Volume 17 Jul - Dec 2009 Number (3-4)

Submit Manuscripts: Editorial office
330 El Sudan Street, Embaba , Egypt
Email : jegyptscts@gmail.com
Tel. 3303 8054 - Mob. 0102327650

EDITORIAL BOARD

Journal of The Egyptian Society of Cardio-Thoracic Surgery

Volume 17 Jul - Dec 2009 Number (3-4) ISSN 1110-578X

CONTENTS

Journal of The Egyptian Society of Cardio-Thoracic Surgery

Volume 17 **Jul - Dec 2009** **Number (3-4)** **ISSN 1110-578X**

Guidelines for Authors

Journal of The Egyptian Society of Cardio-Thoracic Surgery
(J. Egypt. Soc. Cardiothorac. Surg.)

Editorial Office
Please address all correspondence to:
Yasser M W Hegazy MD,FRCS, Editor in Chief
Journal of The Egyptian Society of
Cardio-Thoracic Surgery
330 El-Sudan St., Imbaba, Cairo, Egypt.
Telephone: (+201)06045640
Fax: (+202) 303 8054
E-Mail: hegazyyasser@gmail.com

The Journal of the Egyptian Society of Cardio-Thoracic Surgery [ISSN 1110-578 X] is the official publication of the Egyptian Society of Cardio-thoracic Surgery. The journal is published every three months .

General Instructions

Every submission must include:
Cover letter, indicating the category of article , the Complete manuscript, including title page, abstract, text, tables, acknowledgments ,references and illustrations .

Required Disclosures;

A. Conditions for Publication Form which includes disclosures regarding freedom of investigation and conflicts of interest, signed by all authors. In single Author publication an additional Senior Consultant Signature is required.
B. Written permission from the publisher (copyright holder) is required to reproduce any previously published table(s), illustration(s) or photograph(s) in both print and electronic media.
C. Written permission from unmasked patients appearing in photographs is also required.

Revised_Manuscripts:
Revised manuscripts must be submitted in
three parts as Microsoft word-processing files : (1) cover letter with responses to reviewers' comments (2) revised, marked manuscript showing additions and deletions; (3) revised, unmarked manuscript.

General Information
Three copies of the Manuscripts should be sent preferably prepared in Microsoft Word , typed double-spaced throughout (including title page, abstract, text, references, tables and legends) with one (1) inch (2.5 cm) margins all around. Place Author name and page number in the upper right corner of each page.
Manuscripts written in 12 point Arial or Times New Roman fonts are preferred (Note: Do not submit your manuscript in PDF format it causes problems in processing your submission.)
Arrange manuscript as follows: (1) title page, (2) abstract, (3) text, (4) acknowledgments, (5) disclosures if required, (6) references, (7) tables and Figures (8) legends. Number pages consecutively, beginning with the title page as page 1 and ending with the legend page.
If your manuscript contains illustrations, in addition to submitting them online, you must send two sets of original illustrations to the editorial office labeled with manuscript number, first author, and figure number on back.
Tables and figures should be provided separate from the text while there position in the text should be marked on the manuscript.

Word Limits by Category of Manuscript

Original articles should not exceed 4500 words including title page, abstract of 150-250 words, text, figure legends and references. The combined total of illustrations and tables should not exceed 10 and the number of references should not exceed 40.

Case reports and **"The way I do it"** articles are limited to a total of 1500 words including title page, abstract, text, references and figure legends. For each illustration subtract 100 words and for each table subtract 300 words from the word limit. References are limited to eight. A "how to do it" article should be a description of a useful surgical technique and contain descriptive, illustrative material.

Images in cardiothoracic surgery are limited to 350 words including title and text and to two, possibly three figures. The entire contribution must fit on one printed page .

Review articles are limited to 6500 words including title page, abstract, text, figure legends and all references. The total number of references should not exceed 80. Subtract 100

words for each illustration and 300 words for each table.

Our surgical heritage articles are limited to 2500 words including title page, abstract, text, figure legends and references. Subtract 100 words for each illustration and 300 words for each table.

Correspondence (Letters to the Editor) and commentaries are limited to 500 words. Subtract 100 words for each illustration and 300 words for each table.

Editorials are limited to 2500 words including references. Subtract 100 words for each illustration and 300 words for each table.

Manuscript Preparation

Title Page (first page)

The title is limited to 100 characters and spaces for original manuscripts and to 80 characters and spaces for all other categories of manuscripts. The title may not contain acronyms or abbreviations. All submissions, must have a title.

Running Head. Supply a short title of 40 characters and spaces.

Authors. List all authors by first name, all initials, family name and highest academic degree using "MD, PhD" for holders of both degrees (if more then 7 Authors justifie).

Institution and Affiliations. List the name and full address of all institutions where the work was done. List departmental affiliations of each author affiliated with that institution after each institutional address.

Meeting Presentation. If the paper has been or is to be presented at the annual meeting of The Society, provide the name, location and dates of the meeting.

Keywords. Provide up to 5 keywords selected from the appended list to describe the manuscript. Do not use any keywords that are not on the list.

Word Count. Provide the electronic total word count of the entire manuscript including title page, abstract,text,figure legends and entire reference list.

Corresponding Author. Provide the name, exact postal address with postal code, telephone number, fax number and e-mail address of the author to whom communications, proofs and requests for reprints should be sent.

Abstract Page (Second page)

Original articles
Provide a structured Abstract, no longer than 250 words, divided into four sections: Background or Objective, Methods, Results, Conclusions. Avoid abbreviations and acronyms. Indicate the abstract word count below the abstract.

Case reports, "the way i do it" articles, review articles and our surgical heritage articles. Provide an unstructured abstract of 100 words.

Images, correspondence, commentaries, editorials and updates. No abstract is required.

Text
Text should be organized as follows: Introduction, Methods, Results, Discussion Then references,illustrations and tables in numeric order by order of mention in the text.

Avoid abbreviations. Consult the American Medical Association Manual of Style, 9th edition, for recommended abbreviations. Define abbreviations at first appearance in the text. If 8 or more abbreviations or acronyms are used, provide a separate table of abbreviations and acronyms.

Measurements and weights should be given in standard metric units.Statistical nomenclature and data analysis. Follow the "Guidelines for Data Reporting and Nomenclature" published in The Annals of Thoracic Surgery (1988;46:260-1). Footnotes. Type footnotes at the bottom of the manuscript page on which they are cited. Suppliers of drugs, equipment and other brand mentioned in the article within parentheses , giving company name, city and country .

Acknowledgments
Grants, financial support and technical or other assistance must be acknowledged at the end of the text before the references.

References
Identify references in the text using Arabic numerals in brackets on the line.
Type references double-spaced after text or acknowledgments beginning on a separate sheet. Number consecutively in the order in which they appear in the text. Journal references should provide inclusive page numbers; book references should cite specific page numbers. Journal abbreviations should conform to those used in Index Medicus. follow the formats outlined below:

Journal Article
Jones DR, Stiles BM, Denlinger CE, Antie P . Pulmonary segmentectomy: results and complications. Ann Thorac Surg 2000;76:343-9.(List all authors if 6 or fewer; otherwise list first 3 and add "et al.")

Chapter in Book
12. Vinten-Johansen J, Zhao Z-Q, Guyton RA. Cardiac surgical physiology. In: Cohn LH, Edmunds LH Jr, eds. Cardiac Surgery in the Adult. 2nd ed. New York, NY: McGraw-Hill; 2003:53-84.

Internet Address

3. 1996 NRC Guide for the Care and Use of Laboratory Animals. Available at: http://www.nap.edu/readingroom/books/labrats/contents.html. Accessed October 20, 2003.

Tables :

Tables should be typewritten double-spaced on separate sheets (one to each page). Do not use vertical lines. Each table should be numbered (Arabic) and have a title above. Legends and explanatory notes should be placed below the table. Abbreviations used in the table follow the legend in alphabetic order. Lower case letter superscripts beginning with "a" and following in alphabetic order are used for notations of within-group and between-group statistical probabilities.

FigureLegends :

Figure Legends should be numbered (Arabic) and typed double-spaced in order of appearance beginning on a separate sheet. Identify (in alphabetical order) all abbreviations appearing in the illustrations at the end of each legend. Cite the source of previously published material in the legend and indicate permission has been obtained. Proof of permission must be surface mailed or faxed to the editor .

Illustrations :

You must send two sets of original illustrations to the editorial office labeled with manuscript number, first author, and figure number on back.

Images or figures are submitted online as one or more separate files that may contain one or more images. Within each file containing images, use the figure number (eg, Figure 1A) as the image filename. The system accepts Powerpoint (.ppt) files Most illustrations will be reproduced at a width of one column (8.25 cm; 3 1/4 inches). Black, white and widely crosshatched bars are preferable; do not use stippling, gray fill or thin lines.

Instructions :

Identify print proofs of figures on the back with figure number and name of the first author; when necessary, indicate the top with an up arrow

For figures submitted in electronic format, all images should be at least 5 inches wide. Graphics software such as Photoshop and Illustrator, should be used to create art.

Color images need to be at least 300 dpi.

Gray scale images should be at least 300 dpi .

Line art should be at least 1200 DPI .

Cover letter :

Include with the manuscript a cover letter that provides 1) the category of manuscript (e.g., original research, Brief Communication, Letter to the Editor); 2) statement that the material has not been previously published or submitted elsewhere for publication; 3) information about any personal conflicts of interest of any of the authors; and 4) names of sources of outside support for research, including funding, equipment, and drugs .You may also submit the name of one reviewer of your choice. You should include that individual's mailing address, telephone, fax and e-mail address.

Editorial Policies

Scientific Responsibility Statement

Before publication of an accepted manuscript, each author is required to certify by signing the Conditions for Publication Form that he or she has participated sufficiently in the work and approved the final version of the manuscript to be published.

Exclusive Publication Statement

Each author must certify that none of the material in this manuscript has been published previously in either print or electronic form, and that none of this material is currently under consideration for publication elsewhere. This includes symposia and preliminary publications of any kind except an abstract of 400 words or fewer.

Conflict of Interest :

Authors should disclose any conflict of interests. Authors who have a financial relationship with one or more companies whose products are featured in an article will disclose the existence of this relationship in a box at the bottom of the first page of the published article.

Consultant Statistician and Statistical Methods :

All manuscripts with statistical analysis are required to undergo biostatistical review .The most appropriate way is to involve a biostatistician consultant or coauthor from the investigators' home institution . Manuscripts may undergo further biostatistical review by the Journal after submission. Additional information on statistical methods can be found in "Uniform Requirements for Manuscripts Submitted to Biomedical Journals"(www.acponline.org/journals/resource/unifreqr.htm).

Copyright :

Authors of articles submitted to The J. Egypt. Soc. Cardiothorac. Surg. must transfer copyright to The Egyptian Society of Cardio-Thoracic Surgery by signing the "Conditions for Publication Form." This transfer becomes binding upon acceptance of the article for publication. No part of the published material may be reproduced elsewhere without written permission.

Date of Receipt: The "received for publication" date is the date when the editorial office receives the manuscript, the cover letter, and the Copyright Transfer and Author Declaration Statement, signed by all authors.

For Date of acceptance : letter is provided from the editor.

Checklist

A] Cover Letter

□ Letter to the Editor
□ Manuscript category designation .
□ Single-journal submission affirmation .
□ Conflict of interest statement (if appropriate).
□ Sources of outside funding.
□ Signed Statistical Collaboration .

B] Complete Manuscript

□ Title page .
□ Title of article
□ Full name(s), academic degrees, and affiliation(s) of authors.
□ Corresponding author .
□ Telephones, fax, and e-mail address
□ Abstract (250 words; double-spaced) .
□ Ultramini-abstract (50 words) .
□ Text (double-spaced).
□ References (double-spaced; separate pages).
□ Tables (double-spaced; separate pages).
□ Figures (separate files; on hardcopy; properly identified),
□ Figure legends (double-spaced; separate pages) .
□ Word count.

C] Required Disclosures

□ Conditions for Publication Form signed by all authors. Which transfers copyright to The Egyptian Society of Cardio-Thoracic Surgery
□ Written permission from the publisher to reproduce any previously published material .
□ Written permission from unmasked patients .

Conditions for Publication Form

This form MUST be completed, signed by ALL authors, and returned to the Editorial Office before your manuscript can be accepted for publication.

Scientific Responsibility Statement:

Each author must sign this form to certify that he or she has participated sufficiently in the work to take responsibility for a meaningful share of the content of the manuscript, and that this participation included: (a) conception or design of the experiment(s), or collection and analysis or interpretation of data; (b) drafting the manuscript or revising its intellectual content; and (c) approval of the final version of the manuscript to be published. In addition, each author must indicate whether or not he or she has had full freedom of investigation; defined as freedom from outside interests in controlling the design of the study, collection, analysis, and interpretation of data, and having freedom to full disclose all results.

Exclusive Publication Statement:

Each author must sign this form to certify that none of the material in this manuscript has been published previously in either print or electronic form, and that none of this material is currently under consideration for publication elsewhere. This includes symposia, transactions, books, articles published by invitation and preliminary publications of any kind except an abstract of 400 words or fewer.

Copyright Transfer Agreement:

Each author must sign this form to certify that, if the manuscript is accepted for publication in the Journal of the Egyptian Society of Cardio-Thoracic Surgery (JESCTS), copyright (including the right to obtain copyright registration, whether separately or as part of a journal issue .) in and to the above article transfers throughout the world and for the full term and all extensions and renewals thereof to: THE EGYPTIAN SOCIETY OF CARDIO-THORACIC SURGERY

This transfer includes the right to adapt the article for use in conjunction with computer systems and programs, including reproductions or publication and incorporation in retrieval systems.

Rights of authors:

The ESCTS hereby licenses the following rights back to the author(s):

A. Patent and trademark rights to any process or procedure described in the article.

B. The right to photocopy or make single electronic copies of the article for their own personal use, including for their own classroom use, or for the personal use of colleagues, provided the copies are not offered for sale .

C. The right, subsequent to publication, to use the article or any part thereof free of charge in a printed compilation of works of their own, such as collected writings or lecture notes.

Note:

All copies, paper or electronic, or other use of the information must include an indication of The ESCTS copyright and a full citation of the journal source.

Authorship:

If copyright is held by the employer, the employer or an authorized representative of the employer must sign in addition to the author(s).

Warranties:

The author(s) warrant that the article is the author's original work and has not been published before. The author(s) warrant that the article does not infringe on the rights of others. If excerpts from copyrighted works are included, the author(s) has (have) obtained written permission from the copyright owners and will credit the sources in the article.

Preprints:

The author(s) warrant(s) that if a prior version of this work (normally a preprint) has been posted to an electronic server, such version was accessible to only a small group of individuals and the author(s) will cause its prompt removal from such server.

Conflict of Interest Disclosure Statements:

Each author must indicate below that either (a) no financial conflict of interest exists with any commercial entity whose products are described, reviewed, evaluated or compared in the manuscript, except for that disclosed under "Acknowledgements" or (b) a potential conflict of interest exists with one or more commercial entities whose products are described, reviewed, evaluated or compared in the manuscript through the existence of one or more of the following relationships: the author is a full or part-time employee of a company; has an existing or optional equity interest in a company; owns or partly owns patents licensed to a company; has an ongoing retainer relationship (consultantship, speaker, etc.) with a company for which he/she receives financial remuneration; or has received financial compensation for this publication. If Yes is checked, a box on the first page of the published article will read: ?Dr. X discloses that he/she has a financial relationship with company Y.?

Electronic publishing

Tips for preparing Word documents

1 Learn how to use the Word features under the Tools/ Autocorrect submenu. Some people turn off all autocorrection features because they are disconcerted by Word's default behaviour of adjusting capitalisation and reformatting type on the fly, but these features save a lot of time once you tune them in to match your expectations. In particular, if you have a long word like 'hypergammaglobulinaemia' that you need to type repeatedly, turn on `Replace text as you type' and add it to the replacement list.

2 Keep formatting to a minimum. editors, much prefer manuscripts in a simple one-column layout. Only use fonts that everybody has on their computers: for example, Times New Roman for your main text font and Arial as your font for headings. Turn off type justification, automatic hyphenation, and automatic paragraph numbering. On the other hand, the use of bold, italic, superscript, and subscript text as appropriate is good.

3 Use styles and style tagging rather than formatting the article paragraph by paragraph. This makes it much easier to format an article as you write and easier again if you are asked to change the formatting later. For your level I headings, therefore, define a Heading l style, with the combination of font, spacing, and alignment that you want to use, and then apply this to each heading as you create it. To change all your level I headings later, simply redefine the style and all will be changed without having to select and manipulate each heading.

4 Format text as one continuous flow. Use a page break (Ctrl + Enter) to start a new page (e.g. after your title page) not a stream of hard returns. put only one hard return between each paragraph,. Do not break the article up with Word's section breaks.

5 Keep table formatting simple and consistent. A common error is to place a column of separate items into a single table cell, with each item separated by a hard return: instead each data item should have a table cell of its own. Sometimes tables are formatted with tabs instead of cells: in this case, the key is to set the tab stops for the whole table so that one tab equals one column.

6 Image files should be sent as separate files. The same goes for Excel spreadsheets or charts. If you are embedding images in the file, it is probably best to do it at the end, after the text and references.

7 Be prepared to send the data used to generate graphs. Some publishers will use the data to regenerate the graphs according to their own style rules. In such a case, it helps if you send only the data that are actually shown in the graphs – not the spreadsheet with all of the data generated in the study.

What about PDF?

Send your Manuscript in a Word file. Don't send it as PDF or any other word processor format.

PDF files are not editable in the same way as word processor files. Some publishers will ask for, or even create, a pdf file of your manuscript for use during the peer review process, but a Word file will also be required for editing and production.

Tips for preparing images

Do not make electronic images too small . No effective way exists to increase the resolution of an image beyond its original size, and if an image is reduced in size and saved, picture data is permanently lost. Image files therefore have to be created and saved at high resolution. For a colour image that is to be printed as 4 X 4 in., the required size is (4 X 300) X (4 X 300) = 1200 X 1200 = 1440 000 dots. In many image formats (e.g. tagged image file format, or tiff), each dot will take eight bits (one byte) to store, so the image file will be 1.44 megabytes

Compression techniques can reduce the size of the image file. Zip compression is safe, because it uses an algorithm that packs the data tighter without throwing any of it away; In Compression during which files are saved in jpeg format ,select the option for large file size (maximum picture quality).

Guidelines for Reviewers

Purpose of Peer Review

One is to evaluate objectively the science of the submitted paper and the other is to provide a constructive critique indicating how the paper could be or could have been improved by the authors. Reviewers should avoid unpleasant comments.

Acceptance of a Manuscript for Review

Reviewers should accept assignments to review manuscripts that are within their sphere of expertise, which they plan to review within the 21 day deadline. Reviewers should decline assignments for which a conflict exists between the reviewer and authors or between the reviewer and commercial products that are integral to the content of the article.

Category of the Manuscript

The broad categories of papers for which peer review is undertaken are original scientific articles; new technology papers; case reports, the way i do it articles , images; and review articles. The editor and/or associate editors review correspondence, invited commentaries, editorials, surgical heritage , ethical and statistical papers.

General Requirements for Publication

The paper should conform to the format and restrictions for the category to which it belongs and be written in good, readable English. The paper should address an important or interesting subject and provide new and original information. Illustrative material should be well chosen and of good quality.

Original Scientific Article

The reviewer should assess the articles' interest to readers; strengths and weaknesses; originality; clarity of text, tables, illustrations and figure legends; presentation; analysis of results; credibility of results; importance of the findings; depth of scholarship and relationship of the results to the existing literature Ethical issues, such as prior publication of all or part of the data; plagiarism; transgression of human or animal rights; or dishonesty should be noted, if detected.

The following topics are offered to help guide the reviewer's assessment of an original scientific article.

- 'Title' should reflect the content of the article and be concise and clear
- 'Abstract' should indicate the purpose of the study, subjects and methods used, most important results and the main conclusions supported by results.
- 'Introduction' should indicate the rationale and focus of the study and state the purpose or hypothesis.
- 'Methods' should present the design of the study, fully describe the number and subjects and exclusion and inclusion criteria; whether subjects were enrolled consecutively; methods used to gather data, including follow-up data; methods by which control and experimental groups were assembled; the primary outcome variable; secondary outcome variables; how outcome measurements were made and validated; the statistical design of the study; and the statistical methods used to analyze the study.
- 'Results' should concisely present the most important findings in text . Data should be reported as means or medians with appropriate indicators of variance and exact p values in tables and text. Figures should be well selected to highlight important findings . Survival and event curves should indicate specified confidence limits or subjects at risk. Regression diagrams should include the regression equations, regression coefficient and exact p value in the figure legend. Figure legends should adequately and clearly describe the important information illustrated.
- 'Comment' should not repeat results, but should point out the significance and conclusions of the new data, integrate the authors' new data with that in the prior literature, draw inferences and conclusions regarding the question or purpose addressed by the study and point out the limitations of the study. The 'Comment' section should not be a review of the literature.
- References should be properly cited, reasonably current, accurateand in proper format.

New Technology

Articles describing new technology are necessarily descriptive and do not pose or test a hypothesis. These articles evaluate new devices, systems, monitors, implantable material and similar technology designed for improving patient care and outcomes. The reviewer is asked to evaluate the efficacy, safety and indications of the new technology .

The reviewer needs to inspect the 'Disclosure statement' after the text, before References. This statement should disclose the source of funds used for the evaluation study and whether or not the product was purchased, borrowed or donated by the manufacturer or inventor. Conflicts of interest statements for authors are managed by the editorial staff.

Case Reports, The Way I Do It, Images

Case reports describe interesting presentations of disease and innovative management of the patient's or patients' problem. How to Do It articles emphasize innovations in the operative management of technical challenges and new ways of doing things. Images, which must fit on one printed page, are graphics of interesting presentations of disease within the chest.

Reviewers should evaluate the clarity and completeness of the case or procedure descriptions and the selection and quality of the illustrative material. Reviewers should also note whether or not the paper adheres to the format restrictions enumerated in "Information for Authors". The reference list should be se-

lective rather than inclusive.

Review Article

Reviewers should assess the importance of the subject matter, need for the review and probable interest to readers. Reviews of very rare and unusual diseases are discouraged . Reviewers should note if authors have respected the format and restrictions of this category as stated in "Information for Authors".

The 'Introduction' should provide the rationale for reviewing the subject matter and provide the outlines of what is included and not included in the review. In the 'Methods' section reviewers should assess the methods used to search for articles, including search words and databases probed. The body of the review should be well organized with well chosen topical headings arranged in logical order. Within each topical heading the material should be presented in an integrated, comprehensive, objective manner. Statements should be referenced accurately. Reviewers should look for a "summing up" of the topical content .

The review should provide a general overview of the subject matter assessing progress, pointing out deficiencies in present management and indicating opportunities and directions of future work. The reviewer should also assess the selection of references .

Footnote

The reviewer remains anonymous . The reviewer should direct his or her critique to the authors in the style and format that suits them best. The recommendation to the editor is made separately with or without additio

Events of Interest

The 17th Annual Conference of the Egyptian Society of Cardiothoracic Surgery
Cairo - Egypt (National Heart Institute)

Timing : ..... 8-10 April 2010
Location: ..... Cairo J.W Marriot
Email : ..... jegyptscts@gmail.com

■ **International Joint Meeting on Thoracic Surgery-Barcelona, Spain—November 25-27,**

2009 more information on this meeting, contact Oriol Seto, Acto Serveis, C/ 3onaire, 7, 08301 Matar6, Barcelona, Spain; telephone: 34-937-552-382; fax: ;4-937-552-383; e-mail: thoracic.surgery@actoserveis.com

■ **Surgery of the Thoracic Aorta: November 30-December 1, 2009 Italy**

Fifth Postgraduate Course-Bologna, Italy— telephone: +39 051385 fax: +39 051 221894

■ **139 th Critical Care Congress , Florida – January 13 , 2010**

more information on this meeting -contact the Society of Critical
Care Medicine Customer Service, 500 Midway Dr, Mount Prospect, IL 60056; telephone: (847) 827-6888; fax: (847) 827-6886; e-mail: info@sccm.org; website: www.sccm.org

■ **42nd Annual New York cardiovascular Symposium, New York- December 11-12, 2009**

New York City The Housing Connection _. West Temple, Suite 140,
Lake City, UT 84101; telephone: 505-4606; fax: (801) 355-0250; -- email: thc@housingregistration.

■ **International Symposium on Endovascular Therapy-Hollywood, Florida—January 17-21,2010**

For more information on this meeting, contact Complete Conference Management, 11440 N Kendall Dr, Suite 306, Miami, FL 33176; telephone: (888) 334-7495; fax: (305) 2798221; e-mail: questions@ccmcme. com; website: www.iset.org.

■ **46th Annual Meeting of The Society of Thoracic Surgeons, Florida-January 25-27, 2010***

For more information on this meeting, contact The Society of Thoracic Surgeons, 633 N Saint Clair St, Suite 2320, Chicago, IL 60611-3658; telephone: (312) 202-5800; fax: (312) 2025801; e-mail: sts@sts.org; website: www.sts.org.

■ **European Society of Thoracic Surgeons School of Thoracic Surgery- France—February 15-16, 2010**

For more information on this meeting, contact the European Society of Thoracic Surgeons, PO Box 159, Exeter EX2 5SH, United Kingdom; telephone: +44 13 9243-0671; fax: +44 13 9243-0671; e-mail: sue@ests.org.uk; website: www.estsschool.org.

■ **30th Annual Cardiothoracic Surgery Symposium, California—March 4-7, 2010**

For more information on this meeting contact CREF 2010, 475 W Stetson, Ste. T, #388, Hemet, CA 92543; telephone: (951) 765-2573; fax: (951) 765-2576; e-mail: susan@amainc.com; website: www.amainc.com.

■ **Society for Cardiothoracic Surgery in Great Britain and Ireland 2010 , March 7-9, 2010**

Annual Meeting and Cardiothoracic Forum-Liverpool, United Kingdom—
For more information on this meeting, contact Isabelle Ferner, The Society for Cardiothoracic Surgery in Great Britain and Ireland, 35-43 Lincoln's Inn Fields, London WC2A 3PE, United Kingdom; telephone: +44 020 7869-6893; fax: +44 020 78696890; e-mail: sctsadmin@scts.org; website: www.scts.org.

■ **Interventional Cardiology 2010: 25th Annual International Symposium-Snowmass Village, Colorado—March 7-12, 2010**

For more information on this meeting, contact Promedica International CME, 2333 State St, Suite 203, Carlsbad, CA 92008; telephone: (760) 7202263; fax: (760) 720-6263; e-mail: ic2010@promedicacme.com; web-site: www.promedicacme.com.

■ **European Society ofThoracic Surgeons School of Thoracic Surgery-Antalya, Turkey—March 17-21, 2010**

For more information on this meeting, contact the European Society of Thoracic Surgeons, PO Box 159, Exeter EX2 5SH, United Kingdom; telephone: +44 13 9243-0671; fax: +44 13 9243-0671; e-mail: sue@ests. org.uk; website: www.estsschool.org.

■ **The Houston Aortic Symposium: Frontiers in Cardiovascular Diseases, The Third in the Series-Houston, Texas—April 8-10, 2010**

For more information on this meeting, contact Promedica International CME, 2333 State St, Suite 203, Carlsbad, CA 92008; telephone: (760) 7202263; fax: (760) 720-6263; e-mail: has2010@promedicacme.com

■ **32nd Charing Cross International Symposium-London, United Kingdom—April 10-13, 2010**

For more information on this meeting, contact Biba Conferences, 44 Burlington Rd, Fulham, London SW6 4NX, United Kingdom; telephone: +44 (0) 20-7736-8788; fax: +44 (0) 7736-8283; e-mail: info@ cxsymposium. com; website: www. cxsymposium.com.

■ **International Society for Heart and Lung Transplantation 30th Anniversary Meeting and Scientific Sessions-Chicago, Illinois—April 21-24, 2010**

For more information on this meeting, contact the International Society for Heart and Lung Transplantation, 14673 Midway Rd, Suite 200, Addison, TX 75001; telephone: (972) 490-9495; fax: (972) 490-9499; e-mail: ishlt@ishlt. org; website: www.ishlt.org.

■ **90th Annual Meeting of the American Association for Thoracic Surgery-Toronto**

Ontario, Canada, 2010 For more information on this meeting, contact the American Association for Thoracic Surgen-, 900 Cummings Center, Suite 221-U, Beverly, MA 01915; telephone: (978) 927-8330; fax: (978) 524-8890; e-mail: aats@prri.com; website: www.aats. org.

■ **18th European Conference on General Thoracic Surgery Valladolid, Spain—May 30-June 2, 2010**

For more information on this meeting, contact the European Society of Thoracic Surgeons, PO Box 159, Exeter EX2 5SH, United Kingdom; telephone: +44 13 9243-0671; fax: +44 13 9243-0671; e-mail: sue@ests. org.uk; website: www.estsmeetings.org.

■ **5th International Meeting of the Onassis Cardiac Surgery Center: Current Trends in Cardiac Surgery and Cardiology-Athens, Greece—September 16-18, 2010**

For more information on this meeting, contact Liana Eliopoulou, Triaena Tours & Congress, 206 Sygrou Ave, 176 72 Athens (Kallithea), Greece; telephone: +30 210 749-9353; fax: +30 210 770-5752; website: www. ocsc2009. com.

■ **European Society of Thoracic Surgeons School of Thoracic Surgery-Elancourt, France—September 27-28, 2010**

For more information on this meeting, contact the European Society of Thoracic Surgeons, PO Box 159, Exeter EX2 5SH, United Kingdom; telephone: +44 13 9243-0671; fax: +44 13 9243-0671; e-mail: sue@ests. org.uk; website: www.estsschool.org.

Editorial

Electronic Editing and Plagiarism

Guttenberg with his unparalleled achievement marking the end of middle ages, has disappeared with his outstanding invention to be replaced by the electronic technology , this is the fate of everyone and every thing witth the presnt volume we succeded to preach by the end .

Since the late nineties most of the journals editing ; all over the world and may be lately in Egypt became electronic, i.e. via internet according to Tim Berners-Lee who invented the term in 1990.

Although the articles beforehand were submitted on paper and circulating for reviewing via ordinary postage mail which could have taken ages during that time; The whole process was manual whether typing , marking or correcting etc……and there was no electronic version.

In that volume of our journal we succeeded to get the permission from Blackwell to publish part from Doctor's George Hall book : "How to Write a Paper" which is considered the bible guide for scientific researchers teaching them how to build and write their studies. There is also a part on how to choose the statistical method to be implemented in the research. We have added as well to the editorial section the guidelines for Electronic Publication which is a necessity today for any author, reviewer or journal editing.

We added a paragraph on Plagiarism to inform our Authors and Reviewers; that now any article can be checked for any dishonesty by copying from other articles or international literature . There are now computer programs on the internet for Plagiarism checking, through a very single procedure, so we implore our authors and researchers to take care from this point which is now easily detected and can have a very bad impact on that person career and future as it is considered now in many countries a criminal activity.

Finally we came to know that now most journals will be valued by what is known now as the "Impact Factor" which is a numeric factor translating the number of times our journal is cited in other publications so we urge our readers to cite our journal in their different studies and publications to increase its scientific impact bearing in mind that the real waluation of our Journal infront of the distinguis hed Comniheas will depend on such factors .

Lastly after too many years as Co Editor and Editor to this Journal it is time to jump from the boat …. Oh; I am just joking but as new musicians , actors, and also editors.

Thanks to the issuing date gap Almighty God and to you Readers, Authors, Reviewers ,Editors and Colleagues for your support with special tribute to Dr Magdy Mostafa and Dr. Ezz El Din Mostafa to whom both I owe a lot , wishing for our journal all the prosperity and advancement .

Yours Sincerely

Yasser Hegazy MD , FRCS
Editor - In - Chief
Journal Of The E S C T S

How To Write A Paper

Structure of a Scientific Paper

George M. Hall

The research you have conducted is obviously of vital importance and must be read by the widest possible audience. It probably is safer to insult a colleague's¬ spouse, family, and driving than the quality of his or her research. Fortunately, so many medical journals now exist that your chances of not having the work published somewhere are small. Nevertheless, the paper must be constructed in the approved manner and presented to the highest possible standards. Editors and assessors without doubt will look adversely on scruffy manuscripts – regardless of the quality of the science .All manuscripts are constructed in a similar manner, although some notable exceptions exist, like the format used by Nature. Such exceptions are unlikely to trouble you in the early ' stages of your research career.

The object of publishing a scientific paper is to provide a document that contains sufficient information to enable readers to:

assess the observations you made;

repeat the experiment if they wish;

determine whether the conclusions drawn are justified by the data.

The basic structure of a paper is summarised by the acronym imrad, which stands for:

Introduction **(What question was asked?)**

Methods **(How was it studied?)**

Results **(What was found? and**

Discussion **(What do the findings mean?)**

The introduction should be brief and must state clearly the question that you tried to answer in the study. To lead the reader to this point, it is necessary to review the relevant literature briefly.

Many junior authors find it difficult to write the introduction . The most common problem is the inability to state clearly what question was asked. This should not be a problem if the study was planned correctly – it is too late to rectify basic errors when attempting to write the paper. Nevertheless, ;some studies seem to develop a life of their own, and the original objectives can easily be forgotten. I find it useful to ask collaborators from time to time what question we hope to answer. If I do not receive a short clear sentence as an answer, then alarm bells ring.

BMJ. Books
4th edition-Black well
Dr.George M. Hall
St George' Hospital
University of london
London, Uk

The introduction must not include a review of the literature. Only cite those references that are essential to justify your proposedd study. Three citations from different groups usually are enough to convince most assessors that some fact is `well known' or 'well recognised, particularly if the studies are from different countries. Many research groups write the introduction to a paper before the work is started, but you must never ignore pertinent literature published while the study is in progress.

example introduction:

It is well known that middle-aged male runners have diffuse brain damage,` but whether this is present before they begin running or arises as a result of repeated cerebral contusions during exercise has not been established. In the present study, we examined cerebral function in a group of sedentary middle-aged men before and after a six month exercise programme.

Methods:

This important part of the manuscript increasingly is neglected, and yet the methods section is the most common cause of absolute rejection of a paper. If the methods used to try to answer the question were inappropriate or flawed, then there is no salvation for the work. Next part contains useful advice about the design of the study and precision of measurement that should be considered when the work is planned – not after the work has been completed.

The main purposes of the methods section are to describe, and sometimes defend, the experimental design and to provide enough detail that a competent worker could repeat the study. The latter is particularly important when you are deciding how much to include in the text. If standard methods of measurement are used, appropriate references are alll that is required. In many instances. `modifications' of published methods are used, and it is these that cause difficulties for other workers. To ensure reproducible data, authors should:

- give complete details of any new methods used;
- give the precision of the measurements undertaken;
- sensibly use statistical analysis.

Input from a statistician should be sought at the planning stage of any study. Statisticians invariably are helpful, and they have contributed greatly to improving both the design and analysis of clinical investigations. They cannot be expected, however, to resurrect a badly designed study.

Results :

The results section of a paper has two key features: there should be an overall description of the major findings of the study; and the data should be presented clearly and concisely.

You do not need to present every scrap of data that you have collected. A great temptation is to give all the results, particularly if they were difficult to obtain, but this section should contain only relevant, representative data. The statistical analysis of the results must be appropriate. The easy availability of statistical software packages has not encouraged young research workers to understand the principles involved. An assessor is only able to estimate the validity of the statistical tests used, so if your analysis is complicated or unusual, expect your paper to undergo appraisal by a statistician.

You must strive for clarity in the results section by avoiding unnecessary repetition of data in the text, figures, and tables. It is worthwhile stating briefly what you did not find, as this may stop other workers in the area undertaking unnecessary studies.

Discussion :

The initial draft of the discussion is almost invariably too long. It is difficult not to write a long and detailed analysis of the literature that you know so well. A rough guide to the length of this section, however, is that it should not be more than one-third of the total length of the manuscript (Introduction + Methods + Results + Discussion). Ample scope often remains for further pruning.

Many beginners find this section of the paper difficult. It is possible to compose an adequate discussion around the points given in Box 1.1.

1.1 Writing the discussion

- **Summarise the major findings**
- **Discuss possible problems with the methods used**
- **Compare your results with previous work**
- **Discuss the clinical and scientific (if any) implications of your findings**
- **Suggest further work**
- **Produce a succinct conclusion**

Common errors include repetition or data already given in the results section, a belief that the methods were beyond criticism, and preferential citing of previous work to suit the conclusions. Good assessors will seize upon such mistakes, so do not even contemplate trying to deceive them.

Although IMRAD describes the basic structure of a paper, other parts of a manuscript are important .The title summary(or abstract),and list of Authors are described in chapter 6. It is salutary to remember that many people will read the title of the paper and some will read the summary, but very few will read the complete text. The title and summary of the paper are of great importance for indexing and abstracting purposes, as well as enticing readers to peruse the complete text. The use of appropriate references for a paper ; this section often is full of mistakes. A golden rule is to list only relevant, published references and to present them in a manner that is appropriate for the particular journal to which the article is being submitted. The citation of large numbers of references is an indicator of insecurity — not of scholarship. An authoritative author knows the important references that are appropriate to the study.

Before you start the first draft of the manuscript, carefully read the `Instructions to authors' that every journal publishes, and prepare your paper accordingly. Some journals give detailed instructions, often annually, and these can be a valuable way of learning some of the basic rules. A grave mistake is to submit a paper to one journal in the style of another; this suggests that it has recently been rejected. At all stages of preparation of the paper, go back and check with the instructions to authors to make sure that your manuscript conforms. It seems very obvious, but if you wish to publish in the European Annals of Andrology, do not write your paper to conform with the Swedish Journal of Androgen Research. Read and re-read the instructions to authors.

Variations on the IMRAD system are sometimes necessary in specialised circumstances, such as a letter to the editor , an abstract for presentation at a scientific meeting , or a case report , Nevertheless, a fundamental structure is the basis of all scientific papers.

Introductions:

Introductions should be short and arresting and tell the reader why you have undertaken the study. This first sentence tells you almost everything I have to say and you could stop here. If you were reading a newspaper, you probably would — and that is why journalists writing a news story will try to give the essence of their story in the first line. An alternative technique used by journalists and authors is to begin with a sentence so arresting that the reader will be hooked and likely to stay for the whole piece.

I may mislead by beginning with these journalistic devices, but I want to return to them: scientific writing can usefully borrow from journalism. But let me begin with writing introductions for scientific papers.

Before beginning, answer the basic questions

Before sitting down to write an introduction you must have answered the basic questions that apply to any piece of writing:

- ***What do I have to say?***
- ***Is it worth saying?***
- ***What is the right format for the message?***
- ***What is the audience for the message?***
- ***What is the right journal for the message?***

If you are unclear about the answers to these questions then your piece of writing — no matter whether it's a news story, a poem, or a scientific paper — is unlikely to succeed. As editor of the British Medical Journal, every day I saw papers where the authors had not answered these questions. Authors are often not clear about what they want to say. They start with some sort of idea and hope that the reader will have the wit to sort out what is important. The reader will not bother. Authors also regularly choose the wrong format — a scientific paper rather than a descriptive essay or a long paper rather than a short one Not being clear about the audience is probably the commonest error and specialists regularly write for generalists in a way that is entirely inaccessible.

Another basic rule is to read the instructions to authors (or advice to contributors, as politically correct journals like the BMJ now call them) of the journal you are writing for. Too few authors do this, but there is little point in writing a 400 words introduction when the journal has a limit for the whole article of 600 words.

Tell readers why you have undertaken the study

The main job of the introduction is to tell readers why you have undertaken the study. If you set out to answer a question that really interested you, then you will have little difficulty. But if your main reason for

undertaking the study was to have something to add to your curriculum vitae, it will show The best questions may arise directly from clinical practice and, if that is the case, the introduction should say so:

Examble :

A patient was anaesthetised for an operation to repair his hernia and asked whether the fact that he used Ecstasy four nights a week would create difficulties. We were unable to find an answer in published medical reports and so designed a study to answer the question.

or

Because of pressure to reduce night work for junior doctors we wondered if it would be safe to delay operating on patients with appendicitis until the morning after they were admitted.

If your audience is interested in the answer to these questions then they may well be tempted to read the paper and, if you have defined your audience and selected the right journal, they should be interested.

More commonly, you will be building on scientific work already published. It then becomes essential to make clear how your work adds importantly to what has gone before.

Clarify what your work adds

Editors will not want to publish – and readers will not want to read – studies that simply repeat what has been done several times before. Indeed, you should not be undertaking a study or writing a paper unless you are confident that it adds importantly to what has gone before. The introduction should not read:

Several studies have shown that regular Ecstasy use creates anaesthetic difficulties,' and several others have shown that it does not. We report two further patients, one of whom experienced problems and one of whom did not ,and review the literature.

Rather it should read something like:

'Two previous studies have reported that regular Ecstasy use may give rise to respiratory problems during anaesthesia. These studies were small and uncontrolled, used only crude measurements of respiratory function, and did not follow up the patients. We report a larger, controlled study, with detailed measurements of respiratory function and two year follow up.

Usually, it is not so easy to make clear how your study is better than previous ones and this is where the temptation arises to give a detailed critique of everything that has ever gone before. You will be particularly tempted to do this because, if you are serious about your study, you will have spent hours in the library detecting and reading all the relevant literature. The very best introductions will include a systematic review of all the work that has gone before and a demonstration that new work is needed.

The move towards systematic reviews is one of the most important developments in science and scientific writing in the past 20 years [1]. We now understand that most reviews are highly selective in the evidence they adduce and often wrong in the conclusions they reach [2]. When undertaking a systematic review an author poses a clear question, gathers all relevant information (published in whatever language or unpublished), discards the scientifically weak material, synthesis the remaining information, and then draws a conclusion.

To undertake such a review is clearly a major task, but this ideally is what you should do before you begin a new study. You should then undertake the study only if the question cannot be answered and if your study will contribute importantly to producing an answer. You should include a brief account of the review in the introduction. Readers will then fully understand how your study fits with what has gone before and why it is important.

`In 2007 you should not worry that you cannot reach this high standard because the number of medical papers that have ever done so could probably be numbered on the fingers of one hand' I wrote the same sentence in the first edition of this book only with the year as1994. I then wrote in the first edition:' But by the end of the millennium brief accounts of such reviews will, I hope, be routine in introductions' .I was – as always – wildly overoptimistic. Summaries of systematic reviews are still far from routine in introductions in scientific papers. Indeed, a paper presented at the Third International Congress m Peer Review in September 1997 showed that many randomised controlled trials published in the world's five major general medical journals failed to mention trials that had been done before on the same subject.

This means that authors are routinely flouting the Helsinki Declaration on research involving human subjects. The declaration states that such research should

be based on a thorough knowledge of the scientific literature [3]. Repeating research that has already been satisfactorily done is poor practice. As the CONSORT statement on good practice in reporting clinical trials says: 'Some clinical trials have been shown to have been unnecessary because the question they addressed had been or could have been answered by a systematic review of the existing literature' [4,5].

In 2007 my advice on systematically reviewing previous reports remains a counsel of perfection, but it's still good advice. Perhaps you can be somebody who moves the scientific paper forward rather than somebody who just reaches the minimum standard for publication.

Another important and relevant advance since the first edition is that scientific journals almost all now have websites and publish synergistically on paper and on the Web [6,7]. This at last opens up the possibility of simultaneously being able to satisfy the needs of the reader—researcher, who wants lots of detail and data, and the needs of the reader—practitioner, who wants a straightforward message. The BMJ, for example, introduced a system it calls ELPS (electronic long, paper short) [8]. In this case, it is the editors who produce the shorter paper, although you will have to approve it before publication. In the context of introductions, this synergistic publishing might mean that a proper systematic review might be published on the Web while the paper version might include a short and simple summary. Usually, however, a full systematic review is probably best dealt with as a separate paper.

One interesting feature of revising a chapter 13 years after you wrote the first version is to reflect on how much scientific papers have changed. We might have expected that the appearance of the World Wide Web in the early 1990s would have changed everything. Space is no longer a problem. Video and sound can be added. Hyperlinks are easy. Full data — and the software used to manipulate them — could be included. But the overwhelming impression so far is that very little has changed [9]. In 2004 the BMJ published the 50-year results of the British doctors study [10], providing an opportunity to compare the paper with that giving the first set of results half a century ago [11]. Making the comparison I wrote: 'In the 50 years during which men have landed on the moon, computers and the Internet have appeared, television and cars have been transformed, the scientific article has changed hardly at all. Does this reflect the robustness of the form or a failure of imagination? I suspect the latter' [9].

My suspicion is that new technology will eventually lead to dramatic changes and that if I live to write this chapter again I may have to start completely afresh.

Following the best advice

An important development in medical writing in recent years has been the appearance of suggested structures for certain kinds of studies. These have appeared because of considerable evidence that many scientific reports do not include important information. There are guidelines for randomised controlled trials [4], systematic reviews [12], economic evaluations [13], and studies reporting tests of diagnostic methods [14]. More guidelines will follow and many journals, including the BMJ, require authors to conform to these standards. They will send back reports that do not conform. So authors need to be aware of these guidelines. The requirements for introductions are usually straightforward and not very different from the advice given in this chapter.

Keep it short

You must resist the temptation to impress readers by summarising everything that has gone before. They will be bored, not impressed, and will probably never make it through your study. Your introduction should not read:

Archaeologists have hypothesised that a primitive version of Ecstasy may have been widely used in ancient Egypt. Canisters found in tombs of the pharaohs ... Sociological evidence shows that Ecstasy is most commonly used by males aged 15 to 25 at parties held in aircraft hangars? The respiratory problems associated with Ecstasy may arise at the alveolar-capillary interface. Aardvark hypothesised in 1926 that problems might arise at this interface because of?

Nor should you write:

Many studies have addressed the problem of Ecstasy and anaesthesia.1-9

With such a sentence you say almost nothing useful and you've promptly filled a whole page with references. You should choose references that are apposite, not simply to demonstrate that you've done a lot of reading.

It may often be difficult to make clear in a few words why your study is superior to previous ones, but you must convince editors and readers that it is better. Your introduction might read something like:

Anaesthetists cannot be sure whether important complications may arise in patients who regularly use Ecstasy. Several case studies have described such problems." Three cohort studies have been published, two of which found a high incidence of respiratory problem in regular ecstasy users. One or these studies was uncontrolled and in the other the patients were poorly matched for age and smoking.' The study that did not find any problems included only six regular Ecstasy users and the chance of an important effect being missed (a type II error) was high.' We have undertaken a study of 50 regular Ecstasy users with controls matched for age, smoking status, and alcohol consumption.

A more detailed critique of the other studies can be left for the discussion . Even then, you should not give an exhaustive account of what has gone before but should concentrate on the best studies that are closest to yours. You will also then be able to compare the strengths and weaknesses of your study with the other studies, something that would be wholly out of place in the introduction.

Make sure that you are aware of earlier studies

I've already emphasised the importance of locating earlier studies. Before beginning a study, authors should seek the help of librarians in finding any earlier studies. Authors should also make personal contact with people who are experts in the subject and who may know of published studies that library searches do not find, unpublished studies, or studies currently under way. It's also a good idea to find the latest possible review on the subject an(search the references and to look at the abstracts of meetings on the subject . We know that library searches often do not find relevant papers that have already been published, that many good studies remain unpublished (perhaps because they reach negative conclusions), and that studies take years to conduct and sometimes years to get into published reports.

Editors increasingly want to see evidence that authors have worked hard to make sure that they know of studies directly related to theirs. This is particularly important when editors' first reaction to a paper is 'Surely we know this already'. We regularly had this experience at the BMJ and we then .looked especially hard to make sure that authors had put effort into finding what had gone before.

In a systematic review the search strategy clearly belongs in the methods section, but in an ordinary paper it belongs in the introduction, in as short form as possible. Thus it might read:

A Medline search using 15 different key phrases, personal contact with five experts in the subject, and a personal search of five recent conferences on closely related subjects produced no previous studies

of whether Grandmothers suck eggs.

Be sure your readers are convinced of the importance or your question, but don't overdo it

If you have selected the right audience and a good study then you should not have to work hard to convince your readers of the importance of the question you are answering. One common mistake is to start repeating material that is in all the textbooks and that your readers will know. Thus in a paper on whether vitamin D will prevent osteoporosis you do not need to explain osteoporosis and vitamin D to your readers. You might, however want to give them a sense of the scale of the problem by giving prevalence figures for osteoporosis, data on hospital admissions related to osteoporosis and figures on the cost to the nation of the problem.

Don't baffle your readers

Although you don't want to patronise and bore your readers by telling them things that they already know, you certainly don't want to baffle them by introducing, without explanation, material that is wholly unfamiliar. Nothing turns readers off faster than abbreviations that mean nothing or references to diseases, drugs, reports, places, or whatever that they do not know. This point simply emphasises the importance of knowing your audience.

Give the study's design but not the conclusion

This is a matter of choice, but I asked authors to give a one sentence description of their study at the end of the introduction. The last line might read:

We therefore conducted a double blind randomised study with 10-year follow up to determine whether teetotallers drinking three glasses of whisky a week can reduce their chances of dying of coronary artery disease.

I don't like it, however, when the introduction also gives the final conclusion:

Drinking three glasses of whisky a week does not reduce teetotallers' chances of dying of coronary artery disease.

Other editors may think differently.

Think about using journalistic tricks sparingly

The difficult part of writing is to get the structure right. Spinning sentences

is much easier than finding the right structure, and editors can much more

easily change sentences than structure. Most pieces of writing that fail do so because the structure is poor and that is why writing scientific articles is comparatively easy – the structure is given to you.

I have assumed in this chapter that you are writing a scientific paper. If you are writing something else you will have to think much harder about the introduction and about the structure of the whole piece. But even if you are writing a scientific paper you might make use of the devices that journalists use to hook their readers.

Tim Albert, a medical journalist, gives five possible openings in his excellent book on medical journalism [15]: telling an arresting story, describing a scene vividly, using a strong quotation, giving some intriguing facts, or making an opinionated and controversial pronouncement. He gives two examples from the health page of The Independent. Mike Hanscomb wrote:

In many respects it is easier and less uncomfortable to have leukaemia than eczema?

This is an intriguing statement and readers will be interested to read on to see if the author can convince them that his statement contains some truth. Jeremy Laurance began a piece:

This is a story of sex, fear, and money. It is about a new treatment for an embarrassing problem which could prove a money spinner in the new commercial National Health Service?

Sex, fear, and money are emotive to all of us and we may well want to know how a new treatment could make money for the health service rather than costing it money. My favourite beginning occurs in Anthony Burgess's novel Earthly Powers. The first sentence reads:

It was the afternoon of my eighty-first birthday, and I was in bed with my catamite when Ali announced that the archbishop had come to see me.

This starts the book so powerfully that it might well carry us right through the next 400 or so pages. (I had to look up `catamite' too. It means 'boy kept for homosexual purposes)

To begin a paper in the British Journal of Anaesthesia with such a sentence would be to court rejection, ridicule, and disaster, but some of the techniques advocated by Tim Albert could be used. I suggest, however, staying away from opinionated statements and quotations in scientific papers, particularly if they come from Shakespeare, the Bible, or Alice in Wonderland.

Conclusion :

To write an effective introduction you must know your audience, keep it short, tell readers why you have done the study and explain why it's important, convince them that it is better than what has gone before, and try as hard as you can to hook them in the first line.

Method :

You should describe, in logical sequence, how your study was designed and carried out, and how you analysed your data. If the study is already finished, this should be a simple task. However, do not leave writing the methods until this stage! The sooner you write down the methods, the sooner you can detect and deal with flaws in the design. Write it down, in full detail, before you start the study and ask an experienced colleague to look it over. The challenge of setting down what you intend to do is also a very useful exercise – far better than discovering predictable flaws after months of hard work. In fact, if you are conducting a therapeutic trial, you will have to register the study, and its methods, before you start. If you don't, many journals will not publish it.

What to include in the methods section
How the study was designed

- Keep the description brief
- Say how randomisation was done
- Use names to identify parts of a study sequence

How the study was carried out

- Describe how the participants were recruited and chosen
- Give reasons for excluding participants
- Consider mentioning ethical features
- Give accurate details of materials used
- Give exact drug dosages
- Give the exact form of treatment and accessible details of unusual apparatus

How the data were analysed

- Use a p-value to disprove the null hypothesis
- Give an estimate of the power of the study (the likelihood of a false negative – the B error)
- Give the exact tests used for statistical analysis (chosen a priori)

Testing hypotheses

When readers turn to the methods section, they look for more than details of the apparatus or assay that you used. The methods section should answer the questions 'Who, what, why, when, and where?' Even more important, it should state the hypothesis that was tested – for example, that a treatment has a particular effect, such as increased survival or improved outcome. This is formally tested by assuming that the null hypothesis is true. The observed results indicate how tenable this hypothesis can be – that is, the possibility that the intervention was without effect. Naturally, we would hope that this possibility would be small (much less than 1, which is complete certainty). We state how small this possibility (p-value) has to be to disprove the null hypothesis as the 'mission statement' of the study. A study of two antibiotics might :compare cure rates. The null hypothesis is that there is no difference between these rates. The statistical tests used will estimate the likelihood that the rates are statistically indistinguishable (the rates one might expect if the drugs had been the same, so that both samples were treated equally). A p-value of less than 0.05 (out of a total probability of 1) shows that the possibility that the

rates were statistically similar would have been found in less than I out of 20 experiments. Many papers merely say, adequately, 'p<0.05 was considered significant' '

The other side of the coin of probability, often neglected, is the power of the study. If the null hypothesis survives attempts to destroy its credibility, you cannot conclude immediately that there is no difference between the groups. You have only concluded that the rates are statistically indistinguishable. Are your methods sufficiently exacting to test the null hypothesis properly? A true difference might indeed be present, but it could be small. Another possibility is that a difference may exist, but because the measurements vary, the variations swamp the effect you seek. In both cases, a small ,signal-to-noise' ratio is present. You must therefore also estimate the power of the study to detect what you are looking for, to indicate possibility of a false negative result. This is the 0 error. The value you choose depends on factors such as the precision of the answer needed and may also take into account the practical consequences of an incorrect conclusion. A B-value is often taken as 0.2, which implies a power of 0.8 to avoid a false negative result. In practice, the power of a study depends on the size of the effect, the variability of the data, and the number of observations. A power of 0.8 is often taken as adequate, but this may not always be sensible: take advice if you are unhappy with a false negative result.

Always state clearly the a priori hypotheses – if only to be sure that you Collect appropriate and relevant data and do the correct statistical tests..

Statistics

State the exact tests used to analyse the data, and include an appropriate reference if the test is not well known. State the software, and the version, that you used. The statistical test you should use depends on the type of data. Sometimes the distribution of the data may not be clear before the study is over, so the a priori tests should be chosen conservatively and be non-parametric.

The statistics

Statistics must accompany data. Many papers suffer because the statistics are badly presented. Obviously, many statistical tests exist – conventional as well as esoteric. Choose the test most appropriate for your data analysis. Decide on which statistical test to use when planning your study. Do not take the data of your finished study to your local statistician to see what can be made of them – that is a waste of everyone's time.

Follow some general rules. As data are mostly restricted to tables and figures, that is where you should include most statistical data. Specify the type of statistic, the sample size (n), and the probability value for a test of significance (p-value). When normally distributed data have been analysed statistically, report the mean and a statistic that indicates the variation from the mean (e.g. the standard deviation or the range). When non-normally distributed data have been analysed statistically, report the median and the interquartile range (the range between the 25th and the 75th percentiles).

When you list statistical details in the text, follow some conventional rules. Mean and standard deviation are usually written as 11.4 ± 0.8 (SD) kg' The conventional way to write data that are being compared statistically is: `Body weight increased more in group A than in group B (13.2 ± 1.9 (SD) versus 9.4 ± 0.9 kg in eight patients, P < 0.02)' *This statement contains five types of statistical information ;the mean (13.2 and 9.4kg'), the standard deviation (1.9 and 0.9'), specification of the statistic

used to describe the variation from the mean ('SD'), the sample size (n) (`8 patients'), and the probability value of significance (p <0.02'). Usually, you should provide all five types of statistical information; however, if any of these statistical parameters apply to all data (e.g. SD and sample size), you only need to describe the complete statistical details when You list the data the first time and can omit thereafter those that apply to all data. If you decide to report the confidence interval, the statement can be rewritten as follows: 'Body weight increased more in group A than in group B (13.2 +/- 1.9 (SD) versus 9.4 ± 0.9kg in eight patients; 95% confidence interval for the difference = 1.8 – 5.2 kg, p < 0.02)

When you provide p-values, you should list the actual p-values not only for those differences considered statistically significant (e.g. p < 0.02), but also for differences not considered significant (e.g. p > 0.6 or p = 0.55). By restricting the information to statements like `p > 0.05' or `p = NS, you restrict the reader's ability to interpret the data accurately: a p-value of 0.06 does not exclude the possibility of a statistically significant difference as strongly as a p-value of >0.9.

Do not list data to a greater degree of accuracy than that of the measurement . For example, if you can measure cardiac output with an accuracy of only ±10%, do not quote values for individual results to three decimal places. Make sure that any change described as statistically significant is greater than the error of your measurement. Be particularly careful with calculated values: the errors of the original measurements add up alarmingly.

Take care when you look at associations between variables. Statistical significance needs to indicate how much of an association can be attributed to the dependency of one variable on another and how much is due to chance. Be careful with extrapolation, and do not confuse association with causation.

Statistical presentation is always a problem – too much information, too little space. Present enough information for the intelligent reader to believe what you are saying. Remember: usually, neither your readers, nor your assessors, are expert statisticians. If your statistical tests are too esoteric, be prepared for a lengthy discussion before publication.

References:

I Chalmers I. Improving the quality and dissemination of reviews of clinical research. In: Lock S, ed., The future of medical journals. London: BMJ Books, 1991, pp. 127-48.

2 Mulrow CD. The medical review article: state of the science. Ann Intern Med 1987; 104:485-8.

3 World Medical Association. Declaration of Helsinki. Recommendations guiding physicians in biomedical research involving human subjects. JAMA 1997; 277:925-6.

4 Moher D, Schulz KF, Altman DG. The CONSORT statement: revised recommendations for improving the quality of reports of parallel-group randomised trials. Lancet 2001;357:1191-4.

5 Lau J, Antman EM, Jimenez-Silva J, Kupelnick B, Mosteller F, Chalmers TC. Cumulative meta-analysis of therapeutic trials for myocardial infarction. New Engl J Med 1992;327:248-54.

6 Bero L, Delamothe T, Dixon A, et al. The electronic future: what might an online scientific paper look like in five years' time? BMJ 1997;315:1692-6.

7 Delamothe T. Is that it? How online articles have changed over the past five years. BMJ 2002;325:1475-8.

8 Milliner M, Groves T. Making research papers in the BMJ more accessible. BMJ 2002;325:456.

9 Smith R. Scientific articles have hardly changed in 50 years. BMJ 2004;328:1533.

10 Doll R, Peto R, Boreham J, Sutherland I. Mortality in relation to smoking: 50 years' observations on male British doctors. BMJ 2004;328:1519-33.

11 Doll R, Hill AB. The mortality of doctors in relation to their smoking habits. A preliminary report. BMJ 1954;228(i):1451-5.

12 Moher D, Cook DJ, Eastwood S, Olkin 1, Rennie D, Stroup DE Improving the quality of reports of meta-analyses of randomised controlled trials: the QUOROM statement. Quality of reporting of meta-analyses. Lancet 1999;354:1896-900.

13 Drummond MF, Jefferson TO. Guidelines for authors and peer reviewers of economic submissions to the BMJ. The BMJ economic evaluation working party. BMJ 1996;313:275-83.

14 Bossuyt PM, Reitsma B, Brns DE, et al. Towards complete and accurate reporting of studies of diagnostic accuracy: the STARD initiative. BMJ 2003;326:41-4.

15 Albert T. Medical journalism: the writer's guide. Oxford: Radcliffe, 1992.

Statistics

Clinically useful measures of trial outcome (part three): Absolute risk reduction (ARR) and number needed to treat (NNT).

Ahmed Hassouna, MD.

Besides the previously outlined relative measures such as the relative risk, relative risk reduction, hazard risk and Odds ratio; it is evident that there is a need for also comparative indices which takes into account the changes on the absolute scale. Table 1 shows the risk of sudden death -calculated over 1 year- in a group of 800 patients with either moderate or severe aortic valve stenosis (AS) and who benefited from either medical or surgical treatment. As shown, the risk of sudden death in patients with moderate AS is decreased from 0.01 (1%) in the medical (control) group to only 0.005 (0.5%) in the surgery group. On the other hand, patients with severe AS appear to get more benefit from surgery for the risk of sudden death being decreased from as much as 0.1 (10%) to only 0.01 (1%).

The absolute risk reduction (ARR) that is achieved by surgery is simply calculated by subtracting the risk of death associated in the surgery group from that associated with the medically treated patients (control group). In patients with moderate AS, surgery can reduce the risk of sudden from 1% to only 0.5%; i.e. with sby the same surgery for patient with severe AS is 18 folds that offered to those with moderate AS; ARR= 10%-1% = 9%. As evident, the ARR says a great deal about whether an effect is likely to be clinically meaningful [1]. In other words, the same surgery has a much higher clinical impact on patients with severe AS, compared to those with moderate AS and hence, the treating physician can highly recommend surgery to a patient with severe AS and to a less extent or with some reservations to the patient with a moderate AS.

The ARR can also be expressed in terms of number of events prevented per 100 people treated. In our example, operating upon 200 patients with severe AS reduced the risk of sudden death from as much as 20 patients in the control group to only 2 patients in the surgery group; i.e. 18 more patients were saved by surgery in this study. In other words, surgery can save 18/2 = 9 people from sudden death per each hundred patients with severe AS who are operated upon. How many patients should we operate upon to save one life from sudden death = 100/9 = 11.1 patients; which is known as the number needed to treat or NNT. Hence, the NNT is the inverse of ARR [1/ARR in decimals or 100/ARR in %] or NNT is the number of patients which a clinician needs to treat with a particular therapy to expect to prevent one adverse event [1], i.e. in prophylaxis of a bad outcome. On the other hand, if the event is a good outcome, NNT becomes the number of patients which a clinician needs to treat with a particular therapy to expect to achieve the good event (i.e. in treatment) [2].

*Department of Cardiothoracic Surgery, Ain Shams University Hospitals, Cairo.
E-mail: ahmedhasona@hotmail.com
Codex: 04/07/0911

Aortic valve stenosis (AS) (800 patients)	Event (n, %) Medical group	Surgery group	ARR	NNT
a) Moderate AS (400 patients)	2/200 (0.01)	1/200 (0.005)	0.005	200
b) Severe AS (400 patients)	20/200 (0.10)	2/200 (0.01)	0.09	11.1

Table 1: Risk of sudden death calculated over 1 year period for 800 patients with either moderate or severe aortic stenosis, who were either medically or surgically treated.
Values are presented as numbers (%), AS = aortic valve stenosis, ARR = absolute risk reduction, NNT = number needed to treat.

Though mathematically related to risk differences, the number to treat is considered as a much more meaningful way of expressing the benefit of an active treatment over a control, either in the results of a therapeutic trial or for medical decision making about an individual patient [(1)]. In our first example on patients with AS, we have to perform 200 aortic valve replacements to prevent one sudden death, among patients with moderate AS. The cost of this life saved is, of course, much more expensive than that life saved by performing only 11 aortic valve replacements for patients with severe AS.

We have to note that there are situations in which the treatment group may experience more adverse outcomes, compared to the control group. Let us take the example where the use of epidural anesthesia during the course of labor was associated with a 16% need of Cesarean section, compared to only 6% of control group not receiving epidural anesthesia (2). If we proceed as before we would report our results as follows: the ARR = 0.06-0.16 = -0.1 (or -10%) and the NNT = 100/-10 = (-10). In fact, a negative absolute risk reduction and consequently a negative number needed to treat indicate that the treatment has a harmful effect or undesirable outcome as the need of a Cesarean section in our case. Indeed, it would be more appropriate by just changing the terms to present our case as follows: The absolute risk increase (ARI) is 10% and the number needed to harm (NNTH) is 10 patients. The latter means that for every 10 women in labor who are given epidural analgesia, 1 will have a caesarean section that otherwise would not have had the operation if all 10 women did not receive epidural anesthesia. A negative number needed to treat has been called the number needed to harm: NNTH or NNT (harm); while a positive NNT has been called the number needed to benefit: NNTB or NNT (benefit)(3).

Nevertheless, the NNT is like the arithmetic mean, i.e. it can express the whole of a study in just a single number and the CI at 95% is simply obtained by taking reciprocals of the values defining the confidence intervals for the ARR (3). Applying this to patients with severe AS in Table 1, and as previously shown in this series for calculating the IC of proportions, we begin by calculating both: a common death rate for all patients with severe AS who benefited from both therapies (p) and a common survival rate (q= 1-p). As both groups have the same number of patients, p = (0.1 + 0.01) /2 = 0.055 and by deduction q= 0.945. If Ns and Nc are the respective number of patients in the 2 groups surgery and medical (control) respectively, the common variance for both groups = pq/Ns + pq/Nc = (0.055x 0.945)/200 + (0.055x 0.945)/200 = 0.00052 and the SD equals the square root of the variance = 0.023. The IC at 95% of the ARR = ARR + 1.96 x SD = 0.09 + 1.96 x 0.023 = 0.046 and 0.135 and those of the NNT are just their reciprocal values = 7.5 and 21.7 patients.

The NNT vary with the duration of follow-up and hence, in a time to event study (e.g. survival), the NNT at a specific time represents the number of patients who need to be given the treatment in question, for one additional patient to benefit from the treatment and "not to experience the event; i.e. (e.g. to survive) at that time point (4). In our example, we have calculated the NNT after 1 year of follow up. In the case where we would like to calculate the NNT for a shorter or a longer follow up period, Sackett and colleagues suggested a simple correction by multiplying the observed NNT by the ratio formed of: the average duration of follow up to the duration of interest. Taking the example illustrated in Table 1: the NNT calculated for patients with severe AS 1s 11.1 patients, for an average of 1 year follow-up.

Accordingly, we can calculate the NNT for a 2 years follow-up = 11.2x 1/2= 5.6 patients and the NNT for 6 months follow up = 11.2 x 1/0.5 = 22.4 patients. Although the equation is attractively simple, yet it necessitates 2 strong assumptions that are sometimes hard to verify: the rate by which the event in question occurs as well as the effect of the given treatment are both constant over time (5). A more realistic equation that avoids making those hard assumptions was given by Altman and Anderson. It begins by estimating survival at a certain time point for both: active treatment group (Ss) and control group (Sc) and noting the number of patients remaining alive in both groups Ns and Nc; always at the same time point. The ARR equals (Ss- Sc) and its standard error equals the square root of the quantity [Ss2(1-Ss)/Ns+Sc2(1-Sc)/Nc]. As routine, the 95% IC of ARR = ARR + 1.96 SE and that of NNT is the reciprocals of those values (4). Let us apply the equation to our example and suppose that at 6 months, 10 patients died in medical group in comparison to only 1 patient in surgery group. At 6 months, the ARR = 10/200-1/200 = 0.05-0.005 = 0.045 and, its SE = the square root of the quantity [0.052(1-0.05)/190+0.0052(1-0.005)/199] = 0.0036 and the CI at 95% = 0.045 + 1.96 x 0.0036 = 0.038 and 0.052. The NNT = 22.2 and its CI at 95% = 19.2 and 26.3 patients. Note that the NNT calculated by this equation is nearly the same as calculated by the more simplistic one given above (5). This is because the mortalities after 6 months of follow up were half those reported after double this period; after 1 one year. In other words, we have supposed that the hazard of mortality and effect treatment are constant over the period of the study; which is unfortunately not always the case.

References

1. Laupacis, A, Sackett, DL and RS., Roberts.An assessment of clinically useful measures of the consequences of treatment. N Engl J Med 1998; 318: 1728-33.
2. McQuay, RA and Moore, HJ. Using Numerical Results from Systematic Reviews in Clinical Practice. Ann Intern Med 1997; 126: 712-20.
3. Altman DG. Confidence intervals for the number needed to treat. BMJ 1998; 317: 1309-12.
4. Altman, DG and Anderson, PK. Calculating the number needed to treat for trials where the outcome is time to an event. BMJ 1999; 319: 1492-5.
5. Sackett DL, Haynes RB, Tugwell P, Guyatt GH. Clinical epidemiology: a basic science for clinical medicine, 2nd ed. Boston: Little, Brown, 1991.

Cardiovascular

Off – Pump Left Internal Mammary Artery to Left Anterior Descending Artery, In High Risk Patients

Osama A. Abbas, MD, *
Zainab Abd-el Salam, MD. **

Objectives: **To evaluate the feasibility and safety of Off-Pump left internal mammary artery (LIMA) to left anterior descending artery (LAD) in high risk patients with isolated ischemic single vessel disease, in the form of LAD lesion. This evaluation was done in terms of early survival and post-operative complications. Duration of ventilation, ICU stay, and total hospital stay were also reviewed.**

Methods: **The material of this study consisted of retrospective assessment of 30 high risk patients underwent single graft LIMA to LAD off-pump, in Ain Shams University Hospitals, in the period From March 2007 to October 2008. We defined high risk patients as those patients having one or more co-morbidity rendering them high risk for aortic cannulation and cardiopulmonary bypass. The included comorbidities were, left ventricular dysfunction and low EF (≤ 35), renal dysfunction (not on dialysis) with a serum creatinine > 1.5, sever calcification and atherosclerosis of the ascending aorta, elderly patients with age > 75 years, body mass index (BMI) > 27, history of stroke with carotid and peripheral vascular diseases (PVD), blood diseases with coagulation disorders, malignancy and chronic obstructive pulmonary disease (COPD).**

Results: Hospital mortality was 3.3% due to perioperative MI and low cardiac output. The mean ventilation time was 9.6 ± 4.86 hrs, mean inotropic usage time was 8.7 ± 14.09 hrs, and total ICU stay was 42.76 ± 20.79 hrs. Low cardiac output detected in 10%, with intraoperative insertion of IABP. There was no incidence of emergency conversion to On-Pump. Postoperative total hospital stay ranged from 5 to 15 days (mean of 7.8 ± 2.8).

Conclusion: **Off-Pump LIMA to LAD can be safely done in high risk patients who had single coronary artery disease, LAD lesion with acceptable morbidity and early mortality.**

Address reprint request to: Dr. Osama A. Abbas
Accepted for publication
Address reprint request to : Dr Osama Abbass MD
Department of Cardiothoracic surgery, Ain Shams University Hospitals , EGYPT
Email : jegyptscts@gamil.com
Codex : 04/107/0906

Treatment strategies for ischemic heart disease are complicated by the development of off-pump coronary artery bypass (OPCAB) surgery and drug-eluting stents [1]. Since percutaneous coronary intervention is indicated in a wider range of cases and the remaining sever, high risk cases are subject to surgical revascularization, the best surgical approach should be selected carefully among OPCAB, On-Pump Beating heart coronary artery bypass (CAB), and Conventional CAB [2]. Numerous investigations have shown the OPCAB procedure to hold some advantages over conventional coronary artery bypass (CCAB) using

cardiopulmonary bypass (CPB) [3,4]. OPCAB is advanced by improved surgical techniques and the development of new devices and is introduced aggressively even in high-risk patients for cardiopulmonary bypass (CBP) (e.g., patients with left ventricular dysfunction and low ejection fraction (EF), patients with sever calcification of the ascending aorta, elderly patients, and those complicated by cerebrovascular disorder, malignancy, multiple atherosclerosis, renal failure, and/or respiratory failure [5,6].

The aim of this study was to evaluate the results of OPCAB left internal mammary artery (LIMA) to left anterior descending artery (LAD) in high risk patients with isolated ischemic single vessel disease, in the form of LAD lesion, as regarding early survival and post-operative complications. Duration of ventilation, ICU stay, and total hospital stay were also reviewed.

Methods:

The material of this study consisted of retrospective assessment of 30 high risk patients had ischemic heart disease, single vessel disease, in the form of LAD lesion. All patients underwent single graft LIMA to LAD off-pump, in Ain Shams University Hospitals, in the period From March 2007 to October 2008. We defined high risk patients as those patients having one or more co-morbidity rendering them high risk for aortic cannulation and cardiopulmonary bypass. The included comorbidities were, left ventricular dysfunction and low EF (≤ 35), renal dysfunction (not on dialysis) with a serum creatinine > 1.5, sever calcification and atherosclerosis of the ascending aorta, elderly patients with age > 75 years, body mass index (BMI) > 27, history of stroke with carotid and peripheral vascular diseases(PVD), blood diseases with coagulation disorders, malignancy, chronic obstructive pulmonary disease (COPD). Pre-operative data were collected for all patients including: history taking for age, sex, history of diabetes mellitus (DM), hypertension, hyperlipidemia, previous stroke, previous myocardial infarction (MI), smoking, malignancy, PVD or carotid disease, known blood disease or coagulation disorders. Routine preoperative investigations were done including electrocardiography (ECG), complete blood picture (CBC), serum creatinine, pulmonary function test, liver function tests, and full coagulation profile. All patients underwent preoperative echocardiography for assessment of ejection fraction (EF %), left ventricular end diastolic diameter (LVEDD), and degree of mitral incompetence. Patients with EF ≤ 35 % underwent dobutamine stress echocardiography for viability assessment of anterior wall and septum. All patients with history of cerbrovascular disorders or peripheral vascular disease underwent CT brain, CT chest and heart, carotid duplex, and peripheral vascular duplex. Coronary angiography was done for all patients to asses the LAD lesion and the condition of the other vessels. Anginal symptoms were evaluated on the basis of the Canadian Cardiovascular Classification (CCS), and functional status was evaluated on the basis of New York Heart Association (NYHA) classification.

Surgical technique:

Off-pump coronary artery bypass operation was carried out through a full sternotomy incision after harvesting of pedicled LIMA for all patients in the habitual manner. The LIMA was incised at its distal bifurcation, and then fashioned for distal anastomosis after administration of heparin in the dose of 1.5 mg / kg and activated clotting time (ACT) was maintained at>250 s. The heart was raised up to the level of the sternum using warm big towel and myocardial stabilization was achieved using Octopus 3 stabilizer (Medtronic Inc., Minneapolis, Minn.). The LAD was occluded proximally using 4/0 prolene, and a blower-mister (Medtronic Inc.) was used for enhanced visualization during performing the end-to-side anastomosis using 7.0 Prolene sutures. Silastic shunts were used if there was a distal bleeding troublesome or there were ST changes in the ECG. Shunts were avoided as far as possible if the vessel was diffusely disease. No cell-savers were used. After finishing the anastomosis, the octopus was removed and LIMA pedicle was fixed on the surface of the heart, then protamine sulfate was administered at half the heparin dose. The type and dose of inotropic support was adjusted according to the haemodynamic status and visible cardiac function if needed. Haemostasis and closure in layers after insertion of two mediastinal and one pleural drain. The patient was transferred to the intensive care unit (ICU) and managed as the routine. Full monitoring in the form of invasive arterial blood pressure, centeral venous pressure, oxygen saturation, blood sugar level, arterial blood gases (ABG), urine output, mediastinal and pleural drainage hourly. Baseline ECG, chest x-ray, CBC, serum creatinine, liver function tests, total CPK, and CPK-MB was done. Also full coagulation profile in the form of PT, PTT, INR, and ACT was done.

The following definitions were used for various hemodynamic and biochemical parameters: Hypoxia:

PaO2 of <60 torr with FIO2 of 0.4 Low cardiac output: Clinical criteria of cold peripheries, mean arterial pressure of less than 60 torr, urine output of <0.5 ml/kg/hour, acidosis and a mixed venous saturation of <60%. Renal dysfunction: Serum creatinine >1.5 mg%. Inotropic usage: Dopamine >5 mcg/kg/min or adrenaline >0.05 mcg/kg/min Perioperative myocardial infarction: Development of new Q waves in the ECG or new wall motion abnormalities on echocardiogram or CPK-MB fraction >10% of total CPK. Perioperative insertion of an intraaortic balloon pump (IABP) was defined as a procedure for improving hemodynamics in the presence of cardiogenic shock (The type used was Datascope system 98/XT, Datascope Corp., Montvale, NY). Transient ischemic attack (TIA) was defined as any neurological deficit which disappeared completely within 24 hours. Postoperative cerebrovascular accident was defined as a new neurological dysfunction affecting ambulation or day to day function after operation, and persisting for 24 hours after onset. Atrial fibrillation was defined as sustained atrial arrhythmia requiring treatment.

Statistical analysis:

Demographic, preoperative clinical features, operative and postoperative data were collected and expressed as percentages/mean and standard deviation wherever applicable.

Results:

Preoperative Characteristics:

A total of 30 high risk patients were analyzed in this study. Preoperative patients' characteristics are summarized in Table 1. The mean age of patients was 65.9 ± 8.1 years, with a range of 55 to 84 yrs., 22 (73.3 %) males and 8 (26.6 %) females. Twenty-five patients (83.3%) were in NYHA functional class III–IV. The mean left ventricular EF was 36.8 ± 12.8, eighteen (60 %) patients had EF ≤ 35 %, 7 (23.3%) had EF 35 % - 55 % and 5 (16.6%) patients had EF ≥ 55 %. The preoperative distribution of patient's co-morbidities and risk factors were, DM in 23 (76.6%) patients, 15 of them (50%) on insulin therapy, hypertension in 14 (46.6%) patients, hyperlipidemia in 7 patients (23.3%), COPD in 4 patients (13.3%), renal dysfunction in 9 patients (30%), blood disorders in the form of hyper-spleenism in 1(3.3%) patient with a platelet count of 60.000 per cc, carotid artery disease in 5 patients (16.6%), and peripheral vascular disease in 3 patients (10%). Previous MI presented in 9 patients (30%), and obesity in 6 patients (20%). Six patients (20%) had previous cerebro-vascular accident, 3 of them (10%) with (TIA), 1 patient (3.3%) with monoplegia, and 2 patients (6.6%) with hemiplegia. Aortic calcification was diagnosed by CT chest and heart in 4 (13.3%) patients. There were 10 (33.3%) underwent previous angioplasty, and IABP was inserted in 1 patient (3.3%) during anaesthetic induction due to haemodynamic instability. As regarding distribution of Euro SCORE among the patients, there were four patients (13.3%) with SCORE < 3, 20 patients (66.6%) with score from 3 - 9, and 6 patients (20%) with SCORE > 9.

Variable	Mean ± STD Or No.	Percentage %
No. of patients	30	
Age (years)	65.9 ± 8.1	
Sex		
M	22	(73.3 %)
F	8	(26.66 %)
LVEF %	36.8±12.8	
≤ 35 %	18	(60 %)
35 % - 55 %	7	(23.3%)
≥ 55 %	5	(16.6%)
Preoperative IABP	1	(3.3%)
Previous MI	9	(30%)
Diabetes	23	(76.6%)
Hypertension	14	(46.6%)
Obesity (BMI> 27)	6	(20%)
Blood diseases or coagulation disorders	1	(3.3%)
Hyperlipidemia	7	(23.3%)
COPD	4	(13.3%)
Smoking	15	(50%)
Peripheral vascular disease	3	(10%)
Carotid artery disease	5	(16.6%)
Aortic calcification or atherosclerosis	4	(13.3%)
Previous Cerebrovascular accident	6	(20%)

Renal dysfunction	9	(30%)
Serum creatinine level 1.5-2 (mg/dL)	6	(20%)
Serum creatinine level≥ 2	3	(10%)
CCS angina class		
I	1	(3.3%)
II	4	(13.3%)
III	10	(33.3%)
IV	15	(50%)
NYHA class		
I	-	
II	5	(16.6%)
III	15	(50%)
IV	10	(33.3%)
Previous angioplasty	10	(33.3%)
EuroSCORE (%)		
< 3	4	(13.3%)
3 – 9	20	(66.6%)
9 – 25	6	(20%)
> 25	-	
Mitral incompetence grade		
I	14	(46.6%)
II	3	(10%)
III	-	
IV	-	

Table 1: Preoperative Characteristics
NYHA: New York Heart Association CCS: Canadian Cardiovascular Society
LVEF:Left ventricular ejection fraction IABP: Intraaortic balloon pump
LVEDD:Left ventricular end diastolic dimension
PTCA: Percutaneous transluminal coronary angioplasty.
COPD: Chronic Obstructive Pulmonary Disease
EuroSCORE: European System for Cardiac Operative Risk Evaluation
BMI: Body Mass Index MI: Myocardial Infarction
M: Male F: Female

Operative Details: (Table 2)

All patients underwent single graft LIMA to LAD off-pump. The diagnosis of aortic calcification in four (13.3%) patients done preoperatively by CT chest and heart was confirmed by manual aortic examination and palpation intra-operatively. Intra-coronary shunt was applied in 10 (33.3%) patients, 6 (20%) patients due to sever bleeding at the site of anatomises, and 4 (13.3%) patients due to haemodynamic and ECG changes after proximal snaring. Five (16.6%) patients underwent long LIMA to LAD anatomises in the form of patch arterioplasty due to diffuse disease and successive lesions. Intra-operative transfusion of blood products was needed in 3 patients (10%) due to blood loss and decreased haematocrite level. IABP was inserted intra-operative in 2 patients (6.6%) due to low cardiac-output manifestations. There was no incidence of intraoperative emergency conversion of the procedure from Off-pump to On-pump.

Variable	Mean± STD Or No.	Percentage %
Intracoronary shunts	10	(33.3%)
LAD Patch Arterioplasty	5	(16.6%)
IABP	2	(6.6%)
Blood Transfusion	3	(10%)
Conversion to On-Pump	0	

Table 2: Operative Variable
LAD: Left Anterior Descending IABP: Intra Aortic Balloon Pump

Postoperative findings:

All post-operative data were summarized in Table 3. In this series hospital mortality was (3.3%) one patient, due to perioperative MI and low cardiac output. The patient died after 15 hrs in the ICU with high inotropic support and IABP. The mean of ventilation time for all patients was 9.6 ± 4.86 hrs, inotropic usage time was 8.7 ± 14.09 hrs, and total ICU stay was 42.76 ± 20.79 hrs. Three patients (10%) transferred from operating room to the ICU with low cardiac output manifestations necessitated intra-operative insertion of IABP. The first patient died 15 hrs postoperative, while the IABP was removed with out complications after 24 hours in 2nd patient and 48 hours in the 3rd one. The post- operative mean drainage was 888.9 ± 664.5 ml, and Hb level was 10.2 ± 1.28 mg%. Three patients (10%) were re-ventilated after extubation due to progressive hypoxia, and 2 patients (6.6%) were re-explored for bleeding; one of them had a preoperative hypersleenism with thrombocytopenia (platelets count 60.000 per cc). Postoperative mean blood sugar level in the ICU was 233.8 ± 56.7 mg%, and mean of white blood count was 8773.3 ± 3127.5 per cc. There was no incidence of post-operative cerebro-vascular accidents

Variable	Mean± STD Or No.	Percentage %
Hospital mortality	1	(3.3%)
Periop. MI (no. of pts)	1	(3.3%)
Stroke	0	
Renal dysfunction	12	(40%)
Serum creatinine level 1.5-2 (mg/dL)	8	(26.6%)
Serum creatinine level ≥ 2	4	(13.3%)
Aggravation of preoperative renal dysfunction	3	(10%)
Ventilation time (hrs)	9.6 ± 4.8	
Re – ventilation after extubation	3	(10%)
Drainage (ml)	888.9 ± 664.5	
Blood product (1 unit=350 ml)		
Packed RBCS (unit)	1.7 ± 1.44	
FFP (unit)	4.23 ± 5.1	
Platelets (unit)	3.2 ± 4.38	
Re-exploration for bleeding	2	(6.6%)
Hb (mg %)	10.2 ± 1.2	
Low cardiac output	3	(10%)
Inotrope usage (hrs)	8.7 ± 14.09	
Blood sugar (mg %)	233.8 ± 56.7	
Hypoxia	4	(13.3%)
Respiratory failure	0	
WBC (per cc)	8773.3 ± 3127.5	
ICU stay (hrs)	42.7 ± 20.7	
Post – operative AF	2	(6.6%)
Gastrointestinal complications	1	(3.3%)
Postoperative systemic infection	0	
Deep sternal wound	0	
Hospital stay (days)	7.8 ± 2.8	

Table 3: Post-operative Characteristics
WBC: White Blood Cell Count. ICU: Intensive Care Unite
FFP: Fresh Frozen Plasma RBCS: Red Blood Corpuscles
AF: Atrial Fibrillation Hb: Haemoglobin
MI: Myocardial Infarction

or renal failure needed dialysis, inspite of having 12 patients (40%) with renal dysfunction. Two patients (6.6%) had post-operative AF treated with intravenous (IV) infusion of amiodarone, and 1 patient (3.3%) experienced melena in the 4th day post-operatively with decreased hg level down to 5 mg%. This patient re-admitted to ICU and received blood transfusion, gastrointestinal wash, and IV infusion of proton pump inhibitors. Postoperative total hospital stay ranged from 5 to 15 days (mean of 7.8 ± 2.8).

Discussion:

Many centers have observed an increase in the number of higher-risk patients referred for CABG. [7,8]. Barandon et al,[9] considered patients with additive EuroSCORE > 9, or with at least two of the following criteria, severe LV dysfunction, elderly patients, recent myocardial infarction (MI), terminal renal failure, lung dysfunction, peripheral vascular disease, and body mass index (BMI) > 30, high risk group. Most coronary disorder patients have cerbrovascular disorder concomitantly, which is regarded as a major issue [10]. However, maintenance of pulse pressure with no use of CPB is reported to be effective in case of preoperative multi-organ disorder, such as those complicated by obstructive ventilatory disturbance or by renal dysfunction [11].

The purpose of the present study was to assess early results of Off-Pump LIMA to LAD strategy in patients presenting a high surgical risk, with a wide combination of comorbidities. We defined high risk patients as those patients having one or more co-morbidity rendering them high risk for aortic cannulation and cardiopulmonary bypass. This group of high risk patients included patients with left ventricular dysfunction and low EF, renal dysfunction (not on dialysis), sever calcification and atherosclerosis of the ascending aorta, elderly patients, history of stroke, blood diseases with coagulation disorders, malignancy, chronic obstructive pulmonary disease (COPD). Preoperative analysis of our population confirmed the relatively severe state of all our patients. Twenty percent of our patients had an additive EuroSCORE higher than 9, 30% had previous MI, and 20% of our patients were morbidly obese. Also there were 6 patients (20%) had previous cerebrovascular accident, 30% of patients had renal dysfunction, 3 patients (10%) with sever peripheral vascular disease, and 16% of patients had carotid artery disease. In addition, more than 60% of patients presented impaired left ventricular function (EF< 35%). Off-pump CABG is claimed to have significantly brought down the complications attributed to the use of extracorporeal circulation [12].

The initial fear regarding patency of the grafts done off-ump has been allayed by many reports [13]. This is largely due to the development of excellent stabilizers such as the Octopus III, Genzyme and CTS Guidant systems. Broadly, there are two types of stabilizers, viz. suction devices and compression devices. The Octopus III is the most widely used system and is based on suction stabilization. The Genzyme and CTS Guidant use compression of the target area for immobilization. The Starfish (Medtronics) and Apical Suction Cone (CTS) may further increase the ease and reach of this technique in the future [14]. Occlusion of the coronary artery proximally results in a bloodless field but this could be also achieved by using an intracoronary shunt. These shunts maintain distal perfusion and allow unhurried construction of anastomoses in a blood-free operative field. Furthermore, the arteriotomy could be spread with a soft tensile force applied to the tether of the shunt. This simple method makes it possible to perform safe and precise anastomoses. Also we believe that advancement is attributed to improved anatomises techniques of surgeons and cooperation of anaesthesiologists, medical engineers and operating room staff who can take appropriate measures against changes in haemodynamics on a case-by-case basis. In present study, the Octopus 3 stabilizer (Medtronic Inc., Minneapolis, Minn.) was used for fixation and a blower-mister (Medtronic Inc.) was used for enhanced visualization during performing the end-to-side anastomosis of LIMA to LAD in all cases. As Karagounis and associates,[15] and Ascione et al,[16] applied distal snares in their early experience with perioperative infarcts formation as a clot developed at the site of the distal snare in the postoperative period, we applied 4/0 prolene for proximal control, but no distal control was used. Also silastic shunts were used in 6 patients (20%) due to distal bleeding troublesome, and in 4 patients (13.3%) due to haemodynamic instability and ST changes in the ECG. The results of off-pump coronary artery bypass grafting (OPCAB) will not be very different if aortic anastomosis for inflow is used, because it entails partial clamping of the aorta. Epiaortic scanning and clampless proximal aortic anastomosis may contribute greatly to the reduction of stroke when aortic manipulation is unavoidable[17,18]. Sharony and coworkers,[19] concluded that Off-pump CABG using a single internal mammary artery inflow may circumvent the need for aortic anastomosis. In our study, 4 patients (13.3%) had a preoperatively diagnosed aortic calcification and atherosclerosis, were confirmed

intraoperatively, and all patients had a single vessel disease, LAD lesion, so the only used conduit was LIMA to LAD. Wijeysundera et al,[23] found, in a multicentre trial, that an off-pump approach is associated with lower risk-adjusted mortality and morbidity.

From the available literatures, as Magee et al,[20] it is clear that OPCAB is safe and dependable and is as good as ONCAB in low- and moderate-risk cases, albeit with the possibility of lesser number of grafts per patient. But we believe as well as Al-Ruzzeh et al, [21] that the true value of OPCAB may be in the high-risk group. Hilker and associates,[22] in a review of 507 patients underwent OPCAB with observed mortality of 2.37%, concluded that OPCAB in high risk patients was the superior method with respect to mortality and morbidity. Wijeysundera and associates,[23] reported an operative mortality of 3.2%, and concluded that patients with poor left ventricular function may undergo surgical revascularization using the off-pump technique with good results and low mortality in high risk patients. Barandon et al, [9] in a study of 120 high risk patients underwent OPCAB, reported early mortality of 3%. Parolari et al,[24] in his meta-analysis of currently available randomized trials, previous comparative study of the two techniques of On-pump CAB and OPCAB, concluded that the operative mortality was 1.1 % for ONCAB and 1.4% for OPCAB in their low-risk group. However, in their high-risk group it was 28.5% and 7.7%, respectively. Most of these reports attributed the hospital mortalities in there studies to low cardiac output syndrome and preoperative comorbidities[25,26]. Al-Ruzzeh et al,[27] compared operative results of OPCAB with those of On-Pump/ Beating heart CAB in 305 CAB patients with the LVEF of less than 30 %. While the operative mortality was 6.6 for OPCAB and 14.1% for On-Pump/beating heart CAB, risk factors included ventricular tachycardia and fibrillation but did not include the use of CPB in the patients with reduced left cardiac function. In our study, we had 3.3% hospital mortality, due to perioperative MI and low cardiac output. We believe that small number of studied patients and revascularization with one graft only, LIMA to LAD might be a contributor of this low mortality.

Srinivasan and associates,[28] reported that the systemic inflammatory syndrome is not peculiar to CPB though the degree of inflammation may be more with the use of CPB. In the present study, we experienced no serious infection with a postoperative mean white blood cell count of 8773.3 ± 3127.5 per cc. Stamou et al,[29] reported that the risk of postoperative cerebral infarction was 1.8 times higher for on-pump beating CAB than for OPCAB through a study of 2,320 OPCAB and 8,069 on-pump beating CAB cases. Another report suggested a decrease in neurocognitive function associated with pump use[30]. OPCAB is reported to allow the avoidance of acute pulmonary disorder associated with systemic inflammatory reactions to CPB [31,32]. The total number of complications induced in our study was relatively small, with mean ICU stay hours of 42.7 ± 20.7 and mean total hospital stay of 7.8±2.8 days. Only one of our patients experienced haematemesis and treated in the ICU. Postoperative low cardiac output was evidenced only in 3 (10%) of our patients, another 2 patients (6.6%) experienced postoperative AF, and 12 patients (40%) had post operative renal dysfunction with no renal dialysis needed.

The limitations of this study are that it is nonrandomized, retrospective review of a small cohort of patients and was limited to high risk patients with single graft LIMA to LAD Off-Pump.

In conclusion, Off-Pump LIMA to LAD can be safely done in high risk patients who had single coronary artery disease, LAD lesion with acceptable morbidity and early mortality.The immediate results are satisfactory and long-term follow-up is mandatory. If other workers cooperate in a prospective multicenter studies, OPCAB will be a great advancement in the technique for the care of aged high-risk multivessel candidates for CABG who are seen more often nowadays.

References:

1. Caputo M, Reeves BC, Rogers et al. Monitoring the performance of residents during training in off-pump coronary surgery. J Thorac Cardiovasc Surg 2004; 128:907–915.
2. Hillis GS, Zehr KJ, Williams AW, et al. Outcome of patients with low ejection fraction undergoing coronary artery bypass grafting: renal function and mortality after 3.8 years. Circulation 2006;114(1 Suppl):I-414 –9.
3. Stamou SC, Jablonski KA, Hill PC, et al. Coronary revascularization without cardiopulmonary bypass versus the conventional approach in high-risk patients. Ann Thorac Surg 2005; 79: 552–7.
4. Nurözler F, Kutlu ST, Küçük G, et al. Coronary by-pass for bad ventricle; adoption of “hybrid-pump” bypass. J Cardiothorac Surg 2006, 1:44- 8.
5. Goldstein DG, Beauford RB, Luk B, et al. Multtivessel off-pump revascularization in patients with sever left ventricular dysfunction. Eur J Cardiothorac Surg 2003; 24:72 –80.
6. Puskas JD, Williams WH, Duke PG, et al. Off-pump coronary

artery bypass grafting provides complete revascularization with reduced myocardial injury, transfusion requirements, and length of stay: A prospective randomized comparison of two hundred unselected patients undergoing off-pump versus conventional coronary artery bypass grafting. J Thorac Cardiovasc Surg 2003; 125:797– 808.
7. Weerasinghe A, Athanasiou T, Al-Ruzzeh S, et al. Functional renal outcome in on-pump and Off-pump coronary revascularization: a propensity-based analysis. Ann Thorac Surg 2005; 79:1577–83.
8. Bax JJ, Poldermans D, Elhendy A, et al. Improvement of left ventricular ejection fraction, heart failure symptoms and prognosis after revascularization in patients with chronic coronary artery disease and viable myocardium detected by dobutamine stress echocardiography. J Am Coll Cardiol 1999; 34: 163–69.
9. Barandon L, Richebé P, Munos E, et al. Off-pump coronary artery bypass surgery in very high-risk patients: adjustment and preliminary results. Interact CardioVasc Thorac Surg 2008;7:789-93.
10. Tabata M, Takanashi S, Fukui T, et al. Off-pump coronary artery bypass grafting in patients with renal dysfunction. Ann Thorac Surg 2004;78:2044 – 49.
11. Horai T, Fukui T, Tabata M, etal. Early and mid-term results of off-pump coronary artery bypass grafting in patients with end stage renal disease: surgical outcomes after achievement of complete revascularization. Interact CardioVasc Thorac Surg 2008; 7:218-21.
12. Asimakopoulos G, Karagounis AP, Valencia O, et al. Renal function after cardiac surgery off- vs. on-cardiopulmonary bypass: analysis using the Cockroft-Gault formula for estimating creatinine clearance. Ann Thorac Surg 2005; 79:2024–31.
13. Ascione R, Narayan P, Rogers CA, et al. Early and mid-term clinical outcome in patients with severe left ventricular dysfunction undergoing coronary artery surgery. Ann Thorac Surg 2003; 76:793–799.
14. Ascione R, Lloyd CT, Underwood MJ, et al. Economic outcome of off-pump coronary artery bypass surgery: a prospective randomized study. Ann Thorac Surg 1999;68: 2237–42.
15. Karagounis A, Asimakopoulos G, Niranjan G, et al. Complex off-pump coronary artery bypass surgery can be safely taught to cardiothoracic trainees. Interact CardioVasc Thorac Surg 2006; 5:222-226.
16. Ascione R, Reeves BC, Pano M, et al. Trainees operating in high-risk patients without cardiopulmonary bypass: a high-risk strategy? Ann Thorac Surg 2004; 78:26–33.
17. Kilo J, Baumer H, Czerny M, et al. Target vessel revascularization without cardiopulmonary bypass in elderly high-risk patients. Ann Thorac Surg 2001;71:537– 42.
18. Trehan N, Mishra M, Sharma OP, et al. Further reduction in stroke after off-pump coronary artery bypass grafting: a 10-year experience. Ann Thorac Surg 2001;72(Suppl):1026 –32.
19. Sharony R, Grossi EA, Saunders PC, et al. Propensity casematched analysis of off-pump coronary artery bypass grafting in patients with atheromatous aortic disease. J Thorac Cardiovasc Surg 2004;127:406 –13.
20. Magee MJ, Coombs LP, Peterson ED, et al. Patient selection and current practice strategy for off-pump coronary artery bypass surgery. Circulation 2003;108(Suppl 1):II- 9–14.
21. Al-Ruzzeh S, Nakamura K, Athanasiou T, et al. Does off-pump coronary artery bypass (OPCAB) surgery improve the outcome in high-risk patients? A comparative study of 1398 high-risk patients. Eur J Cardiothorac Surg 2003; 23:50 –5.
22. Hilker M, Oelert H, and Hake U. A review of 507 off-pump coronary bypass patients: a single center experience. Interact CardioVasc Thorac Surg 2003;2:246-250
23. Wijeysundera DN, Beattie WS, Djaiani G, et al. Off-pump coronary artery surgery for reducing mortality and morbidity: meta-analysis of randomized and observational studies. J Am Coll Cardiol 2005; 46:872–82.
24. Parolari A, Alamanni F, Cannata A, et al. Off-pump versus on-pump coronary artery bypass: meta-analysis of currently available randomized trials. Ann Thorac Surg 2003; 76: 37–40.
25. Carr JA, Haithcock BE, Paone G, et al. Long-term outcome after coronary artery bypass grafting in patients with severe left ventricular dysfunction. Ann Thorac Surg 2002; 74: 1531–1536.
26. Sabik JF, Gillinov AM, Blackstone EH, et al. Does off-pump coronary surgery reduce morbidity and mortality? J Thorac Cardiovasc Surg 2002;124:698 –707.
27. Al-Ruzzeh S, Athanasiou T, George S, et al. Is the use of cardiopulmonary bypass multtivessel coronary artery bypass surgery an independent predictor of operative mortality in patients with ischemic left ventricular function? Ann Thorac Surg 2003; 76:444-51.
28. Srinivasan AK, Grayson AD, Fabri BM, et al. On-pump versus off-pump coronary artery bypass grafting in diabetic patients: a propensity score analysis. Ann Thorac Surg 2004; 78: 1604–9.
29. Stamou SC, Hill PC, Haile E, et al. Clinical outcomes of nonelective coronary revascularization with and without cardiopulmonary bypass. J Thorac Cardiovasc Surg 2006; 131:28–33.
30. Diegeler A, Hirsch R, Schneider F, et al. Neuromonitoring and neurocognitive outcome in off-pump versus conventional coronary bypass operation. Ann Thorac Surg 2000; 69: 1162–6.
31. Rastan AJ, Bittner HB, Gummert JF, et al. On-pump beating heart versus off-pump coronary artery bypass surgery— evidence of pump-induced myocardial injury. Eur J Cardiothorac Surg 2005; 27:1057– 64.
32. Mathisen L, Andersen MH, Hol PK, et al. Patient reported outcome after randomization to on-pump versus off-pump coronary artery surgery. Ann Thorac Surg 2005; 79:1584–9.

Incidence and Management of Pleural Effusion after Coronary Artery Bypass Grafting Surgery

Nezar El Nahal MD*,
Mohammed Abdel-Aal MD*,
Ahmed Nageb MD*,
Yasser A.AlRahman MD,**
Bakir M Bakir MD ***

Bakground: **Pleural effusion is very common after coronary artery bypass grafting (CABG) surgery, occurring in up to 50% to 75% of patients in the 1st week after operation. The present study was designed to determine the prevalence and management of pleural effusion after coronary artery bypass surgery.**

Methods: **This study included 568 patients who underwent CABG; 268 developed postoperative pleural effusion. Chest radiographs were obtained during hospitalization, one immediately after surgery, one every postoperative day , one after chest tube removal and when they returned for their regular follow-up visit 2 weeks postoperatively and monthly for 3 months.**

Results: **The size of the pleural effusion was small in most of the patients 57.67%(n = 154); 61patients (22.84%) had a moderate effusion in which it was occupied 25-50% of hemithorax, and 52 patients (19.47%) had a large effusion (occupying > 50% hemithorax. The primary symptom associated with these larger effusions was dyspnea. The effusions tended to resolve spontaneously in most cases and thoracocentesis or thoracostomy tube required in small percentage of patients.**

Conclusion: **47% of patients after CABG surgery developed pleural effusion, about 19.5% that occupies more than 50% of the hemithorax. Most effusions after CABG surgery are left sided. If the effusion is moderate or large and the patients have dyspnea, drainage should be considered.**

Key words: **pleural effusion, coronary bypass**

Pleural effusion is a common consequence following heart surgery, but in most cases the fluid collection is small and not clinically significant. Some patients, however, develop a significant effusion during the initial hospitalization or after hospital discharge, which requires drainage to relieve respiratory symptoms.(1) The reported incidence of symptomatic effusion varies widely in the literature, some reported that approximately 50% of patients undergoing CABG develop a pleural effusion. in other report ,pleural effusion occurs in up to 89% of patients during the first week after coronary artery bypass grafting (CABG). (2) The incidence of post-CABG pleural effusions is higher in patients who receive internal mammary artery (IMA) grafts than in those who receive saphenous vein grafts. The etiology of these persistent effusions remains unknown. Most effusions are diagnosed in the early postoperative course; however, some attain their maximal size weeks to months after surgery. (3) This retrospective study was undertaken to determine the incidence of pleural effusion after coronary artery bypass graft surgery.

Address reprint request to:
Dr. Nezar El Nahal
Accepted for publication : 5/2009
Address reprint request to : Dr. Nezar El Nahal MD Department of cardiothoracic surgery - Zagazig University- Egypt.
Email : jegyptscts@gamil.com
Codex : 04/108/0906

Methods:

The study included 568 patients, who underwent coronary artery bypass graft surgery between June 2005 to may 2008 at King Khalid cardiac center, King Saud University Riyadh, Saudi Arabia. The patients were reviewed and data were collected regarding age, sex, type of surgery, site , size of effusion and timing of occurrence of effusion after CABG. Preoperative and postoperative data were obtained from database registry. Information on postoperative events after hospital discharge obtained from outpatient clinic records during the first three months.

The size of pleural effusion was estimated according to criteria based on semiquantitation method used by Light and coworkers by visually estimating the percentage of the area of the hemithorax occupied by pleural fluid on lateral chest radiograph. The small effusion occupied less than 25% of the hemithorax, a moderate effusion occupied 25% -50%, and a large effusion occupied more than 75% Table 1(2).

Grade of Effusion	Characteristic of Chest Radiograph
0	No pleural effusion
1	Blunting of the costophrenic angle
2 (small)	More than blunting of the costophrenic angle but less than 25% of hemithorax occupied by pleural fluid
3 (moderate)	Pleural fluid occupying 25% to 50% of hemithorax
4 (large)	Pleural fluid occupying 50% to 75% of hemithorax
5 (massive)	Pleural fluid occupying more than 75% of hemithorax

Table 1. Semiquantitation of Size of Pleural Effusions (2)

Effusion	No. of patients & %
Early effusion	189 (70.78%)
Late effusion	78 (29.22%)
Left side	256 (95.5%)
Right side	3 (1.1%)
Bilateral	8 (2.98%)

Table 2: Time of occurrence and size of effusion.

All patients in the study had CABG , most of them were operated by conventional technique that utilized cardiopulmonary bypass and blood cardioplegia, while others were operated with on-pump beating heart technique. Each patient had the mediastinum and the left pleural cavity were drained with two chest tubes. The chest tubes were removed at the first postoperative day when the drainage became less than 80 cc in the previous 8 hours. Otherwise, the tubes were left in place for another 12 to 24 hours or until the drainage became less than 80 cc/8 hours . All patients were started on aspirin (81 mg/ day) from the 1st postoperative day. Anticoagulants or other antiplatelets were used only in special situations. All patients had postero-anterior and lateral chest radiograph during hospitalization, one immediately after surgery, one every postoperative day , one after chest tube removal and when they returned for their regular follow-up visit 2 weeks postoperatively and monthly for 3 months. Effusions with no definite cause were classified as sanguineous or non- sanguineous according to the gross description of the pleural fluid.

Statistical Analysis The data are expressed as the mean ± SD if normally distributed. The characteristics of the patients with no effusion, small effusions, and large effusions were compared using one-way analysis of variance.

Results:

568 patients underwent CABG in our center, mean age was 64 ± 10 years; 410 were male and 158 were female. Most of the patients 87.67% (n=498) included in the study underwent CABG using left internal mammary artery grafts (LIMA) in addition to saphenous vein (SV) grafts ,while 50 patients (12.32%) received only SV grafts due to various causes including age >70 years, emergency cases, inadequate length or flow of LIMA.

In general, there was no significant relationship between the occurrence of a pleural effusion and the age of the patient, the sex of the patient, operative time, or number of grafts received.

The prevalence of pleural effusion in the 568 patients who underwent isolated CABG surgery was 47% (267 patients).The prevalence of pleural effusions was clearly higher in patients who received left internal mammary artery grafts 85.5% (254 patients). The size of the pleural effusion as estimated from the lateral chest radiograph using our criteria was small in most of the patients

57.67%(n = 154); 61patients (22.84%) had a moderate effusion in which it was occupied 25-50% of hemithorax, and 52 patients (19.47%) had a large effusion (occupying 50%-75% of hemithorax) , no patient in this study presented with massive effusion >75% of hemithorax figure (1).

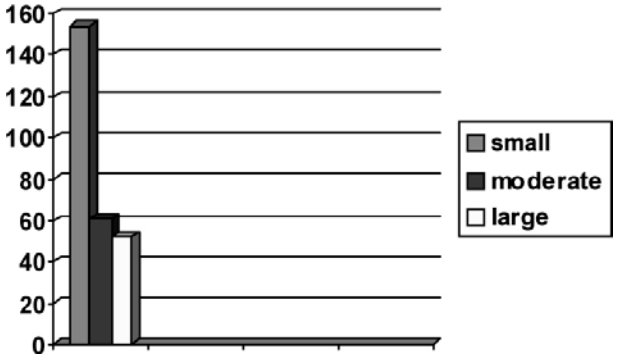


Figure 1: degree of effusion.

Early effusions occurred within the first 2 weeks of surgery in 189 patients (70.78%), whereas 78 (29.22%) developed late pleural effusions after one month of surgery. The overall incidence of symptomatic large pleural effusion in the study was 19.47% (52 of 267), in 38 patients the effusions occurred during the initial hospitalization, but in 14 patients the effusion occurred after hospital discharge. Symptoms include cough 72%, shortness of breath 67% and chest pain in 42% of patients. Most of the effusions were predominantly left sided, 256 patients (95.5%) developed a left-sided pleural effusion, 3(1.1%) developed a right-sided pleural effusion, and 8 (2.98%) developed bilateral pleural effusions, (table 2). The character of effusion was serous in 342 patients (60.2%) while it was serosanginous in 165 patients (29%) and sanginous effusion was observed in 61 patients (10.7%),figure 2. Sanginous and serosanginous effusions reached their maximum size earlier than serous effusions (12 days versus 35 days).

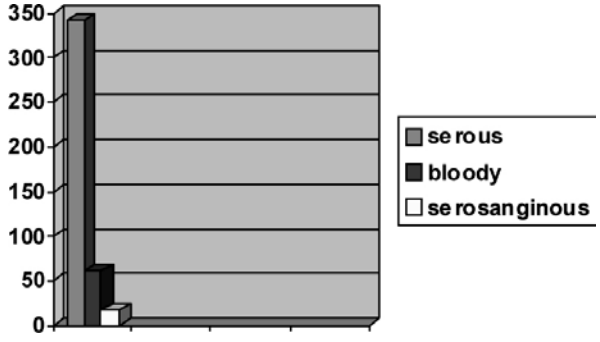


Figure 2: character of effusion

All the patients with small effusion were managed conservatively, whereas thoracocentesis was done in 38 patients (14.17%) with moderate symptomatic effusion. All the patients with large effusion (19.47%) were treated by thoracostomy tube drainage which was removed after complete drainage of the effusion. The average chest tube stay was 2.9±2.1 days. Patients who underwent thoracentesis or thoracostomy tube reported that their dyspnea was effectively alleviated, figure 3. Anti-inflammatory agent (Indomethacin) used in 4 patients who had prolonged significant drainage.

Figure 3: management of effusion.

Discussion:

Many theories had been suggested to explain the etiology of post CABG pleural effusion, the majority of these explanations linked the formation of pleural effusion to the surgical trauma occurred during CABG procedures especially when LIMA was used, this may be attributed to pleurotomy to allow harvesting of LIMA which allows blood to enter the pleural cavity, or creation of row surface at pedicle harvesting site that can be source of fluid that accumulate to form the effusion (1). Several other factors may also contribute to the formation of effusion such as congestive heart failure; pulmonary embolism; atelectasis; and use of drugs such as amiodarone, procainamide hydrochloride, and -adrenergic blocking agents. It is important to include these in the differential diagnosis of effusions occurring in patients who have undergone CABG(4).

The incidence of pleural effusions immediately after surgery in patients undergoing CABG has been reported to be 42% to 89%.(4) This wide variability in the incidence of post-CABG effusions is probably related to the technique used to diagnose the pleural effusion because higher rates have been reported when ultrasound

or computed tomography is used. The etiology of post-CABG effusions and the reasons why some effusions persist while most resolve remain unknown. The mechanism of the formation of pleural fluid after CABG is not clear. (5) Most post-CABG effusions are small, left sided, and regress spontaneously. Occasionally a patient may develop a moderate to large symptomatic pleural effusion after CABG. The prevalence of large effusions after CABG is not definitely known but has been reported to be 1% to 4%. Most effusions occur in the early postoperative period; however, some effusions reach their maximum size only months after CABG. (6)

The present study demonstrates that more than 47% of patients have a pleural effusion after CABG and that 27.1% of patients had a small pleural effusion that occupies less than 25% of the hemithorax at this time and 10.7% of them had moderate effusion and 9.1% had large effusion that occupied more than 75% of hemithorax. The study of Light and coworkers(5) demonstrated that more than 50% of patients had a pleural effusion 1 month after surgery and that approximately 10% of patients had a pleural effusion that occupies more than 25% of the hemithorax at this time. Vargas and coworkers (7) assessed the prevalence of pleural effusion in 47 patients at 30 days after CABG surgery and reported that the prevalence of pleural effusion was 57.4%, which is comparable to the prevalence of 62.4% in the present study. Lancey and coworkers (8) reported that the prevalence of moderate or large pleural effusions 6 weeks after surgery was 11.4%, which is comparable to the prevalence that we found. In this latter series, 5.9% of the patients required a drainage procedure for their pleural effusion in the 6 weeks postoperatively.

The incidence of pleuropulmonary complications is higher with use of IMA grafts compared with saphenous vein grafts. This difference has been attributed to the necessity to enter the adjacent pleural space while mobilizing and harvesting the IMA.(9)

Most of the patients (n=498) included in the our study underwent CABG using left internal mammary artery (LIMA) and saphenous vein (SV) and only in 50 patients SV was used. 254 of the patients with use of LIMA graft (85.5%) had pleural effusion. All the patients with small effusion were managed conservatively, whereas thoracentesis was done in 38 (62.2%) patients with moderate symptomatic effusion. 9.1% of the patients with large effusion were treated by thoracostomy tube which was removed after complete drainage of the effusion.

In a study by Hulburt and associates (3), 200 patients were followed up after surgery (100 with IMA grafts and 100 with saphenous vein grafts); 4% of patients receiving IMA grafts required a thoracentesis or tube thoracostomy by the sixth postoperative day. Two months after surgery, 10.5% of patients who had undergone IMA grafting had a pleural effusion, one of whom required thoracentesis. Areno and associates(10) followed the postoperative course of 200 patients receiving IMA grafts and reported that 8.5% required a thoracentesis immediately after surgery. At 3 months, 20% of patients had a pleural effusion but only 1.5% required thoracentesis. Landymore and Howel (11) reported that none of their 67 patients required a thoracentesis during 3 months of follow-up.

The nature of pleural fluid was studied by Sadikot and associates, they reported that all effusions were exudative in nature, however they found two distinct categories of effusions , the early effusions were usually bloody with high esinophil count mostly linked to surgical trauma, whearse the late effusions had a predominance of lymphocytes which may represents an immune reaction which may be a variant of postcardiotomy syndrome(4) .

The cause of large pleural effusions occurring after CABG is not clear. Sanginous effusions most probably are related to bleeding into the pleural space. Non-sanginous pleural effusions may be due to a variant of the postpericardiotomy syndrome. Other contributing factors may include postoperative pericarditis/pleuritis, congestive heart failure, atelectasis, and an incompletely drained hemothorax. Other possible explanations for nonbloody pleural effusions are interruption of the lymphatics that normally drain the pleural space, leakage of fluid from the mediastinum, damage from topical hypothermia, or a hypersensitivity reaction to a drug(12). Nikas and coworkers(13) reported that 60% of 191 patients who underwent topical hypothermia (bathing the myocardium in iced slush) developed a pleural effusion and 25% required thoracentesis. In contrast, only 25% of 314 patients who did not undergo topical hypothermia developed a pleural effusion and only 8% required thoracentesis. Asanginous pleural effusions seem more difficult to manage. On the basis of our experience, we recommend that these patients initially be managed with therapeutic thoracentesis and an anti-inflammatory agent, such as indomethacin or corticosteroid as prednisone. Payne and coworkers(1) reported that, they could not identify any patient-related or procedure-related factors that were associated with effusion, except

for a higher incidence of insulin-dependent diabetes and chronic obstructive pulmonary disease in patients who subsequently develop a symptomatic effusion. The mechanism by which these factors could mediate pleural effusion is not clear.

In conclusion approximately 47% of patients after CABG surgery may develop a pleural effusion, about in 19.5% of patients effusion occupies more than 50% of the hemithorax. Most effusions after the CABG surgery are left sided. If the effusion is moderate or large and the patients have dyspnea, drainage should be considered, as it is effective in relieving symptoms and in preventing re-accumulation of the effusions.

Refereneces:

1. Payne M, Magovern G, Benckart D, Vasilakis A, Szydlowski G, Cardone J, Marrone G, Burkholder J, Magovern J. Left pleural effusion after coronary artery bypass decreases with a supplemental pleural drain. Ann Thorac Surg 2002;73:149-152.
2. Light R, Rogers J, Cheng D, Rodriguez R. Cardiovascular surgery associates: large pleural effusions occurring after coronary artery bypass grafting. Ann Intern Med 1999;130:891–896.
3. Hurlbut D, Myers M, Lefcoe M, Goldbach M. Pleuropulmonary morbidity: internal thoracic artery versus saphenous vein grafts. Ann Thorac Surg 1990;50:959-964.
4. Sadikot R, Rogers J, Cheng D, Moyers P, Rodriguez M, Light R. Pleural fluid characteristics of patients with symptomatic pleural effusion after coronary artery bypass graft surgery. Arch Intern Med 2000;160:2665–2668.
5. Light R, Jeffrey J, Moyers J, YCG L, Rodriguez M, Alford W, Ball S, Burrus GR. Prevalence and Clinical Course of Pleural Effusions at 30 Days after Coronary Artery and Cardiac Surger. Am J Resp Crit Care Med 2002, 166;1567-1571.
6. Piccoli M, Trambaiolo P, Salustri A, Cerquetan E , Posteraro A, Pastena G, Amici E, Papetti F, Marincola E, La Carruba S. Bedside Diagnosis and Follow-up of Patients With Pleural Effusion by a Hand-Carried Ultrasound Device Early After Cardiac Surgery Chest 2005; 128: 3413 - 3420.
7. Vargas F, Cukier A, Terra-Filho M, Hueb W, Teixeira L, Light R. Relationship between pleural changes after myocardial revascularization and pulmonary mechanics. Chest 1992;102:1333-1336.
8. Lancey R, Gaca C, Vander Salm T. The use of smaller, more flexible chest drains following open heart surgery. Chest 2001;119:19–24.
9. Weissman C. Pulmonary Complications After Cardiac Surgery. Semin Cardiothorac Vasc Anesth 2004; 8(3): 185 - 211.
10. . Areno J, McCartney J, Eggerstedt J, Grafton W, George R. Persistent pleural effusions following coronary bypass surgery. Chest 1998;114:311-314.
11. Landymore R, Howell F. Pulmonary complications following myocardial revascularization with the internal mammary artery graft. Eur J Cardiothorac Surg 1990;4:156-162.
12. Lee Y, Vaz M, Ely K, McDonald E, Thompson P, Nesbitt J, Light R. Symptomatic persistent post-coronary artery bypass graft pleural effusions requiring operative treatment: clinical and histologic features. Chest 2001;119:795–800.
13. Nikas D, Ramadan F, Elefteriades J. Topical hypothermia: ineffective and deleterious as adjunct to cardioplegia for myocardial protection. Ann Thorac Surg 1998;65:28–31.

Predictors of Postoperative Renal Replacement Therapy in patients with Impaired Kidney Function Undergoing on Pump Coronary Surgery

Sameh Sayed MD*,
Mosaad Abolnaser MD,**
Hazem Zaki MD,**
Walid Shaker MD *,**
Mahmoud Daoud MD,**
Sherin Kouderia MD **

Background: **Acute renal failure after Cardiac surgery is associated with high mortality rate. In a trial to determine the predictors for postoperative renal replacement therapy in patients undergoing CABG with impaired kidney function. We performed this study.**

Methods: **This is a retrospective analysis of 565 patients who underwent CABG over 3 years. A total number of 64 patients with preoperative impaired kidney function (serum creatinine ≥ 115 μ mol/L) divided into two groups were enrolled in this study.**

Group 1 (45 Patients) who required postoperative renal replacement therapy. Group 2

(19 Patients) who did not require renal replacement therapy post CABG.

Results: **Both groups were comparable as regards age, sex, and preoperative co-morbid diseases such as diabetes; chronic obstructive pulmonary disease, cerberovascular disease or peripheral vascular disease.**

Serum creatinine was significantly higher in group 1 (135±10) versus (115±9) in group 2 (P = 0.0001). However, creatinine clearance was significantly lower in group 1 (40±20) versus (60±10) in group 2 (P<0.0001).

Patients in group 1 required longer intubation, more inotropic support. Total ICU stay was significantly longer in group 1 (18±7 days) versus (4±2 days) (P= 0.0001).

Group 1 had longer hospital stay than group 2 (35±15 Vs 12±2 days). (P= 0.0001).

Mortality occurred in 40% of patients who required post operative renal replacement therapy in comparison to 15% in patients who did not require renal replacement therapy. (P = 0.02).

Multivariate and logistic regression model analysis identified ; age, diabetes mellitus requiring insulin therapy, creatinine clearance, emergency operation, total bypass time and post operative inotropic therapy as the most important factors which influenced postoperative renal replacement therapy. Odds ratio analysis identified creatinine clearance (3.34), and longer bypasses time (2.3) as the highest odds ratio among the studied factors.

Conclusion: **Creatinine clearance and long bypass time are the most important predictors of renal replacement therapy in our study. Patients requiring post operative dialysis have a high rate of mortality and an alternative way for coronary revascularization should be attempted whenever possible.**

Adress for reprints Dr. Sameh Abd El-Rahman M. Sayed. Prince Salman Heart Center (PSHC)-
King Fahad medical city (KFMC).
P.O Box 59046- Riyadh 11525.
Kingdom of Saudi Arabia.
* Lecturer of cardiothoracic surgery Assiut University. Assiut-Egypt
** Lecturer of Anesthesia and Intensive care. Ain Shams University – Cairo- Egypt.
**** Lecturer of Cardiothoracic surgery Ain Shams University – Cairo- Egypt.
**** Assistant professor of Anesthesia and Intensive care. Ain Shams University – Cairo – Egypt.
Tel: +966-532935840.
E mail: samsayed@lycos.com
Codex : 04/109/0909

Acute renal failure requiring renal replacement therapy is one of the most serious complications occurring after cardiac surgery (1). Several studies reported the incidence and consequences of acute renal failure requiring renal replacement therapy after cardiac surgery; with estimated incidence between 1 to 30% (2).

When renal failure develops following cardiac surgery, renal replacement therapy is usually indicated. This situation is associated with prolonged hospital stay, increasing cost and increased mortality, which may reach to up to 40% (3).

In spite of the great advances in all aspects of coronary surgery, including improvements in operative techniques, post surgery intensive care, safer and more efficient use of cardiopulmonary bypass ; the high mortality associated with postoperative dialysis after cardiac surgery remains unchanged during the past few decades (4).

Elevation in the serum creatinine (Cr) has been commonly used as marker of Acute renal failure (ARF) in hospitalized patients. In some long term studies , renal function has been defined according to elevation in serum creatinine (5) However, these studies did not specify a thresholds for defining clinically effective preoperative renal insufficiency (6). Other studies have consistently identified preexisting renal insufficiency as an independent predictor to the need for postoperative renal replacement therapy (RRT) after coronary surgery (7).

This study was undertaken to evaluate the potential factors which may predict postoperative renal replacement therapy in patients with preoperative impaired kidney function who underwent CABG in our institution.

Our definition for impaired kidney function was according to guidelines from the national kidney foundation (8); Elevated serum creatinine level above the normal limits (90-115 µ mol/l) was defined as impaired kidney function in our study.

Methods:

Between October 2005 and October 2008, total number of 565 consecutive patients who underwent CABG surgery in our institution (King Fahad Medical City (KFMC) - Prince Salman Heart Center (PSHC). Were retrospectively reviewed the charts of the patients. Sixty four patients with impaired kidney function were enrolled in this study.

Our definition for impaired kidney function include those patients with estimated serum creatinine more than 115 µ mol/L (which is the upper limit of normal), and with creatinine clearance (Cr Cl) less than 60 ml /hr, according to Cokcroft- Gault equation (9).

Patients are classified into two groups:

Group 1 (n= 45), include those patients with impaired kidney function who underwent CABG and required post CABG renal replacement therapy.

Group 2 (n= 19) those patients with impaired kidney function who underwent CABG and did not require postoperative renal replacement therapy.

After Approval of our institutional research and ethical committee; all clinical, operative and intensive care postoperative data were reviewed retrospectively through the review of patient charts.

Indications for Renal Replacement Therapy (RRT) were:

1. Hyperkalaemia (> 6.0 m mol) not responding to insulin infusion.
2. Continuous metabolic acidosis in blood gases, not corrected with Sodium Bicarbonate.
3. Anuria or oliguria < 20 ml / h for more than 4 consecutive hours, despite adequate filling and adequate cardiac output, and after trial of Frusmide infusion.

RRT was commenced through the nephrologist's recommendation.

Patients with the following criteria were excluded form our study;

1. Patients who underwent CABG with normal kidney function.
2. Patients who underwent CABG and they are already under renal replacement therapy (dialysis)
3. Patients who underwent CABG without cardiopulmonary bypass (Off Pump).
4. Patients who underwent CABG combined with other procedures such as valve replacement or repair.

Data Analysis:

Factors of interest are analyzed in both groups using univariate and multivariate analysis together with logistic regression analysis. The possible predictors were determined. Software Package SPSS version 11.0 (Spas Inc., Chicago) was used to perform statistical analysis. Results were considered statistically significant when P value was < 0.05.

Definitions:

We used the following definitions in our study:

Operative priority was determined by cardiothoracic surgeons according to standard criteria

- Elective operation means that the medical factors indicate the need for an operation through readmission at a latter date.
- Urgent operation means that the medical factors require the patient to have an operation during the same admission(before discharge)
- Emergency surgery refers to medical factors related to the patient's cardiac condition requiring emergency surgery which should be performed within hours to prevent morbidity or death.

Creatinine clearance was calculated using Cockcroft-Gault equation (9) as follow: Creatinine clearance (ml/ min) = (140- age in years)* actual weight (KG) / serum creatinine (μ mol/l). This formula is applicable to females, and in case of the males the formula is multiplied by 1.2. Normal range of serum creatinine clearance is > 90 ml/ min.

In hospital mortality refers to all mortalities within the same admission post CABG regardless of their length of stay or within thirty days of surgery.

Cardiopulmonary Bypass (CPB):

All operations were performed thorough standard median sternotomy. Following full anticoagulation with heparin given at a dose of 300 IU/ Kg to maintain an activated clotting time of 400-600 sec.

CPB was initiated using ascending aortic cannulation for arterial perfusion and a two stage right atrial venous cannulation for venous return. A membrane oxygenator was used for the CBP (Primox- Sorin group- Sorin Italy). The Extracorporeal circulation was primed with 1500 ml of ringer lactate solution and 5000 IU heparin. CBP was maintained with non pulsatile flow with a minimum flow rate of 2.4 l/m /min and temperature allowed to drift between 32 to 34 ºC. Arterial line filtration was used in all cases.

Shed blood was recycled using cardiotomy suction. Acid base was managed with alpha stat control. Myocardial protection was achieved with intermittent antegrade cold/ tepid blood cardioplegia. On completion of the distal anasotomses, the aortic cross clamp was removed and the proximal anastomoses performed with the partial aortic clamping. Heparin was reversed with protamine at 1:1 ratio on weaning off cardiopulmonary bypass.

Results:

Univariate analysis of individual risk factors and their relevant p value is summarized in table 1. The incidence of acute renal failure requiring renal replacement therapy in our studied patients is (7.9%).

Our study showed that there is no statistically significant difference in the preoperative patient characteristics in both groups in terms of age, sex, and comorbid diseases.

The incidence of diabetes in the group of patients who required renal replacement therapy (group 1) is 73% versus 79% in (group 2) (P = 0.07) hypertension was present in 93% of the patients in group 1 versus 84% of the second group (P= 0.3).

There are also no statistically significant differences in the incidence of chronic obstructive pulmonary disease, cerberovascular disease, or peripheral vascular disease in both groups.

Serum creatinine was significantly higher in the group of patients who required renal replacement therapy (135± 10μ mol/l) versus (115± 9μ mol/l) in the group of patients who did not require renal replacement therapy (P < 0.0001) and serum creatinine clearance was also significantly lower in group 1 (40± 20 ml/min) versus (60± 10 ml/min) in the patients who did require renal replacement therapy (P < 0.0001).

There was no statistically significant difference in the preoperative left ventricular function (EF) in both groups.

Regarding operative factors, there were no statistically significant differences between both the studied groups in the operative priority, timing of coronary angiography within 72 hours before surgery, and the incidence of preoperative insertion of intra aortic balloon pump.

However, aortic cross clamp time, and total bypass time were significantly longer in the patients who require post operative renal replacement therapy (group 1) (112±14 and 143±9 min respectively) versus (73±16 a101± 7 min respectively) in group 2. (P = 0.0001).

Immediate post operative data is depicted in table 2.

Post operative complications were significantly higher in group 1 in comparison to group 2. Hours of mechanical ventilation was longer in group 1 (91.9± 14.6) versus 41.2±12.4 in group 2 (P= 0.0001). Reintuabtion and tracheostomy was required in 45% of the patients in group 1, while it was required only in 16% of the patients in the second group (P= 0.04).

Total ICU stay as well as total length of hospital stay was significantly longer in group 1 in comparison to Group 2 (p = 0.0001).

Hospital mortality occurred in 40% of the patients who required renal replacement therapy in comparison to 15 % of the patients who did not require renal replacement therapy (P = 0.02).

Characteristics	Group (1) Patients required Post operative renal replacement therapy (N= 45)	Group (2) Patients did not require postoperative Renal replacement therapy (N = 19)	P value
Age	68 ± 12	62±15	0.09
Female	14 (31 %)	9 (47%)	0.3
Preoperative renal function:			
Serum creatinine Conc.	135± 10	115±9	*<0.0001
Creatinine Clearance	40± 20	60±10	*<0.0001
Comorbid Diseases			
Diabetes mellitus requiring insulin	33 Pt (73 %)	15 Pt (79 %)	0.7
Hypertension	42 Pt (93 %)	16 Pt (84%)	0.3
Chronic obstructive pulmonary disease	12 pt (26 %)	7 Pt (37%)	0.55
Cereberovascular disease	12 Pt (26 %)	4 Pt (21%)	0.7
Peripheral vascular disease	13 Pt (29 %)	6 Pt (31%)	1.0
Left Ventricular EF			
≥ 60 %	12 pt (26%)	9 pt (46.5 %)	0.16
21- 60%	29pt (64%)	7 pt (37%)	0.055
≤ 20%	4 pt (8%)	3 pt (15.5%)	0.4
Preoperative congestive heart failure	6 pt (13 %)	2 pt (10.5%)	1.0
Coronary Angiography within 72 hours before surgery.	21 pt (46%)	8 pt (42%)	0.7
Operative Priority:			
Emergency	9 pt (20%)	3 pt (15.5%)	1.0
Urgent	23 pt (51%)	8 Pt (42%)	0.5
Elective	14 pt (31 %)	8 pt (42%)	0.4
Intra Aortic Balloon Pump (IABP)	14 pt (31 %)	5 pt (26%)	1.0
Aortic cross clamp time	112± 14	73± 16	*0.0001
Total bypass time	142±22	95±18	*<0.0001

Table 1 Preoperative and operative patient characteristics:
EF Ejection fraction * Statistically significant data - Pt = Patient

	Group (1) Patients required Post operative renal replacement therapy (N= 45)	Group (2) Patients did not require postoperative Renal replacement therapy (N = 19)	P value
Hours of mechanical ventilation	91.9± 50.6	41.2±12.4	* 0.0001
Myocardial support			
Yes	41 pt (91 %)	11 pt (58%)	*0.003
No	4 pt (9 %)	8 Pt (42%)	
Postoperative pulmonary complication			
Non	25 Pt (55%)	16 pt (84%)	*0.04
Reintuabtion- CPAP- Tracheostomy	20 pt (45 %)	3 pt (16%)	*0.04
Total stay in the ICU (days)	18 ± 7	4±2	*0.0001
Total hospital stay (days)	35± 15	12±2	* 0.0001
Mortality	18 pt (40%)	2 pt (15%)	* 0.02

Table2 Immediate postoperative and outcome data:

Predictive modeling:

Logistic regression modeling was used to estimate the risk of dialysis. Variables were selected by performing backward stepwise method to identify factors associated with continuous renal replacement therapy Factors of interest are summarized in table 3

Variable	Odds Ratio	95% confidence Interval	p- Value
Age	0.95	-1.1 – 13.1	0.09
Diabetes mellitus requiring insulin therapy	1.4	0.95- 12.4	0.07
Creatinine clearance	3.34	10.3- 29.7	<0.0001*
Emergency operation	1.9	0.83 – 14.5	0.06
Total bypass time	2.3	31.0 – 47.0	<0.0001*
Post operative inotropic support	0.83	-2.5 – 11.5	0.08

Table 3 Mulivariate analysis of the predictors of renal replacement therapy in the studied groups:

Discussion:

In this study we have attempted to correlate and predict factors predisposing to ARF which ultimately lead to renal replacement therapy (RRT) in patients with already impaired kidney function undergoing CABG. We studied preoperative, intra-operative and immediate postoperative factors. We tried to define those patients who are at high risk of requiring postoperative dialysis in the studied groups. So we can determine the operative risk and predict the postoperative course of them, and if possible find an alternative way of management of those patients.

In our study, we identified older age as one of the predisposing factors for RRT, inspite of that there is no statistically significant differences in the age of both studied groups however, elderly patients with impaired kidney function have low creatinine clearance and hence they are more liable for postoperative renal replacement therapy.

Our results are similar to the results of another study, which also confirmed that elderly patients with low body weight are more liable for RRT after CABG (10)

The effect of diabetes on RRT post cardiac surgery is still controversial. Some studies have shown increased risk of RRT post surgery among diabetics, others failed to show such a risk. This difference maybe due to the prevalence of diabetes among those patients with ischemic heart disease undergoing CABG [11].

In our study, insulin dependant diabetes mellitus was insignificantly associated with postoperative renal replacement therapy (Odds ratio 1.4) (P 0.7) this is may be attributed to that both of the studied groups have almost the same preoperative incidence of insulin dependant diabetes mellitus.

IDDM have been shown to be associated with glomerular sclerosis which increase the risk of renal failure and make patients are more susceptible to ischemic insults during cardiopulmonary bypass [12].

The risks for postoperative dialysis are clearly related to preoperative kidney function. In some long term studies, renal function has been defined according to the levels of serum creatinine and not to estimated glomerular filtration rate (CR Cl) (13). These studies have used serum creatinine level of at least 133 μ mol/L as an indicator of impaired kidney function [14].

The ability of serum creatinine to identify patients with impaired kidney function is limited [15]. Since serum creatinin concentrations are influenced by several other factors than the filtration of creatinine. Therefore, assessment of renal function will be improved by using creatinine Clearance (CR CL).

Our study found that the risk of postoperative RRT increases appreciably when creatinine clearance decreases below 60 ml/min. Our definition of clinically significant preoperative renal impairment (Cr Cl< 60 ml/hr) is in accordance with other recent study; which found that patients with creatinine clearance below 60 ml/min should be considered to have clinically important preoperative renal insufficiency, regardless of their creatinine clearance [17]. Identification of those high risk patients with preoperative creatinine clearance (≤60 ml/ min) would allow better renal protective interventions intra or postoperative. Or find an alternative way of management for those patients.

In hospital mortality has been associated with not only high preoperative serum Cr (6) but also with high postoperative serum creatinine [18]. Conlon et al showed that an increase in serum creatinine by 1.0 (mg. dl) and or new onset of renal failure was associated with mortality post cardiac surgery (19). In one study it was found that there is a distinct association between patients with elevated serum creatinin and low output syndrome and infection, and this may try to explain the high postoperative mortality in those patients. [20].

Thadhani et al reported renal tubular necrosis accounting for 85% of acute renal failure (50%) due to ischemia and 35% due to toxins [21].

Our study showed that there is a high incidence of postoperative mortality (40%) in the group of patients with impaired kidney function who require dialysis post CABG. The mechanism of acute renal failure post CABG maybe due to hypotension which may result from low cardiac output or low pressure from the cardiopulmonary bypass. Nephrotoxins and fluid overloading are also other contributing factors.

Longer cardiopulmonary bypass time was the main operative factor associated with a higher incidence of RRT in our study. The effects of cardiopulmonary by pass on a systemic inflammatory response and endothelial cell-neutrophil adhesion has been established. These changes promote leucocytes adhesions to capillary bed in the kidneys with subsequent release of cytokines and oxygen free radicals causing renal injury [22]. Other studies found that changes in the plasma inflammatory cytokines with an enhanced filtration being more likely to be responsible for tubular damage. These findings are supported by the observation that the incidence of acute renal failure post off-pump CABG is 50% less than that of on Pump CABG [23].

Boldet et al concluded that CPB time > 90 minutes was the most important risk factor for developing ARF post CABG (24). Ascione et al observed that during CPB there has been a significant increase in the creatinine clearance after CBP as a consequence of renal auto-regulation mechanism, thus increasing the glomular filtration capacity. In the mean time, there has been a clearance reduction in the following days followed by an increase of the N-acetyl glucosaminindase (NAG) activity- an important renal tubular damage marker, thus corroborating the influence of CBP use in the pathophysiology of acute renal failure postoperatively (25)

In our study the increased total bypass time in the group of patients who required post operative RRT was obvious (P = 0, 0001), our logistic regression analysis, and high odds ratio (2.3) also confirmed this correlation. So, those patients with impaired kidney function who may require prolonged bypass time, due to additional intraoperative procedures such as endarterectomy or more grafts will benefit form off pump CABG whenever possible.

Other operative factors include emergency operation, in emergency situations; the unstable preoperative state among those patients with recent myocardial infarction and cardiogenic shock are more likely to be associated with reduced renal perfusion secondary to lower cardiac output state. This will be added to the damaging effect of cardiopulmonary bypass making those patients are more vulnerable to renal damage after emergency surgery (26). Although emergency surgery was one of the factors which predispose to postoperative renal replacement therapy, however, our logistic regression analysis did not identify it as a predictor of post operative renal replacement therapy, maybe because the number of studied patients in our cohort is not enough to get statistically significant results.

It becomes clear from our study that those patients with impaired kidney function especially with creatinine clearance less than 06 ml/min are more liable to postoperative complications, and morbidity. Respiratory complications in the form of prolonged intubation, tracheostomy was significantly higher in this group of patients. These complications may also contribute to longer ICU stay, and increase total length of hospital stay.

Identifying those patients with a higher risk of developing post CABG renal replacement therapy is very important to allow more comprehensive informed consent from the family. This will also allow health care providers to estimate with some degree of accuracy the need for postoperative renal replacement therapy in those subgroups of patients.

It is important to manage those patients aggressively, in the first instance, by specific measures include adequate hydration, maintenance of adequate cardiac output, (through adequate filling and inotropic support, the use of continuous infusion of mannitol, frusemide and dopamine (27). Early institution of hemofiltration postoperatively may improve the results (28).

It will be a very important prognostic factor to identify those patients with a high risk of developing post operative permanent dialysis, and try to find an alternative option for coronary management in those patients, as those patients will not benefit more from surgery, and permanent dialysis or death will be one of the major consequences of surgery.

Summary and conclusion:

In summary, our findings demonstrate that patients with preoperatively impaired kidney function have a high incidence of mortality and represent a high risk patients. Estimation of creatinine clearance prior to surgery is a more sensitive indicator of glomerular filtration than serum creatinine. Patients with creatinine clearance less than 60 ml/min are at a higher risk of developing post operative renal replacement therapy. The predictors of postoperative renal replacement therapy, in our study included, low creatinine clearance and long cardiopulmonary bypass time.

An alternative way of coronary revascularization, whenever, possible should be looked for in those high risk group of patients as they are associated with high incidence of mortality and morbidity.

Study limitations

The sample size of the patients is relatively small to get statistically significant data. Besides we are unable to account for any other factors which may influence post operative renal replacement therapy.

References:

1. Thakar CV, Worley S, Arrigain S, Yared J-P, and Paganini EP: Influence of renal dysfunction on mortality after cardiac surgery: Modifying effect of preoperative renal function. Kidney Int67: 1112-1119, 2005.
2. Chartow GM, Lazarus JM, Christiansen CL, et al. Preoperative renal risk stratification. Circulation 1997; 95:878-884.
3. O'Conner GT, Plume SK, Olmstead EM, Coffin LH, Morton JR, Maloney CT, Nowicki ER, Tryzelaar JF, Hernandez F, Addrian L, Casey KJ, Soule CN, Martin CAS, Nugent Wc Charles worth DC, Clough R, Katz S. A regional prospective study of in–hospital mortality associated with coronary artery bypass surgery. J Am Med Assoc 1991; 266:803-809.
4. Ronser MH, Okusa MD: Acute kidney injury associated with cardiac surgery. Clin J Am Soc Nephrol I: 19-32, 2006.
5. Nakayama Y, Sakata R, Ura M, Itoh T. Long term results of coronary artery bypass grafting in patients with renal insufficiency. Ann Thorac surg 2003; 75:496-500.
6. Weerasinghe A, Hornick P, Smith P, Taylor K, Ranatunga C:

Coronary artery bypass grafting in non-dialysis dependant mild to moderate renal dysfunction. J Thorac Cardiovascular Surg 2001:121: 1083-9.
7. Wang F, Dupuis JY, Nathan H, Williams K: An analysis of the association between preoperative renal dysfunction and outcome in cardiac surgery: renal function. Chest 2003; 124: 1852-62.
8. National Kidney Foundation. K/DOQI clinical practice guidelines for chronic kidney disease: evaluation, classification and stratification. Am j Kidney Dis 2002; 39 (suppl.2):S1-S246.
9. Cokcroft DW, Gault MH: Prediction of creatinine clearance from serum creatinine. Nephron 1976; 16:31-41.
10. Doddakula Kishore, Al-Sarraf Nael, Gately Kathy, Huges Anne, Tolan Michael, Young Vincent, McGovern Eillish. Predictors of acute renal failure requiring renal replacement therapy post cardiac surgery in patients with preoperatively normal renal function. Interactive Cardiovascular surgery6 (2007) 314-318.
11. Grayson AD, Khater M, Jackson M, Fox MA, Valvular heart operation is an independent risk factor for acute renal failure. Ann Thorac Surg 2003;75:859-864.
12. Bakris GI, Williiams M, Dworkin L, et al, for the national Kidney foundation Hypertension and diabetes executive committees working group. Persevering renal function in adults with hypertension and diabetes: a consensus approach. Am J Kidney Dis. 2000; 36-646-661.
13. Andresson L-G, Ekroth R, Batteby LE, Hallhangen S, Wasslen O. Acute renal failure after coronary surgery- A study of incidence and risk factors in 2009 consecutive patients. Thaorac Cardiovascu Surg 1993; 41:237-241.
14. Szczech LA, Best PJ, Crowely E, Brooks MM, Berger PB, Bittner V, Gresh BJ, Jones R, Califf RM, Ting HH, Whitlow PJ, Detre KM, Holmes D. Byapss angioplasty revascularization Investigation (BARI) Investigators. Outcomes of patients with chronic renal insufficiency in the bypass angioplasty revascularization investigation. Circulation 2002; 105:2253-2258.
15. Swedko PJ, Clark HD, Paramsothy K, Akbari A: Serum creatinine is an inadequate screening for renal failure in elderly patients. Arch Intern Med 2003; 163:356-60
16. The BARI investigators, Seven –year outcome in the bypass angioplasty revascularization investigation (BARI) by treatment and diabetic status. J Am Col Cardiol 2000; 35:1122-1129.
17. Wijeysundera DN, Karkouti K, Beattie S, Rao V, Ivanov J. Anesthiology 2006;104:65-72.
18. Zanardo G, Michielon P, Paccagnella A, Rosi P, Calo M , Salandin V, Daros A, Michieletto F, Simini G, Acute renal failure in the patient undergoing cardiac operation-Prevalence , Mortality-rate, and main risk factors. J Thorac Cardiovac Sur.1994; 107:1489-1495.
19. Conlon PJ, Stafford-Smith M, White WD, NewmanMF, King S, Winn MP, Landolfo K. Acute renal failure following cardiac surgery. Nephrol Dia Transpl. 1999; 14:1158-1162.
20. Brown JR, Cochran RP, Dacey LJ, Ross C S, Kunzelman KS, Dunton RF, Braxton JH, Charlesworth DC, Clough RA, Helm RE, Leavitt BJ, Mackenzie TA, O'Connor GT, and for the Northern New England Cardiovascular Disease Study Group. Preoperative increase in serum creatinine are predictive of increases 90-day Mortality after coronary artery bypass graft surgery. Circulation 2006; 114; I-409-I-413.
21. Thandhani R, Pascual M, Bonventre JV, Medical progress-Acute renal failure. N Engl J Med 1996; 334:1448-1460.
22. Verrier ED, Boyel EM. Endothelial cell injury in cardiovascular surgery. Ann Thorac Surg 1996; 62:915-922.
23. Stallwood MI, Grayson AD, Mills K, Scawn ND. Acute renal failure in coronary artery bypass surgery: independent effect of cardiopulmonary bypass. Ann Thorac Surg 2004; 77:968-972.
24. Boldt J, Brenner T, Lehmann A, Suttner SW, Kumle B, Isgro F. Is kidney function altered by duration of cardiopulmonary bypass? Ann Thorac Surg. 2003; 75(3) 906-12.
25. Ascione R, Lloyld CT, Underwood MJ, Gomes WJ, Angelini GD. On pump versus off-pump coronary revascularization: evaluation of renal function. Ann Thoarc Surg. 1999; 68(2):493-8.
26. Mehta RH, Grab GD, O'Brien SM, Bridges CR, Gammie JS, Haan CK, Ferguson T B, Petrson ED. And for the society for thoracic surgeons database invstigators. Bedside tool for predicating the risk of postoperative Dialysis in patients undergoing Cardiac surgery. Circulation 2006: 114; 2208-2216.
27. Sirivella S, Gielchinsky I, Parsonnate V. Mannitol, Frusmide and dopamine infusion in post-operative renal failure complicating cardiac surgery. Ann Thorac Surg 2000; 69:501-506.
28. Elahi MM, Lim MY, Joseph RN, Dhammapuneni RRV, Spyt TJ. Early hemofiltration improves survival in post-cardiotomy patients with acute renal failure. Eur J. Cardiothorac Surg 2004; 26:1027-1031.

Mitral Valve Replacement with preservation of whole subvalvular apparatus:Value and Early effects on Left ventricular contractile function.

Talal Ahmed RedaMD,
Sally Mahdy El-Roby MS*,
Medhat Hashem, FRCA, MD,**
Teah Chuan Yeang MBBS ***

Background: **The integrity of both anterior and posterior leaflets as well as the chordopapillary subvalvular apparatus contributes equally to left ventricular function preservation. This study compares short-term (6 months) postoperative follow-up results of mitral valve replacement while preserving both leaflets attachment to the left ventricular wall through the subvalvular apparatus, versus preservation of the posterior leaflet only.**

Methods: **This prospective randomized controlled study was carried out after obtaining informed consent and ethical committee approval. Fifty patients scheduled for MVR were randomized to two equal groups. Group A patients were submitted for mitral valve replacement with preservation of both leaflets' chordal attachments to the left ventricular free wall being anchored to their respective anatomical location along the mitral annulus via teflon-pledgeted sutures. In group B patients', the diseased anterior leaflet with its chordal attachments to both papillary muscles were resected leaving only the posterior leaflet attachments (control group). Perioperative patient evaluation was done on patient visits for clinical examination as well as transthoracic echocardiography.**

Results: **There was no statistical difference regarding cross-clamping time and total operative time in patients in both groups one patient died in group B due to multiple organ dysfunction syndrome and was excluded. In group A, 2 patients had transient arrhythmias (one transient AF, and one transient ventricular tachyarrhythmias). All were controlled by correcting the blood electrolytes without the need for antiarrhythmic drugs. Three patients in group B had low-cardiac output needing prolonged inotropic support and one patient required re-exploration for haemostasis Patients in group A showed a significant improvement in NYHA dyspnea classification and postoperative left ventricular dimensions and ejection fraction than patients in group B.**

Conclusion: In our study group, complete bileaflet chordal preservation lead to a good benefit in patients having mitral valve disease of rheumatic origin. Compared to posterior leaflet chordae only, bileaflet chordal preservation was associated with better short-term improvement in Left Ventricular Contractile Function.

Keywords: RHD: **Rheumatic Heart Disease, MVD: mitral valve disease,** ***MVR:*** **Mitral Valve Replacement, Chordal preservation**

Department of Cardiothoracic Surgery, Faculty of Medicine, Cairo University, Egypt.
Department of surgery, Cardiothoracic unit, Hospital University Kebangsaaan Malaysia (National University of Malaysia), Kuala Lumpur, Malaysia.
*Department of Pediatric Cardiology, National Heart Institute, Cairo, Egypt.
** Department of Anesthesia , Faculty of Medicine, Cairo University, Egypt.
*** Department of Anesthesia, Hospital University Kebangsaaan Malaysia (National University of Malaysia), Kuala Lumpur, Malaysia.
Email : jegyptscts@gamil.com
Codex : 04/110/0906

For many years, the standard technique for mitral valve replacement included excision of both valve leaflets and their attached chordae tendineae. However, the physiologic importance of retaining the geometric integrity of the mitral subvalvular apparatus has been recognized in the early reports of mitral valve surgery by Lillehei et al. [1].

Following their recommendations to respect this anatomical fact, an increased emphasis became placed on retention of all attachments of the mitral subvalvular apparatus during valve replacement [2-5]. Their proof was the practically-confirmed dependence of left ventricular performance on the presence of the mitral valve and subvalvular apparatus, and of the improvement in left ventricular function associated with mitral valvuloplasty as opposed to that associated with standard mitral valve replacement [2-8].

Despite all that, there are still many surgeons who retain only the posterior leaflet and excise all or part of the anterior leaflet claiming that this will prevent both prosthetic valvular dysfunction and left ventricular outflow tract obstruction (LVOTO) [1,3,4,9]. The majority of the still-investigating surgeons fear the possibility of prosthetic valve entrapment by the retained subvalvular apparatus, creation of left ventricular outflow tract obstruction (LVOTO), and the concern of implanting a smaller- sized prosthesis [1-5].

Aim of the work:

This study aimed to compare the value and short-term (6 months) postoperative follow-up results of surgical technique of mitral valve replacement while preserving both leaflets attachment to the left ventricular free wall through the subvalvular apparatus, versus retaining the posterior leaflet only.

Methods:

This prospective randomized controlled study was carried out after obtaining the approval of the local ethical committees. The study population encompassed 50 patients scheduled for elective mitral valve replacement;

Patients diagnosed to have rheumatic heart disease (RHD) complicated by advanced mitral valve disease (combined stenosis and regurge) necessitating mitral valve replacement for the following conditions:

1- Valve heavily calcified or considered unsuitable for valve repair due to extensive scarring.
2- Shortening with severe subvalvular fusion.
3- Previous closed mitral valvotomy or mitral valve repair; subvalvular tissue considered worthy of preservation when calcified mitral leaflets with annular extension were successfully dealt with by squeezing or milking out the calcified debris from the annulus or segmental excision of the calcified segment with the involved chordae tendinae.

Exclusion criteria:

Patients with the following pathologies were excluded from the study;

- Mitral valve pathology associated with heavy subvalvular or annular calcification;
- Associated significant pathology of another cardiac valve eg: aortic valve disease.
- Associated coronary artery disease, and active rheumatic fever.
- Isolated mitral regurgitation amenable for reconstructive procedures.

Patients were prospectively randomized into two equal groups (A and B) using closed envelops method.

Group A patients' had mitral valve replacement with preservation of both leaflets chordal attachments to the left ventricular free wall.

Group B patients' had mitral valve replacement with resection of the anterior leaflet tissue and chordae till their attachment to their respective papillary muscles leaving only the posterior leaflet attachments.

Surgical Technique:

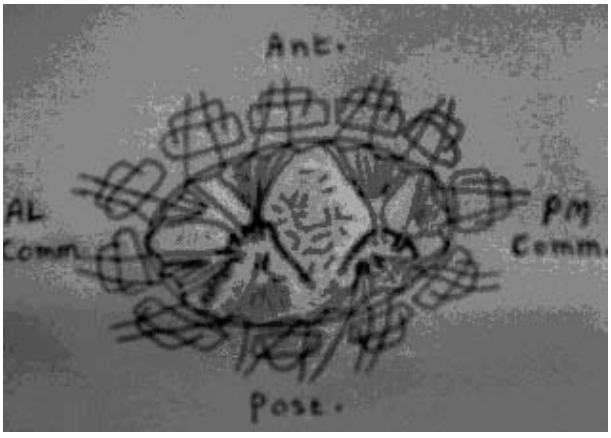
In all patients, median sternotomy was the approach used for cardiac exposure. Moderate hypothermia (28–29°C) was reached during systemic cooling by cardiopulmonary bypass which was established by routine aortic and bicaval cannulation. Antegrade cold blood-enriched crystalloid cardioplegia and topical hypothermia were used for intraoperative myocardial preservation. After dissecting the inter-atrial groove of Waterson, the mitral valve was reached via a left atriotomy incision fashioned behind the groove. In all patients, mitral valve replacement was done using a St Jude mechanical prostheses (St Jude Medical, Inc, St Paul, MN) according to the size of each patient's individual annulus. An everting mattress sutures (ie, pledgets on the atrial side) were used in all cases. Free disc movement was checked for inteXrference of movement and or LVOTO (left ventricular outflow obstruction) after tying all the preserved chordae tendinae.

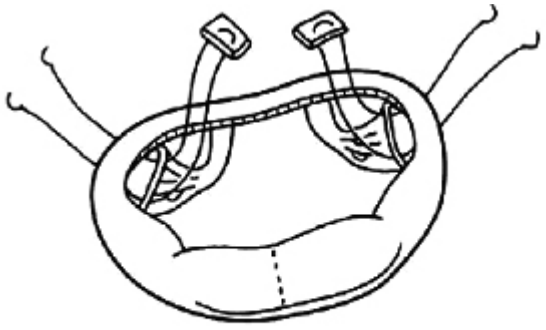
Techniques of Chordal Preservation:

The valve was analyzed intraoperatively in a systematic manner to obtain a detailed imagination of its topography and the site of insertion of the different groups of chordae tendinae. For preserving the subvalvular apparatus, we used the following technique.

The anterior mitral leaflet was incised in the center from edge to base, before separating it about 3-4 mms from the annulus. The major part of the anterior and posterior cuspal (leaflet) tissue was excised, leaving 5-10 mms rim of leaflet free edge attached to the primary first order or marginal chordae tendinae. The anterior and posterior mitral commissures were incised. Excessive cuspal tissue was sliced to make it thin. The fibrous and calcific nodules were excised. The anterior paramedial chorda, or anterior and posterior paramedical chordae tendinae, or paracommissural chordae tendinae were excised along with splitting and partial shaving of the papillary muscle tip on an individualized basis. The separated anterior and posterior segments of the anterior mitral leaflet were shifted and reattached to its presumed original site of detachment by passing itwith the valve stitches respecting its anatomical geometry. In order to accommodate a large size prosthesis, each mitral leaflet was incised from edge to base at two or three additional points between its scallops supported by "cleft chordae." If the posterior leaflet was excessively redundant or the chordae tendinae were elongated, the leaflet was then reefed or imbricated by pledgeted stitches that were passed through it into the mitral annulus. The valve was seated and inserted in its place using 2–0 Ethibond everting sutures (Ethicon, Cincinatti, OH, USA) over polytetrafluoroethylene pledgets. While securing the prosthetic valve on the annulus, the posterior valve sutures were tied first.

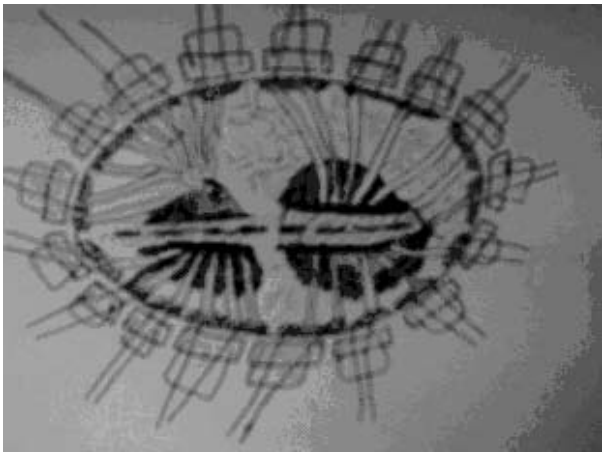
Technique of posterior chordal preservation only: In this technique, the anterior mitral leaflet with its attached chordae tendinae was completely excised with a small part of the apex of its corresponding papillary muscles. The posterior leaflet with the chordopapillary support

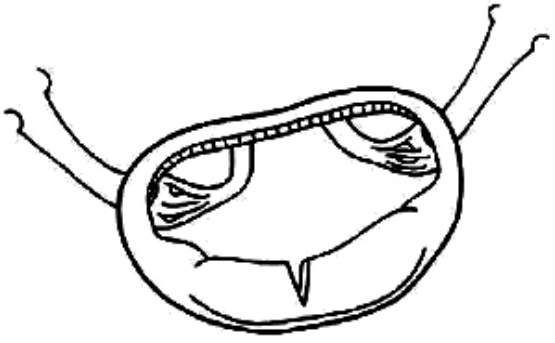




The anterior and posterior parts of the anterior leaflet are re-attached to the mitral ring near the anterolateral and posteromedial commissures

Figure (1): The anterior and posterior leaflets of the mitral valve cut into many tissue islands and teflonized everting stitches were passed through them and into the annulus. Ant: anterior Post: posterior AL comm: anterolateral commissure PM comm: Posteromedial commissure.





These sutures are left untied and are subsequently taken through the prosthetic valve ring along with the other valve sutures

Figure (2) : longitudinal splitting of the two papillary muscles into two 2 halves (transverse dashed line) according to their leaflet belonging to facilitate pulling up and tying each stitch according to its topographic location along the annulus.

mechanism were retained. The posterior mitral leaflet was incised at two or three points in between scallops from edge to base creating islands of leaflet tissue which were reefed or imprecated within the valvular stitches taken in the annulus.

Patient follow-up evaluation was done by clinical examination and transthoracic (TTE) echocardiography using Two-dimensional B-mode, M-mode, and colour-flow Doppler from the parasternal long and short axes as well as from the apical two and four-chamber views. The echocardiographic data have been measured according to the American Society of Echocardiography guidelines. The onset of the Q wave in the ECG defines the onset of end-diastole; the peak downward motion of the interventricular septum is indicative of end-systole. The following echocardiographic data were measured; Left Atrium diameter; Left Ventricular End-Diastolic Diameter (LVEDD) Left Ventricular End-systolic Diameter (LVESD) and Left ventricular ejection fraction (EF). Data were collected in both groups over the first 6 postoperative months.

Intention to treat analysis was performed. Normally distributed numerical data was described in terms of mean and standard deviation and compared using unpaired t-test and one way ANOVA. Non-normally distributed data were compared using Mann-Whitney test. Categorical data were described in terms of number and percentage and compared using Chi square and Fischer Exact tests when appropriate. Data was deemed significant if $p<0.05$.

Results:

There were no significant difference between patients in both groups regarding demographic data, preoperative morbidity and operative durations (Tables 1 and 2)

	Group A (n=25)	Group B (n=24)	p value
Age	54.2 + 6.7	49.6 + 5.9	0.32
Male/Female	20/5	21/3	0.74
Body Mass Index (kg/m2)	24.3 + 2.1	25.1 + 3.2	0.41
Smokers/Non-smokers	18/7	16/8	0.68
Hypertension	7 (28%)	9 (37.5%)	0.50
Hyperlipidaemia	13 (52%)	17 (70.8%)	0.17
Diabetes Mellitus	5 (20%)	7 (29.1)	0.45
COPD	4 (16%)	3 (12.5)	0.72
Renal dysfunction	3 (12%)	5 (20.8%)	0.40
Peripheral vascular disease	5 (20)	7 (29.1%)	0.23
Total Operative time (minutes)	185 ± 13.5	196 ± 11.4	0.39
Cross Clamp Time (minutes)	80 ± 2.5	73 ± 3.5	0.33

Table 1: Preoperative data of patients in both groups.
Values are number (%) except age, body mass index and operative durations: mean + SD. *denotes a significant difference (p<0.05).

NYHA Class			
II-III	13	12	0.88
IV	12	12	0.88
Echocardiographic data			
LA diameter (mm)	57 + 5.4	56.5+4.5	0.46
LVEDD(mm)	62.8+6.5	65.5+8.3	0.32
LVESD(mm)	48.6+8.5	53.3+4.5	0.21
LVEF (%)	44 + 5.6	42 + 4.5	0.28
PASP (mmHg)	50 + 0.7	52 + 0.2	0.31

Table (2): Preoperative cardiological data of patients in both groups.
NYHA: New York Heart Association, LA: Left Atrium, LVEDD: Left Ventricular End-Diastolic Diameter, LVESD: Left Ventricular End-systolic Diameter, LVEF: Left ventricular ejection fraction, PASP: Pulmonary artery systolic pressure.
NYHA data are number (%) and echocardiographic data are mean + standard deviation *denotes a significant difference (p<0.05).

One patient died in group B secondary to muliorgan dysfunction dyndrome (MODS) and was excluded from the results. Postoperative complications occurred in 6 patients (12.24 %). In group B, 3 patients had low-cardiac output needing prolonged inotropic support and one patient was re-explored for haemostasis. In group A, one patient had transient atrial fibrillation and another patient had transient ventricular tachycardia. Both were controlled by correcting the blood electrolytes without the use of antiarrhythmic drugs (Table 3).

	Group A (n=25)	Group B (n=24)	p value
Total morbidity	2 (8%)	4 (16.6%)	0.35
Low Cardiac Output	0	3 (12.5%)	0.07
Re-exploration for bleeding	0	1 (4.1%)	0.31
Transient AF	1 (4%)	0	0.31
Transient VT	1 (4%)	0	0.31

Table 3: Postoperative morbidity in patients in both groups.
AF: Atrial Fibrillation, VT: Ventricular Tachycardia
Values are number (%), *denotes a significant difference (p<0.05).

Postoperative follow-up Data:

During clinical visits, all patients expressed functional improvement, with increased ability to tolerate physical effort as evidenced by their step-up in the NYHA clinical classification. 18 patients in Group A were in NYHA class I as compared to 9 in group B. The difference was statistically significant. Also, patients with NYHA class II or more were significantly more in group B than in group A. Postoperative echocardiography in group A revealed a mean Left Ventricular End-Systolic Dimension of 44 ± 1.2 mms; End-Diastolic Dimensions of 51 ± 1.4 and LVEF% of 54 ± 11.5 % with more clinical improvement reflecting better clinical improvement and better statistical significance than group B patients which gave the following dimensions of 46 ± 3.5; 58 ± 3.5; and 48 ± 3.4 % respectively) (Table: 4).

	Group A (n=25)	Group B (n=24)	p value
NYHA Class			
I	18 (72%)	9 (37.5%)	0.04
II or more	7 (28%)	15 (62.5%)	0.04
Echocardiographic Data			
LA diameter (mm)	51 ± 3	52 ± 4	0.21
LVEDD(mm)	54 ± 1.4	59± 1.5	0.04
LVESD(mm)	43 ± 1.2	49 ± 3.5	0.03
LVEF (%)	54 ± 11.5	48 ± 3.4	0.04
PASP (mmHg)	39 ± 0.5	46 ± 1.7	0.04

Table (4): Postoperative cardiological data of patients in both groups.
NYHA: New York Heart Association, LA: Left Atrium, LVEDD: Left Ventricular End-Diastolic Diameter, LVESD: Left Ventricular End-systolic Diameter, LVEF: Left ventricular ejection fraction, PASP: Pulmonary artery systolic pressure.
NYHA data are number (%) and echocardiographic data are mean + standard deviation *denotes a significant difference (p<0.05).

Discussion:

The normal function of the mitral valve requires the coordinated interplay of all its components namely the annulus, leaflets, chordae tendinae, and papillary muscles(10-12). The saddle-shaped annulus, by virtue of its attachment to the fibrous skeleton of the heart, functions mainly as a fulcrum for the mitral leaflets and to decrease the size of the mitral orifice (by 10% to 20%) during late diastole and systole (13-15) .

Careful understanding as well as evaluation of the different mechanisms inducing mitral valve pathology

is mandatory. Recent reports state that mitral valve dysfunction occurs as a result of small changes in the spatial relations of the different anatomic components of the mitral valve (16). Different studies highlighted the impact of even small changes in annular shape and, more important, the role of the papillary muscles to contribute to the distortion of leaflet coaptation [17],[18].

The mitral valve has two papillary muscles, the anterolateral which has dual blood supply from branches of the circumflex and left anterior descending arteries. The posteromedial PM which, in contrast, is supplied by a single artery arising from either the right coronary or from the terminal circumflex artery and is therefore more prone to infarction (14). Clinical studies have confirmed that MR is more likely to occur after postero-inferior than antero-lateral myocardial infarction [19],[20].

Primary chordae tendinae attach to the free edge of the leaflets and prevent prolapse during systole, whereas the "thicker" secondary chordae attach to the belly of the leaflet [19]. Surgical options for MV repair after intraoperative inspection include chordal or PM shortening, chordal transposition, or neo-chordae construction with Gore-Tex sutures, ring annuloplasty alone or combined with other techniques to embrace the papillary muscles together (pledgeted sutures or slings) [20].

Mitral valve replacement with concomitant preservation of both anterior and posterior leaflets and all chordae tendinea was introduced by David and colleagues [21], who demonstrated improved postoperative left ventricular performance and ejection fraction with increased values both during rest and exercise. Satisfactory results of bileaflet preservation inspired several surgical centers and so they begun to perform techniques to preserve both leaflets during the MVR. Different modifications have been presented [22-26].

The major goal during preserving the subvalvular apparatus is to prevent LVOT obstruction, by preventing the preserved tissue from interfering with prosthetic valve function and at the same time being able to implant an adequate size of valve for the patient [25-28].

LVOT obstruction has been the main feared complication of bileaflet preserving MVR that has been reported recently [23-27]. The remaining redundant chordae tendinea, the postoperative reduction of left ventricular size (cavity) that occurs after surgery; in addition to the systolic anterior motion of the native anterior mitral leaflet after prosthetic mitral valve insertion, can all lead to LVOT obstruction [27],[28].

Several modifications were recently proposed aiming to eliminate LVOT obstruction after bileaflet preserving MVR operations. Feike et al [22]; Wu et al [24] have incised the entire anterior leaflet from annulus and re-affixed close to posterior leaflet and posterior wall. The function of papillary muscle may be preserved but anterior regional wall motion may not always improve in these techniques because the movement of posterior wall may be strengthened excessively by the preserved chordae tendineae [25].

However, many surgeons reported that the preservation techniques using the in situ position of the chordo-papillary attachments may maintain an adequate global and regional cardiac function after MVR (24),(25). Our results, that coincides with other investigators (22-27), confirms this statement. According to our statistical data obtained during the early 6-months of postoperative follow-up, the mean values for LV dimensions (LVEDD, LVESD, as well as their equivalent LVEF%), did show more improvement with the total preservation group (A), compared to conserving the posterior leaflet only (group B). In our opinion, the cornerstone step is to cut the leaflet tissue into separate parts, and then trim its tissues into small islands that retain their attachment to multiple primary or secondary chordae. With this step done carefully, we, as well as other surgeons [22-28], believe that this "debulking" of the leaflet tissue largely protects against any future occurrence of LVOT.

Another crucial step has been the longitudinal splitting of both papillary muscles till its roots from the LV free wall. This step is claimed to maximize the surgical gain by decreasing the tension posed on each chord with the corresponding annular site of attachment, as well as distribute the chordae away from the LVOT direction, in addition to allowing the insertion of a prosthetic valve of an adequate size. Other surgeons also reported the previous finding like [22-25].

Moreover, in the modified bileaflet preserving technique, allowed in situ position of the preserved anterior leaflet, with the remaining tissue anchored between the mitral annulus and the prosthetic valve instead of cutting it loose in the left ventricular cavity.

This also diminishes the risk of LVOT obstruction, compared to other techniques in which just plication of the major bulk of the anterior leaflet tissue was performed [26]. Lastly, the obvious improvement in the patients' quality of life (stepped-up NYHA clinical class), the absent mortality, and the internationally-acceptable lower rate of morbidity complications being all non-fatal and completely controllable, adds more solidity to out data proofs.

Conclusion:

In our patient group, complete bileaflet chordal preservation was possible and was of good benefit in patients having mitral valve disease of rheumatic origin. Compared to posterior leaflet chordae only, bileaflet chordal preservation was associated with better short-term improvement in Left Ventricular Contractile Function.

References :

1. Lillehei CW, Levy MJ, Bonnabeau RC Jr. Mitral valve replacement with preservation of papillary muscles and chordae tendineae. J Thorac Cardiovasc Surg 1964;47:533–43.
2. Spence PA, Peniston CM, David TE, et al. Toward a better understanding of the etiology of left ventricular dysfunction after mitral valve replacement: an experimental study with possible clinical implications. Ann Thorac Surg 1986;41: 363–71.
3. David TE, Burns RJ, Bacchus CM, Druck MN. Mitral valve replacement for mitral regurgitation with and without preservation of chordae tendineae. J Thorac Cardiovasc Surg 1984;88:718–25.
4. Cabrol CE. Comment on [3]. J Thorac Cardiovasc Surg 1984;88:724.
5. Gams E, Hagl S, Schad H, Heimisch W, Mendler N, Sebening F. Importance of the mitral apparatus for left ventricular function: an experimental approach. Eur J Cardiothorac Surg 1992;6(suppl 1):S17–24.
6. Yun KL, Rayhill SC, Niczyporuk MA, et al. Mitral valve replacement in dilated canine hearts with chronic mitral regurgitation. Importance of the mitral subvalvular apparatus. Circulation 1991;84(Suppl 3):112–24.
7. Goldman ME, Mora F, Guarino T, Fuster V, Mindich BP. Mitral valvuloplasty is superior to valve replacement for preservation of left ventricular function: an intraoperative two-dimensional echocardiographic study. J Am Coll Cardiol 1987;10:568–75.
8. Bonchek LI, Olinger GN, Siegel R, Tresch DD, Keelan MH Jr. Left ventricular performance after mitral reconstruction for mitral regurgitation. J Thorac Cardiovasc Surg 1984;88:122–7.
9. Come PC, Riley MF, Weintraub RM, et al. Dynamic left ventricular outflow tract obstruction when the anterior leaflet is retained at prosthetic mitral valve replacement. Ann Thorac Surg 1987;43:561–3.
10. Miller DC. The three-dimensional geometry of the mitral valve: implications for surgical therapy. Proceedings of the 84th Annual Meeting of the American Association for Thoracic Surgery, Toronto, Ontario, 2004.
11. Condado JA, Vélez-Gimón. Catheter-based approach to mitral regurgitation. J Intervent Cardiol 2003;16:523–34.
12. Kodavatiganti R. Intraoperative assessment of the mitral valve by transoesophageal echocardiography: an overview. Ann Card Anaest 2002;5:27–34.
13. Levine RA. Dynamic mitral regurgitation: more than meets the eye. N Engl J Med 2004;351:1681– 4.
14. Piérard LA, Lancellotti P. The role of ischemic mitral regurgitation in the pathogenesis of acute pulmonary edema. N Engl J Med 2004;351:1627–34.
15. Otsuji Y, Kumanohoso T, Yoshifuku S, et al. Isolated annular dilation does not usually cause important functional mitral regurgitation: comparison between patients with lone atrial fibrillation and those with idiopathic or ischemic cardiomyopathy. J Am Coll Cardiol 2002;39:1651– 6.
16. Hvass U, Tapia M, Baron F, Pouzet B, Shafy A. Papillary Muscle Sling: A New Functional Approach to Mitral Repair in Patients With Ischemic Left Ventricular Dysfunction and Functional Mitral Regurgitation. Ann Thorac Surg 2003;75:809 –11.
17. Fasol R, Lakew F, Wetter S. Mitral repair in patients with a ruptured papillary muscle. Am Heart J 2000;139:549–54. G
18. Gorman JH, Gorman RC, Jackson BM, et al. Distortions of the mitral valve in acute ischemic mitral regurgitation. Ann Thorac Surg 1997;64:1026–31.
19. Kumanohoso T, Otsuji Y, Yoshifuku S, et al. Mechanism of higher incidence of ischemic mitral regurgitation in patients with inferior myocardial infarction: quantitative analysis of left ventricular and mitral valve geometry in 103 patients with prior myocardial infarction. J Thorac Cardiovasc Surg 2003;125:135– 43.
20. Iung B. Management of ischaemic mitral regurgitation.Heart 2003;89:459–64.
21. David TE, Burns RJ, Bacchus CM, Druck MN, Weisel RD. Mitral valve replacement for mitral regurgitation with and without preservation of chordae tendineae. J Thorac Cardiovasc Surg 1986;90:432–436.
22. Feikes HL, Daugharthy JB, Perry JE, Bell JH, Hieb RE, Johnson GH. Preservation of all chordae tendinea and papillary muscle during mitral valve replacement with tilting disk valve. J Card Surg 1990;5:81–88.
23. Sintek CF, Pfeffer TA, Kochamba GS, Khonsari S. Mitral valve replacement: technique to preserve the subvalvular apparatus. Ann Thorac Surg 1995;59:1027–1029.
24. Wu ZK, Sun PW, Zhang X, Zhong FT, Tong CW, Lu K. Superiority of mitral valve replacement with preservation of subvalvular structure to conventional replacement in severe rheumatic mitral valve disease: a modified technique and results of 1 year follow-up. J Heart Valve Dis 2000;9:616–

622.
25. Natsuaki M, Itoh T, Tomita S, Furukawa K, Yoshiaki M, Suda H, Ohteki H. Importance of preserving the mitral subvalvular apparatus in mitral valve replacement. Ann Thorac Surg 1996;61:585–590.
26. Vander Salm TJ, Linda AP, Mauser JF. Mitral valve replacement with complete retention of native leaflets. Ann Thorac Surg 1995;59:52–55.
27. Come PC, Riley MF, Weintraub RM, Wei JY, Markis JE, Lorell BH, Grossman W. Dynamic left ventricular obstruction when the anterior E. Kuralay et al. / European Journal of Cardio-thoracic Surgery 22 (2002) 630–632.
28. Waggoner Ad, Perez JE, Barzilai B, Rosenbloom M, Eaton MH, Cox JL. Left ventricular outflow obstruction resulting from insertion of mitral prostheses leaving the native leaflets intact: adverse clinical outcome in seven patients. Am Heart J 1991;122:483–488.

Posterior Mitral Leaflet Augmentation With Autologous Pericar dium

Tarek S. Abdallah, MD*
Dina Soliman, MD**

Background: **Mitral valve repair is the procedure of choice in management of Mitral valve dysfunction of all etiologies. However, patients with rheumatic changes, who have fibrotic and retracted leaflets, pose significant operative challenges due to technical problems.**

Methods: **Fifteen patients underwent Mitral valve repair for rheumatic severe MR due to shortened, retracted posterior leaflet from Jan2007 to Jun2008. All patients presented with severe MR .The mean age of patients was 19.4±5.39 years. All patients were females. Those patients had a mean New York Heart Association (NYHA) class of 3.2 ± 0.4.**

Results: **Mitral valve repair by Posterior Mitral leaflet augmentation with a gluteraldehyde treated Autologous pericardial patch and complete annuloplasty ring were done in all patients for severe MR. The mean ring size was 30.53±0.91mm for Carpentier rings (Edwards Lifesciences LLC, Irvine, CA). The mean aortic clamp time and cardiopulmonary bypass time were 71.26±12.97 minutes and 91±15.49 minute, respectively. Additional surgical procedure included tricuspid annuloplasty were done in 3 patient, by Devega repair and AVR was done in 1 patient.**

Conclusion: **Further information and follow-up is needed to determine the extended long-term durability of patch augmentation of the posterior leaflet. However, our results demonstrate excellent utility of repair and this technique should be considered for patients with severe Mitral Regurge due to retracted, shortened posterior leaflet.**

Mitral valve dysfunction is a well-known complication in long-term rheumatic heart disease. The leaflet changes include fibrosis, thickening, and shortening with or without calcification. Consequently, these changes may result in short, "shrunken" anterior & posterior leaflets. These short leaflets do not provide adequate coaptation and results in Mitral regurgitation and the subsequent development of heart failure.

Historically, Mitral valve replacement has been used to surgically manage these patients. Today the benefits of Mitral valve (MV) reconstruction are well established.

These benefits include low perioperative mortality, preservation of left ventricular function, avoidance of long-term anticoagulation therapy, decreased thromboembolic complications, a low risk of native valve endocarditis, excellent long-term freedom from reoperation, and improved survival as compared to patients undergoing valvular replacement. However, patients with rheumatic changes, who have fibrotic and retracted leaflets, pose significant operative

Address reprint request to :
Dr Tarek S Abdallah
* Department of Cardio - thoracic surgery, Cairo University
** Department of Anesthesiology , Cairo University
Email : jegyptscts@gamil.com
Codex : 04/111/0906

challenges due to technical problems and concerns of long-term results of repair.(1)

All techniques of Mitral valve repair require a 3D understanding of the dysfunction, a vision of the goal of restoring the surface of coaptation, and an experience that will help to answer strategically the different questions that may arise during the repair. The contribution of echocardiography is an absolute must requiring the need for a team approach with an echo cardiographer. (2)

MV surgical repair is a class I indication for intraoperative Transoesophageal echocardiography (TEE) according to ACC/AHA/ASE Guidelines. (3)

In this study we describe our experience in Mitral valve repair by posterior leaflet augmentation using Autologous pericardial patch, in cases of severe Rheumatic Mitral regurge due to shortened, retracted posterior leaflet.

Methods

Fifteen patients underwent Mitral valve repair for rheumatic severe Mitral regurge (MR) due to shortened, retracted posterior leaflet from Jan 2007 to Jun 2008. All patients presented with severe MR. The modes of dysfunction were pure MR in 11 patients and combined MR & MS in 4 patients. The mean age of patients was 19.4±5.39 years. All patients were females. Those patients had a mean New York Heart Association (NYHA) class of 3.2 ± 0.4. Demographic data of the patients are shown in table (1)

Number	15 patients
Age range	13-35y
(mean)	(19.4±5.39)y
Male	-
Female	15 patients
NYHA mean	3.2 ± 0.4
Degree of MR	+4

Table (1): Demographic data of the patients:

Surgical Technique

After induction of general anesthesia, a transoesophageal echo probe was placed. using a Vivid3 machine (General Electric). A detailed Echocardiographic assessment was performed following a standardized protocol to examine the dysfunction. The main goal of prebypass TEE examination was generally to give the surgeon important information regarding etiology, lesions and dysfunction of the Mitral valve apparatus that would influence the strategy necessary to successfully reconstruct the valve rather than quantifying the degree of MR.

The approach to prebypass evaluation of MV apparatus included:

- 2D Exam: in the following views
  - 5ch view, 4ch view.
  - Commissural, 2ch, long axis views.
  - Transgastric basal short axis, midpapillary short axis & long axis views.

The 2D Exam was guided with the systematic Mitral valve exam defined by Lambert and colleagues, which include assessment of leaflet appearance and mobility, localizing the dysfunction (P1, P2, P3), the measurement of Mitral valve annulus (ME long axis view and commissural view, at end systole), subvalvular apparatus (TG two chamber view) and left ventricular shape, dimensions and systolic function. (4)

- Color-flow Doppler (CFD) of MV:
  -Jet area, vena contracta, PISA.
- Spectral Doppler:
- (CWD) of MV regurgitant jet.

The color–flow Doppler & spectral Doppler helped in the evaluation of degree and direction of Mitral regurge. (5)

The nomenclature used all through the procedure as agreed with the surgical & anesthesia team was the Carpentier nomenclature. Carpentier defined the three scallops of the posterior leaflet as (P1, P2 and P3), where P1 is the closest to the left atrial appendage. He also defined three corresponding areas of anterior leaflet (A1, A2 and A3). It was adopted by ASE/SCA guidelines. (6)

The operative procedure was done through median sternotomy. An Autologous piece of pericardium was harvested immediately after median sternotomy and bathed in 3% gluteraldehyde for seven minutes and then rinsed with saline solution in a separate bowl for an additional 30 minutes. Following patch harvesting, patients were placed on standard cardiopulmonary bypass with bicaval cannulation. Standard blood cardioplegic arrest was initiated with antegrade flow in all patients.

Exposure through Lt. Atriotomy was improved by the following steps:

- Complete dissection of the SVC from the surrounding pericardial reflection using both sharp and blunt dissection
- Opening of the oblique sinus by separation of the Lt. Atrium from the reflection of the pericardium to the left of the IVC.
- Dissection of the interatrial groove, starting above the Rt. superior Pulmonary vein passing towards the oblique sinus to the Lt. of the IVC using a knife and sissors to allow the Lt. Atriotomy to be close to the Mitral annulus.

A standard right-sided left atriotomy was made parallel to the interatrial groove and extended and wrapped superiorly and inferiorly in the direction of the right superior and inferior pulmonary veins. Self-retaining retractors were placed. The Mitral valve was brought closer into the operative field by placement of 3 ethibond stitches (2/0). One at the middle of the posterior annulus, the second at the antrolateral commisure and, the third at the postromedial commisure. Those are later used for placement of the annuloplasty ring.

The Lt. Atrium was first inspected for jet lesion, then the appendage for thrombus, then the Mitral annulus whether dilated or not. Two nerve hook retractors were used to stretch the anterior and the posterior Mitral leaflets to detect the presence of prolapse. We also Inspected the thickness of the leaflets, the span, especially the posterior Mitral leaflet (the span of the post. leaflet should be 1/3 the span of the ant. leaflet), and the subvalvular apparatus. № 1 silk strings were passed around the primary chorde groups of the posterior leaflet to facilitate its forward traction. The posterior leaflet was separated from the posterior annulus starting from the middle of the annulus then passing towards the two commisures leaving only small rim of leaflet tissue to facilitate suturing of the patch to the annulus. The 2ry & 3ry chorde of the post leaflet were released whenever needed to allow for better mobilization of the leaflet. That caused the posterior leaflet to "fall" forward towards the anterior leaflet.

The Autologous patch was then generously fashioned. The length of the patch needed was measured by using silk suture to measure the defect from the antro-lateral commisure to the postro-medial commisure taking in consideration the posterior Curve of the annulus. The width was calculated from the width of the anterior leaflet with a 1:3 ratio to avoid systolic anterior motion. Once appropriately sized, the patch was sewn into the defect of the posterior leaflet with two running 5-0 Prolene sutures. The smooth surface of the pericardium is turned toward the atrium for valve repair (Fig. 1).

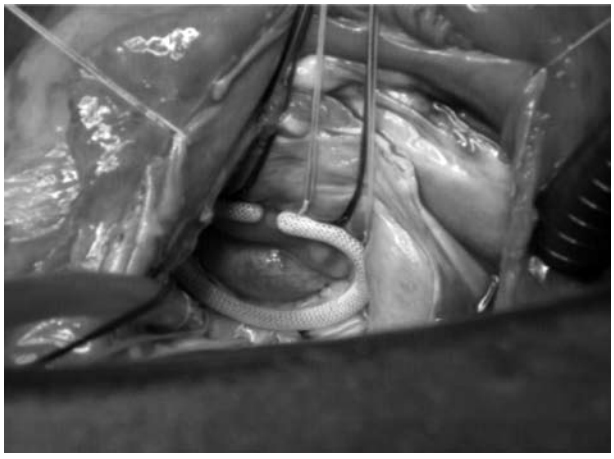


Fig. (1) Pericardial patch in place.

A complete annuloplasty ring was then placed in all patients. The annuloplasty ring size was determined by the trigone to trigone distance of the anterior leaflet. The completed repair restored the intraventricular zone of coaptation.

That Mitral valve repair was tested by filling the LV with saline through a bulb syringe (Fig. 2).

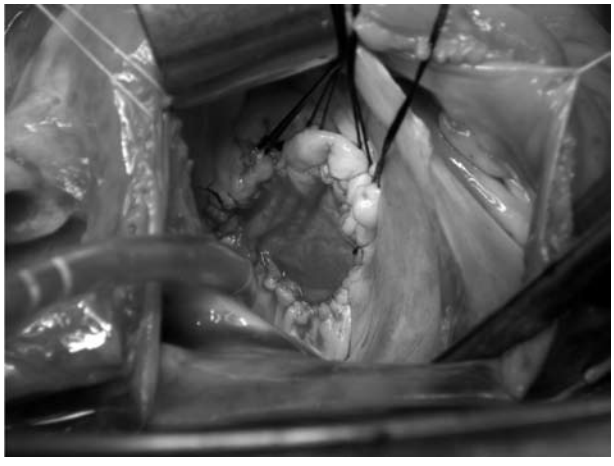


Fig. (2) Competent repair of Mitral valve with ring in place.

Another way of evaluating the Mitral valve competence was also done in some cases, by using Foleys urinary catheter passing it into the Lt. Ventricle through the aorta from the site of cardioplegia cannula. The balloon was inflated by using 5cc of saline, and the aorta was palpated to be sure that the catheter was not folded in the aorta. The Lt. Ventricle was filled with saline to test the Mitral valve repair without distorting the Mitral valve mechanics.

Routine closure of Lt. Atriotomy using 2 prolene (3/0) sutures, and then weaning from Cardiopulmonary bypass were performed.

Appropriate volume loading to maintain a CVP around 7-8 cmH2o, and hemodynamic manipulations to maintain BP within ±10% of preoperative value (using small boluses of norepinephrine or increasing the inotropic support) were important for adequate assessment of repair by TEE.

The post-CPB TEE assessment included initially 2D Exam for evaluating leaflet structure and motion, followed by assessment of residual or new areas of regurgitation, calculation of MV gradient and area, assessment of ventricular function and anatomy, and assessment of SAM.

The grade of MR was evaluated by CFD (Jet area, Jet area/LA area, vena contracta, PISA), and spectral Doppler.

The severity of MR was classified as: No or trivial, mild, moderate and severe MR, corresponding to 0, +1, +2, and +3.

Estimation of effective regurgitant orifice area (EROA) or calculation of regurgitant volume (RV) & regurgitant fraction (RF) were carried out in cases of uncertainty. That was guided with the recommendations of ASE for evaluation of severity of valvular regurge. [7]

Calculation of mean MV gradient was done using (CWD). A mean gradient > 6mmHg or peak gradient >16 was diagnostic of MS.

Measurement of MV area was done by planimetry (TG basal short axis view).

If systolic bending of leaflets into LVOT, turbulent flow in LVOT or posteriorly directed MR jet was demonstrated, gradient across LVOT was measured using CWD in the deep TG view & thickness of septum was obtained to asses the possibility of SAM. (The risk of SAM was evaluated prebypass based on length of MV leaflets, distance from coaptation point to septum and left ventricular internal diameter in systole).

After full satisfaction of the results of Mitral valve repair, decannulation, homeostasis and routine closure of the sternotomy were done.

The patients were transferred to the ICU on physiological doses of inotropic support and vasodilators.

Statistical Analysis

Data were statistically described in terms of frequencies (number of cases), relative frequencies (percentages), mean and standard deviation values (SD). All statistical calculations were done using Microsoft excel 7 computer program (Microsoft cooperation, NY, USA).

Results

Mitral valve repair by Posterior Mitral leaflet augmentation with a gluteraldehyde treated Autologous pericardial patch and complete annuloplasty ring were done in all patients for severe MR. In addition, the following Mitral repair techniques were used: commissurotomy (4patients, 26.6%), papillary muscle splitting and fenestration (1 patient, 6.6%) and release of 2ry and 3ry chorde (5 patients, 33.3%). The mean ring size was 30.53±0.91mm for Carpentier rings (Edwards Lifesciences LLC, Irvine, CA). The mean aortic clamp time and cardiopulmonary bypass time were 71.26±12.97 minutes and 91±15.49 minutes, respectively. Additional surgical procedures included tricuspid annuloplasty which was done in 3 patients, by Devega repair and AVR was done in 1 patient (Table 2).

Mitral surgical procedure	15patients
Posterior leaflet augmentat ion	
Commissurotomy	4 patients (26.6%)
Papillary muscle splitting	1 patient (6.6%)
Release of 2ry & 3ry chorde	5 patients (33.3%)
Additional procedure	
Tricuspid v. repair	3 patients
Aortic v. replacement	1 patients
Ring size Range	30-32mm
Mean	30.53±0.91mm
Cross clamp time	
Range	55-105min.
Mean	71.26±12.97min
Bypass time	
Range	65-130 min
Mean	91±15.49 min

Table (2): Operative data:

Clinical improvements were noted in all patients. The NYHA class improved to class I or II. Patients were anticoagulated for 30 days following repair. There was no operative or 30-dayes mortality in our study. We had 1 case (6.6%) of morbidity in the form of infective endocarditis on the Mitral annuloplasty ring. The patient presented by fever 2 weeks after surgery. Blood culture was positive. The patient received IV antibiotics f or two weeks and reoperation was done one month after the primary repair operation. Debridment of all the infected Mitral tissues was done and MVR using Carbomedics mechanical valve size 29 was used.

Transthoracic Echocardiography was done to all patients, before hospital discharge, and follow up echocardiography were also done at 3 and 6 months after the surgery to asses the degree of Mitral regurge, MVA in cm2 , mean PG across the Mitral valve to detect presence of Mitral stenosis, and whether there was systolic anterior motion abnormality (SAM) due to the pericardial augmentation of the posterior leaflet.

Intraoperative transoesophageal echo revealed: 73.3% of patients (11 patients) showed trivial Mitral regurge, 20% (3 patients) showed mild MR, while 6.6% (1 patient) showed moderate MR. The mean MVA was 2.45±0.11 cm2, with average mean PG across the Mitral valve was 4.26±1.22 mmHg. None of our patients developed SAM abnormality at the Intraoperative TEE. None of our patients underwent 2nd CPB run.

Transthoracic echocardiography was done to all patients before hospital discharge: 80% (12 patients) showed trivial MR, and 20% (3patients) showed mild MR, with mean MVA 2.58±0.09cm2 and average mean PG across the Mitral valve was 4.4±1.12mmHg.

Follow up echocardiography at 3month postoperative showed 78.57% (11 patients) with trivial MR, and 21.4% (3 patients) showed mild MR.(The patient who developed infective endocarditis was excluded) The mean MVA was 2.53±0.10cm2, with average mean PG across the Mitral valve 4.42±1.08mmHg.

Six month follow up echocardiography showed 71.4% (10 patients) with trivial MR, and 28.5% (4 patients) with mild MR. The mean MVA was 2.5±0.08cm2, with average mean PG across the Mitral valve was 4.5±0.85mmHg.

None of our patients showed SAM abnormality at any of the follow up echocardiography. Table (3) shows follow up Echocardiographic data.

	Degree of Mitral regurge	Mean MV area	Mean PG Across MV	SAM
Intra-operative echo	Trivial reg. 11p (73.3%) +1 reg. 3p (20%) +2 reg. 1p (6.6%)	2.45 ±0.11 cm2	4.26 ±1.22 mmHg	No
Before hospital discharge echo	Trivial reg. 12p (80%) +1 reg. 3p (20%)	2.58 ±0.09 cm2	4.4 ±1.12 mmHg	No
3 month follow up echo	Trivial reg. 11p (78.57%) +1reg.3p (21.4%)	2.53 ±0.10 cm2	4.42 ±1.08 mmHg	No
6 month follow up echo	Trivial reg.10p (71.4%) +1reg.4p (28.5%)	2.5 ±0.08 cm2	4.5 ±0.85 mmHg	No

Table (3) Follow up Echocardiography. Preoperative and postoperative transoesophageal echocardiography

Images are shown in Fig. (3) and (4).

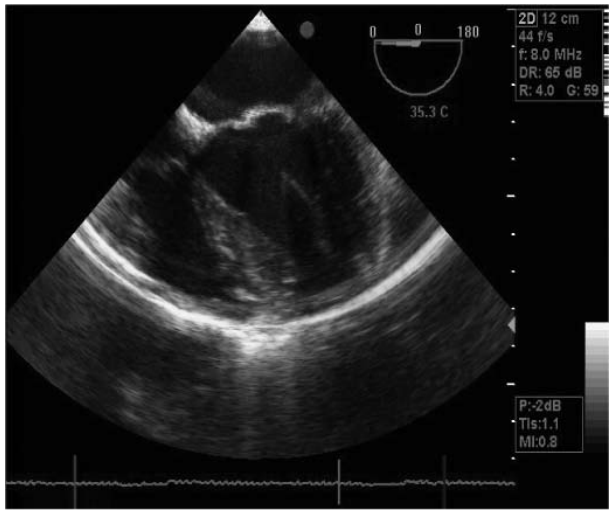


Fig. (3) Preoperative TEE image showing severe MR: morphology of anterior and posterior leaflets in four- chamber view.

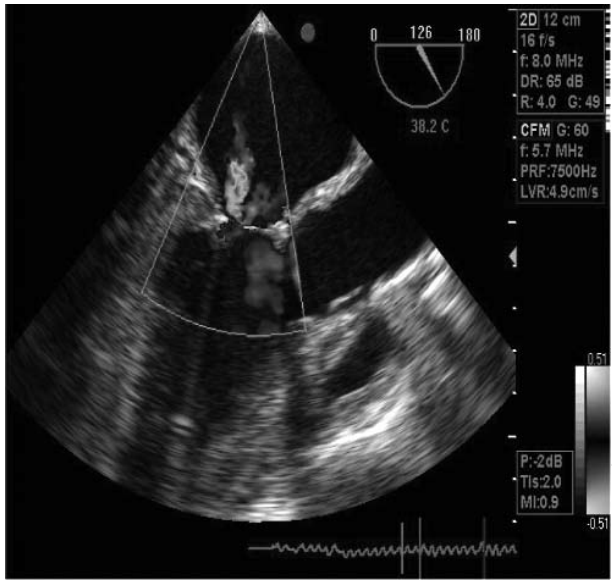


Fig. (4) Postoperative TEE image showing posterior Mitral leaflet augmentation repair showing trivial MR.

Discussion

The long-term results of Mitral repair for rheumatic valve insufficiency are still suboptimal in spite of undersized annuloplasty. Persistence or recurrence of Mitral regurgitation can occur due to severity of tissue retraction precluding leaflet coaptation (Carpentier type IIIa). The concept of repair with posterior patch augmentation is to lengthen the severely shortened and retracted posterior leaflet to reestablish coaptation.

The use of Autologous pericardial patch briefly fixed with gluteraldehyde in Mitral repair offers durable results with no calcification or retraction. It has been applied successfully for anterior leaflet augmentation in case of rheumatic Mitral valve insufficiency. (2)

The severity of tissue retraction involving both the leaflets and the chorde together with valve rigidity due to calcification, are the main pitfalls for valve repair in case of rheumatic Mitral disease and usually lead to valve replacement. To compensate tissue retraction, the technique of posterior leaflet augmentation has been shown to be safe and reproducible. As described, the enlargement concerned not only the posterior leaflet but also the commissural areas. The echocardiography study has shown that the posterior leaflet mobility and the surface of leaflet coaptation were significantly increased. In addition, it allowed the insertion of a larger annuloplasty ring, thereby reducing the risk for stenosis. The post bypass TEE evaluation was consistent with TTE results in the postoperative period. Except for one patient diagnosed as a border-line moderate (mild to moderate +2) MR with a negative leak test Intraoperative, the follow up TTE revealed a mild degree of MR. According to Chitwood, patients with a competent valve following the surgical leak test may still have significant valvular insufficiency by TEE secondary to ischemic wall dysfunction or the presence of SAM in the beating, volume-loaded heart. (8)

In follow up Echocardiography, none of our patients developed SAM abnormality. This may be due to the rheumatic origin of our pathology which leads to rigidity of the Mitral annulus. Also the use of Rigid Mitral ring in our patients reduced the risk of SAM.

Mitral valve repair preserves the Mitral valve apparatus and this has been shown to enhance and maintain left ventricular function. This data indicates that Mitral valve repair should be encouraged, perhaps even when anticoagulation cannot be avoided. In our series, one patient underwent concomitant mechanical aortic valve replacement, thereby necessitating long-term anticoagulation. However, by repairing the Mitral valve, the disruption of the Mitral valve-left ventricular unit is not altered and heart function may be preserved, ultimately this may delay the onset of heart failure and improve quality of life. (1)

Massive calcified Mitral annulus and subvalvular

apparatus may represent a real limit to perform this technique. Careful follow-up will confirm whether a wide use posterior leaflet augmentation substantially improves mid-term and long-term outcome of valve repair in rheumatic Mitral valve disease. (2)

Pericardium has been attractive to the cardiac surgeon for a long time. Its ready availability, its ease of handling, and its pliability make it an obvious choice when a defect must be eliminated. We believe that there is sufficient evidence to support the use of Autologous pericardium, as its long-term durability and low thrombogenicity offer several advantages. The benefits conferred by using autologous tissue are, the easily accomplished surgical technique, the effective functioning of the remodeled valve, and the preservation of the natural shape of the valve. These benefits make this technique a useful surgical alternative for extensive Mitral valve reconstructive procedures. (9)

Use of fresh autologous pericardium has been discouraged because of problems encountered after implantation, namely progressive contracture, thickening fibrosis, loss of pliability, early degeneration, and endocarditis. The method of preserving autologous pericardium is extremely important in determining tissue durability and preventing calcification. The scientific procedure of using Autologous tissue treated with a brief immersion in gluteraldehyde solution was established by Chauvaud and colleagues in 1991. On the basis of that experimental study, the clinical use of gluteraldehyde-pretreated pericardium became established for Mitral valve reconstructive procedures.(10) In our experience, we have observed no evidence of calcification or tearing of the pericardial patch, in our patients based on Echocardiographic evaluation.

Because gluteraldehyde-fixed autologous pericardium is non antigenic, it is preferred over the standard bovine pericardium, and this is corroborated by excellent results. In addition, xenograft tissue carries the small but proved risk of transmission of viral disease, including human immunodeficiency virus. (9)

Annular remodeling and fixation have been regarded as crucial endpoints of the surgical technique in order to assure long-term durability of repair. With the longest follow-up available to date, Carpentier and associates reported a 20-year freedom from reoperation of 92% in all cases which annular remodeling with prosthetic ring were performed. (11)

Valvuloplasty performed by surgeons proficient with the technique can offer outcome advantages suggesting that early operation is feasible before ventricular dysfunction occurs and allows for new strategies in these patients. The ultimate goal is restoration of a neo-valve with “normal” function, avoidance of the use of foreign materials, and gluteraldehyde-treated autologous pericardium could accomplish this goal. (9)

Although there has been question of the durability of valvular repair with pericardial tissue, none of the patients in our series required revision to a mechanical valve at over 6months of follow-up (Except the patient who developed infective endocarditis over the Mitral prosthetic ring). Clearly, more long-term data are necessary to reach firm conclusions.

In the literature, there were several studies on repair of severe Mitral regurge by pericardial augmentation of anterior Mitral leaflet, Micheal et al in 1998 reported a case of anterior leaflet pericardial augmentation for subaortic endocarditis.(12) Edward et al in 2004 described this technique in repair of ischemic Mitral regurge. The leaflet tethering in ischemic MR is directed posterior to the central orifice of the Mitral valve because both papillary muscles lie relatively posterior in the LV. On the basis of this anatomy, they described a method of repair that allows the level of leaflet coaptation to fall more posterior and toward the level of the displaced papillary muscles. (13)

In our study, there was a complete cooperation between the surgeon and anesthesiologist and the use of detailed MV examination by TEE was beneficial particularly in evaluating prebypass lesion and proving the efficiency of our surgical technique.

In the current study, there was an agreement between the prebypass transoesophageal echocardiographic examination and the direct surgical findings, as regard leaflet morphology, Mitral valve apparatus and mechanism of regurge in all patients with missed commissural fusion in one patient.

In their prospective study, Lambert et al, reported a small population of 13 patients who underwent surgery for significant Mitral regurgitation. The mechanism of regurgitation and location of pathology was identified by

using a systematic Echocardiographic examination in 12 patients (92%) compared with surgical inspection of the valve (4).

Intraoperative TEE provides a mean of real-time assessment of Mitral valve repair and a definitive mechanism to improve quality of care for our patients.

It is known that rheumatic valvular heart disease is a progressive and widespread process that may ultimately lead to progressive deterioration of heart function. Further information and follow-up is needed to determine the extended long-term durability of patch augmentation of the posterior leaflet. However, our results demonstrate excellent utility of repair and this technique should be considered for patients with severe Mitral Regurge due to retracted, shortened posterior leaflet.

References

1- Romano MA, Patel HJ, Pagani FD, Prager RL, Deeb GM and Bolling SF: Anterior Leaflet Repair With Patch Augmentation for Mitral Regurgitation, Ann Thorac Surg 2005;79:1500-1504.

2- Aubert S, Flecher E, Rubin S, Acar C and IradjGandjbakhch: Anterior Mitral Leaflet Augmentation with Autologous Pericardium. Ann Thorac Surg 2007;83:1560-1561.

3- Cheitlin MD, Armstrong WF, Aurigemma GP, et al.: ACC/AHA/ASE 2003 guideline update for the clinical application of echocardiography: Summary article: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (ACC/AHA/ASE Committee to Update the 1997 Guidelines for the Clinical Application of Echocardiography).

4- Lambert AS, Miller JP, Merrick SH, Schiller NB, Foster E, Muhiudeen-Russell I and Cahalan MK: Improved Evaluation of the Location and Mechanism of Mitral Valve Regurgitation with a Systematic Transoesophageal Echocardiography Examination. Anesth Analg 1999;88:1205–1212.

5- Iglesias I: Intraoperative TEE assessment during Mitral valve repair for degenerative and ischemic Mitral valve regurgitation. Semin Cardiothorac Vasc Anesth 2007; 11:301-305.

6- Shanwise JS, Cheung AT, Aronson S, et al. Guidelines for performing a comprehensive Intraoperative multiplane transoesophageal echocardiography examination: recommendation of the American Society of Echocardiography Council for Intraoperative Echocardiography and The Society of Cardiovascular Anesthesiologists task Force for Certification in Perioperative Transoesophageal Echocardiography. Anesth Analg 1999;89:870-884

7- Zoghbi WA, Enriquez-Sarano M, Foster E, Grayburn PA, Kraft CD, Levine RA, Nihoyannopoulos P, Otto CM, Quinones MA, Rakowski H, Stewart WJ, Waggoner A and Weissman NJ: Recommendations for evaluation of the severity of native valvular regurgitation with two-dimensional and Doppler echocardiography. Journal of the American Society of Echocardiography 2003; 16:777-782

8- Chitwood WR: Mitral valve repair: An odyssey to save the valves! J Heart Valve Dis 1998;7:255-261.

9- Choi-Keung N, Nesser J, Punzengruber C, Pachinger O, Auer J, Franke H, Hartl P: Valvuloplasty with gluteraldehyde-treated Autologous pericardium in patients with complex Mitral valve pathology. Ann Thorac Surg 2001;71:78-85.

10- Scrofani R, Moriggia S, Salati M, Fundaro P, Danna P, Santoli C: Mitral Valve Remodeling: Long-Term Results With Posterior Pericardial Annuloplasty. Ann Thorac Surg 1996;61:895-899.

11- Bevilacquaa S, Cerilloa AG, Gianettia J, Paradossia U, Mariania M, Matteuccib S, Kallushia E and Glaubera M: Mitral valve repair for degenerative disease: Is pericardial posterior annuloplasty a durable option? Eur J Cardiothorac Surg 2003;23:552-559.

12- Kincaid EH, Riley RD, Hines MH, Hammon HW and Kon ND: Anterior Leaflet Augmentation for Ischemic Mitral Regurgitation. Ann Thorac Surg 2004;78:564–568.

13- Frank MW, Mavroudis C, Backer CL, and Rocchini AC: Repair of Mitral Valve and Subaortic Mycotic Aneurysm in a Child with Endocarditis. Ann Thorac Surg 1998;65:1788 –1790.

Complete Mapping of The Tricuspid Valve Apparatus Using The Three -Dimensional Sonomicrometry.

Hosam Fawzy, MD

Background : **The importance of the tricuspid valve is often overlooked by cardiologists and surgeons because of its unique characteristics. Many surgeons consider the tricuspid valve as a second class structure.**
Our first objective was to determine the normal anatomy and dynamic characteristics of the tricuspid valve apparatus and the complex interrelationships of its various components in vivo. The second objective was to use the constructed three-dimensional geometry of the Tricuspid valve in the design of a tricuspid valve annuloplasty ring model that closely replicates the anatomy and function of the natural valve.
Methods : **The technique used in these studies was 16 channel three-dimensional (3D) digital sonomicrometry (Sonometrics Corp., London, Ontario, Canada) (series 5001 digital sonomicrometer). Sixteen sonomicrometry crystals were placed around the tricuspid annulus, at the bases and tips of the three papillary muscles (PMs), free edges of the three leaflets and the right ventricular apex during the cardiopulmonary bypass (CPB) in five anaesthetized York Hampshire pigs. Animal were studied after weaning of CPB on 10 cardiac cycles of normal heamodynamics.**
Results : **Sonomicrometry array localizations demonstrate the multiplanar shape of the tricuspid annulus (TA). It reaches its maximum area (97.9 ± 25.4 mm2) at the end of diastole and its minimum (77.3 ± 22.5 mm2) at the end of systole and increases again in early diastole, showing a biphasic pattern. Papillary muscles shorten 0.8 - 1.5mm (11.2%) in systole and chordrea tendinea straighten by 0.8-1.7mm (11.4%) in systole.**
Conclusion: **Tricuspid valve apparatus is a complex and has a very efficient valvular mechanism with a geometry that changes continuously during the cardiac cycle. The shape of the tricuspid annulus is not a saddle shape. It has a multiplanar 3D shape with its highest point at the anterseptal commissure and its lowest point at the posteroseptal commissure while the anteroposterior commissure lies in a middle plane in between. Tricuspid annulus area undergoes two major contraction and expansion during the cardiac cycle reaching its maximum during diastole and its minimum during systole. The PMs contract to offset the straightening of the chordae tendinea. This suggests that there is a balance between active structures those contract and passive structures that expand, producing minimal geometrical distortion of the tricuspid apparatus. Optimal tricuspid annuloplasty ring should be a multiplanar 3D one that mimics the normal anatomical shape of the tricuspid annulus.**

Address reprint request to : Dr Hosam Fawzy MD
Department of Cardic Surgery1 Tanta University.
Email : fawzyh@smh.toronto.on.ca
Codex : 04/112/0906

The importance of the tricuspid valve is often overlooked by cardiologists and surgeons because of its unique characteristics. Many surgeons consider the tricuspid valve as a second class structure.1 Most lesions of the tricuspid vale are usually ignored based on the wrong assumption that tricuspid disease is rare and,

in most cases, irrelevant to the patient's outcome after the problem on the left heart that brings the patient to surgery has been solved. One explanation of this maybe the absence of a detailed preoperative study for the tricuspid valve.

A better understanding of the tricuspid valve is necessary to clarify the indications and the limits of tricuspid valve surgery. Because of the importance of the annulus, an annuloplasty ring designed specifically for the tricuspid position should replicate the anatomy and function of the natural tricuspid valves as closely as possible. The specific geometric relationship that is formed by the attachment of the valve leaflets to the papillary muscles (PMs) and to the annulus is likely to be critical in ensuring proper function of both the valve and the right ventricle.2-8 This geometric relationship between the components of the tricuspid valve, however, has not been studied in detail. Previous studies have documented that the mitral valve annulus is saddle shaped, 9, 10 but we do not know if the tricuspid annulus resembles the mitral one and how its shape changes during the cardiac cycle relative to contraction of the PMs. Since the annulus also has both fibrous and muscular components, it likely both dilates and contracts simultaneously within its various sections. Moreover, the network of supporting chordae is highly branched and complex and the load distribution on these chordae has not been studied. It is therefore unlikely that a successful tricuspid valve annuloplasty ring can be developed and properly implanted into patients, unless a thorough understanding of the function of the tricuspid valve apparatus is obtained and implemented. Detailed spatial and temporal information is consequently needed to map the geometric and functional relationships among the contraction of the PMs, the stretching of the tricuspid valve chordae, and the distortion of the tricuspid valve annulus. The unique characteristics of the tricuspid valve, which has been defined as the "Cinderella" of the cardiac valves, are :(1) It is rarely affected in isolation. Most often, the prominent manifestation of other diseased valves minimizes its importance. (2) Located at the entrance of the heart, its symptomatology is primarily extracardiac and is often silent. (3) Its behaviour is closely related to the function of the right ventricle since in most cases, tricuspid regurgitation is secondary to right ventricular dysfunction. (4) Its manifestations maybe directly linked to other pathology. For example, resolution of the mitral problem is often followed by improvement in the degree of tricuspid regurgitation. (5) Because it usually exits in a low –pressure system, it maybe difficult to evaluate its preoperative importance and assess the value of different surgical techniques. These characteristics of the tricuspid valve cause cardiologists and surgeons to often disregard its importance.

Aim of the study:

To study the anatomy and dynamic characteristics of the tricuspid valve apparatus in an open heart pig model. Better understanding of the normal anatomy and function of the tricuspid valve will help to clarify the indications and the limits of tricuspid valve surgery. This can also aid in the design of a tricuspid valve annuloplasty ring that closely replicates the anatomy and function of the natural valve annulus.

Methods:

The technique used in these studies was 16 channel three-dimensional (3D) digital sonomicrometry (Sonometrics Corp., London, Ontario, Canada)(series 5001 digital sonomicrometer). The sonomicrometer measures distances within soft tissue by means of piezoelectric transducers, a high frequency counter, and the time of flight principle of ultrasound. Sixteen of these ultrasonic transducers were implanted into tissue (see below) and sequentially energized with a short electrical pulse. As each transducer was energized, it generated an acoustic wave that propagated through the tissue, away from the transducer in a radial manner. This spherical wave front was detected by each of the other transducers, and the distances between each transmitting crystal and all the receiving crystals were calculated based on the time taken for the wave to travel between an acting transmitter and its corresponding receiver. This cycle was repeated in turn for each of the other 15 transducers, such that each one in turn act as a transmitter during one firing cycle and as a receiver during the other 15 (Figure 1). The digital hardware in the computer controlled sonomicrometer automatically switched between transmit and received modes for the appropriate transducer. This switching was virtually instantaneous compared with biologic motion, since a complete cycle of sequential firing of all 16 transducers could be repeated at 200 Hz.

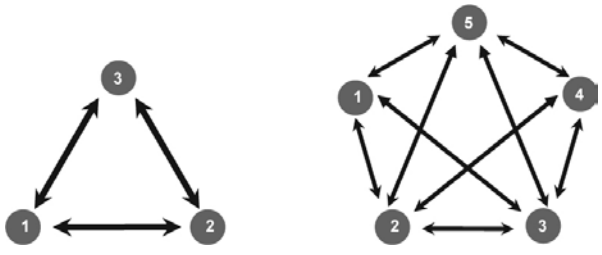


Figure 1. Schematic representation of the number of distances acquired by the digital sonomicrometer. Since each crystal can communicate with all others in the network, the number of distances measured increases exponentially.

Surgical Procedure:

With the approval of the Institutional Animal Use and Care Committee, we developed a porcine model to study the anatomy and the function of the tricuspid valve. We used 2-4 week old Yorkshire pigs (n=5) with an average body weight of 40.6 ± 2 kg. After insertion of an ear vein, the animal was anesthetized with ketamine (20 mg/kg) and acepromazine (1 mg/kg). Following endotracheal intubation, Ventilation was controlled to maintain normocarbia with PaO2 > 150 mmHg. Anaesthesia was maintained throughout the procedure with Isoflurane or enflurane (1-3%). An arterial outline was inserted into the left external carotid artery for continuous blood pressure monitoring and arterial blood sampling for blood gases and serum electrolytes every 30 minutes. A pulmonary artery Catheter was used to measure the pulmonary artery pressure. A right lateral thoracotomy followed by a pericardiotomy was performed to expose the heart and its great vessels. Heparin was given intravenously at the dose of 300 units/kg. Cannulation was carried out using an aortic cannula (Edwards Lifesciences G 22 Fr.) and two separate venous cannulae (Medtronic DLP size 28 Fr.) for the superior and inferior venae cavae. Once activated clotting time (ACT) exceeded 480 sec, cardiopulmonary bypass (CPB) was started. The pump flow rate was 50ml/Kg/min (2-2.5 litres/min). Temperature was kept at 32 - 35 ºC (mild hypothermia) by drift. The aorta was then clamped and cold blood cardioplegia (Fremes solution at 8:1 ratio and temperature of 4 ºC) was infused antegradely into the aortic root to stop the heart. Mean arterial pressure was maintained at 50-60 mmHg throughout CPB. Transverse right atriotomy was performed to expose the tricuspid valve. The 16 sonomicrometer crystals were attached to the tricuspid valve apparatus as follow: Six crystals were placed at the base and apex of the three PMs, two for each one; three crystals were attached at the leaflet ends of the marginal chordae at their sites of attachment to the anterior, posterior and septal leaflets; six crystals were placed around the tricuspid annulus (Three at the commissures and three in between in the middle of annular attachment of each leaflet); and the remaining crystal was attached to the right ventricular apex as a reference (figure 2).

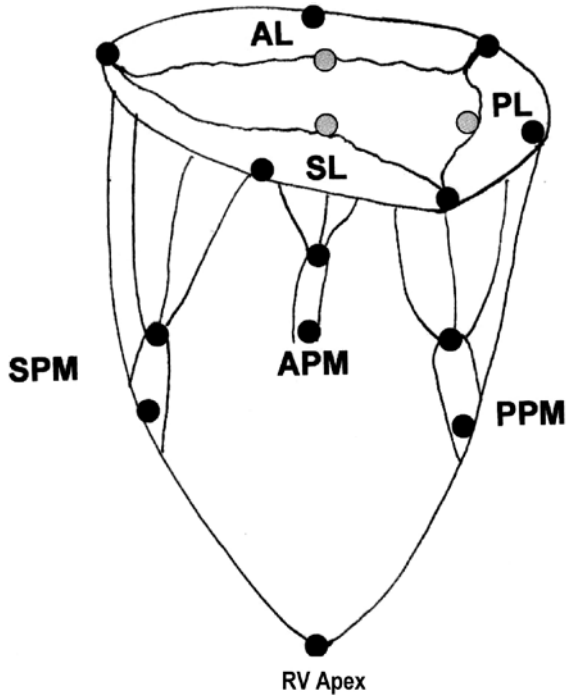


Figure 2: Diagram showing the positions of the crystals that distribute all over the different parts of the Tricuspid Valve apparatus.

All the crystals were fixed using 5/0 prolene sutures. The wires for the crystals fixed to the annulus and the chordea tendinae were routed through the atrium while those fixed to the papillary muscles were routed out through a small incision at the right ventricular apex. Each crystal was connected to the sonomicrometer, and the pig was weaned from cardiopulmonary bypass. Heparin reversal was achieved using intravenous Protamine at a dose of 10 mg per 100 units of Heparin. After hemodynamics reached the normal pre-operative status, we recorded for 5-10 consecutive cardiac cycles data over a 5 sec interval. During each measurement cycle, the 16 channel sonomicrometer stores 300 distances. At a repetition rate of 200 Hz, a typical 5 sec data set would contain 300,000 data points, each 2 bytes long. At the end of the experiment, the animal was sacrificed by exsanguination into the cardiotomy reservoir.

3D RECONSTRUCTION:

Using trigonometry, any set of distance measurements between an array of points was converted to 3D coordinates with respect to some coordinate system. These conversions were done with the use of "triangulation algorithm" that employs the sine and cosine laws for triangles. Method of 3D reconstruction and visualization did not vary than that previously described11.

Statistical Analysis:

Data were expressed as mean ± standard deviation and compared using 2- tailed unpaired student t test. Values of p less than 0.05 were considered significant. Statistical analyses were made by using Microsoft Office Excel 2003 for Windows XP.

Results:

Model Characteristics:

The hemodynamics at the time of recording were: heart rate 95 ± 6 beats/min; arterial pressure 84/45 ± 19/5 mmHg; Pulmonary artery pressure 23/14 ± 3/2 mmHg. All hemodynamic data are shown in table 1. At necropsy, all transducers were in the correct positions in all the animals.

Hemodynamic Parameters	Mean ± SD
Heart Rate (beats/min)	95 ± 6
Systolic Blood Pressure (mmHg)	84 ± 19
Diastolic Blood Pressure (mmHg)	45 ± 5
Mean Blood Pressure (mmHg)	50 ± 10
Systolic Pulmonary Pressure (mmHg)	23 ± 3
Diastolic Pulmonary Pressure (mmHg)	14 ± 2
Mean Pulmonary Pressure (mmHg)	15 ± 3

Table1. Hemodynamic Data during recording. Values are means ±SD where shown.

Measurement	Minimum (mm)	Maximum (mm)	Change (%)
Annulus:			
Anterior Segment:	11.6 ± 1.1	13.5 ± 1.7	16.2 ± 3.1%
Posterior Segment:	15.5 ± 5.7	17.3 ± 5.7	12.5 ± 3.5%
Septal Segments:	16.7± 5.1	18.7 ± 4.4	10.1 ± 0.9%
Chordea Tendinea:			
Anterior Chord:	9.6 ± 3.1	11.0 ± 3.1	14.0 ± 4.6%
Posterior Chord:	9.5 ± 3.3	11.2 ± 2.8	16.9 ± 8.4%
Septal Chord:	14.0 ± 3.5	14.8 ± 3.7	5.2 ± 0.4%
Papillary Muscles:			
Anterior PM:	11.8 + 2.6	13.3 + 3.1	11.1 + 1.3%
Posterior PM:	11.0 + 4.0	11.8 + 3.9	7.5 + 3.4%
Septal PM:	6.5 + 1.7	7.6 + 1.4	16.2 + 6.0%

Table2. Length data for Tricuspid Valve Annulus, Chordae Tendinea and Papillary Muscles. Values are means ± SD where shown.

(1) Tricuspid Annulus changes:

Two-dimensional shape of the tricuspid annulus (TA): the localized distances of each of the six crystals fixed to the TA from the reference crystal at the apex of the right ventricle are summarized in figure 3. Posteroseptal (PS) segment of the TA were close to the right ventricular (RV) apex, and the lowest point from the right atrium was the posteroseptal commissure where the coronary sinus started. In contrast, anteroseptal (AS) and anteroposterior (AP) segments were close to the right atrium, and the highest point of the TA was at the anteroseptal commissure which was close to the RV outflow tract and the aortic valve. Anteroposterior commissure was at a plane in between the anteroseptal and posteroseptal commissures.

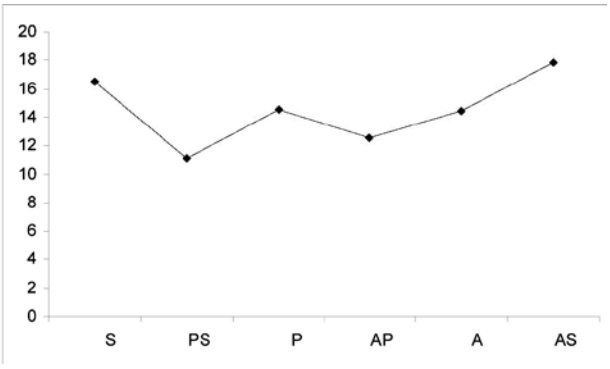


Figure 3: Positions of the six Tricuspid annular Crystals in relation to the right ventricular apex.

Three-dimensional shape of the Tricuspid annulus: The shape of the tricuspid annulus was determined by the 3D coordinates of each annulus crystal. Figure 4 is a comprehensive illustration of the 3D shape of TA at end-systole and end-diastole from one animal. An arbitrary surface passing through each of the annular transducers has been drawn for better demonstration of the multi-planner shape of the annulus which is maintained throughout the cardiac cycle. All pigs studied had annuli of similar shapes. The multiplanar shape of the annulus was defined by the height of each crystal above or below the least-squares plane. The resultant averages determined a multiplanar oriented shape with a maxima corresponding to the AS crystal, intermediate at AP crystal and a minima at PS crystal.

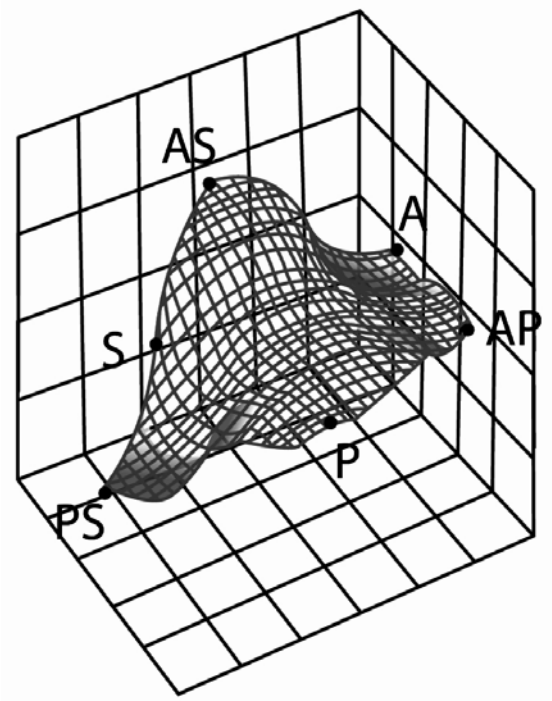


Figure 4: Three Dimensional shape of the Tricuspid Annulus at systole.

Dynamic Changes of the tricuspid annular area: The TA area increased from mid to late diastole reaching a maximum area of 97.9 ± 25.4 mm2 at the end of diastole, decreased during systole reaching its minimum of 77.3 ± 22.5 mm2 at the end of systole, and increased again in early diastole, showing a biphasic pattern with two peaks in early and late diastole (figure 5).

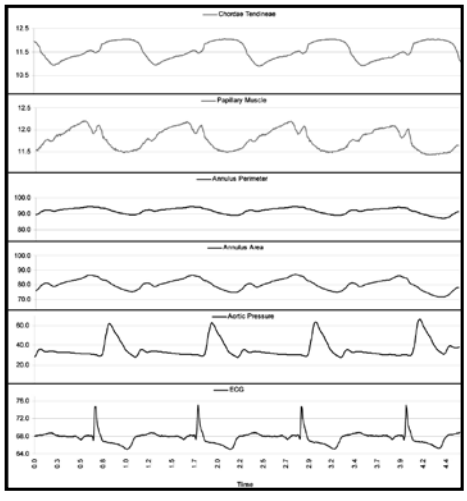


Figure 5: ***Example of dynamic changes of the Tricuspid valve apparatus during the Cardiac Cycle in one animal.***

Dynamic Changes of the tricuspid annular perimeter: The TA perimeter was also calculated to measure the cross-sectional changes during different phases of the cardiac cycle. The annular perimeter expanded from a minimum of 82.5 ± 19.0 mm in late systole to a maximum of 89.0 ± 19.2 mm in late diastole with an expansion of 7.9 ± 0.8%. However, this total expansion was not homogenous in all segments. Most of the changes of the annular circumference were along the anterior annulus (16.2 ± 3.1%). As viewed in the axial plane, there was a relatively little change of the circumference along the posterior annulus (12.5 ± 3.5%). The septal expansion was smaller (10.1 ± 0.9%) than that of the anterior or posterior segments, but not statistically significant (p=0.28 and p= 0.58, respectively). The changes of the different annular segments in the axial plane can be seen at maximum and minimum movements in figure 6.

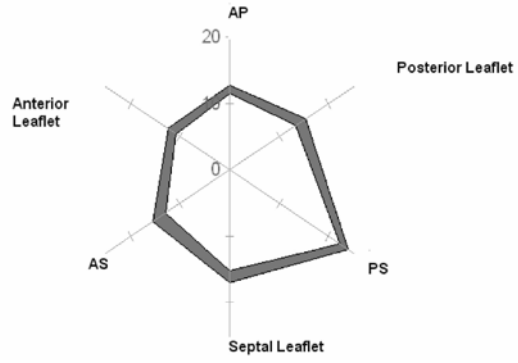


Figure 6: Segmental changes of different parts of the tricuspid annulus.

(2) Leaflet motion:

The opening of the tricuspid valve was determined from the area of the triangles designated by the three crystals on the edges of the three leaflets and the six annular crystals. The leaflets began to open in early diastole, completing their opening by the end of diastole. This is followed by closure during systole. The maximum tricuspid leaflet area occurred near end-systole and averaged 127.3 ± 33 mm2 while the minimum area occurred at the end of diastole and averaged 100.5 ± 29.3 mm2. The mean ratio of leaflet area to orifice area was 1.3.

(3) Chordea tendinae:

The distance between crystals on the chordea tendinea began to lengthen in early systole reaching

its maximum by the end of systole and its minimum in diastole. The intercrystal distance of the anterior chordea tendinea lengthened from 9.6 ± 3.1 to 11.0 ± 3.1 mm (14.0 ± 4.6%) while that of the posterior chords increased from 9.5 ± 3.3 to 11.2 ± 2.8 mm (16.9 ± 8.4%) and that of the septal chords from 14.0 ± 3.5 to 14.8 ± 3.7 mm (5.2 ± 0.4%). The overall chordal intercrystal distance increased by 11.4%.

(4) Papillary muscle movement:

The motion of the PMs relative to the TA plane can be described as a combination of shifting, bending, and twisting of the right ventricle. During the cardiac cycle, the average of the three PMs contracted by 11.2% during systole and the maximum change in length was 0.8 - 1.5 mm.

The PMs were shortest during systole (11.8 ± 2.6, 11.0 ± 4.0 and 6.5 ± 1.7 mm) and longest during diastole (13.3 ± 3.1, 11.8 ± 3.9 and 7.6 ± 1.4 mm) for anterior, posterior and septal PMs respectively (Figure 7).

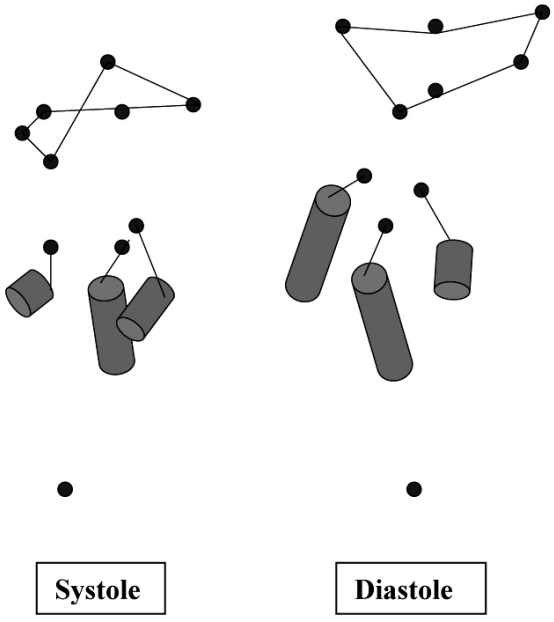


Figure 7. Papillary muscles and their relation to the Tricuspid annulus.

The rotational movement of the PMs during the cardiac cycle indicates considerable changes in the direction of the forces tethering the tricuspid leaflets. However, the torsion along the septal PM (16.2 ± 6.0%) was insignificantly greater in magnitude than anterior PMs (11.1 ± 1.31%, p=0.35), while significantly greater than posterior PMs (7.5 ± 3.4, p= 0.029*).

Angular displacement of the PMs below the corresponding commissures showed that the PM plane was not parallel to the TA plane, and the PMs did not move together in the same direction. In fact, the angles between the two planes were 54.1°, 66.4°, and 41.7° for anterior, posterior and septal PMs respectively.

Figure 8: Optimal Tricuspid Annuloplasty ring.

Discussion:

Because the majority of tricuspid valve pathology has been considered secondary to left-sided lesions and pulmonary hypertension, it was assumed to revert spontaneously after these lesions has been treated. These factors, together with its characteristic paucity of clinical manifestations, have resulted in a lack of solid principals for its diagnosis, indications, and appropriate surgical manoeuvres. Previously, the diagnostic and surgical methods for treating functional tricuspid regurgitation (TR) have very closely followed those applied for the mitral valve. However, the tricuspid valve has distinguishing characteristics that are often ignored. Its tendency to vary in degree following hemodynamic changes makes its evaluation difficult and unreliable. A recent review of 790 patients who underwent a variety of tricuspid annuloplasties showed that between 15% and 37% of these cases had recurrent severe regurgitation at eight years follow up12. These results suggest that the current annuloplasty techniques are either unreliable or insufficient.

A deeper knowledge of the continuous changes in the shape of the tricuspid valve throughout the cardiac cycle should aid in both understanding the mechanisms of its dysfunction and in developing new interventional

therapies.

We demonstrated that the shape of the tricuspid annulus (TA) is multiplanar with non homogenous contraction. The highest point of the TA is at the anteroseptal commissure, which is close to the RV outflow tract and the aortic valve. The lowest point toward the RV apex from the right atrium is the posteroseptal commissure, where the coronary sinus starts. While the anterposterior commissure lies at a middle plane. The geometry of the TA thus may be complicated and appears to be different from the saddle shape of the mitral annulus, suggesting an annuloplasty in TR should be different from that in mitral regurgitation. Fukuda et al. 13 studied the Tricuspid valve in both healthy subjects and patients with Tricuspid regurgitation using real-time 3-D echocardiography and concluded a similar shape of the tricuspid annulus like ours.

There have been many studies of the saddle-shaped mitral annulus 14-17. Many surgeons still believe that the tricuspid annulus has the same saddle shape of the mitral annulus. In a previous study with a small number of subjects, 3D Echo with a reconstruction method was used to measure the 3D structure of the normal TA and showed that the shape of TA is saddle shape 18. A recent experimental study used sonomicrometery in an ovine model with opening of the pericardium19 confirmed the saddle shape of the TA with the highest point corresponding to the midpoint of the posterior leaflet and the lowest point corresponding to the anteroposterior commissure. The anteroseptal commissure and the middle of the anterior leaflet, lies in a middle plane.

In the post-mortem human heart, Deloche et al.20 described the shape of the normal tricuspid annulus as a pear-shaped with its narrow end close to the anteroseptal commissure and its wider end corresponding to the midpoint of the posterior leaflet. The same pear-shaped annulus was also described by Hiro et al.19 in their pressurized post-mortem sheep heart. Recently, in their in vivo ovine model, they observed the opposite shape with the narrow end of the pear-shaped annulus corresponded to the posteroseptal commissure.

The mechanical advantage of the saddle shape of the mitral annulus is well known as it has been shown to reduce valve stress21. These issues have not been fully studied for the tricuspid annulus.

In the present study, we found that changes in the multiplanar shape of the TA during the cardiac cycle also contribute to its orifice area. The decrease in the multiplanar shape during systole has a 'folding' reducing effect on the TA. The high and low points of the annulus move away from each other in the apical/basal direction during TA area dilation. This minimizes leaflet stress and it also allows a 'folding' of the annulus that effectively reduces its orifice area without a dramatic reduction in perimeter. This change in shape is the main mechanism responsible for the TA competence, and questions the efficiency of rigid annuloplasty devices that interfere with this mechanism. Using open heart dog model, Tsakiris group7 found that the Tricuspid valve is not a rigid structure with atrial and ventricular contractions are responsible for TA competence with the major part of annular narrowing caused by atrial contraction. Atrial contraction brought the anterior and posterior leaflets to appose the septal leaflet.

Previous experimental and clinical studies have shown that the TA continuously changes its size and shape during the cardiac cycle7 & 22. In the present study, although the TA area contracted to 21 % of its maximum area, the maximum contraction of the TA perimeter was only 7.9 + 0.8%. These obtained data confirmed those of Tsakiris and associates7 who reported a contraction between 20% and 39% of the maximal diastolic area. In an echocardiographic study of normal humans, Tei et al.6 showed a maximum reduction of 33%. An earlier report by Hiro et al. 19 reported that ratio of the maximum–to-minimum diameters changed from 0.55 + 0.05 mm to 0.46 + 0.04 mm as the TA became less circular.

However, these changes were not homogenous in all segments. As most of the changes of the annular circumference were along the anterior annulus (16.2 + 3.1%), then the posterior annulus (12.5 + 3.5%) and the least reduction occurred at the septal segment (10.1 + 0.9%) with no significant difference. Contrary to these results, Hiro et al.19 reported that the length of insertion of the septal leaflet changed almost as much as the insertion of the anterior and posterior leaflets (10.4 + 1.2% versus 13.0 + 1.5% versus 14.0 + 1.6%, respectively). He believed that the reduction in the annulus orifice was due to a selective shortening of the free wall portions.

The biphasic nature of the TA area curve is comparable

to that observed in the mitral valve by Gorman et al.9 and in the tricuspid valve by Tei et al.6 and Tamiya et al.23. The maximum TA area occurred during late diastole and is most likely due to relaxation of the right ventricular transverse fibers in late diastole24.

The PMs contracted by 11.2% while the chordae tendinae straightened by 11.4%. This implies that the PMs contract to offset the straightening of the chordae tendinea. This has not been reported previously and suggests that there is a balance between active structures those contract and passive structures that expand, producing minimal geometrical distortion of the tricuspid valve apparatus.

The length of the tricuspid valve's septal segment also changes during the cardiac cycle (10.1 + 0.9%). This finding challenges both the standard method of using the length of the septal leaflet base to select the correct ring size as well as the use of open annuloplasty bands, which may not prevent further dilatation of the diseased TA. All these findings question the adequacy of all present annuloplasty techniques that selectively reduce the portion of the annulus corresponding to the right ventricular free wall, assuming that the length of the septal portion of the annulus remains constant in health and disease. Using the length of the base of the septal leaflet to determine the optimal size of the repaired annulus, was derived from the mitral valve, where the fibrous intertrigonal segment was considered stable and constant.

These preliminary data revealed that the tricuspid valve undergoes continuous and complex geometric changes during the cardiac cycle. As in the case of the mitral valve, none of the structures of the tricuspid valve can be studied in isolation. It is hoped that better knowledge of the dynamic changes of the normal tricuspid valve will promote understanding of the mechanisms responsible for its functional regurgitation and consequently help in designing an anatomically and physiologically correct tricuspid annuloplasty ring.

Optimal Tricuspid Annuloplasty Ring:

Tricuspid valve (TV) annuloplasty is now a standard surgical procedure for the treatment of functional TR. Most surgeons consider the use of an annuloplasty ring an essential part of basic repair techniques for obtaining good results. However, the rings currently being used for TV annuloplasty were originally released for mitral valve, and moreover, most rings are formed in a single plane with variation, 25-27 whereas the actual TA has a multiplanar 3D structure28. Therefore, there was a need for a better understanding of the 3D geometry of the TA, especially in the normal heart and that is well illustrated in this study.

A new look at the tricuspid valve based on the better understanding of its functional anatomy should stimulate the development of a new three dimensional design for a tricuspid annuloplasty ring that is not a saddle shape and it is mulitplanar 3D in shape and with its highest point at the anterseptal commissure and its lowest point at the posteroseptal commissure while the anteroposterior commissure lies in a middle plane in between (figure 8) (patent pending).

The tricuspid annuloplasty ring should be made from a semi-rigid material that does not interfere with the folding mechanism of the annulus. The material preferably has progressive degree of flexibility along the whole length of the ring from septal through the posterior and anterior segments. The ring should have its lowest flexibility near the septal leaflet, the highest flexibility near the anterior leaflet.

Conclusion:

Tricuspid valve apparatus is a complex and has a very efficient valvular mechanism with a geometry that changes continuously during the cardiac cycle. The shape of the tricuspid annulus is not a saddle shape. It has a multiplanar 3D shape with its highest point at the anterseptal commissure and its lowest point at the posteroseptal commissure while the anteroposterior commissure lies in a middle plane in between. Tricuspid annulus area undergoes two major contraction and expansion during the cardiac cycle reaching its maximum during diastole and its minimum during systole. The PMs contract to offset the straightening of the chordae tendinea. This suggests that there is a balance between active structures those contract and passive structures that expand, producing minimal geometrical distortion of the tricuspid apparatus. Optimal TV annuloplasty ring should be a multiplannar 3D one that mimics the normal anatomical shape of the TA.

References:

1. Duran CM: Acquired disease of the tricuspid valve. Surgery of the chest; Gibbon's Sabiston and Spencer, 7Th edition; 2005: 1335-54.
2. Fann JI, Miller CD, Moore KA, et al. Twenty-year clinical experience with porcine bioprosthesis Ann thorac Surg 1993; 62: 1301-1312.
3. Smith W, Vesely I: Three dimensional ultrasonic micrometer for use in cardiovascular research. Proceeding of the 13th conference of the IEEE-EMBS, 1991.
4. Angermann CE, Spes CH, Tammen A, et al: Anatomic Characterstics and valvular function of the transplanted heart: transthoracic versus transesophageal echocardiographic findings. J Heart Transplant 1990; 9:331-338.
5. Ohata T, Kigawa I, Tohda E, et al: Comparison of durability of bioprosthesis in tricuspid and mitral position. Ann Thorac Surg 2001; 71: S240-S243.
6. Tei C, Pilgrim JP, Shah PM, et al: The tricuspid valve annulus: study of size and motion in normal subjects and in patients with tricuspid regurgitation. Circulation. 1982; 66:665-671.
7. Tsakiris AG, Mair DD, Seki S, et al: Motion of the tricuspid valve annulus in anesthetized intact dogs. Circulation Res 1975; 36:43-48.
8. Munro AI, Jamieson WRE, Tyers GFO, et al: Tricuspid valve replacement: porcine bioprosthesis and mechanical prosthesis. Ann Thorac Surg 1995; 59:S470-S471.
9. Gorman JH, III, Gupta KB, Streicher JT, et al. Dynamic three-dimensional imaging of the mitral valve and left ventricle by rapid sonomicrometry array localization. J Thorac Cardiovasc Srug 1996; 112: 712-726.
10. Cheng TO, Xie MX, Wang XF, Li ZA, Hu G. Evaluation of mitral valve prolapse by four dimensional echocardiography. Am Heart J 1997; 133: 120-129.
11. Vesely I, Fawzy H, Fukamachi K, Drake M. Use of Three-Dimensional Sonomicrometery to study the Motion of the Mitral Valve. ASAIO Journal 1997; 43:M465-469.
12. McCarthy PM, Bhudia SK, Rajeswaran j, et al. Tricuspid valve repair: Durability and risk factors for failure. J Thorac Cardiovasc Surg 2004; 127:674-685.
13. Fukuda S, Saracino G, Matsumura Y, Daimon M, Tran H, Greenberg NL, Hozumi T, Yoshikawa J, Thomas JD, Shiota T. Three-Dimensional geometry of the tricuspid annulus in healthy subjects and in patients with functional tricuspid regurgitation, a real-time 3-Dimensional echocardiographic study. Circulation. 2006; 114 [suppl I]: I-492-I-498.
14. Ormiston JA, Shah PM, Tei C, Wong M. Size and motion of the mitral valve annulus in man, I: a two-dimensional echocardiographic method and findings in normal subjects. Circulation. 1981; 64: 113–120.
15. Levine RA, Handschumacher MD, Sanfilippo AJ, Hagege AA, Harrigan P, Marshall JE, Weyman AE. Three-dimensional echocardiographic reconstruction of the mitral valve, with implications for the diagnosis of mitral valve prolapse. Circulation. 1989; 80: 589–598.
16. Glasson JR, Komeda MK, Daughters GT, Niczyporuk MA, Bolger AF, Ingels NB, Miller DC. Three-dimensional regional dynamics of the normal mitral anulus during left ventricular ejection. J Thorac Cardiovasc Surg. 1996; 111: 574–585.
17. Kwan J, Qin JX, Popovic ZB, Agler DA, Thomas JD, Shiota T. Geometric changes of mitral annulus assessed by real-time 3-dimensional echocardiography: becoming enlarged and less nonplanar in the anteroposterior direction during systole in proportion to global left ventricular systolic function. J Am Soc Echocardiogr. 2004; 17: 1179–1184.
18. Chandra S, Powell K, Breburda CS, Mikic I, Shekhar R, Morehead AJ, Cosgrove DM, Thomas JD. Three dimensional reconstruction (shape and motion) of tricuspid annulus in normals and in patients after tricuspid annuloplasty with a flexible ring. Comput Cardiol. 1996; 23: 693-696.
19. Hiro ME, Jouan J, Pagel MR, Lansac E, Lim KH, Lim HS, Duran CM. Sonometric study of the normal tricuspid valve annulus in sheep. J Heart Valve Dis. 2004; 13: 452–460.
20. Deloche A, Guerinon J, Fabiani JN, et al. [Anatomical study of rheumatic tricuspid valvulopathies. Applications to the critical study of various methods of annuloplasty]. Arch Mal Coeur Vaiss 1974; 67:497-505 (in French)
21. Salgo IS, Gorman JH 3rd, Gorman RC, Jackson BM, Bowen FW, Plappert T, St John Sutton MG, Edmunds LH Jr. Effect of annular shape on leaflet curvature in reducing mitral leaflet stress. Circulation. 2002; 106: 711–717.
22. Duran CM, Pomar JL, Colman T, Figueroa A, Revuelta JM, Ubago JL. Is Tricuspid valve repair necessary? J Thorac Cardiovasc Surg 1980; 80: 849-860.
23. Tamiya K, Higashidate M, Kikkawa S. Real-time and simultaneous measurement of tricuspid orifice and tricuspid annulus areas in anesthetized dogs. Circ Res 1989; 64:427-436.
24. Coghlan C, Prieto G, Harrison TR. Movement of the heart during the period between the onset of ventricular excitation

and the start of left ventricular ejection. Am Heart J 1961; 62: 65-82.

25.Carpentier A, Deloche A, Dauptain J, Soyer R, Blondeau P, Piwnica A, Dubost C, McGoon DC.
A new reconstructive operation for correction of mitral and tricuspid insufficiency. J Thorac
Cardiovasc Surg. 1971; 61: 1–13.

26. Unger-Graeber B, Lee RT, Sutton MS, Plappert M, Collins JJ, Cohn LH. Doppler echocardiographic comparison of the Carpentier and Duran annuloplasty rings versus no ring after mitral valve repair for mitral regurgitation. Am J Cardiol. 1991; 67: 517–519.

27.McCarthy JF, Cosgrove DM 3rd. Tricuspid valve repair with the Cosgrove-Edwards annuloplasty system. Ann Thorac Surg. 1997; 64: 267–268.

28. Jouan J, Pagel MR, Hiro ME, Lim KH, Lansac E, Duran CMG. Further information from Sonometric study of the normal tricuspid valve annulus in sheep: Geometric changes during the cardiac cycle. J Heart Valve Dis. 2004; 13:452-460.

Evaluation of Left Ventricular Function and Mass After Aortic Valve Replacement in Patients with Different Ejection Fraction

Noureldin Noaman Gwely, MD

Background: **Aortic valve replacement (AVR) is a safe and common operation for aortic regurge (AR) or aortic stenosis (AS) and it must be done prior to the development of left ventricular dysfunction to obtain a good result.**

The aim of this study is to evaluate the left ventricular function (LV function), left ventricular mass (LVM) and LVM index (LVMI) after AVR in patients with AR or AS, and to assess the regression of LVM and LVMI in patients with different ejection fraction (EF) to detect the best time for operation.

Methods: **This is a prospective study which consists of 2 groups, group A included 30 patients with AR and group B included 30 patients with AS. Careful history taking, clinical examination and echocardiography (including LVESD, LVEDD, EF, PG across the valve) were done preoperatively (preop) with calculation of LVM and LVM index. We also classified our patients into patients with poor EF (< 40%) included 16 patients, patients with moderate EF (41 – 54%) included 34 patients and patients with normal EF > 55% included 10 patients. Investigations were repeated early postoperatively (early postop) and after 6 months (6 m postop).**

Results: **There were highly significant difference in each group (P < 0.001) specially the changes of the mean NYHA functional class, LVESD, LVEDD, EF and PG across the aortic valve. Also LVM regressed significantly (P < 0.001) with improvement of the mean LVMI. Also in comparison between both groups, we found significant difference (P < 0.001) as regard parameters of LV function toward group B (AS) while the improvement of LVM and LVMI especially after 6 m postop were more toward group A (AR). There was marked improvement in patients with normal EF in whom the mean LVMI (gms/m2) improved from 230.15 ± 18.12 preop to 180.70 ± 16.52 early postop to 143.10 ± 12.11 after 6m postop and less improvement in patients with moderate EF in whom the mean LVMI (gms/m2) improved from 270.50 ± 13.15 preop to 227.31 ± 17.13 early postop to 188.51 ± 15.5 after 6m postop and the least improvement was detected in patients with poor EF in whom the mean LVMI (gms/m2) improved from 302.20 ± 21.19 preop. to 283.50 ± 19.22 early postop to 235.31 ± 20.15 after 6m postop. Mortalities were 4 patients 2 in both groups, but 3 of them were in patients with poor EF (3/16 = 18.8%), and 1 case with moderate EF 1/34 (2.9%). Morbidity in the form of severe LCOP syndrome occurred in 12/60 patients (20%), 8 patients with poor EF (8/16, 50%) and 4 patients with moderate EF (4/34, 12%) and non of them were with good EF.**

Conclusion: **We conclude that patients with AR or AS must be operated early before deterioration of LV function and LVM to obtain good result with good improvement of LV function and more regression of LVM and LVMI which improve the patient quality of life.Introduction:**

Address reprint request to : Dr Noureldin N Gwely MD
Department of Cardiothoracic Surgery , Mansoura University
Email : dr.noureldin_noaman@yahoo.com
Codex : 04/113/0906

Valvular lesions that imposes a progressive overload on the left ventricular muscle has an effect on the patient quality of life [1] .Progressive volume overload by AR can produce severe left ventricular dilatation and hence myocardial fibrosis and left ventricular dysfunction [2]. In contrast, pressure overload by AS can produce concentric hypertrophy and also myocardial fibrosis, failure or arrhythmia [3].

The potential for left ventricular hypertrophy regression and associated functional improvement may be the underlying mechanisms of results in general after AVR [3, 4].

The long asymptomatic course of AR and AS means that many patients have impaired LV function at diagnosis, and AVR in these patients, is associated with increased mortality and bad longterm results [5]. AVR has become a common surgical procedure for management of AR and AS. While the timing of surgery is critical and it is more clear that surgery must be done prior to the development of prolonged left ventricular dysfunction [6].

Now satisfactory indecis has been developed that allow the clinician to detect and avoid prolonged left ventricular dysfunction. Therefore, patients now undergo surgery sooner, resulting in reduced operative mortality and better long term survival with good left ventricular performance [7].

The aim of this study is to evaluate the left ventricular function and LVM & LVMI after AVR in patients with AR or AS and to assess the regression of LVM and LVMI which occurred after AVR in patients with different EF to detect the best time for operation to obtain good results of LV function and LVMI.

Methods:

This is a prospective study including 60 consecutive patients who had AR or AS and underwent AVR in the Cardiothoracic Surgery Department, Mansoura University Hospitals between January 2005 and December 2007.

These patients were divided into 2 groups, group A included 30 consequetive patients with AR for whom AVR was done, and group B included also 30 consecutive patients with AS for whom AVR was done.

Inclusion criteria included any adult patient with AR or AS for whom AVR was done and we excluded from this study any patient with mixed aortic valve lesions or with associated other valve disease or operations or endocarditis.

Preoperatively, careful history taking with detection of New York Heart Association (NYHA) functional class and clinical examination were done for all patients. Preoperative routine laboratory investigations with chest x-ray and ECG were also done routinely for all patients.

Careful transthoracic echocardiography was done for all patients to confirm the diagnosis, dimension of the left ventricle and parameter of left ventricular function especially ejection fraction (EF), left ventricular end systolic dimension (LVESD), left ventricular end diastolic dimension (LVEDD), and transvalvular pressure gradient (TVPG).

LVM was calculated and correlated to the body surface area (BSA) to get the LVMI as follows (8):

LVM (Gms) = 1.04 x [(LVEDd + IVSd + LVPwd)3 – (LVEDd)3]

LVM corrected tube formula = 0.8 (LVM) + 0.6

LVMI = LVM / BSA expressed in Gms/m2 where LVEDd = left ventricular end diastolic diameter in cm, IVSD = interventiruclar septal dimension in cm, LVPwd = left ventricular posterior wall dimension in cm.

For detection of the high risk for adverse events which may occur if the patients were operated upon late with poor LV function, we classified our patients according to Juraj et al. (9) into patients with poor EF (≤ 40%) included 16 patients, patients with moderate EF (41 – 54%) included 34 patients and patients with normal EF > 55% included 10 patients.

Operative technique, all patients were operated upon electively, the heart was approached through a classic standard median sternotomy in all patients with aortic and bicaval cannulation. Cardiopulmonary bypass (CPB) was done routinely using hypothermia (28ºC) with crystalloid cardioplagic solution injected directly into both coronary orifices after opening the aortic root. AVR

after excession of aortic leaflets was done for all patients using St-Jude bileaflet valve prosthesis and suturing of the valves were done by using interrupted Ticron 2/0. Closure of aortotomy with deairing by left atrial and aortic root venting were done. After beating of the heart, weaning from CPB with decannulation, then closure of the wound with drainage were done.

Postoperatively, all patients were transported to ICU with mechanical ventilation, inotropic support and vasodilators. All patients were examined and investigated as preoperative before discharge and after 6 months postoperatively to detect the change which occurred in LV function and LVM.

Statistical analysis: Data were analyzed using SPSS (Statistical Package for Social Sciences) version 10. Qualitative data were presented as number and percent. Quantitative data were tested for normality by Kolmogrov-Smirnov test. Normally distributed data were presented as mean ± SD. Paired t-test was used for comparison within groups. Student t-test was used to compare between two groups. P value < 0.05 was considered to be statistically significant and P value < 0.001 was considered to be statistically highly significant and P value > 0.05 was considered non significant.

Results:

The preoperative patients data are shown in table I.

Parameter	Group A (30) AR	Group B (30) AS
Age (mean) years	25.57 ± 3.96	24.73 ± 5.25
Sex:		
Male	19 (63.3%)	18 (60%)
Female	11 (36.7%)	12 (40%)
BSA (mean) m2	1.540 ± 0.125	1.557 ± 0.145
Symptomatology:		
Asymptomatic	3	2
Dyspnea	12	15
Angina	8	14
Syncope	5	13
NYHA class		
I	1 (3.4%)	2 (6.7%)
II	4 (13.3%)	4 (13.3%)
III	15 (50%)	16 (53.3%)
IV	10 (33.3%)	8 (26.7%)
Mean	3.13 ± 0.78	3.00 ± 0.83
Echocardiology (mean):		
LVESD (cm)	4.22 ± 0.11	3.96 ± 0.16
LVEDD (cm)	6.80 ± 0.22	5.87 ± 0.19
EF (%)	46.32 ± 3.72	45.77 ± 2.22
TVPG (mmHg)	43.07 ± 5.27	90.63 ± 7.39
LVM (gms)	434.90 ± 14.17	397.23 ± 10.97
LVMI (gms/m2)	249.94 ± 15.97	255.13 ± 7.56

Table (I): Preoperative data of the patients.

The intraoperative data are shown in table II.

Data	Group A (30) AR	Group B (30) AS
Aortic cross clamp time (mean) (min)	47.40 ± 4.50	55.13 ± 10.15
Cardiopulmonary bypass time (mean) (min)	65.50±4.88	75.57±9.97
Valve size (mm):		
19	–	8 (26.7%)
21	2 (6.7%)	13 (43.3%)
23	15 (50%)	6 (20%)
25	10 (33.3%)	3 (10%)
27	3 (10%)	–

Table (II): Intraoperative data.

The changes which occurred in the NYHA functional class postoperatively are shown in table III. Also the changes which occurred in the mean NYHA functional class and in the echocardiographic data in each group are shown in table IV and V, while the comparison between both groups is shown in table VI.

NYHA class	Group A (AR)						Group B (AS)					
	Preop		Early postop		6 m postop		Preop		Early postop		6 m postop	
	No	%	No	%	No	%	No	%	No	%	No	%
Class I	1	3.4	5	17.9	13	46.4	2	6.7	6	21.4	12	42.9
Class II	4	13.3	16	57.1	12	42.9	4	13.3	12	42.9	12	42.9
Class III	15	50	6	21.4	3	10.7	16	53.3	8	28.6	4	14.2
Class IV	10	33.3	1	3.6	-	-	8	26.7	2	7.1	-	-
Total	30	100	28	100	28	100	30	100	28	100	28	100

Table (III): Changes in NYHA functional class.

Data (mean)	Preop	Early postop	T1	P1 value	6 m postop	T2	P2 value
NYHA class	3.13±0.78	2.10±0.71	5.477	<0.001*	1.67±0.66	8.254	<0.001*
LVESD (cm)	4.22±0.11	4.12±0.18	5.298	<0.001*	3.92±0.02	9.681	<0.001*
LVEDD (cm)	6.8±0.22	6.09±0.31	11.018	<0.001*	5.71±0.19	17.626	<0.001*
EF (%)	46.32±3.72	54.21±3.11	15.315	<0.001*	58.12±4.11	21.325	<0.001*
TVPG (mmHg)	43.07±5.27	14.03±2.05	27.770	<0.001*	11.77±1.3	28.903	<0.001*
LVM (gms)	434.9±14.17	377.30±26.91	8.125	<0.001*	295.93±17.6	31.731	<0.001*
LVMI (gms/m2)	249.94±15.97	216.84±12.24	5.278	<0.001*	170.08±14.23	9.812	<0.001*

Table (IV): The changes in group A (AR).
T1, P1 difference between preop. and early postop.
T2, P2 difference between preop. and 6m postop.
**** P < 0.001, highly significant (HS).***

Data (mean)	Preop	Early postop	T1	P1 value	6 m postop	T2	P2 value
NYHA class	3.0±0.83	2.20±0.85	3.188	0.003*	1.73±0.69	5.917	<0.001*
LVESD (cm)	3.96±0.16	3.17±0.14	18.061	<0.001*	2.94±0.17	24.31	<0.001*
LVEDD (cm)	5.87±0.19	5.50±0.37	4.67	<0.001*	4.96±0.11	24.84	<0.001*
EF (%)	45.77±2.22	55.11±2.82	17.632	<0.001*	59.72±3.11	24.283	<0.001*
TVPG (mmHg)	90.63±7.39	19.33±1.39	52.18	<0.001*	13.76±1.10	52.39	<0.001*
LVM (gms)	397.23±10.97	372.83±8.36	10.67	<0.001*	314.10±10.95	35.46	<0.001*
LVMI (gms/m2)	255.13±7.56	239.45±5.21	8.32	<0.001*	201.73±8.32	32.31	<0.001*

Table (V): The changes in group B (AS).
T1, P1 difference between preop. and early postop.
T2, P2 difference between preop. and 6m postop.
**** P < 0.001, highly significant (HS).***

We found highly significant changes in each group as regard all parameters between the preoperative and postoperative data. Comparison occurred between these patients with different EF to detect the optimal time for operation after classification of these patients into three groups (according to Juraj et al. (9)) patients with poor EF (< 40%) included 16 patients, patients with moderate EF (41 – 54%) included 34 patients and patients with normal EF > 55% included 10 patients as shown in table VI.

From the above table, we found good improvement in all parameters in patients with good EF > 55% with less improvement in patients with moderate EF (41 – 54%) and the least improvement in LV function and the least regression of LVMI occurred in patients with poor EF < 40% with long ventilation and long ICU and hospital stay.

Morbidity in the form of low cardiac output syndrome (LCOP) which needed long ventilation and long ICU stay with inotropic support and vasodilators occurred in 12/60

Data (mean)	Patients with poor EF < 40%	Patients with moderate EF (41 – 54%)	Patients with good EF > 54%
No of patients	16	34	10
NYHA class preop	3.89±0.75	3.11±0.55	2.65±0.85
NYHA class early postop	2.88±0.28	2.05±0.11	1.78±0.35
P1 value	< 0.001*	< 0.001*	< 0.001*
NYHA class 6 m postop	2.02±0.11	1.88±0.23	1.31±0.21
P2 value	< 0.001*	< 0.001*	< 0.001*
ICU stay (days)	6.22±2.27	3.62±1.57	1.55±0.51
ventilation (hours)	39.41±5.22	8.35±2.1	3.72±1.78
hospital stay (days)	20.55±3.11	14.22±2.25	10.21±1.88
Preop LVESD (cm)	5.11±0.18	4.78±0.51	3.55±0.11
Early postop LVESD (cm)	4.98±0.72	3.87±0.35	2.79±0.07
P1 value	< 0.001*	< 0.001*	< 0.001*
6 m postop LVESD (cm)	4.02±0.32	2.97±0.11	2.54±0.17
P2 value	< 0.001*	< 0.001*	< 0.001*
Preop LVEDD (cm)	7.16±0.11	6.22±0.27	5.53±0.32
Early postop LVEDD (cm)	6.22±0.32	5.32±0.21	4.87±0.10
P1 value	< 0.001*	< 0.001*	< 0.001*
6 m postop LVEDD (cm)	5.72±0.21	4.81±0.23	3.92±0.12
P2 value	< 0.001*	< 0.001*	< 0.001*
Preop LVMI (gms/m2)	302.20±21.19	270.50±13.15	230.15±18.12
Early postop LVMI (gms/m2)	283.50±19.22	227.31±17.13	180.70±16.52
P1 value	< 0.001*	< 0.001*	< 0.001*
6 m postop LVMI (gms/m2)	235.31±20.15	188.51±15.5	143.10±12.11
P2 value	< 0.001*	< 0.001*	< 0.001*
Morbidity	8/16 (50%)	4/34 (12%)	0/10 (0%)
Mortality	3/16 (18.8%)	1/34 (2.9%)	0/10 (0%)

Table (VI): Improvement in patients with different EF.
P1 difference between preop. and early postop.
P2 difference between preop. and 6m postop.
**** P < 0.001, highly significant (HS).***

patients (20%), eight patients of them with poor EF < 40% (8/16 "50%") and 4 patients with moderate EF (4/34 "12%") and none of them with good EF.

Mortality were 4 patients (6.7%) 2 in both groups from severe uncontrolled LCOP and ventricular arrhythmia and death occurred within 3 – 6 days in ICU, three of them in patients with poor EF (18.8%) and one in patients with moderate EF (2.9%)

Discussion:

Aortic valve replacement for either AR or AS carries good results if done early, but carries high risk for adverse events and poor long term survival when associated with severe LV dysfunction [10].

In AS, the LV compensates for chronic pressure overload by hypertrophy in an attempt to normalize wall stress. Initially, EF and cardiac output are maintained. When wall stress exceeds the compensating mechanism, LV systolic function declines secondary to afterload mismatch, and the mean PG generated by the LV may be low despite the presence of severe AS. Thus, when LV dysfunction is due to afterload mismatch, as seen in severe AS, AVR results in improvement in symptoms and survival (8, 10, 11). LV dysfunction is a major prognostic indicator of the outcome of patients undergoing AVR for AS. AVR for AS decreases ventricular afterload, subsequent changes include adaptation and remodeling, with regression of hypertrophy and LV mass. EF, therefore, would be expected to improve after AVR in patients with reduced preoperative EF. Those who do not improve probably have fixed myocardial damage [10, 11, 12].

Patients with AR develop a compensatory large end-diastolic volume and eccentric cardiac hypertrophy. Chronic AR leads to both, LV pressure and volume overload, imposing a combination of increased preload and afterload on the ventricle. With a severe reduction in diastolic perfusion pressure, a decrease in diastolic coronary flow occurs and some patients may develop angina, especially because myocardial oxygen demand in these dilated hypertrophied ventricles may be very high [13]. The adaptive process leads to myocardial fibrosis, possibly as a result of myocardial ischemia. As aortic insufficiency continues, diastolic wall stress increases without a further increase in wall thickness, and myofibral slippage may develop. This process may be ongoing as a spiral event, leading potentially to an irreversible myocardial damage without the possibility of AVR. Recent long-term studies demonstrated that improvement of LV function after AVR in patients with chronic AR is related significantly to the early reduction in LV dilatation arising from correction of LV overload. In some patients with preoperative severely dilated left ventricles, ventricular dilatation will persist postoperatively [9, 14, 15].

Left ventricular hypertrophy is a well-known predictor of morbidity in hypertensive patients. Preoperative LV hypertrophy is also a negative factor in AVR. Several studies have documented the early and the late prognostic importance of a preoperatively increased LVM (16). On one hand, incomplete recovery of LV function and a lower late survival after AVR are frequently associated with residual hypertrophy. This fact might be due to excessively high initial hypertrophy with an incomplete postoperative reduction in LVM [17, 18].

Changes in LV function and LVM after AVR have been extensively studied during the past several decades. It is well established that, provided a significant pressure drop does not persist, hypertrophy regresses and function improves regardless of whether a biological or a mechanical valve substitute is used. Nevertheless, residual hypertrophy has proved to be an important determinant of long-term ventricular function, arrhythmias, and thus survival [19].

The mean age of our cases was 25.57 ± 3.96 and 24.73 ± 5.25 years in group A and B respectively which matched with El-Fiky et al. [20] which their patients had mean age 28 ± 11 years, because their study from our country and had the same etiology but differ from Marc et al. [21], Colleen et al. [22] and Christopher et al. [23] which the mean age of their patients was 63.3 ± 13.8, 59.7 ± 7.9 and 77 ± 4.9 years respectively because these patients from west countries with different etiologies.

In our study, all patients recorded improvement in the NYHA functional class with significant improvement in the life style of the patients, these improvement occurred from 3.13 ± 0.78 preop to 2.10 ± 0.71 early postop to 1.67 ± 0.66 after 6m postop in group A ($P < 0.001$) and from 3.00 ± 0.83 preop to 2.20 ± 0.85 early postop to 1.73 ± 0.69 after 6m postop in group B ($P < 0.001$), this matched with Giuseppe et al. (11) and Corti et al. (13) which in their studies there were improvement in the mean NYHA functional class from 2.5 ± 0.23 and 2.9 ± 0.80 preop to 1.4 ± 0.32 and 1.6 ± 0.43 postop. respectively.

Also we found marked improvement of all parameters of LV function especially the mean EF (%) which improved from 46.32 ± 3.72 and 45.77 ± 2.22 preop in group A and B to 54.21 ± 3.11 and 55.11 ± 2.82 early postop to 58.12 ± 4.11 and 59.72 ± 3.11 after 6 m postop respectively, these results matched with Juraj et al. (9), Giuseppe et al. (11), Corti et al. (13) and McCarthy (14) who showed improvement in left ventricular systolic and diastolic dimension and also the mean EF which in the study of Juraj et al. (9) increased from 45 ± 3% and 46 ± 2% in his both groups to 59 ± 3% and 64 ± 5% respectively. Also, Giuseppe et al. (11) showed that the mean EF increased from 29 ± 6% to 44 ± 10% and in the study of Corti et al. (13) and McCarthy (14) the mean EF increased from 44.8 ± 10% and 47.6 ± 15% to 54.7 ± 9% and 51.5 ± 17% respectively. Umesh et al. (10) stated that most of the studies (24, 25, 26, 27) have shown an increase in EF after AVR and this increase occurred within the first 6 months of surgery and sustained improvement occurred until 10 years, and he stated that these increase becomes more with good EF preoperatively, but with moderate preoperative EF, the increase became little after operation and became the least improvement with low preoperative EF as proved in these studies (24, 25, 26, 27) and this matched with our study as we found good improvement in the patients with good EF preop. and this improvement became little with moderate preop. EF and became least improvement with poor preop. EF.

We also reported marked fall in PG across the aortic valve in our study especially in group B with aortic stenosis in which the mean TVPG fall from 90.63 ± 7.39 mmHg preop to 19.33 ± 1.39 mmHg early postop to 13.76 ± 1.10 mmHg 6m postop this matched with Ogata et al. (28) and Frederic et al. (29) in which TVPG dropped from 84.31 and 53.5 to 18.76 and 15.2 mmHg respectively with improvement of the life style of the patients.

Also significant changes occurred in our study in the mean LVM (gms) from 434.90 ± 14.17 preop to 377.30 ± 26.91 early postop to 295.93 ± 17.6 gm at 6m postop in group A ($P < 0.001$) and from 397.23 ± 10.97 preop to 372.83 ± 8.36 early postop to 314.10 ± 10.95 gm at 6 m postop in group B ($P < 0.001$). These reflected on the mean LVMI (gms/m2) which improved from 249.94 ± 15.79 preop to 216.84 ± 12.24 early postop to 170.08 ± 14.23 at 6 m postop in group A and from 255.13 ± 7.56 preop to 239.45 ± 5.21 early postop to 201.73 ± 8.32 after 6 m postop in group B ($P < 0.001$).

These matched with other studies which showed marked decrease of LVM after AVR with higher LVMI regression compared with preoperative (12, 16, 17, 18), especially Oli et al. (12) who found regression of the mean LVMI from 200 ± 60 preop to 144 ± 42 (gms/m2) after 1 year. Umesh et al. (10) collected and reviewed the outcome of AVR and found that all the studies are in clear agreement for the regression of the LV mass after surgery and the range of regression remained between 12% to 43% (median value 20%). This agrees with our range of regression which was between 21% and 32% in group A and B respectively (median about 24%). Umesh et al. (10) made a further analysis of more studies (30, 31, 32, 33) that reported the LVMI in more than one allocated period after AVR. They showed a sharp fall in LVMI within the first 6 months of surgery and interestingly, these decrease did not substantially change after 6 months (10) and comparison between preoperative and postoperative follow up yielded a constant regression of LVM ($P < 0.05$) at all instances and indicated an constant decrease in LVMI after surgery and this matched with our results as comparison between preop. and postop. follow up yielded a constant regression of LVM ($P < 0.001$) with constant decrease of LVMI.

Juraj et al. (9) who classified the patients according to EF into poor, moderate and good EF, he found more morbidity in the form of severe LCOP syndrome with long ICU and hospital stays in poor EF patients than moderate EF patients with good postop. follow up in good EF patients. Also, we noticed that our patients with good EF > 54% preop. had good postop. course and this was reflected on ICU and hospital stay which became more stay in the poor EF patients with bad postop. course and moderate course and stay in moderate EF patients. So, morbidity in our study was more in poor EF patients which were 8/16 patients (50%) with severe LCOP syndrome versus 4/34 (12%) in moderate EF patients and none in good EF patients. This matched with other studies (24, 25, 26, 27) who noticed more morbidity in patients with low preop. EF than that with moderate preop. EF and nothing in patients with good preop. EF.

Mortality in our study was more in poor EF patients which were 3/16 patients 18.8% versus one patient with moderate EF patients from 34 (2.9%) with no mortality in good EF patients. Many surgeons (9, 13, 24, 25, 26, 27) faced the same poor results of this type of patients who had AVR with poor EF preoperatively with mortalities ranged from 4.5% to 21% but Umesh et al. (10) on their systematic review of the outcome of AVR stated

that although aortic stenosis patients with preoperative low EF and secondary cardiac diseases constitute a small subset and seem to have relatively higher surgical mortality, these patients should not be denied aortic valve replacement only on the grounds of low EF. The LV mass regression is an independent of age, sex, and types of valve substitutes. The clinical follow-up of these patients should specifically focus on the first 6 months postoperatively as the ventricles revert to their final size within this short but crucial period of time (10).

Conclusion:

We conclude that all patients with aortic valve disease benefited from AVR as regard improvement of all LV function parameters and also regression of the LVM with variable degrees from good to little improvement when they come late with severe left ventricular dysfunction.

So, we recommend that efforts must be taken for avoiding delaying of patients until late stages of aortic valve diseases, these efforts of explaining of the optimal timing of surgery must be directed to the physicians and patients.

References:

1. Durarte IG, Murphy CO, Koniski AS, Toues EL, Craver JM, Gott JP and Gugton RA. Late survival after valve operation in patients with left ventricular dysfunction. Ann Thorac Surg; 64: 1089-1095, 1997.
2. Markus R, Karin D, Tonny DT, Christoph S, Thomas W, Hans HS and Michael D. Aortic valve replacement for aortic regurgitation and stenosis in patients with severe left ventricular dysfunction. Eur J Cardiothorac Surg; 23: 703-709, 2003.
3. Lund D and Evlandson M. Changes in left ventricular function and mass during serial investigations after valve replacement for aortic stenosis. J Heart Valve Dis; 9(4): 583-593, 2000.
4. Bolycal C, Doganci S, Baysan O, Yildirim W and Tonny AB. Evaluation of left ventricular function after aortic valve replacement in a specific young male patient population with pure aortic insufficiency or aortic stenosis, 5 years follow up. Heart Surg Forum; 10(1): 57-63, 2007.
5. Chukunenek A, Rao V, Armstrong S, Ivanov J and David T. AVR a safe and durable operation in patients with impaired left ventricular systolic function. Eur J Cardiothorac Surg; 29: 123-138, 2006.
6. John S, Raukumon E, John CN and Boshic VV. 25 years experience with 456 combined mitral and aortic replacement for rheumatic heart diseases. Ann Thorac Surg; 69: 167-173, 2000.
7. Essop MR, Wisenbaugh J and Saroli P. Discordant changes in left ventricular performance after valve replacement for isolated rheumatic mitral regurgitation versus combined mitral and aortic regurgitation in ten years. Am J Cardiol; 23: 910-917, 1994.
8. Schiller NB. Two dimension Echocardiographic determination of left ventricular function and mass: summary and discussion of the 1989 recommendations of the American Society of Echocardiography. Circulation; 84(3): 1280-1287, 1991.
9. Juraj T, Thomas S, Burkhardt S and Marko T. Predictors of long term outcome after combined aortic and mitral valve surgery. Circulation; 100(II): 48-53, 1999.
10. Umesh C, Sharma MB, Paul B, Saraswati P, Willem RM, Yigal MP and Jos GM. Systemic review of the outcome of aortic valve replacement in patients with aortic stenosis. Ann Thorac Surg; 78: 90-95, 2004.
11. Giuseppe T, Paolo B, Roldaus S, Renato R, Gino G, Giambattista GM, Angelo R and Sobino I. Aortic valve replacement in severe aortic stenosis with left ventricular dysfunction, determinants of cardiac mortality and ventricular function recovery. Eur J Cardiothorac Surg; 24: 879-885, 2003.
12. Oli L, Kristian E, Inge D, Finn TJ and Christian P. Regression of left ventricular hypertrophy during 10 years after valve replacement for aortic stenosis is related to the preoperative risk profile. Eur Heart J; 24: 1437-1446, 2003.
13. Corti R, Binggeli C, Turinn M, Jenni R, Luscher TF and Turina J. Predictors of long term survival after valve replacement for chronic aortic regurgitation. Is M-mode echocardiography sufficient? Eur Heart J; 22: 866-873, 2001.
14. McCarthy PM. Aortic valve surgery in patients with left ventricular dysfunction. J Thorac Cardiovasc Surg; 14: 137-143, 2002.
15. Green GK and Miller DG. Continuing dilemmas concerning aortic valve replacement in patients with advanced left ventricular systolic dysfunction. J Heart Valve Dis; 6: 562-579, 1997.
16. Rafael GF, Jose A, Montero A, Oscan GA, Fernando HS, Sergio CL, Jose DS, Maria BC and Jose AB. Left ventricular mass index in aortic valve surgery, a new index for early valve replacement. Eur J Cardiothorac Surg; 23: 696-702, 2003.
17. Mehta RH, Bruckman D, Dos S, Russman P, Karavite D, Monaghan H, Sonnad S, Shea MJ, Engle KA and Deeb GM. Implications of increased left ventricular mass index on in-hospital outcomes in patients undergoing aortic valve surgery. J Thorac Cardiovasc Surg; 122: 919-928, 2001.
18. Natsuaki M, Itoh T, Ishida H, Humada M, Rikitake K and Naitoh K. Evaluation of postoperative cardiac function and long term results in patients after aortic valve replacement for aortic valve diseases with increased left ventricular mass. Jpn J Thorac Cardiovasc Surg; 48(12): 30-38, 2000.
19. Roldaus S, Christian N, Monica P, Giuseppe T and Sergio DV. Long term survival and functional results after aortic valve replacement in a symptomatic patients with chronic

severe aortic regurgitation and left ventricular dysfunction. J Am Coll Cardiol; 45: 1025-1030, 2005.

20. El-Fiky MM, El-Abbady M and Aboul Fettouh A. Predictors of LV improvement after aortic valve replacement for chronic aortic regurgitation with poor LV function: A comparative prospective study. J Egy Soc Cardiothorac Surg; 10(1): 157-168, 2002.
21. Marc R, Varun K, Joel P, Alexander K, Jan GB and Thierry GM. Natural history and predictors of outcome in patients with concomitant functional mitral regurgitation at the time of aortic valve replacement. Circulation; 114: 541-546, 2006.
22. Colleen CK, Forah KH, Fawzy GE, Floyd DL and Eygone HB. Impact of prosthesis-patient size on functional recovery after aortic valve replacement. Circulation; 111: 3221-3229, 2005.
23. Christopher JB, Nishont DP, Torin PF, Jason A, Pramod NB, Vincent C, Diane EA, Vincent L and William AB. Aortic valve replacement and concomitant mitral valve regurgitation in the elderly: Impact on survival and functional outcome. Circulation; 112: 443-447, 2005.
24. Connolly HM, Oh JK, Drszulok TA, Osborn SL, Roger VL and Hodge DO. Aortic valve replacement for aortic stenosis with severe left ventricular dysfunction. Prognostic indicators. Circulation; 95: 2395-2400, 1997.
25. Robiolio PA, Rigolin VH, Hearne SE, Baker WA, Kisslo KB and Pierce CH. Left ventricular performance improves late after aortic valve replacement in patients with aortic stenosis and reduced ejection fraction. Am J Cardiol; 76: 612-615, 1995.
26. Rediker DE, Boncher CA, Block PC, Akins CW, Buckly MJ and Fifer MA. Degree of reversibility of left ventricular systolic dysfunction after aortic valve replacement for isolated aortic valve stenosis. Am J Cardiol; 60: 112-118, 1987.
27. Connolly HM, Oh JK, Schaff HV, Roger VL, Osborn SL and Hodge DO. Severe aortic stenosis with low transvalvular gradient and severe left ventricular dysfunction, result of aortic valve replacement in 52 patients. Circulation; 101: 1940-1946, 2000.
28. Ogata T, Kamelo T, Obayoshi I, Ishikawa S and Harry SA. Aortic valve replacement for aortic stenosis with small mechanical prosthetic valve. J Card Surg; 17: 70-74, 2002.
29. Frederic VE, Denis B, Ismail EH, Ady B, Philipp D, Michel C, Louis PP, Jean CT and Michel P. Effect of aortic valve replacement for aortic stenosis on severity of mitral regurgitation. Ann Thorac Surg; 83: 1279-1284, 2007.
30. Monrad ES, Hess OM, Murakomi T, Nonogi H, Corin WJ and Krayenbuell HP. Time course of regression of left ventricular hypertrophy after aortic valve replacement. Circulation; 77: 1345-1355, 1988.
31. Villari B, Vassali G, Betocchi S, Briguori C, Chiriello M and Hess OM. Normalization of left ventricular nonuniformity late after valve replacement for aortic stenosis. Am J Cardiol; 78: 66-71, 1996.
32. Rajappan K, Melina G, Bellenger NG, Amrani M, Khaghani A and Pennell DJ. Evaluation of left ventricular function and mass after Medtronic free style versus homograft aortic root replacement using cardiovascular magnetic resonance. J Heart Valve Dis; 11: 60-85, 2002.
33. Gelsomino S, Frassani R, Morocutti G, Nucifora R, Dacol P and Minen G. Time course of left ventricular remodeling after stentless aortic valve replacement. Am Heart J; 142: 556-562, 2001.

Aortic valve replacement with and without aortic root enlargement in patients with small aortic annulus

Ashraf A. Esmat MD,
Ahmed El Nagar MD,
Hassan Elsisi MD,
Ahmed H. Gaafar MD
Mohamed Hagras MD.

Background: **The small aortic annulus is a major problem facing cardiac surgeons performing aortic valve replacement .this study was designed to compare the patients, in whom aortic valve was replaced using mechanical valve size 19 with those undergone aortic root enlargements using Nick's procedure, considering operative and postoperative results.**

Methods: **A prospective study carried out in Cairo University hospitals from January 2007 to October 2008, it included fifty patients randomized into two groups.**

Group A: **25 patients undergone aortic replacement with size 19 mechanical valve.**

Group B: **25 patients undergone aortic root enlargement with insertion of larger valves.**

Results: The two groups were comparable considering patients' demography, NYHA class, and pathology of the valve, LV function and dimensions. The aortic cross-clamp time and bypass time was higher in group B, the postoperative course was not significantly different in both groups, but the postoperative reduction in the gradient across the valve and the reduction in the LV dimensions were higher in group B than group A.

Conclusion: **Aortic root enlargement enables the insertion of larger prosthetic valve in patients who are expected to have prosthesis-patient mismatch that leads to reduction of postoperative gradient without increase in the operative risk.**

LV hypertrophy is a strong independent risk factor for mortality in patients undergoing aortic valve replacement (AVR) (1). Normalization of LV mass is therefore a crucial goal of AVR. Unfortunately, the extent of LV mass regression may vary extensively from one patient to the other and it is often incomplete. These findings underline the importance of identifying and, whenever possible, avoiding risk factors for persisting LV hypertrophy following valve replacement. (2)

The surgical management of the small aortic root accordingly remains a relevant topic. It is intuitive that one would elect to replace a stenotic valve (or for that matter a regurgitant valve) with the least stenotic prosthesis. Therefore, it is not surprising that a number of studies have demonstrated superior left ventricular mass regression,(3) postoperative functional class and exercise tolerance,(4) and patient survival(5) when small valves are avoided. Furthermore, prosthesis–patient mismatch (PPM) (6) specifically has been shown by some investigators to adversely affect left ventricular mass regression (7, 8) and both early and late survival. (9-12)

Address reprint request to :
Dr Ashraf A. Esmat Department of Cardiothoracic surgery ,Cairo University, Egypt
Email : jegyptscts@gamil.com
Codex : 04/114/0906

These arguments are complicated by various definitions of PPM ranging from an indexed effective orifice area (iEOA) of less than 0.6 cm2/m,(13) to less than 0.85 cm2/m2,(13,14) excessive transvalvular gradient immediately post implantation ,or increased transvalvular gradient with exercise.

Regardless of academic argument, the practicing surgeon has a number of options available when confronted with the small aortic root and a circumstance in which he wishes to implant a valve larger than the annulus readily accepts. Among those options is posterior aortic root enlargement (ARE).(15) We find unappealing the more complex alternatives promulgated today using stentless xenografts, homografts, or autografts as full root replacements, a procedure associated with an almost 3-fold higher operative risk than simple aortic valve replacement (AVR).(16)

Methods

This prospective study carried out in the cardiothoracic surgery department in Cairo University hospital and from January 2007 to October 2008, it included 50 patients with small aortic annulus undergoing aortic valve replacement. These patients were equally divided into two groups:

Group A: Aortic valve replacement with 19 mm prosthetic valve.

Group B: Aortic root enlargement using Nick's procedure to admit prosthetic valve of 21 mm or more.

The patients included in this study were selected from the eligible universe of patients; patients were matched in each group for all preoperative variables except for the surgical technique.

Inclusion Criteria

Patients with small aortic annulus relative to body mass index with aortic stenosis or double aortic valve lesion (AR/AS).

Exclusion Criteria

Associated coronary artery disease requiring coronary bypass.

Patients with aortic annulus 21mm or more measured by echocardiography.

Patients with body surface area (BSA)more than 1.9m2

Patients with other valve lesions or redo cases.

Patients were studied for the following main variables:

A-Preoperative variables:

1. Age & Sex
2. Body surface area.
3. Associated medical diseases.
4. Echocardiography to assess:
   - PWT & SWT
   - Gradient across LVOT.
   - Aortic valve area.

B- Intraoperative variables:

1. Type of operation.
2. Valve pathology.
3. Type of valve inserted.
4. Prosthetic valve size.
5. Aortic cross clamp time.
6. Cardiopulmonary bypass time.
7. Need for Inotropic support.
8. Intraoperative bleeding.

C- Postoperative variables:

a- ICU stay:
i. Haemodynamics of the patient.
ii. Ventilator Support.
iii. Blood loss.
iv. ICU stay.
b- Before discharge: Echocardiography to assess:
i. Gradient across LVOT.
ii. Function of the prosthetic valve.
iii. LV function and dimension.
c- 3-6 month's follow-up echocardiography to re-evaluate the same parameters done on discharge.

Surgical technique :

Incision; all patients underwent surgery through a full median sternotomy.

I-Group A patients:

- undergone aortic valve replacement in the usual classic technique cardiopulmonary bypass and cold blood cardioplegia to arrest the heart.

- The sutures used were 2/0 tycron, Teflon-packed taken into interrupted mattress manner, around 12-16 sutures.
- The type of the prosthetic valves inserted were Carbomedics in all patients.

II- Group B patients:

Aortic root enlargement done using the Nick's procedure:

The aortotomy is extended through the annulus in the midportion of the non-coronary cusp, until an acceptable valve sizer can be passed (1-2 sizes more than 19 mm).

The split is patched with a broad based pericardial patch through which the valve sutures in this area are passed.

The remainder of the patch is incorporated in the aortotomy closure which serves to widen the supravalvular region as well.

Postoperative care:

The patient were discharged to the ICU on:
Mechanical ventilation.
Inotropic support if needed (adrenaline infusion)
Follow-up for:
Haemodynamics.
Chest tube drainage.
Electrolyte and acid-base balance.
Urine output.

Statistical Analysis

Results are expressed as means ± standard deviation or number %.Comparison between group A and group B was done using unpaired t-test, while comparison between pre and postoperative values was done using paired t-test. Comparison between categorial variables was done using Chi square test. SPSS computer program (version 10 windows) was used for data analysis. P-values <0.05 was considered statistically significant and <0.001 was considered very highly significant.

Results :

Demographic Data:

In group A: there were 14 males and 11 females while in group B there were 12 males and 13 females.

In group A the age ranged from 20-48 years with a mean of 33.65+- 8.01 years while in group B age ranged 18-55 years with a mean of 37.1+- 12.01 years.(table 1)

	Group A	Group B	Significance
Age	33.65 ± 8.01	37.1±12.01	N.S.
Sex (F/M)	14/11 (60/40%)	13/12 (55/45%)	N.S.
Weight	62.8 ± 8.7	67.6 ± 10.3	N.S.
BSA	1.69 ± 0.43	1.71 ± 0.49	

Table (1): Demographic data
Preoperative clinical data
Patients were classified according to NYHA classification

Group A: 8 patients were class III (32 %) and 17 patients were class IV (68 %) Group B: 9 patients were class III (36 %) and 16 patients were class IV (64 %). (table 2)

NYHA	III	1v
Group A	8 (32 %)	17 (68 %)
Group B	9 (36 %)	16 (64 %)
	N.S.	N.S.

Table (2): Preoperative NYHA classification

Preoperative investigation

In addition to the routine preoperative laboratory investigations, all patients were subjected to M-mode, two-dimensional echocardiography. Standard apical, parasternal and subcostal views were obtained. The following parameters were measured and calculated:

Left ventricular end-diastolic and end-systolic diameters.

The posterior and septal wall thickness.

The aortic annulus and the peak gradient across the aortic valve.

As regard the group A patients the peak gradient across the aortic valve ranged from 68-120 mmHg with a mean of 91.1 ± 23.6 and the posterior wall thickness ranged from 1.3-1.8 cm with a mean of 1.5 ± 0.12 cm, while the septal wall thickness ranged from 1.4-1.8 cm with a mean of 1.4 ± 0.16 cm. the preoperative aortic annulus ranged from 17-21 mm with a mean of 18.4 ± 1.9 mm.

Patients of group B showed that the preoperative peak systolic gradient across the aortic valve of 70-130 mmHg with a mean of 98.7 ± 27.8 mmHg, and the posterior wall thickness ranged from 1.4-1.8 cm with a mean of 1.6 ± 0.13 cm, while the septal wall thickness ranged from 1.3-1.8 cm with a mean of 1.52 ± 0.25 cm. the preoperative aortic annulus ranged from 17-21 mm with a mean of 19.1± 2.1 m. (table 3)

	Group A	Group B	Significance
Mean systolic gradient	91.8 ± 23.6	98.7 ± 27.8	N.S.
PWT	1.58 ± 0.12	1.60 ± 0.13	N.S.
SWT	1.47 ± 0.16	1.52 ± 0.25	N.S.
LVESD	3.75 ± 0.65	3.25 ± 0.57	N.S.
LVEDD	6.15 ± 0.63	5.90 ± 0.58	N.S.
Preoperative aortic annulus	18.4 ± 1.90	19.1 ± 2.1	N.S.

Table (3): Preoperative Echo data

Operative data

In group A 25 patients had aortic valve replacement size 19 mm without doing any of the enlargement procedures.

In group B 25 patients had aortic valve replacement after doing aortic root enlargement using Nick's technique, the size of prosthetic aortic valves were 21 in 22 patients and 23 in 3 patients.(table 4)

Size	Group A	Group B
19	25 (100%)	0 %
21	- (0 %)	22 (88%)
23	- (0 %)	3 (12 %)

Table (4): Prosthetic Valve sizes

The aortic cross clamp time in group A ranged from 40-60 minutes mean (51.6 ± 8.7 min.) the cardiopulmonary bypass time ranged from 55-75 min. with a mean of (64.3 7.5 min.)

In group B the aortic cross clamp time ranged from 70-100 min. with a mean of (83.7± 10.8 min.) while the cardiopulmonary bypass time 85-115 min. with a mean of (103.2 ± 11.4 min.).(Table 5)

	Group A	Group B	Significance
Cross-clamp	51 ± 8.7	83.7±10.8	< 0.001
CPB	64.3 ± 7.5	103.2±11.4	<0.001

Table (5): Cross-Clamp time & Bypass time

ICU course

All patients in both groups required postoperative mechanical ventilation. In group A the ventilation time ranged from 2-6 hours with a mean of 3.6 ±1.2 hours

The blood drainage and blood transfusion required to keep the haematocrite around 25-30% was 120- 430 ml in the first 24 hours in group A with a range of 233.5 ± 80.1 ml,while in group B the blood loss was 180-1300 ml during the first 24 hours with a mean of 408.5 ± 288.66 ml.

No patients required reexploration in group A while 1 patient required in group B due to total blood loss of 1300 ml.in 6 hours.

The amount of blood transfusion in group A ranged from 0-3 units with a mean of 1.1±0.7 units while in group B ranged from 2-6 units with a mean of 3.8±1.4 units this shows that group required more blood with statistically significant difference.

The total ICU stay was comparable in both groups .in group A ranged from 24-48 hours with a mean of 33±12.4 hours, while in group B the range was 24- 72 hours with a mean of 39.7±14.7 hours. (Table 6)

	Group A	Group B	Significance
Ventilation (hours)	3.6±1.2	5.1 ± 2.78	<0.05
Blood loss (ml)	233±80.1	408.5±288.6	<0.05
Blood transfusion(units)	1.1±0.78	3.8±1.44	<0.001
ICU stay (hours)	33.4±12.4	39.7±14.7	N.S.

Table (6): ICU Parameters

Postoperative Course :

Mortality

We had no operative or early postoperative mortality in both groups.

Morbidity

In group A: 1 patient (4 %) suffered from sternal wound infection and mediastinitis, he required wound debridment and rewiring with use of pectoralis major advancement muscle flap and was discharged 2 weeks postoperatively.

In group B: 2 patients (8 %) one patient had atrial fibrillation in the early postoperative period that controlled by amiodarone intravenous infusion with restoration of the normal sinus rhythm, the other patient had superficial wound infection that controlled by repeated dressing and antibiotics.

Follow-up

Postoperative follow-up was done in the form of history taking and echocardiographic evaluation at a mean follow up of 4.5 months.

NYHA class improved in all patients in both groups.17 patients (68 %) in group A became in class I and 21 patients (84 %) in group B.

Post-Operative Echocardiography

In group A: the postoperative peak gradient across the aortic valve ranged from 25-42 mmHg with a mean of 33.5±8.7 mmHg, while in group B it ranged from 11-24 mmHg with a mean of 17.5±5.3 mmHg. With P < 0.001(table 7)

	Pre-operative	Post-operative	Significance
LVESD(cm)	3.75±0.65	3.61±0.67	N.S
LVEDD(cm)	6.15±0.63	5.87±0.33	N.S
PWT (cm)	1.58±0.12	1.43±0.14	<0.001
SWT (cm)	1.47±0.16	1.27±0.12	<0.001

Table (7): Comparison between preoperative and postoperative gradient

This shows that the aortic root enlargement procedure allows placement of a larger prosthesis resulting in significant decrease of the postoperative gradient across the valve.

For both groups there was no significant change in the left ventricular dimensions while the change in the posterior wall and septal wall thickness were significant (table 8 & 9

Group	Pre-operative	Post-operative	P-value
A	91 ± 23.6	33.5 ± 8.7	<0.001
B	98 ± 27.6	17.5 ± 5.3	<0.001

Table (8): Comparison between preoperative & postoperative lt.ventricular
Dimension & wall thickness in group A

	Pre-operative	Post-operative	Significance
LVESD (cm)	3.65 ± 0.57	3.45 ± 0.64	N.S.
LVEDD (cm)	5.90 ± 0.58	5.71 ± 0.23	N.S.
PWT (cm)	1.50 ± 0.13	1.30 ± 0.12	<0.001
SWT (cm)	1.52 ± 0.25	1.20 ± 0.14	<0.001

\

Table (9): Comparison between the preoperative & postoperative
Lt.ventricular dimension and wall thickness in group B

On comparing the percentage of reduction of the posterior wall and septal thickness between both groups we found it is higher in group A than group B but it is statistically non significant this can be explained by the short follow up period that does not allow detection of significant reduction of the left ventricular hypertrophy.(table 10)

	Group A	Group B	Significance
PWT	9.5 %	18.8 %	N.S.
SWT	13.6 %	21.1 %	N.S.

Table (10): Percentage reduction of PWT and SWT in both groups

Discussion :

Our data demonstrate that posteriors ARE can be accomplished during AVR without significantly increasing operative risk. Additionally, our findings confirm small valve size as an independent risk factor for operative mortality. Taken together, these findings support the continued value of this approach as an option when a surgeon wishes to implant a larger prosthesis

than the native aortic annulus would otherwise accept. These findings are consonant with those of other authors. Sommers and David (17) observed a statistically insignificant trend toward a higher mortality rate among patients undergoing ARE (7.1% vs. 3.5%); however, subsequent studies by both Castro and colleagues (18) and Kitamura and associates (19) reported mortality rates among patients undergoing ARE that were actually lower than those observed among patients undergoing isolated AVR (2.5% vs. 4.3% and 3.6% vs. 5.9%, respectively). In none of these studies did multivariate analysis identify ARE as a risk factor for operative death

Choice among the 3 common techniques of root enlargement can be dictated by individual surgeon experience, as well as complexity inherent to the procedure. The Konno–Rastan procedure (20, 21) offers the greatest degree of root enlargement. It is a complex procedure, however, requiring creation of a ventricular septal defect and right ventriculotomy, with double-patch closure of both. This risks damage to the septal arteries, as well as the conduction system. The posterior root enlargement techniques described by Nicks and colleagues (9) and Manouguian and Seybold-Epting(22) are more straightforward technically. Choice between Nicks' and Manouguian's enlargement will likely be largely dictated by the surgeon's preferred aortotomy, oblique or transverse, with the former enlargement representing an extension of the oblique and the latter an extension of the transverse approach(111).

In our study, there was a significant drop of the transvalvular gradient in the group of aortic root enlargement as compared to the other group. As regard the pattern of regression of the left ventricular mass, there was considerable but incomplete regression in both groups, more in group B, but no significant difference between the two groups, this might be due to the limited number of cases in our study and the short period of follow-up.

As reported in other studies, regression of LV hypertrophy was not complete in more than 50% of studied patients 1 to 2 years after operation. The normalization of LV mass is a complex phenomenon that is determined by several patient- and prosthesis-related factors. In this context, the incomplete regression of hypertrophy after the removal of the hypertrophic trigger may be explained by potentially irreversible changes in the hypertrophied myocytes and interstitium that may occur as a consequence of the long standing disease. (23-25)

Del Rizzo and coworkers (7) found a strong and independent relationship between the indexed EOA and the extent of LV mass regression following AVR. There was a mean decrease in LV mass of 23% in patients with an indexed EOA of more than 0.8 cm2/m2 compared with only 4.5% in those with an indexed EOA of equal to or less than 0.8 cm2/m2 ($p = 0.0001$). In contrast to these results, Hanayama and associates(26) found no significant relationship between PPM and regression of LV.

In a study done by Moon and coworkers (27) he found that PPM had a negative impact on survival of young patients but its impact on old patients was minimal. In addition, PPM negatively impact survival for average-size and for large patients.

Our data do not, however, address directly the issue of PPM, and accordingly, we cannot make statements based on our study results concerning this problem.

We conclude that ARE using the Nicks technique can be accomplished with low operative risk, and accordingly, surgeons should not be reluctant to do so when they believe it is otherwise clinically indicated.

References:

1. Orsinelli DA, Aurigemma GP, Battista S, Krendel S, Gaasch WH. Left ventricular hypertrophy and mortality after aortic valve replacement for aortic stenosis. A high risk subgroup identified by preoperative relative wall thickness. J Am Coll Cardiol. 1993;22:1679–1683
2. Mehta RH, Bruckman D, Das S, et al. Implications of increased left ventricular mass index on in-hospital outcomes in patients undergoing aortic valve surgery. J Thorac Cardiovasc Surg. 2001;122:919–928
3. Gonzalez-Juanatey JR, Garcia-Acuna JM, Vega Fernandez M, Amaro Cendon A, Castelo Fuentes V, Garcia-Begoechea JB,et al. Influence of the size of aortic valve prostheses on hemodynamics and change in left ventricular mass: implications for the surgical management of aortic stenosis. J Thorac Cardiovasc Surg 1996; 112:273-280.

Hirooka K, Kawazoe K, Kosakai Y, Sasako Y, Eishi K, Kito Y, et al. Prediction of postoperative exercise tolerance after aortic valve replacement. Ann Thorac Surg 1994; 58:1626-1630. Morris JJ, Schaff HV, Mullany CJ, Rastogi A, McGregor CG, Daly RC, et al. Determinants of survival and recovery of left ventricular function after aortic valve replacement. Ann Thorac Surg 1993;56:22-30 Rahimtoola SH. The problem of valve prosthesis–patient mismatch. Circulation 1978; 58:20-24.

Del Rizzo DF, Abdoh A, Cartier P, Doty D, Westaby S. Factors affecting left ventricular mass regression

after aortic valve replacement with stentless valves. Semin Thorac Cardiovasc Surg 1999;11:114-120

Tasca G, Brunelli F, Cirillo M, DallaTomba M, Mhagna Z, Troise G, et al. Impact of valve prosthesis–patient mismatch on left ventricular mass regression following aortic valve replacement. Ann Thorac Surg 2005;79:505-510

Nicks R, Cartmill T, Bernstein L. Hypoplasia of the aortic root. The problem of aortic valve replacement. Thorax 1970; 25:339-346.

Blais C, Dumesnil JG, Baillot R, Simard S, Doyle D, Pibarot P. Impact of valve prosthesis–patient mismatch on short-term mortality after aortic valve replacement. Circulation 2003;108:983-988

11. Tasca G, Mhagna Z, Perotti S, Centurini PB, Sabatini T, Amaducci A, et al. Impact of prosthesis–patient mismatch on cardiac events and midterm mortality after aortic valve replacement in patients with pure aortic stenosis. Circulation 2006; 113:570-576.

Mohty-Echahidi D, Malouf JF, Girard SE, Schaff HV, Grill DE, Enriquez-Sarano ME, et al. Impact of prosthesis–patient mismatch on long-term survival in patients with small St Jude Medical mechanical prostheses in the aortic position. Circulation 2006; 113:420-426.

Koch CG, Khandwala F, Estafanous FG, Loop FD, Blackstone EH. Impact of prosthesis-patient size on functional recovery after aortic valve replacement. Circulation 2005;111:3221-3229

Medalion B, Blackstone EH, Lytle BW, White J, Arnold JH, Cosgrove DM. Aortic valve replacement: is valve size important? J Thorac Cardiovasc Surg 2000; 119:963-974.

Dhareshmar J, Sundt TM, Dearani JA, Schaff HR, etal. Aortic root enlargement : what are the operative risks? J Thorac Cardiovasc Surg 2007; 134: 916-924. Rankin JS, Hammill BG, Ferguson Jr TB, Glower DD, O'Brien SM, DeLong ER, et al. Determinants of operative mortality in valvular heart surgery. J Thorac Cardiovasc Surg 2006;131:547-557

17. Sommers KE, David TE.Aortic valve replacement with patch enlargement of the aortic annulus. Ann Thorac Surg 1997; 63:1608-1612

Castro LJ, Arcidi Jr JM, Fisher AL, Gaudiani VA. Routine enlargement of the small aortic root: a preventive strategy to minimize mismatch. Ann Thorac Surg 2002; 74:31-36.

Kitamura M, Satoh M, Hachida M, Endo M, Hashimoto A, Koyanagi H. Aortic valve replacement in small aortic annulus with or without annular enlargement. J Heart Valve Dis 1996;5(suppl 3):S289-S293.

20. Konno S, Imai Y, Iida Y, Nakajima M, Tatsuno K. A new method for prosthetic valve replacement in congenital aortic stenosis associated with hypoplasia of the aortic valve ring. J Thorac Cardiovasc Surg 1975; 70:909-917.

21. Rastan H, Koncz J. Aortoventriculoplasty: a new technique for the treatment of left ventricular outflow tract obstruction. J Thorac Cardiovasc Surg 1976; 71:920-927

22. Manouguian S, Seybold-Epting W. Patch enlargement of the aortic valve ring by extending the aortic incision into the anterior mitral leaflet.New operative technique. J Thorac Cardiovasc Surg 1979;78:402-412

Tasca G, Brunelli F, Cirillo M, et al. Mass regression in aortic stenosis after valve replacement with small size pericardial bioprosthesis. Ann Thorac Surg. 2003;76:1107–111

De Paulis R, Sommariva L, Colagrande L, et al. Regression of left ventricular hypertrophy after aortic valve replacement for aortic stenosis with different valve substitutes. J Thorac Cardiovasc Surg. 1998;116:590–598

Lund O, Emmertsen K, Nielsen TT, et al. Impact of size mismatch and left ventricular function on performance of the St. Jude disc valve after aortic valve replacement. Ann Thorac Surg. 1997;63:122

Hanayama N, Christakis GT, Mallidi HR, Joyner CD, Fremes SE, Morgan CD, et al. Patient prosthesis mismatch is rare after aortic valve replacement: valve size may be irrelevant. Ann Thorac Surg. 2002;73:1822–18297–1234

Moon MR, Pasque MK, Munfach NA, Melby SJ: Prothesis-patient size : measurement and clinical implications. J Thorac Cardiovasc Surg, 2005; 129(5):961-965.

Aortic Root Enlargement; should we Hesitate Anymore?

Nezar El-Nahal(MD.),
Mohammad Abd Elaal(MD.),
Ayman Gabal(MD.),
Mahmoud M.Abd Rabo (MD.),
Mamdouh Sharawy (MD.).

Background: **Management of small aortic annulus presents a dilemma and technical challenge to surgeon during aortic valve replacement. Recent studies proved the dangerous effect of postoperative patient-prosthesis mismatch. Patch enlargement of aortic annulus is an effective option for management of such condition, but many surgeons are hesitant to use it due to old conception that it is associated with higher risks than aortic valve replacement. We compare early postoperative outcome of this procedure with that of isolated aortic valve replacement.**

Methods: **fifty two patients with small aortic annulus were included in this prospective study from July 2004 to July 2007, 22 patients had aortic valve replacement in addition to aortic root enlargement, and 30 patients had aortic valve replacement only. Preoperative, operative, and early postoperative data were recorded and statistically analyzed.**

Results: **there was no mortality in both groups, aortic root enlargement group had longer cross clamp and cardiopulmonary bypass time, higher chest tube drainage, but they had significantly higher indexed effective orifice area and better clinical improvement of functional status at 12 months follow up.**

Conclusion: **patch enlargement of the small aortic annulus at time of AVR is a safe, reproducible technique; it can be performed readily and without significant added risk relative to standard AVR. It permits insertion of a larger size valves with better indexed effective orifice area. It should be considered in selected patients especially young age and physically active adults to avoid postoperative patient–prosthesis mismatch.**

Key words: **aortic valve replacement, small aortic annulus, patch enlargement.**

Surgical management of small aortic root at time of aortic valve replacement (AVR) is one of the challenges which every cardiac surgeon had faced during his surgical practice. It has been discussed in the cardiac surgery literature more than 30 years ago (1).Patients with small aortic root especially those with large body surface area are at higher risk for patient-prosthesis mismatch (PPM). Many studies had documented the dangerous effects of PPM on clinical outcome after AVR (2).Nicks and associates proposed a posterior approach for aortic annular enlargement by extension of aortotomy through the non coronary sinus, across the aortic annulus as far the origin of anterior mitral leaflet(3). Manougian introduced extension of aortotomy through the commissure between left and non coronary cusps far into anterior mitral leaflet with opening of left atrium and closure of the defects by two patches(4). Konno-Rastan developed anterior enlargement through right coronary sinus extending into

Address reprint request to :
Dr Nezar El-Nahal MD.
Departmen t of Cardiothoracic surgery, Zagazig University Egypt
Email : jegyptscts@gamil.com
Codex : 04/115/0906

right ventricular outflow tract. Recently two-directional annular enlargement (anterior and posterior) has been reported (5).

Till now, there remains a general perception among many cardiac surgeons that aortic root enlargement (ARE) techniques are associated with more technical difficulties, higher risk for bleeding, and increased morbidity and mortality (6). This conception may be based on old reports at start of experience of surgeons with these techniques. In one of these reports, they had demonstrated up to twofold increase in perioperative deaths when ARE techniques were used (7).Results of recent studies which well documented the serious adverse effects of PPM on clinical outcome, combined with other reports supporting advantages, safety and feasibility of ARE techniques had led to increasing interest of surgeons in the modern cardiac surgery era in ARE techniques (8).

However, as reports are still conflicting regarding this issue, we planned this study for further assessment of safety and early clinical outcome of ARE procedure based on our center experience.

Methods:

This prospective, study included 52 patients who had undergone AVR which was indicated due to sever aortic stenosis in presence of small aortic root which was diagnosed preoperatively or during operation. Study was conducted at Cardiothoracic Surgery department at Zagazig university and King Fahad Cardiac center, King Saud University, Saudia Arabia, in the period from July 2004 to July 2007.Patients were divided into two groups. ARE Group which included 22 patients who had ARE technique in addition to aortic valve replacement with mechanical bileaflets aortic valve prosthesis. AVR Group which included 30 patients who had only aortic valve replacement with size 19 of the same valve prosthesis.

patients in need for combined mitral or tricuspid valve surgery, concomitant coronary bypass surgery, redo valve replacement, previous myocardial infarction, coagulopathy, renal failure were excluded from this study.

Preoperative data of the patients were collected including demographic data, NYHA class, symptoms, associated disease(s), body surface area (BSA), echocardiography data (ejection fraction, peak and mean pressure gradient, effective orifice area (EOA), and indexed effective orifice area (EOAi). Preoperative calculation of minimum accepted prosthetic valve size was based on patient's BSA to prevent PPM as defined by indexed effective orifice area (EOAi) of at least 0.85 cm2/m2.The actual effective orifice area of chosen prosthetic valve was supplied by manufacturing company and reviewed in published literatures.

median sternotomy was performed in all patients, after cardiopulmonary bypass (CPB) was instituted, patients were cooled systemically to30-32 O C,aorta cross clamped, then standard oblique aortotomy was used in all patients. myocardial protection was achieved by antegrade ,tepid (30-32C),blood ,intermittent cardioplegia administered directly into coronary ostia every 20 minutes for better myocardial protection. Native aortic valve was excised carefully with good debridement of the annulus. Annulus was sized with the supplied standard prosthetic valve sizer. At this stage ;decision was taken by the operating surgeon weather to enlarge the annulus ,or to perform standard AVR with size 19 valve (AVR group), taking into consideration patient's age, physical activity, comorbid conditions, BSA, calculated indexed effective orifice area of the prosthesis not to be less than 0.85 cm2/m2, feasibility of ARE according to operative conditions, anatomical, surgical findings (extensive calcification, fragile tissues,…etc) ,surgeon's judgment and comfort level .

In ARE group Nicks' technique with single patch was carried out by extending the aortotomy into the non-coronary sinus and through the annulus, to be stopped 2-4mm below the annulus.. Patch of fresh untreated autologus pericardium was used in all cases, care was taken during tailoring of the patch to adjust its width to be in suitable proportion to the length of incision below aortic annulus . Then the patch sutured by prolene 4/0 suture to the defect starting at its angle , buttressed with small Teflon pledget , then coming up with both limbs of the suture in continuous fashion up to about 1 cm above the annulus level. Again the annulus was resized to select the appropriate valve size which should be suitable to be inserted comfortably without force or excessive tension on sutures, but also large enough to prevent PPM which is the basic goal of the procedure.

Valve sutures were taken by classic everting horizontal mattress technique in the native annulus, but at patch area pledgted sutures were taken at the same level of annulus, with pledgets resting on outer surface of the patch. After the valve tied in place we extend the patch over the reminder of the open aortotomy and trim the patch to fit

comfortably.

Closure of the aortotomy was done very carefully. Hemostasis of sutures lines was carefully examined before declamping of the aorta , this could be done by administration of blood through cardioplegic cannula into the aortic root, if there was any leakage it was controlled before declamping the aorta . the rest of operation was conducted in the routine manner for AVR with special attention to bleeding at patch area. Cross clamp time and total bypass time were recorded.

Postoperative data were collected including mechanical ventilation time, duration of inotropic support, total amount of chest tubes drainage, number of transfused units of blood product, re-sternotomy for bleeding, and ICU stay. Hospitalization period and any complication were recorded.

After discharge of the patients, they were followed up in our out-patient clinic after 6, 12 months postoperatively. They were submitted to full clinical assessment and trans-thoracic echocardiography examination at each visit. Special attention was focused on functional status of the patients and presence of any postoperative complications. All echocardiographic data were collected with focus on peak and mean pressure gradient across the valve which had been calculated according to modified Bernoulli's equation. EOA was calculated according to the continuity equation .Indexed orifice area (EOAi) was obtained by dividing the EOA by the patient's body surface area

Statistical Analysis:

Data were analyzed using a statistical software package (Graph Pad In Stat® version 3.00 for Windows, Graph Pad Software Inc., San Diego, California, USA) and presented as mean (SD), or numbers as needed. The non parametric, paired t test was used to compare data, two-tail P values < 0.05 were considered significant. Chi square test or the fisher exact test were used to compare variables as appropriate.

Results :

preoperative patient characteristics were similar in both groupsThe mean age of the patients in ARE group was slightly younger than patients in AVR group (41.2±6.4 year versus 44.6±9.3 year ARE group versus AVR group, P=NS) .The BSA was higher in ARE group but not statistically significant (1.83±0.22m2versus 1.74±0.23 m2 ARE group versus AVR group, P=NS) table (1).

Characteristics	ARE Group	AVR Group	Pvalue
Age (Years)	41.2±6.4	44.6±9.3	NS
Sex (no) Male	12	16	NS
Female	10	14	NS
NYHA Class	2.5±0,3	2.6±0.6	NS
Etiology			
Rheumatic	19	26	NS
Bicuspid	3	4	NS
BSA	1.83±0.022	1.74±0.23	NS

Table 1: Preoperative Patient Characteristics
ARE= Aortic Root Enlargement, AVR= Aortic Valve Replacement, NYHA= New York Heart Association
BSA= Body Surface Area
Data were presented as mean and standard deviation or percentage.
P Value <0.05 is significant, P Value <0.01 is Highly Significant
NS= Non Significant

Operatively ,the average aortic cross clamp time was significantly prolonged in ARE group in comparison to AVR group (72± 18.4 min. versus46±12.8 min. ARE group versus AVR group, P =0.0001).Also, the total cardiopulmonary bypass time in ARE group was significantly longer than that of AVR group (91±14.9min. versus 60±10.4min. ARE group versus AVR group, P =0.0001) table (2). No intraoperative bleeding occurred in both groups, and weaning from cardiopulmonary bypass was easy in both groups.

Data	ARE Group	AVR Group	P Value
Cross clamp time (min)	72±18.4	46±12.8	0.0001
Cardiopulmonary Bypass Time (min)	91±14.9	60±10.4	0.0001

Table 2: Operative Data
ARE= Aortic Root Enlargement (n=22), AVR= Aortic Valve Replacement (n=30)
Data are presented as mean and standard deviation
P Value <0.05 is significant, P Value <0.01 is Highly Significant
NS= Non Significant

In intensive care unit (ICU) patients who had ARE required slightly longer mechanical ventilation time (9.13±3.2 hours versus 8.7±3.6hours ARE group

versus AVR group, P =NS). There were no statistically significant differences between both groups regarding duration of inotropic support, or ICU stay, but the total chest tubes drain was higher in ARE group (612±185ml. versus 542±140ml. ARE group versus AVR group, P =NS). Inspite of higher drainage in ARE group, the need for transfusion of blood products was similar in both groups (4.1±2.4 units versus 3.3±2.7 units ARE group versus AVR group, P =NS) (table 3).

Data	ARE Group	AVR Group	P Value
Ventilation Time (hours)	9.13±3.2	8.7±3.6	NS
Length Of Inotropic Support (hours)	13.4±7.9	11.2±5.3	NS
Total Chest Tube Drainage (ml)	612±185	542±140	NS
Transfused Blood Units	4.1±2.4	3.3±2.7	NS

Table 3: Intensive Care Unit Data
ARE= Aortic Root Enlargement (n=22), AVR= Aortic Valve Replacement (n=30)
Data are presented as mean and standard deviation
P Value <0.05 is significant, P Value <0.01 is Highly Significant
NS= Non Significant

Rate of reopening in immediate postoperative period due to bleeding was equivalent between both groups, one patient in ARE group had bleeding from aortotomy line just at the annulus level which was heavily calcific, it was controlled easily with single pledgeted stitch, afterward patient had routine uneventful postoperative course. Also one patient in AVR group was reopened for excessive drainage but no active source of bleeding could be identified, after hemostasis and closure he had unremarkable postoperative course .One patient in AVR group developed cerebral stroke diagnosed immediately postoperatively C-T brain showed small infracted area mostly due to extensive calcification of the aortic valve, he had marked improvement during his early postoperative course. Superficial sternotomy wound infection occurred in one patient of ARE group who was insulin-dependent diabetic, he responded well to conservative treatment in the form of antibiotic and frequent dressing. There were no in-hospital or 30 day mortality in both groups, also there were no reported mortality among all patients during follow up period of our study (table 4).

Complication	ARE Group	AVR Group
Re-Sternotomy For Bleeding (no.)	1	1
Cereberal Stroke (no.)	0	1
New Onset AF (no.)	2	3
Sternal Wound Infection (no.)	1	0
30 Days Mortality (no.)	0	0

Table 4: Postoperative Complications
ARE= Aortic Root Enlargement, AVR= Aortic Valve Replacement
AF= Atrial Fibrilation.

Hospitalization period was similar in both groups inspite of being slightly longer in ARE group (10±4.4 days versus 7.8±1.8 days ARE group versus AVR group, P=NS).

Clinical follow up at 6&12 months postoperatively showed clinical improvement of functional class of all patients without significant difference between both groups inspite of better improvement in ARE group (NYHA class1.1±0.2 versus 1.2±0.3 ARE group versus AVR group, P =NS). No patient in both groups was re-admitted to the hospital during follow up period due to heart failure or valve related complication. No mortality recorded in both groups during follow up period.

Echocardiography follow up examination (after 6 & 12 months postoperatively) showed significant reduction of trans-valvular pressure gradients (mean and peak) in both groups when compared to the preoperative gradients (table 5). The peak postoperative pressure gradient was significantly lower in ARE group when compared to AVR group (27.1±4.3mmhg versus32.4±11.3mmhg ARE group versus AVR group P =0.042),also the mean trans-valvular pressure gradient was significantly lower postoperatively in ARE group more than in AVR group (14.8±6.9mmhg versus 18.7±6.1mmhg ARE group versus AVR group, P =0.032). The indexed effective orifice area (EOAi) was significantly greater in ARE group than AVR group (0.88±0.17cm2/m2 versus 0.78± 0.15 cm2/m2 ARE group versus AVR group, P =0.029). No patient in ARE group had PPM, while 6 patients in AVR group had PPM with EOAi <0.85 cm2/m2. Ejection fraction improved in both groups when compared to preoperative value, but the improvement was slightly more in ARE group in comparison to AVR group, table (6). Follow up echocardiography did not show any prosthetic valve dysfunction, mitral regurgitation, or paravalvular leakage in any patient of both groups.

Echo Data	ARE Group			AVR Group		
	Preop.	Postop.	P Value	Preop.	Postop.	P Value
Peak Gradient (mmHg)	65±25.4	27.1±8.1	0.0001	64.1±27.3	32.1±15.1	0.0001
Mean Gradient (mmHg)	46.4±20.7	14.8±6.9	0.0001	41.1±19.3	17.8±8.3	0.0001
EF%	54.4±6.2	56.9±7.6	NS	55.3±4.9	57.2±6.1	NS

Table 5: Comparison of Preoperative and Postoperative Echocardiography Data
ARE= Aortic Root Enlargement (n=22), AVR= Aortic Valve Replacement (n=30)
EF %= Ejection Fraction
EOAi= Indexed Effective Orifice Area
Data are presented as mean and standard deviation
P Value <0.05 is significant, P Value <0.01 is Highly Significant
NS= Non Significant

Data	ARE Group	AVR Group	P Value
Peak Gradient (mmHg)	27.1±4.3	32.4±11.3	0.042
Mean Gradient (mmHg)	14.8±6.9	18.7±6.1	0.032
EF%	57.2±6.1	56.9±7.6	NS
EOAi (cm2/m2)	0.88±0.17	0.78±0.15	0.029

Table 6: Comparison of Postoperative Echocardiography Data
ARE= Aortic Root Enlargement (n=22), AVR= Aortic Valve Replacement (n=30)
EF %= Ejection Fraction
EOAi= Indexed Effective Orifice Area
Data are presented as mean and standard deviation
P Value <0.05 is significant, P Value <0.01 is Highly Significant
NS= Non Significant

Discussion :

Many surgical options had been developed to overcome the serious challenging problem of PPM after AVR in presence of small aortic annulus .These options include-beside ARE techniques- using the modern generations of mechanical valves with superior hemodynamic profiles, larger EOA, and reduced sewing cuff size. Also use of biologic valve (stentless bioprosthesis, homografts,and autografts) was another excellent option (9), however it is surgically demanding and not available in many institutions around the world. Even with all previous options the small aortic annulus may not enable the surgeon to simply insert a valve size enough to prevent development of PPM. In these circumstances one of various aortic root enlargement (ARE) techniques can be safely employed [10].

In the present study there was no operative or early postoperative mortality in both groups. Many investigators had reported similar mortality rates between patients who had AVR with or without ARE. Sakamoto and coworkers in their study proved equivalent 10 years actuarial survival rate for AVR patient with or without annular enlargement (11). Okuyama and his group reported that, the operative mortality and long term survival were similar between patients who had ARE technique during double valve replacement when compared with double valve replacement only. Also, other study (12) showed low mortality rate 2.9% after ARE procedures despite that more than half of patients had concomitant procedures. In a recent report from Mayo clinic, the raw operative mortality was higher in ARE group 5.6% versus 2.9% in AVR group (p=0.032), but the multivariate analysis of results proved that the advanced functional class, preoperative congestive heart failure and smaller valve size were the independent risk factors for operative mortality in this group, but not the addition of ARE technique. So, they supported the safety of ARE procedure and suggested that surgeons should not be reluctant to enlarge the small aortic root (13). As individual experience of the surgeon in ARE techniques had been suspected to be one of the risk factors of the technique .Peterson and associates had conducted study for this point ,they found that the individual surgeon's experience was not identified as an independent risk of mortality ,but the mortality was less with more experienced surgeon, also they observed lower operative mortality in their institute between more recent group of patient operated from 2001 to 2005 when compared to earlier group operated from1995 to 2000 suggested to be related to increased cumulative experience and comfort level of the surgeons with time [9].

Advantages and safety of ARE techniques had been suggested by many investigators, Kulik and co-workers reported that addition of ARE technique enabled the surgeons to insert larger size prosthesis with larger EOA leading to lower postoperative gradient and improved hemodynamics, however it did not improve long term clinical outcome after AVR (14).Controversy regarding better results in ARE patient may be based on selection bias of patients in some studies, as this procedure was generally applied to patients who were deemed to be good operative candidates based upon their preoperative and operative data, then compared to more critically patients where surgeons usually select simple AVR and accept the expected postoperative mismatch inspite of going through surgically demanding ARE procedure (6-7). However, Rammos and associates suggested that even in extremely difficult small annuli, addition of ARE was safe and had superior LV functional improvement [15].

The decision when to enlarge the small annulus should always be judged according to the clinical scenario in light of expected benefits and potential risks. As an example, patients with bad LV function had higher risk for mortality after AVR if an inadequately sized prosthetic valve used, so they will benefit from ARE technique despite the added time and potential risks (9). Also young patients with higher level of activity and motivation who are expected to have postoperative EOAi less than 0.85 cm2/m2after AVR will get greater improvement in their functional class when ARE technique was used (16).In the present study most of our patient were middle aged and active, as the most common etiology of valvular heart disease in Egypt –in contrast to western countries- is the rheumatic heart disease which classically affects patients at childhood and progress with time till valves become damaged and in need for surgical treatment by middle age when the patient is expected to have ; and also in need for his best physical activity during this productive stage of life. This can explain the importance of ARE technique especially in such group of patients in our country when PPM is anticipated.

On the other hand , older patients who are often less active, have more co-morbidities and shorter expected survival seem to be less benefited from ARE techniques. Many studies showed satisfactory long term outcome after implantation of small sized valve in elderly patients. Some investigators implanted prosthesis <21mm in patients older than 70 years , and during follow up period up to 16 years they reported acceptable hemodynamic performance without increased risk of death [17].The best example of such patients was old Japanese women studied by Matsuzaki and associates who had reported that 77% of females who had AVR over 5 year period of study were shorter than 150cm and 87% of them received size 19mm prosthesis with good outcome (18). Also Takaseya and co-worker had studied 11 Japanese women aged 70 year or more who had received size 17mm Regent prosthesis, they had no operative mortality , good clinical and echocardiographic postoperative data suggesting safety and satisfactory hemodynamic results of this small size prosthesis in that group of patients (19). Hashimoto preferred to use high performance small size prosthesis without ARE in elderly Japanese patients>65 year with small annulus and mean BSA 1.39+0.11m2 [20].

Some complications of ARE procedures had been attributed to some points in patch construction technique. Some investigators used a small teardrop-shaped patch of pericardium that is sutured only to base of aortotomy, this may be the culprit predisposing to dangerous bleeding as was their experience [7].Others used diamond shaped patch to close the gap in AML and aorta [21]. While Molina used two separate triangular shaped patches, one of Gore-tex to close the defect in AML and the second one of Dacron to close the aorta, both of them joined at a hinge point at level of annulus with a modified suturing technique resulting in perfect hemostasis , and pliable mobile AML [10].

In another series, the ivestigators used a section cutted from Hemashield tube graft ,they suggested that this material is more stronger than pericardium and more suitable due to its natural curved shape. They found it particularly helpful when facing friable or calcific tissues [8].

As some studies had suggested that MR complicated ARE technique is more linked to type of patch material more than extent of incision into AML, they reported that use of synthetic material may increase incidence of MR more than pericardial patch as it had poor mobility due to progressive stiffness and degeneration of synthetic material which was surprisingly more problematic than the well known degenerative changes of pericardium in form of shrinkage and sclerosing changes [21,22].

In our experience fresh autologus pericardium was excellent material for patch construction,it is easy to handle, already available, with good hemostatic properties and pliability. Also we found that use of small teflon pledgets at angles of suturing lines with proper selection of suture size according to condition of the tissues were very crucial points to prevent bleeding after procedure.

One of the most major concerns of the surgeons is operative and postoperative bleeding associated with ARE technique with subsequent need for blood transfusion or sternal re-opening for control of bleeding. In a recent study comparing outcome of 712 patients, there were no statistical difference between AVR or ARE groups as regard rate of postopertative re-sternotomy due to bleeding (14). Nakano and associates reported safety of ARE technique as they encountered no hemorrhage in there series of 33 patient (23). Rammos and associates reported similar chest tube drainage in in patients underwent AVR wether isolated or associated with ARE procedure (15). In our study, despite that the chest tube(s) drainage was slightly higher in ARE group, the need for replacement of loss by blood transfusion, or the rate of sternal re-opening for bleeding were not statistically different from that required in AVR group thus denoting the minimal impact of this higher drainage on the clinical outcome of the patients in ARE group.

CONCLUSION :

Patch enlargement of the small aortic annulus at time of AVR in the modern era is a safe, reproducible technique. Our early clinical outcome results support its use as it permits insertion of a larger size valves with better EOAi, it can be performed readily and without significant added risk relative to standard AVR. It should be considered in selected patients especially young and physically active adults to avoid postoperative patient–prosthesis mismatch.

References :

1-Nicks R, Cartmill T, Bernstein L. Hypoplasia of the aortic root. The problem of aortic valve replacement. Thorax 1970;25:339–46.

2-Walther T,Rastan A,Falk V,Lehmann S,Garbade J,Funkat A, Mohr F, Gummert F. Patient prosthesis mismatch affects short- and- long-term outcomes after aortic valve replacement. Eur J Cardiothorac Surg 2006;30:15-19.

3- Manouguian S, Abu-Aishah N, Neitzel J. Patch enlargement of the aortic and mitral valve rings with aortic and mitral double valve replacement. Experimental study. J Thorac

Cardiovasc Surg 1979;78:394–401.

4-Manougian S, Seybold-Epting W. Patch enlargement of the aortic valve ring by extending the aortic incision into the anterior mitral leaflet. J. Thorac Cardiovasc Surg 1979;78:402–412.

5-Otaki M, Oku H, Nakamoto S, Kitayama H, Ueda M, Matsumoto T. Two directional aortic annular enlargement for aortic valve replacement in the small aortic annulus. Ann Thorac Surg 1997;63:261–263.

6-Carrier M, Pellerin M, Perrault LP, et al. Experience with the 19-mm Carpentier-Edwards pericardial bioprosthesis in the elderly. Ann Thorac Surg 2001;71(Suppl):249 –52.

7-Sommers K, David T. Aortic valve replacement with patch enlargement of the aortic annulus. Ann Thorac Surg 1997;63:1608–12.

8-Castro L, Arcidi J Jr, Fisher A, Gaudiani V. Routine enlargement of the small aortic root: a preventive strategy to minimize mismatch. Ann Thorac Surg 2002;74:31– 6.

W, Mesana T. Late incidence and predictors of persistent or recurrent heart failure in patients with aortic prostheses. J Thorac Cardiovasc Surg 2004;127:149—59.

9-Peterson M, Borger M, Feindel C, David T. Aortic annular enlargement during aortic valve replacement: improving results with time. Ann Thorac Surg 2007;83:2044 –9.

10-Molina J. Enlargement of the aortic annulus using a double-patch technique: A safe and effective method. Ann Thorac Surg 2002;73:667-70.

11- Sakamoto Y, Hashimoto K, Okuyama H. Prevalence and avoidance of patient-prosthesis mismatch in aortic valve replacement in small adults. Ann Thorac Surg 2006;81:

1305–9.

12-Okuyama H, Hashimoto K, Kurosawa H, Tanaka K, Sakamoto Y, Shiratori K. Midterm results of Manouguian double valve replacement: comparison with standard double valve replacement. J Thorac Cardiovasc Surg 2005;129:869 –74.

13-Dhareshwar J, Sundt T, Dearani J, Schaff H, Cook D, Orszulak T. Aortic root enlargement: What are the operative risks?. J Thorac Cardiovasc Surg 2007;134:916-924.

14-Kulik A, Al-Saigh M, Chan V, Masters R, B'edard P,Lam B,Rubens F,Hendry P, Mesana T,Ruel M. Enlargement of the small aortic root during aortic valve replacement: Is there a benefit?. Ann Thorac Surg 2008;85:94-101.

15-Rammos K,Ketikoglou D,Koullias G, Tsomkopoulos S,Rammos C, Argyrakis N. The Nicks-Nunez posterior enlargement in the small aortic annulus: immediate – intermediate results. Interactive Cardiovasc thorac Surg 2006;5:749-754.

16-Hashimoto K. Patient-Prosthesis mismatch: The Japanese Experience. Ann Thorac Cardiovasc Surg 2006; 12:159-65.

17-Sawant D, Singh A, Feng W, Bert A, Rotenberg F. St. Jude Medical cardiac valves in small aortic roots: follow up to sixteen years. J Thorac Cardiovasc Surg 1997;113:499–509.

18-Matsuzaki K, Unno H,Konishi T. Left coronary ostial obstruction after aortic valve replacement with a supra-annular aortic valve. Jpn J Thorac Cardiovasc Surg 2006;54:199-202 .

19- Takaseya T, Kawara T, Tokunaga S, Kohno M,Oishi Y, Morita S. Aortic valve replacement with 17-mm St.Jude medical prostheses for a small aortic root in elderly patients. Ann Thorac Surg 2007;83:2050-3.

20- Hashimoto K. Patient-prosthesis mismatch: The Japanese experience. Ann Thorac Cardiovasc Surg 2006;12:159-65.

21- Eghtesady P, Hanley F. Posterior aortic annular enlargement for mechanical aortic valve replacement. Operative Tech Thorac Cardiovasc Surg 2002;7:181-187.

22-Imanaka K, Takamoto S, Furuse A. Mitral regurgitation late after manougian's annulusenlargement and aortic valve replacement. J Thorac Cardiovasc Surg 1998;115:727-729.

23- Nakano S, Matsuda H, Shimazaki Y, Taniguchi K, Kaneko M, Ueda T, Mori T, Kawashima Y.Eur J Cardio-thoracic Surg 1992;6:347-349.

Surgical Management of Aortic Coarctation: Experience Of 28 Cases

Sameh M. Amer MD,
Reda E. AL-Refaie MD,
Nour Eldin N. Gwely MD,
and Usama A. Hamza MD

Background: **The surgical repair of aortic coarctation has evolved over time. This study evaluates our experience in surgical management of coarctation of the aorta by different types of repair for detecting the main and common method for each age group .**

Methods: **Mansoura university hospital data was reviewed for patients underwent coarctation repair form 1995 to 2007; 28 patients were available, 13 patients below two years old (Group A) and 15 patients older (Group B).**

Results: **Of 28 patients, 18 (53.3%) were males with mean age of 6.9±8.7 years. In group A, the presenting symptoms were tachypnea (49%), palpitation (40%) disinterest in feeding (35%) and cyanosis (20%) with digoxin and diuretics use in 53.8% and 84.6% respectively while in group B, headache (60%), fatigue (50%) and palpitation (40%) and antihypertensive use (60%) were characteristic. Chest X-ray was suggestive for diagnosis in all patients. In group A; it showed left ventricular enlargement in 4(30.8%) patients and biventricular in 9(69.2%) patients with no other radiological findings while, in group B, it showed figure 3 sign in 1(6.6%) case, rib notching in 11 (73%) patients and left ventricular enlargement in all cases (P <0.001). Echocardiography was diagnostic in all cases. Mean pressure gradient across the coarctation area was 70.6±5.5mmHg in group A versus 72.5±7.5mmHg in group B.with no significant difference between the two groups. CT was done for 3 patients in group B and it showed narrowed aorta in some cuts. CT angiography was done for 7 patients in group B. MR angiography was done in 11 patients in group B. In group A:Resection with extended end to end anastmosis and subclavian flap aortoplasty were done in 7(53.8%) and 2(15.4%) patients respectively while resection with end to end anastmosis was done in 4(30.8%). Onlay patch aortoplasty and interposition tube graft were done only in Group B in 3(20%) and 6(40%) patients respectively. While resection with end to end anastmosis was done in 6(40%) patients. The mean postoperative systolic pressure gradient were 8.8±3.9 mmHg in group A versus 7.5±3.5mmHg in group B.with no significant difference between the two groups Postoperative morbidity in both groups included patch infection 1(3.5%), bleeding 2(7.1%), chylothorax 2(7.1%), left lower lobe collapse 1(3.5%), and right side hemiparesis 1(3.5%). Over all mortality rate was 3.5% only one case died from severe patch infection.**

Conclusion: **There are many surgical techniques for repair of coarctation of the aorta but resection with extended end to end anastmosis is the main method in infant and young children, while interposition tube graft is common in adults and older children.**

Address reprint request to :
Dr Noureldin N Gwely
Department of Cardiothoracic Surgery , Mansoura University
Email : dr.noureldin_noaman@yahoo.com

Codex : 04/116/0906

Coarctation of aorta (CoA) is a congenital narrowing of the aorta usually distal to the left subclavian artery adjacent to the site of insertion of the ductus arteriosus. Usually more than 50% narrowing is needed to be significant. [1, 2] From all congenital heart diseases , (CoA) represents 5-8% , it is three times more common in males than females in cases of isolated coarctation. In (CoA) associated with other cardiac anomalies the percentage is 1:1.[1, 2]

Clinical features of coarctation usually represents itself as heart failure after variable time, delay in onset of failure is related to PDA closure, degree to which collaterals develop and presence of major non cardiac anomalies.[1, 2,]

The first successful surgical repair of (CoA) was performed in Stockholm, Sweden by Craford and Nylin in 1944 by resection and end to end anastmosis [3], in 1957 Vossschulte introduced the concept of patch aortoplasty. For infants as an alternative to "resection with end to end anastmosis" the subclavian flap aortoplasty was first performed by Waldhausen and Nahrrwold in 1966 (1,2,4). The recently introduced technique of resection with extended end to end anastmosis appears to be associated with good intermediate term results. [3]

The aim of this study was to evaluate our experience in surgical management of coarctation of the aorta by different types of repair for detecting the best method for each age group.

Methods

We retrospectively reviewed the files of 28 consecutive patients with coarctation who were operated upon in the Department of Cardiothoracic Surgery, Mansoura university hospital in the period between January 1995 and January 2007 to analyze our experience in surgical management of coarctation of aorta. Patients were divided into two groups; Group A: includes patients less than 2 years old (13 cases) and Group B: includes older children and adults (15cases).

All patients with isolated coarctation of aorta or coarctation with non-obstructive bicuspid aortic valve were included while patients with associated other congenital cardiac anomalies were excluded except associated PDA.

Preoperative evaluation:

Detailed history for group (A) included abnormal feeding behavior, irritability, symptoms of lung congestion or symptoms of low cardiac output and for Group (B) included symptoms of hypertension, heart failure or complications. Past history of admission in ICU or neonatal ICU (NICU), history of drug intake (digitalis, diuretics, or antihypertensive medications), or history of previous coarctation repair was also collected.

The patients were subjected to full general examination with stress on: palpation of pulses in four limbs, measurement of blood pressure in four limbs, palpation of the back of the chest for the presence of collaterals, and presence or absence of other non cardiac congenital anomalies. All patients were subjected to routine laboratory investigations as complete blood picture, liver functions, renal functions, fasting blood sugar and ESR.

ECG and plain chest X-ray were done for all patients at admission and both pre and post-operative for cardiothoracic ratio, lung congestion, and rib notching, enlarged left mediastinal shadow "figure 3". Transthoracic ECHO was done for all patients to determine site and severity of coarctation, pressure gradient, LVH, RVH, biventricular enlargement, any associated lesion as presence of patent ductus arteriosus. CT and CT angiography (CTA) was done in 10 cases in group B while Magnetic resonant angiography, (MRA) was done in 11 patients in same group.

The operative techniques:

All patients in our study were operated upon using one of the following five techniques; resection with end to end anastmosis, subclavian flap aortoplasty, onlay patch aortoplasty, Dacron patch interposition, and resection with extended end to end anastmosis.

Invasive arterial cannula (commonly radial) was put in the right upper limb and another one was put in the lower limb (commonly in dorsalis pedis artery or femoral). If this was not available or difficult, non-invasive cuffs were applied to both right upper limb and lower limb.

Patients were positioned in right lateral decubitus and we used the left posterolateral muscle sparing thoracotomy through the fourth or the third intercostal space in all cases. After meticulous dissection and exposure of the coarcted area, adjacent parts of descending aorta, aortic

arch, patent ductus arteriosus if present, and left subclavian artery; oversewing and division or ligation of the ductus arteriosus when present or the ligamentum was done.

Resection with end to end anastmosis: Cross clamps were put proximally and distally to the coarcted area and time was calculated. Heparin was or was not given. All the coarcted area was removed then approximation of the two ends and anastmosis was done by continuous prolene 3/0, 4/0, 5/0 or 6/0 suture according to the size of aortic wall

Subclavian flap aortoplasty: Cross clamps were put proximally between the left common carotid artery and the left subclavian artery and distally on the descending aorta after the coarcted area. The left subclavian artery was ligated and divided then aortotomy was done along the lateral border of the left subclavian artery extending into the lateral border of the descending aorta passing through the coarcted area then the subclavian flap was folded to be sutured with the incision using prolene 5/0 to enlarge the coarcted area.

Onlay patch aortoplasty: Cross clamps were put proximally and distally to the coarcted area then longitudinal incision in the lateral border of the coarcted area was done then Dacron onlay patch was sutured to the edges of the incision using proline 4/0 to enlarge the coarcted area.

Dacron tube graft interposition: This was done by resection of the coarcted segment and the tube graft is interpositioned between the two ends and continuous sutures were done to anastmose the ends with the tube.

Resection with extended end to end anastmosis: This was done by more dissection of the coarcted segment and whole aortic arch up to the ascending aorta, lower parts of arch vessels and the descending aorta. The cross clamps were put proximally between the brachiocephalic artery and the left common carotid artery and distally after the coarcted area. After excision of the coarcted area, incision in the undersurface of the aortic arch and the appropriate counter-incision in the descending thoracic aorta were done. The descending aorta was freely mobilized of to be anastmosed with the distal end and the inferior border of the arch.

After the repair, cross clamps were removed, good haemostasis, closure of mediastinal pleura, insertion of drainage tubes, and closure of thoracotomy in layers were done.

Postoperative management:

All cases were thoroughly monitored by ECG, pulse oximetry, the blood pressure in upper and lower limbs, and intercostal tubes. Administration of vasodilator infusions to decrease postoperative hypertension was done then tapering of the dose gradually and replaced by antihypertensive medications if needed. Other routine postoperative care was done including the administration of inotropic drugs if indicated and closely monitoring and prediction of complications especially postoperative bleeding.

Follow up:

The patients were submitted to pre-discharge complete physical examination, ECG, chest X-ray and routine laboratory investigations. The patients were followed up at our outpatient clinic in early visits then with cardiology (pediatric or adult) later on.. All patients were subjected to postoperative Echo during our period of follow up which ranged from 6-48 months (mean 28±6.7 months) and their reports were compared with preoperative reports. There was no recorded recurrence at our follow up period.

Statistics:

Our data were subjected to the following statistical non-parametric tests: Wilcoxson signed rank test, Mann-whitney test, Mc nemmar test, Non parametric correlation coefficient test. The probability P value for the calculated values with a degree of freedom was calculated using certain tables with a level of significance less than 0.050.

Results

Of the 28 patients with coarctation who were operated on during the study period, there were 18(53.3%) male and 10(46.7%) female (P = 0.254). The mean age of our patients was 6.9±8.7 years . The mean age for group A (13 cases) was 6.2±4.7 months versus 150.3±103.7 months for group B(15 cases)(P <o.oo1).The mean weight at operation in group A was 5 kg while in group B the mean weight was 21.5 kg.(Table 1).

In group A the main presenting symptoms were tachypnea (49%), palpitation (40%), poor in feeding (35%), and cyanosis (20%) while in group B the presenting symptoms were those of hypertension in most of patients (60%); headache, vertigo, epistaxis; fatigue (50%), and palpitation (40%) as shown in Fig (1 & 2). There were 3 infants in group A presented with acute heart failure and one was on inotropic support in NICU, the other 2 were controlled with digoxin and diuretics. In group B, there were 2 patients presented with complication

of untreated coarctation includes; right side cerebral infarction resulting in left side hemiparesis, while the other patient presented accidentally with post traumatic chest wall large hematoma and during its exploration there was many large blood vessels so Echo was done and it showed coarctation.

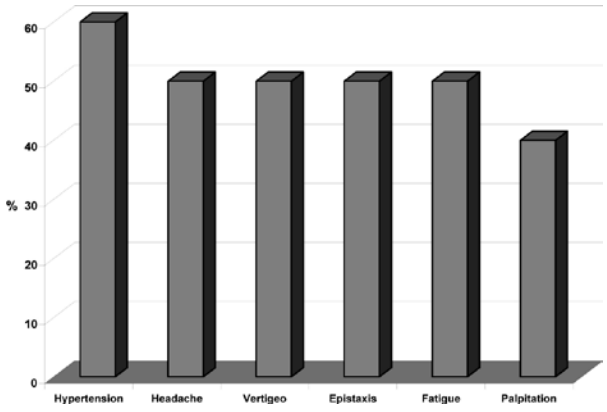


Fig (2): Symptomatology of group B.

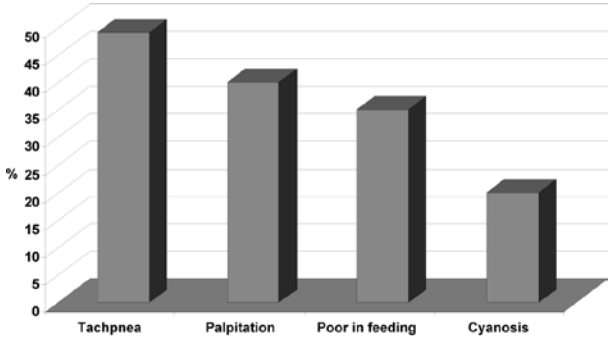


Fig (1): Symptomatology of group A.

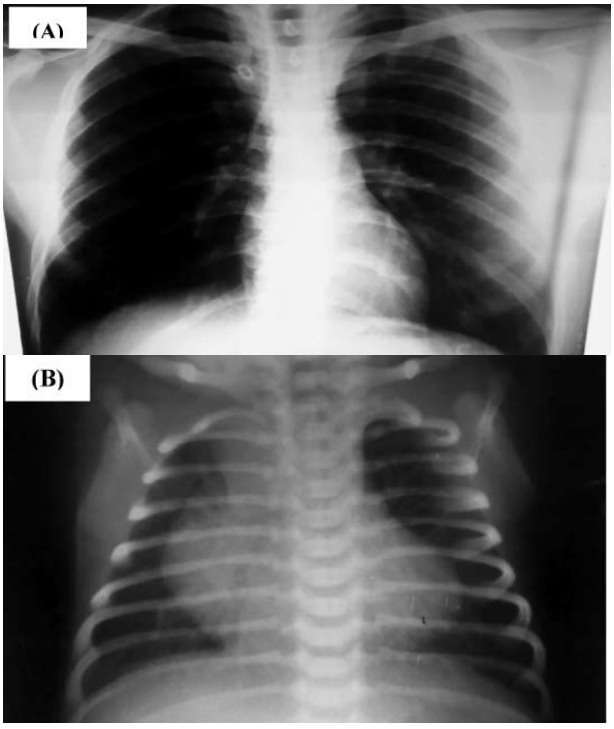


Figure 3 chest x-ray of aortic coarctation with (A) Rib notching. (B) Biventricular enlargement.

Trans-thoracic Echo was done for all patients and it was diagnostic in all patients, PDA was found in 10 patients which were ligated in both groups. Pressure gradient in coarctation area ranged from 35 to 140 mmHg with a mean pressure gradient of about 70.6±5.5mmHg in group A versus 72.5±7.5mmHg in group B.with no significant difference between the two groups.. In all cases, there was turbulence of flow across coarctation area with lack of pulsatility in the descending aorta.. Table (1), Figure (4)

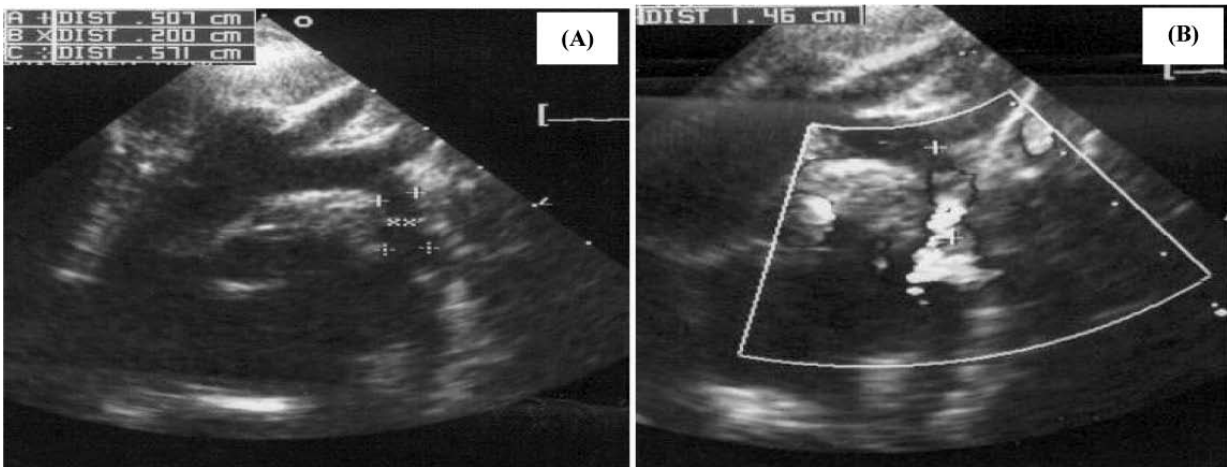


Figure (4) Echocardiography of aortic coarctation showing the diameter and length of coarctation area (A) & (B) respectively.

Data		Group A (n=13) No (%)	Group B (n=15) No (%)	Total (n=28) No (%)	P value
Age	Age (month)	6.2±4.7	150.3±103.7		0.000
Weight	Weight (kg)	5(2.5-11)	21.5(13-65)		0.003
Sex	Male	10(76.9)	8(53.3)	18(63.3)	0.254
	Female	3(23.1)	7(46.7)	10(35.7)	
CXR	Figure 3 sign	0(0)	1(6.6)	1 (3.6)	
	Rib notching	0(0)	11(73.3)	11(39.3)	
	LVE	4(30.8)	15(100)	19(67.9)	0.000
	BE	9(69.2)	0(0)	9(32.1)	
ECHO	PDA	8 (61.5)	2(13.3)	10(35.7)	0.007
	LVE	6(46.2)	14(93.3)	20(71.4)	0.004
	BE	7(53.8)	0(0)	7 (25)	0.049
	Average	0(0)	1(6.7)	1 (3.6)	0.14
preoperative medications	Digitalis	7(53.8)	3(20)	10(35.7)	0.066
	Diuretics	11(84.6)	8(53.3)	19(67.9)	0.082
	antihypertensive	2(15.4)	9(60)	11(39.3)	0.015
Operations	REEA	4 (30.8)	6 (40)	10(35.7)	
	SFA	2 (15.4)	0 (0)	2 (7.1)	
	On lay patch	0(0)	3 (20)	3 (10.7)	0.001
	Tube graft	0 (0)	6 (40)	6 (21.4)	
	REEEA	7 (53.8)	0 (0)	7 (25)	

Table (1): Showing demographic, diagnostic and operative data in each group:
LVE; left ventricular enlargement, BE; biventricular enlargement, PDA; Patent ductus arteriosus, CXR; chest X-Ray, AAH; aortic arch hypoplasia, Bicuspid AV; aortic valve, REEA; resection with end to end anastmosis, SFA; subclavian flap aortoplasty, REEEA; resection with extended end to end anastmosis, Echo; echocardiography.

In group B, CT was done for 3 patients and it showed narrowed aorta in some cuts. CT angiography was done for 7 patients and it was useful in giving an idea about the length of the coarctation area, the flow distal to it and it described the anatomy of the arch and gave a good idea about the development of collaterals (Figure 5-A).

MRA was done in 11 patients in group B, it gave a more illustrated idea about anatomy and collaterals (Figure 5-B).

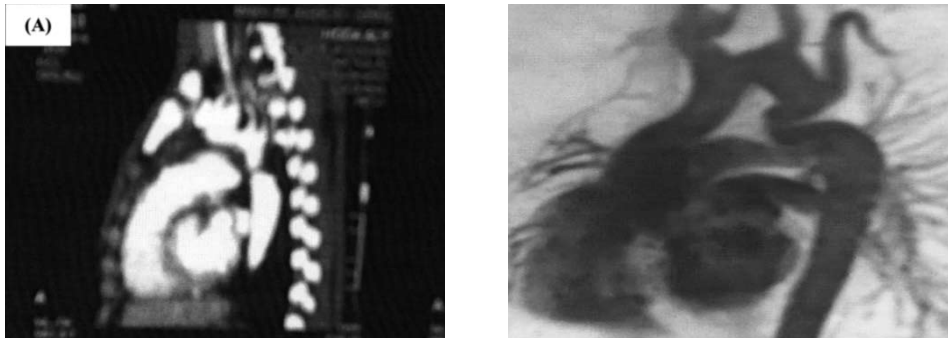


Figure (5) aortic coarctation (A) CT angiography (B) MRA of coarctation with extensive collaterals

Resection with extended end to end anastmosis and subclavian flap aortoplasty were only done in group A in 7(53.8%) and 2(15.4%) patients respectively while onlay patch aortoplasty and interposition tube graft were done only in group B in 3(20%) and 6(40%) patients respectively. Resection with end to end anastmosis was done in 4(30.8%) in group A compared with 6(40%) patients in group B (P = 0.001) (Table (1).

The mean cross clamp time in our study was 26.29±3.67 min. There was no statistically significance difference in cross clamp time in different types of operations as the difference in surgical time was found in time of dissection before cross clamp application. Table (2)

	Cross clamp time Mean ±SD	Nitrocine N (%)	Nipride N (%)
REEA	26.1±3.93	9 (36)	2 (22.2)
SFA	25.21±2.47	2 (8)	1 (11.1)
Onlay	30.0±2.10	3 (12)	1 (11.1)
patch	26.83±4.12	6 (24)	3 (33.3)
Tube graft	25.29±3.40	5 (20)	2 (22.2)
REEEA	26.29±3.67	25(89.3)	9(32.1)
Total			

Table (2): Cross clamp time and postoperative use of vasodilator in each procedure

ICU stay period ranged from 3 days to 25 days with a median of 5 days in both groups. Only 2 patients required mechanical ventilation; one of them(group B) due to delayed recovery from anesthesia and the other was preterm infant (group A) and was on ventilator before surgery that continued after operation. One patient (group B) needed inotropic support (dopamine). Vasodilators; nipride and nitroglycerine were used to control postoperative hypertension in 25(89.3%) and 9(32.1%) respectively Table (2). The chest drains were removed after 2-5 days with mean of 2.7 days. Immediately postoperative, the femoral pulse could be felt well and the dorsalis pedis pulse could be felt, later on both became bounding. The upper to lower limb pressure gradient became less than 18 mmHg postoperatively. Fifty percent of our cases showed no significant change in cardiothoracic ratio postoperatively but all were asymptomatic with no upper to lower limb pressure gradient.

There was one case who died from postoperative patch infection (3.5%) from group A. Two patients suffered from postoperative bleeding (7.1%); one of them from anastmosis line (group A) and the other from the chest wall (group B) and both were explored and controlled. Two patients (7.1%) suffered from postoperative chylothorax (group B) and they were managed conservatively. One patient (3.5%) suffered from left lower lobe collapse (group A) and managed by bronchoscopic suction and another patient (3.5%) was complicated by right side hemiparesis (group B) which improved with physiotherapy..

The mean postoperative systolic pressure gradient were 8.8±3.9 mmHg in group A versus 7.5±3.5mmHg in group B.with no significant difference between the two groups

Discussion

Coarctation of the aorta is a congenital heart disease, which represents 5-8% of congenital heart diseases (1).

In our series, we collected 28 cases in 12 years which represents a relatively small number of cases in comparison with others (1,2.5) but this can be explained by the fact that our department was not a specialized center for congenital cardiac surgery and this number is parallel to the number of congenital cardiac lesions operated. This was clarified in the last two years as the operated cases of CoA were 10 cases representing about 35% of cases. This advance was a part from our increasing interest in congenital cardiac surgery during the last two years.

The age of our patients ranged from 10 days to 35 years with a mean age of about 6.9 years. In Group A the age ranged from 10 days to 1.5 years with mean age of about 6.3 month while in group B the age ranged from 2.5 years to 35 years with mean age of about 12.5 years. **Seirafi** and colleagues reported a nearly similar age range from 1 day to 33 years with mean age of 11 month (6). Some authors studied only neonatal coarctation with mean ages of 10.3 and 13 days respectively (7, 8) while others extended the maximum age of their patients beyond the neonatal to infantile period with mean age of 13.5, 30 days, respectively (5,9). On the other hand Carr and colleagues studied coarctation in adolescent and young adult ranging from 11-53 years with mean age of 21 years (10) **while Aris and associates** collected patients 51-73 years old with mean age of 58 years (11).

In spite of the relative delay in the diagnosis in our

series there were 3 neonatal cases; two of them were in the last two years and they were operated upon electively while the other case was diagnosed and operated emergently because of developing heart failure.

In our series the mean weight at operation in group A was 5 kg while in group B the mean weight was 21.5 kg. The mean weight at operation in Wood and associates collection was 3.7 kg ranging from 0.85 to10.5 kg (9). The difference in mean weight at operation between our series and others is because of their great advance in intensive care management of neonates and infants with aortic coarctation especially in the use of prostaglandin E1 to maintain ductal patency and distal body perfusion.

In our study coarctation was more prevalent in males with male to female ratio of 1.8:1; this was confirmed by many studies (7, 12**).**

In group A, 80% of cases were symptomatic; the main presenting symptoms were tachypnea, abnormal feeding behavior, and cyanosis. Weak or absent femoral pulse were found in all cases and upper extremity hypertension was found in 80% of cases; this matches with the results reported by Zehr and his colleagues (13). Heart failure was the presenting symptom in about 23% of cases of group A; that was similar to Fesseha report (7) while 49% of Wright and colleagues patients presented with shock (14).

In group B, the main presenting symptoms were those of hypertension including headache, fatigue, vertigo, and palpitation; which was the cause of surgical referral. Preoperative complication was seen only in one patient in the form of cerebrovascular accident. The same findings reported by Beur and his associates where all their patients presented with hypertension (15) while in Carr and colleagues series, 78% of cases presented with hypertension and decreased femoral pulse one patient presented with right sided hemiparesis (10). In Wells and colleagues adults' series, the most common reason for surgical referral was recent discovery of hypertension and the most common symptoms were headache, fatigue, claudication, and heart failure (16).

Chest X-ray was done to all of our patients and it showed left ventricular enlargement, biventricular enlargement, and figure 3 sign. Rib notching was found in 11 (73%) cases of group B that represents 39.2% of all cases; this finding matches with **Bouchart and** colleagues (12).

In our series Echocardiography was done for all cases and this goes with many authors (7, 12, 17). While in Carr and colleagues series echo was done only for 67% of cases (10). It was diagnostic to all cases in our series and this was similar to the series of Fesseha, Elkerdany, and Sudarshan and their colleagues (7, 17, 18); While it was diagnostic only in 17% of Carr and colleagues series (10).

PDA was seen in 35.7% in our series; that goes with the series of Dave and colleagues in which it was associated with 35.7% (19) and differs from the series of Zehr and colleagues where it was associated with 57.5%of all patients (13).

Left ventricular enlargement in Echo was found in 71.4 % of cases and that was near that of Bouchart and colleagues series in which it was associated with 82% of all cases (12).

Mean preoperative echocardiography pressure gradient in our series was 70.6 mmHg and 72.5 mmHg that goes with series of Bouchart and Elkerdany and their colleagues where the mean gradient was 69 and 65.7 mmHg respectively (12, 17) while it was different from the mean gradient reported by Carr and colleagues which was 51 mmHg (10).

CT was done for 10 (35.7%) of our patients. In 7 cases, it showed collaterals and was useful to show the coarcted area. In the other two cases, it showed the short coarcted area with PDA.

MRA was done for 11(39.3%) of our patients. It showed the coarcted area, the anatomy of the arch and development of collaterals and it was useful in diagnosis, in determining the aortic arch size, diameter and the presence or absence of PDA. MRA was performed only in one case in the series of Carr and associates (10).

Digoxin was used preoperatively in 10 cases (7 (53%) in group A) and diuretics were used in 19 patients (11(84%) in group A); these results goes with the series of **Zehr** and colleagues which were on infants and neonates, where preoperative Digoxin and diuretics were the mainstay of therapy (13). Preoperative antihypertensive medications were used in 11 cases; 9 (60%) patients in group B; this goes with series of Carr and colleagues

which was on adult cases where about 50% of cases were taking antihypertensive medications (10).

The surgical approach was a left posterolateral thoracotomy through the third or the fourth intercostals space in all our patients; this was the same approach of Sudarshan and Dave and their colleagues (18, 19). Median sternotomy was the approach of surgery in series done by Elgamal and his colleagues (20).

In our series we had performed five types of operations starting with resection and end to end anastmosis in 10 (35.7% of all cases), subclavian flap aortoplasty in 2 (7.1%), onlay patch aortoplasty in 3 (10.7% of all cases). Interposition tube graft 6 (21.4% of all cases), and resection with extended end to end anastmosis in 7 (25% of all cases) which became our preferable operation during the last two years. In group A, we have performed three types of operations; subclavian flap aortoplasty at the beginning of our series and resection with end to end anastmosis then during the last two years all cases of group A were subjected to resection with extended end to end anastmosis while in group B, resection with end to end anastmosis and interposition tube graft had been performed equally while onlay patch aortoplasty was used only at the beginning of the series.

In Carr and colleagues series 18 (40%) patients had either Dacron or Gore-Tex patch enlargements, (22%) had resection with an end-to-end anastmosis, (20%) had interposition Dacron grafts, and (18%) had subclavian flap repairs (10). In Sudarshan and colleagues series, 37.5% underwent subclavian flap angioplasty, one baby had a carotid flap angioplasty, and another had a patch repair using pulmonary homograft. The remaining 13 patients in the group underwent resection with extended end-to-end anastomoses (18). In **Zehr** and colleagues study; the preferred repair technique has changed over the past three decades where resection with end to end anastmosis was the primary mode of repair. Subclavian flap aortoplasty was first performed in their institution in 1978, and the remaining procedures consisted of patch angioplasty, a flap-patch combination, or extra anatomic bypass (13). Later in a recent study, the operative techniques included interposition graft with Hemashield, and Gore-Tex patch aortoplasty repair (21)

In Wells and colleagues series, resection and end-to-end anastmosis, resection and interposition Dacron tube graft, patch angioplasty with Gore-Tex, and a bypass Dacron tube graft from the proximal to the descending thoracic **aorta** were the types of operations (16). In Fesseha and colleagues series patients were operated by resection with extended end-to-end anastmosis or aortic arch advancement (7). In **Seirafi** and colleagues series the extended end-to-end technique was used to augment the hypoplastic aortic arch in 21 neonates and infants. Subclavian flap arterioplasty was performed in 43 neonates and infants. Patch aortoplasty and interposition graft were used primarily in toddlers and older patients, respectively (6). **In Asano** and colleagues series from 33 patients, modified Subclavian flap arterioplasty was performed in 9 patients. The remaining procedures consisted of Subclavian flap arterioplasty in 17 patients, coarctectomy and end-to-end anastmosis in 4 patients, and extended end to end anastmosis in 3 patients (22).

The mean cross clamp time in our series was 26.29 minute with no great difference between different types of operations; this was near that of Dave and colleagues where it was 23.6 (19). In the series of Carr and colleagues, the mean cross clamp time was 31min (10) while in the series of Wright and colleagues it was 17 min (14).

In our series, we used continuous non absorbable prolene suture for anastmosis in all cases and its size was determined according to the age and the size of the aortic wall. In most series the used suture was also prolene suture (7, 12, 20) while Ou and colleagues had reported the use of interrupted and absorbable sutures for repair of the aortic arch (23). Zehr and colleagues reported that the use of monofilament non-absorbable suture is proved to be significantly associated with a reduction in the incidence of restenosis. Monofilament polypropylene is a relatively inert substance, incites minimal adjacent inflammatory reaction, and improves growth of vascular anastomoses (13).

Recoarctation defined as postoperative pressure gradient across site of repair more than 20 mmHg (10) while others defined it as postoperative pressure gradient of more than 15 mmHg (9, 14) where they reported 2.2% and 11% recoarctation rate respectively. Høimyr and colleagues reported 5% of cases with recoarctation that required further intervention after 40 years of follow up (24). During our early postoperative period, the mean postoperative systolic pressure gradient was 11 mmHg where all cases were below 20 mmHg. This was confirmed by Carr and colleagues where the maximum postoperative pressure gradient in their series was 20 mmHg with a mean of 10 mmHg (10). Follow up Echo

was done for all patients during our follow up period, the mean Echo pressure gradient across the site of repair was 8.6±3.7 in group A versus 7.5±3.5mmHg in group B. with a maximum pressure gradient of 15 mmHg and according to the above definitions none of them had recoarctation. Early postoperative paradoxical hypertension was seen in (89%) of patients and it was simply controlled by nitroglycerine infusion in 16 patients and addition of sodium nitropruside in 9 patients; this was near the results of Beur and colleagues where 80% exhibited paradoxical hypertension; which was controlled with sodium nitroprusside infusion and β-adrenergic blockers (15). Carr and colleagues reported that all patients had postoperative reactive hypertension and they were controlled by nitroprusside and nitroglycerin (10), while Wright and colleagues reported it only in 18% of cases and it was controlled by esmolol or sodium nitroprusside (14). In 66% of our cases, nitroglycerine or nitroprusside could be withdrawn without adding other antihypertensive, 34 % required adding oral antihypertensive medications in the form of B blockers or ACEI. During our follow up period half of these patients stopped these medications and became normotensive and the others continued on these medications. These results were near the results of Wright and colleagues where 14% of patients continued on oral antihypertensive medications (14); While Carr and colleagues reported 20% of cases require anti-hypertensives (10).

We have one (3.5%) case of hospital mortality due to onlay patch infection; this was similar to the results of **Seirafi** and colleagues who reported operative mortality of 3.4 % (6). Wright and colleagues reported an incidence of early mortality of 1% (14) while Bouchart and Carr and their colleagues reported 0% of perioperative mortality (10, 12).

In our series, postoperative complications included; 2 patients of postoperative surgical bleeding, 2 patients of chylothorax, one patient of right side hemiparesis, and one of left lower lobe collapse. Thomson and colleagues reported one patient of paraplegia after significant intraoperative bleeding from the anastomotic site, and one patient of transient lower limb weakness but recovered fully. Both **of** these patients underwent extended end to end repair through a thoracotomy. A third patient had a small segmental cerebral infarction on CT after coarctation repair (5). Wells and colleagues reported one patient with persistent left vocal cord paralysis with no other major complications occurred (16). Zehr and colleagues reported; in series of 179 patients, left hemi diaphragm paralysis in 6 patients; vocal cord paralysis in one patient, chylothorax in 4 patients; 2 of them required reoperation for management, Two patients reoperated for the control of postoperative bleeding, and 21 patients of infections; included pneumonia, sepsis, and wound infection in 9, 6, and 6 patients respectively (13). In Carr and colleagues series, the perioperative morbidity rate was 18% that included postoperative bleeding in 3 patients, ventricular tachycardia in one patients, left-hand numbness in one patient, leukocytic vasculitis in one patient, left vocal cord paralysis in one patient, and a lymphocele at the groin cannulation site in one patient (10).

Conclusion: There are many surgical techniques for repair but resection with extended end to end anastmosis is the common method in infants and young children, while interposition tube graft is the common in adults and older children.

References

1. Brouwer MH, Kuntze CE, Ebels TE, Talsma MD and Ejjgelaau AE. Repair of aortic coarctation in infants. J Thorac Cardiovasc Surg 1991; 101: 1093.
2. Horlan JL, Doty DB, Braudt BI and Ehrenhft JE. Coarctation of the aorta in infants. J Thorac Cardiovasc Surg 1984; 88: 1012.
3. Waldhausen JA. The early history of congenital heart surgery: closed heart operations. Ann Thorac Surg. 1997; 64: 5:1533-1539.
4. Backer CL, Paape K, Zales VR, Weigel TJ, Mavroudis C. Coarctation of the aorta. Repair with polytetrafluoroethylene patch aortoplasty. Circulation. 1995; 1; 92(9 Suppl):II132-6.
5. Thomson J D R, Mulpur A, Guerrero R, Nagy Z, Gibbs J L, Watterson K G. Outcome after extended arch repair for aortic coarctation. Heart. 2006; 92: (1):90-94.
6. Seirafi PA, Warner KG, Geggel RL, Payne DD, Cleveland RJ. Repair of coarctation of the aorta during infancy minimizes the risk of late hypertension. Ann Thorac Surg. 1998; 66 (4):1378-1382.
7. Fesseha AK, Eidem BW, Dibardino DJ, Cron SG, McKenzie ED, Fraser CD Jr, Price JF, Chang AC, Mott AR. Neonates with aortic coarctation and cardiogenic shock: presentation and outcomes. Ann Thorac Surg. 2005; 79(5):1650-1655.
8. Conte S, Lacour-Gayet F, Serraf A, Sousa-Uva M, Bruniaux J, Touchot A, Planché C. Surgical management of neonatal coarctation. Thorac Cardiovasc Surg. 1995; 109(4):663-674; discussion 674-675.
9. Wood AE, Javadpour H, Duff D, Oslizlok P, Walsh K. Is extended arch aortoplasty the operation of choice for infant aortic coarctation? Results of 15 years' experience in 181 patients. Ann Thorac Surg. 2004; 77(4):1353-1357; discussion 1357-1358.

10. Carr JA, Amato JJ, Higgins RSD. Long-Term Results of Surgical Coarctectomy in the Adolescent and Young Adult With 18-Year Follow-Up. Ann Thorac Surg 2005; 79: 1950-1956.
11. Aris A, Subirana MT, Ferrés P, Torner-Soler M. Repair of aortic coarctation in patients more than 50 years of age. Ann Thorac Surg 1999; 67: 1376-1379.
12. Bouchart F, Dubar A, Tabley A, Litzler PY, Haas-Hubscher C, Redonnet M, Bessou JP, Soyer R. Coarctation of the aorta in adults: surgical results and long-term follow-up. Ann Thorac Surg. 2000; 70(5): 1483-1488; discussion 1488-1489.
13. Zehr KJ, Gillinov AM, Redmond JM, Greene PS, Kan JS, Gardner TJ, Reitz BA, Cameron DE. Repair of coarctation of the aorta in neonates and infants: a thirty-year experience. Ann Thorac Surg. 1995; 59(1): 33-41.
14. Wright GE, Nowak CA, Goldberg CS, Ohye RG, Bove EL, Rocchini A P. Extended Resection and End-to-End Anastomosis for Aortic Coarctation in Infants: Results of a Tailored Surgical Approach. Ann Thorac Surg 2005; 80:1453-1459.
15. Bauer M, Alexi-Meskishvili VV, Bauer U, Alfaouri D, Lange PE, Hetzer R. Benefits of surgical repair of coarctation of the aorta in patients older than 50 years. Ann Thorac Surg. 2001; 72(6): 2060-2064.
16. Wells WJ, Prendergast TW, Berdjis F, Brandl D, Lange PE, Hetzer R, Starnes VA. Repair of coarctation of the aorta in adults: the fate of systolic hypertension. Ann Thorac Surg. 1996; 61(4):1168-1171.
17. Elkerdany A, Hassouna A, Elsayegh T, Azab S, Bassiouni M. Left subclavian-aortic bypass grafting in primary isolated adult coarctation. Cardiovasc Surg. 1999; 7(3): 351-354.
18. Sudarshan CD, Cochrane AD, Jun ZH, Soto R, Brizard CP. Repair of coarctation of the aorta in infants weighing less than 2 kilograms. Ann Thorac Surg. 2006; 82(1): 158-163.
19. Dave HH, Buechel ERV, Prêtre René. Muscle-Sparing Extrapleural Approach for the Repair of Aortic Coarctation. Ann Thorac Surg 2006; 81: 243-248.
20. Elgamal MA, McKenzie ED, Fraser CD Jr. Aortic arch advancement: the optimal one-stage approach for surgical management of neonatal coarctation with arch hypoplasia. Ann Thorac Surg. 2002; 73(4): 1267-1272; discussion 1272-1273.
21. Backer CL, Stewart RD, Kelle AM, Mavroudis C. Use of partial cardiopulmonary bypass for coarctation repair through a left thoracotomy in children without collaterals. Ann Thorac Surg. 2006; 82(3): 964-972.
22. Asano M, Mishima A, Yamamoto S, Saito T, Manabe T. Modified subclavian flap aortoplasty for coarctation repair in patients less than three months of age. Ann Thorac Surg. 1998; 66(2): 588-589.
23. Ou P, Mousseaux E, Celermajer DS, Pedroni E, Vouhe P, Sidi D, Bonnet D. Aortic arch shape deformation after coarctation surgery: effect on blood pressure response. J Thorac Cardiovasc Surg. 2006; 132(5): 1105-11.
24. Høimyr H, Christensen TD, Emmertsen K, Johnsen SP, Riis A, Hansen OK, Hjortdal VE. Surgical repair of coarctation of the aorta: up to 40 years of follow-up. Eur J Cardiothorac Surg. 2006; 30(6): 910-916.

Feasibility and Accuracy of Real-time Three Dimensional Echocardiographic Assessment of Ventricular Septal Defects Comparative study with 2D echocardiography and surgical findings

Hassan Moftah, MD,*
Maiy El sayed, MD, **
Ghada El shahed, MD, **
Alaa Roushdy, MD, *
Haytham Moussa, Msc*

Background : **Three-dimensional (3D) echocardiography offers the ability to improve and expand the diagnostic capabilities of cardiac ultrasound. However, as with any emerging technology, the enthusiasm to embrace a new technique must be tempered by a critical appraisal of the evidence supporting its use.**

Objective To evaluate the feasibility and accuracy of 3D echocardiography in the assessment of ventricular septal defects

Methods : **The study included 20 patients who were scheduled for VSD closure. All patients were subjected to 2D echocardiogram as well as real time 3D echocardiography with full volume acquisition. Data obtained was tabulated and analyzed to determine the additional value of 3D echocardiography compared to 2D echocardiography in assessment of VSD as well as the accuracy of the 3D echocardiographic data compared to the surgical findings**

Results : **There was complete agreement as regards the morphology of the VSD between RT-3DE and surgery. RT-3DE was of additional value compared with 2DE in detection of other defects. There was no significant difference between RT-3DE and surgery in determination of the site of VSD. There was modest correlation between the size of the VSD measured by 2D echocardiography and that measured by RT-3DE from the LV and RV enface view with an r value of 0.637 and 0.684 respectively. Using linear regression analysis the correlation between the maximal VSD diameter measured during surgery and that measured by both 2DE (r = 0.724) and RT-3DE LV (r = 0.948) and RV enface view (r = 0.938) favored the measurements obtained by RT-3DE.**

Conclusion : **RT-3DE is feasible for quantitative assessment of VSDs with accurate determination of VSD size, number, shape and localization. The use of RT-3DE gives a better understanding of the exact anatomy and size of the defect, which may lead to a further optimization of the planning of surgical or catheter-interventional procedures in patients with a VSD. The enface view of the ventricular septum is a unique capability of the 3D echocardiography.**

Address reprint request to :
Hassan Moftah , M.D.
Departement of Lecturer, cardiothoracic surgery, Ain Shams University
Email : jegyptscts@gamil.com
Codex : 04/117/0906

Ultrasound technology has improved markedly in the past 10 to 15 years, prompting echocardiographers to extend its use in studying cardiac structure and function. New ultrasound equipment and techniques offer superior image quality, greater accuracy, and expanding capabilities. Of these newer techniques, three-dimensional (3D) echocardiography offers the ability to improve and expand the diagnostic capabilities of cardiac ultrasound. However,

as with any emerging technology, the enthusiasm to embrace a new technique must be tempered by a critical appraisal of the evidence supporting its use. It is essential to assess the limitations as well as the unique capabilities it provides. To justify the use of a new 3D modality, its unique contribution to clinical practice must be critically analyzed (1)

Although reports on the incidence of congenital heart disease (CHD) vary considerably from 0.4 to 5% the most widely accepted estimate of the incidence of CHD is 0.8% of all live births. Of the CHD, isolated VSD is by far the most common diagnosis, accounting for 20 % to 30% of all CHD. (2)

Functional and morphologic assessment of VSD is routinely done with 2-dimensional (2D) and color Doppler echocardiography. Usually, this provides adequate information to decide on surgical repair. Nevertheless, the anatomy of the VSD is complex and cannot be presented by actual imaging techniques in a single plane. (3)

Furthermore, advances in cardiac surgical procedures increasingly demand support of highly accurate imaging techniques. 3D echocardiography has been proposed as a new technique able to simulate the intra operative visualization of cardiac structures and to improve the understanding of the anatomy of congenital heart disease. (4)

Methods:

The current study was conducted on 20 patients referred to the Cardiology department at Ain Shams University Hospitals for preoperative assessment of VSD, in the period from February 2008 to July 2008.

Echocardiographic examination: All patients were subjected to full 2D echocardiogram using sequential analysis to establish the diagnoses as well as assess LV dimensions and functions. Real time 3D echocardiogram (RT3DE) was performed from the subcostal, apical and parasternal windows using a commercial scanner (iE33, Philips Medical Systems) equipped with a fully sampled matrix array transducer Care and taken to include the entire LV cavity within the pyramidal scan volume. The displaying mode of RT-3DE includes 3D Live and Full Volume and color Doppler mapping. RT3DE data sets were acquired using wide-angled acquisition mode with minimal possible depth to achieve a high frame rate. Duration of 3D data acquisition was 10±2 minutes. Reconstruction time was 30±10 minutes. RT3DE data sets were stored digitally, analyzed by the quantification lab (Q Lab) and the full volume set was cropped in various planes to assess VSD morphology including site, number, size, shape and relation to other structures.

Surgical Procedure and Assessment of Morphology: Surgical repair was performed, using cardiopulmonary bypass, through a right atriotomy. VSD measurements were made in a flaccid heart after cold cardioplegia from a RV view after transtricuspid exposure of the defect. The surgeon, who was blinded for the 3D data, described and measured the VSD. Using the surgical description as the gold standard, the accuracy of each type of echocardiographic assessment was determined.

Statistical analysis: Data was collected, verified, revised and edited on a personal computer. It was then statistically analyzed using SPSS statistical package version 13 and Microsoft Excel 2003. All data were calculated as mean and standard deviation. T test for independent samples and Pearson's correlation coefficient were calculated whenever needed. Bland-Altman and linear regression analysis was used to correlate the data obtained from both 2D and 3D echocardiography and surgical findings.

Results

This study was conducted on 20 patients with congenital ventricular septal defect referred to the Cardiology department at Ain Shams University Hospitals for preoperative assessment. (Table 1).

Characteristics	Findings
Gender (M/F)	11/9
Mean age (range) in yrs	1.7(0.5-7)
Referral diagnosis	
VSD	(n=13)
VSD -ASD	(n=2)
TOF	(n=3)
DORV	(n=2)

Table: (1) Clinical and demographic characteristics of the patients
VSD= ventricular septal defect, ASD= atrial septal defect, TOF= Tetralogy of fallot, DORV = double outlet right ventricle

In the 20 patients, it was possible to reconstruct a VSD with complete borders from the RV enface view in 19 patients (95%) and from the LV enface view in17 patients (85%) due to the presence of overriding of the aorta which led to the presence of incomplete borders in the remaining patients.

The current study showed that there was complete agreement as regard the morphology of the VSD between RT-3DE and surgical findings, the shape of VSD was well delineated after acquisition of full volume data set and cropping. The VSD shape was seen from both the RV and LV perspective (enface view) (Figs. 1 and 2). RT-3DE was of additional value compared with 2DE in detection of other defects. All patients showed one defect by 2D echocardiography but one of the patients showed 2 adjacent perimembranous subaortic VSDs by 3D echocardiography.

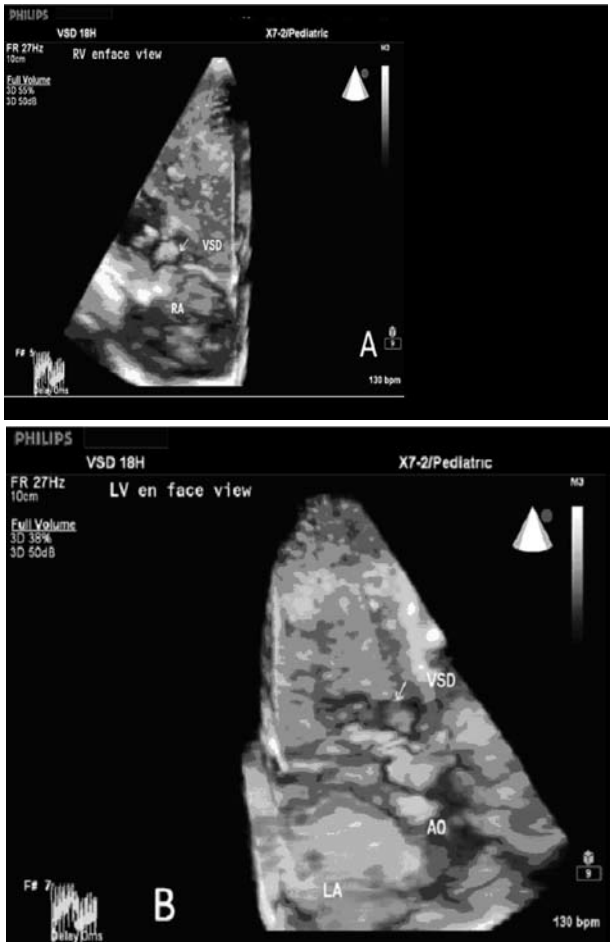


Figure 1. RT-3DE display of typical perimembranous ventricular septal defect (VSD) (arrow) viewed from right (A) and left (B) ventricular surfaces. (RA, right atrium; LA, left atrium; Ao, aorta).

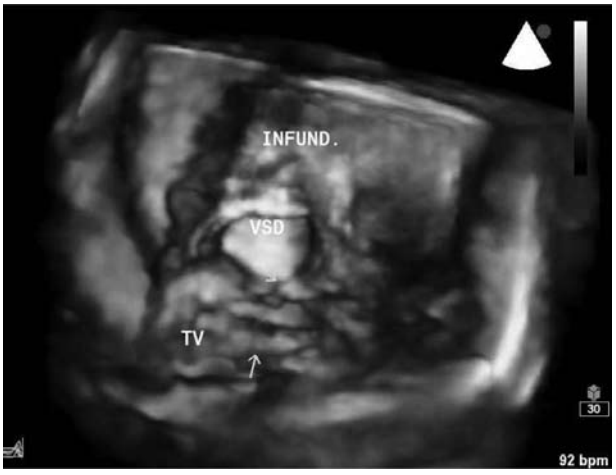


Figure 2. RT-3DE display of perimembranous ventricular septal defect (VSD) viewed from right ventricular surface of interventricular septum. Two papillary muscles (arrows) are seen crossing the VSD. (TV, tricuspid valve; Infund, infundibulum).

There was no significant difference between RT-3DE and surgery in determination of the site of VSD; (P-value = 0.059) with a percentage of agreement between the two methods of 93.33% (in only one case, RT3-DE described an inlet outlet VSD but the surgeon described a perimembranous VSD with inlet outlet extension). This percentage of agreement dropped to 86.6% when comparing 2D echocardiography findings and surgery.

Maximum VSD diameter was 10.9 ± 2.57 mm and 14.367 ± 4.526 mm for 2DE and surgery respectively. Maximum VSD diameter was 12.467 ± 3.493 mm and 12.533 ± 5.105 mm using RT3-DE in LV and RV enface view respectively. Using Bland-Altman analysis, the mean difference between the 2DE measurements and surgical findings was 3.467 ± 3.202 (P-value >0.001), while the mean difference between the surgical diameter and that measured from the LV enface and RV enface views were 1.9±1.647 and 1.833±1.791 respectively.

Using linear regression analysis, the correlation between the maximal VSD diameter measured during surgery and that measured by both 2DE (r = 0.724) (Fig.3) and RT-3DE LV (r = 0.948) (Fig.4) and RV enface view (r = 0.938) (Fig.5) favored the measurements obtained by RT-3DE.

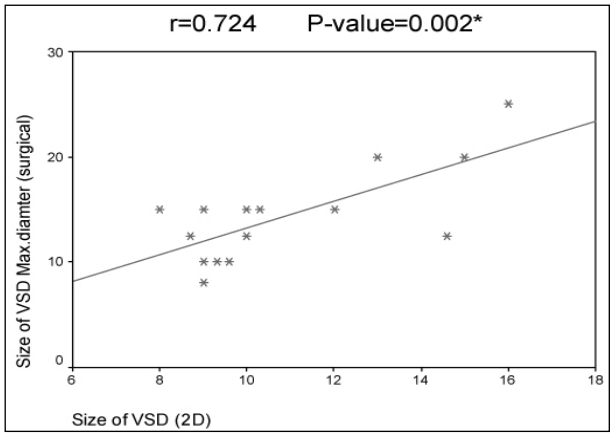


Figure 3: correlation between maximum VSD diameters measured by 2DE and surgery (linear regression)

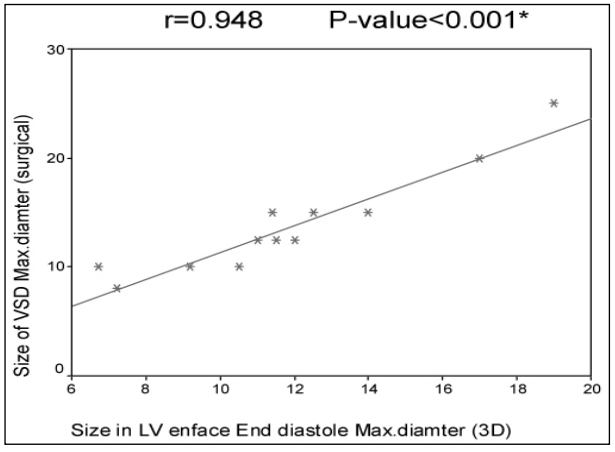


Figure 4: correlation between maximum VSD diameters measured by RT-3DE LV enface view and surgery (linear regression)

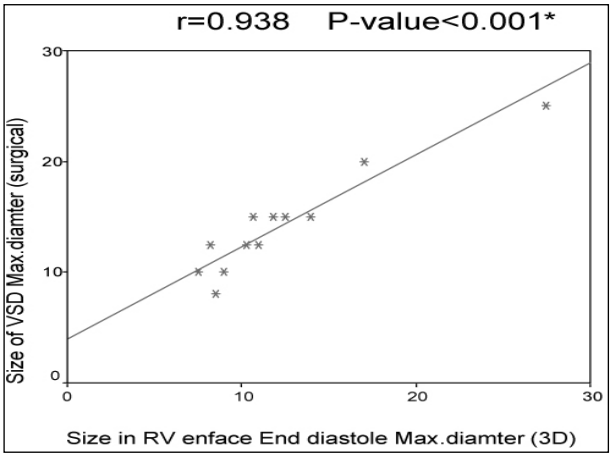


Figure 5: correlation between maximum VSD diameters measured by RT-3DE RV enface view and surgery (linear regression)

By 3D echocardiography both the end diastolic and end systolic dimensions of the VSD could be measured thus giving more accurate data concerning the hemodynamic significance of each VSD. From the LV enface view end diastolic maximum diameter had mean value 13.4±5.547 mm but end systolic maximum diameter had mean value 11.521±5.279mm (P= <0.001). From the RV enface view end diastolic maximum diameter had mean value 13.884±6.449 mm but end systolic maximum diameter had mean value 11.068±5.323 mm (P=0.001).

There was modest correlation between the size of the VSD measured by 2DE and that measured by RT-3DE from the LV and RV enface views with an r value of 0.0.637 and 0.684 respectively.

RT-3DE more clearly depicts sufficient anatomic definition of VSD and adjacent structures in depth perception, and resolution for the preoperative description of VSD for planning and performing surgical closure of VSD.

Discussion

Two-dimensional echocardiography is a well established method for the diagnosis of VSDs, although it requires mental integration of composite orthogonal views to conceptualize the position of the defect within the interventricular septum and its relationship with other anatomic structures. (4)

Accurate assessment of the size and shape of a VSD is also difficult, because complete visualization of the borders of a defect cannot be achieved in a single 2D view. More precise delineation of certain VSDs might be advantageous for surgical and Trans catheter device closure. (5)

Preliminary experience has demonstrated that 3D echocardiography can provide important information pertaining to the morphological and spatial features of various types of VSDs. (6)

In 2006 Chen et al studied 38 patients with VSD using RT-3DE. 3D image database was post processed using TomTec echo 3D workstation. The results were compared with the results measured by 2 DE and surgical findings. RT-3DE produced novel views of VSD and improved quantification of the size of the defect. These results were comparable to the data obtained in our study where the 3D measurements of the VSD showed modest correlation with those obtained from 2D echocardiography. (6)

In 2006 Van den bosch et al studied 34 patients, who were scheduled for surgical closure of a VSD, with (Philips Sonos 7500, Philips Medical Systems, Andover, Mass) system, RT-3DE allowed accurate determination of VSD size, shape, and location. Acquisition of RT-3DE data sets was feasible in 30 of 34 (88%) patients; Duration of 3D data acquisition was 6 ± 2 minutes. Reconstruction time was 23 ± 16 minutes. In our study acquisition of full volume was possible in all patients, however the feasibility to visualize the VSD with complete borders was 95% from the RV enface view and 85% from the LV enface view due to the presence of malaligned defects with overriding of the aorta; Duration of 3D data acquisition was 10±2 minutes and the average reconstruction time was 40±15 minutes dropped to 20±10 minutes as the learning curve progressed. (7)

In the study by Van den bosch et al, There was 100% sensitivity and specificity regarding localization and number of the VSD determined by RT-3DE compared with surgical findings and in 4 patients the localization of the VSD determined on 2DE was not assessed correctly. In these patients the VSD extended to the inlet part of the interventricular septum, which was not visible and could not be retrieved from the stored 2D data set. This is similar with the current study where sensitivity of RT-3DE compared with surgical findings regarding the localization of the VSD was 93.33% but it was less when 2DE compared with surgical findings(sensitivity was 86.67%) which shows the superiority of 3DE over 2DE in assessment of VSD morphology as regard site. (7)

Van den Bosch et al showed weak correlation between maximal 2D and maximal 3D diameters (r = 0.57), and between maximal 2D diameter and surgical findings (r = 0.66). This was in accordance to our study where r was 0.684 between maximal 2D and maximal 3D diameters; r was 0.724 between maximal 2D diameter and surgical findings. (7)

Van den Bosch et al showed excellent correlation of maximal VSD dimension between 3DE and surgical findings (r = 0.95) which is similar to the findings of our study (r = 0.938) when comparing maximal VSD dimension between 3DE and surgery. (7)

Van den bosch et al showed that a VSD increases and decreases considerably in size during the cardiac cycle (mean area change 0.53 ±0.12) and varied markedly between the individual patients. In the current study, the test of change of VSD size during the cardiac cycle was done by comparing end diastolic with end systolic maximal diameter and there was a significant difference between the two measurements (P-value = 0.001). (7)

Our data was also in agreement with those obtained by Mehmood et al, who studied 12 patients who underwent surgical repair for VSD. They concluded in their study that "Live/real time 3DTTE accurately defined VSD location, size, and surrounding anatomy in all patients studied" (8)

Conclusion

RT-3DE is feasible for quantitative assessment of VSDs with accurate determination of VSD size, number, shape and localization. The use of RT-3DE gives a better understanding of the exact anatomy and size of the defect, which may lead to a further optimization of the planning of surgical or catheter-interventional procedures in patients with a VSD. The En face view of the ventricular septum is a unique capability of the 3D echocardiography

References

1- J. Hung, R. Lang, F. Flachskamp et al. 3D Echocardiography: A Review of the Current Status and Future Directions J Am Soc Echocardiogr. 2007; 20 (3): 213-233

2- J. Hoffman, and S. Kaplan. The incidence of congenital heart disease. J Am Coll Cardiol, 2002; 39:1890-1900

3- M. Vogel, S. Yen Ho, C. Lincoln, et al. Three-Dimensional Echocardiography Can Simulate Intraoperative Visualization of Congenitally Malformed Hearts. Ann Thorac Surg 1995;60:1282-1288

4- L. Mercer-Rosa, M. Seliem, A. Fedec, et al. Illustration of the Additional Value of Real-time 3-dimensional Echocardiography to Conventional Transthoracic and Transesophageal 2-dimensional Echocardiography in Imaging Muscular Ventricular Septal Defects: Does This Have Any Impact on Individual Patient Treatment? J Am Soc Echocardiogr. 2006; 19 (12): 1511-1519

5- Butera G, Chessa M, Carminati M. Percutaneous closure of ventricular septal defects. State of the art. J Cardiovasc Med (Hagerstown). 2007 Jan; 8(1):39-45.

6- Chen FL, Hsiung MC, Nanda N, et al. Real time three-dimensional echocardiography in assessing ventricular septal defects: an echocardiographic-surgical correlative study. Echocardiography. 2006; 23(7):562-8.

7- Van den Bosch AE, Ten Harkel DJ, McGhie SJ, et al. Feasibility and accuracy of real-time 3-dimensionsal echocardiographic assessment of ventricular septal defects. J Am Soc Echocardiogr 2006; 19:7–13.

8- Mehmood F, Miller AP, Nanda NC, , et al. Usefulness of live/real time three-dimensional transthoracic echocardiography in the characterization of ventricular septal defects in adults. Echocardiography 2006; 23(5):421-7

Thoracic

Pattern and presentation of blunt chest traumas among different age groups: analysis of 486 cases

Moataz Hanafi 1, 2, MD, Nael Al-Sarraf 2, MRCSI, Hazem Sharaf 1 , MD, Atef Abdelaziz 1, PhD.

Objectives: **To assess the pattern of presentation and the mechanism of injury of blunt chest injuries sustained in different age groups and its impact on management options.**

Methods: **This is a retrospective review of all blunt chest trauma patients managed in a single centerover a 3 year period. There were 486 patients stratified into three age groups: pediatric (≤ 12 years old), adult (>12 and ≤60 years old), and elderly (>60 years old). All clinical dataincluding age, sex, mechanism of chest injury, associated extrathoracic injuries, treatment strategies and mortality were analyzed.**

Results: **Majority of patients were males (418, 86%). The adult group was the commonest age group to sustain blunt chest injury (385 patients, 79%). The commonest cause was motor vehicle accident (MVA) accounting for 93% (452 patients). Isolated chest injuries (335 patients, 69%) were commoner than combined chest injuries (151 patients, 31%). Tube thoracostomy insertion was required in 191 patients (39%) predominantly in the elderly group. The commonest combined chest injury involved bone fractures (102 patients, 21%) followed by head injuries (25 patients, 5%) and abdominal injuries (24 patients, 5%). Majority of blunt chest injuries were treated conservatively (464 patients, 95.5%). Open thoracotomy was required in the remaining 4.5% (22 patients). Indications for thoracotomy were clotted haemothorax (18 cases, 3.7%) followed by empyema (3 cases, 0.6%) and massive air leak (1 case, 0.2%) Overall mortality was 1% (5 patients), predominantly in the adult group.**

Conclusions: **Blunt thoracic injuries are a major cause of morbidity and mortality. Majority of such injuries are related to MVA. Preventative strategies should be enforced by stronger regulations and should concentrate on the middle age adult group where the incidence of blunt chest traumas is highest.**

Trauma is one of the most sudden, dramatic and often irreversible medical conditions and is associated with a significant mortality [1]. Chest injury alone is responsible for 25% trauma related deaths and is a contributing factor in another 25% [1]. Approximately, 80% of blunt chest traumas are due to road traffic accidents. Data on the pattern of blunt chest injuries based on different age groups are currently limited [2-5]. Furthermore, the strategies in dealing with such injuries need to be optimized. In this paper, we sought to examine our experience with such traumas adding further to our understanding on their presentation pattern and management strategies.

Address reprint request to :
Dr Moataz Hanafi
Department of Cardio-thoracic Surgery, University
Email : jegyptscts@gamil.com
Codex : 05/22/0906

Methods:

This retrospective study was performed on all patients who were admitted to a single tertiary center at King Fahad hospital at Al-Baha suffering from blunt chest traumas between August 2004 and August 2007. During this time period, a total of 985 patients were admitted suffering from general trauma, of which 486 patients (49%) were chest traumas. All the 486 patients were reviewed and evaluated regarding age, sex, mechanism of injury, associated injuries, management strategy and short-term outcome. Patients were included in the study if they had sustained blunt chest injury either in isolation or in combination with other system injuries. Patients were stratified into three groups based on their age. First group was the pediatric group which included all patients under the age of 12 years old (n=46, 9.5%). The second group was the adult group which included all patients between the age of 12 and 60 years old (n=385, 79%). The third group was the elderly group which included all patients above the age of 60 years old (n=55, 11.5%). All these patients initially presented to our emergency department and following initial assessment and AP chest x-ray, chest tube thoracostomy was performed if indicated according to standards of Advanced Trauma life support (ATLS) guidelines. This study was approved by our institutional review board.

Results:

There were 486 patients reviewed, of which 418 patients (86%) were males and 68 patients were females (14%). Age ranged from 1 month to 78 years old with a mean of 27 years old. Patients were stratified into three age groups (as described in materials and method section). The main cause of blunt chest trauma was motor vehicle accident (MVA) which occurred in 452 patients (93%) while other causes occurred in 34 patients (7%). These causes and their breakdown are listed in table 1. Intensive care unit (ICU) admission was required 192 cases (40%) while the remaining 294 patients (60%) were managed in a surgical ward.

Mechanism of injury	Number (%)
Motor vehicle accident (MVA)	452 (93%)
- Passenger	372
- Pedestrian	74
- motorcycle	6
Fall from height	22 (4.5%)
Other*	12 (2.5%)

Table 1: mechanism of injury among the study population (n=486) * Includes fall of heavy objects on chest and direct blow to chest with a blunt object

Table 2 is a summary of isolated and combined chest injuries among the study population

	Pediatric (n=46)	Adult (n=385)	elderly (n=55)
Isolated	30 (65%)	260 (68%)	45 (82%)
Combined	16 (35%)	125 (32%)	10 (18%)

Table 2: Isolated vs. combined chest injuries stratified on the basis of age (n=486 patients).

when stratified by age. Pattern of isolated chest injuries among the three age groups is summarized in table 3 and the combined chest injuries among the three groups are summarized in table 4.

Variable	pediatric (n=30)	adult (n=260)	elderly (n=45)
Rib fractures only (no chest tube required)	7 (23%)	132 (51%)	5 (11%)
Rib fractures with haemothorax/pneumothorax / haemopneumothoarx	23 (77%)	72 (28%)	40 (89%)
Bilateral rib fractures with bilateral S/C emphysema requiring bilateral chest tubes	nil	56 (21%)	Nil

Table 3: Isolated chest injuries in the absence of other associated injuries stratified on the basis of age (n=335)

Variable	pediatric (n=16)	adult (n=125)	elderly (n=10)
Bone fractures (n=102)	9 (56%)	89 (71%)	4(40%)
Abdominal injury (n=24)	4 (25%)	17 (14%)	3 (30%)
Head injury (25)	?3(19%)	19 (15%)	3 (30%)

Table 4: Combined chest injuries in the absence of other associated injuries stratified on the basis of age (n=335) basis of age (n=151)basis of age (n=151)

In the elderly group, isolated chest injuries were commoner than combined ones (82%vs. 18%, table 1). In addition, the majority of isolated chest injuries (89%) are rib fractures with an associated pneumothorax, haemothorax or both (table 3). These were all managed by simple tube thoracostomy. Only 11% of elderly patients sustained rib fractures with no associated haemothorax / pneumothorax that did not require tube thoracostomy but merely adequate analgesia to prevent more serious complications such as pneumonias. The high incidence of rib fractures in this group is due to the higher incidence of osteopenic changes seen in elderly population leading to more pronounced outcome when the patient sustain blunt chest injury [6] and consequently higher mortality. Combined injuries in the elderly group involved extremity fractures in 40% of cases while the remainder sustained head or abdominal injuries (30% each, table 4). Open thoracotomy was required in 12 cases (22%) all due to clotted haemothorax. Mortality in this group was seen in 1 case only.

In Pediatric group, majority of patients had isolated chest injury rather than combined one (65% vs. 35%, respectively, table 1). Similar to the elderly group, the majority of patients in the pediatric group with isolated chest injury have sustained rib fractures in association with unilateral haemothorax, pneumothorax or both (77%, table 3). These cases required tube thoracostomy insertion. The remaining 23% had only isolated rib fractures. Open thoracotomy was required in one case due to persistent air leak. There was no mortality seen in this age group. The incidence of traumatic chest injuries in children is lower than adults but carries a higher morbidity and mortality. The serious consequences of blunt chest injuries in pediatric group are related to the mechanical strength of the ribs and thoracic cavity in children which are easily fractured by high speed traumas than in adults [3]. Combined injury occurred in 35% of patients in the pediatric group (table 2). Extremity fractures were commoner than abdominal and head injuries (56% vs. 25% vs. 19%, table 4).

The adult group had sustained the highest incidence of chest injuries among all study population (385 patients of 486, 79% overall). In this group, isolated rib fractures accounted for 51% of isolated chest injuries with the reaming 49% of patients requiring chest tube insertion either unilaterally (28%) or bilaterally (21%, table 3). This signifies the degree of high speed traumas seen in this group. Similarly, the rate of combined injuries sustained in the adult group was higher than the other two age groups combined (125 patients vs. 16 for pediatrics and 10 patients in elderly group, table 2). This also signified the high speed traumas and high energy transfer incurred during MVA collisions among this group. The commonest extrathoracic system injury in this group was extremity fracture (89 patients followed by head and abdominal injuries (19 and 17 patients respectively, table 4). The highest mortality rate was seen in this group (4 cases, 1%). Open thoracotomy was required in 9 cases (2%) mainly due to clotted haemothorax (6 cases) and empyema (3 cases). These are summarized in table 5.

Variable	Pediatric (n=46)	Adult (n=385)	Elderly (n=55)
Open thoracotomy	1 (2%)	9 (2%)	12 (22%)
Indication for thoracotomy	Massive persistent air leak	Clotted haemothorax (n=6) Empyema (n=3)	Clotted haemothorax
In-hospital mortality	0	4 (1%)	1 (1.8%)

Table 5: Indications of open thoracotomy in the study population and in-hospital mortality (n=486).

Discussion :

Blunt chest injuries are usually related to MVA. The relationship between age and the high incidence of blunt chest injuries related to MVA has been previously reported [2-4].

However, data on the pattern of presentation and management strategies among different age groups sustaining blunt chest injuries are rather limited [7]. In this study, we sought to identify the pattern of chest injuries sustained in blunt traumas among differing age groups. In doing so, our understanding of the pattern of their presentation, their incidence and outcome is improved. This will ultimately help in directing resources to both treatment and preventative strategies to those who require it most leading to more cost-effective and optimal approach.

As shown in our work, the commonest age group to sustain blunt chest injury is the middle age group (>12 and <60 years old). In addition, this group also resulted

in the highest incidence of mortality among our study population. It seems logical that tougher regulation should be in place concentrating on this high risk group. Preventative strategies should also go hand in hand with earlier diagnosis and more aggressive management in the high risk category. Approximately half of our patients in this age group required tube thoracostomy insertion in the isolated chest injury group while the other half required conservative management. However, in the combined injury group extremity fractures were seen commonest than the other two system injuries (head and abdominal). Therefore, it seems rational that successful management of major chest injuries requires a prompt diagnosis and collaborative approach between surgeons, anesthesia, ICU team, radiologist and physiotherapists. This will ultimately lead to a better outcome and more streamline management strategies. Specific to say in chest injuries, minimizing pulmonary complications by maintenance of pulmonary and tracheal hygiene, effective eradication of pleural fluid / blood and air, together with more readily used analgesia in the form of epidural infusions will ultimately lower the mortality and morbidity associated with blunt chest injuries [8, 9]. If the conservative management is not sufficient and intra-thoracic organ injuries are detected, early or late thoracotomy should be performed.

Timing of diagnosis is crucial. A chest X-ray film must be performed at the initial presentation not only to identify the number and the extent of rib fractures, but also to determine whether there is an associated pneumothorax, haemothorax and pleural effusion [10] that might require surgical intervention (either by tube thoracostomy insertion or by thoracotomy). Rib fractures usually heal readily if complications are handled properly.

However, the pain associated with the fracture can prevent proper ventilation and coughing, leading to atelectasis, retained secretions and pneumonia, especially in the elderly [8]. The simple effective method of epidural infusion, adequate pain relief, chest physiotherapy with the use of bronchoscopic suction can be of outmost importance in decreasing the complications associated with chest injuries.

The majority of blunt chest injuries can be managed successfully by simple tube thoracostomy [11] with open thoracotomy only required in small cases (4.5% overall in our study). Majority of cases (60%) were managed successfully in surgical ward while the remaining 40% required ICU admission. The multiple disciplinary approach between various teams have lead to a substantially low mortality overall (1%). We recommend that such approach should be employed in any center dealing with chest injuries.

Conclusion:

MVA-related blunt chest injury represents a significant component of the morbidity and mortality from trauma. The most frequent injuries include fractured ribs with pulmonary pneumothorax and haemothorax. Chest trauma can also present with extrathoracic system injuries, especially extremity fractures. There should be preventive measures aimed at preventing these MVA-related injuries, especially among the adult age group. Recognition and treatment depend heavily on a high index of suspicion combined with the appropriate diagnostic tests and multidisciplinary approach to these cases. Conservative management and adequate pain control are of a great value in prognosis and outcome in management of chest trauma Acknowledgments: None.

References:

1) Ahmed Z, Mohyuddin Z. Management of flail chest injury: internal fixation versus endotracheal intubation and ventilation. J Thorac Cardiovasc Surg. 1995;110:1676-80.
2) Bulger EM, Arneson MA, Mock CN, Jurkovich GJ. Rib fractures in the elderly. J Trauma 2000; 48(6):1040-1047.
3) Inan M, Ayvaz S, Sut N, Aksu B, Basaran UN, Ceylan T. Blunt chest trauma in childhood. ANZ J Surg 2007; 77:682-685.
4) Shorr RM, Crittenden M, Indeck M, Hartunian SL, Rodriguez A. Blunt thoracic trauma: analysis of 515 patients. Ann Surg 1987; 206 (2):200-205.
5) Liman ST, Kuzucu A, Tastepe AI, Ulasan GN, Topcu S. Chest injury due to blunt trauma.Eur J Cardiothorac Surg 2003; 23:374-378.
6) Lee RB, Bass SM, Morris JA, MacKenzie E. Three or more rib fractures as an indicator for transfer to a level I center: a population-based study. J Trauma 1990; 30(6):689-694.
7) Boyd AD, Glassman LR. Trauma to the lung. Chest Surg Clin N Am. 1997;7:263-84.
8) Nelson LD. Ventilatory support of the trauma patient with pulmonary contusion. Respir Care Clin North Am. 1996; 2:425-447.
9) Cappello M, De Troyer A. Actions of the inspiratory intercostal muscles in flail chest. Am J Respir Crit Care Med. 1997;155:1085-9.
10) Nadell G, Clevenger FW. Radiographic findings in a blunt trauma patient. Respir Care. 1993;38:1423-7
11) Huber-Wagner S, Korner M, Ehrt A, Kay MV, Pfeifer KJ, Mutschler W, Kanz KG. Emergency chest tube placement in trauma care- which approach is preferable? Resuscitation 2007; 72:226-233.

Invited Commentary

This paper is just a statistical study of a casual activity of a group of surgeons in two different centers. The interest in this paper is that it shows the statistical incidence of motorcar accidents in two countries of the gulf area. Nevertheless, the paper did not tackle any of the major complications of the sudden deceleration either motor car accidents or falling from height accidents . In the first case it is rupture of thoracic aorta is the most common lesion . The fact that the protocol did not insist on doing multiple successive chest X rays at regular intervals to look for a widening of the mediastinum has definitely missed many cases of rupture thoracic aorta. It is well stated that in any case of sudden deceleration motor car accidents where deaths have occurred , causality physicians should look for a case of rupture of a thoracic aorta among the survivors. The first chest X ray may appear normal while the one taken after one or two hours may show widening of the mediastinum. In such a case a multislices tomodensitometry scanner is done to detect the aortic rupture . In this paper such an important issue has not even been discussed. Similarly in cases of fall from heights , search for rupture of supra-hepatic veins is looked for.

Princess Diana died from rupture of pulmonary veins during the sudden deceleration of their motor car.

Without tackling the pathogenesis of sudden deceleration in motor car accidents and in falling from heights accidents the paper is banal. Nevertheless, the paper should be published to show the statistical study about incidence of motor car accidents and age of patients in two of the gulf area countries .

Mohamed AHMED-NASR MD, PHD,FACS
Professor Of Cardio-Thoracic Surgery
National Heart Institute.

Lung Transplantation; Preliminary Experience In Jeddah.

Sameh Ibrahim Sersar MD,
Iskander Al Githmi MD.

Introduction and aim of the work: **Lung transplantation is an expensive operation to improve survival and to have a better quality of life of those patients with end stage respiratory diseases.(1) This retrospective study aims to present our preliminary experience in King Faisal Specialist Hospital and Research Centre; Jeddah (KFSHRC; Jeddah) in lung transplantation and to highlight and underline some of the mortality risk factors in this small group of patients.**

Methods : **This study is a retrospective one that presents our small and limited but growing preliminary experience in lung transplantation. It includes 7 cases of end stage respiratory diseases for whom lung transplantation was done in KFSHRC; Jeddah in 5 years.**

Results: **Seven cases of end stage respiratory disease had lung transplantation in our centre in about 5 years. Three cases had bilateral lung transplant (BLT), 3 left and 1 right lung transplantations;single lung transplant (SLT). The indications were interstitial pulmonary fibrosis in 3 cases, emphysema in 1 case, lymphangioleiomyomatosis in a case, dysmotile cilia syndrome in a case and primary pulmonary hypertension in one case.**

Conclusion: **Lung transplantation results in improved survival and better quality of life. It still remains still very expensive and needs strict compliance of the patient and continous support of the community. The mortality risk factors in our study are bilateral lung transplantations, mismatched recipient donor body surface areas and age and operating in our early experience. Learning curve and good flow volume of cases can improve the outcome.**

Thoracic

Lung transplantation results in better survival and quality of life. It still remains expensive, with cost-effectiveness limited by substantial mortality and morbidity and high costs. Less immunosuppression costs and improvements in quality of life after transplantation can improve and compensate for the cost-effectiveness of lung transplantations.(1) The success of lung transplantation has improved over time as evidenced by better long-term survival and functional outcomes. Despite the success of this procedure, there are numerous problems and complications that may develop over the life of a lung transplant recipient. With proper monitoring and treatment, the frequency and severity of these problems can be decreased. However, significant improvement for the overall outcomes of lung transplantation will only occur when better methods exist to prevent or effectively treat chronic rejection.(2) Despite rarity of donor organs availability and legal and ethical issuues, improved surgical techniques, as well as increased experience and broadened knowledge implemented many changes in pulmonary transplantation. (3)

MBC-J 16
PO Box 40047
Jeddah 21499, Saudi Arabia
Tel: 9662-667-7777 ext 5234
Fax: 9662-6639581
Sameh001@yahoo.com

Methods:

This is a retrospective study presenting our limited preliminary experience in lung transplantations in KFSH&RC;Jeddah. It includes 7 cases of end stage respiratory diseases. Once the diagnosis is achieved as end stage respiratory disease, the pulmonlogists and we start the pretransplant work up and the patient is on our lung transplant waiting list, once we have a suitable cadaveric donor, our harvest team moves to get the donor lung(s). Our Standard Transplant Protocol includes the pre transplant work up, donor search, graft harvest, immune status and HLA system evaluation, our standard surgical technique following the universal world wide surgical techniques described in the literature, postoperative care, in the ICU and transplant ward and immunosuppression regimens. We consider the donors suitable if they were available and fulfilled the following criteria: (1) < 60 years old, (2) clear chest radiograph, (3) a Pao2/Fio2 ratio of > 300 while receiving 100% Fio2 and 5 cm H2O positive end-expiratory pressure, and (4) airways free of purulent secretions or gastric contents on direct examination with bronchoscopy as per Christie et.al.,2003.(3) Donor lung preservation utilized 500 µg of prostaglandin E1 and modified Eurocollins solution or Perfadex solution. The patient is positioned in the thoracotomy position with a groin access. Adhesions are lysed and the hilum is dissected. The pulmonary artery, the superior and inferior pulmonary veins, and the main stem bronchus are isolated. The pulmonary artery is ligated and divided followed by the pulmonary veins then the the main stem bronchus that is stapled and divided, and the native lung is explanted. The donor lung is then placed in the recipient's chest and covered with saline slush and iced laparotomy pads. Once the bronchial anastomosis is complete, the anastomoses of the pulmonary veins are done with a left atrial cuff. The pulmonary arteries are anastomosed with 5-0 polypropylene suture. The pulmonary artery anastomosis is then de-aired. The lung is inflated, and the pulmonary artery clamp is temporarily released to allow flushing of air through the atrial suture line. The left atrial clamp is removed to allow retrograde de-airing of the atrial anastomosis. The pulmonary venous anastomosis is then secured. After hemostasis is ensured, 2 large chest tubes are inserted. The chest is closed in standard fashion. The double-lumen endotracheal tube is exchanged for a single-lumen tube and bronchoscopy is performed to evaluate the bronchial anastomosis. Standard posterior thoracotomy is the access for single lung transplantation(SLT) while the anterior thoraco sternotomy approach was used for bilateral lung transplantation (BLT). We always give methyl prednisolone as a 500-mg bolus prior to reperfusion of the allograft and then at a daily dose of 0.5 mg/kg tapered to 0.15 mg/kg by the third postoperative month. Cyclosporine was initiated immediately postoperatively to achieve a whole blood level of 250 to 350 ng/mL. Azathioprine therapy was initiated at a dose of 2 mg/kg/d. The patient s are intubated at a positive end-expiratory pressure of 5 cm H2O for a week to maintain optimal lung expansion. The chest drainage tubes were kept to suction between -15 to -20 cm H2O and then gradually decreased to water seal in a few days. Bronchoscopy was performed every 48 hours while the patient is still mechanically ventilated to assess the airway viability and to suction any retained secretions. chest physiotherapy was given every 8 hours. Postoperative immunosuppression was a triple-drug therapy consisting of cyclosporine or tacrolimus, azathioprine or mycophenolate mofetil, and corticosteroids. Acute rejection is clinically judged without trans-bronchial lung biopsy and treated with bolus injection of methyl prednisolone. Cytomegalovirus (CMV) prophylaxis with ganciclovir was given to all recipients for the first 3 months. We followed them in the outpatient clinic for any evidence of rejection, infection, bronchiolitis obliterans, blood tests including the cytotoxic drug levels, complete blood picture CBC, hepatic, renal function tests and follow up pulmonary function tests.

Results :

We had 7 cases of lung transplants, 6 females and a male. Three patients had bilateral, 3 left and 1 right lung transplantation. The mean age was 39.8 years, median age was 38 years and ranges between 25 and 60 years. The indications of lung transplantation were; Interstitial pulmonary fibrosis in 3 cases, Emphysema in one case, Emphysema in one case, Dysmotile Cilia syndrome in a cases and Primary pulmonary Hypertension in a case. We followed them up in our outpatient clinic. The mean follow up was 12 months, median was 10 months. The longest follow up was 2 years and the shortest was 2 days. The body surface area of the recipients was 1.8 Kgm/m while that of the donors was 1.94 Kgm/m. The CMV status was –ve for 6 of our patients and +ve for one. Three cases died during follow up one in 2 days, one in a year and the third one after 2 years. Extensive uncontrollable medical bleeding, Liver cell failure, CMV, DVT and Multi Organ Failure(MOF) were the accused causes of deaths. Their mean age was 43.3 years and median age was 38 years. There were 2 female mortalities and a male. They had their transplantation for Dysmotile Cilia syndrome, Primary PHTN and Post TB Table (1-2)

No.	7
Female	6(86%)
Male	1(14%)
Bilateral	3(43%)
Left	3(43%)
Right	1(14%)
Age In Years	
Mean	39.8
Median	38
Range	25-60
Interstitial pulmonary fibrosis	3 (43%)
Emphysema	1(14%)
Lymphangioleiomyomatosis	1(14%)
Dysmotile Cilia syndrome	1(14%)
Primary pulmonary Hypertension	1(14%)
Body Surface Area	1.8
CMV –ve	6 (86%)
CMV+ve	1 (14%)
Follow Up In Months	
Mean	12
Median	10
PostoPerative ventilation time	23hours
Tracheostomy (No.)	2
Compliance To therapy(No.)	5
Donors	
Age In Years	48
Sex	
Male	7 (100%)
Female	0
CMV Status	5 (-ve) and 2
Body surface area	NA.
Ventilation	1.92
Road traffic accident as the cause	8 hours
of death	7

Table(1); Details of the 7 cases. CMV; Cytomegalovirus.

Mortality		
No.	3	
Mean survival	1 year	
Median survival	1year	
Aetiology		
Liver cell Failure		
CMV+ DVT+MOF	1	(33.3%)
Bleeding	1	(33.3%)
Age	1	(33.3%)
Mean		
Median	43.3 years	
Sex	38years	
Female	2	(66.7%)
Male	1	(33.3%)
Indication For transplant		
Dysmotile Cilia syndrome	1	(33.3%)
Primary PHTN	1	(33.3%)
Post TB Emphysema	1	(33.3%)
Operation		
Bilateral	3 s	(100%)
Right	0	
Left	0	
Body surface are	1.7	
CBP use	3	
PostoPerative ventilation time	15 days	
Traceheostmy	0	
Compliance To therapy	1	
CMV status		
Preop.	(3)	
-ve	(100%)	
+ve	0	
Postop.		
-ve	1	(33.43%)
+ve	1	(33.43%)
NA	1	(33.43%)
Donors for the mortality cases		
Mean Age In years	54	
Sex		
Male	3	(100%)
Female	0	
CMV Status	1 (-ve) and 2 NA.	
Body surface area	2.3	
Ventilation	6 hours	
Road traffic accident as the cause of death	3	

Table(2); Details of the 3 mortality cases. Deep Vein Thrombosis (DVT). Multi-Organ Failure (MOF). Pulmonary Hypertension (PHTN) Cardio-Pulmonary Bypass (CPB).

Discussion:

The indications of lung transplantation in our series were; Interstitial pulmonary fibrosis in 3 cases, emphysema in one case, lymphangio leio myomatosis in one case, dysmotile cilia syndrome in a cases and primary pulmonary hypertension in a case. The universal indications include five categories: (1) COPD, including emphysema, chronic bronchitis, bronchiectasis, and bronchiolitis; (2) primary pulmonary hypertension (PPH), exclusive of secondary to congenital cardiac or vascular abnormalities; and (3) cystic fibrosis (CF); (4) fibrotic lung diseases including sarcoidosis and pulmonary fibrosis; and (5) "other," including eosinophilic granuloma, pulmonary alveolar microlithiasis, lymphangioleimatosis, and causes of secondary pulmonary hypertension.(4)

The indications of transplantation in the series of Smith 2006 included chronic obstructive pulmonary disease in 63% (33/52), idiopathic pulmonary fibrosis in 27% (14/52), and others in 10% (5/52). (5)

We had a small series of 7 cases of lung transplants, 6 females and a male. Three patients had bilateral, 3 left and 1 right lung transplantation. The mean age was 39.8 years, median age was 38 years and ranges between 25 and 60 years. Meyer 2005 reported a very large series of 821 cases of lung transplantation of whom 230 cases between 30-49 years with 369 cases between 50-59 years and 222 between 60-69 years.(6)

Three of our patients had bilateral, 3 left and 1 right lung transplantation. This is a very small preliminary series compared to other published ones as Kshettry 1997 who reported 77 single lung and 25 bilateral sequential lung transplantations (7) The age ranged between 25 and 60 years in our study while it ranged between 34 and 63 years in the series of Mason 2007. (8)

Our selection criteria of were the same as Egan et.al.,1995 which included age less than 50 with life expectancy less than 18 to 24 months. No significant renal or hepatic disease. The patient should be able to understand the medical regimen and comply to it. The patient should be also ambulatory and able to participate in rehabilitation program and psychologically stable no extensive adhesions in the pleura or resistant organisms in sputum. One patient was 60 years old in contrary to our selection criteria as it was early in our program that we were very keen to start it. (9)

The data of the United Netwok for Organ Sharing (UNOS) database suggested that recipients older than 60 years should not be excluded from the use of BLT based on concerns of higher early mortality. Long-term outcomes will better define any survival advantage of BLT over SLT, and future studies will help to determine the best use of BLT versus SLT in the older population. The conflict to perform BLT or SLT in elderly patients is still controversial in some major institutions. Although, the guidelines exist with age cutoff limits (BLT in patients < 60 and SLT in patients > 65), several centers perform BLT in all patients preferentially. The proponents and supporters of SLT in Lung transplatation claim that SLT is an easier procedure to perform with less iscaemic time, morbidity and mortality, better early graft function and better early survival than BLT, On the contrary, BLT supporters think that BLT recepients have less ventilation/ perfusion mismatches, are easier to handle and care for in the perioperative period, will provide better overall lung function, are protective against the physiologic manifestations of obliterative bronchiolitis, and offer a better long-term survival. Furthermore, although BLT does entail longer donor ischemic times for the second lung implanted, this has not translated into measurable adverse sequelae. (10)

The follow up in our series is short; (mean of 12 and median of 10 months) as our program is still preliminary and small one, we had 4 cases done in the last 1.5 year. This is one limitation of our study. During follow up, we do Chest X ray, clinical examination for any eveidence of infection, rejection or bronchiolitis obliternans, complete blood picture, pulmonary function tests and drug levels to keep the therapeutic blood levels of these drugs and adjust the drugs accordingly. As per Al Githmi et.al.,2006, Pulmonary function tests are routinely performed after transplantation as persistent decline in FEV1 of 20% or more of the baseline value in the absence of infection and acute rejection is a useful clinical surrogate. The Stanford University group demonstrated a decline in the forced expiratory flow (FEF 25–75%) to less than 70% of the predicted values occurring 4 months earlier than the 20% decline in FEV1 This appears to be a sensitive marker for the early detection of BOS. The methacoline challenging test at 3 months post-transplantation has predicted the early detection of Bronchiolitis obliterans syndrome (BOS) with a positive predictive value of 72% . We never did the methacoline challe

We had 3 mortalities (3/7; 43%) after a mean duration of 1 year due to extensive medical bleeding in

one case, fulminant liver cell failure in an other one and CMV+ DVT+MOF in the third patient. The indications of transplantations in those mortalities were dysmotile cilia syndrome, Primary PHTN and Post TB emphysema. All the three had BLT using cardiopulmonary bypass. Two of them were females and the last one was a male with a mean age of 43.3 years and median of 38 years. Two of them were CMV –ve while the third one died early without screening him for CMV and one was converted to CMV +ve postoperatively. There was a significant donor recipient mismatch of the age and body surface area.(54/43.3 and 2.3/1.7) respectively. The three mortalities were our first 3 cases. Airway complications have been a major factor limiting the development of lung transplantation. The majority of deaths post transplantation are due to bronchial dehiscence. Lung transplantation is the only solid organ transplant in which the systemic arterial blood supply is not routinely anastomosed at the time of transplantation, and bronchial complications have been attributed to ischemia of the donor bronchus. However, other factors such as rejection, immunosuppression, infections and or inadequate organ preservation may delay airway healing. Thus, whereas airway complications comprised up to 80% of the bronchial anastomoses performed before 1983, more recently, the risk of airway complication after transplantation ranges from 10 to 15% per anastomosis, with a related mortality rate of 2 to 3%. Despite these improvements, some controversy still remains regarding the ideal method for management of the bronchial anastomosis at the time of transplantation and thereafter, when a bronchial complication arises. (12)

Analysis of our results shows that bilateral lung transplantation, mismatched body surface areas and age and operating in our early experience are the mortality risk factors. Transplant surgeons carefully evaluate donor lung size to optimize matching to a prospective recipient. However, there is no consensus on the definition of best «size fit» or how to achieve it. Some surgeons size match using donor and recipient height values while taking into account recipient disease diagnosis. (7)

We tried to highlight the effect of body surface area mismatch and we found that high body surface area mismatch between donor recipient is a significant mortality risk factor. Van De Wauwer et.al.,2007 showed mortality in 2.6% of patients during the first year after lung transplantation. In their univariate analysis, they found the anastomotic type; the elescoping technique versus the end-to-end, recipient anastomotic length, donor ventilation between 50–70 hours and recipient male gender were significant predictors of air way complications. As per them, three fremained significant predictors in the multivariate analysis: telescoping tecactors hnique, recipient length and donor ventilation. (13).

Limitations Of The study:

This is a retrospective study with the limitations of retrospective studies with a very small sample size.

Conclusion:

Lung transplantation results in survival and quality of life gains but remains still very expensive and needs compliance of the patient and support of the community. The mortality risk factors are bilateral lung transplantation, mismatched recipient donor body surface areas and age and operating in our early experience. Cumulative learning curve and good flow volume of cases can improve the outcome.

References:

(1) Anyanwu AC, McGuire A., Rogers CA, and Murday AJ. An economic evaluation of lung transplantation. J Thorac Cardiovasc Surg 2002;123:411-420.

(2) Studer S.M, Levy R.D, McNeil K.and Orens J.B. Lung transplant outcomes: a review of survival, graft function, physiology, health-related quality of life and cost-effectiveness Eur. Respir. J., 2004; 24(4): 674 - 685.

(3) Harringer W, Wiebe K, Strüber M, Franke U, Niedermeyer J, Fabel H and

Haverich A. Lung transplantation – 10-year experience. Eur. J. Cardiothorac.

Surg., Nov 1999; 16: 546 - 554.

(4) Christie JD, Kotloff RM, Pochettino A, Arcasoy SM, Rosengard BR, Landis JR and Kimmel SE. Clinical risk factors for primary graft failure following lung transplantation. Chest 2003;124:1232–1241

(5) Smith PW, Wang H, Parini V, Zolak JS, Shen KR, Daniel TM, Robbins MK, Tribble CG, Kron IL and Jones DR. Lung Transplantation in Patients 60 Years and Older: Results, Complications, and Outcomes. Ann Thorac Surg 2006;82:1835-1841.

(6) Meyer DM, Edwards LB, Torres F, Jessen ME and Novick RJ. Impact of recipient age and procedure type on survival after lung transplantation for pulmonary fibrosis Ann Thorac Surg 2005;79:950-958.

(7) Kshettry VR, Kroshus TJ, Hertz MI, Hunter DW, Shumway SJ and Bolman, III, RM. Early and Late Airway Complications After Lung Transplantation: Incidence and Management. Ann Thorac Surg.1997;63:1576-1583.

(8) Mason DP, Brizzio ME, Alster JM, McNeill AM, Murthy SC, Budev MM, Mehta AC, Minai OA, Pettersson GB

and Blackstone EH. Lung Transplantation for Idiopathic Pulmonary Fibrosis. Ann. Thorac. Surg., 2007; 84: 1121 - 1128.

(9)Egan T.M., Detterbeck F.C., Mill M.R., Paradowski L.J., Lackner R.P., Odgen W.D., Yankaskas J.R., Westermann J.H., Thompson T.J., Weiner M.A., Crains E.L., Wilcox B.R. Improved results of lung transplantation for patients with cystic fibrosis. J Thorac Cardiovasc Surg 1995;109:224-235

(10) Nwakanma LU, Simpkins CE, Williams JA, Chang DC, Borja MC, Conte JV, and Shah AS. Impact of bilateral versus single lung transplantation on survival in recipients 60 years of age and older: Analysis of United Network for Organ Sharing database. J. Thorac. Cardiovasc. Surg., 2007; 133: 541 - 547.

(11) Al-Githmi I, Batawil N, Shigemura N, Hsin M, lee TW, He G and Yim A. Bronchiolitis obliterans following lung transplantation. Eur J Cardiothorac Surg 2006;30:846-851

(12) Alvarez A, Algar J, Santos F, Lama R, Aranda JL, Baamonde C, López-Pujol J and Salvatierra A. Airway complications after lung transplantation: a review of 151 anastomoses. Eur. J. Cardiothorac. Surg., Apr 2001; 19: 381 - 387.

(13) De Wauwer C V, Raemdonck DV, Verleden GM, Dupont L, De Leyn P, Coosemans W, Nafteux P, and Lerut T. Risk factors for airway complications within the first year after lung transplantation. Eur. J. Cardiothorac. Surg., 2007; 31: 703 - 710.