



#### CLINICAL CASE

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# Post-operative heart failure management. Report from a case that consisted of correcting the Taussig-Bing disease using an arterial switch. Emphasis on the use of pediatric levosimendan and neural splanchnic monitoring

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#### SUMMARY

Congenital heart disease surgery has importantly evolved in the last decades, among other reasons, due to new pharmacological elements for the management of post extracorporeal by pass low cardiac output syndrome and to new non invasive monitoring devices for cerebral and splanchnic perfusion, which promote better conditions for good outcomes in this set of patients. We report the case of an 8 year old male patient with the following diagnosis: double outlet right ventricle with subpulmonary ventricular septal defect (Taussig–Bing), previous pulmonary artery bandage done at the first year of life, and pseudocoarctation of the aortic arch, that underwent total correction by arterial switch. We focus on the use of levosimendan, a new inodilator drug, and the monitoring of cerebral and splanchnic perfusion using near-infrared spectroscopy.

**Key words:** Congenital heart diseases, levosimendan, near-infrared spectroscopy.

# RESUMEN

La cirugía cardíaca de enfermedades congénitas ha evolucionado en las últimas dos décadas, entre otras razones, gracias a que se han descrito nuevos elementos de manejo farmacológico del bajo gasto posterior a bomba de circulación extracorpórea y nuevos implementos de vigilancia de la perfusión esplácnica y cerebral de forma no invasiva, que brindan condiciones para que estos pacientes tengan una mejor evolución. Presentamos el caso de un paciente masculino de 8 años portador de los siguientes diagnósticos: doble salida de ventrículo derecho, comunicación interventricular subpulmonar (Taussig-Bing), antecedente de bandaje de arteria pulmonar en el primer año de vida y pseudocoartación aórtica, programado para corrección mediante switch arterial. Hacemos énfasis en el empleo de levosimendan, un nuevo fármaco inodilatador y en la vigilancia de la perfusión cerebral y esplácnica con espectrometría cercana a la infrarroja.

**Palabras clave:** Cardiopatías congénitas, levosimendan, espectrometría cercana a la infrarroja.

# INTRODUCTION

Cardiac surgery for congenital diseases has evolved in the last couple of decades; amongst other reasons, due to the arrival of new monitoring technologies and new drugs that allow better post-operative progresses even after a long period of infusion pump use and clamping.

Recently, we have been able to add to the therapeutic armament a new inodilator drug called levosimendan that sensitizes the calcium channels with a minimum amount of phosphodiesterase inhibition, and it has been used in adults with severe heart failure and a resistance to catecholamine.

In cardiac surgery for patients suffering congenital malformations, due to situations that will be elaborated later on, a drug that has inodilatory non-adrenergic properties seems like a desirable option and has great probabilities of being implemented.

On the other hand, some reports recently described the use of near infrared spectrometry (NIRS) for neurological monitoring during corrective surgery for congenital malformations. Its use has allowed a better understanding of the way the regulation of brain circulation behaviors and has led to a better handling of the flow on an extra-corporeal circulation pump (ECCP).

In those situations in which due to technical reasons it is necessary to reduce the blood flow, monitoring brain saturation using the NIRS allows us to make the necessary adjustments and reduce to the fullest the risk of hypoxic brain damage.

Next, we well present a case of a patient that underwent corrective surgery for Taussig-Bing disease using an arterial switch, making emphasis on the use of levosimendan, a new inodilator drug, and the monitoring of brain and splanchnic perfusion with NIRS.

## A CASE REPORT

We are dealing with a male patient of 8 years old, with a weight of 22.05 lbs (ten kg) and a height of 3.61 ft (1.1 m), who has been diagnosed as follows: double outlet right ventricle (RV) and intra-ventricular communication (IVC) subpulmonary of 0.8661 in (Taussig-Bing); post-operative bandaging of pulmonary artery (PA) during his first year of life with narrowness of the aortic arch (aortic pseudo-coarctation).

His condition began at birth with a generalized cyanosis that would intensify during crying and feeding, fatigue, diaphoresis and polypnea; he was diagnosed with the aforementioned condition after 15 days of extra uterine life for which he underwent a pulmonary bandaging when he was 13 months old.

In August 2005, an echocardiography was made that showed: abdominal *Situs solitus*, veno-arterial connections

and concordant arteriovenous, double outlet right ventricle (RV), intra-ventricular communication (IVC), wide subpulmonary (0.8661 in), end to side vessels, anterior and left sided aorta, posterior pulmonary, post-operative state of the pulmonary bandage with a 55 mmHg gradient, aortic arch with a narrowness below the subclavia with a 18 mmHg gradient and a 73% expulsion fraction of the left ventricle (EFLV).

We conducted a cardiac catheterization according to this diagnose, that apart from the previously mentioned results, showed: pulmonary artery systolic pressure (PASP) of 39, pulmonary diastolic pressure (PDP) of 20 and an average (APAP) of 29 mmHg and aortic pseudo-coarctation with an 18 mmHg gradient. Due to all of the above, the patient was scheduled for a total correction through arterial switch (Jatene's procedure).

Preoperative exams showed the following results: Hb 23.9, Hto 66, leucocytes 5,800, lymphocytes 48, platelets 188,000 TP 12.5 (96%), TTP 47", glucose 91, creatinine 0.5, urea 27.8 Na 135, K 4.6, calcium 9.4, chloride 104.

When he arrived at the surgery room his blood pressure was 104/63, heart rate 108x' and 70% of average pulse on the oximetry.

The induction was made using 2 mg. of midazolam, 100-µg fentaline and 1 mg. of rocuronium.

The ventilation control was done through a pediatric volume ventilator (Avea, Bear Co., USA) with the following parameters: tidal volume at 100 mL, respiration rate of 24 per minute, a I:E 1:2 relation and 60% FiO<sub>2</sub>.

After the intubation, pediatric patches for the NIRS, model INVOS 5100 (Somanetics, USA) were placed in the right frontal region and the hepatic region, obtaining the basal data shown in table I.

Hemodynamic monitoring was established through the application of a cannula to the right radial artery with a 22 Gauge catheter and a 5-F central venous catheter of 4.72 in (Arrow International, Inc. 2400 Bernville Road, Reading PA 19605, USA) giving an initial central venous pressure (CVP) of 5 cm. $\rm H_2O$ .

Gasometrical values, brain oxygenation, splanchnic basal and pre-BCEC are shown in table I.

As part of the induction protocol and as an hematic saving, we proceeded to administer aprotinine prior loading dose of 60 thousand UK per kg. of weight and a later upkeep with 7 thousand UK of weight/h.

After intubation and based on the low level of central venous saturation, it was decided to increase the fluid input until it reached a PVC level of approximately 10 in an hour, so as to start later on with dobutamine at a rate of 5 micrograms/kg/minute.

After the first hour, and based on the deterioration of the gasometry and the NIRS brain values, it was decided to add

**Table I.** Gasometric values and preoperatives NIRS. On taking NIRS in second hour prepump, was observed a decrease in 23% with respect to basal in the cerebral sensor, but not exceed to 3 minutes (value in brackets).

	Basal	1a. Sample prepump (First hour)	2a. Sample prepump (Second hour)		
	Dobutamine 5 gammas	Adrenaline starts 0.1 gamma, levosimendan 0.2 gamma y dobutamine 5 gammas	Adrenaline 0.1 gamma levosimendan 0.2, suspended dobutamine		
NIRS (C/S)	70/74	58 (< 23%)/62	69/61		
pH (A/V)	7.36	7.30/7.25			
PaO <sub>2</sub>	68	66			
PvO <sub>2</sub>	35	39			
PaCO <sub>2</sub>	35	37			
PvCO <sub>2</sub>	44	45			
HCO <sub>3</sub>	24.9	16.5			
SaO <sub>2</sub>	91	90			
SvO <sub>2</sub>	64	65			
BA	- 0.5	- 6.4			
Lactate	0.5	0.3			
Diuresis			40		

(Abbreviations: NIRS = Near infrared spectrometry; C = cerebral/S = splachnic, BA = Base excess,  $gamma = \mu g/kg/min$ )

adrenalin at a rate of 0.1 micrograms/kg/minute and a loading dose of levosimendan of 12 micrograms/kg within a 30 minute period, in order to later on apply a progressive dose in continuous infusion starting at 0.05 micrograms/kg/minute until it reached the 0.2 micrograms/kg/minutes, suspending dobutamine from the inotropic scheme.

The cardiopulmonary derivation was started leading to a moderate hypothermia (78.80 °F), with a 150 mL/kg/minute flow corresponding to a 100%.

In order to keep the extra-corporeal circulation, a pH stat metabolic compensation technique was used during refrigeration and an alpha stat technique was used during reheating, adjusting the level of CO<sub>2</sub> in blood to 40 mmHg and a pH of 7.4. This was accomplished by using a blood gas monitor integrated to the lines of the tubes of the extra-corporeal circulation pump, which provided metabolic parameters every 7 seconds, making the necessary changes to reach the goals described with great accuracy.

The refrigeration was accomplished in 20 minutes and once the patient reached a mild hypothermia (78.80 °F) the flow of the extra-corporeal pump was reduced in order to reach 40% of the calculated flow (i.e. the pertaining flow was reduced by 60%), monitoring the levels of neural splanchnic of the NIRS.

Later on, through a median sternotomy and after heparinizing, the aorta, superior vena cava and inferior vena cava were cannulized. The bandage was taken off the pulmonary artery, the aorta was clamped and the arterial switch was made (Jatene's procedure) using the regular procedure, i.e., through right atriotomy and «ventilation» through an inter-auricular septum.

After that, the aorta and coronary buttons were sectioned, later on; the pulmonary artery was also sectioned, re-implanting of the coronary arteries took place at the same time.

Following that, the inter-ventricular communication (IVC) was closed, aligning the left ventricle with the neo-aorta to make a pulmonary artery plasty finally, using two pericardial patches to fill out the spaces left by the resection of the coronary buttons.

Due to the space ratio between the aorta and the pulmonary artery –side-by-side– characteristic of the Taussig-Bing heart, the anastomosis of the neo-pulmonary was done by partially closing the distal orifice of the pulmonary artery and extending the anastomotic mouth, slightly to the right side of it.

Nitric oxide was used in a rate of 40 ppm when the ventilation to remove the extra-corporeal circulation pump had started; the hemodynamic support was based on 0.03  $\mu$ g/kg/min of adrenaline, 6  $\mu$ g/kg/min of nitro-glycerin and 0.2  $\mu$ g/kg/min of levosimendan, the same that had been initialized during the pre-pump period.

The average blood pressure (ABP) at the moment of removing the pump was 73 mmHg, the heart rate (HR) 130x', the central venous pressure (CVP) 11 cm H<sub>2</sub>O, pulse oxime-

try (SpO<sub>2</sub>) 99% with an inspired oxygen fraction (FiO<sub>2</sub>) of a 100%, a level of  $CO_2$  at the end of expiration (EtCO<sub>2</sub>) of 27 mmHg and a temperature of 98.42 °F.

The total amount of aortic clamping was 3 hours and 11 minutes with a total amount of perfusion time of 5 hours and 13 minutes. The total amount of surgical time was 11 hours and 45 minutes. The global evolution during the extra-corporeal circulation can be appreciated in graphic II.

Within the first 24 hours the evolution was accompanied by the support of a mechanic ventilator, apart from  $0.2~\mu g/kg/min$  of levosimendan,  $0.05~\mu g/kg/min$  of adrenaline and  $0.6~\mu g/kg/min$ utes of nitro-glycerin; the ECG showed sinus rhythm, and a diuresis of approximately 4.0~mL/kg/h was obtained, due to the presence of hyperglycemia (related to the metabolic response to the trauma) an insulin infusion had to be set up.

Finally, the levosimendan infusion was suspended (after the first 24 hours) due to a good hemodynamic and clinic evolution, as well as the adrenalin and the nitro-glycerin; patient was discharged with medical treatment after 48 hours of staying in the Intensive Care Unit, with a total amount of inpatient stay of one week. He currently visits the Cardio Surgery Unit of the INP for check ups.

## DISCUSSION

The approach to congenital cardiopathies has evolved from palliative to total correction of pathologies that imply a severe anatomic alteration. Such is the case of diseases like the transposition of large vessels, the hypoplasia of the aortic arch, the univentricular heart and the hypoplasia of the left ventricle. Each one of the corrections to the aforemen-

**Table II.** Evolution of gas and other parameters during transpump. Specifies whether the metabolic management technique was performed using alpha stat or pH stat. The values of CO<sub>2</sub> are already corrected for the temperature level and according to the corresponding strategy.

Para- meter	First hour	First + 7´ Ph stat	First + 14 Ph stat	First + 25´Ph stat	First + 30´Ph stat	First + 40´ Ph stat	Second hour Ph stat	Second hour + 30 Ph stat	Overheating $\alpha$ stat	Overheating + 15° $\alpha$ stat
Heart										
rate			_	_				_		
(bpm)	105	0	0	0	0	0	0	0	50	73
Blood pressure	50/									
(mmHg)	40/									
(S, M, D)	35	34	36	38	43	34	39	39	34	51
Tempe-	00	01	00	00	10	01	00	00	01	01
rature										
(N/R)										
(°C)	30.6/32.6	28.2/29.5	28.2/29.2	28.7/29.2	27.8/28.6	28.3/28.6	28/28.4	28/28.4	37.5/32.7	37/35.6
PVC										
(mean)										
(mmHg)	2	NA	NA	NA	NA	NA	NA	NA	NA	NA
NIRS	70/70	75/05	77/00	77/00	70/07	75/07	70/04	70/04	70/04	74/70
(C/S) pH (A/V)	79/78 7.32	75/85 7.35	77/89 7.3	77/88 7.33	76/87 7.36	75/87 7.38	72/81 7.4	72/81 7.39	70/84 7.48	74/78 7.37
PaO <sub>2</sub>	633	372	323	7.33 297	327	333	341	347	320	317
PvO <sub>2</sub>	000	312	323	231	321	333	J <del>+</del> 1	547	320	317
PaCO <sub>2</sub>	46	37	45	42	41_	41	41	41	33	42
PvCO <sub>2</sub>			1///							
HCO <sub>3</sub>	23.7	22.9	25	24.8	26	26.9	28.3	27.6	25.9	24.3
SaO2	100	100	100	100	100	100	100	100	100	100
BA <sup>2</sup>	-2.4	-4.8	-3.4	-3.1	-1.6	-0.2	1.2	0.4	1.1	-1
Lactate	1.3	1.7	1.6	2.3	2.4	2	1.8	1.6	2.1	2.7
HB/Hto		7.4/24	8.4/27	9.0/29	9.0/29	9.0/29	8.7/28	8.7/28	7.4/24	9.6/31

(Abbreviations: NIRS = Near infrared spectrometry; C = cerebral/S = splachnic, BA = Base excess, S, M, D = Sistolic, mean, diastolic)

tioned conditions implies a complex technique that in many occasions requires a total circulatory arrest (TCA) under deep hypothermia, or else a selective brain perfusion, all of which can lead to potential tissular injuries in different organs such as the brain, kidney or liver.

Within the most difficult challenges one faces when approaching cardiac surgery of congenital diseases, we can find the correction of the transposition of large vessels (TLV) and the surgical correction of coarctation with hypoplasia of the aortic arch (AAO).

In the whole, the procedures that require TCA put the brain and the splanchnic layer at risk; besides, after it has been done, it generates a systematic inflammatory response that will get worse as the patient gets older.

On the other hand, the situations in which a patient goes through a long period of perfusion or aortic clamping, even without TCA, also compromise the brain and splanchnic circulation, and are more frequently seen in surgeries to correct congenital cardiopathies; a good example of this is the TGV correction like the one described in this report.

The main characteristic that can be observed after the cardiopulmonary derivation of the surgical correction is the severe myocardial stun, leading to what is known as *post-pump low output syndrome* (PLOS). This syndrome implies a hemodynamic alteration different to the classic shock syndromes and those that consider the four well-known types.

The PLOS is characterized by the presence of severe myocardial depression (low output) with an important vessel dilatation, which makes the pharmacological approach a lot more difficult.

Regarding the correction of TGV, it is important to remember that these patients suffer from pulmonary hyperflow, which if it is not corrected at a young age (through pulmonary bandaging) it leads to the potential development of a persistent pulmonary hypertension due to the increase of the resistance on the pulmonary vessels.

Even though this phenomenon (the Eisenmenger syndrome) is rare at an early stage of life, the flux redistribution may condition at least a pulmonary hypertension crisis.

In the case that we described above, our patient reached an advanced age for the correction, and even though the Taussig-Bing heart should be treated surgically through an arterial switch during the neonatal stage, in this case the correction was made at age 8. He was referred to our institution after getting a pulmonary artery bandaging at 1 year old, this condition made the peri-operative approach a lot more complex.

This report is of great interest because it suggests that the flow reductions of the BCEC that are calculated in an indirect way, and used «blindly» sort to speak, might not be accurate and contrary to what it is believed, they might be more permissive. This would improve the conditions of the

operative field without the need to resort to circulatory arrest and without compromising the brain-splanchnic functions<sup>(1)</sup>.

In order to achieve the aforementioned flow, we need a monitoring device that uses the near infrared spectrometry principle (NIRS) and that has been widely studied and validated<sup>(1,2)</sup>. The values derived from the transmission to the frontal region of a beam of light in an emission range near the infrared, and it is a known fact that the correlation levels between NIRS and other invasive monitors of venous saturation on the brain (for example, jugular bulb) are high and have been validated under different conditions.

For example, in previous investigations we have analyzed the behavior of the brain NIRS in patients undergoing an exploratory laparoscopy for endometriosis and hence ASA I physical condition; that is to say, without major co-morbidity and young<sup>(3)</sup>. From this and other investigations we get the reference pattern of NIRS normal values in healthy patients, achieving a correlation analysis with the ones obtained from healthy patients, from the blood of the jugular bulb obtained through invasive methods.

This way it was determined that the level of non-invasive venous saturation on the brain ( $ScO_2$ ) that is considered normal is 65 to 70%, which correlates to those that refer to invasive methods<sup>(3)</sup>.

Considering the aforementioned, it is interesting to observe the behavior of the levels of ScO<sub>2</sub> determined through NIRS, in regards to the arterial pressure through out the cardiopulmonary derivation of the present case, given the fact that it exemplifies the use of this technology (Figure 1).

In the picture, we can see that despite maintaining the pressure levels low (at approximately 30 mmHg of average arterial pressure during the procedure) the levels of ScO<sub>2</sub> determined through NIRS and the levels of splanchnic saturation remained acceptable and were never below the basal.

In terms of the interpretation of the levels of NIRS for the monitored area, the  $ScO_2$  values defined as adequate are those that do not reach 20% of reduction in relation to the basal for more than 20 accumulative minutes<sup>(1,4)</sup>.

It is worth mentioning that a basal level is considered the one that is obtained after putting the sensor in the frontal and superciliary region (ideally before intubation), avoiding the longitudinal sinus and after a stabilization period of 15 minutes.

In this case –and considering the brain levels– the levels obtained were the ones that are recommended; however, and in reference to the splanchnic saturation levels, there is yet no consensus as far as the interpretation of NIRS in hepatic locations goes, that is why we resort to previous reports that use invasive methods.

Our group has previously reported an invasive mechanism to monitor supra-hepatic venous blood, which implies

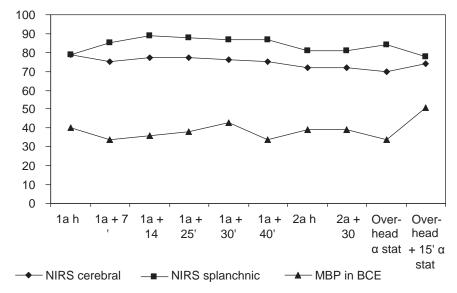


Figure 1. Shows changes in levels of NIRS cerebral or splanchnic, depending on the mean blood pressure (MBP) during extracorporeal circulation. From the firts hour and until before overheading (Overhead). Was used metabolic compensation pH stat. For overheating was used alpha stat technique (see text). Note the level of NIRS splanchnic is always slightly higher than the cerebral and that there were no decreases below 20% respect to baseline in either parameter, despite having levels of MBP up to 30 mmHg.

the insertion of a pulmonary floatation catheter with optical fiber guided through fluoroscopy. From this experience, we have reported what might be considered as normal values of supra-hepatic venous saturation ( $ShO_2$ ) and are between 62 and  $65\%^{(5,6)}$ .

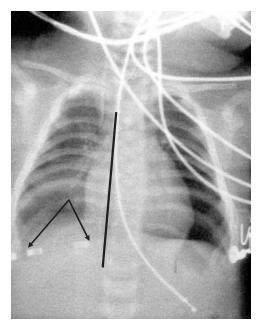
In the INP's Cardiovascular Surgery Service –and based on the aforementioned experiences– we have designed a technique for the insertion of a NIRS sensor in hepatic position, as a sort of strategy for the interpretation of the splanchnic value, considering that there are studies that support the possibility of an hepatic monitoring through this method<sup>(7)</sup>. Experimental studies that analyze the margin of error of the NIRS in hepatic position suggest that it is less than 3% when compared to direct measures on supra-hepatic veins<sup>(7)</sup>.

With this in mind, we inserted a pediatric sensor in the right dorsal region (in this case as well as in those cases of complex congenital cardiac diseases subject to a correction), at the level of the last rib with the free border (the border that does not have the adaptor to connect it to the screen of the monitor), externally in reference to the osseous part of the spinal column and in approximate correspondence with the projection of the hepatic rim in the anterior side (Figure 2).

With the purpose of verifying if the position of the sensor is correct (in the middle of the hepatic shadow); we make a simple abdomen x-ray that must show the diodes of the sensor in the middle of the hepatic shadow (Figure 2).

Once we have done this, we take 15 minutes for the stabilization of the basal ingestion, and with those values, we establish the 20% below which the hepatic saturation should not fall.

In upcoming publications, we will report our experience in relation to splanchnic injuries and NIRS; however, for the time being we can say –in an observational manner–



**Figure 2.** NIRS sensor placed on the hepatic position. The arrows indicate the sender and the receiver diode on the shadow hepatic. The vertical line indicates the edge of the sensor is not on the spine (see text for description of technique for placement).

that if the patients are kept with hepatic NIRS levels 20% below the basal, they will develop a manifested splanchnic failure, caused by either an elevation in the transaminases or a severe postoperative renal failure.

The issue of where the best place to measure the splanchnic profusion is for this method (NIRS) has been thoroughly discussed and the liver is currently accepted as the best place to do it due to its optic stability. As oppose to the measurements taken from the anterior part of the abdomen that are interfered by the movements and the intestinal gases<sup>(7)</sup>.

On the other hand, the case described here went through prolonged periods of clamping and pump and that is why we selected levosimendan to handle SBGP, being this, one of the first reports of its use in the pediatric field.

It is worth highlighting a few physiopathogenic elements in order to fundament de use of this drug.

The physiopathology of severe cardiac failure (CF) has been recently re-analyzed and in that sense we know that currently there are four different types of cardiac failures known, one of them being the one described in this report<sup>(8)</sup>. This implies that the patients develop CF temporarily and related to a modifiable factor, like cardiac surgery; however, patients that suffer congenital cardiopathies are more complex because they have a pre-existent overload since birth, which leads to ultra-structural modifications in an adrenergic receptor level.

In the case in question, the changes that arise in relation to the transposition of large vessels imply alterations in the ventricular architecture that lead to the modification of the expression of the beta adrenergic receptors, central as wells as peripheral, induced by the ventricular inability to handle volume<sup>(8)</sup>.

Summing up, we can say that, basically, four different types of beta adrenergic receptors have been described, namely: the beta 1 and beta 2 that mediate inotropism as well as vasodilatation, the recently described beta 3 that mediates negative inotropism and finally the beta 4 that seems to have a cardio-stimulant role, but depends on the stimulation of beta 1 to exercise its action<sup>(9)</sup>.

In the presence of cardiac failure, we know that<sup>(9)</sup>:

- There is a pronounced activation of the sympathetic system in a patient with CF that relates in an inverse way to the survival rate.
- The beta cardiac receptors –specially the beta 1– are sub regulated and the remaining receptors are disengaged from the proteins G.
- There is a reduction of approximately 50% (percentage related to the severity of the disease) of both beta 1 receptors and their RNAm.
- The beta 2 receptors barely suffer any changes.
- The rest of the beta receptors are desensitize via GRKs (quinines related to protein G)
- The continuous stimulation of beta-receptors 1 induces the apoptosis of the myocyte.

Based on what we said before, and similar to what happens with chronic cardiac failure, the stimulation of beta receptors is associated to a major morbidity, that is why the use of inotropic seems to be more preferable than using adrenergic<sup>(8,9)</sup>.

This case reports a way to use the levosimendan during trans-operative after a pediatric cardiac surgery as an inotropic support mechanism.

In general, current levosimendan indications refer to the approach to refractory cardiogenic shock in adult patients<sup>(10)</sup>; however, there are already a few reports related to the use of levosimendan in certain types of pediatric cardiogenic shock, the same that include the refractory cardiac failure<sup>(11)</sup> and low cardiac output<sup>(12)</sup>.

The novelty of the method presented here, lies in the fact that even though in most reports administration of the drug is initiated, due to the fact that there is an unbalanced cardiac failure refractory to adrenergic treatment (which is generally done during postoperative); in this case, and considering the pharmacokinetics referred to this population<sup>(12)</sup>, we decided to initiate administration during postoperative. We took into account the temporality that we could have as an effect after removing the pump, avoiding thus the hemodynamic deterioration that might arise due to excessive vasodilatation.

It is important to state that in most cases of correction of TGV we found hypoplasic left ventricles in different magnitudes, this is because this ventricle is the one that handles the pressure of the pulmonary system; this will severely compromise the systolic and diastolic functions and cause a cardiac failure, predominantly on the left side, when removing the extra-corporeal circulation pump. If we add to this the fact that we can generally have prolonged periods of clamping time and a BCE instance, we are going to have postoperative myocardial stun almost every time.

Our group uses levosimendan for cases such as the one described here and others similar, calculated based on m2 of body surface in the following way: patients whose ages range from 3-6 months, loading dose of 236  $\mu$ g x m2 of body surface and patients that go from 6 months old to 1 year old, loading dose is 276  $\mu$ g x m2 of body surface. When the patient has either one year or more than one, we administer a loading dose of 12 micrograms/kg.

In all of the cases we administered the dose in a period of 30 minutes through the infusion pump and we later on started a continuous infusion in progressive doses, starting with 0.05 micrograms/kg/minute until we reached the 0.2 micrograms/kg/minute (if there is no excessive vasodilatation).

The approximate time to make a loading dose is 10 minutes before the start of the refrigeration, because it takes an average of 20 minutes to reach the appropriate temperature in order to be able to perform the surgery (generally around 78.80 °F) and considering the amount of average pump time for complex cases; what we are looking for is to have the

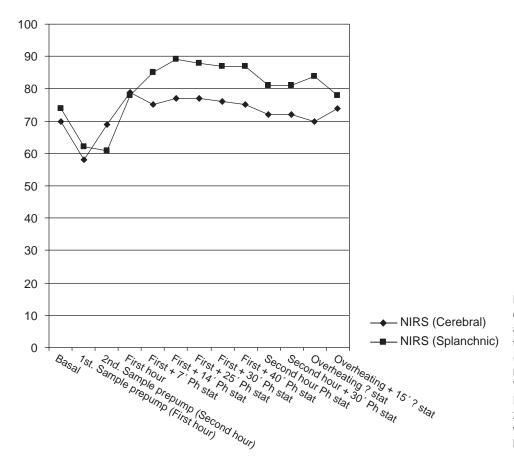


Figure 3. The graph shows the development of NIRS cerebral and splanchnic considering the baseline, prepump period (cooling), during cardiopulmonary bypass and overheating. The first decline in the value of NIRS cerebral (representing a 23% reduction on baseline) was what led to the addition of levosimendan.

drug reach its highest onset of action (that is of 2 hours) when exiting the BCE.

In figure 3, we show the evolution of the NIRS brain and splanchnic values, both basal and the one from the refrigeration process, during cardiopulmonary derivation and reheating. It is important to notice in this picture that by deciding to add levosimendan to the pharmacological scheme (due to the descent below 20% of brain NIRS in relation to the basal level), we managed to achieve a sustained correction of the levels of ScO<sub>2</sub> and an increase on the splanchnic values.

The special increase of the hepatic venous oxygen saturation (ShO<sub>2</sub>) that we can observe and the stability of the brain values, may find an explanation on the brain auto regulation on one hand, and on the other hand, the substantial effect of levosimendan on the splanchnic perfusion that has already been referred to by other authors<sup>(13)</sup>.

To sum up, we presented the experience of a case that underwent TGA correction approached with a new type of non-adrenergic inotropic and monitored by near infrared spectrometry that we describe as a new element of splanchnic monitoring.

With these two maneuvers, we believe that the patients that undergo these types of interventions and others that involve prolonged periods of cardiopulmonary derivation, with the potential of developing a post-pump low output syndrome and the consequent associated postoperative splanchnic failure, might have a better evolution and survival rate.

In order to homogenize the approach of these two novelty techniques, it is required to perform more studies in different groups of corrective surgical techniques for congenital cardiopathies, and we believe that both of them could also be useful in other types of extensive non-cardiac surgeries.

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