Reply to Mohite et al.

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We thank Mohite et al. [1] for their comments and their interest in our work [2] and for adding their experience with the treatment of thrombosis in the HeartWare left ventricular assist device (HVAD).

Implantation of the HVAD has become an established therapy for left ventricular failure. However, the condition of the critically ill patient with heart failure in the ‘real world’ is often complicated by haemodynamic instability, multiple organ failure and coagulopathy and in some cases compounded by surgical trauma, cardiopulmonary bypass injury and related severe complications such as postoperative bleeding, infection and right heart failure. Therefore, the early mortality in patients with INTERMACS level I, destination therapy and biventricular support is high. A novel implantation technique through small incisions, avoiding cardiopulmonary bypass, may minimize surgical trauma and reduce complication rates and early mortality [3, 4].

The device selection strategy in our VAD programme includes primary implantation of an HVAD in all small patients (mostly female), patients with a planned implantation through a lateral thoracotomy (mostly destination therapy), patients with many previous cardiac operations and patients with severely impaired right ventricular function. As all these subgroups have higher perioperative mortality, it is not surprising that our survival rate cannot match the reported survival data from studies carried out for FDA or CE mark approval under completely different conditions.

Thrombosis in the LVAD pumps is a severe complication that often results in high mortality. Pump thrombosis is a gradual process; thus, early detection is crucial for successful treatment. The technical sign for thrombosis of the HVAD pump is increased power consumption due to friction. As the flow is calculated linearly from power consumption, it increases simultaneously. A calculated flow of 10 l/min or higher strongly indicates pump thrombosis. Haemolysis with increased lactate dehydrogenase and free haemoglobin levels accompany thrombus formation in the pump.

Mohite et al. [1] point out that thrombolytic therapy is preferred by many for the treatment of device thrombosis. At our institution, thrombolytic therapy has not been performed; early pump thrombosis detection is followed by rapid device exchange. As the majority of pump thromboses in our series occurred early after the initial operation, we refrained from performing real thrombolytic therapy (e.g. with tissue plasminogen activator) as the risk of severe bleeding complications would have been too high. Instead, we treated a limited number of patients with tirofiban (Aggrastat); however, with limited success: only 1 patient could be discharged home without device exchange. In the most recent 179 patients since publication [2] treated according to our protocol, the device exchange rate has been almost halved to 4.5% (n = 8), with 2 deaths.

Minimally invasive device exchange procedures can be performed with low early mortality and contribute to faster postoperative recovery [5, 6] and should remain an important therapeutic option for patients with pump thrombosis.

REFERENCES