consumption.[3] Others have shown a rapid decline of endothelial ATP especially in the presence of heparin and this depletion occurs within a minute.[4] These effects on intracellular oxygen consumption and ATP availability may account for its direct effect on the cardiovascular system especially on the myocardium. Furthermore, blockade of all potential pathways that may cause hypotension and bradycardia, excluding the direct route, have been unsuccessful in blunting the cardiovascular effect of protamine.[5] These findings strongly indicate a direct influence of this drug on the myocardium.

Finally, protamine is also known to cause both anaphylactoid and anaphylactic reactions through activation of the complement system. Rapid boluses are contributory and should not exceed 50 mg over 10 min.[6] It is possible that the bolus administration of 20 mg was enough to induce such reactions although other indications confirming this as a mechanism of action are not offered.

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REPLY

We assessed possible mechanisms to explain the sudden onset of bradycardia in a cardiac transplant patient following a period of hypotension subsequent to the administration of protamine. We concluded that the change in heart rate could not be explained by cardiac reinnervation, as the patient had undergone transplantation only eight weeks previously. Rather, we suggested that the change was mediated by mechanisms intrinsic to the transplanted heart and extrinsic to the CNS. It was argued that withdrawal of adrenal sympathetic output secondary to stimulation of mechanoreceptors, while possible, was unlikely because of the precipitous change in heart rate. Other possibilities, which we could not discount, included cardiac ischaemia, activation of an intrinsic stretch-related mechanisms in the transplanted heart, and entrainment of the donor to recipient (innervated) atria. Dr. Olutolabi proposes an additional mechanism, which involves a direct effect of protamine on the myocardium of the transplanted heart. This possibility warrants consideration. We thank Dr. Olufolabi for his interest in our report, and for his insight into the possible mechanisms mediating the cardiovascular response produced by protamine in a cardiac transplant patient.

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Falsely reassuring pulse oximetry in the presence of severe hypoxia

To the Editor:

Pulse oximetry is a common means of objectively assessing the adequacy of oxygenation in the neonatal intensive care unit. We report the case of a one-day-old neonate with pulse oximeter saturation readings of >95% despite severe hypoxaemia.

This neonate experienced respiratory failure due to perinatal pneumonia (post-mortem diagnosis), and required mechanical ventilation, fluid and inotropic support. Monitoring included ECG, direct arterial blood pressure measurement and pulse oximetry (Nellcor N25). Clinically, the neonate was cyanosed despite ventilation with 100% oxygen, and a discrepancy was noted between the ABG PO_2 and the pulse oximeter saturation readings, the pulse oximeter persistently reading 95–100% despite ABG PO_2 levels in the 2.1–4.63 kPa range (see Table on page 1324). Pre and post ductal saturations were the same. The probe was attached sequentially to all four limbs with the same readings. Pulse oximeter and ECG heart rates always correlated and the pleythsmograph trace was of good quality. A second probe and pulse oximeter was tried and agreed with the first. Both pulse oximeters were used simultaneously and agreed. Subsequently both machines were found to be faultless by the hospital Bio-engineers.