RML with a mortality of 20%. Since CK plasma levels reflect the severity of muscle cell lysis, it has been suggested that the risk of developing ARF is closely related to CK values, with patients who present with CK > 20 000 IU litre⁻¹ being more at risk. However, there is no prognostic value for irreversible damage or mortality. Our case reinforces the hypothesis that a delay in diagnosis and treatment is more strongly related to the incidence of ARF than the peak CK level. Once RML is detected, vigorous fluid administration and forced diuresis seem to be the best measures to avoid fatal consequences. Padding pressure-points during surgery, positional changes, and strategies to reduce operative time, proposed as preventive measures, have not been proven to be effective in decreasing the incidence of RML. Routine serial postoperative CK monitoring should be carried out in patients at higher risk of RML. Early diagnosis of RML is the cornerstone of successful outcome of this increasingly recognized complication.

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![CK levels](image)

Fig 1 Follow-up of CK levels indicates a peak at 24 h after operation, which decreases rapidly in 4–5 days to the normal preoperative values. Creatinine levels remain at normal values during the entire postoperative period.

Overestimation of intrathoracic blood volume in a patient with atrial fibrillation and subsequent severely reduced atrial blood-flow

Editor—Erroneous measurements during haemodynamic monitoring can have a major impact on assessment and treatment of critically ill patients. We report the case of a 62-yr-old male with chronic atrial fibrillation after a mitral valve replacement 2 yr previously who presented with an acute intracranial haemorrhage. After craniotomy to evacuate a haematoma, the patient remained in a coma for several days requiring mechanical ventilation. He

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developed a ventilator-associated pneumonia with sepsis (temperature 39.1°C). White cell count was 19 200 mm⁻³. The central venous oxygen saturation (S⁰CVo₂) was 80% which we interpreted as reduced oxygen consumption in hyperdynamic sepsis; the PaO₂/FIO₂ ratio was 198. A PiCCO system was used for haemodynamic monitoring (5 F thermistor-tipped catheter, PiCCO, PV 2015L20, version 7.1, software version 1-273.6, Pulsion, Germany). This revealed a high intrathoracic blood volume index [ITBV index: 1632 (range of normal values: 850–1000 ml m⁻²)]. Despite clinical signs of volume depletion: heart rate 100 beats min⁻¹. The patient required norepinephrine administration [6 μg kg⁻¹ h⁻¹ (0.5 mg h⁻¹)] to maintain mean arterial pressure >60 mm Hg and urine output decreased to 0.4 ml kg⁻¹ h⁻¹. The systemic vascular resistance index (SVRI) was markedly decreased [1214 (1700–2400 dyn s cm⁻⁵ m⁻²)]. The cardiac index (CI) was 4.5 (3.0–5.0 litre min⁻¹ m⁻²). Transoesophageal echocardiography (TOE) showed normal valve function [transmitral peak to mean pressure difference (14/6 mm Hg) was within the normal range for this type of mechanical valve]. Marked spontaneous echoes in the atria and auricula, associated with extremely low blood flow, were documented (Vmax 20 cm s⁻¹). There were no echocardiographic signs of volume overload or intracardiac shunts. A conventional chest X-ray did not show any signs of volume overload.

Despite the PiCCO measurement, the patient received an initial infusion of colloid (Gelafundin 4%®) 500 ml. A repeated dose of 10 ml kg⁻¹ of body weight of Ringer’s acetate solution was given and after a total crystalloid solution load of 90 ml kg⁻¹, the continuous norepinephrine supply could be stopped after 48 h. The SVRI increased [1683 (1700–2400)]. At the end of the investigation, ITBV was 1761 (850–1000) and CI was 4.8 (3.0–5.0). After haemodynamic stabilization, the patient could be weaned from the respirator and was subsequently discharged from the intensive care unit.

During therapy, the PiCCO device depicted a markedly prolonged indicator transit time. We suspected an incompetence of the mechanical mitral valve to be responsible for a long transit time of the indicator resulting in a falsely elevated measurement of ITBV. Mitral incompetence can lead to overestimation of ITBV because the ITBV is determined by cardiac output and the mean transit time of the indicator.¹ In valvular incompetence, the thermodilution curve is affected by indicator regurgitation, resulting in a prolonged indicator decay time and may lead to an overestimation of global end-diastolic volume and ITBV. After valvular incompetence was ruled out by TOE, it was obvious that something else was responsible for the erroneous measurement. The TOE revealed almost static haemodynamic conditions in parts of the atria, as demonstrated by massive spontaneous echoes. It is likely that this finding had a major influence on the behaviour of the indicator in the blood after injection. Blood and indicator retention occurred in the atrium. This caused a temporary indicator deposit. As a consequence, the indicator proceeded to the subsequent blood circulation with a significant delay, despite the normal values for CI.

ITBV is a well-validated measurement¹ which is helpful in the monitoring and treatment of critically ill patients, especially in sepsis. Despite widespread use²–⁶ and popularity, the influences of specific cardiac diseases to measurement of the ITBV have not yet been investigated systematically.

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Ondansetron anaphylaxis: a case report and protocol for skin testing

Editor—Ondansetron hydrochloride is a selective serotonin (5-HT₃) receptor antagonist used as an antiemetic agent. Hypersensitivity reactions to ondansetron are rare but have been reported.¹–⁴ Both IgE-mediated⁵ and non-IgE-mediated¹ ³⁶ anaphylactic reactions to ondansetron have been rarely described as has isolated urticaria.⁴

A 44-yr-old female was given ondansetron, vecuronium, and propofol at induction for elective surgery. She immediately became hypotensive with an arterial pressure of 60/30 mm Hg. There was no accompanying urticaria, angioedema, or respiratory distress. She was given i.v. epi-nephrine, promethazine, hydrocortisone, and fluids, after