Hernia (2009) 13:453–454
DOI 10.1007/s10029-009-0503-4

LETTER TO THE EDITOR

Inguinal hernia repair: the hypothesis postulated in the article is not true

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Published online: 15 April 2009

Dear Editor,

I read with interest the recent article of Amato et al. [1] describing a new hypothesis of inguinal hernia repair. Amato et al. stated that "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." This hypothesis itself is highly questionable. The adhesions and fibrosis seen by Amato et al. in the operations performed by them are the results of hernia formation and do not precede hernia formation. They should have carried out inguinal canal dissections in normal individuals or cadavers to draw such conclusions and postulate such a hypothesis. Anson et al. [2] have performed inguinal canal dissection in 500 body-halves and clearly mentioned the detailed anatomy of aponurotic extensions from the transversus abdominis aponurotic arch (dessiduous part). The author of this letter to the editor has further highlighted the importance of this, till today neglected, structure as the basic primary factor that prevents hernia formation in normal individuals [3]. The posterior wall of the inguinal canal from the pubic tubercle to the internal ring is not formed by transversalis fascia alone as a single-layer structure, but is formed by two layers. Another layer is of those aponurotic extensions that give real protective cover. Inguinal hernia formation takes place in those individuals in whom this layer is either absent or deficient. The strength of the posterior wall is directly proportional to the number of fibres it contains and not to the transversalis fascia, which is papery thin [4].

Amato et al. should have given answers to many questions such as: (1) How can anybody draw new conclusions in a retrospective study? (2) Can anybody really remove all fibrotic elements, especially when they are intra-muscular? (3) Can anybody really dilate an already dilated and patulous internal ring? (4) How could they measure the completeness of the adhesiolysis in each patient on which their hypothesis is based? (5) Do they mean that there will not be fibrosis again? (6) Do they mean that the prosthesis inserted by them will not result in fibrosis? (7) The results seen by them in their series are because of the prosthesis alone or because of their manoeuvre of internal ring dilatation and adhesiolysis?

The author feels that "(1) meticulous adhesiolysis, (2) mechanical dilation (divulsion) of the inguinal orifice, (3) allowing viable muscle fibres to contract freely, (4) give a gentle outwards force to induce a reactive contraction of the sphincter-like muscle" are all imaginary without any scientific support. The statements in the conclusions section and elsewhere that "This circular-shaped muscular structure, concentric muscular complex" were never heard of or read by the author. Nobody gave such a description of those muscles in the past in any textbook. Amato et al. should have given evidence to such statements also.

The author to this letter to the editor has already published a new method of inguinal hernia repair without mesh that is based on the physiological principle [5] and the new concepts or theories that prevent hernia formation in normal individuals [3]. The readers are requested to read both of these articles for a better understanding of the surgical anatomy and physiology of the inguinal canal.

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