

Reply to the Letter to Editor by Prof. Desarda: inguinal hernia repair: the hypothesis postulated in the article is not true

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Professor Desarda,

Firstly may I thank you for your letter to the editor [1] with regard to the article “Sphincter-like motion following mechanical dilation of the internal inguinal ring during indirect inguinal hernia procedure” [2]. I think we are in agreement that this is an important subject which deserves expanded debate and deeper scientific enquiry. You have raised several important questions which some time ago we, as a group, also asked and have used to expand our scientific investigations. This is not the forum to discuss all points in detail, but I would like to reply to a few of your important questions.

- I would like to clarify a key point of the article in which you thought we were describing a new hypothesis on inguinal hernia *repair*. We clearly open our discussion by saying “This report should not be intended as a description of a new surgical technique or device.” This statement was to ensure that the description of our procedure was not the main subject, but to give background to the paper. The intent of the article was merely to share with the surgical community our observations following a mechanical dilation of the internal ring during indirect inguinal hernia repair. We clearly declare that it is not a fully formed “theory”, but the observation may lead to a possible hypothesis on which we are currently working.
- You raised the point: “The adhesions and fibrosis seen by G. Amato et al. in the operations performed by them are the results of hernia formation and do not precede hernia formation. They should have done inguinal canal dissec-

tions in normal individuals or cadavers to draw such conclusions and postulate such hypothesis.” We also agree this is a key point and hence are doing extensive cadaver studies both at a macroscopic and, importantly, also at a microscopic level. In this article, we were simply reporting observations and were not drawing any conclusions about normal subjects.

- With regard to looking at the morphopathological changes, we have carried out a study concerning histological examination of tissue samples excised from both patients and cadavers with inguinal hernia. We have collected very interesting data which will form the subject of scientific reports due to be submitted for publication. As a foretaste, I attach one of the many pictures of histological specimens which demonstrate, in the indirect hernia border, the occurrence of phlogistic infiltration; the inflammatory infiltrate is commonly depicted as a cause of adhesion (Fig. 1). In the same picture you can observe the fibrotic and fibroadipous degeneration of the muscle fibers. We are still interpreting the histology to determine if the changes are precursors to the hernia or are a result of it. We would welcome your collaboration in this subject and be delighted to have your opinion.
- You reference the article published by B. J. Anson [3] concerning inguinal canal dissection for anatomical study. We are hoping to build upon this data by performing specific dissections and histological studies in cadavers and patients with hernia. Our study is specifically looking at the muscular structures bordering the hernia to determine what pathological changes have occurred. Our first report on the histological findings of the internal ring in living patients having indirect inguinal hernia has been published in Online First, edition of *Hernia* [4]. We hope this article starts to explain some of the questions you raise.

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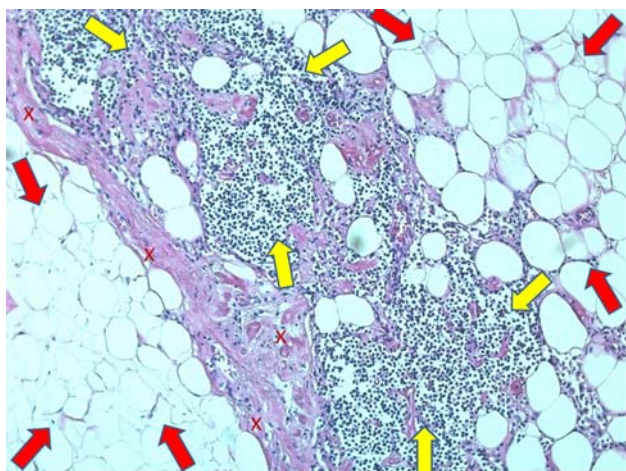
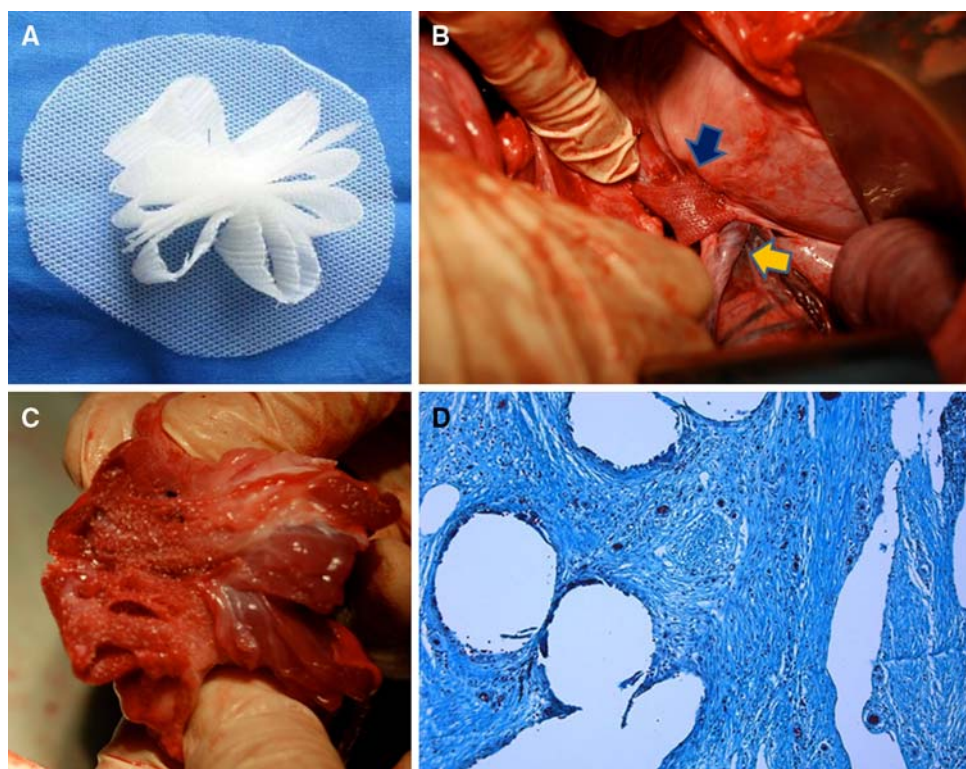


Fig. 1 Histology of cadaver specimen with indirect inguinal hernia: lymphohistiocytic infiltrate (*yellow arrows*). Fibrotic degeneration of the muscle fibers (*X*). Fibroadipous dystrophy of the myocytes (*red arrows*) (H&E $\times 10$)

In the spirit of collaboration, we would like to briefly answer some of the other excellent points you raised (following your numerical ordering)—points which we have also discussed as a group several times.

1. The article is not actually a “retrospective study”. In this article we did not give results of the interventions; those results are due to be published in a formal “retrospective study” in the near future. To clarify, this
2. To be clear, we did not undertake, or wish to imply, the removal of the intramuscular fibrotic elements. The breaking (not removal) of the intramuscular scar fibers was achieved through the divulsion with a properly sized dilation cylinder.
3. As can be seen in Figs. 2, 3, 4 and 5 of the article [2], the inguinal ring is clearly dilated by the hernia protrusion, but covered by a fibrotic enfold (Fig. 2). After adhesiolysis, the “scar” envelope has been removed (Fig. 3). After the dilation (Fig. 4), the sphincter-like motion of the internal ring has been activated again (Fig. 5).
4. Our use of the word “meticulous” was not intended to mean 100% “complete” adhesiolysis but as complete a removal as possible of any impairment to the shuttering motion of the internal ring.
5. We agree, there is the possibility of a recurrent fibrosis, especially if the underlying causes of the adhesions and fibrosis have not been removed. However, this was not the focus of this article. We are thinking of design implications with respect to the implant to try to minimise this recurrence. This is the focus of a future article.
6. This is an important point. We believe that through the careful design of the implant, fibrotic regeneration and

Fig. 2 **a** Handcrafted implant. **b** Implant inserted in the groin of pig 4 months after delivery. Intra-abdominal view: *blue arrow* polypropylene disc, *yellow arrow* spermatic cord. **c** Implant explanted after 4 months transected in the midline. The shape of the implant is still conserved, with no evident shrinkage. **d** Histology of the implant. Mild giant-cell infiltrate. Lax and woven connective in-growth with evident neoangiogenesis (H&E $\times 10$)



implant shrinkage should be minimised. Our implant depicted in this article has been modified to add a preperitoneal shield and inserted into the groins of a porcine experimental model. At the explant after 4 months, we saw that the connective growth within the implant appears to be different with respect to that noted in the usual implanted flat meshes and plugs. We saw in-growth of connective tissue appearing to be lax in nature, instead of the often seen fibrotic scar tissue (Fig. 2). With respect to this, we are preparing other reports which highlight the difference between connective in-growth in static and dynamic implants. But again, this was not the focus of this paper and will inform future scientific papers.

7. We are still working to determine if the dilation or the dynamic implant induces the “gripping action” but suspect it is a combination of both. Again this is subject to ongoing studies and was not to be answered in this paper.

With such an interest in this field, we hope to discuss further how we can collaborate.

Best regards,
Giuseppe Amato

References

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