Cardiotomy suction, but not open venous reservoirs, activates coagulofibrinolysis in coronary artery surgery

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Objectives: Closed and miniaturized cardiopulmonary bypass circuits, which eliminate cardiotomy suction and open venous reservoirs with a reduced priming volume, have been reported to be advantageous. We comparatively examined the respective contribution of cardiotomy suction and open venous reservoirs to perioperative activation in coagulofibrinolysis and inflammation systems, with identical conditions of priming volume and anticoagulation.

Methods: A total of 75 consecutive coronary artery bypass grafting procedures were performed using 1 of the following 3 cardiopulmonary bypass circuits under identical conditions of priming volumes, heparin coating, and protocols of anticoagulation and transfusion, as follows: a circuit with an open venous reservoir and cardiotomy suction (open group, n = 25), a circuit with an open venous reservoir without cardiotomy suction (nonsuction group, n = 25), or a circuit without either (closed group, n = 25). Blood samples were collected at 8 points up to the first postoperative morning.

Results: The thrombin-antithrombin III complex, fibrinogen degeneration products, D-dimer, plasmin-α2 plasmin inhibitor complex, and plasminogen activator inhibitor-1 levels were significantly greater in the open group than those in the other 2 groups ($P < .0001$, for all markers). The C3a and interleukin-6 levels were similar among all the groups. The incidences of perioperative transfusion and postoperative bleeding were increased and the early graft patency rate of saphenous veins was lower in the open group than those in the other 2 groups.

Conclusions: Cardiotomy suction, but not open venous reservoirs, causes perioperative coagulofibrinolysis activation, although neither affects the inflammation system. The use of cardiotomy suction needs to be examined further in association with postoperative PAI-1 elevation and early vein graft occlusion. (J Thorac Cardiovasc Surg 2011;141:1289-97)

Closed and miniaturized cardiopulmonary bypass (CPB) circuits, which have recently been applied, particularly in coronary artery bypass grafting (CABG), appear to be a fundamental and important innovation in CPB technology.[1-7] These circuits are not equipped with cardiotomy suction or open venous reservoirs and, consequently, enable extreme miniaturization of the circuit. In clinical studies, these circuits have been shown to improve the clinical outcomes and suppress various perioperative plasma markers compared with conventional open circuits[1,2] and, furthermore, have been comparable to those found during off-pump CABG (OPCAB).[4,5] The advantageous outcome of closed and miniaturized circuits is supposedly derived from the following 3 components: the elimination of cardiotomy suction, the elimination of open venous reservoirs, and the extreme miniaturization of the circuit.

The activation of coagulofibrinolysis and inflammation systems are important issues in cardiac surgeries using CPB and lead to various adverse outcomes, particularly in high-risk patients.[8,9] Thrombin is a key enzyme in these systems,[9] and anticoagulation with heparin is essential during CPB.[10] Even with standard heparinization and heparin-coated circuits, thrombin generation is inevitable in cardiac surgeries using CPB.[9-11] Cardiotomy suction and open venous reservoirs have been demonstrated to contribute to the activation of the coagulofibrinolysis and inflammation systems[12-16]; however, the contribution of each has not yet been comparatively examined.[7] In the use of closed and miniaturized circuits, highly sophisticated miniaturization can also affect the coagulofibrinolysis and inflammation systems owing to the prevention of hemodilution and a reduction of the blood–circuit contact area.[17]

In the present prospective clinical study, we compared the respective effect of cardiotomy suction and open venous reservoirs on the coagulofibrinolysis and inflammation systems, setting identical conditions of priming volumes,

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Abbreviations and Acronyms

ACT = activated clotting time
APTT = activated partial thromboplastin time
AT = antithrombin III
CABG = coronary artery bypass grafting
CPB = cardiopulmonary bypass
FDPs = fibrinogen degeneration products
IL = interleukin
OPCAB = off-pump CABG
PAI-1 = plasminogen activator inhibitor-1
PIC = plasmin-α2 plasmin inhibitor complex
PRBCs = packed red blood cells
PT = prothrombin time
TAT = thrombin-antithrombin III complex

heparin coating, and protocols of anticoagulation and transfusion.

MATERIALS AND METHODS

Study Population

Between October 2006 and June 2009, 75 elective, primary, isolated CABGs at Osaka City University Hospital (Osaka, Japan) were prospectively randomized for use of 1 of the following CPB circuits: a circuit with an open venous reservoir and cardiotomy suction (open group, n = 25), a circuit with an open venous reservoir without cardiotomy suction (nonsuction group, n = 25), or a circuit without either (closed group, n = 25). The exclusion criteria included single-vessel disease, preoperative administration of heparin, coagulofibrinolysis disorders, renal or liver dysfunction, and inflammatory or malignant disease. Anticoagulants and antiplatelet agents were discontinued at least 7 days before surgery. The institutional review board at our institution approved the study, and all patients provided informed consent for the study.

Study Protocols

Anesthetic protocol and operative procedure. A standardized anesthetic protocol was used. No premedication was administered. Anesthesia was induced with intravenous midazolam and fentanyl and maintained with fentanyl, propofol, vecuronium bromide, and inhalation of sevoflurane. No patient received perioperative corticosteroids, aprotinin, or tranexamic acid.

A median sternotomy approach was used for all patients. The left internal thoracic artery and other graft materials were harvested simultaneously. After systemic heparinization, CPB was established with an arterial cannula to the ascending aorta and a 2-stage cannula through the right atrial appendage. To avoid air entrainment and leaky bleeding through the purse-string suture around the venous cannula, an extra tie was added around the insertion site of the atrial appendage. A left ventricular vent was inserted through the right upper pulmonary vein. During CPB, a nonpulsatile flow of 2.4 L/min/m^2 body surface area was maintained under moderate systemic hypothermia (rectal temperature, 32°C), and the mean arterial pressure was controlled within the range of 50 to 80 mm Hg.

After aortic crossclamping, identical cold blood cardioplegia was administered through the aortic root, followed by intermittent administration in the antegrade and retrograde directions. All proximal, as well as distal, coronary anastomoses were constructed using hand suturing during 1 aortic clamping. After termination of CPB and achievement of hemostasis, all patients were postoperatively admitted to the intensive care unit. The blood from the postoperative chest drains was discarded.

CPB circuits and suction strategy. The circuits in all groups consisted of a hollow-fiber membrane oxygenator (Affinity NT Oxygenator, CB511), a centrifugal blood pump (Bio-Pump, CBBPX80), and an arterial filter (Affinity Arterial Filter, CB351; all 3 components from Medtronic Cardiac Surgery, Minneapolis, Minn). All the components of the circuits in all groups were coated with covalently-bonded heparin (Carmeda BioActive Surface, Carmeda AB, Upplands Vasby, Sweden). The circuits in all the groups were primed with identical fluid (1300 mL), a mixture of 1000 mL lactated Ringer's solution, 200 mL mannitol (200 mg/mL), and 100 mL sodium bicarbonate (84 mg/mL). The differences in the circuits and suction strategy among the groups are described below and shown in Figure 1.

Open group. For the open group, the circuit contained a hard-shell venous reservoir (Affinity CVR Reservoir, 61399409462, Medtronic Cardiac Surgery). During systemic heparinization, pericardial blood was collected in the reservoir through cardiotomy suction. At all other times, the pericardial suction blood was processed by an autotransfusion cell-saving device (Electa with a bowel of BT225, Dideco, Sorin Group, Mirandola, Italy) and transfused.

Nonsuction group. For the nonsuction group, the circuit contained a hard-shell venous reservoir just as in the open group. Cardiotomy suction was not used, and a cell-saving device was applied throughout the operation.

Closed group. For the closed group, the circuit was designed using the basic structure of the Medtronic Resting Heart System (Medtronic Cardiac Surgery). The circuit eliminated an open venous reservoir and was equipped with a collapsible soft reservoir in the side of venous return for temporary blood retention. Cardiotomy suction was not used, and a cell-saving device was applied throughout the operation.

Anticoagulation and blood product use. The protocols for anticoagulation and blood product use were similar for all groups. Before aortic cannulation, all patients received 300 IU/kg bovine heparin. The celite-activated activated clotting time (ACT; Hemochron 801, International Technidyne Corp, Edison, NJ) was used to guide heparin anticoagulation, and the ACT was maintained at 400 seconds or more by the additional administration of heparin, as required. After the termination of CPB, the heparin was neutralized with 4.5 mg/kg of protamine.

Packed red blood cells (PRBCs) were transfused to maintain the hematocrit at 23% or more during CPB and 28% or more after the termination of CPB.

Data collection and measurements. Blood samples were obtained from a radial arterial catheter or the arterial side of the CPB circulation at the following 8 observation times: T1, before the induction of anesthesia; T2, 1 hour after the initiation of CPB; T3, 10 minutes after aortic declamping; T4, 5 minutes after protamine administration; T5, T6, and T7, 1, 3, and 6 hours after the termination of CPB, respectively; and T8, the first postoperative morning. The hematocrit and platelet count were immediately measured using an automated cell counter (MAXM-Retic, Beckman Coulter, Inc, Fullerton, CA). The other citrated samples were centrifuged at 3000*g* for 10 minutes, and the plasma was stored at –80°C for later analysis.

The coagulation, fibrinolysis, and inflammation markers were measured using reagents according to the manufacturer's instructions, as follows. The activated partial thromboplastin time (APTT) was measured using the Langdell method and Hemosil SynthASil (Instrumentation Laboratory, Bedford, MA). The prothrombin time (PT) was determined using the 1-stage method of Quick using Thromborel S (Sysmex Corp, Hyogo, Japan). Antithrombin III (AT) was determined using the absorbance photometry method for chromogenic substrates using Testzyme S ATIII (Sekisui Medical Corp, Tokyo, Japan). The plasma thrombin-antithrombin III complex (TAT) levels, which reflect the amount of thrombin generated in the circulating blood, were measured using an enzyme-linked immunoassay method and TAT [S] (TFB Inc, Tokyo, Japan). The plasma levels of fibrinogen degeneration products (FDPs) were determined using an absorbance photometry method for latex turbidimetry tests and Nanopia P-FDP (Sekisui Medical Corp, Tokyo,

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