day, a metallic density forming loops in the cardiac silhouette was noted (fig. 2). The computed tomograph revealed the intravascular wirelike foreign body with both free ends in the hepatic vein and loops in the right pulmonary artery through the right intracardiac chambers.

On the 64th postoperative day, with informed consent, a 6-Fr pigtail catheter was inserted *via* the right femoral vein to remove the foreign body under fluoroscopic nonsurgical technique.[2] The right pulmonary arterial and intracardiac portions of the foreign body were easily dislodged into the inferior vena cava (IVC) by caudad traction of a part of the foreign body. This technique failed to remove the foreign body from the IVC, because both free ends were fixed hard to the hepatic venous wall. A retrieval catheter with forceps at the tip was inserted, which enabled us to grasp a portion of the foreign body and remove it entirely from the IVC. The foreign body was confirmed to be identical in length to an entire guidewire. Cardiac dysrhythmias and signs and symptoms indicative of pulmonary embolism did not develop throughout the postoperative course.

In conclusion, guidewire retrieval is a crucial step in a catheterization technique wherein a catheter is inserted over a guidewire.

Satoshi Akazawa, M.D.
Associate Professor
Department of Anesthesiology
Yasushi Nakaigawa, M.D.
Instructor
Department of Anesthesiology
Kunihisa Hotta, M.D.
Resident
Department of Anesthesiology
Reiju Shimizu, M.D.
Professor and Chairman
Department of Anesthesiology
Hiroshi Kashiwagi, M.D.
Research Fellow
Department of Surgery
Khoji Takahashi, M.D.
Instructor
Department of Radiology
Jichi Medical School
Minamikawachi-machi, Kawachi-gun
Tochigi-ken, 329-04, Japan

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Problems in Assessing the Effect of Nebulized Prostacyclin in Patients Whose Lungs Are Ventilated

To the Editor:—Pappert *et al.*[1] described the use of nebulized prostacyclin (PGI_2) *versus* inhaled nitric oxide in children with acute respiratory distress syndrome. We realize that the authors studied only three children and, as such, did not derive any conclusion from the study other than that PGI_2 may be an alternative to nitric oxide as a selective pulmonary vasodilator. From this paper, it is questionable whether the clinical benefits of PGI_2 may be offset by a rebound increase in pulmonary artery pressure. The baseline figures of pulmonary artery pressures did not greatly vary between increasing concentrations of nitric oxide. However, in two of the children, there was a marked increase in the pulmonary artery pressure during the interval between the different doses of PGI_2. It would be interesting to know, after each dose of PGI_2, how long the period was before measuring the baseline variables. We also wonder whether the variation of effects of different concentrations of PGI_2 may be due to the effect of positive end-expiratory pressure (PEEP) on the nebulizer. In Glasgow, we have been investigating benefits of nebulizing drugs in patients whose lungs are ventilated. We have had difficulty in obtaining an ultrasonic nebulizer that will perform well in the presence of PEEP. We wonder whether the authors assessed this before their study.

Dr. Gavin Fletcher
Senior Registrar
Dr. Malcolm Daniel
Senior Registrar
Department of Anaesthetics
Royal Infirmary
Glasgow G4 OSF
United Kingdom

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1996; 84:243

In Reply:—Fletcher and Daniel raise an important issue in the use of pulmonary vasodilators. In contrast to our experiences with inhaled nitric oxide, we have not been able to observe any rebound effect on pulmonary artery pressure (PAP) after discontinuation of nebulized prostacyclin (PGI_2).[1] This may be due to the fact that we did not administer PGI_2 for more than 30 min per concentration and vasodilator. At least in children 1 and 2, the study period was long enough to detect any rebound phenomenon-related increase in PAP. During the evaluation of the dose-response relationship, nitric oxide, up to a concentration of 10 ppm, did not decrease PAP to a significant degree, which is in accordance with our results.[1] PAP remained elevated 30–40 min after administration of PGI_2 in child 3 and increased in child 1 at 10 $ng \cdot kg^{-1} \cdot min^{-1}$. However, only the latter observation may give the impression of a rebound phenomenon. Positive end-expiratory pressure (PEEP) should not have influenced the performance of the Siemens ultrasound nebulizer used in our study. On one hand, PEEP was kept constant throughout the study; on the other hand, this nebulizer type is inspiration-triggered, so that the influence of PEEP should be negligible.

Dirk Pappert, M.D.
Rolf Rossaint, M.D.
Abteilung für Anaesthesiologie
und operative Intensivmedizin
HU Berlin, Virchow—Klinikum
D 13353 Berlin, Germany

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Presence of Tumor Necrosis Factor α and Tumor Necrosis Factor Soluble Receptors in Erythrocyte Concentrates

To the Editor:—The proinflammatory cytokines tumor necrosis factor α (TNF), interleukin-1 β (IL-1), and interleukin-6 (IL-6) are assumed to be important mediators for the pathophysiologic changes seen in trauma.[1] Treatment strategies affecting this trauma response may have implications for critically ill patients.

The occurrence of TNF[2] as well as IL-1[2,3] and IL-6[2,3] in platelet concentrates has been described. Stack et al[4] found IL-1 and IL-8 in erythrocyte concentrates, whereas IL-6 was not detectable. TNF was not investigated. Published data concerning cytokine concentrations in blood components yield no information with regard to coexisting soluble cytokine receptors possibly being able to modulate tentative effects of measured cytokines.

The concentrations of TNF and soluble TNF receptors I and II (sTNF-R I and sTNF-R II) in ten buffy coat-depleted erythrocyte con-