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The development and closure of the processus vaginalis

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Summary: It is now well known that the failure of the processus vaginalis (PV) to close off following descent of the testis not only accounts for nearly all inguinal hernias of childhood [Rothenberg et al, 1953], but also for other common congenital conditions of childhood such as scrotal hydroceles and encysted hydroceles of the cord. Despite the direct relationship of the PV to these conditions and its intimate association with the mechanism of testicular descent, surprisingly little is known about this important structure. It is not known, for instance, how the PV develops, what precise role it plays in testicular descent, what makes it close, or exactly when it closes. The purpose of this paper is to review the past and present literature on the PV, examining specifically its role in testicular descent, its postulated modes of development, and the mechanisms of its closure and disappearence following descent of the testis.

Key words: Processus vaginalis — Inguinal hernia — Testicular descent — Gubernaculum

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Development of the processus vaginalis

In males, the testis develops at the cranial end of the mesonephros. It is joined to the area of the future inguinal canal by a continuous column of mesenchyme known as the gubernaculum testis. In the abdomen the gubernaculum forms a well-defined ridge, the plica gubernaculi, projecting into the abdominal cavity and carried on a mesentery continuous with the testicular mesentery, or mesorchium. Prior to inguinoscrotal descent, the *processus vaginalis* (PV) commences to form around the junction of the *plica gubernaculi* and the gubernacular mesenchyme at the site of the future inguinal canal, as an evagination of the coelomic cavity into the gubernacular mesenchyme [Backhouse and Butler, 1960; Bergin et al, 1970]. The evagination extends around three sides of the column of mesenchyme, leaving the posterior surface of the gubernaculum devoid of peritoneum.

The invading PV hollows out the gubernaculum and divides it into three parts [Backhouse and Butler, 1960] (Figs. 1, 2): The *plica gubernaculi*, the intra-coelomic column of mesenchyme extending from the caudal pole of the testis to the distal extremity of the PV. The *pars vaginalis* is a thin layer of mesenchyme surrounding the PV, and from which the cremaster muscle develops. It is continuous posteriorly with the *plica gubernaculi*. Distally it merges with the *pars infravaginalis*. The *pars infravaginalis*, is the gubernacular mesenchyme distal to the PV and into which the processus continues to grow.

The PV closely follows the outgrowth of the gubernaculum with the fundus

of the cavity never being more than 1-2 mm from the gubernacular tip [Wensing, 1968]. In consequence the PV is a blind pouch, open at its cranial end into the peritoneal cavity, and lined with coelomic epithelium. [Wyndham, 1943]. The open sleeve of the processus grows longer as the testis moves downwards [Scorer, 1962]. Its walls are apposed before testicular passage through the inguinal canal.

The PV invades the gubernacular tissue in advance of the testis [Wyndham, 1943; Scorer, 1962]. The testis, gubernaculum and PV do not burrow through the preformed layers of the abdominal wall dragging them to form the coverings of the cord [Backhouse, 1964], as has been suggested [Skandalakis et al, 1993] and still stated in anatomy texts [Johnston and Whillis, 1954]. The abdominal wall muscles and inguinal canal differentiate around the gubernaculum and PV. The cremaster muscle, and both the internal and external spermatic fascia are derived from the *pars vaginalis* [Tayakkanonnta, 1963; Backhouse, 1982], and not from the abdominal wall muscles. The nerve supply to the gubernaculum and cremaster muscle is from the genital branch of the genitofemoral nerve, which traverses the inguinal canal. This nerve supplies branches to the cremaster muscle on its route to the bottom of the scrotum, where it turns back up to supply the gubernaculum from a caudal to cranial direction [Tayakkanonnta, 1963].

Inguinoscrotal descent commences at about the seventh month [Scorer, 1962; Hutson, 1985] with rapid passage of the testis through the internal inguinal ring. After passage through the inguinal canal, the gubernaculum and PV extend toward the scrotal floor [Wensing, 1968]. Further descent of the testis is brought about by lengthening of the *pars vaginalis*, moving the testis, situated at the apex of the *plica gubernaculi*, closer to the bottom of the scrotum [Backhouse, 1964] (Fig 2). Finally, there is shortening of the *plica gubernaculi*, either due to a loss of the extracellular component of the gubernacular mesenchyme [Wensing, 1968] or as a consequence of its involvement by further growth of the testis and cauda epididymis [Backhouse and Butler, 1960]. At the same time, there is also rapid growth of the vas deferens and testicular vessels which allows testicular descent to occur [Wensing, 1968]. Adequate development of the PV is essential for testicular descent, and is dependant on an intact gubernacular core [Backhouse, 1964]. However, a patent PV does not guarantee testicular descent since undescended testes are often accompanied by a patent hernial sac [Jones, 1966]. The PV provides a channel through which the testis can descend [Backhouse, 1964], as the testis is a truly intra-abdominal organ descending inside the patent PV, rather than remaining retroperitoneal.

Following closure of the PV between the internal inguinal ring and the upper pole of the testis, the most distal part of the processus persists as the tunica vaginalis [Gier and Marion, 1969]. It is often stated that the tunica vaginalis has a visceral and parietal layer [Johnston and Whillis, 1954]. This is incorrect. During early development of the testis the coelomic peritoneum is absorbed into the tunica albuginea [Bergin et al, 1970]. As a result, the testis lies wholly within the peritoneal cavity during descent [Gier and Marion, 1969]. The PV becomes only the 'parietal' tunica vaginalis [Gier and Marion, 1969]. Similarly, the ovary, has no true peritoneal coat on the part that projects into the peritoneal cavity.

The initial phase of development of the PV is the same in females [Wilson et al, 1981; Skandalakis et al, 1993]. There is no further development of the PV or gubernaculum after 17 weeks [Backhouse, 1984], with the PV persisting as the Canal of Nuck until its closure. The gubernaculum differentiates and forms the suspensory ligament of the ovary and the round ligament of the uterus [Attah and Hutson, 1991].

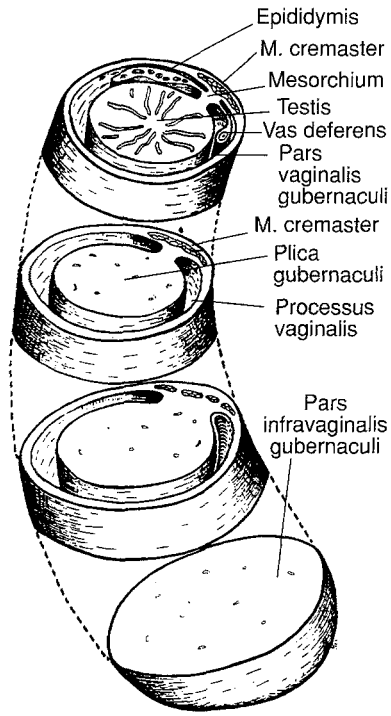

Fig. 1
Schematic sectional representation of PV and gubernaculum testis, removed from the scrotum shortly after testicular descent. Reprinted with permission of Cambridge University Press from Backhouse K.M. [1982]

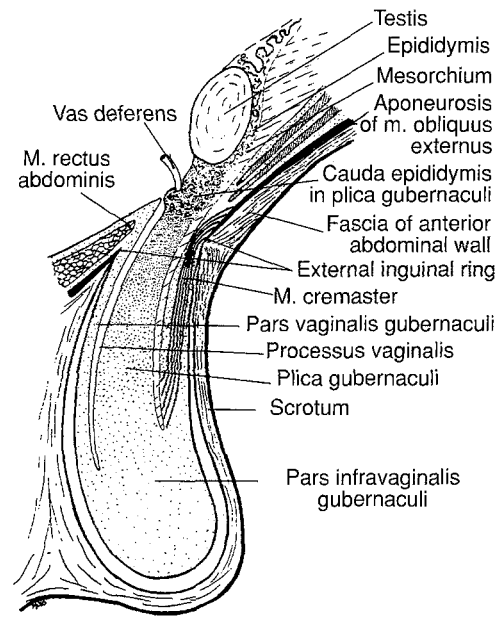

Fig. 2
Schematic representation of the gubernaculum testis and PV shortly before testicular descent, showing its component parts and also its relationship with the surrounding structures. Reprinted with permission of Cambridge Uuniversity Press from Backhouse K.M. [1982]

It is often stated that the PV and round ligament in the female extends as far distally as the labium majorum. This is based on the assumption that the labium majorum and scrotum are homologous, and therefore the gubernacular attachments should be the same in both sexes. In fact the gubernaculum in the female fails to reach the labium majorum and attaches just outside the external inguinal ring [Attah and Hutson,1991]. Moreover the Canal of Nuck extends to a level just distal to the external inguinal ring. This is borne out by the fact that inguinal hernias in girls do not occur in the labial folds, but remain pubic in position.

Despite many theories, the factors controlling evagination of the PV into the gubernaculum remain unknown. Some theories represent the evagination of the PV as a secondary phenomenon, while others propose an actively invading structure.

The most popular theory of PV development is that it is 'pushed out' by raised intra-abdominal pressure. Gier and Marion [1969] propose that the PV represents a hernia in a weak triangle resulting from increased fluid pressure in the abdomen. This increased fluid pressure is thought to result from separation of the intra-coelomic and extra-coelomic cavities following closure of the umbilical ring. Bergin et al [1970] attribute the elongation of the PV to gubernacular swelling, which results in increased fluid pressure on the processus in the inguinal canal. The increased intra-abdominal pressure may result from the rapid growth of the gut and its distension with meconium, while others [Shrock, 1971] have suggested that the development of the PV coincides with the herniation of the mid-gut out of the abdominal cavity at a time of high intra-abdominal pressure.

Hunter [1926] counteracts the argument that intra-abdominal pressure cannot act selectively on the testis by pointing out that each testis lies in a gutter of peritoneum at the entrance of the inguinal canal, and although the pressure within the abdomen acts equally in all directions, it pushes the testis because it lies at the opening of the inguinal canal. Despite this, the pressure theory fails to explain why the PV develops to such a great extent in males, but is only rudimentary in the female. It also fails to explain asymmetrical descent of the testis [Radhakrishnan et al, 1979], which is best explained, rather, by asymmetrical development of the gubernaculum and PV due to local factors [Heyns and Hutson, 1995; Radhakrishnan et al, 1979].

Curling [1841] and others [Pearson, 1956; Lewis, 1948] believed that muscular fibres from the gubernaculum passed up to the testis in the abdomen and that contraction of these fibres served to draw the testis and PV into the inguinal canal. These traction theories of development of the PV fail to explain why the PV forms prior to descent of the testis, because although a close linkage exists between these events, the PV still invades the gubernacular tissue ahead of the testis [Scorer, 1965; Hessert, 1910]. The PV was not thought to be an evagination, but a portion cut off from the peritoneum by encroachment of the anterior abdominal wall during its formation. Wyndham [1943] thought that the PV formed due to growth changes of the abdominal wall, rather than by proliferation.

The most plausible theory of development of the PV is active invasion of the gubernacular core by the PV. Evidence for this comes from the finding of actively growing cuboidal epithelial cells with visible mitotic figures at the tip of the PV, in contrast to the flattened mesothelium of the surrounding peritoneum [Backhouse, 1964]. Active invasion may explain why the PV becomes large and well developed in the male, but remains rudimentery in the female. This theory is supported by the finding in human embryos, that the inguinal canal already contains the gubernaculum, PV, and cremaster muscle prior to both testicular descent and differentiation of the abdominal wall musculature [Pearson, 1956]. That testicular descent is preceeded by very rapid distal growth of the PV provides further evidence for active invasion. [Scorer, 1965; Hessert, 1910].

Active migration of the PV requires a specific signal or controlling factor. The development of the PV is best separated into two distinct phases - an initial phase of development which is the same in both sexes, and a subsequent phase occurring only in males. It is therefore likely that the factors controlling the first phase of development may well differ from those controlling the second phase. Early formation of the PV may be genetically determined by an as yet unknown mechanism. Androgens are implicated in the subsequent PV development in males as this occurs at a time when large amounts of androgen are being produced by the fetal testis [Backhouse, 1964]. However, evidence against a direct role for androgens comes from the failure to identify androgen receptors in samples of human PV from males aged 4 months to 65 years [Johansen and Klein, 1993].

Closure and disappearance of the PV

In females the PV fuses and obliterates, while in males, the upper part of the PV between the internal inguinal ring and the upper pole of the testis undergoes closure and disappearance, leaving the most distal part persisting as the tunica vaginalis [Bergin et al, 1970]. Two poorly understood aspects of this important event in the life of the PV are the exact time at which closure and disappearance takes place, and the mechanisms involved.

There are many theories on the timing of closure and disappearance of the PV, deduced from autopsies, looking specifically at the patency of the PV, and studies looking at the patency of the contralateral PV in inguinal hernial repair. Mitchell [1939] examined 19 term babies and found that the PV was patent in 82% of males. The patency rate in females was only 60% at birth. The results from several studies [Rowe et al, 1969; Clausen et al, 1958; Gilbert and Clatworthy, 1959; McLauthin and Coe, 1960] on the incidence of patency of the contralateral PV at various ages, during bilateral inguinal exploration in cases of clini-

cally unilateral hernia, suggest a patency rate at birth of about 70-80%. This gradually decreases to about 30-40% by 3-4 years of age, although Anson et al [1960] only found 5.1% in 254 adult autopsies. These studies make it difficult to predict the exact ontogeny of closure of the PV. However, PV is partly or completely patent in up to 80% of infants. By 1 year of age, the patency rate seems to fall considerably to around 30-40% . After 1 year of age, the patency rate falls much more gradually, reaching a plateau by 3-5 years of age. Autopsy findings suggest that the left PV closes first. The right sided PV remains patent in infancy twice as often as on the left side. In dissections of newborns, Mitchell [1939] found that the PV was closed on the right side in 30% of cases at birth, compared with 40% on the left. This has always been suspected clinically, as inguinal hernias occur twice as frequently on the right side [Atwell, 1962].

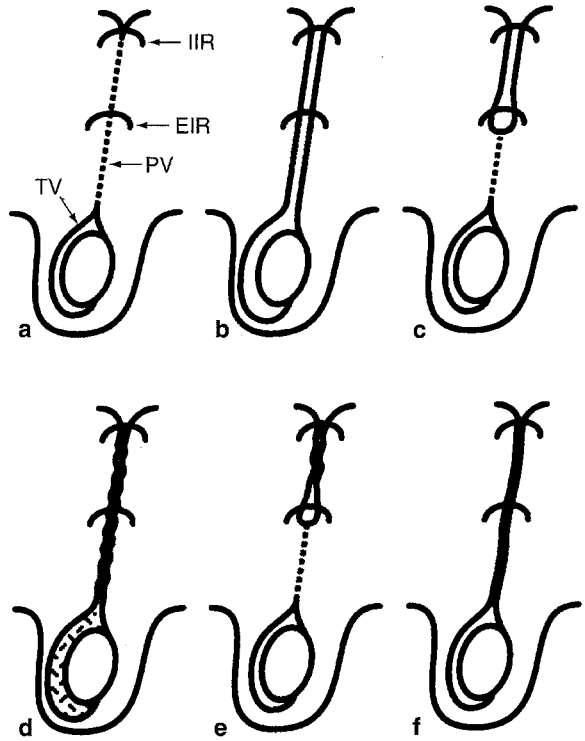

Fig. 3 a-f
a Normal obliteration of the PV between the internal inguinal ring (IIR) and the upper pole of the testis, leaving only the most distal part of the processus vaginalis patent as the tunica vaginalis (TV). **b** Complete patency of the PV which allows abdominal contents to prolapse through the inguinal canal to the bottom of the scrotum, resulting in an inguino-scrotal hernia. **c** Proximal patency of the PV leading to the common inguinal hernia presenting as a lump just outside the external inguinal ring (EIR). **d** Narrow patency of the PV allows intraperitoneal fluid to trickle down and collect around the testis to produce a hydrocele. **e** Narrow patency of the PV extending just beyond the external inguinal ring resulting in an encysted hydrocele of the cord. **f** Obliteration of the lumen of the PV but persistence of the tissues as an inelastic fibrous remnant prevents normal elongation of the cord, leading to relative ascent of the testis out of the scrotum as the boy grows

The first event that occurs in the disappearance of the PV is the fusion of its lumen, followed by subsequent disappearance of the mesenchyme [Mitchell, 1939; Hessert, 1910]. The PV may become occluded by zygosis, which is defined as a process of developmental adhesion, not preceeded by inflammatory changes. Russell [1899] postulated that the mesothelial lining of the PV prevented fusion, since the use of a truss failed to cure infant inguinal hernias. In anatomical dissections carried out by Lockwood [1888] the interior of the PV was always smooth and shining, and he concluded that there was no evidence to suggest that the PV closed by adhesions. It is not known whether PV closure and disappearance occurs in response to a specific signal or whether it is a spontaneous event occurring in the absence of a mechanical barrier. The high incidence of a patent PV in association with congenital undescended testes [Jones, 1966; Johansen, 1988; Herzog et al, 1993] suggests that the PV can only close off once the testis has descended [Scorer, 1962]. Alternatively, the mechanisms of testicular descent and PV closure may be linked. Evidence from patients with testicular feminization (TFM) suggest that androgens are linked to the closure of the PV. Inguinal hernias are present in most cases of complete TFM, in which complete end-organ resistance to androgen occurs [Griffin and Wilson, 1980]. The presence of an undescended testis may be a physical barrier to prevent closure of the patent PV, however, this does not explain patency where the testis is intra-abdominal. Further evidence linking androgens to the closure of the PV comes from the degree of androgen-dependent epididymal development [Elder, 1992], in the presence of a patent PV. Epididymal abnormalities are more frequent when there is a patent PV [Herzog et al, 1992]. Other evidence arises from studies using hormonal treatment for cryptorchidism [Bica and Hadziselimovic, 1993; Herzog et al, 1992]. A combination of luteinizing hormone- release hormone (LH-RH) and human chorionic gonadotrophin (hCG) reduced the frequency of a patent PV from 69% to 31%. LH-RH and hCG both result in increased androgen secretion by the testis, implicating androgens in PV closure. The action of androgens is likely to be indirect, since androgen receptors have not been detected in the PV [Johansen and Klein, 1993]. It has previously been shown [Larkins et al, 1991] that the genitofemoral nerve (GFN) exhibits sexual dimorphism , with the male GFN having a greater number of cell bodies in the spinal cord. It has been suggested [Hutson and Beasley, 1987] that androgens released from the developing testis masculinize the GFN, resulting in the release of the neuropeptide known as calcitonin gene-related peptide (CGRP), to influence gubernacular migration. Recent work in our labororatory [Paxton, 1993; Albano, 1995; Hutson et al, in press] has led us to hypothesize that CGRP released from the GFN causes closure of the PV by bringing about fusion of its inner mesothelial layer. The location of the GFN in the inguinal canal [Tayakkanonnta, 1963] is quite consistent with such an

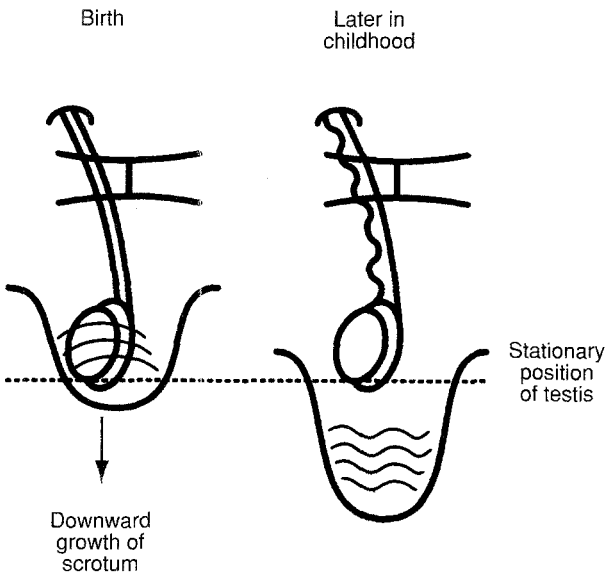

Fig. 4
Schema illustrating 'ascent' of the testis. The testis is fully descended at birth. With further growth the fibrous remnant of the PV prevents elongation of the vas deferens and gonadal vessels. Note that the position of the testis in relation to the pubis has not changed, but appears to ascend out of the scrotum due to progressive growth of the scrotum

hypothesis. It is also postulated that the reason for the postnatal androgen surge found in boys, between 1 and 3 months of age [Gendrel et al, 1980], is to stimulate the GFN to release CGRP in order to bring about closure of the PV. This hypothesis could also explain a role for the GFN in females. The function of the small amount of CGRP present in the female is currently unknown, so it is possible that it is required to bring about closure of the rudimentary PV.

Clinical conditions resulting from the persistence of the PV

Failure of complete closure and obliteration of the PV results in the following clinical conditions. Wide patency at the level of the internal inguinal ring allows abdominal contents, such as bowel, to enter the patent sac, leading to an inguinal hernia (Fig. 3b, c). Entire PV patency results in an inguinoscrotal hernia (Fig. 3b). More commonly, only the proximal portion of the PV remains patent and the hernia presents as a lump at the external inguinal ring (Fig. 3c). Partial involution of the PV leaves a narrow connection to remain between the peritoneal cavity and the tunica vaginalis which only allows peritoneal fluid to enter the sac, thus forming a scrotal hydrocele (Fig. 3d). Occasionally, this narrow connection does not extend all the way down to the tunica vaginalis, and an encysted hydrocele of the cord results (Fig. 3e). It has been proposed recently [Clarnette and Hutson, 1997] that the spectrum of disorders resulting from abnormalities of closure and disappearance of the PV may also extend to include the 'ascending testis', a term used to describe a testis which is undescended later in childhood after apparent normality in infancy [Hutson and Beasley, 1992]. The PV closes, but may fail to disappear, and leave a fibrous remnant within the spermatic cord that prevents elongation of the spermatic cord in proportion to growth [Smith et al, 1989]. Thus, a testis in the scrotum at birth becomes undescended later in childhood (Figs. 3f, 4). The testis does not actually ascend, but rather, remains stationary in position.The persistence of a fibrous remnant at some stage during the normal closure and disappearance of the PV has been noted by several authors [Mitchell, 1939; Johnston and Whillis, 1954], and its presence is a typical finding at operation for correction of the ascending testis .

The hypothesis that the GFN effects closure of the PV via the release of CGRP may explain the indirect role of androgens in the closure of the PV, and, a possible role for the small amount of CGRP present in the female GFN. This is potentially an exciting development as it offers the hope of providing a locally administered agent to bring about closure of the PV, allowing non-operative management for indirect inguinal hernias, scrotal hydroceles, and possibly even, ascending testes.

References

Albano F (1995) Fusion and obliteration of the processus vaginalis. B Sci (Hors) Thesis, University of Melbourne

Anson BJ, Morgan EH, and McVay CB (1960) Surgical anatomy of the inguinal region based upon a study of 500 body halves. Surg Gynecol Obstet 111i, 707-707

Attah AA and Hutson JM (1991) The anatomy of the female gubernaculum is different from the male. Aust. N Z J Surg. 61 : 380-384

Atwell JD (1962) Inguinal hernia in female infants and children. Br J Surg 50 : 294-297

Atwell JD (1985) Ascent of the testis: Fact or Fiction. Br J Urol 57 : 474-477

Backhouse KM (1964). The gubernaculum testis Hunteri: testicular descent and maldescent. A.in Roy Coll Surg Engl 35 : 15-33

Backhouse KM (1982). Embryology of testicular descent and maldescent. Urol Clin N Amer 9 : 315-325

Backhouse KM and Butler H (1960) The gubernaculum testis of the pig. J Anat 94 : 107-121

Bergin WC, Gier HT, Marrion GB, and Coffman JR (1970) A developmental concept of equine cryptorchidism. Biol Reprod 3 : 82-89

Bica TG and Hadziselimovic F (1993) The behaviour of epididymis,processus vaginalis and testicular descent in cryptorchid boys treated with Buserelin. Eur J Pediatr 152 : 38-42

Clarnette TD, Hutson JM (1997) Is the ascending

testis actually 'stationary'? Normal elongation of the spermatic cord is prevented by a fibrous remnant of the processus vaginalis. Pediatr Surg Int 12 : 155-157

Clausen, KG Jake RJ, Binkley FM, Oakland MD, and Calif S (1958). Contralateral inguinal exploration of the unilateral hernia in infants and children. Surg 44 : 735-740

Curling JB (1841). Observations on the structure of the gubernaculum and on the descent of the testis in the foetus. Lancet 2 : 70-74

Elder JS (1992). Epididymal anomalies associated with hydrocele/ hernia and cryptorchidism: implications regarding testicular descent. J Urol 148 : 624-626

Gendrel D, Roger M, and Job J (1980). Plasma gonadotrophin and testosterone values in infants with cryptorchidism. J Pediatr 97 : 217-220

Gier HT and Marion GB (1969). Development of mammalian testes and genital ducts. Biol Reprod 1 : 1-23

Gilbert M and Clatworthy HW (1959). Bilateral operations for inguinal hernia and hydrocele in infancy and childhood. Amer J Surg 97 : 255-259

Griffen JE and Wilson JD (1980). The syndromes of androgen resistance. N Eng J Med. 302 : 198-209

Herzog B, Steigert M, and Hadziselimovic F (1992) Is a testis located at the superficial inguinal pouch (Denis Browne pouch) comparable to a true cryptorchid testis ? J Urol 148 : 622-623

Herzog B, Rosslein R, and Hadziselimovic F (1993) The role of the processus vaginalis in cryptorchidism. Eur J Pediatr 152 : 15-16

Hessert W (1910). The frequency of congenital sacs in oblique inguinal hernia. Surg Gynecol Obstet 10 : 252-256

Heyns CF and Hutson JM (1995). Historical review of theories on testicular descent. J Urol 153 : 754-767

Hunter RH (1926). The etiology of congenital inguinal hernia and abnormally placed testes. Br J Surg 15 : 125-130

Hutson JM (1985) A biphasic model for the hormonal control of testicular descent. Lancet 24 : 419-421

Hutson JM, Albano FR, Paxton G, Sugita Y, Connor R, Clarnette TD, Gray AZ, Watts LM, Farmer PJ, Hasthorpe S (in press). In vitro fusion of human inguinal hernia with associated epithelial transformation. Acta Anat.

Hutson JM and Beasley SW (1987). The mechanism of testicular descent. Aust Paediatr J 23 : 215-216

Hutson JM and Beasley SW (1992) Descent of the Testis. Edward Arnold, London.

Johansen TEB (1988). The anatomy of gubernaculum testis and processus vaginalis in cryptorchidism. Scand J Urol Nephrol 101-105.

Johansen TEB and Klein H (1993) Evidence of androgen receptivity in the pathways of testicular descent in humans. Eur Urol 23 : 466-468

Johnston TB and Whillis J (1954) Gray's Anatomy. Descriptive and applied. 5th Ed. (London, New York, Toronto: Longmans, Green and Co).

Jones PG (1966) Undescended testes. Aust Paediatr J 2 : 36-48

Larkins SL, Hutson JM, and Williams MPL (1991) Localisation of calcitonin gene-related peptide immunoreactivity within the spinal nucleus of the genitofemoral nerve. Pediatr Surg Int 6 : 176-179

Lewis LG (1948) Cryptorchidism. J Urol 60 : 345-356

Lockwood CB (1888). The development and transition of the testis, normal and abnormal. J Anat Physiol 22 : 515-541

McLauthin CW and Coe JD (1960) Inguinal hernia in pediatric patients. Amer J Surg 99 : 45-47

Mitchell GAG (1939) The condition of the peritoneal processus at birth. J Anat 73 : 658-661

Paxton GA(1993) The male gubernaculum. B Med. Sci Thesis, University of Melbourne.

Pearson AA (1956) The relation of the gubernaculum to the development of the inguinal canal in man. Anat Rec 124, 345

Radhakrishnan J, Morikawa Y, Donahoe PK, and Hendren WH (1979). Observations on the gubernaculum during descent of the testis. Invest Urol 16 : 365-368

Rothenberg RE, Barnett T, and Brooklyn NY (1953) Bilateral herniotomy in infants and children. Surg 37 : 947-950

Rowe MI, Copelson LW, and Clatworthy HW (1969) The patent processus vaginalis and the inguinal hernia. J Pediatr Surg 4, 102-106.

Russell RH (1899) The etiology and treatment of inguinal hernia in the young. Lancet 2 : 1353-1358

Scorer CG (1962) The anatomy of testicular descent-normal and incomplete. Br J Surg 49 : 357-367

Scorer CG (1965) The natural history of testicular descent. Proc Roy Soc Med 58 : 933-934

Shrock P (1971) The processus vaginalis and gubernaculum. Surg Clin N Amer 51 : 1263-1269

Skandalakis JE, Colburn GL, Androulakis JA, Skandalakis LJ, and Pemberton LB (1993) Embryological and anatomic basis of inguinal herniorrhaphy. Surg Clin N Amer 73 : 799-802

Smith JA, Hutson JM, Beasley SW, and Reddihough DS (1989) The relationship between cerebral palsy and cryptorchidism. J Pediatr Surg 24 : 1303-1305

Tayakkanonnta K (1963) The gubernaculum testis and its nerve supply. Aust N Z J Surg 33 : 61-67

Wensing CJG (1968) Testicular descent in some domestic mammals. 1.Anatomical aspects of testicular descent. Proc Kon Ned Akad Wetensch c71 : 423-434

Wilson JD, Griffin JE, George FW, and Leshin M (1981) The role of gonadal steroids in sexual differentiation. Rec Prog Horm Res 37 : 1-20

Wyndham NR (1943) A morphological study of testicular descent. J Anat 77 : 179-188