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Front cover:
Panoramic view of Knysna lagoon from Feather bed Nature reserve, Knysna
Photo: Ruchika Meel



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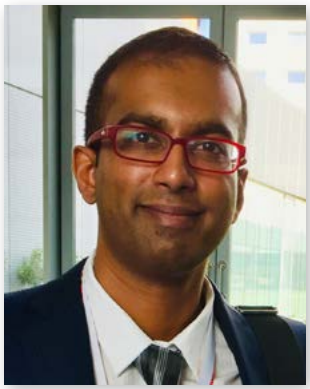
GUEST EDITORIAL

# Cardiac surgery in South Africa: Have we failed our legacy?



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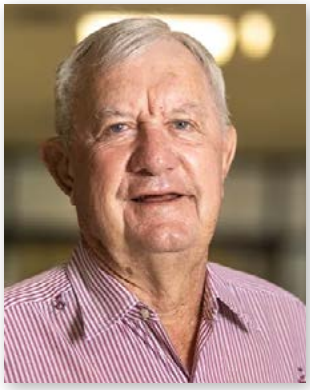

## INTRODUCTION

Cardiac surgery in South Africa (SA) was thrust onto the world stage in 1967 following Christiaan Barnard's world-first orthotopic heart transplant in Cape Town. This pioneering achievement defined the country as an unlikely leader in daring surgical innovation, clinical excellence and laboratory research.[1] The decades following the first heart transplant launched the speciality on a pathway of surgical excellence, evident by world-class surgeon-leaders and internationally renowned training units.

That stellar trajectory has unfortunately reversed, and the current SA cardiac surgery landscape is characterised by the lack of experienced academic leadership, virtually non-existent training and surgical programmes, and the lack of outcomes reporting and benchmarking in both private and public sectors.

## A TRADITION OF EXCELLENCE

In the late 1950s, using a pump-oxygenator donated by the United States government, Christiaan Barnard established the country's first cardiac surgery programme at Groote Schuur Hospital. Although his achievements in the field of heart transplantation are most well publicised, Prof Barnard made immense early contributions to congenital heart surgery, specifically the repair of Tetralogy of Fallot and Ebstein anomaly.[2]

A decade later, Professor Ben le Roux established a world-renown thoracic surgery service at the Wentworth and King George V Hospitals in Durban. Mr Andrew Logan joined the service following his retirement from Edinburgh in 1972, marking the golden years of thoracic surgery training in SA. Prof le Roux's monographs remain some of the most eloquently written texts in thoracic surgery.[3]

Professor Hannes Meyer established the cardiothoracic surgical training programme in Bloemfontein in 1971. This unit developed an excellent coronary artery revascularisation programme, a tissue laboratory and the largest national homograft bank in the country. The research unit was renamed the Robert W.M. Frater Cardiovascular Research Centre in 2015, in recognition of another

outstanding South African cardiac surgeon and researcher. The eponymous Hannes Meyer registrar symposium is held annually as a tribute to his commitment to training.[4]

In the 1980s, the Johannesburg cardiac surgery complex was one of the most productive units in the world, with over 1 000 cardiac cases per year under the leadership of Professor Robin Kinsley. The programme, like the Durban unit, distributed a comprehensive report annually with details of all procedures made, meetings held, training activity and visitors to the unit.

## OUR FINEST EXPORTS

During this era, cardiac surgery in SA was an innovative and dynamic environment with titans in the field keen to attend local meetings, such as the South African Cardiac Society (SACS) meeting at the Carlton Hotel in Johannesburg in 1980, the precursor to today's SA Heart® annual meeting. Many South African surgeons expanded their training abroad, establishing themselves as excellent surgeons leading high-profile surgical centres around the world. In the United Kingdom, this included the likes of Donald Ross, who conceptualised and developed the Ross operation and Sir Terence English, a leader in heart transplantation and president of the Royal College of Surgeons. In the United States, Chris Knott-Craig and Hillel Laks are highly revered congenital heart surgeons, and Lars Svensson leads the Heart and Vascular Institute at the Cleveland Clinic, the highest-ranked adult cardiac surgery programme in the country.

## CARDIAC SURGERY IN SOUTH AFRICA TODAY

The growth and development of high-volume surgical centres of excellence is consistent across the spectrum of high-income countries such as the United States, Germany and the United Kingdom, to previously underdeveloped low-to middle-income countries (LMICs) such as India and China.

In contrast, the phrase that best describes the state of cardiac surgery in SA today is "a ship without a captain, drifting aimlessly". The most significant factors contributing to this situation are the absence of strong surgical leadership, and the lack of accountability from the relevant role players – these include national and provincial health departments, academic hospital management, university training departments, healthcare funders, the Colleges of Medicine of South Africa (CMSA), the Health Professions Council of South Africa (HPCSA), and the Society of Cardiothoracic Surgeons of South Africa (SCTSSA). The fragmentation of responsibility for service delivery, training and research make it difficult to develop a cohesive, constructive strategy to define and improve the situation.

## SERVICE DELIVERY

The national health shift toward primary healthcare necessitated the defunding of state tertiary hospital healthcare services. This destruction of services has significantly hindered service delivery and training in cardiothoracic surgery. Over 80% of the population have no access to private medical care, and the state sector is overwhelmed with the burden of surgical disease in this patient population.[5]

State institutions struggle to deliver appropriate and timeous surgical care to the population due to resource constraints, as is evident from the long surgical waiting lists for routine procedures such as coronary artery bypass grafting (CABG), valve surgery and the correction of congenital heart defects.[6] With diminished surgical capacity, patients present with advanced disease increasing operative risk, and the surgical capacity becomes increasingly crisis-driven.

The situation has reached a disastrous level in paediatric and congenital heart surgery, and no state institution offers comprehensive congenital cardiac surgery.[7] Most provinces offer little to no congenital heart surgery service, and formal referral pathways do not exist for patients to access care elsewhere. Under these circumstances, the waiting list is more accurately a death list.[8] The neglect of congenital heart surgery on the African continent remains a huge challenge, and it is disappointing that SA has been overtaken by other LMIC's such as India and China in this regard.

The lack of funding for specialist surgeon posts, training of specialised nursing, limitations from cardiac anaesthesia and cardiovascular perfusion, lack of consumables and unlimited private practice by specialists have in combination strangulated the potential of many units. The decline of surgical volumes in state hospitals has encouraged an exodus of surgeons to private practice, leaving academic units without experienced specialists.

Many full-time state sector specialists undertake unlimited private practice during normal working hours, leaving trainees without supervision. The general public is well aware of the overall poor service delivery in state hospitals, and the sporadic "dog-and-pony show" type of media release does little to save face, ignoring the multitudes dying on waiting lists every week.[9]

## TEACHING AND TRAINING

Academic units are the mainstay of surgical training, and the success of cardiac surgery programmes is highly dependent on surgical case volumes and senior supervision. Prior editorial reflections within the SA Heart® Journal have questioned the trajectory of cardiothoracic surgery training in SA, asking bluntly: "Where to from here?".[10]

The CMSA serves as the sole examination body, and the introduction of internationally derived continuous assessment programmes, together with recalibration of the examination process using education theory is hoped to improve the assessment of candidates.

Revision of the admission criteria to the examination has been necessary, else no graduates would be produced in a country that desperately lacks cardiothoracic surgeons. The admission

requirement to the Fellowship exam of 75 open heart surgery cases as primary surgeon has been forsaken, as most units in the country do not achieve anything close to this. The most advanced assessment models cannot compensate for the inadequacy of training programmes, and instead the process becomes tailored to the candidate that has seen little and performed even less surgery.

Simulation methodology is a useful adjunct to enhance technical skills in junior trainees, but cardiac surgery is a true apprenticeship and there is no substitute for registrars performing surgery under the direct supervision and guidance of an experienced consultant.

Beyond the final Fellowship examination, junior specialists are seldom ready to embark on safe independent practice, and they require a controlled environment with senior support – this is standard surgical practice globally. Without guidance, many surgeons struggle to manage complex cases or complications, and lose the trust of colleagues in cardiology, anaesthesia and nursing due to poor outcomes.

## RESEARCH OUTPUT AND OUTCOMES DATA

Most research output from academic units comprise of Masters theses published by trainees, usually begrudgingly as a prerequisite for independent specialist registration. Case reports and small case series appear intermittently, but in the absence of high-volume cardiac surgical units it is unsurprising that few contemporary large data series exist. Our unique intersection of late presenting LMIC pathology and novel and unique surgical techniques have much to contribute to the rest of world.[11-14]

Outcomes analysis and reporting are a cornerstone of cardiac surgery quality assessment. The public reporting of surgical outcomes is mandatory in countries such as the United Kingdom, and patient and surgical outcomes data in the United States are submitted to the Society of Thoracic Surgery (STS) database for national benchmarking. Academic units in the past produced annual reports detailing surgical activity and outcomes, but this practice no longer exists. When concerns regarding poor surgical outcomes appear in the media, in the absence of validated surgical data to the contrary, the reputation of an academic surgical unit may be destroyed.

### Private practice

In contrast to the state sector, the private sector is oversupplied with resources, including cardiac surgeons of variable technical proficiency and experience. The proliferation of private cardiac surgery facilities has enticed many new graduates to independent private practice, often prematurely. This is encouraged by private hospital groups and the medical device industry, usually in the interest of profit generation and in many instances a profit-sharing model. Inexperienced surgeons without senior support struggle in an environment that does not tolerate a learning curve of inefficiency, incompetence, and poor outcomes. Under these circumstances, referrals decline and medico-legal troubles brew, and a retreat to diagnostic thoracic procedures is necessary to make a living.

The current fee-for-service private practice model is unsustainable in the long term and has been abandoned by many healthcare systems. Alternatives such as bundled care reimbursement are constantly being pursued by healthcare funders, however poor insight into the complex nature of cardiac surgery makes this challenging. The Health Market Inquiry into private practice in SA highlighted the overservicing of patients, creative billing and fraudulent coding, and cardiothoracic surgery is no exception to this, particularly amongst junior specialists entering private practice.[15]

Surgical outcome reporting is not mandatory in the private sector, and few practices submit data to registries such as the STS database or the World Society of Pediatric and Congenital Heart Surgery (WSPCHS) database, which is cost-free. Efforts by the Society of Cardiothoracic Surgeons of South Africa (SCTSSA) and others to promote national and even regional registries have been unsuccessful.[16,17] In contrast, healthcare funders have a vast quantity of metrics related to reimbursement and can crudely estimate clinical outcomes following surgery. This situation is far from ideal, but in the absence of surgeon-driven data, these remain the only measure of private practice activity. Without self-analysis and benchmarking of surgical outcomes, surgery is conducted in an intellectual vacuum. In a field defined by precision and accountability, the absence of data is itself a form of systemic opacity.

## THE FUTURE OF CARDIAC SURGERY IN SOUTH AFRICA

Cardiac surgery in SA is at a critical inflection point, and in many aspects has already fallen off a precipice that will take decades to resolve. We envisage two potential scenarios in the future:

### Scenario 1 – The downward spiral

At the current trajectory, cardiac surgery in SA is destined to plummet to the levels of our neighbouring countries in sub-Saharan Africa, where comprehensive surgical services no longer exist in either the private or state sectors.

With the decline in surgical volumes and case complexity at academic units, training will eventually cease and ultimately both state and private sectors will be staffed by foreign-national trained surgeons. Patients requiring cardiac surgery in SA will be completely dependent on visiting mission teams, as is the norm on the continent.

Alternatively, patients will be referred to private hospitals on the Indian subcontinent, another common practice in sub-Saharan Africa. This low-cost option is appealing to both the national government and private healthcare funders when complex interventions in adult and paediatric cardiac surgery, transplantation and advanced device therapies are no longer available in SA due to lack of expertise.

## Scenario 2 – Reinventing our legacy

In our view, drastic intervention is required in two specific areas to avoid the collapse of the specialty in SA: Strong leadership in academic units and active private-public integration.

### Leadership

One of the crucial factors contributing toward the decline of the cardiac surgery specialty in SA is the failure of leadership and accountability. The surgeon-leader of an academic unit should be a technically competent and experienced individual that has earned the respect of peers and colleagues, and most importantly is present at the coalface. The ability to teach, train and lead a team are required, ideally with an inclusive / servant leadership style. A toxic leadership style leads to the destruction of interpersonal relationships and is ultimately detrimental to a surgical unit.

### Private-public integration

The private sector retains substantial surgical capacity, infrastructure, and expertise, while the public sector bears the overwhelming burden of disease with limited resources.

Cardiac surgery as a discipline was once best known for coronary artery bypass grafting. Advances in PCI have driven cardiac surgeons to develop alternative areas of expertise and create centres of excellence in mitral valve repair, aortic valve repair, transcatheter aortic valve replacement, aortic vascular surgery, minimally invasive cardiac surgery and complex congenital cardiac surgery.

Much of this expertise lies in the private sector, particularly the fields of cardiac transplantation, mechanical circulatory support and complex congenital cardiac surgery. The highest volume of heart and lung transplantation exists in the private sector, and complex single ventricle congenital heart surgery is not offered in the state sector.

Trainees should be exposed to the best teachers, wherever they may exist. As no formal training programmes exist in the private sector, registrars are not exposed to the advanced surgical techniques and unique pathologies that are managed in private practice. Every such case is effectively a teaching opportunity lost, and many surgeons in private practice welcome the opportunity to mentor junior specialists in a formalised, regulated, mutually beneficial manner, rather than just as a pair of hands to assist. Similarly, academic units have much to gain from the appointment of senior surgeons from private practice in sessional training positions, particularly if they can offer expertise in specific areas.

The seamless integration of academic training units with private practice centres of excellence is essential for quality training of future generations of surgeons.

## CONCLUSION

There is a tendency within SA cardiac surgery to draw reassurance from our proud legacy. The narrative of past excellence—of Barnard, of early innovation, of global leadership—remains deeply embedded in the professional identity of the field. The uncomfortable reality is that legacy has, in some respects, become a substitute for accountability.

Instead, the discipline should be regarded as a pillar of national pride, and all efforts made to restore the former glory in a purposeful manner relevant to global practice patterns. Sophisticated tertiary care specialties such as cardiac surgery and transplantation in general should serve as a bellwether for the national health status, and we believe that by improving services at the apex the "rising tide will lift all boats", including secondary and primary healthcare.

Alternatively, without decisive and visionary leadership the field will continue its gradual decline—losing relevance, capacity, and ultimately, the ability to serve the patients who depend on it.

*"The best way to predict your future is to create it"*
*– Abraham Lincoln*

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EDITORIAL COMMENTARY

# Rethinking cardiac surgical care in South Africa: A call for a dialogue

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The editorial titled "Cardiac surgery in South Africa: Have we failed our legacy?" By Reddy et al. discusses the temporal transition of cardiothoracic surgery in South Africa.[1] Cardiothoracic surgery in South Africa has a strong international legacy, highlighted by the world's first heart transplant in 1967. However, at present services are unevenly distributed, with most specialists and resources concentrated in urban private hospitals, while the public sector faces long waiting lists and limited capacity. Chris Hani Baragwanath Academic Hospital (CHBAH), the third largest hospital in the world, currently lacks onsite cardiothoracic surgical services and has not performed cardiac surgery for nearly 30 years.[2] All surgical cases are referred to the overburdened and under-resourced Charlotte Maxeke Johannesburg Academic Hospital.[2]

The observations by Reddy et al. regarding late patient presentations, long surgical waiting lists and sub-optimal surgical outcomes are supported by recent research works of Banderker et al. and Sumaraj et al.[3,4] The study by Banderker et al. demonstrate that patients with predominantly rheumatic mitral valve disease in the South African public sector experience prolonged delays to surgery, presenting late with advanced heart failure, pulmonary hypertension and atrial fibrillation.[3] Sumaraj et al. show the downstream consequences of these delays: even after mitral valve replacement, patients remain burdened by residual ventricular dysfunction, pulmonary hypertension, high rates of atrial fibrillation and stroke, and poor anticoagulation control.[4] A notable finding was the low rate of concomitant tricuspid annuloplasty, despite the frequent presence of pulmonary hypertension and functional tricuspid regurgitation—conditions for which guidelinedirected surgery would support tricuspid intervention at the time of mitral valve replacement. The absence of tricuspid repair likely reflects late referral, surgical prioritisation under resource constraints, and limited operative time, and may contribute to persistent rightsided dysfunction after surgery. Together, the studies highlight a continuum of care failure in which delayed access to surgery, shortage of experienced surgeons, and inadequate longterm followup jointly drive morbidity. Improving outcomes will require not only surgical capacity expansion but also earlier referral and stronger postoperative care systems. Another study from central South Africa reported generally good outcomes of patients undergoing mitral valve surgery, with in-hospital mortality rates around 3.8 to 4.8%.[5] The author documented overall inhospital complication rate of 14% across the cohort. Data on long-term morbidity was not reported.

Prosthetic valve replacement in young patients as shown by Sumaraj et al. and previously reported by Antunes is associated with comparatively higher morbidity, even with modern valve designs.[4,6] For degenerative mitral valve prolapse a South African study has shown mortality rate for mitral

valve repair of 4.8% at 6 months.[7] However, current data on rheumatic mitral valve repair in adults is scarce in South Africa. Improved understanding of rheumatic valve pathology and advances in repair techniques have led to progressively better results in mitral regurgitation, making mitral valve repair a worthwhile and preferred strategy whenever feasible.[6] The likelihood of successful rheumatic mitral valve repair increases with the surgeon's experience and commitment to valve preservation, highlighting the importance of adequate surgical exposure and training.

The status pertaining to aortic valve disease management also presents a dismal picture. Dlamini et al. conducted a prospective study entitled "A cross-sectional study of clinical and echocardiographic characteristics of adult aortic valve disease at Chris Hani Baragwanath Academic Hospital".[8] Over half of patients with aortic valve disease presented with heart failure and experienced high rates of pulmonary hypertension and atrial fibrillation. A significant proportion awaited surgery. In a retrospective study (2010-2020) by Chingwaru et al "In-hospital mortality post-surgical aortic valve replacement for severe aortic valve stenosis at a quaternary hospital in South Africa" outcomes after isolated aortic valve replacement were poor, with high inhospital mortality largely driven by bleeding and sepsis, reflecting advanced disease and sub-optimal peri-operative care.[9] On the other hand a retrospective study by Scherman et al. reported in-hospital/30-day mortality for the rheumatic mechanical AVR of 1.9% but late mortality was high and primarily related to bleeding and thromboembolic events.[10]

Referral and surgery for complex cardiac pathologies such as aortic aneurysms and congenital heart disease (CHD) remains an ongoing challenge. Meel et al. study on ascending aortic aneurysms showed that only about a third of patients underwent surgical intervention, and overall mortality remained significant, particularly among those presenting with acute aortic dissection. We concluded that ascending aortic aneurysms at CHBAH setting are common, present late, and are associated with considerable morbidity and mortality, especially in association with hypertension and HIV. Rossouw highlighted that CHD is the most common congenital abnormality globally and is associated with higher mortality than any other birth defect.[12] Yet, in Africa most children present late, often only after severe complications and multiorgan dysfunction have developed. Limited access to early diagnosis, including the underuse of simple and inexpensive screening tools such as pulse oximetry, contributes to delayed referral and poor outcomes.

Advances in the field of interventional cardiology offers an alternative to surgery but only to a select group of patients and regions in South Africa. The introduction of transcutaneous aortic valve implantation at major hospitals in the country now offers an alternative for degenerative aortic valve stenosis cases.[13] Similarly, the introduction of mitral clip in South Africa also allows for transcutaneous mitral valve repair in degenerative mitral valve regurgitation.[14] In an 11-year follow-up study Pillay et al. showed that percutaneous pulmonary valve implantation in central South Africa is a feasible, safe, and durable alternative to repeat surgery for right ventricular outflow tract dysfunction.[15] Procedural mortality was low, and long-term valve performance was good, with most patients experiencing sustained haemodynamic and clinical benefit.

The editorial also raises concerns regarding few training posts and the quality of training due to a shortage of experienced cardiothoracic surgery consultants in the state sector.[1] Existing literature makes clear that cardiothoracic surgery training crisis in South Africa is not primarily educational, but structural and political, reflecting underinvestment in specialist posts, weak succession planning, and declining publicsector surgical capacity.[16,17] Without urgent intervention to expand funded registrar and consultant posts in cardiothoracic surgery, cardiology and support staff involved in management of cardiac patients, the existing shortage is expected to worsen, with direct implications for patient mortality and the collapse of tertiary and quaternary cardiac surgical services.

As emphasised in the editorial, future progress will rely on strengthening publicsector capacity, advancing meritbased and competent leadership, expanding highquality training through structured registrar rotations in the private sector via strengthened public–private partnerships, and aligning health policy with the demands of highcost surgical care, supported by locally relevant research on surgical outcomes.[1] Only through effective dialogue between multidisciplinary teams involved in the management of cardiac patients and the Department of Health can the aforementioned challenges be addressed and solutions implemented before irreversible damage occurs and services collapse completely.

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# Temporal trends of transcatheter aortic valve implantation practice in South Africa

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## ABSTRACT

**Background:** The temporal trends in transcatheter aortic valve implantation (TAVI) practice and procedural benefits are well documented in high-income countries; however, data for upper-middle-income countries (UMIC) are sparse.

**Objectives:** This study aimed to describe the evolution of TAVI practice in South Africa, including patient and procedural characteristic profiles and outcomes, from 1 September 2014 to 31 December 2023.

Methods: The South African Heart Association (SHARE)-TAVI registry is a web-based, all-comers prospective registry. The 18 centres that were involved from the outset of the registry in September 2014 were included in our analysis.

**Results:** A total of 2 532 TAVIs were performed across the 18 centres. There was a steady increase in TAVI procedures, with most performed in private hospitals ($n$ = 2 251). Waiting times were shorter in the private hospitals, with a median of 52 days (interquartile range [IQR] 29–82), compared with public hospitals, with a median of 70 days (IQR 61–85). Over time, the median age remained stable at 81 years (IQR 75–85). The European System for Cardiac Operative Risk Evaluation (EuroSCORE) II showed a continuous and significant decline from 4.9% (IQR 4.4, 8.6) in 2014/15 to 3.5% (1.9, 6) in 2023 ($p$ < 0.001). Transfemoral access was the most prevalent access route utilised throughout the study period, and there was a trend of increased use of percutaneous closure devices with lower vascular complications (11% in 2014/15 to 5% in 2023; $p$ < 0.001). There was also a notable reduction in peri-procedural strokes (10% in 2014/15 to 2% in 2023; $p$ < 0.0001). Kaplan–Meier survival curves showed a gradual decrease in mortality risk ($p$ = 0.0344). Accordingly, the 1-year mortality fell from 17% in 2014/15 to 6% in 2022 ($p$ < 0.001).

**Conclusion:** This data showed a steady increase in the number of TAVI procedures during the study period, with a reduction in risk profiles despite the mean age remaining stable, consistent with international recommendations. Technical aspects of the procedures evolved and were associated with reduced complications.

**Keywords:** transcatheter aortic valve implantation practice, temporal trends, procedural benefits.
SA Heart® 2026;23:64-72

## INTRODUCTION

The advent of TAVI marked a paradigm shift in managing symptomatic, severe aortic stenosis, particularly in patients deemed high-risk for surgical aortic valve replacement. Pioneered by Cribier in 2002, this minimally invasive approach improved clinical outcomes, with 1-year mortality rates declining from approximately 50% in inoperable patients to below 20% in contemporary studies.[1,2] The remarkable progress can be attributed to refinements in patient selection, procedural expertise, evidence-based practice, and advancements in transcatheter device technology.[3-5] To our knowledge, South Africa is the only African country performing TAVI in both the

public and private sectors. It is therefore a valuable source of TAVI data in the developing world. [6] In South Africa, the first TAVI was performed in 2009, with subsequent expansion to 30 centres nationwide; the procedure is mostly performed in the private sector.[4,5]

TAVI registries in high-income countries use patient and procedural characteristics to describe temporal trends in TAVI practice. Procedural characteristics include the anaesthesia type, the transcatheter device, and the vascular access used for the procedure. Modifications in these components are associated with reduced peri-procedural vascular complications and shorter hospital stays.[7] Multiple previous TAVI registries demonstrated the importance of these modifications for immediate and long-term outcomes.[8-10] The transfemoral route is preferred, with up to 90% of procedures now performed via this route.[11,12] The current TAVI approach favours conscious sedation (CS) over general anaesthesia, and femoral arterial access via percutaneous femoral puncture and closure over surgical cut down.[13-16]

International guidelines for TAVI are based on data from high-income countries, whereas implementation in a UMIC has not been well characterised.[8,17] Despite the promising data from first-world registries, funding for TAVI is severely limited in South Africa. There is an urgent need for more robust, local data on TAVI to inform patient selection and guide policy and funding for TAVI. To systematically evaluate TAVI outcomes within a local setting, the SHARE-TAVI registry was established in September 2014. This multicentre registry encompasses all TAVI referrals and procedures nationwide and represents the most comprehensive and robust database of TAVI in South Africa. This study seeks to analyse temporal trends in TAVI practice in South Africa based on data from the SHARE-TAVI registry, thereby contributing to the optimisation of patient care and procedural standards in the region.

## METHODS

### Study rationale

This study aims to characterise the demographic and procedural characteristics of patients receiving TAVI in South Africa. Analyses include temporal trends in waiting times, funding sources, hospital stay duration, and short- and long-term clinical outcomes.

### Study population

The SHARE-TAVI registry is a prospectively collected, multicentre registry of all patients referred for TAVI in the South African public and private sectors. From September 2014 to December 2023, this registry enrolled 3 931 patients across 30 participating centres. The analysis was restricted to 18 centres (*n* = 2 532 patients) that contributed to the registry since its inception to detect temporal trends. Twelve centres were excluded from the analysis because they did not enrol patients from the inception of the registry; therefore, data from these centres do not inform the temporal trends of the registry (Figure 1).

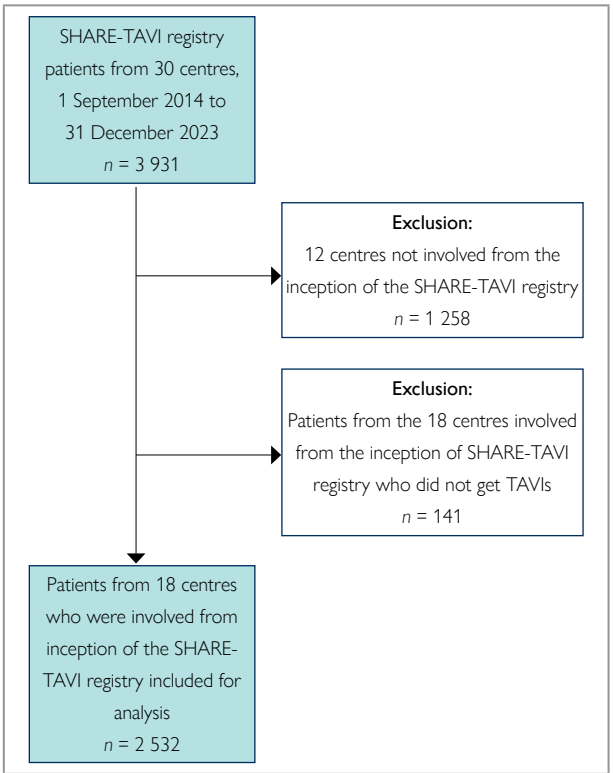



**FIGURE 1: Recruitment of study participants.**
*SHARE-TAVI: South African Heart Association transcatheter aortic valve implantation registry.*

### Statistical analysis

All analyses were performed using descriptive statistical methods. Continuous variables were assessed for normality using the Shapiro–Wilk test and represented as mean ± standard deviation for normally distributed data, or median with IQR for non-normally distributed data. Categorical variables were summarised using frequencies and percentages. For temporal trend analysis, we employed non-parametric Wilcoxon rank-sum tests to

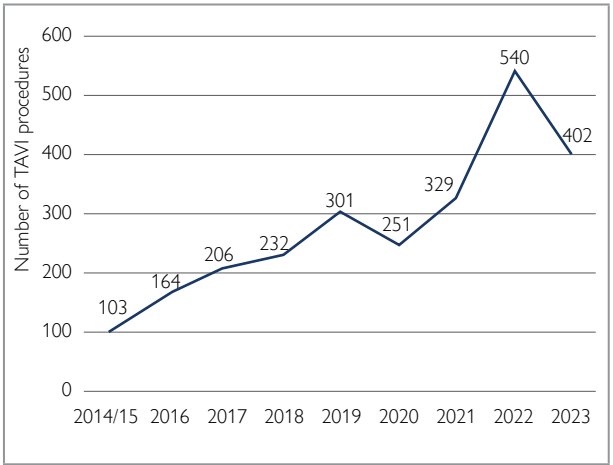



**FIGURE 2: Trends in the number of TAVI (transcatheter aortic valve implantation) procedures from 2014 to 2023.**

evaluate median differences in continuous variables across the study years. Proportional differences in categorical variables between years were assessed using chi-squared tests of association. The log-rank test was used to compare survival differences between groups. For normally distributed, cumulative mortality proportions, we performed one-way ANOVA (analysis of variance) to examine mean differences between study years. All tests were two-tailed, with $p < 0.05$ considered statistically significant.

### Ethical considerations

Ethical approval was obtained from the Health Research Ethics Committee of Stellenbosch University (reference: N14/06/073). All data were anonymised to ensure the privacy and confidentiality of participants' personal information, and each participant was assigned a unique identifier.

## RESULTS

From 1 September 2014 to 31 December 2023, 2 532 patients were prospectively included in the SHARE-TAVI registry at centres participating since its inception (Figure 1). Annual procedural rates increased from 103 in 2014/15 to 402 in 2023 (Figure 2). Most TAVI procedures were performed in the private sector, with the number increasing from 83 in 2014/15 to a peak of 490 in 2022. The number of cases performed in public hospitals annually remained ≤ 50 throughout the study period (Figure 3). Medical insurance companies covered most cases performed in private hospitals, either with full payment or co-payment, with the patient contributing out of pocket for the procedure. There was a decline in the full payment coverage provided by medical insurance companies from 55% in 2014/15 to 24% in 2023 (Figure 4). The waiting times to TAVI were significantly shorter in private hospitals, with a median of 52 days

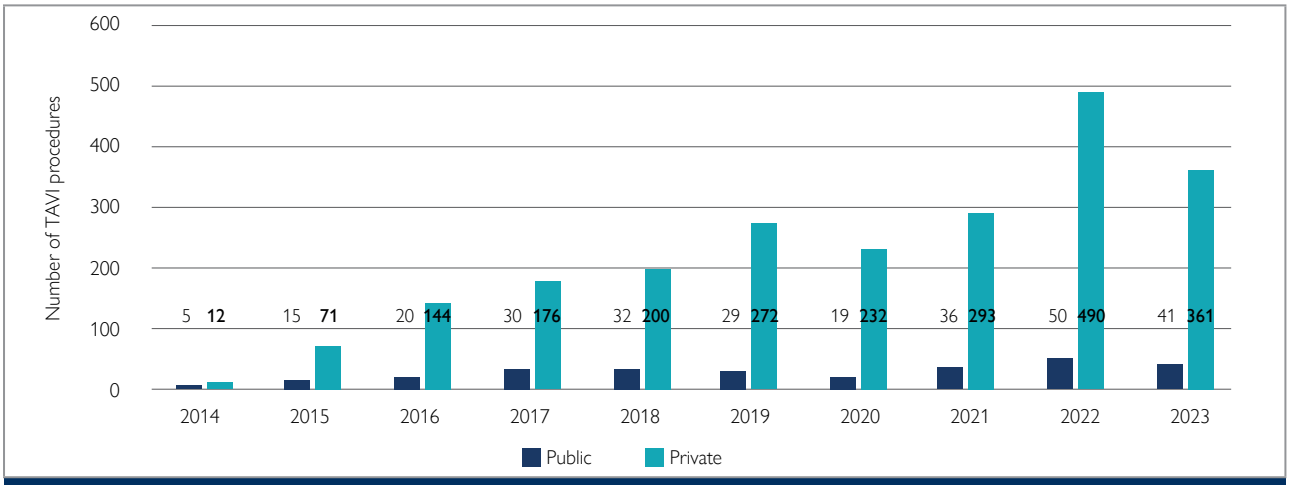



**FIGURE 3: Trends in the number of TAVI (transcatheter aortic valve implantation) procedures in public and private hospitals.**

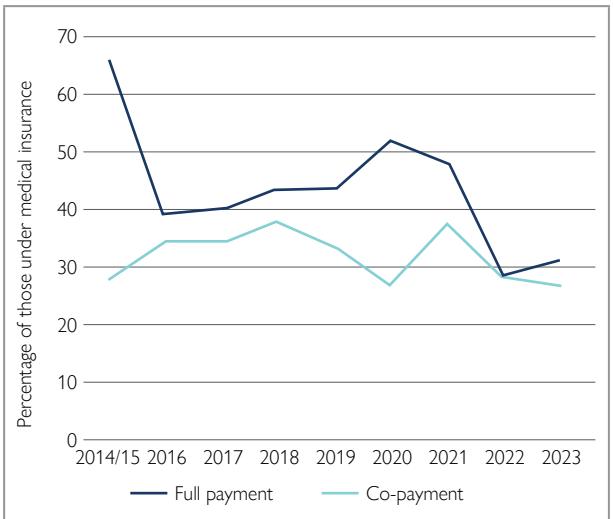



**FIGURE 4: Payment method trends for transcatheter aortic valve implantation procedures performed in private hospitals.**
*Note: Co-payment indicates patients who were required to make an additional payment to supplement their medical insurance coverage.*

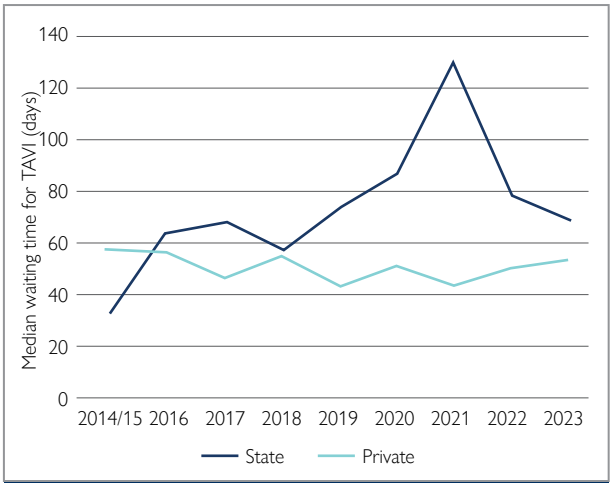



**FIGURE 5: Trends in median waiting times to TAVI (transcatheter aortic valve implantation).**

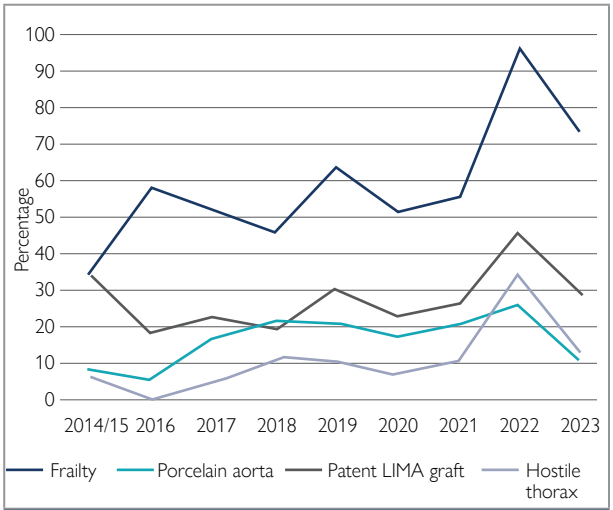



**FIGURE 6: Contraindications to surgical aortic valve replacements leading to transcatheter aortic valve implantation.**
*LIMA: left internal mammary artery.*

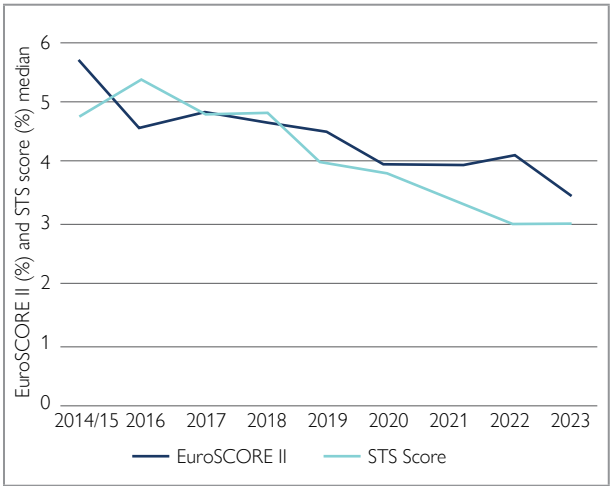



**FIGURE 7: Trends in predicted risk of mortality according to EuroSCORE II and STS score.**
*Note: p < 0.001 comparing the initial scores to the last scores in 2023.*
*EuroSCORE II: European System for Cardiac Operative Risk Evaluation, STS: Society of Thoracic Surgeons.*

**TABLE I: Baseline characteristics of the study population.**

| ***n*** | **2 532** |
|---|---|
| | ***n* (%)** |
| **Demographics** | |
| Age, years median (IQR) | 81 (75–84) |
| Male | 1 491 (59) |
| Female | 1 041 (41) |
| STS score, median (IQR) | 4 (2.6–6.5) |
| Log EuroSCORE II, median (IQR) | 16.3 (8–27) |
| **Comorbidities** | |
| Hypertension | 1 858 (73) |
| Diabetes mellitus | 601 (24) |
| Chronic lung disease | 392 (15) |
| Peripheral vascular disease | 386 (15) |
| **Clinical findings** | |
| Syncope | 518 (20) |
| CCS | 892 (35) |
| Atrial fibrillation | 331 (13) |
| Previous CVA | 218 (9) |
| Previous CABG | 522 (20) |
| Previous aortic valve procedure | 199 (8) |
| Previous PPM | 250 (10) |
| **Lab values** | |
| Haemoglobin, median (IQR) | 13 (11.8–14) |
| **Contraindications to SAVR** | |
| Frailty | 536 (21) |
| Porcelain aorta | 147 (6) |
| Patent LIMA graft | 228 (9) |
| Hostile thorax | 149 (6) |
| **NYHA class, *n* (%)** | |
| II | 853 (39) |
| III | 1160 (53) |
| IV | 192 (9) |
| Not recorded | 323 |

*CABG: coronary artery bypass graft, CCS: chronic coronary syndrome, CVA: cerebrovascular accident, EuroSCORE II: European System for Cardiac Operative Risk Evaluation, IQR: interquartile range, LIMA: left internal mammary artery, NYHA: New York Heart Association, PPM: permanent pacemaker, SAVR: surgical aortic valve replacement, STS: Society of Thoracic Surgeons.*

(IQR 29–82), compared with public hospitals, with a median of 70 days (IQR 61–85). There was an upward trend in TAVI waiting times in public hospitals, peaking in 2021, which coincided with the COVID-19 pandemic; however, waiting times remained unchanged in private hospitals (Figure 5).

## Baseline characteristics

The study population's median age was 81 years (IQR 75–84). The gender distribution revealed 59% males (*n* = 1 491) and 41% females (*n* = 1 041). Tables I and II show patients' baseline characteristics. There were no significant differences in age, sex, and left ventricular ejection fraction (LVEF) throughout the study period. A large proportion of the study population was hypertensive (73%, *n* = 1 858). Diabetes mellitus (24%, *n* = 601) and atrial fibrillation (13%, *n* = 331) were common comorbidities. The median transvalvular aortic gradient was 44 mmHg (IQR 40–66.2), and the median LVEF was 58% (IQR 49–65). The 4 common contraindications to surgical aortic valve replacement (SAVR) were frailty, porcelain aorta, previous coronary artery bypass graft with a patent left internal mammary artery, and hostile thorax (Figure 6). As illustrated in Figure 7, the

**TABLE II: Baseline characteristics per year of transcatheter aortic valve implantation procedure.**

| | All years | 2014/15 | 2016 | 2017 | 2018 | 2019 | 2020 | 2021 | 2022 | 2023 | *p*-value |
|---|---|---|---|---|---|---|---|---|---|---|---|
| ***n*** | **2 532** | **103** | **164** | **206** | **232** | **301** | **251** | **329** | **540** | **402** | |
| **Age, years (range)** | 81 (33–91) | 81 (50–91) | 81 (59–97) | 81 (59–97) | 80 (33–96) | 80 (50–94) | 80 (42–96) | 80 (41–97) | 80 (38–95) | 80 (54–95) | 0.4463 |
| | | | | | | ***n* (%)** | | | | | |
| Men | 1 491 (59) | 54 (52) | 85 (52) | 120 (58) | 128 (55) | 160 (53) | 153 (61) | 211 (64) | 323 (60) | 257 (64) | 0.023 |
| HPT | 1 858 (73) | 71 (68) | 108 (66) | 139 (67) | 174 (75) | 206 (68) | 175 (70) | 210 (64) | 370 (69) | 285 (71) | 0.001 |
| DM | 601 (24) | 25 (24) | 38 (23) | 59 (29) | 51 (22) | 63 (21) | 70 (28) | 79 (24) | 133 (25) | 83 (21) | < 0.001 |
| CVA/embolism | 187 (7) | 8 (9) | 10 (6) | 17 (8) | 16 (7) | 25 (8) | 17 (7) | 39 (12) | 55 (10) | 31 (7) | < 0.001 |
| Previous CABG | 522 (21) | 28 (27) | 41 (25) | 50 (24) | 53 (23) | 61 (20) | 53 (21) | 63 (19) | 104 (19) | 69 (17) | < 0.001 |
| Previous PPM | 250 (10) | 10 (9) | 15 (9) | 23 (11) | 26 (11) | 22 (7) | 23 (9) | 39 (12) | 55 (10) | 31 (7) | < 0.001 |
| AF | 331 (13) | 7 (7) | 27 (16) | 27 (13) | 36 (16) | 27 (8) | 32 (13) | 38 (15) | 89 (16) | 48 (12) | 0.002 |
| **NYHA** | | | | | | | | | | | |
| II | 853 (39) | 27 (26) | 44 (30) | 59 (32) | 64 (29) | 108 (39) | 109 (49) | 138 (47) | 175 (40) | 129 (38) | < 0.001 |
| III | 1160 (53) | 47 (45) | 89 (61) | 108 (59) | 133 (60) | 144 (52) | 96 (42) | 134 (46) | 219 (50) | 190 (56) | < 0.001 |
| IV | 192 (9) | 14 (18) | 12 (9) | 15 (9) | 23 (11) | 24 (9) | 19 (9) | 19 (7) | 44 (10) | 22 (6) | < 0.001 |
| **Median** | | | | | | | | | | | |
| Hb (g/dl) | 13 (11.8, 14) | 12.2 (11.3, 13.8) | 12.4 (11.3, 13.5) | 13 (11.9, 14) | 13 (12, 14.2) | 12.8 (11.3, 13.9) | 13 (11.8, 14.1) | 13.2 (12, 14.3) | 13 (11.8, 14) | 13 (11.9, 14.1) | < 0.001 |
| Mean PG (mmHg) | 44 (40, 66.2) | 47 (40, 60) | 47 (40, 58) | 44 (38, 57) | 45 (38.9, 54) | 45 (40, 35) | 44 (38, 55) | 44 (39, 55) | 44 (39, 55) | 45 (38, 55) | 0.0633 |
| LVEF (%) | 58 (49, 65) | 53 (40, 63) | 57 (45, 65) | 58 (45, 65) | 58 (45, 65) | 56 (45, 65) | 60 (50, 65) | 58 (50, 65) | 59 (50, 65) | 60 (50, 65) | 0.0853 |
| EuroSCORE II | 4.2 (2.3, 7.8) | 5.7 (4.1, 8.7) | 4.6 (3, 8.8) | 4.8 (2.9, 8.9) | 4.7 (2.5, 8) | 4.5 (2.4, 8) | 4 (1.9, 8) | 4.2 (2.1, 8.9) | 4.1 (2.2, 8) | 3.5 (1.9, 6) | < 0.001 |
| STS score | 4 (2.6, 6.5) | 4.8 (3.2, 7.25) | 5.3 (3.3, 9) | 4.8 (3.2, 7) | 4.8 (2.5, 7.5) | 4 (2.5, 6.2) | 3.8 (2.3, 6.9) | 3.4 (2.2, 5.9) | 3 (2.3, 6) | 3 (2.3, 6) | < 0.001 |

*AF: atrial fibrillation, CABG: coronary artery bypass graft, CVA: cerebrovascular accident, DM: diabetes mellitus, EuroSCORE II: European System for Cardiac Operative Risk Evaluation, Hb: haemoglobin, HPT: hypertension, LVEF: left ventricular ejection fraction, NYHA: New York Heart Association, PG: pressure gradient, PPM: permanent pacemaker, STS: Society of Thoracic Surgeons.*

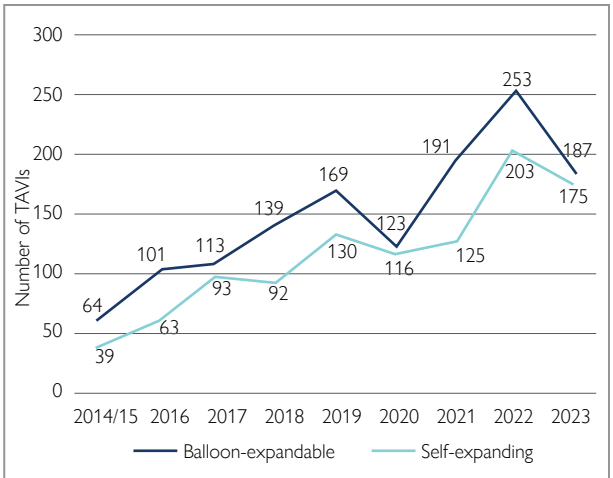



**FIGURE 8: Trends of balloon-expandable and self-expanding valves in TAVIs (transcatheter aortic valve implantations).**

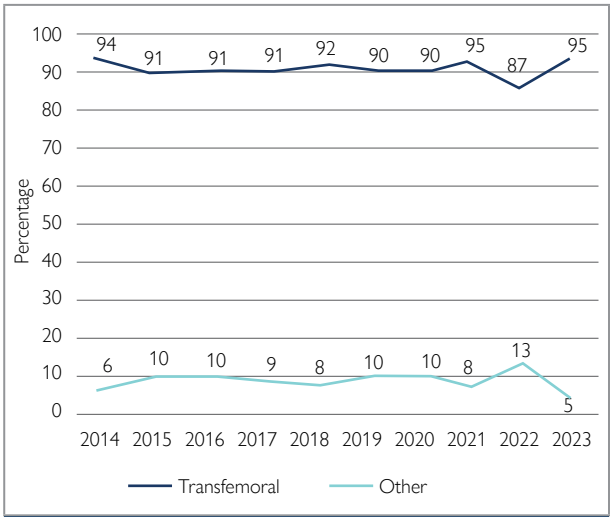



**FIGURE 9: Trends in access sites for transcatheter aortic valve implantation.**
*Note: Other includes transaortic, transapical, subclavian, and carotid.*

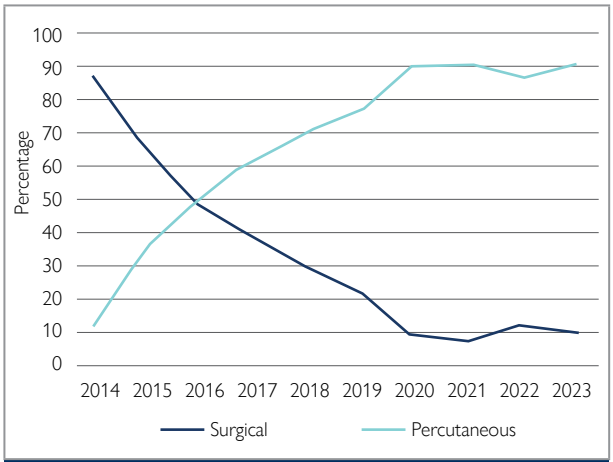



**FIGURE 10: Trends in use of surgical versus percutaneous closure of transfemoral access sites.**

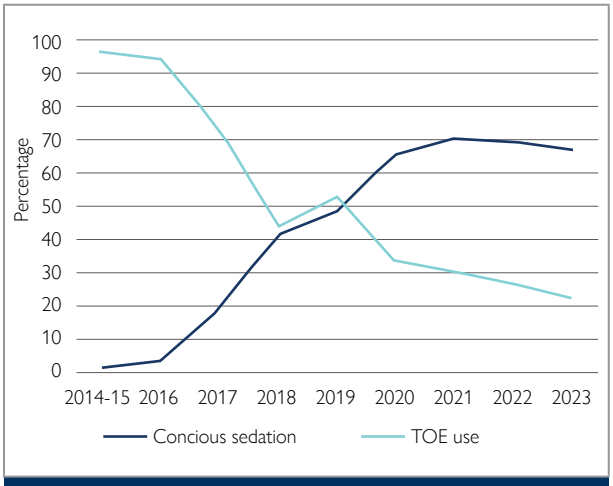



**Figure 11: Trends in TOE (transoesophageal echocardiography) use and conscious sedation.**

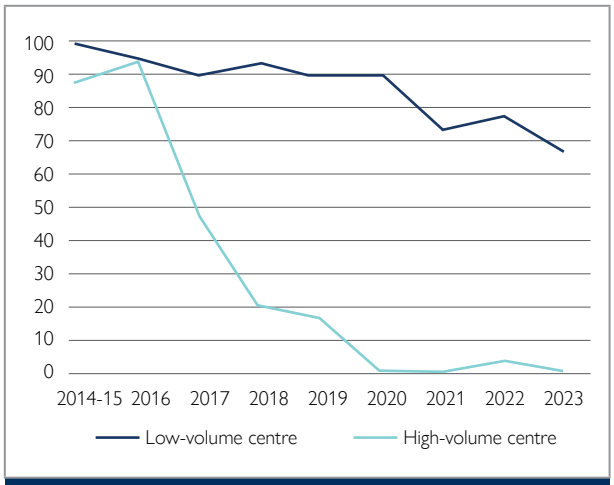



**FIGURE 12: Trends in transoesophageal echocardiography use according to total transcatheter aortic valve implantation rates for the centres.**
*Note: Low-volume means < 200 transcatheter aortic valve implantations performed over the study period; high-volume means > 200 transcatheter aortic valve implantations performed over the study period.*

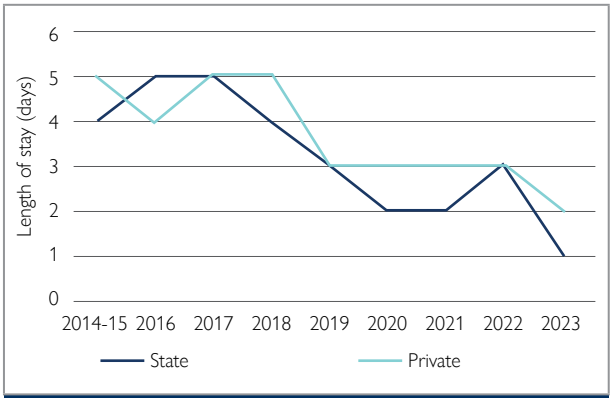



**FIGURE 13: Trends of median length of hospital stay for transcatheter aortic valve implantation patients in public and private hospitals.**

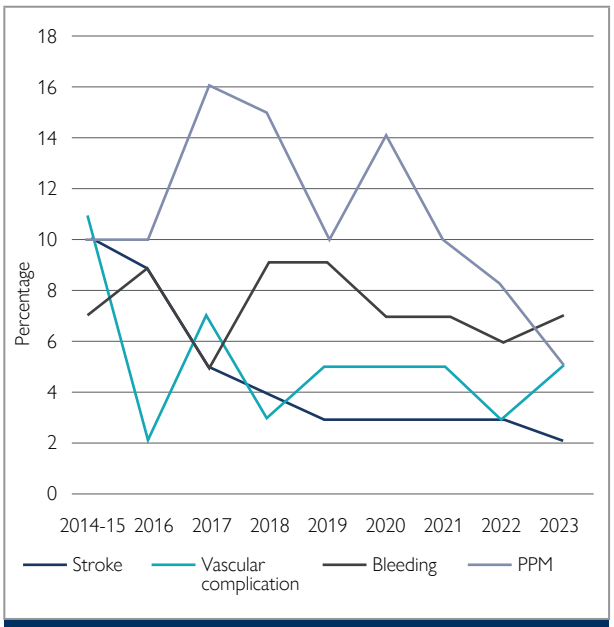



**FIGURE 14: Trends of major adverse outcomes.**
*Note: Bleeding as per VARC-2 (Valve Academic Research Consortium-2) definition. PPM: permanent pacemaker.*

EuroSCORE II declined significantly from 4.8% (IQR 3.2–8.6) in 2014 to 3.5% (IQR 2.3–6.9) in 2023 ($p < 0.001$).

### Procedural characteristics

There was a higher overall utilisation of BEVs, which remained unchanged (Figure 8). The ratio between BEVs and self-expanding valves remained constant at 0.87 to 1. There was no change in the rate of transfemoral access (92%, IQR 87–95) (Figure 9). Alternate access sites for those with poor femoral access included transaortic, transapical, subclavian, and carotid. In cases utilising transfemoral access, there was a temporal increase in the use of percutaneous vascular closure devices (12% in 2014 vs. 90% in 2023; $p < 0.001$), accompanied by a concurrent reduction in surgical closure (Figure 10). There was a statistically significant increase in the use of CS for TAVI (0% in 2014/15 vs. 70% in 2023; $p < 0.001$). There was a notable reduction in the utilisation of intra-procedural transoesophageal echocardiography (TOE) (Figure 11). Another notable reduction was TOE utilisation in centres with higher TAVI rates; centres that performed > 200 cases over the study period had a significant decline in TOE use (100% in 2014 vs. to 1% in 2023; $p < 0.001$), while centres that performed < 200 cases had consistent TOE use > 60% (Figure 12).

### Clinical outcomes

The hospital stay length in both the public and private sectors shortened throughout the study period (4 days in 2014/15 vs. 1 day in 2023) (Figure 13). During the study period, there was a significant decline in peri-procedural complications. The rate of peri-procedural strokes reduced from 10% in 2014/15 to 2% in 2023 ($p < 0.0001$) (Figure 14). The need for permanent pacing declined, possibly reflecting improvements in the implantation technique of self-expanding valves (Figure 14). Kaplan–Meier survival curves showed a gradual decrease in mortality risk over the years ($p = 0.0344$) (Figure 15).

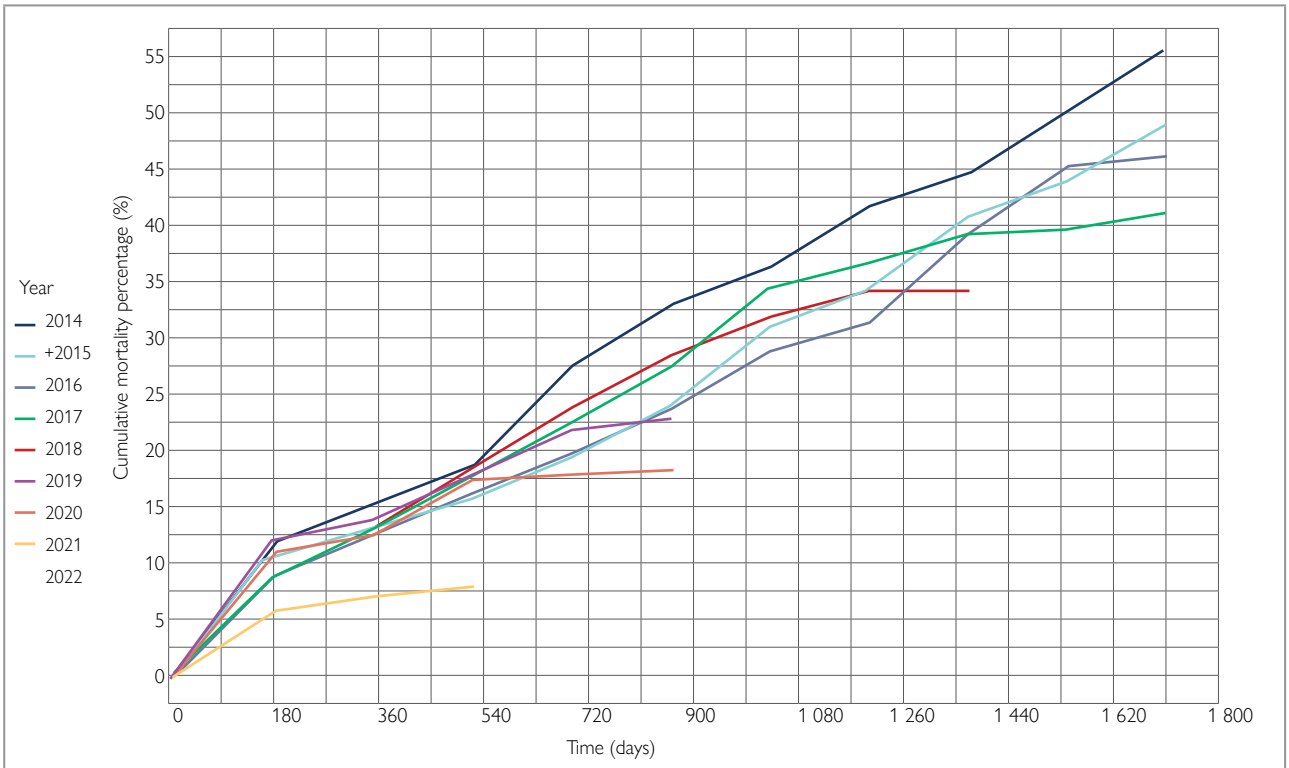
## DISCUSSION

The global adoption of TAVI has demonstrated exponential growth, particularly in high-income countries, whereas UMICs have seen a more gradual uptake. Registry data reveal striking disparities: Germany's procedural volume increased from 637 cases in 2008 to 13 264 in 2014, while the United Kingdom (UK) TAVI registry reported growth from 366 procedures in 2008 to 1 271 in 2012.[14,15] Conversely, UMICs such as Brazil documented only 661 cases over a decade (95 in 2012–2017 vs. 566 in 2018–2019), while the SHARE-TAVI registry recorded 3 931 procedures from 2014 to 2023, demonstrating steady, though comparatively slower, expansion.[19,20] This differential adoption pattern reflects the complex interplay of economic, infrastructural, and healthcare system factors that characterise procedural differences in resource-variable settings.

Consistent with global trends, the SHARE-TAVI registry reflects a transition from treating only high-risk patients to treating those with intermediate and low risk.[18,21] This paradigm shift, particularly evident after 2015, aligns with expanding indications supported by international guidelines and registry data.[22] The median patient age of 81 years corresponds closely with European registries, suggesting comparable patient demographics despite differing healthcare landscapes.[9,10,17] The observed improvement in baseline risk may be multifactorial, potentially reflecting broader patient selection criteria, as recommended by guidelines based on large, randomised trials; however, further research is needed to elucidate these relationships in an African context.[26]

In South Africa, TAVI utilisation remains constrained by financial and systemic barriers. Procedural costs pose a significant burden, particularly in the public sector, where 86% of the population seeks healthcare.[23] Private sector adoption, while more robust, remains hampered by insurance coverage limitations, particularly regarding valve prosthesis reimbursement. This has created a growing disparity in access, with public hospital procedures declining from one-third of total cases to just 10% by 2023 in our study. The resulting inequities lead to prolonged waiting times in the public sector, potentially compromising clinical outcomes and underscoring the need for healthcare system interventions to ensure equitable access to TAVI in the future.

Notable procedural refinements were observed in the SHARE-TAVI registry, paralleling global trends. High-volume centres demonstrated particularly notable progress, reducing TOE utilisation from > 80% to < 5% while increasing CS adoption from 0% to 70%. Transfemoral access was predominant in our registry (> 90% of cases), with percutaneous closure replacing surgical methods (67% in 2014/15 vs. 10% by 2020), which correlated with reduced bleeding complications according to VARC-2 (Valve Academic Research Consortium-2) criteria. Alternative access routes remained uncommon, likely due to low




| Days | 0 | 180 | 360 | 540 | 720 | 900 | 1 080 | 1 260 | 1 440 | 1 620 | 1 800 |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 2014/15 | 103 | 91 | 88 | 84 | 75 | 69 | 66 | 63 | 60 | 58 | 52 |
| 2016 | 164 | 146 | 142 | 138 | 132 | 125 | 113 | 108 | 97 | 92 | 84 |
| 2017 | 206 | 188 | 181 | 173 | 165 | 157 | 147 | 141 | 125 | 113 | 111 |
| 2018 | 232 | 206 | 202 | 191 | 180 | 168 | 152 | 147 | 141 | 140 | 137 |
| 2019 | 301 | 275 | 263 | 246 | 230 | 216 | 205 | 199 | 198 | | |
| 2020 | 251 | 221 | 217 | 208 | 197 | 195 | | | | | |
| 2021 | 329 | 294 | 287 | 272 | 270 | 269 | | | | | |
| 2022 | 540 | 509 | 502 | 497 | | | | | | | |

**FIGURE 15: Mortality at 5 years; Kaplan–Meier curves for all-cause mortality between 2014 and 2022.**

procedural volumes and technical complexity.[24] Transapical access has been phased out due to poorer outcomes. The need for pacemaker implantation was 10% in 2014/15 compared with 11.8% in the United States TAVI registry and 12.4% in the UK registry in a similar time frame.[8,10] The pacemaker implantation rate then fluctuated and stands at 5% (2023), which is comparable to other registries.[18]

The length of hospital stay declined for both private and public sector patients: 5 days in the early study period versus 1 day in recent years in public hospitals, and 6 days versus 2 days in private hospitals. The overall length of stay was similar to the findings in the UK TAVI, FRANCE 2, FRANCE TAVI, and Society of Thoracic Surgeons/American College of Cardiology Transcatheter Valve Therapy (STS/ACC TVT) registries.[8,25] Longer hospital stays in the private sector were likely due to multidisciplinary discharge protocols, including physiotherapy, which is not freely available in the public sector. This decline in length of stay will positively affect procedure costs.

There was a significant temporal trend in survival, showing improved long-term survival with each consecutive calendar year. The mortality reduction likely reflects multiple synergistic factors, including improved patient selection, with more low-to-intermediate risk patients with fewer comorbidities, and it highlights the safety of the procedure in this population. The data reported here help inform our local heart teams' decision-making and patient counselling.

## CONCLUSION

The findings of the SHARE-TAVI registry provide compelling evidence of a successful TAVI implementation in a UMIC, despite continued challenges of healthcare inequity. Temporal trends mirrored those in first-world countries, and refinements in the procedure over time have led to shorter hospital stays, reduced peri-procedural strokes, and lower overall mortality risk.

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PATIENTS HOSPITALISED WITH ACUTE HEART FAILURE

# Characteristics and outcomes of patients hospitalised with acute heart failure at a tertiary hospital in South Africa

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## ABSTRACT

**Aims:** Acute heart failure (AHF) remains a major public health challenge in sub-Saharan Africa, yet contemporary data on its clinical characteristics and management outcomes are limited. This study aims to characterise the epidemiology, treatment patterns, and clinical outcomes of AHF patients in a South African tertiary care setting in the era of modern heart failure (HF) therapy.

**Methods:** We conducted a retrospective study of 339 AHF admissions at Tygerberg Hospital (TBH), Cape Town, during 2022. Comprehensive data, including demographics, clinical characteristics, investigations (echocardiography, coronary angiography), treatment regimens, and outcomes, were analysed. Patients were identified using the International Classification of Diseases, Tenth Revision (ICD-10) codes for HF and cardiomyopathy.

**Results:** The cohort (mean age 53 ± 15.4 years, 51.9% male) demonstrated a high burden of non-ischaemic cardiomyopathy with HF with reduced ejection fraction (HFrEF) predominance (91%). Comorbidities were highly prevalent (74% hypertension, 43.4% diabetes). While conventional guideline-directed medical therapy (GDMT) utilisation was robust (88.7% beta blocker [BB], 87.5% angiotensin-converting enzyme inhibitor [ACEi]/ angiotensin receptor blocker [ARB]), novel therapies were markedly underutilised (3.9% sodium-glucose cotransporter 2 inhibitor [SGLT2i], 1.3% angiotensin receptor-neprilysin inhibitor [ARNi]). Only 42.9% of eligible patients for cardiac resynchronisation therapy (CRT) received implants. Clinical outcomes included rates of 3.9% in-hospital mortality, 27.7% 2-year case fatality, and 44.3% 30-day re-admission.

**Conclusions:** This study reveals a distinct AHF phenotype in South Africa, characterised by younger patients with a predominant non-ischaemic aetiology and high comorbidity burden. Despite adequate conventional GDMT implementation, significant therapeutic gaps persist in advanced therapies. The substantial re-admission burden highlights critical health system challenges, emphasising the urgent need for healthcare policy reforms and optimised care pathways in resource-limited settings.

**Keywords:** acute heart failure, sub-Saharan Africa, dilated cardiomyopathy, guideline-directed medical therapy, cardiac resynchronisation therapy.
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## INTRODUCTION

The incidence of HF has nearly doubled over the past few decades, rising from 33.5 million cases in 1990 to 64.3 million in 2017, with projections indicating continued growth.[1] AHF requires hospitalisation, often on an emergent basis, which is a major driver of healthcare costs.[2,11] Each episode of AHF hospitalisation is associated with an increased mortality risk and a decline in quality of life, making the prevention of recurrent hospital admissions a key priority in HF management.[3]

Despite the growing burden of HF, a paucity of data remains regarding the characteristics and management of patients admitted with AHF in sub-Saharan Africa. Although several HF registries exist, they primarily represent populations from high-income countries, whose ethnic, racial, and socio-economic characteristics differ significantly from those in Africa.[4-6] AHF populations in high-income countries are older, predominantly male, and have a higher prevalence of ischaemic heart disease.[7,8] Limited evidence suggests that the aetiology of AHF in sub-Saharan Africa is distinct, with a higher prevalence of peripartum cardiomyopathy, idiopathic dilated cardiomyopathy (DCM), and HF associated with human immunodeficiency virus (HIV) infection.[9]

Rheumatic heart disease is also an important cause of HF, with an annual incidence of 24.7 per 100 000 South Africans.[10] Additionally, sub-Saharan Africa is undergoing an epidemiological transition, with an increasing burden of noncommunicable diseases (NCD), particularly cardiovascular disease, which now accounts for 37% of NCD-related deaths in the region.[5] It is not well described whether there has been an increase in the contribution of ischaemic heart disease as a cause for HF on the African continent.

This study focused on a population of AHF patients managed with GDMT and device therapy. Given the limited data on the epidemiology, clinical course, and management of AHF in South Africa, there is an urgent need to better characterise these patients to improve care and outcomes. This study aimed to evaluate the aetiology and outcomes of patients admitted with AHF. It sought to characterise the initial emergency department assessment and subsequent inpatient management, and to identify key patient characteristics associated with adverse outcomes. Additionally, the study assessed treatment strategies at discharge, with a particular focus on GDMT and device therapy use in patients with chronic HF. By addressing these knowledge gaps, the study is intended to provide valuable insights to inform future healthcare policies and optimise HF management strategies in South Africa.

## METHODS

TBH is a 1 384-bed tertiary academic hospital serving a population of over 2.6 million people in Cape Town, South Africa.[12] The study was a single-centre, retrospective, hospital-based study of patients admitted with acute decompensated HF to TBH over 1 year (1 January to 31 December 2022). All patient records were obtained from TBH's comprehensive electronic database, including clinical patient records, ICD-10-coded discharge diagnoses, and death registrations. For each patient meeting the inclusion criteria, clinical notes, laboratory results, echocardiographic images, and procedure reports were systematically reviewed. A follow-up review of patient records was conducted 2 years post-discharge to determine the case fatality rate of the study population.

Patients were included in this audit based on admissions with discharge diagnoses of AHF, as determined by ICD-10 coding. Patients with a diagnosis of DCM (I42.0), unspecified HF (I50.9), unspecified left ventricular failure (I50.1), systolic (congestive) HF (I50.2), unspecified cardiomyopathy (I42.9), and acute systolic (congestive) HF (I50.21) were included. AHF was defined as either new-onset HF or decompensation of chronic, established HF with symptoms sufficient to warrant hospitalisation.[13] Patients aged ≥ 18 years were included. Patients with HF as a complication of acute coronary syndrome, valvular heart disease, pericardial disease, congenital heart disease, primary pulmonary hypertension, and cor pulmonale were excluded from the study. The broad exclusion criteria were intended to identify a representative population of patients with AHF who are primarily managed medically, to assess current treatment practices in a South African setting.

The cause of HF was identified based on the information obtained from history, clinical examination, and echocardiography. Echocardiographic records were obtained from an internal database of all transthoracic echocardiograms performed at the Division of Cardiology at TBH. All echocardiograms were performed by qualified healthcare professionals in accordance with contemporary guidelines.[14] The characteristics of our AHF population were compared with previous AHF studies conducted in Africa, as well as high-income countries.

### Ethical considerations

This study was approved by the Health Research Ethics Committee of Stellenbosch University (reference: S22/11/259) and conducted in accordance with the 2013 Declaration of Helsinki. A waiver of individual consent was obtained from the ethics committee to include data from patient records in this retrospective study.

### Statistical analysis

Statistical analysis was performed with IBM SPSS Statistics version 29 for Windows. Descriptive statistics, including means, medians, proportions, and standard deviations, were used. Categorical data were analysed using the chi-squared test with *p*-values. A $p$-value < 0.05 was considered statistically significant. All continuous variables were tested for normal distribution using a histogram for visualisation. Normally distributed data were reported as mean and standard deviation. All statistical analyses were performed in consultation with a statistician.

## RESULTS

A total of 339 patients were admitted with AHF between 1 January and 31 December 2022 (Figure 1). The baseline characteristics of the cohort are summarised in Table I. The study population was evenly distributed between males (51.9%, $n$ = 176) and females (48.1%, $n$ = 163). Most AHF admissions (91%, $n$ = 311) had HFrEF, and the analysis mostly described this HF phenotype. The mean age of patients with HFrEF was 53 ± 15.4 years, with 74.3% ($n$ = 252) of this population aged < 65. The mean age of patients with HF with mildly reduced or preserved ejection fraction (HFmrEF or HFpEF) was 60.9 ± 16.7 years. The mean systolic blood pressure on admission was 137 ± 37.1 mmHg in the entire HF population, with a higher mean systolic blood pressure of 185.9 ± 35.1 mmHg in the HFmrEF/HFpEF groups.

Half of the patients (49.6%, $n$ = 168) had ≥ 2 comorbidities. A large proportion of the HFrEF population (74%, $n$ = 251) had comorbid hypertension. Just under half of the patients (43.4%, $n$ = 147) had diabetes mellitus, with a mean HbA1C of 8.8 ± 2.7%. Regarding recreational substance use, 36.6% of the population ($n$ = 124) were regular cigarette smokers, and 6.5% ($n$ = 22) self-reported methamphetamine use. Of the patients, 89 (26.3%) had chronic kidney disease, with a mean estimated

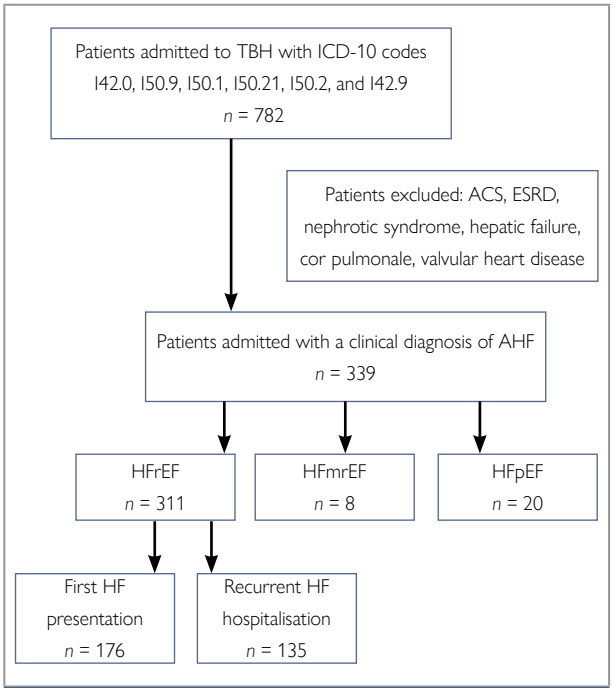


**FIGURE 1: Recruitment of study participants.**

*ACS: acute coronary syndrome, AHF: acute heart failure, ESRD: end-stage renal disease, HF: heart failure, HFmrEF: heart failure with mildly reduced ejection fraction (left ventricular ejection fraction [LVEF] 40–49%)[14], HFpEF: heart failure with preserved ejection fraction (LVEF ≥ 50%)[15], HFrEF: heart failure with reduced ejection fraction (LVEF < 40%)[14], ICD-10: International Classification of Diseases, Tenth Revision, TBH: Tygerberg Hospital.*

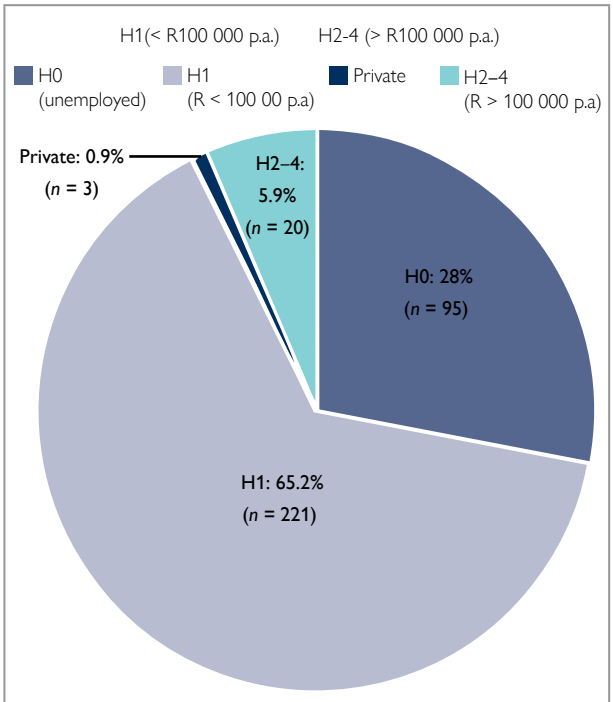


**FIGURE 2: Income status of the population with heart failure with reduced ejection fraction.**

*p.a.: per annum.*

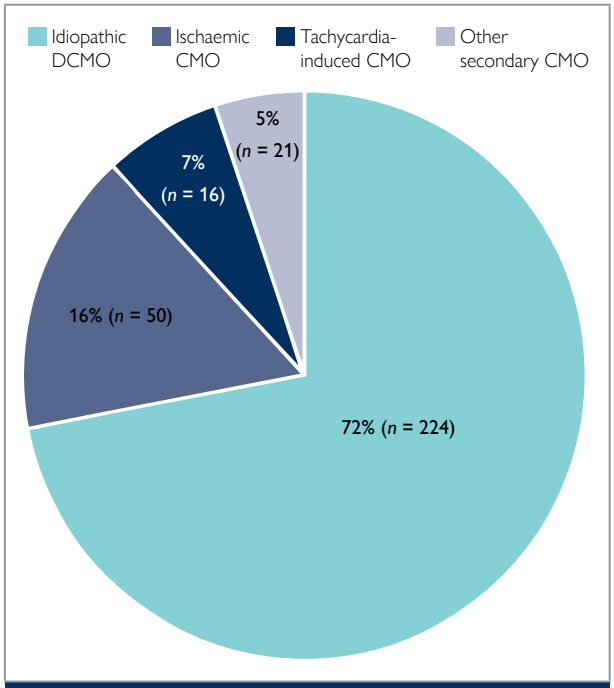


**FIGURE 3: Aetiology of heart failure with reduced ejection fraction based on clinical assessment and echocardiography.**

*CM: cardiomyopathy, DCM: dilated cardiomyopathy.*

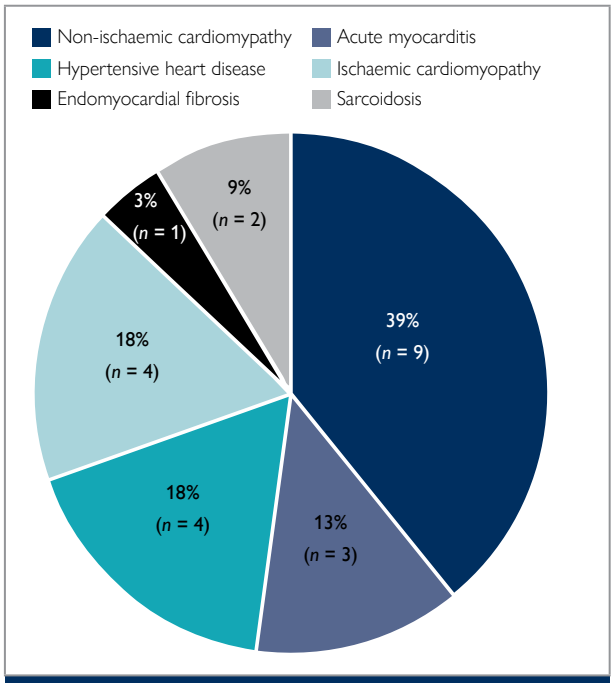


**FIGURE 4: Diagnoses of patients who underwent cardiac magnetic resonance imaging.**

glomerular filtration rate of 34.01 ± 13.08. At the time of the study, 8% of the cohort (*n* = 27) were living with HIV, with a mean CD4 count of 353 ± 294.7. Among them, 3.5% (*n* = 12) had a CD4 count < 200, consistent with advanced HIV.

**TABLE I: Baseline characteristics of acute heart failure admissions.**

| ***n* = 339** | | **HFrEF** | **HFmrEF** | **HFpEF** |
|---|---|---|---|---|
| *n* (%) | | 311 (91.7) | 8 (2.35) | 20 (5.9) |
| **Male, *n* (%)** | 176 (51.9) | 168 (54.1) | 3 (37.5) | 6 (30) |
| **Age, mean (SD)** | 53 (15.4) | 53.4 (15.1) | 54.25 (21.2) | 63.5 (14.3) |
| < 40, ***n*** (%) | 65 (19.2) | 62 (19.9) | 2 (25) | 1 (5) |
| ≥ 40–64, *n* (%) | 187 (55.2) | 172 (55.3) | 4 (50) | 11 (55) |
| ≥ 65, n (%) | 87 (25.6) | 77 (24.8) | 2 (25) | 8 (40) |
| **Systolic BP, mean (SD)** | 137 (37.1) | 133 (34.1) | 168 (32.2) | 193 (3.1) |
| **Diastolic BP, mean (SD)** | 84 (23.0) | 84 (22.7) | 85 (26.2) | 93 (25.4) |
| **HR, mean (SD)** | 96.2 (24.1) | 96.3 (24.1) | 92.3 (18.4) | 81.3 (13.9) |
| **Hypertension, *n* (%)** | 251 (74.0) | 224 (72.0) | 7 (87.5) | 20 (100) |
| **Diabetes mellitus, *n* (%)** | 147 (43.4) | 128 (41.2) | 3 (37.5) | 16 (80) |
| HbA1C, mean (SD) | 8.9 (2.6) | 8.8 (2.7) | 12.8 (2.8) | 8.9 (1.6) |
| HbA1C < 10, ***n*** (%) | 104 (70.7) | 91 (71.1) | 0 (0) | 10 (62.5) |
| HbA1C >10, ***n*** (%) | 41 (27.9) | 36 (28.1) | 2 (66.7) | 6 (37.5) |
| Unknown HbA1C, ***n*** (%) | 2 (1.4) | 1 (0.8) | 1 (33.3) | 0 (0) |
| **Renal insufficiency, *n* (%)** | 89 (26.3) | 80 (25.7) | 1 (12.5) | 8 (40) |
| eGFR (CKD-EPI), mean (SD) | 34.01 (13.8) | 35.78 (14.8) | 40 (0) | 21.6 (9.7) |
| eFGR ≤ 30, ***n*** (%) | 35 (39.3) | 28 (35) | 0 (0) | 7 (87.5) |
| eGFR 31–60, ***n*** (%) | 54 (60.7) | 52 (65) | 1 (100) | 1 (12.5) |
| **Haemoglobin, mean (SD)** | 12.6 (2.4) | 12.4 (2.3) | 12.1 (3.0) | 10.3 (2.3) |
| **PLHIV, *n* (%)** | 27 (8.0) | 26 (8.4) | 1 (12.5) | 0 (0) |
| **CD4 count, mean (SD)** | 353 (295) | 356 (287) | N/A | N/A |
| **Atrial fibrillation, *n* (%)** | 57 (16.8) | 56 (18.0) | 0 (0.0) | 1 (5.0) |

*BP: blood pressure, CKD-EPI: Chronic Kidney Disease Epidemiology Collaboration, eGFR: estimated glomerular filtration rate, HFmrEF: heart failure with mildly reduced ejection fraction, HFpEF: heart failure with preserved ejection fraction, HFrEF: heart failure with reduced ejection fraction, HR: heart rate, N/A: not applicable, PLHIV: people living with human immunodeficiency virus, SD: standard deviation.*

In the HFrEF population, electrocardiogram-documented atrial fibrillation (AF) was identified in 18% (*n* = 56) and atrial flutter in 4.5% (*n* = 14). Nine of these patients underwent catheter ablation (5 for AF and 4 for typical atrial flutter). Of the patients with atrial arrhythmias (*n* = 70), 40% (*n* = 28) had at least 1 electrical cardioversion, 5.7% (*n* = 4) were on digoxin, and 4.3% (*n* = 3) were on amiodarone at discharge.

The income distribution of the study population is depicted in Figure 2. Among patients with HFrEF, 5.9% (*n* = 20) had an annual household income > R100 000 (United States $5 430 at the time of submission), while 0.9% (*n* = 3) had private medical insurance. However, 93.2% (*n* = 316) were either unemployed or had an annual household income < R100 000.

The most common cause of HF in HFrEF patients was idiopathic DCM, diagnosed in 72% (*n* = 224) (Figure 3). In the HFrEF cohort, 16% (*n* = 50) had ischaemic cardiomyopathy, 7% (*n* = 16) had a tachycardia-induced cardiomyopathy, and 5% (*n* = 21) had other secondary causes for cardiomyopathy, including 9 patients with peripartum cardiomyopathy, 1 with cardiac sarcoidosis, 5 with a history of previous exposure to cardiotoxic chemotherapy agents, 1 with confirmed endomyocardial fibrosis, and 1 with Takotsubo cardiomyopathy.

Twenty-three patients with HFrEF underwent cardiac magnetic resonance imaging after being assessed as having atypical cardiomyopathy (Figure 4). Magnetic resonance imaging diagnosis of non-ischaemic cardiomyopathy or acute myocarditis was done in over half of these patients. There were 4 patients with hypertensive heart disease and 4 patients with ischaemic cardiomyopathy.

Iron studies were performed in 33.6% (114/339) of the AHF population. There was evidence of either absolute or relative iron deficiency in 80.7% of those with iron studies (92/114).[22,23] There were 154 patients (45.4%) with evidence of anaemia on laboratory investigations: 54.5% females (*n* = 84) and 45.5% males (*n* = 70). Iron studies were performed in 52.6% (81/154) of the anaemic patients. Of these, 32.1% (26/81) had evidence of iron deficiency. Eleven patients were on oral iron replacement, and only 2 were receiving intravenous iron replacement.

Echocardiographic reports were available for 97% (*n* = 303) of the patients with HFrEF (Table II). Eight patients did not have echocardiographic data available, and 6 demised before echocardiography was performed. In patients with HFrEF, 79.2% (*n* = 240) had a left ventricular ejection fraction (LVEF) < 30%. The mean left ventricular end-diastolic dimension (LVEDD) was

**TABLE II: Baseline echocardiography in heart failure with reduced ejection fraction, acute heart failure admissions.**

| **Echocardiography available, *n* (%)** | 303/311 (97.4) |
|---|---|
| LVEF, mean (SD) | 23.8 (8.2) |
| LVEF ≤ 20%, ***n*** (%) | 126 (41.6) |
| LVEF 21–30%, ***n*** (%) | 114 (37.6) |
| LVEF 31–40%, ***n*** (%) | 63 (20.8) |
| LVEDD (mm), mean (SD) | 60.54 (9.0) |
| LVEDD < 55 mm, ***n*** (%) | 124 (40.9) |
| LVEDD 56–64 mm, ***n*** (%) | 101 (33.4) |
| LVEDD ≥ 65 mm, ***n*** (%) | 78 (25.7) |
| Severe secondary MR, ***n*** (%) | 8 (2.6) |
| LA diameter, mm (SD) | 42.9 (7.1) |
| IVSD (mm), mean (SD) | 8.0 (2.0) |
| IVSD < 8 mm, ***n*** (%) | 123 (40.6) |
| IVSD 8–9 mm, ***n*** (%) | 106 (34.9) |
| IVSD ≥ 10 mm, ***n*** (%) | 74 (24.4) |

*IVSD: interventricular septal thickness in diastole, LA: left atrium, LVEDD: left ventricular end-diastolic diameter, LVEF: left ventricular ejection fraction, MR: mitral regurgitation, SD: standard deviation.*

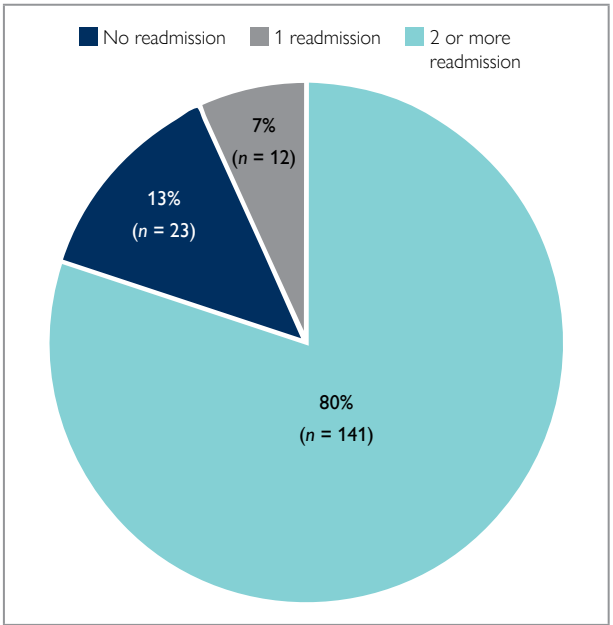


**FIGURE 5: Hospital re-admissions after first heart failure diagnosis.**

also elevated at 60.54 ± 9 mm. The mean left ventricular wall thickness was 8.2 ± 2 mm, and 24.4% (*n* = 74) measured ≥ 10 mm. Eight patients (2.6%) had severe secondary mitral regurgitation.

A total of 39.9% (*n* = 124) HFrEF patients underwent coronary angiography. Significant left main coronary or multivessel coronary artery disease was identified in 7% (*n* = 22), with 3 patients having undergone previous coronary artery bypass grafting. Of these patients, 16 received percutaneous revascularisation, and the remaining 3 were managed medically.

At the time of discharge, 87.5% (*n* = 272) of the HFrEF cohort were on an ACEi or ARB, 88.7% (*n* = 276) on a BB, and 56.3% (*n* = 175) on a mineralocorticoid receptor antagonist. Only 3.9% (*n* = 12) were on an SGLT2i, and 1.3% (*n* = 4) on an ARNi. SGLT2is and ARNis were purchased out of pocket by all patients.

A total of 11.6% of patients (*n* = 36) with HFrEF met the eligibility criteria for CRT based on electrocardiographic and echocardiographic findings. Among them, 77.8% (28/36) had idiopathic DCM, and 22.2% (8/36) had ischaemic cardiomyopathy. The mean QRS duration was 154 ± 20.3 ms; 25% (*n* = 7) had 130–149 ms, and 75% (*n* = 21) had ≥ 150 ms. Of the patients with idiopathic DCM, 75% (21/28) had a Class I indication for CRT per current guidelines.[16] Of those with a Class I indication, 42.9% (9/21) went on to receive a CRT implant.

The in-hospital mortality of the HFrEF patients was 3.9%. The mean age of the 12 patients with in-hospital mortality was 62 ± 15.3 years. There was better survival in patients who had HFmrEF/HFpEF, as all patients in this group were discharged. According to provincial electronic clinical records, the case fatality rate of the entire HF population was 27.7% (*n* = 92/339) at 2 years. The mean hospital stay was 6 ± 5 days in patients with HFrEF, and 28.6% (*n* = 89) had hospitalisations lasting ≥ 7 days. The HFpEF patients had a mean hospital stay of 5 ± 2 days.

Of the HFrEF patients, 51 (16.4%) required either non-invasive ventilatory support or intravenous inotropes, which were administered in the emergency department upon admission or in a high-care unit. No patients with AHF were admitted to the medical intensive care unit for invasive ventilatory support. TBH's medical intensive care unit policy dictates that patients with a LVEF < 40% do not qualify for admission, due to the limited availability of beds for invasive ventilatory support.

In the HFrEF population, 56.6% (*n* = 176) had a new HF diagnosis during their admission in 2022. Of these patients, 19.9% (35/176) had ≥ 1 all-cause re-admission in 2022 (Figure 5). The time to first re-admission was < 1 month in 44.3% (78/176).

Table III compares the Tygerberg cohort with previous HF registries and retrospective studies. Like other African AHF cohorts, this population was younger than those in high-income countries. Additionally, patients in this cohort had more advanced disease at the time of admission, as reflected by a lower LVEF and higher LVEDD compared with prior studies. However, the discharge prescription rate for GDMT was higher than in previous AHF studies.

## DISCUSSION

This study highlights the distinct characteristics of patients with AHF in sub-Saharan Africa, with notable patterns that differ from those observed in previous local and international studies.

**TABLE III: Comparison of patients in published acute heart failure studies and the Tygerberg Hospital cohort.**

| | **Current study (*n* = 339)** | Szymanski, et al.,[9] South Africa (*n* = 119) | THESUS-HF, Africa[4] (*n* = 1 006) | ADHERE, United States[7] (*n* = 105 388) | EHFS II, Europe[8] (*n* = 3 580) |
|---|---|---|---|---|---|
| Age, mean (SD) | **53.0 (15.4)** | 49.9 (16.3) | 52.3 (18.3) | 72.4 (14) | 69.9 (12.5) |
| Females (%) | **48.1** | 58.0 | 50.6 | 52.0 | 38.7 |
| LVEF (%), mean (SD) | **26.2 (11.3)** | 34.1 (16.9) | 39.5 (16.5) | 34.4 (16.1) | 38.0 (15.0) |
| LVEDD (mm), mean (SD) | **60.5 (9.0)** | N/A | 57.7 (11.6) | N/A | 58.0 (7.0) |
| Hypertension (%) | **74.0** | 48.7 | 55.5 | 73.0 | 62.5 |
| Systolic BP (mmHg), mean (SD) | **137.0 (37.1)** | 134.6 (33.2) | 130.4 (33.5) | 144 (13.2) | 135.0 (25.0) |
| Diabetes mellitus (%) | **43.4** | 21.8 | 11.4 | 44.0 | 32.8 |
| Renal insufficiency (%) | **26.0** | N/A | 7.7 | 30.0 | 16.8 |
| Atrial fibrillation/flutter (%) | **21.2** | 5.0 | 18.3 | 31.0 | 38.7 |
| In-hospital mortality (%) | **3.9** | 8.4 | 4.2 | 4.0 | 6.7 |
| Length of stay (days) | **6.0** | 9.0 | 9.2 | 4.3 | 9.0 |
| ACEi/ARB at discharge (%) | **87.1** | 73.0 | 80.0 | 66.1 | 80.2 |
| BB at discharge (%) | **88.7** | 42.7 | 30.0 | N/A | 61.0 |
| MRA at discharge (%) | **56.6** | 26.1 | 75.0 | N/A | 47.5 |

*ACEi: angiotensin-converting enzyme inhibitor, ADHERE: Acute Decompensated Heart Failure National Registry, ARB: angiotensin receptor blocker, BB: beta blocker, BP: blood pressure, EHFS II: EuroHeart Failure Survey II, LVEDD: left ventricular end-diastolic diameter, LVEF: left ventricular ejection fraction, MRA: mineralocorticoid receptor antagonist, N/A: not applicable, SD: standard deviation, THESUS-HF: The Sub-Saharan Africa Survey of Heart Failure.*

This cohort is significantly younger than European and American AHF cohorts.[7,8] A population with mortality peaking at productive age profoundly impacts a group of patients for whom persistent HF symptoms and recurrent HF hospitalisations may result in significant income loss.

Idiopathic DCM is the most common cause of HFrEF in this study, as in the THESUS-HF (The Sub-Saharan Africa Survey of Heart Failure) registry.[4] Although this study did not assess the genetic contribution to DCM, previous local data suggest an equal distribution of familial, idiopathic, and secondary DCM in African populations.[17] Further genetic studies are warranted to explore the hereditary aspects of DCM and identify potential therapeutic targets.

While ischaemic heart disease remains the predominant cause of HF in high-income countries, its contribution to HF in Africa remains relatively low, with 9.7% of our cohort diagnosed with ischemic cardiomyopathy. This aligns with previous African studies, which reported ischaemic cardiomyopathy rates ranging from 0.4% to 9%.[9,21]

HF re-admission rates within 30 days were strikingly high (44.3%) compared with the United States (24.8%).[7] This study illustrates a vulnerable group of patients with lower LVEFs on admission compared with prior studies. Despite lower LVEF on admission, this study population had a shorter length of stay than in high-income countries. Due to constant limitations of hospital beds in South African public hospitals, patients with AHF are often discharged prematurely with inadequate post-discharge treatment plans.

The study found an even distribution of males and females in the HFrEF group, in contrast to predominantly male European HF cohorts.[8] Despite shared risk factors, previous studies suggest that males are more prone to developing HFrEF, whereas females tend to develop HFpEF.[17] This finding suggests that the interplay between sex-specific risk factors and HF pathophysiology may differ in African populations. Further research is needed to explore the sex-based disparities in Africa's HF phenotypes.

This AHF cohort primarily comprised patients with HFrEF due to idiopathic DCM, with hypertension as a frequent comorbidity. The high prevalence of hypertension in this study reflects local epidemiological data, which indicate that 44.1% of individuals < 50 in South Africa have hypertension.[18,19,20] Similarly, European HF registries report hypertension in 70–91% of HF patients.[8]

The interplay between AHF and renal impairment is complex and associated with adverse outcomes in AHF patients.[1] Our cohort showed a higher prevalence of renal insufficiency (26%) than historically reported in African populations (7.7%).[4] This finding aligns more closely with data from AHF populations in high-income countries, likely reflecting the increasing global prevalence of hypertension, diabetes, and chronic kidney disease.[5,7,8] Cardiorenal syndrome also represents an important contributor to renal impairment in advanced HF.

This study showed that 8.7% of the HF population were people living with HIV, a prevalence similar to The Heart of Soweto Study (9.7%).[21] Additionally, HIV was found to be the direct cause of HF in 2.6% of cases, consistent with the THESUS-HF study.[4] The interplay between HIV and HF remains complex, with HIV-associated cardiomyopathy being a recognised contributor to the HF burden in sub-Saharan Africa.

Anaemia was highly prevalent in the AHF cohort (45.4%) compared with European HF registries (14%).[8] Furthermore, routine screening for iron deficiency was not performed in more than half of the HFrEF population. Given that iron deficiency is an independent predictor of poor HF outcomes, routine screening and treatment with intravenous iron therapy could offer a potential therapeutic target to improve functional capacity and reduce HF hospitalisations.[22,23,24]

The study found a higher incidence of AF (16.8%) than previously reported rates in African hospital-based studies (4.6–10.6%).[25,26] This aligns with the global trend of increasing AF incidence.[27] The presence of AF in HF patients is associated with worse outcomes due to increased thromboembolic risk, rapid ventricular rates, and worsened cardiac function.

Obstructive sleep apnoea is highly prevalent among patients with HF, with a reported incidence of 22.8% in South Africa.[30,31] However, access to continuous positive airway pressure therapy remains limited due to its prohibitive cost. In this study, there were limited data on the true prevalence of obstructive sleep apnoea and how it may be linked to our AHF population.

Compared with prior studies, our study showed that the overall prescription of GDMT at hospital discharge for HF improv -ed.[4,7-9] There was an improvement in the prescription of BBs at discharge (42.7% in a previous local HF registry vs. 88.7% in this study).[9] ARNis and SGLT2is remain widely inaccessible in the South African public sector due to financial constraints, prompting hospital policies that limit access despite their proven mortality benefits. Many patients in Africa cannot afford these life-saving therapies[28]. The income distribution of our HF cohort likely reflects the general South African population, in which a substantial proportion lives below the poverty line. To bridge this gap, urgent policy changes are needed to improve the accessibility and affordability of these medications within public healthcare systems. Hydralazine and nitrates were not utilised in our AHF population, despite their ongoing presence in contemporary HF guidelines.

An 11.6% eligibility for CRT was identified in the HFrEF population, aligned with a previous study indicating that 10–15% of AHF patients were eligible for CRT.[29] However, there was a lower uptake of CRT implantation in eligible patients compared with HF registries in the First World (42.9% vs. 58–66%).[29] The lower CRT implantation uptake may have multiple explanations in this AHF population. First, patients hospitalised with AHF may not yet be optimised on HF therapy, and hence not referred for CRT consideration at the first hospitalisation. This is reflected by the fact that over half of our AHF cohort had de novo AHF. Second, there may have been a proportion of patients who were eligible for CRT based on electrocardiographic and echocardiographic criteria but had significant comorbidities or advanced HF, for whom palliative care may have been more appropriate as treatment in a resource-limited setting.

### Limitations

This is a retrospective observational study; therefore, its main limitation is selection bias. The study participants were enrolled based on ICD-10 diagnostic coding, medical history, and management documentation, all of which may be imprecise or incomplete. A further limitation, due to possible selection bias, was the exclusion of approximately 50% of the study population at enrolment. Patients were recruited from a tertiary hospital in sub-Saharan Africa and were likely the sickest HF patients, and the findings from this registry cannot be extrapolated to the general HF population. In addition, this study population was small, limiting the reproducibility of the findings to represent a large population.

## CONCLUSION

This study illustrates a distinct epidemiological and clinical profile of AHF in South Africa, characterised by premature disease onset, predominant non-ischaemic cardiomyopathies, and a high comorbidity burden. Despite satisfactory implementation of conventional GDMT, systemic barriers persist in accessing advanced pharmacological therapies, reflecting critical healthcare disparities in South Africa. The high rates of re-admission and intermediate-term mortality underscore fundamental deficiencies in post-discharge pathways. These findings provide compelling evidence for urgent health policy reforms to expand access to all evidence-based GDMT, with the hope of mitigating the growing HF burden in resource-constrained African settings.

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CARDIAC CATHETERISATION LABORATORY PROCEDURES

# Cardiac catheterisation laboratory procedures and in-hospital outcomes at a tertiary facility: A 1-year review from Groote Schuur Hospital, Cape Town, South Africa

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## ABSTRACT

**Background:** Cardiovascular disease (CVD) is the leading cause of death in South Africa; however, comprehensive data on public-sector cardiac catheterisation laboratory (cath lab) procedural patterns and outcomes remain scarce.

**Methods:** We conducted a retrospective observational study using the Groote Schuur Hospital Cardiac Catheterisation (GSH-CATH) registry at Groote Schuur Hospital (GSH), Cape Town, South Africa, analysing all adult patients undergoing non-electrophysiology procedures between December 2022 and November 2023.

**Results:** A total of 1 694 procedures were performed in 1 239 patients (median age of 58 years, 60.5% female). The primary indications were acute coronary syndrome (ACS) (56.7%) and valvular heart disease (17.7%), with diagnostic coronary angiography (DCA) (40.9%), DCA with percutaneous coronary intervention (PCI), or PCI only (26.0%) being the most frequent procedures. Cardiovascular risk factors were highly prevalent, including hypertension (65.4%), smoking (44.1%), and diabetes (32.5%). The overall procedural complication rate was 6.5%, primarily driven by access-site events (3.2%). Intra-procedural and in-hospital mortality rates were 0.3% and 3.6%, respectively. Systemic hypertension was significantly associated with procedural complications ($p = 0.03$).

**Conclusion:** This study provides the first comprehensive evaluation of cath lab activity at a South African tertiary facility, highlighting high procedural volume and a unique female-dominant demographic, despite a high proportion of patients requiring emergency or time-sensitive interventions. These findings establish a baseline for quality improvement and resource allocation in the South African public health sector.

**Keywords:** catheterisation laboratory, coronary angiography, percutaneous coronary intervention.
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## INTRODUCTION

CVD was the leading cause of death in 2022, both globally and in South Africa.[1,2] In the South African context, CVD management is complicated by a unique quadruple burden: the human immunodeficiency virus pandemic, tuberculosis, non-communicable diseases (NCD), and violence or injury. As populations age and infectious disease management improves, the epidemiological shift toward NCDs, particularly CVD, places an unprecedented strain on healthcare infrastructure.[3] Cath labs are central to CVD diagnosis and management, yet access varies widely. High-income countries generally have 24-hour availability, whereas low- and middle-income countries face infrastructure and workforce constraints.[4] In South Africa, 76.6% of the country's 62 cath labs are privately owned, and consequently, only accessible through the private sector.[4] High rates of cardiovascular risk factors further strain cath lab services. A South African study conducted between 2013 and 2020 found that over half of adults aged > 45 years had hypertension, rising to about two-thirds in those > 55 years.[5] Additional reports indicate that 15.3% of adults have diabetes, 67.3% have dyslipidaemia, and 25.8% smoke tobacco products.[6-8]

International cath lab registries frequently focus on specific procedures, such as DCA or PCI, providing limited insight into overall procedural patterns. Broader registries are reported from Spain, Pakistan, and Uganda. The Spanish national registry reported an average of 906 procedures per cath lab in 2023, including DCA (67.1%), PCI (31.2%), valvular interventions (3.6%), non-valvular structural interventions (1.0%), and adult congenital heart disease procedures (0.8%).[9] In Pakistan, Khan, et al. reported 259 procedures in a single-centre study in 2015, primarily for ACS (53.0%) and chronic coronary syndromes

(29.7%), comprising DCA (43.6%), PCI (46.3%), and valvular interventions (9.7%) in a 67.2% male cohort.[10] In Uganda, 8 years of cath lab activity at a public hospital averaged 365 procedures per year, including DCA (65.2%), pacemaker-related and electrophysiology procedures (17.1%), PCI (14.0%), and valvular interventions (3.0%).[11] To date, no South African study has reported on the full spectrum of cath lab procedures, either at a private or public facility.

Cath lab complications and outcomes are influenced not only by operator skill, but also by patient factors, including age, cardiovascular risk, and the need for urgent intervention. Access-site complications are common; Tavakol, et al. reported rates of 0.2–1.0%.[12] Bhatty, et al. found femoral access-site complications of 1.8% for diagnostic and 4.0% for interventional procedures, with major bleeding in 2.0–6.0% and haematomas in 2.0–12.0%.[13] The multinational RIVAL (radial vs. femoral) trial reported rates of 4.0% for the femoral approach and 3.7% for the radial approach.[14] Other procedural complications identified by Tavakol, et al. included bradyarrhythmias and syncope (3.5%), peri-procedural myocardial infarction (MI) during PCI (5–30%), stroke during PCI (0.2–0.4%), aortic dissection (< 0.1%), and coronary artery perforation (0.3–0.6%).[12] Overall, in-hospital mortality is reported at 1.0%, with intra-procedural mortality at 0.1%.[15] Local South African data remain limited: the Tygerberg Registry of Acute Coronary Syndromes (TRACS) registry, limited to patients with ACS, reported 5.5% in-hospital mortality for ST-segment elevation myocardial infarction (STEMI), and 3.9% for high-risk non-ST-segment elevation acute coronary syndrome (NSTE-ACS); however, not all patients underwent cath lab procedures.[16,17]

GSH in Cape Town, South Africa, is a tertiary public hospital equipped with 2 cath labs. These labs operate primarily between 08:00 and 16:00 on weekdays, with only limited emergency procedures performed outside of these hours. A broad range of procedures is performed for patients referred to GSH, which is the only facility with cath lab services serving a vast geographic area of the Western Cape Province of South Africa. To address the lack of local data, we report the spectrum and outcomes of procedures performed at GSH.

## METHODS

The GSH-CATH registry was established to address the lack of comprehensive cath lab data in South Africa. This prospective, observational registry captures demographic, clinical, and procedural data, as well as procedural and in-hospital outcomes for all adult patients (> 18 years) undergoing cath lab procedures at GSH, with enrolment beginning on 1 December 2022. Pacemaker-related and electrophysiology procedures were excluded, as they are recorded in separate registries. To ensure clarity, the registry defined each cath lab admission as a single analytical entry. Any number of procedures performed during a single cath lab visit (e.g. DCA followed by PCI) were recorded as multiple procedures within 1 registry entry. Procedures performed during separate cath lab admissions were recorded as distinct entries, irrespective of whether they involved the same patient on multiple occasions or occurred during the same hospitalisation.

For this subgroup analysis, we conducted a retrospective observational study of registry data captured between 1 December 2022 and 30 November 2023. The primary objectives were to document the spectrum and frequency of cath lab procedures and evaluate associated peri-procedural complications and mortality. Data variables included patient demographics, cardiovascular risk profiles, diagnostic indications, and procedural outcomes as reported by the lead clinician in the cath lab procedure report. In-hospital mortality was recorded via hospital record review during the initial data capture. Intra-procedural complications and mortality were defined as events occurring during the cath lab admission, whereas in-hospital mortality included deaths occurring after cath lab discharge but prior to hospital discharge. No additional clinical data were collected beyond the registry parameters. Consequently, specific causes of death and the severity of pre-existing comorbidities were not assessed. Patients with incomplete data for any primary study variable were excluded from the final analysis.

**TABLE I: Demographics, diagnostic indications for cath lab procedures, and mortalities.**

| **Characteristic** | ***n* = 1 239**<br>***n* (%)** |
|---|---|
| **Demographics** | |
| Age (years) | 58 (50–67)* |
| Gender, female | 749 (60.5) |
| **Diagnostic indications for cath lab procedures** | |
| NSTE-ACS | 426 (34.3) |
| STEMI | 278 (22.4) |
| Valvular heart disease | 219 (17.7) |
| Chronic coronary syndrome | 189 (15.2) |
| Congenital heart disease | 34 (2.7) |
| Pericardial disease | 34 (2.7) |
| Cardiomyopathy | 17 (1.4) |
| Pulmonary hypertension | 15 (1.2) |
| Workup for transplant | 10 (0.8) |
| Arrhythmia | 5 (0.4) |
| Aortic dissection | 5 (0.4) |
| Transplant donor workup | 3 (0.2) |
| Atrial myxoma | 3 (0.2) |
| Infective endocarditis | 1 (0.1) |
| **Mortalities** | **49 (4.0)**** |
| Intra-procedural mortalities | 4 (0.3) |
| In-hospital mortalities following cath lab discharge | 45 (3.6) |

** Median (interquartile range)*

*** Percentages may not sum to the total due to independent rounding of component figures.*

*Cath lab: cardiac catheterisation laboratory, NSTE-ACS: non-ST-segment elevation acute coronary syndrome, STEMI: ST-segment elevation myocardial infarction.*

**TABLE II: Cath lab procedures and complications.**

| Procedures | *n* = 1 694<br>*n* (%) |
|---|---|
| **Procedure type** | |
| DCA | 693 (40.9) |
| DCA with PCI, or PCI only | 441 (26.0) |
| Left ventriculogram | 367 (21.7) |
| Haemodynamic study | 123 (7.3) |
| Pericardiocentesis | 33 (1.9) |
| Valvular intervention | 15 (0.9) |
| Endomyocardial biopsy | 10 (0.6) |
| Intra-aortic balloon pump insertion | 6 (0.4) |
| Congenital heart disease procedure | 4 (0.2) |
| Other | 2 (0.1) |
| **Complications** | **110 (6.5)*** |
| Access-site complications | 55 (3.2) |
| No-reflow or peri-procedural MI** | 12 (0.7) |
| Coronary dissection or perforation | 11 (0.6) |
| Arrhythmia | 10 (0.6) |
| Vasovagal episode | 7 (0.4) |
| Coronary vasospasm | 5 (0.3) |
| Stroke | 5 (0.3) |
| Thrombus embolisation | 4 (0.2) |
| Aortic dissection | 1 (0.1) |

** Percentages may not sum to the total due to independent rounding of component figures.*

*** As defined by the Fourth Universal Definition of Myocardial Infarction.[18]*

*Cath lab: cardiac catheterisation laboratory, DCA: diagnostic coronary angiography, MI: myocardial infarction, PCI: percutaneous coronary intervention.*

## Statistical analysis

Statistical analysis was conducted in collaboration with the Division of Epidemiology and Biostatistics at the University of Cape Town, using descriptive and comparative techniques. Continuous variables are summarised as medians with interquartile ranges (IQR), while categorical variables are reported as frequencies and percentages. Categorical comparisons were performed using Pearson's chi-squared test. Statistical significance was defined as a *p*-value < 0.05. For analytical precision, complication rates were calculated using the total number of procedures as the denominator (procedural basis), whereas mortality rates were calculated per patient, with each unique cath lab admission as the unit of analysis.

## Ethical approval

Institutional ethical approval for this sub-study was granted by the University of Cape Town Human Research Ethics Committee (reference: 780/2025). The overarching GSH-CATH registry was previously approved by the University of Cape Town Human Research Ethics Committee (reference: R047/2020) on 30 April 2021, with an extension valid until 30 April 2028. All research was conducted in strict adherence to patient anonymity protocols outlined in the original registry framework and in accordance with the Declaration of Helsinki.

## RESULTS

A total of 1 694 procedures were performed during 1 239 cath lab visits, averaging 141 procedures per month or approximately 33 per week. Most patients (66.5%) underwent a single procedure, while 38 patients (3.1%) had ≥ 3 procedures during the same cath lab admission. The most frequently performed procedure was DCA (40.9%), followed by DCA with PCI or PCI only (26.0%), and left ventriculography (21.7%) (Table II). The most frequently performed combination procedure was a haemodynamic study with DCA (performed in 69 patients). Procedural indications were most commonly ACS (56.7%). Of these, NSTE-ACS and STEMI accounted for 34.3% (426 cases) and 22.4% (278 cases) of the ACS cohort, respectively (Table I).

Among 1 694 procedures, 110 complications were recorded, corresponding to a per-procedure complication rate of 6.5%. Across 1 239 cath lab admissions, 94 patients experienced a single complication, while 8 patients sustained 2 complications during the same admission. Access-site complications occurred in 55 procedures (3.2%), while no-reflow or peri-procedural MI (defined by the Fourth Universal Definition of Myocardial Infarction) occurred in 12 procedures (0.7%).[18] Coronary dissection or perforation and arrhythmia each occurred in 0.6% of procedures. Intra-procedural mortality was observed in 4/1 239 patients (0.3%), with an additional 45 patients (3.6%) dying during hospital admission following the procedure (Table I).

This cohort had a median age of 58 years (IQR 50–67 years),

**TABLE III: Complication rates stratified by cardiovascular risk factors.**

| Cardiovascular risk factors | Patients with disease | | Patients without disease | | *p*-value* |
|---|---|---|---|---|---|
| | *n* | Complications, *n* (%) | *n* | Complications, *n* (%) | |
| Hypertension** | 810 | 77 (10.5) | 429 | 25 (6.2) | 0.03 |
| Diabetes*** | 403 | 33 (8.9) | 836 | 69 (9.0) | 0.97 |
| Dyslipidaemia*** | 525 | 46 (9.4) | 704 | 56 (8.6) | 0.61 |
| Active cigarette smoking | 547 | 42 (8.3) | 692 | 60 (9.5) | 0.53 |

** Pearson's chi-squared test.*

*** Systemic hypertension.*

**** Diabetes mellitus and dyslipidaemia of any type.*

with a female predominance (60.5%) (Table I). Among the patients, 65.4% had hypertension, 43.2% had hyperlipidaemia, 32.5% had diabetes, and 44.1% were active smokers. Analysis of individual cardiovascular risk factors revealed that only systemic hypertension was significantly associated with procedural complications (10.5% vs. 6.2%; $p = 0.03$). When risk factors were grouped (e.g. diabetes combined with hypertension), no statistically significant associations with procedural complications were observed (Table III).

## DISCUSSION

The annual procedural volume at GSH (1 694 procedures) demonstrates significant operational efficiency, exceeding single-centre registries in Pakistan (259 procedures) and Uganda (365 procedures), and surpassing the average annual volume per lab in the Spanish national registry (906 procedures).[9-11] This high throughput is particularly notable given the cath lab's primary operation within standard working hours (08:00–16:00), which necessitates a selective referral pattern and often a pharmaco-invasive approach for after-hours ACS cases. Consistent with global trends, DCA predominated at 40.9%, followed by PCI at 26.0%.[9-11] However, excluding pacemaker and electrophysiology procedures, which are typically associated with shorter procedural times and lower inherent risks, likely shifts the reported spectrum toward a more complex workload dominated by urgent, high-risk interventional cases, compared with holistic registries.[19] This comprehensive role is further evidenced by a diverse range of complex diagnostic indications, including transplant donor workups, pulmonary hypertension studies, and endomyocardial biopsies.

The overall complication rate in our study was 6.5%, higher than the 3.6% reported by Khan, et al.; however, direct comparisons are limited by differences in diagnostic indications and procedural scope, as only 4 procedure types were included in their study.[10] Our 3.2% access-site complication rate is lower than recorded in the RIVAL trial (3.7–4.0%), suggesting that vascular access safety at GSH is comparable to high-level clinical trial standards.[14] When excluding minor events, including access-site complications and vasovagal episodes, the major complication rate was 2.8%. Peri-procedural MI occurred in 0.7% of procedures, while serious complications, such as stroke, aortic dissection, or coronary perforation, each occurred in < 0.7%, consistent with previously published data.[12] While mortality rates exceeded global averages (0.3% intra-procedural and 3.6% in-hospital), they must be interpreted alongside the clinical complexity of the GSH population.[15] Unlike low-risk registries, 56.7% of patients in our study presented with ACS, with STEMI accounting for nearly a quarter (22.4%) of the overall study population.

In our cohort, the median age was 58 years, with a marked female predominance (60.5%), contrasting with the standard male predominance (often > 65%) reported in regional and global CVD registries.[9-11,16,17] This is a unique finding that likely reflects the specific risk profile of the South African public sector. In South Africa, women have a significantly higher prevalence of obesity and hypertension than men, which are major drivers of cardiovascular morbidity.[5] Furthermore, valvular heart disease remains a major burden in the public sector, often stemming from rheumatic heart disease, which disproportionately affects females in sub-Saharan Africa.[3] Moreover, the prevalence of active smoking (44.1%) and diabetes (32.5%) significantly exceeds national averages, reflecting the concentration of high-risk patients referred to a tertiary level.[6,8] Dyslipidaemia was lower than the national prevalence, possibly due to underdiagnosis or incomplete testing during admission. Systemic hypertension was identified as the only significant predictor of procedural complications ($p = 0.03$), reinforcing the concept that the pre-procedural physiological state is a critical safety determinant.

### Limitations and recommendations

Several limitations warrant consideration. As a single-centre, registry-based analysis, this study relied on clinician-reported data, which precluded external verification of cardiovascular risk factors, a granular assessment of mortality causes, or the physiological severity of cases, such as the presence of cardiogenic shock at presentation. Furthermore, the outcomes reflect the aggregate experience of multiple operators within a training institution rather than individual clinician performance.

Future research should prioritise multicentre, prospective cohorts that incorporate longitudinal follow-up and operator-level metrics to further refine risk stratification and resource allocation in the South African context. Expanding registry parameters to include pacemaker and electrophysiology data will provide a more holistic representation of cath lab activity and its associated safety profile. Additionally, more detailed recording of specific access-site complications will provide essential data to drive targeted quality improvement initiatives and establish a robust foundation for evidence-based health policy development.

## CONCLUSION

This study provides the first comprehensive audit of cath lab activity within a South African tertiary centre, delineating a procedural spectrum that extends significantly beyond the diagnostic and interventional coronary cases typically reported in global registries. Our findings reveal a population with an exceptionally high burden of cardiovascular risk factors managed within a high-volume public-sector environment characterised by urgent and life-threatening clinical presentations. While complication and mortality rates appeared higher than those in some international high-income country registries, these results must be interpreted through the lens of patient complexity and the diverse, high-risk emergency indications managed at our facility.

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POSITION PAPER

# South African Heart Association Position Statement for the management of valvular heart disease – Part I

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## ABSTRACT

The burden of valvular heart disease (VHD) remains high in South Africa and is associated with considerable morbidity and mortality. While a decline in acute rheumatic fever cases has been observed, chronic rheumatic VHD remains an important cause of index heart failure admission in South Africa. Additionally, with the increased longevity of the African population, degenerative VHD has emerged as an important aetiology. To date, data about VHD epidemiology, diagnosis, management, and patient follow-up remain scarce in this region. Patients with VHD and their physicians face unique challenges in the South African setting. Hence, in this Position Statement, we aim to provide the general cardiologist with a comprehensive review to complement existing guidelines on VHD for adequate patient management in the local setting. This document will comprise 2 parts. Part I focuses on the evaluation and management of native VHD. Part II will focus on prosthetic heart valves, infective endocarditis (IE), preoperative assessment of patients with VHD, VHD considerations in children, and future directions.

**Keywords:** valvular heart disease, management, South Africa.
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## ABBREVIATIONS/ACRONYMS

AF: atrial fibrillation
AR: aortic regurgitation
AS: aortic stenosis
AVR: aortic valve replacement
CABG: coronary artery bypass grafting
CAD: coronary artery disease
$CHA_2DS_2$-VASc score: congestive heart failure, hypertension, age (≥ 75, 2 points), diabetes, stroke or transient ischaemic attack (2 points), vascular disease, age (65–74), and sex category
CMR: cardiac magnetic resonance
CT: computed tomography
ESC: European Society of Cardiology
EuroSCORE II: European System for Cardiac Operative Risk Evaluation II
HIV: human immunodeficiency virus
IE: infective endocarditis
INVICTUS: investigation of rheumatic atrial fibrillation treatment using vitamin K antagonists, rivaroxaban or aspirin studies
LFLG: low-flow, low-gradient
LMIC: low- to middle-income country
LOE: level of evidence
LV: left ventricular
LVEF: left ventricular ejection fraction
LVESD: left ventricular end-systolic diameter
MRAs: mineralocorticoid receptor antagonists
MR: mitral regurgitation
MS: mitral stenosis
NT-proBNP: N-terminal pro–B-type natriuretic peptide
OMT: optimal medical therapy
REMEDY: Global Rheumatic Heart Disease Registry
RHD: rheumatic heart disease
SAVR: surgical aortic valve replacement
SMR: secondary mitral regurgitation
SPAP: systolic pulmonary artery pressure

STS: The Society of Thoracic Surgeons
TAVI: transcatheter aortic valve implantation
TOE: transoesophageal echocardiography
TR: tricuspid regurgitation
TS: tricuspid stenosis
VHD: valvular heart disease
VKA: vitamin K antagonist

## INTRODUCTION

VHD is a major global cause of morbidity and mortality, with varying epidemiology based on location.[1-3] Degenerative and functional valve disease predominates in high-income countries, while rheumatic heart disease (RHD) remains the leading aetiology of VHD in low- to middle-income countries (LMIC), affecting about 41 million people worldwide and contributing significantly to heart failure in endemic areas.[4,5] RHD is strongly associated with poverty and exposure to Group A *Streptococcus*.[6,7] In South Africa, heart failure incidence due to RHD increases with age, reaching over 53 per 100 000 per year in older adults, with a cumulative incidence of 23 per 100 000 per year.[8] Data from the Global Rheumatic Heart Disease Registry (REMEDY) and other studies show high mortality (16.9% at 2 years) and reveal that patients in resource-limited settings often present young with advanced disease and related complications.[9,10]

Given the high burden of RHD in sub-Saharan Africa, the potential rise in degenerative VHD due to increased life expectancy, and the rapid evolution of transcatheter techniques, medical management, and multimodality imaging in VHD, we deemed it necessary to formulate a location-specific Position Statement.[9-11] In Africa, human resources, expertise, and equipment limitations may not allow for dedicated heart valve centres and heart teams at all facilities. However, numerous referral centres offer specialist cardiologists and surgeons, and we urge clinicians involved in the care of patients with VHD to have a low threshold for consultation or referral.

It is of utmost importance to accurately diagnose, quantify, assess the mechanism of, and identify complications in a VHD patient.[12] A baseline physical examination, 12-lead electrocardiogram, chest X-ray, and cardiac imaging with a transthoracic echocardiogram, supplemented by transoeso-phageal echocardiography (TOE) if necessary, form the cornerstone of diagnosis.[14] Coronary artery imaging via computed tomography (CT) coronary angiography or invasive coronary angiography is recommended for coronary artery disease (CAD) assessment in men aged > 40 years, postmenopausal women, those with a history of cardiovascular disease, myocardial ischaemia, left ventricular (LV) systolic dysfunction, and ≥ 1 cardiovascular risk factors before valve surgery or a valve intervention (Class I, level of evidence [LOE] C). In low-risk cases, cardiac CT angiography has good negative predictive value for excluding CAD (Class IIa, LOE C).[12]

In the local context, resource and expertise limitations may preclude multimodality imaging assessment.[9] We advocate for diagnostic investigation, in which a centre has expertise for VHD assessment. The value of routine coronary angiography in the absence of cardiovascular risk factors before VHD intervention was investigated by Meel, et al. in a South African study.[13] They noted a low CAD prevalence (8.6%) in patients with VHD at Chris Hani Baragwanath Academic Hospital. The study's conclusion suggested individualising the decision to perform screening coronary angiography, considering age, symptoms, and cardiovascular risk factors for black patients scheduled to undergo valve replacement surgery in developing countries.

When managing a patient with VHD, their preferences must be considered.[12] The patients and their families must be extensively informed and guided in making the best decision and choosing the appropriate treatment option, considering the patient's life expectancy and expected quality of life. Comorbidities in the elderly should be considered, and therapeutic futility should be avoided, especially in a resource-limited setting such as South Africa. Appropriate patient risk stratification before surgery or intervention is required, using standard risk scores, such as The Society of Thoracic Surgeons (STS) predicted risk of mortality and the European System for Cardiac Operative Risk Evaluation II (EuroSCORE II). In the South African setting, with possible delays in definitive surgery, as shown in a recent study by Banderker, et al., patients with VHD and heart failure must be managed with optimal medical therapy (OMT) as a bridge to surgery.[14] Furthermore, patients who do not qualify for or decline surgery must be placed on optimal anti-failure pharmacotherapy.

IE prevention is crucial in high-risk patients, including those with prosthetic valves or material, previous IE, congenital heart disease, and ventricular assist devices, in which antibiotic prophylaxis is recommended.[12,15] Per the European Society of Cardiology (ESC) guidelines, antibiotic prophylaxis is not routinely indicated for intermediate risk groups, such as patients with RHD, degenerative valve disease, congenital valve abnormalities, cardiac devices, or hypertrophic cardiomyopathy. However, it may be considered on an individual basis. Strict oral hygiene is essential for both high- and intermediate-risk groups, especially in South Africa, where oral health among RHD patients is often poor, necessitating dental care education.[16] The South African Heart Association identifies RHD patients as high-risk and advises routine antibiotic prophylaxis for dental procedures involving gingival or mucosal manipulation. Additionally, increasing intravenous drug use increases IE risk in this population, requiring targeted prevention strategies.[17,18]

In patients with VHD and concomitant atrial fibrillation (AF), direct oral anticoagulants are preferred over vitamin K antagonists (VKA) for those with aortic regurgitation (AR), aortic stenosis (AS), or mitral regurgitation (MR).[12] However, they are not recommended for prosthetic valves or rheumatic moderate-to-severe mitral stenosis (MS).[12] The investigation of rheumatic AF treatment using VKAs, rivaroxaban or aspirin studies (INVICTUS) trial showed that VKAs were superior to rivaroxaban in reducing cardiovascular events and death in RHD

without increasing the bleeding risk.[19] AF ablation should be considered during valve surgery, while evidence for routine left atrial appendage exclusion remains inconclusive.[20] Though large prospective trials are lacking, left atrial appendage resection is advised for patients with a $CHA_2DS_2$-VASc score ≥ 2 undergoing valve surgery, per ESC guidelines.[12]

For established, chronic RHD, long-term secondary prophylaxis against rheumatic fever with intramuscular benzathine benzylpenicillin every 3–4 weeks for 10 years is recommended.[12] Lifelong prophylaxis should be considered in high-risk patients, based on the severity of their valve disease and streptococcal exposure. Primary prevention during acute rheumatic fever focuses on treating Group A *Streptococcus* infection, while echocardiographic screening and secondary prophylaxis in subclinical RHD are under investigation to reduce the prevalence in endemic regions. However, studies such as REMEDY and a recent South African single-centre study highlight the low uptake of secondary prophylaxis, possibly due to older patient populations, a reported decline in acute rheumatic fever incidence, underdiagnosis of latent carditis in chronic RHD, and supply issues in the public sector.[9,14,21]

Sliwa, et al. reported a high prevalence of comorbidities, including renal dysfunction, AF, and anaemia in patients with RHD.[8] Banderker, et al. and Meel, et al. found a high prevalence of comorbidities in patients with rheumatic mitral valve disease.[14,22] Arterial hypertension and human immunodeficiency virus (HIV) infection were the most common concomitant diseases in these studies. Patients with dual HIV and RHD have threefold higher odds of suffering a stroke or transient ischaemic attack than those with isolated RHD.[23] Timely recognition and appropriate management of comorbidities are essential in these patients.

## AORTIC REGURGITATION (AR)

### Aetiology

AR can be caused by primary aortic valve disease or pathology of the ascending aorta (idiopathic root dilatation, Marfan syndrome, aortic dissection, collagen vascular disease, or syphilis). The most common cause in high-income countries is calcific aortic valve disease.[24]

### Assessment

Clinically, AR may be difficult to ascertain or grade. Signs suggestive of severe AR include a longer diastolic murmur with a rougher quality, pronounced peripheral signs (particularly Hill's and Duroziez's signs), an Austin Flint murmur, and signs of increased LV size/LV dysfunction, such as a displaced apex beat or a third heart sound.[25] Patients with suspected AR need to undergo echocardiography to assess its mechanism and severity, and evaluate the LV size and function. Echocardiographic features suggestive of severe AR are summarised in Table I.[26] A CT scan may be required to accurately assess the dimensions of the aortic root and the ascending aorta. If there is doubt, TOE and cardiac magnetic resonance (CMR) imaging are useful alternative modalities to assess the morphological features and AR severity.[12]

**TABLE I: Features of severe aortic regurgitation.**

| |
|---|
| Dilated left ventricle, LVEDD > 65 mm |
| Dense regurgitant jet on CW Doppler profile |
| Jet area ≥ 65% of LVOT |
| Vena contracta > 6 mm |
| Pressure half-time < 200–250 ms, CW max velocity > 3–3.5 m/s |
| Holodiastolic flow reversal with an end-diastolic velocity ≥ 20 cm/s in the descending aorta |

*CW: continuous wave, LVEDD: left ventricular end-diastolic diameter, LVOT: left ventricular outflow tract.*

### Management

Surgical aortic valve replacement (SAVR) is recommended for all symptomatic patients with severe AR. The ESC guidelines advise SAVR for all such patients, regardless of LV function.[12] Even patients with extreme LV enlargement and significant LV dysfunction experience symptomatic and survival benefits from aortic valve intervention.[27,28] SAVR carries a higher risk in this setting, but in the modern surgical era, peri-operative outcomes are acceptable.[29]

In asymptomatic patients with severe AR, surgery should be performed if the left ventricular end-systolic diameter (LVESD) is > 50 mm (indexed LVESD > 25 mm/m$^2$), the left ventricular ejection fraction (LVEF) is ≤ 50%, or if serial imaging indicates significant disease progression. Moreover, patients with incidental, significant AR who are undergoing cardiac surgery for another indication (e.g. coronary artery bypass grafting [CABG]) should undergo SAVR.[12]

SAVR is the preferred intervention method. Transcatheter aortic valve implantation (TAVI) is reserved for carefully selected patients and should be performed only in experienced centres. Although some evidence exists for the symptomatic benefit of renin–angiotensin–aldosterone system inhibitors and dihydropyridines, the role of pharmacotherapy remains adjunctive.[30] Beta blockers and other negatively chronotropic agents must be used with caution in patients with severe, chronic AR. Pharmacotherapy has no established role in delaying AR progression.

It is important to diagnose any significant pathology of the aortic root and ascending aorta. In the absence of traditional risk factors for aortopathy, syphilis should be ruled out as a cause. Dilatation of the aortic root or ascending aorta may need to be addressed at the time of surgery, and should be strongly considered if the aortic diameter measures ≥ 55 mm.[12]

### The South African perspective

Any of the AR causes that are prevalent in the developed world could apply in the South African context. However, RHD remains an important cause of valve pathology, with an estimated

incidence of 23.5 cases per 100 000 per year in our region.[8] While significant aortic valve disease due to RHD is generally associated with other valve pathology, it can occur in isolation.[31,32] Another important cause in LMICs is IE.

Although access to echocardiography may be limited, it remains essential for confirming the diagnosis and guiding management. Specifically in the context of AR, it is important to rule out functional or presystolic MR, as additional mitral valve replacement is usually not required in these cases.[32]

## AORTIC STENOSIS (AS)

### Aetiology

The most common cause of AS in developed countries is calcific aortic valve disease.[33-36]

### Assessment

Clinical features suggestive of severe AS include a systolic murmur (grade 4/6 or greater), central *pulsus parvus et tardus* or a late-peaking murmur, a diminished or absent second heart sound, paradoxical splitting of S2, and a fourth heart sound.[25] Echocardiography supplemented by TOE is required to assess AS morphology and severity.[12] The main determinants of severe AS are summarised in Table II. In patients with diagnostic uncertainty regarding AS severity based on the parameters listed in Table II, LV systolic function, marked aortic valve calcification, and a Doppler velocity time index < 0.25 (LV outflow tract time integral/aortic valve time integral) may be of incremental value.

In the presence of an impaired LVEF, care should be taken not to miss the presence of low-flow, low-gradient (LFLG) AS.[12] It is characterised by a mean transvalvular gradient < 40 mmHg, a valve area ≤ 1 $cm^2$, a LVEF < 50%, and an indexed LV stroke volume ≤ 35 $ml/m^2$.[12] Dobutamine stress echocardiography is required for these patients to rule out pseudo-stenosis. A CT scan with a high aortic valve Agatston score (> 1 200 for females, > 2 000 for males) is further evidence that AS is severe in the setting of LFLG AS.

Further investigations that may help determine the need for surgery, particularly in asymptomatic patients with severe AS, include conventional exercise stress testing to confirm the absence of symptoms and identify blood pressure drop during exercise, a CT scan to assess the severity of aortic valve calcification, and serum N-terminal pro–B-type natriuretic peptide (NT-proBNP) levels.[12]

**TABLE II: Features of severe aortic stenosis.**

| |
|---|
| Mean transvalvular gradient ≥ 40 mmHg |
| Peak velocity ≥ 4 m/s |
| Valve area (according to the continuity equation) ≤ 1.0 $cm^2$, indexed < 0.6 $cm^2/m^2$ |
| Time to peak velocity > 100 ms* |

** Kamimura D, Hans S, Suzuki T, et al. Delayed time to peak velocity is useful for detecting severe aortic stenosis. J Am Heart Assoc. 2016;5(10):e003907. https://doi.org/10.1161/JAHA.116.003907.*

### Management

The first step is to assess whether aortic valve intervention is required. If so, a decision must be made between TAVI and SAVR.[12] Symptomatic patients with severe AS (including LFLG AS) should generally undergo intervention.[12] The decision is more challenging in asymptomatic patients, where expectant management until symptom development before proceeding to intervention has been the traditional approach. Generally, asymptomatic patients with severe AS and a LVEF < 55% should undergo intervention, as should patients with another indication for cardiac surgery (e.g. CABG) who also happen to have significant AS requiring aortic valve replacement (AVR).[12] Preliminary data suggest that asymptomatic patients with a normal LVEF could benefit from early aortic valve intervention; however, a conservative approach is still required for most patients with severe, asymptomatic AS.[34]

Based on currently available evidence, AVR can be considered in patients at low surgical risk who, in addition to having severe AS, manifest: (1) systolic blood pressure decrease > 20 mmHg during exercise stress testing; (2) a mean transvalvular gradient ≥ 60 mmHg or a maximal transvalvular velocity ($V_{max}$) > 5 m/s; (3) annual $V_{max}$ progression ≥ 0.3 m/s; (4) severe aortic valve calcification on CT scan; or (5) significantly elevated NT-proBNP levels.[12,37] Once the decision regarding aortic valve intervention has been made, the heart team must weigh numerous factors to determine whether TAVI or SAVR best serves the patient.

### The South African perspective

AS can be caused by tricuspid or bicuspid degenerative calcific aortic valve disease or RHD. Although rarely the cause of isolated AS, RHD contributes significantly to the burden of AS in South Africa.[36,37] It is usually accompanied by mitral valve involvement. Despite limited resources, the ESC guidelines should be followed regarding indications for aortic valve intervention; SAVR remains the gold standard. Most patients aged < 70 years, particularly if the aetiology is bicuspid aortic valve disease, should undergo SAVR. TAVI is not currently recommended in younger patients with RHD-related AS. As a general guide, patients with a EuroSCORE II or STS score ≥ 4% are at least intermediate risk or higher, and the final risk assessment resides with the heart team. Limited femoral access, bicuspid aetiology, multivessel CAD, and low coronary origins above the annulus favour SAVR.

The decision to forego SAVR in favour of TAVI is complex and should be made by a heart team. TAVI should be considered in the following scenarios: (1) all patients aged ≥ 70 years (regardless of surgical risk); (2) expected, technically difficult open-heart surgery (e.g. previous radiation, porcelain aorta, previous CABG where potential damage to the grafts is anticipated); (3) a very frail patient; (4) major comorbidity (the most common being chronic obstructive pulmonary disease); or (5) a younger patient with a high operative risk.[38,39]

## MITRAL REGURGITATION (MR)

### Aetiology

MR can be primary or secondary (SMR). Primary MR arises from disease of one or more components of the mitral valve apparatus.[12] RHD is the most common MR aetiology in low-income countries, whereas degenerative MR dominates in high-income countries.[12] IE remains an important MR aetiology. SMR arises from left atrial or ventricular remodelling rather than primary leaflet disease.[12] Atrial SMR is linked to AF and enlargement of the annulus (often with heart failure with preserved ejection fraction).[12] Ventricular SMR is associated with LV dilation and resultant papillary muscle displacement in heart failure with reduced ejection fraction.[12]

### Assessment

Severe MR presents with dyspnoea, fatigue, orthopnoea, and palpitations due to AF and LV volume overload. Clinical signs of severe MR include a left atrial lift, high-grade pansystolic murmur at the apex radiating to the axilla, a displaced apex beat, a third heart sound, and, in late stages, signs of pulmonary oedema and right heart failure.[40] Transthoracic echocardiography supplemented by TOE is the recommended first-line modality for MR evaluation.[12] An integrated approach using qualitative, semi-quantitative, and quantitative assessments is crucial for an accurate assessment of MR severity (Table III).[12]

MR mechanism evaluation is important for decision-making regarding surgical or transcatheter mitral valve repair. In cases where two-dimensional echocardiography is insufficient to evaluate mitral valve anatomy or MR severity, additional imaging is indicated, such as advanced echocardiography (strain, three-dimensional, TOE), exercise echocardiography, and CMR imaging.[12] Right heart catheterisation has utility in accurately quantifying systolic pulmonary artery pressure (SPAP), and where there is discordance between echocardiographic MR severity assessment and symptoms. It is also useful to exclude pulmonary hypertension from coexisting lung pathology.

### Management

Urgent surgery is advised in acute, severe MR.[12] Surgery is indicated for chronic, severe, symptomatic, and primary MR (Class I, LOE B). In the absence of symptoms, a LVEF ≤ 60% (Class I, LOE B), a LVESD ≥ 40 mm (Class I, LOE B), a left atrial volume ≥ 60 ml/m$^2$ or diameter ≥ 55 mm (Class IIa, LOE B), SPAP > 50 mmHg (Class IIa, LOE B), and AF (Class IIa, LOE B) portend a poor prognosis, and these patients must be considered for intervention. Watchful waiting is considered safe in asymptomatic patients who do not meet these criteria. Mitral valve repair is favoured over mitral valve replacement depending on the complexity of the lesion (Class I, LOE B) and the availability of surgical expertise.[12]

Management per ESC 2025 guidelines prioritises OMT for heart failure and AF control for SMR, with cardiac resynchronisation therapy indicated for eligible patients.[12] Interventions are considered for symptomatic, severe SMR despite OMT and device therapy, following heart team evaluation. Transcatheter repair is recommended for non-surgical candidates who meet echocardiographic criteria, while surgery is considered primarily during concomitant CABG.[12] In advanced cases unresponsive to

**TABLE III: Quantification of severe primary and secondary mitral regurgitation.[12]**

| | Parameter | Criteria |
|---|---|---|
| Qualitative assessment | Mitral valve morphology | Primary: Flail leaflet or prolapse, large coaptation defect, retracted leaflet<br>Secondary: Normal leaflets with severe tenting, poor leaflet coaptation |
| Qualitative assessment | Colour flow jet area | Primary: Large central jet (≥ 50% of LA) or eccentric wall impinging jet of variable size<br>Secondary: Small to moderate central jet (< 50% of LA) or eccentric jet along the wall |
| Qualitative assessment | Flow convergence | Primary: Large throughout systole<br>Secondary: Small to absent in late systole |
| Qualitative assessment | Continuous wave Doppler jet | Primary: Holosystolic/dense/triangular<br>Secondary: Late systolic/decreased intensity/parabolic |
| Semi-quantitative assessment | Vena contracta width (cm) | Primary: ≥ 0.7 cm (≥ 0.8 cm for biplane)<br>Secondary: ≥ 0.7 cm (biplane) |
| Semi-quantitative assessment | Pulmonary vein flow pattern | Primary: Systolic flow reversal, blunted S wave in one pulmonary vein may be normal if other criteria are met<br>Secondary: Systolic blunting |
| Semi-quantitative assessment | Mitral inflow pattern | Primary: E wave dominant (> 1.2 m/s)<br>Secondary: E wave dominant (> 1.2 m/s) |
| Semi-quantitative assessment | TVI MV/TVI aortic | Primary: > 1.4<br>Secondary: > 1.4 |
| Quantitative assessment | EROA (2D PISA, mm$^2$) | Primary: EROA ≥ 40 mm$^2$/R Vol ≥ 60 ml<br>Secondary: ≥ 30 mm$^2$/R Vol ≥ 45 ml |

*Note: Enlargement of the left ventricle and left atrium is a sign of severe mitral regurgitation.*
*2D: two-dimensional, EROA: effective regurgitant orifice area, LA: left atrium, MV: mitral valve, PISA: proximal isovelocity surface area, R Vol: regurgitant volume, TVI: time velocity integral.*

therapy, LV assist device implantation or cardiac transplantation may be options.

Medical therapy is useful to reduce LV filling pressure and decrease afterload in acute MR.[12] Inotropic support and intra-aortic balloon pump insertion are indicated for hypotension and haemodynamic instability. There is a limited role for medical therapy in chronic MR with preserved LV function. Patients presenting with overt heart failure should be managed per standard guidelines.[41]

In the absence of symptoms, patients with severe MR and preserved LVEF should be followed up every 6 months.[12] Asymptomatic patients with moderate MR and preserved LV function should be followed up 1–2 yearly. Post-intervention follow-up should focus on assessing symptoms, arrhythmias, valve function, and MR recurrence. Serial NT-proBNP may support closer monitoring or earlier referral in borderline cases. Ideally, these patients should be followed up at a heart valve centre.

### The South African perspective

Recent studies from Chris Hani Baragwanath Academic Hospital noted MR secondary to RHD as the most common valve lesion in adults with mitral valve disease.[14,22,42] Most patients were African females with comorbidities, such as HIV and arterial hypertension. Myxomatous mitral valve degeneration was noted in a minority of cases. Most patients were late presenters, manifesting in heart failure. Limited surgical resources resulted in delays in mitral valve intervention. Consequently, many patients were treated with anti-remodelling heart failure drugs, serving as a bridge to surgery.[43]

Notably, rheumatic MR is characterised by eccentric jets; therefore, the value of quantitative parameters, such as the proximal isovelocity surface area method, cannot be relied upon solely for MR quantification, and a multiparametric approach must be utilised. If there is doubt regarding MR severity, additional imaging is indicated, such as TOE when available.[44] CMR imaging in rheumatic MR allows severity assessment of the severity and LV fibrosis through tissue characterisation sequences, which can aid in decision-making regarding surgery and prognostication.[45]

In South Africa, advanced echocardiography and CMR are scarce resources with limited expertise. Mitral valve repair generally offers better outcomes, including preservation of the patient's native valve, which can lead to improved haemodynamics and reduced complications related to prosthetic valves (thrombosis or IE), though it carries a higher risk of reoperation.[46] Most patients in South Africa with severe SMR do not currently have access to percutaneous mitral valve repair. Therefore, patients who are not candidates for surgery should be treated according to the existing heart failure guidelines.

Currently, 2 centres in the Western Cape and 1 in Gauteng perform percutaneous mitral valve repair. Patients with inoperable primary MR or those with SMR and ongoing symptoms despite maximally tolerated guideline-directed therapy (including cardiac resynchronisation therapy if indicated) should be referred for assessment for transcatheter mitral edge-to-edge repair.

## MITRAL STENOSIS (MS)

### Aetiology

RHD is the leading cause of MS.[12] Other aetiologies include degenerative disease, and, less commonly, chest radiation, carcinoid heart disease, and inherited metabolic disorders. In contrast to other aetiologies, the hallmark of rheumatic MS is commissural fusion.

### Assessment

Significant MS presents with dyspnoea, haemoptysis, fatigue, and, in advanced cases, peripheral oedema, hoarseness, and embolic events. On auscultation, a low-pitched mid-diastolic rumbling murmur is present at the apex, accompanied by an opening snap; loud S1/features of AF.[47] Transthoracic echocardiography is the first-line modality for MS evaluation.[12] It assesses aetiology, severity, and haemodynamic consequences. A mitral valve area ≤ 1.5 $cm^2$, a mean transmitral gradient ≥ 10 mmHg, and pulmonary hypertension are considered clinically significant (Table IV).

TOE is indicated before balloon mitral valvuloplasty. It provides detailed information on valve anatomy and excludes left atrial thrombus. Three-dimensional echocardiography with slice rendering is useful for accurate valve planimetry. In patients with equivocal symptoms or borderline MS severity, exercise echocardiography provides objective data on increases in transmitral gradient and SPAP.[48] Proposed mean gradient thresholds for severe MS include values exceeding 15 mmHg during exercise or 18 mmHg during dobutamine infusion.[48]

### Management

Intervention on the mitral valve should be reserved for moderate-to-severe MS.[12] Depending on the patient's clinical characteristics and anatomy of the valve and subvalvular structures based on standardised scores (Wilkins or Cormier) and local expertise, MS can be treated with balloon mitral valvuloplasty (Class I, LOE B) or surgery (Class I, LOE C).[12] Balloon mitral valvuloplasty is not indicated in degenerative MS, since it is characterised by mitral annular calcification and not commissural fusion. In highly symptomatic patients, transcatheter valve replacement or surgery may be considered.

**TABLE IV: Severity grading of mitral stenosis.**

| | Mild | Moderate | Severe |
|---|---|---|---|
| Valve area ($cm^2$) | > 1.5 | 1–1.5 | ≤ 1.5 |
| Mean gradient (mmHg) | < 5 | 5–10 | > 10 |
| Pulmonary artery pressure (mmHg) | < 20 | 30–50 | > 50 |

The entity of LFLG MS is also noteworthy.[49] This is defined as a mean mitral valve area < 1.5 $cm^2$, measured on planimetry, a mean gradient < 10 mmHg, and a transmitral flow < 35 $ml/m^2$. Recent evidence suggests that these patients are older, more ill, with AF, and more subvalvular disease. After valvotomy, they have a lesser reduction in left atrial pressure, and, most importantly, a suboptimal symptomatic response. This may be related to independent ventricular-vascular uncoupling, decreased LV compliance, and a high prevalence of AF in addition to intrinsic MS.

Medical management aims to control heart failure symptoms, achieve rate or rhythm control, and prevent thrombus and systemic embolism.[12] In the absence of contraindications, beta blockers are the mainstay of treatment for AF rate control.[12] Alternative agents for rate control include digoxin and non-dihydropyridine calcium-channel blockers.[12] Amiodarone is effective for maintaining sinus rhythm after cardioversion.[12] Cardioversion and pulmonary vein isolation are not indicated in patients with severe, untreated MS. Cardioversion can be considered in recent-onset AF with a moderately enlarged left atrium and less severe MS.

Oral anticoagulation with a VKA is indicated in AF. The INVICTUS study found that VKAs were associated with a lower rate of a composite of cardiovascular events and death than rivaroxaban, without a higher bleeding rate.[19] In patients in sinus rhythm, oral anticoagulation in MS is indicated in the presence of dense, spontaneous echocardiographic contrast, a history of thromboembolism, and a left atrial diameter > 50 mm or volume > 60 $ml/m^2$.[12]

### The South African perspective

In a recent South African study conducted at a referral centre, isolated rheumatic MS was documented in 23% of patients.[14] Females were predominantly affected, which aligns with data from other LMICs. None of the patients in this study had degenerative (calcific) MS; a likely reflection of the study patients' younger age compared with those in higher-income countries.

In the local setting, where most patients with MS are still young and surgical resources are scarce, balloon mitral valvuloplasty should be the first line of treatment when feasible.[50] In patients with contraindications to balloon mitral valvuloplasty, stabilisation with medical therapy is a reasonable option while awaiting definitive surgical intervention.[14] Zühlke, et al. reported suboptimal use of oral anticoagulation in patients with RHD.[10] Close attention to anticoagulation is mandatory, and an international normalised ratio in the therapeutic range (2–3) should be targeted to prevent stroke.

## TRICUSPID REGURGITATION (TR)

### Aetiology

Most TR (> 90%) is ventricular secondary (i.e. due to leaflet tethering caused by right ventricular or annular dilatation).[51,52] Primary aetiologies (i.e. intrinsic valve disease) are uncommon (e.g. IE, cardiac implantable electronic devices, congenital abnormalities of the tricuspid valve, and RHD). Atrial secondary TR is recognised as another aetiological group, in which right atrial and tricuspid annular dilatation lead to non-coaptation of the valve leaflets.[53]

### Assessment

Multiparametric, transthoracic echocardiography should be used to determine the mechanism and severity of TR (Table V).[54]

### Management

TR intervention comprises surgical valve repair, replacement, or transcatheter valve repair.[55] Treatment is indicated for symptomatic patients with severe primary TR, and in asymptomatic or mildly symptomatic individuals with right ventricular dilatation or declining right ventricular function. Although the survival benefit of tricuspid valve intervention for severe, primary TR is not well established, it can be performed safely in selected patients without severe right ventricular or LV dysfunction and without severe, pre-capillary pulmonary hypertension.

The mortality risk of surgical intervention for isolated TR (i.e. in the absence of concomitant valve disease) is in excess of 10%.[2] Tricuspid valve repair should be performed in patients with severe secondary TR who are undergoing left-sided valve surgery, as well as in those with mild or moderate TR who have dilated tricuspid annuli (a predictor of progressive TR worsening in this population).[56]

Since adding tricuspid valve repair to left-sided valve surgery does not increase operative mortality compared with the very

**TABLE V: Severity grading of tricuspid regurgitation and stenosis.**

| | |
|---|---|
| **Severe tricuspid regurgitation** | |
| Tricuspid valve morphology | Abnormal/flail |
| Colour flow regurgitant jet | Large central jet or eccentric jet impinging on wall at a Nyquist limit of 50–60 cm/s |
| Continuous wave signal of regurgitant jet | Dense or triangular with early peak |
| Vena contracta | > 7 mm at a Nyquist limit of 50–60 cm/s |
| Proximal isovelocity surface area radius | > 9 mm at a Nyquist limit shift of ≈ 30 cm/s |
| Hepatic vein flow | Systolic flow reversal |
| Tricuspid inflow | Dominant E wave ≥ 1 m/s in the absence of other causes of an elevated right atrial pressure |
| Effective regurgitant orifice area | ≥ 40 $mm^2$ |
| Regurgitant volume | ≥ 45 ml/beat |
| Enlargement of cardiac chambers | Right ventricle, atrium, or inferior vena cava |
| **Severe tricuspid stenosis** | |
| Mean transvalvular gradient | > 5 mmHg |

significant risk (> 10%) of late repair of isolated TR, it should be used liberally.[56] Valve replacement is performed when valve repair is not technically feasible or carries a high risk of TR recurrence. Transcatheter valve repair technologies appear promising, although randomised data remain limited.[57]

### The South African perspective

In a contemporary South African cohort of patients with moderate or severe rheumatic MR, moderate or severe TR was documented in 31%.[22] This aligns closely with the prevalence of TR reported in studies of rheumatic MR from Israel and Japan.[58,59] Nyaope (an intravenous mixture of heroin, cocaine, and antiretroviral drugs) abuse is an emerging cause of IE affecting the tricuspid valve.[17] In a recent local study, 60% of patients with nyaope-associated IE had tricuspid valve involvement. Transcatheter valve repair technologies are not commercially available in South Africa and are not currently offered to patients. Loop diuretics and use of c (MRAs) are the cornerstone of the medical therapy of tricuspid incompetence.

## TRICUSPID STENOSIS (TS)

### Aetiology

The most common aetiology of TS is RHD. Other causes are infrequently seen (e.g. congenital heart disease and carcinoid heart disease).

### Assessment

Valve morphology should be carefully assessed on transthoracic echocardiography to determine the aetiology and the suitability for surgical intervention.[60] A mean transvalvular gradient > 5 mmHg indicates significant stenosis (Table V).[12]

### Management

While diuretic therapy may benefit patients symptomatically, durable treatment requires mechanical relief of severe stenosis. Percutaneous balloon valvuloplasty may be considered in cases of isolated TS or when concomitant mitral balloon valvuloplasty is performed.[61] Surgical repair is possible in some patients, but most often valve replacement is required. Implantation of a bioprosthesis is preferred due to the higher risk of mechanical valve thrombosis in the tricuspid position.[12]

### The South African perspective

Sparse local data exist on the prevalence or aetiology of TS.

## MIXED AND MULTIPLE VALVE DISEASES

Mixed disease refers to both a stenotic and regurgitant lesion within the same valve. Multiple valve disease refers to lesions involving > 1 valve, with each lesion at least moderate in severity. The EuroHeart survey showed a 20% prevalence of multiple valve disease in patients with native valves; however, data on multiple valve lesions remain scarce.[62]

### Aetiology

RHD accounts for 8% of clinical heart failure in an urban, South African, black population, with the mitral valve being affected most frequently.[63] A contemporary study by Banderker, et al. found combined disease (MS/MR) and multiple valve disease in 38% and 29% of patients, respectively, mostly due to rheumatic origin.[14] The REMEDY study, which enrolled 3 343 patients from 12 African countries, India, and Yemen, showed that children in the first decade of life presented mainly with isolated MR, while mixed mitral and mixed aortic diseases dominated in the second decade of life.[10]

Another South African study revealed that concomitant primary rheumatic tricuspid valve disease was present in 29% of patients, with rheumatic moderate or severe MR.[22] Of these, 31% had moderate or severe TR with attendant tricuspid valve annular dilation (38 ± 7.2 mm).[22] Assessment for tricuspid disease is important in patients with mitral valve disease, considering the increased morbidity and mortality associated with untreated TR.[64] Multivalve involvement is not uncommonly seen in patients with IE.[14,65,66]

### Principles of assessment

The initial assessment of multivalve pathology is difficult. Many Doppler-derived severity parameters are validated for single-valve pathology only. Nevertheless, it is recommended to assess each valve individually against severity criteria and, subsequently, consider the combined haemodynamic interplay between the valve lesions to determine which severity parameters are useful, erroneous, or redundant.

The clinical presentation and signs, as well as haemodynamic impact, are most frequently determined by the most severe or dominant lesion.[67,68] In patients with balanced involvement of different valves, the pathophysiology and clinical features often reflect the proximal lesion, thereby masking the manifestations of the distal lesion.[67,69] In general, fewer load-dependent parameters should be employed.[69,70] An example is significant AR, which may not be apparent in a patient with severe MS.

Transthoracic echocardiography remains the first-line imaging modality, though more advanced imaging (TOE, CT, and CMR) is frequently required. NT-proBNP, exercise echocardiography, and cardiac catheterisation may also be used, especially in the setting of disproportionate symptoms.[69] A few common valve-specific echocardiography interactions and pitfalls include:[69,70]

- AS and AR: AR pressure half-time is unreliable; the simplified Bernoulli equation for AS gradient determination might not be applicable if the LV outflow tract velocity is elevated.
- AS and MS: The MS pressure half-time method for mitral valve area is unreliable. LFLG MS can occur (three-dimensional echocardiography for mitral valve planimetry is superior).

LFLG AS is common:

- AS and MR: The presence of AS can increase the MR volume. AS and MR jets may be confused on Doppler (look

for aortic valve closure; the AS trace will not extend into the isovolumic relaxation time as in MR).

- AR and MS: An AR jet can be mistaken for a MS jet (if the end-diastolic velocity is > 3 m/s, it is an AR jet). The continuity equation is unreliable when aortic valve flow is used as the reference flow. MS pressure half-time is unreliable for assessing mitral valve area. MS can blunt the increased pulse pressure and LV dilatation of severe AR.
- AR and MR: Doppler volumetric method using left-sided assessment of net forward flow is invalid. AR pressure half-time invalid. This combination is poorly tolerated and more likely to result in residual LV dysfunction, even post-operatively.
- MS and MR: Doppler mitral gradient reflects the severity of both MS and MR.

## Management: General principles and surgical considerations

Data on indications for intervention in mixed valve and multiple valve diseases are limited. Decisions are individually tailored to patients. In those with both regurgitation and stenosis of the same valve, management usually follows the guideline recommendations for the dominant lesion. In balanced disease, greater emphasis is placed on symptoms rather than solely on severity criteria. Generally, the decision to intervene in patients with symptomatic disease and 2 severe lesions is straightforward. In the case of 2 non-severe lesions, symptoms and the degree of LV impairment take precedence.

Age and comorbidities are crucial considerations, and risk stratification based on the combined procedure risk is also important. Both the EuroSCORE II and STS scores underestimate risk in patients with ≥ 2 valves requiring surgery; this is a research gap that requires more study. The risk of surgery is weighed against the risk of the natural history if the diseased valve is left untreated. The presence of concomitant CAD further complicates the decision process. Given the complexity of these decisions, collaboration between cardiologists and cardiac surgeons is paramount.

## Surgical management

VHD treatment may be percutaneous or via open-heart surgery, and may comprise valve repair or replacement. Mitral valve repair is rarely indicated in RHD.[67] In patients with AR/MR, chronicity may impact LV function (which may also persist post-operatively), necessitating early intervention.[71] Combined, severe MR and AR portend a high risk when surgical treatment of both valves is necessary.[71] Even when treated surgically, post-operative outcomes (persistent symptoms, LV dysfunction, and survival) are worse compared with isolated valve disease.[67] Combined MR and AR have a high risk for LV dysfunction in the young after surgery for RHD.[71] The indication for intervention should be based on LV size (dimensions and volumes) and surgical indications for MR, rather than on the higher threshold of LV size for isolated AR.[71]

Percutaneous treatment options for RHD AS are rarely employed, since isolated RHD AS is relatively rare. Before the era of TAVI, percutaneous aortic valvuloplasty was fraught with high complication rates of up to 25%.[67] In a study by Rifaie, et al., 100% success was achieved for RHD AS, though the sample size was small ($n$ = 9).[72] Transcatheter treatment for RHD AR is an active area of research, which may prove viable in the future.[73,74] In the presence of moderate or severe MS and moderate aortic valve disease, percutaneous treatment (percutaneous mitral commissurotomy) may have a role to buy time for subsequent surgical treatment of both valves; however, surgery is preferred in the presence of severe MS/severe aortic valve disease.

## The South African perspective

As highlighted by recent South African studies, multiple valve lesions are common in patients with RHD. However, outcome data remain sparse.

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STATISTICS MADE EASY

# Traditional statistics versus machine learning in clinical registries: A pragmatic workflow for matching methods to data and clinical questions

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## ABSTRACT

**This piece discusses the importance of data type, identification, and organisation for machine learning (ML) and neural network (NN) development, and the applicability of ML for statistical analysis in large clinical and physiological datasets, such as the South African Heart Association Registry (SHARE).**

**Core outcomes/key lessons**

To enable clinicians and researchers to:

- **Systematically assess their clinical dataset (registry data, e.g. SHARE) for variable types, dimensionality, sample size, missingness, and event rates.**
- **Understand when traditional statistical methods are sufficient, when regularised regression is preferable, and when more complex ML approaches are justified.**
- **Recognise common pitfalls (overfitting, multicollinearity, data leakage, mis-specified outcomes), and how to avoid them in both "classic" and ML settings.**
- **Apply a staged workflow to their own data, using the SHARE-transcatheter aortic valve implantation (TAVI) registry as an illustrative case.**

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## INTRODUCTION

Clinical and physiological datasets, such as national and institutional cardiovascular registries, are increasingly high-dimensional and heterogeneous, combining demographic, biometric, diagnostic, and laboratory data with longitudinal outcomes. In this context, there is growing enthusiasm for ML and NNs as powerful tools to complement traditional statistical approaches, particularly because they can, in principle, capture complex, nonlinear relationships and interactions when many covariates are present. We recently proposed using ML and NNs to augment the conventional analysis of clinical and physiological data, highlighting their potential to improve prediction and risk stratification in such multidimensional settings.[1] These developments have led to a perception that any large, clinically rich dataset is "ideal" for advanced ML or NN models.

However, in practice, the most appropriate analytic strategy depends critically on the data's structure and quality, as well as the clinical question being asked. The type of outcome (continuous, binary, time-to-event), the balance between sample size and the number of predictors, the degree of multicollinearity, the mix of categorical and continuous variables, missing data patterns, and the intended use of the model (prediction versus explanation) should be considered. All of the above influence whether traditional regression, regularised regression, or more complex ML methods are likely to be optimal. Simply applying an ML technique because a dataset is "large" or "multidimensional" risks overfitting, loss of interpretability, and, ultimately, limited clinical impact.

In this article, we investigate a general, practice-oriented perspective on an existing clinical database. Using a contemporary TAVI (Transcatheter Aortic Valve Implantation) registry as an exemplar, we suggest a structured way for clinicians and researchers to evaluate their data and align analytic choices with data characteristics and clinical goals.[2] Instead of focusing on the performance of one particular algorithm, we propose a pragmatic workflow that spans traditional statistical models, regularised regression, and more flexible ML approaches. We also discuss when each step along this continuum is likely to be appropriate. Overall, we aimed to provide a practical guide for clinicians, physiologists, and applied researchers working with registries and routinely collected clinical data. By navigating the considerations that arose in our own work, we illustrate how to move from an initial enthusiasm for "doing ML" to a more disciplined, question-driven, and data-aware selection of methods.

## A MOTIVATING CASE: THE SHARE-TAVI REGISTRY

SHARE is a multi-center, prospective, web-based registry of TAVI procedures in South Africa. It captures detailed clinical

histories, demographics, comorbidities, procedural characteristics, TAVI outcomes, complications (classified according to Valve Academic Research Consortium [VARC] definitions), and follow up information, including 30-day and longer term outcomes. This rich, routinely collected dataset was established to inform health policy, promote equitable access to TAVI, and benchmark local practice against international standards.

TAVI is increasingly offered to older patients with multiple comorbidities, in whom post-procedural risk stratification remains challenging. Although procedural success rates in South African TAVI programmes are high, existing risk scores demonstrate only modest discrimination for post-TAVI mortality and other clinically relevant outcomes. Traditional analyses of registry data, typically based on linear or logistic regression, make only partial use of the available high-dimensional information and often assume linear relationships between predictors and outcomes. This has led to interest in applying more flexible approaches, including regularised regression and ML methods, to improve the prediction of outcomes, such as pre-TAVI aortic valve area and 1 year mortality.

Our original analytic plan for the SHARE reflected this enthusiasm: we aimed to develop and internally validate regularised regression models (ridge, lasso, elastic net) to predict pre-TAVI valve area and 1-year mortality, and to explore nonlinear relationships between demographic and clinical variables (including sex and comorbidities) and these outcomes. As we engaged more deeply with the data and the modelling challenges, it became clear that the key questions were not only "which algorithm performs best?", but more fundamentally "what does the structure of this dataset permit, and what is the most appropriate level of model complexity given our goals?"

Specifically, before choosing a method, one should systematically evaluate:

- The number of events available per candidate predictor for the outcome of interest.
- The extent of correlation and multicollinearity among predictors (e.g. between related echocardiographic or laboratory variables).
- How variables are measured and coded (binary, ordinal, continuous, raw versus transformed, derived scores).
- Whether the dataset is truly "big" in a modelling sense, with many more predictors than observations, or primarily "clinically rich" but of moderate size.

These considerations can lead to analytic choices that differ, and sometimes are simpler, than initially envisaged. In the case of SHARE, we propose a general, stepwise workflow for moving from traditional statistics to more advanced methods, while respecting both the data and the clinical questions at hand. This is complemented with practical recommendations for clinicians and researchers using registries (Table I).

## Step 1: Know your data – the taxonomy of clinical datasets

Any analytic journey should begin with a clear understanding of the data at hand. Clinical registries like the SHARE-TAVI registry

**TABLE I: Practical steps and recommendations.**

| Step | Recommendation | Key points |
|---|---|---|
| 1 | Create a "data and question sheet". | Document outcomes (type, prevalence, followup), sample size and events, predictor types and counts, missingness/data quality, and the intended clinical use of any model. |
| 2 | Match method complexity to data and goals. | Start with descriptive statistics and simple regression. Use regularised regression when predictors are many/correlated. Reserve complex ML (ensembles, NNs) for problems where data volume and use case truly justify it. |
| 3 | Respect sample size and EPV constraints. | Calculate EPV for binary/time-to-event outcomes. Limit candidate predictors, interactions, and nonlinear terms to what the data can support and use penalisation and prespecification of key variables. |
| 4 | Prevent data leakage. | Define prediction time point clinically (e.g. preTAVI decision). Exclude variables only known after the outcome. Ensure feature construction does not inadvertently use future information. |
| 5 | Plan validation and transportability early. | Use internal validation (cross-validation, bootstrapping). Choose predictors that are routinely available across centres. Anticipate how casemix, coding, and practice differences may affect performance elsewhere. |
| 6 | Prioritise interpretability and clinical usefulness. | In moderatesized registries, treat regularised regression with thoughtful nonlinear terms as the "advanced default". Prefer models that clinicians can understand and implement, and link outputs to concrete decisions (risk thresholds, example scenarios). |
| 7 | Co-design with a multidisciplinary team. | Involve clinicians, statisticians, and data scientists from planning onwards. Use their combined expertise to define predictors, interactions, and use cases, and to iteratively refine models as data and practice evolve. |
| 8 | Report transparently and support reproducibility. | Clearly describe the cohort, predictors, outcomes, handling of missing data, model specification, validation, and metrics. Provide enough detail (e.g. code lists, transformations) for others to replicate and externally validate the work. |

*EPV: events-per-variable, ML: machine learning, NN: neural network, TAVI: transcatheter aortic valve implantation.*

can feel intuitively "large" and "complex". However, their statistical properties often place them in a very specific niche between small, singlecentre cohorts and true "big data". A simple taxonomy of data structure helps to avoid mismatches between methods and data.

The outcome type strongly shapes the analytic options. In SHARE, pre-TAVI aortic valve area is a continuous outcome, while 1-year mortality is a binary event, and other followup measures could be considered time-to-event or longitudinal outcomes. Each of these outcomes raises different questions about scale, distribution, and appropriate performance metrics, naturally aligning them with different modelling families.[3]

Clinical registries often contain a mix of predictor types. SHARE includes continuous predictors (age and biochemical markers), ordinal variables (the New York Heart Association [NYHA] Functional Classification class or frailty scales), and nominal variables (valve type, sex, and centre), alongside counts (number of binomially labelled comorbidities) and composite scores. Each measurement scale carries implicit assumptions about how differences between categories should be treated, and these assumptions must be honoured or explicitly transformed when moving from traditional regression to ML methods.[4,5]

Dimensionality matters more than sheer row count.[6] A registry may enrol a few thousand patients, but if it captures dozens of demographic, clinical, imaging, and laboratory variables, the ratio of sample size to predictors, and, crucially, events-per-predictor for binary outcomes, may still be modest. This has direct implications for whether complex models can be stably fitted, and for the risk of overfitting when many predictors compete for a relatively small number of events.

Patterns of missing data and measurement error must be examined before selecting an analytic method. Routine registries often show non-random missingness (e.g. laboratory tests ordered only in more severe patients) and variable data quality across centres. While regularised regression and ML methods are sometimes perceived as robust "out of the box", they are no substitute for understanding and, where possible, addressing missingness and misclassification by design or through appropriate imputation strategies.

Combined, these dimensions (i.e. outcome type, predictor mix, dimensionality, and missingness) define a "data profile" that can guide method selection.[7] Many cardiovascular and procedural registries occupy a space that might be called "moderately high-dimensionality but not truly big data". They are rich in variables and clinically valuable, yet remain constrained by sample size, event counts, and data quality. Recognising this helps temper expectations that very deep or highly flexible ML models will necessarily be appropriate or outperform well-tuned regression-based approaches.

## Step 2: Map clinical questions to analytic goals

The second step is to articulate what the analysis is meant to achieve. Different clinical questions map to different analytic goals, and not all goals require – or are even compatible with – highly complex ML models.

A useful starting distinction is between prediction and explanation. In the SHARE context, predicting 1-year mortality after TAVI for individual patients is a prototypical predictive goal, with a focus on accurate risk estimates, and the tolerance for some loss of transparency may be higher if prediction improves meaningfully. Conversely, understanding how sex, renal function, or specific comorbidities relate to mortality, or how valve area changes with age and anatomical features, reflects an explanatory or mechanistic interest, in which the interpretability of effect estimates and their uncertainty are central. Methods that excel at prediction are not always the most informative for explanation, and vice versa.[3]

The outcome focus also matters. Continuous functional outcomes (e.g. pre-TAVI valve area) invite questions about calibration and mean error, whereas binary or time-to-event outcomes highlight discrimination, event prediction, and competing risks. Longitudinal trajectories (e.g. serial echocardiographic measurements or repeated quality-of-life scores) add further complexity and may require hierarchical or time-series approaches.

Finally, the intended use of the results influences the level of complexity deemed desirable. A bedside risk score integrated into clinical workflow demands parsimony and transparency, a model designed to identify quality improvement targets at the programme level can tolerate more complexity, and a model intended to generate mechanistic hypotheses may prioritise clear and robust associations over marginal gains in predictive accuracy – it all depends on the intended use and outcome. In SHARE, one might reasonably wish to develop a simple, implementable risk score for 1-year mortality, an institutional benchmarking tool, or exploratory analyses to determine which patient features drive adverse outcomes.

These distinctions (prediction versus explanation, outcome structure, and intended use) help determine where along the spectrum – from simple regression to complex ML – a given analysis should sit. In many registry settings, the combination of moderate sample size, mixed predictor types, and a strong need for clinical interpretability will favour methods that extend traditional models (e.g. regularised regression with carefully chosen non-linear terms) rather than immediate recourse to opaque algorithms.[6]

## Step 3: A continuum of methods – from classic to machine learning

Once the data profile and analytic goals are clear, methods can be viewed along a continuum rather than as competing camps. A simple conceptual "ladder" can help situate a registry with SHARE-like data.

The base of this ladder consists of descriptive and univariate analyses. These include distributions of key variables, cross-

tabulations, unadjusted associations between predictors and outcomes, and basic checks for missingness and outliers. In SHARE, such analyses might show, for example, the distribution of age and valve area, crude mortality rates by NYHA class, or simple differences between centres.[3] Although sometimes dismissed as preliminary, this level is essential to understand the data and prevent later models from encoding artefacts.

The next rung comprises traditional multivariable models with linear regression for continuous outcomes, logistic regression for binary outcomes, and Cox models for time-to-event data. These are familiar to clinicians, conceptually straightforward, and can often be implemented with standard statistical software. Their strengths include transparency, direct estimates of effect sizes and confidence intervals, and well-understood diagnostics. Their limitations emerge in high-dimensional or highly correlated settings, where linearity assumptions may be implausible, multi-collinearity can destabilise estimates, and the number of candidate predictors threatens to overwhelm the number of events.

Regularised regression methods (ridge regression, lasso, and elastic net) occupy a middle rung and can be viewed as a bridge between classical regression and more flexible ML.[8] They retain the basic regression framework but add penalties on model complexity, shrinking coefficient estimates, and, in some cases, selecting variables. For SHARE, with a large number of patients and many potentially correlated clinical and echocardiographic variables, these methods are particularly attractive, as they address multi-collinearity, reduce overfitting risk, and can yield parsimonious models that remain interpretable for individual predictors.[9]

At the top of the ladder are more flexible ML approaches, including tree-based ensembles (random forests, gradient boosting), support vector machines, and various NN architectures. These methods can, in principle, capture intricate nonlinear relationships and high order interactions without explicit specification. Their advantages become most evident in truly large datasets, in problems involving unstructured data (images, waveforms), or where interactions are numerous and difficult to predefine. However, they often come at the cost of reduced transparency, more complex tuning, and sensitivity to data idiosyncrasies when sample sizes and event counts are modest.

Placed on this ladder, SHARE likely sits in the zone where enhanced regression through thoughtful variable encoding, non-linear terms, and regularisation is the most natural next step beyond traditional modelling. The registry's size and richness justify methods that can handle correlated predictors and mild non-linearities but may not support the stable estimation and validation of very highcapacity ML models (e.g. deep NNs), especially when the number of events is limited and external validation cohorts are not yet available.

## Step 4: A practical workflow for choosing methods

To make these ideas actionable for clinicians and applied researchers, it is helpful to distil them into a simple, reproducible workflow applicable to any clinical dataset, with SHARE as an illustrative case.

First, characterise the data using the taxonomy in step 1. For SHARE, this would mean documenting the total sample size, number of events for each outcome of interest, distributions and types of all candidate predictors, patterns of incompleteness, and any known measurement limitations.[9] This step results in a concise "data profile" that can be shared among collaborators.

Second, define the primary analytic goals and constraints as in step 2. For example, in SHARE, one might prioritise development of an internally validated prediction model for 1-year mortality, with secondary goals of understanding the role of specific comorbidities and generating a simple risk score suitable for routine use. Constraints might include limited events per predictor, absence of external validation data, and the need for a model that can be implemented without complex infrastructure.

In step 3, start with the simplest method that can reasonably address the question. For a continuous outcome (e.g. valve area), this might mean linear regression with a modest set of clinically selected predictors. For 1-year mortality, a logistic regression model with prespecified core predictors might serve as a baseline. These initial models set a reference point for performance and interpretability.

Evaluate model performance and calibration using appropriate internal validation as step 4. Cross-validation, bootstrapping, or split-sample approaches can be used to estimate outofsample discrimination, calibration, and error metrics. In SHARE, this step might show that a simple logistic model achieves only modest discrimination in mortality, suggesting room for improvement.

As a possible step 5, escalate complexity in a controlled fashion. If simple models underperform meaningfully, one can introduce regularised regression to handle larger predictor sets and multi-collinearity, or add carefully chosen nonlinear terms and interactions. For SHARE, this might involve moving from a small logistic regression model to an elastic net model, including a broader set of clinical and echocardiographic variables, while maintaining internal validation at each stage. More flexible ML methods should be considered only if (1) the data volume and signal-to-noise ratio justify their capacity; (2) the incremental gains in performance are likely and clinically meaningful; and (3) the implications for interpretability and implementation are acceptable.

At each step, regardless of the method, it is important to monitor for signs of overfitting, such as overly optimistic apparent performance relative to cross-validated estimates, unstable variable selection across resamples, or implausibly large effect sizes. Multi-collinearity should be assessed, at least

informally, to avoid misinterpreting coefficients. Finally, the clinical plausibility and implementation of model outputs should be scrutinised, and questions such as "Do the identified predictors and their directions of effect make sense?" and "Can the model be used at the bedside or in a policy context without specialised infrastructure?" should be asked. If the answer to these questions becomes less clear as complexity increases, this may be a signal to pause or reconsider. Characterise, clarify goals, start simple, validate, then escalate complexity only when justified by both data and clinical need.[4]

### Step 5: Handling nonlinearities and interactions without overcomplicating

Nonlinear relationships and interactions are often invoked as reasons to adopt complex ML models. Yet, many of the clinically important forms of nonlinearity and interaction can be accommodated within regression or regularised regression frameworks, preserving interpretability while capturing richer patterns.

In a SHARElike registry, one might suspect that age has a nonlinear association with 1-year mortality, that valve area exhibits threshold effects, or that renal function and frailty interact with procedural risk. These hypotheses can be addressed by incorporating transformations and spline functions into regression models. For example, age and key biomarkers can be modelled using restricted cubic splines, allowing their relationship with the outcome to bend smoothly without imposing a single global linear relationship.[10] Valve area could be transformed or segmented around clinically meaningful thresholds to reflect known physiology.

Interactions of clinical interest, such as sex by age, sex by renal function, or comorbidity burden (e.g. hypertension or diabetes) by frailty, can be prespecified based on prior knowledge and encoded as product terms in the model. When combined with regularisation, a model can include a richer set of plausible nonlinear and interaction terms while controlling for overfitting. This strategy allows the analysis to remain grounded in clinically interpretable quantities (e.g. how the risk gradient with age differs between men and women), rather than relying on opaque interaction structures learned automatically by an algorithm.

Whether the added complexity of these terms is justified should be evaluated empirically. In SHARE, one might compare a baseline logistic regression model with linear terms to an extended model including splines and key interactions, using internal validation to assess changes in discrimination, calibration, and clinical utility. If the extended model delivers only marginal gains at the cost of greater complexity, it may be preferable to retain the simpler specification. Conversely, if nonlinear terms substantially improve calibration across the age spectrum or better capture risk in specific subgroups, this justifies their inclusion without necessitating a shift to blackbox ML.

Importantly, this approach underscores that "handling nonlinearity" is not synonymous with "using complex ML".[4] Many clinically relevant nonlinearities and interactions can be captured by thoughtful modelling within regression-based frameworks, particularly when supported by regularisation and rigorous validation. For registries like SHARE, this may offer a balanced path that respects both the richness of the data and the practical needs of clinicians who must interpret and use the results.

**TABLE II: Common pitfalls and how to avoid them.**

| | Pitfall | Alternatives |
|---|---|---|
| **1** | Overinterpreting small, noisy ML models as "AI-driven" applications. | Clinical datasets are often smaller and noisier than they appear, with modest event counts and heterogeneous measurement quality. In such settings, highly flexible ML models can fit idiosyncrasies of the sample rather than the true signal, particularly when internal validation is weak or absent. Transparent reporting of sample size, event counts, and the validation strategy, combined with comparisons to simpler baselines, is essential to avoid overstating these models. |
| **2** | Ignoring EPV constraints. | For binary outcomes, too few EPV is associated with unstable estimates, overfitting, and overly optimistic apparent performance, regardless of whether conventional regression or ML is used. Explicitly calculating EPV and tailoring model complexity (including the number of candidate variables and interaction terms) to this constraint is a critical step in responsible model development. |
| **3** | Data leakage. | Data leakage occurs when information that would not be available at the intended time of prediction is inadvertently used during model training. In clinical prediction models, a classic example is including diagnostic codes or post-procedural variables that are only finalised after discharge in a model intended to predict in-hospital or early post-procedural outcomes. Careful temporal alignment of predictors and outcomes, along with a clear definition of the prediction time point, is needed to avoid this subtle but pervasive error. |
| **4** | Neglecting transportability and generalisability. | Models that perform well in their development registry may fare poorly when applied to new settings, populations, or time periods. Differences in case-mix, practice patterns, data coding, and measurement frequency can all compromise transportability. When external validation is not yet feasible, sensitivity analyses, causal reasoning about predictors and outcomes, and cautious claims about scope are important safeguards. |
| **5** | Presenting models without clear clinical use cases. | Explicitly defining who will use the model, at what point in the care pathway, and how the predictions will change decisions helps align methodological choices with clinical value. |

*AI: artificial intelligence, EPV: events-per-variable, ML: machine learning.*

## COMMON PITFALLS AND HOW TO AVOID THEM

Even when data and questions are well characterised, several recurring pitfalls can undermine the validity and usefulness of statistical and ML models in clinical registries (Table II). These pitfalls include the over-interpretation of data, where apparent "artificial intelligence-driven" performance becomes an illusion of overfitting and noise exploitation.[11-13]

For binary outcomes, it is easy to ignore events-per-variable, as in the case of SHARE, where 1-year deaths are relatively few compared to the dozens of available predictors. Attempts to fit very detailed models, particularly without regularisation, risk producing unstable coefficients and spurious variable importance. Similarly, data leakage can lead to inflated performance estimates.[14] For example, in a SHARE-based context, incorporating variables that are recorded after the TAVI procedure or using follow-up information to engineer predictors for a baseline risk model would constitute leakage, inflating performance estimates.

It is also possible to neglect a registry's generalisability. For instance, a SHARE-derived mortality model might rely on variables whose distributions or meanings differ across other centres or countries, leading to miscalibration and degraded performance elsewhere. Emphasising internal performance alone, without explicit consideration of external validation or likely shifts in population characteristics, can give a misleading sense of robustness.[15]

Lastly, a common pitfall is to develop and report models without articulating a specific clinical use case, mechanistic pathway, or implementation strategy. A complex TAVI risk model may be technically impressive, but if it is too cumbersome for bedside use, lacks clear decision thresholds, or does not address a clinical question, it is unlikely to influence practice. In a SHARE-like scenario, a model that predicts 1 year mortality but does not inform procedural selection, follow up intensity, or patient counselling risks, remains an academic exercise.

Combined, these pitfalls underscore that the value of a model lies not in its algorithmic sophistication, but in the way it respects data limitations, avoids methodological traps, and serves clearly defined clinical purposes.

## CONCLUSION

Clinical registries, such as the SHARE-TAVI registry, offer rich opportunities to improve risk stratification, understand treatment outcomes, and inform policy. Their multi-dimensional nature naturally invites interest in ML and other advanced methods. Yet, as this article has argued, the true power of ML in this context lies not in complexity for its own sake, but in disciplined, question-driven, and data-aware method selection. This stepwise framework emphasises that many registries are "moderately high-dimensional but not big data", and that for such datasets, thoughtful extensions of traditional models, including regularisation and carefully specified nonlinearities, may offer the most appropriate balance between performance and interpretability. Consequently, SHARE illustrates why model choice must be grounded in data structure, event counts, measurement quality, and intended clinical use. The framework we propose is intended to help clinicians, researchers, and registry owners navigate this landscape, enabling them to harness both traditional statistics and ML in ways that respect their data and ultimately serve patients.

**Conflict of interests : none declared**

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WEBINAR: VEIN GRAFT INTERVEN-TIONS

# SASCI-Mayo Clinic Fellows webinar: Vein graft interventions

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## ABSTRACT

This publication is the third instalment in a series of webinars conducted jointly by the South African Society of Cardiovascular Intervention (SASCI) and Mayo Clinic. Hosted by the regular faculty, the webinar began with an in-depth analysis of the pathogenesis of vein graft disease by Dr Barsness. It included a critical appraisal of published data supporting various interventional strategies in this patient population. His presentation was followed by a clinical case study by Dr Engelbrecht, focusing on a patient with recurrent acute coronary syndrome (ACS) events due to vein graft disease. Cardiology fellows from various South African universities participated as discussants.

**Objective:** This manuscript, derived from the webinar series, summarises a multidisciplinary discussion of vein graft intervention complexities. It addresses the underlying pathophysiology, technical considerations, and current evidence-based management strategies.

**Case summary:** A male patient with prior coronary artery bypass grafting (CABG) presented with recurrent episodes of ACS secondary to progressive vein graft disease. The discussion explored the pathogenesis of vein graft disease and the technical challenges of intervention, with a specific focus on the evidence and clinical considerations when deciding between vein graft and native vessel revascularisation. Following recurrent ACS events and percutaneous coronary intervention (PCI) attempts in the diseased vein graft, the patient eventually achieved successful revascularisation through a chronic total occlusion (CTO) procedure in the native left anterior descending artery (LAD).

**Key messages**

- Pathophysiology of vein graft failure: Understand the pathogenesis and underlying mechanisms contributing to the long-term failure of saphenous vein grafts (SVG).
- Procedural complexities: Recognise the technical challenges and complications associated with vein graft interventions, with a focus on the no-reflow phenomenon.
- Mitigation strategies: Evaluate clinical strategies and pharmacological interventions to prevent and manage the no-reflow phenomenon.
- Comparative evidence: Review contemporary evidence guiding the choice between native vessel and vein graft PCI, including an evaluation of the recent PROCTOR trial.
- Clinical considerations: Outline key considerations for native vessel revascularisation in patients with previous CABG.

**Online resource:** Recorded SASCI fellows webinars (restricted to verified healthcare professionals) are available from: https://www.sasci.co.za/content/page/sasci-educational-videos1.

**Keywords:** CABG, vein graft interventions, ACS in CABG, vein graft vs native vessel PCI, pathogenesis of vein graft disease, cardiology education, SASCI fellows webinar.

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## INTRODUCTION

**SASCI convenor:** We are privileged to discuss the important topic of vein graft interventions. There is comparatively little evidence guiding our practice, but the recent publication of new data has renewed dialogue on what constitutes optimal management in patients with degenerated vein grafts. In keeping with our established format, we will begin with a general overview of the topic, followed by an interesting case presentation.

### Overview of vein graft interventions (Barsness)

Despite the risk and clinical effort put forth for open-heart surgery, vein grafts themselves are, at best, palliative. They have a high failure rate over time. Its mechanism can be divided into 3 timeframes:

1. Early (weeks to months): Technical issues or conduit/flow problems.
2. Intermediate (1–5 years): Fibromuscular proliferation and intimal hyperplasia.
3. Delayed (> 5 years): Accelerated atherosclerosis with accumulation of friable, necrotic "gruel"-like plaque.

At 10 years, only about 50% of vein grafts remain patent. The VEST trial randomised patients undergoing CABG to standard vein graft harvest and implantation techniques or use of an investigational external support device to improve graft patency. In this contemporary trial, the rate of vein graft failure in the

control group treated in the usual way was 25–30% at 1 year.[1] On a per-patient basis (because patients usually get more than 1 vein graft), about 40–50% of patients who underwent bypass surgery suffered from a failed vein graft within a year. So, whether on a per-graft or per-patient basis, the failure rate is very high, even in a contemporary series. Thus, if you are caring for patients with vein grafts, you will see disease accumulate, and you'll have to deal with it in some way.

Management selection for this progressive vein graft disease, whether it is repeat surgical intervention or PCI, depends on clinical and anatomic realities in this patient population. We know that surgical risk increases with low ejection fraction, age, female sex, long perfusion times, and triple-vessel disease. Moreover, many of these risk factors describe patients presenting post-bypass with recurrent disease. Hence, people with recurrent vein graft disease are at high surgical risk.

Beyond clinical status, we consider anatomy when deciding between repeat bypass and percutaneous revascularisation. Repeat surgical revascularisation can be an attractive option in some young patients with multivessel disease and multiple failed grafts, especially given the accelerated proximal native vessel disease progression often seen in patients following CABG. Therefore, we would consider repeat bypass in patients with ischaemia in the LAD territory, especially if an unused arterial conduit (the internal mammary artery) is still available. However, we consider vein graft PCI in most patients for clinical and anatomic reasons. In ACS, we think about it with unfavourable native target anatomy within 1–3 years with focal vein graft disease, or especially when there is patency of a prior arterial graft, usually to the LAD.

Until recently, I had been confident that the suitability of a native vessel as a target for PCI was a good reason to proceed with native vessel PCI rather than high-risk, repeat CABG or vein graft PCI. Despite a lack of data, I was intuitively certain that treating native vessels was the right approach in patients with vein graft disease. I'm going to talk about that for a second and why that may or may not still be true.

Long-term outcomes in a Mayo Clinic propensity-matched model suggest worse outcomes in patients who undergo PCI of the diseased vein graft compared with PCI of the native vessel, with a persistent, clinically and statistically significant effect for 10–15 years. This long-term outcome, at least in our series, suggests a benefit of either native vessel intervention or even repeat bypass surgery in these patients.[2] A large pooled analysis of 22 observational studies of 40 000 patients with 2-year follow-up reported consistent findings, suggesting that, compared with vein graft PCI, native vessel PCI was associated with reduced major adverse cardiovascular events (MACE), all-cause death, myocardial infarction (MI), and target vessel revascularisation – all largely statistically significant and with a large risk reduction across all those endpoints, with no difference in acute bleeding or stroke.[3]

Consequently, it seemed like native vessel PCI was the clear winner and should be the preferred option when feasible in patients with recurrent vein graft disease. Then, more recently, many of you will be aware of the PROCTOR trial, which prospectively randomised post-bypass individuals to either native vessel intervention or vein graft intervention. They found that native vessel PCI had a 34% MACE rate at 1 year, whereas vein graft PCI had an event rate of about half that, resulting in significantly reduced adverse events and a clear, statistically and clinically meaningful benefit favouring vein graft intervention.[4] All-cause mortality was not much different, yet there was a significant difference regarding PCI-related MI and repeat revascularisation.

There are a few noteworthy limitations to the study. The trial was stopped early due to funding and enrolment issues, but the investigators were still able to conduct a statistically meaningful endpoint analysis. However, stopping a study early always has its concerns. It was also an open-label design, which may have influenced clinical decision-making, particularly regarding revascularisation options. There was also a short 1-year follow-up, and I'm going to emphasise why that's important. Furthermore, this was a select population that excluded early graft failure, occluded vein grafts, and high-risk vein grafts. We'll talk about what that entails. Moreover, 72% of the treated native lesions were actually CTOs, mostly treated with a retrograde approach, and the stent length was almost 9 cm in patients with treated CTOs, compared with 2 cm in the vein graft patients. So, it speaks to the complexity of the native vessel treatment in this particular trial, as well as its applicability to the treatment of our own patients.

Conversely, a vein graft intervention study from SCAAR (Swedish Coronary Angiography and Angioplasty Registry) shows that at 3 years, all-cause mortality was 20%, MIs occurred in 20%, and any revascularisation in nearly 40%.[5] These population-based event rates are really an important sort of caveat when thinking about vein graft interventions.

Let's look at a case. Here, we see a patient with relatively focal, mid-vessel disease (at least the tightest portion is quite focal). It is easy to wire, but after a single balloon inflation, there is no-reflow, with a tenfold increase in infarction and mortality. So how do we identify this risk and subsequently manage it?

## Anticipating no-reflow

It turns out that plaque volume plays a big role. Plaque volume is directly related to the risk for no-reflow and adverse events associated with vein graft intervention, which can be substantial even early after CABG. Plaque volume can be assessed either by an integrated assessment all along the graft or by using a scoring system (semi-quantitative vein graft degeneration score).[6]

## How to prevent no-reflow

Embolic protection devices (EPD) have some benefits. A pooled analysis of the trials demonstrated the general benefits of EPDs, as well as a relative risk reduction in each quartile of degeneration score.[6,7] This just means you can't really predict who won't

benefit from embolic protection. However, you know that the higher the degeneration score, the higher their absolute risk, and that relative risk reduction translates to a greater absolute risk reduction in these patients. EPDs, at least in this study, showed a clear benefit compared with standard care in the early years. However, not everyone uses an EPD, and it's not universally used in most labs. The reason might be ascribed to a meta-analysis of multiple observational studies and 2 randomised trials, which did not show a statistically significant difference in MACEs with EPDs, leading to a downgrade in United States guidelines from class I to class IIA. There are also technical challenges with EPDs, like how to use them in a distal vein graft lesion or in tortuous anatomy.[8]

Other interventions for management or prevention of no-reflow and distal embolisation include good peri-procedural anticoagulation, which should go without saying. However, GP2B3A inhibitors have a class III indication and should not be routinely used. Other agents such as nicardipine, verapamil, nitroprusside, and adenosine can be considered. It's possible to use these agents to pre-treat a vein graft. In most cases, you can give the agent through the guide. However, when treating no-reflow, it is important to give these agents distally (e.g. using a microcatheter). Other considerations include direct stenting with non-aggressive stent sizing, avoiding pre-dilation and other vessel manipulation as much as possible. In essence, when intervening on a vein graft, we are slightly limited in the tools available with proven efficacy. Regarding PCI, a DES is preferred to POBA in most instances.

### Summary

- The new data from the PROCTOR trial at least gives us pause to consider the benefit of treating focal vein graft lesions, even if the native vessel is amenable to revascularisation, especially if it's a CTO.
- If the vein graft is heavily diseased and there is native vessel disease, even a CTO that can be addressed, treating the native vessel disease in an antegrade fashion, or using the vein graft as a conduit for retrograde CTO treatment, is certainly a reasonable option.
- Optimal medical therapy, as always, is essential.

## CASE PRESENTATION

**Fellow 1:** I'll be jumping straight into our case. A 77-year-old gentleman has cardiovascular risk factors of diabetes, hypertension, and dyslipidaemia. He had CABG in 2008 with left internal mammary artery (LIMA) to LAD, a vein graft to his circumflex, and a vein graft to his first diagonal. In December 2017, he presented with life-limiting angina and a stress echo, which showed inducible ischaemia in his posterior lateral wall. Angiography showed a CTO of his proximal LAD with a dominant left circumflex artery (LCx) system with severe stenosis in the mid-circumflex. Regarding his right system, he had a CTO of his distal right coronary artery (RCA). On injection of his grafts, he had an occluded SVG to the LCx. The SVG to the diagonal was patent and filled the LAD retrogradely. The LIMA was small and atretic, likely due to competitive flow from the SVG to D1. The decision was made to treat the lesion in the LCx with a stent, as it was thought to be responsible for the inducible ischaemia.

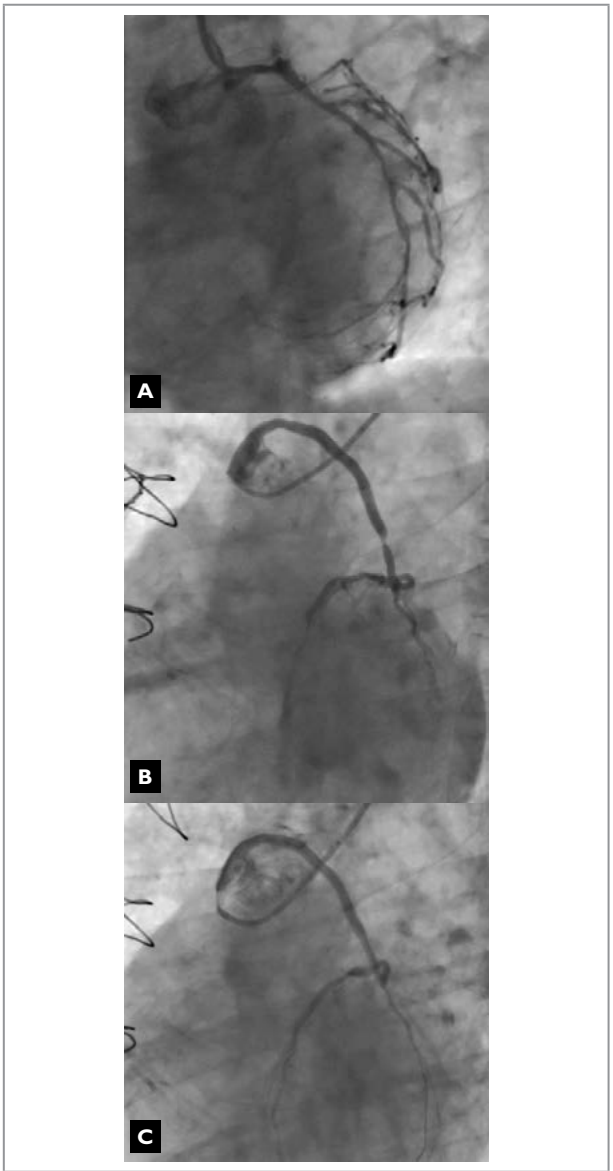


**FIGURE 1: (A) LAO caudal view demonstrating native vessels with CTO of the proximal LAD, (B) vein graft injection demonstrating severe distal lesion in the SVG to diagonal (note concomitant filling of LAD), (C) SVG after PCI with stent to lesion.**
*CTO: chronic total occlusion, LAD: left anterior descending artery, LAO: left anterior oblique, PCI: percutaneous coronary intervention, SVG: saphenous vein graft.*

The same patient presented to us 7 years later in 2024, with non-ST-segment elevation myocardial infarction (NSTEMI) and a troponin leak of 146. Another angiogram was performed, showing findings similar to those before, except that SVG to D1 now had a focal, discrete lesion in the distal vein graft, which was the probable culprit (Figure 1). So, what do we do next?

**Fellow 2:** If you consider that the LAD is effectively being supplied via the diseased graft due to the atretic LIMA, then the diseased SVG becomes a very important vessel. Additionally, it appears anatomically favourable for PCI; a focal lesion in a vein graft that appears otherwise healthy and of good calibre. Alternatively, you could consider a CTO procedure of the native vessel. However, considering the lesion's focal nature and the recent PROCTOR trial, it is probably worthwhile to address the vein graft lesion with PCI.

**Mayo faculty:** This is a fantastic case because it brings up everything we don't know about vein graft intervention. Firstly, this is an ACS case, and it is clearly a high-risk setting in which the patient is truly dependent on this vein graft. We don't have any data about treating vein graft lesions in ACS. There's not a single trial that we can rely on. There are anecdotes, and that's all. But, I will say that apart from the fact that this is ACS, which was not included in the PROCTOR trial, this is the kind of lesion that would have been included in the trial – a very focal vein graft lesion and complex CTO of the LAD. At this point, the data suggest that performing vein graft intervention would yield the best 1-year outcome for this patient. Unfortunately, it is just distal enough to preclude you from using an EPD, especially for the retrograde LAD territory.

A few more technical considerations (remember we are trying to avoid no-reflow):

- Ensure adequate anticoagulation and consider pre-treating with some antithrombotic agent to good levels.
- We don't have any data on GP2B3A inhibition in this case; in fact, we have negative data for GP2B3A inhibition in this patient cohort.
- Remember, it is crucial you do not putter around with this lesion. You wouldn't pre-dilate it because every time you touch it, you increase the potential for problems. You don't want to do much post-dilatation either.
- Regarding stents, you don't necessarily want to oversize or undersize them. You want to treat it with the appropriately sized balloon and ensure the procedure goes well. However, in this particular case, perfection is the enemy of good.

**SASCI convenor:** Ideally, you would like to perform direct stenting (sized 1:1) and provide sufficient inflation to achieve good initial expansion. This vein graft is more than a decade old, so you must anticipate fibrosis and gruel throughout the graft. Even though we don't see irregularities, we know the whole graft is lined with atherosclerotic "junk".

**Fellow 1:** We proceeded with PCI to the SVG lesion. We pre-dilated with a 2.5 × 15 mm semi-compliant (SC) balloon (considering our recent discussion, this might not have been the best strategy), then deployed a 3.5 × 15 mm drug-eluting stent (DES). Our result was good with maintained flow (Figure 1C).

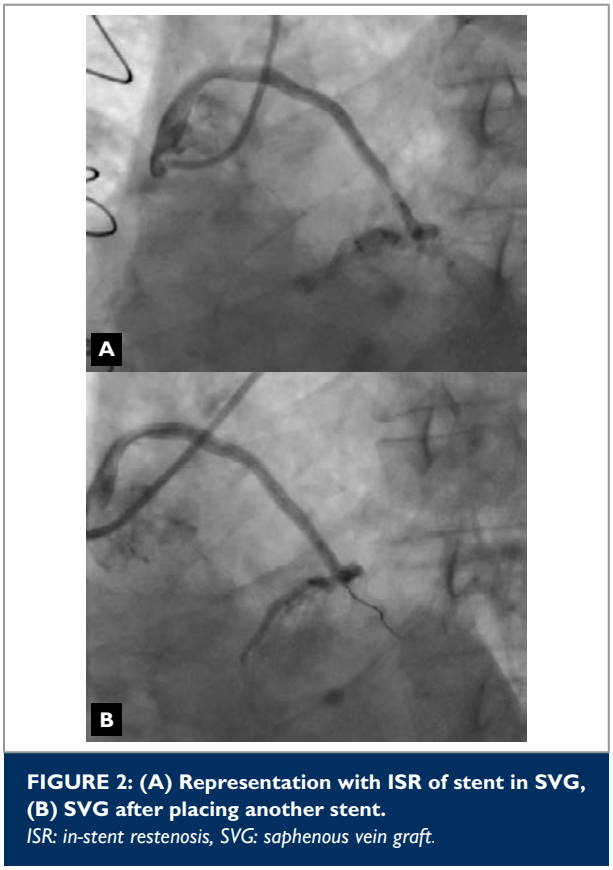


**FIGURE 2: (A) Representation with ISR of stent in SVG, (B) SVG after placing another stent.**
*ISR: in-stent restenosis, SVG: saphenous vein graft.*

Unfortunately, we saw the patient again 5 months later. He presented with another NSTEMI and troponin leak of 625. We performed a new angiogram; the native vessels were unchanged, but there appeared to be in-stent restenosis (ISR) or thrombosis at the distal end of the previously placed stent. We decided to re-intervene on this vein graft. We pre-dilated once more and deployed another 3.0 × 15 mm stent with distal overlap of the old stent. We were quite happy with our result (Figure 2). The patient did well for 9 months. Unfortunately, he presented again with NSTEMI and troponin leak of 922. A repeat angiogram showed recurrent ISR in the distal vein graft. The patient adhered to his medication, and his diabetes was under control, but he did have worsening chronic kidney disease (CKD), which is a risk factor for ISR

**Fellow 2:** I would like to optimise all the patient's risk factors and consider prolonged dual antiplatelet therapy (DAPT) in this case, depending on the bleeding risk. I am reluctant to place another stent; instead, I would consider a drug-coated balloon (DCB) as an option. Also, given that this is his third presentation, it may be time to adopt a different strategy, like native vessel intervention. Another option would be repeat CABG with right internal mammary artery (RIMA) grafting, if feasible.

**Mayo faculty:** Regarding the DCB strategy, the pathology here is different from that in native vessel disease. You have this "peanut butter" inside the vessel, and coating it with a DCB probably won't make much difference.

**Fellow 1:** We were reluctant to intervene a third time, so we opted for medical therapy and intensified low-density lipoprotein (LDL)-lowering. Unfortunately, our plan was short-lived, as we saw him again 1 month later with another NSTEMI. The repeat angiogram showed the same filling defects in the stent portion of the SVG. We thought that the LAD territory was still viable. To summarise, this is a patient with recurrent ACS episodes who had repeated interventions on the SVG and complex CTO of the LAD. We think that our only option here is native vessel PCI and opening the LAD CTO. Any thoughts on this before we continue?

**Mayo faculty:** This is going to be a challenging intervention. There is a retrograde conduit, which was how most of the CTOs were treated in the PROCTOR trial. But the anatomic complexity and calcium burden are high, making intervention potentially difficult. Nonetheless, it is the only real option at this point. I would likely take a dual-injection approach with a guide in the left main stem (LMS) and another guide in the graft, and try to work via a retrograde approach, as getting into the LMS from the LAD will be easier than the reverse.

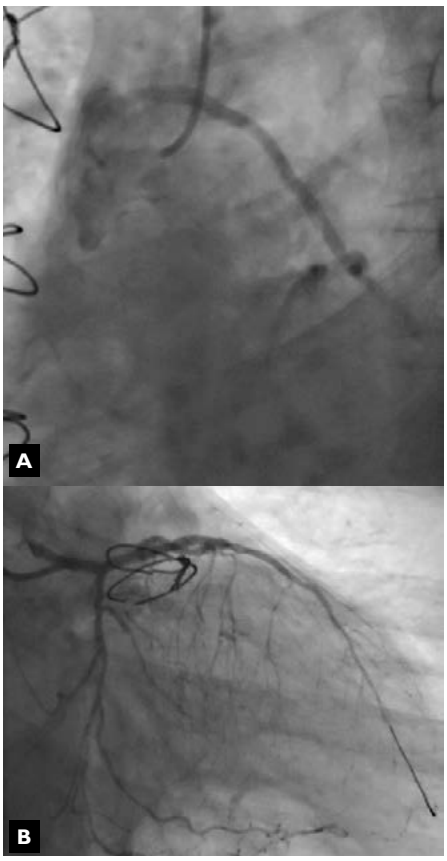


**FIGURE 3: (A) Patient represented with ACS and recurring disease in the vein graft, (B) native vessel treated with CTO procedure of LMS into LAD.**
*ACS: acute coronary syndrome, CTO: chronic total occlusion, LAD: left anterior descending artery, LMS: left main stem.*

**Fellow 1:** We proceeded with the CTO procedure, opting for an initial anterograde approach. We used the right radial access, with an EBU guide. We employed anterograde wire escalation, using a PROGRESS 40 guide wire after failing with a BMW and Fielder XT-A. We were able to deliver the PROGRESS wire into the distal LAD using a FineCross microcatheter. We were unable to deliver balloons over the PROGRESS, so we changed to a Grand Slam wire via the microcatheter for extra support. We could then pass a MINI TREK balloon, progressively escalate to a bigger balloon, and pre-dilate all the way back to the LMS. We proceeded to deploy a 3.0 × 48 mm stent from the LMS into the LAD and used a provisional strategy with proximal optimisation technique (POT) and kissing inflation to optimise the LAD-LCx bifurcation. We were quite satisfied with the result (Figure 3).

**SASCI convenor:** This case highlights that vein grafts are not without problems. We probably faffed around with the vein graft disease for too long. Once they start closing, they don't want to be opened. Whenever you face a vein graft problem, consider tackling the native vessel. That brings our discussion to an end. It has been a terrific session. Thank you to our faculty, Prof Barsness, for the great talk, and Prof Holmes, for the great discussion.

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# ECG Quiz 72

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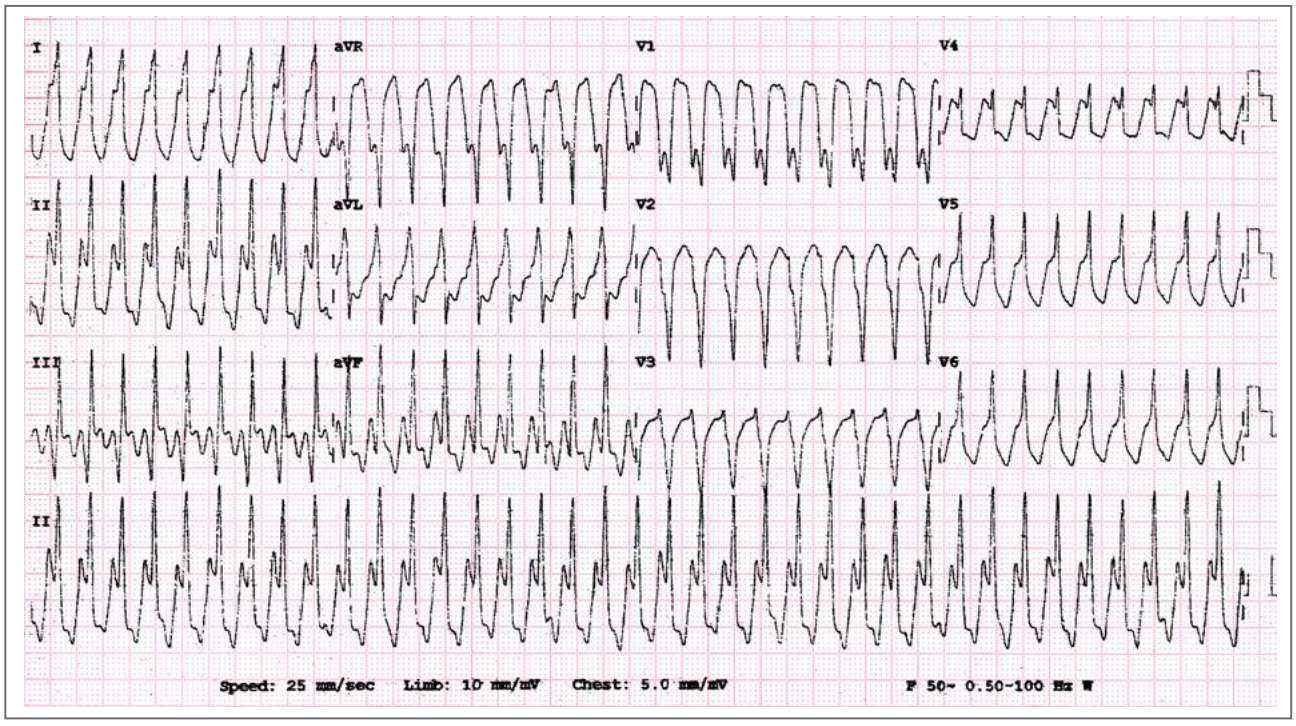
An 8-year-old boy presents with this arrhythmia.




## QUESTION 1: Which one of the following is the best diagnosis?

a. Ventricular tachycardia (VT).
b. Supraventricular tachycardia (SVT) with left bundle branch block (LBBB).
c. Antidromic atrioventricular re-entry tachycardia (AVRT) (Wolff–Parkinson–White [WPW] syndrome).
d. SVT with non-specific intraventricular conduction delay.

## QUESTION 2: What is the most likely clinical diagnosis?

a. Idiopathic right ventricular (RV) outflow tract (RVOT) tachycardia.

b. Arrhythmogenic right ventricular cardiomyopathy (ARVC).

c. Hypertrophic obstructive cardiomyopathy (HOCM).

d. Post-operative tetralogy of Fallot (TOF).

Please analyse the ECG carefully and commit yourself to an answer before checking the explanation.

**ANSWER** on page 110

# ECG Quiz 72

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ANSWER

## 1. OVERVIEW OF THE ECG

This is a fast (250/min), regular, wide QRS (190 ms) tachycardia. Therefore, the default diagnosis is VT (Table I), even in a young boy. However, the other options must be considered.

**TABLE I:** Differential diagnosis of regular, wide QRS tachycardias

- Ventricular tachycardia
- SVT with bundle branch block
- SVT with non-specific intraventricular conduction delay
- Antidromic AV re-entry tachycardia
- Pre-excited SVT
- Paced rhythm

*AV: atrioventricular, SVT: supraventricular tachycardia, WPW: Wolff–Parkinson–White*

## More detailed analysis of the ECG

The key to differentiating VT from SVT with bundle branch block lies in the QRS morphology in the chest leads. V1–V3 are negative, consistent with LBBB. However, the onset of the QRS to the nadir in V1–V2 is 115 ms (Figure 1). This excludes typical

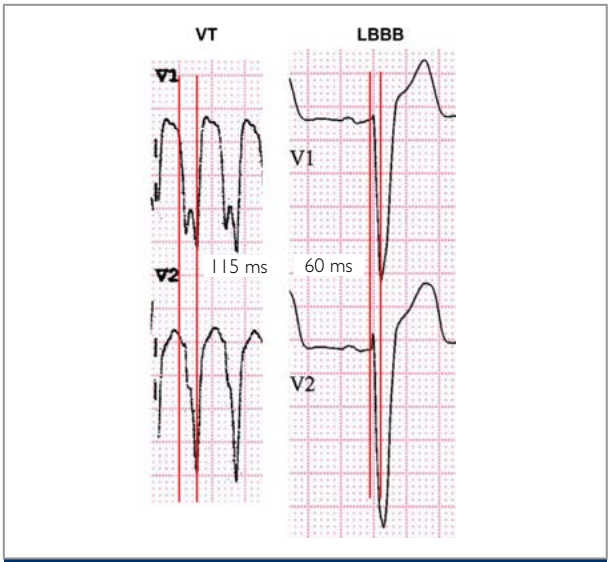


**FIGURE 1: The marked delay in the downstroke of the QRS in this tachycardia (115 ms) excludes LBBB (< 70 ms).**

*LBBB: left bundle branch block, VT: ventricular tachycardia.*

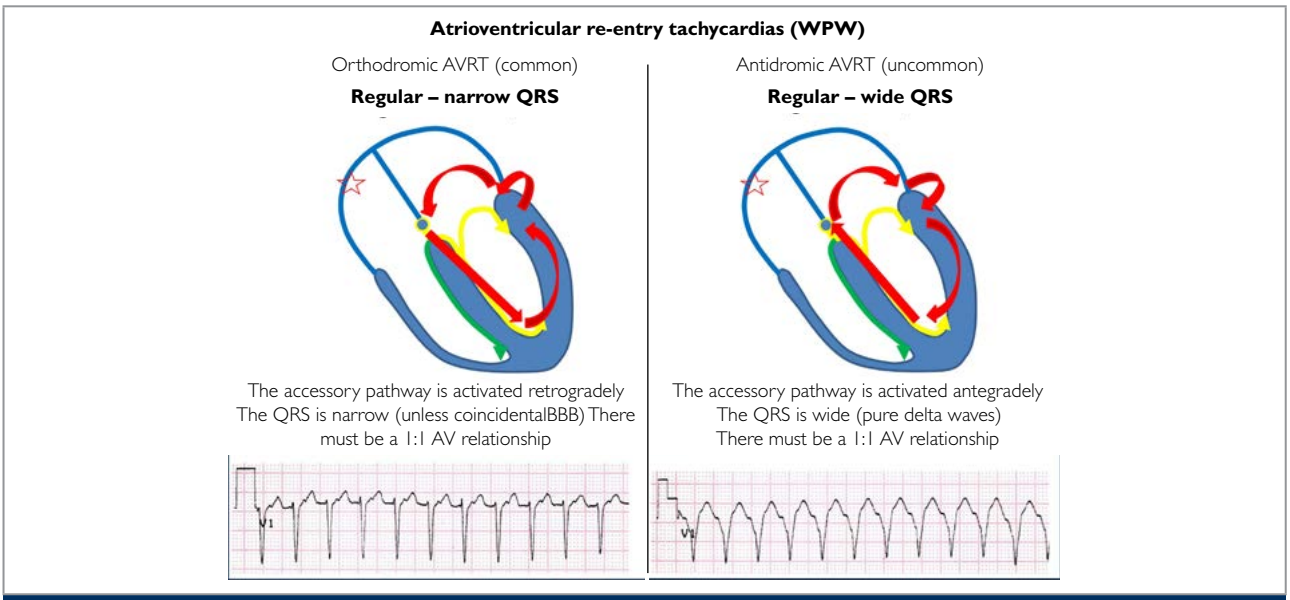


**FIGURE 2: Orthodromic AVRT (wherein the accessory pathway functions as the retrograde limb and the normal conduction system as the antegrade limb of the re-entry circuit) is by far the most common tachycardia in patients with WPW syndrome. Antidromic AVRT (wherein the accessory pathway forms the antegrade limb) constitutes < 10% of clinical tachycardias in WPW syndrome. The QRS is wide because the accessory pathway inserts into the ventricular muscle at the base of the heart and conducts slowly cell-to-cell until it reaches the His-Purkinje system and is conducted retrogradely to the atrium. In both cases, the atria are an obligatory part of the circuit.**

*AV: atrioventricular, AVRT: atrioventricular re-entry tachycardia, BBB: bundle branch block, WPW: Wolff–Parkinson–White.*

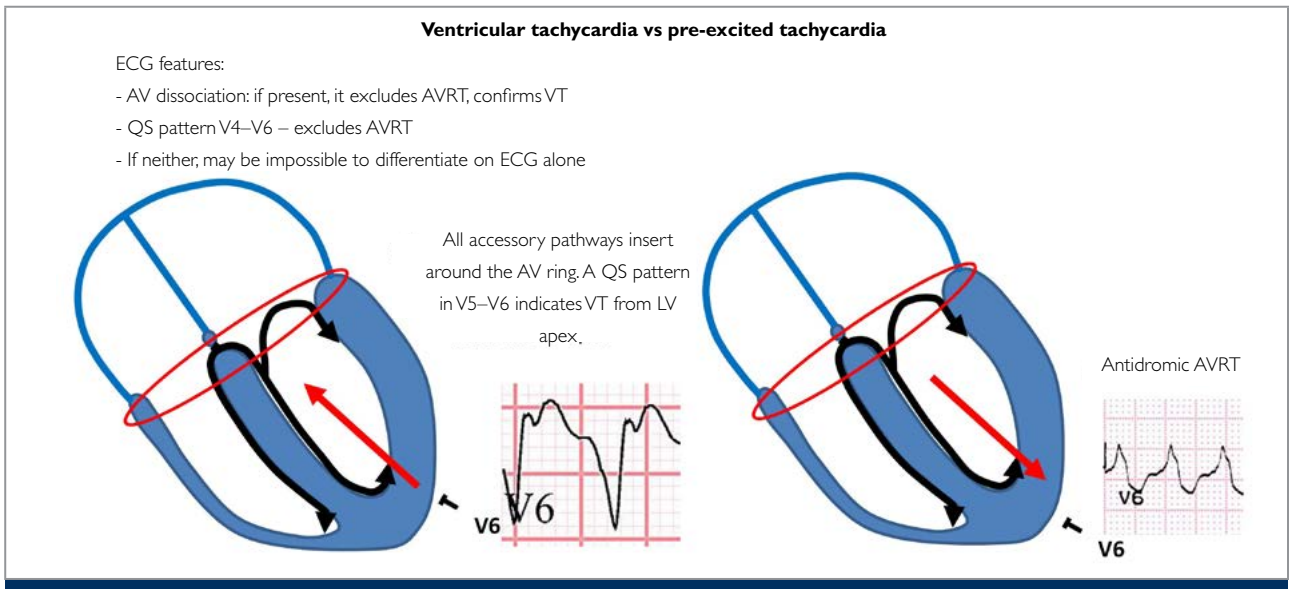


**FIGURE 3: The differentiation of pre-excited tachycardia from VT depends on the QRS morphology and the presence or absence of AV dissociation, as above.**
*AV: atrioventricular, AVRT: atrioventricular re-entry tachycardia, ECG: electrocardiogram, LV: left ventricular, VT: ventricular tachycardia.*

LBBB, which should not exceed 70 ms. Therefore, a diagnosis of SVT with LBBB is not tenable. An SVT with non-specific intraventricular conduction delay is a remote possibility. This only occurs in patients with severe left ventricular (LV) dysfunction due to extensive fibrosis. No clinical details or prior sinus rhythm ECGs (which would show the conduction delay) were given, but it would be very unusual at this age.

Antidromic AVRT is a serious consideration. It is the least common mechanism of tachycardia in patients with WPW syndrome, in which the accessory pathway forms the antegrade limb of the circuit. This results in slow cell-to-cell conduction (pure delta waves) and retrograde activation of the atrium via the normal conduction system (Figure 2). The vast majority of paroxysmal tachycardias in WPW patients are orthodromic AVRT, wherein the accessory pathway forms the retrograde limb, and the atrioventricular (AV) node forms the antegrade limb of the circuit. Therefore, QRS complexes are narrow, unless there is an incidental bundle branch block.

Differentiating antidromic AVRT from VT on the QRS morphology alone can be challenging. While some QRS morphologies, like negative QS waves in V4–V6, are specific for VT (Figure 3, all accessory pathways insert around the AV ring so the delta wave is always positive in V4–V6), this pattern is not present here, and antidromic AVRT cannot be excluded on QRS morphology alone. Looking for signs of AV dissociation now becomes crucial (Figure 3).

In AVRT (orthodromic and antidromic), there is an obligatory 1:1 ventriculo-atrial relationship. Tachycardia cannot occur in the presence of AV dissociation. Careful inspection of the SII rhythm strip reveals occasional dissociated P waves distorting the ST segments (Figure 4 arrows). This clinches the diagnosis of VT. Be aware that AV dissociation is not present or not visible in most VT ECGs, particularly at a rate this fast. A 1:1 retrograde VA conduction is common in VT and does not prove SVT diagnosis.

Therefore, the correct answer is (a): Sustained monomorphic VT.

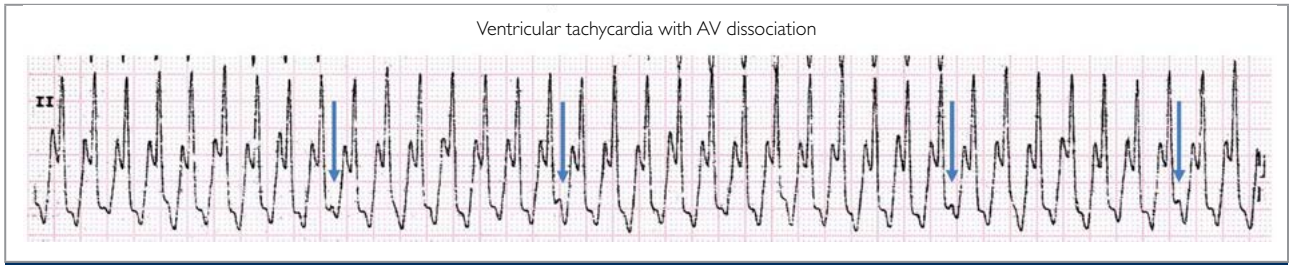


**FIGURE 4: Dissociated P waves (arrows) are visible in the rhythm strip as high-frequency distortions of the ST segment. This excludes antidromic atrioventricular re-entry tachycardia and clinches the diagnosis of ventricular tachycardia.**
*AV: atrioventricular.*

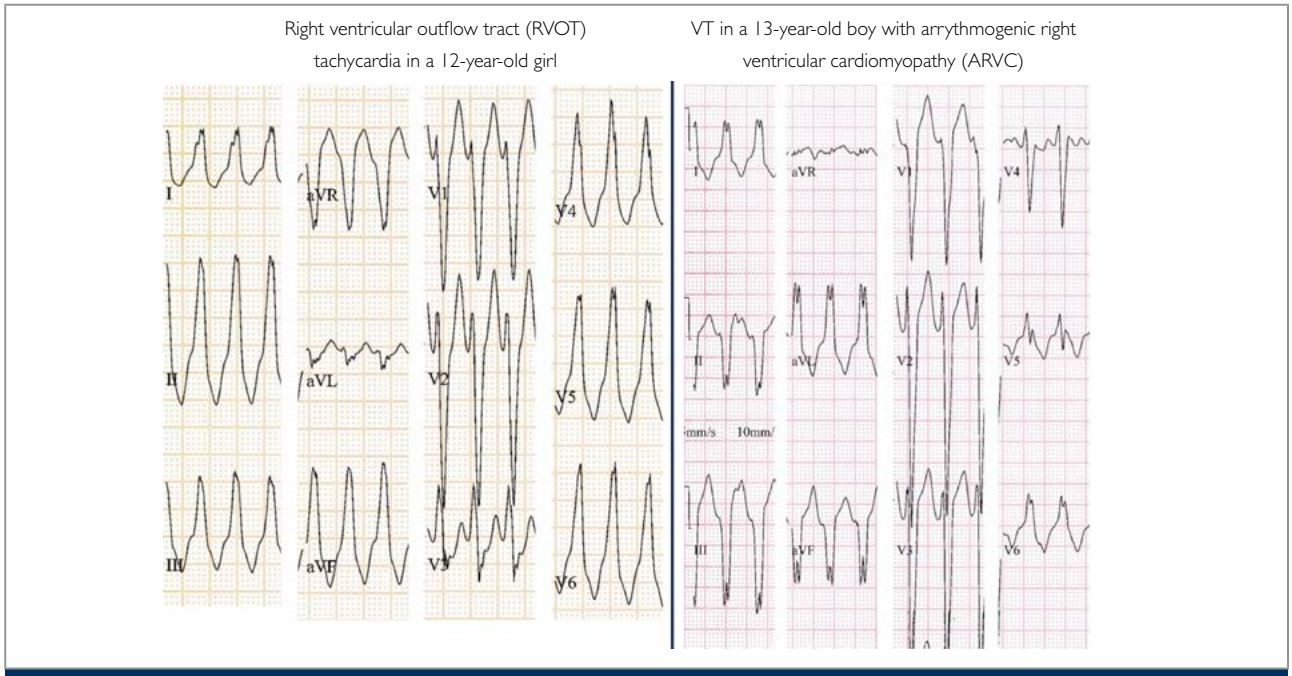


**FIGURE 5: Comparison of proven idiopathic RVOT tachycardia with VT caused by ARVC. The RVOT tachycardia has an inferior QRS axis, with only a small amount of notching and transition by V3. The ARVC VT has a left axis, indicating origin from the right ventricular body, notching in most QRS complexes, and a transition after V5.**
*ARVC: arrhythmogenic right ventricular cardiomyopathy, RVOT: right ventricular outflow tract, VT: ventricular tachycardia.*

## 2. WHAT IS THE MOST LIKELY CLINICAL DIAGNOSIS?

VT in children and young adults has various possible causes. The ECG in VT is not specific to the cause – clinical information and further cardiac imaging are now needed. Nonetheless, there are some helpful features. This tachycardia has a pseudo-LBBB pattern, suggesting a likely RV origin. The axis is about +60 °, suggesting it comes from the RVOT. Is it, therefore, idiopathic RVOT tachycardia, a condition of young people? Contrary to this diagnosis is the very wide QRS with marked notching (best seen in V1), indicating inhomogeneous ventricular activation. This is suggestive of scar-related re-entry, which is not a feature of RVOT tachycardia (Figure 5).[1]

ARVC is an important cause of VT arising in the right ventricle. However, the VT site is usually from the inflow tract or body, which would produce a superior QRS axis. Rarely, VT can also arise from the outflow tract, mimicking this ECG. The notched QRS complexes (best seen in the limb leads) would be compatible with this diagnosis and are probably related to extensive areas of fibrosis and fatty infiltration, which form the substrate for re-entry (Figure 5).

Non-sustained (< 30 seconds) VT is common in HOCM, but sustained monomorphic VT is less so. One mechanism for sustained VT in HOCM is an LV apical aneurysm.[2] Since HOCM predominantly involves the left ventricle, one would expect the VT to have a pseudo-right bundle branch block (RBBB) pattern

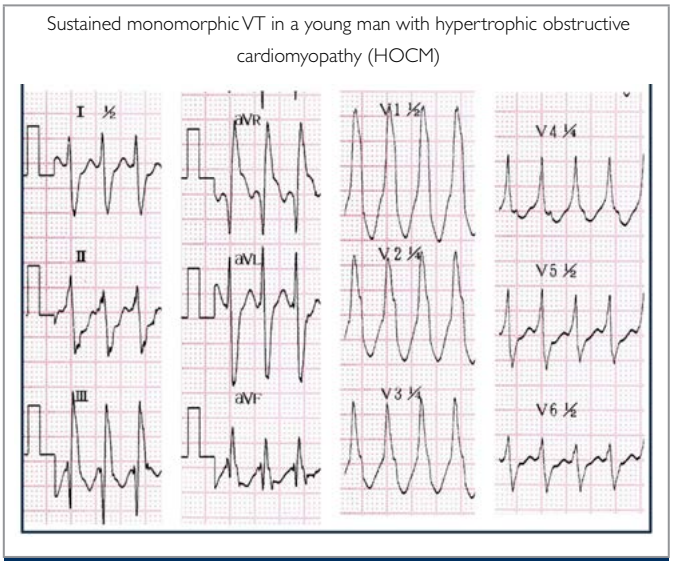


**FIGURE 6: This VT has a pseudo-RBBB morphology, indicating an origin from the left ventricle.**
*RBBB: right bundle branch block, VT: ventricular tachycardia.*

(Figure 6), unlike the ECG in this quiz. Consequently, HOCM is unlikely.

VT may complicate TOF, usually after surgical repair, which involves a right ventriculotomy in the RVOT and resection of muscle to relieve obstruction. The risk factors for VT include late repair, time after surgery, a QRS duration ≥ 180 ms,

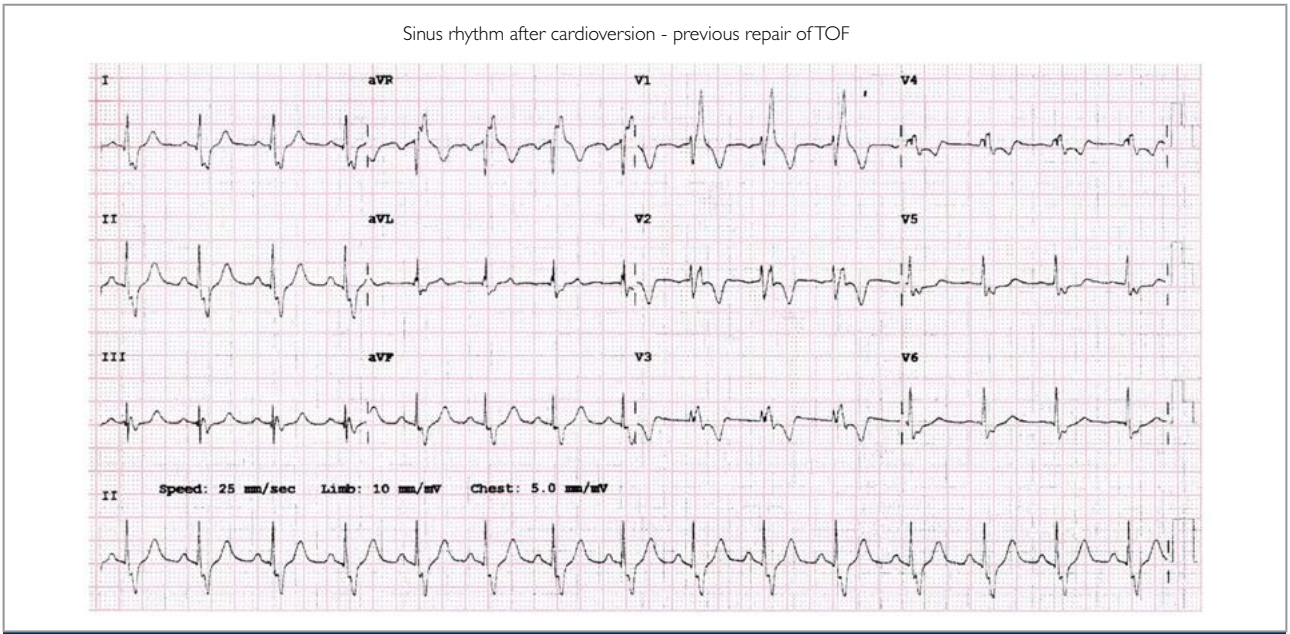


**FIGURE 7: The sinus rhythm electrocardiogram is typical of post-repair of TOF. Right bundle branch block is inevitable if there is a ventriculotomy in the right ventricular outflow tract.**
*TOF: tetralogy of Fallot.*

ventricular dysfunction, and atrial tachyarrhythmias.[3,4] The mechanism is re-entry around the surgical scar.[5] While most patients with VT related to surgery are teenagers or young adults, it is likely that this child had surgery as an infant (date unknown), as the ECG with VT was recorded in 2017. The post-cardioversion ECG (Figure 7) is typical of previously repaired TOF. There is complete RBBB with a QRS duration of 180 ms.

The most likely clinical diagnosis is (d): TOF.

Note that in the absence of clinical information, clinical imaging, or a post-conversion ECG, ARVC is also a possible diagnosis with an uncommon outflow tract VT.

## LESSONS AND CONCLUSIONS

- VT is the most likely cause of a regular, wide QRS tachycardia, even in children.
- AV dissociation, if visible, is diagnostic.
- The QRS morphology is useful to reveal the potential origin site of VT and may point to the cause.
- While surgical repair of TOF is essential for survival and quality of life, it is not a cure, so patients need to be followed up for life.
- Patients with post-operative TOF are at risk of developing VT and sudden death due to scar-related re-entry.

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# CARDIAC IMAGING QUIZ

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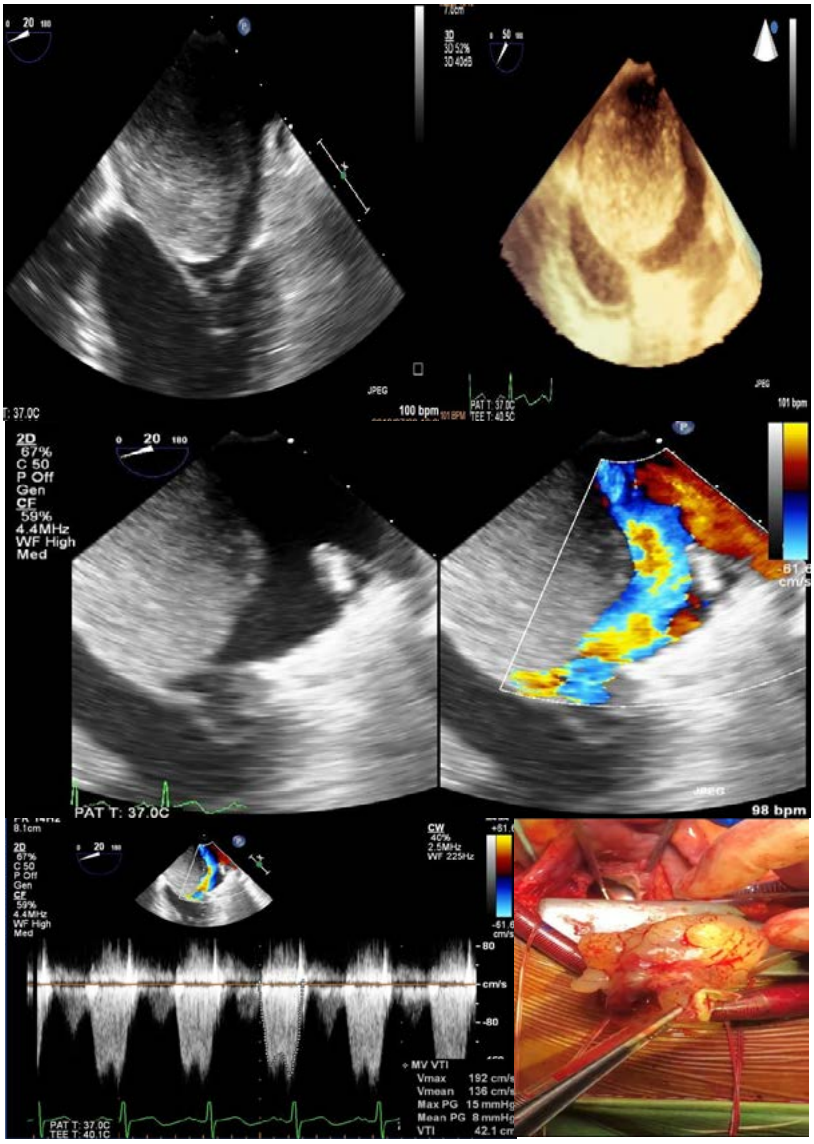


## QUESTION: What is the diagnosis?

A Mitral stenosis with thrombus

B Atrial myxoma

C Infective endocarditis

D Metastatic tumour

## ANSWER

**Correct answer:** B. Atrial myxoma

## CASE PRESENTATION

A young African male with no known comorbidities presented with clinical features of a cerebrovascular accident. On examination, an early diastolic sound ("tumour plop"), accompanied by a diastolic rumble and a loud first heart sound, was heard. The patient was not in heart failure and, on further history, admitted to on-and-off exertional dyspnoea. He had a normal electrocardiogram, and all his blood parameters were within the normal limits. At echocardiographic examination, a left atrial mass was noted. He underwent transoesophageal echocardiography for further evaluation. A large mass in the left atrium, attached to the interatrial septum, was observed (top panels). It straddled the aortomitral curtain and superior aspect of the anterior mitral valve leaflet (middle panels). The mass oscillated in and out of the left ventricle inlet, partially obstructing the mitral orifice in diastole and mimicking mitral stenosis physiology (bottom panels). A preliminary diagnosis of left atrial myxoma complicated by systemic embolism was made, and the patient was referred for surgery. A pedunculated mass consistent with atrial myxoma was removed at surgery. Histology confirmed the diagnosis of atrial myxoma.

## DISCUSSION

Primary cardiac tumours are rare, occurring in only 0.0017–0.03% of autopsy series, and are far less common than cardiac metastases, which are approximately 30 times more frequent.[1] About 75% of primary cardiac tumours are benign, with myxomas accounting for roughly half of adult cases. Atrial myxomas typically affect middle-aged individuals, with a slight female predominance. In African populations, it presents diagnostic and therapeutic challenges.[2] Limited access to echocardiography and specialised cardiac surgery often causes delayed diagnosis, with tumours discovered at large sizes (as in this case), contributing to increased morbidity and mortality.[2,3]

Clinically, myxomas classically present with a triad of obstructive symptoms (dyspnoea, syncope), embolic events (such as stroke), and constitutional features (fever, weight loss).[4] They are most commonly found in the left atrium as pedunculated, mobile masses attached to the interatrial septum, producing a "ball-valve" effect that causes position-dependent symptoms like palpitations or dizziness.

The differential diagnosis of left atrial masses includes thrombi, lipomas, papillary fibroelastomas, metastatic tumours, and infective vegetations, each with distinguishing imaging characteristics.[5] The primary diagnostic tool is echocardiography, supported by multimodality imaging.[4] However, definitive diagnosis relies on histopathological confirmation, including endothelial marker positivity.[4] When treated with prompt surgical resection, the prognosis of atrial myxoma is excellent, with post-operative survival comparable to that of the general population.[4]

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10. Where possible all figures, tables and photographs must also be submitted electronically. The illustrations, tables and graphs should not be imbedded in the text file, but should be provided as separate individual graphic files, and clearly identified. The figures should be saved as a 300 dpi jpeg file. Tables should be saved in a MS Word or PowerPoint document. If photographs are submitted, two sets of unmounted high quality black and white glossy prints should accompany the paper. Figures and photographs should be of high quality with all symbols, letters or numbers clear enough and large enough to remain legible after reduction to fit in a text column. Each figure and table must have a separate self-explanatory legend.
11. Remove all markings such as patient identification from images and radiographs before photographing.
12. Include 3 challenging questions on the content of the manuscript relating to the key messages. The questions will be included in a questionnaire for CPD accreditation purposes. Please supply each question with a choice of 4–5 possible answers of which only one is correct (multiple correct answers not allowed) and highlight the correct answer. Please do not supply questions with a simple yes/no option.

## Submission of manuscripts

The manuscript should be submitted online on the SA Heart® Journal open access platform https://tinyurl.com/y9prlopt. Follow further instructions on this website.

# COMPREHENSIVE CARDIAC CARE

26th Annual SA Heart® Congress
CTICC, Cape Town
30 October – 1 November 2026

SA HEART®

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