CLOSED MITRAL VALVOTOMY IN PREGNANCY

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CONTENTS PAGE

INTRODUCTION

Heart disease remains the most important non-obstetric cause of maternal mortality and morbidity during pregnancy, despite its low incidence of less than 1% (86, 87). This is due to the decline in the number of deaths from haemorrhage, infection and toxemia (36, 90). In addition, a striking change in the pattern of proportional distribution of organic heart disease in pregnant women is being noted, with a decrease in chronic rheumatic lesions and an increase in congenital cardiac disease (92).

In the Third World rheumatic mitral valve disease remains a most important condition during pregnancy. It is currently rarely seen in Europe and the United States. Mitral stenosis is the most commonly encountered rheumatic heart lesion that complicates pregnancy (14, 29, 35, 65, 67, 71, 78, 94, 95). The normal circulatory changes during pregnancy aggravate this lesion as the reduced, fixed valve area obstructs blood flow from the left atrium to the left ventricle, causing pulmonary congestion and oedema. (72, 94).

Careful and regular follow up of these patients is essential, and surgery is indicated if optimal medical management fails (5, 31, 35, 61, 86). Cardiac surgery during pregnancy represents a risk to both the foetus and the mother (31, 86, 105). For most procedures extracorporeal

circulation and heparinization are necessary and adds to the adverse effects of the operation.

Closed mitral valvotomy, however, is an excellent low risk operative procedure in patients with tight mitral stenosis without causing undue harm to the foetus (29, 105). Cuttler described the first attempted surgery of the mitral valve in 1923 and since then the procedure has been improved to benefit many patients with tight mitral stenosis (24). The first reports of closed mitral valvotomy during pregnancy were in 1952 (12, 22, 57, 60). Although a more precise valvotomy can be obtained with an open procedure, the closed operation avoids the risks of extracorporeal circulation, particularly detrimental to the foetus (8, 25, 32, 45, 52, 108).

This report is a review of the Groote Schuur Hospital experience of patients with mitral stenosis requiring closed mitral valvotomy during pregnancy since 1965. The aims of the study are to analyse the outcome of the pregnancy, the effects of valvotomy during pregnancy on both the mother and the foetus, and the outcome regarding restenosis of the mitral valve.

PATIENTS

Since January 1965, 43 closed mitral valvotomies were done using the Tubb's dilator for mitral stenosis complicating pregnancy in 41 patients. Two of these patients required repeat closed valvotomies in subsequent pregnancies. The average age of the patients was 27 years with a range from 18 to 41 years. A history of acute rheumatic fever in childhood was obtained from 15 patients (32.5%).

Thirteen patients presented with acute pulmonary oedema plus haemoptysis, 17 had pulmonary oedema only, 4 had haemoptysis only, and the remaining 9 patients had progressive shortness of breath and deteriorated despite maximal medical treatment. The condition of the patients was graded preoperatively according to the classification of the New York Heart Association (23). Six patients were Class II, 25 Class III, and 12 Class IV, limited, with a mean NYHA Grade of 3.01.

Preoperative electrocardiogram revealed sinus rhythm in 41 patients (Figure 1) and atrial fibrillation in 2. The effects of mitral valve disease, that is, left atrial enlargement and upper lobe venous diversion, were visible on chest roentgenograms in all cases (Figure 2). Evidence of

right ventricular hypertrophy was present in 10 and the classical picture of acute pulmonary oedema in 30 patients.

Thirteen patients required closed mitral valvotomy during the first pregnancy. Of those remaining nine were gravida 2, four gravida 3, five gravida 4, four gravida 5, two gravida 6, four gravida 7, and two gravida 9 (Figure 3). The operations were performed in the first trimester in 2 patients, in the second trimester in 23 and in the third trimester in 18 (Figure 4).

The operative approach was via a left anterolateral thoracotomy through the fifth intercostal space in 41 instances, but a median sternotomy for the closed mitral valvotomy was done in 2 cases where the left atrium was approached from the right side as for mitral valve replacement. The rationale for this was the desire to proceed with an open procedure should the result not be satisfactory in these suboptimal valves, and thereby possibly sacrificing the foetus. The valve orifice was assessed digitally at operation by a finger inserted into the left atrium through its appendage, and the valve was split with a Tubb´s dilator inserted via the left ventricular apex across the orifice of the stenosed mitral valve (Figure 5). Satisfactory dilatation of 32 to 42 mm was achieved in all patients except 2, in whom only the anteromedial commissure could be split (Figure 6).

RESULTS

Forty-three closed mitral valvotomies were done in 41 pregnant women. There were no maternal deaths related to the operation and subsequent delivery and all patients experienced symptomatic improvement from NYHA Grade 3.01 average preoperatively to Grade 1.22 after surgery. Postoperatively, 33 patients were in Class I and 10 in Class II (Figure 7).

Spontaneous abortion occured in 2 cases after midtrimester operations, and 3 women were delivered prematurely of stillborn infants after valvotomies done during the third trimester resulting in a foetal mortality of 11.4% (Figure 8). Foetal morbidity was due to dysmaturity and prematurity. Two babies were dysmature at term, born to mothers who had valvotomies during the first and third trimesters, one infant was premature, born at 36 weeks' gestation 6 weeks after a third trimester valvotomy. Twins were born prematurely to one mother 12 weeks after a mid-trimester valvotomy.

Thirty four normal infants were delivered at term, for an overall survival of 39 babies to 38 mothers (88.6%) (Figure 9). Of these, 3 were delivered by caesarean section, 3 deliveries were assisted by forceps and 1 by vacuum extraction.

In summary, the outcome of pregnancy related to the trimester at the time of operation was as follows:

* Only 2 mitral valvotomies were performed during the first trimester. In both instances the pregancies reached term and resulted in live births, one infant was normal for gestational age and another was dysmature weighing 2100 grams.
* During the second trimester 23 operations were done. Two spontaneous abortions at 17 and 24 weeks gestation followed. Twins were born prematurely at 34 weeks gestation. Both are alive, with birth weights of 2500 and 1800 grams respectively. The other 20 patients were delivered at term of normal infants.
* Eighteen third-trimester valvotomies took place. Three mothers were delivered of stillborn premature babies. One baby was born alive at 36 weeks gestation and weighed 2000 grams. Another was dysmature at term and weighed 1500 grams. Thirteen normal infants were delivered at term (Figure 10).

Ten patients needed 12 subsequent surgical procedures (27.9%) for mitral valve restenosis from 5 to 17 years (mean 10.0 years) after the initial procedure. Repeat closed mitral valvotomy was performed in 4 patients after 5, 7, 8

and 10 years. In two of these, the valvotomy was done during a subsequent pregnancy. Both these patients have since had mitral valve replacements, one 11 years, the other 15 years after the second closed mitral valvotomy.

An open mitral valvotomy was done in one patient after 6 years. Seven patients needed mitral valve replacement; one after 7, two after 11, two after 12, one each after 15 and 17 years. In the rest of the patients (72.1%) further surgical procedures have not been necessary (Figure 11).

There were 2 late deaths. Both patients had significant mitral valve restenosis. One died of pneumonia and meningitis after 10 years, and the other died after 12 years, before mitral valve replacement could be done (Figure 12). Two patients are moderately symptomatic but do not need surgical intervention.

DISCUSSION

1 CIRCULATORY CHANGES IN NORMAL PREGNANCY

The normal cardiovascular response to pregnancy is gradual, but disproportionate as it preceeds and exceeds the demands of the foetus. This may be either in anticipation of the needs of the growing foetus or an adaptive response to the metabolic and nutritional demands of the conceptus (31, 76, 78). Blood volume, stroke volume, heart rate and therefore cardiac output increase, while a decrease is noted in systemic and pulmonary vascular resistance, with resultant decrease in arterial pressure (36, 91). The normal circulatory changes in pregnancy result in a clinical picture which may either simulate or obscure the presence of heart disease (27, 28).

1.1 BLOOD VOLUME

The blood volume increases by 40%, constituted by a rise of red cell volume of about 20% and of plasma of the order of 50%. As a result the haematocrit drops from about 40% to 33% causing the so-called "physiological" anaemia of pregnancy (68, 78, 86). This is not entirely prevented by treatment with iron supplements, but is reduced in severity (16).

There is no accurate correlation between maternal size and the magnitude of increase in plasma volume. A correlation, however, is noted with the weight of the infant in primigravidae (39), with the weight of the placenta (83), and with the combined weight of the baby and the placenta (102).

Mechanisms postulated to be responsible for the increased blood volume include

(i) increased activity of plasma renin and levels of aldosterone despite the rise in glomerular filtration rate (106),

(ii) uterine renin secretion (30, 34),

(iii increased liver production of renin due to oestrogenic substances (98),

(iv) increased renin activity and aldosterone levels due to both oestrogens and progesterone (55),

(v) the arteriovenous leak through the placenta (13, 15) and

(vi) direct action of progesterone in relaxing veins and increasing the capacity of the vascular system (46).

1.2 HEART RATE

There is a slight increase in maternal heart rate early in pregnancy and this continues to rise as the pregnancy

advances to a maximum rate of 80 - 90 beats per minute at term. The mean increase measured is 10 - 20% relative to prepregnancy values (50, 73, 82, 85).

1.3 CARDIAC OUTPUT

Due to the rise in stroke volume and heart rate, an increase in cardiac output is clearly evident by the end of the first trimester and reaches its peak by the 20th to 24th week of gestation, when it exceeds normal values in resting non-pregnant patients by 30 - 50% (6, 50). This level is maintained throughout the pregnancy. It was previously thought that cardiac output returned to normal towards term, but when the position of the patient (supine or lateral) was taken into account this was not substantiated. (1, 10, 28, 36, 47, 78, 85, 103, 104).

The weight of the gravid uterus has simple mechanical effects depending on the position of the patient. Complete caval obstruction occurs in the supine position late in pregnancy with a resultant fall in cardiac output. Venous drainage then follows the azygos, lumbar and paraspinal veins (47, 50). The rise in cardiac output following change from the supine to the lateral position has been confirmed by echocardiographic studies in late pregnancy (85). The supine hypotensive syndrome of pregnancy - the experience of lightheadedness or syncope in the supine position with

bradycardia and a decrease in blood pressure - is rapidly reversed by changing into the lateral position (47, 50, 63, 78).

1.4 SYSTEMIC VASCULAR RESISTANCE

In early pregnancy a fall in systemic arterial blood pressure is noted, with initially a slightly greater reduction of diastolic pressure. The increase in cardiac output coincides with the reduction of peripheral vascular resistance. (50, 78).

1.5 PULMONARY VASCULAR PRESSURES

The pulmonary vascular resistance falls. The increase in pulmonary artery mean pressure, diastolic pressure and right ventricular end-diastolic pressure in the midtrimester may be secondary to intrapleural pressure changes and the effects of hypervolaemia (4).

2 CLINICAL CARDIOVASCULAR SYMPTOMS AND SIGNS DURING PREGNANCY

The circulatory changes during pregnancy increase the workload of the heart, which may then fail if diseased (29, 31, 36, 86, 99). During normal uncomplicated pregnancy symptoms, physical signs, and, electrocardiographic, roentgenographic and echocardiographic changes develop which simulate heart disease (27, 28, 37, 78, 105).

Symptoms include easy fatigueability, dyspnoea, hyperventilation, decreased effort tolerance, palpitations, lightheadedness and even syncope which may also occur in normal pregnancies (47, 69, 78). Peripheral oedema occurs in 50 - 80% of all pregnant women (62, 69, 80, 92). One finds clinically signs of hypervolaemia, like basal pulmonary rales, prominent jugular venous a and v peaks with brisk x and y descents and a more forceful upward and leftwardly displaced left ventricular impulse (37, 69, 78). The first heart sound is increased in intensity and the second tends to exhibit expiratory persistent splitting. A third heart sound is occasionally heard and is due to rapid ventricular filling.

Functional (so-called innocent) murmurs are commonly audible in healthy pregnant women. A pulmonic midsystolic murmur with maximal intensity in the second left intercostal space

is caused by right ventricular ejection into the pulmonary vascular bed. Supraclavicular systolic murmurs originate in the brachiocephalic arteries where they branch off the aortic arch (33, 37, 50, 61, 78, 88). The mammary souffle, finally, is an innocent murmur peculiar to pregnancy occuring either in systole or as a continuous murmur over the breast in late pregnancy and during lactation (96).

Electrocardiographic changes consist, besides the already described increase in heart rate, of a shortening in PR- and QT- interval. The QRS axis shifts leftward, averaging 15 - 40°. Ectopic beats occur commonly in pregnancy, as do bouts of paroxysmal supraventricular tachycardia (17, 78, 86).

Roentgenographic changes include elevation of the diaphragm which causes upward and lateral displacement of the cardiac apex resulting in a relatively horizontal cardiac silhouette on the chest radiograph. The hyperlordosis of pregnancy causes a straightening of the left heart border as well as radiographic prominence of the main pulmonary artery (78).

Echocardiography, a relatively new non-invasive investigation, reveals increased left ventricular end-diastolic volumes and internal ventricular dimensions, which occur normally with pregnancy. Mild volume overload and hyperkinesia of the ventricular walls are also noted towards the end of gestation (43, 50, 85).

2.1 CLINICAL SYMPTOMS AND SIGNS OF HEART DISEASE IN PREGNANCY

Some symptoms and physical signs, however, indicate heart disease in pregnancy. The symptoms of severe progressive dyspnoea, progressive orthopnoea, paroxysmal nocturnal dyspnoea, heamoptysis, syncope with exertion or chest pain related to effort or emotion are not normal and necessitate further investigation.

Findings on clinical examination of cyanosis, clubbing, persistent jugular distention, systolic murmurs of more than grade III/VI intensity, diastolic murmurs, cardiomegaly, sustained arrythmia, persistent split second sound or signs of pulmonary hypertension (loud pulmonary component of the second sound or left parasternal lift) all indicate a pathological condition (61, 69).

3. MITRAL STENOSIS IN PREGNANCY

In women of child bearing age heart disease is usually of rheumatic origin constituting 75% to 90% of structural cardiac complications during pregnancy (78). The proportion of chronic rheumatic heart disease as a cause of heart disease in pregnant women in the Western World decreased from 90% earlier this century to less than 75% now.

As a result of the decline in the overall incidence of rheumatic fever, a decrease in the prevalence of rheumatic heart disease in women of childbearing age was noted in the United States and Western Europe. Szekely reported a 3.5% incidence of rheumatic heart disease in 5617 pregnant patients studied at the Newcastle General Hospital between 1942 and 1947 in contrast to 0.7% of 35426 pregnancies between 1966 and 1969. Mitral stenosis was the predominant lesion (90%), with mitral regurgitation in 6.6%, aortic regurgitation in 2.5% and aortic stenosis in 1% of the remaining patients (71, 95).

In similar reviews of rheumatic heart disease in pregnant women mitral stenosis was present in 65 - 75%, mitral incompetence in 10%, combined mitral and aortic lesions in 10 - 15%, and aortic stenosis and other valvular lesions in a small percentage (14, 65, 67). Mitral stenosis is also

the most common result of acute rheumatic fever with a female to male ratio of 4:1 (19, 29, 36, 78, 94).

In Southern Africa rheumatic valvular disease still constitutes the majority of cardiac defects with mitral stenosis being the predominant valvular lesion complicating pregnancy (87). In 1985 a study performed at King Edward VIII Hospital, Durban, South Africa showed that the incidence of cardiac disease among pregnant patients is 0.4%. Mitral stenosis was diagnosed as an isolated lesion in 56% and the dominant lesion in 76% of patients with combined valvular lesions during pregnancy (76).

3.1 PATHOPHYSIOLOGY OF MITRAL STENOSIS

Mitral stenosis obstructs the flow of blood from the left atrium to the left ventricle during diastole. This is caused by commisural fusion and fibrotic thickening of the leaflets and subvalvar apparatus. The physiological alterations in maternal haemodynamics in pregnancy described before (increased blood volume, heart rate and cardiac output) aggravate this defect by augmenting the left atrioventricular pressure gradient and increasing left atrial, pulmonary venous and capillary pressures. When the latter exceeds approximately 25 mmHg, transudation of liquid into the alveoli may result in pulmonary oedema (67, 72, 76, 78).

Although symptoms may be initially mild in some patients, the risk may be higher later on, emphasizing the unpredictabilty of pregnancy's effects on the circulation (91, 95). The tendency to development of pulmonary oedema even in patients with non-critical mitral stenosis is explained by the increased blood volume, heart rate and cardiac output, while blood pressure in both the systemic and pulmonary circuits remain unchanged, or drops slightly (76).

Left atrial pressure elevation causes dilatation of the chamber, which results in arrhythmias, particularly atrial fibrillation. A dilated left atrium, plus atrial fibrillation, increases the risk of intramural thrombosis and systemic thrombo-embolism (72).

The sudden onset of atrial fibrillation increases the haemodynamic burden by the loss of left atrial contraction; an accelerated heart rate, reduced diastolic duration and sudden increased mitral valve flow and transmitral gradient (76, 78). This often precipitates pulmonary oedema, which is a most alarming and dangerous complication of mitral stenosis in pregnancy.

Acute pulmonary oedema is the most important cardiac cause of maternal death (94). Some patients are less prone to

pulmonary oedema due to a mechanism called "reactive pulmonary hypertension"; pulmonary arteriolar vasoconstriction as a result of increased pressures in the left atrium and pulmonary veins avoids or decreases the severity of the pulmonary capillary hypertension. In addition, some patients with longstanding mitral valve stenosis have developed irreversible obliterative changes in the pulmonary vasculature, with subsequent right ventricular hypertrophy and even right ventricular failure.

Haemoptysis due to rupture of anastomoses between bronchial and pulmonary veins, also occurs and although it is a frightening event, it is seldom life-threatening (72).

3.2 CLINICAL PRESENTATION OF MITRAL STENOSIS

Mitral stenosis during pregnancy is recognised by specific findings, including dyspnoea, orthopnoea, oedema, haemoptysis, easy fatigueabilty or a systemic embolic event. On examination the classical signs of cardiac failure may be present and on auscultation, a loud first sound, an opening snap of the mitral valve, a diastolic rumble with presystolic accentuation at the apex and a loud pulmonary component of the second sound are characteristic of mobile mitral stenosis and pulmonary hypertension (76).

Radiology of the chest may reveal left atrial enlargement,(manifesting as a straight left heart border only or a double density shadow on the postero-anterior film), upper lobe venous diversion, Kerley's B lines, right ventricular hypertrophy and sometimes calcification of the mitral valve. An electrocardiogram may show left atrial enlargement (P mitrale - broad notched P wave in lead II), atrial fibrillation and right ventricular hypertrophy (76).

The echocardiogram is a valuable non-invasive way of investigating the pregnant patient with mitral stenosis. It may demonstrate left atrial enlargement and shows the characteristics of the mitral valve itself. Normally the posterior mitral valve leaflet moves away from the anterior leaflet during diastole. Fusion of the commissures, as in rheumatic mitral stenosis, causes anterior movement of the posterior leaflet with the anterior leaflet and a flattened EF slope (72, 76).

4. MEDICAL MANAGEMENT OF THE PREGNANT WOMAN WITH MITRAL STENOSIS.

The increasing safety of pregnancy in women with cardiac disease is due to the diminishing incidence and severity of mitral stenosis, improved understanding of the lesion and better medical and surgical treatment (72, 100).

The majority of pregnancies complicated by cardiac disease are uneventful, with a good outcome for the mother and foetus. There is agreement that a good maternal cardiac status has a beneficial effect on both maternal and foetal prognosis (44, 101). It is seldom necessary to terminate the pregnancy or to perform surgery for mitral stenosis during pregnancy (93).

4.1 GENERAL GUIDELINES

If a woman with significant mitral stenosis presents in pregnancy, symptoms should be controlled medically. If such measures prove unsuccessful, surgical intervention should be considered during pregnancy (5, 31, 35, 61, 105). Patients should be managed by a combined cardiac and obstetric team, both familiar with the special risks related to a cardiac defect in pregnancy (31, 86).

Good health habits should be established with regular exercise, rest and good nutrition. Ingestion of alcohol, cigarette smoking and treatment with drugs, that may be teratogenic should be avoided. Excess physical activity, emotional stress and anxiety, hot humid environments and large meals are best avoided as they add an unnecessary burden to the heart (1, 61, 76).

In general, it is wise to defer chest roentgenography, cardiac catheterization and angiography until after completion of the pregnancy to avoid the risks of radiation (78,91). Both rheumatic fever and infective endocarditis prophylaxis are indicated in the management of the young pregnant patient with mitral stenosis (42, 50, 89). Coexisting noncardiac diseases may exert undesirable effects on the circulation by increasing the workload of the heart. These should be recognised and treated immediately and effectively, for example: anaemia, hypertension, arrhythmias, infections (upper resiratory and urinary tracts), obesity and hyperthyroidism (1, 61, 78).

4.2 TREATMENT OF CARDIAC FAILURE

The principles of management of cardiac failure are designed to control the amount and distribution of extracellular fluid, limit the demand for cardiac output, improve cardiac

contractility, treat the underlying and complicating conditions and treat the cardiac abnormality (70).

Treatment of cardiac failure is therefore extremely important in avoiding pulmonary oedema (61, 78, 93). All patients should regularly be examined with particular attention given to evidence of fluid retention (1). Daily recording of weight, limited salt intake and the use of diuretics and potassium supplements adjusted to the individual patient´s needs are necessary.

Digitalis preparations are still recommended to treat cardiac failure, although there is controversy whether the inotropic action thereof is of sufficient significance to use the drug in view of its side-effects. Digitalis has the important effect of reducing heart rate by decreasing atrioventricular conduction in the presence of supraventricular tachycardia or atrial fibrillation. Otherwise it reduces the heart rate by a vagal effect on the sinus node. Digoxin is passively transferred across the placenta, but is nonteratogenic and therefore can be given in dosages similar to those in non- pregnant patients (81, 97).

Preload reducing agents, like nitrates, also produce good results by their vasodilatory effect pooling blood peripherally and decreasing systemic venous return (70).

Vasodilator agents which decrease cardiac afterload are reserved for cases where additional complicating factors, like mitral incompetence or poor left ventricular function are present. Vasodilators, however may produce dangerous hypotension in patients with fixed obstructive lesions like mitral stenosis and should be used with the utmost care.

Changes in preload (physiological increase in blood volume) during pregnancy and labour are readily accommodated in the normal heart by increased cardiac output. In mitral stenosis with a fixed mitral valve orifice area and fixed cardiac output fluctuations in preload lead to increased pulmonary capillary wedge pressure and hydrostatic pulmonary edema. Therefore heart rate control (reduction) is important as it prolongs diastolic filling time of the left ventricle (20).

During pregnancy, treatment with a beta-blocking agent (like atenolol 50 - 100 mg daily) inhibits the heart's rate response to exercise and anxiety. This results in a longer diastolic period for transmitral flow (76). By decreasing cardiac output as well, beta-blockers reduce the transmitral pressure gradient and pulmonary wedge pressure in patients with mitral stenosis in sinus rhythm without increasing the left ventricular end-diastolic pressure (50,64). A heart rate of 60 beats per minute is optimal (76)

The use of beta-blockers appears to be safe in pregnancy, although concern remains regarding the possibility of the drug masking the signs of foetal distress (50, 58, 75, 79, 84). Cautious diuresis to control preload and propranolol to optimize heart rate during labor with pulmonary capillary wedge pressure monitoring give excellent results for both mother and infant in cases with severe mitral stenosis (20). Some controversy still exists regarding the safety of beta-blockers during pregnancy. It is thus advisable to observe these patients very meticulously during their pregnancy and labor (97).

4.3 TREATMENT OF ACUTE PULMONARY OEDEMA

Acute pulmonary edema is urgently treated by placing the patient in a sitting or semirecumbent position and thereby diminishing venous return, as well as immediate administration of oxygen. Intravenous medications include Morphine sulphate to relieve anxiety and to act as a vasodilator to pool blood peripherally and thereby reduce the venous return to right atrium. Preload reduction is also achieved by Furosemide, a powerful diuretic with an initial vasodilator effect and Nitrates given either by titration intravenously or sublingually. The control of supraventricular arrhythmias (atrial tachycardia, flutter or fibrillation) which often precipitate acute heart failure and pulmonary oedema is very important and here

administration of intravenous Verapamil is extremely useful. Digoxin also helps to control supraventricular arrythmias and, as an inotrope, is of some value improving cardiac contractility as discussed before. Additional inotropic support with Dopamine and Dobutamine should be used where individually indicated (70, 95). In patients not improving on this therapy, endotracheal intubation and mechanical ventilation with positive endexpiratory pressure is indicated to maintain adequate oxygenation and respiration.

4.4 THE PLACE OF TERMINATION OF PREGNANCY

In those patients, where termination of pregnancy could be a consideration, it is far safer to prevent pregnancy using contraceptive measures.

Termination of pregnancy is reserved for women in whom pregnancy is life-threatening, such as patients with severe pulmonary hypertension, suprasystemic pulmonary vascular resistance, persistent cardiomegaly following peripartum cardiomyopathy, and some patients with Marfan´s syndrome.

If necessary, it should preferably be done in the first trimester, but unfortunately few patients have significant symptoms early in pregnancy. It has the additional disadvantages of sacrificing the foetus, the morbidity and

mortality of the procedure, as well as, today, an admission of defeat rather than offering a form of therapy (36,67).

5. THE PLACE OF CLOSED MITRAL VALVOTOMY IN PREGNANT PATIENTS WITH MITRAL STENOSIS

The first reports of closed mitral valvotomy during pregnancy were in 1952 by Brock, Cooley and Chapman, Logan and Turner, and Mason (12, 22, 57, 60). The operation has gained acceptance since, and numerous patients have undergone the procedure during pregnancy with successful outcome and low operative risk (31, 48, 74). In pregnancy, it must not be forgotten, that both the mother and the foetus are at risk and both should be considered in all decisions regarding management (95).

In 1963 Szekely appropriately stated the three opinions regarding the place of surgery in the management of the pregnant patient with mitral stenosis. Firstly, surgery as a lifesaving procedure is only indicated during pregnancy in the exceptional patient, who is refractory to medical treatment. Secondly, there is no difference in the indications for surgery whether or not the patient is pregnant, and, thirdly, pregnancy should be regarded as an additional indication for surgical intervention (94).

The first mentioned point of view is the commonly practised policy in most units as most physicians and surgeons feel

that operative procedures are preferably avoided during pregnancy (5, 35, 61, 105).

5.1 INDICATIONS FOR CLOSED MITRAL VALVOTOMY IN PREGNANCY

The absolute indications for mitral valvotomy during pregnancy are uncontrollable pulmonary oedema, or, uncommonly, uncontrollable or recurring haemoptysis (11, 48, 61, 70, 86,94). In patients with refractory pulmonary oedema, emergency valvotomy can be lifesaving (35).

We have accepted patients for surgery when an increased susceptibility to pulmonary oedema could be firmly established on reliable history or clinical observation. All patients were initially treated medically, as discussed earlier in this manuscript.

When patients fail to improve on optimal conservative treatment, continuing the medical management outweighs the risk of the operation and these patients should be offered surgery (95).

The excellent maternal outcome in our group of patients, who all survived the operation and pregnancy, supports this view. All patients experienced symptomatic improvement from an average NYHA Grade 3.01 pre-operatively to 1.22 after surgery. Valvotomy can be done at any stage of the

pregnancy once surgery is considered necessary. The prophylactic use of mitral valvotomy during pregnancy is not recommended, but once the decision to operate has been made, the procedure should not be unduly delayed (94, 105).

Metcalfe and co-workers have suggested that, ideally, surgery should be done after twelve weeks gestation, when organogenesis is complete and the risk of spontaneous abortion is less, but before the time of maximal increase in cardiac output at about 20 weeks (70). This, however, is nearly never possible due to the unpredictable response of individual patients with mitral stenosis to the haemodynamic changes of pregnancy.

5.2 OPERATIVE MANAGEMENT

The operative management of these patients is important as both mother and foetus are at risk. Anaesthetic management include meticulous monitoring, pre-oxygenation and careful induction to avoid hypoxia or haemodynamic instability. Most anaesthetic agents cross the placenta.

For a closed mitral valvotomy the patient is placed in the right lateral decubitus position so that the uterus falls

away from the inferior vena cava, which avoids postural changes in cardiac output (31, 47, 50,85). The operation should be accomplished expeditiously, and meticulous technique and haemostasis at the end of the procedure is essential (36).

Post-operatively, monitoring should be continued and where necessary mechanical ventilation maintained for the first 24 hours (31). However, most of our patients could be extubated at the end of the operation without undue effects. Adequate analgesia is necessary to prevent pain and anxiety causing uterine irritability.

5.3 RESULTS OF CLOSED MITRAL VALVOTOMY IN PREGNANCY

5.3.1 MATERNAL OUTCOME

All the patients in our group survived the operation and pregnancy. The low reported maternal and foetal mortality is evidence of the safety of closed mitral valvotomy in pregnancy. The results compare favorably with other reports in the literature (61).

In a review of 514 cases of mitral valvotomy in pregnancy in 1965, Ueland concluded that cardiac surgery during pregnancy is a beneficial procedure compared to medical management alone and that it resulted in lower maternal (1.7%) and foetal mortality (99).

Studying the outcome of 42 pregnant patients, who, over an 8 year period underwent closed mitral valvotomy for mitral stenosis, El-Maraghy and associates concluded that the procedure of closed mitral valvotomy offered excellent low risk palliation (29). In 3 other reviews a maternal mortality of 3.4% was reported by Szekely (92), 3% by Mendelsohn (66) and 1.8% by Harken and Taylor (36).

5.3.2 FOETAL MORTALITY AND MORBIDITY

The foetal mortality in our study (11.4%) is comparable with a perinatal mortality of 15% reported by Knapp and Arditi (48). The foetal loss in the studies of Harken and Taylor, and Ueland were 9% and 7.5% respectively (36, 99).

Foetal mortality is directly related to the severity of maternal heart disease (72). Reduced uterine blood flow, the result of maternal heart disease, changes placental physiology and thus impairs growth, development and viability of the foetus (78).

Loss of the conceptus among patients with cardiac disease who maintained sinus rhythm throughout pregnancy was 7.8% in contrast to 17% in cases where rheumatic heart disease was complicated by atrial fibrillation (91, 92, 93). Foetal mortality similarly was virtually nil in asymptomatic patients (Class 1), but 30% in pregnant women symptomatic at rest (Class 4) (88, 93).

In the current study, two abortions resulted from midtrimester procedures and three babies were prematurely stillborn after surgery during the third trimester. However, only two patients required valvotomy during the first 3 months of their pregnancy. The stress of surgery appears to have little effect on babies born alive.

Although of smaller size, premature and dysmature babies survived the perinatal period.

5.4 LONGTERM VALVE RESULTS

The best results with closed mitral valvotomy are obtained in patients with mobile leaflets and without fibrotic shortening of the chordae and papillary muscles or thickened leaflets (61). Most of our patients were young (average age 27 years) with mobile mitral stenosis and no mitral regurgitation, which would have been a contra-indication to the operation.

An actuarial analysis of results from this institution by Commerford, Hastie and Beck, in 1978 of 654 patients over a 12 year period and an analysis of pre-operative predictors of longterm survival, concluded that closed mitral valvotomy still has a definite role in managing mobile mitral stenosis, particularly in areas where there is a high incidence of rheumatic cardiac disease and a large number of young patients having mobile stenotic valves. A low operative mortality of 2.97% was found (21).

Closed mitral valvotomy does not guarantee the patient freedom from cardiac complications in subsequent

pregnancies. It is recommended that such patients should complete their families without delay following the operation as the incidence of restenosis and recurrence of symptoms increases as time passes (90, 91). Thus close supervision of these patients remains mandatory as they may become symptomatic again during subsequent pregnancies (92).

In this series mitral valve restenosis requiring surgery developed 5 - 17 (mean 10.0) years after closed mitral valvotomy, which would allow enough time for a young woman to complete her family.

Twelve subsequent surgical procedures for mitral valve restenosis were done in 10 patients (23.2%). Repeat closed mitral valvotomy was performed in 4 patients (in 2 during a subsequent pregnancy). An open mitral valvotomy was done in one, and seven patients underwent mitral valve replacement.

5.5 CLOSED MITRAL VALVOTOMY VS PERCUTANEOUS BALLOON MITRAL VALVULOPLASTY

Percutaneous balloon mitral valvuloplasty via the femoral route is emerging as an alternative to closed mitral valvotomy with satisfactory results (2, 3, 40, 56). The candidates for balloon mitral valvuloplasty are identical to those suitable for closed mitral valvotomy - isolated mitral

stenosis with minimal mitral incompetence, pliable leaflets, minimal calcification and no history of systemic emboli.

The advantages are avoidance of general anaesthesia and surgery (resulting in scarring and adhesions), its application in poor surgical risk patients and limited period of hospitalization. Although risks like cardiac tamponade, thrombo-embolism, residual atrial septal defect, mitral incompetence and severe blood loss requiring transfusion do occasionally occur, the results reported are excellent (38, 59).

The initial concern about prolonged exposure to radiation during screening with the use of this procedure during pregnancy may have been overemphasized. Use of modern X-ray equipment and appropriate screening has reduced the radiation dose to the foetus to acceptable levels.

6. CARDIOPULMONARY BYPASS AND PREGNANCY

The increasing safety of cardiopulmonary bypass and valve repair under direct vision has raised a generation of surgeons with limited experience in closed operations, whose claims that open procedures may result in better longterm outcome may well be correct (105). Reporting on non-pregnant patients, Gerami's group and others, found that completeness and overall safety of a closed operation compared less favorable with the results obtained with open mitral valvuloplasty (25, 32, 45, 52).

In 1958, Leyse reported the first case where cardiopulmonary bypass in the eighteenth week of pregnancy was used. Although the pregnancy was completed, the baby was born with multiple congenital defects and died later at the age of four months (54).

In a collective review in 1969, Zitnik concluded that open cardiac procedures did not expose the pregnant mother or foetus to undue risk and recommended that it should preferably be done in the 24th - 28th week after completion of organogenesis. The foetal mortality, however, was 33% (108).

Since this paper, reports of extracorporeal circulation during pregnancy have been limited to sporadic and isolated

cases (18, 26, 51,77, 107), until another collective review was published by Bernal and Miralles in 1986. Forty six patients were divided into two groups - those who had emergency procedures (prosthetic valve thrombosis, rupture of thoracic aortic aneurysm, pulmonary embolism or infective endocarditis) and those who had semi-elective surgery for congenital heart defects, rheumatic valve disease, atrial myxoma or unstable angina. One maternal and nine foetal deaths (19.1%) occured. No relationship between gestational age at operation and foetal mortality could be established (9). Babies with congenital defects were born to mothers placed on bypass in the first trimester as found by other authors as well (8, 9, 51, 53, 54, 108).

Becker, who analysed the experience of the Society of Thoracic Surgeons reported one maternal death (1:5%) and 11 foetal deaths (16%) in 68 procedures where cardiopulmonary bypass was used (7).

The use of cardiopulmonary bypass should be avoided if at all possible and is acceptable only for surgically amenable lesions before resorting to therapeutic abortion. Where extracorporeal circulation is used, it is with much higher foetal risk, which may need to be accepted as the life of the mother is the most important consideration. If cardiopulmonary bypass is necessary, the second trimester seems to be optimal as abortion and congenital malformations

which mostly complicate first trimester procedures and premature labor during the third trimester are the major causes of foetal loss (7, 8, 108). Lately foetal heart and uterine monitoring have contributed to safety of surgery during pregnancy (41, 49, 109).

CONCLUSIONS

During pregnancy the operative mortality for closed mitral valvotomy is low. The risk of the procedure in pregnancy seems no greater than that which would be incurred if the pregnancy was allowed to continue without the operation and valvotomy done postpartum. In addition, foetal survival and the incidence of later mitral valve restenosis is acceptable.

Indications for closed mitral valvotomy include uncontrollable pulmonary oedema or haemoptysis as well as failure to respond to optimal medical therapy, provided that physical and echocardiographic signs of mobility of the valve are present.

Closed mitral valvotomy gives satisfactory results in patients with mitral stenosis during pregnancy and can be performed at any stage.

FIGURE 1

Typical electrocardiogram of a 25 year old woman with tight mitral stenosis presenting with pulmonary edema during pregnancy - left atrial enlargement is manifested by the P mitrale seen in lead II, and pulmonary hypertension by the rightward shift of the QRS axis (early right ventricular hypertrophy).

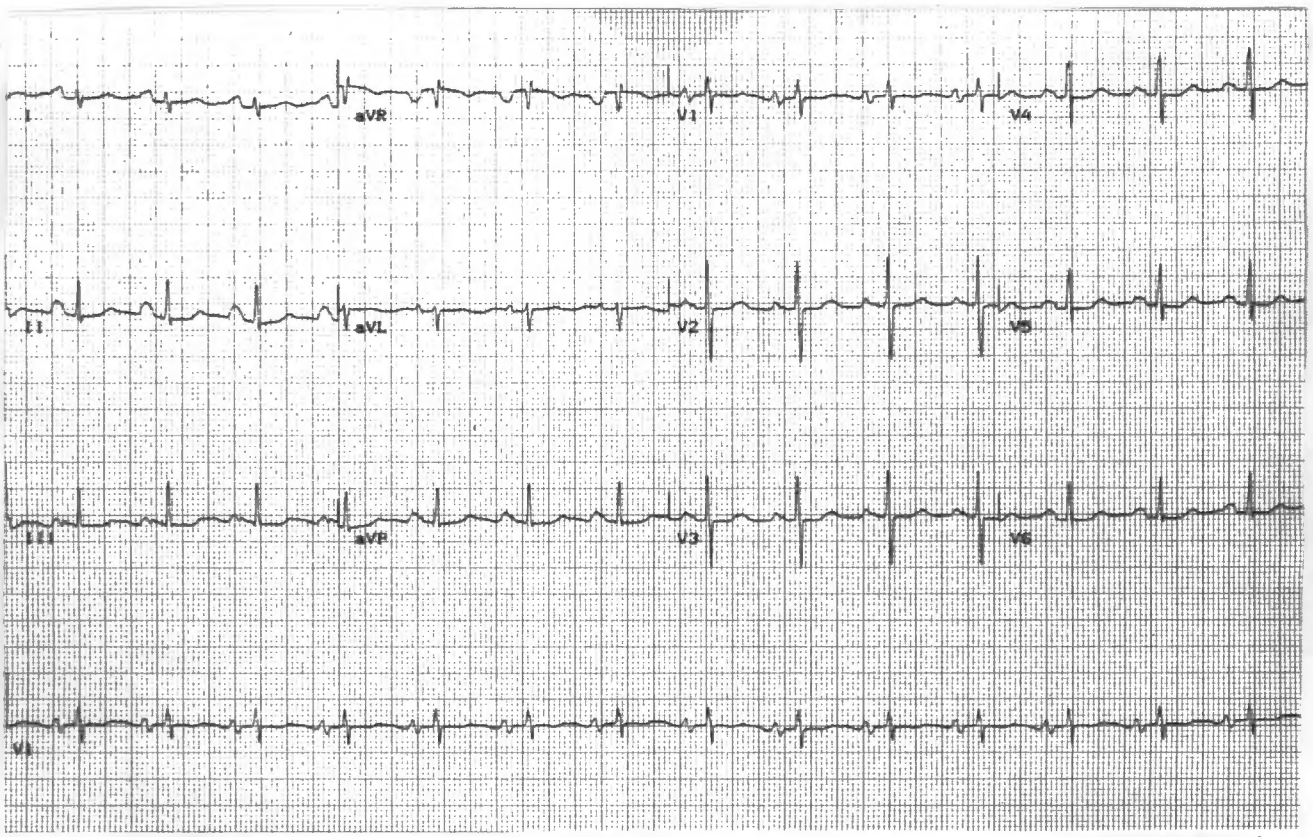


FIGURE 2

Radiological chest appearance of mitral stenosis - enlarged LA and upper lobe venous diversion - plus pulmonary edema in a 19 year old patient in this series.

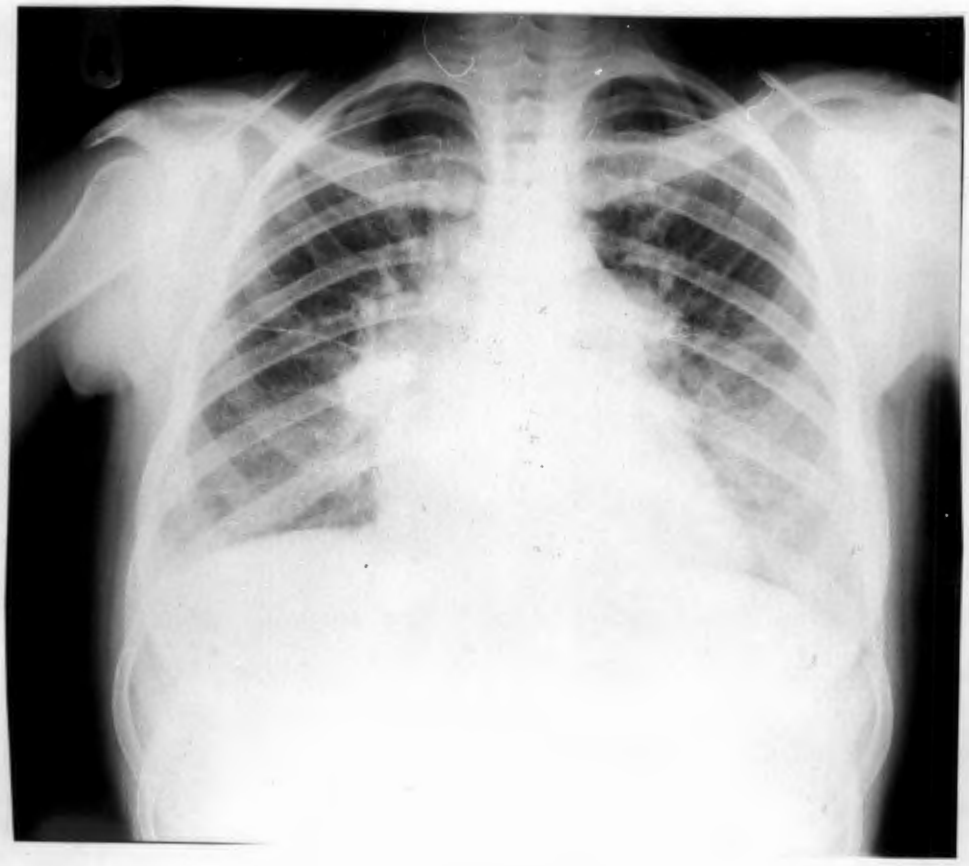


FIGURE 3

Distribution of 43 patients who had closed mitral valvotomy demonstrating number of pregnancies.

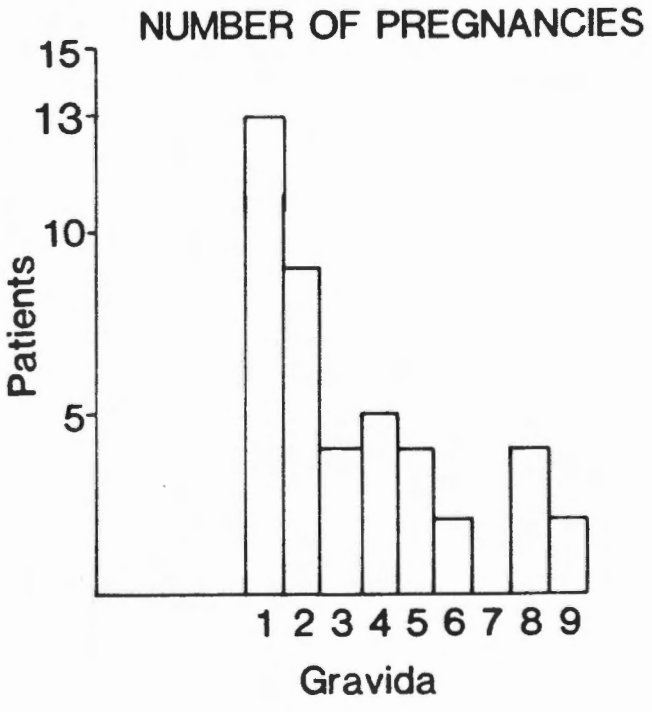


FIGURE 4

Distribution of patients according to trimester of pregnancy at time of operation.

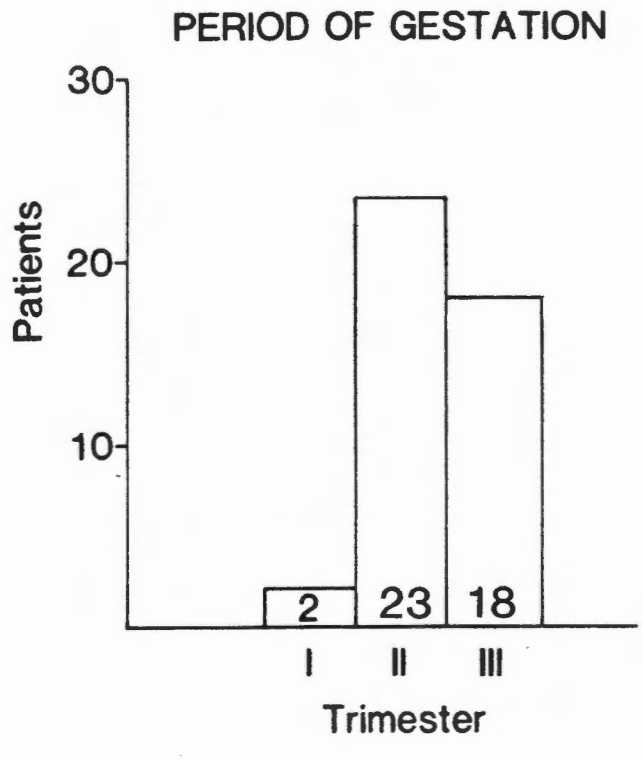


FIGURE 5 (a)

Closed mitral valvotomy using the Tubb's dilator.

(a) The dilator is adjusted precisely before insertion to allow a controlled dilatation of 3 to 4 cm.

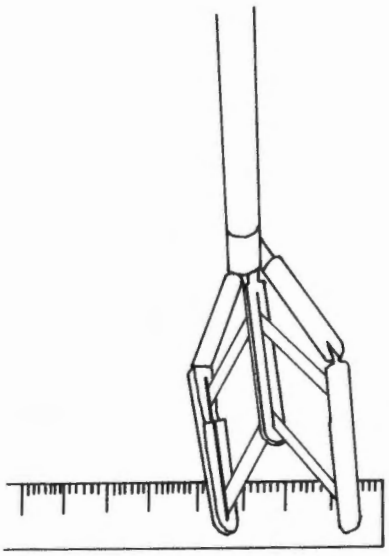


FIGURE 5 (b)

Closed mitral valvotomy using the Tubb's dilator.

(b) The right index finger of the surgeon via the left atrial appendage guides the Tubb's dilator across the stenosed mitral valve orifice, having assessed the valve digitally before to determine leaflet mobility and presence of regurgitation.

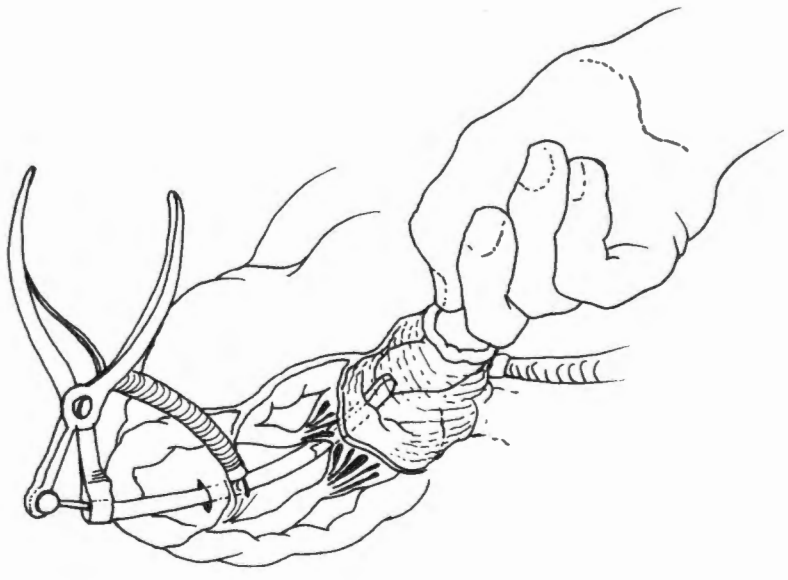


FIGURE 5 (c)

Closed mitral valvotomy using the Tubb's dilator.

(c) The dilator is then opened to split the orifice open along the fused commissures.

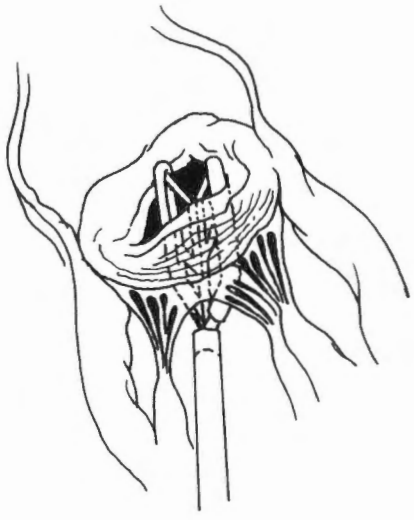


FIGURE 6

An asymptomatic 20 year old woman, pregnant with twins, 8 weeks after satisfactory closed mitral valvotomy during the second trimester.

FIGURE 7

Symptomatic relief in 43 patients after closed mitral valvotomy according to New York Heart Association classification.

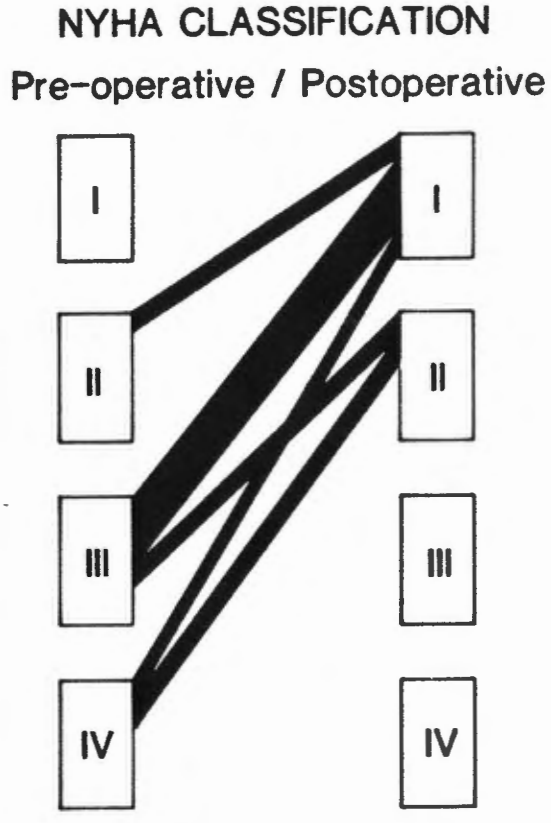


FIGURE 8

Histogram illustrating total baby mortality as well as deaths of babies during individual periods of pregnancy following closed mitral valvotomy

(On Term = Gestation more than 37 weeks

Premature = Gestation 28 - 37 weeks

Abortion = Gestation less than 28 weeks)

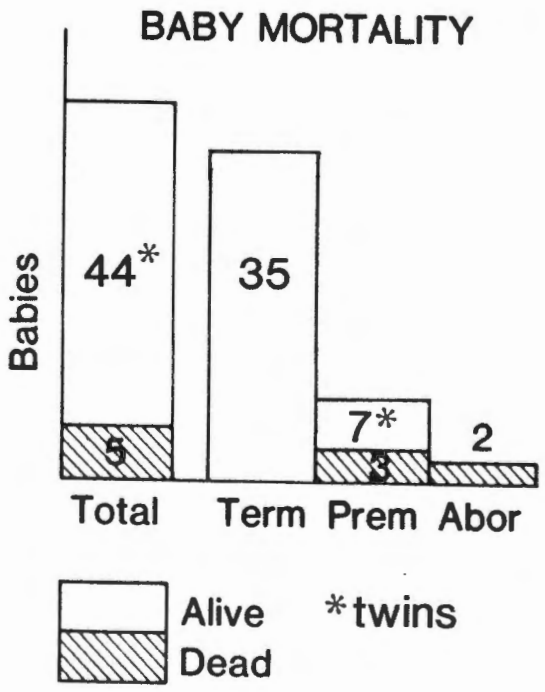


FIGURE 9

Foetal survival rate in 44 babies (once twins) following closed mitral valvotomy during pregnancy (gestation in weeks (w)).

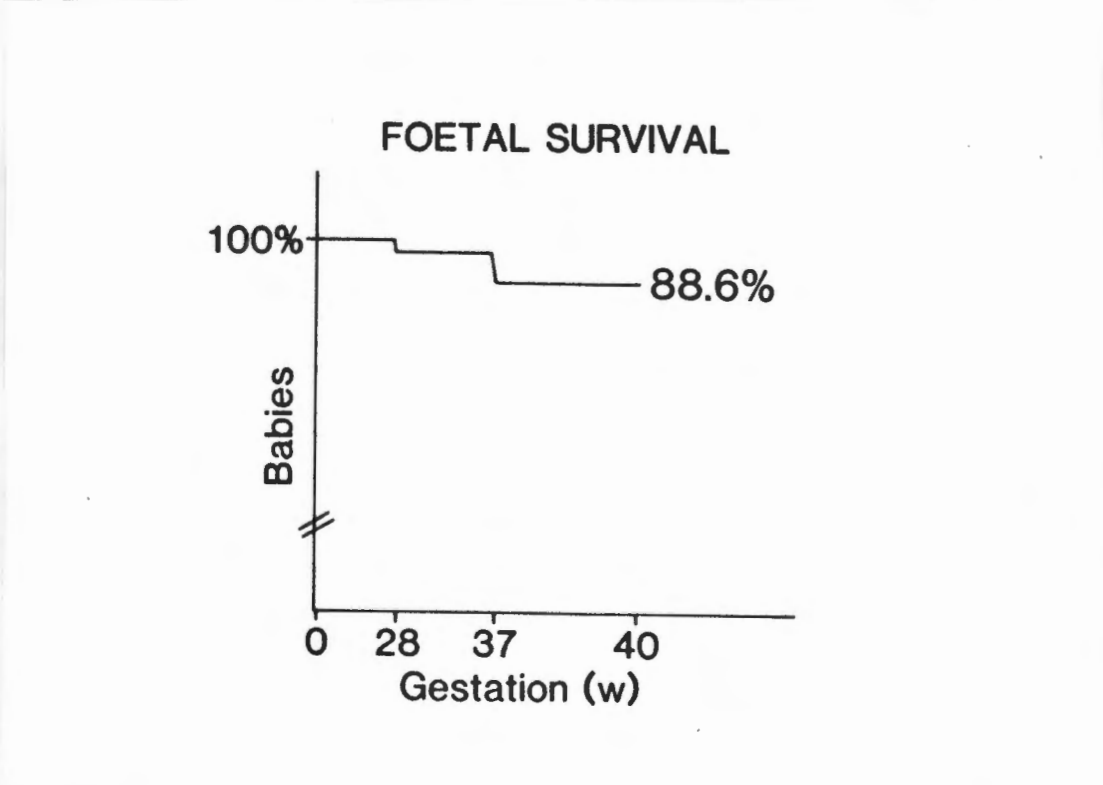


FIGURE 10

Table illustrating the outcome of pregnancy in 43 cases related to the trimester at the time of operation.

TRI-	NO. OF	ABOR-	PREMATURE			TERM		
MESTER	OPERATIONS	TIONS	STILL	DYSM	ALIVE	STILL	DYSM	NORM
1	2					1		1
II	23	2		1*	1*			20
III	18		3		1		1	13

* TWINS

FIGURE 11

Post-operative valve survival curve indicating freedom from reoperation (repeat closed valvotomy, open mitral valvotomy or mitral valve replacement) following closed mitral valvotomy in pregnancy.

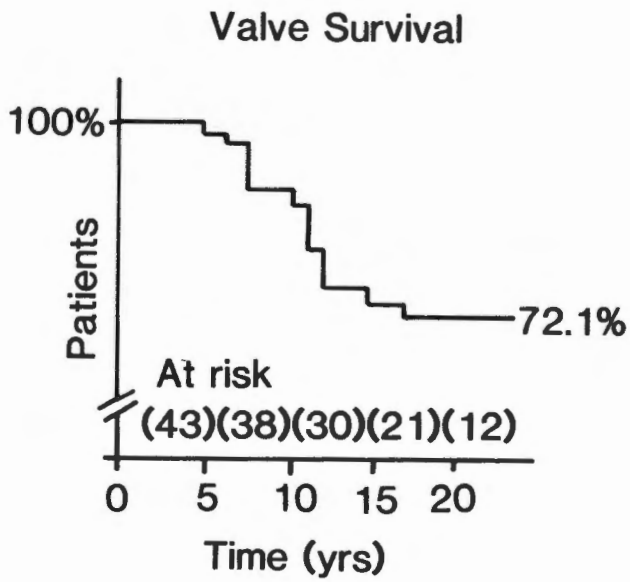
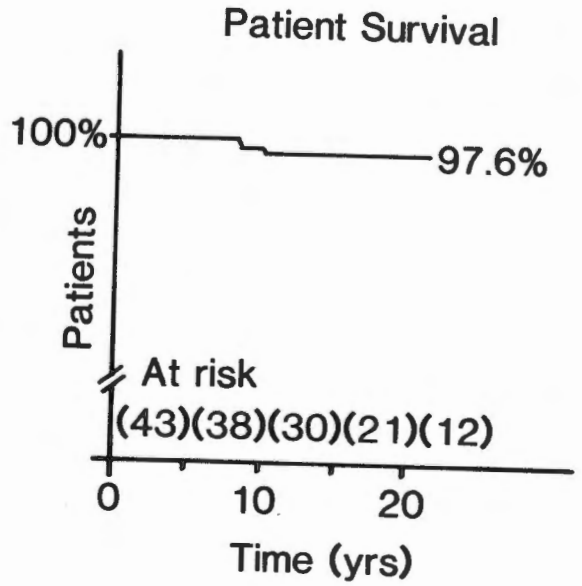


FIGURE 12

Survival rate of patients following closed mitral valvotomy in pregnancy related to time in years post-operatively.



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APPENDIX A SUMMARY OF PATIENT DATA AND RESULTS

Number of patients : 41
Number of operations : 43
Age range : 18 - 41 years
Mean age : 27 years

CLINICAL PRESENTATION

SYMPTOMS AND SIGNS :
- Acute rheumatic fever history : 15 cases (32%)
- Increased dyspnoea : 9 patients
- Pulmonary oedema : 17 patients
- Haemoptysis : 4 patients
- Pulmonary oedema plus haemoptysis : 13 patients

SPECIAL INVESTIGATIONS :

ECG:
- Sinus rhythm : 41 patients
- Atrial fibrillation : 2 patients

CXR:
- Left atrial enlargement : 43 cases
- Upper lobe venous diversion : 43 cases
- Right ventricular hypertrophy : 10 cases
- Pulmonary oedema : 30 cases

OPERATION DATA (Mortality = 0%)

INCISION :
- Left anteriolateral thoracotomy : 41 cases
- Median sternotomy : 2 cases

PROCEDURE :
- Tubb's dilator distance : 32 - 42 mm
- Satisfactory result : 41 cases
- Anteromedial commissure split only : 2 cases

SYMPTOMATIC IMPROVEMENT (according to NYHA classification) :

Class	I	II	III	IV
preoperative (mean 3.01)		6	25	1
postoperative (mean 1.22)	33	10		

MITRAL VALVE RESTENOSIS

Follow-up : 1 - 22 years (1965 - 1987)
Longterm mortality : 2 patients
Symptomatic, not yet requiring reoperation : 2 patients

REOPERATIONS (n = 12) :
- Repeat closed mitral valvotomy : 4 cases
- Open mitral valvotomy : 1 case
- Mitral valve replacement : 7 cases

OBSTETRIC DATA

Number of pregnancy	1	2	3	4	5	6	7	8	9
Number of patients	13	9	4	5	4	2	3	-	2

Trimester at surgery	I	II	III
Number of patients	2	23	18

DELIVERY :
- Caesarian section : 3
- Assisted vaginal : 3 (forceps), 1 (vacuum)
- Normal : 34

FOETAL OUTCOME

Total foetal survival : 37 babies (88.6%)

Foetal mortality (n = 5) :
- Abortion : 2
- Premature stillborn : 3

Foetal morbidity (n = 5) :
- Dysmature : 2
- Premature live : 3