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Anesthetic considerations for surgery involving clamping of superior vena cava.

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ABSTRACT

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Superior vena cava (SVC) lies in the upper mediastinum, laterally to the right in the great vessel area. It serves the venous drainage of the upper part of the body, including the brain, directing the flow to the right atrium. Lung cancer, mediastinal malignancies, benign conditions and a variety of other diseases and clinical conditions in the adult population may lead to surgery involving this large vein. This would be in the presence or not of superior vena caval syndrome, which includes classic features as dilated veins in the upper half of the body, edema of the head, neck and upper extremities, profound collaterals in the chest wall and cyanosis. Problems arising from the problematic SVC drainage, concerning the systemic and cerebral hemodynamics and the perioperative administration of drugs and fluids deserve focusing. The pre- and intraoperative evaluation and management of such patients and the decisions to be made are also for attention.

Etiology of SVC syndrome

Surgery involving SVC

In the adult population, surgery involving the SVC is mainly for lung cancer, either for tumor removal (right pneumonectomy, lobectomies), with or without carinal resection or node resections[**1**]. In a lesser degree, operations for invasive thymomas[**2,3**], thyroidectomies with retrosternal extension[**4**] or operations for other mediastinal masses and vascular lesions may also involve SVC[**5**]. Malignancies of the mediastinum, benign conditions such as idiopathic fibrosis, granuloma and multinodural goiter may cause superior vena cava syndrome. Mediastinal lipomatosis characterized by overgrowth of adipose tissue may be also present in patients under long-term steroid administration or Cushing's syndrome and cause SVC obstruction[**6**]. This large vein may be traumatized during thoracic injuries or during diagnostic or therapeutic procedures[**7**]. Superior vena cava syndrome and septicemia were among the major complications associated with retained pacemaker leads in 119 patients reviewed retrospectively[**8**]. Hemorrhage in patients undergoing mediastinoscopy, sometimes a serious or even fatal complication, may be due to damage of the SVC[**9**]. Complications from pulmonary artery catheters as knotting within the superior vena cava, leading to purse string incision for pulling out, or perforation of this vein, leading to hemothorax, have also been described[**10,11,**

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12]. Catheter-induced thrombosis is not an uncommon complication after central venous access in anesthesia and intensive care medicine and may lead to SVC syndrome[**13**]. Thrombosis of SVC following multiple central venous cannulations that were required for dialysis[**14**], for plasma exchange to treat secondary hyperlipidemia due to nephrotic syndrome[**15**] or Hickman catheter placement [**16**] has also been reported. Extremely rarely, vascular pathology arising from this large vein *per se* may lead the patient to the operating room[**17**]. Finally, a review of 1016 cases of heart transplantation in Stanford University Medical Center revealed three cases of superior vena cava syndrome. All three of these patients underwent transplantation by means of the bicaval anastomotic technique and, in addition, the diameters of the donor and recipient cavae were grossly mismatched[**18**].

Life-threatening complications can occur unexpectedly during general anesthesia in operations of SVC. In case of malignancies involving SVC, decisions concerning patient's management and treatment (radiation, endovascular stenting, surgical treatment, adjuvant therapy) are based on tumor's primary pathology and resectability and patient's condition and operability. Furthermore, surgery-involving SVC, irrespectively of the presence or not of SVC syndrome, may be planned for situations as the above listed. The repair or the revascularization of the SVC may be successfully performed with heterologous 'custom-made' pericardial tube, with prosthetic SVC replacement as ringed polytetrafluorethylene (PTFE) grafts, with partial resection with running suture, with vascular stapler or patch[**1,2,19**]. In these circumstances, with or most often without the presence of superior vena cava syndrome, the peri-operative team has to decide whether to support or not the patient with extracorporeal circulation. Sometimes, although the institution of by-pass is not programmed, the need of using a pump or even of a heart-lung machine with an oxygenator (CPB) may arise intraoperatively under urgent conditions[**1**]. In a series of fourteen consecutive patients at two Boston hospitals, the use of CPB during the resection of locally advanced thoracic malignancies was classified as planned or emergency (8 vs 6), as is the use of CPB due to injury of SCV, IVC or pulmonary artery during surgery [**20**]. Nevertheless, in some of the above operative procedures, because of lack of equipment, surgeon's confidence, emergency character as acute and unexpected bleeding or for other reasons, the temporary by-pass of this vein was not established. In these cases of reduced flow *via* SVC or acute occlusion, the anesthesiologist has to face a number of problems not only arising from the disturbed SVC drainage but also with regard to the systemic and cerebral hemodynamics.

Anatomy

The union of the two innominate veins forms superior vena cava and after two inches it pierces the pericardium and ends in the upper and back part of the right atrium. It has no valves and its tributaries are the two innominate veins, the vena azygos and a few small mediastinal and pericardial veins. The azygos vein begins in the abdomen, originating variably from the posterior aspect of the inferior vena cava, from the right ascending lumbar and subcostal veins, or as a branch of the right renal vein (figures 1,2). It passes into the thorax through the aortic opening of the diaphragm. The hemiazygos vein begins in the left ascending lumbar or renal vein. Ascending on the left side of the vertebral column, it ends in the azygos vein. It receives the lower left intercostals veins, the small left mediastinal and esophageal veins and, sometimes, the lower end of the accessory hemiazygos vein. The latter is small, sometimes entirely absent and often continuous with or drained by the left superior intercostal vein. Normally, flow is directed from the azygos vein (directly from the right and via hemiazygos from the left retroperitoneal spaces) to the SVC[**21**].

If the inferior vena cava (IVC) is obstructed, the azygos and hemiazygos veins are one of the principal means by which circulation is carried out. In this case, flow in these collateral veins is

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Figure 1. The azygos and hemiazygos veins *(ref.: 21, after modification)*.

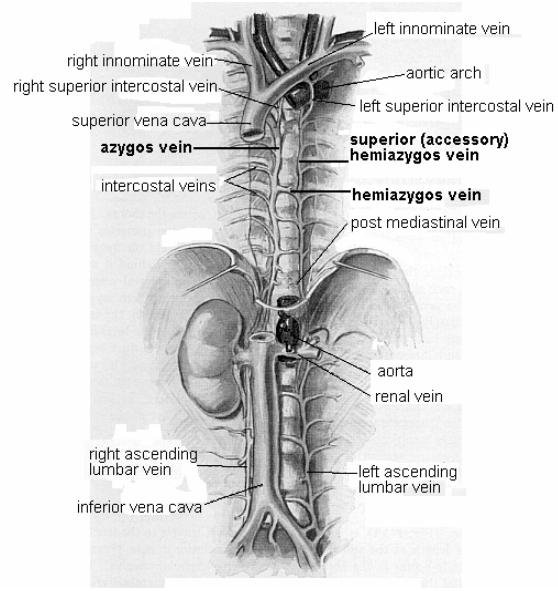
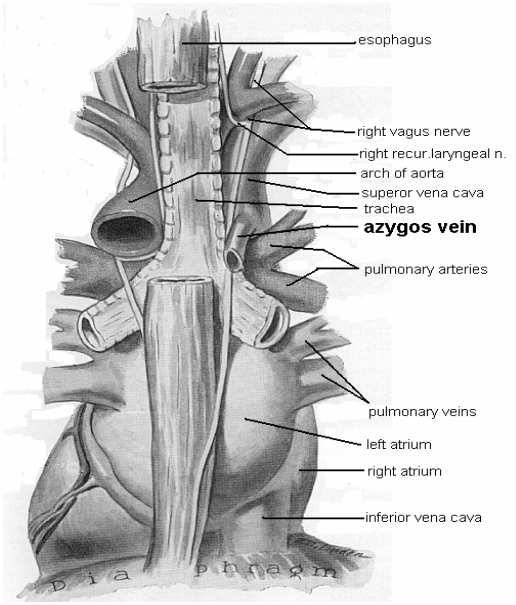


Figure 2. The mediastinal structures from behind *(ref.: 21, after modification)*



augmented and direction is stays normal. But in case of obstruction of the SVC, venous pressure in the upper part of the body is increased and venous drainage may be directed to the right heart via IVC after reversing flow in these collaterals.

Pathophysiology of superior vena caval occlusion

Clamping of SVC acutely reduces venous return with a further, beat-by-beat, reduction to an amount equal to the percentage of stroke volume that goes to the upper compartment. Stroke volume is progressively decreased. Equilibrium is reached when increased venous pressure gradient between the upper and lower compartments leads to supra normal collateral flow and activation of "sleeping" venous networks. Venous drainage may occur mainly via the azygos vein and the left superior intercostal vein (through the hemiazygos system) and in lower degree via the epigastric and phrenic veins and other collaterals into the inferior vena cava infradiaphragmatically. These veins are somehow regulators of venous return between the upper and lower compartments and, in case of SVC occlusion, the flow, reversed or not, is always towards the inferior vena cava. In this situation, the amount of blood volume pooled in the upper compartment is high, the systemic flow and pressure are decreased and the percentage of stroke volume directed to this compartment is equal to the flow by the collateral veins towards the lower compartment (figure 3). In case of subacute occlusion, a kind of equilibrium may be achieved as the time frame allows the veins to dilate and increase the cross-sectional area for adapting the flow. In acute occlusion as during clamping, severe brain damage or hemodynamic deterioration possibly happen before reaching equilibrium. Progressive narrowing of SVC lumen is followed by superior vena cava syndrome whereas acute clamping leads to a severe clinical picture.

Clinical manifestations

Dilated and distended veins and edema of the upper half of the body characterize the progressive narrowing of SVC lumen and the superior vena cava syndrome. The veins do not collapse with the patient in the upright position and distention is augmented in the recumbent position. The same degree of external edema must be expected in the internal structures as mouth, oropharynx, hypopharynx while upper

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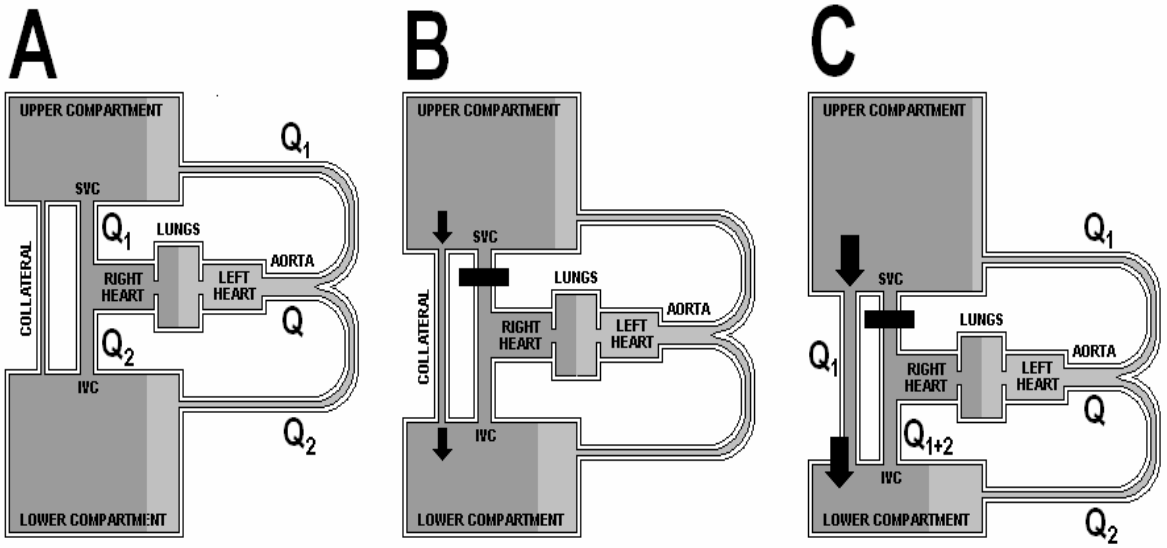
airway and trachea may be compromised by external compression limiting normal movement, or recurrent laryngeal nerve involvement[**22**]. One of our patients, fifteen months after surgical removal of lung cancer and seven months after a stent insertion for treatment of SVC syndrome, was readmitted with a bad dyspneic picture due to vocal cord paralysis. Imaging revealed residual tumor near the carina and a partially occluded SVC along the stent. Chordectomy was performed successfully under general anesthesia. Localized venous dilation and edema differentiate superior vena cava syndrome from pericardial effusions and tamponade, whereas edema is also extended to the lower extremities. Nevertheless, although cardiac involvement is often a late finding in widespread malignancy, the carcinoma of the lung may be presented rarely with such a combined picture making thus SVC involvement difficult to suspect[**23**]. Cardiac involvement in mediastinal tumors may also cause heart rate or rhythm alterations[**22**]. When the narrowing of the lumen of SVC occurs relatively rapidly, all the clinical signs are more prominent.

In case of acute occlusion of an until then normal SVC, as in clamping due to a technically difficult or problematic intraoperative course for a cancer (tumor invading, lysis of adhesions, serious bleeding), the picture of the anesthetized patient includes a sudden and serious drop in the systemic blood pressure, distension of the great veins of the neck, acute and progressively increasing edema of the head, neck, tongue and upper extremities and bulged eyeballs that may become uncovered by the swelling eyelids. The look of the patient is completely changed as his/her color goes to cyanosis. If central venous pressure is measured above the clamp, it goes high synchronously to clamping and continuous elevating in contrast to the reduced arterial pressure. The rate of the peripheral infusions may automatically be reduced or even stopped if the distance from the table is relatively short. In the surgical field, venous bleeding above the clamp is acutely exacerbated. If clamping is done above azygos junction, all the above are more prominent. In case of ongoing occlusion of the SVC, the patient is at risk for arrhythmias, cardiac arrest and bad outcome. A similar picture related to

Figure 3. Schematic representation of the volume and flow alterations during SVC occlusion. A: normal, B: increased flow to the IVC by the collaterals, C: equilibrium (arterial Q1=venous Q1), note the volume transferred to the upper compartment. Gray scale represents the volume changes in arterial and venous blood.



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patient's positioning, including vein distention in the neck and upper extremities, cyanosis and airway obstruction, has been described intraoperatively in anterior mediastinal tumors compressing the SVC, although surgery was not primarily involving this vein[**24**].

Cerebral circulation

As it was previously discussed, venous pressure in the upper half of the body is progressively elevated and collateral flow is augmented when the lumen of SVC is narrowed. Nevertheless, neurological deterioration is not often the case in patients with SVC syndrome as cardiac output and cerebral blood flow are maintained. In these patients, the hemodynamic state is in equilibrium as venous drainage from the upper compartment is almost normal due to the increased venous pressure. In case of clamping without the presence of a well-developed, supra-normal collateral vein network, as in patients without SVC syndrome, cerebral perfusion pressure is acutely decreased as both the systemic arterial and the opposing venous pressure are changing (decreasing and elevating respectively) in a manner that decreases cerebral flow. In a controlled animal setting under CPB with bicaval cannulation, both partial and complete acute occlusion of the SVC resulted in a reduction of cerebral blood flow and furthermore the difference was clinically quite significant for the complete occlusion [**25**]. There were also significant differences in cerebral oxygen delivery between groups and SVC pressure increased to 19.5±4.5 and 32.5±3.1mmHg with partial and complete occlusion respectively. It must be noted, that in these animals arterial pressure was kept stable by the pump throughout the observation. In other words, brain hemodynamic and oxygenation deterioration is expected worse in a patient with a clamped SVC as systemic flow and pressure are falling, a condition that was not allowed in the above setting.

Anesthetic management

Life-threatening complications, even sudden death[**26**], can occur unexpectedly during general anesthesia in patients with problematic SVC. Concerning the patients presenting SVC syndrome who are candidates for surgery not involving SVC clamping, the anesthetic management takes into account the possibility of a problematic airway[**22,27**], the pathology and the increased pressure of SVC during central venous catheterization and, in case of hemodynamic deterioration, the increased possibility of compromised brain oxygenation. Locoregional analgesic techniques seem a reasonable choice for these patients[**28,29**]. In case of general anesthesia, the method chosen depends upon the preoperative condition. At least one intravenous cannula should be inserted in the leg, preferably in the femoral vein, as the upper extremity infusions show very long and unpredictable circulation time. For the adequate ventilation of the patient during induction it is necessary to keep his sitting position. Awake intubation, facilitated or not with the aid of a fiberoptic laryngoscope or bronchoscope, may also appears to be necessary[**30,31**]. Fowler's position is considered to facilitate venous drainage. For surgery of the upper part of the body, venous bleeding and distorted surgical field are encountered the most significant problems, leading to awareness for availability of cross-matched blood.

Patients, presenting or not SVC syndrome, in which the surgical technique involves manipulations of SVC, can be divided to the subcategories listed below.

- *Patients with SVC syndrome in whom clamping of SVC may or definitely be included in the operative technique.*
- *Patients with SVC syndrome in whom clamping of SVC is done unexpectedly.*
- *Patients with normal circulation in whom clamping of SVC may or definitely be included in the operative technique.*
- *Patients with normal circulation in whom clamping of the SVC is done unexpectedly.*

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This categorization takes into account the existence of a supra-normal collateral vein network that may play a protective role during SVC clamping. It has been already discussed that patients with SVC syndrome are exposed to several problems that have impact on the surgical and anesthetic management; at the same time, manipulations or even acute occlusion of SVC without a kind of extra-corporeal circulatory support is expected to cause less deterioration as the lumen of this vein is already narrowed or obstructed. Patients with normal circulation are somehow unprotected from the not supra-developed, normal collaterals when clamping is done. In general, for patients belonging to the first and third category, the operative team has to plan and be prepared for a kind of extracorporeal by-pass. Patients belonging to the fourth category are at a considerably higher risk than these who belong to the second one.

Endovascular techniques

In the past decade, expandable metal stents have been introduced as sole palliative treatment or as an adjunct to other modalities for the treatment of malignant SVC syndrome[**32, 33**]. Endoluminal thrombolysis, balloon angioplasty and stent placement provide rapid and enduring relief of the symptoms. The application of these percutaneous interventions is strongly advocated not only to inoperable malignancies but also to other causes of SVC syndrome, such as a preoperative preparation for general anesthesia, thoracotomy or sternotomy and cardiopulmonary bypass. Anesthetic complications arising from the problematic airway and intraoperative problems like excessive bleeding, may be diminished or avoided through these techniques[**18**].

Use of extracorporeal circulation

The use of cardiopulmonary bypass (CPB) or other forms of extracorporeal circulation as different types of venovenous bypass, for locally advanced thoracic malignancies is highly controversial. Nevertheless, the resection of lung carcinomas or other tumors invading the SVC, although a technically demanding procedure, is demonstrated to be feasible in selected patients by several authors [**1,19,20,34,35,36, 37,38,39**]. Indications, technical aspects, type of vessel reconstruction and type of circulatory support are still objects of debate. The planned use of CPB to facilitate complete resection should be considered only after careful patient selection. The availability of CPB also provides a safety net in the event of injury to vascular structures during tumor resection. However, both pneumonectomy and complete resection of the SVC with prosthetic replacement are associated with a significantly increased risk of death[**1,19**]. The use of extracorporeal circulation is also proposed for surgical correction of other causes of SVC syndrome[**3,39,40**] and for patients presenting significant obstruction of the lower airway irrespectively of the existence of SVC syndrome [**41**].

Time and type of bypass

For patients with significantly compromised airway, many authors recommend venovenous cannulation under local anesthesia, before anesthetic induction, in readiness for upper comparetment's venous decompression. With this technique the symptoms that accompanied SVC syndrome do not worsen and the life-threatening complications during induction of general anesthesia are avoided. Cannulation of the femoral artery and vein in readiness for CPB under local anesthesia is also proposed[**35, 41**]. For patients with SVC syndrome right axillary artery-inferior vena cava cardiopulmonary bypass can be established intraoperatively before the main surgical intervention[**40**]. In case of SVC clamping, both these types of by-pass are based on the collateral patency. Furthermore, the use of oxygenator makes full heparinization mandatory. Alternatively, a temporary bypass between the left or right innominate vein and right auricula[**36,39**] or an axillofemoral[**37,42**], or axillosaphenous[**38**] venovenous bypass can be made, by using artificial vessels before clamping of the SVC. Simultaneous bypasses between right- and left-brachiocephalic veins and right atrium for surgery involving thymus has also been proposed[**3**]. In a case of completely obstructed internal and external jugular veins after

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radical laryngectomy and radiotherapy due to hypopharyngeal carcinoma, venous bypass grafting from cerebral transverse sinus to right atrium was performed[**39**]. For these types of bypass, appropriately sized cannulas are directly introduced into the corresponding veins and/or right atrium and connected together with a tube. Simplicity, nondependency on special equipment as heart-lung machine, reduced costs, avoidance of full heparinization and theoretically lesser inflammatory response are among the advantages of the types of above-mentioned venovenous bypass. However, cardiopulmonary bypass would be of important value intraoperatively in case of problematic oxygenation or significant bleeding and it would allow the application of hypothermia.

Monitoring

In general, for patients belonging in the first and third categories, where a kind of extracorporeal by-pass is planned, monitoring is analogous to the procedure. In case of CPB, monitoring is as for cardiac operations. If a pulmonary artery catheter is decided it must be inserted *via* a femoral vein. It can be inserted under local anesthesia or after induction. Preanesthetic placement and calibration allows early SVO_2 monitoring, which may be proven of extreme value during induction and mechanical ventilation. Intermittent positive pressure ventilation is expected to increase the pressures of intrathoracic venous and atrial structures [**43**] including the collaterals, further deteriorating brain outflow. During veno-venous bypass, fluctuations of SVO_2 values may be due to unstable venous return from the upper compartment. It must be remembered that, in highly anesthetized patients, the jugular oxygen content is a major determinant of SVO_2 further impacting on this value. To evaluate subjectively the preload by looking to the surgical field, the semi-opened pericardium and the position of the patient allow direct vision only of the right atrium. In case of veno-venous bypass, the venous pressure of an internal jugular vein must also be monitored and is expected to decrease immediately after the establishment of the temporary bypass. Even in sitting or Fowler's position, venous pressures up to 40 mmHg have been reported. Significantly decreasing with the temporary bypass denotes adequacy. Alternatively, peripheral venous pressure monitoring has also been reported[**36**]. Although findings from monitoring of brain tissue oxygenation in humans (near-infrared spectroscopy/NIRS) are not available in the literature, the use of this modality is expected to be of most help. In an animal setting, NIRS indicated significant cerebral ischemia to the acute SVC occlusion[**25**]. Finally, in most of the cases, the anesthesiologist must be experienced in managing one-lung ventilation.

Unexpected, acute clamping of the SVC in patients with normal circulation

The majority of the patients belonging in the fourth category are operated for a cancer of the right lung. In these patients, the preoperative diagnostic evaluation was unable to demonstrate SVC invasion, either because of failure or because of no existence and the anesthetic preparation and treatment is as usual, not including special measures. In case of invasion, the intraoperative findings provide a better evaluation and new decisions must be made, including a completely new approach, for the patient's management under the lowest risk. If the surgeon feels confident to proceed to clamping SVC without supportive measures, the anesthesiologist must strongly oppose and ask for time to prepare (heparin administration, vasoactive drug and blood saver preparation, adequate monitoring modalities,) or at least for time to establish a venous line in the lower extremities. Sometimes, acute and unexpected bleeding from the SVC, with or without tumor invasion, leads to the decision of clamping. Under these circumstances, the patient is completely unprotected as the normal collaterals are unable to serve the flow of the upper compartment (*see: pathophysiology & clinical manifestations*). From our experience, clamp placement is followed by abrupt drop of arterial

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pressure and $EtCO_2$, cyanosis and tremendous swelling. The CVP trace in the monitor is lost as the tip of the central venous catheter is usually clamped. In case of a Swan-Ganz catheter, the pulmonary artery pressure wave-form is also lost but the SVO_2 value may be still present and sometimes falsely elevated as the collapsed vasculature brings it in wedge position. Manipulations of SVC, especially stretching near its region of entrance in the right atrium, may increase heart rate. Complex unencapsulated nerve endings forming nets in this region have been demonstrated in a variety of mammals[**44**]. From the other hand, acute reduction of venous return may also be arrhythmiogenic. In this situation, the administration of drugs and volume by the upper compartment's peripheral venous lines must be stopped, as they are not reaching target organs. Urgent administration of vasoactive drugs (i.e. adrenaline) can be done by the endobronchial lumen of the double-lumen tube to the ventilated/perfused lung parenchyma. As the pericardium is usually opened, it is of extreme importance to ask the surgeons to establish a right atrium line. It can solve the problem of reaching central circulation. The anesthesiologist must act on diminishing cerebral metabolic requirements and on improving cerebral perfusion. Periodical unclamping of the SVC may allow venous decongestion. Blood aspiration from the proximal port of the central catheter and readministration by the right atrium line establishes conditions of a manually aided venovenous bypass. A similar technique of decompressing the SVC by active manual aspiration has been described in infants in whom off-pump bidirectional Glenn shunts were performed[**45**]. It must be remembered, that brain cannot tolerate for long time conditions as the previously described (*see: pathophysiology*).

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Λέξεις κλειδιά: superior vena cava; clamping, syndrome

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