developed a ventilator-associated pneumonia with sepsis (temperature 39.1°C). White cell count was 19 200 mm\(^{-3}\). The central venous oxygen saturation (\(S\text{cv}_\text{o}_2\)) was 80% which we interpreted as reduced oxygen consumption in hyperdynamic sepsis; the \(P\text{a}_\text{ao}/F\text{io}_2\) 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\(^{-3}\)), despite clinical signs of volume depletion: heart rate 100 beats min\(^{-1}\). The patient required norepinephrine administration [6 \(\mu g\) 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 (\(V_{\text{max}}\) 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 measurement\(^1\) which is helpful in the monitoring and treatment of critically ill patients, especially in sepsis. Despite widespread use\(^2–6\) 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-HT\(_3\)) receptor antagonist used as an antiemetic agent. Hypersensitivity reactions to ondansetron are rare but have been reported.\(^1–4\) Both IgE-mediated\(^5\) and non-IgE-mediated\(^1,5\) 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
which she became normotensive. Mast cell degranulation was shown by an elevated serum tryptase level (48 μg litre⁻¹, normal 0–13.5 μg litre⁻¹) measured 4 h after the onset of hypotension. Her tryptase level was normal when measured 8 h after the onset of hypotension.

The patient was investigated subsequently with skin prick and intradermal testing to her induction medications. Ten normal controls were used to determine the irritant concentration of ondansetron. The patient had an absent response upon skin prick testing at a concentration of 2 mg ml⁻¹, but did demonstrate a positive wheal reaction on intradermal testing to ondansetron at a concentration of 0.02 mg ml⁻¹. None of the 10 controls produced a positive intradermal test reaction at this concentration. Five of the controls developed a positive reaction with intradermal testing at a concentration of 0.2 mg ml⁻¹ and nine of the controls at a concentration of 2 mg ml⁻¹. The patient did not demonstrate a positive reaction to vecuronium, propofol, or latex.

We suggest that a concentration of 0.02 mg ml⁻¹ be used for intradermal testing in the evaluation of suspected ondansetron allergy. This is in contrast to the only previous case report of a confirmed ondansetron IgE-mediated hypersensitivity, where a concentration of 0.2 mg ml⁻¹ was used to elicit a positive reaction.⁴ This concentration was found to be irritant in our control group.

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