ISSN 2071-4602 (Online)

SA HEART®

Journal of the South African Heart Association

2026
Volume 23 Number 4

Congress of the South African Heart Association

COMPREHENSIVE CARDIAC CARE
30 October – 1 November 2026

Guest Editorial | Editorial Comment | President's Welcome | Message from the Congress Convenor | Congress Abstracts | ECG Quiz | Cardiac Imaging Quiz

SA HEART®

Journal of the South African Heart Association

2026
Volume 23 Number 4

Front cover:
The young girl gazing toward the lighthouse symbolises future generations looking to prevention, early detection, and integrated healthcare systems as guiding lights through the growing storm of multiple chronic conditions.
Photo: Ruchika Meel

Editor-in-Chief:
Ruchika Meel

Sub-editors:
Ashley Chin
John Lawrenson
Elena Libhaber
Mamotabo Matshela
Keir McCutcheon
Philasande Mkoko
Arthur Mutyaba
Anupa Patel
Darshan Reddy
Muhammed Talle

Members of the Editorial Board:
Antoinette Cilliers
Anton Doubell
Sajidah Khan
Farouk Mamdoo
Karen Sliwa
Peter Zilla
Liesl Zühlke

SA Heart® Association:
Erika Dau

Publisher:
Medical and Pharmaceutical Publications (Pty) Ltd. trading as

Medpharm Publications

Guest Editorial
Check my Beat: Hearts behind the wheel – Artificial intelligence to invoke a lifesaving campaign for cardiovascular health promotion 168
M Fine and D Jankelow

Editorial Commentary
Multimorbidity in South Africa: A ticking time bomb 170
R Meel

Welcome Note
Presidential welcome address – SA Heart® Congress 2026 172
M Mpe

Congress Conveners Message
Welcome to the 26th Annual South African Heart Association Congress 174
C Kyriakakis

Index of Abstracts - SA Heart® Congress 2026 176

Abstracts - SA Heart® Congress 2026 177

ECG Quiz 200
RS Millar and A Chin

Answer to ECG Quiz 201

Cardiac imaging quiz 203
R Meel and B Cupido

Instructions for authors 206

GUEST EDITORIAL

Check my Beat: Hearts behind the wheel – Artificial intelligence to invoke a lifesaving campaign for cardiovascular health promotion

M Fine[1] and D Jankelow[2]

[1] FINE Group Global, Westcliff, Johannesburg, South Africa
[2] Netcare Linksfield Hospital, Johannesburg, South Africa

Email:
mandi@finegroupglobal.com

M Fine https://orcid.org/0009-0000-1787-8926
D Jankelow https://orcid.org/0000-0002-8733-5035
DOI: https://doi.org/10.24170/23-4-8588
Creative Commons License - CC BY-NC-ND 4.0

We are excited that our novel community cardiac screening programme, "Check my Beat", is now in its third year. It is a truly positive South African story, epitomising *ubuntu* – a spirit that embodies humanity towards others and affirms the power of collective care.

For World Heart Day, the South African Heart Association (SA Heart®) rolled out a primary cardiac screening programme using artificial intelligence (AI). Our focus is the heart health of South Africa's minibus taxi drivers, who keep the wheels of our economy turning. They move approximately 17 million people every day, and many households depend on them for their daily commute, transporting workers to work and children to school.[1] The life of the taxi driver is difficult, with long days, high stress, lack of benefits, and often unhealthy lifestyles. Their diet is generally of poor quality, and many are sedentary, with a lack of exercise. Smoking, hypertension, obesity, and diabetes are common, putting them at risk for cardiovascular disease (CVD).[2,3] Drivers are often reluctant to attend clinics or hospitals for preventive screening, creating a barrier to early detection of disease risk.

We developed an innovative and culturally engaging approach to heart health promotion. Music, particularly Maskandi, a Zulu folk genre historically linked to migrant workers, forms an integral part of many drivers' daily lives, sustaining them through long hours on the road. Recognising this opportunity, we sought to connect with them in their own language and through the music they enjoy. The result is the Check my Beat Heart Health Tracks campaign: an AI-enabled initiative that transforms biometric data and lifestyle information into personalised songs. Each track carries a positive health message, designed to be shared within their own communities; and is created by combining screening parameters with lifestyle insights, such as exercise status, fruit and vegetable consumption, and added sugar and salt intake at meals. An AI music generator then transforms this data into lyrics and melody. Blood pressure readings dictate the song's rhythm and tempo, ensuring each track is uniquely tied to the person's health profile and reflects their own health journey, while also amplifying CVD awareness.

Over the last 3 years, we have screened 789 drivers and commuters at major taxi hubs in Johannesburg for World Heart Day. Assessments included blood pressure, body mass index, random glucose and cholesterol testing, and lifestyle questionnaires for diet and physical activity. Participants received tailored advice on exercise and nutrition. Those with abnormal results were referred to local clinics for further management. Vitals were measured using advanced AI tools: Syntro-P, a contactless plethysmography-based application developed by BrandMed, was used in 2024 and 2026; and Abby Health mobile screening stations were deployed in 2025. The latter eliminated the need for traditional medical equipment or specialised personnel, enabling scalable and accessible screening in the community. Most importantly, hundreds of South Africans received their first-ever heart health screening.

Preliminary data suggest that at least 70% of individuals are overweight or obese. This presents an opportunity to improve our population's health habits. To do this, our sports science partners (University of the Witwatersrand, Johannesburg, South Africa) created a circuit of exercises that can be done at the taxi rank and at the vehicles. Similarly, our dietitians provided alternative healthy food options that are cheap and easy to implement. The exercises and alternative foods are included as an infographic in the screening booklet, provided to participants.

The Check my Beat results are not only clinical. The project created about 500 minutes of personalised music, and the media campaign reached millions of South Africans. The project received international recognition, with inclusion in the BBC StoryWorks Beats of Change series, the 2025 European Society of Cardiology Congress, and a publication in The Lancet Primary Care.[4,5]

The long-term value of Check my Beat lies in its sustainability and integration into community care, and, most importantly, in creating broad national awareness of CVD. The programme will continue annually around World Heart Day, expand to additional transport hubs and provinces, and strengthen referral pathways to local clinics to ensure care continuity. Future phases will prioritise digital data capture to contribute to a real-world CVD risk registry.

Our success is owed to our many stakeholders, all of whom share an interest in heart health. Our project partners include Anglo American, Betway Cares Foundation, Cipla, Jozi My Jozi, the Nahana Foundation, Syntro-P, and the University of the Witwatersrand. We also acknowledge our incredible team who made this all possible: Katie Andrews, Erika Dau, Kate Gershuni, Ruan Kruger, Obakeng Matlhoko, Ruchika Meel, Georgia Torres, and Thobile Ziqubu.

REFERENCES

1. Kriel R, Burger M. Electrifying South Africa's minibus taxi industry: A data-driven approach [Internet]. TheCityFix; 2023. Available from: https://thecityfix.com/blog/electrifying-south-africas-minibus-taxi-industry-a-data-driven-approach/[S1.1]. Accessed 25 June 2026.
2. Mabetwa EM, Mokwena KE, Mphekgwana PM, Modjadji P. Metabolic syndrome and its components among taxi drivers in the city of Tshwane, South Africa. Appl Sci. 2022;12(3):1767. https://doi.org/10.3390/app12031767.
3. Ramukumba TS, Mathikhi MS. Health assessment of taxi drivers in the city of Tshwane. Curationis. 2016;39(1):1671. https://doi.org/10.4102/curationis.v39i1.1671.
4. BBC StoryWorks Commercial Productions. The initiative helping taxi drivers in South Africa keep on track with their heart health [Internet]. Global film series produced in partnership with the World Heart Federation. 2025. Available from: https://www.bbc.com/storyworks/beats-of-change/south-africa-heart[S2.1][S2.2]. Accessed 14 July 2026.
5. Jankelow D, Meel R, Torres G, et al. Check my Beat: A lifesaving campaign in South Africa for cardiovascular health. Lancet Prim Care. 2026. https://doi.org/10.1016/j.lanprc.2026.100197.

EDITORIAL COMMENT

Multimorbidity in South Africa: A ticking time bomb

R Meel
Department of Internal Medicine, Faculty of Health Sciences, University of the Witwatersrand and Mediclinic Sandton Hospital, Johannesburg, South Africa

Email:
ruchikameel@gmail.com

R Meel https://orcid.org/0000-0002-1405-4259
DOI: https://doi.org/10.24170/23-4-9194
Creative Commons License - CC BY-NC-ND 4.0

Multimorbidity refers to the coexistence of 2 or more chronic health conditions within a single individual at the same time.[1] This differs from comorbidity, which describes the presence and impact of additional diseases in relation to a primary or index condition.[2] The global burden of multimorbidity is expected to increase as populations age, unhealthy lifestyle behaviours become more widespread, and the prevalence of chronic diseases continues to rise.[3] The challenge is particularly pronounced in low- and middle-income countries, where healthcare systems are often fragmented and under-resourced, access to care remains inequitable, and limited local evidence hampers the development of effective policies and interventions.[4]

In South Africa, multimorbidity affects an estimated 45.3% of adults and is driven by the dual burden of noncommunicable diseases (NCD) and chronic infectious conditions, particularly human immunodeficiency virus.[5] This epidemiological transition places substantial pressure on an already strained healthcare system and highlights the need for integrated, patient-centred care models.[5] In the study by Hazell et al., hypertension and diabetes were the predominant conditions, affecting 46.4% and 22.6% of participants, respectively.[5]

The likelihood of developing multimorbidity increases substantially with advancing age. Individuals aged 65–95 years have more than 11 times greater odds of experiencing multimorbidity than those aged 15–24 years.[5] This trend is particularly relevant in South Africa's rapidly ageing population. The number of adults aged 60 years and older increased from 3.5 million (7.6% of the population) in 2002 to 5.4 million (9.1%) in 2020, and is projected to double by 2050, highlighting the growing need for healthcare systems to address the rising burden of multiple chronic conditions.[6] As the population ages, older adults face a substantial disease burden, characterised not only by a high prevalence of NCDs, but also by the continued impact of infectious diseases.[7]

In addition to advancing age, socio-economic deprivation is a major driver of multimorbidity.[8] Individuals in the lowest wealth groups are substantially more likely to experience multiple chronic conditions, with a 47% higher risk of multimorbidity and a 90% greater likelihood of multimorbidity accompanied by severe functional limitations compared with the most affluent populations.[9] These findings highlight the strong association between social disadvantage and poorer health outcomes.

Multimorbidity in South Africa is influenced by a combination of demographic, lifestyle, and health system factors.[5] Higher odds of multimorbidity are observed among women, older adults, and formerly married individuals. Modifiable risk factors, such as obesity and occupational smoke exposure, further increase the likelihood of living with multiple chronic conditions.[5]

At the health system level, the growing burden of multimorbidity presents a major challenge, as South Africa's healthcare services have traditionally been organised around managing single diseases.[10] This highlights the need for integrated, patient-centred primary healthcare approaches that can effectively address the complex coexistence of infectious diseases and NCDs. Consequently, community-based cardiovascular health campaigns, such as "Check My Beat", are essential for facilitating the early identification of multimorbidity and reducing the burden of heart disease through improved awareness, screening, and preventive interventions in underserved communities.[11]

REFERENCES

1. Valderas JM, Starfield B, Sibbald B, Salisbury C, Roland M. Defining comorbidity: Implications for understanding health and health services. Ann Fam Med. 2009;7(4):357-63. https://doi.org/10.1370/afm.983.
2. Nicholson K, Almirall J, Fortin M. The measurement of multimorbidity. Health Psychol. 2019;38(9):783-90. https://doi.org/10.1037/hea0000739.
3. iris.who.int [Internet]. Multimorbidity: Technical series on safer primary care. World Health Organization; 2016. Available from: http://apps.who.int/iris/bitstream/10665/252275/1/9789241511650-eng.pdf. Accessed 23 Sept 2026.
4. Basto-Abreu A, Barrientos-Gutierrez T, Wade AN, et al. Multimorbidity matters in low and middle-income countries. J Multimorb Comorb. 2022;12:26335565221106074. https://doi.org/10.1177/26335565221106074.
5. Hazell M, Kengne AP, Gill P, Taylor D, Uthman O. Prevalence, patterns, and determinants of multimorbidity in South Africa: Insights from a nationally representative survey. PLOS Glob Public Health. 2025;5(5):e0004603. https://doi.org/10.1371/journal.pgph.0004603.
6. statssa.gov.za [Internet]. Protecting South Africa's elderly. Statistics South Africa; 2020. Available from: https://www.statssa.gov.za/?p=13445. Accessed 23 Sept 2026.
7. He W, Aboderin I, Adjaye-Gbewonyo D. Africa aging: 2020 – International population reports [Internet]. U.S. Census Bureau; 2020. Available from: https://www.census.gov/content/dam/Census/library/publications/2020/demo/p95_20-1.pdf. Accessed 23 Sept 2026.
8. Barnett K, Mercer SW, Norbury M, et al. Epidemiology of multimorbidity and implications for health care, research, and medical education: A cross-sectional study. Lancet. 2012;380(9836):37-43. https://doi.org/10.1016/S0140-6736(12)60240-2.
9. Buffel du Vaure C, Dechartres A, Battin C, Ravaud P, Boutron I. Exclusion of patients with concomitant chronic conditions in ongoing randomised controlled trials targeting 10 common chronic conditions and registered at ClinicalTrials.gov: A systematic review of registration details. BMJ Open. 2016;6(9):e012265. https://doi.org/10.1136/bmjopen-2016-012265.
10. Roomaney RA, van Wyk B, Pillay-van Wyk V. Multimorbidity in South Africa: Is the health system ready? J Multimorb Comorb. 2023;13:26335565231182483. https://doi.org/10.1177/26335565231182483.
11. Jankelow D, Meel R, Torres G, et al. Check my Beat—A lifesaving campaign in South Africa for cardiovascular health. Lancet Prim Care. 2026. https://doi.org/10.1016/j.lanprc.2026.100197.

WELCOME NOTE

Dr Martin Mpe
President, South African Heart Association (SA Heart®)
Raslouw Private Hospital and Netcare Pholoso Hospital, South Africa

Presidential welcome address – SA Heart® Congress 2026

Dear colleagues, delegates and friends,

It is my great privilege to welcome you to the 2026 SA Heart® Congress, taking place from 30 October to 1 November at the Cape Town International Convention Centre. Following our Association's 25th anniversary in 2025, we now begin a new chapter that calls on us to translate our shared knowledge, experience, and scientific progress into improved cardiovascular care for every community we serve.

This year's programme reflects the breadth and complexity of comprehensive cardiac care. It brings together adult and paediatric cardiology, cardiac surgery, cardiovascular imaging, electrophysiology, heart failure, interventional cardiology, rehabilitation, research and the allied professions. The programme also recognises that excellence in cardiovascular medicine depends not only on technical expertise but also on multidisciplinary collaboration, ethical leadership, innovation, and developing the next generation.

Over the three days, delegates will engage with contemporary challenges spanning acute coronary syndromes, cardiomyopathies and structural heart disease, as well as congenital heart disease, conduction-system pacing and advanced heart-failure therapies. Sessions on multimodality imaging, artificial intelligence, precision genomics and cardiovascular risk prediction invite us to consider how rapidly evolving technologies can improve clinical decision-making. At the same time, important discussions on access to paediatric cardiac services, clinician-scientist development and the sustainability of specialist programmes remind us that innovation has meaning only when it is accessible, relevant and responsive to our health system's realities.

The Congress maintains a strong practical and educational focus. The pre-congress workshops, technical suites, case-based discussions, clinical conundrums and interactive expert sessions offer opportunities to translate evidence into practice. I particularly welcome the prominent participation of fellows, early-career clinicians, researchers and allied professionals. Their questions, discoveries and emerging leadership are essential to the future of cardiovascular medicine in South Africa.

We are honoured to welcome an outstanding national and international faculty, along with our colleagues from the European Society of Cardiology to the joint ESC-SA Heart® session. These partnerships connect South African cardiovascular practice with global scientific progress and create an opportunity for our experience, ingenuity and scholarship to contribute to the international conversation.

The closing plenary on ethics is particularly significant. Its focus on end-of-life care and on innovative but unproven therapies asks us to confront some of the most difficult questions in modern cardiology. Scientific advancement must always be accompanied by sound judgement, respect for human dignity, and an unwavering commitment to our patients' interests.

I express my sincere appreciation to the Congress Organising Committee, the Scientific Programme Committee, our Special Interest Groups, faculty, abstract presenters, sponsors and industry partners. Your collective commitment has made this ambitious programme possible. I also thank every delegate, whose participation gives the Congress its energy and purpose.

I encourage you to approach the Congress with curiosity and openness: engage with the science, participate in discussions, reconnect with colleagues and form new collaborations. May the knowledge we exchange in Cape Town extend well beyond the Congress halls -strengthening our professions, informing our health system and improving the lives of those who depend on us.

Welcome to Cape Town and to the 2026 SA Heart® Congress.

CONVENORS MESSAGE

Welcome to the 26th Annual South African Heart Association Congress

Dr C Kyriakakis
Life Vincent Pallotti Hospital, Cape Town, South Africa
Congress Convenor, SA Heart® 2026

Email:
charleskyriakakis@gmail.com

It is my great pleasure to welcome you to the 26th Annual SA Heart® Congress held at the Cape Town International Convention Centre from 30 October to 1 November. This year's congress, titled "Comprehensive cardiac care", reflects both the remarkable breadth of contemporary cardiovascular medicine and the increasingly multidisciplinary approach required to provide optimal care for our patients.

Comprehensive cardiac care is about more than the increasing sophistication of our diagnostic and therapeutic capabilities. It requires collaboration between disciplines, an appreciation of the patient as a whole, and recognition that we achieve the best outcomes when we combine our individual areas of expertise. This year's congress was designed with this philosophy in mind.

This year, therefore, marks an important change in the format of our congress. In previous years, multiple parallel tracks – while providing an extensive range of specialised content – presented the challenge of ensuring adequate interest and attendance across the various sessions. Over time, this resulted in a somewhat fragmented meeting, with delegates often having to choose between several competing areas of interest.

Consequently, the SA Heart® Executive Committee made the innovative decision to consolidate this year's congress into a single multidisciplinary track, running alongside a dedicated paediatric cardiology track. This is intended to encourage greater interaction between our various special interest groups (SIG), including SASCI, HeFSSA, CASSA, CISSA, ISCAP, and SASCAR, and to allow delegates to engage with areas of cardiovascular medicine beyond their own fields of expertise.

Complementing the main programme is the introduction of a "learning pod" – a new addition to the congress that will allow our SIGs to host focused and specialised sessions. One example is the interactive session on left ventricular assist device therapy developed by Dr Sarah Kraus on behalf of HeFSSA. This new format has not been attempted at previous congresses, and I am confident our Scientific Steering Committee has risen to the challenge by developing a stimulating, diverse educational programme featuring both local and international experts in cardiovascular medicine. The programme has been deliberately designed to be inclusive, allowing each of our SIGs to share their expertise while enabling delegates to engage with a broader spectrum of cardiovascular medicine.

Cardiology continues to advance at a remarkable pace, and we are fortunate to have an outstanding international faculty joining us this year. Leading experts from the Mayo Clinic – including Profs Leslie Cooper, Rowlens Melduni, Hector Michelena, and Vuyisile Nkomo – will share their expertise in areas ranging from myocarditis and echocardiography to valvular and structural heart disease. Their participation, together with that of our many distinguished local faculty members, offers an opportunity for meaningful exchange between international experience and the unique challenges of cardiovascular care in South Africa.

The congress begins on day 1 with the ever-popular paediatric cardiology, cardiac imaging, and electrophysiology workshops, followed by the opening plenary session. Profs Alfonso Pecoraro,

Lisel Zülke, and Leslie Cooper will discuss the development of innovative cardiac care programmes. A combined SA Heart®/European Society of Cardiology session on adult congenital heart disease will follow, with contributions from Profs Marc Gewillig of the University of Leuven and George McDaniel of the University of Virginia, both of whom will also contribute to the paediatric cardiology programme.

Among the many other highlights is a session on artificial intelligence (AI) in cardiology, developed by Dr David Jankelow. AI represents an important and potentially disruptive development in medicine, and Prof Faisal Syed from the University of North Carolina will challenge us with his provocative presentation, "The ECG will see you now". He will also contribute to the cardiac electrophysiology programme and debate Dr Bradley Griffiths on the role of left atrial appendage occlusion in managing atrial fibrillation.

Structural heart disease will be explored through a challenging case presentation by Prof Hellmuth Weich, while Prof Ruchika Meel, editor of the SA Heart® Journal, will lead an important session for our aspiring researchers, "Clinician–scientist pathway: Education and career tips". HeFSSA will broaden our understanding of myocarditis under the guidance of Prof Leslie Cooper, a leading international expert and founder of the Myocarditis Foundation, and Prof Michael Böhm of Saarland University in Germany will provide further insights into the prevention and management of heart failure. In coronary intervention, one of the current frontiers in the management of coronary artery disease – the modification of coronary calcification – will be addressed by Dr Abdul Mozid from Leeds General Infirmary, together with our local expert, Dr Adie Horak.

The programme also recognises that comprehensive cardiac care extends beyond technological advances and procedural expertise. On day 3, 2 thought-provoking ethics sessions will address the often-neglected issue of end-of-life care in cardiology, as well as the ethical considerations surrounding innovative and unproven therapies, using the experience of a patient with refractory ventricular tachycardia as a starting point for discussion.

Our cardiology fellows will once again present exciting and informative case studies from our various academic units. These sessions are always among the highlights of the congress, providing an opportunity to showcase the knowledge, enthusiasm, and potential of our young and emerging clinicians.

The congress concludes with "Cardiology for non-cardiologists", a HeFSSA-led programme aimed at general practitioners and specialist physicians. This consistently well-attended programme reflects an important principle of comprehensive cardiac care: that improving cardiovascular outcomes depends not only on cardiologists, but on collaboration across the broader medical community.

None of this would be possible without the support of our industry partners. I would like to express my sincere appreciation for their continued commitment to SA Heart® and their contribution to making this year's congress possible. I would also like to thank the members of the Scientific Steering Committee, who have worked tirelessly to develop a stimulating, informative, and memorable programme. Most importantly, I hope this congress gives us an opportunity not only to learn, but also to reconnect with friends and colleagues, establish new professional relationships, and share ideas across the boundaries of our individual subspecialities. I wish you all an enjoyable and rewarding congress, and a very warm welcome to Cape Town.

INDEX OF ABSTRACTS
SA HEART® CONGRESS 2026
ALPHABETICAL LISTING OF FIRST AUTHORS

A Dawood 177

A Kolkenbeck-Ruh 177

A Wentzel 178

A Aboubaker 178

B Abera 179

B Naidoo 179

B Smith 180

C Nel 180

E Nel 181

G Bogoslovskaya 181

H Edwards 182

I Mbanze 182

J Basson 183

K Ekambaram 183

K Sithole 184

K Mokoena 184

L Wille 185

L Hahnle 185

L Giliomee 186

M Terblanche 187

M van Hoogland-van Heerden 187

M Rajah 188

M Cremer 189

N Dullabh 189

N Manzini 190

O Nyekete 190

P Wu 191

P Grobler 191

R Mahomed 192

R Kidula 192

S Khoza 193

S Bakali 193

S Subramoney 194

S Ghulam Hoosain 194

PM Shivaprasad 195

T Dawood 195

T Seedat 196

T Nyawo 196

U Adamu 197, 198

W Naicker 198

Z Magwebu 199

Z Ngwenyama 199

ABSTRACTS
SA HEART® CONGRESS 2026

A rare case of refractory atrioventricular re-entrant tachycardia in a haemodynamically stable infant

A Dawood,[2] L Swanson[1,2] and J Lawrenson[1,2]
[1] Paediatric Cardiology, Red Cross War Memorial Children's Hospital, South Africa
[2] Department of Child Health, University of Cape Town, South Africa

Supraventricular tachycardia (SVT) is the most common sustained arrhythmia in infancy, with atrioventricular re-entrant tachycardia (AVRT) representing a frequent underlying mechanism. While most cases respond to first-line anti-arrhythmic therapy, refractory SVT in haemodynamically stable infants remains a significant management challenge. We discuss a retrospective case report. Written informed consent for publication was obtained directly from the patient. Medical records and echocardiographic reports were reviewed, and relevant data were extracted for analysis.

We report a rare case of refractory AVRT in an infant who presented with persistent SVT unresponsive to medical and electrical cardioversion. On arrival, sustained SVT was confirmed and, following cardiology consultation, intravenous amiodarone therapy was initiated and titrated. Despite combination treatment with amiodarone and propranolol, the tachyarrhythmia remained refractory, although the patient remained haemodynamically stable throughout. Ivabradine was subsequently added without achieving adequate rhythm control.

Following a multidisciplinary discussion involving a paediatric electrophysiologist, AVRT was confirmed as the underlying arrhythmia mechanism. Flecainide therapy was then introduced, with gradual weaning of the previously administered anti-arrhythmic agents. Serial echocardiography initially demonstrated preserved cardiac function with a left ventricular ejection fraction of 67%. However, repeat imaging 3 weeks later showed a decline to 55% with persistent tachyarrhythmia, raising concern for evolving tachycardia-induced ventricular dysfunction.

This case highlights the complexity of managing refractory AVRT in infancy and emphasises the importance of early multidisciplinary involvement, serial cardiac function monitoring, and timely escalation to alternative anti-arrhythmic strategies. Persistent SVT may lead to progressive ventricular dysfunction even in clinically stable patients, underscoring the need for prompt recognition and effective rhythm control.

High prevalence of dyslipidaemia and hypertension, but not obesity and dysglycaemia, among young South African adults: Insights from the Multi-Ethnic Early Vascular Ageing (MEEVA) study

A Kolkenbeck-Ruh, S Halana, N Mapheto, W du Toit, N Mthembu, G Norman, G Tade, A Woodiwiss and V Peterson
Department of Physiology, University of the Witwatersrand, South Africa

Cardiometabolic risk factors, including obesity, elevated blood pressure (BP), dysglycaemia, and dyslipidaemia, are a growing public health concern in South Africa, contributing to noncommunicable diseases (NCD) and premature cardiovascular disease (CVD). Despite the recognised burden of NCDs in South Africa, limited data exist on the prevalence of these cardiometabolic risk factors among young adults. The ongoing Multi-Ethnic Early Vascular Ageing (MEEVA) study aimed to address this gap by investigating the prevalence of obesity, hypertension, dysglycaemia, and dyslipidaemia among young adults in South Africa.

This cross-sectional observational study recruited South African adults (≥ 18 years) living in Johannesburg. Participants provided venous blood samples and underwent cardiovascular assessments. Key outcomes included obesity, elevated BP, dysglycaemia, and dyslipidaemia. Of the 103 participants (median age 29 years), 31.1% were obese. Hypertension prevalence was 58.3%; higher in males (74%) than females (43.4%). Dysglycaemia affected 7.8% of participants and was more prevalent in males. Dyslipidaemia affected 67% of participants, with commonly elevated low-density lipoprotein (LDL) cholesterol (36.9%). Females had a higher overall prevalence of dyslipidaemia, with notable racial and age-related differences.

The findings show that dyslipidaemia and hypertension are the most significant cardiometabolic risk factors among young South Africans, exceeding the impact of obesity and dysglycaemia. Notably, elevated LDL cholesterol was identified as the main factor contributing to dyslipidaemia. These findings suggest that dyslipidaemia and hypertension may be key contributors to the rising early incidence of CVD in young South Africans, underscoring the urgent need for targeted prevention strategies.

An advanced neural network analysis of mental stress electrocardiogram recovery reveals multisystem impairment in non-dipping hypertensive South Africans

A Wentzel,[1,2] Y Breet[1,2] and W Smith[1,2]

[1] Hypertension in Africa Research Team, North-West University, South Africa

[2] South African Medical Research Council Unit for Hypertension and Cardiovascular Disease, North-West University, South Africa

Post-stressor electrocardiogram (ECG) recovery patterns in non-dipping and hypertension may reveal impaired autonomic regulation, abnormal repolarisation dynamics, and delayed myocardial perfusion recovery. We characterised ECG recovery profiles after mental stress using advanced machine learning (ML) models and defined how these trajectories differ by dipping and hypertension status in a South African population.

In 312 South African adults (199 dippers, 113 non-dippers; normotensive and hypertensive), 24-hour ambulatory blood pressure monitoring defined dipping and hypertension status. All participants underwent a standardised colour–word conflict test with continuous ECG recording. ECG segments at rest, peak stress, and 1, 3, and 5 minutes post-stress were used to derive: (1) R-R interval-based heart rate variability (HRV) indices; (2) heart rate recovery (HRR); (3) corrected QT interval (QTc) dynamics; and (4) ST segment deviation and normalisation. A feedforward neural network and a trajectory-aware gated recurrent unit (GRU) network were trained to classify dipper versus non-dipper status from ECG recovery features, evaluating performance using cross-validated area under the receiver operating characteristic curve (AUROC), precision-recall metrics, and calibration indices. Latent GRU representations were clustered to identify physiologically distinct recovery phenotypes across 4 clinical strata: normotensive dippers, normotensive non-dippers, hypertensive dippers, and hypertensive non-dippers.

Non-dippers exhibited slower HRR and blunted vagal HRV rebound following mental stress, with hypertensive non-dippers additionally showing delayed QTc normalisation and more frequent low-grade, persistent ST segment abnormalities during the 5-minute recovery window. A feedforward neural network of ECG recovery features classified non-dippers with high discrimination (AUROC 0.82), while the GRU model, which leveraged the full temporal trajectory of recovery, performed best (AUROC 0.85), with particularly strong discrimination in hypertensive non-dippers (AUROC 0.90) and preserved calibration across subgroups. Clustering revealed 4 recovery phenotypes: (1) rapid autonomic and repolarisation normalisation; (2) an autonomic sluggish cluster with delayed HRR and HRV recovery but preserved QTc/ST behaviour; (3) a high reactivity well-recovering cluster; and (4) a multisystem impaired cluster characterised by persistent autonomic activation, QTc prolongation, and ST segment non-recovery.

Mental stress ECG recovery models revealed a multisystem impaired recovery phenotype specifically in hypertensive non-dippers marked by persistent autonomic activation, delayed repolarisation, and ST segment non-recovery. Using the full ECG recovery time-series, advanced ML models improved discrimination of non-dipping status and revealed previously unrecognised recovery patterns, showing mental stress ECG recovery patterns linked to specific risk clusters of impaired cardiovascular resilience in non-dipping hypertension.

Ineligibility insights: Exploring the challenges of oral stepdown therapy for infective endocarditis in a resource-limited setting

A Aboubaker,[1] A Doubell,[2] A Pecoraro[2] and A Engelbrecht[2]

[1] Department of Internal Medicine, Faculty of Medicine and Health Sciences, Stellenbosch University, South Africa

[2] Division of Cardiology, Department of Medicine, Faculty of Medicine and Health Sciences, Stellenbosch University, South Africa

Oral stepdown therapy is an emerging standard of care for selected patients with infective endocarditis (IE). This study describes the clinical and socio-behavioural challenges that prevent IE patients from receiving this therapy in the South African healthcare system, comparing them with a parallel cohort receiving oral stepdown therapy.

This single-centre study evaluated IE patients admitted to Tygerberg Hospital who were excluded from oral stepdown therapy between August 2024 and March 2026. Comprehensive demographic, clinical, microbiological, and echocardiographic data were analysed to identify factors preventing transition to oral therapy.

The cohort comprised 56 patients (mean age 44 ± 13 years, 75% male). Blood cultures were positive in 59% (*n* = 33), with *Staphylococcus aureus* most frequently isolated. Blood culture-negative IE (BCNIE) accounted for 41% (*n* = 23). *Bartonella* species predominated in the BCNIE group, while no organism was identified in 52% (*n* = 12). Stroke occurred in 21% (*n* = 12), causing swallowing dysfunction in 4% (*n* = 2). Primary reasons for not receiving oral stepdown therapy were pre-enrolment mortality (*n* = 21, 37%) and being unfit for intervention despite surgical indications (*n* = 12, 21%). Other reasons included concomitant infections necessitating continuous intravenous therapy (*n* = 7, 12%), refusal of participation, mostly among people with a history of intravenous drug use (IVDU) (*n* = 6, 11%), belonging to vulnerable populations, such as prisoners or those with intellectual disabilities (*n* = 4, 7%), and fungal endocarditis (*n* = 2, 4%).

In-hospital mortality and high surgical risk, likely driven by late clinical presentation and severe complications, are the leading barriers to implementing oral stepdown therapy in this resource-constrained setting. Stroke-associated swallowing difficulty and concomitant

infections further limit eligibility by necessitating continued parenteral administration. Additionally, patient refusal, predominantly among individuals with a history of IVDU, presents a significant socio-behavioural challenge. Optimising IE outcomes requires decentralised early diagnosis, timely tertiary referral, and integrated psychosocial support models.

An unexpected and potentially lethal cause of paediatric chest pain

B Abera, G Comitis and A Brooks
Department of Paediatric Cardiology, Red Cross War Memorial Children's Hospital, University of Cape Town, South Africa

Congenital coronary anomalies are rare, with an estimated prevalence of 0.1–1%, of which interarterial anomalous aortic origin of the coronary artery (AAOCA) accounts for 0.1–0.7%. AAOCA is a recognised cause of sudden cardiac death, especially in young athletes. Most patients are asymptomatic and diagnosed incidentally during cardiac imaging for other indications. We report the case of a 12-year-old boy who presented with left-sided chest pain associated with exertion and a subjective feeling of dyspnoea and palpitations, without a history of dizziness or syncope.
Physical examination and laboratory tests were unremarkable. A resting electrocardiogram showed sinus bradycardia but no evidence of ischaemia. A transthoracic echocardiogram showed a left main coronary artery origin from the right sinus of Valsalva with its proximal portion coursing intramurally towards the left aortic sinus, as well as interarterially between the aortic and pulmonary artery roots, normal origin and course of the right coronary artery, and normal intracardiac anatomy and ventricular function. Coronary computed tomography angiography confirmed these echocardiographic findings and further showed normal bifurcation of the left main coronary artery into the left anterior descending and the circumflex arteries. An exercise stress test showed subtle signs of myocardial ischaemia. To eliminate the risk of exercise-induced sudden cardiac death, the child underwent surgical left coronary artery repair (osteoplasty and patch with autologous pericardium). The immediate post-operative recovery was uneventful. The child is maintained on daily aspirin and scheduled for a repeat exercise stress test 3 months after the operation to confirm the absence of inducible ischaemia before allowing a return to exercise.
The risk of sudden death is increased in anomalous left coronary artery origin from the right aortic sinus with intramural and interarterial course. Surgical management with anatomical repair is the treatment of choice to prevent sudden death, provided normal coronary anatomy and function are achieved and that extensive return-to-play testing is performed before permitting a return to exercise.

Assessing the cost of heart failure admissions at a tertiary academic hospital in the Western Cape: A retrospective cost analysis

B Naidoo, A Doubell, A Mazaza and H Weich
Division of Cardiology, Department of Medicine, Faculty of Medicine and Health Sciences, Stellenbosch University, Tygerberg Hospital, South Africa

Heart failure is a major cause of hospitalisation, morbidity, mortality, and healthcare expenditure worldwide. Although inpatient care is recognised as the principal contributor to heart failure-related costs internationally, South African public-sector data quantifying the direct cost of heart failure admissions remain limited. This study aimed to determine the direct cost of heart failure admissions at a tertiary academic hospital and identify the principal drivers of admission cost.
A retrospective descriptive cost analysis was conducted among adults with heart failure admitted to Tygerberg Academic Hospital between 1 January 2022 and 31 December 2023. Admissions were identified using the International Classification of Diseases, tenth revision (ICD-10) coding and confirmed by clinical record review. Medical costs were calculated using tariff-based components derived from the Uniform Patient Fee Schedule, including ward stay, cardiac care unit (CCU) stay, intensive care unit (ICU) stay, laboratory investigations, radiological investigations, cardiac investigations, cardiac procedures, and medication. Costs are reported as medians with interquartile ranges (IQR). Predictors of admission cost were evaluated using multivariable log-linear regression.
The study included 900 admissions. The median age was 57 years (IQR 44–67), and 51.2% of admissions occurred in men. In-hospital mortality was 5.1%. A total of 771 individual patients were identified, of whom 103 had repeat admissions, accounting for 232 admissions (25.8% of all admissions). Total expenditure during the 2-year study period was R30.6 million. The median cost per admission was R25 598 (IQR R15 053–R42 339). Inpatient stay was the largest cost component, accounting for 70.7% of total expenditure. Independent cost drivers included length of stay (9.2% increase per day; $p < 0.001$), CCU stay (10.4% increase per day; $p < 0.001$), and ICU stay (8.4% increase per day; $p < 0.001$). Cardiac procedures were independently associated with higher admission costs.
Heart failure admissions impose a substantial financial burden on the South African public healthcare system. Costs are driven predominantly by the duration and intensity of inpatient care, while repeat admissions contribute significantly to resource utilisation. These findings provide important local data to inform healthcare planning and strategies to reduce recurrent hospitalisation and improve the efficiency of heart failure care.

Cardiovascular disease risk factors in patients with fibromyalgia: A scoping review

B Smith,[1] A Orchard,[1] S Leigh[1] and D Constantinou[2]

[1] Department of Pharmacy and Pharmacology, Faculty of Health Sciences, University of the Witwatersrand, South Africa

[2] Department of Exercise Science and Sports Medicine, Faculty of Health Sciences, University of the Witwatersrand, South Africa

Fibromyalgia (FM) is a chronic pain disorder affecting approximately 5% of the global population and is associated with reduced quality of life. FM usually coexists with chronic diseases, including cardiovascular disease (CVD). Metabolically related characteristics in FM patients have been implicated in CVD, potentially via a specific risk profile. These comorbidities contribute to poorer clinical outcomes and warrant screening and monitoring. Studies attribute cardiovascular deaths to established CVD risk factors, such as high blood pressure, dyslipidaemia, and a high body mass index (BMI). This scoping review aimed to map the available evidence on CVD risk factors in patients with FM globally over 3 decades (1990–2023).

The scoping review was conducted in accordance with the Joanna Briggs Institute (JBI) methodology. Databases included Scopus®, ScienceDirect®, and PubMed®, using keywords related to CVD risk factors and FM. The initial literature search yielded 20 658 publications. After applying inclusion and exclusion criteria and removing duplicates, 8 752 titles were screened. EndNote X8 was used for reference management.

Following the selection process, 82 published studies were included in the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) flow diagram. We identified 31 CVD risk factors and mapped their geographic distribution. Of the identified risk factors, 24/31 (77.4%) were elevated in FM patients compared with controls, suggesting an unfavourable cardiovascular risk profile. FM patients had higher heart rate, blood pressure, BMI, and total cholesterol than healthy controls. Given that hypertension is a major risk factor for CVD, it is noteworthy that FM patients exhibit both elevated systolic and diastolic blood pressures and an increased prevalence of hypertension. Geographically, the United States of America and Spain had the most published data.

This scoping review highlights a limited evidence base contributing to significant data gaps regarding CVD risk and FM. The available evidence suggests that FM patients represent a population at increased cardiovascular risk and may benefit from cardiovascular risk assessment and monitoring.

Door-to-electrocardiogram time in patients with ST-elevation myocardial infarction at Tygerberg Hospital: A cross-sectional study

C Nel,[1] R Bowden,[1] C Mostert,[1] N Schoeman,[1] E Ngarande,[2] E Marais[3] and A Doubell[2]

[1] MBChB Student, Faculty of Medicine and Health Sciences, Stellenbosch University, South Africa

[2] Division of Cardiology, Department of Medicine, Stellenbosch University and Tygerberg Hospital, South Africa

[3] Centre for Cardio-Metabolic Research in Africa, Division of Medical Physiology, Faculty of Medicine and Health Sciences, Stellenbosch University, South Africa

Acute coronary syndrome (ACS) remains a significant health burden in South Africa. Guidelines recommend performing a 12-lead electrocardiogram (ECG) within 10 minutes of first medical contact in patients with suspected ACS. The door-to-ECG (DTE) time is a key quality indicator associated with outcomes. However, no published data currently exist on DTE times in South African healthcare settings. This study aimed to determine the average DTE time for patients with an ST-elevation myocardial infarction (STEMI) presenting as undifferentiated patients to triage at Tygerberg Hospital (TBH) and to assess whether the guideline-recommended 10-minute target was met.

A retrospective cross-sectional study was conducted using the TBH STEMI database. Adult patients (≥ 18 years) with a confirmed STEMI who presented to TBH triage between November 2021 and April 2026 were included. Patients referred directly to cardiology and those with missing door-time data were excluded. DTE time was calculated as the interval between first medical contact and ECG acquisition. The study included 98 STEMI patients. The median DTE was 28.5 minutes, with 28.6% of patients achieving a DTE ≤ 10 minutes. Method of transport was the only factor significantly associated with DTE time. Patients arriving by ambulance had a shorter median DTE than those using private transport (20.4 vs. 69.45 minutes; $p = 0.03$).

Fewer than one-third of patients achieved the target DTE time of ≤ 10 minutes, indicating that delays in ECG acquisition remain a significant challenge in STEMI care for undifferentiated patients presenting via the TBH triage service. Although patients arriving by ambulance had significantly shorter DTE times than those using private transport, it remained above the recommended 10-minute target. This highlights the need to identify and address additional system-level factors contributing to delays.

Improving electrocardiographic criteria for the detection of cardiac magnetic resonance-determined left ventricular hypertrophy in high body mass index groups

E Nel and A Doubell
Division of Cardiology, Tygerberg, South Africa

Left ventricular hypertrophy (LVH) requires early detection, both as a marker of underlying cardiac pathology and as a predictor of adverse outcomes. While cardiac magnetic resonance imaging (CMR) is the diagnostic gold standard, electrocardiography (ECG) remains the most accessible screening tool. The prevalence of obesity challenges detection of LVH with conventional ECG criteria. Our group previously demonstrated potential problems in this area: CMR-derived measurements showed that some chest leads were disproportionately displaced from the heart in individuals in higher body mass index (BMI) categories. Moreover, the heart's anatomical orientation shifts slightly leftwards. Consequently, the sensitivities of 3 ECG LVH criteria (Sokolow–Lyon [SL], Cornell voltage [CV], and Peguero–Lo Presti [PLP]) decrease significantly. New criteria that incorporate our CMR observations may improve sensitivity. This study aimed to develop an ECG criterion with improved performance in detecting LVH in an obese population (defined as BMI ≥ 30 kg/m^2).
A retrospective, comparative, descriptive study design was conducted in the Division of Cardiology at Tygerberg Hospital. Convenience sampling was employed to select 447 patients with available concomitant ECG and CMR records. LVH on CMR was defined as > 153 g in males and > 103 g in females. A total of 314 patients were selected as a study group to identify new criteria. The remaining 133 patients were used as a validation group to test the proposed criteria. A new criterion was identified (abbreviated SV1SV3RI):
(S wave V1) + (S wave V3) + (R wave I) ≥ 30 mm
In the first obese group (*n* = 85), SV1SV3RI had a sensitivity of 60.7% (95% confidence interval [CI] 47.3 to 72.9) and a specificity of 87.5% (95% CI 67.6 to 97.3). This was compared with SL (sensitivity 26%, specificity 96%), CV (sensitivity 25%, specificity 92%) and PLP (sensitivity 34%, specificity 92%). In the second obese group (*n* = 62), SV1SV3RI had a sensitivity of 65.6% (95% CI 46.8 to 81.4) and a specificity of 70% (50.6–85.3%). This was compared to SL (sensitivity 31.2%, specificity 96.7%), CV (sensitivity 34.4%, specificity 96.7%) and PLP (sensitivity 40.6%, specificity 90%).
We propose a new criterion for detecting LVH in obese individuals that outperforms the 3 other existing criteria in sensitivity.

Tuberculous perimyocarditis presenting with life-threatening cardiac tamponade and interventricular septal infiltration in a postpartum woman: Case report

G Bogoslovskaya[1] and A Bogoslovskiy[2]
[1] Department of Internal Medicine, Polokwane/Mankweng Hospital Complex, Limpopo University, South Africa
[2] Department of Anaesthesia, Polokwane/Mankweng Hospital Complex, Limpopo University, South Africa

Tuberculous pericarditis is a major cause of cardiac disease in tuberculosis-endemic regions and rarely involves the myocardium, causing life-threatening complications. We describe a postpartum patient with tuberculous perimyocarditis complicated by cardiac tamponade and interventricular septal infiltration, highlighting echocardiographic findings, diagnostic evaluation, haemodynamic compromise, and successful management.
We report a 23-year-old postpartum woman presenting with progressive dyspnoea, chest discomfort, palpitations, hypotension, elevated jugular venous pressure, and ventricular ectopy. Electrocardiogram showed sinus tachycardia, low QRS voltage, and ventricular ectopic beats. Echocardiography revealed massive circumferential pericardial effusion with cardiac tamponade and focal interventricular septal infiltration. Emergency pericardiocentesis resulted in immediate haemodynamic improvement. Tuberculous perimyocarditis was diagnosed based on clinical, echocardiographic, laboratory, and microbiological findings.
Following urgent pericardial decompression, the patient demonstrated rapid clinical and haemodynamic improvement, with resolution of hypotension, normalisation of peripheral perfusion, and a marked reduction in ventricular ectopic activity. Pericardial fluid analysis confirmed tuberculosis, revealing an elevated adenosine deaminase level, positive GeneXpert *Mycobacterium tuberculosis*/rifampin (MTB/RIF) testing, and a positive mycobacterial culture. Standard antituberculosis therapy was promptly initiated and completed over 6 months with good adherence and no major treatment-related complications.
Serial follow-up echocardiography showed progressive reduction and eventual complete resolution of the pericardial effusion. Additionally, previously identified myocardial and interventricular septal infiltration regressed significantly, with preservation of biventricular systolic function throughout follow-up. No recurrent pericardial effusion, clinically significant arrhythmias, or signs of constrictive physiology were observed. At treatment completion, the patient remained asymptomatic with sustained clinical recovery and favourable echocardiographic outcomes.
Tuberculous perimyocarditis should be considered in postpartum patients presenting with haemodynamic instability, pericardial effusion, arrhythmias, and myocardial involvement in tuberculosis-endemic regions. Early echocardiographic evaluation, prompt pericardial drainage, microbiological confirmation, and timely antituberculosis therapy are essential for favourable outcomes. This case highlights the rare occurrence of interventricular septal infiltration and emphasises echocardiography's crucial role in diagnosis, management, and follow-up.

Pregnancy concerns for women with heart disease – Interventions for support, increased knowledge, and self-management: the Nurture Study

H Edwards,[1] K Sliwa,[2,7] M Berghammer,[4] A Burstrom,[5] M Andipatin,[6] T Ahmed,[8] A Osman[8] and L Zühlke[1,2,3]

[1] Children's Heart Disease Research Unit, Red Cross Children's Hospital, South Africa
[2] Cape Heart Institute, Department of Medicine, South Africa
[3] South African Medical Research Council, South Africa
[4] Department of Health Sciences, University West, Sweden
[5] Department of Neurobiology, Care Science and Society, Karolinska Institute, Sweden
[6] Department of Psychology, University of the Western Cape, South Africa
[7] Division of Cardiology, Department of Medicine, University of Cape Town and Groote Schuur Hospital, South Africa
[8] Department of Obstetrics and Gynaecology, University of Cape Town and Groote Schuur Hospital, South Africa

Congenital heart disease (CHD) and rheumatic heart disease (RHD) are among Africa's most common cardiovascular conditions, causing significant morbidity and mortality. Data regarding pregnant women affected by these conditions from low- and middle-income countries are limited. Thus, we aimed to describe the understanding of pregnant women with CHD or RHD regarding symptoms and the consequences of living with heart disease. Participants were recruited at the combined cardio-obstetric clinic at Groote Schuur Hospital, Cape Town.

We employed a mixed-methods research design, divided into 3 phases. The first phase consists of 4 questionnaires, while the second phase will focus on in-depth interviews with participants. The third phase will use results from previous phases to develop a mobile health app to provide knowledge to pregnant women suffering from CHD and RHD. The study completed the first phase, in which 20 participants were enrolled in 2025. Most of the participants (35%) were aged 21–25 years. At enrolment, 85% of participants had a CHD diagnosis, and 15% had an RHD diagnosis. Of the participants, 25% had a comorbidity, and 35% were taking medication.

Participants reported a high pregnancy symptom burden, particularly frequent urination (95%), fatigue and pain (90%), nausea and heartburn (75%), and anxiety (60%). Participants also reported moderate problems with mobility (45%) and usual activities (50%), as well as pain and discomfort (65%). Using the EQ-5D questionnaire, the mean self-reported health at enrolment was 71%. Responses suggested low concern about illness (average 4.4/10), a high sense of control over illness (8.1/10), high confidence in treatment (7.7/10), and a low burden of heart disease symptoms (2.7/10). The mean reported quality of life was 67%, and most participants (45%) were satisfied with their lives.

Despite a high pregnancy-related symptom burden, participants reported a strong sense of control, low concerns about their illness, and reasonable quality of life at enrolment. However, the presence of comorbidities in 25% of participants and medication use in 35% highlights this population's complex needs. The next phase will involve interviews to explore lived experiences and unmet needs and determine health-seeking behaviour.

An ongoing registry of heart failure in the young: Initial findings

I Mbanze,[1,2] S Allie,[2] N Jessen,[1] A Damasceno[1] and K Sliwa-Hahnle[2]

[1] Department of Medicine and Division of Cardiology, Maputo Central Hospital, Mozambique
[2] Cape Heart Institute, Faculty of Health Sciences, South Africa

The pattern of heart failure in Africa differs from other parts of the globe, described as diverse and occurring at much younger ages. However, the prevalence of heart failure in young patients in Maputo, Mozambique, is unknown.

Young patients (aged 18–44 years) admitted with acute heart failure (AHF) to Maputo Central Hospital, Mozambique, have been consecutively enrolled in an ongoing prospective observational study aimed at understanding the spectrum of AHF in the young. Patients willing to participate and able to return for a 6-month follow-up visit were included in the study. All participants underwent clinical evaluation, and available tests (electrocardiogram and echocardiogram) to diagnose AHF were performed. Patients admitted for elective cardiac surgery, and those with oncological and mental disorders or incomplete workup to diagnose AHF were excluded from the study. The primary outcomes include intra-hospital mortality and 30-day survival.

Since February 2026, 25 participants have been enrolled. The mean age was 31.9 years, and 13 (52%) were male. Of the participants, 19 (76%) presented with de novo heart failure. Seventeen (68%) presented in New York Heart Association (NYHA) functional class III, with 8 (32%) in NYHA IV. The mean left ventricular ejection fraction was 31.7% (95% confidence interval [CI] 24.3 to 39.1). Dilated cardiomyopathy was a frequent cause of AHF in males, predominantly alcoholic cardiomyopathy, whereas females had peripartum cardiomyopathy. At 60 days of follow-up, 2/5 participants (40%) with alcoholic cardiomyopathy fully recovered the left ventricular ejection fraction, 2 (40%) improved, and 1 (20%) did not improve. Hospital mortality was 8% (95% CI 0.9 to 26), and 30-day mortality was 81.2% (95% CI 0.602 to 1).

Heart failure is common in young patients in Maputo, Mozambique. Similar to previous large African registries, patients present in advanced stages of the disease, resulting in high mortality. The causes of AHF in this young cohort differ by gender, being more frequently associated with alcohol consumption in males and peripartum cardiomyopathy in females.

Right atrial haemangioma in a patient with Maffucci syndrome: A rare cardiac manifestation

J Basson, L Lorentz and N Niksch
Radiology, Capital Radiology, LIFE Groenkloof Hospital, Pretoria, South Africa

Maffucci syndrome is a very rare, non-hereditary mesodermal dysplasia characterised by multiple enchondromas and vascular malformations. Cardiac involvement is exceptionally uncommon. We present a rare case of an intracardiac haemangioma involving the roof of the right atrium in a female patient with known Maffucci syndrome.
A middle-aged female with Maffucci syndrome underwent contrast-enhanced computed tomography (CT) of the chest during evaluation of nonspecific cardiopulmonary symptoms. CT revealed a hypervascular mass appearing to arise from the interatrial septum. Subsequent transthoracic echocardiography (TTE) demonstrated a well-circumscribed echogenic intracardiac lesion causing mild compression of the pulmonary veins without significant obstruction or haemodynamic compromise. Transoesophageal echocardiography (TEE) was performed for further characterisation and confirmed a vascular mass arising from the roof of the right atrium. No intracardiac thrombus, pericardial invasion, significant valvular involvement, or associated arrhythmia was identified. Multimodality imaging findings were consistent with a right atrial haemangioma causing mild pulmonary venous compression. The patient remained clinically stable, with no evidence of functional cardiac impairment. CT initially identified the lesion, while TTE and TEE provided further anatomical and functional characterisation.
Patient management remains supportive. The patient has restrictive lung disease and remains oxygen-dependent; however, no functional cardiac impairment has been identified. Surgical resection of the haemangioma was not undertaken due to the extent of the disease and limited pulmonary reserve secondary to restrictive lung disease. The patient remains under ongoing clinical and imaging surveillance, with particular attention to potential malignant transformation.
This case highlights a rare cardiac manifestation of Maffucci syndrome in the form of a right atrial haemangioma causing mild pulmonary venous compression. Multimodality imaging with CT, TTE, and TEE played a central role in accurate diagnosis and anatomical characterisation of this unusual intracardiac vascular lesion. In the absence of functional cardiac impairment, supportive management and ongoing surveillance were favoured, given the extent of disease and limited pulmonary reserve.

Calibrated volumetric artificial intelligence-assisted assessment of left ventricular function across echocardiography and point-of-care cardiac ultrasound

K Ekambaram,[1] P Herbst,[2] A Arnab[3] and R Theart[4]
[1] Institute for Biomedical Engineering, Stellenbosch University, South Africa
[2] Division of Cardiology, Stellenbosch University and Tygerberg Hospital, South Africa
[3] Independent researcher, United Kingdom
[4] Department of Electrical and Electronic Engineering, Stellenbosch University, South Africa

Despite major advances in automated echocardiographic analysis, most artificial intelligence (AI) systems estimate ejection fraction (EF) as a point value and provide limited information regarding prediction reliability. This remains problematic in heterogeneous clinical environments, particularly point-of-care cardiac ultrasound, where acquisition conditions differ substantially from traditional echocardiography laboratories.
We developed EchoVerse, a calibrated, volumetric, AI-assisted framework that estimates ventricular volumes, derives EF, and provides evidence to support clinical interpretation and specialist review. EchoVerse uses self-supervised learning and a segmentation-free, volume-first approach to estimate end-diastolic volume (EDV) and end-systolic volume (ESV) distributions directly from apical 4-chamber cine loops. EF and associated uncertainty are derived from predicted volume distributions. Models were trained on EchoNet-Dynamic and externally evaluated on a subset of MIMIC-IV-ECHO. Performance was assessed using volumetric accuracy, prediction interval coverage, calibration metrics, and the ability of model-derived uncertainty to identify studies at risk of clinically important error.
On the EchoNet-Dynamic test set (n = 1 277), EDV and ESV mean absolute error (MAE) were 17.90 ml and 9.73 ml, respectively. Raw 90% prediction interval coverage was 79.6% for EDV and 84.7% for ESV. Derived EF MAE was 5.28%. Under zero-shot external validation on MIMIC-IV-ECHO, EF MAE was 9.10% against apical 4-chamber references and 8.14% against biplane references. Prediction interval coverage deteriorated under external distribution shift (EDV 51.4%, ESV 59.9%), revealing calibration failures that would remain hidden in conventional point-estimate systems. Model-derived uncertainty identified high-error studies ($|\Delta EF| > 10$ percentage points) with an area under the receiver operating characteristic curve of 0.758 on held-out testing and remained informative under external validation. Adaptation using approximately 100 locally labelled studies recovered most of the calibration deficit. The framework also provides phase localisation, anatomical attention maps, and volumetric trajectories to support review of individual predictions.
Calibrated, volumetric, AI-assisted assessment of left ventricular function enables estimation of EDV, ESV, and EF, along with uncertainty and interpretable evidence to support clinical review. Such systems may facilitate safer deployment of cardiovascular AI across echocardiography and point-of-care ultrasound environments while supporting referral, escalation, and specialist decision-making under uncertainty.

Sex-specific central haemodynamic determinants of central pulse pressure in young, normotensive, black South African adults

K Sithole, A Kolkenbeck-Ruh, A Woodiwiss and V Peterson
Department of Physiology, University of the Witwatersrand, South Africa

In South Africa, cardiovascular diseases are responsible for 1 in every 6 deaths, making them a significant public health concern. Although considerable attention is given to treating cardiovascular diseases, it is equally crucial to identify those at risk of developing cardiovascular diseases. This is particularly relevant in South Africa, where individuals of African ancestry develop cardiovascular events approximately 20 years earlier than those in high-income countries. Central pulse pressure (CPP) is a stronger predictor of cardiovascular outcomes than brachial blood pressure. However, the central haemodynamic determinants of CPP in young, healthy South African adults remain poorly understood. Furthermore, sex differences in these determinants have not been investigated using individuals' true aortic flow waveforms. This study aimed to investigate the central haemodynamic determinants of CPP in normotensive, black South African males and females aged 18–34 years.

This cross-sectional study recruited 132 participants (65 males and 67 females). Anthropometric measurements, fasting glucose, cholesterol, brachial blood pressure, and heart rate were assessed. Central haemodynamic measurements, including CPP and pulse wave velocity (PWV), were obtained using the SphygmoCor XCEL device. Forward pressure wave (Pf) and backward pressure wave (Pb) were determined from aortic velocity and diameter measurements obtained with the Philips ultrasound device. Sex-specific multivariable linear regression analyses were performed to identify independent determinants of CPP.

In males, Pf (β = 0.545; $p < 0.0001$) and Pb (β = 1.163; $p < 0.0001$) were positively associated with CPP, whereas heart rate was inversely associated with CPP (β = -0.100; $p = 0.005$). In females, Pf (β = 0.659; $p < 0.0001$), Pb (β = 1.069; $p < 0.0001$), and waist-to-height ratio (β = 10.04; $p = 0.045$) were positively associated with CPP, while heart rate was inversely associated with CPP (β = -0.030; $p = 0.005$). Age, PWV, body mass index, glucose, and cholesterol were not independently associated with CPP in either sex.

The CPP in young, normotensive, black South African adults was mainly influenced by both the Pf and Pb in males and females. Heart rate was negatively associated with CPP in both sexes, while waist-to-height ratio was an additional determinant only in females. These findings suggest that central haemodynamic factors play an important role in shaping CPP before the development of premature cardiovascular events.

Mitochondrial respiratory function in people living with HIV with cardiometabolic disease: A longitudinal study

K Mokoena,[1] I Webster,[1] A Genis,[1] T Boltman,[1] J Cambell,[1] C Kruger,[1] J Holm,[1] V Mbombela,[1] J Adoga,[1] M Kidd,[2] K Erlandson,[3] I Schauer,[3] G Maarman[1] and H Strijdom[1]

[1] Division of Medical Physiology, Stellenbosch University, South Africa

[2] Department of Statistics and Actuarial Sciences, Stellenbosch University, South Africa

[3] University of Colorado Anschutz, United States of America

Cardiometabolic disease (CMD) is increasing in people living with HIV (human immunodeficiency virus) (PLWH). Both HIV and antiretroviral therapy (ART) can impair mitochondrial function, but the role of HIV-associated CMD in mitochondrial function remains poorly characterised, particularly in sub-Saharan Africa. The objective was to investigate the longitudinal relationship between HIV status, CMD, and mitochondrial function in a Western Cape cohort.

This longitudinal study enrolled 182 participants (PLWH and HIV controls) at baseline. CMD was defined as the presence of ≥ 3 cardiometabolic risk factors. Mitochondrial function was measured in isolated peripheral blood mononuclear cells using high-resolution respirometry (Oroboros O2k-FluoRespirometer). Statistical analyses were performed using SPSS Statistics version 30.0 and Statistica version 14.3.

The cohort was predominantly female (~ 73%), aged ~ 40 years, with high smoking rates. PLWH had a higher prevalence of intermediate-to-high liver fibrosis risk (FIB-4) than HIV controls at baseline (23.8% vs. 5.2%; $p < 0.001$) and follow-up (20.3% vs. 6.3%; $p < 0.001$). Estimated glomerular filtration rate (mean ± standard deviation) was significantly lower in PLWH at baseline (99.52 ± 20.99 vs. 112.68 ± 13.72; $p < 0.001$) and follow-up (99.31 ± 20.20 vs. 111.72 ± 13.47; $p < 0.001$). Most participants had low-grade inflammation (high-sensitivity C-reactive protein > 2 mg/L; PLWH ~ 71%, HIV- ~ 58%). Mitochondrial routine respiration (mitochondrial oxygen consumption, pmol/s/10^6 cells/ml) was lower in PLWH than in HIV- at baseline, with means and 95% confidence intervals (CI): 0.78 (0.42 to 1.13) versus 1.74 (1.37 to 2.11) ($p < 0.001$), but not at follow-up. Overall, there was no interaction between HIV and CMD status regarding mitochondrial function. However, HIV status did significantly interact with individual risk factors, such as hypertension and low high-density lipoprotein cholesterol, at multiple respiratory states at both time points, while HIV interacted with elevated fasting glucose at baseline. Haemoglobin A1c was associated with several respiratory states, predominantly in PLWH at both time points, while lipid variables were significantly associated with various respiratory states irrespective of HIV status. Interestingly, mitochondrial function was not associated with CD4 count, viral load, or ART regimen line.

HIV status and CMD risk were associated with altered mitochondrial function at both time points, with evidence of interactions between HIV status and several CMD risk factors. While some trends varied between baseline and follow-up, the findings highlight the importance of longitudinal studies and support further investigation into HIV-associated CMD risk and mitochondrial function in PLWH.

Assessing the impact of specific preventive measures on the rate of infected cardiac implantable electronic devices at Tygerberg Hospital: A retrospective cohort study

L Wille, J Steyn, A Doubell and J Moses
Department of Cardiology, Stellenbosch University, Tygerberg Hospital, South Africa

Cardiac implantable electronic device (CIED) infection is a dreaded complication that causes substantial morbidity, mortality, and expense. CIED infection rates have increased both globally and at our hospital in recent years. We optimised and implemented specific preventive measures in December 2022. This study aimed to assess the impact of these measures by comparing trends in CIED infection rates before and after implementing the infection prevention bundle, and to describe associated risk factors.
We performed a retrospective analysis of all CIED procedures between January 2013 and March 2025, comparing the pre-intervention period (January 2013 to December 2022) with the post-intervention period (April 2023 to March 2025). A 3-month run-in period (January 2023 to March 2023) preceded the post-intervention period to allow staff to become acquainted with the prevention bundle before assessing its impact. Annual infection rates per 100 procedures were described for each year. Associated risk factors were examined by chi-squared and Fisher's exact tests in a case-control dataset.
The study included 2 040 CIED procedures: 1 662 in the pre-intervention period and 378 in the post-intervention period. We identified 69 cases with infection: 53 in the pre-intervention period and 15 in the post-intervention period. The cases were compared with 505 non-infected controls from 2022 to 2025. The annual infection rate was low from 2013 to 2015, peaked at 7.86 per 100 procedures in 2022, and was 3.01, 4.58, and 3.70 in 2023, 2024, and 2025, respectively. Post-intervention rates were below the 2022 peak but did not return to 2013–2017 levels. Nine of 21 risk factors were associated with infection, including raised inflammatory markers, obesity, current smoking, coronary artery disease, previous myocardial infarction, and device type (cardiac resynchronisation therapy pacemaker). Infection rates declined after the prevention bundle was introduced but did not return to the earlier levels seen in 2013–2017. Further analysis with a longer post-intervention period will strengthen the data. Infection-preventive protocols must continue to improve in our hospital. A multicentre approach is also needed to address this important clinical problem.

Predictors of intensive care unit admissions and outcomes in patients with acute heart failure: A substudy of a multicentre cohort study

L Hahnle,[1] S Stewart[2] and C Alphonsus[1]
[1] Department of Anaesthesia and Peri-Operative Medicine, Groote Schuur Hospital, South Africa
[2] National Echo Database of Australia, University of Notre Dame, Australia

Acute heart failure (AHF) drives significant morbidity in sub-Saharan Africa (SSA), often affecting younger cohorts with non-ischaemic aetiologies. Objective triage tools are essential in resource-constrained settings with limited intensive care unit (ICU) capacity. This study aimed to identify clinical and biochemical predictors of ICU admission in an African AHF cohort and evaluate whether ICU admission independently alters 6-month post-discharge survival.
Utilising data from the multicentre THESUS-HF II cohort, we analysed AHF patients from sites with dedicated ICU availability. Multivariable logistic regression identified independent predictors of ICU admission and 6-month post-discharge mortality, adjusting for baseline demographics, cardiovascular risk factors, New York Heart Association classification, blood pressure, and left ventricular ejection fraction. A secondary subgroup analysis incorporated N-terminal pro-B-type natriuretic peptide values.
Of 1 529 patients included, 97% (n = 1 484) were of African descent (predominantly Nigerian, Tanzanian, and Cameroonian), and 50.3% were female. Baseline prevalences of hypertension, diabetes, and dyslipidaemia were 61.7%, 19.8%, and 14.9%, respectively; 37.1% had prior heart failure diagnoses. Overall, 10.1% (n = 147) were admitted to the ICU. In-hospital mortality was significantly higher in the ICU cohort than in ward patients (17.7% vs. 8.4%). In multivariable logistic regression, increasing age (adjusted odds ratio [aOR] 1.03, 95% confidence interval [CI] 1.02 to 1.04; $p < 0.001$) and a history of diabetes (aOR 2.97, 95% CI 1.91 to 4.62; $p < 0.001$) independently predicted ICU admission. Conversely, prior heart failure decreased the odds of ICU triage (aOR 0.59, 95% CI 0.38 to 0.90; $p = 0.014$). Among patients discharged alive, advanced age was the sole independent predictor of 6-month mortality (aOR 1.02, 95% CI 1.01 to 1.03; $p = 0.004$), whereas index ICU admission did not independently affect post-discharge survival ($p = 0.545$).
Advanced age and diabetes are strong independent predictors of requiring ICU care during AHF hospitalisation in SSA. While ICU patients face high in-hospital mortality, an ICU stay does not independently alter 6-month post-discharge survival, suggesting a need to optimise post-discharge transition-of-care strategies.

Pericardial oedema modifies the relationship between inflammation and fibrosis in tuberculous pericarditis

L Giliomee, A Doubell and P Herbst
Department of Internal Medicine, Division of Cardiology, Stellenbosch University and Tygerberg Hospital, South Africa

The relationship between pericardial inflammation and subsequent fibrosis in tuberculous (TB) pericarditis remains incompletely understood. Emerging cardiovascular magnetic resonance imaging (CMR) staging concepts suggest that pericardial inflammation and oedema may represent biologically distinct processes reflecting different phases of disease activity and reversibility. We hypothesised that oedema modifies the relationship between inflammation and subsequent tethering, an imaging marker of pericardial fibrosis.
Participants with confirmed or probable TB pericarditis underwent baseline and 6-month follow-up CMR. The baseline inflammatory burden was quantified using pericardial late gadolinium enhancement at 6-standard deviation (6SD) and 10-standard deviation (10SD) thresholds. Pericardial oedema and follow-up tethering were assessed segmentally using a validated 20-segment pericardial surface area model incorporating T2 short-tau inversion recovery (T2-STIR) and radiofrequency tissue tagging sequences. Generalised estimating equation modelling was performed to evaluate relationships between inflammatory intensity, oedema, and subsequent tethering.
A total of 45 participants underwent paired imaging, contributing 900 segment-level observations. Follow-up tethering developed in 131/633 oedematous segments (20.7%) compared with 138/267 non-oedematous segments (51.7%; Fisher's exact $p = 1.61 \times 10^{-19}$). In 6SD analysis, oedema was associated with a lower likelihood of tethering (odds ratio [OR] 0.29; $p = 3.90 \times 10^{-10}$), whereas increasing inflammatory intensity increased tethering risk (OR 1.32 per standard deviation increase; $p = 0.0011$). Similar findings were observed using 10SD thresholds. Interaction analysis demonstrated that oedema substantially modified the relationship between inflammatory intensity and subsequent tethering. Segments demonstrating intense inflammation in the absence of oedema exhibited the highest probability of future tethering, whereas oedematous segments demonstrated comparatively lower tethering rates across the spectrum of inflammatory intensity.
The prognostic significance of pericardial inflammation in TB pericarditis appears strongly influenced by the associated oedema profile. Oedema may reflect a more acute and potentially reversible inflammatory phenotype, whereas intense inflammation without oedema appears to identify segments at increased risk of subsequent tethering and fibrosis progression. These findings support a multiparametric CMR approach to disease staging and provide mechanistic insight into progression toward constrictive fibrosis.

Ventricular distribution of pericardial tethering and constrictive haemodynamics in tuberculous pericarditis

L Giliomee, A Doubell and P Herbst
Department of Medicine, Division of Cardiology, Stellenbosch University and Tygerberg Hospital, South Africa

The mechanisms by which visceral–parietal pericardial adhesion translates into clinically significant constrictive haemodynamics remain incompletely understood. We hypothesised that the cumulative burden and ventricular distribution of pericardial tethering may influence the development of constrictive physiology in tuberculous (TB) pericarditis.
Participants with confirmed or probable TB pericarditis underwent baseline and 6-month follow-up cardiovascular magnetic resonance imaging (CMR), including radiofrequency tissue tagging. Pericardial tethering was quantified using a validated 20-segment model comprising 11 left ventricular (LV) and 9 right ventricular (RV) segments. LV, RV, and total tethering burden were expressed as percentages of total pericardial surface area. Constrictive haemodynamics were assessed by echocardiography using Mayo Clinic criteria and corroborated by CMR haemodynamic markers.
A total of 45 participants underwent paired baseline and 6-month follow-up imaging. Constrictive haemodynamics were present in 6/45 participants (13.3%) at the 6-month follow-up. Participants with constriction demonstrated significantly greater LV tethering burden (36.3% [36.3–39.7] vs. 18.2% [13.6–27.2]; $p < 0.001$), RV tethering burden (25.0% [22.2–31.9] vs. 0.0% [0.0–8.3]; $p < 0.001$), and total tethering burden (63.6% [58.5–73.5] vs. 22.7% [13.6–37.3]; $p < 0.001$) than participants without constriction. RV tethering burden was less common overall but showed clearer separation between groups than LV tethering burden. Receiver operating characteristic analysis demonstrated excellent discriminatory performance of RV tethering burden (area under the curve [AUC] 1.00), total tethering burden (AUC 0.991), and LV tethering burden (AUC 0.925). All participants with constrictive haemodynamics demonstrated ≥ 4 tethered RV segments, whereas no participant without constriction reached this threshold (6/6 vs. 0/39; Fisher's exact $p = 1.23 \times 10^{-7}$).
Constrictive haemodynamics in TB pericarditis appear to depend not only on overall tethering burden, but also on ventricular distribution. Pericardial tethering occurs more frequently and extensively over the LV surface, whereas RV involvement is less common. However, because total ventricular constraint is required for constrictive haemodynamics to develop, RV tethering appears more closely associated with the transition toward clinically significant haemodynamic compromise. Therefore, segmental pericardial quantification may provide important mechanistic insight into constrictive physiology.

Rheumatic heart disease in Botswana: A systematic review of disease burden and the national efforts towards prevention and control

M Terblanche,[1] L Ryklief,[2] K Rampersadh,[3] J Mwita[4,5] and L Zühlke[2,6]

[1] School of Public Health, Division of Epidemiology and Biostatistics, University of Cape Town, South Africa
[2] Children's Heart Disease Research Unit, Department of Paediatrics and Child Health, University of Cape Town, South Africa
[3] Cape Heart Institute, Department of Global Medicine, Faculty of Health Sciences, University of Cape Town, South Africa
[4] Department of Internal Medicine, University of Botswana, Botswana
[5] Department of Internal Medicine, Princess Marina Hospital, Botswana
[6] South African Medical Research Council, South Africa

Rheumatic heart disease (RHD) remains a major cause of preventable cardiovascular morbidity and premature mortality globally, with sub-Saharan Africa carrying a disproportionate share of the burden. Botswana, an upper-middle-income country with an established healthcare infrastructure, faces persistent gaps in RHD recognition and control despite broader health system achievements. Country-specific evidence on the burden of Group A streptococcal (Strep A) pharyngitis, acute rheumatic fever (ARF), and RHD has not been systematically synthesised, and progress towards implementing comprehensive prevention and control strategies has not been evaluated. This review aimed to address both objectives.

PubMed, Web of Science, Scopus, African Journals Online, African Index Medicus, Pan African Medical Journal, and grey literature sources were searched for Botswana studies published from January 2012 onwards. We included records that addressed Strep A pharyngitis, ARF, or RHD disease burden, or elements of an RHD control programme according to the 25-domain Wyber framework. We assessed study quality using the Joanna Briggs Institute and Mixed Methods Appraisal Tool, and synthesised findings narratively.

A total of 19 records were included. Strep A pharyngitis prevalence was 7.5% among symptomatic children attending primary healthcare clinics (n = 322, 1 study). Community-based RHD prevalence was 4.0 per 1 000 population (n = 4 786, 1 study). In cardiovascular cohorts, RHD accounted for 10.9% of patients admitted with acute heart failure (n = 193, 1 study) and 37.7% of patients with atrial fibrillation (n = 138, 1 study). Evidence of prevention and control identified a strong primary healthcare foundation, with approximately 84% of the population living within 5 kilometres of a health facility and universal access to free cardiac interventions. Nonetheless, gaps remained in key areas, including essential medicine availability, antibiotic prescribing practices, and specialised diagnostic and cardiac care, which remain both limited and centralised.

Botswana represents a high-risk setting for RHD, with limited available country-specific data. Although important healthcare and policy foundations for RHD prevention and care exist, gaps remain in surveillance, service delivery, workforce capacity, and long-term management. Strengthening coordinated national RHD strategies may support earlier detection and improved outcomes.

Distinct arterial and cardiac remodelling patterns

M van Hoogland-van Heerden,[1] P Mntla,[2,3] O Heyneke,[1] T Lechaba,[4] A Woodiwiss,[5] G Norton[5] and H Majane[1]

[1] Department of Physiology, Sefako Makgatho Health Sciences University, South Africa
[2] Department of Cardiology, Sefako Makgatho Health Sciences University, South Africa
[3] Department of Cardiology, Dr George Mukhari Academic Hospital, South Africa
[4] Department of Pharmaceutical Sciences, Tshwane University of Technology, South Africa
[5] Department of Physiology, University of the Witwatersrand, South Africa

Hypertension affects approximately 30% of Africans and is a major contributor to heart failure with preserved ejection fraction (HFpEF), typically in older adults due to arterial stiffness. However, HFpEF has been observed in younger black South African populations. Given the frequent presence of left ventricular hypertrophy (LVH) in hypertensive individuals, this study aimed to determine whether hypertensive patients with HFpEF exhibit distinct clinical, haemodynamic, and cardiac structural characteristics compared with those with hypertension alone.

A community-based case-control study included 125 HFpEF cases and 510 controls. Of these, 95 hypertensive HFpEF cases and 256 hypertensive controls were analysed. Clinically stable adults (> 18 years) were enrolled. Assessments included echocardiographic, anthropometric, haemodynamic, and pulse wave velocity (PWV) measurements.

The mean age was approximately 58 years, with about 80% overweight or obese participants. Compared with controls, HFpEF participants had lower central diastolic blood pressure (DBP) (p = 0.0295) and central pulse pressure (PP) (p = 0.0352), but significantly higher PWV (10.9 ± 4.8 vs. 6.9 ± 3.9 m/s; p < 0.0001), indicating increased arterial stiffness. Concentric LVH predominated in HFpEF (73%), whereas eccentric LVH was more common in controls (25%). Peripheral blood pressure was associated with the ratio of early (E) to late (A) ventricular filling velocities (E/A), while peripheral DBP, peripheral PP, and E/A correlated with antihypertensive treatment. HFpEF was further associated with reduced renal function, higher heart rate, and elevated inflammatory markers (C-reactive protein and galectin-3).

Hypertensive HFpEF is characterised by increased arterial stiffness, concentric LVH, impaired renal function, elevated heart rate, and heightened inflammation. These findings support a role for adverse arterial–ventricular coupling and inflammation-driven concentric remodelling in HFpEF.

Validation of non-invasive end-systolic wall stress estimation on cardiovascular magnetic resonance imaging

M Rajah,[1] J Liebenberg,[2] A Doubell[1] and P Herbst[1]

[1] Division of Cardiology, Department of Medicine, Stellenbosch University and Tygerberg Hospital, South Africa

[2] Winelands Cardiology, Mediclinic Vergelegen, South Africa

End-systolic wall stress (ESWS) associates near-perfectly with left ventricular ejection fraction (LVEF) in the context of normal loading conditions. However, it may add significant additional value in assessing ventricular mechanics under high afterload conditions. Cardiac catheterisation is the gold standard for ESWS measurement, but it is invasive, costly, and requires specialist expertise, equipment, and infrastructure. Cardiovascular magnetic resonance imaging (CMR) offers a more accessible, non-invasive alternative. We aimed to validate non-invasive ESWS on CMR in severe aortic stenosis (AS).

We recruited 17 patients with high-gradient (mean gradient > 40 mmHg) severe AS (valve area < 1.0 cm^2). Gold standard ESWS measurements were acquired invasively during left heart catheterisation using high-fidelity micromanometer-tipped pressure and conductance catheters (CD Leycom, Netherlands). Participants then underwent a standardised research CMR on a 1.5 T MAGNETOM Aera scanner (Siemens, Germany). The left ventricular end-systolic diameter and wall thickness measurements were taken from a basal short-axis cine slice by a reader blinded to invasive measurements. Systolic blood pressure (SBP) was measured at the time of CMR and added to the mean gradient as a surrogate of left ventricular pressure (LVP). Non-invasive ESWS was calculated using the Laplace-derived equation previously validated in a non-AS cohort by Reichek, et al.

The mean invasive ESWS in the cohort was 109.7 ± 42.1 $\times 10^3$ dyn/cm^2 compared with 187.8 ± 126.3 $\times$ 10^3 dyn/cm^2 by non-invasive CMR calculation (p = 0.07). The measurements showed a strong linear relationship in both Spearman analysis (rho = 0.82, 95% confidence interval [CI] 0.55 to 0.94; p = 0.0001) and simple linear regression (standardised beta coefficient = 0.81, 95% CI 1.46 to 3.40; p < 0.001). The mean invasive LVP was 171.5 ± 34.6 mmHg compared with the surrogate LVP of 174.8 ± 24.6 mmHg (p = 0.87). In a simple linear regression analysis, LVP measurements showed an insignificant relationship (standardised beta coefficient = 0.46, 95% CI -0.02 to 0.67; p = 0.06), driven primarily by SBP variation.

Non-invasive ESWS estimation using CMR serves as a robust surrogate for invasive measurements and may be useful for various research applications.

Drivers and implications of left ventricular dilation in pressure overload: Insights from serum proteomics and cardiovascular magnetic resonance imaging

M Rajah,[1,2] E Marais,[2] G Maarman,[2] A Doubell[1] and P Herbst[1]

[1] Division of Cardiology, Department of Medicine, Stellenbosch University and Tygerberg Hospital, South Africa

[2] Centre for Cardio-Metabolic Research in Africa, Division of Medical Physiology, Faculty of Medicine and Health Sciences, Stellenbosch University, South Africa

Left ventricular (LV) pressure overload conditions, including aortic stenosis (AS), are typically associated with hypertrophic (left ventricular hypertrophy (LVH)) remodelling rather than LV dilation. We previously showed that low ejection fraction (left ventricular ejection fraction [LVEF] < 50%) high-gradient (LEF-HG) AS is associated with LV dilation rather than insufficient LVH, as historically hypothesised. The mechanism and haemodynamic implications of LV dilation in LEF-HG AS are unknown. We aimed to evaluate the association between LV dilation and systolic dysfunction in high-gradient AS using cardiovascular magnetic resonance imaging (CMR) and to explore the molecular pathways underlying LV dilation.

We recruited 34 patients with high-gradient severe AS (LEF-HG) and without systolic dysfunction normal ejection fraction-high gradient (NEF-HG). End-systolic wall stress (ESWS), LVH, and LV dilation were quantified and compared using CMR. Association analyses were also performed for LV remodelling and ESWS/LVEF. Serum was isolated at the time of CMR from whole venous blood. The isolated serum underwent untargeted proteomic profiling using liquid chromatography-tandem mass spectrometry to identify potential LV dilation-associated pathways.

Of 34 participants, 17 (50%) had LEF-HG AS (LVEF 26.5 ± 8.6% vs. 62.1 ± 8.8%). Both groups had evidence of LVH (LEF-HG vs. NEF-HG: wall thickness 13 ± 3 vs. 14 ± 2 mm; p = 0.2, and LV mass index 84.9 ± 25.7 vs. 80.6 ± 22.9 g/m^2; p = 0.63). Non-invasive ESWS was higher in the LEF-HG group (278.5 ± 67.1 vs. 126.0 ± 63.8 $\times 10^3$ dyn/cm^2; p < 0.0001). Significant LV cavity dilation was present in the LEF-HG AS group (LV end-diastolic volume index 125.6 ± 37.6 vs. 79.0 ± 15.5 ml/m^2; p = 0.0003). In univariate and multivariate analyses adjusting for age, sex, valve area, and systolic blood pressure, elevated ESWS independently predicted a reduced LVEF, and cavity dilation was independently associated with elevated ESWS. Protein crumbs homolog-2 (CRB2), a known role-player in eccentric LV remodelling, was significantly upregulated in the LEF-HG group (2.82-fold increased expression at a false discovery rate of 5%; p = 0.013). Systolic dysfunction in high-gradient severe AS is mediated through persistently elevated ESWS from LV dilation. Upregulation of CRB2 requires further validation and mechanistic investigation in pressure overload-induced LV dilation.

Longitudinal electrocardiographic changes following COVID-19 pneumonia in an African cohort

M Cremer,[1] N Glover,[2] I Kalla,[1,3] O Ivanova,[4] F Sathar,[2] S Charalambous,[2,5] J Nagudi,[2] A Rachow[4,7,8] and R Meel[3]

[1] Department of Internal Medicine, Charlotte Maxeke Johannesburg Academic Hospital, South Africa
[2] Research Division, The Aurum Institute, South Africa
[3] Department of Internal Medicine, Faculty of Health Sciences, University of the Witwatersrand, South Africa
[4] Internal Medicine, Institute of Infectious Diseases and Tropical Medicine, LMU University Hospital, Germany
[5] School of Public Health, University of the Witwatersrand, South Africa
[6] Infectious Diseases, German Centre for Infection Research, Germany
[7] Unit Global Health, Helmholtz Zentrum München, German Research Centre for Environmental Health, Germany

Cardiovascular sequelae after COVID-19 remain incompletely characterised in sub-Saharan Africa, where comorbidities such as tuberculosis, human immunodeficiency virus (HIV), hypertension, and diabetes may influence long-term outcomes. This study evaluated longitudinal electrocardiographic changes among adults recovering from COVID-19 pneumonia in Johannesburg, South Africa.
A prospective observational cohort study was conducted at a tertiary hospital in Johannesburg, South Africa. Adults aged ≥ 18 years with confirmed SARS-CoV-2 infection participated from March 2021 to November 2022. Standard 12-lead electrocardiograms (ECG) were obtained at baseline, 6 months, and 12 months. The primary outcome was the composite of incident ECG abnormalities in participants without baseline abnormalities. Secondary outcomes were incident conduction abnormalities and longitudinal changes in rhythm. Cox proportional hazards regression was used to identify predictors of incident ECG abnormalities.
The study included 104 participants: 64.4% were female, 93.3% were black Africans, the median age was 48.3 years, 36.5% were HIV-positive, and 18.3% had a history of tuberculosis. At baseline, 65 participants (62.5%) had at least 1 ECG abnormality. The most common findings were sinus tachycardia (n = 44, 42.3%), T wave abnormalities (21.2%), ST-segment abnormalities (13.5%), and atrial fibrillation (12.5%). The prevalence of any ECG abnormality was 54.9% (n = 50/91) at 6 months and 57.6% (n = 38/66) at 12 months. Sinus tachycardia decreased from 42.3% at baseline to 10.6% at 12 months, while atrial fibrillation decreased from 12.5% to 3.0%. Conversely, the prevalence of atrioventricular conduction abnormalities increased from 1.9% at baseline to 9.1% at 12 months. Among 39 participants without baseline ECG abnormalities, 19 developed incident composite ECG abnormalities. Male sex was protective against incident abnormality adjusted hazard ratio (aHR) 0.6, 95% confidence interval [CI] 0.5 to 0.9; p = 0.01), whereas a history of tuberculosis was associated with an increased risk (aHR 1.4, 95% CI 1.1 to 1.9; p = 0.01).
ECG abnormalities were common and frequently persisted for up to 12 months after COVID-19 pneumonia in this African cohort. Rhythm abnormalities improved over time, whereas conduction abnormalities increased, suggesting possible delayed cardiac electrical involvement. Tuberculosis may influence post-COVID-19 cardiovascular risk in high-burden settings and warrants further investigation.

Biomarkers of collagen metabolism in hypertensive aortopathy and correlation with strain in an African cohort

N Dullabh,[1] T Dix-Peek[1] and R Meel[1,2]

[1] Department of Internal Medicine, Faculty of Health Sciences, University of the Witwatersrand, South Africa
[2] Mediclinic Sandton Hospital, South Africa

This study aimed to characterise circulating biomarkers of collagen metabolism and profibrotic signalling in hypertensive aortopathy in an African cohort, and to assess their relationship with aortic mechanical function using deformation-based echocardiography.
This retrospective study was conducted at Chris Hani Baragwanath Academic Hospital (2017–2019) and included adults with confirmed hypertensive aortopathy (n = 72) and healthy controls (n = 72). All echocardiographic measurements followed American Society of Echocardiography guidelines for chamber quantification and speckle-tracking echocardiography. Biomarkers of collagen turnover, including procollagen type III amino-terminal propeptide (PIIINP), procollagen type I carboxy-terminal propeptide (PICP), matrix metalloproteinase-1 (MMP-1), tissue inhibitors of metalloproteinase-1 (TIMP-1), and chronic inflammation (transforming growth factor-beta [TGF-β]) isoforms were quantified in a phenotyping subset.
The mean age of the hypertensive aortopathy cohort was 57.44 ± 14.80 years, and 53.1% were female. Compared with controls, hypertensive aortopathy showed a larger ascending aortic diameter (47.81 ± 9.82 vs. 25.04 ± 3.47 mm; p < 0.001) and a lower left ventricular ejection fraction (47.55 ± 13.32% vs. 61.63 ± 7.25%; p < 0.001). Aortic circumferential strain (CS) (4.4% [3.1–6.0] vs. 11.1% [8.0–14.7]; p < 0.001) were reduced compared with controls. Aortic stiffness, as measured by the β-stiffness index, was higher in the hypertensive aortopathy cohort (p = 0.0003). Hypertensive aortopathy showed selective type III collagen turnover, with higher PIIINP than controls (p = 0.003) and preserved type I collagen synthesis (p = 0.474). Increased MMP-1 (p = 0.023) and MMP-1:TIMP-1 ratio (p = 0.0078) were noted in the hypertensives compared with controls. Pro-inflammatory cytokine TGF-β2 (p = 0.001) and TGF-β3 (p = 0.001) isoforms were also higher in the hypertensive aortopathy cohort. Biomarkers did not correlate with aortic CS.
Hypertensive aortopathy in this African cohort displayed extracellular matrix remodelling likely driven by upstream TGF-β up-regulation and impaired aortic deformation, but without biomarker–strain coupling. Both biomarkers and strain additively serve as tools for early remodelling phenotyping and may refine risk stratification in hypertension-associated aortopathy.

Retrospective analysis of double valve replacement surgery at Groote Schuur Hospital: Long-term outcomes

N Manzini, T Pennel and J Scherman
Chris Barnard Division of Cardiothoracic Surgery, Groote Schuur Hospital, South Africa

Rheumatic heart disease (RHD) remains a major cause of advanced multivalvular pathology in low- and middle-income countries. Data on long-term outcomes after double valve replacement (DVR) in African cohorts remain limited. This study evaluated survival and prognostic factors following concomitant aortic and mitral valve replacement at a tertiary South African centre.
We conducted a retrospective, single-centre cohort study of 245 adult patients who underwent DVR between 2005 and 2017. Patients undergoing concomitant tricuspid valve surgery were excluded to maintain a more homogeneous DVR cohort. Baseline New York Heart Association (NYHA) classification, left ventricular function, valve pathology, prosthesis type, operative data, post-operative complications, anticoagulation status, and survival at 30 days, 1 year, and 5 years were analysed. Kaplan–Meier analysis and Cox proportional hazards modelling were used to evaluate survival and identify prognostic factors.
RHD accounted for most cases. Patients were relatively young, with a mean age of 44 ± 14 years. Advanced functional limitation was common at presentation. Mechanical prostheses were used more frequently than bioprosthetic valves. Pre-operative NYHA class III/IV was associated with poorer survival in the RHD cohort (p = 0.007) but had less prognostic influence in the non-RHD cohort hazard ratio (HR) 0.78; p = 0.278). Male sex was associated with poor survival among patients with RHD (p = 0.025). Acute and long-term post-operative complications were similar between RHD and non-RHD cohorts. Overall survival at 1 and 5 years was 90% and 62%, respectively.
DVR in this South African cohort provided acceptable mid- and long-term outcomes despite advanced disease at presentation and resource constraints. Baseline functional status remained an important prognostic marker. Male sex was associated with poorer survival among patients with RHD, identifying a potentially higher-risk subgroup that may require earlier referral, closer post-operative surveillance, and targeted support for anticoagulation and follow-up in Global South settings.

The conundrum of Takayasu panarteritis: A case of recurrent aortic coarctation in a paediatric patient – A dilemma for low- to middle-income settings

O Nyekete,[1] S Zulu,[2] A Nzimela,[2] E Hoosen[2] and D Reddy[1,3]
[1] Discipline of Cardiothoracic Surgery, University of KwaZulu-Natal, South Africa
[2] Discipline of Paediatric Cardiology, University of KwaZulu-Natal, South Africa
[3] Discipline of Congenital Cardiac Surgery, Lenmed Ethekwini Hospital and Heart Centre, South Africa

Takayasu arteritis is a rare, immune-mediated, large-vessel vasculitis that primarily affects the aorta and its major branches. Delayed diagnosis is common because of its rarity, heterogeneous presentation, and close mimicry of other granulomatous infections, such as tuberculosis (TB). A high index of suspicion and a multidisciplinary team approach are pivotal in managing this pathology. We present an unusual case of Takayasu pan-arteritis in a paediatric patient with recurrent coarctation of the juxtaductal aorta.
A 7-year-old female patient, previously well with no history of TB, presented with systemic hypertension, cardiac failure, and pulmonary oedema. Echocardiogram and computed tomography angiogram revealed a discrete juxtaductal aortic coarctation, which required an urgent Formula stent. Five months later, she presented with restenosis distal to the previous stent and was successfully managed with re-stenting. On the third occasion, she presented 6 months post-stent with a critical distal restenosis in hypertensive emergency, end-organ malperfusion, and elevated acute phase reactants before referral for open surgical revascularisation, where an extra-anatomical conduit was constructed. A Mantoux test was positive, GeneXpert was negative, and connective tissue screen was negative. A diagnosis of type 2B Takayasu was made. The patient was commenced on anti-TB therapy, glucocorticoids, immunomodulators, and antihypertensives, with a good response. There was no residual gradient.
The unusual presenting symptoms, hindering timely diagnosis and multidisciplinary treatment, should be noted. The diagnostic dilemma is amplified further in the areas where other systemic inflammatory diseases, like TB, are prevalent. Upon diagnosis, distinguishing between active and quiescent disease remains a clinical challenge. Revascularisation is limited to lesions that pose a high risk of complications, like end-organ ischaemia. These should be done when the disease is in remission. Data regarding the ideal revascularisation method remain limited.
Rare as it is, Takayasu arteritis is potentially life-threatening. A high index of suspicion, supported by radiological features and a multidisciplinary approach, can result in prompt diagnosis and management. Open surgical revascularisation has proved effective in our case. Resource constraints in low- to middle-income countries are detrimental for such presentations.

Collagen metabolism biomarkers and aortic mechanics: A novel screening approach in people living with HIV

P Wu and R Meel
Department of Internal Medicine, Faculty of Health Sciences, University of the Witwatersrand, South Africa

HIV (human immunodeficiency virus) is an independent risk factor for thoracic aortic aneurysms (TAA), a potentially life-threatening condition that may remain clinically silent until dissection or rupture. Its pathophysiology and natural history in people living with HIV (PLWH) remain poorly understood.
In this substudy, we performed a retrospective secondary analysis of data derived from a previously conducted prospective study on aortic aneurysms at Chris Hani Baragwanath Academic Hospital (2017–2022). The study included 2 control arms: 57 PLWH without aortic disease and 56 age- and sex-matched controls. We enrolled PLWH without aortic disease and healthy control participants at different time points. Echocardiographic assessments and biomarker measurements were performed independently within each group. Aortic dimensions were measured in the parasternal long-axis view per American Society of Echocardiography guidelines, and ascending aortic circumferential strain (CS) was assessed by speckle-tracking echocardiography. Biomarkers of collagen metabolism were analysed using a magnetic Luminex screening assay.
PLWH without overt aortic disease had a mean age of 41.67 ± 8.84 years. Compared with controls, they demonstrated significantly higher levels of matrix metalloproteinase-1 (MMP-1) (15.05 ± 17.08 vs. 8.13 ± 5.80 ng/ml; $p = 0.007$) and procollagen III N-terminal propeptide (PIIINP) (680.06 ± 250.67 vs. 582.07 ± 196.87 ng/ml; $p = 0.003$). The MMP-1-to-tissue inhibitors of metalloproteinase-1 (TIMP-1) ratio (0.06 [0.03–0.09] in PLWH vs. 0.04 [0.03–0.06] in the control group; $p = 0.96$) was not significantly different between groups, possibly indicating a temporal dissociation between matrix turnover activity and aorta remodelling. Consistent with increased aortic stiffness, PLWH demonstrated lower aortic CS than normal controls (8.67 ± 5.06% vs. 10.51 ± 4.27%; $p < 0.005$) and higher β2-stiffness index (0.06 ± 0.04 vs. 0.05 ± 0.02; $p = 0.0125$). Aortic dimensions were greater in PLWH than normal controls at the sino-tubular junction (27.07 ± 3.75 vs. 25.62 ± 2.96 mm; $p = 0.007$) and aortic annulus (27.67 ± 3.52 vs. 25.73 ± 3.09 mm; $p = 0.001$).
PLWH without overt aortopathy demonstrated evidence of subclinical aortopathy. HIV-associated aortopathy represents a progressive disease spectrum developing before the onset of clinically detectable aneurysmal changes. Biomarkers of collagen metabolism and imaging provide additive value to early disease detection. In high HIV-burden settings, these findings identify an opportunity for earlier detection and targeted cardiovascular surveillance, including consideration of routine point-of-care aortic ultrasound screening.

Atrial strain: A sensitive marker for early cardiovascular disease in people living with HIV

P Grobler
Division of Cardiology, Tygerberg Hospital, South Africa

People living with HIV (human immunodeficiency virus) (PLWH) have a higher risk of cardiovascular disease (CVD), with evidence of early cardiac abnormalities demonstrated on cardiac magnetic resonance imaging (CMR). Although this imaging modality can detect these abnormalities early, limited access hinders its clinical utility. The long-term impact of these abnormalities and their interaction with antiretroviral treatment (ART) are unclear. Left atrial (LA) strain analysis to detect early cardiac abnormalities in PLWH is under-investigated. This imaging modality may represent a practical alternative to CMR for identifying subclinical cardiac abnormalities in PLWH.
ART-naïve persons with HIV infection were recruited, along with HIV-uninfected, age- and sex-matched controls. All patients and controls underwent comprehensive two-dimensional transthoracic echocardiography, including atrial strain analysis. The HIV group commenced ART and was reassessed after 9 months. LA strain was measured and compared using both paired and unpaired t-tests as appropriate. We included 66 ART-naïve, HIV-infected participants, and 22 HIV-uninfected controls for analysis.
Compared with controls, ART-naïve individuals had a significantly reduced LA reservoir strain (LARS) (37.00 ± 4.42% vs. 31.31 ± 6.98%; $p = 0.001$). Compared with controls after 9 months on ART, the LARS essentially normalised (37.00 ± 4.42% vs. 36.00 ± 6.06%; $p = 0.91$). A similar pattern of impairment and recovery was recorded with conduit and contraction atrial strain analysis.
Subclinical myocardial abnormalities in ART-naïve PWLH and their improvement on ART, previously demonstrated by CMR, may also be detected by LA strain analysis. These findings suggest that LA strain assessment could serve as a screening tool for subclinical cardiovascular dysfunction in PLWH. Furthermore, it may also be a valuable tool for follow-up of PLWH on ART.

A case of pulmonary hypertension secondary to Abernethy malformation type I in a child with heterotaxy syndrome

***R Mahomed*[1,2]**

[1]Division of Paediatric Cardiology, Rahima Moosa Mother and Child Hospital, South Africa

[2]Division of Paediatric Cardiology, University of the Witwatersrand, South Africa

Congenital extrahepatic portosystemic shunt, or Abernethy malformation (AM), is a rare anomaly caused by the bypass of portovenous blood to the systemic circulation. Two forms exist: type I, with complete absence of intrahepatic portal veins and total diversion of portal blood; and type II, with partial shunting and hypoplastic intrahepatic portal veins. AM is frequently associated with congenital heart defects (CHD). Noncardiac anomalies notably include pulmonary hypertension and portopulmonary syndrome. Heterotaxy syndrome describes the abnormal arrangement of thoracic and abdominal organs. This case report describes a 1-year-old female with type I AM associated with heterotaxy syndrome, multiple CHDs, and severe pulmonary hypertension. A retrospective review of the patient's clinical records, cardiac catheterisation findings, and radiological imaging was performed.

Initial presentation was at 6 months with pneumonia and heart failure. Clinical exam showed cardiomegaly, decreased saturation of 90%, a normal heart rate of 117 beats per minute, and cyanosis. Echocardiography, cardiac catheterisation, and computed tomography angiogram (CTA) demonstrated left atrial isomerism (polysplenia, interrupted inferior vena cava with azygous continuation and direct drainage of hepatic veins into inferior vena cava (IVC) and truncated pancreas), left ventricular non-compaction with preserved systolic function, large inlet ventricular septal defect (VSD) with bidirectional shunting, bicuspid aortic valve with mild stenosis (peak-to-peak gradient 9 mmHg), discrete coarctation of the aorta (COA), and a large conical patent ductus arteriosus (PDA) (18 mm) with left-to-right shunting. Severe pulmonary hypertension was confirmed with a dilated right heart, mean pulmonary artery pressure of 50 mmHg, and pulmonary vascular resistance of 5.1 wood units (WU/m^2) on 21% fraction of inspired oxygen (FiO^2), partially reversible to 2.1 WU/m^2 on 100% FiO^2.

Despite medical management with furosemide and spironolactone, persistent cyanosis and unexplained pulmonary hypertension prompted further investigations. Bilateral intrapulmonary "bubble tests" confirmed pulmonary atrioventricular malformations; CTA revealed intrahepatic portal vein (PV) aplasia and an extrahepatic shunt composed of splenic and superior mesenteric veins converging and draining into the IVC, confirming type I AM.

The patient was lost to follow-up but may have benefited from complete CHD repair and PV shunt ligation. This case highlights AM as an important and rare cause of unexplained pulmonary hypertension in children with CHDs. Early recognition and timely intervention may improve clinical outcomes.

Cardiac magnetic resonance imaging findings in a post-hospitalisation COVID-19 cohort of African descent in Johannesburg, South Africa

R Kidula,[1] N Glover,[2] F Sathar,[2] J Nagudi,[2] S Charalambous,[3] I Kalla,[4] O Ivanova,[5] A Rachow[5] and R Meel[1]

[1] Department of Internal Medicine, Faculty of Health Sciences, University of the Witwatersrand, South Africa

[2] Research, The Aurum Institute, South Africa

[3] School of Public Health, University of the Witwatersrand, South Africa

[4] Department of Internal Medicine, Faculty of Health Sciences, Charlotte Maxeke Johannesburg Academic Hospital, South Africa

[5] Infectious Diseases and Tropical Medicine, LMU University Hospital, Germany

COVID-19 is associated with increased cardiovascular morbidity and mortality, especially among individuals with pre-existing comorbidities. Emerging evidence suggests a complex interplay between chronic systemic inflammation, metabolic dysfunction, and virus-induced myocardial injury. However, data on the long-term cardiovascular effects of COVID-19 in African populations remain limited. This study aimed to evaluate post-COVID-19 cardiovascular sequelae using standard cardiac magnetic resonance imaging (CMR) in an African cohort.

We conducted a secondary analysis of CMR data from a larger, prospective post-COVID-19 cohort study. This study was conducted at Chris Hani Baragwanath Academic Hospital, a tertiary, single-centre hospital in Johannesburg, South Africa. All participants had previously been hospitalised for COVID-19 and were followed prospectively at scheduled visits 6–12 months after discharge. CMR was performed at 6–12-month follow-up visits, between September 2021 and November 2022. The assessment included left ventricular ejection fraction (LVEF), T1/T2 tissue characterisation, late gadolinium enhancement (LGE), and focused evaluation of myocardial and pericardial abnormalities. Findings were interpreted using established CMR criteria for myocardial injury.

The study included 25 individuals, predominantly African females (64%), with a mean age of 52 years (standard deviation 14.6). Common comorbidities were obesity (52%), diabetes mellitus (36%), and hypertension (20%). Six months after hospital discharge, 36% exhibited findings consistent with myocardial and pericardial involvement, 40% had isolated pericardial effusion (PE), and 24% had normal CMR. Non-ischaemic LGE was present in 40% of participants, consistent with residual myocardial fibrosis following prior myocarditis and one participant had ischaemic inferior wall LGE. T1/T2-STIR imaging was normal, without evidence of overt myocardial oedema. At 12

months, LGE and PE persisted overall, although 2 participants (10%) showed partial resolution of LGE and 2 more (10%) showed complete resolution of PE. LVEF worsened in the participant with ischaemic heart disease (IHD).
PE and myocardial fibrosis were the predominant findings. Overall change was minimal at 12 months, with partial resolution of LGE and PE observed in a small proportion of participants.

Cardio-therapeutic effects of quercetin against hypoxia-induced injury in H9c2 cardiomyocytes

S Khoza,[1,2] K Obikeze,[2] C Chauke[1] and Z Magwebu[1]

[1] Primate Unit and Delft Animal Centre, South African Medical Research Council, South Africa

[2] School of Pharmacy, University of the Western Cape, South Africa

Hypoxia is the most essential pathogenic factor contributing to myocardial injury and cardiac dysfunction. This study investigated the cardio-therapeutic effects of quercetin in differentiated H9c2 cells under cobalt (II) chloride ($CoCl^2$)-induced hypoxic conditions.
H9c2 cardiomyoblasts were differentiated into cardiomyocytes using 30 nM retinoic acid in a low-serum-supplemented culture medium at 37 °C in a 5% CO^2 incubator. Differentiated H9c2 cells were exposed to varying concentrations of $CoCl^2$ (400–1200 µM) for 24 hours to induce hypoxia. Based on cell viability and cytotoxicity findings using 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide (MTT) and lactate dehydrogenase (LDH) assays, respectively, 800 µM $CoCl^2$ was selected for further experiments. Thus, H9c2 cardiomyocytes were divided into 4 groups: group 1 (control), group 2 ($CoCl^2$-induced), and groups 3 and 4 were induced and treated with quercetin (10 µg/ml) and the cathepsin S reference compound, LY3000328 (1 µg/ml), for 4 hours. Thereafter, Oil Red O staining, reverse transcription-quantitative polymerase chain reaction (RT-qPCR), and enzyme-linked immunosorbent assay (ELISA) were performed to evaluate quercetin's therapeutic effect.
High levels of lipid deposition in the $CoCl^2$-induced group were associated with impaired lipid metabolism, while quercetin significantly reduced lipid droplets ($p = 0.02$). Moreover, significant downregulation of C-reactive protein (CRP) ($p < 0.0001$) and apolipoprotein A1 (ApoA1) ($p < 0.00001$) were observed after treatment with both quercetin and LY3000328. A similar trend was observed in cardiac Troponin I Type 3 (TNNI3/cTn-I) activity after treatment with quercetin ($p = 0.001$) and LY3000328 ($p = 0.002$), providing a cardio-therapeutic effect against $CoCl^2$-induced hypoxia.
The selected compounds showed potential therapeutic effects by reducing lipid accumulation and inflammatory marker expression, thereby providing a cardio-therapeutic effect against $CoCl^2$-induced hypoxia.

The association of HIV status and antiretroviral treatment use with coagulation markers in adults living in the Western Cape Province

S Bakali,[1] M Pieters,[1,2] H Strijdom[3] and Z De Lange-Loots[1,2]

[1] Centre of Excellence for Nutrition, North-West University, South Africa

[2] South African Medical Research Council Extramural Unit for Hypertension and Cardiovascular Disease, North-West University, South Africa

[3] Centre for Cardio-Metabolic Research in Africa, Stellenbosch University, South Africa

HIV (human immunodeficiency virus) and antiretroviral treatment (ART) are implicated in increased cardiovascular disease (CVD) risk, potentially through altered coagulation. Because the mechanisms are unclear, we assessed the association of HIV and ART with coagulation markers in the EndoAfrica study.
People living with HIV (PLWH) on ART at baseline and follow-up ($n = 146$), PLWH initiated on ART (ART-naïve at baseline, $n = 38$), and HIV-free controls ($n = 104$) participated in this 18-month prospective study. Clot properties (turbidity), fibrinogen (Clauss), high sensitivity D-dimer (D-dimer HS), von Willebrand factor (vWFpp) enzyme linked immunosorbent assay (ELISA), and thrombin generation using Caliberated Automated Thrombogram (CAT) assay and plasmin generation assay (PGA) were compared at baseline and prospectively within and between groups.
At baseline, the ART-naïve group had higher D-dimer ($p = 0.002$) and vWFpp ($p = 0.013$), while PLWH on ART had lower fibrinogen ($p = 0.003$) than the other groups. Clots from PLWH on ART had a lower rate of lateral aggregation ($p < 0.001$) and were less dense ($p < 0.001$) than those from controls. Endogenous thrombin potential (ETP) was lower in PLWH on ART ($p < 0.001$), while time-to-peak (ttpeak) was lower in both groups of PLWH ($p = 0.020$). The rate ($p = 0.001$) and amount ($p = 0.013$) of plasmin generated were highest in the ART-naïve group. D-dimer ($p = 0.008$) and vWFpp ($p = 0.053$) decreased in the ART-initiated group over 18 months. D-dimer levels also decreased in PLWH on ART ($p = 0.032$). ETP increased over time in PLWH on ART ($p = 0.025$) and decreased in the ART-initiated group ($p = 0.006$). Plasmin generation markers increased in PLWH on ART, with no changes in the ART-initiated PLWH group. Over 18 months, increases in CD4 count were significantly associated with decreased D-dimer ($p = 0.010$), vWFpp ($p = 0.042$), and decreased plasmin generation.
ART-naïve PLWH presented with endothelial activation (increased vWFpp) and elevated D-dimer and thrombin generation, suggesting increased clotting activity in untreated PLWH. However, the fibrinolytic system appears to compensate, as shown by the increased

plasmin generation rate and amount. ART offers a protective effect against elevated clotting activity, as shown by less dense clot formation and the reduced fibrinogen and ETP levels in PLWH on ART and after ART commencement in the ART-initiated group. Improved immunity reduced endothelial activation and clotting activity.

Left atrial function and structure as assessed by strain echocardiography – An early predictor of atrial fibrillation risk

S Subramoney,[1,3] A Doubell,[1,2] W Derman,[3] J Moses[1,2] and J Steyn[1]

[1] Division of Cardiology, Tygerberg Hospital, South Africa

[2] Department of Medicine, Stellenbosch University, South Africa

[3] Division of Sport and Exercise Medicine, Faculty of Medicine and Health Sciences, Stellenbosch University, South Africa

Left atrial (LA) enlargement is an established risk factor for atrial fibrillation (AF), and LA remodelling is a common physiological adaptation to endurance training. Some endurance athletes develop atrial enlargement and have an increased risk of developing AF. LA strain is a non-invasive echocardiographic modality that provides invaluable pathophysiological information on the 3 distinct phases of the LA cycle: reservoir (LARS), conduit (LAScd), and contraction (LASct). We investigated this modality's role in identifying potential early signals of increased AF risk in endurance athletes.

This prospective, descriptive cross-sectional study included 4 groups: healthy controls, endurance athletes, professional soccer players, and patients with mitral regurgitation (MR) known to be at high risk of developing AF. An echocardiogram, electrocardiogram, and N-terminal pro-B-type natriuretic peptide (NT-proBNP) level were obtained from all participants.

The study population of 116 patients comprised 28 controls, 27 endurance athletes, 35 professional soccer players, and 26 patients with MR. LA volume index (LAVI) and left ventricle (LV) end-diastolic dimension (LVED) were normal in the control group (LAVI 28 + 7 ml/m^2, LVED 46 mm), with LARS, LAScd, and LASct values of 34 ± 7%, -23 ± 6%, and -11 ± 5%, respectively. LAVI (72 + 30 ml/m^2) and LVED (60 ± 1 mm) were significantly increased in the MR group compared with controls ($p < 0.01$). LA strain was also abnormal in the MR group (LARS 21 ± 9%, LAScd -15 ± 7%, LASct -6 ± 4%). NT-proBNP was significantly increased only in the MR group. The LA size was borderline increased in the endurance athletes (LAVI 35 ± 9 ml/m^2). LA strain parameters in the endurance athlete group were normal and not statistically different from the control group (LARS 33 ± 5%, LAScd -20 ± 5%, LASct -14 ± 4%).

Pathological LV volume overload due to MR was associated with abnormal LA strain and elevated NT-proBNP levels. When assessing atrial strain parameters in endurance athletes, we found no indicator that could identify athletes at risk of developing AF. Determining whether LA strain may reveal subtle abnormalities in endurance athletes that indicate a risk of developing AF would require a larger study population and longitudinal follow-up.

Exertion-induced sudden cardiac arrest in long QT syndrome: Prolonged resuscitation with complete neurological recovery and secondary implantable cardioverter defibrillator prevention

S Ghulam Hoosain[1,2]

[1] Department of Cardiology, Nelson Mandela Children's Hospital, South Africa

[2] Department of Paediatrics and Child Health, University of the Witwatersrand, South Africa

Long QT syndrome (LQTS) is characterised by delayed ventricular repolarisation, predisposing affected individuals to malignant ventricular arrhythmias and sudden cardiac death. It may be congenital or acquired, with drug exposure often unmasking an underlying genetic susceptibility. Early recognition is essential, particularly in children presenting with exertional syncope or sudden cardiac arrest.

We discuss a retrospective case study of a 12-year-old boy who presented to the emergency department while receiving cardiopulmonary resuscitation after collapsing during a soccer match. He reported fatigue and palpitations immediately before losing consciousness. Resuscitation was initiated by a bystander physician after pulselessness was confirmed. On hospital arrival, he was in pulseless torsade de pointes, which degenerated into ventricular fibrillation. Return of spontaneous circulation was achieved after approximately 60 minutes of cardiopulmonary resuscitation and 2 defibrillation shocks. The patient experienced exertional syncope during soccer 2 years earlier, which was misdiagnosed as a seizure; previous computed tomography of the brain, echocardiography, and 24-hour Holter monitoring were normal. There was no family history of sudden cardiac death. He had recently taken chlorpheniramine and pseudoephedrine for flu-like symptoms.

Initial electrocardiography showed sinus rhythm, ST-segment depression, and a corrected QT interval of 467 ms. Serial electrocardiograms demonstrated a minimum corrected QT interval of 488 ms. Electrolytes and thyroid function were normal. A Schwartz score of 7 supported a clinical diagnosis of LQTS. Propranolol was commenced, and the patient made a complete neurological recovery with a normal brain magnetic resonance imaging study. Genetic testing identified heterozygous variants of uncertain significance in *DSP* and *TTN*. One month later, an implantable cardioverter defibrillator was inserted for secondary prevention before the family emigrated.

This case underscores the need for a high index of suspicion for LQTS in children with exertional syncope or cardiac arrest, even without a contributory family history. Recurrent exertional events should prompt careful electrocardiographic review and consideration of inherited arrhythmia syndromes. Prompt recognition and treatment, including beta-blockade and implantable cardioverter defibrillator therapy for secondary prevention where indicated, may be lifesaving.

Outcomes of ventricular septal defect closure in children > 2 years with severe pulmonary arterial hypertension

PM Shivaprasad,[1] S Gupta,[1] R Sivasubramanian,[1] L Kadiyani,[1] A Handa,[1] R Palleti,[2] D Velayoudam,[2] P Ramakrishnan Reddy[2] and L Ramakrishnan[3]

[1] Cardiology and Paediatric Cardiology, All India Institute of Medical Sciences, India

[2] Cardiothoracic and Vascular Surgery, All India Institute of Medical Sciences, India

[3] Cardiac Biochemistry, All India Institute of Medical Sciences, India

Operability assessment in late presenters with large ventricular septal defects (VSD) is complex. In low- and middle-income countries, late presentation is common, but outcome data are limited. The VSD-PROVE study prospectively evaluates outcomes of children older than 2 years with large VSDs deemed operable after comprehensive evaluation.

A total of 53 children with large VSDs and severe pulmonary hypertension (PH) underwent clinical evaluation, echocardiography, and cardiac catheterisation with oxygen vasoreactivity testing. Of these, 37 children (mean age 6.5 ± 3.7 years, range 2–18) were deemed operable, underwent surgical VSD closure, and received dual pulmonary vasodilator therapy (sildenafil and bosentan) for 1 year. Follow-up included clinical and echocardiographic monitoring and repeat catheterisation at 1 year.

Resting and exertional peripheral oxygen saturation (SpO^2) were 97.6 ± 1.9% and 94.4 ± 4.1%, respectively. Baseline mean pulmonary artery pressure (mPAP) and pulmonary vascular resistance index (PVRI) were 70.9 ± 9.2 mmHg and 9.2 ± 3.4 WU/m^2, respectively. With oxygen administration, mPAP, and PVRI decreased to 66.3 ± 11.1 mmHg and 5.5 ± 2.5 WU/m^2, respectively. All 37 patients underwent successful VSD closure with no mortality or PAH crisis. One patient required a pacemaker for post-operative complete heart block. No post-operative heart failure was observed. To date, 26 patients underwent follow-up cardiac catheterisation 1 year after surgical VSD closure. Of these, 22 patients had a mPAP ≤ 30 mmHg (84.6%), 2 had mPAP < 40 mmHg, and 2 had significant residual PAH with mPAP 56 and 64 mmHg, respectively. About 18 patients (70%) had mPAP ≤ 25 mmHg. For the remaining 11 patients, 6-month echocardiography showed a predicted right ventricular systolic pressure of 39.4 ± 5.8 mmHg.

Surgical closure of large VSDs with elevated PVRI in carefully selected children is safe and effective. Near-normalisation of mPAP at 1 year in most children is encouraging. A longer follow-up is needed to confirm sustained benefits.

Predictors of 1-year outcomes in TAVI candidates in South Africa – An analysis of the South African Heart Association TAVI Registry (SHARE-TAVI)

T Dawood[1] and H Weich[2]

[1] Department of Medicine, Faculty of Health Sciences, Stellenbosch University, South Africa

[2] Division of Cardiology, Department of Medicine, Faculty of Health Sciences, Stellenbosch University, South Africa

TAVI (transcatheter aortic valve implantation) is an established treatment for severe aortic stenosis; however, patient selection remains challenging, especially in resource-constrained settings. Conventional pre-procedural scoring systems have limited predictive capacity, and data from developing nations remain scarce. Region-specific data are needed to inform pre-procedural predictors of 1-year mortality.

SHARE-TAVI is a national, multicentre registry for patients receiving TAVI in South Africa. Patients who underwent TAVI between May 2014 and 2024 with 1-year follow-up were included. The primary outcome was the assessment of pre-procedural predictors of 1-year mortality. The performance of traditional risk-scoring systems and peri-procedural 30-day mortality was also evaluated. Univariate analysis of all potential predictors was assessed, followed by multivariate analysis and model evaluation.

A total of 2 903 patients were included. One-year mortality was 13.3%, which is comparable internationally. Significant associations with 1-year mortality included extracardiac arteriopathy, diabetes mellitus, dialysis-dependent chronic kidney disease, chronic lung disease, prior/current malignancy, and an inability to drive independently. An increase in mortality with conversion to open-heart surgery (odds ratio 14.92; $p < 0.01$) was found. Conventional scoring systems did not show significant associations with 1-year mortality beyond the Society of Thoracic Surgeons score, showing only modest predictive ability with an area under the curve (AUC) of 0.6. Two multivariate models were proposed: model 1 from the data, and model 2 based on a model developed in a prior study of a local population. Model 1 (AUC 0.6) showed that extracardiac arteriopathy remained a significant predictor of mortality, and the ability to drive independently showed a protective trend ($p = 0.06$). Model 2 achieved improved discrimination (AUC 0.67) and showed lower ejection fraction as a significant predictor of mortality.

In this national registry from a developing nation, 1-year outcomes were comparable to international data, cementing TAVI as a valuable intervention for the population. Conversion to open-heart surgery as an option in a resource-limited setting may need reconsideration.

Conventional risk scores demonstrated limited discriminatory ability. Measures of independent living may be a future adjunct to assessing risk in our population. These findings support the need for region-specific, frailty-informed approaches to optimise patient selection in resource-constrained settings.

Anomalous origin of the right coronary artery in a 69-year-old male: A case report

T Seedat,[1] R Goncalves,[2] L Rampini[3] and R Meel[1]

[1] Department of Internal Medicine, Faculty of Health Sciences, University of the Witwatersrand, South Africa

[2] Cardiology discipline, Netcare Olivedale Hospital, South Africa

[3] Cardiothoracic Surgery discipline, Netcare Olivedale Hospital, South Africa

Coronary artery anomalies (CAA) are rare congenital abnormalities involving the origin, course, or termination of the epicardial coronary arteries. While typically recognised in younger adults and athletes, CAAs may also present in older patients – often discovered incidentally during evaluation for ischaemic heart disease. Certain high-risk features, particularly an interarterial course, can predispose to myocardial ischaemia, arrhythmias, and sudden cardiac death.

We report a 69-year-old male with hypertension and dyslipidaemia who presented with exertional syncope during treadmill exercise. Initial evaluation revealed a normal electrocardiogram, elevated troponin, and echocardiographic features of "athlete's heart" with preserved ejection fraction. Coronary angiography suggested anomalous origin of the right coronary artery (RCA), confirmed by computed tomography (CT) coronary angiography to arise above the sinotubular junction with an interarterial and a slit-like proximal course. A high plaque burden was also noted. The patient underwent surgical reimplantation of the RCA with aortic root repair. Post-operative recovery was uneventful, and 8-month follow-up demonstrated stable cardiac function and symptom resolution.

This case illustrates that CAAs, though often considered a disease of the young, can present in the elderly, particularly under stress conditions that increase myocardial demand. Interarterial RCA courses with high-risk features, such as a slit-like ostium and acute-angle take-off, can produce dynamic and fixed mechanisms of ischaemia, explaining exertional syncope and potential arrhythmias. CT coronary angiography is the gold standard for anatomical delineation and surgical planning, while echocardiography and invasive angiography remain supportive. Surgical correction remains the definitive therapy in symptomatic patients or those with high-risk anatomy, with excellent intermediate-term outcomes.

This case emphasises that CAAs, though typically considered in younger adults, can present in the elderly and should not be overlooked. CT coronary angiography is the preferred diagnostic tool, while echocardiography and invasive angiography remain valuable in guiding management. Early recognition enables timely intervention, thereby reducing the risk of adverse cardiac events.

Ageing and type 2 diabetes differentially modulate adipose tissue depots and circulating cardiovascular risk markers in db/db mice

T Nyawo,[1,5] S Mazibuko-Mbeje,[3] P Dludla,[6] H Sadie-Van Gijsen,[2] H Strijdom[2] and C Pheiffer[1,2,4]

[1] Biomedical Research and Innovation Platform, South African Medical Research Council, South Africa

[2] Centre for Cardio-Metabolic Research in Africa, Division of Medical Physiology, Faculty of Medicine and Health Sciences, Stellenbosch University, South Africa

[3] Department of Biochemistry, Faculty of Natural and Agricultural Sciences, North-West University, Mafikeng Campus, South Africa

[4] Department of Obstetrics and Gynaecology, School of Medicine, Faculty of Health Sciences, University of Pretoria, South Africa

[5] Department of Biochemistry and Microbiology, University of Zululand, South Africa

[6] School of Medicine, College of Health Sciences, University of KwaZulu-Natal, South Africa

Adiposity contributes largely to the development and progression of cardiometabolic diseases by producing inflammatory, oxidative stress-related cytokines, and other chemokines that contribute to cardiovascular dysfunction. Early detection of circulating cardiovascular disease (CVD) risk markers improves risk stratification and clinical management, reducing disease progression and mortality.

Male db/db and d/+ mice aged 8, 12, and 18 weeks were used to evaluate ageing and diabetic status in retroperitoneal, inguinal, and cardiac adipose tissue depots, as well as selected circulating CVD risk markers. Adipose tissue morphology was assessed, and gene expression associated with inflammation, oxidative stress, and thermogenesis was analysed using quantitative polymerase chain reaction (qPCR). Serum levels of CVD risk markers were measured using a MILLIPLEX MAP Mouse CVD Magnetic Bead Panel 1.

Phenotypic and metabolic parameters worsened with age and disease progression, with db/db mice exhibiting hyperglycaemia, hyperinsulinaemia, hyperlipidaemia, and glucose intolerance. Adipocyte hypertrophy increased over time in all adipose depots; however, cardiac fat adipocytes remained smaller than others. Gene expression analysis showed increased inflammatory and oxidative stress markers (*MCP1* and *GST*) in retroperitoneal and inguinal fat of db/db mice compared with db/+ mice, but not in cardiac fat. In contrast, cardiac fat demonstrated higher expression of the thermogenic gene, *UCP1*. Circulating CVD risk markers, including *MMP9*, *ICAM*, *PECAM1*, and thrombomodulin, displayed heterogeneous expression patterns in both db/db and db/+ mice, suggesting vascular remodelling and endothelial dysfunction during disease progression.

Overall, retroperitoneal and inguinal adipose tissues appeared more susceptible to the detrimental effects of type 2 diabetes and ageing, whereas cardiac fat demonstrated distinct metabolic characteristics that may enhance cardiometabolic health. Additionally, CVD risk markers may provide valuable insight into early cardiovascular dysfunction and progression.

Long-term outcomes of multivessel versus culprit-only percutaneous coronary intervention in patients with non-ST-elevation myocardial infarction and multivessel coronary artery disease: A systematic review and meta-analysis

U Adamu,[1] E Adamu,[2] C Makgoro,[1] M Mashilo,[1] A Patel,[1] A Mutyaba[1] and N Tsabedze[1]

[1] Division of Cardiology, Department of Internal Medicine, University of the Witwatersrand and Charlotte Maxeke Johannesburg Academic Hospital, South Africa

[2] Department of Internal Medicine, Federal Teaching Hospital, Nigeria

Multivessel coronary artery disease is frequently observed in patients with non-ST-segment elevation myocardial infarction (NSTEMI), contributing to worse clinical outcomes. However, the benefit of physiology-guided complete revascularisation in this population remains uncertain. We aimed to evaluate the efficacy and safety of physiology-guided multivessel percutaneous coronary intervention (PCI) versus culprit-only revascularisation in patients with NSTEMI and multivessel coronary artery disease.

PubMed, Embase, and Cochrane CENTRAL were systematically searched from inception to September 2025 for randomised controlled trials (RCT) and observational studies comparing multivessel PCI with culprit-only PCI. The outcomes were major adverse cardiovascular events (MACE)/major adverse cardiovascular and cerebrovascular events (MACCE), all-cause mortality, myocardial infarction, any revascularisation, cardiovascular mortality, and hospitalisation for heart failure. We pooled odds ratios (OR) for binary endpoints with 95% confidence intervals (CI) using a random effects model.

A total of 11 studies (2 RCTs, 9 observational) were included, comprising 33 715 patients, of whom 16 858 (50%) underwent multivessel PCI. Compared with culprit-only PCI, multivessel PCI was associated with a reduced risk of MACE (OR 0.55, 95% CI 0.42 to 0.72; $p < 0.001$), all-cause mortality (OR 0.64, 95% CI 0.53 to 0.77; $p < 0.001$), myocardial infarction (OR 0.58, 95% CI 0.45 to 0.74; $p < 0.001$), and repeat revascularisation (OR 0.53, 95% CI 0.40 to 0.70; $p < 0.001$). However, multivessel PCI was associated with a higher risk of stroke (OR 1.85, 95% CI 1.05 to 3.25; $p = 0.033$). No significant differences were observed for cardiovascular mortality (OR 0.73, 95% CI 0.48 to 1.13; $p = 0.157$) or hospitalisation for heart failure (OR 0.90, 95% CI 0.68 to 1.17; $p = 0.418$). Subgroup analyses showed consistent benefit across study designs, though mortality and stroke findings were largely driven by observational studies.

In this meta-analysis of patients with NSTEMI and multivessel disease, multivessel PCI was associated with lower risks of ischaemic events and repeat revascularisation compared with culprit-only PCI, but at the expense of increased stroke risk. These findings highlight the need for adequately powered randomised trials to refine patient selection and guide clinical decision-making in this high-risk population.

Soluble suppression of tumourigenicity-2 provides incremental prognostic value beyond N-terminal pro-B-type natriuretic peptide for predicting 1-year mortality in acute heart failure

U Adamu,[1] L Khambule,[2] S Currin,[2] T Dix-Peek,[3] C Dickens,[3] R Duarte,[3] M Maseko[4] and N Tsabedze[1]

[1] Internal Medicine and Cardiology, University of the Witwatersrand and Charlotte Maxeke Johannesburg Academic Hospital, South Africa

[2] Department of Chemical Pathology, University of the Witwatersrand, South Africa

[3] Department of Internal Medicine and Translational Research, University of the Witwatersrand, South Africa

[4] Department of Physiology and Nutrition and Hypertension Laboratory, University of the Witwatersrand, South Africa

Accurate risk stratification in patients hospitalised with acute heart failure (AHF) remains challenging. Although natriuretic peptides are established prognostic biomarkers, they primarily reflect haemodynamic stress and may not fully capture inflammatory and fibrotic pathways implicated in HF progression. Soluble suppression of tumourigenicity-2 (sST2) has emerged as a promising biomarker, but its prognostic utility beyond natriuretic peptides remains incompletely defined. We investigated the prognostic significance of sST2 to determine whether it provides incremental predictive value beyond N-terminal pro-B-type natriuretic peptide (NT-proBNP) for 1-year mortality in patients hospitalised with AHF.

We prospectively enrolled 406 consecutive adults hospitalised with AHF between February and November 2023. Plasma sST2, NT-proBNP, and matrix metalloproteinase-9 (MMP-9) concentrations were measured at admission. Patients were followed for 12 months. Survival was assessed using Kaplan–Meier analysis, and multivariable Cox proportional hazards regression was used to identify independent predictors of mortality. Incremental prognostic performance was evaluated using receiver operating characteristic (ROC) curve analysis, likelihood ratio testing, Harrell's C-index, and risk reclassification analyses. Statistical analysis was performed using RStudio version 4.5.3.

Among 388 patients with complete follow-up (mean age 54.7 ± 15.9 years; 49% male), 84 deaths (20.7%) occurred during 12 months. Median sST2 concentrations were significantly higher among non-survivors compared with survivors (66.0 [32.7–120.6] vs. 38.0 [19.3–71.3] ng/ml; $p < 0.001$). Kaplan–Meier analysis demonstrated progressively lower survival across increasing sST2 tertiles (log-rank

$p < 0.0001$). In multivariable analysis, log-transformed sST2 remained independently associated with mortality (hazard ratio [HR] 1.57, 95% confidence interval [CI] 1.24 to 1.98; $p < 0.001$), along with NT-proBNP (HR 1.46, 95% CI 1.15 to 1.84; $p = 0.002$). Adding sST2 significantly improved discrimination beyond NT-proBNP alone (area under the curve 0.708 vs. 0.669; $p = 0.042$), improved model fit (likelihood ratio $\chi^2 = 13.9$; $p < 0.001$), and enhanced risk reclassification (continuous net reclassification improvement 0.35, 95% CI 0.12 to 0.59).

In patients hospitalised with AHF, baseline sST2 independently predicts 1-year mortality and provides incremental prognostic value beyond NT-proBNP. Incorporating sST2 into a multimarker strategy improves risk discrimination and patient risk classification, supporting its potential role in enhancing risk stratification in AHF.

O-acetyl-L-carnitine is independently associated with in-hospital and 12-month mortality in acute heart failure

U Adamu,[1] L Khambule,[2] S Currin,[2] M Maseko[3] and N Tsabedze[1]

[1] Internal Medicine and Cardiology, University of the Witwatersrand and Charlotte Maxeke Johannesburg Academic Hospital, South Africa

[2] Department of Chemical Pathology, University of the Witwatersrand, South Africa

[3] Department of Physiology and Nutrition and Hypertension Laboratory, University of the Witwatersrand, South Africa

Risk stratification of patients in acute heart failure (AHF) remains suboptimal. Although conventional clinical and biomarker-based models predict outcomes, the incremental value of targeted plasma metabolomic profiling for both in-hospital and longer-term mortality remains poorly defined. We aimed to evaluate the prognostic utility of targeted metabolites in patients hospitalised with AHF.

We conducted a prospective cohort study of adults admitted with AHF and followed them for 12 months. Plasma concentrations of 9 targeted metabolites were quantified at admission using liquid chromatography-tandem mass spectrometry. Differences between survivors and non-survivors were assessed. Associations with in-hospital mortality were evaluated using multivariable logistic regression, and associations with 12-month mortality were assessed using Cox proportional hazards models. Prognostic performance was examined using discrimination and risk classification metrics by comparing models with and without key metabolites. Statistical analysis was performed using RStudio version 4.5.3.

Among 406 patients (mean age 54.7 years, 51.3% women), 388 (95.6%) completed follow-up and, 84 (20.7%) died within 12 months. Non-survivors had significantly higher plasma O-acetyl-L-carnitine (carnitine-2) concentrations (median 3.8 vs. 3.1 ng/ml; $p < 0.001$), with lower levels of valine (130.0 vs. 150.8 ng/ml) and arginine (46.9 vs. 53.7 ng/ml) ($p < 0.05$). Elevated carnitine-2 was independently associated with in-hospital mortality (adjusted odds ratio 1.84, 95% confidence interval [CI] 1.03 to 3.28) and 12-month mortality (adjusted hazard ratio 2.24, 95% CI 1.22 to 4.12). In a least absolute shrinkage and selection operator-selected and refitted Cox model, carnitine-2 remained independently associated with mortality (hazard ratio 1.23, 95% CI 1.05 to 1.45; $p = 0.013$), along with N-terminal pro-B-type natriuretic peptide and soluble suppression of tumourigenicity-2. Adding carnitine-2 to a multimarker risk model modestly improved discrimination (Harrell's C-index = 0.77, area under the curve = 0.76) and risk classification.

Plasma carnitine-2 was independently associated with both in-hospital and 12-month mortality in AHF. Although its incremental prognostic value beyond established biomarkers was modest, these findings highlight acylcarnitine metabolism as a potentially informative biological pathway and support further validation in larger, multicentre cohorts.

Can atrial and ventricular strain imaging by echocardiography be used to predict progression of pericardial disease to pericardial constriction? A cardiovascular magnetic resonance imaging and echocardiographic comparative analysis

W Naicker, A Doubell and P Herbst

Division of Cardiology, Tygerberg Hospital, South Africa

Tuberculous (TB) pericarditis remains the most common cause of chronic pericardial constriction (constrictive pericarditis [CP]) in South Africa. Transient effusive–constrictive pericarditis (ECP) is commonly diagnosed after drainage of a large pericardial effusion. However, a subset of ECP patients will progress to irreversible CP. Identifying high-risk patients for CP remains challenging using echocardiography. Pericardial disease staging with cardiovascular magnetic resonance imaging (CMR) shows promise in identifying patients at high risk for developing CP, but access to magnetic resonance imaging (MRI) remains limited. This study aimed to assess whether atrial and ventricular strain after drainage of TB pericardial effusions can predict the development of CP or pericardial tethering burden (using CMR) as a proxy for constriction.

Patients undergoing pericardiocentesis for definite or probable TB pericardial effusions were recruited. Echocardiographic assessment of constriction physiology (Mayo Clinic criteria) and atrial and ventricular strain using speckle-tracking was performed at baseline. CMR was performed at baseline and 6 months after TB treatment. Pericardial oedema, inflammation, tethering, and constriction physiology were assessed.

Of the 45 patients recruited, 42 had the required imaging for analysis. Of these, 22 (52%) had ECP, with 6 (13%) progressing to CP at 6 months. Abnormal global longitudinal strain (GLS) and atrial strain were observed in 39/42 (93%) and 41/42 (98%) patients, respectively, including patients with and without ECP. Median GLS measured -9% (interquartile range [IQR] -7 to -10%). Atrial strain median reservoir, conduit, and contraction function measured 17% (IQR 13% to 20%), -11% (IQR -8% to -15%), and -6% (IQR -5% to -8%), respectively. Neither atrial nor ventricular strain correlated with the development of CP. However, a higher number of tethered segments on CMR at baseline was associated with CP development at 6 months (p = 0.001). Additionally, a lower conduit atrial strain was associated with a higher number of tethered segments at baseline (p = 0.036).
In this cohort of patients with TB pericardial effusions, abnormal atrial and ventricular strain was observed in most patients, though it did not predict CP risk. A higher number of tethered ventricular segments on baseline CMR was associated with CP development at 6 months. Ventricular tethering burden was predicted by abnormal atrial conduit strain.

The effect of quercetin on blood components and paraoxonase genes using a vervet monkey (*Chlorocebus aethiops*) model

Z Magwebu, S Khoza and C Chauke
Primate Unit and Delft Animal Centre, South African Medical Research Council, South Africa

Quercetin is a well-studied dietary polyphenol with diverse biological properties, including anti-inflammatory, antioxidant, antihypertensive, and anticancer effects. This study examined the effects of quercetin on biochemical parameters, paraoxonase gene expression, and associated genes in 8 captive-bred vervet monkeys on a high-fat diet.
The Ethics Committee of the South African Medical Research Council approved all animal procedures (Ethics Committee for Research on Animals reference: 03/19). The control group (n = 4) received a 40% high-fat patty in the morning, and the experimental group (n = 4) received a diet supplemented with quercetin (150 mg/kg/day) for 3 months, followed by a 1-month washout period. Blood samples were obtained for biochemical analysis, genotyping, gene expression, and cytokine markers from baseline through the washout period.
Biochemical analyses showed no statistical differences between groups in body weight, low-density lipoprotein cholesterol (LDL-C), total cholesterol, triglycerides, glucose, and glycated haemoglobin A1C (HbA1c) levels. However, high-density lipoprotein cholesterol (HDL-C) levels were significantly higher in the experimental group than in the control group at baseline (p = 0.03) and washout (p = 0.03). Genotyping of *PON1* showed 4 polymorphisms (*M127V*, *Q192R*, *V215L*, *R331S*), and 1 in *PON3* (*H250Q*), and both genes were downregulated by quercetin administration. Moreover, *V215L* carriers had lower LDL-C and cholesterol levels than the wild type, while *H250Q* carriers had significantly lower HDL-C levels (p = 0.03). Although overall inflammatory cytokine levels were not significant, the reduction in interleukin-6 further supports quercetin's anti-inflammatory role.
The findings suggest that quercetin may offer selective molecular benefits by enhancing HDL-C levels and modulating paraoxonase gene expression. However, a longitudinal follow-up study is recommended to determine whether the identified polymorphisms translate into cardiometabolic outcomes, such as atherosclerosis, coronary heart disease, or diabetes, over time.

Pacemaker implantation to facilitate beta-blocker therapy in a patient with long QT syndrome, bradycardia, and high-grade atrioventricular block

Z Ngwenyama,[1] J Lawrenson,[2] A Greyling[3] and G Comitis[4]
[1] Paediatric Cardiology, Rob Ferreira Hospital, South Africa
[2] Children's Heart Disease Research Unit, University of Cape Town, South Africa
[3] Paediatric Cardiology and Electrophysiology, Mediclinic Vergelegen, South Africa
[4] Western Cape Paediatric Cardiology Service, Red Cross War Memorial Children's Hospital, South Africa

We describe the clinical course of a patient with high-grade atrioventricular (AV) block, sinus bradycardia, and long QT syndrome managed at Red Cross War Memorial Children's Hospital. This case review discusses a patient followed over 2 years.
A 7-year-old girl presented for the first time with syncope secondary to high-grade, second-degree AV block (2:1 block), sinus bradycardia, and long QT syndrome. She has a strong family history of syncope. Sinus bradycardia and high-grade AV block on resting electrocardiogram necessitated implantation of a dual-chamber pacing system to facilitate beta-blocker therapy. During transvenous dual-chamber pacemaker (DDD) implantation, the patient developed torsades de pointes. This was triggered by premature ventricular contractions (PVC) from severe bradycardia. Overdrive pacing at a rate of 80 beats per minute was initiated to prevent further PVCs and subsequent torsades episodes, which could lead to sudden cardiac death.
Following pacemaker implantation and titration of beta-blocker therapy to target doses, no further episodes were noted. Genetic testing was not performed because of the associated financial burden.
Successful pacemaker insertion in a child with a high-grade AV block prevented debilitating symptoms and reduced the arrhythmic risk associated with severe bradycardia, which can trigger episodes of torsades de pointes. With beta-blocker therapy, the patient had no further symptoms or arrhythmic events during the 2-year follow-up.

ECG Quiz 74

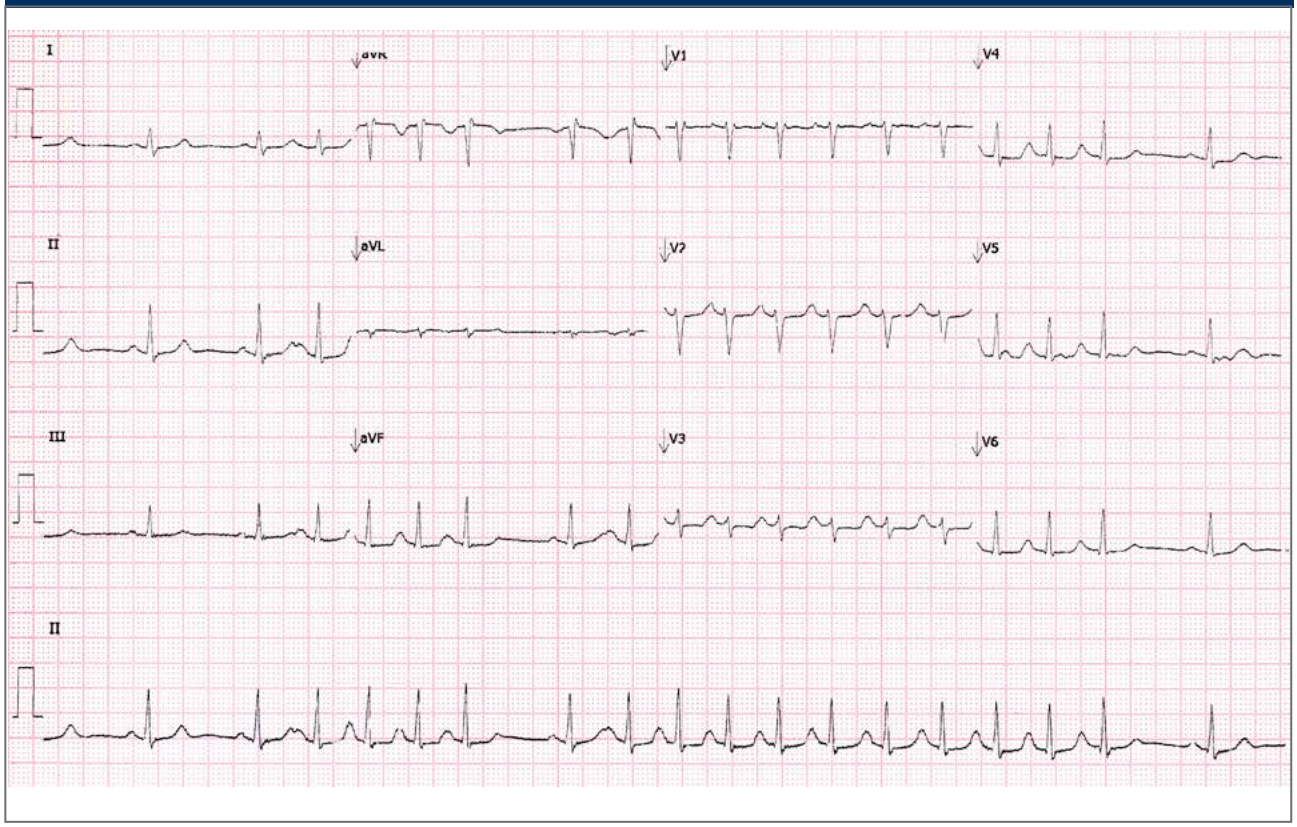
CASSA
CARDIAC ARRHYTHMIA SOCIETY OF SOUTHERN AFRICA
A SPECIAL INTEREST GROUP OF SA HEART®

Rob Scott Millar and Ashley Chin
Cardiac Clinic, University of Cape Town/ Groote Schuur Hospital
Cardiac Arrhythmia Society of Southern Africa (CASSA)

Email:
rob.scottmillar@gmail.com

RS Millar https://orcid.org/0000-0002-5608-0623
A Chin https://orcid.org/0000-0001-6930-3673
DOI: https://doi.org/10.24170/22-4-9195 | https://doi.org/10.24170/22-4-9196
Creative Commons License - CC BY-NC-ND 4.0

The ECG (electrocardiogram) is from a 42-year-old woman who developed palpitations for the first time and presented to an emergency unit (EU).



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

a. Atrial fibrillation.
b. Atrial flutter with variable atrioventricular (AV) block.
c. AV node re-entry tachycardia (AVNRT).
d. Atrial tachycardia.
e. Atrial tachycardia with intermittent exit block.

ECG Quiz 74

CASSA
CARDIAC ARRHYTHMIA SOCIETY OF SOUTHERN AFRICA
A SPECIAL INTEREST GROUP OF SA HEART®

ANSWER

OVERVIEW OF THE ECG

This is an irregular, narrow QRS tachycardia at a mean rate of 108/minute. There are 2 short runs of tachycardia, slightly irregular at about 150/minute, with pauses in between.

More detailed analysis of the ECG

The initial 2 complexes are preceded by P waves with an axis of about +60 ° and a PR interval of 150 ms. The QRS duration is 95 ms, and the QRS axis is about +65 °. The QRS morphology is normal, except for a small terminal r in V1, suggesting an incomplete right bundle branch block. The QT interval of the first and last complex is 390 ms. After the second complex, likely of sinus origin, there are 4 beats of tachycardia. Note the downslope distortion of the T wave, mirrored in Lead III above. This is a premature atrial complex (PAC) (Figure 1), followed by 3 beats of tachycardia, with a cycle length of 400 ms (150/minute). The following pause is 830 ms, ending with a complex likely of sinus origin. This complex is also followed by a PAC, distorting the T wave and initiating a longer run of tachycardia. The cycle length starts at 400 ms and lengthens to 450 ms for the last few beats. The 880 ms pause is followed by a sinus beat. Note the peaking of the T waves in the second run of tachycardia (Figure 1, arrows). A sharp deflection in V1 coincides with the peak of the T waves in Lead II (Figure 2).

COMMENT

The most common cause of an irregular, narrow QRS tachycardia is atrial fibrillation (AF) (Table I).[1,2] From the analysis above, this is not AF; the tachycardia is not randomly irregular. The cycle length increases slightly before the longer run terminates. Each QRS during the tachycardia is preceded by a P wave, which is concealed by the T wave in the Lead II rhythm strip, but is clearly visible simultaneously as a sharp deflection in V1.

While it is possible to have short runs of AF triggered by ≥ 1 PACs, this is unlikely in a young woman with no evidence of structural heart disease and an otherwise normal ECG. Nevertheless, this was the diagnosis made in the EU. The doctor

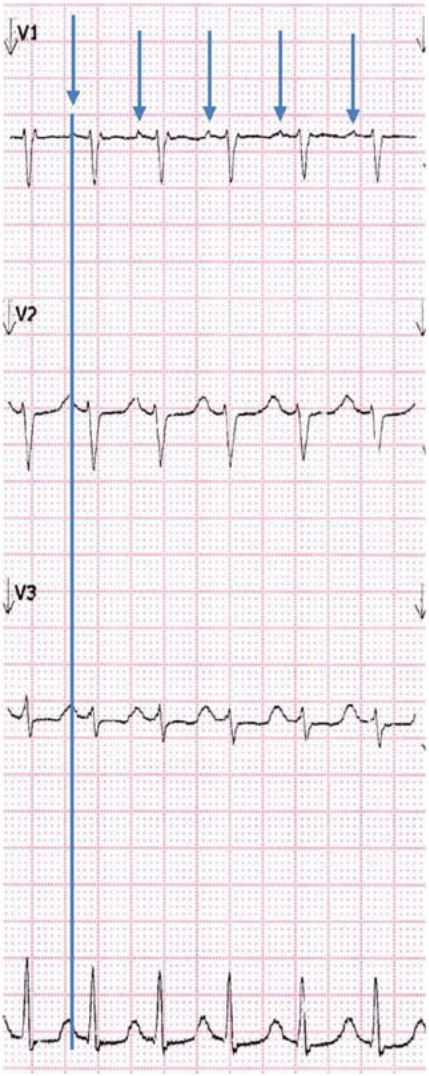


FIGURE 2: Leads V1–3 with Lead II at the bottom, showing the P waves in V1 (arrows) synchronous with the apex of the T waves in Lead II.

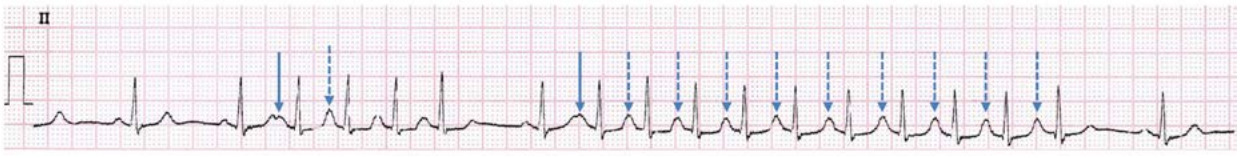


FIGURE 1: Lead II rhythm strip; after 2 sinus beats, a premature atrial complex distorts the T wave and is followed by a run of tachycardia. This is repeated after the pause. The dotted arrows show the P waves distorting the apex of the T waves. The last tachycardia complex is not followed by a P wave.

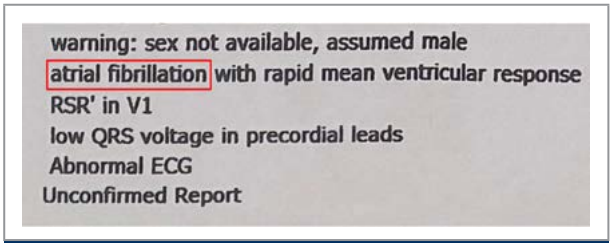


FIGURE 3: Incorrect and misleading computer report falsely diagnosing atrial fibrillation.

in the EU may have been influenced by the computer's false diagnosis of AF, printed on the ECG (Figure 3). The incorrect diagnosis led to her being started on a beta-blocker, which was reasonable, and apixaban, which was not indicated.

The rhythm is not atrial flutter with variable AV block, a common cause of an irregular, narrow QRS tachycardia. There is a single P wave per QRS complex, and all the P waves are conducted. AVNRT must be considered. AVNRT is commonly triggered by a PAC which blocks in the refractory fast pathway. It is then conducted slowly via the slow AV nodal pathway and echoes back to the atrium via the fast pathway, which has now recovered from refractoriness. This results in a very short RP tachycardia, with the retrograde P occurring within or shortly after the QRS. Atypical AVNRT uses the fast pathway as the antegrade limb and the slow pathway retrogradely, resulting in a long RP tachycardia.[3] The case under discussion is a long RP tachycardia. The differential diagnosis of a long RP tachycardia includes sinus tachycardia, atrial tachycardia, atypical AVNRT, and atypical atrioventricular re-entry tachycardia (AVRT).

The P wave axis of the tachycardia can help determine the direction of atrial activation. The P waves are upright in Leads II, III, and aVF, indicating an origin high in the atria and excluding AVNRT and AVRT. The slight slowing before the pauses and irregularity can occur with an atrial tachycardia. Atrial tachycardias are focal in origin with discrete P waves (unlike atrial flutter, a macro-re-entrant arrhythmia with a sawtooth pattern). Atrial tachycardias can cause long RP (common) or short RP (uncommon) tachycardias. Common sites for atrial tachycardias include the high right atrium near the crista terminalis or the pulmonary veins. Incessant atrial tachycardias can cause tachycardia-induced cardiomyopathy. Treatment of atrial tachycardias includes beta-blockers or catheter ablation for resistant cases.

TABLE I: Differential diagnosis of an irregular, narrow QRS tachycardia.

- Atrial fibrillation.
- Atrial flutter with variable atrioventricular block.
- Atrial tachycardia with variable atrioventricular block or intermittent exit block.
- Multifocal atrial tachycardia.

Note: Short runs of otherwise regular supraventricular tachycardias, interrupted by sinus beats, will also give the appearance of an irregular rhythm.

ANSWER

The correct answer is (d.): Atrial tachycardia. While exit block is a possibility, the pauses should be an exact multiple of the tachycardia cycle length, which is not the case here.[4]

Computer analysis of ECG rhythms

Many modern computerised ECG storage systems offer a diagnosis. Although computerised measurements of the various intervals are reasonably accurate, and conditions such as ST-elevation myocardial infarction are usually correctly recognised, rhythm diagnoses are generally appalling and often dangerously incorrect (personal experience). After over-reading many ECGs generated by the system at Groote Schuur Hospital, we had the computer diagnosis removed from the printout. Unfortunately, this is not the case in many hospitals.

LESSONS AND CONCLUSIONS

- Accurate diagnosis of an arrhythmia is vital to ensure appropriate treatment.
- Never accept the computer-generated diagnosis of an arrhythmia without critically examining the ECG and reaching your own conclusion.
- AF is rare under the age of 60 in the absence of structural heart disease ("lone AF").
- Use all available leads to identify P waves.

REFERENCES

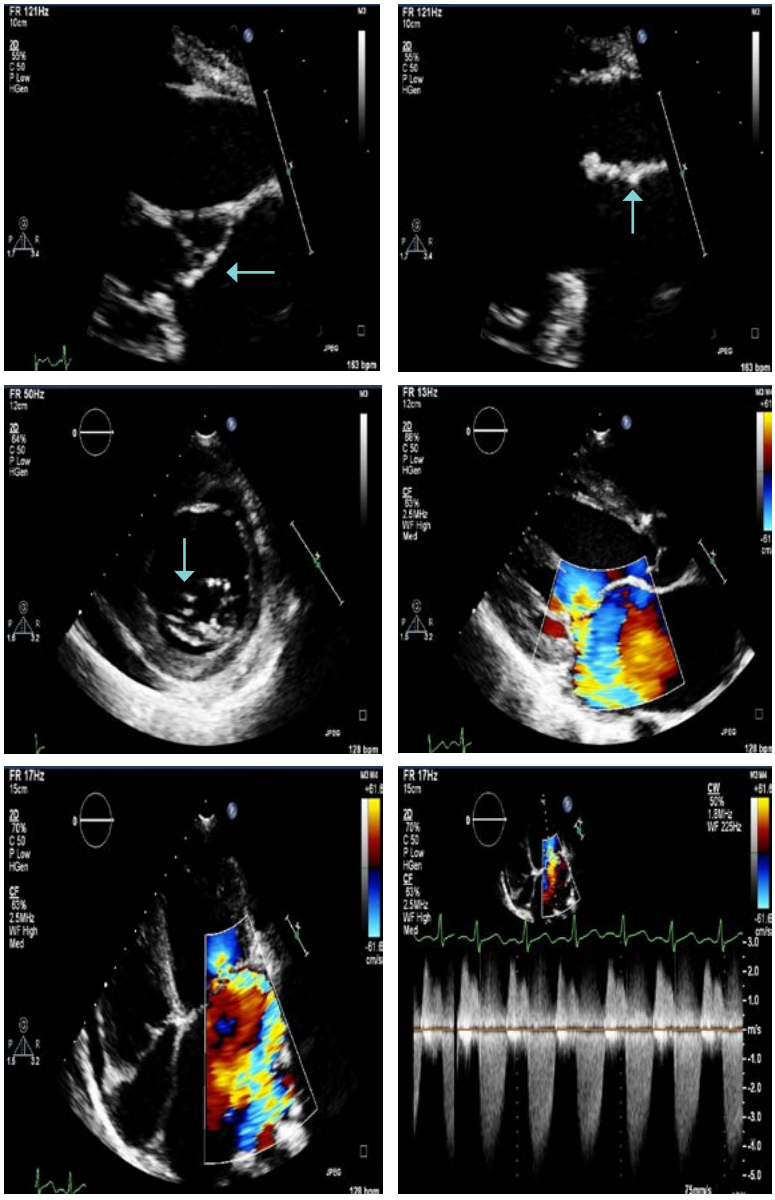
1. Chin A, Vezi B, Namane M, Weich H, Scott-Millar R. An approach to the patient with a suspected tachycardia in the emergency department. S Afr Med J. 2016;106(3):246-50. https://doi.org/10.7196/SAMJ.2016.v106i3.10322.
2. Hoevelmann J, Viljoen C, Chin A. Irregular, narrow-complex tachycardia. Cardiovasc J Afr. 2018;29(3):195-8.
3. Millar RS, Chin A. ECG quiz 37 - Answer. SA Heart J. 2015;12(1);28.
4. Millar RS, Chin A. ECG quiz 70 answers. SA Heart J. 2025;22(4):240-3.

CARDIAC IMAGING QUIZ

R Meel[1] and B Cupido[2]

[1] Faculty of Health Sciences, Department of Internal Medicine, University of the Witwatersrand and Mediclinic Sandton Hospital, Johannesburg, South Africa
[2] Groote Schuur Hospital and University of Cape Town, Cape Town, South Africa

Email: ruchikameel@gmail.com
R Meel https://orcid.org/0000-0002-1405-4259
B Cupido https://orcid.org/0000-0002-6263-7192
DOI: https://doi.org/10.24170/23-4-8589
Creative Commons License - CC BY-NC-ND 4.0



QUESTION: What is the diagnosis?

A. Infective endocarditis.

B. Libman–Sacks endocarditis.

C. Myxomatous degeneration.

D. Rheumatic carditis.

ANSWER

D. Rheumatic carditis.

CASE

A 17-year-old girl presented from a peripheral hospital with heart failure. On examination, she was tachycardic, had a displaced apex beat, and a severe mitral regurgitation holosystolic murmur radiating to the axilla was heard. A transthoracic echocardiogram was performed, which showed features consistent with rheumatic carditis in multiple views. Focal nodular beading on the mitral valve leaflet closure line (arrows in top and middle panels), annular dilation and restricted posterior leaflet motion were present (top and middle panels). Eccentric pathological mitral regurgitation secondary to leaflet malcoaptation extending into the pulmonary veins was noted in 2 valve planes, with a high-velocity jet (4 m/s, holosystolic) (middle and bottom panels). Additionally, an enlarged left atrium, left ventricle, and pericardial effusion were also noted (middle and bottom panels). Acute rheumatic carditis was confirmed by elevated antistreptolysin O titre (ASOT) and antibody to deoxyribonuclease B (anti-DNase B) titres.

DISCUSSION

Rheumatic carditis is a major manifestation of acute rheumatic fever and may present clinically or be detected only on echocardiography (subclinical carditis).[1] Clinically, it is characterised by evidence of cardiac inflammation, most commonly valvulitis causing new mitral regurgitation as in this case, and/or aortic regurgitation murmurs, and may be associated with cardiomegaly, congestive heart failure, or pericarditis with a pericardial rub or effusion. Echocardiography is recommended in all suspected cases because it can identify subclinical carditis, which is now recognised as a major Jones Criteria manifestation.[1,2]

Pathological mitral regurgitation is defined by a regurgitant jet seen in at least 2 views, extending ≥ 2 cm, with a peak velocity > 3 m/s, and a holosystolic pattern. Pathological aortic regurgitation must be seen in at least 2 views, extend ≥ 1 cm, have a peak velocity > 3 m/s, and be holodiastolic. Additional echocardiographic features supporting acute rheumatic valvulitis include mitral or aortic valve leaflet thickening/beading, prolapse or excessive leaflet tip motion, restricted leaflet mobility, chordal elongation, and left-sided chamber dilatation secondary to significant regurgitation.[1] Mitral regurgitation is the most common echocardiographic finding, followed by aortic regurgitation and combined valve involvement.

In acute rheumatic endocarditis, inflammation of the valvular endocardium results in the formation of small, sterile platelet and fibrin thrombi, known as verrucous vegetations (verrucae), along the line of valve closure.[1] These lesions arise from endocardial injury, fibrinoid necrosis, and mechanical stress at the sites where valve leaflets coapt. Echocardiographically, the verrucae appear as multiple small, uniform, echogenic nodules arranged in a characteristic "beaded" or "string of beads" pattern along the leaflet closure line, as in the images above. Unlike the vegetations seen in infective endocarditis, rheumatic verrucae are firmly attached, sterile, rarely embolise, and do not typically cause valve leaflet destruction. Instead, acute rheumatic valvulitis produces valvular regurgitation, most commonly mitral regurgitation, due to leaflet oedema, inflammation, and chordal involvement. Recognising these characteristic morphological features can help differentiate acute rheumatic carditis from other causes of valvular disease.

The main differential diagnoses of rheumatic carditis include infective endocarditis, Libman–Sacks endocarditis, and myxomatous valve degeneration.[1] Infective endocarditis may mimic rheumatic carditis by causing new murmurs and valvular regurgitation; however, it is usually associated with persistent fever, positive blood cultures, evidence of infection, and echocardiographic oscillating masses (vegetations). Libman–Sacks endocarditis, seen in patients with systemic lupus erythematosus or antiphospholipid syndrome, is characterised by sterile vegetations on both sides of the mitral or aortic valve leaflets and is accompanied by other autoimmune manifestations and positive serological markers. Myxomatous degeneration, the pathological basis of mitral valve prolapse, can produce mitral regurgitation and leaflet prolapse on echocardiography, but lacks the inflammatory features, systemic manifestations, and evidence of recent streptococcal infection characteristic of rheumatic carditis. Careful assessment of clinical findings, laboratory markers, and echocardiographic valve morphology helps distinguish these conditions from acute rheumatic carditis.

REFERENCES

1. Gewitz MH, Baltimore RS, Tani LY, et al. Revision of the Jones Criteria for the diagnosis of acute rheumatic fever in the era of Doppler echocardiography: A scientific statement from the American Heart Association. Circulation. 2015;131(20):1806-18. https://doi.org/10.1161/CIR.0000000000000205.
2. Marangou J, Rwebembera J, Mwita J, et al. The echocardiographic diagnosis of rheumatic heart disease: A review of the performance of the World Heart Federation Criteria 2012-2023. Glob Heart. 2024;19(1):47. https://doi.org/10.5334/gh.1327.

SA HEART®

Journal of the South African Heart Association

Recognition by the Department of Education (DoE)

SA Heart® is listed by the Department of Education (DoE) as an Approved Journal since January 2009. This development is important, not only for the stature of the Journal, but also for practical reasons such as the subsidy from the DoE involved for authors affiliated to academic institutions.

International recognition as a National Cardiovascular Journal

SA Heart® is one of an elite group of publications recognised by the European Society of Cardiology (ESC) as a National Cardiovascular Journal.

The invitation to join the ESC Editors' Club highlights the recognition the journal has gained amongst our international peers.

The recognition by the Department of Education and the Academy of Sciences of South Africa (ASSAf) is something we should all take pride in.

Electronic publication

The Journal is published electronically and articles appearing in SA Heart®, both previous and current, are available online at www.saheart.org/journal.

Articles are published in pdf format to facilitate rapid download and easy printing.

SA HEART® IS AN OFFICIAL JOURNAL OF THE SOUTH AFRICAN HEART ASSOCIATION AND APPEARS ON A QUARTERLY BASIS

Instructions for authors

SA Heart® publishes peer reviewed articles dealing with cardiovascular disease, including original research, topical reviews, state-of-the-art papers and viewpoints. Regular features include an ECG quiz, image in cardiology and local guidelines. Case reports are considered for publication only if the case or cases are truly unique, incorporates a relevant review of the literature and makes a contribution to improved future patient management. All articles will have a DOI number.

Publication policy

Articles must be the original, unpublished work of the stated authors. Written permission from the author or copyright holder must be submitted with previously published material including text, figures or tables. Articles under consideration elsewhere or previously published (except as abstracts not exceeding 400 words) may not be submitted for publication in SA Heart®. On acceptance transfer of copyright to the South African Heart Association will be required. No material published in SA Heart® may be reproduced without written permission. Permission may be sought from the Editor (Email: ruchikameel@gmail.com).

Disclosures

Authors must declare all financial disclosures and conflicts of interest in the cover letter and on the title page of the manuscript.

Ethics

All studies must be in compliance with institutional and international regulations for human and animal studies such as the Helsinki declaration (2008) (https://www.wma.net/policies-post/wma-declaration-of-helsinki-ethical-principles-for-medical-research-involving-human-subjects) and the South African MRC ethics guidelines (https://www.samrc.ac.za/research/ethics/guideline-documents). Human studies require ethics committee approval and informed consent which must be documented in your manuscript. Animal studies require ethics committee approval and must conform to international guidelines for animal research. Compliance with these requirements must be documented in your manuscript.

Content

1. Title page: It should contain the title of the manuscript, the names of all authors in the correct sequence, their academic status and affiliations. If there are more than 4 authors, the contribution of each must be substantiated in the cover sheet. The main author should include his/her name, address, phone, fax and email address. All authors must supply their ORCiD ID upon submission of the manuscript.
2. Authors are solely responsible for the factual accuracy of their work.
3. Articles should be between 3 000 and 5 000 words in length.
4. A 200-word abstract should state the main conclusions and clinical relevance of the article.
5. All articles are to be in English.
6. Abbreviations and acronyms should be defined on first use and kept to a minimum.
7. Tables should carry Roman numeral, I, II etc., and figures Arabic numbers 1, 2 etc.
8. References should be numbered consecutively in the order that they are first mentioned in the text and listed at the end in numerical order of appearance. Identify references in the text by Arabic numerals in superscript after punctuation, e.g. …trial.[13]

The following format should be used for references:

Articles

Kaplan FS, August CS, Dalinka MK. Bone densitometry observation of osteoporosis in response to bone marrow transplantation. Clin Orthop 1993;294:73-8. (If there are more than six authors, list only the first three followed by et al.).

Chapter in a book

Young W. Neurophysiology of spinal cord injury. In: Errico TJ, Bauer RD, Waugh T (eds). Spinal Trauma. Philadelphia: JB Lippincott; 1991:377-94.

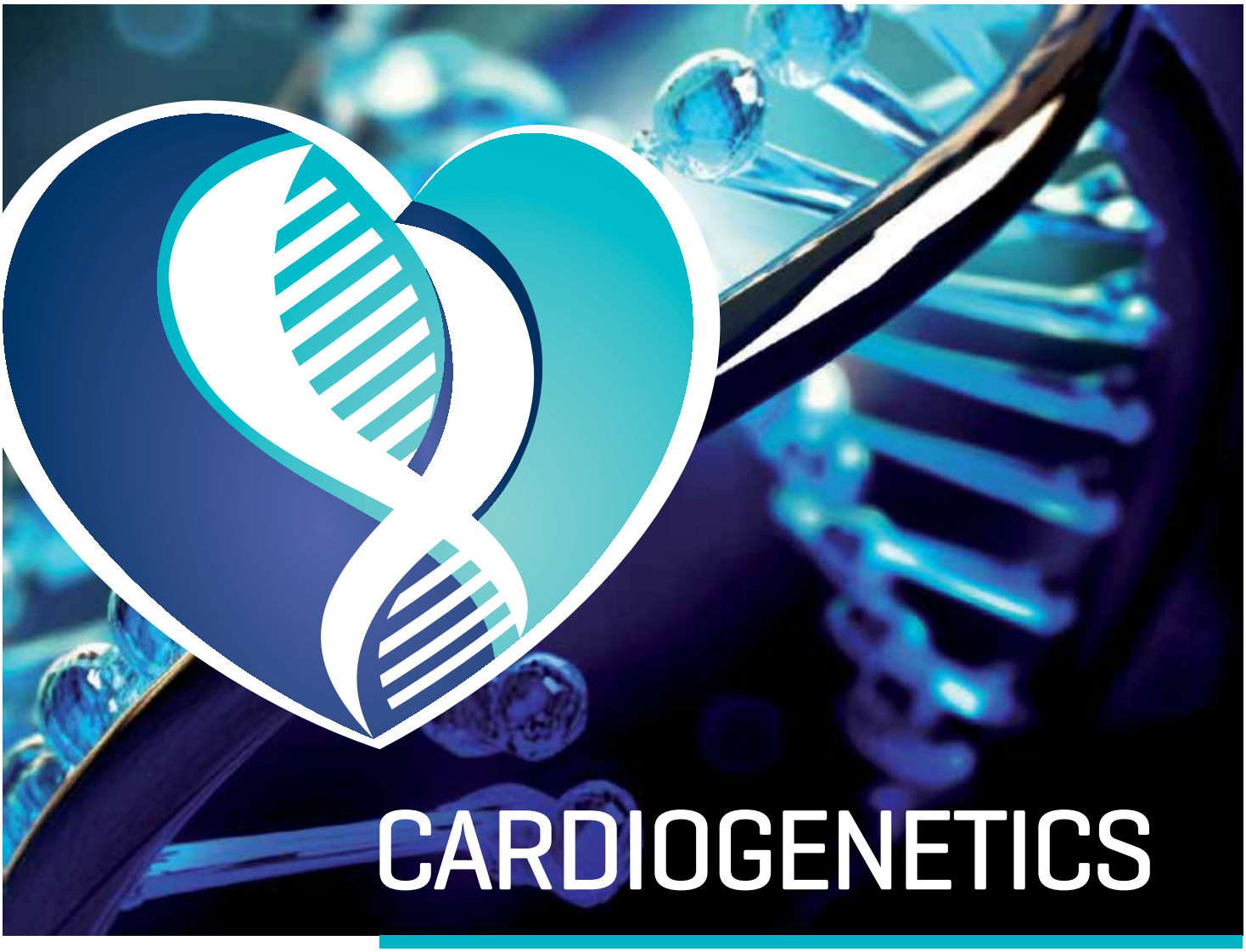
Online media

Perreault, L. (2019). Obesity in adults: Role of physical activity and exercise. UpToDate. Retrieved January 12, 2020, from https://www.uptodate.com/contents/obesity-in-adults-role-of-physical-activity-and-exercise

9. Articles are to be submitted on the online SA Heart® platform https://tinyurl.com/y9prlopt. The text should be in MS Word. Pages should be numbered consecutively in the following order wherever possible: Title page, abstract, introduction, materials and methods, results, discussion, acknowledgements, tables and illustrations, references.
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.



CARDIOGENETICS

FROM DIAGNOSIS TO PRECISION CARE

27th Annual SA Heart Congress 2027

Gauteng Province,
South Africa
5 - 7 November
2027