An Adverse Event Analysis: Inadvertent Exsanguination Following Left Ventricular Assist Device Implantation in a Child

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Neurologic deficit subsequent to cardiac surgery remains a cause of postoperative morbidity and mortality. Although myriad risk factors for postoperative cognitive decline have been identified, their individual influence remains undefined. Although less emphasis is now placed on the heart lung machine as the major source of postoperative cognitive decline, the conduct of cardiopulmonary bypass and, in particular, the management of the bypass circuit remain key to patient safety.We present a case of inadvertent intraoperative exsanguination of a patient following open heart surgery for implantation of a left ventricular assist device. The patient suffered significant neurologic damage. However, the nature of the patient's cerebral injury indicated thromboembolism as the likely cause, rather than hypoxic-ischemic injury caused by hypoperfusion. Subsequent investigation of the incident identified several possible sources and potential causes of embolization to the brain that could not rule out the exsanguination event as a contributing factor.
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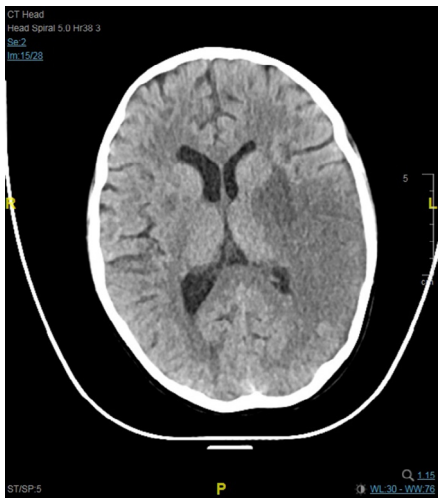
Case Presentation

A 13-year-old girl, weighing 36 kg, presented with heart failure caused by dilated cardiomyopathy. She was placed on venoarterial extracorporeal membrane oxygenation (ECMO) support for a period of 7 days before surgery for implantation of a left ventricular assist device (LVAD) (HVAD; HeartWare Inc., Framingham, MA) as a bridge to cardiac transplantation.

The patient's surgery proceeded uneventfully. Cardiopulmonary bypass (CPB) was performed via the ECMO cannulae, which were situated in the right internal jugular vein and the carotid artery. Venous return to the CPB circuit was augmented with a right-sided femoral venous cannula. The aorta was cross-clamped and the heart arrested for 11 minutes to allow for closure of an atrial septal defect created by a balloon atrial septostomy to decompress the left heart while the patient was on ECMO. The LVAD was implanted with the heart perfused (total CPB time 129 minutes). The patient was successfully weaned from CPB on inhaled nitric oxide and infusions of adrenaline (0.5 μg/kg/min) and milrinone (0.5 μg/kg/min) consistent with the standard management protocol. Poor LVAD flow was noted initially post CPB, which was attributed to poor right ventricular function. The hemodynamics improved in response to increased



Computed tomography scan demonstrating cerebral embolic injury.

Central Message

Neurologic injury following cardiac surgery is a cause of morbidity and mortality. This report demonstrates how it remains possible to navigate the "Swiss cheese" model leading to preventable error.

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adrenaline and protamine was administered. The venous cannulae were clamped, and the right femoral venous cannula was removed, but the arterial cannula was left unclamped to allow volume administration as needed.

At this time, the perfusionist who had conducted CPB (perfusionist A) was relieved by a colleague (perfusionist B). Following protamine administration, the perfusate remaining in the heart lung machine (HLM) was chased through into the cell saver via the CPB hemofiltration circuit by perfusionist B. For this procedure to take place, the arterial line needed to have been clamped adjacent to the arterial cannula in the operating field. During this process, the operating surgeon left the operating theater (OT), and chest closure was commenced by the first assistant surgeon, followed by decannulation of the neck vessels beginning with the right internal jugular venous cannula. Perfusionist B observed these processes and removed the pump boot from the raceway of the hemofilter circuit in anticipation of dismantling the CPB circuit. Perfusionist B then left the OT to process the electronic perfusion record. The arterial limb of the CPB circuit was clamped distal to the oxygenator outside of the sterile field, meaning that the patient exsanguinated relatively slowly via the hemofiltration circuit into the venous reservoir (Fig. 1). As perfusionist B returned to the OT minutes later, the patient suffered a sudden and profound fall in blood pressure and the LVAD controller waveform indicated a "suction event," which was communicated to the team by the ventricular assist device specialist. The anesthetist fluid resuscitated the patient with >1 L intravenous crystalloid and colloid, and the chest was reopened. No source of bleeding was identified. At this time, perfusionist B observed an increase in the level of blood in the CPB circuit venous reservoir and asked the surgical team if the arterial line was clamped. The clamp was noted to be missing from the arterial line in the surgical field. The pump boot was replaced in the raceway and the patient's circulation recovered over the course of 3-4 minutes. The operating surgeon and perfusionist A were recalled to the OT. The operating surgeon asked for volume to be transfused from the CPB circuit. Perfusionist A began this process but noted the nonhomogenous perfusate and the microbubble alert on the HLM alarm. Transfusion was stopped after approximately 5-10 mL and the operating surgeon was informed that the CPB circuit was not viable.

The remainder of the patient's course in the OT was uneventful and the patient was transferred to the cardiac intensive care unit (CICU) without further incident.

Four days after surgery, the patient was noted to be still drowsy, but her pupils were equal and photosensitive. On the following day, however, the CICU record noted "neuro concern" characterized by facial asymmetry, absence of forehead creases, and right arm weakness accompanied by poor speech and cough reflex.

The patient underwent a head computed tomography scan that day (Fig. 2), which reported the following: established middle

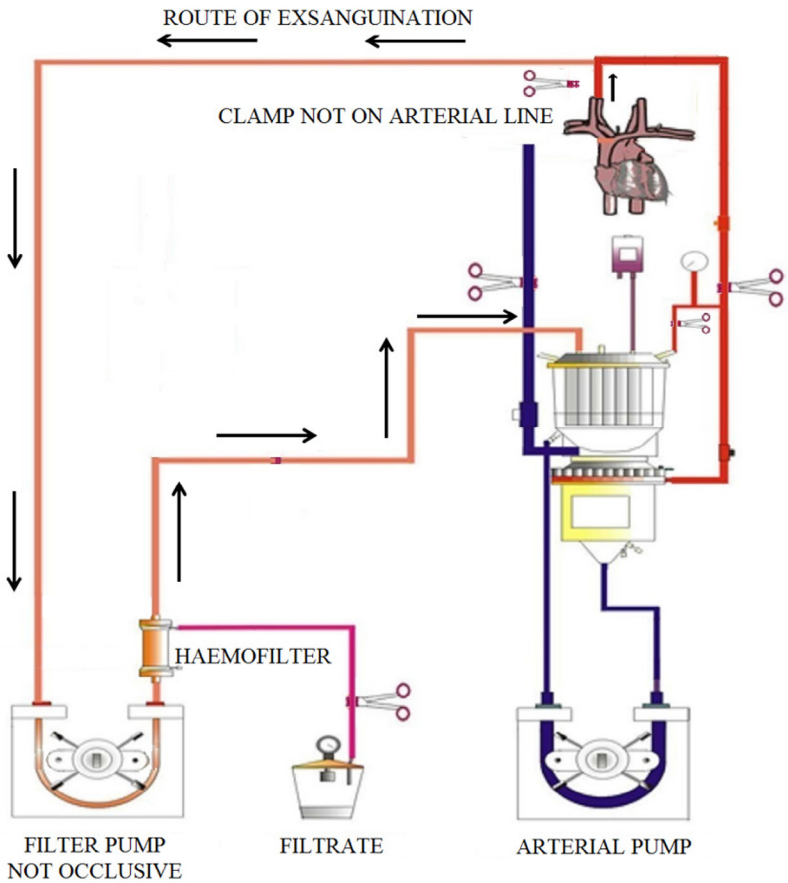

Figure 1 CPB circuit illustrating the clamp missing from the arterial line and the direction of blood flow during the exsanguination event. (Color version of figure is available online.)