THE USE OF PERCUTANEOUS CARDIOPULMONARY BYPASS IN HIGH RISK CORONARY ANGIOPLASTY

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PTCA was performed under cardiopulmonary bypass in 9 high risk patients with a mean left ventricular ejection fraction of 23%. Seventeen French cannulas of the pump-oxygenator system were inserted percutaneously in the catheterization laboratory. Mean bypass time was 50 minutes and flow rate was 2.1 lt/min. All the target lesions were dilated successfully. There was no significant complication related to the bypass procedure. Hemodynamic parameters such as right atrial, pulmonary artery, pulmonary capillary wedge, aortic pressures and cardiac output and heart rate did not change significantly during or after bypass.

In conclusion, percutaneous cardiopulmonary bypass can be used safely in high risk patients undergoing coronary angioplasty.

Key words: Cardiopulmonary bypass,coronary angioplasty,PTCA.

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The use of Percutaneous Transluminal Coronary Angioplasty (PTCA) has dramatically increased in the treatment of multivessel coronary artery disease in recent years[1]. But there is still some limitations of this procedure such as restenosis, acute vessel occlusion and diffuse disease[2].

Acute vessel stenosis occurs in approximately 5% of patients undergoing elective PTCA[3]. This may result in acute myocardial infraction, severe hemodynamic collapse and may require emergency bypass surgery.

Patients who have poor left ventricular function and/or target vessels supplying more than half of the viable myocardium are at high risk and not considered as favorable candidates for PTCA. In order to reduce the potential hemodynamic problems in this high risk group percutaneous cardiopulmonary bypass (CPB) is used as a supporting system[4,5].

In this study we performed PTCA under percutaneous CPB in 9 high risk patients in order to investigate the value and efficacy of this support system.

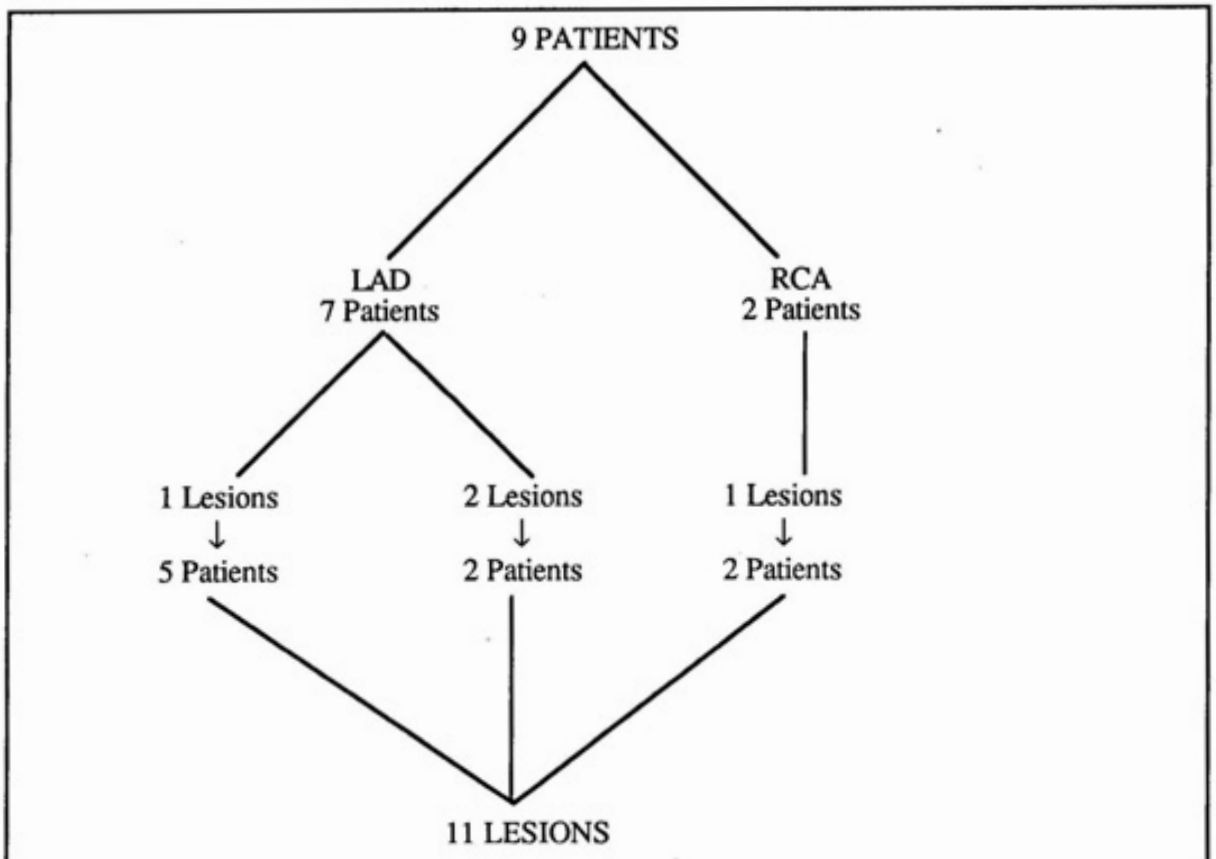

Fig.1. Distribution of vessels dilated.

Material and Method

The study group consisted of 9 male patients with an average age of 54±9 (38-68). Seven of the patients had previous myocardial infarction.

Left ventricular ejection fraction ranged from 10% to 50% (mean 23±13%). Three patients had triple vessel disease and two had double vessel disease.

In 7 patients only one lesion in one vessel (culprit lesion) was dilated. In remaining two patients two lesions in one vessel were dilated. The distribution of the vessels dilated is shown in Fig.1.

The reason for performing PTCA with aid of CPB was very low ejection fraction in 8 patients and in one patient the target vessel which was a dominant right coronary artery was supplying more than half of the viable myocardium. So, all the patients were considered to be poor candidates and carrying high risk of mortality for bypass surgery.

All patients were in NYHA class III or more.

The hemodynamic parameters before, during and after the procedure were statistically evaluated by student's t test.

Technique

All phases of these procedures were performed in the catheterization laboratory. The patients were prepared in usual manner. No hypnotic agents were used other than local anesthetics for both puncture sites. Left femoral artery and vein were usually used for CPB. Eight or 9 French guiding catheter was placed to the right femoral artery. A thermodilution catheter (Spectramed) with an extra lumen permitting the passage of a pacing electrode was placed in the pulmonary artery. The tip of the pacing electrode was positioned in the right ventricular apex.

A stiff 0.038 inch guidewire was placed through the sheath on the left side, and the

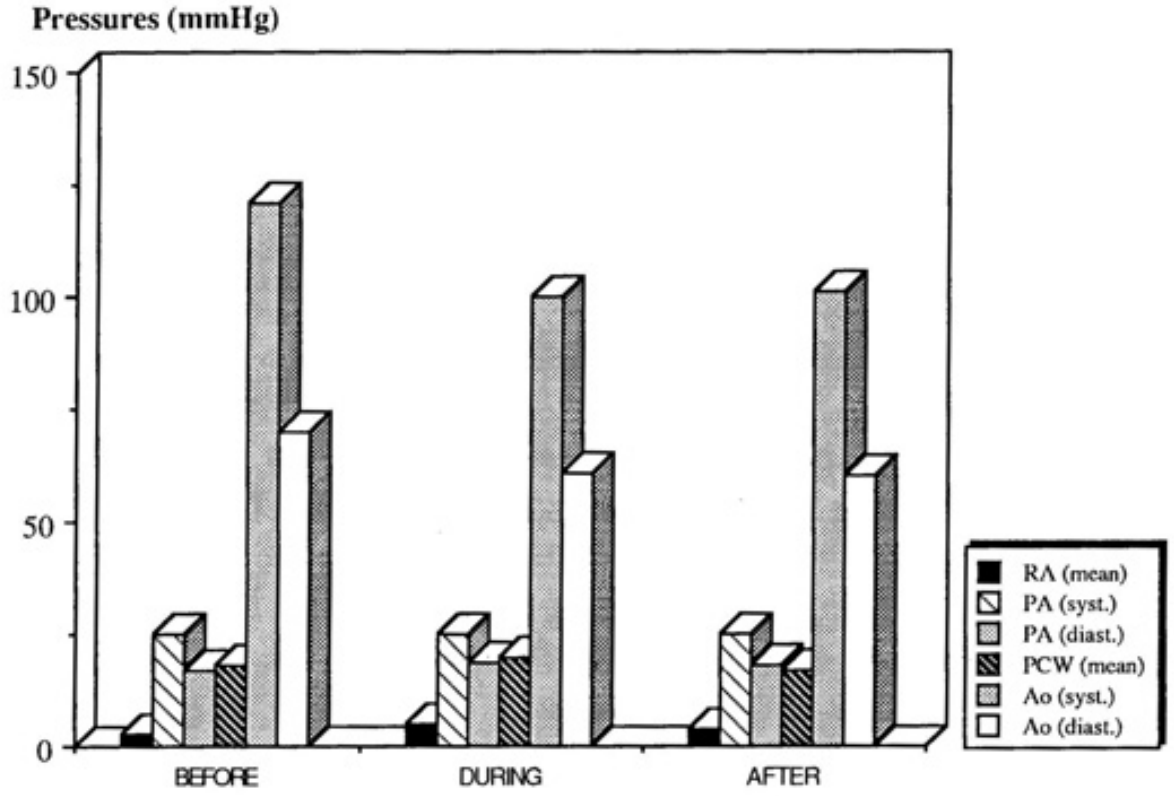

Fig. 2. Right atrial, pulmonary artery, pulmonary capillary wedge and aortic pressures

artery was then dilated with increasing size dilators (12F, 14F) over this extra-stiff guidewire.

Seventeen French cannulas were inserted percutaneously into the left femoral vein and artery. The distal tip of the arterial cannula was left in the iliac artery and distal tip of the venous cannula was advanced to the inferior vena cava-right atrium junction.

The cannulas were connected to the CPB system consisting of Biomedicus Pump with a membrane oxygenator (Scimed Ultrox I) and heat exchanger. As soon as the cannulas were inserted the patients were heparinized with a dosage of 300u/kg to keep Activated coagulation time (ACT) over 350 seconds. This dosage was given as a bolus and ACT was checked every 15 minutes.

Before starting the pump; right atrial, pulmonary artery wedge and aortic pressures were recorded. Cardiac output was measured by thermodilution technique. These parameters were recorded again during the procedure and following the procedure after stopping the pump.

The flow rate delivered by the system ranged between 1.5 and 3.5 lt/min.

PTCA of the target lesions were performed in a standard way with right femoral approach.

After completion of PTCA cardiopulmonary pumping was tapered off over 5-20 minutes. Residual blood in the system was pumped as much as possible to the patient. The cannulas were disconnected but left in place until the patient was transferred to the intensive care unit.

Heparin effect was not reversed with protamine. After waiting for a time for ACT to decrease to about 250 seconds the cannulas were removed. Bleeding from cannula insertion sites was controlled by prolonged manual compression and then a tight bandage was applied.

Results

In all patients percutaneous cardiopulmonary bypass procedure was completed successfully. The average bypass time was 50 minutes. Flow

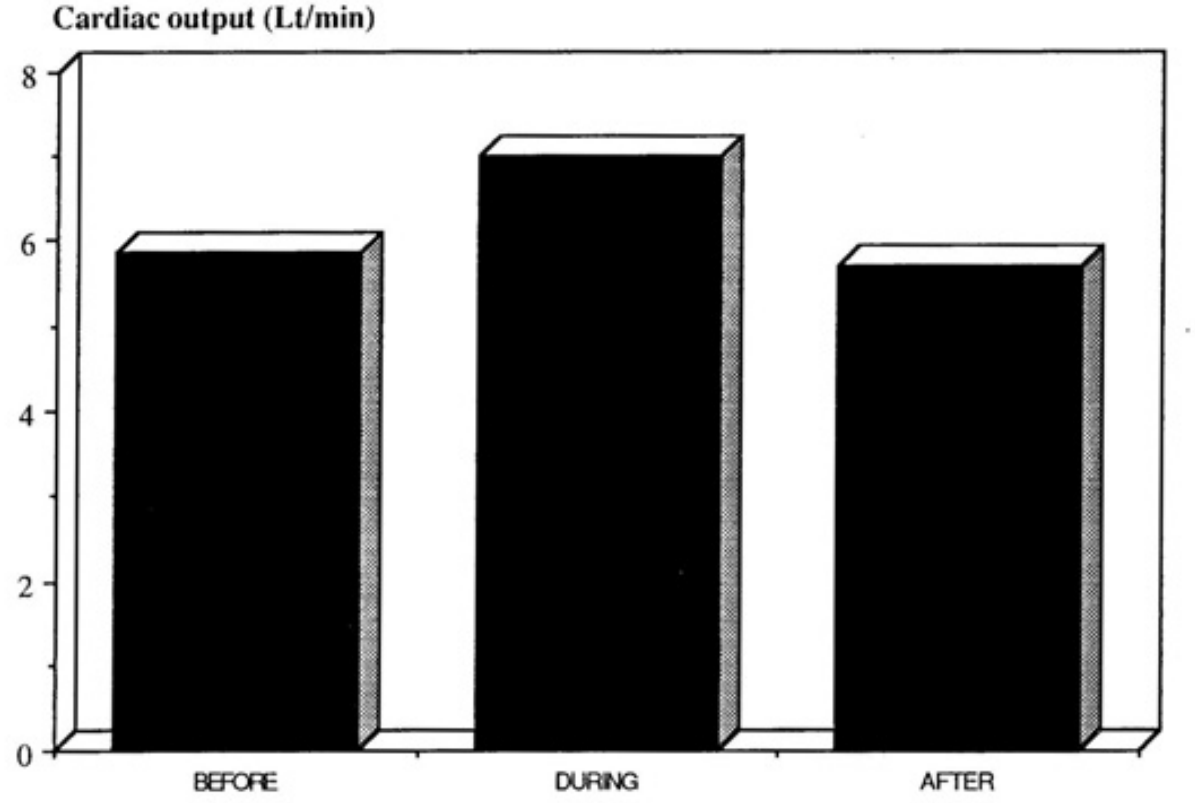

Fig.3. Cardiac output values

rate ranged from 1.5 lt/min. to 3.5 lt/min. with an average of 2.1 lt/min.

Eleven of the 12 lesions attempted were dilated sufficiently. In two patients second lesions in the vessel other than target vessel could not be dilated.

In four patients with left anterior descending lesions chest pain and ST segment depression occurred during balloon inflation.

The mean luminal diameter narrowing in dilated lesions was reduced from 86% to 28% following PTCA.

Hemodynamic parameters before, during and after CPB are outlined in figures 2. and 3. and Table I. There was no statistically significant difference in right atrial, pulmonary artery, pulmonary capillary wedge, aortic pressures, cardiac output and heart rate.

Mean right atrial pressures before, during and after CPB were 3.86, 6.71, and 5.86 mmHg respectively. Pulmonary arterial systolic and diastolic pressures were 30.5-16.5, 31.1-19.0, and 28-17.13 mmHg. Mean pulmonary capillary wedge pressures before, during and after CPB were 17.33, 19.67 and 16.67 mmHg respectively. Aortic systolic and diastolic pressures were 122.1-69.3, 100.7-63.57 and 103.6-60 mmHg before, during and after the procedure.

Cardiac output was 5.88 Lt/min. before CPB, increased to 7.06 Lt/min. during the procedure and was 5.71 Lt/min. after CPB.

In no patient surgical repair of the femoral artery or vein was required. There was also no significant leg pain in any patient due to possible ischemia which could be induced by relatively large size cannulas.

In the first 5 patients hematocrit values were reduced significantly (from 42% to 25%) at the end of bypass procedure and blood transfusion was required. For this reason, the subsequent 4 patients were transfused both during the procedure and immediately following.

Eight of the 9 patients were discharged from the hospital with at least one NYHA class improvement. One patient died 6 hours after supported PTCA probably because of acute vessel occlusion.

Table I. Clinical and hemodynamic characteristics of patients.

	Extent of disease	NYHA class B.A	PCWP (mean) mmHg			PAP (S/D) mmHg			Flow rate (L/min)	By-pass time (min)
			B.	D.	A.	B.	D.	A.		
1. KY	2 vessel disease MI(+)	III I	8	21	20	24/12	38/18	40/22	1.5	90
2. HÖ	1 vessel disease MI(+)	III II	18	25	22	36/22	42/30	42/30	1.8	45
3. NK	1 vessel disease MI(+)	IV II	10	8	10	34/18	20/14	22/12	3.5	50
4. RA	1 vessel disease MI(+)	IV II	20	18	12	28/20	22/17	24/12	3.0	60
5. AL	3 vessel disease MI(+)	III II	22	16	24	30/14	23/16	34/25	1.5	35
6. NT	3 vessel disease MI(+)	III Ex.	8	8	6	24/10	38/22	30/20	1.8	45
7. İS	1 vessel disease MI(+)	III I	6	8	6	18/10	20/10	14/8	2.1	50
8. HU	3 vessel disease MI(+)	III II	30	30	14	50/26	45/25	18/5	2.0	35
9. SC	2 vessel disease MI(+)	III I	16	12	16	24/12	22/14	22/18	1.7	40

PCWP : Pulmonary artery wedge pressure
PAP : Pulmonary artery pressure
MI : Myocardial infarction
B : Before, D: During, A: After
S/D : Systolic/diastolic

Discussion

Patients with severely depressed left ventricular function, extensive coronary artery disease, prior coronary artery bypass surgery, and old age are particularly hard cases for both cardiologist and surgeons. These patients are at high risk during PTCA and severe hemodynamic collapse may easely develop in case of acute vessel occlusion during procedure. Supported angioplasty was considered for this group of patients. Intraaortic balloon counterpulsation, hemopump device and cardiopulmonary bypass are already available modalities for supported angioplasty[2].

Percutaneous cardiopulmonary bypass has some advantages over the other techniques. First, circulatory support can be maintained irrespective of cardiac rhythm or pump function. Second; as cardiopulmonary support accomplishes substantial cardiac unloading, myocardial infarction following coronary occlusion would be minimized. Third; patients going to surgery after failed PTCA would have greater potential for receiving internal mammary artery grafts in a less urgent situation. Fourth; this system permits more prolonged balloon inflation which is suggested to be inducing

improved patency[2]. Patients can tolerate prolonged inflation time with less pain and ST segment changes. This is probably because, less myocardial ischemia is induced due to the left ventricular unloading and lowered end diastolic pressure. In four patients with left anterior descending artery lesions in our study group, however, ST segment depression and chest pain developed proves that myocardial ischemia occured during balloon inflation. Echocardiographic data from some studies also demonstrated myocardial ischemia during balloon inflation. Perfusion of the myocardium distal to the occlusion by using a seperate perfusion system may be a solution to this problem[4].

In all of our patients culprit lesions were satisfactorily dilated. The patients were in stable condition hemodynamically during bypass procedure and PTCA.

Mean pulmonary capillary wedge pressure in our study group was not changed significantly during bypass. This finding is not in accordance with what was found in other studies in which pulmonary capillary wedge pressure decreased to less than 10 mmHg during bypass[4,6]. This could be explained with relatively low pump output we maintained which does not induce sufficient left ventricular unloading. We believe that pulmonary capillary wedge pressure and thereby left ventricular loading can be lowered with increasing the pump output by using larger size cannulas.

In all patients the most significant problem encountered with was the loss of blood and need for blood transfusion. The major reason for this was the blood remained in the bypass system. Therefore in all patients at least two units of blood transfusion was required. Pumping the remaining blood in the system back to the patients following procedure, and use of cell savers and concomitant blood transfusion will probably prevent this untoward effect.

In USA, data from 105 patients undergoing elective supported angioplasty were entered in to the National Registry of Elective Supported Angioplasty in 1988. The most common complication related to the procedure involved vascular problems at the cannulation site[2]. In-hospital mortality rate was 7.6% in this group. Old age (>75) and left main coronary artery stenosis were two major risk factors in these patients.

The major limitation of our study is the absence of a control group. But we would not like to perform PTCA in such a high risk group without the aid of a support system. This issue can be investigated by simply placing guidewires, and inserting the cannulas only when the circulatory support is required because of a hemodynamic collapse.

Although our initial experience shows that percutaneous CPB allows successfull intervention in high risk patients, further controlled studies are needed in larger group of patients whether or not this approach is superior to unsupported PTCA with regard to hemodynamic and long term clinical outcome.

In conclusion; despite the limited number of patients our initial experience suggests that percutaneous cardiopulmonary bypass can be used successfully and with acceptable morbidity in PTCA of high risk patients. This technique not only permits hemodynamic stability during PTCA, but also reduces the operator's anxiety about arrhythmias and possible other complications, and is likely to fill the gap between standart interventions and surgery particularly in extensively diseased high risk patients.

References

1- Holmes DR, Vlietstra RE: Percutaneous transluminal coronary angioplasty: Current status and future trends. Mayo Clin Proc 1986;61:865.

2- Topol EJ: Textbook of Interventional Cardiology. Philadelphia. W.B. Saunders Comp. 1990 pp.363-394.

3- Cowley MJ, Dorros G, Kelsey SF, Van Raden M, Detre KM: Acute coronary events associated with percutaneous transluminal coronary angioplasty. Am J Cardiol 1984;53:12C-16C.

4- Shawl FA: Percutaneous cardiopulmonary bypass support in high risk interventions. J Inv Card 1989;1:287-293.

5- Vogel R: The Maryland experience: Angioplasty and valvuloplasty using percutaneous cardiopulmonary support. Am J Cardiol 1988;62:11K-14K.

6- Shawl FA, Domanski MJ, Hernandez TJ, Punja S: Emergency percutaneous cardiopulmonary bypass support in cardiogenic shock from acute myocardial infarction. Am J Cardiol 1989;64:967-970.