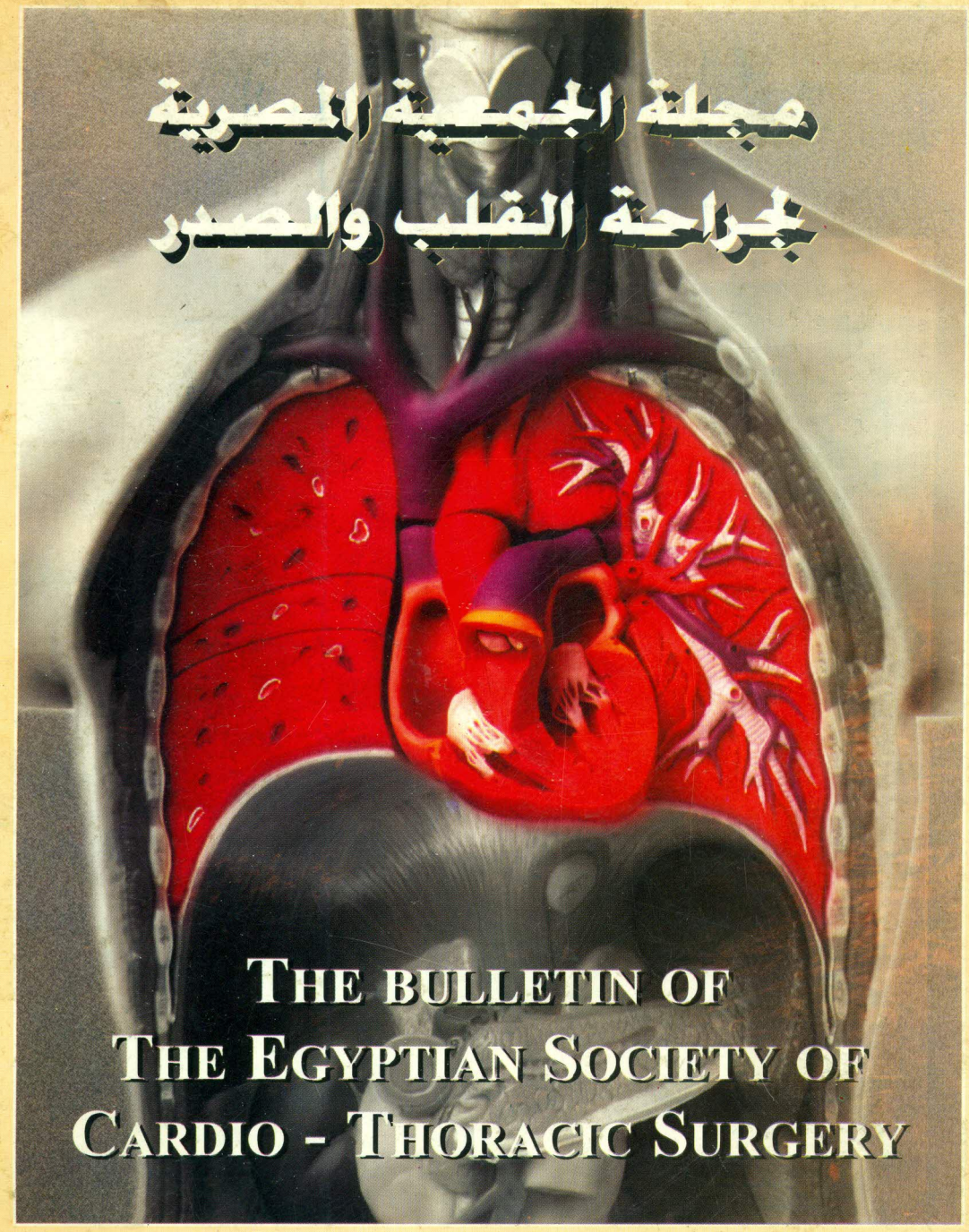
Vol. 2, January 1995

العدد الثانى ، يناير ١٩٩٥

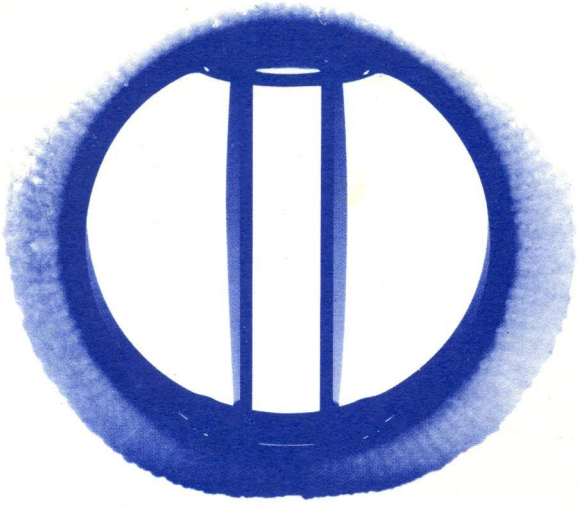


مجلة الجمعية المصرية لجراحة القلب والصدر

THE BULLETIN OF THE EGYPTIAN SOCIETY OF CARDIO - THORACIC SURGERY

Editor
Hassouna Saba F.R.C.S.

ST. JUDE MEDICAL



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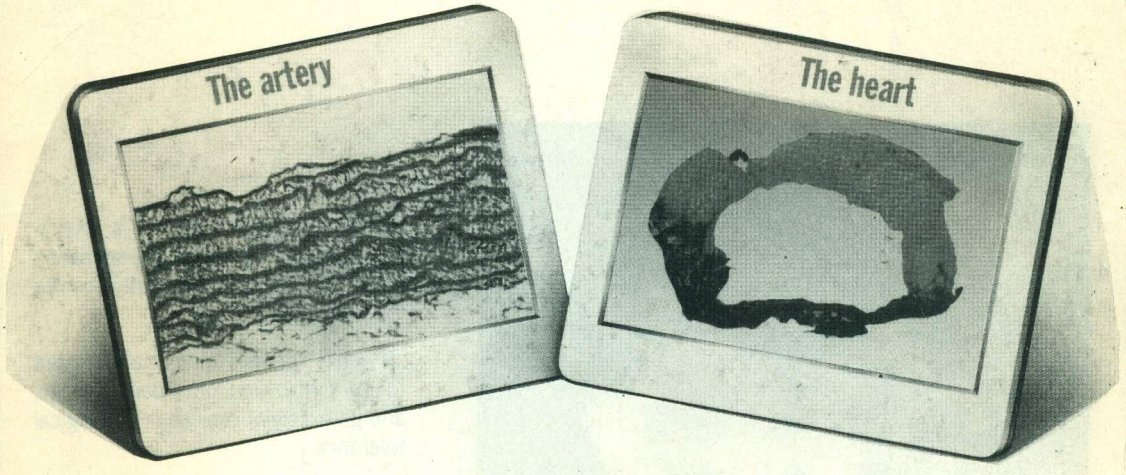
Scoring an ACE in cardiovascular remodeling



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.

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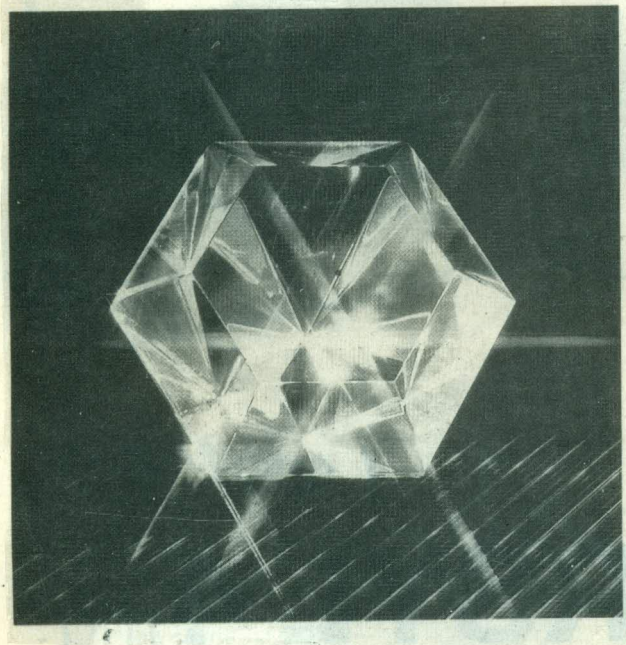
HYPERTENSION

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 excelient 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.

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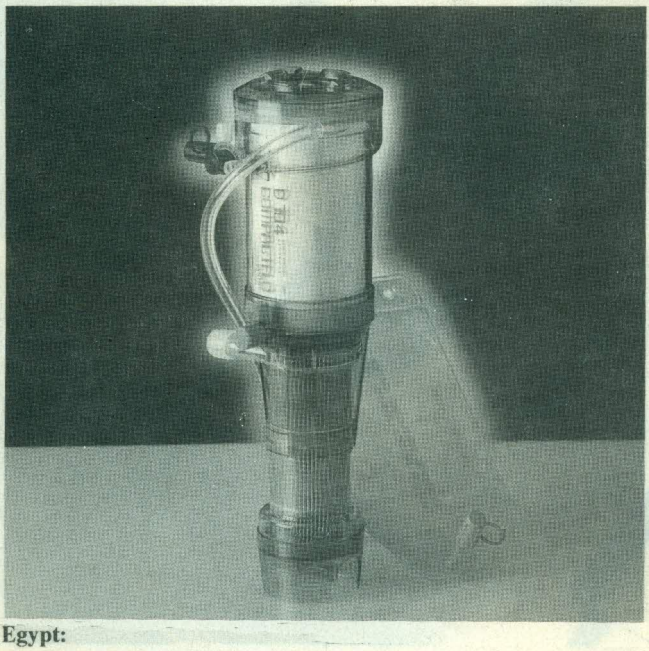

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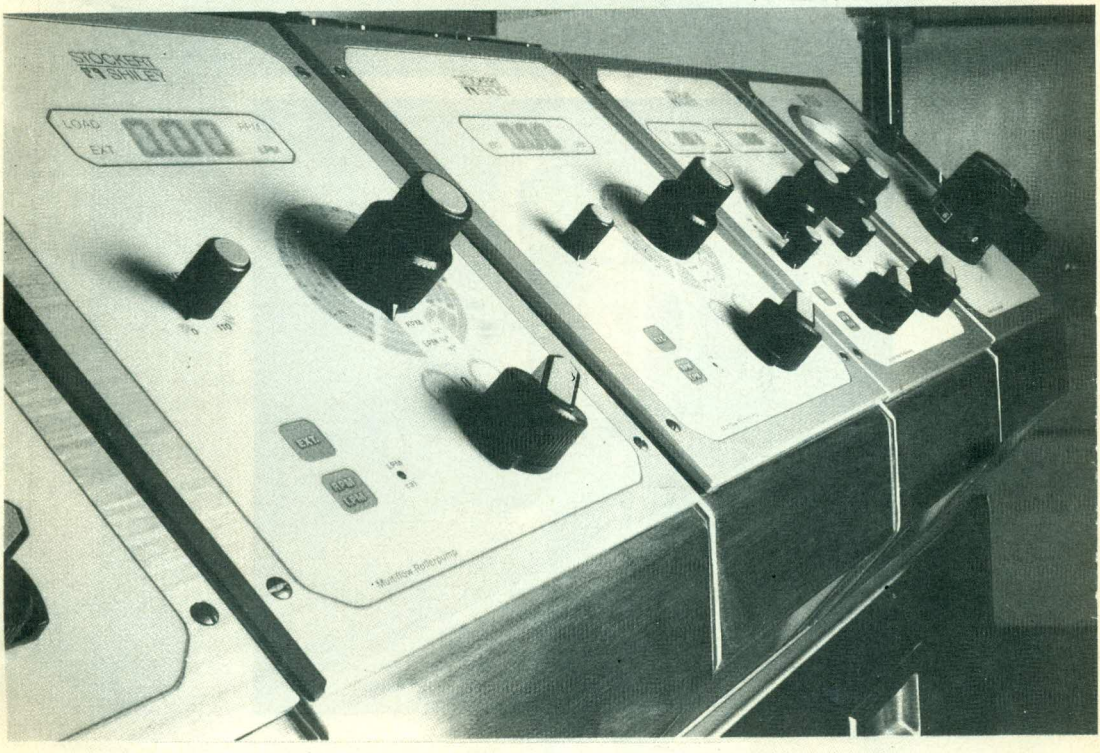
Heart-Lung Machines in a Modular System



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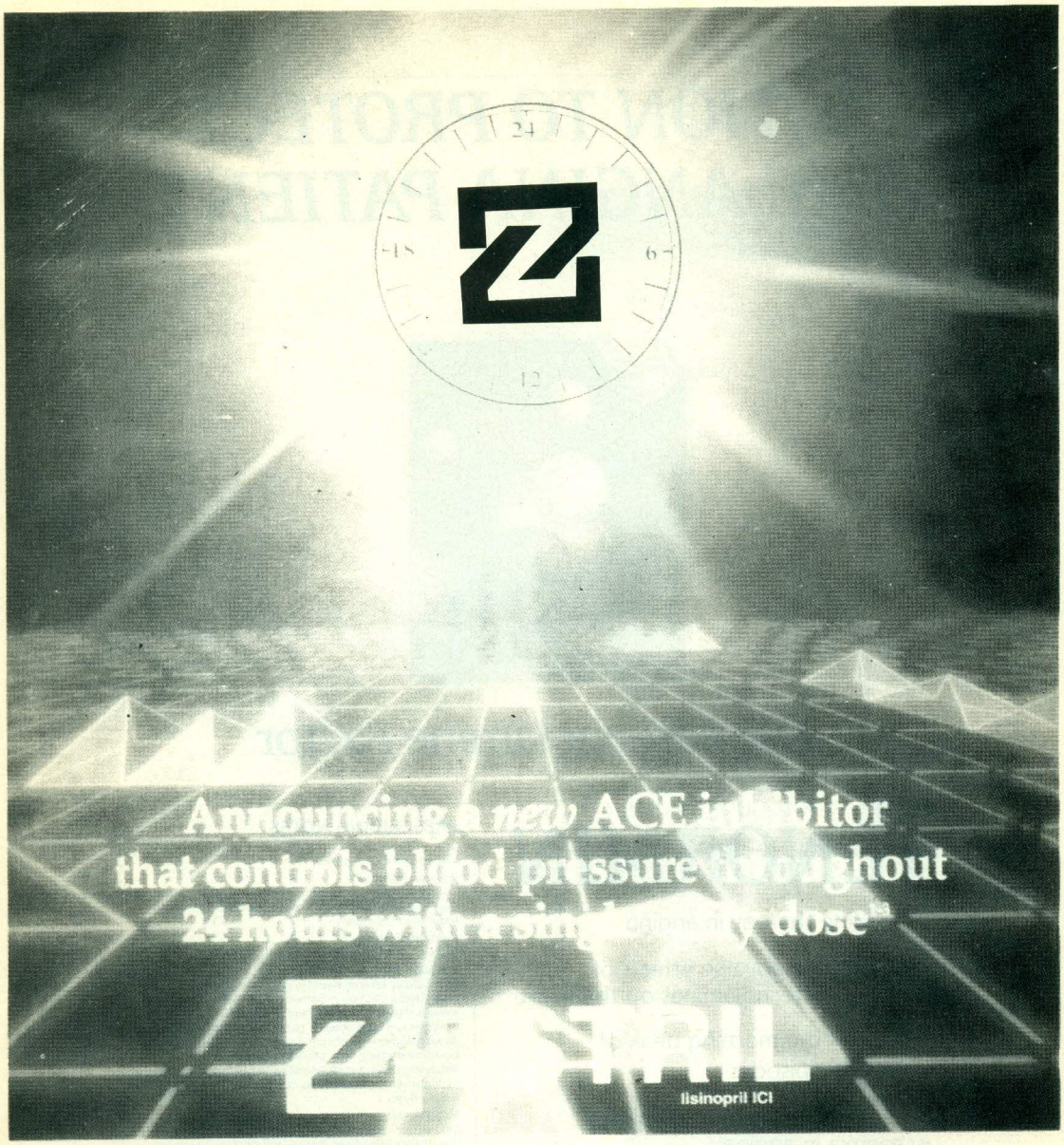

Prescribing Notes

Uses Essential and renovascular hypertension. Congestive heart failure (CHF) as an adjunct to digitalis and/or diuretics.

Dosage *Hypertension*: Usual maintenance dose: 20 mg once daily. If possible discontinue prior diuretic therapy. 10 mg once daily can be used as a starting dose. Lower starting dose required in patients with renal impairment, renovascular hypertension, volume or salt depleted patients, when diuretics cannot be discontinued and some elderly patients. Adjust dosage according to blood pressure response. Maximum 80 mg/day. *CHF*: 5 to 20 mg once daily, starting dose 2.5 mg.

Contra-indications Hypersensitivity to any component of this product. Angioneurotic oedema associated with ACE inhibitors.

Precautions Symptomatic hypotension may occur, particularly in volume depleted patients and congestive heart failure. Caution in patients with ischaemic heart or cerebrovascular disease. Patients with a history of angioedema may be at increased risk of angioedema with an ACE inhibitor. Cough has been reported with ACE inhibitors. Renal impairment (usually reversible) may occur in some CHF patients who experience hypotension on starting 'Zestril'. Hypotension may occur during surgery/anaesthesia. Use in pregnancy not recommended. Caution in nursing mothers. No paediatric experience. The antihypertensive effect of 'Zestril' and diuretics is usually additive. Symptomatic hypotension can be minimised by discontinuing diuretic prior to 'Zestril'. The use of potassium sparing diuretics, potassium supplements, potassium containing salt substitutes, indomethacin and lithium.

Side effects Mostly mild and transient: dizziness, headache, diarrhoea, fatigue, cough, nausea, rash, orthostatic effects, asthenia. Angioneurotic oedema also rarely reported; discontinue 'Zestril' promptly. Side effects which have been reported rarely include myocardial infarction or cerebrovascular accident possibly secondary to excessive hypotension in high risk patients, palpitation, tachycardia, abdominal pain, dry mouth, hepatitis, jaundice, mood alterations, mental confusion, urticaria, diaphoresis, uraemia, oliguria/anuria, renal dysfunction, acute renal failure, impotence, a symptom complex which may include fever, vasculitis, myalgia, arthralgia/arthritis, positive ANA, elevated ESR, eosinophilia and leukocytosis. Rash, photosensitivity, or other dermatological manifestations may occur. Increases (usually reversible) in blood urea, serum creatinine, liver enzymes and serum bilirubin. Small decreases in haemoglobin and haematocrit have occurred. Hyperkalaemia.

Presentation Tablets, containing 5, 10 and 20 mg of lisinopril as the dihydrate.

Consult the full prescribing information before prescribing.

Not all strengths are available in all countries.

References

1. Herpin D, Conte D. *J Hum Hyperten* 1989; **3:** 11-15.
2. Whelton A *et al. J Clin Pharmacol* 1990; **30:** 1074-1080.
3. Whelton A *et al. J Hum Hyperten* 1992; **6:** 325-331.

'Zestril' is a trade mark

Further information is available on request

ICI **Pharmaceuticals**

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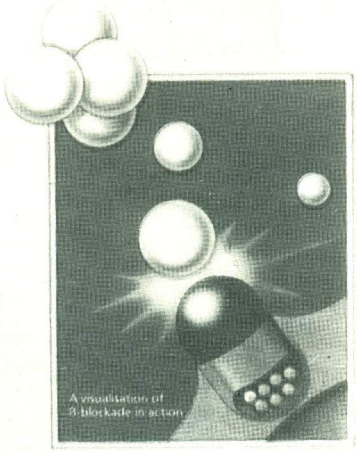
ACTION TO PROTECT THE ANGINA PATIENT



The Catecholamine Factor

CARDIOPROTECTIVE

TENORMIN

- A first-line therapy in angina
- Counters the adverse effects of excessive catecholamines on the heart
- Abolishes the morning peak of ischaemic episodes[1]
- Controls both silent and painful ischaemia in angina[1,2]
- Well tolerated – a factor in quality of life

ZENECA Pharmaceuticals

Alderley House, Alderley Park,
Macclesfield, Cheshire, England.

PRESCRIBING NOTES

Use:
Management of angina pectoris.

Dosage: (Adults):
Usually 100 mg once daily or 50 mg twice daily. Adjust dose in renal impairment. Dosage requirement may be reduced in the elderly.

Contraindications:
Second or third degree heart block, cardiogenic shock.

Precautions:
Poor cardiac reserve. Avoid in overt heart failure. Anaesthesia. Caution in patients with chronic obstructive airways disease or asthma. Withdrawal of beta-blocking drugs should be gradual in patients with ischaemic heart disease. Withdrawal of clonidine. Pregnancy and lactation. Co-administration with verapamil or Class I antidysrhythmic agents. Modifies the tachycardia of hypoglycaemia. If symptoms due to slow heart rate, reduce dose.

Side effects:
Cold extremities, muscular fatigue, bradycardia. Sleep disturbance rarely seen. Rashes and dry eyes have been reported with beta-blockers – consider discontinuance if they occur.

Presentation:
'Tenormin' tablets each containing atenolol 100 mg and 50 mg.

REFERENCES

1. Lancet 1988; **2**: 755.
2. Am J Cardiol 1987; **60**: 36A.

Consult full prescribing information before prescribing.

'Tenormin' is a trademark.
Further information is available on request.

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REMODELING ANNULOPLASTY

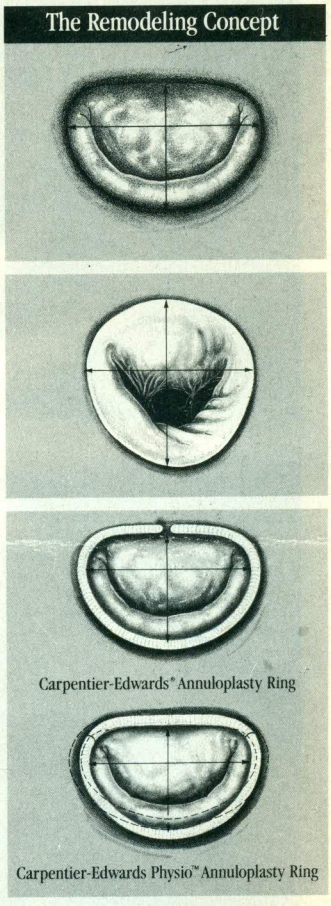
with the Carpentier-Edwards® Mitral Rings

The Carpentier-Edwards® Annuloplasty Ring and the Carpentier-Edwards Physio™ Annuloplasty Ring have been designed to remodel the annulus following mitral valve repair. The remodeling concept, built into the design of both rings, aims at restoring the size and shape of the normal mitral valve.

The normal mitral valve orifice is not circular, but is kidney-shaped with a transverse diameter 33% greater than the vertical diameter.

In a diseased mitral valve, this shape is modified. The annulus is deformed with the vertical diameter equal to or larger than the transverse diameter.

A remodeling annuloplasty involves restoring the physiologic annular ratio to provide maximum orifice area for optimal hemodynamic functioning. The key factor in valve reconstruction is to restore a normal systolic orifice configuration with the characteristic 3:4 ratio between the anteroposterior and the transverse diameters.



The Carpentier-Edwards® Annuloplasty Ring has proven predictability and stability over time, as reflected in the low incidence of reoperation and complications beyond 15 years. The Carpentier-Edwards Physio™ Annuloplasty Ring has the same remodeling design philosophy with a saddle-shaped anterior section and increased posterior flexibility for more natural physiologic functioning.

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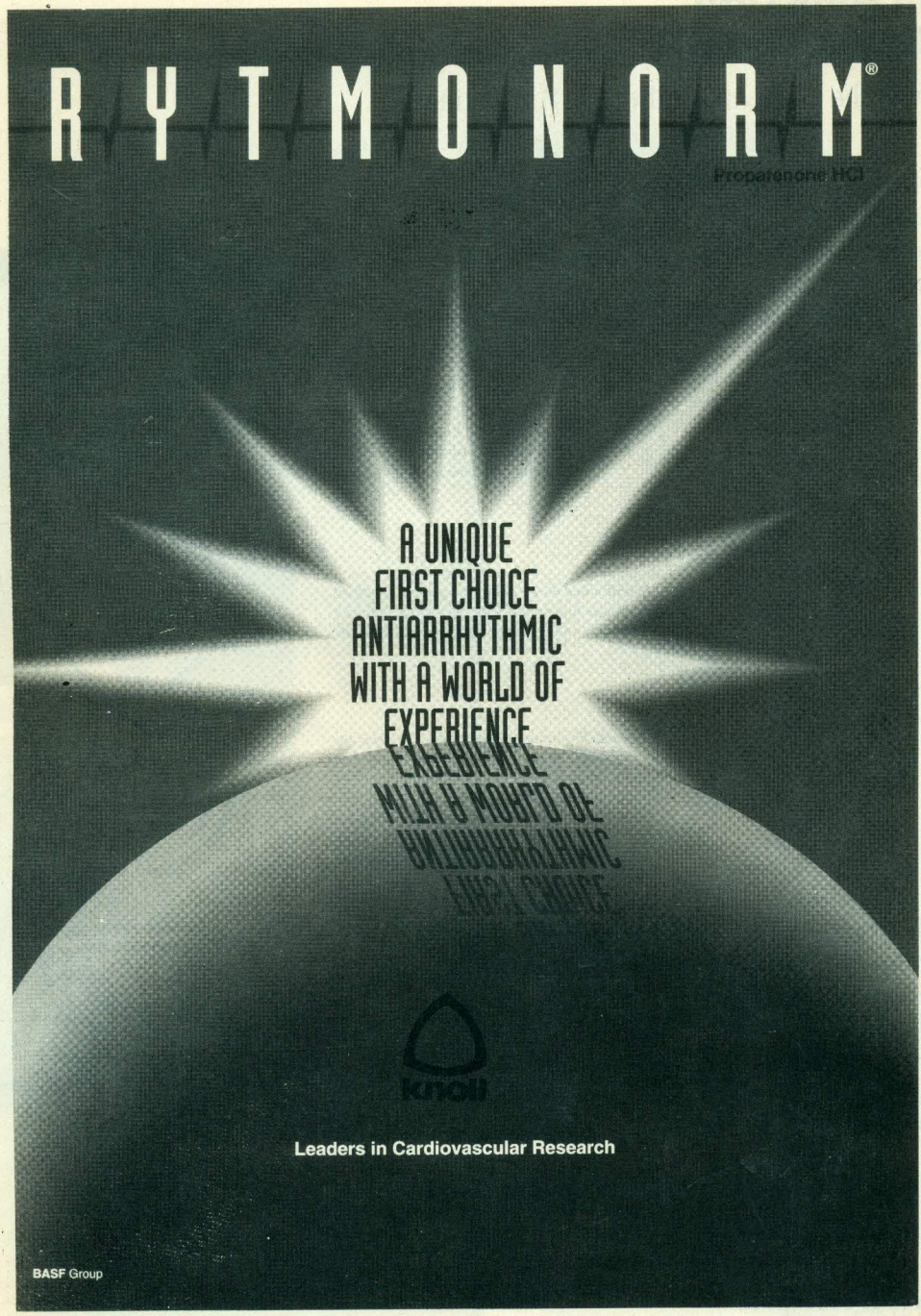

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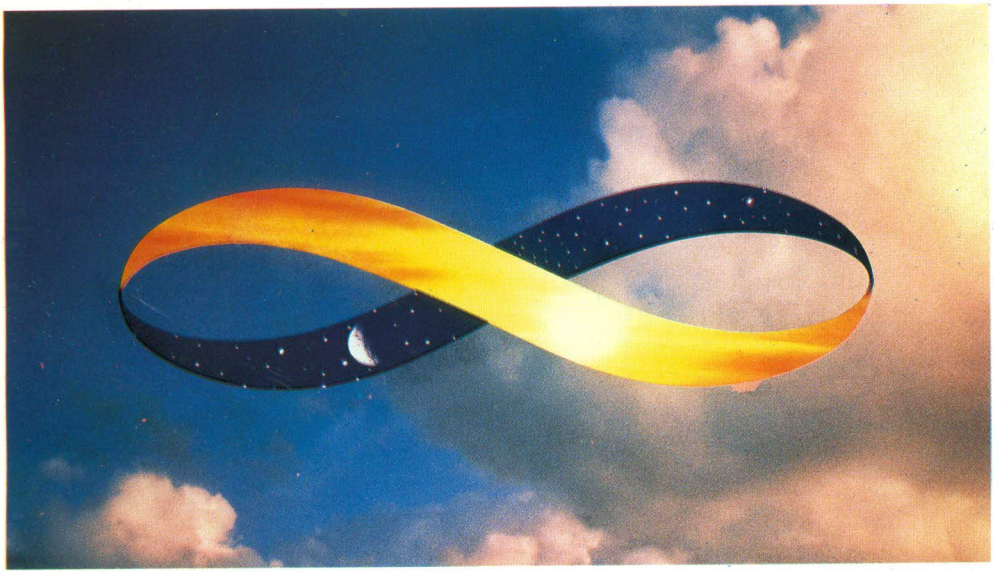
Telefax: (202) 3498308



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
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* 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% [11,12]. 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

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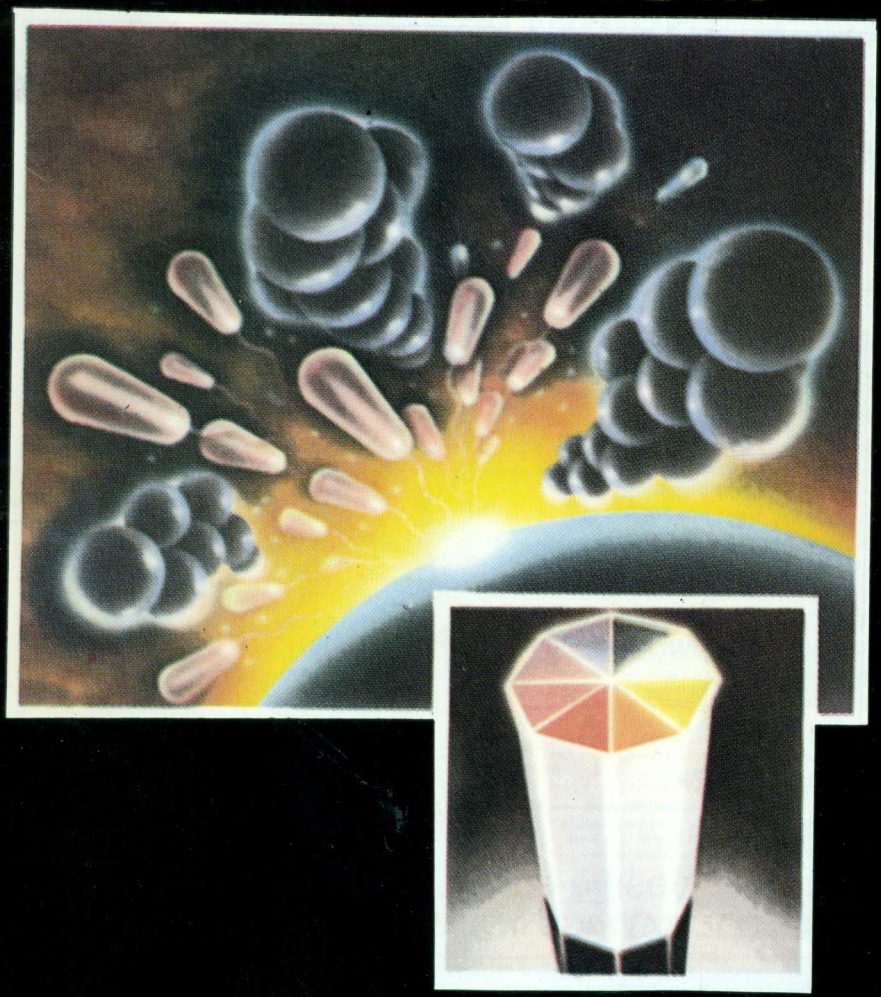
Pfizer

IN SERIOUS INFECTIONS...



Cefobid* IM/IV q12h

(cefoperazone sodium)

THE SOURCE OF STRENGTH BEHIND THE RECOVERY

Cefobid* IM/IV q12h

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Meningitis	11	91%
Urinary tract infection	1,155	83%
Obstetric/gynecological infection	211	93%
Abdominal infection	133	90%
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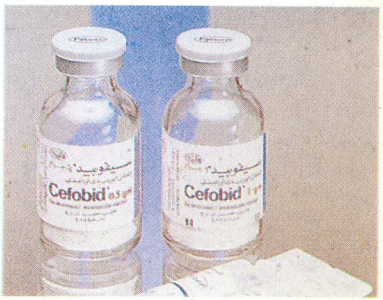

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Presidenial address *

Residency Training

Prof. Anwar Balbaa

Honorable members of the Egyptian Society of Cardiothoracic Surgery Distinguished guests.

What a privilege it is to stand here, this morning, as president of our new society. It is indeed the highest professional honor that I will receive in my lifetime.

In an attempt to choose a subject for my address, very clearly the topic of residency training came to front. Not only can improving the standard of residency training improve the science and practice of our specialty, but also will result in a population of well treated grateful patients.

This will have its tremendous impact on the self confidence within our specialists as well as genuine nationwide appreciation of our specialty.

While preparing this talk, I put down in a small note book, my reflections on what I would like to say, and my impression on what we have good or bad. At the same time, I recorded what, in my opinion, would be a start, to implement the necessary change required for a better future for our coming generations. I am sure, most of you will agree with me on most points.

When I started to go through the literature and review presidential addresses delivered at the different societies and associations, I realized the amazing similarity between what I have recorded and what I was reading.

The ambitions are the same and the problems and limitations are the same. Of course their problems are much less than ours

The analysis and wisdom of Drs Paulson, Grillo, Anderson, Oringer, Lillehei, McGovern, Valdhavsen .... have put clearly the basis for residency training programs.

I would like, for the interest of our new society, to bring to your attention, the cream of their research and recommendation, sometimes in full quotation.

What we have achieved in Egypt over the last 4 decades, especially during the eighties is atonishing. The skill in handling most of the cases in cardiac as well thoracic surgery is quite evident. The results are excellent inspite of the limitations and deficiencies. The younger surgeons, anesthetists, perfusionists, intensivists and nurses have quickly grasped and mastered the handling, interpretation and routine use of the most sophisticated technologies.

This is my personal comment; it reflects the evaluation by the great teachers who visited our centers, both University and Institutional. The progress has been more rapid and most evident in the last few years.

As Professor Anderson puts it, "The era of rapid innovations is slowing down with

* Adress delivered on opening the first annual meeting of the society February 1994

the standardization of the basic techniques and policies". We are now on the threshold of a great change, brought about by the break through in understanding molecular biology. We must be able to use this knowledge to increase the benefits and decrease the risks of cardiothoracic surgery.

We must reexamine the education and training of young surgeons who will be our successors. We expect from them more disciplined, sound and safe performance, as well as more innovations. It is time for all of us to sit together, senior and junior to evaluate, criticize and try.

To improve what we have, we should formulate a better training program. Any delay in this will be disastrous. This program should then be discussed with the administrative authorities at all sectors. A change of the law may be necessary.

On examining the present situation, I realized that we are not manufacturing new generations of cardiothoracic surgery personnel, as efficiently as industry introduces their new generations of equipment. Why ? Because they invest in more evaluation and research than we do. Besides, bureaucracy hampers the implementation of the necessary change. More and more enthusiastic research efforts should be shared and motivated by all of us.

Our younger colleagues are taken away by the glory of cardiac surgery. They seem to forget that the pioneers, teachers and stars of cardiac surgery owe their notable success to their solid general thoracic background.

As Professor Grillo points out "the fact that most of the general thoracic surgery is done by older surgeons indicates clearly that less younger surgeons are willing to participate. We have let the oesophagus surgery to slip away. Let us not leave the empyema and lung surgery by managed by the general surgeon or the cancer surgeon. Let us not allow the new video assisted thoracic surgery to be taken over by the laparoscopic surgeon or the chest physician.

Pride of Cardiothoracic surgery:

Let us not forget that it was the cardiothoracic surgeon who:

1- Clinically introduced cardiac catheterisation; the exact anatomical details, intracardiac pressure changes and shunts are not needed by the cardiologists as they are manadatory for the surgeon.

2- Opened up the field of pediatric surgery, be it in the neonatal period to save lives or in infants and children to definitively treat congenital or inflammatory cardiac or pleuroplumonary lesions.

3- Established the entire field of critical care.

In Egypt as in Europe and America real intensive care and coronary care units started in relation to post operative cardiopulmonary surgery.

4- Develop support systems for cardiogenic shock in the postoperative care of complicated cardiac cases, and later these systems were adopted for medically treated cases.

5- Mastered endoscopies long before fibreoptic scopes became available for the physicians to use.

Basic Principles of change:

These can be discussed taking in consideration three aspects

1- Residency training.

2- Physician patient relationship.

3- Socio economic conditions

Residency Training

The preparation of the future surgeon is a very elaborate process. Sometimes, in view of the work load that a resident is expected to do the main essence of his pressence gets blurred.

The resident is there for education not for service therefore he is entitled to have good sleeping and eating facilities.

He should have reasonable resting hours, as the deep involvement in patient care, which is usually very demanding and fatiguing, results not only in poor decision making but also inability to read, to attend conferences..... etc.

He should have more adequate ancillary staff to help him register vital signs, perform routine procedures, and administer solutions and drugs.

He should have more involvement in preoperative preparation and diagnosis of his cases before they come to surgery. He should also share in the decision making.

He should have more understanding in basic physiology, pathology and pharmacology in a well planned course made available for him and other residents in affiliated specialties.

Patients Surgeon Relationship

This relationship has to be taught, demonstrated and insisted upon in the training of the resident. The surgeon must remain in charge of his patient before operation, in the I.C.U. and after that. This is vital for the well being of the patients. He may delegate part of the job to others but the patient must recognize him as the responsible person who can listen to his remarks & complaints and make a sincere effort to help him.

The aged person requires special handling and sympathy that only the surgeon can offer, because usually, he suffers from other disease conditions like diabetes, cerebral atherosclerosis .... etc. Also he may be more fussy and demanding.

Socio-economic conditions

Limited resources and unavailable funds should never result in poor surgery., even if this results in closing down the centre or limiting the number of patients operated.

Decision in surgery is essentially scientific. However sometimes alteration in this decision may be made, based on socio-economic conditions. The availability of laboratory control of anticoagulant therapy, the presence of important neurologic handicap, can certainly alter the decision or modify it.

Personal suggestions for change

The present situation of residency training in our medical community is definitely inadequate.

Many problems have to be clarified and several radical changes will have to be discussed at the medical policy-making level, at hospital administration level as well as the legislative level.

Changes in our system must entail recruiting residents from undergraduate students and rotating interns. Moreover, changes are needed in the residency and postgraduate programs.

Undergraduate Changes.

To draw the attention and stimulate interest in cardio-thoracic surgery, the undergraduates should be more exposed to lectures, clinical cases, operations and even to postgraduate conferences. Every student should be trained to perform cardiopulmonary resusciation and related procedures.

Rotating Internship Change

Every intern should have a saying whether he chooses to train in the cardiothoracic surgery unit in preference to other special surgery units or not.

The intern should be assigned to a certain number of patients from admission to discharge, including atending their investigation procedures and the operative procedures.

Residency Training Change

This topic will be discussed from three aspects.

A- Duration.

B- Content.

C- Evaluation.

Duration

The present status is very inadequate.

The three years allotted period is distorted by two interferences viz 15 months for military service plus 6 months devoted to general surgery training in the general surgery and casualty departments.

The residency training of the future cardioracic surgeon in the U.S., for example, is around 6-7 years. Three years in general surgical residency precede another 3 years in cardiothoracic surgical residency.

For these reasons three suggestions are made:

1- The military service should be postponed till after the residency training (for negotiation with the authorities).

2- Only general surgery residents after two years training can apply for cardiothoracic surgery.

3- The period of cardiothoracic residency should be two years.

Content of training

As recommeded by Orringer, McGovern, and McKneally should include:

1. Personal experience of a fixed number of patients.

2. A number of Major thoracic operations.

Major cardiac operations.

Minor thoracic procedures.

Endoscopies.

3. Attendance of Chest conference.

Cardiology conference.

Oncology conference.

Mortality & morbidity conference.

4. Participation in research projects.

5. Cardiothoracic teaching sessions in physiology and pathology.

The general thoracic surgical training is essential in every University center. It should be enforced in any center doing only cardiac surgery, at an affiliated center.

This general thoracic surgical teaching course topics should include:

1. Thoracic surgery oncology.

2. Benign oesophageal disorders.

3. Tracheal injury.

4. Pulmonary and pleural infections.

5. Interstitial lung disease.

6. Thoracic trauma.

7. Pediatric thoracic surgery.

8. Thoracic critical care.

9. Mediastinal abnormalities.

10. Lung transplantation.

The general thoracic surgical training course should include:

a. Operations: Pulmonary, oesophageal, bronchoscopy and oesophagoscopy.

b. Outpatient work.

c. Perioperative evaluation and decision making.

d. Methodology and interpretation of pulmonary and oesophageal functions.

Evaluation of the resident after training

This is essential it will evaluate the program itself.

Needless to say the junior surgeon who has finished his residency training program is entitled to be the decision maker for all the work in a small cardiothoracic surgical unit in a provincial hospital or a new medical school.

The fate of the patients who are the victims of accidents, suffer from empyema, spontaeous pneumothorax or resuscitation will be essentially in his hands. Our daily practice exposes many wrong decisions and iatrogenic injuries.

Role of the society

The first role should entail the defining of the number, structure, equipment and personnel of the different centers performing cardiothoracic surgery at prestent. This definition will help put the optimum and the minimum basic requirements for maintaining the present centers and initiating new ones.

Once this definition is clear the society will use every available means to help upgrade the different centers. Every effort should be spent to clarify the role of the physician in collaboration with the surgeon to offer the best possible service for our patients. Any

problems of overlapping interests can be solved by discussions and agreements.

One main task for the society should be to help organize the work of foreign doctors who come to work in Egyptian hospitals and lay down the rules for their function.

Suggestions for the future

I suggest that the first thing to do would be that the society would form a committee of experienced surgeons and anesthetists to study the possible alterations needed for residency training programs. They should present to the society their suggestions and recommendations at the end of a fixed period. Those recommendations should be taken to the different centers for implementation in their programs.

Besides, the authorities in the armed forces should cooperate in the change needed in their enlistment rules. Some alterations in the duration of the residency training will require changes of the law governing the University hospitals.

Regular meetings will be required to increase the awareness of the importance of socioeconomic factors in our basic understanding and dealing with our patients' problems.

Metabolic support by the use of glucose-insulin and potassium infusion has been used successfully in the last decade for managing intractable heart failure and acute cardiac insufficiency after myocardial infarction [45, 46, 47, 48, 49].

Aim of the work

In this study we assess the effectiveness of the metabolic support after hypothermic cardiac arrest in open cardiac surgery.

Patients and methods

Fifty patients were comprised in this study, 34 females [68%] and 16 males [32%]. Their age ranged from 19 to 46 years with a mean of 26±8.4 years. All patients had advanced degree of rheumatic valvular lesions.

Sixteen patients [32%] had double mitral lesion [DM] and tricuspid regurge [TR], twenty two patients [44%] had DM and double aorta [DA]. While twelve patients [24%] had DM, DA and TR. All the patients were presenting in dyspnea grade IV NYHA (50).

Their twelve leads electrocardiogram [ECG] revealed: ten patients [20%] had sinus thythm while forty [80%] patients had atrial fibrillation. Three patients [6%] had P-mitral and seven patients [14%] had P-pulmonale. Nineteen patients [38%] had left ventricular hypetrophy [LVH] while thirty one patients [62%] had biventricular hypertrophy.

Roentgenographic examination of the patients showed marked cardiomegaly and the cardiothoracic ratio ranging between 0.67 with a mean 0.71+0.12. Left atrial enlargement was evident in all patients, while twenty eight [56%] patients had right atrial enlargement. Left ventricular enlargement was detected in twenty patients [40%] while thirty patients [60%] had biventricular enlargement. Pulmonary artery was enlarged and dilated with severe pulmonary venous congestion in thirty patients [60%].

Echocardiographic examination of the patients by M-mode and twodimensional echo with Doppler mapping techniques, revealed poor left ventricular function in all patients with mean ejection fraction of 28% [range from 26 to 30%] and mean systolic pulmonary artery pressure of 74 mmHg ± 10 [range from 62 to 92 mmHg].

The patients have been classified randomly into two groups. Group A comprised 25 patients who received glucose-insulin-potassium [G.I.K] infusion during and immediately after cardiopulmonary bypass [CPB]. Group B comprised 25 patents taken as a control group. They received ringer's solution CPB.

The patients were premedicated over night with intramuscular, valium. Anesthesia was induced with thiopental and maintained with fentanyl 1, pancronium, nitrous oxide and oxygen.

CPB was conducted with bubble oxygenator and a roller pump with nonpulsatile flow. The priming solution consisted of Ringer's lactate, a non-glucose non-lactate crystalloid fluid. Moderate hemodilution [25% to 30%] and hypothermia 28°c to 30°c

were employed. St. Thomas Hospital cardioplegic solution was used for myocardial protection.

In the insulin group [group A] a bolus injection of 25 IU of fast acting insulin [Actrapid] was given, followed by continuous infusion of 1 IU/kg body weight in addition to 100 ml glucosé 25% and 10 mEq KCI per hour dring CPB while group B patients were given Ringer's solution during CPB.

Results

There was insignificant statistical difference between both groups as regards their clinical data and hemodynamic status preoperatively [P 0.05]. Table (1).

Table 1: Pre-operative data.

Data	Group A	Group B	P value
Number of patients mean age	24 ± 3.1	25 ± 1.2	P>0.05
NYHA class IV	25	25	P>0.05
Mean CTR in x-ray	0.70±1.6%	0.69±0.09%	P>0.05
Mean LV EF by 2-D echocardiography	71±2.9	28.4 ± 1.9%	P>0.05
Mean systolic PAP	71 ± 2.9	73 ± 2.1	P>0.05
Clinical diagnosis D.M & T.R. D.M.&D.A. D.M.,D.A.&T.R.	8 12 7	8 10 5	P>0.05 P>0.05 P>0.05

But there was significant statistical difference between both groups during operation.

The cross clamping time was nearly equal in both groups but the mean total bypass time was 85 minutes [range from 60 to 100 min.] in group A patients and 160 minutes [range from 95 to 210 min]. in group B patients [P 0.001] Table (2).

Table 2: Operative data.

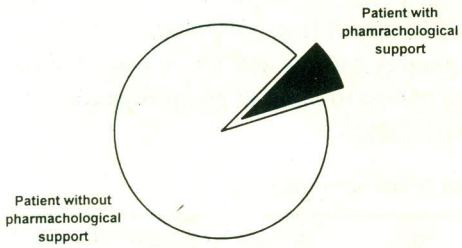
Data	Group A	Group B	P value
MVR & Trepair	8	8	P>0.05
MVR & AVR	12	10	P>0.05
MVR, AVR & DevEga Trepair	7	5	P>0.05
Mean aortic cross Clamping time	72 ± 19	75 æ 12	P>0.05
Mean total CPB time	85 ± 21	160(95-210)	P>0.05
Pharmacologic support for LCOPS	25	-	P>0.05
Operative mortaity	3	-	P>0.05 P>0.05 P>0.05

Table 3: Post-operative data.

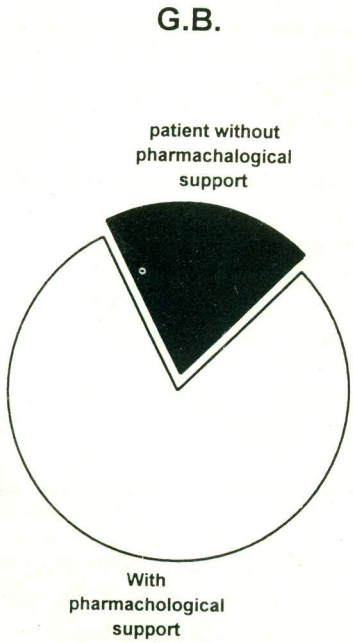
Data	Group A	Group B	P value
Mean artificial ventilation time/hrs	18 ± 6.2	42 æ 9.6	P>0.05
Pharmacologic support for LCOPS	2	20	P>0.05

Pharmacological support of the heart with one or more inotropic drugs [Dobutamine, Dopamine, Isoprel, Adrenaline and Levofed] with or without vasodilator drugs [Nitroglycerin and Nitroprusside] was needed in

G.A.



G.B.



patients of group B to terminate CPB. Table (3).

Operative mortality was 12% [three patients out of 25 patients]. Postoperatively, prolonged ventilation more than 48 hours was essential for three patients in group B patients while all patients of group A were weaned from artificial ventilation within 24 hours [P 0.001].

Discussion

The major finding of this study was that metabolic support of the heart during ECC by G.I.K. infusion improves the outcome of surgery in high risk patients[1,2]. Patients with multiple rheumatic valvular diseases usually presented to surgery in an advanced state [51, 52, 53, 54, 55].

These patients had impaired left ventricular function, severe pulmonary hypertension [56, 57, 58] and variable degrees of rheumatic myocardial insult [rheumatic cardiomyopathy] [59, 60].

Postperfusion ischemia during surgical intervention usually produces refractory heart failure and arrhythmias immediately after CPB in these patients ,(61). Various maneuvers have been utilized to support the failing heart immediately after CPB they are:

a. Mechanical support by assisting the failing heart with ECC or intra-aortic balloon pump [IABP] [3, 4, 5, 6, 7, 8, 9, 10, 11].

b. Pharmacological support by one or more positive inotropic drugs [12, 13, 14, 15, 16,

17, 18, 19, 20, 21, 22, 23, 24, 25, 26, 27, 28, 29, 30, 31, 32, 33].

c. Metabolic support by G.I.K, infusion [34, 35, 36, 37, 38, 39, 40, 41, 42, 43, 44].

Metabolic support of the heart by G.I.K. infusion improves the myocardial tolerance to ischemia, the mechanical function and counteracts the catabolic effects of trauma metabolism [62, 63, 64, 65].

Although the exact mechanisms for the beneficial effects of G.I.K. infusion on myocardium during ECC remain to be elucidated. They may be:

1. Extraction of myocardial potassium that is caused by intracellular potassium shift.

2. Increase myocardial glucose extraction.

3. Decrease myocardial NEFA [non-esterified fatty acids].

The last two mechanisms are caused by shifting of the myocardial metabolism from predominant lipolysis to predominant glycolysis.

4. Increase myocardial lactate extraction, obviously caused by the avoidance of myocardial lactate accumulation by way of stimulated pyruvate oxidation [63].

Summary

This study proved that using metabolic support during ECC eliminates the effect of postperfusion ischemia that manifests by low cardiac output syndrome immediately after ECC particularly in high risk patients. Thus patients of group A could be weaned easily from ECC without operative mortality while those of group B required more time on ECC to assist the failing heart. More than one positive inotropic drug with or without vasodilator drugs have been utilized for group B patients for weaning from ECC. Group B patients had statistically significant operative and post-operative mortality and morbidity.

Conclusion

Metabolic support by G.I.K. during ECC will increase myocardial tolerance to ischemia and counter the catabolic effect of trauma particularly in high risk patients.

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Long-term follow-up of patients after prosthetic cardiac valve reoperation

Abstract

Between January 1980 and January 1990, 75 patients were reoperated upon for prosthetic valve malfunction (PVM): 26 early symptomatizing - Less than 2 years-prosthetic valve disinsertion (EPVD), 16 Prosthetic valve endocartitis PVE, 13 valve thrombosis PVT, 10 bioprosthetic valve failare BPVF and 10 late symptomatizing-more than 5 years - prosthetic valve disinsertion (LPVD). The patients presenting with a mean NYHA FC of 3.7±0.4 benefited from prosthetic valve replacement (81.3%) or reinsertion with a resulting mean NYHA FC of 1.6±0.65 (P< 0.007) for the 65 hospital survivors.

Patients were followed-up for 20-120 months (mean 60 and median 56 months). The annual incidences of late mortality, second EPVD, PVT, PVE and reoperation for a second PVM were 1.5%, 1.8%, 0.9%, 0.3%, and 3% per patient-year; respectively. At 120 months, the actuarial survival rate of the hospital survivors was 86.8%±5.9% and their actuarial reoperation-free rate was 74%±7%.

A second PVM reflected patient-related risk factors: advanced age (P 0.03), cardiothoracic ratio > 0.65 (P< 0.02) and an annual incidence of a recurrent PVM 3 to 10 folds that of newly developing one (P = NS). Late survival tends to be a function of hospital mortality; the comparison of the survival rates based upon the early postoperative NYHA FC class was highly significant: class I & II vs. III & IV : 78.3% ± 12.9% vs. 33.3%+13.5% (P> 0.001). Other comparisons based upon type of PVM, Preoperative NYHA FC and position of prosthesis were insignificant. On the other hand, reoperation does not appear to affect late survival. At 240 months, the actuarial survival rate of the 75 patients calculated since native valve replacement was 63.8%±10.7% with a mortality incidence of 2% per patient-year.

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Cardiac valve replacement is now part of the routine practice of all cardiac surgeons. The follow-up data of 75 patients reoperated upon for prosthetic valve malfunction, at our

institution, were reviewed in this report. The possible relation between such information, the preoperative and early postoperative variables are discussed.

Methods

From January 1980 untill January 1990, 75 patients were reoperated upon for prosthetic valve malfunction (PVM) at our institution. None was discovered accidently during a routine clinical examination. Shortness of breath was the main complaint (49%). All cases were confirmed by echocardiographic and doppler studies. The preoperative characteristics of the patients are outlined in Table I. Cases with disinserted prostheses presenting during the first two years following native valve replacement were termed early prosthetic valve disinsertion (EPVD), while those appearing later on were termed late prosthetic valve disinsertion (LPVD). Both groups have different mechanism of detachement[1], pathological associations[2] and lines of management.

All operative procedures were performed with cardiopulmonary bypass, moderate hypothermia (28°C), and cardioplegic arrest with multidose (every 25 minutes) cold potassium cardioplegia. Both cavae were cannulated for venous drainage and the ascending aorta (80%) or the femoral artery (20%) was used for arterial return. In cases of EPVD with a detachement affecting less than 1/3 of native ring circumference, the prosthesis was reinserted with interrupted matteress sutures butressed with teflon pledgets. In the other cases (46%), the prosthesis was replaced by a similar one. On the other hand, all patients presenting with LPVD benefited from prosthetic valve replacement; an act legitamized by the long period of implantation (117.7±64.2 months) and the frequent association (80%) of small thrombi and pannus formation. Patients with PBVF, PVT or PVE benefited from prosthetic replacement with a bileaflet mechanical prosthesis (72.4%) or a bioprosthesis (27.6%) in patients in whom anticoagulation was presumed difficult or contraindicated as well as young females wishing to have children. Other operative variables are outlined in (Table I)

Surgery resulted in 2 opertive mortalities (2 PVE) and 8 hospital mortalities. Early mortality related risk factors (Table II) included time of cardiopulmonary bypass ($P< 0.001$), advanced preoperative NYHA FC ($P< 0.01$), emergency operation ($P< 0.03$) and type of PVM ($P <0.05$). Other preoperative & operative variables demonstrated in Table I were statistically unsignificant. As guide by the NYHA functional classification, the 56 hospital survivors have shown a significant clinical improvement before hospital discharge ($P< 0.01$). Other postoperative characteristics are outlined in Table III. All patients were placed on an anticoagulation regimen using dicumarol starting on the

T. (II) : Risk factors of Hospital Mortality :

	Hospital Mortalities	Hospital Survivors	"p" value
Number	10 (13.3%) *	65	
1- Type of PVM:			0.05+
PVE	5 (31.25%)	11	
PVT	3 (23%)	10	
LPVD	1 (10%)	9	
EPVD	1 (3.8%)	25	
PBVF	----------	10	
2- Preoperative : NYHA FC mean ± SD	4±0	3.6 ± 0.47	<0,01=
3- Emergency operation	8 (29.6%)	19	<0.02$
4- Time of cardiopulmonary bypass mean ± SD (minutes)	163 ± 49.8	122.5 ± 28.5	<0.001#

Legends : see table I.

* = number in parenthesis indicate percentage of total.

+ = PVE versus PVT versus other PVM (corrcted Chi-square test).

= = paired Student test.

$ = corrected Chi-square test.

= unpaired Student test.

Table (III) : General characteristics of hospital survivors.

1- Number of patients	65
2- Mean age ± SD (years)	31.9 ± 13.6
3- Sex : Males	33
females	32
4- Type of prosthesis.	
St.Jude Medical	24 (34.3%)
Bjork-Shiely	20 (28.6%)
Carpentier-Edwards	17 (24.3%)
Starr-Edwards	9 (12.8)
5- Position of prosthesis	
Mitral	35 (53.8%)
Aortic	25 (38.5%)
Double	5 (7.7%)
6- Mean NYHA FC ± SD	1.6 ± 0.65*

* P = 0.01 as compared to the preoperative NHYA FC (paired Student test).

T.IV : Follow-up data:

Total number of patients	75
Hospital survivors	65
Mean follow-up (years)	5
Median follow-up (years)	4.5
Range follow-up (years)	1.6-10
Cumulative follow-up (patient-year)	333
Late mortality (patient-year)	1.5%
-Prosthesis related	0.6%
- Unrelated	0.9%
Second prosthetic valve malfunction (patient-year)	3%
- Early prosthetic valve disinsertion	1.8%
- Prosthetic valve thrombosis	0.9%
- Prosthetic valve endocarditis	0.3%
lost to follow-up (%)	9.3%

Table (v) : Late mortality :

Type of PVM	Type of prosthesis	Position	Management	cause	Death* timing	No in group+	% of PVM type
PBVF	Carpentier -Edwards	M	-MVR by Carpentier-Edwards bioprosthesis	PVT	5		
EPVD	Starr-Edwards	Ao	-AVR by Starr-Edwards prosthesis	EPVD	16		
	//	M.	-MVR // //	sudden	17		
	//	M.	-MVR // //	cancer ovary	114		
PVE	//	Ao	-AVR by Bjork-Shiely prosthesis	sudden	72		
Total				5	44.6 ±46.8=		7.7%$

Legends : see table I

* period between prosthetic valve replacement and death in moths.

+ number and % of hospital survivors

= mean æ 1 SD.

$ percentage of total number (65) of hospital survivors.

Table (VI): Patients reoperated upon for a second PVM:

Previous PVM	type of prosthesis	Position	Management	No & % of type of PVM	Presenting PVM	Result of operation
1- EPVD	Starr-Edwards	Ao	-AVR by Starr-Edwards prosthesis		EPVD	-post-operative death
2- EPVD	Starr-Edwards	M	-Repair		EPVD	-alive & well
3- EPVD	Carpentier-Edwards	Ao	-Repair		EPVD	-alive & well
4- EPVD	Carpentier Edwards	M	-MVR by St.Jude valve	25 (16%)	EPVD	-alive & well
5- PVT	Starr-Edwards	M	-MVR by Carpentier-Edwards bioprosthesis		PVT	-alive & well
6- PVT	Starr-Edwards	M	- // //		PVT	-alive & well
7- PVT	Bjork-Shiely	M	- // //	10 (30%)	PVT	-alive & well
8- PBVF	Carpentier-Edwards	M	-MVR by		PVT	-post-operative death
		M	Carpentier-Edwards bioprosthesis			
9- PBVF	Carpentier-Edwards		-MVR by St.Jude valve	10 (20%)	EPVD	-alive & well
10 PVE	Starr-Edwards	Ao	-AVR by St-Jude valve	11 (9%)	EPVD	-alive & well
Total				(15.4%) + (10)		

Legends. see table I

* number and percentage of hospital survivors per type of PVM.

+ percentage of total number (=65) of hospital survious

second postoperative day after removal of the drainage tubes. Anticoagulation dosage was adjusted in accordance with a frequent determination of the prothrombin time. By the fourth month, dicumoral was stopped for patients with a bioprosthesis, while a life-long anticoagulation regimen was carried out for patients with a mechanical prosthesis. Follow - up information was collected by patient contact at our outpatient clinic, or a reply on a mailed questionnaire to the refering physician. Time related events, namely death and valve related complications were stored and analyzed as of September 1992 at our Cardiovascular computer data bank facilities. The actuarial method was used to estimate survival and valve related complications necessitating reoperation. Comparison among actuarial curves was evaluated by the log-rank test[3]. Paired and unpaired student tests and Chi-Square test with Yate's correction were used to test non-homogenecity of

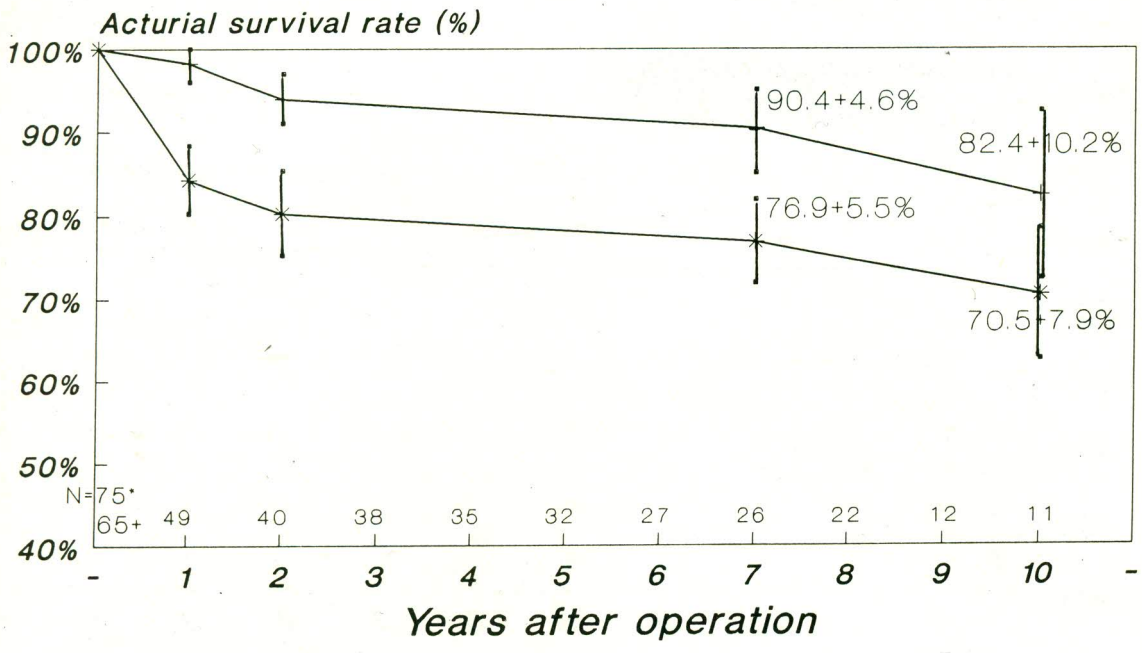

Fig. (1): Actuarial survival rates of 75 patients presenting with PVM* and of the 65 hospital survivors+

N= number of patients living at the begining of the interval. Bars are ± ΣE.

event free and eventfull groups of patients. The coefficient of correlation of ranks (Spearman's test) was used to correlate between the length of the asymptomatic periods preceeding the first and an eventual second PVM.[4]

Results

Table IV summurizes the follow-up data. Seven patients could not be contacted; their information was not computed and they were considered lost to follow-up. Five late deaths were reported (Table V), two of which were definetely related to a second PVM and died shortly after reoperation from low cardiac output. The late mortality rate was 7.7% of hospital survivors which corresponds to an incidence of annual late mortality of 1.5% per patient year. There was no statistically significant relation between late mortality and any of the variables outlined in Table I.

The actuarial survival rate of the 75 patients presenting with PVM was 70.5%±7.9% while that of the 65 hospital survivors was 82.4%±10.2% at 10 years (figure 1). There was no statistically significant difference between the survival rates of patients with different types of PVM (figure 2), position of prosthetic implantation (figure 3) or preoperative NYHA FC (figure 4). On the other

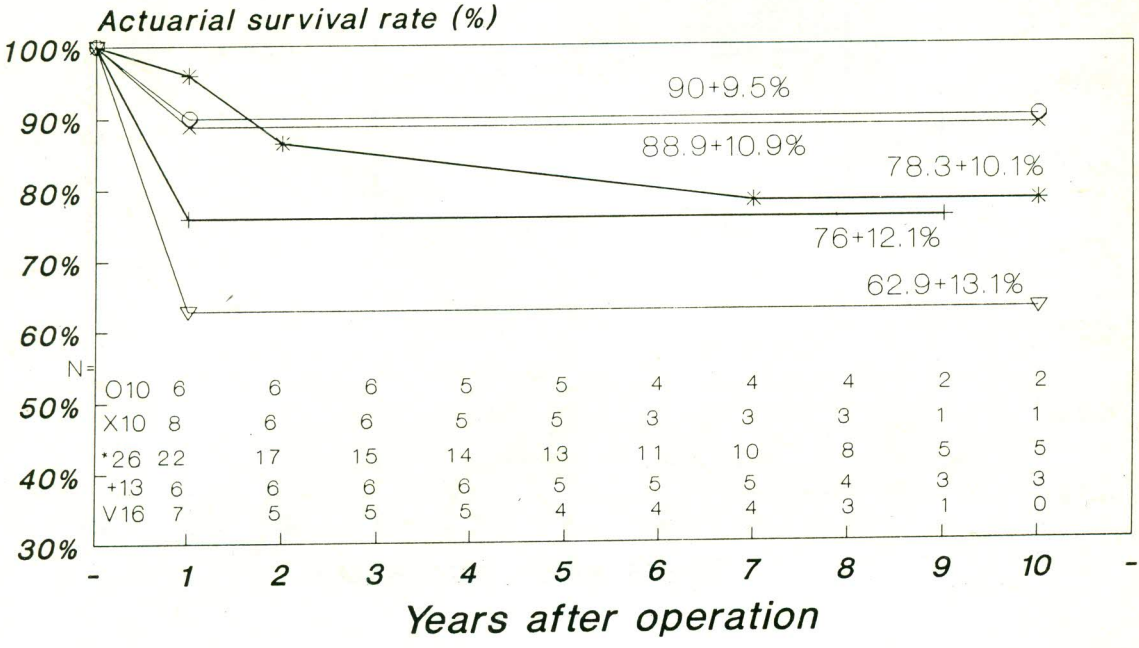

Fig.2 : Actuarial survival rates according to the type of prosthetic valve malfunction.

O primary bioprosthetic valve failure.
X late prosthetic valve disinsertion
*** early prosthetic valve disinsertion**
+ prosthetic valve thrombosis.
V prosthetic valve endocarditis
N= number of patients living at the begining of the interval.
(P= non-significant, log-rank test).

hand, late survival was significantly related to the postoperative NYHA FC (figure 5) with a 10 years survival rate of 78.1%±11.9%% for patients in class I or II and of 33.3%±13.5% for patients in class III or IV (P<0.001)

Ten patients developed a second PVM and were all reoperated (Table VI). They presented with a mean NYHA FC of 3.4±0.69. The mean aortic cross clamping time was 70.5 ± 19.2 minutes and the mean cardiopulmonary bypass time was 137 ± 40.3 minutes. Two patients (20%) died in the early postoperative period from low cardiac output, both were operated upon on emergency basis (20%). The 8 hospital survivors had an uneventful postoperative course and were discharged from hospital with a mean NYHA

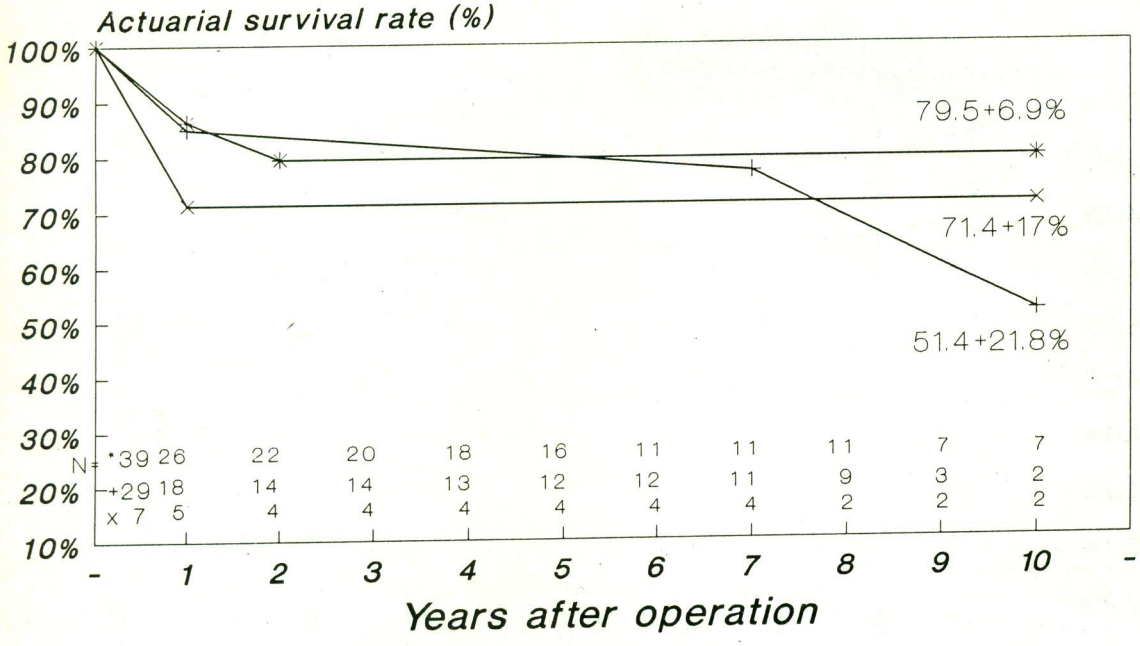

Fig.3 : Actuarial survival rates according to the postion of the malfunctioning prosthesis.

*** mitral position**
+ aortic position
X both mitral and aortic positions
N= number of patients living at the begining of the interval.
(P= non-significant, long-rank test).

FC of 1.37 ± 0,5 (P< 0.01). At 20 years, the actuarial survival rate of the 75 patients calculated since native valve replacement was 63.76% ± 10.7% (figure 6).

At 10 years, the actuarial reoperation - free rate of the 65 hospital survivors was 74% ± 7% (figure 7). The annual incidences of PVE, PVT, EPVD and reoperation for a second PVM were 0.3%, 0.9%, 1.8% and 3% per patient-year, respectively. Out of the 25 hospital survivors initially presenting with EPVD and followed-up for a total period of 133.75 patient-year; four showed a recurrent EPVD which is equivalent to an annual incidence of 3% per patient-year. Similarly the annual incidence of a recurrent PVT was 3.7% per patient-year for the 10 hospital survivors initially presenting with PVT and followed-up for a total period of 53.75 patient-year. On the other hand, the annual incidences of EPVD and PVT calculated for

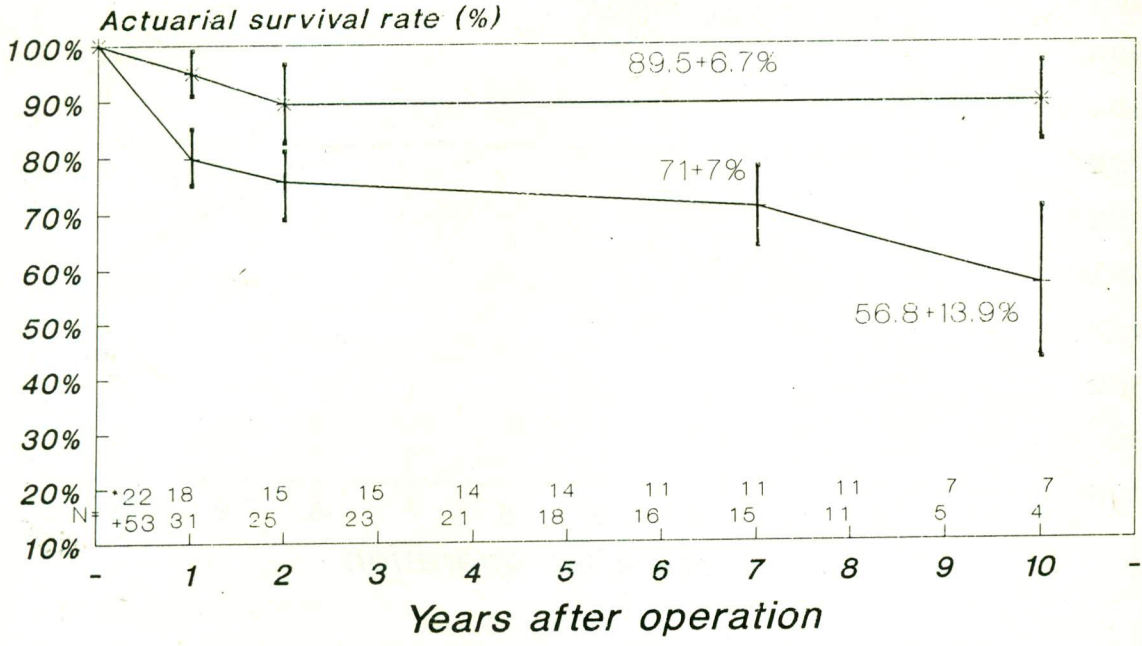

Fig.4 : Actuarial survival rates according to the prepoperative NYHA functional classes III* or IV+.

N= number of patients living at the begining of the interval. Bars are ± ΣE (P= non-significant, log-rank test) .

patients not initially presenting with these malfunctions were 1% and 0.35% per patient-year; respectively (P = Ns)

Risk factors associated with a second PVM (Table VII) were cardiothoracic ratio 0.56 (P< 0.02) and advanced age (P< 0.03). There was a statistically significant negative correlation (P< 0.01) between the asymptomatic periods preceeding the first and the second PVM (Table VIII). Other variables outlined in table I were statistically insignificant.

Discussion

In conformity with the previously reported data[5,6,7,8,9,10] the principle determinants of early survival in this series were the proper timing of surgery and the preoperative NYHA functional class. Our high hospital mortality (13,3%) appears to be the result of a 36% emergency rate in a group, 70.6% of its members were in class IV NYHA. Despite a statistically significant higher mortality rate of PVE and PVT, and as have been noted by others[7,9], patients reoperated upon electively - PVE and PVT included - showed a

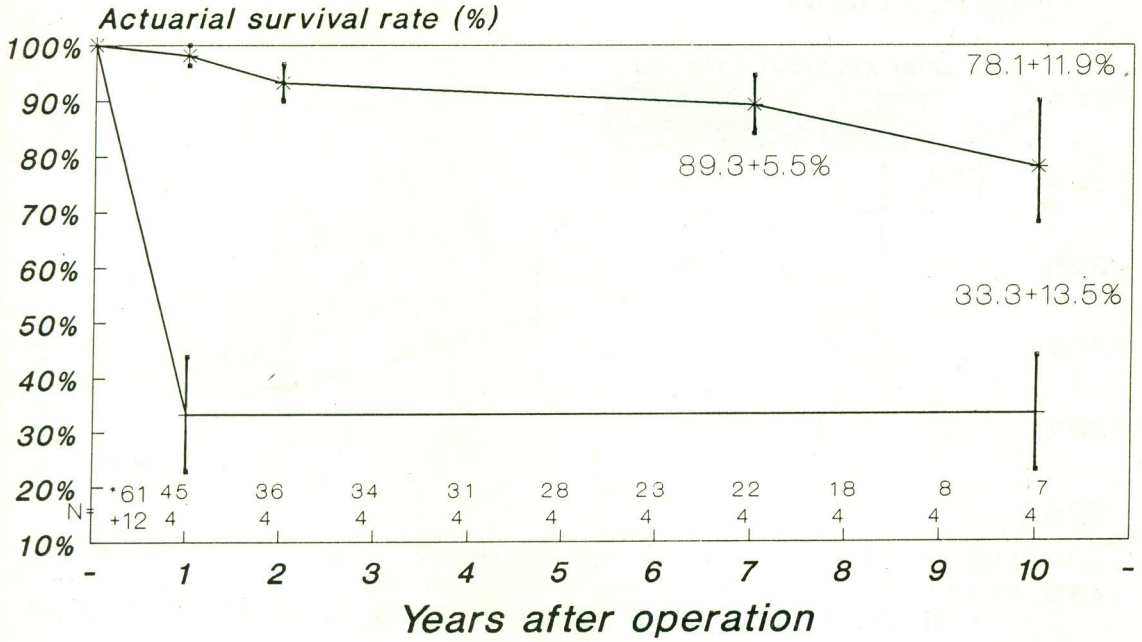

Fig. 5 : Actuarial survival rates according to the postoperative NYHA functional classes I & II* or III & IV+.

N= number of patients living at the begining of the interval. Bars are ± ΣE
(P <0.001, log-rank test)

hospital mortality figure (4.3%) similar to that encountered with native valve replacement. On the other hand, we consider the statistically - related prolonged cardiopulmonary bypass time to be the reflection of already setteled - in preoperative risk factors reponsible for increased mortality and poor results. On condition to be timed properly, the redo procedure does not appear to have and incremental effect on hospital mortality. Our study supports the ongoing trend towards early surgery in patients presenting with PVM before the settlement of functional deterioration[5,7,8,9,10].

The 65 hospital survivors have shown a significant functional improvement (Table III) with 93.8% of patients in class I or II, an annual incidence of late mortality of 1.5% per patient-year and an actuarial survival rate of 82.4%±10.2% at 10 years (figure 1). the actuarial survival rates of the whole group of patients, including hospital mortality, were 76.9%±5.5% and 70.5±7.5% at 5 ad 10 years; respectively (figure 1). Reported risk factors of late mortality are generally those associated with a high hospital mortality; advanced preoperative NYHA functional class [9,11,12], PVM other than PBVF[9,12], PVE[11], double

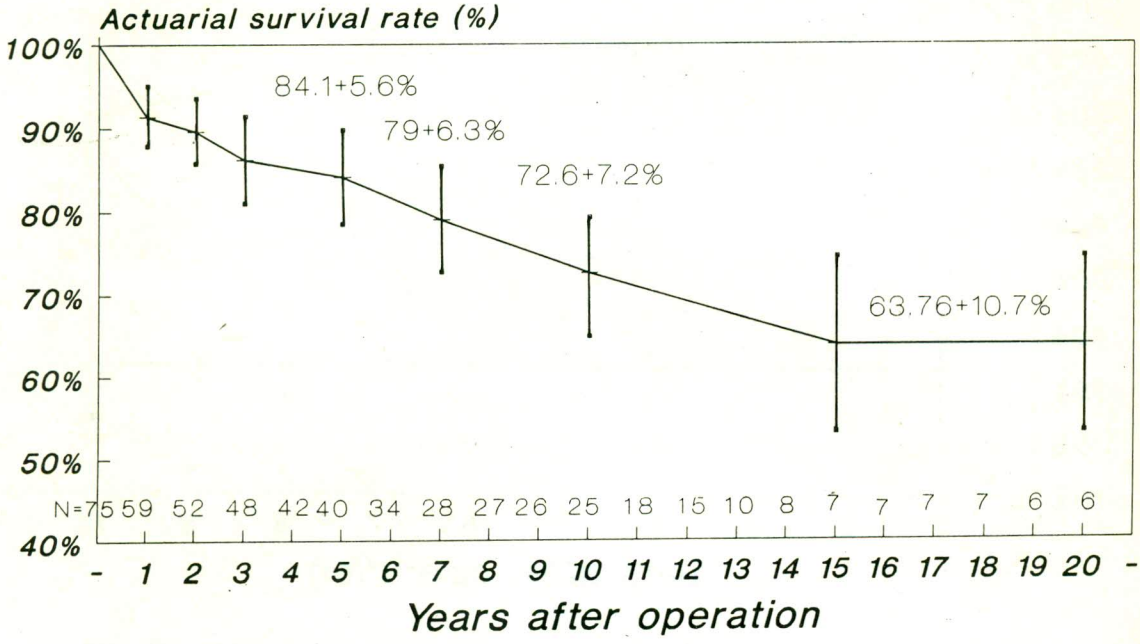

Fig.6 : Actuarial survival rate of the 75 patients calculated since native valve replacement.

N= number of patients living at the begining of the interval. Bars are ± ΣE

valve reoperation[9,11] and mitral valve re-operation[12].

On the other hand, emergency appears to be a qualitative result from the summation of the ongoing functional deterioration. Following the critical early postoperative period, its effect on the convalescing survivors fades with the significant functional improvement. Our study points to the preoperative NYHA FC IV, aortic valve reoperation and PVE as variables associated with lower survival rates, however, statistically insignificant. (figures 2,3,4). In this series, late survival was a function of hospital mortality; being statistically related to an early postoperative NYHA FC III and IV (P< 0.001). (figure 5). Blackstone and Kirklin recorded an early phase of rapidly falling risk of death in the first 2 postoperative weeks, that is followed by a constant phase thereafter[12]. Their reported survival rate (31% at 3.5 years) was low as compa-red to that reported by others: 66.9±5% at 5 years and 57.3%±8% at 7 years[9], 73% at 5 years[7] and 83.3%±9% at 5 years and 65.6%±12% at 10 years[8]. Their 80% survival rate at one month reflected a high hospital mortality rate in comparison to the other series: 8.7%, 8.8% and 5.7%; respectively. All, but them, failed

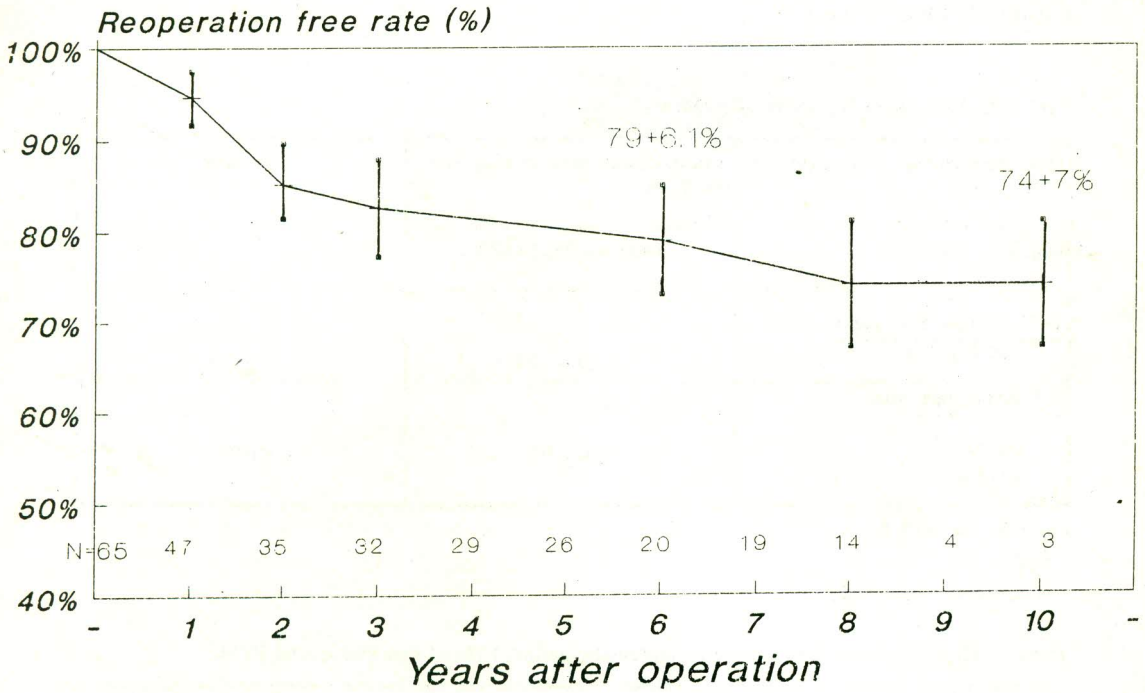

Fig.7 : Reoperation free rate of the 65 hospital survivors.

N= number of patients living at the begining of the interval. Bars are ± ΣE

to demonstrate a statistically signigicant difference between the late survival rates after native valve replacement and first reoperatison. We do not present a group of native valve replacement for comparison however, the actuarial survival rate of the 75 patients calculated since native valve replacement was 63.76%±10.7% at 20 years (figure 6).

After a mean asymptomatic period of 25.8±28.4 months, ten patients presented with a second PVM. As previously reported [7,12], the majority (60%) are disinserted prosthesis. The observations of Bensaid and co-workers suggested that a recurrent EPVD usually manifests with the same delay as the previos one[13]. We can not offer an explanation to the highly significant negative correlation between the asymptomatic periods preceeding the first and second malfunctions (Table VIII). Being all reoperated, the annual incidence of reoperation for a second PVM was 3% per patient-year and the actuarial reoperation-free rate was 74%±7% at 10 years. Patient-related risk factors were associated with the occurence of a second PVM: advanced age ($P < 0.03$), cardiothoracic ratio

Table VII: Risk factors of a second PVM:

patients presenting with a second PVM	patients with a well functioning second prosthesis	"P" value
number = 10 (15.4%)	number = 25 (84.6%)	
1- Mean age + SD (years): 41.6 + 14.2	30.25 + 14.7	< 0.03*
2- Cardiothoracic ratio > 0.56: 8 (80%)	26 (47.3%)	< 0.02+

Legends: see table I,

* Student test.

+ corrected chi-square test.

Table (VIII): Correlation between the asymptomatic periods (AP) of first and second PVM.

First PVM		Second PVM		p value *
type	AP (months)	type	AP (months)	
1- PVE	0.33	+EPVD	24	
2- PVT	0.5	PVE	90	<0.01
3- PVT	4	PVT	64	(r = -0.84)
4- EPVD	8	+EPVD	22	
5- EPVD	9	+EPVD	12	
6- EPVD	12	+ EPVD	6	
7- PBVF	13	+ EBPD	13	
8- EPVD	19	+EBVD	16	
9- PBVF	100	PVT	5	
10- PVT	108	PVT	6	

Legends: see Table I,

* coefficient of correlation of ranks (Spearman's test)

+ "r" = -1.08 and "p" < 0.01 for the 6 patients with EPVD

<0.56 (P< 0.02) and a risk of a recurrent PVM 3 to 10 folds that of new developing one (P = Ns). A higher annual incidence (4.1%), a lower reoperation free rate (50.7%±7.6% at 7 years)[9] and different risk factors (black race, male gender and mitral position)[12] were reported for significantly older (P< 0.01) groups of patients.

The hospital mortality of the second re-operation was high (20%). This percentage reflected severe functional deterioration due to the patient's hesitation in accepting his third open heart surgery in one case and acute bioprosthetic thrombosis in a non-anticoagulated patient previously reoperated for PVT in the second case. Mortality-related risk factors i.e improper timing of surgery and preoperative functional deterioration (P = Ns.), as well as the signficant functional improvement of hospital survivors (P<0.01); appear to be the same as for the first reoperation. In spite of others[9,12] reporting modest survival rates, even at intermediate terms, the small number of hospital survivors (8 patients) in our study does not permit us to comment on the late follow-up of patients after the second reoperation.

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Transesophageal echocardiographic monitoring of closed mitral commissurotomy

Abstract

Closed mitral commissurotomy is considered one of the most popular procedures intended to solve quite a wide section of patients with rheumatic heart disease namely isolated mitral stenosis. Many world wide centrs deny the efficacy of this procedure considering it a blind assessment of the mitral valve apparatus and its function. This study, performed on 46 patients who underwent closed mitral commissurotomy in the period June 1992 till January 1994 in the National Heart Institute. Twenty-four cases underwent commissurotomy with a transesophageal echocardiography probe inserted, the study included 26 females and 20 males whose mean age was 27 years ± 12. Fourteen patients were in sinus rhythm. Nine patients were in NYHA class II, 27 in class III and 10 in class IV, their mean transmitral diastolic pressure gradient dropped after commissurotomy from 14mmHg ± 5 to 4 mmHg ± 3 , the mitral valve area increased from 0.6 cm2 ± 0.4 to 2 cm2 ± 0.6, maximal leaflet separation increased from 0.5 cm ± 0.2 to 1.6 cm ± 0.6, 3 cases had post-commissurotomy mitral incompetence, 2 of them had mild and one had severe regurgitation necessitating urgent mitral valve replacement.

The study proved that experienced surgeons' digital assessment is quite accurate and all the events were confirmed by TEE. Furthermore, some cases with minimal mitral incompetence were denied by the surgeon, yet reported by the echocardiographer to be present but later, echocardiography proved the absence of any degree of mitral incompetence. Neverthaless, the use of TEE during commissurotomy added to the outcome of the procedure especially in cases with severe pulmonary hypertension, those with severe TI and cases with some degree of aortic incompetence.

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Introduction

Since 1956[1] up to the moment, closed mitral commissurotomy remains to be an ideal procedure that solves a prevalent pathology of rheumatic fever complication namely, mitral stenosis[2]. Many centers especially in Europe and United States deny this procedure considering it a blind way of intra-opertive accurate assessment of the mitral valve depending only on digital palpation by the surgeon of the mitral valve orifice,

cusps, commissures and the subvaular apparatus. Many studies proved that experienced surgeons could digitally assess the mitral valve, the efficacy of commissurotomy and incidence of any compilcations during the procedure.

Using the transesophageal echocardiography (TEE) during closed mitral commissurotomy[3], allowed surgeon to "see" the intracardiac events as it provides excellent images of the atrial chambers, mitral morphology and hemodynamics without interfering with surgical techniques[4].

Methods:

In the period from June, 1992 to January, 1994, closed mitral commissurotomy was performed in 46 consecutive patients (26 females and 20 males) with a mean age of 27 years ± 12 (range 15 to 39 years) with symptomatic mitral stenosis. There were 14 patients in sinus rhythm and 28 in atrial fibrillation (kept on oral anticoagulants 15 days pre-operatively), patients with evidence of left atrial thrombus by echocardiography were excluded from the study. Nine patients were in New York Heart Association functional class II, 27 in class III and 10 in class IV. All patients had variable degree of pulmonary hypertension. The mean pulmonary artery pressure was 67 mmHg ± 11 (range from 40 mmHg to 90 mmHg). Nine cases had moderate tricuspid regurgitation (TR) while 12 had mild TR.

Mild mitral incompetence was present in 6 cases and mild aortic incompetence in 4 cases. Three cases had mild affection of subvalvular apparatus and any case with severe affection was excluded from the study.

For assessment of usefulness of TEE during closed mitral commissurotomy, the patients were divided into 2 groups:

Group A: comprised 24 patients who underwent the procedure with TEE inserted.

Group B: comprised 22 patients without TEE.

Most of the cases of group A were adults as the TEE probe available was adult size only (mean age was 30 years ± 11, range 19 years - 33 years).

Transthoracic echocardiographic assessment:[5,6,&7]

All the patients were subjected to echo Doppler assessment before operation. Mitral valve area, maximal leaflet separation, condition of the subvalvular apparatus, the presence of mitral incompetence, pulmonary artery pressure, tricuspid incompetence, and mean transmitral diastolic gradient were all measured pre-operatively. The same parameters were measured immediately post-operatively within 15 days of the procedure.

The mitral valve area was assessed by echo Doppler study using 2 techniques and compared with that assessed by the surgeon at operation. These two techniques are:

Pressure half time mettod (PHT)[8]:

Obtained from the slope deceleration of the early peak velocity curve drawing. The deceleration slope starting from the E point.

MVA (cm2) = 220/PHT (msec)

Continuity equation method (Cont) [9&10]:

a. Cross sectional area = 3.14

$$\left(\frac{\text{LVOT diameter}}{2}\right)^2$$

b. calculation of time velocity integral of the aortic valve (TVI-a).

c. Calculation of time velocity integral of the mitral valve (TVI-m).

$$\text{MVA (cm2)} = \frac{a \times b}{c}$$

Transesophageal echocardiography (TEE):[8]

TEE was performed during CMC using a commercially available echocardiography machine interfaced with a 5-MHz transducer, mounted on a flexible endoscope of an adult size which was introduced while the patient was still in supine position, before the insertion of the endotracheal tube and positioned posterior to the left atrium. Anesthesia was then completed and the patient positioned for thoracotomy.

The TEE first assessed the cardiac chambers and valves. The presence of any degree of mitral regurgitation was semi-quantitatively assessed by transesophageal color Doppler echocardiography. Furthermore, maximal mitral leaflet separation i.e. maximal distance during diastole between the 2 tips of mitral valve leaflets was measured.

Closed Mitral Commissuroromy (CMC):[1]

All patients underwent CMC monitored by TEE were performed by the same surgeon. The patients lied in the supine position with right inclination of 30°, an anterolateral thoracotomy at the level of the 6th rib was performed extending 2 finger breadths from the left sternal border tc a point 2 finger breadths below the lower angle of the left scapula. The left hemithorax was entered through the 5th intercostal space. The pericardium was incised 2 cm in front and parallel to the left phrenic nerve. The left atrial appendage and left ventricle were well exposed by stay sutures on the pericardial edges. A 2/0 ethibond purese-string suture was placed on the left atrial appendage, another figure-of-eight 2/0 ethibond suture was placed near th eleft ventricular apex. The left atrial appendage was incised and the index finger introduced into the left atrial cavity for digital assessment of the mitral valve orifice, cusps and commissures. Tubb's dilator was introduced through the left ventriculotomy and graded mitral commissurotomy was performed starting from 2.5 cm and increased by 0.5 to 1 cm increments with repeated digital and TEE assessment. The left atrial appendage incision was secured by running 2/0 ethibond suture and hemostasis was done, closure of the hemithorax was done with a chest tube in place, connected to an underwater seal.

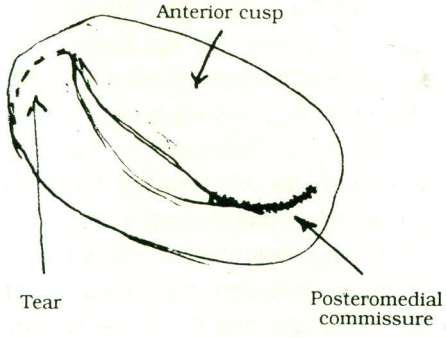
Results

Clinical profile (Sex, age, NYHA functional class, cardiac rhythm, presence of MI,

T.1 Clinical profile of all patients included in our study

Sex	20 males	26 females
Age	15-39 years [27 years + 12]	
NYHA	9 in class II	
	27 in class III	
	10 in class IV	
Rhythm	14 sinus	32 AF
PAP	40-90 mmHg [56 mmHg + 19]	
Presence of TI	Mild in 12 cases	
	Moderate in 9 cases	
	Absent in 25	
Presence of MI	Grade I/IV [mild] in 6 cases	
Presence of AI	I/II in 4 cases	
Affection of subvalvular apparatus	Mild in 3 cases	

Fig.1



AI, TI, or pulminary hypertension are shown in table 1.

During the procedure, 3 cases of group A needed to repeat commissuroromy on instruction of echo operator as on his calculation of transmitral diastolic gradient, it showed that it remained above 10 mmHg. The surgeon also admitted that one of the commissures (the posteromedial in 2 cases and the anterolateral in one case) were still adherent, incompletely opened, careful digital fracture was performed in these and successfully completed the commissuroromy in only 2 cases. Further attempts were denied for fear of cuspal tear.

One case had severe mitral incompetence after commissurotomy which was confirmed by both TEE and the surgeon, the decision of mitral valve replacement was taken and carried out on the same day. On visual assessment of the mitral valve, the anterolateral commissure was totally opened with the posterior cups torn down to the mitral ring about 2 mm from the commissure (fig.1). The posteromedial commissure was almost totally adherent.

In the pre-operative echo studies, in patients of group A, 14 case had dense fibrosis

Table 2 : Subgroups of study patients Group A with TEE, Group B without TEE

		Group A	Group B
No		24	22
Age		19-39 years [28 years ± 11]	14-38 [20 years ± 6]
Sex	males	14	10
	females	10	12
Syptoms	NYHA II	6	3
	NYHA III	16	11
	NYHA IV	2	8
History of hemoptysis		3	4
Rhythm	sinus	8	6
	AF	16	16
PAP		40-90 mmHg [56 mmHg ± 19]	
MI		2	4
TI	mild	6	6
	moderate	3	6
AI		3	1
Affection of subvalvular apparatus		2	1

Table 3 : Compares echocardiographic data pre-operatively and correlates the mitral valve area as palpated by the surgeon intra-operatively

	GROUP A				GROUP B	
	Before		After		Before	After
	Echo	Surgeon	Echo	surgeon	0.8 ± 0.3	2 ± 0.4
MVA (cm2)	0.6±0.4	approx. 0.5	2 ± 0.6	>2		
Mean press. gradient	10-18 [14±4]	-	3-9 [5±4]	-	9-18 [14 ± 5]	4-9 [6 ± 3]
Incidence of MI	2	1 the other denied	4	3 the fourth denied	4	5
Maximal mitral leaflet separation	0.5±0.2	-	1.6 ± 0.6	>2	0.5 ± 0.2	1.5 ± 0.6
Free mobility of the ant mitral leaflet	Restricted	Restricted	Improved	well dooming	Restricted	Improved
Heavy fibrosis	14	14	-	-	9	-
Calcium spikes	3	5	-	-	2	-

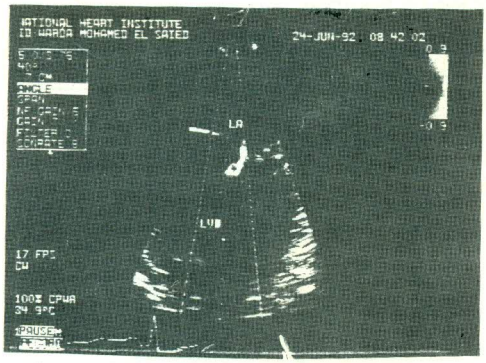


Fig. 2. Mild mitral incompetence jet before commissurotomy confimed by surgeon's digital palpation.

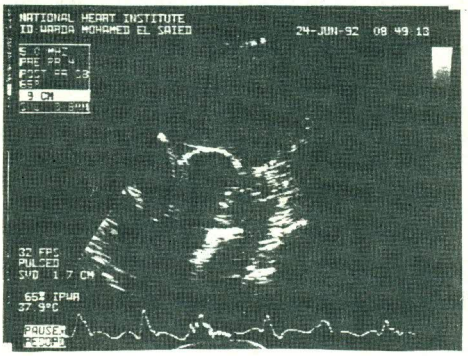


Fig. 4. Pre-commissurotomy digital palpation and assessment of mitral ualve.

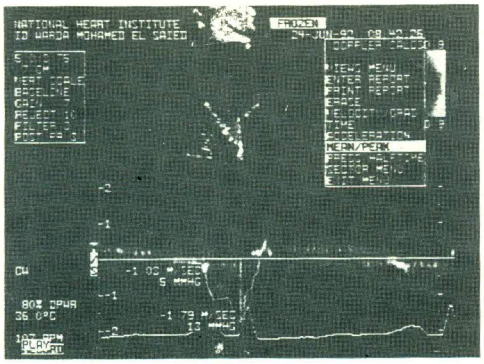


Fig. 3. Pre-commissurotomy Doppler study showing mean and maximum transmitral gradient

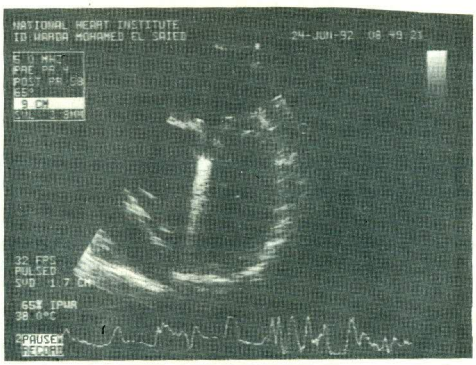


Fig. 5. Tubb's di;ator introduced through the left ventriculotomy and proceed towards the mitral valve orifice.

of the mitral cusps. These data were confirmed by the surgeon who further added that the distribution of fibrosis was either over the tips, the commissures or in the cusp body tissue itself.

The point debate is about the sensitivity of the echo Doppler device to the presence of calcium spikes, calcification and the presence of dense fibrosis, as in 2 cases, calcium spikes, denied by echo study, were

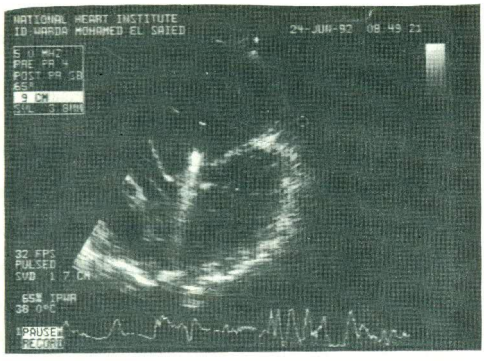


Fig.6. Tubb's dilator positioned in the mitral value orifice.

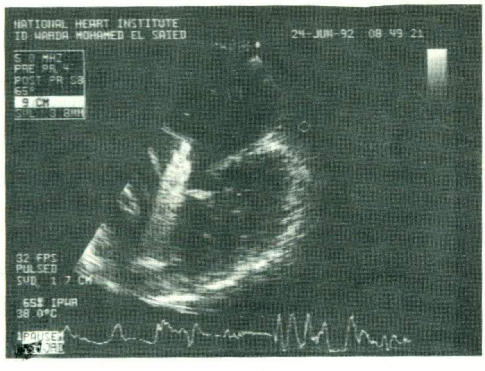


Fig. 7. Start of opening of Tubb's dilator and commissural splitting.

confirmed by the surgeon's digital palpation and futher confirmed on repeated echo post-operatively[11]. Meanwhile, in one case, pre-operative echo study reported the presence of just spikes of calcium on the tip of the anterior mitral leaflet, yet the surgeon palpated dense calcification over the com-

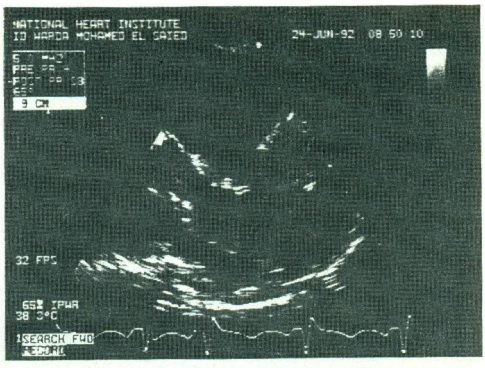


Fig. 8. Maximal leaflet separation seen and free mobility of the anterior mitral leaflet with good subualvular apparatus components.

missure?! The closed precedure in this case was discontinued and an open procedure of mitral valve replacement was done later and the distribution of calcification was confirmed exactly as described by digital palpation by the surgeon[8].

Discussion

During the last decade, many surgeons denied the full usefulness of closed mitral commissurotomy as a solution for rheumatic mitral stenosis. This debate extended nowadays to include invasive cardiologists by the advent of Balloon Mitral Valvuloplasty. The question rises to be, was it the problem of surgical safety or the efficiency of the procedure?. Considering the safety, the morbidity and mortelity rates after mitral commissurotomy is less than 1% in most centers, a percentage accepted internationally. In our study, the use of TEE during the

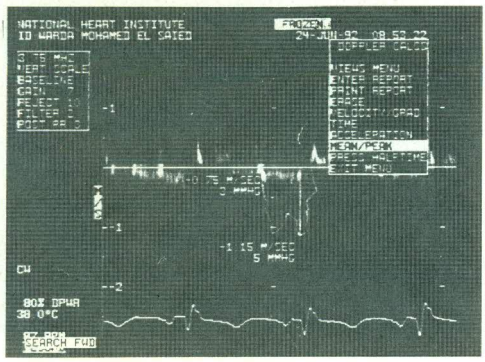


Fig.9. Drop of mean and maximal transmitral pressure gradient after completion of commissurotomy.

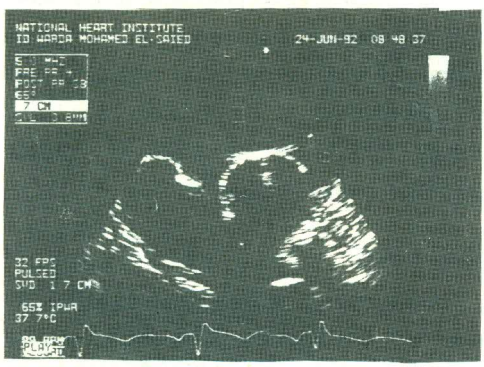


Fig. 10. Post commissurotomy digital assessment of mitral ualve.

procedure confirmed the high efficiency of experienced surgeoun's digital palpation in aseessing the mitral valve both anatomically[12] and functionally[13].

Although TEE has been used during open mitral valve repair to assess its efficiency intra-operatively, no studies have analyzed its usefulness during closed mitral commissuroromy. Closed mitral commissurotomy, yet a simple procedure, still remains dependent on the surgeon's experience. In Egypt, many experienced surgeons perform this procedure with both immediate and long term excellent results (14&15), tiding the patients over for 6-12 years in excellent condition with need of minimal pharmacological control, thus solving the problem of a wide category of patients in developing countries as a cheap and safe procedure needing no sophisticated equipments like that needed in Balloon Mitral Valvuloplasty.

We included in our study 2 groups of patients with tight rheumatic mitral stenosis stenosis who underwent CMV, one was aided by TEE (group A) and the other was not (group B).

In group A, TEE helped in measuring maximal mitral leaflet separation as an index of successful mitral commissurotomy, and the presence and the degree of any mitral incompetence, both of which were recognized by the surgeon almost accurately. Patients in this group also had less incidence of post-commissurotomy mitral regurgitation than those in group B in spite of the larger number of trials for Tubb's dilation and a similar final mitral valve area. In our opinion, this could be explained by the acceptance of the degree of dilation (confirmed by mean pressure drop and mitral leaflet separation) until any whiff

of incompetence appears by TEE that may not be well felt digitally by the surgeon (happened in 2 cases of group A).

TEE had confirmed that digital palpation is an accurate guide[16], as in some cases with efficient mitral valve separation, the surgeon had felt that one of the 2 commissures was not fully opened, and that the calculated orifice area could be increased by trials of digital fracture at the adherent commissures. Furthermore, in the presence of mild mitral incompetence, the degree was confirmed quantitatively by TEE and compared to that experienced by the surgeon.

High pulmonary artery pressure (70-90 mmHg) with severe right ventricular hypertrophy and dilation (3 in group A, 2 in group B), TEE had confirmed a drop in pulmonary artery pressure by a range of 30-50 mmHg within 20 minutes after the commissurotomy denoting an efficient relief of mitral flow.

The point of confusion still remains between digital assessment and echo Doppler study is the pliability of the mitral valve leaflets and extent of calcification or severity of fibrosis in some cases, we suggest repeated echo Doppler studies before surgery in any suspected cases with presence of spikes of calcification, a state in which many experienced surgeons accept a closed mitral commissurotomy to be performed for such patients. A further study is being carried out to assess the efficacy of the procedure for this category of patients in terms of short and long term results[1&16].

In conclusion, Closed Mitral Commissurotomy proved to be an excellent procedure if performed by experienced surgeons in cases with tight mitral stenosis especially those with no subvalvular moderate or severe affection or calcification spikes. Digital palpation of mitral valve apparatus is highly informative and accurate. The use of TEE during the procedure may add to safety and result in selected patients at risk either with the presence of mild mitral incompetence or those with high pulmonary artery pressure.

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Hyperamylasemia and pancreatitis after cardiopulmonary bypass

Abstract

54 Patients who underwent cardiac surgery using cardiopulmonary bypass for Valvular replacement (66.7%) and coronary artery bypass (33.3%) were studied to detect the incidence of clinical or subclinical pancreatitis post operatively.

Determination of the level of serum amylase and clinical examination were the mainstay for diagnosis of panceratitis.

From the study, it was found that hyperamylasemia was common after cardiopulmonary bypass, it's incidence was 61.1%. However, the incidence of overt pancreatitis was much lower (11.1%).

We believe that hyperamylasemia after cardiopulmonary bypass may denote a subclinical affection of the pancrease. So we recommend determination of serum amylase among those patients who had an abnormal clinical course with non-specific abdominal symptoms.

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Introduction

Acute pancreatitis is a reported, but poorly understood complication of cardiac surgery. It's diagnosis and treatment are difficult, since symptoms may be atypical and misleading, often obscured by other complications[1].

The precise incidence of pancreatitis following cardiac surgery is unclear, Early reports suggest a frequency of up to 1%[1,2].

Previous autopsy studies on patients who had undergone cardiac operations showed a higher incidence of pancreatitis. Mild to severe pancreatitis was found in 16% of patients[3]. Post morterm studies found that 11% of patients who died after cardiac surgery had pancreatitis[4].

From the above figures, it is obvious that the frequency of acute pancreatitis after open heart surgery is higher than expected, and the difference can be explained simply by the difficulty of diagnosis and unavailability of specific tests.

Table (1)

Procedure performed	Number of patients	%
MVR	9	16.6%
MVR + TR	12	22%
AVR	12	2%
MVR + AVR	3	5.5%
CABG	18	33%
Total	54	100%

MVR Mitral Valve REplacement.
AVR Arotic Valve Replacement.
TR Tricuspid Repair.
CABG Coronary Artery bypass graft.

In the present study, Serm amylase level was determined, in order to find the percentage of hyperamylasemia after cardio pulmonary bypass and it's relation to clinical and subclinical pancreatic injury.

Materials and Methods

Fifty four adult patients undergoing open heart surgery with cardiopulmonary byass were evaluated.

There were 40 males and 14 females with a mean age of 38 ± 35 years (range from 26 to 55 years).

The operative procedures performed were coronary artery bypass grafts (18 patients), and valve replacement (36 patients).

To determine the prevalance of clinical or subclinical pancreatitis, Serum amylase was determined for all patients before institution of cardiopulmonary bypass, 24 hours and 72 hours post operatively.

Serum amylase level was measured using Enzyline Amylase UV antomates kits from (bio Merieux). Normal level at 37°C varies from 25-125 U/L.

The diagnosis of pancreatic injury was based on both clinical and biochemical data. The presence of abdominal pain, nausea, decreased bowel sounds, with amylase level of 1,000 U/L denotes mild pancreatitis. The presence of these findinges with hemodynamic instability diagnose a severe form of pancreatic injury i.e fulminating type.

So all patients were examined in the early post operative period for the presence of abdominal pain, distension, ileus, vomiting, fever, leucocytosis and haemodynamic par-

Table 2: Pre operative and post operative amylase level (U/L).

		AMYLASE LEVEL			
NUMBER OF PATIENTS	OPERATION PREFORMED	PREOPERATIVE	1ST DAY P.O.	3rd DAY P.O	PATTERN OF PANCREATIC INJURY
1	MVR TR	67	250	137	SUBCLINICAL
2	MVR TR	75	232	138	SUBCLINICAL
3	MVR	52	196	102	
4	AVR	45	184	116	
5	MVR	80	200	156	SUBCLINICAL
6	AVR	63	234	182	SUBCLINICAL
7	CABG	86	229	143	SUBCLINICAL
8	DVR	60	36	100	
9	CABG	45	98	87	
10	AVR	63	70	110	
11	CABG	67	33	99	
12	CABG	43	1300	660	CLINICAL
13	MVR TR	81	2315	710	CLINICAL
14	MVR	82	113	96	
15	AVR	43	41	84	
16	CABG	50	52	95	
17	MVR TR	73	271	139	SUBCLINICAL
18	CABG	65	350	142	SUBCLINICAL
19	MVR TR	67	260	138	SUBCLINICAL
20	MVR TR	75	232	139	SUBCLINICAL
21	MVR	52	160	100	
22	AVR	45	184	117	
23	MVR	80	200	158	SUBCLINICAL
24	AVR	63	234	180	SUBCLINICAL
25	CABG	86	229	145	SUBCLINICAL
26	DVR	60	36	102	
27	CABG	45	98	89	
28	AVR	63	70	110	
29	CABG	67	33	99	
30	CABG	43	1329	665	CLINICAL
31	MVR TR	81	2415	550	CLINICAL
32	MVR	82	113	90	
33	AVR	43	41	82	
34	CABG	50	52	96	
35	MVR TR	73	271	134	SUBCLINICAL
36	CABG	65	350	142	SUBCLINICAL
37	MVR TR	65	258	132	SUBCLINICAL
38	MVR TR	73	235	140	SUBCLINICAL
39	MVR	50	198	101	
40	AVR	43	186	117	
41	MVR	82	201	157	SUBCLINICAL
42	AVR	65	240	183	SUBCLINICAL
43	CABG	87	230	147	SUBCLINICAL
44	DVR	62	37	101	
45	CABG	43	99	87	
46	AVR	65	72	111	
47	CABG	67	33	99	
48	CABG	45	1400	663	CLINICAL
49	MVR TR	80	2440	500	CLINICAL
50	MVR	82	113	96	
51	AVR	43	42	89	
52	OABG	53	55	97	
53	MVR TR	73	271	139	SUBCLINICAL
54	CABG	65	350	142	SUBCLINICAL

MVR = MITRAL VALVE REPLACEMENT
TR = TRICUSPID REPAIR
AVR = AORTIC VALVE REPLACEMENT
DVR = DOUBLE VALVE REPLACEMENT
CABG = CORONERY ARTERY BYPASS GRAFT

Table (3)

Clinical findings	Number of patients	%
1. Early pain Tenderness.	-	-
2. Ileus and abdominal distention.	6	100%
3. Fever > 38 C.	6	100%
4. Late abdominal pain and Tenderness.	6	100%
5. low cardiac output.	6	100%
6. leucocytosis.	6	100%
7. Early Hyperamylasemia.	6	100%
8. late Hyperamylasemia.	3	50%
9. Cardiogenic shock.	3	50%
10. Death	3	50%

ameters for the presence of hypotension, renal dysfunction and the use of inotropic support.

Results

All patients underwent open heart surgery using standered cardiopulmonary bypass.

Myocardial preservation was performed using systemic hypothermia 28°C, oxygenated cardioplegia and local hypothermia using ice slush.

The procedures performed among these patients is shown in (Table 1). Routine medication used in patients with valve replacement was dopamine in a dose of 5 Ug/kg. Those with coronary arterry bypass graft had a combination of Tridil (Nitro glycerin) and Nipride (Sodium nitroprusside) as a coronary and periphral dilator.

Estimation of pre operative serum amylase was in the normal range among all patients, it's level varied from 43 U/L (mean 36 ± 39).

61.1% of patients showed an increase in the levevl of serum amylase postoperatively within 24 hours and the level gradually returned to normal almost in all patients by the 3rd day (Table 2).

Six patients (11.1%) showed a persistent elevation of a mylase level beyond 72 hours.

Patients with elevated amylase level were classified into either clinical or subclinical pancreatic affection according to amylase level and clinical data.

Clinical Pancreatitis

It's diagnosis was based on both clinical and persistent elevation of amylase level. Only six patients (11.11%) were diagnosed as having clinical pancreatitis.

Three patients had mitral valve replacement with tricuspid repair and the rest had coronary artery bypass grafts.

There were three males and three females. Their ages ranged between 31 and 48 years.

All these patients had difficulty in weaning from cardiopulmonary bypass. They received high doses of inotropes in the form of dopamine and adrenalin.

Clinical features among these patients are shown in (Table 3).

The most common symptoms observed were abdominal distention and decreased bowel sounds. These were observed in all six patients within 24-48 hours. On the other hand, abdominal tenderness and pain was observed later on. All patients had low cardiac output starting intraoperatively. Inspite of initial improvement on inotropic support, three patients had subsequent hypotension not responding to the usual treatment. These patients died within 5 days after operation. Only one patient underwent postmortem examination which proved to be severe pancreatitis.

The level of amylase in this group varied from 130 U/L to 2440 U/L at 24 hours postoperatively (mean 1866_50). The level started to decrease gradually but remained at abnormal level, which varied from 710 U/L to 500 U/L at 72 hours (mean 624±66).

It was noted that the three patients who died had a persistent elevation more than 1500 U/L. The other three patients had symptomatic improvement on conservative treatment. These patients were classified as having mild pancreatitis.

Subclinical Pancreatitis

This entity was applied to those who had hyperamylesemia, but showed no clinical manifestation suggestive of pancreatitis, 21 patients (38.9%) showed elevated level of amylase 24 hours postoperatively.

The level varies from 200 U/L to 350 U/L (mean 253±66). The level returned back to normal by 72 hours after operation (mean 184±23).

Discussion

Pancreatitis has been a rare complication of operation not involving direct manipulation of the pancreas.

post pump pancreatitis undergoing cardiac surgery with the use of cardiopulmonary bypass (3,5,6).

The next cause of such a complication is not known. Missavage and colleagues in 1984 proposed a pancreatic ischemia. This was proved experimentally by Feiner in 1976 and Broe in 1982. They found that pancreatic ischemia is secondary to hypoperfusion or microscopic thrombosis which occur during cardiopulmonary bypas. It was found that the pancreas is very sensitive to hypoperfusion which causes marked reduction in pancreatic blood flow, which in turn causes acinar cell damage and release of myocardial depressent factor and proteolytic enzymes[9].

In addition to post operative cardio vascular instability, there is also evidence that cardiopulmonary bypass itself predisposes to pancreatitis. Several studies showed that serum amylase and renal amylase to creatine clearance ratio are increased significantly after CPB[10]. Also the use of non-pulsatile perfusion is associated with elevated amylase level than pulsatile CPB[11].

Pancreatic necrosis was found to occur secondary to hypothermia used in CPB[1].

Other factors that may be involved are atheromatous or cholesterol emboli which may be broken off during cannulation and cross clamping, venous sludging which occurs with extracorporal circulation may injure the pancreas and add to the detrimental effect of the CPB[12].

The diagnosis and treatment of post pump pancreatitis is difficult. Since symptoms may be atypical and misleading, and often obscured by other complications[1].

Prior to 1929, pancreatitis was a postmortem diagnosis. Elman, Arneson and Graham described elevations of amylase level in patients with pancreatic disease and they proposed the use of elevated serum amylase measurements to diagnose pancreatitis.

In our study, diagnosis of pancreatic injury was made on both clinical and chemical estimation of serum amylase.

We found that serum amylase was elevated in 61.1% of patients within 24 hours postoperatively. Those results seem high in comparison to others[14,7], they found an elevation of amylase among 36% and 32% respectively. On the other hand our results were consisent with Leijala and Louhima[15] who found that 33 patients out of 54 had hyperamylesemia postoperatively. The elevation of amylase level was directly affected by the type of operation performed. 49.9% of patients who had coronary artery hypass and 66.6% of patients who had replacement showed elevated amylase. These findinngs were similar to other studies performed[14].

Most patients showed a normal level of amylase after 24 hours, this proved that CPB and hypothermia elevate the amylase level without damaging the pancrease. We were interested in those patients who had persistent elevation beyond that time.

Those patients were categorized as having either clinical or subclinical affection of the pancrease according to Haas study[1].

In 38.9% of our patients, the elevated serum amylase level (mean 253+66) went back to normal within 72 hours without any clinical manifestation suggestive of pancreatitis. These patients were categorized as having subclinical affection of the pancreas, as the level of amylase did not return back to normal within 24 hours.

Evidence of clinical pancreatitis was found in six patients only (11.1%). They had persistent elevation of amylase level (mean 624±66), with symptoms and signs suggestive of pancreatic affection. Ileus, fever, late abdominal pain leucocytosis was found in all patients, while cardiogenic shock was found in 3 (50%) patients only. All these patients had cardiovascular instability and low cardiac output. Our clinical results were similar to those found by others[1,2].

The incidenceof overt pancreatitis after CPB varies greatly. Leijala and Louhima[15], found that 35.2% of their patients had overt pancreatitis. On the other hand, non of the patients in Missavage[7] study had overt pancreatitis inspite that 32% had hypermylesemia.

Our incidence was similar to Svensson[6] who found that 42% of their 135 patients had clinical pancreatitis.

From our study, it is noticed that hyperamylesemia is common in patients undergoing CPB. Although determination of serum amylase can diagnose pancreatitis, misleading results can occur in 30% of cases [7], as many organs contribute to the production of amylase as salivary glands, lacrimal and lung. Also there is a poor correlation between the occurence of postoperative hyperamylesemia and the incidence of overt pancreatitis as seen in our study and in others[6,7]. Although determination of serum amylase to confirm overt pancreatitis is a poor method, it at least helped to find out those patients who had either subclinical or clinical pancreatitis.

We believe that a mild subclinical injury to the pancreas may occur as a consequence of CPB which may progress to severe-ischemic necrosis, if hypoperfusion follows in the post operative period.

So we recommend, that serum amylase assay should be determined among those patients who had an abnormal clinical course with evidence of non-specific abdominal symptoms. Those with elevaled levels should be monitored closely to avoid furthur complications.

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Xenograft valve replaement: ten years follow up

Abstract

Between January 1980 and December 1984, 236 patients had undergone valve replacement by xenografts in the national heart institute, mitral valve replacement (MVR) 120 patients, Aortic valve replacement (AVR) 25 patients MVR+AVR 25 patients, MVR+AVR+Tricuspid valve replacement (TVR) 3 patients, MVR+TVR 12 patients, MVR+TV repair 13 patients, TVR 5 patients 2 of which were with open mitral valvotomy. Mean age was 24.4 years with a female to male ratio 2.6:1. Hospital mortality was 41 patients (17.4%), late mortality of 37 patients (15.7%). Loss of follow up of 31 patients (13.2%). ten years follow up was carried out on 127 patients (53.8%) out of which 31 needed reoperation (24.4%). Functionally, 20 patients (15.7%) of those followed up have an excellent quality of life, 76 patients in functional class II and III NYHA (56.8%) and controlled on anti failure medical treatment. Sixty nine patients (54.3%) are kept on anticoagulation (64 due to chronic atrial fibrillation and 5 due to thromboembolic complications). The results are almost equal in cases whether Carpentier-Edwards or Hancock tissue bioprosthesis was implanted.

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Introduction

Valve replacement as a way for management of degenerated native cardiac valves had been adopted since 1954 (Starr 54). the choice of valve substitute is depending on matching patient parameters to prosthetic valve characteristics in order to minimize morbidity and mortality, including the need of reperation. The thromboembolic complications and the necessity of continuous anticoagulation after prosthetic valve replacement encouraged the production and use of tissue bioprosthesis[1]. Many models have been introduced since 1956. At first these valves were sterilized by formaline, which dissolved the cross linkage of the collagen component with a resultant high failure rate[2]. Carpentier et al., then developed the process of fixation and sterilization of porcine bioprosthesis using a diluted Glutaraldhyde solution, which appeared to promote the stability of the collagen cross linkage in the valve cusps[3]. the valve was then mounted on a semiflexible stent. All tissue valves have the following advantages

- Absence or minimal hemolysis.

- Absence or short term anticoagulation.

- Lower incidence of thromboembolic complications.

- Good flow characters with minimal pressure gradients.

- Structural deterioration when it occurs, is relatively slow and allows elective intervention.

After early enthusiasm, long term follow up revealed that the major disadvantage of tissue valves is their susceptibility to degeneration of its cusp tissue thus affecting its long term function and durability[4].

Methods

During the period between January 1980 and December 1984, 236 patients were operated upon at the National Heart Institute for valve replacement, in which 291 bioprostheses was used either solely or in combination with artificil prosthesis or valve repair.

This study was designed to follow up patients for 10 years postoperatively assessing the patients quality of life, tissue valve function, need of pharmacological treatment (antifailure-anticoagulants) and life span, our patients were divided into 3 subgroups (Table 1), denoting type of operation performed.

Preoperatively all patients were examined clinically, and investigation included ECG, X ray, echocardiography and cardiac cathetrization which was routine at that time for all patients with valvular heart disease.

Patients in our study were 170 females and 66 males (ratio 2.6:1), aged between 14 and 45 years (mean 24.5 yrs) at the time of surgery. Clinically patients symptoms were classified according to NYHA as 92 in functional class II (38.9%) all of which were suffering of isolated aortic valve disease and/or combined with moderate MI, 121 in functional class III (51.3%) and 23 cases in functional class IV (9.7%). All patients were on antifailure measures (diuretics, lanoxin) preoperatively except 6 patients 2 of which were with isolated Aortic stenosis and 4 patients with severe aortic incompetence with a main complaint of palpitation and anginal pains. ECG analysis of all patients showed that 51 patients were in sinus rhythm while 185 in atrial fibrillation, left ventricular hypertrophy in 78 patients (33%), 103 with right ventricular hypertrophy (43.6%), 14 with biventricular hypertrophy (5.9%) and 42 with P. pulmonal (17.8%).

X-Ray examination of patients included in the study revealed an increased cordiothoracic ratio in 218 cases, (92.4%) pulmonary venous congestion in 149 case (63.1%) and double right contour denoting left atrial enlargement in 139 cases (58.9%).

Echocardiographic preoperative assessment confirmed clinical diagnosis and cardiac chamber dimensions were noted (Table 2 & 3).

Echo study revealed that 1 case of AVD was a bicuspid valve with PW thickness 1.2 cm. and 9 cases with calcified aorta, twenty nine cases with densely calcified mitral lea-

Table 1.

Group	Opration	No.	%
Group A	**Single valve replacnt** * AVR * MVR * TVR	**150** 25 pt 120 pt 2 pt	652%
Group B	**Double valve surgery:** * AVR (xeno) + MVR (xeno) * AVR (xeno) + MVR (prosthetic) * MVR (metal) + MVR (XENO) * AVR (XENO) + TVR (XENO) * AVR (XENO) + M. repair * MVR (xeno) + T. plication * TVR (xeno) + M. repair	**83** 34 ptn 7 ptn 9 ptn 12 ptn 5 ptn 13 pyn 3 ptn	34.1%
Group C	**Triple valve operation** * AVR (xeno) + MVR (xeno) + TVR (xeno) * AVR (xeno) + MVR (xeno) + T. plication	6 3 3	3.2%

A.VR: Aortic valve replacement

MVR Mitral valve replacement

TVR Tricuspid valve replacement

Table 2. Echocardiographic diagnosis

Echodiagnosis	**No. of cases**
Aortic stenosis (A.S)	2
Aortic incompetence (A.I)	23
Aortic incompetence + stenosis	4
Aortic incompetence + M. regurge	34
Mitral incompetence	118
Mitral stenosis	12
Mitral stenosis + T. regurge	2
Mitral incompetence + T. regurge	11
Mitral valve disease + T. Valve disease	3
Aortic incompetence + M.S. + T.I.	4
A.S. M.S + TS	2

Table 3. Chamber dimensions.

	MVD	AVD	AVD + MVD	MVD + TVD
LVED	4.1 cm ± 0.6	7.6 ± 0.7	5.8 ± 1	5.6 ± 0.7
LVES	2.0 cm ± 0.7	4.2 ± 0.7	3.4 ± 0.6	3.8 ± 0.6
Ao root	2.1 cm ± 0.3	2.5 ± 0.4	2.3 ± 0.5	2.2 ± 0.3
LA atrium	4 cm ± 0.9	3 ± 0.3	3.8 ± 0.6	4.2 ± 1
RV	2.3 ± 0.9	2.1 ± 0.3	2.1 ± 0.4	2.6 ± 0.4

Table 4. Over all results

	Surviving (127 ptn)				Mortality (78 ptn)		Reoperation (31 ptn)
	Total	NYNA I (20 ptn)	NYHA II (58 ptn)	NYHA III (18 ptn)	Early (41 ptn)	Late (37 ptn)	
Group A	82	18	31	3	14	14	12
Group B	45	2	27	15	22	22	19
Group C	-	-	-	-	5	+	-

flets and 69 cases with dense fibrosis and calcific tips, almost all cases with mitral valve disease had moderate to severe affection of the mitral subvalvular apparatus. Twelve cases with mitral valve disease were suspected to have a left atrial thrombus.

Cardiac catheterization was routinely done at that time for patients with Rheumatic valvular affection, all data obtained confirmed the clinical and echocardiographic data and added to the study pulmonary artery pressure which was as follows.

< 30 mmHg 37 cases (15.7%)

30-50 mmHg 166 cases (70.3%)

> 50 mmHg 43 cases (18.2%)

All patients were submitted to open heart operation using standared techniques venous cannulation by 2 separate venacaval cannulas and snares connected to a roller pump extracorporial machine and standard bubble exygenator was used, hemodilution and hypothermia down to 25°C and myocardial protection by St. Thomas II crystalloid cardioplegia. Cross clamp time ranged from 48-184 mins (mean 89 min) and total bypass time 70-249 mins (mean 114 min) postoperatively all patients were keept on controlled ventilation for at least 12 hours.

Table 5. Cause of early and late mortalities.

Early mortality (41 pt)		Late mortality (37 pts)	
No.	Cause of death	No.	Cause of death
6	Postoperative bleeding	2	Thromboembolism
16	Low cardioc output	1	Cerebral hoemorhage
6	Endocarditis	10	Endocarditis
3	Mediastinitis	16	Intractable heart failare
2	Intractable arrhythmias	4	at reoperation
8	Intractable congestive heart failure	4	unknown

During the period of 10 years follow-up, all patients were followed at the out patient clinical examination, X ray, ECG, anticoagulation monitoring and echocardiographic studies if needed.

Results

Out of 236 patients included in our study only 127 patients (53.8%) were surviving and followed up for 10 years, there was a total of 78 mortalities (33.1%) of which 41 were early mortality (17.4%), and 37 cases (15.7%) with late mortality. Thirty one cases were lost to follow up (Table 4).

127 patients -- survived 10 yrs or more

41 patients -- early hospital mortality

37 patients -- mortality during followup <10 yrs.

31 patients -- loss of follow up.

236

Surviving patients (127 case):

Twenty patients (15.7%) were having an excellent symptom free life. Seventy six patients (56.8%) leading a better quality of life (NYHA II-III), with improved ECG, X ray and echodoppler data but with a need of persistant antifailure treatment. Thirty one patients (24.4%) needed a reopeation due to valve related complications viz: two cases with paravalvular leak, both were cases with mitral valve xenograft replacement. Two patients suffered from infective endocarditis confirmed by repeated blood cultures, both patients were kept on intensive triple antibiotic treatment for 3 weeks and reoperation was carried out just after subsidence of fever by one weak and repeated echodoppler studies revealed the presence of vegitations on top of the bioprosthesis. Twenty seven patients needed reoperation for primary bioprosthesis tissue failure which was degeneration, regression and regurgitation in 18 cases, calcification with decreased leaflet

excurtion in 5 cases and perforation of cusps in 4 cases.

Mortality (Table 5)

Total: 78 patient (33.1%) of which 17.4% (41 cases) suffered early hospital mortality and 15.7% (37 case) late mortality during the period of followup. Analysing the causes of mortality it revealed the following:

(a) All 6 patients with triple valve surgery (study group c) died in the early postoperative period, 5 of which due to low cardic output state that was explained by the operating surgeon being a result of prolonged cross clamp time. The 6th cases was due to persistant postoperative medical bleeding inspite of repeated blood transfusions; Consumption coagulopathy was diagnosed.

(b) Fifteen patients died following mitral + aortic valve replacement (group B) of which:

- five patients having AVR by Bjork shily and MVR using Carpantier Edwards Bioprosthesis.

- Two patients having AVR by Starr Edwards prosthesis and MVR using Carpantier Edwards Bioprosthesis.

- Seven patients with AVR + MVR using bioprosthesis xenograft in both positions.

- One patient with AVR bioprosthesis and Starr Edwards valve at the mitral position.

(c) Six Cases died following mitral valve replacment and Tricuspid valve replacment both were xenografts. and one case had a mitral valve replacement and tricuspid plication (group B)

(d) Twelve patients died having isolated mitral valve replacement (group A)

(e) Two patients who underwent isolated tricuspid replacment died early postoperatively.

Discussion

In the three decades that have elapsed since the introduction of successful intracardiac valve replacement[5&6] thousands of lives have been prolonged and improved by prosthetic valves. During this same period, the limitations of valve design and complications associated with the use of these devices have also been extensively documented and investigated, with significant improvements in valve engineering and composition. Unfortunately, the perfect valve substitute has yet to be developed. From literature, the survival in the patient with the currently used mechanical valves is 65% at the Aortic or mitral position for 10 years [7,8&9] and over 50% of these deaths was due to prostheses related complications. The advent of Bioprotheses aimed at decreasing the thromboembolic complications which is one of the most important drawbacks of (10&11).

In the early eighties, Glutaraldhyde-fixed stent-mounted Bioprostheses was used extensively at the National Heart Institute, our study was designed to evaluate, those patients in whom these valves were implanted both functionally and the incidence of complications 10 years postoperatively.

In our study, the mortality rate was almost as that presented by many other studies (12,13&14). In our study it was 33.2%, the highest incidence was in patients with triple valve surgery, this is explained by the pre-operative patients clinical condition and operative prolonged cross clamp time.

A major advantage of Bioprosthesis is its low embolic rate without routine use of anti-coagulants. However, long-term durability remains in question, In our study 31 patients (24.4%) needed reoperation to re-replace the xenograft by mechanical prosthesis, this was needed in the range between 4-10 years (mean 6 years) which is quite a low durability rate compared to that expressed in other studies [15,16&17], this may be explained by the need of valve replacement in Egypt in a younger age group (mean age 24.5 yrs) with higher incidence of rheumatic exacerbations and a higher incidence of calification, the same data were expressed by Jamieson 1988 who documented the influence of age on primary tissue failure (structural valve deterioration) as the most prominent complication of porcine bioprosthesis, showing that the freedom from deterioration at 10 years time for patients less that 30 years of age is 26.8±17.2% where in patients more than 30 years was 77.4±3% (i.e.: reversed incidence)[15].

Concerning hoemodynamic and functional analysis of Bioprosthesis, many studies have demonstrated that porcine xenografts have reasonable hoemodynamic function, while moderate gradients have been reported in the smaller annular sizes. In our study Carpentier-Edwards and Hancock porcine xenografts were used, the clinical results of both types are satisfactory. However, a careful hoemodynamic evaluation of these valves in both Mitral and Aortic positions has demonstrated that the Carpentier-Edwards porcine bioprosthesis has a lower transvalvular gradients and larger calculated orifice areas than the Hancock porcine valve[18].

Thromboembolic complications were seen in our series in the rate of 2.7% (7 cases), 3 cases fatal, 2 cases major disability (hemiplegia), 2 cases minor complication. The routine in our institution is to keep the patients on anticoagulation (warfarin) for life whenever atrial fibrillation is present; our results were comparable to other studies, in different centers[18].

Tricuspid valve replacement remains to be a difficult task with high mortality rate both operative and postoperative. In our study this was performed in 23 cases with 5 cases mortality (21.7%) which is an acceptable rate[20].

From our study and most of the published results gathered in different centers and different time frames, the late outcome of heart valve replacement can be determined by subjective improvement, improvement of functional capacity and central hoemodynamics, normalization of impaired ventricular functions and by the frequency of complications related to/or induced by the prostheses. Subjective and functional capacity improve-

ment is obviously dependent on the postoperative normalization of the homodynamics[21]. The hoemodynamic properties of modern mechanical prostheses are superior to those of tissue valves because of the significantly more favorable relation between total prosthetic valve area and effective prosthetic valve orifice area, conditioned by design[21].

The main disadvantage of biological valves is their limited durability due to tissue failure resulting in dysfunction. The main advantage is it's less thromboembolic complications and the absence of postoperative anticoagulation, a point that is not adventageous due to the prevalence of patients in atrial fibrillation in our study (78.4%) in whom oral anticoagulation was obligatory. The risk of homodynamica deterioration with consecutive decrease of their functional capacity must be expected a considerable time before a second operation is mandatory.

In conclusion, weighing advantages and disadvantages of both mechanical and tissue valves, a differential therapy and an individualized approach should be preferred. Tissue bioprostheses is recommended for patients in sinus rhythms, females in child bearing periods or in elderly patients over 60 yrs. Tissue valve may play a role also in cases where a proper anticoagulation regimnen and follow up is difficult.

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Acute renal failure following open heart surgery

Abstract

During a 4 year period, 4995 open heart procedures were performed in the Clinics of the Technical University, Aachen; Federal Republic of Germany.

Among these patients, 81 (1.7%) had renal replacement therapy due to acute renal failure postoperatively, with 51 males and 30 females. The mean age of the patients was 61.7 years.

The incidence and the mortality rate of acute renal failure for aortocoronary bypass graft was 0.9% and 75% respectively, for valve replacement was 2,6% and 57% respectively, and for combined aortocoronary bypass graft and valve replacement was 4.5% and 73% respectively.

Preoperative features of these patients requiring postoperative renal replacement therapy included mild to moderate renal failure in 56% of patients, moderate to severe left ventricular dysfunction in 56% of patients, and enlarged right ventricular size, denoting pulmonary hypertension in 42% of patients.

Screening the intraoperative features, the bypass time was 155 +/- 85 min and the aortic occlusion time was 75 +/- 29 min.

Severe systemic hypotension occurred in 67% of these patients, in 87% of patients catecholamine support had to be administered perioperatively, and in 45% of patients Intra-aortic balloon counterpulsation was necessary.
Various procedures of renal replacement therapy were used isolated or in combination: continuous arteriovenous hemofiltration, intermittent mechanical hemofiltration, and hemodialysis.
55 patients (68%) died as a result of their acute renal failure, with no relation to the type of renal replacement therapy used.

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Introduction

Acute renal failure ia a serious complication of cardiac surgery using cardiopulmonary bypass. With the increasing performance of heart operations, the problem of postoperative renal failure has become more important during recent years.

Table 1. Acute renal failure following cardiac surgery; incidence and mortality rate

	Incidence, % (n)	Mortality,%
Doberneck et al, 1962 (1)	3.0 (30/1000)	87
Yeh et al, 1964 (2)	5.6 (10/180)	10
Porter et al, 1967 (3)	2.9 (6/209)	68
Johansson et al, 1967 (4)	3.1 (13/423)	92
Porter and Starr, 1969 (5)	1.3 (12/911)	67
Yeboah et al, 1972 (6)	4.7 (20/428)	70
Abel et al, 1976 (7)	3.6 (18/500)	89
Bhat et al, 1976 (8)	4.3 (21/490)	67
Krian, 1976 (9)	5.3 (157/2945)	72
Mc Leish et al, 1977 (10)	1.6 (25/1542)	28
Hilberman et al, 1979 (11)	2.5 (5/204)	65
Gailiunas et al, 1980 (12)	1.5 (11/752)	27
Heikkinen et al, 1985 (13)	0.9 (15/1686)	67
Koning et al, 1985 (14)	1.9 (27/1403)	--
Morgan et al, 1988 (15)	0.8 (10/1600)	--
Average	2.7 (380/14273)	61

Several studies dealing with acute renal failure after cardiac surgery have been pubilshed (1-16); a survey is shown in table 1. The data are difficult to compare because of marked differences in definitions and selection criteria. Altogether 380 patients were evaluated; the incidence of severs renal failure was similar for all investigators ranging from 0.8 to 5.6% with 2.7% on average. The majority reported a fairly high mortality rate of 65-92%[1,3-9,11,13,-16]; only three groups found substantially better results [2,10,12].

A critical circulation caused by different factors is reported to play an important role in the pathogenesis of acute postoperative renal dysfunction[16]. In order to define risk factors we analyzed a series of clinical variables in a large group of patients with serious acute renal failure following open heart surgery.

Patients and Methods

In this study, acute renal failure requiring renal replacement therapy occurred in 1.7% among nearly 5000 patients undergoing open heart surgery. In total we studied 81 patients -51 males and 30 females - with a mean age of 61.7 years. The incidence of acute renal failure was higher in valve replacement as compared to cronary bypass operation (2.6 versus 0.9%); with a combined surgical procedure it even increased to 4.5% (table 2). The mortality rate corresponded well with the data known from the literature (table 2).

Table 3 shows types of surgical procedures. Valve replacement and bypass operation were equally distributed. Fourteen percent of all operations were performed as

Table 2. Acute failure following cardiac surgery: incidence and mortality rate (present series) (total number of operations: 4995)

	Incidence, %	Mortality, %
All cases (n=81)	1.7	68
Aortocoronary bypass Graft (ACBG)	0.9	75
Valve replacement (VR)	2.6	57
ACBG + VR	4.5	73

Table 3. Acute renal failure following cardiac surgery: types of surgical procedures

Surgical procedure	%	n
Aortocoronary bypass graft (ACBG)		28
Single ACBG	4	
Double ACBG	32	
Triple ACBG	32	
Four or more grafts	32	
Valve replacement (VR)		28
Aortic VR	39	
Mitral VR	39	
Double VR	22	
Combination of ACBG and VR		22
Miscellaneous		3

an emergency intervention, either for unstable angina pectoris or advanced heart failure or acute endocarditis.

Results

Preoperative impairment of renal function was a surprising but frequent finding. Although it was mild in most patients, there were some with creatinine levels exceeding 300 umol/1 (table 4).

Only 39% exhibited completely normal renal function.

Pre-existing heart failure was also frequently observed. According to the NYHA criteria, one third of our patients had to be classified as advanced congestive heart failure (table 5). This was confirmed by routinely obtained chest X-ray; signs of right-sided, left-sided or biventricular heart failure were demonstrable in a total of 51% Global left ventricular performance was calculated during echocardiography by measuring ejection fraction, fractional shortening and end-diastolic diameter. The degree of left ventricular dysfunction was moderate in 33% and severe in 23% (table 6). Furthermore, enlargement of the right heart cavities

Table 4. Acute failure following cardiac surgery: pre-operative renal function

Renal function	%
Normal renal function (creatining < 100 micromol.1)	39
Mild renal failure (creatining < 100 micromol.1)	46
Moderate renal failure (creatining < 100 micromol.1)	10
Severe renal failure (creatining < 100 micromol.1)	5

Table 5. Acute renal failure following cardiac surgery: preoperative classification of heart failure according to the NYHA criteria

Classification	%
NYHA I	2
NYHA II	18
NYHA III	47
NYHA IV	33

Table 6. Acute renal failure following cardiac surgery: preoperative echocardiographic features

Echocardiographic feature	%
Left ventricular function	
Normal	44
Moderate dysfunction	33
Severe dysfunction	23
Right ventricular size	
Normal	58
Enlarged	42

Table 7. Acute renal failure following cardiac surgery: preoperative rhythm disturbances

Rhythm disturbance	%
Sinus rhythm	69
Atrial fibrillation	31
Infrequent ventricular ectopics	58
Frequent ventricular ectopis	42

was observed in more than 40% indicating pre-existing pulmonary hypertension.

The main preoperative electrocardiographic features were ischemia plus hypertophy and myocardial infarction in the past with 38 and 32% respectively. With regard to rhythm disturbances, our data are summarized in table 7. Atrial fibrillation was present in nearly one third of our patients.

With cardiac catheterization including selective angiography of the coronary arteries the most common pathological finding was a triple vessel disease (25%) followed by a double and a single vessel disease (16% and 11% respectively). A main stem stenosis of the left coronary was observed in 9% of the patients. In 39% of our patients we could not reveal any significant obstructive lesions within the main branches of the coronary arteries. The values of some important hemodynamic variables are depicted in table 8. The mean cardiac index was low, but still within the normal limits. The distribution within our group, however, shows a diminished cardiac index in 32%, the corresponding and simultaneously recorded left ventricular end-diastolic pressure was elevated in 70% of the patients. Moreover, an increase in mean pulmonary artery pressure was found in 60% with an average value amounting to 28 mmHg. This hemodynamic pattern indicates a significant impairment of left ventricular function as well as additional increase in pulmonary vascular resistance.

Some intraoperative and early postoperative clinical data are presented in table 9:

Table 8. Acute renal failure following cardiac surgery: preoperative cartheterization data

Cardiac catheterization data	%
Left ventricular enddiastolic pressure	
17 +/- 10 mmHg (mean +/- SD)	30
Normal	70
Elevated	
Cariac index	
2.7 +/- 0.7 l/min-m2 (mean +/- SD)	
Decreased	32
Normal	66
Increased	2
Mean pulmonary artery pressure	
28 +/- 14 mmHg (mean +/- SD)	
Normal	40
Elevated	60

Table 9. Acute renal failure following cardiac surgery: intraoperative data

Total duration of the operation, min	271 +/- 118
Cardiopulmonary bypass time, min	155 +/- 85
Aortic cross-clamping time,min	75 +/- 29
Lowest systolic intraoperative blood pressure, %	
>120 mmHg	1
101-120 mmHg	4
81-100 mmHg	24
60-80 mmHg	53
<60 mmHg	18
Intra - and early postoperative complications, %	
No complications	8
Severe systemic hypotension	67
Recurrent ventricular fibrillation	3
Hypotension + ventricular fibrillation	22
Intraoperative administration of catecholamines, %	
No catecholamines	5
Epinephrine	8
Epinephrine + dopamine	87
Use of Intra-aortic balloon pump (IABP), %	
IABP support	45
No IABP support	55
Intraoperative substitution of blood components, %	
Packed red blood cells or whole blood	
No transfusion	
1-5 units	10
6-10 units	78
> 10 units	9
Freesh frozen plasma	3
No substitution	71
1-3 units	24
> 3 units	5

the relatively long duration of the operation in total, and of cardiopulmonary bypass and aortic cross-clamping in particular, can be interpreted as a general indicator of major intraoperative problems. Corresponding to this finding severe systemic hypotension with temporary systolic blood pressure below 80 mmHg was observed in more than 70% of our patients. This unstable hemodynamic condition was furthermore complicated by recurrent ventricular fibrillation. Despite additional application of catecholamines, treatment with the intra-aortic ballon pump was required in 45% of patients. Intraoperative bleeding complicaions with consecutive systemic hypotension are one of the most important causes of postoperative renal failure. This is reflected by the large number of patients who received substitution of blood components during cardiac surgery.

Discussion

Various factors may contribute to a critical circulation during and after cardiac surgery thus leading to acute renal failure. A prolonged period of cardiopulmonary bypass is generally considered to be one of the major risk factors in the development of postoperative renal dysfunction[1,3,6-9,11,13,14]. The duration of aortic cross-clamping, the total duration of the operation and pre-existing renal damage also closely correlated with the incidence of acute renal failure[3,6,7,9]. The deleterious effect of systemic hypotension on renal perfusion is well known[1,8,13,14]. The significance of several other parameters, such as age, type of operation, NYHA classification, ventricular function, and left ventricular end-diastolic pressure, remains controversial [1,3,6-9,11,13,14]. Some of these previously published potential risk factors, however, have only been studied in small numbers of patients. In our group of 81 patients we frequently found a preoperative impairment of renal function and signs of left-and right-sided heart failure shown by different methods of examination.

With regard to the poor prognosis of acute renal failure following cardiac surgery, Abel et al[7] pointed out that therapy of this postoperative complication, therefore, appears to be better directed toward its prevention rather than treatment once established.

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Segmental annuloplasty versus De-vega annuloplasty in tricuspid regurgitation.

Abstract

Between January, 1991 and March, 1994; 66 patients with rheumatic valvular heart disease associated with significant tricuspid regurgitation (TR), underwent mitral valve replacement or repair and tricuspid valve repair in all cases. There were 39 females (59.1%) and 27 males (40.9%). Their ages ranged from 19 to 54 years (mean 27.2 years + 6.5).

Segmental tricuspid annuloplasty (STA) was done for 36 patients (group A). The results were compared with De-Vega tricuspid annuloplasty (DTA) which was done for 30 patients (group B).

Group A: by one month follow up, thirty-one out of 36 patients (86.1%) had competent valves, the remaining 5 patients (13.9%) had grade I/IV TR. After six months of follow up, no changes in their results were noted.

Group B: One month after the operation, twenty-four patients out of 30 (80%) had competent valve while 6 patients (20%) had grade I/IV TR. By the sixth month of follow-up of these six patients with trivial TR, 3 patients had still grade I/IV TR, 2 patients developed grade II/IV TR and I patient developed grade III/IV TR.

Follow up of the other patients who had competent valves in group B revealed that one patient developed grade III/IV TR and another patient developed severe form of regurgitation (grade IV/IV) and needed urgent surgical interference.

Therefore, we conclude that STA is the procedure of choice for correction of significant TR, as the results are well maintained several months after surgery. DTA although shows good immediate post-operative results, does not maintain these results for long as evidenced by follow up.

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Introduction

As regard the TR, the conservative attitude was the usual policy even with severe degrees of TR believing that it will regress or even disappear after mitral valve correction[1]. Furthermore, after a lot of mitral valve replacement procedures, some patients failed

to improve or even deteriorated and succumbed out of progressive right ventricular failure from severe TR. This explains the more aggressive attitude adopted nowadays towards the diseased tricuspid valve. This started by tricuspid valve replacement by prosthetic valves used for the mitral valve [2]. The results were very unsatifactory, but set the stage for the newer techniques of tricuspid valve correction[3].

Acquired tricuspid valve disease is classified surgically as either functional or organic[4]. The degree of functional impairment is related to the severity of the left sided lesions, and it is duration, as well as the degree of pulmonary artery hypertension and the degree of right ventricular dilatation[4].

So, three general techniques for tricuspid annuloplasty used to correct functional TR are:

1. Annular plication.
2. Annular ring insertion.
3. Semicircular annuloplasty.

a. De-Vega technique (introduced by De-Vega., 1972).

b. Segmental technique.

The aim of all methods are directed to:

1. Narrowing the dilated tricuspid annulus.
2. Maintaining leaflet length and function.
3. Preserve the course of the conductive system from atrium to ventricle[5].

Patients and Methods

This study included 66 patients with rheumatic mitral valve disease who underwent mitral valve correction or replacement with or without aortic valve replacement, and tricuspid repair in the National Heart Institute and Naser Institute in the period between (January, 1991 and March, 1994).

Our patients received mechanical valve prostheses in the mitral position or repair, with or without aortic valve replacement, and had tricuspid valve repair by DTA, in 30 patients and STA in 36 patients.

All the patients were subjected to elective surgical correction after clinical assessment as well as routine investigations for open heart procedure, including ECG, chest x-ray, echocardiography and Doppler study,complete hematological and biochemical study, including complete blood picture, ESR, coagulation profile, serum electrolytes, kidney and liver function tests and urine analysis.

Post-operative clinical, radiological, ECG, and echocardio-graphic data were reviewed and assessed in all patients.

Indication for surgery

A. Pre-operative

1. The severity of the symptoms and clinical signs.

2. Duration of the TR and duration of pulmonary artery hypertension.

b. During operation

1. Enlarged right atrium, thinning out of its wall, with enlarged vena cava means significant disease.

2. Estimation of the tricuspid valve by palpation through the right atrial appendage.

3.High oscillation of the blood in the superior vena caval canula when inserted in the right atrium.

Operation

General anesthesia, median sternotomy, extracorporeal circulation with moderate hypothermia down to 28°c, bubble oxygenator was used, venting through the pulmonary artery, myocardial protection was achieved by intermittent deep selective hypothermia and dilute blood cardioplegia every 20 minutes given in the aortic root or directly into the coronary ostia.

The mitral valve was explored through left atrial or trans-septal approach. While exploring the tricuspid valve, superior and inferior vena caval snaring was needed with right atrial approach.

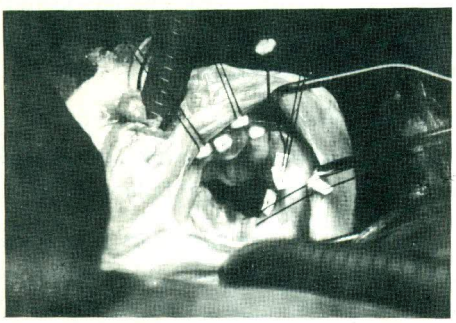

Fig.1

Surgical Technique

The tricuspid valve was repaired by semicircular annuloplasty after declamping of the aorta or beating heart in 60 patients and on the arrested heart in 6 patients.

De-Vega Annuloplasty

In this method, the right atrium is opened in a longitudinal direction. A double armed needle suture of Ethibond 2/0 is started at the anteroseptal commissure and good bites are taken forward every 5 to 6 mm, around the annulus. It is passed around to encircle the base of the anteroseptal commissure, anterior leaflet, anteroposterior commissure, posterior leaflet, posteroseptal commissure and about a half centimeter of the septal ring. The second needle is passed through a teflon pledget, then courses the same way 2 mm parallel to the first to come out through another pledget on the other end. The purse string suture is then tied up with two fingers inside the orifice (or sizer 31) to ensure that the proper diameter is produced (figure 1).

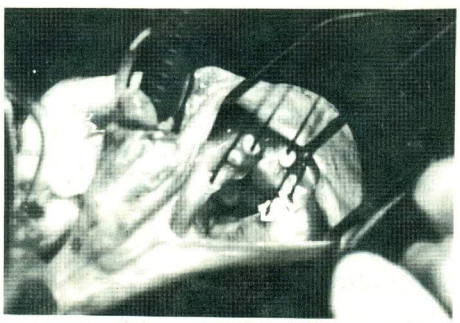

Fig.2

Segmental Tricuspid Annuloplasty

After having the tricuspid valve carefully visualized and the tricuspid leaflets inspected, the tricuspid ring is identified by gentle traction of the anterior leaflet of the tricuspid valve with the valve hook. Then the first stitch

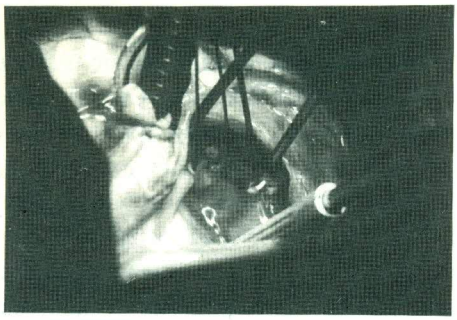

Fig.3

of pledget-supported 2/0 Ethibond suture is placed in the region of the posteroseptal commissure to protect the coronary sinus from being injured. The next series of stitches are placed in counterclockwise manner. The interrupted stitches follow the line that demarcates the tricuspid ring and are placed until the region of the anteroseptal commissure is reached. In general, 5 or 6 stitches need to be placed to complete the annuloplasty. The annuloplasty must include the portion of the annulus corresponding to the anterior and posterior leaflets of the tricuspid valve (figure 2).

Valve testing

When the annuloplasty is complete, competence of the tricuspid valve must be tested by infusing saline solution into the right ventricle via the tricuspid valve. After cardiopulmonary bypass is discontinued, competence of the tricuspid valve is assessed by palpation (intra-operative, transesophageal echo Doppler was used in one case only).

Results

Semicircular tricuspid annuloplasty was done in 66 patients over a period of 39 months. All of our patients had rheumatic heart disease.

Thirty nine patients (59.1%) were females and 27 (40.9%) were males, with a ratio of 1.4:1. Their ages at the time of operation ranged from 19 to 54 years (mean 27.2 years ± 6.5).

The symptoms and signs of right sided decompensation, especially congested pulsating neck veins and enlarged liver with hepatojugular reflux were found in all cases (the congested liver varied from 1 to 6 patient's own fingers).

From laboratory data, the pre-operative blood urea level ranged from 32 to 64 mg/dl (mean 42.2 mg/dl ± 3.6). The pre-operative serum creatinine level ranged from 0.9 to 1.9 mg/dl (mean 1.2 mg/dl ± 0.3).

In the post-operative period, the laboratory data reversed to normal levels after 2 weeks (except one case of renal failure).

The pre-operative pulmonary artery pressure as estimated by echo Doppler ranged from 45mm Hg to 87 mmHg (mean 62.3 mm Hg ± 6.3).

The patients were classified into two groups on the basis of the type of surgical repair of the tricuspid valve:

Group A: comprised 36 patients (54.5%) for whom segmental tricuspid annuloplasty was done.

Group B: comprised 30 patients (45.5%) for whom De-Vega tricuspid annuloplasty was done.

In group: A, two patients had aortic valve replacement in addition to mitral valve replacement, while in group B, 4 patients had aortic valve replacement as well.

Group A: One month follow-up on the patients in this group revealed that 31 patients out of 36 (86.1%) had competent tricuspid valve, 5 patients had grade I/IV TR (13.9%), 3 patients (8.3%) had over correction with minimal tricuspid stenosis noticed in two of them and moderate stenosis moderate degree of congested neck veins and liver just palpable, in one patient who improved by diuretics.

After six months, the follow-up of this group revealed no changes in tricuspid valve function by echo-Doppler.

Group B: One month follow-up of the patients in this group revealed that 24 patients out of 30 (80%) had competent tricuspid valve. Six patients (20%) had grade I/IV TR, 2 patients (6.7%) were overcorrected with minimal tricuspid stenosis, as diagnosed accidentally by echo-Doppler during routine check up without clinical manifestations.

Six months later, follow-up of the patients in this group revealed that two patients out of 24 who previously had competent valves one month after operation developed TR. One had significant degree of TR (grade III/IV). He was controlled by medical treatment and is being regularly followed-up. The other patients was a 34 years old male who had mitral valve replacement with pulmonary artery pressure of 80 mmHg pre-operatively. One month after operation, he had a competent tricuspid valve. Four months post-operatively, he suddenly deterriorated and showed all symptoms and signs of right sided decompensation. He did not respond to medical treatment. By echo-Doppler examination, he was found to have grade IV/IV TR. This patient needed an urgent operation and tricuspid valve repair by segmental annuloplasty was done. His general as well as his cardiac condition are good at the present time.

As regard the other 6 patients with minimal TR one month after operation, their pulmonary artery pressure ranged from 60 to 80 mmHg (mean 70.2 mmHg ± 4.6). Three of them still have grade I/IVTR. Two patients developed grade II/IV TR. They responded well to diuretics. One patient developed grade III/IV TR with good hemodynamics on medical treatment and he is submitted to regular follow-up.

Morbidity

Over all morbidity occured in 14 patients (21.2%). Early post-operative cardiac decompensation that needed hospitalization for more than 2 weeks occurred in 10 patients (15.1%) but improved well before discharge just by medical treatment. This was probably related to a myopcardial factor. Three patients out of six who had aortic as well as mitral valve replacement were comprised in this group of patients.

Delayed recovery occurred in one patient (1.5%). He improved well after 4 days. Two

Comparative results of group A and group B

	Segmental annuloplasty		De-Vaga annuloplasty	
	No	%	No	%
Total number of patients	36		30	
One-menth post-operative competent valves	31	86.1	24	80
One-monthpost-operative TR	5	13.9	6	20
Six-months post-operative competent valves	31	86.1	24	80
Six-months post-operative TR.	5	13.9	8	26.6
Overcorrection with tricuspid stenosis	3	8.3	2	6.7

patients (3%) had transient heart block, both of them developed outside the OR. In one of them, temporary pacing was needed for 48 hours. The other patient developed intraoprative block due to hesitation of stitching with a puncture made at the site of the bundle (both patients had the annuloplasty done with the heart beating).

Acute renal failure occured in one patient (1.5%) but he improved after peritoneal dialysis.

Discussion

De-Vega in 1980, reported on 500 patients with this technique over a period of 10 years with gratifying results[6].

Duran and colleages in 1980 found that approximately 30% of patients had small or moderate post-operative tricuspid systolic gradients after repair. Most residual gradients of incompetence are not clinically important if hemodynamic correction of the left sided heart disease is adequate[7].

Grodin et al (1975) reported 17 patients operated upon with De-Vega annuloplasty with good results and incidence of residual incompetence of a slight degree in about 16% and moderate degree in 6.5%[8].

In our study, there was 10% slight degree of TR, 6.6% moderate of TR, 10% severe degree of TR 6 months after the operation.

De-Vega in 1975, reported the first group of 155 patients, only one of them showed partial detachment of the suture from the annulus[9]. We had only one case out of thirty patients.

Grath and his colleagues (1990), said that "Management of functional tricuspid incompetence is controversial for results reported by groups recommending non-operative treatment of functional TR, have been similar to those reported in patients having various annuloplasty techniques of tricuspid valve replacement. Late death occurs in 40% of the patients who survived the original procedure for tricuspid repair. The majority of these patients died of cardiac failure, which indicates

progressive cardiomyopathy despite apparently successful surgical intervention[10].

In our series, the mortality is excluded because the cause of death was usually not related to the tricuspid valve.

Rackley and his coleagues found that, combining tricuspid valve surgery with surgery for other cardiac valves significantly increased the operative risk. The risk of annuloplasty in combination with aortic and mitral surgery is about 15%[11].

Our experience with aortic and mitral valve replacement with tricuspid annuloplasty is as gloomy as that reported by Rackley as 3 out of 6 patients with triple valve lesion in our series had early post-operative cardiac decompensation.

On the other hand, the number of morbid cases in our series is much higher than reported by others. It reached up to 21%. This may be due to more advanced disease in our patients with neglected rheumatic heart disease who develop severe degree of pulmonary hypertension and left ventricular dysfuction. Elevated mean pulmonary artery pressure, poor left ventricular function and severity of mitral valve lesion have all been indentified as very important predictors of survival after tricuspid valve surgery[12,13,14].

Revulta and Garcia-Rinaldi published that, so far, they have used segmental annuloplasty, this quick, easily performed technique in 35 patients with satisfactory results. Intraoperative Doppler echocardiographic examination demonstrated complete correction of the original tricuspid insufficincy. In 6 months, none of those patients had a recurrent tricuspid insufficiency[15]. This results is comparable to our results in segmental annuloplasty.

Conclusion

1. Segmental annuloplasty is as fast and technically simple as De-Vega technique. It takes about 15 minutes and provides a pliable valve.

2. It eliminates the need for inserting foreign material as in De-Vega technique.

3. Eliminates the risk of atrioventricular block especially when it is done on a beating heart.

4. The group of patients with segmental annuloplasty achieve better results than those with De-Vega procedure.

5. Segmental annuloplasty successfully achieves annular reduction while compensating for suture tearing. Even if one suture should fail or tear from the endocardium, enough sutures would remain to prevent massive tricuspid insufficiency.

Therefore, we conclude that STA is the procedure of choice for the correction of significant TR, as it gives better results in terms of alleviating the incompetence and in terms of durability after surgery. DTA, although shows good immediate post-operative results, does not maintain these results for long after surgery as noticed by follow up.

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Normothermic retrograde continuous blood cardioplegia "Hemodynamic Study"

Abstract

Normothermic retrograde continuous blood cardioplegia (NRCBC) is a new method of myocardial protection that has been reported to yield good clinical results. In this study, 20 patients were randomly selected. These patients had single or multivalvular lesions. They were scheduled electively to undergo open heart surgery. A complete clinical work-up was done preoperatively for every patient. At operation, myocardial protection was afforded by the technique of NRCBC . Induction of heart arrest was achieved antegradely in every patient, unless there is aortic regurgitation, where retrograde induction was needed. Maintenance of arrest was done by retrograde continuous perfusion of hyperkalemic blood. Details of the technique will be described in the text.

Certain parameters chosen to be observed during the perioperative perioperative period. State of the heart during aortic crossclamping and immediately after declamping, pattern of weaning of the heart from the bypass machine, changes in hemodynamic measurements during the immediate post-operative period and the detection of perioperative myocardial infarction are these parameters.

Results showed that the heart was arrested and perfused in 90% of the cases while in 10% repeated potassium shots were needed to maintain the heart arrested. Spontaneous rebeating immediately after aortic declamping was achieved in 85% of the cases. Weaning from the by-pass machine was easy in 95% of the cases. Five patients (25%) needed 5 ug/kg/min. of Dobutamine in the immediate post-operative period. Two patients (10%) needed small doses of adrenaline to counteract the vasodilator effect of this technique. Only one patient (5%) showed positive electrocardiographic and enzymatic changes of perioperative myocardial infarction with no hemodynamic disturbances. We conclude that this technique is a good alternative to the cold crystalloid cardioplegia in valvular heart surgery.

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Introduction

Normothermic Retrograde Continuous Blood cardio plegia (NRCBS) has become the hottest topic in myocardial protection since high potassium solutions were popularized in the mid 1970s[1-2]. A recent survey estimated that 10% of practicing cardiac surgeons are now using warm techniques[3]. The potential advantages of this technique are

Table 1: Patient profiles

No.	20	
Age range	13-42 years	
Sex	Male 8	
	Female 12	
Functional class	III 19	
	IV 1	
Type of valve lesion	MVD	3
	MVD + TVD	6
	AVD	4
	MVD + AVD	3
	MVD + AVD + TVD	4
LVF	Normal 8	
	Dilated 8	
	Concentric hypertrophy 3	
	Generalized hypokinesia 1	
RVF	Normal	15
	Altered	5
PA systolic pressure	> 100	6
	60-100	3
	<60	11

most appealing. Hypothermia leads to a gross imbalance in the metabolic processes of myocardial homeostasis. On the other hand, the normothermic arrested heart is metabolically balancled and provides an excellent milieu for myocardial resuscitation and replenishment during the cross clamp interval. NRCBC can therefore be considered an interval of myocardial resuscitation; The heart is aerobic, perfused and rested[4]. Clinical studies observed that NRCBC is particularly effective in high-risk subgroups undergoing valve operations or revascularization procedures[5].

The aim of this study was to use this technique to benefit from its superior myocardial protection especially in high-risk patients. We also aimed at explaining the technqiue and to try to convince other surgeons to try it.

Patients Population

The patient population consisted of 20 patients having valvular heart disease. They were scheduled electively to undergo open heart surgery to treat their valvular lesions. These patients were 12 females and 8 males. Their age range was 13 to 42 years. Nineteen of them were in functional class III (Dyspnea on mild exertion) and one was in functional class IV (dyspnea at rest). These patients were studied preoperatively by a complete clinical work-up including electrocardiography, chest roentogenography and Doppler echo-cardio-

graphy. The type of valve lesion, left and right ventricular functions and pulmonary artery pressure as well as other data are shown in Table 1.

Operative technique

Two central and 2 peripheral lines were put for every patient. The blood pressure was monitored by a cannula in the radial artery. Anaesthesia was induced with Fentanyl and Pavulon. Priming of the oxygenator and tubes was done using crystalloid solutions. Patients were placed on by-pass by an arterial cannula placed in the ascending aorta and 2 venous cannulas into both cavae. Snares were put. A separate pump was left for cardioplegia delivery. This pump was connected to the cardioplegia outlet of the oxygenator by a tube which was brought up to the table bringing blood having the same temperature as that of the oxygenator. By a side way another line attached to an electric syringe was connected. So at the end, the tube brought up to the table gives blood at normothermia and into this blood we inject our cardioplegia, which consists of potassium and magnesium, in a continuous way by an electric syringe.

Cardioplegia delivery

I. Induction of heart arrest

1. Antegrade: Indicated only if the aortic valve is competent. The cardioplegia line brought up to the table is conncted to Y shaped connector. One side is connected to the cardioplegia cannula inserted into the aortic root and the other end to the coronary sinus cannula which is not yet inserted into the cornary sinus. Proper deairing of the system should be done. After aortic clamping, antegrade injection of cardioplegia started. The flow of blood pumped into the aortoc root should reach 300 ml/min, then, 15 MEq of potassium chlloride was injected slowly over 1 minute. This was usually enough to induce heart arrest. Then clamp this side of the Y connector.

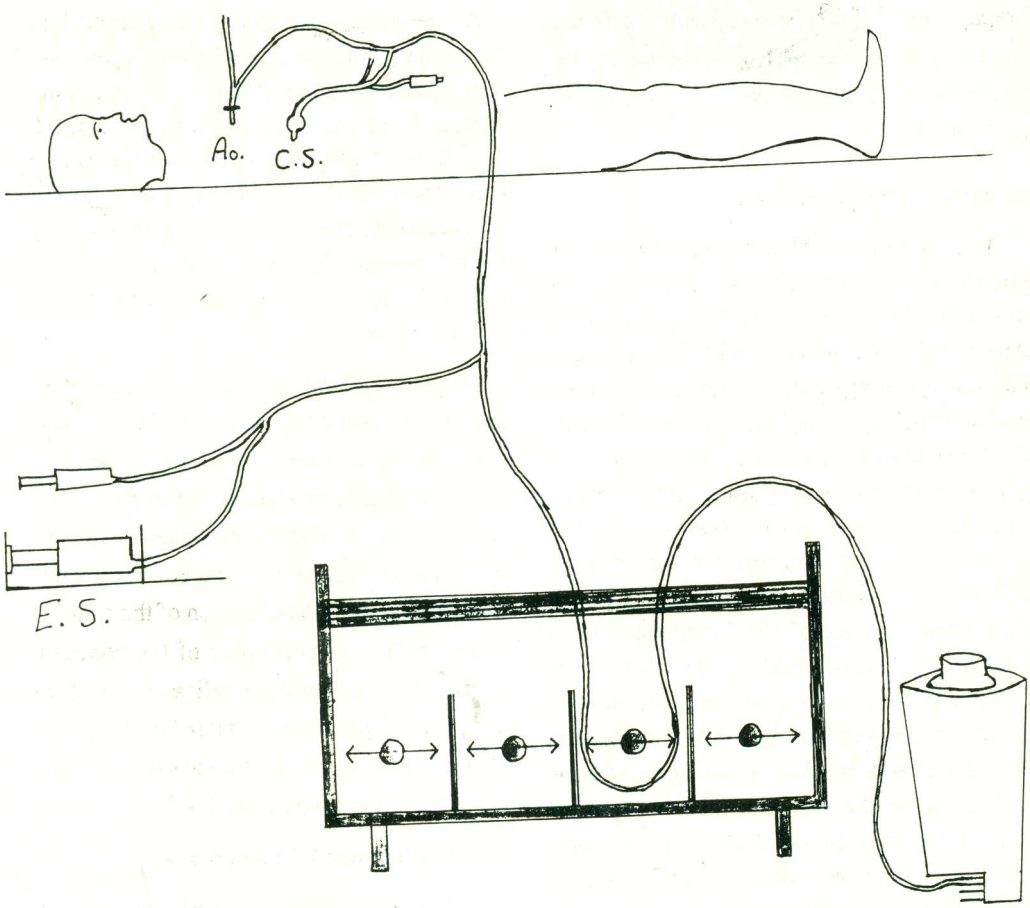
2- Regrograde: Indicated only in the presence of aortic regurgitation. The right atrium was opened and exploration of the right atrium was done to identify the orifice of the coronary sinus. Then aortic clamp was applied and introduction of the coronary sinus cannula was done. The balloon of the cannula should be just at the orifice of the coronary sinus. Then cardioplegia delivery started by a flow of 150 ml/min. After reaching this flow a bolus injection of potassium chloride (15 MEq) was given over 1 min.

II- Maintenance of heart arrest

Whether induction of cardioplegia was given by an antegrade or retrograde route, maintenance of heart arrest was the same. This consisted of retrograde continuous perfusion of hyperkalemic blood. The flow of blood pumped was kept at 150 ml/min. and into this flow of blood we injected our cardioplegia continuously by an electric syringe. This cardioplegia solution consisted of:

a. Potassium chloride 35 MEq/hour.

b. Magnesium chloride 24 MEq/hour.



Technique of delivery of cardioplgia

This was kept until the end of the procedure. Sometimes the heart started to move again so we were obliged to give a bolus of potassium (2-4 MEq) to abort these trials. After finishing the operation, the coronary sinus canula was removed. Deairing of the heart and aortic declamping was usually followed few seconds later by spontaneous rebeating of the heart.

Methods of study

The following parameters were chosen to be observed during the perioperative period:

1- State of the heart during aortic cross clamping and whether it was complerely arrested and perfused or needed repeated potassium injections.

2- Pattern of weaning of the heart from by-pass and changes in hemodynamic measurements during the first 48 hours.

3- Detection of perioperative myocardial infarction. This complication can be presented by a state of cardiogenic shock and difficulty to wean the heart out of the machine, but in the majority of cases it is a small injury that has no hemodynamic effects. Detection of this small injury is of utmost importance to know the efficacy of this technique. So, we followed these patients with serial ECGs and serum enzymes daily for 3 days to exclude the occurrence of perioperative infarction.

For the diagnosis of perioperative myocardial infarction by ECG we used the same criteria that Hultgren et al., 1977 had used. These criteria are the appearance of new persistent Q waves of 0.04 sec. duration or new QS deflections associated with the characteristic evolutionary changes in the ST segments and T waves[6].

Results

Results showed that the heart was kept et standstill all over the procedure in all cases except in 2 (10%) where repeated potassium unjeetions were needed to maintain the heart arrested. Spontaneous rebeating immediately after aortic declamping was obtained in all cases except in 3 (15%) where ventricular fibrillation occurred and DC shocks were indicated. Weaning from the extracorporeal circulation was easy in all cases except in one (5%) where weaning was impossible due to severe pulmonary hypertension and this patient died. Five patients (25%) needed 5 ug/kg/min. of Dobutamine in the immediate postoperative period. Two patients (10%) needed small doses of adrenaline to counteract the vasodilator effect of this technique. Only one patient (5%) showed positive electro-cardiographic and enzymatic changes of perioperative myocardial infarction with no hemodynamic disturbances.

Comment

Despite advances in myocardial preservation, time remains a problem for the cardiac surgeon. Prolonged procedures are associated with higher morbidity and mortality[7]. The advent of cold K+ cardioplegia and hypothermia increased the safe aortic occlusion time. During aortic occlusion, the myocardium becomes ischemic. In ischemic myocardium, anaerobic metabolism is responsible for energy production.

Although hypothermia has gained widespread acceptance, it has many side effects. The adverse effects involve membrane stability, calcium sequestration, adenosine triphosphate generation and utilization, tissue oxygen uptake, pH, cellular osmotic homeostasis and ATPase systems[8].

Electro-mechanical arrest of the heart lowers the energy demand of the myocardium by almost 80%. The concept of normothermic arrest is based on this approach, continuous application of normothermic blood cardioplegia has the advantage of supplying the energy needed during the arrest period. The results showed that myocardial performance in patiens receiving normothermic blood

cardioplegia was better than that in patients receiving cold blood cardioplegia[9].

Retrograde delivery of cardioplegic solution raises questions concerning adequate delivery of cardioplegia to the right ventricle. However, data showed that NRCBC is a safe and effective method of myocardial protection irrespective of pulmonary hypertension, right ventricular hypertrophy, associated left ventricular hypertrophy and prolonged cross clamp times[10-11].

Salerno and associates[12] have recommended infusion flows of 150 to 200 ml/min. with coronary sinus pressures of 30 to 40 mmHG, but higher pressures (as high as 70 mmHg) could be reached when lifting the heart for circumflex anastomosis. Pressures higher then 50 mmHg in the coronary sinus could be dangerous and should not be allowed.

Unexplained dificulties sometimes occur with warm blood cardioplegia. Occasionaly, arrested and continuos to fibrillate or to beat. A warm heart operation is a technically complex endeavor. It is more time consuming, exposure in not usually perfect due to blood flooding the operative field. If used in coronary bypass surgery, visualization of the distal coronary anastomosis is sometimes difficult. Many techniques were developed to solve this problem.

A final problem with normothermic cardiopulmonary byass is that it can lead to systemic vasodilatation that responds to more filling and sometimes vasopressor drugs[4].

Conclusion

In spite of the difficulties and the problems of NRCBC, myocardial protection is superior especially in high risk cases with long cross clamp times.

In our study, we had only one mortality (5%) which was not related to the technqiue or to the myocardial protection. Problems of difficult exposure and blood flooding could be easily dealt with by proper venting. In 10% of our cases we were confronted with some difficulties in maintaining the heart arrested.

We conclude that NRCBC is a valuable alternative especilly in high-risk cases. There is a learning curve to overcome the technical difficulties but still it deserves consideration.

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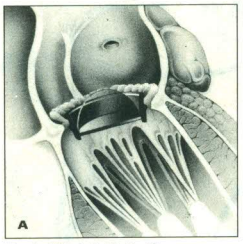

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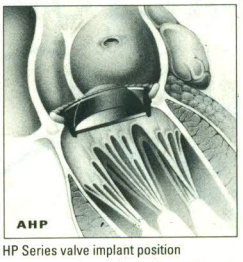

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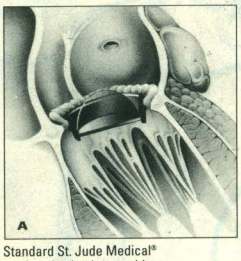

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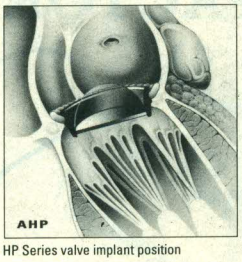

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