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# NONOPERATIVE DILATATION OF CORONARY-ARTERY STENOSIS 

## Percutaneous Transluminal Coronary Angioplasty

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#### Abstract

in percutaneous transluminal coronary angioplasty, a catheter system is introduced through a systemic artery under local anesthesia to dilate a stenotic artery by controlled inflation of a distensible balIoon.

Over the past 18 months, we have used this technic in 50 patients. The technic was successful in 32 patients, reducing the stenosis from a mean of 84 to 34 per cent ( $P<0.001$ ) and the coronary-pressure gradient from a mean of 58 to $19 \mathrm{~mm} \mathrm{Hg}(P<0.001)$. Twenty-nine patients showed improvement in cardiac function during follow-up examination. Because of acute deterioration in clinical status, emergen-


IN 1964, Dotter and Judkins ${ }^{1}$ introduced the technic of transluminal angioplasty for the treatment of atherosclerotic obstruction of the femoral artery. Despite their enthusiasm, the technic has been largely ignored in the United States. In Europe, however, several physicians have used this technic to treat large numbers of patients. ${ }^{2}$

Because of the technical difficulties, complications and limited usefulness of transluminal angioplasty in the treatment of obstruction of the peripheral arteries, we sought to modify the original technic of Dotter and Judkins. A double-lumen dilatation catheter with a nonelastic balloon was developed. ${ }^{3}$ Such catheters have been used since February, 1974, in patients with obstructions of the femoropopliteal and iliac arteries and have yielded low complication rates and no mortality. ${ }^{4}$

Recently, we modified this catheter to allow us to develop a technic for percutaneous dilatation of renalartery ${ }^{5}$ and coronary-artery stenosis. ${ }^{6}$ This technic was used in human beings ${ }^{7}$ after preliminary trials in dogs and cadavers. ${ }^{8-10}$ We present here preliminary results obtained with the technic in the past 18 months.

[^0]cy bypass was later necessary in five patients; three showed electrocardiograpic evidence of infarcts.

Patients with single-vessel disease appear to be most suitable for the procedure, and a short history of pain indicates the presence of a soft (distensible) atheroma likely to respond to dilatation. We estimate that only about 10 to 15 per cent of candidates for bypass surgery have lesions suitable for this procedure. A prospective randomized trial will be necessary to evaluate its usefulness in comparison with surgical and medical management. (N Engl J Med 301:61-68, 1979)

## Materials and Methods

## Technic

The basic equipment consists of two catheters (Firma H. Schneider, Zurich), the guiding catheter, which has an outer diameter of French 8-9, and the dilating catheter. The guiding catheter is inserted into the femoral artery according to the method of Seldinger or through a brachial arteriotomy under local anesthesia. The guiding catheter is advanced in a retrograde manner into the ascending aorta and positioned in the orifice of the coronary artery requiring dilatation. This catheter guides the dilating catheter into the stenotic arterial branch. The dilating catheter contains a double lumen to permit pressure measurements, contrast injection and inflation of the balloon. At the tip of this catheter, a short soft wire, 5 mm long, projects beyond the balloon (Fig. 1) and directs the catheter into the artery, thus avoiding injury to the arterial wall. By means of a fluoroscopic-image intensifier, the dilating catheter is advanced into the stenotic area. The balloon is filled with a liquid mixture of contrast medium and is inflated for three to four seconds at a pressure of 4 to 5 bar ( 400 to 500 kPa ); the balloon is then deflated, blocking the artery for about 15 to 20 seconds (Fig. 2). Inflation and deflation of the balloon are controlled by a calibrated pressure pump (Firma H. Schneider).
To estimate the extent of coronary-artery disease and the effect of dilatation, coronary angiography is peformed immediately before and after transluminal angioplasty. It is important to obtain views laterally, from both oblique angles and hemiaxially. ${ }^{11}$ The degree of stenosis is calculated from the mean stenosis seen in all projections and was determined by one of us (A. R. G.). In all cases, pressure was monitored, and pressure gradients across the lesions were recorded. A pacemaker was readily available during the procedure.

## Drug Treatment

The patient is given aspirin ( 1.0 g per day) for three days, starting the day before the procedure. Heparin and low-molecularweight dextran are administered during dilatation; warfarin is
started after the procedure and is continued until the follow-up study six to nine months later. To prevent coronary spasms, nitroglycerin and nifedipine are given before and during the procedure. The patient is discharged two days after angioplasty.

## Patient Selection and Evaluation

Patients with an accessible stenosis less than 1 cm in length, as judged from coronary arteriograms, and a short history of pain (less than one year) are most suitable for the procedure. The patients should also be likely candidates for operation as a result of disabling symptoms and their clinical status.

Results of previous catheterization studies and coronary angiograms of potential candidates for the procedure are shown to and discussed with the cardiac surgeons. The possible benefits and risks of the procedure and alternative treatments are explained to the patients. After informed written consent is obtained, the procedure is performed when the surgeon, anesthesiologist and operating room with cardiopulmonary bypass equipment are available.

Before transluminal angioplasty, the patient undergoes a baseline, quantitative, submaximal, bicycle ergometric examination in the upright position, ${ }^{12-14}$ which is repeated two days after dilatation. We classified the patients, according to the results of the stress tests, as follows: no pain or electrocardiographic changes with marked exertion (Class I); pain with marked exertion ( $\geqslant 50$ per cent of the predicted age, sex and height-adjusted working capacity consistent with steady state) (Class II); pain with minimal exertion (<50 per cent of the predicted working capacity) (Class III); or pain at rest (Class IV). A ${ }^{201} \mathrm{Tl}$ myocardial-perfusion scintigram can also be performed during the ergometric examination ${ }^{15-17}$ to evaluate perfusion after dilatation.

A 12-lead electrocardiogram and cardiac-enzyme estimation are performed before treatment and repeated every eight hours for the first 24 hours. Stress tests are given every three months for the first year, and coronary angiography is performed six to nine months after dilatation.


Figure 1. Percutaneous Transluminal Coronary Angioplasty and Catheters Used in the Procedure.
(a) Stenosis of the coronary artery is shown. (b) The doublelumen balloon catheter is introduced by use of a guiding catheter positioned at the orifice of the left or right coronary artery; at the tip of the dilating catheter is a short soft wire, which guides the catheter through the vessel. Proximal to the wire is a side hole connected to the main lumen of the dilating catheter. This lumen is used for pressure recording and contrast-material ejection. The dilating catheter is advanced through the coronary artery with the balloon deflated. (c) The balloon is inflated across the stenosis to its predetermined maximal outer diameter of 3.0 to 3.7 mm at a fluid pressure of 4 to 5 bar ( 400 to 500 kPa ), thereby enlarging the lumen. After balloon deflation, the catheter is withdrawn.


Figure 2. Original Tracing with Recording of Mean Pressure and Electrocardiogram during Dilatation, September 16, 1977, in a 39 -Year-Old Man with Severe Angina and 85 Per Cent Stenosis of the Left Coronary Artery.
CoP denotes coronary pressure, and AoP aortic pressure. The left-hand tracing (a) was the proximal pressure ( $114 / 96 / 105 \mathrm{~mm} \mathrm{Hg}$ ); the initial reduction in pressure occurred as the dilating catheter was passed across the lesion $(24 / 16 / \overline{20} \mathrm{~mm} \mathrm{Hg})$, occluding the artery. The position of the catheter was then checked by ejection of contrast medium (CM) distal to the stenosis through the main lumen of the dilating catheter (b). The balloon was inflated across the lesion (c), thereby enlarging the lumen. For 25 seconds, the coronary artery was totally occluded. No pain or change in the S-T segment occurred on the electrocardiogram. Dilatation reduced the stenosis from 85 to 29 per cent and the pressure rose ( $100 / 90 / 95 \mathrm{~mm} \mathrm{Hg}$ ) above the initial distal pressure. There was no substantial further rise in pressure in the pullback pressure curve ( $106 / 91 / \overline{97} \mathrm{~mm} \mathrm{Hg}$ ) (d). This observation indicated a good hemodynamic result, which was confirmed by the second arteriogram taken after coronary angioplasty.

Angiograms of this patient are shown in Figure 5.

## Patients

From September, 1977, to January, 1979, 50 patients underwent coronary angioplasty (Table 1). The patients were 31 to 67 years of age, with a mean of 49 . Forty-six patients were men, and four were women. The mean duration of angina pectoris since the patient first experienced pain had been $13 \pm 30$ months (mean $\pm$ S.D.). Twentysix patients ( 52 per cent) showed electrocardiographic evidence of mural or nontransmural infarction
Thirty patients ( 60 per cent) had single-artery disease (no other artery involved, with 50 per cent or greater reduction in luminal diameter) of the left anterior descending coronary artery, left circumflex artery or the right coronary artery. The remaining patients had two or three affected arteries. Eight patients ( 16 per cent) had previously received coronary-artery-bypass grafts and showed recurrent stenosis and symptoms.
Despite adequate medical therapy (beta-blocking agents and long-acting nitrates), exercise-induced angina pectoris (functional Classes II and III) or angina at rest (functional Class IV) was present in 36 ( 72 per cent) and 14 patients ( 28 per cent), respectively.
Detailed histories on all patients have been deposited with the National Auxiliary Publication Service.*

## Results

We attempted to dilate 53 vessels in 50 patients; 42 patients had severe stenoses of various coronary arteries, and eight other patients were studied after coro-nary-artery-bypass grafting (Table 2). As judged by a

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10 per cent or greater reduction of stenosis and pressure gradient, we successfully treated 34 vessels in 32 patients ( 66 per cent), namely, 29 of the 46 ( 63 per cent) coronary arteries and five of the seven ( 71 per cent) graft stenoses. The mean duration of angina pectoris had been nine months (range, one to 108) in the successfully treated patients.

The coronary stenosis was reduced on average from $84 \pm 9$ to $34 \pm 16$ per cent (paired Student's t-test; $\mathrm{P}<0.001$ ). The mean coronary-artery pressure distal to the stenosis increased from $27 \pm 10$ to $66 \pm 15 \mathrm{~mm}$ Hg ( $\mathrm{P}<0.001$ ), after dilatation, with a mean systemic pressure of 85 mm Hg . The pressure gradient was therefore reduced from $58 \pm 14$ to $19 \pm 13 \mathrm{~mm} \mathrm{Hg}$ ( $\mathrm{P}<0.001$ ). The decrease of the mean pressure gradient correlated well with the decrease of the stenosis after treatment (Fig. 3).

We were unable to dilate 19 vessels in 18 patients. In these subjects, the mean duration of angina pectoris had been 20 months (range, one to 192). Thirteen stenotic arteries could not be repaired because of anatomic factors, such as tortuosity of the right coronary artery, a sharp angle at the point where the left

Table 1. Patient Description and Results of Dilatation.

| No. of Patients | 50 |
| :---: | :---: |
| Age (yr) |  |
| Mean | 49 |
| Range | 31-67 |
| Sex |  |
| Male | 46 |
| Femaie | 4 |
| Duration of angina (mo) mean $\pm$ SD | $13 \pm 30$ |
| Functional classification (no. of patients) |  |
| Class I | 0 |
| Class II | 18 |
| Class III | 18 |
| Class IV | 14 |
| Coronary disease (no. of patients) |  |
| One vessel | 30 |
| Two vessels | 7 |
| Three vessels | 5 |
| Status after coronary-artery-bypass grafting | 8 |
| Failure | 18 |
| Medical therapy (no. of patients) | 1 |
| Elective coronary-artery bypass (no. of patients) | 10 |
| Emergency coronary-artery bypass (no. of patients) | 7 |
| Primary success (no. of patients) | 32 |
| Follow-up period |  |
| Mean (mo) | 9 |
| Range (mo) | 3-18 |
| Success (no. of patients) | 24 |
| Death (no. of patients) | 2 |
| Recurrence | 6 |
| Repeated dilatation (no. of patients) | 3 |
| Coronary-artery bypass (no. of patients) | 1 |
| Medical therapy (no. of patients) | 2 |
| Complications of 60 procedures, including 50 attempts, 3 repetitions \& 7 emergency coro-nary-artery-bypass grafts |  |
| Elevation of creatine phosphokinase <br> (MB fraction) ( $\geq 10 \mathrm{U} / \mathrm{liter})$ | 6 (10\%) |
| Mean of peak values (U/liter) | 35 |
| Range of peak values (U/liter) | 12-74 |
| Myocardial infarction (elevation of creatine phosphokinase [MB fraction] $\&$ positive electrocardiogram) | 3 (5\%) |
| Pulsating hematoma at puncture site (groin) | 1 |

Table 2. Results of Coronary Angioplasty.

| Artery | No. of Patients | Dilatation Completed Successfully | Recurrencein Follow- UP Period |
| :---: | :---: | :---: | :---: |
|  |  | no. of patients | no. of patients |
| Left coronary | 2 | 2 | 0 |
| Left anterior descending coronary | 33 | 21 | 3 |
| First diagonal branch of left anterior descending coronary | 1 | 0 | 0 |
| Left circumflex | 1 | 1 | 0 |
| Right coronary | 8 | 4 | 0 |
| Status after grafting | 8 | 6 | 3 |
| Venous graft | 5 | 3 | 2 |
| Native vessel | 1 | 1 | 0 |
| Both | 2 | 2 | 1 |
| Total | 53 | 34 | 6 |
| No. of patients | 50 | 32 | 6 |

anterior descending coronary artery branches off or tightness and eccentricity of the stenosis. In four patients, the stenotic area was passed and the balloon inflated, but the situation deteriorated; in two subjects, no reduction of stenosis or pressure gradient was possible. Seventeen of these 18 patients required surgical intervention, seven within 24 hours and 10 within four weeks. There were no surgical complications from the procedure.

Clinical success was classified as either primary (by the time of discharge) or late (any time thereafter). The 32 patients treated successfully showed improvement in clinical symptoms and stress tests leading to functional reclassification of 29 patients in the primary period (Fig. 4). Three patients were not functionally reclassified in this period; one improved after discharge, and two had recurrences in the follow-up period.

Fifteen of the 20 patients with single-vessel disease showed marked improvement (into Class I); however, only three of six with multiple-vessel disease could be so reclassified. Of the six other patients who were dilated successfully, one remained in Class II, one improved from Class II to I, two from Class IV to II and two from Class IV to III. Therefore, most patients who failed to reach Class I had many stenoses, but only some of the lesions could be dilated.

The mean working capacity at steady state rose from $86 \pm 48$ to $128 \pm 42 \mathrm{~W}$ ( 32 patients; $\mathrm{P}<0.001$ ). This change represented an increase from 53 to 80 per cent of the predicted age, sex and height-adjusted working capacity. In patients with single-vessel disease, the working capacity increased from $90 \pm 45$ to $143 \pm 31$ per cent ( 20 patients; $P<0.001$ ), which was within the normal range.

Thallium scintigrams performed during exercise were taken before and after the dilatation in 18 of 32 patients; they showed improvement of perfusion in 15 but revealed no change after dilatation in three patients who had normal scintigrams before the operation.


Figure 3. Pressure Recordings across Coronary Stenoses in 32 Patients ( 34 Vessels Dilated) with Primary Success.
The relation of the resting mean pressure gradient across the stenosis to the percentage of reduction in vessel diameter due to stenosis is shown before and after coronary dilatation. Significant decreases in pressure gradient and percentage diameter of stenosis were observed, with a correlation coefficient of $r=0.79 ; \mathrm{P}<0.001$.

Two patients have died. One death was unrelated to the procedure and occurred nine months later. The second death occurred unexpectedly in a 45 -year-old man (Patient 36) who had extensive hypertrophy of the medial smooth-muscle cells of the left main stem; the extent of hypertrophy had been underestimated on the basis of several angiograms, and the vessel was therefore incompletely dilated. The patient died two months after the procedure. Autopsy showed no occlusion or dissection of the left main coronary artery and no infarction. The cause of death was not clear.

Thirty-one patients had at least one follow-up examination, with a mean follow-up time of nine months (range, three to 18 ). Improvement of functional class was maintained in 20 patients; five patients showed further improvement, whereas six showed deterioration to a lower functional class (Fig. 4).

Follow-up angiograms were obtained six to nine months after dilatation for 16 of 25 patients showing consistent clinical improvement. The vessels remained widely patent. There was improvement in caliber and vessel smoothness in 13 of these 16 patients. Recurrence of stenosis ( 30 to 40 per cent) occurred in two patients, although they remained in Class I. Figure 5 shows dilatation of one stenotic left anterior descending coronary artery; the same procedure is shown in Figure 6 for a right coronary artery and in Figure 7 for a bypass-graft circumflex lesion.

By April, 1979, six recurrences had been observed (Tables 1 and 2); all occurred within three months after dilatation. Angiograms were obtained in all six patients. Three recurrences occurred after dilatation of stenotic saphenous-vein bypass grafts. One of these patients underwent repeated angioplasty, and the others were treated medically. The fourth recurrence was recorded in a patient with pseudoxanthoma elasticum and widespread progression of the disease. Cor-onary-artery bypass grafting was necessary. The fifth and sixth recurrences were observed after successful dilatation of severe stenosis of the left anterior descending coronary artery; in those cases, coronary angioplasty was repeated, again with at least initial success.

In the 50 attempts and three repeated dilatations, there were no deaths, no evidence of embolization, no central-nervous-system deficits and one femoral hematoma requiring evacuation (Table 1).

Of the 32 patients who underwent successful dilatation and the three who had repeated dilatations in the follow-up period, only one showed elevation of creatine phosphokinase (MB, or heart, fraction). This patient had renal failure, previous myocardial infarction and pain at rest owing to multiple-vessel disease and a lesion of the left main coronary artery. The ejection fraction was normal. He was the only patient who was denied an operation at the surgical conference because of the poor condition of the distal coronary arteries. The main-stem lesion was dilated, reducing


Figure 4. Analysis of Functional Status before and after Dilatation in 32 Patients Treated Successfully.
Before dilatation, 14 patients were in Class II, 11 in Class III and seven in Class IV. After dilatation, only three did not improve to a better functional class. All except one patient had at least one follow-up examination three months after the procedure. This patient died ( $\dagger$ ) two months after the procedure. Most patients retained higher functional status or showed further improvement (five patients), whereas six had recurrences, which were treated with a second dilatation (PTCA) in three patients, coronary bypass in one (ACBG) and medical therapy in two. The size of each circle is proportional to the number of patients.


Figure 5. A 38 -Year-Old Man with Typical Angina Pectoris since August, 1977 ( 75 W, Three Minutes, Pain, Elevation of S-T Segment, Left-Bundle-Branch Block, 49 Per Cent of Predicted Working Capacity).
(a) Coronary angiography showed 85 per cent stenosis of the left anterior descending coronary artery (right-anterior oblique view, $30^{\circ}$ ). A ${ }^{201} \mathrm{TI}$ scan showed a severe anteroseptal defect after stress testing on September 15. (b and c) Dilatation was performed on September 16. An angiogram taken after dilatation showed a patent vessel with a 29 per cent residual stenosis. The mean coronary-pressure gradient across the lesion had decreased from 85 to 2 mm Hg . After dilatation, marked improvement of the thallium scan and normalization of the stress test occurred ( 200 W , three minutes, S-T segment normal, no bundle-branch block, 121 per cent working capacity, Class I). (d) A coronary angiogram on October 20, four weeks after coronary angioplasty, showed further improvement of vessel patency and wall smoothness. The ${ }^{20171}$ myocardial-perfusion scintigram after exercise was normal on March 6, 1978, six months after coronary angioplasty. Stress testing in July gave the following results: 175 W , three minutes, no pain, S-T segment normal, 115 per cent working capacity, Class I.
the stenosis from 80 to 65 per cent and the mean pressure gradient from 47 to 8 mm Hg , but the balloon blockage of coronary blood flow (already severely impaired) over several 20 -second intervals may have caused myocardial necrosis, producing a maximum creatine phosphokinase (MB fraction) level of 50 U per liter. The electrocardiogram remained unchanged. However, the patient improved for eight months after dilatation and progressed into Class III. The patient then experienced a myocardial infarction resulting from reocclusion of the stenotic left anterior descending coronary artery. Angiograms at autopsy and pathological examination revealed a patent left main coronary artery.

The procedure failed in 18 patients. One patient with severe stenosis of the first diagonal branch of the left anterior descending coronary artery, exercise induced angina pectoris, pain at rest and nontransmural myocardial infarction showed improvement but presented with recurrent spasms after dilatation a likely explanation of the myocardial necrosis
(peak value of creatine phosphokinase [MB fraction], 20 U per liter) and the electrocardiographic evidence of lateral-wall infarction. The remaining 17 patients underwent coronary-artery-bypass grafting, seven of them within 24 hours. Of the 10 elective cases, none showed deterioration of symptoms, elevations of the MB fraction of creatine phosphokinase or electrocardiographic evidence of infarction after attempted dilatation. Six of the seven patients treated surgically within 24 hours were considered to require operations because of complications resulting from the procedure. The seventh patient had been scheduled for an emergency operation, and we were not able to dilate the stenotic area. In five of the six patients showing complications, a critical stenosis became a total stenosis, suggesting imminent infarction. Creatine phosphokinase (MB fraction) was measured after bypass grafting and showed elevations to maximum values of 12 to 74 U per liter (normal, $<10$ ) in four patients, two of whom demonstrated definite evidence of infarct on electrocardiograms. In the sixth patient, severe spasms occurred in the stenotic area after dilatation, resulting in elevations of the $\mathrm{S}-\mathrm{T}$ segment that resolved within a few minutes. Re-examination showed that the stenosis had not been reduced, so we decided to operate on the patient on the same day, even though there was no elevation of creatine phosphokinase. Five of the seven patients who underwent emergency operations had clinical re-evaluations six to eight months later. Four patients reached functional Class I, and one improved from Class IV to II.

## Discussion

The successful application of the balloon-dilatation technic to peripheral arteries ${ }^{4}$ in the past five years encouraged us to try the method on coronary arteries. The technic is comparatively simple and has the advantage of providing instantaneous revascularization without the need for open-heart surgery.
At present, the technic is limited by anatomic factors, such as vessel tortuosity, sharply angled arteries, cul-de-sac-like lesions and fibrotic or calcified stenoses. Most of our 18 failures can be attributed to these factors. Careful evaluation of the angiogram may reduce failures due to anatomic factors, and attention to the patient's history, in which a short duration of symptoms seems to correlate well with the distensibility of atheroma (mean duration of angina, nine months in primary successes versus 20 months in failures), may eliminate unsuitable hard lesions from consideration. These circumstances will limit this therapy to a small number of patients with coronaryartery disease. We estimate that 10 to 15 per cent of our surgically treated patients are suitable for the procedure at present.

Coronary angioplasty was successful in 32 of 50 patients, reducing stenosis from 84 to 34 per cent and decreasing the mean pressure gradient from 58 to 19


Figure 6. A 43-Year-Old Man with Typical Angina Pectoris since July, 1977 ( 150 W, Three Minutes, Pain, Depression of the S-T Segment, 75 Per Cent of Predicted Working Capacity, Class II).
(a) Coronary angiography (left-anterior oblique view, $60^{\circ}$ ) showed 82 per cent stenosis of the right coronary artery, 64 per cent stenosis of the left anterior descending coronary artery and 50 per cent stenosis of the left circumflex artery (not shown). A thallium scan showed an inferior perfusion defect after a stress test. (b and c) Dilatation was performed on November 21, 1977. The stenosis was reduced to 31 per cent. At the same time, stenosis of the left anterior descending coronary artery was reduced from 64 to 33 per cent. The mean coronary-pressure gradient decreased from 51 to 8 mm Hg and from 56 to 27 mm Hg , respectively. Myocardial perfusion, measured on November 28, improved after angioplasty. (d) Restudy eight months after coronary angioplasty, on July 10, 1978, had shown a patent right coronary artery, with further improvement of vessel patency. A thallium scan on April 18 showed normal myocardial perfusion after exercise. January, 1979: 200 W , three minutes, no pain, S-T segment normal, 102 per cent of predicted working capacity, Class I.
mm Hg . However, the pressure gradient across the stenosis provides only an index of the severity of the lesion since insertion of the dilatation catheter (Figs. 3 and 4) contributes to the stenosis. However, if there is no change in heart rate or blood pressure, a decline in the mean pressure gradient after dilatation must represent a reduction in stenosis since the size of the catheter remains the same. The increase in distal coronary pressure after dilatation of the balloon can therefore be used to determine whether the balloon should be inflated again to achieve optimal results.

Restoration of the original lumen, although ideal, is not necessary, since improvement in working capacity from 86 to 128 W was especially clear-cut in patients with single-vessel disease. The improvement of ${ }^{201} \mathrm{Tl}$ myocardial perfusion after dilatation underlines this observation. ${ }^{18}$

The question that now arises is whether the beneficial effect can be substantiated in follow-up studies. Although our experience with the procedure is limited, the first 16 patients restudied six months after dilatation did show results similar to the experience with peripheral vessels ${ }^{4}$ and are thus encouraging. Moreover, in 13 patients, further improvement of vessel patency and wall smoothness was observed. This observation may reflect a self-healing process that took place after the controlled injury caused by balloon inflation, resulting in compression of the atheroma, with intimal tearing and enlargement of the outer diameter of the vessel. Similar changes have been described in histologic studies after dilatation of peripheral arteries. ${ }^{4,9,19}$ The restoration of good blood flow through the dilated segment of the vessel seems to have a major role in this process.

On the other hand, six recurrences occurred in the follow-up period, namely, three of 27 patients who underwent coronary-artery dilatation and three of five undergoing dilatation of saphenous-vein-graft stenosis. Two of the former and one of the latter were subjected to repeated angioplasty, with anatomic and clinical primary success. The different kind of disease may explain the high incidence of recurrence in graft stenosis. Further experience will show whether we should eliminate this lesion from consideration.

Two points must be made. We have not been too successful in dilating stenotic main stems of left coronary arteries. It has been difficult to estimate the extent of disease in this area and the presence of concomitant spasm. We feel that these factors contributed to the death of one patient two months after dilatation. Secondly, although the procedure is relatively simple, it requires special experience. Moreover, the potential complications are both serious and sudden, so that it is mandatory that a competent surgeon be available for emergency coro-nary-artery bypass should it become necessary. The procedure should not be performed in hospitals lacking this facility. We believe that early surgical inter-


Figure 7.
A 43-Year-Old Man Who Received Grafts Bypassing the Right Coronary, Left Anterior Descending Coronary and Left Circumflex Arteries in July, 1977, and Showed Recurrence of Symptoms since July, 1977 ( 50 W, Three Minutes, Pain, Depression of S-T Segment, 32 Per Cent of Predicted Working Capacity; January, 1978: Pain at Rest, Class IV).
(a) Arteriography (right-anterior oblique view, $30^{\circ}$ ) showed stenosis of the graft bypassing the left circumflex artery at the distal implantation site and progression of the disease to the recipient vessel ( 92 per cent stenosis). Occlusion of the bypass graft across the right coronary artery was demonstrated, as was 30 per cent stenosis of the graft bypassing the left anterior descending coronary artery, 64 per cent stenosis of the left anterior descending and 100 per cent of the right coronary arteries. A 201 Tl scintigram showed an inferoposterior-perfusion defect after stress testing. ( $b$ and $c$ ) Dilatation was performed on January 23, 1978. This patient was the first for whom we know the status after coronary-artery bypass. Stenosis of the left circumflex artery decreased from 92 to 20 per cent, and the native vessel of the left anterior descending coronary artery decreased from 64 to 23 per cent. Myocardial imaging returned to normal. Stress-test results improved ( 100 W , three minutes, pain, depression of the S-T segment, 64 per cent of predicted working capacity, Class II). (d) Re-evaluation on August 25, eight months after coronary angioplasty, showed a patent graft and recipient vessel with improved anatomic structure. Myocardial scintiscanning on May 8 had revealed no perfusion defect after exercise, and stress-test results improved ( 125 W , three minutes, pain, depression of the S-T segment, 80 per cent of predicted working capacity, Class II).
vention prevented major infarction in five patients. In two of them, forceful manipulation of the dilatation catheter caused an intimal dissection at the stenosis, whereas in the others, complete occlusion of the stenosis occurred after uneventful dilatation. The precise mechanism of the occlusion is not clear. Inspection of the artery at operation revealed neither vessel damage and extravasation nor peripheral embolization. It is possible that the fibrous "cap" of the atheroma ruptured and that a partial dissection occurred in the diseased intima, creating a flap that occluded the stenotic area.

We emphasize that our results are preliminary. More information and follow-up data are needed before coronary angioplasty can be accepted as one form of treatment for coronary-artery disease. However, the results in patients with single-vessel disease are sufficiently good to make the procedure acceptable for prospective randomized trials. Such trials are clearly needed if we are to evaluate the efficacy of this new technic as compared with current medical and surgical treatments.

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# MINERALOCORTICOID-INDUCED HYPERTENSION IN PATIENTS WITH ORTHOSTATIC HYPOTENSION 

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Abstract The mechanism of recumbent hypertension induced by fludrocortisone was studied in seven patients with orthostatic hypotension. All showed increases in blood pressure in the recumbent and standing positions, and hypertensive levels were achieved on recumbency in four of them. Hypertensive retinopathy developed in two patients and cardiomegaly in one.

Initial blood-pressure elevations were associated with sodium retention and plasma-volume expansion. However, with long-term treatment, plasma volume decreased to control levels despite further bloodpressure increases. Treatment did not affect plasma
levels of catecholamines but did enhance pressor responsiveness to infused norepinephrine in some subjects. Hemodynamic studies indicated that hypertension in the recumbent position was related to increases in total peripheral-vascular resistance and not to changes in cardiac output.
Clinically, hypertension in the recumbent position is an important risk of fludrocortisone treatment in patients with orthostatic hypotension. This unusual model of chronic mineralocorticoid-induced hypertension is not volume dependent but is related to increased peripheral-vascular resistance. (N Engl J Med 301:68-73, 1979)
patients with essential hypertension. ${ }^{2}$ However, other data have suggested that the hypertension may not be directly related to a hypervolemic state, ${ }^{3}$ except possibly in its early phase. ${ }^{4}$ A similar conclusion was recently reached on the basis of experimental studies in dogs treated with deoxycorticosterone acetate or metyrapone. ${ }^{5,6}$

We have examined the effects of fludrocortisone in seven patients with orthostatic hypotension followed for one to 14 years. We have assessed the effects of the drug on sodium balance, plasma volume, plasma renin activity, sympathetic-nervous-system function, vascular reactivity and cardiovascular hemodynamics and have related the findings to changes in blood pressure.

## Materials and Methods

Seven patients with severe orthostatic hypotension, ranging from 21 to 78 years of age, were studied. All were markedly restricted in activity because of orthostatic hypotension and had experienced episodes of syncope, when systolic blood pressure on standing was


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