
MEDICAL HISTORY

CORONARY ARTERY DISEASE IN BLACK AMERICANS 1920-1960: THE SHAPING OF MEDICAL OPINION

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Current opinions regarding the prevalence of coronary artery disease in black Americans are conflicting. Some physicians believe that the prevalence of coronary artery disease in black Americans is less than that in the general population; some find no difference; still others argue that the high prevalence of risk factors, such as hypertension, should result in a higher prevalence of coronary artery disease in black Americans. This article will not attempt to resolve these conflicts but instead will review some of the medical literature that may have influenced prevailing opinions.

Certain segments of the health care establishment have maintained a not too innocent naivete regarding the importance (ie, prevalence) of coronary artery disease in blacks and other minority populations in the United States. Weighing the relative influence of medical literature versus clinical teaching in this matter is interesting, but beyond the scope of this article. Medical opinions appearing in print are probably less prone to careless generalizations than those expressed during daily clinical discourse, principally because published opinions are more carefully formulated and are subject to profession-wide scrutiny. Statistical analysis, which

evolved during the latter half of this century, has further refined the credibility of medical opinion. A review of earlier literature, however, may give us a glimpse of how we have arrived at an age of unprecedented technical ability in a setting of unequal access to costly diagnostics (coronary angiography) and therapeutics (reperfusion and revascularization). Regardless of the strengths or weaknesses of various epidemiologic studies, attitudes that inject a subjective bias may make common problems seem rare, and rare problems are often ignored. Questions regarding physician and patient attitudes brought forth by the Heckler Report¹ have been addressed previously, but a more in-depth analysis is necessary.²

Numerous investigators have written on the subject of racial differences in coronary artery disease; Gillum's reports represent a good example.^{3,4} His work shows that, based on vital statistics, the mortality and morbidity from coronary artery disease in black versus white men is similar, and black females seem to have a slightly increased prevalence over their white counterparts. Gillum points out, however, that the possibility for error in reported statistics may be large. He specifically states that the mortality statistics for nonwhites as a heterogeneous group are inaccurate because minority population percentages vary with time, census data typically underestimate the numbers of minorities, and death certificate errors may be frequent. These potential inaccuracies imply an incidence of coronary artery disease that is less than the true incidence in blacks. This implication is at variance with what appears to be an increased prevalence of coronary disease risk factors in the US black population.⁵⁻⁷

The importance of accurate epidemiologic studies cannot be overemphasized; they have the potential to

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influence physician behavior and decrease reliance on anecdotal, potentially biased, data. Fortunately, physicians and institutions that give differential care based on anecdotal data, race, or socioeconomic status, or who restrict clientele on such grounds are becoming less common.^{8,9} Although this type of information is only a curiosity it should lend some historical perspective to the current concern about differences in coronary artery disease in white and nonwhite Americans.

After reviewing the early literature on the incidence of coronary artery disease in blacks, it is understandable why the medical community regarded this disease as a rarity. Angina pectoris as a specific symptom complex has been described since 1768¹⁰ and early in its history became loosely associated with the wealthy. A reading of Osler's description of angina pectoris will serve today's medical students as well as those found in recent literature.¹¹ He did not seem to attach any racial bias to the symptoms. Both then and now chest discomfort was not tied to a single pathophysiological process.¹²

The treatment of black Americans during the first half of this century was not surprisingly reflective of prevailing attitudes. In an article titled "The Incidence of Heart Disease in the Negro Race" by Woody¹³ in 1924, no mention is made of coronary artery disease or angina pectoris. From experience in Virginia, he claimed that syphilis and hard manual labor produced an inordinate degree of aortic valvular disease and other cardiac lesions in blacks: "As for venereal disease, indiscriminate sexual indulgence and lack of regard for personal cleanliness and prophylaxis, together with a lack of knowledge of the seriousness of both the condition itself and its possible consequences, leads to a relatively much higher rate of occurrences of both syphilis and gonorrhea in the Negro." Regarding cardiac disease in black children, he blamed parents for not following medical advice: "It is very characteristic for them to live today and let tomorrow take care of itself."

The basis for these ideas had already been promulgated by others when Dublin,¹⁴ in 1922, speaking for the Metropolitan Life Insurance Company, stated that ". . . the greater prevalence of heart disease among colored people is notorious. Colored males show rates of heart disease during the main period of life from 65% to 80% higher than for white males at the same ages; those for colored women are twice as high as for white women at a number of age periods of life. Possibly, the higher prevalence of such diseases as syphilis, malaria, and typhoid fever in the colored race plays an important part in creating the excess of heart disease mortality." The eventual recognition of high risk for cardiac disease

including coronary artery disease¹⁵ combined with a decreased likelihood of early treatment or prevention must have had implications for the insurability of black citizens.

The report on "The Etiology of Heart Disease" in 1926 by Wood, Jones, and Kimbrough¹⁶ is a combined study from two geographic locations: Virginia and Massachusetts. The 300 patients from Virginia consisted of 112 blacks and 188 whites. Of the total, 70 were "private room" cases, presumably all white. Of the 323 patients reported from Massachusetts General Hospital, all were white and none were "private room" cases. The etiologic and pathologic categories tabulated are complex and unclear. The classification of heart disease used in that day left room for a considerable degree of subjectivity. Although their methods were not scientific, they arrived at conclusions about race and heart disease that may have contributed to the literature that followed.

They recognized that a large proportion of all organic heart disease was attributed to hypertension and arteriosclerotic conditions regardless of race or geographic region. Rheumatic heart disease was more important in the Northeast compared with the Southeast but in blacks it was considered less important than syphilitic cardiovascular disease as a cause for morbidity and mortality. Although their tabulated data indicated that angina pectoris was present in only two white patients of the entire population (623), the body of their paper reported a higher incidence of "paroxysmal heart pain" in whites (10.2%) than in blacks (5.3%).

The insightful description of angina pectoris by Roberts¹⁷ in 1931 probes the lifestyle components now recognized as "type A" personality. His account is vivid and, ironically, coupled with the most crass forms of racial stereotyping. He used his bias to invalidate angina pectoris as a cause of chest pain in blacks and Asians and concluded that it was a rarity in these groups. In other papers of this period, personal opinions regarding race and socioeconomic status influenced the diagnosis of angina pectoris, but to a lesser degree. All of these works contain useful information. If nothing else, they report the early recognition of regional variation in the incidence of coronary disease, which was perhaps more related to economic status and lifestyle differences between the industrial urban North and the more agricultural South than to race. The marked regional differences in death rates in patients with angina pectoris cannot, however, be equated to death from coronary artery disease, regardless of racial distribution.¹⁸

In 1927, Stone and Vanzant¹⁹ reported on the spectrum of heart disease at the John Sealy Hospital (Univer-

sity of Texas) in Galveston, Texas, over a seven-year period. Black admissions for heart disease were almost twice as frequent (on a percentage basis) as white admissions. They concluded that heart disease was "1.8 times more common in the negro than in the white." By 1931 and 1932, Schwab and Schulze,^{20,21} from the same institution, made a more complete study. They pointed out that because charity patients did not enter the hospital until "they [were] in dire need of hospitalization" and "facilities for Negro charity patients [were limited] as compared with white charity patients," an accurate incidence study was not possible.

A similar pattern emerged in both studies: hypertensive heart disease was prevalent in both races but was consistently higher in blacks in whom onset occurred at a younger age. Syphilitic heart diseases were more prevalent in blacks and arteriosclerotic heart diseases were more common in whites. Angina pectoris was rare in whites and nonexistent in blacks. When angina pectoris did occur in whites, it was almost always diagnosed in "private room" patients. The methodology of disease classification was similar to that used by Wood et al¹⁶; patients were assigned to disease classification, (ie, arteriosclerotic heart disease, coronary artery disease, or syphilitic cardiovascular disease) based on uncertain criteria. Regarding the diagnosis of syphilitic cardiovascular disease, Schwab and Schulze stated, "emphasis was placed largely on the discovery of one of the characteristic structural changes; namely aortitis, aneurysm, or aortic insufficiency, or a combination in the presence of a positive Wassermann reaction, a positive history, or lesions in other parts of the body pathognomonic of a syphilitic infection." The likelihood of assigning a syphilitic origin to lesions that could have another etiology throws doubt on certain aspects of their study and the implications for both blacks and whites. Coronary artery disease and syphilitic aortic stenosis as causes of angina pectoris were certainly not mutually exclusive. In addition, the false positive rate of the Wassermann test could not have been less than that of later reagent tests (ie, 20% to 40% depending on the population studied²²⁻²⁴). The dermatologic diagnostic skills of physicians dealing with black patients at that time is also somewhat in doubt.^{25,26} Aneurysmal disease of the aorta and subsequent aortic valvular disease in a population with a known higher incidence of severe hypertension further compounds the uncertainties of these early studies.

The account by Laws²⁷ in 1933, from Vanderbilt, also offers a black-white comparison. He found the overall incidence of heart disease as high or higher in blacks, with the majority of cases being hypertensive-

arteriosclerotic and syphilitic in origin. In all the reports summarized by Laws, syphilis was the second most common cause of heart disease. He also reported that hypertrophy was the most constant finding in the hypertensive and that substernal pain was not uncommon in blacks, but true angina pectoris was rare. Once again, hypertension occurred at younger ages in black patients. All of the above reports found a lower incidence of both angina pectoris and rheumatic heart disease in southern locations as compared with other areas of the country.

An article by Gager and Dunn²⁸ in 1933 presented findings from their study of 1,200 patients from private practice and clinics at George Washington University Hospital, Gallinger Municipal Hospital (now DC General Hospital), and Freedman's Hospital of Howard University. They found hypertension to be the condition most frequently associated with heart and vascular disease in both racial groups. Coronary arteriosclerosis and syphilis were equally prevalent in blacks and whites. The prevalence of angina pectoris was greater in white private practice patients; however, white and black clinic patients had a similar prevalence (5%) of angina pectoris.

O.F. Hadley of the US Public Health Service studied 450 cases of fatal heart disease in Washington, DC, in 1935.²⁹ This study reaffirmed that blacks died of heart disease at a younger age than did whites. A large proportion of young black male cardiac-related deaths were due to syphilis; death of both sexes was due to hypertension and arteriosclerosis. Hadley regarded coronary arteriosclerosis and thrombosis as uncommon in Negroes, but the study was flawed because a large number of sudden death victims from the coroner's office, which were not examined postmortem, were not included. Articles by Johnston³⁰ in 1936, and Weiss³¹ in 1939, attribute infrequent angina pectoris in blacks to low socioeconomic levels and low intellectual achievement. Whites of the same socioeconomic status were also noted to have a low incidence of angina pectoris, but the authors did not comment on their level of intellectual achievement.

The recognition of coronary artery disease as a significant health problem in black patients continued to gain momentum. Branch³² stated in 1941 "The Negro race is susceptible to every disease that the white race acquired when he attempts to enjoy civilization. The incidence may vary depending upon many complicated immunologic factors . . . Coronary artery disease is not a disease of the intelligent alone. It is seen among poor people also. Some authors have stated that the intellectual criteria of the Negro is low and he does not register.

This is categorically false. The poor uneducated white patient has coronary artery disease. Negro patients act and register identically as white patients where angina is seen."

Farmer,³³ writing on "The Incidence of Heart Disease Among Negroes" in 1940, found a very low prevalence of coronary occlusion or angina pectoris at Mercy Hospital in Philadelphia. His study examined only 28 hospitalized patients over a three-month period, and only four of these patients were male. His article stressed physical diagnosis as the best means of early detection as opposed to blind reliance on the increasingly popular electrocardiogram. His personal experience regarding the rarity of symptomatic coronary artery disease in blacks was refuted by Branch, a physician in the New York-Newark metropolitan area.

The papers of this period are remarkable for their similar conclusions, speculations, and attitudes concerning those of lower socioeconomic class. Although prevailing notions of race and a somewhat arrogant elitism directed at both blacks and whites biased the authors' interpretation of their data, these papers remain important and relevant contributions to medicine and provide insight to our current practice and mode of thought.

An autopsy study comparing black-white differences in coronary artery disease appeared in 1950.³⁴ It refers to a study from Hubbard Hospital (Meharry Medical College), which is referenced in the *Journal of the American Medical Association*, volume 389, page 202, 1946. The correct reference is the *Journal of the National Medical Association*. The article is by Thomas³⁵ and reports on "317 Cases of Heart Disease in Negroes." The preceding article is the "First Annual Oration in Medicine" to the National Medical Association convention, titled "Coronary Atherosclerosis in the Negro" by T.M. Smith of Provident Hospital in Chicago.

The 317 cases (all black) reported by Thomas reflects the overwhelming predominance of hypertensive heart disease (47%) and he differentiated those hypertensives with a large component of arteriosclerosis. Typical angina pectoris was rarely present (no figures given); instead, the predominant symptoms of coronary thrombosis and myocardial infarction were substernal "oppression" and dyspnea. An interesting feature of the hypertensive group was that the average age of those without arteriosclerosis was 50, and those with coronary artery disease was 60. This paper also reported a 25% overall incidence of syphilis in the patients studied.

Smith's oration stands out singularly for its scope.³⁶ He presented data from 155 patients with suspected coronary artery disease at Provident Hospital. Within the

group (there is no indication of the prevalence of the problem in the community at large) the patient profiles, socioeconomic status, incidence of proven myocardial infarction (24%), hypertension (52%), diabetes (9%), claudication (5%), obesity (13%) and chest pain (81%) are characteristic of a more modern series.³⁷ The article probed the etiology of coronary disease in its various forms, and sought to refute the prevalent myths regarding the absence of angina pectoris in blacks. Of particular note is the northern urban setting of the study, the decrease in positive serologies (11%), and a greater proportion of middle class and educated patients. Smith referenced an excellent autopsy study by Hunter³⁸ from the University of Louisville in which 1,000 consecutive black and 1,000 consecutive white patients were compared. Hunter agreed that a decreased index of suspicion is the prime factor for missing coronary ischemia pre-mortem in most black patients and stated that below the age of 70, coronary occlusion is equal in the races. Hunter further implied that dyspnea is a common angina pectoris equivalent in blacks. Smith had noted earlier³⁹ that classical angina pectoris was unlikely to be present in a patient already symptomatic with the dyspnea of compromised ventricular function and that racial predispositions were not "established." The tendency of individuals who are "tense, hard working, and given to overindulgence in eating . . ." were more usually the symptomatic victims of coronary occlusion. The authors discussed the role of hypertension as a cause for silent ischemia in black patients, a clear harbinger of our present day concerns.

The review of Fitzgerald and Yater⁴⁰ examined autopsy material from a single Washington, DC hospital (now the DC General Hospital) from 1940 to 1944. During this period blacks accounted for 64% of all admissions, but the number of final autopsy reports signed as myocardial infarctions was exactly 35 for each racial group. The clinical and social characteristics of the two groups were almost identical except that angina was more prevalent in the black patients studied. These 70 autopsies were taken from a total of 1,904, however there is no racial breakdown of this total pool. Since Fitzgerald and Yater had reviewed some of the earlier literature, they felt safe in concluding that "clinically significant coronary artery sclerosis is probably two and three times more common in white people than in Negroes. Myocardial infarctions are probably about twice as common in Caucasians." This conclusion does not seem to be supported by their own findings. They continued: "there do not appear to be important differences in either the pathologic features or the clinical manifestations

between the two races, although the general impression is that the disease is more often less obvious clinically in Negroes." The authors concluded by stating: "the cause for the difference in the racial incidence of this disease cannot be positively stated, but the differences in temperament and mode of living and the fact that more white people live longer may be factors . . . the trend was for the colored patients to die a decade earlier." Temperament is not mentioned earlier in their paper, and if lifestyle is related to occupation the groups were allegedly identical.

Yater was well respected for his teaching skills and his active interest in the scientific practice of medicine.⁴¹ In 1948 he published an exhaustive study on coronary artery disease in young men.⁴² His analysis, taken from US Army and Veterans Administration data, revealed the incidence of coronary artery disease in World War II Negro soldiers between 18 and 39 years of age to be "somewhat more than two thirds of that in the white soldiers." The autopsy findings in those who died of coronary disease showed no differences between races.

The growing magnitude of coronary artery disease as a national concern inspired the landmark study in Framingham, Massachusetts, which began in 1948. Although the original cohort of more than 5,000 patients included only six blacks,⁴³ the information gained has been applied broadly to presume the natural course of the disease and the identification of risk factors for the entire black population. Hypertension and left ventricular hypertrophy have a strong correlation with the development of coronary artery disease and its morbid events.⁴⁴ Therefore, the high prevalence of hypertension in US black populations would predict that coronary artery disease should not be uncommon. The racial approach to studying the disease continued, however, but with increasing evidence that apparent racial differences were minor in terms of pathophysiology. The comparative study of myocardial infarction patients by Keil and McVay⁴⁵ in 1956 reported an increased frequency of the problem in black females as compared with white females, but a decreased incidence of angina pectoris in black women. The incidence of diabetes in all of their female patients may have played a role in the clinical manifestations. The incidence of hypertension in all four sex-race groups were similar and implies that by working backward from an end-point of established myocardial ischemia, patients with similar risk profiles are selected presumed racially influenced factors are negated. A similar study by Thomas et al in 1961,⁴⁶ reflected no significant racial differences in mode of presentation for black and white myocardial infarction patients and again

confirmed the increased morbidity in black females, which they speculated was related to hypertension and diabetes. The decreased early mortality in blacks compared with whites was also ascribed to hypertension and its possible role in augmenting coronary perfusion.

The 1958 report by Mihaly and Whiteman⁴⁷ (from Harlem Hospital) represented a more recent attempt to clarify the misunderstanding concerning coronary artery disease in black Americans. In analyzing myocardial infarctions in black patients, they found no difference "from the general population as regards the incidence of myocardial infarction in the presence of predisposing factors, nor are the mortality figures different in the race." They found that "87.7% of the patients in this series had precordial pain as a cardinal symptom."

The commentaries of White also revealed a gradual recognition of the importance of coronary artery disease across racial and ethnic lines. The 1931, 1937, and 1947⁴⁸ editions of his textbook on heart disease claim a minimal influence of race in the etiology of coronary disease, but in 1956⁴⁹ he cited the papers of Hunter³⁸ and Smith³⁶ as evidence that the black population may have a higher incidence of coronary disease than previously realized. There is still a distinction between coronary artery disease and angina pectoris since in the earlier editions he stated that, "the pure-blooded Negro rarely or never has [angina pectoris]."

The epidemiologic analysis of coronary artery disease in both a national and international arena has repeatedly indicated the importance of diet-associated disease and other environmental factors as risk determinants for coronary artery disease, far more than any purely racial considerations.⁵⁰ In the era preceding this apparent consensus, and the widespread application of coronary angiography and myocardial revascularization, the sound epidemiologic approach advanced by Stamler^{51,52} had refreshing objectivity, clarity, and useful information concerning the development and prevalence of coronary artery disease. These writings are remarkable for having addressed and answered some of the questions that still nag portions of the medical community.

In reviewing the publications of John B. Johnson, the former department chairman and chief of cardiology at Howard University,⁵³ two papers were found dealing with coronary artery disease and myocardial infarction specifically.^{54,55} In neither did Johnson elaborate on any racial differences; the proper methods of diagnosis and state of the art therapy were all that mattered for "hearts too good to die . . ." Most of his research interest were

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Brief Summary

MINIPRESS (prazosin hydrochloride) CAPSULES

For Oral Use

INDICATIONS AND USAGE: MINIPRESS (prazosin hydrochloride) is indicated in the treatment of hypertension. It is mild to moderate in activity and can be used as the initial agent or in a general treatment program in conjunction with a diuretic and/or other antihypertensive drugs as needed. **CONTRAINDICATIONS:** None known. **WARNINGS:** MINIPRESS may cause syncope with sudden loss of consciousness. In most cases this is believed to be due to an excessive postural hypotensive effect, although occasionally the syncope has been precipitated by a bout of severe tachycardia with heart rates of 120-160 beats per minute. Syncope episodes have usually occurred within 30 to 90 minutes of the initial dose of the drug; occasionally they have been reported in association with rapid dosage increases or the introduction of another antihypertensive drug into the regimen of a patient taking high doses of MINIPRESS. The incidence of syncope episodes is approximately 1% in patients given an initial dose of 2 mg or greater. Clinical trials conducted during the investigational phase of this drug suggest that syncope episodes can be minimized by limiting the initial dose of the drug to 1 mg, by subsequently increasing the dosage slowly, and by introducing any additional antihypertensive drugs into the patient's regimen with caution (see **DOSE AND ADMINISTRATION**). Hypotension may develop in patients given MINIPRESS who are also receiving a beta-blocker such as propranolol. If syncope occurs, the patient should be placed in the recumbent position and treated supportively as necessary. This adverse effect is self-limiting and in most cases does not recur after the initial period of therapy or during subsequent dose titration. The patient should also be cautioned to avoid situations where injury could result should syncope occur during the initiation of MINIPRESS therapy. **PRECAUTIONS: Information for Patients:** Dizziness or drowsiness may occur after the first dose of this medicine. Avoid driving or performing hazardous tasks for the first 24 hours after taking this medicine or when the dose is increased. Dizziness, lightheadedness or fainting may occur, especially when rising from a lying or sitting position. Getting up slowly may help lessen the problem. These effects may also occur if you drink alcohol, stand for long periods of time, exercise, or if the weather is hot. While taking MINIPRESS, be careful in the amount of alcohol you drink. Also, use extra care during exercise or hot weather, or if standing for long periods. Check with your physician if you have any questions. **Drug Interactions:** MINIPRESS has been administered without any adverse drug interaction in limited clinical experience to date with the following: (1) cardiac glycosides—digitalis and digoxin; (2) hypoglycemics—insulin, chlorpropamide, phenformin, tolazamide, and tolbutamide; (3) tranquilizers and sedatives—chloriazepoxide, diazepam, and phenobarbital; (4) antitubercular—isoniazid, rifampin, and probenecid; (5) antiarrhythmics—procainamide, propranolol (see **WARNINGS** however), and quinidine; and (6) analgesics, antipyretics and anti-inflammatories—propoxyphene, aspirin, indomethacin, and phenylbutazone. Addition of a diuretic or other antihypertensive agent to MINIPRESS has been shown to cause an additive hypotensive effect. **Drug/Laboratory Test Interactions:** False positive results may occur in screening tests for pheochromocytoma in patients who are being treated with prazosin. If an elevated VMA is found, prazosin should be discontinued and the patient retested after a month. **Laboratory Tests:** In clinical studies in which lipid profiles were followed, there were generally no adverse changes noted between pre- and post-treatment lipid levels. **Carcinogenesis, Mutagenesis, Impairment of Fertility:** No carcinogenic potential was demonstrated in a 18 month study in rats with MINIPRESS (prazosin hydrochloride) at dose levels more than 225 times the usual maximum recommended human dose of 20 mg per day. MINIPRESS was not mutagenic in *in vivo* genetic toxicology studies. In a fertility and general reproductive performance study in rats, both males and females, treated with 75 mg/kg (225 times the usual maximum recommended human dose), demonstrated decreased fertility while those treated with 25 mg/kg (75 times the usual maximum recommended human dose) did not. In chronic studies (one year or more) of MINIPRESS in rats and dogs, testicular changes consisting of atrophy and necrosis occurred at 25 mg/kg/day (75 times the usual maximum recommended human dose). No testicular changes were seen in rats or dogs at 10 mg/kg/day (30 times the usual maximum recommended human dose). In view of the testicular changes observed in animals, 105 patients on long term MINIPRESS therapy were monitored for 17 α -ketosteroid excretion and no changes indicating a drug effect were observed. In addition, 27 males on MINIPRESS for up to 51 months did not have changes in sperm morphology suggestive of drug effect. **Use in Pregnancy:** Pregnancy Category C. There are no adequate and well controlled studies which establish the safety of MINIPRESS (prazosin HCl) in pregnant women. MINIPRESS should be used during pregnancy only if the potential benefit justifies the potential risk to the mother and fetus. **Nursing Mothers:** MINIPRESS has been shown to be excreted in small amounts in human milk. Caution should be exercised when MINIPRESS is administered to a nursing woman. **Use in Children:** Safety and effectiveness in children have not been established. **ADVERSE REACTIONS:** Clinical trials were conducted on more than 900 patients. During these trials and subsequent marketing experience, the most frequent reactions associated with MINIPRESS therapy are: dizziness 10.3%, headache 7.8%, drowsiness 7.6%, lack of energy 6.9%, weakness 6.5%, palpitations 5.3%, and nausea 4.9%. In most instances side effects have disappeared with continued therapy or have been tolerated with no decrease in dose of drug. Less frequent adverse reactions which are reported to occur in 1-4% of patients are: **Gastrointestinal:** vomiting, diarrhea, constipation; **Cardiovascular:** edema, orthostatic hypotension, dyspnea, syncope; **Central Nervous System:** vertigo, depression, nervousness; **Dermatologic:** rash; **Genitourinary:** urinary frequency; **EENT:** blurred vision, reddened sclera, epistaxis, dry mouth, nasal congestion. In addition, fewer than 1% of patients have reported the following (in some instances, exact causal relationships have not been established): **Gastrointestinal:** abdominal discomfort and/or pain, liver function abnormalities, pancreatitis; **Cardiovascular:** tachycardia; **Central Nervous System:** paresthesia, hallucinations; **Dermatologic:** pruritus, alopecia, lichen planus; **Genitourinary:** incontinence, impotence, priapism; **EENT:** tinnitus; **Other:** diaphoresis, fever. Single reports of pigmentary mottling and serous retinopathy, and a few reports of cataract development or disappearance have been reported. **OVERDOSAGE:** Should overdose lead to hypotension, support of the cardiovascular system is of first importance. Restoration of blood pressure and normalization of heart rate may be accomplished by keeping the patient in the supine position. If this measure is inadequate, shock should first be treated with volume expanders. If necessary, vasopressors should then be used. Renal function should be monitored and supported as needed. Laboratory data indicate MINIPRESS is not dialysable because it is protein bound. **DOSE AND ADMINISTRATION:** The dose of MINIPRESS should be adjusted according to individual blood pressure response. **Initial Dose:** 1 mg two or three times a day. **Maintenance Dose:** Dosage may be slowly increased to a total daily dose of 20 mg given in divided doses. The therapeutic dosages most commonly employed have ranged from 6 mg to 15 mg daily given in divided doses. Doses higher than 20 mg usually do not increase efficacy; however a few patients may benefit from further increases up to a daily dose of 40 mg given in divided doses. After initial titration some patients can be maintained adequately on a twice daily dosage regimen. **Use With Other Drugs:** When adding a diuretic or other antihypertensive agent, the dose of MINIPRESS should be reduced to 1 mg or 2 mg three times a day and retitration then carried out.

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in other areas of cardiovascular hemodynamics and hypertension.

The retrospective or prospective study of any variable that has the potential of providing pertinent information on the pathogenesis of coronary artery disease is obviously important. Race, like sex, is a nonalterable variable, but it could harbor information about coronary artery disease. This article has presented some of the documented opinions concerning coronary artery disease in blacks from a constricted but formative period (1920-1960) in our thinking. But whether or not race is an important factor in the pathogenesis of coronary artery disease still remains unclear. It is our opinion that race is irrelevant if the coronary disease risk factor milieu of blacks and whites is equalized.

This opinion is based partly on the results of a 25-year prospective study by Thomas et al⁵⁶ of 433 black male physicians (former students at Meharry Medical College) and 551 white male physicians (former students at Johns Hopkins University School of Medicine). Follow-up of 80% of both cohorts have shown that 132 of 313 (52%) black physicians developed hypertension versus 73 of 526 (13.8%) of the white physicians; and that 13 (4%) of the black physicians versus 2 (0.3%) of the white physicians developed myocardial infarctions. This observation of increased myocardial infarctions in black physicians is inconsistent with many opinions in the literature. If it can be assumed that the lifestyle and socioeconomic status of these groups have been similar, then the nonequalizing factor could be hypertension. It is probable that the wide variability in the incidence and prevalence of coronary artery disease seen in various subgroups of black Americans is not as closely related to race as it is to longevity and exposure to the risk factors of Western societies. Since 1960, we have learned much more about the numerous manifestations of myocardial ischemia and have recognized that neither classic angina pectoris, disease of the epicardial coronary arteries, racial identity, or middle class status need be present to arouse clinical suspicion.

For the patient suffering from angina pectoris, angina equivalents, silent ischemia, or myocardial infarction with its various sequelae, whether other members of his race or socioeconomic class also suffer is of no immediate importance. Once a patient is afflicted with the disease, the outcome is dictated by his ability to seek treatment and be attended by capable physicians. In this regard, cardiologists and cardiovascular surgeons have created an expanding sphere of cooperation and collaboration. Because the surgical treatment, like the medical

treatment of coronary artery disease, is palliative, these disciplines have developed a unity of purpose and techniques (both invasive and noninvasive) that evolve around patient care in continuity.

Based on this review, there is little evidence to conclude that physicians' attitudes and behavior have somehow escaped the influence of social trends and circumstances. It is obvious that this could have affected clinical decision making. It is hoped that the medical literature after 1960 will reflect an enlightened application of our knowledge base in ischemic heart disease. Unchallenged bias in medical thinking, regardless of its innocence, inhibits and restricts the potential benefits of our technology for the individual patient and his community.

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