IMPROVEMENT IN LEFT VENTRICULAR DIASTOLIC DYSFUNCTION AFTER BALLOON PULMONARY VALVULOPLASTY

Thesis

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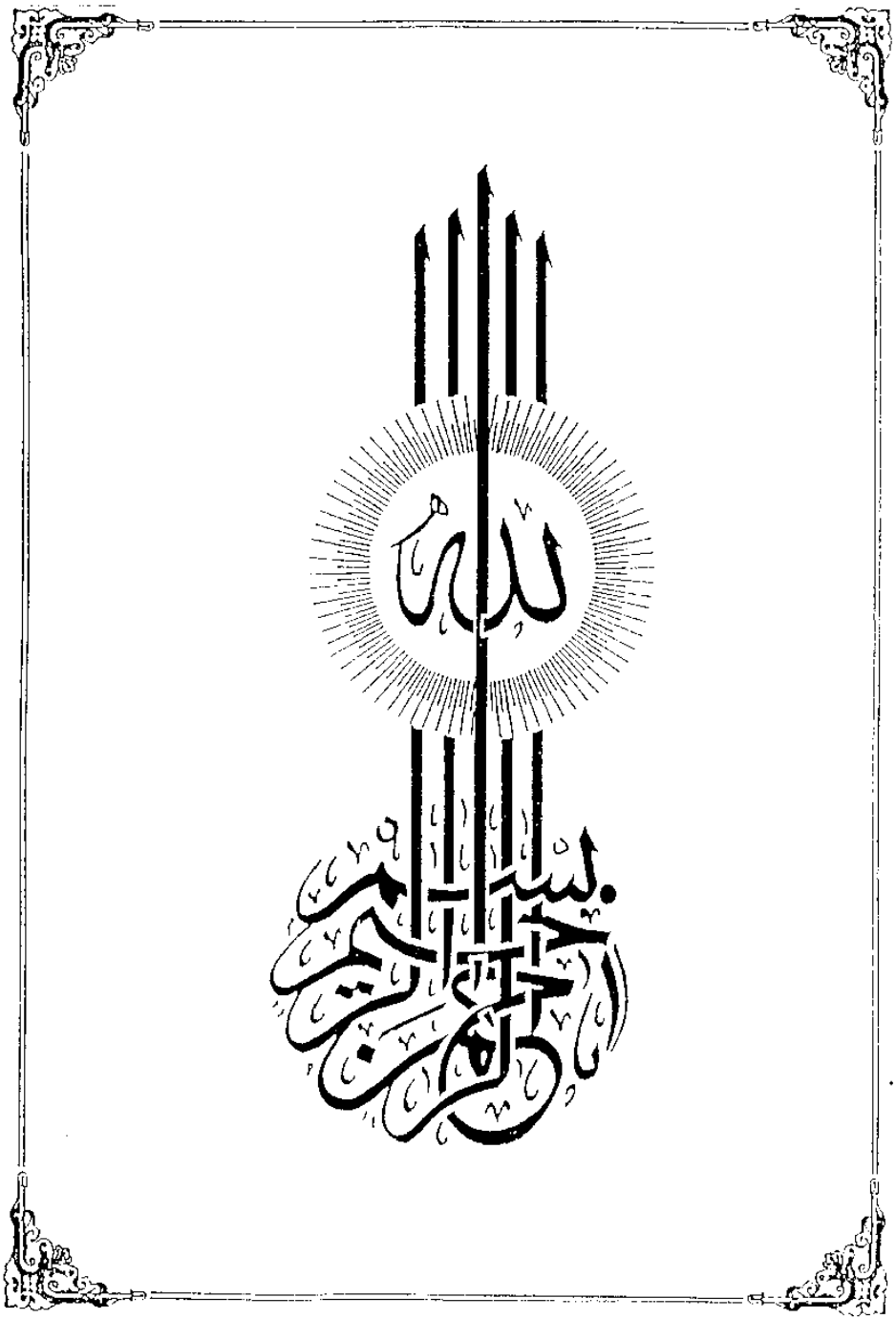
بسم الله الرحمن الرحيم



Dedicated to My Parents

With All Affection and Respect

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INTRODUCTION AND AIM OF WORK

INTRODUCTION AND AIM OF THE WORK

In the past evaluation of the myocardium had been limited to examining the systolic function of the heart. Recent investigatations however have demonstrated that abnormalities of diastolic function of the heart provide an important contribution to the signs and symptoms expressed in patients with heart disease. In addition, abnormalities of diastolic function may preceed abnormalities of systolic function in the early stages of disease.[1]

Chronic right ventricular pressure overload is now a known cause of left ventricular diastolic dysfunction. Louie et al[2] reported abnormal transmitral flow velocity profiles obtained by Doppler echocardiography in patients with chronic right ventricular pressure overload.

In addition Dittrich et al[3], found early improvement of left ventricular diastolic function after reduction of pulmonary artery pressure by pulmonary thromboendarterectomy, despite the presence of long standing pulmonary hypertension. Furthermore, this improvment occur in conjunction with a normalization of interventricular septal position and with an overall increase in left ventricular dimensions.

In the present study we will assess the reversibility of abnormal left ventricular diastolic function in patients with pulmonary stenosis after balloon pulmonary valvuloplasty. This will provide a unique opportunity to assess the reversibility of abnormal left ventricular diastolic function, because patients undergoing balloon pulmonary valvuloplasty show marked reduction in right ventricular pressure

overload without disturbing the pericardium, or influencing the left ventricular geometry or the Doppler echocardiographic indices of left ventricular diastolic function.

AIM OF THE WORK:

To assess the reversibility of abnormal left ventricular diastolic function in patients with pulmonary stenosis after undergoing balloon pulmonary valvuloplasty.

REVIEW OF LITERATURE

PULMONARY STENOSIS

HISTORICAL BACKGROUND :

Valvular pulmonary stenosis was first described in 1761 by John Baptist Morgagni of Pauda and subsequently its pathology was described by Meckel in 1817. However, it was Fallot in 1888 who separated seven cyanotic disorders from 39 cases of tetralogy and introduced the term morbid " triology " to describe the cyanotic form of pulmonary stenosis . Early postmortem studies by Abbott in 1936 suggested that pulmonary stenosis was rare; however, with the introduction of cardiac catheterization numerous reports have appeared , indicating that the fequency of this cardiac malformation is relatively high.[4]

GENERAL FEATURES OF THE PULMONARY VALVE :

The pulmonary valve consists of three cusps, anterior , left and right and their related sinuses , and three commissures that are designated left , right , and posterior .[5] The junction between the sinus portion and the distal tubular portion forms a prominent ridge , the sinotubular junction.[6] The pulmonary annulus contributes to the fibrous cardiac skeleton . it is a dense collagen ring , it assumes the shape of a triradiate crown , the three points of which attain the level of the sinotubular junction and demarcate the commissures . The opening at the level of the sinotubular junction constitutes the anatomic valvular orifice , and the circumference of a semilunar valve at autopsy is measured at this level rather than along the nonplanar annulus .

The plane of the pulmonary annulus faces posteriorly, leftward, and superiorly, the annular circumference measures 7.0 ± 1.0 cm and is approximately 0.5 cm greater in men than in women.[6]

Like the atrioventricular valves, the semilunar valves comprise two major layers histologically. The fibrosa forms the structural backbone of the valve and is continuous with the annulus, where as the spongiosa acts more as a shock absorber along the ventricular surface, especially at the closing edge. Additionally, the ventricularis and arterialis are thin linings along the ventricular and arterial surfaces, respectively. The cusp contains little elastic tissue and accordingly, has no appreciable elastic recoil, i.e. it acts as a passively mobile structure, it apparently has no memory of shape and, therfore has no tendency to assume either an open or closed position.

Since the annulus cannot cotnract, the opening and closing of the semilunar valves is a passive process, which entails cuspid excursion and annulocuspid hinge-like motion. However, during isovolumetric ventricular contraction, expansion of the arterial root may produce commissural separation and thereby initiates valvular opening.[6]

EMBERIOLOGY :

The truncus arteriosus is connected to the dorsal aorta in the embryo by six pairs of aortic arches.[7] By approximately day 32, the dextrosuperior and sinistroinferior truncal swellings have fused and have partitioned the truncus arteriosus. Distaly, along both sides of the truncal septum, tubercles form from each swelling and are destined to become the right and left cusps and sinuses of the pulmonary and aortic valves .

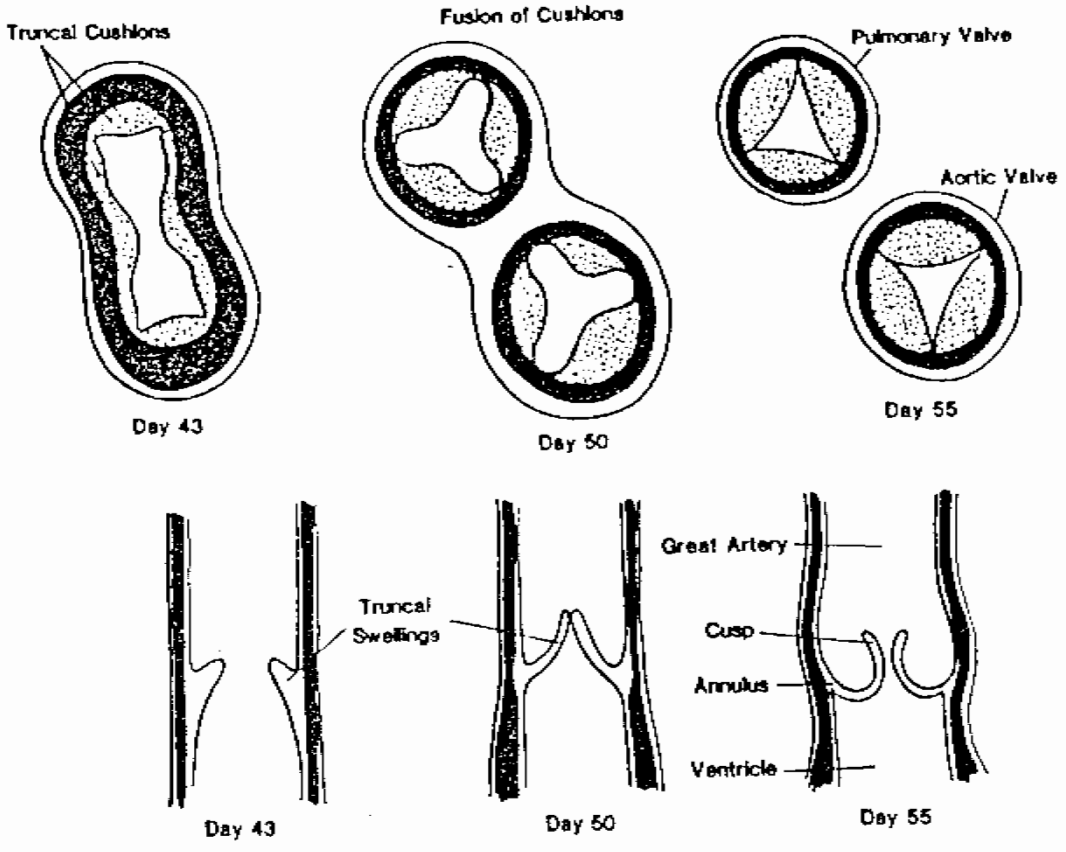

Fig.1 Embryology of the semilunar valves. **Upper**, the aortic and pulmonary valves form as fusion of the right superior and left inferior truncal swellings deviding the truncus arteriosus into two orifices. **Lower**, the cusps are produced by excavation and hollowing of the valve swellings. (From Edwards WD, 1987)[6]

Opposite the septum, between these two pairs of tubercles, arise s a pair of intercalated valve swellings which will form the anterior pulmonary and posterior aortic cusps and sinuses.[6] Rotation of the truncus coils the aorticopulmonary septum and creates the normal spiral relation between the aorta and pulmonary artery.[7] By a process of excavating and hollowing in a proximal direction , the semilunar cusps and their arterial sinuses are molded, between days 35 &55.[6]

Although the six aortic arches appear sequentially, portions of the arch system and dorsal aorta disappear at different times during embryogenesis. The first, second, and fifth sets of paired arches regress completely. The proximal portions of the sixth arches become the right, and left pulmonary arteries, and the distal left sixth arch becomes the ductus arteriosus.[7]

Most congenital anomalies of the pulmonary valve are associated with stenosis. Isolated pulmonary stenosis is almost always due to a dome shaped acommissural valve, with congenital fusion of all three commissures, that occur during mid to late intrauterine development.[7] However forms of pulmonary stenosis which are associated with other cardiac malformations, such as tetralogy of Fallot, usually result from a bicuspid or unicommissural valve (often with a hypoplastic annulus) or from a dysplastic valve with three thickened cusps.[6]

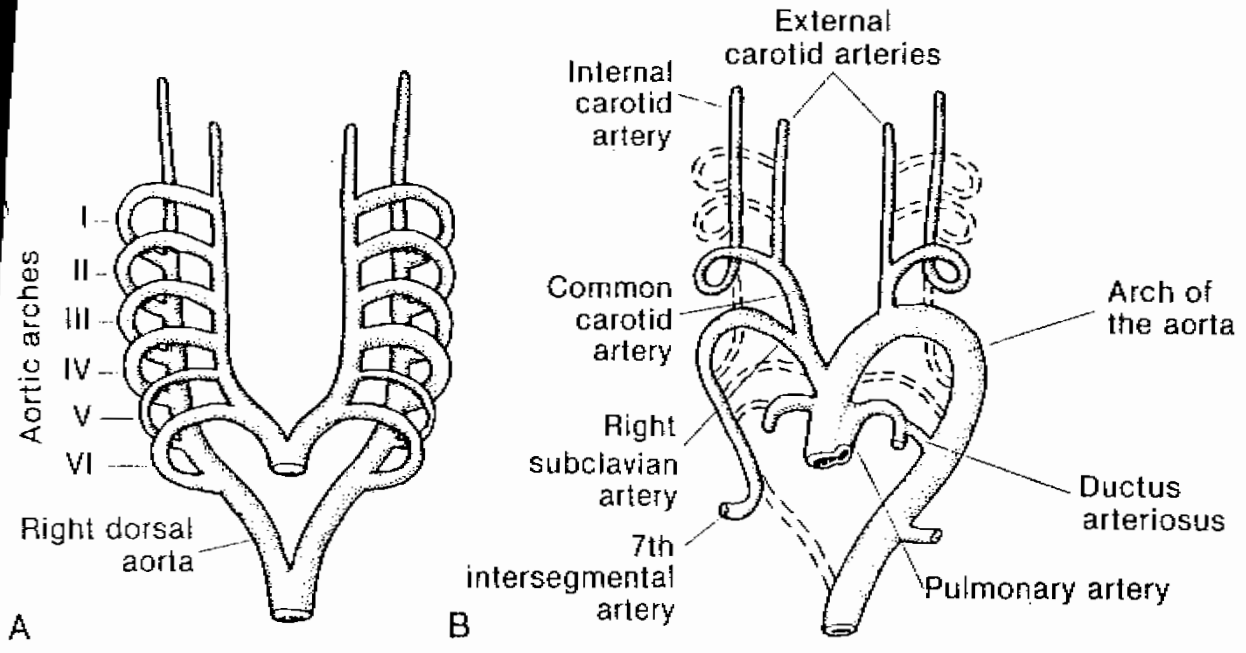

Fig.2 (A) Aortic Arches and dorsal aortas before transformation into the definite vascular pattern. (B) Aortic arches and dorsal aortas after transformation. The obliterated components are indicated by broken lines. (From Friedman WF, 1992)[7]