



شبكة المعلومات الجامعية
التوثيق الإلكتروني والميكرو فيلم

بسم الله الرحمن الرحيم



HANAA ALY



شبكة المعلومات الجامعية
التوثيق الإلكتروني والميكروفيلم



شبكة المعلومات الجامعية التوثيق الإلكتروني والميكروفيلم



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جامعة عين شمس

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قسم

نقسم بالله العظيم أن المادة التي تم توثيقها وتسجيلها
علي هذه الأقراص المدمجة قد أعدت دون أية تغيرات



يجب أن

تحفظ هذه الأقراص المدمجة بعيدا عن الغبار



HANAA ALY

INTRODUCTION

Banding of the pulmonary artery has been proposed as a therapeutic procedure for patients with a large left-to-right shunt and pulmonary artery hypertension not amenable to complete correction. It was theorized that by increasing the resistance to outflow from the right ventricle, and thus lowering the pulmonary artery pressure, the size of the left-to-right shunt would diminish, the high output failure state would improve, and the pulmonary arterioles might be protected from developing progressive intimal changes (*Muller and Dammann, 1952*).

The original proposal by Muller and Dammann was reported in 1952, at a time when surgery with the pump oxygenator was not available. It had been assumed that, with the introduction of cardiopulmonary bypass, this palliative procedure would be outmoded and discarded, and all left-to-right shunts would be repaired in a one-step open-heart Procedure (*Kirklin, 1960*).

The first successful operation for the creation of pulmonic stenosis was performed on July 11, 1951, and reported the following year in Surgery, Gynecology and Obstetrics. The report, entitled “The Treatment of Certain Congenital Malformations of the Heart by the Creation of Pulmonic Stenosis to Reduce Pulmonary Hypertension and Excessive Pulmonary Blood Flow: A Preliminary Report, ” by

William H. Muller, Jr., M.D., and J. Francis Dammann, Jr., M.D. is remarkable in several ways.

It provides a definition and explanation of the pathophysiology of pulmonary hypertension with interventricular communications, and it develops the logic for treatment by the creation of pulmonic stenosis (*Muller and Dammann, 1952*).

They advocated this operation for patients with a single ventricle or large VSD resulting functionally in two ventricles that act as one. The intent was to reduce pulmonary artery size by two thirds in order to decrease pulmonary blood flow by 50% (*Muller and Dammann, 1952*).

The first operation took place on July 11, 1951 on a 5-months old infant who weighed 3.5 kg. The chest was entered anteriorly through the second left interspace, and a U clamp was placed to excise a segment of pulmonary artery (*Nolan, 1987*).

A 1-cm umbilical tape was placed around the pulmonary artery and sutured in place. The excluded part of the pulmonary artery was excised to decrease the chance of the band eroding into the pulmonary artery. The band was placed to prevent the pulmonary artery from growing larger than originally intended (*Nolan, 1987*).

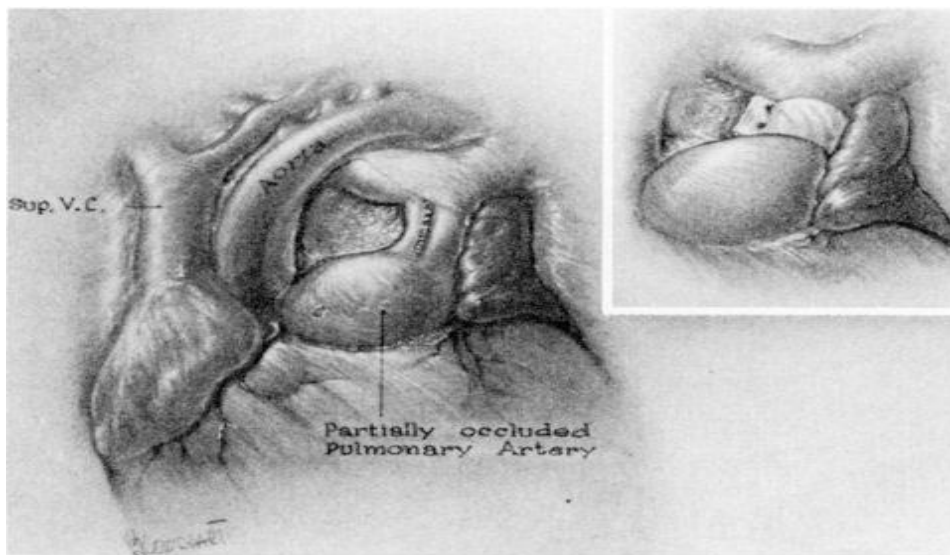


Figure 1: The pulmonary artery has been narrowed to approximately one third of its original diameter. Inset shows the pulmonary artery band, which is composed of umbilical tape wrapped in polyethylene and sutured around the main pulmonary artery (Sup VC: superior vena cava).

Follow-up of the original concept was presented before the Southern Surgical Association on December 8, 1955. Twenty-eight operations had been performed on 25 patients between 1951 and 1955.

There were nine operative deaths and one late death due to excessive narrowing of the pulmonary artery. Five of these deaths occurred in the operating room before the pulmonary artery could be narrowed, and were due to the fragile condition of the patients and the anesthetic techniques of that era (*Muller and Dammann, 1956*).

The authors predicted the patients for whom this operation would be important in the future: "The procedure is a

palliative one, and should be used only on those cases in which an open cardiac procedure with definitive repair of intracardiac defects cannot be effected (*Muller and Dammann, 1956*).

In neonates and early infants with low body weight (BW), cardiopulmonary bypass surgery increases organ edema and dysfunction, leading to high mortality and morbidity (*Oppido et al., 2004*).

Pulmonary artery banding (PAB) remains a useful surgical palliation for small neonates and early infants with excessive pulmonary blood flow who are unable or difficult to withstand an open heart surgery using cardiopulmonary bypass (*Nagashima, 2011*).

PAB can relieve the symptoms of heart failure and respiratory distress and prevent future pulmonary vascular obstructive disease in these infants and intracardiac repair (ICR) can be delayed until they grow (*Nagashima, 2011*).

Trusler and Mustard (1972) advocated that the adequate circumference of the PAB using a 4-mm-wide tape in infants without cyanosis was (20 mm + 1 mm for each kg of the infant BW) (*Nagashima, 2011*).

Recently especially in developed countries with increase of cardiac surgeon experience and possibility of Cardiopulmonary bypass (CPB) in very young infant with low birth weight and also better postoperative care, many of

children that previously had to tolerated initially PA Banding procedure before corrective surgery, today have a chance for total corrective surgery with good result and success and low mortality (*Hoseinikhah et al., 2016*).

At current in some centers there is also noticeable percentage of this palliative surgery for congenital heart disease with pulmonary over circulation to prevent of irreversible pathologic changes in pulmonary vasculature and lung parenchyma (*Hoseinikhah et al., 2016*).

AIM OF THE WORK

Aim of this work is to construct a strategy in management of infants who underwent pulmonary artery banding at Ain Shams University Hospitals Cardiothoracic Department including the following points:

1. Study different operative techniques.
2. Study different postoperative clinical pathways.
3. Study postoperative complications, mortality and morbidity.

ANATOMY OF CONGENITAL CARDIAC DISEASES (CHDs)

Congenital heart diseases (CHD) basically consist of defects of the cardiac architecture that interfere with the venous drainage, septation of the cardiac chambers, and their sequences and regular function of the valve apparatuses without obstacles in blood flow (*Anderson and Becker, 1986*).

Noninvasive imaging that is now available can perfectly detection vivo cardiac abnormalities, thus providing precise diagnosis and rendering it feasible to plan optimal surgical/interventional repair (*Anderson and Becker, 1992*).

When approaching the diagnosis of any CHD, you have to keep in mind that the normal heart is like a three-storey building (atria, ventricles, and great arteries) the atria are the platform, the ventricles the first floor, and the great arteries a second floor, each level connected to each other through valve orifices and each half is totally separated by septa, disposed in such a way to allow deoxygenated venous blood to go to the lungs through the pulmonary artery and the oxygenated venous blood to go to the systemic organs through the aorta. Small and great circulations are in sequence, with no connection to each other than the lung capillary network (*Anderson and Becker, 1992*).

The aim of any surgical/interventional repair is to reconstruct a separate sequence of cardiac segments and to restore the regular physiology of blood circulation (*Anderson and Becker, 1992*).

Anatomy of congenital cardiac diseases with pulmonary artery overflow

The communication between left and right heart accounts or left-to-right shunt in postnatal circulation because of the progressive gradient of resistance in between the great and small circulations. The shunt may occur at:

1. **Venous pole:** This is the case of anomalous pulmonary venous drainage, like the “scimitar syndrome, ” with the Right pulmonary vein draining into the inferior vena cava (Figure 2) or partial anomalous pulmonary venous drainage into the superior vena cava district or coronary sinus (*Anderson and Becker, 1992*).

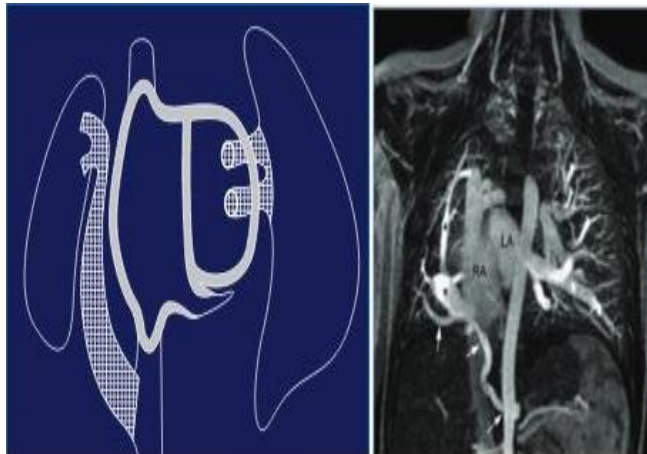


Figure 2: Congenital heart disease with increased pulmonary blood flow. Anomalous pulmonary venous drainage to inferior vena cava (Scimitar syndrome).

2. **Atrial septum:** A communication between the two atria in the atrial septum is usually located at the level of the fossa ovalis [“ostium secundum” or fossa ovalis type atrial septal defect (ASD)] (Figure 3) or just over the AV orifices (“ostium primum” ASD) in the setting of AV canal malformations (partial AV canal) (*Anderson and Becker, 1992*).



Figure 3: Congenital heart disease with increased pulmonary blood flow. Atrial septal defect ostium secundum or fossa ovalis type. (a) A communication between the two atria may does exist in the atrial septum at the level of the fossa ovalies: ostium secundum or fossa ovalies atrial septal defect. (b) view from the right atrium: a large interatrial communication is present at the level of the fossa ovalis (c) same defect viewed from the left atrium.

More rarely the interatrial communication may be observed at the root of superior vena cava, associated with partial anomalous pulmonary venous drainage of the superior right pulmonary vein (superior vena cava ASD) or with unroofed coronary sinus (inferior vena cava ASD) (*Anderson and Becker, 1992*).

3. AV junction (AV septum): This is the case of complete AV septal defect (complete AV canal) where a common AV valve and orifice does exist. An interatrial and interventricular communication are just above and under the common AV valve (Figure 4). This is the largest septal

defect inside the heart, accounting for a huge left-to-right shunt (*Anderson and Becker, 1992*).

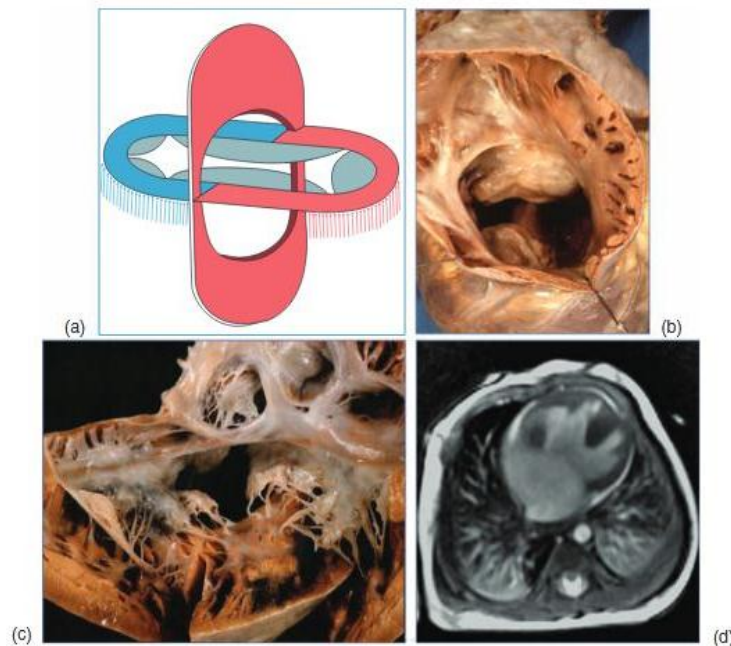


Figure 4: Congenital heart disease with increased pulmonary blood flow. Complete atrioventricular septal defect. (a) Schematic representation: a common atrioventricular valve is present and associated with a large atrioventricular septal defect with interatrial and interventricular communication. (b) view from the above: the common atrioventricular valve shows anterior (superior) and posterior (inferior) leaflets straddling the interventricular septum. (c) view from the right cardiac chambers: a complete form of atrioventricular septal defect is present with a common atrioventricular valve and a large communication at atrial and ventricular level. The anterior leaflet of the common valve shows chordae tendineae attached to the interventricular septum. (d) Four-Chamber view as visualized by cardiac magnetic resonance imaging: this heart presents with a large interatrial communication, a common atrioventricular valve, and a large interventricular septal defect.

4. Ventricular septum: Ventricular septal defects (VSDs) create an interventricular communication at the level or well

around the membranous septum (perimembranous VSD) (Figure 5) or in the inlet, apical or outlet muscular part of the ventricular septum musculature (VSDs) (Figure 6) (*Anderson and Becker, 1992*).

A peculiar VSD is that located in the distal infundibulum, just underneath the semilunar valves (sub arterial VSD), and may or may not be extended to the membranous septum (Figure 7).

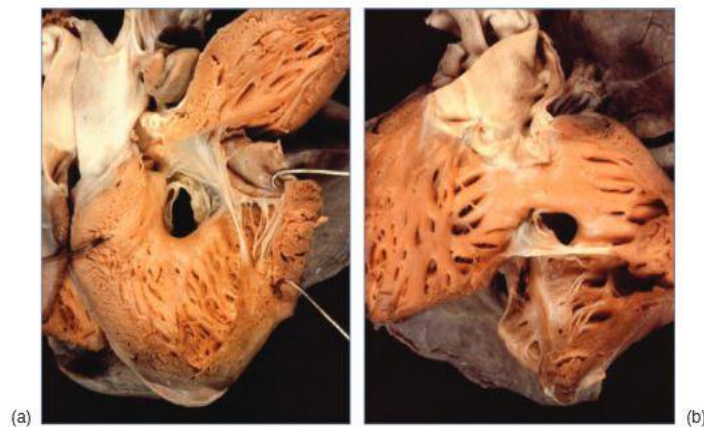


Figure 5: Congenital heart disease with increased pulmonary blood flow. Perimembranous ventricular septal defect (a) View from the left ventricle: the rim of the perimembranous defect is partially muscular and partially fibrous at the level of mitroaortic-tricuspid continuity. In this site (the posteriorinferior rim of the defect), the atrioventricular conduction system is located and at risk of lesion during surgical closure. (b) view from the right ventricle: the defect is located under the septal leaflet of the tricuspid valve. The muscle of Lancisi, located on the superior rim of the defect, indicates a posterior extension of the defect.