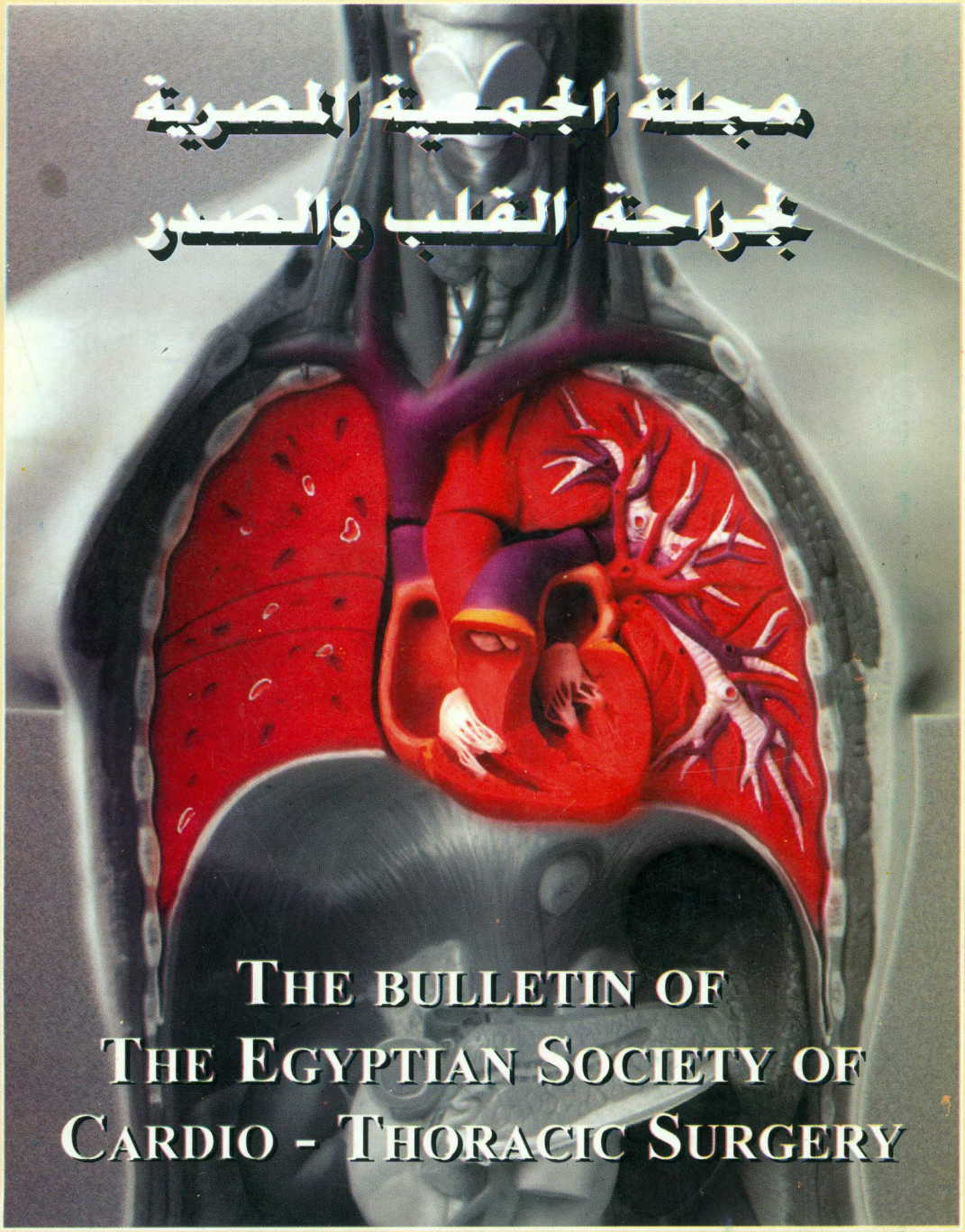
Vol. 3, June 1995

العدد الثالث ، يونيو ١٩٩٥

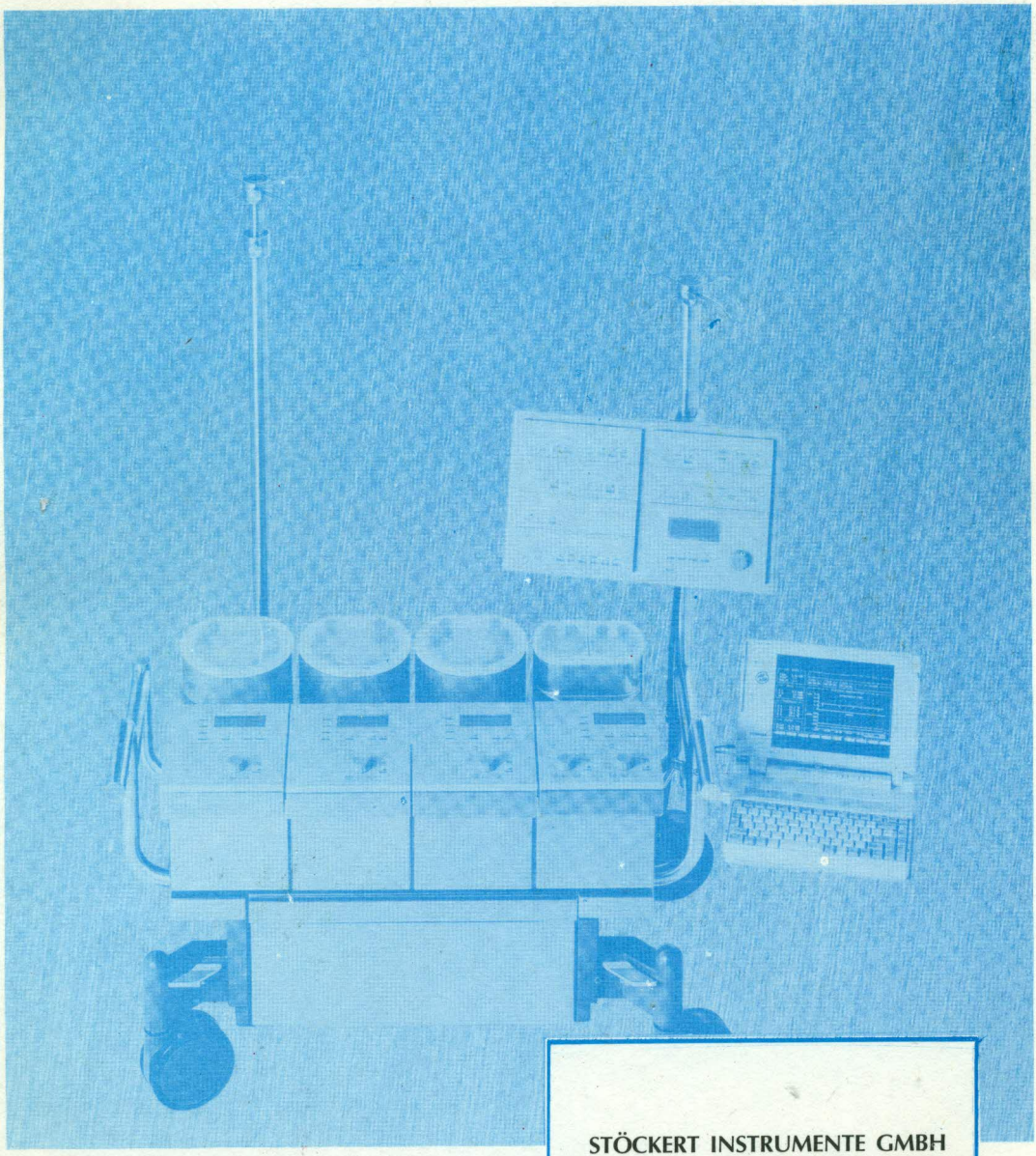


مجلة الجمعية المصرية لجراحة القلب والصدر

THE BULLETIN OF THE EGYPTIAN SOCIETY OF CARDIO - THORACIC SURGERY

Editor
Hassouna Saba F.R.C.S.

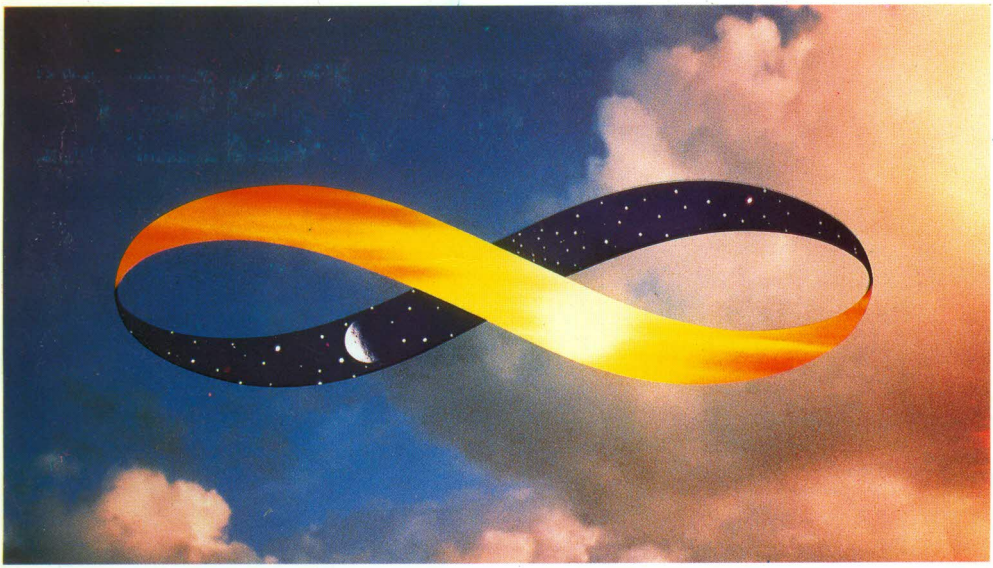
EXPERIENCE DECIDES.



STÖCKERT INSTRUMENTE GMBH
Lilienthalallee 5-7 · D 80939 München
Tel 089.32301-0 · Telex 5215952 stoe d
Fax 089.3234238

WE TAKE RESEARCH TO HEART

STÖCKERT
INSTRUMENTE GMBH



Introducing

The NEW Once-a-Day Calcium Antagonist

NORVASC*

(amlodipine besylate)

24 HOURS OF CONTROL AND PROTECTION IN HYPERTENSION AND MYOCARDIAL ISCHEMIA

Initial Therapy With Excellent Toleration

—Continuous day-and-night blood pressure control[1]

—Predictable 24-hour action allows increased physical activity for your angina patients[2]

—Low incidence of headache, flushing, dizziness and reflex tachycardia[3,4]

*NORVASC is a registered trademark of Pfizer Inc.

1. Heber ME, Brigden G, Al-Khawaja I, et al: 24 h blood pressure control with the once daily calcium antagonist, amlodipine. *Br J Clin Pharmacol* 1989;27:359-365.
2. Taylor SH, Lee P, Jackson N, et al: A four-week, double-blind, placebo-controlled, parallel dose-response study of amlodipine in patients with stable exertional angina pectoris. *Am Heart J* 1989;118(5)(pt 2):1133-1134.
3. Osterloh I: The safety of amlodipine. *Am Heart J* 1989;118(5)(pt 2):1114-1120.
4. Williams DM, Cubeddu LX: Amlodipine pharmacokinetics in healthy volunteers. *J Clin Pharmacol* 1988;28(11):990-994.

For further details, please consult prescribing information on back page.

Pfizer

Further information is available on request
Pfizer Egypt SAE 47 Ramses Street Cairo, Egypt.
* Registered trademark

Norvasc*
(amlodipine besylate)

24 HOURS OF CONTROL AND PROTECTION IN HYPERTENSION AND MYOCARDIAL ISCHEMIA

Pfizer Egypt

PRODUCT DOCUMENT

Name of Medicinal Product: Norvasc*

Qualitative and Quantitative Composition:

Active ingredient: Amlodipine

The tablets contain amlodipine besylate equivalent to 5 mg amlodipine.

Pharmaceutical Form:

Tablets

Clinical Particulars

Therapeutic indications

Amlodipine is indicated for the first line treatment of hypertension and can be used as the sole agent to control blood pressure in the majority of patients. Patients not adequately controlled on a single antihypertensive agent may benefit from the addition of amlodipine, which has been used in combination with a thiazide diuretic, beta adrenoceptor blocking agent, or an angiotensin-converting enzyme inhibitor.

Amlodipine is indicated for the first line treatment of myocardial ischemia, whether due to fixed obstruction (stable angina) and/or vasospasm/ vasoconstriction (Prinzmetal's or variant angina) of coronary vasculature. Amlodipine may be used where the clinical presentation suggests a possible vasospastic/ vasoconstrictive component but where vasospasm/ vasoconstriction has not been confirmed. Amlodipine may be used alone, as monotherapy, or in combination with other antianginal drugs in patients with angina that is refractory to nitrates and/or adequate doses of beta blockers.

Posology and method of administration

Dosage:

For both hypertension and angina, the usual initial dose is 5 mg amlodipine once daily which may be increased to a maximum dose of 10 mg depending on the individual patient's response.

No dose adjustment of amlodipine is required upon concomitant administration of thiazide diuretics, beta blockers, and angiotensin-converting enzyme inhibitors.

Use for children:

No experience is available on use of amlodipine in children.

Contra-indications

Amlodipine is contraindicated in patients with a known sensitivity to dihydropyridines.

Special warnings and special precautions for use

- **Use in patients with impaired hepatic function:**

As with all calcium antagonists, amlodipine half-life is prolonged in patients with impaired liver function and dosage recommendations have not been established. The drug should therefore be administered with caution in these patients.

- **Use in renal failure:**

Amlodipine is extensively metabolised to inactive metabolites with 10% excreted as unchanged drug in the urine. Changes in amlodipine plasma concentrations are not correlated with degree of renal impairment. Amlodipine may be used in such patients at normal doses. Amlodipine is not dialysable.

- **Use in the Elderly:**

The time to reach peak plasma concentrations of amlodipine is similar in elderly and younger subjects. Amlodipine clearance tends to be decreased with resulting increases in AUC and elimination half-life in elderly patients [1,2]. Amlodipine, used at similar doses in elderly or younger patients, is equally well tolerated (3). Therefore normal dosage regimens are recommended.

Interaction with other medicaments and other forms of treatment

Amlodipine has been safely administered with thiazide diuretics, beta blockers, angiotensin-converting enzyme inhibitors, long-acting nitrates, sublingual nitroglycerine, non-steroidal antiinflammatory drugs, antibiotics, and oral hypoglycemic drugs. Special studies have indicated that the co-administration of amlodipine with digoxin did not change serum digoxin levels or digoxin renal clearance in normal volunteers, and that co-administration of cimetidine did not alter the pharmacokinetics of amlodipine.

In vitro data from studies with human plasma indicate that amlodipine has no effect on protein binding of the drugs tested (digoxin, phenytoin, warfarin, or indomethacin).

In healthy male volunteers, the co-administration of amlodipine does not significantly alter the effect of warfarin on prothrombin response time (4).

Pregnancy and lactation

Safety of amlodipine in human pregnancy or lactation has not been established. Amlodipine does not demonstrate toxicity in animal reproductive studies other than to delay parturition and prolong labor in rats at a dose level fifty times the maximum recommended dose in humans. Accordingly, use in pregnancy is only recommended when there is no safer alternative and when the disease itself carries greater risk for the mother and fetus.

Undesirable effects

Amlodipine is well tolerated. In placebo controlled clinical trials involving patients with hypertension or angina, the most commonly observed side effects were headache, edema, fatigue, somnolence, nausea, abdominal pain, flushing, palpitations, and dizziness. Less commonly observed side effects in marketing experience include pruritis, rash, dyspnea, asthenia, muscle cramps, and dyspepsia. As with other calcium channel blockers the following adverse events have been rarely reported and cannot be distinguished from the natural history of the underlying disease: myocardial infarction and chest pain (5). No pattern of clinically significant laboratory test abnormalities related to amlodipine has been observed.

Overdose

In humans, experience with intentional overdose is limited. Gastric lavage may be worthwhile in some cases. Available data suggest that gross overdosage could result in excessive peripheral vasodilatation with subsequent marked and probably prolonged systemic hypotension. Clinically significant hypotension due to amlodipine overdosage calls for active cardiovascular support including frequent monitoring of cardiac and respiratory function, elevation of extremities, and attention to circulating fluid volume and urine output. A vasoconstrictor may be helpful in restoring vascular tone and blood pressure, provided that there is no contraindication to its use. Intravenous calcium gluconate may be beneficial in reversing the effects of calcium channel blockade. Since amlodipine is highly protein-bound, dialysis is not likely to be of benefit.

Pharmacological properties

Pharmacodynamic properties

Amlodipine is a calcium ion influx inhibitor (slow channel blocker or calcium ion antagonist) and inhibits the transmembrane influx of calcium ions into cardiac and vascular smooth muscle.

The mechanism of the antihypertensive action of amlodipine is due to a direct relaxant effect on vascular smooth muscle.

The precise mechanism by which amlodipine relieves angina has not been fully determined but amlodipine reduces total ischemic burden by the following two actions:

1) Amlodipine dilates peripheral arterioles and thus, reduces the total peripheral resistance (afterload) against which the heart works. Since the heart rate remains stable, this unloading of the heart reduces myocardial energy consumption and oxygen requirements [6,7,8]
2) The mechanism of action of amlodipine also probably involves dilatation of the main coronary arteries and coronary arterioles, both in normal and ischemic regions. This dilatation increases myocardial oxygen delivery in patients with coronary artery spasm (prinzmetal's or variant angina) and blunts smoking induced coronary vasoconstriction [9].

In patients with hypertension, once daily dosing provides clinically significant reductions of blood pressure in both the supine and standing positions throughout the 24 hour interval. Due to the slow onset of action, acute hypotension is not a feature of amlodipine administration.

In patients with angina, once daily administration of amlodipine increases total exercise time, time to angina onset, and time to 1 mm ST segment depression, and decreases both angina attack frequency and nitroglycerine tablet consumption [8,10].

In vitro studies have shown that approximately 97.5% of circulating amlodipine is bound to plasma proteins.

Amlodipine has not been associated with any adverse metabolic effects or changes in plasma lipids and is suitable for use in patients with asthma, diabetes, and gout.

Hemodynamic studies and a controlled clinical trial in NYHA Class II-III heart failure patients have shown that amlodipine did not lead to clinical deterioration as measured by exercise tolerance, left ventricular ejection fraction and clinical symptomatology. Studies have not been performed in patients with class IV heart failure [11].

Pharmacokinetic properties

Absorption

After oral administration of therapeutic doses, amlodipine is well absorbed with peak blood levels between 6-12 hours postdose. Absolute bioavailability has been estimated to be between 64 and 80% [1,12]. The volume of distribution is approximately 21 L/Kg [13].

Biotransformation/Elimination

The terminal plasma elimination half life is about 35-50 hours and is consistent with once daily dosing. Steady state plasma levels are reached after 7-8 days of consecutive dosing.

Amlodipine is extensively metabolised by the liver to inactive metabolites with 10% of the parent compound and 60% of metabolites excreted in the urine. Absorption of amlodipine is unaffected by consumption of food [14].

Pharmaceutical particulars

List of excipients

Microcrystalline cellulose, Dibasic calcium phosphate anhydrous, sodium starch glycolate, magnesium stearate.

Shelf life

36 months

Storage conditions

Room temperature (up to 30°C)

Nature and contents of container

Blister pack contains 10 tablets.

Instructions for use/handling

Use as directed by your physician. Keep out of the reach of children.

References

References are available on request in Pfizer-Egypt

Medical Department

47, Ramses street

Cairo, Egypt

* Registered trademark

PF - Egy - L - NOR - 1/94 - Tip In.

Pfizer

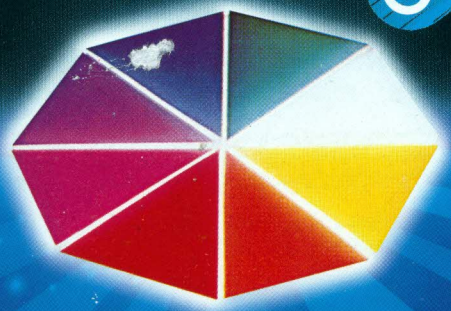
SUPERIORITY

Confirmed by

Efficacy

Toleration

Convenience



Cefobid* IM/IV q12h

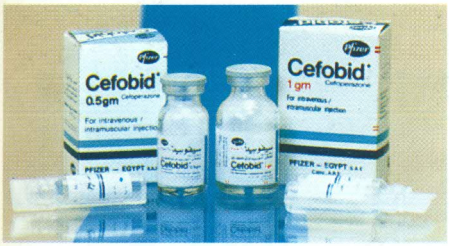
(cefoperazone sodium)

STANDS OUT

Excellent clinical response across a wide range of hospital infections[1]

Diagnosis	No. of patients	Clinical success[1]
Lower respiratory tract infection	205	95%[2]
Upper respiratory tract infection	119	84%
Hepatobiliary tract infection	113	83%
Septicemia	93	96%[2]
Meningitis	11	91%
Urinary tract infection	1,155	83%
Obstetric/gynecological infection	211	93%
Abdominal infection	133	90%
Post-operative/wound infection	12	91.6%
Osteomyelitis	14	78.5%
Dermatological infection	87	89%
Ophthalmological infection	26	85%

Superiority Makes It Stand Out



Usual daily dosage

Adults: *1-2 g IV/IM every 12 hours*

Infants and children: *0.5-1g IV/IM every 12 hours depending on the severity of the infection and bodyweight of the patient.*
or: *50 - 200 mg/kg/day in 2 divided doses every 12 hours.*

These daily dosages may be increased according to the severity of the infection.

References:

1- Ueda Y : Internal Med 1983:2:15-19.
2- Data on file, Pfizer International Inc.

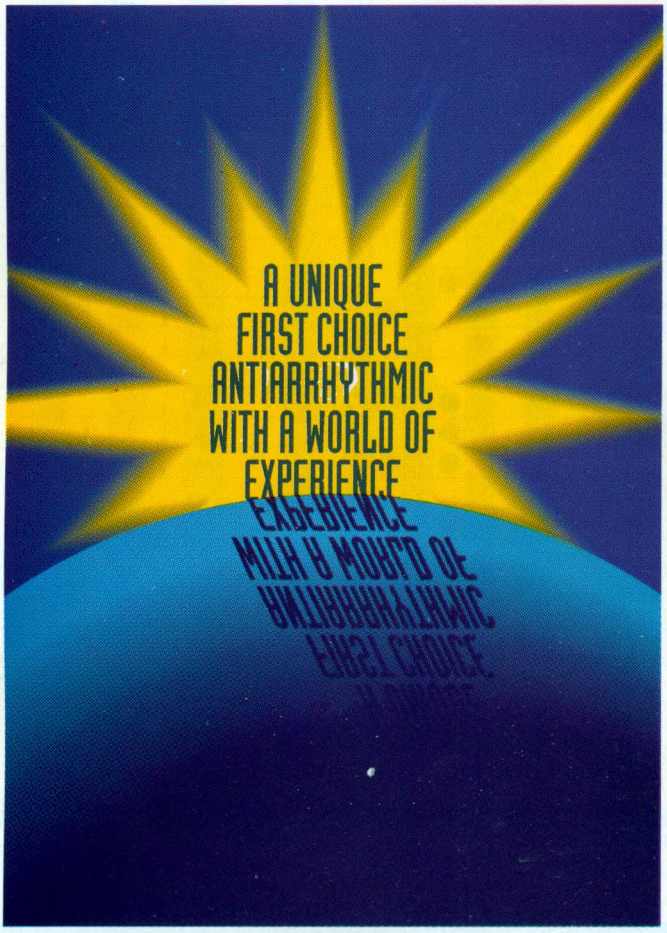
Pfizer

Further information is available on request
Pfizer Egypt SAE 47 Ramses Street
CAIRO - EGYPT

* Registered trademark

RYTMONORM®

Propafenone HCl



Also consider switching to RYTMONORM® because:

Unlike amiodarone Rytmonorm®:

* has no tissue accumulation.
* has no risk of pulmonary fibrosis.
* has no risk of thyroid impairment.
* has no risk of phototoxicity.
* has no effect on the QTc time.

Unlike disopyramide Rytmonorm®:

* has no anticholinergic side effects.
* has moderate ß-blocking effects.
* has a less negative inotropic effect.
* is only 1% excreted via the kidneys.

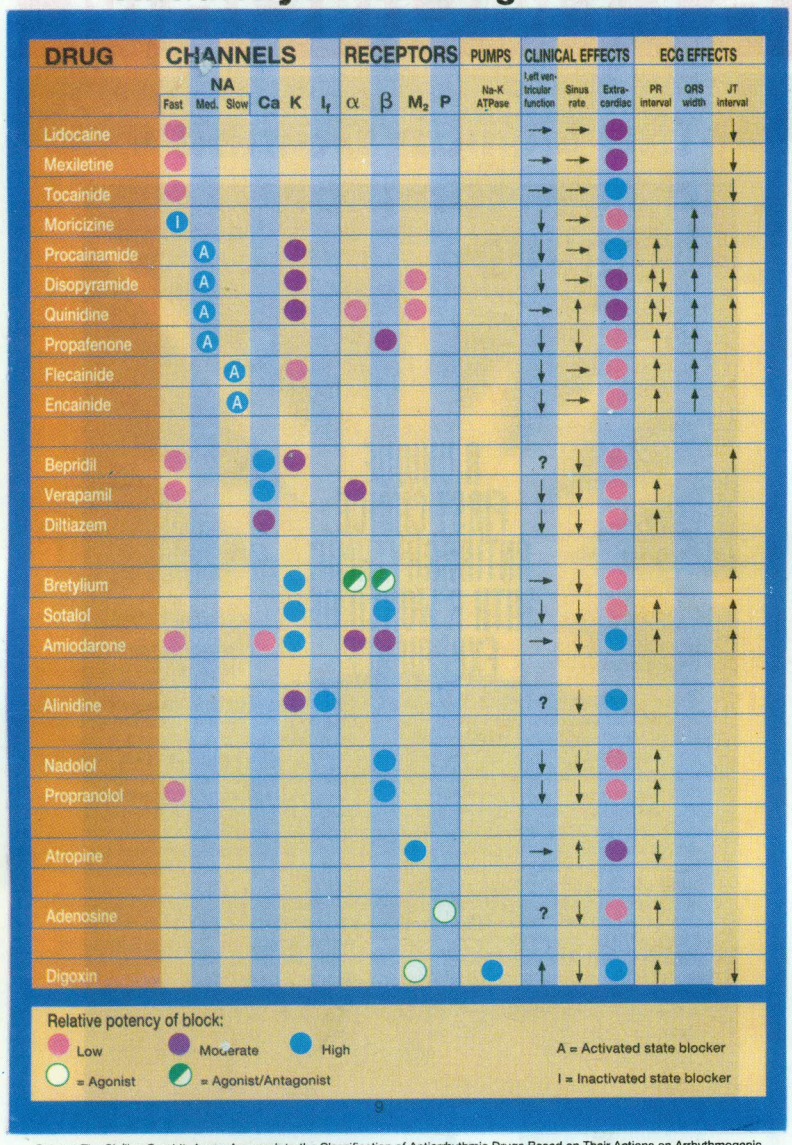
Unlike mexiletine Rytmonorm®:

* is effective in supraventricular and ventricular arrhythmias as well as WPW.
* has moderate ß-blocking effects.

Unlike tocainide Rytmonorm®:

* is effective in supraventricular and ventricular arrhythmias.
* has moderate ß-blocking effects.
* has no risk of pulmonary fibrosis.

The Sicilian Gambit Approach to Antiarrhythmic Drug Actions



Source: The Sicilian Gambit. A new Approach to the Classification of Antiarrhythmic Drugs Based on Their Actions on Arrhythmogenic Mechanisms. Task Force of the Working Group on Arrhythmias of the European Society of Cardiology. Circulation 1991; 84: 1831-1851

Abbreviated Prescribing Information

Composition:
1 film coated tablet of Rytmonorm® 150 mg contains 150 mg propafenone hydrochloride.

Clinical Data:
Rytmonorm® is a highly effective antiarrhythmic agent with a basic local anesthetic effect and a membrane-stabilizing effect on the cardiac muscle cell. It reduces depolarization rate & prolongs conduction time in the atria, AV node, and particularly, the His-purkinje system. Therefore the action of Rytmonorm® on cardiac arrythmias of varying origin is pronounced and reliable.

Indications:
All types of ventricular and supraventricular extrasystoles, ventricular and supraventricular tachycardias and tachyarrhythmias, even in WPW syndrome.

Dosage:
The dosage is as follows unless otherwise prescribed by the physician.
For initial and maintenance treatment a daily dose of 450-600 mg (1 tablet of Rytmonorm® 150 mg 3 times daily) is recommended. Occasionally an increase of the daily dose to 900 mg may be necessary (2 tablets of Rytmonorm® 150 mg 3 times a day). This daily dose should be exceeded only in exceptional circumstances and under strict cardiological control.

Contraindications:
Manifest heart failure, cardiogenic shock (except for shock induced by arrhythmia), severe bradycardia, pre-existing high degree sinoatrial, atrioventricular and intraventricular disorders of impulse conduction, sick sinus syndrome (bradycardia-tachycardia syndrome), manifest disorders of the electrolyte balance, severe obstructive pulmonary diseases, marked hypotension, myasthenia gravis.

Side Effects:
On rare occasions, particularly with higher initial doses, gastrointestinal disturbances, sporadically blurred vision and vertigo, very rarely fatigue and headache, restlessness, nightmares, sleep disorders, and psychological disorders may occur.
These symptoms disappear after reduction of the dose or discontinuation of the drug. Existing heart failure may deteriorate. In the severely damaged myocardium an undesired, marked effect on impulse conduction or myocardial contractility may be observed.
Since treatment with Rytmonorm® may be vital the drug must not be discontinued due to this adverse reaction without consulting the attending physician.

Interactions:
The possibility of potentiation of the effect of Rytmonorm® by local anaesthetics given simultaneously or by other drugs which inhibit the heart rate and/or contractility should be borne in mind. Rises in propranolol and metoprolol or digoxin plasma levels, respectively, under concomitant administration of Rytmonorm® have been reported.
There have been reports of rises of the propafenone plasma concentrations when taken simultaneously with cimetidine. Propafenone may potentiate the effect of oral anticoagulants.

Precautions:
In liver and/or severe renal impairment there may be drug accumulation after therapeutic doses. Even such cases may be well controlled with Rytmonorm® under strict ECG control.

Presentation: 10 film coated tablets (150 mg).

Further information available from : Knoll Scientific Office : 30 Michel Bakhoum Street - Dokki 12311 - Cairo, Egypt
Telephone : (202) 717270, 3603065 Telefax : (202) 3498308

knoll
BASF Pharma

Board of the Egyptian Society of Cardio-thoracic Surgery 1995

President	Hassouna Saba
Vice President	Mohamed El-Fiky
Secretary General	Hussein Gaafar
Assistant Secretary General	Mohamed Bassiouni
Treasurer	Ibrahim Haggag
Members :	Shaaban Abu Elela
	Gamal Abousenna
	Sherif Abd Elhady
	Moustafa Agha
	Mohamed EL Ashkar
	Anwar Balbaa
	Ezzeldin Mostafa
	Ahmed Nasr
	Ayman Shoeb
	Maher Shoeir

The bulletion of the

Egyptian society of cardio thoracic surgery

The Bulletion of

The Egyptian society of cardio thoracic surgery

Volume II-Number II

Contents June 1995

The bulletin of the

Egyptian society of cardio thoracic surgery

EDITOR

Hassonna M. Saba F.R.C.S.
20, Abd el Khalek Tharwat st.
Cairo - Egypt
Tel. Cairo 3912245
Fax 202/3417023

Information for authors

The Journal will consider for publication suitable articles on topics pertaining to thoracic and cardiovascular surgery. All communications relating to the editorial management of the journal should be sent to the editor, at the address given above.

Editorial policies: Statements and opinions expressed in the articles and communications herein are those of the author(s) and not necessarily those of the Editor and the Editor disclaims any responsibility or liability for such material. The Editor does not, guarantee, warrant, or endorse any product or service advertised in this publication.

Review: All articles are reviewed by one or more referees. Acceptance is based upon significance, originality, and validity of the material presented. If the article is accepted for publication, editorial revisions may be made to aid clarity and understanding without altering the meaning.

Manuscripts: An original and two copies of all material, including illustrations, should be submitted. Place author's name and page number in the upper right corner of each page. Use double spacing throughout. Include a covering letter stating that the material has not been previously published or submitted elsewhere for publication. Identify at the letter the name, address, business and home telephone numbers of the author to whom correspondence should be sent. Manuscripts and illustrations that are not accepted for publication will be returned to the authors except for one copy, which will be retained in confidence for three months, to facilitate answering additional correspondence relative to it, and then destroyed.

Title page: Make the title concise. List affiliation and academic degrees of author(s). Restrict number of authors to those making material contributions. Include where the work was done, sources of support (if any),

and name, address, and business and home telephone numbers of individual to whom correspondence and reprint requests are to be addressed.

Abstract: On the first page of the manuscript, an abstract of approximately 150 to 200 words, according to the length of the article but not exceeding one double-spaced, typed page, should be provided. The abstract in an explicit fashion should summarize the data and present the inferences. Tables are not to be included, and acronyms are not to be used. the use of an abstract eliminates the need for a summary.

References: References should be numbered serially in the text and listed, on a separate sheet, double-spaced, at the end of the paper in that order:-

For journals - authors' names and initials, title of article, journal name, date, volume number, and inclusive pages (list all authors when six or less; when seven or more, list only three and add et al.) in that order

Illustrations: Illustrations (three sets of glossy prints) should be numbered in the order of their mention in the text, and marked lightly on the back with the author's name and an arrow to indicate top edge. Original drawings or graphs should be prepared in black India ink or typographic lettering. All lettering must be done professionally and should be in proportion to the drawing, graph, or photograph. Do not send original art work, x-ray films, or ECG strips. Glossy print photographs, 3 by 4 inches (minimum) to 5 by 7 inches (maximum), with good black-and-white contrast or colour balance are preferred. Consistency in size within the article is strongly preferred. Any special instructions regarding sizing should be clearly noted. Suitable legends should be typewritten, double-spaced, and listed on a separate sheet of paper and included at the end of the manuscript. Illustrations for papers that have been accepted for publication will not be returned unless specifically requested by the author.

Tables: Tables should be self-explanatory and should supplement, not duplicate, the text. Type on pages separate from the text. Provide a brief title for each. Abbreviations used in tables should be defined at the bottom of the table.

Reprints: 20 Reprints of articles will be supplied, to the author at cost price. Indivdual reprints of an article should be obtained through the author.

Business communications: Communications of a business, nature and all advertising communications should be addressed to the editor.

EDITORIAL

Thirty years' Experience in the Management of Bronchial Carcinoma

Raymond Hurt FRCS

Formerly Consultant Thoracic Surgeon, North Middlesex Hospital and St. Bartholomew's Hospital, London.

I have been to Cairo several times to visit your ancient sites but this is the first time I have attended a medical conference here and it is indeed a very great honour to address your Society. Professor Saba, thank you very much indeed for allowing me this privilege.

It is clearly impossible to cover completely such an important part of thoracic surgery, which can be realistically described as the modern epidemic of lung cancer since World War II. I would therefore like to emphasise certain aspects of this subject, as it has developed during recent years, in particular the trend towards more conservative resection and the introduction of preoperative staging of the carcinoma to exclude patients from unnecessary exploratory operations and reduce the "open and close" rate.

The history of thoracic surgery is a fascinating subject and it is not alawys realised how recently the speciality has developed, although it does in fact go back to Hippocrates, who knew how to diagnose and drain an empyema. However, thoracotomy only really became a practical procedure with the introduction of the endotracheal tube for anaesthesia early this centrury, and subsequently it became a reasonably safe procedure with the ready availability of blood transfusion and the use of antibiotics during world War II. It was only then that thoracic surgery became established as a speciality in its own right.

Until 1930 lung resection was a hazardous procedure but in the following ten years immediately before the second World War the technique for partial and complete lung resection became standardised through the work of Churchill in the United States and Crafoord in Sweden.

First Resections

The first resection for cancer of the lung was carried out by a 33 year old English surgeon Morriston Davies (Fig. 1) as early as 1912 [1]. This was by a lobectomy and suprisingly by a dissection technique almost identical to that used today, though alas his patient died from a postoperative empyema eight days later. Lung cancer was not common at that time and most lung resections were for bronchiectasis.

Lecture delivered at the second meeting of the Egyptian Society of Cardiothoracic surgery Cairo, January 1995

Fig. 1: Morriston Davies (1879-1965).

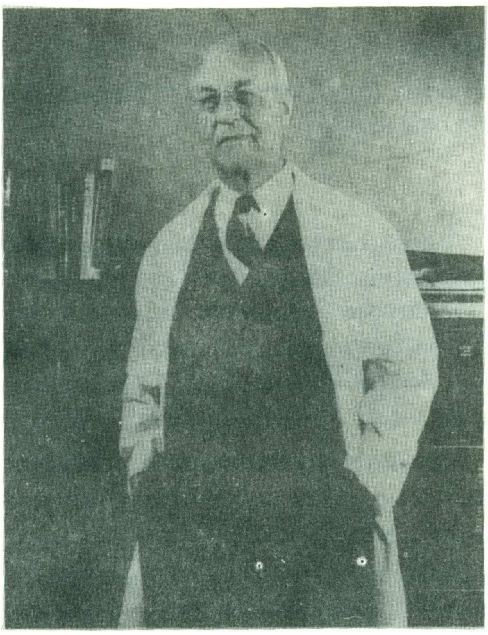


Fig. 2: Evarts Graham (1883-1957).

The first pneumonectomy for carcinoma was carried out in the Untited States in 1933 by Evarts Graham (Fig. 2) [2], who himself died from the disease just over twenty years later.

Diagnosis

Sputum cytology is increasingly being carried out, it is important that the report is signed so that its reliability is known, according to the experience of the pathologist who did the examination. The importance of who has written a report applies, of course, to all reports, not only to those of the sptum.

Always have a good look at the mouth, in case the radiological appearances are due to an inhalation pneumonities from dental sepsis. Even if a cancer is present and operation is required, it is wise to deal with the dental infection first.

Pre-operative assessment

The preoperative assessment of a patient falls into two parts; staging of the extent of the growth and deciding on the fitness of the patient for lung resection.

1. How extensive is the carcinoma outside and inside the chest?

Outside the chest metastases must be excluded by CT scan and bone scan. If there is doubt concerning liver secondaries we have found that ultrasound is more valuable than CT scan.

Inside the chest the value of staging by CT scan, mediastinoscopy and in some cases anterior mediastinotomy, has now been accepted, in particular for the presence of involved mediastinal glands on the contralateral side, which, of course, is always a contraindication to operation. The TNM classification is now generally accepted, though the N2 group is unsatisfactory in that it does not differentiate paratracheal glands from carinal glands. Staging of the cancer has undoubtedly been one of the most important developments in recent years. It has dramatically improved patient selection and has led to a reduction in the "open and close" rate from 10-15% to about 5%. Innumerable articles have now been published in an attempt to estimate accurately the prognosis after resection of a carcinoma with involved mediastinal glands and therefore to exclude from operation those patients with a poor prognosis. The statistics are complicated and confusing, and sometimes one wonders how significant some of them are.

On the CT scan there is often difficulty in knowing whether the enlarged glands are actually involved by growth or merely enlarged due to infection. Even at operation this is sometimes difficult to decide-if the glands are hard then there is no doubt but if they are only firm it may be quite impossible to know without histology. I have had many cases where glands were thought to have been involved, have been resected and histology has shown them to be enlarged due to infection only. In these cases frozen section would be of value, though I myself have not used this.

Some radiological reports go beyond what a radiologist should say: "The carcinoma has metastasised into the pretracheal nodes, one of which is pathologically enlarged. This tumour is therefore thought beyond resection"-I think it is the surgeon himself who should make this decision and I am sorry to say that some chest physicians, who so often are the initial investigators of lung cancer, are being led astray by this type of report, so that some patients are being denied operation, which is the only possible curative procedure.

There is continued controversy concerning the surgical management of non-small cell crcinoma in patients with N2 disease (i.e. ipsilateral mediastinal node involvement), and also on the prognosis after resection if these nodes are involved, ranging from the low figure of 9% described by Pearson [3] in Toronto to the 30% described by Martini [4] in New York. This can only be due to differences in the selection of patients, resulting in the inclusion or exclusion of different subsets of N2 disease, as judged by mediastinoscopy and anterior mediastinotomy. Neither of these investigations are able to assess pulmonary

ligament nodes or paraoesophageal nodes, which are especially significant in lower lobe growths. Furthermore the extent of mediastinal lymph node biopsy varies considerably in different reports and during recent years a much more extensive medistinal exploration has been performed in some surgical units. This also adds confusion to the statistics, the assessment of which was well summarised by Shields in 1990 [5].

As a corollary to the extensive staging now practised by most thoracic surgeons, it appears that the prognosis depends on the extent of node involvement and not on tumour size. This is in keeping with general pathological principles and with what is true of tumours in other parts of the body.

Concerning small-cell (or oat cell) carcinoma there appears to be two types of tumour. Firstly the more common highly malignant type, associated with early widespread metastases, amenable only to cytotoxic therapy and associated with a very poor prognosis. But secondly there does appear to be a very small group of patients in whom the tumor is more peripheral and not so malignant. [6] This type is often diagnosied only after its resection and is not associated with a poor prognosis. This raises problems with the management of such cases diagnosed by sputum cytology only and it may be that some such patients are being denied surgical resection.

The generally accepted procedure in the United kingdom at the present time is first to do a CT scan on all patients who may be suitable for thoracotomy. If mediastinal glands are present then mediastinoscopy is performed and, if the growth is in the left upper lobe, then an anterior mediastinotomy as well.

2. Is the paient fit for operation ?

a) Age of the patient. This depends on the patient's physiological age and not on his actual age. Many patients of 50 years are physiologically twenty years older because of constitutional changes or other associated disease, whereas some patients of 75 are no older physiologically than 55. I have done several lobectomies on patients over the age of 80 who have had a satisfactory quality of life after operation. But having said this, one has to admit that a pneumonectomy over the age of 70 does have a high complication and mortality rate.

b) Chronic bronchitis is so often also present as a smoking related disease, as well as the carcinoma, that it must be considered a very major risk factor. This is best assessed by getting the patient to cough and listening, without the use of a stephoscope, to hear if he sounds "chesty". Bronchospasm, heard with the stethoscope, is, of course, very significant.

c) Examination of the chest is important and a "barrel-shaped" chest, especially in the presence of bronchospasm, is also a significant factor, for these patients take thoracotomy badly.

d) Hypertension itself is not important, unless associated with coronary artery disease.

e) Lung function studies are often difficult to interpret. There are no hard and fast figures but the vital capacity should preferably be greater than 2.5 litres, the FEV1/VC ratio should preferably be greater than 45% and the peak flow should preferably be greater than 200 l/min, BUT far and away the best test is to get the patient to walk up two flights of stairs, and then take his pulse rate and assess his breathing. If the pulse rate has not increased much and if he can still talk easily, this, in my experience, is by far the most reliable test, whatever the other more sophisticated tests show. In other words clinical examination is still very important in the assessment of a patient for possible lung resection.

3. I should like to emphsise that these are NOT contraindications:

a) Phrenic nerve involvement in the presence of a lower lobe growth - the pericardium often appears to act as some sort of barrier to spread and an intrapericardial resection may still be possible. On the other hand if the growth is in the upper lobe then a phrenic paralysis does imply that the growth is inoperable.

b) A pleural effusion-even if blood-stained-unless malignant cells are also present in the fluid. The blood-stained fluid may be due to an infarct distal to the tumour.

c) A raised ESR is not significant as regards operability, though some physicians would not agree.

d) Chest wall involvement by direst extension of the growth on the chest radiograph-though one must admit that the recurrence rate in these cases is somewhat high.

e) Tumour at the origin of the main bronchus at bronchoscopy-sometimes the tumour is a polypoid growth and its attachment to the bronchial wall may be much further distal. This can be assessed at the time of bronchoscopy.

f) Hilar glands on CT scan. These may be inflammatory or, if neoplastic, resectable and the patient should therefore be advised thoracotomy and given the chance of a cure, unless mediastinoscopy has shown them to be hard and fixed.

Operative technique

The radical pneumonectomy operation was developed soon after World War II. Allison (Fig. 3) in England described the technique of intrapericardial division of the pulmonary veins and this led directly to the radical right pneumonectomy operation described by Brock in 1955 [7]. This operation involves a radical dissection of the mediastinum to remove the whole of the lymphatic drainage area alongside the trachea, as well as the lung itself. On the left, of course, such a radical dissection is not possible because of the aortic arch.

Dissection of the pulmonary vessels

a) Once inside the fascial sheath surrounding the vessel the right angled O'Shaughnessy clamp is marvellous for dissection - it has

Fig. 3: Philip Allison (1907-1974).

Fig. 4: Clement Price Thomas (1893-1973).

the correct angle and has a blunt end. The fascial sheath surrounding the vessel must first be picked up with fine-toothed forceps and cut with scissors - then the clamp can be readily passed around the vessel.

b) During a lobectomy don't tent up the arterial or venous branches too much during their ligation, or the ligature may slip off.

c) Price Thomas (Fig. 4) operated on king George Fifth in 1951 and removed his lung for cancer. The Price Thomas manoeurve, which I learnt when his registrar in the 1960s, facilitates division of the right pulmonary artery in difficult cases. The condensation of tissue between the pulmonary artery and the superior vena cava should be deliberately divided with scissors so as to allow the pulmonary artery to be separated from the superior vena cava by blunt dissection; this will provide a considerable extra length of artery for its ligation. I also learnt from Price Thomas how important it is to sit on the pa-

tient's bed when talking to him, so as to be on the same level and put the patient at ease.

d) The right pulmonary artery can also be exposed medial to the superior vena cava and this may be necessary in some cases of extensive growth or in order to control haemorrhage after accidental damage to the pulmonary artery during its dissection. On the left side the pulmonary artery can be divided proximal to the obliterated ductus, in which case it should be closed with a continuous suture, and not merely ligated. In this situation there is a tendency for a simple ligature to slip off.

Closure of the bronchus

For closure of the bronchus there is now an increasing tendency to use the stapling gun, if this is done it is important in a pneumonectomy to use the longer length of staple (i.e. 4.8 mm) as otherwise secure closure may not be obtained. An additional precaution, thought by many to be important in the prevention of a fistula, is to insert a continuous non-absorbable suture along the distal edge of the stapled bronchus.

I somewhat reluctantly became converted to this technique, though many surgeons in England still close the bronchus by hand suture, mostly using the technique originally described by Reinhoff in the United States in the 1930s, in which the posterior membranous wall of the bronchus is approximated to the C-shaped bronchial cartilages (Fig. 5) [8]. If this technique is used I think the use of figure-of-eight stainless steel sutures ap-

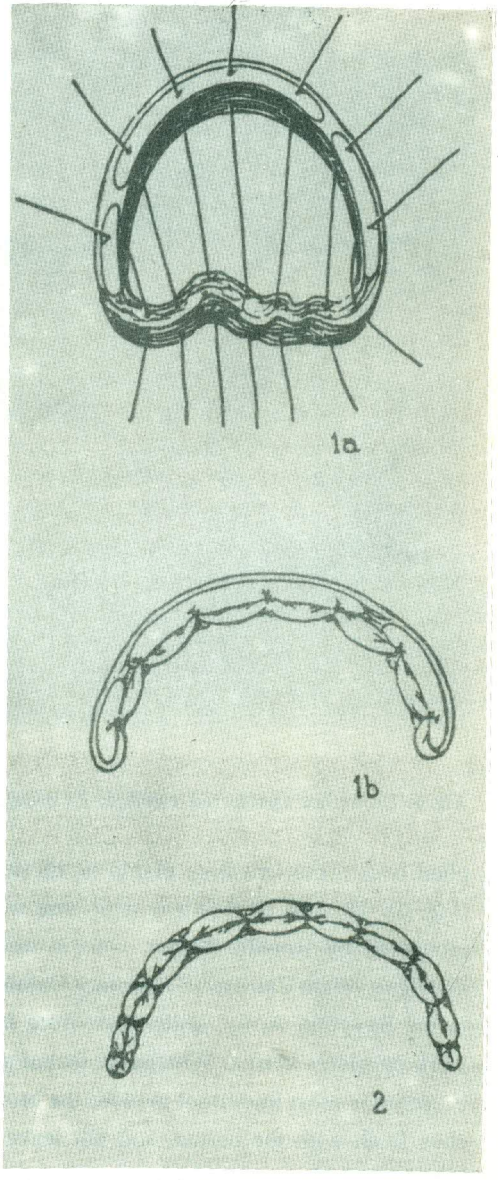


Fig. 5: Rienhoff's method of bronchial closure by approximation of the mucosa to the horse-shoe shaped bronchial cartilage.

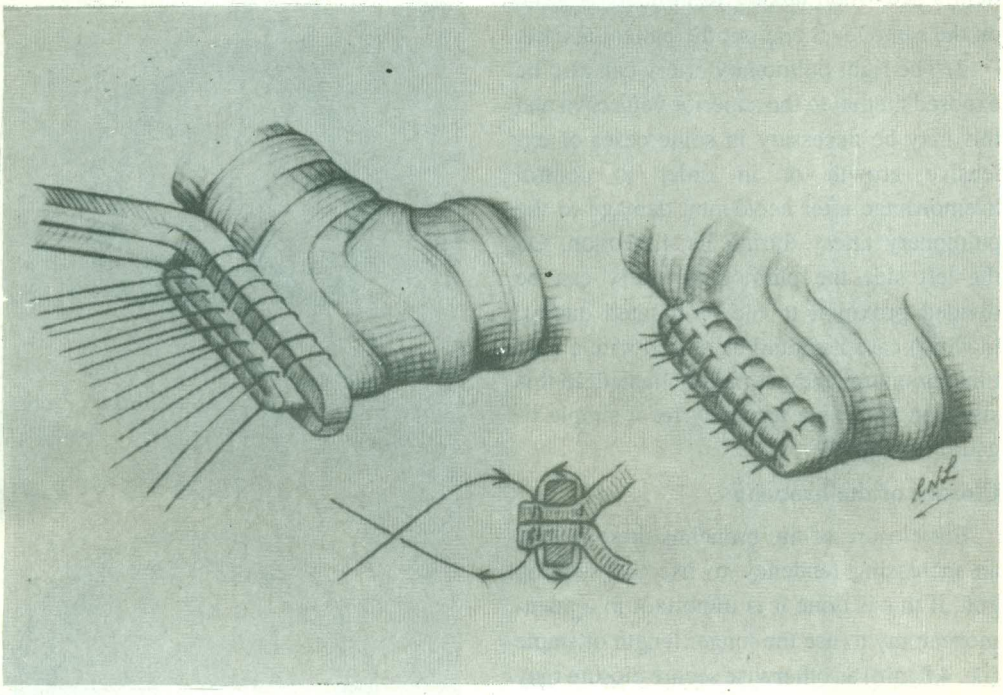


Fig. 6: The author's preferred technique for bronchial closure with figure-of-eight stainless steel wire sutures.

plied over a non-crushing clamp is the best (Fig. 6). The use of stainless steel wire was pioneered by Ronald Belsey, who is well-known to you in Cairo as an honorary Colonel in the Egyptian Army, quite apart from his work on hiatus hernia. Whichever technique is used it is most important to close the bronchus flush with the carina, and not leave a long bronchial stump, which is an important cause of a postoperative fistula.

Some surgeons in England, notably in Manchester, [9] use catgut for bronchial suture - I think this is unwise and I think that a non-absorbable suture is preferable.

Bronchoplastic procedures

Bronchoplastic procedures were developed in the 1950s, first by Gebauer from Honolulu for the treatment of a tuberculous bronchostenosis. He used a full-thickness skin graft, strengthened by a lattice of stainless steel wire, which was inserted into the stenosed main bronchus. Soon afterwards upper lobectomy for carcinoma by sleeve resection of a main bronchus was developed

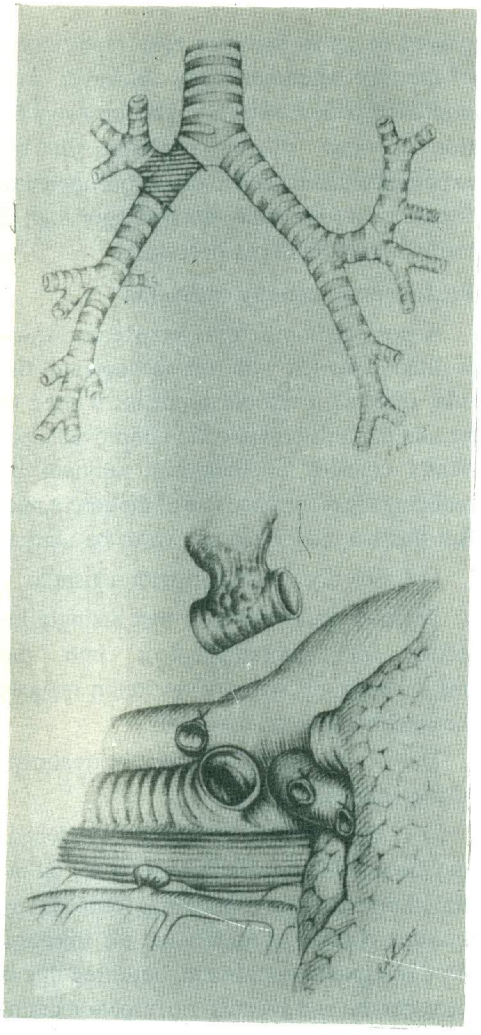


Fig. 7: Upper lobectomy by sleeve resection of the main bronchus.

simultaneously by Paulson in the United States and by Price Thomas in England for growths at the origin of the upper lobe bronchus which would otherwise have required a pneumonectomy (Fig. 7). Somewhat surprisingly there were few postoperative complications. If there is a discrepancy in size between the main bronchus and the intermediate bronchus then rèmoval of a wedge from the main bronchus will allow a satisfactory anastomosis to be made. Price Thomas also reported the resection of a sleeve of pulmonary artery as well as the main bronchus.

These bronchoplastic techniques of sleeve lobectomy and Gebauer skin graft have been dramatically extended by Grillo of Boston, and also other surgeons, to include resection of the carina itself with subsequent anastomosis of the left main bronchus to the trachea or side of the right main bronchus, and also the right main bronchus to the side of the trachea or the left main bronchus [10].

Drainage after pneumonectomy

Remains a most controversial subject and there is a remarkable differece of opinion in the United kingdom concerning this - in fact surgeons are about equally divided. This is most surprising for such a fundamental point in operative technique. It is my personal opinion that drainage for 24 hours is always advisable - by an intercostal tube connected to an underwater seal, which is kept closed and released for one minute every hour. If this procedure is adopted any postoperative haemorrhage is immediately obvious - important because its recognition is not always easy after a pneumonectomy. I have seen patients die from haemorrhage after a

pneumonectomy without its true cause being recognised because of the absence of a drainage tube. There is no risk of infection if the tube is removed after 24 hours. Furthermore the necessity for any postoperative aspiration of the chest is avoided.

If the pneumonectomy space is not drained then the intrapleural pressures should be adjusted to a slightly negative level at the end of the operation - by a temporary intercostal tube in the second interspace when the patient is on his back.

Postoperative care

A minitracheostomy through the cricothyroid membrane, a technique devised by Matthews of Birmingham [11], has proved to be extremely valuable for the treatment of postoperative sputum retention. It has virtually removed the need for bronchoscopic aspiration of sputum. It can be introduced in the ward under local anaesthesia and will provide an easy route for tracheal suction, whilst at the same time allowing the patient to continue to cough with the tube in place. Recently a technique has been developed whereby artificial ventilation by an air jet can be maintined through this minitracheostomy.

Artificil ventilation can also be instituted even more effectively through an endotracheal tube. The modern plastic tubes can be left in place safely for 10-14 days without any risk of vocal cord irritation. If ventilation is required for a longer time then a tracheostomy will be necessary and in the United Kingdom the Bjork type, with an inferior flap of tracheal wall, is almost universally employed so as to facilitate easy replacement of the tube when necessary. However, with improved patient selection artificial ventilation is now seldom required. In my experience the incidence of a bronchopleural fistula is unfortunately depressingly high if artificial ventilation is necessary.

Postpneumonectomy empyema

A postpneumonectomy empyema, which is often associted with a bronchopleural fistula, remains a terrible problem. It used to be said that this complication improved a patient's chances of long-term survival by reducing the recurrence rate of the carcinoma. But this has now been shown to be untrue.

1) Cases not associated with a fistula.

There is some evidence that a single injection of chloramphenicol into the pneumonectomy space at operation reduces the likelihood of an empyema.

There is no agreed method for treatment of the empyema:

a) If the infection is not too virulent it may be possible to sterilise the cavity by initial thoracoscopic aspiration, followed by repeated pleural aspiration and the injection of an intrapleural antibiotic, but often this is unsuccessful and eventual rib resection drainage is necessary.

A few weeks later, when the infection has been controlled, the treatment advised in the 1960s and 1970s was a Roberts chest wall flap operation-i.e. an extensive modified thoracoplasty procedure in which a decostalised portion of chest wall was turned down as a flap onto the mediastinum to obliterate the

pneumonectomy space. This was usually successful but did cause a considerable degree of chest wall deformity and because of this other procedures have been adopted. The most popular treatment now is the daily irrigation of the empyema cavity until it is sterile, followed by chest wall closure. In many cases the space remains sterile, perhps surprisingly in view of the persistence of an intrapleural space.

2) Cases associated with a fistula.

This is a much more difficult problem and immediate rib resection drainage is vital to prevent an inhalation pneumonitis into the contralateral lung. When the infection has been controlled the fistula must be closed by a Roberts type flap thorcoplasty, with or without suture of a pedicled intercostal muscle graft or omentum onto the open bronchus. In a number of cases ultimate success will be achieved but the morbidity is high.

Bronchopeural fistula without empyema

A bronchopleural fistula occurs in a number of cases without pleural infection, sometimes several months or even years after operation. It usually occurs on the right side and is especially likely if an extensive paratracheal lymph node excision has been required, as this inevitably deprives the bronchial stump of some of its blood supply. On the left side I have never had a fistula and in these cases the bronchial stump disappears deep into the mediastinum and remains well covered by fibrin and therefore closed.

My practice has always been to close these fistulae by elective suture at thoracotomy, though it is not easy because of the proximity of the pulmonary artery, superior vena cava and right atrium. It is vitally important first to mobilise the bronchial stump, then to reamputate the bronchus and finally to re-suture the bronchus. Merely to insert a few sutures without re-amputation of the stump inevitably leads to a recurrence of the fistula.

Pain relief after thoracotomy

Various techniques have been used to relieve post-thoracotomy pain- in particular during the last 20 years freezing of the intercostal nerve (cryoablation). Some surgeons speak highly of this procedure but others, including myself, have found it to be unsatisfactory, mainly due to continued chest wall pain, discomfort and numbness due to the procedure itself. One of my patients on whom I had been persuaded to use this technique during a hiatus hernia operation developed persisting chest wall pain for several years, which defied all treatment and was far worse than his preoperative symptoms. Since then I have not used it.

Prognosis

Concerning the prognosis I should like to make just two points:

1. The presence of microscopic growth at the suture line is an interesting subject and 15 years ago a report from Liverpool gave

a surprising 23% five year survival in 64 such patients - all survivors having a small squamous crcinoma [12]. However, most surgeons would advise a further resection or postoperative radiotherapy in such cases.

2. The more lung cancer one sees the more difficult it is to give an accurate prognosis. One of my patients had a right lower lobectomy for an adenocarcinoma in 1964. Two years later in 1966 she developed a mass of paratrachel glands associated with superior vena caval obstruction, and was successfully treated by radiotherapy. She was still alive and well 22 years later. She has defied all the rules, even though the growth was an adenocarcinoma and would not have been expected to have responded to radiotherapy.

Video assisted thoracoscopic resection

I find it very difficult to know what to think of this new technique, a technique that may be called a "high-flying space-age" technique. It has produced a whole range of miniature instruments and is indeed a triumph of modern technology but I have had no personal experience of its use. It would seem to be a reasonable procedure for lung biopsy and the closure of small bullae but I can see little future for its use in lung resections, which are often difficult enough at open thoracotomy. Looking back over the years I can think of many resections that I and others have performed in which a tear in a pulmonary artery or vein, or even atrium, has been readily controlled by finger pressure and subsequent suture without any harm to the patient. Would it have been possible to do this in time if an emergency thoracotomy had to be done first? I doubt it, especially if the damaged vessel had been the pulmonary artery.

Prevention of cancer of the lung

It is a sad fact that in Europe the commonest cancer in men is lung cancer, as also in Scotland it is in women, and that this great killing disease is virtually preventable. And yet remarkbly little is done in the Western World to prevent it.

Twenty years ago the tax on tobacco virtually covered the cost of the National Health Service in the United kingdom - I am sure that this must be a significant fact.

In the United States there is now a campaign against cigarettes which is growing at remarkable speed and some States have banned smoking in all bars, shops and restaurants, and many legal battles are pending concerning possible compensation. In Europe there is now a certain amount of publicity, both by health warnings on cigarettes and on postage stamps, and we are increasingly following the lead of the United States concerning the banning of smoking in public areas.

It is intersting that the danger of smoking was known as long ago as 1604, when king James I of England wrote A Counterblast to Tobacco- "Smoking is a custom loathsome to the eye, hateful to the nose, and in the

black stinking fume thereof nearest resembling the horrible Stygian smoke of the pit that is bottomless."

FINALLY, in these days of often confusing statistics and information, such as we have from survival figures after resection for N2 disease, I should like to emphasise the importance of accurate information and from it logical deduction:

"I should never have drowned", he cried, as he sank in a river of average depth of three feet.

False assumptions may lead to serious consequences.

Acknowledgements

Figures 1-4 are reproduced from Meade, History of Thoracic Surgery, 1961, courtesy of C.C. Thomas, Springfield, Illinois and figures 6-7 from Rob and Smith, Operative Surgery, Thoracic Surgery, 1986, courtesy of Butterworths, London.

REFERENCES

1. Davies HM : Recent advances in the surgery of the lung and pleura. Br J Surg (1913) 1: 228-258.
2. Graham EA and Singer JJ : Successful removal of an entire lung for carcinoma of the bronchus. JAMA (1933) 101: 1371-1374.
3. Pearson FG et al : Significance of positive superior mediastinal nodes identified at mediastinoscopy. J Thorac Cardiovasc Surg (1982) 83: 1-11.
4. Martini N and Flehinger BJ . The role of surgery in N2 lung cancer. Surg Clin N Am (1987) 67: 1037-1049.
5. Shields TW . The significance of ipsilteral mediastinal lymph node metastases (N2 disease) in non-small cell carcinoma of the lung. J Thorac Cardiovasc Surg (1990) 99: 48-53.
6. Shore DF and Paneth M . Survival after resection of small cell carcinoma of the bronchus. Thorax (1980) 35: 819-822.
7. Brock RC and Whytehead LL : Radical pneumonectomy for bronchial carcinoma. Br J Surg (1955) 43: 8-24.
8. Rienhoff WF . A new operative technique for closure of the main bronchus. Bull Johns Hopkins Hosp (1937) 60: 372-373.
9. Sarsam MAR and Moussali H . Technique of bronchial closure after pneumonectomy. J Thorac Cardiovasc Surg (1989) 98: 220-223.
10. Kittle CF . Atypicl resections of the lung: Bronchoplasties, Sleeve Resections and Segmentectomies - their evolution and present status. Curr Probl Surg (1989) 26: 59-132.
11. Matthews HR and Hopkinson RB . Treatment of sputum retention by mini tracheostomy. Br J Surg (1984) 71: 147-150.
12. Soorae AS and Stevenson HM . Survivl with residul tumour on the bronchial margin after resection for bronchogenic carcinoma. J Thorac Cardiovasc Surg (1979) 78: 175-180.

Different techniques for mitral valve reconstruction in rheumatic and degenerative cases

Abstract

This work was done in the National Heart Institute and in 6th October Hospital from 1993 to 1995. 28 mitral valve repairs were done for pure mitral regurgitation. 16 cases were myxomatous degeneration and prolapse of the mitral valve and 12 cases were rheumatic in origin.

Wedge resection of the anterior leaflet was performed in one case (3.6%), 9 cases (32.1%) received only a Carpentier-Edwards ring, 3 cases (10.7%) Duran ring, 7 cases (25%) flexible ring from ST. Jude Medical, 3 cases (10.7%) wedge resection of the anterior leaflet with Carpentier-Edwards annuloplasty ring and 5 cases (17.8%) chordal shortening with Carpentier-Edwards ring in 2 cases, and with flexible ring from ST. Jude Medical in the other 3 cases.
There was one death at the 5th operative day from low cardiac output. Two cases had moderate degree of haemolysis which recovered completely after one month. One case needed reoperation and mitral valve replacement was done due to failure of repair.

Postoperative echocardiography showed decrease in the peak gradient across the mitral valve, good size valve area and no or minimal regurge. All cases were on oral anticoagulant for a period of six months after the repair.

Lotfy M. Eissa*, M.D., Maher Mousa*, M.D., Sherif Abdel Hady*, M.D., Samir El-Mahmoudy, M.D.**

* National Heart Institute. ** Al-Azhar University.

Introduction

Mitral valve prolapse is one of the most common cardiac abnormlities found in the general population. (1) It is a condition that is generally benign with infrequent serious complications, but it is also a condition that increases in frequency with age. (2) The most frequent complication is severe mitral regurgitation, because of progression of the disease that produces changes in the valve, such as ruptured chordae, which may eventually lead to marked regurgitation requiring operation. (3.4) Also, the valve may become infected at any time with endocarditis requiring operation. (5,6) In this work we study the different surgical techniques for mitral valve reconstruction in cases of mitral valve regurgitation due to rheumatic or degenerative disease.

Material and Methods

From January 1993 Until January 1995, 28 patients with mitral valve regurgitation underwent mitral valve repair in the National Heart Institute and 6th October Insurance Hospital. There were 15 women and 13 men, ranging in age from 7 to 26 years (mean 18 years).

Preoperatively all patients were examined clinically, and investigation, included E.C.G, X-ray and echocardiography. Echocardiographic study is very important because it gives a very good idea about the site of regurge and its degree, the site of prolapse, the valve area, ruptured chordae and the peak gradient across the mitral valve.

Operative Technique

All patients were operated on with cardiopulmonary bypass, hemodilution, bicaval cannulation, total body hypothermia down to 27°C, local hypothermia and intermittent crystalloid hyperkalemic cardioplegia. Ring annuloplasty was performed on 27 of 28 patients; 9 had Carpentier-Edwards ring only, 7 had biflexible ring from ST. Jude Medical, 3 had Duran ring, 3 had wedge resection of the anterior leaflet with Carpentier-Edwards ring, chordal shortening was done in five patients with flexible ring from ST. Jude in 3, and with Carpentier-Edwards ring in 2, and one patient had wedage resection of the anterior leaflet with no ring.

The mitral valve after repair was tested by small size catheter passed through the mitral valve from the opened left atrium to the left ventricle, and this catheter connected to a syringe (size 50 c.c), then we inject saline until the left ventricle is distended, and we see the degree and the site of regurge. Another method for testing the mitral valve by removing the aortic clamp and we induce aortic regurgitation by compression on the aortic annulus between two fingers, the left ventricle is distended and we see any degree of mitral regurgitation.

All patients had a two-dimensional echocardiogram after the operation and before hospital discharge, to evaluate the mitral valve area, peak mitral gradient, and degree of MR. All patients (in sinus rhythm and in chronic atrial fibrillation) received anticoagulation with warfarin or dindevan for a period of 6 months.

One patient only, needed reoperation on the second postoperative day, because of low cardiac-output, and by echocardiography there was severe M.R., at reoperation operation, there was complete rupture of the sutureline in the anterior leaflet, the mitral valve was removed and mitral valve replacement was done.

N.B.: This patients had only wedage resection of the anterior leaflet with no ring annuloplasty.

Results

Different surgical techniques for mitral valve repair were done in 28 patients over a period of two years, for primary pure mitral regurgitation (Table 1).

Table I: Operative details in 28 patients undergoing mitral valve repair.

Type of Repair	No. of Patients	%
(1) Wedge resection of the anterior leaflet with no ring annuloplasty	1	3.6 %
(2) Carpentier-Edwards ring annuloplasty	9	32.1%
(3) Flexible ring (ST. Jude Medical)	7	25%
(4) Duran ring	3	10.7%
(5) Wedage resection of anterior leaflet & Carpentier-Edwards ring annuloplasty	3	10.7%
(6) Chordal shortening & Carpentier-Edwards ring.	2	7.1%
(7) Chordal shortening & flexible ring from ST. Jude Medical	3	10.7%
Total		28

In 16 patients, the regurge was due to myxomatous degeneration and prolapse of the mitral valve, and in 12 patients, there was a definite history of rheumatic fever.

There was no operative death. One patient required reoperation (3.6%) due to failure of the repair and low cardiac output, the mitral valve was replaced, but the patient died at the 5th post-operative day due to low cardiac output and shock lung syndrome. This case was the only mortality in our series (3.6%).

All patients received oral anticoagulation therapy for a period of six months after the operation.

Two patients had moderate degree of haemolysis, from the first day of the operation, but they recovered completely after one month, biflexible ring was used with no leaflet resection for these two patients, postoperative echocardiographic studies revealed good size valve area, no or very mild degree of MR. and the peak gradient was less.

Discussion

The advantages of repair of the prolapsed, regurgitant, mitral valve are:

(1) The annulus, continuity is maintained with a papillary muscle valve, which appears to improve postoperative left ventricular function.

(2) The patients are not exposed to the higher morbidity of a bioprosthetic or prosthetic valve, particularly to thromboembolic events. (9)

From experimental, (7,14,15) and clinical data, (12,16,17) annular papillary muscle continuity appears to be important for improvement in the left ventricular function and operative survival as well as for late survival, by improving cardiac output and decreasing the incidence of chronic congestive heart failure. There is still considerable controversy about the timing of surgery for patients with a prolapsed mitral valve. (10,22). Clearly, patients with severe MR should not wait until

there are severe symptoms and severely depressed left ventricular function.

The main morbidity factor with reparative procedures is the failure of the repair and the necessity for reoperation. In our study only one patient out of 28 patients had reoperation for failure of the repair and severe MR. This patient was the first patient in our series. In this case we only resected a wedge from the anterior leaflet without using any type of ring annuloplasty. Mitral valve replacement was done on the second postoperative day, but the patient died on the 5th postoperative day due to low cardiac output and shock lung syndrome.

The most important point in the surgical techniques of repair of the mitral valve is the proper evaluation of the residual regurgitation of the mitral valve, and the proper evaluation of the success of the repair. In our study we use two techniques for evaluation.

(1) We fill the left ventricular cavity with saline solution by a small catheter connected to a 50cc syringe, the catheter passed from the opened L.A via the mitral valve to the left ventricle and we inject the saline.

(2) We turn the head of the patient down, and we remove the aortic clamp, we press by two fingers on the root of the aorta, to make the aortic valve incompetent to fill the left ventricular cavity by blood, and to detect any MR after repair.

Also the evaluation of the mitral valve at operation before repair is important and critical to the evaluation of chordal length, insufficient valve tissue and commissural narrowing. The most difficult valve is that with myxomatous changes in both leaflets with multiple elongated chordae. This type of valve needs good experience for evaluation and repair.

In our study we had 2 patients with haemolysis, we used biflexible ring from ST. Jude Medical, and the rings are of bigger size, then we tighten them more and they became corrugated and traumatizing for the blood. The haemolysis recovered after one month due to endothilum covering these rings.

The treatment of the valve with leaflet resections and chordal shortening is straight forward. The treatment of the annulus, is of some controversy. Carpentier and co-workers (25) used a semirigid ring to stabilize the anulus. Duran and associates (26) have developed a totally flexible ring. More recently ST. Jude Medical developed another more biflexible ring.

David and co-workers (28) have presented data to suggest that a more flexible ring is better for patients with very wide-dimeter marginal anuli, permitting less left ventricular obstruction and consequently better left ventricular performance.

Rigid Carpentier ring may lead to left ventricular outflow obstruction by "telescoping" the valve apparatus resulting in left ventricular outflow obstruction. This was not observed with the Duran ring (29,30). Yacoub and co-workers (31) have stated that in their

large experience, no ring for mitral valve repair is ever indicated, and in fact, the original suture annuloplasty proposed by Reed (32), is still popular and works well.

Conclusion

Repair of the mitral valve is very important and very good operation especially in young patients (Children in the school age), with the wide spread of insurance on these groups of patients in the last years.

Repair of the mitral valve is superior than valve replacement, due to the absence of long-term use of anticoagulation, shorter hospital stay and less frequent chronic congestive heart failure.

The incidence of patients who will be seen with complications of mitral valve prolapse will be increasing, and with the availability of repair techniques, earlier surgical referrals will probably result, since a repaired haemodynamically excellent native valve with an intact subvalvular mechanism, enhances long term survival and reduces morbidity.

Intraoperative transoesophageal echocardiography is very important for good and proper evaluation of the repair, and for detecting any degree of residual mitral regurgitation.

Any surgical techniques for repair of the mitral valve must be ended by ring annuloplasty.

REFERENCES

1. Savage DD, Garrison RJ, Devereux RB, et al. Mitral valve prolapse in the general population: 1.Epidemiological features: the Framingham study. Am Heart J 1983; 106:571-6.

2. Wilcken DEL, Hickey AJ. Lifetime risk for patients with mitral prolapse of developing severe valve regurgitation requiring surgery. Circulation 1988, 78: 10-4.

3. Devereux RB, Hawkins I, Kramer-Fox R. Complications of mitral valve prolapse: disproportionate occurrence in men and older patients. Am J Med 1986; 81:751-8.

4. Hickey AJ, Wilcken DEL, Wright JS, Warren BA. Primary (spontaneous) chordal rupture; relation of myxomatous valve disease and mitral valve prolapse. J Am Coll Cardiol 1985; 5:1341-6.

5. Nishimura RA, McGoon MB, Shub C, Miller FA, IIstrup DM, Tajik AJ. Echocardiographically documented mitral-valve prolpase: long-term follow-up of 237 patients. N Engl J Med 1985; 313:1305-9.

6. Duran DR, Becker AE, Dunning AJ. Long-term follow-up of idiopathic mitral valve prolapse in 300 patients: a prospective study. J Am Coll Cardiol 1988; 11:42-7.

7. Lillehei CW, Levy MJ, Bonnabeau RC. Mitral valve replacement with preservation of papillary muscles and chordae tendinae. J THORAC CARDIOVASC SURG 1964; 47:532-43.

8. Lillehei CW, Gott VL, DeWall RA, Varco RL. Surgical correction of pure mitral insufficiency by annuloplasty under direct vision. Lancet 1957; 77:446-9.

9. Cohn LA, Allred EN, Cohn LA, et al. Early and late risk of mitral valve replacement. J THORAC CARDIOVASC SUTR 1985; 90:872-81.

10. Gaasch WH, Levine HJ, Zile MR. Chronic aortic and mitral regurgitation: mechanicl consequences of the lesion and the results of surgical correction. In: Levine HJ, Gaasch WH, eds. The ventricle: basic and clinical aspects. Boston: Martins Nijhoff Publishing, 1985: 237-58.

11. Kirklin JW. Replacement of the mitral valve for mitral incompetence. Surgery 1972; 72:827-36.

12. Boncheh LI, Olinger GN, Siegel R, Tresch DD, Keelan MH. Left ventricular performance after mitral reconstruction for mitral regurgitation. J THORAC CARDIOVASC SURG 1984; 88:122-7.

13. 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 posible clinical implications. Ann Thorac Surg 1986; 41:363-71.

14. Hansen DE, Cahill PD, DeCampli WM, et al. Valvular-ventricular interaction: importance of the mitral apparatus in canine left ventricular systolic performance. Circulation 1986; 73:1310-20.

15. Salter DR, Pellom GL, Murphy CE, et al. Papillary-annular continuity and left ventricular systolic function after mitral valve replacement. Circulation 1986; 74 (suppl 1): 1-121-9.

16. David TE, Uden De, Strauss HD. The importance of the mitral apparatus in left ventricular function after correction of mitral regurgitation. Circulation 1983; 68 (suppl II): 11-76-11-82.

17. 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 echocrdiographic study. J Am Coll Cariol 1987; 10:568-75.

18. Cohn LH, Kowalker W, Bhatia S, et al,. Comparative morbidity of mitral valve repair versus replacement for mitral regurgitation with and without coronary artery disease. Ann Thorac Surg 1988; 45-284-90.

19. Sand ME, Naftel DC, Blackstone EH, Kirklin JW, Karp RB. A comparison of repair and replacement for mitral valve incompetence. J THORAC CARDIOVASC SURG 1987; 94:208-19.

20. Penkoske PA, Ellis FH, Alexander S, Watkins E Jr. Results of valve reconstruction for mitral regurgitation secondary to mitral valve prolpase. Am J Cardiol 1985:55;735-8.

21. Cosgrove DM, Chavez AM, Lytle BM, et al. Results of mitral valve reconstruc-

tion. Circulation 1986; 74 (suppl 1): 1-82-7.

22. Grossman W. Aortic and mitral regurgitation. JAMA 1984; 252:2447-9.
23. Cohn LH. Intraoperative assessment of the reconstructed mitral valve [letter]. J THORAC CARDIOVASC SURG 1985; 90-311-2.
24. Antunes MJ, Colsen PR, Kinsley RH. Mitral valvuloplasty: a learning curve. Circulation 1983; 68 (suppl 11): 11-70-5.
25. Carpentier A, Chauvaud S, Fabiani JN, et al. Reconstructive surgery of mitral valve incompetence: ten-year appraisl. J THORAC CARDIOVSC SURG 1980; 79:338-48.
26. Duran CG, Poman JL, Revuelta JM, et al. Conservative operation for mitral insufficiency: critical analysis supported by postoperative hemodynamic studies of 72 patients. J THORAC CARDIOVASC SURG 1980; 79:326-37.
27. Murphy JP, Sweeney MS, Cooley DA. The Puig-Massana-Shiley annuloplasty ring for mitral valve repair: experience in 126 patients. Ann Thorac Surg 1987; 43:52-8.
28. David TE, Komeda M. Pollick C, Burns RJ. Mitral valve annuloplasty: the effect of the type on left ventricular function. Ann Thorac Surg (in press).
29. Kreindel MS, Schiavone WA, Lever HM, Cosgrove D. Systolic anterior motion of the mitral valve after carpentier ring valvuloplasty for mitral valve prolapse. Am J Cariol 1986; 57:408-12.
30. Galler M, Kronzon I, Slater, et al. Long-term follow-up after mitral valve reconstruction: incidence of postoperative left ventricular outflow obstruction. Circulation 1986; 74 (suppl I):1-99-1-103.
31. Yacoub M, Halim M, Radley-Smith R, McKay R, Nijveld A, Twoers M. Surgicl treatment of mitral regurgitation caused by floppy valves: repair versus replacement. Circulation 1981; 64 (suppl II): II-210-6.
32. Reed GE. Repair of mitral regurgitation: an 11 year experience. Am J Cardiol 1978; 31:494-6.

Pleural changes after coronary artery bypass grafts

Abstract

It is known that coronary artery bypass grafting (CABG) results in impairment of postoperative pulmonary function and there is also a higher incidence of pleural changes (pleural effusion or pleural thickening) after CABG. The present study reports the incidence of pleural changes after coronary artery bypass grafting in eighty patients, who underwent elective coronary artery grafting, between April 1993 and April 1994, at the Cardiothoracic Surgery Department, Faculty of Medicine, Ain Shams University Hospital. Chest radiographs obtained preoperatively and on the six postoperative day were reviewed. Forty five patients underwent saphenous vein grafts only (56.25%) and 35 patients underwent both saphenous vein and internal mammary grafts (43.75%). Six days postoperatively, 25 patients (31.25%) had a normal chest radiograph, 10 patients 912.5%) had pleural thickening and 45 patients (56.25%) had pleural effusion (either unilateral or bilateral). Twenty five patients (556.6%) had a small pleural effusion and 20 patients (44.4%) had large pleurl effusion. Twenty five patients had right pleural effusion (55.6%), 10 patients had left pleural effusion (22.2%) and 10 patients had bilateral pleural effusion (22.2%). Ten patients (12.5%) had pleural thickening. The incidence of pleural effusion in the group of patients who underwent saphenous vein and internal mammary grafts was higher than with saphenous grafts alone . We believe that the genesis of the pleural effusion and thickening is multifactorial and additional studies that assess both ventricular function and the presence of pericardial fluid using radioisotopes or echocardiography will allow better understanding of these changes postoperatively.

Sherif El-Bouhy, Farag I. Abdel Wahab.*, and Maged M. Refaat Chest and Medical Departments, Faculty of Medicine, Ain Shams University

* Cardiothoracic Surgery Department, Faculty of Medicine, Al-Azhar University

Introduction and aim of the work

Coronary artery bypass grafting (CABG) is commonly performed via a median sternotomy incision with a saphenous vein graft (SVG) and/or an internal mammary artery graft (IMA). The internal mammary artery has become the conduit of choice for CABG because of its superior patency rate (Barner et al., 1985). Hovever, Jenkins et al., 1989,

and Khollef, 1990, have suggested that the use of the internal mammary artery grafts is associated with more postoperative pleural complications and a larger decrease in the pulmonary functions.

Peng et al., 1992, hypothesized that there would be a higher incidence of pleural changes after CABG in patients who underwent IMA grafting because pleurotomy is usually performed but, in their study, they reported an identical incidence of pleural effusions in patients who received IMA grafts (41%) compared with those who received SV grafts (43%) and they found no relationship between the presence of atelectasis and the development of pleural effusions, nor was there an association between the presence of a chest tube and the development of a pleural effusion. They hypothesized that the increased postoperative complications following IMA bypass may be due to the performance of pleurotomy, the placement of a chest tube, pericardial inflammation and/or trauma to the chest wall during dissection.

Vargas et al., 1992, reported that patients who had pleural changes on their chest radiographs, after myocardial revascularization, did have somewhat greater decreases in their lung volumes and in their oxygenation. However, even patients with large effusions did not have a statistically greater decrease in their oxygenation compared with the patients with normal chest radiographs.

Singh et al., 1992, reported that patients who had atelectasis after coronary revascularization tended to have more pleural changes on their chest radiographs and the presence of atelectasis was associated with more significant hypoxia compared with the patients who had normal chest radiographs. Patients who had atelectasis and pleural changes on their chest radiographs had significant decreases in both lung volumes and oxygenation.

The aim of this work is to report the incidence of pleural complications after coronary artery bypass grafting, to review the literature and to find out the possible cause of these pleural complications at the Cardiothoracic Surgery Department, Faculty of medicine, Ain Shams University Hospitals.

Materials and methods

Eighty patients, who underwent elective coronary artery grafting, between April 1993 and April 1994, at the Cardiothoracic Surgery Department, Faculty of Medicine, Ain Shams University Hospitals, were retrospectively evaluated for the occurrence of pleural changes (effusion or thickening) postoperatively.

There were 74 males and 6 females ranging in age between 40 and 56 years. The patients were classified according to the type of graft used either saphenous vein graft alone or both saphenous vein and internal mammary artery grafts (whether right or left internal mammary artery) were used.

All patients had a posteroanterior (PA) and a lateral plain chest radiographs preoperatively (only patients with a normal chest radiographs were included) and on the six

postoperative day. All patients had a small subxiphoid mediastinal drainage tube but no one had an intercostal chest tube. The patient was said to have a pleural effusion after the surgery if a costophrenic angle was blunted on either the PA or the lateral chest radiograph. The effusion was classified as small when it occupied less than one intercostal space and large when it occupied two or more interspaces. The patient was said to have pleural thickening if there was a discrete vertical costal pleural line on the PA radiograph without concomitant blunting of the posterior costophrenic angle.

Results

Eighty patients, who underwent elective coronary artery grafting, between April 1993 and April 1994, at the Cardiothorcic Surgery Department, Faculty of Medicine, Ain Shams University Hospitals, were retrospectively evaluated for the occurrence of pleural changes (effusion or thickening) postoperatively. Chest radiographs obtained preoperatively and on the six postoperative day were reviewed. Forty five patients underwent saphenous vein grafts only (56.25%) and 35 patients underwent both saphenous vein and internal mammary grafts (43.75%).

In this study, the incidence of postoperative pleural changes was high. Six days postoperatively, 25 patients (31.25%) had a normal chest radiograph, 10 patients (12.5%) had pleural thickening and 45 patients (56.25%) had pleural effusion (either unilateral or bilateral). No patient had an intercostal chest tube drainage or pleural aspiration and all effusions responded to medical treatment alone.

Of the forty five patients who had pleural effusion, 25 patients (55.6%) had a small pleural effusion (less than one intercostal space) and 20 patients (44.4%) had large pleural effusion (two or more intercostal spaces). Twenty five patients had right pleural effusion (55.6%), 10 patients had left pleural effusion (22.2%) and 10 patients had bilateral pleural effusion (22.2%).

Ten patients (12.5%), of the 80 patients who underwent CABG, had pleural thickening (discrete vertical costal pleural line on the PA radiograph without concomitant blunting of the posterior costophrenic angle). Of these ten patients 7 patients (8.75%) had underwent both saphenous vein and internal mammary grafts and 3 patients (3.75%) had underwent saphenous vein grafts only.

The incidence of pleural effusion in the group of patients who underwent saphenous vein grafts only (45 patients) was 37.7% (17 patients). Ten patients had right pleural effusion (58.8%), 5 patients had left pleural effusion (29.4%) and 2 patients had bilateral pleural effusion (11.8%).

The incidence of pleural effusion in the group and of patients who underwent saphenous vein internal mammary grafts (32 patients) was 81% (26 patients). Twelve patients had right pleural effusion (46%), 6 patients had left pleural effusion (23%) and 8 patients had bilateral pleural effusion (31%).

The incidence of pleural effusion in the group of patients who underwent both saphenous vein and internal mammary grafts (81%) was higher than those who underwent saphenous vein grafts only (37.7%).

Discussion

The present study as well as several previous studies (Khollef et al., 1988 and Hurlbut et al., 1990), demonstrated that there is a higher incidence of pleural changes after internal mammary artery grafting.

The incidence of postoperative pleural changes, in the present study is 68.75% (55 patients). The incidence of pleural effusion (either unilateral or bilateral) is 56.25% and the incidence of pleural thickening is 12.5%. The effusions did not appear to be related to an enlarged cardic silhouette, atelectasis or the placement of a chest tube. This incidence is high compared to the results reported by Khollef et al., 1992 and Hurlbut et al., 1990.

The internal mammary conduit is preferred to the saphenous vein grafts for coronary revascularization due to its superior patency rates and improved survival (Dangling et al., 1989 and Tyras et al., 1980). More postoperative pleuropulmonary complications are expected after IMA grafts than after SVG for the following reasons. The harvesting of the internal mammary artery may reduce the blood supply to the intercostal muscles and cause additional trauma to the chest wall. The pleural cavity is entered mush more commonly with IMA grafts which will lead to more intrapleural haemorrhage, the placement of more chest tubes and a greater likehood of injury to the phrenic nerve.

The incidence of pleural effusion in the group of patients who underwent saphenous vein grafts only was 37.7% and the incidence of pleural effusion in the group of patients who underwent saphenous vein and internal mammary grafts was 81%. This coincides with previous studies suggested that the incidence of effusion is higher in patients who received IMA grafts. Hurlbut et al., 1990, reported an incidence of 64% in patients receiving IMA graft but only 47% in patients who received only SVC.

The explanation for the occurrence of pleural effusion after CABG is unknouwn, but there are several possibilities:

First, the pleural effusion could be due to heart failure. However, most pleural effusion due to heart failure are bilateral and approximately equal in size (Weiss and Spodick, 1984). Although most of the effusions in this study were unilateral and right sided, right sided in 55.6%, left sided in 22.2% and bilateral pleural effusion in 22.2%, we believe that heart failure is not the explanation for the occurrence of pleural effusion as all the patients did not have heart failure postoperatively.

Second, the pleural effusion could be due to the post cardiac injury syndrome. This is unlikely since this syndrome usually is not manifested until the second or the third week after the injury and usually predominates on the left side (Light, 1990), while in this study

right sided effusions were present in 55.6%, left sided in 22.2% and bilateral in 22.2% of the cases.

Third, the development of effusion could be related to the presence of atelectasis which is common after abdominal surgery (Light and George, 1976). However, in the present study as well as the study of Gale et al., 1979, there was no associtionrelationship between the presence of atelectasis and the development of effusion.

Fourth, the lymphatic drainage from the pleural space could be decreased postoperatively especially in patients who received an IMA grafts as during mobilization of the IMA pedicle the associated lymphatic are transected. This could be expected to decrease the lymphatic clearance from the pleural space and result in accumulation of pleural fluid (Hurlbut et al., 1990). In this study, the incidence of pleural effusion in the group of patients who underwent saphenous vein and internal mammary grfts was 81%. This agrees with Hurlbut et al., 1990, who postulated that the effusion is impaired lymphatic clearance.

Fifth, the performance of pleurotomy could have lead to the accumulation of pleural fluid (Olearchyk and Magovern, 1986). This is another opinion which agrees with the findings in this study as the incidence of effusion was higher in the group of patients who underwent both saphenous vein and internal mammary grafts.

Sixth, the presence of a chest tube could have led to pleural inflammation and the development of the pleural effusion. The results of the present study offer no support for this hypothesis as there was no need for intercostal chest tube drainage or pleural aspiration in any patient because all the effusions responded to medical treatment.

Seventh, the pleural effusion could be related to pericardial inflammation as there appears to be a high incidence of left pleural effusions with inflammatory pericardial diseases (Weiss and Spodick, 1983). One week after cardiac surgery, Ikaheimo and associates, 1988, demonstrated pericardial effusion by echocardiography in 63% of the patients and the incidence of pleural effusion was higher in patients who had pericardial effusion (56%) than in those without pericardial effusion (44%). Although, we did not perform echocardiography, no one of our patients had a clinical evidence of pericardial effusion and as most of the effusions in this were right sided, we believe that pericardial inflammation is a least a contributory factor in some patients.

It appears that the great majority of the unilateral right sided pleural effusion that occurs after coronary revascularization have no clinical significance. In the present study, non of the patients required throacocentesis or intercostal tube drainage and complete resolution of the effusions occurred in all patients by medical treatment. However, it should be emphasized that an occasional patient may require throacocentesis or intercostal tube drainage (Hurlbut et al., 1990).

The overall frequency of pleural thickning, in this series, ws 12.5% (10 patients). The frequency was higher in patients who underwent both saphenous vein and internal mammary grafts (6.75%) than in patients who underwent saphenous vein grafts only (375%0. Pleural thickening was presumably secondary to resolution of the pleural inflammation and/or organization of the residual postoperative hemothorax.

In summary, the present study indicates that the incidence of pleural effusion is 56.25% and the incidence of pleural thickening is 12.5% after coronary artery bypass grafting. Both the pleural effusion and the pleural thickening are much more common on the right side which is against several other studies that reported a left sided predominance of the pleural effusion and thickening. The pleural complications reported in this are much more common in patients who underwent both saphenous and internal mammary artery grafting than patients who underwent saphenous vein grafting only. We believe that the genesis of the pleural effusion and thickening is multifactorial and additional studies that assess both ventricular function and the presence of pericardial fluid via radioisotopes or echocardiography will allow better understanding of these changes postoperatively.

REFERENCES

1. Barner HB, Standeven JW, Rese J. Twelve-year experience with internal mammary artery for coronary bypass. J. Thorac Cardiovasc. Surg. 1985, 90:668-75.
2. Jekins SC, Soutar SA, Forsyth A, Keates FRW and Moxham J. Lung function after coronary artery surgery using the internal mammary artyery and the saphenous vein. Thorax 1989, 44:209-11.
3. Kollef MH: Chronic pleural effusion following coronary revascularization with the internal mammary artery. Chest 1990, 97:750-51.
4. Peng MJ, Vargas FS, Cukler A, Filho MT, Teixeira LR and Light RW. Postoperative pleural changes after coronary revascularization, comparison between saphenous vein and internal mammary artery grafting Chest 1992, 101 (2): 327-30.
5. Vargas FS, Cukier A, Terra-Filho M, Hueb W, teilxeira LR and Light RW. Relation between pleural changes after myocardial revascularization & pulmonary mechanics. Chest 1992, 102 (5): 1333-36.
6. Singh NP, Vargas FS, Cukier A, Terra-Filho M, Teixeira LR and Light RW. Arterial blood gases after coronary artery bypass surgery. Chest 1992, 102 (5): 1337-41.
7. Kollef MH, Peller T, Kondel A and Gragun WH, Delayed pleuropulmonary complications following coronary artery revascularizton with the internal mammary artery. Chest 1988, 94:68-71.
8. Hurlbut D. Myers ML, Lefcoe M and Goldbach M. Pleuropulmonary morbidity internal throacic artery versus saphenous vein graft. Ann. Throacic. Surg. 1990, 50:959-64.

9. Weiss JM and Spodick DH. Laterality of pleural effusion in chronic congestive heart failure. Am. J Cardiology 1984, 53:951.

10. Light RW Pleural effusion due to miscellaneous disease in light RW. Pleural disease, 2nd ed. Philadelphia: Lea & Febiger, 1990:223-36.

11. Light RW and George RB. Incidence and significance of pleural effusion after abdominal surgery. Chest, 1976, 69:621-26.

12. Gale GD, Teasadale SJ, Sanders DF, Bradwell PJ, Russel A and Solaric B. Pulmonary atelectasis and other respiratory complications after cardiopulmonary bypass and investigation of etiological factors. Cana J. Anesth. Soc, 1979, 26:15-21.

13. Olearchyk AS and Magovern GJ. Internal mammary artery grafting. J. Thorac Cardiovasc Surg, 1986. 92:1082-87.

14. Weiss JM and Spodick DH. Association of left pleural effusion with pericardial disease N Eng J Med 1983, 308:696-97.

15. Ikaheimo MJ, Huikuri HV, Airaksinen KEJ, Korhonen UR, Linnaluoto MK and Takka MR. Pericardial effusion after heart surgery: incidence, relatioin to type of surgery, antithrombotic therapy and early coronary bypass graft patency. Am Heart J. 1988, 116:97-102.

9. Tyras DH, Barner HB, Kaiser GC, Godd JE, Pennington DG and Willman VL. Bypass grafts to the left anterior descending coronary artery. J. Thorax. Cardiovasc. Surg. 1980, 80:327-33.

Serial assessment of ventricular performance after relief of left ventricular outflow tract obstruction

Abstract

Between January 1990 and December 1994, 254 patients with chronic left ventricular outflow tract obstruction (LVOTO) have been operated upon. This study included 200 patients with valvular aortic stenosis (GROUP 1), and 54 patients with subvalvular aortic stenosis (GROUP 2).

All patients were studied by echocardiography preoperatively, 24 hours and late postoperatively.

Measurements of cardiac output, mean systemic blood pressure, heart rate and left ventricular ejection fraction were similar before and after operation, however early significant changes in capillary wedge pressure (27 - 13 mmHg), LVEDV (214 - 166 ml) and right ventricular ejection fraction (50 - 68%) have been observed.

Echocardiographic examination three months postoperatively showed increased left ventricular ejection fraction (49-58%), diminished ESV (91 - 59 ml) and EDV (166 - 135 ml) as compared to preoperative data.

We attribute the hemodynamic improvement to the morphologic normalization after relief of LVOTO.

F. Ibrahim, M.D., W. Osman, M.D., M. Ezz-Eldin, M.D., Al H. Gamil, M.D., S. El Mahmoudy, M.D., and I. Sallam, FRCS

*Cardiothoracic Surgery Department-Al-Azhar University ** Heliopolis Cardiac Centre

INTRODUCTION

LVOTO involves two main obstructive lesions, the immediate and late effects of aortic valve replcement in patients with aortic stenosis, and removal of obstructive lesion in patients with subvalvular obstruction, and symptoms of C.H.F, are subjected to detailed study. Documentation of usual changes in cardiac function in this sitting might provide insight into physiologic mechanisms for adaptation to chronic pressure overload by left ventricle and provide insights useful in management of individual patients. Therefore the purpose of this study is to measure early and late changes in left and right ventricular function after aortic valve replacement and relieve the obstructive lesions in subaortic stenosis. Initial echo, was obtained the day before operation and 24 hours after operation; and average of 3.5 months after operation to

assess the early and late response to relief of chronic pressure overload of the left ventricle.

Methods

This project was applied in Al-Azhar University Hospitals and Heliopolis Cardiac Centre between January 1990 and December 1994. The patient population consisted of 254 consecutive patients undergoing aortic valve replacement for aortic stenosis (200 patients) and excision of subvalvular aortic stenosis, (54 patients). were 64 patients in NYHA, III and 16 patients IV. The mean age was 45±15 years. Mean body surface area 1.3±0.3 cm^2. Calcifice aortic stenosis was present in all patients with AS, and discreate membranous subaortic obstruction in patients with suaortic obstruction, with a mean aortic valve gradient of 66±16 mmHg and calculated aortic valve area of 0.7±0.2 cm^2. 80 patients out of the whole group had angina, 90 patients had history of syncope.

Table (l): Serial Echos data: 24-48 hours after operation (mean ± standard deviation)

Variable	Before operation	24 hours	48 hours
Heart rate (beats/min)	74±14	96±13	95±10
		P<0.01	
Mean blood pressure (mmHg)	85±11	75±8	81±12
		P<0.05	
Cardiac output (L/min)	7.3±3.0	7.5±2.2	7.3±2.0
Cardiac index (L/min/m2)	4.1±0.9	4.0±1.1	3.9±1.0
LV ejection fraction	0.52±0.16	0.49±0.16	0.51±0.16
LV end-diastolic volume (ml)	214±50	166±47	169±39
		P<0.01	
LV end-systolic volume (ml)	106±55	91±55	87±45
		P<0.01	
Pulmonary wedge pressure (mmHg)	27±10	13±4	12±4
		P<0.001	
RV ejection fraction	0.54±0.13	0.68±0.06	0.64±0.06
		P<0.001	
Pulmonary blood volume (ml/m2)	700±208	462±73	453±123
		P<0.01	
LV end-systolic pressure volume (mmHg/ml)	1.9±1.1	1.6±0.7	1.8±1.0

Mild aortic insufficiency apparent on echo, was present in 220 patients. Aortic valve replacement using mechanical prosthesis was done in all patients with aortic valve disease. A prosthesis of diameter between 19±23 mm was placed during hypothermic potassium cardioplegic arrest with systemic cooling between 25±28 C, No concomitant CABG was undertaken. Aortic cross clamp time was 45±16 minutes, and total bypass time 60±18 minutes.

Results

Early postoperative assessment after relief of L.V.O.T.O. showed that preload measured by pulmonary capillary wedge pressure, left ventricular end-diastolic volume decreased significantly. In addition to the decrease in left heart filling, the pulmonary blood volume decreased and right ventricular ejection fraction increased (Table 1 and Fig. 1 and 2). Left ventricular end-systolic volume also decreased significantly. Heart rate increased after operation, whereas mean blood pressure decreased (in pts with CHF) and mean cardiac index for the population did not change after operation.

Serial data acquired during the 24±48 hours after operation demonstrated that a reduced level of left ventricular filling was maintained throughout this phase of the study. Left ventricular end-diastolic volume ranged between 142±30 to 185±42 ml. capillary wedge pressure ranged between 9.5±3.2 to 16.0±4.0 mmHg and cardiac index ranged between 3.7±0.9 to 4.8±1.2 L/min/m2, while they were kept on vasodilator to maintain the mean blood pressure near 75 mmHg. left ventricular ejection fraction ranged from

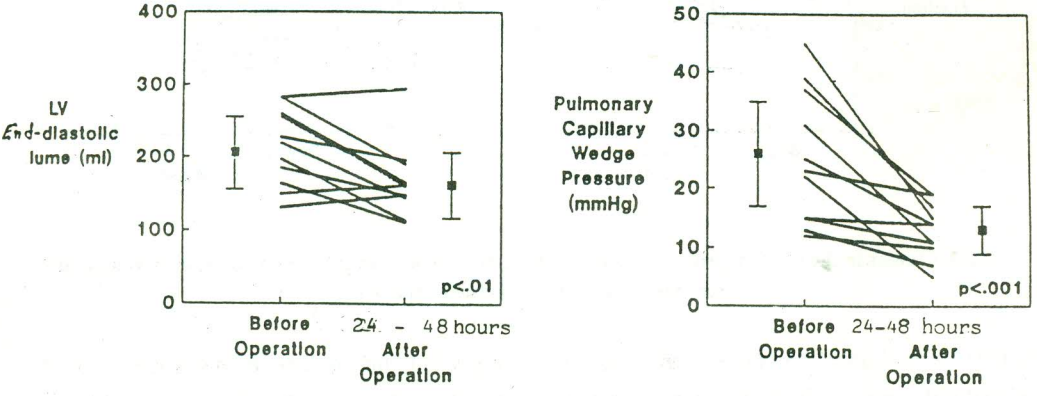
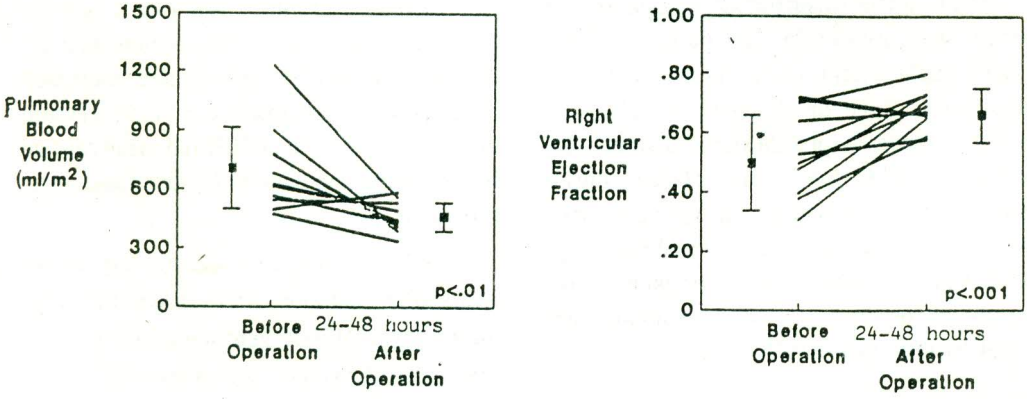


Fig. 1. Change in left ventricular filling is demonstrated with data from left ventricular end-diastolic volume (left) and pulmonary capillary wedge pressure (right).



Fi.g. 2. Change early after valve replacement also included a decrease in total pulmonary blood volume (left) and an increased right ventricular ejection fraction (right).

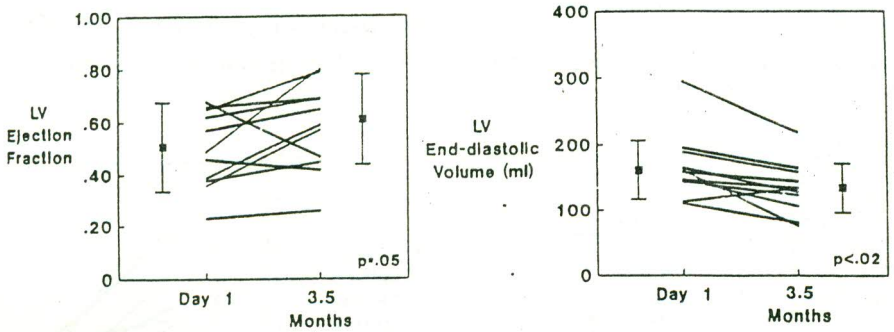


Fig. 3. Change in the left ventricular ejection fraction (left) and left ventricular end-diastolic volume (right) from the early to late period after operation.

0.46±0.14 to 0.58±0.15 (table 1 and fig. 3). 180 patients returned from the operating room while being treated with low-dose intravenous dopamine (<5 ug/kg/min), whereas 60 patients had higher level of support (8 to 10 ug/mg/min). All intravenous inotropic agents were weaned during the first day after operation. Although individual variation was

Table II: Pre and postoperative NYHA classification of all patients.

NYHA Cl.	Preoperative	Postoperative
I	44	90
II	130	100
III	64	56
IV	16	6
Total	254	252 + (Mortality 2)

present, neither student's paired t-test nor an anlysis of trend was able to detect any statistically significant change with respect to time for any of the echoes or hemodynamic variables during the first 48 hours after operation for the entire population.

Serial echoes and hemodynamic data are presented during the first day after valve replacement. Statistical analysis revealed no significant trend with respect to time for any variable, including the comparison of the 24 hours study with the 48 hours study. In addition, no difference was found for the comparison of patients with and without coronary artery disease L.V. Left ventricular, R.V, right ventricular.

Echocardiographic study at rest and after excercise was done at 3.5 months (range 1 to 8 months) after operation cardiac status in all patients was determined as NYHA functional class in table (II). Echo cardiographic results demonstrated a continued decrease in left ventricular filling as measured with end-diastolic volume; resting left ventricular ejection fraction increased, end-systolic pressure volume ratio increased, and left ventricular end-systolic volume decreased. No significant change was observed in the heart rate, cardiac output, right ventricular ejection fraction, or pulmonary blood volume, whereas, the mean blood pressure returned to the preopertive value (Table II, III and fig. 3).

Tachnical difficulties limited the number of adequate exercise test results to 10 patients. An adequate excrecise end point was reached in all patients, with substantial increase in the heart rate and blood pressure. Cardiac index increased from 3.5 to 5.8 L/min/m2 (Tab. III). No change was observed in left ventricular ejection frction, end-systolic volume, or end-diastolic volume (Tab. III and Fig. 4). Two patients of the whole group died

Table III: Echos-data: Early versus late follow-up (mean+standard deviation)

Variable	24–48 hours	*3.5 Month (rest)*	*Exercise*
Heart rate (beats/min)	96 ± 13	87 ± 14	136 ± 2
		$p < 0.001$	
Mean blood pressure (mm Hg)	75 ± 8	96 ± 13	14 ± 13
	$p < 0.02$	$p < 0.001$	
Cardiac output (L/min)	7.5 ± 2.2	6.7 ± 2.3	11.0 ± 5.1
		$p < 0.001$	
Cardiac index (L/min/m^2)	4.0 ± 1.1	3.5 ± 1.1	58. ± 2.5
		$p < 0.001$	
LV ejection fraction	0.49 ± 0.16	0.58 ± 0.16	0.57 ± 0.16
	$p = 0.05$		
LV end-diastolic volume (ml)	166 ± 47	135 ± 38	142 ± 41
	$p < 0.02$		
LV end-systolic volume (ml)	91 ± 55	59 ± 37	62 ± 37
	$p < 0.05$		
RV ejection fraction	0.68 ± 0.06	0.58 ± 0.08	
Pulmonary blood volume (ml/m^2)	463 ± 73	419 ± 130	
LV end-systolic pressure volume (mm Hg/ml)	1.6 ± 0.7	3.1 ± 1.8	
	$p < 0.01$		

Comparison of the changes in echos and hemodynamic parameters from the first day to 3.5 months after valve replacement, as well as the results of the late exercise test are shown. LV, Left ventricular; RV, right ventricular.

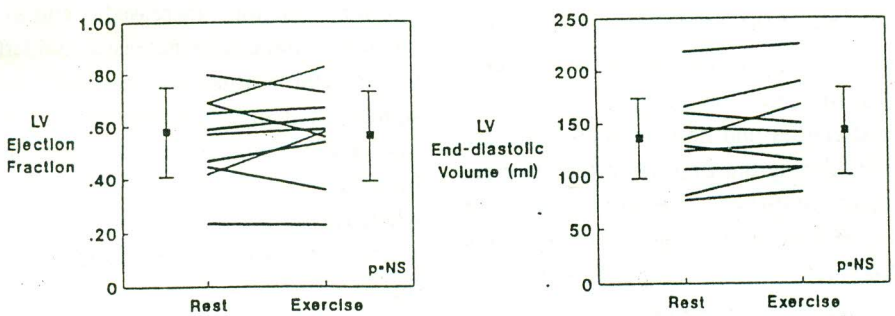


Fig. 4: Response to maximal exercisc 3.5 months after operation is shown for the left ventricular ejection fraction (left) and the left ventricular end-diastolic volume (right).

within one month postoperatively from cardiac causes.

Discussion

The early effects of valve replacement on left ventricular function in patients with aortic stenosis and subvalvular obstruction is probably due to a significantly reduced left ventricular outflow obstruction. Several months after operation other factors including adaptation and myocardial remodeling, may

play a role in a change in function (Kennedy et al., (1) 1991, and Usher, (2) 1991). The purpose of this study was to document by echo, some early and late changes in ventricular function that occur after relief of L.V.O.T.O.

During the first 24 hour after relief of L.V.O.T.O. significant decrease in left ventricular prelod was measured with pulmonary capillary wedge pressure, left ventricular end diastolic volume, and pulmonary blood volume. With a decreased left ventricular outflow obstruction, the left ventricle was able to shift the function curve and maintain a similar cardiac output at a reduced level of filling, (3) this is similar to results by Ross in 1985. The right ventricular ejection fraction also increased because of reduced right ventricular after load (pulmonary artery pressures) and probable increased mobility of the intraventricular septum, since the left ventricular chamber was smaller as that of Borer et al., 1983 (4) and Jehle et al., (5) 1983.

A change was noted in the arterial blood pressure and the heart rate compared with the value before operation. Heart rate was increased after operation, but not to an extent to account for significant decrease measured in left ventricular preload after relief of the obstruction. Although arterial blood pressure decreased slightly, the majority of the decrease in left ventricular afterload after operation was a result of the removal of the left ventricular outflow obstruction.

Ventricular performance, measured by left ventricular ejection fraction, varied after operation + 0.06 units from the mean value before operation, with changes in cardiac output afterload reduction. Therapeutic inotropic support was needed in 60 patients during the initial serial measurements but was rapidly discontinued. The left ventricular performance after valve replacement was similar to that obtained before operation, but it increased with change in filling and afterload. Reduction in late follow up in left Ventricular performance early after operation would be expected if there had been inadequate myocardial protection during cardiopulmonary bypass.

Echo data revealed excellent correlations between left ventricular ejection fraction (r=0.96), end-diastolic volume (r=0.59), and stroke volume (r=0.92).

In 1983 Niemela et al. (6) observed an increase in resting left ventricular ejection fraction (0.62 to 0.72) and a decrease in end-diastolic volume index (106 to 77 ml/m2) in 4 patients who underwent cardiac catherterization before and 1 year after relief of L.V.O.T.O. Schwarz and colleagues in 1978 and Kennedy et al., 1977 (7) (8), described a decrease in left ventricular wall mass and left ventricular end-systolic wall stress six months after operation in eight patients who had valve replacement for aortic stenosis.

Data obtained at the late follow-up studies in our patients showed increased left ven-

tricular ejection fraction, increased systolic pressure/volume ratio, and a continued decrease in left ventricular end-diastolic volume compared with the values obtained 2 days after operation. These late adaptations in resting cardiac function correlated with a regression in concentric hypertrophy and a decrease in myocardial wall mass.

The late postoperative improvement in left ventricular performance was further supported by an improved NYHA functional class in all the patients. Because of the preoperative congestive heart failure in some patients, poor exercise tolerance of these patients, and a concern of creating ventricular arrhythmias with maximal exercise stress, no preoperative exertional testing was undertaken.

However, all patients stated that their exercise tolerance was improved late after operation, and the results of the follow-up echo exmination revealed no changes of maximal exercise.

These data suggest that the postoperative decrease in left ventricular outflow obstruction allowed the ventricle to shift function by Starling mechanism and generate an adequate cardiac output at a decreased level of filling. Adequate myocardial protection during cardiopulmonary bypass maintained left ventricular performance during the early period after operation. The late follow-up data revealed an improved functional class and enhanced ventricular performance and exercise tolerance coincident with ventricular remodeling in this patient population with L.V.O.T.O.

REFERENCES

1. Kennedy, K.D., Nishimura, R.A., Holmes, D.R. et al.: Natural history of moderate aortic stenosis. J. Am. Coll. Cardiol. 17:313, 1991.
2. Usher, B.W.: Valve surgery: Indications and long-term results. Current Opin. Cardiol. 6:219, 1991.
3. Ross J. After load mismatch in aortic and mitral valve disease: implications for surgical therapy. JAM. Call Cardial, 1985; 5: 811-25.
4. Borer J.S. Jason M., Deveraux RB et al. Function of hypertrophied left ventricle at rest and during exercise. Am. J. Med. 1983; 75 34-9.
5. Jehle J. Lauber A, Spiller P. Uhlmann D.: Left ventricular function during exercise in patients with aortic valve disese. Circulation 1983; 68:99-103.
6. Niemela KO, Ikaheimo MJ, Linnaluoto ML, Takkunen JT. Response to progressive bicycle exercise before and following aortic valve replacement. Cardiology, 1983; 70:110-8.
7. Schwarz F, Flaeng W, Thormann J, et al. Recovery from myocrdial failure after aortic valve replacement. J. Thorac cardiovascular Surg. 1978; 75, 854-64.

8. Kennedy JW, Doces J, Stewart DK, Left vetricular function before and following aortic valve replacement. Circulation 1977, 56:944-50.

Malignant pleural mesothelioma: a rare but aggressive tumour, that is difficult to diagnose & manage

Mohamed M. Elsaied**, Salah A. Khalaf**, Mohamed A. El-Gamal**, Ahmed K. Abdallah**, Ayman A. El-Fiky* & Shaaban A. Abul-Ela

From Cardiothoracic Surgery** & Chest Medicine* Department; Mansoura University

Introduction

Malignant pleural mesothelioma is a very aggressive invariably fatal tumour that usually causes progressive pulmonary compromise (Patz et al., 1992). Its relation to asbestos had been described first by Wagner et al. in 1960. However, other possible causes & predisposing factors should be considered especially in childhood (Morgaan & Seaton, 3,4, 1975, Parkes 1983; & Anderson et al., 1985).

The incidence of malignant pleural mesothelioma is one / million/year in general population (Becklake 5, 1976). Two thirds of patients are between 50-70 years of age (Wechsler et al. 6, 1992). This reflects a long latent period of 30-45 years. Its incidence is two to one in males & females (McDonald & McDonald 7, 1983).

Four histological patterns have been described; tubulopapillary (epithelial type) ; sarcomatous type; undifferentiated cell type & mixed cell type (Dunill, 1982 Oparker 8, 1983).

The usual presenting complaints of patients with malignant pleural mesothelioma are chest pain, breathlessness which is progressive, spontaneous hemothorax or it is asymptomatic. Up to 95% of patients have pleural effusion during their clinical course (Gottehrer et al. 9, 1991).

Diagnosis is difficult & should be differentiated from pleural metastases, tuberculous pleurisy & rheumtoid disease (Morgan & Seaton, 1975). Various investigatory modalities should be exhausted ranging from conventional radiography to open pleural biopsy. CT scan, MRI, abdominal ultrasonography, pleural fluid cytology, blind needle pleural biopsy, CT guided biopsy & Thoracoscopic biopsy comes in between.

The accepted therapeutic modalities range from supportive care only (Deslauriers, et al. 10, 1991) to subtotal pleurectomy (Sugarbker et al. 11, 1991), or radical extrapleural pneumonectomy (Rusch et al. 12, 1991). Pleural effusion can be managed by thoracentesis & local sclerosing therapy (Aelony et al., 1991). However, all reported treatment modalities are unsatisfactory or disappointing in relieving symproms or prolonging survival (Deslauriers et al., 1991).

Despite the availability of new techniques such as megavoltage beam irradiation & computerized dosimetry techniques; radiation alone is unstisfactory & is of value only in relieving chest pain (Lederman et al., 1987). The role of chemotherapy is also unclear (Desluriers, et al., 1991).

Aim of the work

To realize the importance of early detection of malignant pleural mesothelioma nd to evaluate these tumours as regards presentation, methods of diagnosis and modalities of treatment since delayed diagnosis and management carry a very bad prognosis.

Patient & Methods

This is a review of eighteen cases of malignant pleural mesothelioma diagnosed at Mansoura in the last twenty years. All patients were subjected to careful history taking, complete clinicl and laboratory examinations; radiological examination in the form of plain radiography to all patients and CT scan to some of them. Histopathological examination of samples obtained by either pleural tapping, pleural needle biopsy, thoracoscopic biopsies or open pleural biopsy had been done. Management was either surgical; radiotherapy; chemotherapy or combined regimens. Follow up of the cases; when possible; was attempted.

Results

Discussion

In this retrospective study, a review of eighteen cases of malignant pleural mesothelioma had been diagnosed. The male to female ratio was 1.5 to 1 approximately (11 males and 7 females) which lies intermediate between the ratio reported by Mc Donald and McDonald, 1983 nd that recently reported by Danson et al., 1993 (2:1 and 1:1 respectively).

Although previous reports had found that two-thirds of patients were between 50-70 years (Wechsler et al., 1992 and Lanpher and Buncher, 1992); most of our cases (61.1%) have an age incidence between 40-50 years and only 16.7% (3 cases) were above 50 years of age.

The relation between malignant pleural mesothelioma and asbestos exposure was proved (Parkes, 1983) and the reported high age of presentation reflects a long latent period of 30-45 years after exposure (Wechsler et al., 1992).

Table I: age & sex incidence of 18 cases with malignant pleural mesothelioma

Age (years)	Males	Females	Total
< 40 Y	2	2	4 (22.2%)
40 - 50 Y	7	4	11(61.1%).
> 50 Y	2	1	3 (16.7%)
Mean age	47.4Y	38Y	43.1Y
Total	11(61.1%)	7(38.9%)	18(100%).

Table II: Side to be affected with malignant pleural mesothelioma

Side affected	Right	Left	Bilateral	Total
No. & % of cases	10 (55.6%)	6 (33.3%)	2 (11.1%)	18 (100%)

Table III: Clinical manifestations in 18 cases with malignant pleural mesothelioma

Clinical feature	No. of cases	Incidence
Symptoms		
Dyspnea	16	88.9%
Chest pain	12	66.7%
Cough	10	55.6%
Haemoptysis	2	11.1%
Signs :		
Pleural effusion	14	77.8%
Haemothorax	1	5.6%
Axillary or supra-clavicular lymph node enlargement	2	11.1%

N.B. : more than one symptom or sign could be encountered in one patient.

Table IV: Methods of samples obtained for histopathological examination in patients with malignant pleural mesotheliomas

Method	- ve for malignancy	suspicious for malignancy	+ve for malignancy	Total.
Aspiration cytology	7 (46.7%)	5 (33.3%)	3 (20%)	15 (100%)
Blind needle pleural biopsy	4 (28.6%)	2 (14.3%)	8 (57.1%)	14 (100%)
Thoracoscopic biopsy	-	-	4 (100%)	4(100%)
Open pleural biopsy	-	-	2 (100%)	2(100%)
Lymph node biopsy	-	-	1(100%)	1(100%)

N.B. : more than one method may be used in one patient.

Pathological type	epithelial	Sarcomatous	mixed	total
No. of cases	14(77.8%)	3 (16.7%)	1(5.5%)	18(100%).

Table V: Pathological type of diagnosed malignant pleural mesothelioma

Table VI: Therapeutic modalities & survival among 18 patients with malignant pleural mesothelioma.

Management pathology	pleurodesis	pleurectomy (subtotal)	chemo-therapy	palliative radiotherapy	survival (months)
1- epithelial	-	+	+	-	12
2- epithelial	failed	-	+	+	11
3- epithelial	success	-	+	-	14
4- epithelial	success	-	+	-	
5- epithelial	-	-	+	-	
6- epithelial	-	+	+	-	15
7- epithelial	-	-	+	-	
8- epithelial	failed	-	+	-	
9- epithelial	-	-	+	+	30
10- epithelial	success	+	+	-	
11- epithelial	success	-	+	-	
12- epithelial	success	-	+	-	
13- epithelial	success	-	+	-	15
14- epithelial	success	-	+	-	
15- sarcomatous	-	+	+	-	12
16- sarcomatous	failed	-	+	-	9
17- sarcomatous	success	-	+	-	8
18- Mixed	success	-	+	+	10

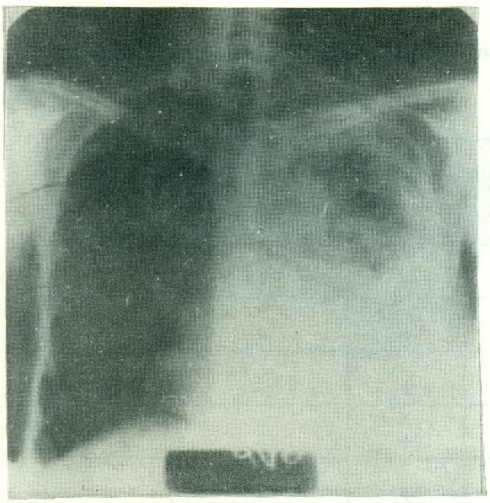


Fig. 1: X-ray of a case with malignant pleural mesothelioma showing Lt. pleural effusion with nodular pleural thickening.

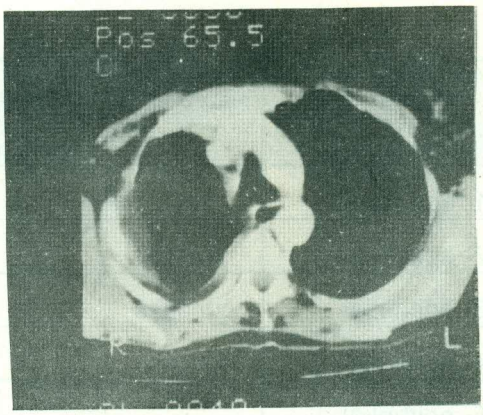


Fig. 3: CT-scan of a case with malignant pleural mesothelioma showing LT. pleural thickening and lung encasement.

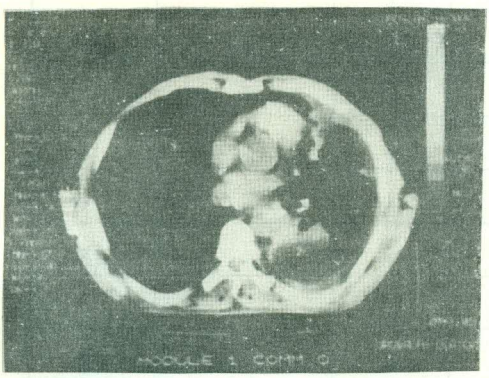


Fig. 2: CT-scan of a case with malignant pleural mesothelioma showing LT. pleural thickening with complete lung encsement and associated Lt. effusion.

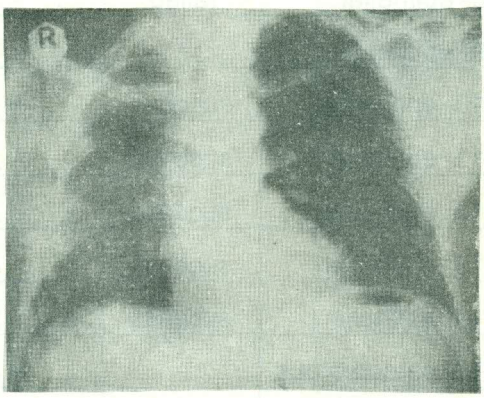


Fig. 4: X-ray of a case with malignant pleural thickening without pleural effusion.

However our area is not involved in asbestos exposure and one of the eighteen patients diagnosed in this study had positive history of exposure; a factor that may be responsible for lowering their age of presentation.

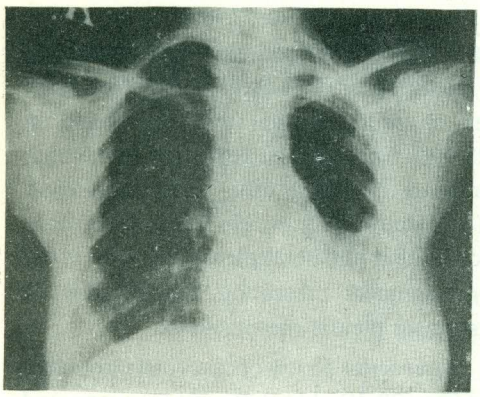


Fig. 5: X-ray chest showing LT. pleural effusion with pleural thickening in a case of malignant pleural mesothelioma.

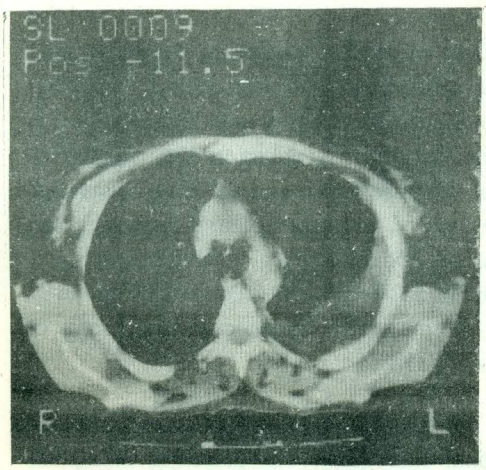


Fig. 6: CT-scan showing LT. pleural effusion with pleural thickening in a case of malignant pleural mesothelioma.

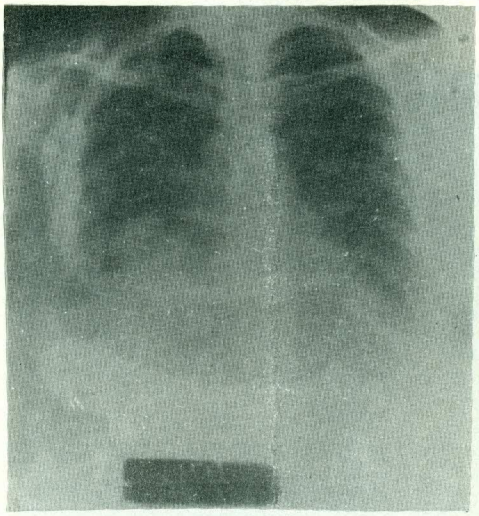


Fig. 7: Post aspiration x-ray film showing RT. basal nodular pleural thickening.

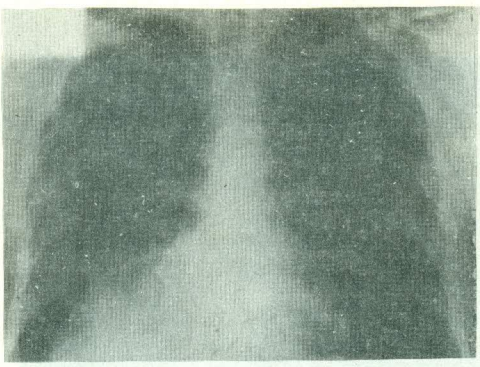


Fig. 8: Localized mesothelioma x-ray showing Rt. basal paracardiac shadow.

The usual presenting complaint of patients with malignant pleural mesothelioma is chest pain often localized and related to nerve and bone involvement of diffuse generalized type.

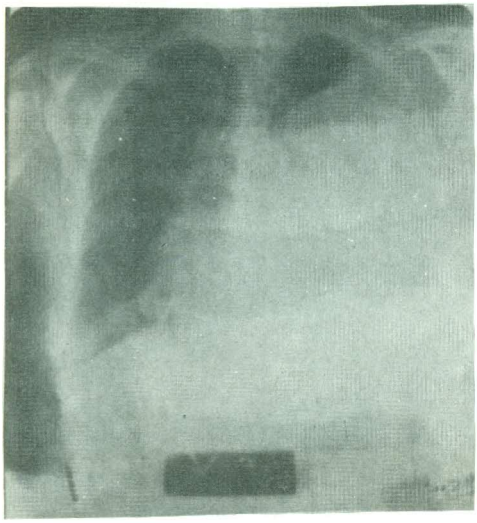


Fig. 9: Localized mesothelioma. Intial x-ray showing Lt. near total opacity with contra-lateral mediastinal shift.

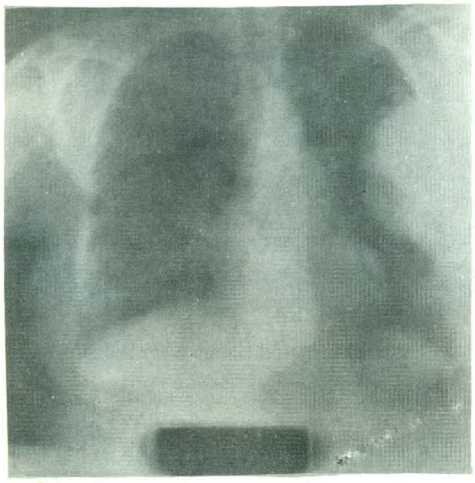


Fig. 9: Localized mesothelioma. x-ray showing Lt. recurrence of the tumour.

It may be referred to abdomen, shoulder and ipsilateral arm (Parkes, 1983). In this study, pain was the second common presenting symptom (66.7%) and was preceded by dyspnea on mild exertion that was found in (88.9%). This may be attributed to the high incidence of pleural effusion & hemothorax in our study (88.9%). Mc Loud, 1992, considered that unilateral pleural effusion is the most frequent presenting manifestation of malignant pleural mesothelioma.

In our series 55.6% of cases had right sided lesions; 33.3% has left sided lesions and 11.1% had bilateral one. The results correspond with that recorded by Devita et al. 15, 1993, who found that 60% of cases had right sided lesions.

Conventional chest radiography; though not diagnostic; is a must. It was done to all patients included in this study, its value was in detecting pleural effusion its site and the extent of pleural thickening before or after thoracocentesis. The appearance varies according to the stage at which the disease is first seen; its distributuion and whether or not it is associated with effusion. Signs suggestive of malignant pleural mesothelioma include nodular thickening of the pleura, such finding is obscured by free pleural fluid until it is removed by thoracocentesis (Adams et al. 16, 1986).

CT scan is more sensitive than conventional radiography in detecting malignant pleural mesothelioma (Godwin 17, 1984) and it was done to 14 cases in this series. All of them showed variable degrees of pleural thic-

kening (100%) and 12 of them (85.7%) had pleural effusion (85.7%). The frequency of subdiaphragmatic extension observed with malignant pleural mesothelioma (Godwin, 1994). raises the importance that CT evaluation of suspected malignant pleural mesothelioma should include scanning of the upper abdomen.

Cytological examination of aspirated pleural fluid was done to all cases with pleural effusion (14 cases) and to the case with spontaneous hemothorx. It was positive in 3 cases (20%); suggestive in 5 cases (33.3%) and negative for malignancy in 7 cases (46.7%). Dunnill, 1980, had reported that pleural fluid when obtained from a case with malignant pleural mesothelioma has a high cell count but the distinction between neoplastic and benign cells may give rise to difficulty. Adam's et al., 1986 had positive results in 32% only.

In this study Adam's pleural needle biopsy was done in 14 cases and the yield result was 57.1% which is higher than cytological examination. Higher yield results were obtained by Chang et al. 19, 1991 and Bechamp et al., 1992 (70% & 71% respectively). These results assure the value of closed pleural needle biopsy before proceeding to thoracoscopy and open pleural biopsy. Moreover the yield result of such technique could be increased by using CT guided approach. Thoracoscopy was utilized in 4 cases after failure of Abram's pleural needle biopsy in our series and the yield result was 100% without postoperative complications. However it must be stressed that multiple biopsies must be taken during the procedure since there is no specific gross lesion that presumably corresponds to early stages of malignant pleural mesothelioma during thoracotomy macroscopic dignosis is not possible.

Magan and seaton, 1975, had found that clinical evidence of distant metastases to other organs is rarely present in cases of malignant pleural mesothelioma and they suggest that the pleural lesion may be itself a metstases. Only one case (5.6%) in this series showed bilateral involvement together with bilateral axillary and supraclavicular lymph node enlargement, no other gross metastases was recorded in any other case. The lymph node biopsy was the diagnostic approach in that case and the yield result was 100%.

Open pleural biopsy was done in only two cases (11.1%) with pleural thickening without effusion in this series, and it allowed direct visualization and palpation of the pleura and the yield result was 100%. However the open pleural biopsy technique was done only in two cases which is a very limited number to estimate the yield result of such approach, especially a lower positivety rate; only 77% had been reported by Adam's et al., 1986. Beauchamp et al., 1992 had 100% yield result of such technique. Although closed pleural needle biopsy, thorarcoscopy; and open pleural biopsy had 100% yield results in diagnosis of malignant pleural mesothelioma in our series; seedling of mesothelioma in chest wall and intractable pain happened in one case.

A major problem in management of malignant pleural effusion is the control of recurrent malignant plaural effusion (Hoad et al. 20, 1986). In this series, twelve cases with massive recurrent pleural effusion underwent pleurodesis through an intercostal tube insertion and twice injection of teracycline and xylocaine. It controlled recurrent effusion in nine cases (75%), failed in three cases (25%) who needed repeated pleural tapping which is a promising result.

Deslouriers et al., 1991; had found that pelurectomy is very effective to control malignant effusions, but it should be restricted to patients in whom less invasive techniques have failed. One advantage of pleurectomy over other procedures is the possibility to surgically reexpand an entraped lung. Pleurectomy was done in four cases in our series and followed by chemotherapy and palliative radiotherapy. In one of these cases; the operation was repeated within 6 months due to local recurrence. Pleurectomy was very effective to control malignant effusion, however its use should be restricted to patients with low operative mortality and it has the advantage over extrapleural pneumonectomy of preserving the lung and diaphragm. Residudal disease over the diphragm is treated with radiotherapy and / or chemotherapy. Extrapleural pneumonectomy was not done in any of our cases. In our study, we found that patients who underwent pelurectomy followed by chemotherapy and radiotherpy have longer survival than chemotherapy and radiotherpy alone (median survival 30 months and 11.75 months respectively) but no difference in survival with epithelial (Median 12 months) and sarcomatous tumours (11 months), although most authors are generally agreed that epithelial type of malignant pleural mesothelioma has a better prognosis than do sarcomatous cell type of tumours (Butlin et al., 1993 and Mansinii et al., 1993) and mixed cell type tumours (Law et al. 21, 1982). However further evaluation with big series is needed.

Summary & Conclusions

In this study a retrospective review of eighteen cases of malignant pleural mesothelioma diagnosed and managed in 11 males & 7 females (1.5 to 1 appox.) at Mansoura in the last twenty years has been carried out 61.1% of patients had an age incidence between 40-50 years. Dyspnea was the presenting symptom (88.9%) followed by chest pain (66.7%). On clinical examination 88.9% had pleural effusion or spontaneous hemothrax; one of them had bilateral effusion associated with enlarged bilateral axillary and cervical lymph nodes.

Malignant pleural mesothelioma was suggested on radiological examination but with no pathognomonic features. CT helped more in diagnosis. Cytological examination of the aspirated pleural fluid was diagnostic in 20% and pleural needle biopsy was diagnostic in 57.1% while thoracoscopic biopsy and open pleural biopsy were diagnostic in 100% of cases. One case was diagnosed by lymph node biopsy.

Pleurodesis was successful to control recurrent malignant effusion in 75% of cases. Pleurectomy was done in four cases and repeated in one case within 6 months followed by chemotherapy and radiotherapy. It was very effective to control recurrent pleural effusion.

The median survival in these cases was higher than those treated by chemotherapy with or without radiotherapy alone.

Malignant pleural mesothelioma should be considered in any case with chest pain and recurrent pleural effusion; even without history of asbestos exposure and all investigations must be exhausted until an accurate diagnosis is made and never relie on aspiration cytology, and / or pleural needle biopsy alone. Surgical interference in the form of pleurectomy in combination with chemotherapy and palliative radiotherapy seems to have promising results and longer survival rates. However more effort and further evaluation is still needed for defining a specific approach for this rare and aggressive type of malignancy.

REFERENCES

1. Patz E.F.; Shffer K. and Worms D.R. (1992): Malignant pleural mesothelioma Value of CT and M.R imaging in predicting resectability. AJR; 159: 961-966.
2. Morgan W.M.K. and Seaton A. (1975): Occupational lung diseases, W.B. Saunders Company, Philadelphia and London; 357.
3. Parkes W.R. (1983): Occupational lung diseases. 2nd ed.; Butterworths, London, Boston and Durban, 233.
4. Anderson K.A., Curley W.C and Hurley B.T. (1985): Malignant pleural mesothelioma following radiotherapy in a 16- year old boy. Cancer; 56: 273-276.
5. Beklake M.R. (1976): Asbestos-related diseases of the lung and other organs, American Review of Respiratory Diseases, 114:55.
6. Wechsler R.J.; Steiner R.M. and Conant E.F. (1992): Occupationally induced neoplasms of the lung and pleura Radiologic clinics of North America; 30: 1245-1268.
7. McDonld and McDonald J.C. (1983): Malignant mesothelioma in North America. Cancer; 1650-1656.
8. Dunnill M.S. (1982): Pulmonary pathology, Vhurcill livingstone, Edinburgh, london and New York, 422.
9. Gottehrer A.; Taryle D.A. and Reed C.E. (1991): pleural fluid analysis in malignant mesothelioma: Prognooctic implications; Chest; 100: 1003-1006.
10. Deslauriers J.; Beauchamp G. and Desmeules M. (1991): Benign and Malignant Disorders of the pleura in : Baue A.E., Glenn's thoracic and cardiovascular surgery, Appleton and lange, California 4th ed., Vol. II, 459.
11. Sugarbaker D.J., Heher E.C. and Lee T.H. (1991): Extrapleural pneumonectomy,

chemotherapy and radiotherapy in the treatment of diffuse malignant mesothelioma. Journal of thoracic and cardiovascular surgery; 102:10-15.

12. Rusch W.W.; Piantadosi; S.; and Halmes E.C. (1991): The role of extrapleural pneumonectomy in malignant pleural mesothelioma. J. Thoracic and Cardiovascular Surgery; 102:1-9.

13. Lanphear B.P. and Buncher C.R. (1992): Latent period for malignant mesothelioma of occupational origin. JOM; 34: 718-721.

14. McLoud T.C. (1992): Conventional radiography in the diagnosis of asbestos related disese. Radiologic clinics of North America; 30: 1177-1189.

15. Devita V.T.; Hellman S. and Rosenberg S.A. (1993): Cancer, Principles and practice of oncology. 4th ed., J.B. Lippincott company, philadelphia, 1491-1507.

16. Aadms V.I.; Unni K.K. and Muhm J.R. (1986): Diffuse malignant mesothelioma of pleura: Diagnosis and Survival in 92 cases. Cancer; 58:1540-1551.

17. Godwin J.D. (1984): Tomogrphy of the chest, J.B. Lippincott company, Philadelphia; 147-148.

18. Chang D.B.; Yang P.C. and Luh K.T. (1991): Ultrasound-guided pleural biopsy with true-cut needle. Chest; 100: 1328-1333.

19. Beuchamp. H.D.; Kundra N.K. and Aranson B. (1992): The role of closed pleural needle biopsy in the diagnosis of malignant mesothelioma of the pleural needle biospy in the diagnosis of malignant mesothelioma of the pleura. Chest; 102 : 1110-1112.

20. Hood R.M. (1986): Surgical diseases of the pleura and chest wall. W.B. Saunders company. London., 229.

21. Law M.R.; Hodson M.E. and Heard B. (1982): Malignant mesothelioma of the pelura. Relation between histological type and clinical behaviour. Thorax; 37.810-815.

22. Mnzini, V.D., Brollo A. and Franceschi S. (1993): Prognostic factors of malignant mesothelioma of the pleura. Cancer; 72: 410-417.

23. Aelony, Y.; King R. and Boutin C. (1991): Thoracoscopic talc powdarage pleurodesis for chronic recurrent pleural effusions. Annals of Internal Medicine; 115:778-782.

24. Boutin C.; Sudan N. and Varette Y. (1975): Thorcoscopy in pleural malignant mesothelioma: A prospective study of 188 consecuative patients. Parts: Prognosis and. Staging. cancer; 72: 394-404.

Emergency coronary artery bypass grafting after failed angioplasty

Abstract

Twenty nine patients out of 4885 percutaneous transluminal coronary angioplasties (PTCA) required immedite emergency coronary artery bypass grafting after angioplasty failure. Among these patients, 15 had single vessel disease and 14 had multiple vessel disease.

The age of the patients ranged from 45-70 years (mean 57) with 25 males and 4 females. The cause of the emergency was dissection in 16 patients, acute vessel occlusion in 17 patients and perforation of a coronary artery in 3 patients.

The technique of controlled coronary reperfusion with warm then with cold blood cardioplegia was used intraoperatively. The surgical interventions were single aortocoronary bypass (ACB) in 14 patients, 2 x ACB in 9 patients, 3 x ACB in 4 patients, and 4 x ACB in 2 patients, with a mean number of 1,79±0.95 grafts per patient. In 3 patients the internal mammary artery was used to bypass the left anterior descending cornary artery.

Intraaortic balloon counterpulsation was necessary in 9 patients postoperatively. The maximal postoperative creatinine kinase (CK) value was 73-2160 (mean 669), and the maximal CK-MB value was 11-138 (average 49). One patient died postoperatively (3.4%), the postoperative morbidity comprised 1 sternal dehiscense and 3 patients had respiratory failure.

A Boseila* and K Emmerich**

Department of Thoracic and Cardiovascular Surgery, Cairo University*; and Department of Cardiology, Herdecken Witten University, Wuppertal; FRG**

Introduction and aim of the work

Since Gruentzing's (1) clinical application of percutaneous transluminal coronary angioplasty (PTCA) in 1977, widespread use of this technique has taken place, with PTCA being performed in community hospitals as well as major medical centres. Despite advances in instrumentation and proficiency in carrying out these procedures, a considerable number of patients require emergency coronary artery bypass grafting after PTCA failure.

This study will try to eluminate the increased hazards in form of morbidity and mortality associated with that emergency procedure.

Patients and methods

A retrospective review was performed on 4885 consecutive patients who underwent

PTCA at the Herdecken Witten University Clinics, Klinikum Barmen; Wuppertal, FRG over a 4 year period. Twenty nine patients required emergency coronary artery bypass grafting after PTCA failure. All patients were in a stable condition with no electrocardiographic or enzymatic evidence of acute myocardial infarction before attempted PTCA.

Failure of PTCA and the decision to proceed with immediate coronary artery bypass grafting rested on the fact that these patients had acute myocardial ischemia, evolving myocardial infarction, or severe rest angina, and who were in impending danger of myocardial loss according to the judgment of the cardiologist, in consulation with the cardiac surgeon. The patients were taken immediately to the operating room. One patient required placement of an intraaortic balloon pump to get him in a hemodynamically stable condition to the operating room. One patient had a previous coronary bypass operation.

The operative strategy was to unload the heart completely using normothermic total cardiopulmonary bypass. After aortic cross-clamping, controlled myocardial reperfusion was started using glutamate and aspartate enriched blood cardioplegia, partly through the ascending aorta and partly retrograde through the coronary sinus. This perfusate was instilled with an initial temperature of 37 degrees Centigrade and then with a temperature of 4 degrees Centigrade, with a flow rate of 200-300 ml/min, then every 20 minutes for 2 minutes, for a mean total duration of 14.29 ± 4.26 (7 min - 25 min). The operative procedure with complete myocardil revascularization was performed, after which the normothermic blood cardioplegia was infused into the aortic root, after which the aortic clamp was removed. The proximal anastomoses were performed with the aid of a partial occlusion clamp, which was removed after their completion and the vein grafts were perfused with flow from the cardiopulmonary bypass circuit.

Electrocardiograms and echocardiography were analyzed for the presence of old myocardial infarction or clinically significant myocardial dysfunction before PTCA .

All charts were reviewed to determine the incidence of postoperative complications and mortality. The dignosis of postoperative myocardial infarction was made by the presence of new, persistent Q waves with loss of R-wave force, and / or elevation of the level of total creatine kinase with greater than 5% MB fraction.

Differences between means of variables were tested for statistical significance using Chi-square and Fisher's exact test. All values were presented as mean ± SD, and the standard error of mean and the median were calculated, and p values were considered significant when less than or equal to 0.05.

Results

The patient collective was composed of 29 patients: 25 males and 4 females, with a

Table 1: Indications for termination of PTCA and proceeding with coronary artery bypass grfting

Indication	Number of patients	Percent (%)
Acute occlusion	17	58.6
Intimal dissection	16	55.2
Perforated coronary artery	3	10.3

Table 2: Opertive data

Varible	
Mean number of bypassed vesels + SD	1.79 + 0.95
CABG X 1	14 (48.3%) ptients
CABG X 2	9 (31.0%) patients
CABG X 3	4 (13.8%) patients
CABG X 4	2 (6.9%) patients
LIMA	3 (10.3%) patients
Mean ischemic time + SD	39.1 + 20 (17 - 89) min
Mean bypass time + SD	78.6 + 28.9 (40-131) min
Retrograde cardioplegia	9 (31%) patients
Mean cardioplegia time + SD	14.29 + 4.26 (7-25) min

mean age of 57±8.2 (age range from 41-70 years).

Diabetes was present in 15.4%, and hypertension in 15.4% of patients as a preoperative finding.

Echocardiography showed that 4 patients (13.8%) had myocardial dysfunction before PTCA, the rest of the patients had normal myocardial function.

Of the 29 patients 15 patients had single-vessel disease and 14 had multiple-vessel disease.

PTCA was performed in 17 patients for the left anterior descending lesions, in 7 patients for right coronary artery lesions, and in 5 patients for the circumflex lesions.

The indications that prompted termination of PTCA and proceeding with subsequent coronary artery bypass grafting are listed in table 1. To achieve hemodynamic and electric stability and to prevent the occurrence of myocardial infarction, a stack was inserted in 6 patients.

Table 2 shows the operative procedures performed for these patients, and the characteristics of these interventions.

Morbidity, as reflected by postoperative myocardial infarction and postoperative complictions were relatively high (tables 3 & 4). It is noticeable that the intubation time was 12-324 hours (mean 58 h), whilst the average

Table 3: Postoperative morbidity

Varible	
Myocardial infarct	9 pts (31.0%)
Q-infarct	7 pts (24.2%)
Non Q-infarct	2 pts (6.8%)
Peak CK (IU/L) mean+SD, median; range	669+570, 452; (73-2160)
Peak CK-MB (IU/L) mean+SD, median; range	49+41, 38; (11-138)
Multiorgan failure	1 pt (3.4%)
Hemofiltration	1 pt (3.4%)
Sternal Dehiscence	1 pt (3.4%)
Low cardiac output	6 pts (20.7%)
Intraaortic balloon counterpulsation	9 pts (31%)
Bleeding	4 pts (13.7%)
Respiratory failure	3 pts (10.3%)
Intubation time (h) mean+SD, median; range	58+83, 33; (12-324)
ICU time (h) mean+SD, median; rnge	92+113, 61.5; (15-480)
Hospital stay (day) mean+SD, median; range	14.7+8.9, 13; (7-41)

intubation time for elective operations is 12 hours. The time in the intensive care unit varied from 15-480 hours (average 92 h), whilst in elective operations the average duration of stay in the intensive care is 24 hours.

By splitting the patients according to the occurrence of postoperative myocardial infarction (table 5), there were no statistically significant differences between the pre-, intra-, and postoperative findings of the different groups, except for the relation of acute vessel obstruction during PTCA with the occurrence of acute myocardial infarction, which was statistically significant ($p = 0.03$), and the number of bypasses performed ($p = 0.04$) . By splitting the patients a second time according to the presence of single-versus multiple-vessel disease, there were no statistically significant differences between the pre-, intra-, and postoperative findings of the different groups.

Thus, by comparing patient variables, no factors were identified that could accurately predict which patients would likely suffer a myocardial infarction after emergency coronary artery bypass surgery for failed angioplasty.

As regards the use of the left internal mammary artery (LIMA), it was used to revascularize the left anterior descending artery in three male patients with an average age of 52.3 years. In two of the three patients there was intimal dissection, and in the third there was acute vessel obstruction which necessitated emergency surgical revascubrization. The patients were brought to the operating theatre in a stable hemodynamic and electric condition. Retrograde cardioplegia

Table 4: Blood loss and use of blood products

Varible	
Chest drainage (ml) median, mode; range	900, 500; (450-6245)
Rethoracotomy for bleeding	1 patient
Need for coagulation factors	1 patient
Packed RBC's units (mean + SD); range	5.8+5.8; (0-19)
FFP units (mean + SD); range	2.21+2.42; (0-9)
Platelet units (mean + SD); tange	1 + 2.5; (0-7)

Table 5: Univariate analysis of risk fctors associated with acute myocardial infarction after failed PTCA and emergency bypass

Variable	No Infarction (20 patients)	Infarction (9 patients)	p-Value
Age, mean+SD (years)	56.4+28.6	57.7+10.2	0.32
Sex (M/F)	16/4	9/0	0.28
Diabetes	18.2%	0%	0.7
Hypertension	9.1%	50%	0.29
Diseased vessels number, mean+SD	1.55+0.69	1.87+0.99	0.17
PTCA vessels (LAD/RCX/RCA)	11/2/7	6/3/0	0.07
Acute vessel obstruction	66.7%	100%	0.03
Perforated coronary	20%	0%	0.52
Stack	4 (66.7%)	2 (33.3%)	0.8
CABG number, mean+SD	1.65+0.75	2.11+1.27	0.04
Ischemia, mean+SD (min)	36.1+16.2	48.3+30	0.24
Bypasstime, mean+SD (min)	74.4+28.6	91.3+30.1	0.63
Cardioplegia time, mean + SD (min)	13.4+3.7	16+4.9	0.45
Retrograde cardioplegia	46.7%	28.6%	0.64
IABC	5 (55.6%)	4 (44.4%)	0.24

was installed in two of the three patients. The ischemic time was 30.3 minutes in average. One patients suffered perioperative infarction, and none required postoperative intra-aortic balloon counterpulsation (IABC). One female patient died 8 days postoperatively with multiorgan failure. During PTCA of a proximal 90% left anterior descending coronary artery (LAD) stenosis, an acute extensive medial dissection occurred, due to her unstable condition, preoperative IABC had to be instituted. The operative intervention was coronary artery bypss grafting to the LAD and its diagonal branch.

Discussion

A collective review of literature (2-19) indicates that the overall failure rate leading

Table 6: Summary of previously published and the present studies

Author	PTCAs done	Emergency CABG	Postop. MI	Postop. death
Jones et al, 84	777	41 (5.3%)	9/41 (18%)	1/41 (2/4%)
Colwley et al, 84	3079	202 (6.6%)	90/202 (45%)	13/202 (6.4%)
Bredlau et al, 85	3500	96 (2.7%)	47/96 (49%)	2/96 (2.1%)
Acinapura et al, 85	198	21 (10.6%)	8/21 (38%)	0 (0%)
Shiu et al, 85	240	14 (5.8%)	5/14 (36%)	1/14 (7.1%)
Pelletier et al, 85	299	35 (11.7%)	10/35 (29%)	0 (0%)
Killen et al, 85	NA	115	46/115 (40%)	13/115 (11/3%)
Golding et al, 86	1831	81 (4.4%)	35/81 (57%)	2/81 (2.5%)
Page et al, 86	NA	44	14/44 (32%)	3/44 (6.8%)
Lazar et al, 87	1045	24 (2.3%)	15/24 (63%)	3/24 (12.5%)
Parsonnet et al, 88	958	67 (7.0%)	19/67 (28%)	8/67 (11.9%)
Connor et al, 88	996	146 (14.7%)	57/146 (39%)	4/146 (4.4%)
Naunheim et al, 89	2418	103 (4.3%)	23/103 (22%)	11/103 (10.7%)
Hrphongse et l, 90	1300	26 (1.9%)	16/26 (61%)	1/26 (3.8%)
Bottner et al, 91	NA	64	34/64 (53%)	9/64 (14.0%)
Sievers et al, 91	1514	55 (3.6%)	25/55 (45%)	3/55 (5.4%)
Buffet et al, 91	2576	100 (3.9%)	57/100 (57%)	19/100 (19.0%)
Lazar et al, 92	2900	53 (1.8%)	29/53 (55%)	6/53 (11.3%)
Average		5.8%	43%	7.3%
This work	4885	29 (0.5%)	9/29 (31%)	1/29 (3.4%)

to emergency coronary artery bypass grafting is 5.8% (table 6). Extended medial dissection and acute occlusion were most often cited as reasons for proceeding with coronary artery bypass grafting in our study. These primary indications were also most frequent in the studies of Jones et al, 1984; Cowley et al, 1984; Pelletier et al, 1985; Golding et al, 1986; Lazar et al, 1987; and by Parsonnet et al, 1988 (2, 3, 7, 9, 11, 12). Many patients have multiple indications for termination of the PTCA and proceeding with coronary artery bypass grafting (ie, dissection plus acute obstruction, occlusion plus unstable angina). In the present study 3 patients had perforated coronary arteries, necessating surgical intervention.

Ferguson and associtates, 1986 (20) reported on the use of an intravascular stents across stenoses after failed PTCA. They noted that the stent allowed stabilization of the patients and performance of a more optimal bypass procedure, including use of the internal mammary artery.

In the present study stacks were used for the same purpose in 6 patients, 4 of which

were saved from perioperative infarction, while 2 other patients had perioperative infarction.

The LIMA was used in 3 patients in this study, none of them had a stack, and one of which had a septoapical infarction perioperatively. It is beleived that if the patient reverts to a state, in which there is no angina and there are no ongoing electrocardiographic changes consistent with ischemia, it would be safe to spend additional time necessary to take down the LIMA. This opinion is shared by Naunheim et al, 1989 (14).

As regards myocardial preservation, we advocated a protocol of normothermic induction of blood cardioplegia, cold maintenance blood cardioplegia, and normothermic reperfusion of blood cardioplegic solution during emergency bypass grafting in the setting of failed angioplasty.

Jones and associates, 1981 (21) stated that prolonged ischemic injury results in depletion of myocrdial energy stores and conversion to anaerobic metabolism, with the subendocardial region being most severely affected.

Rosenkranz and colleagues in 1982 (22) demonstrated in dogs that recovery of high-energy phosphate stores and reversal of anaerobic metabolism, as well as recovery of global left ventricular function, were improved when a 5 minute period of normothermic induction of cardioplegia was employed, suggesting that normothermic induction may result in greater metabolic reserve in the jeopardized myocardial bed.

Lazar and colleagues in 1987 (11) have shown that, in dogs undergoing reversible ischemic damage, a reactive hyperemia ensues when the myocardium is still viable, with consumption of oxygen beyond that necessary to meet the demands of mechanical function. It has been postulated that oxygen may be used in the repletion of high-energy phosphate stores and in cellular repair processess. These authors demonstrated that myocardial oxygen uptake markedly exceeded basal requirements during normothermic reperfusion of blood cardioplegic solution. Inhibiting electromechanical work with cardioplegic solution infusion while maintaining normothermia may allow maximum diversion of oxygen to oxygen-requiring reparative processes. Findings consistent with these hypotheses have been reported by Teoh and associates (23) in 20 patients undergoing elective bypass grafting. The infusion of normothermic cardioplegic solution before crossclamp release resulted in improved metabolic recovery, with higher levels of high-energy phosphates and reduced left atrial pressures during recovery.

When patients were subjected to emergency coronary artery bypass grfting after failed angioplasty, the goal of complete revascularization was not attained according to several reports: Pelletier et al, 1985; Page et al, 1986; Lazar et al, 1987; Parsonnet et al, 1988; and by Greene et al, 1991 (7, 10, 11, 12, 24). They all reported an average of 2.0 grafts or less per patient despite a significantly higher expected number of grafts based on extent of stenotic disease. Zorn (25) has

shown that incomplete revascularization adversely affects 5-year survival. In the present study, the patients were completely revascularized with an average number of grafts 1.8 in the patient group with an average number of stenotic vessels 1.6.

In the present study, 9 patients (31%) required intraaortic balloon pump support postoperatively. This is much higher than the percentages reported by Jones 1984 (4%), Parsonnet 1988 (7.5%), Brahos 1985 (10.3%), and their associates (2, 12, 26).

The reported incidence of postoperative myocardial infarction after emergency coronary artery bypass grafting for failed PTCA ranges from 18% to 63%, with an average of 43% (table 6). In the present study, 9 patients (31%) had electrocardiographic and enzyme evidence of acute postoperative myocardial infarction.

Even though none of the patients in the present study received thrombolytics, the need for blood transfusions and administration of fresh frozen plasma and platelets was increased in patients undergoing emergency CABG. Blood drainage was 1748 ml per patient in average, 5.8 units of packed RBCs, 2.2 units of fresh frozen plasma and 1 unit of platelets were given in average per patient. This same trend was reported by Brahos 1985. Parsonnet 1988, Borkon 1992, and their associates (26, 12, 27). Presumably, antiplatelet agents and high doses of preoperative heparin led to increased intraoperative bleeding (27). Despite this finding, postoperative chest tube drainage and the incidence of reexploration for bleeding were not dissimilar for either the emergency or elective CABG group.

In the present study, the death rate after coronary artery bypass grafting for failed angioplasty was 1/29 (3.4%). As shown in table 6, the average mortality in the collective group of patients who underwent emergency coronary artery bypass grafting for failed PTCA in the reviewed literture was 7.3%.

Conclusion

In summary, emergency coronary artery bypass surgery after failed PTCA carries a greater risk of perioperative death and complications than elective coronary artery bypass grafting. Our study indicates 0.5% of patients undergoing PTCA require emergency coronary artery grafting; 3.4% of those will die, and 31% will sustain perioperative myocardial infarction.

REFERENCES

1. Gruentzig AR: Transluminal dilatation of coronary artery stenosis. Lancet 1978; 1:263.
2. Jones El, Murphy DA, Craver JM: Comparison of coronary artery bypass surgery and percutaneous transluminal coronary angioplasty including surgery for failed angioplasty. Am Heart J 1984; 107:830.
3. Cowley MJ, Dorros GF, Kelsey SF, Van Raden M, Detre KM: Emergency coronary bypass surgery after coronary angioplasty: The NHLBI's PTCA registry experience. Am J Cardiol 1984; 53:22c.

4. Bredlau CE, Roubin GS, Leimgruber PP, Douglas JS Jr, King SB, Gruentzig AR: In-hospital morbidity and mortality in patients undergoing elective coronary angioplasty. Circulation 1985; 72:1044.

5. Acinapura AJ, Cunningham JN Jr, Jacobowitz IJ: Efficacy of percutaneous transluminal coronary angioplsty compared with single vessel bypass. J Thorac Cardiovasc Surg 1985; 89: 35.

6. Shiu MF, Silverton NP, Oakley D, Cumberland D: Acute coronary occlusion during percutaneous transluminal coronary angioplsty. Br Heart J 1985; 54: 129.

7. Pelletier LC, Pardini A, Renkin J, David PR, Hebert Y, Bourassa MG: Myocardial revascularization after fialure of percutaneous trasluminal coronary angioplasty. J Thorac Cardiovasc Surg 1985; 90: 265.

8. Killen DA, Hamaker WR, Reed WA: Coronary artery bypass following percutaneous transluminal coronary angioplasty. Ann Thorac Surg 1985; 40: 133.

9. Golding LA, Loop FD, Hollman JL: Early results of emergency surgery after coronary angioplasty. Circulation 1986; 74 (suppl 3): 26.

10. Page US, Okies JE, Colburn LQ, Bigelow JC, Salomon NW, Krause AH: Percutaneous transluminal coronary angioplasty, a growing surgical problem. J Thorac Cardiovasc Surg 1986; 92: 847.

11. Lazar HL, Haan CK: Determinants of myocardial infarction following emergency coronary artery bypass for failed percutaneous coronary angioplasty. Ann Thorac Surg 1987; 44: 646.

12. Parsonnet V, Fisch D, Gielchinsky I: Emergency operation after failed angioplasty. J Thorc Cardiovasc Surg 1988; 96: 198.

13. Connor AR, Vlietstra RE, Schaff HV, Ilstrup DM, Orszulak TA: Early and late results of coronary artery bypass after failed angioplasty. J Thorac Cardiovasc Surg 1988; 96: 191.

14. Naunheim KS, Fiore AC, Fagan DC: Emergency coronary artery bypass grafting for failed angioplasty: risk factors and outcome. Ann Thorac Surg 1989; 47: 816.

15. Haraphongse M, Na-Ayudhya RK, Burton J, Tymchak W, Lucas A, Humen D, Montague T: Clinical efficacy of emergency bypass surgery for failed coronary angioplasty. Can J Cardiol 1990; 6: 186.

16. Bottner RK, Wallace RB, Visner MS, Stark KS, Recientes E, Katz NM, Hopkins RA, Patrissi GA, Kent KM: Reduction of myocardial infarction after emergency coronary artery bypass grafting for failed coronary angioplasty with use of a normothermic reperfusion cardioplegia protocol. J Thorac Cardiovasc Surg 1991; 101: 1069.

17. Sievers B, Schofer J, Kalmar P, krebber HJ, Bleifeld W: Results of emergency bypass operation following percutaneous transluminal coronary angioplasty. Z Kardiol 1991; 80: 506.

18. Buffet P, Danchin N, Villemot JP, Amrein K, Ethevenot G, Juilliere Y, Mathieu P, Cherrier F: Early and long-term outcome after emergency coronary artry bypass surgery after failed coronary angioplasty. circulation 1991; 84 (suppl 3): 254.

19. Lazar HL, Faxon DP, Paone G, Rajaii-Khorasani A, Jacobs AK, Fallon MP, Shemin RJ: Changing profiles of failed coronary angioplasty patients: impact on surgical results. Ann Thorac Surg 1992; 53: 269.

20. Ferguson TB Jr, Hinohara T, Simpson J, Stack RS, Wechsler AS: Catheter reperfusion to allow optimal coronary bypass grafting following failed transluminal coronary angioplasty. Ann Thorac Surg 1986; 42: 399.

21. Jones RN, Peyton RB, Sabina RC: Transmural gradient in high energy phosphate content in patients with coronary artery disease. Ann Thorac Surg 1981; 32: 546.

22. Rosenkranz ER, Vinten-Johansen J, Buckbery GD, Okamoto F, Edwards H, Bugyi H: Benefits of normothermic induction of blood cardioplegia in energy-depleted hearts, with maintenance of arrest by multidose cold blood cardioplegic infusions. J Thorac Cardiovasc Surg 1982; 84: 667.

23. Teoh KH, Christakis GT, Weisel RD: Accelerated myocardial metabolic recovery with terminal warm blood cardioplegia. J Thorac cardiovasc Surg 1986; 91: 888.

24. Greene MA, Gray LA Jr, Slater AD, Ganzel BL, Mavroudis C: Emergency aortocoronary bypass after failed angioplasty. Ann Thorac Surg 1991; 51: 194.

25. Zorn GL: Stenotic arteriosclerotic coronary artery disease. In: Kirklin J, Barratt-Boyes B, eds. Cardiac surgery. New York: Churchill Livingstone, 1988; 228.

26. Brahos GJ, Baker NH, Ewy GG: Aortocoronary bypass following unsuccessful PTCA: experience in 100 consecutive patients. Ann Thorac Surg 1985; 40: 7.

27. Borkon AM, Trent LF, Piehler JM, Killen DA, Hoskins ML, Reed WA: Risk analysis of operative intervention for failed coronary angioplasty. Ann Thorac Surg 1992; 54: 884.

Dendriform pulmonary ossification a case report

Abstract

Dendriform pulmonary ossification (DPO) is a very rare lung disease of uncertain pathogenesis; It is characterized by the formation of mature bone in the interstitial connective tissue of the lung.

The disease was first desctibed by Luschka in 1856, and since then about one hundred cases have been reported in the literature.

We report a case of DPO found incidentally in an adult female operated on as an emergency case due to ruptured dissecting aortic aneurysm.

Key words: Pulmonary ossification, Idiopathic, Dendriform, Diffuse.

Fayez Khaled Hajjiri, MD Bassam Akasheh Mrcs. Abd Ellatif Oklah F.R.C.S.

From the department of pathology & cardiovascula surgery, King Hussein Medical Center, Royal Medical services, Amman-Jordan

Introduction

Pulmonary ossification is generally divided in two distinct types: nodular and branching. The nodular type is characterized by the formation of mature lamellar bone 2-8mm in diameter free in the alveolar space and usully does not contain marrow elements, this type is not infrequent and can occur in conditions associated with pulmonary venous hypertension, in particular Mitral Stenosis. DPO, the branching type is charcterized by the deposit of branching bony specules, of 1-4mm thickness within the alveolar septae, and usually contains marrow tissue, it is a very rare lung disease and can be found in association with pulmonary diseases that have interstitial fibrosis as a common pathological lesion. DPO is distinct clinically, aetiologically and pathologically from calcinosis of the lung, associated with dystrophic or metastatic calcium deposits, and also unrelated to Bronchopathia/Tracheopathia osteopastica which is pulmonary ossification found beneath the mucous membrane of larger bronchi and trachea of patients with long standing bronchitis.

Case report

A 46-year-old Jordanian female patient was admitted as an emergency case to Queen Alia Heart Institute, Royal Medical Service, Amman, in a state of shock, with a history of severe acute chest pain. Acute aortic dissection was suspected by x-ray film. Emergency cardiac catheterizaion confirmed the presence of ascending aortic dissection extending into the arch and descending thoracic aorta, there was no haemodynamic evidence of any other cardiac abnormality. There was no significant clinical complaint

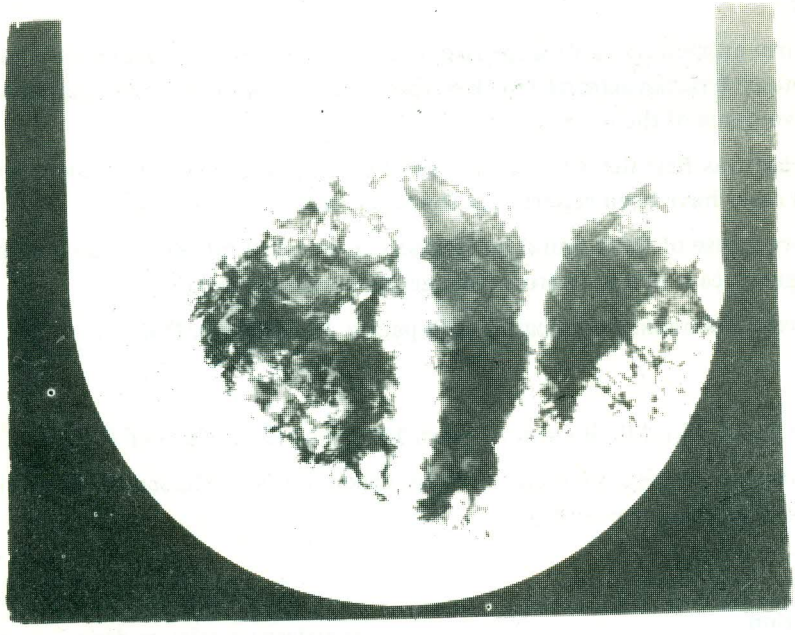


Fig. 1: The branched structure of bone shown in the postmortem radiograph of lung.

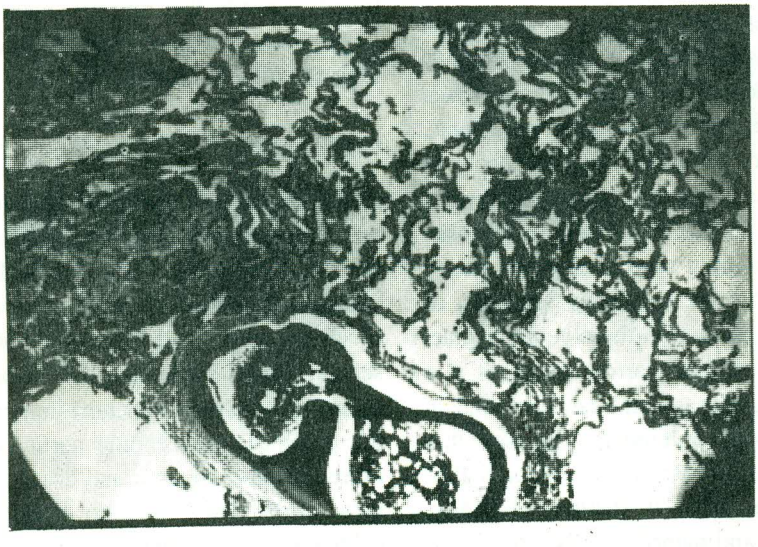


Fig. 2: Rods of bone enclosing haemopeitic tissue situated in areas of alveolar septal fibrosis (H and E X 40).

previously documented regarding her pulmonary or cardiovascular systems.

Emergency surgery was performed and she was found to have an ascending aortic dissection extending into the arch and rupturing into the pericardium with free blood in the anterior mediastinum and left pleura. Replacement of the aortic arch was performed with reimplantation of the head and neck vessels, and the left hemithorax was evacuated. The left lung felt bony hard. The patient died during operation because of uncontrolable bleeding.

No autopsy was performed; The left lung was excised for pathological examination.

The gross examination of formalin-fixed lung revealed hard rods of bone diffusely affecting the lung, a postmortem radiograph of the lung revealed the branched structure of the bony deposits diffusely affecting the whole lung and is shown in figure 1; Decalcifiction of tissue blocks was needed prior to tissue processing and staining. The right lung was normal on palpation during surgery but a minimal degree of involvement can't be entirely excluded. The microscopic examination revealed rods of trabecular bone enclosing normal haemopeitic bone marrow elements situated in areas of alvolar septal fibrosis, the adjecent lung parenchyma shows no remarkable pathology. The fibrosis is an end stage focal pulmonary fibrosis, no active lung disease is seen as to determine its a etiology. An H and E histology section is shown in figure 2.

Discussion

DPO is a very rare lung disease, cases have been reported in association with a group of diseases that have in common pulmonary fibrosis, such as Hamman Rich lung[1], cystic fibrosis[2], amyloidosis[3], histoplsmosis[4], chronic abscess[5] asbestosis[6], tuberculosis[7], and busulfan therapy[8]. Ronald et al[6] reported a case of DPO in association with chronic myeloid leukaemia despite abscence of busulfan therapy, the bone specules encase a hypercellular leukaemic bone mrarow. Daust[9] in 1929 reported two cases, he described the pathogenesis of the ossifiction as a degenerative lesion in the arterial media as an initial process, followed by hyalinization, osteoid deposition and finally formation of bone. There is a tendency for general agreement that the bone deposits are derived from a metaplastic process in areas undergoing fibrous transformation (6,10,11,) found that an extensive degree of interstitial fibrosis was not necessary for bone deposition to take place, he added that the metaplastic bone occurs in the thickened alveolar septae. We fully agree with this evidence, the histopathological findings in our case report are similar, the fibrosis is focal and the metaplastic bone deposits are limited to these areas, we also agree that the cause-and- effect relationship is not predictable since the associated conditions are common world wide and DPO is very rare, to our knowledge probably this is the first documented case in our country.

DPO is an irreversible pathological lesion, progression is slow and regression has not been recorded [12].

A severe degree of involvement of the left lung is noted in our case report, however DPO is predominantly affecting the lower lobes, often bilaterally, and most prominently subpleural[6], the bone deposits are not relted to the bronchi or pulmonary vessels, in its more severe form the whole lobe or lung may become transformed into a mass of oseous tissue [4,6,13].

The degree of pulmonary dysfunction is usually minimal, the diagnosis of DPO in most cases reported in the literature was made at autopsy performed for other reasons. DPO is usually unrecognized radiologically during life. Knowledge about this disease is very helpful in the differential diagnosis of a number of conditions which may show similar reticulated or branching pattern by x-ray films such as diffuse interstitial fibrosis, lymphangitis carcinomatosa, bronchiectasis, and others.

REFERENCES

1. Mendeloff J: Disseminted nodular pulmonary ossification in the Hamman Rich lung. Am Respir Dis 103: 269-274, 1971.
2. Takayama K, Koizuka S, Sudo M, et al: Autopsy report of cystic fibrosis of lung with multiple small nodules of bone. Nippon kyobu shikkan, Gakkai Zasshi 14: 585-591. 1976.
3. Nagy L, Stekker K: Pneumopathia osteoplastica. Zentralbl Allg Pathol 101: 334-337, 1960.
4. Felson B, Schwarz I, Lukin R. Hawkins H: Idiopathic pulmonary ossification. Radiology 153: 303-309, 1984
5. Steinmetz D, Platau E, Daharan M: Pulmonary ossification. Isr J Med Sci 21: 703-705, 1985.
6. Ronald W. Joines, Victor L. Roggli; Dendriform pulmonary ossification. Am J Clin pathol 91: 398-402, 1989.
7. L.T.C. Chow, B.S.F. Shum, W.H. Chow, C.B. Tso; Diffuse pulmonary ossification-a rare complication of tuberculosis, Histopathology 20: 435-437, 1992.
8. Kuplic J.P., Higley C.S., Niewoehner D.E.: Pulmonary ossification associted with long term busulfan therapy in chronic myeloid leukaemia. Am Rev Respir Dis 106: 759-762, 1972.
9. Daust W. Uber verastelte knochenspangenbildung in der lung. Frankfurt Z Patholi 37: 313-327 (Ger.), 1929.
10. Bert Lincoln Pear: Idiopathic disseminated pulmonary ossification. Radiology October 91: 746-748, 1968.
11. Fried ED; Gowin TA: Extensive diffuse pulmonary ossification. chest Nov; 102(5); 1614-5, 1992.

12. Felson B: thoracic calcifications. Dis chest 56: 330-343, 1969.

13. Spencer H: Pathology of the lung (Excluding pulmonary tuberculosis). Third edition, volume 2, pergamon press 689-690.

Index of deterioration of patients with prosthetic valve malfunction

Abstract

To describe the rate of deterioration of patients with malfunctioning prosthetic cardiac valves; we designed a numerical index relating advancement in NYHA functional classing to time of its occurrence. Our proposed "Index of Deterioration" (ID) was calculated for 75 patients (mean ± SD; 2±3.18) previously reoperated for valve malfunction at our institution. Each type of malfunction had significantly different ID from other types, as well as for the same malfunction occurring at different timing (P<0.001). Malfunctions presenting early during the first 2 postoperative years had a higher ID (2.68 ± 3.55) than those presenting later on (0.38 ± 0.62))P<0.01). Moreover, the mean ID of the first postoperative month malfunctions (5.74 ± 1.1) was higher than that of the former (1.1±0.94); (P<0.001).

The calculated ID correlated positively with cardiopulmonary bypass time (P<0.01). The mean ID of hospital mortalities (4.4±3.69), emergently reoperated patients (3.9±4) and patients dependent on positive inotropic support postoperatively (2.23±3.46) was significantly higher (P<0.01) than that of hospital survivors (1.6±0.9) electively reoperated patients (0.9±1.8) and haemodynamically stable patients (0.7±0.7); respectively. Besides its usefulness in comparing patients populations, the suggested ID may help setting surgery timing properly. In this study, ID >1 and >3 were significantly related to emergency reoperation (P<0.001) and hospital mortality (P<0.01); respectively. Other studies however, are warranted to define the true figures.

Ahmed Hassouna, MD.

Department of Cardio thoracic Surgery, Ein Shams University

Introduction

Reoperation upon malfunctioning prosthetic cardiac valves has become part of the routine work of all cardiac surgeons. Authors have noted the time lag between the patient's symptoms and reoperation, even in the so-called catastrophic malfunctions (1,2). On the other hand, from the many reports of such operations, advanced NYHA functional class is the shared common risk factor (3,4,5,6). In this study, we were tended to couple both information by designing a numerical index describing the rate at which such patients deteriorate in terms of NYHA functional classing.

Methods

In the period between january 1980 and january 1990, 75 patients were reoperated upon for prosthetic valve malfunction (PVM) at our institution. They were 38 males and 37 females whose age ranges from 16 to 43 years with a mean of 32.14±14.5 (SD) years. Preoperative selected variables including type of PVM, type and position of cardiac prosthesis, main presenting symptom and auscultatory finding are listed in (Table 1). All patients were hospitalized, benefiting from a full echocardiographic and doppler study as well as routine laboratory investigations. The latter included complete blood picture, ESR, renal and hepatic profiles. Three successive blood cultures were taken from each of the 16 febrile patients before subscription of an antibiotic regimen.

Twenty-seven patients all class IV NYHA- were operated upon on an emergency basis within 3±2.6 hours of taking the operative decision. They were 9 cases with prosthetic valve endocarditis (PVE), 7 with prosthetic valve thrombosis (PVT), 9 with prosthetic valve disinsertion (PVD) and 2 cases with primary bioprosthetic valve failure (PBVF). The other patients -22 class III and 26 class IV NYHA - were electively scheduled for surgery.

All operative procedures were performed under cardiopulmonary bypass, moderate hypothermia (28°C) and multidose -every 25 minutes - cold potassium cardioplegia. Both cavae were cannulated for venous drainage and the ascending aorta (in 80% of the cases) or the femoral artery was used for arterial return. All malfunctioning prostheses were replaced, except in patients with mechanical prostheses implanted for less than 4 years and disinserted over less than one-third of the ring circumference (14 patients ; 18%); they benefited from simple reinsertion using interrupted mattress suturess buttressed with teflon pledgets. We had 2 operative and 8 postoperative mortalities (5 PVE, 3 PVT and 2 PVD; 13.3%) mainly due to low cardiac output. Postoperative positive inotropic support was needed in 78.6% of our cases.

The files of these patients were reviewed. The total duration of implantation of each prosthesis was divided into 2 intervals: the latent and manifest periods of malfunction (LPM and MPM; respectively). The former describes an asymptomatic patient with a well functioning prosthesis. The latter starts when the patient develops the first symptom or sign suggesting PVM and ends with reoperation. Clinical deterioration was assessed according to the guidelines of the NYHA functional classification. In order to obtain a numerical value describing the rate at which such deterioration occurred, and for each patient, we assigned for the NYH functional classes I, II, III, and IV the numbers 1, 2, 3 and 4 ; consecutively. The patient's numerical class at the end of MPM (x) was subtracted from

Table 1: Patients presenting with prosthetic valve malfunsction: preoperative selected variables.

	PVD	PVE	PVT	PBVF	Total	%
1. Number of patients:	36	16	13	10	75	
2. Number of prostheses:	39	18	15	10	82	
3. Type of prosthesis:						
a) Bjork-Shiely (cc)	18	8	8	0	34	41%
b) Starr-Edwards (1620,6120)	17	5	5	0	27	33%
c) Carpentier-Edwards	4	5	2	10	21	26%
4. Position of prosthesis:						
a) aortic	19	7	7	6	39	52%
b) mitral	14	7	4	4	29	39%
c) both mitral & aortic	3	2	2	0	7	9%
5. Main presenting symptom:						
a) shortness of breath	25	1	4	7	37	49%
b) TEM	3	0	2	2	7	9%
c) palpitations	1	0	0	1	2	3%
d) general toxic symptoms	0	2	0	0	2	3%
e) cardiogenic shock	2	1	4	0	7	9%
f) a + b	1	2	3	0	6	8%
g) a + c	3	0	0	0	3	4%
h) a + d	1	10	0	0	11	15%
6. Main auscultatory finding:						
a) appearance of new murmur	31	12	6	10	58	77%
b) changing murmur	0	4	2	0	6	8%
c) absent prosthetic click	0	0	3	0	3	4%
d) irrelevant	5	1	2	0	8	11%

PVD= prosthetic valve disinsertion, PVE= prosthetic valve endocarditis,
PVT= prosthetic valve thrombosis, PBVF= primary bioprosthetic valve failure, TEM= thromboembolic manifestations.

his numerical class during LPM (y), then divided by the length of MPM in months (z). The results was a rate, assigned as the Index of Deterioration (ID) of this patient.

$$ID = \frac{x - y}{z}$$

In the absence of a statistically significant difference in type or position of the cardiac prostheses among the different types of PVM ; our strategy was to correlate the durations of LPM and MPM for the different types of malfunctions as well as for the whole group of patients. In the presence of such correlation, patients were then divided into 2 subgroups : those manifesting during the first two postoperative years of prosthetic implantation (early wave PVM) and patients presenting afterwards (late wave PVM). LPM and MPM of each subgroup were also correlated.

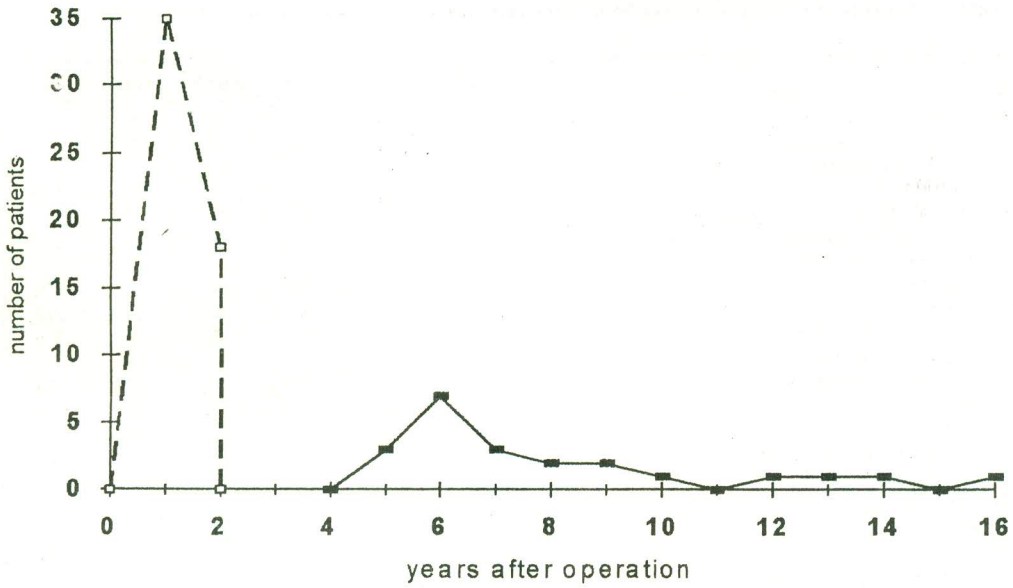


Fig. 1: Waves of prosthetic valve malfunctions

The mean durtion of MPM was initially compared between both waves, then among different types of malfunctions within the same wave, and lastly, for the same given type of malfunction presenting in different waves. The same procedure was followed for the calculated ID of the patients. The value of the latter as a risk factor associated with hospital mortality and morbidity was finally evaluated.

Results were expressed as mean ± standard deviation. Quantitative variables were correlated by using the coefficient of correlation and Spearman's test (7). Means were compared by Student and Kruskall - Wallis tests (7). The independence among qualitative variables was assessed by the Chi-Square test. Statistical significance was considered with a P value < 0.05.

Results

The LPM varied from 1 day to 15.4 years with a mean duration of 31.5±41.3 months. Chronologiclly, 2 groups of patients were distinguished; those presenting during the first two postoperative years of "early wave PVM" and those presenting from the fifth to the sixteenth postoperative year or "late wave PVM" (figure 1). In between, no patient manifested clinically with PVM. With the exception of 4 patients (5.3%) who failed to improve in the early postoperative period

Table 2: Correlation between the latent and manifest periods of malfunctions (in months):

Malfunction	N	LPM	MPM	r	P value
1.PVE	16	27.8±39.9	1.7±1.3	0.35	> 0.05
2.PVT	13	18.3±24.1	6.4±13.3	0.76	<0.01
3. PVD	36	35.2±27.4	14±10.9	0.55	<0.01
4. PBVF	10	41.1±37.1	47±3.4	0.88	<0.01
- Early wave	53	10.7±7.9	4.9±9.9	0.34	0.02
- Late wave	22	81.3±37.6	18.2±17.5	0.47	0.02
Total	75	31.5±41.3	8.8±14.2	0.58	<0.001

N = number of patients, LPM= latent period of malfunction (period between valve replacement and the development of symptoms or signs suggesting valve malfunction), MPM = manifest period of malfunction (period following LPM and ending by reoperation), r= coefficient of correlation of Spearman, (PVE, PVT, PVD and PBVF =see table 1), Early wave = malfunctions presenting during the first 2 postoperative years, Late wave= malfunctions presenting from the fifth to the sixteenth year following valve replacement.

Table 3: Timing of malfunction presentation and the manifest perlod duration (in months).

Malfunction	Early wave			Late wave			P value* (MPM †)
	N	LPM	MPM	N	LPM	MPM	
a) PVT	10	6.1±9.1	0.95±1.5	3	59.3±1.2	24.6±20.2	< 0.02
b) PVE	12	7.1±9.3	1.4±1.3	4	90±27.5	2.5±1.3	>0.05
c) PVD	26	22.9±8.9	9.6±4.8	10	92.9±51.9	25.5±19.8	0.04
d) PBVF	5	8.4±2.2	1.6±0.5	5	73.8±20.6	7.8±1.8	<0.01
P value*		> 0.05	< 0.001		> 0.05	< 0.001	

*** = Kruskall and Wallis test, † = comparison of the manifest periods of a given malfunction presenting in different waves, N = number of patients,**

(Early wave, Late wave, LPM, MPM, PVT, PVE, PVD and PBVF= see tables 1 and 2).

(2 PVD; 5.5%, 1 PVE; 6.3% and 1 PVT ; 7.7%); MPM was always shorter than LPM. It varied from 6 hours to one year with a mean value of 8.81±14.2 months. There was a statistically significant positive correlation between the durations of LPM and MPM (P<0.001). This significance was maintained per wave, and with the exception of PVE, per type of PVM (table 2).

Neither the duration of LPM nor that of MPM were related to the type of malfunction per se. However, when their timing of presentation was considered, early wave PVM had a significantly shorter MPM (4.9±9.9 months) than late presenting malfunctions (18.2±17.5 months) (P<0.001). Ech PVM had its significantly different MPM from that of other malfunctions sharing the same wave of

Table 4: Timing of malfunction presentation as a risk factor in reoperation.

	Early wave		Late wave	P value *
	Initial rise patients †	Other patients		
Number of patients	18	35	22	
1. Emergency reoperation	15 (83%)	10 (28%)	2 (9%)	<0.001
2. Hospital mortality	5 (28%)	4 (11%)	1 (5%)	0.08 ‡
3. Postoperative low cardiac output §	16 (100%)	29 (83%)	14 (64%)	0.03 ‡

(Early wave, Late wave= see table 2), * = Chi-Square test,

† = ptients presenting with valve malfunction in the first postoperative month following valve replacement,

‡ = Early versus Late wave of malfunction § = the 2 operative mortalities are excluded.

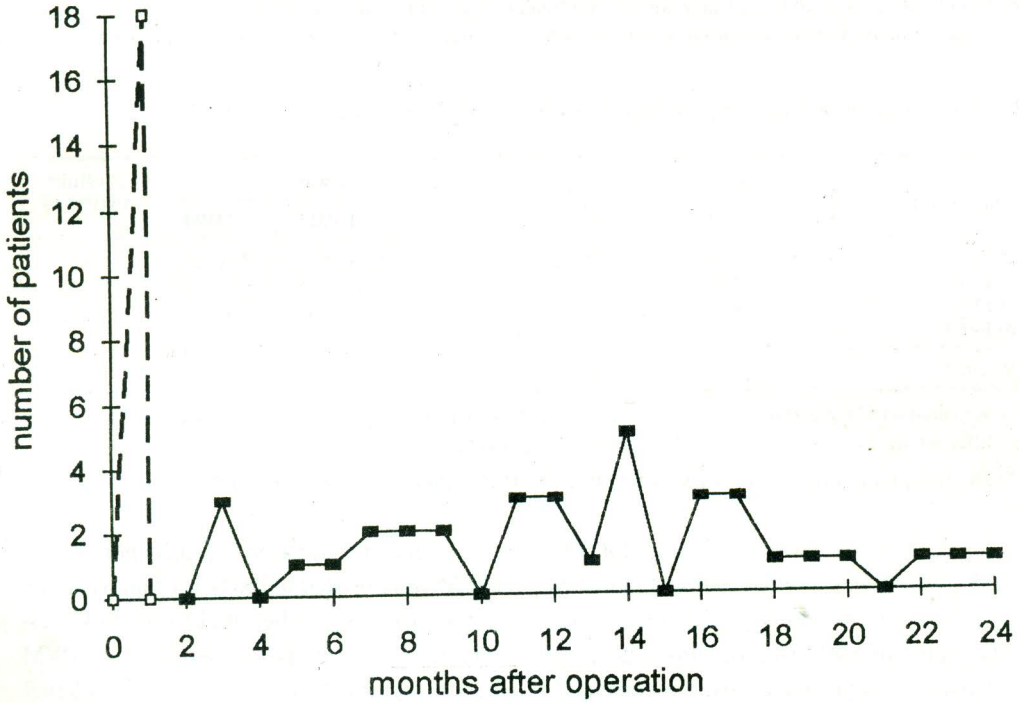


Fig. 2: Early wave of prosthetic valve malfunctions

presentation (P<0.001). Moreover, and with the exception of PVE, MPM of a given malfunction was significantly shorter when presenting early during the first 2 postoper-

ative years following valve replacement than when presenting later on (table 3).

Early presentation of PVM was associated with a higher need for emergency reoperation (P< 0.001), postoperative inotropic positive inotropic support (P = 0.03) and a higher hostpital mortality rate (P = 0.08); when compared to late wave PVM (table 4). Early wave PVM showed an initial rise during the first postoperative month (figure 2), yielding one-third of the patients, equally presented as disinserted, thrombosed and infected prostheses. Most of these patients were reoperated upon on an emergency basis (83%) and all the survivors were dependent on positive inotropic support postoperatively. Moreover, our 2 operative mortalities and half of our overall hospital mortalities were among this group of patients (P = non significant).

In terms of NYHA functional classing, our patients showed a significant deterioration (P<0.001) from a mean value of 1.7±0.86 during LPM to a mean of 3.7±0.45 at the end of MPM; with no statistically significant difference among various types of PVM. On the other hand, the rate of functional deterioration, as assessed by the Index of Deterioration (ID) was highly related to the type and time of malfunction presentation (table 5 and 6). Early wave PVM patients had a higher ID (2.68±3.55) than late wave PVM patients (0.38±0.62) (P<0.001). Moreover, the ID of those presenting during the initial rise (5.7±1.1) was significantly higher than that of others sharing early wave PVM (1.1±0.94) (P<0.01).

Patients reoperated upon on an emergency basis had a significantly higher ID (3.9±4) than electively reoperated ones (0.9±1.8);(P< 0.001). Out of the former, 25 patients (92.6%) had an ID >1 as compared to only 5 patients (10.4%) of the latter (P< 0.01). The mean ID of the 10 hospital mortalities (5 PVE, 3

Table 5: Index of Deterioration calculated for Early and Late waves of prosthetic valve malfunctions.

Malfunction	Early wave			Late wave			P value *
	N	ID		N	ID		
		range	mean±SD		range	mean±SD	
a) PVT	10	0.6-15	6.6±15	3	0.02-0.25	0.14±0.11	
b) PVE	12	1-12	4±3.7	4	0.5-3	1.25±1.2	
c) PVD	26	0.2-3	0.9±0.8	10	0.03-0.25	0.17±0.09	
d) PBVF	5	1-2	1.5±0.5	5	0.1-0.5	0.26±0.09	<0.001
Total	53	0.2-15	2.68±3.55	22	0.02-3	0.38±0.62	<0.01

(Early wave, Late wave, PVT, PVE, PVD and PBVF =see tables 1 and 2),

ID = Index of Deterioration, N = number of patients,

*** = Kruskall and Wallis test.**

Tble 6: Index of Deterioration calculated for Initial rise patients

Malfunction	Initil rise patients			Other Early wave patients			P value *
	N	ID		N	ID		
		range	mean±SD		range	mean±SD	
a) PVT	6	4.5-15	9.6±1.6	4	0.6-6	2.1±2.6	0.05
b) PVE	6	2-12	6±1.8	6	1-3	1.8±3.3	0.01
c) PVD	6	0.66-3	1.6±0.32	20	0.16-2	0.7±0.09	0.01
d) PBVF				5	1-2	1.5±0.5	
Total	18	0.66-15	5.74±1.1	35	0.16-3	1.1±0.94	<0.001

(Initial rise patients, Early wave, Late wave, PVT, PVE, PVD and PBVF= see tables 1,2&4), ID- Index of Deterioration, N= number of patients, * = Kruskall and Wallis test.

Table 7: Index of Deterioration: a risk factor in reoperation of prosthetic valve malfunctions.

Variable	N	%	ID	P value
1) Necessity of reoperation				
a- elective	48	64%	0.9±1.8	
b- emergency	27	36%	3.9±4	<0.01*
- electively reoperated patients with an ID > 1	5	10.4%		
- emergently reoperated patients with an ID > 1	25	92.6%		
2) Outcome of surgery				<0.01 †
a- hospital survivors	65	86.6%	1.65±2.9	
b- hospital mortalities	10	13.3%	4.4±3.69	< 0.01*
- hospital survivors with an ID > 3	8	12.3%		
- hospital mortalities with an ID > 3	8	80%		
3) Time of cardiopulmonary bypass 127.9+31.9 minutes.				
4) Postoperative inotropic support §	75		2±3.18	< 0.01 ‡
a- necessary	59	80.8%	2.23±3.46	
b- unnecessary	14	19.2%	0.7±0.7	<0.001*

N = number of patients, ID = index of deterioration,

*** = Krushall & Wallis test, † = Chi-Square test,**

‡ = Spearman's test, § = excluding 2 operative mortalities (ID = 3 & 6)

PVT and 2 PVD) was significantly higher (4.4±3.39) than that of the 65 hospital survivors (1.65±2.9);(P< 0.01). The calculated ID for 80% of the former was >3 as compared to only 12.3% of the latter (P< 0.01); (table 7).

There was a statistically significant positive correlation between the duration of cardiopulmonry bypass and the patient's ID (P< 0.01). With exclusion of the 2 operative mortalities, 59 patients were dependent on postive inotropic support postoperatively; their mean ID was significantly higher (2.23±3.46) than that calculated for the other 14 patients (0.7±0.7) (P< 0.001); (table 7). None of the latter (7 PVD, 3 PBVF, 2 PVT and 2 PVE) was manifested during the initial rise period nor reoperated upon on an emergency basis and all were discharged from hospital; compared to only 27%, 42% and 86% of the former; respectively. Upon hospital discharge, the mean NYHA of the 65 survivors was 1.6±0.5, with no statistically significant correlation between it and the calculated ID for a given patient.

Discussion

Prosthetic valve malfunctions, as a part of the inherent complication of cardiac valve replacement has been liberally described. Many reports have noted the time lag between valve replacement and the appearance of clinical symptoms or signs suggesting PVM. It varies from a very short period in patients who fail to improve early postoperatively (8.9) to several years after valve replacement in others (10,11).

As all cardiac prostheses carry in their vicinity the curse of malfunction, this lapse of time has been assigned as the latent period of malfunction (LPM). In this study, PVM came into 2 distinct waves:

The early wave carried 71% of malfunctions. It showed an initial rise during the first postoperative month, yielding one-third of the patients, equally presenting with disinserted, thrombosed and infected prostheses. Similar early peaking phases of PVD (4), PVT (8,9) and PVE (4,12) have been reported during the first 2-6 postoperative weeks. By the end of the first year, and as have been noted by short-term reports (13), the majority (66%) of early wave PVM had been already manifested. The mean LPM of this subgroup was 10.7±1.1 months, with no statistically significant difference among the latent periods of various types of malfunctions.

Late wave PVM, on the other hand, was of a lower pitch; taking all the fifth postoperative year for presenting one-third of its patients. It declined gradually over 10 years to end by scattered cases; the last presenting 16 years after valve replacement. The LPM (81.3+8 months) was similarly unrelated to the type of malfunction (P> 0.05).

In concordance with our work, we have observed a dormant period separating the clinical manifestation of early and late malfunctions in reports for PVD (1,14), PVT (10,15), PVE (16,17) and PBVF (18,19). In order to explain the manifestation of a given PVM at different timings; different mechanisms (11,14), predisposing factors (20), route

or type of infection (16) and host-related factors (19) were suggested for PVD, PVT, PVE and PBVF; respectively. Consequently, a difference between early and late PVM in terms of clinicl deterioration, operative risk and outcome of surgery is to be expected :

First, in the absence of infection, there was a statistically significant positive correlation between the durtions of LPM and MPM ($P< 0.001$); (table 2). Thus, the malfunction manifesting in the first 2 years had a shorter symptomtic period than when manifesting several years following valve replacement ($P< 0.05$). Although this was previously noted by Boesch and coworkers in patients with PVE (17), yet it seems that more patients were needed in our study to put into statistical evidence a difference liable to be masked by the accelerated process of infection (table 3).

Secondly, each malfunction had a MPM that is significantly different from that of other malfunctions manifesting during the same wave ($P< 0.01$).

Thirdly, an early presenting malfunction was at a higher risk of emergency reoperation ($P< 0.01$), need of postoperative inotropic support ($P=0.03$) and hospital mortality ($P=0.08$) than a late manifesting one (table 4).

Lastly, despite the absence of a significant difference in the clinical deterioration among various types of malfunctions as assessed by the NYHA functional classification; the rate at which this deterioration was taking place (ID) appeared to be different due to the previously mentioned observations. The operative risk of a class IV patient with a slowly progressive PBVF is far beyond comparison with that of a patient presenting with a "dramatic" PVT or a "fulminating" PVE and sharing the same NYHA functional class. Moreover, the quantitative evaluation of this rate can preclude the use of the previously mentioned literal descriptive terms and retain more information for statistical comparison. Unfortunately, even the new NYHA classification (21) cannot be relied upon as the sole means of evaluating patients with PVM due to the fact that it ignores the time factor; i.e. time during which deterioration occurs. The latter appears to be a determinant factor in the prognosis of these patients.

We designed the Index of Deterioration (ID) by relating the change in a patient's NYHA functional class to the time during which such deterioration occurred. The result was that every PVM, whether occurring early or late, had an ID which is significantly different from other malfunctions; PVE included ($P< 0.001$); (table 5).

ID was found to be highly influenced by the timing of PVM presentation; the mean ID of early wave patients was seven folds that calculated for late wave PVM ($P< 0.01$). Moreover, the ID of patients presenting during the initial rise was five folds that of other early wave patients ($P< 0.001$); (table 6).

The Index of Deterioration was significantly related to the necessity of reoperation, i.e. the mean ID calculated for patients re-

operated upon emergently was four folds that of electively reoperated patients (P< 0.001). Intraoperatively, the higher the patient's ID, the longer was the time necessary to wean him from cardiopulmonary bypass (P< 0.01). Accordingly, the mean ID of the 10 hospital mortalities was three folds that calculated for the 65 survivors (P< 0.01). Non- surprisingly, the mean ID of the 59 patients needing positive inotropic support postoperatively was higher than that calculated for haemodynamically stable patients (P< 0.001); (table 7). None of the latter was manifested during the period of the initial rise, reoperated upon emergently nor participated in our hospital mortality figure.

Advanced NYHA functional class (3,4,5,6), emergency (5,6), PVE (3,5) and the necessity for an additional procedure (5,6) are the most reported risk factors in reoperation upon PVM; our study pointing out the early presentation of the malfunction. Husebey and coworkers suggested that advanced NYHA functional class and emergency have to be treated separately (6). Our suggested Index of Deteriortion overcomes this separation; its numerical value augments whether by increasing its denominator i.e. advanced NYHA functional class or by decreasing its numerator i.e. deterioration in a short period of time necessitating emergency reoperation; thus incorporating both information.

Besides its usefulness in comparing groups, the ID calculated for a given patient will be of value if it helps in setting the proper timing for reoperation. In this study, an ID> 1 was significantly related to the need of an emergency procedure (P < 0.01). On the other hand, 80% of hospital mortalities had an ID > 3 compared to only 12.3% of survivors (P < 0.01). Would an ID approaching 1 be a reliable indication of reoperation before a forecoming emergency situation ? Or would an ID rising up to 3 be a justified claim for cautious management of a patient at high risk of hospital mortality?. Other studies however, are necessary to define the true figures.

REFERENCES

1. Odell JA, Duradt J, Schama DM, Vythiligum S. Spontaneous embolization of a St. Jude prosthetic mitral valve leaflet. Ann Thorac Surg 1985; 39:569-72.

2. Lindblom D, Bjork VO, Semb BKH. Mechanical failure of the Bjork-Shiley valve. Incidence, clinical presentation and management J THORAC CARDIOVASC SURG 1986; 92: 894-907.

3. Stewart S, Deweese JA. The determinants of survival following reoperation on prosthetic cardiac valves. Ann Thorac Surg 1978; 25: 555-7.

4. Blackstone EH, Kirklin JW. Death and other time-related events after valve replacement. Circulation 1985; 72: 753-67.

5. Butchart LG, Breckenridge IM. The timing of prosthetic valve reoperations based on an analysis of risk factors. Z Kardiol 1986; 75:155-9.

6. Husebey DG, Pluth JR, Pielher JM, Schaff HV, Orzulak TA, Puga FY et al. Reoperation on prosthetic heart valves: an analysis of risk factors in 552 patients. J THORAC CARDIOVASC SURG 1983; 86: 543-52.

7. Schwartz D. Les tests non parametriques. In: Schwartz D, ed. Methodes statistiques a l'usage des medecins et des biologistes. 3rd ed. Paris: Flammarion Medecine-Sciences, 1988: 245-57.

8. Davis PK, Myers JL, Pennock JL, Thiele BL. Strut fracture and disc embolization in Bjork-Shiley mitral valve prostheses: diagnosis and management Ann Thorac Surg 1985; 40: 65-8.

9. Ghosh SC, Hartstein ML, Kolker P, Thomson NB. Bjork-Shiley valve malfunction by prolapsed sutures. J Cardiovasc Surg 1986; 27:683-4.

10. Murphy DA, Levine FH, Buckley MJ, et al. Mechanicl valves. A comparative study of the Starr-Edwards and the Bjork-Shiley prostheses. J THORAC CARDIOVASC SURG 1983; 84: 746-52.

11. Silver MD, Butany J. Mechanical heart valves. Methods of examination, complications and modes of failure. Hum Pathol 1987; 18: 577-85.

12. Rutledge R, Applebaum RE, Kin JB, Engler MB, Engler MM. Actuarial analysis of the risk of undergoing repeat cardic valve replacement. Am J Surg 1984; 148: 357-61.

13. Sandza JG Jr, Clark RE, Ferguson TB, Connors JP, Weldon CS. Replacement of prosthetic heart valves. A fifteen -year experience. J THORAC CARDIOVASC SURG 1977; 74: 864-74.

14. Roberts WC, Bulkley BH, Morrow AG. Anatomie pathologique du remplacement valvulaire cardiaque. Etude necropsique de 224 sujets. Acq Nouv Pathol Cardiovasc 1973; 15: 587-639.

15. Moreno-Cabral RJ, McNanara JJ, Mamiya RT, Brainard SC, Chung GKT. Acute thrombotic obstruction with Bjork-Shiley valves. Diagnostic and surgical considerations. J THORAC CARDIOVASC SURG 1978; 75: 321-30.

16. Karchmer AW, Dismukes WE, Buckley MJ, Austen WG. Late prosthetic valve endocarditi s. Clinical features influencing therapy. Am J Med 1978; 64: 199-206.

17. Boesch C, Vernant P, Cachera JP, Block G, Loisance D, Menu P. Les remplacement valvulaires interatifs. A propos de 64 cas. Arch Mal Coeur 1987; 11: 1619-23.

18. Thandroyen FT, Whitton IN, Pirie D, Rogers MA, Mitha AS. Severe calcification of- gluteraldehyde preserved-porcine xenografts in children. Am J Cardiol 1980; 45: 690-6.

19. Magilligan DJ Jr, Lewis JW Jr, Jara FM, et al. Spontaneous degeneration of the porcine bioprosthetic valve. Ann Thorac Surg 1980; 30: 259-66.

20. Yoganathan AP, Corcoran WH, Harrison EC, Carl JR. The Bjork-Shiley prosthesis:

flow characteristics, thrombus formation and tissue overgrowth. Circulation 1978; 58: 70-6.

21. The Criteria Committee of the New York Heart Association: Nomenclature and criteria for diagnosis of diseases of the heart and great vessels. 8th ed., New York Heart Association, Inc., New York, 1979.

The results of laboratory serological screening tests in cardiac patients

Abstract

A total of 300 consecutive cardiac patients admitted at the National Heart Institute were tested serologically to evaluate the incidence of seropositive reactive and nonreactive individuals to Hepatitis HCVAb, hbsAg, HIVAb, and RPRAb among Egyptian cardiac patients.

The sera were tested by ELISA technique for the presence of anti-HCV antibody (IgG) (ABBOTT 2nd generation), ELISA technique for the detection HBsAg (ABBOTT 3rd generation), latex rapid screening test for anti-HIV type 1 antibody, rapid plasma reagin test (RPR) for screening of seropositivity to syphilis. We studied the incidence of reactivity according to the patient's sex, age and cardiac disease.

Results: There were 18.6% patients reactive to HCVAb, and 2.3% reactive to HBsAg, two of them were rective to both HCVAb and HBsAg. All patients were non-reactive to HIV antibodies and non-reactive to RPR. There was significant higher incidence in all male group than in female all group as regard reactivity to HCBAb (20.9%, 14.3%).

Conclusion: There is a non negligible prevlence of seropositivity to HCVAb and HBsAg between cardiac patients even those with no previous history of blood transfusion. It is worthy to take all the possible precautions in dealing with those patients by the medical and paramedical staff.

Azza Abdelmonem Mansy, MD., Ibrahim Abdelmeguid, MD. National Heart Institute, Imbaba, Cairo, Egypt

Introduction

All subjects that are seropositive to Hepatitis B surface antigen (HBsAg), Hepatitis V antibodies (HCVAb), Human deficiency virus antibodies (HIVAb) are potentilly infective by parentral route although they are asymptomatic. Cardiac patients undergoing open heart procedures are subjected to infection either by the surgical procedure or by blood transfusion. They are also a potential source for transmission of infection to the surgical and paramedical team.

Because HCV, HIV, HBV are parenterally transmitted viruses, these agents are of particular interest in Hematology, blood banks and renal dialysis units. Many studies were done to assess the prevalence of seropositivity in special groups of patients (chronic liver

disease, hepatocellular carcinoma, haemophilia and renal dialysis patients).

We would like to assess the prevalence of seropositivity in cardiac patients with variable diagnosis before undergoing cardiac surgery and receiving any blood transfusion to eliminate the route of viral transmission by blood transfusion or its components.

Material and Methods

Patients: Three hundred patients, 181 males nd 119 females (60.4%, 39.6%), were admitted for open heart surgery at the National Heart Institute, Imbaba, Cairo between 01/01/94 nd 31/03/94. Their mean age is 24.1±14.9 years (range from 2 to 71 years). Their diagnoses were 98 Congenital Heart Diseases, 185 Valvular Heart Diseases and 17 Ischamic Heart Diseses (32.6%, 61.6%, 5.9%).

Laboratory Investigations: Four serological tests were done for all patients before surgery to determine the incidence of: anti-HCVAb, HBsAg, anti-HIVAb type-1, and RPRAb in this potentially non-contaminated group of patients.

Venous blood sample was taken in a glass tube without anticoagulant. The serum was obtained by centrifugation and was separated as soon as possible from the clot to avoid haemolysis. The specimens were stored in aliquots immediately at-20°C for the screening of anti-HCVAb and HBsAg by ELISA technique. The sera were tested immediately for anti-HIVAb type-1, RPRAb by rapid screening tests.

All screening tests were performed according to manufacturer's instructions and duplicate repeat testing was performed on samples yielding reactive results.

The sera were tested for:

1- The presence of anti-HCV antibody IgG by (ABBOTT HCV 2nd generation EIA) using polystyrene beads coated with recombinant antigens expressed by structural (core) and non structural regions (NS-3 and NS-4) of HCV genome that increases the sensitivity (95%) and specificity (95%) of the test. We used IgG antibody for the screening of our patients and not for diagnosing them.

2- The presence of hepatitis B surface antigen (HBsAg) by (ABBOTT Ausyme monoclonal 3rd generation EIA) using polystyrene beads coated with mouse monoclonal antibody to Hepatitis surface Antigen with sensitivity ranged from 0.3±0.7 ng/ml and a specificity of 99%.

3- The detection of human antibodies to HIV-type 1 (Cambridge Biotech) by recombinant antigen latex agglutination test using colloudal suspension of latex beads coated with purified recombinant env. polypeptides in large quantities to detect antibodies to HIV-1 with high sensitivity (99.4%) and specificity (99.5%).

4- The detection of Syphilis reagin antibodies in the serum by a nontreponemal

Rapid Plasma Reagin Flocculation test Immutrep RPR (Omega diagnostics) which contains carbon particles to improve the visual reading of the results. (Immutrep-RPR Omega diagnostics.

The results

We studied the results according to the age, sex and diseases' distributions.

Reactivity to different serological tests:

All patients were non-reactive to HIV antibodies tested by recombinant antigen latex agglutination test and non-reactive to RPR tested by carbon particles flocculation test.

- Two patients were reactive to both HCVAb and HBsAg, one male 12 years (MVP) and the other a female 40 (MVP), both were of the rheumatic patient of mitral valve disease category.

Table 1: The age group distribution of patients and the incidence of the reactivity to anti-HCVAb.

HCVAb	<10	10<20	20<30	30<40	40<50	50<60	>60
Total (m, f)	60	78 (51, 25)	60 (38, 22)	44 (24, 40)	41 (23, 18)	8 (7,1)	4 (4,0)
Reactive	3 (1, 2)	9 (6, 2) +1	7 (5, 2)	16 (11, 4) +1	18 (11, 7)	3 (3,0)	0 (0,0)
Nonreactive	57	67	53	28	23	5	4
Percent	5%	12.8%	11.6%	38.6%	38.9%	37.3%	0%

Chart 1: HCVAb age disturbution

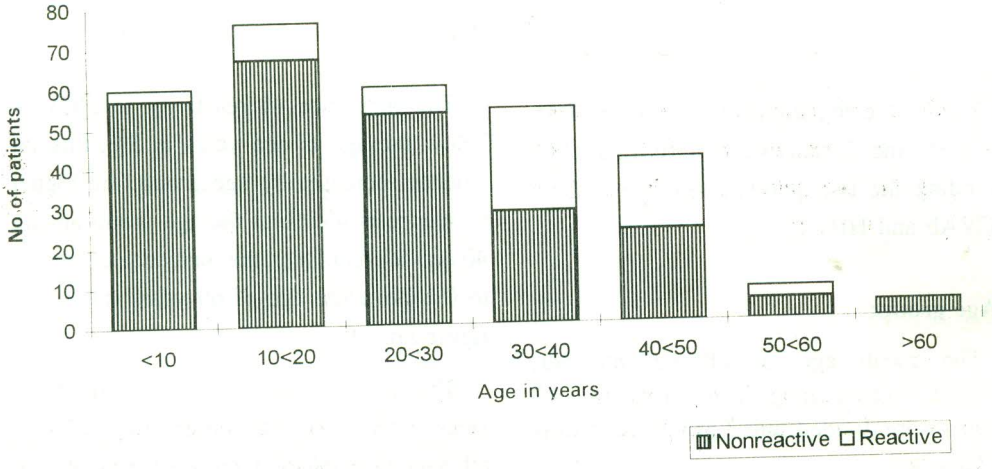
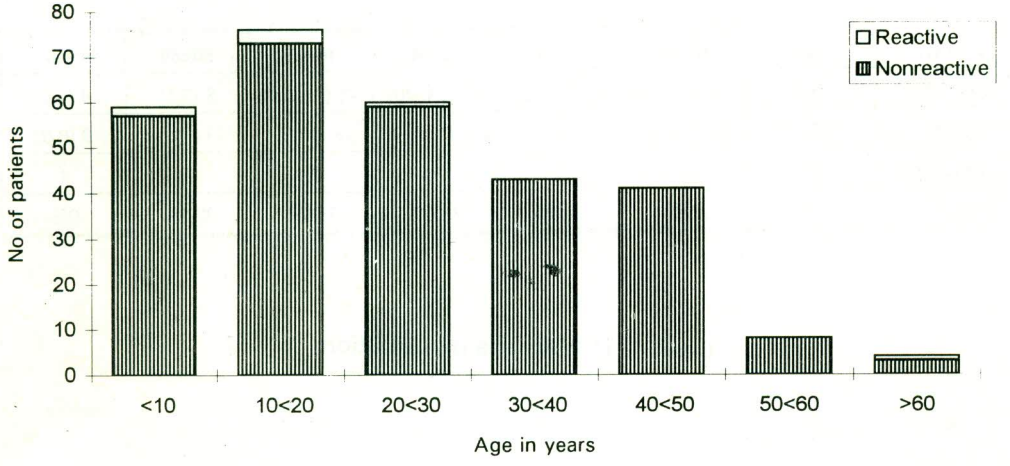


Table 2: The age group distribution of patients and the incidence of the reactivity to HBsAg.

HCVAb	<10	10<20	20<30	30<40	40<50	50<60	>60
Total (m, f)	60	78 (51, 25)	60 (38, 22)	44 (24, 40)	41 (23, 18)	8 (7,1)	4 (4,0)
Reactive	2 (1, 1)	3 (1, 1) +1	1 (0, 1)	1 (0, 0) +1	0 (0,0)	0 (0,0)	1 (1,0)
Nonreactive	57	73	59	43	41	8	3

Chart 2: HBsAg Age disturbution



There were 56 patients reactive to HCVAb (18.6%), and 7 reactive to HBsAg (2.3%) including the two patients reactive to both HCVAb and HBsAg.

- Age group:

The mean age of all patients was 24.1+15.9 years (range 2 to 71 years). All ptients were divided into 7 groups according to their age.

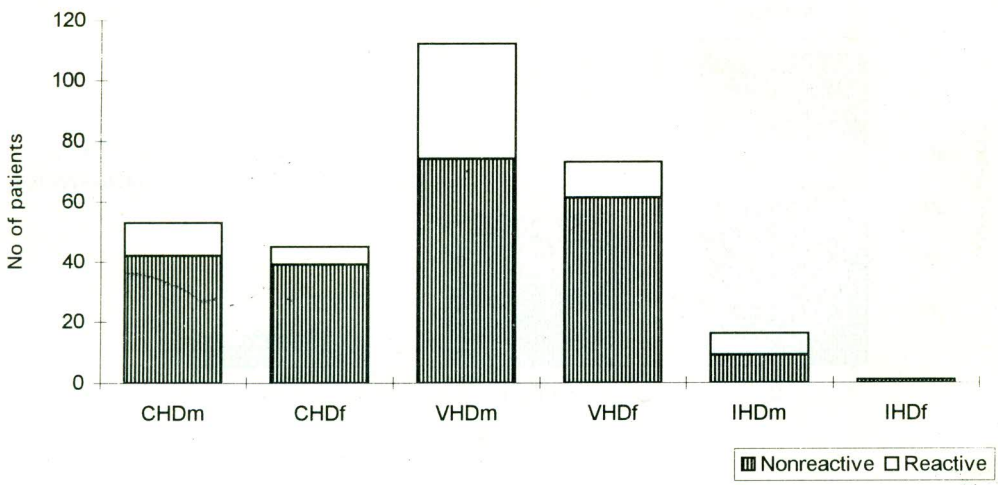
The incidence of reactivity to HCVAb below the age of ten years is 5%, this incidence increases by age group, the higher incidence is 43.9% at the age group among 40 and 50 years then the incidence decreases to 0% bove the age of 60 years (table and figure no. 1).

The incidence of reactivity to HBsAg is weak; only 7 patients representing 2.7% of all patients most of them are below (6 pa-

Table 3: Analysis of all males and all females groups according to reactivity and cardiac diseases.

HCBAb Reactive	Males		Females	
	total	reactive	total	reactive
CHD 11	53	5 (9.4%)	45	6 (13.3%)
VHD 38	112	26 (23.2%)	73	12 (16.4%)
IHD 7	16	7 (43.7%)	1	0 (0%)
Total 56	181 (60.4%)	38 (20.9%)	119 (39.6%)	18 (14.3%)

Chart 3: HCVAb sex disturbution



tients) the age of 30 years (table and figure no.2).

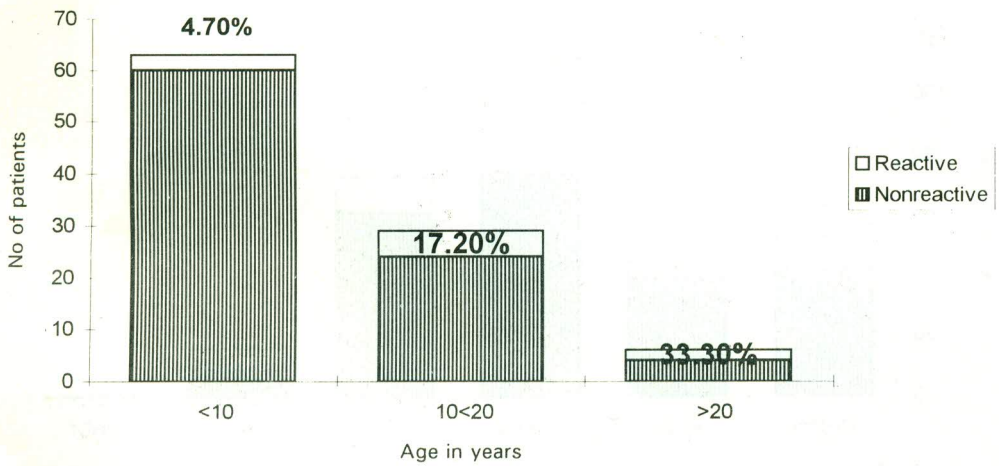
Sex groups:

Studying the whole group by sex revealed that 38 mles and 18 females were reactive to HCVAb (20.9%, 14.3%). This significant higher incidence in all male group is inverted if we consider subgroups by disease diagnosis, as HCV reactivity is significant higher in females with CHD than in males of the same group on the other hand this percentage is higher in males with VHD group than in females. The IHD group is not well distributed by sex (16 males to one female) to study the influence of sex on the incidence of reactivity to HCVAb (table and figure no.3).

Table 4: The age group distribution and the incidence of both HCVAb and HBsAg in group 1 CHD.

Age group	Number (m, f)	HCV antibody	HBsAg
<10	63 (32, 31)	3 (4.7%)	2 (%)
10<20	29 (17, 12)	5 (17.2%)	1 (%)
>20	6 (4, 2)	2 (33.3%)	0 (0%)
mean 10.5+7.8 (2-45)	98 (53, 45)	10 (9.9%)	3 (2.9%)

Chart 4: HCV in CHD



Diseases

Patients were classified in three groups according to their cardiac disease, group 1 (CHD): 98 patients with Congenital Heart Diseases (32.6%), group 2 (VHD): 185 patients with Valvular Heart Diseases (62.6%) and group 3 (IHD): 17 patients with Ischamic Heart diseases (5.6%).

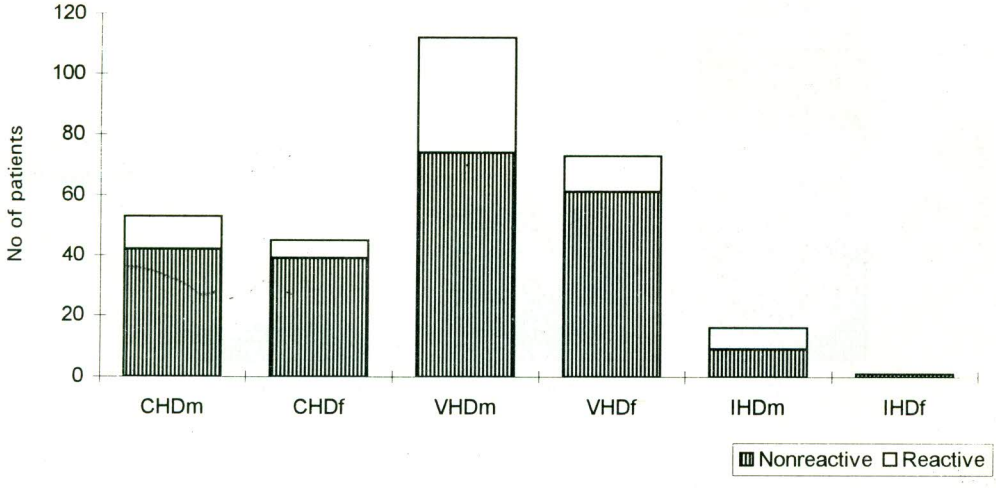
Group 1: Congenital Heart Disease

This group includes 98 patients admitted for total correction of congenital heart diseases, they were 53 males and 45 females (54.1, 45.9%) and most of them were children with mean age 10.5±7.8 (range 2 to 45 years). There were 10 reactive patients to HCV antibody (9.9%) and 3 reactive to HBsAg (2.9%). The age group distribution shows an increase

Table 3: Analysis of all males and all females groups according to reactivity and cardiac diseases.

HCBAb Reactive	Males		Females	
	total	reactive	total	reactive
CHD 11	53	5 (9.4%)	45	6 (13.3%)
VHD 38	112	26 (23.2%)	73	12 (16.4%)
IHD 7	16	7 (43.7%)	1	0 (0%)
Total 56	181 (60.4%)	38 (20.9%)	119 (39.6%)	18 (14.3%)

Chart 3: HCVAb sex disturbution



tients) the age of 30 years (table and figure no.2).

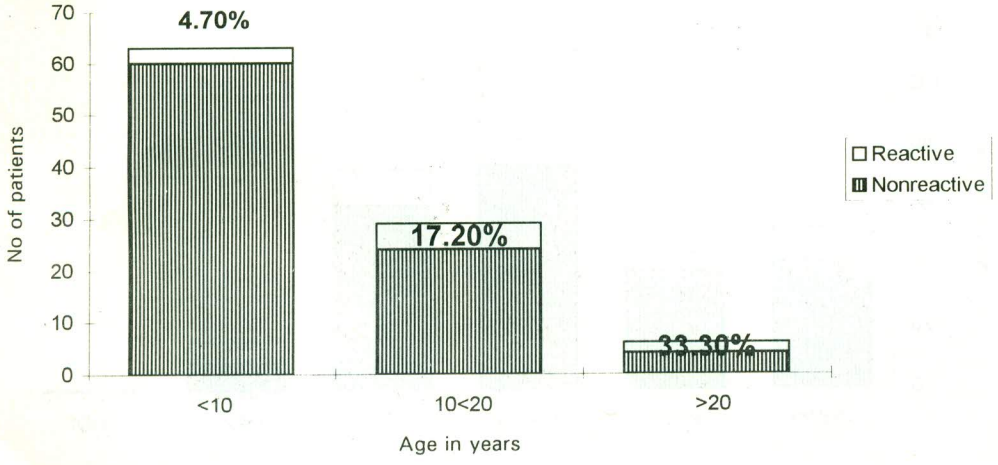
Sex groups:

Studying the whole group by sex revealed that 38 mles and 18 females were reactive to HCVAb (20.9%, 14.3%). This significant higher incidence in all male group is inverted if we consider subgroups by disease diagnosis, as HCV reactivity is significant higher in females with CHD than in males of the same group on the other hand this percentage is higher in males with VHD group than in females. The IHD group is not well distributed by sex (16 males to one female) to study the influence of sex on the incidence of reactivity to HCVAb (table and figure no.3).

Table 4: The age group distribution and the incidence of both HCVAb and HBsAg in group 1 CHD.

Age group	Number (m, f)	HCV antibody	HBsAg
<10	63 (32, 31)	3 (4.7%)	2 (%)
10<20	29 (17, 12)	5 (17.2%)	1 (%)
>20	6 (4, 2)	2 (33.3%)	0 (0%)
mean 10.5+7.8 (2-45)	98 (53, 45)	10 (9.9%)	3 (2.9%)

Chart 4: HCV in CHD



Diseases

Patients were classified in three groups according to their cardiac disease, group 1 (CHD): 98 patients with Congenital Heart Diseases (32.6%), group 2 (VHD): 185 patients with Valvular Heart Diseases (62.6%) and group 3 (IHD): 17 patients with Ischamic Heart diseases (5.6%).

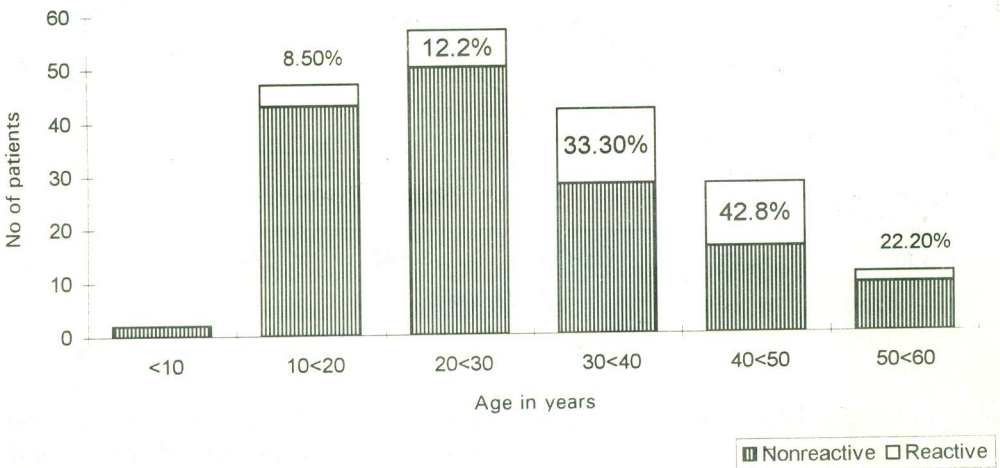
Group 1: Congenital Heart Disease

This group includes 98 patients admitted for total correction of congenital heart diseases, they were 53 males and 45 females (54.1, 45.9%) and most of them were children with mean age 10.5±7.8 (range 2 to 45 years). There were 10 reactive patients to HCV antibody (9.9%) and 3 reactive to HBsAg (2.9%). The age group distribution shows an increase

Table 5: The age group distribution and the incidence of both HCV antibody and HBsAg in group 2 VHD.

AGE	AVD	MVD+MVP	DVD	Total (m, f)	HCV	HBs
<10	0	2	0	2 (1, 1)	0	0
10<20	11	29	7	47 (34, 13)	4 (8.5%)	2 (4.5%)
20<30	14	28	15	57 (36, 21)	7 (12.2%)	1 (1.7%)
30<40	4	31	7	42 (22, 20)	14 (33.3%)	1 (2.38%)
40<50	2	23	3	28 (12, 16)	12 (42.8%)	0
50<60	2	4	3	9 (7, 2)	2 (22.2%)	1 (11.1%)
Total				185 (112, 73)	39 (21%)	5 (2.7%)

Chart 5: HCV in VHD



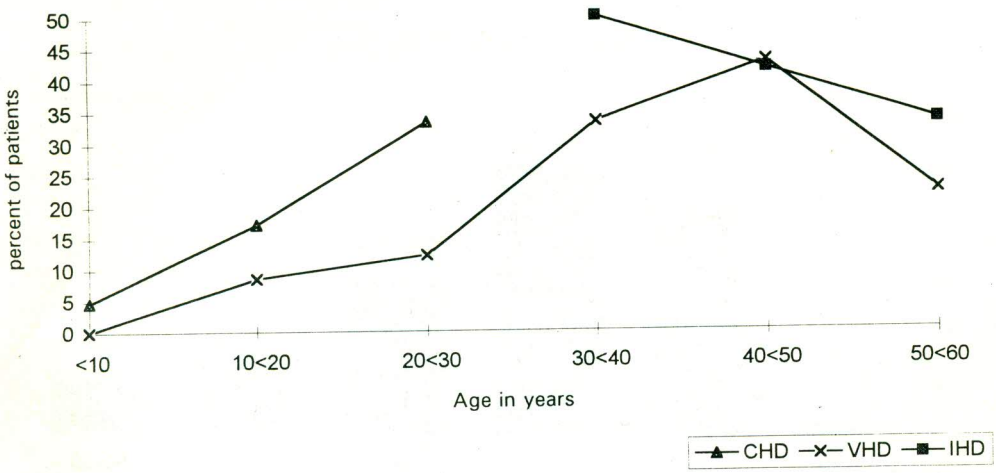
in reactivity to HCVAb with increasing age, however the age group >20 years has a little number of patients (table and figure no.4).

The ten reactive patients to HCV antibody were 3 males and 7 females representing 5.6% and 15.5% for sex distribution in this group. Although there is no significant difference in the number of males and females in this group, there is unexplained significant difference in the number of reactivity as more females are reactive to HCV. Dividing patients into two sub-groups of cyanotic and non-cyanotic does not show any difference neither by sex. There are three patients re-

Table 6: The age distribution and the incidence of HCVAb in group 3 IHD.

Age group	Total number	Reactive	non-reactive
<40	2	1 (50.0%)	1
40<50	12	5 (41.6%)	7
50<60	3	1 (33.3%)	2

Chart 6: Percent of HCVAb reactivity by Disease group



active to HBsAg, two of them were below age of ten.

Group 2: Valvular Heart Disease

This group includes 185 patients admitted for valvular replacement or repair for acquired valvular heart diseases, they are 112 male and 73 females (60.5, 39.5%) and their mean age is 29.2±12.3 (range 6 to 71). There was 39 reactive patients to HCV antibody (21%) and 5 reactive to HBsAg (2.7%). This group ws studied further by their age group and which valve was operated, mitral, aortic or both.

Age correlation

For HCVAb: Patients are divided in 6 groups of age, this shows significant rise in the percentage of reactivity with maximum among 40 and 50 years. Dividing this group of valvular heart disease into two groups of age bellow 30 and above 30 years will show

significant reactivity after age of 30 years (11 patients out of 59 and 28 out of 79 representing 10.3% and 35.4%).

For HBsAg: the total number of reactive patient is small for dividing them into small age groups, if divided into two age groups below and over 30 yers, we will find 3 patients out of 59 and 2 out of 79 patients representing 5% and 2.5, there is no significant difference in the seropositivity to HBsAg below and above 30 years (table and figure no.5).

Group 3: Ischaemic Heart disease

This group includes 17 patients admitted to complete revascularisation for Ischemic heart diseases, they are 16 males and 1 female and there mean age was 46.1±7.9 (range 25 to 60). There were 7 reactive patients to HCVAb (41.1%) and all were non-reactive to HBsAg (100%). Dividing patients into three age groups shows highest incidence among 30 and 40 years with significant decrease in the percentage in increasing of age. This feature is corresponding with the same finding in the group 2 of VHD (table and figure no.6).

The different pattern of reactivity to HCVAb in this group may be due to their small number in relation to group I and group II. A further study is recommended on a larger number of IHD patients.

Finally, figure no.6 resumes the pattern of reactivity to HCVAb in the different disease groups.

Discussion

Many studies were done to assess the prevalence of parenteral transmitted viruses in blood donors and in certain high risk population like hemophilic, cirrhotic or primary liver carcinorma patients.

In our study, we choose three screening tests to study the prevalence of three important parenterally transmitted viruses in cardiac patients admitted for cardiac surgery in the preooperative period and before receiving any blood transfusion.

The seropositivity to HIV ab is 0% in our patient population as they are not from the known high risk population for HIVAb seropositivity e.g intravenous drug abusers, haemophilics or homosexuals.

The seropositivity to HBsAg of the whole number of patients screened without any classification is 2.3%. The seropositivity to HBsAg on classifying the ptients according to their cardiac disease is: 2.9% in group I (CHD) below 10 years old, 2.7% in group II (VHD) below 30 yers old, 2.5% in group II (VHD) above 30 years old, 0% in group III (IHD).

This demonstrates that the incidence of seropositivity to HBsAg is rather homogenous in our patient population regardles to their age group or their cardiac disease.

The seropositivity to HCVAb IgG on grouping the patients with the age or with the cardiac disease is around 10% below 30 years old, then it tends to rise after the age of 30 years with a maximum among 30 and 40 years old where it reaches 40%.

The seropositivity of these patients to either HBsAg or HCVAb may be due to syringe transmission and intramuscular injection without adequate sterilization (Dull 1961).

The seropositive subjects to HBsAg are potentially infective.

The seropositive subjects to HCVAb should be handled with precaution by the medical and paramedicl staff as there is a potential risk of infection.

Conclusion

Our cardiac patients were seronegative to both HIVAb and RPRAb. The seropositivity to HBsAg is 2.3% and to HCVAb is 18.6%. It increases with age with the highest percentage among 40 and 50 years old.

The seropositive cardiac patients must be handled with precaution by the medical and paramedical staff.

More specific tests like polymerised chain reaction (PCR) or recombinant immunobinding assay (RISA), are needed to confirm the screening tests, they are not available in our blood bank as it costs so much for screening tests.

The seropositive patients hve to be followed up by other laboratory investigations, liver function tests, HCVAb, HB markers, and PCR to assess the progress of their medical situation by the time.

REFERENCES

1. Brettler DB, Alter HJ, Dienstag JL et al1990. Prevalence of hepatitis C virus antibody in haemophilia patients. Blood 76: 254-256.
2. Choo QL, Weiner AJ, Overby LR et al 1990. Hepatits C virus: the major causative agent of viral non A, non B hepatitis. Br. Med. Bull 46: 423-441.
3. Coursaget P, Bourdil C, Kastally R et al 1990. Prevalence of heptitis C virus infection in Africa: anti-HCV antibodies in the general population and in patients suffering from cirrhosis or primary liver cancer. Res. Virol. 141: 449-454.
4. Dawson GJ, Lesniewski R.R, stewart JL et al 1991. Detection of antibodies to hepatitis C virus in US blood donors. J Clin. Microbiol. 29: 551-556.
5. Dull HB 1961. Syringe transmitted hepatitis: a recent epidemic in history perspective. Jama: 176: 413-418.
6. Ellis LA, Brown D, Conradie JD et al 1990. Prevalence of hepatitis C in South Africa: detection of anti-HCV in recent and stored serum. J. Med. Virol. 32: 249-251.
7. Johnny D.Cllahan, Neil TConstantine, Peter Kataaha, Xiang Zhang, Kenneth C, Hyaams and Jaya Bansal 1993. Second generation Hepatitis virus Assays: Performance when testing African sera. J. Med. Virol. 41: 35-38.
8. Mc Hutchison JG, Person JL, Govindarajn S, et al 1992. Improved detection of he-

patitis C virus antibodies in high rpopulations. Hepatology 15: 19-25.

9. Rodriguez M, Tevar F 1991. Second generation tests for hepatitis C virus. Intern. Med. 115: 747.

10. Stevens CE, Taylor PE, Pindyek J, Chooa. L, Bradley DW, Kuo G, Houghton M 1990. Epidemiology of hepatitis C virus: a preliminary study in volunteer blood donors. Jama 263: 40-53.

11. T. Jake Ling, Henry C, Bodenheimer Jr, Rolnd Yankee, Nancy V. Brown, Kenneth Chang, Jiakang Huang and Jack R. Wands 1994. Presence of hepatitis B and C viral genomes in US blood donors as detected bt polymerase chain amplification. J. Med. Virol. 42: 151-157.

Preoperative oropharyngeal sterilization for patients with rheumatic valvular heart disese

Abstract

Chronic oropharyngeal infection is probably one of the most common sources of bacteraemia causing infective endocarditis in patients undergoing surgical treatment for their rheumatic valvular lesion. Preoperative oropharyngeal sterilization aims to avoid this serious complication and also to shorten the pre-operative preparing period. 150 patients with rheumatic heart disease were chosen, they were free from any systemic infection.

Each case was subjected to acute phase reactants estimation (ESR, CRP) and ASOT as a serological marker of oropharyngeal infection, before and after treatment. They were arranged into 3 groups according to the method of oropharyngeal sterilization by local antiseptic (60 patients), systemic antibiotic (40 patients), combined local antiseptic and systemic antibiotic (50 patients). Each group was further subdivided into subgroups according to the estimated acute phase reactants values into: moderate and severe cases. The proper antibiotic was selected after throat swab culture and sensitivity and the treatment was continued for one week with each method. We concluded that using the local antiseptic or the combined therapy was highly effective in improving phase reactants levels and ASOT in moderate cases than using systemic antibiotic. For severe cases the combined therapy was more efficient than either local antiseptic or systemic antibiotic therapy.

Abd El Moneim M. Mashaal (M.D.) Wafaa Hussien M. Mahmoud (Ph. D) Mona A. El Atreby (Ph. D)

National Heart Institute-Imbaba-Giza. Egypt

Introduction

It was observed in some patients with rheumatic valvular heart disease admitted for surgery in National Heart Institute, that there was a persistent elevation of acute phase reactants, "Erythrocytic sedimentation rate ESR, C-reactive protein CRP and Antistroptolysin "O" titre ASOT

These findings lead to postponing their surgical interference. Most of these patients

received broad spectrum antibiotics randomly with no significant improvement.

Aim of the Work

Since Chronic oropharyngeal infection is one of the common causes of elevating ESR, ASOT and the positivety of CRP, hence the aim of this work is to evaluate the role of oropharyngeal sterilization on patients with rheumatic valvular disease in shortening the preoperative preparation period, minimizing the use of less effective and expensive antibiotic, avoiding postoperative infective endocarditis and establishing a good postoperative course.

Material

The study included 150 patients with rheumatic valvular heart disease, 86 were males and 64 were females. Their ages ranged from 15 to 38 years with a mean of 23 years . Their clinical diagnosis included: isolated mitral stenosis (25 patients), mitral regurge (15 patients), double mitral valve disease (55 patients), isolated aortic valve regurge (19 patients), double aortic valve disease (26 patients), and mixed mitral and aortic valvular lesions (10 patients). Their functional classes were II (25 patients) and III (125 patients) according to NYHA calssification.

All patients were inpatient at the National Heart Institute, and the following criteria were fullfilled before including any subject in the study:-

- Elevation of ESR and either ASOT or positive CRP or both, and their (throat) swab culture revealed pathognic orgainsms.

- The patients were checked to be free from any systemic infection; in Chest, Ear and Nose, Urinary tract, Gastro - intestinal tract or genital system thyrotoxicosis, diabetes mellitus, liver dysfunction pericardial effusion, severe anaemia and no recent antibiotic administration including long acting penicillin G (LAP). Samples were taken just before administration of LAP doses and one week after menstruation in females.

All Patients had the following labortory investigations: ESR, ASOT, CRP and throat swab culture and sensitivity before treatment and repeated after one week treatment.

Anti heart failure treatment was undertaken for patients in functional class III one week before the study.

Patients were classified into three groups, Group I.: Included 60 patients, subjected to oropharyngeal sterilization by the local antiseptic, benzoxonium chloride lotion and lozenge only.

Group II.: included 40 patients subjected to oropharyngeal sterilization by systemic antibiotic according to culture and sensitivity test.

Group III.: included 50 patients in whom oropharyngeal sterlization was established by combined therapy.

Methods

1- Oral antiseptic : "Local sterilization"

The antiseptic "Benzoxonium chloride lotion and the lozenge forms were used as follows.

- One tea spoonfull for 60 - 90 seconds tds after meals and 2 tablets of the lozenge form t.d.s. between meals, treatment continued for one week.

2- Systemic antibiotic:

The sensitivity to various antibiotics was tested and the proper antibiotic was prescribed for one week.

3- Combined therpy:

Using local oropharyangeal sterilization in addition to systemic antibitic administration according to culture and sensitivity tests for one week.

Results

In our study 150 cardiac patients were examined for persistent elevation of ESR value and / either ASOT or CRP or both, they were divided into 3 groups:

Group I. 60 patients subjected to oral antiseptic treatment.

Group II. 40 patients had systemic antibiotic treatment.

Groups III. 50 patients subjected to combination of oral antiseptic and systemic antibiotic.

Treatment was continued for one week and re-estimtion of the acute phase reactancts "ESR, CRP: and ASOT plus throat swab culture and sensitivity before and after treatment.

We subdevided each group into moderate and severe cases according to the magnitude of elevation of the ESR, ASOT and CRP. Moderate cases had ESR in 1st hour 20-50mm ASOT 300-500iu/ml CRP + ve. Severe cases had ESR in 1st hour > 50 mm., ASOT> 500i.u/ml, CRP >+ ve.

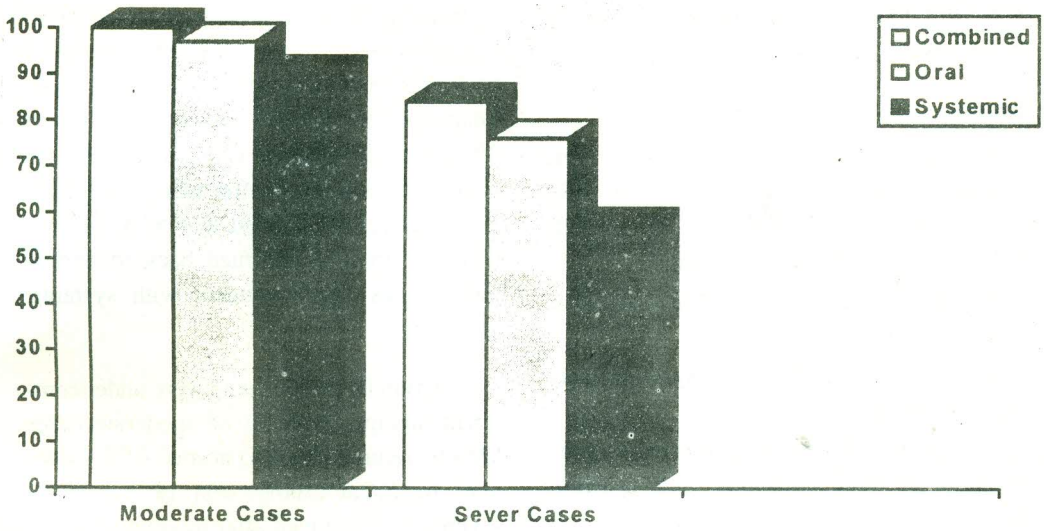
Our results revealed that in group 1 : (table 1 & Diagram 1 for ESR changes). The moderate cases were 34 patients, 33 of them reverted back to normal after oral antiseptic therapy for one week (97%), also 13 patients out of 26 severe cases returned back to normal levels after one week treatment (50%). Collectively 67.7% of patients of group 1 returned back to normal level ESR after oral antiseptic therapy.

In group II (table 1) we had 18 out of 20 moderate cases, whose ESR values returned back to normal level after systemic antibiotic treatment (90%) and we have only 5 out of 20 severe cases whose ESR values returned back to normal levels after treatment (25%). The final result revealed that 57.5% of patients of group II returned back to normal ESR values after treatment with systemic antibiotic.

In group III patients who were under combined therapy (table 1) all moderate cases (23 pts) returned back to normal ESR values after treatment (100%) and 19 out of 27 severe cases resolved after treatment (70%). Totally 84% of patients of group III returned to normal ESR values after combination of oral antiseptic and systemic antibiotic treatment.

TABLE 1 : ***Improvement in ESR After treatment with ORAL (GR I) systemic (GR II) and combined methods of treatment (GR III)***

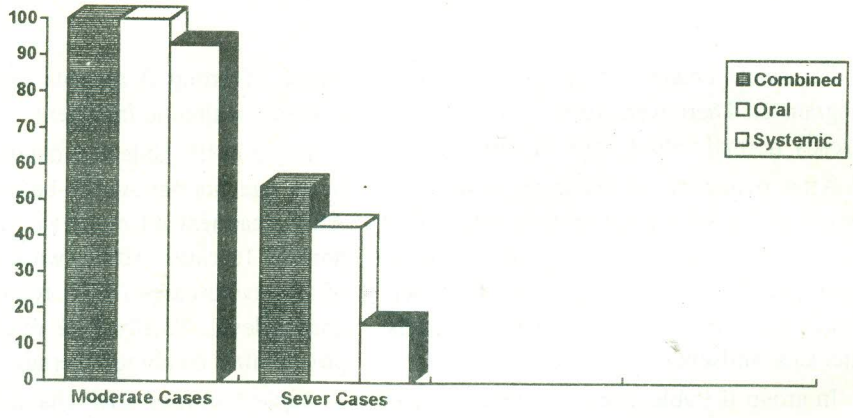
	Group I			Group II			Group III		
	No. of PTS.	No of Improv. Cases	% of Improv.	No. of PTS.	No of Improv. Cases	% of Improv.	No. of PTS.	No of Improv. Cases	% of Improv.
moderate cases 20 - 50 mm	34	33	97%	20	18	90%	23	23	100%
Sever cases > 50 mm	26	13	50%	20	5	25%	27	19	70%
Total	60	46	76%	40	23	57%	50	42	84%



Improvement in ESR after treatment with ORAL, SYSTEMIC and COMBINED methods of treatment

TABLE 2. ***IMPROVEMENT IN ASOT AFTER TREATMENT WITH ORAL (GR I) SYSTEMIC (GR II) AND COMBINED (GR III) METHODS OF TREATMENT***

	Group I			Group II			Group III		
	No. of PTS.	No of Improv. Cases	% of Improv.	No. of PTS.	No of Improv. Cases	% of Improv.	No. of PTS.	No of Improv. Cases	% of Improv.
NORMAL LEVEL < 300 I.U . ML	10	-----	-----	11	-----	-----	11	------	-----
MODERATE CASES 300-500 I.U . ML	29	29	100%	13	9	69%	16	16	100%
SEVERE CASES > 500 I.U . ML	21	9	43 %	16	4	25%	23	16	70%
TOTAL ABNORMAL CASES	50	38	76%	29	13	45%	39	32	82%



Improvement in CRP after treatment with ORAL, SYSTEMIC and COMBINED methods of treatment

TABLE 3 : ***Improvement in CRP After treatment with ORAL (GROUP I) systemic (GR II) combined (GR III) methods of treatment***

	Group I			Group II			Group III		
	No. of PTS.	No of Improv. Cases	% of Improv.	No. of PTS.	No of Improv. Cases	% of Improv.	No. of PTS.	No of Improv. Cases	% of Improv.
Normal - ve	10	---	----	5	---	----	6	---	----
Moderate cases + ve	27	27	100 %	16	15	93 %	18	18	100%
Severecases =/> ++ ve	23	10	43 %	19	3	16 %	26	14	54 %
TOTAL ABNORMAL CASES	50	37	74 %	35	18	51 %	44	32	73 %

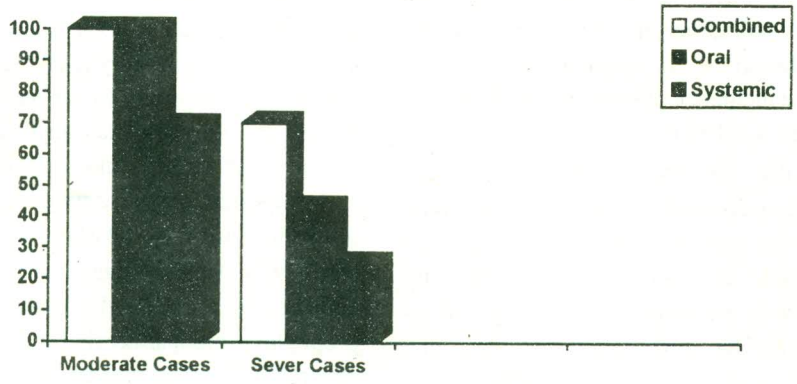
For ASOT changes in group I (table 2 & diagram 2). There were 10 patients who had already normal values before treatment.

After treatment, all moderate cases (29) returned to normal level (100%) while only 9 out of 21 severe cases changed to normal levels (43.1%). Totally 76% of patients group I had improvement of ASOT to normal levels after oral antiseptic treatment.

In group II (table 2 & diagram 2): 11 patients had normal level before treatment. After treatment 9 out of 13 moderate cases had normal ASOT levels (69%) but only 4 out of 16 severe cases became normal after treatment (25%). As regard ASOT changes, 44.8% of group II patients improved after systemic antibiotic treatment.

In group III (table 2 & diagram 2) 11 patients had normal ASOT before treatment. After treatment all moderate cases became normal, 16 cases (100%) while only 16 out of 23 severe cases (70%) returned back to normal level. Totally 82% of all cases improved after combined therapy.

Table 3 showed CRP changes in patients of group I. Ten patients were normal before treatment. All moderate cases (27) were negative CRP after oral antiseptic (100%) while only 10 out of 23 sever cases (43%) became normal. Collectively 74% of total positive



Improvement in ASOT after treatment with ORAL, SYSTEMIC and COMBINED methods of treatment

CRP patients in group I improved after oral antiseptic treatment.

In group II (table 3 & diagram 3) 5 patients had negative CRP before treatment. 15 out of 16 moderate cases became negative after systemic antibiotic treatment (93%). On the other hand 3 out of 19 highly positive cases returned back to normal treatment (16%). Totally 51% of all patients in group II had normal CRP values after treatment.

Finally in group III (table 3 diagram 3) 6 patients had normal CRP value befor treatment. All moderate patients (18) became negative after comined therapy (100%), while 14 out of 26 severe cases (54%) became normal after treatment. As regard CRP positivity of all patients in group III 73% of them became CRP negative after combined therapy.

Discussion

The main aim of preoperative oropharynaeal sterilization is to guard against infective endocarditis (I.E) which is not only responsible for intensification of the rheumatic valvular heart disease but also it causes primary valve lesion (Karchmer, 1988).

In addition it causes early prosthetic valve endocarditis "PVE" (Cowgi et al 1986). The prosthetic valve is more susceptible to colonization by micro-organisms immediatetely after insertion and probably permanently. Most infections which lead to PVE are by bacteria arising from oropharynx, in addition to other sites. The causative organisms usually gain access to blood stream followed by bacteraemia (Everett and Hirchmann,

1977). The risk of early low grade bacteraemia is apparenty higher following surgical procedures (Guntheroth, 1984). Therfore oral health should be maintained, this will decrease the incidence of bacteraemia (Pallasch, 1989. Fekete, 1990).

Recently the committee on rheumatic fever and endocarditis of the American heart Association had recommended strongly the prophylactic prevention of I.E not only for patients with rheumatic heart disease and acquired valvular dysfunction but also after valvular surgery (Dajani et al 1990).

The prohylaxis for I.E was mainly before tooth extraction by administrating broad spectrun antibiotic randomly (Kaye, 1976).

The present study investigated all the available methods of orophayngeal sterilization as a preoperative procedure for patients with chronic rheumatic heart disease CRHD aiming to decrease the preoperative preparation period, and to evaluate the degree of sterilization gained by each method. For these purposes we selected 150 patients with CRHD admitted in National Heart Institute for sugical interference, nearly of the same age group and have functional classses II and III. according to NYH classification (NYH, 1964). We arranged them into 3 main groups. Group I was subjected to oral antiseptic therapy, group II was on systemic antibiotic treatment, and group III was subjected to combined oral antiseptic and systemic antibiotic. We utilized erythrocytic sedimentation rate (ESR) antistroptolysin "O" titre (ASOT) and C-reactive protein (CRP) for assessing the severity of oropharyngeal infection. (Fischbach, 1992, Franke, et al. 1993). Also we used throat swab for culture as a direct visualization of oropharyngeal organisms and testing their senstivity to various antibiotics, before and after oropharyngeal sterilization. Throat swab culture and sensitivity proves to be important in overcoming oropharyngeal infection caused by resistant strain (Karchmer, 1985). Another advantage of throat swab cullture is to prescribe the antibiotic used for prophylaxis preoparatively for therapy of endocarditis if it occurred postoperatively.

In Group I patients: Where throat sterilization established by the local antiseptic Benzoxonium chloride, we found that this method is strongly efficient in maintaining significant oro-pharyngeal sterilization and curing chronic orpharyngeal infection mainly in patients with moderate severity as suggested from the marked reduction in ESR,ASO titre and normaliztion of CRP. Also an improvment but to less extent was observed in severe cases. The efficiency of local antiseptic most probably is due to its direct strong bactericidal effect on the pathogenic oropharyngel organisms (Weibel et al 1987).

In agreement with our finding about the importance of local oral antiseptic is the finding of many workers that "degerming the mouth with antispetic mouth washer can markedly decrease the intensity of subequent bacteria and hence the risk of endocarditis (Bender et al. 1984, Pallasch, 1989).

In group II patients in which we used systemic antibiotic according to throat swab culture and sensitivity tests there was marked decrease in ESR, ASO titre and improvement in CRP., especially in moderate cases but to a less extent in severe cases (tables 1, 1,3).

There is controversy about the use of preoperative antibiotic prophylaxis, while some investigators prove its benefits (Goodman et al 1968) others recorded the occurance of endocarditis despite the use of preopeative antibiotics (Durack, et al 1983). We think that this could be due to the use of antibiotics without proper culture and sensitivity.

In group III patients whom sterilization was established by combined local antisptic and systemic antibiotic we found that oropharyngeal sterilization was more efficient than in the other two groups. This effect was observed not only for moderate cases but also for severe cases as well. The combined method has many advntages. In addition to its efficacy we can use one type of antibiotic with less liability of emergence of resistant strains or superinfection by fungi (Rubinstein, et al 1975) or other opportunistic organisms.

We selected patients at middle age who are more suscibtible to endocarditis (Weinberger et al 1990). We found no relation between severity of infection indicated by ESR, ASOT titre, CRP levels and patients functional class as others found (Rutledge et al 1985). One of the most significant results of this work is the finding that local oral antiseptic, which is cheap and safe is efficient for cases of chronic oropharyngeal infection with moderate elevation of acute phase reactants and ASOT while combined local oral antiseptic and selective systemic antibiotic should be reserved for management of cases with high levels of acute phase reactants and ASOT.

Following this program: no complication has been absereved, patient compliance was good and the preoperative preparation period was reduced greatly due to establishment of sufficient oropharyngeal sterilization following one week treatment especially by the local or the combined method.

Also no cases of infective endocarditis were recorded immediately or two months post opertively which reflect the efficacy of this program.

Summary and Conclusion

150 patients with chronic rheumatic valvular heart didease and chronic oropharyngeal infection were chosen randomly as regard their valvular lesion and having functional classes II and III (HYHA classification). All patients studied had persistent elevation of ESR, and either high ASO titre or positive CRP or both which postponed their surgical interference, and their throat swab culture revealed pathognic organism. Patients were free from any systemic infection acute orophryrgeal infection or acute rheumtic fever. Patients who were under penicillin G prophylaxix, their samples were taken just before the monthly dose administration. The patients were classified into 3 groups

according to the type of treatment: Group I (oral antiseptic) Group II (systemic antibiotic) group III (combined therapy). Each group was further subdevided into subgroups (moderate and sever cases) according to the estimated leveles of ESR, ASO titre and CRP. Our findings revealed that the oral antiseptic treatment is suitable for cases of chronic oropharyngeal infection with moderate elevation of acute phase reactants "ESR, CRP" and ASOT. Combined theapy should be reserved for severe cases with high levels of ESR, CRP and ASOT. Antibiotic therapy is not recommended without culture and sensitivity tests. Following this preoperative preparation progrm the preoprative period was markedly reduced.

REFERENCES

1. Bender, I.B Naidorf I.J. and Garvey, G.J. bacterial indocarditis; A Consideration for physician and dendist. J. am dent. assoc. 1984, 10:415.

2. Cowgill, L.D.,Addonizio, V.P. Hopeman A.R., and Harken, A.H., prsthetic valve endocaditis curr, probl. Cardiol; 1986; 11: 617.

3. Dajani, A.S., Bisno, A.L., Chung, K.J., et. al., Prevention of bacterial endocarditis jama 1990; 264:2919.

4. Durack, D.T, Bisno, A.L. and kaplan. E.L.; Apparent failure of endocarditis prophylaxis, analysis of 52 cases submitted to a national registry jama 1983; 250: 2318.

5. Everett, E.D., and Hirschmann, J.V. Transient bacteraemia and endocarditis prophylxis, a review medicine (Baltimore) 1977; 56:61.

6. Fekete T.: Controversies in the prevention of infective endocarditis related to dental procedures. Dent clin. North AM 1990; 34 (1): 79.

7. Franke, E, Lucente and Steven, M, Soboc: Essential of otolaryngeology 3rd Ed Raven press 1993; P. 247.

8. Fischbach, F.: In Amanual of laboratory & Dignostic tests, Fourth edition, J.B. Lippincoff company, New York 1992; pp. 471-472, 498-499.

9. Goodman, J.S., Schaffner, W., collins, H. A., et al.: Infections after cardiovascular surgery: clinical study including examination of antimicrobial prophylaxis. N. Engl; J. med 1968; 287: 117.

10. Guntheroth, W. G. How important are dental procedues as a cause of infective endocarditis, AM.J. cordial, 1984, 54: 797.

11. Karchmer, A.W.: Antibiotic therapy of non enterococcal streptococcal and staphylococcal endocaditis: current regimens and some future considerations J. Antimicrob. chemother 1988; 21 [suppl.c]: 91.

12. Karchmer, A.W.: staphylococcal endocarditis, laboratory and clinical basis for antibiotic therapy. AM. J. Med. 1985, 78: [suppl. 6 B]: 116.

13. Kaye, D.: Prophylaxis of endocarditis. In kaye, D. (ed): Infective endocarditis. Baltimore, university park press. 1976; PP 245-265.

14. NYH: (New york heart assocation criteria committee: Diseases of the heart and blood vessels, Nomenclature and criteria for diagynosis 6th ed., P. 114. Little, Brown, Boston. 1964; p. 114.

15. Pallasch, T.J.: A critical appraisal of antibiotic prophylaxis. Int. Dent. J. 1989; 39:183.

16. Rubinstein, E., Noriega, E.R. Simberkoff, M.S., et al. Fungal endocarditis. Medicine Analysis of 24 cases and a review of the literature, Medicine, 1975; 54: 331.

17. Rutledge, R., Kim, J., and Applebaum, R.E.: Actuarial analyris of the risk of prosthetic valve endocarditis in 1, 598 patient with mechanical and bioprosthetic valves arch surg, 1985; 120: 469.

18. Weibel, M.A., CORTAT, M., Lebek, G., et al., An approch of the in vivo antibacterial activity of benzoxonium chloride and comparison with other buccopharyngeal disinfectant. Arzeneim - forsch. Drug Ros. 1987, 37 (1), (4) 467-471.

19. Weinberger, I., Rotenberg, Z., Zacharovitch, D., et al. Native valve endocarditis in the 1970's versus the 1980's: underlying cardiac lesions and infecting organims clin. cardiol. 1990; 13:94.

Complications of pulmonary resection: Mansoura experince

Abstrct

566 pulmonary resection operations; 287 in males & 279 in females; were done at Mansoura Cardiothoracic Surgery Department in the form of pneumonectomy (126); lobectomy (258); bilobectomy (78); lobectomy plus segmentectomy (66) and segmentectomy alone (38). Mortality in this series is 0.7%. Intraoperative and postoperative complications are analysed and discussed giving an idea about how to avoid and how to manage these complications. It is also correlated with age, with the specific aetiology for which pulmonary resection has been done and with the type of pulmonary resection. Intraoperative complications included, vascular injuries (3.3%), diaphragmatic injury (0.2%), pericardial injury (0.4%), cardiac arrest (0.2%) and Cardiac arrhythmia (0.5%). Postoperative complications are in the form of rethoracotomy for intrathoracic bleeding (1.6%), persistent pleural pouch (1.4%), empyema (2.6%), bronchopleural fistula (1.9%), collapse of a remaining lobe (0.4%) and wound infection (1.8%).

Mohamed, M. El-Saeid; Mohamed, A. F. El-Gamal; Salah, A. Khalaf; Nasr, L. Gayed; Ahmed K. Abdallah; Shaaban, A. AbulEla; & Fouad, Z. Abdullah.

From Cardiothracic Surgery Department Mansoura University

Introduction

Major advances in surgical technique for pulmonary resection were made by Churchill and Belsey[1]; in 1939, and in 1940, by Blades and Kent[2], who demonstrated the feasibility and advantages of precise anatomic dissection of the bronchovascular structures in pulmonary hilum, with individual suture of each structure rather than mass ligation, as has been the practice until that time.

Resection of a lung or portion thereof carries with it certain attendant risks and potential complications. Some of these complications are common to any surgical endeavor, and others are unique to this type of operative procedure. In order to manage adequately any compliction resulting from pulmonary resection, one must be able to recognize these complications early, and be familiar with all their ramifications.

Aim of the work

Is to estimate the incidence of complications of pulmonary resection to correlate it with the specific aetiology for which pulmonary resection has been done; to correlate it with different types of pulmonary resection and to give an idea about how to avoid and to manage these complications.

Clinical Material

This is a collective review of the series of pulmonary resections done at Cardiothoracic Surgery Department of Mansoura University. It included 566 patients in whom full chest examination and investigations; necessary to diagnose their chest problems; have been done. Also all investigations prior to surgical intervention, as well as those sppecific to this type of surgery have been done. 247 were males (50.7%) & 219 (49.3%) were females. Most of them were in the middle age group (Table I).

The specific indications for pulmonary resection in this series were shown in Table II. The most common cause for which pulmonary resection has been done in our

Table I: Age and sex incidence

Age group	Male		Female		Total	
	No.	%	No.	%	No.	%
< 20 years	72	12.7	96	17	168	29.7
20 - 40 years	128	22.6	83	14.7	211	37.3
41 - 60 years	81	14.3	84	14.8	165	29.1
> 60 years	6	1.1	16	2.8	22	3.9
Total	287	50.7	279	49.3	566	100

Table II: Specific aetology for which pulmonary resection has been done

Specific aetiology	T-B & its complications	non specific infection & infestation	tumours		Cong. mal-formation	Trauma		Total
			adenoma	Carcinoma		early	neglected	
No.	250	162	45	53	44	2	10	566
%	44.2	28.6	7.9	9.4	7.8	0.4	1.8	100

Table III: Types of pulmonary resection operations

Type of pulmonary resection	(P.)	(L.)	(B.L)	(Lts.)	(S.)	Total
No.	126	258	78	66	38	566
%	22.3	45.6	13.8	11.7	6.7	100

locality was tuberculosis or its complications, (44.2%).

Pulmonary resections included in this series (Table III) were in the form of pneumonectomy (22.3%); lobectomy (45.6%); bilobectomy (13.8%; lobectomy and segmentectomy (11.7%) or segmentectomy alone (6.7%). Of these 566 operations, 48.7% were right sided; 51.1% were left one and only 0.2% was a bilateral resection. Table IV detailed these operations. The appoach in all patients was a posterolateral thoracotomy; except in one patient, it was a median sternotomy for middle lobectomy and lingulectomy.

Results

Complications of pulmonary resection in our series are shown in table V; together with its incidence as regards the total number of operations. Moreover, these complications are correlated with age group, (Table VI); with the specific aetiology for which pulmonary resection has been done (Table VII) and with the type of operation (Table VIII).

Discussion

The major problems that if not recognized in the preoperative period might preclude a successful outcome following pulmonary resection are; pulmonary insufficincy preexisting cardiovascular disease and unusual medical problems that compromise the patient's response. Consequently, these factors should be thoroughly evaluated prior to any operative procedure, except in emergency[3] (Jordan, 1981).

Complications in our series go hand in hand with increasing age of patients submitted for pulmonary resection (Table VI). Patients under age of 20 years have the lowest incidence of the overall complications (6%). This could be easily explained by the fact that, surgery in infants and children is much easier than in adults and pulmonary resection in these patients is simplified by the elasticity

Table IV: Details of pulmonary resection operations (566).

Rt. sided operations.

Operation	P	U.L	L.L	M.L	M.L + L.L	A.S. L.L.	A.P.S.-U.L.	B.S of L.L.	M.L. + A.S. L.L.	U.L+ A.S. L.L.	Total
No.	55	52	54	23	78	5	3	2	1	3	276
%	9.7	9.2	9.5	4.1	13.8	0.9	0.5	0.4	0.2	0.5	48.7

Lt. sided operations.

Operation	P.	U.L	L.L.	Ln.,	L.L.+ Ln.	A.S .L.L.	Total
No.	71	49	80	27	59	3	289
%	12.5	8.7	14.1	4.8	10.4	0.5	51.1

Bilateral operation.

Operation	M.L. + Ln.	Total
No.	1	1
%	0.2	0.2

P.= Pneumonectomy
L.= Lobectomy
B.L.= Bilobectomy
L.+S.= Lobectomy & segmeutectomy
S. = Segmentectomy
A.S. L.L. = Apical segmentectomy of lower lobe.
A.P.S.U.L. = Apicoposterior segmentectomy of upper lobe.
B.S.L.L.= Basal segmentectomy of lower lobe.

U.L. = upper lobectomy
L.L. = lower lobectomy
M.L.= Middle lobectomy
Ln. = Lingulectomy

Table V: Specific complications of pulmonary resection among all patients (566).

Complication	No.	%
Intraoperative complications.		
* Vascular injury (V.I)	19	3.3
(Pulmonary vascular injury)	14	2.4
(Slipped ligature)	3	0.5
(Azygos vein injury)	1	0.2
(Subclavian artery injury)	1	0.2
* Diaphragmatic injury (D.I).	1	0.2
* Pericardial injury (P.I.)	2	0.4
* Cardiac arrest (C.A)	1	0.2
* Cardiac arrythmia (C.Ar).	3	0.5
Postoperative complications		
* Rethoracotomy for intrathoracic bleeding (R.T)	9	1.6
* Persistent pleural pouch (P.P.P.)	8	1.4
* Empyema (E).	15	2.6
* Bronchopleural fistula (B.P.F.)	11	1.9
* Collapse of a remaining lobe (C.R.L.)	2	0.4
* Severe wound infection (W.I.)	10	1.8
Mortality	4	0.7
Total	85	15

of the pulmonary vessels which are more visible and usually devoid of fat. Moreover, these patients have less extensive adhesions than in older ones. Also, for the same reason; while correlating the overall incidence of complications with the specific aetiology for which pulmonary resection has been done (Table VII), the lowest incidence has been met with in surgery for congenital malformations (2.3%). However an intimate knowledge of the developmental anatomy is required. Surgery for bronchial adenoma carried a lower incidence of complications than for carcinoma (4.4% & 11.3% respectively). The highest incidence of the overall complications, in our study, have been met with in surgery for tuberculosis or its complications (18.4%) and in surgery for non specific infections (15.1%).

Complications of pulmonary resection also bear a relation to the type of the operation (Table VIII). Segmentectomy, especially if combined with lobectomy carried the highest

Table VI : Correlation of complications of pulmonary resection with age group.

age group	No of patients	Complications																						Total	
		Intraoperative										Postoperative													
		V.I		D.I.		P.I.		C.A		C.Ar		R.T		P.P.P		E.		B.P.F.		C.R.L.		W.I.		No	%
<20Y	168	3	1.8	-	-	-	-	1	0.6	-	-	-	-	3	1.8	1	0.6	-	-	1	0.6	1	0.6	10	6
20-40Y	211	5	2.4	1	0.5	1	0.5	-	-	-	-	4	1.9	2	0.9	8	3.8	3	1.4	1	0.5	3	1.4	28	13.3
41-60Y	165	9	5.5	-	-	1	0.5	-	-	2	1.2	4	2.4	3	1.8	4	2.4	7	-	-	-	5	3.0	35	21.2
>60Y	22	2	9.1	-	-	-	-	-	-	1	4.5	1	4.5	-	-	2	9.1	1	4.5	-	-	1	4.5	8	36.4
Total No. of patients	566	19	3.3	1	0.2	2	0.4	1	0.2	3	0.5	9	1.6	8	1.4	15	2.6	11	1.9	2	0.4	10	1.8	81	14.3

V.I. = vascular injury
D.I. = diaphragmatic injury
P.I. = pericardial injury
C.A. = cardiac arrest
C.Ar. = Cardiac arrythmia.
R.T. = rethoracotomy
P.P.P.= persistent pleural pouch
E. = empyema
B.P.F = bronchopleural fistula.
C.R.L. = Collapse of remaining lobe.
W.I. = Wound infection

Table VII : Correlation of complications of pulmonary resection with specific aetiology.

Specific aetiology	No of patients	Complications																						Total	
		Intraoperative										Postoperative													
		I.I.		D.I.		P.I.		C.A.		C.Ar.		R.T.		P.P.P.		E.		B.P.F.		C.R.L.		W.I.		No	%
T.B or its complications	250	12	4.8	1	6.4	1	0.4	-	-	1	0.4	6	2.4	6	2.4	7	2.8	6	2.4	1	0.4	5	2	46	18.4
non specific infection & infectations	*172	6	3.4	-	-	1	0.6	-	-	1	0.6	3	1.7	2	1.2	4	2.3	4	2.3	1	-	4	2.3	26	15.1
Tumours adenoma	45	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	2.2	-	-	-	-	1	2.2	2	4.4
Carcinoma	53	1	1.9	-	-	-	-	-	-	1	1.9	-	-	-	-	3	5.7	1	1.9	-	-	-	-	6	11.3
Cong. molforation	44	-	-	-	-	-	-	1	2.3	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	2.3
Trauma *	* 2																								
Total	566	19	3.3	1	0.2	2	0.4	1	0.2	3	0.5	9	1.6	8	1.4	15	2.6	11	1.9	2	0.4	10	1.8	81	14.3

* The group of trauma included 12 patient; of whom 2 patients had early intervention for deep seated pulmonary tears or vascular injuries & no complications happened in them. The remaining 10 patients had neglected chest injures & they shifted to the group of non specific infection because they had bronchial stricture & distal pulmonary infection & atelectasis & in whom surgery has been done; in fact, for the distal infection rather than for trauma.

Table VIII: Correlation of complications of pulmonary resection with type of operation.

Operation	No of patients	Complication																						Total	
		Intraoperative										Postoperative													
		V.I		D.I		P.I		C.A		C.Ar		R.T.		P.P.P.		E.		B.P.F.		C.R.L.		W.I.		No	%
P.	126	9	7.1	-	-	-	-	-	-	2	1.6	2	1.6	-	-	12	9.5	5	4	-	-	1	0.8	31	24.6
L.	258	5	1.9	1	0.4	-	-	1	-	-	-	3	1.2	3	1.2	-	-	3	1.2	2	0.4	4	1.6	21	8.5
B.L.	78	2	2.6	-	-	-	-	-	-	-	-	-	-	3	3.8	-	-	-	-	-	-	2	2.6	7	9
L.+S.	66	3	4.5	-	-	2	3	-	-	1	1.5	3	4.5	2	3	2	3	3	4.5	-	-	1	1.5	17	25.8
S.	38	-	-	-	-	-	-	1	2.3	-	-	1	2.6	-	-	1	2.6	-	-	-	-	2	5.3	4	10.5
Total No. of patients	566	19	3.3	1	0.2	2	0.4	1	0.2	3	0.5	9	1.6	8	1.4	15	2.6	11	1.9	2	0.4	10	1.8	81	14.3

incidence of the overall complications (25.8%) and segmentectomy alone had a higher incidence of complications over lobectomy or bilobectomy (10.5%, 8.5%; and 9% respectively). Also a higher incidence of these complications has been met with in pneumonectomy (24.6%). However the individual complications differ in their incidence with the various types of pulmonary resection and each complication will be discussed separately.

Intraoperative complications

1- Vascular injuries (19 patients : 3.3%):

Dissection of the pulmonary vessels during pulmonary resection can be carried out safely; only; if the surgeon has solid knowledge of the vascular anatomy and its variability. As pointed out by Reed and Allbritten, 1969, the veins are less constant anatomically than are the arteries and the arteries vary more than the bronchi. In our series, pulmonary vascular injury happened in 14 patients (2.4%) and it could be controlled without intraoperative mortality, although in some of these patients massive bleeding occured. Injury of pulmonary vessels frequently occurred when an attempt to encircle an inadequately exposed vessel with a clamp in order to pass a ligture and divide the vessel. In those instances when a lebectomy is being performed and the hilar dissection was difficult, we passed a tape around the proximal main pulmonary artery before dissecting the branches. In our experience, we had an injury to main pulmonary artery in 5 cases; injury to the branches of pulmonary artery in 6 cases and in 3 cases, we had an injury to pulmonary veins. The latter are usually less serious than arterial injury, since the arterial branches have been divided prior to dissection of the veins and digital control is rendered easier by the resulting low flow and pressure. However, in one case, massive bleeding happened due to injury of the superior and inferior pulmonary veins at their entry into the atrium by a single trunk and it could be controlled after opening the pericardium. From our experience, in performing a pneumonectomy, the pulmonary artery is dissected and exposed the superior and inferior pulmonary veins are better to be divided between vascular clamps and closed with a continuous vascular suture. In one case of right lower lebectomy, injury to middle lobe artery necessitated its removal, although it was a healthy lobe.

Slipped ligature was of low percentage in this series (3 cases; 0.5%). We always used the technique of tripple ligation and we divide the vessel below bifurcation, whenever possible. However this technique needs more dissection and more time .

We had, also, a single case (0.2%) of injury to azygos vein due to extensive adhesions, since this patient had a previous empyema and it could be ceasily controlled while performing a pleuropneumonectomy.

An interesting vascular injury happened in one case (0.2%) in our series, that is injury to left subclavian artery. It was not an inadvertent injury, but it was ligated and cut in between assuming that it is the left main pul-

monary artery while doing a left pneumonectomy. The subclavian artery was loopnig and very adherent to the undersurface of the left pulmonary apex. After completing dissection, the complication was recognized and overcome by reanastomosing the subclavian artery. Although it did well postoperatively, as monitored by doppler examination of the radial and cubital arteries, it is wise to complete dissection of the arteries before hurrying to ligate and divide any.

Correlation of vascular injury, in our series, is more evident with specific aetiology for which pulmonary resection has been done rather than the operative procedre. However, massive intraoperative bleeding is correlated with both. Pulmonary resection for tuberculosis and suppurative lung disease differ from that for tumours, by the presence of dense adhesions especially with drained empyemas. Also fibrocalcific changes in lymph nodes make dissection of the vessels hazrdous. The highest incidence of vascular injuries, in our series; has been met with in pulmonary resection for tuberculosis (4.8%) followed by nonspecific infection (3.4%). The high incidence of vascular injuries with pneumonectomy in our series (7.1%) could be attributed to the specific a eteology rather than the operative procedure since in most of these cases pneumonectomy was done for tuberculous or suppurative lung diseases. In our locality there is a higher incidence of suppurative lung disease and emergence of drug resistant strains in tuberculosis, togther with late presentation of bronchogenic carcinoma.

2- Diaphragmatic injury (1 patient : 0.2%):

This complication has been met with in one case only due to extensive adhesions between the base of the lung and diphragm, due to a previous empyema and intercostal tube drainage for a long time before doing pulmonary resection; a lower lobectomy. This complication has been recognized and overcome easily by suturing the diaphragm. Again, it was disease ralated rather than operatively correlated, being in a tuberculous patient.

3- Pericardial injury (2 patients : 0.4%):

The pericardium was opened inadvertently in two of our cases during dissection of the mediastinal surface of the lung due to extensive adhesions. It was recognized and sutured early during operation to avoid collection of blood into the pericardium. In our series, this complication was correlated with the type of operation, being lobectomy and segmentectomy in the two cases and also correlated with the specific aetiology, being either tuberculous or non specific infection.

4- Cardiac arrest (1 patient: 0.2%) & cardiac arrhythmia (2 patients: 0.5%):

These complication are commonly associated with inadvertent hypoxia or hypotension. Nevertheless, it is important that the surgeon be aware of the presence of any preexisting cardiovascular disease.

Digitlization is known to be effective in preventing postoperative supraventricular tachyarrhythmias in the elderly, especially

following pneumonectomy and this course should be considered (3). In our series, cardiac arrest intraoperatively, happened in one patient (0.2%) and cardiac arrhythmia in 3 patients (0.5%). These complications, actually, were anaesthesia related, rather than operatively correlated and mostly due to a period of hypoxia during operation, especially due to excessive secretions. Double lumen tubes (Carlen's or Robertshow) were not used routinely in our pulmonary resection operations because it was not available at a time and because of its difficult insertion by the anaesthetist at another. Even at occasions, these tubes have been changed by the anaesthetist during operation by single lumen tubes due to difficulty in suctioning through these tubes. However, the use of these double lumen tubes can provide undeniable advantage in pulmonary resection especially for cavitary or cystic lesions, bronchiectasis, haemoptysis, or bronchopleural fistula (4).

Postoperative complications:

1) Rethoracotomy for intrathoracic bleeding (9 patients: 1.6%):

Bleeding from the chest, following pulmonary resection, usually arises from one of three sources; the major vessels within the thorx; chest wall, or mediastinum (Jordan, 1981). In our experience rethoracotomy has been done in 9 patients (1.6%), for either persistent intrathoracic bleeding or intrathoracic hematoma detected radiographically. In these cases, the source of bleeding was either chest wall, bronchial vessels, or the raw surface of the lung after partial lung resection. None of our cases showed a great vessel as a source of bleeding. In most of our cases, unless a pneumonectomy, we adopted the policy to leave two tubes in the chest; one apical and one basal to avoid obstruction of a single intercostal tube and formation of intrathoracic hematoma. Fore the same reason, we almost always put an intercostal tube after pneumonectomy, although most authors did not use it as a routine. However we leave it for a short period postoperatively and it is removed as soon as possible. In 3 patients, at the time of exploration, after evacuation of clots from the pelural space and expansion of the remaining lung, we did not find obvious bleeding points, even after a thorough search of the entire lung surface, chest wall and mediastinum and these patients did well after closure of the chest. In others (2), we found the source of bleeding to be from the raw surface of the lung after segmental resection or lobectomy with incomplete fissure. Just coagultion or running stitches solved the problem. In 2 patients, bronchial vessels were respensible for rethoracotomy, although we ensisted to control bronchial vessels in all our operations by running stitches and never by diathermy coagulation. In one case, we found the source of bleeding to be from intercostal vessel, most probably injured during closure of the chest in the first thoracotomy. A running stitch of the bleeding vessel overcomed the problem. In another patient, the source of bleeding was the site of extensive adhesions met with in the first thoracotomy in posts-plenectomy pulmonary resection.

Diathermy coagulation failed to control the bleeding sites from the diaphragm and running stitches needed. Rethoracotomy, as a complication after pulmonary resection bears little or no correlation with the type of pulmonary resection as that with the specific pathology for which pulmonary resection has been done. 6 patients (2.4%) were tuberculous and 3 patients (1.7%), had nonspecific infection.

2- Persistant pleural pouch (8 patients : 1.4%):

This complication is peculiar to partial lung resection. Following lobectomy, full re-expansion of the remaining lung on the operated side is essential in order to obliterate the pleural space. After partial lung resection, two chest tubes was our policy as a routine in most of our cases; one in the apex of the pleural space and the other posteriorly at the base. These tubes were connected to under water seal drainage with a negative pressure of approximately-15 cm of water, especially if there is considerable air leak postoperatively. These tubes were tested for patency and milked at frequent intervals while they were in place. Postoperative air leak from a raw parenchymal surfaces may last up to 2 weeks after pulmonary resection. However, if the lobe is fully expanded and there is no residual air space, these leaks will almost always seal off. We have utilized various maneuvers to stop air leaks that have persisted beyond few days postoperatively; ranging from low suction or high suction depending on the amount of air-leak, tube manipulation or pleurodesis. The effectiveness of these procedures varied with the individual case. Occasionally small air caps has been left at the apex of the lung with no evidence of active air leaks. In our experience, it has not been deterrent to a satisfactory recovery and the space usually obliterated by accumulation of some fluid and organization and fibrosis of the apex of the pleural space. In some patients, we anticipated non filling of the remaining lung during operation and we added tailoring thoracoplasty to our pulmonary resection. In others, we added pneumoperitoneum to elevate the diaphragm. However, inspite of these maneuvers to avoid persistent air pouches, we have been faced with this complication in 8 of our patients (1.4%). In 3 infants and children, the problem solved in a short time by tubal drainage and it was mostly, due to early removal of the intercostal tubes after the pulmonary resection. In another two older patients, tubal drainage succeeded to overcome the problem but it took a longer time than in younger ones. Tailoring thoracoplasty was necessary in 3 patients, after failure of tubal draimage, to avoid pleural space infection and possible bronchopleural fistula.

By reviewing our results in tables VII, VIII, it is apparent that this complication is correlated with both, type of operation as well as the specific aettiology for which the operation has been done. Its incidence was 3.8%

after bilobectomy, 3% after lobectomy plus segmentectomy and 1.2% after lobectomy alone, which means that, the greater the magnitude of partial pulmonary resection; the higher the incidence of persistent pleural pouch after resection. Also a higher incidence of this complication (2.4%) is noted in pulmonary resection for tuberculosis or its complications, which may be attributed to the structural changes of the remaining lung by the tuberculous process & the decreased lung compliance.

3- Empyema (15 patients: 2.6%):

Empyema may occur in the pelural space after pulmonary resection in the abscence of a bronchopleural fistula. This is usually the result of a dead space within the pleural cavity which is filled with blood or fluid with subsequent contamination and development of frank empyema. It may be associated with a transient bronchoepleural fistula that close spontaneously and may occur at any time after surgery (5). Postoperative empyema bears some relation to the type of operation especially during its management. It occurred in 15 patients (2.6%), in our series and its incidence was higher after pneumonectomy (9,5%), followed by lobectomy and segmentectomy (3%) or segmentectomy alone (2.6%). By comparing the last two figures, segmentectomy may be accused as a cause of this empyema. Empyema, following partial pulmonary resection (3 in our series) has been managed by tubal drainage, except one case, needed in addition decortication after sterilization of the empyema pouch.

Empyema following pneumonectomy in abscence of bronchopleural fistula creates a slightly different problem in that, there is no lung to expand to fill the space. Various techniques have been described to manage postpneumonectomy empyema. (6) and described their technique of open drainage, daily irrigation with Dakin's solution followed by definitive closure with instillation of neomycin in saline. This method is, also reported by Stafford and Clagett, (7). Moreover, Karkola, et al., 1976, (8) had uniform success, however, in patients with fistula as well. Zumbo, et al., (9) noted that this method is only of value in those patients with no evidence of bronchopleural fistula. On the other hand, Harrison, pointed out that mixed tuberculous and pyogemic empyema has not been successfully treated by the previous techniques and thoracoplasty with or without decortication will probably be necessary if combined chemotherapy and local measures are unable to eliminate the mycobacterial component of the infection. As, Langston, (10), noted, most empyemas that occur in the immediate postoperative period are pyogenic and not tuberculous.

In our experience, rapid collection of serous or serosanguinous fluid after pneumonectomy without symptoms of infection require only aspiration under strict asepsis, in order not to foster the bronchial sutures.

Langston, 1978, reported an incidence of postpneunonectomy empyema in abscence of bronchopleural fistula, to be 2-10%. In our series, it was 7.1% (12 patients). We adopted the technique of Clagett and Geraci, with little modification. After insertion of an intercostal tube, daily lavage of the postpneumonectomy space was done with instillation of proper antibiotic, untill the wash get clear and the fever subside. The tube, then removed, its site closed with instillation of a solution of antibiotic according to culture and sensitivity. However, it must be mentioned that, most of our cases were not a frank empyema, but just turbidity of the fluid collected in the postpneumonectomy space. In one patient, empyema develeped one year after pneumonectomy for bronchogenic carcinoma with postoperative irrdiation and chemotherapy. After failure of tube drainage and daily lavage, open draimage was done using the open flap technique described by Eloeser, (11).

4- Bronchopleural fistula (1.9%):

Although the frequency of bronchopleural fistula is decreasing, it remains a major therapeutic challange for thoracic surgeons and that develop after pneumonectomy is the most dreaded complication after pulmonary resection. Its incidence is 2-7% following all types of pulmonary resection by Malove, et al., (12) (13) and 2-13% by Mc Manigle et al., 1990. Its incidence after pneumonectomy should not exceed 3% (Boyd & Spencer, (14)). Williams and Lewis (15), pointed out that pneumonectomy carries a three fold incidence of fistula over lobectomy (4.5% and 1.5%). In our study, an incidence of 1.9% following all types of pulmonary resection has been encountered. That following pneumonectomy was 4% and after lobectomy, it was 1.2%. However the highest incidence was detected after lobectomy plus segmentectomy (4.5%). Our results bear also a correlation to the specific aetiology, since an incidence of 2.8% and 2.3% followed resection for tuberculosis and non-specific infection respectively. Hood, 1986, reported higher incidence of bronchopleural fistula in tuberculous patients undergoing pulmonary resection than others. However, in our series, although, a little difference is recognized between tuberculous group and the group of non specific infection (2.8% & 2.3%); both had a higher incidence of fistula than others (Table VIII). Moreover, our incidence of fistula formation in tuberculous patients is lower than that reported by Harkin's et al., 1978, where they reported an incidence of 3-7%.

There are many predisposing factors to the development of post operative bronchopleural fistula, but technical errors in bronchial stump closure take the upper hand. The most important principle to prevent bronchopleural fistula is to obtain complete lung expansion early and maintain it continuously. In our expenece, we used pericardial fat pad flap to cover the stump in 6 cases and the non tensions posterior membraneous flap in 2 cases of pneumonectomy and unfortunately we had one chance to use staplers. However; Dart et a., (16), reported that bronchopleural fistula could occur with all types of bronchial closure including stapling. In the early era, in our experience, we used silk sutures for

bronchial closure, that has been changed, since a long time to monofilament prolene sutures which gave good results. Stainless steel sutures used twice in our series but it was difficult to handle, that discouraged its routine use. The monofilament non absorbable sutures, still used and recommended by many authors (Góffi & concalves, (17); Swasaki, et al., (18)). Not only the type of suture material that influences the incidence of bronchopleural fistula, but also, the use of minimal number of sutures for stump closure is very important in detemining the healing of bronchial stump (Rienhoff et al., (19)). It should be tied firmly enough to ensure air tight closure but not too tight to cause necrosis of the intervening tissue (Williams and Lewis, 1976). Avoidance of overzealous dissection of the bronchus and avoidance of long bronchial stumps, also, help in lowering the incidence of bronchopleural fistula (Malove et al., 1971, Harrison, 1980).

In our series, we had 5 (4%) of such fistulae after pneumonectomy that developed after one week or more. Immediate tube drianage was established. Neither of them healed under tube drainage. In three of them, after sterilization of the post-pneumonectomy space, the fistula was dissected down to the bronchial stump, excised down to normal cartilage and the bronchus was closed using a pedicle graft of intercostal muscles as described by Barker et al., (20) and Kirsh et al., 1975 . In the other two cases, thoracoplasty was performed. Also we had 6 cases with postpartial resection bronchopleural fistulae. Three of them were after basal pulmonary resection. Proximal pulmonary resection was done in two cases because the remaning lobe was bronchiectatic. In the third case, decortication was done after drainage of the empyema. In the 3 apical resections followed by bronchopleural fistula, thoracoplasty solved the problem in 2 of them. In the 3 rd patient the fistula developed early with no considerable infection; reamputation of the stump and suturing was effective, especially the remining stump was long enough.

5- Persistent collapse of a remaining lobe (2 patients : 0.4%):

Radiological evidence of residual atelectasis in the immediate postoperative period was frequent in our patients due to secretion and inability to cough and could be easily managed by nasotracheal suctioning (Haight maneuver) or bronchoscopic suctioning. Our problem of persistent atelectasis is different and needs special attention. It was operatively related and it has been met with in two instances of left lower lobectomy due to unrecognized intraoperative encroachment on the lumen of left upper lobe bronchus. Although, the remaining lobe inflated intraoperatively, after operation residual atelectasis has been noticed. Repeated bronchoscopic suctioning was done while following these patients post operatively. The remaining left upper lobe goes into periods of partial inflation and collapse. We did every effort as regards physiotherapy, repeated bronchoscopy and medical tretment and now, we are thinking of proximal resection. This

complication must be put in mind while doing such pulmonary resection.

6- Wound infection (10 patients : 1.8%):

The incidence of wound infection following posterolateral thoracotomy is approximately 1% in most institutions (Adkins and Aaron, 1981). Our results are higher, being 1.8% and should be reevaluated by us to lower this incidence since a part; at least of this incidence; could be avoidable. In our experience, this complication is related to the specific aetiology for which pulmonary resection has been done rather than to the type of the operation; mainly resection for non-specific infection (2.3%) and that for tuberculosis (2%). Our major concern in the management of this problem was the prevention of extension of the superficial wound infection into the pleural space and creation of an empyema which is particularly worrisome in the postpneumonectomy patients. Our policy was to open, early, the entire infected area plus debridement together with subsequent packing and frequent irrigation which resulted in a clean granulomarous area and closure without a dead space.

Mortality in our series (4 patients : 0.7%):

Improved anaesthetic techniques and chemotherapy, selection of patients and the use of stapling devices have contributed to steady decrease in mortality and morbidity of pulmonary resectional procedures (Graensler, (21) ; Moran et al., (22)).

In our series, we have 4 mortalities (0.7%) following pulmonary resection. Although in most of them, the fatality may be attributed to the disease process itself rather than the operative procedure, it is wise to give a brief account on these cases. In three of them, pulmonary resection was done for bronchogenic carcinoma. One of these patients died on the 6 th postoperative day without an obvious cause, although she did well in the early postoperative period and unfortunately we have no postmortem study in our hospital. The second patient had postoperative empyema that necessitated tubal drainage for a time. Repeated attacks of bleeding through the tube happened and the patient has been lost during one of these massive attacks. The 3 rd patient died on the 8 th postoperative day after a severe acute bleeding per rectum, shock and circulatory failure. The remaining patient, who was not malignant underwent left lower lobectomy for bronchiectasis after splenectomy. She died on the 9 th postoperative day from liver cell failure with very high serum bilirubin and renal affection with elevated serum creatinine. However, this patient was a bad risk for pulmonary resection that has been done as a salvage operation, on an emergent basis for the massive recurrent attacks of haemoptysis. This must raise attention to the thoracic complications of splenectomy; the operation frequently done in our locality for bilharziasis.

REFERENCES

1. Churchill, E.D. & Belsey, R.: Segmental pneumonectomy in bronchiectasis; the lingula segement of the left upper lobe. Ann. Surg., 109:481 (1939).

2. Blades, B., & Kent, E.M.: Individual ligation technique for lower lobe lobectomy. J. Thorac. Surg., 10:84, (1940).

3. Kirsk, M.M; Rotman, H., Benrendt, D.M., et al.,: Complications of pulmonary resection. Ann. Thorac. Surg., 20:215. (1975).

4. Harrison, H.: Current aspects of the surgical management of tuberculosis. Surg. Clin. North. Am., 60:883 (1980).

5. Adkins, P.C & Aaron, B.L.: Complications of pulmonary resection. In textbook of complications in surgery & their managment. 4 th ed. Chap. 15, P.P. 313 W.B. Saunders compny (1981).

6. Clagett, O.T, & Geraci, J.E.: A pricedure for the management of postpneumonectomy empyema. J. Thorac cardioovasc. Surg., 45:141 (1963).

7. Karkola, P.; Kiraluma, M.I. & Larmi, K.: Postmeumonectomy empyema in pulmonary carcinma patients. J. Thorac. Cardiovasc. Surg. 72:314, (1976).

8. Stafford, E.G. & Clagett, O.T.: Postpneumonectmy empyema. J. Thorac. Cardivasc Surg., 63:771 (1992).

9. Zumbo, G.L.; Treasure, R.; Geiger, J.P. & Green, D.C.: Empyema after pneumonectomy. Ann. Thorac. Surg., 15:615 (1973).

10. Langston, H.,: Barker, W.L., & Ryle, M.M.: Physiological evaluation of the patient. In Goldsmith practice of surgery. Ellis thoracic surgery. Harper & Row publishers. New Yrk, San Frncisco, London, (1978). chapter 11 PP. 71.

11. Eloesser, L.: An operation for tuberculous empyema. Surg. Gynecol. Obstet 60: 1096, (1935).

12. Malove, G.; Foster, E.D., Wilon, J.A. & Mun, R.D.: Bronchopleural fistula. present day study of an old problem. Ann. Thorac. Surg., 11:1, (1971).

13. McManigle, N.E.; Fletcher, G.L., & Tenholder, M.F.: Bronchoscopy in the management of bronchopleural fistula. Chest, 97:1235-38 (1990).

14. Boyd, A.D & spencer, F.C.: Bronchopleural fistulas: How often should they occur? (Editorial). Ann. Thorac. Sug. 13:195 (1972).

15. Williams, N.S & Lewis, C.T. Bronchopleural fistula. Areview of 86 cases. Br. J. Surg., 63:520, (1976).

16. Dart, C.H.; Scott, SM. & Takaro, T.: Automatic stapling devices for lung resections. Ann. Thorac. Surg., 9: 535-520 (1970).

17. Goffi, F.S & Concalves, F.L.: Closure of bronchial stmp after pneumonectomy: comparison of some techniques through evaluation of tensile strength of suture. Surgery, 42:511, (1957).

18. Swaski, H.; Hore, K.; Yamada, M.; Tojima, G.; Naito, Y., watabe, S.; Katsura, S., Murabayashi, A, Nonaka, T. Fuse, S.: postoperative bronchopleural fistula. Clinical & exprimental study chest, 67:702-705 (1975).

19. Rienhoff, W.G.; Gannan, N. & Sherman, J.: Closure of the bronchus following total

pneumonectomy, Experiental & clinicall observations. Ann. Surg., 116:481 (1942).

20. Barker, W.L.; penfield, L.; Faber, W.E.; Ostermiller, Jr. a langsten, H.T.: Management of bronctopleural fistula J. Thorac. Cardiovasc. Surg., 62: 393-401 (1971).

21. Gaensler, E.A.: The Surgery for pulmonary tuberculosis. Am. Rev. Resp. Dis., 125:73 (1982).

22. Moran, J.F.; Alexander, L.G.; Staub, E.W., et al.,: Long term results of pulmonary resection for atypical mycobacterial disease. Ann. thorac. Surg., 35; 597 (1983).

A selected bibliography on Bronchogenic Carcinoma

The Egyptian National STI Network

Cerebral imaging in the asymptomatic preoperative bronchogenic carcinoma patient: is it worthwhile?

Cole-FH Jr; Thomas-JE; Wilcox-AB; Half-ord-HH-3 rd

Ann-Thorac-Surg. 1994 Apr; 57 (4): 383-40

The issue of screening for cerebral metastatic disease in the preoperative bronchogenic cardinoma patient remains unsettled and changes with advancing technology. A prospective nonrandomized study was designed to compare contrast magnetic resonance imaging (MRI) with computed tomography (CT) after several clinical situations suggested improved sensitivity for the former study. Patients with clinically operable disease and normal neurologic examinations were referred for both enhanced cerebral CT and MRI studies. Forty-two patients were entered and completed the enhanced CT scan; only 30 tolerated the MRI. The demographic data and histology of the patients appeared fairly typical for a series of operative candidates. No unsuspected metastatic lesion was found in this selected and low-risk group. We conclude that neither MRI nor enhanced CT scan is indicated in the asymptomatic bronchogenic carcinoma patient due to expense and lack of positive findings. Magnetic resonance imaging demonstrated more subtle benign pathology, but this study did not allow comparison of the two techniques in detection of metastatic disese.

Multicentricity in resected occult bronchogenic squamous cell carcinoma.

Saito-Y; Sato-M; Sagawa-M; Kanma-K; Takahashi-S; Usuda-K; Nagamoto-N; Endo-C; Chen-Y; Sakurada-A; et-al

Ann-Thorac-Surg. 1994 May; 57(5): 1200-5

The frequency and the treatment of multicentricity in 127 patients with resected roentgenographically occult bronchogenic squamous cell carcinoma were studied. The cumulative rate and the incidence of postoperative metachronous multiple primary lung cancer were 0.11 at 5 years after initial operation and 0.022 per patient-year, respectively. The cumulative rate and the incidence of second primary lung cancer,

which includes synchronous and subsequent metachronous cancer in patients with initial lung cancer, were 0.17 at 5 years after the initial operation and 0.041 per patient-year, respectively. The cumulative rate and the incidence of third primary lung cancer in patients with second primary lung cancer were 0.47 at 5 years, which was significantly higher (p=0.05) than that of second primary lung cancer, and 0.11 per patient year, respectively. In all 12 patients with synchronous multiple primary lung cancer, no recurrence was observed after treatment, but 3 had subsequent multiple primary lung cancer. Among the 13 patients with postoperative metachronous multiple primary lung cancer, recurrence was observed in 1 of the 6 patients who underwent resection and in 2 of the 4 patients treated with laser or radiation therapy or both. The overall survival rate at 5 years after initial operation in patients with solitary and those with multicentric occult bronchogenic squamous cell carcinomas was 0.90 and 0.59, respectively.

Tracheal sleeve pneumonectomy for bronchogenic carcinoma.

Roviaro-GC; Varoli-F; Rebulfat-C; Scalam- bra-SM; Vergani-C; Sibilla-E; Palmarini-L; Pezzuoli-G

J-Thorac-Cardiovsc-Surg. 1994 Jan; 107 (1): 13-8

For a long time, primary tumors arising less than 2 cm distal to the carina have presented a contraindication to surgical excision. Tracheal sleeve pneumonectomy technique allows carinal resection and reconstruction but still carries considerable postopertive complications. from 1983 to 1992 we performed 27 right tracheal sleeve pneumonectomies and one left. Fourteen patients had No nodes, nine had NI, and five had N2. No anastomotic complications, either fistula or stenosis, were observed. Successful outcome depends on meticulous attention to surgical details and careful anaesthetic management with a new ventilation tube. One patient died on the twenty- second postoperative day from myocardial infarction. Complications included pneumonia (one), vocal cord paresis (two), and pleural empyema without bronchial fistula (one).

Conservative treatment allowed complete recovery from all complications. There are seven patients alive at 4 years after operation and one at 5 years. Six patients have been disease-free for between 1 and 32 months. Two patients died free of disease at 13 and 42 months. Two patients died of mediastinal recurrence and 10 of distant metastases within 6 and 54 months.

Bronchoplastic and angioplastic techniques in the treatment of bronchogenic carcinoma.

Maggi-G; Casadio-C; Pischedda-F; Cianci-R; Ruffini-E; Filosso-P

Ann-Thorac-Surg. 1993 Jun; 55(6): 1501- 7

From 1979 to 1991, 51 bronchoplasties, 18 angioplasties, and 4 combined broncho-angioplasties were performed for bronchogenic carcinoma. Sixteen patients underwent operation because of compromised pulmonary function; bronchoplasty, angioplasty, or the combined procedure was performed in the remaining 57 patients because of a suitable anatomic location of the neoplasm. Twenty-four patients had stage 1 disease, 32 stage II, and 17 stage IIIa. Three patients died postoperatively (3.65%). Major postoperative complications occurred in 20 patients (27.3%) (10 early, and 10 late). A completion pneumonectomy was required in 4 patients (5.4%), 2 for anastomotic stricture, 1 becuse of vascular thrombosis after angioplasty, and 1 for local recurrence after angioplasty. Three-year and 5-year survival rates for the entire group were 55.4% and 40.8%, respectively. One-year and 3-year survival rates after angioplasty were 78.6% and 31.4%. Of the 4 patients who underwent a combined bronchoangioplastic procedure, 1 died after 23 months and 3 are alive and well after 11, 15, and 20 months.

Survival was more favorable in the combined No-NI group (62% and 43.1%) than in the N2 group (23.4%), but the ditference was not significant ($p< 0.2$). Three-year survival after angioplasty was found to be lower than, although not significantly different from, the overall 3- year survival rate (31.4% versus 55.4%; p = not significant). No statistically significant differences were found among survival rates of patients with compromised and noncompromised pulmonary reserve. We conclude that bronchoplastic and angioplastic procedures are valid techniques as curative operations in carefully selected patients with bronchogenic carcinoma.

Less than lobar resections for bronchogenic carcinoma.

Weissberg-D; Straehley-CJ; Scully-NM; Margulies-DR

Scand-J-Thorac-Cardiovasc-Surg. 1993; 27 (3-4): 121-6

We performed less than lobar resections for peripheral clinical Stage 1 primary lung cancers on 170 patients treated between 1973 and 1987 at two university centers, one in Hawaii and one in Israel. Most patients were poor risks and several had FEVI < 1 liter There were 6 (3.5%) hospital deaths. There were 58 segmental resections, 97 wedge resections and 15 less than lobar resections not otherwise specified. Seventy-three patients (43%) are

living free of cancer from 5 to 11 years postoperatively and 20 additional patients died of causes unrelated to lung cancer after 5 years; thus disease free five year survival was 54.7%. Patients with adenocarcinoma had poorer prognosis than other cell types. Twenty-three patients (13.5%) had synchronous or metachronous second primary lung cancers. Nine of these patients are long term survivors. Twenty-four ptients (14.1%) developed local recurrences with or without distant metastases. This promising long term cancer-free survival and the frequency of second primary lung cancers justifies less than lobar resection for peripheral, Stage I bronchogenic carcinoma, especially in the poor risk patient.

Limited resection of bronchogenic carcinoma in thepatient with impaired pulmonary function.

Miller-JI Jr

Ann-Thorac-Surg. 1993 Sep; 56(3): 769-71

Surgical resection is the treatment of choice for non-small cell carcinoma of the lung. In some patients with marked impairment of-pulmonary function, cardiac disease, or other medical conditions, the surgeon is faced with performing either a limited resection or carying out nonoperative therapy. Impaired pulmonary functions are defined as a maximum breathing capacity (MBC) of 35% to 40% of predicted; forced expiratory volume in 1 second (FEVI) of less than 1 L; and a forced expiratory volume 25%-75% (FEV25-75) of less than 0.6 L. When MBC values are less than 35% of predicted; the FEVI is less than 0.6 L; and the FEV25-75 is less than 0.6 L; elective resection is contraindicated. Useful criteria for indicating an elective limited resection include the following: (1) TI lesion, (2) peripheral location, (3) margins easily encompassed by resection, and (4) no gross lymph node involvement. In a study of 67 patients, there was 1 postoperative death, with less than an 80% 2-year survival and a 31% 5-year survival. The role of video- assisted thoracoscopy in the management of primary lung cancer remains to be defined. When the high-risk patient can be operated on with attendant low morbidity and mortality, I believe, at the current time, a video- assisted thoracic resection for primary lung cancer is not the best option, as the patient will be offered a compromised operation, and I suspect follow-up studies will prove this correct.

Pulmonary resection after pneumonectomy in patients with bronchogenic carcinoma.

Westermn-CJ; van-Swieten-HA; Brutelde-l-Riviere- A; van-den-Bosch-JM; Duurkens-VA

J-Thorac-Cardiovasc-Surg. 1993 Nov.; 106 (5): 868-74

Eight patients with a previous pneumonectomy for bronchogenic carcinoma underwent an additional resection because of a second primary carcinoma in the remining lung. One patient died of pulmonary embolism in the postoperative period. The postoperative course was otherwise uneventful except for prolonged air leak. Two patients died after 3 months (bone metastasis) and 5 months (recurrent small-cell carcinoma). Two patients were alive at the time this article was written but had evidence of recurrence after 18 months (distant metastasis) and 21 months (local recurrence at the site of positive resection margins). Three patients were alive and doing well without evidence of disease after 16, 17, and 40 months. After careful selection, even patients with a previous pneumonectomy may be good candidates for additional resection of a second primary bronchogenic carcinoma.

Tumor presence at resection boundaries and lymph-node metastsis in bronchil carcinom a patients.

Kayser-K; Anyanwu-E; Bauer-HG; Vogt-Moykopf-1

Thorac-Cardiovasc-Surg. 1993 Oct; 41 (5): 308-11

A prospective study was performed analyzing the bronchial resection boundaries of 120 patients operated on for lung carcinoma. The resection boundary, maximum tumor diameter, distance between tumor and resection boundary, and lymph-node stage were analyzed by serial sections of the surgical specimens (lobes and lungs). The following results were obtained: 20/120 cases (17%) displayed microscopic tumor invasion of the resection boundary (RI status), most frequently adenocarcinoma (21%). The RI status was closely associated with the distance between tumor and resection boundary and postsurgical lymph-node state (pN stage): all 8 tumors excised at disrance 1 mm or less from the bronchial resection boundary revealed bronchial submucous tumor growth, where as none of the tumors located more than 20 mm from the resection boundary was found to display tumor invasion of the bronchial boundary. Curative resection was noted in all 40 tumors operated at pNO stage and in only 11 cases (69%) of tumors with distant lymph-node metastases (pN3 stage). No relationship between tumor infiltration of the resection boundry and type of resection was seen. The data indicate that a) intra- operative control of bronchial resection boundaries is necessary in all lung- carcinoma patients with central tumor localization less than 20 mm from the proposed resection boundary; b) a

"safety distance" between resection Boundary and tumor boundary is of specific importance in bronchial carcinoma with lymph-node metastases.

Predictability of FEVI after pulmonary resection for bronchogenic carcinoma.

Sagalli-M; Spiliopoulos-A; Megevand-R

Eur-J-Cardiothorac-Surg. 1992; 6(5): 242-5

The aim of this study was to review the reliability of prediction of postoperative FEVI in patients with bronchogenic carcinoma using a Tc-99m perfusion scan and simple spirometry. Over a 27 month period, 40 patients without known recurrent disease had their FEVI measured. One quarter of the postoperative values for FEVI differed from predicted values by less than 5% (2/11 pneumonectomies, 5/23 lobectomies, 3/6 segmental resections: and half differed by no more than 10% of predicted FEVI (4/11 pneumonectomies, 12/23 lobectomies, 3/6 segmentectomies). One tenth of the predicted values differed by more than 30% and up to 760 mls (1/11 pneumonectomies, 2/23 lobectomies, 1/6 segmentectomy). Disease recurrence, phrenic nerve paralysis, exacerbation of obstructive pulmonary disease and poor collaboration during spirometry explained the most severe erroneous results. Age, preoperative smoking, tumour stage and histology, absence of symptoms at the time of dignosis and adjuvant radiotherapy showed no statistically significant effect on predictability. Twenty-one patients had a post-operative Tc-99m pulmonary scan simultaneous to the spirometric control. Overestimation of postoperative FEVI was associated with heterogeneous distribution of ventilation and perfusion.

Preoperative laser therapy in a patient with resectable bronchogenic carcinoma and severe coronary artery disease.

Powney-J; Scott-AD; George-PJ; Feneck-RO; Wright-J; Barnes-NC

Thorx-1992 Dec; 47(12): 1075-6

A 67 year old man with severe coronary artery disease was found to have a resectable bronchogenic carcinoma. Myocardial revascularisation and lung resection were considered to be unduly hazardous as either separte or combined operations. Preoperative laser therapy, however, enabled the two procedures to be performed in greater safety in the most appropriate sequence.

Prognostic significance of massive bronchogenic tumor embolus.

Heitmiller-RF

Ann-Thorac- Surg. 1992 Jan; 53(t): 153-5

Massive arterial bronchogenic tumor embolus is rare, and most commonly occurs intraoperatively during pulmonary resection. Arterial obstruction from the tumor embolus carries substantial morbidity and mortlity. For those patients who survive embolectomy, prognosis is most closely correlated with the TNM staging of the primary lung tumor ignoring the tumor embolus.

Results of surgical treatment for roentgenographically occult bronchogenic squamous cell carcinoma.

Satio-Y; Nagamoto-N; Ota-S; Sato-M; Sagawa-M; Kmma- K; Takahashi-S; Usuda-K; Endo-C;

J-Thorac-Crdiovasc-Surg. 1992 Aug; 104 (2): 401-7

Ninety-four patients with roentgenographically occult bronchogenic squamous cell carcinoma had surgical resection. Fifty-three reported having no symptoms. In 83 carcinoma was detected by cytologic examination of the sputum during lung cancer screening. The carcinomas were located in segmental bronchi (34 cases), subsegmental bronchi (19 cases), divisional bronchi (17 cases), and subsubsegmental or more peripher al bronchi (15 cases). The number of cases classified by TNM staging were 16 Tis NO MO, 72 TI NO MO, 4 TI NI MO, and 2 T2 NI MO. Extrabronchial invasion of the resected carcinoma was observed in 17 lesions (16 cases). Five of six patients with lymph node metastasis in the resected specimens had carcinoma with extrabronchial invasion. Multiple primary lung cancers were observed in nine patients at the time of operation and in seven subsequently. Four of seven patients with subsequent primary lung cancer had surgical resection, and no recurrence was observed after the second operation. There were two deaths from lung cancer: One was caused by subsequent primary lung cancer and the other by mediastinal lymph node metastasis. In the 75 patients with intrabronchial cancer invasion and without lymph node metastasis who had complete resection, there was no local recurrence or metastasis of cancer. The 5- year survivals were 80.4% (death from all causes) and 93.5% (death from lung cancer). Although subsequent primary lung cancer is troublesome, operation is a reliable treatment Second surgical intervention for recurrent and second primary bronchogenic carcinomas.

Second surgical intervention for recurrent and second primary bronchogenic carcinomas

Watanabe-Y; Shimizu-J; Oda-M; Tatsuzwa-Y; Hayashi-Y; Iwa-T

Scand-J-Thorac-Cardiovasc-Surg. 1992; 26 (1): 73-8

Second operations were performed in 1961-1990 on 23 patients with non-small cell bronchogenic carcinoma, constituting 2.5% of 906 who had undergone pulmonary resection for such tumor and 3.6% of the 641 with apparently curative surgery. The second operation was performed for recurrent tumor in 15 cases and for second primary tumor in eight. Five-year survival after the first operation was 30% in the former group and 88% in the latter (significant difference). Among the total 23 patients, this survival rate was 51%. The study indicates that an aggressive attitude to second surgical intervention is warrnted. For early detection of second lesions, follow-up at maximally 6-month intervals should be continued for more than 5 years after the first operation.

Surgical treatment of stage III non-small cell bronchogenic carcinoma involving the chest wall.

Lopez-L; Lopez-Pujol-J; Varela-A; Baamonde-C; Socas-L; Salvatierra-A; Freixinet-J; Cerezo-F

Scand-J-Thorac-Cardiovasc-Surg. 1992; 26(2): 129-33

Thirty-five patients who had undergone surgery for non-small cell bronchogenic carcinoma with isolated involvement of the chest wall were reviewed. The diagnosis was preoperatively suspected in 80% of cases. En-bloc resection of the invaded chest wall was per formed in 25 cases and parietal pleurectomy in ten in which the pleura was easily dissectable from the costal plane. Of the eight patients with major complications in the early postoperative period, six, including the two who died perioperatively, had undergone enblock resection. The 5- year actuarial survival rate was 22% overall and 36% in the patients without lymph node involvement. No significant relationship between survival and type of operation or degree of chest wall invasion was found. Isolated involvement of the chest wall by non-small cell bronchogenic carcinoma does not necessarily contraindicate surgery with curative intent. Parietal pleurectomy is valid in selected cases. Long-term survival depends basically on node involvement.

The place for bilobectomy in bronchogenic carcinoma.

DeneuvilleOM; Regnard-JF; Coggia-M; Rojas-Miranda-A; Darteve-elle-P; Levasseur-P

Eur-J-Cardiothorac-Surg. 1992; 6(8): 446-51

From 1978 to 1988, 148 bilobectomies (21 upper and middle and 127 lower and middle) were performed for bronchogenic carcinoma. A conservative procedure was mandatory in 29 patients in whom a pneumonectomy was not functionlly feasible while bilobectomy was deliberately performed in 119 patients with near normal lung function. Overall mortality was 6% compared to 4% and 3%, respectively, following pneumonectomies and lobectomies. Preoperative functional status did not significantly influence mortality. The complication rate was 55%. The incidence of bronchopleural fistula electively observed after lower and middle lobe resection was significantly higher (11%) compared to 4% after pneumonectomy and 1.4% after lobectomy (P less than 0.01). The overall 5-year survival was 43% and was similar to that observed at comparable TNM stage after other pulmonary resections. Residual right pulmonary function demonstrated by perfusion isotopic scan was 24% +/- 10 in 21 long- term survivors. These results indicate that bilobectomy can reasonably be considered in patients requiring more than a lobectomy but in whom lung conservation is mandatory despite a significant increase in morbidity. The lisk appears justifiable regarding late survival results and functional benefit of the remaining right lobe.

The value of selective mediastinoscopy in predicting resectbility of patients with bronchogenic carcinoma.

Maggi-G; Casadio-C; Giobbe-R; Cianci-R; Ruffini-E; Oliaro-

Int-Surr. 1992 Oct-Dec; 77(4): 280-3

From 1980 to 1990, 1505 patients underwent thoracotomy as definitive treatment for non small cell lung cancer. Computed tomography (CT) of the chest has been used rountinely since 1984 for assessment of mediastinal lymph node involvement. A total of 235 cervical medistinoscopies and 71 anterior parastermal mediastinotomies were performed on the same patient population as preoperative staging when CT scan demonstrated mediastinal lymph nodes larger than 1 cm. Radical resections showed a constant increase in number from 70.1% in the period 1980-84 to 82.7% in the period 1985-90. Exploratory thoracotomies and thoracotomies with residual tumor showed a parallel reduction: 14.5% in 1980-84 to 7.4% in 1985-90 for the former, 15.4% in 1980-84 to 9.8% in 1985-90 for the latter. The percentage of N2 disease to

the total number of thoracotomies decreased from 23.6% in 1980-81 to 11.2% in 1989-90. We conclude that a selective use of cervical mediastinoscopy and anterior parasternal mediastinotomy, based upon the results of CT scan, may have contributed to reduce the number of exploratory thorcotomies and thoracotomies with residual tumor.

Tumor fixation of bleomycin labeled with 57 cobalt

Homassn-JP; Peching-A; Roden-S; Angebault-M; Bonniot-JP

Cryobiology. 1992 Oct; 29(5): 543-8

The combined effect of cryotherapy and chemotherapy was studied in 12 patients with bronchial carcinoma. Radiolabeled (57 Co) Bleomycin (BLM) was injected intravenously and initial detection was carried out with a gamma- camera. Plasma half- life and clealance of 57 Co- BLM, as well as tumor/normal tissues ratios were calculated. The same protocol was performed 15 days later immediately after cryotherapy. A mean increase of 30% of radiolabeled BLM was found in the tumor area after cryotherapy, and phannacokinetic data were significantly different after cryotherapy. The vascular component of cryodestruction offers an explnation for these results, with trapping of the anticancer drug in the tumor and immediately surrounding area due to vascular stasis. It seems that chemotherapy may be more effective after cryotherpy, and a multicenter study is in progress to evaluate the association of cryochemotherapy in France.

Wedge resection for bronchogenic carcinoma in high-risk patients.

Temeck-BK; Schafer-PW; Saini-N

South-Med-J. 1992 Nov; 85(1): 1081-3

At the Veterans Administration Medical Center in Washington,

DC, 73 patients with bronchogenic carcinoma had pulmonary wedge resection from February 1967 to March 1988, with a 1.4% perioperative mortality and a 4.1% morbidity. Mean age of the patients was 63 years. Patients were considered poor risk with a mean Goldman index of 9 +/- 4 (class II), mean ASA physical status classification II, mean I-second forced expiratory volume (FEVI) of 1.25 liters (42% predicted), ratio of FEVI to forced vital capacity 30% predicted, and maximum voluntary ventilation 24% predicted. Staging of the bronchogenic carcinomas indicated 68% stage I, 15% stage II, and 17% stage III, and history showed 41% epidermoid, 40% adenocarcinoma, 12% bronchoalveolar, 3% large cell, and 4% small cell type. For the 72 patients eligible for follow-up data were available on 62 for a period ranging from 4 months to 15 years. Survival was 94% at 1 year, 55% at 3 years, 29% at 5 years, 5% at 10 years, nd 2% at 15

years. Within 5 years, 21% of the patients had died of causes other than bronchogenic carcinoma. The rate of recurrence was 16%. Analysis by each stage of lung cancer showed local recurrence in 4% of patients with stage I disease, in 9% of those with stage II disease, and in 59% of those with stage III disease. We conclude that wedge resection provided acceptable surgical treatment in a group of high risk surgical patients.

Carinal resection for bronchogenic carcinoma.

Mthisen-DJ; Grillo-HC

J-Thorac-Cardiovsc- Surg. 1991 Jul; 102(1): 16-22 discussion 22-3

Techniques are available for carinal resection and reconstruction for bronchogenic carcinoma involving the carina. Successful outcome depends on careful patient selection, thorough preoperative evaluation, careful anaesthtic management, strict attention to Surgical technique, and compulsive postoperative care. Since 1973 we have performed 37 carinal resections for bronchogenic carcinoma: 21 right carinal pneumonectomies, 7 carinal resections, 7 carina plus lobe resectins, and 2 carina plus pneumonectomy stump resections. Five patients had diseased N2 nodes and 13 patients had diseased NI nodes. Complications included pulmonary (8), vocal cord paresis (3), atrial fibrillation (9), anastomotic stenosis (4), and anastomotic separation (3). There were 3 early postoperative deaths (8%). All were related to adult respratory distress syndrome and were unresponsive to aggressive treatment. There were 4 late postoperative deaths between 2 and 4 months (10.9%). All late postoperative deaths were related to anastomotic complications (stenosis [1] and separation [3]). There are 5 absolute S-year SUI: vivors and an actuarial S-year survival rate of 19%.

Mediastinoscopy as a predictor of resectability in patients with bronchogenic carcinoma.

Riordin-DS; Buckley-DJ; Aherne-T

Ir-J Med-Sci. 1991 Sep; 160(9): 29-2

Eighty consecutive mediastinoscopies, performed for assessment of patients with bronchogenic carcinoma, were reviewed with regard to accuracy and complications. Thirty patients had mediastinal lymph node metastases: 26 were considered inoperable and thus saved non- therapeutic thoracotomy, 4 were considered operable of whom 3 had a curative resection, and one was inoperable at thoracotomy. Of 50 patients with negative mediastinal nodes 43 had thoracotomy; 42 were resectable and one was unresectable. Seven did not have thoracotomy because of other contraindications. In total of 47 patients

undergoing thoracotomy on the basis of mediastinoscopy, 45 were resectable, giving mediastinoscopy a positive predictive value for resectability of 95.7%. There was no mortality and two superficial wound infections occurred giving a morbidity of 2.5%. Mediastinoscopy is a safe, reliable and accurate predictor or resectability in patients with bronchogenic carcinoma and continues to have a major role in the management of these patients.

Minimal resection for bronchogenic carcinoma. An update.

Crabbe-MM; Patrissi-GA; Fontenelle-LJ

Chest. 1991 Jun; 99(6): 1421-4

Minimal resection with curative intent was performed for 24 patients with stage I bronchogenic carcinoma at our institutions over a 12-year period. This was usually done for patients who could not tolerate more extensive resections. The five-year acturial survival rate was 65 percent. The rate of local recurrences was 13 percent (3/24), and the rate of distant recurrences was 17 percent (4/24), with a median follow-up of 38 months. Survival and recurrence rates are similar for patients undergoing minimal resection and those being reported for patients undergoing more extensive resections for stage bronchogenic carcinom. In selected patients, minimal resection should be considered as an acceptable alternative treatment for patients with stage I bronchogenic carcinoma.

Multimodality treatment for small cell bronchial carcinoma. Preliminary results of a prospective, multicenter trial. The ISC-Lung Cancer Study Group.

Ulsperger-E; Karrer-K; Denck-H

Eur-J-Carciothorac-Surg. 1991; 5(6): 306-9; discussion 310

Preliminary results of the 1984 ISC (International Society of Chemotherpy) lung cancer studies I and II as of June 1990 are based on 146 patients with small cell bronchial carcinoma from 23 departments of thoracic surgery. All patients received surgery for cure in cTNM stages I and II followed by randomization for two different types of chemotherapy. For disease- free patients after completion of postoperative chemotherapy prophylactic cranial irradiation (PCI) was administered. For the two different chemotherapeutic regimens, no statistically significant differences in survival (SVR) could be observed. Each patient was classified by the pTNM system. There were 63 patients with stage I, 44 patients with stage II and 38 patients with stage III disease. Four years after surgery,

63 patients with NO disease had a SVR of 50%, 51 patients with NI disese 31%, and 32 patients with N2 disease, 23%. No prolongation of brin-metastasis- free time for 62 patients receiving PCI was shown. It is concluded that initial surgical resection for small cell lung cancer in stages I and II followed by intensive chemotherapy is an appropriate therapeutic approach.

Multiple primary bronchogenic carcinomas: treatment and follow-up.

Fleisher-AG; McElvaney-G; Robinson-CL

Ann-Thorac-Surg. 1991 Jan; 51(1): 48-51

A second primary bronchogenic carcinoma subsequently developed 8 to 156 months later in 19 patients who underwent curative resection of primary bronchogenic carcinomas. The second primary tumor was treted by surgical resection in 9 patients, 3 patients' tumors were considered unresectable, and the remaining 7 patients, despite having potentially resectable tumors, did not undergo resection because of insufficient pulmonary reserve or unwillingness to undergo resection. Actuarial life- table analysis of survival for the 9 patients who underwent resection showed a median survival time of 110.3 months compared with 19 months for the group with unresected but resectable tumors and 10.5 months for the group with unresectable tumors. There was no operative mortality in the group with resected tumors. We conclude that in patients in whom a second primary carcinoma of the lung develops, surgical resection prolongs survival and can be performed with a low operative mortality

Results of resection for bronchogenic carcinoma with mediastinal lymph node metastases in selected patients.

Regnard-JF; Mgdeleinat-P; Azoulay-D; Dartevelle-P; Deneuville-

Eur-J-Cardiothorac-Surg. 1991; 5(11): 583-6; discussion 587

Between 1982 and 1988, 254 consecutive patients underwent resection for bronchogenic carcinoma with mediastinal lymph node metastases at Marie Lannelongue Hospital. Selection of cases for surgery was carried out using CT and mediastinoscopy. The surgical procedure performed were pneumonectomy (169), lobectomy (65), or bilobectomy (20) associated with resection of ipsilateral mediastinal lymph nodes. Almost all diseased nodes appeared grossly enlarged at surgery and only a few were of normal size. Postoperative mortality was 5.6%. Resection was potentially curative in 191 cases (75%)

and palliative in 63 cases (25%). Almost all patients received adjuvant treatment (mainly radiotherapy). Actiarial 5-year survival was 18% for the entire group, and 23% for those who underv[illegible] curative resection. No patient with palliat[illegible]ection survived 5 years. The following factors proved to be significantly associated with a better prognosis: complete resection, independent lymph node metastases, involvement of only one level, lower paratracheal involvement. On the other hand, there was no difference between pathological types (squamous cell carcinoms) with regard to prognosis. We advocate an aggressive approach in selected cases of N2 bronchogenic carcinoma. Neoadjuvant chemotherapy should be tested in these specific patients with a view to the possibility of improving results.

Simultaneous pneumonectomy and esophagectomy for bronchial carcinoma.

Prauer-HW; Barthlen-W; Siewert-JR

Eur-J-Cardiothorac- Surg. 1991; 5(6): 334-5 Combined-

Two cases of bronchial carcinoma with oesophageal involvement are presented. Both were treated by simultaneous pneumonectomy and oesophagectomy. The postoperative course of each patient was complicated by secondary infection of the pneumonectomy space. One patient expired from a recurrent intratracheal tumour on the 83 rd postoperative day and the other remains tumour free 3 years after treatment. As few therapeutic alternatives exist for non small cell bronchial carcinoma, primary radical surgical treatment should be considered, even in advanced cases where the tumour invades the oesophagus.

Bronchial stump recurrence after surgery for bronchial carcinoma.

Verleden-G; Deneffe-G; Demedts-M

Eur-Respir-J. 1990 Jan; 3(1): 97-100

In 10 out of 295 patients (3.4%), followed-up after radical resection for non-small cell bronchial carcinoma in the period from 1980 until 1986, broachial stump recurrence developed. A good relationship was found between relapse time (4-52 months) and distance between the primary tumour and bronchial resection line (1-7 cm) (i.e. 5-8 months. cm-I) in 8 of the patients (p less than 0.01). The mean survival time after detection of the recurrence was 10 months (range 1-15 months), and was not clearly influenced by the therapy applied (resurgery, chemotherapy, radiation), nor by the TNM stage of the bronchial stump recurrence.

Bronchogenic carcinoma treated by concomitant resection of lung and chest wall.

Warner-R; Ball-SK; Dalton-ML

South-Med-J. 1990 Jun; 83(6): 62-3, 633

Chest wall invasion by bronchogenic carcinoma is found in 5% of all cases of pulmonary carcinoma. During the last 3 years, 11 cases of lung cancer with chest wall involvement have been encountered at the Jackson Veterans Administration Medical Center. We reviewed these cases to reassess the role of concomitant resection of the lung and chest wall. From this experience, we have concluded that (1) chest wall involvement is potentially curable; (2) chest wall resection adds little if any morbidity to the prcedure; (3) resections of fewer than four ribs usually require only soft tissue covetage, without a prosthesis; (4) patients with squamous cell cancer have longer survival; (5) chest wall resection is highly effection in the relief of pain due to invasion of the chest wall; and (6) survival is greater than in other stage III lung carcinomas and is more closely related to nodal involvement than to chest wll invasion.

Bronchoscopic cryotherapy for advanced bronchial carcinoma

Walsh-DA; Maiwand-MO; Nath-AR; Lock-wood-P; Lioyd-MH; Saab-M

Thorax. 1990 Jul; 45(7): 509-13

A prospective study was carried out to assess the value of bronchoscopic cryotherpy for palliation of inoperable bronchial carcinoma with bronchial obstruction. Symptoms, lung function, and chest radiographic and bronchoscopic findings were recorded serially before and after 81 cryotherapy sessions in 33 consecutive patients. Most patients improved in terms of overall symptoms, stridor, and haemoptysis and they had an overall improvement in dyspnoea. Objective improvement in lung function was seen in 58% of patients and the changes in lung function correlated with symptoms. Bronchoscopic evidence of relief of bronchial obstruction was seen in 77% of patients and 24% showed improvement in degree of collapse on the radiograph. There were no important complications. These results compare favourbly with the results in published series of patients having laser therapy. It is concluded that bronchoscopic cryotherapy is valuaable for the palliation of inoperable bronchial carcinoma.

Determination of operability in candidates who undergo lung resection for bronchogenic carcinoma.

Murphy-TP; Casey-MT

Can-J-Surg. 1990 Dec; 33(6): 470-3

In this prospective study the authors attempted to determine the effect of lung resection for bronchogenic carcinoma on final pulmonary function in patients who had severe limitation of lung air flow preoperatively and were therefore likely to have severe, progressive pulmonary failure and in those who had acceptable pulmonary function preoperatively. Preoperative and postoperative pulmonary function tests were performed on 20 patients chosen to undergo various types of resection for bronchogenic carcinoma. Those who underwent pneumonectomy had changes in lung volume that were expected for a resection of that maagnitude. Patients who underwent lesser resections had more variable postoperative lung volumes and flows. The patients whose preoperaative pulmonary function was poorest had the least change postoperatively and even, in some cases, showed some improvement in function, yet they were the ones most likely to be denied surgery, because of their poor preoperative pulmonary function.

En bloc resection for bronchogenic carcinoma with chest wall invasion. Value of pre-operative radiotherapy.

Carrel-T; Nchbur-B; Veraguth-P

Eur-J-Cardiothorc-Surg. 1990; 4(10): 534-7

A small number of patients with lung cancer will have a tumour invading the chest wall. Pre-operative radiotherpy and surgical resection provide the best results in patients with Pancost's tumours, although chest wall invasion is often considered to indicate incurability. We reviewed the outcome in 46 patients with bronchogenic carcinoma and non- apical chest wall invasion and have tried to clarify the role of adjuvant pre- opertive radiotherapy. All patients underwent combined chest wall and lung resection for treatment of lung cancer which had extended grossly and microscopically into the chest wall. In this retrospective study, we identified two groups of patients, those (n = 21) who received and those (n=25) who did not receive pre- operative radiotherapy. Curative resection had been possible in 80% of the patients. There was one early post- operative death, due to pneumonia. The survival in all 46 patients is 32% at 5 years. In the most favourable cases, those without nodal involvement and who received pre- operative radiotherapy, the 5-year survival is 56%. In our series, there was a notable difference in 5-year survival between at every stage of disease.

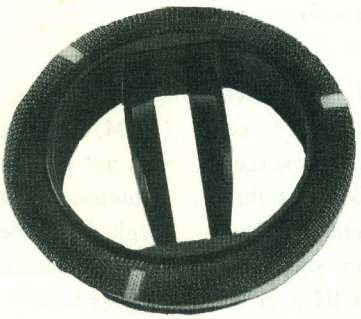
The role of adjuvant surgery in the combined modality therapy of small-cell bronchogenic carcinoma after a chemotherapy- induced partial remission

Holoye-PY; McMurtery-MJ; Mountain-CF; Murphy-WK; Dhin-gra-HM; Umsawsdi-T; Glisson-BS; Lee-JS; Crr-DT; Valdivieso-M; et-al

J-Clin-Oncol. 1990 Mr; 8(3): 416-22

Twenty-six patients with a limited-disease presentation of small cell bronchogenic carcinoma (SCBC) had surgery after achieving a partial remission with three cycles of chemotherapy. Persistent SCBC was found in 15 patients (58%), non- small- cell bronchogenic carcinoma (NSCBC) in six patients (23%), and no malignancy in five patients (19%). Twelve patients have died since surgery. Tumor- node- metastasis (TNM) staging prior to or after chemotherapy was not predictive of outcome, but an NO status found at pathological examination of the surgical specimen was predictive of long- term survival. Median survival for this group of patients was 25 months. Adjuvant surgery is feasible and may be beneficial.

CarboMedics Offers More Options For The Small Aortic Root.



CarboMedics Reduced "R" Series Valve
for intra-annular placement

CarboMedics "Top Hat" Supra-Annular Valve
for increased orifice area

Decide For Yourself.

CarboMedics
A company of SULZER medica

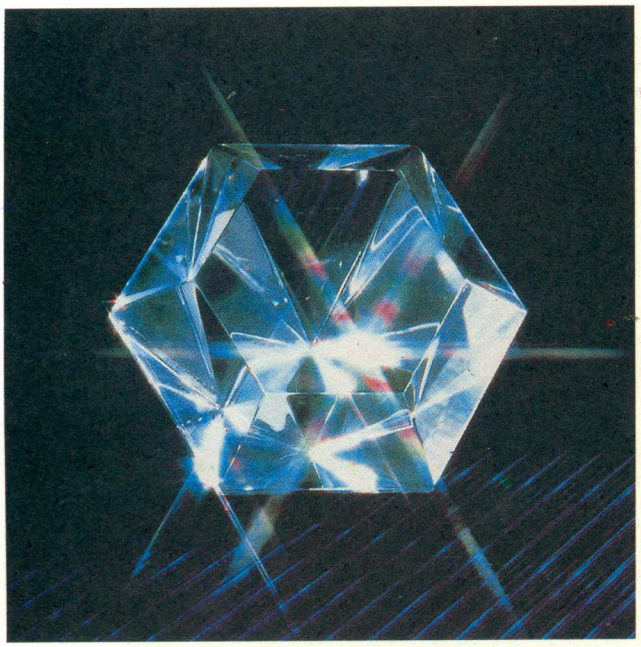
1300 East Anderson Lane, Austin, Texas, USA 78752 (512) 873-3200 FAX (512) 873-3350

For more information, call CarboMedics at (800) 289-5759.

HYPERTENSION

Well-being

the key to success



- Well-being with Natrilix is a spontaneously reported subjective sensation demonstrated by large multicentre studies (1, 2).
- Well-being with Natrilix is also related to its simple and once-daily dosage, and its excellent clinical and biological tolerance.
- Well-being with Natrilix is thus the guarantee of a better adherence of the patient to treatment and the guarantee of a high therapeutic success rate maintained over the long term (3).

(1) Wheeley M. St G. et al. (1982) Pharmatherapeutica, 3 (2): 143-152. (2) Watters K., Campbell D.B. (1986), Concilia Medica, 1 (3): 33-41. (3) Vukovich R.A. et al. (1983), CMRO, 8 (suppl. 3): 109-122.

NATRILIX

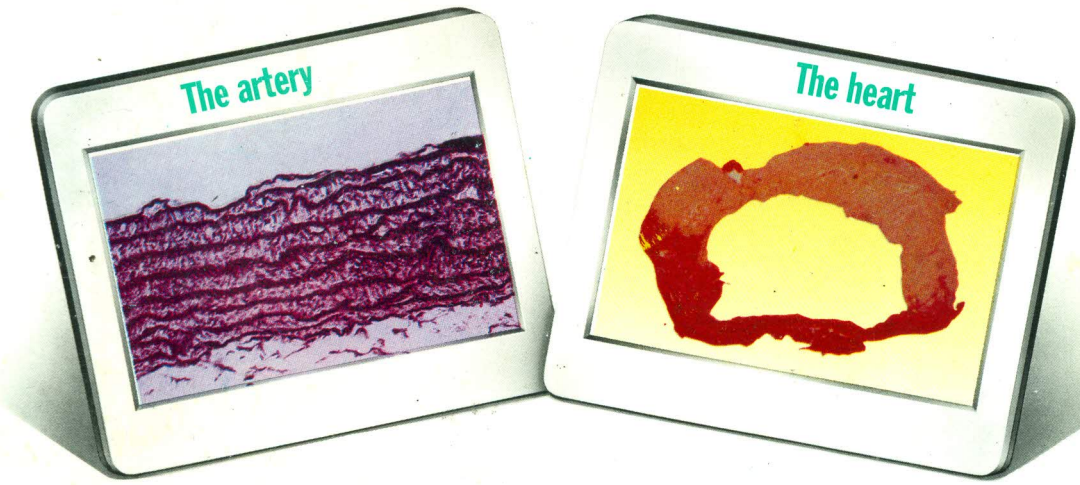
INDAPAMIDE

1 tablet daily

Indication: Essential hypertension. **Contra-indications:** Severe hepatic insufficiency, recent cerebrovascular accidents. **Associated treatments:** In view of its mode of action, Natrilix may be combined with any non-thiazide anti-hypertensive agent. Drug combinations to be avoided: tienilic acid, lithium, fenoxidil, lidoflazine, prenylamine, vincamine. **Precautions:** Monitoring of potassium and uric acid serum levels is recommended especially in subjects with a predisposition or a sensitivity to hypokalemia, and in patients with gout. **Side-effects:** Hypokalemia, fatigue, orthostatic hypotension, allergic manifestations. **Dosage and administration:** One tablet daily. **Composition:** Each coated tablet contains 2.5 mg of 1-(4 chloro-3-sulfamyl-benzamido)-2-methyl-indoline (or indapamide). **Presentation:** Box of 30, 60 or 100 coated tablets.

LES LABORATOIRES SERVIER. Gidy, 45400 Fleury-les-Aubrais - France
Correspondent: Développement International Servier - 24, rue du Pont, 92200 Neuilly-sur-Seine - France

Scoring an ACE in cardiovascular remodeling



1 tablet daily

COVERSYL® 4 mg

PERINDOPRIL

in hypertension and heart failure...

...cardiovascular remodeling is a key pathogenic feature. Coversyl 4 mg actively combats cardiovascular remodeling by correcting the structural and functional alterations of the heart and artery.[1-4]
Coversyl 4 mg is a true once daily ACE inhibitor with guaranteed antihypertensive efficacy right up to 24 hours post dose.[5]
In heart failure, Coversyl 4 mg (half-a-tablet) offers a safer start to treatment thanks to an absence of significant hypotensive first-dose response.[6]
With its original properties, Coversyl 4 mg is a high-performance ACE inhibitor in both its indications.

1. SIHM I et al. *Eur Heart J.* 1993; 14(suppl) : 63 - 2. LEVY BI et al. *Circ Res.* 1988; 63 : 227-239 - 3. ASMAR RG et al. *J Hypertens.* 1988; (suppl 3) : S33-S39 - 4. MICHEL JB et al. *Circ Res.* 1988; 62 : 641-650
5. MORGAN TO et al. *Am J Hypertens.* 1993; 6 : 116 A - 6. MAC FADYEN RJ et al. *Br Heart J.* 1991; 66 : 206-211.

Coversyl is a long-acting ACE inhibitor. **International nonproprietary name** : Perindopril. **Indications :** Essential hypertension. Congestive heart failure (adjunctive therapy). **Dosage and administration :** *Hypertension* : 4 mg once a day in the morning. If necessary, the dose may be increased to 8 mg after one month of treatment. Coversyl should be taken before food. *Congestive heart failure* : Coversyl should be started under close medical supervision at a starting dose of 2 mg in the morning. This may be increased to 4 mg once blood pressure acceptability has been demonstrated. *Elderly patients* : start treatment at 2 mg daily. **Contraindications :** Children. Pregnancy. Lactation. Patients with a history of hypersensitivity to Coversyl. **Precautions :** Assess renal function before and during treatment where appropriate. Renovascular hypertension. Surgery/Anesthesia. Renal insufficiency : the dose should be cautiously adjusted in accordance with the creatinine clearance *(refer to complete data sheet)*. Symptomatic hypotension is rarely seen, but is more likely in volume-depleted patients, those receiving diuretics, or with the first two doses. In diuretic-treated patients, stop the diuretic 3 days before starting Coversyl. A diuretic may later be given in combination if necessary; potassium-sparing diuretics are not recommended. Combination with neuroleptics or imipramine-type drugs may increase the hypotensive effect. Serum lithium concentrations may rise during lithium therapy. **Side effects :** Rare and mild, usually at the start of treatment. Cough, fatigue, asthenia, headache, disturbances of mood and/or sleep have been reported. Less often, taste impairment, epigastric discomfort, nausea, abdominal pain, and rash. Reversible increases in blood urea and creatinine may be observed. Proteinuria has occurred in some patients. Rarely, angioneurotic edema and decreases in hemoglobin, red cells, and platelets have been reported. **Composition :** Each tablet contains 4 mg of the tert-butylamine salt of perindopril. **Presentation :** Packs of 30 tablets of Coversyl 4 mg (scored). *Refer to data sheet for complete prescribing information.*

Les Laboratoires Servier, 45520 Gidy - France. Correspondent : **Servier International,** 6, place des Pléiades, 92415 Courbevoie Cedex - France.

SERVIER

94 CO 388 IA