Appropriate Drug Prescribing for Cardiovascular Disease in the Elderly

Alexandra Papaioannou, MD, George Heckman, MD, and Jo-Anne Clarke

Cardiovascular disease is one of the leading causes of morbidity and mortality in persons older than 65 years. Hospitalizations due to heart failure have doubled over the past 20 years. Two thirds of myocardial infarcts occur in patients older than 65 [1]. Many articles have been published on appropriate use of drug therapy in the elderly, and the underuse of potentially beneficial therapy has been targeted as an area for improvement. However, evidence-based practice is a challenge when there are inadequate data or exclusion of the elderly from research trials [2]. Gurwitz et al [3] found that 60% of individuals aged 75 and older were excluded from therapeutic trials for acute myocardial infarction [3]. There is also a growing interest in employing cost-effective therapies, particularly with a rapidly aging population.

We conducted a systematic literature search to examine pharmaceutical use and appropriate pharmacomnagement of common cardiac conditions in the elderly. Studies were identified by searching the MEDLINE database and Cochrane Registry of Controlled Trials for articles published through November 1999. Search terms included adrenergic-β antagonists and aged, congestive heart failure and aged, hypertension and aged, hypercholesterolemia and statins and fibrates and aged. Only trials looking at morbidity and mortality were selected. References were also handsearched for additional relevant articles.

β Blockade After Myocardial Infarction

β Blockers have been studied in over 50 randomized controlled trials including 55,000 patients. Yusof et al, in their meta-analyses [4,5], examined outcomes of therapy initiated 1 to 2 weeks post–myocardial infarction; follow-up ranged between 1 and 2 years. Pooling of the data reveals that β blockade after myocardial infarction is associated with a 20% reduction in total mortality and 34% reduction in risk of sudden cardiac death. Cohort studies support a similar risk reduction in the elderly (Table 1) [6–8]. Guidelines form the American College of Cardiology/American Heart Association (ACC/AHA) state that the use of β blockers after myocardial infarction decreases mortality and reinfarction and increases the probability of long-term survival by up to 40% [9].

Soumerai et al [7] found in a retrospective cohort study linking Medicare and drug claims that only 21% of elderly patients over the age of 75 were prescribed β blockers. A recent study demonstrated 51% of elderly patients hospitalized with an acute myocardial infarct did not receive early β blockers despite no contraindications to therapy [10].

Clinicians have avoided β blockers owing to concerns about possible contraindications in groups excluded from early randomized trials, including the very elderly. More recent observational data clearly show the benefit of β blockade post–myocardial infarction even in patients over 85 (Table 1). The 1999 ACC/AHA guidelines state that the benefits of β blockers may extend to patients with asthma, insulin-dependent diabetes mellitus, chronic obstructive lung disease, severe peripheral vascular disease, PR interval greater than 0.24 seconds, and moderate left ventricular failure [9]. These recommendations should allow for more elderly patients with comorbidity to receive potentially life-saving treatment, given careful monitoring for adverse effects. Approximately 75% of patients who present with acute myocardial infarction are older than 70 years [11].

Incorporating evidence-based recommendations into clinical practice can be facilitated by medical opinion leaders. In a randomized controlled trial of 37 community hospitals in Minnesota, local opinion leaders increased the appropriate use of aspirin and β blockers for patients hospitalized with acute myocardial infarction. The intervention, which included a guideline-based presentation, increased the median proportion of eligible patients receiving β blockers from 49% to 80% [12].

ACE Inhibitors for Treatment of Heart Failure

Angiotensin-converting enzyme (ACE) inhibitors are recommended for the treatment of heart failure in all patients with left ventricular systolic dysfunction who can tolerate treatment.

Alexandra Papaioannou, MD, Associate Professor, Division of Geriatric Medicine, Department of Medicine, McMaster University, Hamilton, Ontario; George Heckman, MD, Fellow, Division of Geriatric Medicine, Department of Medicine, McMaster University; and Jo-Anne Clarke, Student, Department of Medicine, McMaster University.
with this medication [13]. A meta-analysis of 32 trials found ACE inhibitors decreased the relative risk of mortality and hospitalization for congestive heart failure by 31.2% [14]. A subgroup analysis of the SAVE trial, in which captopril was evaluated in patients with post-myocardial infarction left ventricular dysfunction, demonstrated that physicians needed to treat only 13 patients older than 64 years, compared to 112 patients younger than 55 years, to prevent 1 death. To prevent 1 cardiovascular event, the numbers needed to treat were 12 (older patients) and 39 (younger patients) [15].

Despite evidence to support the use of ACE inhibitors in older adults, utilization rates vary from 25% to 57% [14,16–18]. Advancing age has been linked to underutilization, with an odds ratio of 0.56 that patients over 75 are less likely to receive ACE inhibitors [19].

Losartan, an angiotensin-II receptor blocker, is an effective alternative to ACE inhibitors for patients who are intolerant of ACE inhibitors [20]. β Blockers are indicated in patients with New York Heart Association class II and III symptoms who are on stable ACE inhibitor treatment [21,22]. Spironolactone produced a relative risk reduction of 30% in cardiovascular hospitalization of patients with severe heart failure [23]. Close monitoring of potassium levels and renal function when prescribing ACE inhibitors and spironolactone for heart failure is highly recommended due to the increased prevalence of renal insufficiency in the elderly.

Hospital readmissions account for a large proportion of costs associated with heart failure. A New Zealand study found that 40% of admissions for heart failure were due to readmission during the same year [24]. Nonadherence to prescribed therapy may be an important contributor. A New Jersey Medicaid study found that less than 10% of individuals filled enough prescriptions to have daily heart failure medications for the entire year [25]. Studies have shown that a multidisciplinary approach to therapy that includes interventions such as patient education and telephone follow-up can reduce readmission rates and hospital bed days and improve adherence to therapy and quality of life [26–29].

### Lipid-Lowering Therapy

Over the past 10 years, large well-designed trials have addressed lipid-lowering therapy with hydroxyethylmethylglutaryl-CoA reductase inhibitors (statins) and fibric acid derivatives (fibrates) for the prevention of coronary artery disease (Tables 2 and 3) [30–46]. Four placebo-controlled trials studied statins for the primary prevention of coronary events for patients aged 20 to 86 years [30–33]. Subjects with a broad range of serum cholesterol levels and no prior coronary disease received statins for 1 to 5 years and had fewer cardiovascular events compared with placebo controls. Individuals with additional cardiovascular risk factors seemed to benefit the most. Six placebo-controlled secondary prevention trials assessed statins and fibrates for patients aged 21 to 75 years. In 5 of these trials, there were fewer cardiovascular events in those with established coronary artery disease treated over 5.5 years [34–37]. A meta-analysis of the large statin trials

#### Table 1. Cohort Studies Examining the Benefit of β Blockade on Mortality in the Elderly

<table>
<thead>
<tr>
<th>Study</th>
<th>Design</th>
<th>Patients</th>
<th>Subgroups</th>
<th>RR (95% CI) for Mortality in Patients Prescribed a β Blocker</th>
<th>% Prescribed a β Blocker</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gottlieb</td>
<td>Retrospective cohort from Cooperative Cardiovascular Project database</td>
<td>201,752 patients ≥ 65 yr discharged from hospital with a principle diagnosis of acute MI during an 8-month period</td>
<td>“Ideal patients”*&lt;sup&gt;+&lt;/sup&gt;</td>
<td>0.60 (0.57–0.63)</td>
<td>65.7</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>&lt; 70 yr</td>
<td>0.60 (0.57–0.63)</td>
<td>36.9</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>70–79 yr</td>
<td>0.64 (0.58–0.70)</td>
<td>32.9</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>≥ 80 yr</td>
<td>0.68 (0.63–0.75)</td>
<td>26.7</td>
</tr>
<tr>
<td>Soumerai</td>
<td>Retrospective cohort from a drug claims database</td>
<td>3737 patients ≥ 65 yr discharged from hospital between 1986 and 1990 with a principle diagnosis of acute MI who were eligible to receive β blockers</td>
<td>Overall</td>
<td>0.57 (0.47–0.69)</td>
<td>21</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>65–74 yr</td>
<td>0.50 (0.36–0.72)</td>
<td>NA</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>75–84 yr</td>
<td>0.56 (0.43–0.73)</td>
<td>NA</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>≥ 85 yr</td>
<td>0.72 (0.47–1.11)</td>
<td>NA</td>
</tr>
<tr>
<td>Krumholz</td>
<td>Retrospective cohort from Cooperative Cardiovascular Project database</td>
<td>45,308 “ideal candidates”† (out of 115,015) ≥ 65 yr who survived hospitalization with MI</td>
<td>Overall</td>
<td>0.86 (0.80–0.91)</td>
<td>50.0</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>65–74 yr</td>
<td>0.81 (0.72–0.91)</td>
<td>53.2</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>75–84 yr</td>
<td>0.88 (0.81–0.98)</td>
<td>48.7</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>&gt; 84 yr</td>
<td>0.88 (0.78–0.99)</td>
<td>40.3</td>
</tr>
</tbody>
</table>

<sup>*Patients without contraindications to β blockers.</sup>

<sup><sup>†Patients with MI and no other complications.</sup></sup>

CI = confidence interval; MI = myocardial infarction; NA = not available; RR = relative risk.

48 JCOM April 2000 Vol. 7, No. 4
showed that treatment significantly reduced overall mortality by 21% and major coronary events by 31% [46]. Statins were effective over a broad range of cholesterol levels, though the benefit may be minimal for LDL levels below 125 mg/dL [34,36,37]. Gemfibrozil prevented events in subjects with low HDL in the VA-HIT trial [40]. Posthoc analysis of the BIP trial suggests that individuals with high triglycerides might also benefit from fibrates [39]. Lipid-lowering therapy also reduced the incidence of stroke, a finding supported by meta-analysis [46]. Economic analysis suggests that lipid-lowering therapy is cost-effective for primary and secondary prevention of cardiovascular events in older individuals [47–53]. However, there is insufficient evidence from trials to address appropriateness of lipid-lowering agents in adults over 75. Studies including 80-year-old subjects are underway but will not be completed for several years [54–56]. There is no evidence to suggest pathologic features of atherosclerosis unique to aging. Therefore, the decision to offer lipid-lowering to older adults hinges on whether dyslipidemia is a risk factor in this population [57].

### Table 2. Randomized Trials of Lipid-Lowering Therapy That Included Patients Older Than 65 Years

<table>
<thead>
<tr>
<th>Study</th>
<th>Patients</th>
<th>Patient Age, yr</th>
<th>Lipid Levels, mg/dL</th>
<th>Intervention</th>
<th>Years of Follow-up</th>
<th>Outcomes</th>
<th>% Risk Reduction</th>
</tr>
</thead>
<tbody>
<tr>
<td>PSMG [30]</td>
<td>1062 at high-risk for CAD</td>
<td>20–86</td>
<td>TC: 5.2–7.8</td>
<td>Pravastatin</td>
<td>1</td>
<td>Efficacy/safety/CV event*</td>
<td>N/A 8 (P &lt; 0.001)</td>
</tr>
<tr>
<td>WOSCOPS [31]</td>
<td>6595 with no prior MI</td>
<td>45–64</td>
<td>LDL: 4.0–6.0</td>
<td>Pravastatin</td>
<td>4.9</td>
<td>CAD death/MI/Death</td>
<td>69 (P &lt; 0.001)/78 (P = 0.051)</td>
</tr>
<tr>
<td>AFCAPS/TeXCAPS [32]</td>
<td>6605 with no CAD</td>
<td>45–73</td>
<td>TC: 4.6–6.82 LDL: 3.36–4.91 HDL: ≤1.16 TG: ≤4.52</td>
<td>Lovastatin</td>
<td>5.2</td>
<td>CAD event/CV event</td>
<td>63 (P &lt; 0.001)/75 (P = 0.003)</td>
</tr>
<tr>
<td>ACAPS [33]</td>
<td>919 with carotid plaques</td>
<td>40–79</td>
<td>LDL: 3.36–4.89 TG: ≤4.52</td>
<td>Lovastatin</td>
<td>3</td>
<td>Plaque change</td>
<td>N/A 36 (P = 0.04)</td>
</tr>
<tr>
<td>4S [34]</td>
<td>4444 with angina or prior MI</td>
<td>35–70</td>
<td>TC: ≥5.5 TG: ≤2.5</td>
<td>Simvastatin</td>
<td>5.4</td>
<td>Mortality/CAD event</td>
<td>70 (P = 0.0003)/66 (P &lt; 0.0001)</td>
</tr>
<tr>
<td>LCAS [35]</td>
<td>429 with CAD</td>
<td>35–75</td>
<td>LDL: 2.97–4.91</td>
<td>Fluvastatin</td>
<td>2.5</td>
<td>Plaque regression</td>
<td>N/A 76 (NS)</td>
</tr>
<tr>
<td>LIPID [36]</td>
<td>9014 with prior UAP or MI</td>
<td>31–75</td>
<td>TC: 4–7 TG: &lt;5</td>
<td>Pravastatin</td>
<td>6.1</td>
<td>CAD death/Death/Stroke</td>
<td>76 (P &lt; 0.001)/78 (P &lt; 0.001)/81 (P = 0.48)</td>
</tr>
<tr>
<td>CARE [37]</td>
<td>4159 with prior MI</td>
<td>21–75</td>
<td>TC: &lt;6.2 LDL: 3.0–4.5 TG: &lt;4</td>
<td>Pravastatin</td>
<td>5</td>
<td>CAD death/MI Stroke</td>
<td>76 (P = 0.003)/69 (P = 0.03)</td>
</tr>
<tr>
<td>BIP [38,39]</td>
<td>3122 with CAD</td>
<td>45–74</td>
<td>TC: 4.65–6.5 LDL: ≤1.16 TG: ≤3.4</td>
<td>Bezafibrate</td>
<td>6.2</td>
<td>MI/sudden death</td>
<td>91 (NS)</td>
</tr>
<tr>
<td>VA-HIT [40]</td>
<td>2531 with CAD</td>
<td>&lt;74</td>
<td>LDL: ≤3.6 HDL: ≤1.0 TG: ≤3.4</td>
<td>Gemfibrozil</td>
<td>5.1</td>
<td>CAD death/MI Death</td>
<td>78 (P = 0.006)/89 (P = 0.23)/76 (P &lt; 0.001)</td>
</tr>
</tbody>
</table>

**NOTE:** Due to its importance, WOSCOPS is included despite excluding patients 65 years of age or older. CAD = coronary artery disease; CV = cardiovascular; HDL = high-density lipoprotein; LDL = low-density lipoprotein; MI = myocardial infarction; NA = not applicable; NS = not statistically significant; RCT = randomized controlled trial; TC = total cholesterol; TG = triglyceride; UAP = unstable angina pectoris.

* There were 13 events in the PSMG trial, 12 of which were in placebo group.
† There were 19 events in ACAPS, 14 of which were in placebo group.
‡ There were 72 events in LCAS, 41 of which were in placebo group.
In men older than 65, elevated total cholesterol, LDL cholesterol, and triglycerides were risk factors for cardiovascular events, while HDL cholesterol was not. In older women, low HDL was the strongest predictor, total cholesterol and triglycerides were weakly predictive, and LDL was not significant. The study did not perform age-specific analysis to detect gradients in risk with advancing age. Numerous other studies support elevated total cholesterol and LDL or low HDL as risk factors for cardiovascular disease [59–67].

A major concern in prescribing lipid-lowering agents to older adults stems from the findings of several studies in which lower cholesterol was either not cardioprotective or was associated with higher mortality [59,66,68–76]. Spontaneously low cholesterol has been associated with recently diagnosed cancer, impaired synthetic liver function, low

<table>
<thead>
<tr>
<th>Study</th>
<th>Search Strategy</th>
<th>Selection Criteria</th>
<th>No. of Patients/Characteristics (means)</th>
<th>No. of Trials</th>
<th>Outcomes</th>
<th>RR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blauw et al</td>
<td>MEDLINE through Sep 96; Current Contents through May 96; reference lists</td>
<td>RCTs, double-blinded, statins vs. placebo, strokes observed</td>
<td>20,472 Age: 57 yr TC: 250 mg/dL F/U: 4.3 yr</td>
<td>13</td>
<td>Fatal/ nonfatal stroke</td>
<td>0.69 (0.57–0.83)</td>
</tr>
<tr>
<td>Crouse et al</td>
<td>MEDLINE; reference lists of recent major trials</td>
<td>Clinical trials of statin monotherapy reporting strokes</td>
<td>9770 Age: 57 yr LDL: 175 mg/dL F/U: 4.5 yr</td>
<td>12</td>
<td>Stroke</td>
<td>0.73 (0.60–0.89)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Overall Primary prevention</td>
<td>0.85 (0.73–1.58)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Secondary prevention</td>
<td>0.69 (0.55–0.87)</td>
</tr>
<tr>
<td>Bucher et al</td>
<td>MEDLINE, EMBASE, meta-analyses, and reference lists through Oct 96</td>
<td>RCTs of special diet or drug vs. placebo or usual diet</td>
<td>100,524 Age: 51 yr TC: 165–305 mg/dL F/U: 5.5 yr No prior stroke or heart transplant</td>
<td>28</td>
<td>Fatal/ nonfatal stroke</td>
<td>0.95 (0.86–1.05)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Overall Statins</td>
<td>0.76 (0.62–0.92)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>CVD death</td>
<td>0.87 (0.79–0.94)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Overall Statins</td>
<td>0.69 (0.59–0.80)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Total mortality</td>
<td>0.94 (0.84–1.06)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Overall Statins</td>
<td>0.80 (0.71–0.90)</td>
</tr>
<tr>
<td>Ross et al</td>
<td>MEDLARS, Current Contents, reference lists</td>
<td>Published RCT% of statins vs. placebo, primary or secondary prevention, plaque regression outcomes reported</td>
<td>21,303 Age: 57 yr TC: 250 mg/dL F/U: ≥ 1 yr</td>
<td>17</td>
<td>Total mortality</td>
<td>0.76 (0.67–0.86)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Nonfatal MI</td>
<td>0.66 (0.57–0.77)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Fatal MI</td>
<td>0.61 (0.48–0.78)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Nonfatal stroke</td>
<td>0.69 (0.54–0.88)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Fatal stroke</td>
<td>0.77 (0.57–1.04)</td>
</tr>
<tr>
<td>Leng et al</td>
<td>MEDLINE, EMBASE, investigators, reference lists, pharmaceutical companies</td>
<td>RCTs of treatment (cholestyramine, nicotinic acid, colestipol, betapropylcarbinol, clofibrate, fish oil, sulodexide and/or niacin) vs. placebo</td>
<td>699 with lower-limb atherosclerosis Age: 18–80 yr TC: &gt; 185 mg/dL F/U: 2.1 yr</td>
<td>7</td>
<td>Total mortality</td>
<td>0.21 (0.03–1.17)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Nonfatal events</td>
<td>1.21 (0.80–1.83)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Progression of lower-limb atherosclerosis</td>
<td>0.47 (0.29–0.77)</td>
</tr>
<tr>
<td>Larosa et al</td>
<td>MEDLINE, reference lists</td>
<td>RCTs of statins vs. placebo with F/U ≥ 4 yr, clinical disease or death as endpoints</td>
<td>30,817 Age: 59 yr TC: 155–309 mg/dL F/U: 5.4 yr</td>
<td>5</td>
<td>Major coronary event</td>
<td>0.69 (0.64–0.74)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Death</td>
<td>0.79 (0.72–0.86)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>CVD death</td>
<td>0.73 (0.66–0.81)</td>
</tr>
</tbody>
</table>

CI = confidence interval; CVD = cardiovascular disease; F/U = follow-up; LDL = low-density lipoprotein; MI = myocardial infarction; RCT = randomized controlled trial; RR = relative risk; TC = total cholesterol.

[58]. In men older than 65, elevated total cholesterol, LDL cholesterol, and triglycerides were risk factors for cardiovascular events, while HDL cholesterol was not. In older women, low HDL was the strongest predictor, total cholesterol and triglycerides were weakly predictive, and LDL was not significant. The study did not perform age-specific analysis to detect gradients in risk with advancing age. Numerous other studies support elevated total cholesterol and LDL or low HDL as risk factors for cardiovascular disease [59–67].

A major concern in prescribing lipid-lowering agents to older adults stems from the findings of several studies in which lower cholesterol was either not cardioprotective or was associated with higher mortality [59,66,68–76]. Spontaneously low cholesterol has been associated with recently diagnosed cancer, impaired synthetic liver function, low
albumin, and low hemoglobin, all of which may be markers of comorbidity or frailty [75]. In older adults, low total cholesterol may reflect significant comorbidity or frailty, and hence, decreased life expectancy. Most negative studies did not control for these confounding factors. While spontaneously low cholesterol has been associated in some with higher mortality, there is no evidence to suggest that low cholesterol from lipid-lowering therapy can cause adverse outcomes. Excess cases of breast cancer in the CARE trial were not observed in other trials, suggesting an effect due to chance [37].

Elevated total or LDL cholesterol and low HDL cholesterol should be considered cardiovascular risk factors in active adults over the age of 75 with no significant comorbidity [77]. Treatment should also be considered for older adults with subclinical carotid wall thickening, evidence of peripheral vascular disease, major changes on electrocardiogram, wall motion abnormality, or impaired ejection fraction who are at high risk for clinical disease [78].

**Antihypertensive Therapy**

Hypertension in the elderly is a major independent risk factor for coronary artery disease, stroke, heart failure, mortality, and dementia [79,80]. The prevalence of hypertension rises from 50% in persons aged 50 to 69 years to 65% in those aged 80 or older [81]. Pharmacologic treatment of hypertension in the elderly reduces cardiovascular morbidity and mortality [82,83]. Older hypertensive patients benefit more from treatment than do younger patients. According to 1 meta-analysis, the number needed to treat to prevent a single event in a middle-aged patient is 86, compared with 29 in older persons [84].

In 1997, the National High Blood Pressure Education Program released updated guidelines on the management of hypertension in the elderly [85]. The goal of therapy is a blood pressure of less than 140/90 mm Hg. The guidelines highlight the importance of lifestyle modification, which can reduce the reoccurrence of hypertension and cardiovascular events by 53% in men and women 60 to 80 years of age previously diagnosed with hypertension [86]. Diuretics and β blockers remain the first-line agents, with other agents recommended for specific comorbidity as indicated.

The recently published STOP-2 trial compared conventional first-line therapy with diuretics and β blockers to newer therapy with either ACE inhibitors or calcium channel blockers in 6614 subjects aged 70 to 84 [87]. If the target blood pressure of 160/95 mm Hg could not be achieved, treatment for patients receiving β blockers or ACE inhibitors was supplemented with diuretics, while those on diuretics or calcium channel blockers received additional β blockers. After a follow-up period of 4 years, there was no difference among the 3 groups in the combined incidence of fatal cardiovascular events. The findings of this trial suggest that the different approaches to hypertension therapy in older adults are equally efficacious. In addition, combination therapy is common, as over 46% of patients were on 2 or more agents at the conclusion of the trial. However, economic analysis suggests that diuretics or β blockers as initial treatment are far more economical than treatment with newer drugs [84].

Despite the guidelines and solid evidence for treating hypertension, many patients are either undertreated or not treated at all. Through national education programs, awareness of hypertension in American adults has risen from 51% in 1976-80 to just under 70% in 1991-94 [85]. However, only 53.6% of hypertensive patients are receiving treatment, and only 27.4% have their blood pressure controlled. Inappropriate management of hypertension in the elderly is partially due to patient noncompliance. In a retrospective cohort of elderly Medicare recipients, the rate for filling antihypertensive prescriptions was only 49% [88]. In addition, adherence with guidelines may not be optimal [89].

**Conclusion**

Given the high prevalence of cardiovascular disease in the elderly, the potential impact of appropriate drug prescribing on reducing morbidity is great. Review of the literature has demonstrated that elderly patients with cardiac disease are receiving suboptimal therapy. There is strong evidence to support β blocker use to reduce reinfarction rates and mortality following myocardial infarction. Multidisciplinary interventions have been demonstrated to reduce rehospitalization of patients with heart failure. Lipid-lowering therapy should be considered in older adults with no significant comorbid illnesses, with statins being the agents of choice. Gemfibrozil may be a useful alternative for patients with low HDL. There is growing evidence to suggest that uncontrolled hypertension is a risk factor for dementia as well as cardiovascular disease and that occurrence rates for these endpoints can be reduced with appropriate treatment.

**References**


20. Poole-Wilson PA, Pitt B. Results of the ELITE II study. Presented at the 72nd scientific sessions of the American Heart Association; 1999 Nov.


30. Effects of pravastatin in patients with serum total cholesterol levels from 5.2 to 7.8 mmol/liter (200 to 300 mg/dl) plus two additional atherosclerotic risk factors. The Pravastatin Multinational Study Group for Cardiac Risk Patients. Am J Cardiol 1993;72:1031–7.


