the low platelet counts in this child as there was no clumping of the platelets on the peripheral blood smear. Leukemia was ruled out in this child based on the bone marrow biopsy report. Exposure to unfractionated heparin as well as low-molecular-weight heparin has been implicated in the pathogenesis of heparin-induced thrombocytopenia in cardiothoracic surgical patients.[3] The possibility of heparin - induced thrombocytopenia was not considered, as the baby had low platelet counts even before cardiac catheterization, and the enzyme-linked immunosorbent assay test was negative.

Thrombocytopenia absent radius is a rare syndrome that could be associated with tetralogy of Fallot in which bilateral absence of radii, hypomegakaryocytic thrombocytopenia, and presence of both thumbs are the pathognomonic features.[4] This child did not have limb abnormalities, and the bone marrow report did not corroborate with a diagnosis of thrombocytopenia absent radius syndrome.

Sickle cell trait with cyanotic heart disease causing hemolysis and thrombocytopenia was again ruled out as there was no evidence of hemolysis (bilirubin total: 8 μmol/L [range: 0-20], alanine aminotransferase: 6 IU/L [range: 0-40], alkaline phosphatase: 123 IU/L [range: 90-210], absolute reticulocyte count 141×10^9/L [range: 20-150]; % reticulocyte 1.9% [range: 0.5-3]).

In patients with cyanotic heart disease, the level of cyanosis-induced polycythemia could have an impact on the degree of thrombocytopenia and platelet function abnormalities.[1] This is supported by an observed inverse relationship between platelet count and mean platelet volume. High mean platelet volume with low platelet counts could indicate destruction of platelets despite the bone-marrow-producing platelets and releasing them into circulation. Consumption of platelets may be secondary to increased blood viscosity resulting in intravascular stasis and accumulation of fibrin and platelet deposits.[1] Olgar et al suggested that in patients with cyanotic heart disease and thrombocytopenia, hypoxia-related thrombocytopenia must be considered, and subsequent to reoxygenation by shunt or corrective surgeries, thrombocyte count and functions would recover.[5]

In this child, the D-dimer values were elevated albeit with no evidence of hemolysis. It was presumed that the thrombocytopenia with increased mean platelet volume in the presence of cyanosis and polycythemia (Hb: 17 g/L) was due to consumption of platelets. Thrombocytopenia was reversed once a corrective surgery was performed. Children with cyanotic heart disease presenting with persistent thrombocytopenia and increased mean platelet volume can undergo cardiac surgical procedures that reduce or totally correct hypoxia, with a resultant improvement in platelet levels.

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First Experience With Levosimendan Therapy After Correction of Congenital Heart Disease

To the Editor:

Low-Cardiac-Output Syndrome (LCOS) early after cardiopulmonary bypass (CPB) is a well-known problem in the postoperative course after complex congenital heart surgery, which affects the postoperative recovery of about 25% of patients.[1] It may cause prolonged mechanical ventilation, increased risk of infection and sepsis, longer stay in the intensive care unit, and increased mortality.[2]

At the beginning of 2012, we initiated use of levosimendan (Simdax; Orion Pharma, Espoo, Finland), a novel inodilator agent belonging to the family of calcium sensitizer agents.[3] At that time, a dose of 0.1-μg/kg/min of levosimendan was given continuously by infusion for 72 hours to 25 patients (levo group) who underwent elective pediatric cardiac surgery (children younger than 18 years of age) and arrived in the intensive care unit with a high inotropic score ($IS > 30$).

We compared the levo group with a control group that included 67 patients who underwent surgery between January 2008 and December 2011 and also arrived in the intensive care unit with high inotropic scores ($IS > 30$). During this period no levosimendan was used. All parameters that may have influenced the perioperative management, in particular anesthesia and CPB course, were not modified during the period under review. The standard institutional protocol for CPB weaning consisted of dobutamine, 5-μg/kg/min, started at the end of cross-clamp as the first choice. If mean artery pressure was below 50 mmHg and filling pressures did not indicate the need for fluid replacement, dobutamine was increased to 20-μg/kg/min with the addition of dopamine (3-20 μg/kg/min). If such targets were not achieved, epinephrine (0.02-0.3 μg/kg/min) was added. We did not use milrinone.

There were no differences between groups regarding demographic clinical data, risk stratification by comprehensive Aristotle score, and type of cardiac defects. Mean age was 6.4 ± 7 months in the levo group versus 6.9 ± 5.9 months in the control group ($p = 0.73$). No side effects related to

Table 1
Comparison of Hemodynamic and Metabolic Outcomes Between Levosimendan (25 patients) and Control Groups (67 Patients)

	ICUa		6 h		12 h		18 h		24 h		48 h		72 h	
	Levo	Control	Levo	Control	Levo	Control	Levo	Control	Levo	Control	Levo	Control	Levo	Control
HR (beats/min)	160 ± 13	165 ± 16	168 ± 9	175 ± 10*	170 ± 10	180 ± 14*	165 ± 9	178 ± 15*	165 ± 8	170 ± 10*	160 ± 8	164 ± 11	146 ± 10	150 ± 12
MAP (mmHg)	54 ± 6	56 ± 7	56 ± 6	55 ± 8	55 ± 5	57 ± 6	56 ± 5	58 ± 7	59 ± 7	60 ± 7	60 ± 6	58 ± 5	59 ± 6	60 ± 7
CVP (mmHg)	8 ± 3	7 ± 3	8 ± 2	8 ± 3	9 ± 2	10 ± 3	11 ± 3	12 ± 4	12 ± 3	13 ± 2	10 ± 2	11 ± 3	9 ± 3	10 ± 3
SpO2 (%)	97 ± 3	98 ± 2	99 ± 2	99 ± 2	99 ± 2	99 ± 2	98 ± 3	99 ± 2	99 ± 2	99 ± 2	99 ± 2	98 ± 3	99 ± 2	99 ± 2
ScvO2 (%)	52 ± 7	50 ± 8	55 ± 6	54 ± 6	58 ± 7	55 ± 8	64 ± 7	58 ± 9*	65 ± 7	60 ± 6*	66 ± 7	62 ± 8*	68 ± 6	66 ± 5
Lactates (mmol/L)	4.5 ± 1.1	4.8 ± 1.5	4.7 ± 1.2	5.8 ± 1.7*	4.8 ± 0.9	6.1 ± 1.5*	4 ± 1	5.1 ± 1.2*	3.2 ± 1.3	4.3 ± 1.2*	2.3 ± 1.0	3.1 ± 1.0*	1.5 ± 0.8	1.8 ± 0.7
Diuresis (mL/Kg/h)	–	–	8.5 ± 2.6	8.1 ± 3.0	6.4 ± 2.1	5.6 ± 2.1	6.1 ± 2.2	4.6 ± 1.9*	5.3 ± 1.8	3.8 ± 1.6*	5.2 ± 1.9	4.5 ± 1.7	5.7 ± 1.9	5 ± 2.1
IS (number)	43 ± 8	42 ± 6	40 ± 8	45 ± 10*	35 ± 6	45 ± 10*	32 ± 6	38 ± 7*	25 ± 5	35 ± 7*	23 ± 4	32 ± 7*	20 ± 4	30 ± 6*

Abbreviations: CVP, central venous pressure; HR, heart rate; ICUa, intensive care unit admission; IS, inotropic score; MAP, mean arterial pressure; $S_{cv}O2$, central venous oxygen saturation; SpO2, peripheral oxygen saturation.
*$p < 0.05$.

levosimendan infusion were reported; therefore, the drug was not stopped.

Comparing the 2 groups' postoperative outcomes showed differences in intubation time (5.9 ± 2.3 days versus 7.5 ± 2.5 days, p = 0.006) and intensive care unit and total hospital lengths of stay (12 ± 3.4 days *v* 15 ± 4.1 days, p = 0.002 and 25 ± 5 days *v* 29 ± 6 days, p = 0.004).

Four patients (5.9%) in the control group died (2 postsurgical irreversible left ventricular dysfunction, 2 multiple organ dysfunction syndrome), and 1 (4%) in the levosimendan group died (postsurgical irreversible left ventricular dysfunction) (p = 0.59).

No significant differences were found in hemodynamic outcomes, mean arterial pressure, central venous pressure, systemic oxygen saturation (Table 1) or fluid balance in the first 72 hours (−100 ± 25 *v* −90 ± 30 mL, p = 0.14). Only the postoperative heart rate was lower in levosimendan patients, with a significant difference at 6 (p = 0.003), 12 (p = 0.001), 18 (p = 0.0001), and 24 hours (p = 0.02). As a metabolic outcome (Table 1), mixed venous oxygen saturation was statistically significantly lower in the control group at 12, 24, and 48 hours (respectively, p = 0.0034, p = 0.001, and p = 0.03). Furthermore, diuresis showed a reduction in the control group at 18 and 24 hours (p = 0.002 and p = 0.0002), but the need for dialysis was similar in the 2 groups (2 pts *v* 5 pts; p = 0.62). Finally, lactate levels were lower in the levosimendan group, with a significant difference at 6, 12, 18, 24, and 48 hours (respectively, p = 0.004, p = 0.0001, p = 0.0001, p = 0.0002, p = 0.001), and inotropic score was significantly lower in the levosimendan group up to 72 hours (p = 0.027 at 6 hours, p = 0.0001 at 72 hours). With regard to the pharmacodynamic properties, particularly of the metabolite of levosimendan with a persistence of approximately 1 week, no early or late adverse effects were reported.

The first study investigating levosimendan in children was published in 2004.[4] Thirteen children between the ages of 3 months and 7 years with congenital heart disease received a single bolus of 12-μg/kg during preoperative cardiac catheterization. The changes in hemodynamic variables after this small dose of levosimendan were not statistically significant. The authors did not observe any serious adverse events during the course of the study.

In the only prospective trial found in the literature (32 cases and 31 controls), Ricci et al[5] evaluated the safety and efficacy of levosimendan in neonates with congenital heart disease undergoing cardiac surgery with CPB and concluded that levosimendan infusion was well tolerated, with a potential benefit on postoperative hemodynamic and metabolic parameters of RACHS (risk adjustment for congenital heart surgery) 3-4 neonates. Recently, Joshi et al[6] observed that in 110 patients, levosimendan could be used in all age groups and all complexities of congenital cardiac procedures with minimal side effects, and a inotropic-based regimen showed effective control of LCOS demonstrated by a low LCOS-related mortality of < 5%.

These results may not be applicable to groups that use more inodilators (milrinone) and/or target oxygen delivery or

cardiac output rather than blood pressure in their postoperative vasoactive support strategy. However, we have provided additional data to suggest a randomized large trial is indicated in pediatric patients following cardiac surgery.

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Internal Jugular Vein Valves: Clear and Present Danger

To the Editor:

We recently anesthetized a 55-year-old male for 4-vessel coronary artery bypass grafting. After induction, we prepared and draped the right side of his neck for right internal jugular vein (RIJV) central catheter placement under ultrasound guidance. After identifying the IJV via ultrasound, we accessed the vein using a modified Seldinger technique with an 18-gauge, 2.5-inch Arrow angiocatheter (Teleflex, Morrisville, NC) and removed the needle. Following confirmation of venous placement by manometry, we inserted the wire and removed the angiocatheter. When we directed the catheter and dilator assembly over the wire, we noticed a tenting of the cannula under the skin of the patient's neck. We could not aspirate blood through the catheter and decided to remove it. We repeated the ultrasound imaging and discovered a valve in the IJV, proximal to the insertion site (Fig 1, Video Clip 1), which had not been visualized in the initial scan. Previous literature showed up to 90% of human IJVs contain valves and these valves can disrupt guidewire placement.[1,2] These valves typically are located 0.5-to-2 cm above the union of either the subclavian vein or the innominate vein depending on laterality.[3]

On the second attempt with ultrasound guidance, we again met resistance when we tried to advance the catheter off of the dilator assembly. Although the wire continued to move freely and elicit narrow-complex ectopy, we removed the dilator and catheter assembly. Using ultrasound, we confirmed that the vessel containing the wire had bloodflow characteristics consistent with the RIJV and that the wire traversed the IJV past the valve (Fig 2). As we already planned to use transesophageal echocardiography (TEE) for the surgery, we placed the probe and confirmed that the wire tip was located in the superior vena cava. After we threaded a new catheter, we confirmed proper placement in the central vein by blood return during aspiration and the visualization of a central venous pressure tracing. Postoperatively, we obtained informed consent from the patient and acquired grayscale cinematic ultrasound images of the right neck with the patient seated in an upright position after the central venous access had been removed (Video clip 2). We identified the valve in the right internal jugular vein and visualized the valve leaflets as linear echogenic structures moving within the vessel lumen.

Although IJV valves and the complications with central venous cannulation have been documented previously, and even though ultrasound was utilized on the first attempt at venous cannulation, we did not identify the presence of an IJV valve until a subsequent attempt.[4–6] We demonstrated that TEE can be used to verify the wire tip location in the superior

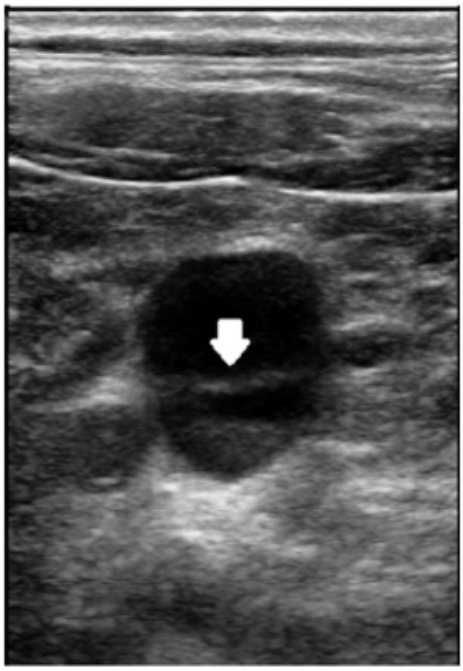

Fig 1. Axial grayscale ultrasound image of the right internal jugular vein shows a valve leaflet as a thin echogenic linear structure in the central lumen.