

Atherosclerotic risk factor control in patients with peripheral arterial disease

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Background: The presence of peripheral arterial disease (PAD), even in the absence of overt coronary artery disease (CAD), confers the same relative risk of death from a cardiovascular cause as in patients with a previous cardiovascular event. Current guidelines recommend atherosclerotic risk factor–reduction strategies in PAD patients identical to those in patients with a recent coronary event. The purpose of this study was to determine the status of atherosclerotic risk factor control in patients with PAD.

Methods: We analyzed the records of patients treated at 2 regional clinics serving 92,940 individuals. Full examination, laboratory, and pharmacy data were available for all patients. Pharmacy data were analyzed to determine prescriptions for β -blocker therapy, angiotensin-converting enzyme inhibitors, and lipid-lowering agents. Lipid control was assessed through fasting lipid data. Glycemic control in diabetics was evaluated by using hemoglobin A_{1c} levels.

Results: We administratively identified 2839 patients with a diagnosis of PAD. The exclusion of 1106 patients with a diagnosis of CAD or validated not to have PAD resulted in a cohort of 1733 patients. Of these, 33.1% (574/1733) were currently receiving β -blockers, 28.9% (500/1733) were receiving an angiotensin-converting enzyme inhibitor, and 31.3% (543/1733) were receiving a statin. Most patients (92%; 1594/1733) had a recent blood pressure recorded. However, 56% (893/1594) had a systolic blood pressure of 130 mm Hg or higher, 45.5% (726/1594) had a diastolic blood pressure of 80 mm Hg or higher, and 13.6% (217/1594) had a diastolic blood pressure of 90 mm Hg or higher. Screening fasting lipid profiles were found in 62.6% (1085/1733) of patients, 56% (508/912) had a low-density lipoprotein of 100 mg/dL or higher, and 21% (187/912) had a value of more than 130 mg/dL. In patients with diabetes, a hemoglobin A_{1c} level of 7.0% or higher was found in 54.2% (198/365) of patients.

Conclusions: Despite national consensus of PAD as a CAD equivalent, patients are currently undertreated with regard to atherosclerotic risk factor modification. Until broader recognition of this disease process exists, vascular surgeons must continue to champion medical as well as surgical treatments for these patients. (*J Vasc Surg* 2005;41:816-22.)

Peripheral arterial disease (PAD) is acknowledged to be a common disease process in elderly patients. Age-adjusted data from several domestic and European studies suggest that the prevalence of PAD nears 15% to 20% in patients older than 65 years.¹⁻⁶ Patients with PAD incur a 3.1-fold increase in all-cause mortality over patients without PAD and a 6.6-fold increased risk of death from coronary artery disease.⁷ Only 10% of patients with lower extremity ischemia have normal coronary arteries by cardiac catheterization, and 28% have severe coronary artery disease.⁸ Furthermore, the presence of PAD, even in the absence of a history of coronary artery disease (CAD), confers the same relative risk of death from a cardiovascular cause as in patients with a previous cardiovascular event.^{9,10}

Taken together, these data suggest that patients with PAD should be considered for secondary prevention strategies comparable to those for patients with a previous

myocardial infarction (MI). The American Heart Association and the National Cholesterol Education Program recommend identical atherosclerotic risk reduction strategies for both PAD and CAD patients.^{11,12} These recommendations include initiation of β -blockade and angiotensin-converting enzyme inhibitors (ACEi) in all patients, use of statins to achieve a low-density lipoprotein (LDL) of less than 100 mg/dL, antiplatelet therapy, and smoking cessation. The purpose of this study was to determine the status of atherosclerotic risk factor modification in patients diagnosed with PAD.

METHODS

All patients in this study were members of a large, group-model, not-for-profit managed care system serving approximately 405,000 patients. Full outpatient medical, pharmacy, laboratory, and radiology information is stored in an electronic medical record, allowing for current and comprehensive analysis.

Patients from 2 large medical offices serving 92,940 patients were administratively screened for a diagnosis of PAD as defined by (1) an International Classification of Diseases 9th revision code for claudication or PAD (443.9), (2) a previous peripheral revascularization procedure, (3) a prescription for either pentoxifylline or cilostazol, (4) an ankle-brachial index (ABI) evaluation or full noninvasive arterial study, or (5) confirmation by a vascular surgeon. Patients who were validated not to have PAD

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because of an ABI greater than 0.9 (in the absence of any revascularization procedure) or on determination by a vascular surgeon were excluded. Patients with CAD (as defined by a history of MI, coronary revascularization, coronary catheterization revealing at least 50% stenosis of at least one vessel, positive thallium stress test with electrocardiogram changes indicating ischemia, or unstable angina) were also excluded.

Centralized computerized pharmacy records for all affiliated regional pharmacies were used to identify any patient in the cohort who picked up at least one prescription from any of the following classes of medications between January 1, 2004, and March 31, 2004: β -blockers, ACEi, angiotensin receptor blockers, hydroxymethylglutaryl coenzyme A (HMG-CoA) reductase inhibitors (statins), bile acid sequestrants, fibric acid derivatives, ezetimibe, antiplatelet agents, or anticoagulants. As members of a managed care organization, most patients have a significant financial advantage in having their prescriptions filled through an affiliated pharmacy. Patients are limited to a 60-day supply of medications.

Computerized laboratory records were queried to identify all patients in the cohort with a fasting lipid profile between March 1, 2003, and March 31, 2004. Diabetic patients were identified by cross-referencing the PAD cohort with an internal validated diabetes registry. Glycemic control in this group was evaluated by using hemoglobin A_{1c} levels during the same interval. The most recent blood pressure value for each patient was obtained from the patient's medical record.

In an effort to ascertain treatment efficacy in this patient population, we analyzed the number of patients receiving a lipid-lowering medication with lipid outcomes and antihypertensive medications with blood pressure data. Patients who had at least one claim for an HMG-CoA reductase inhibitor during the study period were assigned to the "statin users" cohort, while those without such a claim were assigned to the "statin nonusers" cohort. Patients who had at least one claim for an ACEi, angiotensin receptor blocker, or β -blocker were assigned to the "hypertension medication users" cohort, whereas those without a such claim were assigned to the "hypertension medication nonusers" cohort. Patients were categorized as "at goal" for LDL cholesterol (LDL-C) if their LDL-C reading value was less than 100 mg/dL. Diabetic patients were categorized as "at goal" for blood pressure if their systolic and diastolic reading values were less than 130 and 80 mm Hg, respectively. Nondiabetic patients were categorized as "at goal" for blood pressure if their systolic and diastolic reading values were less than 140 and 90 mm Hg, respectively. Assessments were made of the distributions of the LDL-C, total cholesterol, systolic, and diastolic reading values for each cohort, and it was determined that none was normally distributed. Thus, to assess the relationship between LDL-C, total cholesterol, systolic, and diastolic reading values and medication use, individual nonparametric Wilcoxon rank sum tests were performed between the cohorts for each value. To assess the relationship between "at goal"

and medication use, individual χ^2 tests of association were performed between the cohorts for each goal.

RESULTS

A diagnosis of PAD was identified in 2839 patients out of 92,940 records examined. The exclusion of 421 patients confirmed not to have PAD resulted in 2418 patients with a diagnosis of PAD. Of the 92,940 patients, 15.8% ($n = 14,691$) were older than 65 years of age, and 56.8% were female. In the PAD group, 69.7% ($n = 1685$) were more than 65 years old; thus, the prevalence of PAD in patients older than 65 years of age was 11.5% (1685/14,691). The exclusion of 685 patients with a concomitant diagnosis of CAD and the 421 patients confirmed not to have PAD resulted in 1733 patients appropriate for this analysis. The average age of this population was 67.5 years. A mild female predominance (57.2%) was noted.

Of the 1733 patients, only 33.1% ($n = 574$) were currently taking a β -blocker. Prescriptions for an ACEi were received by 28.9% (500/1733) of patients, and an additional 3.6% (62/1733) received an angiotensin receptor blocker. HMG-CoA reductase inhibitors (statins) were received by 31.3% (543/1733) of patients. Few patients received bile acid sequestrants (cholestyramine or colestipol; 0.35%; 6/1733), fibrates (gemfibrozil or fenofibrate; 2.0%; 35/1733), or ezetimibe (0.29%; 5/1733).

Aspirin use was not fully recorded in our system because it is an over-the-counter medication that is not accurately tracked in the pharmacy database. Prescription antiplatelet therapies were documented in 4.6% of patients (clopidogrel, $n = 57$; dipyridamole/aspirin, $n = 23$). Anticoagulation with warfarin was documented in 257 (14.8%) patients.

Screening lipid profiles were found in 62.6% (1085/1733) of patients. No lipid profile or cholesterol determination was performed over the 1-year period in 648 patients. Average total cholesterol was 195 ± 41.6 mg/dL, and 42.7% (464/1085) had a total cholesterol of 200 mg/dL or higher. The average LDL was 106 ± 33.4 mg/dL. More than half of the patients who had a lipid screen (55.7%; 508/912) had an LDL of 100 mg/dL or higher, and 20.5% (187/912) had a value of more than 130 mg/dL. Average high-density lipoprotein (HDL) was 54 ± 15.9 mg/dL, and only 16.7% (168/1005) had an HDL level less than 40 mg/dL. Non-HDL cholesterol is used as a secondary goal in patients with increased triglyceride levels and reflects the overall atherogenic lipoprotein burden. In our patients, average non-HDL cholesterol was 142 ± 39.7 mg/dL (range, 38-429 mg/dL). More than half of the population (58.6%; 589/1005) had a non-HDL cholesterol more than 130 mg/dL. Triglyceride levels were found in 940 patients, with an average of 185 mg/dL, and 52.3% had a value of 150 mg/dL or higher. PAD patients taking a lipid-lowering agent had significantly lower mean LDL and total cholesterol levels (Table I) and were more apt to have reached their target LDL goal (Table II) than patients not taking a statin.

Table I. Relationships between lipid and blood pressure values and medication use in PAD patients

Measurement/medication group	Mean (n, SD)	P value*
LDL-C (mg/dL)		<.001
Statin users	99.1 (448, 31.4)	
Statin nonusers	112.9 (462, 34.1)	
Total cholesterol (mg/dL)		<.001
Statin users	189.4 (476, 37.8)	
Statin nonusers	199.8 (607, 43.9)	
Systolic blood pressure (mm Hg)		<.001
HTN medication users [†]	134.4 (845, 19.5)	
HTN medication nonusers	129.2 (747, 17.9)	
Diastolic blood pressure (mm Hg)		.584
HTN medication users [†]	76.5 (845, 11.8)	
HTN medication nonusers	76.4 (747, 10.0)	

PAD, Peripheral arterial disease; LDL-C, low-density lipoprotein cholesterol; HTN, hypertension.

*Wilcoxon rank sum test.

[†]Includes angiotensin-converting enzyme inhibitor, angiotensin receptor blocker, or β -blocker.

Table II. Relationships between lipid and blood pressure goals and medication use in PAD patients

Measurement/medication group	% at Goal (n)	P value*
LDL-C goal [†]		<.001
Statin users	52.0 (448)	
Statin nonusers	36.6 (462)	
Blood pressure goal [‡]		<.001
HTN medication users [§]	49.6 (845)	
HTN medication nonusers	64.3 (747)	

PAD, Peripheral arterial disease; LDL-C, low-density lipoprotein cholesterol; HTN, hypertension.

* χ^2 test.

[†]>Goal was LDL-C <100 mg/dL.

[‡]Goal was <140/90 mm Hg for nondiabetic and <130/80 mm Hg for diabetic patients.

[§]Includes angiotensin-converting enzyme inhibitor, angiotensin receptor blocker, or β -blocker.

Diabetes mellitus was present in 23.9% of patients (n = 414). Most of these patients (88.2%; n = 365) had a documented hemoglobin A_{1c} level. Mean hemoglobin A_{1c} was 7.41% \pm 1.35%. A hemoglobin A_{1c} level of 7.0% or higher was found in 54.2% (198/365) of patients. Only 26.3% (96/365) had a value of 8.0% or higher, and 11.2% (41/365) had a value of 9.0% or higher.

Most patients (92%; 1594/1733) had a recent blood pressure recorded. However, 56.0% (893/1594) had a systolic blood pressure of 130 mm Hg or higher, and 34.1% (544/1594) had a systolic blood pressure greater than 140 mm Hg. The control of diastolic blood pressure was similar: 45.5% (726/1594) had a diastolic blood pressure of 80 mm Hg or higher, 19.5% (311/1594) had a diastolic blood pressure of 85 mm Hg or higher, and 13.6% (217/1594) had a diastolic blood pressure of 90 mm Hg or higher. Systolic blood pressure was significantly higher in patients taking antihypertensive medications; this may simply reflect an appropriate diagnosis. Diastolic blood pressure control

was identical between groups on or off medications (Table I). Patients taking antihypertensive medications were more likely to meet their recommended¹³ blood pressure goal than those patients not on antihypertensives (Table II).

DISCUSSION

This analysis of unselected patients diagnosed with PAD by primary care physicians attempts to characterize the current care of patients with systemic manifestations of atherosclerosis and the absence of concomitantly diagnosed CAD. To our knowledge, it is the largest and most detailed analysis of pharmacologic and laboratory data in this population. Perhaps not surprisingly, we found that patients with PAD are significantly undertreated and fall well short of nationally established goals for secondary prevention (Table III).

Patients' (and many physicians') attention is often focused on resolution of claudication symptoms through increasingly aggressive revascularization procedures. A more accurate assessment of threat implicates the heart and not the leg. The menace of limb threat in patients with claudication is low,¹⁴⁻¹⁶ but their inherent cardiovascular morbidity and mortality is considerable.^{7,17,18} This compelling evidence led to the national acceptance of PAD as a CAD equivalent with broad recommendation for the use of identical risk reduction strategies in both of these patient populations.¹¹

Despite these recommendations, physician and patient recognition of PAD is poor, and its treatment is generally performed with less attention when compared with patients with CAD.^{4,5,19} Reasons for this disparity may include a simple lack of education, the paucity of focused public health initiatives, apathy, or inexperience. Vascular medicine is a critical component of vascular surgeons' education and practice; however, when compared with other specialties, vascular surgeons may be the least likely to initiate these therapies.^{4,20}

The administration of a β -blocker in secondary prevention of a cardiovascular event may decrease mortality by 26% to 39% and is supported by ample level I evidence. In a retrospective review of 201,752 patients after acute MI, β -blockade was found to reduce mortality by 40% at 2 years.²¹ Freemantle et al²² performed a meta-analysis of 82 randomized trials encompassing 54,234 patients and confirmed a 23% reduction in the odds of death in patients taking β -blockers after MI. It seems to be immaterial which preparation is used (eg, atenolol, metoprolol, or propranolol),²³ but it should be continued indefinitely.²⁴ As a coronary equivalent, all PAD patients should have β -blockade as part of their medical regimen unless absolutely contraindicated. There are few data to support the idea that β -blockers may worsen symptoms of claudication, although this has been a historical concern.^{25,26}

Evidence for the use of ACEi in patients with PAD stems predominantly from the Heart Outcomes Prevention Evaluation trial, in which 9297 high-risk patients without ventricular dysfunction were randomized to ramipril or placebo. In this study, ramipril reduced the risk of cardio-

vascular death, MI, and stroke by 22% over 5 years.²⁷ Subgroup analyses found that 4051 of the 9297 patients had a history of PAD, claudication, or an ABI less than 0.9. This cohort realized a similar benefit from ramipril when compared with patients without PAD, independent of ramipril's effect on blood pressure. The magnitude of this decrement in cardiovascular risk is equivalent to the benefit seen in other studies from β -blockers, aspirin, or lipid-lowering agents. ACEi may achieve even greater benefit in patients with mild congestive heart failure or type 2 diabetes mellitus.^{28,29}

In addition to β -blockade and ACE inhibition, strict control of lipids is well supported as an adjunct in reducing cardiovascular events after acute MI. A correlation between serum cholesterol levels and coronary risk was established through epidemiologic studies.³⁰ Several subsequent large prospective randomized trials³¹⁻³⁴ and a meta-analysis³⁵ have confirmed that statin use results in a 20% to 30% reduction in cardiovascular and all-cause mortality in patients with CAD. In our study, 72.5% of patients either had no cholesterol level checked or had an LDL over the national target level¹² of 100 mg/dL. With recent evidence suggesting that a greater benefit may be achieved with even stricter LDL control,³⁶ these data leave great room for improvement.

Although the intent of aggressive lipid control in patients with PAD is to reduce cardiovascular complications, there is evidence that the use of statins may also specifically limit PAD progression and improve limb function. Mondillo et al³⁷ performed a prospective, randomized, double-blind trial on the effect of daily simvastatin on walking performance in 86 patients with PAD. The patients randomized to the statin group achieved a significant improvement in pain-free walking distance, maximal walking distance, ABI, and claudication symptoms at 6 months. There is also evidence to suggest that lipid control may limit the progression of atherosclerosis in peripheral arteries.³⁸⁻⁴⁰ It is interesting to note that McDermott et al⁴¹ were able to document improvements in leg functioning in patients taking a statin that were independent of the change in cholesterol levels or the presence or absence of PAD. This suggests some as-yet-unexplained non-cholesterol-lowering property of these medications.

The Seventh Joint National Committee (JNC 7) on Prevention, Detection, Evaluation and Treatment of high blood pressure did not recommend a specific target blood pressure for patients with isolated CAD or its equivalents¹³; however, it did recommend starting treatment for healthy patients with a systolic blood pressure greater than 140 mm Hg or a diastolic blood pressure greater than 90 mm Hg. This level of control was not achieved in our PAD population in 34% of patients. In patients with diabetes mellitus or chronic kidney disease, a target blood pressure less than 130/80 mm Hg is recommended.¹¹ No definite first-line agent is described, but the evidence listed in the preceding paragraphs strongly supports the use of β -blockers and ACEi.

Table III. Achievement of national standards for atherosclerotic risk factor control in patients with PAD

<i>Risk factor</i>	<i>AHA/ACC goal*</i>	<i>Achieved goal</i>	
Beta-blockers	100% of patients	33.1%	
ACE inhibitors	100% of patients	28.9%	
Lipid management	LDL <100 mg/dL	23.3%	
Diabetes management	Hemoglobin A _{1c} <7.0%	40.3%	
Blood pressure	Systolic	<140 mm Hg	65.9%
		<130 mm Hg	44.0%
	Diastolic	<90 mm Hg	86.4%
		<80 mm Hg	54.5%
Smoking	Complete cessation	Not available	
Antiplatelet agents	100% of patients	Not available	

PAD, Peripheral arterial disease; *ACE*, angiotensin-converting enzyme; *LDL*, low-density lipoprotein.

*Adapted from the American Heart Association (AHA)/American College of Cardiology (ACC) guidelines for preventing heart attack and death in patients with atherosclerotic cardiovascular disease.¹¹

Patients with diabetes incur an increased risk of developing PAD, and most diabetics die from cardiovascular disease. Aggressive control of hyperglycemia reduces the risk of all-cause mortality by 6% in diabetics, but it seems to affect microvascular disease rather than having any demonstrable benefit on macrovascular or cardiovascular morbidity.⁴² National guidelines suggest intensive control of blood glucose to a target hemoglobin A_{1c} less than 7.0%.⁴³ Of the 414 patients with diabetes in our study, only 167 (40.3%) had a hemoglobin A_{1c} that was within that goal.

Our study has some limitations, including that we could not validate the diagnosis of PAD in all of the patients with noninvasive testing. It is our contention, however, that patients diagnosed with PAD should be treated as such unless there is evidence to contradict that diagnosis. In addition, data regarding tobacco and aspirin use are not robust in our system but are arguably two of the most important factors in PAD etiology and treatment.

The data in this study support those of others^{5,44-46} finding a lack of aggressive risk factor control in patients with PAD. It is striking that this specific group could benefit so greatly with what seems to be little more than improved public and physician education. There seems to be a somewhat paradoxical physician treatment bias skewed toward claudication symptom relief rather than essential risk factor reduction. It is our hope that data such as these will engender broader use of risk factor-control strategies that are well established for the secondary prevention of cardiovascular morbidity.

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DISCUSSION

Dr William Pevec, MD (Sacramento, Calif). Based on a large number of clinical trials, the third report of the National Cholesterol Education Program published in *Circulation* in 2002 concluded that peripheral arterial disease whether diagnosed by ABI, lower limb blood flow studies, or clinical symptoms is a coronary heart disease equivalent. Patients found to be at risk for coronary heart disease can benefit from risk-reducing therapies. However, the 2001 updated guidelines of the American Heart Association and American College of Cardiology guidelines for preventing heart attack and death in patients with atherosclerotic cardiovascular disease published in *Circulation* in 2001 noted that multiple studies of the actual use of these therapies has shown slow improvement, but continues to show a discouraging conclusion that a large proportion of patients at risk for cardiovascular disease do not get the risk-reducing therapies. The report today by Dr Rehring and his associates confirms that disappointing conclusion. I have several questions for the authors.

The authors used relatively soft criteria to define peripheral arterial disease. I agree with their bias mentioned in the manuscript that it is better to treat these patients anyway. However, it is possible that the primary physicians caring for these patients either did not recognize that these patients had peripheral arterial disease or were unconvinced by that diagnosis. My question is might this have been a factor in the relatively low incidence of risk reduction therapy in the study population?

My second question is medication use was determined as described in the manuscript by reviewing prescriptions dispensed by single clinic pharmacy over a three-month period. Could this result in an underestimation of the use of these drug therapies if some study subjects had their prescriptions filled at a different pharmacy or outside this three-month window?

My third question. The authors described a majority of their patients being women which is counter to the usual distribution of disease. I am curious if they could comment on that interesting finding.

The fourth question is the American Heart Association and American College of Cardiology guidelines are somewhat vague about the indications for beta blockers in patients without overt coronary disease, although these guidelines do suggest that ACE inhibitors be considered in patients with vascular disease. These medications are clearly indicated in patients with hypertension and peripheral arterial disease. In this study half the patients had hypertension, a third were on beta blockers, and a third were on ACE inhibitors or angiotensin receptor blockers. Did the authors look at how many patients with hypertension were on either one of these drugs and how many patients with hypertension were on neither of these classes of drugs? This data might give a little more accurate estimate of the true efficacy of this treatment.

And finally I think the most important question is what should we do with these data? How should vascular surgeons intervene on patients with peripheral arterial disease and inadequate risk factor modification? Should the surgeon prescribe the beta blockers, ACE inhibitors, insulin and statins? Does this run the risk of too many cooks spoiling the soup? And also if surgeons prescribe these

medications, should the surgeon then be responsible for followup on the blood pressure, serum glucose and cholesterol profile of these patients in a serial fashion?

I would like to thank Dr Rehring and his associates for getting me the manuscript well in advance of the meeting and I would like to thank the program committee for inviting me to discuss this very good paper. Thank you.

Dr Thomas F. Rehring (Denver, Colo). The first question reflected the relatively soft criteria utilized to diagnose peripheral arterial disease and how that may have impacted risk reduction treatment by primary care physicians. While utilization of a diagnostic code to identify patients in an administrative database is a fairly standard technique, a lack of conviction by the primary care provider may certainly be a confounder. It is still our bias however that if a health care provider labels a patient with a diagnosis of peripheral arterial disease, they should be treated as such. If the primary care doctor is unsure of the diagnosis, the patient should be referred for either a diagnostic study or to a vascular surgeon for consultation. We captured both of these groups by analyzing radiology data for results of a noninvasive arterial exam and evaluating all patients with visits to a vascular surgeon.

In response to your second question, all of the patient medication dispensations are entered in a region-wide, centralized electronic pharmacy record, so if they fill their prescription at any pharmacy in the Kaiser system in the Denver, Boulder, or Colorado Springs area, it would have been captured. These data were obtained over a 90-day period. As prescription refills are limited to 60 days we hoped to capture the vast majority of patients.

We thought the finding of 57% of the cohort being female interesting as well. When I looked at other large PAD prevalence studies however, others have noted that it affected the sexes equally.

With regard to your fourth question stratification of the anti-hypertensive data for efficacy is an excellent suggestion and will be added to the final manuscript.

Your last and obviously the most relevant question is how should we manage these patients? Is it our job to manage their risk factors? I wouldn't presume to tell each surgeon how to manage their practice as patient flow characteristics can be quite variable between physicians. I firmly believe it *is* our job to recommend these risk-reduction strategies to primary care physicians. The decision to initiate the therapy for the primary care doctor to follow must be performed at your own comfort level and on an individual basis. In my own practice, I have no problem initiating both antihypertensive therapy (particularly beta blockers) and statin therapy. This has been welcomed by the primary care doctor.

Dr. Gregory Moneta, MD (Portland, Ore). The Peripheral Arterial Disease Detection, Awareness and Treatment (PARTNERS) study indicated a large underdiagnosis of peripheral arterial disease. My first question would be is there any type of screening program in place or planned for that diagnosis in your patient population? And the second one would be that the American Diabetes Association has actually done analysis of risk factor modification in patients with diabetes and concluded we weren't doing

a very good job with hypertension and we weren't doing a very good job with insulin levels but actually the modification of cholesterol levels was considerably improved over what it was 10 years ago. So my question would be is there a difference in the use of these medications in patients who have diabetes or don't have diabetes in your population?

Dr Rehring. As you recognized, we do believe PAD is underdiagnosed, however we have no plans for initiating a screening program as it would be fairly labor intensive in our population of over 400,000 patients. We have initiated region-wide CME programs on PAD.

As to your second question, I do not have data to determine whether diabetics received better management.

Dr William Krupski, MD (San Francisco, Calif). You mentioned statins just briefly. There has been a lot of literature about use of statins in the coronary patient and to use statins even in the face of normal lipids. Do you recommend using statins in our patients even if their lipids are normal and what parameters do you shoot for now in terms of LDL and HDL levels in view of evidence that lower is better?

Dr Rehring. There is interesting evidence with several of these medications that their effect on reductions in cardiovascular morbidity and mortality are independent of their designed effects. That is, ACE inhibitors improve cardiovascular morbidity and

mortality independent of their effects on blood pressure and statins decrease in cardiovascular morbidity and mortality independent of their effect on cholesterol levels. Statins also improve walking performance, pain-free walking distance, and claudication symptoms independent of their effect on cholesterol. To your point, yes, I think we should initiate statin therapy in our patients with peripheral artery disease. As to targeting a lower LDL in this group, I believe you are referring to a recent study of CAD patients that suggested that patients with an LDL of 70 had better outcomes than those with a target of 100. Another study has failed to duplicate those findings using a separate statin, so I think the jury is still out. Until more definitive data is available, I continue to target an LDL of 100.

Dr Anil Hingorani, MD (Brooklyn, NY). How are you managing your relationship with the primary care physician if you are starting the patients on beta blockers or statins and are you following the liver function tests?

Dr Rehring. Yes, that can be an uncomfortable situation. In some practices and there are primary care doctors who are quite capable and current in their management of these risk factors. Others I've found will welcome some direction. I think it is our responsibility to be experts in this area. In my practice, I recommend and initiate therapy and then turn them over to their primary care doctor for follow-up. That scheme has been welcomed.

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