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## Heparin Management for Cardiopulmonary Bypass in a Patient with Disseminated Intravascular Coagulation

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Severe clotting disorders are seen in patients with spontaneous or post-traumatic bleeding. When acute open heart surgery with cardiopulmonary bypass (CPB) is required in such patients, a modification of the commonly administered heparin loading dose based on body weight or surface area may be appropriate.

### REPORT OF A CASE

A 39-yr-old, 75-kg man was admitted to the emergency room because of an immitigable oozing of blood out of a fistula in the fifth intercostal space of his left chest wall. The patient had been bleeding for several hours. He denied preceding trauma. His medical history included the resection of a post-traumatic cardiac left ventricular aneurysm 7 yr ago, followed by three myocardial and one pulmonary infarctions. The patient had been taking no other medication than the occasional use of a nitroglycerine spray for anginal pain.

On physical examination, a fine pulsating blood stream was coming out of the fistula. The arterial blood pressure was 120/80 mmHg, heart rate 120 bpm, hematocrit 40%, serum potassium 2.9 mEq/l, serum sodium 139 mEq/l, serum lactate 12.7 mmol/l, partial thromboplastin time (PTT) over 90 s, thrombin time (TT) over 90 s, platelet count 70,000 cells/mm<sup>3</sup>, fibrinogen 0.2 g/l, antithrombin III (AT3) 65%, and activated clotting time (ACT) over 1000 s. The chest roentgenogram revealed a cardiac left ventricular aneurysm that necessitated an immediate surgical intervention.

Following the insertion of a right radial arterial line and a central venous catheter via the right internal jugular vein, anesthesia was induced with 0.5 mg fentanyl, 2 mg flunitrazepam, and 8 mg pancuronium iv. The trachea was intubated uneventfully. Before institution of cardiopulmonary bypass (CPB), a bolus of 10,000 IU of heparin was injected iv. As ACT remained over 1000 s during CPB, no additional heparin or AT3 were administered. The ruptured cardiac aneurysm was resected. Total bypass time was 135 min. Weaning from bypass occurred easily. In the post-bypass course, 10 g of fibrinogen,† four units of fresh frozen plasma and five units of fresh blood were administered. After the substitution of blood products, blood clotting findings improved rapidly, as seen in table 1. Therefore, no protamine was given. Once transferred to the ICU, the patient received continuously

through a motor pump 250 IU/h of heparin. No major postoperative bleeding occurred. The trachea was extubated 8 h after the termination of the operation, and the patient made a good recovery. He was discharged 3 weeks later.

### DISCUSSION

The adequate level of anticoagulation with heparin is a prerequisite for the successful completion of surgical procedures employing extracorporeal circulation.<sup>1-2</sup> The administration of heparin before and during CPB is usually monitored by the ACT, allowing the determination of the adequacy of the heparin dose and of the coagulation level at regular intervals.<sup>3</sup> An ACT exceeding 400 s is considered safe for CPB.<sup>4</sup> The consequences of inadequate anticoagulation are either catastrophic thrombosis or low-grade activation of the coagulation cascade leading to the formation of microthrombi and uncontrollable consumption of platelets and blood clotting proteins.

ACT is a global test influenced by many factors. The variability of patients response to heparin is related to the heparin metabolism, as well as to other factors, such as shock, disseminated intravascular coagulation (DIC), AT3 deficiency, and previous heparin or streptokinase therapy.<sup>5-6</sup> Although several protocols for heparin administration have been proposed, an initial heparin loading dose calculated on body weight is commonly used. In our patient, a severe clotting disorder due to the cardiac bleeding had taken place. The pathological findings, *i.e.*, low fibrinogen and elevated PTT and TT (table 1), indicated a DIC in the early consumption phase. DIC had resulted from prolonged bleeding and tissue damage. Released tissue thromboplastin had activated the extrinsic clotting system, while vascular surface injury activated platelets and the intrinsic clotting system. We were pleased to note that no organ dysfunction, such as pulmonary insufficiency, renal failure, cerebral dysfunction, or diffuse hemorrhage, were yet apparent. The acute situation permitted no preoperative therapy to improve the blood-clotting variables.

Before CPB, a decision on the heparin management had to be taken: either no heparin administration, or the administration of the full or reduced heparin loading dose calculated on body weight. The preoperative level of ACT of over 1000 s, as well as the low fibrino-

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† Fibrinogen (Immuno) is registered by national health authorities in Austria and W. Germany. It is equivalent to cryo-precipitate.

TABLE 1. Intraoperative Laboratory Values and Blood and Blood Products

	Time							
	8PM	9PM	10PM	11PM	12AM	1AM	2AM	3AM
Activated clotting time(s)	>1000	>1000			265		165	
Partial thromboplastin time(s)	>90	>90				78		32
Thrombin time(s)	>90	>90				>90		13.5
Fibrinogen (g/l)	0.2	0.12			1.29	1.65		2.38
Antithrombin III (%)	65					58		
Heparin (units)	10,000					250/h		
Fibrinogen (g)				10				
Fresh frozen plasma (units)				4				
Blood (units)				5				
Cardiopulmonary bypass								
Aortic clamping								

gen level of 0.2 g/l, suggested no need for heparin prior to CPB. At an ACT level of over 600 s, blood clotting is so poor that the endpoint of ACT, as well as many other tests of coagulation, are essentially impossible to determine. But, in our case, the omission of heparin could have worsened, or at least sustained, the actual DIC with further consumption of platelets and clotting factors.<sup>7</sup> On the other hand, it seemed dangerous to inject our standard heparin loading dose of 400 IU/kg body weight into this patient. Heparin activates platelets, and platelet counts often decrease after heparin infusion.<sup>8-9</sup> In the early consumption phase of acute and subacute DIC, heparin is used to inhibit fibrin formation and, thereby, the progression of DIC. Yet, if consumption continues, the heparin dose must be reduced by 25–50%. The possible benefit from heparin therapy had to be weighed against the risk of hemorrhage. Therefore, we decided to give our patient a reduced heparin dose, and injected only one-third of the common heparin loading dose calculated on body weight.

From a theoretical point of view, it may seem reasonable, in a similar case, to administer the full loading dose of heparin calculated on body weight in order to avoid the potential risk of inadequate anticoagulation during CPB. This risk being easily circumvented by the determination of the ACT at very short intervals, high heparin doses and their consequences are avoidable. The hazards of excess heparin anticoagulation are not so clearly defined, but it is possible that platelet hemostasis suffered from this derangement, and that the patient may have been more susceptible to bleeding.

In conclusion, a case has been presented in which a patient with substantial clotting disorder secondary to DIC needed emergency cardiac surgery and CPB for resection of a ruptured ventricular aneurysm. The pa-

tient's initial ACT exceeded 1000 s. A low heparin dose arbitrarily chosen was administered for CPB. The aim was to block further consumption of hemostatic components, keep up sufficient anticoagulation for CPB, and prevent hemorrhage and organ dysfunction. Although we do not know whether this patient survived because of the approach we used, we believe that, in this case, the low heparin dosage led to a favorable outcome.

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