Comparison between Snodgrass vs. Mathieu Repair in Correction of Anterior and Mid Penile Hypospadias

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Abstract

Hypospadias remains a common congenital anomaly. Many techniques are used for repair of anterior and mid-penile hypospadias. Two of them are described in this study; Snodgrass and Mathieu. Snodgrass offers easier type of repair with excellent results and requires less time. Snodgrass and Mathieu give equal cosmetic and functional results. Mathieu remains a primary repair in indicated cases, however, Snodgrass has fewer complications.

Keywords:

Hypospadias- Snodgrass- Mathieu

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Introduction

Hypospadias remains one of the most common anomalies in Pediatric Urology with estimates of 3 per 1,000 births[1,2]. Most of the cases being anterior and mid penile.

A variety of operations have been used for correction of anterior and mid-penile hypospadias including Snodgrass and Mathieu repairs. Both are the most commonly used procedures with many modifications. Snodgrass repair is basically forming the tubularized incised plate (TIP), first described in 1994 by Snodgrass. It gives slit meatal opening and normal appearance of the penis. The details of Mathieu repair with its preliminary results were reported in 1931[3,4]. It is described as single-stage, meatal-based flap for hypospadias repair.

Aim of Work

The aim of this work is to compare the results of Snodgrass and Mathieu techniques in correction of Anterior and Mid Penile Hypospadias.

Embryology and Anatomy of Hypospadias

Normal Embryology of External Genitalia

The embryology of the urogenital region remains controversial and confusing, in part related to the fact that observations have been extrapolated to the human from many different species. There are reports in the literature describing mice[5] and rats[6, 7]. Other studies were done in red squirrels[8], sheep[9], pigs[10] and dogs[11]. Lastly, human studies were done[12, 13].

A comparative study of 57 human embryos (crown-rump length 3-65 mm) and over 140 rat embryos (between 11 and 21 days gestation)[14,15,16] was done and the observations have been correlated with the published findings in the literature in all species in an attempt to distil the common developmental steps in the development of mammalian genitalia. Histological sections of the human embryos are housed at the Anatomy and Developmental Biology Department, St. George's Hospital, London; the Department of Anatomy, University of Cambridge; Department of Plastic and Reconstructive Surgery, University Hospital, Rotterdam; and the Institute of Anatomy, UFR Biomedicale des Sts Peres, Paris. The observations form part of a thesis for a Doctorate of Medicine, University of Melbourne[14,15,16].

The Cloacal Cavity and the Cloacal Plate

The cloacal cavity is the most caudal part of the definitive endodermal gut tube in the developing embryo. Cranially it is in continuity with the more caudal part of the gut tube (the hindgut) posteriorly and (in the human) with the allantoic diverticulum anteriorly. The tail gut exits the cloacal cavity caudally.

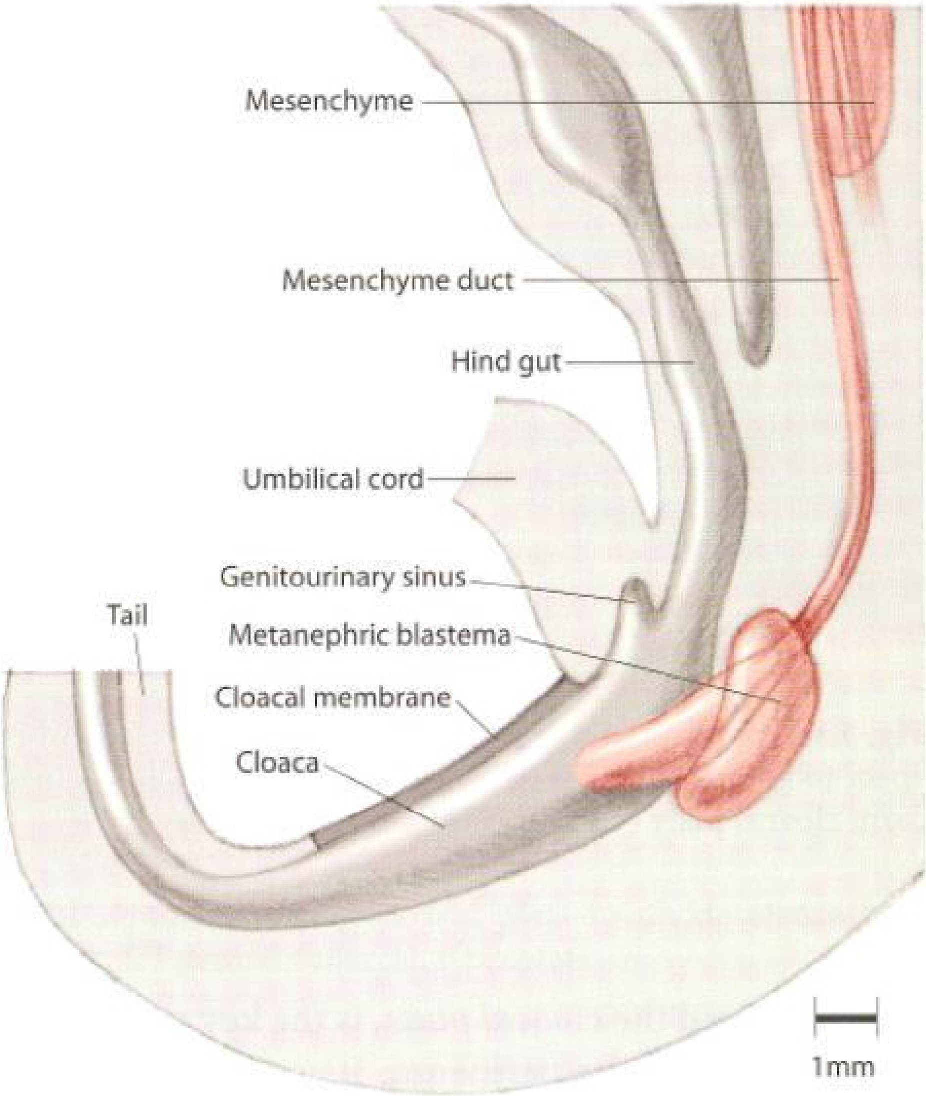

Fig.1: Midsagittal reconstruction with mesenchyme duct and metanephric blastema. It was done by reconstruction of 17 serial sections of day 12 rat embryos. The tailgut is caudal to cloaca, and cloacal membrane is present in tail[14,15,16]

The cloacal membrane limits the cloaca ventrally at first, but proliferation of the infraumbilical mesenchyme in the midline leads to the formation of the early genital tubercle and displaces the cloacal cavity caudally within the tubercle.

In addition, the endodermal lining of the cloaca becomes thickened and apposed side-to-side in the sagittal plane. This results in an endodermal plate of cells orientated in the midsagittal plane extending from the tail fold to the tip of the genital tubercle which then forms the floor of the cloacal cavity over the whole length of what had been the cloacal membrane[14,15,16].

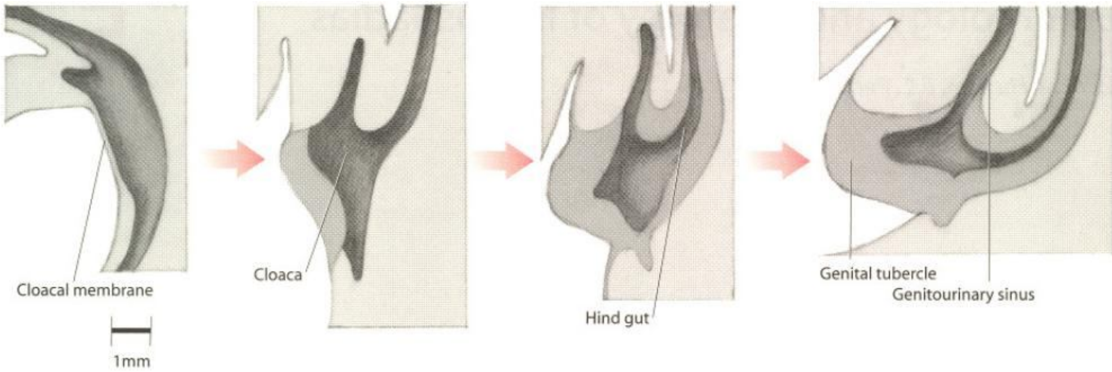

Fig.2: Composite diagrams of midsagittal sections of day 12-13 rat embryos showing formation of genital tubercle[14,15,16].

This nearly solid plate of cells, which can be termed the *cloacal plate,* is the key structure in development of the perineum. Its complete description allows the development of the urethral plate to be more clearly understood[14,15,16].

The Fate of the Cloacal Plate

In both rat and human embryos the cloacal plate gradually loses height posteriorly as the urorectal septum and the perineum approximate and the hindgut gains an outlet onto the perineum. The timing of the changes in the cloacal plate compared with growth and apoptosis in the structures adjacent to it varies across species[14,15,16]. In human embryos (and pigs) the cloacal plate reverts to a two-layer membrane posteriorly, breaking down soon afterwards to expose the anorectal and urogenital tracts, as reported by Nievelstein et al.[13] (1998) and others[17, 18.] Rupture of the cloacal membrane creates a midline groove in the perineum lined by endoderm.

In rats, on the other hand, the urorectal septum meets the top of the cloacal plate (which maintains a vertical height along its entire length), giving the appearance of fusion between the septum and the plate (often described as fusion between the urorectal septum and the cloacal membrane). The anal canal forms by a process of apoptosis through the epithelial plate, and by the time the canal is patent, the urorectal septum mesenchyme forms the perineum as it does in humans, and the posterior part of the cloacal plate is no longer present.

A review of the key embryology papers by Tourneux[9], van der Putte and Neeteson[10] show that the same pattern of development is followed in sheep and pigs. In Tourneux's drawings from sheep embryos the cloacal plate is shown to revert to a membrane over a very short distance posteriorly such that the urorectal septum appears to meet the *back* of the cloacal plate. Tourneux himself, however, noted that the midline of the perineum was initially formed by the endoderm covering the leading edge of the urorectal septum. Van der Putte found a sequence of development in pigs similar to that described for humans[10].

The ventral part of the cloacal plate persists as the urethral plate in both species, but from the time of rupture of the reformed posterior cloacal membrane in the human (and in pigs), the urogenital sinus and the hindgut are open to the amnion, whereas in the rat there is no definite urogenital opening on the perineum until later in development[19].

The Urethral Plate and Formation of the Urethra

The origin of the urethral mucosa has been variously described as endodermal[5,8], ectodermal[20] or mixed[21]. In the 1950s Glenister[22,23] proposed that the male urethra develops by a process of tubularization of a sagittally oriented epithelial plate, which grows as an anterior extension of the endodermal lining of the genitourinary sinus into the perineal surface of the genital tubercle[22,23]. He proposed that the plate and its contained urethra finally meet up with an ingrowth of epithelium at the tip of the glans to form the fossa navicularis.

Although Glenister's description of urethral development gained wide acceptance, his own study included photographs of the cloacal/urethral plate extending to the tip of the very early genital tubercle as discussed above. Several other authors have described the presence of the urethral plate throughout the length of the genital tubercle [8,11,24,25].

The formation of the urethra from the urethral plate is still a matter of speculation. The long-accepted assumption that the urethra forms by infolding of urogenital folds with midline fusion of the opposing sides has been challenged in recent times. Van der Putte's study[10] in pigs failed to find evidence of midline urethral fusion on morphological grounds, and studies looking for evidence of epithelium to mesenchyme transformation, the hallmark of epithelial fusion, also failed to find evidence to prove that fusion plays a role in the formation of the urethra[25].

The presence of the perineal raphe is often cited as evidence for fusion playing a role in the formation of the urethra. It is worth noting, however, that other sites of skin fusion, such as the midline posteriorly over the vertebrae, are not marked by a skin "raphe". In hypospadias, the raphe can be seen to split at the distal limit of the spongiosus, with a "branch" extending to each corner of the dog-ears. Such a bifurcation gives the notion that the skin marking and the urethra form exclusively by a process of fusion. Within the genital tubercle the urethral plate is hourglass in cross section, resembling urethral duplication.

In female rat embryos the urethral plate undergoes massive apoptosis around day 19, exposing the internal surface of the urogenital sinus to the amnion and causing the genital tubercle to become folded down onto the perineum. Unopposed growth of the superior aspect of the genital tubercle accentuates the chordee resulting from involution of the urethral plate[14,15,16]. In the male rat embryo the urethral plate becomes canalized progressively until the lumen finally reaches the tip of the genital tubercle. As also reported by van der Werff et al.[5] (2000), canalization results from apoptosis within the urethral plate. At the same time, the developing urethra becomes gradually separated from the external surface of the genital tubercle by growth of mesenchyme between the plate and the surface skin. There is no evidence of an ectodermal ingrowth from the tip of the phallus joining with the urethral plate.

The urethral plate similarly extends to the tip of the genital tubercle throughout development in human embryos. The urogenital sinus is open to the amniotic cavity from the time of breakdown of the cloacal membrane, but in other respects the anatomy is as described above for the rat. In particular, no significant ingrowth of ectoderm is seen in the developing glans. It is possible that Glenister formed his opinion that ectoderm may be involved in male urethral development because of the stratified squamous epithelium of the urethral plate. The evidence, however, supports the view that this is entirely endodermal in origin, as is the oesophageal mucosa and the female urethra[26].

Some authors have proposed that the urethral plate is derived from an ectodermal fusion of the inner surfaces of paired genital swellings[5, 6,8,27]. However, careful study of both the rat and the human embryo failed to show any ectodermal contribution to the urethral plate.

Phallus and Prepuce

Enlargement of the genital tubercle occurs by growth of the infraumbilical mesenchyme[28]. Simultaneously the opening of the urogenital sinus is carried forward onto the shaft of the phallus by growth of mesenchyme from the urorectal septum and the developing perineal body. As the urethral plate is canalizing by apoptosis, forward growth of the mesenchyme possibly contributes to development of the corpus spongiosum.

The prepuce begins to form as solid lamellae of epithelial cells (the glans lamellae) that dip into the genital tubercle dorsally and laterally, their proximal extent defining the floor of the coronal groove. The glans lamellae extend laterally and ventrally and become confluent with the margin of the urethral plate, and dorsally with its opposite fellow. Epithelial proliferation and desquamation causes a split in the lamella and raises a fold of skin, which then grows distally as a sleeve that covers the dorsum and sides of the glans. The frenulum and mid-ventral foreskin form last as growth of the ventral penile mesenchyme reaches the tip of the penis.

Abnormal Development in Hypospadias

Abnormal morphogenesis in hypospadias affects three main anatomical features: (1) the ectopic urethral orifice, (2) the abnormal foreskin, including irregular penile raphe and dorsal hood, and (3) the chordee, or congenital bend in the penis observed on erection[29].

Ectopic Orifice

The primary anomaly in nearly all cases of hypospadias is failure of the midline perineal mesenchyme to grow ventrally to cover the urethral plate as it canalizes. Incomplete morphogenesis is the commonest embryological defect, and in hypospadias the opening of the urethra is commonly arrested at or near the coronal groove of the glans. This position is normal in 9- and 10-week human embryos as described by Clarnette et al. in 1997[30]. Where the opening is more proximal on the penile shaft or in the perineum, it suggests a more severe local anatomical anomaly or a defect in androgenic action. The most proximal opening in the perineum, representing the original opening of the urogenital sinus and the normal site of the urethra in females, is seen in complex anatomical defects of perineal morphogenesis (often with imperforate anus) or in forms of intersex.

Failure of the urethral plate to be covered by mesenchyme (and skin) leaves the outer genital folds separated as two hemiscrota. An open midline gutter with pin-point orifices of presumed Littre's glands distal to the ectopic orifice indicates the persisting urethral plate.

Dorsal Hood and Raphe

The dorsal hooded prepuce is characteristic of hypospadias and may be explained by failure of androgen-dependent growth of the ventral penile mesenchyme. This leaves a wedge-shaped defect in the ventral prepuce, including an absent frenulum. At each corner of the dorsal hood, the bifurcated penile raphe ends in a "dog-ear". The dog-ears represent the most distal points of external preputial skin that would normally be joined together. In the more severe forms of hypospadias where the dorsal skin of the penis remains fused with the scrotal folds, there are no dog-ears.

The median raphe of the phallus is abnormal in hypospadias. Deficiency of mesenchymal growth along the shaft may lead to a zigzag course of the raphe. Just proximal to the ectopic orifice the raphe bifurcates, with each branch continuing distally to the dog-ears on the prepuce. These raphe branches indicate the distal edge of migration of the mesenchyme that forms the Buck's fascia and subcutaneous tissue, which are lacking in the triangular area between the branches. In cases where the raphe bifurcates some distance proximal to the orifice, the urethra is very superficial and lacks adequate supporting tissue and corpus spongiosum.

Chordee

Chordee is present in most patients with hypospadias, but is related to the severity of the underlying anomaly. In a group study of 46 patients with hypospadias, chordee was present in all perineal or scrotal anomalies (7/7), 15 of 16 cases with penile or coronal hypospadias, and in 11 of 23 of those with glanular hypospadias[31]. The perineal and scrotal anomalies showed feminine development of the phallus consistent with possible testicular dysplasia and/or abnormal function of androgens[32]. The phallus showed a hairpin bend of the corpora cavernosa similar to the clitoris, which is known to be produced by apoptosis of the urethral plate in the absence of androgenic stimulation[14,15,16].

In more distal variants, the arrest of mesenchymal growth and/or the initiation of apoptosis occur later, leading to progressively less severe chordee, with most of the chordee caused by deficient peri-urethral growth rather than a bend in the corpora. In glanular hypospadias, chordee also may result from deficient growth of the distal urethral plate[33], although the appearance of a short urethral plate may simply result from relatively greater growth of the dorsal side of the developing glans.

Blood Supply of the Penile Skin

Most cases of hypospadias, regardless of severity, are associated with a prepuce that could be sufficient to create a neourethra to bridge the existing gap and cover the repair with skin. Success is in a major part dependent on three preconditions:

- The inner layer of the prepuce has to be long enough.
- The epithelial surface of the prepuce has to be adequate.
- The subcutaneous tissue between the outer and inner layer of the prepuce has to be sufficient and carry enough blood vessels for vascularisation of the two layers.

However, any technique using the prepuce for the repair jeopardizes the blood supply, endangering the result. Knowledge of the blood supply of the prepuce is essential so that preservation of the preputial vascularisation is not merely fortuitous.

Embryology

At approximately 8 weeks of gestation the penis and the glans develop from the genital tubercle. At this point the prepuce has not yet developed. At the beginning of the 10th week symmetrical low ridges arise just proximal to the coronal sulcus, progressing dorsally until they form a fold surrounding the glans, leaving the ventral midline uncovered, where they are blocked by the incompletely developed urethra[34]. This nascent prepuce rolls over the base of the glans, leaving a groove between it and the coronal sulcus. This groove is filled simultaneously with actively proliferating glanular lamella, an ingrowth of epithelium of multiple cells in thickness[22,23]. Due to an enormous active proliferation of the epithelium at the apex (proximal end) of the groove, the ridge of the preputial fold is pulled towards the summit of the glans, which is then overtopped owing to proliferation of the mesenchyme of the epithelial layers. By the 30th week of gestation the single epithelial layer between the developed prepuce and the glans begins to split into two layers from the distal ridge[35]. This procedure continues till birth when the two layers have been formed. They are still connected with the glans by desquamated epithelium.