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Influence of Epididymal Anomalies and Development of Processus Vaginalis on Testicular Migration And Cryptorchidism. A Narrative Review

Feridun C. Tanyel [1], Luciano A. Favorito [2]

[1] Turkish Academy of Sciences, Ankara, Türkiye; [2] Unidade de Pesquisa Urogenital - Universidade do Estado do Rio de Janeiro – UERJ, Rio de janeiro, RJ, Brasil

ABSTRACT

The objective of this narrative review is to show the most relevant aspects of the structure of epididymal anomalies (EAs) and processus vaginalis (PV) during testicular migration and in patients with undescended testis. Smooth and striated muscles develop around the processus vaginalis (PV) within the gubernaculum during fetal life. The testis migrates through the PV by the means of peristaltic activity generated by those muscles. After migration is completed, the smooth muscle undergoes programmed cell death, resulting in obliteration of PV and leaving only the dartos muscle. Initiation of programmed cell death requires a shift in autonomic balance characterized by a decrease in sympathetic tone and a concomitant increase in parasympathetic tone. The shift represents a critical step in the migration process. If the shift occurs prematurely, smooth muscle mass decreases before migration is completed, potentially preventing adequate propulsion of the testis and resulting in undescended testis. Premature shift is persistent. Persistence of this shift links the clinical features associated with failed migration. Conversely, if the shift fails to occur or is insufficient, smooth muscle persists and inhibits obliteration of the PV, leading to inguinal hernia or hydrocele depending on the remaining amount of smooth muscle. Epididymal disjunctions anomalies and epididymis atresia could be associated with infertility and are very usual in undescended testis. We can divided EAs in 3 groups: normal variant, disjunction anomalies and epididymis atresia. In the normal variant the epididymis is attached to the testis at the head and tail and totally attached to the testis. The transient presence of SM, its programmed elimination, and regulation by autonomic tone together offer a coherent model that integrates developmental, physiological, and clinical observations. EAs are frequently found in cryptorchidism. Knowledge of anomalies associated with cryptorchidism is relevant in clinical practice, both to prevent accidents during orchidopexy and to counsel and predict infertility in the future, such as in cases of epididymis atresia and total disjunction between the testis and the epididymis.

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Luciano A. Favorito
https://orcid.org/0000-0003-1562-6068

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INTRODUCTION

Undescended testis can be associated with several anatomical anomalies, but epididymal anomalies (EAs) and patency of the processus vaginalis (PV) are among the most frequent with a very variable incidence in the literature: from 36 to 79% (1-4). Knowledge of EA associated to cryptorchidism is relevant in clinical practice to prevent accidents during orchidopexy and to counsel and predict infertility in the future, such as in cases of epididymis atresia, disjunction of epididymal head and total disjunction between the testis and the epididymis (5, 6).

Some studies show an association of the patency of the PV with EAs and cryptorchidism, suggesting that there is a common stimulus, probably androgenic, participating in the epididymis development, testicular migration and closure of the PV (3, 7). The objective of this review is to show the most relevant aspects of the structure of EAs and PV during testicular migration and in patients with undescended testis.

PROCESSUS VAGINALIS AND TESTICULAR MIGRATION

Despite centuries of attempts to explain testicular migration, none has provided a fully satisfactory account. A paradigm shift offers a more coherent mechanistic explanation (8, 9). Migration of the testis can be conceptualized as work, defined as force multiplied by displacement. Hormones alone do not generate force; therefore, migration requires a structure capable of producing force under hormonal modulation. Failure of migration results in an undescended testis, and any explanation must integrate both the mechanism of descent and the reason for its inhibition.

The processus vaginalis (PV) serves as the pathway for testicular migration. Following descent, the PV should undergo obliteration; failure of this process leads to inguinal hernia or hydrocele. Undescended testis is associated with vaso-epididymal anomalies, disrupted mini-puberty, reduced fertility, and increased risk of malignancy. A testis that has initially descended may later retract or ascend (10). A comprehensive mechanism should therefore provide a unified explanation for these associated observations.

Patent processus vaginalis and smooth muscle persistence

Sacs obtained from boys with inguinal hernia or hydrocele consistently present smooth muscle (SM) and myofibroblasts beneath the mesothelial layer, whereas obliterated PV lacks these structures (11-13). Multiple independent studies have confirmed that persistence of SM inhibits obliteration of PV (14-23).

Residual SM not only prevents obliteration but may also contribute to clinical complications. For example, persistence of SM may be associated with an increased risk of incarceration in preterm infants and elevated hydrocele pressures (24-26).

Muscular development in the gubernaculum

The PV develops into the gubernaculum. Youssef and Raslan have reported the development of SM in gubernaculum, which disappears after descent (27).

Our studies have demonstrated that striated muscle fibers and myofibroblasts appear within the gubernaculum at 22 weeks of gestation, whereas SM develops around the PV at approximately the 27th week. Cremaster muscle may transdifferentiate from the vascular SM. Myofibroblasts appear to represent an intermediate stage within the differentiation- dedifferentiation continuum of SM (28).

The gubernaculum that is described as primitive mesenchymal tissue ceases to exist after development of muscles. We have speculated that INSL3 takes part in myogenesis (8). Yuan et al. subsequently provided support for this proposal (29).

The physiological role of smooth muscle

The transient presence of SM around the PV serves a specific physiological role: propulsion of the testis during migration. This propulsion explains both successful descent and the risk of intrauterine torsion (8-10). Once migration is completed, the SM

undergoes programmed cell death, leaving only the dartos muscle. This interpretation provides a framework that integrates the mechanical, developmental, and pathological aspects of testicular descent.

Programmed cell death of smooth muscle depends on shift in autonomic tonus

After a structure completes its function, it disappears through programmed cell death (PCD). PCD involves both intrinsic and extrinsic pathways. The intrinsic pathway involves calcium signaling that increases cytosolic calcium and promotes mitochondrial calcium loading, leading to opening of permeability transition pores (9).

Maintenance of SM appears to depend on sympathetic tone, whereas activation of the inositol 1,4,5-trisphosphate pathway releases calcium from the intracellular stores and initiate PCD.

These observations suggest that a transient decrease in sympathetic tone combined with an increase in parasympathetic tone may represent the physiological mechanism responsible for PV obliteration.

Silencing of the central catecholaminergic activity may contribute to this shift (9). The key to inguinal hernia, hydrocele and undescended testis lie in this shift of autonomic tone. Deviations in the timing, intensity, or duration of this may lead to pathological outcomes. Because sympathetic tone is sexually dimorphic, this shift may occur more readily in females.

Early shift inhibits migration whereas ineffective shift results in inguinal hernia or hydrocele

If the autonomic shift occurs before testicular migration is completed, premature PCD may reduce the amount of SM surrounding the PV. Remaining SM may not suffice to propel the testis. Minimal SM content results in an undescended testis located at a higher position (8).

If the intensity or duration of shift in autonomic tone is not as effective as required, SM does not undergo PCD and persists (30-32). Persistence of SM inhibits the obliteration of PV, resulting in inguinal hernia or hydrocele depending on the remaining amount of SM (Figure-1) (9,10).

Persistence of autonomic imbalance forms basis for the associated features encountered in undescended testis

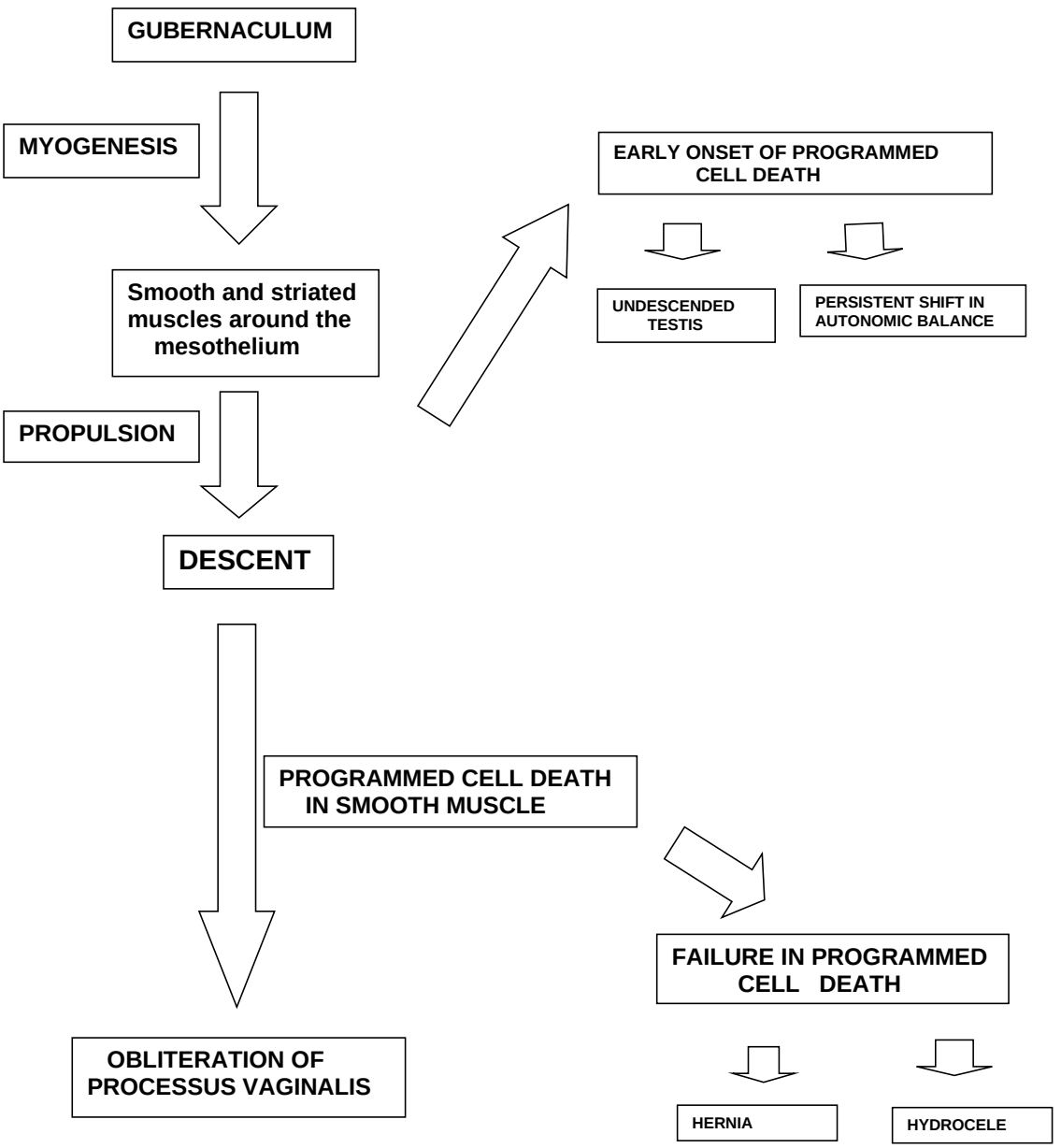
Sacs, cremaster muscles and heart rate variability analysis from boys with undescended testis reveal evidence of defective autonomic innervation via increase in parasympathetic but decrease in sympathetic tones (33-40). Persistence of shift in autonomic tone provides basis for the associated occurrences such as decrease in fertility, increase in risk of malignancy, vaso-epididymal anomalies, disrupted mini-puberty and retractility and ascent.

While parasympathetic tone acts through generating phospholipase C, sympathetic tone acts by generating cyclic adenosine monophosphate (cAMP). Continuous stimulation of phospholipase C pathway by phorbol esters results in the development of tumors. On the other hand, the cAMP pathway is important for spermatogenesis. Differences in signaling pathways with dominance of phospholipase C and decrease in cAMP pathways explain the increase in the risk of malignancy and decrease in fertility (Figure-2) (8, 9). Persistence of signaling towards inducing PCD may also play a role in the reduction of number of germ cells associated with undescended testes.

The gubernaculum also supports the muscular layer of vaso- epididymal structures. Signals towards inducing programmed cell death in SM explains both the reason of vaso-epididymal anomalies and their association with undescended testis (9).

Central catecholaminergic activity also takes place in the regulation of hypothalamic- pituitary-gonadal axis. Since catecholamines take place in the control of pulsatile release of gonadotropin releasing hormone, silencing of central catecholaminergic activity may also affect the hypothalamic- pituitary- gonadal axis (9, 41). On the other hand, testosterone release is also regulated by an efferent neural pathway from the hypothalamus to the testes under the influence of catecholamines (42). Chemical sympathectomy reduces the concentration of testosterone (43). The silencing of central catecholaminergic activity satisfactorily explains the decrease in release of testosterone from the Leydig cells in response to hCG.

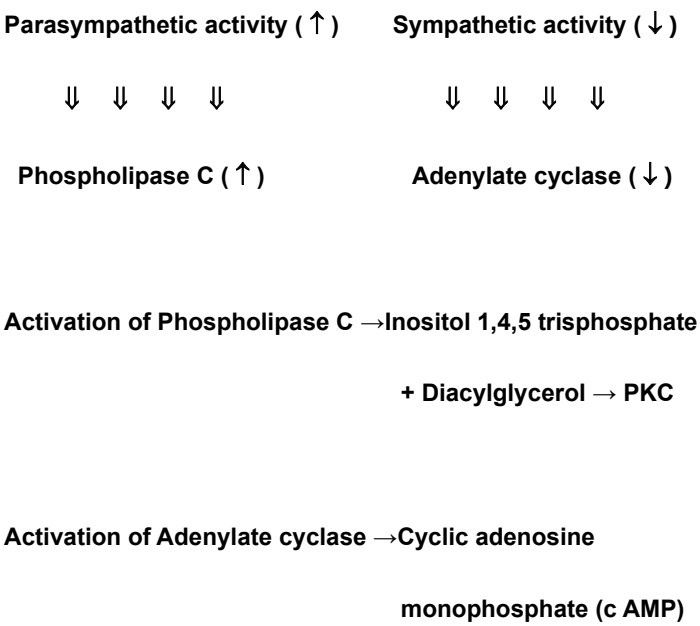
Figure 1 - Schematic summary of Testicular Migration.



Hypogonadism may result from central catecholaminergic changes directed to shift the autonomic tone. Disrupted mini puberty does not provide basis to inhibit descent but shares the persistent shift in autonomic tone (44).

A migrated testis may subsequently retract or ascend. Retractile testis has traditionally been explained by a hyperactive cremaster reflex. However, the cremaster reflex cannot be repeatedly induced without a refractory period due to desensitization. Induction of an already induced superficial reflex is not possible until reflex arc is completed. If the retraction results from a reflex arc, the testis should move up and down and stay in the scrotum after de-

Figure 2 - Signaling during persistent shift in autonomic tonus explains the increase in risk of malignancy and decrease in fertility.



sensitization. However, there are no such testes (45-47). The increase in parasympathetic but decrease in sympathetic tones increases cytosolic calcium. Increase in cytosolic calcium contracts a striated muscle. Increased contractility and contracture define the place in the spectrum as retraction or ascent. Cremaster muscles associated with retractile testes share similar alterations as in patients with undescended testis (48, 49). Retraction or ascent does not reflect a hyperactive reflex but reflects the increase of cytosolic calcium in cremaster muscles due to shift in autonomic tones.

EPIDIDYMIS AND TESTICULAR MIGRATION

Testicular migration is a complex process mediated by endocrine and mechanical factors. The integrity of the axis between the testis, hypothalamus and pituitary, which regulates testosterone production, is important for this process.

Cryptorchidism is a common event in pathologies on this axis, such as hypogonadotropic hypogonadism and 5-alpha reductase deficiency (50). Testosterone appears to play an active role in testicular migration, inducing the development of important structures for testicular migration such as the vaginal process, the vas deferens, the epididymis, the inguinal canal and the scrotum. Another mechanism of action of testosterone would be through stimulation of the genitofemoral nerve, which would induce the production of calcitonin gene-related peptide (CGRP) that acts by stimulating the development of the testicular gubernaculum (51).

Fetal and placental gonadotropins are also implicated in the process of testicular migration. These substances act by stimulating the production of testicular androgens, which induce the growth and development of the vas deferens, the epididymis, the vaginal process and the gubernaculum, itself (52). It is well known that the treatment of cryptorchidism with gonadotropins induces testicular migration at levels ranging from 25 to 55% of cases (53).

Another endocrine substance involved in testicular migration would be descendin. This an-

drogen-independent secreted substance in the testis would play an important role in the growth of the gubernaculum mesenchymal cells. The gubernaculum would therefore be one of the fetal structures implicated in testicular migration, most modified by hormonal action.

Problems with the hypothalamus-pituitary axis are associated with cryptorchidism, which explains the high incidence of this anomaly in patients with Prader-Willi syndrome, Kallman syndrome, pituitary hypoplasia and anencephaly.

Analysis of the nerves of the cremaster muscle of patients with retractile testis also revealed significant differences. These patients had a lower concentration of nerve fibers compared to those with inguinal hernia, but no differences in relation to the group with cryptorchidism. The innervation of the cremaster muscle and the gubernaculum testis originates in the genitofemoral nerve (53). This nerve, through androgenic regulation and the influence of the calcitonin gene-related peptide (CGRP), plays an important role during the testicular migration process, by neuromuscular junction stimulation of the rhythmic contractions of the cremaster muscle during the fetal period (52).

The epididymis is anatomically connected to the gubernaculum, which in turn is attached to the testicle and scrotum (Figure-3). The gubernaculum seems to be the most important anatomical structure in the testicular migration process, by means of contraction and shortening, thus imposing traction strength on the testis and facilitates the transition of the testis by through the inguinal canal (53, 54) (Figure-3).

The growth of the vaginal process divides the gubernaculum into three parts: (a) the main gubernaculum, which corresponds to the portion covered by the visceral layer of the peritoneum of the vaginal process; (b) the vaginal gubernaculum, which corresponds to the portion that externally surrounds the parietal portion of the vaginal process, and (c) the infra-vaginal gubernaculum, corresponding to the caudal region of the gubernaculum, which has not been invaded by the vaginal process (55).

Both the gubernaculum and vaginal process change in harmony during testicular migration. The maintenance of this undifferentiated mesenchyme along the inguinal canal and scrotum is essential for the downward extension of the vaginal process to occur, during which it follows the pathway created by dilation of the gubernaculum, forming the canal through which the testis will reach the scrotum (53, 55).

An interesting and controversial theory suggests that the epididymis would be one of the responsible for testicular migration, through its peristaltic and secretory activity in the second gestational trimester (56). This theory would explain some cases of cryptorchidism, where the epididymis is separated and located more inferiorly than the testis (57). Several observations support the theory described above. The epididymis precedes the testicle in the scrotum; the epididymis is in a privileged position to influence testicular migration, as it is anatomically connected to the gubernaculum, which in turn is attached to the testicle and scrotum. Morphological and functional changes occur in epididymis at the time of migration in certain animal species (58, 59).

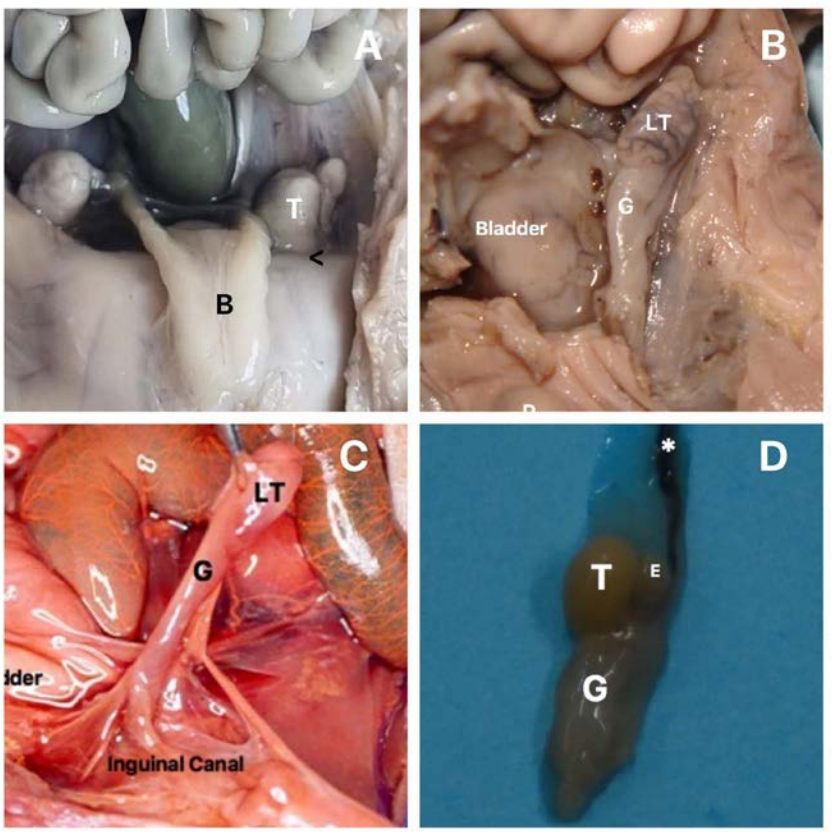
EPIDIDYMAL ANOMALIES AND UNDESCENDED TESTIS

There are several classifications to analyze the relations between the testis and epididymis (60, 61). We can divide this relationship in 3 groups: normal variant, disjunction anomalies and epididymis atresia. In the normal variant the epididymis is attached to the testis at the head and tail (Figure-4) and totally attached to the testis (Figure-5).

We can observe three types of epididymal disjunction anomalies: Epididymis attached to the testis only at the head (Figure-5); epididymis attached to the testis only at the tail (Figure-6) and no visible connection between the testis and epididymis (Figure-6). The EAs with head disjunction and total disjunction are associated to infertility (62). We can observe example of epididymal atresia in Figure-7.

Mollaeian (1) studied 652 undescended testes and observed epididymal disjunction in 16.87% of the cases. Caterino (63) in a study with 895 patients and Kim (2) in a study with 110 patients reports EAs,

Figure 3 - Testicular migration and gubernaculum.



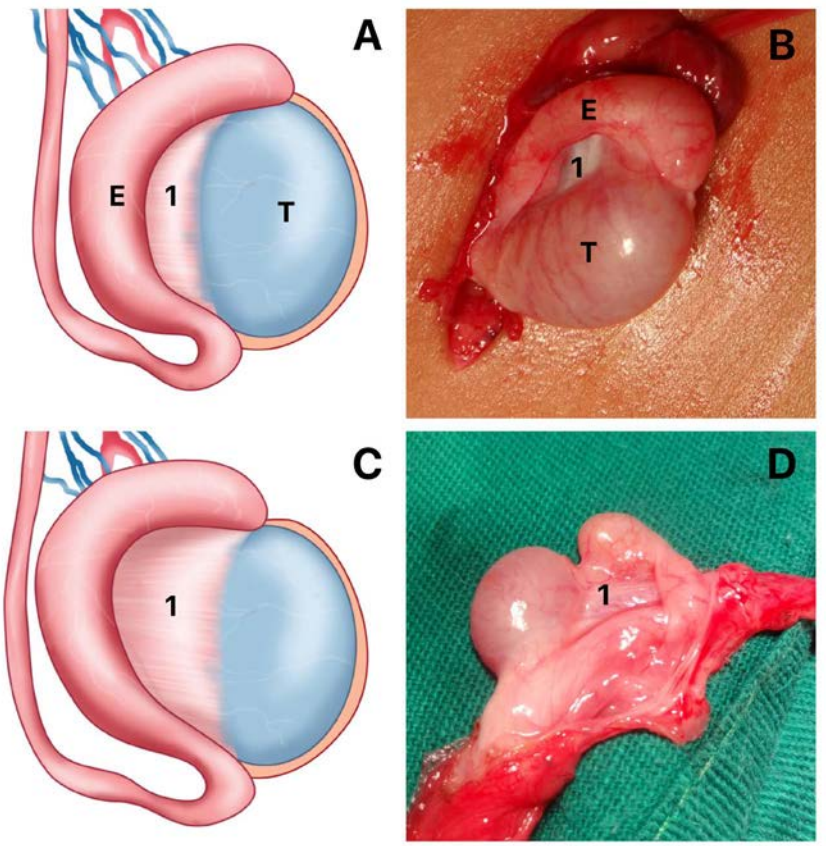
A) The figure shows a fetus with 18 weeks post-conception (WPC) showing the testis (T) beginning its transition in internal inguinal ring (<), B- bladder; B) The figure shows the dissection of the inguinal canal in a fetus with 17WPC. We can observe the gubernaculum (G) attachments with the left testis (LT) and epididymis proximally and with the inguinal region distally; C) In this figure we can observe the dissection of the gubernaculum (G) in a fresh human fetus with 18 WPC, LT – left testis, C- colon and D) The testis (T), epididymis (E) and gubernaculum (G) were removed from a fetus with 20WPC, observe the testicular vessels (*) in proximal position of the testis

respectively, in 96% and 65% - a very high index and probably this high number is associated to a classification that considers normal relationships between testis and epididymis as an anomaly. In a previous study of our group, we observed EAs in 43.7% of the cases; the disjunction of epididymal tail was observed in 25,3%, and disjunctions of epididymal head and total disjunction of epididymis were observed in 18.4% of the cases (62).

The discordant number of EAs associated to undescended testis in several studies can be justified by the lack of definition of the normal pattern of the anatomy of the epididymis in several studies (60, 61). The major risk of infertility in patients with undescended testis may be result from obstruction associated to epididymal disjunctions anomalies and epididymis atresia (64, 65). The disjunction of epididymal head and total disjunction of epididymis are usual in undescended testes and more frequent in testes situated in high position and with patency of the PV (64, 65). The PV is usually obliterated after the end of the testicular migration (66-68). The patency of the PV in patients with cryptorchidism ranges from 21.3 to 81.3% (2, 66).

The epididymal anomalies of disjunction can be explained by the difference in the embryological origin of the epididymis from the mesonephric duct or by changes in the vascularization during the development of the mesonephric tubules and their fusion with the testicular tubules (69). So we suggest

Figure 4 - Relationship between testis and epididymis - Normal variant.



A) Schematic drawing showing a normal variant of epidydimal anatomy - the epididymis (E) is attached to the testis (T) at the head and tail, and we can observe the mesorchium (1); B) We can observe in a undescended testis patient with 3 years old a normal variant of the epididymis (E) – Epididymis is attached to the testis (T) at the head and tail, 1- mesorchium; C) Schematic drawing showing an elongated epididymis with a long mesorchium (1) and D) The same situation in a undescended testis patient with 4 years-old, an elongated epididymis with long mesorchium.

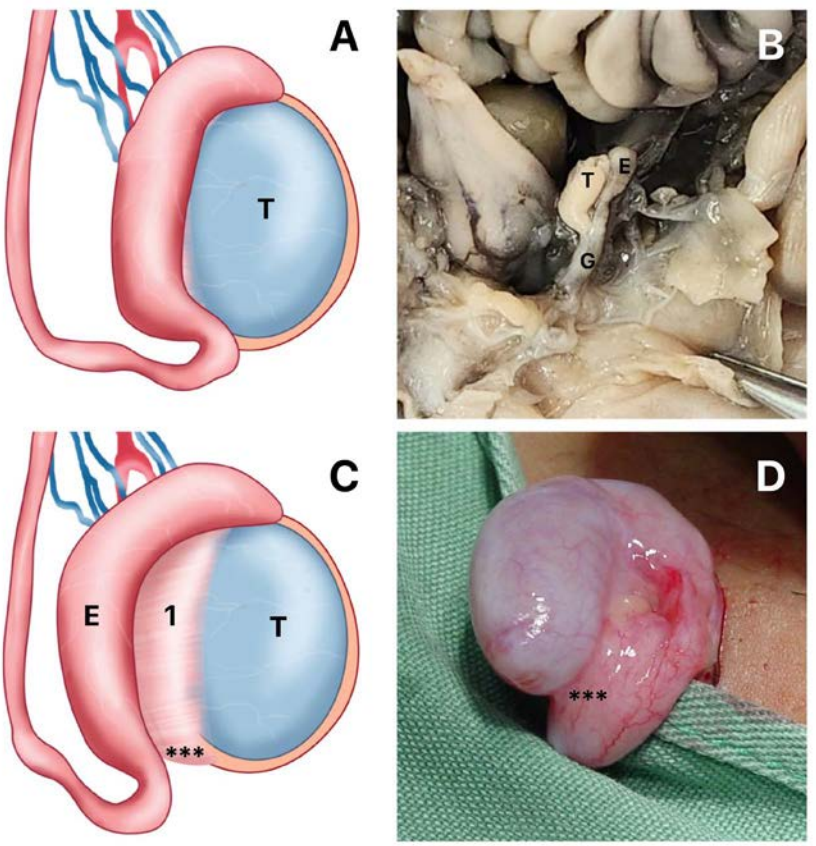
the classification of the epididymal anomalies in disjunctions anomalies, epididymal atresia and elongated epididymis.

EPIDIDYMAL ANOMALIES AND TESTICULAR TORSION

The testicular torsion is an anomaly resulting from changes in the implantation of the tunica vaginalis or epididymal disjunction (70). Normally, the testis is united to the tunica vaginalis, and if the tunica is implanted too high, the testis can present excessive mobility (bell clapper testis) (70). Mesorchium is the ligament that unites the testis to the epididymis (58). In cases of epididymal disjunction or elongated epididymis, conditions that are highly frequent in cryptorchism (69, 71, 72) the mesorchium is long and can contribute to the testicular torsion (58) (Figures 5 and 6).

The type of testicular torsion was classified in three groups according to the anatomy of the tunica vaginalis and the relationship between testis and epididymis (73) (Figure-8): Group A – bell clapper testicular deformity (leading to intravaginal torsion); Group B – torsion of spermatic cord (leading to extravaginal torsion) and Group C – torsion due to long mesorchium (Figure-8). Bell clapper deformity (intravaginal torsion) is the most commonly found type of testicular torsion (80%).

Figure 5 - Testis attached to epidydimis and Epididymal tail disjunction.



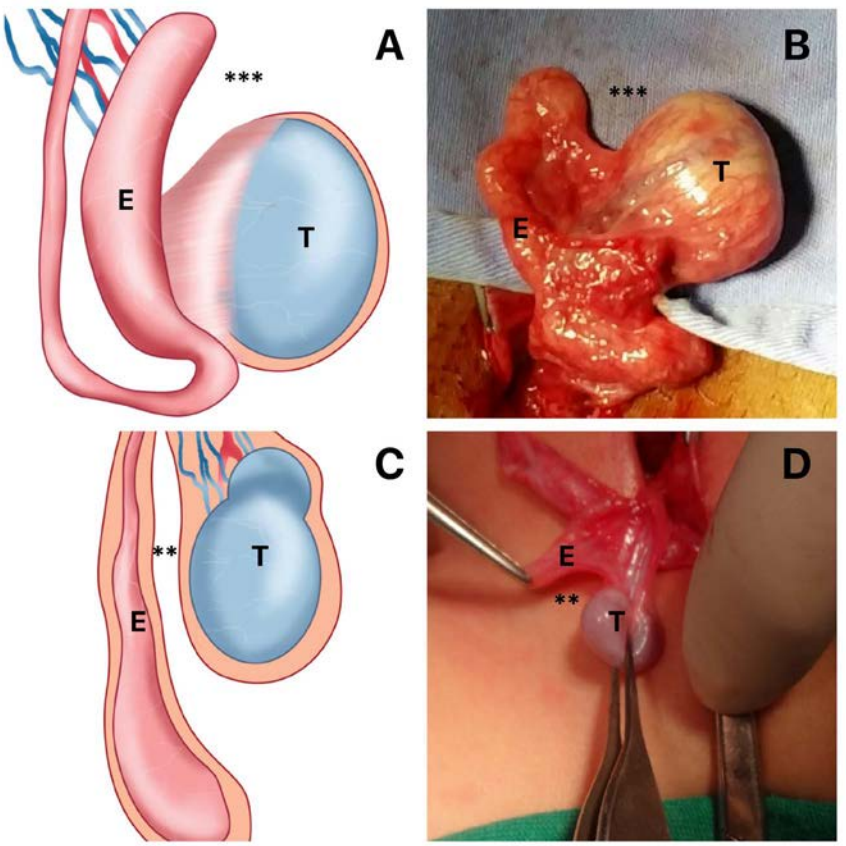
A) Schematic drawing showing a normal variant of the epididymis anatomy - the epididymis is attached to the testis (T) at the head and tail; B) We can observe in a fetus with 16 weeks post-conception a normal variant of the epididymis anatomy - the epididymis (E) is attached to the testis (T), G- gubernaculum; C) Schematic drawing of a epididymal anomaly – disjunction of the epididymal tail (***), T- testis, E- epididymis and 1 – mesorchium and D) We can observe a disjunction anomaly of the epididymal tail (***) in a undescended testis patient with 1 year-old.

Testes present a normal layering of tunica vaginalis. They are involved by this structure on both sides and on their upper portion. The posterior region of testis is not covered by tunica vaginalis. United to the lower pole region of the testis and the epididymal tail, there is the testicular gubernaculum or its remnant, the testicular ligament, which is covered by tunica vaginalis only in its anterior and lateral portions (58). Intravaginal testicular torsion does not occur in testes presenting normal anatomy as described above, because the posterior testicular segment is firmly united to the scrotum, preventing the organ from moving (58, 70).

Normal anatomy of tunica vaginalis or epididymis at the side contralateral to the torsion is very rare and anatomic anomalies occur bilaterally in the vast majority of cases. These findings stress the need for bilateral orchiopexy in cases of testicular torsion (73). Cases of torsion due to long mesorchium most often occur as a consequence of anomalies of epididymal disjunction or elongated epididymis, conditions that are highly frequent in cryptorchism (58). Of the 8 cases with long mesorchium, 3 (37.5%) had cryptorchid testes.

Approximately 20% of cases of testicular torsion occur in patients with cryptorchism (58). Epididymal anomalies associated with long mesorchium are frequent in patients with cryptorchism, with an incidence ranging from 36 to 72% (71) and rare in in-

Figure 6 - Epididymal anomalies.



A) Schematic drawing showing the epididymal anomalies of head disjunction (***), T- testis, E- epididymis; B) We can observe in a undescended testis patient with 4 years-old the epididymal anomalies of head disjunction (***),T- testis, E- epididymis; C) Schematic drawing showing the total disjunction (***) between the testis (T) and epididymis (E) and D) We can observe in a undescended testis in a patient with 2 years-old the total disjunction (***) between the testis (T) and epididymis (E

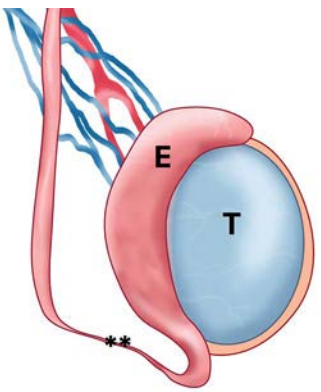
dividuals with topic testes (less than 4%) (60). Due to these changes in mesorchial region, the possibility of testicular torsion must be considered in cryptorchid patients presenting acute scrotal or inguinal pain.

The intravaginal torsion (bell clapper tunica vaginalis) is the most frequent type of torsion, and torsion due to long mesorchium is associated with cryptorchism. The most frequently found anatomical relation between testis and epididymis in the study group was type I (epididymis united to the testis by its head and tail). Historically the bell-clapper deformity is the only anatomical risk factor for the intravaginal torsion but the epididymal dysjunction, despite rare, also play a hole in the pathology. In approximate 90% of the time, a vestigial of Müllerian duct keeps present attached to the upper pole of the testis and is called appendix testis or hydatid of Morgani. This appendix can also twist during lifetime, mimics the testicular torsion clinical presentation and sometimes also requires surgical treatment (74, 75).

CONCLUSIONS

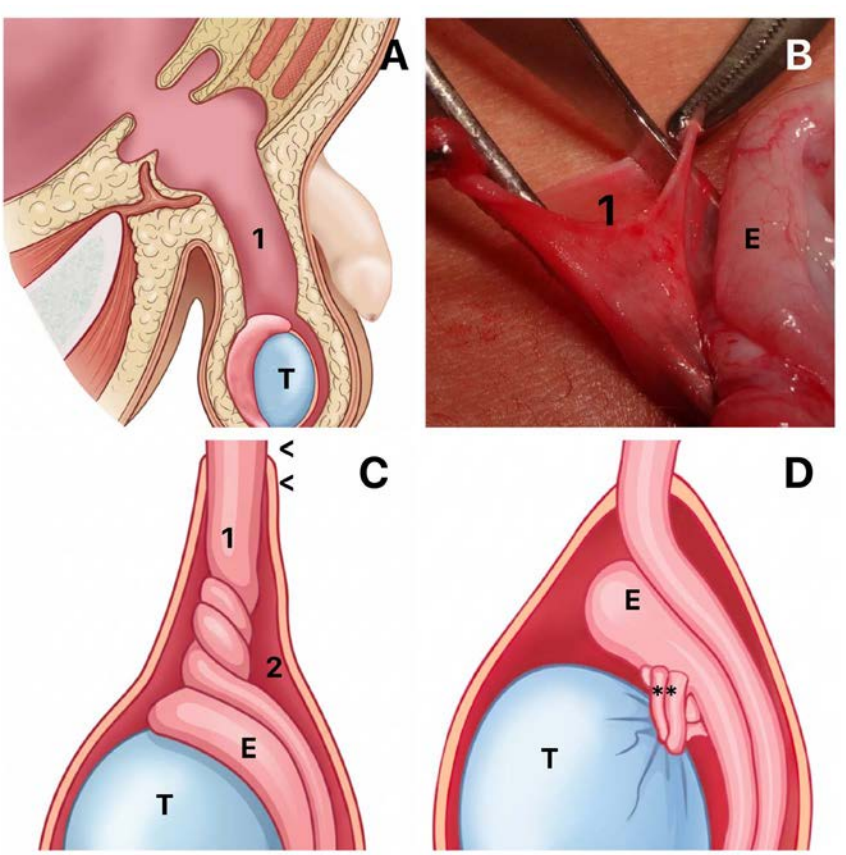
Evaluating testicular migration within this mechanistic framework provides a unified explanation for both successful descent and its associated disorders. The transient presence of SM, its programmed elimination, and regulation by auto-

Figure 7 - Schematic drawing showing the epididymal atresia.



T- testis, E-epididymis and ** - atresia of the epididymal tail.

Figure 8 -Patency of Processus vaginalis.



A) Schematic drawing showing the patency of Processus vaginalis (1), T- testis; B) We can observe in a undescended testis patient with 6 years-old the patency of processus vaginalis (1), E- Epididymis; C) schematic drawing showing the high implantation (<<) of the tunica vaginalis (2) in spermatic vessels (1): the bell clapper testicular deformity (leading to intravaginal torsion), T- testis, E- epididymis and D) Schematic drawing showing the testicular torsion due to long mesorchium (**), T- testis, E- epididymis.

nomic tone together offer a coherent model that integrates developmental, physiological, and clinical observations.

EAs are frequently found in cryptorchidism. Knowledge of anomalies associated with cryptorchidism is relevant in clinical practice, both to prevent accidents during orchidopexy and to counsel and predict infertility in the future, such as in cases of epididymis atresia and total disjunction between the testis and the epididymis.

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CONFLICT OF INTEREST

None declared.

REFERENCES

1. Mollaeian M, Mehrabi V, Elahi B. Significance of epididymal and ductal anomalies associated with undescended testis: study in 652 cases. Urology. 1994;43(6):857-60. doi: 10.1016/0090-4295(94)90152-x
2. Kim SO, Na SW, Yu HS, Kwon D. Epididymal anomalies in boys with undescended testis or hydrocele: significance of testicular location. BMC Urol. 2015;15:108. doi: 10.1186/s12894-015-0099-1
3. Barthold JS, Redman JF. Association of epididymal anomalies with patent processus vaginalis in hernia, hydrocele and cryptorchidism. J Urol. 1996;156(6):2054-6.
4. Lee T, Han SW, Lee MJ, Kim JH, Choi SK, Cho NH, et al. Pathological characteristics in retractile testis comparing cryptorchid testis. Korean J Urol. 1999;40:617-22.
5. Rachmani E, Zachariou Z, Snyder H, Hadziselimovic F. Complete testis-epididymis nonfusion anomaly: a typical association with cryptorchid testis. Urol Int. 2012;89(3):355-7. doi: 10.1159/000342665
6. Sharma S, Sen A. Complete testicular epididymal dissociation in the abdominal cryptorchid testis. J Pediatr Urol. 2013;9(6 Pt B):1023-7. doi: 10.1016/j.jpurol.2013.02.017
7. Aggarwal H, Kogan BA, Feustel PJ. One third of patients with a unilateral palpable undescended testis have a contralateral patent processus. J Pediatr Surg. 2012;47(9):1711-5. doi: 10.1016/j.jpedsurg.2012.01.003
8. Tanyel FC. The descent of testis and reason for failed descent. Turk J Pediatr. 2004;46 Suppl:7-17.
9. Tanyel FC. Obliteration of processus vaginalis: aberrations in the regulatory mechanism result in an inguinal hernia, hydrocele or undescended testis. Turk J Pediatr. 2004;46 Suppl:18-27.
10. Tanyel FC. Obliteration of the processus vaginalis after testicular descent. Balkan Med J. 2024;41(2):89-96. doi: 10.4274/balkanmedj.galenos.2024.2023-12-111
11. Tanyel FC, Talim B, Kale G, Büyükpamukçu N. Differences in the morphology of the processus vaginalis with sex and underlying disease condition. Pathol Res Pract. 2000;196(11):767-70. doi: 10.1016/s0344-0338(00)80109-0
12. Tanyel FC, Müftüoğlu S, Dağdeviren A, Kaymaz FF, Büyükpamukçu N. Myofibroblasts defined by electron microscopy suggest the dedifferentiation of smooth muscle within the sac walls associated with congenital inguinal hernia. BJU Int. 2001;87(3):251-5. doi: 10.1046/j.1464-410x.2001.02028.x
13. Tanyel FC, Dağdeviren A, Müftüoğlu S, Gürsoy MH, Yörüker S, Büyükpamukçu N. Inguinal hernia revisited through comparative evaluation of peritoneum, processus vaginalis, and sacs obtained from children with hernia, hydrocele and undescended testis. J Pediatr Surg. 1999;34(4):552-5. doi: 10.1016/s0022-3468(99)90071-4
14. da Silva AL, Brasileiro FG, Ferreira AP. Morphological study of the inguinal hernial sac. Rev Hosp Clin Fac Med Sao Paulo. 1992;47(2):65-8.
15. Jiang ZP, Yang B, Wen LQ, Zhang YC, Lai DM, Li YR, et al. The etiology of indirect inguinal hernia in adults: congenital or acquired? Hernia. 2015;19(5):697-701. doi: 10.1007/s10029-014-1326-5

16. Mouravas VK, Koletsa T, Sfougaris DK, Philippopoulos A, Petropoulos AS, Zavitsanakis A, et al. Smooth muscle cell differentiation in the processus vaginalis of children with hernia or hydrocele. Hernia. 2010;14(2):187-91. doi: 10.1007/s10029-009-0588-9
17. Krishnan N, Kakkar A, Nag TC, Agarwala S, Goel P, Dhua AK. A comparative study of smooth muscle cell characteristics and myofibroblasts in processus vaginalis of pediatric inguinal hernia, hydrocele and undescended testis. BMC Urol. 2024;24:115. doi: 10.1186/s12894-024-01449-0
18. Piçarro C, Tatsuo ES, Amaral VF, Gomez RS, Cruzeiro PC, Lanna JC. Morphological comparison of processus vaginalis from boys with undescended testis and hernia sacs from boys with inguinal hernia. Eur J Pediatr Surg. 2009;19(3):145-7. doi: 10.1055/s-0029-1202258
19. Barbosa CA, Amaral VF, Silva AL. Histology of the hernial sacs of direct, indirect, relapsed and incarcerated inguinal hernias in adults and children: identification of smooth muscle fibers. Rev Col Bras Cir. 2002;29(1):1-6. doi: 10.1590/S0100-69912002000100002
20. Özdamar MY. Histopathological fate in the inguinal hernia sac in the children. Int J Pediatr Res. 2018;4(1):040. doi: 10.23937/2469-5769/1510040
21. Sfoungaris D, Mouravas V, Koletsa T, Lambropoulos V, Kostopoulos I, Spyridakis I. Differences in the development of the processus vaginalis between children with undescended testis and inguinal hernia. J Clin Neonatol. 2016;5(1):31-4. doi: 10.4103/2249-4847.173270
22. Lazio A, Vasconcelos RS, Gollner AM, Silva AL. Hernial sac histology of the inguinal hernias: identification of smooth muscle fibers and their relation with the blood vessel. Rev Col Bras Cir. 2009;36(4):323-6. doi: 10.1590/s0100-69912009000400009
23. Hosgor M, Karaca I, Ozer E, et al. The role of smooth muscle cell differentiation in the mechanism of obliteration of processus vaginalis. J Pediatr Surg. 2004;39(7):1018-23. doi: 10.1016/j.jpedsurg.2004.03.056
24. Olesen CS, Mortensen LQ, Öberg S, Rosenberg J. Risk of incarceration in children with inguinal hernia: a systematic review. Hernia. 2019;23(2):245-54. doi: 10.1007/s10029-019-01877-0
25. Tanyel FC, Öcal T, Karaağaoğlu E, Büyükpamukçu N. Individual and associated effects of length of inguinal canal and caliber of the sac on clinical outcome in children. J Pediatr Surg. 2000;35(8):1165-9. doi: 10.1053/jpsu.2000.8719
26. Tanyel FC, Öcal T, Büyükpamukçu N. Excessive sac pressures: the pathogenesis and innocence of hydroceles in children. BJU Int. 2001;87(4):372-5. doi: 10.1046/j.1464-410x.2001.00076.x
27. Youssef EH, Raslan NA. Study of factors which affect the descent of the testicles in man. Acta Anat (Basel). 1971;79(3):422-44. doi: 10.1159/000143674
28. Tanyel FC, Talim B, Atilla P, Müftüoğlu S, Kale G. Myogenesis within the human gubernaculum: histological and immunohistological evaluation. Eur J Pediatr Surg. 2005;15(3):175-9. doi: 10.1055/s-2005-837604
29. Yuan FP, Li X, Lin J, Schwabe C, Büllesbach EE, Rao CV, et al. The role of RXFP2 in mediating androgen-induced inguinoscrotal testis descent in LH receptor knockout mice. Reproduction. 2010;139(4):759-69. doi: 10.1530/REP-09-0518
30. Tanyel FC, Erdem S, Büyükpamukçu N, Tan E. Smooth muscle in incomplete obliterations of processus vaginalis lacks apoptotic nuclei. Urol Int. 2002;69(1):42-5. doi: 10.1159/000064359
31. Tanyel FC, Ulusu N, Tezcan EF, Büyükpamukçu N. Total calcium content of sacs associated with inguinal hernia, hydrocele or undescended testis reflects differences dictated by programmed cell death. Urol Int. 2003;70(3):211-5. doi: 10.1159/000068765
32. Tanyel FC, Okur HD. Autonomic nervous system appears to play a role in obliteration of processus vaginalis. Hernia. 2004;8(2):149-54. doi: 10.1007/s10029-003-0196-z
33. Tanyel FC, Sara Y, Ertunç M, Onur R, Büyükpamukçu N. Lack of carbachol response indicates the absence of cholinergic receptors in sacs associated with undescended testis. J Pediatr Surg. 1999;34(9):1339-44. doi: 10.1016/s0022-3468(99)90007-6
34. Tanyel FC, Erdem S, Büyükpamukçu N, Tan E. Cremaster muscles obtained from boys with an undescended testis show significant neurologic changes. BJU Int. 2000;85(1):116-9. doi: 10.1046/j.1464-410x.2000.00362.x

35. Tanyel FC, Erdem S, Altunay H, Ergün L, Özcan Z, Alabay B, et al. Distribution and morphometry of fiber types in cremaster muscles of boys with inguinal hernia or undescended testis. Pathol Res Pract. 2000;196(9):613-7. doi: 10.1016/s0344-0338(00)80003-5
36. Tanyel FC, Müftüoğlu S, Dağdeviren A, Karakoç L, Büyükpamukçu N. Ultrastructural deficiency in autonomic innervation in cremasteric muscle of boys with undescended testis. J Pediatr Surg. 2001;36(4):573-8. doi: 10.1053/jpsu.2001.22285
37. Tanyel FC, Erdem S, Büyükpamukçu N, Tan E. Cremaster muscle is not sexually dimorphic but that from boys with undescended testis reflects alterations related to autonomic innervation. J Pediatr Surg. 2001;36(6):877-80. doi: 10.1053/jpsu.2001.23959
38. Tanyel FC, Ertunç M, Büyükpamukçu N, Onur R. Mechanisms involved in contractile differences among cremaster muscles according to localization of testis. J Pediatr Surg. 2001;36(10):1551-60. doi: 10.1053/jpsu.2001.27042
39. Tanyel FC, Ulusu N, Tezcan EF, Büyükpamukçu N. Less calcium in cremaster muscles of boys with undescended testis supports a deficiency in sympathetic innervation. Urol Int. 2002;69(2):111-5. doi: 10.1159/000065558
40. Duman L, Demirci M, Tanyel FC. Heart rate variability analysis reveals a shift in autonomic balance towards an increase in parasympathetic tonus in boys with undescended testis. Eur J Pediatr Surg. 2010;20(3):150-2. doi: 10.1055/s-0029-1243627
41. Spergel DJ. Modulation of gonadotropin-releasing hormone neuron activity and secretion in mice by non-peptide neurotransmitters, gasotransmitters, gliotransmitters. Front Endocrinol. 2019;10:329. doi: 10.3389/fendo.2019.00329
42. Selvage DJ, Lee SY, Parsons LH, Seo DO, Rivier CL. Hypothalamic-testicular neural pathway is influenced by brain catecholamines, but not testicular blood flow. Endocrinology. 2004;145(4):1750-9. doi: 10.1210/en.2003-0925
43. Rosa e Silva AA, Guimarães MA, Lamano-Carvalho TL, Kempinas WG. Chemical sympathectomy blocks androgen biosynthesis during prepuberty. Braz J Med Biol Res. 1995;28(10):1109-12.
44. Tanyel FC. Commentary: The fate of germ cells in cryptorchid testis. Front Endocrinol. 2024;15:1393040. doi: 10.3389/fendo.2024.1393040
45. Koloğlu-Bingöl M, Sara Y, Tanyel FC, Onur R, Büyükpamukçu N, Hiçsönmez A. Contractility and electrophysiological parameters of cremaster muscles of boys with a hernia or undescended testes. J Pediatr Surg. 1998;33(10):1490-4. doi: 10.1016/s0022-3468(98)90482-1
46. Koloğlu-Bingöl M, Demirci M, Tanyel FC, Büyükpamukçu N. Cremasteric reflexes of boys with descended, retractile, or undescended testes: an electrophysiological evaluation. J Pediatr Surg. 1999;34(3):430-4. doi: 10.1016/s0022-3468(99)90493-1
47. Bingöl-Koloğlu M, Anlar B, Büyükpamukçu N, Tanyel FC. Cremasteric reflex and retraction of a testis. J Pediatr Surg. 2001;36(6):863-7. doi: 10.1053/jpsu.2001.23956
48. Favorito LA, Anderson KM, Costa SF, Costa WS, Sampaio FJB. Structural study of the cremaster muscle in patients with retractile testis. J Pediatr Surg. 2018;53(4):780-3. doi: 10.1016/j.jpedsurg.2017.04.013
49. Florou M, Koletsa T, Raptou G, Spyridakis I, Kaselas C. Evaluation of the immunohistochemical and histological differences of the human cremaster muscle in retractile and undescended testis. J Pediatr Surg. 2026;61:163025. doi: 10.1016/j.jpedsurg.2026.163025
50. Hutson JM, Balic A, Nation T, Southwell B. Cryptorchidism. Semin Pediatr Surg. 2010;19(3):215-24. doi: 10.1053/j.sempedsurg.2010.04.001
51. Favorito LA, Favorito LMM, Morais ARM, Sampaio FJ. Role of gubernaculum testis innervation during the process of testicular migration in human fetuses. Int Braz J Urol. 2024;50(4):519-29. doi: 10.1590/S1677-5538.IBJU.2024.0083
52. Cortes D, Thorup J, Lindenberg S, Visfeldt J. Infertility despite surgery for cryptorchidism in childhood can be classified by patients with normal or elevated follicle-stimulating hormone and identified at orchidopexy. BJU Int. 2003;91(7):670-4. doi: 10.1046/j.1464-410x.2003.04166.x
53. Heyns CF, Hutson JM. Historical review of theories on testicular descent. J Urol. 1995;153(3 Pt 1):754-67. doi: 10.1097/00005392-199503000-00026

54. Favorito LA, Costa SF, Julio JR HR, Sampaio FJ. The importance of the gubernaculum in testicular migration during the human fetal period. Int Braz J Urol. 2014;40(5):722-9. doi: 10.1590/S1677-5538.IBJU.2014.05.18
55. Wensing CJG. The embryology of testicular descent. Horm Res. 1988;30(4-5):144-52. doi: 10.1159/000181052
56. Hadziselimovic F. Mechanism of testicular descent. Urol Res. 1984;12(3):155-7. doi: 10.1007/BF00283413
57. Gapany C, Frey P, Cachat F, Gudinchet F, Jichlinski P, Meyrat BJ, et al. Management of cryptorchidism in children: guidelines. Swiss Med Wkly. 2008;138(33-34):492-8.
58. Scorer CG, Farrington GH. Congenital deformities of the testis and epididymis. London: Butterworths; 1971.
59. Hutson JM, Hasthorpe S. Abnormalities of testicular descent. Cell Tissue Res. 2005;322(1):155-8. doi: 10.1007/s00441-005-1110-6
60. Turek PJ, Ewalt DH, Snyder HM 3rd, Duckett JW. Normal epididymal anatomy in boys. J Urol. 1994;151(3):726-7. doi: 10.1016/s0022-5347(17)35063-4
61. Favorito LA, Sampaio FJB. Anatomical relationships between testis and epididymis during the fetal period in humans (10 to 36 weeks postconception). Eur Urol. 1998;33(1):121-3. doi: 10.1159/000019543
62. Logsdon NT, Gallo CM, Sampaio FJ, Favorito LA. Epididymal disjunction anomalies in undescended testis – a factor associated with spermatic obstruction. Int Braz J Urol. 2022;48(2):336-46. doi: 10.1590/S1677-5538.IBJU.2021.0571
63. Caterino S, Lorenzon L, Cavallini M, et al. Epididymal-testicular fusion anomalies in cryptorchidism are associated with proximal location of the undescended testis and with a widely patent processus vaginalis. J Anat. 2014;225(4):473-8. doi: 10.1111/joa.12226
64. Lee PA, Houk CP. Cryptorchidism. Curr Opin Endocrinol Diabetes Obes. 2013;20(3):210-6. doi: 10.1097/MED.0b013e32835ffc8d
65. Hutson JM, Thorup J. Evaluation and management of the infant with cryptorchidism. Curr Opin Pediatr. 2015;27(4):520-4. doi: 10.1097/MOP.0000000000000233
66. Burgmeier C, Dreyhaupt J, Schier F. Comparison of inguinal hernia and asymptomatic patent processus vaginalis in term and preterm infants. J Pediatr Surg. 2014;49(9):1416-8. doi: 10.1016/j.jpedsurg.2013.12.016
67. Favorito LA, Costa WS, Sampaio FJ. Relationship between the persistence of the processus vaginalis and age in patients with cryptorchidism. Int Braz J Urol. 2005;31(1):57-61. doi: 10.1590/s1677-55382005000100011
68. Han CH, Kang SH. Epididymal anomalies associated with patent processus vaginalis in hydrocele and cryptorchidism. J Korean Med Sci. 2002;17(5):660-2. doi: 10.3346/jkms.2002.17.5.660
69. Gill B, Kogan S. Cryptorchidism. Current concepts. Pediatr Clin North Am. 1997;44(5):1211-27. doi: 10.1016/s0031-3955(05)70558-9
70. Parker RM, Robison JR. Anatomy and diagnosis of torsion of the testicle. J Urol. 1971;106(2):243-7. doi: 10.1016/s0022-5347(17)61621-4
71. Elder JS. Epididymal anomalies associated with hydrocele/hernia and cryptorchidism: implications regarding testicular descent. J Urol. 1992;148(2 Pt 2):624-6. doi: 10.1016/s0022-5347(17)36670-9
72. Marshall FF. Anomalies associated with cryptorchidism. Urol Clin North Am. 1982;9(3):339-47.
73. Favorito LA, Cavalcante AG, Costa WS. Anatomic aspects of epididymis and tunica vaginalis in patients with testicular torsion. Int Braz J Urol. 2004;30(5):420-4. doi: 10.1590/s1677-55382004000500010
74. Noske HD, Kraus SW, Altinkilic BM, Weidner W. Historical milestones regarding torsion of the scrotal organs. J Urol. 1998;159(1):13-6. doi: 10.1016/s0022-5347(01)64008-3
75. Sakurai H, Ogawa H, Higaki Y, Yoshida H, Imamura K. Torsion of appendix of testis and epididymis: a report of 4 cases. Hinyokika Kiyo. 1983;29(12):1657-68.

Correspondence address:
Luciano Alves Favorito, MD, PhD
Rua Professor Gabizo, 104/201
Tijuca, Rio de Janeiro, RJ, 20271-320, Brasil
Fax: + 55 21 3872-8802
E-mail: lufavorito@Yahoo.com.br