

Sphincter-like motion following mechanical dilation of the internal inguinal ring during indirect inguinal hernia procedure

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Abstract

Introduction Even today, there is still great speculation as to the underlying pathogenesis of inguinal hernia. As a result, it could be extrapolated that the vast majority of repairs are based upon conjecture. Most current repairs are founded upon the principle of “closing the defect” in the anatomy, either by suturing closed under tension, covering with a mesh or obliterating the defect with a plug. Many variants of each method are refined to achieve better clinical outcomes. Yet few, if any, strive to understand a fundamental question: “What has gone wrong with the normal physiological and anatomical mechanisms that prevent abdominal structures protruding through the abdominal wall?” We consider, in the normal subject, the muscular structures that converge and wrap around the inguinal canal as a highly dynamic structure, which forms a reactive barrier to the augmentation of intra-abdominal pressures. In effect, the structures work together like a “striated sphincter complex.” Through years of surgical experience, we have seen the formation of adhesions and fibrosis in these delicate and key structures, and hypothesised that they may impair its shuttering action, thus, creating a patency of this jammed inguinal ring leading to hernia. Based upon these observations, we have created a

hernia repair variant that tries to “unblock” the muscles prior to repair, thus, hopefully restoring a degree of physiologic function.

Methods A retrospective study describes the results of 47 patients operated for indirect inguinal hernia with a standardised procedure consisting of meticulous adhesiolysis of the hernia area and mechanical dilation (divulsion) of the inguinal orifice in order to break stiff fibres within the muscle, allowing viable muscle fibres to contract freely once more. After dilation, a proprietary lamellar-shaped implant was delivered into the canal. Its form and function are designed to eliminate impingement of the cord structures and give a gentle outwards force to induce a reactive contraction of the sphincter-like muscle complex during healing. This gentle contraction offers the possibility to eliminate fixation of the implant.

Results The removal of scar tissue, dilation and the introduction of the implant into the internal inguinal ring induced a forceful “gripping” contraction by the sphincter complex in all patients. Even without fixation, it became almost impossible to pull the implant out of the canal. After obliterating the orifice with the lamellar implant, it was clear that there was no dilative compression upon the cord structures.

Conclusion The results of this combined procedure, scar removal, dilation and implant delivery, led to thoughtful suggestions regarding the anatomy and the physiology of the inguinal canal. The procedural adhesiolysis during indirect inguinal hernia repair has always shown the well described concentric muscular arrangement formed by the internal oblique and transversus muscles. This circular-shaped muscular structure is often recognised as a static barrier that, due to weakness and/or together with other causes, fails in its role and allows indirect inguinal hernia protrusion. According to the results of our observations, we

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consider this concentric muscular complex as a dynamic formation: we will use the term “striated sphincter complex.” Its steady tightening motion after divulsion and the insertion of a lamellar implant is always accompanied by a strong gripping action, which is not seen prior to divulsion. This indicates that it could correspond to a sphincter: the “inguinal sphincter.” The impairment of this sphincter could be the cause of the inguinal canal’s patency and the development of hernia.

Keywords Inguinal hernia · Indirect · Inguinal ring · Adhesions · Fibrosis · Sphincter

Introduction

Hernia of the abdominal wall is one of the most common diseases, resulting in over 700,000 hernia procedures performed each year in the USA alone [1]. It is estimated that 75% of all hernias are groin or inguinal hernias, with 66% being indirect and 33% direct [2].

Despite the continuous improvement regarding surgical technique and materials, the amount of intra- and post-operative complications, as well as the recurrence rate, remains unacceptably high, especially when considering non-specialist hernia centres. Also, patient comfort is still a major issue, due to incision size, the amount of dissection required and the nature of the implants currently used today [3–7].

Although over 2 million meshes are placed each year for hernia repair [8], there is still uncertainty about the pathogenesis and the aetiology of inguinal hernia, and, thus, the current repairs are probably based upon conjecture.

In this report, we have revisited the question regarding the genesis of the inguinal hernia, but starting from evidence that is usually not taken into consideration.

The first evidence regards the anatomical structure of the inguinal canal; the internal oblique and transverse abdominal muscles at this level form a muscular structure that wraps around the spermatic cord in a concentric fashion [9, 10]. This resembles, in our opinion, in form and function, an anatomical sphincter. This simple concept seems to be obvious, but is rarely found in the medical literature. There are, in a few scientific reports, the descriptions of an “internal inguinal sphincter” specifically related to the internal inguinal orifice [10, 11]. Conversely, we have not found in the literature a description of an “external inguinal sphincter.” For this reason, we think that this muscular structure should simply be described as the “inguinal sphincter.”

Secondly, some authors emphasise the importance of “dividing” the adhesions between the inguinal canal and the hernia sac during the hernia repair [12]. The existence

of adhesional bands is well known to the surgeons, but the relationship between thicker, fibrotic bands and hernia occurrence has been, so far, undervalued, or not considered, as an element of the progressive hernia disease [13, 14]. The presence of adhesions, in our experience, is not restricted only to the surface of the inguinal ring and hernia sac, but is also clearly evident within the muscle fibres of this “inguinal sphincter.” We have noted this through tactile sensation and histological examination. Adhesions and fibrotic bands are commonly considered as a result of the inflammatory process. So why are these “scar” structures present and what is their impact on the functionality of the tissues? Do these stiff fibrotic bands impinge on the described, dynamic sphincter-like function?

Material and methods

From January 2005 until April 2007, 47 male patients underwent an open anterior inguinal hernia repair. Thirty-five patients had primary indirect inguinal hernia and 12 recurrent hernia. Local anaesthesia was administered in 24 patients, spinal in 19 and general in four.

In all patients, we carried out a standardised procedure consisting of:

1. Dissection of the hernia sac from the cord structures
2. Amputating the redundant hernia sac at the level of the external inguinal orifice and suturing it closed
3. Procedural adhesiolysis between the remaining hernia sac and, respectively, external inguinal orifice, inguinal canal, internal inguinal orifice to the level of the abdominal cavity
4. Returning the amputated and closed sac stump into the abdominal cavity
5. Dilation (divulsion) of the inguinal canal “inguinal sphincter complex” with a dilating cylinder (oversized in relation to the width of the inguinal canal)
6. Insertion of a multi-lamellar polypropylene implant within the inguinal canal (Fig. 1)

Results

Some features of the described procedure, such as steps 1 and 2, are typical aspects of every anterior procedure for inguinal hernia.

Also, as in step 3, the adhesiolysis is usually performed by many surgeons, but we have considered the division of the fibrotic bands between the sac and the canal structures as a highly crucial step in the procedure. Thus, we have always carried out a meticulous division of the adhesions to remove any impingement of the surrounding inguinal



Fig. 1 The lamellar polypropylene implant used for delivery into the internal inguinal orifice

structures (Fig. 2). At the end of this practice, we ensure that the internal inguinal ring, released by the scar tissue, has regained its anatomical identity.

The aim of this intricate step was to isolate the muscular structure surrounding the spermatic cord. After returning the amputated sac to the abdomen (step 4), a clear orifice is seen, which is surrounded by a circular, sphincter-like muscular “frame” (Fig. 3).

At this point, as illustrated in step 5, we performed a radically different technical variant by considering this concentric muscular “frame” of the internal inguinal ring as a “blocked” sphincter. Hence, we have performed a mechanical “stretching” (divulsion) of the muscle ring with the purpose of fracturing any restraining fibrotic bands within the muscular structure, liberating any viable muscle fibres. A metallic cylinder, wider than the dimension of the orifice, was forcefully introduced into the orifice to carry

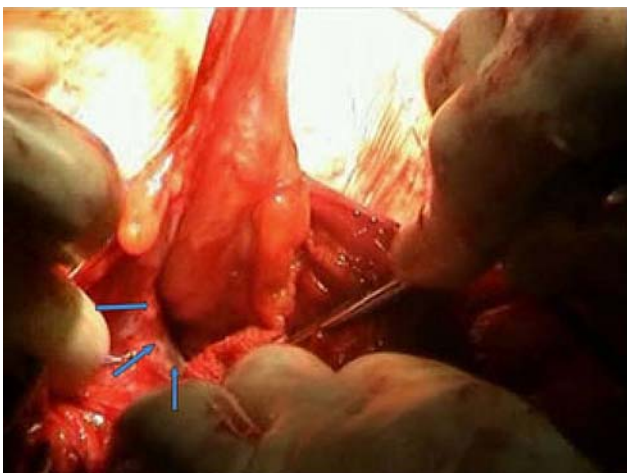


Fig. 2 Before dividing the adhesions, the inguinal orifice is completely blocked by a fibrotic enfold (arrows)

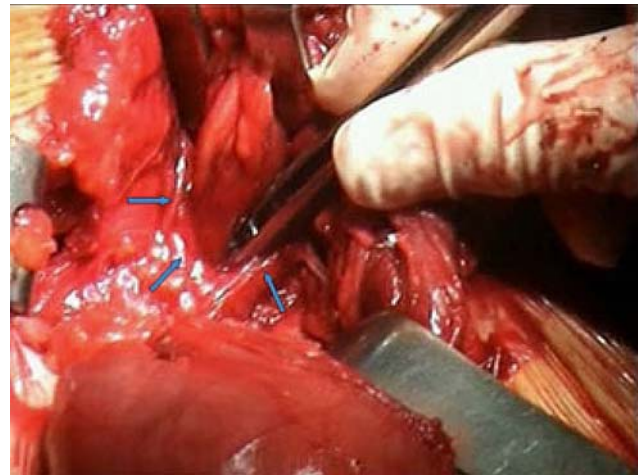


Fig. 3 After dividing the adhesions, the inguinal orifice has been freed from the adhesions (arrows)

out the circumferential “stretching” (Fig. 4). This way, we reached an optimal dilation of the muscle ring in order to break the existing fibrotic bands within the muscle fibres. At the end of the dilation, immediately after the extraction of the cylinder, in all patients, we consistently saw a significant contraction of the sphincter with a remarkable reduction of the residual hole. At this time, as described in step 6, a proprietary implant was delivered into the orifice. To deliver the implant through the contracted inguinal sphincter, we used a cylindrical applicator containing the device. The unique implant had a polypropylene lamellar structure, which, after release, was deployed within the muscular ring. A key design feature is a central point with a connected polypropylene flat mesh. Once the applicator had been removed, the implant remained firmly in place within the inguinal canal. Even trying to remove the implant with force proved futile (Fig. 5). The newly

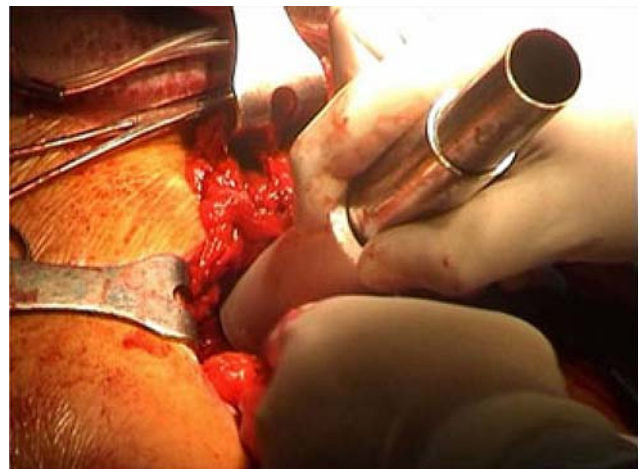


Fig. 4 The applicator is introduced into the inguinal orifice, dilating the “inguinal sphincter.” It has a second cylinder (containing the implant) that slips inside, pushing a plunger

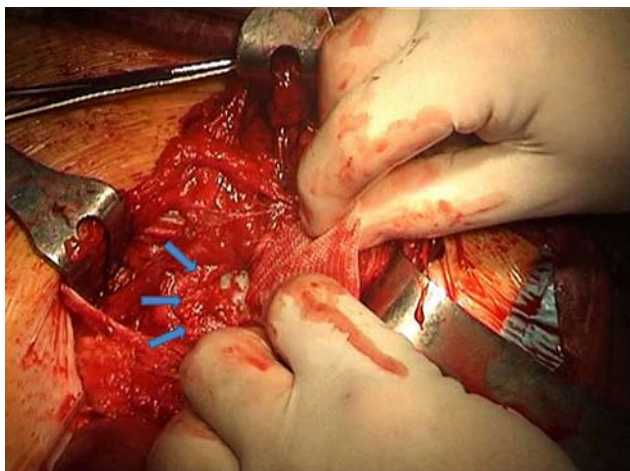


Fig. 5 The implant, connected with a flat mesh, has been released into the inguinal orifice. The internal ring is contracted (*arrows*) and squeezes the implant. Even trying to remove it with force, the implant stays put

functional inguinal sphincter contracted in a powerful way that made implant removal almost impossible. This required no fixation, a step that we feel is critical in reducing chronic pain in hernia patients.

The unique lamellar structure of the implant is thought to allow the spermatic cord to run alongside the lamellas without any dilative compression of the spermatic vessels—a feature of this device in reducing the possible post-operative complications of the cord and testicle (Fig. 6)

The portrayed method has shown consistent observations in all patients; after a methodical take-down of all restraining adhesions, the “inguinal sphincter” can be easily expanded by the dilation cylinder. After the divulsion, the insertion of an adequately sized implant into the

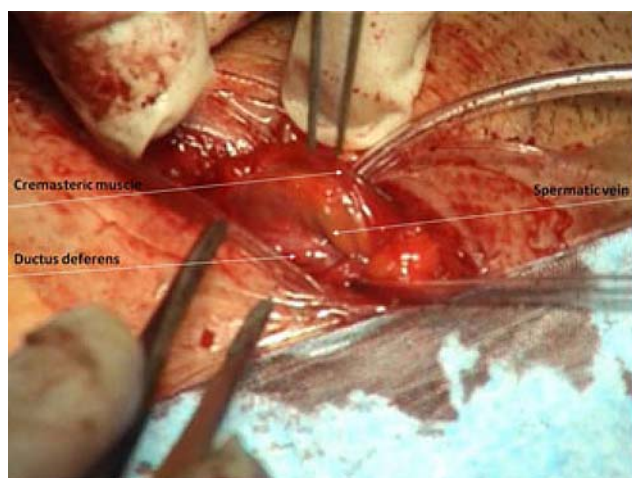


Fig. 6 After obliterating the orifice with the implant, no dilative compression of the spermatic vessels was seen. They are not impinged by the implant

dilated inguinal structure, such as the lamellar implant, constantly led to a powerful “gripping” contraction. It became almost impossible for the inserted implant to be dislodged, even when trying to remove it with force. More so, the implant was forcibly gripped due to a physiologic reflex which induced the sphincter to contract, seemingly a proprioceptive nervous impulse activated by proprioceptive gamma-cell pathways.

Discussion

This report should not be intended as a description of a new surgical technique or device.

The aim of the article is to share with the surgical community the features of our observations. The anatomical structure of the inguinal canal has been accurately defined over centuries. The literature gives a shared description of the muscular ring surrounding the spermatic cord: it is shaped by the fibres of the internal oblique and the transversus abdominis muscle confluencing in a circular fashion and forming a rounded structure. Many authors portray the structure of the internal oblique and the transverse, at the level of the inguinal opening, as a dynamic and not a static barrier [11, 15]. Lytle [16] imagines the motion of these muscular structures as an “inguinal sling” and Stoppa depicts a shutter mechanism such as a curtain closure [17]. These theories are well accepted in the scientific community, although, so far, there is no clinical evidence in this regard. In our opinion, according to our intra-operative observations, we noted that, if stimulated through dilation, the movement of the internal inguinal ring is comparable to a concentric sphincteric motion.

All sphincters, including smooth muscle, combined smooth and striated muscle, and striated muscle sphincters [18], are being subjected to use (or abuse) and structural changes may alter their functional aptitude to close off the inguinal orifice. This may also apply to a striated muscle sphincter-like complex in the inguinal region.

Possibly, the growth of adhesions in the region of the inguinal canal is a result of chronic, repeated microtrauma. The recurring trauma could be the basis of the inflammatory reaction between the parietal peritoneum and the abdominal wall, causing the adherential bands surrounding the inguinal orifice and along the hernia sac [14, 19], as well as fibrotic changes within the muscular structure of the sphincter. Consequently, the contractile functionality of the inguinal sphincter decreases and, at length, the adhesences cause a functional sphincter blockade. Possibly, the dynamic failure of the “inguinal sphincter,” combined with increases in abdominal pressure, could represent the reason for the passage of the abdominal viscera deeper into the inguinal canal. If the sphincter cannot contract, it leaves a

patent orifice. With increased abdominal pressure, viscera are funnelled to the weakened sphincter and, step by step, penetrate across the inguinal orifice, inducing deeper damage and further loss of function to the structure. Without the effective muscular sphincter to act as a “closing mechanism,” the inguinal canal becomes steadily dilated and the hernia may outwardly protrude.

Thus, adhesions and fibrosis could represent the cause of an inguinal sphincter’s insufficiency that leads to the formation of indirect hernia.

In the described surgical practice, a meticulous adhesiolysis within the hernia field, taking care to achieve a complete freeing up of the muscular structure outlining the internal inguinal ring, is the first step in releasing the “inguinal sphincter.” The second step of forceful dilation restores its motile function and contraction. In our clinical experience, the dilation and the successive insertion of a multi-lamellar polypropylene implant in this muscular frame has constantly led to a powerful grabbing contraction of this sphincter-like structure. After the fixation-free delivery, even trying to remove it with force, the implant stays in place.

The observed results after this kind of surgical approach gives us confidence regarding the effective return of the contractile activity of the inguinal muscle ring, “inguinal sphincter.” These suggestions might help to explain why the occurrence of an inguinal hernia in the presence of patent processus vaginalis in childhood is infrequent. The reason appears obvious: despite the patency between the abdominal cavity and the scrotal sac, if the shuttering motion of the “inguinal sphincter” is effective, the intrusion of the abdominal viscera through the inguinal canal (during increases in intra-abdominal pressure) will be avoided. We also consider the results of the latest studies regarding the biochemical changes in the connective tissue in the inguinal region when an inguinal hernia is present [20–22]. We believe that an understanding of the changes in the collagen matrix of patients with hernias is only a partial understanding of the total problem. This may be the underlying genesis of the problem, but we have aimed to describe how the depicted changes in the tissues may result in a chronic process for the actual formation of the hernia. In fact, the fibrotic bands that, in our opinion, impair the inguinal sphincter are also made of connective tissue. Consequently, it is conceivable to depict the formation of adhesions in this area as an unusual and disproportionate inflammatorily response in the framework of the collagen production. Thus, it seems logical that the chronic changes in the collagen matrix (for any proposed reason—smoking, age, genetics) of these patients, combined with the increased stiffness of the muscle fibres, may lead them to a high susceptibility of the injury and inflammatory processes that we describe. Ultimately, this could lead to the

blocking of the inguinal sphincter and the formation of an indirect inguinal hernia.

Conclusion

A standardised adhesiolysis followed by mechanical dilation of the internal inguinal ring performed in patients with indirect inguinal hernia has led us to thoughtful considerations regarding the anatomical configuration and the function of the inguinal structures. The described procedural steps, in our patient sample, has always shown a sudden contraction of the internal inguinal ring, accompanied by a gripping action if a lamellar implant was delivered into this circular muscle frame. This, in our opinion, corresponds to a sphincter-like structure: “the inguinal sphincter.” In our suggestion, in case of impairment resulting from adhesional inflammation, the consequential sphincter blockade could be the cause of the inguinal canal’s patency leading to hernia formation. This implication, resulting from steady intra-operative findings during 47 indirect hernia repair procedures, has to be portrayed only as a suggestion to the surgical community, in order to give opportunity to deepen the depicted characteristics with further physiological and histological studies.

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