The Complications of Surgical Treatment of Incisional Hernia

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Incisional hernia is a highly frequent clinical entity that affects between 1 and 16%, and according to some reports more than 20% of abdominal surgery patients because of additional decisive or favourable factors that may be of a general or local nature [1,2]. Even small incisions that entail the insertion of a trocar in laparoscopic surgery result in an incidence of 1–3% [3,4]. While most incisional hernias appear during the first 6 months post-operatively, adopting different forms with regard to wall areas and entities. The timing, exact manifestation and progression of the pathology are extremely variable. A common and important feature is that the abdominal wall is anatomically and morphologically damaged, with progressive local and general functional involvement. Moreover, this pathology also has psychological and aesthetic consequences for the patient.

The clinical forms of incisional hernia range from the unrecognised or well-tolerated, small, paucisymptomatic ventral hernias with minimal visceral involvement and only a slight tendency to progressively worsen to extreme manifestations of "parietal disaster" that eventually become "incisional hernia disease" or "eventration disease" and result in major functional impairment.

In large incisional hernias, the anatomical-functional relationships of the chest wall, i.e. the rib cage, the diaphragm and the abdominal wall, are radically altered because of the progressive reduction in endo-abdominal pressure caused by the significant visceral hernia. In a median ventral hernia, the tensing of large muscles opens the rectus muscles such that the normal respiratory activity of the intrabdominal muscles is altered, and the spillage of the internal organs through the parietal fault is stimulated.

The creation of an "abdominal volet" leads to a chronic respiratory syndrome with dyspnea, due to the mechanical effort, and a pulmonary emphysema that depends on the dimensions and persistence of the abdominal hernia as well as the atrophy of the parietal musculature. The latter includes muddy and fatty degeneration with progressive atrophy, necrobiosis and fragmentation of fibres such as in myopathy associated with tendinous rupture [5–7]. Alterations of the pressure gradient inside and outside the lumen interfere with the microcirculation of the intestinal wall, resulting in stretching and hypoperistalsis.

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The treatment of incisional hernia dates back to the middle of the twentieth century; the high recurrence rates associated with the repair procedure led to the use of synthetic prostheses by the 1970s. This approach resulted in a marked reduction in the number of recurrences but a relatively high rate of local infectious complications and other, related problems [8]. Beginning in the 1990s, the use of minimally invasive surgical techniques became widespread and greatly influenced prosthetic "open" surgery [6]. Currently, prosthetic surgery makes use of diverse biomaterials, with numerous forms and mesh structure, surgical techniques and implantation sites.

Synthetic prostheses made of polyglactin and polyglycolic acid are reabsorbable and increase the parietal resistance but only temporarily, because reabsorption takes place after 3–6 months. They are especially used in technically difficult repairs with particular anatomical features [9], such as infected tissues or in the "sandwich" technique in association with a non-reabsorbable prosthesis [10–12]. Reabsorbable prostheses frequently cause recurrences, since after their reabsorption by hydrolysis only a loose connective tissue with little mechanical capacity remains [12]. However, there are fewer local complications and better tolerance by infected tissues of organs in which the use of non-reabsorbable mesh would require its removal. Thus, the use of a reabsorbable mesh is recommended until the septic process has resolved, after which other kinds of prostheses can be implanted [13].

Non-reabsorbable prostheses with a permanent structural function consist of mono-constituent or heterogeneous synthetic polymerics, such as polypropylene, polyester and expanded polytetrafluoroethylene (ePTFE). Due to their physicochemical features, they cause only a slight inflammatory reaction in the tissues and generally do not give rise to infective complications. The monoconstituent meshes of polyester and polypropylene used in abdominal-wall surgery have a high structural porosity and resistance, good stability and allow quick and complete tissue integration as well as an intense connective-tissue proliferation. However, when the prosthesis positioning is properitoneal or, even worse, intraperitoneal, these same features produce dangerous visceral adhesions, erosions, and fistulizations [14,15]. The use of a hydrophobic material with a low porosity, such as PTFE, inside the wall, is associated with a reduced infiltration of fibroblasts and thus poor tissue integration and a high recurrence rate [16]. Nonetheless, the low adhesion of this mesh makes it suitable for being placed in close contact with internal organs in either open or laparoscopic repair.

The creation of mesh with pronounced non-stick properties and consisting of layers of hydrophilic reabsorbable polymers on permanent supports (polypropylene/ePTFE; polypropylene/sodium hyaluronate-carboxymethylcellulose foam; polyester/hydrophilic collagen; polypropylene/regenerated oxidised cellulose/polydioxanone), combined with ePTFE with a double microporous structure, allows good apposition to the peritoneal surface.

Possible sites of intraparietal implants where non-reabsorbable prostheses are used include retromuscular-prefascial [17–20] and premuscular-aponeurotical tissues. In such cases, the extent of dissection and the duration of surgery are reduced [21].

Intraperitoneal collocation can be realised with the open technique in certain situations [22], but can also be done in elective procedures, with well-defined indications, in laparoscopy [23]. The surgical strategy, even if perfectly and fully realised, must be compatible with the clinical manifestation of the pathology and characteristics of the patient. For the latter, this takes into account the patient's global state of health as well as biological features of the tissues, psychological state, immunological fitness, life style, individual compliance and readiness to participate in appropriate clinical follow-up.

Many of the elements involved in the pathogenesis of incisional hernia play a role in the development of complications following surgical repair: metabolic disease and organ failure, tissue hypoxia caused by anaemia or ageing, condition of hypo/malnutrition, wall adiposity, chronic bronchopulmonary disease and previous immunodepressant therapies. In addition, there is the risk of an inadequate surgical procedure or the occurrence of technical mistakes and deficiencies, insufficient patient qualification and a lack of preventive and protective measures [24].

A complete analysis of the complications associated with the surgical repair of incisional hernias, by either laparotomy or laparoscopy, should make reference to homogeneous patient groups with respect to the pathological entity, as this provides recourse to shared classifications, technical principles and the chosen approach, as defined by the results of large prospective studies [25,26].

Recurrence

The recurrence rate is an important element to establish the efficiency of surgical treatment. The incidence of recurrence in incisional hernia prosthetic surgery is markedly lower than in direct plasties. Indeed after the autoplasties of the preprosthetic period, the recurrence rate ranged from 14–50% for ventral hernias [27,28]. Chevrel and Flament, in 1990, reported on 1,033 patients who had undergone laparotomy. The recurrence rate at 10-year follow-up was 14–24% for patients treated without the use of prostheses but only 8.6% for those in whom a prosthesis was implanted [10]. A similar incidence was reported by Chevrel in 1995: 18.3% recurrence without prostheses, 5.5% with prostheses [29]. Likewise, Wantz, in 1991, noted a recurrence rate of 0–18.5% in prosthetic laparo-alloplasties [30].

At the European Hernia Society (EHS)-GREPA meeting in 1986, the recurrence rate without prostheses was reported to be between 7.2 and 17% whereas in patients who had been treated with a prosthesis the recurrence was between 1 and 5.8% [25].

A case study published by Flament in 1999 showed a 5.6% recurrence rate for operations with prostheses placed behind the muscles and in front of the fascia, and a 3.6% of such figure consisted of a small-sized lateroprosthetic recurrence. These rates were in contrast to the 26.8% recurrence reported by other surgeons for operations without prostheses [31].

Studies of recurrence are, of course, influenced by the size of the initial defect and the length of follow-up. Nevertheless, it is beyond dispute that the use of prostheses is associated with a lower rate of recurrence independent of the nature of the incisional hernia [32].

The factors that lead to relapse are recognisable in the original features of the ventral hernia, i.e. combined musculo-aponeurotic parietal involvement, septic complications in the first operation, the nature and appropriateness of treatment, the kind of prosthesis and its position. Also important is whether the surgery was an emergency case and the relation to occlusive phenomena, visceral damage and whether these problems were addressed at the same time.

Obesity is also an important risk factor for recurrence. In addition to its association with a higher surgical complications rate, related to the high intra-abdominal pressure, there are deficits in wound cicatrisation as well as respiratory and metabolic pathologies. In such patients, the laparoscopic approach is very useful to significantly reduce the onset of general and wall complications, and the data concerning recurrence are encouraging [33,34], ranging between 1 and 9% in the largest laparoscopic case studies [35–39]. The important multicentric study of Heniford et al., in 2000, reported a recurrence rate of 3.4% after 23 months [1]. In 2003, the same author, in a study with an average follow-up of 20 months (range 1–96) showed a recurrence rate of 4.7% for different, identifiable causes: intestinal iatrogenic injuries and mesh infection with its removal, insufficient fixation of the prosthesis and abdominal trauma in the first postoperative period [40].

The incidence of recurrence after laparoscopic treatment may also be related to general patient factors and to the onset of local complications, mistakes in opting for laparoscopic treatment and deficits in implanting and fixing the prosthesis. With respect to the latter, it is very important to allow a large overlap compared to the diameter of the defect.

Long-term data analysis, with large case studies, is still needed to obtain detailed information about recurrence, and this is particularly true in the assessment of relatively new techniques.

Respiratory Disease Caused by Postoperative Abdominal Hypertension: Abdominal Compartment Syndrome

In the treatment of large eventrations, the forced reduction of the viscera caused by this pathology and by reconstruction and closure of the wall under high tension, may lead to intra-abdominal hypertension and secondary organic malfunction. In addition, the surgical effort to re-establish wall functionality to curb evolution of the pathology through large and multiple prostheses exposes the patient to the risk of a serious intra-abdominal hypertension. Dangerous or even lethal

clinical manifestations can appear during the first 30 h post-operatively. The abdominal hypertension may also have local consequences, including intestinal, renal, hepatic, circulatory, respiratory and neurological ones, which in the absence of proper decompression and identification of the aetiology, can lead to multi-organ failure. Patients may also have an important dyspnoea, tachypnoea and reduction of the tidal volume. Radiological examination of the thorax will highlight a lifting of the diaphragmatic cupulae and an evident basal atelectasis. Blood-gas analysis may indicate hypoxia, acidosis and hypercapnia.

Abdominal hypertension can be determined subjectively as a sensation of heaviness; the pain felt by the patient during palpation of the abdomen is intense.

The alterations in renal haemodynamic parameters described in the abdominal compartment syndrome (ACS) are similar to those of adult respiratory distress syndrome (ARDS), multi-organ failure (MOF) and sepsis. ACS may initially be misdiagnosed or even go unrecognised. Many of its clinical manifestations are identical to those observed in the syndrome of systemic inflammatory response (SIRS) or in septic shock.

It is therefore fundamental to surgically respect the compliance of abdominal cavity [41–43], to administer a respiratory functional evaluation and to suitably prepare the patient for surgery. The technique of pre-operative pneumoperitoneum, which was aimed at reducing many of the above-described complications, was described by Moreno in 1947 [44].

In laparoscopic treatment, the intraperitoneal position of the prosthesis does not restrict the wall, thus complying with the “tension free” principle. While reduction of a large intestinal mass in the abdomen could, at least theoretically, lead to this complication following the treatment of large ventral hernias, there is no consensus as to whether laparoscopy is indicated in such cases.

Mortality

From the above discussion it is clear that there are serious risks in terms of the postoperative respiratory and multivisceral insufficiencies caused by the abdominal compartment syndrome. Postoperative mortality is predominantly a consequence of septic complications, especially in cases of unrecognised intestinal-loop perforation and intra-abdominal abscess, both of which may arise during laparoscopic surgery; this is in contrast to open surgery in which morbidity is most often due to wall complications [45,46].

Throemboembolism causes deaths in 1% of cases [47]. In a large case study carried out in 1990 by the French Surgical Association and involving 1,825 prosthetic alloplasties, the mortality rate was 1.2% [10]. A mortality rate of 0.6% was determined by Flament in a series comprising 1,517 operations carried out in 1999 [31].

Protracted Postoperative Ileum

Postoperative ileum has an unpredictable duration and clinical course. The incidence of this complication following laparotomic surgery was found to be 8% according to a 1998 study [47]. In laparoscopic treatment, it appears occasionally, especially as a consequence of difficult operations, extensive adhesiolysis, intestinal tractions and the use of large prostheses [38]. Heniford quoted an incidence of 2.2%, based on 407 laparoscopic operations carried out in 2002, and 3% in a series of 850 treatments performed in 2003 [40].

Pain

Postoperative pain is reduced in prosthetic surgery compared to direct plasty [48] and is further minimised in laparoscopic surgery. In either case, pain can be well-controlled pharmacologically.

Symptomatology is usually related to areas of particular tension, especially sites of transparietal stitches, and the methods of intraperitoneal fixation. However, with time, pain in these regions eases and disappears due to the plastic adaptation of the involved anatomical structures.

Chronic pain may be a consequence of prosthesis retraction and the method of fixation, both of which may produce algogenic tension on the affected tissues [49]. An inadequately fixed prosthesis or one placed in a reduced space can adopt the conformation of a "meshoma" and act as a pain-producing stimulus [50].

Infiltration with a local anaesthetic prior to skin incision of trocar sites is very useful in laparoscopy [35,38] and provides pain control when the patient wakes up from anaesthesia.

Parietal Rigidity in Prosthetic Surgery

Non-reabsorbable mesh must be able to adapt a form compatible with the parietal wall while maintaining adequate tensile resistance. These prerequisites are fulfilled by most of the currently avalable prostheses. Indeed, with respect to resistance to pressure and tension forces, they are more than adequate.

A surgical technique that does not respect the "tension free" principle, when combined with an exuberant fibroblastic integration, can influence the rigidity of abdominal wall. As a result, the patient may feel constant discomfort, with the potential development of clinical respiratory and/or haemodynamic disorders due to the reduced parietal excursion [51].

When surgeons place a mesh during laparoscopic treatment, attention must

be paid to the distensibility of the prosthesis, which, when fixed after reduction of the pneumoperitoneal pressure, must have a flexible configuration. Also important is the surgeon's awareness of the pressure established with surgical clips, the composition of the clips and the use of biological glues either alone or in combination with other fixation methods. The retraction factor of some prostheses must also be considered; with time, there may be a 7–8% reduction in surface area.

Seroma

Seroma is one of the most frequent complications in laparoscopic prosthetic surgery and in open surgery but its resolution is in most cases spontaneous. It is commonly noted on postoperative ultrasound but it is otherwise subclinical. According to large clinical trials, seromas lasting more than 8 weeks are considered as a complication [52]. An incidence of 1.97% [1] among 407 patients treated with laparoscopic technique in 2000 and 2.6% in a series published in 2003 was noted by Heniford [40].

The disappearance rate of clinically relevant seroma is around 7% (range 4–15%) [18,53] in laparotomic surgery and between 4 and 16% [35,38,54] in laparoscopy.

The tissue reaction to the prosthesis in the first postoperative days resembles that of a physiological inflammatory response and precedes the invasion by fibroblasts. It is a consequence of the residual space and the large detachments of skin flaps.

Seroma may become manifest as late as 6 weeks postoperatively, and even later in cases of encysted chronic seroma, which sometimes have a multilocular structure. It most frequently arises from the use of a premuscular position technique (Chevrel) compared to a position behind the muscles and in front of the fascia [24].

In laparoscopic treatment, seroma appears between the intraperitoneal mesh and the wall, in the cavity of abdominal hernial sac.

Surgeons recommend compression for 4–6 weeks, with a bandage shaped according to the diameter of the defect, to reduce the residual space and to allow adhesion of the prosthesis to the hernial sac. In addition, the use of drainages and local compression, in open surgery and in laparoscopic surgery, reduces the incidence of seroma [55].

The repeated aspiration of inflammatory fluid can lead to contamination, with serious consequences that must be surgically managed. A technique that avoids the appearance of this fluid following surgical placement of the laparoscopic prosthesis is to sear the hernial sac with monopolar current or a "harmonic scalpel" or to treat it with laser-argon applications [56].

Wall Haematoma

The frequency of wall haematomas is variable: for laparo-alloplasty it was 4.7% in a 1990 AFC case study [10], 1.8% in Chrevel's case study of 1997 [57], 3% in the 1998 case study of Leber [47] and 0.7% in a trial carried out in 1999 by Flament [31]. In the large review of Heniford, in 2003, the incidence of haematoma following laparoscopic surgery was 0.7% [40].

Haematoma is a predictable complication in prosthetic laparotomy. It can entail huge abrasions and dissections in patients on anticoagulants for the treatment of cardiovascular pathologies or prophylactically to avoid thromboembolic disease. Nonetheless, it is the responsibility of the surgeon to prevent wall haematomas through rigorous haemostasis and proper use of aspiration drainages.

In laparoscopic surgery, wall haematomas can appear when the surgeons places the trocars but they are not a specific complication of the treatment of abdominal hernias; rather, slight bleeding, haemorrhagic suffusions and haematomas can arise due to vessel damage caused by prosthetic fixation methods. These can be recognised by the surgeon and treated immediately.

Cutaneous Necrosis

Cutaneous damage that appears with necrosis has an incidence of 1.2%, according to the AFC study [10], and 0.9% according to Chevrel [57].

Vascular damage caused by traction, extreme compression or devascularisation and thermal insult can cause large areas of necrosis, thus jeopardising the cutaneous integrity and barrier effect towards pathogens. This can lead to a secondary subcutaneous cellulitis and even deep sepsis, with frank prosthetic infection and fistulation.

Particular attention must be given to wall reconstructions involving extensive dermolipectomies and abdominoplasties, because tractions on the skin flaps can evolve into serious necroses that are detrimental for prosthetic alloplasty.

In laparoscopy, a cutaneous necrosis next to an abdominal hernia may develop as a consequence of diathermocoagulation of the sac.

Prosthesis Infection

Septic complication can appear precociously or after a rather long period of time. Laparotomic techniques are historically linked to cellulitis as well as wall and prostheses infections. Stoppa reported a septic complication rate of 12% in 1989 [18].

Surface sepses following prosthetic laparotomy surgery were found in 5.35% of patients in a 1990 AFC study [10], in 7% in the series of Leber in 1998 [47] and in 1% of cases in the Flament series of 1999 [31].

For laparo-alloplasties, Koehler quoted an incidence of 0.5–6%, based on a case study and a literature review [58]. In the important case study on 850 laparoscopic treatments, Heniford reported an incidence of cellulitis of 1.1% at the trocar site while the frequency of mesh infection was 0.7%, thus establishing that such complication are rare in this approach [40].

Prosthesis infection is not an improbable event in large soft-tissue detachments of the abdominal wall. Laparoscopic prosthetic alloplasty, by contrast, which respects parietal structures, avoids the vascular and tissue damage that causes bleedings, haematomas and serious septic complications.

Prosthetic laparoscopic contamination has repercussions at the visceral level, with the potential development of peritonitis, visceral and parietal adherences and coalescences.

It is essential to observe rigorous asepsis during surgical placement of the prosthesis and to employ all possible devices to avoid the formation of intraparietal haematomas. The use of antibiotic prophylaxis [59,60] and biomaterials impregnated with antimicrobial substances [61] reduces the frequency of such complication. In addition, precautionary measures related to surgical technique and details of the procedure, as well as proper care and instruction of the patient play important roles.

Most prosthetic infections are due to cutaneous pathogens that are transported by contaminated prostheses or cutaneous solutions and promoted by conditions favouring necrosis [52].

Deep sepses in the abdominal wall in laparotomic surgery have dramatic consequences. Frequencies of 0.75% [10], 4% [47] and 2.72% [21] have been reported. The EHS-GREPA published a deep suppuration rate of 3–21% [25].

It may be necessary to remove the prosthesis if it becomes septic and causes problems related to wall reconstruction; in other cases, the prosthesis can be preserved by treatment cleansing, extensive mesh exposure and appropriate dressings.

Late infections depend mostly on the kind of prosthetic material used [62]. The incidence of such complication is low, and for polyester prostheses is 0.2–1% according to the round-table findings coordinated by Wantz at the American College of Surgeouns in 1999 [63]. The frequency quoted by Leber in 1998 was 5.9% of late chronic infections and 3.5% of infections related to enterocutaneous fistulas [19].

With the use of ePTFE (expanded polytetrafluoroethylene) prostheses, Martinez showed, in an important literature review, a global late suppuration rate of 4.1% with the consequent need of mesh removal in 8.2% of cases and thus a recurrence rate of 17.5% [64].

Intestinal migration phenomena in isolated cases have been cited in the literature. True migration, if it exists, must be differentiated from enterocutaneous fistulas, which appear after ignored intestinal lesions, precarious suture or

destructive wall phlogosis with visceral involvement. Leber found an incidence of 3.5% in 1998 [47]. Migration of the prosthetic material in the intestinal lumen is more likely to occur in intraperitoneal as opposed to intraparietal positioning [25].

Veress Needle and First Trocar Visceral Lesions

Veress and first trocar visceral lesions refer to general laparoscopic procedures and have an incidence <1% [65]. However, they represent inauspicious events that can be disastrous in the case of large vascular lesions and intestinal perforations or lacerations with massive septic peritoneal contamination.

The choice of access method improves with experience, sensitivity and surgical preferences, but should be made cautiously. Significant reliability of the open access technique has not been proved. In one study, it was only used in 2.5–9% of cases with the remaining being treated with the Veress technique [66,67].

The insertion site of a Veress needle must be far from surgical laparotomy scars or drainages, common sites of intraperitoneal adherences, and from the defect wall or a defect that is diagnosed preoperatively. Sites with both adherent or outspread viscera, and parenchymatous or pathological organs should also be avoided. When the first trocar is placed, the surgeon must respect the triangulations of the laparoscopic implantation, avoiding the wall defect and bony projections that would limit the instruments' excursion [68].

Intestinal Perforation

Intestinal perforation is a serious visceral complication of laparoscopic procedures. In large case studies, the percentage of accidental enterotomies was between 0.5 and 6% [36,37,69]. It most frequently occurs during adhesiolysis necessary to expose the hernial defect and to establish a wall surface that allows placement of the prosthesis with sufficient overlap.

Intestinal perforations arise by different mechanisms: through the direct action of scissors or traumatic instruments or through the indirect insult of energy sources, with the creation of an eschar, or an ischaemic area of intestinal wall with subsequent necrosis and perforation [47].

Intraoperative recognition of intestinal lesion mandates their immediate repair; since ignored visceral damage causes the fearful complication of a deferred perforation with problems of diagnostic timeliness and treatment.

Adhesiolysis must be done cautiously, without traction, with blunt instruments, and should follow the cleavage and avascular planes. Furthermore, it

must be done “cold”, with cautious use of monopolar coagulation; ultrasound and radiofrequency coagulation spread less heat but are not always safe [58,70].

Identification of intestinal damage may require the laparotomy conversion; this must be considered as a necessary treatment strategy rather than a complication.

Conclusions

Surgery to repair incisional hernia has reached a very high efficiency and safety level. Consolidated and recent techniques allow the treatment of all kinds of structural and functional involvements, with important reductions in complications rates. Nonetheless, current findings must be supported by additional numerical and qualitative data as well as careful observations.

For the surgeon, it is important to operate respecting the traditional techniques but with a desire to know and, as needed, employ state-of-the-art techniques. Knowledge of clinical, technical and organisational aspects must be deep-rooted. Similarly, the various kinds of prevention, clinical assessment, training and choice of treatment must be integrated and supported in the surgical procedure.

Currently, the laparoscopic revolution is gaining increasing attention and credibility, thus confirming its feasibility, advantages and reduced rate of postoperative complications. However, an awareness of the postoperative complications together with efforts to prevent them are fundamental elements in achieving therapeutic success.

References

1. Heniford BT, Park A, Ramshaw BJ, Voeller G (2000) Laparoscopic ventral and incisional hernia repair in 407 patients. J Am Coll Surg 190(6):645–650
2. Basile F, Biondi A, Furci M et al (2002) Trattamento dei laparoceli. EMC Roma: Tecniche Chirurgiche-Addominale, pp 40–165
3. Hesselink VJ, Luijendsk RW, de Wilt JH et al (1993) An evaluation of risk factors in incisional hernia recurrence. Surg Gynecol Obstet 176:228–234
4. Leung TY, Yuen PM (2000) Small bowel herniation through subumbilical port site following laparoscopic surgery at the time of reversal of anesthesia. Gynecol Obstet Invest 49:209–210
5. Trivellini G, Contessini Avesani E (2002) Trattamento chirurgico dei disastri parietali. Atti del 104° Congresso della Società Italiana di Chirurgia. Roma, 13–16 ottobre 2002. Luigi Pozzi, Roma, pp 33–47
6. Chevrel JP, Flament JB (1995) Traitement des éventrations de la paroi abdominale. Techniques chirurgicales. Appareil digestif. pp 40–165
7. Rives J, Lardennois B, Pire JC, Hibon J (1973) Les grandes éventrations. Importance du volet abdominal et des troubles respiratoires qui lui sont secondaires. Chirurgie 99:547–563

8. Rives J, Pire J, Convers C (1977) Traitement des éventrations. EMC Paris: Tecniques chirurgicales pp 40–165
9. Sturniolo G (1990) L'impiego delle protesi reticulate in Dacron nella chirurgia dei grandi laparoceli. Chirurgia 3:573–578
10. Chevrel JP, Flament JB (1990) Les éventrations de la paroi abdominale. In: Rapport au 92ème Congrès Français de Chirurgie. Monographies de AFC. Masson, Paris
11. Champault G (1988) Place de treillis résorbables dans le traitement des éventrations postopératoires. J Chir 125:27–29
12. Trivellino G, Danelli PG (1991) L'impiego di due protesi in contemporanea nella riparazione di grosse perdite di sostanza reale della parete addominale. Chirurgia 4:601–606
13. Dayton MT, Buchele BA, Shirazi SS, Hunt LB (1986) Use of an absorbable mesh to repair contaminated abdominal-wall defects. Arch Surg 121:954–960
14. Marchal F, Brunaud L, Sebbag H et al (1999) Treatment of incisional hernias by placement of an intraperitoneal prothesis: a series of 128 patients. Hernia 3:141–147
15. Romeo G, Catania G, Basile F (1994) L'impiego di protesi nel trattamento chirurgico dei laparoceli. Chirurgia 7:704–708
16. Celi D, Batocchio F, Battaglino D et al (2001) La scelta della protesi nella chirurgia dei grandi laparoceli. Osp Ital Chir 7:107–112
17. Stoppa R, Louis D, Verhaeghe P et al (1987) Current surgical treatment of postoperative eventrations. Int Surg 72:42–44
18. Stoppa RE (1989) The treatment of complicated groin and incisional hernias. World J Surg 13:545–554
19. Acquaviva DE, Bourret P (1948) Cure des éventrations par plaques de nylon. Presse Med 73:892
20. Rives J, Pire JC, Flament JB et al (1985) Le traitement des grandes éventrations: nouvelles indications therapeutiques. A propos de 322 cas. Chirurgie 111(3):215–225
21. Chevrel JP (1999) The use of premusculo-facial prothesis in incisional hernia treatment. Atti del Meeting "La chirurgia del laparocele nel XXI secolo". Napoli, 5–6 luglio 1999
22. Gillion JF, Begin GF, Marecos C, Foutanier G (1997) Expanded polytetrafluoroethylene patches used in the intraperitoneal or extraperitoneal position for repair of incisional hernias of the anterolateral abdominal wall. Am J Surg 174(1):16–19
23. LeBlanc KA, Booth WV (1993) Laparoscopic repair of incisional abdominal hernias using expanded polytetrafluoroethylene: preliminary findings. Surg Laparosc Endosc 3:39–41
24. White TJ, Santos MC, Thompson JS (1998) Factors affecting wound complications in repair of ventral hernias. Am Surg 64:276–280
25. Flament JB, Avisse C, Palot JP, Delattre JF (2000) Complications in incisional hernia repairs by the placement of retromuscolar prostheses. Hernia 4 :S25-S29
26. Chevrel JP, Rath AM (2000) Classification of incisional hernias of the abdominal wall. Hernia 4:7–11
27. Langer S, Cristensen J (1985) Long-term results after incisional hernia repair. Acta Chir Scand 151:217–219
28. Read RC, Yoder G (1989) Recent trends in the management of incisional herniation. Am J Surg 124:485–488
29. Chevrel JP, Flament JB (1995) Traitement des éventrations de la paroi abdominale. EMC Paris, Techniques Chirurgicales-Appareil digestif, pp 40-165
30. Wantz GE (1991) Incisional hernioplasty with Mersilene. Surg Gynecol Obstet 172:129–137
31. Flament JB, Avisse C, Palot JP, Delattre JF (1999) Biomaterials. Principles of implantation. In: Schumpelick, Kingsnorth (eds) Incisional hernia. Springer-Verlag, Berlin Heidelberg
32. Luijendijk RW, Hop WCJ, van den Tol MP et al (2000) A comparison of suture repair for incisional hernia. N Engl J Med 343(6):392–398
33. Novitsky YW, Cobb WS, Kercher KW et al (2006) Laparoscopic ventral hernia repair in obese patients. Arch Surg 141:57–61
34. Birgisson G, Park AE, Mastrangelo MJ et al (2001) Obesity and laparoscopic repair of ven-

tral hernias. Surg Endosc 15:1419–1422
35. Carbajo MA, del Olmo JCM, Blanco JI et al (2000) Laparoscopic treatment of ventral abdominal wall hernias: preliminary results in 100 patients. JSLS 4:141–145
36. Carbajo MA, del Olmo JCM, Blanco JI et al (2000) A laparoscopic solution for an old problem: incisional and abdominal wall hernias. Experience with 200 patients. In: 7th World Congress of Endoscopic Surgery. Monduzzi, Bologna, pp 437–440
37. Franklin ME, Dorman JP, Glass JL (1998) Laparoscopic ventral incisional hernia repair. Surg Laparosc Endosc 8:294–299
38. Le Blanc KA (2000) Current considerations in laparoscopic incisional and ventral herniorraphy. JSLS 4:131–139
39. Toy FK, Bairley RW, Carey S (1997) Prospective multicenter study of laparoscopic ventral hernioplasty. Preliminary results. Surg Endosc 12 (7):955–959
40. Heniford BT, Park A, Ramshaw BJ, Voeller G (2003) Laparoscopic repair of ventral hernias. Nine years' experience with 850 consecutive hernias. Ann Surg 238:391–400
41. Berger P, Nijsten MW, Palin JC et al (2001) Abdominal compartment syndrome: a complication with many faces. Neth J Med 58(5):197–203
42. Meldrum DR, Moore FA, Moore EE et al (1997) Prospective characterization and management of the abdominal compartment syndrome. Am J Surg 174(6):667–672
43. Tons C, Schachtrup A, Rau M et al (2000) Abdominal compartment syndrome: prevention and treatment. Chirurg 71(8):918–926
44. Goni Moreno I (1947) Chronic eventrations and large hernias. Preoperative treatment by progressive pneumoperitoneum. Original procedure. Surgery 22:945–953
45. Moreno Egea DA, Torralba Martinez JA, Morales Cuenca G et al (2004) Mortality following laparoscopic ventral hernia repair: lessons from 90 consecutive cases bibliographical analysis. Hernia 8:208–212
46. Cassar K, Munro A (2002) Surgical treatment of incisional hernia. Br J Surg 89:534–545
47. Leber GE, Garb JL, Alexander AI, Reed WP (1998) Long-term complications associated with prosthetic repair of incisional hernias. Arch Surg 133:378–382
48. Burger JWA, Luijendijk RW, Hop WC et al (2004) Long-term follow-up of a Randomized Controlled Trial of Suture Versus Mesh Repair of Incisional Hernia. Ann Surg 240:578–585
49. Le Blanc KA, Whitaker JM (2002) Management of chronic postoperative pain following incisional hernia repair with Composix mesh: a report of two cases. Hernia 6:194–197
50. Amid PK (2004) A new phenomenon causing chronic pain after prosthetic repair of abdominal wall hernias. Arch Surg 139:1297–1298
51. Klinge U (1996) Pathophysiologie der bauchdecken. Chirurgie 67:229
52. Cobb WS, Kercher KW, Heniford BT (2005) Laparoscopic repair of incisional hernias. Surg Clin N Am 85:91–103
53. Rives J, Palot JP, Buerde A, Corcione F (1986) I grandi laparoceli. Esperienza relativa a 250 casi. Minerva Chir 41:135–144
54. Park A, Bird DW, Lourics P (1998) Laparoscopic and open incisional hernia repair: a comparison study. Surgery 124:816–822
55. Nichter LS, Morgan RF, Dufresne CR et al (1983) Rapid management of persistent seromas by sclerotherapy. Ann Plast Surg 11:233–236
56. Tsimoyannis EC, Siakas P, Glantzounis G et al (2001) Seroma in laparoscopic ventral hernioplasty. Surg Laparosc Endosc Percutan Tech 11(5):317–321
57. Chevrel JP, Rath AM (1997) The use of fibrin glues in the surgical treatment of incisional hernias. Hernia 1:14–19
58. Koehler RH, Voeller G (1999) Recurrences in laparoscopic incisional hernia repair: a personal series and review of the literature. JSLS 3:293–304
59. Gilbert AI, Graham MF (1997) Infected grafts in incisional hernioplasties. Hernia 1:77–81
60. Abramov D, Jeroukhimow I, Yinnon AM et al (1996) Antibiotic prophilaxis in umbilical and incisional hernia repair: a prospective randomised study. Eur J Surg 162:945–948
61. Le Blank KA (2004) Laparoscopic incisional and ventral hernia repair: complications – how to avoid and handle. Hernia 8 (4):323–331

62. Amid PK (1997) Classification of biomaterials and their related complications in abdominal wall hernia surgery. Hernia 1:15–21
63. Wantz GE (1999) Incisional hernia: the problem and the cure. Symposium. J Am Coll Surg 188:429–447
64. Martinez DA, Vasquez JL, Pellicer E et al (1999) Results of expanded polytetrafluoroethylene patches in moderate and large incisional hernias. Hernia 3:149–152
65. Anonymous (2001) Linee Guida EAES. Maastricht, June 2001. Surg End
66. Bonjer HJ, Hazerbrock JF, Kazelier G et al (1997) Open versus closed approach establishment of pneumoperitoneum in laparoscopic surgery. Br J Surg 84:599–602
67. Catarci M, Carlini M, Gentileschi P, Santoro E (2001) Major and minor injuries during the creation of pneumoperitoneum. Surg End 15:566–569
68. Carbajo MA, del Olmo MJC, Blanco JI et al (2003) Laparoscopic approach to incisional hernia. Surg Endosc 17:118–122
69. Ramshaw BJ, Escartia P, Schwab J (1999) Comparison of laparoscopic and open ventral herniorraphy. Am Surg 65:827–831
70. Schenk C, Scheuerecker H, Glaser F (2000) Laparoscopic adhesiolysis: results following prospective surveillance. Chirurgie 1:66–71