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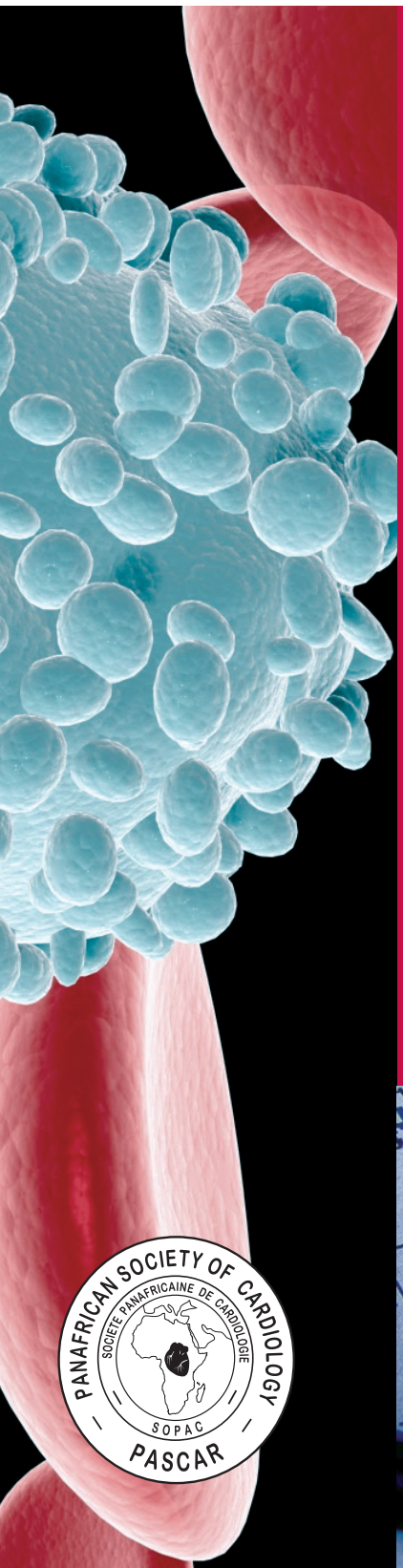

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PRODUCTION EDITOR
SHAUNA GERMISHUIZEN
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e-mail: shauna@clinicscardive.com

CONTENT MANAGER
MICHAEL MEADON (Design Connection)
Tel: 021 976 8129
Fax: 0866 557 149
e-mail: michael@clinicscardive.com

GAUTENG CONTRIBUTOR
PETER WAGENAAR
Cell 082 413 9954
e-mail: skylark65@myconnection.co.za

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Tel: 021 976 8129
Fax: 0866 644 202
Int.: +27 21 976 8129

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From the Editor's Desk

Food and the eating thereof is a universal part of the human condition, and life is obviously impossible without adequate nutrition. The fact that the composition of diets varies so dramatically across the globe and that human populations survive and thrive fairly successfully despite seemingly very different nutrient intakes provides food for thought! Either the exact composition of the diet is irrelevant or else humans are biologically remarkably adaptable to great variation in the composition of their diet. If either of the statements in the previous sentence is correct then those individuals in what I refer to as the 'diet industry', an industry with enormous media appeal and reward, should feel seriously threatened.

In light of the dramatic advances in the pharmacological and interventional management of patients with cardiovascular diseases, it is unfortunate that apparently conflicting advice on such a simple matter as diet is offered to patients and those at risk of disease. Such conflicting advice surely confuses rather than educates those at whom it is aimed. Lifestyle and diet are the topics reviewed by Opie (page 298) in an in-depth and scholarly review, which addresses in a balanced manner many of the current controversies. The accompanying editorial from Raal (page 302) is further valuable commentary.

Awad and colleagues (page 269) report on the high prevalence rates of hypertension in the Gambia and Sierra Leone, as has been previously reported from other parts of Africa. The South African hypertension practice guideline prepared by the Hypertension Guideline Working Group of the Southern African Hypertension Society is published on page 288. It is comprehensive and includes information on lifestyle modification and education, in addition to detailed advice on pharmacotherapy. An accompanying comment (page 296) addresses the value and importance of such guidelines in clinical practice. It would be interesting to hear comment from colleagues in other parts of Africa as to the applicability and relevance of these guidelines to practice in their own countries.

Otaigbe and colleagues (page 265) report on the prevalence of congenital heart disease, detected by echocardiography, among children referred to two specialist paediatric cardiology clinics in the Niger Delta region of Nigeria. Such information adds to our increasing knowledge of patterns of cardiovascular disease in Africa. It must be borne in mind however that this is not a population-based study, referral bias remains possible, and the authors' attribution of the high prevalence to environmental pollution is speculative.

Despite the success of percutaneous interventions, coronary artery bypass grafting is still a very common operation and the impact of interventions and risk factors for complications continues to be investigated. Cingoz and colleagues (page 279) examine the impact of co-morbidity on bleeding after the operation, while Yildiz and co-workers (page 259) examine the value of patient-directed education on patient anxiety after surgery. The importance of the anxiety experienced by patients and families of hospital survivors of major cardiac surgery is often underestimated by healthcare professionals.

PJ Commerford
Editor-in-Chief

The management and staff of Clinics Cardive Publishing wish you and your family a wonderful holiday season and a healthy and peaceful 2015. May your holidays be filled with joy, peace and good cheer!

We take this opportunity to thank you for your loyal support during 2014 and we look forward to being of service in 2015.

Editorial

Towards developing guidelines and systems of care to facilitate early reperfusion for ST-elevation myocardial infarction in Africa

Rhena Delport

Outline

The theme for the 15th annual SA Heart Congress for 2014, 'Bridging the divide' between best practice and current challenges in the management of cardiovascular conditions, inspired this editorial on the feasibility of implementing the European Society of Cardiology 'Stent-for-Life' initiative in sub-Saharan Africa or alternate measures of ensuring early reperfusion for myocardial ischaemia. This editorial explores the changing burden of non-communicable diseases (NCD) in Africa that impact on the occurrence of ST-elevation myocardial infarction (STEMI) in Africa, revisits international guidelines on early reperfusion and implementation of systems of care, and identifies factors related to timely myocardial reperfusion in remote areas.

Current status in Africa

Recent comments by Kengne and Mayosi on the rising incidence of chronic NCD in sub-Saharan Africa in both rural and urban areas express concern about the lack of preparedness of African states for the pending pressure on healthcare services,[1] pertain to South African healthcare services as well.[2,3] The World Health Organisation (WHO) estimates that NCDs will exceed communicable diseases as the leading cause of death in Africa in 2030.[4,5]

An increase in cardiovascular disease (CVD) burden is also expected due to the increased prevalence and incidence of CVD risk factors, paucity of surveillance data and registries, lack of interventional measures, as well as a shortage of physicians and cardiologists, inadequate diagnostic capabilities, and misguided opinions.[6-8] Although CVD remains the leading cause of death in the world,[9] three-quarters of which occur in low- and middle-income populations,[6] the burden of ischaemic heart disease (IHD) remains low in comparison with other causes of heart disease, particularly in people of African descent.[6,10,11] Marked variability is however observed in the incidence, prevalence and mortality rates of IHD across developing countries, as in Africa, mainly due to the differences in composition and severity of risk factors and management thereof, as well as the stage of epidemiological transition.[6,11-20]

Department of Chemical Pathology, University of Pretoria, Pretoria, South Africa
Rhena Delport, PhD, rhena.delport@up.ac.za

Concerted action among the WHO and international cardiac societies to improve cardiovascular health and prevent death from cardiovascular disease is increasingly becoming evident.[21,22] Hopefully African societies will follow suit.

Current guidelines

The majority of recommendations in the European[23] and American[24] guidelines for the management of STEMI were perceived as either identical or overlapping.[25] The detail of the guidelines will not be replicated here, neither is the aim of this editorial to perform further comparisons with other international guidelines. A brief exposition on primary reperfusion strategies will be provided from random sources, with the emphasis on the African context where percutaneous coronary intervention (PCI) facilities are sparsely distributed and emergency medical services (EMS) are not readily available.

Primary percutaneous coronary intervention is the preferred and most effective option for reperfusion, provided that the intervention is performed timely by an experienced operator.[26,27] Although performance metrics such as 'door-to-balloon time' or 'door-to-needle time' are employed to quantify time lapses from the onset of symptoms to definitive treatment, the concept of 'first medical contact (FMC)-to-device time' recognises the need for speedy diagnosis and treatment as the primary outcome.[26]

The patient as well as factors relating to EMS determine the time delay between the onset of symptoms and the FMC, while FMC and the beginning of reperfusion is explained by EMS transport time to a PCI-capable facility and determinants of 'door-to-balloon' time.[27] Ideally the patient should be transported directly to a PCI-capable hospital for primary PCI but if the patient is admitted to a non-PCI facility, the door-in-door-out time should ideally be 30 minutes or less before the patient is transported to a PCI-capable hospital.[26] The FMC-to-device time should be 90 minutes or less, and in the case of necessity to transfer the patient for PCI, 120 minutes or less. If primary PCI is not achievable within 120 minutes thrombolytics should be administered with FMC within 30 minutes of diagnosis of STEMI either pre-hospital by a trained paramedic/clinic nurse, or, alternatively, by a physician in the nearest ER.[26,27]

Additional recommendations of relevance as proposed in the United Kingdom 'NICE' guidelines[28] entail the following (as quoted):

- *Offer coronary angiography, with follow-on PPCI if indicated, as the preferred coronary reperfusion strategy for people with*

acute STEMI if:
  - *Presentation is within 12 h of onset of symptoms and*
  - *PPCI can be delivered within 120 min of the time when fibrinolysis could have been given.*
- *Offer fibrinolysis to people with acute STEMI presenting within 12 h of onset of symptoms if PPCI cannot be delivered within 120 min of the time when fibrinolysis could have been given.*
- *Consider coronary angiography, with follow-on PPCI if indicated, for people with acute STEMI presenting more than 12 h after the onset of symptoms if there is evidence of continuing myocardial ischaemia.*
- *Offer coronary angiography, with follow-on PPCI if indicated, to people with acute STEMI and cardiogenic shock who present within 12 h of the onset of symptoms of STEMI.*
- *Offer an ECG to people treated with fibrinolysis, 60–90 min after administration. For those who have residual ST-segment elevation suggesting failed coronary reperfusion:*
  - *Offer immediate coronary angiography, with follow-on PCI if indicated*
  - *Do not repeat fibrinolytic therapy.*
- *If a person has recurrent myocardial ischaemia after fibrinolysis, seek immediate specialist cardiological advice and, if appropriate, offer coronary angiography, with follow-on PCI if indicated.*
- *When commissioning PPCI services for people with acute STEMI, be aware that outcomes are strongly related to how quickly PPCI is delivered, and that they can be influenced by the number of procedures carried out by the PPCI centre.*

Factors that may contribute to earlier treatment for PCI-treated patients include bypassing non-PCI-capable hospitals and bypassing the emergency department of the PCI-capable hospital, pre-hospital ECG diagnosis of STEMI, and pre-hospital activation of the catheterisation laboratory by emergency physician or EMS, and early (within 20 minutes) activation of the catheterisation laboratory team.[27,28]

Current guidelines for remote areas

For the treatment of STEMI patients living in remote, sparsely populated areas with no ready access to PCI facilities, the pharmaco-invasive strategy is advocated. Fibrinolysis should be commenced as soon as possible if there are no contra-indications, followed by transfer to a PCI facility for rescue PCI or angiography with possible PCI as a routine measure. Patients with contra-indications for fibrinolysis, late presenters, and patients with cardiogenic shock should be transferred to a PCI facility irrespective of the duration of transfer. Clear treatment protocols and a well-organised STEMI network are pivotal in STEMI management in these areas.[29]

From the Australian experience, we learn that direct transport to PCI facilities and inter-hospital transfer for primary PCI positively impact on timely access to primary PCI (defined as 'the proportion of the population capable of reaching a PPCI facility ≤ 120 minutes from emergency medical services activation') and that pre-hospital fibrinolysis significantly improves timely access to reperfusion PCI (defined as 'the proportion of the population capable of reaching a fibrinolysis facility in ≤ 60 minutes from emergency medical services activation') in remote areas.[29] Geographical information systems were employed to integrate hospital, classified as hospitals that provided PCI or fibrinolysis, and population and road network data,[30] which in all probability contributes to informed management of STEMI care.

Concluding remark

In our endeavour to facilitate early reperfusion for ST elevation myocardial infarction in Africa we need to bear in mind that '*Improvements in access to timely care for patients with STEMI will require a multifaceted approach involving patient education, improvements in the emergency medical services and emergency department components of care, the establishment of networks of STEMI-referral hospitals (not PCI capable) and STEMI-receiving hospitals (PCI capable), as well as coordinated advocacy efforts to work with payers and policy makers to implement a much-needed healthcare system redesign. By focusing now on system efforts for improvements in timely care for STEMI, we will complete the cycle of research initiated by Reimer and Jennings 30 years ago. Time is muscle . . . we must translate that into practice*' (Elliott M Antman, 2008).[31]

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Cardiovascular Topics

Effect of standard versus patient-targeted in-patient education on patients' anxiety about self-care after discharge from cardiovascular surgery clinics

Tülin Yıldız, Selami Gürkan, Özcan Gür, Cüneyt Ünsal, Sonay Baltacı Göktaş, Yücel Özen

Abstract

We compared standard and patient-targeted in-patient education in terms of their effect on patients' anxiety. One hundred and ninety-eight patients who were hospitalised for coronary artery bypass surgery were given standard education (group 1) or individualised education (group 2) on the management of their healthcare after discharge. Patients in group 2 were assessed on the patient learning needs scale and were given education according to their individual needs. The level of anxiety was measured by the state–trait anxiety inventory. Anxiety scores were significantly lower in group 2 than group 1 after education ($p < 0.001$). While state anxiety did not change after education in group 1 ($p = 0272$), it decreased significantly in group 2 ($p < 0.001$). For cardiovascular surgery patients, patient-targeted in-patient education was more effective than standard education in decreasing anxiety levels, therefore the content of the education should be individualised according to the patient's particular needs.

Keywords: cardiovascular surgery, anxiety, patient's education, coronary artery bypass surgery

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Department of Surgical Nursing, School of Health, Namık Kemal University, Tekirdağ, Turkey
Tülin Yıldız, MsN, PhD, tyildiz70@hotmail.com
Selami Gürkan, MD
Özcan Gür, MD

Department of Psychiatry, Faculty of Medicine, School of Health, Namık Kemal University, Tekirdağ, Turkey
Cüneyt Ünsal, MD

Department of Surgical Nursing, School of Nursing, Maltepe University, Istanbul, Turkey
Sonay Baltacı Göktaş, MsN, PhD

Department of Cardiovascular Surgery, Kartal Koşuyolu Education and Research Hospital, Istanbul, Turkey
Yücel Özen, MD

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Coronary artery bypass surgery is the absolute treatment for severe coronary artery disease, which is the leading cause of mortality globally.[1] The surgery relieves angina symptoms, improves quality of life, and reduces cardiac-related mortality.[2] To achive the desired benefits of coronary artery bypass surgery, it is important to decrease the risk factors for atherosclerosis and change the lifestyle of patients. For this reason, clinical practice guidelines published by several organisations have recognised the importance of patient self-care measures, and agree that patient education is an important element in the care of patients for whom cardiac surgery is planned.[3-5]

To this end, pre- and postoperative in-patient and discharge education have been shown to demonstrate benefits for patients in cardiovascular surgery clinics.[6,7] Comprehensive in-patient education before discharge improved health outcomes such as survival and quality of life.[8]

For coronary artery bypass surgery, the duration of hospitalisation of patients ranges from four to seven days. During the hospital stay, patients usually feel anxious about surgery and about finding solutions to likely problems they may encounter after discharge. Lack of knowledge and skills necessary for home care and for the new lifestyle after discharge may slow the healing process by causing physical and psychological stress on the patient.[9,10] In-patient and discharge education aims to give patients the necessary information and to decrease their anxiety levels.

Although specific elements of information and standards of patient education have been defined,[11] there are no guidelines or standards for the education of cardiovascular surgery patients for discharge. Cardiovascular surgery clinics usually create patient education content, which focuses on postoperative care, diet, rest and exercise, and implement it via nurses who play a key role in education of hospitalised patients.[12] Furthermore, although the advantages of in-patient education on anxiety, depression and post-discharge health outcomes were described for cardiovascular surgery patients,[13,14] the effectiveness of standardised educational tools in comparison with individualised education has not been studied.

We hypothesised that in-patient education individualised according to the patient's needs would be more targeted and effective than standardised education for patients hospitalised for coronary artery bypass surgery. On the basis of this hypothesis,

we aimed in this study to compare standard and patient-targeted in-patient education in terms of their effect on patients' anxiety about self-care after discharge.

Methods

One hundred and ninety-eight patients who were hospitalised in the cardiovascular surgery clinic between February and August 2013 for coronary artery bypass surgery were included in the study. The study was approved by the institutional ethics committee, and all patients gave consent to participate in the study.

Patients were randomised into two groups based on the content of education: standard education (group 1, $n = 100$) and individualised education (group 2, $n = 100$) on the management of patients' healthcare at home after discharge. Patients in group 2 were assessed with the patient learning needs scale (PLNS) to define their perception of learning needs to manage their healthcare after discharge. These patients were given education that was specified according to their individual needs.

Education was given at the same time daily from the first day of hospitalisation until the day the patient was discharged from the clinic. The duration of hospitalisation was four to five days for all patients.

The level of anxiety of patients was measured by the state–trait anxiety inventory (STAI) before education and on discharge. The STAI scores were compared between the groups and before and after education. Additionally, the effects of socio-demographic variables on the change of anxiety scores in each group were evaluated.

The education was given by one investigator to all patients in both groups. The STAIs were given to patients by another investigator who was blinded to the patients' study groups. Only patients' initials (not full names) and a code for education group were marked on the STAIs; thus the data entry and analysis were blinded to the study groups.

The education and outcomes were evaluated during the patients' hospitalisation, in which time no serious complications were recorded.

The education given to in-patients by nurses in cardiovascular surgery clinics aims to help patients to meet their home-care needs before and after surgery, to facilitate getting help from the healthcare team, to accelerate the healing process, and to ensure the transition to a normal life as soon as possible. On the basis of these aims, a standard education that was developed by Ozcan *et al.*,[15] which includes topics on drug use, coping with pain, surgical wound care, prevention of adverse effects, diet, exercise, rest, hygiene maintenance, constipation, alcohol and tobacco use, sexual activity, mood changes, emergencies, occupations and time of control visits was given face to face to group 1 patients for about 30 to 40 minutes daily. During this time, topics were repeated as necessary, taking into account individual differences. An education booklet including details on all topics was used during the education sessions. Patients in group 2 were given education that was individualised according to their specific need, which were determined by the PLNS.

Study questionnaires

The PLNS was developed to measure patients' learning needs in order to manage their healthcare at home after discharge from hospital.[16] It is completed in less than 20 minutes. It has 50 items scored from 0 to 5, and seven subscales (medication, activities of living, feelings related to condition, community and follow up, treatment and complications, enhancing quality of life, and skin care), yielding a total score of 40 to 200, with higher scores indicating more importance being placed on having information at discharge. The reliability and validity of the PLNS were shown by Bubela *et al.*,[17] and the Turkish version was also shown to be reliable and valid.[18]

The STAI is a commonly used self-report measure of anxiety, which is a four-point Likert scale and consists of 40 questions.[19] The STAI measures two types of anxiety: state anxiety, or anxiety about an event, and trait anxiety, or anxiety level as a personal characteristic. Higher scores are positively correlated with higher levels of anxiety. It can be used to diagnose anxiety and distinguish it from depressive syndromes, and also as an indicator of caregiver distress. Its reliability and validity were demonstrated.[19,20] It is offered in 12 languages, including Turkish.

Statistical analysis

Descriptive statistical methods (frequency, percentage, mean, standard deviation) were used to summarise data, and the Kolmogorov–Smirnov test was used to determine whether data were distributed normally or not. For the comparison of quantitative data between groups, the independent samples *t*-test and Mann–Whitney *U*-test were applied for parameters with and without normal distribution, respectively.

To compare quantitative data of more than two groups with normal distribution, Kruskal–Wallis and Mann–Whitney *U*-tests were used. For in-group comparisons the paired-sample *t*-test was used. Pearson's correlation analysis was performed to define the correlation between quantitative variables, and the outcome was expressed as correlation coefficient (r) and level of significance (p).

The level of statistical significance was set at $p < 0.05$. Statistical analyses were performed using computer software (Statistical Package for Social Sciences, Version 19.0, SPSS Inc, Chicago, Illinois, USA).

Results

The mean ages of the patients in group 1 ($n = 98$) and group 2 ($n = 100$) were 62.1 ± 10.2 and 59.1 ± 9.8 years, respectively, and the number of male/female patients were 73/25 and 72/28, respectively. Other socio-demographic and clinical characteristics of the study patients are summarised in Table 1.

The study groups were homogeneous in terms of gender, marital status, having children, working status, smoking, alcohol consumption, and being on a diet ($p > 0.05$). There was no significant difference between group 1 and group 2 in terms of age, weight, education, income and frequency of exercise ($p < 0.05$) (Table 1).

The STAI scores showed no statistical difference between the study groups before education ($p = 0.168$ and $p = 0.583$, respectively). However, both anxiety scores were significantly lower in group 2 than in group 1 after education ($p < 0.001$ for STAI scores). Furthermore, while state anxiety did not change in group 1 after education ($p = 0272$), it decreased significantly in

Table 1. Socio-demographic characteristics of the study patients

	Group 1 (standard education) (n = 98)	*Group 2 (individualised education) (n = 100)*	p-*value*
Gender			
Male	73 (75)	72 (72)	0.407
Female	25 (26)	28 (28)	
Age (years)	62.1 ± 10.2	59.1 ± 9.8	*0.038*
Weight (kg)	75.6 ± 11.2	80.3 ± 13.5	*0.008*
Marital status			
Married	92 (94)	94 (94)	0.602
Single	6 (6)	6 (6)	
Having children			
Yes	96 (98)	94 (94)	0.146
No	2 (2)	6 (6)	
Education			
Primary school	78 (80)	68 (68)	*0.025*
Middle school	10 (10)	13 (13)	
High school	7 (7)	6 (6)	
University	3 (3)	3 (3)	
Illiterate	0 (0)	10 (10)	
Working status			
Working	30 (31)	30 (30)	0.524
Not working	68 (69)	70 (70)	
Income ($*/TL# per month)	624.0 ± 418.6	406.7 ± 202.3	*< 0.001*
Smoking			
Yes	27 (28)	31 (31)	0.353
No	71 (72)	69 (69)	
Alcohol consumption			
Yes	12 (12)	18 (18)	0.176
No	86 (88)	82 (82)	
Exercise			
Yes	18 (18)	48 (48)	< 0.001
No	80 (82)	52 (52)	
Frequency of exercise			
None	56 (57)	52 (52)	0.002
3–4 times/week	12 (12)	9 (9)	
Daily	10 (10)	30 (30)	
1–2 times/week	20 (20)	9 (9)	
On a diet			
Yes	24 (25)	36 (36)	0.054
No	74 (76)	64 (64)	

Data are given as *n* (%) or mean ± standard deviation.
*$, US Dollar; #TL, Turkish Lira (the exchange rate was 1 TL = $1.9961).

Table 2. State anxiety scores from the STAI

	Group 1 (standard education) (n = 98)	*Group 2 (individualised education) (n = 100)*	t-*value*	p-*value*
Before education	54.34 ± 5.06	55.23 ± 3.94	1.38	0.168
After education	54.96 ± 4.47	26.93 ± 2.56	–54.01	< 0.001
t-value	0.50	65.77		
p-value	0.275	< 0.001		

Table 3. Trait anxiety scores from the STAI

	Group 1 (standard education) (n = 98)	*Group 2 (individualised education) (n = 100)*	t-*value*	p-*value*
Before education	47.36 ± 6.71	46.91 ± 4.48	–0.55	0.583
After education	43.41 ± 5.79	34.45 ± 4.83	–11.82	< 0.001
t-value	4.71	33.83		
p-value	< 0.001	< 0.001		

group 2 ($p < 0.001$). On the other hand, trait anxiety decreased significantly with education in both groups ($p < 0.001$ for both groups) (Tables 2, 3).

Socio-demographic variables had a limited effect on the change in STAI scores with education in both study groups (Table 4). In the standard-education group (group 1), none of the socio-demographic variables had an effect on the education-induced change in state and trait anxiety scores, except gender; in male patients, a larger change in trait anxiety was found with education ($p = 0.017$).

In the individualised-education group (group 2), only working status had a significant effect on the education-induced change in state anxiety ($p = 0.029$). Marital status ($p = 0.017$) and exercise ($p = 0.048$) had significant effects on the education-induced change in trait anxiety.

In both study groups, there was no significant correlation between education-induced change in state or trait anxiety scores and age, weight or income of patients, except a slight significant negative correlation between change in trait anxiety score and weight of patients in group 1 ($r = –0.257$; $p = 0.011$). Accordingly, as weight increased, the education-induced reduction of trait anxiety decreased in patients receiving standard education (Table 5).

Discussion

In this prospective, hospital-based, blind-analysis study, we found that in-patient education was effective in decreasing anxiety levels of patients who were hospitalised in a cardiovascular clinic for coronary artery bypass surgery. More remarkably, our findings showed that in-patient education targeted to the patient's particular needs provided more benefit than standard education in decreasing anxiety of patients about self-care after discharge.

Patients hospitalised for coronary artery bypass surgery in cardiovascular surgery clinics are usually under psychological pressure about the surgery and their new life after discharge. This pressure is greater if they are not aware of and ready for the problems that may develop during home care after discharge. Studies have shown that patients for whom cardiac surgery is planned, want to know about their disease and its treatment, complications and measures that should be taken, and lifestyle after surgery.[9] Goodman[10] evaluated what information and support patients feel they need in the six-week rehabilitation period following discharge after cardiac surgery, and pointed to the need for improvements in the psychological preparation of patients for discharge after cardiac surgery.

In this study, therefore, we focused on the effect of in-patient education on patients' anxiety levels. We used the STAI, a well-established anxiety tool, to determine their anxiety about the period after discharge (presented as state anxiety) and general level of anxiety (presented as trait anxiety). Our study population had high levels of both state and trait anxiety on the first day of hospitalisation before in-patient education.

Table 4. Effect of socio-demographic variables on the change in STAI anxiety scores with education

	Group 1 (standard education, n = 98)				*Group 2 (individualised education, n = 100)*			
	Change in state anxiety with education	p-*value*	*Change in trait anxiety with education*	p-*value*	*Change in state anxiety with education*	p-*value*	*Change in trait anxiety with education*	p-*value*
Gender								
Male	–0.58 ± 5.51	0.922	2.80 ± 7.78	*0.017*	28.42 ± 4.50	0.513	12.33 ± 3.72	0.410
Female	–0.76 ± 6.02		7.32 ± 8.99		28.00 ± 3.80		12.79 ± 3.64	
Marital status								
Married	–0.47 ± 5.51	0.265	3.78 ± 8.02	0.750	28.44 ± 4.21	0.427	12.67 ± 3.65	*0.017*
Single	–3.00 ± 7.18		6.50 ± 12.49		26.17 ± 5.57		9.17 ± 2.56	
Having children								
Yes	–0.72 ± 5.59	0.314	3.88 ± 8.06	0.860	28.13 ± 4.30	0.101	12.45 ± 3.71	0.821
No	4.00 ± 7.07		7.50 ± 21.92		31.00 ± 3.69		12.67 ± 3.56	
Education								
Primary school	–0.50 ± 5.80	0.965	4.49 ± 7.85	0.403	28.10 ± 4.13	0.895	12.00 ± 3.67	0.131
Middle school	–0.90 ± 2.77		5.40 ± 6.92		29.54 ± 5.08		14.31 ± 4.55	
High school	–1.00 ± 6.86		–1.00 ± 11.26		29.50 ± 5.72		12.67±2.16	
University	–2.00 ± 7.21		–3.33 ± 13.65		27.00 ± 3.61		14.00 ± 1.00	
Illiterate					27.70 ± 4.08		12.60 ± 3.37	
Working status								
Working	–2.27 ± 4.86	0.056	2.07 ± 9.27	0.331	29.77 ± 4.03	*0.029*	12.93 ± 3.50	0.339
Not working	0.10 ± 5.80		4.78 ± 7.76		27.67 ± 4.29		12.26 ± 3.76	
Smoking								
Yes	–0.56 ± 4.73	0.936	4.19 ± 9.10	0.790	28.74 ± 4.09	0.555	13.03 ± 4.07	0.248
No	–0.65 ± 5.95		3.86 ± 8.04		28.10 ± 4.41		12.20 ± 3.50	
Alcohol consumption								
Yes	–1.08 ± 3.83	0.761	2.92 ± 8.95	0.774	28.56 ± 3.99	0.850	12.33 ± 3.53	0.850
No	–0.56 ± 5.83		4.09 ± 8.25		28.24 ± 4.39		12.49 ± 3.74	
Exercise								
Yes	–2.00 ± 5.96	0.150	2.17 ± 7.82	0.142	28.50 ± 4.32	0.618	13.29 ± 3.97	*0.048*
No	–0.31 ± 5.53		4.35 ± 8.40		28.12 ± 4.32		11.69 ± 3.25	
Frequency of exercise								
None	–0.23 ± 5.07	0.552	3.95 ± 7.78	0.996	28.12 ± 4.32	0.393	11.69 ± 3.25	0.248
3–4 times/week	–0.50 ± 5.25		4.42 ± 8.59		28.22 ± 3.35		13.78 ± 5.52	
Daily	–3.00 ± 7.94		2.60 ± 11.09		27.83 ± 4.36		13.23 ± 3.70	
1–2 times/week	–0.60 ± 6.10		4.35 ± 8.61		31.00 ± 4.58		13.00 ± 3.46	
On a diet								
Yes	–0.50 ± 5.36	0.944	3.33 ± 6.88	0.459	28.67 ± 4.42	0.650	13.25 ± 4.02	0.172
No	–0.66 ± 5.73		4.15 ± 8.74		28.09 ± 4.26		12.02 ± 3.43	

It has been established in many studies that in-patient and discharge education is essential and beneficial to decrease anxiety levels and depression, hospital re-admissions, non-adherence to medication, and to improve quality of life, survival, and to enable patients to retain a sense of control in their lives.[7,8,13] Education at the time of hospital discharge provides improved clinical outcomes, increased adherence to self-care measures, and reduced cost of care in patients with cardiac disease.[6]

Table 5. Correlation (*r*, correlation coefficient) between change in anxiety score from the STAI and age, weight and income level

		Group 1 (standard education, n = *98)*		*Group 2 (individualised education,* n = *100)*	
		Change in state anxiety with education	*Change in trait anxiety with education*	*Change in state anxiety with education*	*Change in trait anxiety with education*
Age (years)	*r*	–0.072	–0.073	–0.172	–0.020
	p	0.479	0.473	0.088	0.845
Weight (kg)	*r*	0.024	–0.257	–0.133	–0.057
	p	0.818	*0.011*	0.188	0.575
Income	*r*	–0.093	0.127	–0.051	0.091
	p	0.360	0.211	0.611	0.370

Therefore, patient education by nurses during the hospitalisation period is suggested in the common guidelines as part of the routine pharmacological and surgical treatment of patients with cardiovascular diseases.[12] The Joint Commission on Accreditation of Healthcare Organisation defined the standards of patient education by nurses.[11] However, there are currently no guidelines or standards on how to educate cardiovascular surgery patients for discharge with regard to the amount or content of the information necessary to be effective. Therefore we aimed to compare the effectiveness of standard and patient-targeted in-patient education in decreasing patients' anxiety levels in the cardiovascular clinic.

To measure patients' learning needs to manage their healthcare at home after discharge from hospital, we assessed them using the PLNS.[17,18] We modified the standard education

for each individual patient according to the results of the PLNS, and applied this modified and individualised education to the patients in group 2.

We showed that state anxiety decreased significantly only after patient-targeted education but not after standard education given during the hospitalisation period. On the other hand, trait anxiety, which represents the personal anxiety level of the subject independent of the event, decreased in both groups, being significantly lower on discharge in the individualised-education group than in the standard-education group. Therefore, patient-individualised education was more effective than standard education in decreasing both state and trait anxiety levels in cardiovascular surgery patients.

Additionally, we evaluated the effect of socio-demographic factors, and found that these variables had a limited effect on the change in STAI scores. This suggests that the change in anxiety was mainly attributable to the effect of in-patient education.

Our findings are in line with previous studies reporting the advantage and necessity of in-patient education for patients hospitalised for cardiovascular surgery,[7,8,13] but we are the first to demonstrate the superiority of patient-targeted education above standard education in decreasing anxiety levels of patients. Since present evidence in the literature suggests that the psychological stability of patients is associated with better physiological parameters after the procedure and early surgical recovery,[21-23] we propose that decreased anxiety provided by patient-targeted education is related to better clinical outcomes after cardiovascular surgery. However, the effect of decreased anxiety levels produced by patient-targeted education on morbidity and mortality rates after cardiovascular surgery should be evaluated in further clinical studies.

Individual instruction of patients is important to accelerate healing after surgery and to enable the patient's return to social and business life in the shortest possible time. In this context, although gender, marital status, having children, working status, smoking, alcohol consumption, being on diet, and hospitalisation duration showed similarities between the study groups, decreased anxiety levels in patients who received patient-targeted in-patient education resulted in increased levels of self-care and self-confidence.

The main limitations of this study are that we did not follow up on the patients after discharge, and did not evaluate the outcomes of standard versus patient-targeted in-patient education on patients' work and health outcomes after discharge. Nevertheless, it is the first study comparing patient-targeted and standard in-patient education in a cardiovascular surgery clinic, where patients have high levels of anxiety about the period after discharge. The advantage of patient-targeted education over standard education in lowering patients' anxiety levels may decrease patients' physical and psychological stress levels and therefore provide better outcomes after surgery.

Conclusion

Education of cardiovascular surgery patients during hospitalisation about subsequent home care and the new lifestyle after discharge is necessary to decrease patients' anxiety levels, and should be implemented as part of the surgical and pharmacological treatment of patients. Since patient-targeted in-patient education was more effective than standard education in decreasing patients' anxiety, the content of the education should be individualised according to the patient's particular needs. In this way, in-patient education will be more beneficial in decreasing anxiety levels of patients and provide more effective use of resources. Studies on the effect of patient-targeted in-patient education on post-discharge health outcomes are needed to test the further advantages of this type of education.

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AfricaPCR 2015: practical solutions for African problems

The second AfricaPCR course will be taking place in Johannesburg from 26–28 March 2015 at the forum, the campus in Bryanston. The 2015 course will build on the objective of exploring clinical challenges specific to Africa, and forging solutions through a highly interactive process of sharing, learning, questioning, and challenging by the facilitators.

The highlights of the 2015 programme will be: STEMI – exploring solutions to problems defined in 2014; setting up a cathlab service in Africa; and managing patients with limitated resources. There will be a focus on the local environment and the particular circumstances of each African region.

An exciting session on high-risk patients will cover the ideal preparation for procedures on these patients and how to prevent and avoid complications. Atrial septal defect, left atrial appendage closure and left main intervention will be featured in the African context, and the complex issue of peri-procedural antithrombotic pharmacology will be distilled into a rational, practical approach. Parallel sessions will include an interactive case corner as well as a dedicated programme for allied health professionals.

Exhibitors of allied drugs and medical equipment will be sponsoring industry-supported sessions with supporting case studies. Interactive product demonstrations will also be held for delegates in the form of hands-on workshops.

For a detailed programme and to register, visit www.africapcr.com, email: info@eoafrica.co.za or contact Europa Organisation Africa, tel: (011) 325-0020.

Congenital heart disease in the Niger Delta region of Nigeria: a four-year prospective echocardiographic analysis

BE Otaigbe, PN Tabansi

Abstract

Introduction: Echocardiographic evaluation remains the gold standard for the diagnosis of structural cardiac disease. No previous prospective studies have been done on the prevalence of congenital heart disease (CHD) in the Niger Delta area. This study was done to determine the frequency and pattern of congenital heart disease, using echocardiography as a diagnostic tool.

Methods: All patients presenting to the Paediatric Cardiology clinics of two centres, the University of Port Harcourt Teaching Hospital and the Paediatric Care Hospital between April 2009 and March 2013, were recruited and all had echocardiography performed.

Results: Prevalence of CHD in this study was 14.4 per 1 000 children; 277 (83.4%) of the patients had acyanotic CHD and 55 (16.6%) had cyanotic CHD. Ventricular septal defect and tetralogy of Fallot were the commonest acyanotic and cyanotic heart defects, respectively.

Conclusion: The high prevalence of CHD in this study is the highest in the country and Africa, and may be attributable to the increased oil spillage and gas flaring from petroleum exploitation in this region.

Keywords: congenital heart disease, high prevalence, Niger Delta, oil spillage

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Congenital heart disease (CHD) affects approximately eight per 1 000 live births in the general population, making it one of the most common classes of birth defect.[1] CHD is defined as an abnormality in the cardio-circulatory structure or function, which is present at birth, even if it is discovered later.[2]

More children die from CHD each year than are diagnosed with cancer.[3] A large number of these children are in the developing countries. They are often repeatedly admitted and treated for recurrent chest infections and failure to thrive due to ignorance of the attending health caregiver, poor diagnostic tools, poor referral systems, and lack of skilled personnel. These lead to late diagnosis and increased mortality rates.

Department of Paediatrics, University of Port Harcourt Teaching Hospital, Port Harcourt Rivers State, Nigeria
BE Otaigbe, FWACP (Paed), BM BCh, barbiejoe64@yahoo.com
PN Tabansi, BM BCh, FWACP (Paed)

CHD can be life threatening in early childhood, and children born with severe forms are at approximately 12 times higher risk of mortality in the first year of life, especially if they are missed in the neonatal period.[4] About two to three in 1 000 newborns with heart disease will be symptomatic in the first year of life, diagnosis is established by one week in 40–50%, and by one month of age in 50–60% of patients.[5]

The incidence of CHD in Nigeria 30 years ago was 3.5 per 1 000 population.[6] This has increased in recent studies to 4.6/1 000 in the southern,[7] and 9.3/1 000 in the northern[8] parts of Nigeria, apparently due to increased diagnostic facilities and more trained paediatric cardiologists in the country.

Echocardiographic evaluation remains the gold standard for the diagnosis of structural cardiac disease.[7] Paediatric echocardiography has not been widely available in Nigeria as there are few paediatric cardiologists and most of the available echocardiography machines have no paediatric probes. Prior to the procurement of an echocardiography machine with paediatric probes in the reporting echocardiographic centre, history, clinical diagnosis and chest radiography were used as tools for provisional diagnosis of CHD.[9]

There has been no previous report on the incidence of CHD in the Niger Delta region of Nigeria. Previous studies on CHD from Nigeria have been retrospective studies and none from the Niger Delta region of Nigeria, where there have been claims of increasing risks to maternal and child health due to environmental degradation and industrial pollution secondary to petroleum mining and gas flaring in this region. This study was done to determine the frequency and pattern of CHD, using echocardiography as a diagnostic tool.

Methods

This was a prospective study of all patients presenting to the paediatric cardiology clinics of two centres, the University of Port Harcourt Teaching Hospital and the Paediatric Care Hospital, having been referred to the clinics or seen in the wards between April 2009 and March 2013. All the patients enrolled were fully examined by at least one of the two paediatric cardiologists and further evaluated with chest radiographs, electrocardiograms (ECG) and an echocardiogram (echo).

The chest radiographs were read separately by the cardiologist and radiologists and conflicting reports were discussed. The ECG was performed by a technician using a Schiller AT-1 Smart Print machine, standardised at a paper speed of 25 mm/s. Echo diagnosis of all patients was done using Sonosite Micromaxx and Sonosite Edge machines, available only at the Paediatric Care Hospital. Each patient was recruited with a proforma, which contained records of name, age, gender, weight, indication for echocardiography, chest X-ray and ECG findings,

and echocardiographic parameters, diagnosis, comments and outcome.

Statistical analysis

Data were computed and analysed using Epi version 6.02. For the purpose of this report, only the echo findings were included in the analysis.

Results

During the period of four years, 23 124 children presented at the Paediatric Department of the two centres. Of these, 440 children were referred for echocardiographic evaluation. Only 356 (81%) had echo done, out of whom 24 were found to have structurally normal hearts. Therefore 332 patients with cardiac anomalies were analysed. Prevalence of CHD in this hospital-based study was 14.4 per 1 000 children.

Of those analysed, there were 174 males (52.4%) and 158 females (47.6%) in a ratio of 1:1. The ages ranged from 0.25 to 180 months with a mean of 26.1 months. Thirty (9%) of the children were aged one month or less, 62% were between one month and one year, 24.4% between one and five years, and 13.7% above five years of age.

The commonest indications for an echo were murmur in 36% (of which 6% were incidental murmurs), fast breathing in 19.8%, failure to thrive in 11% and cyanosis in 9.9%. Others included features of dysmorphism and easy fatigability (Table 1). Thirty-six (11%) of the patients had features suggestive of Down syndrome (DS), while one patient had dysmorphism suggestive of William's syndrome.[9] Of the 36 patients with DS, over 60% had multiple cardiac defects, with ventricular septal defects (VSD)/patent ductus arteriosus (PDA) and VSD/atrial septal defect (ASD) accounting for 50%. Nine patients had congenital *Rubella* syndrome, with PDA accounting for over 50% of the cardiac defects.

Two hundred and seventy-seven (83.4%) of the patients had acyanotic CHD and 55 (16.6%) had cyanotic CHD. In the acyanotic group, there were 149 males (53.8) and 128 females (46.2%), with no statistically significant difference, while there were 25 males (45.5%) and 30 females (54.5%) in the cyanotic group.

Table 1. Indications for echocardiography

Sign/symptom	*Number of patients*	*% of patients*
Fast breathing	64	19.2
Failure to thrive	36	10.8
Murmur	83	25
Dysmorphism	36	10.8
Cyanosis	32	9.6
Recurrent pneumonia	13	3.9
Recurrent cough	16	4.8
Easy fatigability	23	6.9
Chest pain	6	1.8
Cardiomegaly on chest X-ray	6	1.8
Palpitation	5	1.5
Fainting attacks	6	1.8
Heart failure	21	6.33
Pre-term low birth weight	7	2.1

Table 2. Solitary acyanotic CHD types and frequency of occurrence

Type of acyanotic CHD	*No of patients*	*% of acyanotic CHD (n = 277)*	*% of total CHD (n = 332)*
Ventricular septal defect	90	32.5	27.1
Patent ductus arteriosus	48	17.3	14.5
Atrial septal defect	23	8.3	2.5
Pulmonary stenosis	3	1.1	0.9
Atrio-ventricular canal defect	8	2.9	2.4
Congenital dilated cardiomyopathy	2	3.6	0.6
Congenital hypertrophic cardiomyopathy	2	3.6	0.6

In the acyanotic group, solitary VSDs accounted for 32.5% of cases, solitary PDAs for 17.3% and solitary atrial septal defect (ASD) for 8.3%. Atrio-ventricular canal defect (AVCD) was seen in eight (2.9%) (Table 2). Congenital dilated cardiomyopathy was seen in two patients, one of whom had lost two older siblings in infancy with similar conditions. Of the two patients with hypertrophic cardiomyopathy, one was a macrosomic infant of a diabetic mother, who had spontaneous resolution of the hypertrophy by the fourth month. The other was an infant of a non-diabetic mother, who was managed in a peripheral hospital and died after two months from recurrent heart failure.

In the cyanotic group, transposition of the great arteries (TGA) was seen in 12 patients, and tetralogy of Fallot (TOF) in 28 patients, two of whom had associated ASD (pentalogy of Fallot). Truncus arteriosus was seen in three patients (Table 3). Table 4 shows the number of children with multiple congenital heart diseases.

Of the children studied, 24 (7.2%) have had successful surgeries in India, one in Ghana, one in South Africa and three in the United State of America. Two children with atrio-ventricular septal defect and pulmonary hypertension, who had Eisenmenger syndrome, went to India and were confirmed inoperable after cardiac cathetherisation.

One hundred and thirty-three (40%) of these children have had at least one hospital admission, and 10 (3%) have died. Four died in hospital while the other six were confirmed dead by telephone calls from parents. Of those who died in hospital, two had tetralogy of Fallot with cerebrovascular accident and died after partial exchange of blood for packed cell volume of 88 and 76%, respectively. The other two died of intractable heart failure.

Table 3. Cyanotic CHD types and frequency of occurrence

Type of cyanotic CHD	*No of patients*	*% of cyanotic CHD (n = 55)*	*% of total CHD (n = 332)*
Tetralogy of Fallot	28	50.9	8.4
Transposition of the great arteries	12	21.9	3.6
Tricuspid atresia	3	5.5	0.9
Truncus arteriosus	3	5.5	0.9
Double-outlet right ventricle	3	5.5	0.9
Ebstein's anomaly	3	5.5	0.9
Hypoplastic left heart syndrome	2	3.6	0.6
Partial anomalous pulmonary venous connection	1	1.8	0.3

Table 4. Multiple CHD and frequency of occurrence

Type of CHD	*Number*	*% acyanotic CHD (n = 277)*	*% total CHD (n = 332)*
VSD/PDA	53	19.1	16
VSD/ASD	6	2.2	1.8
ASD/PDA	16	5.8	4.8
ASD/VSD/PDA	12	4.3	3.6
ASD/PAPVC	2	0.7	0.6
AVCD/PDA	2	0.7	0.6

VSD, ventricular septal defect; PDA, patent ductus arteriosus; ASD, atrial septal defect; PAPVC, partial anomalous pulmonary venous connection; AVCD, atrio-ventricular canal defect.

Discussion

The prevalence of CHD in this prospective, hospital-based study of 14.4 per 1 000 is alarmingly higher than studies from other parts of Nigeria, where it was 9.3,[8] and 4.6/1 000,[7] Egypt was 1.01,[10] India was 10.5,[11] the United State of America was 6.5,[12] Norway was 10.6,[13] and Austria was 6.9/1 000.[14] It is comparable with other studies from Qatar where prevalence was 12.25,[15] and Australia was 17.5/1 000.[16]

This very high figure is most likely due to environmental factors. Port Harcourt is in Rivers state in the Niger Delta region, an oil-rich city in the south–south geographical zone of Nigeria where crude oil exploration is rampant, and oil spillage from petroleum exploration commonly affects water quality and terrestrial fauna. Gas flaring constitutes a toxic threat to inhabitants of these areas. Heavy hydrocarbons that cannot be carried into the atmosphere fall back and become inhaled, while others get attached to vegetables grown for consumption. Over time, this may be toxic to the body or cause congenital malformations in babies born in the area.[17] Toxic agents may induce malformation in the foetus during the early weeks of organogenesis.

As with other studies done in Nigeria and other parts of the world, VSD was the commonest acyanotic CHD seen in this study, with a frequency of almost half of the acyanotic CHD (47. 3%); 39.5% of the CHD we saw was similar to that seen in Saudi Arabia,[18] Mysore,[12] and Qatar.[15] The prevalence of VSD was higher than previously reported in studies in Port Harcourt Teaching Hospital, which was 34.1%,[9] 30.3% in Kano,[8] 32.3% in the UK,[19] 32.1% in the USA,[13] and 35.6% in Egypt,[10] but less than the 55.3% reported in Benin.[7]

TOF was the commonest cyanotic CHD, similar to studies in Nigeria,[7-9] and worldwide.[10-16] That TGA ranks second may be due to early mortality of the children. The two cases of dextrocardia had rare presentations of situs solitus with no structural heart defect,[20] dextrocardia, and situs inversus with multiple CHD.[21]

Despite murmurs being the commonest indication for requesting an echo, there are still many patients presenting late to hospital, despite having been seen by numerous doctors managing them for recurrent bronchopneumonia and tuberculosis. This is due to the inability of attending junior medical staff to identify a murmur on auscultation. This is further highlighted in this study with only 9% of the patients reporting to hospital within the first month of life.

Increased risks of structural birth defects and chromosomal abnormalities have been reported to be due to air pollution and proximity to environmental waste. The findings of a large number of multiple congenital heart defects in these children is worrisome and may be related to the teratogenic effect of gas and oil spillage in the Niger Delta.

In this study, although no direct efforts were made to get information about the place of residence of the parents, a cursory review of the addresses showed that 28 (8.4%) of the parents lived close to areas where gas is flared, nine (0.23%) near telecommunication masts and 12 (0.04%) of the mothers, when asked what routine antenatal drugs were ingested, had mentioned a drug called Pregnacare. This contains multivitamins as supplements, including omega-3, folic acid, iron and vitamin B_{12}.

These incidental findings have prompted an ongoing study emphasing description of location and review of drugs ingested by mothers of all children presenting with congenital heart disease in our centres. Hypervitaminosis, potentially teratogenic fumes and ionising radiation are being postulated as contributing factors to this high prevalence of CHD.

It may be worth mentioning that six of the infants with acyanotic heart disease were products of *in vitro* fertilisation (IVF). All six had multiple CHD and were all of multiple gestation. One patient, one of a set of triplets, had hypoplastic left heart syndrome (HLHS) and died within one week of diagnosis, at two weeks of life. The other siblings had structurally normal hearts. Alhough not statistically proven in this study as no comparison was made with children of parents who conceived naturally, the high risk of CHD in IVF patients has been previously documented.[22,23] This has also prompted an ongoing prospective research on cardiac anomalies among patients delivered by IVF in the Niger Delta area.

These results may be the tip of the iceberg, as few of these cardiac patients present to hospital for diagnosis and fewer still can afford the cost of an echocardiograph. Most of the patients are still being managed by pharmacists and herbalists, mostly due to poverty, ignorance and poor access to proper medical care.

Conclusion

The high prevalence rate of CHD reported in this study area, which has many environmental risks for CHD, is worrying. With the increasing availability of echocardiography and paediatric cardiologists in the region, and increased awareness, more cases are likely to be detected in future. There is an urgent need to assess and confirm the impact and causal relationship of oil spillage, gas flaring, use of the drug Pregnacare, and residence close to telecommunication masts in the Niger Delta, to make a genuine case for the prevention and reduction in the prevalence of CHD in this region. We emphasise the need to regulate the deleterious activities of oil companies in the Niger Delta, and establish cardiac centres in our country for cheaper and more easily available diagnostic tools and early surgery to improve outcomes.

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Erratum

Infliximab, an anti-TNF-alpha agent, improves left atrial abnormalities in patients with rheumatoid arthritis: preliminary results

Çetin Süha, Mustafa Gökhan, Keskin Göksal, Yeter Ekrem, Doğan Mehmet, Öztürk Mehmet Akif

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The authors' names should have been:
Süha Çetin, Gökhan Mustafa, Göksal Keskin, Ekrem Yeter, Mehmet Doğan, Mehmet Akif Öztürk

We regret the error.

Prevalence of hypertension in the Gambia and Sierra Leone, western Africa: a cross-sectional study

Morcos Awad, Andrea Ruzza, James Mirocha, Saman Setareh-Shenas, J Robert Pixton, Camelia Soliman, Lawrence SC Czer

Abstract

Background: Hypertension (HTN) is one of the causes of cardiovascular disease (CVD) in Africa, and may be associated with lower socio-economic status (SES). The prevalence of HTN is not well established in the Gambia or in Sierra Leone.

Methods: A cross-sectional, population-based study of adults was conducted in the Gambia in 2000 and in Sierra Leone from 2001 to 2003 and in 2009. The study was conducted as part of the annual visit to countries in western Africa sponsored by a medical delegation from California. People from the Gambia and Sierra Leone were examined by the medical delegation and blood pressures were measured.

Results: A total of 2 615 adults were examined: 1 400 females and 1 215 males. The mean systolic blood pressure (SBP) of the females was 134.3 ± 29.7 mmHg, mean diastolic blood pressure (DBP) was 84.5 ± 17.5 mmHg, and 46.2% were hypertensive. The mean SBP of the males was 132.8 ± 28.5 mmHg, mean DBP was 82.8 ± 16.2 mmHg, and 43.2% were hypertensive. Overall prevalence of HTN in the subjects was 44.8%. Mean SBP, mean DBP and HTN prevalence increased with age decade, both in males and females. In addition, after age adjustment (known age), females had higher mean SBP ($p = 0.042$), mean DBP ($p = 0.001$) and rate of occurrence of HTN ($p = 0.016$) when compared with males.

Conclusions: Prevalence rates of HTN in the Gambia and Sierra Leone were higher than 40% in males and females, and may be a major contributor to CVD in both countries. Due to the association of HTN with low SES, improvements in educational, public health, economic, non-governmental and governmental efforts in the Gambia and Sierra Leone may lead to a lower prevalence of HTN. The cause of the higher prevalence in women may be due to post-menopausal hormonal changes.

Keywords: hypertension, the Gambia, Sierra Leone, prevalence, sodium, age, gender

Division of Cardiology, Cedars-Sinai Heart Institute, Los Angeles, California
Morcos Awad, BS
Saman Setareh-Shenas, MS
J Robert Pixton, BS, Camelia Soliman, BS
Lawrence SC Czer, MD, lawrence.czer@cshs.org

Division of Cardiothoracic Surgery, Cedars-Sinai Heart Institute, Los Angeles, California
Andrea Ruzza, MD, PhD

Section of Biostatistics, Cedars-Sinai Medical Center, Los Angeles, California
James Mirocha, MS

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Hypertension (HTN) is a chronic, slowly progressive disease affecting about one billion people globally and leading to about 7.1 million deaths annually. People of African origin may be particularly susceptible to hypertension.[1-3] Defined as a sustained systolic blood pressure (SBP) above 140 mmHg, a diastolic blood pressure (DBP) above 90 mmHg or both, the aetiology of HTN can be classified as primary or secondary. While there is no known cause for primary (essential) HTN, which accounts for 90–95% of cases, the remaining 5–10% of cases is defined as secondary HTN and is caused by other disease conditions, which may affect the renal, circulatory, endocrine or other organ systems.

Many factors are associated with, and may contribute to the development and persistence of primary HTN, including obesity, stress, smoking,[4] low potassium intake, high sodium (salt) and alcohol intake,[5,6] familial and genetic influences,[7,8] and low birth weight.[9] On the other hand, hyperthyroidism, hypothyroidism and other conditions causing hormonal changes may be associated with primary pulmonary HTN.[10,11] Regardless of the cause, the consequences of HTN include renal failure, heart failure, myocardial infarction, pulmonary oedema and stroke.[12]

Given these undesirable outcomes, treatment and prevention have assumed increasing emphasis in the management of HTN. Modification of risk factors can be achieved by reducing body weight and decreasing sugar intake, along with lowering alcohol consumption,[13,14] as well as reducing salt intake and increasing potassium intake.[15,16] Secondary HTN is managed by treating the underlying cause. Drugs available for the treatment of HTN, whether primary or secondary, include calcium-channel blockers (CCB), angiotensin converting enzyme inhibitors (ACEI), angiotensin receptor blockers (ARB), diuretics, α-blockers and β-blockers.

Race and ethnicity may influence pathogenesis, prevalence and treatment of HTN,[17] perhaps through genetic influences. As a consequence, HTN remains one of the most common CVDs in Africa and one of the most frequent causes of death in the sub-Saharan African region.[18,19] In 2000, the rate of HTN in sub-Saharan Africa was reported to be 26.9% in males and 28.3% in females.[20] Low socio-economic status (SES) may additionally play an important role in the high prevalence of HTN in western and sub-Saharan Africa.

A cross-sectional survey in Tanzania revealed that treatment rates for HTN were very low, especially among people with low SES.[21] Low SES led to inadequate education levels as a factor

correlating with a higher blood pressure (BP) in adults and resulted in a low treatment rate for HTN due to monetary issues.[22]

Stress, in addition, was another factor related to HTN prevalence, especially in Africa.[23] It has been shown that psychosocial stress affects the L-arginine/nitric oxide (NO) system, with a higher susceptibility in black Africans, which in turn contributes to a higher risk of CVD in those individuals.[24]

Therefore, a multiplicity of factors may be associated with and contributing to a high prevalence of HTN among Africans. The current study was undertaken to determine and quantitate the prevalence of HTN in two countries in western sub-Saharan Africa, namely, the Gambia and Sierra Leone.

Methods

This was a population-based, cross-sectional study performed in the Gambia and Sierra Leone. The data were collected from the Gambia in 2000 and from Sierra Leone from 2001 to 2003 and in 2009. The Gambia is a small country, about 11 000 km^2 in 2007, with a population of 1 705 000 by 2009.[25,26] Sierra Leone is a larger country, about 72 000 km^2 in 2007, with a population of 5 696 000 by 2009.[25,26]

This study took place as part of the annual visit to countries in western Africa sponsored by a medical delegation from California. In the Gambia, the visit was to specific areas within the capital city of Banjul, including Serrekunda, Latrikunda and Fajikunda. In Sierra Leone, the medical delegation visited Freetown, Kenema, Lunsar, Bonthe, Bo, Jui and Makeni.

People waited in queues to be examined in a clinic by the team.[27] Subjects underwent a history and general physical examination, had their blood pressure checked, and were given medications depending on the health issues they discussed with the healthcare providers. The current study focused on the BP readings collected for adults aged ≥ 18 years.

People coming for general examinations stayed in a waiting area in front of the clinic to be triaged by a nurse before being checked by a physician. BPs were measured using a sphygmomanometer. Patients whose BP fell in the hypertensive range (SBP ≥ 140 mmHg, or DBP ≥ 90 mmHg) had their BP measured again once or twice by the physician, depending on the initial BP. If more than one BP was recorded, an average value was determined.

In the Gambia and Sierra Leone, one of the additional procedures performed was echocardiography using a hand-carried ultrasound (HCU) to assess left ventricular hypertrophy (LVH) to prioritise HTN treatment.[27] LVH was previously found in 65% of people with HTN.[27]

Statistical analysis

All the data collected during these visits, including BP measurements, medications prescribed, and diagnostic tests, were recorded on a paper form and were later entered in a computerised data spreadsheet and then de-identified. The study was reviewed and certified by the institutional review board (IRB).

Data were analysed statistically using the χ^2-test, and the p-values calculated were classified based on $p < 0.05$ as considered of statistical significance. Other statistical tests included the Fisher's exact test, Cochran–Armitage trend test, Wilcoxon rank sum test, Student's t-test and ANCOVA multivariable-model test. The data were analysed by country prior to and following the combination of both data sets.

Data from Sierra Leone were available for the years 2001–2003 and 2009. Differences in SBP and DBP means were assessed across the years by analysis of covariance (ANCOVA) models. The preliminary model was a two-way full factorial model with factors gender and year and the gender-by-year interaction, and age was the covariate.

In the SBP model, the gender-by-year interaction term was significant ($p = 0.011$), so separate one-way ANCOVA models were assessed in females and males, with age as the covariate. In the DBP model, the gender-by-year interaction term was not significant ($p = 0.17$); however, for comparison, separate one-way ANCOVA models were assessed in females and males, with age as the covariate. The least-squares means (LSmeans) for SBP and DBP were used to present the findings.

The data were divided into three categories: all adults with and without known recorded age ($n = 2\,615$), only adults with known age ≥ 18 years old ($n = 2\,348$) and only adults with known age ≥ 20 years old ($n = 2\,247$). There was one female who did not have a recorded DBP.

The first classification was used to have general demographics for the whole population tested. The second and third classifications were used to observe trends of SBP, DBP and HTN prevalence with age decade, starting with 20-year-old patients. For all results including age decade analyses, the indications ≥ 70s and +70s stand for the age decade 70 years and above, which were combined together with patients over 80 years due to the small sample size in these older groups.

Results

In total, there were 2 615 adult participants: 46.5% males ($n = 1\,215$) and 53.5% females ($n = 1\,400$). Because one female lacked a recorded DBP, the total number of individuals analysed based on SBP, DBP and HTN prevalence were 2 615, 2 614 and 2 614 individuals, respectively.

Of the overall population studied, 44.8% were hypertensive, while mean SBP was 133.6 ± 29.2 mmHg and mean DBP was 83.7 ± 17.0 mmHg. For females, mean SBP was 134.3 ± 29.7 mmHg and mean DBP was 84.5 ± 17.5 mmHg, while 46.2% were hypertensive. For males, mean SBP was 132.8 ± 28.5 mmHg and mean DBP was 82.8 ± 16.2 mmHg, while 43.2% were hypertensive.

The t-test showed no significant difference in mean SBP between males and females ($p = 0.18$). However, for mean DBP, the t-test indicated a significant difference between males and females ($p = 0.008$), with females having a higher mean DBP. Regarding HTN prevalence, the χ^2-test showed that there was no significant difference between males and females, and the Fisher's exact test confirmed this insignificance ($p = 0.119$ and $p = 0.124$, respectively).

From the total number of subjects in the study ($n = 2\,615$), a large proportion ($n = 2\,348$) represented individuals with known age ≥ 18 years old. The demographics of this subpopulation (Table 1) were compared across gender in terms of age, SBP and DBP means using the t-test.

For mean age, males were older on average ($p = 0.018$). For mean SBP, there was no evidence that SBP differed across gender; 133.5 mmHg for females and 132.8 mmHg for males (p

Table 1. Characteristics of patients with known age ≥ 18 years

Variable	*Overall (n = 2 347)*	*F (n = 1 236**)*	*M (n = 1 111)*	p-*value unadjusted*	p-*value adjusted*
Age (years)	39.6 ± 16.1	38.9 ± 15.9	40.5 ± 16.4	0.018*	
SBP (mmHg)	133.2 ± 28.5	133.5 ± 28.6	132.8 ± 28.5	0.57*	0.042*
DBP (mmHg)	83.3 ± 16.7	84.0 ± 17.1	82.6 ± 16.1	0.049*	0.001*
HTN (%)	44.5	45.6	43.3	0.26†	0.016††

Values: mean ± SD or %.
**Females: n = 1 237 for SBP and age, and n = 1 236 for DBP and HTN.
p-values for M vs F: *Student's t-test, †χ^2-test, ††multivariable model (odds ratio = 1.25). Adjustment: for age.
F = females, M = males, SD = standard deviation, SBP = systolic blood pressure, DBP = diastolic blood pressure, HTN = hypertension.

Table 2. Characteristics of patients with known age ≥ 20 years

Age decade (years)	*N*	*Gender*	*n*	*SBP ± SD (mmHg)*	*DBP ± SD (mmHg)*	*HTN % (n/N)*	*HTN Overall % (n/N)*
20s	694	F	386	119.3 ± 19.1	76.0 ± 14.0	21.8 (84/386)	22.3 (155/694)
		M	308	118.6 ± 16.9	74.9 ± 11.7	23.1 (71/308)†	
30s	531	F	284**	125.4 ± 23.1	80.2 ± 15.4	33.8 (96/284)	33.9 (180/531)
		M	247	124.7 ± 21.0	79.2 ± 13.5	34.0 (84/247)†	
40s	373	F	190	143.0 ± 27.6	92.6 ± 16.8	66.3 (126/190)	58.4 (218/373)
		M	183	136.3 ± 29.1	85.9 ± 16.8	50.3 (92/183)*	
50s	312	F	159	151.3 ± 30.5	92.7 ± 16.3	71.7 (114/159)	69.9 (218/312)
		M	153	150.1 ± 32.1	91.2 ± 15.3	68.0 (104/153)†	
60s	201	F	96	160.8 ± 25.5	95.7 ± 13.1	86.5 (83/96)	80.1 (161/201)
		M	105	155.9 ± 30.8	92.6 ± 16.8	74.3 (78/105)*	
≥ 70s	135	F	65	158.0 ± 30.5	93.4 ± 17.9	81.5 (53/65)	75.6 (102/135)
		M	70	153.3 ± 27.5	91.7 ± 14.8	70.0 (49/70)†	

Values: mean ± SD or % (n/N).
**Females: n = 285 for SBP and age, and n = 284 for DBP and HTN.
Fisher's exact test: *significant differences, and †insignificant differences.
F = females, M = males, SD = standard deviation, SBP = systolic blood pressure, DBP = diastolic blood pressure, HTN = hypertension.

= 0.57). However, after age adjustment, females seemed to have a significantly higher SBP compared to males; 134.1 mmHg for females and 132.1 mmHg for males (p = 0.042).

In the case of mean DBP, there was a small difference across gender; 84.0 mmHg for females and 82.6 mmHg for males (p = 0.049). After age adjustment, there was a more significant evidence of the difference in DBP; 84.3 mmHg for females and 82.2 mmHg for males (p = 0.001).

For HTN, the χ^2-test showed no difference across gender (p = 0.26). However, after age adjustment using the multivariable model, it seemed that females had higher odds and hence risk of HTN than males (odds ratio = 1.25, p = 0.016).

SBP, DBP and HTN trends

From the total number of subjects with known age in the study (n = 2 348), a subdivision of this population (n = 2 247) represented individuals with known age ≥ 20 years old. This subpopulation was used to examine the SBP, DBP and HTN prevalence trends with age decade (Table 2).

Mean SBP increased continually with age decade for males and females (Fig. 1). The rate of increase was similar between the genders; the slopes of the regression lines for males and females were 8.036 and 8.806, respectively. As in the case of SBP, mean DBP increased continually with age decade for males and females (Fig. 2). The rate of increase was very similar between the genders; the slopes of the regression lines for males and females were 3.696 and 3.824, respectively.

The Cochran–Armitage trend test showed significant differences in the HTN prevalence between each age decade, overall and gender-wise (p < 0.0001). This meant that within males, females, or overall scores, there was evidence that HTN prevalence increased with age decade (Fig. 3). Meanwhile, female HTN prevalence appeared to be higher than that of males in the age decades 40s, 50s, 60s, and +70s; however, the Fisher's exact test showed evidence for the difference only in the age decades

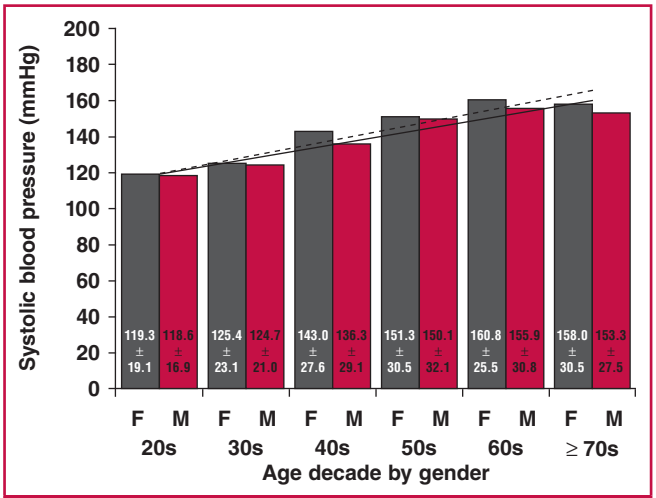

Fig. 1. Mean SBP of patients with known age ≥ 20 years. Histogram of mean SBP ± SD. Females = grey bars and dashed line, males = red bars and solid line. Regression equations: $y = 8.806x + 112.1$ ($R = 0.956$) for females and $y = 8.036x + 111.6$ ($R = 0.954$) for males. F = females, M = males, SD = standard deviation, SBP = systolic blood pressure.

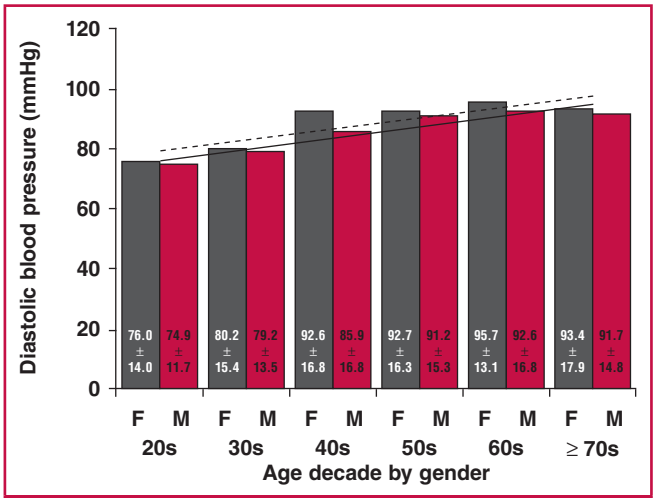

Fig. 2. Mean DBP of patients with known age ≥ 20 years. Histogram of mean DBP ± SD. Females = grey bars and dashed line, males = red bars and solid line. Regression equations: $y = 3.824x + 75.03$ ($R = 0.872$) for females and $y = 3.696x + 72.97$ ($R = 0.938$) for males. F = females, M = males, SD = standard deviation, DBP = diastolic blood pressure.

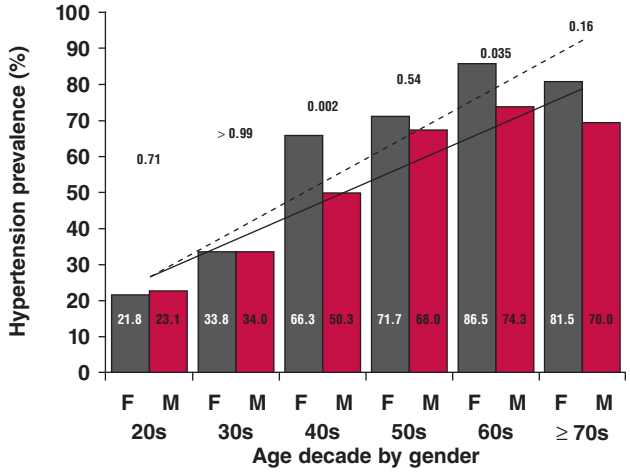

Fig. 3. HTN prevalence in patients with known age ≥ 20 years. Histogram of HTN prevalence rates (%). Females = grey bars and dashed line, males = red bars and solid line. Regression equations: $y = 13.2x + 14.06$ ($R = 0.935$) for females and $y = 10.66x + 15.97$ ($R = 0.944$) for males. p-values: Fisher's exact test. F = females, M = males, HTN = hypertension.

40s and 60s ($p = 0.002$ and 0.035, respectively). The lack of significance in the +70s group could have been due to the small sample size of this age decade.

Of note, the rate of increase in HTN prevalence was somewhat different between the genders; the slopes of the regression lines for males and females were 10.66 and 13.2, respectively. In addition, there was a dramatic increase in HTN prevalence in females between the age decades 30s and 40s, compared to that in males.

The Gambia and Sierra Leone patients

To check whether there were large differences in the demographics of subjects between the Gambia and Sierra Leone, the collected records for the year 2000 in the Gambia and the year 2001 in Sierra Leone were compared for the criteria SBP, DBP and HTN prevalence. Only the year 2001 was chosen to represent the data collected from Sierra Leone because the population sizes in the years 2000 and 2001 were comparable (Table 3).

The χ^2-test indicated more females and fewer males in the Gambia ($p < 0.0001$). The t-test showed that DBP means seemed to be similar between subjects from both countries ($p = 0.21$), while age and SBP means seemed to be different ($p = 0.0001$ and $p = 0.0002$, respectively), with Sierra Leone having higher means.

Furthermore, SBP and DBP means continually increased with age decade for both the Gambia and Sierra Leone subjects (Figs 4 and 5, respectively). In Sierra Leone, there were higher SBP means in the age decades 20s and 30s ($p = 0.013$ and $p = 0.002$, respectively) and lower SBP means in the age decade ≥ 70s ($p = 0.026$) in comparison with SBP means in the Gambia, as shown in Fig. 4.

The increase in mean SBP seemed to be faster in the Gambia when compared with Sierra Leone, based on the regression line slopes of 10.04 and 6.32, respectively (Fig. 4). Similarly, the increase in mean DBP seemed to be faster in the Gambia when

Table 3. Characteristics of patients with known age ≥ 18 years in the Gambia (2000) and Sierra Leone (2001)

Variable	*The Gambia (n = 560*)*	*Sierra Leone (n = 659)*	*p-value†*
Age (years)	36.0 ± 15.3	39.5 ± 16.0	0.0001
SBP (mmHg)	126.7 ± 26.1	132.1 ± 24.6	0.0002
DBP (mmHg)	80.4 ± 15.8	81.5 ± 14.5	0.21

Values: mean ± SD.
*The Gambia: $n = 561$ for age and SBP and $n = 560$ for DBP.
†Student's t-test.
SD = standard deviation, SBP = systolic blood pressure, DBP = diastolic blood pressure.

compared with Sierra Leone, based on the regression line slopes of 4.58 and 3.08, respectively (Fig. 5). As shown in Fig. 5, DBP mean in the Gambia was higher than in Sierra Leone in the age decade ≥ 70s ($p = 0.041$). The Wilcoxon test was more trusted for the small sample size, which was the case in the age decade ≥ 70s.

HTN prevalence appeared to be continually increasing with age decade for both the Gambia and Sierra Leone (Fig. 6). However, this increase seemed to be occurring at a faster rate in the Gambia than in Sierra Leone, as detected by the trend line slopes of 14.07 and 10.30, respectively. In addition, HTN prevalence in Sierra Leone was higher in the age decades 20s and 50s ($p < 0.0001$ and $p = 0.015$, respectively) compared to HTN prevalence in the Gambia.

Overall, among adults with known age ≥ 20 years old, the HTN prevalence rates in the Gambia in 2000 and in Sierra Leone in 2001 were 32.4 and 46.6%, respectively, while the Fisher's exact test showed a significant difference between both values ($p < 0.0001$). The Cochran–Armitage trend test showed a significant difference between the HTN prevalence of each age decade by country ($p < 0.0001$).

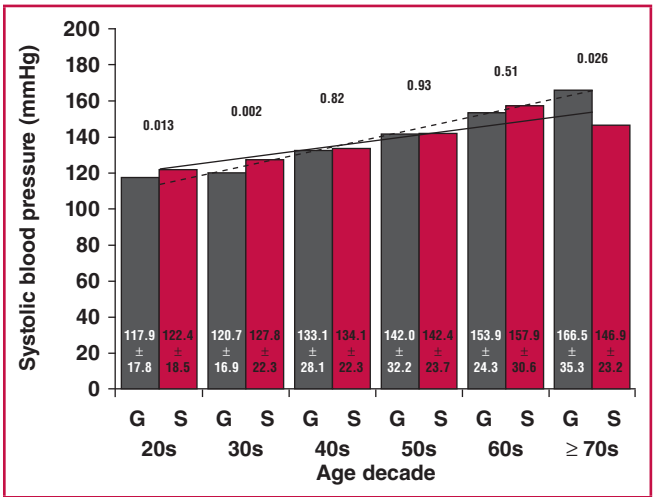

Fig. 4. Mean SBP of patients with known age ≥ 20 years in the Gambia (2000) and Sierra Leone (2001). Histogram of mean SBP ± SD. The Gambia = grey bars and dashed line, Sierra Leone = red bars and solid line. Regression equations: $y = 10.04x + 103.8$ ($R = 0.989$) for the Gambia and $y = 6.318x + 116.4$ ($R = 0.903$) for Sierra Leone. p-values: Student's t-test. G = the Gambia, S = Sierra Leone, SD = standard deviation, SBP = systolic blood pressure.

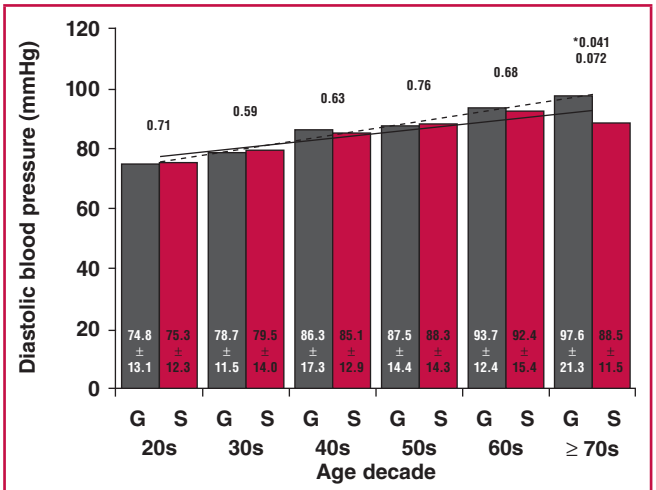

Fig. 5. Mean DBP of patients with known age ≥ 20 years in the Gambia (2000) and Sierra Leone (2001). Histogram of mean DBP ± SD. The Gambia = grey bars and dashed line, Sierra Leone = red bars and solid line. Regression equations: $y = 4.575x + 70.43$ ($R = 0.990$) for the Gambia and $y = 3.078x + 74.07$ ($R = 0.906$) for Sierra Leone. p-values: Student's t-test. *Wilcoxon rank-sum test p-value. G = The Gambia, S = Sierra Leone, SD = standard deviation, DBP = diastolic blood pressure.

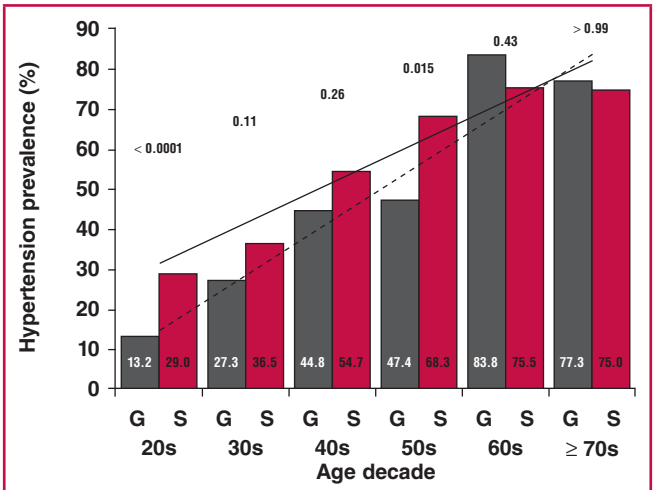

Fig. 6. HTN prevalence in patients with known age ≥ 20 years in the Gambia (2000) and Sierra Leone (2001). Histogram of HTN prevalence rates (%). The Gambia = grey bars and dashed line, Sierra Leone = red bars and solid line. Regression equations: $y = 14.07x - 0.293$ ($R = 0.957$) for the Gambia and $y = 10.30x + 20.44$ ($R = 0.963$) for Sierra Leone. p-values: Fisher's exact test. HTN = hypertension. G = the Gambia, S = Sierra Leone.

Sierra Leone patients

To check whether there was a trend in the data collected in Sierra Leone over the years 2001–2003 and 2009, ANOVA was performed on SBP and DBP LSmeans, adjusted for the relationship with age and separated by gender (Table 4). Adjusted for age, SBP LSmean in females was similar between 2009 and 2003 ($p = 0.84$), higher in 2003 than in 2001 ($p = 0.003$), and higher in 2001 than in 2002 ($p = 0.014$). DBP LSmean in females was higher in 2003 than in 2009 ($p = 0.0002$), similar between 2009 and 2001 ($p = 0.13$), similar between 2001 and 2002 ($p = 0.35$), and lower in 2002 than in 2009 and 2003 ($p = 0.029$ and $p < 0.0001$, respectively).

After age adjustment, SBP LSmean in males was higher in 2003 than in 2009 ($p = 0.043$), higher in 2009 than in 2002 ($p = 0.002$), and similar between 2002 and 2001 ($p = 0.73$). DBP LSmean in males was higher in 2003 than in 2009 ($p < 0.0001$), similar between 2009 and 2002 ($p = 0.068$), similar between 2002 and 2001 ($p = 0.41$), lower in 2001 than in 2009 and 2003 ($p = 0.022$ and $p < 0.0001$, respectively). To summarise, SBP and DBP LSmeans were generally higher in 2003 and 2009 compared to those in 2001 and 2002.

Table 4. Characteristics of patients with known age ≥ 18 years in Sierra Leone

Data collection year	*Gender*	*N*	*SBP (mmHg)*	*p-values*	*DBP (mmHg)*	*p-values*
2001*	F	297	135.1	0.014, 0.003	83.0	0.35, < 0.0001
	M	362	131.7	0.73, < 0.0001	81.3	0.41, < 0.0001
2002**	F	304	130.1	< 0.0001, < 0.0001	81.8	< 0.0001, 0.029
	M	359	132.3	< 0.0001, 0.002	82.2	< 0.0001, 0.068
2003†	F	209	141.8	0.84	92.6	0.0002
	M	74	150.7	0.043	95.9	< 0.0001
2009††	F	108	142.4	0.010	85.6	0.13
	M	74	142.4	0.0009	85.5	0.022

Values: least squares means (LSmeans).
p-values: *2001 vs 2002 and 2003, respectively, **2002 vs 2003 and 2009, respectively, †2003 vs 2009, ††2009 vs 2001.
F = females, M = males, SBP = systolic blood pressure, DBP = diastolic blood pressure.

Discussion

SBP, DBP and HTN trends

Mean SBP was shown to increase with age decade in both males and females (Fig. 1). There was a significant difference in mean SBP between the genders after age adjustment, with females having a higher mean SBP. Previous studies in Kenya, Tanzania, the Gambia and West Africa showed an increase in SBP with increasing age in both genders.[1,21,23,28] The study in Tanzania showed that the increase in mean SBP with age was steeper in females.[21]

Mean DBP increased with age decade and then plateaued as age decade reached +70s in both males and females (Fig. 2), which was similar to a previous study in the Gambia.[23] Prior studies showed that mean DBP increased with age and then plateaued by ages 45–54 and 55–64 years in Tanzania and West Africa, respectively.[21,28] Our study showed that females had a higher mean DBP than males after age adjustment.

HTN prevalence was shown to increase with age decade for both males and females (Fig. 3). Previous studies in Sierra Leone, Kenya and West Africa showed that HTN prevalence rates increased with age in both genders.[1,28,29] Comparing males to females, we found that females had higher odds and risk of HTN than males. Similarly, studies in Tanzania and Uganda showed that HTN was significantly higher in females.[21,30] This may have been due to post-menopausal hormonal changes.[31] Females showed a relatively higher HTN prevalence, starting with the age

decade 40s and above (Fig. 3), consistent with post-menopausal hormonal changes related to the observed increase in androgen levels post menopause.[32]

Knowing that obstructive sleep apnoea/hypopnoea syndrome (OSAHS) is a risk factor for developing HTN, post-menopausal women with OSAHS showed a higher prevalence of HTN when compared to those without OSAHS and to all pre-menopausal women.[33] It was also noted in the same study that among females with OSAHS, post-menopausal women had higher SBP and DBP averages when compared to pre-menopausal women. This may have been due to falling oestrogen levels in post-menopausal women, because oestrogen decline causes a rise in BP via the activation of the renin–angiotensin system, which in turn explains the observed higher plasma renin levels in post-menopausal females compared to males and pre-menopausal females.[32]

Furthermore, endothelin levels are higher in post-menopausal females, which explains in part the observed higher BPs, since endothelin causes sodium re-absorption, which in turn causes higher BP.[32] All of these factors make increasing age a risk factor of acquiring HTN in females, considering also the observation that about 60% of females aged > 65 years are hypertensive.[32]

HTN in Sierra Leone and the Gambia

This study highlights the high prevalence of HTN in the Gambia and Sierra Leone. HTN seems to be highly prevalent as a CVD in the sub-Saharan African region,[19] and may be rising over time. In 2006, a cross-sectional study in Uganda revealed that 252 individuals out of the 842 participants (29.9%) were hypertensive.[30] In 2007–2008, a study in Kenya found that 50.1% of 4 396 subjects were hypertensive.[1] In 1991–1995, HTN prevalence in rural and urban Cameroon was 17.3%; however, in 2003, the rate rose by an additional 7.3%.[34]

HTN in Sierra Leone was reviewed in several studies. Between 1983 and 1992, HTN accounted for about 7.5% on average of all deaths in Freetown, the capital of Sierra Leone.[35] A retrospective study, published in 1993, showed that among 87 subjects, 59 individuals were hypertensive.[36]

HTN prevalence, according to the HTN definition of ≥ 160/95 mmHg, was measured in four Sierra Leonean towns and villages. In 1998, in Njala Komboya and Kychum, HTN prevalence was 24.8 and 17.6%, respectively.[37] Similarly, in 1999, HTN prevalence was 23.4 and 14.7% in Freetown and Port Loko, respectively.[38] Recently, in Bo in 2009, 25.2% of 3 944 individuals aged ≥ 15 years old were hypertensive according to the HTN definition of ≥ 140/90 mmHg; however, the study showed no difference in BPs between males and females.[29] HTN prevalence by calendar year seems to agree with our results, showing that SBP and DBP LSmeans tended to be higher in the later years (2003 and 2009) than in the earlier years (2001 and 2002).

Several studies reviewed HTN in the Gambia. In 1996–1997, the HTN prevalence, according to the definition of ≥ 160/95 mmHg, was 7.1%, whereas by 1998, it rose to 10.2%, an increase of 3.1% in a year.[34] According to the HTN definition in the current study (≥ 140/90 mmHg), van der Sande showed in 1997 that 24.2% of 6 048 individuals in the Gambia were hypertensive.[23] Although the prevalence of HTN seems to be high in the Gambia, a study in 2001 pointed out that HTN prevalence in the Gambia varies with the specific geographical area in the country.[39]

These results show the high prevalence rate of HTN in the Gambia and Sierra Leone. Comparatively, in our current study, the HTN prevalence rate in both countries combined was 46.2% among females (n = 1 399), 43.2% among males (n = 1 215), and 44.8% overall (n = 2 614).

Influence of low SES

One major dilemma in sub-Saharan Africa is the low SES of countries in the region, including the Gambia and Sierra Leone. It was estimated that the total number of hypertensive adults in developing countries in 2000 was 639 million, compared to 333 million in developed countries,[20] which is a result of the difference in SES.[18]

The low SES establishes a variety of factors contributing to the prevalence of HTN, including a low HTN treatment rate, low levels of education and awareness, high salt and low potassium intakes, as well as an increased stress level. All these factors contribute directly or indirectly to the HTN prevalence rate among countries in the sub-Saharan African region.[18,34] Evidently, low SES was linked to high BP means, with a stronger effect on females than males.[40]

HTN treatment and SES

The treatment rate of a chronic disease depends on several factors, including the cost of the treatment associated with the disease. As mentioned, the SES of Sierra Leone and the Gambia is low and this may contribute to lack of availability of antihypertensive treatment.[18] A study in Kenya showed that only 15% of hypertensive individuals were able to obtain treatment for HTN.[1] A low SES contributed to the government not having adequate amounts of medications to distribute among patients.

In a 1999 survey in Cape Town, South Africa, 15.5% of patients reported that during filling prescriptions, insufficient medication was supplied.[41] A low SES also contributed to individual patients not having enough income to pay for the medications. In the Gambia, in 2006, the rate of unemployment was high.[42] Therefore, the inability to obtain medication was a factor contributing to the high HTN prevalence rate.

Education levels and SES

The awareness of HTN was previously correlated with the prevalence rate of the disease.[22] This awareness is usually provided by schools as well as public healthcare facilities. Establishment of schools has been difficult in societies with low SES. Concerning school education, in the Gambia, a research study showed that 10 and 56% of women aged 10–25 (n = 50) and 35–50 (n = 50) years, respectively, were unable to read, whereas 34% of 50 males aged 35–50 years were unable to read.[42] A study in Tanzania also pointed out that SBP was associated with education, which in turn was associated with SES; the higher the SES, the lower the SBP.[21]

Establishment and funding of public healthcare facilities, such as medical schools and nursing schools, has also been difficult in low SES countries. In 2000–2010, there were 0.4 physicians and 5.7 nurses and midwives per 10 000 individuals in the Gambia, while in Sierra Leone, there were 0.2 physicians and 1.7 nurses and midwives per 10 000 individuals. On the other

hand, in the USA, there were 26.7 physicians and 98.2 nurses and midwives per 10 000 individuals.[26]

These low healthcare provider-to-population ratios (61/100 000 in the Gambia and 19/100 000 in Sierra Leone) reflect the inadequate establishment and funding of public healthcare facilities in these countries. It is estimated that by 2015, according to the needs-based model, there will be a total of 45 countries in the world with physician shortage, 32 countries (~ 70%) of which are in Africa.[43]

Potassium and sodium levels and SES

HTN is related to sodium and potassium levels based on renin secretion, cellular sodium–potassium pumps and therefore the individual's nephron mass. The effect of sodium and potassium levels on HTN in the Gambia and Sierra Leone depends on two factors: the intrinsic propensities of the individual being of African descent and the individual's levels of salt intake, as well as vegetable and fruit (potassium) intake. Research in the USA and Europe illustrated that people of African descent had higher HTN prevalence and were at a higher risk of acquiring organ damage due to HTN,[23] in part because of lower nephron mass, macula densa mass, sodium detection levels and sodium–potassium pump activity.[17,44,45]

It was found that higher SBP and DBP means were apparent in individuals with higher sodium intake levels when compared with either intermediate or low sodium intake.[46] It has been shown that there is a linearly increasing correlation between sodium intake and HTN prevalence and mean SBP.[47,48] Globally, it was estimated that sodium intake in children older than five years of age was in excess by about 100 mmol/day.[49] This was a significantly high sodium intake level, considering that a high level of salt intake in infancy and childhood correlated to a high BP later in life.[50,51] In central and South Africa, it was found that sodium levels in cells and in circulating blood were high in hypertensive individuals.[18]

In the Gambia, intake of salt-preserved foods was high due to inadequate refrigeration. As a consequence, there was a high salt and sodium intake.[23] A low SES reduces the likelihood for a household to own a refrigerator and to receive electricity. In the Gambia, 14% of 50 females aged 14–25 years and 34% of 50 males aged 35–50 years did not receive electricity at home.[42] This electricity grid showed inadequate electricity reception in Gambian households, leading to an inability to refrigerate foods.

Furthermore, dietary potassium intake was related to BP.[52] Studies compared sodium to potassium intake and showed that in lower SES communities, the ratio between sodium and potassium intakes was high; however, the situation was nearly reversed in higher SES groups because potassium intake was higher than that in lower SES groups.[40] In Ghana, it was shown that an insufficient fruit and vegetable (a source of potassium) intake in 39.6% of males and 38.2% of females was considered a factor contributing to HTN prevalence.[53] Therefore, this low potassium intake assists in maintaining a high HTN prevalence rate.

Psychosocial status and SES

One of the factors contributing directly to an increased HTN prevalence is the psychological status of the individual, affected by stressors correlating with low SES. Studies have shown that stress, economic transition, and high BP may be correlated.[25,54]

As noted by a recent study, there is a significant association between psychosocial stress and endothelial dysfunction, which contributes to the development of CVD.[24] The study found that cold stress caused a more prominent increase in DBP in white South Africans compared to black South Africans. In addition, black Africans who reported higher levels of psychosocial distress had lower L-arginine/ADMA (asymmetric dimethylarginine) ratio. ADMA is known to be an inhibitor of the endothelial NO synthase, which produces NO from L-arginine. The study concluded that psychological distress significantly affects the L-arginine/NO system, with some ethnic differences.[24]

In a low-SES community, employment levels are very low, therefore leading to an increase in stress levels for individuals in a household due to the lack of an income source to support a living. Another possible source of stress in the Gambia and Sierra Leone was the instability in both societies. The instability in Sierra Leone was due to the persisting civil war from 1991 to 2002, while in the Gambia, it was due to their increasing potential population due to the outmigration of Sierra Leoneans to surrounding countries, one of which was the Gambia. This instability could have served as a psychological stressor that led to the increase in HTN prevalence in both populations.

Additional HTN risk factors

Additional possible risk factors for HTN include smoking, alcohol consumption, schistosomiasis specifically in the Gambia, and certain genotypic correlations. Smoking is associated with CVD and increases HTN risk by two- to three-fold.[55] In the Gambia, by 2006, 29.2% of males and 2.6% of females were tobacco smokers, compared to the USA, where 25.4% of males and 19.3% of females were tobacco smokers.[26]

Alcohol intake also contributes to a higher BP and the prevalence of HTN.[56] A study in Uganda considered past and present alcohol intake as a risk factor associated with HTN prevalence.[30] As for the Gambia and Sierra Leone, by 2005, the total alcohol consumption among adults aged ≥ 15 years in the Gambia was 2.4%, while in Sierra Leone, it was 6.5%, compared to 8.5% in the USA.[26]

In the Gambia, schistosomiasis was another risk factor. Studies showed that prevalence of diastolic hypertension in adults was two- to four-times higher in *Schistosoma haematobium* endemic areas,[23] including parts of the Gambia and Sierra Leone.

Regarding genotypic influences, recent studies relate certain loci and some single nucleotide polymorphisms in the human genome to BP and HTN. An admixture mapping study identified a probable relatioship between chromosomes 6q24 and 21q21 and HTN risk in African Americans.[57] These two regions included two loci on chromosome 21q, and five other markers on chromosome 6q that suggested a genetic linkage to elevated BP.

One genome-wide association study (GWAS) related three genes, previously associated with BP in Americans of European descent, with BP in African Americans.[58] The three genes were *SH2B3*, *TBX3-TBX5*, and *CSK-ULK3*, all of which are genetic variants influencing BP in African Americans, and generally in people of Africa descent.

Influences of lifestyle and economic development

Lifestyle changes, including smoking cessation, lower alcohol

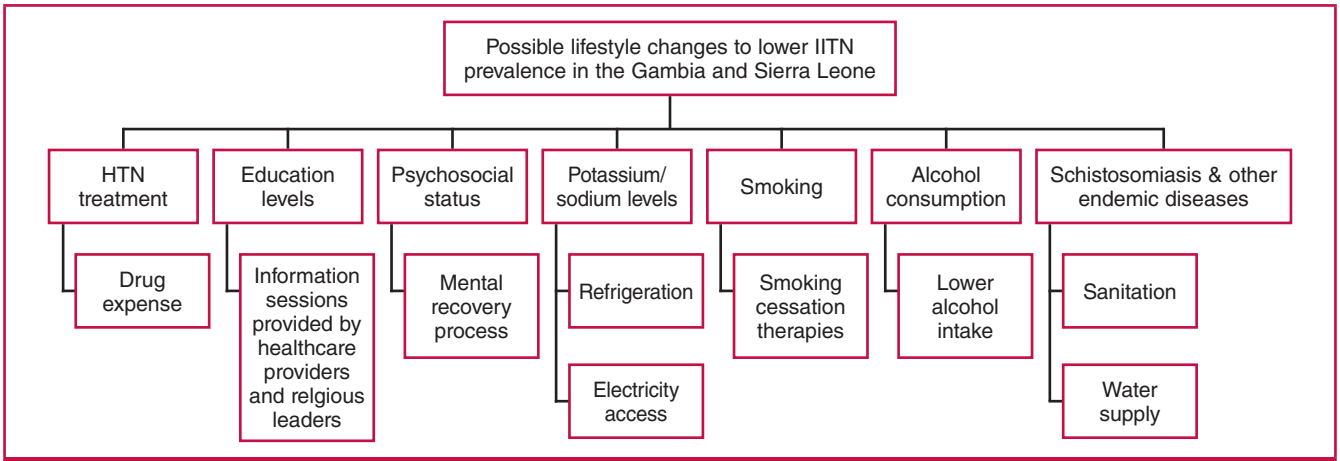

Fig. 7. Summary of possible lifestyle changes to lower HTN prevalence in the Gambia and Sierra Leone. HTN = hypertension.

intake, sanitation, clean water supply, refrigeration, and electricity access may influence HTN prevalence (Fig. 7). The ability to make lifestyle changes may be related to SES, educational level and economic development.

The economic development of both countries could raise both countries' SES, thus diminishing several factors contributing to HTN prevalence. Sources of economic development in the Gambia and Sierra Leone could include the many natural resources present in these countries, which could be used for economic self-sufficiency. With economic development, both governments could offer funds for healthcare systems to lower HTN prevalence.

Concerning HTN awareness, healthcare providers in healthcare centres and religious leaders in religious institutions could routinely make people aware of the disease, its progression and burden, and its preventive means.[53] The government could also establish national policies and programmes so that all individuals, whether educated or not, would have an idea about the existence of the disease HTN. In the Gambia, an improvement in education and disease awareness is already underway.[42]

Increased potassium (vegetable/fruit) intake and lowered sodium intake are needed for protection against HTN.[18,20,46] The high sodium (salt) intake is mainly due to the unavailability of food preservation via refrigeration. Affordable electricity systems could be established using the Berra Kunda waterfalls in the Gambia on the border with Senegal, and the Bumbuna waterfalls in Sierra Leone for hydroelectric power.

Chronic financial stress related to low SES and poor economic conditions is potentially modifiable. A study in Ghana and Cameroon suggested that religious institutions and leaders should encourage the people to overcome their financial problems and to start a recovery process from their stress.[53]

Considering schistosomiasis, the Gambia and part of Sierra Leone fall within the endemic region of the disease.[23,59] The main prevention against such parasitic diseases is the improvement of drinking water sources and sanitation facilities. Improper sanitation and water supply are related to ascariasis, diarrhoea, trachoma, schistosomiasis and other diseases.[60] An analysis showed that cleaner water supplies led to a median reduction in schistosomiasis morbidity rate of 69% for all studies and 77% for four selected rigorous studies.[60]

Study limitations

Study limitations include the gap in data collection in Sierra Leone since the data were collected from 2001 to 2003 and in 2009. The data from the Gambia were only collected in 2000, which may result in a smaller sample size from the Gambia contributing to the findings of the study. In addition, combining the data collected from both countries could potentially be a weakness in the study, taking into account the fact that there were some minor differences between the data collected from the Gambia in 2000 and from Sierra Leone in 2001, as discussed above. Finally, some ages were missing from the records, resulting in the exclusion of these individuals from the statistical analyses involving age.

Conclusion

HTN was highly prevalent in the Gambia and Sierra Leone. This may have been due to low HTN treatment rates, low education and awareness levels, low potassium and high sodium intakes, and high stress levels, all of which are part of the persistently low SES in both countries. Additional risk factors include smoking, alcohol consumption, identified genetic loci and endemic diseases. Lifestyle changes need to be instituted to lower this high prevalence of HTN. Changes include raising the awareness of the disease, initiating a stress-recovery process, finding alternative ways to preserve foods and improving sanitation and water supply sources.

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Is chronic obstructive pulmonary disease a risk factor for epistaxis after coronary artery bypass graft surgery?

Faruk Cingoz, Bilgehan Savas Oz, Gokhan Arslan, Adem Guler, Mehmet Ali Sahin, Celalettin Gunay, Mehmet Arslan

Abstract

Background: Chronic obstructive pulmonary disease (COPD) has customarily been associated with increased surgical morbidity and mortality rates after coronary artery bypass graft surgery (CABG). The aim of this study was to determine whether there is a relationship between epistaxis and COPD after CABG surgery.

Methods: There were 3 443 patients who consecutively underwent isolated CABG from January 2002 to March 2012. We retrospectively analysed the data of 27 patients (0.8%) with newly developed and serious spontaneous epistaxis, which required consultation with the Ear Nose and Throat (ENT) Department. The patients were divided into three groups according to severity of nasal bleeding. Twenty-one (77.7%) patients in the three groups had COPD.

Results: There were 19 males (70%) and eight females (30%). Their ages ranged between 52 and 72 years (mean 61 ± 5). Fifty-five per cent of the patients had hypertension and 78% had COPD. The overall duration of hospital stay was six to 11 days (mean 7.9 ± 1.1). Epistaxis was seen particularly on the fourth and seventh days postoperatively and 17 patients (63%) were treated with anterior, posterior, or anterior and posterior nasal packing (group 1). Nasal bleeding was controlled with electrocautery in six patients (22%) (group 2), and four (15%) were treated with surgical excision and blood transfusions (group 3). All patients (100%) had a good recovery with no mortality.

Conclusion: The high coincidence between epistaxis and COPD made us wonder whether COPD may be a risk factor for epistaxis after CABG surgery. However, we could not find any direct causative link between COPD and epistaxis in patients who had undergone CABG. Epistaxis was more common in patients with COPD and it was more serious clinically in patients who had both COPD and hypertension.

Keywords: epistaxis, chronic obstructive pulmonary disease, coronary artery bypass surgery

Department of Cardiovascular Surgery, Gulhane Military Medical Academy, Etlik, Ankara, Turkey
Faruk Cingoz, MD, fcingoz@yahoo.com
Bilgehan Savas Oz, MD
Gokhan Arslan, MD
Adem Guler, MD
Mehmet Ali Sahin, MD
Celalettin Gunay, MD
Mehmet Arslan, MD

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Epistaxis is the most common otolaryngological emergency that affects up to 60% of the population in their lifetime. Six per cent of all epistaxis cases require medical attention.[1]

Chronic obstructive pulmonary disease (COPD) is often considered a risk factor for postoperative morbidity and mortality after coronary artery bypass graft (CABG) surgery. Postoperative complications such as respiratory failure, re-intubation, sternal dehiscence, prolonged mechanical ventilation, rhythm disturbances and prolonged hospital stays are known complications of COPD in CABG patients.[2]

Epistaxis is a rare complication that is not directly related to heart surgery.[3] Many factors affect bleeding after cardiac surgery, such as thrombolytic agents, hypertension, trauma and nasal oxygen therapy. Data on the association between epistaxis and CABG surgery is less clear.[4] There is a paucity of published data regarding the management of epistaxis in patients with COPD who undergo CABG. We conducted this study to determine whether there was a relationship between epistaxis and COPD after CABG surgery.

Methods

This was a retrospective study. All patients of any age who consulted at the Ear Nose and Throat (ENT) Department with a diagnosis of serious spontaneous epistaxis requiring at least one nasal pack after CABG surgery were included in the study. Patients were divided into three groups according to the severity of nasal bleeding, which was determined by treatment modality. The three procedures included packing (anterior, posterior or anterior-posterior) (group 1), treatment with electrocautery (under direct vision or via endoscopic guidance) (group 2) and surgical ligation of bleeding vessels (group 3).

The 3 443 patients who underwent isolated CABG from 2002 to 2012 were assessed in this study and follow up was obtained from a review of their charts. We focused on only objective data obtained from the medical records, and analysed a total of 27 (0.8%) patients with complete data who consulted at the ENT Department with a diagnosis of spontaneous and incipient epistaxis (Tables 1–3).

All patients were operated on via a median sternotomy, with standard cardiopulmonary bypass procedure and moderate hypothermia. Myocardial preservation was accomplished with intermittent antegrade delivery of St Thomas II solution. Cardiopulmonary bypass was initiated after anticoagulation with bovine lung heparin. The heparin was reversed by protamine at

Table 1. Pre-operative patient characteristics

Male/female	19/8
Age (years), *n* (mean)	52–72 (61 ± 5.09)
Body mass index (kg/m²)	27.55 ± 1.36
Smoking, *n* (%)	17/27 (63)
Diabetes mellitus, *n* (%)	13/27 (48.1)
Hypertension, *n* (%)	15/27 (55.6)
aPTT (s)	31 ± 2.801
INR	1.107 ± 0.1107
Platelet count (/µl)	274444.44 ± 74644.456
LVEF (%)	55.05 ± 3.79

aPTT: activated partial thromboplastin time, INR: international normalised ratio, LVEF: left ventricular ejection fraction.

the end of the cardiopulmonary bypass. Nasal wet oxygen therapy (2–4 l/min) was initiated in all patients after extubation. Aspirin treatment (300 mg/day) was started after mediastinal bleeding had ceased in all patients. Hypertension was diagnosed if the systolic blood pressure (BP) was above 140 mmHg or diastolic BP was above 90 mmHg on two or more occasions.

Postoperative records up to discharge from hospital and available results related to the end-points were compared. The primary end-point was spontaneous epistaxis (with ENT consultation), requiring at least one nasal packing after CABG surgery, and any complications related to nosebleed and treatment, excluding death. The secondary end-point was death related to epistaxis.

Statistical analysis

All available data were analysed with the computer program SPSS (Statistical Package for Social Sciences) for Windows 17.0 (Chicago, IL, USA). Descriptive statistical methods (number, percentage, mean, standard deviation) were used. Differences in variables were analysed using the Mann–Whitney *U*, Kruskal–Wallis and chi-square tests as appropriate, and *p*-values of less than 0.05 were considered significant.

Results

A total of 27 patients with epistaxis consulted at the ENT Department. One patient had a history of epistaxis pre-operatively. There were 19 males (70 %) and eight females (30%), and their ages ranged between 52 and 72 years (mean 61 ± 5); 55% had hypertension, 78% COPD, 48% diabetes mellitus and 63% a history of smoking.

Table 2. Demographics and patient data

	Total number (%)		*Group 1*		*Group 2*		*Group 3*			
			n	*%*	*n*	*%*	*n*	*%*	*p*	χ^2
Gender	27	Female	5	62.5	2	25.0	1	12.5	0.960	0.081
		Male	12	63.2	4	21.1	3	15.8		
Smoking	17 (62.9)	No	6	60.0	3	30.0	1	10.0	0.704	0.703
		Yes	11	64.7	3	17.6	3	17.6		
Diabetes mellitus	13 (48.1)	No	8	57.1	4	28.6	2	14.3	0.708	0.689
		Yes	9	69.2	2	15.4	2	15.4		
Hyper-tension	15 (55.5)	No	9	75.0	3	25.0	0	0.0	0.152	3.772
		Yes	8	53.3	3	20.0	4	26.7		
COPD	21 (77.7)	No	5	83.3	1	16.7	0	0.0	0.415	1.758
		Yes	12	57.1	5	23.8	4	19.0		

*Statistical significance was established as $p < 0.05$.
COPD: chronic obstructive pulmonary disease.

Table 3. Patient characteristics and haematological parameters in the three groups

Variables	*Group*	*n*	*Mean ± SD*	*KW*
Age	Group 1	17	61.760 ± 5.391	0.696
	Group 2	6	60.170 ± 4.708	
	Group 3	4	59.500 ± 5.000	
Hospital stay (days)	Group 1	17	7.470 ± 0.717	11.469
	Group 2	6	8.170 ± 1.169	
	Group 3	4	9.750 ± 0.957	
BSA (kg/m²)	Group 1	17	27.235 ± 1.427	2.454
	Group 2	6	28.033 ± 1.155	
	Group 3	4	28.200 ± 1.178	
LEVF (%)	Group 1	17	54.940 ± 3.944	0.400
	Group 2	6	56.170 ± 3.061	
	Group 3	4	55.750 ± 5.439	
Platelet count (per µl)	Group 1	17	252 352.940 ± 68 786.755	3.963
	Group 2	6	320 000.000 ± 56 213.877	
	Group 3	4	300 000.000 ± 100 000.000	
aPTT (s)	Group 1	17	30.530 ± 2.718	3.217
	Group 2	6	30.830 ± 3.251	
	Group 3	4	33.250 ± 1.708	
INR	Group 1	17	1.100 ± 0.117	0.371
	Group 2	6	1.133 ± 0.121	
	Group 3	4	1.100 ± 0.082	

*Statistical significance was established $p < 0.05$
BSA: body surface area, LVEF: left ventricular ejection fraction, aPTT: activated partial thromboplastin time, INR: international normalised ratio.

The overall duration of hospital stay ranged from six to 11 days (mean 7.9 ± 1.1) (Tables 1–3). Epistaxis was seen specifically on the fourth and seventh days postoperatively. Two patients had two or more epistaxis episodes within 15 days of discharge from hospital. According to the degree of bleeding, group 1 patients ($n = 17$, 63%) were treated with anterior, posterior, or anterior and posterior nasal packing. Group 2 patients ($n = 6$, 22%) were treated by electrocautery. Group 3 patients ($n = 4$, 15%) were treated by surgical ligation of the bleeding vessels and blood transfusions. These four patients in group 3 had two risk factors: hypertension and COPD (Table 2).

All pre-operative patient characteristics and co-morbid factors between the groups were similar ($p > 0.05$) (Tables 1–3). Although all patients with epistaxis presented with discomfort in the postoperative period, all patients (100%) had a good recovery with no mortality. Group 3 patients had profuse nasal bleeding that needed surgical intervention, and both COPD and hypertension were diagnosed in all four of these patients.

Discussion

Epistaxis is classified on the basis of the primary bleeding site being anterior or posterior. A common source of anterior epistaxis is the Kiesselbach plexus, an anastomotic network of vessels on the anterior portion of the nasal septum. Posterior bleeding occurs mainly from the branches of the sphenopalatine artery in the posterior nasal cavity or nasopharynx.[5]

There are many factors causing epistaxis, including environmental factors (humidity, temperature), local factors (inflammation, deviated septum and/or perforation, tumours, foreign bodies, aneurysm), systemic factors (hypertension, haematological abnormalities, renal failure, alcoholism, arteriosclerosis, telangiectasis), and medications affecting clotting (anticoagulants, non-steroidal anti-inflammatory drugs).[6] The literature does not provide a precise definition on the severity of epistaxis, which is often based on subjective impressions (subjective evaluation of the volume of bleeding) or anatomical features, essentially posterior epistaxis.[7]

COPD is a known risk factor for morbidity and mortality in heart surgery. Postoperative complications such as respiratory failure, prolonged mechanical ventilation and oxygen uptake, re-intubation, sternal dehiscence, pulmonary infection, rhythm disturbances and prolonged hospital stays are known complications in COPD patients after CABG.[2]

We could not find any literature on epistaxis in patients with COPD undergoing CABG surgery. In COPD patients, drying and thinning of the nasal mucosa due to long-term nasal oxygen uptake or nebulised use of corticosteroids may cause epistaxis.[8] Irritation by the endotracheal tube in the pulmonary system induces the cough reflex and coughing may cause sudden hypertension in the blood vessels in the nasal cavity. However we do not believe that in our cases, these factors were the cause of excessive nasal bleeding after CABG.

Hypertension and antiplatelet therapy may be a predisposing factor for nasal bleeding in COPD patients post CABG. Aspirin is thought to be a risk factor for epistaxis.[3] The relationship between hypertension and epistaxis is unclear.[4] In our study, neither hypertension nor aspirin were found to be independent risk factors for epistaxis.

However the presence of COPD in all patients (100%) with epistaxis, requiring surgical intervention and blood transfusion, induced us to conduct this study. Profuse nasal bleeding was seen if the patients had both COPD and hypertension. This analysis was conducted in our setting to identify the aetiological profile and to determine the outcome of treatment for epistaxis after CABG surgery. The results of this study may provide a basis for the planning of preventive strategies and the establishment of treatment guidelines.

There was profuse nasal bleeding requiring surgical intervention in all patients in group 3. Both COPD and hypertension were diagnosed in all four of these patients. Although there was no statistically meaningful results for nasal bleeding in these patients because of the low number of cases ($p = 0.415$), there was an interesting connection between COPD and hypertension after CABG surgery. Our results showed that both COPD and hypertension were present in patients with serious nasal bleeding after CABG surgery.

A limitation of this study was that the incidence of epistaxis in the early postoperative period after CABG was low. In our study, only 27 patients had epistaxis. These patients were divided into three groups according to the amount of nasal bleeding and the type of treatment, and each group contained only a few patients. There are also many causes of epistaxis after CABG.

Conclusion

Epistaxis is a co-morbid factor in a small number of patients with COPD after CABG. It may result in a serious clinical situation with regard to the amount of nasal bleeding when seen in patients with COPD alone or with both COPD and hypertension. From to our results, we recommend that when COPD and hypertension coincide, cardiac surgeons should keep in their mind that serious nasal bleeding may occur in these patients after CABG surgery. If so, they should be sent immediately to an ENT specialist for appropriate treatment.

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De novo atrial fibrillation post cardiac surgery: the Durban experience

Ebrahim Mansoor

Abstract

Atrial fibrillation (AF) is the most common complication post cardiac surgery and results in elevated morbidity and mortality rates and healthcare costs. A pilot, retrospective study of the medical records of all adult patients developing *de novo* AF post surgery was undertaken at the cardiac surgical unit in Durban between 2009 and 2012. We aimed to describe the local experience of AF with a view to suggesting an adapted local treatment policy in relation to previously published data. Fifty-nine patients developed AF during the study period. AF occurred predominantly three or more days post surgery. Thirty-five patients required cardioversion and amiodarone to restore sinus rhythm. Return to the general ward (RGW) was 4.6 days longer than the institutional norm. Liberal peri-operative β-blocker and statin use is currently preferred to a formal prophylaxis strategy. Randomised, controlled trials are required to evaluate measures curbing prolonged length of stay and morbidity burdens imposed by AF on the local resource-constrained environment.

Keywords: cardiac surgery, atrial fibrillation, arrhythmia, cardioversion, amiodarone, β-blocker

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Atrial fibrillation (AF) is the most common arrhythmia after cardiac surgery. This complication constitutes significant morbidity and mortality rates for the cardiac surgical patient.[1] Consequential increase in length of stay (LOS), partly on the basis of thromboembolic events, incurs a financial burden on health institutions.

Although the entity of *de novo* AF post cardiac surgery has been intensely studied globally, a grave paucity of data exists from the developing world. The aim of this study was to describe the South African experience of *de novo* AF post cardiac surgery with special emphasis on the issues pertaining to a resource-limited setting.

Methods

A retrospective uni-centre audit of a prospectively collated database between December 2009 and February 2012 was undertaken at the Department of Cardiothoracic Surgery, Inkosi Albert Luthuli Central Hospital (IALCH) in Durban, South Africa. All adult patients who developed *de novo* AF post coronary and valve surgery were included in the study. Paediatric patients and patients with chronic pre-operative AF were excluded from the cohort.

Data was extracted from patients' medical records. Atrial fibrillation was defined as an arrhythmia with irregular irregularity and absent P waves. Diagnosis of AF was confirmed on telemetry and 12-lead electrocardiogram (ECG). AF was managed as per the European Association of Cardiothoracic Surgeons (EACTS) 2006 guideline.[2] Selective deviations from the guideline were on a patient-specific basis upon consultation with the Department of Cardiology.

The following parameters were evaluated: demographics: age, gender, race; type of surgery: coronary, valve or combinations thereof; risk factors for AF (among others hereunder listed): pre-operative withdrawal of β-blockers, prior cardiac surgery, body mass index (BMI), smoking (within six months prior to surgery); nature of surgery: emergency or elective; co-morbidities: hypertension, diabetes mellitus; time of AF presentation: < 24 hours, 24–48 hours and > 48 hours post surgery; medication: pre-operative use of statins and β-blockers; treatment of AF: none, i.e. spontaneously resolved, electrical cardioversion, amiodarone use, or a combination of cardioversion and amiodarone; pre-operative echocardiographic parameters: left ventricular diastolic dimension (LVD), left atrial size (LA), ejection fraction (EF).

Return to the general ward (RGW) is a surrogate concept introduced to quantify LOS and cost burden imposed by the development of AF in the post-operative period. The institutional norm is two days, one day in the intensive care unit (ICU) and another in the high-care ward, after which time the patient returns to the general cardiac surgical ward.

In addition to the above, follow-up information available at the time of data presentation was analysed, particularly the time from surgery, β-blocker use and maintenance of sinus rhythm were recorded. The study was approved by the Biomedical Research Ethics Committee (BREC) at the University of KwaZulu-Natal (BE296/13).

Results

Fifty-nine patients developed *de novo* AF after cardiac surgery in the index cohort during the study period. Considering the total of 997 adult patients who underwent surgical intervention for coronary or valve-related pathology in this period, the institutional AF rate was 5.9%. The number of patients developing AF per surgical procedure is shown in Table 1.

Thirty-three patients (55.9%) had coronary artery surgery, either alone or in combination with valve surgery. All six patients who underwent combination valve and coronary surgery had aortic valve replacements. Off-pump coronary surgery consisted

Department of General Surgery, in association with the Department of Cardiothoracic Surgery, University of KwaZulu-Natal, Durban, South Africa
Dr Ebrahim Mansoor, MB ChB, dremansoor@gmail.com

Table 1. Incidence of AF per surgical procedure

Type of surgery	*Number developing AF*	*Total number of surgeries*	*Percentage of cohort (%)*
Coronary surgery			
Coronary artery bypass graft surgery (CABG)	13	270	45.8
Off-pump coronary bypass surgery (OPCAB)	14	251	
Valve surgery			
Mitral valve replacement (MVR)	7	251	44.1
Aortic valve replacement (AVR)	11	85	
Double valve replacement (DVR)	8	113	
Combination coronary and valve surgery			
CABG + MVR	0	12	10.2
CABG + AVR	6	12	
CABG + DVR	0	3	
Total	59	997	

of 23.7% of the cohort.

Thirty-three patients in the cohort were male and 26 were female. Age ranged from 16 to 82 years (mean: 51.9 years). Thirty-four patients were of Indian descent, while 17 were black and the remaining eight were white patients. The majority (79.4%) of Indian patients had coronary surgery alone or in combination with valve surgery. Fifty-four patients had elective surgery and five had surgery on an emergency basis.

RGW ranged from the institutional norm of two days up to a maximum of 24 days. The mean RGW duration was 6.6 days (n = 54). Six patients died during the index admission period, three of whom had emergency surgery.

Twenty-four patients were diabetic and 35 had hypertension. Twenty patients had both diabetes and hypertension.

Thirty-seven patients used β-blockers and 40 were on statin therapy on a chronic basis. Thirty-two patients in the cohort used a combination of statins and β-blockers prior to surgery. β-blockers were withdrawn in only three patients in the immediate pre-operative period.

Miscellaneous risk factors were BMI, previous cardiac surgery and smoking. The BMI ranged from 17 to 42 kg/m^2, with a mean of 26.4 kg/m^2 (n = 54). Three patients in the cohort had prior cardiac surgery. Twelve patients were still smoking in the immediate pre-operative period. The echocardiographic results are reflected in Table 2.

Table 2. Echocardiographic parameters

Parameter	*Patient number*	*Range*	*Mean*
LVD (mm)	50	42–75	56.1
LA (mm)	50	33–90	51
EF (%)	52	25–66	52.8

An analysis of the subgroup of patients (n = 33) who underwent coronary artery surgery alone or in combination with valve surgery is as follows: mean age was 62.2 years, there were 26 male and seven female, mean BMI (n = 31) was 26.9 kg/m^2, 33 patients were on statins and 31 on β-blockers. Mean echocardiographic parameters (n = 27): LVD = 56.3 mm, LA = 45.9 mm, EF = 51.6%.

The coronary patients were a mean of 10.3 years older than the whole cohort and 78.8% were male. The other parameters closely resembled that of the entire cohort.

The majority of patients (64.4%) developed AF from day three onwards. The incidence of AF in the individual post-operative time periods is reflected in Fig. 1. The various individual and combination treatment modalities used to treat AF after cardiac surgery is shown in Fig. 2.

Follow-up data was available for 40 patients at the time of data presentation. Follow-up duration ranged from 1.5 to 38 months after surgery, with a mean of 16.1 months. At the follow-up visit, all 40 patients were in sinus rhythm. Twenty-two patients were at that stage noted to be using β-blocker therapy for underlying chronic cardiac conditions.

Discussion

De novo atrial fibrillation post cardiac surgery is a post-operative complication associated with significant morbidity and mortality.[3] Hakala *et al.* demonstrated, in a retrospective study of 3 676 Finnish patients, an increase in peri-operative cardiovascular accidents (CVA), confusion, ICU LOS and ICU re-admission rates.[4] Almassi *et al.* showed significantly higher

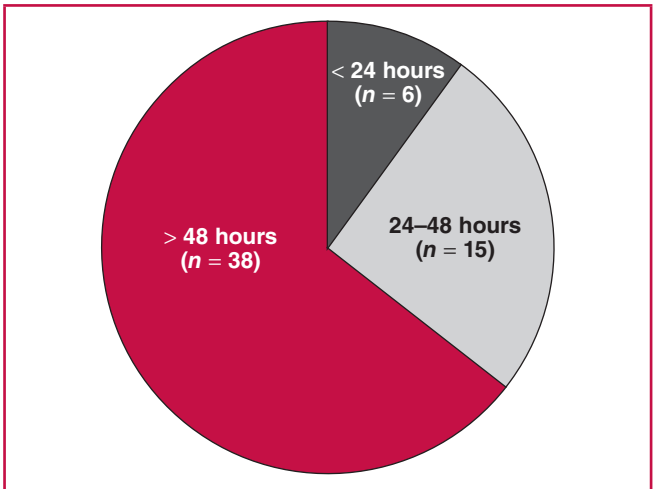

Fig. 1. Timing of AF presentation.

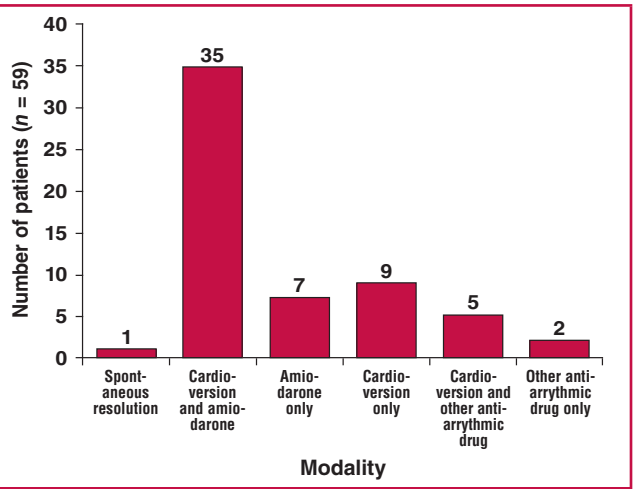

Fig. 2. Modalities used to treat AF.

hospital and six-month mortality rates in patients in whom AF occurred.[1]

The mechanisms involved in the development of AF in the cardiac surgical patient are incompletely elucidated. It is probable that pericardial inflammation, catecholamine increase, autonomic disharmony, atrial stretch, metabolic abnormalities, transcellular fluid and electrolyte shifts, and neurohormonal activation act alone or in concert in the production of AF. These factors shorten the atrial refractory period, slowing atrial conduction. Resultant re-entry wavelets bombard the atrio-ventricular node to produce rapid and irregular ventricular contraction.[5]

Atrial structural alterations, inherent or iatrogenic, affect individual susceptibility to AF. It is widely accepted that the initiation and perpetuation of AF requires a triggering factor and an electrophysiological substrate within the atria. The substrate is mandatory and perhaps explains why some post-operative patients develop AF and some do not.[6] Furthermore, a genetic predisposition has been proposed in patients with the interleukin-6 promoter gene variant.[3]

AF post cardiac surgery is postulated to arise in a milieu of sympathetic hyperactivity in the post-operative period. Several patient and procedure-related factors have been suggested to confer increased individual vulnerability. The inflammatory state induced by cardiopulmonary bypass, atrial incisions and the relative ischaemia of the atrial septum when cardioplegic solution is delivered via the coronary circulation contribute to the complex processes involved in the generation of AF.[7]

The AF incidence of 5.9% in our cardiac surgical unit is within the widely varied range of incidence reported in most international series, between 5.5 and 65%.[7,8] A meta-analysis of 24 trials estimated the incidence at 26.7%.[9] A comparison of eight studies with cohorts of 500 or more patients evaluating the incidence and pre-operative risk factors for atrial arrhythmias after cardiac surgery confirmed a comparable incidence between AF post coronary artery bypass surgery (CABG) and post valve surgery, but an increased incidence after combination surgery.[7]

Coronary and valve surgery patients were virtually equally represented in our cohort, however Creswell *et al.* noted that AF incidence was in fact increased in valve and combination valve–coronary surgery.[10] In the developing world, it is our experience that patients with valve disease present later, once AF has already developed. Since these patients were excluded from our study, this may have accounted for the unremarkable difference in incidence between the valve and coronary surgery groups.

The vast heterogeneity between definitions and study group compositions makes a direct comparison of incidence and other parameters challenging. Several studies do not allude to a definition for AF or method of detection, while others considered only patients requiring intervention for AF. The African incidence of AF post cardiac surgery remains undocumented.

The highest incidence of AF is seen on post-operative days two to three, with fewer patients developing AF in the early post-operative period or beyond four or more days.[7] Seventy per cent of patients develop AF before the end of day four and 94% before the end of day six.[11] Mathew *et al.* documented the peak incidence of AF on postoperative days two and three in their prospective, observational study of 4 657 patients undergoing CABG in 70 centres. Fifty-seven per cent of patients had only a single episode of AF during their hospitalisation.[8]

Our study showed that AF was most prevalent from the third post-operative day and not in the immediate post-operative period (< 24 hours). This supports the notion that AF generation is complex and that it extends beyond the peak adrenergic surge prevalent on the first post-operative day.

Tsikouris *et al.* demonstrated P-wave dispersion and atrial conduction time to be greatest on days two to three, and day three, respectively.[12] It remains unclear whether these electrophysiological alterations contribute wholly or in part to the increased development of AF after the first post-operative day.

A wide array of risk parameters has been evaluated globally, with conflicting results. Mathew *et al.* found age to be an independent risk factor for AF post cardiac surgery and this finding has consistently been reported by others.[7,8] The risk of AF increases by at least 50% per decade and particularly so over 70 years of age. Inflammatory and degenerative changes associated with advanced age cause atrial fibrosis and degeneration.[7,13] The resultant alterations in electrophysiological properties may act as substrates for AF. Most of the parameters considered in this study, albeit few, revealed results that were within normal limits.

Hypertension has been proposed to predict AF after cardiac surgery and this may be related to associated fibrosis and dispersion of atrial refractoriness.[1,8,11] However, several well-conducted trials with large numbers of patients have refuted this proposition.[3,14,15]

Men are more likely to develop AF than women. This disparity may be explained by gender differences in ion-channel expression, and hormonal effects on autonomic tone.[11,15] However, Mathew *et al.* and Echahidi *et al.* expressed a contradictory viewpoint on male predominance.[3,8]

Withdrawal of β-blockers pre-operatively in patients on chronic β-blocker therapy causes withdrawal effects, as described by Kalman *et al.*[16] The poor oral absorption of β-blockers in the post-operative period described by Valtola *et al.* is likely to exacerbate this withdrawal effect.[17]

Beating-heart coronary surgery (OPCAB) did not appear to provide any obvious protective benefit to the development of AF in this study. Notwithstanding the heterogeneity in OPCAB trials, off-pump surgery is still believed to be associated with a significant reduction in post-operative AF.[2]

In our study, only three patients had redo-operations and another three β-blocker withdrawal in the pre-operative period. Therefore no deductions could be made. Twelve patients were noted to be smoking up to the time of surgery. This information was confession-based and furthermore, all patients were advised to cease smoking at the cardiac surgical preview visits.

In their retrospective analysis of 5 058 patients post isolated CABG in patients older than 50 years, obesity was shown by Echahidi *et al.* to be an independent risk factor for AF.[3] Higher cardiac output requirements, left ventricular mass and left atrial size predisposed to AF in the obese population.[18] Filardo *et al.* reported a significant relationship between BMI and AF. Their study of 7 027 consecutive patients post CABG with a mean age of 64.9 years showed a median BMI of 28 kg/m^2.[19] In our study, a mean BMI of 26.4 kg/m^2 indicated an overweight population, as per the World Health Organisation, but no relationship was readily apparent between BMI and AF in our cohort.

In our study, all patients were found to be AF free at follow up. The natural history of AF post surgery is that

of spontaneous resolution within six weeks, irrespective of treatment modality used. Up to 80% of patients convert to sinus rhythm within 24 hours, even without treatment.[7] Less than 10% of patients discharged in sinus rhythm develop AF recurrence within six weeks of discharge.[20] This supports the merit of a more conservative approach locally, especially with haemodynamic stability and low ventricular response. Simple treatment measures such as inotrope reduction, fluid balance management and electrolyte correction (potassium and magnesium) must not be underrated. Anaemia, hypoxia, pain and patient arousal must be addressed as well.

For patients who are haemodynamically unstable, have rapid ventricular responses or features of myocardial ischaemia, initial electrical cardioversion followed by intravenous amiodarone has proved to be a useful strategy locally. We have adopted a rhythm-control strategy as per international trends. A study by Lee *et al.* showed a decreased time to cardioversion, prolonged maintenance of sinus rhythm and decreased overall hospital stay when a rhythm-control strategy was adopted.[21]

In our study, electrical cardioversion was successful only as an isolated modality in nine patients and this is perhaps partially explained by the exclusive use of antero-lateral pad placement.[2] Direct-current cardioversion is recommended as first-line therapy if AF causes haemodynamic instability or ischaemia. The initial shock energy should start with 300–360 J of monophasic waveform or 200 J of biphasic waveform and results in more than 95% success rate in converting to sinus rhythm.[22]

Amiodarone is the preferred anti-arrythmic agent in our setting as it is readily available, has anti-arrythmic efficiency similar to class I agents, can be used in patients with low ejection fractions, has no pro-arrhythmic tendency and is easily converted to oral medication. Intravenous amiodarone leads to sinus conversion in up to 90% of patients within the first 24 hours.[7]

In our study, amiodarone was administered as an intravenous loading dose (300 mg in 200 ml of 5% dextrose water over 45 minutes, followed by 900 mg in one litre of 5% dextrose water over 24 hours) during the first 24 hours of AF onset after failed electrical cardioversion. This was converted to oral agents (300 mg tds) on post-operative day two (or soonest possible) and weaned off gradually in the subsequent weeks. Rho suggests the continuation of amiodarone for a minimum of one week post surgery since the occurrence of AF beyond day seven is rare.[4] Amiodarone has proved to be effective in controlling heart rate in the post-operative period and the intravenous preparation is associated with improved haemodynamic status.[23]

Prophylactic amiodarone has inconsistently proved to effect a reduction in post-operative AF. Several trials evaluating the benefit of prophylactic amiodarone included patients concomitantly treated with β-blockers. Mahoney *et al.* showed that intravenous amiodarone is not cost effective for AF prevention if administered to all patients.[15]Anti-arrhythmic agents other than amiodarone used for the treatment of AF in the study included atenolol, digoxin and diltiazem. These agents are used for rate control in haemodynamically stable patients. The effect of these agents on AF is undoubtedly commensurate with the care with which they are used.

Short-acting β-blockers are the therapy of choice for rate control, especially in ischaemic heart disease, but may be poorly tolerated in asthmatics and patients in cardiac failure.[3] A meta-analysis of 24 randomised, controlled trials by Andrews *et al.* demonstrated a 77% reduction in AF post CABG.[9] The protective effect of pre-operative β-blocker therapy is related to the blunting of the high sympathetic tone occurring after cardiac surgery.[16] Findings of the AFIST II trial suggested that the concomitant use of β-blockers and amiodarone is especially rewarding.[24] Dunning *et al.* recommended β-blockers for the prevention of AF in all patients undergoing cardiac surgery.[2] Pre-operative β-blocker withdrawal is a significant risk factor for AF and must be avoided.[5]

Digoxin is grossly inefficient when adrenergic tone is high and is selectively used in patients with reduced ejection fractions.[3] AV nodal blocking agents such as the non-dihydropyridine calcium channel blockers can be alternatively used for rate control but may cause low cardiac output. These agents must be used cautiously until additional data on their safety profile becomes available.[3]

Historically, several modalities for AF prevention have been used with varying results. These include β-blockers, amiodarone, digoxin, bi-atrial pacing, calcium channel blockers, magnesium, statins, N-3 polyunsaturated fatty acids (PUFAs) and anti-inflammatory agents.[3]

We do not use any AF prophylaxis strategy in our unit. There are no robust risk models or evidence available to govern such a strategy. Moreover, prophylaxis has not been clearly shown to positively impact on morbidity or mortality arising from AF, and we are unaware of any feasibility studies supporting a prophylaxis strategy in resource-constrained environments.[3] The optimal anti-arrhythmic agent, dose, timing of initiation, and route and duration of drug administration for prophylaxis remain elusive.

β-blockers are used liberally for our cardiac surgical patients, mainly for coronary artery disease, as evidenced in this cohort, barring contra-indications. We are currently content to continue β-blocker use in the peri-operative period and enjoy whatever consequential AF reduction it may confer.

For the treating surgeon in the developing world, AF is a harbinger of increased LOS and resultant cost burdens.[4,11,14] The patients in our cohort took 4.6 days more than the institutional norm to return to the general ward. The cost implication is likely to be significant in light of the strain imposed on intensive-care and high-care wards.

In a study in the United States of America conducted by Aranki *et al.*, LOS was increased by 4.9 days, with additional hospital costs amounting $10 000–11 500.[11] AF results in longer ICU and overall hospital stays, even after adjusting for severity of illness.[7] Tamis *et al.* showed an increase in LOS of 3.2 days independent of variables.[25] It is, however, possible that the RGW status in our cohort was affected by other disease processes unrelated to AF.

Statins have been observed to attenuate inflammation and reduce AF post coronary surgery.[26] The ARMYDA-3 trial was a prospective, randomised study that showed atorvastatin 40 mg daily, commenced seven days prior to elective surgery on cardiopulmonary bypass and continued in the post-operative period, reduced the incidence of AF by 61%.[27]

An exciting aspect of *de novo* AF post cardiac surgery not addressed by this study requires discussion; the role of cardiac biomarkers and risk-prediction models in the prediction of post-operative AF. Studies by Gasparovic *et al.* and Pilatis *et al.*

Table 3. Risk factors for AF

Pre-operative
Advanced age
Male gender
Hypertension
Previous AF
History of previous cardiac surgery
Congestive heart failure (CHF)
Chronic obstructive pulmonary disease (COPD)
Right coronary artery (RCA) disease
Peripheral vascular disease
Left ventricular hypertrophy (LVH)
Left atrial enlargement
Electrocardiographic features
Renal failure
Moderate or severe aortic atherosclerosis
Withdrawal of β-blocker or ACEI
Body surface area (BSA)
Obesity and metabolic syndrome
Intra-operative
Aortic cross-clamp time
Bicaval canulation
Pulmonary vein venting
Type of surgery
Need of perioperative IABP
CPB time
CPB inclusive of cardioplegic arrest
Systemic hypothermia
Post-operative
Respiratory compromise
Red cell transfusion

showed elevated B-type natriuretic peptide (BNP) levels to be predictive of AF in patients undergoing CABG.[28,29] The utility of biomarkers in the setting of AF post cardiac surgery requires further clarification prior to a recommendation on their use but preliminary studies certainly show promise for AF prediction and thromboembolic risk stratification.

With regard to risk-prediction models for the development of AF, several have been developed and incorporate risk factors, some of which are mentioned in Table 3. These models have thus far provided controversial and inconsistent results, which have limited their widespread adoption. However, studies by Chua *et al*. and Baker *et al*. demonstrated the $CHADS_2$ and CHA_2DS_2-VASc scoring systems to be predictive of AF post cardiac surgery.[30,31] The limitations of these studies are that they were retrospective in nature, the sample size was small, and the patient population was heterogeneous. A recent study by Sareh *et al*. showed the $CHADS_2$ score to be a powerful and convenient predictor of post-operative AF in a cohort of 2 120 patients.[32]

A large, prospective, multicentric trial will provide a definite answer as to whether the $CHADS_2$ and the CHA_2DS_2-VASc scoring systems reliably predict post-operative AF. Should this be proved to be so, physicians will be guided to develop an effective prophylaxis strategy, including drugs and perhaps even prophylactic ligation of the left atrial appendage for 'high-risk' patients.

Limitations of this study are: it described only post-operative patients developing AF and an analysis of a control group was not undertaken. Furthermore, the cohort number was small relative to other similar studies conducted internationally.

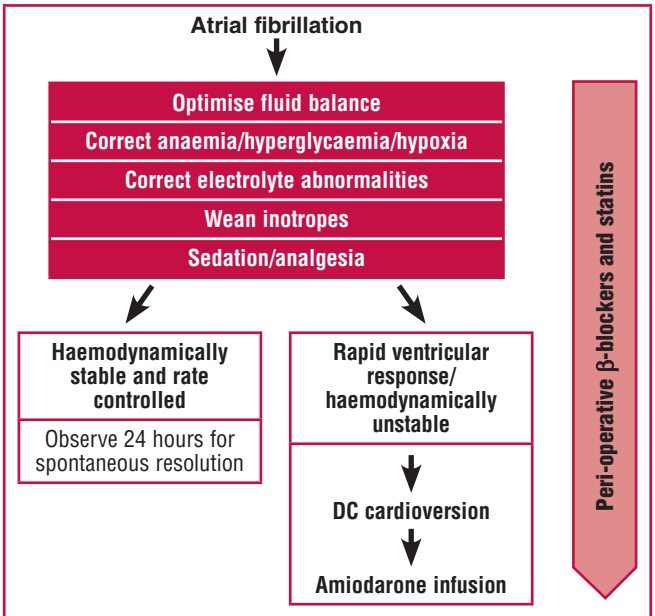

Fig. 3. Proposed strategy for the immediate treatment of AF post cardiac surgery.

Conclusions

This study serves to add to the growing body of information regarding *de novo* AF post cardiac surgery and provides some insight into the problem in developing countries. We propose a simple algorithm, shown in Fig. 3, for the immediate post-operative treatment of AF. Experience locally appears to mirror that of international cardiac surgical units. The aetiopathogenesis of AF is complex and a plethora of risk factors have been proposed (Table 3).[33]

Use of the $CHADS_2$ and CHA_2DS_2-VASc scoring systems and cardiac biomarkers as AF predictors appear promising. Liberal peri-operative β-blocker and statin administration is currently highly recommended. AF prophylaxis for the elderly, obese Indian male undergoing coronary surgery locally requires validation. Opportunistic surveillance for AF is advised at follow-up cardiology visits. Well-designed prospective studies are required for the better understanding and treatment of this common post-operative complication locally. The developing world should concentrate study efforts on LOS and cost reduction.

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Review Article

South African hypertension practice guideline 2014

Hypertension guideline working group: YK Seedat, BL Rayner, Yosuf Veriava

Abstract

Outcomes: Extensive data from many randomised, controlled trials have shown the benefit of treating hypertension (HTN). The target blood pressure (BP) for antihypertensive management is systolic < 140 mmHg and diastolic < 90 mmHg, with minimal or no drug side effects. Lower targets are no longer recommended. The reduction of BP in the elderly should be achieved gradually over one month. Co-existent cardiovascular (CV) risk factors should also be controlled.

Benefits: Reduction in risk of stroke, cardiac failure, chronic kidney disease and coronary artery disease.

Recommendations: Correct BP measurement procedure is described. Evaluation of cardiovascular risk factors and recommendations for antihypertensive therapy are stipulated. Lifestyle modification and patient education are cornerstones of management. The major indications, precautions and contra-indications are listed for each antihypertensive drug recommended. Drug therapy for the patient with uncomplicated HTN is either mono- or combination therapy with a low-dose diuretic, calcium channel blocker (CCB) and an ACE inhibitor (ACEI) or angiotensin receptor blocker (ARB). Combination therapy should be considered *ab initio* if the BP is ≥ 160/100 mmHg. In black patients, either a diuretic and/or a CCB is recommended initially because the response rate is better compared to an ACEI. In resistant hypertension, add an alpha-blocker, spironolactone, vasodilator or β-blocker.

Validity: The guideline was developed by the Southern African Hypertension Society 2014©.

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Department of Medicine, University of KwaZulu-Natal, Durban, South Africa

YK Seedat, MD, PhD, FRCP (London), FCP (SA)

Department of Medicine, University of Cape Town, Cape Town, South Africa

BL Rayner, MB ChB, FCP (SA), MMed (UCT), PhD (UCT), brian.rayner@uct.ac.za

Yosuf Veriava, MB BCh (Wits), FCP (SA), FRCP (London), Hon PhD (Wits)

This is the sixth hypertension guideline published by the Southern African Hypertension Society (SAHS). Currently 30.4% of the adult population have hypertension (HTN),[1] necessitating a simplified approach to assessment and treatment, which reflects realistic objectives that can be implemented by medical practitioners, nurse practitioners and pharmacists to diminish the impact of HTN and related cardiovascular disease (CVD) risk in this country. For full details on management not contained in this document please refer to the more detailed hypertension guideline 2011.[2]

Objective

The objective of this guideline was to promote evidence-based, accessible and comprehensive management of HTN by healthcare professionals in the public and private sectors. Applicable HTN and CVD treatment and prevention guidelines were reviewed as well as HTN trials reporting clinical end-points, including those with individuals with important co-morbidities such as diabetes mellitus and chronic kidney disease.[3-9]

Definition and grading of hypertension

HTN is defined as a persistent elevation of office blood pressure (BP) ≥ 140/90 mmHg (Table 1). The optimal BP is a value < 130/85 mmHg. High normal is BP levels from 130–139 mmHg systolic and 85–89 mmHg diastolic. This high-normal group of subjects is at higher CV risk and is also at risk of developing HTN, but does not require drug treatment.[10] HTN is stratified into three grades depending on severity, which is useful in defining the approach to treatment.

Measurement of blood pressure

BP measurement is a vital clinical sign that is poorly performed by all healthcare professional categories. These recommendations

Table 1. Definitions and classification of office BP (mmHg). Adapted from ref 9

Stage	*Systolic BP (mmHg)*	*Diastolic BP (mmHg)*
Normal	< 120	< 80
Optimal	120–129	80–84
High normal	130–139	85–89
Grade 1	140–159	90–99
Grade 2	160–179	100–109
Grade 3	≥ 180	≥ 110
Isolated systolic	≥ 140	< 90

BP should be categorised into the highest level of BP whether systolic or diastolic.

Table 2. Recommendations for blood pressure measurement

Allow patient to sit for 3–5 minutes before commencing measurement
The SBP should be first estimated by palpation to avoid missing the auscultatory gap
Take two readings 1–2 minutes apart. If consecutive readings differ by > 5 mm, take additional readings
At initial consultation measure BP in both arms, and if discrepant use the higher arm for future estimations
The patient should be seated, back supported, arm bared and arm supported at heart level
Patients should not have smoked, ingested caffeine-containing beverages or food in previous 30 min
An appropriate size cuff should be used: a standard cuff (12 cm) for a normal arm and a larger cuff (15 cm) for an arm with a mid-upper circumference > 33 cm (the bladder within the cuff should encircle 80% of the arm)
Measure BP after 1 and 3 minutes of standing at first consultation in the elderly, diabetics and in patients where orthostatic hypotension is common
When adopting the auscultatory measurement use Korotkoff 1 and V (disappearance) to identify SBP and DBP respectively
Take repeated measurements in patients with atrial fibrillation and other arthythmias to improve accuracy

apply to both clinic and self-measurement of BP. Failure to follow these guidelines leads to significant errors in BP measurement. BP should be recorded using an approved and calibrated electronic device or mercury sphygmomanometer (Table 2). Repeat measurements should be performed on at least three separate occasions within four weeks unless BP is ≥ 180/110 mmHg.

Self- and ambulatory measurement of BP

Self BP measurement (SBPM) and ambulatory BP measurement (ABPM) are recommended in selected circumstances and target groups:[11]

- suspected white-coat HTN (higher readings in the office compared with outside) or masked HTN (normal readings in office but higher outside)
- to facilitate diagnosis of HTN
- to guide antihypertensive medication, especially in high-risk groups, e.g. elderly, diabetics
- refractory HTN
- to improve compliance with treatment (SBPM only).

Masked HTN should be suspected if, despite a normal BP in the clinic, there is evidence of target-organ damage.

All devices used for SBPM and ABPM should be properly validated in accordance with the following independent websites: or http://afssaps.sante.fr.

In general, only upper-arm devices are recommended, but these are unsuitable in patients with sustained arrhythmias. For SBPM the patient should take two early morning and two late afternoon/early evening readings over five to seven days, and after discarding the first day readings, the average of all the remaining readings is calculated.

Wrist devices are recommended only in patients whose arms are too obese to apply an upper arm cuff. The wrist device needs to be held at heart level when readings are taken.

The advantages of SBPM measurement are an improved assessment of drug effects, the detection of causal relationships between adverse events and blood pressure response, and possibly, improved compliance. The disadvantages relate to increased patient anxiety and the risk of self-medication.

Table 3. Definitions of hypertension by different methods of BP measurement

	Office	*Automated office*	*Self*	*Ambulatory*
Predicts outcome	+	++	++	+++
Initial diagnosis	Yes	Yes	Yes	Yes
Cut-off BP (mmHg)	140/90	Mean 135/85	135/85	Mean day 135/85 Mean night 120/70
Evaluation of treatment	Yes	Yes	Yes	Limited, but valuable
Assess diurnal variation	No	No	No	Yes

ABPM provides the most accurate method to diagnose HTN, assess BP control and predict outcome.[12] Twenty-four-hour ABPM in patients with a raised clinic BP reduces misdiagnosis and saves costs.[13] Additional costs of ABPM were counterbalanced by cost savings from better-targeted treatment. It can also assess nocturnal BP control and BP variability, which are important predictors of adverse outcome. However the assessment is limited by access to ABPM equipment, particularly in the public sector, and impracticalities of regular 24-hour ABPM monitoring.

The appropriate cut-off levels for diagnosis of HTN by SBPM and ABPM are listed in Table 3.[11]

Automated office BP measurement

Despite efforts to promote proper techniques in manual BP measurement, it remains poorly performed. Automated office BP measurement offers a practical solution to overcome the effects of poor measurement, bias and white coating.[14] It is more predictive of 24-hour ABPM and target-organ damage than manual office BP measurement. Six readings are taken at two-minute intervals in a quiet room. The initial reading is discarded and the remaining five are averaged. The appropriate cut-off level for HTN is 135/85 mmHg.[14]

CVD risk stratification

The principle of assessing and managing multiple major risk factors for CVD is endorsed. However, because the practical problems in implementing previous recommendations based on the European Society of HTN (ESH) and the European Society of Cardiology (ESC) HTN guidelines, it has been decided to use a modification of this approach.[9]

Once the diagnosis of HTN is established, patients with BP ≥ 160/100 mmHg should commence drug therapy and lifestyle modification. Patients with stage 1 HTN should receive lifestyle modification for three to six months unless they are stratified as high risk by the following criteria: three or more major risk factors, diabetes, target-organ damage or complications of HTN (Table 4).

Routine baseline investigations

Table 5 lists recommended routine basic investigations. The tests are performed at baseline and annually unless abnormal. Abnormal results must be repeated as clinically indicated.

Table 4. Major risk factors, target-organ damage (TOD) and complications. Adapted from the ESH/ESC guidelines[9]

Major risk factors
- Levels of systolic and diastolic BP
- Smoking
- Dyslipidaemia:
  - total cholesterol > 5.1 mmol/l, OR
  - LDL > 3 mmol/l, OR
  - HDL men < 1 and women < 1.2 mmol/l
- Diabetes mellitus
- Men > 55 years
- Women > 65 years
- Family history of early onset of CVD:
  - Men aged < 55 years
  - Women aged < 65 years
- Waist circumference: abdominal obesity:
  - Men ≥ 102 cm
  - Women ≥ 88 cm
  - The exceptions are South Asians and Chinese: men: > 90 cm and women: > 80 cm.

TOD
- LVH: based on ECG
  - Sokolow-Lyons > 35 mm
  - R in aVL > 11 mm
  - Cornel > 2 440 (mm/ms)
- Microalbuminuria: albumin creatine ratio 3–30 mg/mmol preferably spot morning urine and eGFR > 60 ml/min

Complications
- Coronary heart disease
- Heart failure
- Chronic kidney disease:
  - macroalbuminuria > 30 mg/mmol
  - OR eGFR < 60 ml/min
- Stroke or TIA
- Peripheral arterial disease
- Advanced retinopathy:
  - haemorrhages OR
  - exudates
  - papilloedema

Goals of treatment

There has been considerable controversy about BP goals and SAHS accepts that to simplify management, a universal goal of antihypertensive treatment is < 140/90 mmHg regardless of CV risk and underlying co-morbidities.[5] The only exception is that in patients over 80 years of age, therapy should be initiated if SBP is > 160 mmHg and the goal is between 140 and 150 mmHg, based on the HYVET study in which the majority of patients received indapamide and the ACEI perindopril.[15]

SAHS does not support the JNC-8 committee recommendations of a goal BP < 150/90 mmHg for persons over 60 years without diabetes and CKD, as (1) increasing the target will probably reduce the intensity of antihypertensive treatment in a large population at high risk for cardiovascular disease, (2) the evidence supporting increasing the SBP target from 140 to 150 mmHg in persons aged 60 years or older was insufficient, (3) the higher SBP goal in individuals aged 60 years or older may reverse the decades-long decline in CVD, especially stroke mortality.[8,16]

It is also essential to control hyperlipidaemia and diabetes through lifestyle and drug therapy, according to the Society for Endocrine Metabolism Diabetes of South Africa and South African Heart Association/Lipid and Atherosclerosis Society of Southern Africa guidelines, respectively.[17,18] Aspirin should not be routinely prescribed to hypertensives (especially if BP is not controlled),[19] and should mainly be used for secondary prevention of CVD (transient ischaemic attack, stroke, myocardial infarction).

Table 5. Routine investigations

Test	*Comment*
Height, weight, BMI	Ideal BMI < 25 kg/m², overweight 25–30 kg/m², obese > 30 kg/m²
Waist circumference	Men < 102 cm; women < 88 cm. South Asians and Chinese: men < 90 cm and women < 80 cm
Electrolytes	Low potassium may indicate primary aldosteronism, or effects of diuretics
ECG	S in V1 plus R in V5 or V6 > 35 mm or R in aVL > 11 mm or Cornel product (R in aVL + S in V3 + 6 in females) × QRS duration > 2 440 (mm/ms)
Echocardiogram (if indicated and facilities available)	LVH: men > 115 g/m² and women > 95 g/m²
Fasting glucose	Consider HBA_{1c} or GTT if impaired fasting glucose (6.1–7.1 mmol/l)
Cholesterol	If total cholesterol > 5.1 mmol/l – fasting lipogram
Creatinine	Calculate eGFR
Uric acid	High uric acid is relative contraindication to diuretics
Dipsticks urine	If abnormal, urine microscopy and protein estimation

Management of hypertension

All patients with HTN should receive lifestyle counselling as outlined in Table 6, and this is the cornerstone of management. The approach to drug treatment is outlined in Fig. 1. *If the SBP is ≥ 180 mmHg or the DBP is ≥ 110 mmHg then refer to section 8 on severe (grade 3) HTN, as this section does not apply.*

Before choosing an antihypertensive agent, allow for considerations based on the cost of the various drug classes, patient-related factors, conditions favouring use and contra-indications, complications and target-organ damage (TOD) (Tables 4, 7).

In otherwise uncomplicated primary HTN, the initial first choice of antihypertensive drug is a diuretic (thiazide-like or thiazide), ACEI or ARB, and/or CCB used as mono- or combination therapy (Fig. 2). Combination therapy should be considered if clinically appropriate *ab initio* if BP is ≥ 160/100 mmHg (Fig. 1) as this is associated with better clinical outcomes and earlier achievement of goal BP.[20,21] Fixed-drug combinations are preferred because of better patient adherence and control of BP.[22] A treatment algorithm is outlined in Fig. 1 if the goal is not reached after initial treatment.

Table 6. Recommended lifestyle changes

Modification	*Recommendation*	*Approx ↓ SBP (mmHg)*
Weight reduction	BMI 18.5–24.9 kg/m²	5–20 per 10 kg
Dash diet	↓ saturated fat and total fat, ↑ fruit and vegetables	8–14
Dietary Na^+	< 100 mmol or 6 g NaCl/day	2–8
Physical activity	Brisk walking for 30 minutes per day most days	4–9
Moderation of alcohol	No more than two drinks per day	2–4
Tobacco	Complete cessation	–

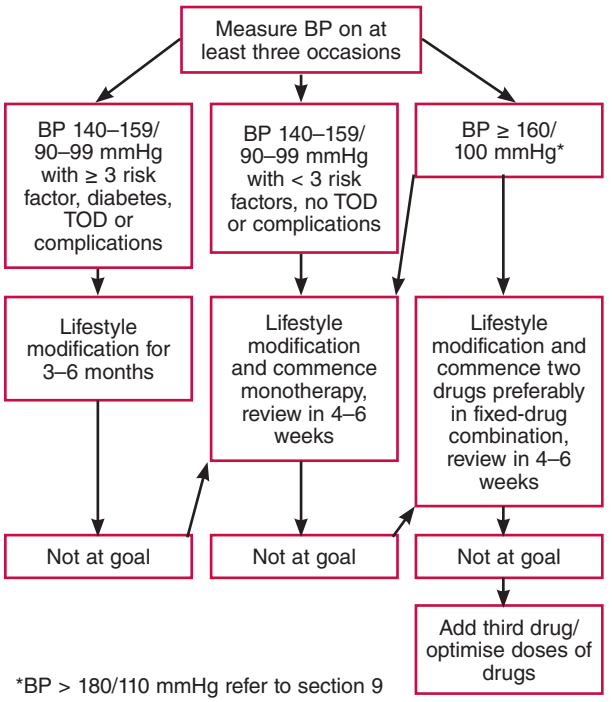

Fig. 1. Overview of approach to treatment.

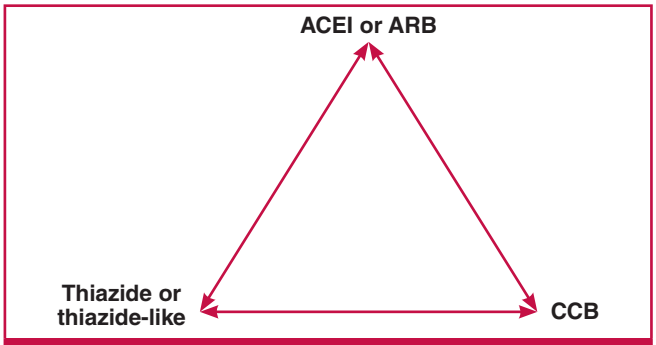

Fig. 2. Initial choices of antihypertensive treatment or combinations.

In black hypertensive patients a diuretic and/or a CCB is recommended.[23] Beta-blockers should generally be avoided in combination with diuretics as first-line therapy because of predisposition to diabetes,[9] but this may not apply to highly selective beta-blockers. Beta-blockers may also be considered if there is intolerance to one of the first-line drugs. Loop diuretics such as furosemide should not be used because of their short duration of hypotensive activity of about six hours, unless there is evidence of chronic kidney disease (CKD) with estimated glomerular filtration rate (GFR) < 45 ml/min.

Table 7. Indications and contra-indications for the major classes of antihypertensive drugs. Adapted from the ESC/ESH guidelines[9]

		Contra-indications	
Class	*Conditions favouring the use*	*Compelling*	*Possible*
Diuretics (thiazide; thiazide-like)	• Heart failure(HF) • Elderly hypertensives • Isolated systolic HTN (ISH) • Hypertensives of African origin	• Gout	• Pregnancy • β-blockers (especially atenolol)
Diuretics (loop)	• Renal insufficiency • HF		• Pregnancy
Diuretics (anti-aldosterone)	• HF • Post-myocardial infarction • Resistant hypertension	• Renal failure • Hyperkalaemia	
CCB (dihydropyridine)	• Elderly patients • ISH • Angina pectoris • Peripheral vascular disease • Carotid atherosclerosis • Pregnancy		• Tachyarrhythmias • HF especially with reduced ejection fraction
CCB non-dihydropyridine (verapamil, diltiazem)	• Angina pectoris • Carotid atherosclerosis • Supraventricular tachycardia	• AV block (grade 2 or 3) • HF	• Constipation (verapamil)
ACEI	• HF • LV dysfunction • Post-myocardial infarction • Non-diabetic nephropathy • Type 1 diabetic nephropathy • Prevention of diabetic microalbuminuria • Proteinuria	• Pregnancy • Hyperkalaemia • Bilateral renal artery stenosis • Angioneurotic oedema (more common in blacks than in Caucasians)	
ARB	• Type 2 diabetic nephropathy • Type 2 diabetic microalbuminuria • Proteinuria • LVH • ACEI cough or intolerance	• Pregnancy • Hyperkalaemia • Bilateral renal artery stenosis	
β-blockers	• Angina pectoris • Post-myocardial infarction • HF (carvedilol, metoprololol, bisoprolol, nebivolol only) • Tachyarrhythmias	• Asthma • Chronic obstructive pulmonary disease • AV block (grade 2 or 3) • Pregnancy (atenolol)	• Peripheral vascular disease • Bradycardia • Glucose intolerance • Metabolic syndrome • Athletes and physically active patients • Non-dihydropyridine CCBs (verapamil, diltiazem)

Management of severe hypertension

Patients with severe HTN (grade 3; BP ≥ 180/110 mmHg) may fall into one of three categories, which determine the urgency of their treatment. Patients should be managed or referred to the appropriate level of care and caregiver in accordance with local resources. Sustained, severe HTN requires immediate drug therapy and lifestyle modification, and close follow up.

Asymptomatic severe hypertension

These patients are asymptomatic but have severe HTN without evidence of progressive TOD or complications. The patient must be kept in the care setting and BP measurement repeated after resting for one hour. If still elevated at the same level, commence oral therapy using two first-line drugs. Follow up within a week or earlier, with escalation of treatment as needed. Early referral is advised if BP is not controlled within two to four weeks.

Hypertensive urgencies and emergencies[24]

While not common, hypertensive emergencies and urgencies are likely to be encountered by all clinicians because of the high prevalence of chronic HTN. It is essential that all professionals are familiar with treatment. There is a paucity of information from well-conducted studies on the outcomes of various antihypertensive drugs and BP-lowering strategies.

Hypertensive urgency[25]

This level of HTN is symptomatic, usually with severe headache, shortness of breath and oedema. There are no immediate life-threatening neurological, renal, eye or cardiac complications, such as are seen in hypertensive emergencies. Ideally, all patients with hypertensive urgency should be treated in hospital.

Commence treatment with two oral agents and aim to lower the diastolic BP to 100 mmHg slowly over 48 to 72 hours. This BP lowering can be achieved with the use of: (1) long-acting CCBs; (2) ACEI, initially used in very low doses, but avoid if there is severe hyponatraemia (serum Na < 130 mmol/l indicates hyper-reninaemia and BP may fall dramatically with ACEI); (3) β- blockers; and (4) diuretics.

Hypertensive emergency

A hypertensive emergency is severe, often acute elevation of BP associated with acute and ongoing organ damage to the kidneys, brain, heart, eyes (grade 3 or 4 retinopathy) or vascular system. These patients need rapid (within minutes to a few hours) lowering of BP to safe levels. Hospitalisation is ideally in an intensive care unit (ICU) with experienced staff and modern facilities for monitoring. If an ICU is unavailable, the patient may be closely monitored and treated in the ward.

Intravenous antihypertensive therapy, tailored to the specific type of emergency, has become the standard of care. Labetalol, nitroprusside or nitroglycerin are the preferred intravenous agents. Overzealous lowering of BP may result in stroke. A 25% reduction in BP is recommended in the first 24 hours. Oral therapy is instituted once the BP is more stable. Although most adult patients with a hypertensive emergency will have BP > 220/130 mmHg, it may also be seen at modest BP elevations; for example, in a previously normotensive woman during pregnancy (eclampsia) or in the setting of acute glomerulonephritis, especially in children.

Severe HTN associated with ischaemic stroke and intracerebral haemorrhage should be managed according to the recommendations of the Neurological Association of South Africa.[26] Great caution should be exercised in lowering BP after an ischaemic stroke due to the risk of extending the ischaemic penumbra.

Resistant hypertension

HTN that remains > 140/90 mmHg despite the use of three antihypertensive drugs in a rational combination at full doses and including a diuretic (hydrochlorothiazide 25 mg or indapamide 2.5 mg) is known as resistant HTN. Common causes of resistant HTN are listed in Table 8.

The therapeutic plan must include measures to ensure adherence to therapy and lifestyle changes. Unsuspected causes of secondary HTN are less common, but need to be considered based on history, examination and special investigations. It is essential to exclude pseudo-resistance by performing SBPM or 24-hour ABPM. Referral to a specialist is often indicated for a patient with resistant HTN.

Table 8. Causes of resistant hypertension in South Africa

Non-adherence to therapy	• Instructions not understood • Side effects • Cost of medication and/or cost of attending at healthcare centre • Lack of consistent and continuous primary care • Inconvenient and chaotic dosing schedules • Organic brain syndrome (e.g. memory deficit)
Volume overload	• Excess salt intake • Inadequate diuretic therapy • Progressive renal damage (nephrosclerosis)
Associated conditions	• Smoking • Increasing obesity • Sleep apnoea • Insulin resistance/hyperinsulinaemia • Ethanol intake of more than 30 g (three standard drinks) daily • Anxiety-induced hyperventilation or panic attacks • Chronic pain • Intense vasoconstriction (Raynaud's phenomenon), arteritis
Identifiable causes of hypertension	• Chronic kidney disease • Renovascular disease • Primary aldosteronism • Coarctation • Cushing's syndrome • Phaeochromocytoma
Pseudoresistance	• 'Whitecoat hypertension' or office elevations • Pseudohypertension in older patients • Use of regular cuff in obese patients
Drug-related causes	• Doses too low • Wrong type of diuretic • Inappropriate combinations • Rapid inactivation (e.g. hydralazine)
Drug actions and interactions	• Non-steroidal anti-inflammatory drugs (NSAIDs) • Sympathomimetics: nasal decongestants, appetite suppressants • Cocaine, Tik and other recreational drugs • Oral contraceptives • Adrenal steroids • Liquorice (as may be found in chewing tobacco) • Cyclosporine, tacrolimus, erythropoietin • Antidepressants (monoamine oxidase inhibitors, tricyclics)

Once the issues relating to lifestyle, adherence to therapy, white coating, etc. outlined in Table 7 have been satisfactorily managed, then consideration should be given to the addition of the fourth- and fifth-line drug. Currently spironolactone (25–50 mg only) with careful monitoring of serum potassium, beta-blockers and/or long-acting doxazasin is recommended.[27,28] Other choices include direct-acting vasodilators (hydralazine, minoxidil), or centrally acting drugs (methyldopa, moxonidine, reserpine).

Initial studies of renal denervation in patients with resistant HTN showed very promising results.[29,30] The recent publication of the Simplicity 3 study showing no significant effect on BP compared to sham procedure, the place of renal denervation in the treatment of resistant HTN remains to be established and is not supported by this guideline.[31]

Special considerations for hypertension in certain populations

Blacks and Asians

Blacks are more prone to complications of stroke, heart failure and renal failure, while the incidence of coronary heart disease, although increasing in frequency, is less common compared with that in whites and Asians.[32] The prevalence of diabetes mellitus and the metabolic syndrome is higher in Asians compared to other racial groups.[33]

Compared to whites, blacks respond poorly to ACEI and β-blockers as monotherapy, but this difference disappears once these drugs are combined with diuretics. Overall, CCBs show the most consistent response in blacks compared to other classes of drugs used as monotherapy.[23,34] However there is a higher incidence of angioedema in blacks treated with an ACEI.[35]

Hypertension in children and adolescents[36,37]

HTN in children is an important issue beyond the scope of this guideline. In adolescents, the HTN is increasingly linked to obesity and affects up to 10% of people between the ages of 15 and 25 years.[38] The international trend of poor diet and lack of exercise in children is leading to an epidemic of obesity, with the early onset of HTN and even type 2 diabetes. The early recognition of HTN in these adolescents will be an important motivation for both children and parents to institute important lifestyle changes.

HIV/AIDS

There are an estimated 5.8 million people living with HIV in South Africa. The co-existence of HIV with HTN and diabetes is increasing, and patients should be screened for associated glomerulonephritis.[39] Prolonged highly active antiretroviral therapy (HAART) is associated with a higher prevalence of systolic HPT,[40] and it is essential that BP is monitored in patients receiving HAART.

Two of the three major classes of antiretroviral drug, the protease inhibitors and the non-nucleoside reverse transcriptase inhibitors, are involved in many drug interactions by inhibiting or inducing the key hepatic enzyme system, cytochrome P450. CCBs are the major class of antihypertensives affected by such drug interactions, leading to inhibition or induction of their metabolism.[41,42] This results in either an enhanced or loss of antihypertensive efficacy.

Disclaimer

This national clinical guideline is a reference and educational document. The SAHS accepts no responsibility or liability arising from any information contained in or any error of omission from the protocol or from the use of any information contained in it.

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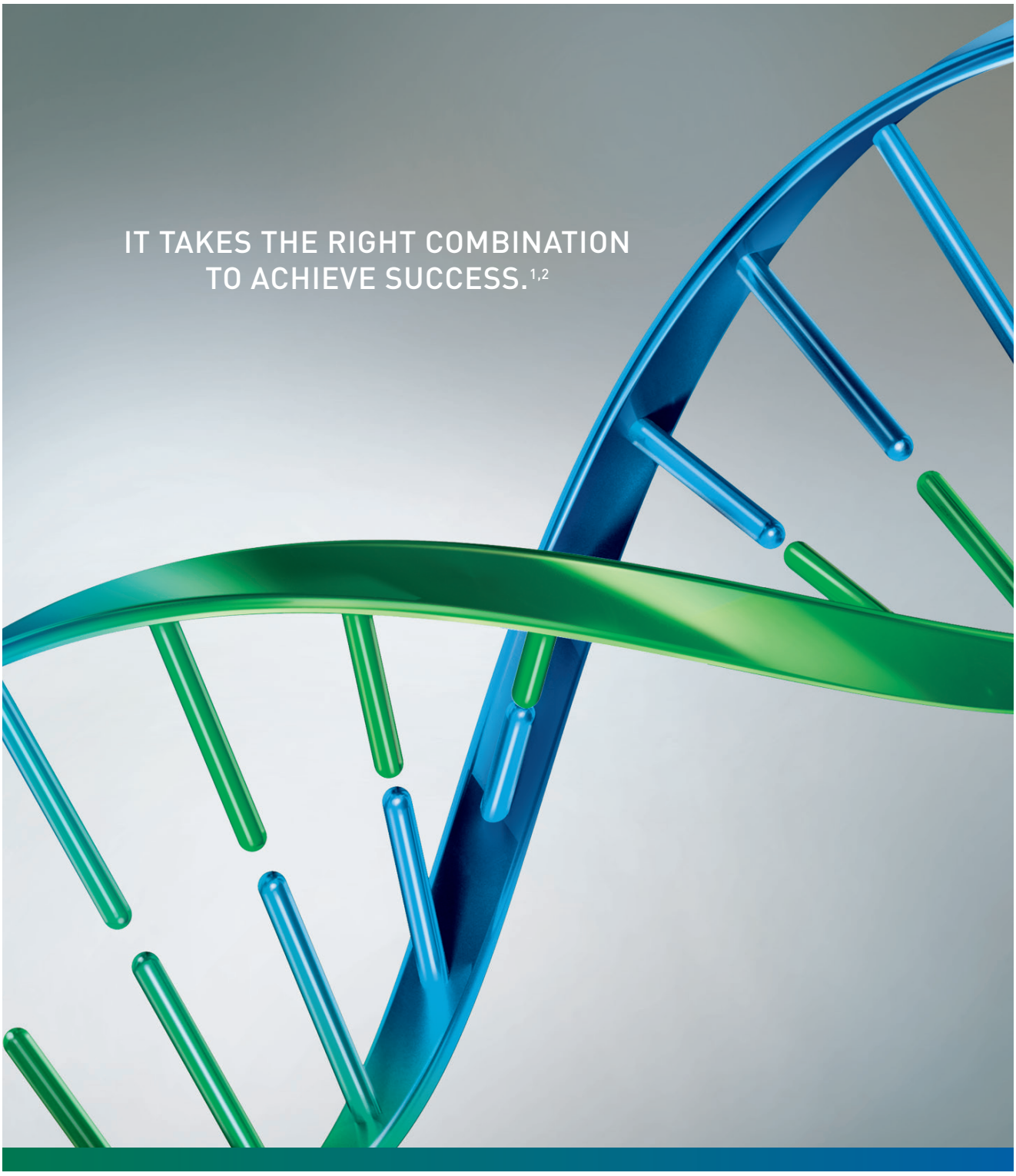

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Comment

The importance of guidelines

Erika SW Jones, Brian L Rayner

The management of chronic diseases crosses the line between primary healthcare and tertiary academic medicine. New technologies are constantly being developed and treatment options being better defined. This has resulted in the development of multiple guidelines[1-3] in order to standardise appropriate therapy for chronic diseases and to disseminate the information. Guidelines highlight current literature and new evidence, and they create an easy step-wise approach to the management of diseases, the targets for disease control and the standards of care.[4]

There is a growing prevalence of patients with hypertension[5] and diabetes mellitus.[6] The cardiovascular complications of these disorders are well documented (including ischaemic heart disease, heart failure, neuropathies, retinopathy, renal failure and stroke) and result in considerable morbidity and mortality. However, with good care, these complications can be decreased, controlled or prevented, limiting the adverse outcomes.[7]

It has been established that the quality of care provided in South Africa is inadequate to prevent these adverse outcomes. Hypertension and its sequelae account for three of the top 10 causes of death in South Africa.[8] This is because blood pressures are uncontrolled, there is poor glycaemic control and screening for complications is inadequate. Guidelines attempt to improve these issues. However, physician compliance with guideline recommendations needs to be addressed in order to improve the outcomes.

Reviews of the major trials in various chronic diseases, such as that by Okpechi and Rayner,[9] summarise the results of the trials but do not make the information practically available. Accessing reviews and applying them to clinical practice requires time and expertise, whereas guidelines are made easily available in their local setting for healthcare providers to peruse as and when needed.

Guidelines provide an easily accessible resource that clinicians can review to expand their knowledge base and determine patient care. This allows clinicians to be able to keep abreast of current knowledge despite the rapidly expanding knowledge that is being continuously developed. Health services and insurers can also access these guidelines to determine standards of care and medication recommendations. This can be the basis for essential drug lists.

The National High Blood Pressure Education Programme (NHBPEP) released their first guideline in 1977. This was the first in the series of hypertension management guidelines produced in the United States to improve blood pressure control and management. The production and implementation of these guidelines resulted in improved patient awareness of blood pressure and the complications that result. As a result of this awareness, people are more likely to visit their doctor for blood pressure checks, the most common reason for adults to visit their doctor.

The NHBPEP is responsible for improving blood pressure control and outcomes; age-adjusted mortality has declined by 70% for heart disease and by 80% for stroke over the four decades of its existence. There has been a steady decline in heart-related deaths over this time period, and malignant hypertension is rare in the USA.[10]

Implementing guidelines can be a difficult task and in some instances may not improve outcomes. A study in Cape Town in 1999[11] showed that the approach to treating hypertension and diabetes with guidelines did not improve blood pressure or glycated haemoglobin levels.

The implementation of the guidelines involved a multifaceted intervention. A structured record was designed and incorporated into the folder. This structured record was a three-sided folded A3 sheet with multiple components: patient details, medical history, referrals, educational topics, algorithms for hypertension and diabetes diagnosis and management, targets, treatment options, and a flow sheet for results. The intervention included an educational package to train the primary healthcare providers in the use of the guidelines.

Unfortunately this intervention did not improve blood pressure control or glycated haemoglobin levels. There are multiple reasons for this; the structured record was only found in 60% of the intervention folders and was generally not used when found in the folders. Other contributing factors include that this was a time when the healthcare system was being changed in South Africa by redistributing patients to primary care facilities. The changes did not include the badly needed increase in staffing. There was also a lack of budget to support the implementation of these guidelines, a lack of facilities within the primary healthcare services, and lack of time to provide the suggested care.

This study[11] highlights two importance aspects of guidelines and interventions. They need to be simple and suited to the environment in which they will be implemented; and in order to implement the guidelines, there needs to be the institutional infrastructure to be able to manage the recommendations.

The American Heart Association has highlighted the cost of hypertension[10] and the resultant cardiovascular complications in the USA. They have issued a science advisory in order to improve control. This document is an attempt to 'identify, disseminate, and implement more effective approaches to achieve optimal control'. They suggest that blood pressure requires a multifactorial approach, and the engagement of all potentially involved persons/health systems. They suggest that best-practice guidelines are essential in achieving the goals of blood pressure control and cost saving. This advisory considers that lack of control can be ascribed to fragmented healthcare services (a major problem in South Africa) and the poor implementation of health-system solutions at a clinical level, as seen by Steyn *et al*.[11]

An evidence-based treatment programme in Kaiser Permanante, northern California,[12] showed system-level success. This programme resulted in an increase in blood pressure control (44 to 80%) despite an increase in numbers of patients with hypertension; possibly due to better and earlier detection, which results independently in better treatment and control.

It is, however, important to recognise the necessity of individualising treatment. This approach was best seen in the physiologically based antihypertensive therapy as described by Spence.[13] Such management can, if successful, be included into guidelines where appropriate.

Guidelines are an essential tool in the care of chronic diseases. They provide a means to update and disseminate information and the standard of care to all health sectors. However, they are only as good as the clinicians who implement them, and the system that provides the infrastructure for their implementation. Furthermore, they need to be appropriate for the system in which they will be implemented.

Erika SW Jones, MB BCh, FCP, Cert Nephrol, PhD,
eswjones@gmail.com

Brian L Rayner, MB ChB, MMed, FCP, PhD
Hypertension Clinic, Groote Schuur Hospital, Cape Town, South Africa

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Review Article

Lifestyle and diet

Lionel H Opie

Abstract

Currently, there is widespread interest in many different diets. The best-known diets include the New Atkins diet in the USA, the Dukan diet in France, and in South Africa the Noakes diet. Two different approaches have emerged, one focusing on a life-long healthy lifestyle and the other emphasising weight loss. These are in fact complementary aims, as will be reviewed and reconciled. Furthermore, besides the dietary approach, there is a valid case for added drug therapy for selected lipid disorders with the use statins. In addition, new drugs are emerging that in the future might eventually considerably reduce the negative health impact of coronary artery disease.

Keywords: diet, cardiovascular risk, Noakes diet, Banting diet, Mediterranean diet

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Lifestyle is life-long

Life-style is life-long in its health implications.[1] Although diet is only one of the five components of a healthy lifestyle,[2] diet has recently come to the fore.[3] When considering overall health, the most important are non-smoking and regular exercise, followed by body weight and diet, in order of importance (Table 1). These proposals are based on a series of important studies on over 100 000 US health professionals over 10 to 25 years, which defined the contribution to health of four major lifestyle factors, only one of which is diet (Table 1).[2,4,5]

While there are many diets to choose from, the majority focusing on weight loss, few diets have had scientifically solid outcome studies to prove that the diet in question actually improves health and increases life span. An exception is the Mediterranean diet, so called because of the very low incidence of heart attacks observed by Ancel Keys in the Mediterranean islands of Corfu and Crete, thus leading to the concept that the Mediterranean diet is an ideal diet,[1,6,7] also protecting against heart failure.[8]

Hatter Institute for Cardiovascular Research in Africa, Groote Schuur Hospital and Medical School, University of Cape Town, Cape Town, South Africa
Lionel H Opie, MD, FRCP, Lionel.Opie@uct.ac.za

Palaeolithic, the oldest diet

What is the paleolithic diet? Mankind evolved over hundreds of millions of years, therefore the paleolithic diet must have been the standard diet that also evolved over that time. Studies on the teeth of the paleolithic man, as found in East Africa (also in its congener from South Africa), showed that the dental bones and teeth had adapted to process large quantities of low-quality vegetation rather than hard objects.[9] The paleolithic diet is now recognised as a nutritional pattern based on the ancient diet of wild plants and animals that our ancestors consumed over 10 000 years ago.

In the Kitava dietary study on isolated tribes in Papua, New Guinea, who even recently ate a pre-Westernised diet of 55 to 65% animal foods and 35 to 45% plant foods, these societies had no incidence of stroke, heart disease, diabetes or hypertension.[10] The diet consisted mainly of fish, grass-fed pasture-raised meats, vegetables, fruits, roots, spices and nuts. There was no restriction on calories or on the foods to be cooked.

Although the Mediterranean diet overlaps with the palaeolithic diet in terms of fibre, antioxidants, saturated fat and mono-unsaturated fat, the paleolithic diet improved glucose tolerance more than did the Mediterranean diet.[10] Furthermore, this diet is more food satiating than a Mediterranean-like diet in persons with ischaemic heart disease.[11] Therefore the paleolithic diet both preceded the Mediterranean diet and was apparently better, so it may be that 'the simpler, the better'.

Diet and lipids

Moving on in history, it was the early Cape Town studies that made the link between fat in the diet and blood cholesterol values. Nearly 60 years ago, Professors John Brock and Brian Bronte-Stuart from Groote Schuur and the University of Cape

Table 1. The 'big-five' components of the healthy lifestyle, with contributions of the various components to give protection from risk of death, with and the proposed mechanisms of action. Note that the missing 21% is probably stress related. From Opie,[1] page 33.

Lifestyle: 'big five'	*Reduced all-cause death risk (%)*	*Mechanism*
Non-smoking	28	Protects arteries
Exercise 30 min or more daily	17	Slows the heart rate, lowers BP
Ideal weight	14	Less toxic chemicals released from fat cells
Ideal diet	13	High unsaturated fatty acids, high vegetables and fruit, low red meat
Modest alcohol	7	Red wine preferred, contains melatonin
All five	79	Remaining 21% may be stress related

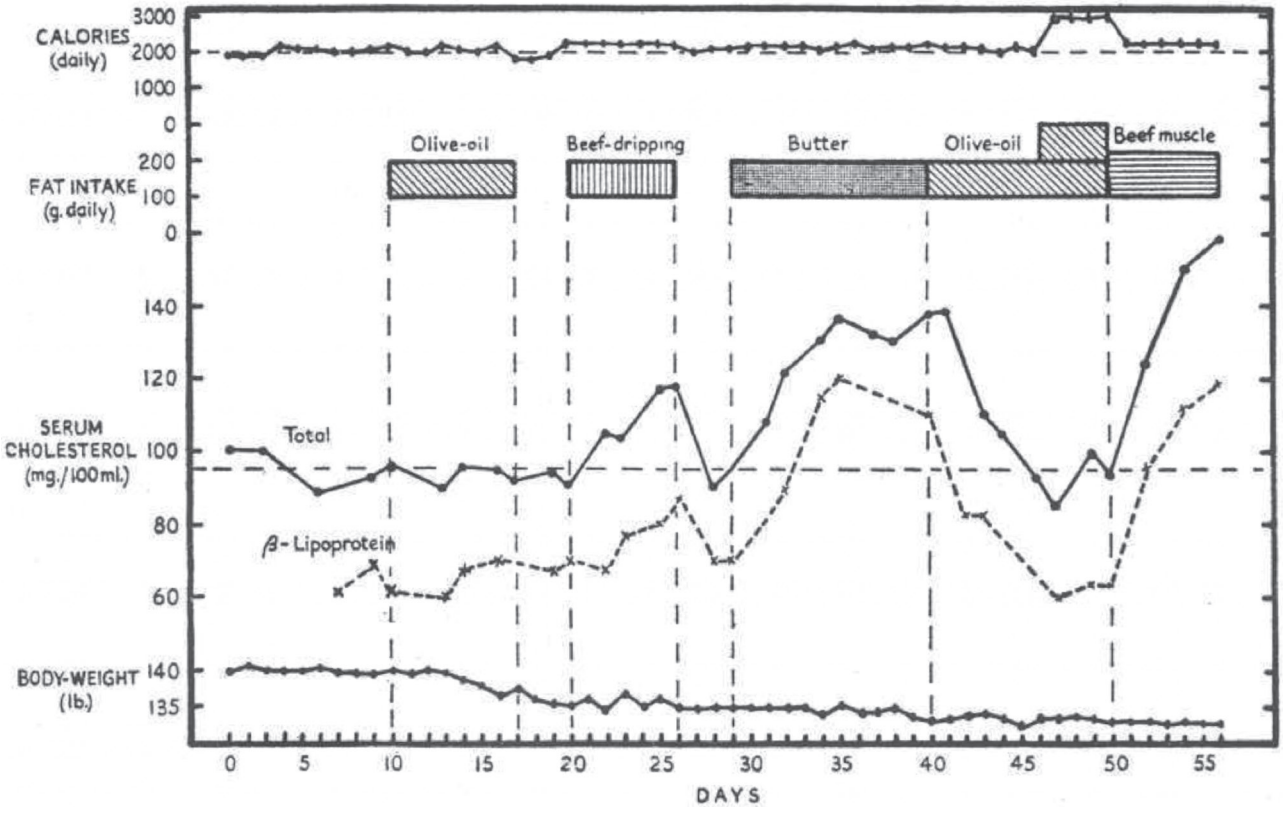

Fig 1. A historical study in Cape Town. The effect of dietary fats on blood lipid levels and their relation to ischaemic heart disease, neutralised by the effect of added olive oil. Note the rapid rise in serum cholesterol levels with the provision of the high-butter diet. All values were obtained in the Metabolic Unit, University of Cape Town, South Africa. From Bronte-Stewart.[14]

Town Department of Medicine used their specialised metabolic unit to give a high-fat diet to subjects with an initially low blood cholesterol level (Fig. 1).[12,13] A butter load of 100 grams given daily increased blood cholesterol by proximately 40% within five days. The addition of large amounts of olive oil to the butter load restored cholesterol levels to their prior low levels (Fig. 1). Therefore the type of fat diet affected blood cholesterol levels.

The decisive further link between circulating cholesterol values and coronary heart disease came from the Framingham study, which found that higher blood cholesterol values were associated with increased cardiovascular and total mortality rates.[15] Over time, the emphasis on selection of drug therapy via statins has shifted to the blood level of low-density lipoprotein (LDL) cholesterol.[16]

In South Africa in 2000, high blood cholesterol levels have been estimated to have caused 24 144 deaths (95% CI: 22 404–25 286) or 4.6% of all deaths.[17] Studies in the Cape Peninsula and in the South African Indian population support links between lipid abnormalities and coronary heart diseases.[18,19] Severely obese South African white women have greatly reduced values for serum high-density lipoprotein (HDL) cholesterol or 'good' cholesterol, rather than high levels of LDL cholesterol.[20]

Lipids in diabetes: the role of statins

The ideal approach to nip diabetes in the bud is by testing HbA_{1c} values in those with the metabolic syndrome or obesity, and then to go for weight loss induced by combined diet and exercise. In those with established type 2 diabetes (DM2), a population study in Hong Kong suggested that statin therapy attenuated the associated increased cancer risk.[21] For diabetes, in a large study with 215 725 person-years of follow up, statin use before the diagnosis of diabetes reduced diabetic retinopathy (hazard ratio 0.60, 95% CI: 0.54–0.66; $p < 0.0001$), diabetic neuropathy (HR 0.66, 95% CI: 0.57–0.75; $p < 0.0001$), and gangrene of the foot (HR 0.88, 95% CI: 0.80–0.97; $p = 0.010$).[22] Regarding the general adult population, statins are recommended as first-line therapy in those up to and including 75 years of age, who have clinical atherosclerotic cardiovascular disease (ASCVD) (Table 4 in Stone *et al.*[23]).

Exercise versus drugs

In studies on the secondary prevention of coronary heart disease and pre-diabetes, randomised trials on exercise interventions suggest that exercise and many drug interventions are often potentially similar in terms of their mortality benefits, rehabilitation after stroke, treatment of heart failure, and prevention of diabetes.[24] This important observation reinforces the essential role of exercise in any programme aimed at overall cardiovascular health (Table 1).

Banting first linked diet to mortality

Banting in his pamphlet[25] in 1869 emphasised the role of diet in weight loss, stating that: 'The dietary is the principle point in

the treatment of corpulence.' The key points in the Banting diet were his method of reducing obesity by avoiding fat, starch and sugar in the food. Therefore the proposal that the Banting diet is similar to the Noakes high-fat diet[3] appears to need re-appraisal. Banting also made wider overall claims that the diet was 'a simple remedy to reduce and destroy superfluous fat; it may alleviate if not cure gout; prevent or eradicate carbuncles, boils, dyspepsia, makes life more enjoyable, and promotes longevity'. One interesting small but important point is that Banting took the fat off the gravy. For these reasons, it seems preferable to separate the Banting diet from the Noakes low-carbohydrate, high-fat diet.

Israeli study and new Atkins diet

The low-carbohydrate, high-fat diets that were introduced by Atkins and his successors[26] have had very wide influence. Some of the key features are as follows, with the relevant book pages given in brackets:

- Protein intake though high has recommended protein ranges (51).
- Fat intake though also high, has a desirable range (70).
- Vegetables including avocadoes are the basis of the permitted carbohydrate intake (102).

In a major landmark Israeli diet, the new Atkins diet was compared with others from the same Israeli population group in a dedicated communal restaurant where the food intake could be monitored.[27] In the group given the new Atkins diet, besides weight loss, the blood cholesterol pattern showed some favourable changes.

In the comparative group taking a calorie-limited Mediterranean diet, similar changes were found in weight loss and blood lipid levels. However, the Mediterranean diet was calorie limited whereas the Atkins group had a spontaneous loss of appetite. The molecular mechanism to explain the appetite loss is not clear. Reservations are that there was no placebo group and the study was too short to judge any clinical effects on cardiovascular events.

A broadly similar conclusion was reached in a meta-analysis of diets of varying carbohydrate and lipid composition. The new Atkins diet is one of several reduced-calorie diets that have all resulted in clinically meaningful weight loss, regardless of which macronutrients they emphasised.[28]

What about high-fat weight-losing diets?

The two potential problems with high-fat diets lie in their adverse effects on the blood lipoprotein pattern, and on the impairment of specific mental functions, as observed by Kieran Clarke in Oxford students. In the Oxford study, a short-term, high-fat, low-carbohydrate diet led to higher circulating free fatty acid (FFA) concentrations, impaired patterns of myocardial high-energy phosphate metabolism, and decreased cognition in healthy subjects.[29]

The site of these deleterious effects on the brain was the hippocampus. In the heart, sophisticated non-invasive nuclear imaging techniques measured levels of high-energy phosphate compounds, which were relatively low in those taking the high-fat diet. The proposal was that elevated circulating FFA levels were underlying the cognitive and cardiac abnormalities.

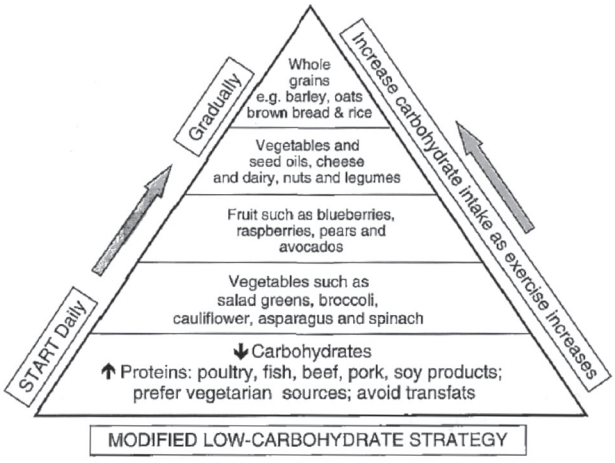

Fig. 2. This dietary pyramid starts at the bottom, with low carbohydrate intake of about 20 g per day, then as exercise increases, works up to 40 to 100 g of carbohydrates per day while maintaining weight loss, with the lifelong aim of maintaining the ideal weight. Note that poultry, fish and beef (free of visible fat) are allowed in the initiating phase. From Opie,[1] page 67.

Therefore Clarke and her associates concluded that high-fat, low-carbohydrate diets are potentially detrimental to the human heart and brain.[29,30]

For these reasons, there are arguments to support the view that the diet overweight persons could best start with is a new Akins type of diet for weight loss, coupled with an exercise programme, and then move onto the Mediterranean-type diet to achieve life-long health benefits, thereby avoiding the cognitive and cardiac changes of high-fat diets. Therefore starting a diet to lose weight, such as the new Atkins or Noakes diet, is complementary with a later switch to the long-term Mediterranean diet. As these diet types come in sequence, they are not competitive.

The future

A safe prediction is that there will be more editions of existing major books (Atkins in the USA, Dukan in Europe, Noakes in South Africa) besides new diet books. New lipid-lowering pharmaceutical agents are already being tested in large new outcomes-based studies on their preliminary promise.

The best self-help policy may well be to start with a dedicated programme for weight loss however achieved, whether by the new Atkins or Noakes diet, but associated with sufficient exercise. The next step would be to move on to the modified Mediterranean diet (Fig. 2) aimed at living longer and living better.

Looking to the far future, having both fish and meat in the daily diet of large populations would need substantial resources, which will be increasingly limited as the human race expands. Maybe the answer will lie in novel fresh nutritional sources such as algae-based diets.

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Comment

The cardioprotective diet: carbohydrates versus fat

FJ Raal

The global burdens of cardiovascular disease, obesity and type 2 diabetes mellitus continue to rise in both developed and developing countries.[1] Much of these burdens are preventable as they are the result of sub-optimal lifestyle, which includes poor diet, excess calorie intake, physical inactivity and cigarette smoking.[2]

As discussed by Lionel Opie, several diets, such as the new Adkins diet, the Noakes diet and the Dukan diet, which encourage the restriction of carbohydrates rather that the restriction of fat, has recently been introduced and many more are likely to follow.[3] Each claim to be better that the next at addressing this global health burden. However one has to consider what these diets are trying to achieve. Are they trying to achieve weight loss and prevention of the onset of type 2 diabetes, or are they trying to achieve cardiovascular protection?

It is correct that excessive carbohydrate intake, particularly refined carbohydrate as found in sugary drinks and energy snacks, is contributing to the global epidemic of obesity and type 2 diabetes mellitus but it is wrong to conclude that a high-carbohydrate intake is the major cause of atherosclerosis, the leading cause of cardiovascular disease worldwide. Atherosclerosis, particularly coronary artery disease, is not a disease of carbohydrate metabolism and there is little evidence to show that a low-carbohydrate diet will prevent atherosclerosis.

Restriction of refined carbohydrates, being our major energy source, will assist with weight reduction in the short term. However in terms of prevention of atherosclerosis and cardiovascular disease in the longer term, restriction of saturated fat is more important. It is therefore incorrect, and in fact it may be harmful, to advocate the substitution of refined carbohydrates with saturated fats.

Increasing the intake of saturated fats raises serum low-density lipoprotein (LDL) cholesterol levels.[4] Innumerable epidemiological studies have shown a positive relationship between serum LDL cholesterol levels and risk for cardiovascular disease, particularly coronary artery disease. In fact the link between LDL cholesterol and coronary artery disease is one of the most thoroughly researched in all of medicine.[5]

There is overwhelming evidence, accumulated over more than three decades, to show that the more you lower LDL cholesterol the lower your cardiovascular risk. For every 1 mmol/l reduction in LDL cholesterol using statins, there is approximately a 12% reduction in total mortality and a 21% reduction in major vascular events.[6] We have not yet identified a threshold below which LDL cholesterol reduction is no longer beneficial but harmful.[7]

So what should we be advising our patients at risk for cardiovascular disease? Obesity is not so much about diet but about energy balance – calories consumed versus those expended. Appropriate restriction of calorie intake whether it be carbohydrate, protein or fat is important for weight maintenance and prevention of obesity and type 2 diabetes. However in terms of achieving cardiovascular protection or maintaining a low LDL level, cholesterol is pivotal.

As Lionel Opie emphasises, we need to encourage and promote a healthy lifestyle with regular exercise, non-smoking and a healthy diet consisting of moderate portions of all three of the major components of our diet, namely carbohydrate, protein and fat.[3] If LDL cholesterol levels remain elevated or if the individual has established cardiovascular disease or diabetes, or is considered at high cardiovascular risk, international guidelines worldwide recommend that statin therapy should be initiated.[8,9] This will be much more beneficial for long-term cardiovascular protection than the short-term benefit of weight reduction achieved with marked carbohydrate restriction.

FJ Raal, FCP (SA), FRCP, FRCPC, Cert Endo, MMed (WITS), PhD, Frederick.raal@wits.ac.za
Division of Endocrinology and Metabolism, Department of Medicine, Johannesburg Hospital, Parktown, Johannesburg, South Africa

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Cardio News

New centre to revolutionise cardiac care in Africa

Ground-breaking new radial angiography service offered in the Western Cape will alleviate cardiac care bottlenecks and set the tone for others in Africa to follow

A pioneering cardiac service in radial angiography, currently not available elsewhere on the continent, will now be offered at a new cardiac care centre at the Division of Cardiology, Tygerberg Hospital in the Western Cape. Launched on 10 November 2014, the new state-of-the art facility, under the directive of SUNHEART and the Division of Cardiology, Department of Medicine, Stellenbosch University and Tygerberg Hospital, has seen a multi-million rand investment by key private and public partners, of which Medtronic is the principle partner. This will enable the Division of Cardiology to offer advanced cardiac care to more patients, especially from previously disadvantaged communities in and around the Western Cape, and alleviate current angiography bottlenecks in the region.

SUNHEART is a newly established, not-for-profit foundation that strives for equal access to advanced cardiac care for all patients. 'In South Africa more than 50 000 patients annually require advanced cardiac care, such as coronary angiography, yet many don't have access to these procedures and are therefore denied potentially life-saving treatments, including coronary stents. The first dedicated radial suite at the Division of Cardiology will see a significant increase in the number of patients we are able to treat', says Prof Anton Doubell, head of the Cardiology Division and director of SUNHEART.

Radial angiography allows patients to have coronary angiography and interventions, including stent placement, by accessing a small blood vessel in the arm rather than having to puncture the large blood vessels in the groin. This improvement on the standard approach has markedly increased the safety of the procedure and the comfort of the patient. The new radial suite situated in the angiography theatre complex will allow patients to be admitted and discharged on the same day. 'Not only are we looking at "business-class coronary intervention" in a state-of-the-art facility, but radial angiography and stenting through our radial suite will significantly reduce the need for an overnight hospital stay', says Doubell.

Medtronic MD, Mike Howe-Ely; Medtronic Director, William Stranix; SUNHEART Chairman, Dr Alfonso Pecoraro; patient; Western Cape Minister of Health, Theuns Botha and SUNHEART Director, Prof Anton Doubell.

'As we shift to a value-based healthcare culture, the need for innovative solutions that result in high-quality, cost-effective healthcare is more important than ever before. In an effort to improve and shape cardiac healthcare in South Africa and sub-Saharan Africa, we have invested in SUNHEART by providing the platform to train a significant number of cardiologists on radial techniques in these regions. This entails providing equipment, training workshops, hands-on support, and our expertise in the African market and the cardiovascular business to ensure the sustainability of the unit', says the managing director of Medtronic South Africa, Mike Howe-Ely, an investment partner in the new facility.

The investment in SUNHEART has seen the building of state-of-the art infrastructure, resulting in an expansion of service delivery and improved workflow. This has meant redesigning the out-patient service, introducing paperless reporting systems, as well as developing echocardiography imaging and stress-test services for an improved 'one-stop' patient offering.

Further support for the improvement in service delivery by the unit comes in the form of an innovative outreach programme where consultants render a decentralised cardiology service at referral hospitals, which will improve the access of patients to cardiac care in remote areas outside of Cape Town. An important outcome of this programme is the training of healthcare professionals at these hospitals.

'By investing in top-quality personnel and through the building of strategic partnerships, this unit has firmly established itself as a cutting-edge institution at the forefront of cardiology service delivery, training and research. SUNHEART is a success story that demonstrates what can be achieved through the building of public–private partnerships', says Western Cape minister of Health, Theuns Botha. 'Facilitating the building of partnerships between the public health sector and private funders is an important ingredient in healthcare planning for the future of South Africa.'

SUNHEART is actively investing in research and training aimed at the eradication of diseases that are relevant to the South African and African context. The first SUNHEART-sponsored research project, Echo in Africa (EIA), was launched this year in collaboration with the British Society of Echocardiography. EIA involves the screening of large numbers of school children for rheumatic heart disease.

Medtronic MD, Mike Howe-Ely; SUNHEART Director, Prof Anton Doubell; Western Cape Minister of Health, Theuns Botha and patient Caureen Amelia Petersen.

The Division of Cardiology has become the premier training facility for young cardiologists in South Africa and even further afield in Africa. An additional funded training fellowship and the renovation of the lecture room, resulting in an ultra-modern lecture facility, has boosted the teaching and training activities at the unit. The unit is a centre of excellence for radial angiography and percutaneous intervention, valvular heart disease, pericardial disease and advanced cardiac imaging, and boasts an echocardiography training and service infrastructure that is world class in Africa and South Africa.

A vision of equal access to advanced healthcare for all can only be achieved through a culture of collective responsibility. SUNHEART believes this culture already exists, and through finding partners who share in their vision, they will make an important contribution to the lives and health of patients.

Rhena Delport

South African Heart Association Congress

The 15th annual South African Heart Congress for 2014 was held at the Durban ICC from 16 to 19 October 2014, with Dr Sajidah Kahn as convener. The *Cardiovascular Journal of Africa* was represented at the conference by the editor-in-chief of the journal, Prof Patrick Commerford, and the regional editor for South Africa, Prof Rhena Delport. The theme for the congress was 'bridging the divide' between best practice and current challenges in the management of cardiovascular conditions.

In keeping with the congress intent, the prize for the best scientific article published in 2013 in the *Cardiovascular Journal of Africa* was awarded by an independent panel to Dr Jane Moses, the first author of the article 'Non-ST elevation myocardial infarction (NSTEMI) in three hospital settings in South Africa: does geography influence management and outcome? A retrospective cohort study'. The contribution of the other authors (Anton F Herbst, Philip G Klusmann, Karl JC Weich and SVH Hellmuth) is also acknowledged. Prof Patrick Commerford presented the award on behalf of the journal.

Prof Pat Commerford.

Dr Jane Moses and Prof Pat Commerford.

Case Report

A case of shoshin beriberi presenting as cardiogenic shock with diffuse ST-segment elevation, which dramatically improved after a single dose of thiamine

Jihye Kim, Sooyoun Park, Jun-Hyun Kim, Sun Woong Kim, Won Chan Kang, Sun Jong Kim

Abstract

Shoshin beriberi is a fulminant form of cardiac beriberi caused by thiamine deficiency. We report on a case of an 87-year-old man with shoshin beriberi presenting as cardiogenic shock with diffuse ST-segment elevation, which dramatically improved after thiamine administration. Because of the rarity of the occurrence, lack of diagnostic test and atypical presentation, diagnosing shoshin beriberi is challenging and requires a high index of clinical suspicion. Shoshin beriberi leads to rapid haemodynamic collapse and death. Therefore, clinicians should consider shoshin beriberi (or cardiac beriberi) as one of the differential diagnoses in patients with heart failure or cardiogenic shock.

Keywords: shoshin beriberi, thiamine deficiency, cardiogenic shock

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Thiamine deficiency (beriberi) has two major clinical manifestations: dry beriberi (peripheral neuropathy) and wet beriberi (cardiovascular disease). Shoshin beriberi is a fulminant form of wet beriberi and is characterised by hypotension, tachycardia and lactic acidosis, rapidly progressing to death if left untreated.[1]

Cardiac beriberi has repeatedly been reported for centuries all over the world, although it is very rare in the modern era, especially in developed countries.[2] Cardiac beriberi is commonly missed without a high index of suspicion. Considering the severity of the potential outcome left untreated, it is essential for clinicians to have an understanding of this disease.

Department of Internal Medicine, Konkuk University Hospital, School of Medicine, Konkuk University, Seoul, Korea

Jihye Kim, MD
Sooyoun Park, MD
Jun-Hyun Kim, MD
Sun Woong Kim, MD
Won Chan Kang, MD
Sun Jong Kim, MD, sjkim@kuh.ac.kr

We herein describe a case of shoshin beriberi presenting as cardiogenic shock with diffuse ST-segment elevation. It improved dramatically after thiamine administration.

Case report

An 87-year-old male in a general ward was transferred to the medical intensive care unit (ICU) with sudden onset of shock. He complained of chest discomfort. His vital signs revealed blood pressure of 85/56 mmHg, respiration rate 30–40 per minute, heart rate 128 beats per minute, temperature 36.5°C and 95% oxygen saturation in room air. An electrocardiogram (ECG) demonstrated ST-segment elevation in lead I, II, III, aVF and V2–V6 (Fig. 1). He was comatose.

He was admitted to our hospital for control of blood sugar level 40 days before the event. During the hospitalisation period, he fell into septic shock secondary to small bowel infarction; small bowel resection surgery was undergone. Since then, he had remained on total parenteral nutrition (TPN).

He had a history of diabetes mellitus, hypertension, asthma and stable angina, and was on regular medication. He had had right hemicolectomy surgery due to Burkitt lymphoma on the terminal ileum 14 months earlier, and a burr hole trephination due to traumatic subarachnoid haemorrhage 11 months before.

On auscultation of the chest, a coarse breathing sound was heard without crackles on both lungs. On laboratory investigation, haemoglobin level was 11.7 g/dl, white blood cell count was 8.43 × 10^3 cells/ml (neutrophils 85.7%, lymphocytes 11.0%), and platelet count was 61 × 10^3 cells/μl.

The biochemical profile showed: total protein 6.2 g/dl, albumin 3.1 g/dl, aspartate aminotransferase 12 IU/l, alanine aminotransferase 10 IU/l, alkaline phosphatase 91 IU/l, total bilirubin 1.7 mg/dl, prothrombin time 85%, blood urea nitrogen 55.7 mg/dl, creatinine 1.08 mg/dl, C-reactive protein 3.58 mg/dl, CK-MB/troponin I 8.3/0.22 ng/ml, brain natriuretic peptide 454.2 pg/ml, D-dimer 1.00 μg/ml, sodium/potassium/chloride 128/5.3/93 mmol/l, anion gap 27 mmol/l and lactic acid 12.2 mmol/l. Arterial blood gas revealed a pH of 7.49, pCO_2 of 22.7 mmHg, pO_2 of 98.8 mmHg, HCO_3 of 16.8 mEq/l, base excess of –4.9 mEq/l and SaO_2 98% on room air.

His chest X-ray showed cardiomegaly with mild pulmonary congestion, both pleural effusion and consolidation in the right upper lung field. This was not changed compared with the previous X-ray (Fig. 2).

We initially suspected cardiogenic shock due to an acute

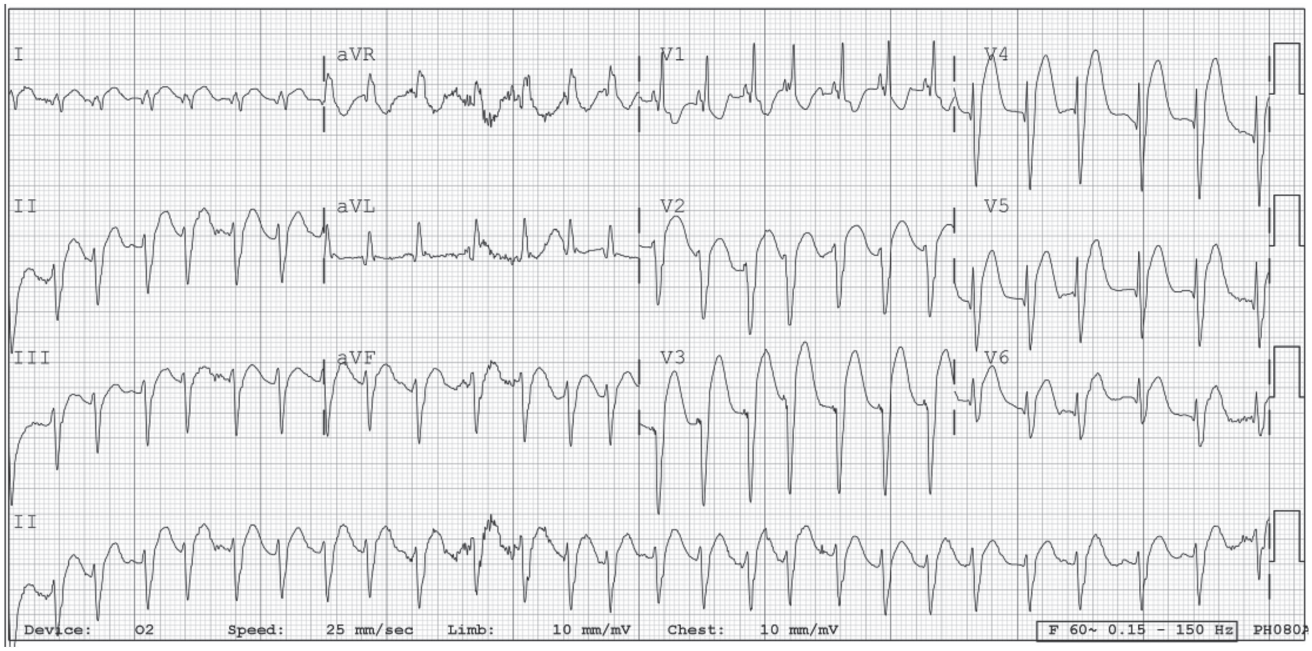

Fig. 1. Electrocardiogram showing diffuse ST-segment elevation involving nine leads, except aVR, aVL and V1.

coronary event, supported by chest pain, sudden onset of hypotension, diffuse ST-segment elevation on ECG, and previous history of coronary disease. Emergency coronary angiography (CAG) was done. It showed 75% focal stenosis on the circumflex artery os and 75% diffuse stenosis on the obtuse marginal branch, but there was no significant interval change compared with the previous CAG (Fig. 3). There was no pulmonary embolism or deep-vein thrombosis on thromboembolism computed tomography.

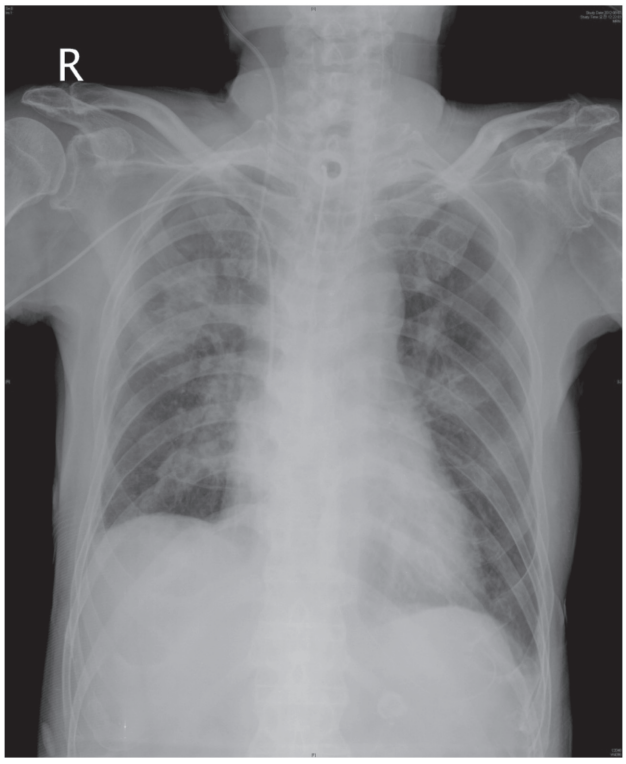

Fig. 2. Chest X-ray showing cardiomegaly with mild pulmonary congestion and pleural effusion from both lungs. However, consolidation on the right upper lung field shows no interval change compared with the previous X-ray.

Due to severe respiratory distress, a mechanical ventilator was applied. The patient developed rapid haemodynamic deterioration, refractory to vasopressor. Dopamine was increased up to 20 mcg/kg/min and norepinephrine up to 300 mcg/min. The pulse was not palpable and his blood pressure was measured at 67/41 mmHg on arterial line monitoring (Fig. 4).

Acidosis progressively deteriorated despite repetitive administration of sodium bicarbonate (Table 1). Based on the patient's history of TPN use for several months in conjunction with severe lactic acidosis and left ventricular (LV) dysfunction on portable echocardiography, cardiac beriberi was highly suspected. Thiamine 100 mg was immediately administered intravenously, resulting in a dramatic improvement of his haemodynamic status. We discovered later that the TPN given

Table 1. The serial arterial blood gas analysis

				Thiamine administration (at 01:30) ↓			
Time	16:31	22:02	00:53	02:04	04:10	08:24	13:25
pH	7.49	7.192	7.175	7.136	7.069	7.284	7.439
PCO_2 (mmHg)	22.7	27.5	21.8	30.1	38.4	37.0	30.0
PO_2 (mmHg)	98.8	150.1	162.9	135.1	81.7	83.4	90.3
FiO_2 (%)	35	30	30	30	30	30	
Bicarbonate (mmol/l)	16.8	10.3	7.9	9.9	10.9	17.2	19.9
Base excess (mmol/l)	–4.9	–16.4	–18.9	–17.8	–18.3	–8.8	–3.5
O_2 saturation (%)	97.8	98.0	98.1	97.4	88.9	94.3	96.3
Lactate (mmol/l)		12.2		15.4			

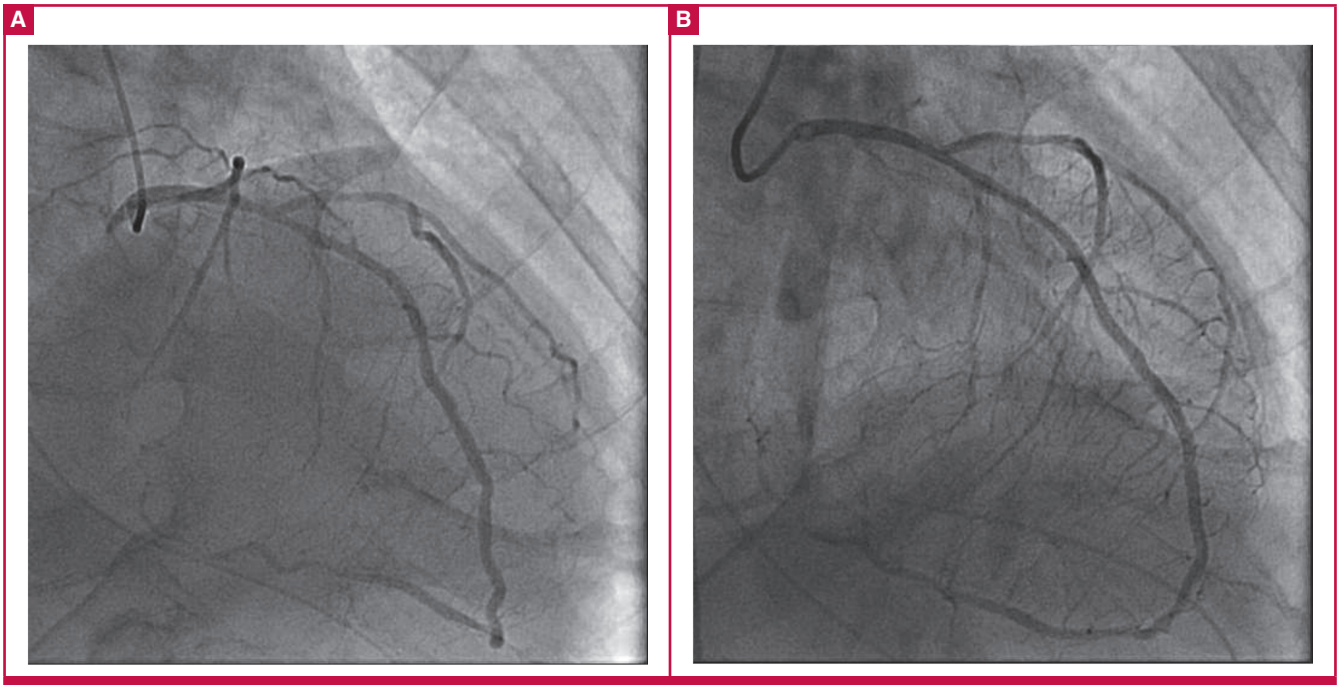

Fig. 3. An emergency coronary angiogram on ICU admission (B) showed no significant interval change compared with the previous CAG (A).

to the patient for several months had not contained any vitamin supplementation.

Three hours after thiamine administration, the patient's pulse was tactile and the systolic blood pressure measured 90 mmHg. Six hours later, urine output was increased to 180 cc per hour. The ejection fraction on portable echocardiography measured up to 50%, which was 25% just before the CAG, and LV motion was much improved.

He completely recovered from acidosis on the next day (Table 1) and the vasopressor was discontinued two days after the event. ECG showed normalisation of the ST-segment, which was elevated previously (Fig. 5). We continued thiamine

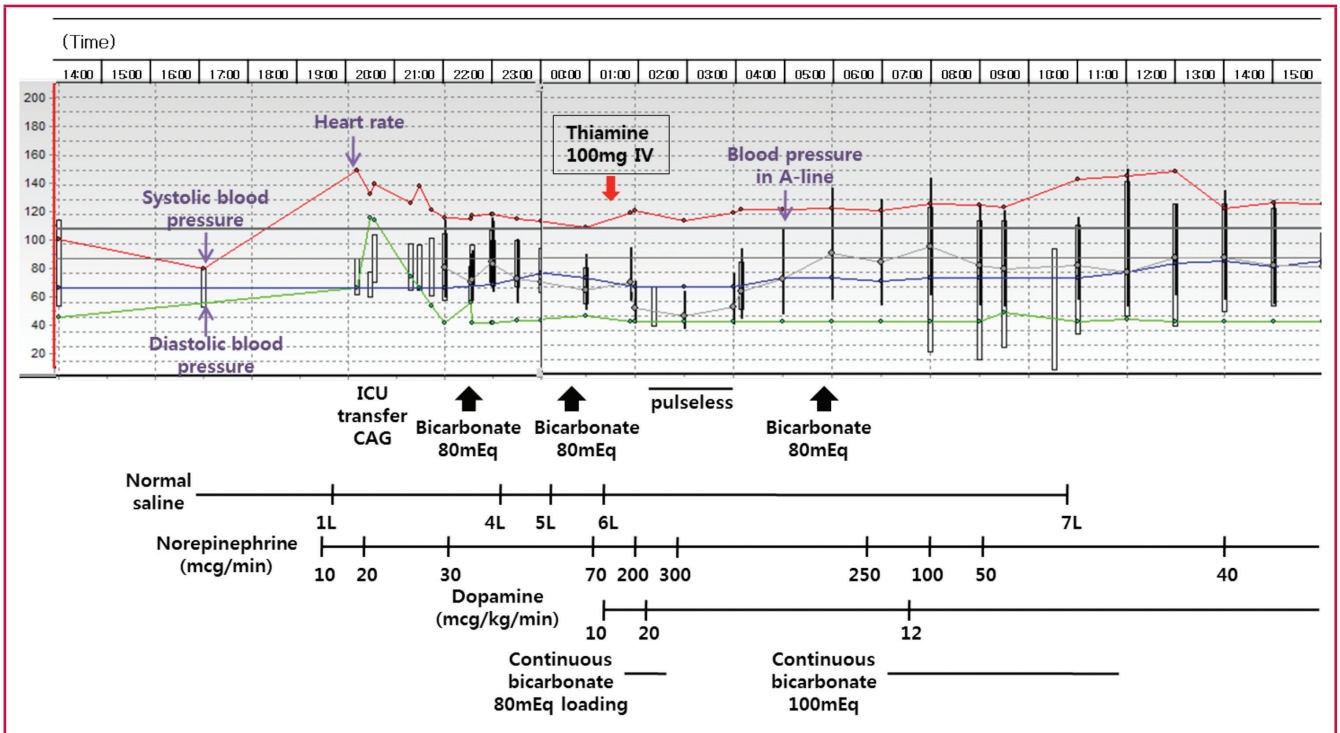

Fig. 4. Time-flow sheet shows blood pressure, respiration rate, body temperature, and administration of fluid, bicarbonate and vasopressors.

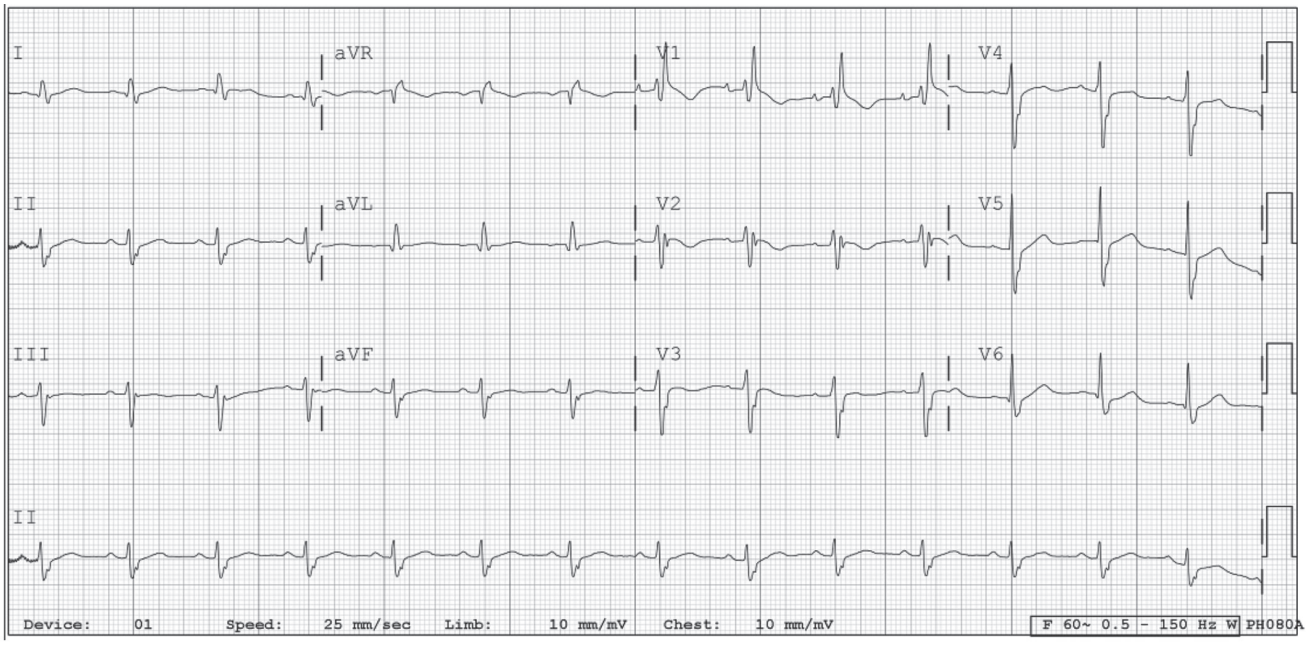

Fig. 5. Five days after ICU admission, ST-segment elevation was normalised on ECG.

supplementation with a daily dose of 100 mg for 11 days, and continued administering multivitamins, including thiamine hydrochloride 3.81 mg.

Follow-up echocardiography performed 15 days later revealed normal LV systolic function with ejection fraction of 58%. The patient's condition was stabilised and he was transferred to the general ward.

Discussion

Thiamine deficiency causes severe reduction in pyruvate dehydrogenase activity, subsequently preventing conversion of its substrate, pyruvate, into acetyl-CoA. This decrease in acetyl-CoA produces deficiency in nicotinamide adenine dinucleotide, resulting in a fall in cellular adenosine triphosphate (ATP).[3] The accumulation of pyruvate and lactate thereby causes intense vasodilatation due to peripheral arterio-venous shunts in the skeletal musculature, with a resulting drop in systemic vascular resistance and an increase in venous return.[4]

Cardiac beriberi is a disorder of thiamine deficiency that results in heart failure. Shoshin beriberi is a fulminant form of this disease, designated as 'a rapidly curable haemodynamic disaster',[5] and is characterised by hypotension, tachycardia and lactic acidosis. The present case manifested classical signs of shoshin beriberi.

Initially we suspected cardiogenic shock due to an acute coronary event, supported by sudden hypotension, diffuse ST-segment elevation on ECG and severe LV dysfunction on portable echocardiography. However, CAG revealed no evidence of acute myocardial infarction. At this point, a medical history of long-standing TPN use, in conjunction with heart failure and lactic acidosis was highly suggestive of a thiamine deficiency, or shoshin beriberi.

In this case, we did not measure serum thiamine concentration or red blood cell transketolase activity, the most commonly used laboratory techniques for diagnosis of thiamine deficiency. These tests are rarely performed in an emergency setting and the results are often non-specific or inconclusive. Therefore, diagnosis of shoshin beriberi is usually established by therapeutic response.[5-7]

In the present case, a single dose of thiamine promptly reversed both profound cardiovascular collapse and metabolic acidosis, and this therapeutic response was diagnostic of shoshin beriberi. According to previous reports, thiamine administration improved the haemodynamics within hours and normalised ECG changes within 24 hours in patients with shoshin beriberi.[6-8]

In this case, ECG characteristically showed diffuse ST-segment elevation in almost all the leads. There have been two case reports showing ST-segment elevation mimicking acute coronary syndrome in shoshin beriberi. One case showed a focal ST-segment elevation, which can be also seen in Brugada syndrome, suggesting autonomic nervous system abnormality as a possible mechanism causing ST-segment elevation.[8] The other case also revealed a focal ST-segment elevation and was associated with electrolyte deficiency and metabolic alkalosis.[9] This is the first case report to demonstrate diffuse ST-segment elevation in shoshin beriberi.

The exact mechanism leading to many different types of ST-segment change and myocardial damage in shoshin beriberi has not been revealed. Myocardial damage in the present case was not likely, due to coronary artery disease. Hypotension and secondary global coronary hypoperfusion may have played a role in the subsequent cardiac dysfunction and myocardial damage. Thiamine therapy may improve this and result in the normalisation of ECG changes.

Myocardial energy depletion may induce myocardial damage with ST-segment elevation because thiamine deficiency impairs myocardial energy metabolism. Moreover, various studies have suggested that activation of sarcolemmal ATP-sensitive potassium channels by ischaemic ATP depletion may result in ST-segment elevation.[10,11] Thiamine deficiency also induces ATP

depletion. Therefore, activation of ATP-sensitive potassium channels by ATP depletion induced by thiamine deficiency may contribute to ST-segment elevation in severe beriberi.

Thiamine deficiency is rare in developed countries and is most commonly associated with chronic alcohol abuse. Other predisposing conditions include chronic dietary malnutrition and impaired absorption or intake of dietary nutrients.[12] In the present case, thiamine deficiency resulted from TPN administration without thiamine supplementation. Because the capacity of thiamine storage in the body is small and the half-life of thiamine is only 10 to 18 days, patients depending on TPN become deficient in thiamine within a few weeks without thiamine supplementation.

Cardiac beriberi, including shoshin beriberi, is well reported in the literature. Shoshin beriberi, if not timeously recognised and promptly treated, can result in rapid haemodynamic collapse and death. However this condition is often under-recognised in clinical practice because of its rarity. In this setting, the diagnosis of shoshin beriberi requires a high index of clinical suspicion, particularly in groups with a high risk of thiamine deficiency.

When in doubt, thiamine should empirically be given because toxicity is not likely. Administration of thiamine is not only a simple and inexpensive treatment that can rapidly reverse a potentially lethal condition, but also a confirmative diagnostic technique. In the present case, thiamine administration dramatically improved the patient's haemodynamic status and saved his life.

Conclusion

Although thiamine deficiency is now relatively infrequent in developed countries, clinician should take into account cardiac beriberi (or shoshin beriberi) as one of the differential diagnoses in patients with heart failure or cardiogenic shock.

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Case Report

Clinical ventricular tachycardia and surgical epicardial ICD implantation in a patient with a Fontan operation for double-inlet left ventricle

Aysen Agacdiken Agir, Umut Celikyurt, Kurtulus Karauzum, Irem Yilmaz, Ersan Ozbudak, Serdar Bozyel, Muhip Kanko, Ahmet Vural, Dilek Ural

Summary

The Fontan operation is the primary surgical technique used for palliation of patients with single-ventricle physiology. Arrhythmias are frequently observed and associated with morbidity and mortality in Fontan patients. The frequency of arrhythmias after the Fontan procedure increases over time and it was reported to reach 50% in a 20-year follow up. Atrial tachyarrhythmias, especially atrial tachycardia and sinus bradycardia, are most frequently observed in these patients. Ventricular arrhythmias are rarely observed.

Generally, medical therapy, catheter ablation, pacemaker or implantable cardioverter defibrillator (ICD) implantation are options in the treatment of these arrhythmias. It may be difficult to implant either a pacemaker or an ICD in patients on whom the Fontan procedure has been performed. In conditions where access to the right ventricle is from the venous system, it is anatomically impossible. Where there is no functional right ventricle, device implantation can be performed with alternative methods other than the conventional transvenous approach.

In this report, we discuss a middle-aged woman with a Fontan operation performed 14 years earlier, who presented with ventricular tachycardia (VT) and in whom an epicardial ICD was implanted. The literature on this issue is also reviewed.

Keywords: Fontan operation, ventricular arrhythmia, implantable cardioverter defibrillator, epicardial

Department of Cardiology, Kocaeli University Medical Faculty, Kocaeli, Turkey
Aysen Agacdiken Agir, MD, aagacdiken@yahoo.com
Umut Celikyurt, MD
Kurtulus Karauzum, MD
Irem Yilmaz, MD
Serdar Bozyel, MD
Ahmet Vural, MD
Dilek Ural, MD

Department of Cardiovascular Surgery, Kocaeli University Medical Faculty, Kocaeli, Turkey
Ersan Ozbudak, MD
Muhip Kanko, MD

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Double-inlet ventricle falls under the umbrella of univentricular atrio-ventricular (AV) connections. These hearts are defined as having more than 50% of each AV connection connected to a dominant ventricle. In about 75% of patients, the dominant (functional) ventricle is a left ventricle, separated from the right ventricle by a rudimentary interventricular septum.[1] These patients usually undergo Fontan surgery with a surgical algorithm such as in those with tricuspid atresia.

The frequency of arrhythmias after the Fontan procedure increases over time and it was reported at 50% in a 20-year follow up.[2,3] Early postoperative arrhythmias can result from sinus node or sinus node artery trauma. In the late period, arrhythmias can result from atrial dilatation and distension and surgical scars. Atrial tachyarrhythmias, especially atrial tachycardia and sinus bradycardia, are most frequently observed in these patients. Ventricular arrhythmias are rarely observed. However, sudden cardiac death is the most common cause of mortality in adult congenital heart disease. Generally, medical therapy, catheter ablation, pacemaker or implantable cardioverter defibrillator (ICD) implantation are the options in the treatment of these arrhythmias.

In this article we report on a middle-aged woman with a Fontan operation 14 years earlier, presenting with ventricular tachycardia (VT) and in whom an epicardial ICD was implanted. The literature on this issue is reviewed.

Case report

A 43-year-old woman was admitted to the emergency department with palpitations accompanied by pre-syncope. There was no history of syncope. She had had a Fontan operation due to a double-inlet left ventricle 14 years earlier. Revision of the previous Fontan connections and an epicardial VVIR pacemaker implantation were done due to bradycardia nine years earlier.

On physical examination, her vital signs showed a body temperature of 36.7°C, a pulse of 190 beats/min, and blood pressure of 80/50 mmHg. Cardiac auscultation revealed beat-to-beat changes and softening of S1, mildly loud S2 and

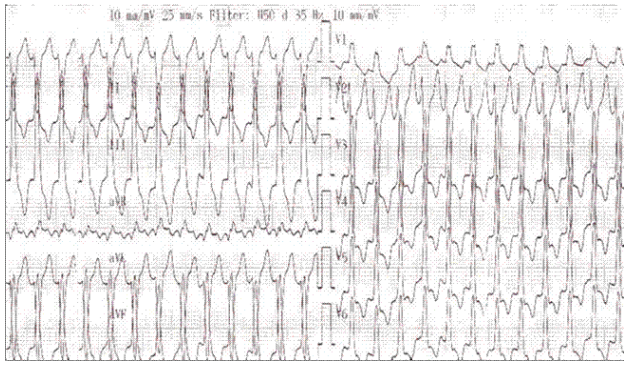

Fig. 1. ECG showing wide QRS complex tachycardia.

2–3/6 holosystolic murmur at the apex. Respiratory system examination revealed rough breathing sounds, but there were no rales or rhonchi.

Chest X-ray showed a mildly increased cardiothoracic index. The electrocardiogram (ECG) showed wide QRS complex tachycardia (Fig. 1). The QRS duration was 150 ms. In the emergency department, adenosine (12 mg iv) was given to the patient to eliminate the supraventricular tachycardia but there was no response in rhythm or rate.

Her basic laboratory findings were glucose: 149 mg/dl, urea: 103 mg/dl, creatinine: 1.02 mg/dl, potassium: 4.03 mg/dl, uric acid: 11.1 mg/dl, AST: 1601 U/l, ALT: 1679 U/l, INR: 2.18. When the laboratory findings and imaging tests were evaluated together with the findings of the physical examination, forefront, ischaemic hepatitis and prerenal azotaemia showing end-organ damage were considered.

Because the haemodynamic parameters were unstable and critical end-organ damage had developed, emergency electrical cardioversion (100 J) was performed. After cardioversion, pacemaker rhythm (60 beats/min) was maintained (Fig. 2) and the blood pressure was 125/75 mmHg.

Transthoracic echocardiogram (TTE) revealed a rudimentary interventricular septum with hypoplastic right ventricle and functional single (left) ventricle, a conduit between the right atrium and pulmonary artery, left atrial dilatation, severe

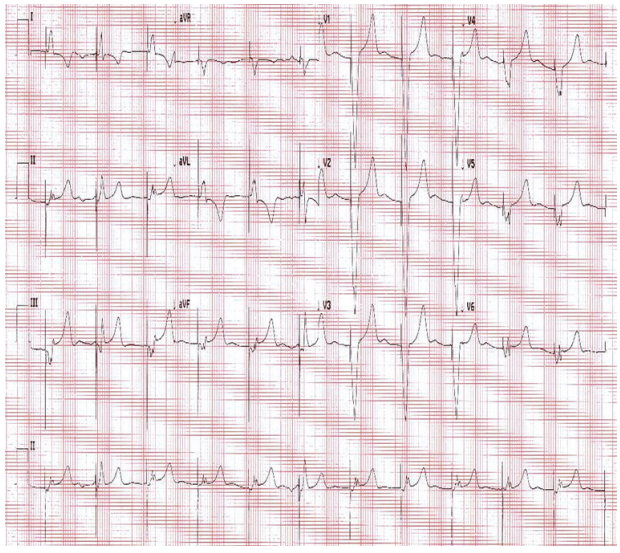

Fig. 2. Pacemaker rhythm (60 bpm) was observed on ECG after cardioversion.

mitral regurgitation (eccentric) and systolic dysfunction with an ejection fraction (EF) of 30% (Fig. 3). Considering structural heart disease with decreased LVEF (< 30%) and spontaneous sustained episodes of ventricular tachycardia (VT), the patient had class I (evidence level B) indication for ICD implantation, according to the recent guidelines.[4]

The cardiac anatomy and venous system was scanned with cardiac computerised tomography (CT) to evaluate for lead placement. On CT, the arcus aorta was located on the left, the left anterior descending artery and right coronary artery were emerging from the same cusp adjacently, and the circumflex artery was emerging from another cusp. The right ventricle was significantly hypoplastic and associated with the left ventricle, there was no main pulmonary artery, and the pulmonary arteries were connected to the right atrium (Fig. 4). Since the conventional transvenous access to the markedly hypoplastic right ventricle was impossible, an epicardial ICD implantation was planned.

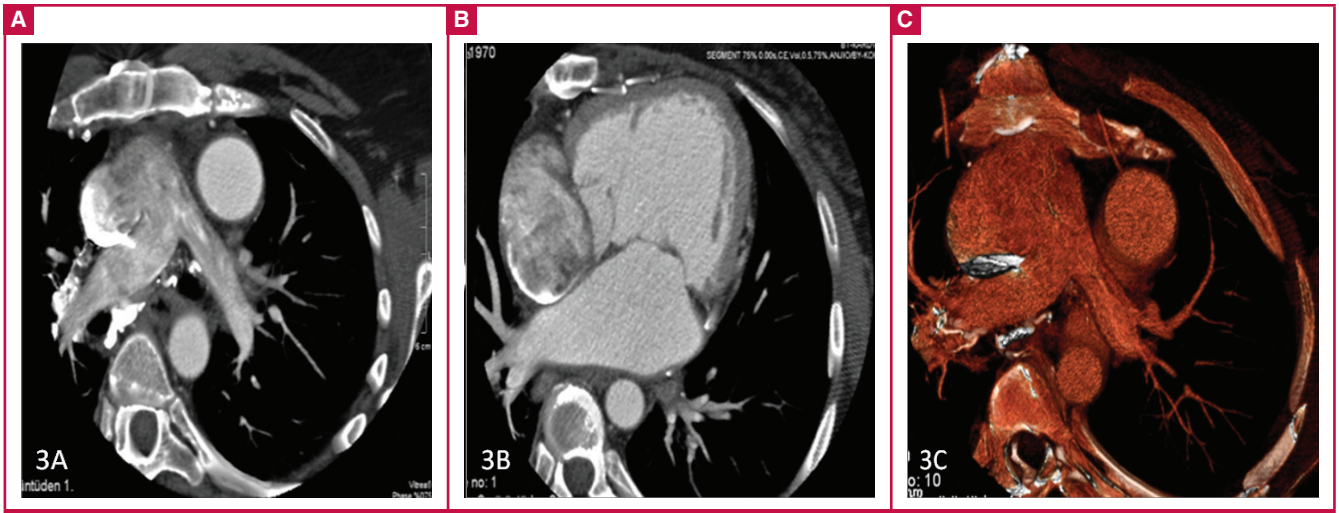

Fig. 3. A, B, CT scan showing systemic venous blood entering the pulmonary circulation through a cavopulmonary conduit. C, a hypoplastic right ventricle associated with the left ventricle was seen on CT scan.

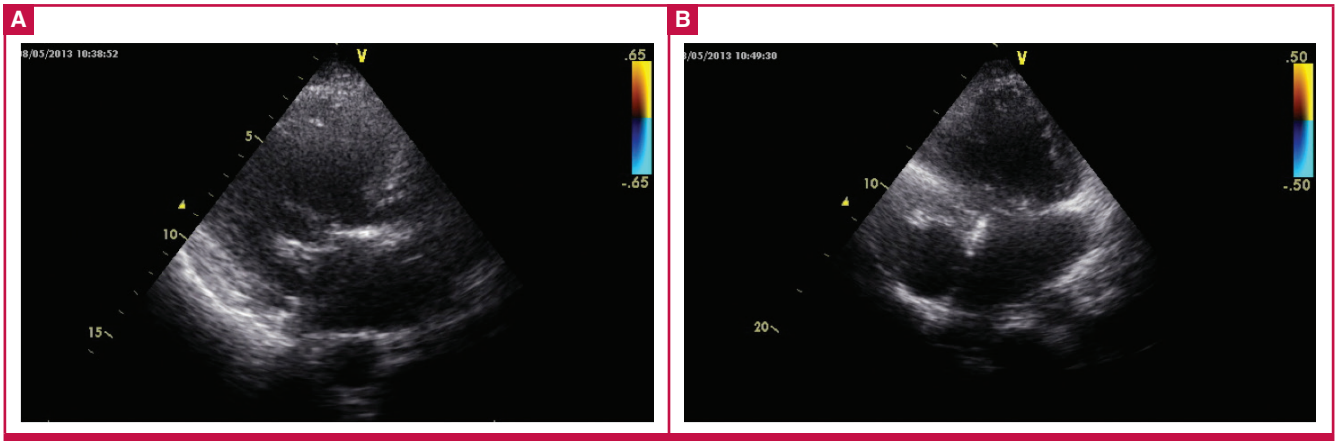

Fig. 4. Transthoracic echocardiographic images in the parasternal long-axis and apical four-chamber views.

Under general anaesthesia, after pealing back the pleura, the pericardium was reached via an incision in the fifth intercostal space by left anterior thoracotomy. The pericardium was opened from the anterolateral site and the left ventricle was explored. The Medtronic single coil (model 6937; Medtronic Inc, Minneapolis, MN) electrode was fashioned as a halo and secured with non-absorbable suture material. This halo was placed between the inferior surface of the heart and the diaphragmatic surface of the pericardium. Through the same incision an active fixation bipolar epicardial lead (model 4968; Medtronic Inc, Minneapolis, MN) was fixed to the lateral wall of the left ventricular epicardium in a position where sensing, pacing and impedance measurements were satisfactory. Ventricular fibrillation (VF) was induced with 'shock-on-T' and the therapy was successful in defibrillating VF at 25 J (a 10-J safety margin). Both leads were then tunnelled through the subcutaneous tissue to be placed inside a generator pocket created in the left sub-pectoral region.

As soon as bleeding was controlled, the layers were closed in the anatomical positions. There were no complications in the peri-operative or postoperative periods. The patient was discharged on medical treatment: acetylsalicylic acid, angiotension converting enzyme inhibitor, beta-blocker, aldosterone receptor antagonist and furosemide. During the eight-month follow up, two appropriate shocks were delivered.

Discussion

The Fontan operation has been the primary surgical technique used for palliation of patients with single-ventricle physiology.[5] It is based on leading systemic venous blood into the pulmonary circulation and pumping pulmonary venous blood into the systemic circulation by a unique functional ventricle.

Arrhythmias are frequently observed and are associated with morbidity and mortality in Fontan patients. It is reported that the frequency of arrhythmias gradually increases, and reaches 50% by 20 years of follow up.[2,3] Postoperative early arrhythmias are a result of trauma to the sinus node and sinus node artery.[6,7] In the late period they may develop due to atrial dilatation and distension,[2,7] and surgical scars.

Major clinical studies investigating late arrhythmias in Fontan patients are summarised in Table 1.[2,8-15] Intra-atrial re-entrant tachycardia and sinus bradycardia are seen most commonly; ventricular arrhythmias are more rare and these arrhythmias arise from surgical scars. In a multicentre, cross-sectional study,[16] ventricular tachyarrhythmia was reported in 18 (3.5%) of 520 patients in whom a Fontan procedure had been performed.

Nakamura and collegues[13] retrospectively reviewed 48 patients who survived and were followed up for more than 15 years, among 110 patients who underwent a Fontan operation. Fifteen years after the Fontan operation, six patients (12.5%) with a higher age at operation developed VT, irrespective of ventricular function. The interval between the Fontan operation and onset of VT correlated inversely with age at Fontan operation. They suspected that pathological changes caused by longstanding cyanosis and volume overload before the Fontan operation may have produced arrhythmogenic areas, resulting in VT, with an additional postoperative factor, age itself, in patients when they got close to their thirties.

In our case, the patient had undergone the Fontan operation 14 years earlier when she was 30 years old. Her left ventricular ejection fraction was also quite low. These factors may have played a role in the development of VT in our patient.

For structural reasons, it may be difficult to implant either a pacemaker or an ICD in patients with congenital heart disease,[7,17] especially those in whom a Fontan procedure had been performed. In conditions where access to the right ventricle from the venous system is anatomically impossible (e.g. tricuspid atresia), or there is no functional right ventricle (e.g. double-inlet left ventricle), device implantation can be performed with alternative methods other than the conventional transvenous approach.

An earlier solution was the placement of epicardial patch electrodes and pacing/sensing leads. The epicardial patch technique has been associated with post-pericardiotomy syndrome, mediastinitis and constrictive pericarditis.[18] Non-traditional alternative surgical methods include video-assisted thoracoscopy, a subxiphoid approach or lateral thoracotomy for placemant of either an epicardial coil and/or ventricular pace-sense leads.[19] In patients with Fontan, although small in number, there are cases in whom an epicardial pacemaker/ICD has been implanted.

In a retrospective study by Cannon *et al.*,[20] eight patients with non-traditional lead placement were identified and included. Four of them had undergone previous Fontan operations because of double-inlet left ventricle or tricuspid atresia. The indications

Table 1. Arrhythmias late after the Fontan operation in some studies

Authors	*Follow up (years)*	*Patients (n)*	*Mean age at Fontan (year)*	*Bradyarrhythmia*	*Supraventricular arrhythmias*	*Ventricular arrhythmias*	*Treatment*	*Outcome*
Driscoll *et al.* (1992)[2]	7.7	352	11.1	PM implantation (13%)	AF/AFL (21%)	PVC (11%) VT (6%)	Anti-arrhythmic drugs	Death (35%) Reoperation (29%) Hospitalisation for arrhythmias (15%)
Gates *et al.* (1997)[8]	7.4	21	27	Complete heart block (5%)	Atrial arrhythmias (37%)	VA (11%)	Anti-arrhythmic drugs	Operative mortality (5%) Late death (5%) Reoperation (16%)
Durongpisitkul *et al.* (1998)[9]	5.9	499	9.5	Sinus bradycardia (3.4%) Junctional rhythm (6.1%)	SVT (all types) (17% at 5 years)		PM implantation	Early mortality 9% 5-year mortality: 13% Sudden death 29% Late SVT risk factors; Age at operation (< 3 or ≥ 10 years) AV valve replacement
Van den Bosch *et al.* (2004)[10]	15	36	12	SND (17%) AV block (6%)	SVT (56%)	VT (6%)	Epicardial PM implantation Anti-arrhythmic drugs Catheter ablation	Deaths (28%) Arrhythmogenic deaths (11%)
Nürnberg *et al.* (2004)[11]	7.9 4.4	ILT (29) ECC (45)	5.8 3.8	Late bradyarrhythmia (11.5%) (0)	Late SVT (27%) (0)		PM implantation	Total mortality (8%)
Stephenson *et al.* (2010)[12]	8.6	520	3.4	Bradycardia (5%) SND (1.3%) Junctional rhythm (0.3%) Complete heart block (0.2%)	Ectopic atrial tachycardia (0.8%) AVRT (1.8%) IART (7.3%)	VT (3.5%)	PM implantation (12%) ICD implantation (0.9%)	IART increased with age; mean age for IART 14.4 years
Nakamura *et al.* (2011)[13]	18.5	APC (26)	5		AT/IART (58%) AF (12%)	NS-VT (15%)	Fontan conversion Full maze procedure Anti-arrhythmic drugs Electrophysiologic study Catheter ablation	
		TCPC (22)			SVT (41%)	(9%)		
Balaji *et al.* (2013)[14]	9.2	ILT (602)		Late bradyarrhythmia (18%)	Late tachyarrhythmia (10%)		–	DC cardioversion (acute) (7%)
		ECC (669)		(9%)	(3%)			(2%)
Lasa *et al.* (2014)[15]	7.1	ECC (87)	2.4	Bradyarrhythmia (30%) – SND (n = 25)	Tachyarrhythmia (7%) – AVNRT (n = 1) – IART (n = 2)	–	PM implantation (n = 2)	Duration of follow up from Fontan was an independent predictor of late PM implantation
	10.5	ILT (106)	1.9	– SND (n = 32) – Complete heart block (n = 1)	AVNRT (n = 1) IART (n = 3) JET (n = 1)		(n = 12)	

PM: pacemaker, AF: atrial fibrillation, AFL: atrial flutter, PVC: premature ventricular contraction, VT: ventricular tachycardia, VA: ventricular arrhythmias, SVT: supraventricular tachycardia, AVRT: re-entrant atrio-ventricular tachycardia, IART: intra-atrial re-entrant tachycardia, AT: atrial tachycardia, JET: junctional ectopic tachycardia, SND: sinus node dysfunction, LTFO: intra-atrial lateral tunnel, ECC: extra-cardiac conduit, APC: atrio-pulmonary connection, TPCP: total cavo-pulmonary connection.

for implantation were VT and ventricular dysfunction in two, VT and syncope in one, and resuscitated sudden cardiac death in one patient. All underwent a midline sternotomy with surgical placement of a subcutaneous ICD coil into the pericardial sac. In addition to the ICD lead, a second epi-myocardial bipolar lead was placed on the ventricle for sensing and pacing in all patients. All patients had a defibrillation threshold of < 20 J.

There were no complications at a mean follow-up period of 22 months despite it being thought that the intrapericardial defibrillation coil could lead to pericarditis, adhesions, infection and pericardial thickening. We have not observed any complications related to epicardial ICD coils in our patients to date. Although defibrillation coil lead dislodgment from the pericardium is a concern, stabilising the tip and body of the coil in a halo shape and securing the lead to the surrounding tissue, as used in our technique, should stabilise the lead in the pericardium.

In another case series, eight patients were identified who had undergone ICD placement or revision requiring non-traditional alternative surgical lead placement.[21] One had undergone the Fontan procedure because of double-inlet left ventricle and pulmonary atresia. For primary prevention due to severe LV failure, the ICD lead was placed subcutaneously along the contour of the left chest wall and the rate-sensing lead was attached directly to the epicardium through a lateral mini-thoracotomy. Both were then connected to the ICD generator placed in the sub-pectoral region. There were no major cardiovascular complications.

It is not clear from the literature which non-traditional method is the best in patients with Fontan and other limited venous access. In a review of multicentre experiences, the authors have described several ICD implantation techniques that did not utilise either transvenous high-voltage coils or epicardial patches in children and young adults.[22]

A total of 22 patients (mean age 9 ± 10 years) underwent a novel ICD implant approach at the 10 centres. Diagnoses included several types of complex congenital heart disease: intracardiac tumours (n = 2), hypertrophic cardiomyopathy (n = 1), long QT syndrome (n = 4), left ventricular non-compaction (n = 1), ARVD (n = 1) and idiopathic VT or VF (n = 6). Three different configurations for the coil were used; 11 patients had subcutaneous arrays placed around the thorax, eight had a transvenous design ICD lead placed on the epicardium, one had a transvenous design lead placed on the epicardium as well as subcutaneous arrays added due to a high defibrillation threshold with the epicardial lead alone, and two patients has a transvenous design ICD lead placed in a subcutaneous position. Most patients underwent a thoracotomy or limited subxiphoid sternotomy for placement of either an epicardial coil and/or ventricular pace-sense lead. A comparison was made between

the defibrillation thresholds of those patients who received subcutaneous systems, and those who had a shock coil placed directly on the pericardium. The epicardial systems were found to have significantly lower defibrillation thresholds. It was concluded that the epicardial coil technique would be preferable in patients with greater body mass.

Conclusion

It is important to recognise that careful consideration must be given to choose the appropriate technique in patients with a Fontan operation and limited venous access. Techniques for implantation of an ICD should be individualised in this population. Long-term outcomes and possible complications of these techniques are not well known because of limited data in this patient group. Therefore regular follow up regarding the longevity and safety of these non-traditional techniques and devices is important.

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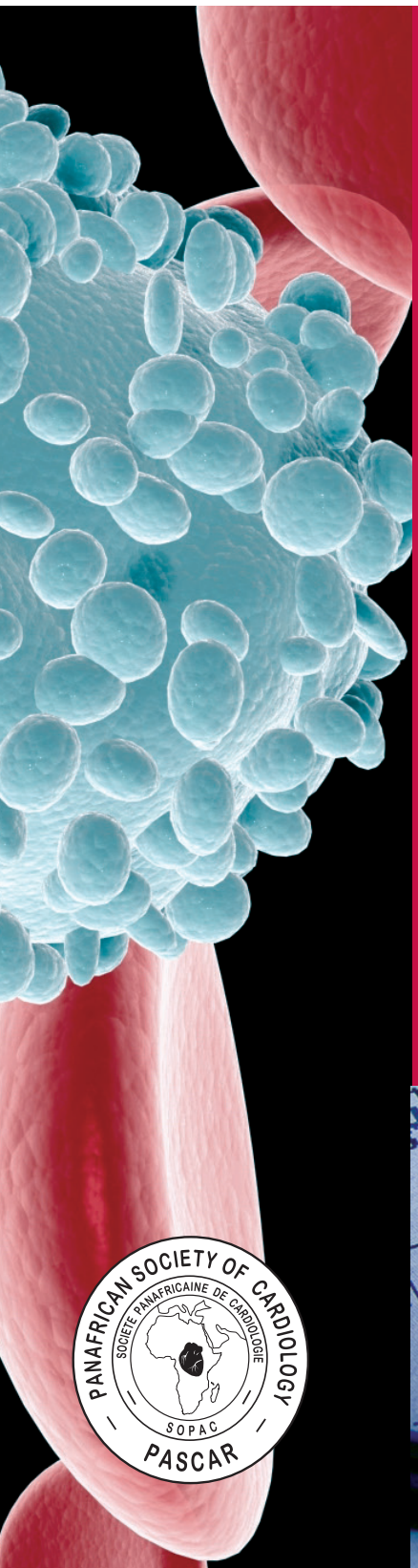

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