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REVIEW

Tetralogy of Fallot: Multimodality Imaging and Key Historical Contributions to Diagnosis and Treatment

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ABSTRACT

Tetralogy of Fallot (TOF) is one of the most common cyanotic congenital heart malformations, characterized by four pathological features: right ventricular outflow tract obstruction/pulmonic stenosis, a ventricular septal defect, an overriding aorta, and right ventricular hypertrophy. It was initially partially defined by Nicholas Steno in the 17th century and completely described by Étienne-Louis Arthur Fallot and Maude Abbott in the 19th and 20th centuries. The advances in multimodality imaging and innovative surgical and transcatheter techniques have led to advances in the management of TOF. While initial management in the mid-20th century favored palliative procedures in infancy followed by complete surgical repair, data now support an early complete surgical repair in infancy. The major post-repair complication is the development of significant pulmonary regurgitation, necessitating either surgical or transcatheter valve replacement. Multimodality imaging is essential to the initial identification of TOF, preoperative planning, and post-procedural complication assessment.

In this review, we provide a historical perspective of the discovery and clinical management of TOF from the 1600s to the present day, as well as the role of multimodality imaging in TOF management.

1 | Introduction

This review provides historical context for the discovery of tetralogy of Fallot (TOF), its clinical manifestations, and procedural advances (Table 1). Some of the cardinal features of the disease were characterized as far back as the 17th century by Nicholas Steno, whose work was later refined by Étienne-Louis Arthur Fallot and Maude Abbott.

The clinical management of TOF has evolved for the most part over the last century, from the invention of palliative surgical techniques in the mid-20th century to contemporary complete surgical repair in infancy. Multimodality imaging is key to understanding the anatomy and pathophysiology of TOF, initial surgical operative planning, surveillance, and eventual pulmonary valve replacement.

TOF is the most common cyanotic congenital heart defect, with a prevalence of 3–6 cases per 10,000 births, accounting for 10% of all congenital heart diseases (CHD) [1]. It occurs equally in males and females and across all races and ethnicities. TOF can be associated with several genetic conditions such as

Abbreviations: BTT, Blalock–Taussig–Thomas; CHD, congenital heart disease; CMR, cardiac magnetic resonance; CT, computerized tomography; PA, pulmonary artery; PR, pulmonary regurgitation; PS, pulmonary stenosis; PV, pulmonary valve; PVR, pulmonic valve replacement; RA, right atrium; RV, right ventricle; RVOT, right ventricular outflow tract; RVOTO, right ventricular outflow tract obstruction; TAP, transannular patch; TOF, tetralogy of Fallot; VSD, ventricular septal defect; VSR, valve-sparing repair; VT, ventricular tachycardia.

TABLE 1 | Outline of major historical events in TOF.

Year	Investigator	Description
1673	Nicolas Steno	Initial partial description of Tetralogy of Fallot (TOF)
1888	Étienne-Louis Arthur Fallot	Full description of TOF published in *Contribution à l'anatomie pathologique de la maladie bleue*
1924	Maude Abbott	Abbott named Fallot's findings "Tetralogy of Fallot"
1944	Alfred Blalock, Helen Taussig, Vivien Thomas	First description of palliative surgery: the Blalock-Thomas-Taussig (BTT) shunt
1954	C. Walton Lillehei and colleagues	First description of complete surgical repair
1970	Stella van Praagh	TOF is a single anomaly: failure of growth of the subpulmonary infundibulum
1976	Alan B. Gazzaniga and colleagues	First description of modified BTT shunt to preserve ipsilateral upper limb blood flow
2000	Philip Bonhoeffer and colleagues	First description of transcatheter pulmonary valve replacement

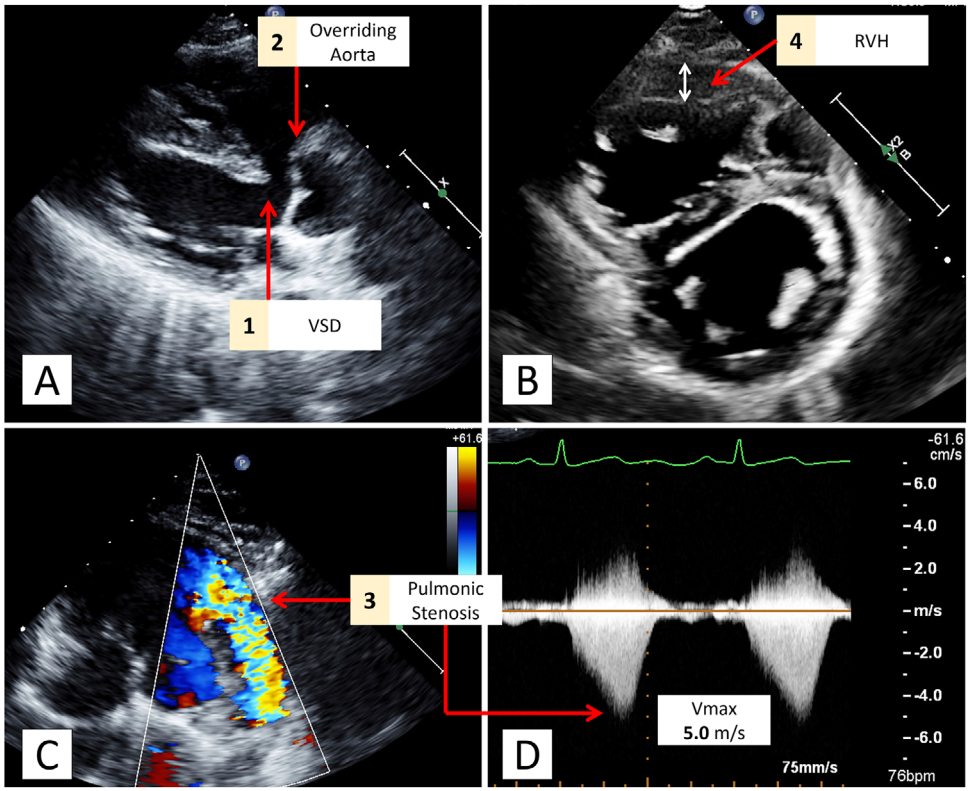


FIGURE 1 | Classic tetrad of unrepaired TOF on TTE Panel A: Parasternal long axis view. Arrow 1 points to a discontinuity in the interventricular septum at the subaortic region. Arrow 2 highlights the overriding of the ascending aorta on the ventricular septal defect. Panel B: Parasternal short-axis view. The double-headed arrow demonstrates increased thickness of the right ventricle due to right ventricular hypertrophy. Panel C: A color Doppler study of the right ventricle outflow highlights aliased velocity and formation of a proximal isovelocity convergence zone (arrow 3) at the level of the pulmonic valve, consistent with significant stenosis. Panel D: Spectral Doppler study of the right ventricular outflow tract at the same level as Panel C shows a peak velocity of roughly 5 m/s, confirming severe pulmonary stenosis. The increased density and late peaking shape of the jet are additional signs of severe obstruction. RVH, right ventricular hypertrophy; VSD, ventricular septal defect.

chromosomal abnormalities, including 22q11.2 deletions, trisomy 21 (Down syndrome), and other birth defects [1–3]. It is classically defined by the following tetrad: Right ventricular outflow tract obstruction (RVOTO) or pulmonic stenosis (PS), malaligned ventricular septal defect (VSD), an overriding aorta, and concentric right ventricular (RV) hypertrophy (Figure 1 and Video S1), (Figure 2 and Video S2), and (Figure 3) [4]. On chest x-ray, the right ventricular hypertrophy gives rise to a boot-shaped heart silhouette referred to in French as "coeur en sabot" (Figure 4, Panel A). In TOF, there is a high incidence (13% to 34%) of a right-sided aortic arch compared to only 0.1% in the general population [5] (Figure 4, Panel B).

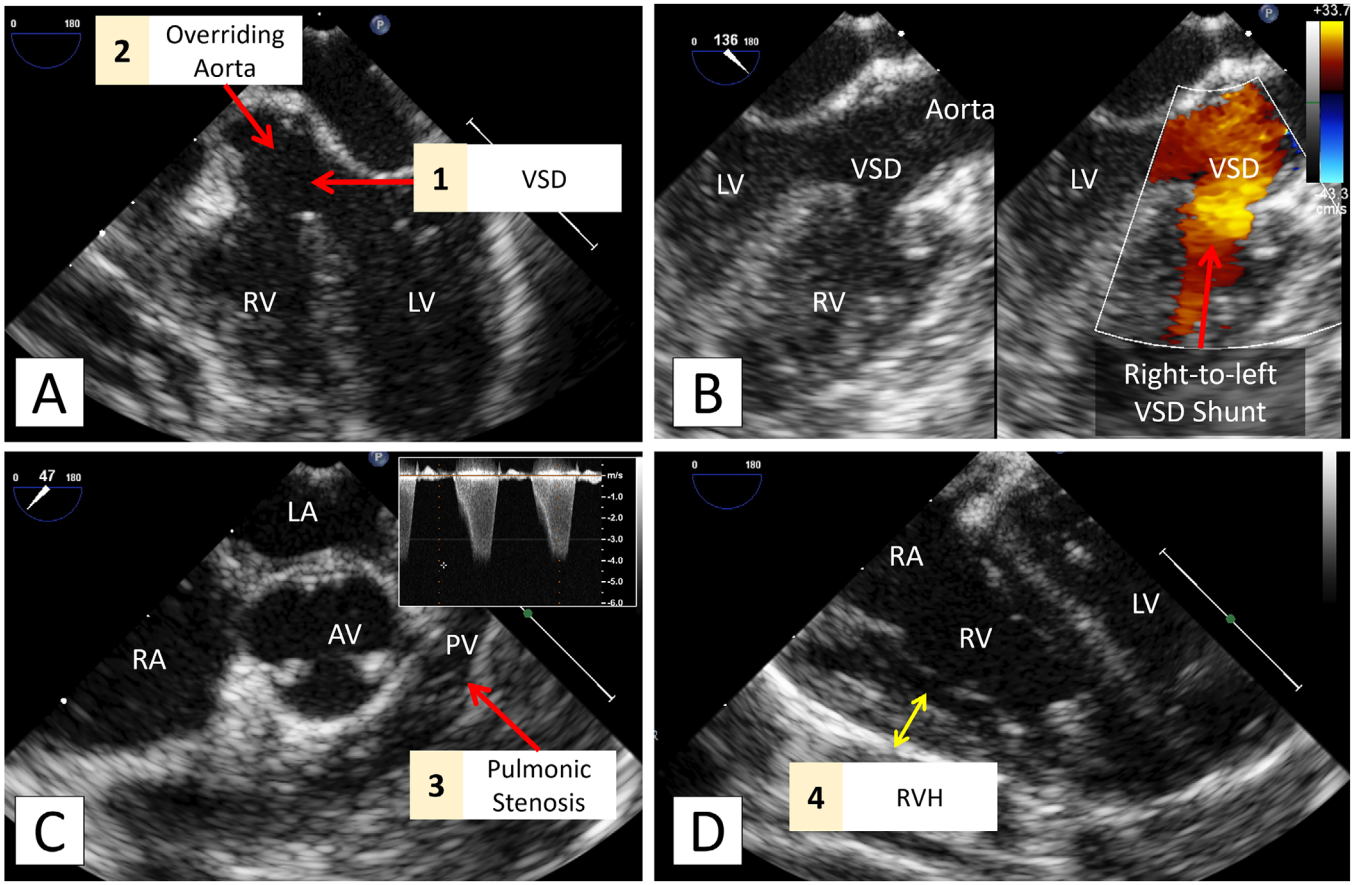


FIGURE 2 | Classic tetrad of unrepaired TOF on TEE Panel A: Mid-esophageal five-chamber view shows the VSD (arrow 1) and overriding of the aorta (arrow 2). Panel B: Modified mid-esophageal views with and without color Doppler demonstrate right-to-left shunt through the VSD. Panel C: Midesophageal short-axis view of the aortic valve. The pulmonic valve leaflets (arrow) are thickened. Spectral Doppler study of the pulmonic valve shows a peak velocity of roughly 4.5 m/s, consistent with severe stenosis. Panel D: Modified view of the right ventricle highlights increased thickness of the RV free wall (double-headed arrow) due to RV hypertrophy. AV, aortic valve; LA, left atrium; LV, left ventricle; PV, pulmonic valve; RA, right atrium; RV, right ventricle; RVH, right ventricular hypertrophy; VSD, ventricular septal defect.

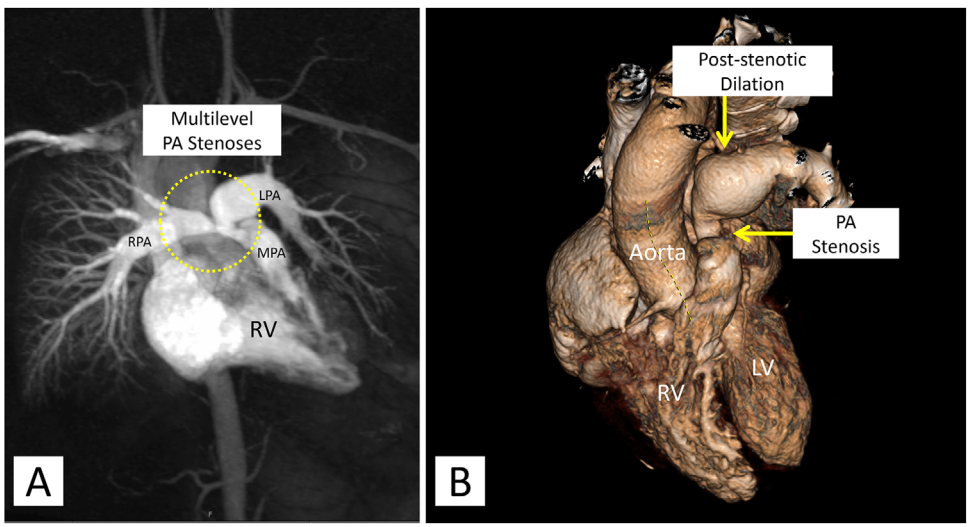


FIGURE 3 | Multilevel pulmonic stenosis Panel A: Contrast-enhanced chest CT shows significant stenosis of the pulmonary valve and origin of the left pulmonary artery (dashed circle). Panel B: Posterior sagittal 3D reformatted image from the same exam shows pulmonary artery stenosis and post-stenotic dilation of the left pulmonary artery. LPA, left pulmonary artery; LV, left ventricle; MPA, main pulmonary artery; RPA, right pulmonary artery; RV, right ventricle.

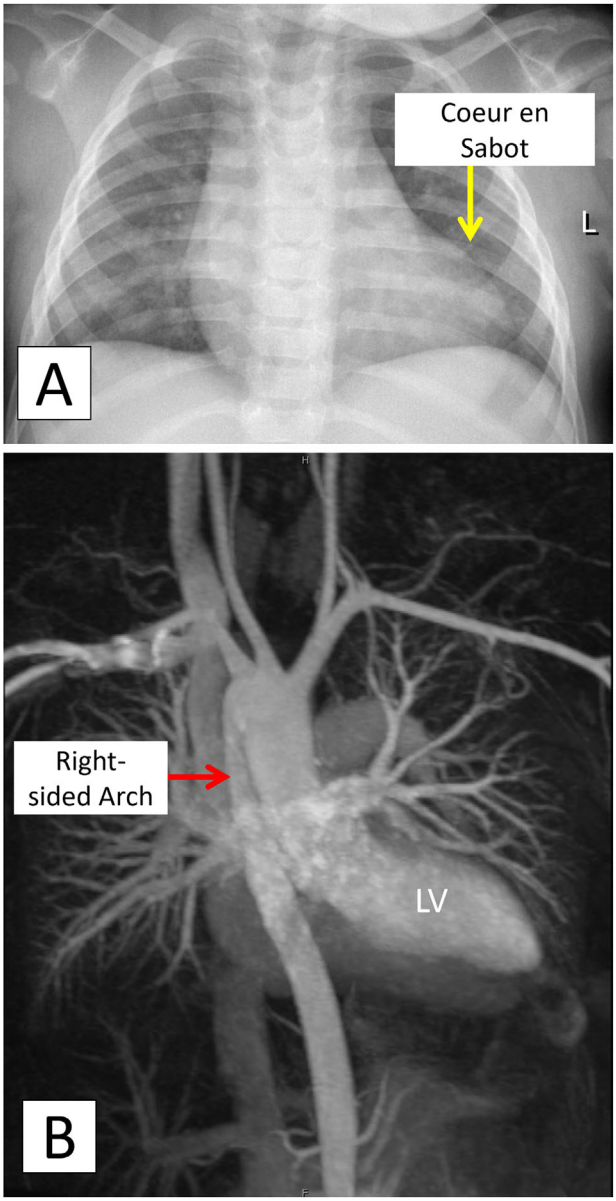


FIGURE 4 | "Coeur en Sabot" & Right-Sided Aortic Arch in TOF Panel A: AP chest radiograph in a TOF patient shows a less convex or concave upper right heart border due to RV outflow obstruction. RV hypertrophy leads to a rounded upward apex. These findings resemble a boot, historically referred to as "Coeur en Sabot." Panel B: Contrast-enhanced axial chest CT in a TOF patient shows a right-sided aortic arch (arrow). LV, left ventricle

Although typically diagnosed early in life, TOF pathology exists on a clinical spectrum, with the degree of symptoms depending on the interplay between the degree of right-to-left shunting across the VSD (leading to cyanosis) and the degree of RVOT/pulmonic stenosis (which may protect against pulmonary bed overcirculation). In the absence of surgical interventions, TOF leads to cyanotic spells and a short life span, typically ending in childhood. Initially, TOF was treated with the creation of a palliative shunt followed by eventual complete surgical repair. However, with advancements in surgical techniques, TOF is now typically treated with complete repair in infancy [6–9] without prior palliative shunts. This surgery has a very low mortality rate, and with regular post-surgical follow-up, the quality of life is greatly improved.

A major complication of surgical repair is residual or worsening pulmonary regurgitation (PR), which leads to right heart failure and arrhythmias, necessitating pulmonic valve replacement (PVR). Thus, despite the benefits of early surgical repair, patients require lifelong care to ensure adequate quality of life [2].

2 | Brief Overview of Unrepaired TOF Pathophysiology

The exact embryologic origin of the anatomical defects seen in TOF is unknown. However, the cardinal features of the disease, including RVOT obstruction/PS, a malaligned VSD, concentric RV hypertrophy, and an overriding aorta, are well-defined and characterized [10, 11]. Indeed, it is now appreciated that VSD formation within the perimembranous region of the ventricular septum leads to secondary displacement of the aorta to the right overlying the VSD, a so-called "overriding aorta" leading to varying degrees of RVOT obstruction. Echocardiography is essential in establishing the diagnosis of unrepaired TOF in children and a few patients who survived into adulthood [12] (Figure 1, Video S1 and Figure 2, Video S2).

The VSD varies in size but allows for bidirectional shunting, with flow across it determined by the pressure gradient between the two ventricles [11]. In this manner, the resistance within the RVOT and systemic circulation dictates the contribution of deoxygenated blood to systemic cardiac output. If the resistance in the RVOT is less, blood will shunt left to right, and the patient will be acyanotic. However, if resistance in the pulmonary bed/RVOT increases to greater than systemic circulation, the shunt will reverse to right-to-left, leading to cyanosis. The RV remodeling seen in TOF is a consequence of the increased pressure in the RVOT/pulmonary trunk and includes RV concentric hypertrophy, dilation, and fibrosis. However, it should be appreciated that TOF exists on a clinical spectrum with varying degrees of RVOT obstruction and pulmonary valve disease/atresia, which dictates both the timing and urgency of repair.

3 | Discovery of TOF: Historical Perspective

The discovery and characterization of TOF went through four key historical eras (Figure 5).

3.1 | Initial Partial Description by Nicholas Steno

The constellation of anatomic findings now referred to as TOF was first described in 1671 by Danish anatomist Niels Steensen (1638–1686), better known by his Latinized name, Nicholas Steno [13]. Steno described a stillborn with numerous abnormalities, including omphalocele, syndactyly, and cleft palate. While performing the autopsy on the stillborn, he noticed a series of findings, including a narrowed RVOT, a connection between the RV and aorta (now appreciated as a VSD), and the aorta originating near the crest of the interventricular septum, implying the presence of an overriding aorta. Thus, with great accuracy, he

FIGURE 5 | Four key contributors to the description of TOF. Niels Steensen (1673): First described TOF Étienne-Louis Arthur Fallot (1888): Described the pathognomonic tetrad. Maude Abbott: Categorized TOF within broader cardiac anomalies. Stella Van Praagh: Influenced surgical management by prioritizing complete repair.

nearly characterized the complete pathology of TOF, except for RV hypertrophy.

3.2 | Full Description by Étienne-Louis Arthur Fallot

After the initial description by Steno, it took another two centuries before the pathognomonic tetrad of TOF was fully described by French physician Étienne-Louis 11 Arthur Fallot (1850–1911). Fallot graduated from the University of Marseille Medical School in 1876. While doing autopsies at the Hospital of Marseilles, he noted a constellation of findings in several cadavers which he named *maladie des enfants bleu* [the blue disease of the infants] for the bluish skin discoloration caused by cyanosis. In 1888, Fallot published the manuscript entitled *Contribution à l'anatomie pathologique de la maladie bleue*, in which he suggested that the tetrad of abnormalities seen in TOF could not have occurred consistently by chance, and there must be an underlying anatomical defect causing the disease [14]. He

also stated that the patent foramen ovale was probably not a fifth defect, given that it was unlikely to significantly contribute to the cyanotic presentation of these patients.

3.3 | Naming of Tetralogy of Fallot by Maude Abbott

The pediatric cardiologist Maude Abbott (1868–1940) was the next crucial figure in the understanding of TOF. She was the very first female Canadian cardiologist and made significant contributions to the understanding and classification of several CHDs. In her seminal publication, *The Atlas of Congenital Cardiac Disease*, she provided detailed descriptions and illustrations of some 1000 cases that revolutionized surgical approaches to these diseases [15].

She furthered our understanding of TOF physiology by noting that increased RVOT/PV resistance leads to intracardiac shunting to the left ventricle/aorta [16]. She also described evidence of RV dilatation and RV hypertrophy, constituting the fourth pathological element of TOF [16]. It was Maude Abbott who was the first to coin the term tetralogy of Fallot. Abbott's case series lists 41 cases of TOF, noting that patients had a relatively short span of life, with most dying in the first or early second decade before adult life(maximum age 36 years old) [17]. Her meticulous documentation of cardiac anomalies and defects in the late 19th and early 20th centuries laid the groundwork for categorizing TOF within the broader spectrum of cardiac anomalies.

3.4 | Modern Contributions to TOF by Stella Van Praagh

More recently, the research of Stella Van Praagh (1927–2006) of the United States significantly influenced the surgical management of TOF by providing a deeper understanding of the anatomical features and guiding surgical techniques. Specifically, her work clarified the critical need for comprehensive surgical intervention that addressed multiple aspects of the condition. She highlighted the importance of precise VSD closure to prevent residual shunting, relieving RVOT obstruction with adequate resection of obstructing tissue, and use of patches or conduits to allow adequate blood flow to the pulmonary artery (PA), particularly when it is hypoplastic or stenosed [18]. Stella van Praagh's work further defined the anatomy of the conal septum (the portion of the outflow tract that separates the ventricles), refining surgical approaches to the conal septum and the infundibulum, which improved outcomes. She also helped establish guidelines on the optimal timing of surgical intervention to maximize benefits and minimize risks, supporting early corrective surgery to prevent long-term complications associated with untreated TOF. Thus, Stella van Praagh's work laid a framework for the current surgical approach to TOF, prioritizing complete repair when possible.

4 | A Historical Perspective on TOF Treatment

In 1944, TOF was the first congenital heart lesion to be palliated [19], and 10 years later, also the first complex cardiac lesion to undergo successful open repair [20].

4.1 | Palliation via Systemic to Pulmonary Shunts

The first documented palliative surgery was the usage of a Blalock-Taussig-Thomas (BTT) shunt in 1944, a technique developed at Johns Hopkins Hospital by surgeon Alfred Blalock, cardiologist Helen B. Taussig, and assistant Vivien Thomas. The original BTT shunt involved the formation of a direct anastomosis between an aortic branch (classically the subclavian artery) to the pulmonary artery (PA) to relieve cyanosis [19, 21].

This technique was refined in 1976 to the modified BTT shunt, which implements a polytetrafluoroethylene graft to create a systemic-pulmonary shunt between the subclavian and pulmonary arteries without compromising the subclavian artery, thus preserving blood flow to the ipsilateral arm [22, 23].

More recently, palliative procedures have expanded to include surgical RV-PA conduit formation as well as percutaneous catheter interventions, first described in the 1990s, including RVOT and patent ductus arteriosus stenting [24–26]. Palliation is typically done in high-risk groups such as low-birth-weight neonates (<3 kg), those with small branch PAs, extracardiac congenital abnormalities, and other evidence of end-organ compromise [8]. Where possible, staged catheter-based palliation is preferable to surgical shunt procedures as a bridge to complete surgical repair.

4.2 | Initial Surgical Repair of TOF Following BTT Shunt

Initially described in 1954 by Lillehei et al., roughly a decade after the first documented use of a palliative BTT shunt, the landscape of complete surgical repair has evolved drastically. This landmark paper described a dual surgical goal of VSD closure and relieving RVOT obstruction or PS while placing patients on cardiopulmonary bypass [20]. Age at surgery ranged from 18 months to 14 years, with an emphasis on palliation in children under 2 years old.

4.3 | Complete Surgical Repair in Infants

After a growing body of evidence pointed to late morbidity with systemic PA shunts such as the BTT shunt, Castaneda et al. in the 1970s demonstrated that complete surgical repair in symptomatic infants (<1 year old) was safe and well-tolerated, thus avoiding a prior palliative operation [27]. In the modern day, complete surgical repair is now routinely done in infancy with excellent postoperative survival reaching >90% at 30 years [9].

Even in asymptomatic/acyanotic "pink" patients, data now support surgical repair between 3–6 months old due to both decreased mortality and length of stay in the intensive care unit/overall hospital stay [6–8]. Neonatal repair is typically reserved for symptomatic patients. The use of palliative shunting has declined to only 7% of primary operations, with the majority performed in neonates [28]. Complete surgical repair is typically performed with the goal of preserving RV lifespan over a patient's lifetime and preventing the need for reintervention when possible.

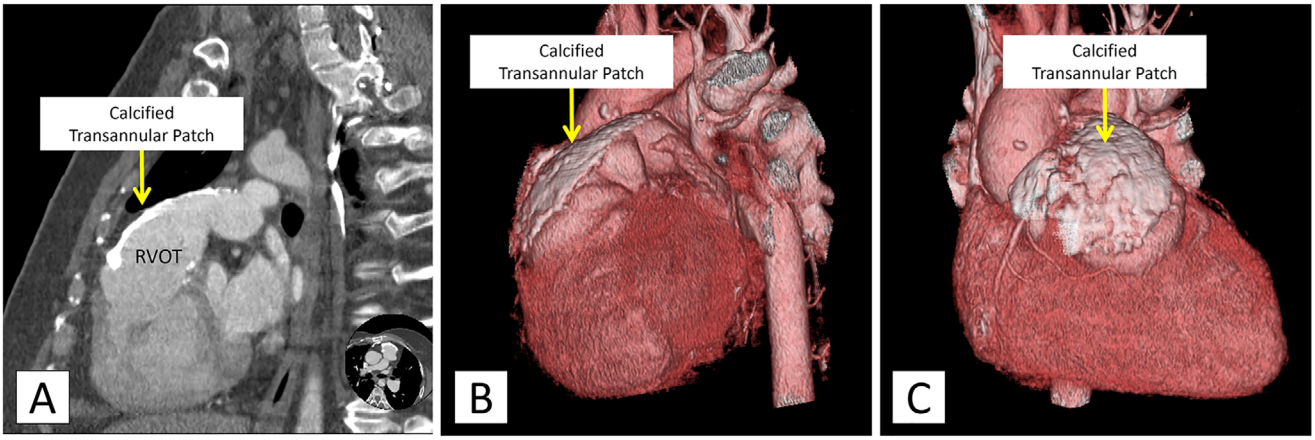


FIGURE 6 | CT imaging of TOF repair 2D (Panel A) and 3D (Panels B and C) CT imaging shows a calcified transannular patch in an adult patient post-childhood TOF repair. RV, right ventricle.

Trans atrial approaches are preferable to ventriculotomy where possible, with the major decisions comprising if the pulmonary valve requires intervention with a transannular patch (TAP) leading to free pulmonary regurgitation or if a valve-sparing repair (VSR) is sufficient [8, 24, 29]. The type of surgical repair is largely dependent on the anatomic/morphologic characteristics of the RVOT obstruction as well as institutional preferences, although ventriculotomy with TAP (44%) remains the most common reparative technique compared to valve-sparing repair (29.1%) or no ventriculotomy (24.8%) [28]. Preoperative echocardiography is an essential diagnostic tool for determining the level of obstruction (subvalvular, valvular, or supravalvular), pulmonary valve anatomy and function, PA anatomy, and coronary artery anatomy for surgical planning. Multimodality imaging is essential in visualizing the results of surgical TOF repair, including calcification of the transannular patch (Figure 6) [8].

4.4 | Long-Term Complications of Surgical TOF Repair

While surgical repair of TOF has dramatically improved the survival of patients into adulthood, many will eventually require reintervention, typically in the form of PVR. Echocardiography (Figure 7 and Video S3) and MRI (Figure 8 and Video S4) are key imaging modalities for the diagnosis and quantification of pulmonic valve regurgitation and its impact on RV remodeling [12]. Changes that occur post-repair include restenosis of the RVOT and branch PAs or a residual RVOT gradient. The use of a trans atrial approach in particular causes chronic PR, which leads to RV overload, ventricular remodeling, and the eventual need for PVR [30]. Patients develop symptoms such as chronic dyspnea, palpitations, and decreasing exercise tolerance in the presence of worsening RV dysfunction and atrial/ventricular arrhythmias [30, 31]. In some patients, RV dilation is severe enough to cause left ventricular dysfunction associated with ventricular arrhythmia, reduced exercise capacity, and sudden death. Sudden cardiac death remains a concern in adults with repaired TOF and might be an indication for ICD implantation.

4.5 | Surgical Reintervention Post TOF Repair

When excessive RV remodeling occurs due to severe chronic pulmonic regurgitation, surgical (Figure 9 and Video S5) or transcatheter (Figure 10 and Videos S5 and S6) implantation of a prosthetic pulmonic valve is indicated. The major parameters for which PVR has been shown to provide benefit in recent work are reduction of mortality and episodes of sustained ventricular tachycardia (VT) [32]. These mortality benefits and reductions in VT occurrence were not demonstrated in prior studies, suggesting improvements in technique and clinical timing of PVR [33, 34]. However, the optimal time for re-intervention with PVR remains unknown, with hypotheses that increased time to PVR would increase RV load from worsening regurgitation.

The greatest benefit of PVR has thus far been shown with advanced disease, given the risk of developing mechanoelectrical cardiomyopathy, arrhythmias, and sudden cardiac death [32, 36]. Parameters including RV end-diastolic volume and QRS duration on electrocardiograms are useful for stratifying the level of RV disease, which can help dictate proper timing for valve replacement [35]. While it would seem early PVR is the most beneficial, younger age has been associated with reduced bioprosthetic valve durability and shorter time to reintervention [36–38]. A multimodality approach, including echocardiography, cardiac CT, and cardiac MRI, is crucial to determine the perfect timing of PVR. The CMR criteria for pulmonary valve replacement in asymptomatic patients include RV end-diastolic volume index more than 150 mL/m^2, RV end-systolic volume index more than 80 mL/m^2 and RV ejection fraction (RVEF) less than47%.

Other hemodynamically significant abnormalities, such as RVOT aneurysm, tachyarrhythmias, tricuspid regurgitation, LV dysfunction, and RVOT obstruction, also play a role [35].

As discussed previously, valve preservation with a valve-sparing repair typically leads to lower rates of reintervention, but this is not always the case if the annulus is spared at the cost of some degree of persistent RVOT obstruction. While VSR initially spares the pulmonary valve, the valve is inherently dysplastic and, without proper support, develops regurgitation as well, necessitating the need for intervention. Family counseling at the

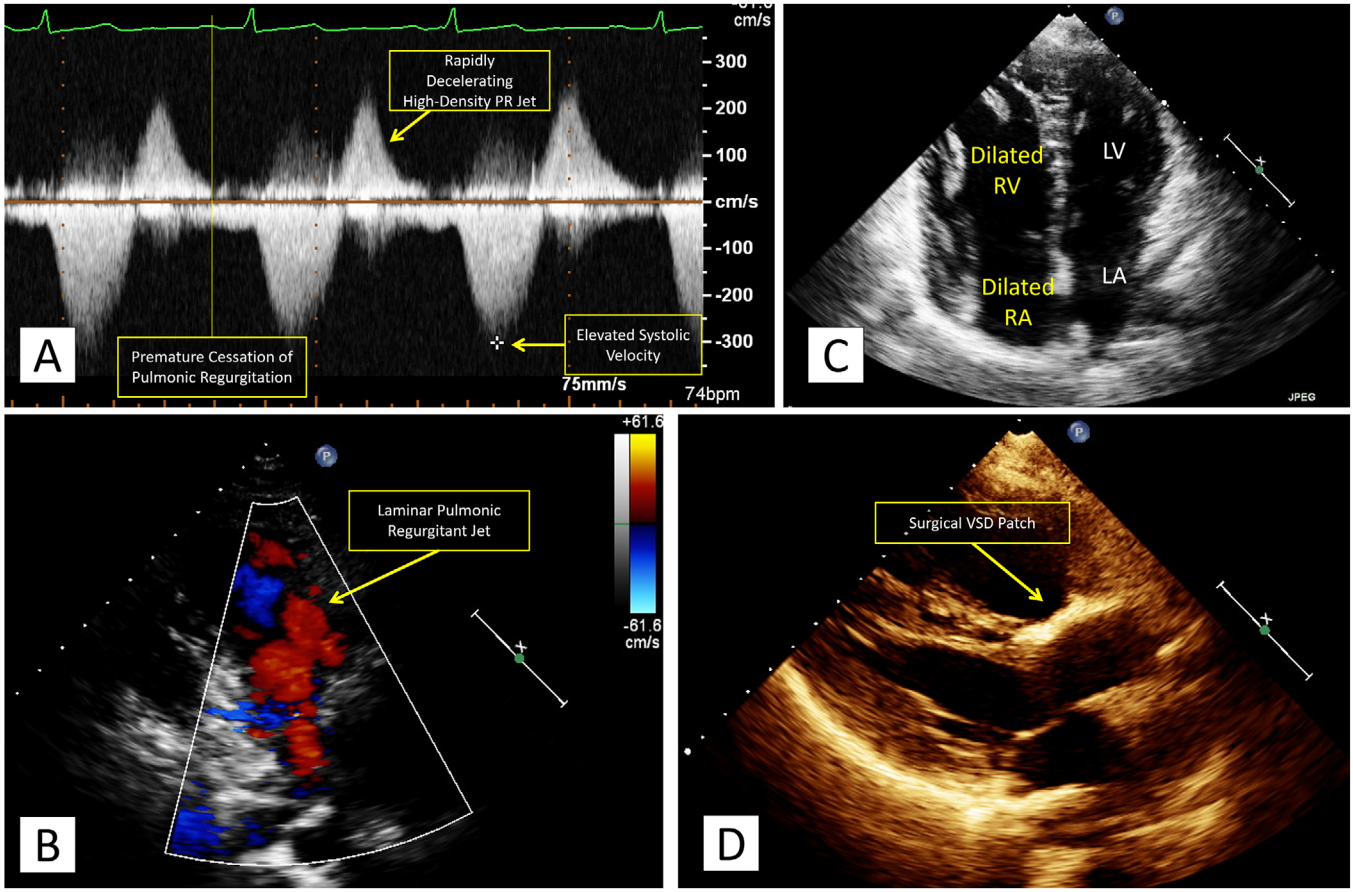


FIGURE 7 | Severe pulmonic regurgitation post TOF repair on echocardiography Panel A: Spectral Doppler shows rapid PR jet deceleration, premature cessation, and elevated systolic velocity (~3 m/s), indicating severe PR. Panel B: Color Doppler shows laminar PR jet flow due to severity. Panel C: Four-chamber view shows severe RV and RA dilation from chronic volume overload. Panel D: Parasternal long-axis view shows increased echo density at the site of surgically placed VSD patches. LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle; VSD, ventricular septal defect.

time of primary surgical repair in infancy or childhood is essential for the potential for reintervention and symptoms that may arise in adulthood.

4.6 | Transcatheter Reintervention Post TOF Repair

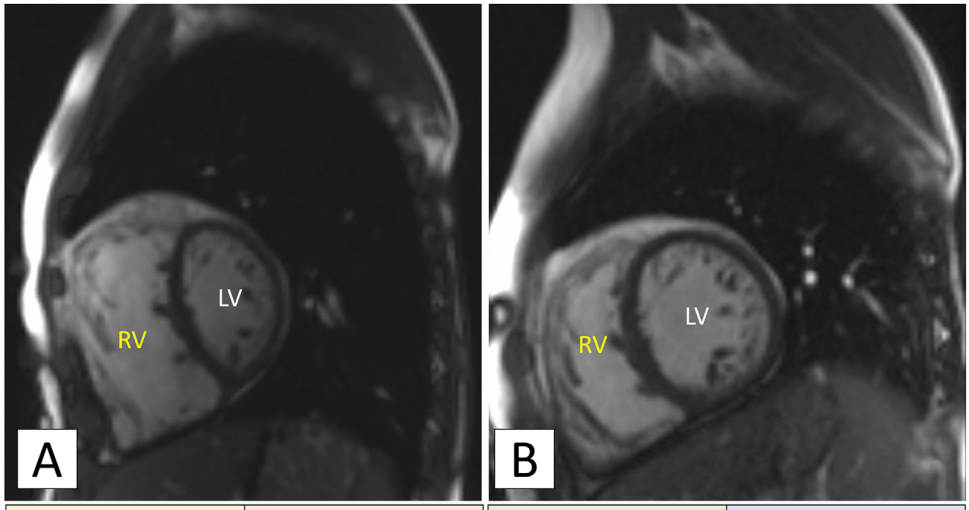
First described in 2000 by Bonhoeffer et al., transcatheter PVR is increasingly becoming the standard of care in large centers that care for CHD patients [39, 40]. While surgical PVR carries increased risk, including re-sternotomy, transcatheter PVR is generally low-risk, and the 10-year rate of freedom from intervention is around 70%–85%. Recent long-term data have suggested a median BPV durability of up to 17 years without significant differences in the type of BPV in data combined from both surgical and transcatheter PVRs [41]. Pre-procedural multimodality imaging plays a crucial role in appropriate case selection for transcatheter pulmonary valve repair/replacement. While echocardiography (especially with 3D) remains the main diagnostic tool, cardiac CT and MRI play a critical role in detailed anatomy evaluation and defining intervention strategies. Transcatheter approaches additionally carry the advantage of performing valve-in-valve repairs after initial valve replacements (Figures 9 and 10, Videos S5 and S6), although they have been shown to carry higher rates of infective endocarditis [41–43].

The currently available transcatheter pulmonary devices include Harmony (Medtronic): a self-expanding valve for native RVOT and Alterra Adaptive Prestent (Edwards): a structure implanted to remodel RVOT, creating a landing zone for the Edwards Sapien S3 valve [44].

The future direction of pulmonary valve repair in TOF will focus on minimally invasive techniques, hybrid approaches of combining surgical and catheter-based, as well as size-adjustable valves, which will allow expanding the valve within the body. These would probably be addressed by using tissue engineering materials to create durable, growing valves that adapt to the developing bodies of children and young adults [44–46].

5 | Conclusions

Multimodality imaging is key to understanding the anatomy and pathophysiology of TOF, initial surgical operative planning, surveillance, and eventual pulmonary valve replacement. Care and management of TOF have vastly improved from initial surgical palliative techniques to the modern era. The landscape



Right Ventricular Parameters	Prior to PVR	One Year Post PVR	Post PVR Improvement
End-Diastolic RV Volume (mL)	408	209	-199
End-Systolic RV Volume (mL)	256	113	-143
RV Stroke Volume (mL)	152	96	-56
RV Ejection Fraction	37%	46%	+ 9%

FIGURE 8 | Severe pulmonic regurgitation post TOF repair on MRI cardiac MRI short-axis views at end-diastole pre (Panel A) and post (Panel B) TOF repair. The table highlights RV size and function improvements post-surgery. LV, left ventricle; RV, right ventricle.

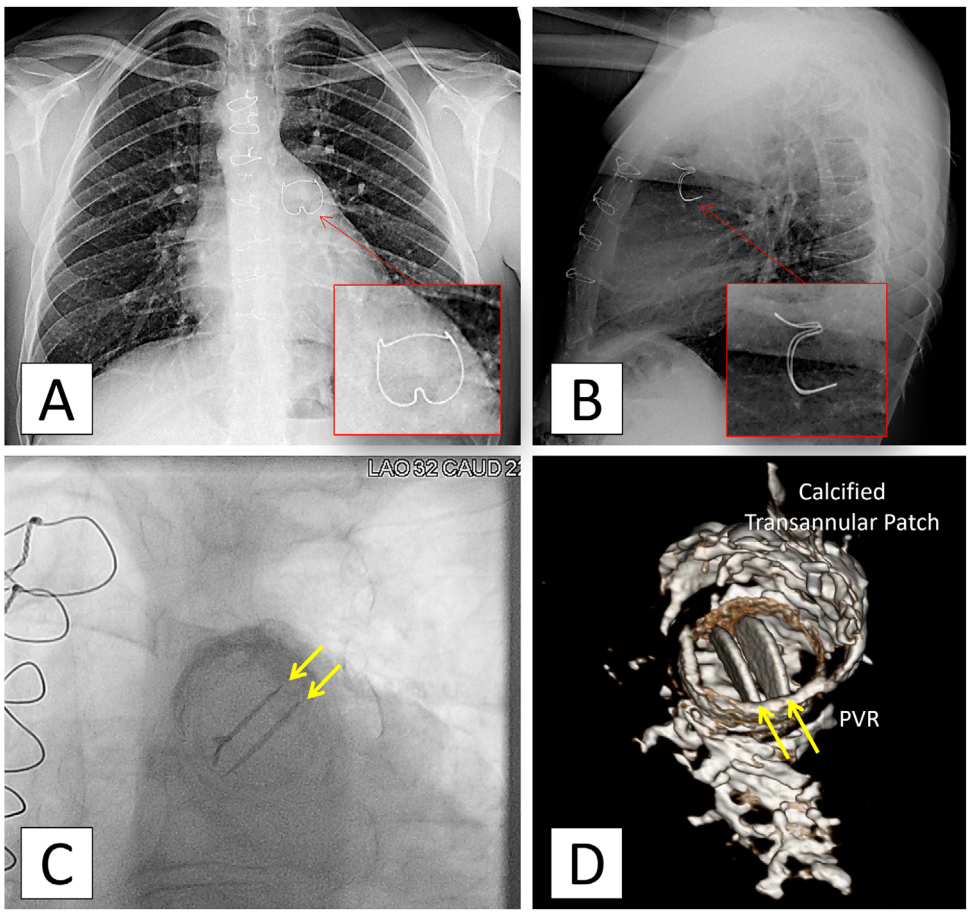


FIGURE 9 | Surgical pulmonic valve replacements Panels A and B: Biological pulmonary valve in a repaired TOF patient. Panel C: Fluoroscopy of a bileaflet mechanical pulmonary valve with normal leaflet position. Panel D: 3D CT of a St. Jude mechanical valve with calcified leaflets and transannular patch calcification (see Figure 6 for details).

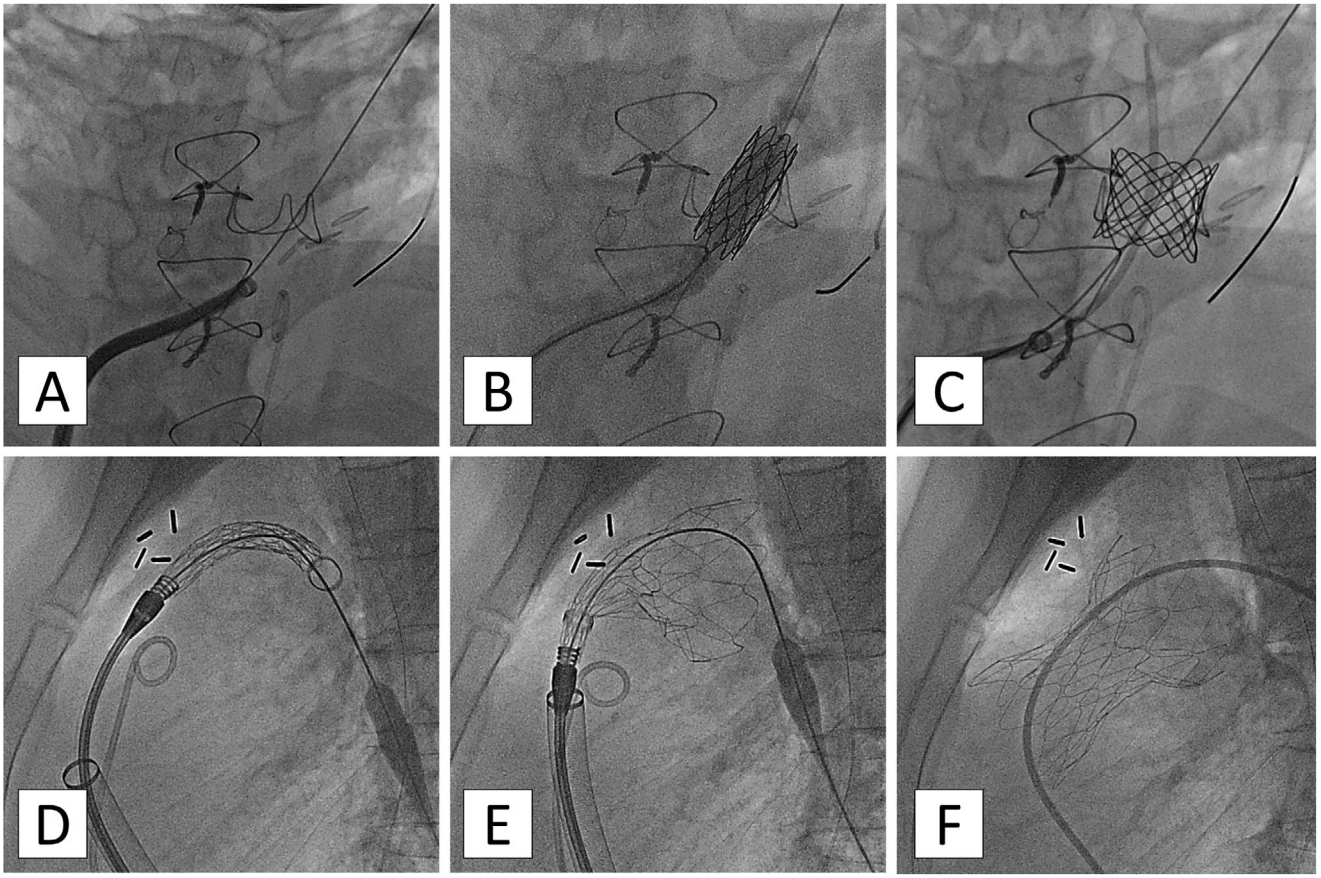


FIGURE 10 | Transcatheter paravalvular leak in repaired TOF with severe pulmonic regurgitation. Fluoroscopy shows transcatheter pulmonary valve implantation in two TOF patients. Panels A–C: Valve-in-valve procedure using a Melody valve in a prior biologic prosthesis. Panels D–E: Transcatheter native valve placement using a Harmony valve in a native pulmonary valve with severe PR.

of TOF management has evolved to prioritize complete repair in infancy, given evidence of improved outcomes. Determining the timing of post-repair PVR to preserve biventricular function will be crucial to improving adult quality of life.

While TOF patients continue to live longer, they face additional cardiac and non-cardiac barriers to a normal life that require regular, specialized follow-up. Gaining insights into the progress of the diagnosis and treatment of TOF patients plays an important role in improving the quality of care provided to this group. Ongoing studies and collaborations between clinicians, researchers, and industry are essential to address challenges and shape future innovations in the management of TOF patients.

Conflicts of Interest

The authors declare no conflicts of interest.

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Supporting Information

Additional supporting information can be found online in the Supporting Information section.

Video 1: echo70306-sup-0001-VideoS1.mp4 **Video 2:** echo70306-sup-0002-VideoS2.mp4 **Video 3:** echo70306-sup-0003-VideoS3.mp4 **Video 4:** echo70306-sup-0004-VideoS4.mp4 **Video 5:** echo70306-sup-0005-VideoS5.mp4 **Supporting Information:** echo70306-sup-0006-VideoS6.mp4 **Supporting Information:** echo70306-sup-0007-VideoS7.mp4