Cardiomyopathy in the Southwest American Indian^{1,2}

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Six Southwestern American Indian patients are reported to have primary myocardial disease or congestive failure of unknown cause. Two are Navajo, three are Laguna, and one is Isleta. Clinical and laboratory features are discussed. This disease appears in culturally traditional Indians and is not associated with contact with American civilization. It occurs in probably adequately nourished Indians in the majority of whom excessive alcohol consumption is not found. No familial cases were noted. This syndrome is probably not uncommon in the American Indian.

Heart failure of obscure cause, or cardiomyopathy, has been described in population groups around the world. There are reports from Uganda, Nigeria, Ghana (Davies and Ball, 1955; Edington, 1954; Shillingford and Somers, 1961), England (Barry and Hall, 1962), U.S.A. (Fowler, Gueron, and Rowlands, 1961; Harvey, Segal, and Gurel, 1964; Dye *et al.*, 1963a, b; Meadows, 1957; Massumi *et al.*, 1965; Thomas *et al.*, 1954), North Brazil (Andrade and Guimarães, 1964; Guimarães and Andrade, 1962), South Brazil (Mattos *et al.*, 1963; Fagundes, 1963), Tanganyika and Kenya (Turner and Manson-Bahr, 1960), Congo territories (Peuchot, Latour, and Puech, 1960; Coelho and Pimentel, 1963), Gabon (Fournier *et al.*, 1961), West Africa (Abrahams and Bridgen, 1961), South Africa (Higginson, Isaacson, and Simson, 1960), Asia and Ceylon (Nagaratnam and Dissanayake, 1959), Colombia (Correa *et al.*, 1963), Jamaica (Stuart and Hayes, 1963), etc.

We are reporting 6 American Indians with heart failure of obscure cause. Since the aetiology of these syndromes is unclear, special attention was given to lineage, acculturation⁴,

family history, diet, and alcohol consumption. The purpose of this report is to call attention to the fact that this syndrome occurs in the American Indian and to investigate some of the factors thought to play a role in the aetiology.

Subjects and methods

The patients were seen in the Bernalillo County Medical Center or the Veterans Administration Hospital. The medical history in each case was taken by a physician and a trained American Indian medical interpreter. The tribal identity of each patient, his parents, and grandparents was expressed in fractions (see Table 1); the first fraction represents the number of parents of the same tribe as the patient out of the two parents possible, the second fraction represents the number of grandparents of the same tribe as the patient out of the four grandparents possible. The degree of acculturation was measured in a grade from 0 to 4. The purely traditional life is represented by the grade of 0; the fully acculturated life is represented by the grade of 4. Upbringing, educational accomplishment, service in the Armed Forces, occupation, travels, living, social and religious practices, and skills with the English language were all considered in this classification. The family history of heart disease symptoms was elicited.

The Indian interpreter collected dietary data in three ways: by a recall of the food intake of the previous 24 hours, or a representative 24 hours, by measuring the amount, frequency, and method of preparation of specific foodstuffs eaten, and by measuring the amount and frequency of sugar and flour purchased at the trading post (Trulson, 1960; Weir and Houser, 1963). These dietary data were then evaluated for nutrition and for degree of acculturation on a 0 to 4 basis. On the nutritional

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⁴ Webster's dictionary: A process of intercultural borrowing between diverse peoples.

scale 0 represents a starvation diet and 4 excellent nutrition. On the dietary acculturation scale 0 is traditional and 4 fully Anglo-American diet. The alcoholic intake also was graded from 0 to 4, where 0 referred to total abstinence and 4 to continuous drinking. The severity of congestive heart failure was classified according to the New York Heart Association criteria (1964).

The physical examination was complete, including special attention to nutritional state and to cardiovascular function. Patients with hypertension, valvular disease, congenital heart disease, historical, or electrocardiographic evidence of coronary artery disease were excluded.

The cardiac output was determined either by the direct Fick method, or the dye-dilution method, using Cardiogreen, a Waters X-300 Densitometer, and an Electronics for Medicine DR-8 Recorder. Oxygen consumption was measured by the open-circuit method. Analyses of brachial arterial and pulmonary artery samples for oxygen were performed in duplicate in the Van Slyke apparatus. Analysis of expired air was performed in duplicate in the Scholander microgasometer. Brachial arterial pressure and pulmonary arterial pressures were recorded with Statham 23 Db gauges and Electronics for Medicine DR-8 Recorder. Arteriovenous oxygen differences were either measured when cardiac catheterizations were done, or calculated from the Fick equation when dye-dilution cardiac outputs were measured. Exercise consisted of alternate leg raising in the supine position with expired air, arterial pressure and cardiac output being determined during the fourth to sixth minute. Red cells labelled with hexavalent anionic chromium 51 (Radiochromate-51¹) and albumin labelled with ¹²⁵I (RISA-125¹) were used to measure the red cell volume and plasma volume simultaneously (Wood and Levitt, 1965) in a twin-scintillation counter and transistorized computer (Volemetron²) (Williams and Fine, 1961).

Results

The 6 Indian patients with heart failure are identified in Table 1. They ranged in age from 41 to 84. Four were men and two were women. One came from the Isleta Pueblo, three from the Laguna Pueblo, and two from the Navajo reservation. Their lineage was pure for their tribal affiliation back through their grandparents with the exception of one (Case 5). On the traditional acculturation scale, two patients were given grade 0, which meant they were fully traditional. None were considered fully acculturated, but Case 2 was given a grade of 3. In general, the patients were more traditional than acculturated.

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TABLE 1 Identification of 6 Indian patients with primary myocardial disease

Case No.	Age (yr.)	Sex	Tribe	Lineage*	Traditional-cultural grade
1	41	M	Isleta	2/2-4/4	2
2	43	M	Laguna	2/2-4/4	3
3	63	F	Laguna	2/2-4/4	2
4	83	M	Laguna	2/2-4/4	0
5	84	M	Navajo	2/2-1/4	0
6	63	F	Navajo	2/2-4/4	2

* See text for explanation.

Other aspects of the history are given in Table 2. The family history was entirely negative in all six patients. There was no consistent pattern in the nutritional history. Two of the patients had an entirely adequate diet, while two had a marginal diet. Cases 1 and 2 were inadequately nourished while drinking but adequately so when not. Only two were given a grade of 3 on the alcoholic scale. The congestive heart failure ranged in severity from I to IV and in duration from one month to 14 years. The current status of the patients is also seen in Table 2.

Certain parameters of the cardiac physical examination are seen in Table 3. Essentially all patients were normotensive. All the patients had abnormal rhythms at one time or another, but four were basically in sinus rhythm. Car-

TABLE 2 Historical features of 6 Indian patients with primary myocardial disease. See text for description of scales.

Case No.	Family history	Dietary nutritional scale	Dietary cultural scale	Alcoholic scale	Congestive heart failure	Current status	
					Severity	Duration (yr.)	
1	Neg.	1-3	3	3	III	4	Died, suddenly
2	Neg.	1-4	3	3	IV	14	Alive, chronic heart failure
3	Neg.	2	2	0	III	3½	Dead, intra-abdominal catastrophe
4	Neg.	2	2	0	II	1/12	Dead*, with syndrome of pulmonary infarction
5	Neg.	4	0	0	II	8/12	Died, suddenly
6	Neg.	4	2	0	I	4	Alive, in fair condition

* Necropsy showed (1) organic heart disease of unknown aetiology, with congestive heart failure, acute and chronic; (2) multiple pulmonary thromboemboli, bilateral lungs; (3) multiple pulmonary infarcts, left lower lobe; and (4) bronchopneumonia, bilateral, moderate.

TABLE 3 Selected cardiac findings on physical examination, electrocardiogram, and chest x-ray

Case No.	Blood pressure (mm.Hg)	LV lift or RV thrust	S-3	Electrocardiogram	X-ray
1	108/72	Present	Constant	Occ. prem. ventric. contraction, left ventric. hypertrophy, sinus tachy. Non-specific ST-T changes, atrial abnormality	Cardiomegaly, left ventricular hypertrophy, pleural effusions, passive congestion of lungs
2	143/86	Present	Constant	Atrial fib. and flutter, with sinus bradyc., and paroxysm. ventric. tachy., left ventric. hypertrophy, antero-lateral T wave changes	Cardiomegaly, left ventricular and left atrial hypertrophy, old pleural reaction in left costophrenic angle
3	140/70	Present	Constant	Various arrhythmias, non-specific ST-T changes	Cardiomegaly, right pleural effusion, passive congestion of lungs
4	136/88	Present	Absent	Minor non-specific ST-T changes, occ. prem. ventric. contraction	Cardiomegaly, chronic inflammation left lower lobe, old fibrocalcific disease in both apices
5	118/60	Present	Variable	Transient atrial fib., occ. prem. ventric. contractions, LBBB, abnormal left axis deviation	Cardiomegaly, right pleural effusion, passive congestion of lungs
6	132/70	Absent	Absent	Transient prem. ventric. contractions and transient abnormal left axis deviation	Minimal cardiomegaly, small fibrotic strands both lung bases, adhesions left costophrenic angle

diomegaly as defined by a left ventricular thrust or a right ventricular lift was found in all but one (Case 6). The second heart sound was physiologically split in all. An S₃ was present in four out of six. An S₄ was heard only variably in one. Four patients had a murmur heard when in heart failure but no murmur of significance when compensated. The electrocardiograms and chest x-rays were abnormal in all patients. Cardiomegaly was defined on x-ray as a cardiothoracic ratio of 50 per cent or greater.

Three of the patients (Cases 3, 5, and 6) were mildly anaemic and iron deficient. Two patients had reactive Venereal Disease Research Laboratory titres with positive fluorescent treponema antibody tests, but neither had organ involvement. Case 6 had been previously treated, while Case 4 was treated for the first time. In five the blood urea nitrogen was within normal limits when the patients were first seen but subsequently in four of the six it became increased with the congestive heart failure. The blood sugar was normal in all but two (Cases 2 and 5). In the patients who had examination of total proteins, only one had a relatively low total protein (Case 6) and only one had a reversal of the A/G ratio (Case 1).

Physiological and haemodynamic data were collected in four of the six patients (Table 4). Two underwent cardiac catheterization which

was repeated to assess the effects of prolonged bed-rest. In one (Case 1) bed-rest appeared to be beneficial, and in the other (Case 2) it did not. Blood volumes were measured to assess the degree of circulatory congestion, and these correlated well with clinical impression. The haemodynamic pattern was characteristic of low output congestive failure without restrictive defects, which is found in constrictive pericarditis or infiltrative myopathies, etc. The initial measurements were made after hospital management of bed-rest, dietary sodium restriction, digitalis, and diuretic therapy. Despite treatment, most patients showed on their initial study a low resting cardiac index. In general, the arteriovenous oxygen difference was increased, and when measured, right heart pressures were increased. Initial blood volume data showed increases above normal despite diuretic therapy. In general, when exercise was performed, there was an inadequate increase in cardiac output with an excessive widening of arteriovenous oxygen difference. One patient (Case 6) was asymptomatic clinically and almost normal physiologically at the time she was studied. She had been in congestive heart failure previously on two occasions and had been successfully treated.

In only one of these patients was necropsy obtained (Case 4), which showed cardiac dilatation with a heart weight of 380 g., evidence of

TABLE 4 *Physiological and haemodynamic findings in 6 Indian patients with primary myocardial disease*

Case No.	Date	Heart rate/min.	Cardiac index* (l./min./m. ²)	O ₂ consumption (ml./min./m. ²)	Pressures (mm.Hg)				Volumes (ml./kg.)			
					PA	RV	RA	BA	Red cell	Plasma	Total blood	
1	19/10/66	Rest	120	2.5 ^F	178	—	63/?	6	119/90	38.3	45.4	83.7
	17/11/67	Rest	86	3.5 ^F	168	24/8	30/0-7	4	136/80	27.9	44.2	72.1
2	23/7/65	Rest	100	2.0 ^F	127	—	45/13	15	143/86	—	—	—
	13/7/66	Rest	50	1.2	145	—	—	—	132/64	31.6	47.5	79.1
3	13/7/66	Exercise	84	1.4	240	—	—	—	—	—	—	—
	12/7/67	Rest	63	1.9	183	—	—	—	158/90	—	—	—
4	12/7/67	Exercise	92	2.5	240	—	—	—	—	—	—	—
	25/10/67	Rest	68	1.9 ^F	143	85/70	85/18	14	170/90	36.8	70.8	107.6
5	20/1/66	Rest	60	2.0	113	—	—	—	107/42	34.1	59.4	93.5
	20/1/66	Exercise	72	1.8	241	—	—	—	—	—	—	—
6	7/11/66	Rest	69	2.7	115	—	—	—	123/55	24.9	39.0	63.9
	7/11/66	Exercise	87	4.9	394	—	—	—	180/66	—	—	—

* Four cardiac outputs were done by the Fick method, indicated by superscript F, the others were done by dye method.

acute and chronic passive congestion, and multiple pulmonary emboli. There were no endocardial lesions. There was minimal interstitial fibrosis and no evidence of myocardial infarction. The coronary arteries were of normal calibre with only slight atherosclerotic plaquing of the intima.

Discussion

Congestive heart failure of unknown cause has been found in populations in many parts of the world; it is here reported in the Southwestern American Indian. This clinical syndrome probably represents many aetiological entities, as the heart has limited means of manifesting functional impairment, congestive heart failure being one of the common ones. After excluding the heart disease associated with Chagas' disease, haemochromatosis, amyloid, muscular dystrophy, certain eosinophilic states, glycogen storage abnormalities, etc., a group of patients with heart failure remains. These have been called cardiomyopathy or heart failure of obscure cause. Even in this group, various pathological entities have been described. Idiopathic hypertrophy with dilatation has been found (Fowler *et al.*, 1961). Adult patients with hypertrophy and considerable fibroelastosis have also been reported (Thomas *et al.*, 1954). The group with conspicuous endomyocardial fibrosis seen in Uganda certainly seems to form a distinct pathological entity.

In the United States and England heart failure of obscure cause may be in some cases related to alcoholism (Regan *et al.*, 1969), in others to genetic and familial factors (Evans, 1949; Gaunt and Lecutier, 1956; Schrader *et*

al., 1961; Beasley, 1960; Campbell and Turner-Warwick, 1956; Paré *et al.*, 1961; Treger and Blount, 1965), and possibly to pregnancy (Meadows, 1957). A tribal predisposition has been described (World Health Organization, 1967). In some it may represent a metabolic defect, or malnutrition of an unknown nutrient, or combinations of these factors. It is thus of interest to note heart failure of obscure cause in a population group in the United States in whom this disease has not been reported.

There are two main groups of Southwestern American Indians, the Athabaskan and the Pueblo, with different languages and customs. The Navajo and the Apache are Athabaskan Indians who were nomadic hunters who arrived some five or six hundred years ago. They found the river watering places occupied by small towns, or Pueblos, inhabited by shorter, more placid, farming Indians. Heart failure of obscure cause has been seen in both the Navajo and Pueblo groups. The patients listed as originating in Laguna and Isleta (Table 1) are Pueblo Indians.

The incidence of heart disease in the American Indian is not well documented. Coronary artery disease is thought to be uncommon (Streeter *et al.*, 1960; Fulmer and Roberts, 1963; Clifford *et al.*, 1963; Hesse, 1964; Sievers, 1967). Some believe heart failure is also uncommon. The stoic Indian accepts slowly developing chronic illness as natural and does not often complain of dyspnoea and exercise intolerance. However, heart failure was not infrequently seen by the authors in Indian reservation hospitals, and a retrospective survey was conducted in four hospitals caring for

TABLE 5 *Hospitals caring for Southwest American Indians, which were surveyed for heart failure patients*

Location	Dates	Total deaths	Heart failure	Congestive heart failure obscure cause
Bernalillo County Medical Center, Albuquerque, New Mexico	May 56-Dec. 63	1,810	35 Pueblo	9
Sage Memorial, Ganado, Arizona	Jan. 49-Dec. 63	863	14 Navajo	6
USPHS Hospital, Shiprock, New Mexico	Jan. 60-Dec. 63	327	40 Navajo	12
USPHS Hospital, Fort Defiance, Arizona	June 57-Dec. 63	541	32 Navajo	10
			18 Navajo	?

Indian patients. This is shown in Table 5. Considering the great rarity of coronary artery disease, heart failure is not uncommon and heart failure of obscure cause is not rare. The 6 patients we are reporting were ones carefully studied by us. The general history and physical examination revealed findings one would expect with heart failure of obscure cause, as did the general laboratory findings. Cardiac catheterization confirmed that most of the symptomatic patients had low cardiac output and did not have restrictive myocardial defects.

The role of acculturation was studied. These patients were predominantly traditional in their outlook, eating a relatively traditional diet and living in traditional housing with traditional customs. The traditional Indian way of life involves at least a moderate level of exercise, predominantly walking, eating certain foods, certain religious beliefs, etc. The development of heart failure of obscure cause is certainly not dependent on contact with modern technological culture, and in fact some feature of the more 'primitive' culture may play a role in this disease.

In none of the patients studied was there any significant family history of heart failure. The American Indian reveres his ancestors and family life is well structured so that any major familial disease would be remembered by the patients.

In Uganda, especially in tribes from Ruanda, endomyocardial fibrosis is common. We have found no special tribal predilections, Pueblo and Athabaskan both having this syndrome. Some workers claim a specific effect of alcohol on the heart (Regan *et al.*, 1969). It certainly seems true that this is a common disease in the American Municipal and Veterans Administration Hospitals where the population comes from a low socio-economic group in which alcoholic consumption is also common. Only two patients in this group had a history of alcoholism. This supports the idea

that alcoholism is either only a contributing factor, or is aetiological in only some examples of this syndrome.

Dietary deficiencies have been implicated in the aetiology of heart failure of obscure cause in Africa. There is some overlap in the kwashiorkor area and that in which heart failure of obscure cause is common, especially that form associated with endomyocardial fibrosis. The Southwest American Indian has great variation in protein intake, and in some of the Pueblos this may be restricted but is probably adequate. These people seem well nourished on physical examination and have no stigmata of vitamin deficiencies. The Athabaskan Indians go through periods of relative famine when the diet consists mainly of corn, grain, etc., and at times through periods of good nutrition when lamb is consumed, so the diet has some intermittent periods of undernutrition. For the most part, these people are not malnourished on physical examination and by taking a dietary history.

The only patient of the six reported in whom a necropsy was available suggests that this type of heart failure is of the idiopathic cardiomegaly or idiopathic hypertrophy type, without significant endocardial lesions, and resembles the pathology described in other American series (Fowler *et al.*, 1961).

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