institutional review board of Fudan University Shanghai Cancer Center. Prospective randomized research of neoadjuvant therapy will be performed soon. We are willing to share the results with our colleagues throughout the world and relate our experience for the treatment of esophageal cancer.

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METHYLENE BLUE, SEROTONERGIC SYNDROME, AND HEART TRANSPLANT
To the Editor:
Grubb and colleagues1 report a case of a 60-year-old man with history of nonischemic cardiomyopathy and end-stage heart failure who underwent placement of a left ventricular assist device (LVAD) and replacement of a mechanical aortic valve with a porcine prosthesis complicated by multiple driveline infections. The heart transplant was the last option. During the operation, the authors reported “episodes of hypotension during the extensive lysis of adhesions for [LVAD] removal. Intermittent boluses of phenylephrine were administered to maintain a sufficient mean arterial pressure.” Subsequently, a methylene blue (MB) 1 mg/kg bolus followed by continuous infusion of 0.5 mg/kg/hour was administered. Postoperatively, the patient presented signals of serotonin syndrome, presumably a consequence of an interaction between MB and antidepressant medications.

This report has two crucial points: the possibility of serotonin syndrome triggered by the association MB and antidepressants, and the routine use of MB to handle vasoplegia in the milieu of heart transplant. Kofidis and colleagues2 reported the first experience of vasoplegia treatment with MB after heart transplant and pointed out that this drug deserves attention because of its catecholamine-saving effect, thus preventing possible malperfusion. Kofidis and colleagues’report is unique in the literature regarding using MB to treat vasoplegia associated with heart transplant.

In 2009, my colleagues and I published an article3 discussing 15 years of questions, answers, doubts, and uncertainties regarding MB for vasoplegic syndrome treatment in heart surgery. In summary, our report shared that heparin and angiotensin-converting enzyme inhibitors are risk factors; in the recommended doses it is safe (the lethal dose is 40 mg/kg); the use of MB does not induce endothelial dysfunction; the MB effect appears in cases of nitric oxide up-regulation; MB is not a vasoconstrictor—by blocking the cyclic guanosine 3,5-monophosphate (cGMP) pathway it releases the cyclic adenosine monophosphate (cAMP) pathway, facilitating the norepinephrine vasoconstrictor effect; the most used dosage is 2 mg/kg administered intravenously as a bolus followed by the same continuous infusion because plasmatic concentrations strongly decay in the first 40 minutes; and there is a possible window of opportunity for MB’s effectiveness.3

To circumvent the morbidity and mortality associated with vasoplegic syndrome Grubb and colleagues1 implemented an intraoperative protocol that includes administration of MB for vasoplegic syndrome resistant to vasopressor drugs. Because serotonin syndrome only occurs in a small percentage of patients and is treatable with benzodiazepines and supportive care, and the possibility of serotonin syndrome is less than the risk of untreated vasoplegia and potential end-organ injury and graft loss, we have continued to include MB in the management of severe vasoplegic syndrome.

I congratulate Grubb and colleagues for reporting the association between MB and antidepressants that may cause serotonin syndrome. Although, to be honest, as a proponent of the use of MB4–5 the purpose of my letter is to suggest Grubb and colleagues publish their MB experience in heart transplant. It would be a valuable contribution to the cardiac surgery literature.

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