develop an experimental model to study the varicocele. We used a pig as our experimental animal and partially ligated the left spermatic vein to increase the blood volume and stasis around the testicle. We then measured substances such as serotonin, epinephrine, and norepinephrine in the testicular vein to determine whether these were elevated as a result of the partial ligation. Unfortunately, our findings were not consistent but it did peak my interest in varicocele and I have followed the literature closely since.

The exact mechanism by which varicocele affects fertility remains elusive, however, the causative relationship seems to be well documented. Although medical evidence is not definitive in the recommendation that an adolescent varicocele be corrected when ipsilateral testicular growth is impaired, there appears to be ample circumstantial evidence to support that recommendation.

This issue of the Dialogues in Pediatric Urology has put together a panel of experts in varicocele, and in particular adolescent varicocele. The contributors discuss the etiology, radiographic evaluation, perspective from the adult andrologist, and surgical correction. This will serve as a reference for the topic of adolescent varicocele for years to come.

I applaud Dr. Diamond and his contributors for putting together a comprehensive review of the topic.
The Adult Urologist’s Perspective on Management of Varicoceles in the Pediatric Population

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For both ethical and practical reasons, studies of semen parameters in the adolescent population are limited. Furthermore, the normal ranges of semen parameters in adolescents are not known. Studies examining semen parameters in adolescents with varicoceles have not consistently demonstrated worse parameters than in adolescents without varicoceles.15, 35 It has been postulated that semen parameters may not be entirely affected until adulthood. Even so, similar to findings in adults, the available data demonstrates significantly lower sperm concentration and motility in Tanner V adolescents with varicoceles and testicular volume differentials greater than 20%, than in those without hypotrophy6, 9. Because of this correlation between semen parameters and varicoceles in adults, several authors have recommended annual semen analyses, when possible, in adolescents as well.

The relationship of varicocele size to its impact on testicular function is less clear. In the adult population, larger varicoceles are associated with lower sperm concentration than smaller varicoceles, and repair of larger varicoceles leads to greater improvement in this parameter.23 Testicular hypotrophy also becomes more common with increasing varicocele grade in adults.30 In adolescents, a direct correlation between varicocele grade and semen parameters has not been observed, while studies of the relationship between grade and hypotrophy have been conflicting.2, 9, 32 Thus, varicocele grade alone is not an indicator for surgical repair.

There is some evidence to suggest that the persistence of a varicocele causes progressive adverse effects on the testis. The majority of this evidence results from studies reporting a significantly higher prevalence of varicoceles in men with secondary infertility than primary infertility. Limited data in adolescents has not found an increased frequency of hypotrophy over time.10 Of note, the prevalence of hypotrophy in adult infertile patients with varicoceles is significantly higher than that reported in adolescent series, supporting the concept of varicocele damage being progressive over time.8, 30

Specific endocrine imbalances and histopathologic findings have been noted in individuals with varicoceles. These include increased testicular oxidative stress and sperm DNA fragmentation.7, 27 A subset of patients manifest an increase in pituitary LH and FSH secretion after GnRH administration, implying a compromise of the hypothalamic-pituitary-gonadal axis. Recent data has also demonstrated a correlation between the GnRH test, testicular hypotrophy, and pathologic semen analysis.14 However GnRH stimulation has not yet been shown to be helpful in identifying adolescents at risk for future infertility.

Based on available data, semen analysis and hypotrophy remain the best primary and secondary outcome parameters, respectively, for the assessment of future infertility among adolescents.

Indications for Repair

According to the current guidelines of the American Society of Reproductive Medicine and the American Urological Association, varicocele repair in adolescents should be considered when there is objective evidence of reduced ipsilateral testicular size. In the absence of objective evidence, patients should be followed with annual ultrasounds or semen analyses in order to detect the earliest signs of varicocele-related testicular injury.4, 5

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Treatment Options

Surgical options for varicocele repair include the traditional inguinal (Ivanissevich) or high retroperitoneal (Palomo) approaches, laparoscopic repair, and subinguinal microsurgical repair. Varicocele embolization is a non-surgical option. Complications of repair include hydrocele formation, persistence or recurrence of varicocele, and rarely, testicular atrophy. It is well accepted that subinguinal microscopic varicocelectomy is associated with the lowest post-operative complication rate. Post-surgical semen parameters and fertility rates of microsurgical repair are comparable to open inguinal and laparoscopic varicocelectomy. The microsurgical approach has also been used in the pediatric population with excellent results, no recurrences, and a low hydrocele incidence of 1%.  

Results of Repair

Varicocele repair is only worth pursuing if it ultimately protects or improves fertility. Since current indications for varicocele repair in adolescents are based on the belief that hypotrophy and impaired semen parameters are indicative of potentially impaired future fertility, it is reasonable to ask if varicocele repair has any effect on these parameters. Three randomized controlled trials of varicocele repair in adolescents have clearly demonstrated that catch-up growth only occurs in the varicocele treatment groups.  

The effect of repair on semen parameters has also been examined. In an uncontrolled retrospective study, better semen parameters were reported in adults who underwent varicocele repair in adolescence versus those who did not undergo repair. More significantly, two randomized controlled studies demonstrated improvement in semen parameters only after varicocele repair. One hundred percent paternity after varicocele repair has been shown in a cohort of young orthodox Jewish men who underwent unilateral or bilateral surgical varicocele repair during adolescence. Unfortunately, the study design does not allow for a cause-and-effect relationship to be established, and whether fertility was actually improved in these patients after the procedure remains unproven.

Whether repair during adolescence is superior to repair during adulthood is unclear. A recent study compared the pre- and post-operative semen parameters among adolescents and fertile and infertile adults with varicocele. The degree of improvement in sperm count, motility, and morphology was not significantly different between the three groups. The adolescent group had overall better endpoints, likely because they had better baseline semen parameters than the adults. A clear advantage to early varicocele repair in adolescence was therefore not demonstrated.

Management

Based on current evidence, abnormal semen parameters and testicular hypotrophy remain the only reasonable parameters potentially predictive of future fertility. The management of varicoceles in the pediatric population, therefore, centers on the presence or absence of hypotrophy and the results of semen analyses, when available.

All children with varicoceles should have an annual assessment of testicular size. The development of ipsilateral testicular hypotrophy is an indication for varicocele repair. In the absence of demonstrable hypotrophy boys should continue to be followed on an annual basis until they are older and Tanner V of sexual development, when a semen analysis can be obtained or hypotrophy develops. As in adults an abnormal semen analysis with or without hypotrophy is an indication for varicocele repair.

The management of varicocele in the setting of ipsilateral hypotrophy but normal semen analysis is more controversial. Whether observation or repair is better in this population has not been established. Some advocate immediate repair while others advocate observation and then repair only if hypotrophy persists until age 17-20. This observation approach is based on the finding that catch-up growth has been demonstrated at least until this age group whereas varicocele repair after this age may not reverse testicular hypotrophy. Prolonged observation of such patients in adulthood is uncommon, but decisions must be made on an individual basis. Hopefully in the future, better markers of future fertility will allow more accurate differentiation between those that need varicocele repair and those that do not.

References


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Varicocele, abnormal dilation of the spermatic veins, especially the pampiniform plexus, is usually left-sided and occurs in ~15% of the general teenage and adult male population but in ~35% of infertile males. This doubling of the incidence of the lesion in infertile men has caused many to infer that the lesion is a significant cause of infertility; however, the fact that only ~5% of the male population is infertile means that 80-85% of men with varicocele exhibit no effects on fertility. This has raised questions about varicocele as a singular cause of infertility; nevertheless, a number of controlled studies have now shown varicocelectomy to be associated with significant improvements in patient fertility. These and similar studies remain under debate, and this clinical controversy has been the background against which basic science investigations into the pathophysiology of varicocele have been conducted. Major issues in that pathophysiology are discussed below.

The Etiology of Varicocele

In 1957, Campbell’s Principles of Urology explained the development of varicoceles as being “...generally induced by faulty sexual hygiene and especially habitual masturbation, repressed sexual desire, or habitual unhomely sex thinking, which maintains a more or less constant pelvic congestion...” Presently, a more enlightened view prevails, but even current theories about the source(s) of varicocele remain largely unsubstantiated. Those theories are:

1. Hydrostatic pressure. The left spermatic vein inserts into the left renal vein whereas the right spermatic vein inserts into the inferior vena cava. This asymmetry is due to a feature of the spermatic veins’ development from the embryo’s cardinal vein system, which leaves the left spermatic vein somewhat longer and more completely vertical than the right. From this, it has been suggested that there is a higher hydrostatic pressure in the left spermatic vein than in the right. This hypothesis has not been directly tested, but the same geometry applies to all men; thus, if the hydrostatic pressure hypothesis is true, why do not all men develop the varicocele? At a minimum, the fact that they do not implies that some other condition must exist for the testicular veins to varicocele in some men but not in others.

2. Inadequate venous valves. It has been proposed that an absence of valves or the presence of incompetent valves in the left spermatic vein contributes to the left varicosity. This hypothesis has not been directly tested, but the same geometry applies to all men; thus, if the hydrostatic pressure hypothesis is true, why do not all men develop the varicocele? At a minimum, the fact that they do not implies that some other condition must exist for the testicular veins to varicocele in some men but not in others.

3. Another hypothesis that could account for specifically left-sided varicocele is the so-called “nut-cracker” phenomenon.
vascular event that occurs when the left renal vein becomes trapped and compressed between the overlying superior mesenteric artery and the underlying aorta. That compression results in a partial obstruction of the left renal vein that is medial to the insertion of the left spermatic vein and causes increased intravascular pressure in the venous tree lateral to the compression, which includes the left spermatic vein. Over time, this can cause a left varicocele vascuosity to develop. Pediatric patients might be particularly susceptible during puberty as the testes mass and testicular blood flow develop concomitantly. When the nutcracker condition is mimicked in laboratory animals by a partial ligation of the left renal vein, a left varicocele does develop and that procedure has been widely used for the study of the pathophysiology of varicocele.

The Pathophysiology of Varicocele

As with the etiology of varicocele, discussed above, a number of hypotheses have been advanced regarding the pathophysiology of varicocele.

1. Testicular bloodflow: The most most common finding and best documented in laboratory animals is that varicocele increases testicular blood flow. This may appear counterintuitive since a unilateral increase in vascular resistance on the left side, which should occur due to the restriction of venous flow in the left renal vein, should decrease testicular blood flow on the left side. It has been suggested that the increased collateral circulation that develops along with the varicocele allows for the increase in flow. Only two blood flow studies have been conducted in human males with varicocele. Neither study found statistically significant increases in testicular blood flow, but both demonstrated trends in that direction. The lack of uniformity of the lesion in the patient population may be reason for the statistically insignificant findings.

2. Testicular temperature: Early studies of scrotal surface temperature in the human suggested that varicocele causes an increase in testicular temperature. Studies using rat, dog, and monkey models have subsequently demonstrated bilateral increases in intratesticular temperatures and this has been documented in the human, as well. An increase in testicular temperature is consistent with the aforementioned finding of increased testicular blood flow in patients with varicocele. An increase in testicular blood flow decreases the efficiency of the countercurrent heat exchange mechanism in the pampiniform plexus and allows warmer blood to reach the testis, which increases intratesticular temperature.

3. Reflux: Reflux of blood from the left renal and adrenal veins down the left spermatic vein to the testis is often presumed a priori to be a part of the varicocele condition because scrotal vessels fill during a Valsalva maneuver and because radiographic evidence can show contrast agents flowing from the distal left spermatic vein in a retrograde fashion down toward the testis. It is often overlooked that retrograde flow is not the only explanation for scrotal filling during Valsalva or with rising to an upright position. Further, proper perfusion technique is essential in venographic studies of blood flow and the details of technique are rarely, if ever, presented. It is also important to note that radiographic contrast media have a much higher specific gravity than blood and can gravitate downward against the direction of positive left spermatic vein blood flow.

Studies of experimental left varicocele have shown that the testicular pathology of varicocele occurs in the absence of venous reflux and that reflux occurs as commonly in control human male subjects as in men with varicocele. Further, reflux would be expected to bring renal or adrenal products to the testis or at least to the proximal testicular vasculature, but to our knowledge this has not been shown to happen. Also, adrenal products play no role in the testicular effects of experimental varicocele in monkeys. All these data suggest that reflux is not an obligate part of the pathology of varicocele.

4. Endocrinopathy: Endocrine effects of varicocele are controversial. Studies in animal models have shown bilateral decreases in intratesticular testosterone, but the models have given inconsistent results regarding where along the hypothalamo-hypophyseal-gonadal axis a deficiency might exist. At a minimum, findings from the varicocele models and the histology of adolescent varicocele testes suggest some form of Leydig cell dysfunction leading to an intratesticular androgen environment inadequate for the full support of Sertoli cells and spermatogenesis.

5. Autoimmunity: Shook et al reported that unilateral left varicocele in the rat causes a higher serum antisperm antibody titer than in control animals. It is difficult to say how such antibodies arise or have access to sperm when it has been shown in both rats and humans that the blood-testis barrier remains intact under conditions of varicocele. Studies in human patients have found both for and against a correlation between antisperm antibodies and varicocele, but the conclusion consistent with the continued presence of the blood-testis barrier is that the underlying pathology of varicocele is not an autoimmune one.

6. Oxidative stress: Testicular oxidative stress has been reported in the testes and semen of varicocele patients. The increased testicular blood flow and increased testicular temperature discussed previously may play a role in testicular oxidative stress since an increase in blood flow and temperature may mimic a mild inflammatory condition. Also, Ozdamar et al have found that experimental varicocele in the rat is associated with a decrease in antioxidant capacity of the rat testis, and Hendin et al found similarly studying the semen of varicocele patients. Interestingly, increases in NO and lipid peroxidation have been linked in both human varicocele patients and rats with experimental varicocele, which implies that peroxinitrite, a relatively harsh oxidant, plays a role in the stress of varicocele.

Conclusion

The strongest line of evidence suggests that varicocele induces a bilateral increase in testicular blood flow, temperature, and oxidative stress, which in turn alters the activity of specific enzymes in the steroidogenic pathway in Leydig cells. This, in turn, reduces intratesticular testosterone, ultimately causing a reduction in spermatogenesis. It is unlikely that reflux of blood down the spermatic vein has a significant role in varicocele or that varicocele causes an increase in antisperm antibodies. The fact remains, however, that both the varicocele lesion and its effect on male infertility are highly variable.
Obvious questions remain, e.g., how does the unilateral varicocele cause a bilateral testicular response? Studies in the rat model have shown that the bilateral effect is not mediated through the ipsilateral testis, but rather the contralateral testis. However, the ipsilateral testis is not affected by surgery itself. This suggests that the lesion has the potential to threaten testicular growth and spermatogenesis in adolescents and post-pubertal boys. The incidence of varicocele is vanishingly small in pre-pubertal boys but increases through the adolescent years and reaches an incidence in postpubertal boys approximating that of adults. Since 80% of adults appear unaffected by their varicocele, pediatric urologists are faced with the challenge of deciding which boys with varicocele will benefit from surgical intervention and which will not.

The most widely accepted criterion for surgical intervention has been atrophy or impaired growth of the ipsilateral testis. Defining “impaired growth” has been relatively subjective with some investigators using an absolute volume differential of 2ml and others suggesting a relative volume differential of 10%. Another confounding factor is that a small percentage of normal boys exhibit asymmetric timing of testicular growth during puberty; thus, some specialists suggest observing a mild difference of testicular volume for 6-12 months before considering surgery. That period of watchful waiting should allow the urologist to decide if the physiological factors relevant to varicocele discussed here, i.e. testicular blood flow, temperature, oxidative stress, and endocrine response, have a sufficient cumulative effect in a particular patient to be a threat to testicular development.

References
Varicocele is most commonly diagnosed by physical examination. However, physical examination may be unreliable in the presence of a hydrocele, thickening or scarring of the scrotal tissues. The term “sub-clinical varicocele” refers to a varicocele that is not palpable on physical examination and requires imaging for diagnosis. The American Urological Association and the Practice Committee of the American Society of Reproductive Medicine (ASRM) currently recommend that treatment be restricted to palpable varicoceles. At this time, there is no consensus regarding the diagnosis of subclinical varicocele, although venography is most commonly accepted as the gold standard for the diagnosis of reflux into the testicular vein. However, venography is invasive and is generally reserved for patients undergoing sclerotherapy.

The venographic diagnosis of varicocele was first described by Ahlberg et al in 1966 and has been employed by others with minor variations.12 Diagnosis is based on selective retrograde venography of the renal and testicular veins. Using a transfemoral approach, a catheter is placed into the left testicular vein at a point just beyond the valve most proximal to the left renal vein. Five to 10cc of contrast medium are then manually injected through the catheter as the patient performs a Valsalva maneuver. When the valve at the orifice of the testicular vein is absent or incompetent, contrast medium flows from the renal vein inferiorly into the testicular vein; i.e., the direction of physiologic flow is reversed. If no reflux into the testicular vein is detected, the left renal vein is examined for collateral venous insufficiency from the segmental renal veins laterally to the inferior vena cava (IVC) medially. In the absence of left-sided reflux, the right renal vein, the IVC and the left common iliac vein are studied with retrograde venography to identify right-sided reflux.3

One concern regarding the venographic diagnosis of varicocele is that venous reflux may occur under conditions of variable injection pressure and catheter tip placement. The catheter tip may be located beyond the valve or may interfere with its function, and thus may not reflect the usual physiologic condition in a particular patient.1 Further, injection into the renal vein may also not represent the actual physiologic condition under which a patient may reflux into the spermatic vein, particularly when the patient is not fully erect.

In addition to venography, a number of noninvasive imaging modalities have been used in an attempt to more accurately diagnose clinically significant varicoceles, including scrotal contact thermography, radionuclide imaging and color Doppler ultrasound (US). Color Doppler US is currently the most widely employed and accepted of the noninvasive imaging modalities.

By US a varicocele consists of multiple serpiginous, anechoic structures greater than 2 mm in diameter located adjacent or proximal to the epididymal head and upper pole of the testis.5 Scrotal US is performed using a high-frequency transducer and low-flow Doppler settings in order to optimize the detection of slow-flow within the varicocele. Augmentation of venous flow is achieved with the patient in the upright position or during performance of the Valsalva maneuver. Flow augmentation is especially well demonstrated with color Doppler (Figure).

Color Doppler US has been touted as a more diagnostically accurate technique than physical examination.6,7 However, there is currently no consensus as to which US measurements should be used in the diagnosis of varicocele.8 The presence of multiple veins in the pampiniform plexus greater than 2 to 3 mm in diameter with reversal of flow with the Valsalva maneuver are the most commonly described diagnostic criteria.9,14 This variability within the literature regarding size criteria for diagnosis of varicocele is a reflection of the absence of any well-established normative measurements. To date, there have been no clinical trials where pre- and postoperative spermatic vein measurements have been correlated with semen parameters and subsequent fertility. Many clinicians also believe that color Doppler US documentation of flow reversal with the Valsalva maneuver is essential to the diagnosis of varicocele.11,14 However, flow reversal with the Valsalva maneuver has been demonstrated in healthy men with normal semen parameters with spermatic veins less than 3 mm in diameter.15 Meacham et al16 studied 34 asymptomatic young men with normal semen parameters and found varicoceles in 15% on physical examination, and flow reversal by color Doppler US in 35%. In that study a varicocele was defined by gray scale US as 2 or more veins with at least one vein having a diameter of 3 mm or greater. A diagnosis of varicocele was made by color Doppler US if retrograde flow was identified within the pampiniform plexus spontaneously and/or during the performance of a Valsalva maneuver. The findings indicate that the mere presence of venous reflux by color Doppler US is not necessarily clinically significant.

To date, no quantitative color Doppler US studies have been published that distinguish between significant and insignificant levels of flow reversal. Cornud et al in 1999 attempted to classify venous reflux with pulsed Doppler US according to its duration after the Valsalva maneuver.17 Three grades were described: grade 1 (brief) reflux lasted less than 1 second and was considered physiologic; grade 2 (intermediate) reflux lasted 1–2 seconds and decreased during the Valsalva maneuver, then disappeared prior to the end of the maneuver; and grade 3 (permanent) reflux lasted more than 2 seconds and demonstrated a plateau throughout the Valsalva maneuver. Grade 3 reflux was associated with a palpable varicocele in 60% of cases, whereas grade 1 and 2 reflux were never associated with a palpable varicocele. The authors also noted that only treatment of subclinical varicoceles with grade 3 reflux resulted in changes similar to those seen after repair of palpable varicoceles. A recent study by Cina et al highlights the current dilemmas in US diagnosis of varicocele.18 The authors performed scrotal color Doppler US studies in 145 healthy, asymptomatic subjects with normal clinical examinations and semen analyses. They noted that the upper limit of scrotal vein diameter (3.7-3.8 mm) exceeded the values commonly employed for a diagnosis of varicocele. In addition, 53% of the subjects had reflux in the scrotal veins when performing the Valsalva maneuver.

In summary, as recently noted by Lee et al, until the reliability and validity of our measurement tools is ensured and strict diagnostic criteria for the different imaging modalities are developed, it will not be possible to accurately identify those patients with varicocele who might benefit from surgical intervention.8

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Figure. Left varicocele. Transverse (a) and sagittal (b) sonograms depict a varicocele located medial and superior to the left testis. There is an increase in diameter of the dilated veins of the pampiniform plexus when a sagittal image obtained at rest (c) is compared to an image obtained while the patient performs the Valsalva maneuver (d). Color Doppler imaging with spectral analysis depicts flow reversal within the varicocele during the Valsalva maneuver (e).

References
The first step in the evaluation of an adolescent referred for evaluation with a diagnosis of a varicocele or, as sometimes occurs, a scrotal mass which is a varicocele is to obtain a good history and physical exam. The history should ascertain how the lesion was discovered, how long it has been present and whether or not there has been any significant associated pain (which is uncommon). Then there should be an objective measurement of bilateral testis sizes with an orchidometer and/or an ultrasound exam.

Prior to recommending surgical or radiological correction of a varicocele for an adolescent boy it is absolutely necessary to have an objective discussion with the family about the rationale for the intervention including a discussion of the incidence of varicocele, infertility in the general population and in men with a varicocele. The discussion should include a careful review of the logistics and potential complications of the procedure as well as the outcomes of the intervention such as catch up growth of the left testis. The results of varicocele repair in older infertile men should be addressed. Finally, the fact that there is no firm data available to determine which of the young men with a varicocele will eventually have a fertility problem must be emphasized. It should be explained to the patient and his family that a varicocele has no significant endocrine effect so that his potency, virilization and all the other components of normal male development and function will remain normal whether or not the varicocele is repaired.

Our indications for recommending intervention include a significant difference in testicular size (greater than 2ml) if the side with the varicocele is smaller, a definite history of pain with a warning that the pain may not be secondary to the varicocele, and, therefore, may not resolve after intervention, or in the case of a large varicocele a desire by the family after the above described discussion to proceed even in the absence of a size differential or pain. Glassberg presented data at the annual meeting of the American Urological Society in May 2008 which indicated that a significant minority of boys with a varicocele and a smaller left testis will experience catch up growth without correction of their varicocele. There is other published data that does not agree with this finding. In spite of Glassberg’s work we have not changed our indications for recommending correction of an adolescent varicocele. If there is a recurrent or persistent varicocele we suggested radiologic correction. This is an option only in settings where there is a skilled interventional radiologist available.

Prior to recommending surgical or radiological correction of a varicocele for an adolescent boy it is absolutely necessary to have an objective discussion with the family about the rationale for the intervention....

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Each surgical approach to the adolescent varicocele has advantages and disadvantages. The currently utilized techniques for varicocele repair include the standard inguinal approach, the microscopic subinguinal approach, laparoscopic techniques, and the Palomo procedure. Each technique will be described in the following paragraphs.

Inguinal Approach

The inguinal approach to varicocele correction was first described by Ivanissevich in 1960. This procedure is performed at the internal inguinal ring with the patient in reverse Trendelenburg. The internal spermatic veins are doubly ligated with 3-0 or 4-0 silk sutures. Inspection of the floor of the inguinal canal will reveal any aberrant external spermatic veins or venous collateral veins which are also ligated. Attempts are made to identify and spare the spermatic artery and lymphatic channels. Kass and Marcol used a modified Ivanissevich approach and reported a 16% recurrence rate even when optical magnification with 2.5 to 4.5 power loupes is typically used to enhance visualization.

Subinguinal Approach

Goldstein et al described a subinguinal approach to varicocele repair with the use of the intraoperative microscope providing 10 – 15X power. This technique provides better visualization of the spermatic cord and allows a very thorough dissection of the very small testicular vessels and lymphatics. The goal is to identify and ligate all spermatic veins while preserving the testicular artery and lymphatic structures. The procedure begins with the patient in the supine position. A 2.5 – 3 cm incision is made above the external inguinal ring and the spermatic cord is identified and mobilized. The testicle is delivered into the operative field to allow for identification of any gubernacular veins which are subsequently ligated. Once the testicle has been returned to the scrotum, the operating microscope is used to facilitate meticulous dissection of the entire spermatic cord. All venous branches are either clipped or doubly ligated with 4-0 silk and then divided. The vas deferens, spermatic artery and any lymphatics are identified and preserved. Any vasal veins that are >2.5 mm in diameter are also ligated. Schiff et al using this technique reported no varicocele recurrence and a 1% hydrocele rate with only 10.1 months of follow up. Yaman et al used a technique similar to Goldstein but did not deliver the testicle in order to look for veins in the gubernaculum. They described a 98% success rate with no secondary hydroceles in their study of 92 patients. The authors discussed that they did not have any difficulty identifying the testicular artery using the microscopic subinguinal approach in prepubertal children. One concern with the subinguinal microscopic approach is the rare but devastating potential for testicular atrophy. Chan et al reviewed 2102 adult men undergoing microscopic subinguinal varicocele repair and described a 1% risk of inadvertent ligation of the testicular artery. In patients with subinguinal artery ligation they found a 5% risk of testicular atrophy. This risk has the potential of being even greater for a less experienced microscopic surgeon operating on the very small testicular arteries in children and adolescents.

Laparoscopic Approach

In 1988, Sanchez de Badajoz et al performed the first laparoscopic varicocele ligation. The procedure is performed transperitoneally with the patient in the Trendelenburg position. After adequate pneumoperitoneum is established via a 5-mm laparoscopic port at the umbilicus, two 3-5mm working ports are placed in the right lower quadrant and the suprapubic area. The peritoneum overlying the external spermatic vessels is incised and the entire spermatic vessel chain is isolated and ligated with clips or staples.

Prato and Mackinlay reported their results of 41 patients who underwent laparoscopic Palomo varicocelectomy. They reported a 98% success rate with no secondary hydroceles in their study of 92 patients. The authors discussed that they did not have any difficulty identifying the testicular artery using the microscopic subinguinal approach in prepubertal children. One concern with the subinguinal microscopic approach is the rare but devastating potential for testicular atrophy. Chan et al reviewed 2102 adult men undergoing microscopic subinguinal varicocele repair and described a 1% risk of inadvertent ligation of the testicular artery. In patients with subinguinal artery ligation they found a 5% risk of testicular atrophy. This risk has the potential of being even greater for a less experienced microscopic surgeon operating on the very small testicular arteries in children and adolescents.

Surgery for the Adolescent Varicocele

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The retroperitoneal approach to varicocele repair was first described by Palomo in 1949. The surgical procedure is performed through a short transverse incision just medial to the anterior iliac crest. The peritoneum is mobilized medially and the spermatic cord is identified and confirmed with traction on the ipsilateral testicle. Once mobilized, the entire internal spermatic vessel chain is encircled with a vessel loop to insure that all of the structures of the internal spermatic cord are identified and included. A mass ligation of the entire internal spermatic cord is then performed with two 3-zero silk ligatures. The spermatic artery and veins are doubly ligated, and may or may not be divided. Optical magnification with 2.5 to 4.5 power loupes is typically used to enhance visualization.

In our long term experience with the Palomo technique, we retrospectively reviewed the charts of 312 adolescent boys who underwent surgical ligation of a varicocele using mass retroperitoneal ligation of the spermatic artery and vein from August 1986 and December 2006. Our overall success rate with the Palomo technique for varicocele repair is 96.1%. Varicocele persistence occurred in 3.9% of the patients. After an average follow up of 14 months, 29% of patients presented with a palpable hydrocele. The majority of hydroceles were small, asymptomatic and only discovered on postoperative physical examination. Only 5% of all the patients undergoing varicocelectomy ultimately required hydrocelectomy because of large or increasing size and discomfort. Misseri et al described similar success rates of 97% with the standard Palomo procedure having only discovered a 3% recurrence rate. They also described a 28% hydrocele rate after a mean follow up of 22.1 months. Ten percent of patients who underwent the varicocelectomy ended up with a secondary hydrocelectomy. To our knowledge, there has never been a report of testicular atrophy with the retroperitoneal approach.

Due to fear of testicular atrophy, a modification of the Palomo approach spares the testicular artery during high ligation. Kass and Marcol reported an 11% varicocele recurrence rate when sparing the testicular artery. Gorenstein et al performed spermatic artery sparing retroperitoneal varicocele ligation with recurrence rates of 12%. Kraeft et al described an even higher failure rate with 18/48 (37.5%) children having a persistent or recurrent varicocele after retroperitoneal ligation with internal spermatic artery preservation. We believe that the artery sparing technique has a higher failure rate than the standard Palomo because ligation of small venous collaterals that are intimately attached to the artery may be missed with artery sparing techniques.
success rate with 34% secondary hydrocele formation. Hassan et al performed laparoscopic retroperitoneal ligation of the spermatic vessels in 89 boys between January 2000 and December 2003. Only 1.3% of patients had a persistent varicocele with a mean of 20 months follow up. Post operative hydroceles developed in 22.8% of their cohort.

The limitation with both the standard and laparoscopic Palomo varicocele ligation has been the increased risk for hydrocele formation. The cause for the high postoperative hydrocele rate relates to ligating the lymphatic channels. Even though the majority of patients who developed secondary hydroceles remain asymptomatic and do not require surgical correction, some investigators have tried to utilize techniques to limit secondary hydroceles. Kocvara et al describe lymphatic preservation during a laparoscopic Palomo. They reported a hydrocele rate of 2.9% in 104 patients who had the lymphatics preserved compared to 17.9% in the conventional group. However, they reported a 6.7% varicocele persistence rate in the lymphatic sparing group and an 8.9% persistence rate in the standard laparoscopic Palomo procedure. Schwenter et al randomized 50 patients and injected isosulphan blue into the space between the dartos and the tunica vaginalis. They reported a 0% hydrocele rate and a 4% varicocele persistence which resolved spontaneously at 6 months. Eight percent of patients experienced a blue stained scrotum for up to 6 months following surgery. Glassberg et al retrospectively reviewed 174 patients who underwent a laparoscopic lymphatic sparing Palomo with 88 patients who underwent a non-lymphatic sparing procedure. He reported an incidence of secondary hydrocele formation of 3.4% in the lymphatic sparing group, compared to 11.4% in the standard laparoscopic group. Varicocele recurrence rates were not significantly different with 2.9% recurrence in the lymphatic sparing group and 4.5% recurrence in the non-lymphatic sparing group.

In summary, we think that the Palomo procedure and the subinguinal microscopic approach both are equally successful. However, we believe that the laparoscopic Palomo or the open Palomo technique should be preferred for varicocele ligation in adolescents. They both have high success rates, a low risk for complications and testicular atrophy if the artery is advertently ligated. The Ivanissevich is the least successful technique, and, in our opinion, should no longer be used for adolescent varicocele ligation.

References

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