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**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 ( $\text{PGI}_2$ ).<sup>1</sup> This may be due to the fact that we did not administer  $\text{PGI}_2$  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.<sup>1</sup> PAP remained elevated 30-40 min after administration of  $\text{PGI}_2$  in child 3 and increased in child 1 at  $10 \text{ ng} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ . 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.

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## Presence of Tumor Necrosis Factor $\alpha$ and Tumor Necrosis Factor Soluble Receptors in Erythrocyte Concentrates

**To the Editor:**—The proinflammatory cytokines tumor necrosis factor  $\alpha$  (TNF), interleukin-1  $\beta$  (IL-1), and interleukin-6 (IL-6) are assumed to be important mediators for the pathophysiologic changes seen in trauma.<sup>1</sup> Treatment strategies affecting this trauma response may have implications for critically ill patients.

The occurrence of TNF<sup>2</sup> as well as IL-1<sup>2,3</sup> and IL-6<sup>2,3</sup> in platelet concentrates has been described. Stack et al<sup>4</sup> 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-