Influence of plaque morphology on the mechanism of luminal enlargement after directional coronary atherectomy and balloon angioplasty

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Abstract

Objective—To relate the mechanism of luminal gain after directional atherectomy and balloon angioplasty to the morphological characteristics of the coronary lesions, assessed by intravascular ultrasound imaging.

Design—Intravascular ultrasound imaging was performed before and after the revascularisation procedure to assess the contribution of wall stretching and plaque reduction in luminal gain.

Subjects—32 patients undergoing balloon angioplasty and 29 undergoing directional coronary atherectomy.

Main results—The main luminal area in vessels treated by balloon angioplasty increased from 1.51 (SD 0.30) to 3.91 (1.09) mm² ($P < 0.0001$) with a concomitant increase in total vessel area from 11.44 (2.73) to 13.07 (2.83) mm² ($P < 0.0001$). Therefore stretching of the vessel wall accounted for 68% of the luminal gain while plaque reduction accounted for the remaining 32%. This mechanism ranged from 45% in non-calcific plaques to 81% in echogenic plaques. The main luminal area in vessels treated by directional atherectomy increased from 1.49 (0.32) to 4.68 (1.73) mm² ($P < 0.0001$), with a concomitant increase of total vessel area from 13.61 (4.67) to 15.2 (4.04) mm² ($P = 0.006$). Thus stretching of the vessel wall accounted for 49% of the luminal area gain and plaque reduction for the remaining 51%. The presence of calcium influenced the relative contribution of these two mechanisms to the final luminal gain after directional atherectomy, since in calcific plaques stretching of the vessel wall accounted for only 9% of the luminal gain as compared to 56% in non-calcific plaques. After balloon angioplasty there was greater evidence of coronary dissections (32% *v* 3% after directional atherectomy, $P < 0.01$) and plaque fissure (60% *v* 0%, $P < 0.01$). Plaque fissure was more frequently seen in echolucent and concentric lesions, whereas dissections prevailed in echogenic and eccentric lesions.

Conclusions—Intravascular ultrasound imaging may allow the assessment of acute changes in lumen and vessel wall after revascularisation procedures, and help in evaluating the potential effect of

the structure and morphology of coronary lesions on the mechanism of luminal enlargement.

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Keywords: coronary atherectomy; balloon angioplasty; plaque morphology; intravascular ultrasound

Knowledge of the exact mechanism of action of balloon coronary angioplasty and directional coronary atherectomy in different kinds of atherosclerotic plaques is based on animal studies or pathological findings,¹⁻⁵ but few data exist in living humans. Intravascular ultrasound is the only method that offers the possibility of visualising the components and structure of atherosclerotic lesions in vivo and evaluating the effects of interventional procedures.⁶⁻¹¹

Most of the available data relating plaque structure to the final results of revascularisation procedures are derived from intravascular ultrasound studies performed only after revascularisation.¹²⁻¹⁴ However, it would be desirable to know about the plaque morphology before the procedure in order to assess the acute changes induced by different interventions. Recent investigations aimed at evaluating the mechanism of lumen enlargement achieved by directional atherectomy as compared to coronary angioplasty have given conflicting results,^{15,16} since the relative contribution of vessel stretching and plaque removal differs in the various studies. It is likely that these discrepancies are dependent on the different morphology of the atherosclerotic plaques of the patients included in those studies. To test this hypothesis we undertook a study to relate the mechanism of luminal enlargement achieved by directional atherectomy and coronary angioplasty to the morphological characteristics of the coronary lesions.

Methods

STUDY POPULATION

The study was carried out in 61 patients: 56 men and five women, with a mean age of 44 (SD 12) years, scheduled for elective coronary angioplasty (32 patients) or directional atherectomy (29 patients). The indication for coronary revascularisation was stable angina in 33 patients and unstable angina in 28

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patients. Arteries studied were the left anterior descending coronary artery ($n = 44$), the right coronary artery ($n = 8$), the left circumflex coronary artery ($n = 8$), and a saphenous vein graft in one patient. The procedure was selected only on the basis of angiographic findings: proximal short lesions also including branch vessels in non-tortuous segments were treated with directional atherectomy, whereas coronary angioplasty was used for all other types of lesion in the proximal or mid-part of the arteries. Exclusion criteria were limited to total occlusions in which predilatation morphometric analysis with intravascular ultrasound was not feasible.

Informed consent for the studies was obtained from all patients.

PROCEDURE

Before the procedure, intravenous heparin (10 000 units) and aspirin (500 mg) were given. Selective coronary angiography in multiple projections was performed before and after the revascularisation procedure. A variety of commercially available over-the-wire catheters was used to perform coronary angioplasty, and the Simpson Atherocath was used for coronary atherectomy. In three atherectomy procedures, the stenosis was predilated with an undersized standard 2 mm balloon. The number of balloon inflations and the pressure exerted were determined routinely as necessary to achieve an optimal angiographic result. Intravascular ultrasound examination was performed before and after the procedures in all patients. The ultrasound device used was a 25 MHz 3.9 F (Intertherapy). The imaging catheter consists of a single ultrasound transducer on the distal end of a flexible motor driven shaft with a mirror that reflects the ultrasound beam perpendicular to the long axis of the probe. The catheter is connected to a motor driven unit which rotates at 1800 rpm to provide real time cross sectional images. The ultrasound subassembly was inserted through a plastic introducing sheath to protect the arterial lumen from

injury during movement of the catheter. The introducing sheath was placed over a 0.014 inch guide wire and was advanced in the coronary artery distal to the treated area under fluoroscopic visualisation. The ultrasound transducer was then moved slowly in a retrograde direction through the guiding sheath; the fluoroscopic picture which displayed the position of the ultrasound catheter was shown on the same video screen as the ultrasound images to ensure correlation between the cross sectional ultrasound images and the position on the angiogram along the length of the artery. Warm saline was injected intermittently by hand to dislodge any small air bubbles adhering to the transducer. After completion of the preintervention imaging, the intravascular ultrasound catheter was removed with the guide wire left in place. At the end of the dilatation procedure the angioplasty or the atherectomy catheter was exchanged for the intravascular ultrasound catheter and imaging of the treated site was repeated.

IMAGE ANALYSIS

The images were recalled from the ultrasound computer disk and evaluated by two observers.

The following dimensions were calculated: (1) Total vessel area confined within the internal elastic membrane was measured to assess the extent by which stretching to the vessel wall contributed to enhanced lumen area after the procedure. Vessel stretch at the lesion site was defined as the percentage increase in postintervention total area as compared to the preintervention area.

(2) Luminal cross sectional area was measured before and after the revascularisation procedure, and injection of iodinated contrast medium was performed to confirm definition of the vessel wall. Luminal area gain was calculated as luminal area postprocedure minus luminal area preprocedure.

(3) Plaque cross sectional area was defined as the difference between the total vessel area and the luminal area. Plaque reduction was calculated as plaque area postprocedure minus plaque area preprocedure.

To evaluate whether the outcome of interventional procedures was influenced by plaque morphology, the gain in luminal area, plaque area, and total vessel area was calculated in each type of plaque.

ULTRASOUND MORPHOLOGY

Qualitative analysis included determination of plaque composition, plaque eccentricity, and presence of calcium (fig 1). Intravascular ultrasound images recorded after revascularisation were also analysed for plaque rupture and dissections. According to plaque composition, plaques were defined as echolucent when echo signals were weaker than those of the reference adventitia; plaques of this type have a high content of lipid, thrombus, loose connective tissue, or intimal hyperplasia. Echogenic plaques had brighter echo signals than those of the reference adventitia; this

Figure 1 Representative intravascular ultrasound image from a patient undergoing coronary angioplasty

Table 1 Plaque morphology by intravascular ultrasound in 29 patients treated by directional atherectomy (DCA) and 32 treated by balloon angioplasty (PTCA).

	DCA	PTCA
Echolucent	18 (62%)	14 (44%)
Echogenic	11 (38%)	18 (56%)
Eccentric	18 (62%)	16 (50%)
Concentric	11 (38%)	16 (50%)
Calcific	13 (45%)	24 (75%)
Non-calcific	16 (55%)	8 (25%)

type of plaque has been shown to contain dense fibrous tissue.

To determine plaque eccentricity, an eccentricity index (thinnest dimension of the plaque divided by the width of the opposite wall) was used. When the index was below 0.5 the plaque was considered eccentric, whereas the plaque was considered concentric if the index was above 0.5. Calcium was identified as bright dense echoes, with corresponding shadow in the far field. A plaque fissure was defined as an irregular thin echolucent separation extending from the lumen for a variable length into the plaque; a dissection between media and atheroma was defined as an echo-free space behind the atherosclerotic plaque of more than 0.3 mm thickness, or if movement of the plaque was seen as the plaque wavered in the flow of blood behind it. Equivocal dissections were confirmed by hand injection of contrast medium during ultrasound imaging.

STATISTICAL ANALYSIS

Results are given as mean (SD). A Student's *t* test for paired and unpaired data or χ^2 test were used when appropriate. *P* values less than 0.05 are considered to be statistically significant.

Results

PROCEDURAL ANGIOGRAPHIC RESULTS

Coronary angioplasty was successful in 30/32 patients (94%) whereas in two patients a Palmaz-Schatz stent had to be implanted because of major dissections. Directional atherectomy was successful in all 29 patients.

INTRAVASCULAR ULTRASOUND RESULTS

The morphological characteristics observed by intravascular ultrasound are shown in table 1.

The mean luminal area in vessels treated by coronary angioplasty increased from 1.51 (0.30) to 3.91 (1.09) mm² (*P* < 0.0001). Total vessel area increased from 11.43 (2.73) to 13.07 (2.83) mm² (*P* < 0.0001) and plaque area decreased from 9.85 (2.61) to 9.15 (2.13) mm² (*P* = 0.009). Therefore stretching of the vessel wall accounted for 68% of the luminal gain, while plaque reduction accounted for the remaining 32%. The mean luminal area in vessels treated by directional atherectomy increased from 1.49 (0.32) to 4.68 (1.73) mm² (*P* < 0.0001) with a concomitant increase of total vessel area from 13.61 (4.67) to 15.2 (4.04) mm² (*P* = 0.006) and a reduction of plaque area from 12.64 (4.43) to 10.61 (3.48) mm² (*P* < 0.0001). Therefore stretching of the vessel wall accounted for 49% of luminal area gain and plaque reduction for the remaining 51%.

As shown in fig 2, the luminal area gain achieved by directional atherectomy in all groups of plaques was higher than the gain achieved by coronary angioplasty; however, the only significant difference was found in non-calcific plaques treated with directional atherectomy as compared to those treated with coronary angioplasty: 3.70 (2.02) *v* 2.45 (1.17) mm², *P* = 0.007.

MECHANISM OF CORONARY ENLARGEMENT AFTER DIRECTIONAL ATHERECTOMY IN RELATION TO DIFFERENT PLAQUE CHARACTERISTICS

Vessel expansion accounted for only 32% of luminal gain in echogenic plaques, for 38% in both concentric and eccentric plaques, and for 39% in echolucent plaques. Plaque reduction was the predominant mechanism to achieve optimal luminal area gain irrespective of plaque composition or plaque eccentricity. However, in calcific plaques, stretching of the vessel wall accounted for only 9% of the luminal gain as compared to 56% in non-calcific plaques (table 2). In particular, in none of the patients with echogenic and calcific plaques was the postatherectomy vessel area greater than the vessel area measured before atherectomy. Conversely, in patients with echogenic plaques without evidence of calcium, stretching accounted for 90% of luminal gain, with only 10% due to plaque area reduction (fig 3).

Figure 2 Gain of luminal area as assessed by intravascular ultrasound in relation to different plaque morphologies in patients undergoing directional coronary atherectomy (DCA) and percutaneous transluminal coronary angioplasty (PTCA). *P* = 0.007

Table 2 Mechanism of lumen enlargement after directional atherectomy in relation to different plaque characteristics. Values are means (SD).

	Luminal area gain (mm ²)	Total area gain (mm ²)	Plaque area reduction (mm ²)
Echolucent	3.44 (2.13)	1.36 (1.95) [39%]	-2.14 (2.20) [61%]
Echogenic	2.69 (0.83)	0.86 (2.32) [32%]	-1.83 (2.27) [68%]
Eccentric	3.62 (1.99)	1.37 (2.50) [38%]	-2.43 (2.33) [62%]
Concentric	2.36 (1.03)	0.90 (1.28) [38%]	-1.38 (1.88) [62%]
Calcific	2.56 (1.31)	0.22 (1.60)* [9%]	-2.33 (2.36) [91%]
Non-calcific	3.70 (2.02)	2.06 (2.11)* [56%]	-1.72 (2.05) [44%]

*P = 0.002.

Figure 3 Gain of luminal area, and total vessel area and plaque area reduction, as assessed by intravascular ultrasound, in echogenic and calcific plaques compared with echogenic and non-calcific plaques in patients undergoing directional coronary atherectomy. P < 0.001

Table 3 Mechanism of lumen enlargement after balloon angioplasty in relation to different plaque characteristics. Values are means (SD)

	Luminal area gain (mm ²)	Total area gain (mm ²)	Plaque area reduction (mm ²)
Echolucent	2.73 (1.25)	1.50 (1.60) [55%]	-0.97 (1.30) [45%]
Echogenic	2.15 (1.03)	1.75 (1.28) [81%]	-0.29 (1.15) [19%]
Eccentric	2.41 (1.24)	1.82 (1.50) [75%]	-0.40 (1.57) [25%]
Concentric	2.39 (1.04)	1.34 (1.25) [56%]	-0.89 (1.35) [44%]
Calcific	2.45 (1.17)	1.84 (1.47) [75%]	-0.36 (1.59) [25%]
Non-calcific	2.27 (1.18)	1.03 (1.08) [45%]	-1.24 (0.92) [55%]

Figure 4 Relative contribution of plaque fissure, dissection, and stretching alone as assessed by intravascular ultrasound in relation to different plaque morphologies in patients undergoing coronary angioplasty.

In no case was plaque fissure observed after directional atherectomy, whereas dissections were seen in only one case.

MECHANISM OF CORONARY ENLARGEMENT AFTER CORONARY ANGIOPLASTY IN RELATION TO DIFFERENT PLAQUE CHARACTERISTICS

Vessel expansion was the most relevant mechanism for improvement in luminal area after coronary angioplasty, ranging from 45% in non-calcific plaques to 81% in echogenic plaques (table 3). Plaque fissure was seen in 19 cases, prevailing in echolucent and concentric lesions, whereas dissection was documented in 10 plaques and prevailed in echogenic and eccentric lesions. The incidence of coronary dissection, plaque fissure, or both after coronary angioplasty according to the different plaque characteristics is shown in fig 4. As compared to directional atherectomy, the only significant difference in vessel expansion was seen in calcific lesions in which stretching of the vessel wall accounted for 75% of luminal enlargement in the coronary angioplasty group, in contrast to 9% in the directional atherectomy group (P = 0.002). After coronary angioplasty there was also greater evidence of dissections (coronary angioplasty 32%, directional atherectomy 3%; P < 0.01).

Discussion

It has been documented that catheter based two dimensional intracoronary ultrasound imaging provides accurate dimensional information on the arterial lumen as well on the detailed morphological appearances of the arterial wall.⁶⁻⁸ Moreover, intravascular ultrasound imaging may allow in vivo assessment of the mechanism of coronary interventions in humans.¹⁰⁻¹³ Results of several recent studies have shown an excellent correlation between intravascular ultrasound images and histological samples with respect to morphological and

dimensional measurements.^{7,8} However, few studies have defined the plaque morphology before coronary interventions; therefore scant information exists about how baseline plaque characteristics may influence the outcome of different revascularisation procedures.^{17,18} In this study we were able to obtain intravascular ultrasound images before coronary interventions; therefore we could evaluate the potential effect of the composition and eccentricity of the lesions on the mechanism of luminal enlargement. Moreover we assessed the acute changes to the lumen and vessel wall produced by two different revascularisation procedures and defined the mechanism of lumen enlargement. Since the luminal area gain was calculated as the difference between postprocedure and preprocedure luminal areas, it could be objected that measurements of lumen dimensions before percutaneous interventions could have been affected by wedging of the ultrasound catheter into the stenotic lumen, completely abolishing the flow before coronary angioplasty and directional atherectomy. However, since the preintervention lumen dimensions clustered around the size of the catheter in nearly all patients, luminal area gain measurements were substantially dependent on postprocedure luminal area and were not affected by preprocedure dimensions.

LUMEN ENLARGEMENT ACHIEVED BY DIRECTIONAL ATHERECTOMY

Previous angiographic studies¹⁹ have suggested that the amount of tissue retrieved by directional atherectomy is not enough to account for the resultant increase in luminal diameter and that the Dotter effect of the catheter may also contribute to lumen enlargement. By using intravascular ultrasound imaging, Suneja *et al.*¹⁶ recently showed that vessel expansion resulting in increased total vessel area tended to be greater in patients treated by directional atherectomy than in those treated by coronary angioplasty. Our data show that plaque reduction accounts for 51% of the luminal gain after directional atherectomy, while stretching of the vessel wall accounted for the remaining 49%. However, the contribution of vessel expansion to lumen enlargement was highly variable, ranging from 56% in non-calcific plaques to 9% in calcific plaques. Our results differ from those reported by Tenaglia *et al.*,²⁰ who did not find any significant difference in internal elastic lamina area between the treated and the reference sites, implying that vessel stretching did not occur. Our data are also at variance with those reported by Braden *et al.*,¹⁵ who found that only 22% of the luminal gain obtained by coronary atherectomy was due to vessel stretching and that plaque reduction was responsible for 78% of the luminal enlargement. Tenaglia *et al.*²⁰ also pointed out that plaque removal by coronary atherectomy was less when echogenic plaques rather than echolucent plaques were present, whereas Braden *et al.*¹⁵ found that plaque reduction was more frequently associated with lumen

enlargement after coronary atherectomy in eccentric than in concentric lesions. In our study plaque removal by directional atherectomy was significantly less when non-calcific plaques rather than calcific plaques were present.

LUMEN ENLARGEMENT ACHIEVED BY CORONARY ANGIOPLASTY

The mechanism of lumen enlargement after coronary angioplasty remains controversial. A combination of vessel expansion, plaque fissure, and dissection produces the improvement in lumen dimension after coronary angioplasty.^{1-5,21} However, little information in living humans exists. In our study we found that the enlargement produced by coronary angioplasty was associated with a 68% increase in total vessel area. These data are similar to those reported by Tenaglia *et al.*²⁰ and by Braden *et al.*¹⁵ These investigators showed that the gain in luminal area was primarily associated with stretching of the vessel wall. At variance with those studies, we also analysed the presence of plaque fissure and dissections: our data show that only in 12% of echolucent concentric and non-calcific lesions, and in 6% of echogenic eccentric and calcific lesions, was vessel stretching the sole mechanism of lumen enlargement. In the majority of cases a variable percentage of plaque fissure and dissection was seen. In particular, we found that dissection was higher in eccentric and in echogenic plaques than in concentric and echolucent plaques. These data are similar to those reported by Fitzgerald *et al.*²²: in 41 patients studied, 31 (76%) had ultrasound evidence of dissection or plaque fissure after balloon dilatation.

CONCLUSIONS

Our data show that lumen enlargement after coronary angioplasty is achieved predominantly by vessel stretching. A variable percentage of plaque fissure and dissection was also seen. Plaque fissure was more frequently seen in echolucent and concentric lesions, whereas dissections prevailed in echogenic and eccentric lesions. On the other hand, plaque rupture and dissections were uncommon in vessels treated with directional atherectomy. After this procedure the improvement in lumen enlargement was due to both plaque reduction and vessel stretching and was influenced by plaque composition and particularly by the presence of calcium. The results of this study show that intravascular ultrasound imaging may allow us to assess acute changes of lumen and vessel wall after revascularisation procedures, as well as to evaluate the potential effect of the structure and morphology of coronary lesions on the mechanism of luminal enlargement.

- 1 Waller BF. Early and late morphologic changes in human coronary arteries after percutaneous transluminal coronary angioplasty. *Clin Cardiol* 1983;6:363-72.
- 2 Soward AL, Essed C, Serruys P. Coronary arterial findings after accidental death immediately after a successful percutaneous transluminal coronary angioplasty. *Am J Cardiol* 1985;56:794-5.
- 3 Potkin B, Roberts W. Effects of percutaneous transluminal

- coronary angioplasty on atherosclerotic plaques and relation of plaque composition and arterial size to outcome. *Am J Cardiol* 1988;62:41-50.
- 4 Faxon D, Weber V, Handenschild C, Gottsman SB, McGovern WA, Ryan TJ. Acute effects of transluminal angioplasty of normal canine coronary arteries. *Arteriosclerosis* 1982;2:125-33.
 - 5 Block P, Baughman K, Pasternak RC, Fallon JT. Transluminal angioplasty: correlation of morphologic and angiographic findings in experimental model. *Circulation* 1980;61:778-85.
 - 6 Nissen SE, Gurley JC, Grines CL, Booth DC, McKlure R, Berk M, *et al.* Intravascular ultrasound assessment of lumen size and wall morphology in normal subjects and patients with coronary artery disease. *Circulation* 1991;84:1087-99.
 - 7 Tobis JM, Mallery J, Mahon D, Lehmann K, Zalesky P, Griffith J, *et al.* Intravascular ultrasound imaging of human coronary arteries in vivo. Analysis of tissue characterization with comparison to in vitro histologic specimens. *Circulation* 1991;83:913-26.
 - 8 Potkin BN, Bartorelli A, Gessert JM, Neville RF, Almagor Y, Roberts W, *et al.* Coronary artery imaging with high-frequency ultrasound. *Circulation* 1990;81:1575-85.
 - 9 Yock PG, Fitzgerald PJ, Linker DT, Angelsen B. Intravascular ultrasound guidance for catheter-based coronary interventions. *J Am Coll Cardiol* 1991;19:39-45B.
 - 10 Honye J, Mahon DJ, Jain A, White C, Ramee S, Wallis J, *et al.* Morphological effects of coronary balloon angioplasty in vivo assessed by intravascular ultrasound imaging. *Circulation* 1992;85:1012-25.
 - 11 Potkin BN, Keren G, Mintz GS, *et al.* Arterial responses to balloon coronary angioplasty: an intravascular ultrasound study. *J Am Coll Cardiol* 1992;20:942-5.
 - 12 Keren G, Pichard AD, Kent K, Satler L, Leon M. Failure or success of complex catheter-based interventional procedures assessed by intravascular ultrasound. *Am Heart J* 1992;123:200-7.
 - 13 Isner J, Rosenfield K, Losordo DW, Rose L, Langevin RE, Razvi S, *et al.* Combination balloon-ultrasound imaging catheter for percutaneous transluminal angioplasty. Validation of imaging, analysis of recoil, and identification of plaque fracture. *Circulation* 1991;84:739-54.
 - 14 Tenaglia AN, Buller CE, Kisslo KB, Phillips HR, Stock RS, Davidson C. Intracoronary ultrasound predictors of adverse outcome after coronary artery interventions. *J Am Coll Cardiol* 1992;20:1385-90.
 - 15 Braden G, Herrington D, Downes T, Kutcher M, Little W. Qualitative and quantitative contrasts in the mechanism of lumen enlargement by coronary balloon angioplasty and directional coronary atherectomy. *J Am Coll Cardiol* 1994;23:40-8.
 - 16 Suneja R, Nair R, Reddy K, Raseed Q, Sheehan H, Hodgson J. Mechanism of angiographically successful directional coronary atherectomy. Evaluation by intracoronary ultrasound and comparison with transluminal coronary angioplasty. *Am Heart J* 1993;126:507-13.
 - 17 Macaya AF, Goicolea J, Alfonso F, Iñiguez A, Hernandez R, *et al.* Angiographic changes induced by intracoronary ultrasound imaging before and after coronary angioplasty. *Am Heart J* 1993;125:877-80.
 - 18 Mintz G, Pichard A, Kovach J, Kent K, Satler L, Javier S, *et al.* Impact of preintervention intravascular ultrasound imaging on transcatheter treatment strategies in coronary artery disease. *Am J Cardiol* 1994;73:423-30.
 - 19 Safian RD, Gelbfish JS, Erny RE, Schnill SJ, Schmidt DA, Baim DS. Coronary atherectomy: clinical, angiographic and histologic findings and observations regarding potential mechanism. *Circulation* 1990;82:69-79.
 - 20 Tenaglia A, Buller C, Kisslo K, Stack R, Davidson C. Mechanism of balloon angioplasty and directional atherectomy as assessed by intracoronary ultrasound. *J Am Coll Cardiol* 1992;20:685-91.
 - 21 Waller BF. "Crackers, breakers, stretchers, drillers, scrapers, shavers, burners, welders, and melters": the future treatment of atherosclerotic coronary artery disease? A clinical morphological assessment. *J Am Coll Cardiol* 1989;13:969-87.
 - 22 Fitzgerald PJ, Ports TA, Yock PG. Contribution of localized calcium deposits to dissection after angioplasty: an observational study using intravascular ultrasound. *Circulation* 1992;86:64-70.