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

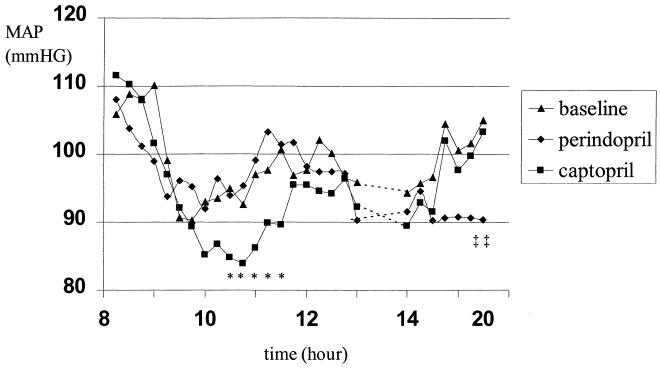

Figure 1. Course of blood pressure at baseline, after 6.25 mg captopril and 2 mg perindopril. MAP = mean arterial blood pressure.
*$P < .05$ captopril compared with perindopril and baseline; †$P < .05$ perindopril compared with captopril and baseline.

median time to the lowest MAP after captopril was 2 hours (range 0.75–5 hours) and after perindopril was 5 hours (range 0.25–10 hours). Plasma ACE values before the first dose of captopril or perindopril were both 0.62 ± 0.2 U/l.

The present findings confirm that 2 mg perindopril causes less blood pressure reduction after the first dose than does captopril. Even in a high-risk group of older patients with heart failure with proven FDH (>25 Hg MAP) after 6.25 captopril, blood pressure reduction after perindopril was gradual and mild and caused no symptoms, even when diuretics were not withdrawn.

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RISK FACTORS FOR DEVELOPING CARDIAC DISEASE IN LATE MIDDLE-AGED AND OLDER MEN AND WOMEN: A PROSPECTIVE STUDY

To the Editor: Previous studies have identified several risk factors for developing cardiac disease (CD), particularly in younger samples. Hypertension and coronary heart disease were found to be predictive for the development of congestive heart failure (CHF).[1,2] However, neither Seeman et al.[3] nor Penninx et al.[4] found, in older persons, any significant association between blood pressure and cardiac events. Diabetes mellitus also emerged as an important risk factor for CD, with greater risk for older women than for older men.[2,3] Other independent risk factors for the development of CHF were (past) smoking behavior, obesity, and (psychological) stress.[5] Depressive symptomatology was found to be related to CD, particularly myocardial infarction and ischemic heart disease.[4,6–10] However, Mendes de Leon et al. reported a significant association between depressive symptoms and myocardial infarction only in older women,[9] whereas Penninx et al. found that depressed mood was related to cardiovascular events only in older men.[10]

Although most of these studies suggested that subjects with a history of hypertension, diabetes mellitus, obesity, and smoking are at risk for developing CD, these main, well-known risk factors have mostly been studied in samples of middle-aged men.[3] Studies on risk factors for older men and women produced inconsistent findings regarding the risks associated with smoking, high blood pressure, depressive symptoms, and obesity.[3] In addition, several of the studies mentioned above did not differentiate between men and women, did not use large population-based samples, and focused on only a few risk factors, and several studies also used samples including only younger or younger and older subjects. Furthermore, most research included only cardiac mortality or hospitalization as endpoint of observation. Cardiac events may then have induced risk factors for mortality or hospitalization (such as depression and nonadherence with medical regimen).[11]

In the present prospective study of older people, we included hypertension, heart disease, diabetes mellitus, depressive symptomatology, obesity, and past smoking behavior as potential risk factors for developing subsequent CD in a large sample of late middle-aged and older persons. Endpoint of measurement included CD irrespective of mortality or hospitalization The analyses were conducted separately for men and women.

The investigation was part of the Groningen Longitudinal Aging Study.[12] In 1993, 5,279 persons age 57 and older from the patient panels of 27 family physicians who register every doctor-patient encounter in a computerized health information system were interviewed. The interviews collected data on six risk factors: body mass index, life-time smoking exposure, depressive symptoms, heart disease, hypertension, and diabetes mellitus, plus age, level of education, and number of chronic medical conditions. Patients who had a new postbaseline cardiac event were recruited through the 27 family physicians. Two cardiac events were included: CHF and acute myocardial infarction (AMI). Each fortnight, from the baseline wave in 1993 until January 1, 1998, these physicians passed on the