In contrast, haemoconcentration may produce resistance to neuromuscular blocking drugs as observed in two children with congenital cyanotic heart disease (Fallot tetralogy; transposition of great vessels with VSD). In both children before operation, \(P_{aO_2}\) was 30 mmHg, Hct was 70 and plasma protein concentrations were within the normal range. Anaesthesia was induced with ketamine 2 mg kg\(^{-1}\), followed by pancuronium. Optimal relaxation necessary for tracheal intubation was only achieved with pancuronium 0.35 mg kg\(^{-1}\) in the patient with Fallot tetralogy and 0.40 mg kg\(^{-1}\) in the patient with transposition. These doses are about three or four times the usual doses.

Such resistance to pancuronium cannot be explained by the right-to-left intracardiac shunt, which can slow the rate of uptake of inhalation anaesthetics (Eger, 1974). Conversely, drugs administered i.v. will reach the systemic circulation rapidly via the shunt without traversing the lung. The unexpected resistance to i.v. pancuronium may be attributed to the excessive haemoconcentration associated with the cyanotic heart diseases. Haemoconcentration will produce a mirror-image effect of haemodilution. It increases the viscosity and peripheral resistance, resulting in a decrease of regional blood flow to muscles. It is also possible that pancuronium and perhaps other neuromuscular blocking drugs are directly bound to the structural stroma that envelops the RBCs, or to the anionic sites of the stroma-bound acetylcholinesterase enzyme. Increased binding to the high RBC mass can lead to a lower concentration of the unbound free drug. It may be concluded that smaller doses of neuromuscular blocking drugs are required in patients having normovolaemic haemodilution, while larger doses are needed with haemoconcentration.

**REFERENCES**
