- of C1 esterase inhibitor activity by heparin. *Clin Exp Immunol* 1976;23:264-71.
- 97 Packham MA, Cazenave JP, Kinlough-Rathbone RL, Mustard JF. Drug effects on platelet adhesion to collagen and damaged vascular walls. *Adv Exp Med Biol* 1978;109:253-76.
 - 98 Wallis J, Moses JW, Borer JS, Weksler BB, Goldberg HL, Fisher J, et al. Coronary blood flow in coronary artery disease. Heparin-induced potentiation caused by prostacyclin release. *Circulation* 1982;66 (suppl II):263.
 - 99 Vinazeer H, Sternberger A, Haas S, Blumel E. Influence of heparin, of different heparin fractions, and of low molecular weight heparin-like substances on the mechanism of fibrinolysis. *Thromb Res* 1982;27:341-51.
 - 100 Boyle E, Bragdon JH, Brown RK. Role of heparin in vitro production of alpha-1-lipoprotein in human plasma. *Proc Soc Exp Biol Med* 1952;81:475-77.
 - 101 Graham DM, Lyon TP, Gofman JW, Jones HB, Yankley A, Simonton J, White S. Blood lipids and atherosclerosis. II: the influence of heparin upon lipoprotein metabolism. *Circulation* 1951;4:666-73.
 - 102 Jaques LB. Drug prophylaxis in atherosclerosis. *Artery* 1987;14:209-15.
 - 103 Engelberg H, Kuhn R, Steinman M. A controlled study of the effect of intermittent heparin therapy on the course of human coronary atherosclerosis. *Circulation* 1956;13:489-98.
 - 104 Bottiger LE, Carlson LA, Engstedt L, Oro L. Long-term heparin treatment in ischaemic heart disease: effects on clinical condition and plasma lipids. *Acta Med Scand* 1967;182:245-57.
 - 105 Gertler MM, Leetma H, Koutrouby RJ, et al. Long-term use of mini-dose heparin in post-myocardial infarction. *Adv Exp Med Biol* 1975;52:341-50.
 - 106 Neri Serneri GG, Rovelli F, Gensini GF, Pirelli S, Carnovalli M, Fortini A. Effectiveness of low-dose heparin in prevention of myocardial infarction. *Lancet* 1987;1:937-42.
 - 107 Hellgren M, Hagnevik K, Blomback M. Heparin aerosol: effect on blood coagulation and pulmonary function. *Thromb Res* 1981;21:493-502.
 - 108 Borchgrevink CF. Long-term anticoagulant therapy in angina pectoris. A follow up study. *Lancet* 1962;1:449-51.
 - 109 Second Report of the Sixty-Plus Reinfarction Study Research Group. Risks of long-term oral anticoagulant therapy in elderly patients after myocardial infarction. *Lancet* 1982;1:64-8.
 - 110 Smith P, Arnesen H, Holme I. The effect of warfarin on mortality and reinfarction after myocardial infarction. *N Engl J Med* 1990;323:147-52.
 - 111 Meade TW. Low-dose warfarin and low-dose aspirin in the primary prevention of ischaemic heart disease. *Am J Cardiol* 1990;65(Suppl C):7-11.

ABSTRACTS IN CARDIOLOGY

Do coronary angiograms reflect progression of atherosclerosis?

Are angiographic studies and event counts equally valid in assessing progression of coronary atherosclerosis? The abstract below shows that the first is an independent predictor of the second. Both methods may be

measuring a common mechanism for progression and event production, and the most likely contender is thrombosis on plaques.

MJ DAVIES

Coronary disease progression predicts subsequent cardiac events

David Waters, Tim Craven, Jacques Lesperance

Abstract

Progression of coronary disease is frequently used as an endpoint in clinical trials, yet its prognostic significance has not been well documented. Among 383 patients enrolled in such a trial, 335 underwent repeat coronary arteriography at 2 years. At study entry all patients were ≤ 65 years of age, had at least 4 stenoses $\leq 75\%$ and had an ejection fraction ≥ 0.40 . Coronary lesions were measured quantitatively using the system of Reiber et al. Progression, defined as an increase in diameter stenosis $\geq 15\%$, occurred in 141 patients (42%). During a subsequent follow up of 44 ± 10 months, 107 patients had one or more cardiac events: cardiac death (19), MI (36), or revascularisation (85). The relative risk (95% confidence intervals) of patients with progression, compared with those without, was 7.34 (2.18-24.7) for cardiac death, 2.29 (1.26-4.19) for cardiac death or

MI, 1.41 (0.98-2.03) for revascularisation, and 1.69 (1.24-2.31) for any event. The results were similar if progression was defined as a decrease in minimum diameter ≥ 0.4 mm. The relative importance of clinical and angiographic variables was assessed using a stepwise multivariable Cox regression model of time to event. Independent predictors were ejection fraction ($p = 0.002$), progression ($p = 0.003$), HDL cholesterol ($p = 0.005$), and hypertension ($p = 0.013$) for cardiac death; ejection fraction ($p = 0.001$) and progression ($p = 0.002$) for cardiac death or MI; and progression ($p = 0.04$), angina ($p = 0.05$), ejection fraction ($p = 0.05$), and HDL cholesterol ($p = 0.05$) for any clinical event.

Conclusion—Coronary progression is a powerful, independent predictor of subsequent coronary events and its use as a surrogate endpoint in clinical trials is justified. (*Circulation* 1992;86:I-159A.)