

EFFECT OF PREOPERATIVE PULMONARY ARTERY PRESSURE
ON SURGICAL OUTCOME IN CHILDREN BELOW TWO YEARS
WITH VENTRICULAR SEPTAL DEFECT (VSD)

Thesis

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cardiothoracic surgery**

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Introduction

Ventricular septal defects (VSDs) are among the most common congenital heart anomalies; isolated VSDs represent about 20% to 30% of all congenital cardiac malformations and have a prevalence of 1 to 2 per 1,000 live births. [1]

The first VSD closure was performed in **1954** by **Lillehei** and associates and now the primary closure for symptomatic patients is the surgical treatment of choice. [2]

Long-standing and neglected cases of ventricular septal defects may develop progressive pulmonary obstructive disease with severe pulmonary hypertension which may affect surgical outcome significantly including mortality, morbidity and lifestyle. [3]

However, some authors like **Blackstone** and colleagues, reported that preoperative pulmonary artery pressure and pulmonary vascular resistance are not determinants of early mortality at present, although they affect late results. [4]

This is supported by studies of **Rabinovitch** and colleagues, in which it was also found that surgical cure is likely to result in any infant in whom the VSD is repaired before age 6 to 9 months, irrespective of degree of pulmonary vascular disease. [5]

Aim of the work

This work will include 160 patients below two years coming for primary closure of ventricular septal defect at Ain-Shams university hospitals.

They will be divided into 2 groups (80 patients in each group) according to the severity of their preoperative mean pulmonary artery pressure. [6]

- **Group I:** patients with pre-operative pulmonary artery pressure below 55 mmHg (mild and moderate pulmonary hypertension).
- **Group II:** patients with pre-operative pulmonary artery pressure exceeding 55 mmHg (severe pulmonary hypertension).

These patients will be studied as follows:

1) Preoperative study including.

- Age
- Body weight
- Clinical picture
- X- ray findings
- ECHO findings especially
Right ventricular pressure (RVP), pulmonary artery pressure (PAP)
and pressure gradient across the ventricular septal defect.
- Cardiac catheterization data especially pulmonary artery pressure,
pulmonary vascular resistance, Qp/Qs and the effect of vasodilator
and 100% oxygen on these parameters.

2) *Operative study which include:*

- Aortic cross clamp time
- Total bypass time.
- Type and size of the ventricular septal defect.
- Pulmonary artery pressure before and after cardiopulmonary bypass by direct needle measurement.
- Weaning off bypass.

3) *Post operative study which include:*

*** Early postoperative course including:**

- weaning from mechanical ventilation.
- Inotropic support.
- Vasodilators.
- Pulmonary hypertensive crisis.
- Other morbidity and mortality.
- Postoperative ECHO after one week of surgery.

*** Late postoperative follow-up (up to 6 months) by:**

- Clinical evaluation.
- ECHO.
- Chest x-ray.

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تأثير ضغط الشريان الرئوي علي نتيجة الجراحة للأطفال تحت عمر السنتين المصابين بالثقب بين البطينين

رسالة مقدمه من الطبيب
باسم مفرح محمود

توطئه للحصول علي درجة الدكتوراه في جراحة القلب
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الملخص العربي

تعتبر حالات الثقب بين البطينين بالقلب هي أكثر الحالات شيوعا بين العيوب الخلقية للقلب. وتشكل مانسبته من ٢٠% الى ٣٠% من هذه العيوب وتحدث بمعدل من ١ الى ٢ لكل ١٠٠٠ مولود حي .

وتتمت أول جراحة لإغلاق الثقب بين البطينين عام ١٩٥٤ بواسطة ليليهي وزملائه. ولقد أصبح الآن إغلاق الثقب بين البطينين في مرحلة واحدة هو الحل الأمثل لمعظم هذه الحالات.

وتأخر هذه الحالات في إجراء التدخل الجراحي اللازم يؤدي الي ارتفاع مستمر في ضغط الشريان الرئوي وما يصاحبه من انسداد بهذه الشرايين الرئوية نتيجة زيادة سمك جدار هذه الأوعية الدموية مما يؤثر سلبا علي نتائج العمليات الجراحية اللازمة لإغلاق هذا الثقب بين البطينين.

وتشمل هذه الدراسة عدد ١٢٠ طفل تحت سن السنتين مصاب بهذا العيب في القلب وهو الثقب بين البطينين وهذه الحالات تعرضت لتدخل جراحي لإغلاق الثقب وذلك بمستشفيات عين شمس الجامعية ويتم تقسيم الحالات إلي مجموعتين في كل مجموعه ٦٠ حالة

المجموعة الأولى:

حالات مصحوبة بضغط الشريان الرئوي قبل العملية أقل من ٥٥ ملم زئبقي

المجموعة الثانية:

حالات مصحوبة بارتفاع ضغط الشريان الرئوي فوق ٥٥ ملم زئبقي

ويتم دراسة هذه الحالات من الجوانب الآتية

أولاً: دراسة العوامل قبل إجراء التدخل الجراحي مثل:

- السن
- وزن المريض
- الاعراض المرضية
- أشعة الصدر العادية
- موجات فوق صوتية علي القلب وخاصة بعض البيانات مثل
ضغط البطين الأيمن و ضغط الشريان الرئوي و تباين الضغط علي جانبي الثقب
- قسطرة القلب التشخيصية وخاصة بعض البيانات مثل:
ضغط الشريان الرئوي ومقاومة الشرايين الرئوية لتدفق الدم و معدل تدفق الدم بالشريان
الرئوي مقارنة بالشريان الاورطي وتأثير موسعات الشرايين ونسبة ١٠٠% أكسجين علي
هذه البيانات.

ثانياً: دراسة العوامل أثناء إجراء العملية وتشمل

- الوقت الكلي لتوقف القلب واستخدام ماكينة القلب الصناعي
- وقت إغلاق الشريان الاورطي وتوقف مد القلب بالدم عن طريق الشرايين التاجين
- أنواع وأحجام الثقوب بين البطينين
- ضغط الشريان الرئوي قبل توقف القلب وتشغيل ماكينة القلب الصناعي وبعد رجوع
القلب للانقباض وتوقف ماكينة القلب الصناعي.
- المضاعفات التي تحدث أثناء الجراحة

ثالثاً: دراسة العوامل بعد العملية وتشمل:

* متابعة المريض فترة ما بعد العملية مباشرة وتشمل:

- فصل المريض من جهاز التنفس الصناعي.
 - استخدام الأدوية المنشطة للقلب والدورة الدموية.
 - استخدام الأدوية الموسعة للأوعية الدموية.
 - الإرتفاع الحاد والمفاجئ لضغط الشريان الرئوي.
 - المضاعفات الأخرى ومعدل الوفيات.
 - موجات فوق صوتية على القلب بعد أسبوع من العملية.
- * متابعة المريض على المدى البعيد حتى فترة ستة أشهر بعد العملية وذلك عن طريق:

- الفحص الإكلينيكي.
- موجات فوق صوتية على القلب.
- أشعة عادية على الصدر.

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Historical Notes

The initial description of the clinical signs and symptoms of a VSD was ascribed to **Roger** in **1879**, and a small, flow-limiting VSD with associated normal pulmonary artery pressures carries the eponym (malady de **Roger** ventricular septal defect). [1]

At the other end of the clinical spectrum is a large VSD with severe pulmonary hypertension and fixed pulmonary vascular resistance resulting in cyanosis with right-to-left shunting through the VSD; this is known as the (**Eisenmenger** complex) following the description of such patient in **1897** when **Victor Eisenmenger** reported a case of a 32-year-old man with cyanosis and exercise intolerance. The patient developed congestive heart failure and died following an episode of hemoptysis. The autopsy demonstrated a large membranous ventricular septal defect VSD with an overriding aorta. [2]

Banding of the pulmonary artery as a palliative maneuver was first described in **1952**. [3] This decreased left-to-right shunting and as a consequence prevented the development of pulmonary vascular obstructive disease and left-sided volume overload. Until the mid-**1960s** when primary VSD closure became safer, pulmonary artery banding was the procedure of choice in managing VSDs. [4]

The first VSD closure was performed in **1954** by **Lillehei** and associates at the University of **Minnesota**, using controlled crosscirculation between the child and parent. Nineteen of the 27 patients who underwent this procedure survived. [5]

In **1955** **Kirklin** and associates at the **Mayo** Clinic closed a VSD using a heart–lung machine. [6] In **1958** transatrial VSD closure was performed by **Stirling** and **Lillehei** [7] followed in **1969** by the popularization of primary repair in symptomatic infants by **Barratt-Boyes** and associates using cardiopulmonary bypass, deep hypothermia, and circulatory arrest.[8]

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List of Abbreviations

ABG: Arterial Blood Gas
ACC: Aortic Cross Clamp
ANOVA: Analysis of Variance
Ao: Aorta
ARDS: Adult Respiratory Distress Syndrome
ASD: Atrial Septal Defect
CAVB: Complete Atrio-Ventricular Block
CHD: Congenital Heart Disease
CHF: Congestive Heart Failure
CPB: Cardio Pulmonary Bypass
CRBBB: Complete Right Bundle Branch Block
F: French
ICU: Intensive Care Unite
LA: Left Atrium
PA: Pulmonary Artery
PDA: Patent Ductus Areriosus
NO: Nitric Oxide
PT: Pulmonary Trunk
PVR: Pulmonary Vascular Resistance
PAH: Pulmonary Arterial Hypertension
Qp: Pulmonary Blood Flow
QS: Systemic Blood Flow
RV: Right Ventricular
SD: Standard Deviation
SVR: Systemic Vascular Resistance
TBP: Total Bypass
TV: Tricuspid Valve
VSD: Ventricular Septal Defect

Embryology Of the Ventricular Septum

The part of the cardiac loop that joins the inflow and outflow limbs was designated the bulboventricular fold in the classical literature. The bulboventricular fold is a narrowed area that represents the division between what will be the right ventricle (“BULBUS”) and the left ventricle (Figure 1). Both ventricular chambers are formed by ballooning of the outer curvature myocardium. This occurs concurrently with formation of trabeculae, which are myocardial diverticula extending from the compact, outer wall of the myocardium into the cardiac jelly toward the lumen. Formation of the trabeculae is the first manifestation of the differentiation of the nascent left and right ventricles. [9]

The ventricular chambers enlarge by growth and expansion of the myocardial wall on the greater or outer curvature.[10] Growth is closely followed by increasing trabeculae, which give the ventricular lumen a spongy appearance (Figure 1). The luminal passage through the bulboventricular fold is called the primary ventricular foramen (or bulboventricular foramen). The ventricular septum grows from the greater curvature at the site of the bulboventricular fold (Figure 1). However, the primary ventricular foramen is maintained at the leading edge of this crescent-shaped septum. In fact, the primary ventricular foramen never closes but actually enlarges in the fully developed heart to give left ventricular access to the aortic vestibule. [11]

The functional significance of trabeculae in early heart development is unknown. Histological studies in chick and human suggest that coalescence of the trabeculae results in formation of the muscular ventricular septum. [12] Other studies suggest that the muscular septum originates from the part of the ventricular wall that is interposed between the expanding free walls of the future right and left ventricles. [13] In fact, both processes may contribute to the growth of the ventricular septum. [11]

In human embryos at 5 weeks, a muscular septum is identifiable between the trabecular portions of both ventricles. The muscular ventricular septum is divided into regions designated the inlet portion of the septum near the atrioventricular septum and valves while the portion nearest the ventricular outflow is designated the outlet septum. The atrioventricular bundle of His is found on the crest of the caudal portion of the muscular ventricular septum. [14]

The last part of the ventricular septum to form is the membranous septum which forms from cushion tissue that fills in the opening left between the crest of the ventricular muscular septum and the atrioventricular mesenchymal septum. As the membranous portion of the septum develops, it also forms the primordium for the septal cusp of the tricuspid valve. During sculpting of the septal leaflet it is detached from the interventricular septum at the fifth month of human fetal development.[15]

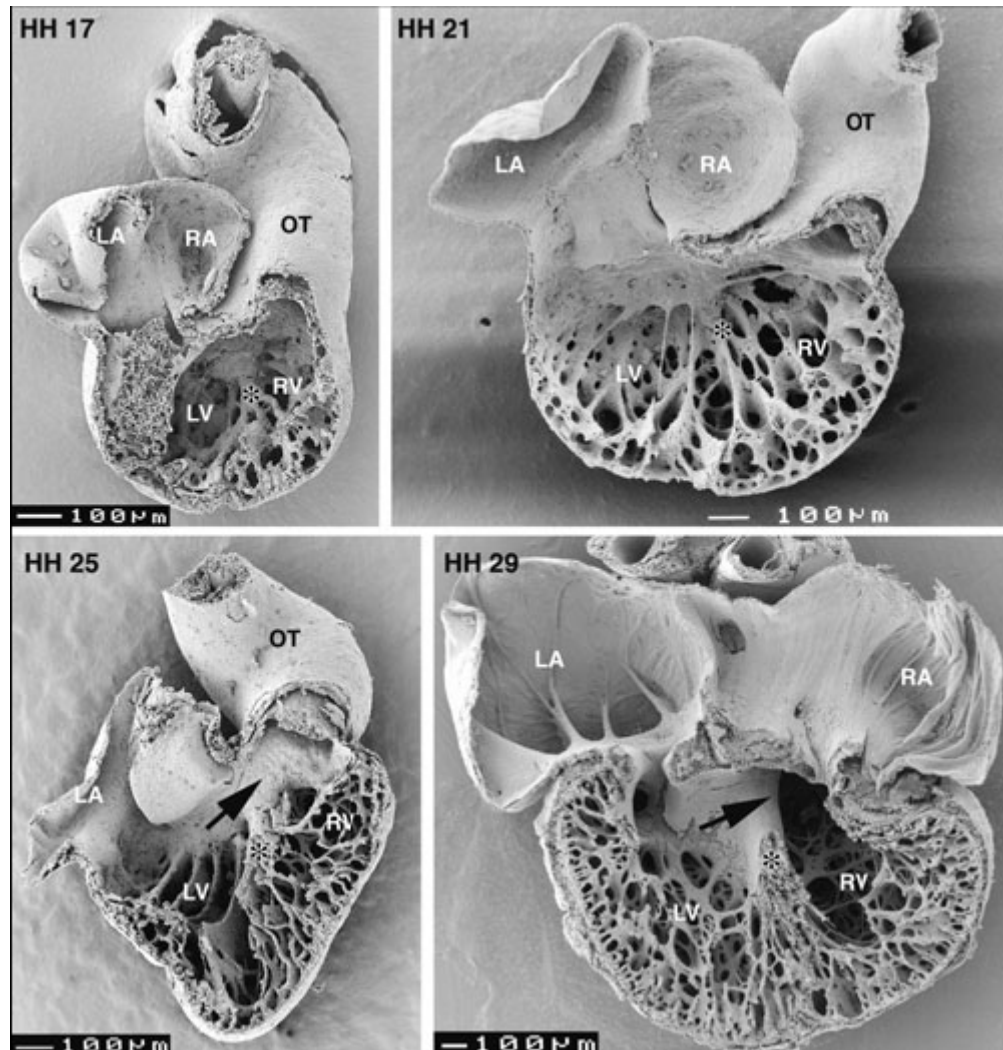


Figure: 1 Ventricular morphology in scanning electron micrographs of chick embryos during chamber formation. Ventral halves of frontally dissected stage 17, 21, 25, and 29 hearts viewed from the back. Arrows indicate the primary interventricular foramen. LA, left atrium; RA, right atrium; LV, left ventricle; RV, right ventricle; OT outflow tract; asterisk, crest of developing muscular interventricular septum. [16]