PTCA of chronically occluded coronary arteries

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The progression of a coronary artery stenosis to total occlusion does not necessarily imply complete myocardial necrosis of the entire flow-dependent region of the vessel involved.[1] Various degrees of perfusion can be maintained by a collateral circulation, so that the area of necrosis can be limited in size or infarction can even be prevented. During increased demand for myocardial oxygen the collateral blood flow may become insufficient, and symptoms of myocardial ischemia may arise. Signs of viable myocardial tissue are: angina pectoris in the absence of other significant coronary artery lesions, absence of Q waves on the electrocardiogram, the preservation of left ventricular wall motion as shown by angiographic or echocardiographic techniques, and redistribution on thallium 201 scintigraphy.[2,3] Angina pectoris (even unstable angina pectoris) may persist in spite of an optimal antiischemic drug regimen.

Percutaneous recanalization, followed by balloon dilatation, appears to be an attractive approach, since coronary bypass graft surgery, although a safe procedure, is now generally not considered to be the best treatment for single-vessel coronary artery disease. Percutaneous transluminal coronary angioplasty (PTCA) of chronically occluded coronary arteries, first described in 1982,[4,5] comprises approximately 10% of the total number of PTCA procedures.[2,6-9] This article reviews the factors that determine the initial success of angioplasty of chronic total coronary artery occlusions, as well as the clinical and angiographic follow-ups. A decision scheme for the approach to treatment of the patient with a chronic total occlusion is presented, and guidelines for the PTCA procedure are proposed. New techniques are described, which might be used if "older" techniques fail.

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PRIMARY SUCCESS OF PTCA OF CHRONICALLY OCCLUDED CORONARY ARTERIES

The primary success rate of PTCA of chronically occluded vessels is mainly determined by the following factors: (1) the presence of functional or total occlusion, (2) the duration of the occlusion, (3) the length of the occluded segment, (4) the presence of collateral circulation, (5) the presence of a vessel stump, (6) the underlying pathology, and (7) the availability of new or additional technical devices.

Functional or total occlusion. Traditionally, coronary artery occlusion has been classified as either functional occlusion or total occlusion. Functional occlusion is defined as antegrade, faint and late opacification of the vessel segment beyond the occlusion with or without visible continuity of the artery.[10] With the guide wire across the area of stenosis, complete occlusion commonly occurs. Total occlusion is defined as the absence of antegrade filling distal to the occlusion. In some cases, distal filling can be demonstrated through collaterals that originate proximally from the area of stenosis or from other vessels. Generally, the primary success rate of dilatation in the case of a functional occlusion is higher than that in the case of a total occlusion (Table I).[6,7,10-13]

Duration of the occlusion. The second most important determinant of primary success of PTCA of occluded vessels is the duration of the occlusion because the duration determines the consistency of the lesion. Occlusions of short duration often consist solely of fresh thrombus, whereas in later stages organization and calcification occur. It is often difficult to estimate the duration of the occlusion. The medical history (abrupt changes of symptoms), a sustained myocardial infarction, and the time of the diagnostic coronary angiogram are often helpful in making an approximation of the duration of the oc-

Table I. Functional and total occlusion and initial success of PTCA

	Total occlusion		*Functional occlusion*	
Authors (year)	*Patients*	*Success*	*Patients*	*Success*
Dervan et al (1983).[10]	4	3 (75%)	9	4 (44%)
Kereiakes et al. (1985)[6]	68	34 (50%)	8	6 (75%)
Geuskens et al. (1986)[7]	51	24 (47%)	29	22 (76%)
DiSciascio et al. (1986)[11]	23	13 (57%)	23	16 (70%)
Melchior et al. (1987)[12]	100	56 (56%)	—	— (—)
Safian et al. (1988)[13]	169	86 (51%)	102	81 (79%)
Totals	415	216 (52%)	171	129 (75%)

Table II. Duration occlusion and success of PTCA

Authors (year)	*Duration of occlusion*	*Patients*	*Success*
Holmes et al. (1984)[8]	≤12 weeks#	19	13 (68%)
	>12 weeks#	5	0 (0%)
Kereiakes et al. (1985)[6]	<20 weeks#	55	33 (67%)
	≥20 weeks#	17	3 (18%)
Geuskens et al. (1986)[7]	≤8 weeks*	18	16 (89%)
	>8 weeks*	11	6 (55%)
	≤8 weeks#	29	16 (55%)
	>8 weeks#	22	8 (36%)
DiSciascio et al. (1986)[11]	<2 weeks†	19	14 (74%)
	2-4 weeks†	9	6 (67%)
	>4 weeks†	18	9 (50%)
Melchior et al. (1987)[12]	<1 month#	52	36 (69%)
	1-6 months#	38	19 (50%)
	>6 months#	9	1 (11%)
Safian et al. (1988)[13]	<1 month†	60	40 (67%)
	1-6 months†	49	29 (59%)
	>6 months†	60	33 (55%)

#Total occlusion.
*Functional occlusion.
†Total and functional occlusions.

clusion. Table II shows studies that deal with the duration aspect.[6-8, 11-13] It may be concluded that the primary success rate is substantially lower if the duration of the total occlusion is estimated to be more than 2 months as compared to an estimated duration of less than 2 months. The same holds true for the functional occlusion.

Length of the occluded segment. The length of the occluded segment can sometimes be assessed because of filling of the distal part of the vessel by adequate collateral circulation. This permits estimation of the length of the "missing" segment. Kereiakes et al.[6] showed that the outcome of angioplasty was significantly influenced by the length of the nonvisualized segment. PTCA of occluded segments less than 1.5 cm in length was successful in 78% of the cases versus 42% in which the length of the occlusion was greater than 1.5 cm.

Collateral circulation. The collateral circulation provides useful information on the length of the occlusion, opacification of the vessel distal to the occlusion that delineates the course of the vessel beyond the lesion ("roadmapping"), and possible distal abnormalities.[9] Knowledge of the anatomy of the distal vessel may greatly facilitate crossing the area of stenosis with the wire, and it may reduce the risk of perforation of the vessel wall. The possible detrimental effects of collaterals on restenosis rate will also be discussed.

Other anatomic factors. The presence of a vessel stump beyond the last side branch (Fig. 1) may facilitate the procedure by giving direction to the guide wire and/or perfusion catheter. An occlusion in a straight vessel segment is another favorable factor. Great difficulty in crossing may arise if the occluded segment is located in a bend or if side branches arise from the most distal end of the stump.

Pathologic considerations. Although it may be unknown to the angiographer the underlying pathology of total occlusions may be different in individual cases (Fig. 2), and this may determine, to a great extent, the ultimate short- and long-term outcome[14]: the "ideal" occlusion consists of a small, fresh, central thrombus surrounded by a firmer fibrocellular narrowing that permits easy passage of the wire within the vessel lumen. On the other hand, the lesion may consist of a rupture of complex plaque with a fibrous cap overlying soft atheromatous tissue or may even consist of massive fibromuscular tissue. In these situations the "path of least resistance" may run in the subintima, and attempted recanalization may be hazardous because of the considerable risk of vessel wall perforation (Fig. 3).

Availability and use of new devices and techniques. For successful perforation of the occlusion an optimal "pushability" of the guide wire and a good and stable fit of the guiding catheter in the ostium of the

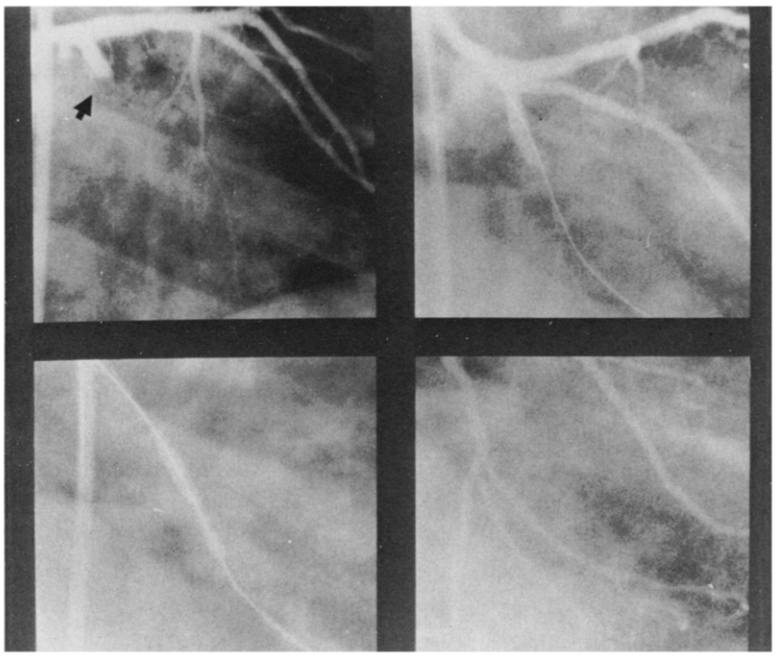

Fig. 1. Example of a vessel stump of the left circumflex coronary artery *(upper panel, left)*. After perforation of the occlusion with the guidewire, the balloon catheter can be advanced across the lesion *(upper panel, right)*; the lesion is dilated *(lower panel, left)* with good angiographic results *(lower panel, right)*.

coronary artery is even more important than with conventional lesions. If an approach with a standard soft-tipped guide wire fails, a stiffer guide wire may be useful. The guide wire should not be screwed into the occlusion because there is a risk of wire fracture, and it should be rotated no more than one revolution in either direction. Sometimes it is helpful to position a perfusion catheter in the vessel stump for better support of the guide wire.[15] Specially designed guidewire delivery system ("hollow wire") for this particular purpose have been described,[16] and information on more sophisticated systems, that employ tipped wires with high pushability ("magnum") or slow rotating wire systems will be available soon. After successful perforation, a dilatation with a small caliber low profile balloon catheter can be performed. Contrast media can be injected through the perfusion or the balloon catheter to visualize the vessel segment beyond the area of stenosis.

Newer techniques in cardiology, such as ultra-low profile balloon catheters, "balloon-on-a-wire" devices, direct laser energy,[17] laser balloons,[18] laser wires,[19] slow rotational angioplasty systems,[20] atherectomy catheters,[21] abrasive tips,[22, 23] and thrombolytic regimens may improve the initial results of angioplasty of occluded vessels. Since angiographic techniques provide only limited information on the structure and consistency of a coronary vessel occlusion, intravascular imaging devices such as angioscopy and ultrasound are needed to delineate the intraluminal pathway as a guide for the recanalization devices and to prevent a "fausse route."

Table III. Potential complications of PTCA of occluded vessels

-Perforation of the vessel wall
-Dissection of the dilated vessel
-Dissection of side branches proximal to the dilatation site by manipulations of the guidewire
-Acute reocclusion, probably caused by competitive antegrade and retrograde flow because of well-developed colaterals
-Occlusion of side branches beyond the lesion by the "snow plow" effect
-Dilatation of side branches or collateral vessels by a "fausse route" of the guidewire and the balloon catheter
-Guidewire fracture by entrapment in the occlusion
-Embolization of thrombi
-Myocardial infarction
-Arrhythmias

COMPLICATIONS OF PTCA OF TOTAL OCCLUSIONS

PTCA of a total occlusion is sometimes regarded as a low-risk procedure because preexisting 100% stenoses cannot become worse and because a well developed collateral circulation is present. The complications mentioned in Table III may also occur during angioplasty of conventional lesions, but some of them are likely to be more frequent in PTCA of total occlusions. A creatinine kinase elevation of more than

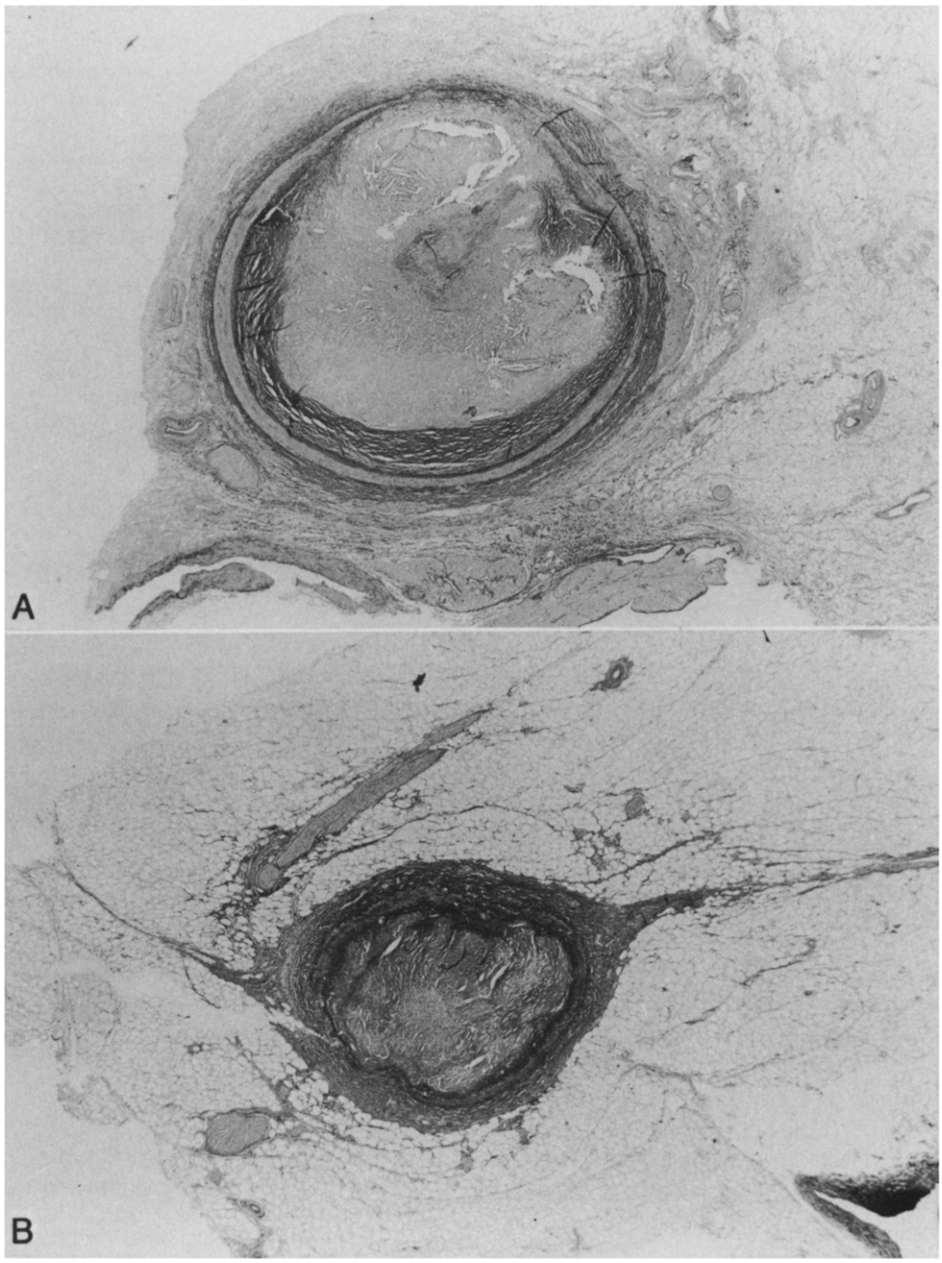

Fig. 2. Examples of the pathologic appearance of a chronic total coronary artery occlusion. **A**, The lumen is predominantly filled with atheroma and only marginal fibromuscular tissue. (H.A. stain). **B**, The lesion is mainly composed of fibromuscular tissue. (Elastic-van Gieson stain).

Table IV. Recurrence of angina pectoris after successful PTCA of occluded coronary arteries

Authors (year)	*Follow-up (months)*	*Patients*	*AP*
Kereiakes et al. (1985)[6]	7,3	40	10 (25%)
Safian et al. (1985)[24]	16	47	21 (45%)
Clark et al. (1985)[26]	≥6	45	20 (44%)
Geuskens et al. (1986)[7]	3-48	46	15 (33%)
DiSciascio et al. (1986)[11]	4-12	29	14 (48%)
Melchior et al. (1987)[12]	1-48	49	14 (29%)
Totals	1-48	256	94 (37%)

AP, Angina pectoris.

twice the normal value occurs in 5.3% to 16.3%,[2, 6-8] a definite myocardial infarction in 1.1% to 2.5%[7, 12, 13] occlusion of distal vessels or side branches in 2.2% to 5.3%,[6, 11] abrupt reocclusion in 5.3% to 7.7%,[6, 10, 24] emergency coronary artery bypass grafting in 1.3% to 4.3%,[2, 6-8, 13, 25] guide-wire fracture in 0.6%,[13] rupture of the coronary artery in 1% (12), and significant arrhythmias (ventricular tachycardias, ventricular fibrillation, and symptomatic bradycardias that necessitate temporary pacing) in 2% (12) of the cases.

Most complications can be explained by coronary

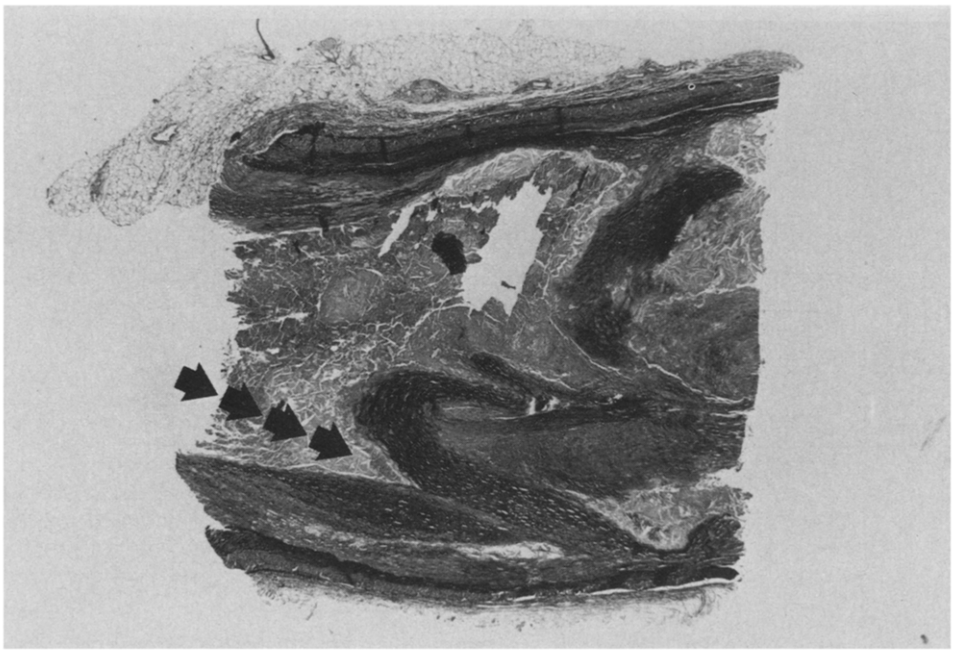

Fig. 3. Longitudinal histologic section through a totally occluded coronary artery showing atheroma and massive fibromuscular tissue; the arrows indicate a potential "fausse route" into the subintima if the lesion is negotiated by the guidewire in this direction (the filling defect is a fixation artefact.) (H.A. stain).

Table V. Restenosis after successful PTCA of occluded coronary arteries

Authors (year)	*Follow-up (months)*	*Definition*	*Patients*	*Restenosis*
Holmes et al. (1985)[8]	7	>40% increase	10	2 (20%)
Clark et al. (1985)[26]	≥6	?	45	25 (56%)
Libow et al. (1985)[27]	6	>50% loss	42	18 (43%)
Geuskens et al. (1986)[7]	3-48	reocclusion	32	9 (28%)
DiSciascio et al. (1986)[11]	4-12	?	29	14 (48%)
Melchior et al. (1987)[12]	1-48	≥60% DS	40	22 (55%)

Definition, Definition of restenosis; increase, increase in percent of diameter stenosis; loss, loss in percent of initial gain of PTCA; DS, diameter of stenosis.

thrombus formation and/or dissection and embolization. There is no convincing evidence that angioplasty of total occlusions is more hazardous than angioplasty of conventional lesions. To reduce the risk of abrupt reocclusion after the procedure, full heparinization during at least 24 hours after PTCA is advised. Careful follow-up is mandatory and in the case of signs of restenosis (symptoms and/or noninvasive tests) coronary angiography should be performed. Our current practice is to perform angiographic follow-up within 2 to 3 months, and if restenosis is present, regardless of symptoms, repeat angioplasty is attempted during the same session. However, the merits of this approach have never been established.

Follow-up. The recurrence of angina pectoris and angiographic restenosis after successful PTCA of chronically occluded coronary arteries is presented in Tables IV and V.* Angina pectoris recurs in 25% to 48% of the cases, which appears to be higher than after PTCA of conventional lesions. The reported incidence of restenosis depends on the completeness of the angiographic follow-up and also on the definition of restenosis. As there is considerable variation in the methodology adopted, the available studies are not very comparable. However, one may conclude that restenosis and reocclusion are more frequent findings after PTCA of occluded vessels than after PTCA of conventional stenoses. A study by Clark et al.[26] showed that restenosis was more frequent if lesions required more balloon inflations and higher inflation pressure. DiSciascio et al.[11] found that restenosis appeared to be related to a higher degree of post-PTCA residual narrowing. Although angio-

*References 6-8, 11, 12, 24, 26, 27.

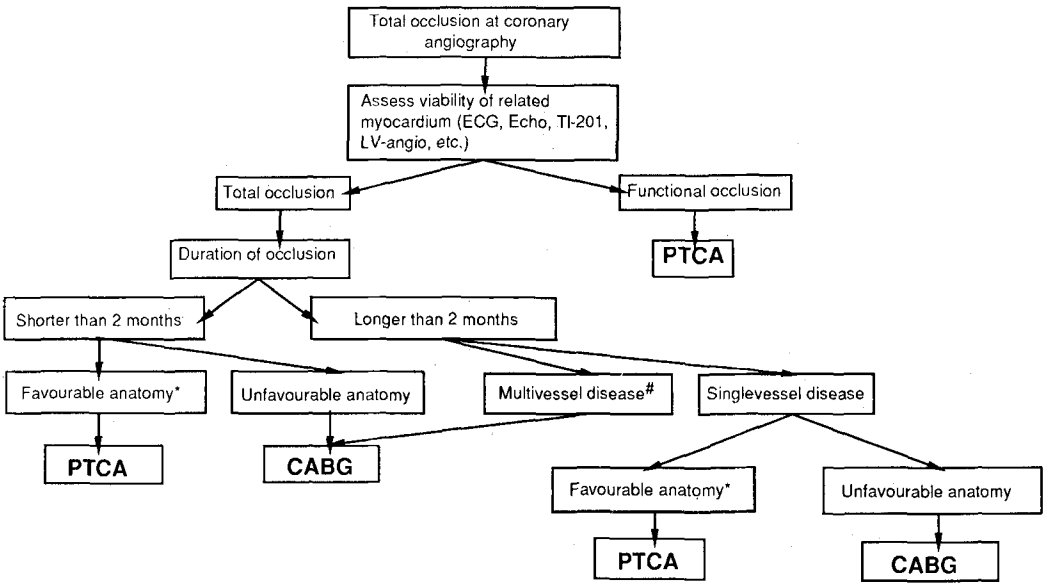

Fig. 4. Decision scheme for the approach to treatment of the patient with a chronic occluded coronary artery during coronary angiography. This scheme presumes anginal complaints in spite of optimal medical treatment. *Favorable anatomy consists of short length of occluded vessel segment, occlusion in a straight segment, good visualization of the segment beyond the occlusion by collaterals, the absence of stenoses distal to the occlusion, and the presence of a vessel stump. In particular in the presence of severe stenoses in a coronary artery that supply the myocardial region of the occluded vessel by collaterals (jeopardized collaterals). *ECG,* electrocardiography; *TI-201,* thallium 201 scintigraphy; *LV-angio,* left ventricular angiography.

graphically visible collaterals provide information on the course of the occluded vessel and thereby favorably influence the initial success rate of angioplasty, some authors have demonstrated that a well developed collateral circulation is associated with a higher restenosis rate.[10, 27] The degree of collateral supply is represented by the measured coronary wedge pressure, which is highest in the presence of chronic total occlusions.[28] Therefore patients with high coronary wedge pressures, which is especially the case in vessels with a chronic total occlusion and a visible collateral supply, are at increased risk for restenosis. This could be explained by a decrease of transstenotic gradient, which might cause accelerated progression of stenosis severity at the dilatation site.[29]

With a large series of patients, Ellis et al.[30] demonstrated that restenosis does not appear to reach the plateau phase at 6 months follow-up as is the case in PTCA of nonocclusive stenoses in which restenosis at the dilatation site occurs predominantly within the first 6 months after PTCA.[31] Furthermore, it was shown that drug therapy with aspirin, dipyridamole, or warfarin did not influence the long-term results. Whether restenosis and reocclusion can be reduced by additional techniques (e.g., laser balloon angioplasty or stenting) or alternative percutaneous techniques (e.g., high-speed rotational or direct laser angioplasty, whether or not in combination with pharmacologic regimens, remains to be established.

Conclusions. The initial success of PTCA of chronically occluded coronary arteries depends mainly on the duration of the occlusion and on several anatomic factors. The follow-up of these patients is characterized by a higher percentage of restenosis and recurrence of angina pectoris. Against this background, a strategy must be formulated for each patient with an occluded coronary artery and myocardial ischemia in the related flow region. In Fig. 4 a decision scheme for the approach to treatment of the patient with a chronic total occlusion is presented. This scheme presumes ischemia in the myocardial region supplied by the occluded vessel and angina pectoris in spite of optimal medical therapy. It is evident that no patient fits perfectly into such a scheme, which is no more than a basis for clinical management. Whether newer techniques in cardiology may improve the initial and long-term results of angioplasty of occluded vessels

remains to be established by carefully designed prospective studies with angiographic follow-up in all patients.

SUMMARY

The occlusion of a coronary artery does not necessarily imply the existence of nonviable myocardium of that flow-dependent region, because the presence of a well developed collateral circulation may be a sufficient nutrient source. During an episode of increased demand for myocardial oxygen, this collateral blood supply may become insufficient, and symptoms of myocardial ischemia may arise. PTCA of the occluded vessel appears to be an attractive approach to relieve ischemia in this situation. The primary success of dilatation of totally occluded segments depends largely on the duration of the occlusion but also on anatomic factors such as total or functional occlusion, the length of the occluded segment, and good angiographic visualization of the coronary artery distal to the occlusion by collaterals. The primary success rate (±60%) of PTCA of occluded vessels is lower than the success rate (>90%) of PTCA of nonocclusive stenoses. Also the restenosis rate (±40%) and subsequent recurrence rate of angina pectoris is higher, compared to the 30% restenosis rate after dilatation of conventional lesions. Newer percutaneous techniques such as lasers, newly designed guide wires, and intravascular imaging devices are necessary to increase the primary success rate. Whether these techniques will also improve the long-term results remains uncertain.

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Frequency and significance of chamber collapses during cardiac tamponade

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Diagnosis of a tamponading pericardial effusion is often elusive, partly because cardiac tamponade is a continuum. Even clinically non-tamponading effusions are physiologically active; noninvasive studies in patients with small to large clinically nontamponading amounts of pericardial fluid showed definite physiologic effects that could only be due to increased ventricular interaction via increased coupling of the heart and pericardium by the fluid.[1] Thus it is not surprising that the earliest description of chamber collapses by Shiina et al.[2] offered this finding as a sign of "impending" tamponade. Later, Engle et al.[3] distinguished non-tamponading from tamponading pericardial effusions by M-mode echocardiography, showing persistence of posterior motion of the right ventricular endocardium beyond 0.05 second after mitral valve opening. Subsequently, right ventricular and right atrial chamber collapses were identified as diagnostic correlates of cardiac tamponade, occurring individually or in combinations and even in low pressure tamponade.[4-7]

Left atrial collapse is relatively uncommon, reported by Fast et al.[6] in 5 of 39 patients, four of whom also had right atrial and ventricular collapse. Left ventricular collapse is reported rarely and as an isolated phenomenon in exceptional cases of postoperative effusion loculated over the left ventricle[8] and in a nonloculated effusion in one patient with pulmonary hypertension and abnormal right cardiac chambers.[9]

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METHODOLOGY

We evaluated the frequency and reliability of right-sided cardiac chamber collapses in diagnosing cardiac tamponade, analyzing only reports of investigations including both a control population and patients in whom tamponade had been established by both hemodynamic and clinical criteria, as well as by meticulous M-mode and two-dimensional echocardiographic investigations (Table I). The methodology of each series was scrutinized for non-echographic criteria that would correspond to established diagnostic requirements[10] including (1) hemodynamics showing virtual equalization of central diastolic pressures, (2) clinical observations of reduced arterial systolic pressures with (3) pulsus paradoxus, (4) jugular venous distension, and (5) improvement following pericardiocentesis. Thus these results were selected because the diagnosis and characterization of tamponade would satisfy the clinician and the hemodynamicist as well as the echocardiographer.

OBSERVATIONS (Table I)

Out of many careful descriptions and analyses of echocardiographic chamber collapses, four reports completely satisfied all requirements for both hemodynamic and clinical evidence. These series included