

# Percutaneous Cardiopulmonary Support System for the Treatment of Fulminant Myocarditis

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## Abstract

We have developed a simple percutaneous cardiopulmonary support (PCPS) system. The circuit of the system is set up and sterilized beforehand and can be used promptly in an emergency situation. We used the PCPS on three patients with fulminant myocarditis and treated them successfully.

Case 1 was a 53-yr-old man and Case 2 was a 12-yr-old girl. They had failed to respond to treatment of their circulatory states with conventional therapy. Their circulatory states improved dramatically after application of PCPS with 2 L/min blood flow. They were weaned from PCPS after 107 and 227 hours use, respectively. Case 3 was a 37-yr-old woman who developed ventricular fibrillation which was resistant to drug therapy and cardioversion. After starting PCPS with 4 L/min blood flow, arrhythmia disappeared and her circulatory state improved. She was successfully weaned after 39-hour of PCPS.

Patients with fulminant myocarditis fall into circulatory collapse which is resistant to conventional therapy, but there is a possibility that cardiac function will recover after the acute phase of myocarditis. Therefore, PCPS can be the only treatment of the circulatory collapse of fulminant myocarditis, which is refractory to conventional therapy.

## Introduction

The advent of percutaneous cannulas has dispensed with surgical procedures such as the exposure of the vessels and thoracotomy and enabled rapid and expedient circulatory and / or respiratory support (percutaneous cardiopulmonary support, PCPS). Recently, supported angioplasty and cardiopulmonary resuscitation in cardiogenic shock and cardiac arrest have become the order of the day. PCPS has been applied broadly, particularly in cases calling for emergency treatment [1-3]. In our institution, we have been practicing PCPS with a system developed to rapidly cope with these cases.

We applied our system with satisfactory results to 3 patients with fulminant myocarditis, after having succumbed to cardiogenic shock. We herein present the courses

of these cases and our method of extra-corporeal circulation.

## Materials and Methods

Our PCPS system is comprised of a centrifugal pump (Bio Pump®) which formed a closed circuit with various membrane oxygenators shown in Table 1. These components were preassembled and sterilized to enable prompt treatment in emergency situations (Figure 1). Furthermore, with the exception of Case 1, Silastic Medical-Grade Tubing®, a silicone tubing, was chosen as the circuit tubing. Figure 2 is an overall view of the system in its clinical setting (Case 3). The percutaneous cannulas used are shown in Table 1. In all cases, cannulation was performed from the femoral artery and vein. Activated clotting time was maintained at 200 - 300 seconds by continuous infusion of heparin.

### Results 1. Course of cases (Table 2)

#### Patient No.1: A 53-year-old man.

Chills and fever had persisted since May, 1989, and on May 29th he was admitted to a local hospital after several episodes of syncope. During his hospitalization, complete atrioventricular block occurred and he was transferred to the intensive care unit (ICU) with a temporary pacing and catecholamine treatment.

Hospital course (Figure 3,4): Two days after admission to ICU, despite increased catecholamine doses, progression of respiratory failure and oliguria persisted. Thus intraaortic balloon pumping (IABP) was instituted due to hypotension. Although his hemodynamics briefly showed some improvement, circulatory insufficiency progressed further, leading to hepatic and renal failure, and disseminated intravascular coagulation. Fifteen hours after initiation of IABP, he entered a state of shock and PCPS was immediately instituted. Thereafter, he was free from multiple-organ failure, norepinephrine could be discontinued, and his hemodynamic state was stable. He was weaned 5 days (107 hours), after initiation of PCPS. The disturbed circumferential wall motion (left ventricular ejection fraction: 26%) observed at the initiation of PCPS recovered and the ejection fraction improved to 55%. He was discharged on

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- 540 Bio Console, External Motor Unit and Pump Head (BP-80), Medtronic Inc., (Bio-Medicus, Inc.), Minneapolis, MN
- Cat. No. 601-565, Dow Corning Corp., Midland, MI.

day 43 after admission and has by now returned to a normal life.

**Patient No.2: A 12-year-old girl.**

She experienced precordial pain upon waking on February 19th, 1990, and went to a local hospital. She had repeated episodes of fever and precordial pain, without symptomatic improvement. She was transferred to the emergency outpatient unit of our hospital on February 22nd. Systolic blood pressure was 48 mmHg, heart rate was 150 beats per minute. Cyanosis was found. The cardiothoracic ratio was 68%. Catecholamine was administered for control of the shock state.

Hospital course (Figure 5,6): After 8 hours, the blood pressure fell to 65/50 mmHg. Therefore, IABP was initiated immediately, but without satisfactory effect. Administration of norepinephrine failed to improve the hemodynamics and PCPS was instituted. In echocardiography, wall motion was virtually non-existent. To prevent multiple-organ failure and assist water management, continuous hemofiltration was performed concurrently. On day 6 after initiation of PCPS, wall motion at the papillary muscle level was confirmed on echocardiogram, and blood pressure became stable. She was weaned on day 10, after 227 hours of PCPS. On the 14th day, IABP was removed and on the 27th day, she was discharged from ICU. As of February 1991, she has been hospitalized because of a transition to dilated cardiomyopathy.

**Patient No.3 : A 37-year-old woman.**

She experienced exertion-related palpitation beginning on June 12, 1990, and went to a local hospital. ST Elevation was found on electrocardiogram, suggesting acute myocarditis. She was referred to our hospital on June 14th. The cardiothoracic ratio was 57%. Emergency catheterization revealed a normal coronary artery, decreased wall motion of the left ventricle (ejection fraction: 27%), and a high end diastolic pressure of 27 mmHg.

Hospital course (Figure 7,8): Administration of catecholamine helped maintain blood pressure but multifocal ventricular extrasystole and short run occurred frequently. Arrhythmias were poorly controlled and blood pressure fell. At 8 hours after admission, atrioventricular dissociation occurred and suddenly ventricular tachycardia, ventricular flutter and fibrillation developed. Tracheal intubation and cardiac massage were performed. Precordial thump and cardioversion were repeated. She did not respond to these treatment, and PCPS was therefore initiated. From intubation to commencing PCPS took 10 minutes. IABP was inserted to reduce left ventricular afterload [4]. To treat arrhythmias, drugs and cardioversion were repeated. However, she was unresponsive and ventricular tachycardia persisted therefore catecholamine was discontinued under PCPS. After 2 hours without direct treatment, sinus rhythm was restored spontaneously. Echocardiography revealed minimal wall motion. The IABP induced a pulsatic aortic pressure waveform, thus pul-

monary artery pressure waveform was flat. To prevent thrombus formation, catecholamine was reinstated, which produced remarkable responses with the appearance of aortic and pulmonary artery pulse wave patterns and recovery of wall motion of the heart on echocardiography. Thereafter, she had repeated episodes of ventricular tachycardia and extrasystoles. 15 hours after initiation of PCPS, arrhythmias disappeared and stable hemodynamics were obtained. She was weaned 37 hours after initiation of PCPS. After one month, the left ventricular ejection fraction was 79% and the cardiothoracic ratio was 48%. On the 59th hospital day, she was discharged.

**2. Estimation of extra-corporeal circulation**

In all cases, there was no gross evidence of thrombus in the circuit after use. In case 2, the cannula which acted as the sheath for IABP was used as the arterial cannula. A polyvinyl chloride tube, 1/4 inch in diameter, was used at the joint, and an overt thrombus was found locally.

The Bio-Pump head showed neither functional failure nor thrombus formation during continuous operation. In case 1, upon changing the oxygenator, the rotational speed of the Bio-Pump had to be increased by more than 50% because of a high pressure loss of the new oxygenator. The pump speed was increased from 1,980 RPM (flow rate 2.02 L/min) to 3,050 RPM (flow rate 1.90 L/min). After this change, a sharp increase of total bilirubin (T-Bil), lactate dehydrogenase (LDH), and red urine indicative of hemolysis were observed (Figure 4). In case 2, no increase of free hemoglobin levels in the serum and urine was found and the haptoglobin level was stable (Figure 6). However, after weaning both serum and urinary levels of free hemoglobin increased abnormally.

In all percutaneous cannulas, there was practically no bleeding from the site of insertions and the peripheral blood flow was well maintained. In removing the cannula, surgical suture of the vessel was performed. Although along with heparinization, bleeding was the problem. The blood transfusion during PCPS was necessary 1,120 mL, 11,800 mL and 7,020 mL, in case 1, 2 and 3, respectively. In cases 2 and 3, the main bleeding was from the insertion point of the intraaortic balloon, and in case 2, digestive tract bleeding increased.

## Discussion

In many cases of acute myocarditis, if the acute phase is overcome, the prognosis is good. Many lifesaving application of IABP even in cases where medical treatment has failed to improve the circulatory failure, have been reported by Akiyama et al. and others [5-9]. In regard to supported circulation for myocarditis, a life saving case has been reported by Splaingard et al [10]. Our cases 1 and 2 showed circulatory failures with resistance even to IABP, yet the most critical phase was overcome by circulatory support with PCPS. It is noteworthy that with the fatal arrhythmia of case 3, the use of PCPS for cardiopulmonary resuscita-

tion also resulted in recovery from the increased irritability of the heart muscle during circulatory support [11]. In acute myocarditis in which serious circulatory insufficiency or fatal arrhythmias occur, myocardial lesions are likely to recover in a short period. Thus, the critical phase of acute myocarditis can be overcome by PCPS, even if the condition seems refractory to drugs and IABP. Cardiopulmonary support using a percutaneous cannula enables rapid flow assistance in such cases.

On the other hand, hemolysis may be mentioned as one of problems in long-term cardiopulmonary support. While the drawing force of venous cannula and biochemical factors were also causes, case 1 developed hemolysis after we change to a new oxygenator with high pressure loss. In explanation of this, an increased fragility of red blood cells at the time of oxygenator change cannot be disregarded. However the sudden change of condition suggests that the increased afterloading of the Bio-Pump head resulted in an increased shear stress within the pump head which, in turn, caused a breakdown of red blood cells.

As for hemorrhage, there is no other way than the reduction of heparin. We are concerned about devices which do not require heparinization.

In summary, our percutaneous cardiopulmonary support system enables us to quickly institute a long-term flow assistance in an emergency state, and is especially effective in the treatment of fulminant myocarditis.

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## Questions and Comments

Q. How did you identify a good candidate to use this technique on as opposed to not using the technique on a candidate?

A. Emergent cases.

Jim Beavers, Corpus Christi, Texas

Q. Did you notice any problems with the distal limb? The cannulas were in — especially in the 12-year-old girl — a long period of time. Any ischemia or distal flow problems?

A. It may cause problems in long-term support.

Table 1. Summary of Percutaneous Cardiopulmonary Support

Patient No.	Arterial Cannula (Fr.)	Venous Cannula (Fr.)	Duration of PCPS (h)	Oxygenator	Duration (h)	Reference on oxygenator
1	PEBS <sup>a</sup> (16)	PEBS <sup>b</sup> (16)	107	· MERA EXCELUNG <sup>f</sup>	60	Serum leakage (36 hrs. later)
				· MERASILOX HSO 2.5 <sup>g</sup>	47	None
2	PERCOR Percutaneous Introducer <sup>c</sup> (for IABP) (11.5)	PEBS <sup>b</sup> (16)	227	· CAPIOX II 36 <sup>h</sup>	62	Serum leakage (40 hrs. later)
				· M - 2000 <sup>i</sup>	165	None
3	Femoral Cannula <sup>d</sup> (17)	Femoral Cannula <sup>e</sup> (21)	39	CAPIOX E <sup>j</sup> <Modified >	39	None

<sup>a</sup> Percutaneous Extracorporeal Bypass Set (Cook Inc., Bloomington, IN).effective length 9.5cm, ID 5.4mm, OD 6mm  
<sup>b</sup> Percutaneous Extracorporeal Bypass Set (Cook Inc., Bloomington, IN).effective length 28cm, ID 5.4mm, OD 6mm  
<sup>c</sup> PERCOR 11.5Fr. × 11" long percutaneous introducer (Datascope, Inc., Paramus, NJ).effective length (cut at) 13cm, ID 3.9mm, OD 4.3mm.  
<sup>d</sup> Femoral Cannula 96017( DLP, Inc.,Grand Rapids, MI). effective length 17.3cm, ID 4.5mm, OD 5.7mm  
<sup>e</sup> Femoral Cannula 96017( DLP, Inc.,Grand Rapids, MI). effective length 52.7cm, ID 5mm, OD 7mm  
<sup>f</sup> Senko Medical Trading, Tokyo, Japan; Microporous polypropylene hollow fiber membran oxygenator  
<sup>g</sup> Senko Medical Trading, Tokyo, Japan; Silicon hollow fiber membran oxygenator  
<sup>h</sup> Terumo, Inc.,Tokyo, Japan; Microporous polypropylene hollow fiber membran oxygenator  
<sup>i</sup> Shiley, Inc., Irvin CA; Pleated microporous polypropylene membran oxygenator  
<sup>j</sup> Terumo, Inc.,Tokyo, Japan; Microporous polypropylene hollow fiber membran oxygenator. <The reservoir and heat exchanger parts were removed.>

Table 2. Summary of Patients

Patient No.	Age	Sex	Weight (Kg)	Diagnosis	ECG	Other Artificial Support	Complications	Outcome
1	53	M	50	Viral (Coxsackie A,B or ECHO) or Idiopathic Myocarditis	ST elevation Complete A-VB <sup>a</sup> Sustained VT <sup>b</sup>	Pacemaker IABP <sup>d</sup>	None	Survived
2	12	F	38	Viral (ECHO) or Idiopathic Myocarditis	ST elevation Sinus tachycardia	Pacemaker IABP <sup>d</sup>	Digestive tract hemorrhage	Survived
3	37	F	56	Viral (Coxsackie A or Adeno) or Idiopathic Myocarditis	ST elevation Sustained VT <sup>b</sup> Vf <sup>c</sup>	IABP <sup>d</sup>	Hemorrhage (cannulation site of IABP <sup>d</sup> )	Survived

<sup>a</sup> atrioventricular block, <sup>b</sup> ventricular tachycardia, <sup>c</sup> ventricular fibrillation, <sup>d</sup> intraaortic balloon pumping

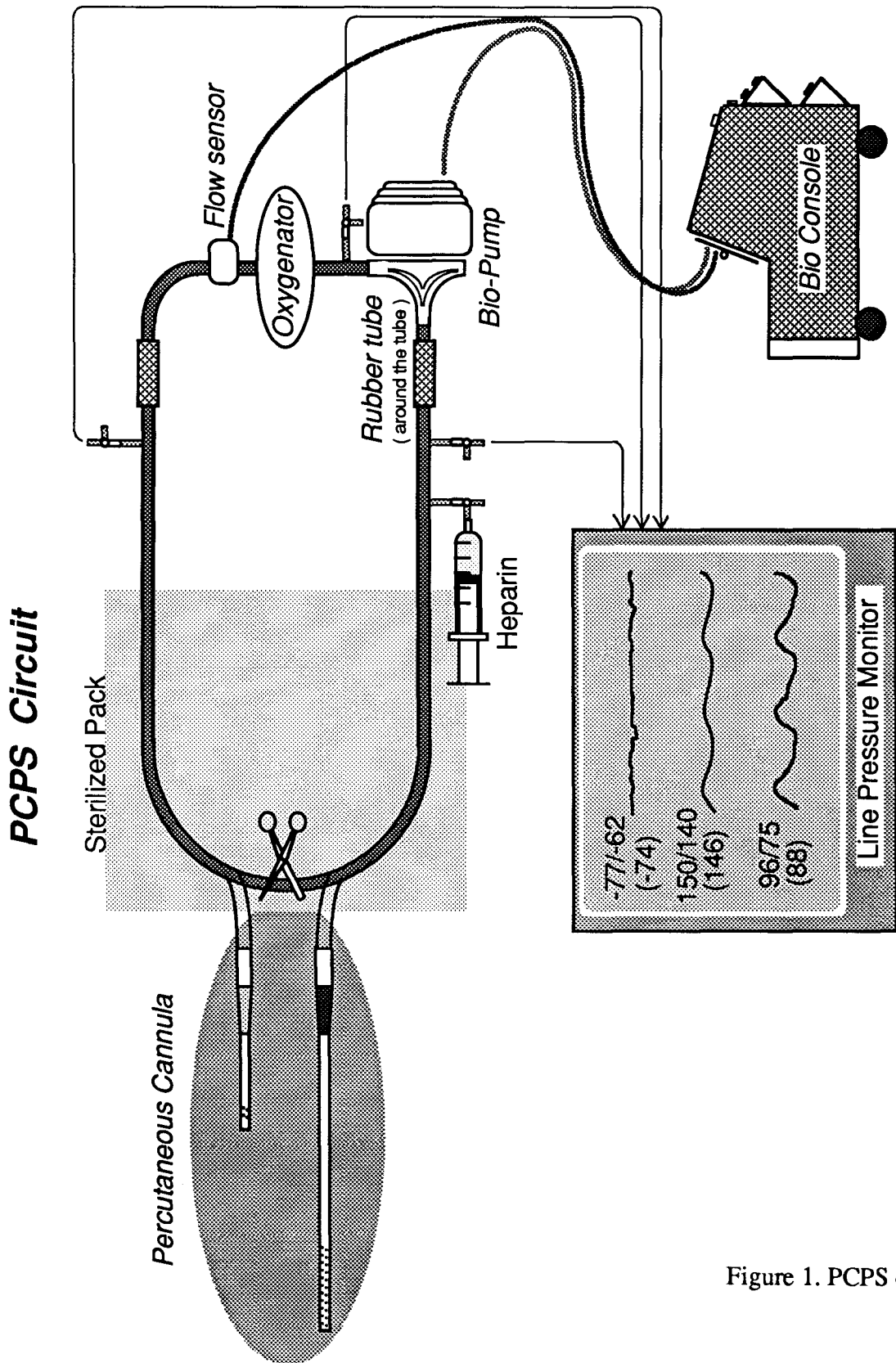


Figure 1. PCPS circuit

Figure 2. The System in Clinical Setting (Patient No. 3)

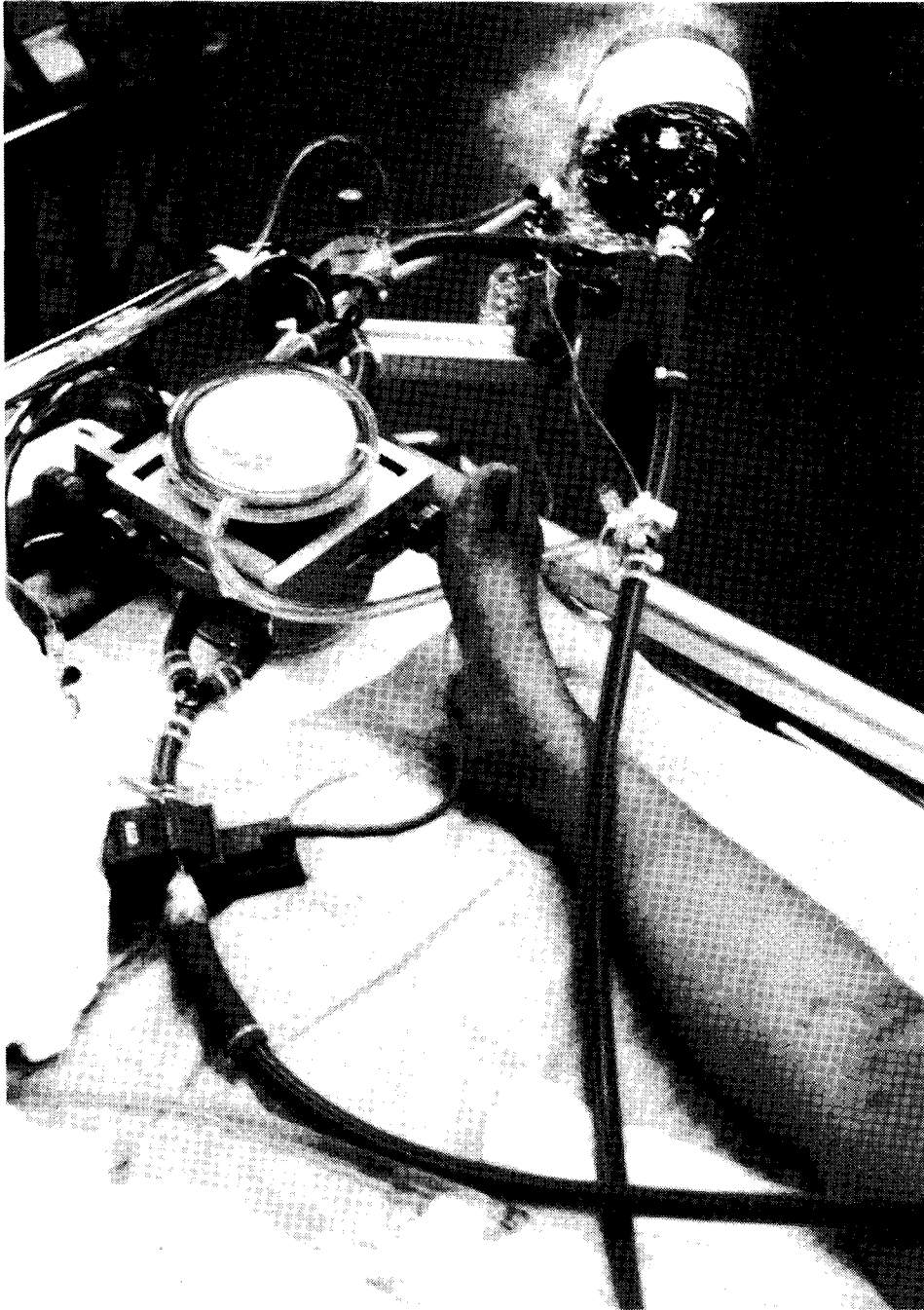
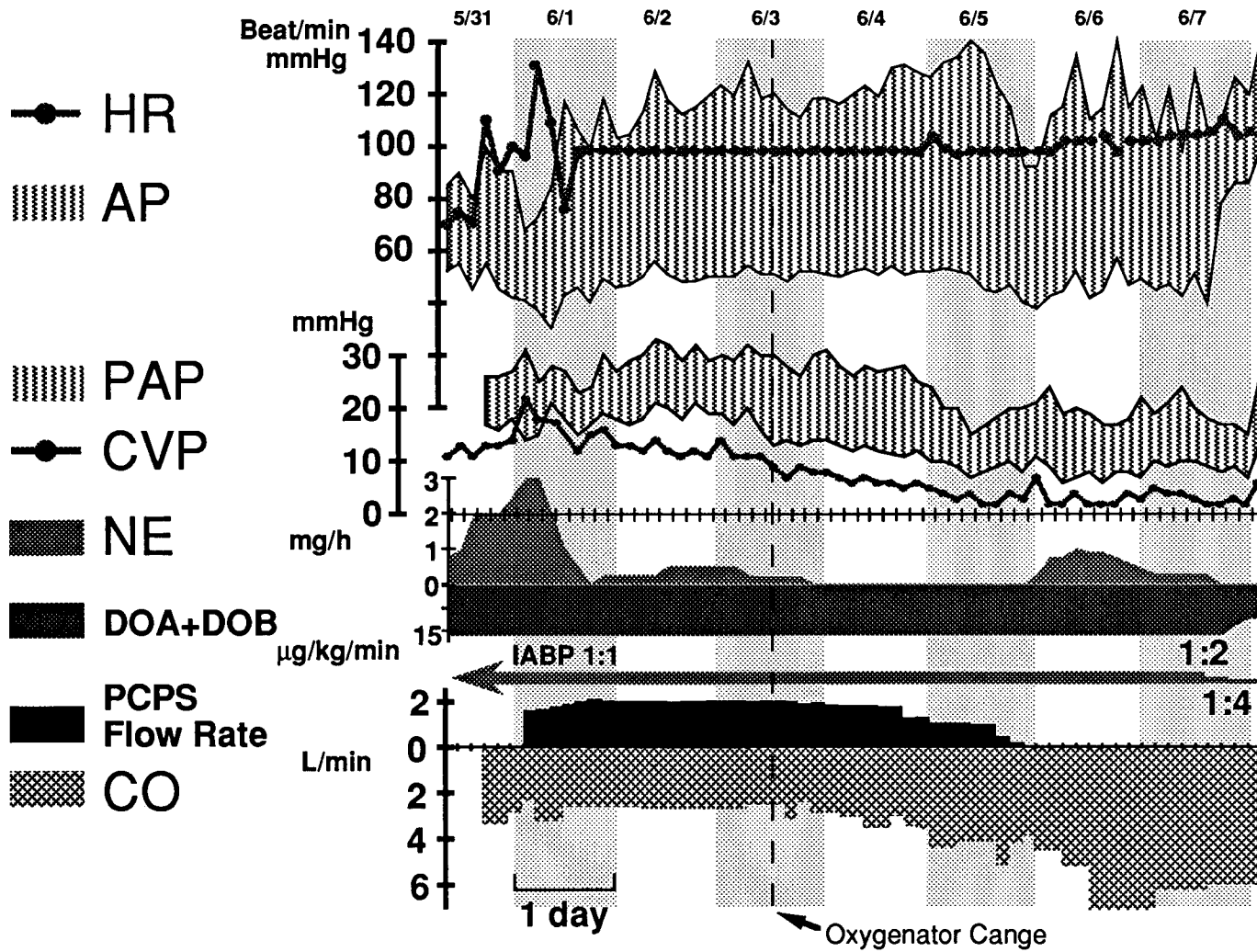
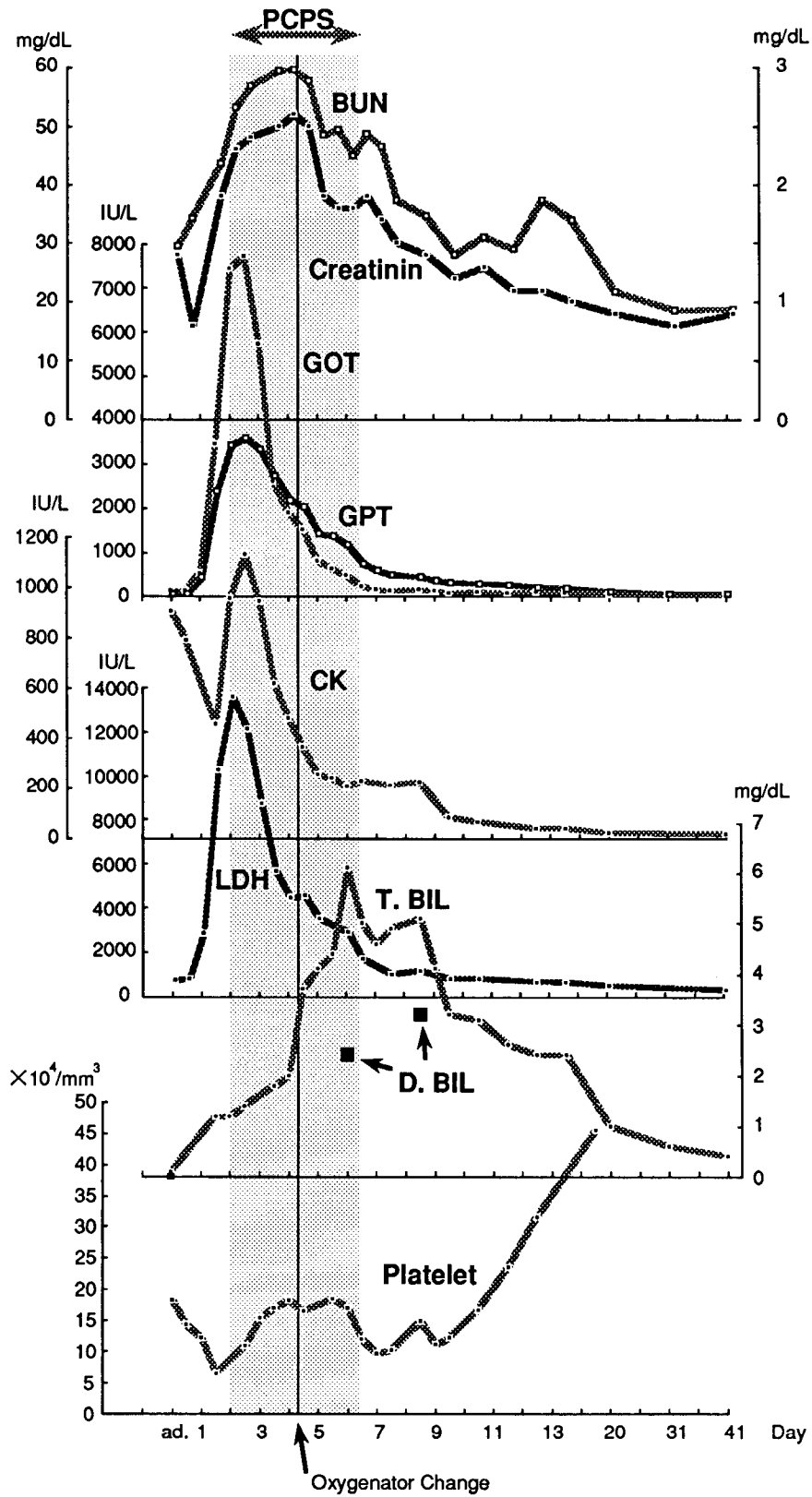


Figure 3. Changes of Hemodynamics (Patient No. 1)



HR = heart rate; BP = arterial blood pressure; PAP = pulmonary artery pressure; CVP = central venous pressure; NE = norepinephrine; DOA = dopamine; DOB = dobutamine; PCPS = percutaneous cardiopulmonary support; CO = cardiac output; IABP = intraaortic balloon pumping

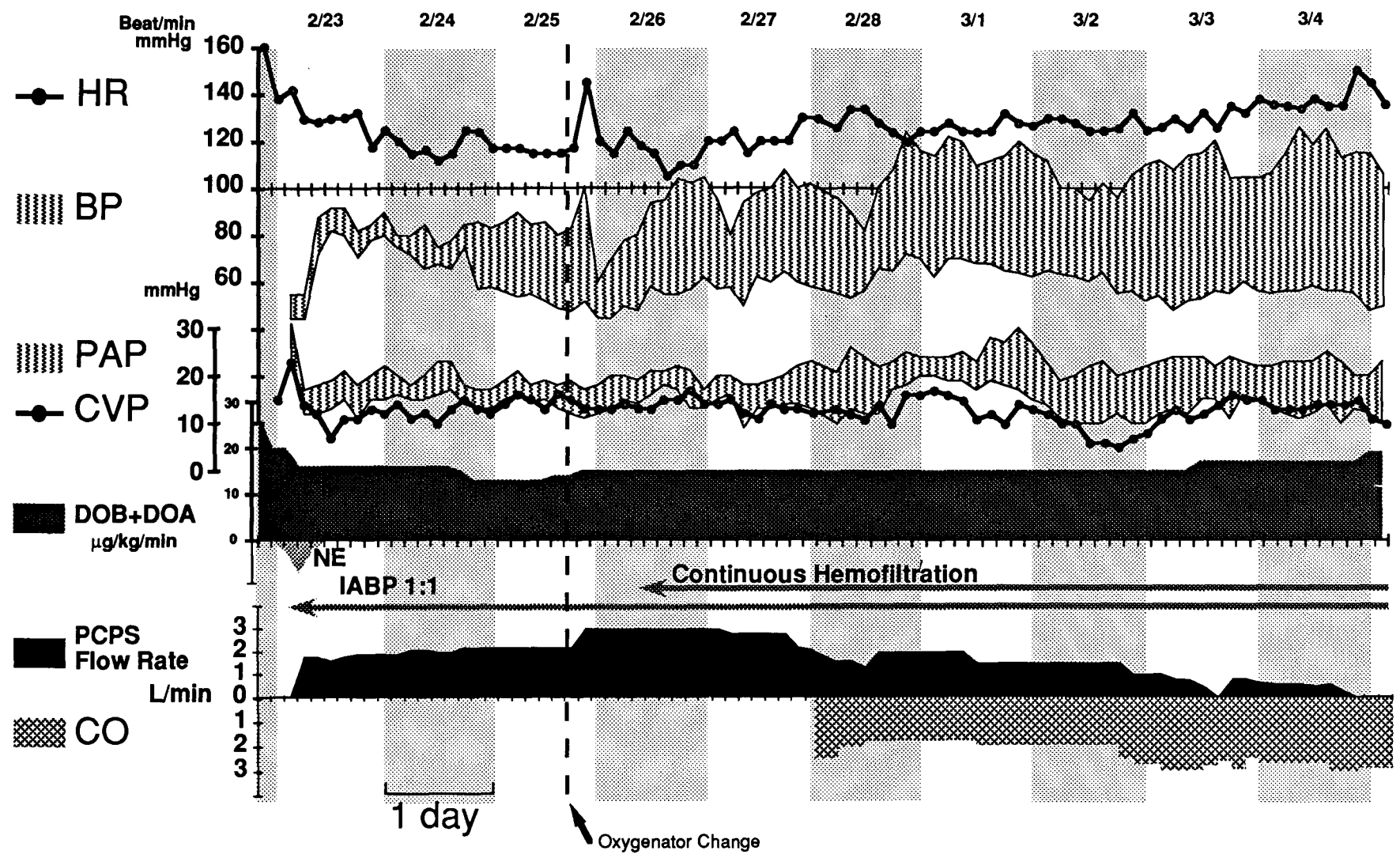
Figure 4. Laboratory Data (Patient No. 1)



BUN = blood urea nitrogen; GOT = glutamic oxaloacetic transaminase;  
 GPT = glutamic pyruvic transaminase; CK = creatin kinase; LDH =  
 lactate dehydrogenase; T.Bil = total bilirubin; D.Bil = direct bilirubin

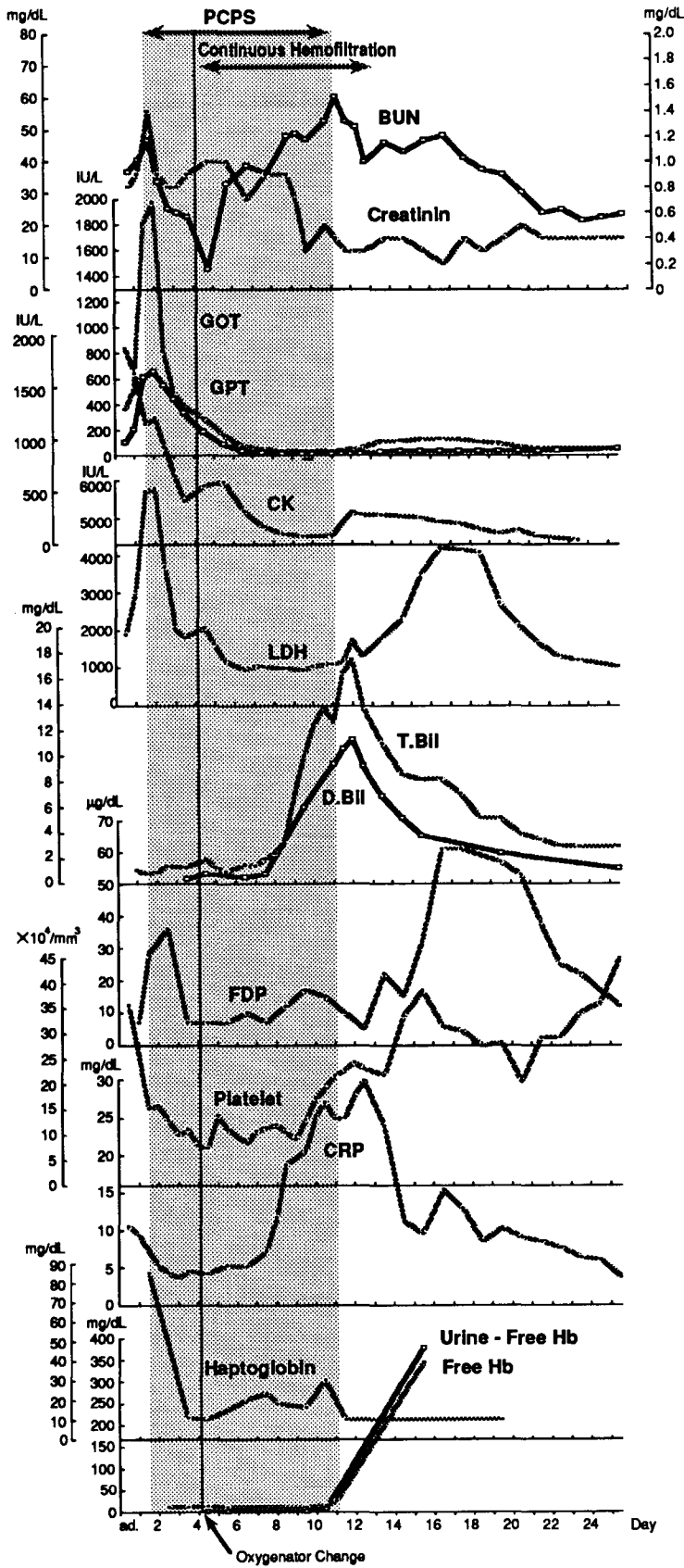


Figure 5. Changes of Hemodynamics (Patient No. 2)



HR = heart rate; BP = arterial blood pressure; PAP = pulmonary artery pressure; CVP = central venous pressure; DOA = dopamine; DOB = dobutamine; NE = norepinephrin; PCPS = percutaneous cardiopulmonary supprt; CO = cardiac output; IABP = intraaortic balloon pumping

Figure 6. Laboratory Data (Patient No. 2)



BUN= blood urea nitrogen; GOT= glutamic oxaloacetic transaminase; GPT= glutamic pyruvic transaminase; CK= creatin kinase; LDH= lactate dehydrogenase; T.Bil= total bilirubin; D.Bil= direct bilirubin; FDP= fibrinogen and fibrin degradation products; CRP= C-reactive protein; Hb= hemoglobin

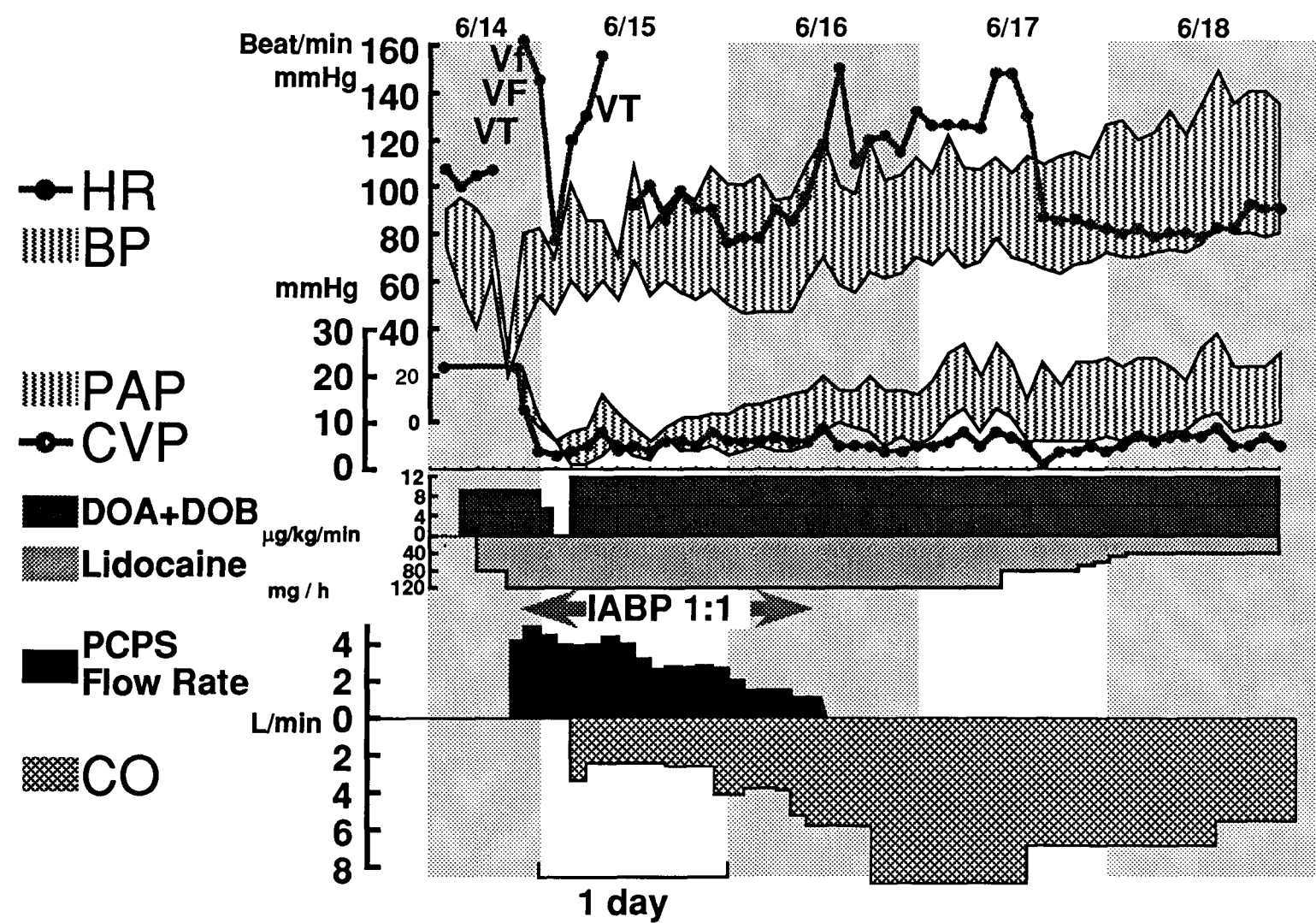
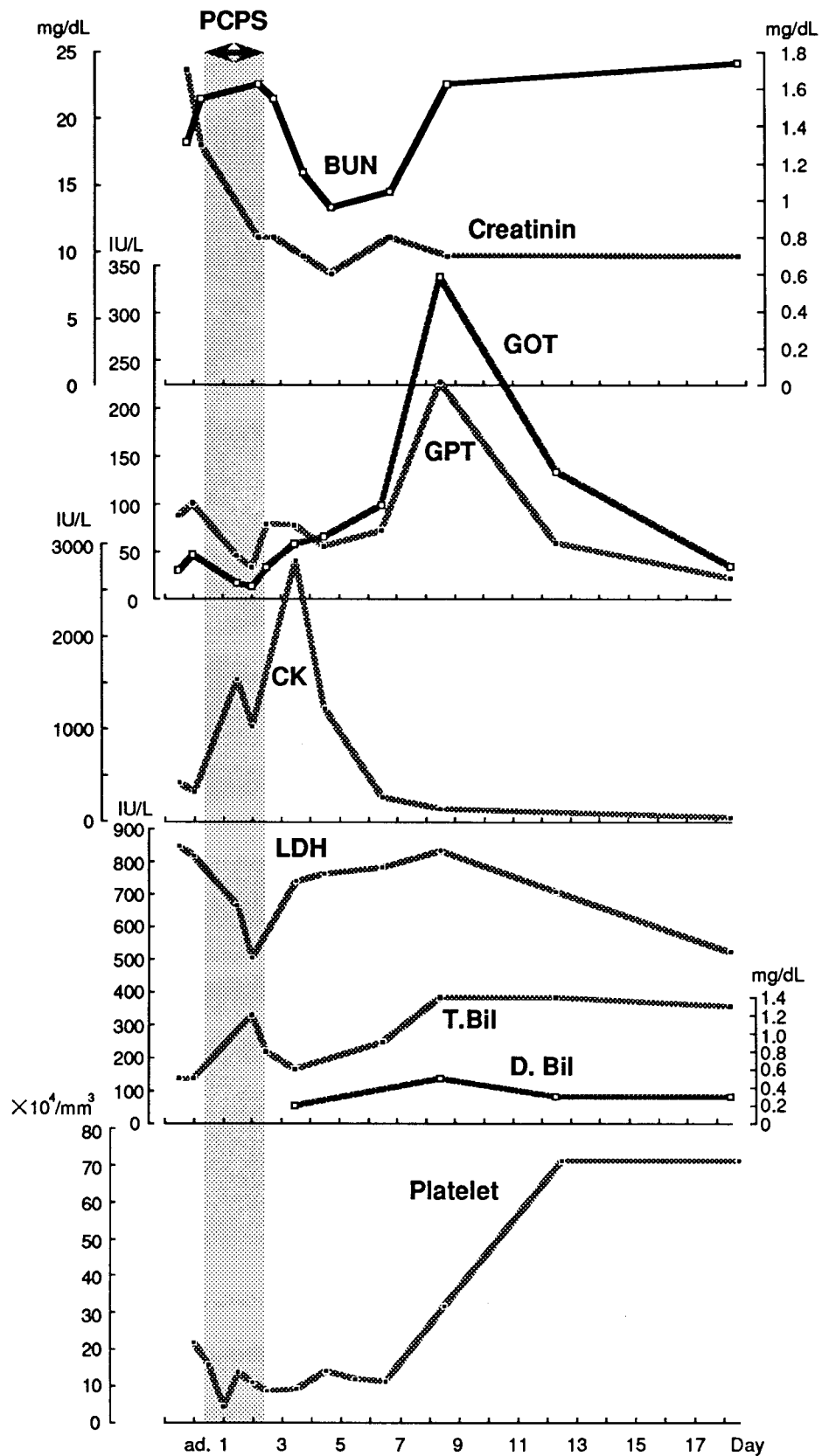


Figure 7. Changes of Hemodynamics (Patient No. 3)

HR = heart rate; BP = arterial blood pressure; PAP = pulmonary artery pressure; CVP = central venous pressure; DOA = dopamine; DOB = dobutamine; PCPS = percutaneous cardiopulmonary supprt; CO = cardiac output; VT = ventricular tachycardia; VF= ventricular fibrillation; IABP = intraaortic balloon pumping

Figure 8. Laboratory Data (Patient No. 3)



BUN = blood urea nitrogen; GOT = glutamic oxaloacetic transaminase;  
 GPT = glutamic pyruvic transaminase; CK = creatin kinase; LDH =  
 lactate dehydrogenase; T.Bil = total bilirubin; D.Bil = direct bilirubin