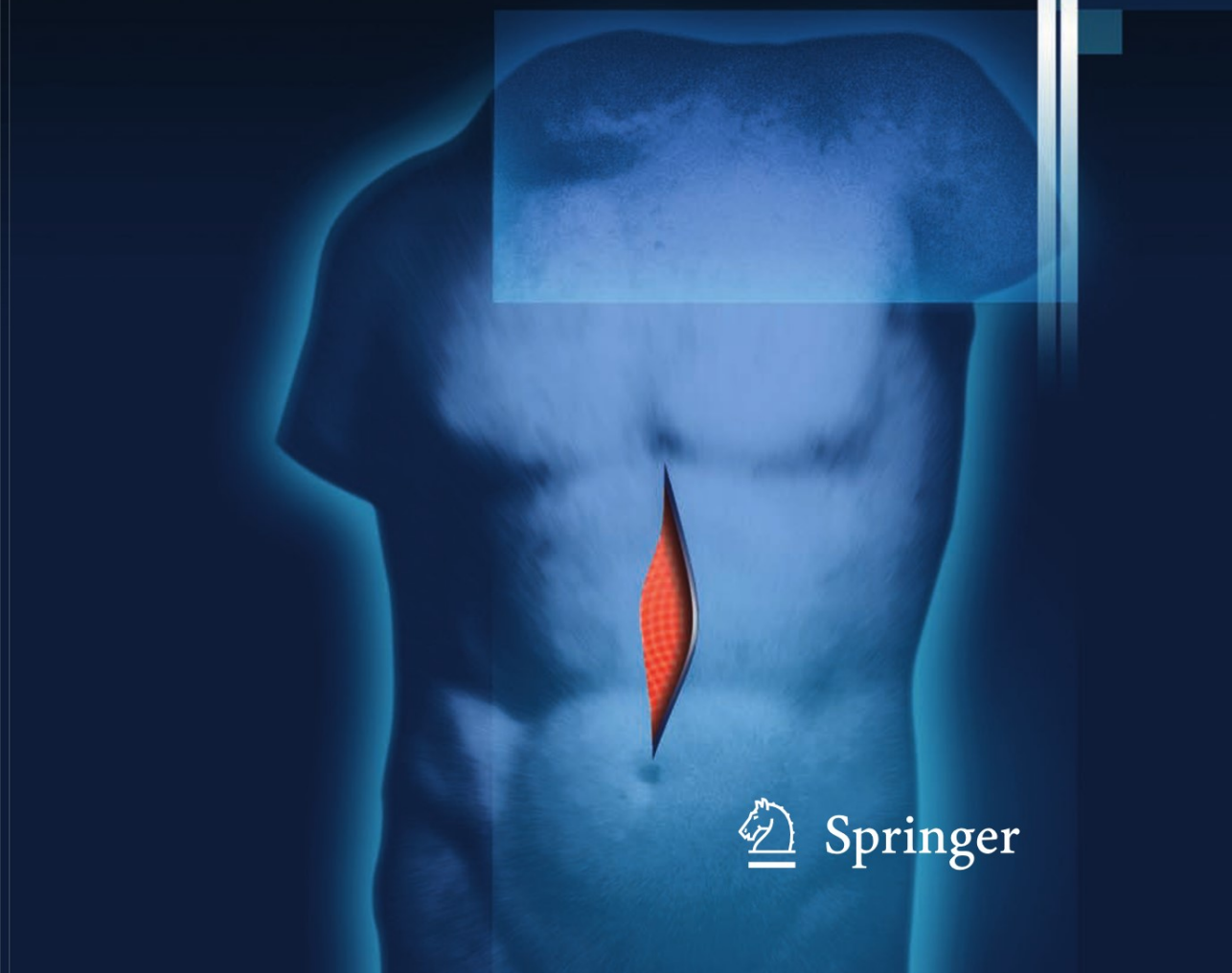
V. SCHUMPELICK
R. J. FITZGIBBONS

Recurrent Hernia

Prevention and Treatment



Springer

Volker Schumpelick
Robert J. Fitzgibbons (Eds.)

Recurrent Hernia
Prevention and Treatment

Volker Schumpelick
Robert J. Fitzgibbons (Eds.)

Recurrent Hernia

Prevention and Treatment

With 144 Figures and 97 Tables

Springer

Prof. Dr. Volker Schumpelick (Ed.)
Chirurgische Klinik
Universitätsklinikum Aachen
Pauwelsstraße 30
52074 Aachen
Germany
e-mail: vschumpelick@ukaachen.de

Prof. Dr. Robert J. Fitzgibbons (Ed.)
Department of Surgery
Creighton University
601 North 30th Street
Suite 3740
Omaha, NE 68131
USA
e-mail: fitzjr@creighton.edu

ISBN 978-3-540-37545-6 Springer Medizin Verlag Heidelberg

Bibliographic information Deutsche Bibliothek
The Deutsche Bibliothek lists this publication in Deutsche Nationalbibliographie; detailed bibliographic data is available in the internet at <http://dnb.ddb.de>.

Springer Medizin Verlag

springer.com

Cover: deblik, Berlin
Typesetting: Hilger VerlagsService, Heidelberg
Printing and Binding: Stürtz AG, Würzburg

Printed on acid-free paper SPIN 11820598 18/5135/BK – 5 4 3 2 1 0

Preface

The field of hernia surgery has changed markedly within the past decade. Today, every patient and every surgeon has the choice between various techniques and devices to repair inguinal, incisional or hiatal hernias. Lots of publications confirm, that most of them can be applied with success. The overall low recurrence rates published make it difficult to decide, which one is the best. Large randomised trials or meta-analysis only provide mean rates to be compared, limited by the heterogeneity of surgeons and patients. In contrast, the many personal series published focus on successful treatment and are characterized by almost absence of any recurrences. However, epidemiological data repeatedly miss the prove of a significant improvement of our results, if regarded on the level of populations. In Germany, despite marked changes of repair techniques and the use of meshes in more than 60% of the patients we still have to face a constant rate of recurrent inguinal hernias of more than 12%. This discrepancy rises questions about the true reproducibility of clinical trials and the cause for recurrence, e.g. improper techniques too difficult to teach, lack of technical skill or biological failure of wound healing?

To compare the good results of various techniques is a traditional, sometimes boring attitude of hernia congresses. The tradition of Suvretta meetings has always been to talk about failures and mistakes in order to learn for the future. After the first meeting in 1995 on "inguinal hernia", the second on "incisional hernia" in 1998 and the third on "meshes" in 2003 this meeting in 2006 on "recurrent hernia" is the fourth in a 11-year-tradition. – The intention of this expert workshop is to elaborate precise recommendations, to help the surgeons to avoid mistakes and to treat recurrences after different types of non-mesh or mesh-repair in inguinal, incisional and hiatal hernia.

V. Schumpelick

List of First Authors

Amid, P. K.
Lichtenstein Hernia Institute
Suite 207
5901 West Olympic Boulevard
Los Angeles, CA 90036
USA
e-mail: pamid@onemain.com

Arlt, G.
Chirurgische Klinik
Park-Klinik Weißensee
Schönstraße 80
13086 Berlin
Germany
e-mail: arlt@park-klinik.com

Bay-Nielsen, E.
Department of Surgical Gastroenterology
Hvidovre Hospital
Kettegaard Alle 30
2650 Hvidovre
Denmark
e-mail: morten.bay.nielsen@hh.hosp.dk

Bellón, J. M.
Department of Morphological Sciences and Surgery
Faculty of Medicine
University of Alcalá
Crta. Madrid-Barcelone Km 33, 500
28871-Alcalá de Henares
Madrid
Spain
e-mail: juanm.bellon@uah.es

Bendavid, R.
614-120 Shelborne Avenue
Toronto, Ontario
M6B2M7
Canada
e-mail: rbendavid@sympatico.ca

Berger, D.
Klinik für Viszeral-, Gefäß- und Kinderchirurgie
Stadtklinik
Balger Straße 50
76532 Baden-Baden
Germany
e-mail: D. Berger@stadtklinik-Baden.de

Bittner, R.
Klinik für Allgemein- und Visceralchirurgie
Marienhospital Stuttgart
Böheimstraße 37
70199 Stuttgart
Germany
e-mail: reinhardbittner@vinzenz.de

Carlson, M. A.
University of Nebraska Medical Center
Surgery 112, VA Medical Center
4101 Woolworth Ave
Omaha, NE 68105
USA
e-mail: macarlso@unmc.edu

Ceydeli, A.
2608 Berkshire Road
Augusta, GA 30909
USA
e-mail: adilc@excite.com

Chan, C.K.
Shouldice Hospital
7750 Bayview Avenue
Thornhill, Ontario L3T 4A3
Canada
e-mail: ggordon@shouldice.com

Chan, K.L.
Division of Paediatric Surgery
Department of Surgery
University of Hong Kong Medical Centre
Queen Mary Hospital
Hong Kong SAR
China
e-mail: klchan@hkucc.hku.hk

Chung, Lucia
University of Glasgow
Department of Surgery
Western Infirmary
Glasgow G11 6NT
United Kingdom

Chowbey, P.
Department of Minimal Access Surgery
Sir Ganga Ram Hospital
Ayushman 13, DS Market
R-Block, New Rajinder Nagar
New Dehli 11006
India
e-mail: chowbey1@vsnl.com

Conze, J.
Chirurgische Klinik
Universitätsklinikum Aachen
Pauwelsstraße 30
52074 Aachen
Germany
e-mail: jconze@ukaachen.de

de Vries Reilingh, T.
Department of Surgery
University Medical Center Nijmegen
PO Box 9101
6500 HB Nijmegen
Netherlands
e-mail: t.deVriesReilingh@chir.umcn.nl

Deysine, M.
S.U.NY. at stony brook
American Hernia Society
2000 N. Village Avenue
Rockville Centre, NY 11570
USA
e-mail: maxdey@optonline.net

Dutta, S.
Department of Surgery
Stanford University
780 Welch Road
Suite 206
Stanford, CA 94305
USA
e-mail: sdutta1@stanford.edu

Elieson, M. J.
Harris Methodist HEB Hospital
1600 Hospital Parkway
Bedford, Tx 76022
USA

Ferzli, G. S.
Department of Surgery
Staten Island University Hospital
65 Cromwell Avenue
Staten Island, NY 10304
USA
e-mail: info@drferzli.com

Fitzgibbons, R. J.
Department of Surgery
Creighton University
601 North 30th Street
Suite 3740
Omaha, NE 68131
USA
e-mail: fitzjr@creighton.edu

Franz, M.G.
Division of Gastrointestinal Surgery
University of Michigan Health System
2922H Taubman Center
1500 East Medical Center Drive
Ann Arbor, Michigan
48109-0331
USA
e-mail: mfranz@umich.edu

Franzén, T.
Department of Surgery
University Hospital
Linkoping 58185
Sweden
e-mail: thomas.franzen@lio.se

Frantzidis, C. T.
Minimally Invasive Surgery
Evanston Northwestern Healthcare
Northwestern University
2650 Ridge Avenue, Burch 106
Evanston, IL 60201
USA
e-mail: cfrantzides@enh.org

Gilbert, A. I.
Hernia Institute of Florida
6250 Sunset Drive 200
Miami, FL 33143
USA
e-mail: Bigart32@aol.com

Haapaniemi, S.
Department of Surgery
Vrinnevi Hospital
SE-60182 Norrköpping
Sweden
e-mail: Staffan.Haapaniemi@lio.se

Halm, J.A.
Laboratorium voor Experimentele Chirurgie
Erasmus MC
Universitair Medisch Centrum Rotterdam
Postbus 2040
3000 CA Rotterdam
The Netherlands
e-mail: j.halm@erasmusmc.nl

Israelsson, L.
Kirurgkliniken
Sundvalls Sjukhus
Sundsvall Hospital
85186 Sundsvall
Sweden
e-mail: leif.israelsson@lvn.se

Itani, K.
Boston University
VA Health Care System (112A)
1400 VFW Parkway
West Roxbury, MA 02132
USA
e-mail: kitani@med.va.gov

Junge, K.
Chirurgische Klinik
Universitätsklinikum Aachen
Pauwelsstraße 30
52074 Aachen
Germany
e-mail: karsten.junge@post.rwth-aachen.de

Kehlet, H.
Juliane Marie Center
Section for Surgical Pathophysiology 4074
Rigshospitalet
Blegdaarmsvej 9
2100 Copenhagen
Denmark
e-mail: Henrik.Kehlet@rh.dk

Kim, B.
VA Medical Center San Francisco
Surgical Service (112)
4150 Clement Street
San Francisco, CA 94121
USA

Kingsnorth, Andrew
Plymouth Postgraduate Medical School
Level 07 Derriford Hospital
Plymouth
Devon PL6 8DH
United Kingdom
e-mail: andrew.kingsnorth@phnt.swest.nhs.uk

Köckerling, F.
Klinikum Hannover-Siloah
Chirurgische Klinik/Zentrum
für Minimal-Invasive Chirurgie
Roesebeckstraße 15
30449 Hannover
Germany
e-mail: ferdinand.koeckerling.siloah@klinikum-hannover.de

Kukleta, J. F.
Klinik Im Park
Seestraße 220
8029 Zürich
Switzerland
e-mail: jfkukleta@bluewin.ch

Kurzer, M.
24 Prothero Gardens
London NW4 3SL
United Kingdom
e-mail: martin@kurzer.co.uk

Lynen-Jansen, Petra
Chirurgische Klinik
Universitätsklinikum Aachen
Pauwelsstraße 30
52074 Aachen
Germany
e-mail: plynen@ukaachen.de

Ma, S.Z.
Beijing ChaoYang Hospital
Capital Medical University
Cell: 13901291518
Beijing 100020
China
e-mail: masongzhang2004@yahoo.com.cn

Machairas, A.
3rd Department of Surgery
University of Athens
Faculty of Medicine
Attikon University Hospital
Rimini 1
12462 Haidari
Athens
Greece
e-mail: anmach@med.uoa.gr

Mertens, P.
Medizinische Klinik II
Universitätsklinikum Aachen
Pauwelsstraße 30
52074 Aachen
Germany
e-mail: pmertens@ukaachen.de

Miserez, M.
Department of Abdominal Surgery
University Hospitals Leuven
Herestraat 49
3000 Leuven
Belgium
e-mail: marc.miserez@uz.kuleuven.ac.be

Morales-Conde, S.
University Hospital Virgen Macarena
Avda Dr. Fedriani sn
41009 Sevilla
Spain
e-mail: smoralesc@mixmail.com

Muschaweck, Ulrike
Arabella-Klinik
Arabellastraße 5
81925 München
Germany
e-mail: um@hernien.de

Nixon, S.
The Royal Infirmary of Edinburgh at Little France
26 Mayfield Gardens
Edinburgh, EH9 2BZ
United Kingdom
e-mail: stephen.nixon@ed.ac.uk

Nordin, P.
Department of Surgery
Östersund Hospital
831 83 Östersund
Sweden
e-mail: par.nordin@jll.se

Peiper, C.
Evangelisches Krankenhaus Witten
Pferdebachstraße 27
58455 Witten
Germany
e-mail: ch.peiper@dwr.de

Pettinari, D.
Department of Surgical Sciences – Pad. Beretta Est
Ospedale Maggiore Policlinico, Mangiagalli
and Regina Elena
Foundation I.R.C.C.S. Public Nature
University of Milan
Italia
e-mail: renato.pietroletti@cc.univaq.it

Pointner, R.
Department of General Surgery
and Division of Clinical Psychology
Hospital Zell am See
5700 Zell am See
Austria
e-mail: Rudolph.pointner@kh-zellamsee.at

Ramshaw, B.
Emory University
1364 Clifton Road NE
Suite H-124
Atlanta, GA 30322
USA
e-mail: ramshawb@health.missouri.edu

Read, R. C.
304 Potomac Street
Rockville, MD 20850
USA
e-mail: read@post.harvard.edu

Rosch, R.
Chirurgische Klinik
Universitätsklinikum Aachen
Pauwelsstraße 30
52074 Aachen
Germany
e-mail: r.rosch@chir.rwth-aachen.de

Sarr, M. G.
Department of Surgery
Mayo Clinic and Mayo Foundation
Rochester, MN 55902
USA
e-mail: Sarr.michael@mayo.edu

Schippers, E.
Allgemein- und Viszeralchirurgie
Juliusspital
Juliuspromenade 19
97070 Würzburg
Germany
e-mail: e.schippers@juliusspital.de

Schumpelick, V.r
Chirurgische Klinik
Universitätsklinikum Aachen
Pauwelsstraße 30
52074 Aachen
Germany
e-mail: vschumpelick@ukaachen.de

Schwab, R.
Department of General Surgery
Central Military Hospital
Rübenacher Straße 170
56072 Koblenz
Germany
e-mail: Robert.schwab@web.de

Simons, M.
Onze Lieve Vrouwe Gasthuis
Postbus 95500
1090 HM Amsterdam
The Netherlands
e-mail: mpsimons@worldonline.nl

Sorensen, L. T.
Department of Surgery
Bispebjerg Hospital
Bakke 23
2400 København
Denmark
e-mail: lts@dadlnet.dk

Stumpf, M.
Chirurgische Klinik
Universitätsklinikum Aachen
Pauwelsstraße 30
52074 Aachen
Germany
e-mail: m.stumpf@chir.rwth-aachen.de

Targarona, E. M.
Service of Surgery
Hospital de Sant Pau
Autonomous University of Barcelona
08025 Barcelona
Spain
e-mail: etargarona@santpau.es

Verhaeghe, P.
Service de Chirurgie generale et Digestive
CHU Amiens Nord
80054 Amiens cedex 01
France
e-mail: verhaeghe.pierre@chu-amiens.fr

Van Geffen, E.
Department of Surgery,
Jeroen Bosch Hospital (GZG)
Nieuwstraat 34,
5211 s-Hertogenbosch,
The Netherlands
e-mail: e.v.geffen@jbz.nl

Contents

V Abdominal Wall Closure

VI Incisional Hernia

VII How to Treat the Recurrent Incisional Hernia

VIII Primary Inguinal Hernia

IX Treatment of Recurrent Inguinal Hernia

X Treatment of the Other Hernia

XI What Can We Do to Improve Our Results?

XII Concluding Recommendations to Prevent the Recurrence

XIII Appendix

Recurrence as an Important Endpoint

1 Present State of Failure Rates (Clinical Studies and Epidemiological Database, Short- and Long-Term)

1.1 Inguinal Hernia

S. Haapaniemi, P. Nordin

Introduction

Hernia treatment has been a challenge to surgeons for more than 2000 years. Modern hernia surgery started in Italy, more than 100 years ago, with Eduardo Bassini's presentation of a new method of repair. Bassini did not just invent a new method of inguinal hernia repair [1]; one of his major contributions was that he performed adequate audit and follow-up of patients [2]. Notable improvements in herniology after that were the development of the Shouldice technique and the introduction of prosthetic mesh.

Today many methods of repair are used, the majority including reinforcement with various mesh devices. Excellent results have been repeatedly reported from specialized hernia clinics with almost total absence of recurrences [3–5]. However, in general surgical practice, in Sweden and elsewhere, recurrent hernia still is a problem, even though the new techniques have been adopted and the outcome improved. In Sweden, with its 9 million inhabitants, each person has a personal identification number [6]; this, together with the national death register [7, 8] and the positive attitude to medical quality registers [9], makes it possible to study hernia surgery using epidemiological methods.

The aim of this chapter is to try to estimate the present failure rate following surgery for inguinal and femoral hernia by reviewing recent data from the Swedish Hernia Register.

Background to our Epidemiological Data

The Swedish Hernia Register

The Swedish Hernia Register (SHR) [10, 11] was established in 1992 and started as a regional project, including eight hospitals, with prospective registration of all procedures for inguinal and femoral hernia surgery on people 15 years of age and older, the use of Person Numbers making it possible to link re-operations to previous operations performed within the framework of the register. The SHR has expanded each year and is now a truly "national" register with 90 units aligned (2004). Our estimation is that approximately 95% of Swedish groin hernia surgery is prospectively registered today.

Once a surgical clinic is aligned to the voluntary register, a contract outlining responsibilities concerning data collection and delivery is signed by the head of the

clinic. The aligned unit also agrees to participate in an external review (visits from SHR representatives) if the hospital is selected. External review is necessary to keep data validity high, and approximately 10% of aligned units are controlled each year. The SHR has been found to include 98% of eligible operations [12].

The aim with the register is to describe and analyze hernia surgery and to be used as a tool in improvement processes at the hospitals participating [11]. From the beginning, our register was funded by the Federation of County Councils and the National Board of Health and Welfare. Since 2001 all aligned hospitals must pay a small fee (30 SKR or approximately € 3.–) for each repair registered, to cover total costs. Recently, a decision was made to increase insight and make some of the data public on the Internet, making it possible to compare results reported from participating units. Hopefully that will stimulate Swedish hernia surgeons to further improve their results. The results of individual surgeons, however, will be reserved for internal quality audit.

Endpoints and Definitions

The two most important outcome measures following hernia surgery are recurrence rate and chronic postoperative pain. Many variables affecting outcome may be studied in the SHR, such as method of repair, suture material, classification of anatomy and size, type of anaesthesia and postoperative complications [11]. Other quality measures such as days off work (or normal activity) following surgery, costs etc. are not as yet registered in the database, but the register can be used as a tool to identify individuals suitable for such analyses.

The focus here will be on rate of recurrence, an endpoint that is not readily available in the SHR. To be able to calculate the true recurrence rate, follow-up of all patients including a physical examination (for instance 3 years after surgery) is necessary. However, in most general surgical departments it is impossible to perform this on an annual basis because of the resources required [13]. Physical follow-up examination is optional but not mandatory for participation in the SHR.

Instead of the ultimate outcome variable recurrence rate, re-operation for recurrent hernia is used as surrogate endpoint. The definition of re-operation for recurrence is listed below. Re-operation for chronic groin pain (tension-reducing procedure including mesh removal, decompression or ligation of nerves) was added in the protocol as indication for surgery in 1999, but numbers of such procedures registered are still so low that meaningful analyses is not yet possible.

Processing of Data

Every year (usually in May) each surgical clinic aligned to the SHR is sent a report with its results and accumulated national data for comparison. The personal identification numbers on re-operated patients are listed to facilitate retrieval of patient files (which can be used for internal quality work, such as seminars).

Data are processed at the Register Centre once a year after certain control measures have been taken (controls of personal identification number and so-called logic controls are today included in the web-based SHR protocol). Prior to analysis, data are matched with the Swedish Cause of Death Register and dates of death are incorporated into the database [11].

An index hernia repair entered into the database is followed from date of surgery until reported date of re-operation on the operated side or, if there is no re-operation, until the person's death. The cumulative incidence for re-operation at various times after an index repair is the main measure of interest and is estimated by actuarial life table analysis. Relative risk analyses are estimated with the Cox's proportional hazards model[14], first performing univariate analyses for assumed risk variables and then selecting variables with the highest or lowest univariate risks for multivariate analysis. Statistical analyses are performed using the SPSS programme.

Definition on Re-Operation for Recurrence in SHR Protocol

"Any hernia operation in a groin previously operated upon for hernia irrespective of type of hernia at the initial and subsequent procedure". (However, a second operation on an adult patient following a simple hernia sac extirpation in the same groin during childhood is not defined as a recurrent groin hernia repair).

Results

Re-Operation as Surrogate Endpoint

To evaluate recurrence rate and chronic groin pain 3 years after hernia repair and to validate a postal questionnaire with selective physical examination as

Table 1.1. Variables associated with increased risk of re-operation

Indications	Methods of repair
– Recurrent hernia – Absorbable suture material (Vicryl, Dexon) – Direct hernia – Postoperative complication (registered by the operating unit)	– Shouldice – Other open techniques without mesh – Unspecified mesh techniques, inguinal incision – Preperitoneal open techniques with mesh – Plug methods – Laparoscopic methods

a method of follow-up, a prospective cohort study[13] was done at a hospital aligned to the SHR. The study comprised 272 repairs and the follow-up rate was 96% with a median follow-up time of 36 months. We found that the re-operation rate requires to be multiplied by a factor within the range 1.7 to 2.3 (depending on method of follow-up and definition of recurrence [15,16]) to gain the true recurrence rate. A similar conclusion was reached in a previous Swedish study[17].

Risk Factors for Re-Operation

The SHR may be used to identify risk factors for re-operation for recurrent hernia [18–20]. The large numbers of operations registered make it possible to use multivariate statistics, and analyses have been done in close cooperation with a professional statistician connected to the register from the start.

The last annual report from the SHR (available on the Internet in Swedish [21]) includes 107,838 hernia repairs done between January 1, 1992, and December 31, 2004. Variables associated with, statistically significant, increased relative risks for re-operation for recurrence can be found in Table 1.1. In two recent multivariate comparisons of anaesthetic alternatives on SHR data with local anaesthesia as reference, both general anaesthesia and regional anaesthesia were associated with decreased relative risk. Using the Lichtenstein technique as reference, all other methods of repair carried increased relative risk of re-operation.

Operation for Recurrent Hernia

The percentage of repairs done for recurrent hernia may be used as a quality measure (but note that these figures also include surgical mistakes incurred before the start

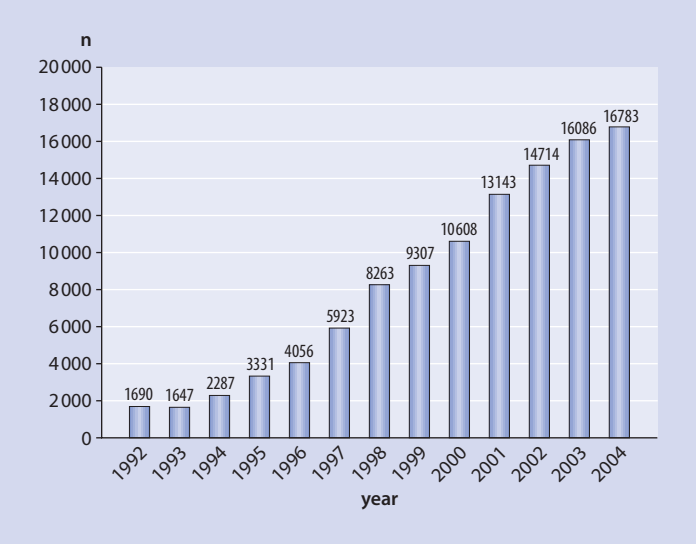


Fig. 1.1. Operations per year in the SHR 1992–2004

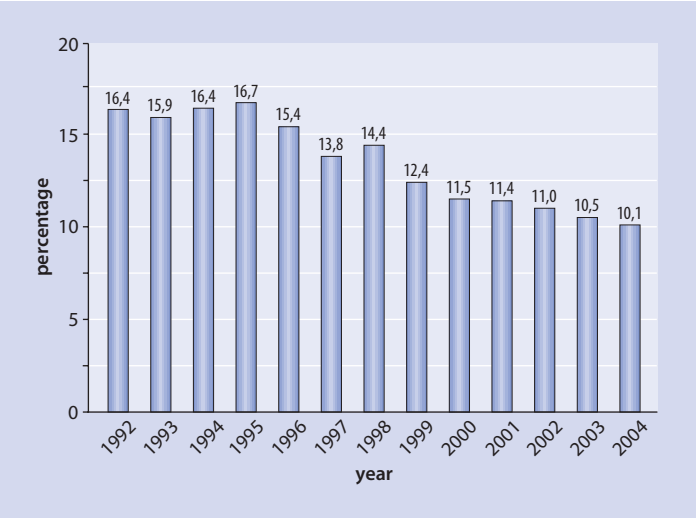


Fig. 1.2. Re-operated hernias 1992 to 2004

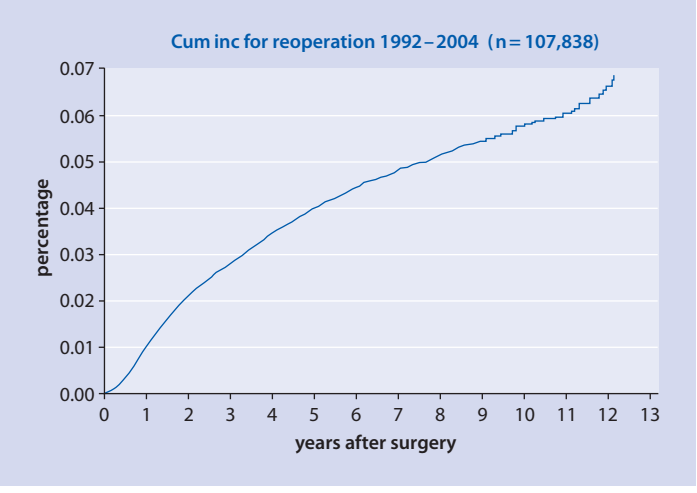


Fig. 1.3. Cumulative incidence for re-operation 1992–2004 (n = 107,838)

of the SHR). Figure 1.1 illustrates the growing numbers of hernia repairs included in the database; in 2004 a total of 16,090 repairs were done at the 90 units aligned. In Fig. 1.2 the change in percentage of repairs done for recurrent hernia during the past 13 years is shown. As can be seen, the improvement has slowed down and has not reached statistical significance every year.

Cumulative Incidence for Re-Operation

The cumulative incidence of re-operation for recurrent hernia is the major outcome measure. In Fig. 1.3 all 107,838 hernia repairs so far registered (both primary and recurrent repairs) are included in the analysis. The cumulative incidence of re-operation 5 years after surgery was approximately 4% with no confidence intervals given in the figure.

Discussion

Over the past 15 years great changes have taken place concerning the methods of repair used in Swedish groin hernia surgery. The Swedish Hernia Register, today comprising more than 120,000 inguinal and femoral hernia repairs, has become an important tool

in the analyses of what we have done, and where there is room for improvement in the future.

Participation in the register is voluntary for the surgical departments aligned but mandatory for individual surgeons working at those units. The register has developed to become nation wide, covering approximately 95% of Swedish groin hernia surgery. It is important to remember that repairs recorded are performed by surgeons at all levels, from specially interested consultants to trainees with various degrees of experience and supervision. The results obtained under such conditions are a measure of "effectiveness" as compared to "efficacy", which reflects "what a method can accomplish in expert hands when correctly applied to an appropriate patient" [22]. However, there are, naturally, limitations in information reached from national epidemiological databases; register studies with multivariate analysis cannot replace randomized trials.

Results from randomized controlled studies are generally considered the highest level of evidence. In order to interpret outcomes after surgical RCTs not only the techniques tested but also inclusion/exclusion criteria, funding and surgical experience [23] have to be considered. We have to keep this in mind when we estimate the external validity of conclusions reached in RCTs. Guidelines for reporting RCTs have been published (CONSORT [24, 25]), but are not always followed. An interesting example of the importance of surgical dexterity in hernia surgery is illustrated by two RCTs published in 1998 with the Bassini repair in one arm; the recurrence rate approximately 3 years after surgery was 2% in one study [26] and 20% in the other [27]. It very clearly helps us to remember that an eponym is not an operation.

Systematic reviews and meta-analyses may increase generalizability (external validity) in findings in RCTs. Meta-analyses [28–31] in the field of hernia surgery undertaken during the past decade bring information with high scientific impact.

Data from the SHR illustrate significant improvements regarding cumulative incidence for re-operations as well as for the percentage of operations done for recurrent hernia since the start in 1992. However, recurrent hernia still constitutes a quantitative problem in our country, approximately 10% of all registered procedures being a repair for a recurrence, the speed of improvement in the last years, regarding the percentage of operations for recurrent hernia, has also decreased. Reports from the Danish Hernia Database [32] and from Germany [33] give similar (or slightly higher) figures.

In a recent Swedish randomized multicentre study by Arvidsson et al. [34] on hernia surgery there was a significant correlation between surgeon's performance score and the recurrence rate. The importance of experienced surgeons in hernia surgery was also recently reported by Neumayer et al. [35] and by Wilkiemayer et al. [36]. Education of surgeons seems to be one important way to further improvement, and with continuing prospective registration we will follow the future outcome.

Acknowledgements. The authors wish to thank all surgeons and secretaries at aligned units for their contribution to the SHR. Special thanks to our Register Statistician Lennart Gustafsson for making the database and the analyses what they are. We also thank our colleague Peter Cox for skilful language correction and the SHR for permission to publish tables and figures based on data previously published in The Annual SHR Report 2004. Financial support for the SHR has been received from the National Board of Health and Welfare and the Federation of County Councils, Sweden.

References

1. Bassini E (1890) Ueber die Behandlung des Leistenbruches. Archiv fur Klinische Chirurgie 40: 429–476
2. Devlin HB, Kingsnorth AN (1998) Management of abdominal hernias. 2nd edn. London: Chapman and Hall Medical.
3. Amid PK, Shulman AG, Lichtenstein IL (1996) Open "tension-free" repair of inguinal hernias: the Lichtenstein technique. Eur J Surg 162(6): 447–453
4. Bendavid R (1997) The Shouldice technique: a canon in hernia repair. Can J Surg 40(3): 199–205, 207
5. Kark AE, Kurzer MN, Belsham PA (1998) Three thousand one hundred seventy-five primary inguinal hernia repairs: advantages of ambulatory open mesh repair using local anesthesia. J Am Coll Surg. 186(4): 447–455
6. Lunde MN, Lundeborg S, Lettenstrom GS, et al (1980) The person-number systems of Sweden, Norway, Denmark and Israel. Vital Health Stat 2: 1–59
7. Statistics Sweden (2004) Statistic yearbook of Sweden. ISBN 91–618–0740–0
8. Statistics Sweden (http://www.scb.se)
9. The Federation of Swedish County Councils and the National Board of Health and Welfare (2000). National Health Care Quality Registries in Sweden 1999. Stockholm: The Federation of Swedish County Councils and Ordförrådet AB
10. Nilsson E, Haapaniemi S (1998) Hernia registers and specialization. Surg Clin North Am. 78(6): 1141–1155, ix
11. Haapaniemi S (2001) Quality assessment in groin hernia surgery – the role of a register. Linköping University, Sweden, Medical dissertation 695
12. Nilsson E, Haapaniemi S, Gruber G, Sandblom G (1998) Methods of repair and risk for reoperation in Swedish hernia surgery from 1992 to 1996. Br J Surg 85(12): 1686–1691

13. Haapaniemi S, Nilsson E (2002) Recurrence and pain three years after groin hernia repair. Validation of postal questionnaire and selective physical examination as a method of follow-up. Eur J Surg 168(1): 22–28
14. Cox D (1972) Regression models and life tables. J R Stat Soc 208: 187–220
15. Marsden AJ (1959) The results of inguinal hernia repairs: a problem of assessment. Lancet i: 461–462
16. Shuttleworth KED, Davies WH (1960) Treatment of inguinal herniae. Lancet i: 126–127
17. Kald A, Nilsson E, Bragmark M, et al. (1998) Reoperation as surrogate endpoint in hernia surgery. A three-year follow-up of 1565 herniorrhaphies. Eur J Surg 164(1): 45–50
18. Haapaniemi S, Gunnarsson U, Nordin P, Nilsson E (2001) Reoperation after recurrent groin hernia repair. Ann Surg 234(1): 122–126
19. Koch A, Edwards A, Haapaniemi S, Nordin P, Kald A (2005) Prospective evaluation of 6895 groin hernia repairs in women. Br J Surg. 92 (12):1553–1558
20. Nordin P, Haapaniemi S, van der Linden W, Nilsson E (2004) Choice of anaesthesia and risk of reoperation for recurrence in groin hernia repair. Ann Surg 240 (1): 187–192
21. Swedish Hernia Register (https://sbr.norrnod.se)
22. Institute of medicine (1985) Assessing medical technologies. National Academy Press, Washington 1985, pp 71–71
23. Van der Linden W (1980) Pitfalls in randomized surgical trials. Surgery 87: 258–262
24. Begg C, Cho M, Eastwood S, et al (1996) Improving the quality of reporting of randomized controlled trials. The CONSORT statement. JAMA 276: 637–639
25. Pollock AV (1998) Guidelines for the correct conduct of clinical research in surgery. Eur J Surg 164: 243–249
26. Strand L (1998) Randomised study of three types of repair used for 324 consecutive operations of inguinal hernia. In Danish with English summary. Ugeskr Laeger 160: 1010–1013
27. Dirksen CD, Beets GL Go PMNYH, et al (1998) Bassini repair compared with laparoscopic repair for primary inguinal hernia: a randomised controlled trial. Eur J Surg 164: 439–447
28. EU Hernia Trialists Collaboration (2000) Laparoscopic compared with open methods of groin hernia repair: systematic review of randomised controlled trials. Br J Surg 87: 860–867
29. EU Hernia Trialists Collaboration (2000) Mesh compared with non-mesh methods of open groin hernia repair: systematic review of randomized controlled trials. Br J Surg 87: 854–859
30. Simons MP, Kleijnen J, van Geldere D, et al (1996) Role of the Shouldice technique in inguinal hernia repair: a systematic review of controlled trials and a meta-analysis. Br J Surg 83: 734–738
31. Schmedt CG, Sauerland S, Bittner R (2005) Comparison of endoscopic procedures vs Lichtenstein and other open mesh techniques for inguinal hernia repair: a meta-analysis of randomized controlled trials. Surg Endosc 19 (2): 188–199
32. Kehlet H, Bay-Nielsen M (2004) In: Schumpelick V, Nyhus LM (eds) Meshes: Benefits and Risks. Springer, Berlin Heidelberg New York
33. Klinge U (2005) Personal communication (letter)
34. Arvidsson D, Berndsen FH, Larsson LG. et al. (2005) Randomized clinical trial comparing 5-year recurrence rate after laparoscopic versus Shouldice repair of primary inguinal hernia. Br J Surg. 92 (9): 1085–1091
35. Neumayer L, Giobbie-Hurder A, Jonasson O, Fitzgibbons R Jr, Dunlop D, Gibbs J et al (2004) Open mesh versus laparoscopic mesh repair of inguinal hernia. N Engl J Med 350: 1819–1827
36. Wilkiemayer M, Pappas TN, Giobbie-Hurder A, Itani KM, Jonasson O, Neumayer LA (2005) Does resident postgraduate year influence the outcomes of inguinal hernia repair? Ann Surg 241 (6): 882–884

Discussion

Schumpelick: *How do you explain the high rate of recurrences in Lichtenstein repair in female compared to TEP?*

Haapaniemi: *You have to read our full report on that, but one important thing is that there are lots of missed female hernias. We cannot really explain why with this method. I think it was done or created for male patients from the beginning. From our material it looks as if it is not suitable for women.*

Read: *In regard to the excellent results of the Lichtenstein, it seems to me that the Lichtentein operation was done more recently. In other words, it is the modern procedure. Some of your dates from the Shouldice, for instance, would be older, so it seems to me that we as surgeons probably know better than we did 10 years ago. Isn't there a little bias in your data?*

Haapaniemi: *It may be so. There have been great changes and perhaps it is so that it is not the same surgeons today that do the primary hernias that did the hernias 10 years ago. So it's difficult to say.*

Read: *It may be that you should compare some dates for the same year. In other words during the year 2003, that the Lichtenstein was this and the Shouldice was this.*

Haapaniemi: *We have done such an analysis but even if the figures are exactly the same, the pattern isn't the same.*

Read: *Oh yes, I am not denying that that is important.*

Kehlet: *It's an impressive amount of data and in contrast to the randomized trials. We know that the suture repairs should not be done, as you also have shown in your large epidemiological series. So my question is: why does it take so long, it's the same in Denmark, for surgeons to change their method despite the evidence? What is your experience in Sweden? Why do 25% continue to do suture repairs?*

Haapaniemi: *We have tried not to point out and say you have to do this, you have to do that. Our register is more*

a tool to follow what is really happening. But of course we have our annual meetings where Swedish surgeons are represented and we tell them this is the result and they can draw their own conclusions.

Kehlet: *I can just answer that in Denmark this is public. So we have just written to the departments to say that this is on the public website. It's official that if you are doing surgery you should do it according to the evidence. But they still do it.*

Haapaniemi: *In a few weeks from now our results will also be available for every hospital on the Internet. So perhaps that will put some extra pressure on Swedish surgeons as well.*

Jeekel: *The problem is that some techniques keep on having a recurrence and some don't, as we found in our prospective randomized study of Lichtenstein versus Bassini. In our long-term follow-up we found that in the Bassini the recurrence came repeatedly for 10 years, but not with the Lichtenstein. So, what was your mean follow-up and do you have any information about the differences in recurrence rate among the techniques? Where there no recurrence rate after a suitable number of years?*

Haapaniemi: *I think with our data that these are the figures when non-specialists use these techniques. We know this from various randomized studies. You mentioned, for instance, Bassini technique. I saw randomized studies from 1998, the same technique but different studies. In one study you had Bassini with a 2% recurrence rate after 3 years and in the same year another randomised study with the Bassini arm you had 20 or 22% recurrence rate. So it's not the name of the method, it's not the eponym; it's how we do it.*

Jeekel: *But we found no recurrence at all in the course of 10 years after Lichtenstein versus the randomized other arm, where we found recurrence up to 10 years. So, do you have any information that, for example, with the Lichtenstein you don't have any recurrence rate after 1, 2, or 3 years?*

Haapaniemi: *No, I can't answer that question right now. But it seems that it's not so.*

Schumpelick: *But are there different time courses for recurrence in different methods?*

Haapaniemi: *I understand what you mean, but I cannot answer that question now. Perhaps you can come back to this later this week.*

Schumpelick: *Is there any method without recurrence?*

Haapaniemi: *No.*

Schumpelick: *O.k. I think that is the answer.*

Kurzer: *I'd like to endorse what Prof. Kehlet said. It has troubled me for a long time why certain surgeons persist with an operation that the evidence in the literature says is no good. There has been a recent paper from Poland that, with some others, looked at factors that will make surgeons change their practice. Published evidence in the literature doesn't seem to make the ordinary general surgeon change his practice. Fitzgibbons said in his opening remarks, what do I hope to learn from this conference? My feeling is that what we should all learn that it is our duty as surgeons from individual countries to go back to our countries and think about how we are going to educate our colleagues; there is a lot of evidence now that the way we will do it is simply by showing other people, making ourselves available, having workshops. The general surgeons will change their practice if they are shown what to do, if they are shown the evidence of their mistakes. The Swedish databases have shown that when you give surgeons feedback about their mistakes and their errors and their recurrences they will change their practice. I think that this is something we should learn from this conference. It's not enough that we learn how to stop recurrence but we have to learn how to teach our colleagues and as "experts" I think it's our duty to go back to our countries because every person in this room knows that hernias recur because they are not done properly in the main and, as Haapaniemi just said, you can call an operation what you want, you can hear a surgeon say "I do an Lichtenstein" you can go and watch him but I have heard Amid say this: "I watch the people do the operation, they call it a Lichtenstein but it is simply not a Lichtenstein operation". So we have to take on a role as teachers and go back and educate our colleagues in our home countries.*

Schumpelick: *Comment on that?*

Haapaniemi: *No, I do agree. I think it's the way to go, to improve their education.*

Verhaeghe: *Another answer to your question about recurrences after TEP in the female, it is probably the same problem for TEP techniques and GRPVS. I mean that the important point is the parietalization of the cord. On women it's very difficult to perform because the teres uteri ligament is more adhesive to the peritoneum and on the male it's easy to stick, so for women often the prosthesis may not stay in place.*

Chan: *For any surgery people come over to see how we operate, and I have somebody who has been there for 1 week, for example, and I go back to see how he operates and I find he is doing very well after 1 week; he is actually doing the real Shouldice technique.*

Schumpelick: *Dr. Chang, but you are a well-equipped and well-educated Shouldice hospital. You have recurrences of operation done by yourself. Is that so?*

Chang: *Yes!*

Schumpelick: *Me too! There must be more than only technical differences.*

Chang: *Yes, we have around 1% recurrences. For primary hernias it will be a little bit lower; as you can see in my first paper in 1987. We saw the recurrence rate go up the more recurrences you do. But then we learned how to put in another mesh, which is underneath the muscle. We go down to the level of the cooper ligament. I think we did it a little bit better now than at former times. But we are learning, too. We changed our thinking in 1987 when we started to say we can't do all primary hernia with suture.*

1.2 Incisional Hernia

K.M.F. Itani

Introduction

Although the rate of ventral incisional hernia (VIH) is about 4% [1], the reported incidence varies from 0.5 to 11% [2, 3]. Recurrence of the hernia is among the more problematic adverse outcomes following incisional hernia repair [4, 5] with progressively higher rates of recurrence after repeated repairs [5, 6]. Repeat recurrence rates after initial repair has varied between 4 and 54%, regardless of the surgical technique used [7–9]. This variability in recurrence rate is due, at least in part, to methodological factors involved in the design of these studies (e.g., heterogeneous study populations and varying study design, end points, and length of follow-up), technical factors involved in the conduct of the operation (e.g., use of autogenous tissue or prosthetic grafts), and patient-related factors (e.g., characteristics of the hernia and co-existing chronic illnesses [7].

A Population-Based Analysis of Incisional Hernia Repair

In 2003, Flum and colleagues published their findings on a total of 10,822 patients undergoing VIH repair extracted from an administrative database in the state of Washington [10]. Of patients undergoing VIH repair, 12.3% underwent at least one subsequent re-operative VIH repair within the first 5 years after initial repair (23.1% at 13 years follow-up). The 5-year re-operative rate was 23.8% after the first re-operation, 35.3% after the second and 38.7% after the third (◘ Fig. 1.4). The use of synthetic mesh in incisional hernia repairs increased from 34.2% in 1987 to 65.5% in 1999. When controlling for age, sex, comorbidity index of the patient, year of the initial procedure, and hospital descriptors, the hazard for recurrence was 24.1% higher if no mesh was used compared to the hazard if mesh was used (◘ Fig. 1.5). After similar adjustments, no differences were found in the hazard of re-operation based on the era of the operative repair [10].

Several important and definitive conclusions can be drawn from this population-based study.

1. Recurrence is not limited to the first 2–5 years after VIH repair but continues over the course of follow-up.
2. Recurrence after each subsequent repair is higher.
3. The use of a mesh in VIH repair decreases recurrence.
4. The rate of recurrence has not changed in time despite newer technology and material.

Effect of Repair Technique on Recurrence

Conventional Non-Prosthetic Ventral Incisional Hernia Repair

Primary repair of ventral incisional hernia without prosthesis can be divided into simple or complex repairs. Simple repairs include edge approximation, vest over pants repair, advancement procedures, a Darn repair, as well as multiple modifications of the above. Complex repair includes components separation, abdominal wall partitioning, the use of tissue expansion-assisted closure, as well as multiple modifications of the above. A summary of the largest series of primary repairs reported in the literature is presented in ◘ Table 1.2. Recurrence rates have varied from a minimum of 25% to a maximum of 54% with a mean follow-up of 1.1 years to 7 years.

The components separation technique, which was first popularized by Ramirez [18], has a recurrence rate of 2–11% in series of 7–26 patients reported between 1994 and 2001. In a more recent publication by DeVries, the recurrence rate was 32% [19].

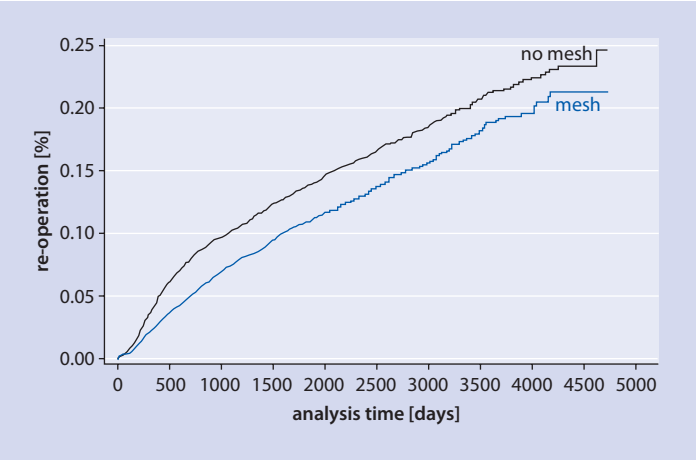


Fig. 1.4. Failure rates after re-operation on a cohort of 10,822 patients in the State of Washington [10]

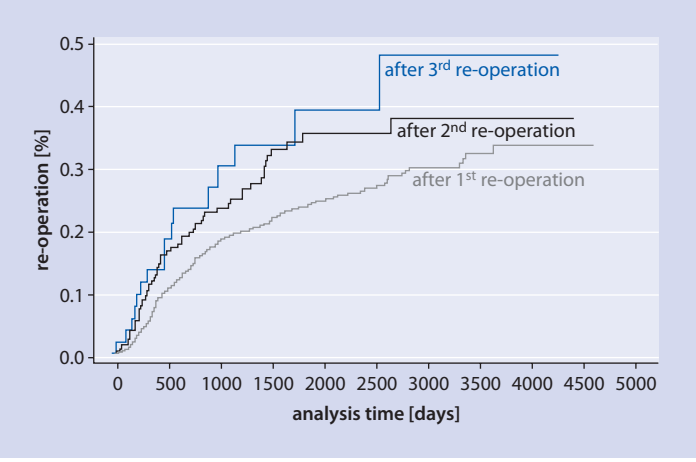


Fig. 1.5. Progression to re-operative repair, by use of mesh in a cohort of 10,822 patients in the State of Washington Patients [10]

Conventional Ventral Hernia Repair with Prosthesis

Three categories of repair have been described in the repair of VIH with prosthesis: direct fascial attachment (simple or Usher techniques), the onlay mesh (Sandwich technique, Chevrel technique), and the sublay mesh popularized by Flament, Rives, and Stoppa. Various modifications and combinations of the above techniques have been described. The recurrence rate after the onlay repair has varied from 5.5–14.8% with a mean follow-up of 1 to 6.7 years (Table 1.3). Various types of prosthetics and repairs are reported in these series. The recurrence rate after the sublay prosthetic technique has varied from 1 to 23% at a mean follow-up of 1.7–6.7 years (Table 1.4).

In a prospective randomized trial of open primary VIH repair vs. repair with sublay mesh, the recurrence rate was 43 and 24% after 3 years, respectively, [17]. The 10 year cumulative rate of recurrence rose to 63% after suture repair and 32% after mesh repair in the same patients [35].

It is clear from the presented data that, irrespective of the technique, the use of mesh to repair VIH reduces recurrence rates in all series by about half.

The sublay mesh technique as described by Flament, Rives, and Stoppa has also been associated with the lowest recurrence rate (5.93%) in the hands of its originator [36]. Although the European Society of Hernia Surgery has adopted the sublay mesh repair as the standard open repair, the complication rate as-

Table 1.2. Recurrence rate with simple repair of ventral incisional hernias

Author, country	Year	No. of patients	Follow-up [years]	Recurrence rate [%]
Langer, Sweden [5]	1985	72	7.0	31
George, U.K. [11]	1986	81	1.1	46
Van der Linden, Netherlands [12]	1988	47	3.3	55
Read, USA [8]	1989	169	5.0	25
Manninen, Finland [13]	1991	57	4.5	34
Hesselink, Netherlands [14]	1993	231	2.9	36
Geçim, Turkey [9]	1996	109	3.6	45
Luijendijk, Netherlands [15]	1997	68	Varying	54
Paul, Germany [16]	1997	111	5.7	53
Anthony, USA [7]	2000	48	3.8	54
Luijendijk, Netherlands [17]	2000	97	2.2	46

Table 1.3. Recurrence rate with onlay prosthetic repair of ventral incisional hernias

Author, country	Year	No. of patients	Prosthesis	Follow-up [years]
Chevrel, France [20]	1986	50	Mersilene/Prolene	1–20
Molloy, USA [21]	1991	50	Marlex	4
Kennedy, USA [22]	1994	40	Goretex	4
Liakakos, Greece [23]	1994	49	Marlex	8
Küng, Switzerl.[24]	1995	47	Marlex	6
Vestweber, Germany [25]	1997	36	Prolene	3
Leber, USA [26]	1998	118	Marlex	6.7

sociated with this type of repair remains high and is associated with a steep learning curve. When originally reported by Stoppa in 1985 on 247 patients, the recurrence rate was 18.5% [37] dropping to 5.93% in 1998 [36].

Laparoscopic Ventral Incisional Hernia Repair

Laparoscopic VIH repair has revolutionized the care of patients with these problems. Laparoscopy is accepted as a more rational technique for repair of a

Table 1.4. Recurrence rate with sublay prosthetic repair of ventral incisional hernias

Author, country	Year	No. of patients	Prosthesis	Follow-up [years]	Recurrence [%]
Adloff, France [27]	1987	130	Mersilene	3	5
Stoppa, France [28]	1989	368	Mersilene	5	15
Amid, USA [29]	1996	75	Marlex	varying	1
Schumpelick, Germany [30]	1996	82	Marlex	5.3	7
Sugerman, USA [33]	1996	98	Marlex	1.7	4
Temudom, USA [34]	1996	50	Prolene	2	4
Leber, USA [26]	1998	82	Marlex Prolene or Mersilene	6.7	20
Feleshtinskii, Ukraine [33]	1999	57	Polyuretan or Marlex	1–5	2
Petersen, Germany [34]	2000	50	Gore-Tex or Prolene	1.5	10
Luijendijk, Netherlands [18]	2000	84	Marlex or Prolene	2.2	23

Table 1.5. Recurrence rate after laparoscopic repair of ventral incisional hernias

Author	Year	No. of patients	Recurrence [%]	Follow-up [months]
Toy [38]	1998	144	4.4	7
Chowbey [39]	2000	202	1.6	35
LeBlanc [40]	2001	100	9.3	23
Berger [41]	2002	150	5.4	28
Henniford [42]	2003	850	4.7	20
Carbajo [43]	2003	270	4.4	44
Rosen [44]	2003	100	17	30

VIH than for repair of an inguinal hernia, because an abdominal procedure and general anaesthesia are requirements for VIH repair whether by an open or a laparoscopic technique, while an inguinal hernia can readily be repaired using local anaesthesia without a laparotomy. The technique of laparoscopic VIH

repair has been standardized with the use of intraperitoneal polytetrafluoroethylene mesh. In addition, the peritoneal sublay method that is used during laparoscopic ventral herniorrhaphy is based on the Stoppa technique for open ventral herniorrhaphy. Some few controversies continue to exist regarding technique such as the extent of mesh overlap and the placement of transabdominal mesh fixation, all of which might affect recurrence. All reports since the introduction of the laparoscopic technique in 1992 consist of retrospective reviews of personal series or prospective collection of data on a cohort of patients undergoing this procedure.

Recurrence rate has varied between 1.6 and 9.3% at 0.6 to 3.6 years mean follow-up (◘ Table 1.5). This will amount to a mean recurrence rate of 4.9% at a mean follow-up of 27 months. In a meta-analysis of eight studies comparing open to laparoscopic repair, no conclusion could be made regarding recurrence due to the short follow-up and lack of standardization [45]. A prospective randomized trial comparing a standard open mesh repair to a standard laparoscopic repair is currently underway in the United States [46].

Other Technical Factors Contributing to Recurrence

Other technical factors within each category of repair have been shown to contribute to recurrence. These include the type of mesh used, type of suture (tacking alone versus tacking and transabdominal suture fixation in the laparoscopic repair), mesh overlap and details of the specific techniques as perfected by its originator and which made it a success in the hands of experts. In addition, one should not ignore the associated learning curve with any procedure; although the learning curve was best described with the laparoscopic technique, it applies as well to the various open techniques.

Each of these issues is mentioned here, but will be the subject of a complete discussion in other chapters.

Patient Risk Factors for Recurrence

Despite the frequency with which incisional hernias complicate the postoperative course of patients undergoing laparotomy, they remain relatively poorly studied. There are only a limited number of studies assessing the impact of various patient-related factors on long-term outcome. In general, previous studies have been retrospective reviews of an institution's experience over a prolonged period of time (10–20 years). The cohort examined is often heterogeneous as patients with ventral hernias at various sites and from a myriad of prior operations are often considered together. Furthermore, the results of repeated repairs are often included with those of the initial attempt, thus confounding the accurate definition of recurrence risk. The impact of various patient-related factors such as chronic illness has received relatively little attention in these previous studies and will be addressed in a more complete discussion in subsequent chapters of this book.

Conclusion

Several conclusions can be made from the above discussion.

1. Mesh repair of VIH is superior to suture repair and will reduce recurrence by half.
2. Repair of recurrent VIH is associated with higher recurrence rates for each subsequent repair.
3. The type of open-mesh repair seems to favour the sublay technique. Other types of repair in the hands of experts can match the sublay repair with similar recurrence rates.
4. The laparoscopic repair of VIH is gaining popularity and is currently under study in a prospective randomized trial.
5. To appropriately assess recurrence after VIH, long follow-up of at least 5 years is required.

References

1. Larson GM, Vandertoll DJ: Approaches to repair of ventral hernia and full thickness losses of the abdominal wall. Surg Clin North Am 64: 335–369, 1984
2. Carlson MA, Ludwig KA, Condon RE: Ventral hernia and other complications of 1000 midline incisions. South Med J 88: 450–453, 1995
3. Khaira HS, Lall P, Hunter B, Brown JH: Repair of incisional hernias. JR Coll Surg Edinb 46: 39–43, 2001
4. Mudge M, Hughes LE: Incisional hernia: a 10 year prospective study of incidence and attitudes. Br J Surg 72: 70–71, 1985
5. Langer S, Christiansen J: Long-term results after incisional hernia repair. Acta Chir Scand 151: 217–219, 1985
6. Costanza MJ, Heniford BT, Arca MJ, et al.: Laparoscopic repair of recurrent ventral hernias. Am Surg 64: 1121–1127, 1998
7. Anthony T, Bergen PC, Kim LT, et al.: Factors affecting recurrence following incisional herniorrhaphy. World J Surg 24: 95–100, 2000
8. Read RC, Yoder G: Recent trends in the management of incisional herniation. Arch Surg 124: 485–488, 1989

9. Gecim IE, Kocak S, Ersoz S, et al.: Recurrence after incisional hernia repair: results and risk factors. Surg Today 26: 607–609, 1996
10. Flum DR, Horvath K, Koeprell T: Have outcomes of incisional hernia repair improved with time? A population-based analysis. Ann Surg 237(1): 129–135, 2003
11. George CD, Ellis H: The results of incisional hernia repair: a twelve year review. Ann R Coll Surg Engl 68(4): 185–187, 1986
12. Van der Linden FT, Van Vroonhaven TJ: Long-term results after surgical correction of incisional hernia. Neth J Surg 40(5): 127–129, 1988
13. Manninen MJ, Lavarius M, Perhoniemi VJ: Results of incisional hernia repair. A retrospective study of 172 unselected hernioplasties. Eur J Surg 157(1): 29–31, 1991
14. Hesselink VJ, Luijendijk RW, de Wilt JH, Heide R, Jeekel J: An evaluation of risk factors in incisional hernia recurrence. Surg Gynocol Obstet 176(3):228–234, 1993
15. Luijendijk RW, Lemmen MH, Hop WC, Woreldsma JC: Incisional hernia recurrence following "vest-over-pants" or vertical Mayo repair of primary hernias of the midline. World J Surg 21(1): 62–65, 1997
16. Paul A, Korenkov M, Peters S, Kohler L, et al.: Unacceptable results of the Mayo procedure for repair of abdominal incisional hernias. Eur J Surg 164(5): 365–367, 1998
17. Luijendijk RW, Hop WC, Van der Tol MP et al.: A comparison of suture repair with mesh repair for incisional hernia. N Engl J Med 343(6): 392–398, 2000
18. Ramirez OM: Abdominal herniorrhaphy. Plast Reconstr Surg 93(3): 660–661, 1994
19. De Vries Reibiugh TS, Van Goor H, Rosman C et al.: Components separation techniques for the repair of large abdominal wall hernias. J Am Coll Surg 196(1): 32–37, 2003
20. Chevrel JP, Dilin C, Marquette H: Treatment of median abdominal hernia by Muscular autograft and pre-musculoaponeurotic prothesis. A propos of 50 cases. Chirurgie 112(9): 616–622, 1986
21. Molloy RG, Moran KT, Waldron RP, et al.: Massive incisional hernia: abdominal wall replacement with marlex mesh. Br J Surg 78(2): 242–244, 1991
22. Kennedy GM, Matyas JA: Use of expanded polytetrafluoroethylene in the repair of the difficult hernia. Am J Surg 168(4): 304–306, 1994
23. Liakakos T, Karonikas I, Panagiotidis H, Dendrinos S: Use of Marlex mesh in the repair of recurrent incisional hernia. Br J Surg 81(2): 248–249, 1994
24. Kung C, Herzog U, Schuppisser JP, Ackermann C, Tondelli P: Abdominal citatricial hernia-results of various surgical techniques. Swiss Surg 6: 274–278, 1995
25. Vestweber KH, Lepique F, Hoof F, Horatz M, Rink A: Meshplasty for recurrent abdominal wall hernias – results. Zentralbl Chir 122(10): 885–888, 1997
26. Leber GE, Garb JL, Alexander AI, Reed WP: Long-term complications associated with prosthetic repair of incisional hernias. Arch Surg 133(4): 378–382, 1998
27. Adloff M, Anaud JP: Surgical management of large incisional hernias by an intraperitoneal mesh and an apauneurotic graft. Surg Gynecol Obstet 165(3): 204–206, 1987
28. Stoppa RE: The treatment of groin and incisional hernias. World J Surg 13(5): 545–554, 1989
29. Amid PK, Lichtenstein IL: Retromuscular alloplasty of large scar hernias: a simple staple attachment technique. Chirurg 67(6): 648–652, 1996
30. Schumpelick V., Conze J, Klinge U: Preperitoneal mesh plasty in incisional hernia repair. A comparative retrospective study of 272 operated incisional hernias. Chirurg 67(10): 1028–1035, 1996
31. Sugerman HJ, Kellum JM Jr, Reines HD, et al.: Greater risk of incisional hernia with morbidly obese than steroid dependant patients and low recurrence with prefascial polypropylene mesh. Am J Surg: 171 (1): 80–84, 1996
32. Termudom T, Siadati M, Sarr MG: Repair of complex giant or recurrent ventral hernias by using tension-free intraparietal prosthetic mesh (Stoppa technique): Lessons learned from our initial experience (fifty patients). Surgery 120(4): 738–743, 1996
33. Felesktinskii IOP: Alloplasty of giant postoperative abdominal hernia in middle aged patients. Klin Khir 7: 24–26, 1999
34. Petersen S, Henke G, Freitag M, et al. Experiences with reconstruction of large Abdominal wall cicatricial hernias using Stoppa–Rives pre-peritoneal mesh plasty. Zentralbl Chir 125(2): 152–156, 2000
35. Burger JW, Luijendijk RW, HopWC, et al. Long term follow-up of a randomized controlled trial of suture versus mesh repair of incisional hernia. Ann Surg 240(4): 578–583, 2004
36. Stoppa R: Long-term complications of prosthetic incisional hernioplasty. Arch Surg 133(1): 1254–1255, 1998
37. Louis D, Stoppa R, Henry X, Verhaegh P: Postoperative eventrations. A propos of 247 surgically treated cases. J Chir (Paris) 122(10): 523–527, 1985
38. Toy FK, Bailey RW, Carey S, et al.: prospective multicenter study of laparoscopic ventral hernioplasty. Preliminary results. Surg Endos 12(7): 955–959, 1998
39. Chowbey PK, Sharma A, Khullar R, et al.: laparoscopic ventral hernia repair. J Laparoendosc Adv Surg Tech A 10(2): 79–84, 2000
40. Le Blanc KA, Booth WV, Whitaker JM, Bellanger DE: Laparoscopic incisional and ventral herniohaphy: our initial 100 patients. Hernia 5(1): 41–45, 2001
41. Berger D, Bientzle M, Muller A: Postoperative complications after laparoscopic incisional hernia repair. Incidence and treatment. Surg Endocs 16(12): 1720–1723, 2002
42. Heniford BT, Park A, Ramashaw BJ, Voeller G: Laparoscopic repair of ventral hernias: nine years experience with 850 consecutive hernias. Ann Surg 238(3): 391–399, 2003
43. Carbajo MA, Martp del Omo JC, Blanco JI, et al.: Laparoscopic approach to incisional hernias. Surg Endosc 17(1):118–122, 2003
44. Rosen M, Brody F, Ponsky J, et al.: Recurrence after laparoscopic ventral hernia repair. Surg Endosc 17(1): 123–128, 2003
45. Goodney PP, Birkmeyer CM, Birkmeyer JD: Short term outcomes of laparoscopic and open ventral hernia repair. Arch Surg 137: 1161–1165, 2002
46. Itani KMF, Neumayer L, Reda D, Kim L, Anthony T: Repair of ventral incisional Hernia: The design of a randomized trail to compare open and laparoscopic surgical techniques. Am J Surg 188(6A): 22S-29S, 2004

1

Discussion

Jeekel: *Consider the Luijendijk study that we published in the New England Journal of medicine 2000, after that a long-term follow-up was published in the annals of surgery and recently in the annals and then you see that the recurrence rate is much higher at 10 years follow-up. So then the mesh result had a 32% recurrence rate and in the primary closure it was 67%. It is amazing, so high. So that means that you need a long-term follow-up as you say, for a good study. So on what should we then agree? Should we say, we no longer trust on data with a follow-up of less then 4, 5 years, or do we, as you may do, extrapolate. What should we do? Another small question is that in the incidence of incisional hernia you see so many differences. I think in the literature you find between 5 and 20% incidence of incisional hernia. In Holland when we calculated a number of years ago it was 15%. Is there a difference in races, in countries, in Caucasians people versus, Chinese or what ever?*

Itani: *These are very good comments and questions. You might know when we planned the inguinal hernia trial with the NDA the budget for this study was six million dollars and for a follow-up of 2 years. So you can imagine what the budget for a study would be for a follow-up of 5 years or even 10 years. So I think it is impossible to go to 5 years without having a budget of millions of dollars. I think the way to do it is to go to population-based studies such as the Flum study in order to understand the progression of the disease. I think that we have enough evidence now to show that 75–80% of the recurrences are going to occur in the first 5 years but that you will continue having recurrences beyond that, as long as we keep that in mind. For your second question regarding races, I don't think it has been studied anywhere in the literature and nobody knows what the exact answer to that is. In any study that we perform, whenever it is a prospective randomized control studies such as the VA study, we take race into consideration but we have a higher proportion of one race over the other so that it would be inappropriate or statistically impossible to reach a good conclusion about race difference.*

Jeekel: *Doing laparoscopic surgery, just one remark: We will close our laparoscopic versus open randomized study in 2 months I think and then we shall have some answers.*

Amid: *In all the reported randomized studies the issue is open versus laparoscopic repair. But what is meant by open? There are many different types of open and there is not only one kind of laparosopic. Do you have any idea?*

Itani: *That´s another very important point, Dr. Amid, and you know those few studies that I've shown you, small studies that have looked at open versus laparoscopic. The VA trial that we've just started standardized the open repair with all details and particular attention was payed to each single issue within the repair in order to come up with an evaluated conclusion about the repair. But as you might know, even if you adopt one repair over the other, you will have proponents of that repair and you will have detractors as well that will tell you should have used a different one because it is better.*

Amid: *So the consensus of the previous meeting in Suvretta was that Rives was superior to the other types of open repair. Would it be possible to get the same consensus in this meeting, because it is very important to see which open repair we have to do?*

Schumpelick: *I think we will come to that topic again, but I would like to comment on that. We have done a prospective randomized study of eight centres in Europe, now published in the British Journal of Surgery, and in three centres we have no recurrence at all, in five centres a large number of recurrences; it is a question of technique. There is no question that the technique is a very important point and you can use different techniques in open approach but there will still be a biological reason we don't understand at the moment; we can talk about this in the coming days.*

Franz: *I agree with your conclusion that the majority of recurrences of primary incisional hernias are probably forming early and, as group of scientific surgeons being scared away from a long-term follow-up that may be required to get better numbers, certainly a physical exam as determent factor of surrogates could be used or radar imaging studies or ultrasound, for example, to detect these defects early. There are recent reports in the literature showing that a gap in the facial closure occurring even in the first month with great accuracy will predict a downstream hernia rate. In your VA trial perhaps you consider surrogate markers for the defects such as ultrasound.*

Itani: *A very good question. If there is any question we do recommend a radiological study such as an ultrasound or CT scan to look more carefully at whether a recurrence is there. We did not adopt surrogate endpoints in our study at the VA. However, I would like to also caution you because you are introducing now a new parameter whereby if your radiologist is not properly trained to detect these small recurrences, they are going to be missed and you will have to standardize among radiologists reading these studies and maybe have one or two radiologists reading all the studies from all the centres in order to come up with a valid surrogate endpoint rather than saying that*

each centre can have one radiologist reading the studies. I don't think you will have a good standardization that way.

Franz: *To the use of the ultrasound, we provide exactly that service at the University of Michigan and when the team is dedicated, it is amazing how accurate they can be with defining what you are going to see in the operating room, but it does require their extreme interest.*

Miserez: *I would like to expand to the previous speaker. We need more standardization. Conferences like this need to work on standardization and especially if you talk about recurrences with the laparoscopic technique we should not forget postoperative bulging and diastases as an important point also to register and to measure because for some this is kind of pseudorecurrence with a lot of complaints for the patients, so we should not forget this.*

Deysine: *I congratulate you, this is progress. There is a problem with standardization. You are talking about VA programs that train first- to fifth-year residents in surgery with different skills. So you are comparing the first year to the fifth, which is totally different. There was an article published showing an improvement from the first to the fifth year in the recurrence rate of inguinal hernias. However the attendants taking care of those residents were the same. So there is a fault in the training program and in the teaching program that permits a first-year resident operating with an attendant to have a very high recurrence rate.*

Itani: *Very, very good point. Actually excellent point. Dr. Fitzgibbons and I were on that publication that looked at PJV level and recurrence rate and your comments are very well taken. We have adopted a much stricter approach with ventral incisional hernia because the operation itself is more complex than inguinal hernia repair and the attending physicians are very involved in that trial and making sure that they are doing the right thing.*

Read: *I would like to make one short comment. I think we should stop calling this operation the Rives or the Rives-Stoppa procedure. Rives did some pioneering work in this area in the early 1970s. Stoppa did further work in the next decade. But this operation is the Flament operation because he has struggled with it for the last 25 years. As Fitzgibbons says, this is the Flament operation. It is the Flament operation and he is with us today and I think he should get all the credit.*

Flament: *I am a very faithful man so I don't want to forget the people who were behind me. As my boss told me, when you work on a heritage, you can take the heritage for yourself but you must not forget the people who succeeded before you. It is Rives-Stoppa.*

1.3 Hiatal Hernia

R. Pointner, F.A. Granderath

In 1951 Philip Allison [1] emphasized the association between esophagitis and hiatal hernia, and hiatal hernia became synonymous with gastro-esophageal reflux disease. Soon thereafter, attention shifted to the lower esophageal sphincter, and investigators related sphincter function to the presence of GERD. It became evident that in patients with hiatal hernia the altered geometry at the cardia could potentially affect lower esophageal sphincter function. Recently, much work has been done to elucidate the effect of the hiatus hernia in the pathophysiology of reflux disease and we are now beginning to understand this complex relationship. A hiatus hernia disrupts the anatomy and physiology of the normal antireflux mechanism. It reduces lower esophageal sphincter length and pressure and impairs the augmenting effects of the diaphragmatic crura. The presence of a hiatus hernia is supposed to be associated with symptoms of gastro-esophageal reflux and increased prevalence and severity of reflux esophagitis, although there are no data available regarding whether patients are more impaired by symptoms corresponding to the insufficiency of lower esophageal-sphincter pressure or hiatal hernia. The fact that esopagitis and reflux were deemed a predictable consequence of hiatus hernia became untenable with the observations that not all patients with hiatus hernias had reflux disease and that not all patients with esophagitis had concomitant hernias and that simple repair of a hiatus hernia did not resolve GERD. Although this fact is well known in only a few papers dealing with recurrences of large hiatal hernias, a differentiation between radiological recurrences and symptom recurrence due to postsurgical anatomical changes or GERD-related problems is worked out.

There is no exact definition of a hiatus hernia, as the "normal" hiatus is well described in regard to its

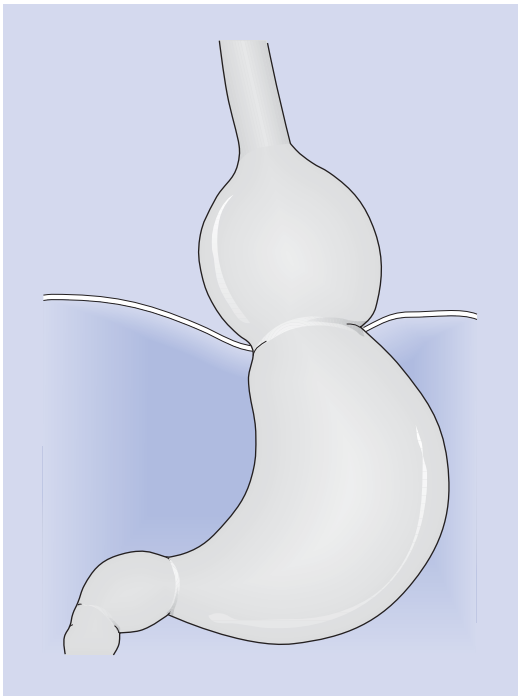


Fig. 1.6. Type-I hiatal hernia

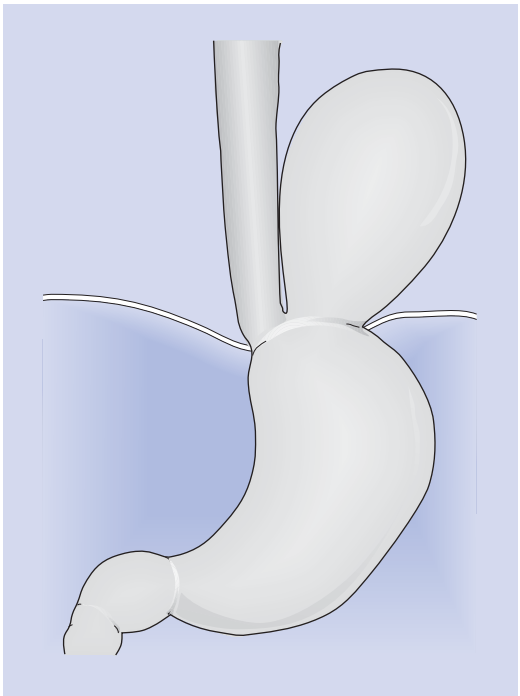


Fig. 1.7. Typ-II hiatal hernia

function but not to its size regarding the anatomy. A hiatus hernia is defined as a proximal displacement of the proximal part of the stomach through the diaphragmatic hernia. There are two different ways to describe a hiatal hernia, the endoscopical and radiological:

- Endoscopically, a hiatal hernia is present when the Z-line can be identified above the crural ring with the folds of gastric mucosa between the crura and the Z-line. The distance between the Z-line and the crura indicates the size of the hiatal hernia. The current practice of diagnosing a hiatus hernia and measuring its size using the centimetre markings on the endoscope is inaccurate. There is no standardization regarding the degree of air insufflation or at which phase of respiration the measurement is made.
- Radiologically the hiatal hernia is specified in three major types:
  - Type I: The sliding hiatus hernia: the gastro-esophageal junction migrates through the hiatus (Fig. 1.6).
  - Type II: The para-esophageal hiatus hernia (PEH): the gastric fundus herniates through the hiatus with the gastro-esophageal junction maintaining its normal intra-abdominal position (Fig. 1.7).
  - Type III: represents a combination of type I and type II: the gastric fundus and gastro-esophageal junction hernia through the hiatus into the thorax (Fig. 1.8).
  - Type IV: this is a type-III hernia with the addition of other organs herniating through the hiatus into the thorax.

The examination technique for diagnosis of hiatal hernias is standardized for neither the endoscopic nor the radiological approach, therefore the size of hiatal hernias depends on different and not standardized examination techniques. There are few published data on the correlation between upper endoscopy and barium studies in the diagnosis of hiatus hernia [2, 3]. Upper GI endoscopy significantly underestimates the size of hiatus hernias compared with barium studies. At present, neither radiology nor endoscopy is an accurate method of measuring hiatus hernia size.

For restoration of normal hiatal anatomy, the knowledge of contents of the hernial sac as well as the distance between Z-line and the diaphragmatic crura is necessary. The most important fact for restoration is the knowledge of the length of the pillars and the width of the maximum distance between the pillars. Measur-

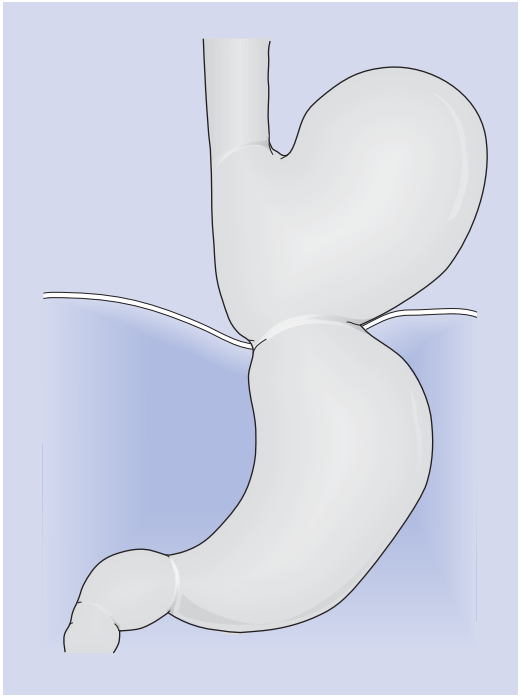


Fig. 1.8. Type-III hiatal hernia

ing these distances, the size of the hiatal surface area (HSA) can be calculated as the only exact parameter for dividing indivduals into patients with normal, small and large hiatal hernias [4].

The precise etiology of large or para-esophageal hernias (PEH) is unknown. The current theory is that large and para-esophageal hernias result from progression of sliding hiatal hernias. Sliding hernias are more common in younger patients and more common than para-esophageal hernias. Increased intra-abdominal pressure, enlargening of the diaphragmatic hiatus and stretching of the phreno-esophageal membrane are key factors in large hiatal hernia formation.

Complications of gastric incarceration or volvulus have been described by Skinner and Belsey [5] with a grade of severe complications in 30% of asymptomatic patients treated conservatively for para-esophageal hernia. Recently, Allen [6], who followed 23 PEH-patients for a medium of 78 months documented a very low incidence, and Stylopoulos [7] created a decision analytical model to determine if asymptomatic patients with large hiatal hernias benefit from elective hiatal repair. For asymptomatic patients, a higher risk for surgery is calculated in the paper of Stylopoulos, and this study adds support to the conservative treatment approach towards asymptomatic PEH.

In 1951 Philip Allison [1] reported very enthusiastically on 33 patients operated over a 5 year period with 30 of them having excellent short-term results. Twenty-two years later, he was courageous enough to report his long-term results and recurrence rates of almost 50% to the American Surgical Association meeting in 1973 [8]. Supported by a grant from the American Surgical Association, he reviewed 421 of his 553 surgically treated patients, of whom 118 were dead and the condition of 14 was unknown. This study of Philip Allison, one of the pioneers of hiatal hernia surgery, is the only one with a nearly complete follow-up of patients in the long-term run for open hiatal surgery. After radiological re-examination of these 421 patients, in cases with presence of a supradiaphragmatical gastric pouch, irrespective of the pouch size, a surgical intervention was indicated for determination of recurrence.

By these rigid standards, radiological recurrence was found in 33% of former para-esophageal hernias and in 49% of former sliding hernias. An important aspect is that recurrences increased steadily with the years after operation.

In the group of patients operated by Allison, there were 27 recurrences in the first year, 28 between 1 and 5 years, 15 between 5 and 10 years and 11 after 10 years. Similar results were found in the group of the other surgeons in this trial. Beneath this high recurrence rate, Philip Allison made clear that a lot of patients were completely free of symptoms but were found to have radiological recurrence, pointing out that there is no correlation between radiological recurrence and symptom recurrence. Over the next decades, no radiologically controlled mid-term or long-term results of hiatal hernia surgery were published, until Hashemi [9] followed 54 patients with type-III hiatal hernias for a medium of 27 months, 27 of them having undergone laparoscopic hiatal hernia repair and 27 open hiatal hernia repair.

The symptomatic outcomes were similar in both groups, with excellent or good outcomes in 76% of the patients of the laparoscopic repair and in 88% after an open repair. A recurrent hernia was present in 12 of the 41 patients (29%) who returned for a follow-up video esophagogram; 42% (9 of 21) of the laparoscopic group had a recurrent hernia compared with 15% (3 of 20) of the open group. Five years later, a similar study was published by Ferri [10], comparing 25 patients with para-esophageal hernia after an open approach with 35 patients after a laparoscopic

hernia repair. No significant difference in general or disease-specific quality of life was documented. Radiographical follow-up was available for 78% open and 91% laparoscopic repairs, showing anatomical recurrence rates of 44% and 23%, respectively. These data are exactly contrary to those published by Hashemi [9] 5 years before. Although the data for the open transabdominally and laparoscopic approaches are contradictory in both papers, the overall recurrence rate in the two studies is exactly the same, 30%! These 30% recurrences were detected also by Jobe [11]. He evaluated the long-term effectiveness of the laparoscopic management of giant type-III hiatal hernia in 52 patients at a mean of more than 3 years. Esophagograms revealed a recurrent hernia in 32% (11 of 34) of patients of whom 36% (4 of 11) were asymptomatic. Of these 11 recurrences, 4 occurred within the first 2 years, 3 between the second and fourth year and 4 between years 4 and 7. According to the increasing rate of recurrences, the rate of patients presenting no adverse symptoms dropped from 91% 3 months postoperatively to 81% 3 years postoperatively. These results were confirmed by Targarona [12] in 2004 in a study of mid-term analysis of safety and quality of life after the laparoscopic repair of para-esophageal hiatal hernia in 46 patients he had operated on. Eight patients (21%) had postoperative gastro-intestinal symptoms in a follow-up of more than 6 months. Barium swallow was performed in 30 patients (81%) and showed a recurrence in 6 of them (20%). However, follow-up of the patients with recurrent hernia was significantly longer than that of the patients without recurrence, suggesting that the risk of recurrence is highly correlated with time. In his study, Targarona pointed out that the quality of life of patients postoperatively reached normal values and did not differ significantly from the standard values for the Spanish population of similar age and with similar comorbidities. Successfully operated patients reached a gastro-intestinal quality-of-life index value comparable to standard population; however, symptomatic patients had significantly lower gastro-intestinal quality-of-life index scores than the asymptomatic or the X-ray-recurrent group.

The main object of Targarona´s study was to assess the incidence of recurrences of hiatal hernia repair and to investigate its correlation with the patients' postoperative quality of life. One interesting finding was that a number of patients with recurrent radiological hernia remained asymptomatic, whereas, as shown also by Jobe [11], increase of adverse symptoms or low quality of life index is not obviously correlated with anatomical recurrence.

Going through the literature of laparoscopically performed hiatal hernia repairs (Table 1.6), there is general agreement that a wrap has to be constructed and should hold the stomach intra-abdominally. Whereas the majority of authors prefer a Nissen fundoplication, about 50% of them anchor the stomach intra-abdominally in addition to the wrap by performing a gastropexy. The incorporation of a fundoplication has gained popularity, since it became evident that most of patients with giant hernias report symptomatic reflux pre-operatively. If, and this should be oblique, 24-h pH monitoring and esophageal manometry is performed on these patients, abnormal reflux and incompetence of the lower esophageal sphincter pressure can be demonstrated in almost all of these patients.

Only regarding the utility of performing a gastropexy is controversy likely to remain. Up to now, there are no randomized trials validating the use of a gastropexy in preventing hiatal hernia recurrences. All published studies (see Table 1.6) have demonstrated that complete sac excision and the reduction of viscera into the abdomen is unalterable, as shown by Edye [19]. In his study patients treated without sac-excision experienced a recurrence rate of 20% versus no recurrence in the sac-resection group. The closure of the hiatus is the most essential step in hernia repair. Assessing the failures and problems of antireflux surgery, it is well known that the majority of complications and failures leading to redo surgery in 80% are related to problems of the hiatal closure [20].

Most authors prefer crural closure with simple non-absorbable sutures posteriorly to the esophagus. Buttressing the hiatal closure, typically with a mesh onlay, is advocated if the crura are not of sufficient girth and adequate suture purchase is not possible. Tension-free hiatal closure using prosthetic material seems superior to simple closure, if the gap between crura is excessive and undue tension is placed on the sutures [21].

By now, it is impossible to compare open and laparoscopic results. For both procedures only a few studies are available which routinely include esophagograms to identify asymptomatic recurrences. Based on the only available long-term investigation with a nearly complete follow-up in X-ray documentation, one must conclude that for the open approach recurrence-rates have been increasingly high [8]. For the laparoscopic approach the follow-up time is too short to compare these studies with the long-term study of Philip Allison. Nevertheless, anatomical recurrence rates vary between 15 and 43% (Table 1.7) with a clear

Table 1.6. Laparoscopic hiatal hernia repair

Author	No. (conversion)	WRAP			Gastropexie	Collis	Sac excision	Crural closure		
		Nissen	Toupet	Hill				Sutures	Pledgets	Mesh
Perkidis [13]	53 (2)	52	1	–	24/53 (45%)	–	Yes	Post.	–	–
Mattar [14]	136 (3)	136	–	–	–	6 (5%)	Yes	Post.	136	–
Jobe [11]	52 (0)	–	–	52	–	–	Yes	Post.	>4 cm	–
Khaitan [15]	31 (6)	19	6	–	13/25 (52%)	–	Yes	Post.	15	–
Diaz [16]	119 (3)	108	6	–	48/116 (41%)	6 (5%)	Yes	Post.	116	6 (5%)
Andujar [17]	166 (2)	127	23	–	14/166 (8%)	1	Yes	Post.	–	–
Smith [18]	94 (8)	92	-	–	92/94 (98%)	6 (6%)	Yes	Post. Prae.	–	–

Table 1.7. Recurrence rates after laparoscopic hiatal hernia repair

Author	No.	Follow-up [months]	X-ray (% of N)	Recurrences	Redo	Satisfaction (exc./good)
Perkidis [13]	53	18 (2–54)	46/53 (87%)	7/46 (15%)	0	49/53 (92%)
Mattar [14]	136	40 (12–82)	32/125 (25%)	14/32 (43%)	1	25/28 (90%)
Jobe [11]	52	37 (2–84)	34/52 (65%)	11/34 (32%)	2 (+4)	32/37 (86%)
Khaitan [15]	25	25	15/25 (60%)	6/15 (40%)	0	Not done
Diaz [16]	116	8 (6–12)	66/96 (69%)	21/66 (32%)	3 (2,6%)	Not done
Andujar [17]	166	15	120/166 (72%)	34/120 (28%)	10 (8,3%)	Not done
Smith [18]	94	27 (3–93)	47/94 (50%)	11/47* (23%) 10/86 (12%)	10 (12%)	*= asympt. *=sympt. (before X-ray)

sign that recurrence rates increase with time. These high recurrence rates for the open as well as for the laparoscopic approach necessitate further consideration to ameliorate the results of hiatal hernia repair. One of these new concepts could be the application of meshes at the hiatus [21].

Although the recurrence rate of hiatal hernia repair is extremely high, we know little about the effect of diaphragmatic stressors on recurrent hiatal hernia. Kakarlapudi and Filipi [22] investigated the correlation between the various diaphragmatic stressors and anatomical disruption of the diaphragmatic closure. They conducted a retrospective analysis utilizing a standardized diaphragm stressor questionnaire for the study group and a control group of 50 patients without hiatal hernia recurrence. Only vomiting and weight lifting were significant, using a logistic regression to determine the significant predictors of hiatal hernia recurrence.

Beside these stressors there is discussion about the existence of a so-called short esophagus and whether this entity might influence recurrence rates. There is also discussion, whether decreased adhesion formation due to a wide use of ultrasonic devices can increase the recurrence rates.

Looking at the radiographical features of recurrences, exact descriptions of the new and recurrent pictures are required. Terms like "sliding" hernia or "para-esophageal" hernia in patients with recurrences are incorrect, leading to misinterpretations, and can by no means have influence on the indication for surgery. For recurrences, we need other characteristics, since a patient with a wrap around the distal esophagus can experience neither a „sliding„ nor a "para-esophageal" hernia.

Recapitulating, a high incidence of 30–50% of anatomical recurrences has been demonstrated with routine postoperative radiological studies for both the open and laparoscopic approach. Half of these patients remain asymptomatic, whereas a group of patients of unknown incidence is symptomatic without showing anatomical recurrence.

References

1. Allison PR (1951) Reflux esophagitis, sliding hiatal hernia, and the anatomy of repair. Surg Gynaecol Obstet 92: 419–431
2. Panzuto F, Di Diudio E, Capurso G, et al. (2004) Large hiatal hernia in patients with iron deficiency: a prospective study on prevalence and treatment. Aliment Pharmacol Ther 19: 663–670
3. Sloan S, Rademaker AW, Kahrilas PJ (1992) Determinants of gastroesophageal junction incompetence: hiatal hernia, lower esophageal sphincter, or both? Ann Intern Med 117: 977–982
4. Granderath FA et al. (2006) Laparoscopic antireflux surgery: tailoring the hiatal closure to the size of hiatal surface area. Surg Endosc 21: 542–548
5. Skinner DB, Belsey RH (1976) Surgical management of esophageal reflux and hiatus hernia. Long-term results with 1030 patients. J Thorac Cardiovasc Surg 53(1): 33–54
6. Allen MS, Trastek VF, Deschamps C, et al. (1993) Intrathoracic stomach. Presentation and results of operation. J Thorac Cardiovasc Surg 105(2): 253–258
7. Stylopoulos N, Gazelle GS, Rattner DW (2002) Paraesophageal hernias: operation or observation? Ann Surg 236(4): 492–500
8. Allison PR (1973) Hiatus hernia: A 20-year retrospective surgery. Ann Surg 178(3): 273–276
9. Hashemi M, Peters JH, DeMeester TR, et al. (2000) Laparoscopic repair of large type III hiatal hernia: objective follow-up reveals high recurrence rate. J Am Coll Surg 190(5): 553–560
10. Ferri LE, Feldman LS, Standbridge D, Mayrand S, Stein L, Fried GM (2005) Should laparoscopic paraesophageal hernia repair be abandoned in favor of the open approach? Surg Endosc 19: 4–8
11. Jobe BA, Aye RW, Deveney CW, Domreis JS, Hill LD (2002) Laparoscopic management of giant type III hiatal hernia and short esophagus: objective follow-up at three years. J Gastrointest Surg 6: 181–188
12. Targarone EM, Novell J, Vela S, et al. (2004) Mid term analysis of safety and quality of life after the laparoscopic repair of paraesophageal hiatal hernia. Surg Endosc 18: 1045–1050
13. Perdikis G, Hinder RA, Filipi CJ, Walenz T, McBride PJ, Smith SL, Katada N, Klingler PJ (1997) Laparoscopic paraesophageal hernia repair. Arch Surg 132: 586–590
14. Mattar SG, Bowers SP, Galloway KD, Hunter JG, Smit CD (2002) Long-term outcomes of laparoscopic repair of paraesophageal hernia. Surg Endosc 16: 745–749
15. Khaitan L, Housten H, Sharp K, Holzmann M, Richards W (2002) Laparoscopic paraesophageal hernia repair has an acceptable recurrence rate. Am Surg 68: 546–551
16. Diaz S, Brunt LM, Klingensmith ME, et al. (2003) Laparoscopic paraesophageal hernia repair, a challenging operation: medium-term outcome of 116 patients. J Gastrointest Surg 7: 59–66
17. Andujar JJ, Papasavas PK, Birdas T, Robke J, Raftopoulos Y, Gagne DJ, Caushaj PF, Landreneau RJ, Keenan RJ (2004) Laparoscopic repair of large paraesophageal hernia is associated with a low incidence of recurrence and reoperation. Surg Endosc 18: 444–447
18. Smith GS, Isascson JR, Draganic BD, Baladas HG, Falk GL (2004) Symptomatic and radiological follow-up after paraesophageal hernia repair. Dis Esophagus 17: 279–284.
19. Edye M, Salky B, Posner A, Fierer A (1998) Sac excision is essential to adequate laparoscopic repair of paraesophageal hernia. Surg Endosc 12(10): 1259–1263
20. Granderath FA, Schweiger UM, Kamolz T, Pointner R (2005) Dysphagia after laparoscopic antireflux surgery: a problem of hiatal closure more than a problem of the wrap. Surg Endosc 19: 1439–1446
21. Granderath FA, Carlson MA, Champion JK, Szold A, Basso N, Pointner R, Frantzides CT (2006) Prosthetic closure of the esophageal hiatus in large hiatal hernia repair and laparoscopic antireflux surgery. Surg Endosc 20(3): 367–379
22. Kakarlapudi GV, Awad ZT, Haynatzki G, Sampson T, Stroup G, Filipi CJ (2002) The effect of diaphragmatic stressors on recurrent hiatal hernia. Hernia 6: 163–166

Discussion

Frantzides: *What you point out is exactly what we see in the literature. We saw the high recurrence rates of hiatal hernia repairs. A colleague here said: „What is it, that we have to change with our technique? " You pointed, that there is up to 40% recurrence rate and we are still wondering why but we are doing the same thing: Placing a few stitches on the crura, expecting that this would be the best treatment. Of course this topic is near to my heart. I've been working on this for 20 years and I was very disappointed when I saw that you didn't mention our work, that is the only prospective, randomised study up to now. I've shown that if you use mesh the recurrence rate should be much less. Actually our study,was a 9-years study published in The Annals of Surgery 2000 with a medium follow-up of 3.5 years. We've shown that the use of mesh should result in 0 recurrence of hiatal hernia. I recognize that mesh is something we are very leery to use around the hiatus. There are reports of erosions especially with prolene mesh. With PTFE we haven't seen that. So I would like to hear your comments. In this forum it is evident that we need to change a lot of things. As said by others before we have to send a message out about when you operate: If there are symptoms, if it is para-esophagial or sliding? When is it time to make decision? The placement of the mesh will be discussed in another forum.*

Pointner: *Thank you Dr. Frantzides. I know your work and I'll mention your work in the afternoon. You know we use meshes as you do and I think that meshes should be used in the correction of this region and they are very important but that's the topic for this afternoon.*

Fitzgibbons: *I can ensure you that we see a lot of redoes and we see plenty of erosions of PTFE into the oesophagus after the hiatus was repaired with Gore-Tex. And we think that material in this area is nonsense because we have seen many of them.*

LeBlanc: *I think this is a problem that we see in all the other hernia repairs. There is no standardized technique: Where do we have to place the sutures, what type of knots and which instruments should be used and even which meshes should be used and where should they be placed? So there is no standardization of any of that. We haven't seen any erosion but we certainly have seen a lot of redoes without the use of mesh. So I'm proposing to use the mesh, particularly for the redo, but I think we need to standardize the operation just like all the others. But I guess we will never eliminate recurrences.*

Pointner: *You are right, there is no standardization of the operation and we don't know which technique – but one thing is clear to me: we have a recurrence rate of about 30% for open and laparoscopic procedures and the recurrence rate for patients with meshes is very, very low. We have to talk about which mesh, which shape of mesh, but we see that we have a lower recurrence rate but that's the topic for this afternoon.*

1.4 Results of Unpublished Studies

M.G. Sarr

Introduction

When asked to write this chapter on Results of Unpublished Studies, I thought my task to be very easy and very short (indeed, very, very short!): unpublished studies are unsubstantiated and therefore not peer-reviewed; thus, these "studies" are neither substantiated nor reliable, and thus my report is over! However, many physicians, both the serious and the pedantic, talk of results (often their own) of unpublished trials, so several questions arise. Who does this? What are these studies? Why do these "studies" get discussed? And finally, what are the perils of this non-science? The following discussion represents my thoughts on this topic as it deals with the subject of the management of hernia disease.

Who Does This?

Who would refer to unpublished studies as dictum or truth? Well, we all do, or at least most of us do. We talk of our own experience (usually a flawed surrogate of a "study"), not disingenuously, but rather based on our believed memory, i.e. our experience. Yet how often our memory fails us – we forget much morbidity and even mortality, though we may have suffered

equally with the patient and their family. Indeed, some memories of complications are just too painful – after all, we often remember the good and protect ourselves psychologically from remembering the bad. I tend to believe that many of us practice this invisible and unknowing selective memory, not out of malice or disinformation, but rather because we may believe strongly and honestly in what we do and how we do it; the important lesson is that we acknowledge this potential fallacy and recognize it for what it is, and keep an open mind such that we try to either prove our "experience" to be correct or, equally important, prove it to be wrong, and then change our practice according to evidence-based studies.

Other possibilities, however, also occur. Ego is often blind. "I've done about 300 of these operations." When I hear this type of a boast, I usually divide the number immediately by a factor of two (or greater depending on the presumed "head size" of the boaster, i.e. here the "presumer" is the boaster himself/herself!). This calculation seems especially pertinent when the boaster is discussing (long-term) morbidity and mortality! I have no scientific data to support my impressions and thus I also write without data, but I always question any non-published, self-aggrandizing "personal experience" when delivered with undeserved authority.

Still another possibility is ignorance. "I've never had a recurrent hernia." Well, it might be true that Surgeon A has never had to repair a recurrent hernia, but that does not mean, necessarily, that none of his (the term "his" from now on will be gender-neutral!) herniorrhaphies have recurred. You don't see what you don't look for! Maybe his patients with a recurrence are asymptomatic, maybe they don't want to tell him because of their respect for him or they believe he will be embarrassed, or more likely, they have gone to another surgeon for repair because Surgeon A failed the first time to fix it. Again: "You don't see what you don't look for." Therefore, Surgeon A may be well-meaning and not untruthful, but just ignorant of his results.

What Are These Unpublished Series?

We have all heard about these series: "I've done 300 of these complicated, huge, multiply recurrent hernias." Remember the divide by 2 (or greater) rule! "My infection rate (or recurrence rate) is zero," or "I've never had a wound infection (or a recurrence)." Right! We have all hopefully learned the lesson of recurrent incisional hernia by the long-term studies from The Netherlands and the Washington State Medical database showing not only an (unbelievably but documented!) high recurrence rate but also the relentless, steady increase year-by-year, not just in the first year or two [1–3]. One can argue about personal experience, but an evidence-based approach is dissociated from emotion, no matter how fervent one might be about his "beliefs" – they remain "beliefs" until proven to be facts. While surgeon A is hopefully in the minority of the rest of us evidence-based surgical scientists, nevertheless surgeon A, especially if a well-renowned leader in his university hospital or community, can promulgate quite a bit of disinformation – "tissue repairs of inguinal hernias have low recurrence rates" – try and argue this point with an enlightened, evidence-based surgeon in Denmark [4]!

Why Do These Unpublished Studies Get Discussed?

There are a multitude of reasons that emanate from many of the points raised above. "My repairs are better," or "It can't happen to me." Divine ignorance. Or in the well-meaning but ignorant surgeon – we never looked, or the follow-up is too short, or the patients seek out another surgeon. Remember, hernias don't recur in the operating room (!) and, admittedly, the infection rate of a herniorrhaphy wound is zero as the patient leaves the operating room and will remain so (in the surgeon's mind) until he looks objectively for a wound infection or a recurrence. Finally, while ideally all operative procedures (in our case herniorrhaphy procedures) should be studied in an evidence-based manner, i.e. well-designed class-I data with long-term follow-up preferably by a double-blind, randomized controlled study, such studies are expensive, difficult to design, impossible to have accepted by the local or national community of all potential participating surgeons, and take a lot of time. Because all of our procedures/approaches cannot fully be confirmed by such studies, we need to continue to question our practices continually and not relay on these unpublished studies.

What Are the Perils of Unpublished Studies?

Beware of the phrase, "in my experience!" Remember the problems with anecdotal "experience", e.g. the scare of port-site recurrence (of colon cancer) after laparoscopic colectomy. Similarly, the implications of validating an operation based on too short a follow-up rings so true when one attempts or continues to justify the practice of repairing incisional hernias with

autogenous suture repairs [1, 2]. Another trap we as surgeons also fall into is the belief in "expert testimony", often the expert is our mentor, whom so many of us "worship." Similarly, our often unwavering support and loyalty toward institutional tradition has also too often clouded our judgment; for instance, at my institution, talk of the Mayo repair of umbilical hernias still lingers in some hallways! Progress continues; new operations are designed; techniques change; we need to maintain an open mind (albeit a critical open mind) – witness the fate of our ancestors who said that laparoscopic gallbladder removal will never catch on. Along these lines, however, we also need to remain cognizant of what we don't know, e.g. duodenal ulcer disease and *Helicobacter pylori*; or pre-1990 the lack of a prosthetic material for repair of direct inguinal hernias, or maybe even the avoidance of prosthetic-based repair for any incisional abdominal wall hernia! We need to learn more about the biology of hernia development and repair, thus, the Suvretta Symposium!

How, then, do we approach the future in the field of herniology when class-I evidence is absent? We will be approached (undoubtedly and hopefully) by industry with new devices, new products, new techniques etc! This is good, this is opportunity, and we need to embrace such a partnership! But we need to question animal models, avoid relying on sensationalism or expert testimony and accept case reports and anecdotal "experience" for what they are, i.e. preliminary observations. Moreover, we need to support study of these advances and to compare them to our (documented) gold standards. Change is (often) good, change is (often) an opportunity, but change must be justified or at the very least accepted with a critical eye and with "The Data!"

The Future

While no one can predict the future, many new programs in the healthcare field are reassuring and offer potential optimism. The proliferation of quality-control initiatives, both at the local (hospital-based) and national level, such as proliferation of participation in National Study of Quality Improvement (NSQIP), the voluntary participation in the Danish herniorrhaphy database, the multi-centre trials in Germany, France, the Netherlands, Sweden and finally in the United States through the Veterans Administration (VA) hernia trials – here is the future of an evidence-based practice. We need to partner with industry, foundations, insurance providers, universities, and the government to evaluate best practice in herniorrhaphy; indeed, this may even be the lack of the need for herniorrhaphy, i.e. watchful waiting [5]! And hopefully through meetings like this Suvretta conference, we will be able to educate our peers in the biology of hernias.

References

1. Burger JWA, Luijendijk RW, Hop WCJ, Halm JA, Verdaasdonk EGG, Jeekel J. Long-term follow-up of a randomized controlled trial of suture versus mesh repair of incisional hernia. Ann Surg 2004; 240: 578–585
2. Luijendijk RW, Hop WCJ, van den Tol MP, et al.. A comparison of suture repair with mesh repair for incisional hernia. New Engl J Med 2000; 343: 392–398
3. Flum DR, Horvath K, Koepsell T. Have outcomes of incisional hernia repair improved with time? A population-based analysis. Ann Surg 2003; 237: 129–135
4. Bay-Nielsen M, Kehlet H, Strand L, Malmstrom J, Andersen FH, Wara P, Juul PM, Callesen T. The Danish hernia database – four years' results. Ugeskr Laeger 2004; 166:1894–1898.
5. Fitzgibbons RJ Jr, Giobbie-Hurder A, Gibbs JO, et al. Watchful waiting vs repair of inguinal hernia in minimally symptomatic men: a randomized clinical trial.. JAMA 2006; 295: 328–329

Discussion

Bendavid: *I really enjoyed your paper and it is true that you have touched on a point that we all have experienced – the fact that every organization actually needs a maverick, and unfortunately this can be very difficult. A good story that I have heard also is: Mark Ravitch was being interviewed once and the topic was division of nerves and, as you certainly know, Dr. Amid does triple neurectomies and a lot of us have done neurectomies for the past 20 years, all of them as routine operations. And the answer of Mark Ravitch to the question "What would happen if your resident cut the nerve?" was "You mean my former resident!" Thanks for the good talk.*
Sarr: *Just the topic of vagotomy. "Should we ever do a vagotomy now?" that's hearsay from 20 years ago.*
Schumpelick: *Dr. Sarr, say something about your unpublished opinion: can we always treat a hernia successfully?*
Sarr: *Can we always treat a hernia successfully? I think no. I think some of them are too big. We can operate on them – but do we really help them? The small ones we should be able to fix as long as we are not ignorant in our knowledge (i.e. primary suture repairs); but as we work with a lot of ignorant surgeons, and many of us are ignorant, it is basic practice that we really have to do just that. But I think there are some hernias we can't fix and some we shouldn't fix. Based on no data!*

2 Recurrence as a Problem of the Trainee

M.P. Simons

The evidence concerning results of a trainee (resident) versus an attending surgeon or specialist surgeon is scarce. There are no randomized clinical trials concerning hernia surgery that compare the outcome of trainee versus surgeon or specialist.

For this chapter a Medline search was performed and the experience with new training methods in the OLVG hospital in Amsterdam are described. The results of inguinal hernia surgery over a period of 10 years in a teaching hospital are presented. The general conclusion of the Medline search is that specialists publish the best results, often in retrospective studies with many flaws in the methods. From general practice, most articles indicate that results of hernia surgery are disappointing but it is usually not clear what recurrences are caused by trainees versus those caused by attending surgeons.

The article of Davies [1] published in 1995 describes how (probably in many countries) surgeons were trained in hernia surgery; "see one, do one, teach one" was the strategy. After about eight inguinal hernia repairs as an assistant the resident would perform an average of nine repairs under supervision of a consultant after which he was on his own. We know now that many were trained in performing the wrong technique. Simons [2] showed in a study that in The Netherlands (and probably elsewhere) almost no surgeon performs the technique the way the inventor had originally described it. Corrupted Bassini's and Shouldice operations were the result.

Few articles describe the results of residents performing inguinal hernia surgery. The article of Danielson [3] reports a RCT in which residents had 9/89 recurrences after Shouldice and 0/89 after Lichtenstein, indicating that its not just the training but also the difficulty of the technique that must be taken into account. The long learning curve for endoscopic hernia repair is well documented [4]. Wilkiemeyer [5] (Table 2.1) recently reported that junior residents had significantly more recurrences performing supervised inguinal hernia repair than senior residents but many studies show that the outcome is not different in teaching hospitals.

Table 2.1. Postgraduate years surgical trainees, their recurrence rate and operating times in a study published by Wilkie on the influence of resident experience on results. They should not be. It is the surgeons duty to the patient to make sure that the outcome is comparable. This can only be achieved by good training

	Recurrence [%]	Operating time [min]
PGY 1+2	6,4	76
PGY 3	3,0	79
PGY 4–5	1,1	71
p = 0.01		

Table 2.2. Patient, hernia and surgical characteristics in 2243 patients with 2535 hernias

No. of patients	1994–1998 (n = 578)	1999–2001 (n = 808)	2002–2004 (n = 857)
No hernias	650	906	979
Age [years]	56.0	54.1	55.1
Length of surgery [min. ± SD]	56.7 ± 27.9	56.2 ± 24.1	58.2 ± 21.1
Acute operation [%]	3.3	1.9	3.0
Recurrence total [%]	15.8	10.4[a]	10.6
Recurrence previous repair OLVG [%]	6.6	3.2[a]	2.5
Re-operation neuralgia [%]	0.4	0.5	0.5
Local anaesthetic [%]	3.5	4.8	4.1
Ambulatory care [%]	14.7	57.4[a]	65.2[a]
Length of stay [days ± SD]	4.3 ± 2.4	2.1 ± 1.7[a]	1.9 ± 1.6

[a]Significantly decreased or increased compared to the previous data period ($p \leq 0.05$).

Table 2.3. Techniques used for primary hernia repair from 1994 to 2004

Technique	1994–1998 [%]	1999–2001 [%]	2002–2004 [%]
Non-mesh	307 (56.1)	73 (9.0)[a]	21 (2.4)[a]
Bassini	100 (18.3)	0[a]	0
Shouldice	203 (37.1)	63 (7.8)[a]	17 (1.9)
Other[b]	4 (0.7)	10 (1.2)	4 (0.5)
Prostheses	240 (43.9)	739 (91.0)[a]	854 (97.6)[a]
Lichtenstein	220 (40.2)	634 (78.1)[a]	713 (81.5)
Endoscopic	19 (3.5)	101 (12.4)[a]	141 (16.1)[a]
Other[b]	1 (0.2)	4 (0.5)	0
Total	547	812	875

[a]Significantly decreased or increased compared to the previous data period ($p \leq 0.05$). [b]Hernial sac resection, McVay, [c]Plug and Patch, Wantz, Stoppa

Table 2.4. Skill of operating surgeon in teaching hospital performing inguinal hernia repair

No hernias	1994–1998 [%] (n = 650)	1999–2001 [%] (n = 906)	2002–2004 [%] (n = 979)
Surgeon [%]	57 (8.8)	64 (7.1)	70 (7.1)
Surgeon + resident [%]	127 (19.5)	234 (25.8)[a]	283 (28.9)
Resident + surgeon [%]	301 (46.3)	382 (42.2)	461 (47.1)[a]
Resident [%]	140 (21.5)	115 (24.9)	165 (16.9)[a]
Unknown [%]	25 (3.9)	0	0

[a]Significantly decreased or increased compared to the previous period ($p \leq 0.05$)

Many new training methods have been described and are being developed internationally. In the OLVG hospital in Amsterdam many of these methods have been implemented and are used in the attempt to improve the results of inguinal hernia surgery.

Surgical training starts in the first year with theoretical training and skillslabs. Residents must have knowledge of the Dutch Guidelines [6, 7], learn the anatomy and observe training videos that were developed by Dr. Amid of the Lichtenstein Hernia Institute. Part of this training is in the lab using models and cadavers. There is an internet-based preparation by residents with interactive learning of anatomy of the abdominal wall and a test of their knowledge. In the operation theatre residents are supervised by a dedicated hernia surgeon.Usually around five or six inguinal hernias will be performed by this surgeon with one resident in one single operating day. The resident must first observe the surgeon performing the operation, then show knowledge of the procedure by telling the surgeon how to perform it step by step and after that he or she is supervised for 40–60 inguinal hernias until qualified enough to perform simple primary one-sided inguinal hernia with supervision not at the operating table, but close by if necessary. The method consists of knowing how, showing how, performing unsupervised and teaching how (knows, shows, does, teaches). Bilateral and recurrent hernias are performed by a dedicated hernia surgeon training only one senior resident at a time to perform endoscopic and other techniques. Dilution of expertise is avoided respecting the long learning curve for difficult hernia techniques.

Following this strategy, a study was performed to compare the results of inguinal hernia surgery before implementing the guidelines and during the period of very infrequent supervision (1994–1996) with a period after implementation of the guidelines and the new training techniques (2002–2004).

Between these periods there was a significant increase in the use of mesh and the supervision of residents (Tables 2.2–2.4). The significant decrease in operations for recurrent inguinal hernia is probably due to the changes in strategy.

In a prospective study of 111 patients with primary inguinal hernia operated in the OLVG all with a follow-up by physical examination of 4 years (2000–2005) the recurrence rate of Lichtenstein repair was 1.8%. In 1990–1994, a RCT was performed in the same hospital comparing modified Bassini and modified Shouldice [8] with recurrence rates after 2 years follow-up of respectively 10.7% and 5.6%. It was concluded that changing of technique and better training with more supervision improved the results. In a RCT studying the value of prophylactic antibiotics performed in three non-teaching and one teaching hospital, there were no significant differences measuring recurrences after 4 years and quality of life (Tables 2.5–2.7) [9–12].

The results show an increase in operating time in the teaching hospital but comparable results for recurrence and other complications.

In conclusion although it seems logical that residents perform less than attending surgeons, this is not proven in literature. It could be that general surgeons who do not perform dedicated hernia surgery have results comparable to residents. The fact is that in general

Table 2.5. Patient and hernia characteristics of 254 patients divided between teaching and non teaching hospitals

No. of actual responders	Teaching hospital (n = 111)	Non-teaching hospitals (n = 143)	Total (n = 254)
No. of eligible patients	130	152	282
Age – years (mean ± SD)	57.5 ± 12.3	58.3 ± 11.2	57.9 ± 11.7
Sex – male [%]	104 (93.7)	138 (96.5)	242 (95.3)
Preoperative painful hernia [%] VAS median (25–75% quartiles)	81 (73.0) 31 (0–60)	98 (68.5) 25 (0–50)	179 (70.5) 25 (0–50)
Operations in day surgery [%]	80 (72.1)[a]	29 (20.3)[a]	109 (42.9)
Level of surgical expertise no. [%]			
Certified surgeon Resident with surgeon Unsupervised resident	10 (9.0)[a] 88 (79.3)[a] 13 (11.7)[a]	136 (95.2)[a] 7 (4.8)[a] 0*	146 (57.5) 95 (37.4) 13 (5.1)
Duration of surgery [min]			
Median (25–75% quartiles)	45 (40–60)[b]	28 (24–40)[b]	36 (25–45)

[a]Chi-square test (Fisher's exact test), [b]Mann-Whitney U test

Table 2.6. The short-term postoperative complications and follow-up of 254 patients divided between teaching and non teaching hospitals

	Teaching hospital (n = 111)	Non teaching hospitals (n = 143)	p value
Re-operation no. [%]			
– Postoperative bleeding	0	1 (0.7)	0.56[a]
– Orchidectomie	0	1 (0.7)	0.56[a]
Wound infection, no. (%)	1 (0.9)	2 (1.4)	0.59[a]
Bladder retention – no. (%)	0	2 (1.4)	0.32[a]
Percutaneous drainage of seroma, no. (%)	1 (0.9)	2 (1.4)	0.59[a]
Total of complications with intervention	2 (1.8)	8 (5.6)	0.11[a]
Follow-up 1 week, no. (%)			
– Pain	28 (25.2)	24 (16.8)	0.10
– Swelling	56 (50.5)	83 (58.0)	0.23
– Haematoma	26 (23.4)	29 (20.3)	0.55

Table 2.6. *Continued*

	Teaching hospital (n = 111)	Non-teaching hospitals (n = 143)	p value
Follow-up 2 weeks, no. (%)			
– Pain	10 (9.0)	14 (9.8)	0.83
– Swelling	38 (34.2)	45 (31.5)	0.64
– Haematoma	9 (8.1)	8 (5.6)	0.43
Follow-up three months, no. (%)]			
– Pain	9 (8.1)	6 (4.2)	0.28

[a]Chi-square (Fisher's exact test)

Table 2.7. The long-term (4 years) postoperative complications of 254 patients divided between teaching and non-teaching hospitals

	Teaching hospital (n = 111)	Non-teaching hospitals (n = 143)	p value
Physical examination at outpatient clinic – no. [%]	94 (84.7)	116 (81.1)	0.46
Recurrence, no. (%)	2 (1.8)	2 (1.4)	0.59[a]
Testicular atrophy, no. (%)	0	1 (0.7)	0.56[a]
Mesh wrinkled or palpable cranial stitch, no. (%)	8 (7.2)	11 (7.7)	0.88
Pain score pre-operatively			
– Number with pain, no. (%)	81 (73.0)	98 (68.5)	0.44
– VAS median (25–90% quartiles)	31 (0–75)	25 (0–66)	0.10[b]
Pain score (resting) 4 years post-operatively	21 (18.9)	13 (9.1)	0.02
– Number with pain, no. (%)	0 (0–20)	0 (0–5)	0.04[b]
– VAS median (25–90% quartiles)			
Frequency of pain 4 years post-operatively, no. (%)	82 (73.9)	100 (69.9)	0.49
– Never painful	22 (19.8)	41 (28.7)	0.11
– Sometimes	3 (2.7)	0	0.08[a]
– Frequently	3 (2.7)	2 (1.4)	0.38[a]
– Always			
Some level of pain during, no. (%)			
– Resting	7 (6.3)	6 (4.2)	0.45
– Coughing	10 (9.0)	2 (1.4)	0.01[a]
– Standing up	9 (8.1)	2 (1.4)	0.01[a]
– Weight lifting	18 (16.2)	18 (12.6)	0.41
– Sports activities	16 (14.4)	11 (7.7)	0.08

Table 2.7. *Continued*

	Teaching hospital (n = 111)	Non-teaching hospitals (n = 143)	p value
Pain location on physical exam			
▬ Pain pressing pubic tubercle, no. (%)	45 (40.5)	36 (25.2)	0.01
▬ VAS median (25–90% quartiles)	0 (0–36)	0 (0–15)	0.01[b]
▬ Pain pressing Poupart's ligament, no. (%)	17 (15.3)	22 (15.4)	0.99
▬ VAS median (25–90% quartiles)	0 (0–10)	0 (0–10)	0.66[b]
Physical exam (sensory), no. (%)			
Reduced sensory function:			
▬ On the scar	17 (15.3)	28 (19.6)	0.38
▬ Below the scar	26 (23.4)	10 (7.0)	0.46
Absent sensory function:			
▬ On the scar	1 (0.9)	2 (1.4)	0.59[a]
▬ Below the scar	1 (0.9)	1 (0.7)	0.68[a]
Sensory disturbance associated with nerves, no. (%)			
▬ Ilioinguinal	2 (1.8)	1 (0.7)	0.41[a]
▬ Iliohypogastric	5 (4.5)	2 (1.4)	0.13[a]
▬ Genitofemoral	2 (1.8)	1 (0.7)	0.41[a]
Satisfied with the operation result, no. (%)	100 (90.1)	135 (94.4)	0.19
Wants the operation performed in the same way	104 (93.7)	134 (93.7)	1.00

[a]Chi-square test (Fisher's exact test), [b]Mann-Whitney U test.

practice results are far inferior to results described by specialists although no RCTs have been performed. National data bases from Sweden, Denmark and Scotland show that re-operation rate yearly for recurrent inguinal hernia is still 8–15% indicating that much effort has to be put into training surgeons and residents to use the best technique, the right way respecting the proven learning curves for all techniques. Recurrences are thus never a problem of the trainee. They are a problem of insufficient training by the trainers. All efforts must be put into excellent training and quality control by sound scientific methods.

References

1. Davies BW, Campbell WB. Inguinal hernia repair: see one, do one teach one? Ann R Coll Surg Engl 1995; 77(6 Suppl):299–301
2. Simons MP, Hoitsma HFW, Mullan FJ. Primary inguinal hernia repair in The Netherlands. Eur J Surg 1995; 161: 345–348
3. Danielsson P, Isacson S, Hansen MV. Randomized study of Lichtenstein compared with Shouldice inguinal hernia repair by surgeons in training. Eur J Surg 1999; 165: 49–53
4. Neumayer L, Giobbie-Hurder A, Jonasson O, et al. Open mesh versus laparoscopic mesh repair of inguinal hernia. N Engl J Med 2004; 350: 1819–1827
5. Wilkiemeyer M, Pappas TN, Giobbie-Hurder A, Itani KM, Jonasson O, Neumayer LA Does resident post graduate year influence the outcomes of inguinal hernia repair? Ann Surg 2005; 241: 879–882
6. Lange de DH, Aufenacker ThJ, Roest M, Simmermacher RK, Gouma DJ, Simons MP. Inguinal hernia surgery in The Netherlands: a baseline study before the introduction of the Dutch Guidelines. Hernia 2005; 9: 172–177
7. Simons MP, de Lange D, Beets GL, van Geldere D, Heij HA, Go PM. The 'Inguinal Hernia' guideline of the Association of Surgeons of The Netherlands. Ned Tijdschr Geneeskd 2003; 147: 2111–2117
8. Simons MP, Koelemay MJW, Luitse JSK, Hoitsma HFW, Obertop H. Modified Shouldice versus modified Bassini by residents. A randomised study. Ned Tijdschr Heelkd, jaargang 8, nr. 6, December 1999;189–194

9. Aufenacker TJ, Geldere D van, Mesdag T van, et al. The role of antibiotic prophylaxis in prevention of wound infection after Lichtenstein open mesh repair of primary inguinal hernia. A multi-center double-blind randomized controlled trial. Ann Surg 2004; 240: 955–961
10. Aufenacker TJ, de Lange DH, Burg MD, Kuiken BW, Hensen EF, Schoots IG, Gouma DJ, Simons MP. Hernia surgery changes in the Amsterdam region 1994–2001. Decrease in operations for recurrent hernia. Hernia 2005; 9(1): 46–50
11. Aufenacker TJ, Schmits SP, Gouma DJ, Simons MP. Do guidelines influence results in inguinal hernia treatment? A descriptive study of 2535 hernia repairs in one teaching hospital from 1994–2004. Hernia (submitted)
12. Aufenacker TJ, van Geldere D, Scheijde E, Juttmann JW, Gouma DJ, Simons MP. Complications after Lichtenstein inguinal hernia repair. A comparison of teaching and non teaching hospitals in a prospective study. Hernia (submitted)

Discussion

Amid: *One thing that Maarten forgot to say: Out of the two or three patients that I have operated in Holland there was one recurrence and that is a recurrence rate of 30–50%. What I really want to say the efficacy of Lichtenstein and laparoscopic repair is between 0 and 0,5% recurrence rate. But when the operation goes to the surgeons at large it goes up to 4% with Lichtenstein and 10% with laparoscopy, as the VA study showed. That means really that training and experience are very important. As a part of what I do every year I have to review approximately 500 operative reports for different reasons and having reviewed thousands of operative reports, mainly in the US, I can seriously say that surgeons do not know how to do a hernia operation as you can clearly find out while reading these useless, sketchy operative reports. It is really the job of the educators to educate the young surgeons so we get out of this mess.*

Schumpelick: *A very clear statement.*

Jeekel: *Excellent lecture. But I don't agree with one conclusion, that the resident is inferior. As you said there is one level-three evidence study from last year; one study is no study. You found yourself the same results in teaching hospitals as in non-teaching hospitals. So I don't think there is real evidence at all from one level-three study that residents are inferior. Isn't what you are saying more that – besides technique – volume is so important and we haven't heard very much about volume today yet. But volume – of course the technique principles should be right – but then the volume is so important. So a resident in his first year has less volume, but shouldn't we just focus a little bit more on the question of volume, volume per surgeon. I agree very much with training the trainers and have volume for the trainers and for everybody. We know that volume often gives quality.*

Simons: *I agree completely. What I found out is that residents really enjoy following me for a month doing 1 day a week of hernia surgery and in 1 day six or seven Lichtensteins one after another. So volume can be even better if you combine it with all the other strategies, the techniques that I showed here, and then the repetition of doing it over and over again is, of course, enormously important. This takes a lot of planning, seeing all the challenges that we have getting the residents in to do these operations. I think the problem is that volume will never be available in general practice with large training programs so you have to focus on all these other aspects also. But your point is right and I am going to put it in my next talk because we shouldn't forget it.*

Fitzgibbons: *There is a small trap in what you say, Prof. Jeekel. In the academic centres sometimes we go to the operation which is most teachable and maybe not the best; for example, the Lichtenstein is a very teachable operation where the Shouldice isn't. So most of us like to use the Lichtenstein in a training centre because it is a very easy operation to supervise as opposed to the Shouldice operation. I worry a little about this.*

Deysine: *First of all, Maarten, I have to congratulate you on this wonderful speech. It is very interesting to see how after these conferences a kind of thought develops in people who are involved with hernias and we all come out with just about the same conclusion, that training teaching is the basis of success. I know of whole regions in the area where I live that when the Shouldice procedure was performed the transversal fascia was never opened, which was exactly an old-fashioned Bassini. The problem that we have is not to convince each other of something that we already know. The difficulty we have is now how are you going to convey your thoughts to people so they will start training residents properly in the management of these patients? To the previous speaker I can tell you that whenever I had a recurrence I knew it already in the operative room. I knew I had done something that was not perfect, and my patient recurred.*

Young: *One of the things that we do with PHS repairs which hasn't been mentioned yet is, in fact, you have the opportunity to do an operation that is relatively easy to perform and you have an opportunity to visualize the anatomy while you do that, as one of the difficulties while doing a Shouldice repair, which is very difficult to do once you even find the anatomy or a Lichtenstein repair obviously, you don't get into the posterior space in that situation.*

Ferzli: *I just want to confirm how important training is and the experience in laparoscopic inguinal hernia in the*

United States. Dr. Fitzgibbons asked me to present to the American College 3 years ago how we work at graduating chief residents in the United States over a 10-year period when the laparoscopic inguinal hernia started. Actually, graduating chief residents in the United States graduate with two laparoscopic inguinal hernias on the average exposure, while they graduate with about 50–60 laparoscopic cholecystectomy exposures. It poses a major problem. I looked at the laparoscopic fellowship that I currently presented in the United States, just to share with you: less than 15% of them train in laparoscopic hernia. I enjoyed your talk very much and I think it is fundamental to see how a model can try to get the hernia repair to another level when we are really not training junior residents in the technique.

Simons: *One short comment. Exactly for that reason I have agreed in my hospital that there is only one older resident who helps me with the endoscopic surgery because we only do the recurrences and the bilateral and a few on request, that's about 40 to 50 a year. So only one resident follows me for 2 years and after his term he will have done about 30 or 40 endoscopic repairs, the rest zero. It is just for one person now.*

3 Failures in Hernia Surgery Done by Experts

A.I. Gilbert, M.F. Graham, J. Young

Introduction

As an invitee to Dr. Volker Schumpelick's fourth triennial meeting in St. Moritz (2006), my assignment was to present answers to why expert hernia surgeons don't always have perfect results. Stated another way, why do some repairs done by experts fail? Clearly, this was one of the most difficult topics I have been asked to write about. Research in the printed surgical literature has been less than fruitful. Textbooks and journals mention generally accepted factors related to hernia repair failure, usually as a prologue to the subject of recurrence. These articles and texts do not distinguish causes of failure by experts from non-expert hernia surgeons. Finding limited value from the printed literature for answers to the assigned question, I sought information directly from colleagues who have demonstrated unusual interest, additional experience, or have recognized expertise in herniology.

Methods

My first attempt to gather information regarding the causes of failures by experts was by sending an e-mail request to a specific group of surgeons asking for their opinions (◘ Fig. 3.1). This group (group 1) was comprised of

- senior authors of articles published in the past 3 years in *Hernia, The World Journal of Hernia* and *Abdominal Wall Surgery*, and
- some other recognized hernia experts whose work has contributed to the science.

Specifically, I asked for their opinions of the causes of failure by experts who repair:

- groin hernias,
- primary abdominal wall hernias,
- incisional hernias, and
- hiatus hernias.

The answers rendered by surgeons in group 1 were divided into four hernia-type categories (◘ Tables 3.1 to 3.4). Within each category five different temporal segments were designated (segments 1–5 in Tables 3.1–3.4). Mostly, the answers and terminology used by the responder was recorded verbatim.

My second attempt to gain answers to this question was to send e-mail requests to the invitees to this St. Moritz meeting. To reach them, I used their current e-mail addresses as listed and furnished by the organizing committee. In this request I asked for their personal results with the technique(s) they had used most often. I included our own results as an example of the information I sought.

Request for "Expert" Professional Assistance

As you are a published expert in the field of herniology I am asking for your input to assist me in preparing a presentation that deals with your views of reasons for recurrence of abdominal hernias. I am sending this same survey to many others who have expertise in this field.
My topic is "Recurrence as a problem of the expert". Note that the emphasis is on EXPERT. The essence of the presentation leaves room for reference to open and laparoscopic approaches to groin hernias, other primary abdominal wall hernias, incisional hernias, and hiatus hernias. It requires answers based on your individual experiences and observations.
Please EMAIL REPLY to me what you believe are the two or three main causes for failed repairs of each group by experts in each field.
(Some causes may apply to all four groups):

Groin Hernias: .....
Primary Abdominal Wall Hernias: .....
Incisional Hernias: .....
Hiatus Hernias: .....

Excuse the BCC format of this correspondence. It is the simplest, quickest, and least expensive way to get responses from a large group while keeping your emails private.

Thank you for taking the time to answer this survey.
This will be the only questionnaire I will send.

Arthur I. Gilbert, M.D.

Fig. 3.1. Request for "Expert" Professional Assistance

Results

The initial mailing was to 112 surgeons. I used the e-mail addresses that were noted in each article. Twelve e-mails were returned as undeliverable due to unrecognized addresses. From the 100 e-mails that were not returned as undeliverable I received 46 responses (46%). The causes of hernia repair failures they reported are itemized in Tables 3.1–3.4. I received no response from the other presumed recipients.

The 46 responders (group 1) noted 180 answers listing 46 different causes of groin hernia repair failure, 150 answers listing 27 different causes of primary ventral hernia repair failure, 149 answers listing 31 causes of incisional hernia repair failure, and 132 answers listing 35 causes of hiatus hernia repair failure. Several responders noted many of the same causes. For each type of hernia repair I separated the causes of failure into the same five temporally related categories.

From my second attempt to gain answers from the 62 invitees to the 2006 St. Moritz meeting, I received only 14 responses (22.5%) to my e-mail inquiry (group 2). The majority of surgeons in this group noted many of the same causes noted by surgeons in group 1. The causes of failures noted by group 2 experts are itemized in Table. 3.1. Thirteen of the 14 furnished an overview of their preferred techniques and their personal results. Since anonymity was promised to the responders the details of techniques and personal numeric results furnished by each responder is not included.

Discussion

A true expert expects success, but always looks for his/her own failures. I received information about recurrences from 13 of 14 surgeons in group 2. Despite my specific request for the details of their own failures, only 4 of the 13 furnished information of those causes. Responses from surgeons in group 2 included faults in Lichtenstein, plug, and laparoscopic repairs. These causes were basically the same as furnished by group 1. While group 1 surgeons noted metabolic defects, col-

Table 3.1. Reasons for failure by experts (group 1) – groin hernias

Reason for failure	No.
1. Surgeon's personal preparation	
Poor understanding of anatomy/patho-physiology	7
Poor training in lap hernia repair	7
Surgeon's limited knowledge, experience, skill	6
Poor training in open hernia repair	5
Failure to recognize multiple defects	4
Ignorance of MPO	3
Poor teaching of residents	2
Surgeon's age-related factors	1
Non expert pressured to do LIH vs. lose case	1
2. Patient profile and habits	
Collagen disorders	4
Smoking	3
Obesity	2
Genetic factors	2
Ascites	1
3. Various intra-operative factors	
Inadequate dissection	13
Repair without mesh	10
Inadequate size of mesh	10
Technical mistakes	9
Inadequate overlap of mesh	9
Errant fixation of mesh	7

Table 3.1. *Continued*

Reason for failure	No.
3. Various intra-operative factors	
Plug migration	6
Tension in repair	5
Plug not in pp space for direct hernias	5
Choice of wrong procedure	4
Missed hernia sac	4
Mesh wrongly placed	3
Lichtenstein poor shutter reconstruction	3
No coverage of femoral canal from groin	3
Incision too small	2
Unrecognized lateral hernias	2
Lichtenstein poor overlap at pubis	2
LIH poor closure of keyhole	2
Wrong anesthetic modality	2
Not fully creating pp space for mesh	1
4. Wound problems	
Infection	8
Mesh shrinkage	3
Hematoma	2
Use of absorbable suture material	2
Intestinal obstruction	1
Seroma	1
5. Postoperative events	
Strenuous activity too soon	3

lagen disorders, and patient's biological features as some causes of failed repairs, the group 2 surgeons did not attribute even one failure to poor patient selection or to patients' biological features. All attributed their failed repairs to faulty personal observation or technique.

Table 3.2. Reasons for failure by experts – primary abdominal wall hernias

Reason for failure	No.
1. Surgeon's personal preparation	
Poor understanding of anatomy and physiology	7
Surgeon's limited knowledge, experience, and skill	5
Surgeon underestimating extent of hernia	4
2. Patient profile and habits	
Genetic factors	7
Obesity	4
Collagen disorders	4
Previous contaminated or infected wound	3
Smoking	1
Concurrent diastasis recti	1
3. Various intra-operative factors	
Failure to use mesh	14
Mesh too small	13
Tension on repair	12
Inadequate fixation of mesh	12

Table 3.2. *Continued*

Reason for failure	No.
3. Various intra-operative factors	
Inadequate overlap of mesh	10
Using onlay method of mesh repair	7
Overlooked multiple defects	5
Failure to use component separation tissue repairs	3
Poor exposure	2
Inadequate dissection	2
Wrong anesthetic modality	2
Rapidly absorbing suture material	2
Lap hernia poor alignment of mesh	1
Fascia not strong enough for repair	1
4. Wound problems	
Infection	11
Seroma	3
Hematoma	2
5. Postoperative events	
Resuming forceful activity too soon	2

Regarding ventral hernia repairs, Awad [1] identified certain technical causes of failure. Inlay mesh repairs were associated with higher failure rates compared to onlay, sublay, and sandwich techniques. The lowest rate of failure was in the sandwich technique. Infection, lateral detachment of the mesh, and inadequate fixation of the mesh were shown to be the most common factors related to failed repairs.

At the 2003 St. Moritz meeting of hernia experts, the question was posed to the group, "Do you believe you can always prevent a hernia recurrence by doing the procedure properly?"; 24% of attendees responded they thought they could; 76% did not feel they could. The audience proffered that failures were related to poor technical skill in 83% of failures, to poor teaching in 57% of failures, and to the patient's defective biological features in 28% of cases.

My personal observations of causes of failed groin preperitoneal hernia repairs by experts include the surgeon's failure to sufficiently develop the preperitoneal space (Bogros space) in doing TEP, TAPP, Ugahary, Kugel, or PHS repairs. Other causes in open repairs were related to inadequate mesh size and poor mesh fixation. I personally was responsible for two failed PHS repairs due to my own poor knot tying.

Causes for failed Lichtenstein repairs were detailed by Amid [2], and Read [2, 3]. With the help of ultrasonography I have identified persistent hernia sacs under the onlay mesh of patients who had Lichtenstein tension-free hernioplasties and presented complaining of inter-

Table 3.3. Reasons for failure by experts – incisional hernias

Reason for failure	No.
1. Surgeon's personal preparation	
Surgeon underestimating extent of hernia	11
Poor understanding of anatomy and physiology	3
Surgeon's limited knowledge, experience, and skill	2
2. Patient profile and habits	
Obesity	7
Genetic factors	6
Smoking	3
Collagen disorders	1
Previous contaminated or infected wound	1
Not fully prepared preoperative.	1
3. Various intra-operative factors	
Mesh too small	15
Inadequate fixation of mesh	13
Inadequate overlap of mesh	11
Tension on repair	10
Inadequate exposure	9
Inadequate dissection	8

Table 3.3. *Continued*

Reason for failure	No.
3. Various intra-operative factors	
Overlooked multiple defects	8
Using onlay method of mesh repair	8
Fascia not strong enough for tissue repair	3
Failure to use mesh	3
Fixation failure at iliac crest and/or pubis	2
Lap hernia inadequate lysis of adhesions	2
Inadequate lysis of adhesions open procedure	2
Rapidly absorbing suture material	1
Lap hernia sutures breaking or tearing tissue	1
Bowel injury	1
Failure to use component separation tissue repairs	1
4. Wound problems	
Infection	9
Hematoma	3
Mesh shrinkage	1
5. Postoperative events	
Resuming forceful activity too soon	2
Drains removed too soon	1

mittent postoperative pain. My personal observations from treating failed plug operations include plug migration into the scrotum in two patients, bowel perforation in two patients, and failure to protect the area surrounding the plug resulting in recurrent interstitial hernias through the lateral triangle and femoral hernias.

For incisional and ventral hernias, my observation in failed repairs has been the surgeon's failure to use mesh large enough to get far wide of the original defect(s). Ventral and incisional hernia failures also are closely related to wound complications that lead to infection. Impatience by the surgeon and/or patient dealing with a postoperative seroma has led to infection because of single or multiple wound aspirations that might have been unnecessary if treated expectantly. My own failures following those repairs were related most often to infection. Once infected, the wound has a high chance of herniation. Additional factors leading to failure include consenting to operate too soon on patients with inadequate pulmonary preparation or insufficient weight loss. Such failures represent compromised judgment by the surgeon who lowers basic principles in response to the patient's pleadings, despite increased chance of failure.

Table 3.4. Reasons for failure by experts – hiatus hernia

Reason for failure	No.
1. Surgeon's personal preparation	
Surgeon's limited knowledge, experience, and skill	9
Poor understanding of anatomy and physiology	2
Surgeon underestimating extent of hernia	4
No. of surgeons that don't do this operation	11
2. Patient profile and habits	
Obesity	4
Collagen disorders	3
Poor preoperative evaluation	2
3. Various intra-operative factors	
Inadequate fixation of mesh	13
Failure to use mesh	9
Inadequate dissection	8
Tension on repair	8
Using onlay method of mesh repair	8
Short esophagus	4
Failure to remove hernia sac	3
Inadequate exposure	3
Fascia not strong enough for tissue repair	3
Lap division of the short gastric vessels	3

Table 3.4. *Continued*

Reason for failure	No.
3. Various intra-operative factors	
Not approximating crura	2
Suture tear through	2
Fixation failure at iliac crest and/or pubis	2
Lap hernia inadequate lysis of adhesions	2
Inadequate lysis of adhesions open procedure	2
Mesh too small	1
Rapidly absorbing suture material	1
Lap hernia sutures breaking or tearing tissue	1
Bowel injury	1
4. Wound problems	
Infection	9
Crura too tight	5
Incomplete closure of hiatus	5
Hematoma	3
Not anchoring fundoplasty	2
Mesh shrinkage	1
Slipped Nisson	1
5. Postoperative events	
Vomiting or gagging	4
Resuming forceful activity too soon	2

At the 2005 meeting of the European Hernia Society, Kingsnorth reported that in a plan to improve surgical education he proposed a scheme of teaching hernia repair he refers to as "Surgery by Numbers." In this plan he had identified 42 separate technical steps that have to be learned to properly perform uncomplicated groin hernia repair. Based on pure mathematical probabilities of successful completion of any endeavor, a standard probability chart (Table 3.5) shows that the more steps or factors involved or needed to complete the job, the greater becomes the chance of failure. To better appreciate how the demand for perfection in every surgical procedure must be, if one presumes that only seven steps are involved in the surgical procedure, and further assumes a 95% probability that each step was completed successfully, the probability of the total success of the operation

Table 3.5. Probability table of successful results. First row: probability of successful completion of each step in the process. Percentages in the table represent the probability of successfully completing the entire process. In a 7-step process, if probability of success in each is step 95%, the probability of a successful outcome is less than 70%

Step	99%	98%	97%	96%	95%	94%	93%	92%	91%	90%
1	0.9900	0.9800	0.9700	0.9600	0.9500	0.9400	0.9300	0.9200	0.9100	0.9000
2	0.9801	0.9604	0.9409	0.9216	0.9025	0.8836	0.8649	0.8464	0.8281	0.8100
3	0.9703	0.9412	0.9127	0.8847	0.8574	0.8306	0.8044	0.7787	0.7536	0.7290
4	0.9606	0.9224	0.8853	0.8493	0.8145	0.7807	0.7481	0.7164	0.6857	0.6561
5	0.9510	0.9039	0.8587	0.8154	0.7738	0.7339	0.6957	0.6591	0.6240	0.5905
6	0.9415	0.8858	0.8330	0.7828	0.7351	0.6899	0.6470	0.6064	0.5679	0.5314
7	0.9321	0.8681	0.8080	0.7514	0.6983	0.6485	0.6017	0.5578	0.5168	0.4783
8	0.9227	0.8508	0.7837	0.7214	0.6634	0.6096	0.5596	0.5132	0.4703	0.4305
9	0.9135	0.8337	0.7602	0.6925	0.6302	0.5730	0.5204	0.4722	0.4279	0.3874
10	0.9044	0.8171	0.7374	0.6648	0.5987	0.5386	0.4840	0.4344	0.3894	0.3487

would be only 69.83%. And this probability considers only one of the five categories (intra-operative factors) mentioned above as reasons for failure. Nevertheless, certainly as related to mesh repairs, technical skills are the most critical factor in the equation of success.

Finally, this verbatim quote from Schroder is worth considering: "Expert surgeons become expert based on repetitive experience, enthusiasm and dedication to a particular field of expertise, hand-eye coordination skills, and intellectual stimulation. Eventual failure of technique is inherent with age, as enthusiasm tends to wane over time, hand-eye coordination skills can diminish, and the fatigue factor plays more of a role with age. As the expert surgeon becomes more known for his/her skills, more work is thrust upon them, which may cause him to rush through their cases, take short cuts that may be inadvisable, and have mental lapses simply due to fatigue which takes more of a toll as we age. Being the expert lends itself to a failure in the expertise, not due to wanton carelessness or overconfidence, but due to the volume of cases and the imperfection of the human being. If you walk a high wire enough times, you will fall. I believe this general statement is applicable for each of the operations requested."

Tough as it may be for expert surgeons to accept this fact gracefully I believe Schroder's comments should be seriously considered. Just as Babe Ruth, Pele, Mohammed Ali, and other notable experts enjoyed being at the top in their field as the result of their excellent ability, dexterity, and performances, there came a time when their physical skills and performances began to slip down the ladder of excellence. Usually, it is the physical component that declines before the cognitive. While value is given to judgment, dexterity, and tenacity, it becomes clear why perfection at best is asymptotic, and that there certainly comes a time in each expert surgeon's career when reputation and desire are not the most reliable predictors of successful out-comes.

References

1. Awad ZT, Puri V, LeBlanc K, Stoppa R, Fitzgibbons RJ, Iqbal A, Filipi CJ (2005) Mechanisms of ventral hernia recurrence after mesh repair and a new proposed classification. JACS 201: 132–140
2. Amid P (2002) How to avoid recurrences in Lichtenstein tension-free hernioplasty. Am J Surg 184: 259–260
3. Read R, Gilbert AI (2004) Interstitial recurrence, with chronic inguinodynia, after Lichtenstein herniorrhaphy. Hernia 8: 264–267
4. Kingsnorth A (2005) Quality of care in hernia surgery: Educational aspects. Hernia (Special Issue) 9: 74

Biological Reasons to Fail

4 Pervasive Co-Morbidity and Abdominal Herniation: an Outline

R.C. Read

Introduction

For most of the 20th century, there was unanimity regarding the etiology of herniae. They arose from congenital defects in normal abdominal wall. The ability to resist protrusion was compromised by straining, poor muscular tone, obesity, debility, ascites, or advancing age. Care was to be undertaken solely by surgeons since repair was curative, provided that recurrence from faulty technique, surgical error, or infection was avoided. Keith [1] did state in 1924 that a pathologic change in the connective tissues of the belly might render certain individuals particularly liable to hernia, but his insight was ignored. The purpose of this review is to outline the evidence which has led to our present understanding regarding the role of co-morbidity in the development of herniae and the implications thereof.

Collagen Malformation and Destruction

In 1967, a young Navy veteran, operated on for an inguinal hernia, was found not to have the expected indirect defect but a direct protrusion. Fasciae and aponeuroses were attenuated with hypertrophied muscles [he was a weight lifter] bulging through the many tears in the transversalis fascia. The appearance prompted the question: is this patient suffering from some unrecognized connective tissue disorder [2]? I had been sensitized to such a possibility by publishing, in 1964–1965, cardiovascular research describing both multiple aneurysms from disseminated cystic medial necrosis [3] and the floppy valve syndrome [a possible forme fruste of Marfan's disease] [4].

In 1970, we reported on a large series of veterans who demonstrated similar atrophy, more marked in those with bilateral or direct defects [5]. Biochemical studies revealed a striking loss of collagen related to decreased synthesis by poorly proliferating fibroblasts [6]. Despite normal cross-linking, which ruled out lathyrism [7], fibrils were cystic with varying diameter and diminished polymerism [8]. Precipitability and hydroxylation were affected, providing further evidence that collagen was not only scarce but abnormal [9]. Skin and pericardium showed similar involvement [10]. We concluded [1977] that a cause of inguinal herniae was systemic disease of collagen [11, 12]. In 1981, another series of veterans with groin herniation was found to have leukocytosis and increased elastolytic activity in the bloodstream accompanied by a decrease in antiproteolytic capacity [13]. Thus, the co-morbidity not only damages fibroblasts, reducing their synthesis of collagen, which is no longer normal, but by inducing an inflammatory reaction destroys existing connective tissue.

Cigarette Smoking

Fortuitously and fortunately, our research was conducted in a veteran's hospital attached to a University medical center. Many of the patients had become addicted to nicotine while serving in the military, free cigarettes being sent up with the rations. Two-thirds with inguinal herniae admitted to the habit, a significantly higher incidence than that found in those admitted with other complaints. Since smoke evokes inflammation in the lungs with recruitment of primed granulocytes and monocytes, we ascribed destruction of collagen to spillover of proteases into the peripheral circulation [14]. Protease-antiprotease imbalance in the lungs was being transferred to other parts of the body (metastatic emphysema).

Toxins present in smoke could be responsible for damaging fibroblasts, thereby decreasing collagen synthesis. They would, in addition, enhance collagenolysis by their known effect on inhibitors, i.e., alpha 1 antitrypsin [15].

These initial studies were later confirmed and extended. Thus, Weitz et al. [16], using a new technique, documented increased neutrophil elastase activity in the blood of smokers. The latter were more likely to develop inguinal herniae, especially women [17]. Defective collagen synthesis, in those who smoke, was noted by Ajabnoor et al. [18] and Jorgensen et al. [19]. Yin et al. [20] showed that in vitro tobacco smoke decreases collagen synthesis by cultured fibroblasts. Knuutinen et al. [21] showed that smoking affects collagen synthesis in skin. Its systemic effects were reviewed [22], while Sorensen et al. [23] showed in 2002 that smoking was a risk factor for groin hernia recurrence.

Collagen Type I/III Ratio

Collagen, the principal component of the extracellular matrix, consists of 20 types. I and III predominate, comprising 95% of the whole. They interact to form the bundle architecture. Type I are strong and big, whereas type III are thin and flexible. Normally, the former are approximately four times as prevalent as the latter. In 1982, Busuttil, a vascular surgeon now renowned for transplantation, discussed our presentation of metastatic emphysema [13]. He reported that the type I/type III ratio was decreased in the aortic media of patients with abdominal aortic aneurysm (AAA). A decade later, Friedman et al. [24] described a similar change in cultured fibroblasts obtained from the skin of patients with inguinal herniation. They concluded that the presence of such a change would render an individual liable to herniation, incisional breakdown, and recurrence.

Much of the recent research on the collagen type I/type III index has been conducted by Schumpelick's group at Aachen. Thus, in the groin, using fascia and skin from patients with hernia, primary and recurrent, they confirmed Friedman's finding of a significant increase in synthesis of type III collagen [25–27]. Using skin, fascia, fibroblasts and peritoneum from patients with primary and recurrent incisional herniation, a similar fall in the Type I/III ratio was noted [28–31]. This index has emerged as a possible clinical tool for determining in any adult with abdominal herniation whether connective tissue disease is present and to what degree. Based on the result, a surgeon should be able to assess pre-operatively which type of repair would be performed and the likelihood of recurrence.

Matrix Metalloproteinases (MMPs)

In 1970, vascular surgeons began investigating the etiology of aneurysms (AAA). In 1985, Busuttil et al. recognized a new protease in the media. Analysis suggested it could be a zinc-dependent metallo-proteinase released by monocytes infiltrating the arterial wall [32]. Their work was soon substantiated. It was not until 1996 that Jackson et al. [33] reported MMPs in the vaginal wall of premenopausal women with genito-urinary prolapse.

Bellon et al. [34, 35] noted that patients with direct inguinal defects had more MMP activity in their transversalis fascia than those with indirect. Zheng et al. [27] identified MMP expression mainly in their patients with recurrent inguinal herniation. These authors showed that MMPs were also released by fibroblasts. Normally, they appear during the inflammatory phase of healing which, in the presence of comorbidity, may be extended.

Like collagen, MMPs have many forms, approximately two dozen being numbered. Secreted in a latent form, they are subjected to inhibitors when activated. One or other may predominate in response to injury, healing or comorbidity. In the future, similarly to the collagen I/III ratio, they may prove useful as markers pre-operatively to signal the presence and degree of comorbidity. They are intimately involved with collagens in processes associated with herniation, repair and recurrence.

Aging and Healing

The incidence of abdominal herniation is known to be higher at the extremes of life. In the young, apart from those with heritable diseases of collagen, there is no evidence that factors other than congenital malformation are involved. Thus, Rosch et al. [36], in a 3-month-old infant with bilateral recurrence of inguinal hernia, found no evidence that his collagen metabolism differed from that of similar patients with primary unilateral defects. The elderly tend to have attenuated fasciae [37] which can be explained by degeneration of elastin and collagen fibrils, observed by Rodrigues et al. [38] in the transversalis fascia of senile men operated on for inguinal herniation. Nikolov and Beltschev [39] found similar changes. Lenhardt et al. [40] reported that collagen deposition after surgery in elderly men was below normal. This effect was not seen in older women, perhaps because of estrogen. The latter, when premenopausal were found by Jorgensen et al. [41], after injury, to produce more collagen than men. Lenhardt et al. [40] speculated that their findings might explain why senile men have twice the incidence of postoperative dehiscence. Good nutrition is essential for good healing. Older men enhanced accumulation of collagen after herniorrhaphy when their diet was supplemented with essential amino acids [42].

Aging impairs injury induced secretion of MMP tissue inhibitors [43]. Increased concentrations of MMP extend the early inflammatory phase of healing when weaker type III collagen fibrils form a temporary matrix. Thereby the risk of recurrence, sometimes delayed, is enhanced. Failure to form a healthy scar can negate the beneficial effect of prostheses since they are not properly incorporated [44]. Thus, it would appear that aging duplicates the effects of comorbidity or evokes a latent connective tissue disease.

Genetic Influences

There is a strong familial tendency to herniation of the groin. Watson (1938, cited in [45]) reported that a quarter of his patients gave a history of a similar diagnosis being made in their parents or grandparents. A Chinese study of indirect inguinal herniae in 280 families indicated transmission was autosomal dominant with incomplete penetrance of a preferential paternal factor [46]. Various connective tissue disorders are known to be heritable or caused by genetic mutation. These include congenital hip dislocation (CDH), homocystinuria, elastosis, Marfan's and Ehlers-Danlos syndromes, along with hypermobility. They are all associated with a high incidence of hernia [47]. Affected patients have disorganized collagen fibers with inadequate cross-linking. Lysyl hydroxylase deficiency was described by Pinnell et al. [48]. Jensen et al. (1986) observed a reduced type 1/type III ratio associated with CDH as Friedman et al. [24] did with hypermobility.

Morris-Stiff et al. [49] and Lederman et al. [50] provided evidence that autosomal dominant polycystic disease, the most common genetic illness, is frequently [43%] coupled with abdominal herniation. Alterations in collagen and elastin have been documented. The abnormality in the basement membranes of the kidney is known to result from disordered production of the extracellular matrix. Renal and hepatic cysts develop along with cerebral aneurysms [10–19%] and floppy mitral valves [26%]. Increasingly, recent data regarding comorbidity and herniae relate to collagen genes and RNA expression [24, 51]. Radiation produces mutations and other environmental factors, i.e., smoke, drugs, etc., may similarly influence transmission of the genotype [19, 52].

Are Aneurysms Herniae of the Arterial Wall?

It is remarkable that, as mentioned above, pioneer vascular surgeons, investigating the etiology of aneurysm, provided data regarding the collagen I/III ratio and MMPs a decade or more before herniologists. As noted previously, Busuttil, in his 1981 discussion [13], stated that the latter markers were not present in the occluded aorta. This information led us to determine the incidence of hernia (inguinal) in veterans with aortic disease. Patients with AAA, but not those suffering from Leriche syndrome, had a history of twice the normal incidence of inguinal herniation even though the latter smoked even more than the former [53]. This suggested an inherited susceptibility to the same acquired comorbidity as that described originally in herniated veterans. These findings have been confirmed. Pleumeeckers et al. [54] showed that the prevalence of AAA was more than three times higher in the elderly with a history of inguinal herniorrhaphy, when compared to those without.

Heightened elastolytic activity was identified by us (1982) in the blood stream of veterans with AAA but not those having Leriche syndrome [14]. Busuttil's group made a similar finding (metalloproteinase) [32]. These observations were confirmed by Cohen et al. in the rabbit [55]. Concentration of MMP may predict

the risk of expansion or rupture of AAAs [56]. Mice, experimentally susceptible to develop AAA, fail to do so when genetic expression of MMPs is denied [57]. The association between cigarette smoking and aneurysm of the aorta was first revealed by Hammond and Horn [58]. The formation, expansion, and rupture of intracranial aneurysms, once considered congenital, is now also ascribed to cigarette smoke. Their incidence rose eight times in patients suffering from alpha-1-antitrypsin deficiency, another cause of systemic protease-antiprotease imbalance [59]. As mentioned, heritable disorders result not only in herniation, but also in aneurysm. Armstrong et al. [60] demonstrated differential gene expression in the aorta which determines whether aneurysm or occlusion results. AAA not only has a familial tendency but is seen more often with aging.

A Pervasive Comorbidity

Other Herniae

Connective tissue disease was first associated with inguinal herniae, then incisional and later genito-urinary prolapse (a euphemism for hernia). This latter is caused by failure of the arcus tendineous fasciae pelvis to support the anterior wall of the vagina, related to a fourfold increase in MMP activity [33] coupled with a decreased collagen I/III ratio [61]. In the groin, we [5] and Bellon et al. [34, 35] noted that atrophy of fasciae and aponeuroses was more severe with direct as opposed to indirect defects. Nevertheless, the latter predominated. Originally [5] this finding was blamed on a patent processus vaginalis facilitating protrusion. However, herniologists have long thought the integrity of the internal ring is maintained by the musculature, despite a greater than 20% retention of the peritoneal sac. Overlapping of the internal oblique is complemented by three sphincters. Failure of these safeguards raises the question "does the comorbidity affect skeletal muscle"?

The observed degeneration of tendons and aponeuroses affects contractility. It may even cause rupture, as in the Achilles tendon, associated with a decreased collagen type-I/III ratio at the rupture site [62]. Further, muscle bundles are bound together into functioning units by a delicate architecture of connective tissue septae [endomysium, perimysium, and epimysium] derived from their fascial sheaths. Deterioration wrought by comorbidity or injury affects muscle collagen, Lehto et al. [63]. Ajabnoor et al. [18] detected, in vitro, reduced proline uptake by cultured myocytes taken from patients with inguinal herniae. Such changes are presumably responsible for muscle wasting, frequently described in the herniated and aged. Disaggregation of muscle bundles explains interstitial herniation in the adult as described in the groin, anterior abdominal wall (Spigelian), pelvis, and diaphragm. In regard to the latter, Filipi et al. [64] have suggested that hiatus herniae may be caused by comorbidity.

Other Organ Systems

The first evidence that in patients with inguinal hernia the identified comorbidity was not restricted to the groin was obtained when similar changes were found in the skin [8], indicating a systemic process. A decade later, the arterial system was shown by Busuttil to be susceptible to the connective tissue disease, resulting in aneurysm [13]. In 1989, Capasso et al. [65] determined that alterations in collagen cross-linking impair myocardial contractility in the mouse heart. Recently, similar data have been obtained in the human with heart failure and other problems associated with changes in the collagen type-I/III ratio [Pauschinger et al. 1999] [66]. Stumpf et al. [67] reported that diverticular disease of the colon is associated with a decreased collagen I/III ratio and reduced expression of MMPs. If Filipi's belief in a role of comorbidity in hiatus herniae is borne out [64], it will show that Saint's triad [68, 69] is, at least for two components, a relationship and not co-incidence. Stumpf et al. [70] later pointed out that a change in the extracellular matrix was a risk factor for anastomotic leakage after bowel surgery and Crohn's disease was accompanied by a reduced collagen type-I/III ratio with increased MMP expression [71]. Recently, damage to smooth muscle, the respiratory tract, skeletal system, liver, fat, and the eye have been attributed to collagen disorder.

In conclusion, the fact that the comorbidity associated with abdominal herniae affects the whole body means that care is no longer the sole province of the herniologist with knowledge of anatomy. Other clinicians need to be involved. Further, information regarding the implications of associated connective tissue disease has to be obtained from the experience of other surgical specialists, pathologists, geneticists, and biologists.

In the future, patients need to be tested for the presence and severity of changes in the extracellular matrix. Prophylaxis or treatment may become available to ward off protrusions and render repair more successful.

References

1. Keith A (1924) On the origin and nature of hernia. Brit J Surg 11: 455–475
2. McVay CB, Read RC, Ravitch MM (1967) Inguinal hernia. Curr Probl Surg: 28–29
3. Read RC, Wolf P (1964) Symptomatic disseminated cystic medial necrosis: report of a case with multiple arterial rupture. N Engl J Med 271: 816–819
4. Read RC, Thal AP, Wendt VE (1965) Symptomatic valvular myxomatous transformation (the floppy valve syndrome): a possible forme fruste of the Marfan syndrome. Circulation 32: 897–910
5. Read RC (1970) Attenuation of the rectus sheath in inguinal herniation. Am J Surg 120: 610–614
6. Wagh PV, Read RC (1971) Collagen deficiency in rectus sheath of patients with inguinal herniation. Proc Soc Exp Biol Med 137: 382–384
7. Wagh PV, Read RC (1972) Defective collagen synthesis in inguinal herniation. Am J Surg 124: 819–822
8. Read RC, Wagh PV, Sun CN, White HJ (1973) Inguinal herniation in men: possible collagen disease. Eur Surg Res 5: 38
9. Sun CN, White HJ, Wagh PV, Read RC (1974) Alteration of collagen fibrils in the direct inguinal herniation of men. 8th Inter Congr Electron Microscopy 2: 482–483
10. Wagh PV, Leverich AP, Sun CN, White HJ, Read RC (1974) Direct inguinal herniation in men: a disease of collagen. J Surg Res 17: 425–433
11. White HJ, Sun CN, Read RC (1977) Inguinal hernia: a true collagen disease? Lab Invest 36: 359
12. Read RC, White HJ (1978) Inguinal herniation 1777–1977. Am J Surg 136: 651–654
13. Cannon DJ, Read RC (1981) Metastatic emphysema: a mechanism for acquiring inguinal herniation. Ann Surg 194: 270–278
14. Cannon DJ, Read RC (1982) Blood elastolytic activity in patients with aortic aneurysm. Ann Thorac Surg 34: 10–15
15. Read RC (1992) A review: the role of protease-antiprotease imbalance in the pathogenesis of herniation and abdominal aortic aneurysm in certain smokers. Postgrad Gen Surg 4: 161–165
16. Weitz JI, Crowley KA, Landman SL, et al. (1987) Increased neutrophil elastase activity in cigarette smokers. Ann Intern Med 107: 680–682
17. Bielecki K, Pulawski R (1988) Is cigarette smoking a causative factor in the development of inguinal hernia. Pol Tyglek 43: 974–976
18. Ajabnoor MA, Mokhtar AM, Rafee AA, et al. (1992) Defective collagen metabolism in Saudi patients with hernia. Ann Clin Biochem 29: 430–436
19. Jorgensen LN, Kallehave F, Christensen E, et al. (1998) Less collagen production in smokers. Surgery 123(4): 450–455
20. Yin L, Morita A, Tsuji T (2000) Alterations of extracellular matrix induced by tobacco smoke extract. Arch Dermatol Res 292; 188–194
21. Knuutinen A, Kokkonen N, Risteli J, et al. (2002) Smoking affects collagen synthesis and extracellular matrix turnover in human skin. Brit J Dermatol 146: 588–594
22. Read RC (1984) Systemic effects of smoking. Am J Surg 148: 706–711
23. Sorensen LT, Friis E, Jorgensen T, et al. (2002) Smoking is a risk factor for recurrence of groin hernia. World J Surg 26: 397–400
24. Friedman DW, Boyd CD, Norton P, et al. (1993) Increases in Type III collagen gene expression and protein synthesis in patients with inguinal hernias. Ann Surg 218(6): 754–760
25. Klinge U, Zheng H, Si ZY, et al. (1999) Altered collagen synthesis in fascia transversalis of patients with inguinal hernia. Hernia 4(3): 181–187
26. Klinge U, Zheng H, Si ZY, et al. (1999) Expression of the extracellular matrix proteins collagen I, collagen III, fibronectin and matrix metalloproteinase-1 and -3 in the skin of patients with inguinal hernia. Eur Surg Res 31: 480–490
27. Zheng H, Si Z, Kasperk R, et al. (2002) Recurrent inguinal hernia: disease of the collagen matrix? World J Surg 261: 401–408
28. Klinge U, Si ZY, Zheng H, et al. (2000) Abnormal collagen I to III distribution in the skin of patients with incisional hernia. Eur Surg Res 32: 43–48
29. Klinge U, Si Z, Zheng H, et al. (2001) Collagen I/III and matrix metalloproteinases MMP-1 and -3 in the fascia of patients with incisional hernia. J Invest Surg 14: 47–54
30. Si Z, Rhanjit B, Rosch R, et al. (2002) Impaired balance of type I and type III procollagen mRNA in cultured fibroblasts of patients with incisional hernia. Surgery 131(3): 324–331
31. Rosch R, Junge K, Knops M, et al. (2003) Analysis of collagen-interacting proteins in patients with incisional hernias. Langenbecks Arch Surg 387: 427–432
32. Brown SL, Backstrom B, Busuttil RW (1985) A new serum proteolytic enzyme in aneurysm pathogenesis. J Vasc Surg 2(3): 393–399
33. Jackson SR, Avery NC, Tarlton FR, et al. (1996) Changes in metabolism of collagen in genitourinary prolapse. Lancet 347: 1658–1661
34. Bellon JM, Bajo A, Honduvilla NG, et al. (1997) Study of biochemical substrate and role of metalloproteinases in fascia transversalis from hernial processes. Eur J Clin Investigation 27: 510–516
35. Bellon JM, Majo A, Honduvilla NG, et al. (2001) Fibroblasts from the transversalis fascia of young patients with direct inguinal hernias show constitutive MMP-2 overexpression. Ann Surg 233 (2): 287–291
36. Rosch R, Junge K, Lynen P, et al. (2004) A case of bilateral inguinal hernia recurrence in infancy: investigations on collagen metabolism. Hernia 8(2): 160–163
37. Peacock EE, Madden JW (1974) Studies on the biology and treatment of recurrent inguinal hernia: II. Morphological changes. Ann Surg 179: 567–571
38. Rodrigues AJ, Tolosa EM, de Carvalho CA (1990) Electron microscopic study on the elastic and elastic-related fibers in human transversalis fascia at different ages. Gegenbaurs Morpkal Jahrb 136: 645–652
39. Nikolov VS, Beltschev B (1990) Some ultrastructural features of fascia transversalis in direct hernias in senile men. Anat Anz Jena 170: 265–272
40. Lenhardt R, Hopf HW, Marker E, et al. (2000) Perioperative collagen deposition in elderly and young men and women. Arch Surg 135: 71–74

41. Jorgensen LN, Sorensen LT, Kallehave F, et al. (2000) Premenopausal women deposit more collagen than men during healing of an experimental wound. Surgery 13: 338–343
42. Williams JZ, Abumrad N, Barbul A, et al. (2002) Effect of a specialized amino acid mixture on human collagen deposition. Ann Surg 236(3): 369–375
43. Ashcroft GS, Horan MA, Herrick SE, et al. (1997) Human aging impairs injury-induced in vivo expression of tissue inhibitor of matrix metalloproteinases (TIMP)-1 and -2 proteins and mRNA. J Pathol 183: 169–176
44. Junge K, Klinge U, Rosch et al. (2004) Decreased collagen type I/III ratio in patients with recurring hernia after implantation of alloplastic prostheses. Langenbecks Arch Surg 389: 17–22
45. Smith MP, Sparkes RS (1968) Familial inguinal hernia. Surgery 57: 809–812
46. Gong Y, Shao C, Sun Q et al. (1994) Genetic study of indirect inguinal hernia. J Med Genet 31: 187–192
47. Uden A, Lindhagen T (1988) Inguinal hernia in patients with congenital dislocation of the hip: a sign of general connective tissue disorder. Acta Orthop Scand 59(6): 667–668
48. Pinnell SR, Krane S, Kenzora JE, et al. (1972) A heritable disorder of connective tissue: hydroxylysine-deficient collagen disease. N Engl J Med 286: 1013–1020
49. Morris-Stiff G, Coles G, Moore R, et al. (1997) Abdominal wall hernia in autosomal dominant polycystic kidney disease. Brit J Surg 84: 615–617
50. Lederman ED, McCoy G, Conti DJ, et al. (2000) Diverticulitis and polycystic kidney disease. Am Surgeon 66; 200–203
51. Rosch R, Klinge U, Si Z, et al. (2002) A role for the collagen I/III and MMP-1/-13 genes in primary inguinal hernia. BMC Medical Genetics 3: 2–6
52. Hein R, Mauch C, Hatamochi A, et al. (1988) Influence of corticosteroids on chemotactic response and collagen metabolism of human skin fibroblasts. Biochem Pharmacol 37: 2723–2729
53. Cannon DJ, Casteel L, Read RC (1984) Abdominal aortic aneurysm, Leriche's Syndrome, inguinal herniation, and smoking. Arch Surg 119: 387–389
54. Pleumeeckers HJC, de Gruijla A, van Beck AJ, et al. (1999) Prevalence of aortic aneurysm in men with a history of inguinal hernia repair. Brit J Surg 86: 1155–1158
55. Cohen JR, Sarfati I, Wise L (1989) The effect of cigarette smoking on rabbit aortic elastase activity. J Vasc Surg 9: 580–582
56. Lindholt JS, Vammen S, Fasting H, et al. (2000) The plasma level of matrix metalloproteinase 9 may predict the natural history of small abdominal aortic aneurysms. A preliminary study. Eur J Vasc Endovasc Surg 3: 281–285
57. Buckley C, Wyble CW, Borhani M, et al. (2004) Accelerated enlargement of experimental abdominal aortic aneurysms in a mouse model of chronic cigarette smoke exposure. J Am Coll Surg 199(6): 896–903
58. Hammond EC, Horn D (1958) Smoking and death rates: report on forty-four months of follow-up of 187,783 men. II: Death rates by cause. JAMA 166: 1294–1308
59. Baker CJ, Flore A, Connolly ES Jr, et al. (1995) Serum elastase and alpha-1-antitrypsin in patients with ruptured and unruptured cerebral aneurysms. Neurosurgery 37(1): 56–61
60. Armstrong PJ, Johanning JM, Franklin DP, et al. (2002) Differential gene expression in human aorta: aneurysmal versus occlusive disease (abstract F9). Program and Abstracts of the Annual Meeting of the Society for Vascular Surgery, Boston, MA
61. Moalli PA, Talarico LC, Sung VW (2004) Impact of menopause on collagen subtypes in the arcus tendineous fasciae pelvis. Am J Obstet Gyn 190: 620–627
62. Eriksen HA, Pajala A, Leppilahti J, et al. (2002) Increased content of Type III collagen at the rupture site of human Achilles tendon. J Orthop Res 6: 1352–1357
63. Lehto M, Sims TJ, Bailey AJ (1985) Skeletal muscle injury – molecular changes in the collagen during healing. Res Exp Med (Berl) 185(2): 95–106
64. Puri V, Kakarlapudi GV, Awad ZT, Filipi CJ (2004) Hiatal hernia recurrence: 2004. A review. Hernia 8(4): 311–317
65. Capasso JM, Robinson TF, Anversa P (1989) Alteration in collagen cross-linking impairs myocardial contractility in the mouse heart. Cir Res 65: 1657–1664
66. Pauschinger M, Knopf D, Petschaver S, et al. (1999) Dilated cardiomyopathy is associated with significant changes in collagen type I/III ratio. Circulation 99(21): 2750–2756
67. Stumpf M, Cao W, Klinge U, et al. (2001) Increased distribution of collagen Type III and reduced expression of matrix metalloproteinases in patients with diverticular disease. Int J Colorectal Dis 16(5): 271–275
68. Muller CJB (1948) Hiatus hernia, diverticula, and gallstones. S African Med J: 376.
69. Saint CFM (1966) Saint's triad. The origin and story of its recognition. Rev Surg 23(1): 1–4
70. Stumpf M, Klinge U, Wilms A, et al. (2005) Changes of the extracellular matrix as a risk factor for anastomotic leakage after large bowel surgery. Surgery 137(2); 229–234
71. Stumpf M, Cao W, Klinge U, et al. (2005) Reduced expression of collagen Type I in patients with Crohn's disease. J Invest Surg 18(1): 33–38

Discussion

Schumpelick: *Is the hernia disease a collagen disease or is it a mechanical disease based on weak collagen? What is first? Is it a mechanical or a collagen disorder? Where does it start?*

Read: *Well, it can be a congenital disease. I think in the adult there a lot of acquired factors.*

Schumpelick: *And the collagen is only one factor?*

Read: *Exactly. I think we need to look at things like muscle cells, fibroblasts and other cells.*

Schumpelick: *Could you imagine prophylactic procedures if we knew the collagen type of the patient?*

Read: *We should. I think it vital that we start to know how much collagen disease an individual patient has. We need a screening test.*

Jeekel: *Of course the correlation between the inguinal hernia disease and aneurysms is well known; we also found that there is a relation between inguinal hernia*

and incisional hernia. Patients with the two together, inguinal and incisional hernia, will have a 38% chance to have or develop an aneurism.

Read: *I think this is a very important contribution. And the fact that this comorbidity is pervasive. It seems to affect every organ in the body so we have to be concerned about such things: if we have an inguinal hernia, are we able to handle it? We need to have a broader view. As herniologists, we tend to somewhat confine our studies but we have the concern that this is the human body, with systemic problems.*

Chowbey: *1. Do you feel that it is a bilateral disease to begin with? 2. Is there any way to predict that a patient will develop a hernia in the course of time?*

Read: *I think it is bilateral. When we first started all this work there was a great tendency to think that this was a local phenomenon, maybe affected by the sac or other problems in one area and then it became understood it is bilateral and affects other parts of the body as well. We should be able to predict and maybe give prophylaxis if we can identify the presence and extent of the comorbidity in any individual patient.*

Chowbey: *We are all doing laparoscopic inguinal hernia repair and for many years we have seen that it provides a good opportunity to do a bilateral repair. Do you think it is justified for a patient with a unilateral hernia to do a bilateral repair?*

Read: *I think it is up to the surgeon. I believe that it's probably wise to do a bilateral repair if you can do it without increasing the problems of the operation itself. You have to be pretty skilled to take on that responsibility.*

Kehlet: *I think this is fascinating research but I should like to take your hypothesis one step further into clinical practice and the future. Given that we have a test, given that we have a pharmaceutical agent to treat this collagen disorder – and this should be lifelong treatment – do you really think that this is the future compared to doing a simple and cheap mesh repair?*

Read: *I just don't know. We need more data. This is research for the future.*

Campanelli: *In your last slide you talk about drugs. What do you think about stem cells that restore the right collagen in the future? Second, if the hernia is a collagen disease, we don´t need any controlled and randomized trial to decide to use always the prosthesis? If it is a collagen disease, the simple suture repair does not make sense. What do you think?*

Read: *Well, I agree with you that we should never use sutures if there is presence of some connective tissue disease. Secondly, stems cells are an interesting observation. I think that this is the future. Maybe we can grow some new fibroblasts that are not damaged. I believe that fibroblasts can be damaged by smoking. There is evidence for this. I also believe that fibroblasts can be damaged by congenital changes in the DNA.*

Mertens: *I believe that it is a collagen disease. Actually we are very close to having a test system. The next thing is, we have to understand genomics much more than we do right now. When you say congenital, you believe that we have one set of genes that don't alter at all during a lifetime – and that is not true. Like you die because your telomer length is shortened. So there is a change over life. And the same happens with the collagen gene. There are changes over time and such a test system will prove that over time there are changes within the collagen promotor region. This is what we are doing at present and I will present it on Saturday. So I believe that we have to understand that congenital does not mean that it has to happen all the time and that there are confounding factors like smoking which also affect these processes over time and that in the end will lead to recurrence. I hope that we will have this test system in the near future that provides the advice whether to use mesh material or not. Because I believe that meshes are required in most instances in these patients, but these mesh materials should not be inert but should somehow improve the scar quality.*

Read: *I think that is a very important comment. I don't know anything about DNA and I know less about RNA. But the point is that you people have demonstrated that the RNA expression may change. We cannot completely separate congenital from acquired factors.*

Schumpelick: *Professor Mertens, can we treat the collagen disease by any drug or method?*

Mertens: *I think that you produce a collagen disease every day in your daily practice. When you use ACE-inhibitors you block MMP-2 in the whole body. When you use statins you have a huge effect on MMP-2 levels in your cells. By the drugs that we use in our everyday practice we change a lot of these things that are related to collagen. When you have a vitamin C deficiency you get scurvy. In all the medications we use we have not assessed their impact on collagen turnover. My point of view is: we have a set of genes that have some predictive factors for a collagen disease. I even want to put it further: when you look at patients that I normally treat like patients with diabetes mellitus only 30% progress to end stage renal disease. These 30% are different from the other 70%, and we don't know yet why. This is some kind of disturbed wound healing in the kidney from my perspective.*

Kingsnorth: *A drug is being developed for the treatment of hernia which is calcitonin gene-related peptide. It is not a drug that affects collagen metabolism but it is in*

research in Australia and that may be appropriate for treating infantile hernias because it seems to shrink the infantile hernia sac and may cause a retraction of the infantile hernia sac, therefore not require herniotomy. So the future is here, possibly very soon around the corner with this peptide. My question is: you talked about targeting different groups of patients that have an increased incidence of hernias. Patients with Ehlers Danlos syndrome – of which there are seven types – don't all have an increased incidence of hernia. So, would it be appropriate to take out one group and find out what is going on in their genes? As we stated earlier, this is certain to be a genetic polymorphism because it is a multifactorial thing. Secondly, what about patients who have diastasis of the rectus abdominis? These patients who have spontaneously their linea alba stretched and these problems must be a prime target for looking at patients who have a collagen disorder and get ventral hernias.

Read: *I think we have to build on the points that Dr. Kingsnorth has made. I think the important thing is that this is an enormous field now and the day that the hernia was strictly a problem of the surgeon has gone. We need help from different medical disciplines.*

Bendavid: *I should like to support the idea that not only direct inguinal hernias – as you mentioned in the older population – but now stronger evidence is beginning to appear for the chemical basis also for indirect inguinal hernias starting in children and there is a wonderful work being done in Turkey, so that I think eventually all hernias should be considered as a chemical or metabolic collagen disease and where the mechanical aspects will affect the chemical basis and the mechanical aspects would be a strict reflection of the metabolic aspect. And I do not want to finish without praising Professor Read for his incredible research and for really giving a boost to this area of hernia knowledge. Thank you, Dr. Read.*

5 Non-Surgical Risk Factors for Recurrence of Hernia

L.T. Sorensen, L.N. Jorgensen

Introduction

Abdominal wall hernias may recur as long as 15 years after herniotomy. Recurrences which appear within 6 months following surgery are regarded as technical failures due to inadequate surgical technique. Recurrences which appear later than 6–9 months after the primary surgical procedure may be considered as a result of the abnormal collagen metabolism, which originally led to herniation [1, 2].

The pathological mechanisms for the non-surgical risk factors associated with recurrence of inguinal hernia or formation of abdominal wall hernia, for that matter, are not fully understood. Roughly, these risk factors may be divided into non-modifiable and potentially modifiable risk factors.

The aim of this study is to review the literature regarding non-surgical physiological and biochemical mechanisms involved in both primary development and recurrence of abdominal wall hernia.

Non-Modifiable Risk Factors for Hernia Formation and Recurrence

A higher prevalence of inguinal hernia is well known among patients suffering from congenital connective tissue disorders like osteogenesis imperfecta, cutis laxa, Ehlers-Danlos syndrome, Hurler-Hunter's and Marfan's syndrome [3–5]. In children with congenital hip dislocation, inguinal hernia occurs more frequently [6], and patients with Ehlers-Danlos syndrome exhibit a higher incidence of recurrent incisional hernia [7]. There is no evidence, however, to suggest that other genetically predisposed patients undergoing hernia repair are more likely to experience recurrent hernia formation.

The prevalence of inguinal hernia increases significantly with patient age [8]. Experimental studies show that the activity of collagen-degrading enzymes is higher in elder patients, presumably due to a reduced inhibition of collagenase [9, 10]. It is not clear whether patient age is associated with recurrence of inguinal hernia. In a study of 544 patients undergoing inguinal hernia repair, a multivariate analysis showed that patient age was not an independent risk factor for recurrence within 2 years postoperatively [11]. In a recent study of incisional hernia, patient age was inversely associated with recurrence [12].

The incidence of inguinal hernia is higher in males, a difference partly due to embryological characteristics of each gender. It is puzzling, however, that one fifth of men pass into adulthood with a patent processus vaginalis, but less than half develop clinical herniation [1, 13]. In addition, indirect inguinal hernia may appear first in a man over 40 years of age [1]. These observations suggest that other factors may play a role in the development of an indirect hernia including structural abnormalities of the internal ring, acquired attenuation of transversalis fascia or abnormal muscle function ac-

companying a congenital defect [1, 14–16]. More recent studies have found that men accumulate less collagen in surgical test wounds compared to women presumably due to discrepancy in systemic concentrations of female reproductive hormones [17–19].

Further, there is evidence of a higher incidence of abdominal incisional hernia in men than in women [20, 21]. Male patients with abdominal aortic aneurism specifically demonstrate high incidence of both, primary and recurrent inguinal hernia [22–26], as well as a 30% risk of incisional hernia following open aneurism repair [22–25, 27–29]. These findings support the view of a common causative connective tissue metabolism defect in abdominal aortic aneurismal disease and abdominal wall hernia formation and recurrence [24, 26].

Potentially Modifiable Risk Factors for Hernia Formation and Recurrence

Prolonged strain of the fascia transversalis caused by chronically raised intra-abdominal pressure is considered a facilitating factor for the formation of groin hernia [5, 15, 30, 31]. This condition may be secondary to heavy work load, coughing, ascites, hyperplasia of the prostate, constipation and pregnancy [5, 8, 16, 31–33]. However, raised intraabdominal pressure as a causative factor in abdominal wall hernia recurrence is speculative [34] and not supported by recent evidence [11, 21]. More likely, it is a structural weakness in the connective tissue that facilitates the hernia formation rather than a momentary increase of the intra-abdominal pressure.

Smokers have a two-fold higher risk of inguinal hernia recurrence and a four-fold higher risk of incisional hernia independent of other risk factors [11, 21, 34]. Whether smoking is a risk factor for the formation inguinal hernia, too, remains unclear, as the available few and small studies report conflicting results [5, 32, 33, 35].

A biochemical study of smokers with inguinal hernia, especially the direct type, has demonstrated significantly higher blood levels of elastine degrading activity and lower levels of protease inhibitors [36]. Smoking appears to induce a protease-anti-protease imbalance, and may also partly explain the higher incidence of other tissue-destructive disorders including abdominal aorta aneurism, pulmonary emphysema and parodontosis [26, 36–40].

Accordingly, recent studies have found a higher neutrophil collagenase level and a higher reactivity of neutrophils and monocytes in smokers compared to non-smokers [41, 42]. Another explanation is impaired collagen biosynthesis induced by smoking. Smokers accumulate less collagen in surgical test wounds than non-smokers [43] and tissue hypoxia induced by nicotine and carbon monoxide reducing the concentration of molecular oxygen in the tissue has been suggested as causative factors.

Connective Tissue Attenuation as a Predisposing Factor for Abdominal Wall Hernia and Recurrence

Structural studies on tissue samples obtained from the anterior rectus abdominis sheath and from the fascia transversalis in patients with groin hernia have shown a significant attenuation of the fascia transversalis on the asymptomatic contralateral side [1]. An increase in levels of biological elasticity and maximal distension of the fascia transversalis has also been found with the highest levels in patients with direct inguinal hernia and intermediate values in patients with indirect inguinal hernia [44]. Corresponding alterations in the architecture of groin connective tissue have been reported, suggesting higher levels of immature collagen and loss of resiliency of the fascia transversalis [45, 46]. Significantly lower levels of proline and lysine hydroxylation have been reported in transversus abdominis fascia samples from patients with direct hernia compared to those with indirect hernia [30, 47]. This indicates that the stability of the collagen in the transversus abdominis fascia is compromised in patients with direct hernias.

The ultrastructure of the connective tissue biopsies from the anterior rectus abdominis sheath reveals a lower collagen diameter and periodicity in patients with direct inguinal hernia as compared to patients with indirect inguinal hernia or controls [47]. Biopsies obtained from the fascia transversalis or the peritoneum from hernia patients show no differences in collagen fibril diameter [30], but unevenly arranged collagen microfibrils and more abundant interfibrillar matrix and collagen build-up in the subserosal fibrous tissue of hernial sacs. These findings – being at variance with the most current surgical concept of hernia formation – were more pronounced in direct than indirect inguinal hernia [30, 48, 49]. There is evidence of alterations in the elastic system of the fascia transversalis of hernia patients with a decrease in oxytalan fibres and an increase in amorphous substance of the elastic fibres, resulting in a lowered resistance of the fascia transversalis [50]. Interestingly, the qualitative changes observed

in the rectus sheath biopsies from patients with direct hernia have also been found in other tissues such as the pericardium, supporting the view that the disease is systemic [51].

Higher levels of soluble collagen in hernia patients have also been observed [47, 52, 53]. The collagen I: III ratio in fascia transversalis in hernia patients has shown a significant ratio decrease and a concomitant increase in collagen type III protein synthesis in direct and indirect inguinal hernia as well in incisional and recurrent incisional hernia. Similar results were obtained when tissue from hernial sacs and skin fibroblasts was examined [54–60]. Supportive of this finding is a significantly lower procollagen I:III ratio in skin fibroblasts of hernia patients compared to controls [2]. However, other studies report conflicting results failing to detect any difference in the collagen I:III ratio in fascia transversalis or anterior rectus abdominis sheath between direct and indirect inguinal hernia patients [30, 45]

It is unclear whether increased collagenolysis predisposes to hernia formation or recurrence. The higher prevalence of an inguinal hernia in patients with abdominal aorta aneurysm (26–41%) suggests the view that there is a common causative proteolytic factor [24, 26]. Metalloproteinase I (MMP-1) expression was more pronounced in patients with recurrent incisional or inguinal hernias than in controls [60]. Significantly higher levels of Metalloproteinase II (MMP-2) have been found in transversus abdominis biopsies and in fibroblasts from fascia transversalis from patients with direct inguinal hernia compared to patients with indirect hernia; this may reflect an overall proteolytic effect [30, 31] A concomitant significant increase of MMP-1 mRNA and protein was found in skin fibroblasts in patients with a recurrent inguinal hernia compared to controls [61], but it remains unclear whether this is associated with hernia formation [54, 58].

Impaired Wound Healing as a Predisposing Factor for Hernia Formation and Recurrence

Cultured fibroblasts from biopsies of patients with hernia have showed a longer generation time and a lower incorporation of radioactively labelled (^{14}C) proline, indicating lower rates of cell proliferation and retarded cellular biosynthesis. The lowest values were found in patients with direct hernia, but the number of patients in this study was too small to allow for a proper statistical evaluation [53]. In a larger study, it was found that fibroblasts obtained from the internal oblique muscle and the cremasteric muscle in hernia patients exhibited retarded proliferation compared to controls [62]. In addition, the incorporation of radioactively labelled proline in the tissue from cultured fibroblasts was significantly depressed, suggesting a lower rate of synthesis of matrix in hernia patients. A study from our group did not show any difference between patients with inguinal hernia and controls in the deposition of collagen as measured in a subcutaneous test wound in the arm after 10 days [63].

We have assessed the possible link between concentrations of proteinases (MMP-2 and MMP-9) in the wound fluid from hernia patients and the amount of collagen deposited in an implanted ePTFE model within the surgical wound [64]. High levels of MMP-9 were found after 24 h, reflecting the inflammatory phase of healing. The concentration of MMP-9 24 h after surgery correlated negatively with the amount of collagen deposited after 10 days in the implanted ePTFE fibre. It was concluded that MMP-9 might be a predictor of impaired healing in this type of wound. There are still no data, however, on whether the wound fluid is representative of the healing processes taking place in the transversalis fascia.

Potential Treatment of Tissue Destruction and Impaired Wound Healing

Transplantation of autologous tissue such as the anterior rectus sheath or the fascia lata has been shown to accelerate the synthesis and deposition of collagen for at least 2 years [65]. However, it has been argued in many papers that patients with inguinal hernia present a generalized defect in fibrillogenesis. The use of autologous tissue may not be the perfect solution, therefore, as this tissue may express the same abnormalities. There is reason to believe that synthetic materials such as polypropylene are to be preferred. Such materials provide scaffolding and induce an intense inflammatory response with brisk secondary fibrillogenesis.

In experimental studies, the implantation of type-I collagen sponges seeded with fibroblasts or coated with the growth factor bFGF raised the collagen deposition and the tensile strength of dermal wounds [66]. Future clinical studies will show whether there is place for the application in hernia wounds of similar material with stimulatory effects on collagen synthesis and deposition resulting in more durable tissues.

There is still too little knowledge on how to classify hernia patients into different categories of risk for compromised healing and the development of hernia recurrence in particular. If simple and relevant assays on collagen metabolism should become available in the future, there may be a place for a specific choice of surgical technique based on the individual patient's wound-healing potential.

Conclusion

Although the literature contains diverging results, it seems most likely that the pathophysiology of hernia formation and recurrence is due to metabolic alterations in the connective tissue. Data from smokers and patients with abdominal aortic aneurismal disease indicate that the connective tissue alterations toward poorer or more immature collagen structure are systemic.

The results from examinations of the fascia transversalis or the anterior rectus abdominis sheath also support the theory of a systemic connective tissue disease. This is reflected by the frequent reports of inclining connective tissue alterations with inclining disease – indicating worse results in direct inguinal hernias compared to controls, with intermediate values in indirect inguinal hernias. A systemic alteration is also supported by the fact that the connective tissue of the asymptomatic contralateral side of inguinal hernia patients already is affected when compared to controls. The potential of preventing hernia formation and recurrence is present if future studies show that medical agents may reduce tissue degradation or stimulate connective tissue formation of he abdominal wall.

References

1. Peacock EE Jr, Madden JW (1974) Studies on the biology and treatment of recurrent inguinal hernia. II. Morphological changes. Ann Surg 179: 567–571
2. Friedman DW, Boyd CD, Norton P, et al. (1993) Increases in type III collagen gene expression and protein synthesis in patients with inguinal hernias. Ann Surg 218: 754–760
3. Liem MS, van der GY, Beemer FA, van Vroonhoven TJ (1997) Increased risk for inguinal hernia in patients with Ehlers-Danlos syndrome. Surgery 122: 114–115
4. Hayakawa A, Fujimoto K, Ibayashi H (1982) Two cases of Ehlers-Danlos syndrome with gastrointestinal complications. Gastroenterol Jpn 17: 61–67
5. Abrahamson J (1998) Etiology and pathophysiology of primary and recurrent groin hernia formation. Surg Clin North Am 78: 953–72
6. Uden A, Lindhagen T (1988) Inguinal hernia in patients with congenital dislocation of the hip. A sign of general connective tissue disorder. Acta Orthop Scand 59: 667–668
7. Girotto JA, Malaisrie SC, Bulkely G, Manson PN (2000) Recurrent ventral herniation in Ehlers-Danlos syndrome. Plast Reconstr Surg 106: 1520–1526
8. Abramson JH, Gofin J, Hopp C, Makler A, Epstein LM (1978) The epidemiology of inguinal hernia. A survey in western Jerusalem. J Epidemiol Community Health 32: 59–67
9. Ashcroft GS, Horan MA, Herrick SE, Tarnuzzer RW, Schultz GS, Ferguson MW (1997) Age-related differences in the temporal and spatial regulation of matrix metalloproteinases (MMPs) in normal skin and acute cutaneous wounds of healthy humans. Cell Tissue Res 290: 581–591
10. Ashcroft GS, Herrick SE, Tarnuzzer RW, Horan MA, Schultz GS, Ferguson MW (1997) Human ageing impairs injury-induced in vivo expression of tissue inhibitor of matrix metalloproteinases (TIMP)-1 and -2 proteins and mRNA. J Pathol 183: 169–176
11. Sorensen LT, Friis E, Jorgensen T, Vennits B, Andersen BR, Rasmussen GI, et al. (2002) Smoking is a risk factor for recurrence of groin hernia. World J Surg 26: 397–400
12. Sauerland S, Schmedt CG, Lein S, Leibl BJ, Bittner R (2005) Primary incisional hernia repair with or without polypropylene mesh: a report on 384 patients with 5-year follow-up. Langenbecks Arch Surg 390: 408–412
13. Conner WT, Peacock EE Jr (1973) Some studies on the etiology of inguinal hernia. Am J Surg 126: 732–735
14. Menck J, Lierse W (1991) The fascia of the inguinal canal ring. Chirurg 62: 117–120
15. Stelzner F (1994) Function of the abdominal wall and development and therapy of hernias (among others: the paracolostomy hernia). Langenbecks Arch Chir 379: 109–119
16. McArdle G (1997) Is inguinal hernia a defect in human evolution and would this insight improve concepts for methods of surgical repair? Clin Anat 10: 47–55
17. Lenhardt R, Hopf HW, Marker E, et al. (2000) Perioperative collagen deposition in elderly and young men and women. Arch Surg 135: 71–74
18. Jorgensen LN, Sorensen LT, Kallehave F, Vange J, Gottrup F (2002) Premenopausal women deposit more collagen than men during healing of an experimental wound. Surgery 131: 338–343
19. Ashcroft GS, Greenwell-Wild T, Horan MA, Wahl SM, Ferguson MW (1999) Topical estrogen accelerates cutaneous wound healing in aged humans associated with an altered inflammatory response. Am J Pathol 155: 1137–1146
20. Bucknall TE, Cox PJ, Ellis H (1982) Burst abdomen and incisional hernia: a prospective study of 1129 major laparotomies. Br Med J (Clin Res Ed) 284: 931–933
21. Sorensen LT, Hemmingsen UB, Kirkeby LT, Kallehave F, Jorgensen LN (2005) Smoking is a risk factor for incisional hernia. Arch Surg 140: 119–123
22. Hall KA, Peters B, Smyth SH, et al. (1995) Abdominal wall hernias in patients with abdominal aortic aneurysmal versus aortoiliac occlusive disease. Am J Surg 170: 572–575
23. Raffetto JD, Cheung Y, Fisher JB, et al. (2003) Incision and abdominal wall hernias in patients with aneurysm or occlusive aortic disease. J Vasc Surg 37: 1150 1154

24. Lehnert B, Wadouh F (1992) High coincidence of inguinal hernias and abdominal aortic aneurysms. Ann Vasc Surg 6: 134–137
25. Adye B, Luna G (1998) Incidence of abdominal wall hernia in aortic surgery. Am J Surg 175: 400–402
26. Cannon DJ, Casteel L, Read RC (1984) Abdominal aortic aneurysm, Leriche's syndrome, inguinal herniation, and smoking. Arch Surg 119: 387–389
27. Stevick CA, Long JB, Jamasbi B, Nash M (1988) Ventral hernia following abdominal aortic reconstruction. Am Surg 54: 287–289
28. Augestad KM, Wilsgaard T, Solberg S (2002) Incisional hernia after surgery for abdominal aortic aneurysm. Tidsskr Nor Laegeforen 122: 22–24
29. Holland AJ, Castleden WM, Norman PE, Stacey MC (1996) Incisional hernias are more common in aneurysmal arterial disease. Eur J Vasc Endovasc Surg 12: 196–200
30. Bellon JM, Bujan J, Honduvilla NG, et al. (1997) Study of biochemical substrate and role of metalloproteinases in fascia transversalis from hernial processes. Eur J Clin Invest 27: 510–516
31. Bellon JM, Bajo A, Ga-Honduvilla N, et al. (2001) Fibroblasts from the transversalis fascia of young patients with direct inguinal hernias show constitutive MMP-2 overexpression. Ann Surg 233: 287–291
32. Carbonell JF, Sanchez JL, Peris RT, et al. (1993) Risk factors associated with inguinal hernias: a case control study. Eur J Surg 159: 481–486
33. Flich J, Alfonso JL, Delgado F, Prado MJ, Cortina P (1992) Inguinal hernia and certain risk factors. Eur J Epidemiol 8: 277–282
34. Clark JL (2001) Ventral incisional hernia recurrence. J Surg Res 99: 33–39
35. Bielecki K, Pulawski R (1988) Is cigarette smoking a causative factor in the development of inguinal hernia? Pol Tyg Lek 43: 974–976
36. Cannon DJ, Read RC (1981) Metastatic emphysema: a mechanism for acquiring inguinal herniation. Ann Surg 194: 270–278
37. Vardulaki KA, Walker NM, Day NE, Duffy SW, Ashton HA, Scott RA (2000) Quantifying the risks of hypertension, age, sex and smoking in patients with abdominal aortic aneurysm. Br J Surg 87: 195–200
38. Lindholt JS, Jorgensen B, Klitgaard NA, Henneberg EW (2003) Systemic levels of cotinine and elastase, but not pulmonary function, are associated with the progression of small abdominal aortic aneurysms. Eur J Vasc Endovasc Surg 26: 418–422
39. Janoff A (1985) Elastases and emphysema. Current assessment of the protease-antiprotease hypothesis. Am Rev Respir Dis 132: 417–433
40. van Laarhoven CJ, Borstlap AC, Berge Henegouwen DP, Palmen FM, Verpalen MC, Schoemaker MC (1993) Chronic obstructive pulmonary disease and abdominal aortic aneurysms. Eur J Vasc Surg 7: 386–390
41. Knuutinen A, Kokkonen N, Risteli J, et al. (2002) Smoking affects collagen synthesis and extracellular matrix turnover in human skin. Br J Dermatol 146: 588–594
42. Sorensen LT, Nielsen HB, Kharazmi A, Gottrup F (2004) Effect of smoking and abstention on oxidative burst and reactivity of neutrophils and monocytes. Surgery 136: 1047–1053
43. Jorgensen LN, Kallehave F, Christensen E, Siana JE, Gottrup F (1998) Less collagen production in smokers. Surgery 123: 450–455
44. Pans A, Pierard GE, Albert A, Desaive C (1997) Adult groin hernias: new insight into their biomechanical characteristics. Eur J Clin Invest 27: 863–868
45. Pans A, Albert A, Lapiere CM, Nusgens B (2001) Biochemical study of collagen in adult groin hernias. J Surg Res 95: 107–113
46. Rodrigues Junior AJ, Rodrigues CJ, da Cunha AC, Jin Y (2002) Quantitative analysis of collagen and elastic fibers in the transversalis fascia in direct and indirect inguinal hernia. Rev Hosp Clin Fac Med Sao Paulo 57: 265–270
47. Wagh PV, Leverich AP, Sun CN, White HJ, Read RC (1974) Direct inguinal herniation in men: a disease of collagen. J Surg Res 17: 425–433
48. Baradi AF, Heslop JH, Rao NS (1986) Peritoneal fine structure of inguinal hernia: a scanning electron microscope study. Histol Histopathol 1: 89–92
49. Baradi AF, Parry BR, Heslop JH (1992) Peritoneal fine structure of inguinal hernia: a transmission electron microscope study. Histol Histopathol 7: 251–257
50. Rodrigues Junior AJ, de Tolosa EM, de Carvalho CA (1990) Electron microscopic study on the elastic and elastic related fibres in the human fascia transversalis at different ages. Gegenbaurs Morphol Jahrb 136: 645–652
51. White HJ, Sun CN, Read RC (1977) Inguinal hernia: a true collagen disease. Lab Invest 36: 359
52. Wagh PV, Read RC (1971) Attenuation of the rectus sheath in inguinal herniation. Proc Soc Exp Biol Med 137: 382 384
53. Wagh PV, Read RC (1972) Defective collagen synthesis in inguinal herniation. Am J Surg 124: 819–822
54. Klinge U, Zheng H, Si ZY, Schumpelick V, Bhardwaj R, Klosterhalfen B (1999) Synthesis of type I and III collagen, expression of fibronectin and matrix metalloproteinases-1 and -13 in hernial sac of patients with inguinal hernia. Int J Surg Investig 1: 219–227
55. Klinge U, Zheng H, Si Z, et al. (1999) Expression of the extracellular matrix proteins collagen I, collagen III and fibronectin and matrix metalloproteinase-1 and -13 in the skin of patients with inguinal hernia. Eur Surg Res 31: 480–490
56. Klinge U, Si ZY, Zheng H, Schumpelick V, Bhardwaj RS, Klosterhalfen B (2000) Abnormal collagen I to III distribution in the skin of patients with incisional hernia. Eur Surg Res 32: 43–48
57. Klinge U, Si ZY, Zheng H, Schumpelick V, Bhardwaj RS, Klosterhalfen B (2001) Collagen I/III and matrix metalloproteinases (MMP) 1 and 13 in the fascia of patients with incisional hernias. J Invest Surg 14: 47–54
58. Rosch R, Klinge U, Si Z, Junge K, Klosterhalfen B, Schumpelick V (2002) A role for the collagen I/III and MMP-1/-13 genes in primary inguinal hernia? BMC Med Genet 3: 2
59. Si Z, Bhardwaj R, Rosch R, Mertens PR, Klosterhalfen B, Klinge U (2002) Impaired balance of type I and type III procollagen mRNA in cultured fibroblasts of patients with incisional hernia. Surgery 131: 324–331
60. Rosch R, Junge K, Knops M, Lynen P, Klinge U, Schumpelick V (2003) Analysis of collagen-interacting proteins in patients with incisional hernias. Langenbecks Arch Surg 387: 427–432

61. Zheng H, Si Z, Kasperk R, et al. (2002) Recurrent inguinal hernia: disease of the collagen matrix? World J Surg 26: 401–408
62. Ajabnoor MA, Mokhtar AM, Rafee AA, Taha AM (1992) Defective collagen metabolism in Saudi patients with hernia. Ann Clin Biochem 29 (Pt 4): 430–436
63. Jorgensen LN, Sorensen LT, Kallehave F, Schulze S, Gottrup F (2001) Increased collagen deposition in an uncomplicated surgical wound compared to a minimal subcutaneous test wound. Wound Repair Regen 9: 194–199
64. Ågren MS, Jorgensen LN, Andersen M, Viljanto J, Gottrup F (1998) Matrix metalloproteinase 9 level predicts optimal collagen deposition during early wound repair in humans. Br J Surg 85: 68–71
65. Peacock EE Jr (1975) Subcutaneous extraperitoneal repair of ventral hernias: a biological basis for fascial transplantation. Ann Surg 181: 722–727
66. Marks MG, Doillon C, Silver FH (1991) Effects of fibroblasts and basic fibroblast growth factor on facilitation of dermal wound healing by type I collagen matrices. J Biomed Mater Res 25: 683–696

Discussion

Franz: *Our group is very impressed by your results. In fact, we now require our patients to cease smoking for the month around the recurrent incisional hernia operation. We check urinary nicotines to make sure they are compliant with our request. We don't have any data that this makes any difference, but we were so impressed with your excellent work that that is how we practice now. Do you have any idea what mechanism is responsible for the effect? Is it oxygen delivery, is it some direct effect of nicotine, is it coughing, is it a physiological effect? Finally, in your last slides you showed an increased risk for infections. Which comes first: is the cigarette smoking a risk factor for wound infections or are they independent?*

Jorgensen: *We have other data suggesting that cigarette smoking increased the risk for wound infection. But for sure, in the majority of patients who did not have wound infection there is also a higher risk for hernia formation. So it is multifactorial. I showed a slide with all potential mechanisms: it concerns breakdown, hypoperfusion of the tissue due to the nicotine action. Smoking one cigarette diminishes subcutaneous perfusion for approximately 45 minutes.*

Deysine: *The first portion of your presentation I found fascinating: you measure hydroxyproline deposition in those PTFE implants. Have you measured collagen quality and type, because this is an enormous science and, for example, as mentioned before, patients with Ehlers Danlos type 4 just do not produce collagen as we do?*

Jorgensen: *We are doing that at the moment but we just do not have the data. But I had to refer to the group from Aachen where they have done a lot of these studies which is very interesting to consider the type-I/III ratio.*

Kingsnorth: *Your data are very interesting. Perhaps, that is suggesting that there are different etiological factors, biological factors in failure of incisional as opposed to inguinal hernia because the failure rate curves are different. You can follow the failure rates for inguinal hernia that show a constant increase of failure rate even after 20 and 25 years. Your data show us that with incisional hernia most of these hernias are symptomatic after 3 months. Now either that suggests a different biological phenomenon or maybe it is all mechanical, maybe it is all the surgeon's failure – an incisional and not a collagen failure?*

Jorgensen: *Yes, a hard question! The idea of our study was merely to point out that there are some common risk factors but for sure there are several other factors to take into consideration and I am not sure how each factor weighs in this specific area of surgery.*

Kingsnorth: *Do you think that the surgeon has got more to do with the failure than the collagen?*

Jorgensen: *Perhaps. But if you implant a mesh at the primary surgical procedure it is perhaps to a higher degree associated with the collagen potential. But these are speculations.*

Schumpelick: *How do you explain the fact that ex-smokers have bad results, too? Is it a chronic disease after smoking? How long was the duration of ex-smoking?*

Jorgensen: *It seems that there are long-lasting effects of smoking perhaps during the first couple of years after quitting but I do not have data about the duration of abstinence. But it supports the idea of a pathophysiological factor.*

6 The Instable Scar

R. Rosch, M. Binnebösel, K. Junge, P. Lynen-Jansen, P.R. Mertens, U. Klinge, V. Schumpelick

Introduction

There is increasing evidence that inguinal hernia formation is based on a disorder of the connective tissue biology [1–12]. Similarly, secondary herniations as incisional hernias or diverse recurrent hernias are supposed to be associated with biological factors that provoke an instable scar formation during the wound-healing process. Patients with congenital connective tissue disorders have a higher risk of developing of incisional or recurrent hernias [13, 14]. Furthermore, aneurismal disease, as another collagen disorder, has repeatedly been shown to be associated with an increased risk for the development of incisional hernias [15–17]. Previous work was able to support the hypothesis of a dysregulation of the collagen metabolism in patients with incisional hernias. In patients with either primary or recurrent incisional hernias, a significantly decreased ratio of collagen type I to type III and alterations of collagen-interacting proteins were found [18–21]. An alteration of the collagen composition was further verified by a comparative immunhistochemical analysis of surgical mesh explants where patients operated for hernia recurrence had a lowered collagen I/III ratio as compared to patients operated due to mesh infection or mesh-related pain [22].

In order to understand what leads to this altered connective tissue quality in (recurrent) incisional hernia patients, we performed an immunohistochemical characterization of factors with potential impact on the wound-healing process in comparison to non-hernia patients.

Materials and Methods

Abdominal skin scars from patients with recurrent incisional hernias were excised in the course of hernia repair. Abdominal skin scars of patients without any history or clinical evidence of hernia who underwent relaparotomy due to diverse intra-abdominal diseases served as controls. Patients under steroid therapy, extraordinary obesity (body mass index > 35) or history of connective tissue diseases were excluded from the study.

Immunohistochemical and cross polarization microscopy studies were performed with paraffin embedded tissue sections. The following primary antibodies were applied: catenin, c-myc, factor XIII, notch, SMA, ESDN, TGF-β, PAI, uPAR, YB-1, COX-2, p53. Collagen-I/III ratios were analyzed as described previously [21]. The expression of immunohistochemical parameters was analyzed by an immunoreactive score (IRS) where the score ranges from 1 to 20 [23].

In order to characterize the functional network of the investigated parameters, associations between variables were calculated through two-sided Spearman correlation test within each group. Associations between variables were assumed with p values < 0.05 and shown graphically. Additionally, linkages between parameters were calculated by the clustering coefficient c (see Fig. 6.1):

$$c = \frac{E}{K(K-1)/2}$$

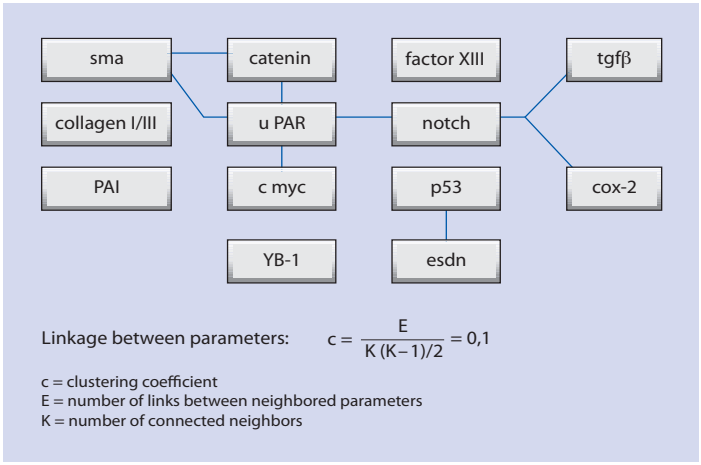
Results

Functional interferences of the matrix parameters within the postulated network were assessed by two-sided Spearman correlation analyses. ◘ Figures 6.1 and 6.2 show the graphic representation of this network with related parameters linked by bars (significant correlation coefficient with $p < 0.05$). In summary, we found differences in association patterns of matrix parameters between skin scars from recurrent hernia and from control patients. In the recurrent hernia group associations were found for collagen I/III with factor XIII and uPAR, PAI and c-may and TGF-β and COX-2. In the control group there were associations between SMA and catenin, ESDN and p53, notch and TGF-β and COX-2 and between uPAR and c-myc, notch, catenin and SMA.

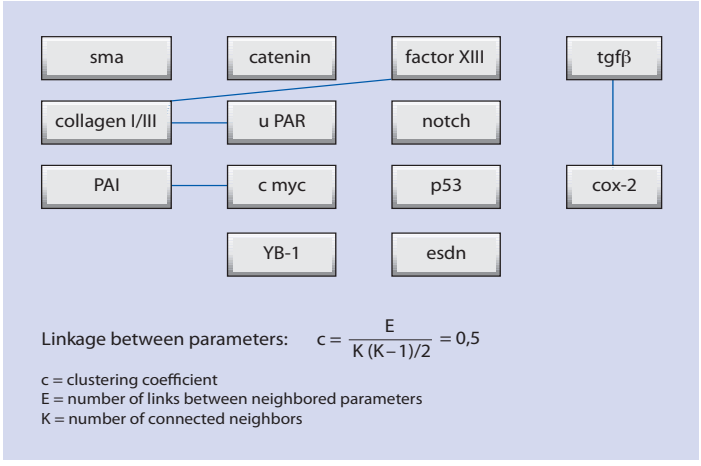
Calculated cluster coefficients (c) within the two networks also showed pronounced differences between both groups with a higher degree of crosslinking in controls ($c = 0.1$) as compared to recurrent hernia ($c = 0.05$).

Discussion

Wound healing and scar formation are tightly regulated and highly dynamic and complex processes characterized by permanent cell turnover and matrix remodelling. The modulation of this network is influenced by the interplay of numerous cellular and extracellular factors, hereby determining the quality of scar formation. Already mild disturbances of this



◘ **Fig. 6.1.** Spearman correlations – control scar



◘ **Fig. 6.2.** Spearman correlations – recurrent hernia scar

system may thus predispose to insufficient scar formation, hereby leading to incisional or recurrent hernia formation. For inguinal hernia recurrence, which might be regarded as a subtype of incisional hernia showing with similar alterations of collagen quality, Sorensen et al. have shown that smoking is an important risk factor, presumably due to an abnormal connective tissue metabolism in smokers [24, 25].

Previous investigations about the collagen-interacting proteins in scar tissue from patients with incisional hernias showed divergent expression patterns for the matrix-metalloproteinase MMP-1 and the discoidin domain receptor DDR-2 when compared to controls [21]. Additionally, in in vitro studies about fibroblast function in patients with recurrent incisional hernias we found a specific cell response after biomaterial contact as compared to control fibroblasts [26].

These results indicated an altered remodelling and phenotype in a population at risk that might be regarded as additional causative factors for a defective scar formation. However, focusing on single alterations of expression profiles does not reflect the complex cross talk within the cellular and extracellular matrix network during wound healing. With the analyses of correlations and clustering of diverse parameters with known impact on cell-cell adhesion and interaction, migration, angiogenesis, cell differentiation and proliferation we thus tried to map the scar architecture in patients with recurrent incisional hernias as compared to controls. Here, the different associations between matrix parameters and respective clustering coefficients indicate a different intercommunication within the (cellular and extracellular) matrix in recurrent incisional patients that possibly is responsible for a defective scarring process.

Conclusion

In addition to a modified cell function our results indicate a disturbed intercommunication and a comprehensive change of matrix composition and turnover within the scar tissue in recurrent incisional hernia patients as a potential cause for the development of an insufficient scar formation. Further studies are needed for the understanding of the complex functions of this biological network.

Acknowledgements. We are grateful to Mrs. Ellen Krott for most excellent and careful assistance during this investigation.

References

1. Liem MS, van der Graaf Y, Beemer FA, van Vroonhoven TJ. Increased risk for inguinal hernia in patients with Ehlers-Danlos syndrome. Surgery 122 (1997) 114–115
2. Uden A, Lindhagen T. Inguinal hernia in patients with congenital dislocation of the hip. A sign of general connective tissue disorder. Acta Orthop Scand 59 (1988) 667–668
3. Pleumeekers HJ, De Gruijl A, Hofman A, Van Beek AJ, Hoes AW. Prevalence of aortic aneurysm in men with a history of inguinal hernia repair. Br J Surg 86 (1999) 1155–1158
4. Wagh PV, Read RC. Defective collagen synthesis in inguinal herniation. Am J Surg 124 (1972) 819–822
5. Conner WT, Peacock EE Jr. Some studies on the etiology of inguinal hernia. Am J Surg 126 (1973) 732–735
6. White HJ, Sun CN, Read RC. Inguinal hernia: a true collagen disease. Lab Invest 36 (1977) 359
7. Ajabnoor MA, Mokhtar AM, Rafee AA, Taha AM. Defective collagen metabolism in Saudi patients with hernia. Ann Clin Biochem 29 (Pt 4) (1992) 430–436
8. Bellon JM, Bujan J, Honduvilla NG, Jurado F, Gimeno MJ, Turnay J, Olmo N, Lizarbe MA. Study of biochemical substrate and role of metalloproteinases in fascia transversalis from hernial processes. Eur J Clin Invest 27 (1997) 510–516
9. Klinge U, Zheng H, Si ZY, Bhardwaj R, Klosterhalfen B, Schumpelick V. Altered collagen synthesis in fascia transversalis of patients with inguinal hernia. Hernia 3 (1999) 181–187
10. Klinge U, Zheng H, Si Z, Schumpelick V, Bhardwaj RS, Muys L, Klosterhalfen B. Expression of the extracellular matrix proteins collagen I, collagen III and fibronectin and matrix metalloproteinase-1 and -13 in the skin of patients with inguinal hernia. Eur Surg Res 31 (1999) 480–490
11. Klinge U, Zheng G, Si ZY, Schumpelick V, Bhardwaj R, Klosterhalfen B. Synthesis of type I and III collagen, expression of fibronectin and matrix metalloproteinases-1 and -13 in hernial sac of patients with inguinal hernia. Int J Surg Investig 1 (1999) 219–227
12. Rosch R, Klinge U, Si Z, Junge K, Klosterhalfen B, Schumpelick V. A role for the collagen I/III and MMP-1/-13 genes in primary inguinal hernia? BMC Med Genet 3 (2002) 2
13. Girotto JA, Malaisrie SC, Bulkely G, Manson PN. Recurrent ventral herniation in Ehlers-Danlos syndrome. Plast Reconstr Surg 106 (2000) 1520–1526
14. McEntyre RL, Raffensperger JG. Surgical complications of Ehlers-Danlos syndrome in children. J Pediatr Surg 12 (1977) 531–535
15. Augestad KM, Wilsgaard T, Solberg S. Incisional hernia after surgery for abdominal aortic aneurysm. Tidsskr Nor Laegeforen 122 (2002) 22–24
16. Adye B, Luna G. Incidence of abdominal wall hernia in aortic surgery. Am J Surg 175 (1998) 400–402
17. Papadimitriou G, Pitoulias G, Papaziogas B, Koutsias S, Vretzakis G, Argiriadou H, Papaziogas T. Incidence of abdominal wall hernias in patients undergoing aortic surgery for aneurysm or occlusive disease. Vasa 31 (2002) 111–114
18. Klinge U, Si ZY, Zheng H, Schumpelick V, Bhardwaj RS, Klosterhalfen B. Abnormal collagen I to III distribution in the skin of patients with incisional hernia. Eur Surg Res 32 (2000) 43–48

19. Klinge U, Si ZY, Zheng H, Schumpelick V, Bhardwaj RS, Klosterhalfen B. Collagen I/III and matrix metalloproteinases (MMP) 1 and 13 in the fascia of patients with incisional hernias. J Invest Surg 14 (2001) 47–54
20. Si Z, Rhanjit B, Rosch R, Rene PM, Klosterhalfen B, Klinge U. Impaired balance of type I and type III procollagen mRNA in cultured fibroblasts of patients with incisional hernia. Surgery 131 (2002) 324–331
21. Rosch R, Junge K, Knops M, Lynen P, Klinge U, Schumpelick V. Analysis of collagen-interacting proteins in patients with incisional hernias. Langenbecks Arch Surg 387 (2003) 427–432
22. Junge K, Klinge U, Rosch R, Mertens PR, Kirch J, Klosterhalfen B, Lynen P, Schumpelick V. Decreased collagen type I/III ratio in patients with recurring hernia after implantation of alloplastic prostheses. Langenbecks Arch Surg 389 (2004) 17–22
23. Stegner HE, Remmele W. Recommendation for uniform definition of an immunoreactive score (IRS) for immunohistochemical estrogen receptor detection (ER-ICA) in breast cancer tissue. Pathologe 8 (1987) 138–140
24. Zheng H, Si Z, Kasperk R, Bhardwaj RS, Schumpelick V, Klinge U, Klosterhalfen B. Recurrent inguinal hernia: disease of the collagen matrix? World J Surg 26 (2002) 401–408
25. Sorensen LT, Friis E, Jorgensen T, Vennits B, Andersen BR, Rasmussen GI, Kjaergaard J. Smoking is a risk factor for recurrence of groin hernia. World J Surg 26 (2002) 397–400
26. Rosch R, Lynen-Jansen P, Junge K, Knops M, Klosterhalfen B, Klinge U, Mertens PR, Schumpelick V. Biomaterial-dependent MMP-2 expression in fibroblasts from patients with recurrent incisional hernias. Hernia 10 (2006) 1–6

Discussion

Franz: *There are some recurrences so early so that there has to be a mechanical surgical component and yet groups like your own are demonstrating clear biological effects in these complicated patients. I have two questions: can you predict who is going to develop an incisional hernia? If you had a pre-operative biopsy, are you able to predict who, after the laparotomy, will develop an incisional hernia? Obviously that would be the ultimate goal. And finally do you have any idea how possibly a failed wound might lead to a systemic change? I personally think that a lot of early surgical failures are the mechanism behind these incisional hernias and yet you are demonstrating a systemic effect. Are you able to predict prospectively who develops a hernia and have you any idea how maybe an early laparotomy wound might induce changes you are seeing in the skin?*

Rosch: *With regard to the first part of the question I hope that we will in future have a test and Dr. Mertens has told us already that he will talk about this part. It might be possible, but maybe not for all hernia patients because there are also the technical reasons that have nothing to do with a pathological scar formation or the collagen metabolism. So we have to separate the patients at risk from technical reasons and also from other factors like smoking or medication. We still have to study a lot to understand the system and it is still quite unclear where to focus on – maybe on different parts of this network structure.*

Franz: *But the skin of all these patients has healed despite the measurement of a matrix disorder?*

Rosch: *Yes, the skin is easy to investigate; of course, it would be better to investigate the fascial structures. But what we found in the fascial structures before with regard to the collagen type-I/III ratio was the same as in the skin.*

Kehlet: *I totally agree with you regarding incisional hernias. But what about the inguinal hernias? Do you think that this is important in inguinal hernias and is it going to replace a sufficient surgical technique or should we simply not consider this for inguinal hernias?*

Rosch: *You have to separate the primary hernias from the recurrent inguinal hernias, which in my opinion are similar to incisional hernias with regard to their pathogenesis – they are also a kind of incisional hernia.*

Kehlet: *But you see the problem with these series with no recurrences so maybe it is a question of surgical technique and not of the collagen problem in inguinal recurrences?*

Rosch: *If you have a mesh structure in your inguinal region it is more difficult to develop a recurrence.*

Kingsnorth: *I think we must separate the two problems. Dr. Read was talking about direct herniation which is a primary phenomenon of collagen. We must separate this from the patients with wound failure, which is incisional hernia. These patients have had their fascia disturbed and it is a failure of the wound rather than a primary failure of the fascia. So I think it is certain that there are differences between these two mechanisms and we should not confuse the two.*

Rosch: *Of course not. But the patients who will develop primary hernias also more often develop secondary hernias, for example incisional hernias. The reason why the collagen type-I/III ratio is disturbed might be different in the secondary as compared to the primary hernias. But the problem remains the same.*

Kingsnorth: *Yes, I agree, because we have just seen the data this morning that show that patients with direct hernias have a higher instance of recurrence. This is probably because they already have a primary phenomenon that caused the direct hernia but in addition they may have a secondary phenomenon which is scar failure, wound failure itself. But I think that these are two separate mechanisms.*

Rosch: *Yes, possibly different factors that are combined.*

7 Biomaterials: Disturbing Factors in Cell Cross-Talk and Gene Regulation

P. Lynen-Jansen, U. Klinge, D.H. Lovett, P.R. Mertens

Introduction

Surgical mesh materials are the most frequently used medical devices designed to reinforce fascial structures and thereby treat hernia disease [1, 2]. The implantation of non-absorbable polymeric biomaterials excites perpetual activation of cytokine cascades and proteases that are a chronic inflammatory reaction and postoperative complications like seroma, mesh shrinkage and migration, adhesion, infection and pain may ensue [2]. To circumvent such an on-going foreign body reaction, gold standard meshes have been designed to improve biocompatibility. Such meshes meet the demand for a reduced amount of implanted material, have optimized pore size and adjustment to physiological requirements [3]. Besides this approach to optimize mesh integration and concurrently replace fascial structures in hernia patients, an open question is whether there are alternate means of beneficially influencing the foreign-body reaction. To address this, an in-depth understanding of the molecular mechanisms that guide the extent of foreign-body reactions is required.

Mesh-Induced Foreign-Body Reaction

The fact that tissue cells respond to biomaterial implantation is illustrated by granuloma formation and cell infiltration surrounding mesh materials over time (Fig. 7.1). Common cellular components of such a reaction are infiltrating macrophages that are dispersed in the developing granuloma. These cells have the propensity to synthesize a plethora of pro-inflammatory cytokines [transforming growth factor-β (TGF-β)], platelet-derived growth factor (PDGF), vascular endothelial growth factor (VEGF)) and are regarded as key players directing the extent of fibrosis with influence on the phenotypic behaviour of surrounding fibroblasts [4, 5]. Residential fibroblasts independently contribute to the regulation of tissue remodelling and wound healing. They occur as activated myofibroblasts encapsulating the mesh filaments and are constitutionally involved in extracellular matrix (ECM) remodeling by synthesizing type-I and type-III collagen. Furthermore, fibroblasts are the source of enzymes involved in matrix degradation such as matrix metalloproteinases (MMPs) that may affect the ongoing foreign-body reaction. MMPs are the most abundant proteases in wound healing [6] and MMP-2 (72-kDa collagenase, gelatinase A) enzymatic activities are upregulated in diseases associated with inflammatory reaction such as arthritis [7], cancer [8], atheroma [9] and tissue ulceration [10]. A pivotal role for MMP-2 in hernia disease was determined by a study that detected elevated levels of MMP-2 enzymatic activity in wound fluids of hernia patients [10]. Beyond their capability to hydrolyze components of the ECM, MMP-2 directly affects cellular phenotypes, proliferation rates and the inflammatory reaction, and several studies indicate that MMP-2 is centrally involved in the inflammatory and fibrotic response [11, 12]. Regarding

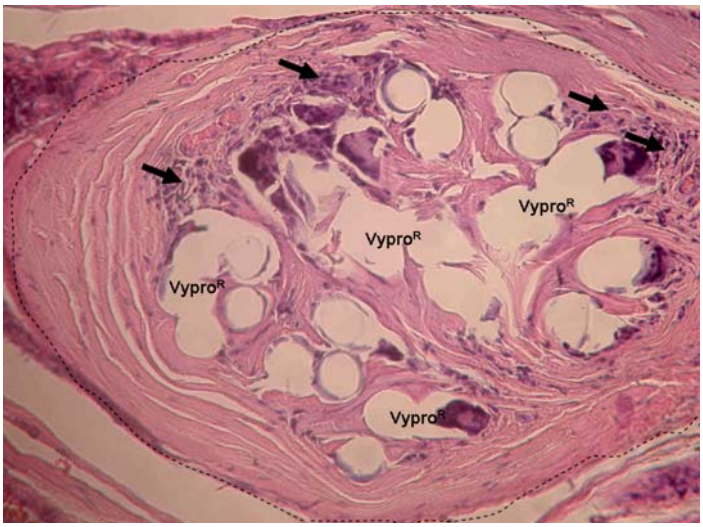


Fig. 7.1. Granuloma formation (*dashed lines*) and cell influx (*arrows*) 24 months after implantation of a Vypro mesh that was explanted because of chronic pain. HE staining, 200x magnification

foreign-body reaction, it is known that macrophages are activated by polymeric nanoparticles and secrete MMP-2 in vitro [13]. Blockage of MMP-2 activation with MMP inhibitor Ilomastat dampens the inflammatory cell infiltration, indicating that MMP-2 mediates the cross-talk of cells and ECM components [15]. In vivo, stimulation of MMP-2 expression may result from a complex cross-talk between cells, especially fibroblasts and macrophages in wound healing. These findings hint at a pivotal role of MMP-2 in wound healing and foreign-body reaction and suggest the investigation of the molecular mechanisms that govern MMP-2 gene transcription after biomaterial implantation.

Transgenic Mice: Models to Grasp Gene Function in Wound Healing and Foreign-Body Reaction

Transgenic mouse models are useful tools to illuminate the precise molecular regulation of wound healing and the impact of mesh implantation on this highly complex and well-balanced process [13]. Histomorphological studies revealed three major phases of wound healing: early inflammation, followed by proliferation and initial matrix deposition, and finally formation of granulation tissue and a "stable" scar. The differential expression of growth factors, cytokines and specific matrix components, especially fibrillar type-I and -III collagens during each of these phases leads to the approach of topical application of particular cytokines aiming to alter wound healing kinetics [14]. Transgenic as well as knockout models were established to elucidate the underlying gene regulation required for wound healing. Transgenic technology has focused on growth factors (FGF, TGF-β) as well as matrix components (plasminogen, stomolysin) [13]. Such transgenic mice are created by injection of a gene of interest into a recently fertilized one-cell embryo. The gene expression is governed by a chosen regulatory element that was incorporated into the injected DNA construct (transgene). Regulatory elements (promoters) contain sequences activated by tissue-specific transcription factors and are small segments of DNA positioned in front of specific genes. Previously, we have outlined specific regulatory elements for the MMP-2 gene that reside up to -1686 bp of the 5'- flanking region [15, 16]. A strong response element denoted RE-1 was identified at -1282/1322 bps, to which the transcription factors activating protein-2 (AP2) [17], Y-box protein-1 (YB-1) [18], non-metastasizing protein 23 (nm23) [19], signal transduction and activator of transcription factor 3 (Stat3) [20] and p53 [21], all may bind, mostly cooperatively. Given the pivotal role of this enzyme in wound healing, it was our primary goal to unravel the molecular mechanisms that govern MMP-2 gene transcription after biomaterial implantation in vivo. Transgenic reporter mice were established that take advantage of a LacZ reporter gene driven by MMP-2 gene regulatory sequences. Transgenic mice harbouring bps -1686/+423 of the rat MMP-2 gene including the response element RE-1 were utilized to determine timely and spatial transcriptional regulation of the MMP-2 gene after biomaterial implantation.

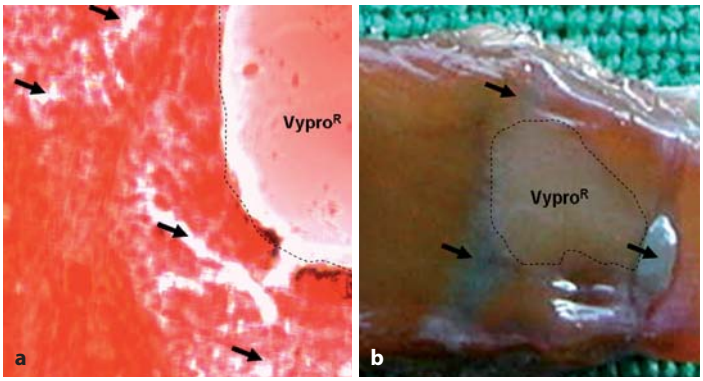


Fig. 7.2. a MMP enzymatic activity (white holes, indicated by arrows) 90 days after implantation of Vypro (*dashed lines*), in situ zymography, 400x magnification. **b** MMP-2 gene expression (blue color, indicated by arrows) in MMP-2/LacZ transgenic mice 90 days after implantation of Vypro (*dashed lines*) β-galactosidase assay

Meshes Interfere with Cell Cross Talk and MMP-2 Gene Regulation

Meshes interfere with MMP-2 gene regulation due to soluble factors, ECM modification or cell cross-talk. In MMP-2/LacZ transgenic mice the impact of mesh implantation on MMP-2 gene expression can be evaluated and compared to MMP-2 enzymatic activity, protein synthesis and expression/binding of transcription factors. As depicted in Fig. 7.2, implantation of polymeric meshes induces MMP-2 gene expression (Fig. 7.2, right panel). By performing a β-galactosidase assay 90 days after mesh implantation, we detected reporter gene expression surrounding the mesh material indicating that the MMP-2 regulatory element –1686/+423 drives mesh-induced MMP-2 gene expression. In accordance, in situ zymography assessed that matrix turnover in vicinity to alloplastic material is predominantly provided by MMP-2 proteolytic activity (Fig. 7.2, left panel). Immunfluorescence analysis was performed to analyze mesh-induced MMP-2 gene transcription at the cellular level. These experiments revealed that macrophages and fibroblasts are distinctively involved in MMP-2 gene transcription: macrophages that exhibit the MMP-2 transcriptional phenotype are widely distributed throughout the foreign-body granuloma whereas macrophages in more than 80–100 μm distance to the mesh filaments are not involved in MMP-2 transcription. Fibroblasts form band-like structures surrounding the mesh filaments and exhibit strong staining patterns for MMP-2 protein, but only a subgroup of these cells is involved in MMP-2 gene transcription. In contrast to our findings for macrophages, different MMP-2 regulatory elements govern transcription in fibroblasts that reside outside the tested promoter sequences. Such findings indicate an intimate cell cross-talk initiated by implantation of alloplastic materials and reflect a zonal as well as cell-specific MMP-2 gene regulation in mesh-related foreign-body reaction.

Outlook

The pivotal role of meshes for MMP-2 gene expression may lead to novel therapeutic strategies in mesh modification that utilizes meshes as carriers for medication to improve biocompatibility. Such bioactive meshes may take corrective action in mesh-related impaired tissue repair due to siRNA, mesh coating or comedication. In a similar approach that aimed at a modified transcriptional regulation of MMP-2, Miyake et al. demonstrated that treatment with anti-NFκB and Ets chimeric decoy oligonucleotides reduced the activities of MMP-2 and MMP-9 and therefore prevented the development of abdominal aortic aneurysms in a rabbit model [22]. In view of the significance of optimizing biocompatibility and avoiding short- and long-term postoperative complications after mesh repair, the MMP-2/LacZ animal model may be a useful model to test the effect(s) of mesh modifications that are created to interfere with MMP-2 transcription.

Acknowledgements. This work was supported by the German Research Association (Deutsche Forschungsgemeinschaft, DFG, grant KL 1320/2–1 (U.K., P.R.M.) and JA 1123/1–1 (P.L.J., U.K.,P.R.M., M. J.), SFB 542 project C4 (P.R.M.), IZKF-BIOMAT, RWTH-Aachen, project no. NTV 41 and NIH grant DK039776 (to D.H.L.).

References

1. Jansen PL, Mertens PP, Klinge U, Schumpelick V: The biology of hernia formation. Surgery 2004, 136:1–4
2. Junge K, Klinge U, Rosch R, Mertens PR, Kirch J, Klosterhalfen B, Lynen P, Schumpelick V: Decreased collagen type I/III ratio in patients with recurring hernia after implantation of alloplastic prostheses. Langenbecks Arch Surg 2004, 389: 17–22
3. Klinge U, Klosterhalfen B, Muller M, Anurov M, Ottinger A, Schumpelick V: Influence of polyglactin-coating on functional and morphological parameters of polypropylene-mesh modifications for abdominal wall repair. Biomaterials 1999, 20: 613–623
4. Chapman HA: Disorders of lung matrix remodeling. J Clin Invest 2004, 113: 148–157
5. Li Y, Yang J, Dai C, Wu C, Liu Y: Role for integrin-linked kinase in mediating tubular epithelial to mesenchymal transition and renal interstitial fibrogenesis. J Clin Invest 2003, 112: 503–516
6. Woessner JF Jr: MMPs and TIMPs – an historical perspective. Mol Biotechnol 2002, 22: 33–49
7. Ishikawa T, Nishigaki F, Miyata S, et al.: Prevention of progressive joint destruction in collagen-induced arthritis in rats by a novel matrix metalloproteinase inhibitor, FR255031. Br J Pharmacol 2005, 144: 133–143
8. Mandal M, Mandal A, Das S, Chakraborti T, Chakraborti S: Clinical implications of matrix metalloproteinases. Molecular and Cellular Biochemistry 2003, 252: 305–329
9. Wu M, Li YG: The expression of CD40-CD40L and activities of matrix metalloproteinases in atherosclerotic rats. Mol Cell Biochem 2006, 282: 141–146
10. Agren MS: Gelatinase activity during wound healing. Br J Dermatol 1994, 131: 634–640
11. Turck J, Pollock AS, Lee LK, Marti HP, Lovett DH: Matrix metalloproteinase 2 (gelatinase A) regulates glomerular mesangial cell proliferation and differentiation. J Biol Chem 1996, 271: 15074–15083
12. Marti HP, Lee L, Kashgarian M, Lovett DH: Transforming growth factor-beta 1 stimulates glomerular mesangial cell synthesis of the 72-kd type IV collagenase. Am J Pathol 1994, 144: 82–94
13. Arbeit JM, Hirose R: Murine mentors: transgenic and knockout models of surgical disease. Ann Surg 1999, 229: 21–40
14. Mustoe TA, Pierce GF, Thomason A, Gramates P, Sporn MB, Deuel TF: Accelerated healing of incisional wounds in rats induced by transforming growth factor-beta. Science 1987, 237: 1333–1336
15. Harendza S, Pollock AS, Mertens PR, Lovett DH: Tissue-specific enhancer-promoter interactions regulate high level constitutive expression of matrix metalloproteinase 2 by glomerular mesangial cells. J Biol Chem 1995, 270: 18786–18796
16. Harendza S, Lovett DH, Stahl RAK: The Hematopoietic Transcription Factor PU.1 Represses gelatinase a transcription in glomerular mesangial cells. J Biol Chem 2000, 275: 19552–19559
17. Mertens PR, Alfonso-Jaume MA, Steinmann K, Lovett DH: A synergistic interaction of transcription factors AP2 and YB-1 regulates gelatinase A enhancer-dependent transcription. J Biol Chem 1998, 273: 32957–32965
18. Mertens PR, Alfonso-Jaume MA, Steinmann K, Lovett DH: YB-1 regulation of the human and rat gelatinase A genes via similar enhancer elements. J Am Soc Nephrol 1999, 10: 2480–2487
19. Cheng S, Alfonso-Jaume MA, Mertens PR, Lovett DH: Tumour metastasis suppressor, nm23-beta, inhibits gelatinase A transcription by interference with transactivator Y-box protein-1 (YB-1). Biochem J 2002, 366: 807–816
20. En-Nia A, Reisdorff J, Stefanidis I, Floege J, Heinrich PC, Mertens PR: Mesangial cell gelatinase A synthesis is attenuated by oscillating hyperbaric pressure. Biochem J 2002, 362: 693–700
21. Mertens PR, Steinmann K, Alfonso-Jaume MA, En-Nia A, Sun Y, Lovett DH: Combinatorial interactions of p53, AP2 and YB-1 with a single enhancer element regulate gelatinase A expression in neoplastic cells. J Biol Chem 2002, 277: 24875–24882
22. Miyake T, Aoki M, Nakashima H, et al.: Prevention of abdominal aortic aneurysms by simultaneous inhibition of NF[kappa]B and ets using chimeric decoy oligonucleotides in a rabbit model. Gene Ther 2006, 13(8): 695–704

Discussion

Schumpelick: *Is it of importance? With the mesh we construct a wall in front of the hernia, should we be interested in what happens behind this wall?*

Lynen-Jansen: *We heard about the prospect of developing a bio-active mesh. As suggested by Dr. Sarr, mesh modification might be the future. But if we want to create such bio-active meshes we have to understand the biology, and we still do not know the molecular biology for simple polypropylene meshes. This understanding is fundamental for future developments.*

Schumpelick: *Or could it be that the fixation of the mesh in the long run – 20 years – is related to how the collagen is formed behind the mesh?*

Lynen-Jansen: *We want to guide foreign-body reaction. We want to have an impact on the pathophysiology of our individual patient. Therefore we want to optimize mesh materials.*

Fitzgibbons: *There are meshes being developed now. Is there any drug or material that is anywhere near clinical level in this line of research?*

Lynen-Jansen: *We are making a drug coating; Dr. Junge will describe this on Saturday. In my opinion we have to specify our modulations. In 2006 there was a study on gene therapy and they used a rabbit model where the development of aortic aneurism could be prevented by blocking transcription factors and lowering the MMP-2 expression. We have to specify on the cellular level to optimize a complex system such as the foreign-body reaction.*

Fitzgibbons: *But there is nothing on the clinical level?*

Lynen-Jansen: *Not yet.*
Miserez: *We also know that growth factors play a role in bio-active regulation and using new meshes. Firstly, what makes you think that MMP-2 is the way to go and not growth factors? Secondly, if I am correct, there are also some protective MMPs such as MMP-13. Could you elaborate a little on that?*
Lynen-Jansen: *We know the studies on MMP-13 from Dr. Klinge and he found an overexpression of MMP-13 in incisional hernia, too. However, I have no idea about the importance of MMP-13 on the cellular level. But I think that not only growth factors will play an important role, as has been presented by Dr. Rosch. Unfortunately, it is a very complex system. I present a model which allows us to specify MMP-2 regulation. In future, I guess, we have to create a new balance in these processes, but it is evident that if we manipulate on one side, especially via MMP-2, we alter the complex reaction in a different direction.*
Duh: *You are studying the material itself and how it affects the expression of some of these genes. Do you know whether the stress by itself also can change some of the expressions? Because when we fix hernias in addition to putting a mesh in, there are obviously changes in stress in the wound.*
Lynen-Jansen: *No, it is a standardized animal model for the cellular response where we did not investigate mechanical stress.*
Mertens: *When you have hyperbaric stress you can induce this gene very heavily. So every form of cellular stress most likely will have some kind of transduction into this promotor. I would agree with your hypothesis that stress may have an impact.*
Sarr: *All the studies that we do with meshes are abnormal healing. As our body developed we did not develop a healing response to a clean not contaminated incision. We used that as a model but maybe it is the wrong model; 500 years ago there were no clean incisions, only external and dirty trauma. Shouldn't we really be looking at a normal healing response, which is the whole idea behind bioprostheses, and try to let the body engineer a neo-abdominal wall rather than a scar abdominal wall?*
Lynen-Jansen: *I think we have to look at the normal healing because we want to modulate the collagen ratio. I cannot adapt such an animal model to the in vivo situation in the patient. But my knowledge can provide new tools for the modulation of the collagen ratio in the end.*
Franz: *In your model, was there a normal mouse abdominal wall? Not a hernia model?*
Lynen-Jansen: *These were normal mice with an integrated transgene.*
Franz: *I think it will be important eventually to see what you can observe in the situation of a hernia. There are animal models of incisional hernias. Do you have any human data that supports your animal observation?*
Mertens: *This should not be an experimental model for hernia disease. This should exemplify how an animal treats a biomaterial. There are macrophages invading and these macrophages talk to fibroblasts, and the fibroblasts that are close to the mesh respond differently from the others that are far away. So this is a permanent reaction that is going on in every patient that you treat with a mesh. When you want to design a biocompatible mesh, then with this knowledge you now can address this question. If we want to get rid of MMP-2 in macrophages, if we want to target these macrophages and fibroblasts in a cell-specific fashion, then we have to know which factors regulate MMP-2 in these cells. This is the answer that Dr. Lynen has given, she can name these factors. I believe we have some compounds we can use to target these cells in a cell-specific way. This is my understanding of this model.*
Schumpelick: *I would like to conclude from the Swedish data that there is a high recurrence rate according to the different types of operation; there is still a high rate of recurrence in incisional hernia. We learned about the evidence of published and non-published data. I think the overall conclusion is that we still have a problem of hernia recurrence, and we will talk in the coming days about how to prevent this. Today we learned a little about the biology, the collagen, the MMPs and the extracellular matrix in hernia disease.*

Hiatal Hernia

8 Technical Pitfalls and Factors that Promote Recurrence (Small Defects) Following Surgical Treatment of Hiatal Hernia

M.E. Targarona, C. Balague, R. Berindoague, M. Trias

Introduction

The successful development of laparoscopic fundoplication has made it a valid alternative to medical therapy in the treatment of gastro-esophageal reflux. As experience has grown, the laparoscopic approach is now used to treat more complex conditions such as type II (paraesophageal hernia, PEH) or type-III (mixed) hiatal hernia [1]. Results from several series have shown that laparoscopic repair is feasible and safe, in spite of the increased technical difficulty, and its immediate and short-term results are excellent (◘ Table 8.1) [2–13]. However, the incidence of recurrences may be high, reaching 42% in one series [3].

The Problem

Experience over the past 15 years suggests that surgical strategy for the laparoscopic treatment of PEH includes viscera reduction, sac excision, retrogastric crural closure and fundoplication [1, 14, 15]. Pexy of the gastric plicature, abdominal wall gastropexy and gastrostomy are the most controversial technical steps in maintaining the stomach in place in the abdomen. Though controlled comparative trials with the open approach are lacking, the immediate clinical outcome of laparoscopic repair of PEH is highly satisfactory. However, the recurrence rate is higher than expected at midterm follow-up – as high as 42%, when compared with the open approach (◘ Table 8.1), and some authors have suggested that the laparoscopic technique is unsuitable in this setting [3]. Recurrence has been related to several factors [16, 17] – none of which is clearly responsible – but the main reason for failure of the hiatal repair is tension. Treatment of all hernia repairs, such as the Lichtenstein repair or incisional hernia repair is currently tension-free. However, performing a tension-free repair in the hiatus is controversial and technically very demanding due to the oblique situation of the pillars and the difficulty in securing the mesh. Furthermore, in inguinal or ventral hernia repair, the mesh provides passive support to the intra-abdominal viscera, while the hiatus is a complex anatomical structure in which the esophagus moves during respiratory excursion of the diaphragm. Any prosthetic mesh will therefore be in contact with the esophagus, so there is a theoretical risk of esophageal erosion and complications.

Recurrences

Surgical treatment of gastro-esophageal reflux disease (GERD) or PEH may fail due to relapsing symptoms or to true anatomical failure, associated or not to clinical symptoms. This anatomical failure may be the result of a problem with the fundoplication (too tight or broken), or a hiatal recurrence. This chapter deals only with anatomical hiatal recurrence. The incidence of recurrence is variable. Initial experience of fundoplication for

Table 8.1. Recurrence after laparoscopic treatment of PEH in series with systematic radiological control

Author	No.[a]		Recurrence [%]	PEH recurrence	Sliding Symptoms	
Wu [2]	35/38	92%	23%	2	5	35%
Hashemi [3]	21/27	78%	42%	ns	ns	40%
Wiechmann [4]	44/60	73%	7%	3	0	100%
Khaitan [5]	15/25	60%	40%	1	5	50%
Jobe [6]	34/52	65%	32%	8	3	64%
Mattar [7]	32/125	26%	33%	ns	ns	43%
Keidar [8]	ns		15%	0	5	40%
Diaz [9]	66/96	69%	32%	7	14	62%
Targarona [10]	30/37	81%	20%	1	5	50%
Andujar [11]	120/166	72%	28%	6	24	33%
Watson [12]	60/100	60%	30%	5	13	30%
Ferri [13]		91%	23%	ns	ns	

[a]No. of patients with esophagogram

GERD was followed by a 10% recurrence rate, mainly related to difficulty in closure of the hiatus [18]. With current experience, recurrence rates of less than 5% are expected in cases of pure G-E reflux or small type-I sliding hernia. However, the incidence is highly variable in the case of type-II–IV hernias, reaching 42% in one series. Analysis of recurrences shows different patterns of time and form of presentation (see Table 8.1). Immediate postoperative recurrent hernias are usually secondary to total disruption of the hiatal closure with a relapsing PEH. Long-term recurrences may adopt several patterns: complete recurrent PEH, fundoplication migration, or a small sliding hernia, without a clear recurrence of the paraesophageal sac. In the latter subgroup, the incidence of symptoms is variable, and most are identified only by esophagogram. Symptom recurrences should be treated surgically, depending on the severity. However, there is tacit agreement that non-symptomatic recurrences, especially in cases of small sliding hernia, do not require repair. Recurrent hernias of any type should be considered technical failures, although the long-term outcome of asymptomatic recurrent hernias is unknown.

Factors Related to Hiatal Hernia Recurrence

Many factors have been related to hiatal hernia recurrence. They include local or anatomical factors, technical-related factors and functional (patient-related) factors (Table 8.2). Few studies have analyzed the individual responsibility of any of these factors as the definitive cause of recurrence.

Local factors are of paramount importance because the anatomical elements of the hiatus are widely distorted, especially in PEH. Nevertheless, these elements will be needed for the surgical repair (pillars). All the anatomic factors are inter-related. Besides, the size of the hernia and the amount of the herniated stomach are related to the type of hernia, and may be type II, III or IV. All correlate with the size or width of the hiatus, and some paraesophageal hernias may be as large as 10 cm. Consequently, surgical repair of type-I or pure GERD diseases without hernia have a recurrence below 9%, but recurrence after type II–IV is up to 40%.

It is not surprising that another factor favouring recurrence is redo surgery. Re-dissection of a previously operated area logically implies the use of fibrous and

Table 8.2. Results of comparative studies of paraesophageal hernia repair. Laparoscopy vs. laparoscopy + mesh

Author/year	Groups	No.	T. Op	Conv	Follow-up	Recurrence	Comment
Basso [27][a]	Lap	65	78	1.5%	48.3	14%	Ten-free (polypr.)
	Lap-mesh	67	70	0	22.5	0	
Hui [28]	Lap-mesh	12	226	8%	37	0	Se+ pcr + fp (ptfe + polipr.)
	Lap	12	202	ns	37	0	Se+ pcr + fp
Kamolz [29][c]	Lap	100	70	0	12	9%	Pcr + fp
	Lap mesh	100	70	0	12	ns	Pcr + fp + mesh polypr.
Frantzides [30][d]	Lap	36	ns	0	40	22%	Se + pcr + fp
	Lap mesh	36	ns	0	40	0	Se + pcr + fp + ptfe
Granderath [32]	Lap	50	58	0	12	26%	Pcr + fp
	Lap mesh	50	58	0	12	8%	Pcr + fp + mesh polypr

[a+c]Non-randomized, include all types of hiatal hernias. [d]Prospective randomized trial. *Se* sac excision, *Pcr* posterior crural repair, *Fp* fundoplication, *ns* not stated.

scar tissue. The incidence of recurrence is higher and it may occur especially when a recurrent hernia is found in the redo procedure [20].

Another factor related to recurrence is the anatomy of the pillars. The hiatal crura are a fleshy structure without tendinous reinforcement. Standard sutures may cut the muscle, and if the hiatus is particularly wide, when the pillars are approached, the lateral portions of the diaphragm near the crura become tense, especially on the right, and there is a potential risk of disruption.

A second important group of factors which play a relevant role in recurrence are technical aspects. In spite of the success of laparoscopic surgery for gastroesophageal reflux, fundoplication and hiatal dissection should be performed by means of a precise technique which requires advanced laparoscopic surgical skills. In the literature, many of the series of patients undergoing surgery for large hiatal hernias were operated on in the early days of laparoscopic fundoplication, and there is inevitably a steep learning curve with this technique, as demonstrated by the reduction in operative time and associated morbidity as experience is gained. The current technique for the laparoscopic approach is well systematized, and includes stomach reduction, sac excision, esophageal mobilization, hiatus closure and funduplicature. Any variation, pitfall or mishap could be followed by a relapse [1, 14, 15].

One of the key factors for technical success is crural closure, inevitably related with tension. Gentle intraoperative manoeuvers and manipulation are needed to avoid the tearing or rupture of the pillars. Crural closure poses some technical challenges, and in function of the size and shape of the hiatus opening, posterior or anterior stitches to the esophagus or the placement of a mesh may be needed. Such technical options may favour a defective closure of the hiatal passage and facilitate recurrence. The routine use of calibration has been also suggested as a measure to minimize hiatal recurrence [17]. Although it is rare, a short esophagus is another controversial factor, especially in PEH which do not have longstanding esophagitis.

Additional factors which are difficult to evaluate regarding their role in recurrence are the type of knot performed (double knot, square knot, pledgets), the type of knotting technique [intracorporeal, extracorporeal, Endo-StitchTM (Tyco)], and the material used.

Some authors suggest the use of non-resorbable sutures, as they consider that silk-braided string may degrade over time and favour recurrence [21, 22]. Using mesh to reinforce the pillars' approximation may logically help to avoid recurrence. The mesh may be placed by one of several methods and as yet there is no consensus regarding the method of choice. However, mesh placement in this setting continues to be controversial. The hidden side is the number of underreported severe complications secondary to the presence of a mesh near the esophagogastric junction [14, 15].

Functional factors associated with the patient's general condition are sources of complications which should also be taken into consideration. A number of situations, mainly chronic disorders, are associated with episodes of increased intra-abdominal pressure and may have a direct effect on the repaired anatomical area [16, 17]. Another factor that may enhance the effect of these functional stressors is the reduction of postoperative adherences, a well-known characteristic of laparoscopic surgery [12, 18, 23–25]. Obesity, chronic pulmonary disease, constipation or gastro-esophageal symptoms (gagging, belching, retching, hiccuping, vomiting) may promote recurrence. It goes without saying that early or chronic weight lifting is also related to recurrence.

Other Manoeuvers

Additional manoeuvers to secure the stomach in the abdomen in an attempt to reduce recurrence include a range of techniques: pexy of the fundoplication to the diaphragm, pexy of the gastric body to the abdominal wall, gastrostomy and ligamentum teres pexia [1, 14, 15]. Fundoplication itself may have some fixation effect. Some authors consider the Toupet technique may help to avoid recurrence because the posterior placement of the fundus covers the crural closure and fixes it to the diaphragm. However, as yet there are no definitive data from randomized trials to support the routine use of any of these measures.

It is not known whether collagen disorders are related to the appearance of hiatal hernia or favour recurrence, as has been observed in incisional hernia [26].

Analysis of the Factors Responsible for Recurrence

There was little interest in this topic during the prelaparosopic era, as is evident if we compare the number of papers published before or after the description of laparoscopic repair. Besides, there are no well-defined prospective trials analyzing the importance of different factors on the appearance of a recurrence. One major drawback is the failure to stratify patients according to a homogeneous model. Some studies include a variety of criteria (more than 30, 50%, intrathoracic stomach, gastric volvulus) that make comparison difficult. Furthermore, patients' associated medical conditions which may also impair the anatomical outcome are not considered.

Factors related to hernia recurrence are shown in ◘ Tables 8.3 and 8.4. Soper [18] and Filipi's group [17, 23, 24] worked on a group of GORD patients and both found hernia size and diaphragmatic stressors were the main factors related to recurrence. However, Watson's group [12] analyzed the same factors in the case of PEH hernia and found that only age and obesity were predictors for recurrence.

Comparative trials addressing hiatal closure with or without the use of a mesh are few and their methodology has some drawbacks. However, based on the observation of minimal recurrence with the use of a mesh, they add further support to the hypothesis that tension is the reason for failure. Four comparative studies have been published (◘ Table 8.3) [27–30], but only two were prospective and randomized trials. In addition, two of the comparative trials included patients with all types of hiatal hernias, and only one focused on PEH hernia repair. Basso et al. [27] compared simple, tension-free closures using an onlay piece of polypropylene, and divided their personal series chronologically into two parts. Kamolz et al. [29] compared simple closure with a reinforcement procedure that places the stitches over a piece of polypropylene covering the hiatal closure. Neither study was randomized; they were merely comparisons of initial experiences without mesh and more recent experiences with mesh. They also counted hiatal repair of all types, including type-I hernias or pure GERD without hernia. Mesh placement was followed by a lower incidence of recurrences, without specific morbidity.

Frantzides et al. [30] reported their results of a prospective randomized trial comparing simple closure with PTFE onlay reinforcement for PEH hernia repair, in cases with hiatus over 8 cm wide. Recurrences were significantly reduced after mesh placement (20% vs. 0, $p < 0.00$), without long-term sequel after a 40-month

Table 8.3. Multivariate analysis of factors related to hernia recurrence

Author	Soper [18]	Karkalapudi [24]	Aly [12]	Iqbal [23]
Year	1999	2002	2005	2006
N	290	37	100	100
Hernia type	I	I	II	I-II
Predictive factor	Learning group ($p < 0.05$) Vomiting ($p < 0.0001$) Other stressors[a] ($p < 0.001$) Hiatal size ($p < 0.005$)	vomiting ($p < 0.03$) weight lift (< 0.02)	age ($p < 0.05$) obesity ($p < 0.05$)	gagging ($p < 0.005$) belching ($p < 0.02$) hernia size ($p < 0.04$)

[a]Diaphragmatic stressors: cough, sneezing, vomiting, motor vehicle accident, weight lifting.

Table 8.4. Factors related to hiatal hernia recurrence

Type of hernia	– I/II–IV – Size – Primary/secondary – Pillars characteristic – Short esophagus
Technical factors	– Approach: laparoscopic vs. open – Surgical experience – Knots (type, intra-/extracorporeal) – Material – Calibration – Type of suture – Mesh – Redo
Patient condition	– Obesity – Pulmonary disease – Constipation – Symptoms of GERD recurrence – Gagging/belching/retching/hiccoughing – Associated diseases – Weight lifting
Other manoeuvers	– Gastric pexia – Gastrostomy – Mesh – Ligamentum teres pexia

follow up period. Granderath et al. [31] recently showed similar results with satisfactory long-term function, but with only reinforcing the hiatus with a portion of polypropylene mesh.

Discussion and Conclusions

Treatment for PEH and type-III mixed hernias has been a challenge to digestive surgery for the past 30 years. Surgical treatment was an option for a subset of elderly patients, some of whom were particularly frail, and in some cases it was associated to emergencies such as gastric volvulus or gastric incarceration. However, the results from centres with extensive experience showed low morbidity and good long-term outcome after standard open transthoracic or transabdominal approaches, though in most series the results were merely assessed on the basis of the presence or absence of symptoms, without any anatomical (X-ray) evaluation. Available experience shows the efficacy of the laparoscopic approach for treatment of PEH [1]. Although the intra-operative technical difficulty is greater, and although there are no randomized trials comparing it with the open approach to conclusively determine its relative merits, the immediate outcome clearly endorses the use of this minimally invasive approach in a population that is generally at a higher risk than conventional patients with GERD or small type-I hiatal hernia. The large number of series published in recent years (20 series related to

open approach in 33 years, compared with 46 series in 12 years for the laparoscopic approach) bears witness to the success of, and the interest in, the application of laparoscopic techniques in PEH repair.

The most common technical approaches for surgery of PEH include stomach reduction, sac excision and closure of the hiatal defect – on occasion over 8 cm wide – with or without the addition of some type of pexy. The controversy arises after the definitive observation of a variable recurrence rate (up to 42%) when a routine radiological control is conducted. Some authors have suggested that alternative approaches (open or thoracic) may be better for this disease. Arguments put forward to account for this unacceptably high recurrence rate include the learning curve due to the technical difficulty of the procedure, poor technical crural closure, or a short esophagus. The learning curve for a difficult laparoscopic procedure undoubtedly plays a role, and it has been observed in several large series that the recurrence rate falls as surgeons gain experience. The significance of a short esophagus continues to be a controversial issue. It has been considered a potential cause of failure, but most PEH patients do not have advanced GERD disease with esophageal scarring. The need to perform a Collis gastroplasty to lengthen the esophagus varied from 0% to 70% in the series analyzed and as yet there is no clear agreement on whether this technical step is needed during PEH repair.

Clearly, as with other abdominal wall defects, the aim is to achieve adequate closure. In contrast with the accepted standard concept for inguinal or ventral hernia, which is tension-free, the most widely supported approach is to close the hiatus under tension, with the obvious risk of disruption. The rationale for this judgment is that, unlike the abdomen or groin, where repair aims to achieve passive contention, the cardial region – including the hiatus and the GE junction – is a highly dynamic area and anatomical repair is thus justified. However, since PEH repair causes wide-ranging anatomic distortion and the risk of disruption is high, reinforcement with a mesh is a logical forward step. Hiatal closure is occasionally difficult. Surgeons who do not generally favour the placement of mesh in the hiatus are sometimes obliged to use the procedure to correct the gap, either because of the size of the hernia or because it is technically impossible to proceed otherwise.

There are no clear explanations for the differences in outcome after open or laparoscopic approach to PEH. The final results of laparoscopic repair are possibly not as good because the approach is more technically demanding [32]. However, performance of a systematic radiological esophagogram in patients operated by the open approach, including asymptomatic patients, has evidenced a high number of recurrences. Haas et al. [33, 34], for example, found an anatomical recurrence rate of 42% after systematic radiological evaluation. This suggests that the recurrence may also have been high in the open era, but has only become relevant since the laparoscopic revolution and the increased interest in this topic.

One of the main arguments against mesh placement is the emergence of complications, due in the main to visceral erosion, a risk that is intrinsically related to the existence of a foreign body [14, 15]. Based on this rationale, many surgeons contra-indicate routine placement. However, there are clear differences between the placement of a mesh and insertion of an Angelchik device or bands for gastric banding in obese patients. The latter devices are placed directly over the cardia, creating sustained tension and favouring potential erosion. On the other hand, a mesh in the hiatus to reinforce diaphragmatic closure is placed outside the esophagus and direct contact is avoided. Though several severe complications have been reported, the morbidity rate associated with mesh placement is low.

Another controversial point is whether the use of mesh for hiatal repair in PEH should be routine or selective. The local conditions of the hiatus after sac excision may cause results to differ and sometimes, even though the hernia sac is large, the pillars may be of good quality and can be approached without difficulty. Regarding recurrence after laparoscopic repair of PEH, few studies to date have investigated the predictive factors [2, 18, 23, 24] possibly involving anatomical features of the hiatus (such as the size of the gap, tension, diaphragmatic weakness), the type of repair (single stitches, pledget, etc.), additional fixation manoeuvers (Toupet, pexy, gastrostomy, etc.) and patient characteristics (heavy work, constipation, chronic cough, etc.). Some authors recommend a tailored approach, placing a mesh in cases of major risk of recurrence, and this practice seems more advisable in the case of redo operations. However, the final decision whether or not to place a mesh will evidently depend on the experience of the surgeon.

The controversy surrounding recurrence after surgical treatment of hiatus hernia will end when the long-term follow-up of patients in whom a mesh has been placed has been analyzed, and when randomized trials have been performed. These should be designed to resolve the controversial technical aspects regarding the type of mesh to be used, location of the lesion, selective vs. routine use and additional manoeuvers such as pexy and, Collis esophageal lengthening, and the definitive role of diaphragmatic stressors.

References

1. Draaisma WA, Gooszen HG, Tournoij E, Broeders IA. in paraesophageal hernia repair: a review of literature. Surg Endosc 2005, 19: 1300–1308
2. Wu JS, Dunnegan DL, Soper NJ. Clinical and radiologic assessment of laparoscopic paraesophageal hernia repair. Surg Endosc 1999, 13: 497–502
3. Hashemi M, Peters JH, DeMeester TR, et al. Laparoscopic repair of large type III hiatal hernia: objective follow up reveals high recurrence rate. J Am Coll Surg 2000, 190: 553–561
4. Wiechman RJ, Ferguson MK, Naunheim KS, McKesey P, Hazelrigg SJ, Sanntucci TS, Macherey RS, Landrenau RJ. Laparoscopic management of giant paraesophageal herniation. Ann Thorac Surg 2001, 71: 1080–1087
5. Khaitan L, Houston H, Sharp K, Holzman M, Richards W. Laparoscopic paraesophageal hernia repair has an acceptable recurrence rate. Am Surg 2002, 68: 546–551
6. Jobe BA, Aye RW, Deveney CW, Domreis JS, Hill LD. Laparoscopic management of giant type III hiatal hernia and short oesophagus. Objective follow up at three years. J Gastrointest Surg 2002, 6: 181–188
7. Mattar SG, Bowers SP, Galloway KD, Hunter CD, Smith CD. Long-term outcome of laparoscopic fepair of paraesophageal hernia. Surg Endosc 2002, 16: 745–749
8. Keidar A, Szold A. Laparoscopic repair of paraesophageal hernia with selective use of mesh. Surg Laparosc Endosc 2003, 13: 149–154
9. Diaz S, Brunt M, Klingensmith ME, Frisella PM, Soper NJ. Laparoscopic paraesophageal hernia repair, a challenging operation: medium-term outcome of 116patients. J Gastroint Surg 2003, 7: 59–67
10. Targarona EM, Novell J, Vela S, et al. Mid term analysis of safety and quality of life after the laparoscopic repair of paraesophageal hiatal hernia. Surg Endosc 2004, 18: 1045–1050
11. Andujar JJ, Papasavas PK, Birdas T, et al. Laparoscopic repair of large paraesophageal hernia is associated with a low incidence of recurrence and reoperation. Surg Endosc 2004, 18: 444–447
12. Aly A, Munt J, Jamieson GG, Ludemann R, Devitt PG, Watson DI. Repair of large hiatal hernias. Br J Surg 2005, 92: 648–653
13. Ferri LE, Feldman LS, Stanbridge D, Mayrand S, Stein L, Fried GM. Should laparoscopic paraesophageal hernia repair be abandoned in favor of the open approach? Surg Endosc 2005, 19: 4–8
14. Targarona EM, Bendahan G, Balague C, Garriga J, Trias M. in the hiatus: a controversial issue. Arch Surg 2004, 139: 1286–1296
15. Targarona EM, Balague C, Martinez C, Garriga J, Trias M. The massive hiatal hernia: dealing with the defect. Semin Laparosc Surg 2004, 11: 161–169
16. Rice TW. Why antireflux surgery fails. Dig Dis 2000, 18: 43–47
17. Puri V, Kakarlapudi GV, Awad ZT, Filipi CJ. Hiatal hernia recurrence: 2004. Hernia 2004, 8: 311–317
18. Soper NJ, Dunnegan D. fundoplication failure after laparoscopic antireflux surgery. Ann Surg 1999, 229: 669–676
19. Catarci, M, Gentilkeschi, P, Papi, C, Carrara, A, Marrese, R, Gaspari, AL, Grassi, GB. Evidence-Based apparisal of antireflux fundoplication. Ann Surg 2004, 239: 325–337
20. Smith CD, McClusky DA, Rajad MA, Lederman AB, Hunter JG. When fundoplication fails: redo? Ann Surg. 2005, 241: 861–869
21. Greenwald D, Shumway S, Albear P, Gottlieb L. Mechanical comparison of 10 suture materials before and after in vivo incubation. J Surg Res 1994, 56: 372–377
22. Neo EL, Patkin M, Watson DI. Suturing efficiency during hiatal repair for laparoscopic fundoplication. ANZ J Surg 2004, 74: 13–17
23. Iqbal A, Kakarlapudi GV, Awad ZT, et al. Assessment of diaphragmatic stressors as risk factors for symptomatic failure of laparoscopic Nissen fundoplication. J Gastroint Surg 2006, 10: 12–21
24. Kakarlapudi GV, Awad ZT, Haynatzki G, Sampson T, Stroup G, Filipi CJ. The effect of diaphragmatic stressors on recurrent hiatal hernia. Hernia 2002, 6: 163–166
25. Stein HJ, Feussner H, Siewert JR. Failure of antireflux surgery: causes and management strategies. Am J Surg 1996, 171: 36–40
26. Roisch R, Junge K, Knops M, Lynen P, Klinge U, Schumpelick V. Analysis of collagen-interacting proteins in patients with incisional hernias. Langenbecks Arch Surg 2003, 387: 427–432
27. Basso N, DeLeo A, Genco A, Rpsato P, Rea S, Spaziani E, Primavera A. 360º laparoscopic fundoplication with tension free hiatoplasty in the treatment of symptomatic gastroesophageal reflux disease. Surg Endosc 2000, 14: 164–169
28. Hui, TT, David, T, Spyrou, M, Phillips, EH. Mesh crural repair of large paraesophageal hiatal hernias. Am Surg, 2001, 67:1170–4.
29. Kamolz, T, Granderath, FA, Basmmer, T, Pasiut, M, Pointner, R. Dysphagia and quality of life after laparoscopic Nissen funduplication in patients with and without prosthetic reinforcement of the hiatal crura. Surg Endosc, 2002,16:572–7.
30. Frantzides CT, Madan AK, Carlson MA, Stavropoulos GP.. A prospective, randomised trial of laparoscopic polytetrafluoroethylene (PTFE) patch repair vs simple cruroplasty for large hiatal hernia. Arch Surg. 2002, 137:649–52.
31. Granderath FA, Schweiger UM, Kamolz T, Asche KU, Pointner R. Laparoscopic Nissen fundoplication with prosthetic hiatal closure reduces postoperative intrathoracic wrap herniation: preliminary results of a prospective randomized functional and clinical study. Arch Surg 2005, 140: 40–48
32. Oelschlager BK, Pellegrini CA. Paraesophageal hernias: open, laparoscopic, or thoracic repair?. Chest Surg Clin N Am 2001, 11: 589–603
33. Haas O, Rat P, Christophe M, Friedman S, Favre JP. Surgical results of intrathoracic gastric volvulus complicating hiatal hernia. Br J Surg 1990, 77: 1379 1381
34. Low DE, Unger T. Open repair of paraesophageal hernia: reassessment of subjective and objective outcomes. Ann Thorac Surg 2005, 80: 287–294

Discussion

Franzidis: *Prof. Fuchs, you mentioned that, at the end of the procedure or the hiatal hernia repair, the surgeon is always happy with the repair. I would disagree with that. I am often not happy with the primary repair. And I have some parameters where I would say that these patients, if I leave it the way it is, need one blow and then it will fall apart.*

Fuchs: *There is a randomized trial showing that it is helpful for the patient if you use a bougie, especially if you are not very experienced to prevent a long persisting dysphagia. I always advise in courses, that a bogie should be used in order to prevent a persisting dysphagia. I always tend to be happy at the end of the operation, and I am not happy when I try to change what I have done. As a matter of fact, I am pretty often happy. If the condition is bad, then I use a mesh. I shall be happy, when you have finished the study so that we have some data on it.*

Schumpelick: *If you do a normal hernia, stitching together, it doesn't work in an inguinal hernia or an incisional hernia. Should it work here? Why? It is a permanently moving muscle, you stitch it together and rely on that and say that this is hernia repair, and don't talk about reflux disease. I am talking about hernia repair. I will not be certain that this suture repair of the hiatus in the long run is sufficient. Have you any data? In my opinion, we are not treating the hernia.*

Fuchs: *Of course we are not sure. Later in the summary I will show some data on the number of patients that have a migration. You can ask a lot of people doing reflux surgery that having a migration is one of the problems. First of all again, fixing the oesophagus at the diaphragm with all its moving doesn't help. People who have done this, and I did this for a certain period, too, will experience that it becomes loose, because of all the movement and tension that there is. That is not enough. On the other hand, we have to narrow it in order to have at least some kind of resistance there. So the door is not wide open, but we cannot close it, this is our problem. What we at least can do is make sure that the narrowing that we can create during the operation will stay like this. We know from the randomized trials that the recurrence rate was 15%. That was reflux recurrence. We don't know the number of hiatal recurrences from the very few references where this is always documented. I agree with your opinion, that we don't treat the hernia.*

Köckerling: *I agree with Prof. Schumpelick's comment. The recurrences we have seen have always the same appearance. The Nissen fundoplication was intact, but the complete fundoplication slipped back into the thorax and again we have a widening of the hiatus, which is the problem. In my opinion we need a prospective randomized study comparing simple suture reconstruction and a reconstruction using additional mesh material.*

You have mentioned the close anatomical relation between the hiatus and the aorta. One very important step is to really dissect the aorta so that you can grasp enough of the muscle.

Fuchs: *I agree with the second, maybe also with the first comment. I have done two or three stitches in the aorta, and with compression there was never a problem. This can be really a problem for somebody who has no experience. Regarding the first comment, again I must say that I am sure that the meshes do have a role in narrowing the hiatus and making it stable. But, on the other hand, you cannot close this hernia as you can close an incisional or an inguinal hernia because you have a food passage here. If you close it more you will have side effects that the patient will not like. Even if you do a mesh on every patient you will still have a gap that you will need for the oesophageus, and through this gap you will have some kind of recurrence.*

Köckerling: *I tend now to say that the dysphagia we sometimes see in patients is induced more by the Nissen fundoplication and not by the very close suturing of the hiatus. This is our experience. What we do now is make a Toupet fundoplication and close the hiatus very densely with four to five stitches using additional latches. Since we have been doing this, we have never seen a patient with postoperative dysphagia. In my opinion it is more the fundoplication and not the closing of the hiatus.*

Fuchs: *I would disagree to that, because we have done a thousand Nissens. And others who have done more than a thousand Nissen fundoplications have not had this dysphagia as others have.*

Franzidis: *If you review the surgical literature it is not an American problem and not a European problem, it is a world-wide problem. The main reason for recurrence of symptoms in patients with hiatal hernia reflux is disruption of the hiatal hernia. When you claim that you can leave the hiatal defect unrepaired, I think it is a disservice to the patient. What must be done is prevent recurrence of hiatal hernia.*

Fuchs: *I agree completely with you. But you will not be able to do this even if you use a mesh. I have done redos where I found meshes all over the place. It also can create other problems.*

Read: *Dr. Targarona, some of these recurrences occur through the diaphragm itself to do the lateral cross, and they do not herniate through the esophageal hiatus.*

Targarona: *It is clear that hernia is mainly a disease of the elderly. I don't know if that favours the recurrence or*

if the older patients have more comorbidities, or difficult tissues that make solution different. Also it is important to remark that this special group of patients is sometimes frail, which is also to be considered. In order to know that you need a perfect anatomical hernia repair or we can have some tolerance with this. This is also a matter of discussion from the clinical point of view. If the hernia is through the oesphagus or through the lateral pillow I can not answer it really.

Ferzli: *A quick comment on what you have said. I saw your video and your standardization. Do you take a short gas track, because there is a recent paper from Kleiber, who uses the mesh routinely here in Switzerland? And they also don't take a short gas track.*

Targarona: *We take out the short vessels to avoid this for every dysphagia. In these patients it is probably much easier to dissect the sac. My practice now is to pull the stomach to go through the short vessels till the beginning of the sac in the inferior part of the left pillar and then you begin to dissect the sac and you can take it out.*

Fuchs: *There is an interesting discussion based on some randomized trials regarding the division of the short gastrics. If you summarize the four randomized trials that are available you are tempted to say it is not necessary, but it depends also on to what extent you dissect on the right side. If you minimize your dissection on the right side you need something on the left in order to dissect the hiatus. I also mobilize the fundus very posteriorly to make a symmetric wrap, but looking at the evidence from some randomized trials we must confess that the evidence is not clear, or rather controversial.*

Schippers: *I have a comment on technique and a question. You are in favour of placing a tube order to calibrate your fundoplication. I was afraid about this technique, because I had some better experience with our anaesthologists. From that time I switched to doing an intra-operative endoscopy after my procedure. If it is able to pass the hiatus without pushing, I am quite lucky with my operation.*

You mentioned cases of big defects in the diaphragm. With respect to the comment before, that we treat the defect and not the disease, do we really have any evidence-based literature which proves that we have to add a fundoplication after our repair of the defect?

Targarona: *I don't use calibration. I think it is finally not necessary. I am also afraid, because sometimes it can hurt the hiatal oesophagus and it is much more difficult to handle this disruption. With the cutting of the short vessels we can assure a really floppy Nissen.*

The disease is at the hiatus. But we destroy all the paraoesophageal attachment to the oesophagus. And at this moment the most accepted technique is to add a fundoplication.

Schippers: *I was not talking about the defect in the hiatus. I was talking about lateral defects in the diaphragm. Do we have to add a fundoplication in these patients?*

Targarona: *Then you need to put a mesh on the defect.*

Ferzli: *It is very controversial, because we are here as experts. But we are in the area of GIA on the one hand, and we have the experience that we are witnessing in these patients that when we do a band on them, they all get reflux; within a year when the laparoscopic lap bands all have oesophagitis and reflux. Yet when we scope a gastrectomy, they do not have a reflux. When we do the vertical banded gastroplasty with the resection of the upper part of the stomach, which is now the new vertical gastric, these patients have no reflux. My question is, shouldn't we move into a new area of technique where there is no wrap? There is now fear of migration of wrap, maybe there is no need to reconstruct a hiatus which is constantly under motion.*

9 Anatomical Limitations of Surgical Techniques

M. Stumpf, U. Klinge, J. Conze, A. Prescher

Mesh repair for reinforcement of large hiatal hernias is being increasingly used [1]. Guidelines for this indication or publications about standard procedures are still lacking.

The intention of our investigations was to study some anatomical limitations for the usage of mesh repair in the hiatal region. Therefore fixed and fresh-frozen corpses were investigated.

In most publications a dorsal hiatal closure is used for repair of hiatal hernia. This is also the technique used at our clinic; so at first we focused on the question of what size of overlap behind the oesophagus is possible.

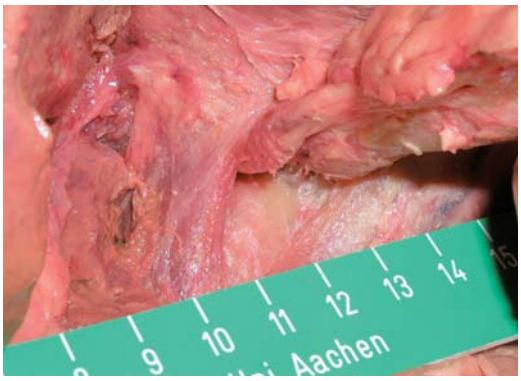


Fig. 9.1. Space between aorta and oesophagus

Due to position of the aorta passing the diaphragm, the posterior space behind the oesophagus is limited. In the case of normal anatomy, the distance between aorta and oesophageal wall is around 2 cm (Fig. 9.1). In the case of a large hiatal hernia, this space may be increased after suture of the left and right crus, but still remains the place of the smallest overlap.

In summary, a big 4–5 cm overlap as claimed in abdominal hernia surgery is not possible to reach [2].

The second interesting aspect concerns the contact between the mesh and the oesophageal wall, when placed behind the oesophagus. Many publications recommend placing the mesh at a distance to the edge of the oesophagus, to prevent direct contact with the prosthetic material. Our anatomical studies revealed a different problem. During the presence of pneumoperitoneum and laparoscopic preparation, an angle between the hiatal crura and the oesophagus is imitated (Fig. 9.2). In a normal and relaxed situation the hiatal crura and therefore the implanted mesh will have broad contact to the posterior oesophageal wall. With implanted retro-oesophageal mesh a broad contact and possible fixation of the oesophagus and therefore potential complications have to be taken in account.

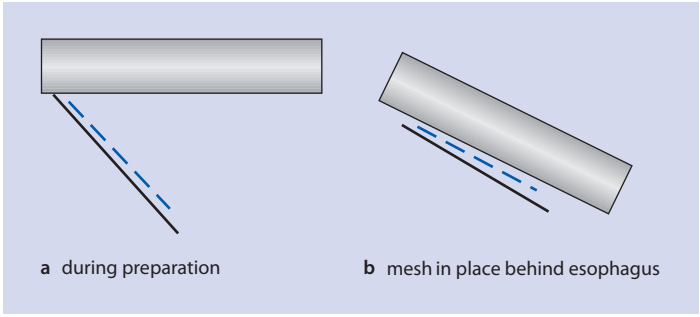


Fig. 9.2. Mesh placed behind the oesophagus

References

1. Granderath FA, Schweiger UM, Kamolz T, Asche KU, Pointner R. Laparoscopic Nissen fundoplication with prosthetic hiatal closure reduces postoperative intrathoracic wrap herniation: preliminary results of a prospective randomized functional and clinical study. Arch Surg 2005;140(1): 40–48
2. Klinge U, Conze J, Krones CJ, Schumpelick V. Incisional hernia: open techniques. World J Surg 2005; 29(8): 1066–1072

Discussion

Deysine: *At the moment I am feeling very humble. I don't know how to repair a hiatus hernia, and only did a couple when I was a resident. But I have been listening to this conference for a long time. With all respect, but it seems to me that we are in the pre-Bassini rea. The complications are terrible and we still suture muscle to muscle, which is something that we don't do anymore anywhere in the body. On top of that, every time we try to place a prosthesis, it may migrate into the oesophagus. I don't see the results coming as fast and as well as for other parts of our science in hernia. The question is, have you tried biological meshes to repair these hernias?*

Fitzgibbons: *We have considered the use of biological prostheses and we use them all, all types. I hear this strong criticism of repairing hiatal hernias, but we have got to realize that in most series the quality of life is remarkable improved, even though there may be recurrence of the hernia. You may have a small sliding hernia or you have a huge para-oesophageal hernia. Almost all studies show 80–90% quality of life in long-term follow-up. So, to think we are not doing any good by closing the crural is ridiculous.*

Pointner: *You have shown that you are talking about 2 or 3 cm. The normality is, that hernias are 4 to 5 cm, and those are the large hernias. We have no contact between the mesh and the oesophagus, because we are doing a wrap and we have contact between the stomach and the mesh. I don't know if it is not necessary to have this contact. I am not sure if we don't need adhesions from the stomach to the mesh. That is another problem.*

Conze: *If you do a fundoplication and wrap around it you don't have contact. But I think we should keep in mind that we are in a situation where there is a lot of mobility. The diaphragm itself moves, DeMeester says it moves 25,000 times a day, so there is lots of mobility. So you have the mesh, you have your adhesions and you have this continuous up and down. I don't know if you really get that much adhesion there; it might even act like a saw.*

N.N.: *I did use this biological mesh, but I have no long experience, for two reasons, so I cannot say anything about the long time. But to manipulate in the laparoscopy is not easy, because it is a material that is too thin and that makes the same problem as if you would use polyester in laparoscopy.*

Kukleta: *I want to make some personal comments on this. Jacobs has used it routinely for several years and seems to be very happy to advocate meshes. But I have completely different information from somebody else, who found an absolute catastrophe, having very long stenosis of the oesophagus which he had to resect. But maybe we can discuss this together with the meshes.*

10 Prevention by Selection?

T. Franzén

Introduction

Fundoplication is the most common surgical treatment for both gastro-oesophageal reflux disease (GORD), with sliding hernia and para-oesophageal hernia.
We must realize that there is no best before date for a fundoplication, and recurrence can appear several years after operation. The long-term outcome after fundoplication depends on several factors such as: the skill of the surgeon, the severity of GORD, the size of the hiatal hernia and the surgical technique.

Background

Mechanism of Action of the Fundoplication

The technical principles of surgical repair remain the same in laparoscopic practice as in conventional transabdominal procedure. Both, total and partial fundoplication, anterior or posterior, work in similar fashions. The principle is to mobilize the lower oesophagus and to wrap the fundus of the stomach around the oesophagus to create a functional valve. As a complement, the crural diaphragm is narrowed with sutures to prevent migration of the wrap up in the chest and to prevent postoperative para-oesophageal herniation.

The goal is to overcompensate the antireflux barrier, which will be done from both an anatomical and a physiological point of view by

- reducing the hiatal hernia, stretching out the oesophagus and repositioning the lower oesophageal sphincter (LOS) into the abdominal positive pressure environment;
- increasing the abdominal length of the LOS in order to increase the resting pressure.

Postoperative Side-Effects After Fundoplication

The most frequent side-effects after fundoplication are solid food dysphagia (temporary or persistent), inability to vomit, decreased ability or inability to belch, epigastric pain, postprandial fullness, increased meteorism and increased flatulence. When performing a fundoplication, especially a total, it is necessary to do the wrap both floppy and short, trying to avoid these side-effects.

As a surgeon you have to consider these side-effects and put them in relation to the durability and efficiency of the fundoplication.

Problem of Recurrence After Fundoplication

The different types of surgical failure after fundoplication are a wrap that is too tight or too loose, incorrectly positioned or disrupted. The failure rate for total fundoplication (360°), at a follow-up interval of 5 years, is 10

to 15%. A number of patients therefore need a second operation because of dysphagia or recurrent heartburn. The indication for re-operation is more urgent for patients with dysphagia than for patients with heartburn, but when slippage occurs dysphagia might also develop as a late symptom.

When there is a suspicion of failure, it is necessary to detect the anatomical deficiencies with endoscopy and/or barium swallow investigations. Hinder et al. [1] have defined the underlying abnormalities responsible for the need for re-operation and various radiological types of failure have been demonstrated:

- Type I represents complete or almost complete disruption of the fundoplication, with recurrence of the hiatal hernia in most cases.
- Type II involves slippage of part of the stomach above the diaphragm. An hourglass defect is created, with part of the stomach above and part below the oesophageal hiatus in the diaphragm. This is frequently caused by the fundoplication having been incorrectly placed around the upper stomach rather than around the oesophagus.
- Type III, so-called slipped Nissen. Part of the stomach lies above and part lies below the fundoplication and may also be associated with an hourglass defect. This may occur as a result of slippage of the stomach through the fundoplication or incorrect placement of the fundoplication around the stomach at the time of surgery.
- Type IV occurs when the intact fundoplication herniates through the oesophageal hiatus into the chest.

Another problem is postoperative para-oesophageal herniation of the stomach into the chest, which is reported to be more common after laparoscopic fundoplication [2]. Acute para-oesophageal hernia should be repaired early to prevent gastric strangulation [3, 4].

Hiatal insufficiency with migration of an intact repair into thorax is reported as the most common complication after laparoscopic fundoplication [4–7]. One explanation is that patients operated on laparoscopically have less postoperative pain and might return to normal activity earlier than patients operated on transabdominally.

Early activity raises the abdominal pressure before adhesions have been established in the hiatal area [7]. When slippage and mechanical failure such as dysphagia is presented, the medical treatment is not sufficient and the patient is in a worse condition than before the operation. A recurrence of a partial fundoplication is probably not so dramatic.

Prevention of Recurrence

It is a fact that patients who really need an operation are the most difficult to repair. To prevent recurrence you have to select patients with mild disease and small hernias, which is not acceptable because they can be well treated with medication. The only way to prevent recurrence is to improve the surgical technique. A common finding at re-operation is that the short vessels of the major curvature are divided except the difficult part, the upper vessels and the peritoneum between the fundus and the diaphragm [8]. When the wrong part of the fundus, or a too low part of the stomach, is used for the valve there is a high risk of including the fat pad at the oesophagogastric junction in the fundoplication. The valve functions perfectly early postoperatively but there is then a high risk of slippage and rupture later on.

To prevent migration of the wrap it is useful to add two extra sutures from the upper part of the fundoplication to the undersurface of the diaphragm [9] in combination with crural repair.

Some authors claim that it is essential to choose an operation that is tailored to the patient's physiology and that a total fundoplication is an absolute contraindication in the presence of a primary oesophageal motility disorder [3, 10, 11]. However, contradictive results are presented in a randomized trial showing no difference in outcome between total and partial fundoplication [12]. In patients with oesophageal stricture and oesophageal shortening the fundoplication can be combined with Collis gastroplasty [13]. This uses the stomach adjacent to the lesser curvature to create a longer tubular oesophagus. The procedure can be done with both open and laparoscopic technique. This is a popular procedure as a redo operation because it is believed that oesophageal shortening is often involved in failure of a fundoplication [1]. In patients with recurrence, despite good fundal mobilization, we must suspect inadequate suturing technique. It is important to take good bites without tearing the tissue. It is also obligatory to use non-absorbable sutures. Maybe it is also important to choose between conventional open surgery and laparoscopy to prevent recurrence. To compare open and laparoscopic total fundoplication, we performed a randomized study in our hospital [14]. Adult patients with hiatal hernia and uncomplicated GORD were included during the years 1994–1998 in this prospective clinical trial. Two senior surgeons well trained in laparoscopic antireflux surgery performed the 45 laparoscopic operations. Forty-eight patients underwent open surgery, performed and su-

pervised by two other senior surgeons well trained in gastro-oesophageal surgery. Early postoperative reflux control was similar for laparoscopic and conventional fundoplication. At long-term follow-up significantly more patients were satisfied after laparotomy (91%) than after laparoscopy (62%). Our findings are in accordance with a questionnaire study in Sweden, concerning antireflux surgery [15]. This found a failure rate of 29% for laparoscopy and 14% for laparotomy 4 years postoperatively.

Outcome of redo fundoplication has a somewhat lower success rate than after first operation, with 79% satisfied patients [16]. The success rate falls to 66% after a third operation and less than 50% after a fourth procedure [17]. These success rates are after primary open fundoplication and open redo surgery.

Laparoscopic redo fundoplication should be carried out only by surgeons with a large experience in laparoscopic antireflux surgery, because of technical difficulties [1].

References

1. Hinder RA, Klingler PJ, Perdikis G, Smith SL (1997) Management of the failed antireflux operation. Surg Clin North Am 77: 1083–1098
2. Seelig MH, Hinder RA, Klingler PJ, Floch NR, Branton SA, Smith SL (1999) Paraesophageal herniation as a complication following laparoscopic antireflux surgery. J Gastrointest Surg 3: 95–99
3. Ferguson MK (1997) Pitfalls and complications of antireflux surgery. Nissen and Collis-Nissen techniques. Chest Surg Clin N Am 7: 489–509
4. Watson DI, Pike GK, Baigrie RJ, Mathew G, Devitt PG, Britten-Jones R, Jamieson GG (1997) Prospective double-blind randomized trial of laparoscopic Nissen fundoplication with division and without division of short gastric vessels. Ann Surg 226: 642–652
5. Granderath FA, Kamolz T, Schweiger UM, Pasiut M, Haas CF, Wykypiel H, Pointner R (2002) Long-term results of laparoscopic antireflux surgery. Surg Endosc 16: 753–757
6. Watson DI, Jamieson GG, Devitt PG, Mitchell PG, Game PA (1995) Paraoesophageal hiatus hernia: an important complication of laparoscopic Nissen fundoplication. Br J Surg 82: 521–523
7. Dallemagne B, Weerts JM, Jehaes C, Markiewicz S (1996) Causes of failures of laparoscopic antireflux operations. Surg Endosc 10: 305–310
8. Franzén T, Johansson K-E (2002) Symptoms and reflux competence in relation to anatomical findings at reoperation after laparoscopic total fundoplication. Eur J Surg 168: 701–706
9. Hunter JG, Swanstrom L, Waring JP (1996) Dysphagia after laparoscopic antireflux surgery. The impact of operative technique. Ann Surg 224: 51–57
10. Fuchs KH et al. (1994) Management of gastroesophageal reflux disease 1995: tailored concept of antireflux operations. Dis Esophagus 7: 250
11. Kauer WK, Peters JH, DeMeester TR, Heimbucher J, Ireland AP, Bremner CG (1995) A tailored approach to antireflux surgery. J Thorac Cardiovasc Surg 110: 141–146; discussion 146–147
12. Rydberg L, Ruth M, Abrahamsson H, Lundell L (1999) Tailoring antireflux surgery: A randomised clinical trial. World J Surg 23: 612–618
13. Collis JL (1957) An operation for hiatus hernia with short oesophagus. Thorax 188: 341–348
14. Franzén T, Anderberg B, Wirén M, Johansson K-E (2005) Long-term outcome is worse after laparoscopic than after conventional Nissen fundoplication. Scand J Gastroenterol 40: 1261–1268
15. Sandbu R, Khamis H, Gustavsson S, Haglund U (2002) Long-term results of antireflux surgery indicate the need for a randomized clinical trial. Br J Surg 89: 225–230
16 Jamieson GG (1993) The results of anti-reflux surgery and reoperative anti-reflux surgery. Gullet 3: 41–45
17. Skinner DB (1992) Surgical management after failed antireflux operations. World J Surg 16: 359–363

Discussion

Ferzli: *In your data about the recurrence, in the laparoscopic group you had six and in the open group you had two; now you show twenty-one. Similar to Dr. Filipi's recent paper again, from Omaha, mostly related to wrap migration and tightness. What did you do for these patients? Could you at least please tell us what the operation was, was it transthoracic, was it open? What operation did you do for this redo, because we know that these redo carry over 10% if not more?*

Franzén: *We did transabdominal open operations in every redo. There is no short oesophagus in any patient, and we use no mesh.*

Fitzgibbons: *Your explanation of the difference between open and laparoscopic is not consistent with the rest of the literature. And later to your point: how many times did you link the oesophagus in the whole series? I didn't hear anything about that. Because if you have 0%, then I expect you have no recurrence because of the short oesophagus. Did the Swedish surgeons not believe in the short oesophagus?*

Franzén: *We believe in it. But in these cases we found no short oesophagus. If we found any short oesophagus on the first operation, they were not included in the study.*

Fuchs: *What is your explanation for these differences between laparoscopic and open?*

Franzén: *We must remember that this was 10 years ago. But I think laparoscopic treatment was the same then today.*

Read: *From your extensive experience, I know you didn't operate difficult cases in your study. With your own understanding today, do you believe that the reflux operation helps the oesophagus or not?*
Franzén: *If you look at the international literature, there was an accumulation of more than 14,000 fundoplications worldwide, and that report has shown 91% of the patients with a high success rate. So how do you explain your low success rate with the laparoscopic approach, when the rest of the world has better results?*
Fuchs: *I cannot explain it. I can only explain it with the lack of skill of the surgeon.*

Redo-Operations Open/Laparoscopically: Change of Technique or Make it Better?

11 The Failed Laparoscopic Hiatal Hernia Repair: "Making it Better" at Redo Operation

S. Dutta

Introduction

Despite over 85 years of experience in its surgical management [1], hiatal hernia remains a tremendous challenge to the gastro-intestinal surgeon. The difficulty in effectively managing this disorder is evident in the large amount of literature devoted to the topic and the myriad of surgical options that are described, including open transabdominal and transthoracic and, more recently, laparoscopic and thoracoscopic approaches. The enthusiasm for laparoscopy and its many potential benefits has made it the standard of care for antireflux surgery [2–4], and there is compelling evidence to suggest that redo surgery is also feasible [5–7]. This enthusiasm has naturally carried over to the surgical management of hiatal hernia, which frequently co-exists with reflux. Laparoscopic hiatal hernia surgery, however, is not yet well established, and concern has been voiced that it may not be a suitable approach for those patients with large hiatal hernia, and for those with recurrent hernia after a primary laparoscopic repair [8].

Concern with laparoscopic hiatal hernia repair stems from outcome studies that suggest high recurrence rates. Although some investigators have found the recurrence rates for laparoscopic repair to be comparable to open approaches [8], the highest quoted recurrence for laparoscopy (42%) [9] by Hashemi and colleagues compared unfavourably to their recurrence with open techniques (15%). Other investigators, using mainly transthoracic techniques, have quoted anatomical recurrence rates as low as 2% [10]. These excellent results from open surgery are attributed to extensive mobilization and surgical lengthening of the shortened esophagus (interestingly these maneuvers can also be achieved with laparoscopy) [11]. Given the potentially high recurrence rates with laparoscopic techniques, the laparoscopic surgeon who is confronted with a recurrent hiatal hernia in a patient who has undergone primary laparoscopic repair is faced with a dilemma: should the redo operation be performed laparoscopically, or should the surgeon abandon this approach in favour of an open operation?

Technically speaking, the components of hiatal hernia repair are essentially the same whether done laparoscopically or through an open incision. The difference is the tools that one uses, and the way those tools are used. Laparoscopy can be seen as an addition to the surgeon's armamentarium, albeit one that requires considerable technical skill; success is dependent on the facility of the surgeon and the limitations of the technology. As laparoscopic technology is continuously improving, based on necessity and capability, the limitations are reduced. In 1965, Gordon Moore, co-founder of Intel Corporation, observed that the number of transistors per square inch of an integrated circuit had doubled yearly since the integrated circuit was invented [12]. Since then, progress has slowed from Moore's prediction; however, this technology continues to double in capacity every 18 months. Technophiles are fond of applying this law to other aspects of technological development. If this is true for laparoscopy, then in time minimal access techniques will accomplish

feats not possible by open surgery. Hints of this eventuality are already emerging in the fields of robotic and translumenal surgery.

Proponents of laparoscopic surgery feel that every aspect of hiatal hernia surgery that can be performed through an open approach can be accomplished using laparoscopic and/or thoracoscopic techniques. Therefore, it is possible through attention to specific details of the operative technique to use laparoscopic tools to effect a secure hiatal hernia repair. This article briefly reviews the classification of hiatal hernia and the nature of its recurrence, discusses the factors that may lead to recurrence after laparoscopic hiatal hernia repair, and proposes strategies that should be employed during the laparoscopic procedure to prevent recurrence.

Classification of Hiatal Hernia and the Nature of Recurrence

Hiatal hernia is composed of a widening of the esophageal hiatus large enough to allow intra-abdominal components of the GI tract to enter into the thoracic cavity. Hernias are classified into three primary types [13], and a fourth type is described for classification purposes [14, 15]. The most common is type 1 (sliding hernia), which generally involves a small hiatal defect with intrathoracic herniation of the gastro-esophageal junction (GEJ) and proximal stomach. These make up 90–95% of all hiatal hernia, and can either be aymptomatic or manifest with gastro-esophageal reflux symptoms. Type 2 (para-esophageal) hernia comprises a larger defect with normal infrahiatal placement of the GEJ but significant herniation of the gastric fundus. These patients are at risk of gastric ulceration with hemorrhage, and gastric volvulus with necrosis and perforation. Prior to these life-threatening sequelae, the hernia may be asymptomatic and many surgeons consider an incidental finding to indicate operative repair. Others believe in a more selective approach [16] in patients who are at poor surgical risk. Type-3 (mixed) hernia exhibits components of both type 1 and 2, and clinically behaves as a paraesophageal hernia. A type-3 herniation that also involves other viscera such as colon, small bowel and liver is referred to as a type 4.

Surgeons may embark on a hiatal hernia repair as a component of an antireflux operation, or for the specific goal of correcting a type-2 or -3 defect. Either way, a fundoplication should be a component of the repair [17–19]. Extensive hiatal and para-esophageal dissection during antireflux surgery can disrupt the integrity of the hiatus and ligamentous fixation of the esophagus, creating a defect that predisposes to herniation. The most common failure pattern for laparoscopic antireflux surgery and hiatal hernia repair is intrathoracic wrap migration (84% of failures) [20, 21], which results from crural repair breakdown [22]. These patients present with dysphagia and/or recurrent reflux. Interestingly, this is not the case for open antireflux surgery, which has a wrap herniation rate of about 22% [20].

Factors Promoting Hiatal Hernia Recurrence

Recurrence of hiatal hernia can be traced back to a number of factors that may have contributed to the failure of the initial operation. These factors are related to the experience of the surgeon, the anatomy and nature of the disease, the comorbidities of the patient and the consequences of a laparoscopic approach. Understanding these factors gives insight into strategies the surgeon can use to maximize success of primary and redo laparoscopic hiatal hernia repair.

The Surgeon

The surgeon who tackles hiatal hernia repair must have considerable experience in esophagogastric surgery in order to expect optimal results. As such, repair of these defects should be performed at specialist centers where critical volumes can be accrued and adequate expertise is present, particularly with laparoscopy. Reviewing the Austrian experience with redo fundoplication, Wykpiel and colleagues [22] demonstrated an inverse relationship between complication rate and experience with fundoplication procedures.

The ability to perform a successful open hiatal hernia repair does not necessarily implicate immediate success when changing to a laparoscopic approach. Laparoscopy requires an entirely novel set of psychomotor skills, and it is commonplace to acquire additional training in advanced laparoscopic surgery through established fellowships or “mini-residencies” [23]. The surgeon who is newly adopting laparoscopy cannot rely on his open surgical skills as a foundation for his learning, and must often “unlearn” or replace his open skills in order to gain laparoscopic facility. Although dissection is essentially the same in laparoscopic and open approaches, the principles of exposure and the techniques of suturing are very different.

Steep learning curves have been demonstrated for a range of minimal access procedures [24–26]. Soper and Dunnegan [27] found that laparoscopic fundoplication failure was significantly higher early in their learning curve, with a rate of 19% in the first 53 patients as compared to 4% in the subsequent 237 patients. Also of note, these surgeons did not routinely mobilize the fundus and repair the crura in the early patients, but subsequently felt this to be an important factor. It can be expected that the learning curve plays a similar, if not greater role, in the success and failure of the technically more difficult laparoscopic hiatal hernia repair. Ferri and colleagues [8] found their recurrence rate after laparoscopic para-esophageal hernia repair to be higher in their first 15 patients (5 recurrences, 33%) as compared to their subsequent 20 patients (2 recurrences, 10%). Overall, the recurrence rate at their institution for open repair was higher (44%) than for laparoscopic repair (23%).

The Disease

A number of anatomical features of hiatal hernia predispose to recurrence after repair. These issues must be acknowledged and addressed by the operating surgeon at the primary repair, and again at redo operation. All hiatal hernias have an accompanying hernia sac extending into the mediastinum. Because the sac is composed of peritoneum, attempting crural repair with the sac intact results in poor healing between the two peritoneal surfaces, leaving a path for recurrence [5]. The sac acts to tether the stomach and esophagus in the mediastinum, impeding reduction into the abdomen and, furthermore, an intact sac can progress to a mediastinal retention cyst which is at risk of infection and mass effect [16].

A second important feature is the size of the hiatal defect. Smaller defects (usually type 1) can be re-approximated with little or no tension. Type-2 and -3 defects tend to be larger, and significant tension is placed on the tissues with primary suture repair [28]. As we have learned from groin hernia surgery, tension in the repair can lead to recurrence. A further complicating factor is that the tissues at the edge of the defect may be attenuated or friable, and sutures may easily tear through.

A final, and critical, anatomical concern is the esophageal length. A number of investigators have identified shortened esophagus as a source of crural repair breakdown [10, 20, 29–31]. Patients with hiatal hernia frequently have severe reflux disease which results in inflammation, fibrosis and consequent shortening of the esophagus. Hernias of types 1 and 3 are most likely to have a shortened esophagus, with the GEJ situated in the mediastinum. A hiatal repair under these conditions experiences tension when the fundoplicated esophagus attempts to re-establish its intrathoracic position, and the repair eventually breaks down, resulting in intrathoracic wrap herniation.

The Patient

The surgeon must recognize pre-operatively the patient-specific factors that can compromise success of hiatal hernia repair. Patients with respiratory disease may have chronic cough which can place great stress on a crural repair through repetitive violent contractions of the diaphragm and severe transient increases in intra-abdominal pressure. Patients with chronic obstructive pulmonary disease or asthma may be on steroid medications which can compromise tissue integrity and healing, further detrimenting the success of a hiatal repair. Furthermore, patients with large paraesophageal hernias are often elderly with poor nutrition and healing ability.

Patients who retch or vomit are also at risk for recurrence, and prone to acute postoperative wrap herniation [5]. These patients can often be identified pre-operatively, and the degree of their retching can increase postoperatively, leading to considerable tension on the repair. Pediatric surgeons are very familiar with this problem in their neurologically impaired patients, many of whom retch and gag as a consequence of poor gastric motility, promoting a higher recurrence rate [32].

The Laparoscopic Approach

Some benefits of the laparoscopic approach can also be a detriment. Laparoscopy results in fewer adhesions than open approaches, and this allows for redo laparoscopic fundoplication. However, the Achilles' heel [20] of laparoscopic fundoplication, intrathoracic wrap herniation, is thought to be due to relatively reduced adhesions posterior to the esophagus where most of these herniations occur [20]. At redo fundoplication, it is common to find multiple adhesions of the liver to the wrap, but much reduced adhesions posteriorly at the crural repair. Laparoscopists must pay particular attention to this area in order to decrease the rate of crural breakdown.

Performing an Effective Laparoscopic Redo Hiatal Hernia Repair

When confronted with a recurrent hiatal hernia following primary laparoscopic repair, addressing the issues outlined above and paying attention to key technical details can help to prevent recurrence and effect a secure redo operation. The surgeon can derive clues from the pre-operative work-up about the technical source of the failure. For example, intrathoracic wrap migration where the GEJ has also relocated above the hiatus most likely represents inadequate esophageal length in addition to crural repair breakdown. Alternatively, if the GEJ is still intra-abdominal but there is herniated fundus, then the culprit is the hiatal repair alone. Adherence to the following principles may help to prevent recurrence after primary laparoscopic repair, or effectively treat it at re-operation.

Traversing the Learning Curve

Laparoscopic repair of large hiatal hernia is clearly an advanced procedure that requires considerable skill and comfort with minimal access surgery. Surgeons should not attempt such a procedure early in their learning curve. Learning laparoscopic surgery should not be viewed as mastery of a successive series of operations, but instead a gradual accumulation and refinement of a repertoire of skills. Eventually, the surgeon masters enough of these skills such that they can be applied to virtually any operation. This comfort level is indicated by a change in the way the surgeon approaches a surgical problem – they begin to think "laparoscopically".

Although the literature discusses specific numbers of cases that must be done of a particular operation before being considered competent, experience with laparoscopic training shows that this can be highly variable. Some surgeons can learn in two operations what it might take another surgeon ten operations to learn. A surgeon well advanced in his repertoire of skills may be quite facile at a novel laparoscopic procedure despite never having performed it before.

Surgeons interested in performing laparoscopic hiatal hernia repair should first make an assessment of their skill level. Skills are best accumulated by first performing operations of lesser technical difficulty such as cholecystectomy and appendectomy. Once working in a laparoscopic environment becomes comfortable, surgeons should accumulate experience with uncomplicated fundoplications, where less difficult intracorporeal suturing can be performed. It may also be useful to use inanimate video box trainers and computer-based virtual reality platforms for purposes of practice [33]. Working with a preceptor who is advanced in the learning curve can also accelerate learning [34]. Laparoscopic hiatal hernia repair should only be performed once a solid foundation is developed in the skills of laparoscopic dissection (blunt, sharp, and thermal), stapling, and suturing.

Redo hiatal hernia surgery requires an even greater degree of skill. In a review of the Austrian experience with redo laparoscopic fundoplication, a six times greater conversion rate as compared to primary laparoscopic fundoplication attested to the difficulty of this procedure. This was due mainly to adhesions between the liver and the stomach and wrap, a factor responsible for the more frequent complications of esophageal and gastric perforations [35].

Preparing the Patient for Surgery

A number of steps can be taken in patient preparation that will maximize chances for a successful operation. Patients with COPD, asthma and other conditions that can lead to chronic cough should have their medical therapy optimized. A concerted effort should be made to control symptoms with nonsteroid medications and steroid use should be minimized. Anesthetic reversal and extubation should be performed carefully to prevent violent coughing, and consideration given to prolonged intubation to allow for a slow, easy wean off the ventilator. Patients with a history of retching and vomiting should be agressively treated peri-operatively with anti-emetics. Finally, nutritional maximization should be instituted prior to surgery.

Operative Strategy

To minimize hiatal hernia recurrence, special attention must be paid to a number of key aspects of the laparoscopic procedure that address the disease-specific problems discussed above. These recommendations pertain both to the primary operation and redo procedures.

Hernia Content Reduction, Sac Mobilization, and Sac Resection

After mobilization of the fundal wrap off adjacent structures, it is necessary to reduce the hernia contents. In redo operations, it is important to first dissect

the often dense adhesions between the wrap and the liver. This is best accomplished by following the liver surface, to avoid gastric perforation, and a lighted bougie in the stomach may be helpful. The herniation of the stomach typically occurs posteriorly and, depending on the type of hernia, there may be varying degrees of stomach and other viscera involved. All hernia content must be completely reduced and adhesive attachments to the mediastinum must be released, otherwise there is tension on the reduced structures and a tendency to reherniate. Moving these structures away from the area also greatly helps in exposure when repairing the hernial defect.

The hernia sac should then be completely excised. This is done by incising the sac circumferentially around the edge of the defect. Continuous traction is applied while the sac is reduced from the mediastinum and loose adhesions are transected bluntly or with a thermal energy source. The entire sac should be removed, en bloc if possible, so as not to leave sac remnants.

Assess and Address Esophageal Length

Because the esophagus is a dynamic structure that contracts and extends in relation to the hiatus, it is difficult to estimate its length using endoscopy or esophagogram, and only approximations can be made with these modalities. Patients with severe esophagitis or stricture, Barrett's disease, para-esophageal hernia, and those undergoing redo surgery have a higher likelihood of foreshortened esophagus [36]. More accurate assessment of esophageal length is made intra-operatively with the laparoscope. Specifically, it is important to have more than 2.5 cm [36, 37] of esophagus sitting without tension below the hiatus. This ensures that there will be no upward tension by the fundoplication on the hiatal repair.

Inadequate esophageal length can in most cases be addressed laparoscopically by generous mobilization of the mediastinal esophagus [30, 31, 38]. Care must be taken to not induce pneumothoraces by avoiding violation of the pleura. In severe situations, mediastinal mobilization is insufficient and an esophageal lengthening procedure must be utilized. This has been done both laparoscopically [39] and thoracoscopically [40], and both are technically challenging. Lengthening typically takes the form of a Collis gastroplasty in which a circular stapling device is used to make a defect on the fundus from which a second linear stapler is fired toward the angle of His adjacent to a bougie placed in the esophagus to tubularize the proximal stomach. More recently, surgeons experienced in these procedures have recommended the use of a simpler wedge gastroplasty [41]. This technique is more easily done using conventional roticulating endoscopic staplers, and involves an initial transverse staple fire across the fundus followed by an inferior to superior staple fire parallel to the left side of the esophagus. A wedge of fundus is removed, while tubularizing the proximal stomach. The lengthened portion of the esophagus can then be fundoplicated, and care must be taken to incorporate the superior-most fundoplication sutures into normal esophagus in order to prevent an obstructive effect.

The Large Hiatal Defect – Mesh or No Mesh?

Some investigators define a large hiatal defect as those greater than 4–5 cm in diameter [16], and others consider 8 cm [42] as the cutoff. In general, a large defect is one that cannot be closed primarily without excessive tension. This latter definition, although subjective, accounts for the quality of the tissues that are being re-approximated. The problem with tension is that sutures ultimately tear through, particularly with diaphragmatic contraction, and friable tissues are more prone to this. Poor tissue integrity is seen in the elderly, malnourished, and those on corticosteroids. It is important also to note that the magnified view provided by the laparoscope may lead the surgeon to get insufficient crural purchase during suturing of the hiatus, and that bigger bites should be taken to compensate for this. Magnification may also lead the surgeon to overestimate the size of a defect, and so more objective means of measurement should be employed.

When conditions exist that are not ideal for primary closure, the surgeon must consider alternate forms of tension-free repair such as with mesh. Many surgeons choose to avoid mesh due to concerns over erosion, infection and stricture; however, when a large hiatal defect recurs, mesh repair should be seriously considered at redo operation. In addition to reducing tension, the mesh most likely perpetuates robust adhesions posterior to the esophagus that are otherwise lacking with a laparoscopic approach. These adhesions bolster the hiatal closure and secure the wrap in the abdomen.

Mesh can be used in two ways. One approach is to suture the mesh patch to the edges of the defect without re-approximating the crura [14]. This is the purest form of tension-free repair of the hiatal defect. The concern, however, is that the mesh cannot be anchored to the esophagus at its anterior border, thereby leaving a po-

tential defect. To avoid this, the mesh must abut the esophagus in order to minimize the defect. The esophagus is a dynamic structure that moves in a vertical plane relative to the crura, and the resultant chronic abrasion can result in mesh erosion into the esophagus.

As a remedy to this situation, a more popular approach is to re-approximate the crura primarily with suture, then place a mesh onlay that is anchored to both crura [43]. Although some authors describe creating a horseshoe- or ovoid-shaped [16] mesh that encircles the esophagus, there are concerns over mesh shrinkage and consequent esophageal stricture [44], hence a rectangular mesh situated posterior to and away from the esophagus may be a better option. The mesh onlay distributes tension more evenly, hopefully reducing the chance of tissue tear at any one place. Once again, the mesh must not abut the esophagus to avoid erosion, and some surgeons recommend placing it such that it abuts the fundal wrap [45]. Some authors advocate an A-shaped mesh as optimal, based on studies of crural mechanics [14, 46].

There has been excellent success reported with mesh cruruplasty using both polypropylene and expandable polytetrafluorethylyne (ePTFE; DualMesh, Gore-Tex; W.L. Gore & Associates, Flagstaff, AR). Granderath and colleagues [45] recently reported their experience in 100 consecutive fundoplication patients that were randomized to crural closure with and without polypropylene mesh overlay. A rectangular piece of mesh was secured with suture to both crura after primary suture closure. Postoperative intrathoracic wrap migration on fluoroscopy was significantly lower in the mesh group than the non-mesh group (8 versus 26%). In an earlier study, Frantzides and colleagues randomized a total of 72 hiatal hernia/reflux patients to ePTFE mesh onlay or no mesh. An ovoid piece of mesh with a "keyhole" was situated around the esophagus and secured with tacks. In a follow-up ranging from 6 months to 6 years, there were 8 (22%) recurrences in the non-mesh group versus none in the mesh group [43].

A distinction must be made between polytetrafluoroethylene (PTFE) and expanded polytetrafluoroethylene (ePTFE). There is a tendency in the surgical literature to incorrectly refer to ePTFE as PTFE. Strictly speaking, PTFE, also known as Teflon (Dupont, Wilmington, Delaware), is frequently the material of which pledgets are made and it has also been used as a mesh onlay in the past. In distinction, ePTFE (expanded polytetrafluoroethylene) is a processed form of PTFE that is microporous and has unique mechanical properties.

Concern with mesh repair as a potential source for fistulization and esophageal erosion arises from reports of polypropylene mesh erosion into the esophagus [47, 48] and PTFE into the stomach [49] after para-esophageal hernia repair. There is one report in the literature of Teflon pledgets used in hiatal hernia repair fistulizing to the esophagus [50]. In addition, I recently operated on a 12-year-old neurologically impaired patient who had had mesh repair of a hiatal hernia as a 3-year-old using a PTFE (Teflon) patch. The mesh had almost completely eroded into the esophagus, causing obstruction, and was successfully removed using a laparoscopic transgastric approach (narrowly avoiding a much more involved operation such as esophagogastric resection) [53].

Although ePTFE is safely used for other diaphragmatic defects such as Bochdalek and Morgagni hernias, the hiatus represents a unique situation where the esophagus may be exposed to chronic abrasion against the mesh. Since PTFE (in Teflon mesh form) clearly can erode into the esophagus, ePTFE (Gore DualMesh) should also be held in suspicion for erosion as an eventuality. Nevertheless, when the surgeon is left with no choice but to use mesh for the hiatal repair, ePTFE is most likely the best compromise [37].

Hopefully future development of biological meshes, such as those derived from porcine intestinal submucosa [48, 51], will obviate the need for prosthetic materials and eliminate the concern over erosion.

Conclusion

Although there are few randomized prospective data to make definitive conclusions, proponents of minimal access esophagogastric surgery assert that proper use of laparoscopic tools and approach can result in effective repair of hiatal hernia with minimal recurrence [52]. These success rates are subject to the experience of the surgeon, the nature of the disease and the comorbidities of these complex patients. Attention to these issues, with specific modifications in peri-operative care and technical approach, should minimize their negative effects. Recurrence rates can be further expected to decline with introduction of improved laparoscopic technologies and less troublesome biologically derived prostheses. Patients with hiatal hernia, particularly the fragile elderly, greatly benefit from a minimal access approach. For this reason alone, surgeons should focus their efforts on maximizing the success of laparoscopic hiatal hernia repair rather than abandoning it.

References

1. Stylopoulos N, Rattner DW. The history of hiatal hernia surgery: from Bowditch to laparoscopy. Ann Surg 2005; 241(1):185–193
2. Anvari M, Allen C. Five-year comprehensive outcomes evaluation in 181 patients after laparoscopic Nissen fundoplication. J Am Coll Surg 2003; 196(1):51 7; discussion 57 8; author reply 58–59
3. Peters JH, DeMeester TR, Crookes P, et al. The treatment of gastroesophageal reflux disease with laparoscopic Nissen fundoplication: prospective evaluation of 100 patients with “typical” symptoms. Ann Surg 1998; 228(1): 40–50
4. Bammer T, Hinder RA, Klaus A, Klingler PJ. Five- to eight-year outcome of the first laparoscopic Nissen fundoplications. J Gastrointest Surg 2001; 5(1):42–48
5. Dutta S, Bamehriz F, Boghossian T, et al. Outcome of laparoscopic redo fundoplication. Surg Endosc 2004; 18(3):440–443
6. Granderath FA, Kamolz T, Schweiger UM, Pointner R. Long-term follow-up after laparoscopic refundoplication for failed antireflux surgery: quality of life, symptomatic outcome, and patient satisfaction. J Gastrointest Surg 2002; 6(6): 812–818
7. Smith CD, McClusky DA, Rajad MA, et al. When fundoplication fails: redo? Ann Surg 2005; 241(6):861 9; discussion 869–871
8. Ferri LE, Feldman LS, Stanbridge D, et al. Should laparoscopic paraesophageal hernia repair be abandoned in favor of the open approach? Surg Endosc 2005; 19(1):4–8
9. Hashemi M, Peters JH, DeMeester TR, et al. Laparoscopic repair of large type III hiatal hernia: objective followup reveals high recurrence rate. J Am Coll Surg 2000; 190(5):553 60; discussion 560–561
10. Maziak DE, Todd TR, Pearson FG. Massive hiatus hernia: evaluation and surgical management. J Thorac Cardiovasc Surg 1998; 115(1):53 60; discussion 61–62
11. Luketich JD, Grondin SC, Pearson FG. Minimally invasive approaches to acquired shortening of the esophagus: laparoscopic Collis-Nissen gastroplasty. Semin Thorac Cardiovasc Surg 2000; 12(3):173–178
12. Vosburgh KG, Newbower RS. Moore’s Law, disruptive technologies, and the clinician. Stud Health Technol Inform 2002; 85: 8–13
13. Hill LD. Incarcerated paraesophageal hernia. A surgical emergency. Am J Surg 1973; 126(2): 286–291
14. Casaccia M, Torelli P, Panaro F, et al. Laparoscopic tension-free repair of large paraesophageal hiatal hernias with a composite A-shaped mesh: two-year follow-up. J Laparoendosc Adv Surg Tech A 2005; 15(3): 279–284.
15. Hill LD, Tobias JA. Paraesophageal hernia. Arch Surg 1968; 96(5): 735–744
16. Keidar A, Szold A. Laparoscopic repair of paraesophageal hernia with selective use of mesh. Surg Laparosc Endosc Percutan Tech 2003; 13(3): 149–154
17. Swanstrom LL, Jobe BA, Kinzie LR, Horvath KD. Esophageal motility and outcomes following laparoscopic paraesophageal hernia repair and fundoplication. Am J Surg 1999; 177(5): 359–363
18. Oelschlager BK, Pellegrini CA. Paraesophageal hernias: open, laparoscopic, or thoracic repair? Chest Surg Clin N Am 2001; 11(3): 589–603
19. Lal DR, Pellegrini CA, Oelschlager BK. Laparoscopic repair of paraesophageal hernia. Surg Clin North Am 2005; 85(1): 105–118
20. Hunter JG, Smith CD, Branum GD, et al. Laparoscopic fundoplication failures: patterns of failure and response to fundoplication revision. Ann Surg 1999; 230(4): 595–604; discussion 604–606
21. Frantzides CT, Carlson MA. Laparoscopic redo Nissen fundoplication. J Laparoendosc Adv Surg Tech A 1997; 7(4): 235–239
22. Wykypiel H, Kamolz T, Steiner P, et al. Austrian experiences with redo antireflux surgery. Surg Endosc 2005; 19(10):1315–1319
23. Chou DS, Abdelshehid CS, Uribe CA, et al. Initial impact of a dedicated postgraduate laparoscopic mini-residency on clinical practice patterns. J Endourol 2005; 19(3): 360–365
24. Voitk AJ, Tsao SG, Ignatius S. The tail of the learning curve for laparoscopic cholecystectomy. Am J Surg 2001; 182(3): 250–253
25. Watson DI, Baigrie RJ, Jamieson GG. A learning curve for laparoscopic fundoplication. Definable, avoidable, or a waste of time? Ann Surg 1996; 224(2): 198–203
26. Ferguson GG, Ames CD, Weld KJ, et al. Prospective evaluation of learning curve for laparoscopic radical prostatectomy: identification of factors improving operative times. Urology 2005; 66(4): 840–844
27. Soper NJ, Dunnegan D. Anatomic fundoplication failure after laparoscopic antireflux surgery. Ann Surg 1999; 229(5): 669–676; discussion 676–677
28. Gryska PV, Vernon JK. Tension-free repair of hiatal hernia during laparoscopic fundoplication: a ten-year experience. Hernia 2005; 9(2): 150–155
29. Pearson FG, Langer B, Henderson RD. Gastroplasty and Belsey hiatus hernia repair. An operation for the management of peptic stricture with acquired short esophagus. J Thorac Cardiovasc Surg 1971; 61(1): 50–63
30. Gastal OL, Hagen JA, Peters JH, et al. Short esophagus: analysis of predictors and clinical implications. Arch Surg 1999; 134(6): 633–636; discussion 637–638
31. Horvath KD, Swanstrom LL, Jobe BA. The short esophagus: pathophysiology, incidence, presentation, and treatment in the era of laparoscopic antireflux surgery. Ann Surg 2000; 232(5): 630–640
32. Pearl RH, Robie DK, Ein SH, et al. Complications of gastroesophageal antireflux surgery in neurologically impaired versus neurologically normal children. J Pediatr Surg 1990; 25(11):1169–1173
33. Hamilton EC, Scott DJ, Fleming JB, et al. Comparison of video trainer and virtual reality training systems on acquisition of laparoscopic skills. Surg Endosc 2002; 16(3):406–411
34. Heniford BT, Backus CL, Matthews BD, et al. Optimal teaching environment for laparoscopic splenectomy. Am J Surg 2001; 181(3):226–230
35. Neuhauser B, Hinder RA. Laparoscopic reoperation after failed antireflux surgery. Semin Laparosc Surg 2001; 8(4):281–286

36. Urbach DR, Khajanchee YS, Glasgow RE, et al. Preoperative determinants of an esophageal lengthening procedure in laparoscopic antireflux surgery. Surg Endosc 2001; 15(12):1408–1412
37. Granderath FA, Carlson MA, Champion JK, et al. Prosthetic closure of the esophageal hiatus in large hiatal hernia repair and laparoscopic antireflux surgery. Surg Endosc 2006; 20(3): 367–379
38. O'Rourke RW, Khajanchee YS, Urbach DR, et al. Extended transmediastinal dissection: an alternative to gastroplasty for short esophagus. Arch Surg 2003; 138(7): 735–740
39. Johnson AB, Oddsdottir M, Hunter JG. Laparoscopic Collis gastroplasty and Nissen fundoplication. A new technique for the management of esophageal foreshortening. Surg Endosc 1998; 12(8):1055–1060
40. Swanstrom LL, Marcus DR, Galloway GQ. Laparoscopic Collis gastroplasty is the treatment of choice for the shortened esophagus. Am J Surg 1996; 171(5):477–481
41. Terry ML, Vernon A, Hunter JG. Stapled-wedge Collis gastroplasty for the shortened esophagus. Am J Surg 2004; 188(2):195–199
42. Frantzides CT, Carlson MA. Prosthetic reinforcement of posterior cruroplasty during laparoscopic hiatal herniorrhaphy. Surg Endosc 1997; 11(7):769–771
43. Frantzides CT, Madan AK, Carlson MA, Stavropoulos GP. A prospective, randomized trial of laparoscopic polytetrafluoroethylene (PTFE) patch repair vs simple cruroplasty for large hiatal hernia. Arch Surg 2002; 137(6):649–652
44. Scholz S, Richards WO. Thoughts on the implantation of prosthetic material at the esophageal hiatus in children. Pediatr Surg Int 2003; 19(1 2):131–132
45. Granderath FA, Schweiger UM, Kamolz T, et al. Laparoscopic Nissen fundoplication with prosthetic hiatal closure reduces postoperative intrathoracic wrap herniation: preliminary results of a prospective randomized functional and clinical study. Arch Surg 2005; 140(1):40–48
46. Casaccia M, Torelli P, Panaro F, et al. Laparoscopic physiological hiatoplasty for hiatal hernia: new composite "A"-shaped mesh. Physical and geometrical analysis and preliminary clinical results. Surg Endosc 2002; 16(10):1441–1445
47. Trus TL, Bax T, Richardson WS, et al. Complications of Laparoscopic Paraesophageal Hernia Repair. J Gastrointest Surg 1997; 1(3):221–228
48. Edelman DS. Laparoscopic herniorrhaphy with porcine small intestinal submucosa: a preliminary study. JsIs 2002; 6(3): 203–205
49. Coluccio G, Ponzio S, Ambu V, et al. [Dislocation into the cardial lumen of a PTFE prosthesis used in the treatment of voluminous hiatal sliding hernia, A case report]. Minerva Chir 2000; 55(5): 341–345
50. Baladas HG, Smith GS, Richardson MA, et al. Esophagogastric fistula secondary to teflon pledget: a rare complication following laparoscopic fundoplication. Dis Esophagus 2000; 13(1): 72–74
51. Oelschlager BK, Barreca M, Chang L, Pellegrini CA. The use of small intestine submucosa in the repair of paraesophageal hernias: initial observations of a new technique. Am J Surg 2003; 186(1): 4–8
52. Draaisma WA, Gooszen HG, Tournoij E, Broeders IA. Controversies in paraesophageal hernia repair: a review of literature. Surg Endosc 2005; 19(10): 1300–1308
53. Dutta S. Prosthetic esophageal erosion after mesh hiatoplasty in a child, removed by transabdominal endogastric urgery. J Pediatr Surg 2007; 42(1): 252–256

Discussion

Read: *I would like to point out that you mentioned chronic cough and maybe you mentioned smoking. But smoking not only causes chronic cough, but is chronic and a big comorbidity. For decades, because I have been around for decades, I have heard hernia surgeons or general surgeons talking about chronic cough. In other words, it is a mechanical problem. The cough and the increased abdominal pressure blow the repair apart. I have heard that for so long, that now, when you present this work again, with that slide, you need to give not only chronic cough but smoking as comorbidity.*

Dutta: *Thank you for the kind comments. I agree with you that there are two aspects to smoking as a causal factor, the chronic cough and the systemic effect on tissue integrity.*

Franzidis: *I would like to congratulate you on this excellent presentation. I would agree with you, when you have finished with the challenge of the redo, there are so many factors that play a role, the adhesions with the left lobe of the liver and so on. I wonder sometimes if you are clear on the anatomy, because many times no matter how many positions you have done, the anatomy is not clear. What we have used is a light and bougie. I know that it is controversial to introduce anything, especially if the device is introduced by an anesthesiologist. But if you have someone who can do this, he can clearly find the anatomy in the gastro-oesophageal junction, so that it is clear where you have mobilization of the oesophagus. Also, when you are dealing with a young child, obviously there is going to be a growth of tissue and the oesophagus is going to become larger; do you account for that when you place the mesh? I feel a little bit uncomfortable with the idea of placing a mesh on a child.*

Dutta: *I like the idea of the bougie. I have not talked about that, because my approach is just to stick onto the liver. Liver bleeding always stops on its own or with some pressure. With reference to your second question, I do worry about that, but once again, it is a compromise, and a judgment. With congenital diaphragm hernia we sometimes use Gore-Tex mesh to close, sometimes we use a muscle flap. When we use a mesh we see later on in life that these children have an indentation of the rib cage on the left side, where we*

typically have the mesh. So yes, there is some issue with growth.

LeBlanc: *Do you have any comment on or experience with the incision in the diaphragm and putting a mesh there and taking the tension of?*

Dutta: *No, but I have read about it. There is something intuitively that I worry about by making a hole somewhere, where there was no hole. But I understand the concept, that it is a relaxing incision that has been done for hernia surgery and groin hernia surgery. My answer is, personally I would say that I would not do that, because of making another hole where I didn't have one before.*

LeBlanc: *It is just an option.*

12 Change of Technique: With or Without Mesh?

R. Pointner, F.A. Granderath

Introduction

Despite increasing experience with laparoscopic sliding or para-oesophageal hernia repair, authors are continuing to report recurrence rates between 30 and 40% with simple primary suture repair of the hiatus [1–3]. This high recurrence rate is also documented for the open approach in long-term follow-up series [4]. As there is a paradigm shift in the repair of inguinal and ventral hernias, discussion arises also for the hiatus, whether to close it by simple suture technique, tension-free, or by the use of meshes. The experience during the past 10 years suggests that the most important technical steps for maintaining the stomach in place in the abdomen are visceral reduction and sac excision, fundoplication and crural closure [5]. Whereas there is wide agreement concerning sac excision and fundoplication, controversy exists about the technique to close the crura. There are no exact data available as to why hiatal hernias recur. The tension on the crura, the diameter of the hiatus, the anatomy of the pillars and the intra-abdominal pressure of the patient are suggested as the main reasons for the failure of hiatal repair. As simple sutures seem to be unable to restore the hiatal anatomy for a long time and cannot provide a tension-free repair, attention is being paid by a few surgeons to the use of prosthetic material for repair or re-inforcement of the hiatus. There are only two randomized trials [6, 7] comparing simple suture techniques to mesh techniques, demonstrating extremely low recurrence rates for the mesh techniques compared to simple sutures. The concept of using prosthetic meshes is based on the lessening of tension on the hiatal crura or the reinforcement of simple sutured crura to prevent postoperative hiatal disruption. Since the first description of prosthetic hiatal closure by Kuster and Gilroy [8] in 1993, a number of techniques has been published. There has been debate regarding the shape, material and the placement of the mesh, and especially whether a prosthetic hiatal reinforcement has to be tension-free. Additionally, there is no agreement regarding the question of selective versus routine use of mesh. Some authors recommend the routine use of prosthetic mesh in order to prevent tension on the hiatal crura and therefore decrease hiatal hernia recurrence. Other authors use mesh selectively – for example in patients in whom a sufficient tension-free hiatal closure cannot be achieved with simple sutures. For some authors, the indication for reinforcement of the hiatal crura with prosthetic material depends on the size of the hiatal defect.

Methods

1. A search of electronic databases was performed to identify available articles regarding prosthetic hiatal closure for hiatal hernia repair. Feasibility, safety and complications related to the use of meshes for hiatal closure as well as recurrence rates were reviewed and compared.
2. Additionally, our own patient material was followed up:

- Thirty-three patients presenting with recurrent large hiatal hernia underwent prosthetic hiatal closure with a circular polypropylene mesh. The mesh was cut from a larger sheet of mesh, cutting a circular defect of 3 cm as a keyhole in the centre. The patch was applied as an onlay to the suture diaphragmatic repair and was anchored in place with a laparoscopic hernia stapler. Out of 33 patients, who were all controlled radiologically by barium X-ray, 24 patients had a follow-up time of 60 months or more.
- Between 11/2003 and 02/2005 in 15 patients with a large hiatal hernia without any possibility of approximating the crura by simple sutures, a tension-free procedure, using a composite PTFE mesh (BARD Crurasoft) was performed. This special V-shaped mesh was fixed with interrupted sutures on the edges of the mesh and secured with staples on the lateral side of the mesh. An X-ray control was performed in all these patients in December 2005.
- Between 10/2003 and 12/2005 a mesh onlay procedure was performed in 20 patients with a large hiatal hernia and weak crura using a dual mesh (Parietex). This mesh has a three dimensional weave of polyester on one side with a hydrophilic collagen material on the other. With the specially designed U-shape of the mesh it can specifically be used as an additional reinforcement of primary sutured hiatal crura and is secured to the diaphragm with a hernia stapler. In all these patients an X-ray control was performed in December 2005.

12

Results

Review of the Literature

Kuster and Gilroy [8] were the first to report on tension-free anterior repair of a hiatal defect. In six patients with large para-oesophageal hernias, a non-absorbable polyester fibre mesh was placed on the hiatus as an anterior onlay patch, overlapping the hiatal crura approximately 2 cm in all directions and securing the crural edges with staples. No intra-operative or postoperative mesh-related complications occurred during a follow-up period of 8–22 months and X-ray showed no evidence of postoperative hernia recurrence. A similar technique was used by Paul [9] with a 5×10 cm PTFE mesh in three patients, showing no complications and no hernia recurrences for a mean follow-up period of 10 months.

In a series of 65 patients who underwent simple sutured hiatal closure, Basso et al. [10] experienced a hiatal hernia recurrence rate of 13.8% during a mean follow-up of 48.3 months. After reviewing the video tapes of these patients, it became clear that the crural sutures were under tension leading to hiatal disruption and intrathoracic migration of the fundic wrap. Due to these findings, Basso et al. began using a 3×4-cm polypropylene mesh for posterior hiatal reinforcement. The mesh was secured with staples as a tension-free hiatoplasty. This technique was used in a subsequent group of 67 patients who underwent Nissen fundoplication for GERD. During a mean follow-up of 22.5 months, there were no complications related to the prosthetic mesh and no hiatal hernia recurrence.

Champion et al. [11] preferred a prosthetic reinforcement of primarily sutured crura. After placing interrupted permanent sutures posteriorly to the esophagus, a 3×5-cm polypropylene mesh was placed as an onlay prostheses and then fixed with a hernia stapler along the crural edges. This technique was performed in 52 consecutive patients with symptomatic GERD and a large hiatal hernia. During a mean postoperative follow-up of 25 months, only one patient developed a postoperative intrathoracic wrap migration. No mesh migrations or visceral erosion occurred in this series of patients.

Keidar and Szold [12] used a circular mesh with a shape similar to that used by Frantzides and Carlson. Out of a sample of 33 patients, 10 patients with large para-oesophageal hernias underwent laparoscopic prosthetic hiatal repair. The simple cruroplasty was then reinforced with a polypropylene mesh. The mesh was precut to an oval sheet, placed around the esophagus and fixed to the diaphragm using a hernia stapler. During a follow-up of 46–76 months, the satisfaction score was good to excellent for the majority of patients. Only one of the mesh-repaired patients developed a hiatal hernia recurrence compared to four patients who underwent repair without mesh. No complications related to the use of the mesh were seen in this study. To increase the theoretical safety of the procedure, they began using a preformed composite mesh with polyester on one side and a hydrophilic collagen material on the other. In any diaphragmatic hernia measuring 4 cm or larger, a loose primary repair was performed and reinforced with the precut Parietex mesh. The mesh was anchored with hernia tacks at two or three points. During a period of 7 years, a total of 238 patients had a diaphragmatic hernia repair. Of these, a mesh was used in 55 patients (23%). Twenty patients were operated on for a recurrent diaphragmatic hernia and in 33 a mesh was used for repair

of a defect larger than 4 cm. During a follow-up of 58 months, there were two symptomatic hernias (3.6%) that necessitated a second repair. In addition, in four patients (7%) a small, so-called sliding hernia was diagnosed that necessitated no intervention. There were no long-term complications that could be related to the use of the mesh.

Encouraged by a series of Condon [13] with 44 patients of open mesh repair with a polypropylene onlay to the diaphragm showing a clinical recurrence rate of zero during a 15-year period, Frantzides and Carlson [14] were the first to address the problem of an unacceptably high recurrence rate of the sutured hiatal herniorrhaphy by using a mesh-reinforced cruraplasty with a minimally invasive approach. They hypothesized that the benefit from mesh placement would most likely be seen in patients with a large hiatal defect; 72 patients with GERD and large defect hiatal hernia were enrolled in a trial randomizing the subjects between simple posterior cruroplasty with or without PTFE onlay re-inforcement followed by performing a floppy Nissen fundoplication. After a mean follow-up of 3.3 years the recurrence rate in the cruroplasty onlay group was 22% (8/36) and the rate in the cruroplasty plus PTFE group was zero. There were no mesh-related complications. The PTFE patch was cut from a larger sheet of mesh with a 3.5-cm circular defect as a keyhole in the centre of the mesh to accommodate the esophagus.

Results of Own Patient Material

1. All 33 patients with a recurrent hiatal hernia who were treated with a circular polypropylene mesh underwent X-ray-control in December 2005. A recurrent hiatal hernia was seen in two patients (6%); 24 patients had a follow-up time of 5 years or more. The recurrences occurred in one patient after 1 year, in the other after 4 years.
2. All 15 patients in whom a tension-free procedure due to giant hiatal hernia was performed were controlled by radiological barium swallow in December 2005. Before December a re-operation had to be performed in three of them (20%): in one of them because of increasing dysphagia caused by a suggested impression of the mesh leading to an erosion of the esophagus. Two patients had to be operated on because of recurrences, one complete and one partial recurrence (recurrence rate 13.3%). The performed X-ray-control showed no further recurrences.
3. In all 20 patients with large hiatal hernias and treated by a mesh onlay procedure an X-ray control was performed in December 2005. One patient had experienced an accident with a sternum fracture a few months before and had to be reoperated on. No recurrences were found in this group of patients.

Complications

The use of prosthetic materials in surgery for large hiatal hernia repair is accompanied by a low incidence of foreign-body complications. Visceral erosions, foreign-body migrations or gastro-oesophageal fistulas after surgery are reported. The focus is on the possibility of erosion or migration of the mesh into the esophagus or stomach as well as complications due to severe mesh adhesions or the development of fibrotic strictures on the hiatal area. Beneath these complications (Table 12.1), there has been one fatal complication described by Kemppainen [15] not primarily related to the use of a mesh but to the use of a hernia stapler: after fixation of the mesh to the diaphragm, the patient developed a cardiac temponade caused by a stapler laceration of a coronary vein.

Discussion

The incidence of 30–50% of anatomical recurrences following simple sutured cruroplasty for both the open and laparoscopic approach is unacceptably high. Condon [13] was the first to show that the recurrence rates for the open approach could be minimized by using meshes. Especially Frantzides and Carlson were encouraged by these results, leading to their well-known randomized trial with 72 patients [6]. There are only a few comparative studies and trials of laparoscopic hiatal closure with simple sutures versus mesh hiatoplasty. All of them have shown that patients with a prosthetic hiatal closure have a lower rate of postoperative hiatal hernia recurrence in comparison to patients with simple hiatal repair (Table 12.2). There is debate not only whether to use prosthetics but also when to employ them. Champion [11] prospectively measured the hiatal diameter in 476 primary laparoscopic antireflux procedures with simple posterior suture closure of the hiatus, and demonstrated a recurrence rate of 0.9% if the initial crural diameter was <4.5 cm and a 10.6% recurrence risk if the diameter was >4.5 cm. The difference was highly significant. Since Frantzides and Carlson had an impressive difference in outcome between the control and mesh groups, they felt justified in broadening the indication for mesh usage and decreased their threshold

Table 12.1. Complications of prosthetic crural closure

Author	Type	Complications	Re-operation
	Prolene	Esophageal stenosis due to mesh-induced fibrosis	Laparoscopic revision
Trus [22]	–	Mesh-induced esophageal scarification	Relaparotomy with esophageal myotomy
Carlson [14]	Prolene	Esophageal mesh erosion	Transhiatal esophagectomy
Kempainen [16]	PTFE	Cardiac tamponade secondary to mesh fixation by tacks	
v. d. Peet [23]	Polyester	Hiatal fibrosis	Relaparotomy with mesh removal
Casabella [24]	–	Fibrotic hiatal damage/esophageal mesh ersion	Relaparotomy with distal esophagectomy
Coluccio [25]	PTFE	Penetration of the cardial lumen	Relaparotomy with distal esophagectomy
Zilberstein [26]	Dacron	Esophageal mesh migration	Laparoscopic mesh removal

for mesh usage to hiatal defects whose diameter is 5 or 6 cm. The original indication for the utilization of PTFE reinforcement during hiatal hernioraphy was a defect size of more than 8 cm.

As documented in various papers before, in our own patient material on the symptoms of GERD with hiatal hernia, we experienced a significantly higher recurrence rate with simple suture herniorraphy compared to patients with mesh usage [7, 16]. The evaluation of our database led us to attempt different methods of crural closure, depending on the size of the hiatal defect, by measuring the hiatal surface area (HSA). This HSA (Fig. 12.1) can be calculated with the length of the crura measured in centimetres beginning at the crural commissure up to the edge where the pars flaccida begins and the circuit between the both crural edges is measured. The HSA corresponds to the space of any hernia ring in square centimetres. This proceeding is equivalent to the way of fixing the threshold for mesh usage as Frantzides or Champion do. Patients with an HSA of <4 cm² undergo crural closure by simple interrupted non-absorbable sutures. Patients with an HSA >4 cm² with strong crura undergo simple sutured crural closure and additional application of a 1×3-cm polypropylene mesh which is cut out of a 10×15-cm mesh, which is usually taken for laparoscopic inguinal hernia repair. Patients with an HSA >4 cm² with weak crura or narrow crura undergo primary simple sutured crural closure and additional reinforcement with a composite mesh. Basically in all patients with extra large hiatal hernias with a HSA over 8 cm², the crural closure is performed without simple sutures in a tension-free technique. After hiatal dissection, a special v-shaped mesh with porous PTFE is positioned on the crura as a tension-free posterior onlay. By thus tailoring the method of crural closure to the size of HSA, the recurrence rates with a follow-up of more than 2 years are very low. In all patients with large hiatal defects undergoing laparoscopic redo-surgery after failed primary hiatoplasty, the crura were approximated by simple non-absorbable sutures and the hiatus reinforced with a circular polypropylene mesh [17]. Out of 33 patients with recurrent hiatal hernia with a circular polypropylene mesh, only two patients (6%) experienced recurrences in a follow-up of more than 5 years. Although this follow-up is short compared to the 20-year survey of Philip Allison [4], it has to be taken into consideration that these patients are of higher risk for experiencing recurrences, as they all had large hiatal defects, weak crura and most of them were obese. None of the papers, including our own dealing with [19] mesh usage, reported about mesh erosion or mesh migration into the esophagus or stomach. In contrast to only a few reported prosthetic erosions and

Table 12.2. Prosthetic hiatal closure

Author	Patients (no.)		Mesh	Follow-up (months)	Recurrences Hernia	
	Mesh	Non-mesh			Mesh	Non-mesh
Carlson [14]	44		Prolene	52	0	
Frantzides [15]	17	18	PTFE	36	0	3
Basso [11]	67	65	Prolene	22,5	0	9
Frantzides [6]	36	36	PTFE	6–72	0	8
Champion [12]	52		Prolene	7–60	1	
Keidar [13]	10	23	Prolene	46–76	1	4
Szold [20]	55	183	Parietex	58	2	NA
Gryska [19]	135		PTFE	64	1	

migrations associated with mesh at the hiatus, there are no complications in larger series with prosthetic mesh closure. Especially Gryska and Vernon [18] examined the safety and efficacy of a tension-free crural repair with a PTFE mesh in 135 patients with a mean follow-up of 64 months. They reported one reherniation but no migrations or erosions in that huge number of patients.

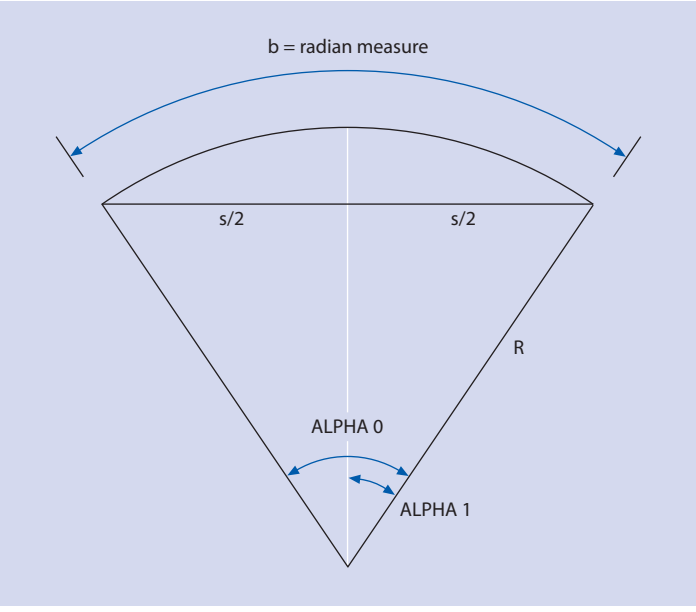


Fig. 12.1. Hiatal surface area (HSA)

With the paradigm shift in the repair of inguinal and ventral hernias to tension-free and mesh-inforced procedures, the unacceptably high recurrence rate of primary sutured repair of diaphragmatic hernias came under discussion. As with any other hernia, the goal for repair of the hiatus should be the creation of a tension-free repair. The diaphragm is a dynamic area with constant motion, even when at rest, and that may explain why the repair of the diaphragmatic hiatus is so difficult, with recurrence rates up to 50%. Of all the trials yet published comparing primary suture repair to mesh repair in hiatal hernia surgery, an advantage for the mesh group was documented with significantly lower recurrence rates. Although it seems to be evident that mesh usage is superior to simple suture repair, a lot of questions are unresolved: the technique for placement of meshes varies; there is also no agreement as to which mesh should be used, including the problem of tension-free or non-tension-free repair. Above all, it remains unclear how a recurrence is defined and what the indications for re-operations are.

References

1. Hashemi M, Peters JH, Deemeester TR, et al. (2000) Laparoscopic repair of large type III hiatal hernia: objective follow up reveals high recurrence rate. J Am Coll Surg 190: 553–561
2. Ferri LE, Feldman LS, Standbridge D, Mayrand S, Stein L, Fried GM (2005) Should laparoscopic paraesophageal hernia repair be abandoned in favor of the open approach? Surg. Endosc 19: 4–8
3. Blair A, Jobe MD, Ralph W, Aye MD, Clifford W, Deveney MD, John S, Domreis MD, Lucius D, Hill MD (2002) Laparoscopic management of giant type III hiatal hernia and short esophagus: objective follow-up at three years. J Gastrointest Surg 181–186
4. Allison PR (1951) Reflux esophagitis, sliding hiatal hernia, and the anatomy of repair. Surg Gynaecol Obstet 92:419–431
5. David I, Watson, MD, Nicholas Davies MD, Peter G, Devitt MS, Glyn G, Jamieson MS (1999) Importance of dissection of the hernial sac in laparoscopic surgery for large hiatal hernias. Arch Surg 134: 1069–1073
6. Frantzides CT, Madan AK, Carlson MA (2002) A prospective randomized trial of laparoscopic polytetrafluoroethylene (PTFE) patch repair vs simple cruroplasty for large hiatal hernia. Arch Surg 137: 649–652
7. Granderath FA, Schweiger UM, Kamolz T, Asche KU, Pointner R (2005) Laparoscopic Nissen fundoplication with prosthetic hiatal closure reduces postoperative intrathoracic wrap herniation. Arch Surg 140:40–48
8. Kuster GG, Gilroy S (1993) Laparoscopic technique for repair of paraesophageal hiatal hernias. J Laparoendosc Surg 3:331–338
9. Paul MG, DeRosa RP, Petrucci PR, Palmįer ML, Danovitch SH (1997) Laparoscopic tension-free repair of large paraesophageal hernias. Surg Endosc 11: 303–307
10. Basso N, De Leo A, Genco A, Rosato P, Rea S, Spaziani E, Primavera A (2000) 360° laparoscopic fundoplication with tension-free hiatoplasty in the treatment of symptomatic gastroesophageal reflux disease. Surg Endosc 14: 164–169
11. Champion JK, Ben-Shlomo II (1991) Erosion of marlex mesh collar after vertical banded gastroplasty. Obes Surg 1:443–444
12. Keidar A, Szold A (2003) Laparoscopic repair of paraesophageal hernia with selective hernia with selective use of mesh. Surg Laparosc Endosc Percutan Tech 13:149–154
13. Carlson MA, Condon RE, Ludwig KA, Schulte WJ (1998) Management of intrathoracic stomach with polypropylene mesh prosthesis reinforcement transabdominal hiatus hernia repair. J Am Coll Surg 187(3): 227–230
14. Frantzides CT, Carlson MA (1997) Prosthetic reinforcement of posterior curoplasty during laparoscopic hiatal herniorraphy. Surg Endosc 11: 769–771
15. Kemppainen E, Kiviluoto T (2000) Fatal cardiac tamponade after emergency tension-free repair of a large paraesophageal hernia. Surg Endosc 14(6): 593
16. Granderath FA, Schweiger UM, Kamolz T et al. (2002) Laparoscopic antireflux surgery with routine mesh-hiatoplasty in the treatment of gastroesophageal reflux disease. J Gastrointest Surg 6: 347–353
17. Granderath FA, Kamolz T, Schweiger UM et al. (2003) Laparoscopic refundoplication with prosthetic hiatal closure for recurrent hiatal hernia primary failed antireflux surgery. Arch Surg 138: 902–907
18. Gryska PV, Vernon JK (2005) Tension-free repair of hiatal hernia during laparoscopic fundoplication: a ten-year experience. Hernia 9: 150–155
19. Granderath FA, Carlson MA, Champion JK, Szold A, Basso N, Pointner R, Frantzides CT (2006) Prosthetic closure of the esophageal hiatus in large hiatal hernia repair and laparoscopic antireflux surgery. Surg Endosc 20: 367–379
20. Edelman DS (1995) Laparoscopic paraesophageal hernia repair with mesh. Surg Laparosc Endosc 5: 32–37
21. Trus TL, Bax T, Richardson WS, Branum GD, Mauren SJ, Swanstrom LL, Hunter JG (1997) Complications of laparsocopic paraesophageal hernia repair. J Gastrointest Surg 1: 221–228
22. Van der Peet DL, Klinkerberg-Knol EC, Alonso A, Sietses C, Eijsbouts QAJ, Cuesta MA (2000) Laparoscopic treatment of large paraesophageal hernias. Surg Endosc 14: 1015–1018
23. Casabella F, Sinanan M, Horgan S, Pellegrini CA (1996) Systematic use of gastric fundoplication in laparoscopic repair of paraesophageal hernias. Am J Surg 171: 485–489
24. Coluccio G, Ponzio S, Ambu V, Tramontano R, Cuomo G (2000) Dislocationinto the cardial lumen of a PTFE prosthetic used in the treatment of voluminous hiatal sliding hernia, a case report. Minerva Chir 55: 341–345
25. Zilberstein B, Eshkenazy R, Pajecki D, Granja C, Brito ACG (2005) Laparoscopic mesh repair antireflux surgery for treatment of large hiatal hernia. Disease of the Esophagus 18: 166–169

Discussion

Fuchs: *From the very few cases I have done, in two patient relaparoscopies where previously mesh was put in similarly to this technique. What I could see was that the hiatus in that posterior reach was very firm and scary. The mesh was incorporated, it was hard to see that it was a mesh, the colour had changed to the colour of the muscle infect. But you could still feel it, when you touched it.*

Pointner: *In those patients, we don't see complete wrap migration intrathoracically.*

Ferzli: *Do you know any case, or any situation while you are doing the dissection and are planning to put a mesh and you have an iatrogenic injury of the oesophagus or the stomach? Would you go ahead and put a mesh; have you had iatrogenic injury in this series that you have repaired and put a mesh?*

Pointner: *In a few cases I had an injury of the stomach and I put a mesh in and left it in, that is no problem. I have never had an injury of the oesophagus.*

Fuchs: *I would like to confirm this from doing a Collis together with a mesh. I have a suture line to the stomach and this has been no problem.*

Schippers: *Do we not have to learn how to fix the mesh? As I realized you changed suturing, you had tackers, you mentioned one patient dying after spiral tackers; during the coffee break I heard about two patients dying after spiral tackers.*

Pointner: *You are completely right.*

Köckerling: *Why do you use the circumferential mesh design in the recurrences, and in the primary case just the small 1- to 3-centimetre piece?*

Pointner: *Because the recurrences had larger hernias, and the other ones were just prospective, randomized only non-mesh versus mesh, independent of which hernia they had.*

Fuchs: *Without the study, would you still do this, or would you use different sizes of mesh adapted to the anatomical problems?*

Pointner: *I do not know if I would do it without the studies. But we are working now according to the hiatal surface area.*

13 Some Laparoscopic Hiatal Hernia Repairs Fail – Impact of Mesh and Mesh Material in Crural Repair

J. F. Kukleta

Introduction

The breakdown of crural repair occurs in 6–40% of laparoscopic hiatal surgery [5, 17] and often leads to recurrence with intrathoracic wrap migration or para-oesophageal herniation. In order to prevent this complication, various surgeons attempt to reinforce the repair or patch the unsutured crural defect with prosthetic material.

Similarly to the problematic of intraperitoneal prosthetic repair of incisional hernias, the use of mesh in hiatal repair is still controversial. The impact of the surgical technique and the unique behaviour of specific mesh materials is recognized but far from being well investigated, understood and clearly standardized. Despite significant decrease in recurrence rate, some sporadic dangerous complications have been reported [9, 28]. One can assume that the numbers and complexity of these adverse events are strongly under-reported.

Method

Besides the review of the available literature published in English between 1995 and 2005, a personal communication of unpublished information to this rare topic from various experts is added. Not unexpectedly, sometimes the personal opinion of experienced laparoscopists differs from the trends imposed by the latest scientific papers.

Problem Analysis

Many causes of recurrence are suggested and discussed in the literature, but very few are supported by data, like surgeon's inexperience, postoperative vomiting, retention of the hernia sac and heavy lifting [1]. Although statistically not proven, chronic cough, smoking-related impairment of collagen synthesis and any other chronic increase of intra-abdominal pressure are logical promoting factors of recurrence.

Possible additional mechanisms directly related to laparoscopic procedure include no nasogatric tube in the early postoperative course, too early return to normal activities before the scar tissue is formed, less adhesions in laparoscopic surgery when compared to open technique.

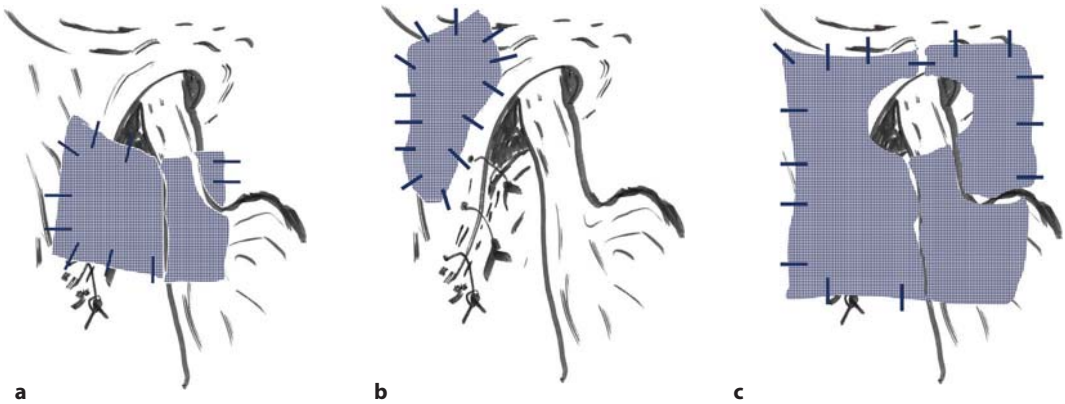
The early experience with laparoscopic repair of hiatal hernias of type II and III demonstrated higher recurrence rate than the open technique [2]. The individual learning curve, failure analysis and corrections of surgical technique, especially complete hernia sac removal from mediastinum or its excision, improved the durability of the repair [3, 4]. The significance of oesophageal shortening caused by chronic inflammation is still under debate. Due to fear of postoperative dysphagia, the crurorhaphy tends to become too loose rather than too tight, especially since the hiatal calibration with large bougies is being given up by many to avoid possible intra-operative perforation.

The principle cause of crural disruption is the tension: either the defect is too big, the repair too weak from the very start or it becomes insufficient due to acute or chronic increase of intra-abdominal pressure. The anatomical recurrence rate of non-reinforced crurorhaphy in type-II and -III hernias is after longer follow-up too high, but less than 50% of these patients are symptomatic.

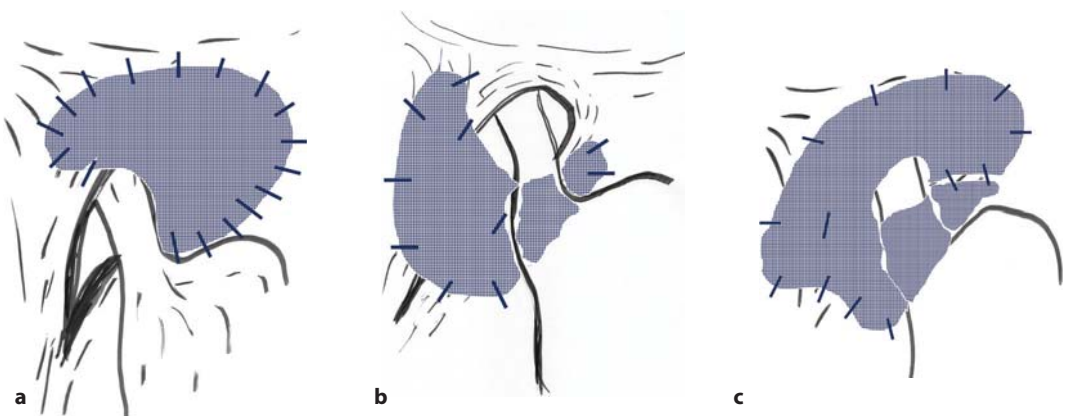
During the laparoscopy the diaphragm is distended and stretched. This effect makes the available tissue bites smaller and the repair weaker [1]. In redo surgery, the crural repair is even more difficult, because the disruption leads to a rigid defect and the crurorhaphy increases the tension even more. In large defects the posterior crural repair displaces the GE junction too far ventrally, potentially resulting in impaired transit. Although the diaphragm becomes thinner ventrally of the oesophagus, the anterior crural repair appears to be at least as good in the short term as posterior suturing as a method of narrowing the hiatus during laparoscopic Nissen fundoplication [13].

Results

As the use of prosthetic material is no longer taboo, many investigators use various materials under unequal conditions, and with different indications and additional technical modifications. Therefore a comparison of the methods and their outcome at this stage is nearly impossible (◘ Figs. 13.1 and 13.2).



◘ **Fig. 13.1. a** Reinforced posterior repair. **b** Relaxing incision. **c** Reinforced circular repair



◘ **Fig. 13.2. a** Patched anterior repair. **b** Patched posterior repair. **c** Patched circular repair

Most of the published experiences with the use of mesh in hiatal hernia are from small series with limited or rather short follow-up. Few comparative studies have demonstrated significant reduction of recurrence mesh vs. non-mesh, with a mesh-related complication rate close to zero [5, 8, 12]. The overall mesh complication reported is less than 2% [18].

Analysis of the complex issue of a prosthetic repair shows at least five important mesh related variables: the mesh material itself, its anchorage, its shape, position and function.

Function

Intraperitoneal onlay mesh can be used to reinforce the crural repair (not tension-free) [8, 9, 10, 11] or bridge/patch the enlarged hiatus without crural approximation, leaving the passage for the abdominal oesophagus free in different ways (true tension-free repair) [6, 7, 15].

Fixation

The mesh can be anchored to crura with sutures, tacks or staples. Sutures are more time consuming, staples and tacks can be more dangerous, inconstantly not deep enough and distort the mesh, depending on the material used. Cardiac tamponade was reported following tack fixation.

Position

Irrespective of the mesh purpose it can lie anteriorly [3, 6, 13] or posteriorly in relation to the oesophagus. Most authors are used to perform a posterior crural repair and therefore they buttress or patch posteriorly [7, 12]. The posterior total or partial fundic wrap protects the oesophagus from direct contact with the implant or at least from the transverse mesh edge.

Shape

A certain degree of creativity is still an important part of our profession. Numerous shapes were suggested: oesophagus totally encircling [19, 20] (A-shape, keyhole), partially encircling (U-shape, Arc de Triomphe-shape [3]) or not encircling triangular, rectangular, etc. (reinforcing, patching or covering the relaxation incision of the right crus).

Mesh Material

See ◘ Table 13.2.

Implant Site

The mesh-underlying tissue interface is similar, but not identical with the one in inguinofemoral or laparoscopic incisional hernia repair. The contact surface in hiatus is a thin muscle with a good blood supply with vital structures in the vicinity. The respiratory movements, the heartbeat and the oesophageal peristalsis make the region very difficult to be "just" stabilized.

Porosity

The macroporous meshes will induce and permit a complete tissue ingrowth. After maturation of collagen, a solid scar tissue is present thus incorporating the mesh. The meshed area of the hiatus oesophagei is in constant motion, therefore there must be a solid fixation guaranteed in the early postoperative period to prevent mesh dislocation and consecutive recurrence. The appreciated inflammatory reaction reinforces the interface, but bears an uncontrollable risk of oesophageal erosions or stenosis. The microporous meshes require better fixation. The biological meshes permit a complete ingrowth and cause a strong inflammatory reaction, which can lead to oesophageal stenosis.

Transparency

Transparent meshes add more security to mesh fixation, eliminate unrecognized bleeding when not blindly applying penetrating fixation and permit more generous suture bites.

Stiffness

The biggest disadvantage of polypropylene and polyester meshes is the loss of local elasticity due to fibrotic fixation, and the mesh margins may become sharp. The first may cause dysphagia due to impairment of peristalsis or stenosis, the latter erosions, migration or late oesophageal perforation. The resulting stiffness of the traditional "heavy" materials is not existent in lightweight meshes.

Table 13.1. Incomplete overview of prosthetic materials

Absorbable	Polyglactin 910	Vicryl
	Polyglycolic acid	Dexon
Non-absorbable	Polypropylene	Prolene, Marlex, Surgipro, Trelex, Parietene, Prolite, TiMesh
	Polyester	Mersilen, Parietex
	PTFE	Goretex, Dualmesh
	Composites	PP/e-PTFE Composix
		PP/RCO Proceed
		PP/Sepra Sepramesh
		PP/Polyglactin 910, Vypro, Vypro-2
		PP/Polyglecaprone Ultrapro
		PP/collagen film Parietene composite
		PE/collagen film Parietex composite
	PVDF/PE	Dynamesh
Biomaterials	Porcine SIS	Surgisis
	Porcine skin	Permacol
	Human skin	Alloderm

Less risk-bearing appears e-PTFE (without any objective proof), because it stays much softer and is less prone to adhesions, but is non-transparent and difficult in handling. Gryska reported no erosions (135 patients) after 10 years of experience [5].

Shrinkage

All mesh materials alter their extent after the primary scar tissue reaction is over. This "hot or overheated" issue in inguinofemoral hernia repair does not seem to be of clinical importance in mesh-supported hiatal repairs. The well-known pronounced shrinkage of the PTFE products or heavy polypropylene meshes could theoretically cause late dysphagias in patched repairs of large hiatal defects. The use of light-weight meshes as a consequence of the above fact has not yet been reported.

Infection Resistance

The incidence of infection of the prosthetic material in this specific location is so low, that it does not seem to be of significance as long as the digestive tract remains intact.

Mesh-Related Complications

In the early postoperative course a higher incidence of dysphagia of longer duration was reported [29].

The inflammatory reaction, which is a material-specific host response to a foreign body, can cause a material-specific morbidity even many years later. Erosions have been reported after 3 years with polypropylene [9], late oesophageal perforation with PTFE, Teflon pledget intrusion in oesophagus 9 years

after repair [28]. The adhesiogenic potential is in given localisation not of big concern as long as the oesophagus is not encircled, the direct contact of mesh and oesophagus can be avoided and materials, that turn to be stiff when fibrotic reaction takes place, are not used (heavy PP). Stiffness and wrinkles, that will become sharp edges are the main problem.

Discussion

For well-known reasons the information and knowledge being elaborated by studies even of a low level of evidence pass through many different filters, suffer from heterogeneity, difficulty of standardization and often from the impossibility to exclude the major variable factor in any clinical study – the personal experience of the surgeon. Unpublished opinion of opinion leaders is a different kind of information. It might be very subjective, it might not be scientifically correct, but it mostly reflects a personal attitude of a professional based on experience. To compare this personal information with the published literature, the author contacted 30 experienced surgeons by e-mail. More than 50% answered the simple question: what about a mesh in hiatal hernia repair and what is your preferred solution?

Mesh at All?

The vast majority would use the mesh very selectively. Some try to avoid prosthetic around oesophagus per principle, some reinforce the suture with pledgets or bicrural strips. Mesh as seldom as possible, most often only under difficult conditions in redos. The fear of erosion is understandable after a personally experienced disaster, but the general opinion seems to be overimpressed by few reported cases. If prosthetic material is used, the distance of the mesh margin to the oesophagus has to be warranted and encircling is not recommended (Table 13.1).

Table 13.2. Reports on materials used

Source	Material used
Frantzides [8]	Circular PTFE, PCR
Granderath [29]	Circular PP, PCR
Kamolz [12]	PCR + PP retrooesophageal strip
Casaccia [6]	Parietex composite, A-shape
Basso [7]	PP, retrooesophageal rectangular patch
Keidar [19]	Composix
Szold [20]	Parietex composite
Gryska [5]	PTFE retro-oesophageal, V-shape
Oelschlager [21]	Surgisis
Aregui	Surgisis, PCR, relaxing incision
Gagner	Surgisis
Jacobs	Surgisis
McKernan	Surgisis
Dallemagne	Pledgets, Surgisis
Filipi	PTFE, halfcircle
Himpens	PTFE, slit mesh
Bailey M	PP, bicrural strip, PCR
Giulianotti	Teflonpledgets, PTFE, semicon-cave, PCR

PP polypropylene, *PCR* posterior crural repair

What Material?

Most of the known materials did well in published reports (Table 13.2). The more personal experience with the use of mesh in hiatus, the more often the biomaterial Surgisis is proposed. The satisfaction with this product ranges from negative to very positive, from the danger of being too reactive (leads to stenosis and oesophageal-gastric resection) to a trend to reinforce even small sliding hernias to reduce the chance of rehernia-tion. The most frequently used material is still e-PTFE (according to the literature), being the best documented and having the longest follow-up.

Which Additional Manoeuvres?

As already analyzed [22], there is no available evidence on the use of additional "anti-re-herniation" surgical steps like fixation of the wrap on the crural repair or

gastropexy or gastrostomy. The importance of Collis oesophagus lengthening gastroplasty is unclear.

Routinely or Selective Approach?

Surgeons who can rely on their own results reinforce not only the redos or large type III, but even the small sliding hernias, to secure their good functional results, especially in long-term follow-up.

Is the Fear of Potential Mesh Complication Justified?

The indication for a surgical intervention in the case of large para-oesophageal hernia has often a prophylactic character due to its known natural course with possible serious complications. The minimally invasive solutions make the decision for a repair easier even in the elderly, but do not resolve the problem of recurrence. The vast majority of experienced laparoscopists are very reserved to foreign material in hiatus and would try to avoid it in primary repairs. Despite the fact that reported experience with biomaterials is of singular nature, more than half of the reviewed experts would advocate their use. The use of mesh in crural repair will have to stay selective until the mesh-related complications can be eliminated by improved materials.

Conclusion

The evidence of the most reports is low (II c–V). The very few existing comparative studies [7, 8, 12] have demonstrated the superiority of mesh repair.

The incidence of serious mesh-related complications is very low. Due to the fact that the reason for a breakdown of crural repair is multifactorial and the incidence of type-III hernias is low, there are no objective data available to justify the exclusive choice of one or another mesh material. Based on the reported information, the potential risk of visceral erosions, late fistulization and wound sepsis known from inguinal and incisional hernia repairs should not be transferred 1:1 to hiatal repair. However, the principles learned from experience should finally influence the operative strategy of crural repair: celebrating precise surgical technique and choosing light-weight or tissue-separating coated meshes. The objective value of biomaterials, although already very promising, must be demonstrated in more extensive studies.

References

1. Puri V, Kakarlapudi GV, Awad ZT, Filipi CJ (2004) Hiatal hernia recurrence. 8: 311–317
2. Hashemi M, Peters J, Demeester T, Huprich J, Queck M, Hagen J, Crookes P, Theisen J, Demeester JR, Sillin L, Bremner C (2000) Laparoscopic repair of large type III hiatal hernia: objective follow-up reveals high recurrence rate. J Am Coll Surgeons 190: 553–560
3. Leeder PC, Smith G, Dehn TC (2003) Laparoscopic management of large paraesophageal hiatal hernia. Surg Endosc 17:1372–1375
4. Edye MB, Canin-Endres J, Gattorno F, Salky BA (1998) Durability of laparoscopic repair of paraesophageal hernia. Ann Surg 4: 528–535
5. Gryska PV, Vernon JK (2005) Tension-free repair of hiatal hernia during laparoscopic fundoplication: a ten-year experience. Hernia 9(2):150–155
6. Casaccia M, Torelli P, Panaro F, Cavaliere D, Ventura A, Valente U (2002) Laparoscopic physiological hiatoplasty for hiatal hernia: new composite A-shaped mesh. Surg Endosc 16:1441–1445
7. Basso N, DeLeo A, Genco A, Rosato P, Rea S, Spaziani E, Privaera A (2002) 360° laparoscopic fundoplication with tension-free hiatoplasty in the treatment of symptomatic gastroesophageal reflux disease. Surg Endosc 14: 164–169
8. Frantzides C, Madan A, Carlson M, Stavropoulos G (2002) A prospective, randomized trial of laparoscopic polytetrafluoroethylene (PTFE) patch repair vs simple cruroplasty for large hiatal hernia. Arch Surg 137: 649–652
9. Carlson MA, Condon RE, Ludwig KA, Schulte WJ (1998) Management of intrathoracic stomach with polypropylene mesh prosthesis reinforced transabdominal hiatus hernia repair. J Am Coll Surg 187: 227–230
10. Morales-Conde S, Bellido J, Cadet I, Martin M (2002) Indications and management of prostheses to close the crura during laparoscopic repair of paraesophageal hernias. Surg Endosc 16: 284
11. Champion JK, Rock D (2003) Laparoscopic mesh cruroplasty for large paraesophageal hernias. Surg Endosc 17: 551–553
12. Kamolz T, Granderath FA, Bammer T, Pasiut M, Pointner R (2002) Dysphagia and quality of life after laparoscopic Nissen fundoplication in patients with and without prosthetic reinforcement of the hiatal crura. Surg Endosc 16: 572–577
13. Watson DI, Jamieson GG, Devitt PG, Kennedy JA, Ellis T, Ackroyd R, Lafullarde TO, Game PA (2001) A prospective randomized trial of laparoscopic Nissen fundoplication with anterior vs posterior hiatal repair. Arch Surg. 136: 745–751.
14. Granderath F, Kamolz T, Schweiger U, Pointer R (2003) Laparoscopic refundoplication with prosthetic hiatal closure after primary failed antireflux surgery. Arch Surg 138: 902–907
15. Paul MG, DeRosa RP, Petrucci PE, Palmer ML, Danovitch SH (1997) Laparoscopic tension-free repair of large paraesophageal hernias. Surg Endosc 11: 303–307
16. Targarona EM, Balague C, Martinez C, Garriga J, Trias M (2004) The massive hiatal hernia: dealing with the defect. Semin Laparosc Surg 11(3): 161–169

17. Targarona EM, Novell J, Vela S, et al. (2004) Mid-term analysis of safety and quality of life after the laparoscopic repair of paraesophageal hiatal hernia. Surg Endosc 18: 1045–1050
18. Targarona EM, Bendahan G, Balague C, Garriga J, Trias M (2004) Mesh in the hiatus – a controversial issue. Arch Surg 139: 1286–1296
19. Keidar A, Szold A (2003) Laparoscopic repair of paraoesophageal hernia with selective use of mesh. Surg Laparosc Endosc Percutan Tech 13: 149–154
20. Szold, Sagie B. Laparoscopic mesh repair of diaphragmatic hernias (2004) 26th Grepa congress in Prague
21. Oelschlager BK, Barreca M, Chang L, Pellegrini CA (2003) The use of small intestine submucosa in the repair of paraesophageal hernias: initial observations of a new technique. Am J Surg 186(1): 4–8
22. Draaisma WA, Gooszen HG, Tournoij E, Broeders IAMJ (2005) Controversies in paraesophageal hernia repair. Surg Endosc 19: 1300–1308
23. Carlson MA, Richards CG, Frantzides CT (1999) Laparoscopic prosthetic reinforcement of hiatal herniorrhaphy. Dig Surg 16: 407–410
24. Frantzides CT, Richards CG, Carlson MA (1999) Laparoscopic repair of large hiatal hernia with polytetrafluoroethylene. Surg Endosc 13: 906–908
25. Edelman DS (1995) Laparoscopic paraesophageal hernia repair with mesh. Surg Laparosc Endosc 5: 32–37
26. Champion JK, McKernan JB (1998) Prosthetic repair of diaphragmatic crural defects during laparoscopic fundoplication. Hernia 2: 511
27. Cadiere GB, Bruyns J, Himpens J, Vertruyen M (1996) Intrathoracic migration of the wrap after laparoscopic Nissen fundoplication. Surg Endosc 10: 187
28. Arendt T, Stuber E, Monig H, Fölsch UR, Katsoulis S (2000) Dysphagia due to transmural migration of surgical material into the esophagus nine years after Nissen fundoplication. Gastrointest Endosc. 51: 607–610
29. Granderath FA, Schweiger UM, Kamolz T, Asche KU, Pointner R (2005) Laparoscopic Nissen fundoplication with prosthetic hiatal closure reduces postoperative intrathoracic wrap herniation – Preliminary results of a prospective randomized functional and clinical study. Arch Surg 140: 40–48

Discussion

Carlsson: *Today I have heard a lot of anecdotal reports about the possible danger about the prosthetic oesophageal hiatus and in specific reference to a PTFE I have not been able to find published evidence of PTFE as a primary cause of erosion in the oesophagus. Now there are cases where there was a secondary problem, for example if a surgeon preparated the oesophagus and the stomach and then the PTFE was found in the preparation. Then this was called an erosion. But I have not been able to find a situation where PTFE eroded into the lumen primarily. I would encourage anyone in this room to report in published form these cases of a mesh erosion, so we can get this out of the table.*

Miserez: *I will not ask any questions about meshes, but in your second slice you mentioned the absence of an oesogastric tube postoperatively as a risk factor for an early recurrence. How long do you keep this tube in and on what evidence is this decision based?*

Kukleta: *We remove it before the patient wakes up in the laparoscopic repair. In the open repair, we have a longer ileus time so we keep it in. When the patients are fine they start eating and then they go. Probably they go very early they don't have an oesogastric tube after the operation. It is taken out in the recovery room.*

Franzidis: *This was an excellent presentation and what you show is that we are not in a perfect world. This is an imperfect world with problems, and we want to have a perfect operation and perfect prosthesis. I still believe that when the literature shows that you have a 30–50% rate of recurrence, then someone should come up with an alternative. Until then we have to accept the consequences. The reported erosion of PTFE or ePTFE is an anecdotal report. The same applies to dual mesh. Maybe these complications are under-reported or anecdotal. If the experts in the field would agree that this is a standardization of the technique we might avoid erosions. The advice is, that anybody embarking on this type of operation should be a very experienced laparoscopic surgeon and should have done his homework in the laboratory.*

Kukleta: *But certainly we end up with the technical details. This is an evolution of 10 years, and in 10 years you always add something to this, because it is difficult to stay with the same regime. If some people can reach these, we have to orient ourselves on those. That is my belief.*

Schumpelick: *There is something that I don't understand in this session. I hear that very small meshes fit, I hear that big meshes are used, I heard that you use different types of meshes, difficult localisations and you always mesh a reflux as a criterion that works. Are there any animal or anatomical or postmortem studies that show how the mesh really works? I think it is a bit like evidence level five. Everybody says I have good results, but how does it work? Some say better adhesions, some say it is better to have a patch on it; it is absolutely confusing for me. Are there better results in the literature than here?*

Kukleta: *We certainly have a problem with the incidence of these big hernias. They are not so numerous as inguinal hernias. If you have seen Dr. Pointners setup, there are very few papers that have enough numbers, just seven or eight studies with more than 100 cases. That is, why I cannot answer this.*

Ferzli: *Carlsson made a report about PTFE and you mentioned about the erosion. Phillip Chowbey mentioned the erosion of PTFE with a hiatal hernia into the oesophagus, and Eric DeMaria from Virginia reported one erosion of PTFE in the oesophagus. Just to clarify that.*

Pointner: *Prof. Schumpelick, thank you for your comments. In my opinion this is the important point. We don't know how large the hiatus really is. We have no anatomical studies, and today this was the first presentation I have seen, heard or found.*
Fuchs: *Most important was the pre-operative radiography; but this is an unreliable tool. Because you have patients where these 5 cm you do two times with a swab and they are down, and you have other patients where you are busy for half an hour clearing it. So it is not reliable. Probably it is much more reliable, as you suggested, to mesh the hiatus and then go on from there.*
Dutta: *I have two ideas. One is that mesh produces adhesions; the other is that mesh produces tension. I was fascinated by Dr. Pointner's report of using small mesh. I am thinking of the box a little bit and am wondering a little if that small mesh probably is reducing tension if it is causing adhesions. Has anyone thought about injecting a sclerosing agent into the crural to introduce adhesions?*
Kukleta: *But the muscle does this.*
Köckerling: *We can have our experimental experience with the different types of polypropylene meshes. I agree, obviously it is better to use light-weight polypropylene meshes. What we have learned in our experimental studies is that after 3 months, when we sacrifice the animals, this type of mesh behaves like nearly normal connecting tissue. It has no sharp edges, it does not fold due to shrinkage and other things, whereas the heavy-weight polypropylene meshes do that, they have sharp edges, they fold, they are stiff. From our point of view I would always prefer, if you use polypropylene mesh, then the light-weight mesh. Because it is like normal connective tissue.*

Concluding Remarks

Ferzli: *What we heard this evening is much more controversial than we thought. Now we are not able to say what is the best. Most speakers have repeated the significant points that are still unresolved. From the fixation to the wrap, the fixation of the oesophagus. I cannot go ahead and say we have a consensus. From what we have seen, we still have to go a long way. Hopefully the future will bring us some better answers.*
Fuchs: *If we look together at what to avoid, I think what we have learned this evening, or what we have discussed this afternoon, that we have here not one problem or not one disease. We have basically two, the reflux problem and the hiatal problem. In some patients, I would say in most patients, the reflux problem is foremost, but in some other patients it is maybe 10 to 20% it is the hiatus. The hiatal problems are really those that must have a higher priority. If I look at our experience of redos, there are some patients who come for the second or the third, fourth or even the fifth time. And if you come for several times, migration is still, of course, a problem. Also a spectrum of other reasons; we have to clarify, when mesh, for example, can help. To start with, you have to avoid that an operator who really has experience neither in laparoscopic surgery nor in the reflux disease or hernia repair, because that is really bad. Of course, you have to avoid the oesophageal perforation, destroying the crurals or injecting a sclerosing agent. This can be a real problem, because then you have nothing to put together. Too much tension on the suture, as we all know in the area of the body is a problem. Placing too many sutures and that is limiting, can be a problem. Placing too many sutures creates an angle that might have the effect of dysphagia. Or creating a stenosis is bad. Narrowing the hiatus insufficiently, even a gap, then the road is free for migration, and using insufficient crural alone for narrowing, as we have learned, is also a problem. So we need some material over the next 5 years to learn what size, what material we can use.*

Abdominal Wall Closure

14 Finding the Best Abdominal Closure – An Evidence-Based Overview of the Literature

A. Ceydeli, J. Rucinski, L. Wise

Introduction

Despite advances in surgical technique and materials, abdominal fascial closure has remained a procedure that often reflects a surgeon's personal preference with a reliance on tradition and anecdotal experience. Several theoretical and practical facts have been described about operative site healing and include the physiology of fascial healing, the physical properties of specific closure methods, the properties of the available suture materials and patient-related risk factors [1, 2]. Yet the ideal techniques and materials, although suggested by the surgical literature, have not been uniformly accepted.

The value of a particular abdominal fascial closure technique may be measured by the incidence of early and late wound complications. Early complications include wound dehiscence (sometimes associated with evisceration) and infection, while late complications are hernia, suture sinus, and incisional pain.

The best abdominal closure technique should be fast, easy, and cost-effective, while preventing both early and late complications. Traditionally, individual authors have advocated one technique over another for theoretical or practical reasons but, until recently, evidence-based principles have not been applied to the subject as a whole. Relevant factors for review include: 1) layered closure, mass closure, and retention sutures, 2) continuous closure and interrupted closure, 3) suture material and 4) suture thickness and the suture-length-to-wound-length ratio. Careful analysis of the current surgical literature, with the identification of evidence-based conclusions, indicates that there is relative consensus regarding the most effective method of midline abdominal fascial closure.

Methods

A MEDLINE (National Library of Medicine, Bethesda, MD) search was performed. All articles related to abdominal fascia closure published from 1966 to 2003 were included in the review.

Discussion

Layered Closure, Mass Closure and Retention Sutures

Layered closure is described as the separate closure of the individual components of the abdominal wall, specifically the peritoneum and the distinct musculoaponeurotic layers. Mass closure is the closure of all the layers of the abdominal wall (except the skin) as one structure.

Layered closure, often in conjunction with a paramedian incision, is a technique that was viewed as essential to adequate and appropriate wound closure in the past. Discussion of the technique, however, has disappeared from current surgical writing and it is little used in

practice. The proponents of layered closure believed that the approach reduced intra-peritoneal adhesions, contributed to wound strength, discouraged dehiscence, prevented leakage of intraperitoneal contents and promoted hemostasis [3–8].

Smead first described a mass closure technique in 1900. Jones described the same technique in 1941 and thereafter it was called the Smead-Jones technique. Dudley, in an experimental study in 1970, showed that mass closure was superior to layered closure when using stainless steel wire [9]. In 1975 Golligher supported the concept of mass closure by demonstrating a dehiscence rate of 11% with layered fascial closure compared to a rate of 1% with mass closure. (It should be noted, however, that chromic catgut, with its own inherent reasons for wound failure, was used for layered closure and was compared to stainless steel wire for mass closure) [10]. In 1982 Bucknall and co-authors prospectively studied 1129 abdominal operations and demonstrated that layered closure was associated with a significantly higher dehiscence rate compared to mass closure (3.81 vs. 0.76%) [11].

Subsequent investigators, further questioning the beneficial effects of layered closure, compared it with mass closure techniques producing a number of conclusions favoring the latter. Peritoneal closure, specifically, has been shown to be associated with an increased incidence of adhesions, compromise of the adequacy of closure of the subsequent layers and increased duration of operation [12–25]. Recently published meta-analyses have confirmed a statistically significant reduction in hernia formation and dehiscence with mass closure [26–28].

Retention sutures (involving the entire thickness of the abdominal wall including the skin and subcutaneous tissue) were first described by Reid in 1933 but have lost much of their popularity in recent years. It has been shown that the additional security of retention sutures is largely hypothetical, that they are associated with increased postoperative pain and that they make site determination of enteral stomas difficult [13]. In addition, retention sutures have not been shown to decrease the incidence of fascial dehiscence [13].

Continuous Closure and Interrupted Closure

Multiple reports show no difference in the incidence of dehiscence or hernia formation when either technique is used [29–32]. Proponents of continuous closure cite an evenly distributed tension throughout the length of the incision and a more cost-effective closure, requiring half as much time and less suture material, as definite advantages of continuous mass closure [26, 29–38]. It has also been shown experimentally that the bursting strength of a wound is significantly higher when a continuous closure is used [39–40]. Continuous closure minimizes the number of knots and has been shown to be associated with an equivalent or lower incisional hernia rate in four meta-analyses [26, 27, 28, 41]. The only theoretical disadvantage of continuous closure is that the security of the wound is dependent on a single strand of suture material and a limited number of knots. Disruption of the knot or the suture, however, has been shown to be a rare cause of wound dehiscence [33, 42].

Suture Material

Nonabsorbable, slowly absorbable, and rapidly absorbable suture materials are available. In addition, such materials are available in monofilament and multifilament (braided) form. The choice of material for closing the abdominal fascia should be made in the light of what is known about fascial healing and the physical properties of suture material (strength, durability, ease of handling, and resistance to infection) [43]. It was demonstrated in the early 1950s that the healing process of abdominal fascia after surgical incision lasts 9 to 12 months [44, 45]. Abdominal fascia regains only 51 to 59% of its original tensile strength at 42 days, 70 to 80% at 120 days and 73 to 93% by 140 days. Tensile strength never rises to higher than 93% of the strength of unwounded fascia [44, 45].

Nonabsorbable materials have been widely used for abdominal fascial closure since the 1970s. The most common nonabsorbable materials used are polypropylene (Prolene), nylon (Nurolon), polyethylene (Ethibond) and polyamide (Ethilon) [46]. Stainless steel wire and silk are only of historical note and are infrequently used in current surgical practice. Stainless steel is difficult to handle and tie and tends to develop fractures. Braided silk is a long-lasting biomaterial but is associated with a rapid loss of tensile strength (similar to absorbable sutures), a high association with infection, and an intense inflammatory reaction [48–50]. Other braided nonabsorbable suture materials have much better tensile strength characteristics but are less resistant to infection than nonabsorbable monofilament or absorbable materials [48–50].

Non-absorbable monofilament suture materials have been shown to have more tissue reactivity compared to stainless steel but less than that of absorbable materials. They are more resistant to infection but their use

is associated with a higher incidence of sinus formation, wound pain, and button-hole hernia [47–54]. The benefits of nonabsorbable materials lie in the fact that they retain their strength as the fascia develops intrinsic strength in the process of wound healing.

Absorbable materials are designed to approximate the fascia during the critical early healing period and subsequently to undergo absorption in order to avoid the complications of sinus formation, pain, and button-hole hernia associated with nonabsorbable sutures. The incidence of chronic wound pain and suture sinus formation has been found to be significantly less with absorbable material [28, 47, 52, 53]. Absorbable sutures may be classified as rapidly absorbable and slowly absorbable. Catgut, chromic catgut, polyglycolic acid, and polyglactin 910 are examples of rapidly absorbable materials.

In surgical practice catgut and chromic catgut are no longer widely used for fascial closure. Polyglycolic acid (Dexon) and polyglactin 910 (Vicryl) are the most commonly used rapidly absorbable suture materials. Absorption of such materials lasts 15 to 90 days, although most of their tensile strength is lost in 14 to 21 days [46]. Dexon and Vicryl are braided materials but are less reactive than silk or catgut since they are absorbed by hydrolysis. Their absorption may be delayed by infection and they may act as a focus for infection and as a foreign body with an associated delay in healing [26–28, 41, 49]. The rapidly absorbable suture materials have been associated with increased rates of incisional hernia formation when compared to nonabsorbable sutures [28, 46, 47].

Polydioxanone (PDS) and polyglyconate (Maxon) are the most commonly used slowly absorbable suture materials. Absorption of such materials takes about 180 days and they maintain 50% of their tensile strength for about 4 weeks [46, 56–61]. PDS has been shown to have 1.7 times the tensile strength of Prolene. Maxon, the newest of the synthetic absorbable materials, has been shown to be 16% stronger than Vicryl [39]. PDS and Maxon are more similar to nonabsorbable materials than are Vicryl and Dexon in that they retain their strength for a longer period during fascial healing. They are absorbed slowly by hydrolysis and are not subject to enhanced absorption by bacterial enzymatic activity. Several studies have shown no statistically significant difference in the incidence of incisional hernia formation, wound dehiscence, or infection between the slowly absorbable and the nonabsorbable suture materials. In contrast, nonabsorbable suture materials have been associated with statistically higher rates of incision pain and suture sinus formation [28, 41, 44, 47, 52, 53].

Suture Size and Suture-Length-to-Wound-Length Ratio

The mechanical reasons for wound dehiscence are as follows:

- the suture breaks,
- the knot slips, or
- the suture cuts through the tissues.

Generally the first two reasons are rare and wound dehiscence occurs when the suture material tears through the fascia. The strength of a particular suture material increases as its cross-sectional diameter increases and smaller diameter sutures are associated with a greater likelihood of tearing through the tissue [32, 33, 42, 62, 63].

Most of the studies in the current surgical literature employ a number zero or larger-sized suture to close the fascia. It should be noted, however, that one series found no increase in the incidence of wound dehiscence when size 2–0 suture material was used to close the fascia [52]. The double-loop closure method provides the most tensile strength, but in one study was associated with a significantly increased rate of pulmonary complications and postoperative death, possibly related to decreased compliance of the abdominal wall [64]. The suture thickness chosen, then, must provide adequate tensile strength as well as adequate elasticity to accommodate an increase in intra-abdominal pressure in the postoperative period.

The suture-length-to-wound-length ratio involves a geometric approach that aims to avoid wound dehiscence and hernia formation. It has been shown experimentally by Jenkins that the length of a midline laparotomy incision can increase up to 30% in the postoperative period in association with a number of factors that increase the intra-abdominal pressure [65]. If the bites taken in suturing (and the associated length of suture material used) are not large enough to accommodate the potential increase in wound length, then the suture may cut through the fascia, resulting in wound dehiscence. Jenkins, using the principles of geometry and the rules that apply to the component sides of triangles, studied the relationship of the bites of tissue taken in suturing to the amount of suture material used. He concluded that the bite of tissue needed to avoid suture pull-through could be expressed in terms the length of suture material needed for the incision under consideration. In the study it was determined that a suture-length-to-wound-length ratio of 4:1 would incorporate a large enough bite of tissue such that suture pull-through could not occur even with maximal

lengthening of the incision in the postoperative period [65, 66, 67]. The 4:1 suture-length-to-wound-length ratio was achieved in Jenkins' study by placing the sutures approximately 2 cm away from the fascial edge and approximately 2 cm from one another.

Conclusion

The best abdominal closure technique should be fast, easy, and cost-effective while preventing both early and late complications. The early complications that are to be avoided are wound dehiscence and infection and the late complications to be avoided are hernia, suture sinus, and incisional pain. Careful analysis of the current surgical literature, with the identification of evidence-based conclusions, indicates that there is an optimal technique. The most effective method of midline abdominal fascial closure involves mass closure, incorporating all of the layers of the abdominal wall (except skin) as one structure, in a simple running technique, using #1 or #2 absorbable monofilament suture material with a suture length to wound length ratio of 4 to 1.

References

1. Riou JP, Cohen JR, Johnson H. Factors influencing wound dehiscence. Am J Surg 1992; 163: 324–330
2. Poole GV. Mechanical factors in abdominal wound closure: The prevention of fascial dehiscence. Surgery 1985; 97:631–639
3. Brennan TG, Jones NAG, Gillou PJ. Lateral paramedian incision. Br J Surg 1987; 74: 736–737
4. Gilbert JM, Ellis H, Foweraker S. Peritoneal closure after lateral paramedian incision. Br J Surg 1987; 74: 113–115
5. Donaldson DR, Hegarty JH, Brennal TG. The lateral paramedian incision – experience with 850 cases. Br J Surg 1982; 69:630–632
6. Donaldson DR, Hall TJ, Zoltowski JA. Does the type of suture material contribute to the strength of the lateral incision? Br J Surg 1982; 69: 163–165
7. Giullou PJ, Hall TJ, Donaldson DR. Vertical abdominal incision – a choice? Br J Surg 1980; 67: 395–399
8. Cox PJ, Ausobsky JR, Ellis H, et al. Towards no incisional hernias: lateral paramedian versus midline incisions. J R Soc Med 1986; 79: 711–712
9. Dudley HAF. Layered and mass closure of the abdominal wall—A theoretical and experimental analysis. Br J Surg 1970; 57: 664–667
10. Golligher JC, Irvin TT, Johnston D. A controlled clinical trial of three methods of closure of laparotomy wounds. Br J Surg 1975; 62: 823–829
11. Bucknall TE, Cox PJ, Ellis H. Burst abdomen and incisional hernia: a prospective study of 1129 major laparotomies. Br Med J 1982; 284:931–933
12. Lewis RT, Wiegand FM. Natural history of vertical abdominal parietal closure: Prolene vs dexon. Can J Surg 1989; 32: 196–200.
13. Wasiljew BK, Winchester DP. Experience with continuous absorbable suture in the closure of abdominal incisions. Surg Gynecol Obstet 1982;154: 378–380
14. Hugh TB, Nankivell C, Meagher AP, Li B. Is closure of the peritoneal layer necessary in the repair of midline surgical abdominal wounds? World J Surg 1990; 14: 231–234
15. No authors listed. Why suture the peritoneum? Lancet 1987; 1: 727
16. Kendall SWH, Brennan TG, Guillou PJ. Suture strength to wound length ratio and the integrity of midline and lateral paramedian incisions. Br J Surg 1991; 78: 705–707
17. Stark M. Clinical evidence that suturing the peritoneum after laparotomy is unnecessary for healing. World J Surg 1993; 17: 419
18. Ellis H, Heddle R. Does the peritoneum need to be closed at laparotomy? Br J Surg 1977; 64: 733–736
19. Chana RS, Sexena VC, Agarwall A. A prospective study of closure techniques of abdominal incisions in infants and children. J Indian Med Assoc 1993; 91: 561
20. Spencer EE, Akuma A. Layered versus mass closure of vertical midline laparotomy wounds in Negro Africans. Trop Doct 1988; 18: 67–69
21. Kiely EM, Spitz I. Layered versus mass closure of abdominal wound in infants and children. Br J Surg 1985; 72: 739–740
22. Hoerr SO, Allen R, Allen K. The closure of the abdominal incision: a comparison of mass closure with wire and layered closure with silk. Surgery 1951; 30: 166–173
23. Humphries AL, Corley WS, Moretz WH. Massive closure versus layer closure for abdominal incisions. Am Surg 1964; 30: 700–705
24. Leaper DJ, Pollock AV, Evans M. Abdominal wound closure: a trial of nylon olyglycolic acid and steel sutures. Br J Surg 1977; 64: 603–606
25. Irvin TT, Stoddard CJ, Creaney MJ, et al. Abdominal wound healing: a prospective clinical study. Br Med J 1977; 2: 351–352
26. Weiland DE, Bay C, Del Sordi S. Choosing the best abdominal closure by meta-analysis. Am J Surg 1998;176: 666–670
27. van't Riet M, Steyerberg EW, Nellensteyn J, Bonjer HJ, Jeekel J. Meta-analysis of techniques for closure of midline abdominal incisions. Br J Surg 2002; 89: 1350–1356
28. Rucinski J, Margolis M, Panagopoulos G, Wise L. Closure of the abdominal midline fascia: Meta-analysis delineates the optimal technique. Am Surg 2001; 67: 421–426
29. Cleveland RD, Zitsch RP, Laws HL. Incisional closure in morbidly obese patients. Am Surg 1989; 55: 61–63
30. Fagniez P, Hay JM, Lacaine F, Thomsen C. Abdominal midline incision closure. Arch Surg 1985; 120: 1351–1353
31. McNeill PM, Sugerman HJ. Continuous absorbable versus interrupted nonabsorbable fascial closure. Arch Surg 1986; 121: 821–823
32. Richards PC, Balch CM, Aldrete JS. Abdominal wound closure. A randomized prospective study of 571 patients comparing continuous vs. interrupted suture techniques. Ann Surg 1983; 197:238–243

33. Gislason H, Gronbech JE, Soreide O. Burst abdomen and incisional hernia after major gastrointestinal operations – comparison of three closure techniques. Eur J Surg 1995; 161:349–354
34. Trimbos JB, van Rooji J. Amount of suture material needed for continuous or interrupted wound closure: An experimental study. Eur J Surg 1993; 159: 141–143
35. Colombo M, Maggioni A, Parma G, Scalambrino S, Milani R. A randomized comparison of continuous versus interrupted mass closure of midline incisions in patients with gynecologic cancer. Obstet Gynecol 1997; 89: 684–689
36. Brolin RE. Prospective, randomized evaluation of midline fascial closure in gastric bariatric operations. Am Surg 1996; 172: 328–332
37. Trimbos JB, Smith IB, Holm JP, Hermans J. A randomized clinical trial comparing two methods of fascia closure following midline laparotomy. Arch Surg 1992; 127: 1232–1234
38. Sahlin S, Ahlberg J, Grantstrom L, Ljungstrom KG. Monofilament versus multifilament absorbable sutures for abdominal closure. Br J Surg 1993; 80: 322–324
39. Rodeheaver GT, Powell TA, Thacker JG, Edlich RF. Mechanical performance of monofilament synthetic absorbable sutures. Am J Surg 1987; 154: 544–547
40. Poole GV, Meredith JW, Kon ND, Martin MB, Kawamoto EH, Myers RT. Suture technique and wound-bursting strength. Am Surg 1984; 50:569–572
41. Hodgson NC, Malthaner RA, Ostbye T. The search for an ideal method of abdominal fascial closure: a meta-analysis. Ann Surg 2000; 231: 436–442
42. Alexander HC, Prudden JF. The causes of abdominal wound disruption. Surg Gynecol Obstet 1966; 122: 1223–1229
43. Wadstrom J, Gerdin B. Closure of the abdominal wall: how and why? Acta Chir Scand 1990; 156: 75–82
44. Rath AM, Chevrel JP. The healing of laparotomies: a review of the literature. Part 1. Physiologic and pathologic aspects. Hernia 1998; 2: 145–149
45. Douglas DM. The healing of aponeurotic incisions. Br J Surg 1952; 40: 79–84
46. Luijendijk RW. Incisional hernia; risk factors, prevention, and repair. Thesis. Erasmus University, Rotterdam. Scheveningen: Drukkerji Edauw and Johannissen, 2000
47. Wissing J, van Vroonhoven TJMV, Eeftinck Schattenkerk M, et al. Fascia closure after laparotomy: Results of a randomized trial. Br J Surg 1987; 74: 738–741
48. Bucknall TE, Teare L, Ellis H. The choice of suture to close abdominal incisions. Eur Surg Res 1983; 15: 59–66
49. Bucknall TE. Factors influencing wound complication: A clinical and experimental study. Ann R Coll Surg Engl 1983; 65: 71–77
50. Sharp WV, Belden TA, King PH, Teague PC. Suture resistance to infection. Surgery 1982; 91: 61–63
51. Krukowski ZH, Matheson NA. "Button-hole" incisional hernia: A late complication of abdominal wound closure with continuous non-absorbable sutures. Br J Surg 1987; 74: 824–825
52. Larsen PN, Nielsen K, Schultz A, Mejdahl S, Larsen T, Moesgaard F. Closure of the abdominal fascia after clean and clean-contaminated laparotomy. Acta Chir Scand 1989; 155: 461–464
53. Corman ML, Veidenheimer MC, Coller JA. Controlled clinical trial of three suture materials for abdominal wall closure after bowel operations. Am J Surg 1981; 141: 510–513
54. Knight CD, Griffen FD. Abdominal wound closure with a continuous monofilament polypropylene suture. Arch Surg 1983; 118: 1305–1308
55. Bucknall TE, Ellis H. Abdominal wound closure: a comparison of monofilament nylon and polyglycolic acid. Surgery 1981; 89: 672–677
56. Schoetz DJ, Coller JA, Veidenheimer MC. Closure of abdominal wounds with polydioxanone. Arch Surg 1988; 123:72–74
57. Ray JA, Doddi N, Regula D, Williams JA, Melveger A. Polydioxanone (PDS), a novel monofilament synthetic absorbable suture. Surg Gynecol Obstet 1981; 153:497–507
58. Gys T, Hubens A. A prospective comparative clinical study between monofilament absorbable and non-absorbable sutures for abdominal wall closure. Acta Chir Belg 1989; 89:265–270
59. Israelsson LA, Jonsson T. Closure of midline laparotomy incisions with polydioxanone and nylon: the importance of suture technique. Br J Surg 1994; 81: 1606–1608
60. Carlson MA, Condon RE. Polyglyconate (Maxon) versus nylon suture in midline abdominal incision closure: a prospective randomized trial. Am J Surg 1995; 61: 980–983
61. Krukowski ZH, Cusick EL, Engeset J, Matheson NA. Polydioxanone or polypropylene for closure of midline abdominal incisions: a prospective comparative clinical trial. Br J Surg 1987; 74: 828–830
62. Wallace D, Hernandez W, Schlaerth JB, Nalick RN, Morrow CP. Prevention of abdominal wound disruption utilizing the Smead-Jones closure technique. Obstet Gynecol 1980; 56:226–230
63. Gallup DG, Talledo OE, King LA. Primary mass closure of midline incisions with a continuous running monofilament suture in gynecologic patients. Obstet Gynecol 1989; 73:675–677
64. Niggebrugge AH, Trimbos JB, Hermans J, Steup WH, Van de Velde CJ. Influence of abdominal wound closure technique on complications after surgery: a randomized study. Lancet 1999; 353: 1563–1567
65. Jenkins TPN. The burst abdominal wound: a mechanical approach. Br J Surg 1976; 63: 873–876
66. Israelsson LA, Jinsson T. Suture length to wound length ratio and healing of midline laparotomy incisions. Br J Surg 1993; 80: 1284–1286
67. Varshney S, Manek P, Johnson CD. Six-fold suture: wound length ratio for abdominal closure. Ann R Coll Surg Engl 1999; 81: 333–336

Discussion

Deysine: *In the 1970s Dr. Goligher introduced a continuous suture with nylon for the closure of laparotomies. At that time the number of laparotomies exploded in the world because of vascular surgery and they used be closed by a running suture. This technique by Dr. Golligher is very well depicted and those who practice it, like me, are*

very happy with it. It is a continuous suture with a thick no.1 nylon and it accommodates to the changes in the abdominal wall and, to my surprise, it does not include the skin but all the other layers; the patients have very little pain with this kind of closure.

Ceydeli: *Yes, in the NY State survey also the nonabsorbable, monofilament nylon suture was the most common suture but in the review the most common one was PDS, late absorbable.*

Jeekel: *But nylon causes more pain.*

Amid: *We really need a correct terminology. The most common mistake that is made is the issue of fascia vs. aponeurosis. When we close midline the abdominal wall we don't close fascia, we close the linea alba or rectus sheath; the fascia is a very thin investing layer of the muscle that has absolutely no role in hernia surgery.*

Jeekel: *The suture-length-wound-length ratio, please one remark to small or large bites.*

Israelsson: *I was a bit concerned about the recommendation of taking 2-cm-large bites. There are several clinical studies that show that by taking that big size of the bite you will end up with a high rate of incisional hernia and wound infection. There is also strong evidence by experimental studies that a suture-length-wound-length ratio of 4:1 should be achieved by small tissue bites at short intervals.*

Jeekel: *But this is only experimental evidence.*

15 Closure of Transverse Incisions

J.A. Halm, J. Jeekel

Incisions

Any incision chosen for access to the abdominal cavity needs to provide access to the viscera or the lesion to be treated. Furthermore, an incision needs to provide extensibility and permit subsequent secure closure. A further demand may be the postoperative preservation of function [1] such as containment of abdominal organs and respiration. Additional considerations in choosing the incision are the speed of entry, presence of scars, possibility of hemostasis and a cosmetically pleasing outcome.

Secure closure must be possible and various suture materials are used in this day and age. Suture materials should ideally: be sufficient to hold parts together; disappear as soon as its work is accomplished; be free of infection; and be non-irritant.

To appreciate the different incisions and problems with closure, thorough knowledge of the anatomy of the abdominal wall is mandatory.

Anatomy Ventral Abdominal Wall

The ventral abdominal wall consists of the rectus abdominis muscle on contralateral sides of the line alba. The origo of the rectus muscle are the 5th, 6th and 7th rib, the insertion is the pubic bone. The rectus muscles are each contained in a fascial layer, the anterior and posterior rectus sheath, which is made up of the aponeurosis (insertion) of the internal, external and transverse muscle. The rectus muscle is horizontally incised by the three inscriptiones tendinea. Lateral to the rectus abdominis the abdominal wall is made up of the afore-mentioned external oblique, the internal oblique and the transverse muscle, which extend over the ventral and lateral part of the abdomen (the part not covered by the rectus muscle). The origo of the external oblique muscle runs from the 5th to the 12th rib. The internal oblique originates from the iliac crest. The transverse muscle, with its horizontal fibre direction, originates from the previously mentioned iliac crest, the lumbodorsal fascia and the lower six ribs superiorly. The lateral border of the rectus muscle forms the linea semilunaris. At the symphysis pubis the posterior sheath ends in the thin curved margin, the linea semicircularis (Douglasi). Below this level the aponeuroses of all three muscles passes in front of the rectus abdominis and the fascia transversalis is responsible for the separation of the rectus from the peritoneum. The pyramidalis muscle (if present) lies anterior to the lower part of the rectus abdominis muscle. It arises from the superior surface of the pubic ramus and inserts at the linea alba.

The vasculature of the muscles of the abdominal wall consists of the superior and inferior deep epigastric vessels as well as transverse segmental branches of the aorta. The superior and inferior deep epigastrics are located in front of the posterior rectus sheath and the rectus muscle and form its blood supply through perforating vessels. The inferior deep epigastric ar-

tery branches from the external iliac artery whereas the superior deep epigastric is a branch of the internal thoracic artery. The deep epigastric arteries are anastomosed and thus form the deep epigastric arcade. The transverse segmental arteries supply the transverse muscle, the internal and external oblique and are situated between the transverse and internal oblique. Blood supply to the relatively avascular linea alba originates from the perforating vessels of the superior and inferior deep epigastrics.

Innervation of the abdominal wall is achieved through intercostals nerves, the ilioinguinal and the iliohypogastric nerve. The intercostals nerves are ventral branches of thoracic nerves originating from levels Th 5 through Th 12 of the spinal cord.

Midline Incisions

The midline incision is possibly the most popular incision amongst surgeons today. When investigating alternatives to it, the baseline characteristics need to be described. Midline incisions incise the skin, subcutaneous tissue, linea alba and the peritoneum vertically. Midline incisions are easy, relatively little blood is lost and the incision takes an average of 7 min to perform [2–4]. The exposure achieved through a midline incision encompassing the umbilicus is excellent, and includes access to the retroperitoneum. The upper or lower abdominal midline incisions may be utilized in case the expected pathology is situated in the upper or lower quadrants of the abdomen respectively. Extensions may be made in cranial or caudal direction when deemed necessary. The qualities mentioned above make the midline incision the most ideal for emergency and exploratory surgery.

Transverse Incision

Transverse incisions are possible at all levels of the abdomen. Common examples are the Pfannenstiel incision just above the pubic bone and the upper right quadrant transverse incision just below the costal margin.

The Pfannenstiel incision is approximately 8–12 cm in length (distance between the superfiscial epigastric arteries) and transsects the superficial fascia and the fibrous rectus sheath. Further access is achieved by a slightly more cranial, vertical incision of the fascia transversalis, the preperitoneal fat and the peritoneum [5]. Luijendijk has described incisional hernia formation in Pfannestiel incisions most recently and came to 2.1% in 243 patients after a follow-up between 1.6 and 7.8 years [6].

The upper right quadrant transverse incision requires transsection of the oblique and transverse musculature as well as the rectus muscle. The linea alba is incised most commonly when extending the transverse incision across the midline. Dividing the rectus muscle requires ligating the epigastric arcade yet poses minor damage to the intercostals nerves and superficial arteries supplying the transverse and oblique musculature [7]. The transverse incision is thus accompanied by more blood loss than the midline incision and takes longer to achieve [4, 8]. Exposure of the lesion is generally good, although unilateral incisions may provide a somewhat limited view.

Closure of Incisions

Midline Closure

Studies describing closure of incisions have been performed focusing on continuous, interrupted, layered closure and various suture materials (absorbable and non-absorbable). A recent meta-analysis reviewed 13 [9–21] clinically homogeneous randomized controlled trials comparing absorbable, non-absorbable, continuous and interrupted closure of abdominal incisions [22]. Non-absorbable sutures were found to reduce incisional herniae when compared with absorbable sutures. The odds ratio (OR) favouring non-absorbable sutures was 0.68 (95% CI 0.52–0.87) combining data from nine trials [9–12, 15–18, 21]. Neither wound infection nor wound dehiscence was statistically more likely in absorbable sutures. In contrast, suture sinuses and wound pain were significantly more frequent in the non-absorbable suture group with respective odds ratios of 2.18 (95% CI 1.48–3.22) and 2.05 (95% CI 1.52–2.77).

Six trials were identified in the afore-mentioned meta-analysis comparing interrupted and continuous suture technique disregarding suture type [9, 12, 14, 17, 20, 21]. Continuous sutures compared favourably to interrupted sutures (OR 0.73; 95% CI 0.55–0.99). No statistical differences were found for wound dehiscence and wound infection.

When taking into account the differences in technique (nine trials), continuous non-absorbable suturing outperformed the continuous absorbable suture in incisional hernia prevention (OR 0.61; 95% CI 0.46–0.8) [9–11, 14, 16–18, 21]. No significant differences were found when comparing interrupted absorbable and interrupted non-absorbable closure.

A subgroup analysis revealed that use of slowly absorbable polydioxanone (PDS) and polyglycolic acid (Dexon) did not significantly increase the risk for incisional hernia formation compared to polypropylene. Polyglactin (Vicryl) compared unfavourably with non-absorbable sutures. Previously Wissing et al. have found that nylon has the lowest incidence of incisional hernia yet is unfavourably associated with more wound pain and suture sinuses than polydioxanone sutures [21].

Transverse Closure

Randomized studies, not mentioned earlier, specifically describing incisional hernia formation with respect to midline, transverse and oblique incisions are summarized in ◘ Table 15.1. Transverse incisions were found to be prone to incisional hernia formation in 3.6 – 40% of patients. Fassiadis et al. used continuous single-layered closure with nylon in the trial reported. The hernia incidence in high-risk patients undergo-

◘ **Table 15.1.** Randomized studies on incisional hernia

Author	Year	Patients [N]	Incision(s)	Follow-up [months]	Rate of incisional hernia [%]	Technique, suture type, layers [L]	p value
Blomstedt [24]	1972	30	Transverse	8–24	9.5	Various suturesa, 2 L	ns <0.01
	RCT	115	Midline		13.9	Various suturesa, 1 L	
		80	Oblique		3.8	Various suturesa, 2 L	
Greenall [8]	1980	235	Transverse	>6	6.4	Variousa, 1 L, cont.	ns
	RCT	234	Midline	>6	8.1	Variousa, 1 L, cont.	
Ellis [25]	1984	50	Transverse	<12	14.0	Nylon, 1 L, cont.	ns
	RCT	46	Paramedian	<12	17.4	Nylon, 1 L, cont.	
Schoetz [26]	1988	28	Transverse	1–12	3.6	PDS, 1 L, cont.	ns
		172	Midline	1–12	2.9	PDS, 1 L, cont.	
Lord [27]	1994	126	Transverse	12–72	13.5	Nylon, 2 L, cont.	ns
	RCT	109	Midline		16.5	Nylon, 1 L, cont.	
Fassiadis [23]	2005	15	Transverse	>48	40	Nylon, 1 L, cont.	<0.01
	RCT	22	Vertical		91	Nylon, 1 L, cont.	
Halm	Sub.	60	Transverse	12–36	2	Vicryl, 2 L, comb.	p = 0.02
	RCT	63	Vertical		14	Vicryl, 1 L, inter.	

[a]absorbable/non-absorbable. *RCT* randomized controlled trial; *ns* not significant; *cont.* continuous; *inter.* interrupted; *comb.* one layer cont. and one layer inter.; *L* layer; *sub.* submitted

ing abdominal aortic aneurysm surgery was reported to be 40%. In the transverse incisions studied by Fassiadis (using ultrasound) the incisional hernias were found predominantly at the lateral border [23].

Schoetz found the most encouraging results in closure of transverse incisions, 3.6% incisional hernia incidence after continuous closure with polydioxanone.

No studies were found specifically comparing different methods of closure (materials or technique) for the transverse incision.

Currently unpublished (submitted) results from a randomized study (n = 150) performed at our own institute confirmed the results that transverse incisions (2% incisional hernia) are significantly less likely to develop hernias compared to upper abdominal midline incisions (14% incisional hernia) in the patients seen at follow-up (◘ Table 15.1). Closure of the transverse incision of the abdominal wall was achieved by closure of the peritoneum and the posterior rectus fascia using a continuous, polyglactin 910 suture (Vicryl). The anterior rectus sheath and the fascia of the internal and external transverses were closed using simple interrupted polygalactin 910 sutures (Vicryl).

Complications: Pain, Wound Infection and Burst Abdomen

Armstrong et al., reporting a randomized study comparing midline and transverse incisions in 60 patients, have documented significantly reduced postoperative pain for transverse incisions [28], a result that we confirmed in our own (submitted) randomized trial. Halasz et al. found a reduction in the use of analgesics in patients after an oblique incision when compared to a paramedian approach [29]. A similar result was found by Garcia-Valdecasas comparing oblique to midline incisions [30]. The review by Burger et al. concluded that none of the trials performed to date reported a significant difference in surgical site infection rates [31].

Burst abdomen has an incidence between 0 and 2.5% and was found to be more likely after vertical incisions. Pooling of data by Grantcharov and coworkers revealed a significant difference between the incidence of burst abdomen after vertical incision of 1% (46/4480) and after transverse incision of 0.34% (15/4365) [32]. An odds ratio of 2.86 favouring transverse incision 95% CI 1.72–4.73 was subsequently calculated (◘ Table 15.2).

Randomized Controlled Trial

The POVATI trial (ISRCTN 60734227), as initiated by researchers from Heidelberg, Germany (Prof. Dr. M.W. Büchler), compares the two most common incisions in general surgery, midline and transverse [34].

The trial, which was started in July 2003, proposes abdominal wall closure in a standardized way in both groups: four Mikulicz clamps are to be placed at the edges of the abdominal fascia and a continuous, all-layer closure technique with two Mono Plus loops

◘ **Table 15.2.** Data on burst abdomen incidence

Author	Type of publication	No. of patients	Incision(s)	Rate of burst abdomen [%]	p value
Greenall [8]	RCT	292	Transverse	0	0.2453
		287	Midline	0.69	
Thompson [33]	Retr.	760	Transverse	0.5	0.004
		603	Midline	2.5	
Halasz [29]	Retr.	3313	Transverse	0.33	0.009
		3590	Midline	0.81	

RCT randomized controlled trial; *Retr.* retrospective

(Aesculap, Tuttlingen, Germany) performed, starting from both ends of the incision with a 4:1 ratio (suture length:wound length). Neither subcutaneous closure nor subcutaneous drainage is proposed. Skin closure is to be achieved with skin clips.

Primary outcome measures are the requirement of analgesics and patient satisfaction. Secondary outcomes are incisional hernia 1 year postoperative (diagnosed by ultrasound). Burst abdomen, pulmonary infection and wound infection are secondary endpoints, but are also defined as adverse events.

Closure of the Transverse Incision: How We Do It

Currently, hepaticopancreaticobilliary surgeons of the Erasmus MC propose double-layered closure of transverse incisions, reasoning that the cosmetic outcome is more pleasing since, in their experience, the skin inadvertently inverts when single-layered closure is employed.

In detail, a USP 0 PDS loop (Ethicon, Johnson & Johnson Amersfoort) is used to close the posterior fascia in a continuous fashion starting at the lateral border of the incision. Upon reaching the medial border of the incision, the same loop, without interruption, is employed to approximate the anterior fascia and the internal and external obliques. A suture-length-to-wound-length ratio of 4 to 1 is maintained throughout. Subcutaneous closure is achieved in case the dead space observed is deemed too large in the eyes of the surgeon. For reduction of dead space interrupted Vicryl (Ethicon, Johnson & Johnson, Amersfoort) sutures are used. Skin closure is achieved by intracutaneous, continuous suturing using Monocryl 5–0 (Ethicon, Johnson & Johnson, Amersfoort, The Netherlands).

Conclusion

Closure of transverse incisions can be achieved securely using single as well as double-layered closure. Non-absorbable or slowly absorbable sutures seem to be advantageous in the prevention of incisional hernia, as is continuous suturing technique. Slowly absorbable sutures seem to reduce the incidence of wound pain and suture sinuses. Further research in the form of randomized controlled trials seems warranted in light of the lack of data on the topic of transverse closure techniques.

References

1. Skandalakis LJ, Gadacz TR, Mansberger AR, et al. Modern Hernia Repair: the embryological and anatomical basis of surgery. New York: Parthenon Publishing Group, 1996
2. Guillou PJ, Hall TJ, Donaldson DR, et al. Vertical abdominal incisions – a choice? Br J Surg 1980; 67(6): 395–399
3. Kendall SW, Brennan TG, Guillou PJ. Suture length to wound length ratio and the integrity of midline and lateral paramedian incisions. Br J Surg 1991; 78(6): 705–707
4. Lacy PD, Burke PE, O'Regan M, et al. The comparison of type of incision for transperitoneal abdominal aortic surgery based on postoperative respiratory complications and morbidity. Eur J Vasc Surg 1994; 8(1): 52–55
5. Pfannenstiel HJ. Ueber die Vortheile des suprasymphysären Fascienquerschnitts für die gynäkologischen Koeliotomien, zugleich ein Beitrag zu der Indikationsstellung der Operationswege. Volkmann's Sammlung klinischer Vorträge, Leipzig, 1900, n F. 268 (Gynäk. Nr. 97), 1735–1756
6. Luijendijk RW, Jeekel J, Storm RK, et al. The low transverse Pfannenstiel incision and the prevalence of incisional hernia and nerve entrapment. Ann Surg 1997; 225(4): 365–369
7. Nahai F, Hill L, Hester TR. Experiences with the tensor fascia lata flap. Plast Reconstr Surg 1979; 63(6): 788–799
8. Greenall MJ, Evans M, Pollock AV. Midline or transverse laparotomy? A random controlled clinical trial. Part I: Influence on healing. Br J Surg 1980; 67(3): 188–190
9. Bucknall TE, Ellis H. Abdominal wound closure--a comparison of monofilament nylon and polyglycolic acid. Surgery 1981; 89(6): 672–677
10. Cameron AE, Gray RC, Talbot RW, Wyatt AP. Abdominal wound closure: a trial of Prolene and Dexon. Br J Surg 1980; 67(7):487–488
11. Carlson MA, Condon RE. Polyglyconate (Maxon) versus nylon suture in midline abdominal incision closure: a prospective randomized trial. Am Surg 1995; 61(11): 980–983
12. Cleveland RD, Zitsch RP, 3rd, Laws HL. Incisional closure in morbidly obese patients. Am Surg 1989; 55(1): 61–63
13. Corman ML, Veidenheimer MC, Coller JA. Controlled clinical trial of three suture materials for abdominal wall closure after bowel operations. Am J Surg 1981; 141(4): 510–513
14. Irvin TT, Koffman CG, Duthie HL. Layer closure of laparotomy wounds with absorbable and non-absorbable suture materials. Br J Surg 1976; 63(10): 793–796
15. Kronborg O. Polyglycolic acid (Dexon) versus silk for fascial closure of abdominal incisions. Acta Chir Scand 1976; 142(1):9–12
16. Krukowski ZH, Cusick EL, Engeset J, Matheson NA. Polydioxanone or polypropylene for closure of midline abdominal incisions: a prospective comparative clinical trial. Br J Surg 1987; 74(9): 828–830
17. Larsen PN, Nielsen K, Schultz A, et al. Closure of the abdominal fascia after clean and clean-contaminated laparotomy. Acta Chir Scand 1989; 155(9): 461–464
18. Leaper DJ, Allan A, May RE, et al. Abdominal wound closure: a controlled trial of polyamide (nylon) and polydioxanone suture (PDS). Ann R Coll Surg Engl 1985; 67(5): 273–275
19. Lewis RT, Wiegand FM. Natural history of vertical abdominal parietal closure: Prolene versus Dexon. Can J Surg 1989; 32(3):196–200

20. Richards PC, Balch CM, Aldrete JS. Abdominal wound closure. A randomized prospective study of 571 patients comparing continuous vs. interrupted suture techniques. Ann Surg 1983; 197(2): 238–243
21. Wissing J, van Vroonhoven TJ, Schattenkerk ME, et al. Fascia closure after midline laparotomy: results of a randomized trial. Br J Surg 1987; 74(8): 738–741
22. Hodgson NC, Malthaner RA, Ostbye T. The search for an ideal method of abdominal fascial closure: a meta-analysis. Ann Surg 2000; 231(3): 436–442
23. Fassiadis N, Roidl M, Hennig M, et al. Randomized clinical trial of vertical or transverse laparotomy for abdominal aortic aneurysm repair. Br J Surg 2005; 92(10): 1208–1211
24. Blomstedt B, Welin-Berger T. Incisional hernias. A comparison between midline, oblique and transrectal incisions. Acta Chir Scand 1972; 138(3): 275–278
25. Ellis H, Coleridge-Smith PD, Joyce AD. Abdominal incisions--vertical or transverse? Postgrad Med J 1984; 60(704): 407–410
26. Schoetz DJ, Jr., Coller JA, Veidenheimer MC. Closure of abdominal wounds with polydioxanone. A prospective study. Arch Surg 1988; 123(1): 72–74
27. Lord RS, Crozier JA, Snell J, Meek AC. Transverse abdominal incisions compared with midline incisions for elective infrarenal aortic reconstruction: predisposition to incisional hernia in patients with increased intraoperative blood loss. J Vasc Surg 1994; 20(1): 27–33
28. Armstrong PJ, Burgess RW. Choice of incision and pain following gallbladder surgery. Br J Surg 1990; 77(7): 746–748
29. Halasz NA. Vertical Vs Horizontal Laparotomies. I. Early postoperative comparisons. Arch Surg 1964; 88: 911–914.
30. Garcia-Valdecasas JC, Almenara R, Cabrer C, et al. Subcostal incision versus midline laparotomy in gallstone surgery: a prospective and randomized trial. Br J Surg 1988; 75(5): 473–475
31. Burger JW, van 't Riet M, Jeekel J. Abdominal incisions: techniques and postoperative complications. Scand J Surg 2002; 91(4): 315–321
32. Grantcharov TP, Rosenberg J. Vertical compared with transverse incisions in abdominal surgery. Eur J Surg 2001; 167(4):260–267
33. Thompson JB, MacLean KF, Coller FA. Role of the transverse abdominal incision and early ambulation in the reduction of postoperative complications. Arch Surg 1949; 59(6): 1267–1277
34. Reidel MA, Knaebel HP, Seiler CM, et al. Postsurgical pain outcome of vertical and transverse abdominal incision: design of a randomized controlled equivalence trial [ISRCTN60734227]. BMC Surg 2003; 3: 9

Discussion

Schumpelick: *How should we close transverse incisions, what is your recommendation: single or double layer?*
Jeekel: *I close by single layer when it is a small muscle and when it is a big muscle I do a double layer.*

16 Biological Reasons for an Incisional Hernia

J.M Bellón

Introduction

Incisional hernia continues to represent a significant problem within the context of abdominal wall pathologies.

The incidence of incisional hernia has remained constant over the past decade, despite numerous modifications in the techniques and materials used. It is a frequent complication of abdominal surgery, with a reported incidence of 2–11%. After procedures such as aortic surgery, the rate can be as high as 16–20%. In the USA, 4 to 5 million laparotomies are performed annually, which means that at least 400,000 to 500,000 incisional hernias can be expected to develop each year. Incisional hernia repair is performed approximately 200,000 times per year. The total financial cost of these operations could be around 2.5 billion dollars [1].

In general, the wound-healing process can be divided into three stages: an inflammatory stage, a fibroplastic stage and a stage of maturation. The inflammatory stage lasts for 4–6 days, during which time the wound is prepared for subsequent healing by removal of necrotic tissue and bacteria. During this period, the wound has no intrinsic strength and its integrity is entirely dependent on the suture and the suture-holding capacity of the tissues. This stage is followed by a fibroplastic phase characterized by collagen synthesis. During this second stage, the wound rapidly gains in tensile strength by the bridging over of collagen fibres. The fibroplastic stage is gradually followed by a prolonged phase of maturation in which collagen fibres are remodelled.

The tensile strength of a sutured aponeurosis after 2–3 weeks is about 20% that of unwounded tissue, and after 4 weeks is about 50%. After 6–12 months, the aponeurosis attains about 80% of its original strength, but complete recovery is never achieved.

Factors Contributing to the Genesis of Incisional Hernia

Why do incisional hernias occur? Incisional hernias occur as the result of a biomechanical defect in acute fascial wound healing, which affects the normal capacity of the abdominal wall to support increasing tension during the postoperative recovery period.

Most studies now support the theory that acute fascial separation occurs early in the postoperative period, during the course of acute wound healing at a time when wound tensile strength is very low or absent (postoperative days 0–30), and leads to the delayed clinical development of abdominal wall incisional hernias [2].

It is during this early period of acute wound healing that the scar depends entirely on the integrity of the suture to keep the abdominal wall closed. This integrity, in turn, also depends on the success of the wound repair process in each individual.

Several factors have been implicated in the aetiology and pathogenesis of the incisional hernia [3].

The most frequently identified clinical risk factors for fascial wound failure and primary incisional hernia formation include:

- Type of laparotomy
- Suboptimal closure technique
- Infections
- Malnutrition
- Preoperative hypotension
- Jaundice, anaemia, corticosteroid therapy
- Biological disorders (collagen-related)

Transverse laparotomies generally show a lower incidence of incisional hernia than vertical ones [4].

Many laparotomy closures are incorrectly undertaken and basic rules such as the 4:1 Jenkins rule are neglected [5]. In many cases, closure is undertaken by surgeons early on in the learning curve with insufficient training.

Infection has been directly linked to over 75% of incisional hernias. In addition, malnutrition and substantial blood loss during surgery have been related to a greater incidence of incisional hernia. Other factors such as jaundice, anaemia and steroid treatment interfere with the entire healing process in general and therefore contribute to the appearance of this abdominal wall pathology.

Finally, there is also a series of factors related to the tissue biology of each individual. These factors are associated with the biological wound repair, or scarring process. The scarring process in one subject obviously differs to that in another, mainly because of tissue components and inducers that mediate the process.

Biological factors include the components of the extracellular matrix such as collagens and the enzymes metalloproteinases (MMPs). Exogenous variables can also predispose an individual to incisional hernia such as smoking or a concurrent disease whose underlying cause is a collagen alteration, including aortic aneurysm, cutis laxa, Marfan's syndrome, osteogenesis imperfecta, and Ehlers-Danlos syndrome.

Biological Factors

The search for biological factors involved in the appearance of incisional hernia has been limited, unlike the case for biological factors contributing to the genesis of groin hernias. This is possibly because the pathogenesis of incisional hernia depends on many other factors other than those strictly classed as biological factors.

Biological factors, in an individual manner, closely modulate the repair process at the level of the fascia; this is the only retaining structure after a laparotomy closure.

In fascial tissue, the mechanisms regulating the proliferative and synthesizing capacity of fibroblasts have not yet been defined. Neither do we know the reason for the failure of a surgical wound that generates incisional hernias.

To date, it has not been possible to establish a correlation between the proliferative response of fascial fibroblasts at the level of the cell cycle and wound healing failure [2].

Ischemia at the level of the fascial continuum could arrest the cell cycle of the fibroblast as a reparatory cell. This could occur in a technically deficient closure (when the suture is too tight or closure is under tension) or in cases of sustained intra-operative hypotension when the oxygen supply to the tissues is reduced.

Notwithstanding, in the past few years some investigations have centred on those factors or diseases that could condition the appearance of an incisional hernia following laparotomy. Many of the factors identified so far have also been implicated in the genesis of other types of hernia such as groin hernias.

Experimental Models

Role of Cytokines: TGF-beta and FGFb

In a rat model, Franz et al. [6] created incisional hernias after performing a midline laparotomy closed with a suture that was absorbable in the short term. This generates a defect in the abdominal wall that produces a postlaparotomy hernia. Topical treatment of laparotomy closures with recombinant TGF-β2 in an aqueous medium has been noted to diminish the appearance of incisional hernia and to increase fibroblasts, and collagen type-I and -III deposition, detected by immunohistochemistry.

Using the same experimental model, DuBay et al. [7] reported that by treating the fascia with FGFb loaded in a polymer vehicle, the appearance of incisional hernia was significantly reduced. In animals treated with this growth factor, angiogenesis and collagen deposition were also found to improve.

Another hypothesis proposed by the group of Franz and Dubay [6,7], is that the aponeurotic tissue of the abdominal wall is also dependent on mechanical signals to regulate the homeostasis of the fascial fibroblast. This mechanico-transduction theory pro-

poses that the load on soft tissue or bone is transmitted to structural cells through the extracellular matrix, and that there are integrin type receptors on the cell surface. Mechanical failure or reduced mechanical signals, for instance, when a suture fails, could lead to the impaired kinetics and proliferative capacity of the reparative fibroblast.

It has been well established that during the repair of tendons and ligaments, the mechano-transduction pathway is important for triggering the repairing actions of fibroblasts. A wound in the fascia could show similar behaviour.

Clinical Studies

Role of Collagen

Collagen plays a predominant role in any wound-repair process. It constitutes the main axis of wound healing along with the enzymes metalloproteinases (MMPs), which balance their production and lysis.

Klinge et al. [8] observed an imbalance between collagen I and III in patients with inguinal and incisional hernia.

In cultures of fibroblasts taken from the skin of patients with recurrent incisional hernia, Si et al. [9] also noted an imbalance between collagen type I and III. These authors also reported generally disorganised levels of collagens in the extracellular matrix.

Rosch et al. [10] also described a reduction in the collagen I/III ratio in patients with incisional hernia.

MMPs and Incisional Hernia

A balance between extracellular matrix synthesis and degradation is important for tissue integrity, because remodelling occurs continuously. MMPs are the enzymes that regulate the components of the extracellular matrix. Changes or defects in matrix molecules may also alter tissue architecture, impairing the proper assembly of the matrix components and modifying the mechanical properties of the tissue. Some of these enzymes play an important role in the general scarring process [11,12]. Thus, wounds that are difficult to repair such as in patients with diabetes show high MMP levels. In these patients, skin fibroblasts have been found to show increased amounts of MMP-2 [13].

In incisional hernias, Klinge et al. [14] found reduced MMP-1 expression compared to controls through Western blot analysis of fascial tissue.

Aortic Aneurysm and Incisional Hernia

The relationship among disorders in which extracellular matrix components are involved, such as aortic aneurysm, has been widely described in the literature.

Stevick et al. [15] first pointed out the link between post-laparotomy incisional hernia and aortic aneurysm, although Cannon et al. [16], had previously observed a relationship between patients with inguinal hernia and aneurysm.

In subsequent studies [17–19], a high incidence of aortic aneurysm was correlated with a similar incidence of incisional hernia.

The rate of incisional hernia has been reported to be as high as 31% following midline laparotomy for abdominal aortic-aneurysm repair [20, 21]. In a recent randomized study performed on patients undergoing surgery for aortic aneurysm, Fassiadis et al. [22] noted a lower incidence of incisional hernia in transverse laparotomies compared to midline procedures.

Alterations to the extracellular matrix have been reported by several authors.

In 1993, White et al. [23] reported that adventitial elastolysis was a primary event in aneurysm formation. Later, enhanced MMP-2 and MMP-9 expression was reported by Patel et al. [24], Skalihasan et al. [25], and Tamarina et al. [26]. In cultured muscle cells harvested from the medial layer of the aortic aneurymal wall, increased MMP-2 expression has been described [27].

Smokers

Smokers have a high risk of incisional hernia formation independent of other recognized risk factors, presumably owing to the detrimental effect of smoking on wound healing. Diminished collagen deposition in surgical test wounds has been observed in smokers [28].

The link between inguinal hernia, aortic aneurysm and smoking was first suggested by Read [29]. According to Read, the degradation of connective tissue caused by imbalance between proteases and their inhibitors could also be a contributing factor. Smoking has been related to increased proteolytic activity, activation of neutrophils and macrophages and the release of oxidants, impairing the antiprotease defence mechanism, leading to increased collagenolysis and inappropriate repair [30].

In a recent study, Sorensen et al. [31] linked smoking with the appearance of incisional hernia. In this study, the incidence of incisional hernia is four times higher in smokers than non-smokers. A relationship between smoking and hernia recurrence had already been reported [32] in a study in which recurrence was found to occur more frequently in smokers undergoing herniorraphy.

In general terms, all the biological factors that could induce the appearance of an incisional hernia are inter-related. It thus becomes obvious that in the absence of other risk factors (infection, an inappropriate closure technique, malnutrition, jaundice etc.), the biology of the individual plays a pivotal role. Hence, when several biological risk factors are present these could have a synergistic effect on the repair process.

A smoker who also has a collagen disorder will have a greater risk of developing an incisional hernia after a laparotomy. This would explain why hernia recurrence sometimes occurs after the successful surgical repair of an incisional hernia. This event was described in a recent report [33], in which recurrence mechanisms of operated incisional hernias were classified.

References

1. Wedbush Morgan Securities. Biotechnology in wound care 2001; 4: 1–82
2. Franz MG, Robson MC. The use of the wound healing trajectory as an outcome determinant for acute wound healing. Wound Repair Regen 2001; 8: 511–516
3. Carlson MA. Acute wound failure. Wound healing. Surg Clin North Am 2001; 77: 607–635
4. Grantcharov TP, Rosenberg J. Vertical compared with transverse incisions in abdominal surgery. Eur J Surg 2001; 167: 260–267
5. Jenkins TNP. The burst abdominal wound: a mechanical approach. Br J Surg 1976; 63: 837–876
6. Franz MG, Kuhn MA, Nguyen K, Wang X, Ko F, Weig TE, Robson MC. Transforming growth factor β2 lowers the incidence of incisional hernias. J Res 2001; 97: 109–116
7. DuBay DA, Wang X, Kuhn MA, Robson MC, Franz MG. The prevention of incisional hernia formation using a delayed-release polymer of basic fibroblast growth factor. Ann Surg 2004; 240: 179–186
8. Klinge U, Si ZY, Zheng H, Schumpleick V, Bhardwaj RS, Kloterhalfen B. Abnormal collagen I to III distribution in the skin of patient with incisional hernia. Eur Surg Res 2000; 32: 43–48
9. Si ZY, Rhanjit B, Rosch R, Mertens R, Klosterhalfen B, Klinge U. Impaired balance of type I and type III procollagen mRNA in cultured fibroblasts of patients with incisional hernia. Surgery 2002; 131: 324–331
10. Rosch R, Junge K, Knops M, Lynen P, Klinge U, Schumpelick V. Analysis of collagen-interacting proteins in patients with incisional hernia. Langebecks Arch Surg 2003; 387: 427–432
11. Agren MS. Jorgensen LN, Andersen M, Viljanto J, Gottrup F. Matrix metalloproteinase 9 level predicts optimal collagen deposition during early wound repair in humans. Br J Surg 1998; 85: 68–71
12. Nwomeh BC, Liang HX, Cohen IK, Yager DR. MMP-8 is the predominant collagenase in healing wounds and nonhealing ulcers. J Surg Res 1999; 81: 189–195
13. Wall SJ, Sampson MJ, Levell N, Murphy G. Elevated matrix metalloproteinase-2 and 3 production from human diabetic dermal fibroblasts. Br J Dermatol 2003; 149: 13–16
14. Klinge U, Si ZY, Zheng H, Schumpelick V, Bhardwaj RS, Klosterhalfen B. Collagen I/III and matrix metalloproteinases (MMP) 1 and 13 in the fascia of patients with incisional hernias. J Invest Surg 2001; 14: 47–54
15. Stevick CA, Long JB, Jamasbi B. Ventral hernia following abdominal aortic reconstruction. Am Surg 1988; 51: 287–289
16 Cannon DJ, Castel L, Read RC. Abdominal aortic aneurysm, Leriche syndrome, inguinal herniation, and smoking. Arch Surg 1984; 119: 387–389.
17. Hall KA, Peters B, Smyth SH, Warmeke JA, Rappaport WD, Putnam ChW, Hunter GC. Abdominal wall hernias in patients with abdominal aortic aneurysmal versus aortoiliac occlusive disease. Am J Surg 1995; 170: 572–576
18. Holland AJA, Castleden WM, Norman PE, Stacey MC. Incisional hernias are more common in aneurysmal disease. Eur J Vasc Endovasc Surg 1996; 12: 196–200
19. Rogers M, McCarthy, Earnshaw JJ. Prevention of incisional hernia after aortic aneurysm repair. Eur J Vasc Endovasc Surg 2003; 26: 519–522
20. Adye B, Luna G. Incidence of abdominal wall hernia in aortic surgery. Am J Surg 1998; 175: 400–402
21. Raffetto JD, Cheung Y, Fisher JB. Incision and abdominal wall hernias in patients with aneurysm or occlusive aortic disease. J Vasc Surg 2003; 37: 1150–1154
22. Fassiadis N, Roidl M, Hennig M, South LM, Andrews SM. Randomized clinical trial of vertical or transverse laparotomy for abdominal aortic aneurysm repair. Brit J Surg 2005; 92: 1208–1211
23. White JV, Haas K, Phillips S, Comerota AJ. Adventitial elastolysis in primary event in aneuriysm formation. J Vasc Surg 1993; 17: 371–381
24. Patel MI, Melrose J, Ghosh P, Appleberg M. Increased synthesis of matrix metalloproteinases by aortic smooth muscle cells is implicated in the etiopathogenesis of abdominal aortic aneurysms. J Vasc Surg 1996; 24: 82–92
25. Sakalihasan N, Delvenne P, Nusgens BV, Limet R, Lapière ChM. Activated forms of MMP-2 and MMP-9 in abdominal aortic aneurysms. J Vasc Surg 1996; 24: 127–133
26. Tamarina NA, McMillan WD, Shively VP, Pearce WH. Expression of matrix metalloproteinases and their inhibitors in aneurysms and normal aorta. Surgery 1997; 122: 264–272

27. Crowther M, Goodall S, Jones JL, Bell PRF, Thompson MM. Increased matrix metalloproteinase 2 expression in vascular smooth muscle cells cultured from abdominal aortic aneurysms. J Vasc Surg 2000; 32: 575–583
28. Jorgensen LN, Kallehave F, Christensen E, Siana JE, Gottrup F. Less collagen production in smokers. Surgery 1998; 123: 450–455
29. Read RC. A review: The role of protease-antiprotease imbalance in the pathogenesis of herniation and abdominal aortic aneurysm in certain smokers. Postgraduate General Surg 1992; 14: 161–165
30. Read RC. Why do human beings develop groin hernias? In: Fitzgibbons, R.J., Jr. Greeburg A.G. eds. Nyhus and Condon´s hernia. Philadelphia: Lippincolt Williams&Wilkins 2002; 3–8
31. Sorensen LT, Hemmingsen RN, Kirkeby LT, Kallehave F, Jorgensen LN. Smoking is a risk factor for incisional hernia. Arch Surg 2005; 140: 119–123
32. Sorensen LT, Friis E, Jorgensen LN, Vennits B, Andersen BR, Rasmussen GI, Kjaergaard J. Smoking is a risk factor for recurrence of groin hernia. World J Surg 2002; 26: 397–400
33. Awad ZT, Puri V, LeBlanc K, Stoppa R, Fitzgibbons Jr RJ, Iqbal A, Filipi ChJ. Mechanisms of ventral hernia recurrence after mesh repair and a new proposed classification. J Am Coll Surg 2005; 201: 132–140

Discussion

Franz: *In our experimental work we never found a collagen synthesis defect in our animals. We can generate something in the animal that looks very much like a human incisional hernia without any recognizable biological defect, and that is what bothers us as surgeons, that so many patients will fail despite any easily recognizable biological defect; however, once the failure occurs and we are able to measure postmechanical failure defects on the fibroblastic level, one of our first surprising observations was that there never was a defect in the collagen production either in the wound or in the isolated fibroblast. The German group is good about demonstrating isonomic imbalances and showing perhaps pathology level that way, but we were never able to measure a collagen total synthesis defect.*

Kingsnorth: *What has not been mentioned are two small randomized trials using meshes prophylactically to support the wound, in aortic aneurysms and bariatric surgery. This is probably working better than trying to supplement the biological factors in the wound. What is your view of prophylactic mesh in patients with high risk?*

Bellon: *I think that is the future ….*

17 Technical Pitfalls Favouring Incisional Hernia

17.1 Technical Factors Associated With the Development of Incisional Hernia

M.A. Carlson

Introduction

"Occasional contributions have appeared on the subject of disruption of wounds for a long time, but more than forty articles have been found in the American literature alone during the last few years as evidence of its importance" (Singleton and Blocker 1939 [1]).

Postoperative abdominal incision failure remains as much a problem and topic of controversy today as it did nearly a century ago. The predominance of the surgical literature on incisional hernia describes and evaluates various repair techniques; less is written on predisposition and prevention. In the latter subset of the literature, emphasis has been placed upon patient-associated risk factors in the pathogenesis of incisional failure. Over the past several decades, however, the idea that surgeon-associated (i.e., technical) risk factors may be important in the etiology of incisional hernia has been gaining more acceptance [2]. The postulation that the surgeon could be the most important risk factor for this complication, however, is a more radical concept. This brief review will emphasize the role of surgeon-related factors in the development of incisional hernia.

Dehiscence vs. Incisional Hernia: Separate or the Same?

Abdominal wound dehiscence (variably known as wound disruption, acute wound or fascial failure, burst abdomen, etc.) and incisional hernia often are thought of as two separate entities, but they probably are ends of the same continuum. In general, the fascial disruption of wound dehiscence occurs in the early postoperative period (within the first several weeks); with incisional hernia, the disruption manifests later. The skin remains intact in the latter, having had ample time to heal, while in the former the skin either disrupts with the fascia or leaks fluid. So does an incisional hernia develop in a scar that has healed and then weakens over time? The current data suggest that a patient who acquires an incisional hernia will have had evidence of that hernia in the early postoperative period, i.e., during the time that a wound dehiscence presents. This has been demonstrated in midline incisions with the use of metal clips and plain radiographs [3] or by measuring the distance between the recti on CT scans [4].

This would suggest that the hernia formation begins very early. In other words, an incisional hernia might be thought of as a "subclinical dehiscence" in which the fascial failure, while not catastrophic and/or eviscerating, results in a gradually widening fascial defect. Of course, not all incisional hernias would fall under this etiology (see later discussion about buttonhole hernias), but the realization that postoperative abdominal wall hernia may have a very early origin implies that its cause could be similar to that of wound dehiscence; and the cause of wound dehiscence in the vast majority of cases is a technical inadequacy [5–7]. That is, the fault lies with the surgeon.

Choice of Incision

So if the responsibility for abdominal incisional hernia formation is technical inadequacy, what can the surgeon do to circumvent this? In other words, what are the forms of the technical inadequacy? The first (in temporal order) is the choice of incision. The best incision the surgeon can choose which will minimize the risk of incisional hernia is a minimal incision. If properly closed, trocar punctures from a minimally invasive procedure produce incisional hernia in ~1% of cases [8], which is much less than the 10–30% rate of herniation typically quoted for conventional incisions. Furthermore, since emphasis is being placed on the utilization of laparoscopic instruments with a diameter of ≤ 5 mm, the incidence of trocar hernia most likely will decrease.

If a laparoscopic approach is not feasible, then for a major intra-abdominal procedure the surgeon has a variety of incisional choices; for simplicity, these will be classified as either vertical (most commonly midline, through the linea alba) or transverse. There is a large amount of historical, retrospective data which suggests that the transverse incision has a lower incidence of dehiscence and hernia; for an early example of this, see Singleton and Blocker's review of 9000 incisions [1]. This retrospective data is influenced by various confounding factors (e.g., use of short transverse incisions for cholecystectomy vs. longer midline incisions for emergency procedures), but the preponderance of the data (not reviewed here) favors the transverse incision.

Three randomized controlled trials comparing hernia rates in vertical vs. midline incisions have been published [9–11], and these provide some support for a lower risk of incisional hernia in transverse incisions. The most recent trial [11] found a large, statistically significant increase in the incidence of hernia in midline compared to transverse incisions in a small group (<40) of aortic aneurysm patients. This finding needs to be tempered by the fact that the hernia incidence in the midline group was 94% (certainly the highest ever recorded in a hernia trial), which suggests a problem with suture technique (an uncontrolled variable in this trial). Currently there are no controlled data comparing transverse to midline incisions in which the suture technique is optimized and constant.

Two UK institutions reported a very low (1% or less) incidence of postoperative hernia with the lateral paramedian incision in trials during the 1980s [12–17]. This is a vertical incision through the lateral portion of the rectus sheath, about two-thirds the distance from the medial edge of the rectus. The rectus muscle is reflected medially during the operation, so upon layered closure of the rectus sheath, the muscle covers the fascial incisions. This provides a splinting effect which, the authors claim, is the basis for the robustness of the incision. The lateral paramedian incision generally takes longer to perform, and requires more expertise than the midline incision. Unfortunately, there have been no corroboratory publications from other institutions which validate the superiority of the lateral paramedian incision.

Abdominal Entry

The next choice the surgeon has which may influence the risk of wound failure is the act of incising the layers of the abdominal wall. Animal experimentation has shown that a small amount of tissue injury (such as delivered with a scalpel blade) is important to incite the appropriate amount of inflammation which will produce the strongest scar [18]. On the other hand, too much injury (such as that delivered with coagulation current from the cautery blade) inhibits healing because of fascial necrosis [19]. Even more dramatic is the effect of delayed primary or secondary wound closure which, in animals, can increase wound breaking strength (fascial or dermal) by as much as 100% at 60 days compared to primary closure [20, 21]. The presumptive cause of this effect is the greater fibrotic reaction inherent with an open wound. Data from humans in this area are absent and, of course, no one would recommend delayed primary or secondary wound closure as the standard operating procedure for elective laparotomy closure. The time-honored tradition of entering the abdomen with a clean swipe of the scalpel [22], however, still applies.

Choice of Suture Material

After the intra-abdominal procedure has been completed, the next choice the surgeon faces that may influence the risk of incisional hernia is suture material. There is a wealth of both retrospective and controlled data (not to be reviewed here) that scrutinizes suture material. The bottom line is that with modern suture material, the suture choice is of much less importance than how the surgeon actually places it (see below). That being said, there have been a number of meta-analyses and systematic reviews which have favored either nonabsorbable suture material (e.g., nylon, polypropylene) or slowly absorbable suture material (e.g., polydioxanone) in the closure of laparotomy incisions [23–26]. The perceived detraction to using nonabsorbable suture is the development of buttonhole hernia [27, 28] which is a fascial defect created by the perpetual sawing motion of the suture where it penetrates the fascia. A patient can develop a cluster of these hernias and end up with a so-called Swiss cheese abdomen. Buttonhole hernia may be the reason why incisional hernias continue to develop years out from the index procedure [29]. It is difficult to say if the incidence of buttonhole hernia is less with a slowly absorbable suture.

Suture Technique: Suture-Length-to-Wound-Length Ratio

The single most important surgeon-related factor in the risk for incisional hernia is suture technique, which entails items such as tissue bite, stitch interval, stitch tension, and so on. In cases of wound dehiscence not involving fasciitis, the most common cause of failure is suture tearing through the fascia [5]. One possibility suggested by this observation is that an inadequate tissue bite during incisional closure will predispose the patient to tissue tearing, which can result in acute wound failure or delayed hernia. It is not surprising that in animal and cadaver studies, a wider bite of fascia with the suture results in a higher pull-out strength [30–32]. Furthermore, it has been shown that suture holding capacity in experimental incisions of both the abdominal fascia and hollow viscera actually decreases during the early postoperative period [33], presumably because the region immediately adjacent to the incision is biochemically active (e.g., matrix metalloproteinase activation) and becomes "soft" [34]. So, taking a wide bite with the suture needle would avoid this biochemically active wound region.

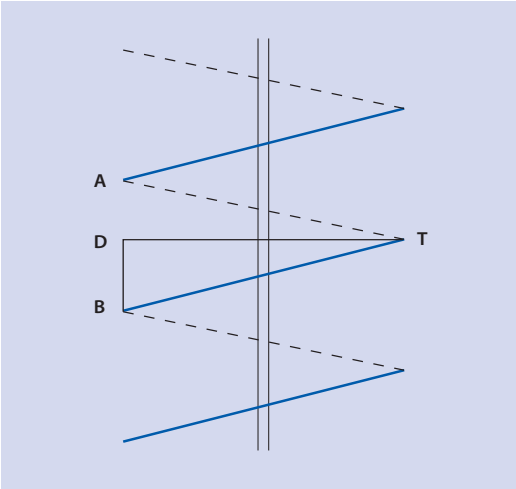


Fig. 17.1. Suture-length-to-wound-length ratio [6]

So how wide a bite should be taken? If 1 cm is better than 5 mm, then why not 2 or 3 cm? Indeed, in some of the early experience with wide bite closure, some surgeons routinely placed retention sutures. For example, Kennedy [35] informally described the performance of around 30,000 abdominal incisions over a 56-year period (between him and his mentor, Joseph Price), and could recount only one case of dehiscence and no hernias (!). Their technique of closure involved through-and-through (all layers, dermis to peritoneum) silk sutures, placed 1 inch (2.5 cm) back from the wound edge, three for every inch of incision, and tied loosely. They also closed the fascia with buried sutures prior to tying the through-and-through sutures. The silk retentions typically were removed on postoperative day 10. Such routine retention suture placement probably would not be readily accepted today, but the above experience is illustrative of the benefit of generous tissue bites and short stitch interval on the prevention of wound failure.

The first individual to apply some science to wide bite closure was TPN Jenkins [6, 36]. He introduced the concept of suture-length-to-wound-length ratio (SL:WL), as shown in Fig. 17.1. This applied to continuous closures, and was equal to the length of suture used to close the incision divided by length of the incision. The suture length was dependent on two parameters: the stitch interval (distance AB in Fig. 17.1) and the tissue bite (one half of the distance TD in Fig. 17.1). Jenkins determined that a SL:WL of ≥ 4 was protective of dehiscence; he had only one burst abdomen in 1500 closures in which he maintained this ratio (0.07%

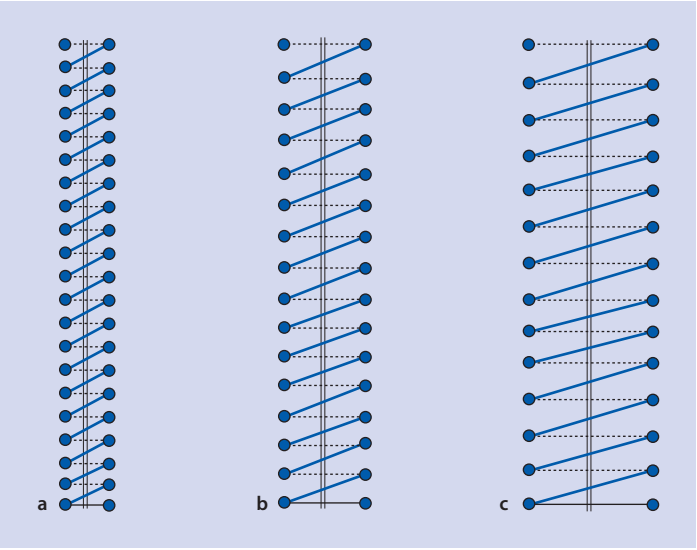


Fig. 17.2. Role of stitch interval vs. tissue bite in rat vertical midline wounds closed with a constant SL:WL of 4. The wound in C was the strongest immediately after closure, but the wound in A and B were stronger on postoperative day 4 [45]

dehiscence rate). Jenkins also applied this technique to primary suture repair of incisional hernia and, employing SL:WL as high as 44, he achieved a relatively low recurrence rate of 8%.

The use of SL:WL in abdominal incision closure was popularized by Israelsson and colleagues during the 1990s [2, 7, 37–40]. They demonstrated that maintenance of a SL:WL greater than 4 (particularly in regard to vertical midline incisions) minimized the occurrence of both dehiscence and hernia. The primacy of a SL:WL of 4 in the prevention of wound failure was corroborated experimentally by the Aachen group [41, 42]. But, analogous to the question above, if a SL:WL of 4 is good, would 5 or more be better? Perhaps not; clinically it was observed that a SL:WL ≥ 5 was associated with an increased incidence of wound infection (and subsequent wound failure), especially in obese patients [43, 44].

Experimentally, excessively wide bites have disadvantages. In rat incisions closed with a constant SL:WL of 4 [45], wounds with a relatively short stitch interval and small tissue bite were stronger on postoperative day 4 than wounds with a relatively long stitch interval and large tissue bite (Fig. 17.2). That is, the wounds with more stitches and smaller bites fared better. In a study with pigs [46], closing a vertical midline incision with wide interrupted bites through the rectus sheath and then maintaining 20 mmHg of intra-abdominal pressure for 3 h resulted in rectus muscle tearing and hemorrhage with greater wound edge separation (as marked with metal clips), as compared to wounds in which stitches took only bites of the anterior sheath. Early wound separation is, as noted above, an early indicator of incisional hernia. The implication of these experimental data and the above clinical studies was that a mass stitch in wide bite closure might be detrimental to incisional healing. So the simple concept of "more is better" in wide bite closure may be subject to some qualifications. The final word probably has not been heard in this arena.

Suture Technique: Tension

There are two types of tension which are relevant to incisional healing. The first type is tension that the surgeon (or first assistant) places on the suture during closure. It has been shown experimentally that excessive suture tension decreases wound strength [30, 42, 47–49] and perfusion to the central portion of the wound [50]. Of course, inadequate tension on the suture (i.e., too loose) will result in protrusion of intestinal loops, peritoneal fluid leaks, wound edge separation, and eventual hernia. One group found that compression suture of vertical midline incisions (in which each individual loop of a continuous suture was tightened with 5 kg of force) in patients resulted in fewer wound complications compared to a closure with nontightened loops [51]. This finding is somewhat counterintuitive to the clinical adage of "approximate, don't strangulate." Currently, there is no consensus on the amount of tension to place on suture during closure.

The second type of tension relevant to incisional healing is that required to bring the wound edges together, or tissue tension. This also is the tension across the wound, or suture line, after closure has been completed. Another maxim in surgery is that suture lines under tension will be at an elevated risk for failure; this has been confirmed in the laboratory [47, 52]. There are some experimental conditions, however, in which suture line tension actually increased wound disruption strength [3, 53]; in addition, tension stimulated granulation tissue growth in animal excisional wounds [54, 55]. There may be some level of tissue tension that is optimal for incisional healing; clinically, however, this has not been defined. Furthermore, a critical level of tissue tension beyond which the risk for incisional failure is unacceptable also is not known.

Suture Technique: Other Issues

Perhaps less controversial in the recent literature are choices between continuous vs. interrupted sutures and mass vs. layered technique. There have been multiple retrospective reviews that document the efficacy of various combinations (running mass, interrupted layered, etc.) which will not be reviewed here. There also have a number of meta-analyses which have concluded that continuous sutures are superior to interrupted [23–26, 56]. One large randomized controlled trial comparing running vs. interrupted laparotomy closure [57] demonstrated that the former had fewer wound complications (mainly dehiscence; follow-up was for 30 days).

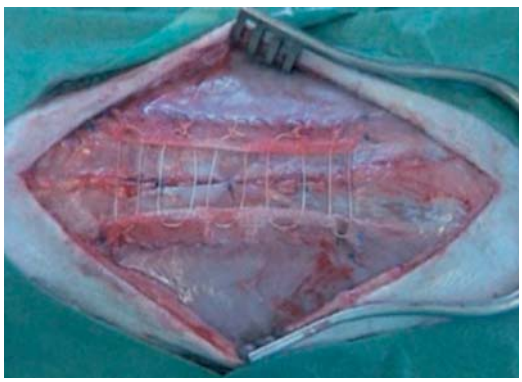
The Smead–Jones suture technique [58], also known as far-near near-far sutures, intermittently has been touted (with uncontrolled clinical data) as protective against wound failure. A variant of this technique, the continuous double-loop suture, was shown to be acutely stronger than other techniques in the rat; interestingly, this technique failed in comparison to conventional running suture in a clinical trial [59]. Retrospective data has demonstrated that routine retention suture placement (Mont Reid type [60]) at the index laparotomy prevents acute wound failure [35, 61]. This was confirmed experimentally in dogs [62], but not in a clinical randomized trial [63]. Other than the salutary effect of closely spaced retention sutures on hernia prevention in older retrospective data [35], the efficacy of retentions in modern-day hernia prophylaxis is unknown.

Prophylaxis of surgical wound infection, while not completely under control of the surgeon, should be mentioned in an article such as this, because infection repeatedly has been shown to be an independent risk factor in the development of incisional hernia (data not reviewed here). Of note, the Israelsson group has shown that a SL:WL of 4.0–4.9 is optimal value for minimizing wound infection risk and subsequent incisional hernia [38, 43].

Novel Techniques for Prevention of Incisional Hernia

Recently, the feasibility and efficacy of prophylactic mesh placement for reinforcement of laparotomy closure has been demonstrated in one small randomized trial of high-risk patients [64] and two small series of bariatric [65] and aortic aneurysm [66] patients. The optimal placement technique (e.g., sublay vs. onlay) is not known. In regard to intestinal stomas, there has been one small randomized trial of routine placement of a light-weight composite mesh (Vypro) at the time of stomal creation [67], which demonstrated a reduction of parastomal hernia formation in the mesh patients. Mesh reinforcement of primary hiatal herniorrhaphy also was efficacious in reducing hernia recurrence in a randomized trial [68]. Prophylactic mesh placement is an exciting and intriguing area in abdominal wall surgery, and needs further study.

A novel technique of laparotomy closure recently described in animals by the Aachen group is tension banding or the bridging technique [50, 69], in which the fascial edges of a vertical midline incision are coated by polylactide (slowly absorbable synthetic) U-stitches placed into two parallel polylactide strips that have been affixed to the anterior sheath (◘ Fig. 17.3). This technique provided equivalent or better wound perfusion and strength compared to conventional suturing or onlay mesh placement. The advantage of the bridg-



◘ **Fig. 17.3.** Tension banding for laparotomy closure [50]

ing technique has been postulated to be the avoidance of both foreign material at the wound edge and the strangulating effect of incisional sutures. Clinical data are not yet available.

Recommendations

The ability to prevent both abdominal wound dehiscence and incisional hernia primarily lies with the surgeon and the technique used to close the laparotomy incision. That being said, the technical recommendations to minimize the risk of incisional hernia after major laparotomy which are promoted by this article are as follows:

- Avoid large incisions by performing a minimally invasive procedure whenever possible.
- Consider transverse incision as an alternative to the vertical midline incision.
- Avoid the coagulation current of the cautery when incising the aponeurosis.
- Utilize either a nonabsorbable or a slowly absorbable suture.
- In a running closure of a vertical midline incision, maintain the suture-length-to-wound-length ratio between 4 and 5.
- Avoid excessively wide suture bites which incorporate large masses of muscle and fat.
- Avoid incisional closure in the presence of excessive tissue tension.
- Maintain adequate suture tension to coapt the fascial edges, but do not strangulate the tissue.
- Choose running suture over interrupted.
- Minimize the risk of surgical wound infection.
- Consider prophylactic mesh placement for the patient at high risk for wound failure.

Acknowledgements. Supported in part by a grant from the United States National Institutes of Health (K08 GM00703).

References

1. Singleton A, Blocker T. The problem of disruption of abdominal wounds and postoperative hernia: a review of 9,000 consecutive abdominal incisions. JAMA 1939; 112(2): 122–127
2. Israelsson LA. The surgeon as a risk factor for complications of midline incisions. Eur J Surg 1998; 164(5): 353–359
3. Pickett BP, Burgess LP, Livermore GH, Tzikas TL, Vossoughi J. Wound healing. Tensile strength vs healing time for wounds closed under tension. Arch Otolaryngol Head Neck Surg 1996; 122(5): 565–568
4. Burger JW, Lange JF, Halm JA, Kleinrensink GJ, Jeekel H. Incisional hernia: Early complication of abdominal surgery. World J Surg 2005; 29(12): 1608–1613
5. Carlson MA. Acute wound failure. Surg Clin North Am 1997; 77(3): 607–636
6. Jenkins TPN. The burst abdominal wound: a mechanical approach. Br J Surg 1976; 63(11): 873–876
7. Israelsson LA, Jonsson T. Closure of midline laparotomy incisions with polydioxanone and nylon: the importance of suture technique. Br J Surg 1994; 81(11): 1606–1608
8. Tonouchi H, Ohmori Y, Kobayashi M, Kusunoki M. Trocar site hernia. Arch Surg 2004; 139(11): 1248–1256
9. Ellis H, Coleridge-Smith PD, Joyce AD. Abdominal incisions – vertical or transverse? Postgrad Med J 1984; 60(704): 407–410
10. Greenall MJ, Evans M, Pollock AV. Midline or transverse laparotomy? A random controlled clinical trial. Part I: Influence on healing. Br J Surg 1980; 67(3): 188–190
11. Fassiadis N, Roidl M, Hennig M, South LM, Andrews SM. Randomized clinical trial of vertical or transverse laparotomy for abdominal aortic aneurysm repair. Br J Surg 2005; 92(10):1208–1211
12. Brennan TG, Jones NA, Guillou PJ. Lateral paramedian incision. Br J Surg 1987; 74(8): 736–737
13. Cox PJ, Ausobsky JR, Ellis H, Pollock AV. Towards no incisional hernias: lateral paramedian versus midline incisions. J R Soc Med 1986; 79(12): 711–712
14. Donaldson DR, Hall TJ, Zoltowski JA, Guillou PJ, Brennan TG. Does the type of suture material contribute to the strength of the lateral paramedian incision? Br J Surg 1982; 69(3): 163–165
15. Donaldson DR, Hegarty JH, Brennan TG, Guillou PJ, Finan PJ, Hall TJ. The lateral paramedian incision – experience with 850 cases. Br J Surg 1982; 69(10): 630–632
16. Kendall SW, Brennan TG, Guillou PJ. Suture length to wound length ratio and the integrity of midline and lateral paramedian incisions. Br J Surg 1991; 78(6): 705–707
17. Gilbert JM, Ellis H, Foweraker S. Peritoneal closure after lateral paramedian incision. Br J Surg 1987; 74(2): 113–115
18. Miro D, Julia MV, Sitges-Serra A. Wound breaking strength and healing after suturing noninjured tissues. J Am Coll Surg 1995; 180(6): 659–665
19. Rappaport WD, Hunter GC, Allen R, Lick S, Halldorsson A, Chvapil T, Holcomb M, Chvapil M. Effect of electrocautery on wound healing in midline laparotomy incisions. Am J Surg 1990; 160(6): 618–620
20. Fogdestam I. A biomechanical study of healing rat skin incisions after delayed primary closure. Surg Gynecol Obstet 1981; 153(2): 191–199
21. Scott PG, Chambers M, Johnson BW, Williams HT. Experimental wound healing: increased breaking strength and collagen synthetic activity in abdominal fascial wounds healing with secondary closure of the skin. Br J Surg 1985; 72(10): 777–779
22. Moynihan B. The ritual of a surgical operation. Br J Surg 1920; 8: 27–35
23. Weiland DE, Bay RC, Del Sordi S. Choosing the best abdominal closure by meta-analysis. Am J Surg 1998; 176(6): 666–670

24. van,t Riet M, Steyerberg EW, Nellensteyn J, Bonjer HJ, Jeekel J. Meta-analysis of techniques for closure of midline abdominal incisions. Br J Surg 2002; 89(11): 1350–1356
25. Hodgson NC, Malthaner RA, Ostbye T. The search for an ideal method of abdominal fascial closure: a meta-analysis. Ann Surg 2000; 231(3): 436–442
26. Rucinski J, Margolis M, Panagopoulos G, Wise L. Closure of the abdominal midline fascia: meta-analysis delineates the optimal technique. Am Surg 2001; 67(5): 421–426
27. Krukowski ZH, Matheson NA.,Button hole' incisional hernia: a late complication of abdominal wound closure with continuous non-absorbable sutures. Br J Surg 1987; 74(9): 824–825
28. Read RC, Yoder G. Recent trends in the management of incisional herniation. Arch Surg 1989; 124(4): 485–488
29. Mudge M, Hughes LE. Incisional hernia: a 10 year prospective study of incidence and attitudes. Br J Surg 1985; 72(1): 70–71
30. Sanders RJ, DiClementi D, Ireland K. Principles of abdominal wound closure. I. Animal studies. Arch Surg 1977; 112(10):1184–1187
31. Campbell JA, Temple WJ, Frank CB, Huchcroft SA. A biomechanical study of suture pullout in linea alba. Surgery 1989; 106(5): 888–892
32. Tera H, Aberg C. Tissue strength of structures involved in musculo-aponeurotic layer sutures in laparotomy incisions. Acta Chir Scand 1976; 142(5): 349–355
33. Hogstrom H, Haglund U, Zederfeldt B. Suture technique and early breaking strength of intestinal anastomoses and laparotomy wounds. Acta Chir Scand 1985; 151(5): 441–443
34. Adamsons RJ, Musco F, Enquist IF. The chemical dimensions of a healing incision. Surg Gynecol Obstet 1966; 123(3): 515–521
35. Kennedy JW. Tragedies of the abdominal incision. Am J Surg 1934; 25: 512–520
36. Jenkins TPN. Incisional hernia repair: a mechanical approach. Br J Surg 1980; 67(5): 335–336
37. Israelsson LA, Jonsson T. Incisional hernia after midline laparotomy: a prospective study. Eur J Surg 1996; 162(2): 125–129.
38. Israelsson LA, Jonsson T, Knutsson A. Suture technique and wound healing in midline laparotomy incisions. Eur J Surg 1996; 162(8): 605–609
39. Israelsson LA, Jonsson T. Suture length to wound length ratio and healing of midline laparotomy incisions. Br J Surg 1993; 80(10): 1284–1286
40. Israelsson LA. Continuous closure of laparotomy incisions: aspects of suture technique. In Schumpelick V, Kingsnorth AN, eds. Incisional hernia. Springer, Berlin Heidelberg New York 1999. pp. 246–252
41. Hoer J, Klinge U, Schachtrupp A, Tons C, Schumpelick V. Influence of suture technique on laparotomy wound healing: an experimental study in the rat. Langenbecks Arch Surg 2001; 386(3): 218–223
42. Hoer JJ, Junge K, Schachtrupp A, Klinge U, Schumpelick V. Influence of laparotomy closure technique on collagen synthesis in the incisional region. Hernia 2002; 6(3): 93–98
43. Israelsson LA, Jonsson T. Overweight and healing of midline incisions: the importance of suture technique. Eur J Surg 1997; 163(3): 175–180
44. Millbourn D, Israelsson LA. Wound complications and stitch length. Hernia 2004; 8(1): 39–41
45. Cengiz Y, Blomquist P, Israelsson LA. Small tissue bites and wound strength: an experimental study. Arch Surg 2001; 136(3): 272–275
46. Cengiz Y, Gislason H, Svanes K, Israelsson LA. Mass closure technique: an experimental study on separation of wound edge. Eur J Surg 2001; 167(1): 60–63
47. Villegas-Alvarez F, Olvera-Duran J, Rodriguez-Aranda E, Carmona-Mancilla A, Vigueras-Villasenor RM, Mendez-Ramirez I. Esophageal anastomotic failure: an experimental study. Arch Med Res 2003; 34(3): 171–175
48. Nelson CA, Dennis C. Wound healing; technical factors in the gain of strength in sutured abdominal wall wounds in rabbits. Surg Gynecol Obstet 1951; 93(4): 461–467
49. Poole GV, Jr., Meredith JW, Kon ND, Martin MB, Kawamoto EH, Myers RT. Suture technique and wound-bursting strength. Am Surg 1984; 50(10): 569–572
50. Hoer J, Tons C, Schachtrupp A, Anurov M, Titkova S, Oettinger A, Wetter O, Schumpelick V. Quantitative evaluation of abdominal wall perfusion after different types of laparotomy closure using laser-fluorescence videography. Hernia 2002; 6(1): 11–16
51. Mayer AD, Ausobsky JR, Evans M, Pollock AV. Compression suture of the abdominal wall: a controlled trial in 302 major laparotomies. Br J Surg 1981; 68(9): 632–634
52. Borgstrom S, Sandblom P. Suture technic and wound healing; an investigation based on animal experiments. Ann Surg 1956; 144(6): 982–990
53. Thorngate S, Ferguson DJ. Effect of tension on healing of aponeurotic wounds. Surgery 1958; 44(4): 619–624
54. Carlson MA, Longaker MT, Thompson JS. Wound splinting regulates granulation tissue survival. J Surg Res 2003; 110(1):304–309
55. Carlson MA, Thompson JS. Wound splinting modulates granulation tissue proliferation. Matrix Biol 2004; 23(4): 243–250
56. Wadstrom J, Gerdin B. Closure of the abdominal wall; how and why? Clinical review. Acta Chir Scand 1990; 156(1): 75–82
57. Fagniez PL, Hay JM, Lacaine F, Thomsen C. Abdominal midline incision closure. A multicentric randomized prospective trial of 3,135 patients, comparing continuous vs interrupted polyglycolic acid sutures. Arch Surg 1985; 120(12): 1351–1353
58. Jones TE, Newell ET, Jr, Brubacker RE. The use of alloy steel wire in the closure of abdominal wounds. Surg Gynecol Obstet 1941; 72: 1056–1059
59. Niggebrugge AH, Trimbos JB, Hermans J, Steup WH, Van De Velde CJ. Influence of abdominal-wound closure technique on complications after surgery: a randomised study. Lancet 1999; 353(9164): 1563–1567
60. Reid MR, Zinninger MM, Merrell P. Closure of the abdomen with through-and-through silver wire sutures in cases of acute abdominal emergencies. Am Surg 1933; 98: 890–896
61. Holman CW, Eckel JH. Prevention of wound disruption with through-and-through silver wire stay sutures. Surg Gynecol Obstet 1941; 72: 1052–1055
62. Miles RM, Nash JP, Gillespie H, Dacus D. Value of retention sutures in the prevention of experimental abdominal wound disruption. Am Surg 1967; 33(11): 898–905

63. Hubbard TB, Jr., Rever WB, Jr. Retention sutures in the closure of abdominal incisions. Am J Surg 1972; 124(3): 378–380
64. Gutierrez de la Pena C, Medina Achirica C, Dominguez-Adame E, Medina Diez J. Primary closure of laparotomies with high risk of incisional hernia using prosthetic material: analysis of usefulness. Hernia 2003; 7(3): 134–136
65. Strzelczyk J, Czupryniak L, Loba J, Wasiak J. The use of polypropylene mesh in midline incision closure following gastric by-pass surgery reduces the risk of postoperative hernia. Langenbecks Arch Surg 2002; 387(7 8): 294–297
66. Rogers M, McCarthy R, Earnshaw JJ. Prevention of incisional hernia after aortic aneurysm repair. Eur J Vasc Endovasc Surg 2003; 26(5): 519–522
67. Janes A, Cengiz Y, Israelsson LA. Randomized clinical trial of the use of a prosthetic mesh to prevent parastomal hernia. Br J Surg 2004; 91(3): 280–282
68. Frantzides CT, Madan AK, Carlson MA, Stavropoulos GP. A prospective, randomized trial of laparoscopic polytetrafluoroethylene (PTFE) patch repair vs simple cruroplasty for large hiatal hernia. Arch Surg 2002; 137(6): 649–652
69. Hoer J, Klinge U, Anurov M, Titkova S, Oettinger A, Schumpelick V. Tension banding closure of laparotomies: results of an experimental study in dogs. Langenbecks Arch Surg 2002; 387(7 8): 309–314

Discussion

Schumpelick: *What about the time course of incisional hernia development? There are interesting investigations, for example by CT scan from Prof. Jeekel. The second question is about suture tension: what is tension? How do you measure it?*
Carlson: *The time course is difficult to say. The risk remains forever. A number of people have documented hernia formation 10 or 20 years after the operation, I think those are typically buttonhole hernias that you see after these years, but the risk remains as long as you live. The suture tension remains a secret, we know it is important but we have no way to quantify. How to standardize so that 1000 surgeons can do the same thing, we don't know.*
Schumpelick: *About 3 years ago we developed a tensiometer, measuring the knotting tension of different doctors. At a different time of the day and at different types of operations. It showed that we use too much tension, with a little less in the afternoon than in the morning, and in a re-operation there is a higher tension, that means we have to measure it. More studies concerning the problem and validation of tension are necessary.*
Jeekel: *In the mentioned CT investigation it was shown that in people who developed a hernia it was already visible on the CT scan with the fascia edges widening.*
Deysine: *I heard several times to use a mesh when the fascia has not enough strength, in other words support a wound with a mesh. I want to caution that when you do that you have to double the caution to keep the wound clean, asepsis and antisepsis and antibiotics, in other terms, the rate of wound infection will increase.*
Kehlet: *I think we have a great problem to translate science into the daily clinical practice. Every lecture says transverse incisions and suddenly you say you are not using it and when I look around seeing fast-track surgery they never use transverse incisions, so how can we spread the message and why is it that you are using vertical instead of transverse incisions? Here in the lecture room you say transverse but when you go home to your operation room you do the opposite.*
Jeekel: *And then, Dr Kehlet, we talk about prevention by using a mesh instead of using the right incision, yes that's amazing.*

17.2 Technical Pitfalls Favouring Incisional Hernia From an Expert in Laparoscopic Surgery

S. Morales-Conde

Introduction

Laparoscopic surgery continues to advance in achieving further benefits over the conventional approach for certain pathologies. In 1991 LeBlanc et al. carried out the first laparoscopic repairs of ventral hernias [1]. Although not originally considered to be a pathology that could benefit from this approach, laparoscopic repair of ventral hernias has attained wide acceptance in recent years because of the significant advantages afforded by improvements in prosthetic materials and in attachment methods, as well as in the surgical technique used. The laparoscopic procedure offers greater comfort during the postoperative period, reduces hospitalization time and lowers complication rates. Even though many series still have a limited follow-up, the technique has shown lower rates of recurrence than the open methods, making it a procedure that solves a long-standing challenge for the surgeon. The relationship

between laparoscopic surgery and ventral hernias could be established in three senses. On the one hand, the fact that the rate of recurrences after laparoscopic ventral hernia repair is lower compared to an open repair, due to different factors that could be analyzed; on the other hand, there are some technical aspects of the laparoscopic repair of ventral hernias that may influence in the possibility of having a recurrence. Finally, laparoscopy, as an approach, could be involved in the production of hernias, such as trocar site hernias or tack hernia, as we will describe during this chapter.

Factors That May Influence a Lower Incidence of Recurrences After Laparoscopic Incisional Hernia Repair Compared to an Open Repair

There are different comparative studies published in the literature comparing laparoscopic and open ventral hernia repair (Table 17.1). The purpose of these studies was to determine whether laparoscopic intraperitoneal prosthetic patch repair of a ventral hernia is superior to open-mesh repair. Laparoscopic hernioplasty is as safe and effective as the traditional open technique with shorter length of stay and decreased hospital costs. In these series that have been published [2–6, 8–10], laparoscopic ventral hernioplasty compares favourably also with respect to wound complications, hospital stay, operative time and recurrence rate, and only one study [7] shows that laparoscopic incisional hernia repair of at least moderate complexity had no demonstrable advantage over the open repair.

Different questions arise after analyzing these studies, such us why laparoscopic repair shows better results than open conventional mesh repair and, especially why the rate of recurrences is lower. Different factors have been related to recurrences after open repair including wound infection and other local wound complications, size of the hernia, obesity, age, respiratory disease, sex (male), site of the her-

Table 17.1. Comparative studies between laparoscopic and open ventral hernia repair

	Technique	No.	Compl. [%]	Operative time [min]	Hospital stay [days]	Infection [%]	Recurrences [%]
Holzman et al. (1997) [2]	Open Laparoscopic	16 20	31 23	98 128	5 1.6 d	6 5	13 10
Park et al. (1998) [3]	Open Laparoscopic	49 56	37 18	78 95	6.5 3.4	2 0	35 11
Ramshaw et al. (1999) [4]	Open Laparoscopic	174 79	26 15	82 58	2.8 1.7	3 0	20.6 2.5
Carbajo et al. (1999) [5]	Open Laparoscopic	30 30	50 20	112 87	9.1 2.2	18 0	7 0
De Maria et al. (2000) [6]	Open Laparoscopic	18 21	72 57	– –	4.4 0.8	33 10	0 6
Chari et al. (2000) [7]	Open Laparoscopic	14 14	14 14	78 124	5.5 5	0 7	– –
Robbins et al. (2001) [8]	Open Laparoscopic	23 31	– –	– –	– –	30 16	– –
Wright et al. (2002) [9]	Open mesh Open no m Laparoscopic	90 119 86	28 22 24	102 70 131	2.5 1.5 1.5	13 10 9	6 9 1
MacGreevy et al. (2003) [10]	Open Laparoscopic	71 65	21 8	1.7 h 2.2 h	1.5 1.1	– –	– –

nia, number of times the hernia have recurred and the presence of non-palpable defects. Some of the advantages of laparoscopic repair are the possibility that this approach offers to reduce the presence of some of the factors involved on having a recurrence. The rate of local wound complications, and especially wound infection, is lower after a laparoscopic repair, as has been demonstrated in different studies, decreasing from 30 to 16% in some series that specifically have measured the rate of this complication, while some series demonstrated that this rate can even be reduced to 0% [3–5] after the laparoscopic approach. We believe the main reason for this is the fact that the presence of seroma after an open and a laparoscopic repair is very frequent. After an open repair the possibility of infection of the seroma is higher since the incision performed is localized at the top of this fluid, which is close to the mesh. In these situations, bacteria from the skin could easily come into contact with the seroma, and the possibility of contamination infection of the seroma and the mesh increases. On the other hand, after a laparoscopic approach, trocar sites are far from the area where the seroma and the mesh are, so this area could be maintained under sterile conditions and the possibility of infection decreased (Fig. 17.4a,b).

Another factor that is involved in the presence of hernia recurrences after an open repair is obesity. Patients who are morbidly obese traditionally have been considered poor surgical candidates for ventral hernia repair because of their associated comorbidities and risk of postoperative wound infection and hernia recurrence. Laparoscopic repair of ventral hernias in patients who are morbidly obese is both safe and feasible, and can be performed with minimal morbidity. Birgisson et al. [11] have demonstrated that this factor does not play a role in recurrences during laparoscopic repair, since the rate of recurrences after this approach has no relation to the BMI of the patient.

One of the advantages of laparoscopic surgery is that it offers the possibility to find the presence of non-palpable defects that have not been detected during clinical examinations. The laparoscopic approach allows a total exposure of the incision once adhesiolysis is completed, and those small defects or weakness of the anterior abdominal wall can be easily detected, factors that could be involved in the presence of further recurrences and that are difficult to identify during an open repair. One of the recommendations during the laparoscopic approach of incisional hernias is to expose the whole area of the incision to detect these weak areas, which must be covered with the mesh with a proper overlap. In fact, it has been published [12] that 13.1% of the patients who undergo a laparoscopic repair of an incisional hernia have multiple defects, and the average number of defect found is 4.8, more than the number detected during clinical examination [13].

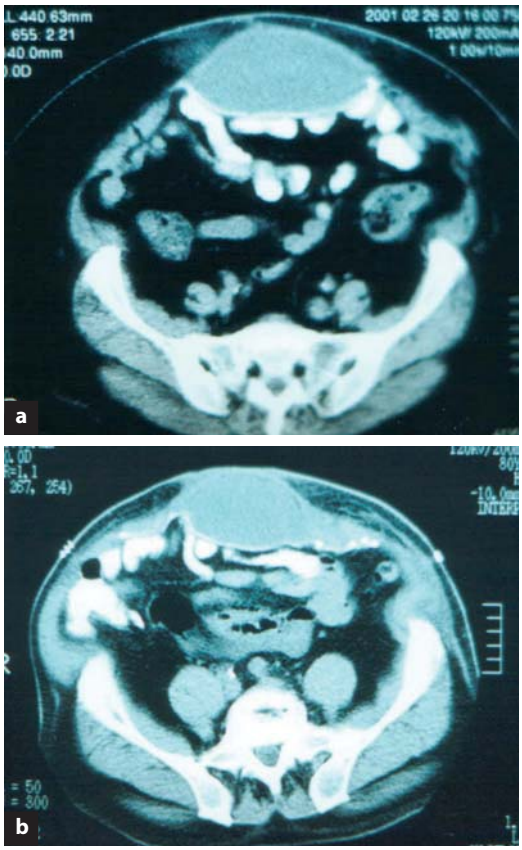


Fig. 17.4. a Seroma after open mesh repair. Incision is at the top of the fluid collection, which increases the possibility of contamination infection. **b** Seroma after laparoscopic mesh repair. Trocar site is far from the serma and the mesh, which are under sterile conditions

Technical Aspects of Laparoscopic Ventral Hernia Repair That May Influence in Hernia Recurrences

Incisional hernia underwent a change from conventional techniques to laparoscopic approach. The relevance of different factors, such as operative technique, mesh material and fixation, concerning the outcome following laparoscopic repair, are still under debate. Laparoscopic repair revealed acceptable recurrence

rates with high patient comfort. From a surgical point of view, the most important prognostic factor following mesh repair is the surgeon's experience, although some factors can be analyzed to try to reduce the presence of recurrences.

What Area Should Be Covered?

New hernias below original hernias have been described as a factor of recurrence after open repair. This factor has also been described after laparoscopic approach [14], which has led to recommending to cover the entire incision even in those cases in which a weak area is not detected, since this damaged tissue could be involved in the presence of a new hernia. At present, it appears evident that when undertaking laparoscopic repair of an incisional hernia, adhesiolysis must cover the entire area of the previous scar in order to identify possible wall defects at this level, other than those originally destined to be repaired. This is precisely one of the advantages of laparoscopy over traditional open repair. Defects that were not identified during the clinical examination and that were the cause of recurrence or appearance of a new defect after open repair can be detected and repaired in the same surgical procedure [15].

How Should the Area of Placing the Mesh Be Prepared?

There are two factors that may influence a proper repair of a ventral repair regarding the area where the prosthetic material is going to be implanted. On one hand, the intraperitoneal fatty tissue of the anterior abdominal wall must be removed to guarantee a proper fixation of the mesh. This fatty tissue, including the round and the falciform ligament, should be removed so the spiral tacks could reach the aponeurosis of the muscle to fix the mesh more consistently.

On the other hand, the process of removing this fatty tissue, together with the adhesiolysis, will produce an inflammatory reaction of the peritoneum, which will increase the ingrowth of the prosthetic materials. This fact is especially important when composite materials with polypropylene mesh in the parietal side are used, since this material needs this inflammatory reaction to improve the ingrowth. This does not occur with meshes of ePTFE since this material follows a pattern of encapsulation to be attached to the anterior abdominal wall.

How Should Size of the Mesh Be Selected?

The two factors that should be considered during a laparoscopic repair regarding mesh size are the following: it is safer to avoid recurrences by using one large mesh than two pieces of the material, since the area where the two meshes overlap is a weak area that has been related to the presence of recurrences [16].

On the other hand, it has been described that recurrences were reduced because of the use of an increased overlap of the biomaterial [14]. Prostheses initially recommended were small; overlapping the defect by only 2 [17, 18] to 2.5 cm [3] in all directions, and not the minimum of 3–5 cm currently recommended. Recently, we have demonstrated in an experimental study that expanded polytetrafluoroethylene (ePTFE) prostheses decreases in size once they have been implanted (1.63 cm out of 4 cm after 5 weeks), probably because of the scar tissue reaction and the encapsulation process experienced by the mesh [19], so recurrences in these initial experiences could have happened mainly because of the smaller size of the mesh.

How to Fix the Mesh?

One of the most interesting points currently being debated is whether or not it is necessary to use sutures and tacks or tacks alone, following the double crown technique (Fig. 17.5), or other additional methods of fixation, such as biological glues or the new method of fixations available.

Despite the lower recurrence rate, various authors have made efforts to analyze the causes for recurrence in order to adequately define the laparoscopic technique and thereby achieve an even lower recurrence rate. Initial laparoscopic ventral hernia repair series established a direct correlation between recurrence and the absence of transfascial sutures [9, 18, 21]. In fact, they demonstrated that one of the essential factors to avoid recurrence is the use of these sutures [22]. Analysis of the data derived from these early series, data which were later the basis for recommendations on the use of sutures, shows that there could have been other factors involved in the development of recurrence in these patients besides the use or not of transfascial sutures: prostheses initially recommended were small, the method of fixation was also inadequate, since tacks were not yet available and mesh patches were anchored with the old endostaplers that did not ensure secure attachment of the material, and

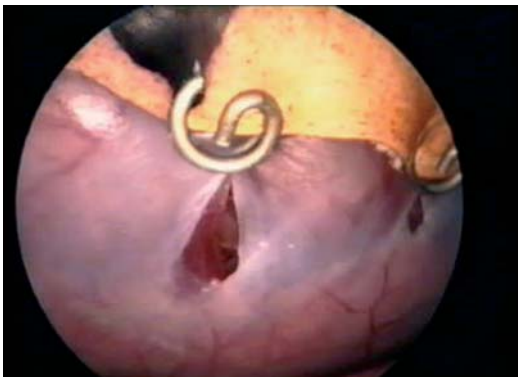


Fig. 17.5. Double crown technique for laparoscopic ventral hernia repair. Tear in the peritoneum and the muscle produced by a tack in a pig after increasing intra-abdominal pressure after a laparoscopic incisional hernia repair. These tears could be a tack hernia in the future

lastly, the learning curve of these initial series could be more directly related to the appearance of recurrences.

An analysis of our recurrences following the double crown technique shows that they were not directly related to the use of sutures [20] In a series that advocates the use of sutures, the recurrence rate ranges anywhere from 0 to 8.3%, with a mean of 3.98% [21]. The recurrence rate of our series is 2.14% with a mean follow-up of 40 months [20].

This surgical technique, double crown, has some advantages over the use of transfascial sutures that could be related to the presence of recurrences, since this technique reduces the dead space between the mesh and the anterior wall of the abdomen, which will reduce the amount of fluid from the seroma in this area, what will favour the ingrowth of the mesh, accelerating the biological fixation of the mesh. On the other hand, the inner crown of spiral tacks will guarantee a proper fixation of the mesh since the scar tissue at the edge of the defect will offer a consistent fixation.

How Could the Seroma be Decreased?

Seroma is a frequent complication of laparoscopic or open repair of ventral hernias. The presence of seroma is due to different factors, one of them related to the fact of leaving the sac in place during the procedure, since the sac is not excised once adhesiolysis is completed. A recent study revealed the presence of seroma in 100% of patients when an ultrasound examination is done,

Table 17.2. Decrease in the rate of seroma after injection of fibrin glue (Tissucol, Baxter Biosurgery) in the sac of the hernia

	1 week	1 month	3 months
Seroma without fibrin glue [%]	95.2	52.9	0
Seroma with fibrin glue [%]	66.6	8.3	0

while it is diagnosed clinically in only 35% of cases [23], this rate being very variable in the literature; but the real incidence has not been established properly. Seroma can produce pain and discomfort in the abdominal wall of the patients, and could also have some influence in recurrences for two reasons: the fluid between the mesh and the abdominal wall will delay ingrowth of the prosthetic material and, on the other hand, aspiration of the content has the risk of introducing bacteria, resulting in infection and the recurrence of the hernia.

Different methods have been proposed to decrease the incidence of seromas with not too good results, by cauterizing the sac by monopolar cautery, harmonic scalpel [24] or using argon beam. For these reasons we have been working to reduce the presence of seroma after the laparoscopic repair. The injection of fibrin glue (Tissucol, Baxter Biosurgery) in the sac of the hernia, after the repair has been completed, has reduced the rate of seromas after the surgery in a study we are conducting in our hospital. Our preliminary results show a reduction of the presence of the seroma 1 week after surgery from 95.2 to 66.6% and from 52.9 to 8.2% after 1 month (Table 17.2).

Laparoscopy as a Factor in Production of Hernias

Hernia at Trocar Sites

Incisional hernia after laparoscopic surgery is related to trocar sites. Such hernias are attributed to the difficulty of applying standard suturing techniques to wound closure, and to the fact that intra-operative

dislodgment and re-insertion of working trocars may create fascial defects larger than the actual size of the trocar. Trocar-site incisional hernias and their complications are reported in 1 to 6% of patients [25], and they have even been described after using 3-mm trocars [26]. The following risk factors for the development of trocar site hernias have been identified: the trocar diameter, the trocar design, pre-existing fascial defects, and some operation- and patient-related factors. Peritoneal and fascial closure should be done when blunt trocars of >10 mm have been employed. In order to avoid these hernias, fascial defects of 10 mm or larger should be closed, including the peritoneum. Opinion varied if a 5-mm trocar site defect should be closed [27], although complications have been reported in the literature related to this trocar size especially in infants, this fact have lead to recommend to close this trocar in children [28]. The site of the trocar is also important since umbilical trocars are more likely to produce incisional hernia than extraumbilical hernias. The frequency of incisional hernias at extraumbilical 10 and 12 mm trocar insertion sites is 0.23 and 3.1% [29], respectively, rates lower than series in which umbilical trocars are included.

With increasing numbers of laparoscopic procedures more postoperative trocar site hernias can be expected; 23% of the laparoscopic ventral hernias repair we performed in our unit in 2005 were hernias at the trocar sites. This complication of minimally invasive surgery is rare but potentially dangerous. Among trocar site hernias, Richter's hernias are the most frequent, accounting for two-thirds of all small intestinal hernias [30].

Umbilical Hernia and Laparoscopic Approach

The incidence of umbilical hernias following laparoscopic surgery varies from 0.02–3.6%. The incidence of pre-existing fascial defects, however, may be as high as 18% in patients undergoing abdominal laparoscopic surgery [26]. The presence of pre-existing fascial defects can cause increased morbidity in any laparoscopic surgery, and may predispose the patient to site herniation. The detection and management of these defects is crucial in preventing postlaparoscopic complications.

In our institution, 6.4% of the patients who underwent a laparoscopic cholecystectomy in 2005 showed the presence of an umbilical hernia, which is considered a factor that increases the possibility of developing an incisional hernia at this trocar site. Our approach to the combination of these pathologies in a patient who undergoes a laparoscopic cholecystectomy is as follows: in case the hernia is smaller than 3–4 cm a herniorraphy is performed, but if the hernia is larger than 4 cm a laparoscopic mesh repair is carried out, if the condition of the gallbladder allows this repair to be performed without any risk of contamination of the mesh.

Tack Hernias

Laparoscopic repair of incisional and ventral hernias is evolving into the armamentarium of many surgeons. Currently, most surgeons use transfascial permanent sutures to fixate the mesh securely or the double crown technique to perform this surgery. The placement of additional tacks along the periphery of the prosthesis, in those who use transfascial sutures, serves to approximate the patch so that ingrowth of tissue can occur and also to prevent the migration of bowel between the sutures. Since the helicoidal tacks have been available, surgeons have used them to secure the mesh without apparent complications. The increased use of this technique may identify unusual or unexpected outcomes. One of these has been identified as a tack hernia. Tack hernia was first described by LeBlanc in 2003 [31], due to the presence of a hernia in two patients previously submitted to a laparoscopic ventral hernia repair. This new entity is a hernia at the site were the tacks were placed. This discovery of fascial disruption by these devices that has resulted in herniation is a completely new finding. Conceivably, there may be several of these "failures" noted in the future. Conversely, it is also possible that some of the recurrent hernias that have been reported in the literature may, in fact, have been the result of the development of these tack hernias. It is believed that once these hernias have enlarged significantly, it may be quite difficult to identify the etiology with absolute certainty.

In a study conducted in our institution, we have demonstrated that the increase in the intra-abdominal pressure in the immediate postoperative period may be the etiologic pathology that results in the production of this new entity. Ten pigs underwent a laparoscopic repair of a ventral hernia following the double crown technique in our lab. Following the implantation of a ePTFE mesh, pigs were submitted to the maximal human physiological increase of intraabdominal pressure from 113 mmHg (Valsalva manoeuvre) to 277 mmHg (during weight lifting in male athletes). This increase in intra-abdominal pressure was performed immediately following patch implantation, five times during 20 s duration, and during a single duration of 5 min. Any observable changes between the interface of the mesh

and the abdominal wall of the pigs were observed. Tears in the peritoneum, fascia and/or muscle were observed in five of the ten pigs studied (50%) subsequent to the initial increase in intra-abdominal pressure. These tears were observed along the edge of the mesh at the tack sites and resulted from the movement of the mesh that was caused by the increase of the intra-abdominal pressure and the distension of the abdominal wall.

The conclusion of this study shows that the avoidance in any cause of the increase in the intra-abdominal pressure following surgery during the post-anesthesia period or by postoperative emesis may prevent this entity.

References

1. LeBlanc KA, Booth WV. Laparoscopic repair of incisional abdominal hernias using expanded polytetrafluoroethylene: preliminary findings. Surg Laparosc Endosc 1993; 3 (1): 39–41
2. Holzman MD, Purut CM, Reintgen K, Eubanks S, Pappas TN. Laparoscopic ventral and incisional hernioplasty. Surg Endosc 1997;11(1): 32–35
3. Park A, Birch DW, Lovrics P. Laparoscopic and open incisional hernia repair: a comparison study. Surgery 1998;124(4): 816–821
4. Ramshaw BJ, Esartia P, Schwab J, Mason EM, Wilson RA, Duncan TD, Miller J, Lucas GW, Promes J. Comparison of laparoscopic and open ventral herniorrhaphy. Am Surg 1999;65(9): 827–831
5. Carbajo MA, Martin del Olmo JC, Blanco JI, de la Cuesta C, Toledano M, Martin F, Vaquero C, Inglada L. Laparoscopic treatment vs open surgery in the solution of major incisional and abdominal wall hernias with mesh. Surg Endosc 1999;13(3): 250–252
6. DeMaria EJ, Moss JM, Sugerman HJ. Laparoscopic intraperitoneal polytetrafluoroethylene (PTFE) prosthetic patch repair of ventral hernia. Prospective comparison to open prefascial polypropylene mesh repair. Surg Endosc 2000;14(4): 326–329
7. Chari R, Chari V, Eisenstat M, Chung R. A case controlled study of laparoscopic incisional hernia repair. Surg Endosc 2000; 14(2): 117–119
8. Robbins SB, Pofahl WE, Gonzalez RP. Laparoscopic ventral hernia repair reduces wound complications. Am Surg 2001;67(9): 896–900
9. Wright BE, Beckerman J, Cohen M, Cumming JK, Rodriguez JL. Is laparoscopic umbilical hernia repair with mesh a reasonable alternative to conventional repair? Am J Surg 2002;184(6): 505–508
10. McGreevy JM, Goodney PP, Birkmeyer CM, Finlayson SR, Laycock WS, Birkmeyer JD. A prospective study comparing the complication rates between laparoscopic and open ventral hernia repairs. Surg Endosc. 2003;17(11): 1778–1780
11. Birgisson G, Park AE, Mastrangelo MJ Jr, Witzke DB, Chu UB. Obesity and laparoscopic repair of ventral hernias. Surg Endosc 2001;15(12): 1419–1422
12. Bageacu S, Blanc P, Breton C, Gonzales M, Porcheron J, Chabert M, Balique JG. Laparoscopic repair of incisional hernia: a retrospective study of 159 patients. Surg Endosc 2002;16(2):345–348
13. Carbajo MA, Martp del Olmo JC, Blanco JI, Toledano M, de la Cuesta C, Ferreras C, Vaquero C. Laparoscopic approach to incisional hernia. Surg Endosc 2003;17(1): 118–122
14. LeBlanc KA, Whitaker JM, Bellanger DE, Rhynes VK. Laparoscopic incisional and ventral hernioplasty: lessons learned from 200 patients. Hernia 2003; 7(3): 118–124
15. Condon RE, DeBord JR. Expanded polytetrafluoroethylene prosthetic patches in repair of large ventral hernia. In: Nyhus LM, Condon RE eds. Hernia (4th edn). Philadelphia: Lippincott Willians & Wilkins 1995; 20: 328–336
16. Morales-Conde S, GRETHAL. Laparoscopic ventral hernia repair: our experience with 1005 cases. 12th International Congress of the European Association for Endoscopic Surgery (EAES) 9–12 June 2004: p 54
17. Park A, Gagner M, Pomp A. Laparoscopic repair of large incisional hernias. Surg Laparosc Endosc 1996; 6(2): 123–128
18. LeBlanc KA, Booth W, Whitaker JM. Laparoscopic repair of ventral hernias using an intraperitoneal onlay patch: report of current results. Comtemp Surg 1994; 45(4)
19. Morales-Conde S, Cadet I, Tutosaus JD, Carrasco P, Palma F, Morales-Méndez S. Macroscopic evaluation of mesh incorporation placed intraperitoneally for laparoscopic ventral hernia repair. Experimental model. Proceedings of the 7th World Congress of Endoscopic Surgery (Singapore June 1–4, 2000). Monduzzi Editore, Bologna, Italy. 2000: 455–460
20. Morales-Conde S, Cadet H, Cano A, Bustos M, Martin J, Morales-Mendez S. Laparoscopic ventral hernia repair without sutures--double crown technique: our experience after 140 cases with a mean follow-up of 40 months. Int Surg 2005; 90(3 Suppl): S56–62
21. Morales-Conde S. Laparoscopic ventral hernia repair: advances and limitations. Semin Laparosc Surg 2004; 11(3):191–200
23. Susmallian S, Gewurtz G, Ezri T, Charuzi I. Seroma after laparoscopic repair of hernia with PTFE patch: is it really a complication? Hernia 2001; 5(3): 139–141
24. Tsimoyiannis EC, Siakas P, Glantzounis G, Koulas S, Mavridou P, Gossios KI. Seroma in laparoscopic ventral hernioplasty. Surg Laparosc Endosc Percutan Tech 2001;11(5): 317–321
25. Di Lorenzo N, Coscarella G, Lirosi F, Pietrantuono M, Susanna F, Gaspari A. Trocars and hernias: a simple, cheap remedy. Chir Ital 2005; 57(1): 87–90
26. Bergemann JL, Hibbert ML, Harkins G, Narvaez J, Asato A. Omental herniation through a 3-mm umbilical trocar site: unmasking a hidden umbilical hernia. J Laparoendosc Adv Surg Tech A 2001; 11(3): 171–173
27. Tonouchi H, Ohmori Y, Kobayashi M, Kusunoki M. Trocar site hernia. Arch Surg 2004; 139(11): 1248–1256
28. Nakajima K, Wasa M, Kawahara H, Hasegawa T, Soh H, Taniguchi E, Ohashi S, Okada A. Revision laparoscopy for incarcerated hernia at a 5-mm trocar site following pediatric laparoscopic surgery.Surg Laparosc Endosc Percutan Tech 1999; 9(4): 294–295
29. Kadar N, Reich H, Liu CY, Manko GF, Gimpelson R. Incisional hernias after major laparoscopic gynecologic procedures. Am J Obstet Gynecol 1993; 168(5): 1493–1495

30. Holzinger F, Klaiber C. Trocar site hernias. A rare but potentially dangerous complication of laparoscopic surgery. Chirurg 2002; 73(9): 899–904
31. LeBlanc KA. Tack hernia: a new entity. JSLS 2003; 7(4): 383–387

Discussion

Read: *You talked about removing fat from the abdomen, and I think we have to be careful when we describe fatty deposits we have to remember there is an extraperitoneal fatty layer and this may herniate, then there can be separate lipomas, there can even be leiosarkomas, so I think we have to be careful in the use of terms in regard to the abdominal fat.*

Van Geffen: *I want to go back some steps to the indication, because in the whole group of patients with incisional hernias there is on the one hand a primary incisional and on the other hand patients with an abdominal disaster, such as severe pancreatitiy, treatment with open abdomen. Where are the borders of laparoscopic approach? Within which indications do you try a laparoscopic approach in a patient or not?*

Morales Conde: *Talking about indication we have our philosophy. We can talk about size and adhesions, but the cases where you should avoid a laparoscopic approach are those with previous tuberculosis or patients after radiotherapy. The problem about the other cases with peritonitis … you have to find a safe access to the abdomen.*

Another thing is, it depends how the rectus muscle is, so maybe in some cases you need to approximate the rectus muscle to have a physiological function of the abdominal wall, something we don't know yet.

Van Geffen: *So if I understand you right, beside the patients with an enormous loss of abdominal wall there is no contra-indication to trying to get access and trying a laparoscopic approach.*

18 Bioprostheses: Are They the Future of Incisional/Acquired Hernia Repair?

M.G. Sarr, E. Podgaetz, J.S. Lane

Introduction

Our understanding of the pathobiology of hernia formation has grown tremendously in the past decade. Indeed, several well-designed, molecular-based studies have shown that many acquired hernias (e.g. direct inguinal hernia) as well as incisional hernias arise in the setting of measurable abnormalities in tissue healing – either in the production of the various types of collagen or in the remodeling and breakdown via matrix metalloproteinases [1, 2]. In addition, several well-conducted, long-term studies (>5 years follow-up) have shown that the rate of recurrences after primary autogenous repairs of incisional hernias is really much greater than we ever appreciated previously [3, 4].

These types of studies in the basic science of hernia formation, complemented by clinical experience with prosthesis-based repairs, have virtually revolutionized the field of inguinal herniorraphy. Few surgeons currently would consider repairing a direct inguinal hernia without some form of permanent prosthetic material – quite a change from the classic, accepted autogenous repairs of Bassini and McVay of just 20–30 years ago.

But, where are we currently with the concept of optimal repair of incisional hernias? The scientific data supporting the presence of a basic abnormality in the biology of tissue healing in the majority of patients who develop incisional hernias (in the absence of any technical factors or tissue loss) are compelling [1, 2]. Combine this mechanistic data with the (now appreciated) inordinately high rate of recurrence after primary autogenous repair [3, 4], and one might wonder why all abdominal incisional hernias are not also repaired routinely with some form of prosthesis reinforcement.

Several explanations prevail. First, the rate of recurrence after repair is not well appreciated. We all (as surgeons) think we do excellent repairs (they all look great intra-operatively), yet we also know that many patients will seek a different surgeon when a recurrence occurs, and often we never find out about the recurrence (unless we look, look for up to 10 years!). Second, prosthetic-based repairs involve more dissection, especially when performed as a sublay repair, take more time, and require a more involved anesthetic (and possibly a longer and more costly hospitalization). Third, the prostheses are expensive, especially when placed with broad lateral overlap. And, fourth, and possibly the most important, all surgeons are worried about mesh infection and the potential of mesh erosion/bowel fistula.

Concepts of Bioprostheses

If one accepts the concept that some additional form of support is necessary for incisional herniorraphy because of the unacceptably high recurrence rate with autogenous repairs, and that lack of a permanent foreign body would be optimal, then several approaches have been proposed. Currently, most reinforced incisional hernia repairs use synthetic pros-

theses such as polypropylene or expanded polytetrafluoroethylene (ePTFE). The polypropylene mesh generates an intense, perifilamentous inflammatory reaction resulting in the mesh being encapsulated by a rigid scar plate and shrinking in surface area by as much as 30% [5, 6]. Similar changes occur at the interface of the native abdominal wall and the ePTFE. The permanent scar plate represents a chronic, foreign-body reaction that reduces abdominal wall compliance, increases the risk of recurrent hernia, and continues to predispose to infection. Such concerns have spawned the interest in bioprostheses designed both to limit the foreign-body reaction (eventually the components of the bioprosthesis will be broken down and fully absorbed) and to regenerate as normal and compliant a neo-abdominal wall as possible.

Autografts

Fascia lata autografts (from the same patient) have been utilized to bridge fascial gaps or to reinforce tenuous, autogenous closures. These nonvascularized, free fascial transfers, though initially strong, are degraded via an inflammatory reaction and are eventually re-absorbed. Long-term studies of durability have not yielded great success. Similarly, homografts of fascia from cadavers would be even less successful, in part, because of an allogeneic immune response.

Xenograft Bioprostheses

Nontreated xenografts (animal tissue) are, of course, contra-indicated because of the intense immune response generated by a multitude of foreign antigens. Several technologies, however, have emerged to chemically treat such xenografts in a way that removes essentially all cells and most associated foreign antigens, leaving (allegedly) just the components of the extracellular matrix. The basic concept is that the resultant biograft minimizes the immune reaction, and by serving as a lattice work, stimulates the ingrowth of tissue stem cells and fibroblasts, vascularization, and ultimately the laying down of a strong, dynamic, plastic neofascia. The components of the underlying xenograft are then re-absorbed slowly and replaced by a more functional, host-derived neofascia and host-derived extracellular matrix. Being a re-absorbable bioprosthesis, which allows vascularization, both the acute and especially the long-term risks of infection are less than with permanent prosthetic materials.

The principle behind the xenograft bioprostheses relies on processing the initial xenograft to minimize both the immune and inflammatory reaction and maximizing host-derived tissue repair. The process must thus stimulate vascularization, recruitment of growth factors, and ultimately the development of a stable, neo-abdominal wall from host-derived tissue healing/repair. Examples of treated xenografts include porcine, submucosal bioprostheses (Surgisis, Cook Surgical, Bloomington, IN, USA), treated porcine dermis bioprostheses (Permacol, Tissue Science Laboratories, Covington, GA, USA) and treated bovine pericardial collagen bioprosthesis (Veritas, Synovis Surgical Innovations, Lino Lakes, MN, USA).

Allograft Bioprostheses

Similar in principle to treated xenografts, allogeneic (from human tissue) biografts have been developed as well. Human dermis harvested from cadavers can be treated to remove all cells and allogeneic antigens, leaving intact the three-dimensional extracellular matrix, as well as the structural components of the vascular channels devoid of endothelial cells. Being allogeneic as opposed to xenogeneic, allegedly the ability to recruit tissue stem cells and to initiate the host-derived healing/repair process is superior to xenograft bioprostheses. In addition, maintaining the vascular tubes allows so-called inosculation, i.e., host-derived endothelialization of these vascular channels, which speeds the vascularization of this extracellular matrix. By maintaining the three-dimensional structural component of the extracellular matrix and allowing inosculation, allegedly the process of tissue repair/remodeling can generate a structurally complete, plastic, and pliable neo-abdominal wall rather than the post-inflammatory scar/fibrosis often generated by the treated xenograft bioprostheses. An example of an allograft bioprosthesis is treated human dermis matrix (Alloderm, LifeCell, Branchburg, NJ, USA).

Unknown Considerations in Bioprostheses

Proponents of the expanded use of bioprostheses cite the advantages of an increased resistance to infection, the lack of permanent foreign material and its primary inflammatory response, and the host's ability to form endogenously a functional neo-abdominal wall. This latter contention is potentially problematic for the same reasons that the incisional or acquired hernias form in

the first place. Patients who form these hernias have a basic defect in tissue healing/repair. Whether or not these bioprostheses can induce a robust-enough response and a durable neofascia/neoabdominal wall, though suggestive, is not proven, as will be discussed below. Indeed, good, objective, long-term (>5 years) studies (or experience), confirmed by several medical centers, remain absent.

Clinical Studies

Porcine Submucosal Acellular Extracellular Matrix (Surgisis)

This bioprosthesis is a resorbable biomaterial derived from the submucosal extracellular matrix of the pig small intestine. After removing the muscularis externa and the mucosa, the 80-μm submucosa is treated with 0.1% peracetic acid, which removes all cells, is rinsed, and sterilized with ethylene oxide; a construct of eight separate, stacked sheets of this three-dimensional extracellular matrix oriented at 45° angles to adjacent sheets are then subjected to vacuum compression. The resultant eight-sheet complex is perforated with 0.9-mm holes spaced about 7 mm apart [7]. In a dog model replacing a partial thickness, abdominal wall defect with an inlay of Surgisis at 3 months postimplantation, a well-organized, smooth, dense connective tissue of collagenous material appeared to be well-incorporated into adjacent fascia and muscle, closely resembling the native fascia [8,9]. The non-cross-linked preparation composed primarily of fibrillar collagens (types I, III, and IV) and various glycosamine glycans, proteoglycans, and glycoproteins, the relative lack of cellular antigens, and the minimal immune response allegedly supports a site-specific tissue remodeling [10,11].

Clinical studies specifically of ventral hernia repair using Surgisis, however, are limited to preliminary, short duration studies; there are no long-term (>5-year) studies available. When reviewing the reported studies [12–15], one must be cognizant of several considerations: first, follow-up is short and poorly defined in terms of definitions of recurrence and actual repeat physical examination/imaging; and second, many patients were recruited because of contaminated or grossly infected surgical fields [13–15]. Small, uncontrolled studies suggest good initial strength in preventing evisceration and minimizing dehiscence, and early studies suggested a minimal recurrent rate in clean wounds of 0% [12], 0% [14] and 15% [15], but allegedly with short <2-year mean follow-up. When carried out in potentially or grossly contaminated wounds, recurrence rates increased to 20–80% [13,15]; several episodes of infection and partial or total degradation (digestion) of the Surgisis were described, stimulating Helton et al. [15] to suggest leaving the skin edges open with the Surgisis exposed and the placement of a wound vacuum device. Superficial infection may lead to a peeling off of the superficial layer(s) of the eight-ply prosthesis but ingrowth into the deeper layers of the biograft. In addition, these latter investigators described a short-lived, noninfective inflammatory reaction characterized by erythema, induration, and pain that responded to nonsteroidal anti-inflammatory agents; this reaction was neither noted nor described, curiously, in the other series [12–14].

In summary, use of Surgisis as a bioprosthesis has performed reasonably well as a biologic dressing in contaminated wounds, is considerably cheaper than several of the other bioprostheses (Permacol, Alloderm), and appears effective in the short term; however, long-term studies with objective criteria of follow-up and recurrence are lacking.

Porcine Acellular Cross-Linked Dermal Collagen Implant (Permacol)

Permacol is made from pig dermis, and, unlike Surgisis or Alloderm, is subjected to trypsinization and extraction of all cellular elements and genetic material followed by gamma irradiation and a proprietary cross-linking process using hexamethylene diisocyanate to provide a strong, chemical bonding stability of the extracellular matrix; this chemical cross-linking makes the bioprosthesis more resistant to degradation by human and bacterial collagenase. The resulting biograft is a three-dimensional material consisting largely of elastin and types-I and -III collagen. This technology also is designed to elicit a minimal inflammatory response but to maintain form and strength while host tissues integrate into the bioscaffold. Published experimental [16] and clinical [17] studies of Permacol are very limited, but unpublished, anecdotal abstracts presented at national meetings are supportive.

Implantation in a rat model showed infiltration by neovascular channels and a present, albeit less intensive, foreign-body reaction; there were no neutrophils, lymphocytes, or macrophages at 12 weeks postimplant, and no (ostensible) structural changes in the collagen organization and content, presence of elastin, and thickness of the biograft, although these parameters were not quantitated or assessed biomechanically [16].

Clinically, Adedeji et al. [17] report one patient repaired successfully in a contaminated wound without recurrence at 12 months. Several unpublished "reports" available from the manufacturer of a total of 72 patients with ventral hernias repaired with Permacol relate a recurrence rate of about 8%, but no data are available concerning duration and type of follow-up, and these reports remain unpublished.

Allogenic (Human) Acellular Dermal Matrix (Alloderm)

This biograft was engineered to maintain a three-dimensional structure of extracellular matrix from human tissue and without chemical cross-linking, thereby further decreasing the possibility of immune reaction. In brief, partial thickness sheets of cadaver skin are harvested with a dermatome; using proprietary methodology, the epidermis separated off, and all cells are removed by sodium deoxycholate which simultaneously eradicates donor major histocompatibility class-I and -II antigens [18, 19]. After freeze-drying, the biomaterial contains a structurally intact basement membrane with the overlying matrix containing glycosaminoglycans, intact fibers and bundles of type I, III, IV, and VII collagen, and intact elastin and laminin [20, 21]. Moreover, the biomaterial maintains the extracellular matrix of the vascular tubes devoid of endothelial cells, allegedly permitting a more rapid revascularization (inosculation) of the bioprosthesis (personal communication, J. Harper, LifeCell).

Experimental work in animal models is supportive in principle. Rabbit [22] and pig [23] models of abdominal wall defects confirmed a rapid revascularization and cellular repopulation without a substantive inflammatory foreign-body reaction of the bioprosthesis yet maintenance of essentially normal breaking strength as evaluated by tensiometry.

As with Permacol, published clinical studies are limited and represent initial preliminary results [18, 24–28]. After an anecdotal report [26], Hirsch and colleagues [25] introduced its use for early definitive closure of the open abdomen, which had been reported previously in nine patients by Guy and colleagues [27] with good results. The use of Alloderm in the elective setting, either for repair of fascial weaknesses/repairs in 18 patients after TRAM flaps [28] and in 85 patients for abdominal wall hernias or defects [18,28,24] also is promising, although again, follow-up is short, and details of the follow-up are lacking; recurrences after repair of ventral hernias or postresection abdominal wall defects were noted but were <15%.

Summary

Current standard of care for repair of incisional hernias probably should involve some form of permanent prosthetic reinforcement unless the hernia is secondary to technical mishap, a limited defect secondary to tissue loss, or the operative field is potentially (clean-contaminated) or grossly contaminated. Currently, under these latter situations, the use of a bioprosthesis has considerable support in the literature and possibly is a better choice than a temporary polyglycolic acid closure which will lead to an abdominal wall defect. Although very attractive in theory, and preliminary, largely anecdotal experience suggests good results using a bioprosthesis rather than a permanent prosthesis for elective repairs. The current literature is as yet too immature in terms of breadth of experience, duration of detailed objective follow-up, and well-designed, prospective, randomized studies to support the replacement of the newer, soft, lightweight permanent prostheses with a bioprosthesis in elective, clean incisional herniorrhaphies.

References

1. Jansen PL, Mertens PR, Klinge U, Schumpelick V. The biology of hernia formation. Surgery 2004;136: 1–4
2. Bellon JM, Bajo A, Ga-Honduvilla N, Gimeno MJ, Pascual G, Guerrero A, Bujan J. Fibroblasts from the transversalis fascia of young patieints with direct inuibnal hernias show constitutive MMP-2 overexpression. Ann Surg 2001;233: 287–291
3. Burger JWA, Luijendijk RW, Hop WCJ, Halm JA, Verdaasdonk EGG, Jeekel J. Long-term follow-up of a randomized controlled trial of suture versus mesh repair of incisional hernia. Ann Surg 2004;240: 578–585
4. Luijendijk RW, Hop WCJ, van den Tol MP, de Lange DCD, Braaksma MMF, Ijzermans JNM, Boelhouwer RU, de Vries BC, Salu MKM, Wereldsma JCJ, Bruifninckx CMA, Jeekel J. A comparison of suture repair with mesh repair for incisional hernia. New Engl J Med 2000;343: 392–398
5. Klinge U, Klosterhalfen B, Muller M, Schumpelick V. Foreign body reaction to meshes used for the repair of abdominal wall hernias. Europ J Surg 1999;165: 665–673
6. Klinge U, Kosterhalfen B, Muller M, Ottinger AP, Schumpelick V. Shrinking of polypropylene mesh in vivo: an experimental study in dogs. Erop J Surg 1998;164: 965–969
7. Pu LL, and the Plastic Surgery Educational Foundation Committee. Small intestinal mucosa (Surgisis®) as a bioactive prosthetic material for repair of abdominal wall fascial defect. Plast Reconstr Surg 2005;115: 2127–2131
8. Clarke KM, Lantz GG, Salisbury SK, Badylak SF, Hiles MC, Voytik SL. Intestine submucosa and polypropylene mesh for abdominal wall repair in dogs. J Surg Res 1996;60: 107–114

9. Dejardin LM, Arnoczky SP, Clarke RB. Use of small intestinal submucosal implants for regeneration of large fascial defects: an experimental study in dogs. J Biomed Mater Res 1999;46: 203–211
10. Voytik-Harbin SL, Brightman AO, Waisner BZ, Robinson JP, Lamar CH. Small intestinal mucosa: a tissue-derived extracellular matrix that promotes tissue-specific growth and differentiation of cells in vitro. Tissue Eng 1998;4: 157–173
11. Allman AJ, McPherson TB, Merrill LC, Badylak SF, Metzger DW. The Th2-restricted immune response to xenogenic small intestinal submucosa does not influence systemic protective immunity to viral or bacterial pathogens. Tissue Eng 2002;8:53–62
12. Eid GM, Mattar SG, Hamad G, Cottam DR, Lord JL, Watson A, Dallal RM, Schauer PR. Repair of ventral hernias in morbidly obese patients undergoing laparoscopic gastric bypass should not be deferred. Surg Endosc 2004;18: 207–210
13. Ueno T, Pickett LC, de la Fuente SG, Lawson DC, Pappas TN. Clinical application of porcine small intestinal submucosa in the management of infected or potentially contaminated abdominal defects. J Gastrointest Surg 2004;8: 109–112
14. Franklin ME Jr, Gonzalez JJ Jr, Glass JL. Use of porcine small intestinal submucosa as a prosthetic device for laparoscopic repair of hernias in contaminated fields: 2-year follow-up. Hernia 2004;8: 186–189
15. Helton WS, Fisichella PM, Berger R, Horgan S, Espat NJ, Abcarian H. Short-term outcomes with small intestinal submucosa for ventral abdominal hernia. Arch Surg 2005;140: 549–560
16. Macleod TM, Wiliams G, Sanders R, Green CJ. Histological evaluation of Permacol™ as a subcutaneous implant over a 20-week period in the rat model. Brit J Plast Surg 2005;58:518–532
17. Adedeji OA, Bailey CA, Varma JS. Porcine dermal collagen graft in abdominal-wall reconstruction. Br J Plastic Surg 2002;55: 85–86
18. Holton LH, Kim D, Silverman RP, Rodriguez ED, Singh N, Goldberg NH. Human acellular dermal matrix for repair of abdominal wall defects: review of clinical experience and experimental data. J Long-Term Effects of Med Implants 2005;15: 547–558
19. Wainwright D, Madden M, Luterman A, Hunt J, Monafo W, Heimbach D, Kagan R, Sittig K, Dimick A, Herndon D. Clinical evaluation of an accellular alograft dermal matrix in full-thickness burns. J Burn Care Rehabil 1996;172: 124–136
20. Eppley BL. Experimental assessment of the revascularization of acellular human dermis for soft-tissue augmentation. Past Reconstr Surg 2001;107: 757–762
21. Adhikary S, Beniker HD, Garfield J, Griffey ES, Harper JA, Livesey SA, McQuillan DJ, Ott D, Owens RT. Biochemical and ultrastructural characterization of an acellular extracellular matrix scaffold (AlloDerm® an dCymetra™): utility in tissue regeneration and potential for gene delivery. American Society for Matrix Biology, 2002
22. Menon NG, Rodriguez ED, Byrnes CK, Girotto JA, Goldberg NH, Silverman RP. Revascularization of human acellular dermis in full-thickness abdominal reconstruction in the rabbit model. Ann Plast Surg 2003:50: 523–527
23. Silverman RP, Li EN, Holton LH 3rd, Sawan KT, Goldberg NH. Ventral hernia repair using allogenic acellular dermal matrix in a swine model. Hernia 2004;8: 336–342
24. Butler CE, Langstein HN, Kronowitz SJ. Pelvic, abdominal, and chest wall reconstruction with AlloDerm in patients at increased risk for mesh-related complications. Plast Reconstr Surg 205;116: 1263–1277
25. Scott BG, Feanny MA, Hirshberg A. Early definitive closure of the open abdomen: a quiet revolution. Scand J Surg 2005;94:9–14
26. Hirsch EF. Repair of an abdominal wall defect after a salvage laparotomy for sepsis. J Am Coll Surg 2004;198: 324–328
27. GuyJS, Miller R, Morris JA Jr, Diaz J, May A. Early one-stage closure in patients with abdominal compartment syndrome: fascial replacement with human acellular dermis and bipedicle flaps. Am Surg 2003;69: 1025–1028
28. Buinewicz B, Rosen B. Acellular cadaveric dermis (AlloDerm): a new alternative for abdominal hernia repair. Ann Plast Surg 2004;52: 188–194

Discussion

Halm: *In our own rat animal model we tested quite a number of prostheses, resorbable and non-resorbable, and also the bioprosthesis to mesh, Pericard*
We found absolutely no adhesions to the Tuto mesh.
The question I have is: *you spoke briefly of fistula formation when placing a polypropylene mesh within the abdominal cavity. We have one study that was performed by a colleague of mine, who found in a retrospective setting of a larger group of patients no fistula and I recently concluded a series of patients all with an intraperitoneal polypropylene mesh and found very little fistula formation, 3%, that was two patients. Is fistula formation really a problem?*
Sarr: *I think fistula formation is a problem and 3% is unsatisfactory, if you ask me. It usually occur at the edge where it rolls over because the bowel gets stuck up under it. Your idea of using it as a barrier in the abdomen is a very attractive concept of bioprosthesis because at least in theory it shouldn't form any adhesions. I maintain that the concern of permanent intraperitoneal prostheses that is not a non-mesh, Gore Tex is really a non-mesh, it is a problem.*
LeBlanc: *I am really surprised how many people use these bioprostheses without any data. I have used almost all of them and I had a 100% failure rate, every single one of them. I think the only time that it might be effective is in combination with an abdominal component separation technique, and laying something like Permacol on top. I had good results with that in an infected field.*
Schumpelick: *Dr. Sarr, do you see any future for stem-cell implantation?*
Sarr: *It has all been done as far I can say. The whole idea behind tissue engineering is to get the tissue-specific stem cells in the environment with the growth factors in that*

area to regenerate the linea alba, to regenerate fascia, to regenerate bowel, to regenerate bladder.. we must keep an open mind. I think that this is the future!
Köckerling: *You have mentioned the disadvantages of the polypropylene meshes with 30% shrinkage rate and scar formation. This is really true for the heavy-weight polypropylene meshes, but no longer true for the light-weight meshes because we have done some experimental work and the shrinkage rate of the modern meshes is around 3% and you will find no scar formation, just around the filaments not over the whole surface.*
Sarr: *But does it regenerate a neo-abdominal wall?*
Köckerling: *Yes, as far as we can say from our experience.*

Concluding Remarks

Read: *I just want to say that in this session and in this conference here, we have learned that we have to separate the responsibility of the surgeon in the wound and repair from the biological problem. I think for the future that when we pre-operatively have some assessment of the degree of biological comorbidity and when we have a series of patients in whom we can separate out the importance of biological comorbidity, then we can determine the importance of the surgeon. We are moving on rapidly in that field and I am pleased about it.*

Jeekel: *Closing the abdominal wall we know we should not, and should not have to close the peritoneum, we should close the line alba probably not with too large bites, very large bites can be very dangerous, do continuous suturing with non-resorbable or slowly resorbable suture with a suture-length-to-wound-length ratio of more than 4. Amazing is that we all recognize that we can prevent hernias and that we don't do it. As Dr Kehlet says, we don't do the transverse incision, … we don't use it because it is so easy to use the midline.*

Incisional Hernia

19 Whom to Operate?

C.K. Chan, G. Chan

Indications for Repair of Recurrent Incisional Hernia

The treatment of incisional hernias has changed dramatically with the innovation of mesh prostheses and with the introduction of laparoscopy, while the indications for repair of a recurrent incisional hernia have remained unchanged (Table 19.1). The decision regarding the technique and timing of a repair should be based on the characteristics of the hernia and the management of the risk factors for re-recurrence. Since a previous recurrence is a significant risk factor for failure [1], a strong emphasis should focus on the pre-operative preparation of the patient to ultimately improving the success rate of repair.

There are three common indications for elective repair of an incisional hernia recurrence. The main indication is symptoms. Clearly, pain and discomfort can adversely affect the patient's occupation and quality of life. In addition, dissatisfaction with the cosmetic appearance of a disfiguring bulge can negatively impact self-esteem and perception. Repair is also indicated for significant and progressive defect enlargement, in effect to prevent a loss of abdominal domain. The third indication is to prevent complications, such as strangulation, intestinal obstruction or incarceration.

The indication for semi-urgent repair is an association with either an entero-cutaneous fistula, skin erosion with exposed mesh or chronic infection. Any repair would be in a contaminated or dirty field. There is also a significant likelihood of concomitant bowel resection [2]. The operation should not be undertaken until the optimal treatment of wound sepsis has been achieved. The pre-operative medical management of infected meshes involves local wound care, drainage of collections and antimicrobial therapy. Once the degree of infection has been resolved or minimized, the infected mesh can be resected and the recurrence repaired, either immediately or staged. Immediate repair can be done primarily with sutures or with abdominoplasty [3], such as bilateral sliding rectus abdominis myofascial advancement flaps [2, 4]. The use of a permanent mesh has been described but is at very

Table 19.1. Indication for repair of incisional hernia recurrence

Elective	– Symptomatic (pain, disfiguring) – Enlargement and prevention of loss of domain – Prevention of complications (strangulation, obstruction incarceration)
Semi-urgent	– Chronically infected mesh – Skin erosion and mesh exposure – Enterocutaneous fistula
Urgent	– Strangulation and intestinal gangrene – Sepsis

high risk of complication in a dirty field [5–7]. More recently, intestinal submucosal meshes were developed and intended to diminish the rate of infection and re-recurrence in clean-contaminated wounds [8, 9], but long-term studies of these bioprostheses have yet to be published. In addition, preliminary evidence shows a high risk of recurrence and dehiscence when used in a dirty wound [10]. In staged repairs, the temporizing measures include using an absorbable mesh and possibly a skin graft [7, 11]. The delay of definitive repair should be for at least 6 months and up to 1 year, until such time as the hernia field is free of infection and the wounds are matured [12]. The options for definitive repair include primary repair, component separation or mesh. Postoperatively, closed suction drains and antimicrobial therapy are used commonly, although their effectiveness in preventing infection and re-recurrence has yet to be proven in clinical trials.

If the wound sepsis cannot be managed medically, urgent debridement of the infected mesh is necessary to obtain source control. A minimal attempt at hernia repair should be made at this initial stage, either by suture repair or with absorbable mesh. The emphasis is to conserve the maximal amount of tissue for the delayed definitive repair.

An urgent repair is also required for compromised or gangrenous intestine incarcerated in the recurrent hernia. A conservative staged approach would be indicated. Initially, the hernia should be reduced with intestinal resection, and the infected mesh and any necrotic tissue should be debrided. Preservation of healthy tissue is vital for the staged repair. The definitive repair can be delayed for up to a year allowing for wound maturation and patient recovery, as necessary.

Pre-Operative Preparation and Risk Factor Management

The pre-operative assessment of patients with incisional hernia recurrence has to be comprehensive. The management should be individualized and begins upon initial assessment. Several factors are important, including the characteristics of the hernia, the planned surgical technique and the risk factors for recurrence and re-recurrence. The principles should focus on the preparation of the abdominal wall for repair, and the treatment of comorbidities to optimize the patient medically. All of this is in a concerted effort to increase the likelihood of a successful repair.

The characteristics of the hernia can be defined according to location, size, recurrence, reducibility and symptoms [13]. In addition to physical examination, imaging with CT accurately determines the size, sac contents, and the number and location of defects [14]. Full knowledge of the quality of a hernia allows proper planning and avoids intra-operative surprises.

Today, the widespread use of imaging has widened the detection of incisional hernia recurrences. It is useful in situations of difficult clinical examination secondary to body habitus, pain or scarring. CT scanning is valuable in the diagnosis of occult hernias in obese patients with aspecific symptoms, and provides the definition of the location, sac and contents [15]. It is actually superior to physical exam in confirming a suspected symptomatic recurrence [16]. However, there is no indication for repair of occult or incidental hernias in asymptomatic patients [17]. The precise role of pre-operative imaging requires further study, and could address cost-effectiveness and the impact on recurrence.

The options of the surgical repair include the technique, the type of mesh prosthesis, location of mesh placement and the timing. The open sublay mesh repair or the Rives-Stoppa technique is the current gold standard. Mesh hernioplasty is indicated in the majority of recurrences, particularly for primary approximation under tension, proximity to bony landmarks, and multiple defects [18]. The choice of laparoscopic repair depends largely on the surgeon and hospital, and is applicable only to specific patients [19]. There are particular benefits in terms of postoperative pain and length of stay and, perhaps most appealing, the decrease in the amount of abdominal wall dissection [20]. The timing of the repair should not be rushed. Wound healing and maturation can take up to 1 year, so the elective repair should not be undertaken during this period.

The preparation of the abdominal wall for surgery is vital for successful repair. The two most significant risk factors for repair failure are infection and obesity. Obesity is a commonly cited risk factor for recurrence [22, 23] and for the development of incisional hernia [24, 25], even specifically in prosthetic repairs [21]. A massive volume of abdominal fat increases intra-abdominal pressure and limits the reducibility of the hernia for repair. The enormous weight of the panniculus literally pulls apart the surgical incision [26]. Weight loss is obviously desirable and definitely possible prior to repair. A supervised weight loss programme can contribute to good clinical results, as our group has previously published [18]. Our programme includes a diet eliminating the excess fat and carbohydrates that is typical to Western diets, multivi-

tamin supplementation, and a daily physical exercise routine to strength the abdominal muscles by sit-ups or straight leg lifts. If compliant over several months, the majority of overweight and obese patients can attain a BMI under 30 kg/m^2. The regime should also be enforced postoperatively.

The other major risk factor for recurrence is postoperative wound infection [18, 23, 27]. The vast majority of these complications can be treated conservatively with local wound care and occasionally require antibiotic treatment. Once a recurrence has occurred, it is of utmost importance to allow the infection to clear prior to attempting a repair. We will wait at least 1 year from the resolution of the infection before repair. Although chronic infection of the prosthesis may be adequately treated with drainage [28], a recurrence in this setting would necessitate a conservative staged repair. Placement of a permanent mesh in an infected wound has a high rate of complications [6].

Smoking is a risk factor for incisional hernia development [29], increased anaesthesia-related respiratory complications and postoperative wound infection [30]. Clinical trials have shown that abstinence from smoking (> 4 weeks) can decrease the rate of infection and peri-operative complications [31, 32]. Although there are no studies specific to incisional hernia recurrences, there is no reason not to implement a smoking cessation programme for this population.

There are a host of other comorbidities that may affect the durability of the repair (Table 19.2), such as chronic lung disease, ascites, cirrhosis, jaundice, diabetes, aneurismal disease, renal failure, malignancy and steroid treatment [33]. Many factors have been suggested, with a variable quality of evidence. Some of these may require medical optimization, although the actual effect on the outcome of hernioplasty is unknown.

The pre-operative management of an incisional hernia recurrence must be tailored to eliminate the factors that contributed to the failure of a repair. This requires dedicated time and effort to be addressed properly prior to re-operation.

Table 19.2. Risk factors for recurrent incisional hernias

Abdominal wall factors	– Infection – Obesity – Seroma/hematoma – Location – Size – Previous surgical technique – Surgeon
Comorbid disease	– Older age – Male sex – Collagen – Smoking – Renal failure – Chronic lung disease – Diabetes mellitus – Steroid use – Malignancy – Malnutrition – Anticoagulation therapy – Jaundice and cirrhosis

Conclusion

Incisional hernia repairs are common procedures whose results have improved significantly. The indications and goals of elective repair must be understood clearly by the patient, whether it is for symptomatic relief, cosmetic or preventative reasons. The factors that contributed to a recurrence need to be identified and addressed explicitly for significant degrees of compliance with any peri-operative care and management plans. Non-elective repairs require specialized care to save the maximal amount of healthy tissue, resolve the concomitant infection and then ultimately to fix the recurrence. The newest mesh technology aims to tackle this subpopulation; however, until clear evidence of improved outcomes is published, a conservative staged approach is the most prudent.

References

1. Manninen MJ, Lavonius M, Perhoniemi VJ (1991) Results of incisional hernia repair. A retrospective study of 172 unselected hernioplasties. Eur J Surg 157 : 29–31
2. Szczerba SR, Dumanian GA (2003) Definitive surgical treatment of infected or exposed ventral hernia mesh. Ann Surg 237: 437–441
3. Van Geffen HJ, Simmermacher RK (2005) Incisional hernia repair: abdominoplasty, tissue expansion, and methods of augmentation. World J Surg 29: 1080–1085
4. Disa JJ, Goldberg NH, Carlton JM, Robertson BC, Slezak S (1998) Restoring abdominal wall integrity in contaminated tissue-deficient wounds using autologous fascia grafts. Plast Reconstr Surg 101: 979–986
5. Voyles CR, Richardson JD, Bland KI, Tobin GR, Flint LM, Polk HC Jr (1981) Emergency abdominal wall reconstruction with polypropylene mesh: short-term benefits versus long-term complications. Ann Surg 194: 219–223
6. Jones BV, Sanchez JA, Vinh D (1989) Acute traumatic abdominal wall hernia. Am J Emerg Med 7: 667–668

7. 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
8. Franklin ME Jr, Gonzalez JJ Jr, Michaelson RP, Glass JL, Chock DA (2002) Preliminary experience with new bioactive prosthetic material for repair of hernias in infected fields. Hernia 6: 171–174
9. Ueno T, Pickett LC, de la Fuente SG, Lawson DC, Pappas TN (2004) Clinical application of porcine small intestinal submucosa in the management of infected or potentially contaminated abdominal defects. J Gastrointest Surg 8: 109–112
10. Helton WS, Fisichella PM, Berger R, Horgan S, Espat NJ, Abcarian H (2005) Short-term outcomes with small intestinal submucosa for ventral abdominal hernia. Arch Surg 140: 549 560; discussion 560–542
11. Fabian TC, Croce MA, Pritchard FE, Minard G, Hickerson WL, Howell RL, Schurr MJ, Kudsk KA (1994) Planned ventral hernia. Staged management for acute abdominal wall defects. Ann Surg 219: 643 650; discussion 651–643
12. Cohen M, Morales R Jr, Fildes J, Barrett J (2001) Staged reconstruction after gunshot wounds to the abdomen. Plast Reconstr Surg 108: 83–92
13. Korenkov M, Paul A, Sauerland S et al. (2001) Classification and surgical treatment of incisional hernia. Results of an experts' meeting. Langenbecks Arch Surg 386: 65–73
14. Zarvan NP, Lee FT Jr, Yandow DR, Unger JS (1995) Abdominal hernias: CT findings. AJR Am J Roentgenol 164: 1391 1395
15. Ianora, AA, Midiri M, Vinci R, Rotondo A, Angelelli G (2000) Abdominal wall hernias: imaging with spiral CT. Eur Radiol 10: 914–919
16. Gutierrez de la Pena C, Vargas Romero J, Dieguez Garcia JA (2001) The value of CT diagnosis of hernia recurrence after prosthetic repair of ventral incisional hernias. Eur Radiol 11: 1161–1164
17. Rodriguez HE, Matsumura JS, Morasch MD, Greenberg RK, Pearce WH (2004) Abdominal wall hernias after open abdominal aortic aneurysm repair: prospective radiographic detection and clinical implications. Vasc Endovascular Surg 38: 237–240
18. Chan G, Chan CK (2005) A review of incisional hernia repairs: preoperative weight loss and selective use of the mesh repair. Hernia 9: 37–41
19. SSAT patient care guidelines (2004) Surgical repair of incisional hernias. J Gastrointest Surg 8: 369–370
20. Cobb WS, Kercher KW, Heniford BT (2005) Laparoscopic repair of incisional hernias. Surg Clin North Am 85: 91–103, ix
21. Anthony T, Bergen PC, Kim LT, Henderson M, Fahey T, Rege RV, Turnage RH (2000) Factors affecting recurrence following incisional herniorrhaphy. World J Surg 24: 95–100; discussion 101
22. Sauerland S, Korenkov M, Kleinen T, Arndt M, Paul A (2004) Obesity is a risk factor for recurrence after incisional hernia repair. Hernia 8: 42–46
23. Hesselink VJ, Luijendijk RW, de Wilt JH, Heide R, Jeekel J (1993) An evaluation of risk factors in incisional hernia recurrence. Surg Gynecol Obstet 176: 228–234
24. Sugerman HJ, Kellum JM Jr, Reines HD, DeMaria EJ, Newsome HH, Lowry JW (1996) Greater risk of incisional hernia with morbidly obese than steroid-dependent patients and low recurrence with prefascial polypropylene mesh. Am J Surg 171: 80–84
25. Christou NV, Jarand J, Sylvestre JL, McLean AP (2004) Analysis of the incidence and risk factors for wound infections in open bariatric surgery. Obes Surg 14: 16–22
26. Wantz G (1991) Atlas of Hernia Surgery. Raven Press, New York
27. Burger JW, Luijendijk RW, Hop WC, Halm JA, Verdaasdonk EG, Jeekel, J (2004) Long-term follow-up of a randomized controlled trial of suture versus mesh repair of incisional hernia. Ann Surg 240: 578 583; discussion 583–575
28. Petersen S, Henke G, Freitag M, Faulhaber A, Ludwig K (2001) Deep prosthesis infection in incisional hernia repair: predictive factors and clinical outcome. Eur J Surg 167: 453–457
29. Sorensen LT, Hemmingsen UB, Kirkeby LT, Kallehave F, Jorgensen LN (2005) Smoking is a risk factor for incisional hernia. Arch Surg 140: 119–123
30. Myles PS, Iacono GA, Hunt JO, Fletcher H, Morris J, McIlroy D, Fritschi L (2002) Risk of respiratory complications and wound infection in patients undergoing ambulatory surgery: smokers versus nonsmokers. Anesthesiology 97: 842–847
31. Sorensen LT, Karlsmark T, Gottrup F (2003) Abstinence from smoking reduces incisional wound infection: a randomized controlled trial. Ann Surg 238: 1–5
32. Moller AM, Villebro N, Pedersen T, Tonnesen H (2002) Effect of preoperative smoking intervention on postoperative complications: a randomised clinical trial. Lancet 359: 114–117
33. Yahchouchy-Chouillard E, Aura T, Picone O, Etienne JC, Fingerhut A (2003) Incisional hernias. I. Related risk factors. Dig Surg 20: 3–9

Discussion

Schumpelick: *Dr Chan, is a 1-year waiting period today still justified in our fast-living society? 1 year with an incisional hernia can be a long period.*

Chan: *If you talk to the patient they can carry on their life very easily, with some abdominal impairment, if they have a really big hernia.*

Schumpelick: *But if he wants a repair within a quarter of a year, if he is a sportsman who wants the repair now?*

Chan: *That's why I say, there is no clinical trail, it's a feeling but not rational. Like the last case I had, a patient after a perforated appendicitis. When I go in, even after 1 year, it's like concrete.*

Itany: *We have a real problem with obesity in the United States, and when I looked at it last year in our hospital about 80% of our patients undergoing abdominal surgery had a BMI above 30, and 40% had a BMI above 35%. Most of our patients are above the age of 60. Do you have any advice for these patients or for us surgeons that are operating on these incisional hernias, and how to address the problem of obesity?*

Chan: *I think it's a question of motivation. They usually come to see us and say: Doctor, you are our last resort. So we can tell them: you do what I say and we fix your hernia! You need motivated patients.*

20 How to Create a Recurrence After Incisional Hernia Repair

20.1 How to Create a Recurrence After Incisional Hernia Repair as an Expert of Suture Repair

M.P. Simons

Should Suture Repair be Considered at All?

In a Pubmed search using the terms incisional hernia limited to clinical trials, 122 hits were found. There were two randomized trials. The best-known is the Luijendijk study with after long-term follow-up a significantly lower recurrence rate after mesh sublay repair when compared to suture repair (Table 20.1) [1].

In a randomized clinical trial by Korenkov, results after a follow-up of 13 months in 100 patients with non-complex incisional hernia (< 10 cm) showed the following recurrence rates: suture repair 4/33, polypropylene onlay mesh 3/39 and autodermal graft 4/28 [2]. The differences were not significant. Mesh repair showed (marginally significantly) more infectious complications than suture repair. In 4 non-randomized studies recurrence rates for suture repair varied from 25–63% (Table 20.2) [3].

Flum et al. indicated in a statewide population-based study that the hazard for recurrence after suture repair was 24%; on the other hand, introducing new mesh-based repairs had not shown a decrease in the yearly number of incisional repairs performed [4].

As shown above, suture repair should not be first choice for treatment of incisional hernia.

Table 20.1. Long-term recurrence rate Luijendijk study

	3 years	10 years
Suture	43%	63%
Mesh	24%	32%
	(p = 0,02)	(p < 0,001)

Table 20.2. Non-randomised trials suture versus mesh repair for incisional hernia

	Suture [%]	Mesh [%]
Liakakos	25	8
Schumpelick	33	13
Koller	63	13
Clark	36	23

What Suture Repair Techniques Are There?

Many suture techniques have been described, but none has proven to be superior to the other in well-performed clinical trials. Documented suture repair techniques are, for example: Keen, Nattal, fascial, mass nylon, layered steel wire, rectus-relieving incision, sliding door, Cardiff and Mayo [3]. The recurrence rates varied from 0–49%, depending on technique and methods of follow-up. Ramirez described the component separation technique with good personal results [5]. In a randomized trial (submitted for publication), results of Ramirez could not be reproduced, with a high percentage of recurrences after 2 years. Often relaxing incisions in the rectus are advised to reduce tension on sutures. Many devices have been used and discarded, e.g. the Ton device, subcutaneous rods etc. There is little evidence to indicate what results one can expect. The studies are heterogenous, there is no consistent classification system and thus the level of evidence does not exceed 3 (retrospective case control studies).

On a theoretical basis an incisional hernia has an increased risk of occurring after a number of factors that are difficult, if not impossible, to influence.

Patient- and Hernia-Related Risk Factors for Incisional Hernia

- Gender
- Age
- Intra-abdominal pressure
  - Obesity
  - COPD
- Impaired wound healing
  - DM
  - Corticosteroids
  - Malnutrition
  - Smoking
  - Oncologic disease (chemo/RT)
  - Obstructive jaundice
- Collagen disease
- Previous repairs
- Previous wound dehiscence
- Previous laparotomies
- Hernia size
- Number of hernias
- The hernia-free interval

These factors, usually predominant in patients with incisional hernia, preclude the goal (and possibility) of approximating healthy tissue in a tension-free manner.

Suture Repair or Mesh Repair?

It is the opinion of many experts that non-mesh repairs are indicated only when the operation is performed under septic circumstances or when mesh is not available. Small incisional hernia (< 2–4 cm) is a relative indication for mesh repair. There is no level-1 evidence whether these need mesh repair.

It seems obvious to follow the principles of closure of a laparotomy wound when performing suture repair of an incisional hernia. As described in other chapters and in the meta-analysis of van t'Riet, probably the best technique is tension-free running suture of slowly absorbing or non-absorbable type [6]. Where possible, tension on sutures and tissues must be avoided by using relaxing incisions or the principles of component separation technique as described by Ramirez.

How to Create a Recurrence After Incisional Hernia Repair as an Expert of Suture Repair?

The short answer is: by using it. The longer answer: close the defect with knotted catgut stitches under as much tension as possible.

References

1. Burger JWA, Luijendijk RW, Hop WCJ, Halim JA, Verdaasdonk EGG, Jeekel J. Long-term follow-up of a randomized controlled trial of suture versus mesh repair of incisional hernia. Ann Surg 2004; 240(4): 578–585
2. Korenkov M, Sauerland S, Arndt M, Bograd L, Neugebauer EAM, Troidl H. Randomized clinical trial of suture repair, polypropylene mesh or autodermal hernioplasty for incisional hernia. Br J Surg 2002; 89: 50–56
3. Cassar K, Munro A. Surgical treatment of incisional hernia. Br J Surg 2002; 89: 534–545
4. Flum DR, Horvath K, Koepsell T. Have outcomes of incisional repair improved with time? A population based analysis. Ann Surg 2003; 237(1): 129–135
5. Ramirez OM, Ruas E, Dellon AL. "Components separation" method for closure of abdominal wall defects: an anatomic and clinical study. Plast Reconstr Surg 1990; 86: 519–526
6. Van t Riet M, Steyerberg EW, Nellensteyn J, Bonjer HJ, Jeekel J. Meta-analysis of techniques for closure of midline abdominal incisions. Br J Surg 2002; 89: 1350–1356

Discussion

Schumpelick: *How do you handle small trocar hernias? Mesh or suture?*

Simons: *I think there is a place for randomizing the small hernias, suture vs. mesh. We are going to do a trial with Rotterdam on umbilical hernia looking for what do you do in 2-cm hernias or 1-cm hernias. I don't know whether you have to use a mesh in that case. There is only little evidence, and we should randomize these patients.*
Halm: *In our study we advised abandoning suture repair. Now you say that when you have to do a suture repair you have to do it in the following way. Maybe you should go one step further and say never do suture repair, and follow the patients until they have serious problems. Is there any indication for doing suture repair in the first place? It gives so many problems; one should never do it anyway.*
Simons: *Are you talking about the non-operative treatment?*
Halm: *Yes, perhaps the non-operative treatment is a far better choice than the suture repair.*
Simons: *I think in asymptomatic patients there is a lot of room for non-operative treatment. Don't operate on people that don't complain, and in very large hernias I send them home also, because the risks don't outweigh the benefit.*
Simons: *Covering the mesh or trying to close the abdominal wall over the mesh vs. leaving the defect as it was or only approximating it. When you leave a defect, do you suture the borders of the fascia to the mesh or do you just stick to stitches that you have at the bilateral sides?*
Flament: *In my opinion, closure of the tissue in front of the mesh is only to prevent contact between the skin and the mesh. Sometimes, if we want to close the muscles, we use some relaxing incisions, but not very often. We use anything we can, e.g. a small amount of the peritoneal sac, but we never stitch the limits of the abdominal wall to the prosthesis.*
Simons: *In what percentage would you estimate that you leave a defect after the Rives-Stoppa-Flament repair?*
Flament: *If we give enough tension on the prosthesis, we usually close the fascia in all cases.*
Kingsnorth: *The Rives technique in the hand of experts produces extremely good results. There are no national surveys; we don't really know what proportion of general surgeons uses this technique. But it is my impression that most general surgeons will choose the onlay technique because it is simpler. Do you think we should have a randomized trial concerning sublay vs. onlay. We have never had one; the two techniques have been around for 30 years, but a randomized trial has never been done? Why?*
Flament: *I don't know. Maybe everybody believes that his technique is the best and has good results. If you promote a prospective trial on the two techniques I will never see the results.*
Kingsnorth: *All we can say is that it produces good results in the hand of experts and we can say nothing more than that. We don't know whether it produces good results in the hand of ordinary general surgeons.*
Flament: *The only objection we have with the Chevrel procedure is the need for big skin flaps, sometimes with necrosis. Chevrel saw a lot of seromas before he glued the prosthesis.*
Kingsnorth: *Do you think a recommendation of this meeting would be to encourage the industry to support a trial of sublay vs. onlay?*
Flament: *Maybe.*
Fitzgibbons: *I just would like to make a point: you showed that the Reverdins needle goes through the skin. Do you routinely do this or do you ever bring it out in the subcutaneous tissue?*
Flament: *As someone said, usually we have fatty patients. The needle with the stitches is not long enough when you have 10 cm of fat below the skin, so to go through the skin you have to use a long needle. As I have shown in other communications, the laparoscopist use the Gor needle which looks exactly like the Reverdin needle to pass transfixing stitches in laparoscopic procedures.*

20.2 Open Onlay Mesh Reconstruction for Incisional Hernia

T.S. de Vries Reilingh, O.R. Buyne, R.P. Bleichrodt

Introduction

Nowadays, prosthetic repair is the standard technique to repair incisional hernias. Basically there are three methods for implantation of prosthetic meshes when used for reconstruction of abdominal wall defects: inlay, onlay or sublay. The choice of each method is predominantly based on the surgeon's preference. For a proper reconstruction the prosthetic mesh must have a sufficient overlap with the fascia. The onlay and sublay techniques both provide a proper overlap between the mesh and the fascia, whereas the inlay technique does not provide enough contact be-

tween the myoaponeurotic fascia and the mesh to guarantee proper anchorage. Therefore the latter technique must be abandoned [1].

The onlay technique is simple, no extensive adhesiolysis is needed, and fixation of the mesh is easy and can be an attractive alternative to the more difficult sublay technique.

Operative Technique

The skin and subcutaneous fat are dissected free from the hernia sac and the anterior fascia, far laterally. The hernia is reduced and the fascia is closed primarily, if possible. When primary closure is not possible, the peritoneum covering the bowels or the greater omentum is used as an interface between the intra-abdominal viscera and the mesh. Subsequently, a prosthetic mesh is positioned on the ventral fascia, with an overlap of at least 5 cm between the fascia and the mesh. The prosthetic mesh is fixed to the fascia with non-resorbable sutures or staples. The prosthetic mesh must be firmly fixed to the fascial edges to prevent herniation between the ventral fascia and the mesh [1]. Scarpa´s fascia and skin are closed over the prosthetic mesh. (◘ Figure 20.1a,b) If no full thickness skin is available the greater omentum or a composite myocutaneous flap should is used to cover the prosthetic mesh [2].

Patients and Methods

From 1996 to 2000, 17 patients (9 women and 8 men) with a ventral hernia were operated using the onlay technique using polypropylene mesh. All patients received standard thrombo-embolic and antibiotic prophylaxis.

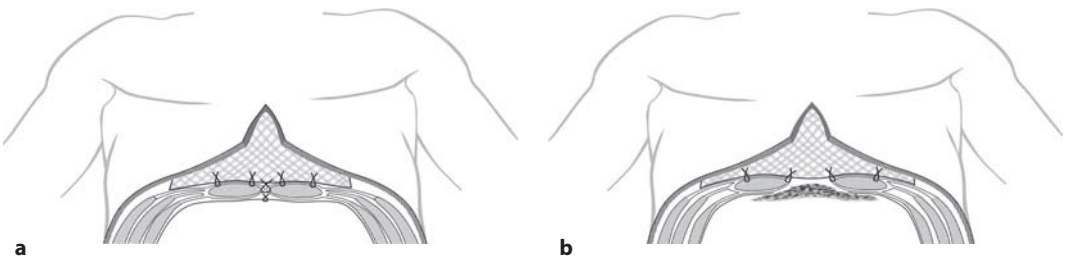
The records of the patients were reviewed. The following data were extracted from the medical record: size and cause of the hernia, pre- and postoperative mortality and morbidity, with special attention to wound complications. All patients were invited to come to the outpatient clinic for physical examination of the abdominal wall, at least 1 year after operation.

Results

Reconstruction was performed under clean conditions in all patients. The cause of the hernia was open treatment of generalized peritonitis in four patients and a recurrent hernia in two patients. In four patients the abdominal wall was closed primarily, covered with an onlay polypropylene mesh. In 9 patients the fascial gap was bridged with an onlay polypropylene mesh. In all patients, the mesh was fixed to the fascia with iron staples.

The postoperative course was uneventful in four patients. Wound complications occurred in 13 patients: one patient had a wound infection, two patients had skin necrosis and 12 patients had a seroma. In one of these 12 patients the seroma became infected after puncture, another patient developed skin necrosis secondary to seroma.

Two patients died within 1 year after the operation, not related to the hernia operation. Fifteen patients were seen in the outpatient clinic after a median follow-up of 18.5 months (range 12–28 months). Three patients had a recurrent hernia (20%), five patients complained about a rigid abdominal wall.



◘ **Fig. 20.1a,b.** Reconstruction of an incisional hernia using the onlay reconstruction. **a** The rectus abdominis muscle is approximated in the midline. The polypropylene mesh should be fixed to the fascia with an overlap of at least 5 cm in all directions and with a double row of non-resorbable sutures. **b** The fascia cannot be approximated under the mesh. Omentum is placed between mesh and bowels. The inner row of sutures should be positioned from the fascial edges. If this inner row of sutures is placed away of the fascial edge, the intra-abdominal pressure might push the mesh away from the fascia and a recurrence can easily to occur

Discussion

Abdominal wall hernia reconstruction using an onlay polypropylene mesh seems the most straightforward method, but is associated with serious postoperative complications.

The prosthetic mesh can be used in two ways. First, as a support when the fascia can be closed primarily. Then the mesh can be positioned either as an onlay or a sublay, because the biomechanical circumstances are similar. Still, the sublay technique is preferred since wound complications such as seroma formation and infection are rather frequent. Using the sublay technique, the retromuscular position will prevent the exposure of the prosthesis if wound complications occur. Second, prosthesis can be used to bridge fascial defects if the fascia cannot be closed primarily [1, 3–5]. Under these circumstances, the sublay technique, where the intra-abdominal pressure (0.2–2.0 kPa) presses the prosthesis against the ventral abdominal wall, is preferred as well. If properly fixed, the forces on the mesh are counteracted by the abdominal wall, thus preventing reherniation [6]. The sutures in concert with the fibro-collagenous tissue that surrounds the prosthetic mesh will counteract the small sheering forces on the prosthesis (Fig. 20.2).

When using the onlay technique, the intra-abdominal pressure is not counteracted and the much larger forces will put a continuous stress on the fixating sutures and the fibro-collagenous tissue, with the risk of tearing the prosthesis from the fascia (Fig. 20.3). Although the sublay mesh reconstruction is superior, the onlay mesh reconstruction might be helpful in selected patients, for example, to prevent contact between the prosthesis and the bowel and when the sublay technique is not possible for technical reasons.

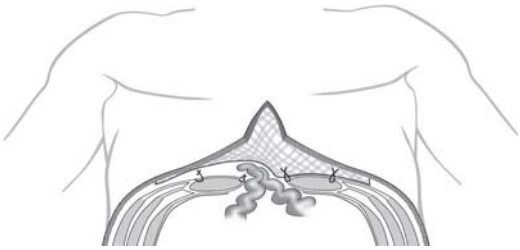


Fig. 20.2. Due to the intra-abdominal pressure, a reherniation occurred

In the literature, ten series report the results of onlay mesh reconstruction [7–16] (Table 20.3). All but one of the series are retrospective case series. The number of patients included varies from 9–70. The series have a wide range of follow-up and the method of follow-up was mentioned in none of the studies. The reherniation rate varied between 0 and 13%. The reherniation rate in our series was 20%, but it is the only series where all patients were seen in the outpatients' clinic after an adequate follow-up period. The results are similar to other series with adequate follow-up [4].

Several prosthetic materials can be used to repair incisional hernias. Expanded-polytetrafluoroethylene (ePTFE) patch and polypropylene mesh (PPM)-based prosthesis are the most frequently used prosthetic materials. PPM is the preferred prosthetics material when the onlay technique is used. First, because the anchorage of the prosthesis to the adjacent fascia is superior to the ePTFE patch. Fixation of the ePTFE patch depends solely on the fixating sutures, because the micropores (20 μm) in ePTFE patch are too small to allow ingrowth of fibro-collagenous tissue [17, 18]. PPM is completely

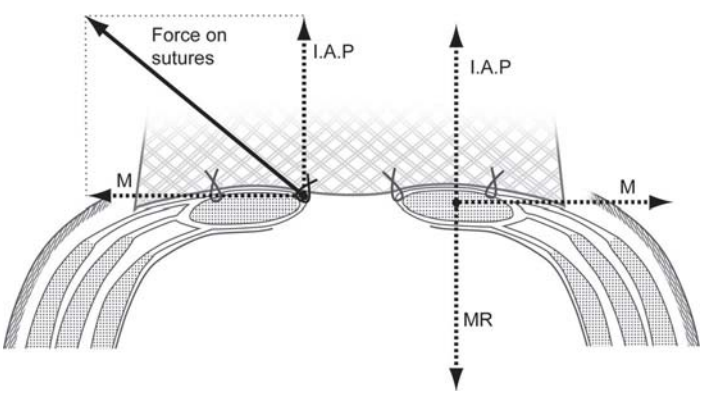


Fig. 20.3. In an intact abdominal wall the intra-abdominal pressure (*I.A.P.*) is compensated by the muscle strain (MR). In the midline of the abdominal wall there always a muscle strain to the lateral border caused by the oblique abdominal muscles and compensated by the opposite site, there is a balance. The intra-abdominal pressure (I.A.P.) on the inner row of sutures of an onlay reconstruction is not compensated by muscle strain (*MR*), but the muscle still gives a constant strain to the lateral border (*M*). This result is a constant force on the sutures (*in black*)

Table 20.3. Onlay technique

Author	Year	Patients	Complications n (%)	Reherniation n (%)	Follow-up mean (range) months
Larson	1978	9	0	0	? (12–60)
Deitel	1979	36	4 (11%)	2 (6%)	42 (?)
Lewis	1984	50	5 (10%)	3 (6%)	30 (?)
Wagman	1985	9	0	0	14 (?)
Molloy	1991	50	10 (20%)	4 (8%)	45 (6–120)
Liakakos	1994	49	4 (8%)	4 (8%)	? (0–16)
Birolini	2000	20	5 (25%)	0	? (12–84)
Korenkov	2002	70	14 (20%)	6 (9%)	14 (11–24)
De Vries Reilingh	2004	17	13 (76%)	3 (20%)	18.5 (12–28)
Machairas	2004	43	9 (21%)	4 (9%)	54.4 (4–106)
Kingsnorth	2004	16	5 (31%)	2 (13%)	? (6–60)

incorporated into fibro-collagenous tissue and firmly anchors to the adjacent fascia. Second, because PPM is rather resistant against infection, whereas infected ePTFE patches have to be removed. Since wound infections occur in 17–50% of patients, the use of ePTFE patch to repair incisional hernias by the onlay technique is too risky [19–21]. Korenkov et al. performed a randomized clinical trial comparing onlay polypropylene mesh repair with suture repair and onlay dermal graft repair [16]. This trial is the only randomized clinical trial comparing onlay reconstruction with two different biomaterials. Wound complications occurred in 20%. Although none of the meshes had to be removed because of infection, the trial was stopped because of the high complication rate.

In our series, 76% of patients suffered from seroma after the operation, compared to 0–31% in other series (Table 20.3). Seromas are a consequence of the large subcutaneous wound surface that is created to fix the prosthetic mesh with an adequate overlap to the fascia. Seromas are a frequent complication after reconstruction of large abdominal wall hernias occurring in up to 30% [19, 22]. Moreover, wound infections are frequent. In our series, 24% of patients suffered a wound infection, which is similar to the frequency found in other series [14, 16]. Wound infection may also occur secondary to skin necrosis. Separation of the epigastric perforating arteries endangers the vascular supply of the skin, which may interfere with wound healing and may result in skin necrosis and subsequent infection.

In conclusion, onlay prosthetic repair of abdominal wall hernias is easy but, because of the increased chance of reherniation and loss of the prosthesis in the case of wound complications, the use of onlay prosthetic repair must be discouraged and be performed only when the superior sublay repair is not possible.

Acknowledgements. The authors wish to thank Mr. F. Bosch (Tilburg, The Netherlands), medical illustrator, for making the illustrations.

References

1. de Vries Reilingh TS, van Geldere D, Langenhorst B, de Jong D, van der Wilt GJ, van Goor H et al. Repair of large midline incisional hernias with polypropylene mesh: comparison of three operative techniques. Hernia 2004; 8(1): 56–59

2. Bleichrodt RP, Malyar AW, de Vries Reilingh TS, Buyne OR, Bonenkamp JJ, van Goor H. The omentum-polypropylene sandwich technique: an attractive method to repair large abdominal wall defects in the presence of contamination or infection. Hernia 2007; 11(1): 71–74
3. Burger JW, Luijendijk RW, Hop WC, Halm JA, Verdaasdonk EG, Jeekel J. Long-term Follow-up of a Randomized Controlled Trial of Suture Versus Mesh Repair of Incisional Hernia. Ann Surg 2004; 240(4): 578–585
4. Luijendijk RW, Hop WC, van den Tol MP, de Lange DC, Braaksma MM, IJzermans JN et al. A comparison of suture repair with mesh repair for incisional hernia. N Engl J Med 2000; 343(6): 392–398
5. Stoppa RE. The treatment of complicated groin and incisional hernias. World J Surg 1989; 13(5): 545–554
6. Klinge U, Klosterhalfen B, Conze J, Limberg W, Obolenski B, Ottinger AP et al. Modified mesh for hernia repair that is adapted to the physiology of the abdominal wall. Eur J Surg 1998; 164(12): 951–960
7. Birolini C, Utiyama EM, Rodrigues AJJ, Birolini D. Elective colonic operation and prosthetic repair of incisional hernia: does contamination contraindicate abdominal wall prosthesis use? J Am Coll Surg 2000 191(4): 366–372
8. Deitel M, Vasic V. A secure method of repair of large ventral hernias with Marlex mesh to eliminate tension. Am J Surg 1979; 137(2): 276–277
9. Larson GM, Harrower HW. Plastic mesh repair of incisional hernias. Am J Surg 1978; 135(4): 559–563
10. Liakakos T, Karanikas I, Panagiotidis H, Dendrinos S. Use of Marlex mesh in the repair of recurrent incisional hernia. Br J Surg 1994; 81(2): 248–249
11. Molloy RG, Moran KT, Waldron RP, Brady MP, Kirwan WO. Massive incisional hernia: abdominal wall replacement with Marlex mesh. Br J Surg 1991; 78(2): 242–244
12. Wagman LD, Barnhart GR, Sugerman HJ. Recurrent midline hernial repair. Surg Gynecol Obstet 1985; 161(2): 181–182
13. Kingsnorth AN, Sivarajasingham N, Wong S, Butler M. Open mesh repair of incisional hernias with significant loss of domain. Ann R Coll Surg Engl 2004; 86(5): 363–366
14. Machairas A, Misiakos EP, Liakakos T, Karatzas G. Incisional hernioplasty with extraperitoneal onlay polyester mesh. Am Surg 2004; 70(8): 726–729
15. Lewis RT. Knitted polypropylene (Marlex) mesh in the repair of incisional hernias. Can J Surg 1984; 27(2): 155–157
16. Korenkov M, Sauerland S, Arndt M, Bograd L, Neugebauer EA, Troidl H. Randomized clinical trial of suture repair, polypropylene mesh or autodermal hernioplasty for incisional hernia. Br J Surg 2002; 89(1): 50–56
17. de Vries Reilingh TS, Malyar AW, Walboomers XF et al. Impregnation of e-PTFE abdominal wall patches with silver salts and chlorhexidine diminishes biocompability and is associated with an increased reherniation rate (submitted)
18. van der Lei B, Bleichrodt RP, Simmermacher RK, van Schilfgaarde R. Expanded polytetrafluoroethylene patch for the repair of large abdominal wall defects. Br J Surg 1989; 76(8): 803–805
19. de Vries Reilingh TS, van Goor H, Charbon J et al. Repair of large midline abdominal wall hernias: Components Separation Technique versus Prosthetic Repair. Interim analysis of a randomised controlled trial. World J Surg 2007; 31: 756–763
20. Lowe JB, Garza JR, Bowman JL, Rohrich RJ, Strodel WE. Endoscopically assisted "components separation" for closure of abdominal wall defects. Plast Reconstr Surg 2000; 105(2): 720–729; quiz 730
21. de Vries Reilingh TS, van Goor H, Rosman C, Bemelmans MH, de Jong D, van Nieuwenhoven EJ et al. "Components separation technique" for the repair of large abdominal wall hernias. J Am Coll Surg 2003; 196(1): 32–37
22. Conze J, Kingsnorth AN, Flament JB, Simmermacher R, Arlt G, Langer C et al. Randomized clinical trial comparing lightweight composite mesh with polyester or polypropylene mesh for incisional hernia repair. Br J Surg 2005; 92(12): 1488–1493

Discussion

Flament: *I am surprised that no one has mentioned relaxing incisions today, because with them a suture repair may be achieved in cases where non-absorbable meshes are not suitable, e.g. in infected cases. Main part of the onlay repair by Chevrel was a relaxing incision of the anterior sheath of the rectus muscle and a prosthesis covering, reinforcing and recreating the anterior rectus sheath. That is a little different from what you have shown compared to the 400 cases of Chevrel published in Hernia.*

deVries Reilingh: *There is a randomized clinical trial including patients for Ramirez technique with and without mesh reinforcement, and the mesh is placed in the sublay position, not onlay. We choose this technique because of the large wound complication described by onlay mesh plasty and also with the Ramirez technique, and it seems not suitable to put a mesh in areas where they might cause problems.*

Kurzer: *I was interested, but not surprised, to see your high rate of wound complication and abdominal wall stiffness. I am interested that Prof. Flament and his colleagues have a vast experience with sublay mesh and have shown over many years that it works very well. Prof. Kingsnorth, with respect, is advocating a randomized trial of a bad operation against a good operation done badly, and I can't see the point in doing that. Do a good operation well. We should be teaching the people to do the good operation, not doing more randomized clinical trials of two very different operations, one of which doesn't work well at all. I am pleased that you are moving over to sublay mesh.*

Chan: *In my study and review we have taken a lot of onlay mesh, that's all I can tell you, especially for big ones. It just doesn't work, because most of the time the defect is just so big, its too tight to put it in, so it just won't work, I would recommend not to use it at all.*

Kingsnorth: *I would like to speak up in favour of the onlay technique. Firstly, we must not ignore the results of Prof. Chevrel, that are every bit as good as the sub-*

lay technique; we cannot call the onlay a bad operation. Secondly, I think it is very versatile; the best place for the sublay technique is only in the upper abdomen because you can then put it in front of the posterior rectus sheath; once you get below the linea arcuata, you then only have peritoneum, that often tears and then you have mesh in direct contact with bowel, so I think in the lower abdomen the onlay technique maybe advantageous. We must give the onlay technique a chance, it is more versatile, it is easier, and general surgeons are capable of using it under more circumstances than the sublay technique.

Schumpelick: *I would also like to say something in favour of the onlay technique, even as a sublay man. In the recurrent cases, where the retromuscular space is already obliterated by a mesh, it is sometimes very difficult to place another mesh in the same space. With the new meshes you can do an onlay repair. The main problem with the old meshes in the onlay position was infection, something we don't see with the new large pore meshes that are better integrated. And even in the case of infection there is no need for explantation. We have done some in this technique with good results.*

20.3 Technical Factors Predisposing to Recurrence After Minimally Invasive Incisional Herniorrhaphy

C.T. Frantzides, L.E. Laguna, M.A. Carlson

Introduction

Since 1993, experience in minimally invasive incisional hernia repair has accumulated such that we now have some basic understanding of how to optimize the technical outcome of this procedure. In this review we will summarize technical maneuvers which we believe will minimize the risk of recurrence after minimally invasive incisional herniorrhaphy. The conclusions and recommendations of this review are based on our own clinical experience [1] and a review of the surgical literature. As is the case in most areas of surgery, the recommendations given in this review are based on uncontrolled clinical series and expert opinion; there are little to no data available from randomized controlled trials in the field of minimally invasive incisional hernia surgery.

Methods

An internet search of the literature was performed (PubMed/National Library of Medicine, www.ncbi.nlm.nih.gov/entrez/) using various combinations of the following keywords: minimally invasive, laparoscopic, ventral, incisional, hernia. The inclusion criteria were papers that contained adequate data on > 10 patients undergoing minimally invasive incisional or ventral herniorrhaphy. To be included, a paper needed to describe patient demographics, surgical technique, perioperative events, and some follow-up/recurrence data. In addition to internet search, the references of selected papers were searched manually to identify any possible manuscripts that were missed (none were found with this secondary search). In some instances, a group of authors had multiple publications on the same series of patients; in these cases only the most recent update of a given patient series was included in the present review.

Results for Hernia Recurrence

A total of 53 manuscripts met the inclusion criteria (◘ Table 20.4); these papers described 5227 minimally invasive incisional or ventral herniorrhaphies (a comprehensive analysis will be submitted for later publication.) Certain aspects of herniorrhaphy technique were virtually identical among all 53 manuscripts: intraperitoneal sublay of prosthetic mesh which extended beyond the margins of hernia in all directions, with no excision of the hernia sac. The papers differed in the type of mesh used, the amount of mesh overlap of the defect, and in the technique of mesh fixation (see discussion below). The rate of hernia recurrence in these 5227 published procedures was 3.98%. Of course, this result is mostly the product of specialty centers in which minimally invasive surgery is prominent, so the recurrence rate for all operators is likely to be higher. The results from the 53 manuscripts of this review also is subject to publication bias (i.e., better results have a greater likelihood of being submitted than mediocre results). The reported recurrence rate from open in-

Table 20.4. Papers included in review of minimally invasive incisional/ventral hernia surgery

Ref. no.	Year	Authors	Institution	Procedures
[7]	1997	Holzman et al.	Duke	21
[8]	1998	Toy et al.	Multicenter	144
[9]	1998	Tsimoyiannis et al.	Hatzikosta General Hospital, Ioannina	11
[10]	1999	Koehler et al.	Martha's Vineyard Hospital	32
[11]	1999	Kyzer et al.	Tel Aviv Univ	53
[12]	1999	Sanders et al.	Tulane Univ, Henry Ford Hospital	12
[13]	2000	Chari et al.	Meridia Huron Hospital, Cleveland	14
[14]	2000	Chowbey et al.	Sir Ganga Ram Hospital, New Delhi	202
[15]	2000	DeMaria et al.	MCV, Richmond	21
[16]	2000	Farrakha	Abu Dhabi, UAE	18
[17]	2000	Reitter et al.	UI Peoria, IL	49
[18]	2000	Szymanski et al.	Scarborough Hospital, Canada	44
[19]	2001	Birgisson, Park et al.	UKY	64
[20]	2002	Andreoni et al.	UNC Chapel Hill	13
[21]	2002	Aura et al.	Aulnay-Sous-Bois, France	86
[22]	2002	Bageacu et al.	Saint-Etienne, France	159
[23]	2002	Ben-Haim et al.	Tel Aviv Univ	100
[24]	2002	Berger et al.	Baden-Baden	150
[25]	2002	Gillian et al.	Southern Maryland Hospital	100
[26]	2002	Kirshtein et al.	Ben Gurion Univ, Beer Sheva, Israel	103
[27]	2002	Kua et al.	Royal Brisbane Hospital, Queensland, Austral	30
[28]	2002	Lau et al.	Univ Hong Kong Med Ctr	11
[29]	2002	Parker et al.	Univ South Carolina	50
[30]	2002	Raftopoulos et al.	UI Chicago	50
[31]	2002	Salameh et al.	Baylor, Houston TX	29
[32]	2002	van't Riet et al.	Erasmus U Med Ctr, Rotterdam	25

Table 20.4. *Continued*

Ref. no.	Year	Authors	Institution	Procedures
[33]	2002	Wright et al.	Hennepin County Med Ctr, Minneapolis	90
[34]	2003	Carbajo et al.	Valladolid, Spain	270
[35]	2003	Chelala et al.	Univ Hosp Tivoli, Belgium	120
[36]	2003	Chowbey et al.	Sir Ganga Ram Hospital, New Delhi	34
[37]	2003	Eid et al.	UPitt, VAMC Pitt, UMN	79
[38]	2003	Heniford et al.	Carolinas Medical Center, UKY, Emory, UTN	850
[39]	2003	LeBlanc et al.	Min Invas Surg Inst, Baton Rouge	200
[40]	2003	McGreevy et al.	Dartmouth-Hitchcock Med Ctr, VAMC VT	65
[41]	2003	Mizrah et al.	Ben Gurion Univ, Beer Sheva, Israel	231
[42]	2003	Rosen et al.	Cleveland Clinic	114
[43]	2004	Bamehriz and Birch	McMaster Univ, Hamilton, Can	28
[44]	2004	Bencini and Sanchez	Florence, Italy	64
[45]	2004	Bower et al.	East Carolina Univ, Greenville	100
[46]	2004	Franklin et al.	Texas Endosurgery Institute, MGH, Monterrey	384
[1]	2004	Frantzides et al.	NWU, UNMC, UTN	208
[47]	2004	Gal et al.	Bugat Pal Hosp, Hungary	15
[48]	2004	Kannan et al.	Changi General Hosp, Singapore	20
[49]	2004	McKinlay and Park	Univ Maryland	170
[50]	2004	Moreno-Egea et al.	Murcia, Spain	90
[51]	2004	Muysoms et al.	Ghent, Belgium	52
[52]	2004	Sanchez et al.	Florence	90
[53]	2004	Ujiki et al.	NWU, UHawaii, Hines VA	100
[54]	2004	Verbo et al.	Catholic Univ, Rome Italy	45
[55]	2005	Angele et al.	Ludwig-Maximilians Univ, Munich	28
[56]	2005	Johna	Loma Linda Univ, CA	18
[57]	2005	Olmi et al.	Monza, Italy	50
[58]	2005	Perrone et al.	Washington Univ	121

cisional herniorrhaphy (not reviewed here) is widely variable, from several percent to 20% or more. Needless to say, a prospective randomized comparison of open vs. minimally invasive incisional hernia repair has not been done. Considering the inherent advantages of minimally invasive surgery, however, it would be reasonable to predict that the overall results (including recurrence, infection, pain, patient satisfaction, etc.) of the minimally invasive approach would be as least as good, if not better, than the open approach.

Technical Factors: Entry and Exposure

For any laparoscopic procedure, the surgeon can minimize the risk of port-site hematoma by transilluminating the abdominal wall prior to trocar insertion. This maneuver minimizes the risk of abdominal wall vessel laceration. It is not clear, however, whether a port site hematoma predisposes a patient to recurrent hernia. In order to prevent port-site hernia, the surgeon should close all port sites for trocars > 5 mm, and for 5mm if the site has become stretched or enlarged [2].

Probably the first major technical issue that the surgeon encounters during a minimally invasive incisional hernia is intra-abdominal exposure. Retrospective analysis has determined, not surprisingly, that inadequate dissection of the hernial defects will increase the risk of hernia recurrence [3]. Nearly all authors of the 53 manuscripts of the present review stress complete exposure of the ventral abdominal wall with takedown of all adhesions to the viscera. The entire incision needs to be visualized. Such a maneuver will prevent the surgeon from missing a small, asymptomatic defect which later could enlarge into a symptomatic one. This is especially important with long midline incisions closed with running nonabsorbable suture, in which the so-called Swiss cheese abdomen (i.e., multiple small hernias deriving from the cutting action of the suture) can develop. Small hernias can be hidden in a mass of dense adhesions, so complete adhesiolysis is essential.

Technical Factors: Mesh Type

The next choice of potential consequence during minimally invasive incisional hernia repair is the mesh type. Expanded Polytetrafluoroethylene (ePTFE) was the prosthetic material used in the majority of procedures in 41 (77%) of the 53 manuscripts; of these 41 papers, 33 (62%) specified their ePTFE as the dual-surface construct available from W. L. Gore and Associates, Inc. (i.e., DualMesh). This mesh has a closed structure surface on the side facing the viscera; this is intended to reduce tissue attachment. The other side (facing the abdominal wall) has a macroporous structure (corduroy), which is intended to enhance tissue attachment. Interestingly, an improvised dual-surface mesh for minimally invasive incisional herniorrhaphy already was in use by the early 1990s [4]. This was a bilaminar prosthesis consisting of a sheet of ePTFE and a sheet of polypropylene sewn together; the polypropylene side was applied to the abdominal wall while the ePTFE side contacted the viscera. This dual-surface arrangement encouraged tissue ingrowth on the abdominal wall side, thereby increasing the robustness of the repair, yet minimized intestinal reaction to the mesh. So far, published clinical experience with the dual-surface mesh configuration has shown it to be safe. To our knowledge, there have been no published cases of primary erosion of ePTFE into the viscera after incisional herniorrhaphy with ePTFE. In laparoscopic incisional hernia repair the prosthesis is typically placed in direct contact with the viscera which, in the case of heavy-weight polypropylene mesh, introduces the risk of visceral erosion. The dual-surface mesh configuration appears not to have this risk.

The use of ePTFE has undergone a resurgence with the advent of minimally invasive incisional hernia repair. This material was less popular in open hernia repair because it was more prone to infection and incorporated less well than other materials (e.g., polypropylene). Since mesh infection appears to be less of a problem with the minimally invasive approach, and with the introduction of the dual-surface product which incorporates strongly into the abdominal wall yet is benign to the viscera, dual-surface ePTFE has become the material of choice for the majority of the authors in this review. It should be noted, however, that there are a number of light-weight/composite polypropylene hernia meshes now available which may be suitable (or even better) alternatives to ePTFE. Long-term comparative data in patients are not available.

Technical Factors: Mesh Overlap

As indicated above, the universal approach to minimally invasive repair of hernia of the ventral abdominal wall in manuscripts of this review is sublay positioning of prosthetic mesh, a technique originally described in open surgery by Rives and Flament [5] and also by Stoppa in the groin [6]. For repairs of this type, one

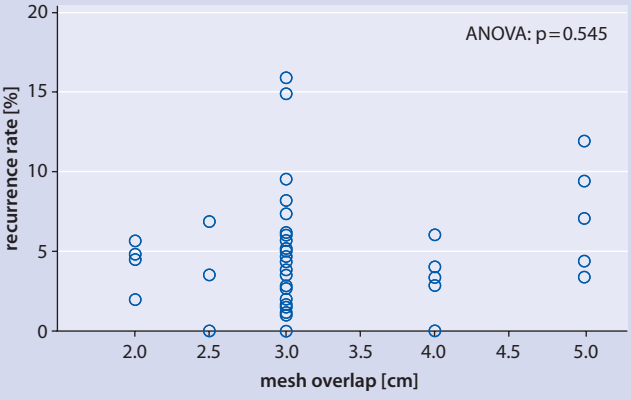


Fig. 20.4. Plot of hernia recurrence rate vs. minimum mesh overlap of the hernial defect for minimally invasive incisional/ventral herniorrhaphy. Complete data were available from 45 of the 53 manuscripts shown in Table 20.4

requirement for the mesh is that it should have adequate overlap (a more accurate term would be underlap) of the hernial defect [3]. That is, the margin of the mesh should extend beyond the margin of the defect by an appropriate amount throughout the defect's entire circumference. The range of mesh overlap in the 53 manuscripts of this review is shown in Fig. 20.4. Most (60%) of the authors favoured a minimum of 3cm of overlap; 24% indicated 4cm or more. One might hypothesize that the recurrence rate would decrease as the overlap increased, but this is not supported by plotting these two variables, as shown in Fig. 20.4 (it should be admitted that this is a relatively unscientific manipulation of uncontrolled data). The final answer to an appropriate amount of mesh overlap during minimally invasive incisional herniorrhaphy is not known, although 3cm most commonly is chosen. The optimal distance most likely is dependent on multiple variables, and may not be simply defined by "more is better."

Technical Factors: Mesh Fixation

One of the more controversial issues in minimally invasive incisional herniorrhaphy is the technique of mesh fixation. At a minimum, the laparoscopically performed sublay technique requires some fixation to keep the mesh anterior while pneumoperitoneum is present. Further fixation beyond this would be intended to prevent mesh migration/slippage with subsequent reherniation. The basic choices for fixation are (1) tacking/stapling, (2) transabdominal fixation sutures, or (3) a combination of both. Of the 53 manuscripts in this review, 44 contained sufficient details regarding mesh fixation; 69% of the papers utilized a combination of tacking/stapling and fixation sutures, while 29% utilized tacking/stapling alone (one paper used sutures alone). A plot of fixation technique vs. recurrence rate is shown in Fig. 20.5; there was no statistical difference in recurrence with respect to fixation. Nevertheless, given that a common cause of recurrent herniation is mesh slippage, it would seem reasonable to use the maximum amount of mesh fixation (i.e., lots of tacks/staples + lots of fixation sutures). Unfortunately, fixation sutures are associated with long-term abdominal pain, and they also require additional stab incisions in the skin and more operating time. We have spoken with surgeons who anecdotically claim that their recurrence rate is less with the combined use of tacks/staples and sutures, but controlled data are lacking. Furthermore, there are details of fixation technique (e.g., spiral tacks vs. straight staples, single vs. multiple rows of tacks, spacing between tacks and/or sutures, etc.), which further complicate the fixation issue. One of us (C.T.F.) utilizes a single row of straight staples at 1cm intervals (having obtained a 1.4% recurrence rate [1], while the other (M.A.C.) has changed his technique to a single row of spiral tacks at 1cm intervals with 2–0 polypropylene transabdominal fixation sutures placed every 5–7cm. The first author (C.T.F.) places each staple radially so that one end is buried into the PTFE while the other end takes tissue. In addition, he is careful that each staple enters the abdominal wall perpendicularly (using the two-handed stapling technique) to ensure maximum tissue penetration. It is this type of technical detail that could make the difference between a 1% vs. a 5% recurrence rate. In any event, it is difficult to recommend one fixation technique over another without

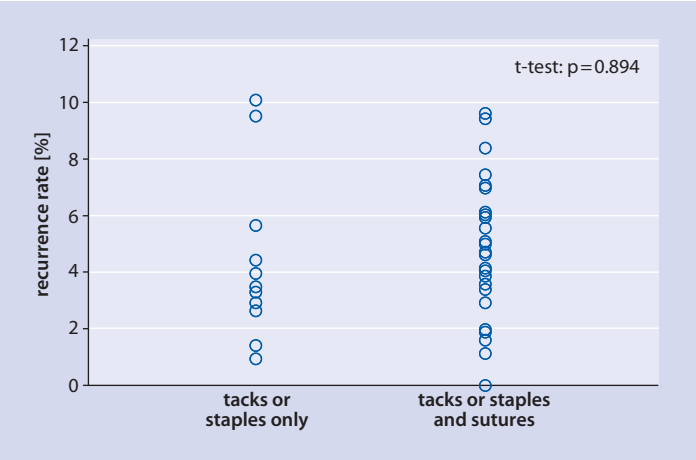


Fig. 20.5. Plot of hernia recurrence rate vs. technique of mesh fixation for minimally invasive incisional/ventral herniorrhaphy. Complete data were available from 44 of the 53 manuscripts shown in Table 20.4

controlled data. This is another area of surgery which will continue to be dictated by training environment, local experience, and so forth.

Technical Factors: Infection

Wound infection has been shown to be an independent risk factor for recurrence after open incisional hernia repair in numerous clinical series (data not reviewed here). Port-site infection after laparoscopic incisional hernia repair usually can be handled with antibiotics and local care without endangering the mesh; infection of ePTFE mesh itself, however, invariably means mesh removal with subsequent hernia recurrence. Although seemingly less common with the minimally invasive approach, mesh infection still had an incidence of 0.89% in the 5227 procedures of this review. There are a number of recommendations (expert opinion, not necessarily standard of care) to minimize the risk of major wound/mesh infection in minimally invasive incisional herniorrhaphy:

- pre-operative bowel preparation (mechanical and oral antibiotics);
- appropriate use of antibiotic prophylaxis;
- use of an antimicrobial-impregnated adhesive drape;
- avoidance of ePTFE contact with skin;
- changing surgical gloves prior to handling the mesh;
- careful surgical dissection with minimal blood loss;
- deferral of operation in the presence of incisional inflammation or stitch abscess.

Smoking should be minimized/eliminated pre-operatively, as this has been shown to be a risk factor for failure in open incisional herniorrhaphy. If the patient develops a large seroma postoperatively, then the surgeon should avoid the temptation of aspiration/drainage. The vast majority of these seromas will resolve without intervention; unnecessary violation of the space may introduce bacteria.

An issue related to infection is the management of intra-operative small bowel perforation. This complication occurred in 81 (1.6%) of the 5227 cases of this review. Details on the management of these cases were not available for all of them. In general, however, a surgeon has at least three options when a small bowel perforation is recognized intra-operatively: (1) convert to an open procedure, repair the enterotomy, and close the hernial defect primarily without a mesh; (2) if there is no enteric spillage, then repair the enterotomy laparoscopically and complete the mesh herniorrhaphy as planned; (3) repair the enterotomy laparoscopically, place the patient on IV antibiotics for several days, and then perform the minimally invasive incisional herniorrhaphy with mesh (usually the authors choice). There are variations to these options, but the essential choice is conversion vs. laparoscopic bowel repair and herniorrhaphy vs. laparoscopic bowel repair with delayed herniorrhaphy. The idea of placing a piece of PTFE in the face of potential enteric contamination (option 2 above) may not seem safe, but there are numerous successful examples of this management in the 53 articles of this review. Since the incidence of this complication is relatively low, it will be difficult to ascertain the optimal management, especially with respect to patient comorbidities. Consequently, treatment for each case

of intra-operative small bowel perforation will depend on the characteristics of the injury, surgeon's bias and experience, patient comorbidities, and so on. Intra-operative colon injuries are more rare; since the bacterial concentration in the colon is at least a millionfold of that in the small bowel, however, one should be wary of simultaneous repair of a colon injury and mesh placement.

Summary

At this relatively early stage in the history of minimally invasive repair of ventral/incisional hernia, a few recommendations for optimizing technique and reducing recurrence may be given:

1. Completely, yet carefully, expose the entire incision and anterior abdominal wall.
2. For intraperitoneal mesh placement, a dual-surface mesh which incorporates into the abdominal on one side while remaining relatively nonreactive to the viscera on the other appears optimal.
3. The ideal amount of mesh overlap of the defect is not known; a 3cm overlap seems reasonable.
4. The optimal form of mesh fixation needs to be studied by a carefully designed and controlled trial. At this point tacks/staples ± fixation sutures are the most popular techniques.
5. Minimize the risk of mesh infection; have a plan ready in the event of an intra-operative small bowel enterotomy.
6. Close all port sites for trocars >5mm.

Acknowledgements. Supported in part by a grant to MAC from the United States National Institutes of Health (K08 GM00703).

References

1. Frantzides CT, Carlson MA, Zografakis JG, Madan AK, Moore RE. Minimally invasive incisional herniorrhaphy: a review of 208 cases. Surg Endosc 2004; 18(10): 1488–1491
2. Tonouchi H, Ohmori Y, Kobayashi M, Kusunoki M. Trocar site hernia. Arch Surg 2004; 139(11): 1248–1256
3. Lowham AS, Filipi CJ, Fitzgibbons RJ Jr., Stoppa R, Wantz GE, Felix EL, Crafton WB. Mechanisms of hernia recurrence after preperitoneal mesh repair. Traditional and laparoscopic. Ann Surg 1997; 225(4): 422–431
4. Frantzides CT, Carlson MA. Minimally invasive ventral herniorrhaphy. J Laparoendosc Adv Surg Tech A 1997; 7(2): 117–120
5. Flament JB, Avisse C, Palot JP, Delattre JF. Biomaterials: Principles of Implantation. In: Schumpelick V, Kingsnorth AN (eds) Incisional hernia. Springer, Berlin Heidelberg New York, 1999
6. Stoppa R, Ralmiaramanana F, Henry X, Verhaeghe P. Prosthetic repair of recurrent groin hernias. In: Schumpelick V, Kingsnorth AN (eds) Incisional hernia. Springer, Berlin Heidelberg New York, 1999
7. Holzman MD, Purut CM, Reintgen K, Eubanks S, Pappas TN. Laparoscopic ventral and incisional hernioplasty. Surg Endosc 1997; 11(1): 32–35
8. Toy FK, Bailey RW, Carey S et al. Prospective, multicenter study of laparoscopic ventral hernioplasty. Preliminary results. Surg Endosc 1998; 12(7): 955–959
9. Tsimoyiannis EC, Tassis A, Glantzounis G, Jabarin M, Siakas P, Tzourou H. Laparoscopic intraperitoneal onlay mesh repair of incisional hernia. Surg Laparosc Endosc 1998; 8(5): 360–362
10. Koehler RH, Voeller G. Recurrences in laparoscopic incisional hernia repairs: a personal series and review of the literature. Jsls 1999; 3(4): 293–304
11. Kyzer S, Alis M, Aloni Y, Charuzi I. Laparoscopic repair of postoperation ventral hernia. Early postoperation results. Surg Endosc 1999; 13(9): 928–931
12. Sanders LM, Flint LM, Ferrara JJ. Initial experience with laparoscopic repair of incisional hernias. Am J Surg 1999; 177(3): 227–231
13. Chari R, Chari V, Eisenstat M, Chung R. A case controlled study of laparoscopic incisional hernia repair. Surg Endosc 2000; 14(2): 117–119
14. Chowbey PK, Sharma A, Khullar R, Mann V, Baijal M, Vashistha A. Laparoscopic ventral hernia repair. J Laparoendosc Adv Surg Tech A 2000; 10(2): 79–84
15. DeMaria EJ, Moss JM, Sugerman HJ. Laparoscopic intraperitoneal polytetrafluoroethylene (PTFE) prosthetic patch repair of ventral hernia. Prospective comparison to open prefascial polypropylene mesh repair. Surg Endosc 2000; 14(4): 326–329
16. Farrakha M. Laparoscopic treatment of ventral hernia. A bilayer repair. Surg Endosc 2000; 14(12): 1156–1158
17. Reitter DR, Paulsen JK, Debord JR, Estes NC. Five-year experience with the „four-before" laparoscopic ventral hernia repair. Am Surg 2000; 66(5): 465 468; discussion 468–469
18. Szymanski J, Voitk A, Joffe J, Alvarez C, Rosenthal G. Technique and early results of outpatient laparoscopic mesh onlay repair of ventral hernias. Surg Endosc 2000; 14(6): 582–584
19. Birgisson G, Park AE, Mastrangelo MJ Jr., Witzke DB, Chu UB. Obesity and laparoscopic repair of ventral hernias. Surg Endosc 2001; 15(12): 1419–1422
20. Andreoni KA, Lightfoot H, Jr., Gerber DA, Johnson MW, Fair JH. Laparoscopic incisional hernia repair in liver transplant and other immunosuppressed patients. Am J Transplant 2002; 2(4): 349–354
21. Aura T, Habib E, Mekkaoui M, Brassier D, Elhadad A. Laparoscopic tension-free repair of anterior abdominal wall incisional and ventral hernias with an intraperitoneal Gore-Tex mesh: prospective study and review of the literature. J Laparoendosc Adv Surg Tech A 2002; 12(4): 263–267
22. Bageacu S, Blanc P, Breton C, Gonzales M, Porcheron J, Chabert M, Balique JG. Laparoscopic repair of incisional hernia: a retrospective study of 159 patients. Surg Endosc 2002; 16(2): 345–348

23. Ben-Haim M, Kuriansky J, Tal R, Zmora O, Mintz Y, Rosin D, Ayalon A, Shabtai M. Pitfalls and complications with laparoscopic intraperitoneal expanded polytetrafluoroethylene patch repair of postoperative ventral hernia. Surg Endosc 2002; 16(5): 785–788
24. Berger D, Bientzle M, Muller A. Postoperative complications after laparoscopic incisional hernia repair. Incidence and treatment. Surg Endosc 2002; 16(12): 1720–1723
25. Gillian GK, Geis WP, Grover G. Laparoscopic incisional and ventral hernia repair (LIVH): an evolving outpatient technique. Jsls 2002; 6(4): 315–322
26. Kirshtein B, Lantsberg L, Avinoach E, Bayme M, Mizrahi S. Laparoscopic repair of large incisional hernias. Surg Endosc 2002; 16(12):1717–1719
27. Kua KB, Coleman M, Martin I, O'Rourke N. Laparoscopic repair of ventral incisional hernia. ANZ J Surg 2002; 72(4): 296–299
28. Lau H, Patil NG, Yuen WK, Lee F. Laparoscopic incisional hernioplasty utilising on-lay expanded polytetrafluoroethylene DualMesh: prospective study. Hong Kong Med J 2002; 8(6): 413–417
29. Parker HH, 3rd, Nottingham JM, Bynoe RP, Yost MJ. Laparoscopic repair of large incisional hernias. Am Surg 2002; 68(6): 530–533; discussion 533–534
30. Raftopoulos I, Vanuno D, Khorsand J, Ninos J, Kouraklis G, Lasky P. Outcome of laparoscopic ventral hernia repair in correlation with obesity, type of hernia, and hernia size. J Laparoendosc Adv Surg Tech A 2002; 12(6): 425–429
31. Salameh JR, Sweeney JF, Graviss EA, Essien FA, Williams MD, Awad S, Itani KM, Fisher WE. Laparoscopic ventral hernia repair during the learning curve. Hernia 2002; 6(4): 182–187
32. van't Riet M, Vrijland WW, Lange JF, Hop WC, Jeekel J, Bonjer HJ. Mesh repair of incisional hernia: comparison of laparoscopic and open repair. Eur J Surg 2002; 168(12): 684 689
33. Wright BE, Niskanen BD, Peterson DJ, Ney AL, Odland MD, VanCamp J, Zera RT, Rodriguez JL. Laparoscopic ventral hernia repair: are there comparative advantages over traditional methods of repair? Am Surg 2002; 68(3): 291 295; discussion 295–296.
34. Carbajo MA, Martp del Olmo JC, Blanco JI, Toledano M, de la Cuesta C, Ferreras C, Vaquero C. Laparoscopic approach to incisional hernia. Surg Endosc 2003; 17(1): 118–122
35. Chelala E, Gaede F, Douillez V, Dessily M, Alle JL. The suturing concept for laparoscopic mesh fixation in ventral and incisional hernias: preliminary results. Hernia 2003; 7(4): 191–196
36. Chowbey PK, Sharma A, Khullar R, Soni V, Baijal M. Laparoscopic ventral hernia repair with extraperitoneal mesh: surgical technique and early results. Surg Laparosc Endosc Percutan Tech 2003; 13(2): 101–105
37. Eid GM, Prince JM, Mattar SG, Hamad G, Ikrammudin S, Schauer PR. Medium-term follow-up confirms the safety and durability of laparoscopic ventral hernia repair with PTFE. Surgery 2003; 134(4): 599 603; discussion 603–604
38. Heniford BT, Park A, Ramshaw BJ, Voeller G. Laparoscopic repair of ventral hernias: nine years' experience with 850 consecutive hernias. Ann Surg 2003; 238(3): 391–399; discussion 399–400
39. LeBlanc KA, Whitaker JM, Bellanger DE, Rhynes VK. Laparoscopic incisional and ventral hernioplasty: lessons learned from 200 patients. Hernia 2003; 7(3): 118–124
40. McGreevy JM, Goodney PP, Birkmeyer CM, Finlayson SR, Laycock WS, Birkmeyer JD. A prospective study comparing the complication rates between laparoscopic and open ventral hernia repairs. Surg Endosc 2003; 17(11): 1778–1780
41. Mizrahi S, Lantsberg L, Kirshtein B, Bayme M, Avinoah E. The experience with a modified technique for laparoscopic ventral hernia repair. J Laparoendosc Adv Surg Tech A 2003; 13(5): 305–307
42. Rosen M, Brody F, Ponsky J, Walsh RM, Rosenblatt S, Duperier F, Fanning A, Siperstein A. Recurrence after laparoscopic ventral hernia repair. Surg Endosc 2003; 17(1): 123–128
43. Bamehriz F, Birch DW. The feasibility of adopting laparoscopic incisional hernia repair in general surgery practice: early outcomes in an unselected series of patients. Surg Laparosc Endosc Percutan Tech 2004; 14(4): 207–209
44. Bencini L, Sanchez LJ. Learning curve for laparoscopic ventral hernia repair. Am J Surg 2004; 187(3): 378–382
45. Bower CE, Reade CC, Kirby LW, Roth JS. Complications of laparoscopic incisional-ventral hernia repair: the experience of a single institution. Surg Endosc 2004; 18(4): 672–675
46. Franklin ME Jr., Gonzalez JJ Jr., Glass JL, Manjarrez A. Laparoscopic ventral and incisional hernia repair: an 11-year experience. Hernia 2004; 8(1): 23–27
47. Gal I, Balint A, Szabo L. Results of laparoscopic repair of abdominal wall hernias using an ePTFE-polypropylene composite mesh (in German). Zentralbl Chir 2004; 129(2): 92–95
48. Kannan K, Ng C, Ravintharan T. Laparoscopic ventral hernia repair: local experience. Singapore Med J 2004; 45(6): 271–275
49. McKinlay RD, Park A. Laparoscopic ventral incisional hernia repair: a more effective alternative to conventional repair of recurrent incisional hernia. J Gastrointest Surg 2004; 8(6): 670–674
50. Moreno-Egea A, Torralba JA, Girela E, Corral M, Bento M, Cartagena J, Vicente JP, Aguayo JL, Canteras M. Immediate, early, and late morbidity with laparoscopic ventral hernia repair and tolerance to composite mesh. Surg Laparosc Endosc Percutan Tech 2004; 14(3): 130–135
51. Muysoms F, Daeter E, Vander Mijnsbrugge G, Claeys D. Laparoscopic intraperitoneal repair of incisional and ventral hernias. Acta Chir Belg 2004; 104(6): 705–708
52. Sanchez LJ, Bencini L, Moretti R. Recurrences after laparoscopic ventral hernia repair: results and critical review. Hernia 2004; 8(2): 138–143
53. Ujiki MB, Weinberger J, Varghese TK, Murayama KM, Joehl RJ. One hundred consecutive laparoscopic ventral hernia repairs. Am J Surg 2004; 188(5): 593–597
54. Verbo A, Petito L, Pedretti G, Lurati M, D'Alba P, Coco C. Use of a new type of PTFE mesh in laparoscopic incisional hernia repair: the continuing evolution of technique and surgical expertise. Int Surg 2004; 89(1): 27–31
55. Angele MK, Lohe F, Dietz J, Hernandez-Richter T, Jauch KW, Heiss MM. Laparoscopic incisional hernia repair – an alternative to the conventional procedure? [German]. Zentralbl Chir 2005; 130(3): 255–259
56. Johna S. Laparoscopic incisional hernia repair in obese patients. Jsls 2005; 9(1): 47–50

57. Olmi S, Magnone S, Erba L, Bertolini A, Croce E. Results of laparoscopic versus open abdominal and incisional hernia repair. Jsls 2005; 9(2): 189–195
58. Perrone JM, Soper NJ, Eagon JC, Klingensmith ME, Aft RL, Frisella MM, Brunt LM. Perioperative outcomes and complications of laparoscopic ventral hernia repair. Surgery 2005; 138(4): 708 715; discussion 715–716

Discussion

Itani: *One of the issues that nobody addresses with laparoscopic surgery is the issue of cosmesis. As you know, in open surgery in all these deformed abdominal walls it is very easy to remove the scar, doing an abdominal plasty if needed, remove excess skin, but you cannot do that with the laparoscopic procedure.*

Frantzides: *You can do that with a laparoscopic procedure at the latest stage, which means a second operation later on.*

LeBlanc: *One thing that you didn't mention when you look at the fixation, and I know that you are not a proponent of suture as I am, there is no good consensus, but a lack of adequate follow-up in the majority of series that allow anyone to make a firm determination. There are only two or three series that have followed up beyond 2 or 3 years, so there are just not enough data; we need more prospective randomized trials to answer that question.*

21 Anatomical Limitations – Where Are the Layers?

J. Conze, A. Prescher

The surgical armamentarium to solve the persisting problem of incisional hernia has grown over the decades and recently expanded essentially by the laparoscopic techniques. However, the multitude of techniques is a typical sign that no procedure meets all requirements to answer every fascial defect of the abdominal wall. This might be explained by the different and difficult anatomy of the abdominal wall in the median compartment and both lateral compartments and their complicated transition zone of muscles, fascias and aponeuroses (Fig. 21.1).

The muscles of the abdominal wall, antagonistic to the muscles of the back, are important for every movement of the trunk. They are essential for erect position, regulate the intra-abdominal pressure, support defecation and furthermore they are permanently involved in supporting breathing.

From topographic-anatomical aspects the abdominal wall closes the skeletal gap between the lower thoracic aperture and the pelvis, the so-called lacuna sceleti sternopubica, according to August Rauber. The abdominal wall consists of different muscles, fascial structures, aponeuroses, peritoneum and intercalated nerves and vessels fixed within the osseous frame [1, 2].

On both sides of the midline the rectus abdominis muscle runs in vertical direction from the fifth to the seventh rib to the pubic bone (Fig. 21.2a). The muscle is separated by three to four horizontal tendineous intersections that fix the muscle to the anterior rectus sheath. At the lower insertion it is overlayed by the rudimentary pyramidalis muscle. The medial compartment is mainly a single muscle layer structure that is surrounded by the rectus sheath. This collageneous structure orgin-

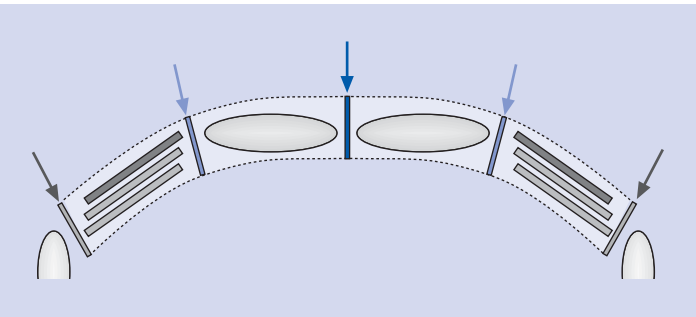


Fig. 21.1. The four compartments of the abdominal wall, separated by the Linea alba (*dark blue arrow*), the lateral margins of the rectus sheath (*light blue arrows*) and the osseous frame (*grey arrows*)

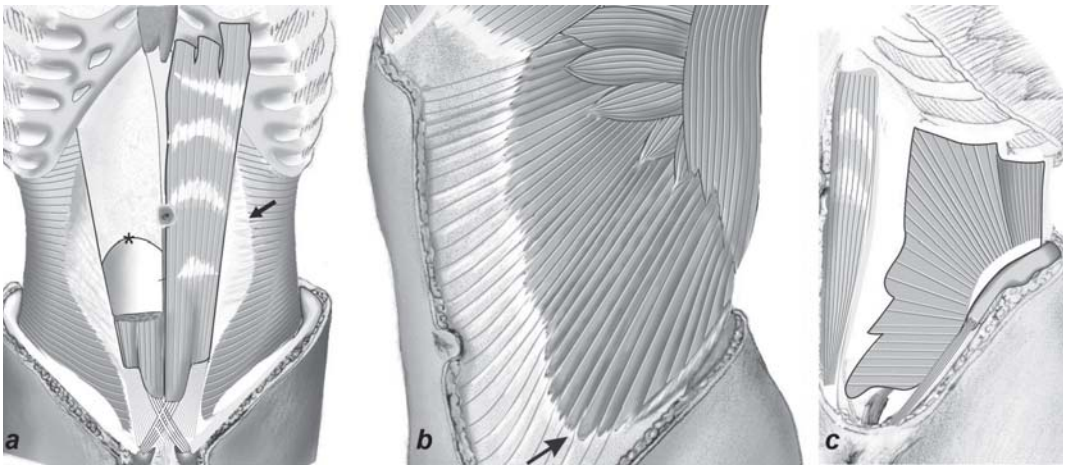


Fig. 21.2a–c. Schematic drawings of the muscular and fascial components of the abdominal wall. **a** M.rectus abdominis and M.transversus abdominis (star: arcuate line of Douglas in the posterior lamina of the rectus sheath; *arrow*: semilunar line of Spighel). **b** M.obliquus abdominis externus abdominis. **c** M.obliquus internus abdominis; note the different fibre directions in the different parts of the muscle

ates from the aponeuroses of the oblique muscles of the lateral compartment of the abdominal wall.

The lateral compartment of the abdominal wall is formed by three oblique muscles that run in different directions [3]. The external oblique muscle runs in a cranial to caudal direction from the fifth to the twelfth rib to the iliac crest, pubic tubercle and linea alba (Fig. 21.2b). Beneath this structure lies the internal oblique muscle, presenting different parts with different fibre directions (Fig. 21.2c). This muscle originates from the iliac crest, the lumbodorsal fascia and from the lateral part of the inguinal ligament; it terminates at the ribs and the linea alba. Between these two muscles an avascular layer of loose connective tissue can be found. The transverse muscle runs more horizontally from the seventh to the twelfth rib, the deep sheet of the lumbodorsal fascia, the iliac crest and the lateral part of the inguinal ligament of Poupart to the xiphoid process, the linea alba and the medial parts of the pubic bone (Fig. 21.2a). Between these muscles the neurovascular bundles are intercalated.

The rectus sheath presents a different architecture above and below the arcuate line (Fig. 21.3). Above this variable line the anterior rectus sheath is formed by the aponeurosis of the external oblique muscle and the ventral part of the aponeurosis of the internal oblique muscle. The posterior rectus sheath, on the other hand, is formed by the posterior part of the aponeurosis of the internal oblique muscle and the aponeurosis of the transverse muscle. Approximately 3–5cm below the umbilicus the structures forming the posterior rectus sheath above also join the anterior rectus sheath. The zone where this change takes place is the arcuate line of Douglas (Fig. 21.2a). According to these conditions, the posterior lamina of the rectus sheath beneath the arcuate line is formed only by the transversal fascia.

Incisional hernia repair with mesh is principally an augmentation of the abdominal wall. To achieve sufficient and stable mesh integration, a tissue overlap of 5cm has been shown to be the minimum to prevent hernial recurrence at the mesh border. The amount of overlap seems to be independent of the mesh position within the abdominal wall, with exception of the inlay technique where the prosthesis is placed to bridge the fascial defect; but even in the laparoscopic bridging technique a sufficient overlap is postulated.

In the onlay technique, where the meshes are placed epifascially, there are no anatomical limitations. The mesh implantation with a sufficient overlap can be easily performed. Limitation must be expected only if the fascial defects are neighbouring osseous structures such as the xiphoid process, the ribs or pubic bone. The same applies for the open or laparoscopic IPOM techniques, where the mesh is placed onto the parietal peritoneum within the abdominal cavity. The extension to osseous structures is achievable in the pubic region by dissection of the urinary bladder and opening the preperitoneal space as in the inguinal TAPP procedure. To cover defects which are bordered by osseous structures in the upper abdomen, the mesh is placed onto

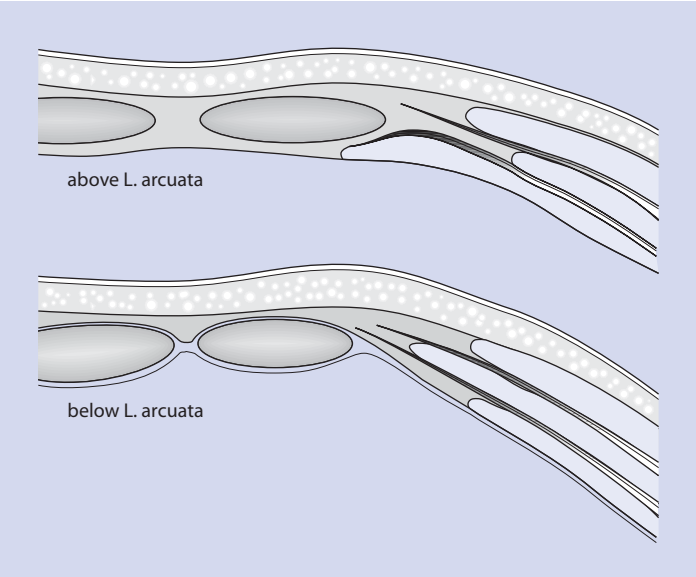


Fig. 21.3. Anatomical-topographical view of the components of the rectus sheath above and below the arcuate line

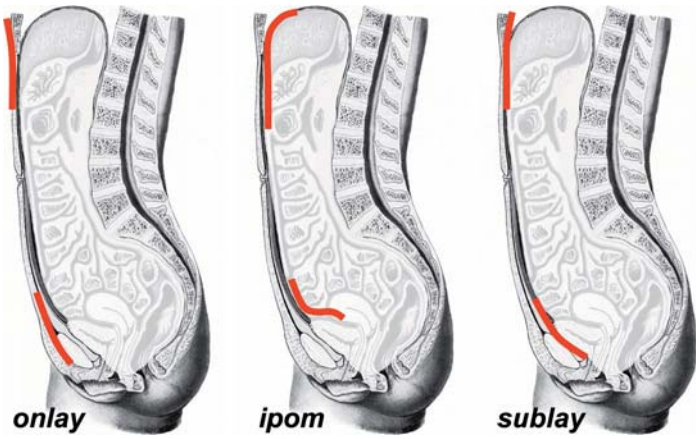


Fig. 21.4. Mesh position and neighbouring osseous structures in different techniques

the diaphragm with limited options for mesh fixation. It should be kept in mind that mesh-related complications threaten if meshes are in direct contact with intra-abdominal structures.

The standard procedure for incisional hernias of the midline is the sublay technique. The mesh is covered by tissue of the abdominal wall on both sides, the rectus muscle externally and the posterior rectus sheath internally, thus preventing a direct contact with the intestines. A sufficient mesh subduction cranial and caudal of the defect can be achieved by incision of the posterior rectus sheath on both sides of the linea alba, opening the preperitoneal space that appears like a fatty triangle [4]. In the case of neighbouring osseous structures, the preparation can be extended into the retroxiphoidal or retropubic area (Fig. 21.4) [5].

This is different when the defect neighbours or crosses the lateral margin of the rectus sheath, as occurs in transverse or pararectal incisional hernias. Due to the different muscular and fascial composition of the lateral and medial compartment, the preparation of a mesh layer is more challenging. In the lateral compartment the ideal anatomical layer is between the external and internal oblique muscle. This avascular connective tissue plane is known from the abdominal wall separation technique of Ramirez [6]. In the case

of incisional hernia defects crossing the compartments, a mesh extension from the medial-retromuscular to the lateral-intermuscular layer (between external and internal oblique muscle) is a possibility to fulfil the postulates of mesh repair.

References

1. Prescher A, Lierse W (2000) Anatomie der ventralen Leibeswand. In: Schumpelick V (Hrsg) Hernien. Thieme, Stuttgart, pp 1–27
2. Prescher A (1999) Surgical anatomy of the abdominal wall. In: Incisional Hernia. Springer, Berlin Heidelberg New York, pp 45–60
3. Klinge U, Prescher A, Klosterhalfen B, Schumpelick V (1997) Development and pathophysiology of abdominal wall defects. Chirurg 68: 293–303
4. Conze J, Prescher A, Klinge U, Saklak M, Schumpelick V (2004) Pitfalls in retromuscular mesh repair for incisional hernia: the importance of the "fatty triangle". Hernia 8: 255–259
5. Conze J, Prescher A, Kisielinski K, Klinge U, Schumpelick V (2004) Technical consideration for subxiphoidal incisional hernia repair. Hernia 9(1):84-7
6. Ramirez OM, Ruas E, Dellon AL (1990) "Components separation" method for closure of abdominal-wall defects: an anatomic and clinical study. Plast Reconstr Surg 86:519–526

Discussion

Frantzides: *I don't advocate an overlap of 2 cm, but what I use personally is at least 3cm overlap. The data show, however, based on the 53 papers that I have reviewed, that it doesn't matter, there is no statistical significant difference if the overlap is 2 or 5cm.*

Conze: *If we talk about evidence and prospective studies, there are only two studies, that is the study from Luijendyk/The Netherlands and the Vypro I study. The Luijendyk study had an overlap of 2cm and didn't close the fascia in front of the mesh in all cases. This study, with the follow-up by Burger, has a high recurrence rate and is always mentioned to show the limitations of this technique; but we should also look at the limitations of this study protocol, where augmentation and bridging techniques are mixed together. In the Vypro I study there was an overlap of 5cm, with a result of 12% recurrences after 24 months compared to 23% in the Luijendyk study. So I believe there is considerable importance concerning the overlaps and again, the mesh polymer and structure has also a great impact.*

Deysine: *You have presented us with a challenge that will demand another conference. Basically, if you approach a flank hernia, e.g. postnephrectomy, it is easy to anchor the mesh in the front, but then at the top you have to anchor it to the rib and in the lower abdomen you have nothing to anchor to. There is no answer to this. Most of the talks on abdominal ventral hernia repair don't face this problem. It will require a lot of imagination, so I congratulate you on opening this problem.*

Conze: *It's not only in the talks that you don't find this topic, its also missing in all the hernia books.*

Bendavid: *I have seen at least six cases of iliac crest hernias that were quite generous, and I have never had any problem, because all I have done was drill holes, up to nine of them, and anchor a Marlex or polypropylene mesh of any kind.*

Conze: *How is the mobilization of the patient afterwards? I am afraid that might cause some limitations, most certainly if you take heavy-weight meshes.*

Bendavid: *None whatsoever.*

Flament: *The only point where I disagree totally with you is when you write "no mesh fixation to body structures". At the end of the 19th century, anatomists showed that with three stitches through the Cooper ligament you can lift the cadaver. Why not use these thick structures, e.g. the iliac crest, to put stitches in?*

Conze: *I personally believe that the abdominal wall is something dynamic and I want to keep it like this. Mesh fixation to osseous structures will have an influence on the mobility and dynamic.*

Schumpelick: *We have learned from Rene Stoppa that a large overlap is better than fixation, and there is no fixation in the Stoppa procedure!*

22 Biomechanical Data – "Hernia Mechanics": Hernia Size, Overlap and Mesh Fixation

R. Schwab, U. Klinge, O. Schumacher, M. Binnebösel, K. Junge, V. Schumpelick

Introduction

Mesh repair has not been able to eliminate hernia recurrence. Therefore several possible biomechanical causes have been accused: the size of the prosthesis, the extent of surgical dissection, the overlap of the mesh and whether it is properly secured, all have been shown to affect the risk of recurrence after hernia repair.

Secure mesh fixation is intended to prevent the risk of recurrence due to implant dislocation caused by abdominal shear forces. The fixation of biomaterials is required until sufficient ingrowth has made collagen impregnation sufficiently strong to ensure repair of the fascial defect. Whereas there is controversy about the need for fixation in preperitoneal inguinal hernia repair (sublay position), there is consent that an additional mesh fixation in anterior inguinal (onlay position) and all types of incisional hernia repairs seems to be essential.

In preperitoneal repairs, the fixation of the prosthesis is postulated to be strong enough based only on the physiological intra-abdominal pressure and no additional suturing or fixing is mandatory in the case of a sufficient overlap. On reviewing the literature, a lack of biomechanical data regarding this problem becomes apparent. Therefore we developed a standardized hernia simulation model to investigate possible correlations between hernia size, overlap and mesh fixation.

Design of the Hernia Test Stand and Methods

In co-operation with the Fraunhofer Institute for Production Technologies, Aachen, a standardized test stand was realized to simulate abdominal wall hernias and their reconstruction in a sublay and onlay setup. According to our previous investigations, the physiological landmarks to simulate different abdominal peak pressures of up to 200 mmHg and an abdominal wall elasticity of 20 to 30% at a pressure level of 150 mmHg were set.

The so called hernia test stand" (◘ Fig. 22.1) is characterized by four main components:

- The pressure chamber to simulate the abdominal cavity. This includes a highly elastic and ultrathin silicone sac to display the peritoneum, which can be insufflated by air pressure.
- The standardized abdominal wall is patterned by a silicone sheet of 20 to 30% of elasticity combined with fresh porcine muscular tissue as mesh layer.
- The digital imaging unit to monitor the face of contact and mesh deformation during abdominal pressure enhancement.
- The measurement device to determine the protrusion of the mesh and abdominal wall during abdominal pressure enhancement

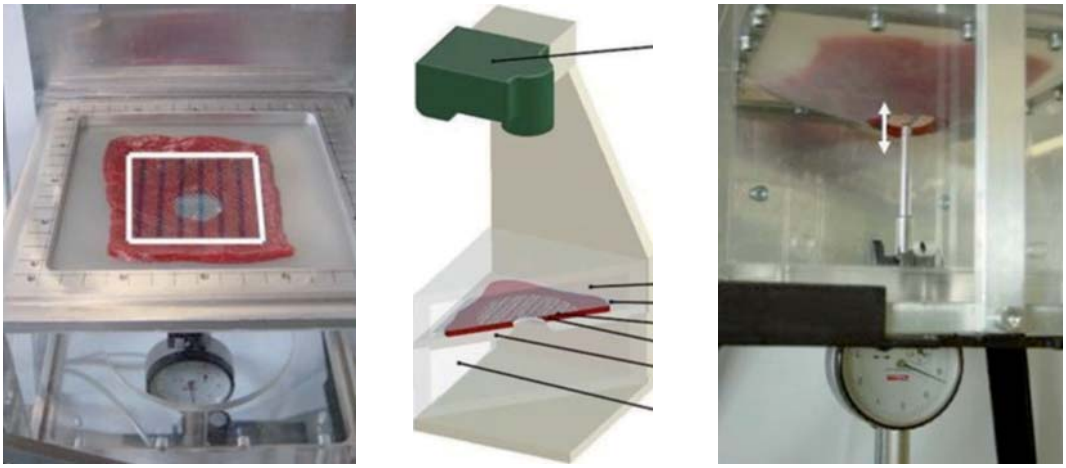


Fig. 22.1. Standardized model for abdominal wall hernia simulation, the hernia test stand. Monitoring of the mesh dislocation (*left*) and protrusion of the mesh and abdominal wall (*right*) during pressure enhancement

By replacing the genuine abdominal wall by a standardized silicone membrane with comparable biomechanical properties, it is possible to eliminate a main source of errors due to varying anatomical specimen. The porcine muscular tissue as mesh layer performs no mechanical work but serves as gliding and fixation sheet for the mesh.

Therefore it is possible to investigate the impact of a varying overlap, defect size, mesh or fixation technique in a model of otherwise static biomechanical parameters.

Overlap and Mesh Fixation: Sublay Setup

Using this standardized in vitro model of the abdominal wall, the compressive, tensile and shear forces were simulated at abdominal pressures of 0–200 mmHg. Mesh deformation and dislocation at the abdominal wall and mesh protrusion into the bridged defect were determined during abdominal pressure enhancement in a sublay setup (Figs.22.1 and 22.2). The biomechanical properties of ten most frequently used meshes (Marlex®, Atrium®, Premilene LP®, Mersilene®, Dual

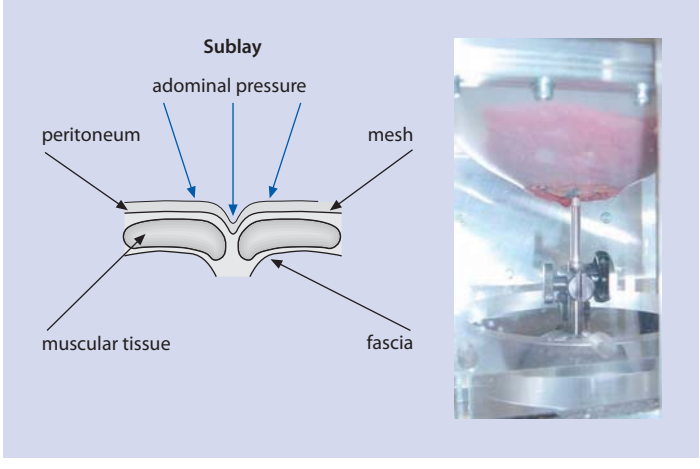


Fig. 22.2. Circular defect in a simulated sublay repair

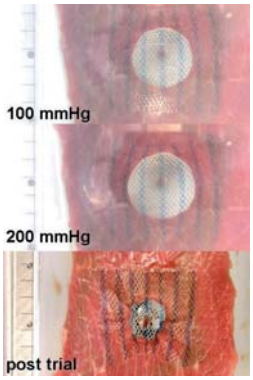


Fig. 22.3. Simulation of a sublay repair without mesh fixation (initial overlap 2 cm) and movement of the mesh on the muscle during pressure enhancement

mesh®, Ti-mesh light®, Ti-mesh extra light®, Vypro®, Vypro II® and Ultrapro®) were examined.

During consecutive test series with a varying defect size of 1 to 3 cm and a decreasing overlap starting by 3 cm (defect to overlap = 1:1) the critical overlap of non-fixation, associated with mesh dislocation through the defect, was detected. The material properties of all ten meshes brought into the trial are found to be sufficiently stable. There was no rupture or destruction of any single mesh found following simulated abdominal pressures of up to 200 mmHg. Without fixation, in all cases a relative movement between the mesh and the muscular layer of the abdominal wall was observed (Fig. 22.3). Starting by a 3-cm overlap and a defect size of 3 cm (defect tooverlap = 1:1), only a minimal relative movement was monitored. In the case of a 2 cm overlap (defect to overlap = 3:2) the gliding is significant, but it was not yet possible to push a mesh through the defect at a peak pressure of 200 mmHg (Fig. 22.3). The critical overlap was determined at a defect-overlap ratio of 2:1. In a simulated defect of 3 cm and a 1.5 cm overlap nine in ten meshes dislocated through the defect at a mean pressure of 160 mmHg with a range of 125 to 200 mmHg. In the same setup, at the critical overlap of 1.5 cm bridging a 3-cm defect, the meshes were fixed at certain points using either eight single sutures (Prolene® 2/0) or glued to the whole muscular surface by 1ml fibrin sealant (Tissucol®). The quality of fixation was assessed by analyzing the mesh deformation, mesh dislocation and the protrusion of the abdominal wall during pressure enhancement.

Fixing meshes by suturing or gluing reliably prevents the mesh dislocation even at a peak pressure of 200 mmHg (Fig. 22.4). None of the ten meshes was observed to be pushed through the defect. Analyzing

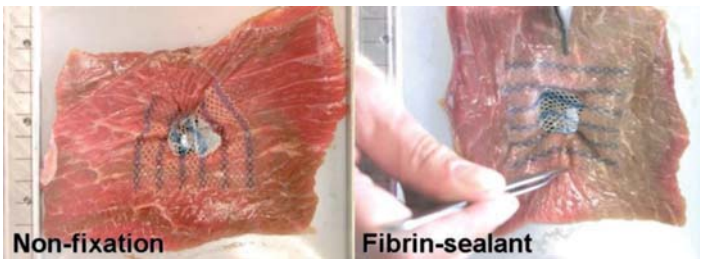


Fig. 22.4. Post trial: sublay simulation (1.5 cm overlap) following non-fixation (dislocation through the defect at 150 mmHg) and fibrin sealant (no movement or dislocation after pressures up to 200 mmHg)

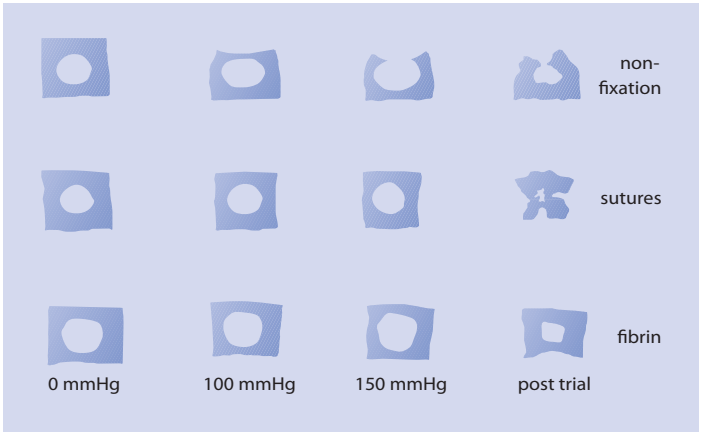


Fig. 22.5. Face of contact between mesh (Ultrapro®) and abdominal wall following different fixation techniques (non-fixation vs. eight single sutures vs. fibrin glue. Mesh in a sublay position bridging a 3-cm defect by a 1.5-cm overlap

the protrusion of the mesh and abdominal wall leads to a lower protrusion and higher stability of the mesh and abdominal wall compound for the glued group. The difference was significant comparing fibrin with non-fixation at pressures between 0 and 150 mmHg, but did not even reach a significant level comparing sutures and fibrin at pressures of up to 200 mmHg.

The surface of contact between mesh and abdominal wall was assessed by digital imaging (Fig. 22.5). It could be demonstrated that any kind of fixation prevents the mesh from dislocation through the defect, but a fixation of the whole surface of the mesh by gluing guarantees a secure position of the implant on the layer without folding after pressure release.

Fixation of Meshes: Onlay Setup

An onlay setup was chosen to evaluate the biomechanical properties and stability of mesh fixation following various suture techniques and fibrin sealant in a simulated Lichtenstein repair. The onlay mesh was fixed by four and six single sutures (Prolene® 2/0) or by the use of fibrin glue (Tissucol®). In two further test series an additional closure of the 3 cm hernia orifice was performed by a running suture (Prolene® 2/0). The simulated abdominal pressure was continuously elevated until a mesh dislocation was observed. Furthermore, the pressure level was determined when the hernia orifice re-opened, depending on the technique of repair (Fig. 22.6).

In onlay repair sealing the meshes with fibrin is superior to four single sutures and at least equivalent to all mere suture techniques. The best biomechanical results can be achieved by combining a continuous suture closure of the defect and mesh fixation using fibrin glue. Based on the data gathered, the clinical use of mesh fixation with fibrin sealant is considered to be sufficiently safe and stable as far as inguinal hernia surgery is concerned.

Conclusions

In sublay repair, a permanent mesh fixation is not mandatory if the overlap exceeds a defect-overlap ratio of 2:1. Therefore an overlap of more than 2 to 3 cm in all directions should be sufficient for most inguinal defects. These data gathered in vitro are in accordance with the clinical research results of individual series. In the case of large medial hernia defects, it could be indicative for additional mesh fixation. Fibrin gluing represents a reliable method of mesh fixation to prevent early mesh dislocation even at peak pressures of 200mmHg.

Larger longitudinal defects such as found in incisional hernia have to be further investigated using a recently designed modified test device. Preliminary results will lead us to a requested overlap of at least 4 to 6 cm. Whether a postulated ratio of defect-overlap has to exceed 2:1 has to be verified by further investigations.

Discussion

Miserez: *Firstly, there was a paper published 2 or 3 years ago with a similar high-pressure chamber where Vypro did not perform very well; with a collapse of the mesh in the different experimental setups. Secondly, I am still struggling with the tensile strength of the abdominal wall,*

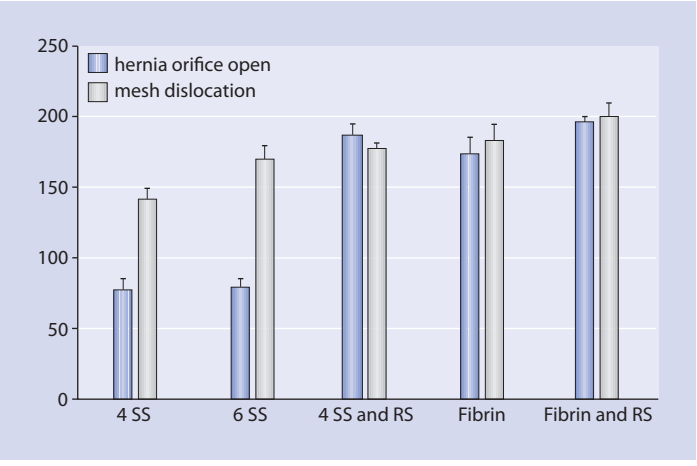


Fig. 22.6. Onlay simulation and fixation of the mesh by single sutures (SS) or fibrin sealant (fibrin) and additional closure of the defect by running sutures (RS)

16 or 32 N, is this in both the transversal and longitudinal direction the same or is it different?
Schwab: *They explanted, we have standardized. These are the calculated forces, we don't know the forces.*
Kukletta: *It is very impressive how fibrin glue, which is not a glue, improved your results. Did you think of using a glue that really is a glue, because fibrin doesn't have any adhesive strength?*
Schwab: *Although fibrin is not glue it has adhesive strength; you can take some fibrin and lift a Swiss chocolate of 200 g without any problem. It is not a question of taking a glue at each edge point. The future could be to coat the mesh with a glue so that the mesh doesn't move at all. The peaks at square millimetres are smaller than if you have three or four fixation points.*

Concluding Remarks

Carlson: *My summary of how to perform a suture repair: in general don't do it except in very limited indications like small defects (fingertip size) or infected wounds.*
Flament: *In open incisional hernia repair we favour the open sublay technique, for most but not all types of incisional hernia, with a low rate of septic complications, and good functional and cosmetic results. When this is not possible, we need a very dedicated mesh for intra-abdominal placement. We must not forget that these hernias are a general disease, and we must have a very long follow-up, since 8–10% of the recurrences occur after 10 years. So retromuscular seems better, though I still do not understand why not fix the mesh also to the bone as you stitch the mesh all around the abdominal wall? For a small number of patients we need a good prosthesis for intra-abdominal placement.*

How to Treat the Recurrent Incisional Hernia

23 Open Repair

23.1 How to Treat the Recurrent Incisional Hernia: Open Repair in the Midline

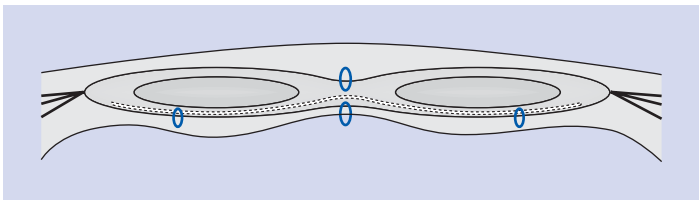
V. Schumpelick, U. Klinge, R. Rosch, J. Conze, K. Junge

Introduction

Even with the routine use of mesh, repair of an incisional hernia is a challenge. Increasing evidence of impaired wound healing in these highly selected patients supports routine use of an open prefascial, retromuscular mesh repair (◘ Fig. 23.1). Basic pathophysiological principles dictate that a wide overlap underneath healthy tissue is required for a successful long-term outcome and prevention of recurrence. The extent of overlap should be 5 cm in all directions: surrounding the fascial wound closure, subxiphoidal, underneath the ribs, below the arcuate line and retropubic. Generally, it is not the preparation of the hernia sac or the dissection of the mesh area that generates difficulty during the management of recurrent incisional hernias, but it is the patients and their comorbid conditions which may cause some troubles and delay the reconvalescence.

Pathophysiological Concept

For decades incisional hernia was hypothesized to be caused mainly by technical problems with suture technique. Consequently, correction of this problem was undertaken by a repeat, but more meticulous, suture repair with a variety of configurations to prevent recurrence. Additional doubling of the fascia to reinforce the abdominal wall was performed in some cases. Whereas the intra-operative aspects were ostensibly satisfying, the long-term results were disappointing. Recurrence rates of 50% after suture repair of an incisional hernia were reproduced in several studies [2, 19]. It was the introduction of mesh by Usher et al. in 1958 that opened a new era [20]. Reinforcement of the abdominal wall with strong polyester or polypropylene nets produced a resilient scar–mesh compound that



◘ **Fig. 23.1.** Mesh position in retromuscular, prefascial (sublay) position

prevented recurrences through the mesh. Indeed, recurrences through mesh are still a rarity. In accordance with the widespread use of mesh, several personal series reported excellent results, with recurrence rates of far less than 10%. Examination of the literature shows that the results are independent of mesh type and operative technique [11, 18]. This early euphoria has been recently clouded by the results of the only randomized, controlled trial that has compared mesh and suture repair [1, 16]. In this Dutch trial, a mesh was applied in a prefascial, retromuscular position with an overlap of 2 to 4 cm. After 3 years there were 43% recurrences in the suture group, as expected. However, the authors found 24% recurrence in the mesh group as well. Interestingly, the results were not affected by the size of the hernia. Furthermore, in 2003 a retrospective population-based cohort study by Flum et al. analyzed data from 10,822 patients operated on for incisional hernia by either suture or unspecified mesh repair [5]. Within 5 years, 14% of the patients after suture repair underwent at least one subsequent re-repair compared to 11% after mesh repair (log-rank differences, $p < 0.001$). First, both studies clearly reveal a reduced recurrence rate after mesh implantation at any time point. The lower rate seen by Flum et al. might be due to the fact that several patients with a relapse have not undergone reoperation. Nevertheless, the most striking fact is that both studies unexpectedly found a constantly rising incidence of hernia recurrence over the years of follow-up, not only in the suture group but in the mesh group as well. Over a decade, this recurrence rate shows an almost linear curve. Comparing suture to mesh, the implants seem only to delay the recurrence for 2 to 4 years and the time course never reaches a plateau. These data substantiate that the development of a recurrence in an incisional hernia repair is not primarily a technical one. Recently, molecular biological investigations have proven the theory of disturbed composition of the extracellular matrix in patients with recurrent hernia. In particular, there is a decreased ratio of collagen types I and III [7, 9, 10, 12, 13]. Furthermore, in situ dysfunction in fibroblasts has been found, indicating a primary malfunction of the cells independent of their local environment.

Reinforcement of the closed hernial gap by mesh is based on the concept of ingrowth of fibrous tissue into prosthetic material, forming a scar–mesh compound. Although the intensity of the scar formation is influenced by the amount of material, its quality is not improved [8]. As a consequence, mesh fixation by fibrosis cannot prevent recurrence unless a wide overlap underneath healthy tissue can be achieved. Although such data are not yet published, it may be hypothesized that the width of the overlap correlates with the duration of the delay. However, clinical experience with all techniques developed in recent years has uniformly shown a trend towards utilizing larger prostheses.

In conclusion, a defective process of wound healing should be assumed in patients suffering from incisional hernia. Rare exceptions refer to patients with a traumatic defect or an obvious technical fault (e.g. unclosed trocar incisions or broken suture material). Consequently, every connection end-to-end has to be regarded as a weak point and should be avoided.

In accordance, every surgical technique has to consider this biological cause for the incisional hernia. Taking into account an insufficient scar formation, this lead to the requirement of an extended overlap, and it is the frontal coverage with a large area of healthy tissue which hinders the hernia recurrence at the mesh border.

This principle must be carefully considered regarding technique and performance, and the surgeon has to decide where to place the mesh, and how to achieve the demanded overlap.

From our experience, the placement of a mesh behind the rectus muscles, above the linea arcuata in front of the posterior rectus sheath is advantageous. In contrast to a mesh in front of the fascia in onlay position, the sublay mesh position facilitates a sufficient subduction of intact linea alba, even behind the xiphoid or pubic bone. It is the retromuscular mesh with a fascia closure in front, which is kept in position just by tissue ingrowth and intra-abdominal pressure, whereas the onlay mesh has to be fixed additionally by permanent sutures. The mesh in the space behind the rectus muscle can be easily dissected, whereas the extended preparation of the subcutaneous space in the case of the onlay position frequently is accompanied by haematoma or seroma, in the case of infection sometimes even leading to a disaster.

Incisional Hernia Repair in the Midline Using a Retromuscular, Prefascial Mesh Prosthesis (Sublay Repair)

Apart from surgical repair, there is no alternative treatment for incisional hernia. Hernia repair should be considered early because of the tendency for the defect to increase in size and impair quality of life. Apart from previous technical faults or traumatic defects of the abdominal wall, the surgical technique should

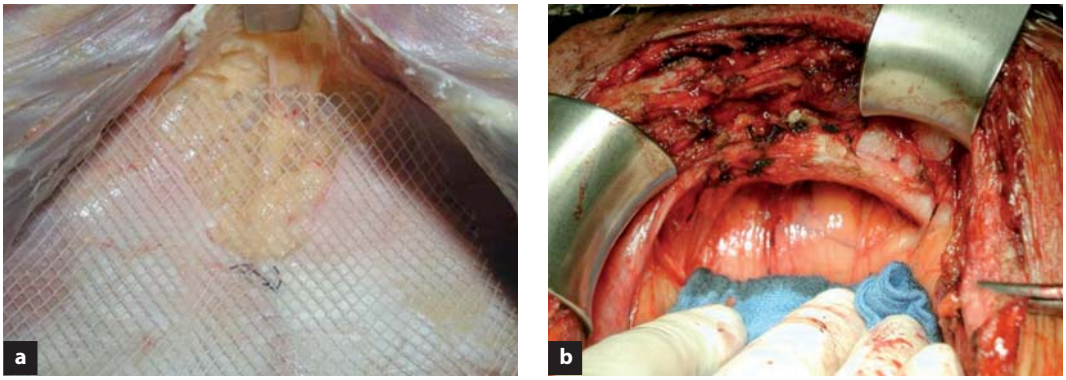


Fig. 23.2a,b. Preparation of the (**a**) cranial and (**b**) caudal border

routinely include the use of mesh. The only exception may be small defects of less than 3 cm, which can be closed by a continuous nonabsorbable suture repair (a suture length/wound length ratio of 4:1). In the case of a giant hernia or obesity, pre-operative improvement of respiratory function and reasonable weight reduction [3] should be encouraged. Additionally, the skin should be in optimal condition to minimize the risk of infection. Pre-operative bowel preparation and perioperative antibiotics are advisable. In principle, flat mesh is placed in a prefascial, retromuscular position to reinforce the fascia closure and form an extended mesh–scar compound. After excising the entire skin scar, the hernia sac is prepared down to the margins of the fascia. The sac then is opened, and local adhesiolysis eases the complete opening of the previous incision. For midline incisions, the retromuscular space behind the rectus muscles and in front of the posterior rectus sheath (prefascial) is bluntly dissected. The neuro-vascular bundles at the lateral part should be preserved as carefully as possible. At the cranial margin, the posterior sheath is incised on both sides parallel to the linea alba. A triangle of preperitoneal fat with separating fascial margins them becomes apparent (Fig. 23.2a) [4]. To realize a sufficient overlap (at least 5 cm), preparation continues far behind the xiphoid. Similar preparation is needed at the caudal margin of the fascial incision, where below the arcuate line the disappearing posterior rectus sheath demands dissection in the fatty preperitoneal space. Finally, the mesh is placed behind the pubic bone in front of the bladder (Fig. 23.2b). It is advisable to complete the circular preparation of the preperitoneal mesh placement before closing the peritoneum to avoid damage during the dissection to closely attached bowel or organs.

A major task is the prevention of direct contact between the bowel and the mesh prosthesis to avoid dense adhesions or late fistulas. Thus, the peritoneum or, if necessary, hernia sac tissue must be carefully closed by continuous absorbable suture. A further interposition of omentum might be helpful, especially in cases of peritoneal defects. After careful control for bleeding, the mesh is trimmed to fit the specific dimensions of the defect to be treated. Usually, implants have a width of 12 to 14 cm and a length of 20 to 35 cm (Fig. 23.3).

Respecting the physiological elasticity of the abdominal muscle fibres, the mesh should feature its main elasticity in a vertical direction. This ensures adaptation to the physiological stretchability of the abdominal wall and reduces craniocaudal shrinkage by mesh deformation. An overall overlap of at least 5 cm in all directions is mandatory (Fig. 23.4). To prevent early dislocation, the unfolded mesh is fixed circularly to the

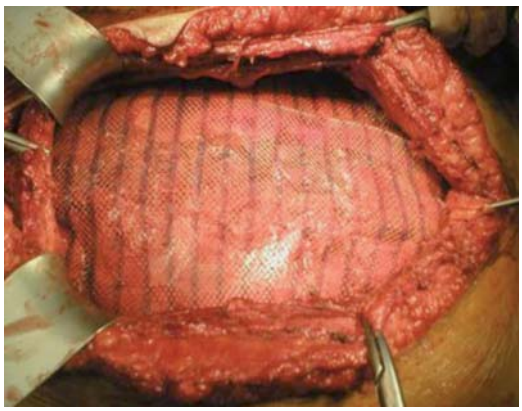


Fig. 23.3. Mesh placed in retromuscular, prefascial position

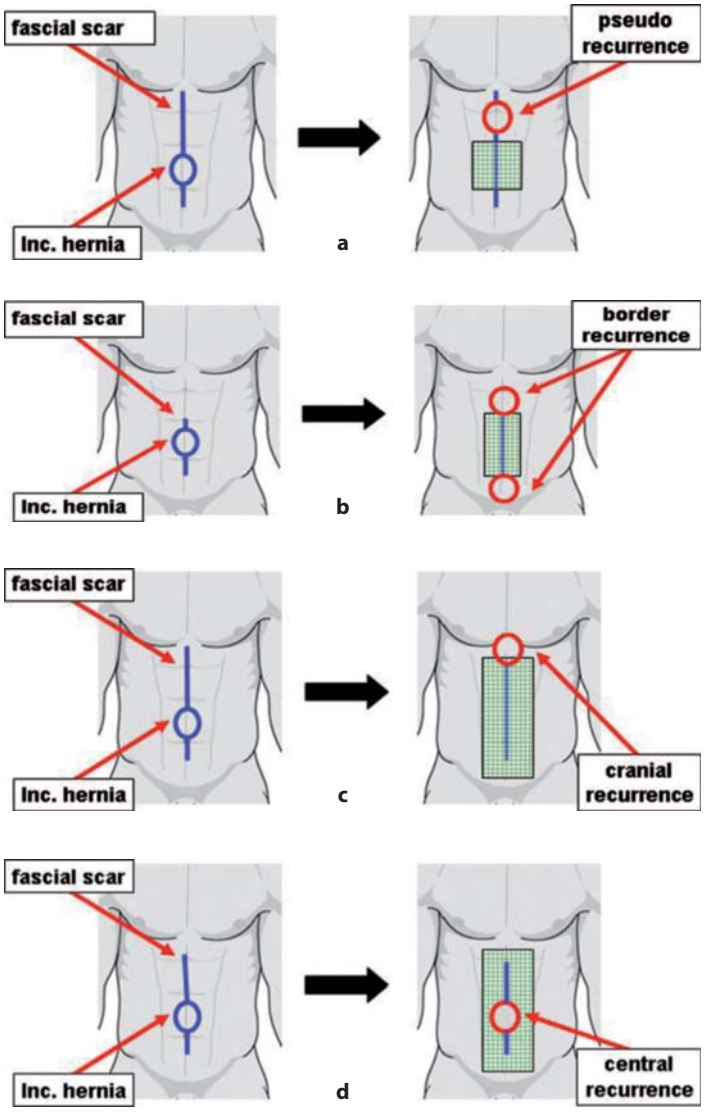


Fig. 23.4a–d. Technical pitfalls: **a** pseudo-recurrence due to insufficient excision of the entire fascial scar. **b** Recurrence at the borders due to insufficient overlap at the margins (fatty triangle, retropubic space). **c** Cranial border recurrence due to insufficient preparation behind the xiphoid (subxiphoidal space). **d** Central mesh recurrence due to insufficient closure of the anterior rectus sheath

posterior rectus sheath and the peritoneal sac below the arcuate line, respectively. It remains controversial as to whether the use of non-absorbable sutures is absolutely indicated. During fascia closure, wrinkling of the mesh should be avoided. After placing drains in the retromuscular space, the anterior fascia is closed by non-absorbable continuous suture respecting a 4:1 ratio for suture/wound length. Working as thrust-bearing and preventing early strain to the mesh, closure of the fascia is imperative. If closure of the anterior fascia occurs with undue tension, relaxing incisions in the anterior rectus sheath or an additional Ramirez component separation is added. Skin closure follows as usual. Postoperative care is mainly directed to the control of wound problems. Because the mesh is assumed to be integrated, mobility restriction is required for no longer than 1 week. Only the repair of giant hernias sometimes demands prolongation of postoperative artificial respiration until respiratory function has fully recovered.

Technical Pitfalls

Lateral to the rectus sheath and crossing the linea semilunaris, a sufficient overlap of healthy tissue is more difficult to achieve. In such cases, the posterior rectus sheath must be cut in proportions similar to the preparation lateral of the linea semilunaris. Further laterally, the dissection has to create a new preperitoneal space. If this is not feasible, the transverse and internal oblique muscles must be separated. Preservation of the nerves innervating the rectus muscles and running at the dorsal side of the internal oblique is mandatory.

However, sufficient overlap of the ribs sometimes cannot be achieved owing to the insertion of the diaphragm. In cases of extended abdominal wall defects and failure to achieve closure of the fascia in front of the mesh, materials with a tensile strength of > 32 N/cm are recommended to avoid central mesh rupture.

Results from our institution have shown that large-pore meshes with a tensile strength of 16 N/cm are insufficient in such cases if used as a single layer.

Results

Postoperative results are frequently complicated by seroma formation, wound infections, wound discomfort and recurrence. Whereas a sizable seroma is seen in about 30% of the patients, it rarely requires re-intervention apart from intermittent aspiration. However, there are always some few patients with excessive fluid accumulation around the wound who require surgical intervention and removal of the seroma capsule, which may have persisted for months. Infections may be expected in about 10% of the patients. Usually restricted to the subcutaneous space, they should be treated conservatively as common wound infections. Even if the infection encroaches into the mesh itself, a conservative attempt is justifiable, provided the mesh is porous. Late infections appearing after months or even years are more challenging. They are often combined with complex fistulas including bowel [14]. In these cases, preservation of the mesh is likely to fail, and sooner or later most of the mesh has to be removed. After a temporary mesh-free closure, any subsequent mesh repair should be performed no sooner than 6 months later. Moderate complaints after incisional hernia repair are quite common, especially in patients with a long history of previous incisions.Fortunately, the development of a stiff abdomen is rare, although it sometimes requires a mesh exchange [15]. Whether modern large-pore meshes with preserved elasticity can prevent this unpleasant complication is not yet clear.

Summary

Despite recurrences after mesh implantation, the recently published data are encouraging. These series prove the superiority of mesh compared to simple suture repair. In summary, the use of mesh can reduce the recurrence rate from 40 to 50% to about 10% [17]. Even if this effect represents only a delay in the appearance of a recurrence, it reduces morbidity and the rate of re-operation required for re-recurrence. Perhaps an extensive overlap can prolong this delay for the rest of the patient's life. Lacking valid data, mesh should be positioned behind the abdominal wall muscles (sublay technique) using physiological abdominal wall pressure for further fixation of the implant. If (and only if) retromuscular placement cannot be achieved, an onlay implant is justified. In the absence of results from randomized trials, closure of the covering fascia is preferred with non-absorbable suture material. Extended defects of the abdominal wall where the fascia cannot be closed and the mesh is used to bridge the defect must be reinforced by materials with a tensile strength of > 32 N/cm.

Further technical pitfalls mainly refer to anatomy but usually can be answered successfully. Mesh explantation is strictly limited to patients with complex infections or a stiff abdomen.

References

1. Burger JW, Luijendijk RW, Hop WC, Halm JA, Verdaasdonk EG, Jeekel J. Long-term follow-up of a randomized controlled trial of suture versus mesh repair of incisional hernia. Ann Surg 2004; 240(4): 578–583
2. Cassar K, Munro A. Surgical treatment of incisional hernia. Br J Surg 2002; 89(5): 534–545
3. Chan G, Chan CK. A review of incisional hernia repairs: preoperative weight loss and selective use of the mesh repair. Hernia 2005; 9(1): 37–41
4. Conze J, Prescher A, Klinge U, Saklak M, Schumpelick V. Pitfalls in retromuscular mesh repair for incisional hernia: the importance of the "fatty triangle". Hernia 2004; 8(3): 255–259
5. Flum DR, Horvath K, Koepsell T. Have outcomes of incisional hernia repair improved with time? A population-based analysis. Ann Surg 2003; 237(1): 129–135
6. Israelsson LA. The surgeon as a risk factor for complications of midline incisions. Eur J Surg 1998; 164(5): 353–359
7. Jansen PL, Mertens PP, Klinge U, Schumpelick V. The biology of hernia formation. Surgery 2004; 136(1): 1–4

8. Junge K, Klinge U, Klosterhalfen B, Mertens PR, Rosch R, Schachtrupp A et al. Influence of mesh materials on collagen deposition in a rat model. J Invest Surg 2002; 15(6): 319–328
9. Junge K, Klinge U, Klosterhalfen B, Rosch R, Stumpf M, Schumpelick V. Review of wound healing with reference to an unrepairable abdominal hernia. Eur J Surg 2002; 168(2): 67–73
10. Junge K, Klinge U, Rosch R, Mertens PR, Kirch J, Klosterhalfen B et al. Decreased collagen type I/III ratio in patients with recurring hernia after implantation of alloplastic prostheses. Langenbecks Arch Surg 2004; 389(1): 17–22
11. Kingsnorth A, LeBlanc K. Hernias: inguinal and incisional. Lancet 2003; 362: 1561–1571
12. Klinge U, Si ZY, Zheng H, Schumpelick V, Bhardwaj RS, Klosterhalfen B. Abnormal collagen I to III distribution in the skin of patients with incisional hernia. Eur Surg Res 2000; 32(1): 43–48
13. Klinge U, Si ZY, Zheng H, Schumpelick V, Bhardwaj RS, Klosterhalfen B. Collagen I/III and matrix metalloproteinases (MMP) 1 and 13 in the fascia of patients with incisional hernias. J Invest Surg 2001; 14(1): 47–54
14. Leber GE, Garb JL, Alexander AI, Reed WP. Long-term complications associated with prosthetic repair of incisional hernias. Arch Surg 1998; 133(4): 378–382
15. LeBlanc KA, Whitaker JM. Management of chronic postoperative pain following incisional hernia repair with Composix mesh: a report of two cases. Hernia 2002; 6(4): 194–197
16. Luijendijk RW, Hop WC, van den Tol MP, de Lange DC, Braaksma MM, IJzermans JN et al. A comparison of suture repair with mesh repair for incisional hernia. N Engl J Med 2000; 343(6): 392–398
17. Millikan KW. Incisional hernia repair. Surg Clin North Am 2003; 83: 1223–1234
18. Morris-Stiff GJ, Hughes LE. The outcomes of nonabsorbable mesh placed within the abdominal cavity: literature review and clinical experience. J Am Coll Surg 1998; 186(3): 352–367
19. Paul A, Korenkov M, Peters S, Kohler L, Fischer S, Troidl H. Unacceptable results of the Mayo procedure for repair of abdominal incisional hernias. Eur J Surg 1998; 164(5): 361–367
20. Usher F, JL O, Tuttle LJ. Use of Marlex mesh in the repair of incisional hernias. Am Surg 1958; 24: 969–974
21. van 't RM, Steyerberg EW, Nellensteyn J, Bonjer HJ, Jeekel J. Meta-analysis of techniques for closure of midline abdominal incisions. Br J Surg 2002; 89(11): 1350–1356

Discussion

Jeekel: *You talk about bridging. Why don't you put the mesh in and leave it with 6-cm overlap and why do you take so much effort to close the fascia?*
Schumpelick: *I think that bridging is only necessary if you have no other chance of making a sublay or onlay. If you have a big defect and can't close the defect, we call that bridging when you replace the abdominal wall by mesh. We put the mesh behind or in front of the fascia. But this in only necessary in very rare cases with gigantic defects. Usually we try to close this defect.*
Jeekel: *We never close the defect, because you get tension on the muscles again.*
Schumpelick: *We are afraid of blowing the mesh through the defect, when there is no thrust bearing in front of it.*
Jeekel: *In our trial we did not see that in any case. Should we still do a randomized controlled trial on the kind of mesh? Because I do not think we have real problems with heavy polypropylene meshes. Should we also try to do a randomized controlled trial to prove, for example, whether to put it retromuscular or to put the retrorectus sheath on top of the peritoneum or to put it intraperitoneally on top of the omentum?*
Schumpelick: *I think it is not proven which mesh is the best. We should do randomized controlled trials. We need good criteria to describe the effects, not only elasticity of the abdomen, infection morbidity etc.*
Jeekel: *Maybe also in the intraperitoneal position?*
Schumpelick: *In the beginning we tried to put it between rectus sheath and peritoneum, this space is really difficult to get at. Untill now we are reluctant to place meshes within the abdominal cavity, because we do not know how it works. The new meshes may work well in the abdomen.*
Kingsnorth: *First, no one has yet made any mention of the material analysis done by Korenkov, presented at the experts' meeting that took place in 1999, seven studies with sublay and 11 with onlay, and no differences between the results of the two techniques. You have been telling us, that it is the best. But the best compared between what? Because there really are no data to compare, to look at individual series, and one meta-analysis by Korenkov in fact shows a similar quality between the two. The next thing is, the laparoscopic versus open trial in Europe using the sublay as the open method. In America they use the onlay as the open method, because I think that is the best practice for incisional hernia repair. So, we have a difference here between Europe and America, they are using different techniques. My final point is about the mesh; you didn't mention a lot about the data from the trial that was published in December 2005, which was the primary outcome looking at the quality of life and the abdominal wall compliance. There is no difference between the two groups. This scar-plate impression of the polypropylene versus light-weight mesh does not exist, because all the symptoms were the same in both groups. The recurrence rate was three times higher with light-weight mesh. I don't think the case is proven yet. Light-weight mesh is a good concept, but I think it is one thing we need to pursue, and at the moment there is no proof and no comparative data that suggest that it is better.*

Schumpelick: *Yes, you are right. Our randomized multicenter trial did not show the expected results. Recurrences were seen only in 3 of 8 centres, making a technical reason very likely. You are right, so far there is no proven study to show that light-weight meshes are better, but also no proven study shows the opposite.*
Kingsnorth: *Well, is there a randomized study? Lightweight versus heavy-weight? Is there any study existing on the abdominal wall compliance and the quality of life measures? In the Vypro II group, which is a very light mesh against polypropylene mesh there was no difference: this was a randomized trial.*
Schumpelick: *Make some more studies, with a standardized surgical technique and you will see that it works. There is no recurrence by mesh, but by technique. From theoretical point of view, it will be better, but it is only an opinion. I think the onlay technique is simple and feasible, but we fear the problems of subcutaneous infection. We have seen skin necrosis, even in your study. That is a real problem of onlay meshes.*
Itani: *I'd like to make a quick comment about the American trial. When the investigators met to decide which technique to use in the open repair, they were very much aware of the European consensus and about the sublay repair. They decided concisely that, in order not to replicate the laparoscopic repair with the open one, an onlay repair using the Chevrel technique would be very appropriate. So far, we have had very good results. Of course, this study is randomized, so we don't know which one is better, but the investigators have now adopted this repair in their general practice, because they were so happy with it on trial.*
Schumpelick: *We will see the results.*

23.2 Sublay: Incision Crossing the Linea Semilunaris

M. Stumpf, J. Conze, A. Prescher, U. Klinge

Introduction

The treatment of incisional hernias outside the rectus sheath is still a challenging procedure, when done as retromuscular sublay repair. There are an amazingly small number of publications about this field of hernia surgery and therefore any evidence-based data are lacking. It is also impossible to find any guidelines helping to perform such an operation.

All these facts lead to the hypothesis that the repair of incisional hernia outside the midline is a kind of freestyle surgery.

To test this hypothesis and to reject it, we performed some anatomical studies using fresh-frozen corpses to find rules of treatment. We also present some cases of treated patient with different kinds of lateral hernias.

The main problem of sublay repair is to maintain the principle of adequate overlap of the mesh underneath healthy tissue. The challenge regarding lateral hernias is the question, of which anatomical layer has to be used to create the maximal overlap with the minimal side-effects?

In our view, we have to distinguish between two main types of lateral hernias: hernias partly outside the rectus sheath and lumbar hernias.

Hernias Partly Outside Rectus Sheath

If the preparation has to be done below the arcuate line, the lateral extension of the preparation can be done easily on the layer of the preperitoneal space. A mainly blunt dissection is able to create an adequate overlap to both lateral sides. More challenging seems to be the preparation above the arcuate line, e.g. repair of an incisional hernia after subcostal incision. Theoretically, there are two possible layers of preparation. It is possible to dissect between the external and the internal oblique muscle or between the internal oblique and the transverses muscle. ◘ Figure 23.5 illustrates why the anatomical circumstances lead to a preparation between internal oblique and transversal muscle when leaving the rectus sheath laterally. This is, in fact, a useful layer to place the mesh with adequate overlap, if you do not have to extend the preparation into the lumbar region.

If an extended preparation to the lateral side is necessary, a dissection between the two oblique muscles is recommended, because the segmental nerves and blood vessels are lying on the transversal muscle laterally. To reach the layer between external and internal oblique muscle, e.g. repair a hernia after subcostal incision, the dissection should begin at the lateral side and then go further to the lateral border of the rectus sheath.

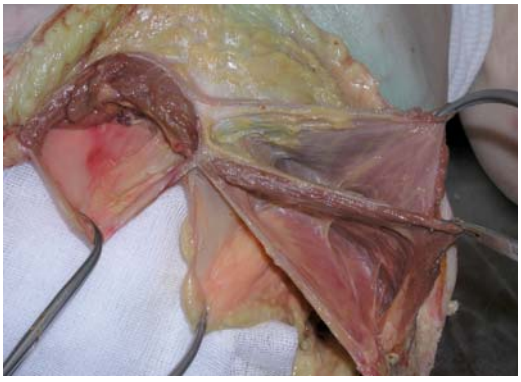


Fig. 23.5. Dissected layers of the lateral abdominal wall

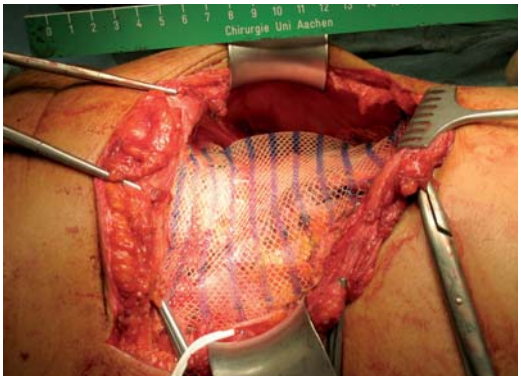


Fig. 23.6. Repair of lateral incisional hernia with Ultrapro mesh placed in retromuscular position between external and internal oblique muscle and posterior rectus sheath

Lumbar Hernia

Repair of a lumbar incisional hernia has to respect the same principles as described above. Because of the segmental nerves positioned on the transversus abdominis muscle laterally, this layer of dissection is not recommended for this hernia type. If there is enough lateral abdominal wall left, a dissection between external and internal oblique muscle can be done easily. After this preparation it is possible to create an adequate medial overlap with incision of the lateral rectus sheath and preparation of the posterior layer to place the mesh (Fig. 23.6). If the hernia has a relevant lateral defect, without adequate muscle and fascia inserting at the crista iliaca, the preperitoneal space has to be used for dissection, to create an adequate lateral overlap underneath healthy tissue. If an extended lateral preparation is done in the preperitoneal space, it has to be certain, that a correct positioning of the mesh, without folding, is guaranteed.

In summary, the repair of lateral hernias has to follow the same principles as the median sublay repair. An adequate mesh overlap has to be assured. With adequate knowledge of the anatomical layers of the abdominal wall this can be achieved for any kind of lateral hernia. Therefore, the hypothesis that repair of incisional hernia outside the midline is "free-style" surgery has to be rejected.

Discussion

Miserez: *If you have a very giant hernia, which is extending outside the semilunar line, then you put your mesh in the lower part of the abdomen, in the preperitoneal space. Now we hear from you, in the upper part, that you should put it between the external and internal oblique. How do you make the connection between both, because the meshes were on two different planes? It is impossible. What is your solution?*

Young: *An option in these situations is to do two layers of mesh with a partial sandwich technique, and go through the muscle, catching the lower edge of the outer layer and the upper edge of the lower layer, which can overlap. They can be on opposite sites of the muscle; but light-weight mesh is better.*

Bendavid: *Why would you bother, when extraperitonally you have a full thickness and have never had difficulties with it? Being in the extraperitoneally space, at any level, it is easy, you have the full thickness of the abdominall wall, when even try to find an intramuscular plane?*

Conze: *The extraperitoneal space is easy to dissect beneath the linea arcuata down behind the pubic bone, but above the linea arcuata the preperitoneal space behind the posterior rectus sheath is very challenging to dissect, and should not be promoted.*

23.3 Closure of a Laparostomy

A. Kingsnorth

Introduction

Closure of a laparostomy is the most challenging operation a hernia surgeon can undertake. Because the abdominal compartments syndrome is a relatively recently recognized phenomenon, the operation of laparostomy to manage this condition is increasing in incidence and the management strategies for closing a laparostomy are evolving [1, 2].

The hernia surgeon should not manage these wounds alone but should work in partnership with intensivists, respiratory physicians, trauma surgeons and plastic surgeons. The surgical skills required for closure of laparostomies are wide-ranging and include a detailed understanding of the anatomy of the anterior abdominal wall, function of its constituent parts, knowledge of the abdominal compartments syndrome and methods of reconstruction of the abdominal wall, including vacuum-assisted closure, free flaps, tissue expansion, and components separation.

Staged Management

Once the decision has been made to omit fascial closure of the abdomen because of the potential risk of abdominal compartments syndrome, phased management should begin at that time, in the operating room [3]. The most important prognostic factor for the success of management of the laparostomy is the surgeon's experience [4]. Other important factors significantly influencing the outcome are patient obesity and size of the hernia. Although staged management is possible, no technique is the "best" solution; knowledge of a wide variety of surgical options will be of benefit [5]. Although laparoscopy is an involving technique for the repair of an incisional hernia, it has no role to play in the management of laparostomy [6, 7].

The aims of the operation should be adequate soft tissue coverage to achieve prevention of visceral eventration, and restoration of abdominal wall function [8]. In the vast majority of cases, laparostomy has been performed through a vertical midline incision, therefore limiting the techniques required to close the defect.

Acute Phase

Shock resuscitation leads to visceral oedema precluding abdominal wall closure. This may be compounded by a retroperitoneal haematoma and packing to prevent haemorrhage. The ideal temporary abdominal wound closure should provide containment of intra-abdominal viscera, protection of the viscera from mechanical injury, prevention of bowel desiccation, minimize abdominal wall tissue damage, prevention of contamination of the peritoneal cavity, control of the egress of peritoneal fluid, rapid application lend itself to multiple applications and be relatively inexpensive [9]. Open packing is generally unsatisfactory because it fails to contain the viscera and results in large quantities of fluid loss. An inexpensive option is the use of a sterilized soft three-litre plastic cystoscopy fluid irrigation bag cut to an oval shape and sutured to the skin or fascial edges of the wound [10]. This method was originally used in Columbia and has acquired the name of the Bogotá bag [11]. The plastic bag holds sutures well, helps retain body heat, minimizes fluid loss, is quick and easy to apply and non-irritant to the viscera. A similar alternative is the use of a temporary abdominal closure with silicone sheeting which also allows acute management of visceral oedema before planned surgery to close the laparostomy defect [12].

Recently, a vacuum-assisted closure method has been successfully used for temporary management of the open abdomen [13]. The original technique described the placement of a perforated polyethylene sheet over the viscera, which was covered with moist sterile surgical towels, and two suction drains were positioned over these followed by the application of a plastic polyester adhesive drape to the wound and skin edges. Following this procedure, suction was applied, which assisted resolution of the visceral oedema and wound repair with minimal tissue damage. In this report Barker and colleagues managed 112 trauma patients and applied 216 vacuum-assisted devices (VAD). Fifty five percent of these patients went on to primary fascial closure during the same hospital admission, with the development of fistulas in 5% of patients and intra-abdominal abscesses in a further 5%. This was a significant advancement on previous strategies, in two respects: first, the number of patients that left the hospital with primary

fascial closure was greatly increased and second, the size and complexity of the residual incisional hernias requiring surgical treatment several months later was reduced. Other reports have confirmed the success of the VAD in shortening hospital stay and preventing re-admission for hernia repair [14, 15]. In a relatively small series, Stonerock and colleagues reported that 67% of patients had successful fascial closure of the abdominal wall within 11 days of initiating use of the VAD. Predictors of successful primary closure included duration of VAD placement (less than 12 days), the total amount of VAD output (less than 3 l), the patient's cumulative fluid balance within the first 2 weeks (less than 2 l) and the presence of systemic infection at the time of attempted closure.

VAD is therefore the temporary abdominal wall closure of choice in the acute phase for patients undergoing open abdominal management.

Intermediate Phase

If fascial closure cannot be completed within 1 to 3 weeks of creating an open abdomen, then various strategies exist to manage this intermediate phase before the final stage of definitive reconstruction. In the intermediate phase, granulation tissue covers the exposed viscera (Fig. 23.7) and two options exist: placement of temporary absorbable mesh or skin grafting. The absorbable mesh placed over the abdominal viscera at this stage may later be pleated to assist in fascial closure [16]. After 2 or 3 weeks, when healthy granulation tissue covers the exposed viscera, the temporary absorbable mesh can be removed and split skin grafts applied to bridge the defect between the skin edges [17]. This effectively results in a planned ventral hernia for which definitive reconstruction could be planned at a later stage. During this intermediate phase there is further resolution of soft tissue oedema affecting the bowel, and other complications such as intra-abdominal sepsis or fistulas can be managed and treated definitively. This intermediate phase was generally of a duration of approximately 6 to 12 months, but in some centres the timing to fascial closure has been reduced to 3 or 4 months [18].

Definitive Closure

The aetiology of the open abdomen correlates with the likelihood of fascial closure [19]. If the laparostomy followed an operation for trauma, the likelihood of fascial closure is greatest. If the operation was for GI sepsis, closure is more likely to be achieved with the utilization of supplementary mesh; and if the original operation was for pancreatitis, definitive closure is less likely. Reconstruction is complicated because the open abdomen has resulted in lateral migration of the rectus muscles, decreased compliance of the oblique muscles, suboptimal skin quality and availability, and the need for enterolysis, possible ostomy reversal and poor pulmonary function [20]. In addition, the reconstructive surgeon may be faced with the difficult removal of skin grafts which are densely adherent to underlying bowel loops (Fig. 23.8).

By far the most useful adjunct for definitive closure of a midline laparostomy is the components separation technique (see below) and success rates are greatly enhanced if plastic and other specialty surgeons are involved in the definitive abdominal wall reconstruc-

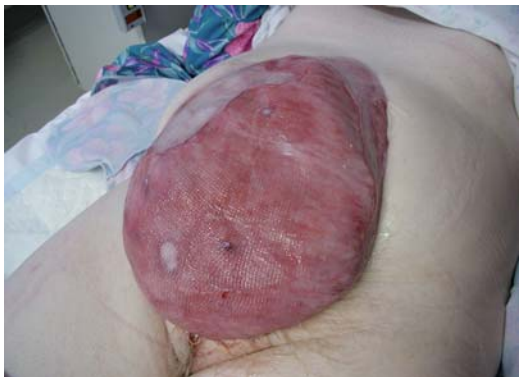


Fig. 23.7. Open laparostomy wound showing mature granulation tissue covering exposed bowel loops

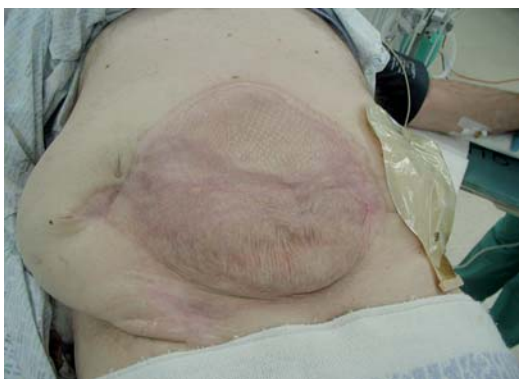


Fig. 23.8. Mature split skin grafts covering bowel loops with widely separated rectus abdominis muscles

tion. For instance, between 5 and 10% of patients will have developed fistulas through the temporarily closed laparostomy wound which will require management by a GI surgeon [16].

Components Separation Method

With this technique, an innervated rectus abdominus – internal oblique – transversus abdominis muscle complex is mobilized bilaterally into the midline [21, 22]. At the waist line a 10-cm advancement on each side can be achieved allowing for closure of 20-cm defects. The external oblique is released 1 to 2 cm from its attachment to the rectus abdominis muscle and separated from the underlying internal oblique in an avascular plane. In addition, the rectus muscle and the underlying anterior rectus sheath can be elevated from the underlying posterior rectus sheath by incision in the midline of its fascia, thus allowing further migration into the midline of the rectus muscles. The skin flaps must be raised to the anterior axillary line. A modification of the technique has been devised for closure of abdominal wall defects in the presence of an enterostomy [23, 24].

In a series of 43 patients deVries Reilingh reported a complicated postoperative course in 17 patients with fascial dehiscence occurring in one, haematoma in five, seroma in two, wound infection in six, skin necrosis in one and respiratory insufficiency in two patients. At 15 months follow-up the recurrent hernia rate was 32% [25]. This high recurrence rate calls into question whether the components separation operation should be supplemented with prosthetic mesh. It is now our practice to supplement this operation with an onlay mesh attached to the two lateral cut edges of the external oblique aponeurosis (Fig. 23.9).

Tissue Flaps

For large defects of the lower abdomen where fascial closure is not possible, a tensor fascia lata (TFL) flap is a useful adjunct [26]. This should be considered where, in addition to a large tissue defect, there is absence of stable skin coverage, recurrence after prior closure attempts, infected or exposed prior mesh or compromised tissues and fistulas. The TFL flap is suitable for reconstructive procedures in the lower abdomen because it has a reliable vascular pedicle and a safe arc of rotation to this zone of the abdominal wall.

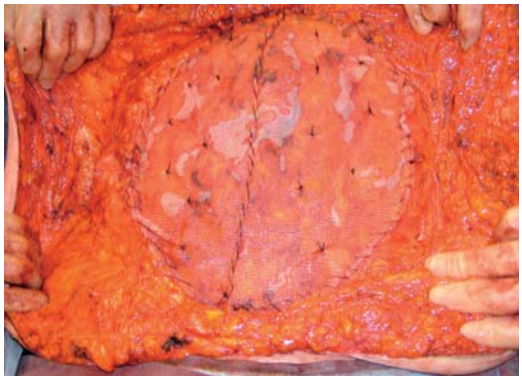


Fig. 23.9. Onlay mesh sutured to cut lateral edges of the external oblique aponeurosis, supplementing components separation closure of a laparostomy wound

Tissue Expansion

Rarely, a subcutaneous or subfascial tissue expander placed between the external and internal oblique muscles may be required before fascial closure can be achieved [27]. The expander is usually required to be in place for at least 3 months and the final volume achieved is usually between 500 to 2000 ml. These patients often require supplementary prosthetic mesh for the definitive procedure.

Mental and Functional Outcomes

Most patients who have undergone life-saving abdominal surgery followed by open abdominal wound management and staged abdominal wall reconstruction experience a stress reaction [18]. However, although these patients experience a decrease in physical, social and emotional health while they are awaiting definitive surgery, their mental health is not affected and after completion of fascial closure, their health status is equivalent to that of the general population. Following surgery, the majority of patients are then able to return to their pre-injury employment.

Conclusion

Closure of a laparostomy requires a multidisciplinary approach which in the intermediate and late phase should be co-ordinated by the hernia surgeon assisted by a team of other specialists. Outcomes are optimized by this team approach.

References

1. Kingsnorth A, LeBlanc K. Hernias: inguinal and incisional. Lancet 2003; 362: 1561–1571
2. Kingsnorth AN, Sivarajasingham N, Wong S, Butler M. Open mesh repair of incisional hernias with significant loss of domain. Ann R Coll Surg Engl 2004; 86: 363–366
3. van Geffen HJAA, Simmermacher RJK, van Vroonhoven TJMV, van der Werken C. Surgical treatment of large contaminated abdominal wall defects. J Am Coll Surg 2005; 201: 206–212
4. Langer C, Shaper A, Liersch T, Kulle B, Flosman M, Fuzesi L, Becker H. Prognosis factors in incisional hernia surgery: 25 years of experience. Hernia 2005; 9: 16–12
5. Dumainian GA, Denham W. Comparison of repair techniques for major incisional hernias. Am J Surg 2003; 185: 61–65
6. Rohrich RJ, Lowe JB. An algorithm for abdominal wall reconstruction. Plast Reconstr Surg 2000; 105: 202–216
7. Gonzalez R, Rehnke RD, Ramaswamy A, Smith CD, Clarke JM, Ramshaw BJ. Components separation technique and laparoscopic approach: a review of two evolving strategies for ventral hernia repair. Am Surg 2005; 71: 598–605
8. van Geffen HJAA, Simmermacher RKJ. Incisional hernia repair: abdominoplasty, tissue expansion, and methods of augmentation. World J Surg 2005; 29: 1080–1085
9. Barker DE, Kaufman HJ, Smith LA, Ciraulo DL, Richart CL, Burns RP. Vacuum pack technique of temporary abdominal closure: a 9-year experience with 112 patients. J Trauma 2000; 48: 201–207
10. Fernandez L, Norwood S, Roettger R, Wilkins HE. Temporary intravenous bag silo closure in severe abdominal trauma. J Trauma 1996; 40: 258–260
11. Feliciano DV, Burch JM: Towel clips, silos, and heroic forms of wound closure. Advances in Trauma and Critical Care, vol 6. Mosby-Year Book, 1991, pp 231–250
12. Howdieshell TR, Proctor CD, Sternberg E, Cue JI, Mondy JS, Hawkins ML. Temporary abdominal closure followed by definitive abdominal wall reconstruction of the open abdomen. Am J Surg 2004; 188: 301–306
13. www.wsacs.org
14. Navsaria PH, Bunting M, Omoshoro-Jones J, Nicol AJ, Kahn D. Temporary closure of open abdominal wounds by the modified sandwich vacuum pack technique. Br J Surg 2003; 90: 718–722
15. Stonerock CE, Bynoe RP, Yost MJ, Nottingham JM. Use of a vacuum-assisted device to facilitate abdominal closure. Am Surg 2003; 69: 1030–1035
16. Jernigan TW, Fabian TC, Croce MA, Moore N, Pritchard FE, Minard G, Bee TK. Staged management of giant abdominal wall defects: acute and long-term results. Ann Surg 2003; 238: 349–357
17. Fabian TC, Croce MA, Pritchard E, Minard G, Hickerson ML, Howell RL, Schurr MJ, Kudsk KA. Planned ventral hernia: staged management for acute abdominal, wall defects. Ann Surg 1994; 219: 643–653
18. Cheatham ML, Safcsak K, Llerena LE, Morrow CE, Block EFJ. Long-term physical, mental and functional consequences of abdominal decompression. J Trauma 2004; 56: 237–242
19. Tsuei BJ, Skinner JC, Bernard AC, Kearney PA, Boulanger BR. The open peritoneal cavity: etiology correlates with the likelihood of fascial closure. Am Surg 2004; 70: 652–656
20. Huttman CS, Pratt B, Cairns BA, McPhail L, Rutherford EJ, Rich PB, Baker CC, Meyer AA. Multidisciplinary approach to abdominal wall reconstruction after decompressive laparotomy for abdominal compartment syndrome. Ann Plast Surg 2005; 54: 269–275
21. Ramirez OM, Ruas E, Dellon AL. "Components separation" method for closure of abdominal-wall defects: an anatomic and clinical study. Plast Reconstr Surg 1990; 86: 519–526
22. Shestak KC, Edington HJD, Johnson RR. The separation of anatomic components technique for the reconstruction of massive midline abdominal wall defects: anatomy, surgical technique, applications, and limitations revisited. Plast Reconstr Surg 2000; 105: 731–738
23. Kuzbari R, Worseg AP, Tairych G, Deutinger M, Kuderna C, Metz V, Zauner-Dungl A, Holle J. Sliding door technique for the repair of midline incisional hernias. Plast Reconstr Surg 1998; 101: 1235–1244
24. Maas SM, van Engeland M, Leeksma NG, Bleichrodt RP. A modification of the "components separation" technique for closure of abdominal wall defects in the presence of an enterostomy. J Am Coll Surg 1999; 189: 138–140
25. deVries Reilingh TS, van Goer H, Rosman C, Bemelmans MHA, deJong D, van Nieuwenhoven EJ, van Engeland MIA, Bleichrodt RP. "Components separation technique" for the repair of large abdominal wall hernias. J Am Coll Surg 2003; 196: 32–37
26. Mathes SJ, Steinwald PM, Foster RD, Hoffman WY, Anthony JP. Complex abdominal wall reconstruction: a comparison of flap and mesh closure. Ann Surg 2000; 232: 586–596
27. Tran NV, Petty PM, Bite U, Clay RP, Johnson CH, Arnold PG. Tissue expansion – assisted closure of massive ventral hernias. J Am Coll Surg 2003; 196: 484–488

Discussion

Franz: *Can you tell us what mesh do you use for this procedure in addition to the components operation technique, and when and why you started to do that? You have shown some pretty white skin flaps. Do you have any problems concerning overlapping that mesh?*

Kingsnorth: *The data that were shown this morning, about my series of onlay, was my very early experience. When I first came to this meeting, and was learning about the Rives technique, I decided that a good incisional hernia surgeon should do sublay, because it was the best operation, and then as I gained experience, people sent me the very challenging hernias. The only way I could do them was with an onlay repair. Sublay was not an option, because of the destruction of the lower abdominal wall. With these really big ones I started doing an onlay and got excellent results. So practically my practice has shifted. I still do sublay for the upper midline incisional hernias, because there you have got a very good posterior rectus sheath.*

I work quite a lot with plastic surgeons, and I noticed that they have particular techniques to use skin flaps. They are not frightened of closing with a small amount of tension. So you never leave a skin flap loose. You should pull the skin flap in the midline where the overlap is and take that skin away. And then you close it quite carefully into two layers. You have a danger of necrosis if, for example, you don't treat the skin around stoma properly, or you don't take regard of where previous incisions have been made. And you don't get a lot of serum formation if you treat with skin flaps, properly. We follow the full Chevrel package, which is relaxing incisions if necessary, usually no incisions in the anterior rectus sheath but Ramirez, and we use tissue glue, fibrin glue. That is the Chevrel package that we use.

Jeckel: *What I learned from a plastic surgeon is to use the tensia fascia lata, which is fantastic, maybe not strong enough in all cases, and you cannot reach far above the umbilicus. But it is sometimes a good technique to use. Do you use Gore-Tex and in that same line more or less Vicryl? Do you use both or was it either/or, because Vicryl gives always a hernia and not Gore-Tex.*

Kingsnorth: *Personally, we actually use a dual mesh now, and we don't use it very often. If we do, we just do a temporary bridging of the mesh. We will actually use a dual mesh and stick it to the fascia. There are options, some stick it to the skin, some stick it to the fascia.*

Schumpelick: *It is very expensive for temporary closure.*

Kingsnorth: *It is, but these are expensive patients.*

Schumpelick: *Why don't you use a Vypro mesh? It works. Did you fix the mesh with glue as Chevrel did, if you make an onlay? Or did you fix it with suture?*

Kingsnorth: *This mesh thing is a question of belief. The glue itself, some people believe, is a wallpaper effect. You glue the wall, and then you put the mesh over it, which is the way they sold it to me. But actually I believe, though I have no evidence for this, I believe that it seals the lymphatics in the skin flaps. I use most of the glue to spray the undersurface of the skin flaps, and the remainder I spray over the mesh itself. Again, I have no evidence.*

Schumpelick: *Is there any contra-indication for you to closing a laparostomy, for example, in a fistula case? And when you have fistulas, do you always close the abdomen or are there any contra-indications?*

Kingsnorth: *Yes, we will close a stoma at the same time as we close laparostoma. We cover patients for 5 days with intravenous antibiotics, and have no major septic problems.*

Sarr: *I have been sitting here and we are talking about hernias. I have not heard anybody talk about infection and chronic mesh infection. I look at your laparostomy closure with these wide flaps in a patient who is compromised. I can't imagine that there is not a high rate of infection.*

Kingsnorth: *Absolutely. When I see these patient it is the first thing I tell them. Their wound will almost certainly not heal primarily. They will get at least a small area of dehiscence, they may have a small area of infection, and I make it quite clear to them. There is a high instance, but considering what we are dealing with, I mean we don't have meshes swimming around and we don't have complete dehiscence of the abdominal wall. But one of the major issues is actually getting a perfect skin closure, which you never get.*

23.4 Onlay

A. Machairas

Introduction

The treatment of choice for a large incisional hernia using open techniques is the tension-free repair with mesh, placing it in the retromuscular-preperitoneal space (sublay technique) or subcutaneously in the prefascial space (onlay technique) [1–4]. Weight loss and optimization of pulmonary and cardiac function are important. Calculation of lung functional volumes and muscle strength, pre-operatively, with the hernia intact and after hernia reduction are very important [1,5,6]. These calculations under protocol can be done intra-operatively at the closure of peritoneum, determining the postoperative respiratory mechanical workload [6].

Open-Onlay Technique

The important steps of the onlay technique are the following [1, 7]:

1. Administration i.v. of antibiotics upon anesthesia induction or 2 h earlier and one more dose 12 h later

(Ampicillin plus Sulbactam/second generation Chephalosporin/ Vancomycin).
2. Skin cleansing and draping.
3. Excision of the skin scar.
4. Identification and preparation of the hernial sac. The skin-cutaneous flaps, rectus abdominis fascia and fascial margins are all prepared.
5. Opening of the hernial sac permits the thorough exploration of the abdominal cavity, checking for sac crypts and safe lysis of intestinal or omental adhesions and subsequent reduction.
6. Excision of the protruding peritoneum (hernial sac).
7. Closure of the hernial gap with complete or partial re-approximation of the rectus abdominis muscles to the midline by peritoneum-fascia adaptation with non-absorbable sutures. This is important depending on the intra-operative assessment of respiratory mechanics.
8. Onlay, tension-free mesh fixation on the anterior rectus fascia, extending 6–8 cm beyond the gap borders in all directions.
9. Tension-free mesh fixation on the aponeurosis by the means of two rows of interrupted non-absorbable sutures, in a 1- to 2-cm distance from each other.
10. Suction drains and trauma closure.

Some reasonable questions are raised about the open-onlay technique/method:

- Is it an easy or a difficult technique? Are there any technical problems concerning the application of this method?
- What about morbidity and mortality?
- What is the incidence of trauma-mesh infection (early complication) and hernia recurrence (late complication)?
- Is this methods comparable to the sublay technique?

There is no doubt that the open-onlay technique is easily performed by a low-experienced surgeon or a senior resident with no need for extensive dissection in the preperitoneal space and blood preparation [1, 7]. In a midline subxiphoidal hernia, mesh fixation with an overlap 6–8 cm in all directions is difficult or impossible because this theoretically will restrict chest mobility. Failed stitch or stitches of the first suture row near the rectus fascia gap may lead to the development of buttonhole hernia (recurrent hernia between the fascia and the mesh). It should be remembered that the main difference between the open-sublay and open-onlay methods is that in the first case the mesh is held in place by the positive intra-abdominal pressure against the closed fascia of the abdominal wall, but in the latter by the stay-anchoring sutures [2, 7]. The incidence of morbidity in open-onlay technique ranges between 4 and 28% and mortality between 0 and 2.7% [2, 7, 8, 9]. The incidence of wound infection ranges between 5 and 16% and the recurrence rate between 2.5 and 11% [3, 7, 8, 10].

Comparisons between the open-sublay and open-onlay techniques are difficult in a high level of evidence-based data for many reasons:

- There are no prospective randomized or controlled studies that have tested the onlay technique versus the sublay technique.
- A small number of operations for incisional hernias are performed, even in large surgical clinics, per year.
- The type of technique, sublay or onlay, is mainly dependent on the surgeon's experience and choice.
- The type of mesh that is used in hernia repair depends on the surgeon's preference, the financial background of the hospital and it may change even within the period of this study [1, 3, 7, 11, 12]. In conclusion, the open-onlay technique is an easily performed and safe method, with an acceptable complication rate (especially wound infection and recurrence). For real comparison, with the open-sublay technique, however, randomized trials or control studies are needed.

References

1. Kingsnorth A, LeBlanc K: Hernias: Inguinal and Incisional. The Lancet 2003, 362: 1561–1570
2. Stumpf M, Conze J, Klinge U, Rosch R, Schumpelick V: Open mesh repair. Eur J Surg 2003, 35: 21–24
3. Korenkov M, Raul A, Sauerland S, et al: Classification and surgical treatment of incisional hernia. Langenbeck's Arch Surg 2001, 386: 65–73
4. Israelsson L: The surgeon as a risk factor for complication of midline incisions. Eur J Surg 1998, 164: 353–59
5. Kavvadia V, Greenough A, Laubscher B, et al: Perioperative assessment of respiratory compliance and lung volume in infants with congenital diaphragmatic hernia: prediction of outcome. J Pediatr Surg 1997, 32: 1665–1669
6. Munegato G, Brandolese R: Respiratory physiology in surgical repair for large incisional hernias of the abdominal wall. J Am Coll Surg 2001, 192: 298–304
7. Machairas A, Misiakos E, Liakakos T, et al: Incisional hernioplasty with extraperitoneal onlay polyester mesh. Am Surg 2004, 70: 726–729
8. Petersen S, Henke G, Freitag M, et al: Erfahrungen mit der reconstruction bauchwadnarbenhernien mittels praeritonealer nach Stopa-Rives. Zentralbl Chir 2000, 125: 152–156

9. Cobb W, Harris J, Lokey J, et al: Incisional herniorrhaphy with intraperitoneal composite mesh: a report of 95 cases. Am Surg 2003, 69: 784–787
10. Luijenndijk R, Hop W, van den Tol P, et al: Comparison of suture repair with mesh repair for incisional hernia. N Engl J Med 2000, 343: 392–398
11. Chevrel J: Hernias and surgery of the abdominal wall. Springer, Berlin Heidelberg New York, 1998
12. Chrysos E, Athanasakis E, Saridaki Z, et al: Surgical repair of incisional ventral hernias: tension-free technique using prosthetic materials (ePTFE Goretex dual mesh). Am Surg 2000, 66: 679–682

Discussion

Simons: *It is better not to go into the abdominal cavity, if it is not necessary, not to do an adhesiolysis, because the adhesions will come back. What is the reason why you do an adhesiolysis when you don't really have to be in the abdominal cavity anyway? Do you agree with me that cutting adhesions means that you will have a recurrence of adhesions?*

Machairas: *Sometimes there is no need to open this up. It is understandable in small hernia defects, smaller than 6 cm in length. The advantage of opening the abdominal cavity is the opportunity to explore the abdominal cavity and to free the borders. Also we can free loops and see, if there are any other defects that cannot be recognized by examining the patient from the outside.*

Deysine: *I observe that your infection rate is about 10%. I know that you inject the patient an antibiotic. What other measures do you take to prevent bacteria from falling from the air into your wound, from your hands and everywhere? Not to create this famous film that would allow them to survive? And would you use any antibiotic locally? And if you don't, why not? Actually the orthopaedic literature is very clear on this, their prosthesis surfaces are smaller than ours. They use irrigational antibiotics, they use intravenous antibiotics to prevent bacterial infection. They have lamina air flow in the operating rooms, and they have a drop in their infection rate from initially about 60% to below 1%.*

Machairas: *No. In the past we used to use antibiotics locally, or hypertonic solutions, like natrium chloride. The committee against infections in the hospital doesn't permit local antibiotics.*

Sarr: *I am going to ask again about wound infections. If there is a 10 or 12% incidence of wound infections, how many of these patients are left with a chronic mesh infection? We are including these mesh infections in our calculation of recurrences.*

Machairas: *In 43 patients we had three wound infections. There was need to remove the mesh in the first 3 weeks in only one patient. The other two patients developed wound infection and we had to remove the mesh 4 and 6 months later.*

Schumpelick: *I have some concern about the skin necrosis, especially these cases with big flaps. I have seen some skin necrosis here; how do you handle that?*

Machairas: *No, we had no skin necrosis. Because we take care to preserve the vessels of the skin.*

23.5 Long-Term Results of Reconstructing Large Abdominal Wall Defects With the Components Separation Method

H.J.A.A. Van Geffen, D. Kreb, R.K.J. Simmermacher, J. Olsman, Ch. Van der Werken

Introduction

Despite better understanding of possible predisposing factors and preventional measures, 10 to 15% of all patients, having had a midline laparotomy, still develop an abdominal wall defect (AWD). Introduction of prosthetic mesh in the repair of these defects has reduced recurrence rates during recent years, but long-term results of reconstruction of large abdominal wall defects remain poor with recurrence rates still up to 44%. Among others, incorrect application of the mesh might be an important factor for this number. Moreover, surgical repair of recurrences is demanding and entails considerable concomitant morbidity in major surgery.

In theory, the goal of any reconstruction of an AWD should be full restoration of abdominal wall function with an intact muscular coverage, prevention of visceral eventration and adequate soft tissue conditions. Various techniques to achieve this have been advocated, but up to now there is still no gold standard for surgical repair of AWDs. Important factors for the choice of technique are the size and site of the defect, availability of viable tissue and degree of contamination. One possible solution for closure of large median AWDs is the use of local

tissue after a tension-relaxing procedure, i.e. components separation method (CSM), first described by Ramirez [1] in 1990. With this technique, the abdominal midline can often be reconstructed in a one-stage procedure without the need of a musculofascial transfer (distant flaps) or the use of prosthetic material.

The purpose of this study is to evaluate our long-term results of large abdominal wall reconstruction by means of the CSM with special regard to recurrences, the influence of contamination and additional use of prosthetic mesh.

Patients and Method

In a 6-year period, we treated 95 patients with large mid-line abdominal AWDs at the University Medical Centre in Utrecht and at the Jeroen Bosch Hospital in 's-Hertogenbosch. Defects exceeding 5 cm in width and 50 cm2 were considered as large. All patients had debilitating symptoms or local conditions which urged surgical intervention (e.g. an AWD with atrophic skin coverage and subsequent imminent enterocutaneous fistulation). Population characteristics are shown in Table 23.1. All operations were planned procedures (no emergencies) and performed by an experienced surgeon under peri-operative antibiotic prophylaxis using amoxicilline/clavulan acid 1200 mg i.v. 30 min prior to incision (repeated after 3 h if necessary). In cases of bacterial peritonitis or drained abscesses antibiotics were continued for 3 days. Bowel preparation was not routinely performed. Contamination was classified according to the National Research Council (NRC) [2]. Previously implanted mesh was removed if possible and the component separation method was performed, as illustrated in Fig. 23.10. After bilateral mobilization of skin and subcutaneous tissues, the aponeurosis of the external oblique muscle was incised pararectally, about 1 cm lateral to the rectus muscle. Then the external and internal oblique muscles were separated by blunt dissection, which is rather easy due to loose connective tissue and the avascularity in this plane (Fig. 23.11). This mobilization was carried out as far as the posterior axillary line in order to facilitate medialization of the rectus abdominus muscle to achieve tension-free closure of the abdominal wall defect.

Due to this extensive dissection, large wound surfaces are created, essentially including the entire ventral abdominal wall. To diminish the risk of skin necrosis and seroma formation we used an alternative approach in three cases: instead of bilateral subcutaneous mobilization starting at the midline, bilateral skin incisions were made directly at the level of the rectus

Table 23.1. Patient characteristics (n = 95)

Age [years]	52
Male/female	48/47
Median body mass index (BMI)	28
Chronic obstructive pulmonary disease (COPD)	13
Previous laparotomies (mean no.)	3
Ostomy	22
Fistulae	19
Skin defect	30
Size of defect (mean size in cm^2)	230 (60–800)
Failed mesh repair	26

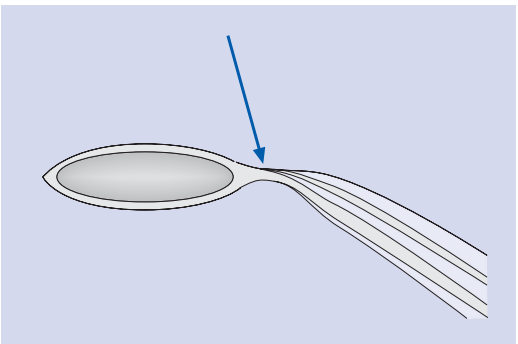


Fig. 23.10. Skin and subcutaneous tissue are mobilized and the aponeurosis of the external oblique muscle incised pararectally, about 1 cm lateral to the rectus muscle

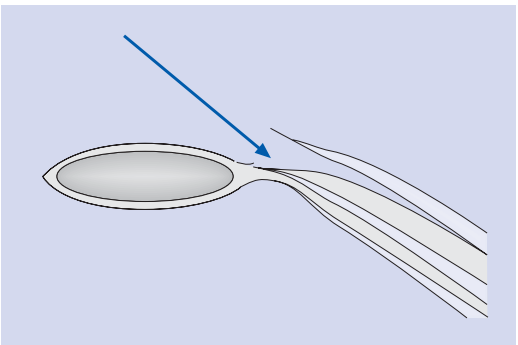


Figure 23.11. The external and internal oblique muscles can be separated by blunt dissection

abdominis/external oblique junction. The separation of the external and internal oblique muscle could then be made directly through this approach. After closure of the linea alba, the consequent lateral skin defects were covered by split-skin grafts. This method was not routinely performed for cosmetic reasons.

In addition, the rectus muscles were separated from the posterior rectus sheath which increases its medialization by several centimetres (Fig. 23.12). This procedure was normally performed bilaterally, but in cases with an ostomy this was only done contralaterally in order to prevent skin necrosis around the ostomy. After excision of the fibrotic fascial edges, the midline was closed with looped PDS (polydioxanone-S, no. 1) in one layer. In 26 randomly chosen defects a non-resorbable prosthetic mesh (18 Mersilene, 6 Prolene and 2 Marlex) was used as augmentation, being fixed with a running PDS suture in the retromuscular space between the rectus muscle and the posterior rectus sheath, with at least 5-cm overlap at all sides (Fig. 23.13). The decision as to whether mesh augmentation was used was strictly at random because these patients participated in different randomized trials. Bilateral suction drainage was used in the subcutaneous space.

The skin was closed with staples in cases with NRC-III contamination. When NRC-IV was encountered, the skin was just covered with a dressing. An abdominal binder was used for 7 days, in order to limit seroma and haematoma formation. We defined postoperative wound infections according to the criteria for surgical site infections of the US Centers for Disease Control [3].

Information was obtained from the patient's general practitioner whether he or she was still alive and had not moved. All patients were invited to visit the outpatient clinic for an interview and physical examination with special attention to recurrences. Patients who were unable or unwilling to travel were visited at their private address by the authors, hereby accomplishing 100% follow-up.

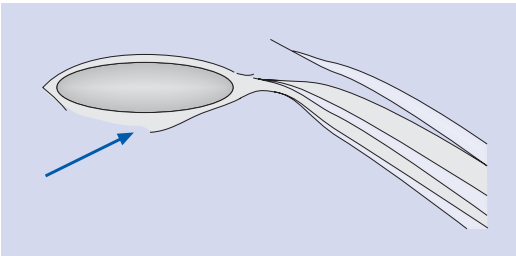


Fig. 23.12. Additionally, the rectus muscle can be separated from the posterior rectus sheath

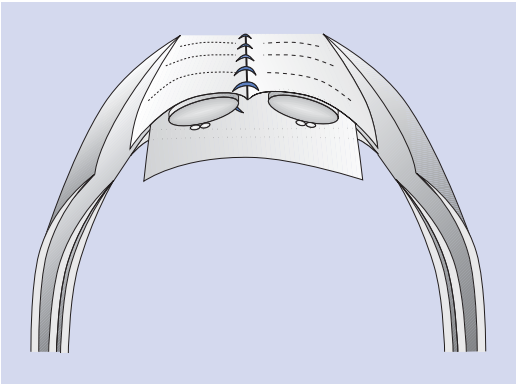


Fig. 23.13. Retromuscular mesh position on the posterior rectus sheath

Table 23.2. Complications of the components separation method

Complications	n
Minor complications	
Superficial wound infection	24
Seroma/haematoma	23
Pneumonia	13
Enterocutaneous fistulae	6
Major complication	
Death	2 (2.1%)

Short-Term Results

Patients were operated in a median operating time of 120 min (30–240) and with a median blood loss of 500 ml (100–2500 ml). In 88% of the operations a bilateral procedure was performed (n = 84) of which more than half without mobilization of the posterior rectus sheath (n = 46). We encountered contamination in 34 operations (36%) of which 4 were NRC-IV contaminated. Patients were hospitalized for a median stay of 11 days (3–201) during which 58% developed one or more complications. Most complications were grade-I and required no intervention. Seven patients (7%) needed a re-operation during hospitalization: four

cases because of progressive seroma formation, two patients due to postoperative hemorrhage. One patient suffered an anastomotic disruption and died after 60 days due to severe peritonitis and multiple organ failure. Another patient died after 4 days due to massive pulmonary embolism.

The most frequent complications are listed in Table 23.2. All wound infections were superficial and could be treated by local drainage and secondary healing of the wound. Most seromas could be aspirated at the surgical ward or in the office.

Long-Term Results

Five patients died during the follow-up period, all by unrelated causes (malignancies four of five). These patients were not excluded from follow-up, but we decided to use their last visit as date of follow-up which ranged from 2 to 5 years. After a median follow-up period of 48 months (1–95) we found 15 patients with a recurrence (15.7%). Thirteen of these patients were asymptomatic and fully satisfied with their situation. Two patients with a recurrence needed a re-operation during which the CSM was performed on the contralateral side (they had had a unilateral CSM initially) in combination with a retrofascial, preperitoneal non-absorbable mesh. We found only one small asymptomatic recurrence (3.8%) in the group with initial mesh augmentation versus 14 (20%) in patients without augmentation (Table 23.3). The only recurrence found in the mesh group concerned a 60-year-old male (BMI = 30), who had a recurrent fascial defect measuring 400 cm2 which was treated with the CSM, augmented by Mersilene mesh. In his postoperative period he developed a wound infection and had prolonged seroma formation. After 40 months we discovered an asymptomatic recurrence of 3 cm in diameter in the most cranial part of the scar, about 2 cm below the xiphoid, possibly due to a technical error.

In six patients we found postoperative entero-cutaneous fistulae. Three of those patients had fistulae pre-operatively and in all three, previously implanted Marlex mesh could initially only be partially removed and all three required re-operation. Three other patients had low-volume fistulae which healed spontaneously, so eventually all patients recovered from fistulae. In seven patients a non-infected skin defect was present but no signs of entero-cutaneous fistulae were observed. Thirteen patients had complaints about the cosmetic result of the operation and 12 patients had used psychological counselling for more than 6 months in the postoperative period.

Table 23.3. Patient characteristics (mesh vs. non-mesh)

n = 95	Component separation method + mesh augmentation	CSM
Patients [n]	26	69
Male/female	13/13	36/33
Age [years]	58	52
Body mass index (BMI)	29	27
Defect size [cm^2]	225	251
Median operation time [min]	115	120
Median total blood loss (TBL) [ml]	500	650
Contamination	5 (19%)	29 (42%)
Wound infection	7	17
Seroma	4	19
Recurrence	1 (3.8%)	14 (20%)

All patients had completed the questionnaire concerning daily life activities. Twenty-nine patients (30%) reported having one or more serious restriction in daily life activities. In most cases this concerned restrictions in heavy weight bearing (n = 22) and/or tying their shoe laces (n = 12).

Discussion

Long-term results of patients treated for massive and often recurrent abdominal wall defects in large series are lacking in the literature. Due to the great variations in aetiology, abdominal wall history and present pathology, treatment has to be tailored individually and often customized in detail, as we stated before [4]. Different

treatment strategies for these patients are therefore difficult to investigate in randomized controlled trials and the optimal treatment for an individual patient cannot be derived from an algorithm.

Although minimal invasive approaches are being used more often for incisional hernia repair in general, most patients with massive and multiple recurrent ventral hernias will need an open reconstruction. Laparoscopic series report recurrence rates up to 17%. Recently, Perrone et al. [5] reported a recurrence percentage of 9.3% in his short-term follow-up of laparoscopic incisional hernia repair in 116 patients. Only 21% of his patients were operated for a recurrent hernia and the mean fascial defect measured 115 cm2. These patients were operated in 157 min, with a conversion rate of 17% and a recurrence rate after 22 months of 8%. One of the specific risks of laparoscopic repair is accidental enterotomy, which may remain undetected during surgery. In this study 11.4% of patients with recurrent ventral hernia suffered from this hazardous complication. All were treated by laparotomy, bowel resection and primary closure of the fascial defect because contamination apparently ruled out an additional procedure. One patient developed multiple organ failure and died.

Because of the alleged high risk of infection in the case of encountered contamination, traditional one-stage reconstruction, with or without mesh, is abandoned and a multistage procedure is chosen [6, 7]. The latter procedure is time-consuming, often not finalized and accompanied by considerable morbidity. However, recent reports suggest that definitive closure despite contamination is successful and does not necessarily exclude the use of synthetic mesh [8–10].

For open reconstruction, abdominal wall defects can be treated either by bridging (with synthetic material or the patient's own tissue) or closure of the abdominal wall after tissue expansion or relaxing incisions [4]. The latter will restore circumferential functional muscular support and (by avoiding the use of mesh) prevent complications of direct contact between non-resorbable mesh and the bowel. The ultimate goal of reconstruction of the abdominal wall is preventing visceral eventration by dynamic, muscular support and adequate soft-tissue coverage. We therefore prefer closure of large, recurrent abdominal wall defects in a one-stage manner by using the components separation method.

Oscar Ramirez [1] first described the possible medial mobilization of the rectus muscle by using his tension-reducing technique. Nahas [11] documented that these relaxing incisions and undermining of the external oblique muscles resulted in a reduction of the necessary force for medial mobilization. In a cadaver study, we additionally found that release of the external oblique muscle produces more benefit to abdominal wall closure than release of the posterior rectus sheath [12].

Few reports have been published about the results of the CSM during the past decade with recurrence rate varying from 5 to 32% [1, 13–18]. Most studies are hampered by either study size or time of follow-up (Table 23.4).

Table 23.4. Results of the components separation method

Author	Year	Study size (no. of patients)	Contamination (no. of contaminated procedures)	Recurrences [%]	Follow-up [months]
Ramirez [1]	1989	11	3	0	?
DiBello [14]	1996	35	15	8.6	22
Girotto [15]	1999	33	3	6.1	21
Shestak [16]	2000	22	?	5	52
Lowe [17]	2000	30	?	10	12
De Vries [18]	2003	43	15	32	15.6
Van Geffen [?]	2006	95	34	15.7	48

These results are in accordance with a recent population-based study of more than 10,000 patients. Flum [19] reported a 12.3% re-operation rate within the first 5 years after initial incisional hernia repair (irrespective of the technique). He also found a 5-year re-operative rate of 23.8% after the first and 35.3% after the second incisional hernia repair.

In this study we analyzed a large series of patients with massive abdominal wall defects treated with the CSM, during a follow-up period of 4 years. At this long-term follow-up we found 15, mostly asymptomatic, patients with a recurrence (15.7%). Most patients could perform daily life activities without limitations, with the exception of heavy weight bearing in 22 patients and tying their shoe-laces in 12 patients (Table 23.5). Theoretically, the abdominal wall can become less flexible after synthetic mesh augmentation and can therefore cause more restrictions in daily life, as opposed to non-mesh repair. However, it was striking to see that patients with postoperative limitations in heavy weight bearing or tying shoe-laces were equally divided between treatment with and without mesh augmentation. In this series, the application of synthetic mesh for augmentation of abdominal wall reconstruction by means of the CSM did not create more limitations in daily life activities.

A large number of patients admitted to have had psychological problems during hospitalization and 12 patients needed psychological counselling for more than 6 months after discharge. All 12 patients held the longevity of their illness, characterized by multiple operations and intensive care admissions, accountable for this.

At the present time, there are no reports on the additional benefit of mesh augmentation during the CSM regarding recurrence. Dibello [14] could not achieve a tensionless repair with the CSM in 15 of his 35 patients. In these cases, reconstruction was augmented by using a resorbable mesh (Vicryl) as an overlay and anchored beyond the semilunar line, but the specific results of these augmented repairs are unknown. Lowe [17] reported the additional use of mesh in 10 of the 30 patients who underwent an open CSM but the indication, position and type of synthetic mesh is unclear as well as the follow-up of this group of patients.

The coincidental (random) use of non-absorbable mesh in a preperitoneal position in our study, for augmentation of the CSM, provided remarkable results. Both groups of patients (with and without mesh augmentation) are comparable, as shown in Table 23.3, with the exception of the amount of contaminated procedures. We found only one small asymptomatic recurrence in the group with initial mesh augmentation (3.8%), versus 14 in patients without augmentation (20%). Analysis with a Fisher's exact test proved this difference to be statistically significant ($p = 0.036$). The latter 14 patients were equally divided between contaminated and non-contaminated procedures. The higher recurrence rate in patients without mesh augmentation, therefore, does not seem to be correlated to the presence of contamination. Although we could not derive it from the operative report, we suspect technical failure to account for the single recurrence in the mesh-augmented group. Careful dissection of the "fatty triangle" and sufficient mesh overlap are essential in this area to prevent a subxiphoidal recurrence, as Conze et al. [20, 21] reported recently.

Table 23.5. Limitations in daily life activities

Patients	Tying shoe-laces	Heavy weight bearing	Total no.[a]
CSM + mesh (n = 26)	3	6	8 (30.7%)
CSM (n = 69)	9	16	21 (30.4%)
Total (n = 95)	12	22	29 (30.5%)

[a]total number of patients with one or more serious limitations.

We conclude that massive and recurrent abdominal wall defects can be safely treated by using the components separation method, given the grotesque pathology. The combination of the CSM with non-absorbable mesh augmentation in the prefascial retromuscular space clearly shows favourable results over mesh-less reconstruction with the CSM. Future investigation in a large prospective randomized trial is needed to validate this finding.

References

1. Ramirez OM, Ruas E, Dellon AL (1990) "Components Separation Method" for closure of abdominal-wall defects: an anatomic and clinical study. Plast Reconstr Surg 86: 519–526
2. National Research Council. Postoperative wound infections. Ann Surg 1964; 160: S1–192.

3. Horan TC, Gaynes RP, Martona WJ, et al. CDC definitions of nosocomial surgical site infections, 1992: A modification of CDC definitions of surgical wound infections. Infect Control Hosp Epidemiol 1992; 13: 606–608
4. Van Geffen HJAA, Simmermacher RKJ. Incisional hernia repair: abdominoplasty, tissue expansion, and methods of augmentation World J Surg 2005 29(8): 1080–1085
5. Perrone JM, Soper NJ, Eagon Chr, Klingensmith ME, Aft RL, Frisella MM, Brunt M. Perioperative outcomes and complications of laparoscopic ventral hernia repair. Surgery 2005, 138(4): 708–716
6. Fabian TC, Croce MA, Pritchard FE, et al. Planned ventral hernia. Staged management for acute abdominal wall defects. Ann Surg 1994; 219: 643–650
7. Jernigan TW, Fabian TC, Croce MA, et al. Staged management of giant abdominal wall defects; acute and long-term results. Ann Surg 2003; 238: 349–355; discussion 355–357
8. Vix J, Meyer CH, Rohr S et al. The treatment of incisional and abdominal hernia with a prosthesis in potentially infected tissues. Hernia 1997; 1: 157–161
9. Campanelli G, Nicolosi FM, Pettinari D, Contessini Avesani E. Prosthetic repair, intestinal resection and potentially contaminated areas: safe and feasible? Hernia 2004; 8: 190–192
10. Van Geffen HJAA, Simmermacher RKJ, van Vroonhoven TJ, van der Werken Chr. Surgical treatment of large contaminated abdominal wall defects J Am Coll Surg 2005, 201(2): 206–212
11. Nahas FX, Ishida J, Gemperli R, Ferreira MC. Abdominal wall closure after selective aponeurotic incision and undermining. Ann Plast Surg 1998; 41: 606–617
12. Van Geffen HJAA, Simmermacher RKJ, Bosscha K, van der Werken C, Hillen B. Anatomical considerations for surgery of the anterolateral abdominal wall Hernia 2004; 8: 93–97
13. Thomas WO, Parry SW, Rodning CB. Ventral/incisional abdominal herniorhaphy by fascial partition/release. Plast Reconstr Surg 1993; 91: 1080–1086
14. Dibello JN, Moore JH. Sliding myofascial flap of the rectus abdominus muscles for the closure of recurrent ventral hernias. Plast Reconstr Surg 1996; 98: 464–469.
15. Girotto J, Ko M, Redett R, et al. Closure of chronic abdominal wall defects: a long-term evaluation of the components separation method. Ann Plast Surg 1999; 42: 385–395
16. Shestak K, Edington H, Johnson R. The separation of anatomic components technique for the reconstruction of massive midline abdominal wall defects. Plast Reconstr Surg 2000; 105: 731–738
17. Lowe JB, Jaime RG, Bowman JL, Rohrich RJ, Strodel WE. Endoscopically assisted "Components Separation Method" for closure of abdominal wall defects. Plast Reconstr Surg 2000; 105: 720–727
18. De Vries Reilingh TS, van Goor H, Rosman C, Bemelmans MH, de Jong D, van Nieuwenhoven EJ, van Engeland MI, Bleichrodt RP. "Components separation technique" for the repair of large abdominal wall hernias. J Am Coll Surg 2003; 196: 825–826
19. Flum DR, Horvath K, Koepsell Th. Have outcomes of incisional hernia repair improved with time? Ann Surg 2003, 237 (1): 129–135
20. Conze J, Prescher A, Klinge U, Saklak M, Schumpelick V. Pitfalls in retromuscular mesh repair for incisional hernia: the importance of the "fatty triangle". Hernia 2004; 8(3): 255–259
21. Conze J, Prescher A, Kisielinski K, Klinge U, Schumpelick V. Technical consideration for subxiphoidal incisional hernia repair. Hernia 2005; 9(1): 84–87

Discussion

Jeekel: *You had a high incidence of pneumonia. What was the reason, just the sick patients? Or was it that you made it too tight? The other question is that the group with the mesh is significantly better in recurrence. Couldn't you just use the mesh instead of the combination, because as I said earlier, you don't need to bridge the gap, you can just use the mesh as the abdominal wall.*

van Geffen: *I honestly do not know what is the best way to do this, because Prof. Schumpelick told us that he puts in a retromuscular mesh and then maybe a relaxing incision. So maybe we do your components separation method, the other way round. We do the components separation method and maybe with a mesh. But I do not know which is the best technique, because an advantage for the retromuscular mesh, at least we think, is the approximation of the rectus muscle and the muscular coverage.*

Jeekel: *This is just what we never do. That is the strange thing.*

van Geffen: *It is, but to me it produces more tension, and probably more pulmonary problems. We have seen a lot of infections with pneumonia, but there is no ratio about that, because there was no tension. They were all tension-less repairs. I do not think that this is the reason for pneumonia.*

Kingsnorth: *Can I ask you where you are getting the recurrences? We have had two, and they both occurred in the area that had been left bare after the component separation. The sublay may not be sufficient in those areas, which is why in that area I use mesh that goes right to the full limits, where you cut the external oblique aponeurosis.*

van Geffen: *With the recurrences I agree with you. There are a lot of recurrences in the lateral side and there where some recurrences in the epigastric area. Of course, theoretically an onlay mesh is covering that, or an inlay mesh is covering these sides to the flanks. I don't know, it could be.*

de Vries: *We had our recurrences in the epigastric region and the cause is probably the fatty triangle. I want to react to Prof. Jeekel. Before we started with our trial we did a trial for large hernias with the Ramirez plastic for prosthetic bridging. We have had to stop this trial, because we had high mesh failure, probably because we did not use the right mesh, we used Gore-Tex mesh and everything became infected.*

23.6 Redo Following Mesh Repair

J. Conze, M. Binnebösel, U. Klinge

Introduction

With a cumulative incidence of about 20%, the repair of incisional hernia following laparotomy remains a perpetual task for general surgeons [5]. Regarding recurrence rates of about 50% after conventional suture repair, a repeat of this technique seems to be insufficient and even obsolete. The introduction of meshes reduced the number of recurrences significantly. In a multicentre, prospective trial with a follow-up of 24 months a recurrence rate of 12.1% was found [2]. In contrast to the crucial importance of a proper wound healing for a successful suture repair, the long-term success of a reinforcing implantation of non-absorbable meshes seems mainly dependent on technical aspects.

Though there are several well-described surgical options for primary mesh repair of incisional hernias, whether open or laparoscopically, there is no literature that addresses the problem of re-operation of failed mesh repair. Mesh failure can mean recurrence but also mesh infection or persisting postoperative pain.

For this reason we analyzed the re-operations after mesh failures that were performed in our surgical department between 1995 and 2004. In this period of time we performed 88 operations following mesh repair. The investigation was complicated due to the different techniques of mesh repair and different type of meshes. Since almost 50% of the primary mesh repairs were performed in other hospitals, the surgical notes were not available in all cases.

In our series, the main reason for a mesh failure was a hernia recurrence (77/88). The different procedures of primary mesh repair mirror the historical advancements of mesh techniques. Different meshes were found in various positions: 46 sublay, 23 onlay, 6 inlay and 2 open IPOM procedures. The primary mesh repair was performed with a heavy-weight, small-pore polypropylene mesh (PPHW) in 31 patients, with a light-weight, large-pore polypropylene mesh (PPLW) in 38 patients, a mesh made from ePTFE in seven patients and polyester mesh (Pol) in one patient.

Type of Mesh Material and Location of the Recurrence

Depending on the mesh size, extent of overlap and mesh position within the abdominal wall, the recurrent fascia defect most often occurred at the mesh border, and only in very rare cases through the mesh (Table 23.6). In patients with PPHW meshes, the allocation of hernia recurrences was located equally on all sides of the mesh. This stands in contrast to the recurrences after PPLW implantation, where the fascia defect occurred almost exclusively at the cranial edge, where the intact linea alba

Table 23.6. Allocation of recurrence position in dependence on the implanted mesh

Location	Cranial			Central			Lateral			Caudal		
Position	Med	Lat	Total	Med	Lat	Total	Med	Lat	Total	Med	Lat	Total
PPHW n = 31	6	3	9	–	–	–	11	4	15	2	5	7
PPLW n = 38	16	8	24	2	–	2	–	7	7	1	3	4
ePTFE n = 7	1	2	3	–	–	–	1	2	3	–	1	1
POL n = 1	–	–	–	–	–	–	–	1	1	–	–	–

prevented a sufficient mesh overlap. In our series only two recurrences through a mesh were described. In both cases the previous repair had been performed with a PPLW.

The recurrences after ePTFE mesh repair also occurred on all sides.

Description of intra-operative adhesiolysis was found also predominantly in patients with PPHW or ePTFE prosthesis, making a small bowel resection necessary in two patients after PPHW implantation (Table 23.7).

Shrinkage of Mesh Area and Adhesion Formation

Due to the retrospective character of this investigation, the degree of shrinkage and adhesion formation was recorded only by the description in the surgical notes, not by detailed measurements. Noticeably, there was an obvious tendency to mesh area shrinkage described for PPHW and ePTFE in more than 50% of the patients, in contrast to the patients after PPLW implantation, where only 5% of the implants showed shrinkage of mesh area. This confirms the experimental results of previous animal investigations, where the different degrees of shrinkage in correlation to the mesh prosthesis have been described [3, 8].

Surgical Procedure at the Revision Operation

In principle, there are three different surgical options to deal with the initial mesh prosthesis following a failed mesh repair.

- Mesh exchange: In the case of mesh deformation and obvious shrinkage of the mesh area, the explantation of the fibrotic modified prosthesis most often becomes inevitable. The following mesh repair should be performed with a large-pore mesh that shows less tendency to mesh area shrinkage, favouring the retromuscular mesh position. In our patients we performed a mesh exchange typically after PPHW and ePTFE meshes (Table 23.8).

Table 23.7. Incidence of mesh area shrinkage and adhesion formation mentioned in the surgical notes

	Shrinkage mentioned		Adhesions		
Mesh	No	Yes	No/little	Remarkable	Severe with, bowel resection
PPHW	42%	58%	74%	13%	13%
PPLW	95%	5%	89%	11%	0%
ePTFE	43%	57%	71%	29%	0%

Table 23.8. Revision operation in dependency of mesh material and incision used

REDO	Exchange			Extension			Explantation and Suture		
Position	Med	Lat	Total	Med	Lat	Total	Med	Lat	Total
PPHW (n = 31)	15	8	23	2	3	5	2	1	3
PPLW (n = 38)	–	4	4	16	12	28	2[a]	4[a]	6
ePTFE (n = 7)	2	1	3	–	1	1	2	1	3
POL (n = 1)	–	–	–	–	1	1	–	–	–

[a]One suture and onlay mesh

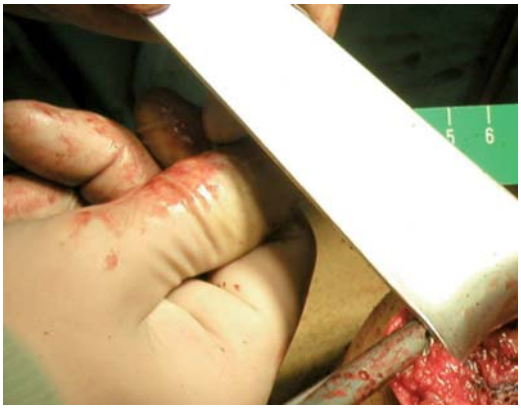


Fig. 23.14. Mesh extension with a second PPLW mesh sutured to the initial mesh prosthesis

- Mesh extension: In the case of a mesh border recurrence at the cranial mesh border, an optimization of the initial repair technique with a sufficient mesh subduction behind the linea alba should be aspired, usually by preparation of the "fatty triangle" or the retroxiphoidal space. In these patients, the initial prosthesis shows no deformation, so it can be left in place and combined with another mesh to facilitate a sufficient mesh subduction (Fig. 23.14).
- The third option is a mesh explantation with abandonment of further mesh material, reducing the repair to a suture procedure.

The surgical decision depends on the initial mesh material and its position within the abdominal wall. Due to extensive fibrotic reaction caused by the initial mesh and/or the considerable tissue trauma after its explantation, it can sometimes be difficult to mobilize the retromuscular space. In these patients, a change of the mesh position, e.g. to the epifascial onlay position, must sometimes be considered. The same principles account for laparoscopic procedures. In expert centres, a re-operation can also be performed by laparoscopy with a mesh extension by a second prosthesis (Fig. 23.15).

In the case of re-operation for infection or persisting postoperative pain, any prior mesh has to be removed. Due to the infection, a new mesh implantation should be avoided and replaced by a standard suture repair with a continuous, non-resorbable suture. Mesh explantation because of persisting complaints was necessary in five patients after PP-small and one patient after ePTFE mesh implantation. On exchange to prostheses that are better adjusted to the physiological elasticity of the abdominal wall, these complaints disappeared.

From our knowledge of re-operations, the reasons for failure after mesh repair are mainly related to technical pitfalls. Most recurrences occur at the border of the meshes near their fixation by surrounding scar tissue. The increasing evidence of a basic defect in wound healing mainly forming collagen of poor quality may lead to an insufficient incorporation of the layers, in so far as meshes can still not improve the quality of collagen [6]. Correspondingly, a too small subduction underneath

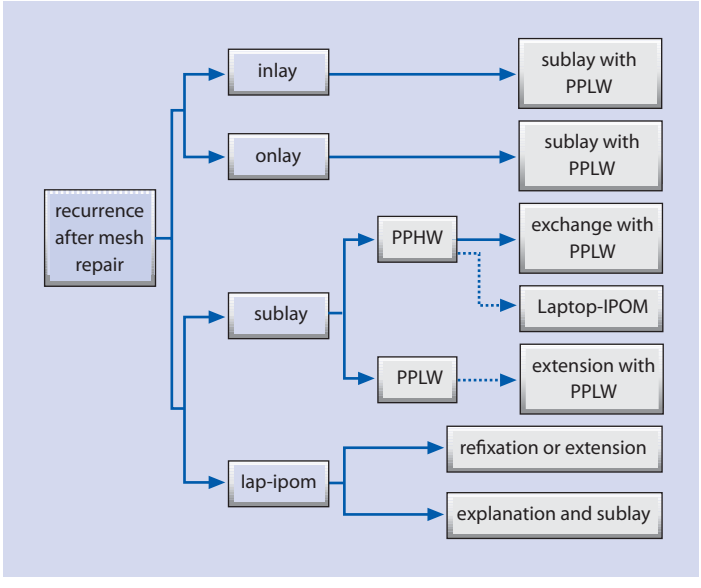


Fig. 23.15. Algorithm for re-operation of recurrent mesh repair for incisional hernia

healthy tissue will accelerate the process of relapse. In contrast, an extended overlap might prevent the recurrence life-long. According to the current literature, an overlap of 5 cm in all directions should be sufficient. Furthermore, a basic defect in scar processing could explain that independently of the intra-operative appearance of recurrence, the entire incision needs to be reinforced in any case. Heavy-weight polypropylene meshes with high tendency to shrinkage seem to encourage a relapse. Our recent insight into the pathogenesis of incisional hernias confirms that even extended scar formations cannot prevent the recurrence if these are of poor quality [7].

The epidemiological data of Flum et al., that showed only a delay of hernia recurrence after mesh repair and long-term results, e.g. Burger et al. with a 10-year cumulative recurrence rate above 30%, underline the importance of this new surgical entity [1, 4].

References

1. Burger JW, Luijendijk RW, Hop WC, Halm JA, Verdaasdonk EG, Jeekel J (2004) Long-term follow-up of a randomized controlled trial of suture versus mesh repair of incisional hernia. Ann Surg 240: 578–583
2. Conze J, Kingsnorth AN, Flament JB, et al. (2005) Randomized clinical trial comparing lightweight composite mesh with polyester or polypropylene mesh for incisional hernia repair. Br J Surg 92: 1488–1493
3. Conze J, Rosch R, Klinge U, Weiss C, Anurov M, Titkowa S, Oettinger A, Schumpelick V (2004) Polypropylene in the intra-abdominal position: Influence of pore size and surface area. Hernia 8: 365–372
4. Flum DR, Horvath K, Koepsell T (2003) Have outcomes of incisional hernia repair improved with time? A population-based analysis. Ann Surg 237: 129–135
5. Hoer JJ, Junge K, Schachtrupp A, Klinge U, Schumpelick V (2002) Influence of laparotomy closure technique on collagen synthesis in the incisional region. Hernia 6: 93–98
6. Jansen PL, Mertens PP, Klinge U, Schumpelick V (2004) The biology of hernia formation. Surgery 136: 1–4
7. Junge K, Klinge U, Rosch R, Mertens PR, Kirch J, Klosterhalfen B, Lynen P, Schumpelick V (2004) Decreased collagen type I/III ratio in patients with recurring hernia after implantation of alloplastic prostheses. Langenbecks Arch Surg 389: 17–22
8. Klinge U, Klosterhalfen B, Muller M, Ottinger AP, Schumpelick V (1998) Shrinking of polypropylene mesh in vivo: an experimental study in dogs. Eur J Surg 164: 965–969

Discussion

Amid: *You mentioned in a case of infection that the mesh has to be removed. Do mean all kinds of meshes?*
Conze: *It was mainly the ePTFE meshes that we had to remove. Very rarely did we have to remove normal-weight mesh, and never light-weight meshes because of infection.*
Amid: *If the mesh is monofilament it really does not need to be removed.*
Deysine: *I enjoyed your presentation very much. Most patients with umbilical hernias in the States, because of the obesity endemic, come with a defect. On top is a triangular defect which is part of the rectus diastasis. My policy is to tell the patients that they have an umbilical hernia that I am going to fix and when it is finished they are going to have a bulge above. That is the way they are going to live, because if you start repairing under the sternum, that is a big operation. So I prefer not to touch it, and most of the patients agree with that.*
Conze: *We all agree that we should not touch rectus diastasis as a primary indication. But if you have an umbilical hernia to repair and it develops rectal diastasis after a year, you have to act and you have to do something.*
Kingsnorth: *Do you think you could assist your problem with this rectus diastasis recurrence by using a non-absorbable stitch to close the posterior rectus sheath? I noticed you used an absorbable stitch. Would you avoid this problem if you used non-absorbable? Why did you use an absorbable stitch for the posterior rectus sheath?*
Conze: *Because the problem that you have is a very broad linea alba. The main tension is on the anterior fascia, on the anterior suture, not on the posterior closure.*
Kurzer: *I did not understand that last point. With umbilical hernias, you are not making a new incision. You are placing a sublay mesh through the umbilical hernia defect, that is correct, isn't it?*
Conze: *If we do mesh repair for an umbilical hernia, and we don't see the necessity very often, than we do a retromuscular, sublay repair.*
Kurzer: *A sublay repair, so you are not making an extra incision, you are using the defect itself. So there is not a posterior sheath to close?*
Conze: *We preparated the fascia defect, incise the rectus sheath on both sides, close the mobilized posterior rectus sheath/hernia sac, and than perform a retromuscular repair, placing the mesh onto the closed posterior rectus sheath. That is where you get to close the posterior rectus sheath.*
Sarr: *For an umbilical hernia with a diastasis rectus why not use a PTFE intraperitoneally. Then you don't have to take down where the posterior rectus comes and joins that attenuated fascia. It could be an ideal place for an intraperitoneal mesh.*
Conze: *If you like to place intraperitoneal meshes, that is an option, yes. But we still hesitate with the intraabdominal position. I don't believe that we have the right material yet for intra-abdominal positioning of meshes or ideal fixation method.*

23.7 Trocar and Small Incisional Hernia

J.A. Halm, J.W.A. Burger, M. van't Riet, J.F. Lange, J. Jeekel

Introduction

Trocar hernia can be defined as the development of a hernia at the cannula insertion site [1]. To this definition Tonouchi and colleagues added the prerequisite that a trocar hernia need not necessarily have a hernia sac (peritoneal covering) [2]. Hernias without a hernia sac are the earliest trocar hernias by the definition of Tonouchi et al., late onset hernias are defined as being contained in a hernia sac [2]. In 2002 Holzinger and Klaiber remarked that a "herniation" at a trocar site without parietal peritoneum should, in fact, be regarded as a mini-platzbauch and not as a hernia [3]. Port-site hernias probably occur after the total spectrum of laparoscopic (minimal invasive) surgery.

The first mention of the problem of trocar herniation must be credited to Fear et al. in their large series of laparoscopy for gynecological diagnosis [4]. Erich Mühe performed the first successful minimally invasive cholecystectomy on September 12, 1985, although Rosen and Ponsky credit Mouret [5]. Six years passed until the first incisional hernia was described after laparoscopic cholecystectomy. Maio and Ruchman must be credited with the first publication exposing incisional hernia, after laparoscopic cholecystectomy, as a problem of minimally invasive digestive surgery. They were also the first to use imaging in the diagnosis of trocar hernia [6].

The rate of incisional hernia after midline incision is commonly underestimated but probably lies between 2 and 20% [7, 42–45]. In The Netherlands alone, 100,000 laparotomies and approximately 4000 incisional hernia repairs are preformed annually (data obtained from Prismant) [7]. The Chevrel classification divides incisional hernias according to size into small (< 5 cm in width/length), medium (5–10 cm in width/length) or large (> 10 cm in width/length) [46]. It is the small hernias that are of interest in this discussion.

Trocar Hernia

Incidence

Investigation of all incisional hernia repairs and the underlying surgery in The Netherlands yielded the following results. A total of 14,526 laparoscopic cholecystectomies were performed in 2001. In 2002 a total of 3853 incisional hernia repairs were performed. It was found that 110 hernia repairs were completed after prior laparoscopic cholecystectomy. The percentage of patients who receive treatment for a trocar hernia after laparoscopic cholecystectomy is 0.8% [7].

Furthermore, five hernia repairs were completed after prior laparoscopic appendectomy. A total of 814 laparoscopic appendectomies were performed in 2001, hence 0.61% of patients received treatment for an incisional hernia after laparoscopic appendectomy [7]. These numbers are the absolute minimum found and the fact that asymptomatic patients may not seek medical attention needs to be appreciated.

Two prospective studies reporting on the incidence of trocar hernia find 1.5 and 1.8% [8, 9], respectively. Mayol et al. based his findings on patients after a variety of procedures (range of follow-up: 3–51 months) and Nassar et al. after laparoscopic cholecystectomy (range of follow-up: 2–6 months). The incidence of trocar-site hernia, taken from literature reporting the complication rates of laparoscopic cholecystectomy, varies between 0.15 [10] and 7.7 [11] (◘ Table 23.9).

Recently, a study describing complications of laparoscopic fundoplication for gastro-esophagal reflux disease reported a trocar site hernia incidence of up to 2.8% [12]. The hernia incidence after transabdominal preperitoneal (TAPP) inguinal hernia repair was reported by Ridings and Evans [11] to be 7.7% and found to be more frequent after TAPP inguinal hernia repair than after TEP surgery by Felix et al. in a series of 1087 patients. A total of six trocar hernias were found (at median follow-up of 42 months), 5 after TAPP (n = 395) and one after converted TEP (n = 692, 14 converted to TAPP) [13].

Trocar Size

The use of large-diameter trocars and cannulas in minimally invasive surgery is often regarded as a predisposing factor for the development of trocar hernia [14–22]. Theoretically, longer incisions, with larger wound surfaces to heal, are at increased risk of wound failure [23]. Based on this theory, small incisions used during laparoscopy (0.5 cm), should be associated with a concomitant small risk of incisional hernia.

Table 23.9. Selection of large prospective and retrospective studies describing trocar hernia incidence

Reference	Study design	No. of patients	Operation(s)	Incidence	Follow-up
Bhoyrul et al. [33]	Randomised; sharp vs. radially expanding	244	Cholecystectomy, hernia, fundoplication, colon surgery, other	0% (sharp) 0% (blunt)	6–18 months
Mayol et al. [8]	Prospective	403	Cholecystectomy, fundoplication, colon surgery, other	1.5%	3–51 months
Nassar et al. [9]	Prospective	870	Cholecystectomy	1.8%	2–6 months
Bowrey et al. [12]	Retrospective	320	Fundoplication	3%	6 weeks-81 months
Azurin et al. [15]	Retrospective	1300	Cholecystectomy	0.77%	Post-operative visit
Ridings et al. [11]	Retrospective (re-usable port changed to disposable port)	1700	TAPP	7.7% (re-usable pyramidal ports) 3.2% (disposable pyramidal ports)	Not reported
Larson et al. [10]	Retrospective	1983	Cholecystectomy	0.15%	Not reported

In a survey of the American Association of Gynecologic Laparoscopists regarding the rate of incisional hernia after laparoscopy, Montz and colleagues found that 725 out of 840 (86.3%) trocar-site hernias occur in locations in which the diameter of cannula used was at least 10 mm. A herniation rate of 2.7% was observed when the diameter of the trocar used was less than 8 mm [24]. The previously mentioned prospective studies by Mayol and Nassar found all hernias except one at 10-mm trocar sites. A single hernia was diagnosed in a 5-mm trocar site [8, 9]. Case reports of five patients, however, have also reported hernias in incisions created by 5-mm trocars after cholecystectomy and fundoplication/Nissen [20, 25–28].

Trocar Type

Numerous trocar designs are available to surgeons these days. The most common trocars are the blunt-conical, pyramidal, radially expanding and cutting dilating types.The trocar-cannula system design has been studied extensively in order to determine the damage inflicted on the abdominal wall during surgery and the number of incisional complications. Experiments in an animal setting have revealed that pyramidal and cutting-dilating trocars require the least force for introduction, yet that both create significantly larger postinsertion defects than blunt trocars [29, 30].

Clinical research suggests that non-cutting trocars reduce the wound surface and thus the consequent risk of developing trocar hernias. In a study of 70 patients in whom blunt conical (muscle splitting) trocars were used, the postoperative defect was found to range between 6 and 8 mm. Of 180 trocar cannula systems placed, 110 were between 10 and 12 mm (61%). None of the defects was closed and no incisional hernias were diagnosed after a median follow-up of 11 months [31]. Leibl et al. demonstrated a difference in trocar hernia rate when comparing sharp and blunt trocars. Sharp trocars were responsible for incisional hernia in 1.83% while the blunt (conical) trocars were to blame for 0.17% [32]. A randomized controlled trial of 244 patients demonstrated no difference in incisional hernia

rates between radially expanding and cutting trocars (see Table 23.10). Hemorrhage, however, was significantly less present in the group of patients randomized for the radially expanding trocar [33].

Location of Entry

Midline sites are the common sites leading to hernia after minimally invasive surgery and umbilical sites are most common [12, 15, 20, 21, 34]. Anatomical considerations are brought forward commenting on the inherent weakness of the para-umbilical region and the use of the largest cannula to facilitate the camera.

From an embryological point of view, the umbilical defect is the fusion of ectoderm and embryonic mesoderm to form the fascial margin of the umbilical ring. To allow the passage of the umbilical arteries and the umbilical vein to the umbilical cord, an abdominal wall "defect" is present from the 3rd week of gestation onwards. After birth, thrombosis of both the arteries and the vein occurs, and thus facilitates contraction of the umbilical ring by cicatrization. Subsequently, the weakest area of the umbilical ring is the superior aspect of it, the area between the umbilical vein and the cranial margin of the umbilical ring. The relative lack of elastic fibres in the obliterated umbilical vein is held responsible for this weakness cranially. In adults, the anatomical margins of the umbilical canal are the umbilical fascia from posterior, the linea alba from anterior and the medial edges of the rectus sheaths.

Azurin and Ahmad hold incidental umbilical hernias responsible for trocar-site hernias [14, 15]. The incidence of para-umbilical and umbilical fascial defects is reported to be 12% in 870 patients undergoing laparoscopic cholecystectomy, the majority of the patients being unaware of the defect (83.7%) [9]. Bowrey described all hernias found in the analysis of laparoscopic fundoplication to be at the midline, open Hassan technique, supra-umbilical initial port [12]. From a questionnaire among the members of the American Association of Gynecologic Laparoscopists, Montz found that 75.7% of all hernias in which the site was noted (n = 152) occurred in the umbilical, the remainder in the flank (23.7%) and at a suprapubic site (0.7%) [24, 35].

Retrieval through Port-Site

Enlargement of umbilical wounds for the retrieval of gallbladders (or other surgical specimens) from the abdomen may be involved in the rate of occurrence of port-site hernias [8]. Nassar and colleagues view the enlargement of the midline (umbilical) fascial defect as the most significant risk factor for trocar-site hernia and advocate avoiding unnecessary wound extension, if possible [9].

Closure of Trocar Site Defect

In order to prevent trocar hernia, authors have advocated closure of all fascial defects after minimally invasive surgery [1, 8, 15, 18, 36–38]. In a study by Kadar, closure of the 12-mm trocar sites significantly reduced the incidence of trocar-site hernias after major laparoscopic gynecological surgery [39]. The authors promote closure of all extra-umbilical fascial defects created by trocars larger than 1 mm and raise the concern that three out of five 12-mm port hernias in their study occurred after closure had been attempted.

Several techniques for the closure of incisions after minimally invasive surgery have been proposed. Di Lorenzo and colleagues propose the use of the Deschamps ligature needle to close defects under direct vision and conclude that the use of the Deschamps needle is straightforward and cost-effective [40].

Petrakis et al. describe a technique utilizing a 15-gauge spinal tap needle and a continuous, non-absorbable suture (USP size 0) for the primary closure of fascial defects as well as the placement of mesh [41].

Conclusion

Laparoscopic surgery is faced with an incidence of incisional hernia of around 2%. Numerous methods have been studied to reduce this incidence. The use of reduced diameter cannula, novel trocar designs and alternative location of entry are brought forward, few of which have been studied prospectively.

Closures of abdominal defects after laparoscopy are discussed in length in the literature. The most commonly suggested factor influencing a surgeon's decision whether or not to close the defect is cannula size. We feel that leaving any fascial defect unclosed is correlated with a higher incidence of trocar-site hernia and that more research, taking into account the type of suture used, perhaps even in the form of a randomized controlled trial, is warranted.

Small Incisional Hernia – Subgroup Analysis of an RCT

Introduction

In 2000, a randomized controlled, multicentre trial performed by our group indicated that mesh repair of incisional hernia is superior to suture repair [47]. The results were confirmed by long-term follow-up of the trial in 2004 [48]. A number of authors previously proposed that there are still indications for suture repair of incisional hernia [49–51]. Upsetting data point out that surgeons are still performing suture repair, in spite of the clinical evidence presented. In 1997, in Germany, 85% of incisional hernias repaired were still performed without mesh [52], while in 1999, in Washington State, 35% of incisional hernias were repaired without the use of mesh [53]. In 2002, Dutch surgeons failed to use mesh in 40% of incisional hernia repairs [7]. The argument that small incisional hernias need not be closed using mesh to achieve excellent results is often heard. We performed this subgroup analysis to explore whether or not this argument is valid.

Patients and Method

From the initial randomized study [47, 48], which included 181 patients, a subgroup of 51 patients was identified. Maximum incisional hernia defect in patient selection was defined to be 10 cm^2. Patient demographics previously recorded in our study included: gender, age, smoking history, presence of prostatism, presence of diabetes mellitus, presence of obstipation, body mass index (BMI) and glucocorticoid use.

Statistical Analyses

Fractions and continuous variables were compared using Fisher's exact test and the Mann-Whitney U test, respectively. The analysis of cumulative percentage of recurrences over time was performed using Kaplan-Meier curves and comparisons were analyzed by the log rank test. Null hypotheses were tested two-sided and a p value of 0.05 or less was considered statistical significant. Statistical analyses were performed using Statistical Package for Social Sciences for Windows (SPSS Inc., Chicago, IL., USA). Hernia recurrence was defined as the primary endpoint.

Results

The 51 patients who formed the study group had a median age of 55 years (range: 23–78 years). The median follow-up was 79 and 66 months for suture repair and mesh repair patients, respectively. Gender, age, body mass index, smoking habits, as well as obstipation, prostatism and COPD were equally distributed between the groups (◘ Table 23.10).

◘ **Table 23.10.** Baseline characteristics and hernia recurrence rate of patients with small incisional hernia (n = 51), according to study group

Variable[a]	Suture repair (n = 30)	Mesh repair (n = 21)
Gender, M:F	1:1.1	1:1.3
Median age, years (range)	67 (25–78)	57 (23–78)
Median BMIb, kg/m^2 (range)	25.3 (20–41.5)	25.8 (20–41.5)
BMI > 30 kg/m^2 [%]	4/30 (13.3)	3/21 (14.3)
Smoking [%]	5/29 (17.2)	8/20 (40)
Prostatism, no. of males [%]	2/13 (15.4)	1/11 (9.1)
Obstipation [%]	4/29 (13.8)	4/20 (20)
Diabetes [%]	0/29 (0)	2/21 (19)
Steroids [%]	1/29 (3.4)	2/20 (10)
Haematoma [%]	3/30 (10)	1/21 (4.8)
Mean intra-operative area of hernia, cm^2 (sd.)	6.5 (3.2)	5.8 (3.5)
Discomfort [%]	9/21 (42.9)	2/12 (16.7)
Recurrence rate [%]	67	17
	p = 0.0029	

[a]Data were not available for all patients; bBMI body mass index

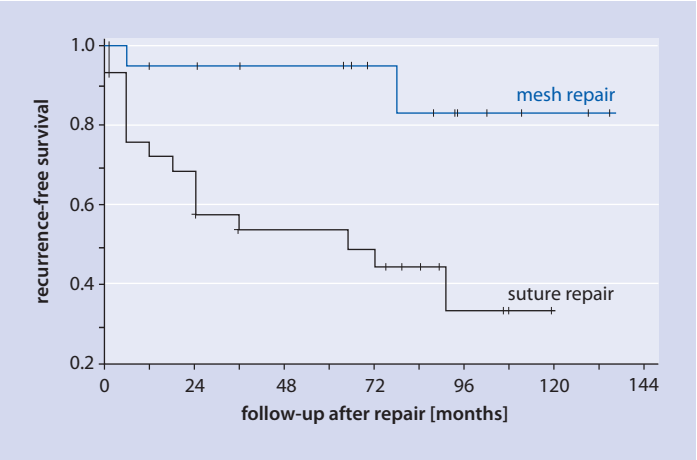


Fig. 23.16. Kaplan-Meier survival curve for recurrence of incisional hernia after repair of a primary or first recurrent small incisional hernia according to study groups. There were significantly fewer recurrences in patients who were assigned to mesh repair (p = 0.0029)

The subgroup analysis of 51 patients with small incisional hernias (≤ 10 cm²) revealed that the 10-year, age adjusted, cumulative recurrence rate was 67% after suture repair, compared with 17% after mesh hernia repair (p = 0.0029; Table 23.10). These aforementioned results are represented in Fig. 23.16.

From our available data (21 suture and 12 mesh-repair patients) scar pain, abdominal pain and discomfort were not significantly associated with either incisional hernia repair method.

Suture and mesh repair patients rated the postoperative cosmetic appearance as 7/10 and 7.5/10 on the VAS, respectively (ns). Fourteen out of 21 patients (67%) after suture repair versus 11/12 (92%) after mesh repair were satisfied after surgery (ns). Dissatisfaction was most commonly caused by hernia recurrence.

Discussion

Subgroup analysis of data collected in a RCT comparing suture and mesh incisional hernia repair provides evidence that mesh repair of small incisional hernias is superior to suture repair on the long run.

The current subgroup analysis reveals that the recurrence rate after suture repair of small (≤ 10 cm²) incisional hernia increases to an undesirable level 10 years after surgery (67%). Although the results of mesh repair are somewhat poor (17% recurrence), a recurrence rate reduction by 75% when not utilizing suture repair is nevertheless a marked improvement. Naturally, all possible complications of mesh repair reported in international literature need to be weighed before the individual patient is treated. This study is the only RCT studying recurrence rates after suture and mesh repair of incisional hernia repair. In order to consolidate the data, new trials are certainly necessary.

Conclusion

In conclusion, the study published by our group is the first and only one to provide prospective long-term follow-up of incisional hernia repair. It proves that mesh repair is superior to suture repair even in small incisional hernias. Mesh repair results in lower, age-adjusted, cumulative recurrence rates without causing significantly more discomfort, abdominal and scar pain or fistula.

References

1. Crist DW, Gadacz TR. Complications of laparoscopic surgery. Surg Clin North Am 1993; 73(2): 265–289
2. Tonouchi H, Ohmori Y, Kobayashi M, Kusunoki M. Trocar site hernia. Arch Surg 2004; 139(11): 1248–1256
3. Holzinger F, Klaiber C. [Trocar site hernias. A rare but potentially dangerous complication of laparoscopic surgery]. Chirurg 2002; 73(9): 899–904
4. Fear RE. Laparoscopy: a valuable aid in gynecologic diagnosis. Obstet Gynecol 1968(31): 297–309
5. Rosen M, Ponsky J. Minimally invasive surgery. Endoscopy 2001; 33(4): 358–366
6. Maio A, Ruchman RB. CT diagnosis of postlaparoscopic hernia. J Comput Assist Tomogr 1991; 15(6): 1054–1055
7. National Medical Registration: http://www.prismant.nl. 2004
8. Mayol J, Garcia-Aguilar J, Ortiz-Oshiro E, De-Diego Carmona JA, Fernandez-Represa JA. Risks of the minimal access approach for laparoscopic surgery: multivariate analysis of morbidity related to umbilical trocar insertion. World J Surg 1997; 21(5): 529–533

9. Nassar AH, Ashkar KA, Rashed AA, Abdulmoneum MG. Laparoscopic cholecystectomy and the umbilicus. Br J Surg 1997; 84(5): 630–633
10. Larson GM, Vitale GC, Casey J, Evans JS, Gilliam G, Heuser L, McGee G, Rao M, Scherm MJ, Voyles CR. Multipractice analysis of laparoscopic cholecystectomy in 1,983 patients. Am J Surg 1992; 163(2):221–226
11. Ridings P, Evans DS. The transabdominal pre-peritoneal (TAPP) inguinal hernia repair: a trip along the learning curve. J R Coll Surg Edinb 2000; 45(1): 29–32
12. Bowrey DJ, Blom D, Crookes PF, Bremner CG, Johansson JL, Lord RV, Hagen JA, DeMeester SR, DeMeester TR, Peters JH. Risk factors and the prevalence of trocar site herniation after laparoscopic fundoplication. Surg Endosc 2001; 15(7): 663–666
13. Felix EL, Michas CA, Gonzalez MH, Jr. Laparoscopic hernioplasty. TAPP vs TEP. Surg Endosc 1995; 9(9): 984–989
14. Ahmad SA, Schuricht AL, Azurin DJ, Arroyo LR, Paskin DL, Bar AH, Kirkland ML. Complications of laparoscopic cholecystectomy: the experience of a university-affiliated teaching hospital. J Laparoendosc Adv Surg Tech A 1997; 7(1): 29–35
15. Azurin DJ, Go LS, Arroyo LR, Kirkland ML. Trocar site herniation following laparoscopic cholecystectomy and the significance of an incidental preexisting umbilical hernia. Am Surg 1995; 61(8): 718–720
16. Bender E, Sell H. Small bowel obstruction after laparoscopic cholecystectomy as a result of a Maydl's herniation of the small bowel through a trocar site. Surgery 1996; 119(4): 480
17. Boughey JC, Nottingham JM, Walls AC. Richter's hernia in the laparoscopic era: four case reports and review of the literature. Surg Laparosc Endosc Percutan Tech 2003; 13(1): 55–58
18. Fitzgibbons RJ, Jr., Annibali R, Litke BS. Gallbladder and gallstone removal, open versus closed laparoscopy, and pneumoperitoneum. Am J Surg 1993; 165(4): 497–504
19. Freedman AN, Sigman HH. Incarcerated paraumbilical incisional hernia and abscess--complications of a spilled gallstone. J Laparoendosc Surg 1995; 5(3): 189–191
20. Plaus WJ. Laparoscopic trocar site hernias. J Laparoendosc Surg 1993; 3(6): 567–570
21. Sanz-Lopez R, Martinez-Ramos C, Nunez-Pena JR, Ruiz de Gopegui M, Pastor-Sirera L, Tamames-Escobar S. Incisional hernias after laparoscopic vs open cholecystectomy. Surg Endosc 1999; 13(9): 922–924
22. Wagner M, Farley GE. Incarcerated hernia with intestinal obstruction after laparoscopic cholecystectomy. Wis Med J 1994; 93(4): 169–171
23. Pollock AV, Greenall MJ, Evans M. Single-layer mass closure of major laparotomies by continuous suturing. J R Soc Med 1979; 72(12): 889–893
24. Montz FJ, Holschneider CH, Munro M. Incisional Hernia Following Laparoscopy: A Survey of the American Association of Gynecologic Laparoscopists. J Am Assoc Gynecol Laparosc 1994; 1(4, Part 2): S23–24
25. Matter I, Nash E, Abrahamson J, Eldar S. Incisional hernia via a lateral 5 mm trocar port following laparoscopic cholecystectomy. Isr J Med Sci 1996; 32(9): 790–791
26. Nakajima K, Wasa M, Kawahara H, Hasegawa T, Soh H, Taniguchi E, Ohashi S, Okada A. Revision laparoscopy for incarcerated hernia at a 5-mm trocar site following pediatric laparoscopic surgery. Surg Laparosc Endosc Percutan Tech 1999; 9(4): 294–295
27. Reardon PR, Preciado A, Scarborough T, Matthews B, Marti JL. Hernia at 5-mm laparoscopic port site presenting as early postoperative small bowel obstruction. J Laparoendosc Adv Surg Tech A 1999; 9(6): 523–525
28. Waldhaussen JH. Incisional hernia in a 5-mm trocar site following pediatric laparoscopy. J Laparoendosc Surg 1996; 6 Suppl 1: S89–90
29. Tarnay CM, Glass KB, Munro MG. Incision characteristics associated with six laparoscopic trocar-cannula systems: a randomized, observer-blinded comparison. Obstet Gynecol 1999; 94(1): 89–93
30. Tarnay CM, Glass KB, Munro MG. Entry force and intra-abdominal pressure associated with six laparoscopic trocar-cannula systems: a randomized comparison. Obstet Gynecol 1999; 94(1): 83–88
31. Liu CD, McFadden DW. Laparoscopic port sites do not require fascial closure when nonbladed trocars are used. Am Surg 2000; 66(9): 853–854
32. Leibl BJ, Schmedt CG, Schwarz J, Kraft K, Bittner R. Laparoscopic surgery complications associated with trocar tip design: review of literature and own results. J Laparoendosc Adv Surg Tech A 1999; 9(2): 135–140
33. Bhoyrul S, Payne J, Steffes B, Swanstrom L, Way LW. A randomized prospective study of radially expanding trocars in laparoscopic surgery. J Gastrointest Surg 2000; 4(4): 392–397
34. Duron JJ, Hay JM, Msika S, Gaschard D, Domergue J, Gainant A, Fingerhut A. Prevalence and mechanisms of small intestinal obstruction following laparoscopic abdominal surgery: a retrospective multicenter study. French Association for Surgical Research. Arch Surg 2000; 135(2): 208–212
35. Montz FJ, Holschneider CH, Munro MG. Incisional hernia following laparoscopy: a survey of the American Association of Gynecologic Laparoscopists. Obstet Gynecol 1994; 84(5): 881–884
36. Callery MP, Strasberg SM, Soper NJ. Complications of laparoscopic general surgery. Gastrointest Endosc Clin N Am 1996; 6(2): 423–444
37. De Giuli M, Festa V, Denoye GC, Morino M. Large postoperative umbilical hernia following laparoscopic cholecystectomy. A case report. Surg Endosc 1994; 8(8): 904–905
38. Velasco JM, Vallina VL, Bonomo SR, Hieken TJ. Postlaparoscopic small bowel obstruction. Rethinking its management. Surg Endosc 1998; 12(8): 1043–1045
39. Kadar N, Reich H, Liu CY, Manko GF, Gimpelson R. Incisional hernias after major laparoscopic gynecologic procedures. Am J Obstet Gynecol 1993; 168(5): 1493–1495
40. Di Lorenzo N, Coscarella G, Lirosi F, Gaspari A. Port-site closure: a new problem, an old device. Jsls 2002; 6(2): 181–183
41. Petrakis I, Sciacca V, Chalkiadakis G, Vassilakis SI, Xynos E. A simple technique for trocar site closure after laparoscopic surgery. Surg Endosc 1999; 13(12): 1249–1251
42. Anthony T, Bergen PC, Kim LT, Henderson M, Fahey T, Rege RV, Turnage RH. Factors affecting recurrence following incisional herniorrhaphy. World J Surg 2000; 24(1): 95–100;discussion 101
43. Manninen MJ, Lavonius M, Perhoniemi VJ. Results of incisional hernia repair. A retrospective study of 172 unselected hernioplasties. Eur J Surg 1991; 157(1): 29–31

44. Paul A, Korenkov M, Peters S, Kohler L, Fischer S, Troidl H. Unacceptable results of the Mayo procedure for repair of abdominal incisional hernias. Eur J Surg 1998; 164(5): 361–367
45. Read RC, Yoder G. Recent trends in the management of incisional herniation. Arch Surg 1989; 124(4): 485–488
46. Korenkov M, Paul A, Sauerland S, Neugebauer E, Arndt M, Chevrel JP, Corcione F, Fingerhut A, Flament JB, Kux M, Matzinger A, Myrvold HE, Rath AM, Simmermacher RK. Classification and surgical treatment of incisional hernia. Results of an experts' meeting. Langenbecks Arch Surg 2001; 386(1): 65–73
47. Luijendijk RW, Hop WC, van den Tol MP, de Lange DC, Braaksma MM, JN IJ, Boelhouwer RU, de Vries BC, Salu MK, Wereldsma JC, Bruijninckx CM, Jeekel J. A comparison of suture repair with mesh repair for incisional hernia. N Engl J Med 2000; 343(6): 392–398
48. Burger JW, Luijendijk RW, Hop WC, Halm JA, Verdaasdonk EG, Jeekel J. Long-term follow-up of a randomized controlled trial of suture versus mesh repair of incisional hernia. Ann Surg 2004; 240(4): 578 583; discussion 583–585
49. Kingsnorth A, LeBlanc K. Hernias: inguinal and incisional. Lancet 2003; 362(9395): 1561–1571
50. Korenkov M, Sauerland S, Arndt M, Bograd L, Neugebauer EA, Troidl H. Randomized clinical trial of suture repair, polypropylene mesh or autodermal hernioplasty for incisional hernia. Br J Surg 2002; 89(1): 50–56
51. Korenkov M, Sauerland S, Paul A, Neugebauer EA. [Incisional hernia repair in Germany at the crossroads: a comparison of two hospital surveys in 1995 and 2001]. Zentralbl Chir 2002; 127(8): 700 704; discussion 704–705
52. Paul A, Lefering R, Kohler L, Eypasch E. [Current practice of incisional hernia reconstruction in Germany]. Zentralbl Chir 1997; 122(10): 859–861
53. Flum DR, Horvath K, Koepsell T. Have outcomes of incisional hernia repair improved with time? A population-based analysis. Ann Surg 2003; 237(1): 129–135

24 Laparoscopical Repair

24.1 Laparoscopic Repair of Incisional Hernias – Reasons for Recurrence

D. Berger, M. Bientzle

Introduction

The incidence of incisional hernias after major abdominal surgery persists at around 20%, implicating the need of a safe and effective technique for repair [7, 23]. Today the need of meshes for augmentation of the abdominal wall is generally accepted because the hernia disease can be explained by a pathological scar formation [16]. The mostly recommended open sublay technique needs separation of the different layers of the abdominal wall, explaining the sometimes high rates of complications reported in the literature [11, 15, 22, 24]. Therefore the laparoscopic approach gains increasing acceptance. It has been generally demonstrated that the rate of wound complications is dramatically decreased after laparoscopic procedures [17, 22, 24, 25]. However, besides the possible complication of unrecognized enterotomy the recurrence rate is still under debate. Since the widespread application of the method the recurrence rates seem to be increasing sometimes exceeding 10% [1–6, 8–10, 13, 17, 22, 26]. The presentation should summarize our experience after more than 600 laparoscopic procedures done for incisional, umbilical, epigastric and parastomal hernias in terms of the reasons for recurrences as well as the treatment of this complication.

Patients and Methods

The demographic data of the prospectively documented patients are shortly summarized in ◘ Table 24.1.

In summary, 425 patients were enrolled in the study between 9/1999 and 9/2005; 94/425 patients with recurrent hernias = 22%, no umbilical/epigastric or parastomal hernias were included!

Technique

The pneumoperitoneum is established by an open access via minilaparotomy. Sometimes the Verres needle is used if an untouched upper quadrant was available. Using three trocars on one and one trocar on the opposite site, an adhesiolysis of the complete abdominal wall is performed using sharp or blunt dissection without any energy-driven device. Fatty tissue such as the falciform ligament or between the plicae mediales is removed to provide safe fixation of the mesh in fascial structures. For suprapubic incisions the space of Retzius is opened and the mesh is fixed at the pubic bones and the symphysis. Generally the whole original incision is covered by the mesh. The overlap should exceed the incision

Table 24.1. Summary of patient data

	Median	Range
Hospital stay [days]	8	4–64
Operating room time [min]	85	30–220
Hernia size [cm²]	110	1–600
Mesh size [cm²]	430	25–884
BMI	30	18–54
Age	65	24–88
Observation period [months]	24	3–72

by at least 5 cm in all directions. The mesh is fixed by non-resorbable stay sutures at the corners and spiral tacks. ePTFE, Dualmesh®, was used between 9/1999 and 12/2002. Covered polypropylene meshes were introduced in 2003. Since May 2004 we have generally used Dynamesh IPOM®, which represents a real mesh structure allowing the use of multiple meshes overlapping each other. The mesh is made of polyvinylidene fluoride with a minor amount of polypropylene on the parietal side which provides efficient incorporation in the abdominal wall. In the meantime we have used Dynamesh IPOM in more than 200 patients with all kinds of ventral hernias without any adverse effects.

Results

Up to now we have observed ten recurrences corresponding to 2.4%. Five of them were localized in the suprapubic region, which can be easily explained by fixation without removing the fatty tissue between the plicae mediales and opening the space of Retzius. Therefore the mesh was fixed only in the fatty tissue and could slip upwards, releasing the original fascial gap. The re-repair was generally done by a conventional sublay procedure.

Two further patients developed a recurrence after small midline incisions due to asymmetric fixation of the mesh. In both cases the right margin was completely fixed with tacks by pressing the abdominal wall against the tacker. After releasing the abdominal wall the mesh moved to the right and the overlap on the left side was reduced. In combination with a small mesh and primarily small overlap of the fascial gap a recurrence must occur. Both patients underwent laparoscopic re-repair with removal of the original mesh.

A central dislocation of the mesh was observed in two patients leading to recurrences. In one patient the mesh was centrally thinned, implying real mesh failure. However, the ePTFE mesh of the second patient was dramatically shrunken, it had lost 56% of its original surface! Therefore the combination of shrinkage and dislocation due to inadequate fixation may explain the recurrences in both cases. One patient was treated by laparoscopic removal of the original mesh and laparoscopic re-repair. The other patient underwent open sublay repair.

One further subcostal recurrence occurred. Again, the ePTFE mesh had lost 63% of its original size. Obviously, the overlap in the upper abdomen was too small and combined with inadequate fixation. This patient underwent laparoscopic re-repair by inserting a mesh with a broad overlap to the diaphragm and overlapping the ePTFE mesh.

In summary, all recurrences were observed in our series with ePTFE meshes. All patients treated with covered polypropylene and polyvinylidene fluoride meshes have been recurrence-free up to now.

Ninety-four patients of our series suffered from a recurrent hernia after conventional repair. No conversion was necessary in these patients and only one suprapubic recurrence has been observed up to now. The laparoscopic technique was essentially the same as described above. The original meshes, if used, were left in place and the whole incision was covered by the IPOM.

Discussion

As recently reviewed by LeBlanc, the recurrence rates after laparoscopic repair of incisional hernias are reported in the literature to range from 1 to 16% [17]. Unfortunately, the exact reasons for recurrences have been rarely defined. Heniford et al. [13] found recurrences associated with only stapling the mesh. As expected, the hernia recurred after mesh removal due to infection. But the suggested reason could be defined in only 14 out 35 patients with recurrences. Statistical evaluation revealed the size of the defect, the mean operating time, the presence of previous failed repairs, and the occurrence of postoperative complications as independent risk factors for recurrence after laparoscopic repair.

LeBlanc et al. defined the coverage of the entire incision and the amount of the overlap as the main aspects for prevention of recurrences [18, 19]. His observations are supported by our experience. All our recurrences can be explained by inadequate overlap. Over the years the mesh size increased in the series of LeBlanc as well as in our series. The recurrence rate of LeBlanc dropped from 9 to 4% with increasing mesh size [20]. As outlined above, our recurrences have been observed in patients treated with ePTFE which is known to shrink more than other materials [14, 21]. Using bigger meshes with a lower shrinkage rate, no further recurrence occurred. The new mesh material, which possesses a real pore structure, allows the application of multiple meshes overlapping themselves, which is impossible for ePTFE and is not studied for covered polypropylene meshes.

The laparoscopic treatment of recurrent hernias after a conventional repair seems to be an effective approach with a very low complication and recurrence rate, as shown above in 94 patients of the own series.

In summary, the laparoscopic treatment of primary or recurrent incisional hernias seems to be an effective technique concerning the recurrence rate. However, some preconditions must be strictly followed:

1. Coverage of the whole incision.
2. Broad overlap, at least 5 cm.
3. Adequate mesh material with no or only little shrinkage.
4. Adequate fixation with sutures and tacks if necessary at the pelvic bone or the costal arch.

Therefore the limitation of the laparoscopic technique is clearly given by the defect size. If an overlap of at least 5 cm is not possible, another technique may be advisable which allows the reconstruction of the abdominal wall such as the component separation technique [12, 27].

References

1. Aura T, Habib E, Mekkaoui M, Brassier D, Elhadad A (2002) Laparoscopic tension-free repair of anterior abdominal wall incisional and ventral hernias with an intraperitoneal Gore-Tex mesh: prospective study and review of the literature. J Laparoendosc Adv Surg Tech A 12: 263–267
2. Bageacu S, Blanc P, Breton C, Gonzales M, Porcheron J, Chabert M, Balique JG (2002) Laparoscopic repair of incisional hernia: a retrospective study of 159 patients. Surg Endosc 16: 345–348
3. Bencini L, Sanchez LJ, Scatizzi M, Farsi M, Boffi B, Moretti R (2003) Laparoscopic treatment of ventral hernias: prospective evaluation. Surg Laparosc Endosc Percutan Tech 13: 16–19
4. Berger D, Bientzle M, Muller A (2002) Postoperative complications after laparoscopic incisional hernia repair. Incidence and treatment. Surg Endosc 16: 1720–1723
5. Bower CE, Reade CC, Kirby LW, Roth JS (2004) Complications of laparoscopic incisional-ventral hernia repair: the experience of a single institution. Surg Endosc 18: 672–675
6. Carbajo MA, Martin dOJ, Blanco JI, de la Cuesta C, Toledano M, Martin F, Vaquero C, Inglada L (1999) Laparoscopic treatment vs open surgery in the solution of major incisional and abdominal wall hernias with mesh. Surg Endosc 13: 250–252
7. Cassar K, Munro A (2002) Surgical treatment of incisional hernia. Br J Surg 89: 534–545
8. Chari R, Chari V, Eisenstat M, Chung R (2000) A case controlled study of laparoscopic incisional hernia repair. Surg Endosc 14: 117–119
9. Chowbey PK, Sharma A, Khullar R, Mann V, Baijal M, Vashistha A (2000) Laparoscopic ventral hernia repair. J Laparoendosc Adv Surg Tech A 10: 79–84
10. Eid GM, Prince JM, Mattar SG, Hamad G, Ikrammudin S, Schauer PR (2003) Medium-term follow-up confirms the safety and durability of laparoscopic ventral hernia repair with PTFE. Surgery 134: 599–603
11. Gilbert AI, Graham MF (1998) Problems associated with prosthetic repair of incisional hernias. Arch Surg 133: 1137
12. Gonzalez R, Rehnke RD, Ramaswamy A, Smith CD, Clarke JM, Ramshaw BJ (2005) Components separation technique and laparoscopic approach: a review of two evolving strategies for ventral hernia repair. Am Surg 71: 598–605
13. Heniford BT, Park A, Park AF, Ramshaw BJ, Voeller G, (2003) Laparoscopic repair of ventral hernias: nine years' experience with 850 consecutive hernias. Ann Surg 238: 391–400
14. Johnson EK, Hoyt CH, Dinsmore RC (2004) Abdominal wall hernia repair: a long-term comparison of Sepramesh and Dualmesh in a rabbit hernia model. Am Surg 70: 657–661
15. Klinge U, Conze J, Krones CJ, Schumpelick V (2005) Incisional hernia: open techniques. World J Surg 29: 1066–1072
16. Klinge U, Si ZY, Zheng H, Schumpelick V, Bhardwaj RS, Klosterhalfen B (2001) Collagen I/III and matrix metalloproteinases (MMP) 1 and 13 in the fascia of patients with incisional hernias. J Invest Surg 14: 47–54
17. Le Blanc KA (2005) Incisional hernia repair: laparoscopic techniques. World J Surg 29: 1073–1079
18. LeBlanc KA (2001) The critical technical aspects of laparoscopic repair of ventral and incisional hernias. Am Surg 67: 809–812
19. LeBlanc KA, Booth WV, Whitaker JM, Bellanger DE (2001) Laparoscopic incisional and ventral herniorraphy: our initial 100 patients. Hernia 5: 41–45
20. LeBlanc KA, Whitaker JM, Bellanger DE, Rhynes VK (2003) Laparoscopic incisional and ventral hernioplasty: lessons learned from 200 patients. Hernia 7: 118–124
21. McGinty JJ, Hogle NJ, McCarthy H, Fowler DL (2005) A comparative study of adhesion formation and abdominal wall ingrowth after laparoscopic ventral hernia repair in a porcine model using multiple types of mesh [In Process Citation]. Surg Endosc 19: 786–790
22. McGreevy JM, Goodney PP, Birkmeyer CM, Finlayson SR, Laycock WS, Birkmeyer JD (2003) A prospective study comparing the complication rates between laparoscopic and open ventral hernia repairs. Surg Endosc 17: 1778–1780

23. Millikan KW (2003) Incisional hernia repair. Surg Clin North Am 83: 1223–1234
24. Olmi S, Magnone S, Erba L, Bertolini A, Croce E (2005) Results of laparoscopic versus open abdominal and incisional hernia repair. JSLS 9: 189–195
25. Robbins SB, Pofahl WE, Gonzalez RP (2001) Laparoscopic ventral hernia repair reduces wound complications. Am Surg 67: 896 900
26. Sanchez LJ, Bencini L, Moretti R (2004) Recurrences after laparoscopic ventral hernia repair: Results and critical review. Hernia 8: 138–143
27. Vries Reilingh TS, van Goor H, Rosman C, Bemelmans MH, de Jong D, van Nieuwenhoven EJ, van Engeland MI, Bleichrodt RP (2003) "Components separation technique" for the repair of large abdominal wall hernias. J Am Coll Surg 196: 32–37

Discussion

Schippers: *It is just one technical point. You analyzed your recurrences and you mentioned there was one mesh failure. But you didn't specify. What is that mesh failure?*

Berger: *That was the one patient with a protrusion of central dislocation of the mesh. When we removed the mesh we found that it was quite thinned at the central part. It was thick as normal on the lateral part, but very thin in the central part. Nevertheless, it was a recurrence, and I think it was due to a technical mistake.*

LeBlanc: *I found in a lab that even the dual meshes always shrink 60%, which is what you demonstrated. For this reason we have accounted for a larger and larger overlap over the years. The other thing is, that we use suture fixation, and in the first 100 cases, the only recurrences we had were those we did not fixate. Why do you switch to different meshes?*

Berger: *Because of the price and because I was convinced in 2003 that the Parietene-composite mesh was a good mesh for intraperitoneal placement. The PVDF mesh now is elastic and is a real mesh structure. So I can use more than one mesh overlapping each other, which I think is not very good for ePTFE. Since that time it is quite astonishing how often I need more than one mesh for repairing the defect. I like to overlap two meshes in order to get a very big overlap to the costal arc and down to the symphysis.*

LeBlanc: *If you go below the costal arc, how do you fix the mesh to the diaphragm?*

Berger: *I do not fix it to the diaphragm; I fix it to the costal arc, to the ribs. That makes pain, of course, but only for a few days. I don't fix the mesh at the diaphragm, because that is not necessary if it is adequately fixed at the costal arc. The pressure of the liver and the stomach and spleen will do the rest in preliminary fixation until it is incorporated.*

Schumpelick: *An amount of shrinkage of more than 60% is a lot. Is it really shrinkage, or is it folding up and rolling up?*

Berger: *We have removed the mesh and retried to cut out everything of tissue of the mesh. It was not possible to get any enlargement of the shrinkaged mesh. That was the real size of the mesh at that time. There was only a little folding, but not very strong to explain this large amount of shrinkage of that mesh.*

24.2 The Local Patch

M. Miserez, K. Tomczyk, F. Penninckx

Is it Necessary to Cover the Whole Incision in the Case of a Well-Circumscribed Incisional Hernia?

The reappearance of "another" hernia after previous repair can be due to a true recurrence, a new hernia formation or the clinical manifestation of a missed hernia. The question on how this could be prevented differs according to the situation.

In the case of a missed hernia, the question is: could this have been prevented by a better (pre- or intra-operative) examination? Therefore the physical examination by the surgeon him- or herself immediately or shortly before the surgical intervention is extremely important. Apart from the localization of the hernia defect and the necessary mesh overlap, especially with respect to the bony edges (xiphoid, costal margin, pubis), the exact delineation of any bulging, rectus diastasis or other abdominal wall hernia formation (previous incisions, epigastrium, umbilicus, groin etc.) is necessary. This is even more important in laparoscopic repair, where the installation of the patient and the positioning of the trocars are influenced by these factors. The patient should be examined in supine and erect position. Radiological

Table 24.2. Comparison of mesh size vs. hernia size for different hernia defects

Hernia size [cm]	Mesh size [cm] (5 cm overlap)	Mesh-size-to-hernia-size ratio
2 x 2	12 x 12	36
3 x 3	13 x 13	19
5 x 5	15 x 15	9
7 x 7	17 x 17	6
10 x 10	20 x 20	4
10 x 3	20 x 13	9
10 x 5	20 x 15	6

examinations such as ultrasound or CT scan can be very helpful especially in obese patients. In any case, intraoperative exploration of the whole scar is the gold standard, since Swiss-cheese defects have been described in almost one third of patients [1]. In most cases, this can be accomplished ideally by laparoscopy.

Could this have been prevented by a better technique in the case of a recurrent hernia (i.e. at the previous hernia site or at the edges of it)? The key to success in any hernia repair is always to use a large mesh centred over the defect and bulging around it, with a wide overlap of at least 5 cm circumferentially, and adequate fixation. There is still a lot of controversy in the literature concerning the ideal type of fixation (transfascial sutures vs. fixation devices) [2].

Table 24.2 shows some common hernia defects and mesh size to hernia size ratio using a mesh with circumferential overlapping of the defect by 5 cm. The range of this ratio in the examples varies from 4 to 36. It might be that a systematic 5 cm overlapping is not necessary in smaller hernias, whereas more overlapping is needed in larger hernias. Therefore, it might be that this ratio is a better parameter to determine the adequate mesh size. The question on the ideal ratio remains, however, unanswered and is probably between 6 and 9.

Could this be prevented by a larger mesh covering the whole incision (supposing this had not been done previously) in case of a new hernia? Figure 24.1 depicts our current vision about the possible mechanisms for a new hernia development and the interaction between technical failure and underlying connective tissue disorders. Some believe a hernia is in the first place the result of a technical failure, whereas others say it is mainly the result of a connective tissue disorder resulting in a biological defect in scar tissue formation, the so-called hernia disease [3]. The truth probably lies in between, with different proportions playing a role in different subgroups. Other exogenous factors can be obesity and other causes of chronic increased intra-abdominal pressure.

Some authors have put forward arguments for a systematic coverage of the whole previous incision [4–6]. Arguments pro are the decreased recurrence rates postoperatively due to systematic prevention of a new hernia which might otherwise arise as a result of the proposed connective tissue disorder. Moreover, this strategy allows the systematic treatment of missed (single or multiple) small hernias. Arguments contra are that this might be overtreatment in some cases with other potential complications due to a more extensive adhesiolysis and larger mesh.

Of course, in some circumstances the decision is relatively straightforward. Figure 24.2 shows two patients with a localized and well-circumscribed abdominal wall defect without additional bulging or diastasis; Fig. 24.2a shows a patient where coverage of the whole previous incision in the epigastrium requires a mesh of only 2 cm longer, whereas Fig. 24.2b shows

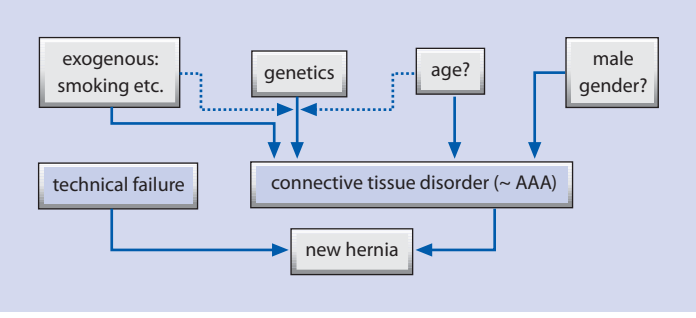


Fig. 24.1. Possible mechanisms for a new hernia development (*AAA* aneurysm of the abdominal aorta)

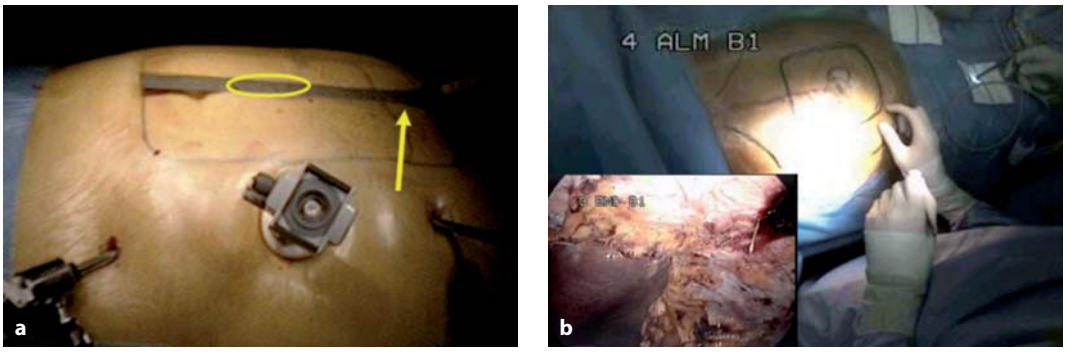


Fig. 24.2. a Patient with supra-umbilical incisional hernia and the previous incision extending into the epigastrium (arrow). **b** Patient with peri-umbilical incisional hernia where the epigastric part of the previous incision is covered by dense adhesions of the liver (inset)

a patient where most surgeons would probably not reinforce the entire incision because of dense adhesions between the liver and the abdominal wall.

In addition, what should be the attitude with respect to other incisions in the vicinity of the hernia defect, e.g. post-appendectomy scars, without any weakness? Should they be covered, and what should be the mesh overlap in these cases without any hernia? It is also clear that in some of these cases the positioning and fixation of such a large mesh or different meshes might be difficult (laparoscopically).

In conclusion, for the time being, we use a tailored approach based on different parameters to determine the need for the coverage of the whole incision in a patient with an incisional hernia:

- Thorough clinical examination preoperatively to exclude rectus diastasis or bulging.
- Intra-operative exploration of the whole scar with minimal morbidity.
- Patient-specific parameters favouring systematic coverage of the previous incision: young age, short interval since the primary surgery (since most incisional hernia recurrences develop in the first 3 years postoperatively [7, 8]), no major extent of the incision (in the case of open repair), minimal adhesions or easy to lyse (in the case of laparoscopic repair) and presence of possible risk factors for a connective tissue disorder such as smoking and aortic aneurysm [8–10].

Indications for Laparoscopic Repair of a Recurrent Incisional Hernia

Even in cases where conversion to an open repair is preferred, a diagnostic laparoscopic approach to recurrent incisional hernia as a first step in the operative procedure can be rewarding to explore the scar for other hernias and, after previous intra-abdominal mesh repair, to determine the mechanism of recurrence (mesh dislocation, mesh shrinkage etc.) and the extent and tenacity of adhesions to the mesh and/or fixation devices. The latter will also be a major determinant in the decision to perform a (new) laparoscopic repair.

For treatment after previous open (= anterior) repair, laparoscopy has definitely a place since it allows repair of the abdominal wall defect through a different (= posterior) approach, following the same concept as in groin hernia repair.

Although a new laparoscopic approach after previous laparoscopic repair is feasible, some specific points need to be taken into account: the risks of the necessary adhesiolysis must be weighed against the potential benefits of the laparoscopic repair; furthermore, mesh-mesh overlap intra-abdominally needs permanent suture fixation in order to diminish the risk for recurrence at the mesh-mesh interface. In our opinion, a relative contra-indication to a laparoscopic repair is the giant hernia with a transversal hernia orifice diameter of more than 10 cm. We believe that in these patients there is a higher risk of abdominal wall bulging and/or recurrence. Indeed, large fascia defects require some type of closure or approximation anterior to the (intra-abdominal) prosthesis, in order to prevent its bulging (with potential dehiscence of some lateral fixation points) and to increase parietal ingrowth of the prosthesis. Some authors mention the importance of this systematic (attempt at) closure of the hernia orifice with sutures in patients undergoing laparoscopic repair [11]. Although we believe this is not necessary in the small- and medium-sized hernias, it might be useful in the above-mentioned giant hernias to try to approximate the hernia edges laparoscopically. In these

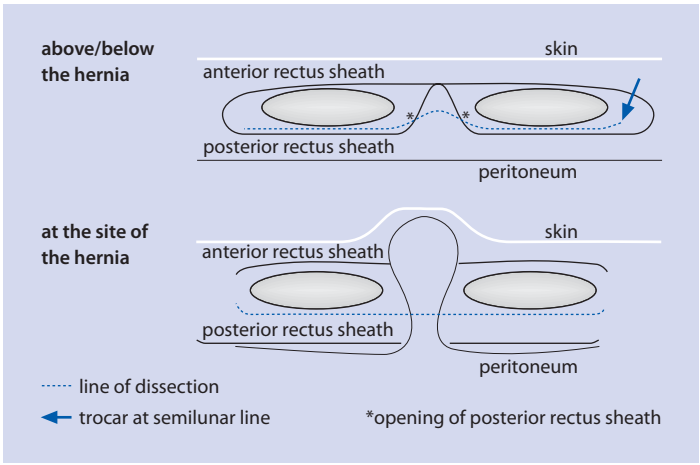


Fig. 24.3. Endoscopic extraperitoneal mesh repair. *Left:* Schematic drawing of the retromuscular dissection plane. *Above:* Patient with epigastric hernia where closure of the hernia sac was possible and a polypropylene mesh was used

hernias, more mesh overlap and more fixation might also be necessary.

In some cases we prefer to avoid the intra-abdominal route and use an endoscopic extraperitoneal mesh repair as described before [12]. Briefly, after entering the retromuscular space at the semilunar line with a 10/12 mm trocar, dissection is carried out bluntly and sharply towards the midline. Away from the hernia site, the posterior rectus sheath is opened on the midline bilaterally in order to create a retromuscular plane with a short rim of preperitoneal fat on the midline (Fig. 24.3a). At the site of the hernia, the hernia sac is opened circumferentially. If the hernia sac can be closed, a simple polypropylene mesh can be used (Fig. 24.3b). Moreover, due to natural fixation of the mesh in this virtual plane, minimal additional mesh fixation is probably necessary, which might be useful in hernias close to bony edges (lumbar hernias, hernias close to the pubic bone or xiphoid and costal margins), cases in which a true laparoscopic approach is less obvious because of more difficult fixation. This technique is more demanding but avoids an extensive abdominal adhesiolysis and the contact of the mesh and/or the fixation devices to the intestinal organs (except at the site of the hernia orifice in larger hernias where the peritoneum cannot be closed).

A Critical Analysis of our Results

We analyzed the results of 156 patients operated laparoscopically between January 2000 and December 2004 in whom a follow-up of more than 10 months was available. These patients, with both primary (32%) and incisional (68%) hernias, were operated by different surgeons, including our learning curve period for this technique. With 89.1% of patients in follow-up (7% lost to follow-up and 3.8% unrelated mortality), the mean follow-up period is 2.6 years (range 0.86–5.6 years). One third of incisional hernia patients had a recurrent incisional hernia after previous open mesh repair (9% of all patients) or open primary repair (14%). Six percent of all patients had a parastomal hernia. One third of all patients (34%) underwent an endoscopic extraperitoneal repair.

Two patients (1.44%) developed a postoperative peritonitis due to unrecognized or delayed intestinal perforation. Both events occurred during the first year of our experience, stressing the importance and potential risks of the adhesiolysis. Mesh removal was necessary in both cases; both patients survived and the hernia recurred in one patient. In another patient (0.72%), who had a recurrence after previous open mesh repair, a partial excision of the skin was performed because of ulcerated and thin overlying skin at the time of the laparoscopic repair. This evolved to a chronic wound with exposed mesh and subsequent deep prosthetic infection. The mesh was removed and a giant hernia recurred. Three patients (2.16%) developed a superficial infection (cellulitis, erythema +/- fever) which was successfully treated with temporary antibiotic treatment.

We noticed 11 patients with a recurrence (7.9%). In all patients, mesh overlap was 5 cm circumferentially and fixation was performed with fixation devices and four non-resorbable or slowly resorbable transfascial sutures, one in the middle on each side, unless stipulated otherwise in Table 24.3. This table shows some factors

Table 24.3. Overview of patients with primary or incisional hernia and factors potentially related to the recurrence

Primary		Incisional	
Small umbilical hernia – morbid obesity	Intra-abdominal pressure?	Appendectomy scar – morbid obesity	Mesh overlap, no sutures?
Epigastric hernia – morbid obesity	Intra-abdominal pressure?	Postoperative peritonitis	Mesh removal
Epigastric hernia – Surgisis	Mesh?	Subcostal incision	Mesh overlap, no sutures?
Umbilical hernia – morbid obesity	Intra-abdominal pressure?	Parastomal hernia	Central incision too large
		Periumbilical (AOD, hand-assisted)	Early failure (loosening fixation devices)
		2nd recurrent umbilical hernia	Dual Mesh fixed with PDS and fixation devices (recurrence at the edges)

AOD aortic occlusive disease

possibly related to recurrence in individual patients, although not all patients were re-operated.

In the primary hernias, the surgery is relatively straightforward and the recurrence rate should be low with enough mesh overlap and adequate mesh fixation. In this group, we noticed a recurrence rate of 8%. Most of the time, meshes were fixed with an intra-abdominal pressure of 8–10 mmHg, in order to avoid mesh wrinkling. However, in morbid obesity, the intra-abdominal pressure is likely to be higher than in other patients [13]. Thus, the higher tension on the mesh in this subgroup of patients might have led to a higher recurrence rate. Now, we keep the intra-abdominal pressure at 15 mmHg in morbidly obese patients when fixing the prosthesis. Of course, also mesh shrinkage should be taken into account, but the data available with respect to the mesh used are not consistent [14]. One hernia recurred after treatment with a non-permanent collagen mesh made from porcine small intestinal submucosa (Surgisis Gold®). The use of this type of prosthesis is based on the idea of a temporary reinforcement of the abdominal wall until autogenic remodelling at the site of the hernia is accomplished. Although the available data in the literature are promising [15–17], large randomized controlled trials are needed to determine the long-term effects of this kind of meshes.

In the incisional hernia group, we noticed a recurrence rate of 5.2% (excluding the parastomal hernias where the recurrence rate was 22.2%). As stated above, there is still a lot of controversy on the optimal mesh fixation. For the time being, we use a combination of fixation devices and transfascial sutures, in both endoscopic extraperitoneal and intraperitoneal repair. We use four slowly resorbable PDS sutures, not only for fixation, but also for easy manipulation and centring of the mesh exactly over the defect. The rationale for the use of slowly resorbable sutures is, on the one hand, a temporary additional fixation until mesh ingrowth is accomplished and, on the other hand, to diminish the risk of chronic pain or sinus formation due to the presence of sutures. However, it seems logical to use a permanent fixation when using less adherent purely ePTFE meshes or in the case of large-sized hernias. The fixation devices should be applied every 2 cm [18] and be very deep with good counterpressure from outside in order to ensure deep penetration into the mesh and the musculofascial layers.

In addition, our data confirm that even with the advent of laparoscopic repair, parastomal hernias remain very difficult to treat adequately [19].

Another complication, more specifically related to laparoscopic incisional hernia repair where the ab-

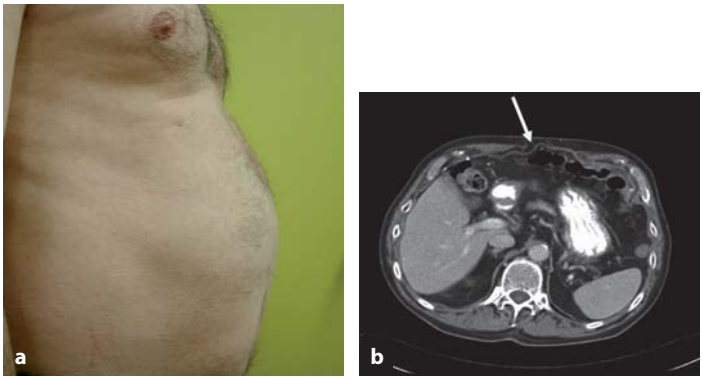


Fig. 24.4a,b. Postoperative bulging in patient after endoscopic extraperitoneal mesh repair (**a**) and in another patient after laparascopic repair for a recurrent incisional hernia after previous open mesh repair (**b**) due to mesh wrinkling (*arrow)*

dominal wall is not reconstructed and the hernia defect not closed, is the occurrence of postoperative bulging (Fig. 24.4). We noticed 11 patients (7.9%) with this problem, which was more frequent after extraperitoneal (14.9%) vs. intraperitoneal (4.3%) repair. Although a rather subjective feature, usually not causing any complaints to the patient, it can give some (aesthetic) discomfort to some patients and we believe that in a minority it should be regarded as a hidden recurrence. Therefore, this specific parameter should be reported together with the recurrence rate during the follow-up. We believe that the following factors are possibly related to a higher risk for postoperative bulging:

- An associated diastasis of the rectus muscle, especially in case of epigastric hernias.
- The incision of the posterior rectus sheath in endoscopic extraperitoneal repair.
- Too much wrinkling of the mesh during mesh fixation.
- The giant hernia with a transversal hernia orifice diameter of more than 10 cm without anterior support, as mentioned above.

Other often neglected items during the follow-up are prolonged postoperative pain and trocar site hernias.

Prolonged postoperative pain might be due to the use of both transfascial sutures or fixation devices and there is no consensus in the literature [2]. There is no clear definition on what exactly is prolonged pain. We noticed 30 patients (21.6%) with some degree of prolonged postoperative pain (need for narcotic analgetics, delayed discharge because of pain etc.). In the majority of cases, this is only a temporary phenomenon, but it is important to mention to the patients that the postoperative course (pain, reconvalescence) might not be comparable with other minimally invasive procedures.

It is very distressing both for the patient and the surgeon to see a patient with a trocar-site hernia after a minimally invasive repair of an incisional hernia. Since these hernias have small orifices, they often cause pain and the risk of incarceration is not to be underestimated. We noticed four patients with a trocar site hernia (2.9%). In one of these patients, the trocar site was not closed because of exposure problems in an obese patient. All trocar sites > 10 mm should be closed. This can be done by coverage with the same mesh or by use of a transfascial suture under laparoscopic guidance. We feel that simple closure of the fascia at the end of the procedure is facilitated by the placement of two traction sutures on the fascia before placement of the 10/12 mm trocar, especially in (morbidly) obese patients.

All these data show that a long-term follow-up of more than 3–5 years postoperatively can reveal important additional information. Large randomized trials comparing the effectiveness of open and laparoscopic mesh repair for ventral and incisional hernias are needed.

References

1. Perrone JM, Soper NJ, Eagon JC, Klingensmith ME, Aft RL, Frisella MM, Brunt LM (2005). Perioperative outcomes and complications of laparoscopic ventral hernia repair. Surgery 138: 708–716
2. LeBlanc KA (2005). Incisional hernia repair: laparoscopic techniques. World J Surg 29: 1073–1079
3. Jansen PL, Mertens Pr P, Klinge U, Schumpelick V (2004). The biology of hernia formation. Surgery 136: 1–4
4. Berger D, Bientzle M, Müller A (2002). Postoperative complications after laparoscopic incisional hernia repair. Surg Endosc 16: 1720–1723.
5. LeBlanc KA, Whitaker JM, Bellanger DE, Rhynes VK (2003). Laparoscopic incisional and ventral hernioplasty: lessons learned from 200 patients. Hernia 7: 118–124

6. Schumpelick V, Klinge U, Junge K, Stumpf M (2004). Incisional abdominal hernia: the open mesh repair. Langenbecks Arch Surg 389: 1–5
7. Luijendijk RW, Hop WC, van den Tol MP et al. (2000) A comparison of suture repair with mesh repair for incisional hernia. N Engl J Med 343: 392–398
8. Burger JW, Luijendijk RW, Hop WC, Halm JA, Verdaasdonk EG, Jeekel J (2004). Long-term follow-up of a randomized controlled trial of suture versus mesh repair of incisional hernia. Ann Surg 240: 578–585
9. Read RC. Milestones in the history of hernia surgery. Prosthetic repair (2004). Hernia 8: 8–14
10. Fassiadis N, Roidl M, Hennig M, South LM, Andrews SM (2005). Randomized clinical trial of vertical or transverse laparotomy for abdominal aortic aneurysm repair. Br J Surg 92: 1208–1211
11. Chelala E, Gaede F, Douilez V, Dessily M, Alle JL (2003). The suturing concept for laparoscopic mesh fixation in ventral and incisional hernias: preliminary results. Hernia 7: 191–196
12. Miserez M, Penninckx F. Endoscopic totally preperitoneal ventral hernia repair (2002). Surg Endosc 16: 1207–1213
13. Nguyen NT, Wolfe BM (2005). The physiologic effects of pneumoperitoneum in the morbidly obese. Ann Surg 241: 219–226
14. Coda A, Bendavid R, Botto-Micca F, Bossotti M, Bona A (2003). Structural alterations of prosthetic meshes in humans. Hernia 7: 29–34
15. Franklin ME Jr, Gonzalez JJ Jr, Glass JL (2004). Use of porcine small intestinal submucosa as a prosthetic device for laparoscopic repair of hernias in contaminated fields: 2-year follow-up. Hernia 8: 186–189
16. Eid GM, Mattar SG, Hamad G, Cottam DR, Lord JL, Watson A, Dallal RM, Schauer PR (2004). Repair of ventral hernias in morbidly obese patients undergoing laparoscopic gastric bypass should not be deferred. Surg Endosc 18: 207–210
17. Helton WS, Fisichella PM, Berger R, Horgan S, Espat NJ, Abcarian H. Short-term outcomes with small intestinal submucosa for ventral abdominal hernia. Arch Surg 140: 549–562
18. van't Riet M, de Vos van Steenwijk PJ, Kleinrensink GJ, Steyerberg EW, Bonjer HJ (2002). Tensile strength of mesh fixation methods in laparoscopic incisional hernia repair. Surg Endosc 16: 1713–1716
19. LeBlanc KA, Bellanger DE, Whitaker JM, Hausmann MG (2005). Laparoscopic parastomal hernia repair. Hernia 9: 140–144

Discussion

Jeekel: *It is very important to know what the future will be. We will, hopefully, have enough patients now for laparoscopic versus open trial next month and then we will get to the follow-up. What I don't understand is, you say whenever the defect is larger than 10 cm you hesitate to do this. In my opinion, the mesh is the new abdominal wall and you don't need to close anything above it. You don't need to and maybe you should not do it, because you get extra tension again on the wall. The mesh is your new wall, so why do you hesitate at 10 cm?*

Miserez: *Because I think that reconstruction of the abdominal wall, especially if you have a large orifice, and if you look at the experimental data from the group of Aachen this morning, you have more tendency that the mesh will bulge and bulging is maybe the beginning of a recurrence. So you need more overlap, you need better fixation in the large hernias. I would first like to see that laparoscopy works in small and medium-sized hernias. If it works there I will feel much more confident to extrapolate my experience to the large hernias. At this time I do not feel confident to say to the patients that I can guarantee that the result will be as good with my laparoscopic approach in those giant hernias. A giant hernia, for my understanding, is more than 10 cm. That is my reason.*

Jeekel: *A very important point is always that you should not put a mesh in the abdominal cavity, because you get adhesions and what ever. I don't say that, but did you have any data about adhesions and bowel obstructions etc.?*

Miserez: *No, I do not have any data. I know that in your hospital you have been using Prolene® intra-abdominally and some of the American colleagues have also been doing it. On the other hand, I know that the companies put that as a strict contra-indication on their instruction sheet. I have not only medical reasons not to do it. I would have a very difficult time in court to defend the use of a simple Prolene®-mesh intra-abdominally with intestinal erosion and a laparostomy as a final result. So that is the reason I don't do it.*

Schumpelick: *Is it true that a laparoscopic exploration is superior to a good ultrasound concerning analyzing of the defect?*

Miserez: *Again it will be a feeling, sorry for that. I think yes. I think ultrasound is dependent on experience; it is more difficult in obese patients. Laparoscopic exploration is operator-dependent too, but it depends on the adhesiolysis. You need to see the whole scar, I agree with that. But with some experience and the ultrasound also needs experience, I think you can differentiate when you feel from outside and you see from inside: there is bulging, there is weakness, there is a hole.*

Fitzgibbons: *It is consensus trying to determine the best practices. I have been surprised listening to all these talks about laparoscopic ventral hernia repair and I have heard no word about the major problem in this operation, which is an intra-abdominal injury with a huge and high mortality rate. I think it is so crucial. If this procedure is going on, we have to eliminate this problem of intra-abdominal injury. Because of the postoperative pain the patients have it is hard to recognize.*

Miserez: *You are completely right. Two remarks on that: First of all, this is why you will never hear me say that the laparoscopic repair is better, until we have some more evidence. We have intra-operative non-recognized enterotomies with postoperative peritonitis. Unfortunately, we had to take out the mesh when one occured, resulting in a long stay in intensive care. But that was during the first year of our experience. So we have been much more selective in choosing our patients and this is a very important point that I didn't stress, because I wanted to stress recurrence. But you are completely right.*
Deysine: *You mentioned it at the beginning of the conference, but I should like to emphasize the importance of obtaining a CT scan, whenever you have any doubt, particularly in obese patients. It has been extremely helpful and has allowed me to do what I want. I operate them all open since I don't know how to do them laparoscopically. But the CT scan has saved me a lot of trouble, particularly in people who are over 300 pounds in weight.*
Miserez: *I agree in these patients. But on the other hand, we sometimes see that every groin hernia patient now comes from the GP with an ultrasound, at least in Belgium. On the other hand, many patients with incisional hernias come from the physician or the GP with a CT scan which you don't need at all. I think again, we need to do it selectively, in patients where we cannot judge by our physical examination a CT scan might be helpful.*
Deysine: *I would not touch a ventral hernia without a CT scan.*
Bendavid: *About 10 years ago I published a study of 30 cases where I inserted a double mesh of Gore-Tex inside the abdomen and outside polypropylene. At that time already Gore-Tex was not recommended for intra-abdominal use. If you carefully read the insert, and this was absolutely stunning, here they are marketing it and selling it, and in the insert it is not recommended for intra-abdominal use.*
Miserez: *This has clearly changed. I don't think you will find it in the recommendation any more. I mean whether we use composite or we use a Gore-Tex mesh, both with ePTFE, but most literature is on the Gore-Tex mesh, the results are excellent. Despite the fact that people say that it gives less ingrowth. I think we have to face the evidence and honour the good effect of this mesh in laparoscopy.*

24.3 Laparoscopic Parastomal Hernia Repair

M.J. Elieson, J.M. Whitaker, K.A. LeBlanc

Introduction

Parastomal hernias have long plagued surgeons and the patients they treat. Although the true incidence is most likely unknown, the incidence of parastomal henias has been reported to be as high as 48% in patients with stomas [1–7]. The incidence appears to be highest in colostomies, lower in enterostomies, and still lower in urostomies [6, 7]. Two retrospective studies have suggested that ostomies located within the rectus sheath have a lower incidence of hernia formation [3]. These findings have led to the widely accepted convention that stomas should be placed within the rectus sheath. There have been several other studies, however, that have not supported this conclusion [3]. Yet, it is largely undisputed that stomas should not be brought through the laparotomy incision due to the unacceptably high incidence of associated complications (infections, dehiscence, and hernias) [8–11].

An overly large opening in the abdominal wall for the stoma has been suggested to increase the risk of parastomal hernia [8, 11–15]. Obesity, wound infection, old age, corticosteroid use, chronic respiratory disorders, and malnutrition are other factors that have been suggested to place patients at risk for the development of a parastomal hernia [8, 16–18].

All stomas are at risk of hernia formation as a result of the physical alteration of the anatomy of the anterior abdominal wall to allow the egress of the intestinal conduit. The necessity to leave the abdominal fascia open sufficiently large enough to allow passage of intestinal contents through the stoma and the compressible nature of the intestinal conduit combined with any of the standard risk factors for incisional hernia formation make for a high rate of hernia formation [19].

Repair of the enlarged fascial defect is indicated when the parastomal hernia is associated with a complication. Complications of parastomal hernias can be unique to the stoma or common to all hernias. Stoma complications include difficulty with care of the stoma such as an ill-fitting appliance, leakage of intestinal contents around the appliance, difficulty with irrigation, skin excoriation, or stomal obstruction as a result of extrinsic compression from the

hernia contents. Hernia complications include pain, incarceration, strangulation, obstruction of a herniated loop of intestine, or a significant cosmetic deformity [19].

The successful repair of these defects is challenged by the need to maintain the defect in the anterior abdominal wall. The multiplicity of techniques to repair parastomal hernias attests to the high failure rate of any of the methods currently employed. Perhaps the laparoscopic approach might provide a secure method of hernioplasty for these difficult hernias. We shall present our experience with such a repair. We have prospectively followed these patients in an effort to evaluate the most effective laparoscopic method of repair to utilize for these hernia defects.

Local aponeurotic repair has shown recurrence rates from 46 to 76% [18, 20]. This is clearly not an acceptable rate of recurrence. As such, stoma relocation was long purported to be the technique of choice to repair parastomal hernias. However, recurrence rates would be expected to be similar to primary herniation rates and have been documented to be from 33–40% [18, 20]. This repair also carries the risk of incisional hernia formation at the laparotomy site as well as the old stoma site. Interest in repairing parastomal hernias with mesh has developed as biomaterials have shown promise in the repair of other hernias. Repair with a polypropylene mesh via an open approach has improved these statistics, but still has recurrence rates as high as 26–29% [21, 22].

The success of the laparoscopic repair of incisional hernias has lead to the application of these techniques to the repair of parastomal hernias [19]. Many variations have been proposed for the laparoscopic repair of parastomal hernias. These have included single or multiple pieces of mesh, with and without slits or keyholes cut into the mesh, as well as a variety of different biomaterials. We have utilized most of these methods in an effort to identify the best method of repair.

Materials and Methods

The authors describe their technique of previously published results and current at the time of this writing [19]. The patients have been followed prospectively for hernia recurrence or other complications of their repair. None of the patients has been lost to follow-up.

All patients are placed in the supine position. The ipsilateral side of the patient is elevated to allow easier access to fixate the prosthesis laterally by placing a roll underneath the ipsilateral iliac wing. An antimicrobial impregnated drape is used to avoid any contact of the prosthesis with the skin of the patient. Following introduction of trocars, adhesiolysis commences and represents the most tedious portion of the procedure. Usually three to four 5 mm trocars are placed as the adhesiolysis permits. The use of an energy source of any kind is avoided in close proximity to the intestine that is encountered. This is done to minimize the risk of an unintended intestinal burn. The hernia defect and the intestine entering it is completely dissected (Fig. 24.5).

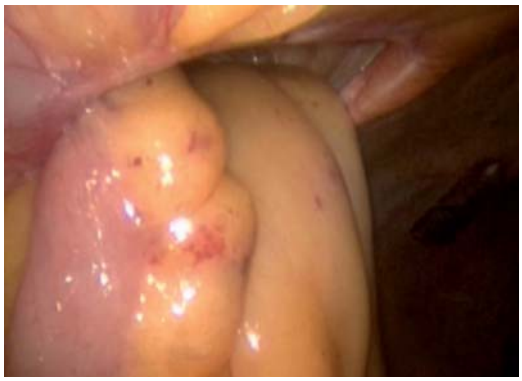


Fig. 24.5. Completed dissection of a parastomal hernia. The ileum is exiting an enlarged fascial defect

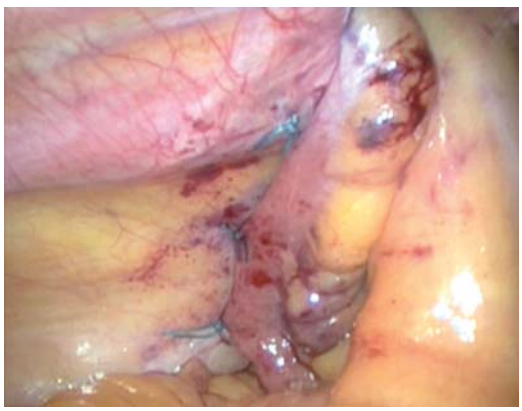


Fig. 24.6. The ileum has been sewn to the lateral abdominal wall with three non-absorbable sutures

The repair mimics that of the open repair described by Sugarbaker [23]. In this method, the intestine is lateralized along the abdominal wall by sutures. Prior to this lateralization, we prefer to cauterize the peritoneum of the overlying abdominal wall to propagate adhesions between the intestinal conduit and the abdominal wall as healing ensues. The intestine then is secured with the use of two to three non-absorbable sutures (Fig. 24.6).

The fascial defect is measured after the abdomen is deflated of all of the carbon dioxide to avoid any inflation artifact resulting in an expansion of the fascial defect. Based upon this measurement, a DualMesh Plus® biomaterial (W.L. Gore and Associates, Inc., Flagstaff, AZ, USA) is chosen so that it provides at least a 5-cm overlap of the fascial edges. Most often a 15 x 19-cm DualMesh Plus® prosthesis is sufficient to cover the bowel and the hernia defect with adequate fascial overlap. If there is an accompanying midline incisional hernia, an appropriately sized larger prosthesis is used to repair both hernias with one mesh, if feasible. The mesh is marked with a marker to denote the midline on each of the four edges, placing a single mark on the axial midline and a double mark on the longitudinal midline, by convention. There is no defect created in the DualMesh Plus® biomaterial. A CV-0 ePTFE suture (W. L. Gore & Associates, Flagstaff, AZ) is placed at the midline of one of the shorter edges and tied in place leaving both ends of the suture sufficiently long to pull through the abdominal wall and tie above the abdominal fascia. Two additional ePTFE sutures are placed on the edge opposite the first stitch. They are centred on the edge, separated just sufficient to allow the passage of the lateralized intestinal conduit between the patch and the abdominal wall. Commonly, the distance between these two sutures is a measured 7 cm (Fig. 24.7).

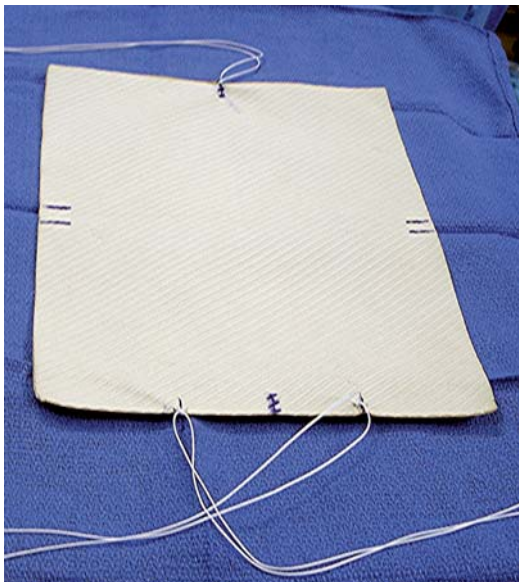


Fig. 24.7. A large DualMesh Plus® patch has been chosen. One suture has been placed on the patch and will be placed on the side opposite of the lateralized intestine. Two sutures have been placed 7 cm apart to accommodate the intestine beneath the patch

The sutures are folded inside the mesh as it is folded in half multiple times along its axis, leaving one end slightly tapered. Because the DualMesh Plus® prosthesis is 50% air by volume, it is compressed into a tight roll to allow for the introduction through the abdominal wall. One end will be rolled to that it is slightly tapered and this end will be pulled first into the abdomen (see below) [24].

The mesh is pulled into the peritoneal cavity by passing a grasper into a trocar on one side of the abdominal cavity and then out of a second trocar on the opposite side. The second trocar is removed, the tapered end of the folded mesh is grasped, and the mesh is slowly pulled into the peritoneal cavity as it is turned in a corkscrew fashion (Fig. 24.8). Since this procedure is performed with 5 mm trocars only, occasionally the skin incision is enlarged to better accommodate the mesh as it is pulled through the abdominal wall, although the authors usually find that this is unnecessary unless a large patch is used. The mesh is unwound just prior to pulling the last end of the mesh through the abdominal wall. It is then unfolded, using two or three graspers inside the abdominal cavity [24].

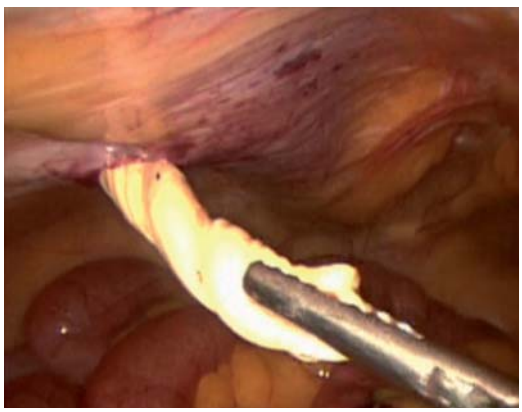


Fig. 24.8. A tightly rolled patch is pulled into the abdomen via a 5 mm port site

The mesh is fixed to the abdominal wall similarly to the traditional laparoscopic incisional hernia repair that we have reported previously [25]. This repair differs in that the intestine must tract along the side of the abdomen tucking under the mesh to exit through the stoma. The pair of sutures are brought through the abdominal wall and tied down on either side of the intestinal conduit by passing a suture passer through small skin incisions lateral to the lowest portion of the lateralized intestine. The single suture on the opposite end of the patch is secured in a similar fashion such

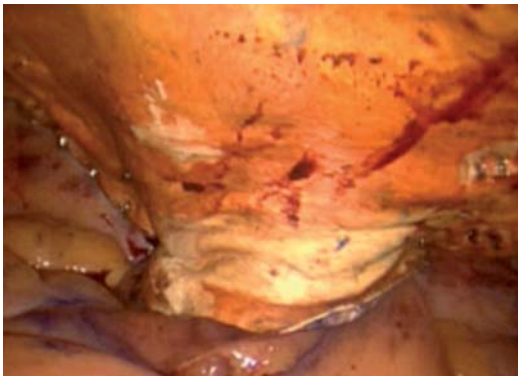


Fig. 24.9. The completed repair of the parastomal hernia. Note how the DualMesh Plus® covers the intestine with laxity to allow for its entrance toward the stomal site

that the mesh is pulled taut. Once this two-sided fixation is accomplished, the two unsecured edges are then grasped and spread out to provide equal coverage above and below the stoma and its associated defect. The edges of the mesh are then secured to the abdominal fascia by placing tacks or constructs circumferentially around the edge of the mesh approximately 1 cm apart. The fixation devices should be placed right on the edge of the mesh to avoid adhesions to the curled edge [26]. A second row of tacks is placed between and a few centimetres inside the first row. Additional transfascial sutures are placed around the periphery of the mesh no more than 5 cm apart to provide additional fixation of the mesh to the abdominal wall fascia [25, 27]. It is believed that this is the most important factor to assure adequate fixation of the patch to the abdominal wall (Fig. 24.9).

Once the repair is completed, it is important to inspect the abdominal contents carefully to assure that there is not an unrecognized intestinal injury or that hemorrhage is not present. Once this is assured, the trocars are removed under visualization.

Results

The results of our 11 laparoscopic parastomal hernia repairs utilizing the modified Sugarbaker technique are shown in Table 24.4. None of these patients have been lost to follow-up. The average length of follow up is 25.3 months (1–48). The average patient age is 59 years (41–85).

Table 24.4. Laparoscopic parastomal hernia repair results

Hernia type	Patient age [years]	Length of follow-up [months]	Recurrence	Complications
Ileostomy	55	48	–	–
Colostomy	44	46	+	–
Colostomy	64	44	–	SBO
Urostomy	78	39	–	Enterotomy
Colostomy	80	29	–	LBO
Urostomy	41	27	–	–
Urostomy	51	12	–	–
Ileostomy	46	6	–	–
Colostomy	55	3	–	–
Colostomy	59	2	–	–
Colostomy	76	1	–	–

SBO small bowel obstruction, *LBO* large bowel obstruction.

An intra-operative complication occurred in one patient. An inadvertent enterotomy occurred as the bowel adhesions were lysed from a prior polypropylene repair of the same hernia. The enterotomy was closed laparoscopically and the hernia was not repaired. The patient was returned to the operating theatre in 4 days and underwent a successful repair of the paraurostomy hernia.

One patient developed a small bowel obstruction postoperatively as a result of adhesions. She was returned to the operating theatre where she underwent a laparoscopic lysis of adhesions. The source of the adhesions was not related to the hernia repair. She recovered uneventfully following the second procedure.

One patient developed a postoperative colonic obstruction. She had undergone a prior relocation of her left lower quadrant colostomy to the right lower quadrant. Her recurrent hernia was repaired in a fashion similar to the other patients. She failed to have bowel function postoperatively and was re-explored laparoscopically. At re-operation, it was apparent that the resultant angulation of her left colon as it exited from under the right-sided mesh laterally resulted in a mechanical obstruction. This was treated laparoscopically by splitting the patch where the colon entered underneath the patch to relieve the tension. Subsequent to that procedure, she has maintained normal bowel function.

One recurrence occurred 3 years following repair of a paracolostomy hernia. On re-operation, the patch was found to have pulled off the abdominal wall laterally. A second patch was placed to cover the lateral aspect of the previous repair.

Discussion

Parastomal herniation is problematic to the patient and is a significant challenge to the surgeon. The many different techniques that have been reported attest to the inability to identify a single permanent and effective solution. It appears that the treatment of the entity is undergoing the same evolution that was seen for the repair of incisional hernias. Primary fascial repair with non-absorbable sutures has consistently been shown to have an unacceptable rate of recurrence that is as high as 76% [18, 20, 28, 29]. Relocation of the intestinal stoma is frequently touted as the best method to treat this malady. However, most patients would rather not move the location of their ostomy because of its familiarity and the need to undergo a fairly extensive operation. Furthermore, failure of this attempt is at least 33–40% [18, 20]. Therefore, these surgical attempts are not considered the appropriate approach by many surgeons unless factors require this choice.

The open placement of a prosthetic mesh in some fashion without the relocation of the stoma does appear to have decreased the rate of recurrence. However, there have been mixed reviews on this approach. Sugarbaker found that this method was very effective as he had no recurrences [23]. Morris-Stiff and Hughes reported a 5-year follow-up of seven patients in whom two pieces of polypropylene mesh were sewn to the intestine by "fingers" that were cut into the biomaterial [21]. A recurrent hernia developed in 29% of these patients. Moreover, serious complications such as obstruction or dense adhesions and mesh-related abscess formation were seen in 57 and 15% of these patients, respectively. Tekkis et al. reported the use of a polypropylene mesh to reinforce the in situ repair of fascial repair in five cases and reviewed the literature of other prosthetic repairs of parastomal hernias [30]. No recurrences were seen in their patients after a short-term follow-up of less than 2 years. In their review of the literature of stomal repairs that included a mesh of some type, a recurrence rate of 8.3% was cumulatively seen in 72 patients. This is less than others have reported. Certainly, one could make the argument that all of these series had few patients and that they were reported to detail favorable results and that unfavorable results are not generally reported. Others, however, have recently reported 0–20% rates of recurrence [31, 32]. Steele et al. reported on 58 patients who were repaired with polypropylene mesh over a 4-year period, with a mean follow-up of 50.6 months [22]. He placed the mesh as an onlay above the fascia via an open approach rather than as Sugarbaker or as we describe herein. There was an alarming rate of complications that were related to the mesh in 36% of these individuals. These were recurrence (26%), bowel obstruction requiring surgery (9%), prolapse (3%), wound infection (3%), fistula (3%), and mesh erosion (2%). While they concluded that the use of polypropylene was safe and effective, others, such as the authors, would not agree. However, the recurrence rate still represents an improvement on that of primary fascial repair or relocation. Based on these results with polypropylene, it would seem that this is not a preferred prosthetic biomaterial for this procedure by any technique.

The laparoscopic repair of parastomal hernias is in evolution. It is hoped that the diminution of the recurrence and complication rates that have been seen with the minimally invasive approach of incisional hernias will be extended to these very challenging hernias. There are few studies that have been reported with this technique. The largest series has been that of Berger,

which includes 15 patients [4]. He used an on-lay technique that uses fixation with transfascial sutures and tacks. Unless the mesh was greater than 20 cm, he did not use any more than four sutures. He, too, prefers an overlap of 5 cm for this procedure. In the immediate postoperative period, one patient in his series developed a hematoma and one patient required re-operation because of incarceration of small bowel between the patch and the abdominal wall (due to a dislocated tack). Three of these patients (20%) developed a recurrent hernia at between 2 and 4 months. One could certainly postulate that the method of fixation may be inadequate because of the relatively few transfascial sutures that were used in this repair. We believe that it is critical that transfascial sutures are used at not more than 5 cm apart along the entire periphery of all patches unless there is a structure such as the iliac bone that prohibits its placement [33]. In this instance, the patch should be secured with many more of the metal fixation devices than would be the usual recommendation. Some surgeons have trephined the bone to place sutures to ensure fixation, however.

There have been single case reports that have placed polypropylene mesh circumferentially around the exiting intestine to repair the defect. Pekmezci et al. used a polypropylene mesh to repair the hernia [34]. They chose to use the biomaterial because "it was cheaper and easier to manipulate", while acknowledging the fact that it is associated with enteric erosion and extrusion. Dunet et al. and Deol et al. used a similar type of technique for ileal conduits but used expanded polytetrafluoroethylene as the prosthesis [35, 36]. None of these patients experienced a complication but all of the follow-up was always very short.

Our series is one of the largest and with significantly longer follow-up than most others. Based upon our prior experience with DualMesh Plus® with incisional hernia repair and longer-term evaluation postoperatively, we believe that this is the best biomaterial that is available currently for this procedure. It is effective and has a low rate of postoperative adhesion formation [37].

We feel that the laparoscopic onlay technique is the best alternative to parastomal hernia repair. It is easy to perform and mimics the successful open repair of Sugarbaker [23]. We are continuing to follow all of these patients to assess the long-term outcome. The complication rate of 27% is somewhat high, but these were mostly minor and not associated with any long-term adverse consequences. With the exception of the large bowel obstruction secondary to a patch that was too tight because of the path of the bowel, none was absolutely preventable. The 2-year recurrence rate of 9% shows promise. Given the current results, we do not see any need to modify this approach to these hernias.

Conclusion

The laparoscopic repair of parastomal hernias appears to be a successful technique whose evolution has followed that of the laparoscopic incisional hernia repair. The laparoscopic onlay technique appears to be successful in the short term. Critical factors appear to be effective lateralization of the intestine, use of a large amount of prosthetic overlap, and secure fixation with transfascial sutures. Longer follow-up of these patients will be necessary to ascertain its efficacy.

References

1. Phillips P, Pringle W, Evans C, Keighley M Analysis of hospital based stomatherapy service. Ann Roy Coll Surg Engl 1985; 67: 37–40
2. Sjodahl R, Anderberg B, Bolin T. Parastomal hernia in relation to the site of the abdominal wall stoma. Brit J Surg 1988; 75: 339–341
3. Ortiz H, Sara MJ, Armendariz M, de Miguel M, Marti J, Chocarro C. Does the frequency of para-colostomy hernias depend on the position of the colostomy in the abdominal wall? Int J Colorect Dis 1994; 9: 65–67
4. Berger D. Laparoscopic parastomal hernia repair: indications, technique, and results. In: Morales-Conde S (ed) Laparoscopic ventral hernia repair. Springer, Berlin Heidelberg New York, 2002, pp 383–387
5. London-Schimmer EE, Leong APK, Phillips RKS. Life Table analysis of complications following colostomy. Dis Col Rect 1994; 37: 916–920
6. Marshall FF, Leadbetter WF, Dretler SP. Ileal conduit parastomal hernias. J Urol 1975; 113: 40–42
7. McDougal WS. Use of intestinal segments and urinary diversion. In: Walsh PC, Retik AB, Vaughan ED, Wein AJ (eds) Campbell's Urology. Saunders, Philadelphia, 1998, pp 3121–3161
8. Goligher JC. Surgery of the anus, rectum and colon 4. Bailliere Tindall, London, 1980
9. Hulten L, Kewenter J, Kock NG. Complications of ileostomy and colostomy and their treatment. Chirurg 1976; 47:16–21
10. Pearl RK, Prasad NL, Orsay CP, et al. A survey of technical considerations in the construction of intestinal stomas. Ann Surg 1988; 51: 462–465
11. Todd IP. Intestinal stomas. Heinemann, London, 1978
12. Pearl RK, Prasad NL, Orsay CP, et al. Early local complications from intestinal stomas. Arch Surg 1985; 120: 1145–1147
13. Kronberg O, Kramhohft J, Backer O, et al. Late complications following operations for cancer of the rectum and anus. Dis Colon Rectum 1974; 17: 750

14. De Ruiter P, Bijnen AB. Successful local repair of paracolostmy hernia with a newly developed prosthetic device. Int J Colorect Dis 1992; 7: 132–134
15. Martin L, Foster G. Parastomal hernia. Ann Roy Coll Surg Engl 1996; 78: 81–84
16. Kingsnorth AN, LeBlanc KA. Parastomal hernia. In: Management of abdominal hernias, 3rd edn. Oxford University Press, London, 2003, pp 257–266
17. Leslie D. The Parastomal hernia. Surg Clin North Am 1984; 64: 407–415
18. Rubin MS, Schoetz DJ, Matthews JB. Parastomal hernia. Is relocation superior to fascial rep"air? Arch Surg 1994; 129: 413–418
19. LeBlanc KA, Bellanger DE, Whitaker JM, Hausmann MG. Laparoscopic parastomal hernia repair. Hernia 2005; 9:140–144
20. Cheung M-T, Chia N-H, Chiu W-Y. Surgical treatment of parastomal hernias complicating sigmoid colostomies. Dis Colon Rectum 2001; 44: 266–270
21. Morris-Stiff G, Hughes LE. The continuing challenge of parastomal hernia: failure of a novel polypropylene mesh repair. Ann R Coll Surg 1998; 80: 184–187
22. Steele SR, Lee P, Martin MJ, Mullenix PS, Sullivan ES. Is parastomal hernia repair with polypropylene mesh safe? Am J Surg 2003; 185(5): 436–440
23. Sugarbaker PH. Peritoneal approach to prosthetic mesh repair of paraostomy hernias. Ann Surg 1985; 201(3): 344–346
24. LeBlanc KA. A new method to insert the Dual Mesh prosthesis for laparoscopic ventral herniorrhaphy. JSLS 2002; 6: 349–352
25. LeBlanc KA, Whitaker JM, Bellanger DE, Rhynes VK. Laparoscopic incisional and ventral hernioplasty: lessons learned from 200 patients. Hernia 2003; 7(3): 118–124
26. LeBlanc KA, Stout RW, Kearney MT, Paulson DB. Comparison of adhesion formation associated with Pro-Tack (US Surgical) versus a new mesh fixation device, Salute (Onux Medical). Surg Endo 2003; 17: 1409–1417
27. Heniford BT, Park A, Ramshaw BJ, et al: Laparoscopic repair of ventral hernias. nine years' experience with 850 consecutive hernias. Ann Surg 2003; 238: 391–400
28. Thorlakson RH. Technique of repair of herniations associated with colonic stomas. Surg Gynecol Obstet 1965; 120: 347–350
29. Allen-Mersch TG, Thompson JP. Surgical treatment of colostomy complications. Br J Surg 1988; 75: 416–418
30. Tekkis PP, Kocher HM, Payne JG. Parastomal hernia repair. Modified Thorlakson technique, reinforced by polypropylene mesh. Dis Col Rect 1999; 42: 1505–1508
31. Venditti D, Gargiani M, Milito G. Parastomal hernia surgery: personal experience with use of polypropylene mesh. Tech Coloproctol 2001; 5: 85–88
32. Kald A, Landin S, Masreliez C, Sjödahl R. Mesh repair of parastomal hernias: new aspects of the Onlay technique. Tech Coloproctol 2001; 5: 169–171
33. LeBlanc KA, Booth WV, Whitaker JM, Bellanger DE. Laparoscopic incisional and ventral herniorraphy in 100 patients. Am J Surg 2000; 180(3): 193–197
34. Pekmezci S, Memisoglu K, Karahasanoglu T, Alemdaroglu K. Laparoscopic giant patastomal hernia repair with prosthetic mesh. Tech Coloproctol 2002; 6: 187–190
35. Dunet F, Pfister C, Denis R, Pascal T, Khalil H, Peillon C. Laparoscopic management of parastomal hernia in transileal urinary diversion. J Urol 2002; 167: 236–237
36. Doel ZK, Shayani V. Laparoscopic parastomal hernia repair. Arch Surg 2003; 138: 203–205
37. Koehler RH, Begos D, Berger D, Carey S, LeBlanc K, Park A, Ramshaw B, Smoot R, Voeller G. Minimal adhesions to ePTFE mesh after laparoscopic ventral incisional hernia repair: reoperative findings in 65 cases. JSLS 2003; 7: 335–340

Discussion

Ramshaw: *I was wondering if you could describe how you prepare the stoma, and whether it is different if it's a diversion as against a bowel diversion.*
LeBlanc: *If it is colon I do bowel preparation. For parastomy I would put a catheter into the ileum to drain it. Actually, we had tried in the past to put a thick catheter in, thinking it would help, but it is so stiff that it actually hinders the operation. So I don't do this anymore.*
Ramshaw: *After that obstruction, do you ever manipulate the bowel? Do you have an opening at the mesh level with some sort of calibration?*
LeBlanc: *The only thing I do is put my finger in the stoma itself just to see how it feels. But I can't feel an obstruction, because it is too deep.*
Miserez: *A technical question: If possible, do you try to orientate your loop 180° versus the hernia defect? If you are doing the Sugarbaker, you want to cover your defect as good as possible, did you try to put your loop at 180°?*
LeBlanc: *I actually look from the exact opposite of the abdominal wall laparoscopically and thin the bowel right there and I actually stitch a suture to know exactly the thinness of the defect.*
Ramshaw: *I think you run the bowel not over the defect, but away from the defect.*
Ferzli: *Every time we do a colostomy we are actually inducing a hernia, because we are cutting the fascia and bringing the bowel through. Did you look at those 70 patients how the colostomy was created, by what technique? Essentially what I am trying to say is: are you going to be selective in your approach to paracolostomia hernia, based, number one, on the patient population with connective diseases? What we are dealing with is a substance.*
Number two: How was the colostomy made? What was the operative report? All these factors influence the approach to the incision whether open or relocation. To me it seems, and I have suffered actually through these challenging cases, that there are multiple problems, not just one simple parastomal hernia that requires a repair.
Berger: *What would you propose if you find a lateral defect, a fascia defect lateral from the stoma loop?*
LeBlanc: *I would do the same, I might put a stitch right to the fascia defect itself.*

24.4 Reasons for Recurrence After Laparoscopic Treatment of Parastomal Hernias

D. Berger, M. Bientzle

Introduction

The incidence of parastomal hernias is so high that sometimes the herniation is interpreted as inevitable rather than as a complication of the surgical technique itself [3, 16,17]. However, the pathogenesis of parastomal hernias seems to be similar as to that described for incisional hernias in terms of a biological disease with disturbed collagen synthesis [8–10]. Therefore the recurrence rates after simple suture repair or relocation of the stoma can be easily explained and each kind of repair should be based on the insertion of a mesh [8, 21, 23]. In 1985 an open IPOM technique was introduced with very promising long-term results in a small number of patients [24]. Increasing experience with laparosocopic repair of incisional hernias suggests a laparoscopic adaptation of the original Sugarbaker technique which is based on the coverage of the abdominal wall by a non-resorbable mesh and fixing the stoma loop between the abdominal wall and the mesh for at least 5 cm from medial to lateral. This technique was introduced in November 1999 and modified in October 2003. The presentation is limited to the description of the surgical technique and the results in terms of the recurrence rate.

Surgical Technique

Repair of a parastomal hernia was recommended if the hernia was growing rapidly , the stoma care experienced increasing problems or the patients suffered from pain due to recurrent incarceration. Pre-operative colonic preparation was used routinely. The pneumoperitoneum was established by an open access of a 10-mm trocar in the right or left upper quadrant usually on the opposite site of the stoma. One 30° optic and two further 5- and 10-mm trocars were used on the same side for the adhesiolysis of the complete abdominal wall. Fatty tissue such as the falciform ligament or between the plicae mediales was removed and the prevesical space of Retzius was opened. The stoma loop must be clearly identified and isolated from adherent greater omentum and small or large bowel. The parastomal defect is accurately measured as well as the accompanying defect of the midline if present. For the first 41 patients Dualmesh® of W.L. Gore & Assoc., Flagstaff, USA, was used for covering the abdominal wall. The size of the mesh should allow an overlap of the whole original incision and the stoma of at least 5 cm. The stoma loop was placed between the mesh and the abdominal wall from medial to lateral for again 5 cm, which is called lateralization, and then turned down to the abdominal cavity at the lateral margin of the mesh. The mesh was fixed with transfascial sutures and spiral tacks.

The so-called sandwich technique represents a modification which is based on the primary introduction of an incised mesh. This mesh is primarily placed around the stoma loop covering the lateral part of the abdominal wall with the non-incised part of the mesh. The medially placed incision is closed by two transfascial sutures and spiral tacks which were also used for fixing the margins of the mesh. The stoma loop crosses the centre of the mesh via a 2 x 2 cm defect. A second mesh was placed as previously described in the simple IPOM technique. So the stoma loop passes the abdominal wall and the first mesh proceeding between both meshes to the lateral margins providing a lateralization of at least 5 cm. In order to provide incorporation of both meshes, covered polypropylene or polyvinylidene fluoride (between October 2003 and April 2004 Parietene composite® of Sofradim Co., Lyon, France, and since may 2004 Dynamesh IPOM®, Dahlhausen Co., Cologne, FRG) was used.

Results

Between November 1999 and September 2005, 60 patients with different kinds of terminal ostomies and symptomatic parastomal hernias were treated laparoscopically; 22 out of the 60 patients suffered from recurrent hernias after open repairs previously attempted between 1 and 12 times! The follow-up ranges from 3 months to 6 years with a median of 24 months. No conversion was necessary.

In the meantime, recurrences were observed in eight patients. One patient underwent emergency laparotomy due to ileus not related to the primary IPOM repair 9 months before. When opening the abdomen, the ePTFE mesh was incised and sutured again. The suture broke down and a giant incisional and parastomal hernia developed. The repair was done laparoscopically with the sandwich technique.

Common features of the remaining seven patients were a lateral fascial gap and the IPOM technique. One patient was laparoscopically repaired by using again the IPOM technique and developed a further recurrence after 3 months. Three other patients were laparoscopically re-treated with the sandwich technique and remained recurrence-free. Three patients did not accept further surgical treatment.

Discussion

Parastomal hernias are a challenging problem of hernia repair. Suture repair or relocation of the stoma is associated with high recurrence rates exceeding 50% [8, 20, 21, 23]. It is generally accepted that, due to the biochemical nature of the hernia disease in terms of altered collagen metabolism, mesh-based techniques should be used. However, the open augmentation of the abdominal wall is accompanied by a high rate of wound complications and recurrence rates again exceeding 10% [4, 7, 8, 19, 23, 25]. The experience with laparoscopic repair of incisional hernias clearly demonstrated the superiority over the open techniques concerning the rate of infection or hematomas and general wound complications [1, 2, 12]. Recurrence rates after laparoscopic repair of incisional hernias seem to be at least as good as those obtained after open sublay techniques. So the laparoscopic adaptation of the open IPOM technique originally described by Sugarbaker seems to be a logical consequence. In the literature, some minor series have been published dealing with the laparoscopic repair of parastomal hernias [11, 13, 15, 22, 26]. Urological as well as enteric ostomies were included, and different techniques were used. However, the main limitation was the small number of patients included in the studies, rarely exceeding ten. Therefore, a clear statement about the real value of the laparoscopic approach is impossible. On the other hand, most studies dealing with the open repair also suffer from small patient numbers and do not allow any evaluation or even a recommendation of the most suitable technique. The same fact holds for the very recent description of the relocation of the stoma with a prophylactic mesh and the mesh-based repair of the original defect by Israelson et al. [5].

The present series comprises 60 patients with an adequate median follow-up of 24 months. The original technique exactly followed the suggestions of Sugarbaker [24]. Evaluating the results, we must recognize that, in contrast to the original results, the recurrence rate in our early series was high. All patients experiencing recurrences held in common a laterally localized fascial defect. All patients with a medial defect experienced a definitely stable abdominal wall. Lateralization of the stoma loop, which is a crucial step of the Sugarbaker-technique, implies that the lateral defect is covered by the stoma loop underlying the mesh. Obviously the defect was not stabilized and could grow, leading to the lateral recurrence. So we concluded that the lateral defect must be covered by a mesh providing ingrowth and long-lasting stabilization of the abdominal wall. Incising a mesh and placing around the stoma loop proved to be very effective. A second mesh was further inserted as originally described. Thus, the defect can be repaired and the crucial lateralization of the stoma loop is provided.

In 19 patients who underwent the sandwich repair, we observed no further recurrence. Although the original one-mesh technique seems to be sufficient for medially localized defects, it has been completely replaced by the sandwich technique because the latter has proved to be very effective and safe in all cases of parastomal hernias. Furthermore, the availability of a real mesh material such as Dynamesh IPOM® (polyvinylidene fluoride) provides incorporation of overlapping meshes, which is impossible when ePTFE meshes are used. To our knowledge, there is no information available concerning the behaviour of overlapping covered polypropylene meshes. Furthermore, the shrinkage of ePTFE is most pronounced, so that an incised ePTFE mesh placed around the stoma loop may lead to a stenosis in the long run [6, 18]. Polypropylene meshes in direct contact with the viscera are known to erode the bowel, producing enteric fistula [14].

In conclusion, the laparoscopic repair of parastomal hernias should be performed with the sandwich technique using the most inert material available today, which is polyvinylidene fluoride.

References

1. Berger D, Bientzle M, Muller A (2002) Postoperative complications after laparoscopic incisional hernia repair. Incidence and treatment. Surg Endosc 16: 1720–1723
2. Cassar K, Munro A (2002) Surgical treatment of incisional hernia. Br J Surg 89: 534–545
3. Cheung MT (1995) Complications of an abdominal stoma: an analysis of 322 stomas. Aust N Z J Surg 65: 808–811
4. Hamy A, Pessaux P, Mucci-Hennekinne S, Radriamananjo S, Regenet N, Arnaud JP (2003) Surgical treatment of large incisional hernias by an intraperitoneal Dacron mesh and an aponeurotic graft. J Am Coll Surg 196: 531–534
5. Israelsson LA (2005) Preventing and treating parastomal hernia. World J Surg 29: 1086–1089

6. Johnson EK, Hoyt CH, Dinsmore RC (2004) Abdominal wall hernia repair: a long-term comparison of Sepramesh and Dualmesh in a rabbit hernia model. Am Surg 70: 657–661
7. Kasperk R, Klinge U, Schumpelick V (2000) The repair of large parastomal hernias using a midline approach and a prosthetic mesh in the sublay position. Am J Surg 179: 186–188
8. Kasperk R, Willis S, Klinge U, Schumpelick V (2002) Update on incisional hernia. Parastomal hernia. Chirurg 73: 895–898
9. Klinge U, Conze J, Krones CJ, Schumpelick V (2005) Incisional hernia: open techniques. World J Surg 29: 1066–1072
10. Klinge U, Si ZY, Zheng H, Schumpelick V, Bhardwaj RS, Klosterhalfen B (2001) Collagen I/III and matrix metalloproteinases (MMP) 1 and 13 in the fascia of patients with incisional hernias. J Invest Surg 14: 47–54
11. Kozlowski PM, Wang PC, Winfield HN (2001) Laparoscopic repair of incisional and parastomal hernias after major genitourinary or abdominal surgery. J Endourol 15: 175–179
12. Le Blanc KA (2005) Incisional hernia repair: laparoscopic techniques. World J Surg 29: 1073–1079
13. Le Blanc KA, Bellanger DE, Whitaker JM, Hausmann MG (2005) Laparoscopic parastomal hernia repair. Hernia 9: 140 144
14. Leber GE, Garb JL, Alexander AI, Reed WP (1998) Long-term complications associated with prosthetic repair of incisional hernias. Arch Surg 133: 378–382
15. LeBlanc KA, Bellanger DE (2002) Laparoscopic repair of paraostomy hernias: early results. J Am Coll Surg 194: 232–239
16. Leong AP, Londono-Schimmer EE, Phillips RK (1994) Life-table analysis of stomal complications following ileostomy. Br J Surg 81: 727–729
17. Leslie D (1984) The parastomal hernia. Surg Clin North Am 64: 407–415
18. McGinty JJ, Hogle NJ, McCarthy H, Fowler DL (2005) A comparative study of adhesion formation and abdominal wall ingrowth after laparoscopic ventral hernia repair in a porcine model using multiple types of mesh. Surg Endosc 19: 786–790
19. Morris-Stiff G, Hughes LE (1998) The continuing challenge of parastomal hernia: failure of a novel polypropylene mesh repair. Ann R Coll Surg Engl 80: 184–187
20. Pearl RK (1989) Parastomal hernias. World J Surg 13: 569–572
21. Rubin MS, Schoetz DJ Jr, Matthews JB (1994) Parastomal hernia. Is stoma relocation superior to fascial repair? Arch Surg 129: 413–418
22. Safadi B (2004) Laparoscopic repair of parastomal hernias: early results. Surg Endosc 18: 676–680
23. Stephenson BM, Phillips RK (1995) Parastomal hernia: local resiting and mesh repair. Br J Surg 82: 1395–1396
24. Sugarbaker PH (1985) Peritoneal approach to prosthetic mesh repair of paraostomy hernias. Ann Surg 201: 344–346
25. Tekkis PP, Kocher HM, Payne JG (1999) The continuing challenge of parastomal hernia: failure of a novel polypropylene mesh repair. Ann R Coll Surg Engl 81: 140–141
26. Voitk A (2000) Simple technique for laparoscopic paracolostomy hernia repair. Dis Colon Rectum 43: 1451–1453

24.5 Meshes in Recurrent Incisional Hernias

Jan F. Kukleta

Introduction

The event of laparoscopic minimal invasive techniques has profoundly changed the abdominal surgery in the last 15 years. The feasibility of even advanced visceral procedures and the improvement of the applied material created an expectation of general availability of excellent results reported by a few experts. There is a discrepancy between the expected reduction of morbidity and recurrence rate and the average clinical outcome.

Incisional hernia, as an important, late complication of abdominal surgery, is still too frequent, and the outcome of its traditional surgical therapy is too poor to be accepted. It took several decades for the alloplastic prosthetic material to become widely accepted, being only randomly understood in its diversity and complexity. The potential danger of infection, failure, chronic pain and unknown other adverse effect have made this evolution even longer.

The naked fact of high recurrence rate in suture repair of incisional hernias [1] was the driving force for a change: the failure rate of a surgical effort of over 50% was reason enough to take the risk and challenge of implanting a foreign body. The results have improved since then.

But what are the results? Another issue exposed to the pressure of the societal change. Not only recurrence, but infection, chronic pain and disability, length of hospital stay and of off-work period, the total societal cost and, last but not least, the resulting improvement of the individual patient's quality of life. A fact which very often becomes difficult to be seen in the haze of scientific randomized activities.

What are the realistic expectations of today's surgical therapy? The mesh has become an essential part of the repair of incisional and ventral abdominal hernias. There is a variety of synthetic and biological prosthetic materials which can be used in the repair of primary incisional and recurrent abdominal hernias, irrespective of the kind of approach. The various different properties of these meshes, their unequal behaviour during the healing process and the product-specific potential disadvantages [9], force the operating surgeon to become familiar with this new

segment of surgical knowledge and select the appropriate product according to the specific patient's situation. Moreover, he has to learn to accept that something we learned to be trivial years ago is no longer trivial today.

What is the Present State of the Art of Being Wrong?

As the meshes used in the LIHR are placed intraperitoneally (with very few exceptions), they have to fulfil at least the one and only condition, to cause no harm to the abdominal wall and viscera like chronic pain, infections, adhesions, intestinal complications like bowel obstruction and erosions with intestinal fistulas (Table 24.5) [2].

As the intention is not only not to cause any harm, but to treat, the prosthetic material has to add the specific strength to the repair to help to prevent the recurrence. It is desirable and expected that the mesh will become incorporated, a functional part of the entire abdominal wall. This means it should help to restore the malfunction caused by the herniation. Therefore it has to mimic the integrity of the abdominal cavity by bridging or reinforcing the defect and mimic its elasticity now and for a long time, if not for ever. To become a part of the abdominal wall for a long time, the mesh has to be either perfectly fixed to the defect edges by any ideal permanent artificial internal or eternal fixation, or it has to be kept in place by the in-growth of the host scar tissue in the so-called sufficient overlap area.

One of the reasons for my guarded optimism about the state of the art is the fact that the necessary overlap margin grew from 2–3 cm up to 5 and more just within the last 10 years. The prosthetic material has to be or become compatible to being placed intraperitoneally and it has to reach a permanent bond with the defect edges to fulfil the demand on integrity. Besides this, the bigger the substituted area becomes in relation to the surface of the whole abdominal wall, the more its physical properties have to be respected to restore its original function [3].

Table 24.5. Meshes used in incisional hernia repair

ePTFE	Dual mesh, Dulex
Polypropylene PP	Prolene, Marlex, etc.
Polyester PE	Mersilene, etc.
Composites	
– PP + Collagen	Parietene composite
– PE + Collagen	Parietex composite
– PP + Hyaluronate	Sepramesh
– PP + PDS + ORC	Proceed
– PP + ePTFE	Composix
– PP + PVFD	Dynamesh
– PP + Polyglactin	Vypro, Vypro 2
– PP + Polyglecaprone	Ultrapro
Biomaterials	
– Porcine	Surgisis, Permacol
– Human	Alloderm

Key Properties

The intraperitoneal mesh is, in contrast to the intraparietal one, exposed to two different environments. Therefore it has to satisfy a unique demand to have two differently behaving surfaces. Both interfaces, mesh-abdominal wall and mesh-viscera, have their own problematic.

Parietal Interface

Ingrowth

Among the available materials we have a quite clear understanding about the parietal side of the ideal mesh. The stronger the ingrowth is, the better [4]. The physical property that enables this is a macroporous, non-absorbable and light-weight mesh structure. To reach the state of incorporation, we have to span the time-dependent process of fibroblast colonization, collagen deposition and its maturation by the use of different means of permanent, slowly absorbable or absorbable fixation to keep the mesh in place.

Shrinkage

The known effect of surface reduction of the implanted mesh is a mesh-specific reaction of healing in [4, 10]. This can be negative by uncovering the insufficiently

overlapped defect (leading to recurrence), by increasing the tension on fixing devices (e.g. tack hernia [5]) or the tension on transparietal sutures (chronic pain). Shrinkage can lead to the positive effect of medialization of lateral defect edges [6].

The faster a tension-resistant ingrowth can be reached, the less pronounced seem to be the resulting compression of the mesh. Is this the reason for the most pronounced shrinkage of ePTFE?

Porosity

The bigger the mesh pores, the faster the ingrowth. The marked shrinkage of inguinally implanted heavy polypropylene in comparison with the light one is obviously visible. The question, if less fibrotic reaction weakens the stability of the mesh–abdominal wall compound is still not responded.

Extent of Protrusion

Some of the light-mesh constructs seem to have less memory or too much elasticity and facilitate a protrusion into the defect (pseudohernia, bulge) even without complete dislocation. This property becomes of importance when bridging a large single defect, rather than a long Swiss-cheese defect.

Strength

The tensile strength of the mesh material should exceed 32 N/cm when bridging a defect [7].

Host Body Reaction

There is not only the foreign-body reaction with resulting fibrosis which helps to secure the mesh in place; especially when using biological mesh material there is the concern of unfavourable immunological responses (toxic reactions) related to material origin [9].

Infection Resistance

Monofilament meshes are less susceptible to bacterial infections than the multifilament ones or microporous materials [8]. Adding antibacterial agents could diminish the risk of infection (e.g. silver/chlorhexidine) [11]. Macroporous meshes do not harbour the bacterial infection in the way the microporous do, the coating of composite meshes increases the risk of infection. Biomaterials promise high infection resistance due to their fast vascularization.

Visceral Interface

Adhesion Prevention

The visceral side of the mesh for intraperitoneal positioning is still a subject of wishful thinking. The capital problem is the adhesiogenesis. The foreign-body response of the host ranges from mild to very strong inflammatory reaction with loose/dense adhesions as a consequence. The justified reservation against unprotected polypropylene or polyester meshes is the possibility of late erosions of intestinal loops and chronic fistulization [2].

Adhesion prevention is either the permanent property of the visceral interface (ePTFE, PVFD, polyurethan) or a temporary tissue separating concept of the composite meshes. An absorbable coating protects viscera until the underlying non-absorbable component is overgrown by neoperitoneum. The protective layer may be a collagen film (Parietene®/Parietex composite®), hyaluronate sodium with carboxymethylated cellulose (Sepramesh®) or oxidized regenerated cellulose (Proceed®).

The results of experimental comparative studies concerning the extent and dignity of adhesions, the peel-off strength or shrinkage of various products are still not so convincing to determine clearly the “best” material.

Biomaterials

Despite the fact that the non-absorbable mesh materials used today are chemically inert, they still cause a local chronic inflammatory process lasting for years. There is strong hope that the biomaterials will do better. The rational of these products is the remodelling of the extracellular matrix of porcine or human origin by the host tissue. This site-specific tissue transformation is called intelligent tissue remodelling. The acellular scaffold enables a rapid colonization by fibroblasts and promotes intensive vascularization. The gradual substitution of the original patch by the host’s tissue increases the initial strength of the repaired area. The strength exceeds that of the native tissue when used as a body wall repair device [33].

The clinical reports are promising, indicating the possibility of use even in contaminated environment. There are not enough data available to confirm this expectation yet [17, 18, 29, 30].

Critical Report

An important information was communicated by Robinson et al. [9]. Over 250 adverse event reports of the FDA's Manufacturer User Facility Device Experience Database concerning the use of mesh in hernia repair were analyzed. According to their results, specific mesh materials are related to specific complications. Although the results are not representative for conclusions: infections were related to PTFE (75 vs. 41% all other), mechanical failure to Sepramesh (80 vs. 14%), reaction to biomaterials (57 vs. 7%) and intestinal complications to composix(14 vs. 7%).

Outlook

The hydrophobic surface of polypropylene fibres prevents cell growth. Embedding polypropylene with extracellular matrix enables the fibroblast proliferation. The faster the mesenchymal cells cover the prosthetic material, the less adhesions are to be expected. The feasibility of precoating mesh material with living human fibroblasts has been demonstrated [31]. The use of a patient's own cultivated fibroblasts for this purpose seems to be the next step.

Local release of a basic fibroblast growth factor in a surgical wound improves the resulting stability of the healed incision and significantly helps to prevent the formation of incisional hernias [32]. Dubay's report shows a realistic possibility of applying FGF in high-risk hernia patients in prophylactic manner in the near future.

Discussion

A major part of the available information about mesh behaviour in the intraperitoneal position comes from animal studies [12–15]. Some of the results are very contradictory [14–16], or at least difficult to interpret, especially concerning shrinkage and extent of adhesions.

The unanswered questions are:

1. What is the dignity of the adhesions between the protective layer and the abdominal content?
2. How efficient is the neoperitoneum as a protection against the chronic inflammatory process caused by macroporous light weight meshes of the parietal mesh face?
3. Can the tissue ingrowth replace the function of permanent fixation in bridging bigger defects?
4. If yes, how long does it take?
5. What is the impact of the factor surgical technique in relation to the final outcome in front of the above background?

The true reproducibility of animal studies and randomized clinical trials with too small numbers of patients and too many variables (e.g. multicentre studies) has to be questioned from time to time. Maybe it is time again to rethink the significance of the reports of level-5 evidence and try to focus on the details of the individual surgical performance of experienced experts too [24, 26, 28, 29].

Conclusions

The mesh-reinforced incisional hernias still recur. What can be done better? Approach, technique, mesh size, bigger overlap, better fixation? Weight reduction? Growth factors? Can a change of mesh material type bring the necessary impact?

Despite the better knowledge of specific mesh properties and their behaviour during the in-growth, none of the known meshes is significantly much better than the rest. Despite the diversity of the available products the real choice is limited.

According to today's information, unprotected macroporous meshes should not be used in intraperitoneal position, although the role of light-weight polypropylene meshes in this position is not determined yet.

The composite/coated meshes and the extracellular matrix-based materials carry the hope of improvement in intraperitoneal incisional hernia repair. Before a general recommendation can be made, more clinical outcome results are necessary.

References

1. Luijendijk RW, Lemmen MHM, Hop WCJ, Wereldsma JCJ. Incisional hernia recurrence following "vest-over-pants" or vertical Mayo repair of primary hernias of the midline. World J Surg 1997, 21, 62–66
2. Leber GE, Garb JL, Alexander AI, Reed WP. Long-term complications associated with prosthetic repair of incisional hernias. Incisional hernioplasty with polyester mesh. Arch Surg 1998; 133: 378–382

3. Ammaturo C, Bassi G. The ratio between anterior abdominal wall surface/wall defect surface: a new parameter to classify abdominal incisional hernias. Hernia 2005; 9: 316–321
4. Gonzalez R,Fugate K, McClusky DW, Matt Ritter E, Lederman A, Dillehay D, Smith, CD, Ramshaw BJ. Relationship between tissue ingrowth and mesh contraction. World J Surg 2005; 29: 1038–1043
5. LeBlanc KA. Tack hernia: a new entity. JSLS 2003; 7: 383–387
6. Van Sickle KR, Baghai M, Mattar SG, Bowers SP, Ramaswamy A, Swafford W, Smith CD, Ramshaw BJ. What happens to the rectus abdominus fascia after laparoscopic ventral hernia repair? Hernia 2005; 9: 358–362
7. Klinge U, Conze J, Krones CJ, Schumpelick V. Incisional hernia: open techniques. World J Surg 2005; 29: 1066–1072
8. Klinge U, Junge K, Spellerberg B, Piroth C, Klosterhalfen B, Schumpelick V. Do multifilament alloplastic meshes increase the infection rate? Analysis of the polymeric surface, the bacteria adherence, and the in vivo consequences in a rat model. J Biomed Mater Res 2002; 63: 765–771
9. Robinson TN, Clarke JH, Schoen J,Walsh MD. Major mesh-related complications following hernia repair. Events reported to the Food and Drug Administration. Surg Endosc 2005; 19: 1556–1560
10. Klinge U, Klosterhalfen B, Muller M, Ottinger AP, Schumpelick V. Shrinking of polypropylene mesh in vivo: an experimental study in dogs. Eur J Surg 1998; 164: 965–969
11. Carbonell AM, Matthews BD, Dreau D, Foster M, Austin CE, Kercher KW, Sing RF, Heniford BT. The susceptibility of prosthetic biomaterials to infection. Surg Endosc 2005; 19: 430–435
12. Kramer K, Senninger N, Herbst H, Probst W. Effective prevention of adhesions with hyaluronate. Arch Surg 2002; 137: 278–282
13. Alponat A, Lakshminarasappa SR, Teh M, Rajnakova A, Moochhala S, Goh PM, Chan ST. Effects of physical barriers in prevention of adhesions: an incisional hernia model in rats. J Surg Res 1997 68(2): 126–132
14. McGinty JJ, Hogle NJ, McCarthy H, Fowler DL. A comparative study of adhesion formation and abdominal ingrowth after laparoscopic ventral hernia repair in a porcine model using multiple types of mesh. Surg Endosc 2005; 19:786–790
15. Demir U, Mihmanli M, Coskun H, Dilege E, Kalyoncu A, Altinli E, Gunduz B, Yilmaz B. Comparison of mesh materials in incisional hernia repair. Surg Today 2005; 35(3): 223–227
16. Jacob BP, Hogle NJ, Fowler DL. Tissue ingrowth and bowel adhesion formation animal comparative study: polypropylene vs. proceed vs. Parietex composite. Communication at 27th international congress of European Hernia Society, 2005
17. Holton LH, Kim D, Silverman RP, Rodriguez ED, Singh N, Goldberg NH. Human acellular dermal matrix for repair of abdominal wall defects: review of clinical experience and experimental data. J Long Term Eff Med Implants. 2005; 15(5): 547–558
18. Kolker AR, Brown DJ, Redstone JS, Scarpinato VM, Wallack MK. Multilayer reconstruction of abdominal wall defects with acellular dermal allograft (AlloDerm) and component separation. Ann Plast Surg. 2005; 55(1): 36–41
19. Arnaud JP, Tuech JJ, Pessaux P, Hadchity Y. Surgical treatment of postoperative incisional hernias by intraperitoneal insertion of dacron mesh and an aponeurotic graft. A report on 250 cases. Arch Surg 1999; 134: 1260–1262
20. Bellon JM,. Garcıa-Honduvilla N, Serrano N, Rodrıguez M, Pascual G, Bujan EJ. Composite prostheses for the repair of abdominal wall defects: effect of the structure of the adhesion barrier component. Hernia 2005; 9: 338–343
21. Bower CE, Reade CC, Kirby LW, Roth JS. Complications of laparoscopic incisional–ventral hernia repair. The experience of a single institution. Surg Endosc 2004; 18: 672–675
22. Napolitano L, Di Bartolomeo N, Aceto L, Waku M, Innocenti P. Use of prosthetic materials in incisional hernias: our clinical experience. G Chir 2004; 25(4): 141–145
23. Anthony T, Bergen PC, Kim LT, Henderson M, Fahey T, Rege RV, Turnage RH. Factors affecting recurrence following incisional herniorrhaphy. World J Surg. 2000; 24: 95–101
24. Frantzides CT, Carlson MA, Zografakis JG, Madan AK, Moore RE Minimally invasive incisional herniorrhaphy – A review of 208 cases. Surg Endosc 2004; 18: 1488–1491
25. Chelala E, Gaede F, Douillez V, Dessily M, Alle JL. The suturing concept for laparoscopic mesh fixation in ventral and incisional hernias: preliminary results. Hernia 2003; 7: 191–196
26. LeBlanc KA, Whitaker JM, Bellanger DE, Rhynes VK. Laparoscopic incisional and ventral hernioplasty: lessons learned from 200 patients. Hernia 2003; 7: 118–124
27. LeBlanc KA, Booth WV. Laparoscopic repair of incisional abdominal hernias using expanded polytetrafluoroethylene: Preliminary findings. Surg Endosc 1993; 3: 39–41
28. Heniford BT, Park A, Ramshaw BJ, Voeller G. Laparoscopic repair of ventral hernias. Nine years' experience with 850 consecutive hernias. Ann Surg 2003; 238: 391–400
29. Franklin ME Jr, Gonzalez JJ Jr., Glass JL. Use of porcine small intestinal submucosa as a prosthetic device for laparoscopic repair of hernias in contaminated fields: 2-year follow-up. Hernia 2004; 8: 186 – 189
30. Franklin Jr ME, Gonzalez Jr JJ, Glass JL, Manjarrez A Laparoscopic ventral and incisional hernia repair: An 11-year experience. Hernia 2005; 8: 23–27
31 Kapischke M, Prinz K, Tepel J, Tensfeldt J, Schulz T. Precoating of alloplastic materials with living human fibroblasts – ≠a feasibility study. Surg Endosc 2005; 19: 791–797
32. Dubay DA, Wang X, Kuhn MA, Robson MC, Franz MG. The prevention of incisional hernia formation using a delayed-release polymer of basic fibroblast growth factor. Ann Surg 2004; 240(1): 179–186
33. Badylak S, Kokini K, Tullius B, Whitson B. Strength over time of a resorbable bioscaffold for body wall repair in a dog model. J Surg Res 2001; 99(2): 282–287

Discussion

Halm: *In our series of patients that received open mesh repair, we looked at 66 patients who underwent re-operation for various reasons. When we compared intraperitoneal placement, which was open intraperitoneal mesh placement, with the extraperitoneal mesh placement, we found the shocking results that the groups were divided fifty/fifty. When going into the abdomen with the polypropylene intraperitoneally, 20% of the patients required small bowel resections just to get in.*

Kukleta: *That is why I would never use that. But I still respect the opinion of several people here who do and in no small numbers. Maybe Chowbey could comment on this?*
Chowbey: *I think there are certain factors which we have to keep it in mind when we are doing a laparoscopic incisional hernia. The first factor, I would say, is serosal injury to the bowel, while you are dissecting the bowel, taking the bowel out from the defect. It is very important, if there is a serosal injury; you take one suture and cover that defect so as not to leave it for later repair. The second important thing is the presence of a good omentum. If the omentum cannot be interposed between the bowel and the mesh, I think I would change to another type of mesh rather than using polypropylene. We never use scissors with diathermy for preparation, we use sharp scissors without diathermy and if there is bleeding we try to control it on another way. Most of the Indian patients do not have an inflammatory bowel disease as it is so common in this part of the world. I think inflammatory bowel disease, like diverticulitis, is also an important factor which plays a role. But I think it is the intra-operative injury of the bowel-serosa, which is the factor that can cause serious complications.*
Halm: *I understand that minimizing all surgical trauma decreases adhesions etc. What happens if you re-enter the abdomen? We have no hard data on that, we cannot randomize, so we should really study it critically.*
Köckerling: *We have also tested experimentally many of the meshes which are on the market. What we can conclude from our results is the necessity of special measures for intra-abdominal meshes: no membranes but large pores, we need light-weight polypropylene meshes, that allow perfect ingrowth, with no adhesions to the bowel if the bowel is not injured in the experimental setting. You just get adhesions to the great omentum and the liver but, not to the bowel. But we apply the same principles in the intra-abdominal use as in the inguinal use.*
Kukleta: *I had it in one of the slides. Probably one of the next steps is that we have to investigate light-weight meshes in the intraperitoneal position, and not any others.*

24.6 How to Treat the Recurrent Incisional Hernia Laparoscopically – Fixation

B. Ramshaw

Introduction

Inadequate fixation is one of the common causes of recurrence following laparoscopic ventral/incisional hernia repair. One unique aspect of the laparoscopic ventral/incisional hernia repair involves placing the mesh in the abdominal cavity and fixing it directly on the parietal peritoneum.

The fixation should be secure to prevent the mesh from eventuating out of the defect or migrating along the peritoneum, which is a mobile organ, or contracting too much, allowing a recurrence. The mesh should also be fixed adequately to the peritoneum to prevent internal herniation between the mesh and the abdominal wall. Besides recurrence, other issues concerning mesh fixation to be discussed include pain caused by bleeding in the abdominal wall, the amount of time it takes to perform the fixation, and the costs of the fixation devices and materials.

Keeping all of these issues in mind, the main goal of the fixation, especially for a recurrent hernia, is to prevent a hernia recurrence.

Body

A variety of fixation strategies have been proposed. The most common fixation strategy in the literature is a combination of full-thickness abdominal wall suture fixation with permanent sutures and point fixation with "tacks" [1–11]. There are other 5-mm point fixation options, including the Salute construct, the EndoAncho, and the 10-mm fixation options, including Ethicon and Autosuture hernia staplers and the Sofradim pariefix, which was the first device to deploy absorbable point fixation. There are also a few 5-mm absorbable point fixation devices now available or soon to be available. The use of glues for mesh fixation has also been proposed, but because the mesh is placed on the peritoneum, this has not become a popular fixation technique. Another strategy for fixation is to use point fixation only. Some surgeons use the point fixation only at the edges of the mesh while others have adopted the double crown technique, placing point fixation around the edges of the mesh and at the edges of the hernia defect [12–19]. Proponents of these strategies cite the

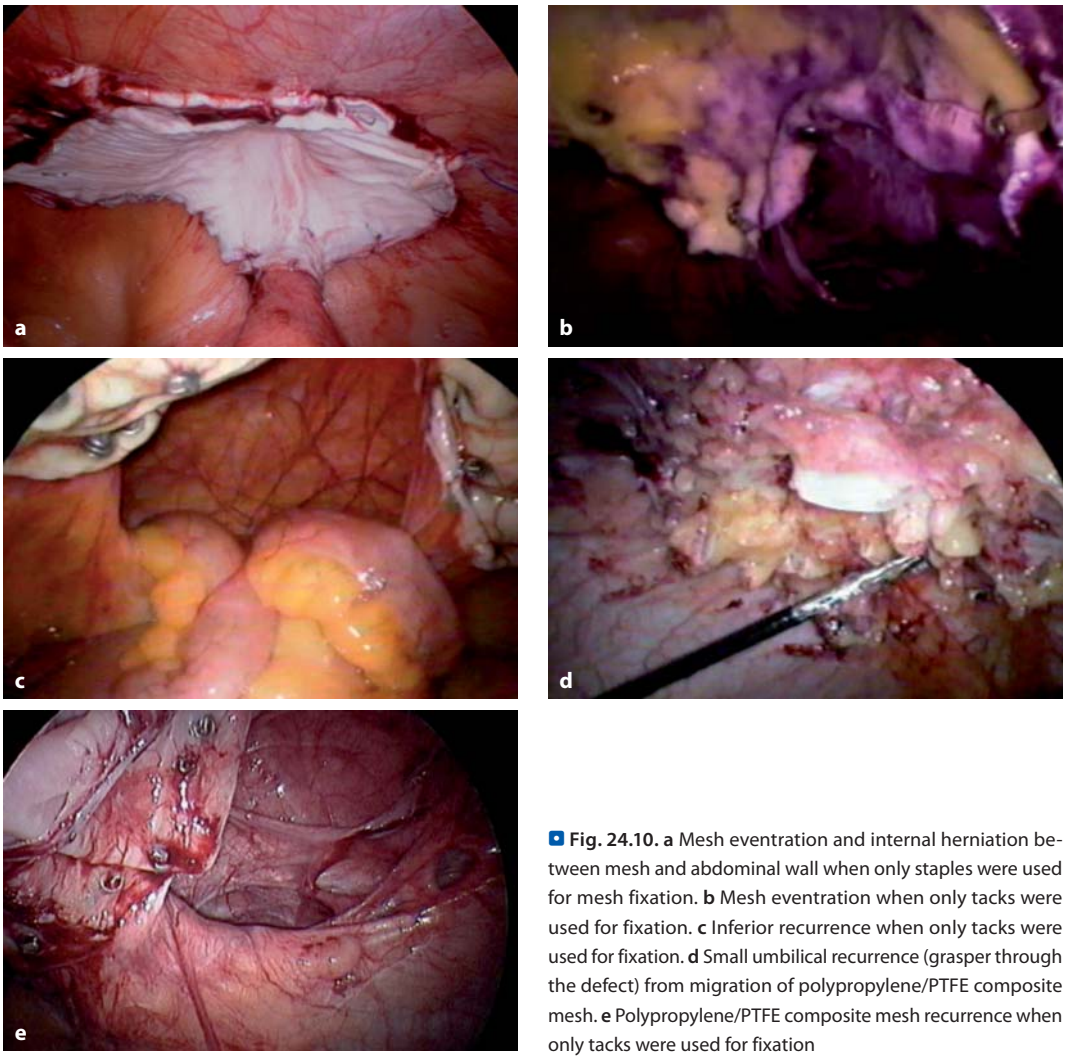


Fig. 24.10. a Mesh eventration and internal herniation between mesh and abdominal wall when only staples were used for mesh fixation. **b** Mesh eventration when only tacks were used for fixation. **c** Inferior recurrence when only tacks were used for fixation. **d** Small umbilical recurrence (grasper through the defect) from migration of polypropylene/PTFE composite mesh. **e** Polypropylene/PTFE composite mesh recurrence when only tacks were used for fixation

advantages of decreased operative time, decreased abdominal wall bleeding, and less postoperative pain. Proponents of suture fixation in addition to point fixation recognize these potential advantages; however, they believe these advantage are minimal and do not outweigh the increased potential for recurrence when sutures are not utilized. Several articles in the literature have documented a higher recurrence when only tacks were used, and this experience led to the use of sutures [6, 7, 9, 20]. Despite this, there are some series in the literature demonstrating a low recurrence rate with the double crown technique, although the authors of the series from Spain note that sutures are now used in select situations, such as subcostal hernia locations [13]. There are no published prospective randomized studies comparing these techniques although some re planned and/or underway. Regardless of the results of these studies, it will be difficult to convince surgeons who currently use sutures, including the author, to abandon the technique because of the numerous recurrences seen in their patients or patients from other surgeons when only point fixation was used. Some case examples are shown in Fig. 24.10.

Animal data have shown that sutures provide a statistically significant increase in mesh-holding force compared to tacks alone [21, 22]. The animal models

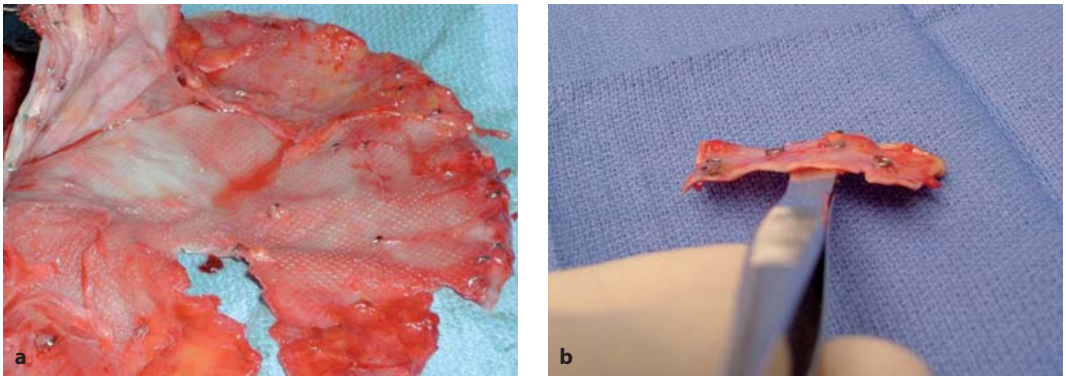


Fig. 24.11. a The mesh explant shows the abdominal wall side of the mesh and the amount of constructs available for fixation. **b** This mesh explant shows the abdominal wall side of the mesh and the amount of the tacks available for fixation

used were the rabbit and the pig. Even with the large animal model (the pig), it is easy to imagine that these same devices do not fix as well in a human, given the increase in preperitoneal fat and abdominal wall thickness compared to the pig models. Figure 24.11 show the amount of the fixation device (construct and tack) that is available to go through the peritoneum, the preperitoneal fat and into the muscle/fascia. Especially in the obese patient, who has increased intra-abdominal pressures and more preperitoneal fat, there is concern that a no-suture technique might lead to a higher likelihood of recurrence.

To minimize bleeding with suture placement, it is important to visualize the abdominal wall to identify and avoid the inferior epigastric vessels and their branches. Bleeding from accidental injury to an abdominal wall vessel is usually controlled with direct pressure and/or tying down the suture. Persistent bleeding can be controlled with suture ligation proximal and distal to the bleeding site, placing sutures through the same skin incision.

Suture site pain may be lessened by injecting local anesthetic prior to skin incision and by tying the knots gently to avoid entrapping nerves and tissue [23] Tying the knots gently might also help prevent a rare cause of recurrence – herniation at the suture site. Placing the suture about 1 cm inside the edge of the mesh and making sure the mesh covers the suture site should also help to prevent a suture site hernia recurrence. The exact interval between sutures will vary depending on the size and type of defect (Swiss cheese vs. single defect) and the amount of mesh overlap. In general, the larger the defect, the closer the suture interval should be. For example, when repairing a 1-cm recurrent umbilical hernia using a 10×15 cm mesh, the initial four sutures (top, bottom and each side) should provide adequate suture fixation. For a large single defect involving an entire midline incision, suture intervals of 3–5 cm is recommended. On the other hand, for a Swiss cheese defect of the same mid-line incision, an interval of 5–8 cm between sutures should be adequate.

Proper placement of the tacks or other point fixation devices includes placing the devices within 1 cm of each other inside the edge of the mesh to prevent internal herniation between the mesh and the abdominal wall. It is important to place the point fixation device as flush with the mesh as possible. Any portion of the tack that is hanging below the mesh could be a site for increased adhesion formation, or worse, could cause injury to abdominal organs. Bowel fistulas, apparently caused by exposed tacks, have been reported [24, 25]. Other complications from point fixation devices include pain, bleeding, tack site hernias, and inadvertent injury to organs outside the abdominal cavity, including the heart.

Conclusion

In summary, the best approach to prevent recurrence following the laparoscopic repair of a recurrent ventral/incisional hernia is to use both permanent full-thickness abdominal wall sutures and point fixation devices. Initially two to five sutures are placed on the mesh about 1 cm from the edge. After these sutures are brought out of the abdomen and tied down gently under the skin, the point fixation device is used to fix the mesh along the edges at 1 cm or less intervals. Additional sutures are then placed at the edges of the mesh at smaller intervals for large single-defect hernias and at larger intervals for

Swiss-cheese type and smaller hernia defects. Despite this opinion of a majority of experts in the literature, various other forms of fixation are being used and have similar published results. Prospective studies and new fixation options may lead to improved knowledge and better techniques for mesh fixation.

References

1. Bageacu S, Brenton C, Blanc P, et al. Laparoscopic repair of incisional hernia. A retrospective study of 159 patients. Surg Endosc 2002; 16: 345–348
2. Ben-Haim M, Kuriansky J, Tal R, et al. Pitfalls and complications with laparoscopic intraperitoneal expanded polytetraflurorethylene patch repair of postoperative ventral hernia. Surg Endosc 2002; 16: 785–788
3. Birgisson g, Mastrangelo MJ, Park A, et al. Obesity and laparoscopic repair of ventral hernias. Surg Endosc 2001; 15: 1419–1422
4. Bower CE, Kirby W, Reade CC, et al. Complications of laparoscopic incisional-ventral hernia repair. Surg Endosc 2004; 18: 672–675
5. Gonzalez R, Duncan T, Mason E, Ramshaw BJ, Wilson R. Laparoscopic versus open umbilical hernia repair. JSLS 2003; 7: 323–328
6. Heniford BT, Park A, Ramshaw BJ, et al. Laparoscopic repair of ventral hernias, nine years' experience with 850 consecutive hernias. Ann Surg 2003; 238(3): 391–400
7. LeBlanc KA, Rhynes VK, Whitaker JM, et al. Laparoscopic incisional and ventral hernioplasty: lessons learned from 200 patients. Hernia 2003; 7: 118–124
8. Park A, Birch DW, Lovrics P. Laparoscopic and open incisional hernia repair: A comparison study. Surgery 1998; 124(4): 816–822
9. Ramshaw BJ, Duncan TD, Esartia P, Lucas G, Mason EM, Miller J, Promes J, Schwab J, Wilson RA. Comparison of laparoscopic and open ventral herniorrhaphy. AM Surg 1999; 65: 827–832
10. Robbins SB, Gonzalez RP, Pofahl WE. Laparoscopic ventral hernia repair reduces wound complications. Am Surg 2001; 67(9): 896–900
11. Roth JS, Mastrangelo MJ, Park AE, Witzke D. Laparoscopic incisional/ventral herniorraphy: a five hear experience. Hernia 1999; 4: 209–214
12. Carbajo MA, Blanco JI, de la Cuesta C, Inglada L, Martin F, Martin JC, Toledano M Vaquero C. Laparoscopic treatment vs. open surgery in the solution of major incisional and abdominal wall hernias with mesh. Surg Endosc 1999; 13: 250–252
13. Carbajo MA, Blanco JI, Martin del Olmo JC, et al. Laparoscopic approach to incisional hernia. Surg Endosc 2003; 17: 118–122
14. Chari R, Chari V, Chung R, Eisenstat M. A case controlled study of laparoscopic incisional hernia repair. Surg Endosc 2000; 14: 117–119
15. Eitan A, Bickel A. Laparoscopically assisted approach for postoperative ventral hernia repair. J Laparoendo Adv Surg Tech 2003; 12(5): 309–311
16. Frantzides CT, Carlson MA, Zografakis JG, et al. Minimally invasive incisional herniorrhaphy. Surg Endosc 2004; 18: 1488–1491
17. Gillian GK, Geis WP, Grover G. Laparoscopic incisional and ventral hernia repair (LIVH): an evolving outpatient technique. JSLS 2002; 6: 315–322
18. Holzman MD, Eubanks S, Pappas TN, Purut CM, Reintgen K. Laparoscopic ventral and incisional hernioplasty. Surg Endosc 1997; 11: 32–35
19. Sanchez LJ, Bencini L, Moretti R. Recurrences after laparoscopic ventral hernia repair: results and critical review. Hernia 2004; 8: 138–143
20. Tagaya N, Mikami H, Aoki H, Kubota K. Long-term complications of laparoscopic ventral and incisional hernia repair. Surg Laparosc Endosc Percutan Tech 2004; 14(1): 5–8
21. Joels CS, Matthews BD, Kercher KW, Austin C, Norton HJ, Williams TC, Heniford BT. Evaluation of adhesion formation, mesh fixation strength, and hydroxyproline content after intraabdominal placement of polytetrafluoroethylene mesh secured using titanium spiral tacks, nitinol anchors, and polypropylene suture or polyglactin 910 suture. Surg Endosc 2005; 19(6): 780–785
22. van't Riet M, Steenwijk PJ, Kleinrensink GJ, Steyerberg EW, Bonjer HJ. Tensile strength of mesh fixation methods in laparoscopic incisional hernia repair. Surg Endosc 2002; 16(12): 1713–1716
23. Carbonell AM, Harold KL, Mahmutovic AJ, Hassan R, Matthews BD, Kercher KW, Sing RF, Heniford BT. Local injection for the treatment of suture site pain after laparoscopic ventral hernia repair. Am Surg 2003; 69(8): 688–691
24. Ladurner R, Mussack T. Small bowel perforation due to protruding spiral tackers: a rare complication of laparoscopic incisional hernia repair. Surg Endosc 2004; 18(6): 1001
25. DeMaria EJ, Moss JM, Sugerman HJ. Laparoscopic intraperitoneal polytetrafluoroethylene (PTFE) prosthetic patch repair of ventral hernia. Surg Endosc 2000; 14: 326–329
26. LeBlanc KA. Tack hernia: a new entity. JSLS 2003; 4: 383–387

Discussion

Kukleta: *Don't you think that the medialization that you describe in your group is a consequence of shrinkage? Because you fix it very well and you found it in nearly 90%. Nine out of ten patients had a substantial medialization. That would be the only positive effect of shrinkage.*

Ramshaw: *Actually, on a few re-operations which we had done with large meshes, we saw a little buckling in the mesh inside. So I don't think it is shrinkage, because I think it is a true natural healing contractor, just as we see with the skin. If you eliminate the intra-abdominal pressure, it contracts over time. So I don't think it is actually contraction of the mesh doing that.*

Frantzidis: *One issue that hasn't been raised with these very large hernias: Do you offer your patients a binder to reduce seroma formation and may help to incorporate the mesh into the tissue?*
Ramshaw: *With those very large defects I think that dense spaces are always going to fill with fluid. I offer patients a binder. I explain to them that it may be helpful in two ways, to eliminate those dense spaces and possibly with the security of eliminating movement that can cause especially early fixation pain postoperatively. So I definitively offer it and ask them to wear it. I don't make it mandatory, but if they wear it, I think they end up with a better result.*

Primary Inguinal Hernia

25 How to Create a Recurrence

25.1 Bassini

M. Bay-Nielsen, H. Kehlet

Introduction

For many years, repair of inguinal hernias was primarily based on Bassini-like repairs, aiming to re-enforce or re-establish a weak or absent posterior wall of the inguinal canal by using the anatomical structures bordering the defect, with many of the differences in the various open surgical techniques described being rather subtle.

Previous studies have shown recurrence rates of non-mesh repairs in the range of 20–30% with highest recurrence rates after Bassini repair [1–4], and in most large series, the rate of operation for a recurrence approaches 16–18%, confirming the high recurrence rates of past non-mesh inguinal hernia repairs.

This study presents the results after Bassini repair, based on data from the Danish Hernia Database.

Material and Methods

The analysis was based on 74,131 elective inguinal herniorraphies recorded in the Danish Hernia Database in the period 1 Jan. 1998 to 30 June 2005 (■ Table 25.1). The setup and organization of the Danish Hernia Database is described elsewhere [5, 6]. In brief, the database records basic information, including type of repair, on all (> 98%) inguinal and femoral herniorraphies performed in Denmark, based on schemes filled out by the operating surgeon at time of operation. The database uses rate of operated recurrences as a proxy for recurrence and patient-specific observation time is calculated by the use of unique social security numbers. Cumulative re-opera-

■ **Table 25.1.** Number of herniorraphies, age, operative findings and rate of operation for recurrence. Danish Hernia Database 1 Jan. 1998 to 30 June 2005

	Bassini	Lichtenstein
No. of herniorraphies	1383	48,400
Median age	56 years	58 years
Direct/indirect hernias	60/40%	56/44%
Primary/recurrent hernias	88/12%	89/11%

tion rates are shown as Kaplan-Meier plots and compared by use of log rank test. Hazard ratios for risk factors are calculated using multivariate Cox proportional-hazards regression. $P < 0.05$ is considered significant.

Results

Of the 74,131 elective inguinal herniorraphies recorded in the Danish Hernia Database, 1383 (1.8%) were Bassini repairs. The use of Bassini repairs declined, from 4% in 1998, to < 0.5% in 2005, concomitant to an increase in the use of Lichtenstein repairs from 34 to 78% (Fig. 25.1). Only small differences were found, comparing age, ratios direct/indirect hernias and primary/recurrent repairs for Lichtenstein and Bassini repairs.

Kaplan-Meier estimates of re-operation rates show a significantly higher re-operation rate after Bassini repair, compared to Lichtenstein repair (Fig. 25.2) while analysis of re-operation rates after Bassini repair, shows a re-operation rate after repair of direct inguinal hernia being twice that of indirect hernias, and recurrent repairs having almost three times the re-operation rates of primary hernias (Table 25.2).

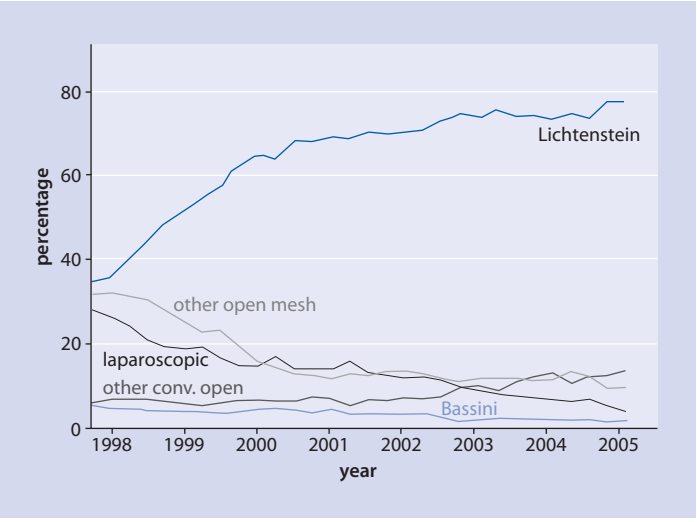


Fig. 25.1. Changes in use of operative techniques, Danish Hernia Database Jan. 1988 to June2005. n = 74,131 elective inguinal herniorraphies

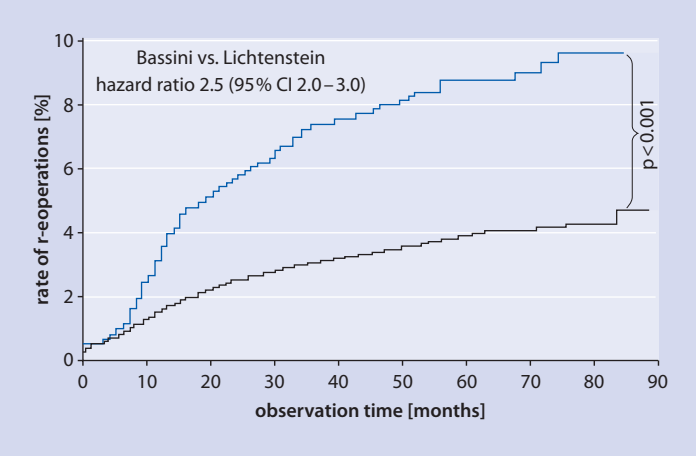


Fig. 25.2. Kaplan-Meier estimates of re-operation rates, Bassini (n= 1383) and Lichtenstein (n = 48,000) repair of elective inguinal hernia, Danish Hernia Database 1 Jan. 1998 to 30 June 2005. $p < 0.05$ comparing Bassini and Lichtenstein repair (log rank test)

Table 25.2. Risk factors, comparing elective inguinal Bassini (n = 1383) and Lichtenstein repairs (n = 48,400)

Risk factor	Hazard ratio, comparing Bassini and Lichtenstein (95% CI)
Age (> 65, ≤ 65)	1.2 (0.8–1.8)
Direct vs. indirect	2.1 (1.4–3.1)
Recurrence vs. primary	2.7 (1.7–4.2)

Discussion

These data from the Danish Hernia Database confirm a high rate of reoperation after Bassini repair (10% after 7 1/2 years).

Although mesh implantation in itself has been suspected to be a factor in chronic postherniorraphy pain, previous studies do not confirm this relation [7] and no evidence exists showing an advantage of the Bassini repair in other outcome parameters.

As a consequence of the unacceptably high risk of recurrence after Bassini (and other open non-mesh repairs) and the absence of data supporting the use of Bassini repair, the use of Bassini repair should be abandoned.

Conclusion and Consequences

To create a recurrence after a Bassini-type inguinal herniorraphy is easy: you just do it and leave the rest to time and gravity. The use of Bassini repair should be abandoned.

References

1. Tran VK, Putz T, Rohde H (1992) A randomized controlled trial for inguinal hernia repair to compare the Shouldice and the Bassini-Kirschner operation. Int Surg 77: 235–237
2. Paul A, Troidl H, Williams JI, Rixen D, Langen R (1994) Randomized trial of modified Bassini versus Shouldice inguinal hernia repair. The Cologne Hernia Study Group. Br J Surg 81: 1531–1534
3. Hay JM, Boudet MJ, Fingerhut A, Poucher J, Hennet H, Habib E, Veyrieres M, Flamant Y (1995) Shouldice inguinal hernia repair in the male adult: the gold standard? A multicenter controlled trial in 1578 patients. Ann Surg 222: 719–727
4. Strand L (1998) Randomized trial of three types of repair used in 324 consecutive operations of hernia. A study of the frequency of recurrence. Ugeskr Laeger 160: 1010–1013
5. Bay-Nielsen M, Kehlet H, Steering committee of the Danish hernia data base (1999) Establishment of a national Danish hernia data base: preliminary report. Hernia 3: 81–83
6. Bay-Nielsen M, Kehlet H, Strand L, Malmstrom J, Andersen FH, Wara P, Juul P, Callesen T (2001) Quality assessment of 26,304 herniorrhaphies in Denmark: a prospective nationwide study. Lancet 358: 1124–1128
7. Bay-Nielsen M, Nilsson E, Nordin P, Kehlet H (2004) Chronic pain after open mesh vs. sutured repair of indirect inguinal hernia in young males. Br J Surg 91: 1372–1376

Discussion

Campanelli: *I don't agree with your conclusion. If you follow the original steps of Bassini repair, it's a perfect repair. You can do it ambulant under local anesthesia and you can achieve the same results as with a mesh repair. So what are your specific steps of Bassini repair?*
Bay-Nielsen: *It's not my repair. I just described how surgeons do the Bassini in Denmark.*
Kingsnorth: *I think the problem is that you don't have control over the surgeons. But you have more control over surgeons doing Lichtenstein because they are able to apply the principle of the repair better and achieve results close to Lichtenstein, while general surgeons don't appear to be able to apply the basic principles of the Bassini to get his results.*
Bay-Nielsen: *It gives us the ability to say: you do a Bassini repair, these are your results, and you should do something else.*
Read: *I was surprised that the incidence of indirect hernia was less than the incidence of direct hernia in the population who are operated upon. This is against the main experience with this type of hernia.*
Bay-Nielsen: *I cannot comment on that.*

25.2 Shouldice

R. Bendavid

Introduction

To the serious and dedicated surgeon, it would be unthinkable to expect a career without being competent in the performance of a pure tissue repair for inguinal hernias. It would be unrealistic if not careless. To accomplish this competence will not be easy, for it will take valiant and diligent effort not to be overwhelmed or intimidated by the manufacturers and salesmen of surgical prosthetics, instruments and implements. Although it is a necessary evil of marketing strategies to sell indiscriminately if not wantonly, the onus is on the surgeon to be steadfast and show perspicacity, for he is the guardian of his patient's most prized possession: his well-being.

This is not a plea for blind conservatism but a call for an informed liberal choice. A pure tissue repair is always a proper operation when the pathology consists of an indirect inguinal hernia. This is nearly always the case for children, young adults, females as well as many adults who present with a pure indirect inguinal hernia. That is, unless an indirect sac has a neck wide enough to involve the posterior inguinal wall. The use of mesh is properly indicated for direct inguinal hernias, femoral hernias whether or not associated with an indirect inguinal hernia and recurrent hernias. Perhaps the most important reason to be adept with a pure tissue repair is that it imparts knowledge that will enable you to manage any situation in the groin, particularly during emergencies when incarceration, strangulation or bacterial contamination of the operative site may proscribe the use of prostheses. Important, too, is that you are the one to decide what is best for your particular need. No one else has made that decision for you at a sales strategy powwow!

The number of pure tissue repairs derived from Bassini's technique is now well over 80 and counting [1]. All have quietly disappeared but for the Shouldice repair. The Shouldice repair itself has been recognized by many, quietly, to be really a Bassini with no difference to justify a new appellation. The fact, too, is that no particular technique was ever described by Shouldice himself. The present discussion and recommendations will apply therefore to the Shouldice as well as the Bassini repairs. I always find it ironic to read that the Shouldice repair yields better results than the Bassini repair. The good results of the Shouldice Hospital are, in no small measure, the result of an expertise acquired from doing thousands of procedures to the exclusion of all other surgical operations, by a team of dedicated surgeons. Who among us does not recall that the Bassini repair was taught as a "modified Bassini" and therefore, by not resecting the cremasters and not opening the posterior inguinal wall but imbricating it instead, one did a corruption of that repair which evidently leads to poorer results! Shouldice respects the very steps introduced by Bassini, adding a second running suture in the reconstruction for good measure.

Another digression, about the McVay repair this time, begs to be made since the dissection is entirely a Bassini-Shouldice dissection without the resection of the cremaster. It is still performed by a few surgeons, though their number is dwindling. McVay's contribution was made at a time when mesh was not in common use and, when used, was fraught with and evoked unwarranted fears. The McVay contribution was one of exquisite understanding of the anatomy of the groin. As a hernia repair, it was beset by a moderate incidence of recurrence, suffered from too much tension and pain and was associated with a constant, if low, incidence of femoral vein complications. Most notable is the fact that a recurrence from a true McVay repair is always the most difficult dissection one can expect while doing open surgery on a recurrent inguinal hernia.

How does one then, "create" a recurrence while performing a Shouldice repair? The answer must be provided under five headings:

- Magnitude of the problem.
- Corruption of the established technique.
- Shouldice against odds. Attempting to perform a Shouldice repair in a class of hernia where a pure tissue repair is known to yield poor results.
- Inadequate knowledge of the anatomy and pathology of the groin.
- Specifics.

Magnitude of the Problem

The incidence of recurrence following inguinal hernia repairs varies between 8 and 33%, and depends on the operative technique [2]. In the hands of the Shouldice Hospital surgeons who rely on the Shouldice repair only, that is to say when mesh is not used, that incidence varies between 1 and 20% [3]. Looking at pre-mesh days (up to 1983), the results can be assessed from ◘ Table 25.3.

With reference to recurrence rates following primary inguinal hernia repairs, the Shouldice Hospital claims an incidence of less than 1%. However, I have

Table 25.3. Shouldice Hospital: own re-recurrences from 1057 operations [3]

1x	recurrent inguinal hernia	18/775	2.3%
2x		15/212	7%
3x		6/49	12%
4x		1/14	7%
5x		1/5	20%
6x		0/2	0%??

never seen a study emanating from the Shouldice group analyzing the extent of the follow-up that would be acceptable to a statistician. My own attempt at following 400 patients from their records alone, from 1986 to 1996, yielded a dismal follow-up of 10% only [4]. The literature reports incidences of recurrences as high as 12.5% at 4 years [5]. On a yearly basis, 13–15% of all patients presenting at or, referred to the Shouldice Hospital are already recurrences and persisting with the use of the Shouldice repair may well be a way of "creating" a hernia! At least by their own admission.

Corruption of Established Techniques

A chronic bane in surgery is the blind improvisation of a particular step in a well-established operation. This variation is often perpetrated without the benefit of a defining study to confirm the premise or pretence of that variation. This is seen when the Bassini or Shouldice repairs are carried out without the resection of the cremaster or without the division of the posterior wall of the inguinal canal or when reconstruction is carried out by "imbrication" of the transversalis fascia or when the external oblique aponeurosis is approximated under the cord, leaving the latter in the subcutaneous position. These shortcuts usually lead to shortcomings.

Shouldice Against Odds

The Shouldice Hospital, which remains a bastion of pure tissue repairs, has finally conceded that, in fact, there are situations when "mesh is indicated"! Like a man who has long been used to suspenders, wearing a waist belt only feels somewhat unsafe still! Their own statistics do reveal at last that mesh must be the order of the day when dealing with recurrences. Often, unfortunately, recurrences are due to missed and overlooked hernias during a previous attempt at herniorrhaphy. If such a missed hernia is an indirect inguinal sac, a Shouldice repair may well be safely attempted. A Shouldice repair should not be attempted in the presence of a direct inguinal hernia. The reason will be examined under the next heading. The Shouldice repair should not be modified to include the ligament of Cooper. This has been proposed by a surgeon, resulting in a McVay type of repair in order to correct a co-existent femoral hernia [6]. In such a modification, the resulting operation would be closer to a McVay than to a Shouldice. Though this modification may perhaps handle a small femoral hernia, a double-blind study was never carried out and cases needing mesh were excluded from the series since they were too large to handle by a suture repair! In other words patient selection took place, negating the study and casting much doubt on the results [6, 7].

Inadequate Awareness of Inguinal Anatomy and Pathology

It is often said that the anatomy of the groin is the most difficult with which one has to contend. That is probably the case and this is reflected by the unaltered incidence of hernia recurrences in the past three decades despite the addition of prosthetic sheets, prosthetic gadgets and laparoscopic mastery [8]. Adequate textbooks abound which discuss the anatomy of the groin; however, many can be confusing, unless one is to dedicate the necessary time to study them. The anatomy lab and the operating room are ideal places to identify, confirm and crystallize the acquired knowledge.

Hernia pathology used to imply progressive changes in anatomy secondary to the mechanical strains and stresses of daily life, work and ageing. Today, there is a resurgence of interest in the biological and metabolic aspect of hernia disease, particularly, more recently, at the cellular, nuclear, chemical and molecular level. Scientific activity is centred on the nature and changes taking place within the collagen tissue as a result of inherited factors or external deleterious stimuli e.g. smoking [9].

Specifics

How then, specifically, does one "create a recurrence" during a Shouldice-Bassini repair? The answer is, of necessity, speculative, since no-one has ever gone about

to knowingly create such hernias. Logic and a smattering of detective work will help.

1. The skin incision is often suggested to be, in most textbooks, 2 to 3 cm above a line joining the anterior superior iliac spine to the pubic spine. Personal and practical experience dictates that the incision must be on and along that very line from the anterior superior iliac spine to the very level of the pubic spine to provide an optimal exposure of the relevant site of surgery. This location displays easily the medial portion of the floor of the canal where recurrences occur most often. The undersurface of the ligament of Poupart becomes clearly visible at the level of the femoral triangle, where a femoral hernia can be routinely searched for and excluded. An incision so situated minimizes tension by the retractors, for these are often the very source of marked discomfort during surgery under local anaesthesia.
2. Resection of the cremasterics permits the accurate identification, without fail, of an indirect sac at the medial aspect of the cord at the internal ring. In the series of 1057 recurrences seen and reported by Obney and Chan [3], 37% of the recurrences turned out to be indirect inguinal hernias (missed hernias?)! 45% were direct inguinal hernias, 8% were femoral hernias (most likely overlooked also) and in 10%, two or more hernias were discovered.
3. Division of the posterior wall of the inguinal canal allows the examination of the preperitoneal space, the identification of femoral pathology and rare hernias. But above all, it affords the identification of good tissue layers which will allow for a solid repair. The posterior inguinal wall will not be made up of a weak, thin and translucent transversus abdominis fascia and its posterior layer, the true transversalis fascia which is part of the endopelvic fascia.
4. Division of the cribriformis fascia is a small surgical step, requires little time and pays off handsomely in terms of discovering a femoral hernia which would otherwise have become a missed hernia and therefore a recurrence.
5. Nowadays, the division of the posterior wall of the inguinal canal must come under scrutiny. Is it a necessary step in all patients undergoing the Bassini or Shouldice repairs? Many of the Shouldice surgeons with whom I shared surgical opinions over many years varied in their approach. Oftentimes, when the wall and the tissues were good, the wall was not divided. Why divide a good structure and run the risk of a recurrence which, if it takes place, will do so at the very medial end of this wall just lateral to the pubic spine? Some surgeons take the middle of the road by dividing the posterior inguinal wall halfway only. I very rarely divide the posterior inguinal wall in women because they seldom have direct hernias or in patients who have an indirect inguinal hernia with a good posterior wall and in children. In women the occurrence of a direct hernia is low, 1 out of 12 primary inguinal hernias compared to 1 out of 2 in men [10]. If one considers that women make up 5% of the hernia population, their chance of having a direct inguinal hernia is 0.4% of all inguinal hernias!
6. When direct inguinal hernias are present, they must be considered to be secondary to metabolic, genetic and chemical factors which lead to tissue degeneration and therefore hernia formation. In these patients, the use of prostheses is justified and recommended [9]. We have seen above that in the hands of the Shouldice surgeons, the incidence of re-recurrence can be between 2.3 and 20% when they repair recurrences without mesh. The patients at their hospital present with a recurrence number 12–16% of the total number of patients [11]. Yet, mesh was used in only 0.86% of recurrent indirect inguinal hernias and in 5.78% of direct inguinal hernias. Somehow, logic is being ignored and a reasonable conclusion would be that the Shouldice Hospital is instrumental in "creating hernias" while doing a Shouldice repair [12]!
7. The relaxing incision is a most trusted manoeuvre in relieving tension in pure tissue repair. Introduced by Wolfler in 1892, it was re-introduced by Berger in 1902 and Halsted in 1903 [13]. It has since been adapted in 12 variations [13]. Koontz confirmed experimentally that" not only does an incision over fascia over good muscle not weaken the structure, but the fascial covering is rapidly regenerated [14]. I have used a relaxing incision in over 2200 cases without a single cause for regret. I have often seen, while performing a generous relaxing incision as far as the level of the internal ring that an interstitial or low Spigelian hernia becomes evident which will invariably require a mesh repair. In this case, the hernia was not "created", it was discovered!

Conclusion

Alexis Carrel, the Nobel laureate in medicine in 1912, remarked that "the very fame of a specialist renders him dangerous". I thought a long time about this. Did he mean that man becomes welded to his thoughts and techniques and promotes them to the reckless exclusion

of all logic and deference to worthy and newer challenges? This may well be. It is a form of slavery from which man must detach himself. For his sake, for the sake of science, but above all for the sake of man.

References

1. Bendavid R. New techniques in hernia repair. World J Surg 1989; 13: 522–531
2. Weber A, Garteiz D, Valencia S. Epidemiology of inguinal hernia: A useful aid for adequate surgical decisions. In: Bendavid R, Abrahamson J, Arregui ME, Bernhard J, Phillips EH (eds) Abdominal wall hernias. Springer, Berlin Heidelberg New York, 2001
3. Obney N, Chan CK. Repair of multiple time recurrent Inguinal hernias with reference to common causes of recurrence. Contemp Surg 1984; 25: 25–32
4. Bendavid R. Introduction to pure tissue repairs. In: Bendavid R, Abrahamson J, Arregui ME, Bernhard J, Phillips EH (eds) Abdominal wall hernias. Springer, Berlin Heidelberg New York, 2001, p 353
5. Champault G, Barrat C, Catheline JM, Rizk N. Groin hernias- 4 year result of two randomized prospective studies. Hernia 1998; 2: 19–23
6. Welsh DRJ, Alexander MAJ. The Shouldice repair. Surg Clin N Am 1993; 73: 451–469
7. Degani CT. Femoral hernia repair. A five year prospective study. American Hernia Society Meeting; February 9–12/2005 in San Diego, Ca. Abstract #19–1
8. Schumpelick V. Herniosis: Recent understanding. San Diego conference of American Hernia Society, Feb 9–12, 2005:Abstract Book, # 46-I, page 114
9. Bendavid R. The unified theory of hernia formation. Hernia 2004; 8: 171–176
10. Glassow F. Inguinal hernia in the female. Surgery, gynecology and obstetrics. 1963; 116: 701–704
11. Bendavid R. The Shouldice repair. In: Nyhus LM, Condon RE (eds) Hernia IV. Lippincott, Philadelphia, 1995, p 223
12. Shouldice EB. The Shouldice repair for groin hernias. Surg Clin N Am 2003; 83: 1181
13. Bendavid R. Relaxing incisions. In: Bendavid R, Abrahamson J, Arregui ME, Bernhard J, Phillips EH (eds) Abdominal wall hernias. Springer, Berlin Heidelberg New York, 2001, pp:343–346
14. Koontz AR. Hernia. Appleton-Century-Crofts; New York, 1963, pp 52–53

Discussion

Kingsnorth: *Does the suture material you use contribute to recurrence? You still use stainless steal wire?*
Bendavid: *I have switched to polypropylene suture material. The danger of stainless steel is that you have sharp ends and you very often get pricked.*
Miserez: *I have two questions. Do you advocate doing anything with the posterior wall in the young adult and in women, or do you just leave like it is? Do you still use four layers for a Shouldice repair?*
Bendavid: *In women, direct hernias are never a problem, they normally don't occur. In indirect hernias we just narrow the internal ring and we rarely see a recurrence. Concerning the second question: we still do the four-layer repair. In about 15 to 20% we add a relaxing incision to make it as tension-free as possible.*
Flament: *Five years ago during a meeting organized by Prof. Lange, 500 surgeons were asked what to do with a 2 cm lateral hernia and 95% answered that they would do a Shouldice repair. This is just a statement.*
Kingsnorth: *This is an important statement.*
Kurzer: *When we talk about the Shouldice or the Bassini repair, we talk about procedures which have a high efficacy, but only in the hand of experts. But a hernia repair is only effective if it can be spread easily to the rest of the surgical community. The advantage of the onlay mesh repair is that it can be spread to the general surgeon and still gives good results. The suture repairs have efficacy but only the onlay mesh repair has effectiveness as an operation in the surgical community.*
Bendavid: *I agree with you. The Lichtenstein repair can be done even by worse surgeons with good results. There is no doubt that the Shouldice is not an easy operation.*
Chan: *There are two things that I should like to say. First of all, direct hernia recurrence is not only from a Shouldice repair. If you use big bites and put too much tension on your suture, you will have a medial recurrence with any type of repair. Furthermore, you have to be aware not to miss a small indirect hernia, which still accounts for several recurrences. The third is that doing the Shouldice repair you may open the femoral canal by putting too much tension on your suture line. If you want to see the anatomy and the exact technique, come to the Shouldice Hospital and follow us for a week or two. Mesh is seldom necessary, we use mesh in only about 1% of the cases.*
Kehlet: *I want to come back to the results of the two nationwide registers we have, Sweden and Denmark. Here we see that Shouldice is a catastrophe. Do you really still recommend the Shouldice? When you do the Shouldice it might provide good results because you are experts. But it has been proven in several studies that it does not function in the general community.*
Bendavid: *The registers just reflect what you put in them. But you don't have any control over the surgeons.*
Deysine: *You cannot learn a Bassini or a Shouldice from a book or a drawing. The only way for a general surgeon to learn the Shouldice is to go to Toronto and to watch you operating. In the region where I live nobody can do a Shouldice besides Berliner.*

Bendavid: *I agree, the Shouldice is not an easy operation and you have to move and learn it from an expert.*
Schumpelick: *I would like to comment on Dr. Kehlet. I agree with you. Very often where Shouldice is written about, it is no Shouldice. A lot of surgeons do the operation with no knowledge of anatomy. We re-operated more than 200 of these cases. I think the name Shouldice is not the operation of Shouldice.*
Muschaweck: *Dr. Kehlet, I think the Shouldice is not a catastrophe. You have to do the Shouldice correctly and then you will have excellent results.*
Bendavid: *The advantage of having learned the Shouldice technique is that afterwards you are able to manage any problems in the groin and use any other technique of repair, including all the mesh techniques.*
Kehlet: *We are here as hernia experts and we have to give advice for hernia surgeons around the world. I do not say that Shouldice is a disaster in expert hands, but when every surgeon is doing it, it is probably not a Shouldice at all and they should not do it or even try to do it.*
Kingsnorth: *A lot of surgeons want to have a fall-back operation when they do not want to proceed with a Lichtenstein repair. In those countries where surgeons believe that they should not put a mesh in a group of patients, they need a tissue repair. There is no doubt that the Shouldice is the best tissue repair if it is applied correctly. In countries where surgeons believe that the Lichtenstein can be applied universally, the Shouldice repair is irrelevant. But in countries where a tissue repair is still supported by a group of surgeons, it is their duty to apply the correct Shouldice, which unfortunately is quite difficult.*

25.3 Lichtenstein

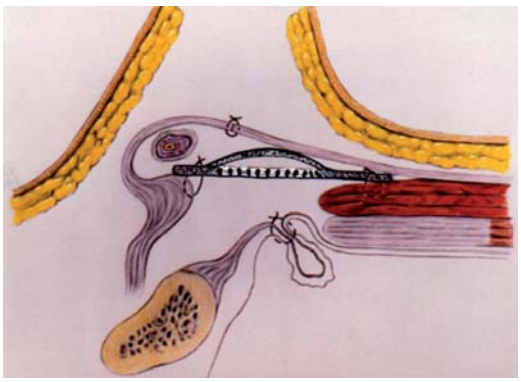
P. Amid

25

Introduction

Lichtenstein tension-free hernioplasty began in 1984. In the late 1980s, analyzing data from our own hernia registry, published in 1987, we identified the following flaws [1].

1. The mesh did not extend beyond the pubic tubercle to overlap the pubic bone.
2. The mesh was too small (only 5×10 cm) to provide enough mesh tissue contact beyond the inguinal floor.
3. The mesh was kept flat (◘ Fig. 25.3, broken line), and, therefore, was subject to tension when the patient stood up from the supine position of the operation.
4. The upper edge of the mesh was fixed using a continuous suture, which potentially left the iliohypogastric nerve at risk.
5. Passing the genital nerve and external spermatic vessel through a gap along the suture line of the mesh with inguinal ligament exposed the nerve to potential risk of entrapment.



◘ **Fig. 25.3.** Cross-section of the tension-free repair demonstrating an inverted direct hernia sac and the dome-shaped laxity of the mesh versus a completely flat mesh (dotted line)

In 1989, to correct the above problems, a set of principles (outlined below) was established by our group, employed with satisfactory results, and reported in 1993 [2].

Key Principles of the Lichtenstein Tension-Free Repair

1. Use a large sheet of mesh that will extend approximately 2 cm medial to the pubic tubercle, 4–5 cm above the Hesselbach triangle, and 5–6 cm lateral to the internal ring (◘ Fig. 25.4). We suggest using a 7×15 cm sheet of mesh for easy handling, then trimming 3–4 cm from its lateral side.

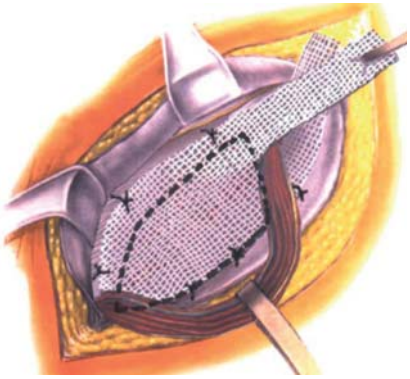


Fig. 25.4. Extension of mesh beyond the boundary of the inguinal floor (dotted line) 1 1.5–2.0 cm medial to the pubic tubercle, 2 4.0–5.0 cm above the inguinal floor, 3 5.0–6.0 cm lateral to the internal ring

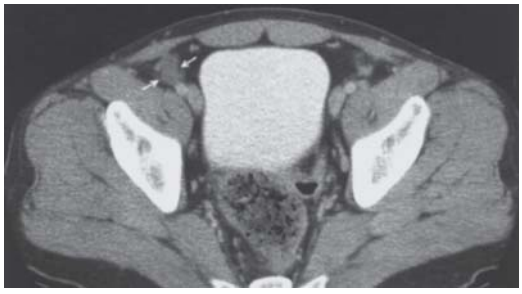


Fig. 25.5. CT scan image of a meshoma (*above*). The explanted meshoma (*below*)

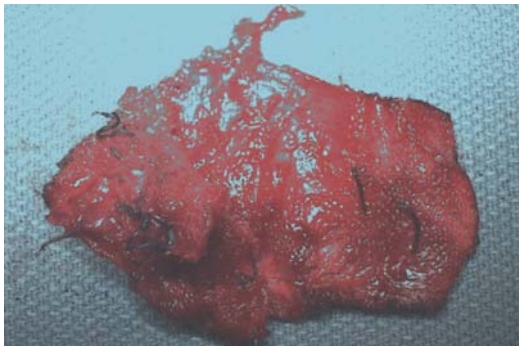


Fig. 25.6. Neuro-anatomy of the inguinal canal

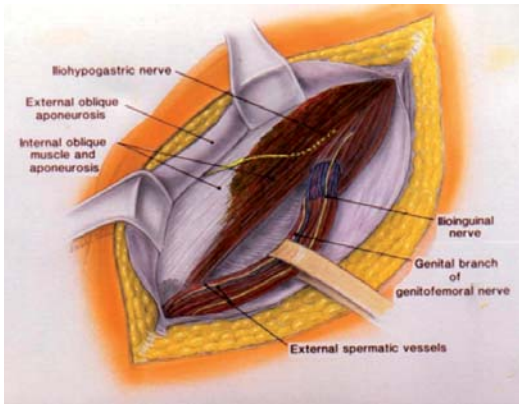


Fig. 25.7. Paravasal nerves within the lamina propria of vas deferens

2. Cross the tails of the mesh behind the spermatic cord to avoid recurrence lateral to the internal ring (see Fig. 25.4). Suturing the tails together in a parallel position, without crossing, is a known cause of recurrence lateral to the internal ring area.
3. Secure the mesh with two interrupted sutures on the upper edge and one continuous suture with no more than three to four passes on the lower edge of the mesh to prevent folding and movement of the mesh in the mobile area of the groin (see Fig. 25.4). Fixation of the mesh prevents movement, folding, and wadding of the mesh (meshoma) (Fig. 25.5), which can cause chronic pain and recurrence of the hernia [3].
4. Keep the mesh with a slightly relaxed, tented up, or buckled configuration (see Figs. 25.3, 25.4) to counteract the forward protrusion of the transversalis fascia when the patient stands up from the intra-operative supine position, and to compensate for contraction of the mesh.
5. Visualize and protect the ilioinguinal, iliohypogastric, and genital nerves throughout the operation (Fig. 25.6). The iliohypogastric nerve can be identified easily, while the external oblique aponeurosis is being separated from the internal oblique layer to make room for the mesh. Because of a natural anatomic cleavage, separation of these two layers from each other is easy, fast, and bloodless. The most vulnerable part of the iliohypogastric nerve is its intramuscular segment (Fig. 25.6, dotted line), which runs along the lower edge of the internal oblique muscle (the so-called conjoint tendon). Passing a suture through the internal oblique muscle to approximate this layer to the inguinal ligament (during tissue approximation repairs) to a plug (during mesh plug repair) or to the upper edge of the mesh (during Lichtenstein repair) is liable to injure the intramuscular portion of the iliohypogastric nerve with the needle or entrap the nerve with the suture [4]. The genital nerve is protected by not removing

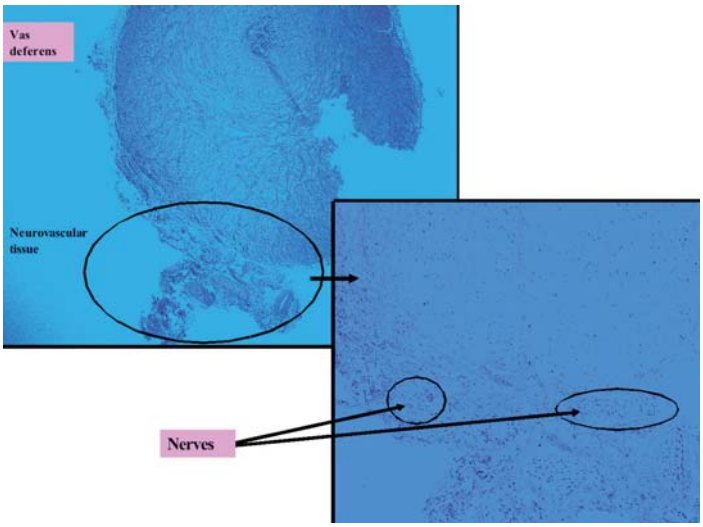


Fig. 25.8. Central narrowing of mesh in vertical direction with the mesh stretched in horizontal direction

the cremasteric muscle and keeping the easily visible blue external spermatic vein with the spermatic cord while it is being lifted from the inguinal floor [4]. Removing the cremasteric muscles exposes the genital nerve, paravasal nerves (Fig. 25.7), and the vas deferens to the mesh, which may lead to chronic inguinodynia, orchalgia, and/or possible infertility [5] respectively. The ilioinguinal nerve can easily be located over the spermatic cord. Manipulating and lifting the nerve from its natural bed will increase the risk of perineural fibrosis and chronic postherniorrhaphy inguinodynia [4].

Causes of Recurrence after Lichtenstein Tension-Free Hernia Repair

Causes of recurrence can be grouped in two categories: (1) material-related causes and (2) technique-related causes.

Material-Related Causes

- Mesh shrinkage: According to our clinical and laboratory studies reported in 1995, after implantation in vivo, mesh shrinks by approximately 20%. Shrinkage of mesh can lead to recurrence of hernia. Recurrence, however, can be prevented by extending the mesh well beyond the boundary of the inguinal floor.
- Mesh deformity related to the textile engineering of the mesh: Certain structural designs of meshes leads to narrowing of the mesh in the perpendicular direction of stretching the mesh (Fig. 25.8). As a result the narrowed centre of the mesh can pull away from its attachment to the host tissue and lead to recurrence.

Technique-Related Causes

These include:

1. Failure to extend the mesh for approximately 1.5–2.0 cm medial to the pubic tubercle, 4–5 cm above the inguinal floor, and 5–6 cm lateral to the internal ring.
2. Failure to keep the mesh slightly relaxed or buckled up to account for forward protrusion of abdominal wall in response to increased intra-abdominal pressure when the patient stands up from the surgical supine position and begins routine daily activities.
3. Inadequate mesh fixation that can lead to wrinkling of the mesh and recurrence of hernia.

References

1. Amid PK. Lichtenstein tension-free hernioplasty: Its inception, evolution, and principles. Hernia 2004; 8: 1–7
2. Amid PK, Shulman AG, Lichtenstein IL. A critical scrutiny of the open tension-free hernioplasty. Am J Surg 1993; 165: 369–371

3. Amid PK. Radiologic images of meshoma. A new phenomenon causing chronic pain after prosthetic repair of abdominal wall hernias. Arch Surg 2004; 139: 1297
4. Amid PK. Causes, prevention, and surgical treatment of postherniorrhaphy neuropathic inguinodynia: Triple neurectomy with proximal end implantation. Hernia 2004; 8: 343–349
5. Peiper C, Junge K, Klinge U, Strehlau E, Ottinger A, Schumpelick V. Is there a risk of infertility after inguinal mesh repair? Experimental studies in the pig and the rabbit. Hernia 2006; 10(1): 7–12

Discussion

Bendavid: *In one of your drawings you showed a lateral recurrence. An indirect hernia should never be a problem as a recurrence.*
Amid: *I have seen several recurrences lateral of the cord. To avoid this, the mesh should be extended lateral to the internal ring. Except for the one suture, you don't have to put sutures lateral to the internal ring because you can damage the ilioinguinal nerve.*
Miserez: *Does chronic testicular pain arise from an internal ring which is too narrow? How can the ring be calibrated?*
Amid: *It is difficult to prove that the pain arises from a narrow lateral ring. Much more likely, for example, is a lesion of the paravascular nerves.*
Kingsnorth: *The question is still: how do you calibrate the ring?*
Amid: *I use the tip of my finger.*
Read: *With the preservation of the cremasteric muscle I am concerned that the surgeon might miss an indirect hernia.*
Amid: *Around the internal ring we do a longitudinal incision on the cremasteric muscle. Doing this I can identify any indirect hernia sac and dissect it as high as I want.*
Young: *Without opening the cremasteric fascia it might be difficult to dissect the sac correctly in some cases.*
Amid: *The vertical incision of the muscle opens the entire spermatic cord like a book and you can dissect the sac very easily.*
Young: *How do you handle the nerves? You showed a ligation on a nerve which provoked chronic pain. On the other hand, you have to ligate the nerves to prevent a neuroma.*
Amid: *It is a fundamental difference if the nerve is intact or not. If you put a suture to an intact nerve, you will provoke pain. If you cut the nerve, you have a dead nerve and this nerve has to be ligated at the proximal end.*
Schumpelick: *You recommended not to resect the cremasteric muscle in order to keep the spermatic duct away from the mesh. But on the back there is no muscle at all.*
Amid: *You are right. The muscle is like a half-moon shape. But on the back you still have the cremasteric fascia, which will avoid direct contact between duct and mesh.*

25.4 Plug and PHS Technique

D. Pettinari, M. Cavalli, G. Campanelli

Introduction

Inguinal hernia repair is one of the most common operation performed by general surgeons. It is considered a routine procedure, > 150,000 of these repairs are performed annually in Italy, and > 730,000 in the United States [1]. Recurrences of hernia represent failure to achieve the operative goal. It remains a significant clinical problem despite advances in surgical techniques. Reasonably, we can say that the most important yardstick for the success of a hernia repair is still the recurrence rate [2, 3].

Comprehensive audit from national hernia registers in Sweden [4] and Denmark [5] has shown an incidence of recurrence of 16–18% following primary repair, but a recurrence rate of even over 30% has been reported [6]. In a surgical reference centre, with hernia surgery specialization, this rate is above 0.3%, this means about 1100 recurrences a year, despite the introduction of laparoscopy and marked increase in the use of prosthethic materials for the repair of hernia in the wide community [7]. Applying the meshes has not in fact solved the problem of recurrence, but called for different strategies for handling those recurrences [8, 9].

One must examine the factors and influences which come to bear on the choice of technique and quality in our performance.

There are factors beyond the control of the surgeon such as genetics, metabolic disorders, collagen diseases

and smoking, which are now being recognized. Equally, in the hands of surgeons there are factors like the surgical technique, the choice of prosthesis and the necessary knowledge of the inguinal anatomy.

The aim of this work is to clarify some aspects linked to recurrent inguinal hernia, despite the increasing use of prostheses like PHS and hernia plug repair.

Materials and Methods

Mesh is a surgically designed, sterile woven material, made of a synthetic plastic (i.e. polypropylene), specifically used to repair hernias. Prostheses by definition are adjunct foreign materials used in the repair of hernias and traditionally have come usually in flat sheets of various sizes [7].

The plug is a 3-D filler cap that we insert in internal inguinal ring to ensure a correct obliteration near the issue of the spermatic cord (Fig. 25.9)

The Prolene Hernia System 3 in 1 is a unique and innovative design that combines the three most popular tension-free techniques utilized today in the repair of inguinal hernias (Fig. 25.10).

Its onlay patch covers the entire floor of the canal; it overlaps the pubic tubercle for added support, and provides the security of conventional patch techniques.

Its connector provides the simplicity of a plug repair. Additionally, it secures the underlay patch to prevent migration. Its profile is a significant improvement over the bulk of conventional plugs.

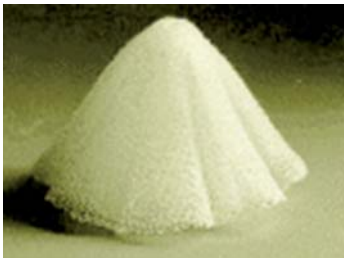


Fig. 25.9. Plug

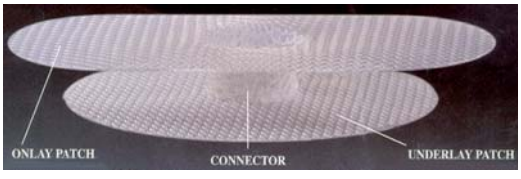


Fig. 25.10. The Prolene Hernia System 3 in 1

The underlay patch, like a laparoscopic repair, provides posterior support; however, it accomplishes that support from a much simpler anterior approach. This underlay patch lies in the preperitoneal space and opens to cover the entire myopectineal orifice. This key and exclusive feature of the Prolene Hernia System ensures that both the femoral and inguinal regions are protected to minimize the possibility of recurrence.

Discussion

Here again, a thorough understanding of the anatomy will lead us to applying our prosthesis in the proper plane. The anatomy of the inguinal hernia region has never been easily mastered by anyone. This delicate aspect of anatomy was taken up and discussed with a clear sense of rediscovery and grateful acknowledgement especially by Cooper, who had enunciated with uncanny accuracy anatomical features which have been since overlooked, neglected or simply forgotten [10].

One further aspect which illustrates anatomical difficulty as an obstacle to good surgery is the presence of a vasculature in the preperitoneal space [11].

The PHS presents an interesting dichotomy; it will work in exactly the same way as a sheet of polypropylene will work if properly placed in the preperitoneal space under direct vision or as practised in the Lichtenstein technique with evidently good result.

The most telling series to indict the poor knowledge of anatomy was the publication of Obney and Chan [12]; these authors reported a series of 1057 repairs on recurrent hernias and noted that 37% of the patients had an indirect inguinal hernia. An accurate knowledge of the anatomy could not yield this level of recurrence. It is evident that sacs are not being identified and often overlooked. A proper dissection would not only discover hernias but would expose the proper planes and tissues to be incorporated in a reliable reconstruction with or without a prosthesis.

Another very difficult point to by-pass is excessive body weight. Obesity is the bane of all surgery. Obese people require larger amounts of sedation, local or general anaesthesia, larger incision and longer operating time; their tissues show marked fatty infiltration, lipomas, and therefore proneness to wound infections. A proper dissection plane to plane is more difficult. Obesity implies excessive tension along any suture line and at peripheral sites where suture or staples maintain a prosthesis in place [3, 8, 13].

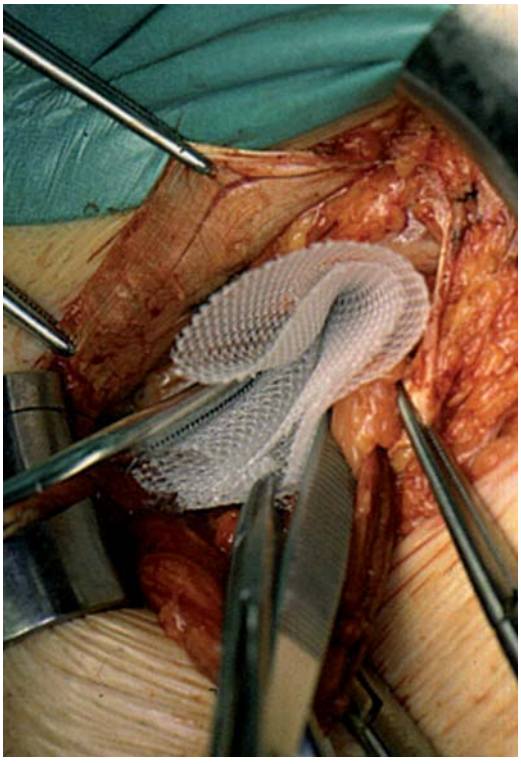


Fig. 25.11. Insertion of PHS

An inadequate tissue dissection may cause a haematoma, and this could be a cause of mesh lifting and recurrence [14]. It will be a problem especially in patients with a difficult dissection of anatomical plane like obese patients, or with a scar tissue caused by previous intervention (e.g. appendicectomy, abdominal way for prostatectomy).

So, we have to evaluate the anatomy of inguinal region, choosing the correct graft plane, we have to take more care of our patient tissues, but we have also to know the surgical technique for positioning properly our prosthesis.

The technical position of PHS will take more care and will need a short training. The PHS is inserted as a plug, into the internal ring (Fig. 25.11).

The underlay patch has to be extended in the preperitoneal space.

The knowledge of this space is the most important because we have already said that in this space (space of Bogros) we have a great vascularization [11].

The connector is, like the plug, to obliterate the internal inguinal ring [15].

The plug repair is one of the most common techniques, and it's used combined in open anterior approach (Trabucco hernia repair) [16].

The plugs secure the larger internal inguinal ring defect. Most people do not use the plug (Guidelines European Hernia Society 2005) but choose to close the internal inguinal ring with one or two stitches using reabsorbable material and so the mesh on the inguinal canal floor is the only "device" that protects from probable recurrence.

In overweight patients, the correct plane dissection and thereby the correct sac isolation just near the internal inguinal ring is very difficult [3, 8, 13]. The closure of the internal inguinal ring is also more difficult with or without the plug.

Conclusion

There are factors beyond the control of the surgeon (genetics and metabolics), but there are factors in the hands of the surgeons.

We have a wide spread use of prostheses but this is not a solution for all recurrences. We have to know the anatomy, the surgical technique and the proper position of all prostheses that we use in our reparations.

References

1. Rutkow IM (1998) Epidemiologic, economic and sociologic aspect of hernia surgery in United States in the 1990's. Surg Clin North Am 78: 941–951
2. Nilsson E, Haapaniemi S, Gruber G (1998) Methods of repair and risk for reoperation in Sweden. Br J Surg 85: 1686–1691
3. Bendavid R. Archives of the Shouldice Hospital. Unpubl. data
4. Nilsson E (1999) Outcomes. In: Kurzer M, Kark AE, Wantz GE (eds) Surgical management of abdomina wall hernias. Martin Dunitz, London, pp 11–19
5. Bay-Nilsson E, Kehlet H (1999) Steering committee of the Danish hernia database. Establishment of a national Danish hernia database: preliminary report. Hernia 3: 81–83
6. Ijzermans JNM, de Wilt H, Hop WCJ (1991) Recurrent inguinal hernia treated by classical hernioplasty. Arch Surg 126: 1097–100
7. Bendavid R (2003) Recurrences: the fault of the surgeon. In: Schumpelick V, Nyhus LM (eds) Meshes: benefits and risks. Springer, Berlin Heidelberg New York, pp 51–62
8. Bendavid R (2002) The Shouldice repair. In: Fitzgibbons R Jr, Greenburg AG (eds) Nyhus and Condon hernia, 5th edn. Lippincott Williams & Wilkins, Philadelphia, pp 129–138
9. Campanelli G, Pettinari D, Nicolosi FM, Cavalli M, Contessini Avesani E (2005) Inguinal hernia recurrence: classification and approach. Hernia 2006; 10(2): 159–161

10. Bendavid R (2001) The transversalis fascia. New observation. In: Bendavid R (eds) Abdominal wall hernia, chap 10. Springer, Berlin Heidelberg New York, pp 97–100
11. Bendavid R (1992) The spaces of Bogros and the deep inguinal venous circulations. Surg Gin Obst 174: 355–358
12. Obney N, Chan CK (1984) Repair of multiple time recurrent inguinal hernia with reference to common causes of recurrence. Contemp Surg 25: 25–32
13. Israelsson LA (2002). Wound failure and incisional hernia. In: Fitzgibbons R Jr, Greenburg AG (eds) Nyhus and Condon hernia, 5th edn. Lippincott Williams & Wilkins, Philadelphia, pp 328–340
14. Lowham AS, Filipi CJ, Fitzgibbons R Jr, Stoppa R, Wantz GE, Felix EL, Crafton WB (1997) Mechanisms of hernia recurrence after preperitoneal mesh repair: traditional and laparoscopic. Ann Surg 225 (4): 422–431
15. Nienahuijs SW, Van Oort I, Keemers-Gels ME, Strobbe LJA, Rosman C. (2005) Randomized clinical trial comparing the prolene hernia system, mesh plug repair and Lichtenstein method for open inguinal hernia repair. Brit J Surg 92: 33–38
16. Prieto-Dias-Chavez E, Medina-Chavez JL, Gonzalez-Ojeda A, Coll-Cardenas R, Uribarren-Berrueta O, Trujllo-Hernandez B, Vasquez C (2005) Tension-free hernioplasty versus conventional hernioplasty for inguinal hernia repair. Surg Today 35: 1047–1053

25

Discussion

Bendavid: *Using the PHS in the case of a large direct hernia you would put two layers of Marlex mesh to the floor which seems by far too much mesh material.*
Campanelli: *You are right, but in cases of large indirect hernias you really need the connector of the PHS device.*
Jeekel: *The principle of the operation is good because a part of the mesh is in the preperitoneal space in a sublay position. But how can you be sure that you prepared the right space, that means the preperitoneal space? And the second question is, what are the results?*
Campanelli: *You cannot see the space, therefore you have to prepare the space very exactly.*
Flament: *We have investigated the transinguinal preperitoneal mesh placement for many years. It is just the Rives procedure. In our experience it is easy to create the preperitoneal space. The sublay mesh is mandatory for the repair and the PHS connector is only for to keep the sublay mesh in place.*
Kehlet: *I think in inguinal hernia surgery we have too many procedures. To decide which method can be preferred we need results. What are the efficacy and the effectiveness data of the PHS procedure?*
Campanelli: *I do not have much experience with the PHS operation. But I think we really need many different procedures to provide a tailored approach in hernia surgery.*
Kingsnorth: *You recommended the PHS for indirect but not for direct hernia. In contrast, Prof. Flament uses his approach especially for direct hernias.*
Campanelli: *In direct hernia you often have a large bulge of the posterior wall and not a defect. In these cases its better to place a mesh on the bulge than to destroy the wall which it's intact.*
Schumpelick: *You did not talk about the Rutkow plug. Is there still a place for this procedure?*
Campanelli: *I don't use the plug. But the operation technique and the indication are the same as for the PHS device.*
Köckerling: *The plug just creates a meshoma in the preperitoneal space. I will show this in the afternoon.*
Kingsnorth: *We don't have to be too dismissive concerning the plug. About two million plugs have been implanted and a lot of surgeons still use this method.*
Deysine: *I have to defend the plug. I have put in about 1500 plugs up to now. Normally, I use the plug in indirect hernia. In direct hernia I prefer the Rives operation. The only six or seven times I saw a problem with the plug it was because of incomplete dissection of the preperitoneal space.*
Young: *We remove more plugs than any other type of mesh in our practise for recurrence or chronic groin pain. We have done about 1500 plug repairs during the past 3 years. Follow-up is difficult because the operation is rather young, but the method seems to be successful also in hands of surgeons who are not experts in hernia surgery.*

25.5 Transabdominal Preperitoneal (TAPP) Inguinal Hernia Repair

B. Kim, Q.Y. Duh

Introduction

Laparoscopy has provided surgeons with new and innovative ways to treat common surgical problems. Over the past 10–15 years, this technology has been applied to the treatment of inguinal hernias, where laparoscopy has introduced a host of alternative surgical techniques. Ger et al. first described the application of laparoscopy to inguinal hernia repair in 1990. In this paper, Ger and colleagues described repair of indirect inguinal hernias through laparoscopic stapling of the abdominal opening of the patent processus vaginalis [1]. Other minimally invasive techniques were later developed including a plug and patch repair [2] and an intraperitoneal onlay mesh (IPOM) repair [3]. The plug and patch repair was not widely adopted due to high recurrence rates coupled with small bowel obstructions related to adhesions [4]. The IPOM repair, as described by Fitzgibbons et al., involved placing a prosthetic mesh over the inguinal hernia defect intraabdominally without performing a groin dissection [5]. While the advantage of this technique was its simplicity, this repair allowed for direct contact of the mesh with viscera and the potential for mesh erosion into bowel. As a result, this method of inguinal hernia repair was largely abandoned.

Today, most laparoscopic inguinal hernia repairs are performed with placement of a prosthetic mesh into the preperitoneal space. This can be accomplished in one of two ways: the totally extraperitoneal (TEP) approach and the transabdominal preperitoneal (TAPP) approach. The TAPP approach was the first to be developed and was initially described by Arregui and colleagues [6]. This repair starts with entry into the abdominal cavity, followed by incision into the preperitoneal space and blunt dissection and reduction of the hernia sac. Once this is done, a piece of prosthetic mesh is placed over the hernia defects with subsequent re-approximation of the peritoneum. The TAPP approach allows for a large working space and a good view of the inguinal anatomy bilaterally. However, this technique requires laparoscopic access into the peritoneal cavity, placing the patient at potential risk of trocar injuries, preperitoneal hernias from the peritoneal incision and port sites, and intra-abdominal adhesive complications.

TAPP vs. TEP

The TEP approach was eventually developed in response to concerns about the need for intra-abdominal laparoscopic access required in the TAPP repair [7]. This method allows for direct access to the preperitoneal space while avoiding the peritoneal incision. However, this procedure is also felt to be technically more demanding given the smaller working space provided compared to the one found in the TAPP repair.

When Felix et al. compared the two methods to each other, they found that the TAPP repair had a higher incidence of intra-abdominal complications than the TEP repair, including port-site hernias, small bowel obstruction, and small bowel injury. However, several TEP repairs needed conversion to a TAPP approach in the study. Additionally, the study showed no appreciable differences with regards to postoperative pain and return to normal activity [8].

In a review of the available literature comparing TAPP vs. TEP repairs, Wake et al. found no statistical difference in length of operation, length of stay, time to return to normal activity, or recurrence rates between the two techniques. They did find higher rates of intra-abdominal injuries and port site hernias in TAPP repairs [9]. In another review, Leibl and colleagues reported similar findings. Of note, however, they stated that the TAPP approach, in general, has a shorter learning curve than the TEP repair [10]. While this conclusion has not been supported by prospective studies, these authors suggested that because of the shorter learning curve, the TAPP repair might be more easily adopted into further surgical education.

Further randomized controlled trials are needed to compare the TAPP to the TEP repair to see which method is superior. Nevertheless, we find the TAPP repair to be useful in some clinical circumstances, for example in patients with large indirect inguinal hernias and in patients with incarcerated inguinal hernias. In addition, the TAPP approach is easier than the TEP approach in patients who have had prior operations in the preperitoneal space.

Operative Steps

The patient is placed supine with both arms tucked and under general anaesthesia. The operation is performed using three trocars: one 10-mm port subumbilically and two 5-mm ports, one in the right lower quadrant and one in the left lower quadrant. Pneumoperitoneum is established, and a 10-mm, 30° angled laparoscope is inserted. The groin anatomy is identified, specifically the inferior epigastric vessels and the internal inguinal ring, through which the spermatic vessels and the vas deferens run (Fig. 25.12). The peritoneum is incised several centimetres above the hernia defect, from the edge of the medial umbilical ligament out laterally towards the anterior superior iliac spine. The peritoneal incision should be made in close proximity to where the upper edge of the mesh used for repair will most likely be positioned. The preperitoneal space is then dissected bluntly in the avascular plane between the peritoneum and the transversalis fascia (Fig. 25.13).

Indirect Hernia

The cord structures are dissected free from the surrounding tissues, as the indirect hernia sac is isolated out. The indirect sac is found on the anterolateral side of the cord and is adherent to it. When separating the sac from the cord, it is important to handle the vas deferens and the spermatic vessels with care so as to minimize trauma. If the sac is small, it can be completely dissected free from the cord, becoming part of the peritoneum (Fig. 25.14). A large sac can be divided a few centimetres distal to the internal ring with the subsequent peritoneal defect closed with an endoloop suture (Fig. 25.15).

Direct Hernia

Direct hernias are typically easier to reduce than indirect hernias. Once the preperitoneal space has been entered, the direct hernia defect is dissected by simply

25

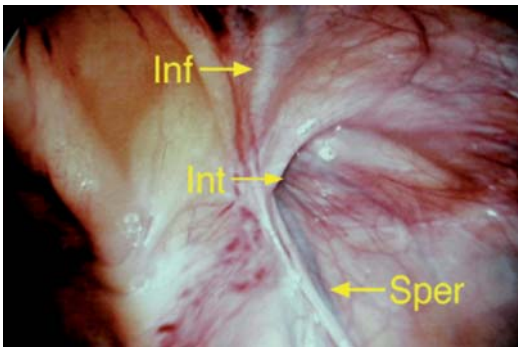


Fig. 25.12. Right groin anatomy view from a 30° laparoscope placed below the umbilicus. *Inf* inferior epigastric vessels; *Int* internal ring; *Sper* spermatic vessels

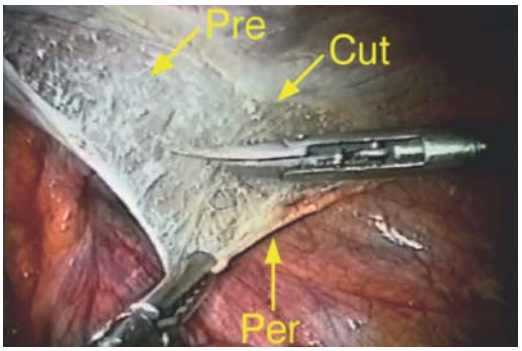
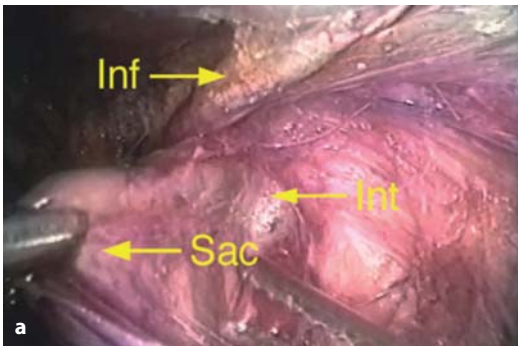


Fig. 25.13. Peritoneal incision exposing the preperitoneal space in the right groin. *Pre* preperitoneal space; *Cut* cut edge of peritoneum; *Per* peritoneum



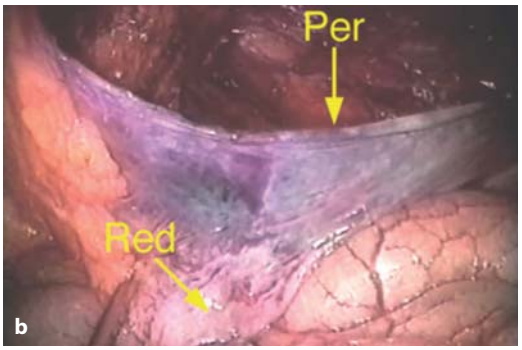


Fig. 25.14a,b. Right indirect inguinal hernia. **a** Dissection of indirect hernia sac from cord structures. *Inf* inferior epigastric vessels; *Int* internal ring; *Sac* indirect hernia sac. **b** Reduced indirect hernia sac. *Per* peritoneum; *Red* reduced hernia sac

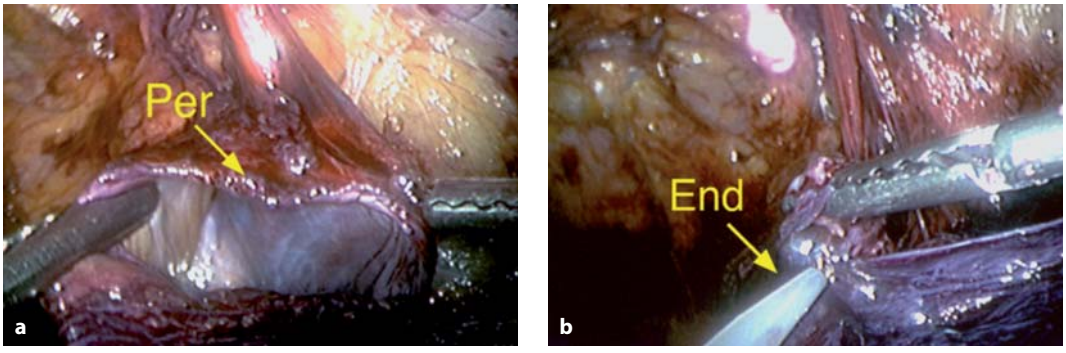


Fig. 25.15a,b. Large right indirect inguinal hernia sac divided just distal to the internal ring. **a** Divided indirect hernia sac with exposed intra-abdominal cavity. *Per* cut edge of sac. **b** Endoloop closure of peritoneal defect. *End* endoloop suture

separating the peritoneum from the overlying myopectineal orifice. When reducing the direct hernia sac, a pseudosac is usually encountered (Fig. 25.16). The pseudosac is part of the transversalis fascia that overlies and adheres to the peritoneum; it invaginates into the preperitoneal space when the surgeon pulls on the true sac during the dissection. The pseudosac must be separated from the true hernia sac in order for the peritoneum to be fully released back into the peritoneal cavity. Once the pseudosac is freed, it will typically retract back into the direct hernia defect.

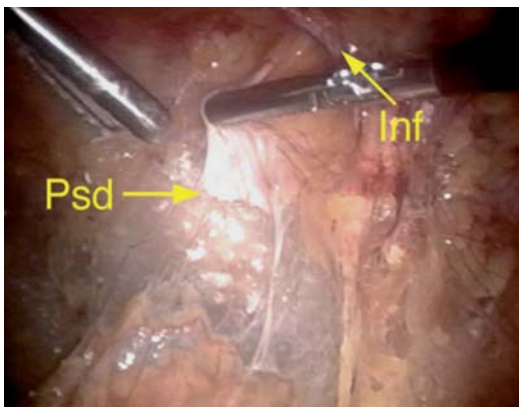


Fig. 25.16. Right direct inguinal hernia. Dissection showing the "pseudosac", which is the retracted transversalis fascia. *Psd* pseudosac, *Inf* inferior epigastric vessels

Placement of Mesh

Prosthetic mesh is required for TAPP repairs. We routinely use a large piece of polypropylene mesh (16×12 cm) to cover all the myopectineal orifices, including the direct, indirect, and femoral hernia spaces. For direct hernias only, we have also used a preformed, contoured mesh (Bard 3D Max Mesh) for coverage. For indirect hernias, we usually use a 16×12 cm mesh that is slit medially. The lower tail is wrapped around the spermatic vessels and the vas deferens in a lateral to medial fashion. The upper edge of the lower tail and the lower end of the upper tail are then fixed onto Cooper's ligament (Fig. 25.17). The slit in the mesh allows it to lie flat in the preperitoneal space while still providing complete coverage of the indirect hernia defect. It is important that the preperitoneal space is completely dissected out (Stoppa described this step as "parietalization") so that the mesh does not fold within this space and compromise the repair. The mesh needs to be fixed medially at Cooper's ligament and laterally above the iliopubic tract to prevent movement of the mesh. The mesh should also slightly overlap the midline to avoid recurrence through the direct hernia space.

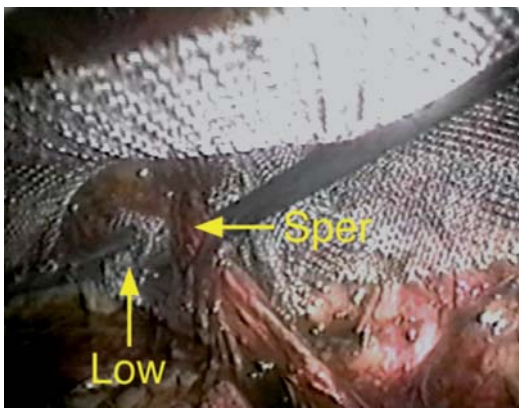


Fig. 25.17. Placement of slit mesh wrapped around cord structures in a lateral to medial direction for a right indirect inguinal hernia. *Sper* spermatic vessels, *Low* lower tail of mesh

Issue of Recurrence

In the hands of experienced surgeons, laparoscopic inguinal hernia repair should result in a recurrence rate of 1% or less [11]. Several reviews of recurrence from TAPP repairs are summarized in Table 25.4.

A number of factors have been noted to lead to higher rates of recurrence. These are:

- Surgeon's inexperience.
- Inadequate dissection of the preperitoneal space.
- Missed hernia defects.
- Insufficient size of the mesh.
- Insufficient overlap of the mesh beyond the edges of the hernia defects.
- Improper fixation of the mesh.

Technical reasons appear to dominate these factors [16] and, as a result, suggest that operative modifications can lead to better outcomes.

Surgeon inexperience has been well accepted as a critical factor in determining the risk of recurrence after a TAPP hernia repair. Bobrzynski et al. specifically addressed this and found that their recurrence rates after TAPP repairs dropped from 2.84 to 1.14% after accounting for their learning curve [17]. Similarly, Voitk suggested that at least 50 cases were required before operating times and complication rates stabilized [18]. Certainly, understanding the preperitoneal groin anatomy and the subtle layers and variations found in this area is important, and this understanding is largely gained through experience. Only by fully understanding this anatomy can complete dissection of the preperitoneal space be ensured. This includes full dissection of the midline and Cooper's ligament, full dissection and reduction of direct and indirect hernia sacs, removal of cord lipomas, and identification of all myopectineal orifices and cord structures. Incomplete dissection can result in missed hernias, leading to persistent rather than recurrent hernias. Complete dissection avoids what Lowham and colleagues refer to as secondary mechanisms of hernia recurrence, which include insufficient size and overlap of the mesh, improper fixation of the mesh, and lipomatous hernia "recurrences" [19].

Factors pertaining to the mesh prosthesis also influence recurrence rates after TAPP repairs. Several studies reported decreased recurrence rates as larger pieces of mesh were used [20–22]. After the preperitoneal space is dissected and all of the myopectineal orifices are identified, the mesh used needs to properly cover all of the existing and potential defects. At least 3 cm of the mesh should overlap of the edge of the hernia defects for complete coverage. This is necessary because the hernia defect tends to enlarge and the mesh tends to shrink over time. Potential hernia defects need to be covered to prevent the future development of hernias in those spaces. We routinely use a 16×12-cm mesh to provide enough overlap medially and laterally.

Mesh fixation prevents recurrence as well. Mesh can be fixed in place with tacks, sutures, or staples. Fixing the mesh avoids early mesh migration and mitigates the effects of mesh shrinkage. Over time, tissue ingrowth will keep the mesh in place. Early on, however, the mesh needs to be anchored. We routinely fix the mesh to Cooper's ligament medially and above the iliopubic tract laterally. This immobilizes the mesh and prevents it from folding. When fixing the mesh laterally, it is important to do so above the iliopubic tract to avoid injury to the lateral femoral cutaneous nerve and subsequent neuralgias.

Using a slit mesh during laparoscopic inguinal hernia repairs has not been universally adopted. Some surgeons believe that if the tails of the slit mesh are not properly reapproximated, the opening will cause a recurrence [19]. Felix, however, noted that a slit mesh is necessary to prevent the cord structures from lifting the mesh off of the inguinal floor [8]. In our experience, a slit mesh is preferred to cover indirect hernia defects. We slit the mesh medially and then fix both tails to Cooper's ligament. For direct hernias, however, slit mesh is not necessary. We prefer to use a preformed, contoured mesh (Bard 3D Max Mesh) with no slit made in it.

Finally, a preperitoneal hernia is a unique problem that may develop after a TAPP repair. In TAPP repairs, a peritoneal incision is required to gain access to the preperitoneal space and to dissect and repair all of the hernia defects. This peritoneal incision needs to be

Table 25.4. Summary of TAPP repair studies

Author	Year	Hernias [n]	Recurrence rate [%]
Bittner et al. [2]	2002	8050	0.70
Kapiris et al. [13]	2001	3530	0.62
Schultz et al. [14]	2001	2500	1.04
Birth et al. [15]	1996	1000	1.10
Felix et al. [8]	1995	733	0.30

closed to prevent herniation of intra-abdominal contents into the dissected preperitoneal space. Closing the peritoneal incision also prevents the bowel from directly contacting the mesh. Because of the need to incise and close the peritoneum, surgeons who prefer the TEP technique frequently cite this as a significant disadvantage of the TAPP repair.

Conclusion

The TAPP approach to inguinal hernia repair has proven to be a safe and effective alternative to conventional open hernia repairs. A recurrence rate of 1% or less should be expected provided the surgeon has gained the appropriate experience. Several factors contribute to better outcomes and a reduced recurrence with the TAPP repair. These include overcoming surgeon inexperience, adequately dissecting out the preperitoneal space and identifying all potential hernia defects, using a large piece of mesh with sufficient overlap beyond the edges of all myopectineal orifices, and fixing the mesh.

References

1. Ger R, Monroe K, Duvivier R, Mishrick A. Management of indirect inguinal hernias by laparoscopic closure of the neck of the sac. Am J Surg 1990; 159(4): 370–373
2. Schultz L, Cartuill J, Graber JN, Hickok DF. Transabdominal Preperitoneal Procedure. Semin Laparosc Surg. 1994; 1(2): 98–105
3. Kingsley D, Vogt DM, Nelson MT, Curet MJ, Pitcher DE. Laparoscopic intraperitoneal onlay inguinal herniorrhaphy. Am J Surg 1998; 176(6): 548–553
4. Tetik C, Arregui ME, Dulucq JL, Fitzgibbons RJ, Franklin ME, McKernan JB, Rosin RD, Schultz LS, Toy FK. Complications and recurrences associated with laparoscopic repair of groin hernias. A multi-institutional retrospective analysis. Surg Endosc 1994; 8(11): 1316–1322; discussion 1322–1323
5. Fitzgibbons RJ Jr, Salerno GM, Filipi CJ, Hunter WJ, Watson P. A laparoscopic intraperitoneal onlay mesh technique for the repair of an indirect inguinal hernia. Ann Surg 1994; 219(2): 144–156
6. Arregui ME, Davis CJ, Yucel O, Nagan RF. Laparoscopic mesh repair of inguinal hernia using a preperitoneal approach: a preliminary report. Surg Laparosc Endosc 1992; 2(1): 53–58
7. Soper NJ, Swanstrom LL, Eubanks WS. Mastery of endoscopic and laparoscopic surgery. Lippincott Williams & Wilkins; Philadelphia, PA, 2005, p 49
8. Felix EL, Michas CA, Gonzalez MH Jr. Laparoscopic hernioplasty. TAPP vs TEP. Surg Endosc 1995; 9(9): 984–989
9. Wake BL, McCormack K, Fraser C, Vale L, Perez J, Grant AM. Transabdominal pre-peritoneal (TAPP) vs totally extraperitoneal (TEP) laparoscopic techniques for inguinal hernia repair. Cochrane Database Syst Rev. 2005; 25: CD004703
10. Leibl BJ, Jager C, Kraft B, Kraft K, Schwarz J, Ulrich M, Bittner R. Laparoscopic hernia repair-TAPP or/and TEP? Langenbecks Arch Surg 2005; 390(2): 77–82
11. Cameron JL et al. Current surgical therapy. Elsevier Mosby; Philadelphia, PA, 2004, pp 1207–1213
12. Bittner R, Schmedt CG, Schwarz J, Kraft K, Leibl BJ. Laparoscopic transperitoneal procedure for routine repair of groin hernia. Br J Surg 2002; 89(8): 1062–1066
13. Kapiris SA, Brough WA, Royston CM, O'Boyle C, Sedman PC. Laparoscopic transabdominal preperitoneal (TAPP) hernia repair. A 7-year two-center experience in 3017patients. Surg Endosc 2001; 15(9): 972–975
14. Schultz C, Baca I, Gotzen V. Laparoscopic inguinal hernia repair. Surg Endosc 2001; 15(6): 582–584
15. Birth M, Friedman RL, Melullis M, Weiser HF. Laparoscopic transabdominal preperitoneal hernioplasty: results of 1000 consecutive cases. J Laparoendosc Surg 1996; 6(5): 293–300
16. Phillips EH, Rosenthal R, Fallas M, Carroll B, Arregui M, Corbitt J, Fitzgibbons R, Seid A, Schultz L, Toy F et al. Reasons for early recurrence following laparoscopic hernioplasty. Surg Endosc 1995; 9(2): 140–144; discussion 144–145
17. Bobrzynski A, Budzynski A, Biesiada Z, Kowalczyk M, Lubikowski J, Sienko J. Experience--the key factor in successful laparoscopic total extraperitoneal and transabdominal preperitoneal hernia repair. Hernia 2001; 5(2): 80–83
18. Voitk AJ. The learning curve in laparoscopic inguinal hernia repair for the community general surgeon. Can J Surg 1998; 41(6): 446–450
19. Lowham AS, Filipi CJ, Fitzgibbons RJ Jr, Stoppa R, Wantz GE, Felix EL, Crafton WB. Mechanisms of hernia recurrence after preperitoneal mesh repair. Traditional and laparoscopic. Ann Surg 1997; 225(4): 422–431
20. Leibl BJ, Schmedt CG, Kraft K, Ulrich M, Bittner R. Recurrence after endoscopic transperitoneal hernia repair (TAPP): causes, reparative techniques, and results of the reoperation. J Am Coll Surg 2000; 190(6): 651–655
21. Felix E, Scott S, Crafton B, Geis P, Duncan T, Sewell R, McKernan B. Causes of recurrence after laparoscopic hernioplasty. A multicenter study. Surg Endosc 1998; 12(3): 226–231
22. Fitzgibbons RJ Jr, Camps J, Cornet DA et al. Laparoscopic inguinal herniorrhaphy-results of a multicenter trial. Ann Surg 1995; 221: 3–13

Discussion

Kukleta: *Up to now, I did not understand the bad results of the Neumayer study. Now I do. Your task was to show how to create a recurrence and you did.*

Duh: *In the Neumayer study 90% of the endoscopic repairs were done with the TEP repair.*

Kingsnorth: *Dr. Kukleta, you tell us that he is doing a bad operation. So tell us why?*

Kukleta: *The strong part of the TAPP is that you visualize everything. You haven't seen anything on this video. The 6-cm opening of the peritoneum is not enough to see down below the important structures. I have not seen the Coopers ligament, and the mesh was too big as well.*

Flament: *In France last year we saw three deaths and severe complications from laparoscopic surgery and none from open surgery. How would you explain this difference?*
Duh: *The severe complications after laparoscopic repair depend a lot on the learning curve problem. Laparoscopic operation is harder to learn and if you make mistakes, some of these mistakes can be deadly.*
Peiper: *Do you have an age limitation for your laparoscopic approach?*
Duh: *In general, the indication depends on the type of hernial defect. I examine the patients and then decide whether a large mesh is necessary or not. Under the age of 20 I would not do a TAPP.*
Halm: *In bilateral hernia I use a large mesh for both hernias with a special shape and I have achieved excellent results. Would you comment on this?*
Duh: *You are right. The problem is where the weakness occurs, and in bilateral hernia that is in the middle.*
Fitzgibbons: *I want to emphasize that Dr. Duh is an excellent hernia surgeon and that this video actually does not show his surgical expertise. Besides, the Neumayer study was TEP in 90% and not TAPP, and the high recurrence rate in the endoscopic group was not because the surgeons were not good. I don't want to leave this impression in the audience here.*
Kurzer: *Dr. Duh, what were your indications? Concerning the contra-indications you mentioned one contra-indication is a future prostatectomy, but that's virtually all the patients you operated upon.*
Duh: *A lot of our hernia patients will be candidates for prostatectomy and there might be a problem in the future. We have to think about it. Concerning indications, we know that recurrent and bilateral hernias are good but I also do a TAPP in single primary hernia. I don't have a recurrence in a TAPP or a TEP repair.*
Kurzer: *So one message from this meeting is that you should have your TAPP or TEP done by an expert, who has gone through his learning curve, and a single primary hernia should be done by an open onlay mesh.*
Duh: *If I were the patient, I would choose my surgeon and not my technique.*
Ferzli: *Concerning future prostatectomy, it is not fair to discuss this only with TAPP or TEP repair. During all the years when open preperitoneal mesh repair according to Rives, Stoppa or Wantz was done, nobody talked about future prostatectomy.*
Duh: *I have been working with urologists at our department and I know they hate us for putting mesh in the preperitoneal space.*

25.6 TEP

P. Chowbey

Introduction

Endoscopic total extraperitoneal repair (TEP) for treatment of inguinal hernia was first described by Dulucq et al, followed by Mc Kernan and Laws in early 1990 and reported by Schultz [1]. The main advantage of the TEP approach is that the entire dissection is done in extraperitoneal space without transgressing into the abdominal cavity. Laparoscopic groin hernia repair totally reinforces the myopectineal orifice of Fruchaud.

Recurrence in TEP

Most important end point of any hernia surgery is the rate of recurrence. Several studies have focused on causes of recurrence after endoscopic hernia repair. Some surgeons have cited early displacement, folding or invagination of mesh during early postoperative period [2]. Lowan et al. have reported factors leading to recurrence including surgeons' inexperience, inadequate dissection, insufficient prosthesis, overlap of hernial defects, improper fixation, folding and twisting of prosthesis, missed hernias and mesh lifting secondary haematoma formation [3].

Recurrence after TEP has been reported to be as low as 0.4% [4]. Phillips et al. have reported recurrence in patients with small mesh size (6×10 cm) [5]. Here we present our experience of more than a decade of TEP repair and lessons learnt regarding causes of recurrence.

The Sir Ganga Ram Hospital (SGRH)

The Department of Minimal Access Surgery at the Sir Ganga Ram Hospital, the first of its kind in the subcontinent, was founded in 1996 to focus exclusively on

evaluation, development and expansion of procedures and techniques in minimal access or key hole surgery. Minimal access surgery (MAS) has introduced a sweeping revolution in surgical practice since its dramatic entry more than a decade ago. We perform more than 2500 basic and advanced laparoscopic surgeries per year, including about 500 endoscopic hernia repairs.

Patient-Related Factors

Improper patient selection in early period of experience may become a leading cause of increased recurrence.

We have proposed a classification system based on expected level of intraoperative difficulty of endoscopic hernia repair. This functional classification grades groin hernias according to the pre-operative predictive level of difficulty of endoscopic surgery.

SGRH Classification for TEP Repair

Grade I

- Small, direct, reducible hernia
  - Swelling appears on coughing/straining and disappears on lying down
  - Finger breadth-size defect in the functional direct floor (Hesselbach triangle)
  - Endoscopically – minimal dissection of sac from fascia transversalis is required

Grade II

- Small, indirect, incomplete, reducible hernia
  - Hernial swelling limited to inguinal canal
  - Endoscopically – the sac can be reduced completely and may not require transection or ligation
- Moderate-sized direct hernia
  - Swelling is present in standing and reduces in the supine position
  - Thumb-sized defect in the direct floor
  - Endoscopically, the sac needs to be dissected from the fascia transversalis
- Reducible femoral hernia

Grade III

- Moderate-sized indirect reducible inguinal hernia
  - Hernial swelling (sac) extends beyond superficial ring, up to the neck of scrotum but does not descend to the testis
  - Endoscopically – this type of hernia will require transection of sac and ligation of its proximal part of sac
- Large reducible direct hernia
  - Involvement of the entire direct floor
  - Big bulge on clinical examination over the triangle of Hesselbach
  - Endoscopically, creation of space in the midline is difficult. Anatomical distortion – stretching and lateral displacement of inferior epigastric vessel
- Recurrent groin hernia
  - Endoscopically – difficult dissection in region of spermatic cord and the space lateral to it.

Grade IV

- Large reducible indirect inguino scrotal hernia
  - Large sac extending up to the testis. The testis cannot be palpated separately from hernia in erect position
  - The sac may contain omentum or small bowel, which require manual reduction in supine position
  - Endoscopically – the internal ring is enlarged with a wide mouthed sac. There is difficulty in dissecting sac from cord structures. Medial displacement and stretching of the inferior epigastric vessels may occur. Inadvertent opening of peritoneum may lead to pneumoperitoneum and dissection of sac becomes difficult
  - There is higher incidence of post-operative seroma/haematoma because of traction on sac
  - The chances of damage to the cord structures are increased

Grade V

- Large, complete, indirect inguinal hernia, which is only partially reducible or irreducible
- Irreducible femoral hernia
  - The sliding component includes bowel or bladder
  - Endoscopically the sac is bulky. Adhesions between contents of the sac and sac wall. The sac often needs to be opened and the contents reduced laparoscopically. Injury to the contents (bowel, bladder and omentum) while reducing them is likely

A Beginner Should Operate

- Patient with small direct hernias (grade I)
- Patient with incomplete indirect sacs (grade 2)
- Patients fit for general anaesthesia
- Patients who can safely withstand longer duration of surgery

A Beginner Should Not Operate

- Patient with obstructed hernia (grade V)
- Patient with complete irreducible hernias (grade V)
- Obese patients
- Patients unfit for general anaesthesia

Equipment-Related Factors

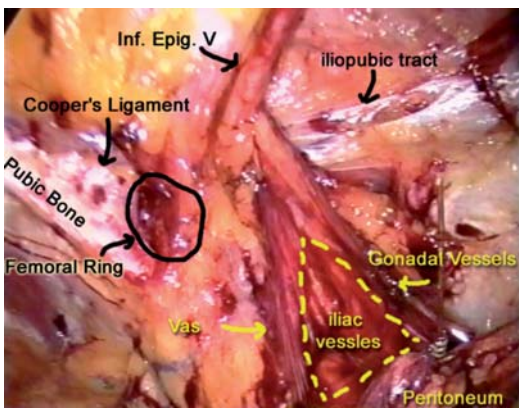
- The core equipment required for any laparoscopic procedure is the endovision system, which consists of the telescope, endovision camera, light source, fibreoptic cable and video monitor.
- Better vision results in better surgery and 10 mm 30° scope is used.
- Poor vision can lead to surgical difficulties and complications.
- An electronic insufflation is necessary for all laparoscopic surgery but for an extraperitoneal hernia repair, high capacity insufflator, i.e. 18–30 l/min, is required because of small working space and loss of space when pneumoperitoneum occurs.

Surgeon-Related Factors

- There is no question that the endoscopic approach is difficult and intense study is required to master this technique [6], but if appropriate skill sets are achieved, the reported result are excellent, reproducible and quite different from those reported by inexperienced surgeons [7].
- Some say that repair is too difficult, takes too much time to perform or too costly. If a repair works, all patients should benefit from this modern approach, including those patients who have only a unilateral hernia.
- Before attempting the endoscopic repair of complex or bilateral hernias a surgeon must learn the anatomy and technique by repairing simple ones. The learning curve can be steep and sometimes prolonged but with proper instruction and supervision operative times are short and equal to open approaches.
- In the hands of experienced surgeons beyond the learning curve, time for laparoscopic repairs are usually equal to or even faster than times reported for open repairs [8]. Hernia surgery should only be attempted after adequate experience of minimum of 50 to 100 basic laparoscopic procedures.

Technique-Related Factors

- Most important endpoint of any hernia surgery is the rate of recurrence. It is the single most factor which judges all repair methods. Several studies have focussed on causes of recurrence after laparoscopic hernia repair.
- Good understanding of endoscopic anatomy of preperitoneal space is important prerequisite for performing endoscopic total extraperitoneal repair for groin hernia. It is a potential space created between fascia transversalis above and peritoneum below (◘ Fig. 25.18).
- The lateral extent is from one anterior superior iliac spine to the other. The region which marks site of femoral and inguinal hernias lies within a quadrangle known as myopectineal orifice of Fruchaud.
- The anatomical space includes the preperitoneal space of Bogros and Retzuis and endoscopic view is horizontal plane from level of umbilicus.
- Creation of preperitoneal space is important step and all precautions should be taken. An infraumbilical, transverse 12-mm incision is made on anterior rectus to avoid inadvertent opening of peritoneum. A space is created and balloon is introduced and



◘ **Fig. 25.18.** TEP apporoach for right inguinal hernia – anatomical landmarks

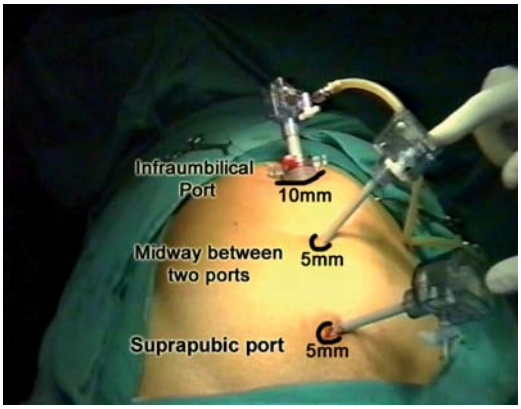


Fig. 25.19. TEP repair – port placements

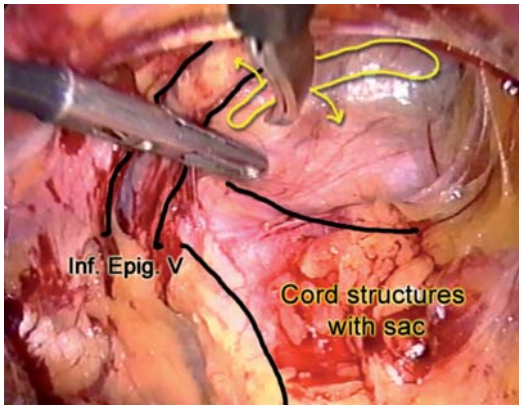


Fig. 25.20. Extraperitoneal landmarks on the right side

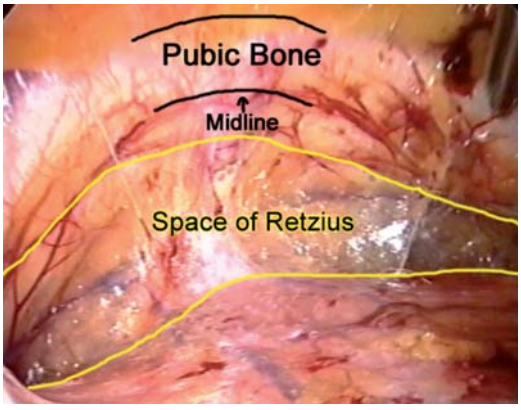


Fig. 25.21. Retropubic dissection

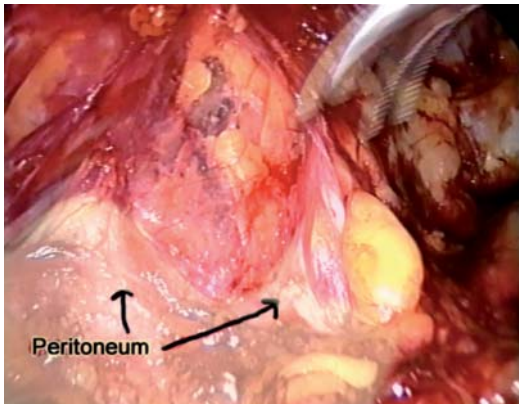


Fig. 25.22. Extraperitoneal dissection

inflated with 100–150 ml saline. The balloon is made by tying two fingerstalls of size 8 latex surgical gloves on 5-mm laparoscopic suction cannula and Hassan's trocar is placed. Accessory ports which are 5 mm should be put in midline under vision to avoid haemorrhage and injury to bladder (Fig. 25.19).

- Injury to peritoneum during trocar insertion can lead to pneumoperitoneum with decrease in working space. The urinary bladder should be kept empty at the time of surgery.
- Sharp dissection is done with use of short burst of cautery, which helps in creating adequate space and ensuring proper haemostasis. This space has loose areolar tissue, and blunt dissection can lead to staining of tissue (Fig. 25.20).
- First structure to identify is the pubic bone and next is the cooper's ligament as it may be get occluded by hernial sac (Fig. 25.21).
- In case of direct inguinal hernia, an indirect sac should be looked for along cord structures and treated. In indirect hernia, sac should be separated from cord structures.
- Dissection should be done closer to deep ring. In case of direct inguinal hernia after reduction of inguinal hernia sac, margins of defect should be free all adhesions otherwise peritoneum can slide along adhesions and cause recurrence (Fig. 25.22).
- The peritoneum should be well reflected proximally from cord structures and complete parietalization should be done (Figs. 25.23, 25.24).
- Indirect sac should be transected in case of complete inguinal hernia so as to avoid pneumoperitoneum. Transected sac may be closed using endloop or free suture tie.
- No dissection should be done in triangle of doom (Fig. 25.25). Lateral dissection till anterior su-

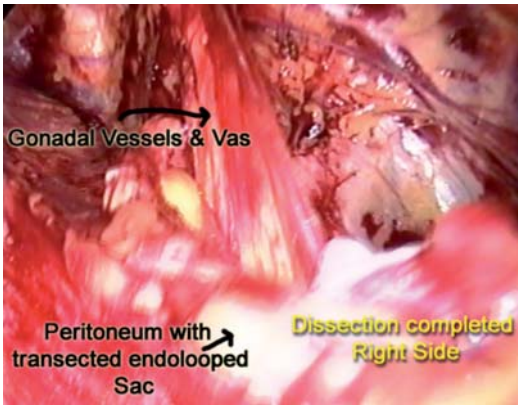


Fig. 25.23. Extraperitoneal dissection on the left side

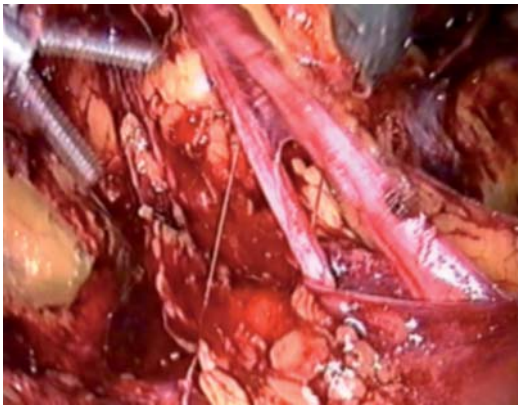


Fig. 25.24. Extraperitoneal dissection on the right side

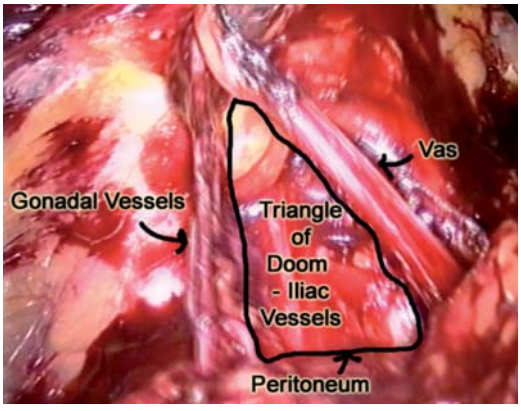


Fig. 25.25. Dissection on left side – triangle of doom

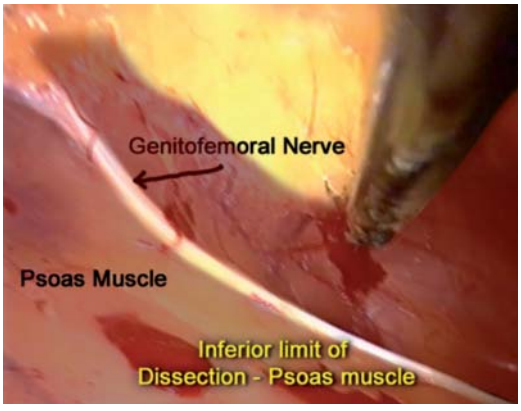


Fig. 25.26. Inferior limit of dissection

perior iliac spine over psoas muscle should be done and injury of nerves should be avoided (Fig. 25.26).

- The minimum size of mesh to avoid recurrence is 15×15 cm.
- To handle a mesh of this size in restricted preperitoneal space is not easy. Thus we have developed a technique of introducing a rolled mesh in this space for easy handling and accurate fixation. The mesh is rolled like a carpet to 2/3 of its length leaving 5 cm free, Stay sutures are tied using absorbable sutures 3 cm away from margins to keep the rolled mesh in position (Fig. 25.27). The rolled mesh is put through 10-mm subumbilical port and the free margin of mesh is pushed into retropubic space medially and psoas muscle laterally.
- A two-point fixation at cooper's ligament should be done to prevent migration. In case of large deep inguinal ring, lateral fixation should be done above iliopubic tract.
- After cutting the stay sutures mesh is unrolled to lie within the preperitoneal space and none of the edges of the mesh should be partially rolled at the time of exsufflation, as this may lead to further rolling and the likelihood of future recurrence of hernia.

Summary

- Dissect the entire myopectineal orifice to ensure extensive parietalization of peritoneum – the more proximal, the better
- Do not slit the mesh. Immobilize the mesh with two point fixation over Cooper's ligament – avoid mesh migration

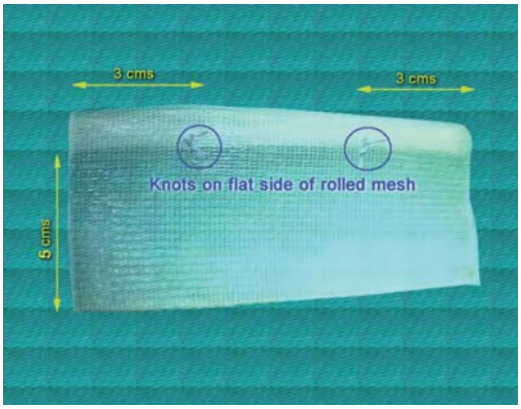


Fig. 25.27. Preparation of mesh

- Use two separate meshes for bilateral hernias – prevents recurrence
- No suture or tacks below iliopubic tract – avoids neuralgia
- Mesh implanted without creases – avoids pressure on nerves
- Laying the mesh on the roof and not the floor – aids in better placement of mesh
- Use large mesh at least 15×12 cm – mesh shrinks postoperatively
- Bulge postoperatively may be seroma or haematoma – wait and watch
- Adequate dissection, complete coverage of myopectineal orifice and proper fixation – must for endoscopic inguinal hernia repair

References

1. Schultz L, Graber J, Pietrapitta J et al. Laser laparoscopic herniorraphy: A clinical trial: Preliminary results. J Laparosc Surg 1990; 1(1): 41–45
2. Leibl BJ, Schmedt C, Kraft K, Ulrich M, Bittner R. Recurrence after endoscopic transperitoneal hernia repair (TAPP): causes, reparative techniques and results of reoperations. J Am Coll Surg 2000; 190: 651–655
3. Lowan AS, Filipi CJ, Fittzgibbons JR, Stoppa R, Wantz GE, Felix EL, Crafton WB. Mechanisms of hernia recurrence after preperitoneal mesh repair, traditional and laparoscopic. Ann Surg 1997; 225: 422–431
4. Felix E. 10 year experience with laparoscopic hernioplasty. Presented 9th World Congress of Endoscopy, Cancun Mexico, February 2003
5. Phillips EH, Rosenthal R, Fallas M, et al. Reasons for early recurrence following lap. Hernioplasty Surg Endsc 1995; 9(2): 140–144
6. Wright D, O' Dwyer P. The learning curve for laparoscopic hernia repair. Semi Laparosc Surg 1998; 54: 227–232
7. Felix E, Scott S, Crafton B, Geis P, Duncan T, Swell R, Mckernan B. Causes of recurrence after laparoscopic hernioplasty. Surg Endosc 1998; 12: 226–231
8. Schurab J, Beaird D, Ramshaw B, et al. After 10 years and 1903 inguinal hernias, what is outcome for laparoscopic repair? Surg Endosc 2002; 168; 1201–1206

Discussion

Ferzli: *A short comment: we avoid opening or tying the sack. We just prepare it and reduce it.*
Chowbey: *If you have a large sac to prepare, the patients suffer from cord indurations postoperatively but if you transect the sac and leave the distal part in place the patients don't have this problem.*
Miserez: *Two remarks: very delicate manipulation of the vas is important, and secondly the lateral lowest part of the mesh should be exactly fixed when you desufflate.*
Chowbey: *We fix the lateral lower part of the mesh with a forceps during desufflation to avoid the peritoneal sac going under the mesh.*
Köckerling: *You showed how you dissect the medial extension of the transversalis fascia. Our experience is that it's good to reduce this extension. We grasp the transversalis fascia and suture it to the Cooper's ligament to avoid a sudden recurrence.*
Chowbey: *There are two ways to handle a big direct sac. One is to pull it down and fix it to the Cooper's ligament. Another is to fenestrate the transversalis fascia to avoid a big seroma formation.*
Kingsnorth: *Prof. Schumpelick asked the chairmen to give some recommendations at the end of a session. I would like to give recommendations not to the experts but to the surgical community. I would say the Lichtenstein repair could be the first operation of choice for a general surgeon with a patient with a straightforward primary hernia. In those places where they prefer a tissue repair, they should teach an adequate technique of the Shouldice operation. But there are countries where a tissue repair is obsolete. The PHS and the plug repair are still discussed controversially. More results are necessary. Those surgeons who are beyond their learning curve can apply the TAPP or TEP repair very well, but extrapolation of these techniques to other surgeons must be done carefully and with caution.*

25.7 GPRVS

P. Verhaeghe, F. Dumont, R. Stoppa

Introduction

The large interposition [1] of a synthetic non-absorbable mesh is able to hold face with the neighbouring layers and to support instantly and permanently the inguinal wall between the deeper inguinal layer and the visceral sac (◘ Fig. 25.28) in the retroperitoneal cleavable space [2]. This prosthesis, extended broadly beyond the weak inguinal area in all directions, completely covering the Fruchaud's musculo pectineal hole [3] is pressed by intra-abdominal pressure against the inner face of the abdominal wall and quickly attached by the development of connective tissue through the mesh. This definition includes all the technical aspects that can explain a recurrence after a GPRVS.

25

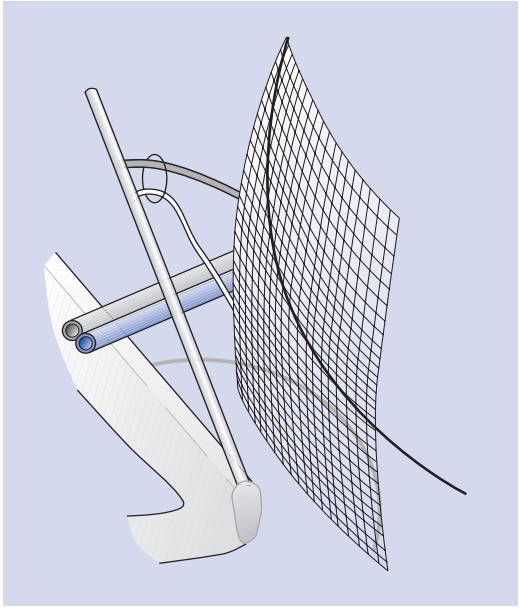
Operative Circumstances: Foreseeable

Diffuse medial scarrring represents a rare but difficult challenge following transvesical prostatectomy or traumatic lesion of the pelvic rim. Trying to dissect pro vesical space leads to a high risk of a vesical wound so at the beginning of our experience we cut the medial part of the prosthesis vertically and we observed a lateral sliding of the sheets and internal inguinal recurrence occurred (◘ Fig. 25.29).

A previous aorto-femoral prosthesis, needing an inguinal incision and often a femoral inguinal section, is often associated with large scarring: it is a contraindication for GPRVS use.

Complicated appendicectomy will produce a diffuse scarring in the right iliac fossa. In this case the peritoneal dissection must be performed upper from the Douglas arch and by a circular movement and a limited opening of peritoneal bag, which will be closed in a second time.

Female inguinal hernia is more difficult, because the teres uteri ligament adheres strongly to the peritoneal bag and cannot be parietalized correctly. Cutting the teres uteri ligament liberates the region and allows a large posterior peritoneal bag dissection.



◘ **Fig. 25.28.** Mesh between the deeper inguinal layer and the visceral sac

Medial Preperitoneal Approach

Limited parietalization of the spermatic cord is probably the most frequent pitfall.

Two mechanisms explain this occurrence:

- If the dissection is not begun medially in contact with the anterior part of the peritoneal bag, the novice surgeon, laterally, is embarrassed by constant limited bleeding because he is progressing too superficially and wounding small epigastric vessels branches. The limited vision explains why he stops too early.
- Lack of knowledge of embryology explains why some surgeons open the prosthesis (◘ Fig. 25.30) to let the cord go through. The uro-genital fascia [4] is a protection for deferent canal and spermatic vessels; this fascia will also protect the iliac vessels from prosthesis contact. A precise dissection of the limit between peritoneal hernia sac and uro-genital fascia is bloodless, can lead to a limited opening of the peritoneum (sutured) but the posterior dissection will be easy and must be done as far as the psoas muscle.

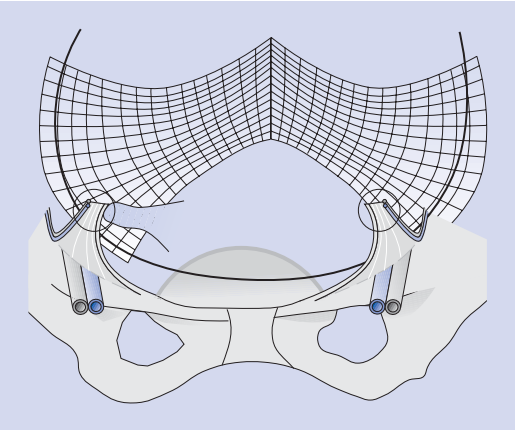


Fig. 25.29. Medial part of the prosthesis is cut because of postoperative previous fibrosis

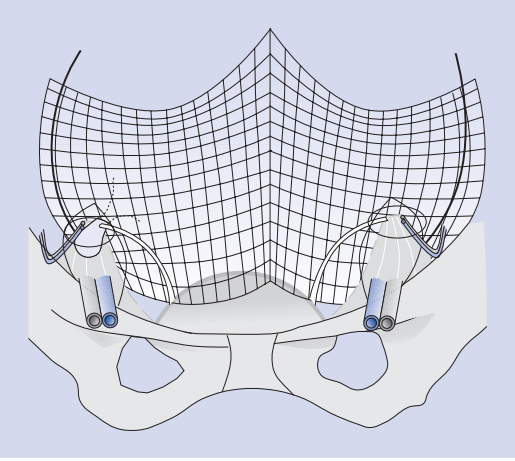


Fig. 25.30. Vertical sections in front of the internal inguinal ring create a weak point

Prosthesis Preparation

A large prosthesis is an essential element of the success; this prosthesis largely covers the peritoneal bag.

- Some surgeons use folded prosthesis (two or three layers!). So the prosthesis cut large (25 or 28 cm), finally covers no more than 14 or 9 cm, which is certainly too small for bilateral hernia [6].
- A small prosthesis, shrunken by fibrosis, will not cover the two inguinal holes
- A horizontal inferior section of the prosthesis is not favourable to a good wrapping: the three-dimensional curvature of the pelvis needs a chevron section of the inferior line of the prosthesis, so the inferior triangles will develop inferiorly and medially.
- Insertion of two separate prostheses on each side seems to cover the same surface, but the Pascal principle is not respected, so these prostheses can move laterally more easily.

Prosthesis Handling

The use of too small forceps (less 30 cm long) does not allow the surgeon to keep the prosthesis far enough back and does not facilitate a large wrapping.

Conclusions

The GPRVS is a reproductible technique providing a recurrence rate of less than 1% in skilled hands; most recurrence can be avoided by good anatomical knowledge.

References

1. Stoppa R, Petit J, Abourachid H (1973) Procédé original de plastie des hernies de l'aine l'interposition sans fixation d'une prothèse en tulle de dacron par voie médiane sous-péritonéale. Chirurgie 99: 119–123
2. Odimba BFK, Stoppa R, Laude M (1980) Les espaces clivables sous pariétaux de l'abdomen. J Chir (Paris) 117: 621–627
3. Fruchaud H (1956) Anatomie chirurgicale des hernies de l'aine. Doin edit, Paris (The surgical anatomy of hernias of the groin. Translated by R. Bendavid and P. Cunningham. Pandemonium books. Toronto, 2006)
4. Stoppa R, Diarra B, Mertl P (1997) The retroparietal spermatic sheath. An anatomical structure of surgical interest Hernia 1: 55–59
5. Verhaeghe P, Rohr S (2001) Chirurgie des hernies inguinales de l'adulte. Arnette edit, Paris
6. Stoppa R (2003) Wrapping the visceral sac into a bilateral mesh prosthesis in groin hernia repair. Hernia 7: 2–12

Discussion

Kingsnorth: *There is a large publication of 1000 prostatectomies from the USA looking at a subgroup of patients who previously had laparoscopic mesh repair. There was no increase in complications in this subgroup. So carry on with mesh placement in the preperitoneal space, there will be no dispute with the urological surgeons.*

Verhaeghe: *Prof. Stoppa never told us to use GPRVS in all inguinal hernias. The indications are recurrences and bilateral hernia, which accounts for about 10% of all hernia.*

Kingsnorth: *Are you still using Mersilene?*
Verhaeghe: *Yes, and as with polypropylene, we also never put Mersilene in contact with the bowel. If you do so, it will migrate into the bowel. It might take 4 or 5 years, but it will happen. Recently, I re-operated a patient of Prof. Stoppa. He had done a mesh repair of an umbilical hernia 15 years before and in histology there was a penetration of the mesh into the layers of the bowel. It was only a question of time until a complete fistula would occur.*
Bendavid: *In large hernias there might be a bulge in the region of the defect. Do you have an idea how to avoid this phenomenon?*
Verhaeghe: *This is a real problem in very large inguinal hernia. The bulge is a result of seroma formation. You have to explain this to your patients. You might reduce seroma formation by leaving the peritoneal sac in the defect and just cut the peritoneum in these large hernias rather than preparing it completely.*
Bendavid: *Could it happen that the mesh itself bulges into the defect in very large hernias?*
Verhaeghe: *This event is very rare. If it happens, you can repair it by a simple transinguinal suturing of the caudal margin of the mesh to the Cooper ligament.*

25.8 Anaesthesia and Recurrence in Groin Hernia Repair

P. Nordin, S. Haapaniemi

25

Introduction

Groin hernia surgery is one of the most frequent operations performed in general surgery. The introduction of mesh techniques has led to a marked reduction in recurrence rates and attention has now shifted to other aspects of hernia surgery. For the important question as to method of anaesthesia, there is still no consensus about the best choice. In general surgical practice, regional and general anaesthesia are the preferred choice [1–3], whereas local anaesthesia is almost exclusively used in centres with a special interest in hernia surgery [4–8]. Most reviews and case series [9–12] as well as randomized trials [13–17], bear witness to its advantages over regional and general anaesthesia. Its reported major advantages are simplicity, safety for high-risk patients, extended postoperative analgesia, early mobilization without post anaesthesia side effects and low cost [4–7].

The long-term outcome of hernia repair is generally assumed not to be affected by the method of anaesthesia used [18]. However, the evidence on which this assumption is based is far from convincing. The few studies on the topic have produced conflicting results [19–21].

The Swedish Hernia Register (SHR) records detailed information on the great majority of groin hernia repairs performed in Swedish hospitals [22, 23]. During 1992–2004, 107,838 hernia repairs were prospectively recorded. With such large numbers of data it is possible to study time trends for anaesthetic methods and repair techniques. Furthermore, multivariate analysis of risk for re-operation associated with alternatives in anaesthesia may also be undertaken with appropriate adjustment for possible confounding factors. In the present investigation SHR data was used to estimate the relative risk of re-operation for recurrence with the three anaesthetic alternatives.

Patients and Methods

Data were retrieved from the SHR and comprise all patients over 15 years of age who underwent groin hernia repair between 1 January 1992 and 31 December 2004. In 2004 approximately 95% of all hernia operations done in Sweden were documented in the SHR. In this register each operation for groin hernia on patients 15 years of age and older is recorded according to a protocol where variables such as patient characteristics, type of hernia, method of repair, suture material, anaesthesia, complications and re-operation for recurrence (if applicable) are noted. Clinical follow-up is not mandatory, but any complication that is observed by the operating unit has to be recorded in the register. External review of register data is performed on an annual basis [23]. Patients are identified through a person number [24] unique for each citizen in Sweden, thereby making it possible to link re-operations to previous operations performed within the framework of the register.

Table 25.5. Swedish Hernia Register 1992–2004

Year	Participating units	Hernia repairs	Recurrent hernia [%]
1992	8	1690	16.4
1993	8	1647	15.9
1994	9	2287	16.5
1995	19	3331	17.1
1996	21	4056	15.8
1997	29	5923	14.4
1998	37	8263	15.2
1999	45	9307	13.1
2000	53	10602	12.3
2001	73	13143	11.4
2002	79	14714	11.0
2003	86	16092	10.5
2004	90	16783	10.1
Total	–	107838	

Statistics

Statistical analyses were performed using SPSS version 12.0.1 (Chicago, IL). Differences between groups were tested by chi-square analysis and cumulative incidence of re-operation was measured through actuarial analyses. Relative risks of re-operation were estimated with the Cox proportional hazards regression model [25], first performing univariate analyses for assumed risk variables and then selecting variables with the highest or lowest univariate risks for multivariate analysis. Re-operation, for the Cox analysis, was defined as a hernia repair in the same groin as the previous repair, performed within the framework of the SHR.

Results

Hernia repairs, participating units and percentage of operations done for recurrent hernia in SHR are given in Table 25.5. During the 13-year period 107,838 hernia repairs have been performed; 60,925 (57%) repairs were performed under general anaesthesia, 30,398 (28%) in regional anaesthesia and 16,515 (15%) in local anaesthesia. Since the start of the registration, 12,982 operations for recurrent hernia have been carried out. This amounts to 12% of all operations registered. The percentage of operations done for recurrent hernias has decreased from above 16% 1992–95 to 10% 2004. As can be seen in Fig. 25.31, great changes have taken place concerning the choice of anaesthesia. In the early

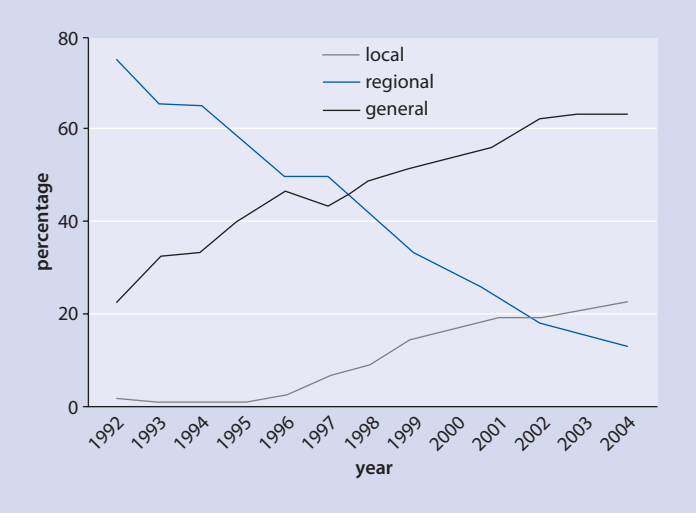


Fig. 25.31. Choice of anaesthesia in the SHR between 1992 and 2004 (107,838 hernia repairs)

1990s the use of local anaesthesia was limited to a few cases, but by the middle of that decade its use had started to increase and reached 23% in 2004. During the same 13-year period the use of regional anaesthesia decreased from 75 to 14%.

The percentage of patients treated on an outpatient basis increased from 33% in 1992 to 74% in 2004 (Fig. 25.32) with wide variations among participating units. Of all repairs performed under local anaesthesia, 82% were carried out on an outpatient basis. The corresponding percentages for regional and general anaesthesia were 48 and 61%, respectively. The differences between the three groups are statistically significant ($p < 0.001$).

Univariate analysis (without any adjustments) of the cumulative incidence of re-operation for recurrence after 5 years following all 107,838 hernia repairs was 4.0%, and for the three anaesthetic alternatives local, regional and general anaesthesia it was 3.7, 4.0, and 4.0%, respectively (Fig. 25.33).

The data of all 107,838 operations were also used to analyze the relative risk for re-operation after surgery with the three forms of anaesthesia. As indicated in Table 25.6, no significantly difference in risk for re-operation was found between local, regional or general anaesthesia in univariate analyses. However, multivariate analyses demonstrated a significant increased relative risk for re-operation with local anaesthesia in pri-

Fig. 25.32. Day-case surgery in the SHR, 1992 to 2004 (107,838 hernia repairs)

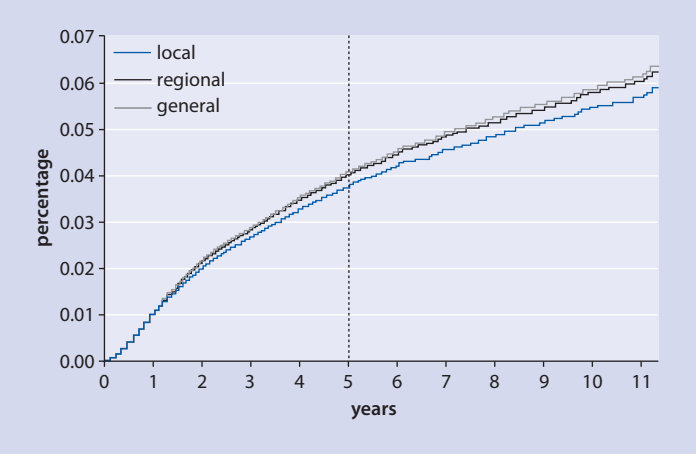


Fig. 25.33. Univariate analysis of the cumulative incidence of reoperation with 107,838 primary and recurrent hernia operations

Table. 25.6. Choice of anaesthesia and relative risk (RR) for re-operation following 107,838 primary and recurrent hernia operations registered in the SHR 1992–2004

Variable	Operations [n]	RR	95% CI
All hernias[a]	107,838	–	Reference
Local anaesthesia	16,515	1.00	–
Regional anaesthesia	30,398	1.07	0.94–1.20
General anaesthesia	60,925	1.08	0.97–1.21
All hernias[b]	107,838	–	–
Local anaesthesia	16,515	1.00	Reference
Regional anaesthesia	30,398	0.76	0.67–0.87
General anaesthesia	60,925	0.86	0.76–0.97
Primary hernias[b]	94,856	–	–
Local anaesthesia	15,365	1.00	Reference
Regional anaesthesia	26,530	0.74	0.63–0.85
General anaesthesia	52,961	0.83	0.71–0.95
Recurrent hernias[b]	12,982	–	–
Local anaesthesia	1,150	1.00	Reference
Regional anaesthesia	3,868	0.77	0.57–1.02
General anaesthesia	7,964	0.88	0.67–1.15

[a]Univariate analysis, [b]multivariate analysis according to Cox's proportional hazards model

mary but not recurrent hernia. Using local anaesthesia as a reference in primary hernia repairs, the RR was 0.74 (95% CI 0.63–0.85) and 0.83 (95% CI 0.71–0.95) for regional and general anaesthesia, respectively. For recurrent hernia repairs the RR was 0.77 (95% CI 0.57–1.02) for regional anaesthesia and 0.88 (95% CI 0.67–1.15) for general anaesthesia, i.e. there were no significant differences between the methods of anaesthesia.

Discussion

The main findings in the present study are that the use of local anaesthesia was associated with a significantly increased risk of re-operation for recurrence in primary hernia repair. No significantly increased re-operation rate was found when local anaesthesia was used in operations for recurrent hernia.

The study's strong points are the large number of collected data and the data quality obtained through numerous controls and annual external review [23]. The results are based upon detailed information from 107 838 prospectively documented hernia repairs. With such a large number of observations, it is possible to perform multivariate analyses of risk for re-operation using appropriate adjustments for possible confounding factors. Another strong point is that surgeons with varying background and experience of hernia surgery perform the operations. Hence, the outcome reflects the results obtained in routine practice, effectiveness, as distinct from results obtained by experts under optimal conditions in selected patients, and efficacy. Register studies with multivariate analysis cannot replace randomized trials (RCT). Some aspects of hernia surgery cannot, however, easily be studied in RCT. An example is late recurrence. Therefore, epidemiological studies based on validated quality registers can have an important complementary role.

The main limitation of the study, which is a problem inherent in all large-scale register studies of this type, is the use of re-operation as the endpoint which does not reflect total number of recurrences. However, the recurrence rate can be estimated by multiplying re-operation rates by 1.4–2.3, depending upon definition of recurrence and method of follow-up used [26, 27].

This study is not the first to find that local anaesthesia has been associated with a somewhat higher risk of recurrence. In a recent study on the effect of smoking, Sorensen and coworkers [21] accidentally found local anaesthesia to carry a higher risk than general and regional anaesthesia lumped together. In contrast, no such association was found in the Danish national hernia database [1].

In spite of the fact that univariate analysis showed a somewhat lower risk for re-operation in the local anaesthesia group, the multivariate analysis showed that local anaesthesia was associated with a significantly increased risk for re-operation in primary but not in recurrent hernia repair. Sorensen et al. also found similar differences between the univariate and the multivariate analyses [21]. One conceivable explanation for this discrepancy may be the fact that local anaesthesia is selectively used in a higher frequency with techniques associated with a decreased relative risk for re-operation.

Local anaesthesia has gradually become more and more popular among Swedish surgeons during the past decade. In 1992 no more than 1.7% of all operations were performed under local anaesthesia, but that number had risen to 23% in 2004. The technique is quite easy to learn, but only successful if the surgeon handles the tissues gently and has patience. Since skill and experience seem to be of such great importance, substandard results are likely to occur if surgeons use the technique without appropriate training. Already when he introduced local anaesthesia for hernia surgery, Cushing [28] pointed out that the technique is only successful if the surgeon is fully conversant with all its details.

Later, Kingsnorth et al. [19] even found that the surgeon's personal experience was the factor that most strongly influenced recurrence. Since skill and experience seem to be of such great importance in this demanding technique, substandard results are likely to occur during a period when many of the surgeons using the technique are still in the learning stage. The surgeon's skill should be kept in mind in the evaluation of all operative data, even data from RCT [29].

To sum up, local anaesthesia is slowly becoming more popular in Swedish hernia surgery. Its use was found to be associated with a somewhat higher re-operation rate in primary hernia repair. This leads to stressing the importance of proper training before adopting the technique, and that further investigations on the effect of anaesthesia on recurrence after hernia repair are motivated.

Acknowledgements. The authors thank the surgeons at the aligned hospitals for their collaboration. Register secretary Annika Enarsson and statistician Lennart Gustafsson have provided invaluable work for the SHR. Financial support for the SHR has been received from the National Board of Health and Welfare and the Federation of County Councils, Sweden.

References

1. Bay-Nielsen M, Kehlet H, Strand L, et al. Quality assessment of 26,304 herniorrhaphies in Denmark: a prospective nationwide study. Lancet 2001; 358: 1124–1128
2. Hair A, Duffy K, McLean J, et al. Groin hernia repair in Scotland. Br J Surg 2000; 87: 1722–1726
3. O'Riordan DC, Kingsnorth AN. Audit of patient outcomes after herniorrhaphy. Surg Clin North Am 1998; 78: 1129–1139
4. Amid PK, Shulman AG, Lichtenstein IL. Open tension-free repair of inguinal hernias: the Lichenstein technique. Eur J Surg 1996; 162: 447–453
5. Bendavid R. Symposium on the management of inguinal hernias. 4. The Shouldice technique: a canon in hernia repair. Can J Surg 1997; 40: 199–207
6. Callesen T, Bech K, Kehlet H. One thousand consecutive inguinal hernia repairs under unmonitored local anaesthesia. Anesth Analg 2001; 93: 1373–1376
7. Kark AE, Kurzer MN, Belsham PA. Three thousand one hundred seventy-five primary inguinal hernia repairs: advantage of ambulatory open mesh repair in local anaesthesia. Am Coll Surg 1998; 186: 447–455
8. Kingsnorth AN, Porter C, Bennett DH. The benefit of a hernia service in a public hospital. Hernia 2000; 4: 1–5
9. Cheek C, Black N, Devlin HB, et al. Groin hernia surgery: a systematic review. Annals of the Royal College of Surgeons of England. 1998; 80 (Suppl 1): S1–S80
10. Flanagan LJR, Bascom JV. Repair of groin hernia: out-patient approach with local anaesthesia. Surg Clin N Am 1984; 64: 257–268
11. Young DV. Comparison of local, spinal, and general anaesthesia for inguinal herniorrhaphy. Am J Surg 1987; 153: 560–563
12. Makuria T, Alexander Williams J, Keighley MR. Comparison between general and local anaesthesia for repair of groin hernias. Ann R Coll Surg Engl 1979; 61: 291–294
13. Godfrey PJ, Greenan J, Ranasinghe DD, et al. Ventilatory capacity after three methods of anaesthesia for inguinal hernia repair: a randomized controlled trial. Br J Surg 1981; 68: 587–589
14. Knapp RW, Mullen JT. Clinical evaluation of the use of local anaesthesia for the repair of inguinal hernia. American Surgeon 1976; 42: 908–910
15. Song D, Greilich NB, White PF, et al. Recovery profiles and costs of anaesthesia for outpatient unilateral inguinal herniorrhaphy. Anesth Analg 2000; 91: 876–881
16. Teasdale C, McCrum AM, Williams NB, Horton RE. A randomised controlled trial to compare local with general anaesthesia for short-stay inguinal hernia repair. Ann R Coll Surg Engl 1982; 64: 238–242
17. Nordin P, Zetterström H, Gunnarsson U, Nilsson E. Local, regional,or general anaesthesia in groin hernia repair: multicentre randomised trial. Lancet 2003; 362: 853–858
18. Devlin HB, Kingsnorth AN. Management of abdominal hernias. 2nd edn. Chapman & Hall; London, 1988
19. Kingsnorth AN, Britton BJ, Morris BJ. Recurrent inguinal hernia after local anaesthetic repair. Br J Surg 1981; 68: 273–275
20. Morris GE, Jarrett PE. Recurrence rates following local anaesthetic day case inguinal hernia repair by junior surgeons in a district general hospital. Ann R Coll Surg Engl 1987; 69: 97–99

21. Sorensen LT, Friis E, Jorgensen T, et al. Smoking is a risk factor for recurrence of groin hernia. World J Surg 2002; 26: 397–400
22. Haapaniemi S. Quality assessment in groin hernia surgery – the role of a register. Linköping University, Sweden, Medical Dissertation No. 695, 2001
23. Nilsson E, Haapaniemi S. Assessing the quality of hernia repair. In: Fitzgibbons R Jr, Greenburg AG (eds) Hernia. Nyhus and Condon, Lippincott, Philadelphia, 2000, pp 567–573
24. Lunde MN, Lundeborg S, Lettenstrom GS, et al. The person-number systems of Sweden, Norway, Denmark, and Israel. Vital Health Stat 1980; 2: 1–59
25. Cox D. Regression models and life tables. J R Stat Soc 1972; 208: 187–220
26. Kald A, Nilsson E, Anderberg B, Bragmark M, Engström P, Gunnarsson U, et al. Reoperation as surrogate endpoint in hernia surgery: A three year follow-up of 1565 herniorrhaphies. Eur J Surg 1998; 164: 45–50
27. Haapaniemi S, Nilsson E. Recurrence and pain three years after groin hernia repair. Validation of postal questionnaire and selective physical examination as follow-up method. Eur J Surg 2002; 168. 22–28
28. Cushing H. The employment of local anaesthetics in the radical cure of certain cases of hernia with a note on the nervous anatomy of the inguinal region. Ann Surg 1900; 31: 1
29. van der Linden W. Randomized surgical trials. In: Delaney JP, Varco RL (eds) Controversies in Surgery II. Saunders, Philadelphia, 1983, pp 1–5

Discussion

Campanelli: *We also do our operations under local anaesthesia. Do you have an explanation why only 20% of the hernia repairs in Sweden are done under local anaesthesia?*

Nordin: *It is difficult to introduce local anaesthesia because in most clinics in Sweden the service of the anaesthesiologists is very good and surgeons like to have a sleeping patient. It takes time to introduce a new technique.*
Simons: *What is the reason that you have more recurrences if you have done the repair under local anaesthesia?*
Nordin: *My theory is that the repair can be more difficult when you are not completely convinced with the technique of the local anaesthesia. When the patient has pain during the surgery, both the patient and the nurse will push you to complete the surgery more rapidly and you might make surgical mistakes.*
Simons: *I agree with the stress idea. Especially in training it is difficult to use local anaesthesia. I think this is one of the reasons why in Holland the local anaesthesia is stable at about 2% and it does not go up.*
Verhaeghe: *Have you found a difference with local anaesthesia for primary or recurrent hernia?*
Nordin: *In recurrent surgery, one has to be more familiar with the technique of local anaesthesia. Normally, a recurrence is done only by experts in local anaesthesia.*
Young: *Only under local anaesthesia can you do an intraoperative test of the hernia site and the repair. We always have an anaesthesiologist who gives a sedation during our local procedures and I think there are certain parts of the repair where you need more sedation.*
Nordin: *I agree with you.*
Bendavid: *A common mistake is that the sedation is not given long enough beforehand. We never have any problems when we start the sedation one and a half hours beforehand.*

26 How to Treat Recurrent Inguinal Hernia

26.1 Open Suture

U. Muschaweck

Introduction

The wrong decision on the repair technique for a primary hernia or technical failure is the beginning of the natural history of a recurrence. Despite the development of implants and refinement of surgical techniques over the years, recurrences in inguinal hernia surgery still remain. Our aim was to evaluate the techniques used to treat recurrent hernia after suture repair.

Table 26.1. Surgical techniques for primary hernias 2003 and 2004

	2004	2003
Suture repair	31.5%	48.0%
Mesh repair	30.0%	27.1%
Laparoscopy	38.5%	24.9%

Database: BAQ Bayerische Qualitätssicherung 2004, Modul 12/3 without data of Hernia Centre Dr. Muschaweck, Munich

The Bavarian Centre for Quality Assurance published for the years 2003 and 2004 following distribution of surgical techniques for primary hernias (Table 26.1).

With our data, the rate of suture repair would be as high as 40%. These data, however, do not contain the cases of the Hernia Center Dr. Muschaweck, because we do not operate on patients with a compulsory health insurance. Even within 1 year, there are marked changes in the surgical technique, but recurrences remain. The percentage of recurrent hernia operations averages over 6 years just below 13% with no trend to decrease (Table 26.2).

Material and Methods

In the Hernia Centre Dr. Muschaweck, I have operated a total of 12.115 patients with an inguinal hernia of whom 1781 patients (14.7%) were recurrent hernias.

Of these 1781 patients, 1446 patients had a first recurrence (81.2%), 253 patients a second-recurrence (14.2%) and 82 a third or higher re (4.6%).

In this article, we are focusing on the group of the first recurrences only, containing 1404 referred recurrences and all our known cases of own recurrences, 42 in total (since 1993).

Table 26.2. Percentage of recurrent hernia operations in Bavaria

Year	%
1999	13.3
2000	12.8
2001	12.4
2002	12.2
2003	12.1
2004	13.0

Database: BAQ Bayerische Qualitätssicherung 2004, Modul 12/3

It was interesting that by the end of the year 1994, after having operated on more than 1000 patients per year, the number of recurrences decreased sharply and fell below 1%, clearly indicating that the rate of having a recurrence after primary hernia surgery drops significantly with the experience of the surgeon. Figure 26.1 shows our own numbers of recurrences plotted over time and frequency of performed groin surgeries. The year 1997 is marked red, because we performed a 5-year follow-up study of all patients operated, which gave us detailed information about our outcome (only about two more patients developed a recurrent hernia).

Analysis of All 42 Known Cases of Our Own Recurrences

Out of the total number of 42 cases, 88.0% had been previously repaired with an open suture repair (Shouldice) and 12.0% with TIPP (suture plus mesh).

Analysis of Selected 55 First Recurrent Hernias (referred) from January 2004 until December 2005

Looking at the 100 cases of the past 3 years, we found that nearly half of them had their primary repair longer than 10 years ago, ranging even up to 65 years. Beyond 10 years there is practically no information about the technique obtainable. So, we selected a group which had a latency of no longer than 10 years, matching our own recurrences:

- 65.4% after suture repair
  - 34.5% Shouldice
  - 16.4% Bassini
  - 14.5% not classified
- 21.8% after laparoscopic repair (TAPP, TEP)
- 12.8% after different mesh repairs (Lichtenstein, Plug)

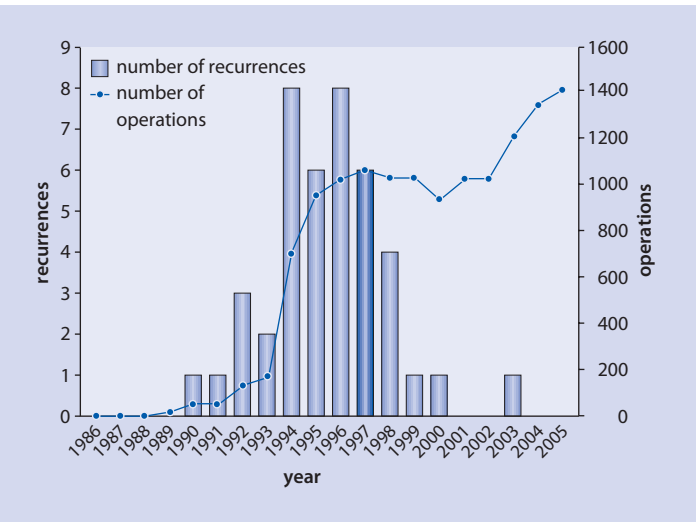


Fig. 26.1. Forty-two known cases of our own recurrences from 1989 until 12/2005

Probable Causes for the Development of a Recurrent Hernia

Analyzing all the patient´s specifications, we could find following coherences:

Age Peak. The referred patients show two age peaks, an early one around age 29–41 years and a second peak at age 64–68 years. My own patients showed only 1 peak at age 63–71 years.

Status of the Transversalis Fascia. In this group, only 10% of the patients who have been operated by a third surgeon, had a scar in the transverse fascia; in 90% it was completely intact without any signs of sutures or scars. This is a technical failure which attributes wrong negative results to the Shouldice repair.

Smoking. The history of smoking shows no difference between these two groups. As we operate on many athletes and businessmen, our clientele seems to consist of predominantly non-smokers anyway.

BMI. There is a peak between 22 and 26. So obesity does not seem to be a real cause in our clientele.

Type of Primary Hernia. I only can refer to my own recurrences; 68% were direct hernias, 20% indirect hernias and 12% were combined. This is exactly the proportion we encountered in the past 5 years in all patients undergoing surgery, so none of the types of hernias has an increased risk.

Type of Recurrent Hernia. In my own group there are more suprapubic recurrences. In the group of referred patients the lateral recurrences were slightly more frequent. Medial recurrences occurred with similar frequency in both groups.

Localization of the Primary Hernia and the Influence on the Site of Recurrences. The indirect and direct hernia showed nearly the same outcome of medial and lateral recurrences. In the group of combined hernias, the number of medial recurrences prevailed.

Different Surgical Techniques Used for a Recurrent Hernia. For our own cases, the plug repair was nearly always performed, rarely a Shouldice. Nowadays, the Lichtenstein repair is also chosen. In the group of referred recurrent hernias, 90% had an intact fascia transversalis, so in two thirds of those patients an open suture repair could be performed. Preferred surgical technique in case of a small defect (the so-called R 1 – recurrences after Campanelli) was the minimal-repair technique (Muschaweck repair) or the Shouldice technique in R 2.

In only one third of the patients was a mesh repair technique chosen. Plug repairs are performed when the fascia is weak and the defect is small. Lichtenstein or TIPP is performed when the whole groin floor is weak. This is typically found in cases of combined hernia.

Results

Although hernia surgery is the most commonly performed surgery in today's surgical world, there are as many different types of recurrences as different techniques, and even the most famous surgical techniques are still not done correctly:

In our experience, only 20% of the Shouldice repair are done correctly! In 80% of recurrent hernias where a Shouldice was supposedly performed, there was an absolute intact transverse fascia.

This gives us reason to believe that technical failure contributes a major factor to the high frequency of a recurrent hernia. Also, we learned that:

- Even specialists have a learning curve.
- Mesh repair is performed more than ever – however, meshes do not prevent recurrences!
- For combined hernias, a Shouldice repair is not sufficient and should be supplemented by a mesh, or a Lichtenstein repair should be performed.
- Elder patients are more prone to suffer from a recurrent hernia, probably due to instability of the tissue.

How Could Recurrent Hernia Be Avoided?

- Do the right thing: choose the appropriate type of repair ("tailored surgery").
- Do it right: communicate with the expert.
- Specialize: collect experience.

Routine must not lead to imprudence.

References

1. Hermanek P, BAQ München. Leistenhernien: Gibt es immer noch Rezidive? Münster, 2003
2. Campanelli G, Pettinari D, Nicolosi FM, Cavalli M, Avesani EC. Inguinal hernia recurrence: classification and approach. Hernia 2006; 11: 1–3
3. Klinge U, Krones CJ. Can we be sure that the meshes do improve the recurrence rates? Hernia 2005; 9: 1–2

4. Aufenacker TJ, Lange DH, Burg MD, Kuiken BW, Hensen EF, Schoots IG, Gouma DJ, Simons MP. Hernia surgery changes in the Amsterdam region 1994–2001: Decrease in operations for recurrent hernia. Hernia 2005; 9: 46–50
5. Janu PG, Sellers KD, Maniante EC. Recurrent inguinal hernia: preferred operative approach. Ann Surg 1998; 64(6): 569–573

Discussion

Kingsnorth: *I am surprised by the repair figures in your region of about one third of suture, open mesh and laparoscopic repair. Do you think there is any connection with the 16% recurrence rate you mentioned?*
Muschaweck: *I don't know. I was astonished also when I saw these numbers.*
Kingsnorth: *What I am wondering is, if you still promote the Shouldice repair in Germany; are you teaching this approach enough?*
Muschaweck: *You are right, this is a problem. In a lot of Shouldice repairs I saw that the transversalis fascia had not been touched and perhaps a lot of surgeons do not know how to perform a proper Shouldice repair. I think the teaching is worse in Germany, with the exception of Aachen. But one workshop a year in Aachen is probably not enough.*
Deysine: *Dr. Muschaweck, when you approach a recurrent hernia, what is your operation of choice?*
Muschaweck: *I first inspect the transversalis fascia. If there is a good fascia I intend to do a suture repair, if not I do a mesh repair with the Lichtenstein or the plug technique.*
Verhaeghe: *We did a nationwide questionnaire in France and we got the same results concerning the recurrence rates as you in Bavaria. Furthermore, we found no difference in repairs done at the universities, community hospitals or in private practise.*
Kingsnorth: *Dr. Verhaeghe, what did you find concerning the techniques of repair?*
Verhaeghe: *In France we also have a lot of surgeons doing laparoscopic repairs, but in spite of mesh repair and laparoscopic repair we have a nationwide recurrence rate of 16%, as in Bavaria.*
Schumpelick: *We have a 15% recurrence rate in our country and that has not changed over the years despite of 60% mesh repair in Germany. I think the surgeon is a risk. If you have a bad surgeon, you will have bad results with every technique. So the message from this meeting should be that we need more training to achieve better results.*
Bendavid: *A comment to Dr. Muschaweck and Dr. Deysine: it is difficult to decide the technique beforehand. You have to do your dissection and then decide the adequate method or repair.*
Muschaweck: *I agree with you.*
Chan: *If you do a proper operation in indirect hernia, identify the indirect sac and dissect the sac, you should not have a problem with a recurrence. The only concern is with the femoral region. Sometimes with your suture you lift up the ligament and open a femoral defect. Only in these cases do we put a preperitoneal mesh.*
Simons: *In cases of multiple recurrences after a transinguinal repair, the Dutch guidelines, based on a sound evidence, give the advice to use a preperitoneal approach for another repair. The recurrent transinguinal approach causes a lot of complications. We should advise the surgeons to send these patients to the experts who can do the recurrent repair laparoscopically or even by an open preperitoneal approach.*
Muschaweck: *In the complicated cases you have to use a tailored surgery.*
Simons: *I agree with that, but I would never go back anteriorly when the patients has been operated by that route once or twice before.*
Kingsnorth: *I think that is an important remark, thank you.*

26.2 Open Mesh Repair

M. Kurzer, A.E. Kark

Introduction

The two major changes in the field of hernia surgery over the last 20 years have been the use of prosthetic mesh and the development of laparoscopic hernia repair. It is now generally accepted that the use of mesh for inguinal hernia repair, whether open or laparoscopic, gives better results than sutured repair [1]. The efficacy of laparoscopic repair of groin hernia has also been clearly demonstrated [2]. However, because of the need for a high degree of technical skill, a consequent long learning curve, a risk of serious complications and increased cost, laparoscopic

repair has not been widely adopted for routine repair of inguinal hernias [3, 4]. Thus at present the majority of primary inguinal hernias are repaired using an open anterior method, usually a modification of the original Lichtenstein technique. The open tension-free mesh operation is technically more straightforward than laparoscopic repair – it is easy to teach and learn – and good results are readily obtainable with a low recurrence rate and low morbidity. It is also cost-effective and is the operation favoured by the vast majority of surgeons for the repair of primary inguinal hernia [4–7].

Increasing use of mesh and a greater interest in hernia surgery by general surgeons has meant that the overall incidence of recurrent hernia seem to be falling [8–11]. Nevertheless, despite the excellent overall results recurrences do occur – an indication that open mesh repair, despite it apparent ease, does undoubtedly have a learning curve and has to be carried out correctly to obtain good results [8, 12].

Does the Type of Recurrence Following Open Mesh Repair Differ from that Following Sutured Repair?

From what little evidence there is in the literature, from discussion with colleagues and personal experience, it seems that recurrence following an open mesh repair can take one of two forms: either a "direct" recurrence occurring medially or inferomedially, where mesh has pulled away from its attachment – or (less commonly) as an indirect sac, possibly missed at the initial operation, emerging through the reconstructed deep ring [12–15]. The mesh may have been incorrectly positioned, insecurely attached, may have contracted or may have been too small originally.

How Should These Recurrences Be Dealt With?

The important factors that should be considered in the evaluation of any hernia repair are [16]:

- Recurrence rates
- Complication rates
- Seriousness of potential complications
- Technical difficulty
- Rehabilitation
- Socio-economic factors.

It should be noted that not operating on, say, an elderly asymptomatic patient with comorbid conditions is also an option. The surgical choices are either to re-operate through the original incision or to approach the affected area from behind – a pre-peritoneal approach. The preperitoneal operation can be either open or laparoscopic.

In this article we would like to review the theoretical arguments for these three procedures, then examine the evidence available for each and finally to look at what is actually being done in practice.

Anterior Open Repair – Re-Operating Through the Previous Scar

The advantage of this method is that one simply redoes the original operation – no special equipment and, in theory, no special training is required. The procedure can also be done under local or regional anaesthetic if general anaesthetic is contra-indicated. The drawback is that one has to re-operate through scar tissue and distorted anatomy. Identifying the hernia may be more difficult, especially for an inexperienced or trainee surgeon, and there is a risk of damage to the testicular vessels. Are these difficulties and risks increased because of a theoretically greater degree of fibrosis if mesh has been used previously?

Does the Presence of Mesh Make Open Re-Operation More Difficult?

There is little information in the literature to guide us. Richard's retrospective review of the management of recurrent inguinal hernia in a general hospital identified 18 recurrences that had previously been repaired by an open mesh technique [17]. Thirteen subsequently had the recurrence also repaired by an open anterior method, and on analysis of the case notes the majority of these open operations were recorded as straightforward. The defect was usually readily identifiable and often only required repair with a nylon suture – presumably to reattach the edge or corner of the mesh. Gianetta's series of 141 patients with a recurrent inguinal hernia included nine failed open mesh repairs (four had been for a previous recurrence) [15]. All were re-repaired with further mesh (under local anaesthetic) and no mention was made of any increased difficulty in the presence of previous mesh. A large nationwide Scandinavian study which had access to the case notes of 87 recurrent inguinal hernias following previous open mesh repair made no comment regarding the degree of difficulty of the subsequent procedure [13]. The

authors' personal experience is that, as with all recurrent hernias, the difficulty of the dissection in the presence of scar tissue is unpredictable.

Outcomes of Open Anterior Reoperation After Previous Mesh Repair

Unfortunately, there is also very little in the literature to guide us on outcomes in this situation – the numbers are simply too small and surgeons' perceptions regarding the degree of difficulty of an operation may not be the best guide to how good the results are. One recent randomized study, although not a series of failed open mesh repairs, and therefore not strictly applicable to this situation, found a high re-recurrence rate (14.1% at 2 years) following open anterior re-operative surgery, even when carried out by experienced surgeons [18].

Testicular Complications

Testicular ischemia and atrophy following re-operative groin surgery seems to have received little attention in the literature. Figures for the incidence of this complication are conspicuous by their absence in most recent articles. Yet testicular atrophy is an important outcome measure as well as being a distressing complication with potential medico-legal implications [19, 20]. Not knowing the incidence of testicular problems makes it impossible for surgeons to assess the merits of a procedure or for the patient to make a truly informed decision when surgical options are discussed pre-operatively.

Posterior Approach – the Preperitoneal Repair

The advantages of operating through unscarred virgin territory are considerable. The procedure, in theory, is not affected by the previous surgery and can be standardized. Not operating through the fibrosis of the inguinal canal virtually eliminates the risk of damage to the testicular vessels, and the whole of the myopectineal orifice can be viewed. The ability to view all potential hernia sites is a great advantage, as recurrent hernias following sutured repair often consist of complex multiple defects, or are femoral hernias [21, 22]. Following a failed mesh onlay repair multiple defects may be less likely, but unsuspected femoral hernias do form a significant proportion of cases [13].

A preperitoneal repair can be open or laparoscopic. All preperitoneal repairs require wide dissection and exposure of the myopectineal orifice and all potential hernia sites as well as accurate placement of a sufficiently large piece of mesh in order to obtain the best results [23]. The drawbacks of laparoscopic, in contrast to open preperitoneal repair are the equipment costs, the long learning curve and the risk of serious complications [24].

Laparoscopic Repair for Recurrent Inguinal Hernia

Perhaps not surprisingly, laparoscopic repair of an inguinal hernia following a failed anterior mesh repair is not more complex than for a primary repair., although peritoneal tears are more likely [25, 26]. NICE – the National Institute for Clinical Excellence – in the UK, recommends considering laparoscopic repair in this situation because of the perceived difficulties and historically poor results generally obtained when re-operating using an anterior approach [27, 28]. Some series, however, have reported disappointingly high re-recurrence rates following laparoscopic repair, emphasizing the technically challenging nature of the procedure [29].

In the main, however, figures from large series suggest that outcomes, in terms of re-recurrence, differ little between anterior reoperation and laparoscopic repair as long as mesh is used for the anterior repair [9, 18].

Laparoscopic or Open Preperitoneal Repair?

The introduction and development of laparoscopic repair might seem to have made the operation of open preperitoneal repair redundant. However, with laparoscopic surgery there is the expense of equipment, the need to have trained, skilled laparoscopic surgeons, and the risk of serious complications [30, 31]. It is interesting that where surgeons have directly compared open preperitoneal with laparoscopic repair, the open repair is thought to be quicker or easier, with a shorter learning curve than laparoscopic repair [32–35]. This concurs with the authors' own experience [36]; we feel that open preperitoneal repair is an underrated procedure, which has great advantages in terms of a reduced learning curve and increased cost-effectiveness when compared with laparoscopic repair [37] (◘ Table 26.3).

Table 26.3. Repair of recurrence following open mesh repair

	Open anterior	Lap.	Open preperit.
Avoid scar	–	+	+
Testicular problems low	–	+	+
Post-op pain low	–	+	+
Rehabilitation rapid	–	+	+
No special equipment	+	–	+
Learning curve	+	–	±
Low risk of serious complications	+	–	+
Recurrence rate low	?	+	+

What Are General Surgeons Doing in Practice?

In the UK about 5% of all inguinal hernias are repaired laparoscopically [3, 38], although in Germany, for instance, the figure approaches 30%. A survey of current practice by surgeons in the Southwest of England showed that 90% of respondents used an anterior mesh repair for recurrent hernias if the previous repair had been sutured [4]. However, if mesh had been used initially, only 55% would carry out a further open mesh repair. While 7% of the surgeons surveyed recommended laparoscopic repair for any recurrent hernia, this increased to 17% if the primary repair had been done with mesh. A retrospective cohort study of inguinal hernia repair in one Scottish region found that 42% of recurrent hernias were being repaired laparoscopically [3]. Data from the Swedish Hernia Register showed that 25% of 2600 recurrent hernias operated between 1996 and 1998 25% were done laparoscopically [9]. In Denmark, where the use of laparoscopic hernia surgery is fairly low (about 5%) half of these are done for recurrence [5].

Recommendation – What Is the Best Practice?

Why are more recurrent hernias not repaired using preperitoneal techniques? Despite the obvious theoretical advantages of re-operating on failed open anterior repairs using a preperitoneal approach, surgeons who are uncomfortable with carrying out a laparoscopic repair will either use an open anterior operation again or refer patients to a surgical colleague skilled in laparoscopic techniques [4]. Reluctance to use an open pre-peritoneal repair is presumably related to unfamiliarity with the method, and a perception that it is a "difficult operation". Further evidence from randomized trials are not the answer – many surgeons will do the operation they feel most familiar with despite the "evidence" [39]. Recent data suggest that learning in small group training sessions and workshops is the activity most likely to change surgeons' practice [40]. Expert groups and meetings such as this one are probably the best route through which this can be achieved.

References

1. EU Hernia Trialists Collaboration (2002) Repair of groin hernias with synthetic mesh: meta analysis of randomised controlled trials. Ann Surg 235: 322–332
2. McCormack K, Scott NW, Go PM, Graham P, Ross SJ, Grant AM (2003).Laparoscopic techniques versus open techniques for inguinal hernia repair. In: Cochrane Database Systematic Review. CD001785. Oxford: Cochrane Library
3. Beattie DK, Foley RJE, Callam MJ (2000) Future of laparoscopic inguinal hernia surgery. Br J Surg 87: 1727–1728
4. Richards SK, Earnshaw JJ (2003) Management of primary and recurrent inguinal hernia by surgeons from the South West of England. Ann R Coll Surg 85: 402–404
5. Bay-Nielsen M, Kehlet H (2001) Quality assessment of 26,304 herniorrhaphies. Lancet 358: 1124–1128
6. Hair A, Duffy L, McLean J, et al (2002) Groin hernia repair in Scotland. Br J Surg 87: 1722–1726
7. Wara P, Bay-Nielsen M, Juul P, Bendix J, Kehlet H (2005) Prospective nationwide analysis of laparoscopic versus Lichtenstein repair of inguinal hernia. Br J Surg 92: 1277–1281
8. Aufenacker TJ, de Lange GH, Burg MD, et al. (2005) Hernia surgery changes in the Amsterdam region 1994–2001: Decrease in operations for recurrent hernia. Hernia 9: 46–50
9. Haapaniemi S, et al. (2001) Reoperation after recurrent groin hernia repair. Ann Surg 234: 122–126
10. Nilsson E, Haapaniemi S, Gruber G, Sandblom G (1998) Methods of repair and risk for reoperation in Swedish Hernia surgery from 1992 to 1996. Br J Surg 85: 1686–1691
11. Sandblom G, Gruber G, Kald A, Nilsson E (2000) Audit and recurrence rates after hernia surgery. Eur J Surg 166: 154–158
12. Amid PK (2002) The Lichtenstein repair in 2002: an overview of causes of recurrence after Lichtenstein tension-free herniorrhaphy. Hernia 7: 13–16

13. Bay-Nielsen M, Nordin P, Nilsson E, Kehlet H (2001) Operative findings in recurrent hernia after a Lichtenstein procedure. Am J Surg 182: 134–136
14. Johanet H, Cossa JP, Chosidow D, Marmuse JP, Benhamou G (1999) (Laparoscopic treatment of recurrent inguinal hernia after only mesh [French]. Ann Chir 53: 29–31
15. Gianetta E, Cuneo S, Vitale B, Camerini G, Marini P, Stella M (2000) Anterior tension-free repair of recurrent inguinal hernia under local anesthesia: a 7-year experience in a teaching hospital. Ann Surg 231: 132–136
16. Kingsnorth AN (2004) Treating inguinal hernias. BMJ 328: 403–404
17. Richards SK, Vipond MN, Earnshaw JJ (2004) Review of the management of recurrent ingunal hernia. Hernia 8: 144–148
18. Neumayer L, Giobbie-Hurder A, Jonasson O, Fitzgibbons R Jr., Dunlop D, Gibbs J, et al. (2004) Open mesh versus laparoscopic mesh repair of inguinal hernia. New Engl J Med 350: 1819–1827
19. Reid I, Devlin HB (1994) Testicular atrophy as a consequence of inguinal hernia repair. Br J Surg 81: 91–93
20. Wantz GE (1993) Testicular atrophy and chronic residual neuralgia as risks of inguinal hernioplasty. Surg Clin N Am 73: 571–581
21. Felix E (1995) Laparoscopic repair of recurrent hernias. Surg Endosc 9: 135–139
22. Scheurlein H, Schiller A, Schneider C, et al (2003) Totally extraperitineal repair of recurrent inguinal hernia. Surg Endosc 17: 1072–1076
23. Lowham A, Filipi C, Fitzgibbons RJ, et al. (1997) Mechanisms of hernia recurrence after preperitoneal mesh repair. Ann Surg 225: 422–431
24. Arregui ME (2005) Groin hernia repair by laparoscopic techniques: current status and controversies. World J Surg 29: 1052–1057
25. Nixon SJ, Kumar S (2005) The totally extraperitoneal approach (TEP) to inguinal hernia repair. Surgeon 3: 281–287
26. Lau H (2004) Endoscopic totally extraperitoneal inguinal hernioplasty for recurrence after open repair. ANZ J Surg 74: 877–880
27. National Institute for Clinical Excellence (2001) Technology Appraisal Guidance No. 18: Guidance on the use of laparoscopic surgery for inguinal hernia.
28. Guthy E, Boom H (1983) Multiple recurrences in inguinal hernia. Langenbecks Arch Surg 361: 316–318
29. Knook MTT, Weidema WF, Stassen LPS, van Steensel CJ (1999) Endoscopic totally extraperitoneal repair of primary and recurrent inguinal hernias. Surg Endosc 13: 507–511
30. Bittner R, Schmedt CG, Schwarz J, Kraft K, Leibl BJ (2002) Laparoscopic transperitoneal procedure for routine repair of groin hernia. Br J Surg 89: 1062–1066
31. Tamme C, Scheidbach H, Hampe C, et al. (2003) Totally extraperitoneal endoscopic inguinal hernia repair (TEP). Results of 5023 hernia repairs. Surg Endosc 70: 190–195
32. Beets GL (1999) Open or laparoscopic repair for recurrent inguinal hernia. Surg Endosc 13: 323–327
33. Champault GG (1997) Totally preperitoneal laparoscopic approach versus Stoppa operation: randomized trial of 100 cases. Surg Lap Endosc 7: 445–450
34. Janu PG (1998) Recurrent inguinal hernia; preferred operative approach. Am Surg 6: 569–573
35. Huang C-S (1999) Surgical treatment of recurrent groin hernia. J Formos Med Assoc 98: 122–127
36. Kurzer M, Belsham PA, Kark AE (2002) Prospective study of open preperitoneal mesh repair for recurrent inguinal hernia. Br J Surg 89: 90–93
37. Johansson B (1999) Laparoscopic mesh versus open preperitoneal mesh versus conventional technique for inguinal hernia repair: a randomizes multicenter trial (SCUR Hernia Repair Study). Ann Surg 230: 225–231
38. Bloor K, Freemantle N, Khadjesari Z, Maynard A (2003) Impact of NICE guidance on laparoscopic surgery for inguinal hernias: analysis of interrupted time series. BMJ 326: 578
39. Mc Greevy M (1998) Groin hernia and surgical truth. Am J Surg 176: 301–304
40. Smietanski M, Lukasiewicz J, Bigda J, et al. (2005) Factors influencing surgeons' choice of method for hernia repair technique. Hernia 9: 42–45

Discussion

Bendavid: *This was a very good paper, congratulations. I should also like to add the agreement of Prof. Nyhus. Though he prepared the extraperitoneal approach for many years, it did not in retrospect favourable, and rightly so because the recurrences went up to 25 and 30%; but from the day mesh started being used it became an absolute success story. Certainly, I support what you are saying. I personally feel that it's a much simpler operation than laparoscopic surgery and certainly a lot cheaper, and I agree with you that it ought to be done far more often than it is.*

Kurzer: *Thank you, Robert. It's interesting. Obviously the published series are very few and they have come from experts and you could criticize this as a procedure that has efficacy but not effectiveness. I very much like to hear from laparoscopic TEP surgeons about whether they are happy just to try it once or even to use it to teach their trainees, because I think it does have a place, it could have a place particularly in hospitals, in countries where laparoscopic equipment is very expensive. And people can then move on. I haven't tried this because I am not a laparoscopic surgeon, but I have done a lot of open preperitoneal repairs and I would like to try a TEP repair to see what the differences are. There must be people who have done both. I don't know whether this room feels that open preperitoneal repair should be given a chance?*

Bendavid: *That's a very good appeal and I think Dr. Duh will comment on this. Something I want to include in my conclusions eventually is: we started with animosity between the laparoscopic and the open surgeons. I am glad to see that over the years this has disappeared. Unfortunately, we still see some individual surgeons who maintain this animosity. The idea is to develop an opera-*

tion that adapts to the patient and that the patient adapts to the technique that the surgeon is doing. There is no doubt that everyone is excellent in his own technique and that good results can be obtained with every technique.
Duh: *A great, great paper. The comment you asked for about the laparoscopic perspective: I find that the TEP repair is a great repair for every kind of hernia. There is one population that it is probably better for than the open preperitoneal repair. In the US we have lots of patients with a huge, huge belly, big BMI. In these patients any open repair takes so much more work to get to the space. If you are able to get into the preperitoneal space with the scalpel, the operation is so much easier.*
Kurzer: *Can I ask you, have you done an open preperitoneal?*
Duh: *I have tried a few and found it difficult enough so that I stuck with laparoscopic repair.*
Franz: *I have just lived the question you're asking. I was trained to do both, the open and the laparoscopic approach at the same time. Recently I used an open approach for a difficult patient. I felt, based on what I had learned and knowing the TEP approach, that it is a fantastic operation in the current situation, it was much easier for me to perform an open preperitoneal approach having the better understanding of the anatomy I had learned laparoscopically.*
Kurzer: *Right. I learned my open preperitoneal from Georg Wanz when I was with him for about 3 days and he was a great teacher. I think that with all this, in fact it has been the mood of this meeting: you can't learn any of this from both, even Lichtenstein repair. You have just got to go to someone who does it well. I think these are the messages that must emerge. Perhaps not from your boss who doesn't do it well but from someone who does do it well. You just have to go and do it and watch it two or three times.*
Bendavid: *I agree that an obese patient is actually a totally different patient and I can also understand the hesitation of many people and I have referred them to laparoscopic surgery as well.*

26.3 TAPP

R. Bittner, J. Schwarz

Introduction

Recurrent inguinal hernia after laparoscopic and endoscopic hernioplasty is seldom [1, 2]. Therefore, it is not astonishing that although there are some anecdotal reports, only very few studies involving larger numbers of patients describing laparoscopic re-intervention (re-TAPP) due to recurrent hernia after transabdominal preperitonal patch plasty (TAPP) are available [3]. Re-TAPP is considered very difficult because the operating field can contain extensive scar tissue, depending on the size and type of mesh that was implanted primarily. Any amount of dissection that respects the tissue layers is extremely difficult, and the risk of complications is high. For this reason, it is said that posterior access should be used for recurrent hernia following anterior repair (e.g. Shouldice or Lichtenstein), and anterior access should be used following posterior repair (preperitonal mesh implantation, e.g. TAPP, TEP or Stoppa procedure). With this rule, scar tissue is circumvented, thus decreasing the complication rate (haematoma, infection, spermatic cord injury).

The following study investigated whether recurrent hernia following TAPP can be treated with the same type of access (re-TAPP), provided that the surgeon has adequate experience. Also, the complications that can be expected and factors influencing the rate of these complications were analyzed.

Patients and Methods

Between April, 1993 and December, 2005 12,687 laparoscopic hernia repairs were performed in the Marienhospital, Stuttgart (◘ Table 26.4). 1577 patients who presented with recurrent hernia had primary anterior repair, and 135 patients had preperitoneal mesh repair. Of the latter group 73 had TAPP repair at our institution and 62 came from other hospitals. ◘ Figure 26.2 shows the change in recurrence rate per year for our patients. Re-operation for recurrent hernia was performed using the laparoscopic approach in all 73 patients who had been treated in our hospital. This provided us with precise information regarding the cause of recurrence. Insufficient overlapping of the hernial defect during the primary operation was the cause in 18 (20.9%) patients, because initial meshes were too small (8×12 cm). Insuf-

Table 26.4. Laparoscopic hernia repair (TAPP)

Patients (n = 10,250)	Hernia (n = 12,678)
Unilateral repair n = 7813 (76.2%)	Primary hernia n = 10,962 (86.4%)
Bilateral repair n = 2473 (23.8%)	Recurrent hernia n = 1725 (13.6%)

ficient occlusion of the keyhole mesh was the cause for recurrence in 16 (18.6%) patients who received meshes with slit, which were also used in the beginning years. The most frequent cause of recurrence (39 patients, 45.3%) was mesh dislocation due to either inadequate fixation or lateral parietalization. The remaining 13 patients have not yet been operated on, so the cause cannot be determined. Our operative technique is described in the book Laparoscopic Hernia Surgery by Karl LeBlanc [4]. The important features of the operation are listed below:

1. The peritoneal incision must be performed clearly above the upper rim of the mesh or region of scar tissue, which is usually easily discernible. Usually, the dissection is made between the mesh and abdominal wall, strictly along the anatomical land marks (rectus muscle, symphysis and Cooper's ligament, transverse fascia).
2. When necessary, the medial umbilical plica is severed in order to gain access to the symphysis through a region that is free of scar tissue.
3. The same applies to the epigastric vessels, which usually cannot be left undamaged when dissection proceeds strictly along the rectus muscle and transverse fascia.
4. The correct direction of dissection is from normal tissue to scar tissue.
5. The dissection should proceed from familiar structures to scar tissue.
6. Thorough haemostasis should always be performed.
7. Usually the mesh is not removed.
8. There is usually no scar tissue in the region of the hernia, which makes dissection here comparatively easy.
9. The dissection is usually finished by completely depicting the anatomic structures, including the large blood vessels and psoas muscle, as is also the case during primary surgery.
10. A second mesh is implanted with sufficient overlapping of the defect.
11. If it is not possible to completely separate the spermatic cord from the lateral-caudal region of the mesh, then a mesh with a slit should be implanted and the slit should be secured with an adequately sized mesh using the double-buttress technique.

Results

The results of primary laparoscopic hernioplasty, recurrent hernia repair after anterior access and recurrent hernia repair after preperitoneal access are pre-

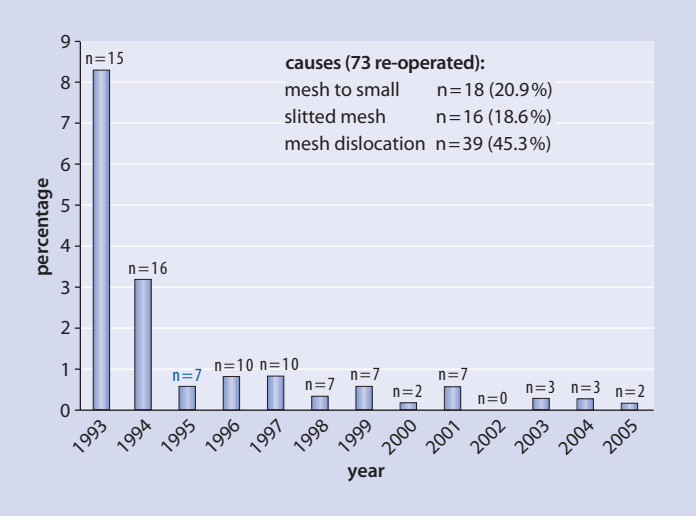


Fig. 26.2. Recurrence after TAPP

Table 26.5. Laparoscopic hernia repair (TAPP) – results

	Primary hernia (n = 10,962)	Recurrence after anterior repair (n = 1590)	Recurrence after preperitoneal mesh repair (n = 135)
Operating time (median) [min]	40	45	75
Morbidity [%]	2.8	3.5	8.1
Re-operation rate [%]	0.3	0.6	2.2
Recurrence rate [%]	0.7	1.0	0.74
Return to work (median) [days]	14	17	17
Age (median)	59 (17–97)	61 (18–92)	59 (29–90)
BMI (median)	25	25	25

sented in Table 26.5. While the results of the first two groups differ only slightly, the operating time and morbidity rate are significantly longer and higher for the last group. The list of complications is presented in Table 26.6.

Table 26.6. TAPP after posterior repair – complications

Total complications	n (%)	Therapy
Bladder injury	3 (2.2)	1x lap. rev., 2x cons. ther.
Bowel injury	2 (1.5)	1x lap. rev., 1x open rev.
Mesh infection	1 (0.73)	Ant. + post. rev. drain
Orchitis/testicular atrophy	1 (0.73)	Conservative
Urinary retention	1 (0.73)	suprapubic cath.
Wound infection	1 (0.73)	Conservative
Trocar-site hernia	1 (0.73)	Operative
Ileus	1 (0.73)	Operative external hosp.
Total	11 (8.1)	

Table 26.7 shows a reduction in complication rate with increasing experience of the surgeon. The most important complication was bladder injury in the first observational period. One of these patients had a recurrent inguinoscrotal hernia due to mesh slit insufficiency, one had the fourth recurrence after previous anterior, and posterior repair as well. The first patient was treated laparoscopically after 5 days, and there were no further complications. The other two were treated conservatively. Adhesiolysis caused both cases of bowel injuries. One patient had the fourth recurrence (three anterior and one posterior repair) and the other patient had the eighth recurrence after multiple conventional and laparoscopic operations. Both patients had extensive abdominal adhesions of small intestine or sigmoid colon with the inguinal region.

A medial recurrence can be repaired much faster and without as many complications as a lateral recurrence, as can be seen in Table 26.8.

Table 26.9 shows that there is a certain connection between the cause of recurrence and complication rate.

Discussion

Laparoscopic surgery of recurrent hernia following laparoscopic/endoscopic repair (re-TAPP) is a difficult operation. Operating time and complication rates are significantly longer and higher than for both primary hernia repair and recurrent hernia after anterior repair. Our experience indicates that the most important com-

Table 26.7. TAPP after preperitoneal mesh repair. Results (n = 135), "learning curve"

n (Prof.)	1–45 (1–20) (6/1993–12/1998	46–90 (21–40) (12/1998–02/2002)	91–135 (41–56) (02/2002/–11/2005)
Operating time (median) [min] (Prof.)	82.5 (87.5)	71.0 (85)	77.0 (57.5)
Morbidity [%]	14.0	8.0	2.0
Re-operation rate [%]	2.2	2.2	2.2
Recurrence rate [%]	–	–	2.2
Return to work (median) [days]	18.0	17.0	17.0

Table 26.8. TAPP after preperitoneal mesh repair

	Medial (direct) n = 57	Lateral (indirect) n = 78
Operating time (median) [min]	60.0	92.5
Morbidity [%]	5.2	10.2
Re-operation rate [%]	–	2.6
Recurrence rate [%]	–	1.3
Return to work (median) [days]	16.0	17.0

Table 26.9. TAPP after preperitoneal mesh repair

	Slit insufficiency n = 16	Mesh dislocation n = 62
Operating time (median) [min]	98.5	89.5
Morbidity [%]	6.25	11.2
Re-operation rate [%]	–	3.2
Recurrence rate [%]	–	1.6
Return to work (median) [days]	14.0	19.0
Hospital stay	7.0	6.0

plication is bladder injury. Bowel injuries occur only when extensive adhesions are present between bowel and inguinal region after multiple previous operations. Surgery of recurrent direct inguinal hernia is easier than for recurrent indirect hernia. A correlation between the type and size of mesh that was implanted during the first operation and the amount of scar tissue could be seen in isolated cases. Scarring was less pronounced for smaller, lighter meshes than it was for heavier meshes, which were used in the early phase. Noteworthy is that despite all of these difficulties, only one patient had testicular atrophy. The results are clearly dependent

on the experience of the surgeon. With adequate surgical experience, the complication rate is not higher than for primary operation. Since the re-recurrence rate and length of time off work does not differ significantly from primary hernia surgery, the indication for at least diagnostic laparoscopy is justifiable. There is no question that conversion is necessary when serious difficulties arise or when conditions in the operating field are too complex.

Conclusions

The recurrence rate following laparoscopic hernia repair is low. The classic approach for repair is an anterior reparation. Re-TAPP is difficult and technically demanding. The morbidity rate is dependent on the experience of the surgeon and the location of the defect as well. Furthermore, the morbidity rate appears to be dependent on the type and size of mesh and individual scar tissue formation. In experienced hands, re-TAPP can be performed with the same effectiveness and efficiency as primary repair.

References

1. Bittner R, Schmedt CG, Schwarz J, et al. Laparoscopic transperitoneal procedure for routine repair of groin hernia. Br J Surg 2002; 89: 1062–1066
2. Schmedt CG, Sauerland S, Bittner R. Comparison of endoscopic procedures vs Lichtenstein and other open mesh techniques for inguinal hernia repair. A meta-analysis of randomized controlled trials. Surg Endosc 2005; 19: 188–199
3. Leibl BJ, Schmedt CG, Kraft K, et al. Recurrence after endoscopic transperitoneal hernia repair (TAPP): causes, reparative techniques, and results of the reoperation. J Am Coll Surg 2000; 190: 651–655
4. Bittner R, Schmedt CG, Leibl BJ. Transabdominal pre-peritoneal approach. In: LeBlanc K (ed) Laparoscopic hernia surgery – an operative guide. Arnold, London, 2003

Discussion

Duh: *You obviously have fantastic experience in laparoscopic repair. I just have a comment and I want to ask you your opinion about this. I have done about 20 re-TAPPs, but I have done them differently from what you recommended here, because always almost the mesh migrated towards the head, and the defect is frequently lateral and inferior. So I just cut only the inferior border of the previous mesh and I find it easy to leave the other mesh, not even to dissect it. What do you think of that technique?*
Bittner: *I am sorry that I didn't comment on this during your talk. We discussed it with Kukleta. The problem is, as demonstrated in your video, that you didn't do lateral parietalization. So far, it must come to a recurrence. And your question is the right answer. In our opinion, it is not necessary to make a slit; moreover it is better to do no slit.*

26.4 TEP

G.S. Ferzli, G. Elie Al-Khoury

Introduction

There has never been a shortage either of technical creativity or of methodology for treating inguinal hernia. In De Medicina, Cornelius Celsus (circa 25 B.C. to A.D. 50) [1] described his approach to this condition (bubonocele). Even before that, the mummy of Pharaoh Merneptah (1224–1214 BC) showed evidence of a wound in the groin, with the scrotum separated from the body, indicating that surgery may have been performed [2]. Since Bassini's procedure was developed, more than 70 techniques have been described [3]. Among them is the preperitoneal approach described by Stoppa [4]. It provides direct access to the entire myopectineal orifice of Fruchaud, and reinforces the transversalis fascia through the placement of a large mesh. Over the past 15 years, meanwhile, the laparoscopic management of inguinal hernia has evolved from a simple plug and patch to the transabdominal preperitoneal repair (TAPP) and the totally extraperitoneal approach (TEP) [5–7], which essentially reproduces the Stoppa repair. Results of the TEP have been quite satisfactory, and it is often recommended for bilateral and recurrent inguinal hernias (8). However, recurrences after TEP are inevitable [3, 6, 9–12], with treatment for them being either the open standard approach, a TAPP, or another TEP. This report describes the management of these recurrences and our 10 years of experience with TEP after a first TEP.

Methods

The approach to TEP after TEP does not vary from the standard TEP repair [7, 13]. The patient is placed in a supine, slightly flexed position, with arms tucked in. A slight Trendelenburg position is helpful. A general endotracheal anaesthesia is used, and the video monitor is positioned at the patient's feet. A small incision is made in the inferior aspect of the umbilicus at the site of the old scar. Dissection is continued down to the rectus fascia, which is incised horizontally over either the left or the rectus muscle. The rectus muscle is bluntly separated from the midline linea alba, and blunt and sharp dissection is done medial to the rectus muscle in the direction of the pubic symphysis on the midline. Further blunt finger dissection is performed extraperitoneally on the midline toward the pubic symphysis, and must not be forced. (Blunt finger dissection across the midline would not be feasible.)

This process allows the placement of a 10-mm blunt-tip trocar, which is secured in place. CO_2 insuflation tubing is attached to the cannula, with a pressure of 10 mm of mercury being maintained. A 10-mm 30o operative scope is introduced in the trocar. It is important to stay in the midline parallel to the rectus muscle and as anterior as possible, in order to avoid bleeding or peritoneal tearing. Rectus muscle fibers will be visible. The thin, translucent, filmy fascial layer that represents the posterior lamina or the inner portion of the transversalis will be absent. The scope is gently advanced on the midline, tangential and parallel to the rectus. It is best to choose the least resistant pathway toward the bone (the pubic symphysis on the midline). This process is carried out above, meaning anterior, to the old mesh.

In some cases, the CO_2 insufflation pressure must be increased to 12 or 15 in order to increase the volume of working space, which is small at best. As soon as there is room enough to make it possible, an additional 5-mm trocar is introduced at the old infraumbilical trocar site. Once the space is further developed, a second 5-mm trocar is introduced, also on the midline (Fig. 26.3). Adhesions are taken sharply without cautery. Small branches of the epigastric vessels are divided. Excellent hemostasis should be maintained, and the epigastric vessels ligated to avoid bleeding and to provide a slightly larger working area. If possible, Bogros's space should be accessed first, rather than the midline. It is important to remember that if there is a hernia, there will be no adhesions around it. Therefore, if the midline is much scarred, there is no direct hernia.

The usual anatomical landmarks present in a primary TEP (midline pubic symphysis, Cooper's ligament, Hesselbach's triangle, and the transverse abdominis muscle) will not be visible. What can be seen are the epigastric vessels. These will lead to the hernia, either medially (direct) or laterally (indirect). The assistant should reach across to the scrotum and pull on the cord structures. This helps the surgeon recognize where these structures are. Everything is to be fibrosed and sclerosed. Applying pressure from the outside at the level of the anterosuperior iliac spine or on the cord structures will help with orientation. At any point when the surgeon feels that a bladder injury might have oc-

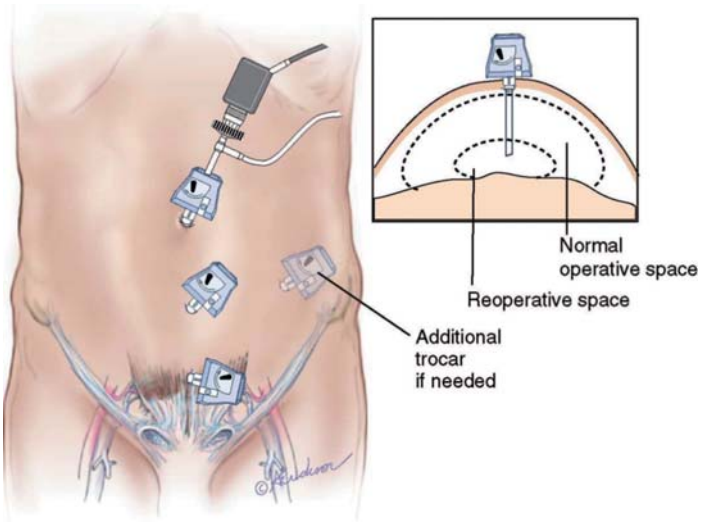


Fig. 26.3. Trocar placement

curred, a Foley catheter should be inserted. The bag will be automatically distended with CO_2. The surgeon may also choose to administer methylene blue for further identification of possible urine leakage, and a close suction drain should be left in the preperitoneal space. If bowel injury is identified at any time during this dissection, conversion is mandatory.

Contrary to what is expected during a primary TEP, the contralateral space will not open up. Likewise, the Retzius space will not open, which leaves the working area extremely small. There are several cardinal rules that serve as guides during primary TEP, such as: inability to see the Cooper femoral canal indicates a direct hernia, and inability to see the vas deferens indicates an indirect hernia. Neither of these is applicable in the TEP after a TEP. Also, if a peritoneal tear occurs and pneumoperitoneum follows, it should not be vented.

In cases where the working space cannot be maintained, conversion is advisable. After the Retzius space is developed in primary TEP, the weight of the bladder keeps it open despite a pneumoperitoneum; but in TEP after TEP this is simply not the case. It is always advisable to scan the cord for the possibility of a lipoma. For an indirect hernia, dissection of the cord structures is done in a direction perpendicular to these structures, sweeping them gently, medially and posteriorly, while holding the sac with the other grasper laterally and superiorly (Fig. 26.4). An alternative procedure is to pivot the hernia sac medially and posteriorly, dissecting the cord structures in a perpendicular fashion while sweeping in a lateral and posterior direction (Fig. 26.5). An advantage of this approach is easy identification of a cord lipoma and the margins of the indirect sac, as well as avoidance of injury to the vascular structures.

The surgeon can also alternate dissection between the medial and lateral approaches. A window is thus created between the cord structures and the sac. The latter is either totally invaginated and reduced or else transsected, leaving the distal end in situ and closing the proximal end, after ensuring that the contents are not injured. The cord structures must be totally parietalized with the posterior wall, so that no element crosses the preperitoneal prevesical space. In a direct hernia, the contents are always freed with gentle traction and counter traction, a process rarely requiring sharp dissection. A rolled edge or fold nicely demarcates the separation between the redundant thickened transversalis and the peritoneal sac (Fig. 26.6). Dissection continues along this fold, reducing the entire contents of the defect. The margins of the direct hernia defect should be cleansed of all tissue.

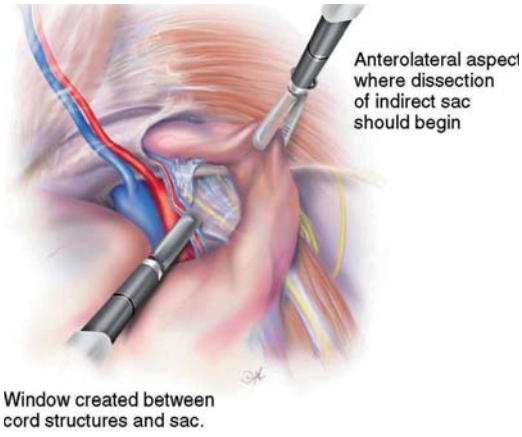


Fig. 26.4. Indirect hernia dissection

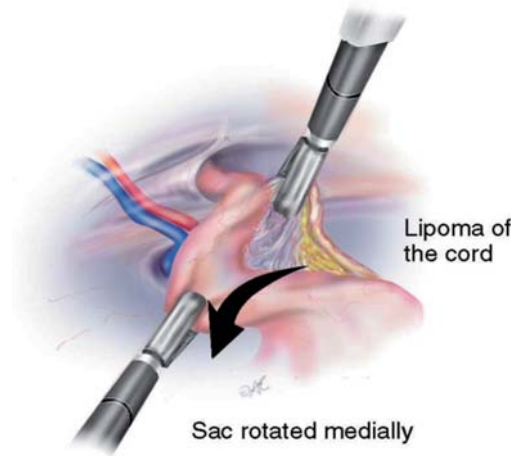


Fig. 26.5. Lipoma of the cord

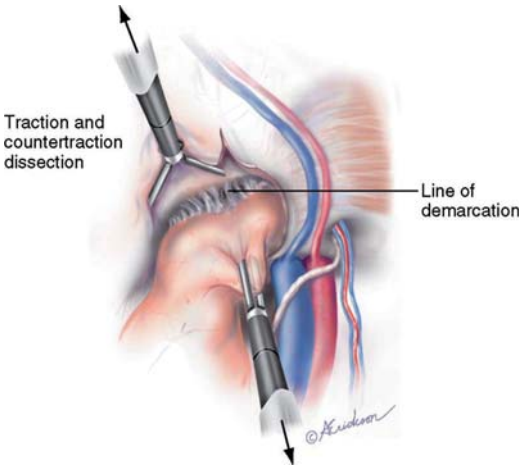


Fig. 26.6. Direct hernia dissection

Polypropylene mesh was used in all our patients. A Jackson Pratt drain was left in one, and he also required a Foley catheter. Small peritoneal tears occurred in seven cases. In one patient, pneumoperitoneum mandated conversion to an open Lichtenstein repair, while in another case bleeding obscured the field, mandating a conversion.

Results

From September 1991 to September 2005 we repaired 1526 inguinal hernias in 1156 male patients, using a totally extraperitoneal TEP approach, with 786 of them being unilateral and 370 bilateral. Of these, 141 patients were operated on for recurrence (12.2%), including 21 after a previous TEP. In our early experience we approached recurrence via the conventional anterior method, but after 1995 as we gained experience with the procedure, we began to perform TEP after TEP repair. Of the 21 TEP repairs after a primary TEP approach, 18 were indirect hernias and three direct. All patients were male. The mean operative time was 47 minutes (31 to 120). The mean age was 52 (29 to 79). We also operated on 22 patients who presented with primary hernias contralateral to a TEP repair done between 13 months and 12 years before. The mean operative time was 36 min (20 to 100). The mean age was 56 (35 to 84). In three patients with TEP after TEP, the space could not be opened and the cases were converted to an open preperitoneal Lichtenstein repair. Seven patients had peritoneal tears, with one of them obliterating the space and requiring conversion to the open Lichtenstein [13]. The epigastric vessels were ligated in seven patients, and one had excessive bleeding in the field, leading to conversion. There were no complications, no bladder or bowel injuries, and no fatalities. In one patient a Jackson Pratt drain was left owing to the possibility of a bladder injury (though none was found). No patient required transfusion or developed a preperitoneal haematoma, and all were discharged on the day of surgery.

Discussion

Re-operation by the inguinal approach is the method of choice for recurrence after an open preperitoneal Stoppa repair [15, 16]. Thus it would be intuitive that a recurrence after TEP be repaired by an open tension free Lichtenstein repair [17]. While this method is followed by most surgeons, reports concerning use of the TAPP for these recurrences are frequently found in the literature (Table 26.10) [10, 18–23]. Leibl et al. [18] and Felix et al. [19] concluded that endoscopic re-repair is only possible by the transperitoneal approach. In 2003, Tamme et al. [20] reported on 29 recurrences following 5023 TEP procedures, with 18 treated by open repair, three by TAPP, two by TEP, and six being done at another institution.

There is no question that re-entry into the preperitoneal space is challenging. Wide cleavage and dissection of the Retzius space may be particularly difficult, so much so that in three of our patients the space could not be opened at all. In addition, mesh placement behind

Table 26.10. Repair of recurrence after endoscopic hernia repair

Study	Recurrent hernias after endoscopic repair	TEP original operation	Repair
Felix 1998 [19]	34	11	TAPP 29, Open 4
Knook 1999 [21]	34	9	TAPP
Leibl 2000 [18]	46	0	TAPP
Chowbey 2003 [23]	6	6	TAPP 4
Tamme 2003 [20]	23	23	TEP 2, TAPP 3, Open 18
Richards 2004 [22]	8	0	TEP 1, Open 7
Ferzli 2004 [10]	12	12	TEP

the transversalis fascia in the preperitoneal space has led to growing concern among urologists and vascular surgeons. This centres on the extreme risk and difficulty, if not impossibility, of performing urological and vascular operations, in particular radical prostatectomy and lymph node dissection, subsequent to open and laparoscopic preperitoneal hernia repair [3, 24–26].

Recently, however, Stolzenburg et al. [27, 28] reported on 750 patients who underwent endoscopic extraperitoneal radical prostatectomies (EERPE). Of these, 14 had had previous laparoscopic hernia repair with mesh placement, eight had had prior TEP (two bilateral), and six had had prior TAPP. In one of the cases with prior bilateral TEP, the space could not be created and the procedure was performed by a transperitoneal approach. Their conclusion was that previous minimally invasive hernia repair with mesh placement made EERPE more demanding, but was not a contraindication. In a different study, Patterson et al. [29] discussed the problem of re-entering the preperitoneal space after prior lower abdominal surgery. They found that TEP could indeed be carried out safely in the presence of scars from such procedures, including lower midline suprapubic and paramedian incisions.

At this institution, our experience with TEP repair after a primary TEP has been documented in the Results section. In addition to the 21 successful procedures noted, we were also able to operate on 22 patients who presented with primary hernias contralateral to a TEP. The incidence of peritoneal defects associated with TEP depends on the presence of adhesions and prior scarring [11]. These defects are reportedly as high as 46% in this group [30]. In our series, seven patients (33%) developed peritoneal tears. During primary TEP, pneumoperitoneum should not lead to conversion, nor does it occlude the working space. However, in TEP after TEP, the occurrence of peritoneal tears may lead to conversion, as happened in one of our patients owing to loss of the working space. Venting the pneumoperitoneum transperitoneally is not helpful. As previously noted, in primary TEP the weight of the bladder will keep the Retzius space open, maintaining a working space despite a full pneumoperitoneum. This is not the case in TEP after TEP.

Arregui [6] concluded that the overall complication rate for laparoscopic repair is the same as for the open procedure, with the potential for serious injury being higher in laparoscopy. There were no complications in our series, but in one patient the possibility of a bladder injury led to the placement of a Jackson Pratt drain, the injection of methylene blue, and the use of a Foley catheter. There was in fact no demonstrable injury. In another study, Stolzenburg [27] reported two bladder injuries in 14 patients who underwent EERPE after previous laparoscopic hernia repair. None of those in our series required transfusion, nor was there any haematoma formation, but we found it essential to maintain proper haemostasis during the entire procedure because the presence of minute bleeding can obscure the clarity of vision. This results in difficult identification of the anatomy and possible conversion, as occurred in one of our cases. That brought the total conversion rate in our series to five out of 21 patients (24%).

TEP has a prolonged learning curve for the general surgeon [31, 32], with some stating that it is in the range of 30 to 50 procedures [9]. The cooperative Veteran Affairs study [33] suggests that the number is much higher. Indeed, the recurrence rate associated with laparoscopic repair was greater than 10% for the 58 surgeons who had performed 250 or fewer such repairs in any category. Recurrence was less than 5% for the 20 surgeons who reported having performed more than 250 laparoscopic repairs. The causes of recurrence have been clearly identified as missed hernia, missed lipoma, migration of the mesh, slit in the mesh, and one poorly shaped or too small [6, 23, 34, 35]. In our series, 18 patients had an indirect hernia resulting from missed hernias and migration of the mesh (especially in recurrent large scrotal hernias). The three direct hernias also resulted from displacement of the mesh.

Arregui [6] was quite correct in stating that it is imperative to remove all excess preperitoneal fat and cord lipoma, to reduce the sac, and to identify the anatomical structure properly prior to mesh placement. The size of mesh used in TEP after TEP is certainly much smaller than in primary TEP, yet it must be as large as the working space. It should be fluffy and fairly redundant, because of the shrinking process over time [3].

Conclusions

The key to successful hernia repair is mastery of the anatomy. It is easy for urologists to learn endoscopic extraperitoneal pelvic lymph node dissection, because they are familiar with the anatomy and have only to learn the technique. The same applies to general surgeons learning laparoscopic cholecystectomy. They are familiar with the terrain, and need only learn the method. TEP has a very prolonged learning curve for general surgeons because they have to learn the anatomy as well as the procedure, both at the same time. It has been recommended that they perform ten open Stoppa repairs before undertaking TEP [36]. In fine,

general surgeons should master the anatomy before attempting a TEP, and most certainly before attempting a TEP after TEP. Finally, it is surgeons' recognition of their own skills, expertise, and limitations that will dictate their choice of the proper procedure.

References

1. Papaoramidou MS, Chirstopoulou-Aletras H (2005) Treatment of hernia in the writings of Celsus (first century AD). World J Surg 29: 1343–1347
2. Lau WY (2002) History of treatment of groin hernia. World J Surg 26: 748–759.
3. Amid PK (2005). Groin hernia repair: open techniques. World J Surg 29: 1046–1051
4. Stoppa R, Warlaumont CR (1989) The preperitoneal approach and prosthetic repair of groin hernia. In: Nyhus LM, Condon R (eds) Hernia. Lippincott, Philadelphia, pp 1898–225
5. Ferzli GS, Massad A, Albert P (1992) Extraperitoneal endoscopic inguinal hernia repair. J Laparoendosc Surg 2 (6): 281–286
6. Arregui ME, Young SB (2005) Groin hernia repair by laparoscopic techniques: current status and controversies. World J Surg 29: 1052–1057
7. Ferzli GS, Kiel T (1995) Evolving techniques in endoscopic extraperitoneal herniorrhaphy. Surg Endosc 9 (8): 928–930
8. Grunwaldt LJ, Schwaitzberg SD, Rattner D, Jones D (2005) Is laparoscopic inguinal hernia repair an operation of the past? J Am Coll Surg 200, No. 4, 616–620
9. Davis CJ, Arregui ME (2003) Laparoscopic repair for groin hernias. Surg Clin N Am 83: 1141–1161
10. Ferzli GS, Shapiro K, DeTurris SV, et al. (2004) Totally extraperitoneal (TEP) hernia repair after an original TEP. Is it safe, and is it even possible? Surg Endosc 18 (3): 526–528
11. Scheuerlein H, Schiller A, Schneider C, Scheidbach H, Tamme C, Konckerling F (2003) Total extraperitoneal repair of recurrent inguinal hernia results with 179 consecutive patients. Surg Endosc 17: 1072–1076
12. Feliu X, Jaurrieta E, Vinas X, Macarulla E, Abad JM, Fernandez-Sallent E (2005) Recurrent inguinal hernia: a ten- year review. Hernia 9(2):120–4
13. Ferzli GS (1996) Extraperitoneal herniorrhaphy. In: Lanzafame RJ, Prevention and Management of Complications in Minimally Invasive Surgery. New York, Tokyo, Igaku-Shoin, pp 139–154
14. Amid PK (2004) Lichtenstein tension-free hernioplasty: its inception, evolution, and principles. Hernia 8: 1–7
15. Stoppa RE (1995) The preperitoneal approach and prosthetic repair of groin hernias. In: Nyhus LM, Condon R (eds) Hernia. Philadelphia, Lippincott pp 188–206
16. Wantz GE (1995) Personal experience with the Stoppa technique. In: Nyhus LM, Condon R (eds) Hernia. Philadelphia, Lippincott pp 206–210
17. Barrat C, Surlin V, Bordea A, Champauld G (2003) Management of recurrent inguinal hernias: a prospective study of 163 cases. Hernia 3: 125–129
18. Leibl BJ, Schmedt CG, Kraft K, Ulrich M, Bittner R (2000) Recurrence after endoscopic transperitoneal hernia repair (TAPP): causes, reparative techniques and results of the reoperation. J Am Coll Surg 190: 651–655
19. Felix E, Scott S, Crafton B, Geis P, Duncan T, Sewell R, McKernan B (1998) Causes of recurrence after laparoscopic hernioplasty: a multicenter study. Surg Endosc 12: 226–231
20. Tamme C, Scheidbach H, Hampe C, Schneider C, Kockerling F (2003) Totally extraperitoneal endoscopic inguinal hernia repair (TEP). Surg Endosc 17: 190–195
21. Knook MTT, Weidema WF, Stassen LPS, van Steensel CJ (1999) Laparoscopic repair of recurrent inguinal hernias after endoscopic herniorrhaphy. Surg Endosc 13: 1145–1147
22. Richards SK, Vipond MN, Earnshaw JJ (2004) Review of the management of recurrent inguinal hernia. Hernia (8) 144–8
23. Chowbey PK, (2003) Recurrent hernia following endoscopic total extraperitoneal repair. J Laparoendosc Adv Tech A 13(1):21–5
24. Katz EE, Patel RV, Sokoloff MH, Vargish T, Brendler CB (2002) Bilateral laparoscopic inguinal hernia repair can complicate subsequent radical retropubic prostatectomy. J Urol 167: 637–638
25. Cook EL, Afzal N, Cornaby AJ (2003) Laparoscopic hernia repairs may make subsequent radical retropubic prostatectomy more hazardous. BJU Int 91: 729
26. Cooperberg MR, Downs IM, Carroll PR (2004) Radical retropubic prostatectomy frustrated by prior laparoscopic mesh herniorrhaphy. Surgery 135: 452–453
27. Stolzenburg JU, Anderson C, Rabenalt R, Do M, Ho K, Truss MC (2005) Endoscopic extraperitoneal radical prostatectomy in patients with prostate cancer and previous laparoscopic inguinal mesh placement for hernia repair. World J Urol 27: 1–5
28. Stolzenburg J, Ho KMT, Do M, Rabenalt R, Dorschner W, Truss M (2005) Impact of previous surgery on endoscopic extraperitoneal radical prostatectomy. Urology 65: 325–331
29. Paterson H, Casey J, Nixon S (2005) Total extraperitoneal laparoscopic hernia repair in patients with previous lower abdominal surgery. J Laparoendosc Adv Surg 15 (2) 121–124
30. Lau H (2004) Endoscopic totally extraperitoneal inguinal hernioplasty for recurrence after open repair. J Laparoendosc Adv Surg Tech 14 (2): 93–96
31. DeTurris SV, Cacchione RN, Mungara A, et al. (2002) Laparoscopic herniorrhaphy: beyond the learning curve. J Am Coll Surg 194 (1): 65–73
32. Kaafarani H, Itani K, Giobbie-Hurder A, et al. (2005) Does surgeon frustration and satisfaction with the operation predict outcomes of open or laparoscopic inguinal hernia repair? J Am Coll Surg 200 (5): 677–683
33. Neumayer L, Giobbie-Hurder A, Jonasson O, et al. (2004) Open mesh versus laparoscopic mesh repair of inguinal hernia. N Engl J Med 350 (18): 1819–1827
34. Liem MS, van Duyn E, van der Graaf Y, van Vroonhoven T (2003) Recurrences after conventional anterior and laparoscopic inguinal hernia repair. A randomized comparison. Ann Surg 237: 135–141
35. Choy C, Shapiro K, Patel S, Graham A, Ferzli G (2004) Investigating a possible cause of mesh migration during totally extraperitoneal (TEP) repair. Surg Endosc 18: 523–525
36. Pawanindra L, Kajla RK, Chander J, et al. (2004) Laparoscopic total extraperitoneal (TEP) inguinal hernia repair. Overcoming the learning curve. Surg Endosc 18: 642–645

Discussion

Fitzgibbons: *Brilliant presentation, I really enjoyed it. But I can't believe that you could tell us that we shouldn't be concerned about the urologists having to do laparoscopic prostatectomy to that kind of mass. Literature says that prostatectomy is more difficult after a laparoscopic herniorraphy. I am not sure which article you are referring to. The last article you see on the screen, is a German paper and they had 14 cases. They describe a lot of difficulties and they had to adapt their technique. Actually they are such good surgeons that they had no more complications. But still the procedure is much more difficult. I just rise to say that we don't really do these laparoscopic operations.*

Ferzli: *Mr. Fitzgibbons, I did laparoscopic prostatectomy from 1997 and I can tell you that if a patient had a prior TAPP or TEP I do an open prostatectomy. Prostatectomy is extremely difficult, you have to respect the anatomy more than with any other organ and you should not start with already difficult conditions. I totally agree with you.*

Dysine: *I rise because I am a person that has never done a laparoscopic repair over hernia. Actually, I admire all the speaker this morning. I have an enormous admiration for their capacity to suffer without even winking. Operations that can be done in about 25 min seem to be quite difficult to me. I apologize I really cannot see the reason why you are doing that. I have to keep on studying that.*

Köckerling: *Congratulations for your talk and wonderful technique. As you know, we have an experience of more than 8000 TEP repairs and we have done a few recurrences after TEP also in a TEP technique but we prefer to offer the patient a Lichtenstein or TAPP repair because to do a re-TEP is very, very demanding; you could show us how demanding it is and it is reserved for the absolute specialist in the field.*

Ferzli: *I am totally in agreement and I just wanted to show you what the space is like when you have to go back in. One thing to doctor Deysine: the laparoscopic mesh repair is far easier than an open preperitoneal repair. Therefore I am a great advocate: if you are an open preperitoneal surgeon, quite honestly for the sake of the patient, it is better to do it laparoscopically.*

Concluding Remarks

Basic Mistakes of Suture Repair (Bendavid, CAN). All I have to say is that:

1. It does help a great deal when you know the anatomy.
2. Be able to assist the kind of tissues that you are handling and therefore think of using mesh.
3. Most important: do not hesitate to refer to someone who may be doing a different technique; in other words you should adapt your techniques to the patient and not vice versa.

Basic Mistakes of Mesh Repair (Kingsnorth, GB). The list I have drawn up applies the same: I thought with anatomy that one must prepare oneself before entering the operating room; case selection is also important with the particular techniques that are available. When you get into the operating room you are faced with a lot of technical details. It is very important that if we are using a particular mesh technique we must absolutely adhere to what the originators of the technique have shown and not make our own little modification that we think is going to improve it because our trainee will also make his own little modification and then it gets completely corrupted. Also I think: know your results. A lot of surgeons don't know what their results are and if surgeons are getting bad results, they need to go back and think about what they are doing wrong. Finally, if in doubt about a difficult case, refer to an expert.

Treatment of Recurrent Inguinal Hernia

27 Recurrence and Infection: Correlation and Measures to Decrease the Incidence of Both

M. Deysine

Introduction

Since the early 1900s herniorrhaphists or general surgeons writing on the subject have insisted on the "radical cure" of hernia. However, they were mostly referring to recurrences, which were considered the only significant problem at that time. The subsequent evolution of our science demonstrated that recurrences can be lowered by implementing a variety of technical measures, which involved proper groin dissection as popularized by the Shouldice clinic, followed by repairing the defect with various adjunct measures, which so far have proven effective in the hands of dedicated surgeons. This has lowered the recurrence rate in many series to below 1% [1]. However, the 2003 Suvretta Conference revealed that the recurrence rate for most European countries still ranged from 7 to 12%, demonstrating that, leaving aside those dedicated to the field, there is a significant number of surgeons who are responsible for a large failure rate [2]. Unfortunately, reliable statistical data from the USA are unobtainable due to a shift in medical care management by third-party payers (◘ Table 27.1).

The discrepancy between these two groups concerning the recurrence rate can also be observed in the field of post-herniorrhaphy wound infection. In the hands of experienced surgeons, the infection rate for inguinal herniorrhaphy should be below 1%. However, reports are still published with infection rates around 7%. These very high figures, apparently published without being challenged by editorial boards, give the impression that those parameters should be accepted by the readership [3] (see ◘ Table 27.2).

Meanwhile, the real incidence of post-inguinal and ventral herniorrhaphy infections remains an elusive figure particularly because the impact of these complications has not been accurately projected. Reports of small and large series either fail to mention the subject or cavalierly describe incidences of 7% as an unavoidable mishap. The projection of such incidence would signify that the 2,000,000 yearly inguinal herniorrhaphies performed between Europe and the USA would be followed by 140,000 infections [3] (see ◘ Table 27.3).

◘ **Table 27.1.** Projected recurrence rates of inguinal herniorrhaphy by those who publish their results and those who do not

Primary IH [n]	10% RR (not publishers)	1% RR (by publishers)
2,000,000	200,000	20,000
Second operation	RR 25% = 50,000	RR 5% =1000
Third operation	RR 25% = 12,500	RR 5% = 50
	12,000 vs. 50	

Table 27.2. Projected infection rates of inguinal herniorrhaphy by surgeons who publish their results and those who do not

No. of primary IH	4% IR	1% IR
2,000,000	80,000	20,000
Second operation	20,000	200
	20,000 vs. 200	

Table 27.3. Reported recurrences and infection rates of ventral herniorrhaphy

Author	Year	No.	RR	Infection
Girotto	2003	284	20%	28%
Bower	2004	100	2%	2%
White	1998	250		14%
Rosen	2003	96	17%	4%
Constanza	1998	31		6%
Gorge	1986	81		25%
Terzi	2005	395		2% (E. coli)
Finan	2005	3661		5%
Hesselink	1993	417		7%

Is There a Correlation Between Infection and Recurrence?

This is a very pertinent question because the location of the infective process will dictate the outcome and seriousness of the possible recurrence. The correlation between wound infection and recurrence is not clear as most publications do not specify their incidence nor do they detail the exact location of the infection [4].

Dealing with inguinal herniorrhaphy, if the infection involves only the subcutaneous tissues above the external oblique aponeurosis, then the treatment consists of wound opening and debridment plus the administration of appropriate antibiotics. These infections seldom involve the repair itself and should not be followed by a recurrence. However, delay in recognition and treatment may permit the downward invasion of the microbes with a subsequent mesh infection.

If the infection is located deep to the external oblique aponeurosis, it will involve the repair itself. If the repair was done anatomically as in the Shouldice procedure, the removal of the sutures, debridement of the wound and antibiotics may take care of the problem. On the other hand, if it was a mesh repair, the treatment requires a long period of local wound care taken to allow the granulation tissue to penetrate the mesh interstices. This evolution may take weeks or months eventually requiring mesh removal. PTFE meshes should be removed as soon as there are found to be infected. Evidently, both kinds of infections require a different treatment and harbor a different prognosis.

In any event, an infection will be onerous to both the welfare of the patients and the medical system as it requires multiple procedures, several anaesthetics, and may end up with a recurrence taxing both the patient and the surgeon [5–8].

The literature on ventral hernia repair is clearer when dealing with infections and the rates range from 2 to 28%. In this regard, wound size, the extent of the dissection, mesh area, and particularly operating time are all significant factors affecting the incidence of infection.

As with inguinal hernias, ventral superficial subcutaneous infections usually respond well to wide debridment and drainage, irrigations, frequent dressing changes and judicious antibiotic utilization. Deep infections involving the prosthesis are serious complications demanding massive wound and prosthetic debridment plus long-term outpatient wound care until the wound finally heals. Judicious surgical judgment will dictate if portions of mesh invaded by granulation tissue may be allowed to remain in the wound as a permanent repair or to be incorporated by suture to newly inserted prosthesis. While mesh removal is almost always followed by recurrence, mesh re-implantation can only be accomplished when the surgeon is certain of wound sterility. This question can be solved by percutaneous microbiological wound sampling. In some of these patients the final care may take years [8–18].

Is it Possible to Teach Anti-Recurrence and Anti-Infection Measures?

Congresses like this one have successfully dedicated innumerable hours of debate dealing with the subject of recurrence meaning that – in our hands – the

recurrence rate of inguinal herniorrhaphy has fallen significantly.

This has occurred because we have adopted a series of sequential tactical anti-recurrence measures consisting of:

1. Adequate exposure of the anatomical elements in the inguinal region.
2. Evaluation of the hernia defect and its tissue quality.
3. Selection of mesh type and size that would best fit that particular defect and the patient.
4. Selection of a specific technique for mesh anchoring.

The judicious utilization of this knowledge emanating from the work of recognized herniorrhaphists has produced very good results in inguinal and ventral herniorrhaphy. I call these measures anti-recurrent measures.

Can We Equate Those Results in the Field of Infection?

In the field of surgical infection, starting and following the work of Semmelweis, Pasteur and Lister, surgeons adopted so-called antiseptic or aseptic measures in order to lower the infection rate of "clean surgery", but unfortunately, these very effective measures have not been applied in a consistent manner, particularly in the field of herniorrhaphy. One way to analyze the validity of these principles is to note the evolution of orthopaedic joint replacement, which was plagued by infection rates of about 15% at its inception. The stringent application of well-proven aseptic and antiseptic measures by Charnley's group, including ambient bacterial control by laminar flow ventilators with HEPA filters, strict adherence to antiseptic technique, systemic, and local antibiotics, and particularly the utilization of hoods to cover the heads and necks of the surgeons and assistants, have lowered their infection rate to below 1%. I believe that such levels of excellence could be achieved in the hernia field.

Unfortunately, in the discipline of hernia repair and in spite of the fact that some of our meshes have a larger surface exposed to microbes than orthopaedic metal prosthesis do, those measures are not being implemented yet. Accordingly, we still operate on hernias dressed in the same garb as we did in 1920, showing a significant lack of sophistication and a reluctance to accept microbiological truisms.

On the other hand, the field of microbiology has broadened its base going from the study of the bacteria itself to the study of their biofilm. This biological bacterially produced glue permits bacterial adhesion, colonization and wound invasion. Across such biological glue, bacteria communicate with each other about food location and better colonization sites. They seem to be more intelligent than we thought because this sophisticated biological condition permits their survival and further procreation. Its control or elimination may be a clue to future infection control

A recent congress held in Seattle, Washington, USA, in which this subject was discussed, led to the audacious forecast of a future variety of measures that will deal with infections control concentrating all efforts on the inhibition of biofilm production by biochemical means. This suggests the possibility that antibiotics may not be the future answer to infections [20].

Can the Road to Recurrence Control Meet the One About Infections?

Regardless of their correlation with recurrence, infections tax the confidence and credibility between surgeon and patient forcing multiple procedures that strain both. In order to lower our infection rate, we need to adopt more stringent anti-infection measures which have proven to be as successful as those utilized for the reduction of recurrences [19].

It is evident that those who are interested in the field of hernia have fewer complications than general surgeons performing these operations. This, in turn, suggests that those responsible for recurrences and infections are not mindful of the advances made on infection and recurrence control actually applied by dedicated surgeons. In other words, that their training in those areas may be incomplete or at least outdated.

One of the ways to bridge the educational gap between those dedicated to hernia and those who operate on hernia as part and parcel of a broad general surgical practice would be to re-evaluate our training system.

Table 27.2 depicts in a simplistic way the usual hernia-related training pattern of surgical residents. From it, it is obvious that their only real contact with the subject is during their first year, when they dedicate most of their operating time to acquiring the handicraft of instrument utilization and the necessary eye–hand coordination. They have little opportunity to understand the underlying anatomy or the repair technique. Their second, third and fourth year are spent assisting or performing other "larger surgeries" and they may perform a few difficult hernias during their senior year.

Later, in practice, and being the youngest of a large cohort of general surgeons, they are referred inguinal hernias. It is then that they start comprehending the mechanics of a good repair but only after a learning curve of different dimensions. This is probably the reason behind the published high recurrence rate.

How Can We Better Train Our Surgeons?

Evidently, we need a different type of education for trainees who will end up operating on hernias, and I suggest that following ◘ Table 27.3, we put together a program to be inserted within resident training of general surgeons. It would be beneficial if they could spend a certain amount of time in a service that dedicates itself to hernia repair or with individuals who do that too. Ideally, this should involve second-year trainees because the first-year trainees are too involved in his eye–hand coordination process and during that time they may not be able to understand the complications and intricacies of the repair itself. The program should include lessons in anatomy, up to date techniques, polymer availability, anti-infection and anti-recurrence control, postoperative care, and follow-up. The trainee would first assist surgeons with a recognized competence in the field and may at the end of the period repair hernias under supervision. This should be followed by an oral and practical examination after which the resident can continue his training and graduate as a general surgeon.

The necessity to turn our discipline into a specialty is still uncertain; however, the actual fragmentation of general surgery has been successfully producing fields in which individuals can better dedicate themselves and concentrate their efforts in one entity for the benefit of the patients.

In summary, if there is no direct evidence that an infection brings recurrence, there is evidence that infections require re-operations and they are a source of human discomfort, pain and even death. In order to lower both recurrence and infection rates we need to re-focus our attention on the training of our residents in the field of hernia.

References

1. Bendavid R (2001) The Shouldice repair. In: Bendavid R, Abrahamson J, Arregui M, Flament JB, Phillips EH (eds) Abdominal wall hernias. Principles and management. Springer, Berlin Heidelberg New York, pp 370–375
2. Schumpelick V, Nyhus LM (2004) Meshes: benefits and risks. Springer, Berlin Heidelberg New York
3. Cingi A, Manukyan M, Gulluoglu B, Barlas A, Yegen C, Yalin R, Yilmaz N, Aktan AO (2005) Use of resterilized Polypropylene mesh in inguinal hernia repair: A prospective, randomized study. J Am Coll Surg 201: 834–840
4. Sanchez I, Sanchez J, Deysine M (2004) Incidence and epidemiology of infection after external abdominal wall herniorrhaphy. In: Deysine M (ed) Hernia infections; pathophysiology, diagnosis, treatment and prevention. Marcel Dekker, New York, p 17–57
5. Deysine M (1991) Inguinal herniorrhaphy. Reduced morbidity by service standardization. Arch Surg 126: 628–630
6. Deysine M (1998) Pathophysiology, prevention and management of prosthetic infections in hernia surgery. Surg Clin N Am 78: 1105–1115
7. Deysine M (2001) Hernia clinic in a teaching institution: creation and development. Hernia 5: 65–69
8. Deysine M (2006) Infection control in a hernia clinic: 24 year results of aseptic and antiseptic measure implementation in 4,620 "clean cases". Hernia 10: 25–29
9. Jones NW (1998) Laparoscopic re-de repairs of recurrent inguinal hernias using double-mesh technique. J Soc Laparoendosc Surg 2: 175–176
10. Constanza MJ, Heniford BT, Arca MJ, Mayes JT, Gagner M (1998) Laparoscopic repair of recurrent ventral hernia. Am Surg 64: 1121–1125
11. Bower CE, Reade CC, Kirby LW, Roth JS (2004) Complications of laparoscopic incisional – ventral hernia repair: the experience of a single institution. Surg Endosc 18: 672–675
12. Rosen M, Brody F, Ponsky J, Walsh RM, Rosenblatt S, Duperier F, Fanning A, Siperstein A (2003) Recurrence after laparoscopic ventral hernia repair. 17: 123–128.
13. Le Blanc KA (2004) Laparoscopic incisional ventral hernia repair: Complications-how to avoid and handle. Hernia 8: 323–331
14. Terzi C, Kilic D, Hosgorler F, Fuzun M, Ergor G (2005) Single – dose oral ciproflaxin compared with single – dose intravenous cefazolin for prophylaxis in inguinal hernia repair: a controlled randomized clinical study. J Hosp Infect 60: 340–347
15. Finan KR, Vick CC, Kiefe CI, Neumayer L, Hawn MT (2005) Predictors of wound infection in ventral hernia repair. Am J Surg 190: 676–681
16. George CD, Ellis H (1986) The results of incisional hernia repair: a twelve year review. Ann R Coll Surg Engl 68: 185–187
17. Hesselink VJ, Luijendijk RW, de Wilt JH, Heide R, Jeekel J (1993) An evaluation of risk factors in incisional hernia recurrence. Surg Gynecol Obstet 176: 228–234
18. White TJ, Santos MC, Thompson JS (1998) Factors affecting wound complications in repair of ventral hernias. Am Surg 64: 276–280
19. Deysine M (2004) The catastrophe: Mesh infection and migration with fistula formation – Life long risk? In: Schumpelick V, Nyhus LM (eds) Meshes: benefits and risks. Springer, Berlin Heidelberg New York, pp 207–227
20. Bugs and biomaterial: bacterial and the biointerface. University of Washington Summer Symposium. Seattle, Washington. August 24–26, 2005

Discussion

Conze: *Let me speak for the young surgeon who has just finished his studies. I absolutely agree with you that he needs close and intensive teaching of the anatomy and of the preparation that has to be done. But if he is doing a few hernias in his second and third and fourth year, that will not really help him. I think the repetition of the procedure is what he needs. So let him do ten a week or five a day; that will help him to keep the technique in mind, so he will remember later on in practice. If he does one a month – forget it, it is waste of time.*

Read: *You said all the right things. As a teacher of surgeons for 40 years I think the problem is not what the particular surgeon teaches. The problem is the evolution of the politics and the establishment. Herniology as a subspecialty, as we know, has become very successful. We can be proud and we have done it very fast. But we have to catch up with the establishment and the politics because general surgery is trenched and all of this takes time. I think you have the right thing but you have to be patient because we as herniologists have done very well.*

Deysine: *I am afraid that unless we take charge, other people who are not as much interested as we are will take charge. So I believe in politics.*

Schumpelick: *I want to make some comments. First, I miss a something about mesh and infection. How should we handle it? The second thing is: is a hernia simulator available in the American hernia society? I know that you have some very good devices training surgeons on simulators as a pilot? Is there anything on the way?*

Deysine: *Your first question is: how to handle an infected mesh? Carefully! If it's polypropylene and it is superficial, it can be treated locally. If the mesh belongs into a large ventral hernia it's a year and a half of work! Some people do it faster by excising part of the mesh and suturing the remaining together. If it is PTFE you had better remove it as soon as it becomes infected because it will not heal. For superficial infections in general, if the surgeon is well trained in treating infections: incision and draining as soon as you think about it, and then, of course, antibiotics. I prefer prevention and I think we should operate our patient with the same precaution as the orthopaedic surgeons use to insert a new hip, we should use three pairs of gloves, hepafilters; they have reduced their infection rates below 1%. I think we should follow them.*

The simulators? I don't know anything about it, but I think it's a good idea. This is pure anatomy. Recurrences are anatomy, and anatomy is not mysterious.

Bendavid: *One short comment in terms of teaching. If you want to succeed perhaps we should emulate Billroth and talk a little more about our failures.*

Deysine: *Actually, our conference is a mixture of that. It's very good advice. But I see that a group of surgeons who do not go to the conference or read our journals are not getting up to date with technique. Every one of my first-year residents that assists me on hernias, when I asked him "are you familiar with the Shouldice repair" said "no". So they come to surgery without reading before. That can be arranged; it is not difficult.*

Chowbey: *It has been documented that laparoscopic repair of hernia dramatically reduces the infection rate. Would you comment on that?*

Deysine: *Yes.*

Chowbey: *Should we not move on to do more and more laparoscopic repairs to reduce the infection?*

Deysine: *No*

Bendavid: *You mustn't forget that with laparoscopic surgery you're dealing with a very wide area – peritoneal-extraperitoneal. You have got 2 m2 of peritoneal surface which is extremely bacteriostatic as compared to a small wound that is connected with the outside. This is probably the explanation of the lesser number of infections.*

Deysine: *Dr. Wanz once got up and got very, very red in the face and was very angry. "From this podium I can go to downtown and then to my hotel room or to my room directly – the difference between both procedures is enormous". But those who do them well – good luck.*

28 Inguinal Hernia Recurrence and Pain

L. Chung, P.J. O'Dwyer

Introduction

Recurrent hernias account for between 10 and 20% of all inguinal hernias repaired. When addressing the issue of recurrence and pain there are at least three questions that come to mind:

1. Are recurrent hernias more painful than primary inguinal hernias?
2. Is an increase in post-operative pain after primary inguinal hernia repair associated with recurrence?
3. Are recurrent inguinal hernias more likely to be associated with chronic pain after repair?

We have reviewed the literature pertaining to these topics and in addition obtained data from our own patient population in an effort to provide some answers to these questions.

Results

Are Recurrent Hernias More Painful Than Primary Inguinal Hernias?

The visual analogue pain scale (VAS) is the most reliable and sensitive tool for measuring pain (Fig. 28.1). These scales should not be corrupted by placing additional points on the 100-mm scale, for example, marking where mild, moderate or severe pain is thought to be represented. Patients tend to cluster their responses around such markings and reduce the reliability of the test [1].

In a prospective study we obtained pre-operative VAS scores on 323 patients undergoing primary inguinal hernia repair [2]. The mean pain score was 7 mm at rest and 17 mm on moving in a consecutive series of unselected patients. A quarter of the patients had no pain whatsoever at rest, whilst one-sixth had no pain on moving from their inguinal hernia (Table 28.1).

Table 28.1. Severity of pain in 323 consecutive patients with a primary inguinal hernia

Visual Analogue Scale (VAS) [mm]	Severity of pain	No. of patients (%)	
		At rest	On movement
0	No pain	86 (26.6)	53 (16.4)
< 10	Mild	174 (53.9)	137 (42.4)
10–50	Moderate	58 (18.0)	100 (40.0)
> 50	Severe	5 (1.5)	33 (10.2)

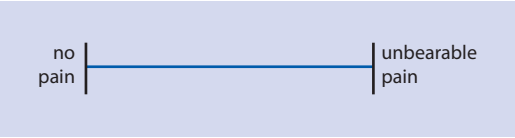


Fig. 28.1. 100-mm visual analogue scale (VAS)

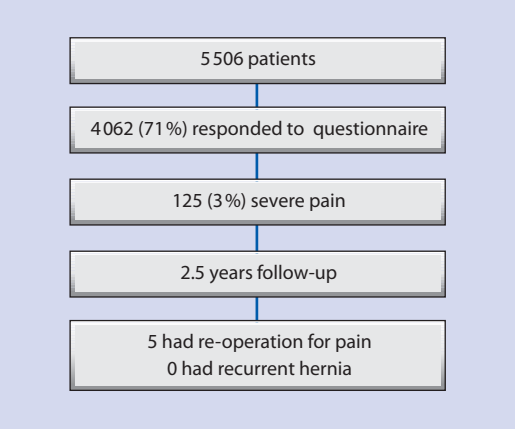


Fig. 28.2. Outcome of patients with severe pain from the Scottish Audit of Hernia Repair

Although we have no equivalent data for recurrent hernias, we can conclude from clinical trials with long-term follow-up that they are likely to cause pain similar to a primary hernia. In the study by Beets et al. looking at the 12- and 15-year follow-up of various sutured repairs in 324 patients, there were 83 recurrences, 35 (42%) of which were completely asymptomatic [3]. Also we know that few patients who go on to develop severe chronic pain after repair of a primary inguinal hernia actually have a recurrent hernia. When 125 patients with severe chronic pain from the Scottish Audit of Hernia Repair were followed for 2½ years, none had represented in that period with a recurrent hernia (Fig. 28.2) [4].

Is an Increase in Post-Operative Pain After Primary Inguinal Hernia Repair Associated with Recurrence?

There is some evidence from the Swedish Multicentre Study (SMIL) that patients undergoing a Shouldice repair who have severe postoperative pain are significantly more likely to develop a recurrence (Table 28.2) [5]. Interestingly, in patients who underwent laparoscopic repair, no such association was evident. This may indicate that excess postoperative tension in sutured repairs is associated with an increase in recurrence rate.

Are Recurrent Inguinal Hernias More Likely to Be Associated with Chronic Pain After Repair?

There is no evidence in the literature that patients who undergo an operation for a recurrent hernia are more likely to suffer chronic pain. In the Danish Nationwide Questionnaire Study, 34% of patients reported pain following repair of a recurrent hernia compared to 28% following repair for a primary hernia [6]. This difference was not significant. The respective figures for functional impairment secondary to pain were 13.7% and 10.9%. Similar results are obtained when only severe chronic pain has been assessed. In a 5-year follow-up of primary and recurrent hernias treated under our care, although a trend for increased severe pain in patients undergoing repair of a recurrent hernia was evident, this difference was not significant (Table 28.3).

Table 28.2. Association between recurrent hernia rates and postoperative pain in patients undergoing Shouldice repair of primary inguinal hernia

Combined VAS score in first post-op week	Numbers with recurrence	Numbers without recurrence	P value
>200 mm	18	143	0.006
≤200 mm	9	251	

Table 28.3. Severe chronic pain[a] at a 5-year follow-up for primary and recurrent inguinal hernias

	Primary hernia (n = 300)	Recurrent hernia (n = 99)
Recurrence	6 (2%)	0 (0%)
Chronic pain	5 (1.6%)	6 (6%)

[a]Severe chronic pain was defined as pain requiring referral to a pain clinic.

Conclusions

Recurrent inguinal hernias are often asymptomatic but will usually cause some mild to moderate discomfort like their primary counterpart. An increase in postoperative pain after open sutured repair may be associated with a higher rate of recurrence.

However, further studies are required in this area. Finally, although severe chronic pain is probably more common after repair of a recurrent hernia, the difference is small and should be discussed pre-operatively with the patient when consenting for operation.

References

1. Ong KS, Seymour RA. Pain measurement in humans. Surg J R Coll Surg Edinb Irel. 2004; 2: 15–27
2. Page B, Paterson C, Young D, O'Dwyer PJ. Pain from primary inguinal hernia and the effect of repair on pain. Br J Surg 2002; 89: 1315–1318
3. Beets GL. Oosterhuis KJ. Go PM. Baeten CG. Kootstra G. Long-term follow-up (12–15 years) of a randomized controlled trial comparing Bassini-Stetten, Shouldice, and high ligation with narrowing of the internal ring for primary inguinal hernia repair. J Am Coll Surg. 1997; 185:352–357
4. Hair A, Duffy K, McLean J, Taylor S, Smith H, Walker A, Macintyre IMC, O'Dwyer PJ. Groin hernia repair in Scotland. Br J Surg 2000; 87:1722–1726
5. Arvidsson D, Berndsen FH, Larsson LG, Leijonmarck C-E, Rimbäck G, Rudberg C, Smedberg S, Spangen L, Montgomery A. Randomized clinical trial comparing 5-year recurrence rate after laparoscopic versus Shouldice repair of primary inguinal hernia. Br J Surg 2005; 92:1085–1091
6. Bay-Nielsen M, Perkins FM, Kehlet H. Pain and functional impairment 1 year after inguinal herniorrhaphy: a nationwide questionnaire study. Ann Surg 2001; 233: 1–7

Discussion

O'Dwyer: *I think there's an incredibly wide variation in what people regard as chronic pain. I suspect one has to take the definition from pain after the wound has healed. Whether it is a good definition I really don't know. Most people think that is 3 months. But I think probably a year is better than 3 months because some people will still have persistent pain because of the persistence of a haematoma or a complication.*

Kingsnorth: *I just think its better a 6- to 3-months period because it is internationally recognized amongst anaesthesiologists. Do you think it is a recommendation we should stick to?*

O'Dwyer: *I think as long as we stick to that we are all regarding the same thing.*

Amid: *I just want to confirm exactly what you said. I have performed about 350 operations because of pain and these are patients that have been referred by pain clinic centres. There is no correlation between pain and recurrence. The incidence of pain after open versus laparoscopic repair is the same. There is no correlation between the pain and a mesh, and the result of surgery is much more successful after anterior repair.*

Kurzer: *Looking at long-term postoperative pain as a significant outcome at least as important as recurrence that is going to have an effect on how we deal with hernia with respect to the recent watchful trial results. We are going to have reconsider who we operate on.*

O'Dwyer: *I have not read Bobs trial yet, but certainly in our trial on symptomatic patients, there was no increase in pain in those we operated on but there was a slight decrease.*

Schumpelick: *If recurrence is not associated with pain, how does the patient notice that he has a recurrence?*

O'Dwyer: *He notices a lump in the groin. If there is no lump there is probably no hernia.*

29 Recurrence and Mesh Material

F. Köckerling, C. Schug-Pass

Introduction

A meta-analysis of the EU Hernia Trialist Collaboration of all available prospective randomized trials could prove a significant lower recurrence rates for techniques with the use of mesh after primary repair of groin hernias (Table 29.1) [1, 2]. There was no statistical difference between the open and the endoscopic techniques in the recurrence rate after primary repair when a mesh was used. In the nationwide Danish hernia database as large prospective observational study of 26,304 herniorrhaphies the re-operation rates 30 months after anterior mesh repair and laparoscopic repair were significantly lower than after sutured posterior wall repairs in primary inguinal hernia (2.2 and 2.6% vs. 4.4%; $p < 0.0001$). Re-operation rates were also lower with anterior mesh repair (6.1%; $p < 0.0001$) and laparoscopic repair (3.4%) after recurrent hernia (Table 29.2) [3].

Table 29.1. Incidence of recurrences after primary repair [2]

Open non-mesh	4.4%	–
Open mesh	1.4%	$p < 0.001$
Endoscopic mesh	2.1%	$p = 0.026$

Table 29.2. Re-operation rates after repair of recurrent hernia [3]

Sutured posterior wall repair	10.6%	–
Anterior mesh repair	6.1%	$p < 0.0001$
Endoscopic repair	3.4%	$p < 0.0001$

Mesh and Recurrences

Recurrence after inguinal herniorrhaphy continues to be a problem, although the mesh techniques are associated with reduced recurrence rates [4]. Operative findings in recurrent hernia after a Lichtenstein procedure showed direct recurrences in 62%, whereas the remaining recurrences were either indirect (17%), femoral (13%) or unclassified (8%) [4]. The main reasons for recurrences after primary mesh repair are technical faults during the operation and shrinkage of the mesh (see list below) [5].

A typical technical fault of the endoscopic repair of primary hernias is the use of too small meshes. The Neumayer study has shown the significant influence of the mesh size on the recurrence rate, although the differences between the mesh sizes were not great (Table 29.3) [6, 7].

Table 29.3. Size of the mesh used for endoscopic repair of a primary hernia [6]

	Recurrence	No recurrence	p
Mean vertical dimension [cm]	8.1 ± 0.6	8.5 ± 1.3	< 0.001

Reasons for recurrence after primary mesh repair [4]:
- The recurrent hernia develops in 99% of all cases at the free edges of the mesh
- Hernias in the area of the mesh seems to be a rare exception
- Technical faults during the operation (mesh size, fixation, etc.)
- Shrinkage of the mesh
- Alteration of the extracellular matrix

Mesh Shrinkage

Considering mesh size as an important factor in the avoidance of recurrences shrinkage of meshes also has a substantial influence on the concrete choice of a mesh. Klinge et al. (1998) could show that the amount of polypropylene influences the extent of inflammatory response and the corresponding consecutive fibrosis. Shrinkage might be responsible for secondary folding in cases of poor elasticity and small pores [8]. Meshes that contain a lot of polypropylene shrink to about 30–50% of their original size after 4 weeks (Table 29.4). Reduction in the polypropylene content decreases both the inflammatory response and the shrinkage. Meshes with lower content of polypropylene and larger pores are less likely to fold and improve compatibility [8].

Table 29.4. Mesh shrinkage. The amount of polypropylene influences the extent of the inflammatory response and the corresponding consecutive fibrosis. Shrinking might be responsible for secondary folding in cases of poor elasticity and small pores [8]

Monofilament, polypropylene, 95 g/m²	46%
Multifilament, polypropylene, 55 g/m²	34%

Light-Weight Polypropylene Meshes – Experimental Data

The development of light-weight monofilament, large-pore polypropylene meshes has led to an appreciable improvement in biocompatibility. This has been shown in a number of experimental studies [8–14].

Using an experimental model with domestic pigs (containing 11 pigs in each group) with the endoscopic total extraperitoneal approach different meshes (Atrium, Vypro II, Parietene, TiMesh extralight and Ultrapro) were placed in the preperitoneal space. After 3 months the animals were sacrificed and a diagnostic postmortem laparoscopy performed. Specimens comprising both mesh and tissue were then removed, and the dimensions of the meshes measured in the fresh, tension-free specimens and recorded. Tissue samples were studied histologically, including immunohistochemistry and electronmicroscopy.

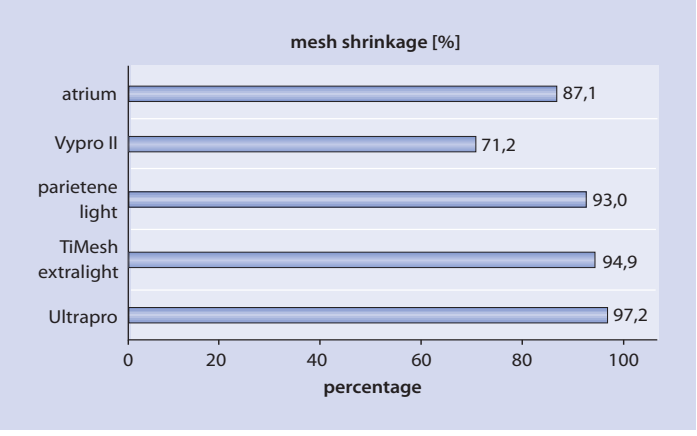


Fig. 29.1. Comparison of mesh shrinkage (% of original size).

Table 29.5. Histological properties of various polypropylene meshes used for endoscopic inguinal hernial repair – impact on their shrinkage rates and inflammatory tissue reaction, cellular reaction

	Atrium	Vypro II	Parietene light	TiMesh extralight	Ultrapro
Mesh surface on explantation [%]	87,1 ± 7,3	71,2 ± 21,6	93,0 ± 12,0	94,9 ± 7,4	97,2 ± 4,9
Partial volume of inflammatory cells [%]	21,0 ± 8,2	34,1 ± 13,6	29,0 ± 19,8	13,1 ± 5,3	15,8 ± 7,9
KI67 (proliferation) [%]	17,3 ± 9,4	25,8 ± 10,7	19,2 ± 11,9	12,3 ± 4,3	5,8 ± 4,5
Apoptotic index (cell death)	7,6 ± 4,0	10,3 ± 5,4	9,5 ± 4,8	8,7 ± 2,8	2,1 ± 2,1
CD 11A (granulocytes, macrophages, monocytes, lymphocytes)	12,2 ± 5,8	18,0 ± 6,9	11,7 ± 3,0	1,1 ± 0,7	7,4 ± 7,2
CD 68 (macrophages/ monocytes)	7,9 ± 3,7	9,1 ± 6,0	5,1 ± 2,3	0,2 ± 0,4	5,1 ± 3,4

Clear differences were found in the shrinkage characteristics of the meshes. In the first experiment Vypro II mesh shrinkage (28%) was significantly greater in comparison with Atrium (12%), Parietene (7%) and TiMesh extralight (5%). The lowest chronic inflammatory reaction was seen with TiMesh extralight (13% PV of inflammatory cells) (Table 29.5). With regard to cell proliferation, Parietene and TiMesh appeared to have slight advances. No differences were observed in the apoptotic rate. In this experiment the TiMesh, as a light-weight and titanium-coated mesh, proved to be significantly superior to all the other implanted meshes. Vypro II as a multifilament mesh with a large surface and partially absorbable mesh (polyglactic acid) induced a pronounced inflammatory and fibrotic reaction, which exceeds that of heavy-weight monofilament polypropylene meshes [9, 15].

In a further study, Ultrapro, another light-weight polypropylene mesh (technical data in comparison to TiMesh extralight, Table 29.6) showed an overall mean reduction of the surface area of the mesh of 2.8%, which was the least shrinkage rate of all meshes (see Table 29.5, Fig. 29.1), even in comparison to TiMesh extralight (surface area 97.2% vs. 94.9%), but

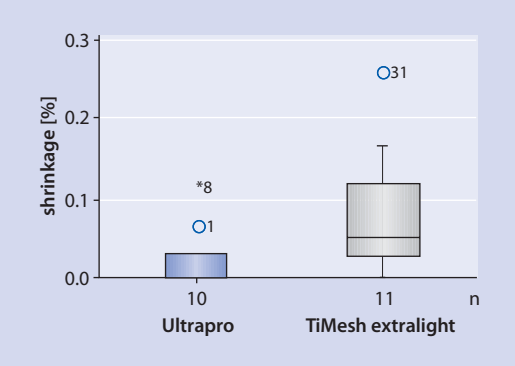


Fig. 29.2. Comparison of mesh shrinkage (% of original size) – lightweight meshes, p = 0.111

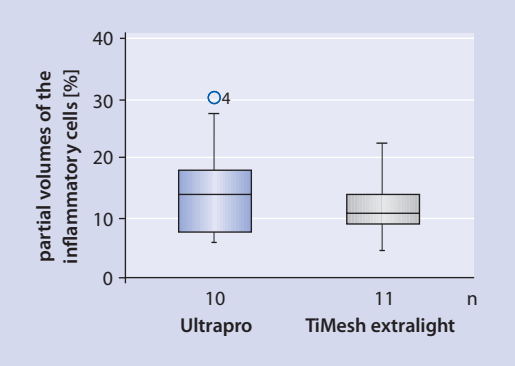


Fig. 29.3. Comparison of the partial volumes (%) of the inflammatory cells – lightweight meshes, p = 0.376

Table 29.7. Technical data of light-weight meshes

	Ultrapro	TiMesh extralight
Material	PP + Poliglecaprone (absorption within 90–120 days)	PP + titanium coating
Weight	28 g/m^2	16 g/m^2
Pore size	~3–4 mm	~ 1.0 mm
Filament thickness	0.09 mm	0.065 mm
Mesh thickness	0.5 mm	0.2 mm
Elasticity (max. abd. pressure)	17.5% 16 N/cm	
Max. tensile strength (manufacturer's data)	650 mmHg (~ 69 N/cm)	> 8.5 N/cm

without significance (p = 0.111) (Fig. 29.2). The partial volumes of inflammatory cells measured for TiMesh extralight showed only slightly lower, but not significant figures in comparison with Ultrapro (13.1% vs. 15.8%, p = 0.374) (Fig. 29.3). By the addition of the Monocryl supplementation (resorbable), in contrast to TiMesh extralight a large proportion of the inflammatory cells comprised macrophages or monocytes.

Conclusion

The development of light-weight monofilament, large-pore polypropylene meshes has led to an appreciable improvement in biocompatibility. As a result of its good biocompatibility and elastic properties, the light-weight large-pore meshes as TiMesh extralight and Ultrapro showed a very low foreign-body reaction and therefore only a very slight tendency to "shrink". This renders these meshes extremely well suited for clinical utilization in hernia repair surgery.

In the daily clinical routine situation, this is usually underscored by a reduction in the foreign-body sensation, and the less frequent development of seromas, which are also more minor. Reduced rates of chronic pain have also been reported [16, 17]. It is possible that the elastic properties of meshes also play a role, but this has yet to be confirmed clinically over the long term. As a result of the only slight tendency of these meshes to shrink, the risk of a recurrence following an endoscopic hernial repair is likely to be small, provided that the mesh was initially appropriately dimensioned and accurately placed.

References

1. EU Hernia Trialist Collaboration (2000) Laparoscopic compared with open methods of groin hernia repair: systematic review of randomized controlled trials. Br J Surg 87: 860–867
2. EU Hernia Trialist Collaboration (2000) Mesh compared with non-mesh methods of open groin hernia repair: systematic review of randomized controlled trials. Br J Surg 87: 854–859
3. Bay-Nielsen M, Kehlet H, Strand L, Malmstrom J, Andersen FH, Wara P, Juul P, Callesen T; Danish Hernia Database Collaboration (2001) Quality assessment of 26.304 herniorrhaphies in Denmark: a prospective nationwide study. Lancet 358: 1124–1128
4. Bay-Nielsen M, Nordin P, Nilsson E, Kehlet H; Danish Hernia Database Collaboration (2001) Operative findings in recurrent hernia after a Lichtenstein procedure. Am J Surg 182: 134–136
5. Klosterhalfen B, Klinge U, Rosch, Junge K (2004) Long-term inertness of meshes. In: Schumpelick V, Nyhus LM (eds) Meshes: benefits and risks. Springer, Berlin Heidelberg New York, pp 170–178
6. Neumayer L, Giobbie-Hurder A, Jonasson O, et al. (2004) Open mesh versus laparoscopic mesh repair of inguinal hernia. N Engl J Med 350: 1819–27
7. Neumayer L (2004) Open mesh versus laparoscopic mesh hernia repair. N Engl J Med 350: 1463–1465; comment on: N Engl J Med 350: 1819–1827
8. Klinge U, Klosterhalfen B, Müller M, Öttinger AP, Schumpelick V (1998) Shrinking of polypropylene mesh in vivo. An experimental study in dogs. Eur J Surg 164: 965–969
9. Scheidbach H, Tamme C, Tannapfel A, Lippert H, Köckerling F (2004) In vivo studies comparing the biocompatibility of various polypropylene meshes and their handling properties during endoscopic total extraperitoneal (TEP) patchplasty. Surg Endosc 18: 211–220
10. Cobb WS, Kercher KW, Heniford BT (2005) The argument for lightweight polypropylene mesh in hernia repair. Surg Innov 12: 63–69
11. Greca FH, de Paula JB, Biondo-Simmoes ML, da Costa FD, da Silva AP, Times S, Mansur A (2001) The influence of differing pore sizes on biocompatibility of two polypropylene meshes in the repair of abdominal defects – experimental study in dogs. Hernia 5: 59–64
12. Junge K, Rosch R, Bialasinski L, Klinge U, Klosterhalfen B, Schumpelick V (2003) Persistent extracellular matrix remodelling at the interface to polymers used for hernia repair. Eur Surg Res 35: 497–504

13. Klinge U, Klosterhalfen B, Birkenhauer V, Junge K, Conze J, Schumpelick V (2002) Impact of polymer pore size on interface scar formation in a rat model. J Surg Res 103: 208–214
14. Schumpelick V, Klinge U, Welty G, Klosterhalfen B (2000) [Meshes within the abdominal wall]. Chirurg 70: 876–887
15. Knook MTT, van Rosmalen AC, Yoder BE, Kleinrensink GJ, Snijders CJ, Looman CWN, van Steensel CJ (2001) Optimal mesh size for endoscopic inguinal hernia repair: a study in a porcine model. Surg Endosc 15: 1471–1477
16. O´Dwyer PJ, Kingsnorth AN, Molloy RG, Small PK, Lammers B, Horeyseck G (2005) Randomized clinical trial assessing impact of lightweight or heavyweight mesh on chronic pain after inguinal hernia repair. Br J Surg 92: 166–170
17. Tamme C, Garde N, Klingler A, Hampe C, Wunder R, Köckerling F (2005) Totally extraperitoneal inguinal hernioplasty with titanium-coated lightweight polypropylene mesh: early results. Surg Endosc 19: 1125–1129

Discussion

Miserez: *You said that the light-weight meshes potentially cause less recurrences and a better quality of life because of better biocompatibility. We have seen with the Vypro so far that this is not the case. Could you comment on that?*
Köckerling: *I think the problem with the Vypro concept is that the pore size was so large that, especially in our model where we don't fix the meshes, probably this was a mechanical problem. The shrinkage was not due to the heavy reaction. The reaction was more due to the fact that it was a multifilament.*
Read: *I don't have any scientific comment but I just want to tell you that your presentation was a model. It was a work of art.*
Kingsnorth: *I do appreciate the scientific content of this and its very nice preclinical data. You did begin to tell us a bit about the clinical data but we made mistakes with the early light-weight meshes we used in terms of the technical details, of not fixing the mesh with open repair. So do you have any technical tips for using the meshes for laparoscopic repair or are they equally amenable for use in laparoscopic and open hernia repair?*
Köckerling: *I can tell you from my clinical experience that you can use this mesh in any type of hernia repair. So we use it for endoscopic hernia repair, open hernia repair, especially Lichtenstein, and I also use it for incisional hernia repair where we perform a specific open technique. You have seen the video in Turin. We always use this type of a mesh – it is strong enough, it is excellently biocompatible, the ingrowth is for me more than enough and the surgical handling is excellent.*
Duh: *I also want to congratulate you, it was a wonderful talk. In fact you may have convinced me that we can fight it in San Francisco if we start trying it. I have some question about whether you have studied meshes that have different shapes, which are, for example, three-dimensional. Do they prevent shrinkage?*
Köckerling: *My personal opinion is that I would prefer any type of two-dimensional reconstruction, not a three-dimensional one. Hopefully, I could show you what would happen if you use a three-dimensional material. It ends up in more reaction, more connective tissue. It's more material and this is not necessary. In this manner I would say less is more.*
Kukleta: *I can agree to that exactly. Since we use Ultrapro it seems that it doesn't shrink at all, Vypro II did, and that is a big difference.*

30 Mesh Explantation in the Groin

G.D. Arlt

Introduction

The use of prosthetic mesh in inguinal hernia surgery is a well-accepted practice. Adverse effects of mesh implantation in the long term seem to be seldom and mesh removal is still a rare procedure today. Nevertheless, several case reports have been published about severe complications following mesh repair.

Most of these deal with the phenomenon of mesh migration. Mesh plugs have been found in the scrotum mimicking a third testicle (Dieter 1997; Novak 2005) or dislocated in the preperitoneal space causing severe and disabling pain (Moorman 2004). Flat meshes are more likely to migrate into the bladder (Hume 1996; Bodenbach 2002; Riaz 2004) or into the colon (DeGuzman 1995; Hamy 1997). The patients suffer from persistent infections or recurrent bleeding and there is an urgent indication for the surgical or endoscopic removal of the mesh material.

Besides mesh migration, other adverse effects of mesh placement are infection and chronic groin pain. Mesh removal may be necessary under these conditions (Avtan 1997; Schumpelick 1997; Taylor 1999; Athanasakis 2000) but indication and timing of revisional surgery is debatable. Our own experience of indication, operative technique and results of mesh removal after mesh repair of inguinal hernia are presented here.

Patients and Methods

Patients

From October 1998 to October 2005, 25 patients with severe complaints following an inguinal hernia repair with a mesh were identified for mesh removal at the Surgical Department of the Park-Klinik in Berlin-Weissensee. Several attempts of conservative treatment on an outpatient basis had been done in all of the patients. Most of them had been treated with chronic intake of analgesic drugs. In some cases a psychosomatic therapy had been recommended. Since the TAPP repair is the most popular mesh procedure in Berlin to date, most cases had a TAPP repair in their history. Patients with mild or moderate symptoms responding to a conservative therapy are not included in this series.

All patients underwent a standardized program of examinations, including a careful history with special attention to the characteristics of inguinal pain, clinical examination and ultrasound. Local anaesthesia of the ilioinguinal nerve with 10 ml of Xylocain 1% at the SPA (spina iliaca anterior superior) was performed in all patients except two cases with a mesh infection. The data of the patients, postoperative interval until presentation at the Park-Klinik and type of the last repair are given in ◘ Table 30.1.

Table 30.1. 25 patients identified for mesh removal at the Surg. Dept. in Berlin-Weissensee from 10/1998 to 10/2005 (19 male/6 female, mean age 48 (14–71) years. Interval from index operation 10–46 months

Mesh procedures	Patients [n]
TAPP	18
TEP	2
Lichtenstein	4
Mesh plug	1

Indication for Operation

In patients with an inguinal abscess or sinus due to an infected mesh a revision of the groin through an inguinal incision was done. Removal of the mesh was intended in only two cases of a mesh "swimming" in an abscess. The wound was left open for secondary healing. The hernia was fixed later by a Shouldice repair.

In nine patients with chronic inguinal pain not responding to a blockade of the ilioinguinal nerve with local anaesthesia (ilioinguinalis syndrome) at the SPA, a transinguinal revision was performed. When a small clinical inapparente recurrence was ruled out intraoperatively and the genital nerve could not be visualized, the removal of the mesh was recommended. The defect of the posterior wall was repaired either with a two-layer Shouldice procedure or a Lichtenstein repair using a light-weight mesh.

In 14 patients with severe groin pain not related to the ilioinguinal nerve and a clinical apparent recurrence, a re-operation with removal of the mesh was done. The defect was closed by a Shouldice repair, a Lichtenstein mesh repair or a Rives procedure.

Operative Technique of Mesh Removal and Reconstruction

The inguinal canal is opened through a transinguinal approach. In cases with a Lichtenstein repair, the cremasteric muscle has to be dissected and the spermatic duct and the vessels are armed with a loop. In a typical Lichtenstein repair the cremasteric muscle is preserved, which facilitates the identification and preparation of the cord. The posterior plane of the cremasteric fascia is taken together with the underlying mesh and excised from the internal oblique muscle. Mostly the caudal margin of the mesh cannot be dissected from the basis of the inguinal ligament without destroying this structure. A small strip of the mesh (about 3 mm) could be left in place in these cases. Later, the caudal stitches of the Shouldice repair can be anchored to this mesh-ligament strip. After total or nearly total excision of the muscle mesh component the defect of the posterior wall is closed with a two-layer Shouldice repair of the untouched cranial transversalis fascia.

In patients with a preperitoneal mesh position, the deep epigastric vessels must be divided caudal near the femoral vessels. Then the posterior wall is opened from the lateral side and the incision is completed medially to the pubic bone. The exact position of the mesh must be explored by digital palpation and an edge of the mesh is armed with a sharp clamp. Under light tension laterally and caudally the medial and cranial part of the mesh is excised from the preperitoneal tissue by sharp dissection. The epigastrics are ligated and divided also at the cranial margin of the mesh. At the internal inguinal ring, the dissection of the testicular vessels, the spermatic duct and the femoral vessels from the mesh is the most challenging part of the procedure. Depending on the individual situation, it might be necessary to leave small strips of the mesh on these structures. Vascular defects occur most likely at the femoral vein. After complete or near-total removal of the mesh, the defect is closed by a two-layer Shouldice procedure. In cases of a large destruction of the transversalis fascia, a Lichtenstein repair or a Rives procedure using a light-weight mesh (Vypro II) is performed.

Peri-Operative Care and Follow-Up

Informed consent was taken from all patients regarding an increased risk of intra- and postoperative complications (estimated percentage of ischemic orchitis about 5%, vascular injury about 5%, and recurrent hernia about 10%).

A single-shot antibiotic prophylaxis was used in all operations. All patients were mobilised within 4–6 h postoperatively. Suction drains were removed between the 2nd and 5th postoperative day. Patients were discharged due to their own wish between day 3 and 10. Physical activity was restricted for 2 weeks after discharge from hospital.

In October 2005 all patients were examined clinically and by ultrasound. The follow-up interval ranged from 12 to 86 months. Due to the small number of patients with different complaints, no statistical analyses were done.

Results

Two patients presented with a late manifestation of a deep infection. The interval after the index operation was 23 and 46 months. In both cases the mesh was found to be "swimming" in an abscess. The mesh could be easily removed and the hernia was repaired by a Shouldice procedure 6 and 9 months later. No signs of a persistent infection were found at follow-up. Both patients were completely satisfied.

In nine cases the groin pain did not respond to local anaesthesia of the ilioinguinal nerve. All these had their meshes removed and got a Shouldice repair. A partial bladder resection was necessary in one case with mesh migration into the bladder. Follow-up examination after 12 to 72 months revealed excellent results in eight patients. In one case a recurrence was found.

Fourteen patients presented with a clinical apparent recurrence and severe inguinal pain not responding to a nerve blockade. The meshes were removed totally or nearly totally. Small strips of the mesh material were left on the spermatic duct in three cases. Suturing of a large defect of the femoral vein was necessary in one case. In all but two patients, the reconstruction of the posterior wall was possible with a two-layer Shouldice procedure. In a 57-year-old female the excision of the mesh-fascia-muscle specimen resulted in a large defect of the posterior wall which had to be closed by a Rives procedure with a Vypro II mesh. In another male the inguinal floor was augmented with a Vypro II-Lichtenstein repair.

Early postoperative complications in these 14 patients were seroma formation and an ischemic orchitis with a consecutive testicular atrophy in each case. At follow-up after 12 to 86 months no recurrence was found. Eight patients were totally satisfied, with no symptoms at all. Mild inguinal pain during physical exercise was reported by five patients. The 58-year-old male with the testicular atrophy had no inguinal pain but was not satisfied with the result (Table 30.2).

Table 30.2. Results of mesh removal after inguinal mesh repair. Data of 25 patients, follow-up 12–86 months

Recurrent hernia	1
No complaints at all	18 (72%)
Mild/moderate pain during physical activity	5 (20%)
Dissatisfied (testicular atrophy)	1

The explanted meshes showed macroscopically the shrinkage of 50 to 70% of the surface, and additional kinking and folding. During histological examination the mesh material was found to be a heavy (> 90 g/m^2) polypropylene mesh in 23 cases and a polyester mesh in two patients.

Conclusion

In the literature there are only two other series of mesh removal after groin hernia surgery. In 1998 Heise and Starling described the transinguinal mesh removal in 20 patients with severe groin pain following Lichtenstein (n = 17) or laparoscopic mesh repair (n = 3). Outcome was excellent or good in 12 (60%) of the 20 cases. Besides a tendency of better results after additional neurectomy during mesh removal, they were not able to identify other factors that could improve postoperative results or help to select better patients for surgery (Heise 1998).

Recently Rosen and coworkers (Rosen 2006) presented their experience with mesh explantation on the basis of ten cases selected from 1998 to 2004. Indication was chronic groin pain not responding to local anaesthesia of the inguinal nerves. The index operation was a Lichtenstein procedure in nine of the ten cases. In contrast to Heise and Starling and our approach they used a combined procedure with transinguinal removal of the mesh and laparoscopic repair of the hernia defect. Results were good or excellent in nine of ten patients.

These data and our results show that transinguinal mesh explantation is feasible and may reveal good long-term results. An important supposition is a careful selection of patients. Indication should be restricted to cases with conservatively intractable symptoms. Favourable indications are persistent infection with abscess formation around the mesh, invasion into a hollow organ (gut/bladder), severe groin pain when other reasons had been ruled out (ilioinguinalis syndrome, hip degeneration, etc.) and recurrent hernia with severe pain which is not related to the recurrence.

The operation is challenging. The surgeon should be experienced in hernia surgery and vascular surgery as well. Informed consent from the patient concerning the increased risk of testicular and vascular complications is mandatory (Arlt 2003).

References

1. Arlt G, Lamm T, Klosterhalfen B (2003) Mesh removal in inguinal hernia repair. Eur Surg 35:42–44
2. Athanasakis E, Saridaki Z, Kafetzakis A et al. (2000) Surgical repair of inguinal hernia: tension free technique with prosthetic materials (Gore-Tex Mycro Mesh expanded polytetrafluoroethylene). Am Surg 66:728–731
3. Avtan L, Avci C, Bulut T, Fourtanier G (1997) Mesh infections after laparoscopic inguinal hernia repair. Surg Laparosc Endosc 7:192–195
4. Bodenbach M, Bschleipfer T, Stoschek M, et al. (2002) Intravesical migration of a polypropylene mesh implant 3 years after laparoscopic transperitoneal hernioplasty. Urologe-A 41:366–368
5. DeGuzman LJ, Nyhus LM, Yared G, Schlesinger PK (1995) Colocutaneous fistula formation following polypropylene mesh placement for repair of a ventral hernia: diagnosis by colonoscopy. Endoscopy 27:459–461
6. Dieter RA (1999) Mesh plug migration into the scrotum: a new complication of hernia repair. Int Surg 84:57–59
7. Heise CP, Starling JR (1998) Mesh inguinodynia: A new clinical syndrome after inguinal herniorraphy? J Am Coll Surg 187:514–518
8. Hume RH, Bour J (1996) Mesh migration following laparoscopic inguinal hernia repair. J Laparoendosc Surg 6:333–335
9. Moorman ML, Price PD (2004) Migrating mesh plug: complication of a well-established hernia repair technique. Am Surg 70:298–299
10. Novak DD, Chin AC, Singer MA, Helton WS (2005) Large scrotal hernia: a complicated case of mesh migration, ascites, and bowel strangulation. Hernia 9:96–99
11. Riaz AA, Ismail M, Barsam A, Bunce CJ (2004) Mesh erosion into the bladder: a late complication of incisional hernia repair. A case report and review of the literature. Hernia 8:158–159
12. Rosen MJ, Novitsky YM, Cobb WS, Kercher KW, Heniford BT (2006) Combined open and laparoscopic approach to chronic pain following open inguinal hernia repair. Hernia 10:20–24
13. Schumpelick V, Arlt G, Schlachetzki A, Klosterhalfen B (1997) Chronischer Leistenschmerz nach transperitonealer Netzimplantation (TAPP) – Kasuistik einer Netzschrumpfung. Chirurg 68:1297–1300
14. Taylor SG, O´Dwyer PJ (1999) Chronic groin sepsis following tension-free inguinal hernioplasty. Br J Surg 86:562–565

Discussion

Simons: *Very interesting talk and a very difficult problem. Do you ever do only triple neurectomy and leave the mesh in place? And another question I would like to attach is: what I sometimes do is I give a block of the nerve with Bubivacain and if it works then my idea is the nerve can be influenced to treat the pain so neurectomy might work. You're saying if the anaesthesia doesn't work you take out the mesh. So I'd like some more clarification on these two questions: Do you do triple neurectomy only without taking out the mesh ever and how do you use the local anaesthesia to your diagnostic work?*

Arlt: *We use local anaesthesia to block the ilioinguinal nerve and the hypogastric nerve and I think it's really impossible to reach the genital branch and the difference between responder and non-responder is responding to the block on the ilioinguinal nerve and the hypogastric nerve. And if we have a responder we do nothing more than a neurectomy.*

Amid: *Thank you very much for the great presentation. I would like to sympathize with you because I have to remove mesh at least once a month. I know how difficult and tedious it is. Just a few suggestions: having a CAT scan before mesh removal gives the surgeon a roadmap on where to go. Particularly if the CAT scan shows that what you want to remove is close to the great vessels for somebody like me who has no experience in vascular surgery or make sure a vascular surgeon in the house is available. Also one suggestion for semantic in order to understand each other: I think it is a good idea to make a distinction between the term shrinkage and wrinkle. It is better to lead shrinkage when the mesh becomes smaller but very uniformly without creating three-dimensionality or sharp edges and then use wrinkle when we have the things that you show because if we use shrinkage for both of them, then people who read or listen to us really don't understand what we're talking about.*

Arlt: *I will follow your advice during the next Suvretta meeting. Thank you. And to your first question:*

Amid: *It was not a question, I only confirmed your comment that it is very difficult to remove this and your approach is the best approach.*

Fitzgibbons: *I don't understand your local anaesthesia test for triple neurectomy because how do you differentiate the physiological effect of local anaesthesia to get rid of the pain versus some type of therapeutic effect. How do you know if you inject local anaesthesia and the patient gets relief how that it is just the physiological effect of blocking the nerves as it posed to be a neuralgia.*

Arlt: *No, I think this is not the target. I want to block the two nerves, not three nerves, the two nerves. And if they respond I have a concrete target. I can leave the mesh. The mesh is responsible for fixing the hernia and I'm not interested in creating another hernia. I'm just interested in pain relief and if this could be done by simple neurectomy of ilioinguinal nerve and hypogastric nerve I will do this. It's much more simple and much quicker. I don't know if I answered your question.*

Fitzgibbons: *First thing I do discover from local anaesthesia is to block those 2 nerves, that's a very effective way to block those nerves but it's only temporary. I just don't understand.*

Arlt: *No, no this test is only to make the diagnosis.*
Young: *I backed up having done vascular surgery before hernia surgery. I do feel comfortable having that knowledge going into doing this. I have one comment or a specific question: they are extremely difficult to do particularly if you are going into remove laparoscopically placed mesh entirely. The question I have is if you have a patient with a recurrence and chronic pain, obviously what we would suggest – and I guess you do – is a neurectomy: when you repair the hernia do you also take that mesh out or do you leave it in?*
Arlt: *I take only those meshes from which I suppose that they are responsible for the chronic pain and if somebody has a recurrence it's one thing, if he has groin pain it's another. There were 14 patients where both things came together and we removed the mesh but to make it quite clear: we didn't remove the mesh because of the recurrence but because of the pain caused by the mesh.*
Ma: *I think this topic is very interesting for me. In China now some very difficult cases come to me especially for the infection. The question is after you removed the mesh how to treat it. Your say give the Shouldice procedure immediately or put in a mesh. That is correct?*
Arlt: *Yes, that's correct.*
Ma: *You don't wait for some time for the second wound healing? But I think if there is infection when you put in a mesh that's indication or contra-indication?*
Arlt: *Yes, you're quite right. So we were happy that in these two cases where we got a swimming mesh in the abscess and a groin sepsis we were able to do a Shouldice repair and it was not necessary to place another mesh in this situation.*
Ma: *According to my experience if you take out the mesh and immediately give a hernia repair it is very difficult so my experience is to wait. But so far I have maybe 12 cases so I know recurrence. I don't know why, maybe skin do no repair the hernia I think.*
Miserez: *If you have a mesh in the preperitoneal space will it sometimes be safer to go through a small midline incision and do a kind of a Stoppa approach to get it out? Secondly, is there a place for a partial remove of the mesh? If you have a hard rim which you can feel which is very painful to the patient, is there a place just to get this hard rim out or not?*
Arlt: *I think it depends on the concrete case and to my experience it's wiser to leave small parts of the mesh in place especially near the vessels near the duct and I think by doing this you can save the testicle and even can save the limb and doing another approach than transinguinal may be a possible way – I don't have any experience in this. I think there has to be a plan before you go to the operation theatre. You have to have a concrete plan. This can be done by CT, it can be done by ultrasound – I'm much more familiar with ultrasound – so for me it's much easier to do such an examination.*
Kehlet: *We published a review of the literature on surgical treatment for chronic pain in the British Journal of Surgery last year and the overall conclusion was that the literature is chaotic regarding pre-operative assessment, characterization of the pain and the patients, intra-operative handling and especially postoperative follow-up are so variable and without any details that it is impossible to make any conclusion on this important topic. I'm really afraid that the number of patients operated for this is increasing. Even Amid, you never saw a patient with chronic pain in a meeting in Sweden on pain 10 years ago or 12 years ago. But now you're saying you're operating more and more and more and you are operating and several other people are operating. I think we need to work together and have exactly the same detailed assessment of the pain and the patient pre-operatively, intra-operatively, diagnostic tests and the follow-up, most important the follow-up because if you talk to a neurologist on this topic with a neurectomy and all the others they will say: You're crazy? What I simply say it's not true? So I really plea for this and I also say that we – Clifford Woolfe and I – have a major review on this topic in the Lancet on chronic postoperative pain and the pathophysiology behind it and please in the future collaborate. We have to do this; otherwise it will just be a disaster in the future. More patients will be operated and they will not be pain-free, I'm sure.*
Arlt: *Thank you. I think this is a very important remark. Since your paper was published in 2005 it was – and I can't rely on this and I can't use this for the patients I have already operated upon – but for the future I think it's good advice to have standards and to collect the patients.*
Kukleta: *I have a question regarding the immediate repair of the hernia. I had the chance to explant several infected plaques and anterior meshes and these patients never had hernia afterwards. Is that because they did not have any before or does it apply to somebody's knowledge that there are less recurrences than expected after explantation even if you don't do anything more than explant?*
Arlt: *I can't answer this question.*
Deysine: *I had several patients who deposited enough collagen after an infection in the area to prevent a recurrence. I even have one that had a large ventral in which we remove Marlex for about a year and than we didn't have to do anything else.*
Ferzli: *A comment on the infection and mesh in the preperitonal space on the person on experience bases. Number one is quite hard to diagnose on clinical ground. Often the diagnosis is made on CAT-scan when the nerve*

fluid level is seen above the bladder and that is how the diagnosis is made. Quite often the approach they do is midline and sometimes the mesh is just floating as someone just described and they remove it.

Schumpelick: *I would like to confirm the comment of Dr. Kehlet. I think the patients come with pain and say: I have a mesh, can you remove it? We had many of these cases in the – I would say 5 years ago. This story is down now and you have seen the figures of Arlt decreasing the risk and I agree with you don't remove a mesh because there is some pain. Many pains will stay after mesh removal. But there are some cases, which we must define, where a mesh removal makes sense, but some do not – don't rely on the pain of patients only.*

31 The Mesh and the Spermatic Cord

R.J. Fitzgibbons Jr.

Introduction

The fibrotic reaction of polypropylene mesh after tension-free (TFR) inguinal herniorrhaphy causing vasal obstruction has been implicated as the reason for infertility in a group of patients presented in a recent paper by Shin and colleagues [1]. The purpose of this presentation is to examine the controversial subject of infertility and inguinal hernia repair and analyze some of the currently available evidence.

Discussion

Can prosthetic material actually cause infertility by virtue of the dense fibroblastic reaction which it is designed to produce? The manuscript referred to above by Shin and colleagues incriminating the polypropylene mesh fibrotic reaction as a cause of infertility would seem to provide evidence of this [1]. Fourteen patients with infertility secondary to obstructive azoospermia (normal sperm in a testicular biopsy yet no sperm in the ejaculate) felt to be related to the fibroplastic involvement of the vas deferens after a heterogeneous group of mesh repairs (conventional, laparoscopic, unilateral, bilateral) are presented. All patients underwent surgical exploration with intra-operative vasography. The vasogram determined the site of the obstruction in the inguinal region and the surgical exploration identified the cause of the obstruction to be the mesh.

But could there be another explanation for these findings? Experienced surgeons who perform re-operative groin explorations after mesh inguinal hernia repairs for reasons other than fertility such as recurrence or postherniorrhaphy groin pain know that the intense fibrotic response described in the manuscript is invariably present. Polypropylene and the other mesh materials used in hernia surgery are supposed to incite a dense fibroplastic tissue response for the purpose of creating a strong mesh-aponeurotic complex to replace weakened native tissue. Eight hundred thousand groin hernia repairs are performed in the US per year, of which approximately 90% are now mesh repairs [2]. Given the fact that inguinal hernias occur at all ages of life and inguinal herniorrhaphies are performed in sizable numbers of patients who are still planning to bear children, why then are we not seeing an epidemic of infertility? Do these 14 patients represent a subset that is exquisitely sensitive to the normal fibroblastic response to mesh? Or was the real cause of the vasal obstruction described in this manuscript the result of a more traditional injury (see list below) followed by scarification to the most convenient structure, which in this case would be the mesh?

Causes of vasal obstruction related to inguinal herniorrhaphy:

- Division
- Ligation
- Clipping
- Stapling

- Electrocauterization
- Devascularization
- Scarification.
- Traction injuries [3]

Infertility caused by inguinal hernia surgery can be related to either the vas deferens or the testicle. The incidence of injury to the vas deferens during inguinal herniorrhaphy has been estimated at 0.3% for adults and between 0.8 to 2.0% for children [4]. Injury to the testicle which eventually leads to atrophy is estimated to occur in about 0.5% for primary hernia repairs but increases tenfold to 5% for recurrent hernia repair [5, 6]. The routine use of prosthetic material for inguinal hernia repair has resulted in a marked decrease in the historical recurrence rate when compared to population-based studies of non-tension-free herniorrhaphies [7]. The irony of this discussion of polypropylene mesh causing infertility is the theoretical effect of decreasing the recurrence rate in the general population from 10–15% seen with Bassini and its variants to less than 5% with the mesh tension-free approach. One should expect a parallel decrease in infertility because of the decreased need for re-operative surgery for recurrence.

We know that the overall incidence of infertility after inguinal herniorrhaphy is higher than the general population. Yavetz et al. looked at 8500 infertile patients and found that 565 or 6.65% gave a history of an inguinal hernia repair [8]. However, this does not shed light on the incidence of the infertility caused by the operation. The issue is clouded by the fact that that many herniorrhaphy patients have no intention of conceiving a child, so fertility status cannot be known; the fertility status of the patient prior to herniorrhaphy is usually not known and the time period between the herniorrhaphy and the diagnosis of infertility introduces the variable of intervening causation. We must look to investigators like Shin and colleagues who conduct specialty infertility clinics to try to extrapolate the incidence. But that literature is dominated by case reports or small series calling into question the quality of the estimates [9]. It is possible that the incidence is so low that the fertility advantages of mesh repair as the result of the avoidance re-operation for recurrence outweighs it.

If one were to assume that polypropylene mesh does indeed cause obstruction of the vas, then one logically must consider the mechanism. Is it caused by an exaggerated fibroblastic response in some patients? If so, why is not the entire structure obliterated? Or does it have only to do with sites where the vas comes in contact with edges of the mesh? It should then occur only at the external and internal rings where the cord rides over these edges. Would the modified Lichtenstein operation in which the tails of the split mesh are simply approximated lateral to the cord at the internal ring put the patient at greater risk than the classic operation in which the inferior surface of the superior tail is sutured to the inferior surface of the inferior tail and the inguinal ligament which creates a shutter valve effect?

Additional Clinical Papers

This is not the first report of abnormality of the vas deferens after mesh inguinal herniorrhaphy. For example, an often-quoted case report by Silich et al. describes a patient who presented 4 years after an inguinal herniorrhaphy with a painful subcutaneous nodule in the repair site [10]. At groin exploration the patient was found to have a spermatic granuloma "imbedded in surrounding fibroareolar tissue and mesh". The authors concluded that cut edges of the mesh where the tails had been wrapped around the cord eroded into the cord, and even provided a diagram depicting this, despite the fact that the original operation was performed at "an ambulatory surgery centre" and no details of that operation were available. One might speculate that an isolated injury to the vas deferens was the more likely explanation, as a spermatic granuloma is by definition an immunological response to extravasated sperm. A direct injury to the vas resulting in a sperm leak might be a more plausible explanation than the gradual erosion by the edge of the mesh. Similarly, a case report published by Seifman et al is often purported to show unequivocal evidence that mesh can cause obstruction of the vas [11]. The 32-year-old patient was diagnosed with secondary infertility (infertility which develops after a successful conception) 1 year after a right inguinal hernia repair with mesh. The patient underwent a groin exploration after he was determined to have obstructive azoospermia on the right based on the absence of viable sperm in a seminal vesical aspirate compared to a right testicle aspirate showing many sperm. An isolated segment of vas was resected that was "incorporated into a scarification process involving the mesh and the vas was totally obstructed" and a reconstruction performed. The patient successfully conceived a child 6 weeks later. It seems pretty clear: the site of blockage was identified precisely, the problem corrected surgically, and the patient was almost immediately able to conceive a child. However, what is commonly omitted when this article is referenced is that the patient also underwent a simultaneous varicocelectomy on the op-

posite side. The authors felt that the short time interval between the varicocelectomy and the conception was too brief to have any effect. It must be left to the reader whether the correction of a known cause of infertility, a varicocele, or a technically challenging reconstruction was responsible for the pregnancy.

There is literature other than case reports useful for the purposes of a discussion concerning infertility that addresses not only vasal obstruction but other potential causes. Aydede and colleagues looked at a group of 60 patients who had undergone TFR herniorrhaphies, 30 of whom were preperitoneal and 30 conventional anterior [12]. The study parameters included spermiograms and testicular perfusion with color Doppler ultrasonography. The spermiograms were identical pre-operatively and postoperatively in both groups. The testicular perfusion studies showed a significant difference between pre-operative and early postoperative values but not late postoperative values in either group. The authors concluded that the results "supported the idea that inguinal mesh application is still a safe procedure in patients with no children or who are undergoing infertility treatment, where testicular function is important."

Color Doppler ultrasonography was used to assess testicular perfusion in another study by Dilek et al. [13]. Twenty-six patients were randomly assigned to undergo a totally extraperitoneal preperitoneal or a standard Lichtenstein TFR hernia repair. The specific blood flow indexes of the spermatic artery studied included end diastolic velocity, peak systolic velocity, and the resistive index. Studies were performed immediately pre-operatively and then 3 months after surgery. No differences were found between the pre-operative and postoperative measurements.

Laboratory Models

Several experimental studies in varying animal models have been published with mixed conclusions. The more widely referenced are summarized to illustrate this point. One of the studies was published in the Journal of Urology in 1999 by Uzzo and others [14]. Twelve male beagle dogs had inguinal hernia defects surgically created on one side of each animal. Six were repaired using a polypropylene TFR and the other six with a Shouldice technique. Study parameters included testicular temperature and volume, peripheral and testicular vein testosterone levels, testicular blood flow, vasography, testicular and cord histology, and sperm motility and morphology. The side without a hernia defect acted as a control. Postoperative testicular temperature, blood flow, and volume were similar to controls from both the mesh and Shouldice groups although there was a trend toward decreased volume in the TFR group(17.8 cc pre vs. 12.6 cc post, p = 0.17). Contralateral (control) testicular vein testosterone levels were higher in animals repaired with mesh than by Shouldice. There was a significant decrease in cross-sectional vasal luminal diameter in both repair groups compared to their respective contralateral controls. Microscopic examination disclosed a marked foreign-body reaction in the soft tissues surrounding the vas in the TFR group. All vasograms demonstrated patency. Three of the six TFR dogs had grossly abnormal pathology (two hydroceles and one ischemic testis). None of the Shouldice dogs demonstrated such findings. Sperm morphology and motility did not differ between the two groups.

Our group conducted a study to determine whether congenital indirect inguinal hernias in male pigs could be repaired by placing a polypropylene mesh prosthesis over the defect intra-abdominally [15]. The study design called for an assessment of the effect on fertility. The project differed from other mesh fertility studies in that the prosthesis was placed using an intraperitoneal onlay mesh technique (IPOM) meaning that peritoneum separated the prosthesis from the cord structures. Twenty-six healthy Yorkshire cross feeder male pigs weighing between 23 and 30 kg with congenital unilateral or bilateral indirect inguinal hernias were divided into two groups. In group 1, 13 pigs had a total of 19 indirect inguinal IPOM herniorrhaphies performed at the time of a laparotomy. Thirteen pigs in group 2 underwent the same operation laparoscopically and a total of 16 indirect inguinal hernias were repaired. All pigs were followed for 6 weeks postoperatively and allowed unrestricted physical activity and then sacrificed. There were no signs of erosion or damage to cord structures in any pig. There was normal flow of methylene blue without obstruction or extravasation (Fig. 31.1). A standard electrical transrectal ejaculation protocol employed in the livestock industry for artificial insemination was used to harvest sperm before sacrifice. Spermiograms were then performed and were normal.

Another evaluation of fertility was conducted by the respected group from Aachen, Germany, in pigs and rats [16]. Fifteen pigs underwent a TFR-type procedure on one side and a control operation Shouldice on the other. Three animals were sacrificed weekly until 35 days. On the TFR side, foreign-body reaction with diffuse infiltrating inflammatory cells was found in all. Five pigs were noted to have venous thrombosis of their spermatic veins and one animal was shown to have focal fibrinoid necrosis of the wall of the vas. On the control

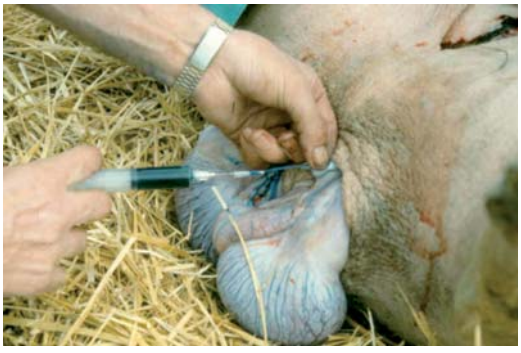


Fig. 31.1. Patency of the vas deferens was assessed with methylene blue injection

operated side, only minor postoperative changes were observed. The same operative scheme was used in eight chinchilla rabbits, but the study parameters in these animals included in addition to histological evaluation of the foreign-body reaction, testicular size, testicular temperature, testicular and spermatic cord perfusion, and spermatogenesis using the Johnsen scoring system. Just as in the pigs, there was much more foreign-body reaction on the mesh side than the Shouldice. In addition, there was decreased arterial perfusion and lower testicular temperature on the mesh side when compared to Shouldice. The TFR operation appeared to have adversely effected the Johnsen scale, which measures the rate of seminiferus tubules with regular spermatogenesis (TFR: 48.1%, Shouldice: 63.8%, controls: 65.8%). The authors voiced concern about this potential influence on spermatogenesis.

A study from Brazil included 18 dogs divided into three groups: group 1 (n = 7) underwent bilateral groin exploration with mesh being placed on the left side while the right had a non TFR repair [17]. In group 2 (n = 7), the sides were reversed (left side without mesh versus right side with mesh. Group 3 (n = 4) had no surgical manipulation (control group). The results were that there was increased chronic inflammatory reaction in all operated groups compared to controls, increased chronic inflammatory reaction on the mesh side compared to nonmesh, and decreased vas deferens diameter size on mesh side.

Taneli et al. examined testicular function, testicular nitric oxide metabolism, and germ cell-specific apoptosis in 40 rats who were divided into two groups consisting of a study group in whom a 0.5×1-cm polypropylene mesh patch was implanted behind the left inguinal spermatic cord and a sham-operated control group [18]. They concluded that long-term polypropylene mesh implantation has no effect on testicular hormonal function and only a limited effect on nitric oxide levels, and this effect is not sufficient to cause apoptosis in testis that could lead to infertility.

Another experimental study in rats evaluating how different types of mesh affect the spermatic cord was published in European Surgical Research by Berndsen and colleagues [19]. They divided 30 rats into 3 groups:

1. Conventional non-TFR repair,
2. TFR repair with a heavy-weight polypropylene mesh, and
3. TFR repair with large-pore, light-weight polypropylene/polyglactin composite.

Vasography was performed after 90 days. Study endpoints included cross-sectional area of the vas deferens and S-testosterone measured from the spermatic vein using the contralateral side as control. Light microscopy of the inguinal canal was performed and inflammation and fibrosis were graded. The vasography revealed patent vas deferens in all animals. In group III, there was a lower S-testosterone in the spermatic vein and a reduced cross-sectional area of the vas deferens on the operated compared to the control side. However, there was no difference in the other groups and there was no significant difference in S-testosterone levels between the groups. There was significantly more inflammation and fibrosis after mesh repair compared to suture repair, but there was no difference between the two mesh groups. The authors had no reason to believe that fertility would have been affected by any of these findings.

Conclusion

Infertility is a known complication of inguinal hernia surgery with or without mesh, and can be caused by a variety of mechanisms. The findings in the Shin manuscript which were the reason this review was undertaken are provocative and certainly provide an invitation for further study. However, careful analysis of the patients reported in that paper, as well as review of other pertinent literature, fails to unequivocally prove that polypropylene mesh can cause vasal obstruction as an independent aetiology. It seems logical that there might be a subset of patients in whom vasal obstruction will occur because of exquisite sensitivity to the fibroplastic response intended with the use of mesh material in hernia surgery. However, this subset must be quite small given that larger numbers of infertile patients are not being identified despite the fact that many patients having mesh hernia repairs are in an age group

still intending to father children. This is not just a matter of staunch TFR enthusiasts turning their backs and hiding their collective heads in the sand rather than face this "new revelation". On the contrary, the concern is overreaction to these level-4–5 evidenced based findings resulting in a return to the routine use of the Bassini operation or one of its nonprosthetic variants, which will inevitably lead to the need for more re-operative surgery for recurrence, which places the patient at the greatest risk of loss of fertility as a consequence of testicular atrophy.

References

1. Shin D, Lipshultz LI, Goldstein M, et al. Herniorrhaphy with polypropylene mesh causing inguinal vasal obstruction: A preventable cause of obstructive azoospermia. Ann Surg. 2005; 241: 553–558
2. Rutkow IM. Demographic and socioeconomic aspects of hernia repair in the United States in 2003. Surg Clin North Am 2003; 83: 1045–51, v–vi
3. Ceylan H, Karakok M, Guldur E, Cengiz B, Bagci C, Mir E. Temporary stretch of the testicular pedicle may damage the vas deferens and the testis. J Pediatr Surg 2003; 38: 1530–1533
4. Sheynkin YR, Hendin BN, Schlegel PN, Goldstein M. Microsurgical repair of iatrogenic injury to the vas deferens. J Urol 1998; 159: 139–141
5. Iles J. Specialisation in elective herniorrhaphy. Lancet 1965; 17: 751–755
6. Wantz GE. Testicular atrophy and chronic residual neuralgia as risks of inguinal hernioplasty. Surg Clin North Am 1993; 73: 571–581
7. Amid PK. Groin hernia repair: Open techniques. World J Surg 2005; 29: 1046–1051
8. Yavetz H, Harash B, Yogev L, Homonnai ZT, Paz G. Fertility of men following inguinal hernia repair. Andrologia 1991; 23: 443–446
9. Ridgway PF, Shah J, Darzi AW. Male genital tract injuries after contemporary inguinal hernia repair. BJU Int. 2002; 90: 272–276
10. Silich RC, McSherry CK. Spermatic granuloma. an uncommon complication of the tension-free hernia repair. Surg Endosc 1996; 10: 537–539
11. Seifman BD, Ohl DA, Jarow JP, Menge AC. Unilateral obstruction of the vas deferens diagnosed by seminal vesicle aspiration. Tech Urol. 1999;5:113–115.
12. Aydede H, Erhan Y, Sakarya A, Kara E, Ilkgul O, Can M. Effect of mesh and its localisation on testicular flow and spermatogenesis in patients with groin hernia. Acta Chir Belg. 2003; 103: 607–610
13. Dilek ON, Yucel A, Akbulut G, Degirmenci B. Are there adverse effects of herniorrhaphy techniques on testicular perfusion? evaluation by color doppler ultrasonography. Urol Int 2005; 75: 167–169
14. Uzzo RG, Lemack GE, Morrissey KP, Goldstein M. The effects of mesh bioprosthesis on the spermatic cord structures: A preliminary report in a canine model. J Urol. 1999; 161: 1344–1349
15. Fitzgibbons RJ,Jr, Salerno GM, Filipi CJ, Hunter WJ, Watson P. A laparoscopic intraperitoneal onlay mesh technique for the repair of an indirect inguinal hernia. Ann Surg 1994; 219: 144–156
16. Peiper C, Junge K, Klinge U, Strehlau E, Ottinger A, Schumpelick V. Is there a risk of infertility after inguinal mesh repair? Experimental studies in the pig and the rabbit. Hernia. 2006; 10: 7–12
17. Goldenberg A, Paula JF. Effects of the polypropylene mesh implanted through inguinotomy in the spermatic funiculus, epididium and testis of dogs. Acta Cir Bras. 2005; 20: 461–467
18. Taneli F, Aydede H, Vatansever S, Ulman C, Ari Z, Uyanik BS. The long-term effect of mesh bioprosthesis in inguinal hernia repair on testicular nitric oxide metabolism and apoptosis in rat testis. Cell Biochem Funct 2005; 23: 213–220
19. Berndsen FH, Bjursten LM, Simanaitis M, Montgomery A. Does mesh implantation affect the spermatic cord structures after inguinal hernia surgery? An experimental study in rats. Eur Surg Res 2004; 36: 318–322

Supported by a Grant from the United States Agency for Healthcare Research and Quality (5 R01 HS09860–03) and the Department of Veterans Affairs Cooperative Studies Research and Development Program (CSP #456).

Discussion

Deysine: *I would like to start the discussion by telling the audience that when this paper arrived I lost some sleep. But I also read a commentary that Dr. Fitzgibbons wrote on the same issue of Annals of Surgery and that cleared the air completely. It was extremely well written to the point and with all the information necessary to take away the initial panic that people may have had and I have to congratulate you for that. Thank you!*

Kingsnorth: *I agree completely with the way you have analyzed this very difficult topic and the literature review you have done on the animals and so on. Thinking a bit laterally, because you know we are in a chronic pain session as well: do you think that damage to the vas deferens done either by the surgeon or by stenosis caused by the mesh is a source of chronic pain? It is something we don't consider. But do you think there are some patients in whom a transscrotal vasogramm may be beneficial in helping us to diagnose the cause of chronic pain?*

Fitzgibbons: *That's a very interesting question. As you know, Dr. Bendavid has described the dysejaculation syndrome, which is a specific syndrome obviously related to the vas. Whether there'll be patients that have just generalized pain not associated with the ejaculation is interesting. I suppose it's a possibility, but a speculation for me, though.*

32 Principle Actions for Re-Recurrences

R. Schwab, U. Klinge

Introduction

Since the use of meshes for inguinal hernia repair has become increasingly popular, hernia surgery has undergone major changes in the past 15 years. Approximately 200,000 inguinal hernia operations are performed annually in Germany. Whereas synthetic meshes were used in less than 10% of cases in 1995, they are today applied in about every second patient using either a conventional or endoscopic approach. In England, Scotland and the United States, the percentage of mesh repairs is even between 70 and 86% [1, 2].

The main argument for a wide expansion of mesh techniques is an expected lower recurrence rate compared to suture techniques. A model for calculating the recurrence rates with the assumption of meta-analysis leads to expected 15% recurrent hernias following suture repairs and a total of 5% after mesh techniques. But the use of meshes did not eliminate the recurrence. In spite of the wider application of meshes, the rate of operations for recurrent inguinal hernias in Germany continues to be 8–15% [2, 5, 10]. Mesh material that was introduced during a previous operation is currently detected in about 10% of recurrences or an annual number of approximately 2500 patients in Germany [5, 10]. With an increasing use of meshes, we have to face more recurrences following previous mesh repair in the future (◘ Fig. 32.1).

The implantation of a mesh leads not only to excessive scarring and thus to the desired stabilization of the hernia site but also to massive adhesions in many cases. For this reason, the surgical revision of a previous mesh repair is a major technical challenge for every surgeon. In the case of multiple recurrences, the degree of difficulty of a revision operation increases drastically with the number of previous operations.

How to Treat the Re-Recurrence?

To debate this problem, multiple factors have to be considered. There is a huge variety of previous techniques performed for inguinal hernia repair. None of these procedures is suitable as a standard for all types of recurrences. Furthermore, the indication to explant the former mesh has to be clarified. Therefore an individual approach considering the previously applied technique has to be performed. This requires an extended experience in various techniques.

In order to assess the situation in the management of recurrent hernia, we analyzed the medical records of 672 patients who underwent recurrent inguinal hernia repair in Aachen (n = 438) and Koblenz (n = 234) between January 2000 and September 2004. In 92 of these cases (13.7%), a mesh had been used in a previous repair (Aachen: n = 63 or 14.4%; Koblenz: n = 29 or 12.4%). Altogether 44 patients presented with a first recurrence, 26 patients with a second recurrence, 10 patients with a third recurrence and 12 patients with multiple recurrences (4 to 11). Of the 92 included patients 47.8% had a first and 52.2% had multiple recurrences after previous mesh repair.

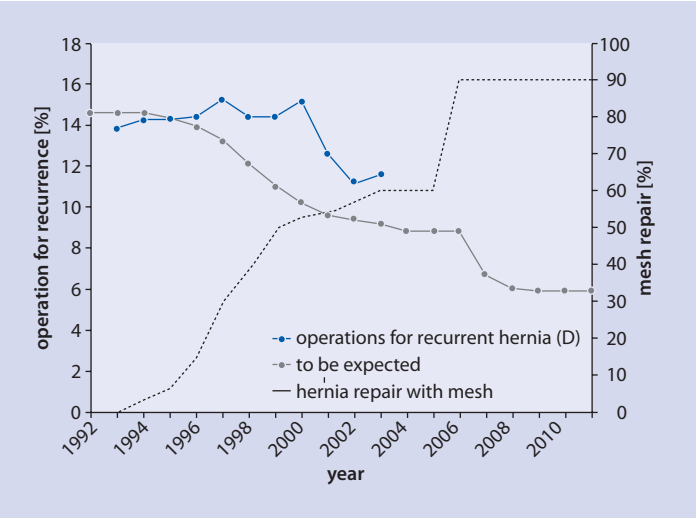


Fig. 32.1. In Germany, the mesh did not eliminate recurrence as should be expected

In the previous operation, the mesh had been placed anterior to the posterior wall of the inguinal canal in 55 cases (59.8%) and in a preperitoneal position using a posterior approach in 37 cases (40.2%). A Lichtenstein repair as anterior onlay mesh had been carried out in the majority of previous operations (56.5% or n = 52). A previous endoscopic technique (13 TEP and 19 TAPP procedures) had been used in 34.8% of the patients (n = 32).

There was a wide variety of reasons why a particular operative procedure was chosen for the repair of a recurrent hernia. The ultimate decision as to which technique to use was made as late as during surgery in 71 cases (77.2%). In a mere 21 cases (22.8%), the surgeons decided before surgery to perform either an endoscopic procedure or a conventional (Stoppa, Wantz) approach (Table 32.1).

After a previous anterior approach (Lichtenstein: n = 52, plug and patch: n = 3), an anterior repair technique was again chosen in 24 cases. In 12 of these cases, the surgeons used a Shouldice procedure or a direct suture for the closure of small defects. The mesh was removed in 8 of these cases. A Lichtenstein repair was performed for the repair of both the previous and recurrent hernias in 10 cases (a larger medial overlap was created in the majority of these cases). In one case, the Lichtenstein technique was chosen after a previous plug and patch repair. A total of 31 of the 55 patients who had undergone a previous anterior repair had a preperitoneal repair for a recurrent hernia. An endoscopic (TEP) approach was used in 7 of these cases and a conventional TIPP repair was chosen in 15 cases (6 meshes were removed). Last but not least, a Wantz repair was performed in 5 cases and a Stoppa repair in the remaining 4 cases (Table 32.1).

After a previous preperitoneal repair (32 endoscopic TAPP or TEP procedures, 5 conventional Stoppa, Wantz or TIPP procedures), the technique was changed and an anterior placement of the mesh was chosen in 30 patients. A Lichtenstein repair (TAPP or TEP) was performed in 15 of these cases, a Tipp repair in one case and a direct suture or a Shouldice repair in another 15 cases. In six cases with a previous posterior repair, a preperitoneal mesh was implanted again using a Stoppa repair after a Wantz procedure in two cases, a TIPP repair after a TEP procedure in one case, a Wantz repair after a TAPP procedure in another case, a TAPP repair after a Stoppa procedure in one case and a TAPP repair was repeated in one case (Table 32.1).

An analysis of the records showed that the decision as to which repair technique to use was mostly made on the basis of each individual case. In the majority of cases, it is not possible to identify a definitive algorithm for the selection of a technique. The following statements can be made:

- There is a huge variety of previous techniques performed for inguinal hernia repair.
- A transinguinal repair technique was usually used for revision in patients presenting with pain and a recurrent hernia.
- Where multiple recurrences could not be managed using the commonly employed technique, a minimal direct suture repair (either with or without the placement of an additional small mesh) was used

Table 32.1. Repair techniques used in the previous and revision operations

Previous operation	Revision operation							Total
	Shouldice or suture	Lichten-stein	TIPP	TAPP	TEP	Wantz	Stoppa	
Lichten-stein	12	10	15	0	7	4	4	52
TEP	4	8	1	0	0	0	0	13
TAPP	10	7	0	1	0	1	0	19
Wantz	1	0	0	0	0	0	2	3
Stoppa	0	0	0	1	0	0	0	1
Plug and patch	1	1	0	0	0	1	0	3
TIPP	0	1	0	0	0	0	0	1
Total	28	27	16	2	7	6	6	92

Table 32.2. High rate of re-recurrences following non-mesh repair after previous mesh repair[a]

Re-recur-rence	Suture	Lichten-stein	TEP	TIPP	TAPP	Wantz	Stoppa
No	20	25	7	14	2	5	5
Yes	6 (23%)	1	0	1	0	0	1

[a]Follow-up of 87 Patients (94.6%) after 36.3 months (13–68); re-recurrence rate 10.3% (n = 9)

for small defects or a preperitoneal (Wantz, Stoppa or TAPP) approach was used for inserting a new large mesh.

To follow up the patients, telephone interviews were performed on the basis of a questionnaire in order to assess the outcome of revision operations for recurrences after previous mesh repairs (Table 32.2). The mean follow-up was 36.3 months (13 to 68 months; median: 33) or, in other words, slightly more than 3 years. It was possible to conduct interviews with 87 of the 92 patients. One patient had died of another cause, but had had no recurrence. Another 4 patients could not be contacted. Accordingly, a follow-up rate of 94.6% was achieved. Whereas 9 patients (10.3%) had undergone surgery for a re-recurrence by the time of follow-up, all other patients had had no recurrence. The re-operations had been performed after an average of 19.9 months (9–38 months) after the last repair. Only patients with previous multiple recurrences were affected. Of the 26 patients who had undergone a non-mesh repair, 6 had a recurrence. This group of patients showed the highest re-operation rate (23.1%).

The surgical management of recurrent inguinal hernia after a previous mesh repair is a technically demanding challenge for a surgeon. Compared with a suture repair, the mesh technique leads to considerably more scarring, thereby making it usually much

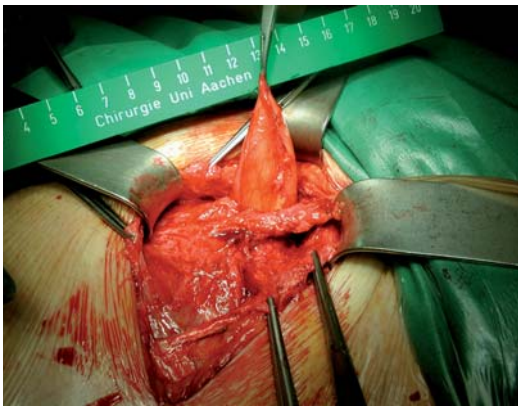


Fig. 32.2. Difficult dissection in scarry tissue with increased risk for spermatic cord and nerves

more difficult for the surgeon to identify anatomical landmarks and in most cases impossible to preserve selective nerves (Fig. 32.2). Especially the traditional heavyweight small-pore meshes are often associated with the formation of massive scar and fibrous tissue [1, 6, 12].

Altogether 22 of 92 meshes were explanted in our patient population. All Rutkow plugs were removed during the revision operation. In the absence of pain or signs of infection, meshes were left in situ during the revision operation. Meshes were removed only if the patient reported a relevant foreign-body sensation or pain. Our approach is according to the literature. The removal of a previously introduced mesh is indicated if the patient complains of chronic pain that cannot be managed by neurolysis, if the foreign material causes discomfort or if a massive infection with abscess formation develops around the mesh [1]. In addition, it is postulated that there should be very strict indications for the removal of mesh material and that the surgeon must have extensive experience in hernia surgery and experience in vascular surgery. Especially in the presence of massive adhesions in the region of the major vessels, it is better to leave mesh material in situ than to risk vascular or spermatic cord damage. A mesh that is not causing a problem can usually be left in place.

There are no generally accepted guidelines and only a paucity of data on the choice of repair technique for recurrences after a previous mesh repair. Whereas some authors recommend repeating the primary procedure and the placement of an additional mesh [4, 9], others advocate changing the procedure and using an anterior approach after a posterior procedure and vice versa [3, 7, 8, 11].

In our experience, the choice of technique depends on the previous repair technique and on the need for removing the foreign material that was inserted beforehand (Fig. 32.3). The mesh must be removed if there

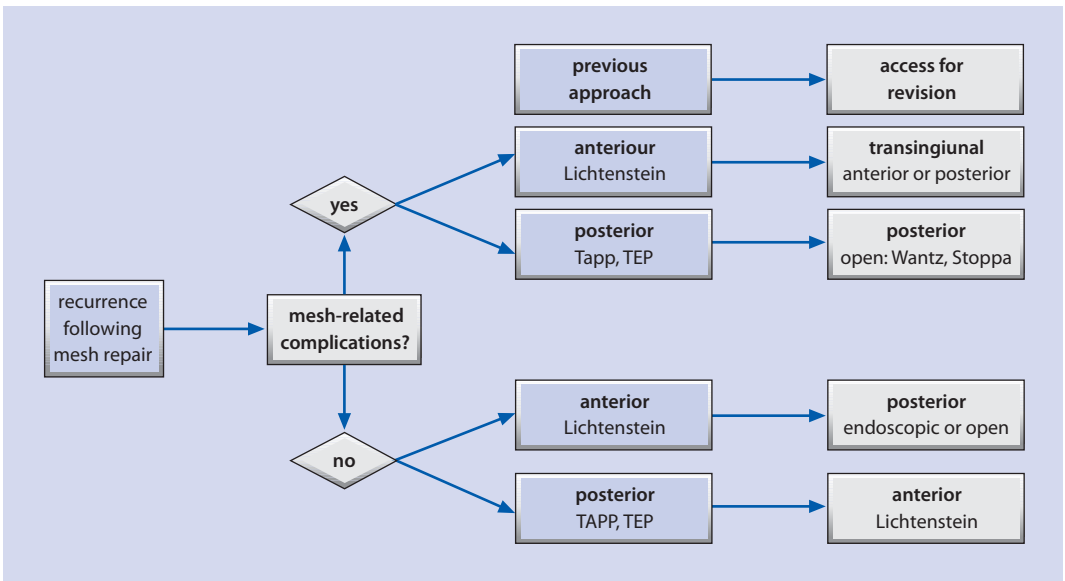


Fig. 32.3. Algorithm for selecting the most appropriate type of revision operation for the management of recurrent hernia after a previous mesh repair

are complications such as a foreign-body sensation and pain. The presence of these symptoms appears to require a conventional transinguinal approach for the revision operation. The use of a posterior technique for reconstructing the posterior wall of the inguinal canal after a previous anterior procedure or vice versa makes it easier for the surgeon to perform the operation since mesh is placed in a non-operated area. Likewise, a change of surgical approach in patients where the mesh causes no complications has the main advantages that the trauma of access is minimized and the surgeon can operate through intact tissue.

An algorithm (Fig. 32.3) is provided to advice on the selection of the most appropriate repair technique. Depending on local expertise, it is also possible to repeat previous TAPP or TEP procedures, which, however, are highly demanding and much more difficult to perform than a repair after a change in technique. Patients with multiple recurrences after a previous Stoppa repair (GPRVS) present a particular challenge for the surgeon. In our opinion, the best repair approach in these cases appears to be a transabdominal reinforcement of the abdominal wall using a TAPP approach. Both a laparoscopic and an open repair are possible.

Conclusions

There is currently neither an algorithm for selecting the most appropriate type of revision operation in the management of recurrent hernia after a previous mesh repair, nor is there general agreement on how to choose a technique. The increasing use of mesh techniques requires that we address this problem in a constructive and effective way. As a general rule, re-operations after mesh repairs are technically more demanding than re-operations after previous Shouldice repairs and require a high level of professional skill on the part of the surgeon. A change of technique from an anterior to a posterior approach and vice versa enables the surgeon to operate through intact tissue. The mesh should be removed in patients presenting with complications such as pain and a foreign-body sensation. Multiple recurrences require a mesh repair and a preperitoneal placement of the new mesh. This is emphasized by our follow-up data, suggesting a high rate of failure for the suture repair of recurrent hernias after a previous mesh repair. The best way to minimize the number of revision operations after mesh placement is a thorough knowledge of potential weaknesses and limitations of the primary operations and thus to avoid recurrences due to technical failures.

References

1. Arlt G (2004) Explantation of meshes as a routine in future? In: Schumpelick V, Nyhus LM (eds) Meshes: benefits and risks. Springer, Berlin Heidelberg New York, pp 413–426
2. Atkinson H, Nicol S, Purkayastha S, Paterson-Brown S (2004) Surgical management of inguinal hernia: retrospective cohort study in southeastern Scotland, 1985–2001. BMJ 329 (7478): 1315–1316
3. Barrat C, Surlin V, Bordea A, Champault G (2003) Management of recurrent inguinal hernias: a prospective study of 163 cases. Hernia 7(3): 125–129
4. Ferzli GS, Shapiro K, DeTurris SV, Sayad P, Patel S, Graham A, Chaudry G (2004) Totally extraperitoneal (TEP) hernia repair after an original TEP. Is it safe, and is it even possible? Surg Endosc 18(3): 526–528
5. Hermanek P (2004) Qualitätssicherung der Leistenhernienoperation. Viszeralchirurgie 39:8–12
6. Klinge U, Zheng H, Si Z, Schumpelick V, Bhardwaj RS, Muys L, Klosterhalfen B (1999) Expression of the extracellular matrix proteins collagen I, collagen III and fibronectin and matric metalloproteinase-1 and -13 in the skin of patients with inguinal hernia. Eur Surg Res 31: 480–490
7. Kurzer M, Kark AE, Belsham PA (2005) Open preperitoneal mesh repair for recurrent inguinal hernias. Hernia 9(1): 105
8. Kurzer M, Belsham PA, Kark AE (2002) Prospective study of open preperitoneal mesh repair for recurrent inguinal hernia. Br J Surg 89(1): 90–93
9. Leibl BJ, Schmedt CG, Kraft K, Ulrich M, Bittner R (2000) Recurrence after endoscopic transperitoneal hernia repair (TAPP): causes, reparative techniques, and results of the reoperation. J Am Coll Surg 190(6): 651–655
10. Mohr D, Bauer J, Döbler K, Fischer B, Woldenga C (2003) BQS-Qualitätsbericht 2002- Modul 12/3: Hernienoperation. Bundesgeschäftsstelle Qualitätssicherung gGmbH, Düsseldorf
11. Richards SK, Vipond MN, Earnshaw JJ (2004) Review of the management of recurrent inguinal hernia. Hernia 8(2): 144–148
12. Schumpelick V, Klinge U (2003) Prosthetic implants for hernia repair. Br J Surg 90(12): 1457–1458

Discussion

Miserez: *I agree with the lower parts of your slide completely. With the upper parts I would just like to say what has been stressed by the previous speakers. If you have to take out the mesh which is probably the case in infection, even if it's difficult then there is no problem in taking out the mesh entirely and doing an endoscopical repair posteriorly to place a new mesh if necessary, so there is, I think, definitely place in those difficult cases for a combined approach.*

Schwab: *Combined approach was exactly what I also made possible and it also depends on the skill of the surgeon who performs it. If you are an absolute expert in TEP or in TAPP, you will have probably an easier ap-*

proach to the posterial wall than Prof. Flament with his anterior method and his expertise, so it's not a question of this council here, it's a question for the surgeons out on the field performing 99.9% of the hernia repairs not the 0.1% we perform here.

Amid: *Many surgeons are afraid of doing anterior repair after an original mesh repair because it's more scar tissue. If I'm given a choice of doing a recurrent hernia repair I will pick a patient who had a previous mesh repair and this is, at least in my mind, for a very logical reason. When there is mesh in the groin, that mesh for me is a point of reference. I can stay on the mesh, shave off everything else the mesh and then do the rest of the operation. Whereas when there is no mesh in the inguinal canal it is all scar tissue. My reference point is gone. If I go too deep I may end up in the bladder. If I go too superficially I may end up in the spermatic cord and cause testicular problems. But when the mesh is there at least in one direction I'm safe and I have repeatedly mentioned that, but it seems that it is only my preference. Nobody else agrees with me. People are afraid of that extra scar tissue when there is a mesh there, but the presence of mesh, as I said, is good for me, it is a point of reference for me that makes my operation safer at least in one direction.*

Schwab: *While writing the paper on our patients and on our results I looked in the literature and find that most surgeons suggest doing the redo in an untouched layer. It's easier for most surgeons, but might not be true for you.*

Amid: *I know. As I said, this is surgeon-dependent. I'm more comfortable with the anterior approach and I mentioned the reason, but recurrent hernias are difficult, no matter what you do.*

Young: *Dr Amid, I would agree with you. However, there are many situations where I do refer these patients to laparoscopists, even though I don't do this procedure myself.*

Treatment of the Other Hernia

33 Laparoscopic Repair of Recurrent Childhood Inguinal Hernias After Open Herniotomy

K.L. Chan

Introduction

Repair of inguinal hernia (IH) is one of the most common operations in paediatric surgical practice [1]. The incidence of IH ranges from 0.8 to 4.4% in children of all ages. It is particularly common in the first year of life.

Open repair is still the popular method of treatment for paediatric IH [2, 3] which is the result of a patent processus vaginalis only. There is no need for muscle strengthening procedure after the division and ligation of the hernia sac. However, the recurrence rate still ranged from 1.76 to 6.3% [4–6]. The high recurrence rate was attributed to the setting of a general department, where several surgeons and residents operated upon a limited number of paediatric patients [6], the other reasons suggested being junior surgeons or surgeons without specific paediatric surgical training performing the operations.

In boys, re-operations are difficult and required tedious and careful dissection of dense fibrous tissue resulting from the previous surgery. There is a definite risk of damaging the vas deferens and testicular vessels, which are situated in the midst of the dense fibrous tissue.

Our centre reported a safe laparoscopic method for paediatric IH repair [7–9]. The operative site is above the previous operative field if it is a recurrent hernia after an open operation. The laparoscopic method should have less chance of damaging the vas deferens and testicular vessels.

The present study was to evaluate our laparoscopic repair for paediatric recurrent inguinal hernia after open repair. The results were also compared with the historic data of the same laparoscopic method used as the first attempt at IH repair

Materials and Methods

The medical records of all paediatric patients who were treated laparoscopically in our institution for recurrent IH after open surgery were reviewed retrospectively. The parameters of sex, age, follow-up duration, operation time, success rate and complications of the patients were noted. The data were compared with the historic data from our previously reported IH patients who were treated laparoscopically as the first initial hernia operation [9].

Continuous data were expressed as mean +/- standard deviation (SD) and statistical significance with two-tail t test or Mann-Whitney test. For proportion data, Chi-square or Fisher's exact test was used. Statistical significance was set at $p < 0.05$.

Surgical Technique

The detailed technique has been reported elsewhere previously [7–9]. Briefly, after the induction of general endotracheal anaesthesia, the patient was placed in the Trendelenburg position. A 5-mm port was then inserted through the umbilicus. Pneumoperitoneum of pres-

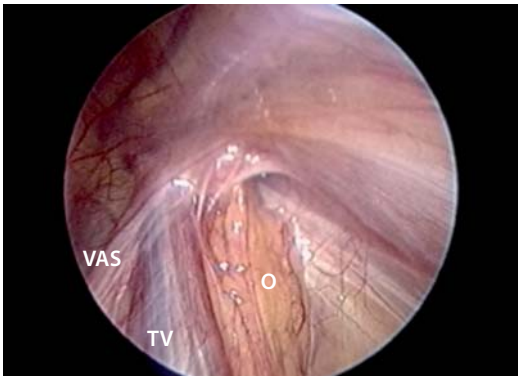


Fig. 33.1. Laparoscopic photo showing the right internal inguinal opening of the recurrent hernia. O omentum; TV testicular vessels; VAS Vas deferens

sure between 8 and 10 mmHg was created with carbon dioxide. The internal opening of the hernia was first confirmed and then the opposite side was inspected. Two more 3-mm ports were placed under telescopic vision via the abdominal wall medial to the anterior superior iliac spine. Contents of the hernia, such as omentum or bowel loop were gently dissected from the hernia sac (Fig. 33.1). For girls, 3/0 prolene stitch was placed into the peritoneal cavity through the abdominal wall. A purse-string suture was placed around the internal hernia opening and tied using intraperitoneal knotting. The ends of the stitches were then cut after the needle was passed out through the abdominal wall.

For boys, to separate the important structures of vas deferens and testicular vessels from the peritoneum, normal saline injection was given at the extraperitoneal space with the injector (6F, 155 mm, NM-3k injector, Olympus, Tokyo, Japan) which was guided by a metal cannula (Stryker, Santa Clara,LA) (Fig. 33.2). On placing the needle for the purse-string stitch, "needle sign" was emphasized. "Needle sign" is the sign in which the

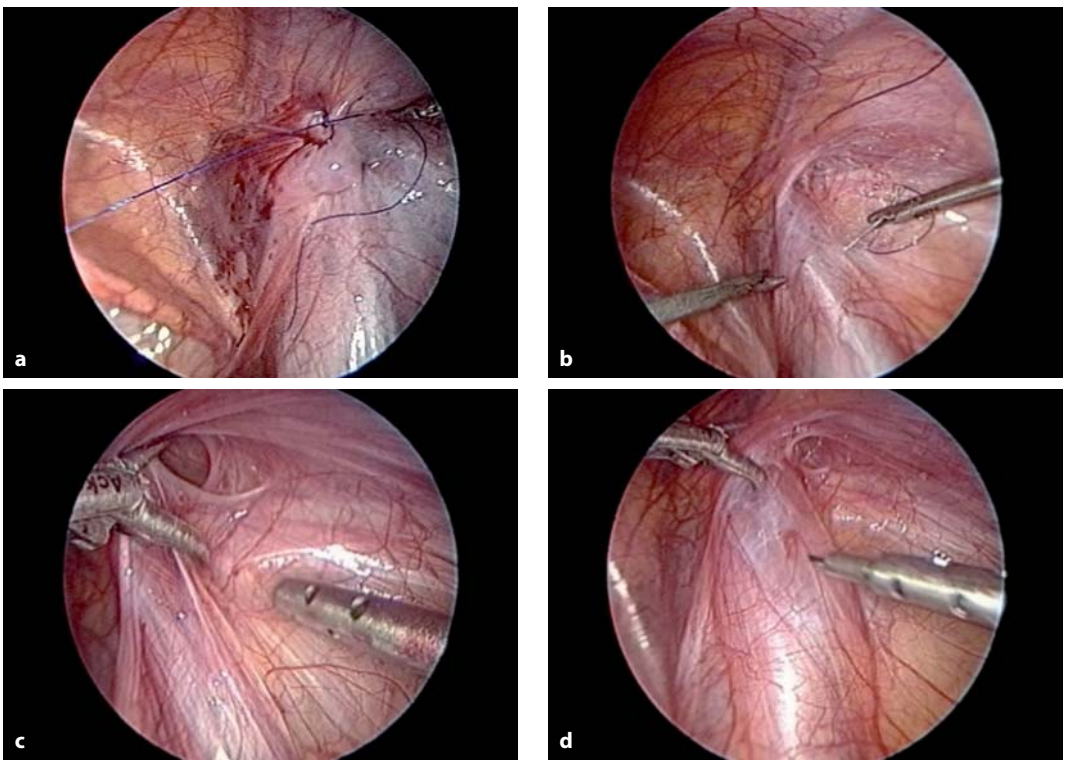


Fig. 33.2. a Appearance of the internal inguinal opening after the portion of omentum dissected from the opening. There was not much fibrous tissue around the opening. **b** Extraperitoneal saline injection easily separated the testicular vessels and vas deferens from the peritoneum. **c** Purse string stitch was put around the internal inguinal opening. d An intracorporal knot tightly closed the internal inguinal opening.

Table 33.1. Comparison between laparoscopic repair of recurrent childhood hernias with historic data for first laparoscopic attempt repair of childhood hernias

	Recurrent lap hernias (n = 5)	Historic lap hernias (n = 41)	P value[a]
Sex (male:female)	4:1	34:7	0.634
Age [months]	58.8 +/– 68	56+/– 45.67	0.91
Follow-up [months]	21 +/– 13	12.2 +/– 2.83	0.121
OT time(unilateral)	25 +/– 5.58 min	23.25 +/– 6.26 min	0.842
OT time(bilateral)	35 min	34.0 +/– 6.26 min	0.642
Successful rate [%]	100	100	> 0.05
Testis atrophy [%]	0	0	> 0.05
Recurrence [%]	0	0	> 0.05

[a]Statistic significance is $p < 0.05$; data expressed as mean +/– SD

needle could be seen clearly underneath the peritoneum without the vas and the testicular vessel in between. The sign further protected these important structures to be included in the stitch.

The stitch ends were pulled and tightened slightly before they were tied together. A complete ring of peritoneum without the presence of visible significant portion of raw stitch was named the complete ring sign. Only then were the ends tied and the opening closed completely. The complete ring sign was used to prevent recurrence.

After the pneumoperitoneum was released, the ports were removed. The umbilical wound was closed with absorbable stitches and the lateral ones with sterile strips.

Results

From September, 2002, to October, 2005, four boys and one girl were treated in our institution for recurrent IH after open operation. Their mean age was 58.8 months (Table 33.1). One patient had bilateral hernias after an open operation on one side in another institution. Both hernias of the patient were treated laparoscopically in one operative setting.

All patients were treated successfully with our laparoscopic technique. There was no recurrence detected in the group of patients with the mean follow-up period of 21 months. There was no testicular atrophy nor other possible complications detected on follow up.

The present data such as operative time, complications, when compared with our previous reported data from a series of patients who had laparoscopic hernia repair as the first operation and their data were collected prospectively [9] and showed no statistical significance (Table 33.1).

Discussion

After reviewing 71 recurrent IH after open repair in 62 children, Grosfeld et al. [10] suggested adequate high ligation at the internal ring, snugging of a large internal ring, avoidance of injury to the canal floor and closure of the internal ring in girls to prevent indirect hernia recurrence. From the above technical considerations, the laparoscopic method theoretically can avoid recurrence. However, the recurrence rate was reported to be 3.4% in a three-centre experience with 933 repairs [11]. The main reason may be due to the presence of testicular vessels and vas deferens in close proximity to the peritoneum at the expected site of closure near the internal ring (see Fig. 33.1). Our technical refinement in the use of saline injection to separate these structures from the peritoneum (see Fig. 33.2) and the emphasis of the complete ring sign during surgery has reduced the recurrence rate to 1% [8].

In a first initial operation for IH, laparoscopic repair is also found to be superior to open operation with regard to postoperative pain, recovery and cosmesis. It can also allow detection of contralateral hernias and have them repaired at the same operation [9]. The findings were based on our prospective randomized single-blinded control study to compare the two forms of operation for paediatric IH.

For recurrent hernias after open operation, re-operation with the open method needs to go through the old operation site which in boys almost always has the vas deferens and testicular vessels embedded in dense fibrous tissue. The operation is always tedious and possesses the danger of damaging these important structures. From the present retrospective study, the laparoscopic method is the preferred operation for recurrent hernias after open hernia repair. It has all the superior aspects of laparoscopic method and can also avoid the previous operation site. Thus, it can avoid damaging the vas deferens and testicular vessels. Further, it is as simple as a fresh hernia repair because the time taken for the repair of recurrent hernia laparoscopically was the same as the fresh laparoscopic repair (see ◘ Table 33.1). There was no added complication nor was it less successful as compared with the initial laparoscopic operations. There was no recurrence in the present group of patients after a mean follow-up of 21 months.

In conclusion, laparoscopic repair is the preferred operation for recurrent childhood IH after open operation. With refinements in the technique in laparoscopic repair, recurrence can be prevented even in this group of patients.

References

1. Cheung TT, Chan KL (2003) Laparoscopic inguinal hernia repair in children. Ann Coll Surg HK 7: 94–96
2. Levitt MA, Ferraraccio D, Arbesman MC, Brisseau GF, Caty MG, Glick PL (2002) Variability of inguinal hernia surgical technique: A survey of North American pediatric surgeons. J Pediatr Surg 37: 745–751
3. Antonoff MB, Kreykes NS, Saltzman DA, Acton RD. (2005) American academy of pediatric section on surgery hernia survey revisited. J Pediatr Surg 40: 1009–1014
4. Carneiro PM (1990) Inguinal herniotomy in children. East Afr Med J 67: 359–364
5. Harvey MH, Johnstone MJ, Fossard DP. (1985) Inguinal herniotomy in children: a five-year survey. Br J Surg 72: 485–487
6. Nazir M, Saebo A (1996) Contralateral inguinal hernial development and ipsilateral recurrence following unilateral hernia repair in infants and children. Acta Chir Belg 96:28–30
7. Chan KL, Tam PK (2003) A safe laparoscopic technique for the repair of inguinal hernias in boys. J Am Coll Surg 196: 987–989
8. Chan KL, Tam PK. (2004) Technical refinements in laparoscopic repair of childhood inguinal hernias. Surg Endosc 18: 957–960
9. Chan KL, Hui WC, Tam PK (2005) Prospective randomized single-center, single-blinded comparison of laparoscopic vs repair of pediatric inguinal hernia. Surg Endosc 19: 927–932
10. Grosfeld JL, Minnick K, Shedd F, West KW, Rescorla FJ, Vane DW. (1991) Inguinal hernia in children: factors affecting recurrence in 62 cases. J Pediatr Surg 26: 283–287
11. Schier F, Montupet P, Espostito C (2002) Laparoscopic inguinal herniorrhaphy in children: A three-center experience with 933 repairs. J Pediatr Surg 37: 395–397

Discussion

Ceydeli: *Thanks, Dr. Chan, for this great presentation and I think that as pediatric surgeon I have to say that this is really a revolution in how we're doing hernia surgery on children. I just have one quick comment and then a couple of questions for you. Firstly I'm doing this operation laparoscopically as well but I do not put the sutures in place intracorporally. I find that managing a suture, especially in a premature infant, and a needle is not necessarily an easy task and so what we're doing is replacing a 2-mm incision – just a stab incision – over the internal ring and then passing the suture circumferentially around the neck of the hernia sac and tying it down in the subcutaneous tissues. This we find is faster than trying to place the suture inside. I agree with you that the recurrent hernia – I've had one recurrent hernia in a child who was constipated in straining and the suture released – and the recurrent hernia is as easy as doing the initial hernia operation. A couple of questions: How do you decide whether you should close the opposite side or not, given the high chance of spontaneous closure of the pin processes? The next question is how young are these patients and also what about patients who have ascites, or are you using laparoscopy for these patients?*

Chan: *Thank you for the comment and also for your questions. There are a number of ways to kill a cat and you have mentioned one and then I mention mine. I think I can do the knotting. I find no problem. You found that there is a problem in diagnosis. I think you just continue the operation and there is a contralateral repair. I think if we are doing a laparoscopic method we find holes in the other side because is a sign to put stitches with minimal or no chance of damaging anything. So whenever we see something, we close it if we are doing the laparoscopic repair; for closure I think there is no prospective study proof that the patent process will definitely close. So there is no evidence of this kind. So I think at operation you have to close the other side as well if you find the holes open on the other side.*

Ceydeli: *The patients that may have ascites – are you using laparoscopy on them?*
Chan: *At the present moment we do not. Maybe later we'll try, but the thing is that we don't know the cause of ascites.*
Read: *Dr. Chan, in your first statement regarding the cause of these hernias in infants you mentioned a patent processus vaginalis. We know that the patent processus vaginalis can persist through life without any hernia development. My own son, who is now 54 years of age, as a neonate had a communicating hydrocele of the cord. That went away. He has never had a hernia. But we do know he did have a real patent processus vaginalis. I'd like you to comment on that.*
Chan: *If it's a hernia it means there is a big patent processus vaginalis, then I think at the present moment there is no definition as to how or why the patent processus vaginalis is a hernia. I suppose if it is more than half a centimetre, then the bowel can get in and it can become a hernia. There was a paper published in the Asian Journal of Surgery in the recent issue. They will close a patent processus vaginalis that is half a centimetre in diameter – but the thing is that is no data.*

34 The Femoral Hernia – the Bête Noire of Hernias!

R. Bendavid

Introduction

"An error made on your own is safer than ten truths accepted on faith" (Ayn Rand, Atlas Shrugged 1957). Rand's aphorism summarizes all the fears one must experience to become familiar with the difficult clinical diagnosis and surgical treatment of femoral hernias. And more than one error it will be! The cause of this all too common fear is the lack of familiarity with the problem. Femoral hernias are less frequently seen than inguinal hernias and make up only 2 to 5% of all groin hernia series. If the average general surgeon treats 50 hernias a year, this means that he may handle from one to perhaps two femoral hernias a year [1].

Femoral Laws

At the risk of sounding repetitive and trite and to hammer a point home (is it not what Madison Avenue advertising agencies do with publicity spots?), some platitudes about femoral hernias must be enshrined as "Femoral Laws".

First Law of Femoral Hernias. Remember that the first operation is the best chance of a cure. All subsequent attempts will be attended by danger, fear, failure and complications [2].

Second Law of Femoral Hernias. You must search for and exclude femoral hernias during all surgeries in the groin. These hernias account for more than 8% of all recurrences and can be especially difficult [3].

Third Law of Femoral Hernias. Whether surgery is carried out through an open or a laparoscopic technique, never disturb any fat pad or lymph node present at or within the femoral ring [4].

Fourth Law of Femoral Hernias. All femoral hernias must be repaired with a mesh from, or within, the preperitoneal space. Suture repairs, however small the defect, can no longer be trusted [5].

Fifth Law of Femoral Hernias. Surgery for femoral hernias must be done at the earliest convenience if elective. In emergencies, whether incarcerated of strangulated, never delay. In strangulation, complications and mortality vary directly and proportionally with the duration of the delay [6].

Femoral hernias have been described as the most treacherous of all hernias and when incarcerated, they outnumber all other forms of incarcerated abdominal wall hernias combined [6]. The diagnosis is missed in 25% of cases [7]. Incarceration and strangulation have been reported in 2–25% [6, 7].

Table 34.1. Re-recurrence rate of femoral hernias

1x recurrent femoral hernia	11.8%
2x recurrent femoral hernia	34.7%
3x recurrent femoral hernia	34.6%
4x recurrent femoral hernia	30.0%
5x recurrent femoral hernia	75.0%
Average	22.0%
Mean	37.2%

The incidence of recurrence is often quoted as being between 0 and 1.1% after mesh repairs and from 0–6.5% after sutured repair [8]. I have long suspected these figures to be low. The suspicion was confirmed when one of the largest series ever reported and with which I was associated (508 cases) revealed that 50% of femoral hernias admitted to the hospital were already recurrences. This pattern had been noted in previous years. That same series which reflected a careful follow-up of the patients (84.7% after 4 years), revealed that recurrences ranged from 11.8–75% depending on the number of previous operations (Table 34.1) [9].

In the selection of the patients to be followed, those who were included were patients who had had a femoral hernia confirmed at surgery. When a recurrence took place, only those patients who had a femoral hernia recur, in other words a true recurrence of the original pathology, were included in the follow-up study. If the recurrence after a femoral repair was an inguinal hernia or if a femoral hernia followed an inguinal repair, these patients were not included in the study. The aim of the study was to identify and confirm a pure femoral hernia and document the recurrence of a pure femoral hernia. Interesting additional facts which emerged was that women made up 52.5% of 251 primary femoral hernias while they made up only 18% of 257 recurrent femoral hernias. All these patients underwent elective surgery. However, when patients present in emergencies with incarceration or strangulation, sometimes requiring a bowel resection, 76.7% turn out to be females [10].

Three significant factors have accounted for the complexity of femoral hernia as a clinical entity. These factors are: the intricacy of the anatomy, the flimsy nature of the tissues available for repair, and tension.

Intricacy of the Anatomy

True understanding of the femoral canal was the major contribution of Chester McVay [11] and Fruchaud [12]. In simplest terms, the femoral canal is formed by the development of the femoral vessels which drag along with them, the true
into the thigh. This transversalis fascia is that part of the endopelvic fascia, flimsy as it is. It is not to be confused with what is commonly called the transversalis fascia but is, in fact, the transversus abdominis aponeurosis. The latter on its deepest surface is adjacent to the true transversalis fascia and both are referred to, erroneously, as the "transversalis fascia". The femoral canal is therefore lined with true transversalis fascia which comes to lie and fit against nearby elements. These surrounding elements create a funnel shaped structure with an inlet and a body.

The inlet is rigid and its limits are:

- Posteriorly: the pubic crest and Cooper's ligament.
- Anteriorly: the inguinal and Thomson's ligaments.
- Medially: the lateral edge of the lacunar ligament of Gimbernat.
- Laterally: the femoral vein.

The body of the funnel, however, is walled by:

- Anteriorly, the anterior leaf of the fascia lata.
- Posteriorly: the pectineus fascia (medially) and the posterior leaf of the fascia lata (laterally).
- Medially: the lacunar ligament of Gimbernat.
- Laterally: the femoral vein.

It is important to distinguish, as pointed out by Fruchaud, that the crural canal is that which houses the femoral artery, femoral vein and the lymphatic canal as they descend from the abdominal cavity into the thigh, while the femoral canal is the most medial part of the crural canal, covered superiorly by a fat pad and or a lymph node. It is the canal into which a femoral hernia will descend and enlarge in the direction of the fossa ovalis where the latter makes room for the hook of the saphenous vein.

Nature of the Tissues

It becomes readily apparent that the tissue forming the femoral canal is of no substance. Laterally, where it is called the femoral sheath and is adjacent to the femoral vein, it is so thin that the naked eye can rarely identify it. Certainly, it is of no surgical value in terms of retaining a suture. Whence, the tenuous nature of suture repairs resulting in frequent failures.

Tension

All suture repairs of femoral hernias imply tension. This tension is generated by the architecture of the groin. The area in question is triangular with the base formed by the femoral vein, the rounded apex of this triangle, by the lacunar ligament of Gimbernat, the posterior side of the triangle being the pubic ramus and pectineal ligament while the anterior side is the iliopubic tract of Thomson and inguinal ligament. These structures are fixed rather rigidly and attempts to approximate them effectively will either cause a tear in tissues or impinge on the femoral vein. The latter is a constant if low occurrence in McVay repairs [13, 14]. It is in femoral hernias that meshes have found their most efficient expression. The literature detailing this definite advance in hernia surgery is abundant and is the subject of an entirely different interest.

Conclusion

There is little doubt that femoral hernias are among the most difficult hernias to repair. Certainly the most stressful! How does one go about "creating a femoral hernia". One sure way is to be unfamiliar with anatomy. The other is to insist on a suture repair. No matter how tension-free a suture repair may look and feel, it is only an appearance without substance. One must not succumb to that illusion. The mesh repairs of femoral hernias must avoid the use of gadgets for which there is "no need to know anatomy"! A simple sheet of mesh 6 to 8 cm in diameter (with a suture threaded at its centre if need be) can be inserted by any method that one is most familiar with: infrainguinal, transinguinal, suprapubic or laparoscopic. The net result of the repair should be a preperitoneal position of the mesh.

References

1. Bendavid R. Femoral hernias: why do they recur? Probl Gen Surg 12 (1995) 147–149
2. Koontz A. The disaster of recurrent hernia. Curr Med Digest 29, 1962
3. Obney N, Chan CK. Repair of multiple time recurrent inguinal hernias with reference to common causes of recurrence. Contemp Surg 25 (1984) 25–32
4. Georgievski A. Surgeon-in-chief, Shouldice Hospital (1995–2000). Personal communication (1990)
5. Bendavid R. A femoral "umbrella" for femoral hernia repair. Surg Gynecol Obstetr 165 (1987) 153–156
6. David T. Strangulated femoral hernia. Med J Aust 1 (1967) 256–261
7. Ponka JL, Brush BE. The problem of femoral hernia. Arch Surg 102 (1971) 417–423
8. Bendavid R. The need for mesh. In: Bendavid R (ed) Prosthesis and abdominal wall hernia. Landes Biomedical Publishers, Austin, 1994, pp 116–122
9. Bendavid R. Femoral hernias: primary vs. recurrence. Int Surg 74 (1989) 99–100
10. Xavier H, Bouras-Kara T. Should prostheses be used in emergency hernia surgery? In: Bendavid R (ed) Abdominal wall hernia: principles and management. Springer, New York, 2001, pp 557–559
11. McVay CB. Hernia. The pathologic anatomy and their anatomic repair of the more common hernias. Charles C Thomas, Sprinfield, IL, 1954
12. Fruchaud H. Surgical anatomy of inguinal hernias in the adult, translated and edited by Bendavid R and P. Cunningham; University of Toronto Press (in press)
13. Barbier J, Carretier M, Richer JP. Cooper ligament repair; An update. World J Surg 13 (1989) 499–505
14. Brown R, Kinateder RJ, Rosenberg N. Ipsilateral thrombophlebitisand pulmonary embolism after Cooper's ligament herniorrhaphy. Surgery 87 (1980) 230–232

Discussion

Fitzgibbons: *Do you think that we can reliably differentiate an indirect inguinal hernia from a femoral hernia in a female? The reason I ask this question is because, after publishing the watchful waiting trial showing it was safe to observe men, we specifically excluded women on the basis of the fact you can't reliably make the distinction and they should have immediate operation. We're getting lots of calls from women's societies that we were chauvinists and subjecting these women to surgery: Do you agree with this statement that you can't reliably differentiate an indirect inguinal hernia from a femoral in a female?*

Bendavid: *I have found that differentiating it has been easy most of the time because if you draw a line which is called the Brown line between the anterior superior iliac spin and the pubic crest, obviously the femoral will be below it. It will be much more difficult to differentiate between a direct and an indirect but I have seen situations where the femoral sac is so large that it would actually dissect itself back up so that it feels like either direct or an indirect hernia. From that standpoint you cannot tell them apart: so to answer your question: you cannot tell them with 100% certainty.*

Fitzgibbons: *I personally think it's dangerous to observe any female with a hernia.*

Bendavid: *Well, I agree, I agree. That's a tricky question, though: are you using that on exams?*

Fitzgibbons: *No, not on exams. I questioned myself, that's why it's a personal question.*

Read: *Dr. Little, the great surgical anatomist from England, has presented, as you know, quite a few studies about the surgical anatomy of femoral hernia; his concept was that a femoral hernia doesn't occur until the hernia so-called has passed the exit of the femoral canal as opposed to the entry. Would you comment on that?*
Bendavid: *Well it was a nebulous area. Now I understand that anatomically in fact the funnel does go all the way down to the saphenous opening. It's there, it's been described by many people, interestingly enough the work of Little also was done in the 18th century in France and I found that he derived a lot of his comments from that work; but what happens is the fact that once a hernia does develop and takes on volume, its covering is so thin that it can start bulging before it gets down to the femoral opening, the saphenous opening, and I have personally never seen, perhaps once if I remember, a sac extending all the way. Have you? I feel that the covering of the transversalis fascia is so thin, so distensible, and that you should intervene way before it becomes a problem. I think, theoretically anatomically, he is right.*
Young: *Two comments, one is that ultrasound can be an extremely accurate tool for diagnosis on femoral hernia and we do this in our office very frequently; second point since you are going into that, we have two ways of repairing femoral hernias with PHS which I think might be very relatively straightforward. One is going directly through the opening inessentially opening the underlay on the inside attaching it to the ligamentum anterior or the Coopers's ligament on the outside and then cutting off the overlay, the second way is going through and doing essentially a direct repair but in that case we anchor the underlay to Cooper's ligament just medial to the femoral vein and then the additional portion of the underlay lies down in front as if you had place it in there.*
Bendavid: *That's a lot of invasion but, however…*
Kehlet: *I just want to add some information from the real world in Denmark and an analysis that I will show tomorrow. In more than 2000 femoral hernias the results are terrible. We have a 9% recurrence rate with an observation period over 6 years.*
Bendavid: *Following what kind of technique?*
Kehlet: *All the classical techniques, including the mesh; the laparoscopic technique is half. So I want to ask also the Swedish database, because you published a paper in about 800 patients some years ago, if you can comment on your nationwide results. You didn't mention laparoscopic repair for femoral hernia. Isn't that the ideal technique?*
Bendavid: *Certainly you get to the area and you will cover it. In fact, the laparoscopic surgeons are beginning to report incidences of femoral hernia that are far beyond what was suspected. Some surgeons have even told me that they see it at least 20% of the time but this is why I have commented on the fact that if anything looks like a meniscus, don't disturb it, leave it alone. If you see a sunken lymph knot, leave it alone, leave it in place and don't dissect it because where there was no femoral hernia before you will definitely have one now. I think we have seen it often enough, so one has to be careful.*
Berndsen: *We made an analysis a couple of years ago on 600 femoral hernias, but we couldn't see any differences between the various methods. There was a slight difference in the material in favour of methods using mesh. There were no statistically significant findings.*
Schippers: *Dr. Bendavid, during my surgical education I was taught at least for the inguinal approach to approximate the inguinal ligament and the Cooper ligament in order to close the femoral hernia. Did I understand you right that those structures are not reliable any longer?*
Bendavid: *I don't recommend any suture repair any more. When you see the angle and you see the size of the vein and when you see drawings you cannot avoid tension and I've seen one case of a leg that was terribly reflective of what I'm talking about. Today I think we have to move with the time and I would not recommend any sutured repair. Of course, when you look at the old texts, they said something like you must make sure that you have at least 2 mm between the last suture and the femoral vein. It's a difficult thing to do because don't forget that the patient starts moving and then you have a completely different anatomy and different physiology. The moment the person stands you cannot compare the anatomy even in surgery with a leg outside the table dangling on the side of the table with a bag under the pelvis in order to duplicate the position and the function during the standing posture of the patient. So I'm not so sure, and as I've said, if you can see up to 3 or 4% that's high when you are doing such a benign procedure to end up with such a nasty complication.*
Chan: *I think from the way that we have developed the need to use femoral mesh is by experience in the past – you know until 1986 then we put the mesh in. Before that we knew that once we get femoral hernia recurrent we threw up our hands! Now we can't really repair. So what I mean is, Bendavid, you first put the mesh in and then forget about.*
Kukleta: *I should like to make a comment on anatomy. As laparoscopist I see it a little differently. I agree that one should not remove a lymph knot out of the femoral canal because maybe there is no hernia at all, but I'm not absolutely sure if you're right with the preperitoneal fat. Sometimes when we pull on that 5–7 cm of preperitoneal tissue comes which was the reason for the symptom. We've learned something if we suspect femoral hernia*

and don't have any peritoneal sac, we have to open to make sure that we don't have this preperitoneal tissue in there.

Bendavid: *I would like to disagree with you strongly because we have in fact learned this. There was a time when we did the femoral hernia from below and often we used to find fat tab and it was so easy to actually pull on this fat tab until you got as much of it as possible and resected it and put it in a simple suture and that's all. It would certainly recur as a femoral hernia. The attitude has changed and I think it's fairly convincing that we leave it alone. If you happen to be below you simply don't dissect it, don't put it out. A fat tab is a very effective plug so far and I don't see why you should go looking for trouble. The Americans have a good saying: "If it isn't broken, don't fix it".*

Kukleta: *We do it only for those who are symptomatic and this is the reason why we open there because if it was just diagnostic laparoscopy nobody would ever open the peritoneum to look for fat pads.*

35 The Umbilical Hernia

J. Conze, A. Prescher, M. Schlächter, O. Schumacher

Introduction

In early development a connecting stalk between the caudal end of the embryo and the chorion is established. This stalk contains at the embryonic end a small allantoenteric diverticulum. Furthermore, it contains the umbilical (allantoic) vessels: one umbilical vein and two umbilical arteries and the urachus. It must be mentioned that a small exocoelomic recess is also included in the proximal (embryonic) part of the umbilical stalk. This recessus is also termed umbilical coelom and it is in continuation with the intra-embryonic coelom of the embryo. During the 6th to 10th week of development this umbilical coelom forms a sac, which receives the physiological umbilical hernia of the midgut. After the retraction of the physiological umbilical hernia, the umbilical coelom is usually obliterated and does not further exist. At birth these structures are dispensable, leading to an obliteration of the umbilical cord structures. The following granulation and scarring process typically leads to a fibrotic, collagenous plate characterized by criss-crossing fibre fusion with the neighbouring umbilical ring. According to this complicated development, the definitive umbilicus is a locus minoris resistentiae with a lifelong risk for herniation.

Two main groups of umbilical hernias can be differentiated easily: the infantile umbilical hernias and the adult umbilical hernias. The first group can be derived without any problems from a disturbed development in the umbilical region, where the rectus abdominis muscles fail to approximate in the midline after the retraction of the physiological umbilical hernia. The second group is always an acquired hernial entity.

It is absolutely essential not to confuse the other defects of the anterior abdominal wall (omphalocele, gastrochisis and intussusception at the umbilicus) with an umbilical hernia. An exact terminology and clear definitions are given by Moore and Stokes, so that a precise differential diagnosis can be established [8].

Infantile Umbilical Hernia (Hernia Funiculi Umbilicalis)

Non-fusion of the obliterated umbilical cord structures with the surrounding umbilical ring and disturbances in the closure of the umbilical foramen may lead to protrusion of the peritoneal sac. After hydroceles and inguinal hernias they are the third common surgical disorder in infancy, with an incidence of up to 20% in white children and even up to over 50% in black infants. There seems to exist a familial predisposition of 9–12%. Most often they appear in premature and low-weight newborns.

Beside the obvious protrusion, infantile umbilical hernias rarely enlarge over time or become symptomatic. In up to 90% they even disappear without any surgical action within the first 2 years. The probability of spontaneous closure seems to correlate with defect size. Umbilical hernias with defect diameter of more than 15 mm are unlikely to close spontaneously.

Therefore, the indication for surgical repair should not be made before the age of 2 years. In the case of operation, the typical surgical procedure is a simple single stitch or continuous suture repair with resorbable suture material (Spitzy repair). This can be performed with a short general anaesthesia in a day-care setting.

Adult Umbilical Hernia

In the adult the umbilical hernia are most often acquired. The over-all incidence is approximately 5–6% of all abdominal wall hernias. Typical predispositions are rise of the intra-abdominal pressure, for example in extreme obesity, history of multiple pregnancies, ascites or large intra-abdominal tumours. Contrary to the infantile umbilical hernias, the risk of incarceration is much higher in the adult.

In the literature there is sometimes a differentiation between direct umbilical and para-umbilical hernias, though in clinical practice this remains without effect. Direct hernias appear as a symmetric protrusion with a circumferentially symmetric bulge after yielding of the cicatrix tissue closing the umbilical ring. Direct umbilical hernias result from a persistent elevation of the intra-abdominal pressure. This is typical for patients with ascites formation or peritoneal dialysis. If not as an emergency, a primary therapy of the actuating disease should be aspired before any surgical action in these cases.

In indirect, para-umbilical hernias, the yielding of tissue around the umbilical ring leads to a semicircular protrusion above or below the umbilicus with the naval column building part of the hernia.

Already 2000 years ago Aulus Cornelius Celsus, author of De Medicina described the umbilical hernia as an "indecent prominence of the naval". He suggested a tight constriction of the hernia with flaxen thread and burning the part beyond the ligature with caustics. Today, the surgical armamentarium for umbilical hernia repair has evolved with a broad spectrum of different procedures (Fig. 35.1). As in inguinal or incisional hernia, we can observe the same tendency favouring a repair with mesh prosthesis; but unlike these hernias, the recurrence rates after suture repair are not as desolate.

The suture repair of umbilical hernias can be performed as a single stitch to stitch, or a continuous suture with absorbable or non-absorbable material. In recent publications these conventional techniques reach recurrence rates between 8 and 14% (Table 35.1). Using the Mayo repair, suturing the overlapping fascia downward from above, the results are even better with recurrence rates around 4%. These results appear inconsistent compared to recurrence rates of more than 40% in incisional hernia repair. A possible explanation could be the longitudinal suture direction, with an angle of 90° to the transverse fibre direction of the fascia.

The surgical options for mesh implantation in umbilical hernias are similar to inguinal and incisional hernia repair. So far, there is no final conclusion in terms of technique, material or mesh position, or mesh necessity at all. In the literature the open mesh technique shows recurrences between 0 and 25%, with infection in up to 15% (Table 35.2). Recent descriptions using PHS (Prolene Hernia System) or laparoscopic procedures show promising results, though limited by small numbers and short follow-up (Tables 35.3 and 35.4).

Comparing the different techniques and their results the suture repairs facilitate a success rate in over 90% of the patients with a minimum of costs and a surgical procedure that can be performed in local anaesthesia in an outpatient setting. Mesh repair is more expensive, adding the costs for mesh material and longer opera-

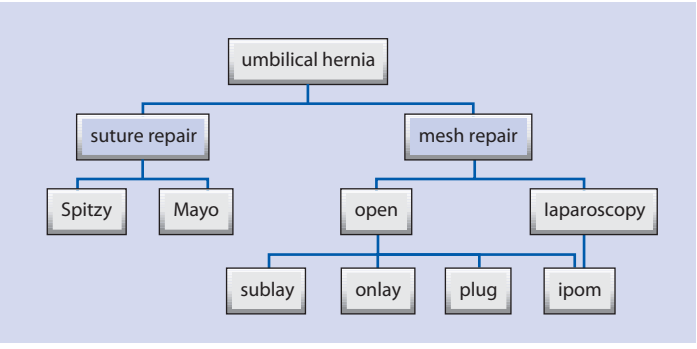


Fig. 35.1. Surgical options for umbilical hernia repair

Table 35.1. Umbilical hernia repair with suture repair (single stitch; continuous suture or Mayo repair)

Author	No.	Follow-up [months]	Technique	Ser.	Inf.	Rec.
Arroyo et al. 2000** [1]	100	64	Suture, non-absorbable	5.5%	3.0%	11.0%
Wright et al. 2002 [13]	66	30	Suture, non-absorbable	9.0%	9.0%	9.0%
Schumacher et al. 2003 [11]	108	30	Suture, absorbable	?	6.5%	13.0%
Gonzales et al. 2003 [3]	24	28	Suture, absorbable	?	?	8.0%
Halm et al. 2005 [4]	98	32	Suture?	?	9.2%	14.3%
Aachen 2006	369	72	Spitzy	14.4%	6.0%	9.7%
Mayo (1901)	75	?	Mayo	?	?	2.6%
Bowley and Kingsnorth 2000	393	25	Mayo	?	?	4.0%
Menon and Brown 2000 [7]	32	24	Mayo, non-absorbable	?	6.0%	0.0%

Table 35.2. Umbilical hernia repair with open mesh techniques

Author	No.	Follow-up [months]	Technique	Ser.	Inf.	Rec.
Bowley and Kingsnorth 2000	80	25	Mesh	?	?	2.5%
Arroyo et al. 2002 [1]	213	64	147 PP-Plug (<3 cm) 70 PP-onlay mesh (>3 cm)	5.6%	1.4%	0.95%
Wright et al. 2002 [13]	20	28	Open mesh (PP) onlay or sublay	15.0%	10.0%	25.0%
Gonzales et. al. 2003 [3]	20	25	Open onlay mesh	40.0%	15.0%	20.0%
Kurzer et al. 2004 [5]	54	43	sublay mesh/plug (PP)	?	12.9%	0.0%
Sinha and Keith 2004 [12]	34	14	Plug (PP)	3.0%	3.0%	3.0%
Halm et al. 2005 [4]	11	32	Sublay mesh (PP)	0.0%	0.0%	0.0%

Table 35.3. Umbilical hernia repair with open Prolene hernia system (PHS)

Author	No.	Follow-up [months]	Technique	Ser.	Inf.	Rec.
Perrakis et al. 2003 [9]	48	13	PHS	2%	0%	0%
Del Pozo and Marin 2003 [2]	14	?	PHS	0%	0%	0%
Polat et al. 2005 [10]	17	22	PHS	6%	6%	0%

Table 35.4. Umbilical hernia repair with laparoscopic IPOM

Author	No.	Follow-up [months]	Technique	Ser.	Inf.	Rec.
Lau and Patil 2003 [6]	26	24	Lap-IPOM (ePTFE)	0%	0%	0%
Wright et al. 2002 [13]	30	23	Lap-IPOM (ePTFE)	10%	3.3%	0%
Gonzales et al. 2003 [3]	32	25	Lap-IPOM (PP or ePTFE)	56%	0%	0%

tion time, plus general anaesthesia for laparoscopic procedures.

In 2003, Schumacher et al. performed a follow-up study after umbilical hernia repair and looked at the possible risk factors for hernia recurrences. They found a significant relationship between recurrence and body mass index (BMI). In patients with a BMI below 30 the recurrence rate was 8.1% compared to 32% recurrences with a BMI above 30% [11]. These findings were recently confirmed by Halm et al. [4].

Another risk factor for hernia recurrence identified by Schumacher et al. was the size of the fascia defect. After suture repair of an umbilical hernia, recurrence occurred significantly more often in patients with fascia defects of more than 3 cm diameter. Excluding the patients at risk (BMI > 30, defect > 3 cm), the suture repair was successful in 96% of all patients. In contrast to incisional hernia repair, the implantation of mesh prosthesis seems to be an overtreatment in most umbilical hernias. Mesh repair should be reserved for patients at risk with a BMI above 30 and a defect diameter of more than 3 cm. In the patients that Schumacher et al. followed up there were 22% at risk, concluding that approximately 80% of all umbilical hernias can therefore be treated successfully with a suture repair and only in 20% would a mesh repair have been indicated. Besides, the ideal technique for umbilical mesh repair has yet to be found. There is no evidence on mesh position, mesh size, mesh material or mesh fixation. Future studies need to investigate the ideal mesh procedure.

References

1. Arroyo A, Garcia P, Perez F, Andreu J, Candela F, Calpena R (2001) Randomized clinical trial comparing suture and mesh repair of umbilical hernia in adults. Br J Surg 88: 1321 1323
2. del Pozo M, Marin P (2003) Three-dimensional mesh for ventral hernias: a new technique for an old problem. Hernia 7: 197 201
3. Gonzalez R, Mason E, Duncan T, Wilson R, Ramshaw BJ (2003) Laparoscopic versus open umbilical hernia repair. JSLS 7: 323 328
4. Halm JA, Heisterkamp J, Veen HF, Weidema WF (2005) Long-term follow-up after umbilical hernia repair: are there risk factors for recurrence after simple and mesh repair. Hernia 9: 334 337
5. Kurzer M, Belsham PA, Kark AE (2004) Tension-free mesh repair of umbilical hernia as a day case using local anaesthesia. Hernia 8: 104 107
6. Lau H, Patil NG (2003) Umbilical hernia in adults. Surg Endosc 17: 2016 2020
7. Menon VS, Brown TH (2003) Umbilical hernia in adults: day case local anaesthetic repair. J Postgrad Med 49: 132 133
8. Moore TC SG (1953) Gastroschisis; report of two cases treated by modification of Gross operation for omphalocele. Surgery 33: 112 120

9. Perrakis E, Velimezis G, Vezakis A, Antoniades J, Savanis G, Patrikakos V (2003) A new tension-free technique for the repair of umbilical hernia, using the Prolene Hernia System early results from 48 cases. Hernia 7: 178 180
10. Polat C, Dervisoglu A, Senyurek G, Bilgin M, Erzurumlu K, Ozkan K (2005) Umbilical hernia repair with the prolene hernia system. Am J Surg 190: 61 64
11. Schumacher OP, Peiper C, Lorken M, Schumpelick V (2003) [Long-term results after Spitzy's umbilical hernia repair]. Chirurg 74: 50 54
12. Sinha SN, Keith T (2004) Mesh plug repair for paraumbilical hernia. Surgeon 2: 99–102
13. Wright BE, Beckerman J, Cohen M, Cumming JK, Rodriguez JL (2002) Is laparoscopic umbilical hernia repair with mesh a reasonable alternative to conventional repair? Am J Surg 184: 505–508

Discussion

Deysine: *Thank you, Dr. Conze. I will tell you that my experience is a bit different. I used to do my umbilical hernia repairs with either a stitch if they were very small or a Mayo repair. My recurrence rate was close to 100%. Then I switched to mesh repair and that improved dramatically. I didn't have another recurrence, but all my little umbilical hernias that I stitched came back.*

Hahn: *I want to raise two points. Magnificent of you, thank you very much, I want to apologize for going ahead and mentioning it already yesterday. I'm very sorry about that. Two things: you mentioned that you went to the financial department and they worked out the price for you and it included 2 days of in-house stay. I think that's very long. I think all patients in The Netherlands in our series were operated in day-care so they were sent home straight away. That's one thing I would like you to comment on. The second is in your overview of possible techniques, I think you left out one maybe little experiment but it's the TAPP procedure of the umbilical hernia with the transabdominal praeperitoneal placement of mesh which is currently practiced by surgeons in The Netherlands. Well, there is no report, but it looks lovely.*

Conze: *Give me some time. I think the problem in Germany is if you look at the numbers that we get from our insurance companies you'd be surprised. I don't know why the Germans love the hospitals, they like to be in hospital and they feel they should stay there until they're really safe and everything is OK. Now 2 days is not overtreatment in Germany, it's usually around 6 days for umbilical hernia. I know you would not survive a stay like this in America, they kill you immediately. Germans are a little different concerning their stay in the hospital. It will change but it takes time. We do this also in outpatient but it's a small number.*

Schumpelick: *Fitzgibbons told us watchful waiting in the hernia business. What about watchful waiting in umbilical hernia? We see a lot of hernias, should we operate them all? What is the indication to operate?*

Conze: *Well, every big hernia was once a small hernia.*

Schumpelick: *Operate every hernia?*

Conze: *Yes.*

Schumpelick: *Is there agreement here? Every seen hernia should be operated? Symptomatic hernia always has to be operated; asymptomatic hernia, what about that?*

Conze: *We should specify. We are not talking about children. We are talking about adults and we are talking about adults without risk factors. Who would operate every umbilical hernia at the age over 25 with a bulge?*

Chowbey: *Well I think we should also keep in mind the possibility of strangulation and obstruction.*

Schumpelick: *Absolutely.*

Chowbey: *The smaller hernias are more notorious to have obstruction.*

Schumpelick: *We all see a lot of patients because of other diseases and you feel the umbilicus and you see small hernia. Should we say you must be operated? No.*

Chowbey: *My question is, why are you debating from the meshes to sutures just for the matter of costs or recurrence when today we are talking about all hernias including the hiatal hernia, we are saying that sutures are practically out. Why are we going back into the umbilical hernia. Prof. Deysine just said that there is a very high recurrence rate when you use just the suture, not the mesh.*

Conze: *The literature shows a little difference in our numbers so we have a success rate of 90% with a follow up of 17 months. That's quite a number. Again, there is the trauma that you cause by placing a mesh in a small defect of 1 cm. We saw the same problem yesterday when we talked about trocar hernias. So the trauma you set to repair this defect with a mesh is far bigger than trying to do a suture repair. But this is not the question I wanted to pose. What I meant to ask is what is a recurrent umbilical hernia? Is it an incisional hernia or is it a recurrent umbilical hernia? Because some papers mix them up; the second question from the anatomical point of view: is a para-umbilical hernia not an epigastric hernia?*

Deysine: *The pathogenesis of an epigastric hernia is totally different, it is located in the linea alba and the pathogenesis has been well described. You may find there is a weakness above the umbilical hernia, that's very common.*

Conze: *I didn't want to get from the track, so back to the size of the defect. If you have an umbilical hernia of 1 cm and you want to place a mesh into it, you certainly have to enlarge the defect to place the mesh, depending on what kind of mesh you take. If you put in a composix*

you might have to enlarge the defect. So I think in these cases you should try to have 90% recurrence free by taking an anterior approach. They say if it's an obese patient with a higher body mass index and a larger defect, no question about it, take a mesh. But I think we need to find the risk factors for people that develop a recurrence after a suture repair. In our view it is the mesh, the size of the fascia defect, and the body mass index that are the important factors at the moment.

Chowbey: *I think also there is a case for laparoscopic repair where you show good results.*

Conze: *Yes.*

Young: *Regarding the DRGs. Our experience in the United States is that as soon as they realize that you are sending those patients home on days with zero days and doing an outpatient that 2000 € will probably very quickly go down to 500, so I don't think that's necessarily the best way.*

Chan: *Most of the time we do umbilical hernia with stitch and we are doing around 200 or 300 a year. When the defect is bigger than 2.5 cm or larger then I will put a mesh in. Otherwise I won't.*

Fitzgibbons: *Just two sentences about the asymptomatic umbilical hernia, and it's only worth two sentences because nobody has any doubt. We have no large population of people who have their umbilical hernias repaired so there is no way to get any natural history of why we have many hernias. The hernia that I refer to: where's a little pulp of defect I would not be concerned, but I am concerned about observing patients that have palpable visible bulges. I think it is dangerous to observe it until we have more data.*

Bendavid: *It's not my personal experience but I'm beginning to read the reports of as high as 20% infection rate with umbilical repairs. I don't use prophylactic antibiotics. Should I? Have you observed any statistics or do you have any statistics on this?*

Conze: *As far as I know there's only one paper from The Netherlands, I think, that used a prophylactic antibiotics, not only for umbilical hernia but also for inguinal or incision hernia. If you take a mesh, certainly I would suggest a single-shot antibiotic and I think more important actually is the cleaning of the umbilicus before you incise, because it's not enough just do a little sponge the night before with iodine. What I always do is after narcosis and after local anaesthesia just take a swab and really clean it to make sure that you don't have problems.*

Bendavid: *You really need to do that 2 or 3 days before hands. Do you use antibiotics for umbilical repair?*

Deysine: *You have to. It's one of the dirtiest places in the human body.*

36 Parastomal Hernia: Prevention and Treatment

L.A. Israelsson

Introduction

Parastomal hernia is a very common complication in gastro-intestinal surgery. Some degree of parastomal herniation has been claimed to be an almost inevitable complication of any ostomy formation [1]. Several surgical techniques have been tried in order to prevent the development of parastomal hernia, but these efforts have not been very successful [2]. Although a great variety of methods for the repair of parastomal hernias have been attempted, very high recurrence rates have been reported [2]. However, new types of prosthetic mesh materials have been developed that offer an opportunity for both the prevention and the treatment of parastomal hernia.

Incidence

Since a uniform definition of parastomal hernia is not used at follow-up it is difficult to compare results between surgical departments. The rate of parastomal hernia has been reported in the range of 5 to 50% [3–17]. Different definitions of parastomal hernia used at follow-up, rather than actual differences in prevalence between surgical centres, probably explains why herniation is reported within this great range. Thus, the very high hernia rates reported during the past decade in studies including evaluation with a CT scan at follow-up probably reflect that also very small parastomal hernias are then detected [9, 10, 18].

Essentially, a parastomal hernia is an incisional hernia related to an abdominal wall stoma [19]. In congruence with incisional hernia- follow-up should in clinical studies not be earlier than after 12 months and any palpable defect or bulge adjacent to the stoma when the patient is supine with elevated legs or erect and coughing or straining should be regarded as a parastomal hernia [2, 20–22]. In general surgical practice, the rate of parastomal hernia is probably between 30 and 50%.

The rate of parastomal hernia is probably similar to an ileostomy and to a colostomy, although a higher rate has been suggested with the latter in some studies [18, 23]. An enterostoma brought out through the laparotomy wound is associated with an extremely high rate of infection, wound dehiscence and herniation [24–27]. To bring out the enterostoma through an extra peritoneal path has not been proved to reduce the rate of parastomal hernia development [9, 18, 28]. Mesenteric fixation has also not been established to decrease the rate of herniation [9].

Enterostomas should probably be brought out through the rectus abdominis muscle since this has, in two clinical reports, been associated with a lower rate of parastomal herniation than if brought out lateral to the muscle [12, 29]. There are other retrospective studies, however, that have not confirmed these findings [9, 10, 18, 28].

Making too big an opening in the abdominal wall for the enterostoma may increase the risk of parastomal

hernia developing but the proper size of the abdominal opening is, of course, difficult to standardize in the clinical setting [11, 24, 27, 30–32]. Old age, obesity, chronic respiratory disorders, malnutrition, corticosteroid use and wound infection have been suggested as risk factors for the development of parastomal hernia [24, 28, 33–35].

Surgical Treatment

In 15 to 70% of patients with a parastomal hernia, surgical repair seems to be demanded [4, 12, 18, 30]. Local aponeurotic repair is not an acceptable method of repair since recurrence rates between 50 and 76% are reported with this method [24, 32, 33, 35, 36]. Relocating the stoma into another quadrant of the abdominal wall has also produced very high recurrence rates, reported in some studies to be as high as 76% [2, 33, 35, 37, 38]. When relocating the bowel, the defect in the abdominal wall at the index ostomy also presents a problem, since it is often very large and must be repaired as an incisional hernia [2, 39, 40]. An incisional hernia may develop at the original enterostoma site in 52% of patients [35]. If the first attempt to treat a parastomal hernia fails, the recurrence rate after the following procedures increases dramatically [35].

The best results in parastomal hernia repair have been reported with the use of a prosthetic mesh. A non-absorbable mesh can be placed in either a sublay [41–44] or an onlay position [31, 34, 45–49]. Randomized studies are not available, but with prosthetic mesh repair of parastomal hernias, lower recurrence rates have been reported than with other methods of repair [32, 35, 41, 45, 50].

Prevention

Placing a prosthetic mesh adjacent to the bowel may be associated with the development of fistulas, adhesions or strictures. The rate of complications has been very high without peritoneum interposed between the prosthetic mesh and abdominal visceral contents [51]. However, meshes with a large pore size of about 5 mm with a reduced polypropylene content and a high proportion of absorbable material have been available for several years (Vypro, Ultrapro, Ethicon, Norderstedt, Germany). These meshes are associated with a lesser degree of inflammation in the vicinity of the mesh [52]. Such meshes have been used for the repair of large parastomal hernias, and with a modest grade of inflammation, the tendency of the mesh eroding into bowel has been suggested to be diminished [41].

These low-weight meshes can be utilized to prevent the development of parastomal hernia. In a clinical study 54 patients were randomized to either a conventional enterostomy through the rectus abdominis muscle or to the same procedure with the addition of a low-weight mesh placed in a sublay position. The mesh was not associated with infection or other early complications and at 12-month follow-up the rate of parastomal hernia was significantly lower with a mesh (5 vs. 50%) [53, 54].

Placing a low-weight mesh with a reduced polypropylene content and a high proportion of absorbable material in a sublay position at the primary operation is as yet the only method that has significantly reduced the rate of parastomal hernia in a randomized study. No adverse effects have been reported so far, but late effects cannot be ruled out before long term follow-up is completed, and a multicentre study confirming the results is, of course, desirable.

Considering the obvious similarities between incisional hernia and an enterostoma, it is perhaps not surprising that the path towards reducing the rate of parastomal hernia seems to include a mesh at the primary operation. With both entities abdominal contents protrude through a defect in the abdominal wall – in the first case due to defect wound healing and in the second as an inevitable consequence of stoma formation. If enterostomas are regarded as deliberately formed incisional hernias they should in consequence primarily be treated as an incisional hernia – that is with a sublay mesh.

Technique for a Prophylactic Mesh

The abdominal cavity is accessed through a midline incision. After dissection, the bowel intended to be brought out as an enterostoma is divided with a linear cutting stapler. At the spot marked for the stoma the skin is grasped with a clamp and a circular excision of the skin is made. After subcutaneous dissection, a cross-incision is made in the anterior rectus sheath, which should not be larger than just to let the bowel pass through (◘ Fig. 36.1).

Corresponding to the stoma site, peritoneum and the posterior rectus sheath are opened along the midline for a length of 10 to 15 cm (◘ Fig. 36.2). Dorsal to the rectus muscle dissection is then easily continued to the lateral border of the muscle, since it is an almost avascular plane. A certain caution is required to not sever the epigastric vessels under the rectus muscle. Through

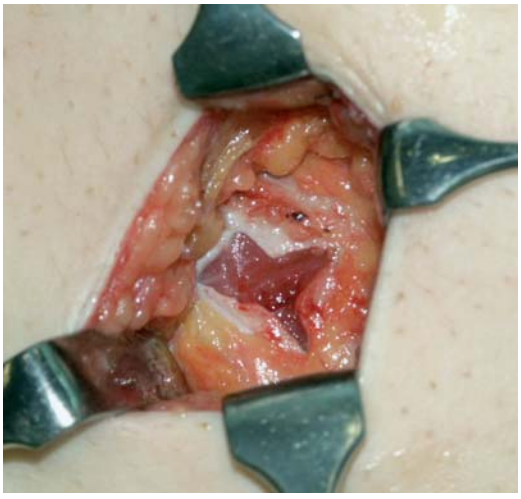


Fig. 36.1. After circular excision of the skin and subcutaneous dissection, a cross-incision is made in the anterior rectus sheath. The incision should not be larger than just to let the bowel pass through

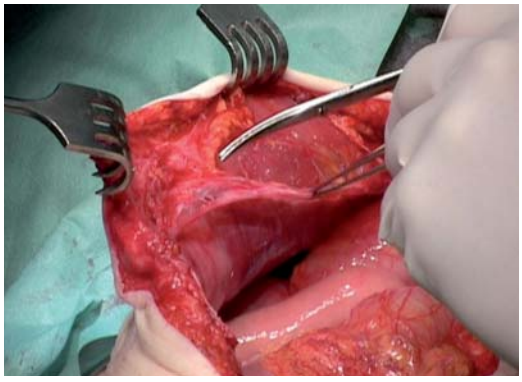


Fig. 36.2. Corresponding to the stoma site, peritoneum and the posterior rectus sheath are opened along the midline for a length of at least 10 cm. Dorsal to the rectus muscle dissection is then continued to the lateral border of the muscle

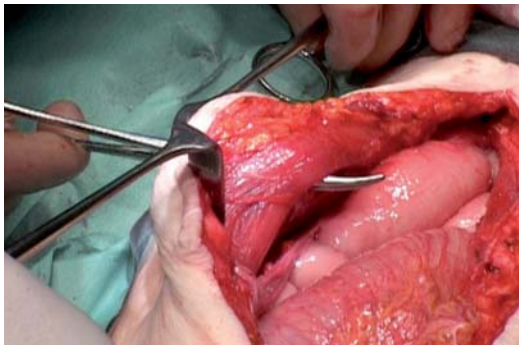


Fig. 36.3. Through the stoma opening in the skin and the anterior rectus sheath a clamp splits an opening through the centre of the rectus muscle for the bowel to be brought out later

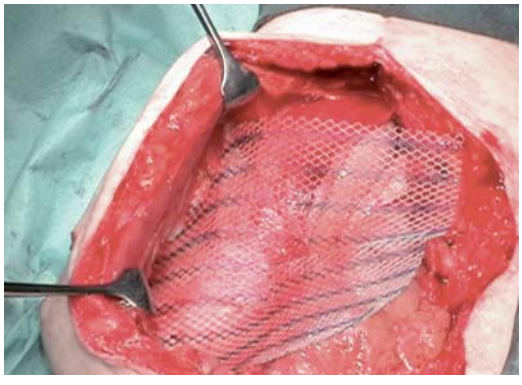


Fig. 36.4. A partly absorbable low-weight mesh 10x10 cm with a cross cut open in its centre is placed in the retro-muscular plane created. The upper and lower lateral corners of the mesh must be anchored to the dorsal rectus sheath with a single stitch. Here to be placed corresponding to the tip of the retractors

the stoma opening in the skin a clamp then splits an opening through the centre of the rectus muscle, for the bowel to be brought out at a later stage (Fig. 36.3).

A partly absorbable low-weight mesh (Vypro or Ultrapro) is used (Fig. 36.4). The mesh should be 10×10 cm. The mesh is foiled and a cross is cut in its centre. The opening should not be made larger than just to let the bowel pass through. The mesh is then placed in the retro-muscular plane created. The upper and lower lateral corners of the mesh must be anchored to the dorsal rectus sheath with a single stitch, using an absorbable monofilament suture.

Peritoneum and the dorsal rectus sheath are opened at the intended stoma site. Firstly, the bowel is brought out through the opening made in the dorsal rectus sheath (Fig. 36.5). Then the bowel is brought out through the opening cut out in the mesh (Fig. 36.6). The length of the bowel and the size of the opening in the mesh can then be checked. Lastly, the bowel is brought out through the rectus muscle and the skin (Fig. 36.7).

The anterior rectus aponeurosis is closed by a continuous suture technique with a slowly absorbable or non-absorbable monofilament suture. The upper and lower medial corners of the mesh must also be anchored (Fig. 36.8). This is achieved by the running stitch closing the aponeurosis also incorporating peritoneum and the mesh, at the medial corners of the mesh. Along the

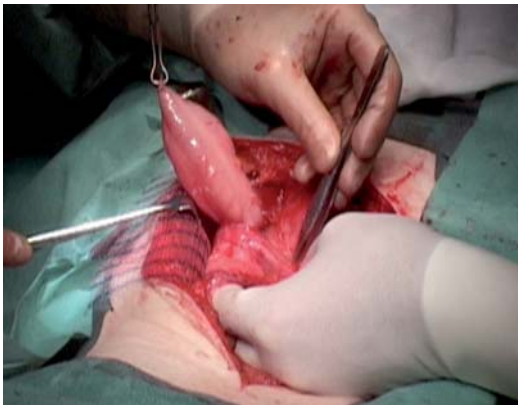


Fig. 36.5. The bowel, that has previously been divided with a cutting stapler, is first brought out through the dorsal rectus sheath

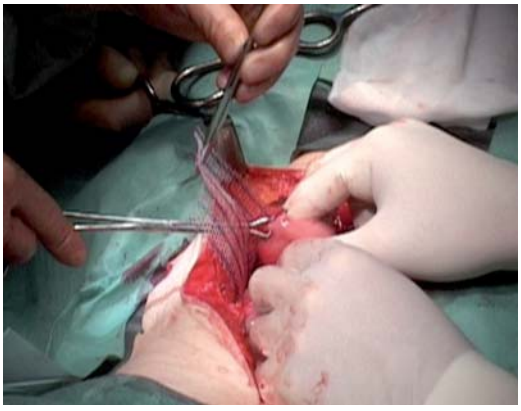


Fig. 36.6. The bowel is then brought out through the opening cut out in the mesh. The length of the bowel and the size of the opening in the mesh can then be checked

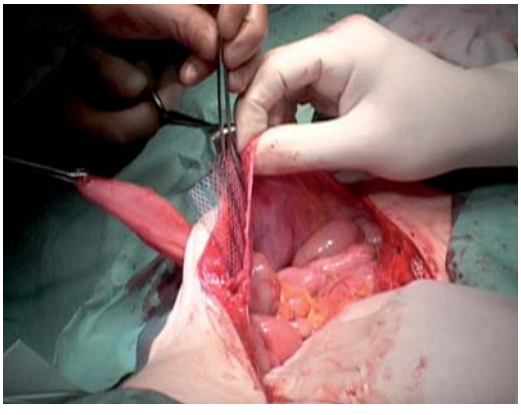


Fig. 36.7. Lastly, the bowel is brought out through the rectus muscle and the skin. Here, the bowel can be seen passing through the posterior rectus sheath, the mesh, the rectus muscle, the anterior rectus sheath and skin

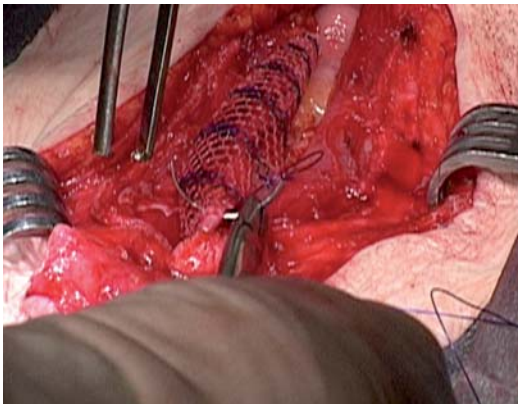


Fig. 36.8. The upper and lower medial corners of the mesh must also be anchored. This is achieved by a stitch of the running suture closing the aponeurosis in the midline also incorporating peritoneum and the mesh. Along the mesh at least every second stitch in the aponeurosis should also include peritoneum to prevent the mesh from coming into contact with visceral contents

mesh at least every second stitch in the aponeurosis should also include peritoneum. This makes certain that bowel does not come into contact with the mesh. The mesh can also be included in these stitches.

Relocation with a Mesh

The defect in the abdominal wall at the site of the parastomal hernia may be very large. That the enterostoma is brought out through a large defect in the abdominal wall complicates the repair of parastomal hernias. Also the risk of incisional hernia is increased when an abdominal incision is re-entered [55] and parastomal hernias are often in proximity to the midline or a concomitant incisional hernia is present. When an enterostoma is relocated into another quadrant of the abdominal wall, the incisional hernia at the primary site and at the celiotomy incision can be repaired in a standardized way with a mesh in a sublay position [21].

Unfortunately, the risk of a parastomal hernia developing at the new site is even higher after stoma

relocation than after the primary enterostoma [33, 35, 37,38]. However, with a prophylactic mesh at the stoma site it may be possible to considerably reduce the risk of parastomal hernia developing at the new site. Thus, relocation of the ostomy into another quadrant with a prophylactic mesh at the new site in combination with a sublay mesh repair at the primary enterostoma and of a concomitant incisional hernia may be a logical way of treating parastomal hernia.

This way of treating parastomal hernias has been used routinely at our department for some years. Although the number of patients treated as of yet is less than 40, it is in our experience a technically rather easy procedure that can be done in a standardized way. The rate of early complications has been low, as has the rate of recurrence in those patients having had a 12-month follow-up [40]. These results must, of course, be confirmed in long-term follow-up and in larger clinical series.

References

1. Goligher JC (1984) Surgery of the anus, colon and rectum (5th edn.). Bailliére Tindall: London; pp 703–704
2. Carne PW, Robertson GM, Frizelle FA (2003) Parastomal hernia. Br J Surg 90: 784–793
3. Birnbaum W, Ferrier P (1952) Complications of abdominal colostomy. Am J Surg 83: 64–67
4. Burns FJ (1970) Complications of colostomy. Dis Colon Rectum 13(6): 448–450
5. Abrams BL, Alsikafi FH, Waterman NG (1979) Colostomy: a new look at morbidity and mortality. Am Surg 45(7): 462–464
6. Burgess P, Matthew VV, Devlin HB (1984) A review of terminal colostomy complications following abdominoperineal resection for carcinoma. Br J Surg 71: 1004
7. Cevese PG, D'Amico DF, Biasiato R, Frego MG, Tropea A, Giaconi MA, et al. (1984) Peristomal hernia following endcolostomy: a conservative approach. Ital J Surg Sci 14(3): 207–209
8. Cheung MT (1995) Complications of an abdominal stoma: an analysis of 322 stomas. Aust N Z J Surg 65(11): 808–811
9. Londono-Schimmer EE, Leong AP, Phillips RK (1994) Life table analysis of stomal complications following colostomy. Dis Colon Rectum 37(9): 916–920
10. Ortiz H, Sara MJ, Armendariz P, de Miguel M, Marti J, Chocarro C (1994) Does the frequency of paracolostomy hernias depend on the position of the colostomy in the abdominal wall? Int J Colorectal Dis 9(2): 65–67
11. Pearl RK, Prasad ML, Orsay CP, Abcarian H, Tan AB, Melzl MT (1985) Early local complications from intestinal stomas. Arch Surg 120(10): 1145–1147
12. Sjodahl R, Anderberg B, Bolin T (1988) Parastomal hernia in relation to site of the abdominal stoma. Br J Surg 75(4): 339–341
13. Baslev A (1973) Kolostomitilvaerelse. Ugeskr Laeger 135: 2799–2804
14. Stelzner S, Hellmich G, Ludvid K (1999) Die Versorgung der Parakolostomiehernie nach Sugarbaker. Zbl Chir 124 (Suppl 2): 13–17
15. Makela JT, Turko PH, Laitenen ST (1997) Analysis of late stomal complications following ostomy surgery. Ann Chir Gynaecol 86(4): 305–310
16. Everingham L (1998) The parastomal hernia dilemma. World Council of Enterostomal Therapists Journal 18: 32–34
17. Tretbar L (1988) Kirurgi vid stomikomplikationer. Stomijournalen: nordisk tidskrift för stomi vård 2(4): 10–11
18. Williams JG, Etherington R, Hayward MW, Hughes LE (1990) Paraileostomy hernia: a clinical and radiological study. Br J Surg 77(12): 1355–1357
19. Pearl RK (1989) Parastomal hernias. World J Surg 13: 569–572
20. Abcarian H (1995) Peristomal hernias. Igaku-Shoin, New York
21. Cengiz Y, Israelsson LA (2003) Parastomal hernia. Eur Surg 35: 28–31
22. Cengiz Y, Israelsson LA (1998) Incisional hernias in midline incisions: an eight-year follow up. Hernia 2: 175–177
23. Marshall FF, Leadbetter WF, Dretler SP (1975) Ileal conduit parastomal hernias. J Urol 114(1): 40–42
24. Goligher JC (1980) Surgery of the anus, rectum and colon. 4 ed. Bailliere Tindall, London
25. Hulten L, Kewenter J, Kock NG (1976) [Complications of ileostomy and colostomy and their treatment]. Chirurg 47(1): 16–21
26. Pearl RK, Prasad ML, Orsay CP, Abcarian H, Tan AB (1988) A survey of technical considerations in the construction of intestinal stomas. Ann Surg 51: 462–465
27. Todd IP (1978) Intestinal stomas. Alden Press, Oxford, London
28. Marks CG, Ritchie JK (1975) The complications of synchronous combined excision for adenocarcinoma of the rectum at St Mark's Hospital. Br J Surg 62(11): 901–905
29. Eldrup J, Wied U, Bishoff N, Moller-Pedersen V (1982) Parakolostomihernier. Incidens og relation till stomiens placering. Ugeskr Laeger 144: 3742–3743
30. Kronberg O, Kramhohft J, Backer O, Sprechler M (1974) Late complications following operations for cancer of the rectum and anus. Dis Colon Rectum 17: 750
31. de Ruiter P, Bijnen AB (1992) Successful local repair of paracolostomy hernia with a newly developed prosthetic device. Int J Colorectal Dis 7(3): 132–134
32. Martin L, Foster G (1996) Parastomal hernia. Ann R Coll Surg Engl 78(2): 81–84
33. Devlin HB, Kingsnorth AN (1998) Parastomal hernia. In: Devlin HB, Kingsnoth A (eds). Management of abdominal hernias, 2nd edn. Devlin, London, pp 257–266
34. Leslie D (1984) The parastomal hernia. Surg Clin North Am 64(2): 407–415
35. Rubin MS, Schoetz DJ, Jr., Matthews JB (1994) Parastomal hernia. Is stoma relocation superior to fascial repair? Arch Surg 129(4): 413–418
36. Horgan K, Hughes LE (1986) Para-ileostomy hernia: failure of a local repair technique. Br J Surg 73(6): 439–440
37. Pearl RK, Sone JH (2002) Management of peristomal hernia: techniques of repair. In: Fitzgibbons RJ, Greenburg AG (eds) Nyhus and Condon's Hernia, 5th edn. Lippincott Williams & Wilkins, Philadelphia, pp 415–422

38. Allen-Mersh TG, Thomson JP (1988) Surgical treatment of colostomy complications. Br J Surg 75(5): 416–418
39. Cassar K, Munro A (2002) Surgical treatment of incisional hernia. Br J Surg 89: 534–545
40. Israelsson LA (2005) Preventing and treating parastomal hernia. World J Surg 29: 1086–1089
41. Kasperk R, Klinge U, Schumpelick V (2000) The repair of large parastomal hernias using a midline approach and a prosthetic mesh in the sublay position. Am J Surg 179: 186–188
42. Rives J, Lardennois B, Flament JB, Hibon J (1971) [The utilisation of a dacron material in the treatment of hernias of the groin]. Acta Chir Belg 70(3): 284–286
43. Rives J, Pire JC, Flament JB, Palot JP, Body C (1985) [Treatment of large eventrations. New therapeutic indications apropos of 322 cases]. Chirurgie 111(3): 215–225
44. Stoppa R, Petit J, Abourachid H, Henry X, Duclaye C, Monchaux G, et al. (1973) [Original procedure of groin hernia repair: interposition without fixation of Dacron tulle prosthesis by subperitoneal median approach]. Chirurgie 99: 119–123
45. Stephenson BM, Phillips RK (1995) Parastomal hernia: local resiting and mesh repair. Br J Surg 82(10): 1395–1396
46. Rosin JD, Bonardi RA (1977) Paracolostomy hernia repair with Marlex mesh: a new technique. Dis Colon Rectum 20:299–302
47. Abdu RA (1982) Repair of paracolostomy hernias with Marlex mesh. Dis Colon Rectum 25(6): 529–531
48. Venditti D, Gargiani M, Milito G (2001) Parastomal hernia surgery: personal experience with use of polypropylene mesh. Tech Coloproctol 5(2): 85–88
49. Bayer I, Kyzer S, Chaimoff C (1986) A new approach to primary strengthening of colostomy with Marlex mesh to prevent paracolostomy hernia. Surg Gynecol Obstet 163: 579–580
50. Amin SN, Armitage NC, Abercrombie JF, Scholefield JH (2001) Lateral repair of parastomal hernia. Ann R Coll Surg Engl 83(3): 206–208
51. Morris-Stiff G, Hughes LE (1998) The continuing challenge of parastomal hernia: failure of a novel polypropylene mesh repair. Ann R Coll Surg Engl 80(3): 184–187
52. Schumpelick V, Klosterhafen B, M Iler M, Klinge U (1999) Minimized polypropylene meshes for preperitoneal mesh plasty in incisional hernia. Chirurg 70: 422–430
53. Jänes A, Cengiz Y, Israelsson LA (2004) Randomized clinical trial of the use of a prosthetic mesh to prevent parastomal hernia. Br J Surg 91: 280–282
54. Jänes A, Cengiz Y, Israelsson LA (2004) Preventing parastomal hernia with a prosthetic mesh: a randomized study. Arch Surg 139: 1356–1358
55. Lamont PM, Ellis H (1988) Incisional hernia in re-opened abdominal incisions: an overlooked risk factor. Br J Surg 75: 374–376

Discussion

Deysine: *My question really is: Do you take any anti-infection precautions like of locally irrigation intravenous antibiotics because you have a contaminated wound?*
Israelsson: *We follow standard procedure. We have Tetracycline and Metronidazole orally before operation at one instance and nothing else.*
Deysine: *There is a bowel prep?*
Israelsson: *No.*
Deysine: *No? Just systemic antibiotic?*
Israelsson: *Yes. Orally.*
Deysine: *And nothing else? And you haven't had any infections?*
Israelsson: *We've had wound infections, we've had intra-abdominal abscesses as usual, but we have not had any infection of a mesh, none, all meshes are still in place.*
Köckerling: *I agree with your observation. This is an advantage of the new light-weight polypropylene meshes with large pores. We also apply these new mesh types into infected areas, for example in incarcerated hernias or something like that, and it works. The risk of infection of the mesh is reduced dramatically due to the large pores because this makes the way free for cellular reaction against material.*
Deysine: *Actually, the diameter of the fibre has little to do with the addition of the bacteria to the fibres: I presume that your technique is very pure and very delicate and that has helped the infection rate, but it is a contaminated wound and I have to congratulate you for not having any significant infection of the mesh.*
Israelsson: *Today we place a prophylactic mesh in all enterostomas, that means that we place a mesh even if we have faecal peritonitis and we still haven't had any infection of the mesh. Strange, yes, but it is a fact.*
Schumpelick: *The point is that we have meshes integrated by sound and healthy tissue. The pores are 3 mm, that means around the fibres – monofilament fibres – there is enough space to fight infection. That makes the difference to the old meshes.*

37 Central Mesh Rupture – Myth or Real Concern?

E. Schippers

Introduction

The use of meshes in repairing incisional hernia first described by Usher [1] in 1958 is now widely accepted. The reinforcement of the wall with meshes led to a satisfactory reduction of recurrence rates of less than 10%. This effect was due to the mechanical properties of the mesh and the induced scar acting as a scar-mesh compound. However, with increasing numbers of implanted meshes, reports of undesirable complications arose as well. These mesh-related consequences included infection, seroma or shrinkage of the mesh [2]. Even the potential risk of malignant transformation due to a persistent foreign-body reaction was a concern. During the third Suvretta meeting in 2003, benefits and potential risks of meshes were discussed in all variants. The take-home message at that time was that with the recently developed meshes no real concern remained. Meantime, in the past 3 years this optimistic view for mesh implants has been spoiled by alarming reports on recurrence rates in incisional hernia repair. Flum [3] reported 10,822 patients operated on for incisional hernia by either suture or mesh repair. It was a retrospective population-based cohort study in Washington State. The recurrence rate over 4000 days reveals an almost linear curve for both repairs. In comparison, a percentage of 20% re-operations occurred after approximately 3500 days after suture repair and 4000 days after mesh repair. The introduction of meshes for the repair of incisional hernia only delayed the re-operation. Furthermore, Burger [4] updated the follow-up of a randomized control trial of suture versus mesh repair of incisional hernia in 2003. He found a 10-year cumulative rate for recurrence of 63% after suture repair as expected. Surprisingly, the 10-year cumulative rate for the mesh repair was 32%. So the results of mesh repair are disappointing as well in the long run. Although the rate of recurrences after mesh repair is well documented in the literature, the reasons in detail for recurrence are not mentioned, or data are rarely available (◘ Table 37.1). Main causes were recurrences cranial, caudal or lateral from the mesh [5, 6] an inadequate overlapping [7, 8] and infection or loosening [9]. Morris-Stiff [10] analyzed in 1998 the outcomes of non-absorbable mesh placed within the abdominal cavity in a literature review and from clinical experience. In his conclusion he stated that all recurrences have been reported as occurring lateral to the mesh and no cases of mesh weakness were identified for any of the materials.

Rumours

In contradiction to this clear statement, rumours between surgeons occurred that central mesh rupture has been observed. So Morris-Stiff reported in 1999 a central mesh rupture in a patient after Marlex mesh repair of an incisional hernia at the incisional hernia symposium in Aachen [11]. However, he did not publish the case since the mesh had been resterilized, against the recommendations of the manufactures. Next, rumours appeared during an investigator meeting in London in

Table 37.1. Cause of recurrence after open incisional hernia repair with prosthetic mesh

Year	First author	No.	Technique	Material	Cause/location
1997	McLanahan [5]	3	Sublay	PP	Upper/lower edge
1991	Molloy [6]	4	Onlay	PP	Lateral edge
1999	Schumpelick [7]	5	In-/sublay	PP	Inadequate, overlapping
1997	Vestweber [8]	2	Onlay	PP	Inadequate, overlapping
1999	Wantz [9]	3	Sublay	Polyester	Infection/loosening

2003. A randomized clinical trial comparing a standard mesh with new light-weight meshes in patients undergoing incisional hernia repair was finished. Discussing the recurrences in this trial in detail, three drawings of the intra-operative findings during re-operation clearly indicated recurrences in the middle of the formerly implanted mesh (Fig. 37.1). After a clinical follow-up of 24 months, the final evaluation of the randomized clinical trial revealed similar outcomes for light-weight composite mesh to polypropylene or polyester mesh with the exception of a non-significant trend towards increased hernia recurrence. Those recurrences were related to the surgical technique. In particular, problems in achieving sufficient mesh coverage, suture technique, material for mesh fixation and closure of the anterior fascia were discussed. Central mesh rupture as documented during the investigator meeting was not mentioned in the final publication [12].

Facts

Beside those rumours which occurred and disappeared after a given period of time, the first description of a central mesh recurrence was published 2001 by Langer [11]. He observed a central recurrence after incisional hernia repair with mesh. The repair was performed in a patient with a BMI of 35 using a Marlex mesh in sublay position without complete closure of the anterior rectus fascia. Ultrasound examination displayed a central mesh defect of 3.5×3 cm in accordance with the palpable mass. Sagittal and transverse section during MRI investigation pre-operatively documented a 3.8 cm-central mesh defect with visible surrounding biomesh structures. Intra-operatively, a central defect of at least 3 cm was confirmed and photographically proved. A second central mesh rupture after Marlex mesh repair was reported from the same group [13].

Fig. 37.1. Analysis of recurrences during the investigator meeting of the multicentre trial. Postoperative drawing indicating formerly mesh position and location of the defect during re-operation

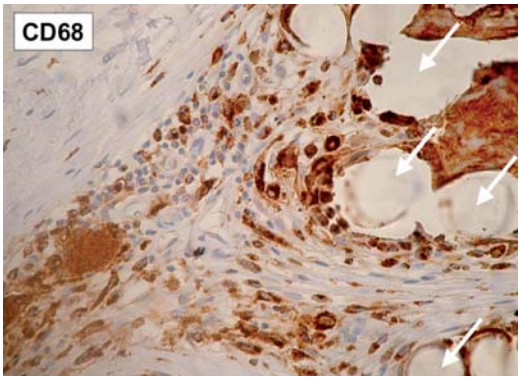


Fig. 37.2. Typical histological finding from implanted mesh: CD 68 positive macrophages (*brown*) infiltrating the foreign body granuloma, mesh filaments indicated by *white arrows*. Biopsies were taken from the edges of the central defect

Furthermore, Klinge [14] reported in 2005 three cases of abdominal wall defects the recurrent hernia passing through the mesh.

In our own, series a 64-year-old female patient presented with clinical signs of recurrence following three consecutive midline incisional hernias. The last repair was carried out using a Vypro mesh (17×30 cm) in the sublay position with complete closure of the anterior rectus fascia. Physical examination of the patient (BMI 33) led to the discovery of a palpable mass in the midline in the supra-umbilical region at the side of the former repair. Ultrasound examination displayed a central defect of (2.5×3×4 cm). Intra-operatively, a supra-umbilical recurrence in the midline with a diameter of minimum 2 cm was found. A sharp dissection was necessary to identify the Vypro mesh, which was well incorporated and almost invisible. In order to prove the rupture in the centre of the mesh, biopsies were taken all around the defect edges. The defect was closed with a running non-absorbable suture. A new Vypro mesh (18×20 cm) widely (5 cm) overlapping the former defect was attached to the first one using interrupted non-absorbable sutures. Histological examination confirmed mesh filaments in all biopsies and proved the central mesh rupture (Fig. 37.2).

Meanwhile, two surgical centres (Aachen/Lyon) specialized on hernia surgery documented four patients (2/2) with central mesh rupture after incisional hernia repair (personal communication J. Conze, 2006; J.B. Flament 2006). All four patients had a repair with Vypro mesh in sublay position and a pathological BMI (34–44). Fascia was closed in two patients during the primary repair (Table 37.2).

Discussion

Despite the superiority of mesh compared to simple suture repair of incisional hernia it has been proven that the ghost of recurrence did not totally disappear in this field of surgery. Central mesh rupture appeared as a rumour, was discussed as a myth and is now documented reality. Pathomechanism for recurrences in hernia surgery usually are related to patient characteristics, speci-

Table 37.2. Documented cases of central mesh rupture. Characteristics of patients, mesh and operative technique

Year	First author	No.	BMI	Mesh	Position	Fascia closed
2001	Langer [11, 13]	2	>35	Marlex	Sublay	No
			>35	Marlex	Sublay	No
2006	Schippers	1	33	Vypro	Sublay	Yes
2006	Conze [15]	2	37	Vypro	Sublay	No
			44	Vypro	Sublay	No
2006	Flament [16]	2	34	Vypro	Sublay	Yes
			40	Vypro	Sublay	Yes

fication of the implants and to the surgical technique. Analyzing the available data in seven documented patients (Table 37.2), no general conclusion on possible pathomechanism can be drawn. As far as patient-related factors are concerned, a corresponding finding in all patients was a pathological (> 26) BMI which varied between 33 and 44. With respect to the implanted material, we have to realize that it was polypropylene in all patients. Pore size and weight of the meshes were different. After Marlex, a heavy-weight mesh a central mesh rupture was documented two times. There is no doubt that these meshes are theoretically strong enough with regard to their tensile strength in comparison with the physiological forces of the abdominal wall. In the case of the rectus muscles, widely separated as mechanism for central mesh rupture, the imbalance between the elasticity of the especially stiff Marlex mesh and the greater elasticity of the abdominal wall at the transitional zone of the fixed and mobile parts of the mesh has been discussed [11].

The conclusion was that heavy meshes consisting of a large amount of biomaterial are in greater danger of being damaged than the light, more flexible, variants. Five of the seven documented patients had a light-weight mesh implanted. Klinge [14] postulated, in cases of extended abdominal wall defects and failure to achieve closure of the fascia in front, mesh materials with a tensile strength of > 32 N/cm must be implanted to avoid mesh rupture. Large-pore meshes with a tensile strength of 16 N/cm are insufficient in such cases if used as a single layer.

As contradictory as the role of mesh weight in the genesis of mesh rupture is the role of closure of the fasica above the implanted mesh in the documented cases. The position of all meshes was sublay, beneath the rectus muscle. Fascia was closed in the midline above in three patients. In four patients, closure of fascia was not achieved during primary repair. If the failed closure of fascia as a potential cause of central mesh rupture is seriously discussed, a great number of central mesh ruptures after laparoscopic procedures for incisional hernia have to be expected. Due to the laparoscopic technique, in none of these patients will the fascia be closed over the implanted mesh.

In conclusion, central mesh rupture is no longer a myth, it is reality. Data available in the literature are rare. Beside pathological BMI, no general risk factors are obvious up to now. Experimental studies have to clarify the role of closure of the fascia and the right material with respect to weight and pore size, especially for patients with risk factors such as pathological BMI.

References

1. Usher F, Öchsner J, Tuttle LL Jr (1958) Use of Marlex mesh in the repair of incisional hernias. Am Surg 24: 969–974
2. Leber GE, Garb JL, Alexander AI, et al (1998) Long-term complications associated with prosthetic repair of incisional hernias. Arch Surg 133: 378–382
3. Flum DR, Horvath K, Koepsell T (2003) Have outcomes of incisional hernia repair improved with time? A population-based analysis. Ann Surg 237: 129–135
4. Burger JW, 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 204: 578–585
5. McLanahan D, King LT, Weems C, Novotney M, Gibson K (1997) Retrorectus prosthetic mesh repair of midline abdominal hernia. Am J Surg 173: 445–449
6. Molloy RG, Moran KT, Waldron RP, Brady MP, Kirwan WO (1991) Massive incisional hernia: abdominal wall replacement with Marlex mesh. Br J Surg 78: 242–244
7. Schumpelick V, Klinge U, Welty G, Klosterhalfen B (1999) Meshes in der Bauchwand. Chirurg 70: 876–887
8. Vestweber KH, Lepique F, Haaf F, Horatz M, Rink A (1997) Netzplastiken bei Bauchwand-Rezidivhernien – Ergebnisse. Zentralbl Chir 122: 885–888
9. Wantz GE, Fischer E (1999) Prosthetic incisional hernioplasty: indications and results. In: Schumpelick V, Kingsnoth AN (eds) Incisional hernia. Springer, Berlin Heidelberg New York, pp 303–311
10. Morris-Stiff GJ, Hughes LE (1998) The outcomes of non-absorblable mesh placed within the abdominal cavity: literature review and clinical experience. J Am Coll Surg 186: 352–366
11. Langer C, Neufang T, Kley C, Liersch T, Becker H (2001) Central mesh recurrence after incisional hernia repair with Marlex – are the meshes strong enough? Hernia 5: 164–167
12. Conze J, Kingsnorth AN, Flament JB, Simmermacher R, Arlt G, Langer C, Schippers E, Hartley M, Schumpelick V (2005) Randomized clinical trial comparing lightweight composite mesh with polyester or polypropylene mesh for incisional hernia repair. Br J Surg; 92: 1488–1493
13. Langer C, Kley C, Neufang T, Liersch T, Becker H (2001) Zur Problematik des Narbenhernienrezidivs nach Netzplastik der Bauchwand. Chirurg 72: 927–933
14. Klinge U, Conze J, Krones CJ, Schumpelick V (2005) Incisional hernia: open techniques. World J. Surg. 29, 1006–1072

Discussion

Miserez: *Congratulation! For me, this was the best lecture of the meeting so far. I think we should go further and I wonder if with this lecture we should not be very very cautious in the new book on the use of Vypro in large hernias where we cannot close the defect. What is your opinion? I think the general surgical community should take notice from this group of experts.*

Schippers: *What I would conclude is that a problem exists; if we use meshes we have to be aware of the problem that*

they might rupture. I cannot go so far as to say we have to beware of light meshes because we had heavy meshes with the same problem and I cannot say it is the closure of the fascia up to now because we have had it in patients with closure and in patients without. The only conclusion we can make at the moment is to be aware that it might happen and might be a cause of recurrence, and we have to do further mechanical studies, which kind of mesh, and maybe we have to diversify in the kind of patient and indication which kind of mesh we have to use in the future. But to give any recommendation I think it's too early.

Deysine: *We are supposed to conclude with some conclusions. After listening to all these speakers today my conclusion is that we could have a complete conference on every one of the subjects we touched. We have increased the amount of knowledge and the amount of questions that came out on every subject and that will keep us busy for a long time.*

Chowbey: *I think when we talked about other types of hernia, it appears that pediatric hernia is also gaining importance and there are expected recurrence rates and it seems that it can all be handled either by conventional or laparoscopic surgery. Also the femoral hernias are fairly common, maybe a little more common than is generally expected, and this should be handled with caution and it is a technical challenge to the surgeon to deal for the first time with a femoral hernia because that is the best time when it can be handled. There are other rare hernias like parastomal hernias, where there is little debate whether we should put in a mesh or not. I feel that we're looking at the present concept of hernia repair, where we should definitely consider a mesh as a choice of repair; however, the high recurrence has been noticed with the suture technique. When coming to the rare hernias like the parastomal hernias, this is something which can be prevented. As the studies are of short duration, we will have to wait for a longer period to know. Also we discussed a very interesting aspect about rupture of the mesh especially with high-BMI obese patients. We should especially keep in mind the possibility of rupture, which is a reality. With this we have many more questions which are unanswered and I'm sure we can continue for days and days and many congresses like this to find the answer. Thank you very much for your patience.*

Personal Comment to the Paper of E. Schippers

U. Klinge, J. Conze

Among reasons leading to recurrence following mesh repair, the recurrence through a mesh due to a mesh rupture is rare. However, it has been reported, though the genesis is still obscure.

The description of a central mesh rupture through a Marlex mesh [1] with its known excessive textile strength may indicate a possible filament breakdown induced by the permanent bending within a flexible abdominal wall, probably favoured by the tendency of polypropylene to become stiffer over time. Further studies may reveal the impact of permanent alternate strains on the textile properties dependent on polymer, type of filaments and various mesh structures.

In contrast, the description of a central mesh rupture through a material reduced large-pore mesh may be due to an insufficient tensile strength of the mesh, when creating a thrust bearing for the implanted mesh has been disregarded. This can happen if the anterior fascia was not closed or in the case of early dehiscence. Burger et al. described early fascia dehiscence in the early postoperative period visible only by CT scan and not clinically detectable, that lead to incisional hernia formation at a later stage [4]. Estimations of the physical strength necessary clearly indicate that in the case of reinforcement with sufficient fascia closure, a tensile strength of about 16 N/cm is sufficient, whereas in the case of abdominal wall replacement without fascia closure a tensile strength of 32 N/cm is appropriate [2].

Unfortunately, the differentiation between reinforcement and defect-bridging of the abdominal wall, important for the selection of the right mesh material, has long been overlooked. This might explain the fact that many trials lack detailed description of this aspect.

In the case of a midline suture line including the underlying mesh, a local weakness could develop, favouring later mesh rupture.

We have seen three central ruptures of material reduced large-pore meshes ourselves [3] always in cases of missing anterior fascia closure. Since restricting the use of large pore meshes just for reinforcement of the abdominal wall with a sufficient fascia repair, we have seen no more central ruptures. If a fascia closure is not

feasible, we use a double layer of large pore meshes or, alternatively, small-pore, heavy meshes.

Considering all re-operations for recurrence following mesh repair, it is evident that the main problem is the recurrence at the mesh border due to insufficient overlap. However, in particular, the long-term outcome of mesh repair has to be surveyed carefully, to define the best compromise between sufficient tensile strength of the mesh prosthesis on the one hand and as little foreign material as possible for an improved biocompatibility on the other hand.

References

1. Langer C, Neufang T, Kley C, Liersch T, Becker H. Central mesh recurrence after incisional hernia repair with Marlex are the meshes strong enough? Hernia 2001; 5: 164 167
2. Schumpelick V, Nyhus L. Meshes: benefits and risks. Springer, Berlin Heidelberg New York, 2003
3. Klinge U, Conze J, Krones CJ, Schumpelick V. Incisional hernia: open techniques. World J Surg 2005; 29: 1066 1072
4. Burger JW, Lange JF, Halm JA, Kleinrensink GJ, Jeekel H. Incisional hernia: early complication of abdominal surgery. World J Surg 2005; 29: 1608 1613

What Can We Do to Improve Our Results?

38 Improved Teaching and Technique

S. Nixon, R. Rosch

Facing the persisting problem of hernia recurrence, improvement of the outcome might be achieved by an optimized method and by an improved teaching of the different surgical procedures.

With regard to the applied surgical method to do the hernia repair, randomized controlled trials of surgical operations are flawed because the surgeons are biased and blinding is not possible. Another problem is the surgeon himself, who is often guided by three rules that anticipate progress in operative therapies: don not believe another surgeon; never believe an expert surgeon; only believe trials that confirm pre-existing prejudices

An example might be the introduction of endoscopic hernia repair techniques. Investigations by Knook et al. revealed that in The Netherlands only 16% of surgeons do laparoscopic hernia repair and only 50% use it for primary hernia repair. The authors conclude that improvement in training of both surgical residents and surgeons is necessary to enhance the acceptance of this technique for inguinal hernia repair [1].

Concerning the impact of surgical skill, experience and teaching on hernia recurrence rates, it has been shown that the statistical methods used for assessing learning effects in health technology assessment have been crude and the reporting of studies poor [2]. Nevertheless, the literature review reveals some interesting information on this topic:

Investigating the effects of training and supervision on inguinal hernia recurrence, Robson et al. showed that supervised trainees had recurrence rates similar to seniors, whereas unsupervised junior trainees had poor results [3]. A comparison of open versus laparoscopic mesh repair of inguinal hernia revealed that the experienced surgeon had 4% recurrence rates in open surgery and 5% in laparoscopic surgery, whereas less experienced surgeons had 2.5% recurrence rates in open and 12% in lap hernia repair [4].

Regarding the training of TEP hernia surgery, it is of practical importance to clarify the necessary duration, the appropriate number of cases, how training can be accelerated and the costs. Additionally, it has to be decided who should be trained and whether it is worth the effort.

Following the learning curve, Haidenberg et al. showed that the recurrence rate is less than 1% with trainees performing TEP under supervision [5].

In another investigation, surgical inexperience was a strong predictor of recurrence after TEP and open hernia repair [6]. This again underlines the importance of supervision of trainees.

Investigations on the learning curve for TEP by Lau et al. showed that the time of operating reached a plateau after 80 cases. In this study all other parameters showed no change, and even during the learning curve, a low morbidity and conversion rate was found [7].

In order to further decrease the length of the learning curve, artificial TEP models have been introduced such as the Guildford Minimal Access Therapy

Training Unit (MATTU) that includes lectures, live demonstrations and practical training. This model has been described as an accurate replicate of the TEP repair, as robust, easy to use, cost-effective, easy to maintain and widely available in future [8]. Lal recommended a minimum of ten open Stoppa procedures before a trained surgeon starts TEP operations independently [9].

In order to face the problem of too few trainers, too many trainees and insufficient time, in Scotland it is recommended that a trainee has a standard experience of 40+40 hernia repairs, that consultant courses and congresses are visited, and that other media, such as the internet or DVD, are used.

Altogether, the basic rule of teaching operations is to teach the right operations and to do the operations right. Prolonged supervision has been shown to be highly cost-effective regardless of the higher costs for personal resources per operating minute [10]. To reduce hernia recurrence rates, failures should be studied.

References

1. Knook MT, Stassen LP, Bonjer HJ. Impact of randomized trials on the application of endoscopic techniques for inguinal hernia repair in The Netherlands Surg Endosc 2001; 15: 55–58
2. Ramsay CR, Grant AM, Wallace SA, Garthwaite PH, Monk AF, Russell IT. Statistical assessment of the learning curves of health technologies. Health Technol Assess 2001; 5: 1–79
3. Robson AJ, Wallace CG, Sharma AK, Nixon SJ, Paterson-Brown S. Effects of training and supervision on recurrence rate after inguinal hernia repair. Br J Surg 2004; 91: 774–777
4. Neumayer L, Giobbie-Hurder A, Jonasson O, Fitzgibbons R Jr, Dunlop D, Gibbs J, Reda D, Henderson W. Open mesh versus laparoscopic mesh repair of inguinal hernia. N Engl J Med 2004; 350: 1819–1827
5. Haidenberg J, Kendrick ML, Meile T, Farley DR. Totally extraperitoneal (TEP) approach for inguinal hernia: the favorable learning curve for trainees. Curr Surg 2003; 60: 65–68
6. Neumayer LA, Gawande AA, Wang J, Giobbie-Hurder A, Itani KM, Fitzgibbons RJ Jr, Reda D, Jonasson O. Proficiency of surgeons in inguinal hernia repair: effect of experience and age. Ann Surg 2005; 242: 344–348
7. Lau H, Patil NG, Yuen WK, Lee F. Learning curve for unilateral endoscopic totally extraperitoneal (TEP) inguinal hernioplasty. Surg Endosc 2002; 16: 1724–1728
8. Slater GH, Jourdan I, Folscher DJ, Snook AL, Cooper M, D'Allessandro P, Rangeley C, Bailey ME. The Guildford MATTU TEP hernia model. Surg Endosc 2001; 15: 493–496
9. Lal P, Kajla RK, Chander J, Ramteke VK. Laparoscopic total extraperitoneal (TEP) inguinal hernia repair: overcoming the learning curve. Surg Endosc 2004; 18: 642–645
10. Koperna T. How long do we need teaching in the operating room? The true costs of achieving surgical routine Langenbecks Arch Surg 2004; 389: 204–208

Discussion

Kehlet: *I think this was a good start up in this morning, very provocative. I think some US surgeons have to respond to this.*

Fitzgibbons: *Prof. Kingsnorth performed a study trying to teach non-physicians to do hernia surgery. He absolutely could not teach nurses to do a Lichtenstein operation. They could not make the decision about the sac and things like that. So I do not think that we can teach just anybody to do hernia surgery. We surgeons have to continue to do this work.*

Nixon: *We have a problem in the UK. Our government pays people less to do more and they want to pay nurses to do all the things in medicine, not just operate. I absolutely agree with you. We make a lot of academic decisions even during simple surgery and without that academic support I do not think these people can not do this kind of operation.*

Kurzer: *How many operations do you think you need to take a trainee through to be happy to let him off on his own as a trained TEP surgeon? Furthermore, do you have to train all the trainees or do you have to select out certain ones and just have a selected number?*

Nixon: *I think TEP surgery requires laparoscopic skills and some surgeons are better than others. There is no doubt. You are working in two dimensions. I have come across a few who I am not sure I could actually train or at least do not have the time to train them. I think that hernia surgery is special, as we discussed yesterday. Should hernia surgery be a speciality? I think it has to be, probably in the future. Because everything else is specialized. There is not much left. So I totally agree. Not everybody can do it, not everybody should do it, and we should promote the concept of a hernia being a specialized surgery.*

Kurzer: *Just give me a rough number when you take a raw person. How many operations do you think you have to take him through and afterwards say right, you are on your own. I am happy not to watch you any more. Thirty? Forty? Fifty?*

Nixon: *If I am honest, our trainees watch forty, do forty. But we really do not know.*

Deysine: *I have been training resident surgeons for a long, long time. The problem starts in medical school. Many of them choose to become a surgeon and they really do not have the hand coordination which is necessary.*

Sarr: *Dr. Berndsen, do you think every operation should have a standard set of points which should be checked like a clinical pathway?*

Berndsen: *I think so. I think if you take an operation and divide it into steps, you will never go to step two before*

you have done step one. Then it is easy to teach surgical trainees to do the operation.

Chan: *Since we are a specialized clinic we had to take on a new surgeon from time to time. So we know that we have to observe and assist them in about fifty cases before they even start, and when they really start we assist them in fifty cases. In general, we know that in the 1950s we had a recurrence rate of about 20%, over years it came gradually down to 4 or 5% and in 1960 we reached an acceptance of recurrent hernias of 1%. Just keep on looking on your own results and follow it.*

Berndsen: *I think this is very important. You must know what you do. It is not enough to check the results every hundred years. You have to have a continuous quality control.*

Schumpelick: *Do you really think it is justified to have specialized centres of hernia repair as Dr. Nixon said?*

Berndsen: *In the study mentioned there were two groups of surgeons. Twelve surgeons performed the TEP operation and thirteen the Shouldice operation and they were not mixed, but not specialized as well. They were interested in hernia surgery, but no specialised hernia surgeons.*

Schumpelick: *So do you think there should be one specialized surgeon performing the laparoscopic repair and one surgeon performing the open technique?*

Berndsen: *I think a surgeon specialized in hernia surgery can do more than one type of operation. This would be the best situation. One surgeon specialized in hernia surgery could have many operations so that he can tailor the operation for each patient. So the general surgeon can send the difficult cases to the centres.*

Duh: *One simple comment and one question. In the VA study the Lichtenstein repair recurrence rate was 4% for primary and 14% for recurrent hernias. So you need to put this in the slides of your Lichtenstein evaluation.*

Berndsen: *Yes, that is right.*

Duh: *My question is: it has been a problem for us to figure it out if someone has a recurrence. What is your gold standard for deciding? Examination by the surgeon himself? By somebody else? By a physician? By ultrasound? What do you think?*

Berndsen: *In the first study it was a questionnaire. We asked the patient if he had any discomfort or a bulge in the groin. Those who had this discomfort were examined. If there was no bulge and we could not find anything we made a herniography to see if he has a hernia.*

Duh: *This is a problem. If you only look at the symptomatic ones you will underestimate the recurrence rate.*

Berndsen: *Yes, this is right. And this is always the problem with follow-up. I think in a questionnaire study, you get a follow-up rate of 95% and you get most of the recurrences. You get the symptomatic ones and these are the ones who are clinically important. Maybe you miss some asymptomatic. But that's the way it is.*

39 Analyzing Reasons and Re-Operation for the Inguinal Hernias Recurring After Mesh-Plug Procedure

S. Ma

Introduction

Inguinal hernias have been widely repaired with mesh in China since the end of 1997. In the summary on my first 500 cases, there were 84 cases of recurrent inguinal hernias, among which 11 cases after repairing with mesh. The 11 patients were aged between 59 and 78 and all of them were male. Three of the patients suffered from bilateral hernias and one patient had a family history of hernia.

Results

Before the 11 cases underwent repair with mesh, 41.6% cases received conventional hernia repair. Among the 11 recurrent cases, 9 cases underwent perfix plug (Bard) repair, two cases underwent Lichtenstein operation with Marlex mesh; 91.6% cases recurred within 12 months post operation. All these 11 cases received re-operations. In re-operations, in five cases the newly recurred hernia sac was found to be located at the upper and outboard the pubic tubercle; in five cases newly recurred hernias were found at one side of the originally inserted but hardened mesh plugs; in one case mesh migrated to one side of the original repair site. To analyze by combining the findings of re-operations and following up the previous surgical records, the recurrent reasons were summarized as follows:

- The newly recurred hernia sac was found to locate at the upper and outboard the pubis tubercle. The reasons are:
  - The mesh contracted longitudinally and no mesh covered the direct hernia triangle area.
  - The mesh contracted in breadth and no mesh covered the pubic tubercle.
  - The mesh migrated totally and the onlay patch was not found during re-operation.
- The newly recurred hernias were found to locate at one side of the originally inserted but hardened mesh plugs. The reasons are:
  - The last operations were not consummate, and the mesh-plugs were not fixed at the strong tissue surrounding the hernia deep ring.
  - The mesh plug contracted.

Re-Operation Methods for the Eleven Recurrent Patients

- To seek and dissect the hernia sac at a high position.
- If there is a small defect only and the tissue surrounding the defect is complete and solid, insert a cut plug and anchor surrounding tissue.
- Insert a flat mesh into preperitoneal space through the defect and overlap 3–4 cm with normal tissue which surrounded the defect area.

- Or to insert a flat mesh above the transversalis fascia under cord and external oblique aponeurosis. The mesh was fixed with a Lichtenstein procedure.
- Or to insert one mesh separately into the preperitoneal space and the space between transversalis fascia under cord and external oblique aponeurosis.

One patient died from a traffic accident among the 11 cases, and all the other patients were followed up. One patient who had received repair four times recurred locally 1 year and 10 months after re-operation. None of the other patients recurred, the mean follow-up period was 5 years and 3 months.

Opinions

When the recurrent inguinal hernia patient with mesh-plug repair undergoes re-operation, the hernia sac should be sought and dissected at high position. Then, according to the condition of the tissue surrounding the defect, a big enough mesh should be inserted into the preperitoneal space or the space above the fascia transversalis. If there is a small defect only and the tissue surrounding the defect is complete and solid, to insert a cut plug and anchor the surrounding tissue still is the better procedure.

Currently, the exact recurrence rate of inguinal hernia after repair with mesh is still not clear. So an international prospective clinical study with 2-year follow-up on the recurrence rate of inguinal hernia is suggested. This clinical study should be performed under a uniform classification, uniform surgical procedure, and should be in participation with an independent third part for follow-up monitoring. This task could be accomplished by organizing a collaboration group.

Discussion

Kehlet: *In those two patients where you had a recurrence, did you make any histological examination of the material?*
Ma: *I reviewed the patients and the first may be a problem of the technique.*
Kehlet: *No, I did not asked for the reasons of the recurrence. You did not take out a piece of the material and look for invasion of new collagen? So you did not made a biopsy of the mesh?*
Ma: *No, we did not. We just started.*
Itani: *Yesterday we heard about central mesh failure and I noticed that you were putting holes into your mesh to allow drainage and I am wondering if this is one aspect of your recurrence because you disrupted the mesh.*
Ma: *That is a good idea. Maybe they caused the recurrence.*
Sarr: *Most of the studies with alloderm, which is the US equivalent, do not suggest putting holes in the mesh, because the ingrowth is supposed to be very quick.*

40 Standard Procedures for Standard Patients?

H. Kehlet, M. Bay-Nielsen

Introduction

A variety of procedures is available to repair a groin hernia, including several techniques for open repair and a couple for laparoscopic repair. Most of these techniques have been assessed in multiple, randomized controlled trials and evaluated in meta-analyses, showing similar recurrence data from laparoscopic vs. Lichtenstein technique [1], but with more recurrences after conventional sutured repairs [1] (Bassini, Shouldice, McVay, anuloplasty, etc). However, many experts' series from single centres continue to report excellent results with each of the available techniques. However, for the general surgeon, the key question is whether a specific technique is preferable in specific patients and also the effectiveness, i.e. generalizability of the technique for more widespread use outside expert centres. For this discussion, we have therefore chosen to show nationwide results from Denmark, based upon the Danish Hernia Database collaboration [2], which covers > 98% of all hernias performed in Denmark with focus on specific groups of patients with a groin hernia.

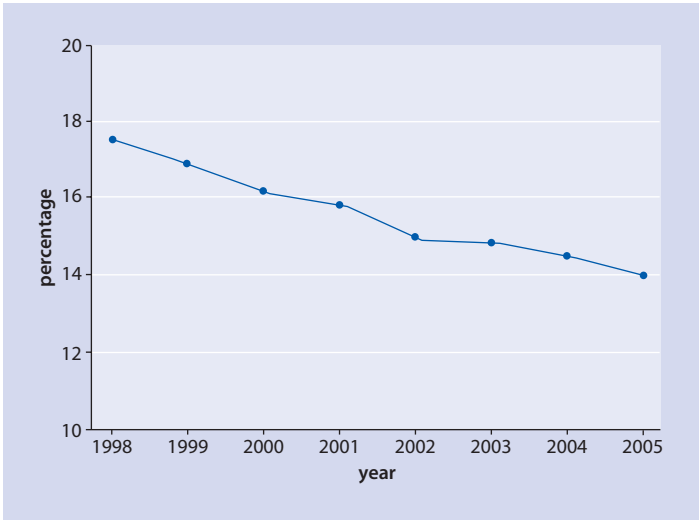


Fig. 40.1. Percent of groin hernia repairs for a recurrence in Denmark

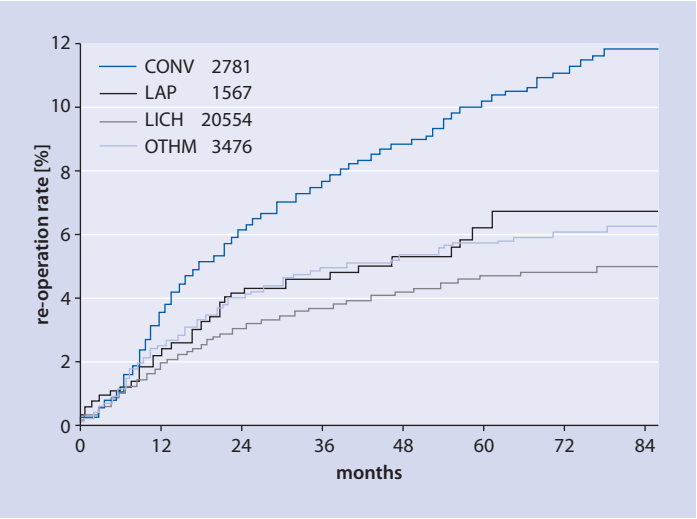


Fig. 40.2. Elective primary medial inguinal hernia

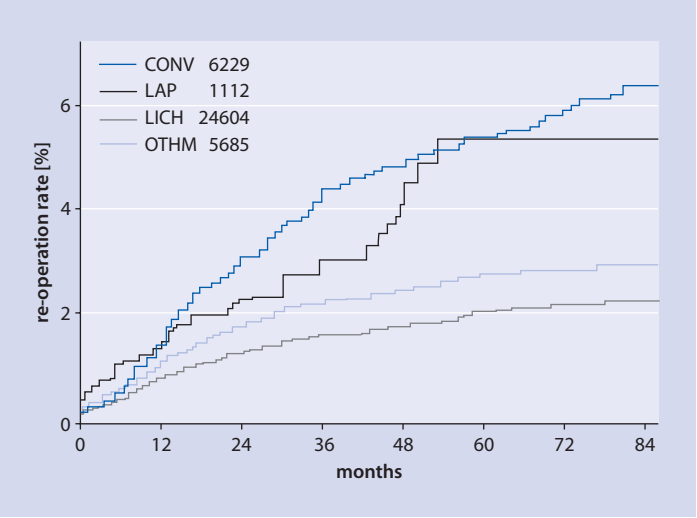


Fig. 40.3. Elective primary lateral inguinal hernia

All-Over Recurrence Rates Over Time in Denmark

Figure 40.1 shows the changes in the rate of operations for a recurrence in Denmark from the start of the Hernia Database collaboration on Jan. 1 1998 until the end of 2005. In accordance with the increased use of a Lichtenstein repair (about 70%), a relatively stable use (about 10%) of laparoscopic repairs and sharp decrease in use of conventional sutured repairs and other mesh types [2], the rate of operations for a recurrence have been steadily decreasing from about 18 to 14%. The high proportion of operations for a recurrence is explained by recurrences occurring from an operation performed before 1998.

Primary Medial Inguinal Hernia

As shown in Fig. 40.2, the re-operation rate (Kaplan-Meier Plot) is significantly higher after the sutured repairs than other repairs and with no significant differences between the mesh and laparoscopic techniques.

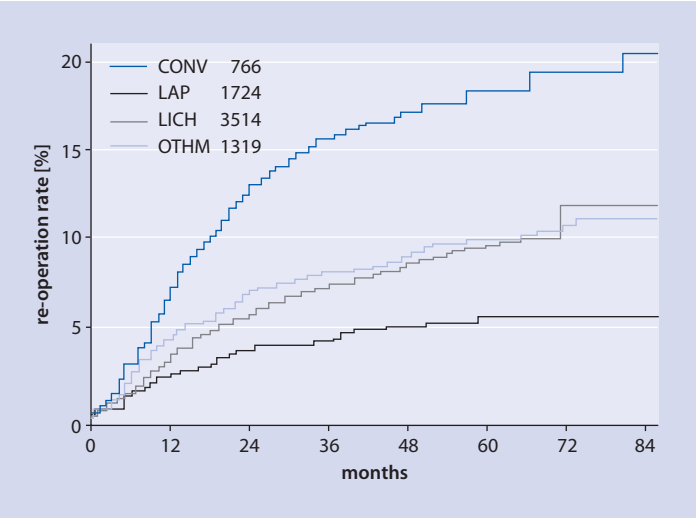


Fig. 40.4. Elective recurrent medial inguinal hernia

Primary Lateral Inguinal Hernia

As seen in Fig. 40.3, re-operation rates are significantly higher after conventional sutured repairs and laparoscopic repairs than other mesh techniques. The unexpectedly increased re-operation rate after laparoscopic repairs may be explained by insufficient surgical technique, especially in bilateral hernias [3].

Recurrent Medial Inguinal Hernia

Re-operation rates after recurrent medial inguinal hernia repairs are again significantly higher with sutured repairs, but lower with a laparoscopic repair compared with other mesh techniques (Fig. 40.4).

Recurrent Lateral Inguinal Hernia

As shown in Fig. 40.5, again sutured repairs have the highest re-operation rates with no differences between the three other mesh techniques.

Female Hernia

We have previously reported [4] that re-operation rates are unexpectedly higher in female inguinal hernias than in males, as also shown from data from the Swedish Hernia Database [5]. Figure 40.6 shows again high re-operation rates with conventional sutured repairs, but unexpectedly high recurrence rates with a Lichtenstein mesh repair. The uses of other meshes (plug) and laparoscopic repairs have the lowest re-operation rates.

Femoral Hernia

In Denmark it has been recommended to use either laparoscopy or a Lichtenstein-type mesh repair (ad modum McVay or plug) for femoral hernias. As seen in Fig. 40.7, the lowest re-operation rates occurred after a laparoscopic or mesh McVay repair.

Re-Operation Rates in High- vs. Low-Volume Centres

In an ongoing analysis (unpublished), a linear relationship has been found between the risk of a re-operation and the number of repairs performed in a given department. Thus, in low-volume departments (< 65 operations/year) an about 40% higher re-operation rate was found compared to high-volume departments (> 130 operation/year) analysed for Lichtenstein repairs only. Although these data could not be analyzed for surgeon volume, other data suggest again an about 40% lower re-operation rate after Lichtenstein repair performed in the few Danish private clinics with predominantly high-volume surgeons compared to operations performed in hospitals with assumed lower-volume surgeons (unpublished).

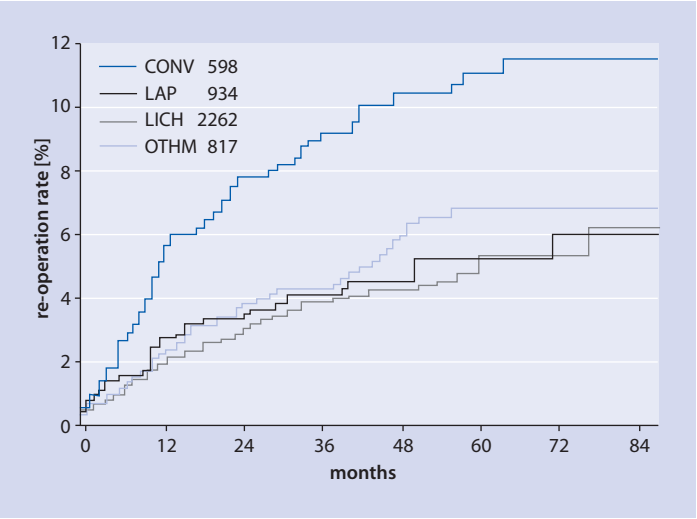


Fig. 40.5. Elective recurrent lateral inguinal hernia

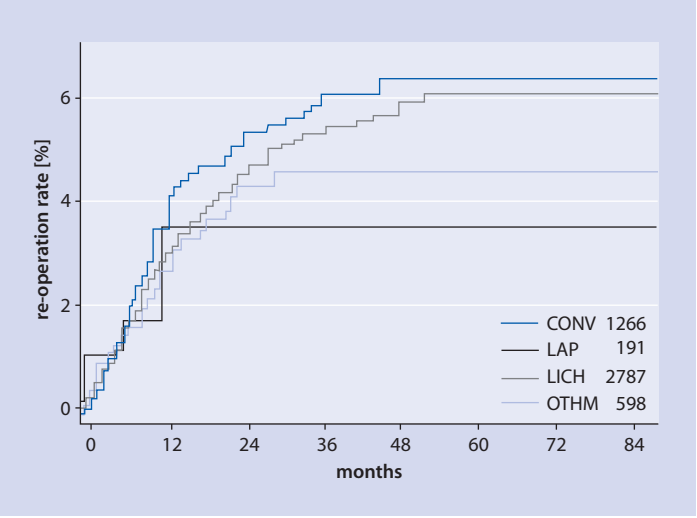


Fig. 40.6. Elective female inguinal hernia

Conclusions

When discussing standard operations for standard patients in general practice, the conclusions based on nationwide data must depend on the reality that most operations are performed by relatively low-volume surgeons in median-volume departments. The data clearly suggest that the Lichtenstein mesh repair has the lowest re-operation rates (or comparable with the laparoscopic technique) in both medial and lateral primary inguinal hernias compared to other techniques, except that in Denmark there is an apparent insufficient technique when performing a laparoscopic repair of a bilateral/lateral hernia [3]. For recurrent hernias the data support the use of a Lichtenstein repair or a laparoscopic repair, probably to some extent depending on type of previous repair, where final results will be available from a large (about 500 patients), just finished randomized trial. The data in female inguinal repairs show an unexpectedly higher re-operation rate, probably due to overlooked femoral hernias [4, 5] and support the use of a laparoscopic technique or at least exploration for a concomitant femoral hernia at the primary open operation. In femoral hernias a laparoscopic repair or a mesh repair a.m. McVay or plug is recommendable. Based upon the nationwide data, sutured repairs should be abandoned,

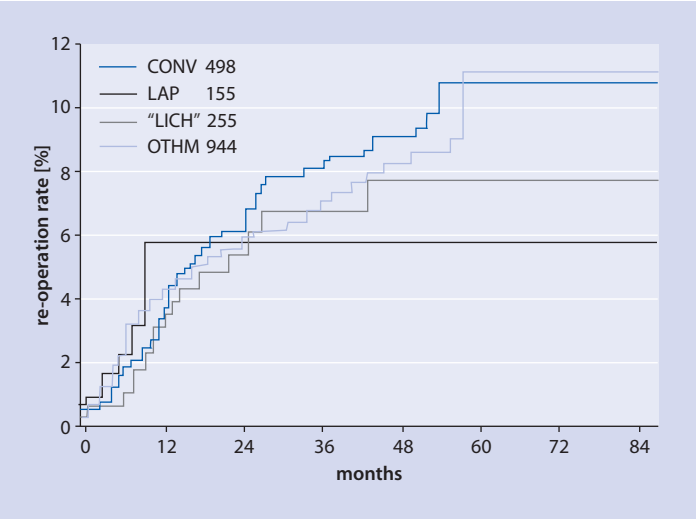


Fig. 40.7. Elective femoral hernia

despite positive results in the literature from specialized surgeons/clinics. Most importantly, the data suggest that further improvements probably need further specialization, i.e. high-volume surgeons and high-volume departments with close monitoring of the results. Also there is a need for future studies on other specific subgroups, i.e. patients with increased body weight, as well as more data are required on femoral hernias where recurrence rates are higher than after inguinal hernia repairs. In this context, collaborative efforts are important since femoral hernias are relatively rare and therefore cannot be studied sufficiently in single or a few centres. Finally, it must be remembered that after the introduction of Lichtenstein mesh repair and laparoscopic repairs, re-operation rates are generally low (2–3%), except for a twofold higher risk after surgery for a recurrence. This is in contrast to other sequelae such as chronic pain problems [6, 7], which must be considered of increasing importance, since they occur at the same or higher rate, but where treatment results so far are undocumented [8]. Therefore, despite being a relatively small, although common operation, groin hernia repair continues to demand further improvements in the general surgical profession.

References

1. EU Hernia Trial Lists Collaboration. Repair of groin hernia with synthetic mesh: Meta-analysis of randomized controlled trials. Ann Surg 2002; 235: 322–332
2. Bay-Nielsen M, Kehlet H, Strand L, et al. Quality assessment of 26,304 herniorrhaphies in Denmark: A prospective nationwide study. Lancet 2001; 358: 1124–1128
3. Wara P, Bay-Nielsen M, Juul P, Bendix J, Kehlet H. Prospective nationwide analysis of laparoscopic vs Lichtenstein repair of inguinal hernia. Br J Surg 2005; 92: 1277–1281
4. Bay-Nielsen M, Kehlet H. Inguinal herniorrhaphy in women. Hernia 2006; 10: 30–33
5. Koch A, Edwards A, Haapaniemi S, Nordin P, Kald A. Prospective evaluation of 6895 groin hernia repairs in women. Br J Surg 2005; 92: 1553–1558
6. Aasvang E, Kehlet H. Chronic postoperative pain: the case of inguinal herniorrhaphy. Br J Anaesth 2005; 95: 69–76
7. Aasvang EK, Møhl B, Bay-Nielsen M, Kehlet H. Pain-related sexual dysfunction after groin hernia repair. Pain 2006; 122: 258–263
8. Aasvang E, Kehlet H. Surgical management of chronic pain after inguinal hernia repair. Br J Surg 2005; 92: 795–801
9. Wilkiemeyer M, Pappas TN, Giobbie-Hurder A, Itani KMF, Jonasson O, Neumayer LA. Does resident post-graduate year influence the outcomes of inguinal hernia repair? Ann Surg 2005; 241: 879–884

Discussion

Amid: *One of the standard steps of Lichtenstein repair is looking for femoral hernia, no matter if the patient is male or female. No relative to failure of Lichtenstein for repairing of femoral hernia, most surgeons that I have seen they used the standard-shaped mesh for femoral hernia as well as for inguinal hernia, but that does not work because if you suture the lower edge of the mesh to the Cooper ligament then you are not going to be able to overlap the mesh to the pubic tubercle. Because of that, the shape of the mesh has been modified and the modified shape of the mesh has been demonstrated in my slices and*

my article. That mesh has a small dropdown triangular extra mesh. The normal Lichtenstein mesh is placed over the pubis tubercle and that dropdown which is triangular is sutured to the Cooper ligament. So I am sure that the reason that they failed is the fact that they used the standard-shaped mesh, not the modified one.

Kehlet: *I agree.*

Sarr: *But how do you fix the mesh? You have raised the question. Should there be an insert with the mesh that has a picture drawn for a femoral hernia versus an inguinal hernia?*

Amid: *Well the insertion is very easy.*

Sarr: *No, I mean should there be a picture in the package of the mesh for a femoral hernia?*

Amid: *There is no commercially available mesh for femoral hernia repair. It has to be tailored by the surgeon in the operating room. It is very easy. It takes 30 s to make that little triangular dropdown.*

Kehlet: *Good advice.*

Miserez: *We noticed that laparoscopy was mainly bad in primary bilateral hernias. How do you explain this? Are we talking about different surgeons even on a nationwide level?*

Kehlet: *Yes, different surgeons, but the volume of each department is very, very low. Thirty operations a year or so. We could not make any correlation between the number of operations performed and the re-operation rate in laparoscopic repair because the numbers were so small. Again, remember we have just a few laparoscopic operations in Denmark, just 5–6%.*

Sarr: *But even in your volume data it was a 1% difference, not a 15% difference.*

Kehlet: *Yes, 1% is very small. But if we want to get the recurrence rate down, even this 1% is important.*

Deysine: *When the general surgeons were trained about 30 years ago to perform orthopedic procedures, there were all kinds of operations with terrible results. Since they have become a speciality their results are now magnificent.*

Kehlet: *I totally agree. In Denmark we are five and a half million inhabitants and we are doing this operation in a about 70 departments and in 10 to 12 private clinics and that should be in my opinion reduced to at least half.*

Young: *There is actually a mesh designed with excellent characteristics for repair of femoral hernia, the PHS mesh. This mesh has an underplayed portion that covers the femoral area and it also has the overlay which essentially covers the myopectineal orifice.*

Kehlet: *I agree. But as you see, we are trying to decrease the number of techniques in order to increase experience. So we should not like to introduce that without really hard data showing that it is better.*

Young: *We are working on it.*

Schumpelick: *Despite your trying to standardize your technique you reduced your nationwide recurrence rate from 18 to 14%. So you still have 14%. Everywhere the same percentage, in Denmark, in Germany. How do you explain that?*

Kehlet: *Well, we are optimistic; we think that these are the old failures. We have to analyze it again.*

Schumpelick: *Could it be a biological reason?*

Kehlet: *Could be. We still have a lot of open repair and they have to finish.*

Sarr: *I think this is the question I hope to answer in the next 15 min.*

41 Tailored Approach for Non-Standard Patients

C. Peiper, S. Schinkel, K. Junge

Introduction

Many different therapeutic principles compete in the treatment of inguinal hernias. Due to their training, many surgeons prefer just one kind of inguinal hernia repair. Many institutions provide one single therapeutic principle in high volumes. Examples are the Shouldice Hospital in Toronto, which up to now has performed more than 270,000 suture repairs, while specialized laparoscopic clinics do more than 1000 laparoscopic repairs every year. This principle of doing one single operation at a high level of routine and standardization for any patient may lead to a good quality of the used procedure, but is not accepted everywhere. Due to great variations in patients concerning body constitution, size of the hernia or collagen metabolism, the idea of one operation suitable for all patients has to be discussed.

It is accepted that the use of a mesh hernioplasty will reduce recurrence rates, at least within the first 5–10 years [3, 8]. On the other hand, there are certain mesh-related complications reported in the literature. These include late mesh infections with cutaneous fistulization and inflammatory changes of the spermatic cord. Therefore, one standardized therapeutic procedure for all patients seems to be no longer the best way. An individual approach including patient-depending factors, which may lead to a mesh implantation only in selected cases, seems to fit better. The remaining question is: which patient is at risk of developing a recurrency, and therefore benefits from mesh implantation? This question has to be answered, if therapy of the inguinal hernia is to be improved.

Identification of the Risk Population

The decision about the therapeutic procedure for any hernia patient may be influenced by:

- Analysis of the collagen subtypes produced by the individual.
- Estimation of the risk factors in the patient's history.
- Suggestion by the surgeon.

Collagen Test

A 5-mm skin specimen harvested in local anaesthesia at the lumbar region is analyzed under Sirius red and fast green stains. With colour spectroscopy, distinction between collagen type I and type III is possible. Patients with recurrent hernias present a larger amount of type-III collagen, while the collagen I/III ratio is larger in controls. We regarded any ratio > 10 as normal and any ratio < 5 as pathological.

HEAD Score

The HEAD score (Hernia of the Adult Disease) was developed to enable an objective and scientifically based estimation of the individual risk to develop an inguinal hernia recurrency. The score is based on the proven risk parameters:

Table 41.1. HEAD-Score

Gender	Male Female	3 1
Age	> 50 years < 50 years	3 1
Hernia	Primary hernia First recurrency Multiple recurrency	2 4 8
Size	> 3 cm < 3 cm	3 1
Localization	Several locations One location	4 1
Smoking	Yes No	2 1
Family	Occurance of hernias in > 2 first-grade relatives Occurance of hernias in < 2 first-grade relatives	3 1
Collagen disorders	Proved alteration in collagen No evidence of alterations in collagen	5 1
Total		

Table 41.2. Current operative procedures (selection)

Suture repair	Marcy/Zimmerman Shouldice Bassini Lotheisen/McVay
Open mesh repair	Lichtenstein
Plug repair	Rutkow Gilbert
Preperitoneal repair	Ugahary TIPP/Rives Moran Nyhus Wantz Stoppa's GPRVS
Endoscopic repair	TAPP TEP

- Gender [9)
- Age [19)
- Presence of recurrency [13)
- Size of the hernial gap [19)
- Existence of several hernias [16)
- Smoking [22)
- Family-related disposition [12)
- Collagen disorders [1, 23)

We developed the score system according to the clinical relevance of the single parameters (Table 41.1).

In a retrospective evaluation we used the HEAD score in 293 Shouldice repairs in 1992. The patients were followed up for 10 years. We observed a recurrence rate of 7.7% in the primary hernias. The patients with a HEAD score of 15 or less presented a recurrence rate of only 2.7%. Therefore, we regarded a score of more than 15 points as increased risk for recurrency.

Suggestion by the Surgeon

The decision about the surgical procedure is made during the preoperative discussion between surgeon and patient. The following parameters may have an influence:

- Expectation of the patient. Most patients are informed by papers, internet or friends about different methods and have certain ideas about the best proceeding.
- Provision by the surgeon. Due to their training or conviction, many surgeons provide just one or two surgical techniques.
- Results in world literature.

Guided by these points, surgeon and patient are to chose the best procedure out of many current possibilities (Table 41.2).

Prospective Study

Materials and Methods

The HEAD score was used in a county hospital within a prospective study. During 17 months we performed 405 cases (Table 41.3). Collagen testing was carried out in 20 cases. Statistics were neglected due to the small sample sizes.

We asked for all relevant risk factors using a standardized question form and calculated the individual HEAD score. We used the score as an argument in the pre-operative discussion with the patient about the sur-

Table 41.3. Patients

Recruitment	01.06.2003 to 30.11.2004
Operations	n = 405
Male/female	358/47
Age (mean, range)	57.0 (10–97) years
Hospital stay (mean + SD)	4.4 + 2.3 days
Follow-up after 14 days	n = 351 (86.7%)
Follow-up after 12 months	n = 301 (74.3%)

gical strategy. Mesh repair was suggested in scores of more than 15: In bilateral and recurrent cases following anterior repair TAPP (10), in all other cases open mesh repair (2) was suggested. If the score was below 15 we suggested a Shouldice repair (20). The final decision about the surgical procedure was made by the patient.

All patients were invited for follow-up after 2 weeks and 12 months. We asked about complaints, return to physical activity, and operative re-intervention. Ultrasound of the inguinal region was added to the physical examination.

Statistical analysis was done with the Mann-Whitney-test. Statistical significance was assumed, if $p < 0.05$. All data are presented as mean + standard deviation (SD).

Results

In patients with a primary inguinal hernia the collagen I/III ratio was smaller than in recurrent cases (Fig. 41.1). Of course, primary hernias were more often treated by suture repair than recurrent hernias (Fig. 41.2); 61.5% of all cases were operated using a mesh repair. The correlation between the HEAD score and the therapeutic procedure is shown in Fig. 41.3. Showing a score of more than 16, only 8 patients underwent Shouldice repair; 351 patients (86.7%) could be followed up 14 days after the operation. These results are shown in Table 41.4. Seroma was detected in more than every third patient by clinical examination and ultrasound. Intervention, however, was the exception. Seromas occurred more often after open mesh implantation than after laparoscopic operation ($p = 0.001$) or after suture repair ($p = 0.03$) (Fig. 41.4). We observed more seromas after any mesh repair (48.5%) than after the Shouldice operation (16.6%).

All other complications were distributed similarly to the operations. Surgical revision was necessary because of bleeding in one patient and due to wound infection in two cases. Two more patients underwent a second operation of an ilioinguinalis syndrome.

In the second follow-up after 12 months 301 patients (74.3%) took part. In the whole group we detected eight recurrences (2.6%). Concerning return to full physical activity we observed only minor advantage for the Lichtenstein repair without difference of statistical significance (Shouldice: 5.5 + 6.3 weeks, Lichtenstein: 3.9 + 4.2 weeks, TAPP: 4.2 + 3.5 weeks).

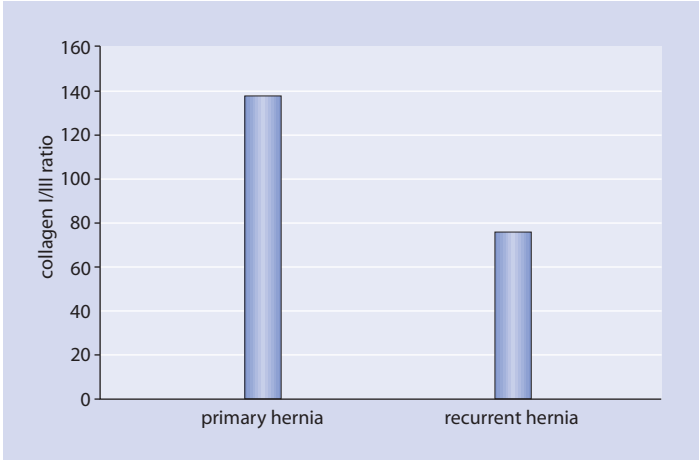


Fig. 41.1. Collagen I/III ratio in hernia patients (mean)

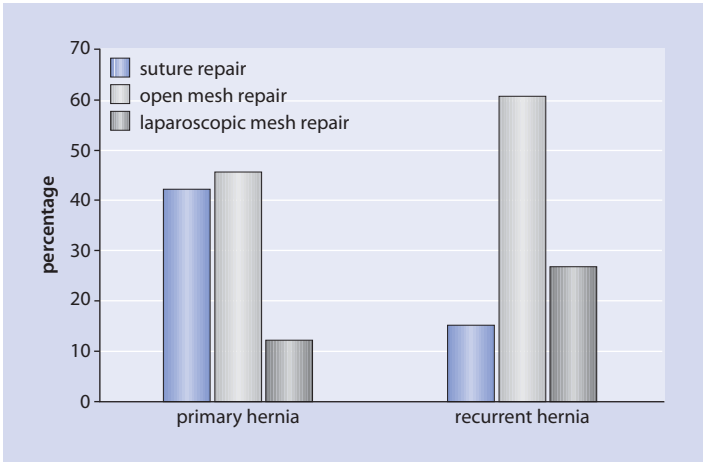


Fig. 41.2. Operations for primary and recurrent inguinal hernia (%)

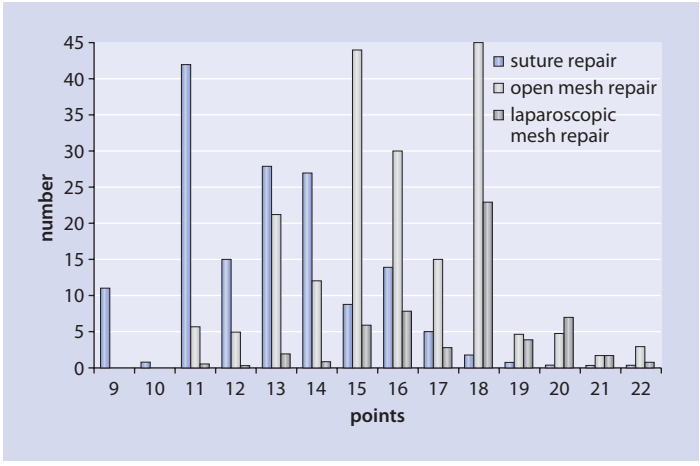


Fig. 41.3. HEAD score and operation used

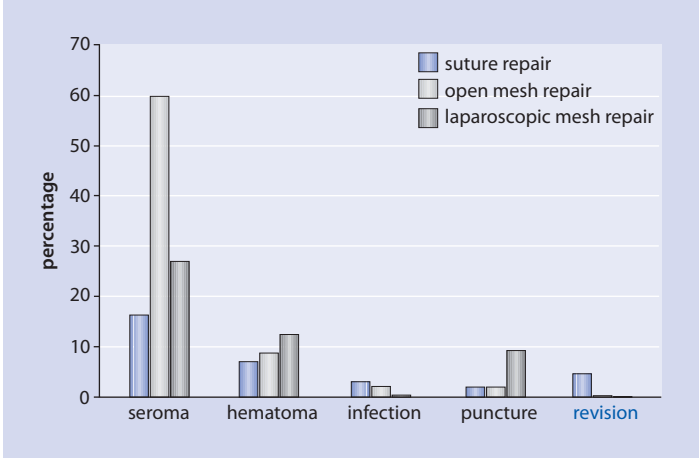


Fig. 41.4. Complications and different methods of operation (%)

Table 41.4. Early postoperative results (14 days, n = 265)

	n	%
Seroma	140	39.9
Hematoma	28	8.0
Infection	8	2.3
Puncture	11	3.1
Revision	5	1.4

Discussion

The tailored approach for the repair of an inguinal hernia is popular, but needs an objective tool to support the correct decision. Pre-operative tests on a molecular biological basis may help in certain cases, but they are not 100% reliable and standardized, and are still in their experimental stadium. Therefore we depend on anamnestic factors. The HEAD score developed on this basis was used in a county hospital starting in June 2003. We found it useful for the pre-operative discussion with the patient. Many of them, however, believe to be informed by the internet or other doctors. Their decision was rarely changed by our information. This led to several operations beyond our therapeutic concept.

The results of our follow-ups support the theory of early physical recovery after mesh repair. On the other hand, we observed a strong foreign-body reaction after Lichtenstein repair. Lacking histological evaluation we found the highest rate of seroma formation.

The rate of recurrence was small (2.6% after 1 year), especially in a non-specialized county hospital. Under comparable conditions, other authors observed rates between 2.3% (TAPP) [6] and 3.5% (Lichtenstein) [7]. We concluded that despite a narrow indication for mesh repair, the recurrence rate did not increase.

The individualization of inguinal hernia therapy with the help of the HEAD score did not increase complication rates. We believe that the risk for complications associated to the mesh, which are still published as case reports (colonic arrosion [5, 14], intestinal obstruction (11, 18), bladder arrosion [4, 17]), will decrease with a tailored approach. Especially young patients might also be protected from possible urological side effects of mesh implantation (obstructive azoospermia [21], dysejaculation [15]).

References

1. Adye B, Luna G (1998) Incidence of abdominal wall hernia in aortic surgery. Am J Surg 175: 400–402
2. Amid PK, Shulman AG, Lichtenstein IL (1994) Lichtenstein herniotomy. Chirurg 65: 54–58
3. Bay-Nielsen M, Kehlet H, Strand L, Malmstrom J, Andersen FH, Wara P, Juul P, Callesen T; Danish Hernia Database Collaboration (2001) Quality assessment of 26,304 herniorrhaphies in Denmark: a prospective nationwide study. Lancet 358: 1124–1128
4. Bodenbach M, Bschleipfer T, Stoschek M, Beckert R, Sparwasser C (2002) Intravesical migration of a polypropylene mesh implant 3 years after laparoscopic transperitoneal hernioplasty. Urologe 41: 366–368
5. Celik A, Kutun S, Kockar C, Mengi N, Ulucanlar H, Cetin A (2005) Colonoscopic removal of inguinal hernia mesh: report of a case and literature review. J Laparoendosc Adv Surg Tech 15: 408–410
6. Czechowski A, Schafmayer A (2003) TAPP versus TEP: a retrospective analysis 5 years after laparoscopic transperitoneal and total endoscopic extraperitoneal repair in inguinal and femoral hernia. Chirurg 74: 1143–1149
7. Dieterich K, Eichhorn J (2004) Five hundred outpatient hernioplasties using the Lichtenstein method. Chirurg 75: 890–895
8. EU Hernia Trialists Collaboration (2002) Repair of groin hernia with synthetic mesh: meta-analysis of randomised controlled trials. Ann Surg 235: 322–332
9. Eypasch E, Paul A (1997) Abdominal wall hernias: epidemiology, economics and surgical technique – an overview. Zbl Chir 122: 855–858
10. Felix EL, Michas CA, McKnight RL (1994) Laparoscopic herniorrhaphy. Transabdominal preperitoneal floor repair. Surg Endosc 8:100–103
11. Ferrone R, Scarone PC, Natalini G (2003) Late complication of open inguinal hernia repair: small bowel obstruction caused by intraperitoneal mesh migration. Hernia 7: 161–162
12. Gong Y, Shao C, Sun Q, Chen B, Jiang Y, Guo C, Wei J, Guo Y (1994): Genetic study of indirect inguinal hernia. J Med Genet 31: 187–192
13. Kald A, Nilsson E, Anderberg B, et al. (1998) Reoperation as surrogate endpoint in hernia surgery. A three year follow-up of 1565 herniorrhaphies. Eur J Surg 164: 45–50
14. Lange B, Langer C, Markus PM, Becker H (2003) Mesh penetration of the sigmoid colon following a transabdominal preperitoneal hernia repair. Surg Endosc 17: 157
15. Langenbach MR, Schmidt J, Lazika M, Zirngibl H (2003) Urological symptoms after laparoscopic hernia rapair. Reduction with a variant of polypropylene mesh. Urologe 42: 375–381
16. Melis P, van der Drift DG, Sybrandy R, Go PM (2000) High recurrence rate 12 years after primary inguinal hernia repair. Eur J Surg 166: 313–314
17. Rieger N, Brundell S (2002) Colovesical fistula secondary to laparoscopic transabdominal preperitoneal polypropylene (TAPP) mesh hernioplasty. Surg Endosc 16: 218–219
18. Rink J, Ali A (2004) Intestinal obstruction after totally extraperitoneal laparoscopic inguinal hernia repair. JSLS 8: 89–92

19. Rios A, Rodriguez JM, Munitiz V, Alcaraz P, Perez D, Parrilla P (2001) Factors that affect recurrence after incisional herniorrhaphy with prosthetic material. Eur J Surg 167: 855–859
20. Schumpelick V (1984) Shouldice repair in inguinal hernia. Chirurg 55: 25–28
21. Shin D, Lipshultz LI, Goldstein M, et al. (2005) Herniorrhaphy with polypropylene mesh causing inguinal vasal obstruction: a preventable cause of obstructive azoospermia. Ann Surg 241: 553–558
22. Sorensen LT, Friis E, Jorgensen T, Vennits B, Andersen BR, Rasmussen GI, Kjaergaard J (2002) Smoking is a risk factor for recurrence of groin hernia. World J Surg 26: 397–400
23. Uden A, Lindhagen T (1988) Inguinal hernia in patients with congenital dislocation of the hip. A sign of general connective tissue disorder. Acta Orthop Scand 59: 667–668

Discussion

Kehlet: *I think it is a nice hypothesis, but I am a little concerned about so few numbers and with a follow-up rate of 75%. That makes it a little dangerous to make conclusions when you miss 25%.*

Peiper: *No one can compete with your database. I had the same impression. I also think that a follow-up of 75% is not enough. But the problem is, if you want to make it higher you have to go home to every patient and perform your investigation there. And we made no questionnaire, we examined every patient because in our opinion it is the only way to get the real recurrence rate.*

Kehlet: *Well the approach is very nice with the HEAD score and so but that's a good topic for collaboration between different centres to get higher numbers and really get some good answers.*

Simons: *Maybe I missed a slide. But how many surgeons performed these operations? Do you have residents or are there dedicated surgeons that do more operations than others?*

Peiper: *It is just a regular county hospital and we are nine surgeons including residents at the beginning of their residency. And everyone is doing these operations. Not the laparoscopic one, but the opens ones were performed by any.*

Simons: *I think we showed that in a residency program you can get your recurrence rate down and after four years to 1.8% by a less tailored approach with residents in general surgery and I think the tailoring is that endoscopic surgery is only done by one surgeon. So all the difficult and the high risk patients stay in expert's hands and general surgery can do the others techniques.*

Peiper: *I think if you just focus on the recurrence rate as we just heard by Prof. Kehlets talk there is no way except of the Lichtenstein technique. But I think there are additional problems we just discussed also. And that's why I think that it is not correct just to focus on the recurrence rate. The other problems are not evaluated. No one asks for testicular pain of dysejaculation or anything else. These data are not published but might be a problem.*

Simons: *That is the whole point. We do not know exactly if these problems are only mesh related.*

Kehlet: *And if people will follow your score really need to have some specialised surgeons for the open repair.*

Kurzer: *I think everybody agrees that it makes sense that you have to perform the operation for the patient and so you have to have surgeons who can deal with a range of operations, because if your patients really requires a laparoscopic repair and comes to a surgeon who does not do a laparoscopic repair we all know that in the real world this surgeon does not want to loose his patient and he will not send his patient to a laparoscopic surgeon even knowing that this patient should have a laparoscopic repair.*

Peiper: *If you want to do the best for the patient you should include a laparoscopic surgeon in your team. This would be the best. But even on the other hand. If a laparoscopic surgeon does not provide a Shouldice or Lichtenstein repair in local anaesthesia he should refer his patients who are suitable for this to someone else. This would be good team work. Of course it is difficult in reality, I know.*

Kurzer: *So it would be easier to have one surgeon doing all this operations.*

Peiper: *Would be the best way. I learned hernia surgery also in a kind of monoculture but you can learn it.*

Sarr: *One of the ways that might happen is that the insurance companies might be the one that determine who does the operation. The chair of the department might be.*

42 Identification of the Patients at Risk (for Recurrent Hernia Disease)

P.R. Mertens, P. Lynen-Jansen, U. Klinge

What Do We Know About the Pathogenesis of Recurrent Hernia Disease?

Since its first description by Keith in the year 1924 on the origin and nature of hernia (Keith A, Brit J Surg 11: 455–475), ample evidence has been collected that the collagen composition is altered in wound-healing disturbances that are summed up as recurrent hernia disease. The predominant observation is that the type-I to type-III collagen ratio is altered in such a way that type-III collagen is more abundant in wound-healing disturbances. Klinge et al. have confirmed this finding in patients with inguinal and incisional hernia disease [1, 2], and novel work indicates that such a disturbance in collagen composition is also prevalent in patients with hemorrhoids as well as diverticulosis [3]. Using different protein and molecular biology techniques, this finding was confirmed in tissue specimens from patients who underwent surgical intervention for failure of mesh implantation [4]. The main question arising is which factor(s) direct the altered collagen ratio. Undoubtedly, tissue fibroblasts are the major source of interstitial collagens in tissue. Fibroblasts may not be regarded as uniform cell type with uniform synthesis profile. Interaction of fibroblasts with infiltrating macrophages may have significant influence on their expression profile [5]. Therefore, Klinge et al. set up an in vitro model system with outgrowths of primary fibroblasts from healthy controls and from patients with recurrent hernia disease [6, 7]. This model system allowed them to test whether the collagen synthesis rate is different in fibroblasts obtained from these two groups and circumvents the possible interference of infiltrating mononuclear cells. As major finding, Klinge et al. determine that the fibroblasts in vitro exhibit the same alterations of collagen synthesis as has been determined in tissue specimen, indicating that a defect must reside within the inherent fibroblast synthesis program.

How Is Wound Healing Coordinated?

Wound healing may be subdivided into different phases, inflammatory, proliferative, and maturation [8]. The dynamic nature of this process has to be kept in mind. It is well known that exogenous factors like smoking may affect wound-healing processes, e.g. by reducing the blood flow due to its vasoconstrictive action [9] and by interfering with the vitamin-C metabolism which leads to a relative vitamin-C deficiency and diminished collagen cross-linking [10]. Other exogenous factors are comedications that are administered due to comorbidities like congestive heart failure or metabolic syndrome, namely angiotensin-converting enzyme inhibitors and statins. These have a well-proven impact on the activity of matrix-degrading enzymes like matrix metalloproteinase-2 (MMP-2) and on the collagen synthesis rate itself [11]. However, the data outlined above indicate that such confounding factors are not predominantly acting, as fibroblasts in cell culture retain the same alterations

of the collagen synthesis rate or the factors must have a long-lasting influence. Given epidemiological data and the observation that a collagen disease may prevail in some families, it is conceivable that a considerable effect and influence must reside within the genes.

What Is Known About Collagen Genes and Their Regulation?

The advent of molecular biology has revolutionized our understanding of diseases. The number of genetic diseases is still increasing as mutations within genes that are causative are found. The most studied collagen disease hitherto is the Ehler-Danlos syndrome in which a mutation within the coding sequence of the type collagen genes COL5A1 and COL3A1 are present [12, 13]. Other known collagen diseases are the Marfan syndrome, in which mutations of FBN1 on chromosome 15 or FBN2 on chromosome 5 are present [14] and the osteogenesis imperfecta with mutations in the COL1A1 gene [15]. For recurrent hernia disease there is no single mutation (e.g. single nucleotide polymorphism, SNP) known from the literature that is causative for disease; however, an in-depth understanding of the collagen gene synthesis requires that the respective regulatory elements relevant for gene transcription are known. Such elements may be localized in immediate context of the coding sequence, denoted as proximal promoter sequences, interspersed in the gene within introns, or they are localized in distance to the coding sequence, up to 20 kb and more [16]. Regulatory elements may act by enhancing or silencing gene transcription. It is notable that the different components of collagens, e.g. the alpha-1 and alpha-2 strand of the collagen type-I trimer are regulated co-ordinately at the transcriptional level [17]. Several groups have identified key regions for the transcriptional regulation of type-I and type-III collagen. These elements are bound by transcription factors, amongst others activating protein 1 (AP1), Sp1, Sp3, YB-1 and C/EBP [18–21], which are activated after cellular contact with cytokines like TNF-α, TGF-β, IFN-α. For the understanding of gene regulation, it is of paramount importance to keep in mind that the programs for gene regulation are mostly set up in a cell-specific fashion, meaning that fibroblasts may orchestrate collagen synthesis completely differently from macrophages, and knowledge from one cell type may not be transferred to another cell type.

Hypothetically, there are two different ways of altering the collagen gene regulation in a cell. The first disorder relates to an altered signalling within a cell, e.g. cytokine signalling transmitted to transcription factor activation. Such alterations of signalling may have the same impact as gene mutations within regulatory elements, which constitute the second possibility. Such mutations within regulatory elements may prevent binding of transcription factors or enhance their binding affinities. The complexity of the whole system cannot be stressed enough, given that the transcription initiation complex consists of at least 20 different proteins that have to gather around the transcription start site.

Are There Candidate Genes for Wound Healing Disturbances?

From other diseases with gradual and/or late onset in life, like arterial hypertension or diabetes mellitus, it might be anticipated that not a mono-causal genetic defect exists but rather a whole array of genetic alterations come together, making an individual susceptible for a certain disease. However, knowledge about such polygenetic traits has to be gathered before one may assume such a defect. Regarding collagen gene regulation, it is noteworthy that embryos with three copies of chromosome 18 (trisomy 18) have only type-III collagen in their skin, and lack type-I collagen [22]. Such a profound alteration of collagen type-III synthesis surprises, as the gene locus is located on chromosome 2q31 and the type-I collagen gene Col1A1 locus resides on chromosome 17q21.33, the Col1A2 on 7q22.1. One may postulate that there is an important genetic factor located on chromosome 18 and that this factor regulates the expression of the collagen genes inversely. The identification of chromosome 18 as dominantly acting on collagen expression renders all genes located on this chromosome interesting as candidate genes for wound-healing disturbances. Similarly, the collagen genes themselves and aberrations within their promoter sequences may be of relevance for wound-healing disturbances. Recent results from our group indeed indicate that such aberrations exist, that alter transcription rates of collagen genes and that might be useful as predictive markers for wound-healing disturbances.

References

1. Klinge U, Zheng H, et al. (1999) Altered collagen synthesis in fascia transversalis of patients with inguinal hernia. Hernia 4: 181–187
2. Klinge U, Si ZY, et al. (2000) Abnormal collagen I to III distribution in the skin of patients with incisional hernia. Eur Surg Res 32(1): 43–48

3. Stumpf M, Cao W, et al. (2001) Increased distribution of collagen type III and reduced expression of matrix metalloproteinase 1 in patients with diverticular disease. Int J Colorectal Dis 16(5): 271–275
4. Junge K, Klinge U, et al. (2004) Decreased collagen type I/III ratio in patients with recurring hernia after implantation of alloplastic prostheses. Langenbecks Arch Surg 389(1): 17–22
5. Wynes MW, Frankel SK, et al. (2004) IL-4-induced macrophage-derived IGF-I protects myofibroblasts from apoptosis following growth factor withdrawal. J Leukoc Biol 76(5): 1019–1027
6. Si Z, Bhardwaj R, et al. (2002) Impaired balance of type I and type III procollagen mRNA in cultured fibroblasts of patients with incisional hernia. Surgery 131(3): 324–331
7. Rosch R, Junge K, et al. (2003) Hernia a collagen disease? Eur Surg Res 35(1): 11 15
8. Waldorf H, Fewkes J (1995) Wound healing. Adv Dermatol 10: 77–96; discussion 97
9. Silverstein P (1992) Smoking and wound healing. Am J Med 93(1A): 22S–24S
10. Yin L, Morita A, et al. (2000) Alterations of extracellular matrix induced by tobacco smoke extract. Arch Dermatol Res 292(4): 188–194
11. Jesmin S, Sakuma I, et al. (2003) Role of angiotensin II in altered expression of molecules responsible for coronary matrix remodeling in insulin-resistant diabetic rats. Arterioscler Thromb Vasc Biol 23(11): 2021–2026
12. Zoppi N, Gardella R, et al. (2004) Human fibroblasts with mutations in COL5A1 and COL3A1 genes do not organize collagens and fibronectin in the extracellular matrix, down-regulate alpha2beta1 integrin, and recruit alphavbeta3 Instead of alpha5beta1 integrin. J Biol Chem 279(18): 18157–18168
13. Malfait F, De Paepe A (2005) Molecular genetics in classic Ehlers-Danlos syndrome. Am J Med Genet C Semin Med Genet 139(1): 17–23
14. Rokicka A, Rokicki W, et al. (2002) [Genetic aspects of Marfan syndrome]. Wiad Lek 55(1 2): 107–111
15. Qin W, He JX, et al. (2005) [Mutation detection of COL1A1 gene in a pedigree with osteogenesis imperfecta]. Yi Chuan Xue Bao 32(3): 248–252
16. Mautner J, Behrends U, et al. (1996) c-myc expression is activated by the immunoglobulin kappa-enhancers from a distance of at least 30 kb but not by elements located within 50 kb of the unaltered c-myc locus in vivo. Oncogene 12(6): 1299–1307
17. Norman JT, Lindahl GE, et al. (2001) The Y-box binding protein YB-1 suppresses collagen alpha 1(I) gene transcription via an evolutionarily conserved regulatory element in the proximal promoter. J Biol Chem 276(32): 29880–29890
18. Mertens PR, Harendza S, et al. (1997) Glomerular mesangial cell-specific transactivation of matrix metalloproteinase 2 transcription is mediated by YB-1. J Biol Chem 272(36): 22905–22912
19. Chen SJ, Artlett CM, et al. (1998) Modulation of human alpha1(I) procollagen gene activity by interaction with Sp1 and Sp3 transcription factors in vitro. Gene 215(1): 101–110
20. Dhalla AK, Ririe SS, et al. (1998) chk-YB-1b, a Y-box binding protein activates transcription from rat alpha1(I) procollagen gene promoter. Biochem J 336: 373–379
21. Mertens PR, Steinmann K, et al. (2002) Combinatorial interactions of p53, activating protein-2, and YB-1 with a single enhancer element regulate gelatinase A expression in neoplastic cells. J Biol Chem 277(28): 24875–24882
22. Brand-Saberi B, Epperlein HH, et al. (1994) Distribution of extracellular matrix components in nuchal skin from fetuses carrying trisomy 18 and trisomy 21. Cell Tissue Res 277(3): 465–475

Discussion

Kehlet: *I think you have opened up a new field in hernia surgery. Very important. If you can use this test, what will it cost?*

Mertens: *We are at a very early stage. We have to test this with a larger cohort of patients but I think it is very promising. When you do it like that you are in a range of 40–50 €. That's it.*

Franz: *Maybe I missed a slide. Where do the fibroblasts from the control patients come from? And where do the fibroblasts from the hernia patients come from?*

Mertens: *This is DNA extracted from blood. It is not DNA extracted from fibroblasts. Actually, this is a very interesting question we are addressing right now. Because when you are a mosaic, that means not everywhere in the body that microsatellite has to be the same. Maybe on the head it is different compared to the feet. At the side of your hernia where you have damaged your tissue it might be different. One of the aspects which is very interesting is that you told me smoking has a bad impact on recurrence rate and you do not lose that even if you are a long-term non-smoker. So maybe smoking affects a lot of things, like folat metabolism, like vitamin C metabolism and what we are just trying to address is whether this microsatellite instability is brought forward by these deficiencies so that you keep this trait, this genetic trait, lifelong, which would make sense. The other thing is age-dependent changes. You do not have the same incidence of recurrences at every age. So there must be something that changes with age.*

43 The Biological Treatment of the Hernia Disease

M.G. Franz

The biological treatment of hernias is perhaps best defined as a bio-molecular or biomechanical therapy that results in the complete and sustained correction of the structure and function of a hernia defect. Biological therapy of hernias could be applied as an adjuvant to surgical reconstruction, or, potentially stand alone as a primary therapy.

Growing evidence suggests that recurrent hernias are most often the result of surgical wound failure. Our group approaches the problem of incisional hernias with the hypothesis that the majority of incisional hernias develop following the mechanical disruption of early laparotomy wounds. By extension, most recurrent hernias are the result of mechanical wound failure during the lag phase of the wound healing trajectory (Fig. 43.1). Traditional surgical teaching is that laparotomy wound failure is a rare event, with reported dehiscence rates clustered around 0.1% [1–3]. The parallel wound-healing literature therefore suggested that incisional hernias were the result of late laparotomy wound failure and scar breakdown [4] This concept was first challenged by clinical studies of incisional hernias that recorded high primary and secondary recurrence rates after short-term follow-up, typically 2–4 years [5,

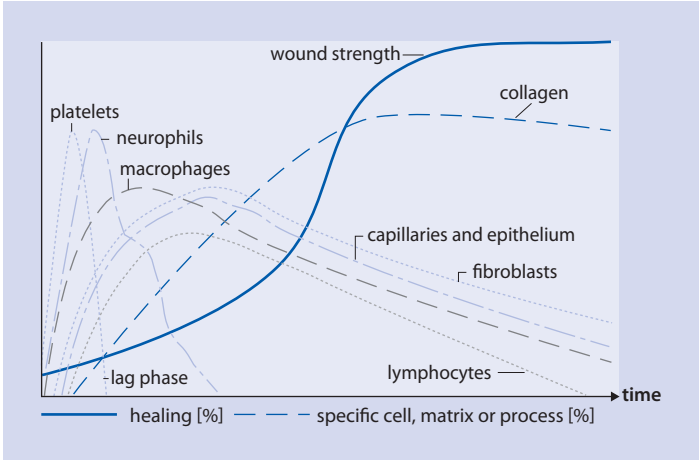


Fig. 43.1. The cellular and molecular pathways of surgical wound repair are time-dependent. During the initial lag-phase, wound tensile strength is low

Table 43.1. Human laparotomy wound-edge gap on post-operative day 30

Laparotomy wound outcome at 43 months	Less than 12 mm	More than 12 mm
Healed [%]	95 (140/147)	6 (1/18)
Incisional hernia [%]	5 (7/140)	94 (17/18)

6]. One prospective study found that the true rate of laparotomy wound failure is closer to 11%, and that the majority of these (94%) go on to form incisional hernias during the first 3 years after abdominal operations [2] (Table 43.1). The real laparotomy wound failure rate is therefore 100 times what most surgeons think it is. In simplest terms, most incisional hernias and recurrent hernias are therefore derived from clinically occult dehiscences. The overlying skin wound heals, concealing the underlying myofascial defect. This mechanism of early mechanical laparotomy wound failure is more consistent with modern acute wound healing science. There are no other models of acute wound healing suggesting that a successfully healed acute wound goes on to breakdown and mechanical fail at a later date. This mechanism is also unique in that it assumes that the majority of abdominal wall laparotomy wound failures occur in hosts with no clearly identifiable wound-healing defect. The majority of surgical patients undergoing laparotomy are considered safe surgical candidates from a wound-healing perspective.

The time-course of recurrent hernia formation is, however, debated [7, 8]. Incisional hernias detected many years after an operation have been ascribed to impaired collagen and tissue protease metabolism [9, 10]. Incisional herniation was associated with increased collagen solubility, increased ratios of early wound matrix collagen isoforms (e.g. collagen III) and increased metalloprotease 9 levels (MMP-9) [11, 13]. These studies were limited by the small numbers of patients and no confirmatory animal model data exist. A decreased ratio of type I: type III collagens was detected in fascial and skin specimens obtained from patients with incisional hernia disease at both the mRNA and the protein level [9, 14]. Morphological changes were present not only in the fascial tissue, but also in the hernia sac, skin specimens and scar tissue surrounding explanted meshes of hernia patients; collectively, the changes indicate a generalized alteration of collagen metabolism. MMPs are required for the proper progression and maturation of wound healing. Whereas MMP-2, -7, -8, and -9 are absent in healthy skin, MMP-1, -2, -3, -9, -11, -13, -14 are upregulated after injury. MMP-2 overexpression has been measured in fibroblasts of patients with direct inguinal hernia formation and MMP-13 overexpression was detected in specimens obtained from patients with recurrent inguinal hernias. In most cases, recurrent inguinal hernias most likely are a form of incisional hernia. Interestingly, there is a link between MMP-2 and collagen gene expression, as collagen binding to discoidin domain receptor II (DDR 2) regulates MMP-2 gene transcription [15]. Correspondingly, alterations in both MMP-2 and fibrillar collagen can interfere with the wound-healing process. Studies hypothesizing that hernia formation is a disease of the extracellular matrix (ECM) are a new approach to the biology of recurrent inguinal hernia and incisional hernia formation.

Surgical approaches to hernias are primarily based on mechanical principles. Fundamental techniques involve the sturdy apposition of durable aponeurotic tissues like the transversalis fascia, conjoined tendon, inguinal ligament, Cooper's ligament or rectus sheath. Consistently high recurrence rates led to the concept of a tension-free repair and the introduction of alloplastic mesh implants [16, 17]. The first suggestion of a biological approach soon followed, when it was hypothesized that part of the mechanism for the reduced recurrence rates observed with mesh implants was the avoidance or replacement of diseased or structurally defective tissue in hernia repairs. The current widespread use of plastic mesh implants has not, however, solved the problem of hernia recurrence. The problem is especially evident with recurrent incisional hernias, where population-based and administrative database analyses find a consistently high recurrence rate of between 24 and 58% and the more recent conclusion that the mesh repair of incisional hernias does not reduce the incidence of recurrence, but only increases the time-interval until the recurrence is recognized [18].

We hypothesize that the natural delay in acute wound healing prevents the establishment of adequate tensile strength within laparotomy wounds to off-set the loads placed across it by recovering surgical patients. An improved understanding of the cellular and molecular mechanisms of acute tissue repair now makes a biological approach to the problem of incisional hernia formation possible. Fundamentally, an incisional hernia occurs as the result of the biomechanical failure of an acute laparotomy wound. It is during the earliest period of acute wound healing when the wound depends

entirely on suture integrity to maintain abdominal wall closure. Recurrences following mesh repairs occur almost exclusively at the suture line interface between the implant material and the laparotomy wound. Numerous studies conclude that the breaking strength of mesh implants themselves far exceeds wound-breaking strength, and that, in fact, most of the mesh implants in clinical use are over-engineered [19]. It is an unusual event for a permanent mesh implant to mechanically fail. Most hernia repair patients are recovering from their procedures and returning to increased levels of activity and placing increasing loads across the acute wound during its weakest phase.

The potential for biomolecular intervention for surgical problems has improved with the continued maturation of applied biomolecular engineering and the wealth of data generated by the various genome projects. It is no longer impossible to imagine important applications of biotherapy in the field of wound healing (benign neoplasia or regenerative medicine) for unsolved problems like surgical wound failure (recurrent hernias), hypertrophic scar, bony non-union, gastrointestinal anastamoses, vascular endoleaks and intravascular stent patency.

Growth factors were one of the first groups of bioactive molecules identified for clinical application. Derived primarily from tissue macrophages and wound-repair fibroblasts, tissue growth factors are an important class of tissue repair, signaling peptides upregulated during the lag phase of acute wound healing [20, 21]. Five to seven days are required, however, before peak levels of fibroproliferative growth factors like transforming growth factor beta (TGF-β) are reached within acute wounds. Acute wound therapy with proliferative growth factors is known to accelerate the appearance of fibroblasts and collagen into the wound thereby shortening the natural lag phase for gain in injured tissue tensile strength. Several reports have demonstrated the ability of platelet-derived growth factor (PDGF) and TGF-β to accelerate the recovery of tensile strength in acute skin and laparotomy incisions [22–24].

A series of time-dependent cellular and molecular events must be activated and orchestrated during the establishment of a surgical wound matrix (Fig. 43.1). One strategy for the biological treatment of recurrent hernias would be to accelerate tissue repair, or shift the temporal wound-healing trajectory to the left (Fig. 43.2). It was already noted, that in addition to the natural delay in the activation of tissue repair, abnormal wound matrix structure may contribute to the mechanism of recurrent hernias. Biological therapies might also be designed to correct abnormal collagen isoform expression, or block overexpressed MMP activity. Ideally, normal aponeurotic, fascial, muscle or tendon structure would be regenerated following hernia repairs. Biological approaches for normal laparotomy wounds might be guided by information gained from identified genetic or epigenetic pathways associated with hernia formation like abnormal collagen matrix structure in the Ehlers-Danlos syndrome or MMP/TIMP expression in abdominal aortic aneurysm (AAA) disease or in other chronic wounds.

Exogenous application of cytokine growth factors has been used to accelerate healing of chronic wounds. Steed et al. first reported a successful clinical trial using recombinant platelet-derived growth factors (PDGF-BB) to accelerate healing of diabetic foot ulcers [25]. PDGF has also been reported to shorten the time of healing for

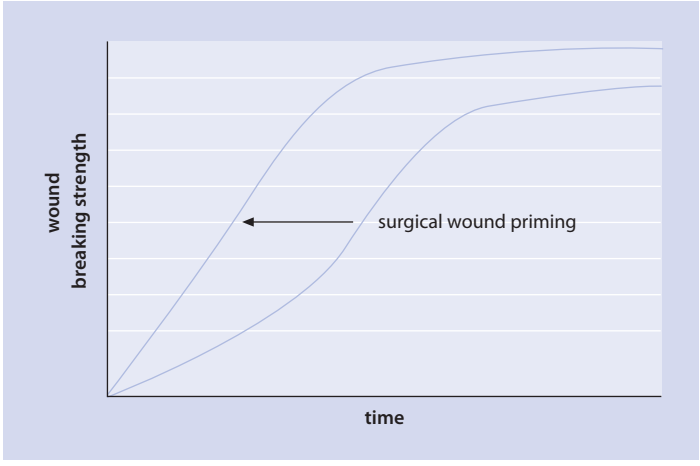


Fig. 43.2. Accelerating the pathways of surgical wound healing can be visualized as a shift-to-the-left of the wound-healing trajectory. We call this surgical priming

pressure ulcers. Robson et al. demonstrated that basic fibroblast growth factor (bFGF) could decrease the size of pressure ulcers more effectively than a placebo control [26]. Keratinocyte growth factor-2 (KGF-2) appears to have been effective in achieving closure in venous stasis ulcers, as has transforming growth factor beta-2 (TGF-β2). Bone morphogenetic proteins (BMP) are in the family of fibroblast growth factors and are now approved by the United States Food and Drug Administration (FDA) for the acceleration of interbody fusion following anterior diskectomy during spine operations [27].

Success in treating chronic wounds with exogenous cytokine growth factors has been disappointing. Despite numerous clinical trials, only one growth factor has been approved by the FDA for clinical use. There are several reasons for this. The chronic wound has a significant bacterial burden which is often not controlled prior to treatment with growth factors. Since bacteria degrade cytokine growth factors, larger amounts are required to be effective. In addition to the degradation of the cytokine growth factors, the bacteria produce proteases and MMPs both by themselves and as a product of bacterial action on tissue cells. These proteases and MMPs can degrade not only the growth factor molecules but also the cytokine receptors.

The acute wound, especially the surgical incision, is a closed wound with a bacterial burden 100–1000-fold less than the chronic wound [28]. The surgical wound may therefore be the ideal application for the tissue growth factors shown to accelerate the rate of gain of tensile strength in experimental incisions (Fig. 43.2). Preliminary studies showed that not only can growth-factor therapy be done at the time of surgical incision, it can be effective when initiating the inflammatory phase of healing prior to tissue injury by injecting cytokine growth factors in the area of the intended incision [23, 29]. Combining this pretreatment with a second growth-factor application at the time of incision nearly doubled the breaking strength of the acute wound (Franz, personal communication, **date??**).

Botsford first introduced the concept of accelerating surgical wound healing when describing delayed primary closures. It was observed that surgical wounds closed 7 days after incision regained tensile strength faster than incisions closed primarily [30]. The improved understanding of the cellular and molecular mechanism of surgical wound healing led to the proposal that it is the recruitment of repair cells and molecules into the open surgical incision that is the mechanism for accelerated repair following delayed primary closure. We call this phenomenon surgical priming. The availability of cloned biological signalling molecules now makes it possible to biopharmacologically induce the appearance of tissue repair cells, avoiding the practical difficulties associated with the surgical technique of delayed primary closure. The technique of delivering tissue repair growth factors at the time of incision is called simple priming and the technique of delivering the same growth factors several days prior to incision is called "early priming".

Growth factor strategies in hernia repairs:
- Accelerate recovery of wound tensile strength
- Optimize wound collagen structure and function
- Stabilize implant incorporation

Materials and Methods

We developed a model of incisional hernias in order to study the mechanism of acute wound healing during hernia formation and recurrence. We based our animal model of laparotomy wound failure and incisional hernia formation on the best available clinical evidence. Well-controlled, prospective studies conclude that most laparotomy wound disruptions progressing to incisional hernias begin to form soon after laparotomy wound closure [2, 31]. In the model, laparotomy wounds on the ventral abdominal wall of rats are temporarily repaired with rapidly absorbed plain 6–0 catgut suture. Laparotomy wound-edge separation occurs, progressing to incisional hernia formation due to incompletely supported mechanical loads. The incisional hernias that develop have well-defined hernia rings, hernia sacs and

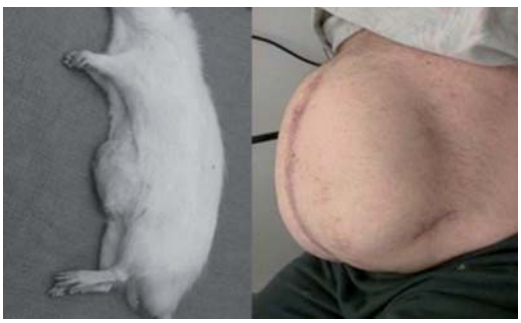


Fig. 43.3. In our animal models, mechanical failure of the laparotomy wound occurs and progresses to incisional hernias. The gross and microscopic structure is very similar to the human defect. In the rat, the skin flap serves as a sling to support the viscera for experiments requiring early disruption of the laparotomy wound

visceral adhesions, all characteristic of the incisional hernias that develop in humans (Fig. 43.3). In one variant of this model, the laparotomy incision is intentionally left open. These animals go on to form chronic, giant, incisional hernias [32]. We feel our model is an improvement over those that use abdominal wall excisions with inherent loss of abdominal wall muscular and tendinous structure. That approach does not control for the function of intact anatomical structures during laparotomy repair, including distractive load forces generated by the lateral oblique and midline rectus muscle and fascia components.

Aqueous Priming with TGF-beta2

Under our experimental protocol, 100 µl of vehicle (sodium acetate aqueous solution) or 100 µl of vehicle containing recombinant human TGF-β2 (1 µgm) (Genzyme, Framingham, MA) was injected evenly into the linea alba using a tuberculin syringe immediately prior to fascial incision (simple priming, Fig. 43.4). A 5.0-cm midline celiotomy was then performed and the laparotomy incision was immediately closed with a single, 5–0 plain catgut suture placed across the midwound, as described above.

All rats were sacrificed on postoperative day (POD) 28 by intraperitoneal Nembutal overdose (100 mg/kg). The entire ventral abdominal wall was then excised and the skin was separated from the musculo-fascial layer. The peritoneal and subcutaneous surfaces of each abdominal wall were then carefully inspected for the presence of herniated abdominal contents. A minimum of 2 mm fascial separation was required for a wound to be scored as containing an incisional hernia. Sagittal fascial wound and/or hernia sections were then cut and immediately fixed in 10% neutral buffered formalin in preparation for histological analysis.

Continuous Release Therapy with Basic Fibroblast Growth Factor

The first set of experiments was designed to test the activity of bFGF in preventing the development of incisional hernias. In the hernia prevention group (n = 20), a pliable, delayed-release polygalactone polymer-rod formulation of basic fibroblast growth factor (bFGF-rod; 10 µg, Scios Inc, Sunnyvale, CA) was sewn to the ventral edge the primary fascial wound closure (Fig. 43.5). Each rod was 5 cm in length and 1 mm in diameter. Previous experience in our laboratory established 10 µg

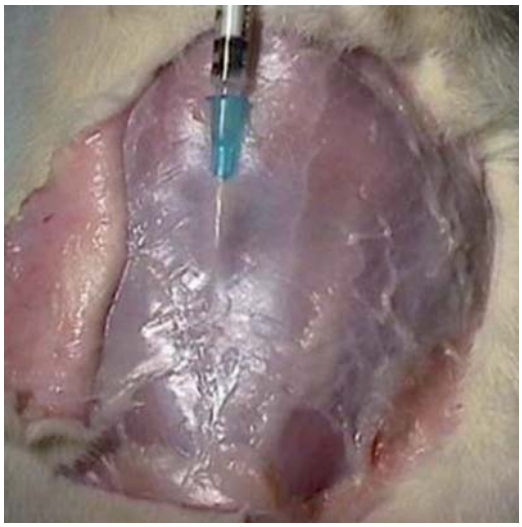


Fig. 43.4. During biological priming, a potential surgical wound site is treated with fibroproliferative and angiogenic tissue repair growth factors before the incision is made

as the optimal dose to stimulate the angiogenic and fibroplastic properties of bFGF in vivo. A growth factor delivery control group (n = 20) had rods without bFGF incorporated into the fascial closure (empty rod control). In the hernia model control group (n = 30), no rods were placed (hernia model control). The incidence of primary incisional hernia was determined during necropsies performed on postoperative day (POD) 28 on ten animals each in the bFGF rod treatment and empty rod control groups.

The recovery of fascial wound mechanical strength was measured on POD 7 using ten rats each from the bFGF-rod treatment and empty-rod control groups. The rats were sacrificed with an overdose of an intraperitoneal injection of Nembutal (100 mg/kg) and the entire ventral abdominal wall was excised. Following separation of the skin, the musculo-fascial layer was divided into three 1-cm-wide by 4-cm-long strips cut

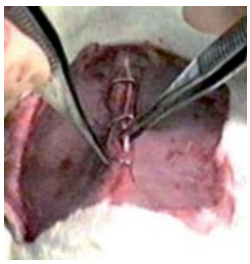


Fig. 43.5. A delayed-release polymer containing tissue repair growth factors can be incorporated into a surgical wound during hernia repairs

perpendicular to the fascial wound for biomechanical analysis. Breaking strength analysis was performed on the fascial wound strips using an Instron pneumatic tensiometer (Model MN44, Instron Corporation, Canton MA) set at a crosshead speed of 10 mm/min. The breaking strength was defined as the force in Newton's required to rupture the resultant incisional scar. Blood samples were obtained at necropsy and centrifuged for ELISA immunoassay analysis of serum bFGF levels (R&D Systems Inc. Minneapolis, MN).

The second set of experiments was designed to test whether bFGF augments the acute fascial repair of mature incisional hernias (recurrent hernia experiment). Twenty seven incisional hernias developed in the hernia model control group of 30 rats. The hernias were reduced and fascial defects repaired. The rats were then randomized to receive either bFGF rod hernia treatment (n = 13) or control empty-rod therapy (n = 14). The incidence of recurrent incision hernias was determined during necropsies performed 28 days after hernia repair. Several 2-mm biopsies taken of the fascial: fascial interface or hernia ring were snap frozen in liquid nitrogen for gene expression analysis. Inflammatory cell infiltrate and wound angiogenesis were measured using hematoxylin and eosin and trichrome staining. The intensity of wound collagen type-1 deposition was measured by quantifying the digital intensity of staining over three HPF using computer-aided image analysis software (Scion Image, Scion Corp., Bethesda, MD).

Statistical Analysis

Data were analyzed using SigmaStat software (Jandel Scientific, Corte Madera, CA). The Fisher's exact test was used to determine differences in the incidence of incisional hernias. Student's t test or ANOVA was used to determine differences in cell count, collagen staining, angiogenesis and collagen mRNA band intensity following RT-PCR. P values of < 0.05 were considered significant.

Results

TGF-beta 2

Incisional hernias developed in 88% (35/40) and 79% (11/14) of the untreated or vehicle-treated fascial incisions, respectively, after 28 days. No incisional hernias (0/16) occurred in the TGF-β2 treated wounds

Table 43.2. Growth factor priming (TGF)

Treatment modality	No.	Incisional hernias [%]
Untreated	40	35 (88)
Vehicle prophylaxis	14	11 (79)
TGF-β2 prophylaxis	16	0 (0)[a]

[a]Significantly less than untreated or vehicle prophylaxis.

(Table 43.2; $p < 0.05$). All animals gained weight during the study period and there was no difference in the mean weight between all three groups after 28 days (group 1 306 +/– 11.7 g vs. group 2 312 +/– 14.1 g vs. group 3 311 +/– 12.7 g). There were no deaths during the study period and no evidence of evisceration and or intestinal incarceration, obstruction or strangulation.

Cellular infiltration into the wounds on day 28 consisted mainly of macrophages, lymphocytes and fibroblasts. Fibroblast number after 28 days was higher within the TGF-β2-treated fascial incisions ($p < 0.05$). Trichrome and collagen immunostaining also was increased in fascial wounds treated with TGF-β2.

bFGF Delivery

Sustained delivery of bFGF was documented 7 days following surgical implantation. Analysis of POD 7 serum revealed a significant increase in systemic bFGF delivery compared to controls (0.033 μ + 0.017 bFGF rod treatment, 0.012 μ + 0.0097 empty rod control, and 0.010 μ + 0.008 hernia model control, $p < 0.05$)

Breaking Strength

All wounds mechanically disrupted at the fascial:fascial interface. Wounds treated with bFGF impregnated rods developed increased breaking strength in the early postoperative fascial incision. The bFGF rod-treated animals breaking strength was 12.29 N +/– 2.98 vs. 8.61 N +/– 3.17 in the empty-rod control animals' ($p < 0.05$).

Table 43.3. Growth factor priming (bFGF)

Treatment modality	No.	Incisional hernias [%]
Untreated	40	35 (88)
Placebo prophylaxis	10	6 (60)
bFGF prophylaxis	10	3[a] (30)
Placebo therapy	14	12 (86)
bFGF therapy	13	3[b] (23)

[a]for $p < 0.05$ compared to untreated; [b]for $p < 0.05$ compared to placebo therapy (Fisher's exact test)

Prevention of Incisional Hernia Development with bFGF

Incisional hernia formation was significantly reduced with bFGF rod therapy. Incisional hernias developed in 90% (27/30) of control incisions compared to 60% (6/10) in empty rod control-treated incisions, and only 30% (3/10) of the bFGF impregnated rod-treated incisions after 28 days ($p < 0.05$, Table 43.4). Full-thickness fascial sectioning perpendicular to the wound revealed the polymer rod incorporated into the provisional matrix. There were no deaths during the study period and no evidence of evisceration, intestinal incarceration, obstruction or strangulation. There were no obvious systemic effects of the bFGF observed in the postoperative period or at necropsy.

Treatment of Mature Incisional Hernias with bFGF

Incisional hernia recurrence was significantly reduced with bFGF rod therapy. The 27 incisional hernias that developed in the hernia model control group were operatively repaired 28 days after the initial procedure. Prior to repair, the rats were randomized to receive the 10 μg bFGF polymer or placebo polymer incorporated into the fascial closure. After another 28 days, 86% (12/14) of the empty rod control-treated hernia repairs developed recurrent hernias compared to only 23% (3/13) of the bFGF treated group ($p < 0.05$) (Table 43.3).

Inflammatory Infiltrate

Basic histology demonstrated that rods were still present, although approximately 50% smaller than when initially applied to the wound, demonstrating a slow absorption pattern of the polygalactone rod polymer. There was no difference in size between the bFGF-rod and empty-rod absorption or incorporation into the wound. There was a modest non-granulomatous inflammatory infiltrate surrounding the bFGF and empty-rod polymers. H&E staining suggested a gradient effect for polymorphonuclear leukocytes and macrophages in the bFGF-treated incisions.

Angiogenesis

Grossly, there was a visible increase in granulation tissue deposition adjacent to the bFGF rods when compared to the control animals. Dense capillary and provisional matrix deposition was observed in bFGF-treated incisions, resulting in macroscopic nodules similar to small hemangiomas. Microscopic neovascularization was most pronounced adjacent to the bFGF rods, and decreased as a function of distance from the rod.

Collagen Production

Basic FGF had no significant effect on wound collagen mRNA levels by POD 28. Histology and immunohistochemistry, however, demonstrated a gradient effect for fibroblast chemotaxis and collagen protein production in bFGF-treated incisions. Collagen I staining was significantly increased adjacent to the polymer by POD 7 ($103.72 \mu + 1.70$ vs. $24.42 \mu + 6.56$, $p < 0.01$). This intense collagen staining decreased as a function of distance from the rod.

Discussion

New approaches to the problem of abdominal wall wound failure are needed because the 11% incidence of primary and 24–58% incidence of recurrent incisional hernias remain unchanged over 50 years [5, 18, 33]. Mechanical approaches alone including optimized suture length to wound length ratios, incision location and orientation, layered versus mass closure and even mesh herniorrhaphy have all failed to significantly impact this very common surgical complication. Mesh herniorrhaphy is the only surgical

intervention that has decreased recurrent incisional hernia formation. However, the incidence of recurrent incisional hernias remains unacceptably high even following mesh repair (34–48%). The cloning of fibroproliferative tissue growth factors along with our improved understanding of the cellular and molecular mechanism of normal acute wound healing now make the biological augmentation of acute tissue repair a possible clinical alternative.

We developed a model of incisional hernia formation to better study and define the molecular mechanisms responsible for acute laparotomy wound failure. The model is based on early abdominal wall mechanical wound failure during the lag phase of acute healing when wound tensile strength is essentially zero. This results in an 80% incisional hernia rate after 28 days. In one experiment, incisional hernia formation was completely eliminated by priming the linea alba with aqueous TGF-β2 prior to midline celiotomy [29]. We have now repeated this study three times and believe that the overall reduction in the rate of recurrent hernias is 85% when priming with TGF-β2 (Franz, personal communication, 2006). The prevention of early acute wound failure was associated with enhanced macrophage and fibroblast chemotaxis in addition to increased collagen-I and -III production in TGF-β2-treated incisions. Two potential limitations identified with TGF-β2 were its biological property of inducing limited angiogenesis, and the technical problem of delivering aqeous suspensions to hernia wounds.

We then hypothesized that the combined fibroproliferative and angiogenic properties of bFGF would induce improved accelerated laparotomy and hernia wound repair. We also believed that a gradient release was important to the mechanism of action of tissue repair growth factors. To test this hypothesis, a bFGF-impregnated polymer was designed and incorporated into primary fascial closures and incisional hernia repairs. Early postoperative myofascial incisions recovered enhanced breaking strength at POD 7 in the bFGF therapy animals compared to the empty-rod controls, demonstrating accelerated early postoperative fascial wound repair. Prophylactic therapy of the celiotomy wound site with a delayed-release polymer formulation of bFGF resulted in a significant decrease in incisional hernia formation. Control animals had a 90% incidence of incisional hernia formation while bFGF-treated incisional hernia rate was only 30%. In addition to preventing incisional hernia formation, treatment of established incisional hernias with bFGF polymer resulted in a decreased recurrent hernia rate [34]. This distinction is important because a mature incisional hernia physiologically shares many characteristics of a chronic wound as opposed to the TGF-β2 experiments where an acute wound was biologically modified. Primary apposition of laparotomy or hernia edges with incorporation of "empty rods" in the controls resulted in an 86% recurrent incisional hernia rate, whereas bFGF impregnated rod therapy decreased the recurrent hernia rate to 23%.

Basic FGF treatment induced pronounced angiogenesis, earlier fibroplasia and enhanced collagen production within the laparotomy and hernia wounds. In vivo, bFGF appears in high initial peak concentrations in the early acute wound fluid (within 48 h), and then tapers off to baseline levels within a few days. This pharmacokinetic pattern is characteristic of insulin-like growth factor, epidermal growth factor, platelet-derived growth factor and transforming growth factor B-1. The early-response growth factors are known to recruit the cellular and molecular events necessary for the transition to the fibroproliferative stage. Specifically, bFGF stimulates endothelial cell proliferation and induces the production of proteases, required for neovessel growth through the extracellular matrix [34]. These biological functions theoretically make bFGF the ideal growth factor for signalling the remodelling of the mature, dense scar that characterizes hernia rings through protease activation and stimulation of neovascularization to support laparotomy wound healing following the apposition of hernia wound edges. Several other regenerative clinical applications for bFGF have been demonstrated in experimental animal models of acute wound healing in tissues with relatively poor blood supply. Incorporation of tracheal autographs, devascularized sternal wound healing, fractured bone healing and tendon repair all have been demonstrated to have enhanced wound healing with the application of bFGF to the acute wound [34].

Growth-factor delivery remains a difficult problem in models of acute wound healing. Applying growth factors as an aqueous preparation to an open incision makes containment within the wound difficult and equal distribution throughout the wound nearly impossible. Carboxymethylcellulose (CMC) suspensions improve the handling and delivery of growth factors for open-wound models, but are cumbersome and unreliable for closed primary wounds. Fibrin based carriers can also improve growth-factor delivery but may act as mechanical barriers to incision healing at specific concentrations. We developed the "priming" of the tissue prior to injury to allow ease of handling and to improve growth-factor delivery to the wounded tissue, but this first approach had the shortcomings of

a single-dose delivery and difficult application in the treatment of developed incisional hernias with separated fascial edges.

Semi-rigid polygalactone polymers aid in the delivery and handling of growth-factor preparations. The delayed-release formulation greatly improves sustained tissue infiltration, resulting in a better chemotactic gradient within the tissue when compared to wound-surface application alone. It is well known that a decreasing wound-tissue growth factor gradient is one of the important mechanisms for fibroblast and macrophage recruitment [35]. We measured sustained delivery of bFGF at 7 days after incorporation into the hernia incision, well beyond the time point when the physiological postinjury bFGF levels return to baseline. The main proposed drawback of semirigid rods is that unless the carrier polymer rapidly absorbs and is pliant, it may also act as a mechanical barrier to acute tissue approximation and repair. However, empty-rod therapy in this study resulted in a slightly decreased trend in the incidence of primary incisional hernia formation compared to the hernia model control animals (60% – 6/10 vs. 90% – 27/30). We observed a modest inflammatory reaction adjacent to the polymer which in itself stimulates angiogenesis and may account for the decreased incisional hernia trend observed with empty rod controls. Alternatively, the polymer rod may mechanically stent the fascial defect, resulting in this trend. In contrast to the hernia prevention studies, empty-rod therapy resulted in an 86% incidence of recurrent incisional hernia formation in the treatment of mature incisional hernias, similar to the control rate of 90%, suggesting no therapeutic benefit. Based upon these data, the mechanical presence of the polymer itself at least did not function to impede acute wound healing and may have a mild therapeutic effect on primary wounds.

The mechanism for the majority of recurrent hernias most likely involves early acute mechanical wound failure occurring at a time when acute fascial wound strength is less than 10% of normal tissue tensile strength. During this 4–6-week period, a celiotomy wound relies almost exclusively on the integrity of the suture line. If a suture fails, pulls through the wound and adjacent tissue or loosens as the patient recovers and increases activity, the natural load placed across the early acute wound will frequently lead to mechanical wound failure, occult fascial dehiscence and clinical hernia formation. Enhanced delivery of bFGF to the early postoperative wound functionally shortens the lag phase of tissue repair, thereby reducing the period of total mechanical reliance. Primary incisional hernia formation was decreased with bFGF, as was the incidence of recurrent incisional hernia development following repair. Not surprisingly, the mechanism includes enhanced fibroblast and macrophage recruitment into the region of the fascial incision as well as enhanced collagen and extracellular matrix synthesis, including markedly increased neovascularization. In the future, a combined biomechanical approach similar the one reported here may be applied clinically, especially in cases where there is a high risk of acute hernia wound failure. These would include, for example, recurrent herniorrhaphy, acute wounds in patients older than 60, those closed during hemodynamic instability or in the setting of abdominal sepsis, severely malnourished patients and those wounds associated with abdominal aortic aneurysm or morbid obesity surgery. Recurrent hernia wound therapy with proliferative growth factors such as bFGF offers a promising new strategy for reducing the incidence of this common surgical complication.

References

1. Brolin RE. Prospective, randomized evaluation of midline fascial closure in gastric bariatric operations. Am J Surg 1996; 172(4): 328–331
2. Santora TA, Rosylyn JJ. Incisional hernia. Hernia surgery. Surg Clin N Am 1993; 73(3): 557–570
3. Carlson MA, Ludwig KA, Condon RE. Ventral hernia and other complications of 1,000 midline incisions. Southern Med Jl 1995; 88(4): 450–453
4. Peacock Jr EE. Fascia and muscle. In: Peacock Jr EE (ed) Wound repair. 3rd edn. W.B. Saunders, Philadelphia, 1984, pp 332–362
5. Luijendijk RW, Hop WCJ, van den Tol MP, de Lange DCD, Braaksma MMJ, Ijezermans JNM, et al. A comparison of suture repair with mesh repair for incisional hernia. NEJM 2000; 343(6): 392–398
6. Korenkov M, Sauerland S, Arndt M, Bograd L, Neugebauer EAM, Troidl H. Randomized clinical trial of suture repair, polypropylene mesh or autodermal hernioplasty for incisional hernia. Br J Surg 2002; 89(1): 50–56
7. Bendavid R. The unified theory of hernia formation. Hernia 2004; 8(3): 171–176
8. Carlson MA. Acute wound failure. Wound healing. Surg Clin N Am 2001; 77(3): 607–635
9. Klinge U, Zheng H, Si ZY, Schumpelick V, Bhardwaj R, Klosterhalfen B. Synthesis of type I and III collagen, expression of fibronectin and matrix metalloproteinases -1 and -13 in hernial sac of patients with inguinal hernia. Int J Surg Invest 1999; 1(3): 219–227
10. Klinge U, Zheng H, Si ZY, Schumpelick V, Bhardwaj R, Klosterhalfen B. Synthesis of type I and III collagen, expression of fibronectin and matrix metalloproteinases -1 and -13 in hernial sac of patients with inguinal hernia. Int Jf Surg Invest 1999; 1(3): 219–227

11. Junge K, Klinge U, Rosch R, Mertens PR, Kirch J, Klosterhalfen B, et al. Decreased collagen type I/III ratio in patients with recurring hernia after implantation of alloplastic prostheses. Langenbecks Arch Surg 2004; 389(1): 17–22
12. Klinge U, Klosterhalfen B, Muller M, Schumpelick V. Foreign body reaction to meshes used for the repair of abdominal wall hernias. Eur J Surg 1999; 165(7): 665–673
13. Junge K, Klinge U, Klosterhalfen B, Rosch R, Stumpf M, Schumpelick V. Review of wound healing with reference to an unrepairable abdominal hernia. Eur J Surg 2002; 168(2): 67–73
14. Rosch R, Junge K, Knops M, Lynen P, Klinge U, Schumpelick V. Analysis of collagen-interacting proteins in patients with incisional hernias. Langenbecks Arch Surg 2003; 387(11 12): 427–432
15. Olaso E, Ikeda K, Eng FJ, Xu LM, Wang LH, Lin HC, et al. DDR2 receptor promotes MMP-2 mediated proliferation and invasion by hepatic stellate cells. J Clin Invest 2001; 108(9): 1369–1378
16. Usher FC. Repair of incisional and inguinal hernias. Surg Gynecol Obstet 1970; 131(3): 525–530
17. Lichtenstein IL, Shulman AG, Anderberg B, et al. The tension-free hernioplasty. Am J Surg 1989;157: 188
18. Flum DR, Horvath K, Koepsell T. Have outcomes of incisional hernia repair improved with time? A population based analysis. Ann Surg 2003; 237(1): 129–135
19. Klinge U, Klosterhalfen B, Conze J, Limberg W, Obolenski B, Ottinger AP, et al. Modified mesh for hernia repair that is adapted to the physiology of the abdominal wall. Eur J Surg 1998; 164(12): 951–960
20. Wahl SM, Wong H, Mccartneyfrancis N. Role of growth factors in inflammation and repair. J Cellular Biochemi 1989; 40(2): 193–199
21. Robson MC, Dubay DA, Wang X, Franz MG. Effect of cytokine growth factors on the prevention of acute wound failure. Wound Repair Regen 2004; 12(1): 38–43
22. Mustoe TA, Pierce GF, Thomason A, Gramates P, Sporn MB, Deuel TF. Accelerated healing of incisional wounds in rats induced by transforming growth factor-β. Science 1987; 237: 1333–1336
23. Smith PD, Kuhn MA, Franz MG, Wachtel TL, Wright TE, Robson MC. Initiating the inflammatory phase of incisional healing prior to tissue injury. J Surg Res 2000; 92: 11–17
24. Kuhn MA, Smith PD, Nguyen K, Ko F, Wang X, Franz MG. Abdominal wall repair is delayed during hepatic regeneration. J Surg Res 2001; 95: 54–60
25. Robson MC, Mustoe TA, Hunt TK. The future of recombinant growth factors in wound healing. Am J Surg 1998;176 (Suppl 2A): 80–82
26. Robson MC, Hill DP, Woodske ME, Steed DL. Wound healing trajectories as predictors of effectiveness of therapeutic agents. Arch Surg 2000; 135: 773–777
27. DuBay DA, Franz MG. Acute wound healing: the biology of acute wound failure. Surg Clin N Am 2003; 83: 463–481
28. Robson MC, Shaw RC, Heggers JP. The reclosure of postoperative incisional abscesses based on bacterial quantification of the wound. Ann Surg 1970; 171: 279
29. Franz MG, Kuhn MA, Nguyen K, Wang X, Ko F, Wachtel TL, et al. Transforming growth factor-b2 lowers the incidence of incisional hernias. J Surg Res 2001; 97: 109–116
30. Danielson CC, Fogdestam I. Delayed primary closure. Collagen synthesis and content in healing rat skin incisions. J Surg Res 1981; 31: 210–217
31. Franz MG, Smith PD, Wachtel TL, Wright TE, Kuhn MA, Robson MC. Fascial incisions heal faster than skin: a new model of abdominal wall repair. Surgery 2001; 129(2): 203–208
32. Dubay DA, Wang X, Adamson B, Kuzon WM, Jr., Dennis RG, Franz MG. Progressive fascial wound failure impairs subsequent abdominal wall repairs: a new animal model of incisional hernia formation. Surgery 2005; 137(4): 463–471
33. Cassar K, Munro A. Surgical treatment of incisional hernia. Br J Surg 2002; 89(5): 534–545
34. Dubay DA, Wang X, Kuhn MA, Robson MC, Franz MG. The prevention of incisional hernia formation using a delayed-release polymer of basic fibroblast growth factor. Ann Surg 2004; 240(1): 179–186
35. Banda MJ, Dwyer KS, Beckmann A. Wound fluid angiogenesis factor stimulates the directed migration of capillary endothelial cells. J Cell Biochem 1985; 29: 183–193

Discussion

Mertens: *I have two questions. How did you applied these growth factors?*

Franz: *In two different ways. The one is to simply inject them.*

Mertens: *The half-life of these growth factors is extremely short. Have you measured that? Because in our experience you have to use a continuous pump system to, for example, get damage in the kidney when you add substance. So have you checked for the half-life of these growth factors?*

Franz: *The ones which have been delivered by the bFGF rod we have indirectly measured because we analyzed systemic absorption, and over 3 days in the bFGF group there is a systemic absorption. It is indirect but showing us that at least there is a release.*

Mertens: *I think it is about 14 min.*

Franz: *Yes, but the difference compared to trying to induce scar in a kidney model is that we are trying to pre-activate the cells to be ready for tissue repair. But your point is well taken. Pharmaco-kinetics matters, but I refer you to Mustoe and PDGF, in Science 1999.*

Mertens: *Have you tested for macrophage influx, because TGF-beta is the most potent immunosuppressant agent?*

Franz: *Macrophages in which assay?*

Mertens: *No assay, if you knock out mice for TGF-beta they die because of an inflammatory response of the whole body. So have you checked for the macrophage influx and the number of them in this area?*

Franz: *I presented that TGF-beta slide that showed this; don't you agree that it looked like inflammatory cells?*

44 Pharmacological Treatment of the Hernia Disease

K. Junge, R. Rosch, M. Anurov, S. Titkova, A. Öttinger, U. Klinge, V. Schumpelick

Introduction

Hernia repair using mesh implants is one of the most commonly performed procedures in surgery. For incisional hernia repair, recurrence rates could be decreased to below 10% by means of supportive mesh materials [31]. Even in inguinal hernia repair, laparoscopic and open mesh techniques are nowadays widely accepted and increasingly performed. Classical studies concerning risk factors implicated in the development of primary and recurrent inguinal and incisional herniation usually focus on surgical techniques, the local anatomy, and physical alterations like raised intra-abdominal pressure and hernia size. Nowadays, the centre of attention has shifted to alterations of the extracellular matrix (ECM), its major component collagen and degrading matrix metalloproteinases (MMPs) leading to the occurrence of herniation [2,3,8,13,14]. Collagens represent the quantitatively most abundant protein of the body and are the most important scleroproteins of the ECM. Within the first phases of wound-repair processes, tissue formation is characterized by fibroplasias, neovascularization, migration and ECM production. Later phases of wound repair are characterized by matrix remodelling with the transformation of initial granulation tissue into connective tissue. The initially abundant immature type-III collagen is mainly replaced by mature type-I collagen during the physiological wound healing. The mechanical stability and tensile strength of connective and scar tissue increases due to intermolecular cross-linkage between collagen type I and type III [7,9,21]. Importantly, a decreased ratio of type-I to type-III collagen corresponds to a reduced tissue stability [4]. Concerning inguinal and incisional herniation, several studies have demonstrated alterations of the collagen concentration and ratio of collagen type I/III in fibroblasts isolated from skin and fascial scar [13,14,28]. A cohort study of 78 mesh prostheses implanted for repair of inguinal and incisional hernia samples that were subsequently removed because of hernia recurrence exhibited a significantly decreased collagen type-I/III ratio when compared with samples removed because of pain [11]. Altogether, an alteration of collagen metabolism with an insufficient scar formation and a disturbed balance of collagen type I and type III [10] has to be assumed as a major cause for the development of recurrent hernia. Considering the surgical challenge of recurrence treatment, further improvement of mesh materials to modulate tissue ingrowth seems pivotal. The aim of the present study in rats was to analyze the modulation of collagen quantity and quality of supplemented mesh samples with different pharmacological agents which are supposed to have an influence on scar formation.

Material and Methods

Mesh Materials

Mersilene mesh samples were used in this study. Before implantation, mesh samples (3×2 cm) were incubated for 30 min with the following agents: doxy-

cycline (20 mg/ml, Doxycyclin-ratiopharm, Ratiopharm GmbH, Ulm, Germany), TGF-β3 (0.1 µg/ml, Sigma-Aldrich, Steinheim, Germany), zinc-hydrogene-aspartate (3 mg/ml, Unizink, Koehler-Pharma GmbH, Alsbach, Germany), ascorbic acid (10 mg/ml, Canea Pharma GmbH, Hamburg, Germany) and hyaluronic acid (10 mg/ml, Sigma-Aldrich, Steinheim, Germany). Incubation with a physiological 0.9% NaCl solution served as control.

Animals

Thirty-six male Wistar rats (250–300 g) were housed under conditions of constant light and temperature and received a complete diet of rat feed and water ad libidum throughout the entire study, which was performed according to the rules of the Deutsche Tierschutzgesetz and to the NIH guidelines for the use of laboratory animals. The animals were randomly divided into three groups (n = six animals each).

Surgical Procedure

Following anaesthesia, the skin was shaved and disinfected with polividone-iodine solution. After midline skin incision and subcutaneous preparation of two lateral sides, the mesh samples (2×3 cm) were implanted. Skin closure was applied using a running 3/0 silk suture (Fig. 44.1).

Observation Periods

Three animals from each group (n = 6) were sacrificed for morphological observations 7 and 90 days after mesh implantation. Throughout the whole observation period all animals were objectively controlled and underwent daily clinical investigation to assess local and systemic complications.

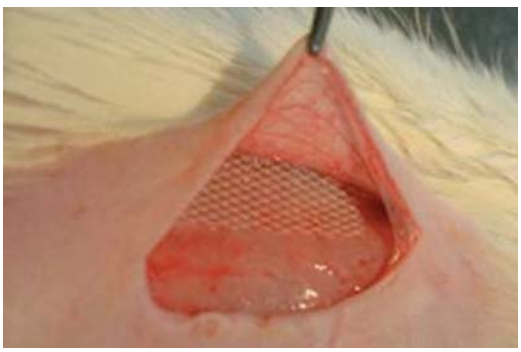


Fig. 44.1. Placement of the Mersilene mesh in subcutaneous position

Collagen/Protein Ratio

Specimens of paraffin-embedded tissue samples 15-µm thick were obtained from each group and placed in test tubes. After deparaffination, the slices were stained with Sirius red and fast green (Polysciences, Warrington, PA, USA). The specimens were rinsed several times with distilled water until the supernatant was colourless. Subsequently, the dyes were eluted from the sections by incubation with 0.1 N NaOH in absolute methanol. The fluid was read immediately in a spectrophotometer at the wavelengths corresponding to the maximal absorbance of Sirius red (535 nm) and fast green (605 nm). Results are expressed as the ratio of collagen (µg) to non-collagenous protein (mg) to level out the differences of weight of the slices and were performed with six samples in each animal [20].

Cross Polarization Microscopy

For cross-polarization microscopy (CPM) 5-µm sections were stained for 1 h in Picrosirius solution (0.1% solution of Sirius Red F3BA in saturated aqueous picric acid, pH 2) according to Junqueira [12]. The sections were washed for 2 min in 0.01 N HCl, dehydrated, cleared and mounted in synthetic resin. To analyze collagen type-I/III ratios, tissue samples were evaluated using cross-polarization microscopy (CPM). Thicker collagen type-I fibres were stained in red-orange shades, whereas thinner collagen type III appeared as pale-green shades. For each sample ten regions within the interface (400x, area 100 µm x 100 µm) were captured by a digital camera (Olympus C-3030, Hamburg, Germany). Collagen I/III ratios were obtained by analysis of the amount area of collagen type I and III using a digital image analyzing software (Image-Pro Plus, Media Cybernetics, Silver Spring, MD, USA). Results are expressed as ratio of area of collagen type I and type III.

Statistics

Statistical analysis was carried out using Statistical Package for Social Sciences (SPSS) software. Data was organized according to mesh modification and duration of implantation. Results were compared using an

independent t-test. P-values < 0.05 were considered to be significant. All data are presented as means ± standard deviation.

Results

Macroscopic Observations

Overall, macroscopic clinical observations after implantation of up to 90 days did not show haematomas, seromas or infections. One rat (TGF-β3) died because of an intraabdominal tumour and there was one mesh protrusion through the skin (ascorbic acid). Most animals (without relation to supplementation) showed local signs of abacterial inflammation (red skin) during the early postoperative period which disappeared later spontaneously.

Collagen/Protein Ratio

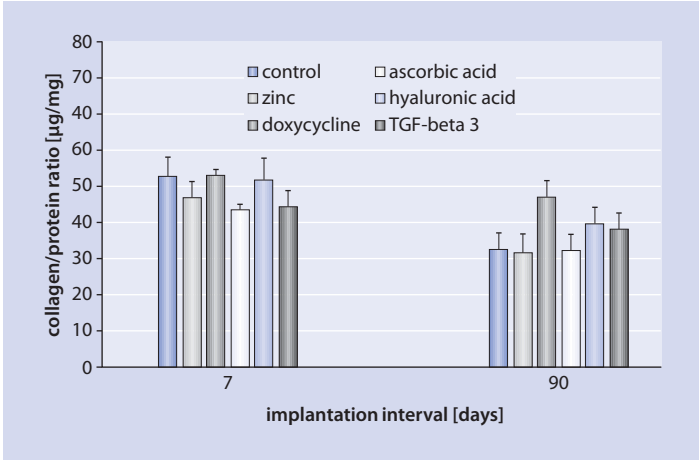
Quantity of collagen was analyzed investigating the collagen/protein ratio. Following an implantation interval of 90 days supplementation with doxycycline (39.3 ± 7.0 µg/mg) and hyaluronic acid (34.4 ± 5.8 µg/mg) was found to have a significantly increased collagen/protein ratio compared to implantation of the pure Mersilene mesh samples (28.3 ± 1.9 µg/mg; ◘ Fig. 44.2).

Cross Polarization Microscopy

Overall, an increase of the collagen type-I/III ratio was found in all groups, indicating scar maturation over time. However, no significant differences were found after 7 and 90 days of implantation comparing collagen type-I/III ratio of supplemented mesh samples and control group ($p < 0.05$, ◘ Fig. 44.3).

Discussion

Up to now, the number of studies investigating the effect of a local administration of bioactive substances to improve wound healing and scar formation has been rather limited. A significantly decreased incisional hernia formation has been reported by Robson et al. [5, 30] with local administration of transforming growth factor beta 2, basic fibroblast growth factor (bFGF) and interleukin-1β. Primary incisional hernia formation was decreased, as was the incidence of recurrent incisional hernia development with bFGF-coated rods. The improved wound healing was associated to an enhanced fibroblast and macrophage recruitment in the region of the fascial incision as well as to an enhanced collagen and extracellular matrix synthesis with markedly increased neovascularization [5, 30]. On the other hand, Korenkov et al. were not able to find an augmentation of the anterior abdominal wall using local application of transforming growth factor beta 1 [15]. Unfortunately, none of the above-mentioned studies analyzed quality of scar formation and collagen type-I/III ratio expressed. Furthermore, up to now there is no bioactive mesh material available leading to an improved mesh integration modulating a depressed collagen type-I/III ratio. Therefore, different bioactive agents which have a documented influence on wound healing were supplemented to a multifilamentous Mersilene mesh and quantity as well as quality of collagenous mesh integration analyzed.



◘ Fig. 44.2. Collagen/protein ratio following supplemented Mersilene mesh implantation compared to control

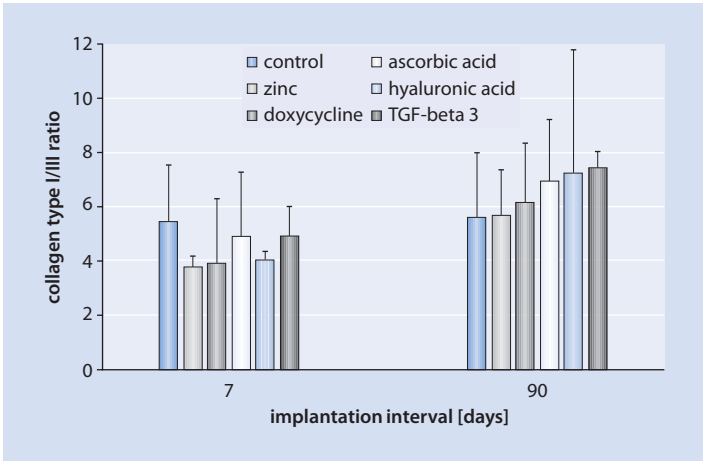


Fig. 44.3. Collagen type I/III ratio following supplemented Mersilene mesh implantation compared to control

Topical zinc is widely used in wound treatment although the beneficial effect of zinc has been documented only in zinc-deficient patients who were given zinc orally. Whereas the impact of zinc on collagen-degrading enzymes (MMPs) is described in detail, till today no data are available investigating the influence of zinc supplementation on collagen type-I/III ratio. Norman et al. analyzed the development of tensile strength in incised wounds in rats and guinea pigs at 7 and 14 days after wounding in animals given supplements of zinc salts by either the oral or parenteral route. No difference in tensile strength was observed at these times in the wounds of either rats or guinea pigs given zinc supplements [27]. Agren reported a stimulated leg ulcer healing by enhancing re-epithelialization, decreasing inflammation and bacterial growth [1]. However, no significant effect was observed comparing collagen quantity and qualitity of the zinc supplemented and the pure Mersilene mesh sample.

Tetracyclines have been shown to inhibit the activity of mammalian matrix metalloproteinases, i.e. type I collagenase (MMP-1) and type IV collagenase/gelatinase (MMP-2) [32]. Lauhio et al. demonstrated that a 2-month regimen of doxycycline can reduce MMP-8 levels in serum and especially in body fluids (i.e. saliva) containing inflammatory exudates and thus may contribute to reduced tissue destruction in reactive arthritis [18]. In Toluene diisocyanate-induced asthma doxycycline significantly reduced airway inflammation, airway hyperresponsiveness, and reduced expression of MMP-9 mRNA and protein [19]. Whereas no effect could be observed analyzing collagen type-I/III ratio, the overall collagen deposition measured using the collagen/protein ration was significantly higher following doxycycline supplementation of the mesh samples. These data are in accordance with findings of Lamparter et al., who found a higher collagen concentration in doxycycline-treated rats [17].

Vitamin C (ascorbic acid) is required for the growth and repair of tissues in all parts of body. It is necessary to form collagen and essential for the healing of wounds, and for the repair and maintenance of cartilage, bones and teeth. A severe form of vitamin C deficiency is known as scurvy, which mainly affects older, malnourished adults. Moreover, Vaxman et al. found out that vitamin C increased the collagen synthesis-associated metals Fe, Cu and Mn levels in the healing process of colonic anastomoses. Vitamin C enhanced the colonic wound-healing process in the rabbit, acting together in synergy in vivo as well as in vitro [33]. Even local application of ascorbic acid induces benefical effects including promoting of collagen synthesis, photoprotection from ultraviolet A and B and improvement of a variety of inflammatory dermatoses [6]. Whereas Zhang et al. found a stimulated cell proliferation, type-I collagen and alkaline phosphatase synthesis in vitro for ascorbic acid released from a glycerol-polyethylene glycol matrix, no significant effect on collagen/protein and collagen type-I/III ratio was observed following the implantation of supplemented mesh samples within our study.

Hyaluronic acid, also known as hyaluronan or HA, is a major component of the extracellular matrix and plays an important role in tissue repair. It is known to influence a number of events critical to successful wound healing, including inflammation, cell migration, angiogenesis, re-epithelialization and scar formation. Due to hyaluronidase activity and metabolism by cells,

naturally occurring hyaluronan has a short residence in tissues. Studies in animals showed that hyaluronan accelerated wound healing in both rats [29] and in the cheek pouches of hamsters [16]. Although hyaluronan is known to play an important role in wound healing, clinical experience with this polymer is limited. However, positive results have been recorded in a number of indications, and hyaluronan has been used successfully for many years in ophthalmology and the treatment of joint conditions [26]. It has also been used to ameliorate wound healing in the treatment of venous and mixed etiology leg ulcers and diabetic foot ulceration, post-surgical wounds and burns [34, 34]. The extracellular matrix of fetal rabbit wounds contains an abundance of the hyaluronic acid but is devoid of excessive collagen. Thus, fetal wounds heal without scarring with a tissue repair resembling regeneration [23]. Mast et al. observed an increased fibroblast infiltration, collagen deposition and capillary formation in hyaluronidase-treated wounds [22]. Despite these facts, we found a significantly increased collagen/protein ratio at the interface without any significant changes in the collagen type-I/III ratio in hyaluronan supplemented mesh materials.

Following injury with the adhesion, aggregation and degranulation of circulating platelets within the forming fibrin clot, a plethora of mediators and cytokines are released, including transforming growth factor beta (TGF-β). Recent studies, particularly in genetically manipulated animal models, have highlighted the impact of TGF-β on various aspects of wound healing and, surprisingly, not all of its effects are conducive to optimal healing. Acute wound therapy with proliferative growth factors is known to accelerate the appearance of fibroblasts and collagen into the wound thereby shortening the natural lag phase in injured tissue tensile strength. Several reports have demonstrated the ability of TGF-beta 3 to accelerate the recovery of tensile strength in acute skin and laparotomy incisions [25]. However, there were no significant effects applying TGF-beta 3 during mesh implantation within our animal model.

In summary, in our study about the impact of supplemented mesh materials, the investigated bioactive agents with reported influence on wound healing were not associated with an improved scar formation. Further research should focus on a controlled temporary drug release to optimize local administration of bioactive substances as well as an investigation of additional agents which are probably suitable for an optimized integration of foreign-body materials.

References

1. Agren MS. Studies on zinc in wound healing. Acta Derm Venereol Suppl (Stockh) 1990; 154: 1–36
2. Bellon JM, Bajo A, Ga-Honduvilla N, Gimeno MJ, Pascual G, Guerrero A et al. Fibroblasts from the transversalis fascia of young patients with direct inguinal hernias show constitutive MMP-2 overexpression. Ann Surg 2001; 233(2): 287–291
3. Bellon JM, Bujan J, Honduvilla NG, Jurado F, Gimeno MJ, Turnay J et al. Study of biochemical substrate and role of metalloproteinases in fascia transversalis from hernial processes. Eur J Clin Invest 1997; 27(6): 510–516
4. Birk DE, Mayne R. Localization of collagen types I, III and V during tendon development. Changes in collagen types I and III are correlated with changes in fibril diameter. Eur J Cell Biol 1997; 72(4): 352–361
5. Dubay DA, Wang X, Kuhn MA, Robson MC, Franz MG. The prevention of incisional hernia formation using a delayed-release polymer of basic fibroblast growth factor. Ann Surg 2004; 240(1): 179–186
6. Farris PK. Topical vitamin C: a useful agent for treating photoaging and other dermatologic conditions. Dermatol Surg 2005; 31(7 Pt 2): 814–817
7. Friedman DW, Boyd CD, Mackenzie JW, Norton P, Olson RM, Deak SB. Regulation of collagen gene expression in keloids and hypertrophic scars. J Surg Res 1993; 55(2): 214–222
8. Friedman DW, Boyd CD, Norton P, Greco RS, Boyarsky AH, Mackenzie JW et al. Increases in type III collagen gene expression and protein synthesis in patients with inguinal hernias [see comments]. Ann Surg 1993; 218(6): 754–760
9. Henkel W, Glanville RW. Covalent crosslinking between molecules of type I and type III collagen. The involvement of the N-terminal, nonhelical regions of the alpha 1 (I) and alpha 1 (III) chains in the formation of intermolecular crosslinks. Eur J Biochem 1982; 122(1): 205–213
10. Jansen PL, Mertens PP, Klinge U, Schumpelick V. The biology of hernia formation. Surgery 2004; 136(1): 1–4
11. Junge K, Klinge U, Rosch R, Mertens PR, Kirch J, Klosterhalfen B et al. Decreased collagen type I/III ratio in patients with recurring hernia after implantation of alloplastic prostheses. Langenbecks Arch Surg 2004; 389(1): 17–22
12. Junqueira LC, Cossermelli W, Brentani R. Differential staining of collagens type I, II and III by Sirius Red and polarization microscopy. Arch Histol Jpn 1978; 41(3): 267–274
13. Klinge U, Si ZY, Zheng H, Schumpelick V, Bhardwaj RS, Klosterhalfen B. Abnormal collagen I to III distribution in the skin of patients with incisional hernia. Eur Surg Res 2000; 32(1): 43–48
14. Klinge U, Zheng H, Si Z, Schumpelick V, Bhardwaj RS, Muys L et al. Expression of the extracellular matrix proteins collagen I, collagen III and fibronectin and matrix metalloproteinase-1 and -13 in the skin of patients with inguinal hernia. Eur Surg Res 1999; 31(6): 480–490
15. Korenkov M, Yuecel N, Koebke J, Schierholz J, Morsczeck C, Tasci I et al. Local administration of TGF-beta1 to reinforce the anterior abdominal wall in a rat model of incisional hernia. Hernia 2005; 9(3): 252–258

16. Krasner Ri, Young G. Role of hyaluronidase and the hyaluronic acid capsule in the survival and dissemination of group A streptococci in the hamster cheek pouch. J Bacteriol 1958; 76(4): 349–354
17. Lamparter S, Slight SH, Weber KT. Doxycycline and tissue repair in rats. J Lab Clin Med 2002; 139(5): 295–302
18. Lauhio A, Konttinen YT, Tschesche H, Nordstrom D, Salo T, Lahdevirta J et al. Reduction of matrix metalloproteinase 8-neutrophil collagenase levels during long-term doxycycline treatment of reactive arthritis. Antimicrob Agents Chemother 1994; 38(2): 400–402
19. Lee KS, Jin SM, Kim SS, Lee YC. Doxycycline reduces airway inflammation and hyperresponsiveness in a murine model of toluene diisocyanate-induced asthma. J Allergy Clin Immunol 2004; 113(5): 902–909
20. Lopez-De Leon A, Rojkind M. A simple micromethod for collagen and total protein determination in formalin-fixed paraffin-embedded sections. J Histochem Cytochem 1985; 33(8): 737–743
21. Madden JW, Peacock EE, Jr. Studies on the biology of collagen during wound healing. I. Rate of collagen synthesis and deposition in cutaneous wounds of the rat. Surgery 1968; 64(1): 288–294
22. Mast BA, Flood LC, Haynes JH, DePalma RL, Cohen IK, Diegelmann RF et al. Hyaluronic acid is a major component of the matrix of fetal rabbit skin and wounds: implications for healing by regeneration. Matrix 1991; 11(1): 63–68
23. Mast BJHTKRDIC. In vivo degradation of fetal wound hyaluronic acid results in increased fibroplasia, collagen deposition and neovascularisation. Plast Reconstr Surg 1992; 89,3:503–509
24. Moore AR. Hyaluronan--a review of the recent patent literature. IDrugs 2000; 3(2): 198–201
25. Mustoe TA, Pierce GF, Thomason A, Gramates P, Sporn MB, Deuel TF. Accelerated healing of incisional wounds in rats induced by transforming growth factor-beta. Science 1987; 237(4820): 1333–1336
26. Niethard FU. Pathogenesis of osteoarthritis--approaches to specific therapy. Am J Orthop 1999; 28(11 Suppl):8–10
27. Norman JN, Rahmat A, Smith G. Effect of supplements of zinc salts on the healing of incised wounds in the rat and guinea pig. J Nutr 1975; 105(7): 822–826
28. Pans A, Albert A, Lapiere CM, Nusgens B. Biochemical Study of Collagen in Adult Groin Hernias. J Surg Res 2001; 95(2): 107–113
29. Ren GY, Dong FS, Wang J, Shi PK. [The effect of hyaluronic acid external film on rats wound healing]. Zhonghua Zheng Xing Wai Ke Za Zhi 2004; 20(5): 380–383
30. Robson MC, Dubay DA, Wang X, Franz MG. Effect of cytokine growth factors on the prevention of acute wound failure. Wound Repair Regen 2004; 12(1): 38–43
31. Schumpelick V, Conze J, Klinge U. [Preperitoneal mesh-plasty in incisional hernia repair. A comparative retrospective study of 272 operated incisional hernias]. Chirurg 1996; 67(10): 1028–1035
32. Suomalainen K, Sorsa T, Ingman T, Lindy O, Golub LM. Tetracycline inhibition identifies the cellular origin of interstitial collagenases in human periodontal diseases in vivo. Oral Microbiol Immunol 1992; 7(2): 121–123
33. Vaxman F, Chalkiadakis G, Olender S, Maldonado H, Aprahamian M, Bruch JF et al. [Improvement in the healing of colonic anastomoses by vitamin B5 and C supplements. Experimental study in the rabbit]. Ann Chir 1990; 44(7): 512–520
34. Vazquez JR, Short B, Findlow AH, Nixon BP, Boulton AJ, Armstrong DG. Outcomes of hyaluronan therapy in diabetic foot wounds. Diabetes Res Clin Pract 2003; 59(2): 123–127

Discussion

Kehlet: *If the experimental model is appropriate to study the role of vitamin C and zinc supplementation, because in humans we know that many elderly patients are relatively insufficient in these substances, although you have a negative experimental trial it probably has to be tested in the clinical situation.*

Junge: *Yes, of course, we have to try to study it in the clinical situation but till today there are no relevant data available.*

Franz: *So I should come to Aachen and learn more about the thought behind the collagen isoforms. I love these papers but isn´t it normal for a wound healing to have in the early period a greater amount of collagen type-III isoform? Look at your time course. You have a 7- and up to 90-day time course in which the mesh was implanted. Is that not too soon, isn´t it is just a normal time response.*

Junge: *This 90-day implantation interval is part of our standardized animal model. Of course I do not know what will happen after, for example, 2 years with this kind of mesh. We are trying right now to investigate long-term studies with this mesh but there is an effect compared to the unsupplemented mesh and I suppose there will be an effect after 2 years as well. Concerning these early changes, we recently had a published study in The Netherlands and they showed us that using CT scans you already can see that there is a dehiscense of the linea alba right after the operation and so using this early effect of this supplemented mesh it will probably work.*

Franz: *Exactly, if it is a normal response to have an inversion of these collagen isoforms, how would an intervention like this affect that?*

Junge: *I actually do not understand your question. We performed controls as well and what we found was a significant difference comparing the control group and the supplemented mesh group. The control group has to be regarded as the normal response.*

Sarr: *Dr. Mertens, could you come up to the microphone as well. I give you the hypothesis that I think we are working from the wrong direction. We are trying to deal with an abnormal response of the human body that is a healing*

to a surgical scar. Should we not talk about tissue regeneration? I maintain that a surgical scar is not a normal response from the body. We are getting out of it by putting prosthesis in it and trying to decrease the inflammatory cell response and the scar response. Should we not try to regenerate normal tissue.

Junge: *Probably gentamicine is able to regenerate normal tissue.*

Sarr: *But you have mesh in there.*

Junge: *We additionally try to investigate the gentamicine effect using just suture techniques. We just inject this gentamicin and see what will happen.*

Mertens: *I completely agree, but this is not a physiological situation and the question is whether the human body has a capability to respond to this injury. What I find very interesting, that with mesh you have a chronic foreign body reaction which persists and Dr. Lynen has shown that macrophages like this chronic reaction. They go there, they stay there and there is a response which is ongoing. The nice thing is and I believe in all the data hinting a deficient collagen type I/III composition can be reproduced under different conditions, but I am not sure whether you can transfer all these date from the animal to the human situation. This is my problem that I have with most of the experiments. But this substance of gentamicine is something that we also use for different diseases where translation of mRNA is changed like cystic fibrosis. There are clinical trials proving that it has a major impact so I believe what Dr. Junge has proposed is a very nice concept to alter the natural response.*

Franz: *Again, what I was trying to say is what he just said. Isn't is just a normal response or is it really something more.*

Junge: *I think it is something more. We in our study tried to regenerate the local scar following mesh implantation and this is what we got.*

Schumpelick: *I think the question of Prof. Sarr is justified. We are on a wrong level of healing. Is there any chance to go back to fetal healing? They do not have this problem. Anything concerning sterm cells?*

Junge: *If think there will be o role of sterm cells in future but it will take some more time. And concering the mesh materials I would like to stress, that of course it would be the best to have an absorbable mesh who is supplemented by this gentamicine and following regeneration of the tissue will disappear and everything is ok. This mesh works just as a carrier of our substances.*

Concluding Recommendations to Prevent the Recurrence

45 Questionnaire (39 Participants)

1. Total Number of Hernias Performed/Year

- Inguinal 9075
- Incisional 2232

2. Percentage of Operations Performed

- Inguinal
  - Lichtenstein 35.4 %
  - Shouldice 24.2 %
  - TAPP 7.8 %
  - TEP 17.6 %
  - TIPP-Rives 1.4 %
  - Stoppa 1.0 %
  - Wantz 1.0 %
  - Ugahary 0.0 %
  - Plug 0.6 %
  - Others 11.1 %
- Incisional
  - Sublay 51.6 %
  - Onlay 6.8 %
  - Inlay 0.0 %
  - IPOM 27.4 %
  - Others 14.2 %

3. Type of Mesh Actually Used (Multiple Answers Possible)

	Inguinal	Incisional	Hiatal
Polypropylene (Atrium, Marlex)	63.4%	56.1%	–
Low weight Polypropylene (Vypro, UltraPro)	46.3%	46.3%	12.1%
Polyester (Mersilene)	4.9%	9.8%	–
ePTFE (DualMesh)	2.4%	29.3%	9.8%
Others	9.8%	9.8%	–

4. Do You Think That Operations Performed Without Any Technical Failure Are Able to Eliminate Recurrences in All Cases?

- Yes 10.3%
- No 89.7%

5. Is There Any Prophylactic Use of Meshes Imaginable?

- Yes 90.3%
- No 9.7%

6. Which Incision Is Preferable for an Elective Laparotomy?

- Midline 58.5%
- Transverse 41.5%
- Paramedian 12.2%
- Oblique 0.0%

7. Which Incision Should Be Avoided?

- Midline 17.1%
- Transverse 2.4%
- Paramedian 34.2%
- Oblique 36.6%

8. Mesh Repair for Incisional Hernias

A. How should the overlap at the rib cage be dealt with?
- Sublay 58.5%
- Onlay 12.2%
- Periosteum suture fixation 9.8%
- Suture around rib 22.0%

B. How should the overlap at the xiphoid be dealt with?
- Sublay 78.0%
- Onlay 12.2%
- Periosteum suture fixation 9.8%

C. How should the overlap at the pubic bone be dealt with?
- Sublay 51.2%
- Onlay 9.8%
- Periosteum suture fixation 7.3%
- Fixation to Coopers ligament 56.1%

D. How should the overlap lateral to the rectus sheath be dealt with?
- Preperitoneal 34.1%
- Between muscles 51.2%
- Onlay 19.5%

E. How should a mesh be fixed?
- Non-absorbable suture 46.3%
- Absorbable suture 39.0%
- Glue 7.3%
- No fixation at all 14.6%

F. Which overlap to all sides should be achieved in incisional hernia repair?
- < 2 cm 0.0%
- 2-5 cm 28.6%
- > 5cm 71.4%

9. Mesh repair for inguinal hernias

A. Do you believe in one single standard procedure or a tailored surgery?
- Single standard 27.5%
- Tailored approach 72.5%

B. Does every hernia (even in young patients with a small lateral hernia) demand a mesh repair?
- Yes 36.6%
- No 63.4%

C. What do you think are the major reasons for the constantly high recurrence rates reported by health care analyses (multiple answers possible)?
- Poor technical skill 92.7%
- Insufficient teaching 85.4%
- Patients´ biology 53.7%

D. Which patient related factors do you think are of importance for the development of a recurrent hernia (multiple answers possible)?
- Gender 46.3%
- Age 53.7%
- Weight 70.7%
- Affected relatives 31.7%
- Medication 41.5%
- Smoking 87.8%

E. Are there any limitations (hernia size, location, previous surgery) that restrict the use of the following mesh technique?
- Lichtenstein Yes 55.8% No 44.1%
- TAPP Yes 93.3% No 6.7%
- TEP Yes 90.3% No 9.7%
- TIPP-Rives Yes 87.1% No 12.9%
- Plug Yes 90.0% No 10.0%

F. Which overlap to all sides should be achieved in the inguinal area?
- < 2 cm 7.5%
- 2–5 cm 80.0%
- > 5cm 12.5%

D. Which factors are related to hiatal hernia recurrence?
- Poor technical skill 63.4%
- Insufficient teaching 46.3%
- Patients´ biology 36.6%

8. Mesh Repair for Hiatal Hernias

A. Does every hiatal hernia demand a mesh repair?
- Yes 9.4%
- No 90.6%

B. The hiatoplasty should be performed?
- anterior 0.0%
- posterior 66.7%
- both 33.3%

C. Is excision of the hernial sac mandatory?
- Yes 80.0%
- No 20.0%

Summary

Today surgical repair of abdominal wall offers a huge variety of different techniques, mostly using meshes as reinforcement. However, the problem of recurrence still exist, either due to technical mistakes or to a patient related inadaequate wound healing. More than 50 international experts joined the 4rd Suvretta meeting to discuss their experience with creating, avoiding and repairing recurrent hernia. This book summarizes the today risks and opportunities of most current techniques to treat hiatal hernia as well as incisional or inguinal hernia. The lively discussions reflect controversies and reveal open questions, that have to be examined in future.

Appendix

Subject Index