Lone ventricular cardiomyopathy, 1993 - 1996

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Objective. To identify subjects with univentricular idiopathic structural and/or functional myocardial disorders (as defined) and to describe the characteristic features.

Design. Over a period of 4 years, 1993 - 1996, all adult subjects were obtained consecutively from a centralised referral cardiological service. The subjects had to fulfil a set of formulated diagnostic criteria for each isolated type of univentricular disease — symptomatic or asymptomatic. The subjects were diagnosed on the basis of clinical features, supported by electrocardiographic, radiological and echocardiographic evidence of lone ventricular disease, with a further definition of abnormalities based on appropriately selected standard left and right heart assessments, *inter alia:* (*i*) cardiac catheterisation, including coronary arteriography and pulmonary angiography; (*ii*) radio-isotope studies — mibiscan; (*iii*) ventilation perfusion scan; and (*iv*) laboratory tests to identify likely cause(s) of difffuse myocardial damage as well as to recognise nonspecific effects of tissue damage and organ dysfunction.

Setting. A referral cardiological service of a tertiary academic hospital, which provides a consultative service for inpatients and ambulatory cases. All subjects were studied on admission to hospital.

Participants. A set of criteria was formulated for each category of lone ventricular myopathy. A total of 30 patients were thus identified and included in the study — men and women ranging in age from 18 years to 84 years, with an average of 48 years. All were investigated after admission to hospital by means of a detailed set of investigations that rigorously excluded overt or occult causes of diffuse myocardial damage and any severe myocardial dysfunction secondary to haemodynamic conditions. Seven patients with significant coronary artery disease were excluded. Any subject with pulmonary or systemic hypertension was also excluded.

Main outcome measures. Total number of patients, number of patients in each subgroup were analysed by age, sex, clinical features, and by special investigation. The mode of presentation and electrocardiographic features were analysed separately.

Results. Twenty-two left ventricular and 8 right ventricular cases of lone ventricular cardiomyopathy were diagnosed. All but 1 patient with right ventricular disease were symptomatic and 5 subjects with left ventricular myopathy were incidentally discovered. There were 17 men and 13 women in the series. Of the 8 patients with right ventricular disease, 6 were women, while of the 22 patients with left ventricular cardiomyopathy, 15 were men.

Conclusion. The study supports the previously described existence of lone ventricular idiopathic cardiomyopathy. Further studies are, however, indicated in order to define its prevalence and nature more accurately, as well as to describe any relationship with univentricular cardiomyopathies, and define the characteristics of each category and the possible evolutionary patterns. Right ventricular cardiomyopathy is a new entity which may pose difficult diagnostic challenges, while left ventricular disease is generally accepted as a stage in the clinical spectrum of classic idiopathic dilated cardiomyopathy.

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Dilated idiopathic cardiomyopathy is a universal clinical problem. It is one of the commonest cardiological causes of morbidity and mortality, especially among South African blacks. Gillanders[1] set the scene in South Africa with a report of 30 cases of what he called 'nutritional heart disease'. The causative nutritional factors suspected to be due only in part to thiamine deficiency, continued to be favoured for some time.[2] From Grusin's later series,[3] it became clear that this syndrome of 'cryptogenic heart disease' of blacks, was not a simple nutritional disorder. Curiously, some of Grusin's patients responded to a low-thiamine diet (although bedrest was probably just as effective at an early stage). Different clinical stages and the impact of duration of the disease began to be appreciated. From a paper by Brink and Webber,[4] on a case report of endomyocardial fibrosis, and from discussions at an international seminar in Johannesburg,[5] the concepts of the cardiomyopathies that occur in Africa were formerly consolidated. Reports from various English-speaking African countries confirmed the existence of obscure types of cardiomyopathy, with two distinct forms. The term 'cardiomyopathy', used without qualification, was generally adopted in 1971.[6]

The dilated form of cardiomyopathy, a heterogeneous condition initially called congestive,[7] is a clinical syndrome. There remain many unanswered questions on aetiology, pathology and other aspects. It is assumed that the common clinical presentation, with biventricular, four-chamber dilatation, usually but not always starts as a left-sided disorder.[8] However, left ventricular dilatation is not invariable;[8] neither does left ventricular concentric hypertrophy negate the diagnosis.[8] There are at least three subtypes of dilated idiopathic cardiomyopathy, viz. a left ventricular disorder, a peripartum disease and a right

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ventricular form.[8,9] Although knowledge on cardiomyopathy is incomplete, especially on aspects of predictors of outcome, pathology and aetiology, we set out to identify two morphological/functional forms at a consulting clinic that deals with a large number of 'typical cases'. Our period of study is 1993 - 1996.

Subjects and methods

Thirty consecutive adult subjects were identified selected from a large group of patients with clinical central cardiovascular diseases. Table I shows the patients' profiles, as well as the electrocardiograms and the clinical presentation of each patient. Table II shows crude statistics of Ga-Rankuwa Hospital's activities for the Departments of Medicine and Cardiology for 1 year. Of the inpatients, about 3 - 4% would have a major cardiological problem, virtually all with symptomatic disease. About 30 - 40% of all subjects with cardiac failure have cardiomyopathy. Very few asymptomatic patients are routinely admitted for investigation. For this study, 'scarce beds' were 'sacrificed' by admitting minimally symptomatic or asymptomatic subjects or subjects who would ordinarily have been treated at home. Approximately one-quarter of patients with heart failure, admitted to this hospital, would have idiopathic dilated cardiomyopathy. Table III lists the criteria to be fulfilled for each univentricular cardiomyopathy category.

Table I. The patients, 1993 - 1995

No.	Age (yrs)	Sex	Ventricle affected	ECG	Clinical presentation
1	43	F	Right	Atrial flutter with block, T-wave inversion on right precordial leads, QRS axis normal	Right heart failure
2	32	F	Left	T-wave inversion on V1-V6, AVL and S1, sinus rhythm	Left ventricular symptoms — pulmonary congestion
3	62	M	Left	Normal	Acute pulmonary oedema
4	38	M	Right	Normal	Cardiomegaly on chest radiograph — no symptoms
5	28	F	Right	Atrial fibrillation, low voltage, normal QRS axis	Right heart failure
6	30	F	Right	Sinus rhythm, T-wave inversion V1-V3	Right heart failure
7	60	M	Left	Sinus rhythm, normal QRS, voltage and axis, flat T waves V3-V6, AVL, S1	Pulmonary congestion symptoms
8	43	M	Left	Sinus rhythm, T-wave inversion S2, S3, AVF	Chest pain, symptomatic pulmonary congestion
9	60	M	Left	Normal sinus rhythm, normal QRS axis	X-ray cardiomegaly, no symptoms
10	52	M	Left	Sinus rhythm, flat T wave, V3-V6	Symptoms of pulmonary congestion
11	60	M	Left	Normal ECG	Cardiomegaly only — query 'decapitated' hypertension
12	30	F	Left	Sinus rhythm, flat T waves globally, left axis QRS — 30°.	Peri-pregnancy symptoms of pulmonary congestion
13	18	M	Left	Concentric hypertrophy, left axis QRS — 30°, sinus rhythm	Acute pulmonary oedema, left ventricular thrombus on echocardiogram
14	29	F	Left	Sinus rhythm, T wave, inversion V4, V6, AVL, S1	Severe pulmonary congestion symptoms
15	43	M	Left	Pathological Q waves, inferior heart, sinus rhythm, normal QRS axis	Pulmonary oedema, possible acute myocardial infarction
16	40	F	Left	Voltage, left ventricular hypertrophy and strain over left precordial leads	Pulmonary congestion symptoms
17	32	F	Left	Sinus rhythm, flat T waves over left V leads	Peri-pregnancy pulmonary oedema
18	70	M	Left	Sinus rhythm, T-wave inversion globally, normal QRS axis	Pulmonary oedema, 'minimal right heart failure', normal right ventricle
19	40	F	Left	Left ventricular concentric hypertrophy	Pulmonary congestion
20	29	M	Left	Sinus rhythm, normal QRS axis, left ventricular voltage, criteria for hypertrophy	No symptoms, cardiomegaly
21	74	M	Left	Low voltage, sinus rhythm, normal QRS axis, flat T wave on left ventricular leads	Congestive cardiac failure
22	60	M	Left	Normal ECG	Cardiomegaly, no symptoms
23	42	M	Right	Right QRS axis, sinus rhythm	Right heart failure
24	48	M	Left	Normal ECG	Pulmonary congestion symptoms
25	68	M	Left	Atrial fibrillation, normal QRS axis	Stroke — cerebral infarct
26	45	F	Right	Sinus rhythm, right QRS axis, low voltage	Right heart failure
27	84	F	Right	Atrial fibrillation, normal QRS axis	Right heart failure
28	32	F	Right	Atrial fibrillation, QRS axis right, right bundle-branch block, normal voltage, right ventricular hypertrophy	Right heart failure
29	83	M	Left	Sinus rhythm, low voltage, normal QRS axis	No symptoms
30	57	F	Left	Sinus rhythm, Q waves + ST elevation on inferior leads, anterior leads	Chest pain, no pulmonary congestion

Every patient underwent a meticulous clinical evaluation, independently checked by another person, with subsequent discussion. Basic laboratory tests were performed in every patient, viz. a full blood count, erythrocyte sedimentation rate, urea and electrolyte assessment, full urinalysis, as well as a chest radiograph and an electrocardiogram. The investigations listed in Table IV were not done in every case, but were chosen and grouped on the basis of clinical indications or a specific situation with regard to a cardiac disorder. Ventricular dysfunction was defined on the basis of echocardiography and/or by cardiac catheterisation in the case of left ventricular disease, according to accepted criteria. Radio isotope studies (mibiscan) were a better method of defining the structure and function of the right ventricle; but scans of both ventricles were scrutinised. Patients in atrial fibrillation were first slowed down and the ventricular rate controlled before functional studies were done, either by mibiscan or echocardiography. Echocardiography was also employed to assess ventricular size, left ventricular wall dimensions and motion abnormalities and for a detailed study to exclude valvular dysfunction and intra-chamber abnormalities (thrombi). A ventilation-perfusion scan was performed in all cases of right-sided disease, despite the fact that all patients with severe pulmonary hypertension and any abnormality of the right ventricle (echocardiographic or on catherisation) were excluded. Cardiac catherisation, in addition to standard procedures for left and right heart study, was specifically employed to determine pulmonary pressures, pulmonary wedge pressure (for left ventricular dysfunction, especially) and ventricular end-diastolic pressures. Minimal coronary artery disease was defined as the presence of less than 40% stenotic lesions, not more than one lesion per vessel, and not more than two vessels thus affected. Patients with significant coronary artery disease were excluded. There were 7 such patients. Only the 24 patients with left ventricular disease who fulfilled our criteria were included (Table IV). Of the patients who underwent coronary arteriography, only 5 had minimal disease. All were men, aged 52 - 74 years. These patients all happen to have lone left ventricular disease. Peripheral phlebography was undertaken in most cases of right-sided disease, while pulmonary angiography was undertaken in all. Coronary arteriography was omitted in 4 patients with right-sided disease. A total of 35 patients underwent coronary arteriography. Artefactual signs of dysfunction were recognised in a few cases, but this did not interfere with important critical decisions. Of particular note is that although determination of an ejection fraction is not as precise in the presence of atrial fibrillation, reduced function in the presence of altered dimensions was a useful additional abnormality to weight a decision, provided atrial fibrillation was controlled.

Table II. 1994 medical hospital statistics

1.	Total medical admissions	= 5 684
2.	Medical (general) consultations — outpatients	= 11 152
3.	Hypertensive heart clinic consultations	= 2 277
4.	Cardiology clinic consultations	= 3 609

Consultations are not all 1-patient consultations. Admissions include re-admissions. Follow-up clinics' repeat visits are largely one visit per month by each patient.

Table III. Criteria for a ventricular disease

Lone left ventricular disease — criteria
- No present or known past systemic hypertension
- No organic valvular heart disease — acquired or congenital (including shunts)
- No clinical echocardiographic or radio-isotopic evidence of structural or functional disorder of the right ventricle except pulmonary hypertension in some cases
- Demonstrable left ventricular disease — symptomatic or asymptomatic — on clinical grounds, electrocardiography, echocardiography and catheterisation data (pressures, ventriculogram)
- Healthy coronary vessels or very minimal disease, if any
- No diabetes mellitus

Lone right ventricular disease — criteria
- No structural or functional disorder of the left ventricle
- No pulmonary hypertension or primary or secondary disorder of the respiratory system, including the vasculature
- No intracardiac or extracardiac shunts
- Demonstrable structural and/or functional disorder of the right ventricle — with or without clinical presentation — on echocardiography, radio-isotope studies and catheterisation data (pressure, ventriculogram, angiogram)
- Healthy coronaries or minimal disease, if any

Table IV. Investigations specifically undertaken to further definitions of ventricular disease

Basic electrocardiography	(all patients)
Exercise stress test	(12)
Chest radiograph	(all patients)
Transthoracic echocardiogram	(all patients)
Transoesophageal echocardiogram	(10)
Standard left and right heart catheterisation	(24)
Right heart catheterisation only	(10)
Coronary arteriography	(24)
Pulmonary arteriography and right ventriculogram	(10)
Radio-isotope studies	(20)
Ventilation-perfusion scan	(8)
Routine laboratory tests, including serology	(all patients)
Phlebography	(8)
Cardiac enzyme tests	(2)
Mibiscan	(14)

Number of patients who underwent tests are shown in brackets.

Results

The 30 subjects studied are shown in Table V, and are obviously a very small fraction of the patient populations at different levels of the hospital (Table II). Both sexes are represented, with an age range of 18 - 74 years, and an average of 48 years. There are more men overall. There were 6 women with right ventricular disease out of 8 patients. Of the 22 patients with isolated left ventricular disease, nearly three-quarters are men. Five asymptomatic subjects were discovered with left ventricular disease, compared with only 1 with right-sided cardiomyopathy. The only 2 pregnant patients in the series had isolated left-sided disease. This was not unexpected. Atrial fibrillation occurred in 5 patients, all with right-sided cardiomyopathy. In our experience atrial

fibrillation does occur in classic dilated cardiomyopathy, seemingly in the chronic phase. Electrocardiograms were normal in 5 patients with left-sided disease, and in 1 with a right-sided ventricular disorder. The 'electrocardiographic pattern' of myocardial infarction seemed more likely to be mimicked by left ventricular disease. Sinus rhythm was present in 25 cases. The electrocardiogram voltages were normal in all except 4 patients. Of the latter, 3 satisfied voltage criteria for left ventricular hypertrophy and 1 had low QRS forces. The QRS axes were inconsistent, while primary nonspecific T-wave changes were common in left ventricular disease.

Table V. Ventricular disease categories

Sex	Left ventricle	Right ventricle	Total
Men	15	2	17
Women	7	6	13
Total	22	8	30

Discussion

The primary purpose of this exercise was to discover and verify the existence of univentricular cardiomyopathy, if any, especially with regard to right ventricular disease. We suspect that early cardiomyopathy is a left-sided ventricular problem. We are aware that early idiopathic cardiomyopathy is generally assumed to be a left ventricular disease. Our series, however, is too small to allow firm conclusions on either typical characteristics or any differences between the possible two categories. There is evidence, although scant, that univentricular forms of cardiomyopathy do exist. Most literature is on the classic biventricular disorder of cardiomyopathy. The right ventricular form is a newly described entity. More work needs to be done to support this notion and to define accurately the features of these subtypes, as well as the relationships between them. The absence of histopathological support in the series reported does not argue strongly against the postulate that these were cases of idiopathic cardiomyopathy. Biopsy is neither routinely recommended nor of material benefit in the general work-up of cardiomyopathies.[8]

The description of primary myocardial disorders as diseases characterised by loss of contractile systolic function together with appropriate symptoms and signs has varied over recent years. This terminology was biased in favour of left ventricular dysfunction, which is a primary component of a clinical syndrome that commonly manifests as biventricular cardiac failure with classic four-chamber dilatation. With modern-day techniques, cardiomyopathy can readily be distinguished from other diffuse myocardial disorders, such as end-stage and late valvular lesions, ischaemic heart disease, hypertension, cor pulmonale and 'silent' congenital shunts. It is implied, and has been accepted, that the term 'cardiomyopathy', as coined by Goodwin and Oakley,[6] describes the end result of diverse (yet unknown) causes and is always initially a left ventricular disorder. But insufficient emphasis has, however, been placed on 'pure left ventricular disease', as an entity in its own right, especially at an early or presymptomatic stage.

In our practice, cardiomyopathy usually manifests as a biventricular four-chamber dilatation. The reasons for late presentation are socio-economic and cultural. Among our patients with pure left ventricular disease we excluded many with pulmonary hypertension and possible early right ventricular involvement. We recognise that classic idiopathic cardiomyopathy has a typical clinical spectrum. The patients with left ventricular diseases are therefore a highly select category in this series.

Another subtype of cardiomyopathy that has since been well recognised is peripartum cardiomyopathy.[10,11] This manifests predominantly as a left ventricular disease or a typical biventricular disorder. It is clear now that pregnancy is merely incidental, and may serve as an additional trigger or provide conditioning circumstances, e.g. in Nigerian women. Earlier reports had suggested erroneously that peripartum cardiomyopathy was a specific entity.[12,13] Right ventricular cardiomyopathy may be another subtype, as was recently suggested.

Many workers in the field are familiar with the epidemiology of (classic) cardiomyopathy, such as the slight male preponderance, and the typical diagnostic clinical features, taken in combination. The myopathic status is readily demonstrated. Landau and co-workers,[9] in a case description, comment that right ventricular idiopathic dilated cardiomyopathy is rare. They further pose the question of whether this subtype is part of a continuum of disorders, including Uhl's anomaly and right ventricular dysplasia, both arrhythmogenic and non-arrhythmogenic. They allege that right ventricular cardiomyopathy has been overlooked, on account, *inter alia*, of technical difficulties inherent in the study of the complex right chamber. Braunwald,[8] however, discusses the entity of right ventricular cardiomyopathy with no reservations. He also alerts readers to the possibly outdated or inappropriate use of the term 'dilated' in the present-day definition of these cardiomyopathies. Lastly, it must be pointed out that the complexities of this heterogeneous disorder, with its subtypes, are still many. The aetiology is obscure, and malnutrition and alcohol toxicity are not completely excluded.[1,2] There remain no agreed criteria with regard to prognosis or natural history. There are still gaps in our knowledge of the evolution of some subtypes (e.g. whether it is right-sided first or left-sided first at onset). It is therefore of interest to make some further observations on the subject, summarised as follows:

1. In a study of 31 patients, left ventricular involvement was reported in primary initial right ventricular disease,[14] in 19 in whom the left ventricle was initially normal.
2. There was a subset of subjects in whom the degree of biventricular involvement was discordant[15] — morphological subsets with possible implications for clinical outcome. It would seem that severe left ventricular disease (defined as dilatation) confers an unfavourable outcome.
3. The distinction between selective right ventricular cardiomyopathy and a dysplastic right ventricle[16] can be made by histology and recognition of certain inflammatory forms of right ventricular disease.[17]

The discovery of early selective left ventricular disease is not unexpected. In fact, we believe this to be the initial stage of a common disease. With the successful implementation of primary health care strategies, we believe that this subtype may emerge as the commonest form —

silent, early symptomatic or even with unusual manifestations.[18] A selective right ventricle subtype is rare and more difficult to identify, especially in a practice like ours, where some type of cor pulmonale is commoner. However, we excluded all right ventricular disorders with pulmonary hypertension. These preliminary findings are interesting and exciting, especially the discovery of right ventricular cardiomyopathy. The shortcomings of our study need to be highlighted, however: (*i*) selective ventricular disease may only be present in initial stages — follow-up is necessary to describe the natural history. This was not done; (*ii*) organic tricuspid regurgitation is not easy to define or to distinguish from secondary valvular dysfunction, yet this 'lesion' is common in right ventricular cardiomyopathy. One patient who had undergone an operation seemed to have had organic tricuspid valve disease, possibly unexplained infective endocarditis; (*iii*) we cannot claim to have collected representative cases and many were possibly missed. Too few cases were analysed; (*iv*) the absence of histological study material is a disadvantage; and (*v*) all patients were studied invasively after treatment, when some parameters might have changed. It is questionable whether it is possible for any hypertension, pulmonary or systemic, to drop on account of severe ventricular dysfunction.

Conclusion

A preliminary study has highlighted 30 cases with selective univentricular disease. The subjects, consecutively collected from a central consultative cardiology service, had to fulfil special criteria. A wide range of investigations was used for the diagnosis of lone disease as dysfunction, dilatation or both. The main purpose was to ascertain the existence of univentricular cardiomyopathies. There were 22 subjects with left ventricular disease and 8 with isolated right ventricular cardiomyopathy. The latter is a recently recognised but rare entity. The subtypes of cardiomyopathy, a disease of obscure origin and uncertain pathology with a multitude of environmental causes, include these ventricular subtypes, clinicopathological subtypes in each category and peripartum cardiomyopathy. The relevant literature has been reviewed and the nuances of cardiomyopathy subsets discussed. Some limitations of the study were highlighted after the subject had been discussed in terms of the history, the terminology, pathology and clinical aspects. Further work needs to be done and more questions answered. The relationship between selective ventricular diseases must still be defined, and more knowledge on cardiomyopathy in general must be obtained in respect of other issues — prognosis, pathology and the place of univentricular cardiomyopathy in the spectrum of heart muscle diseases of obscure origin, which may well form a continuum of possibly related disorders. Right ventricular cardiomyopathy may exist as an entity, though rarely.[8,17] However, its natural history remains to be elucidated.[14-16] The findings of some workers suggest that right ventricular cardiomyopathy may predate left ventricular dysfunctions; this has yet to be proved. We continue to believe that classic dilated cardiomyopathy is always initially a left ventricular problem. The patients in this series, whose condition has been described as isolated left ventricular cardiomyopathy, have an early form of idiopathic dilated cardiomyopathy.

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