Intracoronary artery shunt: An assessment of possible coronary artery wall damage

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Multivessel off-pump coronary artery bypass (OP-CAB) is currently feasible in many patients^{1,2} and yields anastomoses of excellent quality. Intracoronary shunts are routinely used to ensure an optimal bloodless operative field and to reduce the onset of myocardial ischemia.³ However, the effects of these devices on coronary artery endothelial integrity have not been thoroughly assessed.

The aim of this study was to investigate such effects on coronary endothelial cells and the artery wall in a human model that would exactly mimic the atheromatous coronary artery scenario of patients with ischemic coronary artery disease.

Patients and Methods

Informed consent was obtained. Eight patients who underwent heart transplantation for dilated cardiomyopathy caused by ischemic coronary artery disease were included in the study group. Intravenous heparin (3 mg/kg) was administered to maintain an activated clotting time of greater than 400 seconds. Before the start of cardiopulmonary bypass (CPB) on the beating heart, an intracoronary shunt (DLP, Medtronic, Inc, Minneapolis, Minn) was inserted, with the left anterior descending coronary artery (LAD) as the target vessel (Figure 1).

Stabilization of the target artery was accomplished with the Octopus stabilizer (Medtronic, Inc). The LAD was occluded by being encircled with a 5-0 polypropylene suture buttressed with a 1-cm piece of silicone tubing as abutment and lashed with a soft silicone tube. After the arteriotomy, the proximal snare was tightened, avoiding excess tension to obtain a bloodless operative field, and the intraluminal shunt was inserted. The vessel was then reperfused by releasing the snare; after a 10-minute period of coronary shunting, CPB was started, the aorta was clamped, and the heart was excised and placed in a modified Krebs-bicarbonate solution.

The LAD was dissected with the adjacent myocardium (Figure 2) and epicardial tissue and was divided in rings 4 mm in width.

Figure 1. Intraoperative view of the left descending coronary artery with the inserted intraluminal shunt, Prolene 5-0 suture (proximal and distal), and a single arm of the Octopus stabilizer. Note that the patient is cannulated, with the rapid possibility to switch in CPB any time it was requested.

Figure 2. Block (10 cm) of ventricular wall that included the target coronary vessel with the suture snares and the intraluminal shunt.

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Seven different segments of the LAD were analyzed: the site of proximal snare application, the site between the proximal snare and coronary arteriotomy, the segment of coronary arteriotomy, the site between coronary arteriotomy and the distal snare, and the site of the distal snare application, therefore accounting for all the coronary segments in contact with the shunt. The proximal and distal unmanipulated LAD segments respective to the snares served as controls.

Figure 3. Sample of coronary artery ring between the anastomotic and suture snare sites; this coronary segment contains a brushing of the endothelial surface caused by the shunting maneuvers. At scanning electron microscopy, only partial endothelial lining is present (arrow).

The rings were first fixed with buffered paraformaldehyde 4% and then washed for 1 minute with *N*-2-hydroxyethylpiperazine-*N*-2-ethanesulfonic acid sucrose buffer solution. Gross, light microscopy, and scanning electron microscopy examinations were performed on the LAD, grading endothelial and coronary wall damage according to severity: grade I, intact endothelial layer; grade II, partial endothelial loss; and grade III, endothelial denudation with the subendothelial layers exposed to blood flow.⁴

For histologic study, the tissues were dehydrated and embedded in paraffin. Sections of tissue 5- to 7- μ m thick were stained with hematoxylin and eosin and Weigert van-Gieson stain. For scanning electron microscopy, the specimens were dehydrated at the critical point dryer, covered by gold, and examined (Philips XL30 scanning electron microscope).

Results

Insertion of the shunts was always effective in preventing myocardial ischemia without ECG modification, regional motion abnormalities assessed by means of 2-dimensional echocardiography, or both. In all cases the shunts ensured a bloodless operative field.

At histologic and electron microscopic examinations, the specimens used as controls showed normal endothelial layers without mechanical damage to the coronary artery wall. By contrast, the insertion of the endoluminal shunts always determined partial endothelial denudation of the coronary artery wall because of the unavoidable brushing effect on the vessel lumen caused by the insertion maneuver (grade II, Figure 3).

In one case the insertion of the intraluminal shunt led to catastrophic disruption of the coronary artery wall caused by the presence of an atherosclerotic plaque, even if the shunt insertion was not troublesome (grade III; Figure 4, A and B).

Figure 4. A, Sample of anastomotic site exposed to the catastrophic action of the inserted intraluminal shunt. Note the knocked-down coronary wall with the vessel disruption. (Hematoxylin and eosin staining; original magnification 5 $\times$.) B, At scanning electron microscopy, the original artery wall is destroyed with exposure of adventitial layers to the blood flow, and red cells with platelets are in direct contact with denuded collagen fibers (arrows).

Discussion

This study investigates the mechanical effects induced by shunting devices routinely used during beating-heart surgery in a human atherosclerotic disease coronary artery model.

In OPCAB surgery high-quality anastomoses are crucial for graft patency,⁵ and shunts are extremely effective in granting an optimal bloodless operative field. Additionally, the intracoronary shunt insertion, avoiding blood flow interruption, might prevent intraoperative ischemia and its consequences (arrhythmia, S-T tract modification, and functional impairment of the left ventricle). However, the assumption of clinical safety and the minimal effects on the coronary artery wall integrity of the shunt insertion might be overestimated.

In our view the animal model, which has healthy and large coronary arteries, appears to be inadequate for testing such devices.^{6,7}

A recent study⁸ focused on a human model and used the postischemic dilated heart during heart transplantation to test occlusive devices of target vessels. In this model the coronary arteries have features identical to those of atherosclerotic coronary arteries of patients undergoing coronary artery bypass. In appearance, the method of Hangler and colleagues⁸ corresponds to the real clinical setting of patients with coronary artery disease undergoing OPCAB. Nevertheless, at careful observation, this method is unreliable because the heart is unloaded during CPB. This modifies chamber sizes, systolic-diastolic pressure, and left ventricular wall stress and introduces a fundamental bias in interpreting and discussing the results. By contrast, in our study we applied the devices to the beating heart for 10 minutes before starting CPB, thereby exactly mimicking the real OPCAB scenario.

To the best of our knowledge, no authors have previously reported histologic and ultrastructural coronary artery wall findings observed after insertion of an intracoronary shunt in coronary arteries affected by atherosclerotic disease. According to our results, insertion of intraluminal shunts always led to endothelial denudation (grade II) with collagen fibers exposed to blood flow and sometimes determined catastrophic endothelial damage (grade III) in the presence of diffuse calcified coronary artery vessels. This can lead to coronary disruption.

The loss of endothelial cell coverage might be important clinically because regenerated endothelium presents a selective dysfunction that might accelerate the occurrence of vasospasm. Additionally, the loss of coronary artery wall integrity might accelerate the atherosclerosis process.^{9,10}

In this study we have tested the shunt by using only one model of shunt, which is currently recognized to be stiff and blunt and therefore currently avoided by many surgeons. We are continuing the investigation with several flexible devices available on the market, and the results will be the object of a further study.

However, the major inference from our study is the clear evidence that, in minimally invasive coronary artery bypass grafting, the underhand complications of shunting devices on coronary

endothelial integrity might be avoided through full knowledge of the effects of the devices and their careful manipulation. The results of our study are the first real human evidence that the use of a shunt at diseased sites of vessels can cause significant damage, and surgeons need to be clearly aware of this potential hazard.

References

1. Cartier R, Brann S, Dagenais F, Martineau R, Couturier A. Systematic off-pump coronary artery revascularization in multivessel disease: experience of three hundred cases. *J Thorac Cardiovasc Surg.* 2000; 119:221-9.
2. Bull DA, Neumayer LA, Stringham JC, et al. Coronary artery by-pass grafting with cardiopulmonary bypass versus off-pump cardiopulmonary bypass grafting: Does eliminating the pump reduce morbidity and cost? *Ann Thorac Surg.* 2001;71:170-5.
3. Lucchetti V, Capasso F, Caputo M, et al. Intracoronary shunt prevents left ventricular function impairment during beating heart coronary revascularization. *Eur J Cardiothorac Surg.* 1999;15:255-9.
4. Ip JH, Fuster V, Badimon L, Badimon J, Taubman MB, Chesebro JH. Syndromes of accelerated atherosclerosis: role of vascular injury and smooth muscle cell proliferation. *J Am Coll Cardiol.* 1990;15:1667-87.
5. Gundry SR, Romano MA, Shattuck OH, et al. Seven-year follow-up of coronary artery bypasses performed with and without cardiopulmonary bypass. *J Thorac Cardiovasc Surg.* 1998;115:1273-7.
6. Perrault LP, Menasché P, Wassef M, et al. Endothelial effects of hemostatic devices for continuous cardioplegia or minimally invasive operations. *Ann Thorac Surg.* 1996;62:1158-63.
7. Perrault LP, Menasché P, Bidouard JP, et al. Snaring of the target vessel in less invasive bypass operations does not cause endothelial dysfunction. *Ann Thorac Surg.* 1997;63:751-5.
8. Hangler HB, Pfaller K, Antretter H, Dapunt OE, Bonatti JO. Coronary endothelial injury after local occlusion on the human beating heart. *Ann Thorac Surg.* 2001;71:122-7.
9. Shimokawa H, Aarhus L, Vanhoutte PM. Porcine coronary arteries with regenerated endothelium have a reduced endothelium-dependent responsiveness to aggregating platelets and serotonin. *Circ Res.* 1987; 61:256-70.
10. Cartier R, Pearson PJ, Lin PJ, Schaff HV. Time course and extent of recovery of endothelium-dependent contractions and relaxation after direct arterial injury. *J Thorac Cardiovasc Surg.* 1991;102:371-7.