lesion debulking and intravascular ultrasound need to be further defined in randomized trials. Although there are some reports favoring their use in ULM PCI, they are based on small, nonrandomized, single-center trials.[12,14]

Study Limitations
The ULTIMA investigators acknowledge a number of important limitations to this study. The registry's design and the absence of a surgical control group limit the outcome analysis to comparisons with historical controls. However, such controlled randomized clinical trials are unlikely, given the prohibitive sample size and cost limitations. The use of platelet glycoprotein IIb/IIIa inhibitors was limited in this study (4.3%); additional benefit in periprocedural outcomes might be expected with more liberal use of these agents.[15,16] ∎

References
1. Takaro T, Hultgren HN, Lipton MJ, Detre KM, for participants in the Study Group. The VA cooperative randomized study of surgery for coronary arterial occlusive disease. II. Subgroup with significant left main lesions. *Circulation*. 1976;3:107–117.
2. Farinha JB, Kaplan MA, Harris CN, et al. Disease of the left main coronary artery: surgical treatment and long term follow-up in 267 patients. *Am J Cardiol*. 1978;42:124–128.
3. Varnauskas E, for the European Coronary Surgery Study Group. Twelve-year follow-up of survival in the randomized European Coronary Surgery Study. *N Engl J Med*. 1988;319:332–337.
4. O'Keefe JH, Hartzler GO, Rutherford BD, et al. Left main coronary angioplasty: early and late results of 127 acute and elective procedures. *Am J Cardiol*. 1989;64:144–147.
5. Eldar M, Schulhoff RN, Hertz I, et al. Results of percutaneous transluminal coronary angioplasty of the left main coronary artery. *Am J Cardiol*. 1991;68:255–256.
6. Crowley ST, Morrison DA. Percutaneous transluminal coronary angioplasty of the left main coronary artery in patients with rest angina. *Cathet Cardiovasc Diagn*. 1994;33:103–107.
7. Stertzer SH, Myler RK, Insel H, et al. Percutaneous transluminal coronary angioplasty in left main stem coronary stenosis: a five year appraisal. *Int J Cardiol*. 1985;9:149–159.
8. Ellis SG, Tamai H, Nobuyoshi M, et al. Contemporary percutaneous treatment of unprotected left main coronary stenoses: initial results from a multicenter registry analysis 1994–1996. *Circulation*. 1997; 96:3867–3872.
9. Macaya C, Alfonso F, Iniguez A, et al. Stenting for elastic recoil during coronary angioplasty of the left main coronary artery. *Am J Cardiol*. 1992;70:105–107.
10. Garcia-Robles JA, Garcia E, Rico M, et al. Emergency coronary stenting for acute occlusive dissection of the left main coronary artery. *Cathet Cardiovasc Diagn*. 1993;30:227–229.
11. Wong P, Wong V, Tse K-K, et al. A prospective study of elective stenting in unprotected left main coronary disease. *Cathet Cardiovasc Intervent*. 1999;46:153–159.
12. Park SJ, Hong MK, Lee CW, et al. Elective stenting of unprotected left main coronary artery stenosis: effect of debulking before stenting and intravascular ultrasound guidance. *J Am Coll Cardiol*. 2001;38:1054–1060.
13. Silvestri M, Barragan P, Sainsous J, et al. Unprotected left main coronary artery stenting: immediate and medium-term outcomes of 140 elective procedures. *J Am Coll Cardiol*. 2000;35:1543–1550.
14. Kosuga K, Tamai H, Ueda K, et al. Initial and long-term results of directional coronary atherectomy in unprotected left main coronary artery. *Am J Cardiol*. 2001;87:838–843.
15. The EPISTENT Investigators. Randomized placebo-controlled and balloon-angioplasty-controlled trial to assess safety of coronary stenting with the use of platelet glycoprotein-IIb/IIIa blockade: evaluation of platelet IIb/IIIa inhibitor for stenting. *Lancet*. 1998;352:87–92.
16. The PURSUIT Trial Investigators. Platelet glycoprotein IIb/IIIa in unstable angina: receptor suppression using integrilin therapy: inhibition of platelet glycoprotein IIb/IIIa with eptifibatide in patients with acute coronary syndromes. *N Engl J Med*. 1998;339:436–443.

Atherosclerosis

Exploring the Relationship Between Hyperlipidemia and Aortic Stenosis

Reviewed by Arthur E. Weyman, MD
Cardiac Unit, Massachusetts General Hospital, Boston, MA

Valvular aortic stenosis is a relatively common disorder, occurring in between 2% and 7% of the population older than 65 years. Aortic stenosis is most frequently caused by progressive calcification and degeneration of the aortic cusp. The disease typically shows a progressive course, which accelerates after the threshold to mild stenosis has been crossed. Similar pathologic mechanisms of aortic valve stenosis and atherosclerosis have been reported, but the relationship of cardiovascular risk factors to progression of aortic stenosis has been inconsistent.

Progression of Aortic Valve Calcification: Association with Coronary Atherosclerosis and Cardiovascular Risk Factors
Pohle K, Mäffert R, Ropers B, et al.
Circulation. 2001:104:1927–1932.

In this study the investigators used electron beam tomography (EBT) to quantitate the degree of aortic valve calcification in a group of 104 patients (age 64.7 ± 8 years, 89 male) to determine the rate of progression and the influence of cardiovascular risk factors on the course of calcium accumulation. They also investigated the influence of the low-density lipoprotein cholesterol (LDL) level, other standard cardiovascular risk factors, and the extent of coronary calcification on the progression of aortic valve calcification, as quantified by EBT. Patients were selected because of a positive EBT scan for aortic valve calcium and coronary calcium. Aortic valve calcium was quantified using a volumetric score. EBT was repeated at a

mean interval of 15 months (range 10–36 months) and the progression of aortic valve calcium and coronary calcium determined. Patients were divided into two groups according to their LDL levels. Group 1 contained 57 patients with LDL ≤ 130 mg/dL, and Group 2 contained 47 patients with LDL > 130 mg/dL.

The mean progression of aortic valve calcium for Group 1 patients was 9% ± 22%, whereas for Group 2 patients it was 43% ± 44% ($P ≤ .001$). There was no significant influence of the amount of aortic calcification in the initial scan on the rate of progression. There was a significant correlation between the progression of coronary and aortic valve calcification ($R = .42$, $P < .001$); the mean coronary calcium progression was 16.1% ± 22% in Group 1, compared with 39.7% ± 46% in Group 2 ($P < .001$). There was no influence of smoking, hypertension, diabetes, or patient age on the rate of progression, possibly because of the small size of the respective subgroups. Although the use of cholesterol-lowering medication by itself had

---

The data suggest that a limitation in the progression of aortic valve calcification, and hopefully in aortic stenosis, may be a secondary outcome of treatment of hyperlipidemia.

---

no significant influence on the progression of aortic valve calcification, when patients treated with statins were divided according to their LDL levels a statistically significant difference of annualized aortic valve calcium progression was found.

The authors note a number of limitations to their study. First, it was a retrospective analysis of patients referred for coronary calcification scanning and therefore subject to selection bias, because patients with cardiovascular risk factors tend to be over-represented. Second, the sample size was small. Most importantly, they assessed only aortic valve calcification, with no measure of the functional status of the valve. Thus an increase in calcification may not have been associated with an increase in severity of stenosis. Despite the limitations, a commonality in response of coronary calcification and aortic valve calcification to LDL levels reinforces the concept of the common pathologic mechanism of these two disorders. The data do suggest that a limitation in the progression of aortic valve calcification, and hopefully in aortic stenosis, may be a secondary outcome of treatment of hyperlipidemia. Further studies are required that relate these findings to the more important question of progression of stenosis and clinical outcomes. ■

Hypertension

Risk Associated with "Normal" Blood Pressure

Reviewed by Norman E. Lepor, MD, FACC, FAHA
Cedars-Sinai Medical Center, Los Angeles, CA

Impact of High-Normal Blood Pressure on the Risk of Cardiovascular Disease

Vasan RS, Larson MG, Liep EP et al.
N Engl J Med. 2001:345:1291–1297.

It has been accepted dogma that increasing systolic and diastolic blood pressure remains a continuum of risk–the higher the measured pressure, the higher the risk of cardiovascular (CV) events. There is very little data documenting the absolute risk differential between patients in the nonhypertensive categories as defined by the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC- VI) and World Health Organization–International Society of Hypertension (WHO-ISH) criteria:

- Optimal blood pressure (OBP): systolic < 120 mm Hg, diastolic < 80 mm Hg
- Normal blood pressure (NBP): systolic between 120 and 129 mm Hg or diastolic between 80 and 84 mm Hg
- High normal blood pressure (HNBP): systolic pressure between 130 and 139 mm Hg or diastolic between 85 and 90 mm Hg

The authors of this study have used that segment of the Framingham Heart Study population with no evidence of heart disease (n = 6859) as the population studied.

Blood pressure increases that occur within the "normal range" are associated with increase CV events.

In terms of the characteristics of the study subjects, women were more likely to have optimal blood pressure than men. Subjects with HNBP were more likely to be older, heavier, and have higher levels of cholesterol than those with OBP. About one third of the study population were smokers and about 2% were diabetic.