Magnetic Resonance Imaging Role in Evaluation Of Cardiac Function of Tetralogy Of Fallot

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Abstract

Background: Survivors of tetralogy of Fallot (TOF) repair constitute a large and growing population of patients. Although postsurgical outcome is generally favorable, as these patients move into adulthood, late morbidity is becoming more prevalent and the notion that TOF has been "definitively repaired" is increasingly being challenged. Recent evidence suggests that adverse long-term postsurgical outcome is related to chronic pulmonary regurgitation, right ventricular dilatation, and deteriorating ventricular function. Cardiac magnetic resonance (MR) imaging has been established as an accurate technique for quantifying ventricular size, ejection fraction, and valvular regurgitation. Cardiac MR imaging does not expose the patient to ionizing radiation and is therefore ideal for serial postsurgical follow-up. Familiarity with the anatomic basis of TOF, the surgical approaches to repair, and postrepair sequelae is essential for performing and interpreting cardiac MR imaging examinations. For example, awareness of the complications and sequelae that can occur will assist in determining when to intervene to preserve ventricular function and will improve long-term outcome. Technical facility is necessary to tailor the examination to the individual patient (eg, familiarity with non-breath-hold modifications that allow evaluation of young and less compliant patients). The radiologist can play an essential role in the treatment of patients with repaired TOF by providing noninvasive anatomic and physiologic cardiac MR imaging data. Further technologic advances in cardiac MR imaging are likely to bring about new applications, better normative data, and more examinations that are operator independent.

Keywords: Magnetic Resonance Imaging, Tetralogy Of Fallot

Introduction

The first anatomical description of a case of Fallot's tetralogy was made by **Stensen (1)**. Others described some of features of the anomaly before Fallot's description. It was in 1888 when Fallot described the four anatomic findings as one entity. However the diagnosis of tetralogy of Fallot was not made with any consistency until 1930 **(2).**

Anatomy of Fallot's tetralogy

"Arthur Louis Etienne Fallot" French scientist describe Tetralogy of Fallot (TOF) In his own words, "la maladie bleue," he described four anatomical traits **(3)** consist of a tetrad of:

1. **Ventricular septal defect (VSD)** This condition is usually a large non-restrictive defect (i.e., no obstruction to flow across the VSD). It is often located in the perimembranous and muscular regions of the ventricular septum, allowing shunting of blood between the ventricles **(4).**

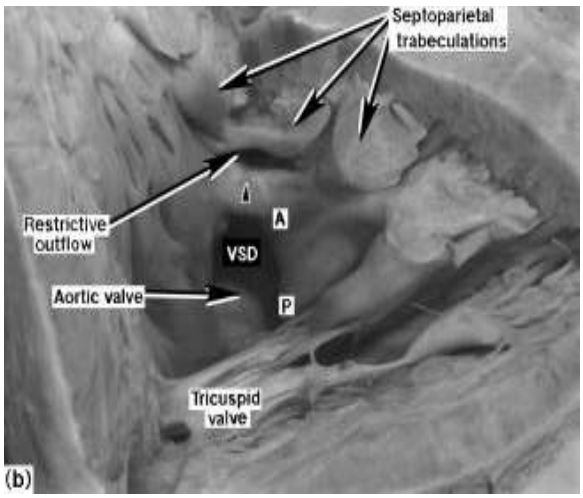

Fig. 1 : The right ventricle of a heart with tetralogy of Fallot . resembling subcostal right anterior oblique echocardiographic views, The overriding of the aortic valve through the VSD is evident.The septal insertion of the ventriculo-infundibular fold is attenuated (*), leaving an area of fibrous continuity between aortic, triscupid and mitral
valves. The deviated outlet septum (-), cut in profile, narrows the pulmonary outflow tract. and a specimen with tetralogy of Fallot **(2).**

Aortic overriding: When the LV supplies more than 50% of the aorta, the manner of ventriculo-arterial connection may be concordant, or when the RV supports more than 50% of the aorta, a double-outlet ventricular connection may emerge. This is the TOF DORV variant. **(5).**

2. **Right ventricular outflow tract (RVOT) obstruction:** Hypertrophy of the infundibular septum and anterior muscle bundles contributes to pulmonary outlet obstruction in tetralogy of Fallot. Hypoplasia of the pulmonary valve and main pulmonary artery is common. RVOT can be classified into sub-valvular, valvular, and supra-valvular levels of obstruction (**5).**

The caliber of the pulmonary valvular orifice was judged in comparison to that of the aortic orifice, to develop designation of the degree of pulmonary valvular stenosis based upon comparison of diameters **(**6**)**

1- Normal: The diameters of the pulmonary valve orifice was greater than 80% of the aortic orifice.
2- Mild to moderate stenosis: The pulmonary valve orifice was greater than 50% and less than 80% of the aortic orifice.
3- Severe stenosis: The pulmonary valvular orifice was less than 30% of the aortic orifice.
4- Pulmonary atresia: There was no communication between the infundibulum of the right ventricle and the pulmonary trunk.

Chacko, et al. (6) judged the degree of pulmonary artery narrowing by comparing its diameter to that of the aorta as follows:

1- Normal: The diameter of the pulmonary trunk was greater than 80% of that of the aorta.
2- Moderate hypoplasia: The diameter of the pulmonary trunk was between 50 and 80 percent of that of the aorta.
3- Severe hypoplasia: The diameter of pulmonary trunk was less than 50% of that of the aorta
4- Atresia: There is no lumen in the pulmonary trunk which represented by a fibrous like strand of the tissue.
5- Poststenotic dilatation: The relative size of the pulmonary trunk was disproportionately large compared to the orifice of the pulmonary valve.

4. Right ventricular (RV) hypertrophy: RV hypertrophy develops as a consequence of the RVOTO because increased RV pressure needs to be generated to maintain pulmonary blood flow. This condition also alters the RV cavity size and muscle mass, which are important issues after TOF repair **(7)**.

Nomenclature and classification of tetralogy of Fallout (8):

Four diagnostic subgroups of TOF exist:
1- TOF, absent pulmonary valve syndrome.
2- TOF, common atrioventricular canal (AVSD).
3- TOF, pulmonary atresia.

4- TOF, pulmonary stenosis.
5- TOF , A patent foramen oval "PFO" .
*TOF, absent pulmonary valve syndrome is a form of TOF with a severely dysplastic pulmonary valve and markedly dilated pulmonary arteries. represents only 3% to 5% of all cases of TOF. TOF with absent pulmonary valve is commonly associated with respiratory difficulties.
*TOF, common atrioventricular canal (AVSD) is the presence of both TOF and complete Atrioventricular Septal Defect (AVSD). represents only 2% of all cases of TOF. Complete surgical repair of this lesion is associated with more risk than either repair of TOF.
*TOF, pulmonary atresia is a form of pulmonary atresia with VSD in which the intracardiac anatomy is TOF. TOF with pulmonary atresia is commonly associated with hypoplastic branch pulmonary arteries and may be associated with Major Aorto-Pulmonary Collateral Arteries (MAPCAs).
*TOF, pulmonary stenosis is the commonest "garden variety" form of TOF. TOF with Pulmonary Stenosis (PS) will be the focus of this manuscript. In TOF with PS, the PS may be at the subvalvar, valvar, or supravalvar level or the stenosis may involve any possible combination of these three levels.(**8**)
*****Pentology of Fallot: A patent foramen ovale "PFO" or an ostium secondum ASD is a common association with TOF and is known as pentalogy of Fallot **(5).**

Diagnosis of Fallot tetralogy

To reach proper diagnosis of TOF, two items which complements each other (clinical picture and investigations), must fulfill the criteria within the diagnosis.

-Clinical picture:

The majority of cases of TOF are diagnosed on antenatal foetal ultrasound scanning. This procedure allows an appropriate counselling of parents, delivery in a cardiac centre and planning of postnatal care(**9).**

The diagnosis may be made after birth with the time of presentation depending on the severity of the TOF. Many neonates will be detechted by routine pulse oximetry screening, or the presence of a muermur on a neonatal examination. The classic presentation is of a cyanotic neonate or young infant, who has no features of respiratory distress, fails to respond to oxygen therapy and has signs of a good cardiac output. (**10).**

-Investigations:

***Blood gases:**

The pH and PCO_2 are normal at rest .The PO_2 is variable depending upon the severity of the pulmonary stenosis. Arterial oxygen saturation falls, sometimes dramatically with physical activity. **(11)**.

***Hematologic studies:**

The hemoglobin, hematocrit and erythrocyte count is usually elevated. The magnitude of the increase is generally proportional to the cyanosis with hematocrit values varying from normal to as high as 90%, the majority being between 50% and 70%, very high levels of hemoglobin and hematocrit have serious consequences. Blood viscosity rises strikingly with with hematocrit levels greater than 60% . **(12)**.
Polycythemia also alters blood coagulation and deficiency of one or more coagulation factors becomes very common if the hematocrit exceeds 65%. The specific factors involved and the severity of the hemostatic abnormality vary from patient to patient. The platelet count and total blood fibrinogen are frequently slightly diminished **(12)**.

***Radiologic features:**

Cardiac imaging has rapidly evolved, from relying exclusively on cardiac catheterization and chest radiography to today's more advanced imaging techniques: echocardiography, cardiac magnetic resonance and cardiovascular computed tomography. Becouse of the complex nature and diversity of these population, as well as the limitations of each modality, it is increasingly recognized that an expert multidisciplinary team with a multimodality imaging approach is necessary for their clinical management. **(13)**

-X ray:

Findings: On radiographs, the cardiac silhouette is normal in size; however, right ventricular hypertrophy can elevate the left ventricle. Together with a small or absent main pulmonary artery segment, the heart can have the classic boot-shaped appeara**n**ce. Typically, the vascularity of the pulmonary artery is reduced. Large collaterals may give the appearance of normal vascularity **(14)**.

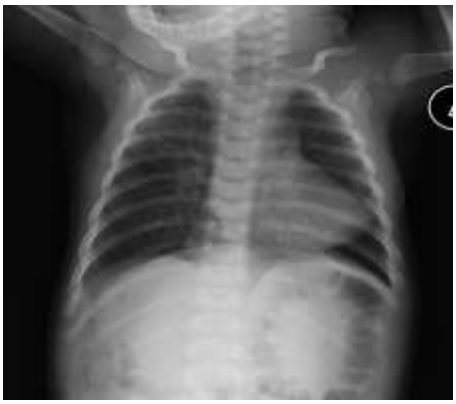

Fig 2. Tetralogy of fallout by x ray. **(14)**.

-CT:

CT infrequently has a role in the evaluation of tetralogy of Fallot. CT is useful for the evaluation of surgical complications such as infection or pseudo aneurysm formation. Helical CT can be used to identify airway compression caused by a large ascending aorta associated with tetralogy of Fallot **(15)**.

MRI:

Findings:

Spin-echo MRI can be used to identify the morphologic abnormalities of tetralogy of Fallot, which are as follows: right ventricular outflow tract obstruction, ventricular septal defect, right ventricular hypertrophy, and overriding aorta **(2).**

The confluence, presence, and size of the branch pulmonary arteries can be identified. MRI measurements are as accurate as angiographic measurements, and they can be used to calculate the McGoon ratio and Nakata index Postoperative evaluation of pulmonary artery stenosis is better with MRI than with echocardiography. Cine imaging can be used to identify pulmonary stenosis or regurgitation, which is depicted as flow voids. Right ventricular enlargement is best quantified with MRI. Flow analysis quantification of pulmonary regurgitation is unique to MRI. Although gradients can be measured with echocardiography, only MR flow analysis enables accurate cross-sectional measurement of flow **(16)**.

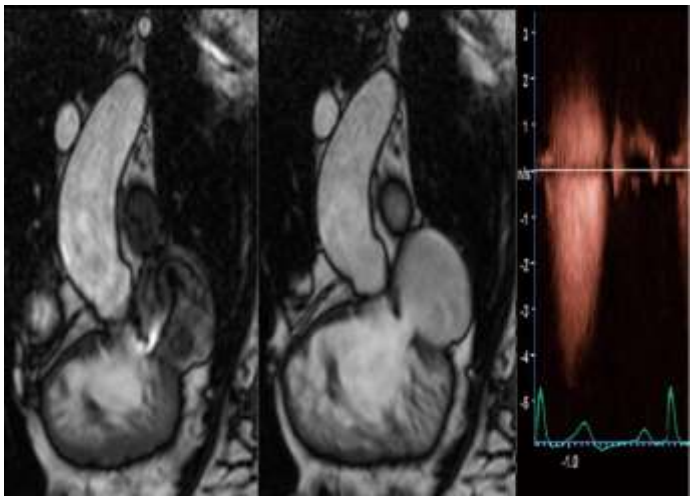

Figure 3 CMR cine images depicting a patient with PS in systole (left) and diastole (center). The doppler curve (right), with a maximum velocity close to 5 m/s, resembles an aortic stenosis curve, and corresponds to severe PS. CMR, cardiac magnetic resonance; PS, pulmonary stenosis. **(16)**.

-Electrocardiographic features:

The common findings on the ECG are right axis deviation and right ventricular hypertrophy. There is usually a dominant R wave in the right precordial leads and a dominant S in the left leads, but the R and S may be of similar height in V_6 or there may be a dominant R wave. T waves may be upright or inverted in V_1-V_3 **(11)**.

Treatment strategy

-Medical management:

Initial medical management is aimed at relieving hypoxemia and preventing cyanotic spells. A progressive decrease in systemic saturation usually is associated with fixed right ventricular outflow tract obstruction and does not respond to medical intervention. **(17).**

- Surgical repair and its complication:

***Decision making and timing for repair of Fallot tetralogy:**

Although the results of complete repair of TOF and pulmonary stenosis have improved greatly, occasional deaths still occur the early post-operative period with some patients. But without surgical treatment very young patients with TOF in general are at greater risk of dying within 1 to 2 years than are older patients **.**The risk is considerably greater in those having severe cyanosis and syncopal attacks. At present date very young age (less than 3 months) remains in general a risk factor for death after repair, however when the RVOT was favorable for repair, young age carried only a small predicted risk which is probably lesser than those of natural history **(18)**.

***The goals of surgical therapy are to:**

(1) Close intracardiac shunts,

(2) Provide relatively unobstructed pulmonary blood flow,

(3) Maintain normal function of the right ventricle,

(4) Maintain normal function of the pulmonary and tricuspid valve, and

(5) Maintain normal cardiac rhythm with minimal cumulative morbidity or mortality)(**19**)

***Palliative Surgery:**

Palliative systemic to pulmonary shunts are used to improve pulmonary blood flow and hence encourage pulmonary artery growth in individuals who have tiny or hypoplastic pulmonary arteries. A palliative shunt used to be the therapy of choice, but as the results of early corrective surgery have improved, it is now rarely performed. **(20):**
However, certain patients may benefit from a palliative shunt in the first weeks or months of life:

- Newborns with duct dependent pulmonary circulation (pulmonary atresia)
- Marked RVOTO and cyanosis
- Very small pulmonary arteries
- Anomalous coronary artery anatomy

Types:

1-Blalock-Taussig shunt (classical): Subclavian artery-to-pulmonary artery anastomosis (end-to-side). Infrequently, this may lead to pulmonary hypertension. **(21)**

2-Blalock-Taussig shunt (modified): The modified Blalock Taussig (mBT) shunt: A 3-4 mm Gortex is placed between one of the arch arteries and the right or left pulmonary artery, depending on the position of the aortic arch and the structure of the pulmonary arteries. The pulmonary arteries might be continuous or discontinuous (confluent/non-confluent), or one of them can be larger than the other. A palliative mBT shunt's flow may foster the growth of tiny pulmonary arteries. Depending on the surgical needs, surgery may be conducted by a thoracotomy or sternotomy. **(5).**

3-Waterston shunt: Ascending aorta-to-main or right pulmonary artery (side-by-side). No artificial material used; shunt grows with the patient. May lead to pulmonary hypertension. Infrequently, problems have been encountered with pulmonary artery disruption, requiring extensive arterioplasty. (**22)**

4-Potts shunt: is a side-to-side anastomosis is created from the left pulmonary artery to the descending aorta, resulting in a right-to-left shunt. (**22)**

5-Glenn operation

A-The classic (unidirectional) Glenn shunt involved:

- ligation of the distal end of the superior vena cava (SVC)
- Anastomosis of the side of the SVC above the ligation with the distal end of the divided right pulmonary artery.

B-Modified Glenn or hemi-Fontan shunt, anastomosis is created between the transected end of the SVC and the side of the undivided right pulmonary artery.

6-The Fontan procedure is basically a bidirectional Glenn shunt combined with a conduit or tunnel through or around the right atrium, shunting blood from the inferior vena cava to the pulmonary arteries

7-Some centres perform transcatheter interventions such as ballooning or stenting of the RVOT, PDA, or branch pulmonary arteries in place of a mBT shunt prior to definitive surgery, but this is not common practice.

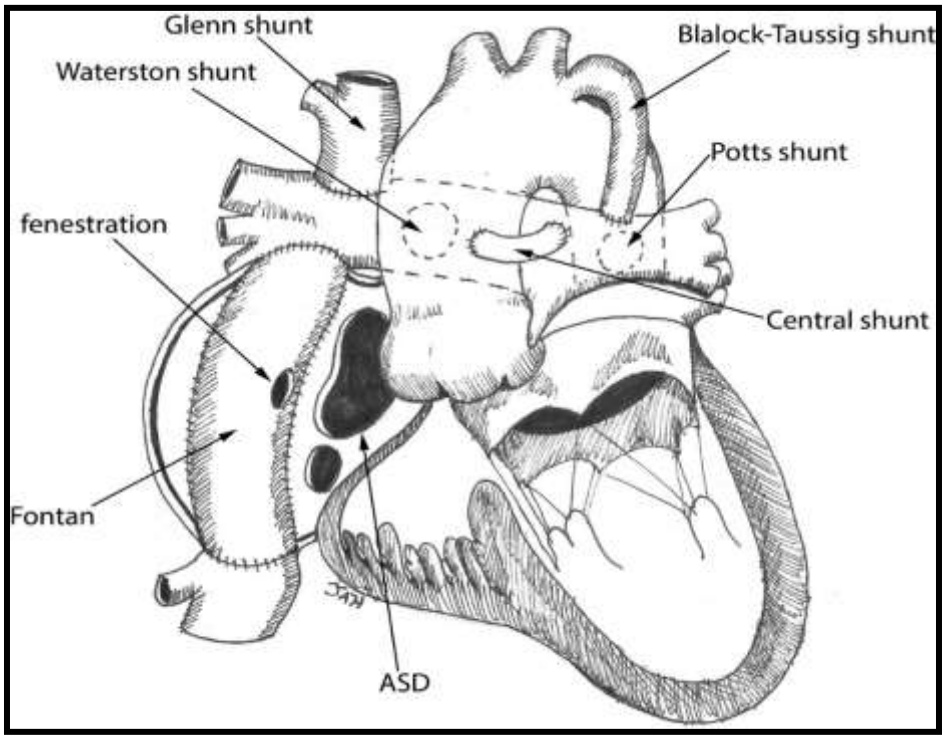

Fig. (4): Diagram shows different types of palliative shunts. **(5).**

Complete surgical repair of TOF:
Consists of closure of the VSD and relief of the RVOTO via median sternotomy on cardiopulmonary bypass. It is best done in the first year of lifes.
A trans-atrial approach is taken to repair the VSD using a bovine pericardial patch. The RVOTO's is reduced by:
=Muscle bundle resection in the pulmonary infundibulum by a transatrial approach ,RVOT patch, leaving the pulmonary annulus intact
=Transannular patch across the pulmonary outflow tract
=Pulmonary valvotomy or valvectomy
=Pulmonary conduit or homograft from the right ventricle to pulmonary artery may be required in children with pulmonary atresia or coronary abnormalities.
The long-term complications including :
1-PR and RV Dilatation
One of the most prevalent complications of TOF surgery is a recurrence of PR due to RV hypertrophy, PR is well tolerated in the short term, but it eventually leads to RV dilatation and right heart failure.(**23**).
2-Conduit stenosis.
3-RVOT Aneurysm:
An RVOT aneurysm (dyskinesia) is described as the movement of a portion of the ventricular wall or its repaired outflow channel outward during systole. **(24)**
4-Recurrent VSD:
Is an important finding that may require intervention despite its rarity. **(25).**
5-Residual pulmonary artery stenosis (PAS):
In roughly 10%–15% of individuals who have undergone TOF repair, residual or recurrent branch PAS occurs **(20).** Stenosis of the peripheral or central PAs is possible. Furthermore, branch PAS can occur as a result of previous palliative Blalock-Taussig shunt surgery, due to scarring at the anastomotic site or postoperative PA distortion and kinking.

-Pulmonary Regurgitation After TOF Repair

The severity of PR can increase over time , The degree of PR is determined by (1) regurgitation orifice area; (2) RV compliance; (3) diastolic pressure difference between the main pulmonary artery (MPA) and the RV; (4) capacitance of the pulmonary arteries; and (5) duration of diastole. Some of the key factors influencing PR volume are captured by the Torricelli principle (**26):**

A growing clinical experience and experimental evidence point to striking similarities between the pathophysiologic response of the left and right ventricles to severe chronic volume load. **(27)**

RV Mechanics After TOF Repair

After TOF repair, additional factors related to the operation further impact the pathophysiology. Relief of RV outflow obstruction typically involves incision of the infundibular free wall, resection of obstructive muscle bundles, disruption of the pulmonary valve with partial or complete excision, and placement of an outflow patch, which often extends across the plane of the pulmonary valve into the MPA. In some patients a conduit between the RV and the pulmonary arteries is required to provide antegrade pulmonary blood flow. The ventricular septal defect is closed with a patch, a procedure that can impair tricuspid valve function. In addition to the nearly universal occurrence of PR (discussed above), these procedures often lead to akinesis or dyskinesis of the RVOT, outflow patch aneurysm fibrosis of the RV free wall, and conduction delay .Human studies in repaired TOF patients using CMR provide insights regarding ventricular mechanics and clinical outcomes in this population . Several authors have demonstrated a close relationship between the degree of PR and RV diastolic dimensions and stroke volume . Similar to LV function in severe chronic aortic regurgitation, once the compensatory mechanisms of the RV fail, mass-to-volume ratio decreases, end-systolic volume increases, and ejection fraction decreases (**28)**

RV-LV Interaction After TOF Repair

The right and left ventricles function in series and, in the absence of shunts, have similar net outputs. In 1910 the French physiologist Bernheim first recognized interdependence between LV and RV function. He postulated that alterations in the size and function of the LV adversely impact the geometry and function of the RV, a phenomenon termed 'Bernheim effect' . Many subsequent studies have demonstrated that alterations in the size and function of the RV lead to LV dysfunction.(**29).**

=Treatment Strategies Late After Tof Repair:

***Indications for Pulmonary Valve Replacement (30).**

Indications for PVR in patients with repaired TOF or similar physiology with moderate or severe pulmonary regurgitation (regurgitation fraction ≥25%):

I Asymptomatic patient with two or more of the following criteria

- RV end-diastolic volume index >150 ml/m^2 or Z-score >4. In patients whose body surface area falls outside published normal data: RV/LV end-diastolic volume ratio >2
- RV end-systolic volume index >80 ml/m^2
- RV ejection fraction <47%
- LV ejection fraction <55%
- Large RVOT aneurysm
-QRS duration >140 ms
- Sustained tachyarrhythmia related to right heart volume load
- **Other hemodynamically significant abnormalities:**

○RVOT obstruction with RV systolic pressure ≥2/3 systemic
○ Severe branch pulmonary artery stenosis (<30% flow to affected lung) not amenable to transcatheter therapy
○ ≥ Moderate tricuspid regurgitation
○ Left-to-right shunt from residual atrial or ventricular septal defects with pulmonary-to-systemic flow ratio ≥ 1.5
○ Severe aortic regurgitation
○ Severe aortic dilatation (diameter ≥5 cm)

II. Symptomatic Patients

Symptoms and signs attributable to severe RV volume load documented by CMR or alternative imaging modality, fulfilling ≥1 of the quantitative criteria detailed above. Examples of symptoms and signs include:

- .Exercise intolerance not explained by extra-cardiac causes (e.g., lung disease, musculoskeletal anomalies, genetic anomalies, obesity), with documentation by exercise testing with metabolic cart (≤70% predicted peak VO_2 for age and gender not explained by chronotropic incompetence)
- Signs and symptoms of heart failure (e.g., dyspnea with mild effort or at rest not explained by extra-cardiac causes, peripheral edema)

- Syncope attributable to arrhythmia

III. Special considerations

a. Due to higher risk of adverse clinical outcomes in patients who underwent TOF repair at age ≥3 years , PVR may be considered if fulfill ≥1 of the quantitative criteria in section I

b. Women with severe PR and RV dilatation and/or dysfunction may be at risk for pregnancy-related complications . (**30).**

***Timing of Pulmonary Valve Replacement**

There is strong evidence that without intervention severe PR in repaired TOF leads to severe RV dilatation and dysfunction, tricuspid valve regurgitation, LV dysfunction, tachyarrhythmias, diminished exercise tolerance, heart failure symptoms, and death . PVR leads to marked reduction of PR, improved biventricular mechanics, improvement in tricuspid regurgitation, and symptomatic improvement. (**(30).**

Recent evidence clearly demonstrates that relying on symptoms as the major criteria for PVR results in patients receiving a pulmonary valve when their RV is markedly dilated (mean RV end-diastolic volume 201 ± 37 ml/m^2 With an average preoperative RV end-diastolic volume of 142 ± 43 ml/m^2, RV end-systolic volume of 91 ± 18 ml/m^2, and RV ejection fraction 47 ± 8%, RV size and function was, on average, within normal limits one year after PVR. The authors, therefore, recommend PVR before RV end-diastolic volume exceeds 150 ml/m^2. We analyzed pre-PVR predictors of normal post-PVR RV size (end-diastolic volume index ≤114 ml/m^2) and function (ejection fraction ≥48%) in 64 patients with severe chronic PR**(30).**

Thus, the timing and indications for PVR after TOF repair must balance the benefits of elimination of RV volume load before irreversible dysfunction occurs. **(30).**

***Benefits of Pulmonary Valve Replacement**

Clinically, many patients report less cardiac symptoms after PVR and several studies have reported a significant improvement in NYHA functional class , Within approximately one year after PVR RV end-diastolic and end-systolic volumes decrease by 30-40% as compared with their preoperative values **(30).**

LV end-diastolic volume increases slightly whereas global LV systolic function remains unchanged . The degree of tricuspid valve regurgitation tends to improve with or without concomitant tricuspid valve surgery. **(30).**

***Risks of Pulmonary Valve Replacement**

The operative mortality for surgical PVR is low . There is, however, continued low risk of death after PVR. (**31).**

When considering the risks associated with PVR, the risk of valve failure should also be considered. All valves inserted in the pulmonary position have a limited life expectancy, with wide variations in rates of freedom from valve failure and reoperation, depending of the type of valve and patient age.

The advent of transcatheter pulmonary valve implantation ,however, may provide a new non-surgical option for the treatment of failed bioprosthetic pulmonary valve. Although the currently available catheter-delivered, stent-mounted valves are limited by the size and geometry of the RVOT to mostly patients with RV-to-pulmonary artery conduits, future developments will likely expand the clinical application of this technique to patients with dilated RVOT. Even with currently available technology, catheter-based pulmonary valve implantation can be performed inside a failing bioprosthetic valve or conduit **(32).**

=Table 1 Structural and functional abnormalities encountered in repaired TOF (**33)**

Structural Abnormalities	**Functional Abnormalities**
Inherent to TOF repair	RV volume overload
Partial or complete removal of pulmonary valve tissue	Pulmonary regurgitation
Infundibulotomy scar	Tricuspid regurgitation
Resection of RV/infundibular muscle bundles	Left-to-right shunt
Right atriotomy scar	Ventricular septal defect
VSD patch	Atrial septal defect
Residual or recurrent lesions	Aorto-pulmonary collaterals
RV outflow tract obstruction	RV pressure overload

Structural Abnormalities	Functional Abnormalities
Main or branch pulmonary artery stenosis	RV outflow or pulmonary artery stenosis
Ventricular septal defect	Pulmonary vascular disease
Atrial septal defect	Pulmonary venous hypertension secondary to LV dysfunction
Acquired lesions	RV systolic dysfunction
Tricuspid valve abnormalities	RV diastolic dysfunction
RV outflow tract aneurysm	LV dysfunction
RV fibrosis	Ventricular conduction delay
Associated anomalies	Arrhythmias
Dilated aorta	Atrial flutter
Associated congenital cardiovascular anomalies	Atrial fibrillation
Associated genetic and non-cardiac anomalies	Ventricular tachycardia
	Co-morbidities
	Renal, pulmonary, musculoskeletal, neurodevelopmental abnormalities

***Goals of CMR in patients with repaired TOF include (34)**

- Quantitative assessment of left and right ventricular volumes, mass, stroke volumes, and ejection fraction.
- Evaluation of regional wall motion abnormalities.
- Imaging the anatomy of the right ventricular outflow tract, pulmonary arteries, aorta, and aorto-pulmonary collaterals.
- Quantification of PR, tricuspid regurgitation, cardiac output, and pulmonary-to-systemic flow ratio.
- Assessment of myocardial viability with particular attention to scar tissue in the ventricular myocardium aside from sites of previous surgery (e.g., ventricular septal defect and RVOT patches).

Imaging protocol of repaired TOF:

Localizing images: ECG-gated steady-state free precession (SSFP) localizing imaging in the axial, coronal, and sagittal planes followed by real-time interactive sequences for identification of key imaging planes and structures targeted for additional sequences (e.g., ventricular long- and short-axis planes, short-axis of the proximal MPA for subsequent measurements of PR).

- **ECG-triggered, breath-hold cine SSFP** in the following planes:

▪ LV 2-chamber
▪ RV 2-chamber
▪ 4-chamber
▪ Ventricular short-axis .
▪ Oblique sagittal parallel to the RVOT and proximal MPA
▪ Parallel to the left ventricular outflow (LV 3-chamber view).
▪ Axial plane for imaging of the outflow tracts and branch pulmonary arteries (all first studies, optional thereafter).

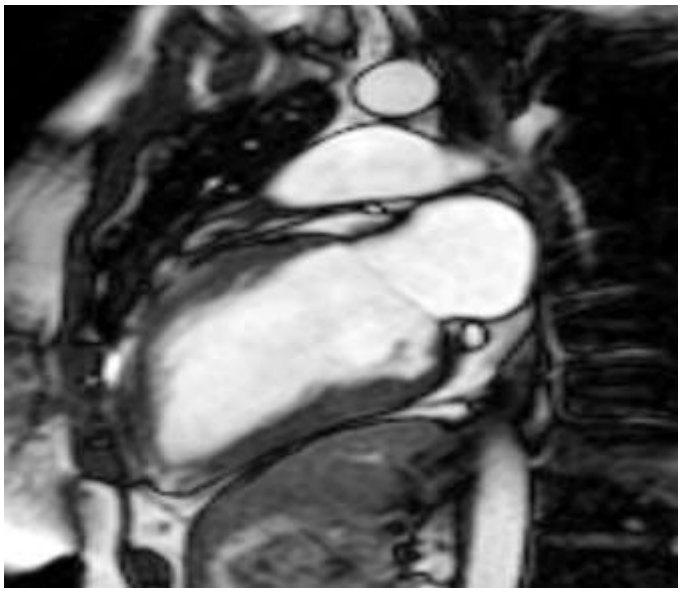

Figure (5) Evaluation of ventricular size and function by ECG-gated cine SSFP MR in repaired TOF: Left ventricular 2-chamber (vertical long-axis) plane. (34)

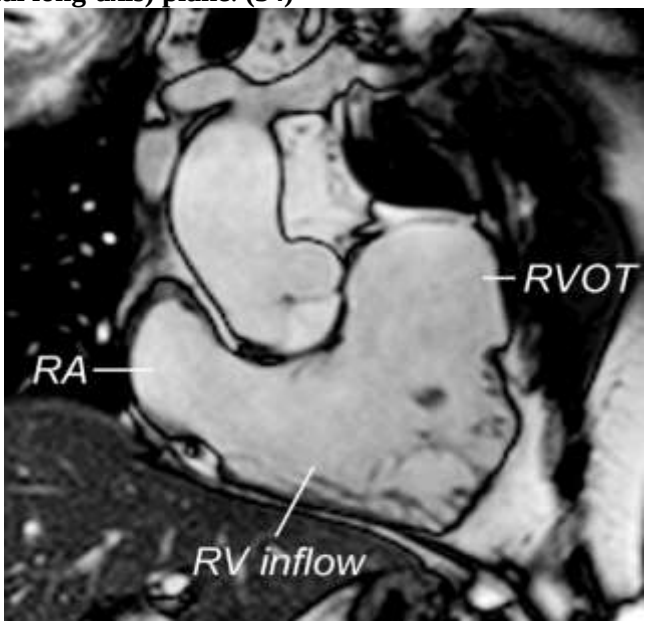

Figure (6) Evaluation of ventricular size and function by ECG-gated cine SSFP MR in repaired TOF: Right ventricular 2-chamber (vertical long-axis) plane. RA = right atrium; RV = right ventricle; RVOT = right ventricular outflow tract. (34)

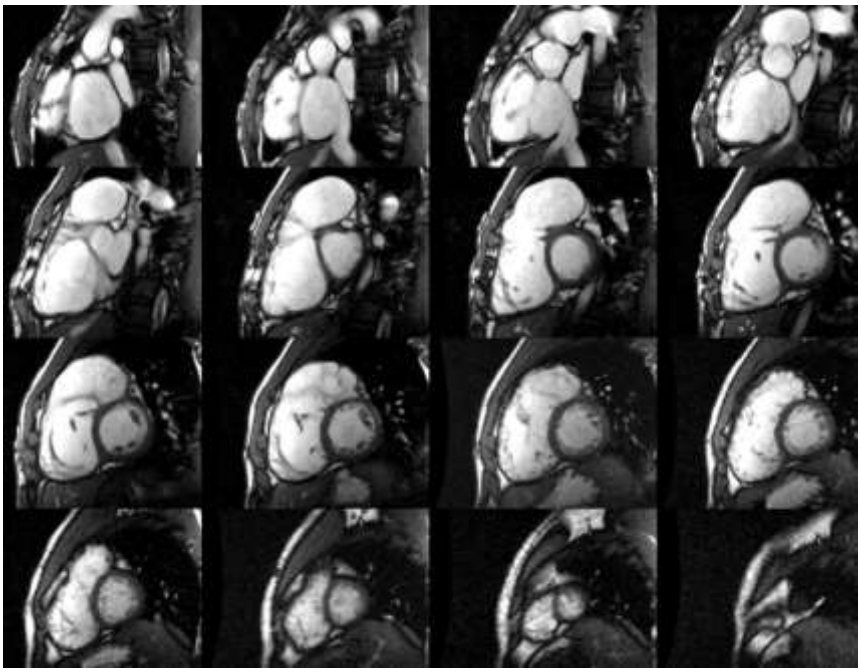

Figure (7)Evaluation of biventricular size and function by ECG-gated cine SSFP MR in repaired TOF: Ventricular short-axis. Note that 16 short-axis slices were required to fully cover the markedly dilated RV in this patient. (34)

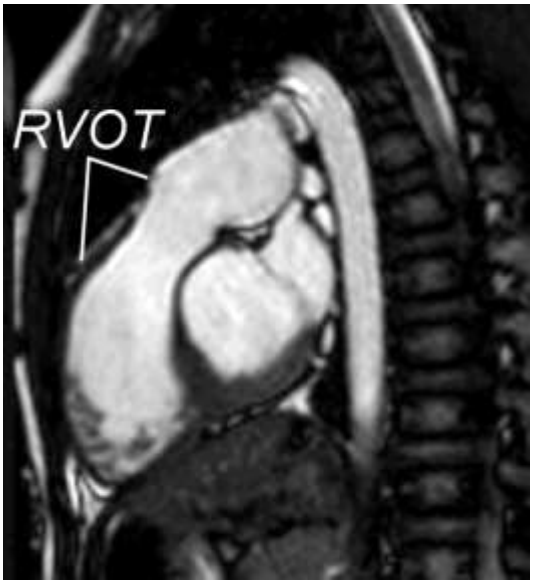

Figure (8) Evaluation of the right ventricular outflow tract (RVOT) long-axis by ECG-gated cine SSFP MR. Along with the RV 2-chamber plane, this view demonstrates patency of the RVOT and main pulmonary artery, presence or absence of pulmonary valve tissue, and wall motion abnormalities. (34)

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