A Method to Detect Rupture of the Arterial Wall during Balloon Angioplasty

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Abstract
Restenosis limits the long-term success rate of Percutaneuos Transluminal (Coronary) Angioplasty (PT(C)A) significantly. A relation between ruptures resulting from PT(C)A and restenosis has been suggested in literature. We developed a method to detect arterial lesions during the balloon inflation by measuring the pressure in the balloon. We state that a sudden drop in balloon pressure indicates an arterial rupture. Our preliminary results support this hypothesis in 92% of all cases. This method can be useful when investigating the relationship between modified PT(C)A procedures and occurence of lesions.
Keywords: restenosis, angioplasty, balloon pressure, rupture

Introduction

Percutaneous Transluminal (Coronary) Angioplasty (PT(C)A) has proven to be an effective technique to treat both coronary and peripheral sclerotic arterial disease, and will pertain this role in the near future. Though this technique is commonly used, the knowledge about the mechanical behaviour of plaque and arterial wall during PT(C)A still is scarce [1,2,3,4]. PT(C)A is a fairly safe technique with a high immediate success rate and a small number of severe shortterm complications like dissection, rupture, perforation and occlusion. Longterm success rate is significantly lower because of restenosis. Between 25 an 40% of all patients with succesful first PT(C)A suffer from restenosis within 6 months [5,6,7]. Many modified procedures to overcome these limitations of PT(C)A are proposed, but so far none of them seems to decrease rate of restenosis significantly [4,6,7]. Several reports suggest a relationship between restenosis and lesions resulting from balloon inflation. In order to investigate the relationship between arterial lesions resulting from balloon inflation and modified procedures we developed a method to detect arterial lesions as soon as they occur, i.e. during the balloon inflation. The method we suggest here is based on measurement of the pressure in the balloon. A sudden change in balloon parameters (e.g. pressure or volume) to detect rupture of the arterial wall has been suggested before. Demer [2] suggest that the discontinuities she found in the relations between pressure and volume of the balloon are caused by ruptures of the arterial wall. Evidence showing the relation between a pressure 'drop' and wall rupture has not been published yet. In this paper we present preliminary results of the tests we performed to verify possible relations.

Methods

We developed a system for computer controlled balloon inflation that consists of a motor-driven syringe [8]. This system is used to perform balloon dilations in an in-vitro experiment with normal and atherosclerotic, human, post-mortem, femoral arteries. Arteries were obtained after informed consent of the family of the deceased. With the motor-driven syringe it is possible to apply constant strain rates to the arteries, using the relation between diameter and volume of the unopposed balloon we measured in advance. Diameters of the balloon and the arterial segment are measured using 4 linear variable displacement transducers (Jensen LDT-5L, reproducibility of mean arterial diameter 0.012 mm). Possible translations of the balloon or the arterial segment are eliminated in calculating the mean outer diameter by using two orthogonal diameters. Volume of the balloon is measured by measuring the position of the plunger with one LVDT (HBM). We use Olbert angioplasty balloon-catheters of Meadox Surgimed A/S because of reproducible volume-diameter relation ($\pm$ 0.07 mm diameter change at specified volume at 37°C after at least one hour of rest). The pressure-diameter relation at the specified strain rate is also measured before the experiment. Strain is defined as the relative increase of the inner diameter of the segment. Calculations of strain are made under the assumption of wall incompressibility. Pressure is measured using a transducer (Baldwin-Lima-Hamilton) placed between the syringe and the balloon catheter entrance. With the pressure-diameter and volume-diameter relations it is possible to calculate the dilation pressure inside the arterial wall. Dilation pressure is defined as the difference between the pressure in the balloon during dilation of the arterial segment and the pressure in the unopposed balloon at the same volume. Because of visco-elastic effects it is necessary to perform both measurements with the same strain rate. Before balloon inflation mean inner and outer diameter of the arterial segment are obtained by ultra-sound (10 MHz mechanical sector scanner, Diasonics Master, resolution 0.15 mm,

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reproducibility ±0.2 mm). The arterial segment is conditioned before the experiment in an oxygenated Tyrode solution at physiological temperature and pH by changing the intraluminal pressure gradually between 20 and 100 mmHg until a reproducible pressure-diameter relation is obtained. During the experiments pressure-time (figure 1) and diameter-time curves can be plotted real time. As soon as a decline of pressure was detected we stopped the inflation of the balloon, to be sure no ruptures would be caused after the first pressure drop was detected. If no decline was detected, inflation was performed until maximum balloon pressure or diameter was reached. After the experiment the segment was filled under pressure with gelatin and fixed in 4% neutral buffered formaldehyd. Subsequently the segments were cut transversely at the site of dilatation and 2cm next to it. Paraffin sections were stained with Elastica van Gieson and H&E for histological examination.

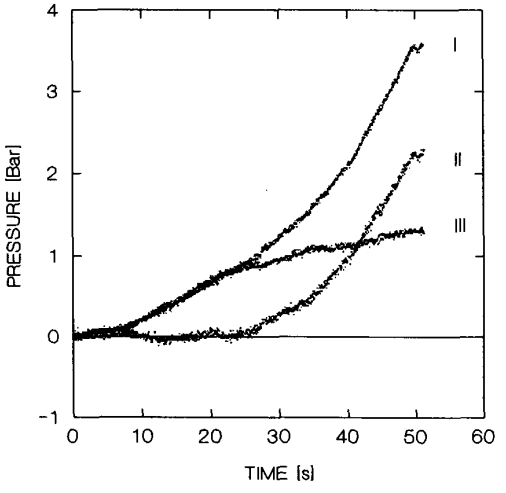

Fig. 1: Experimental pressure curves; (I) pressure during angioplasty, (II) dilation pressure, (III) pressure in unopposed balloon. Partial rupture at t=50s.

Results

We performed 18 experiments with human femoral arteries, applying constant strain rates varying from 0.10%/s to 2.4%/s. Five experiments were inconclusive because of technical problems or undecissive histological results. In 8 experiments we detected a sudden decrease in dilation pressure. Histological results of the 8 experiments with a pressure decline show a rupture of the arterial wall. Balloon inflation did not cause a sudden pressure drop in 5 experiments. One of these 5 experiments showed arterial wall rupture at microscopy, 4 showed intact arterial wall.

Conclusions

Our preliminary results support the hypotheses that a decrease in balloon dilation pressure effectively can be used to indicate rupture of the arterial wall during PT(C)A in 92% of all cases. They demonstrate the importance of further research on this subject. We also plan to investigate on the relationships between rate and duration of dilation and diameter, pressure, stress and strain at which rupture in the arterial wall takes place.
The effectiveness of our method has not yet been tested in-vivo. Clinical measurement of the pressure inside the angioplasty balloon is difficult because of the use of viscous contrast fluids and the great and sudden changes in balloon pressure that are induced by manual inflation of the balloon. If these complications can be overcome (e.g. by the use of a computer-controlled inflation system as we described) we assume that the method we propose here can be of importance clinically. More research will be needed for final conclusions for in-vivo situations.

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