What is the "Vasoplegic Syndrome" (VS)?
The complications related to the cardiovascular surgery are multiple and they have been studied from the beginnings of this type of procedures. [1,2] Among them the cardiovascular ones are the most frequent and they constitute also, the more common causes of death. [3,4]

Since a few years ago there has been evident the relatively frequent appearance of a postoperative picture characterized basically by severe systemic hypotension, low ventricular filling pressures, and partial response to the expansion of the intravascular space with important amounts of liquids. Some medical groups named initially to this complication VS, alluding to the pronounced vasodilatation that characterizes it. [5,6] Its worse expression is the vasodilatory shock. [7]

It is necessary to remember that more than two decades ago, the predominant phenomenon in the immediate postoperative period of cardiovascular surgery was the vasoconstriction, shown as perioperative systemic hypertension. [8,9]

The states of pronounced vasodilatation also have been observed, in specific sub-groups of patients submitted to cardiovascular surgery: the patients with advanced heart failure to whom a device of ventricular left assistance is implanted; [7] and those in the immediate postoperative period of the cardiac transplant, in whom the mortality increases.[10]

The denomination of this state of vasodilatación does not keep uniformity of criteria. The used terms include from hypotension, [11] alluding to the most showy sign, up to shock, used to express maximum gravity and sudden appearance. Another is state of low systemic vascular resistance, [12] that emphasizes the hemodynamic mechanism involved.

Also one has alluded to this type of complication, confusing it with the systemic inflammatory response syndrome. [13,14] Although the vasodilatación come along with inflammatory response, other causes can produce the first one.

In relation to the used shock denomination, its habitually goes followed by some term that tends to specify the mechanism that produces it. Some years behind the expression distributive shock was frequent [15] whereas at present it is used vasodilatory shock. [16]

In the context of the cardiovascular surgery the term VS is used. [17]

The incidence of the VS immediately after cardiac surgery changes in a wide status as the reports. In Brazil, W. Gomes observed an incidence of 0,4 %, being located between the lowest. [17]

Whereas in the opposite end, Argenziano et al found in the United States 42 % of patients with this complication. [7]

Other authors, showed intermediate values. It is the case of Carrel et al who studied 800 coronary
and aortic valvular patients in Switzerland, finding that 21.9% of them developed the VS. [18]


The diagnosis of VS in the course of the postoperative period can be established by clinic or hemodynamics indicators, when a flotation catheter is placed in the patient pulmonary artery. [22,23]

The clinical signs include: 1) systemic hypotension (systolic pressure below 90 mmHg) that does not recover appropriately to the fluids infusion. 2) diuresis low, normal or high. 3) partial response to the intravascular fluids infusion. 4) vasoconstrictors drugs requirement (Dopamine, Norepinefrine, etc.) to support the hemodynamic status of the patient.

Due to the clinical signs low sensibility and especificity in the Consensus of Definitions in Cardiac Surgical Intensive Care published recently by the Argentine Federation of Cardiology it is demanded to accomplish the clinical diagnosis of this syndrome all the mentioned criteria, simultaneously. [24]

The hemodynamic diagnosis of this complication is based in: 1) low systemic vascular resistance is the key element in the diagnosis since it expresses vasodilatation. The systemic vascular resistance index is always below the normal values (2000-2400 dyn.seg.cm-5.m2) and in the most serious cases it has reached 700 dyn.seg.cm-5.m2. [25] 2) high or normal cardiac index. 3) low right and left ventricular filling pressures.

At the present moment the VS etiology is not clear. Prolonged time of extracorporeal circulation (CEC), use of normothermia during extracorporeal circulation, administration of big volumes of cardioplegic solution, depressed preoperative ventricular function, preoperative treatment with angiotensin-converting enzyme inhibitors and with intravenous heparin were investigated and proposed as causal factors. [18,26-30]

Some of these conditions are related to the development of the systemic inflammatory response to the surgery.

Several hypotheses have been proposed to explain the pathophysiology of the severe vasodilatation states. Nevertheless, the most plausible seem to be the three that are summed up next.

The first one relates the trigger insult to the production of acidosis, or more globally tissue hypoxia and through this the generation of vasodilatation or vasoconstrictors resistance. [31,32]

The second one supposes that the generating factor or trigger produces severe hypotension which causes neurohypophysis vasopressin liberation as compensating mechanism. [26,33-36] This fact is related with the later deficit of vasopressin and relatively low hormone blood levels in relation to the hypotension grade.

The third conjecture is related to the nitric oxide, a compound of well-known vasodilating action. In this case the process trigger produces the expression of the inducible nitric oxide synthase. This can occur by mean of several cytokines (interleukins 1 and 6, tumor necrosis factor, interferon gamma) in the context of a syndrome of inflammatory systemic response. The expression of the enzyme leads to an excess of nitric oxide production. [37,38]

Although these hypotheses are schematically described in separated way probably mixes mechanisms or superposition take place in different grade.

In a beginning, the VS prognosis was considered to be benign, probably because many cases showed high cardiac output.

Nevertheless, frequently it is observed that the patients need high doses of vasoconstrictors drugs with persistence of the critical state for several days. [25]

This form of presentation is related to appearance of serious complications, being the most frequent the renal failure, the respiratory failure and the multiple organ failure. On the other hand, some investigators have published high mortality for this group of patients. [17,20,21]

The treatment is based on the use of drugs with vasoconstrictor effect with the purpose of supporting the organs perfusiön pressure.

The current use drugs are the catecholamines with adrenergic alpha effects (Dopamine, Norepinefrine, Adrenaline). [5,16,27]
At present, due to the greater knowledge of this syndrome new therapeutic strategies have begun to arise with promising results but without conclusive evidence up to the moment. Between them, the drugs used in the absence of response to the support treatment are Vasopressin or Terlipressin [25,26,39] Methylene blue [20] and corticosteroids low doses.[40,41].

**How seem to be the ventricular function of the patients with a Vasoplegic Syndrome?**

The released studies showed the VS after cardiovascular surgery as a high cardiac output state being supported so the ventricular function is normal or high.

In one of the first descriptions of this complication, where there was used the denomination of VS, W. Gomes et al commented on 6 cases with cardiac index between 2,97 and 3,82 L/min/m². The patients received high doses of Norepinefrine for their treatment. [5]

The same group, in 1998, described 16 patients with cardiac indexes between 2,93 and 7,7 L/min/m². All the patients needed Norepinefrine. [17]

In a cases and control study done in France, Mekontso-Dessap et al analyzed 36 vasoplegic patients, to whom they defined, between other criteria by a cardiac index higher than 2,5 L/min/m². More than 40 % of the patients was treated with Dopamine and 58 % with Norepinefrine. The control group (72 patients) did not receive vasoconstrictors drugs. It is interesting to notice that these investigators defined the cardiogenic shock only by a cardiac index below to 2,5 L/min/m² and constituted it in a exclusion criterion, like with the patients they named “mix shock” (cardiogenic and vasoplegic). [27]

Cremer et al, in a work done in Germany, studied 10 patients with what they named “hyperdynamic circulatory instability” produced by systemic inflammatory response syndrome. The patients showed a mean cardiac index after extracorporeal circulation of 5,2 L/min/m² and 3 hours later of 4,4 L/min/m². All the patients were treated with Dopamine or Norepinefrine. The control group (another 10 patients studied in equal moments) presented values of cardiac index of 2,5 and 2,9 L/min/m². [13]

Argenziano et al, in the USA, in a pilot study detected 20 patients with severe systemic hypotension that needed Norepinefrine. They labelled with the term vasodilatory shock to 11 of them which presented cardiac index higher than 2,5 L/min/m² and with the term cardiogenic shock to the remaining patients (with cardiac index below 2,5 L/min/m²). [26]

In Switzerland, Carrel et al analyzed 175 patients with VS who had to meet two of four conditions between which it was counted to have a cardiac index higher than 3,5 L/min/m². All the patients were treated with Norepinefrine. It is interesting to highlight that the latter investigators informed that 4 % of the patients presented “low cardiac output syndrome”. [18]

**What is the behavior of the ventricular function in other states with vasodilatación and inflammatory response as the sepsis?**

Nevertheless the previously mentioned, in some vasodilatation states similar to the VS, it has detected ventricular function deterioration.

It is the case of the sepsis, which as the VS, has been characterized as a hyperdynamic state with high cardiac output. Also, the sepsis is considered to be a systemic inflammatory response to the infection [42] whereas the systemic inflammatory response to the surgery has been located in a place of privilege in the VS etiology. But, in contrast to it, in some patients and at different moments of its evolution decrease of the ventricular function has been observed, as we will see next.

Some years later, investigators used the curve of Frank-Starling to show the ventricular function depression in the patients with sepsis. It is the case of Ognibene et al who measured the left ventricular work index and the end diastole volume index of left ventricle. They found a flattening of the curve in patients with septic shock. In these patients, increases in the end diastole volume produced small increases in the ventricular work compared with the critical patients without sepsis. [43]

The mentioned finds, which could have due to contractility deterioration or to decrease of the left ventricular compliance, were clarified later by Parker et al. This group, using radioisotopic technique showed left ventricular ejection fraction decreased as expression of contractile deterioration and acute dilation of this cardiac camera in patients with sepsis. [44]

On the other hand, studies with echocardiography suggested that the left ventricle diastolic function also is altered in the patients with sepsis.
Jafri et al. [45] showed the ventricular filling delayed and Munt et al. [46] and Poelaert et al. [47] found alterations of the left ventricular relaxation.

In a similar way, the exploration of the systolic and diastolic right ventricular function showed pronounced deterioration in the patients with sepsis.

Kimchi et al. [48] and Parker et al. [49] showed the ventricular systolic dysfunction demonstrated by decrease of the ejection fraction and right ventricular dilation that happened independently of the pulmonary vascular resistance and the pulmonary artery pressure.

Kimchi et al. [48] and of Schneider et al. [50] found a decrease of the right ventricular compliance expressed by an absence of correlation between end diastolic right ventricular pressures and volumes.

What effects have the chemical mediators of the systemic inflammatory response on the ventricular function?

It previously exhibited in relation to the depression of the ventricular function in the sepsis is emphasized by the analysis of the chemical mediators of the systemic inflammatory response feature that it shares with the SV.

In sepsis or cardiac surgery models studies on animal and human it has been seen that many of the chemical mediators of the systemic inflammatory response have negative inotropic effect and therefore they can potentially cause ventricular function depression. It has indicated, between others, to the group of the prostaglandins, the leukotrienes, the thrombocytes activator factor, the histamine and the endorphins. [51] Nevertheless, some citokines (as the Tumor Necrosis Factor α (TNF) and the interleukins) seem to play a central role and therefore we will analyze them especially, next.

Investigations in animals as in human healthy volunteers showed that the experimentally raised levels of TNF produce systemic hypotension, increase of the cardiac output, decrease of the systemic vascular resistance, fever, lactic acidosis, disseminated intravascular coagulation, acute pulmonary injury and death. [52-55]

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In isolated rat heart, Schulz et al showed that the TNF and the interleukin 1 β induce pronounced depression of the contractile function through the nitric oxide. [66]

Also, Barth et al in a model of sepsis in mice registered deterioration of the ventricular function, measured as Norepinefrine needs worse in the group control with regard to genetically modified mice and therefore unable to synthesize inducible nitric oxide and to mice that received an inhibiting drug of inducible nitric oxide synthesis. [67] Other investigators found similar information in the same experimental model. [68,69]

**What do we note with regard to the systolic ventricular function hemodynamics parameters in the patient care with Vasoplegic Syndrome?**

Between April 15, 2003 and September 15, 2005 we enrolled prospectively the adult patients submitted to cardiovascular surgery with extracorporeal circulation in the Rosario’s Cardiovascular Institute, to locate those patients who presented the basic features of the VS.

In this period 515 cardiac surgeries registered and the mentioned postoperative complication was diagnosed in 158 patients (30,67 %) by clinical criteria.

In 44 of 158 patients (8,54 %) we had also hemodynamic diagnosis of the mentioned complication as the patient needed the placement of a pulmonary artery flotation catheter.

Three patients must be excluded for the analysis of his ventricular function because the scarce number of hemodynamics measurements that could be done.

To the remaining 41 patients, with clinical and hemodynamic diagnosis of VS we did 426 sessions of hemodynamics measurements and calculations along their stay in the cardiac surgical intensive care room.

We did hemodynamic diagnosis of systolic ventricular function depression when the measured stroke volume index (SVI) was below 25 mL/m² still in patients who were receiving inotropics drugs or when the basal SVI estimated by the linear regression model was below this value in the patients who were receiving Dopamine. The latter methodology was used to estimate the value of the IVS in absence of dopamine, in those patients who needed it during the measurement of the variable.

The differences in the values of SVI were related with the linear regression model to the differences in the dopamine doses in consecutive moments. This allowed the calculation of the SVI value before to the first measurement of the variable under drug effect that we named “estimated basal SVI”. It should reflect the value that the variable had had without the effect of the treatment.

In 19 of 41 patients (46,3 %) we found at least a value of measured SVI below to the fixed threshold in spite of being receiving drugs with positive inotropic effect.

In 16 of 40 patients (40,0 %) who were receiving Dopamine as the only drug a basal SVI was estimated below to the threshold.

To delimit the patients with depression of the systolic ventricular function we decided to use the combination of two criteria previously mentioned in an additive way.

Therefore, we considered with depression of the systolic ventricular function 22 of 41 patients (53,7 %) in whom it was present the first criterion or the second criterion. This latter way of taking into account the depression of the systolic ventricular function seemed to us conservative and the nearest to the reality.

Also in 29 of 41 patients (70,7 %) right atrial and pulmonary wedge pressures were raised as expression of diastolic ventricular dysfunction.

We were ruled out any influence of the preoperative ventricular function.

The patients with depression of the postoperative ventricular function developed a higher number of major complications [Median (Percentile 25 %-Percentile 75 %)]: 3 (1-4) vs. 1 (0-2), (p=0,01) and showed more postoperative hospitalary long of stay: 13,5 (10,5-30,5) vs. 8,0 (7,0-9,0) days, (p=0,001).

The number of patients who presented myocardial infarction and atrial fibrilation showed a trend to be major in the group with depression of postoperative the ventricular function.
The same happened with the maximum dose of required Dopamine (when it was used as the only drug) and with the number of patients who needed two or more vasoconstrictors drugs or intraaortic counterpulsation balloon on the first postoperative day.

The number of deceased patients was an approximately the double in the group with depression of the postoperative ventricular function without reaching statistical significance.

Table 1 shows some of the possible effects of postoperative ventricular function depression in patients with Vasoplegic Syndrome.

<table>
<thead>
<tr>
<th></th>
<th>FV postop No Deprimida</th>
<th>FV postop Deprimida</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pt es con otras complicaciones mayores:</td>
<td>0/19 0,0 5/22 22,7 0,05</td>
<td>4/19 21,0 5/22 22,7 1</td>
</tr>
<tr>
<td>IAM pop</td>
<td>1/19 5,3 2/22 9,0 1</td>
<td></td>
</tr>
<tr>
<td>Sangrado excesivo</td>
<td>3/19 15,0 5/22 22,7 0,70</td>
<td></td>
</tr>
<tr>
<td>Insuficiencia renal aguda</td>
<td>4/19 21,0 10/2 45,4 0,19</td>
<td></td>
</tr>
<tr>
<td>Insuficiencia respiratoria aguda</td>
<td>1/19 5,3 6/22 27,3 0,09</td>
<td></td>
</tr>
<tr>
<td>Fibrilación auricular</td>
<td>3/19 15,8 3/22 13,6 1</td>
<td></td>
</tr>
<tr>
<td>Síndrome confusional</td>
<td>1/19 5,3 1/22 4,5 1</td>
<td></td>
</tr>
<tr>
<td>Neumopatía</td>
<td>0/19 0,0 3/22 13,6 0,23</td>
<td></td>
</tr>
<tr>
<td>Sepsis</td>
<td>1/18 5,6 1/20 5,0 1</td>
<td></td>
</tr>
<tr>
<td>Disturbio Orgánico Múltiple</td>
<td>2/19 10,5 3/20 15,0 1</td>
<td></td>
</tr>
<tr>
<td>Tratamiento:</td>
<td>3/19 15,8 5/22 22,7 0,70</td>
<td></td>
</tr>
<tr>
<td>Ptes con IABP et 1* día pop</td>
<td>3/19 15,8 7/21 33,3 0,28</td>
<td></td>
</tr>
<tr>
<td>Muerte en RCCV</td>
<td>3/19 15,8 6/22 27,2 0,46</td>
<td></td>
</tr>
<tr>
<td>Muerte hospitalaria</td>
<td>3/18 16,7 7/19 36,8 0,27</td>
<td></td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Mn</th>
<th>P25%</th>
<th>P75%</th>
<th>P25%</th>
<th>P75%</th>
<th>p**</th>
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<tbody>
<tr>
<td>Creatinina pop (mg/dL)</td>
<td>1,1</td>
<td>0,9-1,5</td>
<td>1,1</td>
<td>1,0-2,5</td>
<td>0,53</td>
<td></td>
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<tr>
<td>Pao2/Fio2 pop (mmHg)</td>
<td>275</td>
<td>240-345</td>
<td>290</td>
<td>190-260</td>
<td>0,11</td>
<td></td>
</tr>
<tr>
<td>Balance hídrico pop dia 0</td>
<td>1650</td>
<td>704-2090</td>
<td>2057</td>
<td>1390-2706</td>
<td>0,74</td>
<td></td>
</tr>
<tr>
<td>Diuresis (mL/Kg/h)</td>
<td>3,4</td>
<td>2,2-4,8</td>
<td>3,3</td>
<td>1,8-4,3</td>
<td>0,86</td>
<td></td>
</tr>
<tr>
<td>n° de otras complicaciones mayores/pte</td>
<td>1</td>
<td>0-2</td>
<td>3</td>
<td>1-4</td>
<td>0,01</td>
<td></td>
</tr>
<tr>
<td>Tratamiento:</td>
<td>8,0</td>
<td>5,0-10,0</td>
<td>11,0</td>
<td>7,0-20,0</td>
<td>0,11</td>
<td></td>
</tr>
<tr>
<td>Tiempo de estadía en RCCV(h)</td>
<td>110</td>
<td>81-141</td>
<td>149</td>
<td>52-192</td>
<td>0,36</td>
<td></td>
</tr>
<tr>
<td>Tiempo de estadía en RCCV y CC(d)</td>
<td>8,0</td>
<td>7,0-9,0</td>
<td>13,6</td>
<td>10,5-20,5</td>
<td>0,001</td>
<td></td>
</tr>
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</table>

**Table 1:** Effects of postoperative ventricular function depression in patients with Vasoplegic Syndrome

Postop VF: postoperative ventricular function, p*: associated probability (Fisher’s exact test), p**: associated probability (Wilcoxon’s test, normal approximation), pt/s: patient/s, AMI: acute myocardial infarction, pop: postoperative, IAB: intraaortic balloon, CSICR: cardiac surgical intensive care room, Mn: median, P25%-P75%: percentile 25 & 75%, day 0: day of surgery, max: maximal, LOS: long of stay, GW: general ward.

**Why the ventricular function of the patients with Vasoplegic Syndrome can seem normal or high?**

Some reasons can explain the ventricular function of the patients with VS seems to be normal or high in some studies.

First of all it is clear that many investigators have not taken in account the positive inotropic effect of the vasoconstrictors drugs used for the treatment of the VS.

M. R. Johnson does this type of critique in a editorial [12] on a study of Kristof et al [70] in which a...
quarter of the patients considered without low systemic vascular resistance were finding in treatment with vasoconstrictors drugs with inotropic effect.

The hemodynamics measurements done under the effect of the treatment in none of these studies were an object of some subsequent analysis to deprive them as we did in our investigation.

On the other hand, in several studies were excluded the patients with low cardiac index and they are considered to be carriers of another entity as it is the cardiogenic shock or "mixed shock" (cardiogenic and vasoplegic) without taking in account the systemic vascular resistance. Some grade of confusion seems clear between the VS and others complications that look like it.

In the pathological states in which the basic abnormality is the depression of the ventricular function as the cardiogenic shock the majority of the patients exhibit high systemic vascular resistance. [71]

Therefore the patients’ inclusion or exclusion in studies with VS should do only for the presence of vasodilatation markers (as systemic vascular resistance) and not for indicators of the ventricular function deterioration (as cardiac output or cardiac index).

In this sense can be quoted the study of Carrel et al who studied the patients with post surgical VS and in the postoperative outcome they described a percentage of patients with "Low cardiac output syndrome". [18] In this latter complication known from the beginnings of the cardiac surgery and long before the recognition of the VS the basic abnormality is the ventricular function depression which can evolve to the grade of cardiogenic shock and is accompanied of systemic vascular resistance normal or high. [24]

Also, is illustrative the study of Rosseel et al on inotropics drugs for the low cardiac output syndrome in which before receiving the drugs the patients exhibited an average systemic vascular resistance index of 2976 dinas.seg.cm-5.m2 and 60 of 70 patients (85,7 %) needed drugs with vasodilating effect. [72]

Therefore, it is possible that the group of Carrel actually showed a percentage of patients with VS who underwent depression of the ventricular function.

Another cause of conflict between studies as ours and the studies previously mentioned in which the patients with VS appears with normal or high ventricular function, can reside in the hemodynamic variable chosen to represent the systolic ventricular function. Since the patients have a high grade of heart rate variability this can influence the values of cardiac output or cardiac index.

Practically the totality of the studies quoted previously used the mentioned indicators, in contrast to our work in which we used the stroke volume index (SVI) that, for his form of calculation, is not affected by the heart rate (cardiac index /heart rate).

Finally, another potential source of conflict might be that the patients included in our study constituted a sub-group of major gravity since they needed the placement of a pulmonary artery catheter, habitually for treatment refractoriness. Nevertheless, several patients groups studied by other investigators also showed the same clinical severity. [7,39]

**What are the implications of the recognition of the postoperative depressed ventricular function in the patients with Vasoplegic Syndrome?**

The recognition of postoperative ventricular function depression associated with VS is important especially in the patient therapeutic aspects.

Among the vasoconstrictors drugs that can be used for treating the vasodilatation states we can find the catecholamines with pure alpha effect without clearly demonstrated positive inotropic effect (e.g. Phenytolephrine, Metaraminol, Ephedrine). [73]

The administration of this type of drugs to these patients can worsen the depression of the ventricular function by afterload increase without concomitant increase of the inotropic state. Better safety and therapeutic efficacy is supplied in this type of patients with adrenergic drugs with vasoconstrictor alpha effect and positive inotropic effect on the myocardium (e.g. Dopamine, Norepinefrine, Adrenaline). In this way two deficits situations are covered.

In addition, the intraaortic counterpulsion balloon, device of circulatory mechanical assistance used frequently to treat the ventricular left failure in the perioperative period of cardiac surgery [74] bases its effect on two mechanisms that are the decrease of the left ventricular afterload and the increase of the coronary flow. [75]
The patients with a vasodilatation state have for this reason the left ventricle afterload diminished. Therefore if the patient does not present ischemia on going the use of the counterpulsation balloon loses sense.

Also, the presence of an intravascular not endothelized device can increase or prolong the systemic inflammatory response that many of these patients suffer and that has been involved in the genesis of the VS.

Finally, if the problem is focused only from the deterioration of the ventricular function, it might be recommended the use of vasodilating drugs or inotrops drugs with vasodilating effect (ej. Dobutamine, Isoproterenol, Milrinone) since the reduction of the afterload is a therapeutic target of the heart failure. [23,76] In these patients, the use of these drugs is not possible because it aggravates more the arterial hypotension.

On the other hand, as we have exposed earlier in this paper, the appearance of the postoperative ventricular function depression in the patients with VS seems to be a severity marker. The group that presented it exhibited a worse outcome and a higher consumption of resources in the postoperative period.

Therefore, the patients with the coexistence of two mentioned conditions should be subject of study to the recognition and adoption of new interventions leading to reduce the morbidity, the mortality and the use of resources.

Conclusions
It has been supported up to the present that patients’ ventricular function with Vasoplegic Syndrome in the immediate postoperative period of cardiac surgery is normal or high.

Nevertheless, in similar states of vasodilatación and systemic inflammatory response, as the sepsis, investigators have shown depressed ventricular function.

Also, the chemical mediators of the systemic inflammatory response which is associated to the Vasoplegic Syndrome etiology possess a clear negative inotropic effect.

Our investigation showed not less than 40 % of the patients with Vasoplegic Syndrome in the immediate post surgical period of a cardiac surgery presented postoperative depressed ventricular function by hemodynamic criterion. This proportion can be higher according to the criterion used. It is of making to notice that the said percentage happened in a critical patients’ sub-group within the group of patients with Vasoplegic Syndrome, who needed hemodynamic monitoring for their care.

The presence in these patients of postoperative ventricular function deterioration has implications in their current care and it seems to be a marker of severity and resources use.

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