UNDERTREATMENT OF CARDIOVASCULAR DISEASE IN ETHNICALLY DIVERSE OLDER ADULTS: WHO SHOULD RECEIVE AN ELECTROCARDIOGRAM?

To the Editor: There is an increased prevalence of cardiovascular disease and its associated risk factors such as hypertension in older African Americans and probably other ethnic groups as well.[1,2] Several factors likely contribute to this increased prevalence, including genetics, environment, culture, education, socioeconomic status, and access to health care. However, the explanation remains incomplete.[3–7] While screening ethnically diverse seniors for an exercise study, we inadvertently discovered that many subjects and their doctors were not aware of a large number of cardiovascular problems. Perhaps this finding can help to shed light on the high rate of cardiovascular morbidity and mortality in older minority populations.[8]

More specifically, we studied 36 older adults (mean age ± standard deviation: 71.1 ± 6.6 years; 21 women) who received written permission from their primary care physician (PCP) to participate in an "aggressive" exercise program at a multicultural senior center in Dorchester, Massachusetts. Center participants typically have a lower socioeconomic status and are predominately of non-European ancestry. Thirty of the 36 the subjects we studied did not speak English and 28 could not read or write. Thirty-three were not European American. Interpreters were used to communicate with the participants.

The PCP of each eligible subject was asked, in writing, whether their patient was an appropriate candidate for a vigorous exercise program and to report on their patient's medical history and whether they had any cardiac concerns. We acquired a medical history, performed an electrocardiogram (ECG), and measured blood pressure. A cardiologist who had no knowledge of the PCP's report reviewed the ECG. ECG abnormalities noted included ischemia, atrial fibrillation, left ventricular hypertrophy, and atrioventricular-block.

Of the 36 subjects, 24 had an ECG abnormality. In the 24 subjects with ECG abnormalities, the primary care physician or the patients themselves reported cardiac problems in four instances (16%). Twenty-eight subjects had hypertension, but only 23 had received a prescription for antihypertensive medication. Of the 23 subjects on antihypertensive drugs, 13 still did not have controlled hypertension. Hypertension and ECG abnormalities are summarized in Table 1 and Figure 1. By definition, all subjects had a PCP.

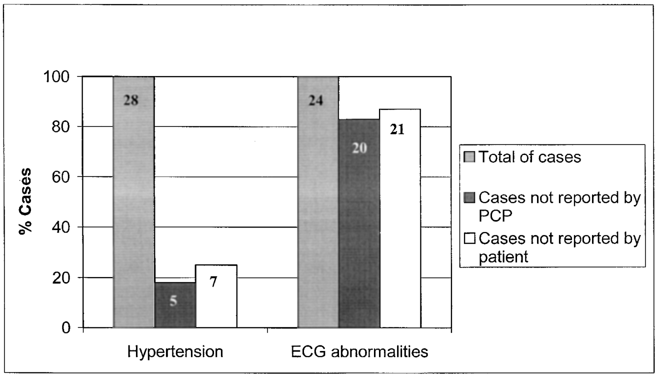

Figure 1. Undetected and unreported cardiovascular abnormalities. The subjects did not report more than 80% of the cases of electrocardiogram (ECG) abnormalities. This appears to have been largely a result of the physicians' failure to test for the presence of ECG changes. Note that all subjects received written clearance by their PCP to participate in an aggressive exercise program.

The subjects or their PCP did not report more than 80% of the cases of ECG abnormalities. This appears to have been largely a result of the physician's failure to test for (or at least report) the presence of ECG changes. In contrast to the recommendations of the PCPs, based on our measures, the study physician permitted only 19 of the 36 subjects to participate in the exercise study.

The benefits of an ECG are well known, yet our findings raise a potentially important question: why does it appear that the ECG is underutilized in older minorities? There are no firm guidelines recommending when older adults should have an ECG performed even though the yield of irregular ECGs is very high, the cost is relatively low, and, often, proven therapeutic interventions are at hand. Among this group, high-cost cardiac interventions are also underutilized.[4,5] This situation contrasts dramatically with the recommendation for other age-associated diseases. For example, for osteoporosis screening, bone densitometry scans are recommended for all persons age 65 and older. Although very important to the health of older people, these tests are relatively costly, and, when an abnormality is detected, the treatment is expensive and requires long-term therapy to achieve only modest gains. Perhaps similar guidelines for ECG evaluation in older adults, especially those who are not fluent in English, would help to reduce the relatively high

Table 1. Undertreated and Undetected Cardiovascular Abnormalities

Abnormality	Cases n (%)	Not Reported by Doctor n (%)	Not Reported by Patient n (%)
Hypertension	28 (78)	5 (18)	7 (25)
Dyspnea or dyspnea on exertion	8 (22)	6 (75)	7 (87)
Arrhythmia	3 (8)	2 (66)	3 (100)
CAD/MI	13 (36)	9 (69)	9 (69)
Other ECG abnormality	8 (22)	8 (100)	8 (100)
Total ECG abnormality detected	24 (61)	20 (83)	21 (87)
Total cardiovascular abnormalities detected	60	31 (52)	35 (58)

CAD = coronary artery disease; MI = myocardial infarction; ECG = electrocardiogram.

degree of cardiovascular morbidity and mortality among non–European Americans.

These preliminary findings highlight the problem of the underdiagnosis and undertreatment of hypertension and ECG abnormalities in older minorities. Future studies are needed to further assess the role of education, acculturation, and socioeconomic status as they affect the care of older minorities. Further examination of the efficacy, utility, and cost-effectiveness of more widespread testing of the ECG is needed in older adults, especially among ethnically diverse seniors, where the barriers of language and culture may also exacerbate the difficulty of obtaining accurate self-reporting. It would appear that the ECG is an invaluable but underutilized tool for assessing cardiovascular risk among older minorities, many of whom may not speak fluent English and may be illiterate. To help reduce cardiac morbidity and mortality, perhaps it would be appropriate to enact a national guideline that calls for yearly ECG screening.

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BLOOD PRESSURE REDUCTION AFTER THE FIRST DOSE OF CAPTOPRIL AND PERINDOPRIL

To the Editor: Initiation of treatment with angiotensin-converting enzyme (ACE) inhibitors is frequently complicated by the occurrence of (sometimes severe) hypotension after the first dose. Profound hypotension, requiring termination of the treatment, was present in about 5% of the patients after the first dose of 6.25 mg captopril and of 10 mg enalapril.[1,2] First-dose hypotension (FDH) increases the risk of falls, myocardial infarction, and stroke. A positive correlation between FDH and mortality was found.[3] Older patients are especially at risk, presumably because of impaired adaptive and autoregulation functions. To prevent FDH, it is advised to stop diuretics for 24 to 48 hours and to give a low dose of 6.25 mg captopril or 2.5 mg enalapril. However, in older people, a delicate treatment equilibrium often exists, and discontinuation of diuretics is not always possible. In general practice, the advice to stop diuretics is not always followed up.[4] Clinically relevant differences between ACE inhibitors in the magnitude of FDH might exist. Studies showed that perindopril caused less FDH than did captopril, enalapril, or lisinopril.[5–7]

We studied 10 patients age 70 and older with chronic stable heart failure and proven severe FDH, defined as a decrease of mean arterial blood pressure (MAP) >25 mmHg, after 6.25 mg captopril. A consent was obtained from each patient.

Blood pressure was measured supine with an automatic ambulatory device (Spacelab®, SpaceLabs, Inc., Workingham, Beckshire, England) every 15 minutes from 8 a.m. on for 5 hours and in sitting position every hour for the next 8 hours. During the whole study period, patients were closely monitored for clinical signs of FDH. Blood pressure was measured on 3 different days: at baseline; the following day, receiving 6.25 mg captopril at 9 a.m.; and, in the case of severe FDH, receiving 2 mg perindopril at 9 a.m. after a 3-day washout period. All other drugs, including diuretics, were not changed from 72 hours before the study until the end of the study period. The difference between the lowest MAP measured in the hour before captopril or perindopril and the lowest of 24 MAPs measured within 12 hours after captopril or perindopril was calculated. The differences in blood pressure reduction after captopril and perindopril were tested with Wilcoxon signed rank test. Effects with a P-value $<$ .05 were regarded as significant.

Of 25 patients, 10 (mean ± standard deviation (SD) age 84 ± 6 years) suffered a blood pressure reduction of >25 mmHg MAP after 6.25 mg captopril. No differences were found for age, gender, serum creatinine level, or New York Heart Association class between patients with and without severe FDH. The 10 patients with severe FDH used at mean a lower dose of furosemide (44 ± 13 mg) than did the 15 patients without severe FDH (77 ± 53 mg). The mean ± SD maximum fall of MAP in these 10 patients was 31.3 ± 4.5 mmHg (range 27–40). Two patients became drowsy for a short period. No intervention was needed. After the first dose of perindopril, mean ± SD maximum fall of MAP was 18.5 ± 10.6 mmHg (range 5–38) in the 10 patients. Two patients had a fall of >25mmHg MAP. None of the patients had symptomatic FDH. The difference in blood pressure reduction after the first dose of captopril and perindopril was statistically significant (P = .007).

Figure 1 shows the course of the MAP at baseline, after captopril, and after perindopril. On all 3 days, a marked and comparable blood pressure reduction was present between 8:00 a.m. and 9:00 a.m. A further blood pressure reduction was found 1 hour after use of captopril, between 10:00 and 11:00 a.m. After 12:00 noon, the blood pressure–lowering effect of captopril disappeared again. Captopril was not continued after the first dose. The blood pressure reduction caused by perindopril started gradually, 4 to 5 hours after the dose, and persisted until the last measurements. The