# Superior Vena Cava Replacement in Thoracic Malignancies: a Review of Literature and Experience of 33 Procedures

Victor A. TARASOV, *Professor, Head of the Department of Thoracic Surgery, Medical Academy of Postgraduate Education, St.Petersburg, Russia*
Evgeny S. POBEGALOV, *Associate Professor, Department of Thoracic Surgery, Medical Academy of Postgraduate Education, St.Petersburg, Russia*
Vladimir V. STAVROVIETSKIY, *Chief of the Thoracic Surgery Unit, Municipal Hospital No. 26, St.Petersburg, Russia*
Yuriy K. SHAROV, *Associate Professor, Department of Thoracic Surgery, Medical Academy of Postgraduate Education, St.Petersburg, Russia*
Maria V. VINOGRADOVA, *Assistant Professor, Department of Thoracic Surgery, Medical Academy of Postgraduate Education, St.Petersburg, Russia*

## 1. Aetiology of superior vena cava syndrome

The first description of the superior vena cava syndrome (SVCS) dated by 1757 belongs to Dr. William Hunter of London who observed this condition in a patient with a syphilitic aneurysm of the thoracic aorta. This condition remained one of the commonest causes of SVCS up to the middle of the XXth century. After that, certain pathomorphosis of benign and malignant diseases has crucially changed the structure of conditions causing SVCS.

### 1.1. Dynamic changes in the aetiology of SVCS.

Changes in nosological structure of the diseases underlying SVCS during the XXth century are presented in Table 1.

*Table 1. Changes in the aetiology of SVCS during the XXth century*

| Author(s) | Year | Number of patients | Malignant tumours, including: | | | | Thoracic aortic aneurisms | Other benign conditions |
|---|---|---|---|---|---|---|---|---|
| | | | Lung cancer | Mediastinal tumours | Metastases | Total | | |
| McIntire F.T., Sykes E.M., | 1949 | 502 | | | | 38% | 30% | 32% |
| Kamiya K. et al. | 1967 | 734 | 71,0% | | 2,6% | 73,6% | 4,6% | 21,8% |
| Banker V.P., Maddison F.E. | 1967 | 438 | 65% | 13% | 4% | 82% | 18% | |
| Schraufnagel D.E. et al. | 1981 | 107 | | | | 85% | 15% | |
| Ahmann F.R. | 1984 | 1980 | 85% | 15% | | 90% | 10% | |
| Fincher R.E. | 1987 | 39 | | | | 87% | 13% | |
| Chen et al. | 1990 | 45 | 58% | 19% | 4% | 94% | - | 6% |

In the first half of the XXth century, 1/3 of SVCS cases were caused by thoracic aortic aneurisms, 1/3 – by malignant neoplasms, and the remaining third – by chronic fibrosing mediastinitis (mainly of tuberculosis and hystoplasmosis origin, less by primary idiopathic one) (McIntire F.T., Sykes E.M., 1949). Since the beginning of the 50ies, on one

hand, successes in treatment of syphilis led to disappearance of its visceral forms, while medico-social combat with granulomatous diseases – tuberculosis and hystoplasmosis – sufficiently decreased the incidence of chronic fibrosing mediastinitis. On the other hand, rapid growth of incidence of thoracic malignancies (first of all, lung cancer and mediastinal neoplasms) is being observed since the 40ies – 50ies, and as a result they cause 78 – 97% cases of SVCS in our days [1, 2, 3]. The rest of modern cases are still being caused mainly by chronic fibrosing mediastinitis though incidence of benign superior vena cava (SVC) thrombosis is growing last years – due to prolonged central venous catheterisation and intravascular electrode placement for cardiac pacing [3].

Among rare causes of SVCS, thyroid cancer [4, 5, 6], primary leiomyosarcoma [7, 8, 9] and lymphoma of the SVC [10], primary mediastinal amelanotic melanoma [11], intravascular metastatic melanoma of the SVC [12], right atrial angiosarcoma [13] have been reported so far. As for benign lesions, plasmocytomas of inflammatory origin [14], benign fibrous pleural mesothelioma [15], Beck sarcoidosis [16], intravascular lipoma of the SVC [17] have also been reported to cause SVCS. There is also a description of prolonged intravascular growth of invasive thymoma through the lumens of the thymic vein, IV (IV) into the SVC and further on into the right atrium [19].

### 1.2. SVCS as a complication of thoracic malignant neoplasms.

As we have already mentioned in the previous paragraph, the most common causes of the SVCS are now lung cancer and invasive mediastinal tumours (first of all, those located in the upper anterior mediastinum).

According to data of P.Thomas et al. [20], 6% cases of right-sided lung cancer present with invasion of the SVC. L.Szur and L.L.Bromley (1956) found invasion and obstruction of the SVC in 104 (5,2%) of 2,000 lung cancer patients. I.Steinberg and C.T.Dotter (1952) evaluated incidence of SVCS in lung cancer as much as 10-15%, B.T.Le Roux [21] - 4,6%, W.Stanford and D.B.Doty [22] - 6-7%, I.Robinson and J.Jackson [23]- в 5%. According to data of P.Dartevelle et al. [24], SVC invasion in lung cancer may be due to:
-   a tumour of the right upper lobe;
-   a tumour localized in the pulmonary hilus;
-   paratracheal lymph node metastases.

As regards mediastinal neoplasms, there is much less analysis of incidence of SVC invasion in literature, due to comparative rarity of this kind of pathology. J.Remy et al. [25] found reports of SVCS in 55 (9,2%) of 596 thymomas described in literature by the moment of their publication. It would be logical to suppose the incidence of SVC invasion to be higher than the registered incidence of clinically presented SVCS.

## 2. Pathophysiological peculiarities of SVCS.

The block of venous outflow from the head and the upper half of the body is known to cause a number of pathophysiological effects:
-   decrease in venous return to the right ventricle;
-   decrease of cardiac output resulting from the previous effect;
-   systemic hypotension resulting from the previous effect;
-   increase of venous pressure in SVC system which may cause thrombosis in cerebral vessels.

The result of the last two effects is decrease of arterio-venous pressure gradient in cerebral vessels, which may cause irreversible changes of the brain.

Yet, if the occlusion of the SVC grows gradually, most patients are able to compensate these events by developing more or less enough collateral venous outflow [1] through the following routs:

- the system of azygos vein (provided the latter is patent);
- the system of internal mammary veins and their anastomoses with upper and lower epigastric veins – into the system of external iliac veins;
- vertebral veins – into the inferior vena cava [26].

Due to elastic walls of the SVC and to low pressure in its lumen, the moment of its invasion is commonly preceded by a rather prolonged period of external compression [27], and this time is usually enough for collateral venous outflow to be formed. Owing to the compensating mechanisms mentioned above, patients with SVCS die not so of this syndrome but mostly of other consequences of the principal disease [28]. Besides that, patency of SVC in advanced lung cancer is usually preserved for a long time despite its invasion [29].

Nevertheless, only 10 – 20% of patients with malignant SVCS survive more than 2 years [30]. Median life duration of patients with malignant tumours of the thorax after onset of SVCS does not exceed 10 months [31].

## 3. Clinical presentation and diagnosis of SVCS.

The clinical picture of SVCS described by William Hunter in 1757 is so typical that physical examination is commonly believed to be quite enough for its diagnosis [32]. Yet, it is necessary to keep in mind that in a number of patients who develop adequate collateral venous outflow clinical presentation of SVCS may be significantly "washed out"; in some cases, there may be no typical complaints at all. Pemberton's manoeuvre[1] or other simple tests[2] may help to reveal SVCS in these occult cases.

Diagnostic techniques are needed not so to reveal SVCS but rather to confirm this diagnosis, to provide quantitative evaluation, and – which is of importance for treatment planning – to detect accurately the level and extent of central venous occlusion.

Phlebography, which was the "golden standard" in the diagnosis of lesions of SVC system up to the middle of the 80ies, nowadays seems to present mostly historical interest. Nevertheless, classification of SVC obstructions developed on the basis of phlebographic data still has a certain practical significance up to our days; thus, we'll present this classification here.

---

Classification of SVC obstructions by phlebographic data (W.Stanford, D.B.Doty, 1986):

Type I: partial (less than 90%) SVC obstruction with preserved patency at the level of the azygos vein.

Type II: complete or almost complete (90-100%) SVC obstruction with preserved patency at the level of the azygos vein.

Type III: complete or almost complete (90-100%) SVC obstruction with retrograde blood flow in the azygos vein system.

Type IV: complete SVC obstruction with obstruction of one or more of its tributaries, including the azygos vein.

---

Now, it is needless to speak about the real revolution in surgical diagnosis as a whole, and in the diagnosis of SVC lesions in particular, produced by modern techniques of ray diagnosis, and first of all - magnetic resonance imaging presenting the opportunity of non-invasive angiography.

---

[1] The technique of this maneuver is the following: the patient is asked to raise his arms upwards and stay in this position for several minutes. In presence of SVC occlusion, characteristic signs of SVCS (cyanosis of the face and neck, tension of jugular veins, injection of conjunctival vessels, and so on) appear immediately [33].

[2] Signs of SVCS may appear after the patient performs 15 – 20 fast leans forward.

## *4. Historical and modern approaches to management of malignant SVC lesions.*

For the most part of the past century were the physicians trying to help patients with clinically advanced SVCS – first of all for benign cases, but in a number of cases for malignant ones as well. After general successes of surgery, anaesthesiology, oncology, and with appearance of new minor-invasive surgical technologies, a wider problem arose before surgeons and oncologists: treatment of malignant lesions of the SVC and its tributaries, without reference to presence or absence of SVCS. Yet, it is impossible to imagine attempts of solving this problem without the precedent stage – development of methods to manage SVCS itself.

### 4.1. Historical aspects of SVCS correction

As SVCS is a condition caused by these or those mechanical reasons: extrinsic compression, obstruction by a thrombus or a tumour, - it is natural that the problem of its correction was being solved mainly by surgeons.

C.Gluck made first experimental attempts of venous replacement in 1898, followed by A.Carrel and C.C.Guthrie in 1906. The first to suppose the possibility of surgical treatment for SVCS was P.G.S.Killen (1917). F.Gerbode et al. (1934) demonstrated the possibility of direct vessel-to-heart anastomoses in experiments with mice, and advocated these methods for management of SVC obstructions.

Later on, a tremendous experimental work was carried out in a number of surgical clinics to find optimal ways of SVCS correction. In the field of surgical strategies, several principal approaches to restoration of venous flow in SVC system resulted:
1) External decompression
2) Bypass:
   a) internal (intrathoracic)
   b) external (extrathoracic)
3) Thrombectomies
4) Plastic procedures
5) Major resections with immediate reconstructions
6) Percutaneous endovascular angioplasty and stenting

The majority of approaches listed above demanded development of optimal materials for shunting, plastic reconstruction, or replacement of SVC. In experimental SVC reconstructions, R.A.Deterling (1954) used homologous aorta, E.G.Aschburn (1956) and A.Riberi, T.C.Moore (1958) – heterogeneous blood vessels, T.W.Jones (1958) and L.R.Sauvage (1960) – autologous pericardium, R.S.Todd (1963) – autologous peritoneum and even homologous trachea, C.A.Brea (1965) – autologous fascia, J.L.Chevrier (1964) – autologous derma, P.C.Petropoulos (1963) - homologous dura mater. Perspectives of synthetic materials were studied in experiments by R.A.Deterling and S.B.Bhonslay (1955), M.Y.Peter et al. (1960), T.C.Moore et al. (1960), R.J.Botham et al. (1960), T.Hasegawa et al. [34], A.C.Leshnower et al. [36]. By the middle of the 60ies, enough laboratory experience was accumulated to transfer experimental works into clinical surgery. L.R.Sauvage and R.E.Gross used the first compound autovenous graft for SVC replacement in experiment in 1960.

It was only 10 years later that Y.Fujiwara et al. [36] published the results of first experiments with polytetrafluorethylene (PTFE) blood vessel grafts, which later on contributed greatly to extension of possibilities of surgery for great vein lesions in clinic. Having been tried in clinical surgery, this model appeared to be quite suitable [37, 38, 39, 40]. H.Masuda et al. [41] suggested enforced PTFE grafts for venous reconstruction.

Thus, a solid basis for introduction of auto-and alloplastic venous reconstructions into surgical oncology has been created.

**External SVC decompression.** The world-first successful surgical procedure for SVCS correction was external decompression of the vein performed by W.Ehrlich et al. in 1934. Further on, this method of SVCS correction became a common procedure in a lot of benign SVC obstructions, and also in management of thoracic malignancies without direct venous invasion.

**Internal bypass.** The first anastomosis between SVC system and the right atrium was performed by F.T.McIntire and E.M.Sykes в 1949. D.A.Cooley and G.C.Hollman in 1964 were the first to perform a "side to side" anastomosis between the azygos vein and inferior vena cava to correct SVCS[1]. K.P.Klassen et al. (1951) were the first in clinical application of autovenous graft between the azygos vein and SVC. D.J.Effeney et al. [43] were the first to use autopericardial blood vessel graft for SVCS correction in clinic.

**External bypass.** Subcutaneous sapheno-jugular bypass was first used in clinic by R.Schramel and H.Olinde (1961). Later on, their idea found support and further development due to certain advantages of external shunting before internal one: this kind of shunt requires neither general anaesthesia nor thoracotomy; morbidity and mortality rates are minimal; thrombosis of such a shunt is rare because of gravitation effect and elevated blood pressure in SVC system; patients do not require anticoagulants after the procedure – low-molecular dextran infusion is enough [1, 44]. B.A.Hoak et al. [31] used subcutaneous axillo-axillar and axillo-femoral bypass by synthetic grafts for palliation of SVCS in a patient with malignant invasion of SVC and left subclavian vein, claiming this method to be fast, technically simple, safe, and feasible under local anaesthesia if necessary. Besides that, intrathoracic SVC shunt in conditions of a persisting malignant tumour as well as fibrosing mediastinitis is at risk of secondary extrinsic compression from the same pathological process [45].

It is necessary to take into consideration that bypass procedures are merely a palliation and do not contribute to life duration of oncological patients [1]. Thus, extrathoracic bypass is indicated in malignant SVCS only in case of impossibility or ineffectiveness of more radical treatment [46].

**Thrombectomies.** The first endovenectomy for SVC thrombosis was performed by T.O'Neil in 1954. Nowadays, removal of the so-called malignant thrombi from great veins is becoming a common procedure in surgical oncology.

**Plastic procedures.** The first wedge resection of the SVC in malignant invasion was performed by L.G.Lome and I.M.Bush in 1972 [47]. Now, wedge and tangential SVC resections are widely used in thoracic surgery; the venous defect is usually closed with a suture or a patch (most often – of autopericardium) [20, 24, 48].

**Wide resections with consecutive reconstructions** demanded maximum efforts for development of both surgical / anaesthesiological techniques and materials for reconstruction. A doubtless advantage of this approach, especially important for management of malignant SVCS, is a combination of natural venous flow restoration with an oncologically radical excision. Disadvantages are comparatively high postoperative mortality and morbidity rates.

The first attempt in the USSR to replace the SVC as a stage of combined pneumonectomy for locally advanced lung cancer, though unsuccessful, was made in 1960 by E.P Dumpe.

In 1983, R.P.Andersson and W.I.Li [49] became the first to perform a successful segmental resection of the SVC with immediate reconstruction of the removed segment in a patient with recurrent upper anterior mediastinal leiomyosarcoma. As their patient did not

---

[1] In 1975, D.B.Miller [42] modified Cooley's technique by performing "end to side" anastomoses between the azygos vein and inferior vena cava in 2 patients with SVCS.

present with signs of SVCS before surgery, this procedure may be considered to be the first successful attempt of radical surgery for a malignant neoplasm invading the SVC.

Overall, the following methods have been used in clinical surgery so far for SVC bypass and replacement:
- autovenous grafting with a non-modified femoral vein Gladstone, 1965), spiral autovenous grafts [50, 51, 52]; A.W.Lee et al. [53] were the first to use the reversed azygos vein to reconstruct the SVC (resected for lung cancer) by anastomosing the peripheral end of the azygos vein and the confluence of IVs;
- free [43, 54] and pedicled [55] autopericardial grafts;
- synthetic grafts made of Dacron and Teflon [27, 56];
- PTFE grafts [57, 58, 59].

Till the end of the 80ies, the choice of material for reconstruction of SVC and IVs had been made between PTFE grafts, autopericardium and autovein; now, modern PTFE grafts have fully conquered pericardium and are successfully concurring with autovenous grafts [3, 20, 29, 59]. Doubtless advantages of PTFE grafts are their immediate readiness for application, technically simple adaptation by length and diameter, resistance for extrinsic compression; with time, their inside surface becomes delineated with neointima. Disadvantages seem to be low resistance for infection (though theoretical) and risk of thrombosis (though infrequent). Autovenous grafts do not bear these disadvantages but certain time is needed for their preparation during surgery, thus prolonging complex procedures that are significantly long by themselves. As for pericardium, it is resistant for infection, a graft may be prepared of this material in a rather short time, but it will never become delineated with vascular neointima.

**Percutaneous endovascular angioplasty and stenting.** The first attempt of SVC stenting for malignant obstruction made in 1986 was complicated by early thrombosis [60]. The first successful stenting was reported a year later by J.Rosch et al. [61]. In our days, when the stocking trade of interventional radiologists includes a variety of self-expanding stents of enough diameters to maintain a nearly normal lumen of the SVC, the method seems to be optimal when treatment options are limited with palliative venous outflow restoration only. Contraindications are few: complete stable SVC obstruction; severe coagulopathy; chronic heart diseases [62].

## 4.2. Evolution of approaches to management of malignant SVC lesions

During decades, a sceptical attitude towards the possibilities of radical surgery in cases with malignant SVC invasion dominated in surgical oncology. This conservatism was based upon poor prognosis after surgical removal of these advanced tumours, lack of material suitable for SVC replacement, and on a possibility of compensation of SVCS by collateral venous outflow while patients are being treated non-surgically – by chemo- and radiotherapy [3, 24].

M.Turina et al. [2] in 1982 considered surgery to be contraindicated in SVCS of malignant origin reserving this option only for cases of thrombotic or long-standing SVCS. Even in these benign conditions, they advocated internal bypass using an autologous vein or PTFE graft as a method of choice. Thus, all the spectrum of malignant SVCS had to be subjected to radio- and chemotherapy.

Yet, radiotherapy, which has traditionally supposed to be the "golden standard" in treatment of malignant SVC obstructions, is really effective in 90% of these patients – but only within the first week; afterwards, 20 – 50% patients present with recurrence. In case of combination of invasion and thrombosis of the SVC, radiotherapy is mostly ineffective [23, 46].

In 1990, J.C.Chen [28], observing 42 patients with malignant SVCS managed with radiotherapy, reported median survival time of 3 months, and mean SVCS-free time of 4

weeks. The cause of death in the majority of his patients appeared to be respiratory failure due not so to SVCS itself but mostly to other complications of locally advanced tumours.

Similar observations made M.Zembala et al. [54] conclude in 1986 that surgical methods of management are more effective in malignant SVCS than radiotherapy.

Dissatisfaction with non-surgical methods of management of malignant SVCS, on one hand, and a number of brilliant experimental works which developed surgical technologies for effective reconstruction of great veins, on another hand, were the reasons of certain changes. Since 1979, some clinics of the world started to regard patients with invasion of the SVC by primary malignant tumours of the lung and mediastinum as candidates for radical surgery [24]. First results appeared to be surprisingly encouraging.

## 5. Modern surgical strategy in malignant SVCS.

### 5.1. Indications for surgery in malignant SVCS.

Clinical experience accumulated by today presents the opportunity of significant extension of operability for patients with malignant thoracic tumours invading great veins. P.E.Magnan et al. [3] suppose surgical reconstruction of SVC system to be indicated in patients with expected life duration of more than 6 months who fail to respond to radiotherapy and/or chemotherapy. T.Jeanfaivre et al. [63] do not consider malignant invasion of the SVC to be contraindication for radical surgery. The same aggressive surgical approach is being demonstrated in a number of thoracic clinics of Japan and Italy. Thus, today malignant invasion of great veins in the mediastinum has no principal difference with other types of local advance of malignant neoplasms: SVC replacement is becoming a standard procedure in thoracic surgical oncology [24].

**5.1.1. Indications in invasive mediastinal neoplasms.** Malignant mediastinal tumours may be removed safely enough even in cases of invasion of chest organs and structures. Radical removal is quite important in achievement of satisfactory long-term survival time [48]. SVC resection presents new potentials of radical surgery in invasive upper anterior mediastinal masses. Acceptable postoperative mortality rate and unexpectedly high survival time justify the attempts of development and standardization of radical surgical procedures with great vessel reconstruction [24].

**5.1.2. Indications in lung cancer.** In modern staging scheme for lung cancer, mediastinal invasion is referred to IIIB stage which is generally characterised by poor prognosis and considered to be a contraindication for surgery. At the same time, most of patients with mediastinal spread of lung cancer are known to die not so of generalization of the disease but rather of local complications caused by the primary tumour, which advocates aggressive surgical approach [20]. Other advantages of this strategy are:
- complete removal of symptoms of venous flow block;
- prevention of local recurrence (only 13% local recurrence was reported after radical surgery for lung cancer with mediastinal spread).

Besides that, non-surgical methods – chemo- and radiotherapy – fail to provide significant life duration in most of these cases [20]. L.Spaggiari et al. [59] lined out the following advantages of radical surgery for locally advanced lung cancer with SVC invasion:
- possibility of cure;
- good palliative effect in any case;
- acceptable postoperative mortality rate (12% as reported by authors).

Yet, in the light of long-term results, major surgery of advanced lung cancer is now commonly considered to be of benefit only in T4N0-1 cases, because only in these patients may combined surgery provide effective control of the process: 5-year survival rate of 36% was shown after radical surgery for NSCLC T4N0-1 [64]. In this regard, a number of

authors underline the necessity to differ SVC invasion by the primary tumour (T4) of invasion by mediastinal metastases (N2-3), the last being considered by the majority to be inoperable due to poor long-term results.

Thus, conditions of operability for lung cancer invading the SVC which justify resection of the latter may be presented at the moment in the following way:
- morphologically proved SVC invasion;
- absence of mediastinal lymph node metastases (N limited to 0-1);
- absence of extrathoracic metastases.

## 5.2. Contraindications for surgery in malignant SVCS.

Contraindications for SVC resection are few:
- lung cancer N2-3 or M1;
- invasion of the left phrenic nerve (one usually has to sacrifice the right phrenic nerve when resecting the invaded SVC);
- impossibility of radical surgical procedure (R0)
  [24, 48, 58].

Yet, we should mention that no one of these contraindications seems to be absolute, which will be discussed below.

Another matter is a question of contraindications for reconstruction of the SVC and its tributaries after their resection. Reconstruction in case of extended distal venous thrombosis is not only senseless but dangerous as well. It is also risky in patients with well-developed collateral venous outflow which may slow down the blood flow through a graft, thus contributing to possible thrombosis [24, 48, 58].

## 5.3. Surgical technique in malignant invasion of the SVC and IVs.

*5.3.1. Choice of access.* Optimal surgical access in most cases of mediastinal neoplasms, including the cases demanding pulmonary resections, seems to be median sternotomy [24]. Additional cervicotomy provides full control of all the SVC system [3, 20]. The same access is preferential for bypass procedures without removal of neoplasms [3]. Yet, it is not enough in some cases: E.A.Bacha et al. [48] had to perform thoracotomies or combined accesses (sternotomy plus thoracotomy, bilateral or clamshell thoracotomy, cervicomanubriotomy) in 21% patients operated for invasive mediastinal masses involving SVC system.

Optimal access for invasive lung cancer is usually thoracotomy because IVs are commonly intact and free for manipulations in these cases [3, 24, 29]. Cross-section of the SVC before division of pulmonary vessels facilitates access to the retrocaval portion of the pulmonary artery [29]. L.Spaggiari et al. [59] use posterolateral or lateral thoracotomy in lung cancer invading the SVC, choosing median sternotomy if an IV is involved. K.Nakahara et al. [65] prefer combined access: sternotomy plus thoracotomy, adding local supraclavicular incisions when manipulations with the arteries are also needed. S.Larsson and V.Lepore [66] advocate refusal of thoracotomy in order to preserve as much natural collateral venous anastomoses as possible to maintain collateral venous outflow; yet, considering reconstruction of central venous outflow, this point does not seem to be of importance.

*5.3.2. Choice of method for reconstruction of SVC system.* After resection of the SVC and/or IVs, venous outflow may be restored by different ways, depending greatly upon the extent of resection. Small wedge and tangential resections may be completed with a simple suture; after larger wedge resection, an autovenous or autopericardial patch may be necessary [59].

Extended resection of the SVC with immediate reconstruction is indicated in extended invasion [24] occupying more than 50% of its circumference [3, 59] when wedge or tangential resections with suturing or patching of the defect are not feasible [48].

***5.3.3. Peculiarities of technique for SVC grafting.*** The goal of central venous reconstruction is not only restoration of venous outflow but also prevention of venous thrombosis. As for possible complications of venous reconstruction, prevention of thrombosis is much more actual than prevention of bleeding [56].

Blood flow through the graft is optimal when its diameter matches exactly the diameter of the vein [67]. S.S.Mikhailov and I.A.Pismenov [68] found the conical shape of the venous prosthesis to be optimal in their experimental work. R.B.Avasthi and K.Moghissi [69], based also on experimental data, formulated the following conditions for long-term patency of a venous graft:

1.  Non-thrombogenous internal surface.
2.  Resistibility to extrinsic compression. (T.Moore and I.Mandelbaum showed in 1963 that enforcement of a venous graft prevents its occlusion [35].)
3.  Non-thrombogenous suture line at the site of anastomosis.
4.  The lumen of the graft slightly exceeding the lumen of the vessel. (As demonstrated by J.Heimburger et al. in 1960: the more is the diameter of the graft, the less is the risk of occlusion [35]; this is related to delineation of the graft with neointima, which may thicken with time, thus obstructing the lumen [20].)
5.  Good blood flow and sufficient internal pressure in the graft. (As stated by H.Mitsuoka et al. (1966), H.C.Stancel (1964), W.Dale and H.Scott (1963), the more intensive is blood flow through the graft, the less is the risk of occlusion [35]. W.Dale and H.Scott (1963) suggested ligation of the azygos vein to accelerate blood flow in the SVC after its reconstruction.)

It was stated by H.Mitsuoka et al. (1966) that the less is the length of the graft, the less is the risk of occlusion [35]. Some authors advocate creation of arterio-venous fistulas with subclavian or axillary artery to support intensive blood flow in the long venous grafts [70, 71].

In 1992, S.Larsson и V.Lepore, being the advocates of autovenous reconstruction, based on their clinical experience of 12 SVC reconstructions, pointed out the following technical principles [66]:

-   The optimal method for reconstruction of the SVC system is separate autovenous reconstruction of both IVs anastomosing the grafts with the right atrium (the advantages of this kind of anastomosis are: simple technique; convenient cross-clamping of the auricle; large lumen [69]);
-   The sum of diameters of two autologous vein grafts should be approximately equal to ½ of diameter of the SVC.

Among synthetic venous grafts, PTFE graft proved to be the best. Its advantages are: technically simple application; resistibility to kink and extrinsic compression; resistibility to postsurgical irradiation [3, 20]. P.Dartevelle et al. [24, 29, 58], based on their clinical data, suggested the following guidelines for SVC allografting:

-   The diameter of a PTFE graft for SVC reconstruction should not be less than 18 – 20 mm.
-   In case of invasion of IVs, reconstruction of only one of them may be enough, providing more intensive blood flow through the graft.
-   If both IVs have to be reconstructed, two separate grafts are at less risk of thrombosis than a single Y-shaped graft.
-   Enforced PTFE graft 10 – 14 mm in diameter is preferable for reconstruction of IVs to avoid kink and extrinsic compression.
-   The peripheral anastomosis should be formed prior to the central one.

- If the airways were opened during the procedure (i.e. pneumonectomy), the graft should be covered with a pleural flap to prevent infection.

P.E.Magnan et al. [3] underline the importance of the peripheral anastomosing within the intact tissues above the level of venous obstruction, preferring to perform central anastomosis of the graft with the right auricle, as many others. They suppose anastomosing of the graft with a thrombectomized portion of the vein to elevate risk of rethrombosis. Most often these authors restored venous outflow by reconstruction of the left IV; more rarely SVC, right IV, subclavian and internal jugular veins have been reconstructed. L.Spaggiari et al. [59] also use to reconstruct the left IV only, in order to avoid blood flow slowdown which may cause thrombosis of the graft.

In cases of initial total obstruction of the SVC, even prolonged manipulations with this vessel produce no haemodynamic disturbances at all, but cross-clamping of partially or totally patent SVC may cause undesirable pathophysiological effects stated above in Paragraph 2: decrease of venous return, decrease of cardiac output, systemic hypotension.

Increase of venous pressure in SVC system may cause thrombosis in cerebral vessels or decrease of cerebral arterio-venous pressure gradient; the latter may result in irreversible changes of the brain. To avoid these effects, H.Yoshimura et al. [72] suggested temporary bypass of the SVC for the time of its reconstruction. K.Nakahara et al. [65] consider central venous pressure monitoring to be necessary, and use temporary bypass only if central venous pressure after cross-clamping of the SVC exceeds 40 cm of water column. To prevent early thrombosis of the graft, the same authors apply heparinization of 100 units per kg of body weight immediately before reconstruction of the vein, and avoid placement of central venous lines near the graft.

It is worth mentioning that a number of authors have abandoned temporary SVC bypass or do not suppose it to be of importance. According to their data, cross-clamping of the SVC for the average of 53 – 62 minutes is tolerated without complications or neurological symptoms even by patients with initially patent SVC [20, 24, 59]. As for haemodynamic effects, they may be prevented by prior excessive fluid infusion, vasopressor injection, adequate anticoagulation, and decreasing of time of SVC cross-clamping [29, 48, 59]. S.Larsson and V.Lepore [66] reconstruct SVC tributaries separately, starting with the more obstructed IV: in such an order of actions, venous outflow in the SVC system does not become decreased during the procedure, making temporary bypass needless.

***Prevention of postoperative thrombosis.*** There is an opinion [23, 73] that no postsurgical anticoagulation therapy is needed if the diameter of venous graft and the blood flow through the graft are adequate. Yet, the majority of authors advocate prolonged (not less than 3 months) postoperative therapy with indirect anticoagulants [66]. Optimal duration of anticoagulation is still questionable, varying from 3 [65] to 6 months [24, 48]. Placement of central venous lines inside or near the graft is discommended in the early and long-term postoperative period [24, 58].

## 5.4. Results of surgery.

***5.4.1. Immediate results.*** Despite initially severe condition of the patients, high grade of surgical aggression in combined procedures, published data of postsurgical mortality and morbidity seem to be acceptable. Overall mortality after combined procedures with resection of SVC and IVs for advanced thoracic malignancies varies from 0 to 12%, morbidity - from 0 to 36%. Table 2 presents immediate results of surgery for malignant SVCS reported by different authors.

*Table 2. Immediate results of surgery for malignant SVCS.*

| Authors, year of publication | Pathology | Number of patients | Mortality, % | Morbidity, % |
|---|---|---|---|---|
| P.G.Dartevelle et al., 1991 | Mediastinal neoplasms, lung cancer | 22 | 4,5 | 4,5 |
| P.Thomas et al., 1994 | Lung cancer | 15 | 7 | 27 |
| P.-E.Magnan et al., 1994 | Lung cancer | 9 | 0 | 20 |
| N. Shimizu et al., 1994 | Invasive thymomas | 20 | 10 | - |
| P.G.Dartevelle et al., 1997 | Lung cancer | 14 | 7,1 | 21,4 |
| S.Watanabe et al., 1999 | Mediastinal neoplasms, lung cancer | 37 | 0 | 0 |
| L.Spaggiari et al., 2000 | Lung cancer | 25 | 12 | 36 |

Morbidity of venous reconstruction itself is comparatively rare, being related mainly to early thrombosis. P.Dartevelle et al. [24] observed only 1 (4,5%) graft thrombosis; L.Spaggiari et al. [59] reported 2 cases (8%), both of them occurred after wedge SVC resections with simple suturing. As supposed by P.Dartevelle et al. [24], besides thrombosis of the graft, there is a "theoretical" risk of its infection with spread to mediastinitis or pleural empyema, but there are no reports of these complications in literature so far.

*5.4.2. Long-term results.* By the opinion of all the authors having the experience of surgery in malignant lesions of major thoracic veins, combined procedures in this condition not only increase life duration and disease-free interval in most cases, but also improve the life quality level by radical elimination of the most distressing complications of the disease. Long-term results of surgery for malignant SVCS are presented in Table 3.

*Table 3. Long-term results of surgery for malignant SVCS.*

| Authors, year of publication | Pathology | Number of patients | Survival rate, % | | | | Life duration, months | |
|---|---|---|---|---|---|---|---|---|
| | | | 1 year | 2 years | 3 years | 5 years | Median | Maximal |
| P.G.Dartevelle et al., 1991 | Total | 22 | | | 57 | 47 | 25 | 95 |
| | Mediastinal neoplasms | 16 | | | 60 | 60 | 36 | 95 |
| | Lung cancer | 6 | | | 50 | 0 | 13 | 51 |
| P.Thomas et al., 1994 | Lung cancer | 15 | 46,7 | 32 | | 24 | 8,5 | 62 |
| P.-E.Magnan et al., 1994 | Lung cancer | 9 | 70 | 25 | | 12,5 | 17 | 62 |
| N. Shimizu et al., 1994 | Invasive thymomas | 20 | | | | | 30,8 | 96 |
| P.G.Dartevelle et al., 1997 | Lung cancer | 14 | | | | 31 | | 65 |
| K. Yagi et al., 1996 [77] | Invasive thymomas | 12 | | | | | 41,3 | 92 |
| S.Watanabe et al., 1999 | Lung cancer | 28 | | | 26,2 | 11,2 | | |
| | Mediastinal neoplasms | 11 | | | | 45,5 | | |

Results of non-radical (R1-2) surgery are definitely worse than those of radical (R0) ones. This is more characteristic for palliative bypass procedures than for tumour-reductive ones. W.Stanford и D.B.Doty [22] reported median life duration of 10.8 months in 7 cases of intrathoracic bypass between an IV and right auricle for malignant SVCS; yet it is worth mentioning that the same figure for the same patients who did not undergo surgery made up only 1.4 months. 4 patients observed by M.Okada et al. [78] were completely relieved of signs of malignant SVCS by bypass procedures but died in terms of 1 to 7 months

postsurgically. The comparison of results of radical and tumour-reductive procedures is presented in table 4.

*Table 4. Long-term results related to surgical radicalism.*

| Authors, year of publication | Pathology | 5-year survival rate, % | | Life duration, months | | | |
|---|---|---|---|---|---|---|---|
| | | | | Median | | Maximal | |
| | | R0 | R1-2 | R0 | R1-2 | R0 | R1-2 |
| K.Fukushima et al., 1994 | Thymic neoplasms | | | | | 139 | 26 |
| R. Tsuchiya et al., 1994 | Lung cancer | 19 | 0 | 13,8 | 6,5 | | |
| P.-E. Magnan et al.1994 | Lung cancer | | | 24 | 14 | 62 | 20 |
| T.Takahashi et al.,1999 | Lung cancer | 18 | 0 | | | | |

At the same time, I.Robinson and J.Jackson [23] reported median life duration of patients with malignant SVCS to be less than 6 weeks without treatment, and 6-7 months with chemo- and radiotherapy. Only 1% of these patients survive 12 months since the time of diagnosis. Data presented by M.Oudkerk et al. [62] are still more pessimistic: median life duration after palliative stenting of malignant SVC obstructions made up 3 months.

Both radical and non-radical surgical procedures remove SVCS immediately and steadily in all the cases, thus improving life quality [3]. PTFE grafts were reported to preserve their patency despite even postsurgical mediastinal fibrosis being aggravated by radiotherapy [24]. There are only few reports of late thrombosis of SVC and IV grafts, with occurrence varying from 5 to 14% [24, 56].

## 6. Our experience of surgical treatment of 33 patients with malignant invasion of SVC and IVs.

### 6.1. Characteristic of patients.

33 patients (21 male and 12 female) aged 23 – 75 (average 52.4) with locally-advanced and metastatic thoracic malignant neoplasms complicated by invasion of SVC system underwent surgery at the Department of Thoracic Surgery of St.Petersburg Medical Academy of Postgraduate Education. 22 patients had various malignant tumours of the mediastinum (invasive thymomas, thymic carcinoma, angiosarcomas), 8 patients – lung cancer, 2 patients – mediastinal metastases of renal-cell and nasopharyngeal carcinoma, and 2 patients - mediastinal lymphoma. 14 patients presented with advanced symptoms of SVCS, 5 of them presented with signs of major airway obstruction.

SVC was invaded in 20 cases, IVs – in 24, jugular vein – in 1, subclavian artery and vein – in 2 patients with Pancoast tumour, common carotid artery – in 2 cases. Overall 52 major vessels of the chest (48 of them being veins) were invaded in 33 patients.

### 6.2. Peculiarities of surgical procedures.

All the patients underwent combined radical R0 (26 patients, or 79% cases) or tumour-reductive R1-2 (7 patients, or 21% cases) procedures. Supposing the possibility of manipulations on major thoracic veins, we always placed central venous lines into both SVC system (through subclavian or jugular vein) central venous pressure monitoring, and inferior vena cava system (through femoral and external iliac vein) to maintain intravenous infusion at any moment.

If less than a half of circumference of a vein was invaded, we limited venous resection to a wedge or tangential one with simple suturing of the defect; this procedure

was performed for 16 vessels. After more extended resections, vessels were either ligated (7 cases) or replaced with "Vitaflon ™"[1] PTFE grafts (26 vessels).

The subclavian vein was ligated without reconstruction in both cases of invasion. The indication for ligation of the IV was extended distal thrombosis; in other cases – in absence of thrombosis or after successful thrombectomy – we tried to reconstruct both IVs, taking into consideration the possibility of postsurgical thrombosis of one of the grafts. SVC was ligated without reconstruction in only one case of extended thrombosis in the systems of both IVs, in other cases it was reconstructed

A bypass between the right IV and the right auricle in course of a tumour-reductive procedure was performed in 1 patient with invasive mediastinal lymphoma complicated by SVCS (the diagnosis of lymphoma was stated only at surgery by frozen section studies).

Prior to cross-clamping of the SVC, moderate hypervolaemia was created by fluid infusion. Vasopressors were used only in case of significant arterial hypotension. Immediately before reconstruction of SVC or IVs, heparinization was used in a dose of 100 units per kg of body weight.

Different methods of reconstruction were used to restore blood outflow in the SVC system:
- SVC reconstruction by a linear PTFE graft anastomosed with the confluence of IVs and right auricle or the central stump of the SVC;
- Reconstruction of both IVs with a single linear PTFE graft, anastomosing it in "end to end" mode with one of the IVs, and in "end to side" mode with another  (see Fig. 1);
- SVC reconstruction by a linear PTFE graft anastomosed with one of the IVs and right auricle or the central stump of the SVC, ligating the other IV (see Fig. 2);
- Separate reconstruction of the right IV with a linear PTFE graft anastomosed to the right auricle, and of the left IV – with an autovenous graft;
- Reconstruction of IVs with two separate linear PTFE grafts anastomosing one of them with the right auricle and another – with the first graft ("end-to-side");
- Reconstruction of IVs with two separate linear PTFE grafts anastomosing one of them with the right auricle and another – with central stump of the SVC (Fig. 3);
- PTFE graft reconstruction of one of the IVs when another one and SVC are intact (Fig. 4).

If blood pressure in the SVC system after SVC cross-clamping exceeded 400 mm of water column, we performed a temporary bypass between the right IV and the right auricle prior to mobilization of the tumour; no neurological complications related to intraoperational venous hypertension in the SVC system were observed.

In the early postoperative period, prophylaxis of thrombosis was continued by direct anticoagulants till the 7th day postoperatively, after that it was switched to disaggregants and Trental. After discharge, patients were recommended to continue Trental; no indirect anticoagulants were used.

---

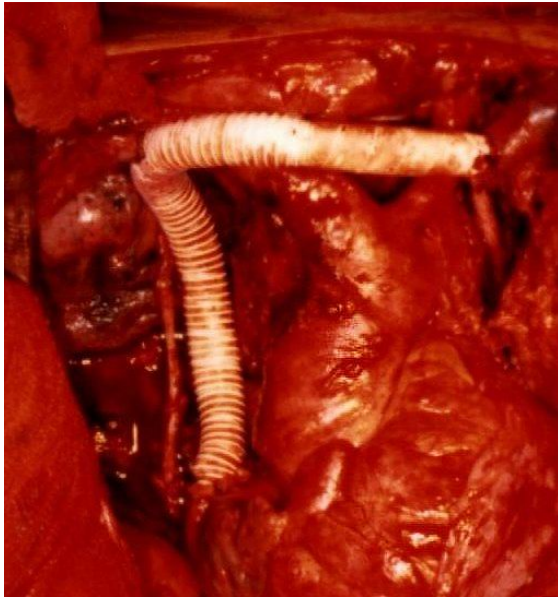[1] Production of "Ecoflon" enterprise, St.Petersburg, Russia.

*Fig. 1. SVC and both IVs resected; left IV anastomosed "end to end" with the PTFE graft, right IV anastomosed "end to side"; the graft anastomosed with central stump of the SVC.*
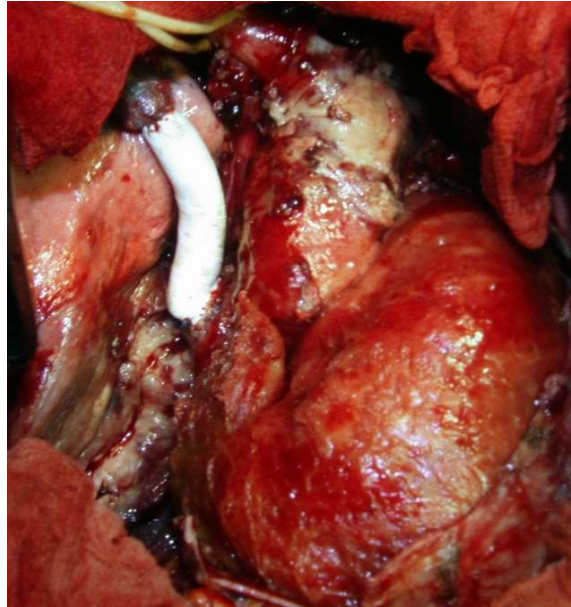


*Fig. 2. SVC and both IVs resected; PTFE graft anastomosed with the stump of right IV and right auricle; left IV ligated.*
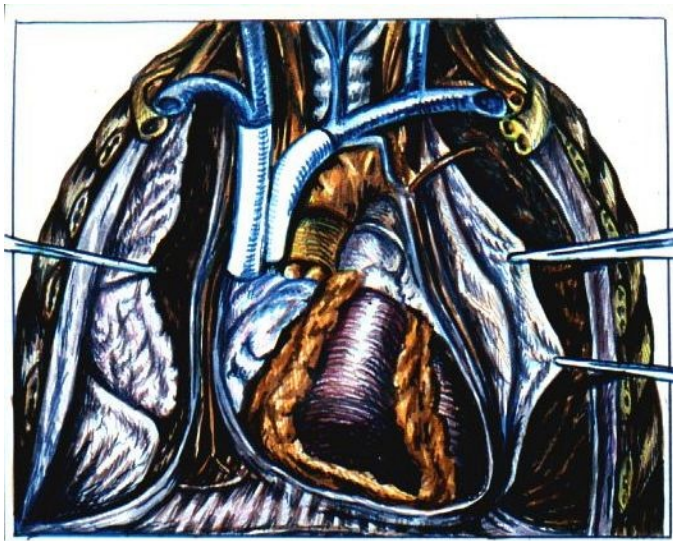


*Fig. 3. Scheme of separate reconstruction of both IVs with linear PTFE grafts anastomosed with the right auricle and SVC stump.*
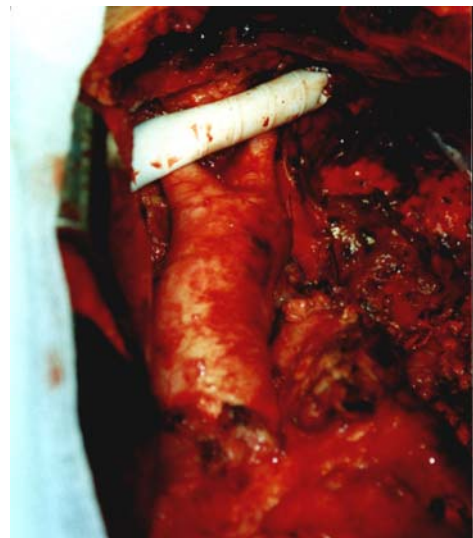


*Fig. 4. PTFE graft of the left IV anastomosed "end-to-side" with the SVC.*

## 6.3. Results.

Despite the variety of techniques used for venous reconstruction, there was no tendency to PTFE graft thrombosis in any of them. One patient who underwent removal of an invasive thymic carcinoma with SVC resection and reconstruction developed distal thrombosis in the system of right IV which was successfully managed with conservative therapy; after his death of generalized disease (vertebral metastasis) 5 months later, autopsy revealed the patent PTFE graft without signs of thrombosis. In other patients, no venous thrombosis was observed immediately after surgery, neither in long-term follow-up.

Speaking of modern indications for SVC resection, we must mention that all 3 of our patients who underwent bilateral phrenic nerve resection due to malignant invasion succeeded to survive. One of them required artificial ventilation for 12 days postsurgically to manage respiratory failure which was finally cured. Other two patients recovered effective spontaneous ventilation at the day of surgery or next day. We believe that bilateral phrenic nerve resection is possible in case of their invasion because respiratory system in these conditions is already prepared do denervation of the diaphragm and able to compensate the loss of phrenic nerve function.

After discharge, all the patients were sent to specialized oncological institutions for adjuvant or curative chemo- and radiotherapy; yet, no one of them was given this therapy for reasons we are unaware of.

Life quality evaluated by Karnofsky's scale made up a median of 35 before surgery and 88 two months postsurgically.

It has to be admitted that we failed to obtain long-term results in the majority of our patients for a significantly prolonged period, due to their migration to other regions of the country as well as to foreign countries. This fact, along with morphological variability of the cases, does not permit us to analyse long-term results of surgery. We have only incomplete data about 12 of our patients: 9 of them survived at least 1 year, 2 – 2 years, one patient survived more than 4 years.

## 7. Conclusion.

Main causes of malignant SVCS are lung cancer and malignant mediastinal neoplasms located mostly in upper anterior and middle mediastinum. Appearance of SVCS in a patient with thoracic malignancy proves local advance of the disease and catastrophic lack of time for diagnosis and treatment. Life duration and life quality in these patients undergoing symptomatic treatment or chemo- and radiotherapy are significantly lower than after successful surgery.

Modern angiosurgical technologies being applied to surgical oncology permit surgeons to extend operability by radical plan (R0) for patients with malignant SVCS syndrome up to 80% and more. Principal advantages of surgery for these cases are: the most accurate diagnosis and staging with morphological verification; immediate elimination of tumour-related complications - fatal and/or decreasing life quality; increase of survival time related to surgical radicalism.

Analysis of literature demonstrates that combined surgery for patients with locally advanced lung cancer may be justified by good long-term results even in presence of mediastinal lymph node metastases including contralateral ones.

Life duration and life quality are comparatively lower after tumour-reductive surgery than after radical one but higher than after palliative bypass procedures, endovascular stenting, and chemo/radiotherapy. Thus, even non-radical surgery may be more preferable for these patients than conservative approach.

In diagnostically doubtful cases of SVCS we advocate open surgery for the following reasons:
- Only open surgical procedure with incisional biopsy of several organs and structures leaves almost no chance for a diagnostic error;
- Only after thorough open inspection may a surgeon judge upon operability and choose radical, tumour-reductive, or palliative plan;
- Even in case of a locally-advanced tumour sensitive to chemo- and radiotherapy, such as lymphoma, a decompressive tumour-reductive procedure with restoration of venous flow may immediately improve the patient's condition by removal of mediastinal compression and intoxication;

- Complete radical removal of a malignant mass with resection and reconstruction of the SVC presents some patients an opportunity of significantly prolonged survival time, increasing life quality level more than twice in all the patients.

Differentiated approach is necessary for patients who are likely to demand bilateral phrenic nerve resection for surgical radicalism, which does not cause severe respiratory failure in some cases. Based on our experience, we believe the danger of bilateral phrenic nerve section to be overestimated at some extent. It seems that involvement of phrenic nerves into a malignant infiltrate affects their function much earlier than time for surgery comes, and this period is mostly enough for a patient to compensate respiratory muscle insufficiency by accessory respiratory muscles. Otherwise, this respiratory muscle failure may be corrected within 2 weeks postsurgically using modern ventilators. We suppose that such a patient needs profound preoperative evaluation of respiratory-muscular functions, in particular - phrenic nerve and diaphragmatic function. At least some patients with bilateral phrenic nerve invasion may be operated by radical plan.

The optimal prosthesis for reconstruction of SVC system seems to be enforced PTFE graft. Its undoubted advantages before autovenous grafts are fast and simple preparation for use, resistibility to extrinsic compression; at the same time, its atrombogenic properties are comparable with those of autovenous graft. As for different techniques of reconstruction of SVC system, our experience did not prove any of them to be significant for risk of thrombosis. To prevent the latter, therapy with direct anticoagulants during the first week after surgery and disaggregation later on seem to be enough.

In conclusion, the authors would like to express their conviction of necessity to use all the potential of modern surgical and anaesthesiological technologies to extend operability of advanced malignant neoplasms invading great vessels, because effectiveness of all existing alternative methods remains lower than effectiveness of surgery in the aspects of long-term results.

Reference List

1. Taylor GA, Miller HA, Standen JR, Harrison AW. Bypassing the obstructed superior vena cava with a subcutaneous long saphenous vein graft. J Thorac Cardiovasc Surg 1974; 68(2):237-40.

2. Turina M. [Surgical diseases of the superior vena cava]. Helv Chir Acta 1988; 54(5):611-6.

3. Magnan PE, Thomas P, Giudicelli R, Fuentes P, Branchereau A. Surgical reconstruction of the superior vena cava. Cardiovasc Surg 1994; 2(5):598-604.

4. Puglionisi A, Picciocchi A, D'Ugo DM, Bruni V, Lemmo GM. Venous involvement by follicular carcinoma of the thyroid gland. Ital J Surg Sci 1986; 16(2):133-7.

5. Bordigoni L, Blin D, Magnan PE, Giudicelli R, Fuentes P. [Ectatic tumoral thrombosis of the superior vena cava revealing thyroid cancer]. Ann Chir 1992; 46(2):194-8.

6. Tarao M, Nitta T, Hayashi M, Ichihashi M, Goto A. [A case of thyroid cancer invading into mediastinum that was in need of resection of both IVs for complete cure]. Kyobu Geka 1997; 50(7):531-4.

7. Levett JM, Meffert WG, Strong WW *et al*. Leiomyosarcoma of the superior vena cava and azygos vein. Ann Thorac Surg 1995; 60(5):1415-7.

8. Spaggiari L, Regnard JF, Nottin R *et al*. Leiomyosarcoma of the superior vena cava. Ann Thorac Surg 1996; 62(1):274-6.

9. Tovar-Martin E, Tovar-Pardo AE, Marini M, Pimentel Y, Rois JM. Intraluminal leiomyosarcoma of the superior vena cava: a cause of superior vena cava syndrome. J Cardiovasc Surg (Torino) 1997; 38(1):33-5.

10. Nomori H, Nara S, Morinaga S, Soejima K. Primary malignant lymphoma of superior vena cava. Ann Thorac Surg 1998; 66(4):1423-4.

11. Shishido M, Nagao N, Miyamoto K. [Mediastinal amelanotic melanoma presenting as superior vena cava syndrome]. Nihon Kyobu Shikkan Gakkai Zasshi 1997; 35(2):240-4.

12. Blanco P, Ly S, Beylot Barry M *et al*. Surgical treatment of an endovascular metastatic melanoma of the superior vena cava. Dermatology 1999; 199(2):156-7.

13. Benyass A, Boulahya A, Aithoussa M *et al*. [Angiosarcoma of the right atrium. Presentation of a surgically treated case and comparison with data of the literature]. Ann Cardiol Angeiol (Paris) 1997; 46(10):674-8.

14. Brachet A, Thevenet F, Gilly FN *et al*. [Inflammatory pseudotumor of the superior vena cava: rare etiology of mediastinal tumor]. Ann Chir 1993; 47(2):170-3.

15. Gomez Huelgas R, Martin Villasclaras JJ, Ruiz Morales MJ, Guijarro Merino R, Benitez Domenech A, Campos J. [A superior vena cava syndrome secondary to a pleural fibrous mesothelioma]. Arch Bronconeumol 1994; 30(5):269-71.

16. Narayan D, Brown L, Thayer JO. Surgical management of superior vena caval syndrome in sarcoidosis. Ann Thorac Surg 1998; 66(3):946-8.

17. Trabut JB, Duong Van Huyen JP, Artru B, Bruneval P. [Intravascular lipoma of the superior vena cava]. Ann Pathol 1999; 19(6):529-31.

18. Sato S, Hirano J, Itsubo K, Mashiko K, Nakano M, Kurosawa H. [Intravascular invasion of a thymoma from the thymic vein to the right atrium--a case report]. Nippon Kyobu Geka Gakkai Zasshi 1996; 44(7):998-1002.

19. Minato N, Rikitake K, Ohnishi H, Takarabe K, Ishida H. Invasive thymoma with intracaval growth

extending and directly invading the right atrium. J Cardiovasc Surg (Torino) 1999; 40(6):915-7.

20. Thomas P, Magnan PE, Moulin G, Giudicelli R, Fuentes P. Extended operation for lung cancer invading the superior vena cava. Eur J Cardiothorac Surg 1994; 8(4):177-82.

21. Le Roux BT. The presentation of bronchial carcinoma. Scott Med J 1968; 13(2):31-7.

22. Stanford W, Doty DB. The role of venography and surgery in the management of patients with superior vena cava obstruction. Ann Thorac Surg 1986; 41(2):158-63.

23. Robinson I, Jackson J. New approach to superior vena caval obstruction. BMJ 1994; 308(6945):1697-9.

24. Dartevelle PG, Chapelier AR, Pastorino U *et al*. Long-term follow-up after prosthetic replacement of the superior vena cava combined with resection of mediastinal-pulmonary malignant tumors. J Thorac Cardiovasc Surg 1991; 102(2):259-65.

25. Remy J, Herbeau D, Sautiere M, Ribet M, Voisin C. [Thymic tumors and superior vena cava syndrome]. Ann Radiol (Paris) 1971; 14(11):895-904.

26. Gomes MN, Hufnagel CA. Superior vena cava obstruction: a review of the literature and report of 2 cases due to benign intrathoracic tumors. Ann Thorac Surg 1975; 20(3):344-59.

27. Bergeron P, Reggi M, Jausseran JM *et al*. [Our experience with surgery of the superior vena cava]. Ann Chir 1985; 39(7):485-91.

28. Chen JC, Bongard F, Klein SR. A contemporary perspective on superior vena cava syndrome. Am J Surg 1990; 160(2):207-11.

29. Dartevelle PG. Herbert Sloan Lecture. Extended operations for the treatment of lung cancer. Ann Thorac Surg 1997; 63(1):12-9.

30. Perez CA, Presant CA, Van Amburg AL 3rd. Management of superior vena cava syndrome. Semin Oncol 1978; 5(2):123-34.

31. Hoak BA, Chapman J, Tiley E, Boland JP. Quick and easy management of superior vena caval syndrome. Am Surg 1986; 52(11):622-3.

32. Parish JM, Marschke RF Jr, Dines DE, Lee RE. Etiologic considerations in superior vena cava syndrome. Mayo Clin Proc 1981; 56(7):407-13.

33. Anders H, Keller C. Pemberton's maneuver - a clinical test for latent superior vena cava syndrome caused by a substernal mass. Eur J Med Res 1997; 2(11):488-90.

34. Hasegawa T, Mizumo A, Matsumoto H, Fukushima K, Fuse K. Use of copolymer graft developed to serve in venous prostheses. Surgery 1973; 74(5):696-702.

35. Leshnower AC, Shumacker HB Jr, Shariatzadeh AN. Experimental studies with synthetic venous bypass grafts, including a new experimental model. Am J Surg 1974; 128(3):392-7.

36. Fujiwara Y, Cohn LH, Adams D, Collins JJ Jr. Use of Gortex grafts for replacement of the superior and inferior venae cavae. J Thorac Cardiovasc Surg 1974; 67(5):774-9.

37. Heydorn WH, Zajtchuk R, Miller J, Schuchmann GF. Gore-Tex grafts for replacement of the superior vena cava. Ann Thorac Surg 1977; 23(6):539-44.

38. Heydorn WH, Geasling JW, Moores WY, Lollini LO, Gomez AC. Changes in the manufacture of expanded microporous polytetrafluoroethylene: effects on patency and histological behavior when used to replace the superior vena cava. Ann Thorac Surg 1979; 27(2):173-7.

39. Husfeldt KJ, Gall FP, Schulz HP, Zirngibl H. [Vein substitution using PTFE prosthesis--results of

animal experiments and initial clinical experiences]. Chir Forum Exp Klin Forsch 1979; 11-5.

40. Fiore AC, Brown JW, Cromartie RS *et al*. Prosthetic replacement for the thoracic vena cava: an experimental study. J Thorac Cardiovasc Surg 1982; 84(4):560-8.

41. Masuda H, Ogata T, Kikuchi K, Tanaka S. Longevity of expanded polytetrafluoroethylene grafts for superior vena cava. Ann Thorac Surg 1989; 48(3):376-80.

42. Miller DB. Palliative surgery for benign superior vena caval syndrome. Am J Surg 1975; 129(4):361-4.

43. Effeney DJ, Windsor HM, Shanahan MX. Superior vena cava obstruction: resection and bypass for malignant lesions. Aust N Z J Surg 1973; 42(3):231-7.

44. Vishnevskii AA, Adamian AA, Khariton AS, Mazhorov VA. [Jugular-femoral shunting in the vena cava superior syndrome]. Grudn Khir 1978; (5):57-62.

45. Pokrovskii AV, Klioner LI, Kazakov EN, Ansatarov EA. [Surgery in the superior vena cava syndrome using semibiological protheses]. Grudn Khir 1970; 12(5):3-8.

46. Graham A, Anikin V, Curry R, McGuigan J. Subcutaneous jugulofemoral bypass: a simple surgical option for palliation of superior vena cava obstruction. J Cardiovasc Surg (Torino) 1995; 36(6):615-7.

47. Lome LG, Bush IM. Resection of the vena cava for renal cell carcinoma: an experimental study. J Urol 1972; 107(5):717-9.

48. Bacha EA, Chapelier AR, Macchiarini P, Fadel E, Dartevelle PG. Surgery for invasive primary mediastinal tumors. Ann Thorac Surg 1998; 66(1):234-9.

49. Anderson RP, Li WI. Segmental replacement of superior vena cava with spiral vein graft. Ann Thorac Surg 1983; 36(1):85-8.

50. Doty DB, Baker WH. Bypass of superior vena cava with spiral vein graft. Ann Thorac Surg 1976; 22(5):490-3.

51. Doty DB. Bypass of superior vena cava: Six years' experience with spiral vein graft for obstruction of superior vena cava due to benign and malignant disease. J Thorac Cardiovasc Surg 1982; 83(3):326-38.

52. Doty DB, Doty JR, Jones KW. Bypass of superior vena cava. Fifteen years' experience with spiral vein graft for obstruction of superior vena cava caused by benign disease. J Thorac Cardiovasc Surg 1990; 99(5):889-95; discussion 895-6.

53. Lee AW, Reed CE, Kratz JM. Superior vena cava bypass: use of the azygos vein. Ann Thorac Surg 1988; 46(6):686-7.

54. Zembala M, Kustrzycki A, Ostapczuk S *et al*. Pericardial tube for obstruction of superior vena cava by malignant teratoma. J Thorac Cardiovasc Surg 1986; 91(3):469-71.

55. Lemmer JH Jr, Behrendt DM, Beekman RH, Hernandez RJ. Pedicled right atrial-pericardial tissue conduit for bypass of the obstructed superior vena cava in children. J Thorac Cardiovasc Surg 1989; 98(3):417-20.

56. Shimizu N, Date H, Moriyama S, Ando A, Teramoto S. Reconstruction of the superior vena cava in patients with mediastinal malignancies. Eur J Cardiothorac Surg 1991; 5(11):575-8.

57. Dartevelle P, Levasseur P, Rojas A, Pouliquen E, Le Brigand H, Merlier M. [Replacement of the superior vena cava with PTFE prostheses. Apropos of 12 cases]. Chirurgie 1982; 108(8):671-7.

58. Dartevelle P, Chapelier A, Navajas M *et al*. Replacement of the superior vena cava with polytetrafluoroethylene grafts combined with resection of mediastinal-pulmonary malignant tumors. Report of thirteen cases. J Thorac Cardiovasc Surg 1987; 94(3):361-6.

59. Spaggiari L, Regnard JF, Magdeleinat P, Jauffret B, Puyo P, Levasseur P. Extended resections for bronchogenic carcinoma invading the superior vena cava system. Ann Thorac Surg 2000; 69(1):233-6.

60. Charnsangavej C, Carrasco CH, Wallace S *et al*. Stenosis of the vena cava: preliminary assessment of treatment with expandable metallic stents. Radiology 1986; 161(2):295-8.

61. Rosch J, Bedell JE, Putnam J, Antonovic R, Uchida B. Gianturco expandable wire stents in the treatment of superior vena cava syndrome recurring after maximum-tolerance radiation. Cancer 1987; 60(6):1243-6.

62. Oudkerk M, Heystraten FM, Stoter G. Stenting in malignant vena caval obstruction. Cancer 1993; 71(1):142-6.

63. Jeanfaivre T, Pegis JD, Enon B, Tuchais E. [Surgical vein reconstruction in patients with tumor invasiveness of the superior caval system. Retrospective study of 7 cases]. J Chir (Paris) 1996; 133(2):61-4.

64. Takahashi T, Akamine S, Morinaga M, Oka T, Tagawa Y, Ayabe H. Extended resection for lung cancer invading mediastinal organs. Jpn J Thorac Cardiovasc Surg 1999; 47(8):383-7.

65. Nakahara K, Ohno K, Mastumura A *et al*. Extended operation for lung cancer invading the aortic arch and superior vena cava. J Thorac Cardiovasc Surg 1989; 97(3):428-33.

66. Larsson S, Lepore V. Technical options in reconstruction of large mediastinal veins. Surgery 1992; 111(3):311-7.

67. Scherck JP, Kerstein MD, Stansel HC Jr. The current status of vena caval replacement. Surgery 1974; 76(2):209-33.

68. Mikhailov SS, Pismenov IA. Effect of prosthetic design on the results of vena cava alloplasty. Experimental and morphologic investigation. Am J Surg 1969; 117(3):369-74.

69. Avasthi RB, Moghissi K. Malignant obstruction of the superior vena cava and its palliation: report of four cases. J Thorac Cardiovasc Surg 1977; 74(2):244-8.

70. Moore WM Jr, Hollier LH, Pickett TK. Superior vena cava and central venous reconstruction. Surgery 1991; 110(1):35-41.

71. Gloviczki P, Pairolero PC, Toomey BJ *et al*. Reconstruction of large veins for nonmalignant venous occlusive disease. J Vasc Surg 1992; 16(5):750-61.

72. Yoshimura H, Kazama S, Asari H, Itoh H, Tominaga S, Ishihara A. Lung cancer involving the superior vena cava: pneumonectomy with concomitant partial resection of superior vena cava. J Thorac Cardiovasc Surg 1979; 77(1):83-6.

73. Beattie EJ Jr, Martini N, Choudhry KU, Wright W, Howland WS. Emergency reconstruction of the superior vena cava. Surg Clin North Am 1970; 50(5):959-67.

74. Watanabe S, Oda M, Tomita Y *et al*. [Extended resection of the great vessels for primary lung cancer and mediastinal tumor]. Kyobu Geka 1999; 52(1):4-7.

75. Tanaka H, Miyoshi S, Okumura A *et al*. [Reconstruction of great vessel for patients with advanced lung cancer or malignant mediastinal tumor]. Kyobu Geka 1999; 52(1):19-24.

76. Fukushima K, Ohara T, Yamaguchi T *et al*. [Long-term follow-up results of stage III and IV thymomas and thymic carcinomas invading the great veins]. Nippon Geka Gakkai Zasshi 1993; 94(11):1201-8.

77. Yagi K, Hirata T, Fukuse T *et al*. Surgical treatment for invasive thymoma, especially when the superior vena cava is invaded. Ann Thorac Surg 1996; 61(2):521-4.

78. Okada M, Sugimoto T, Yamamoto H. [Surgical strategy for invasive pulmonary and mediastinal

tumors requiring superior vena cava reconstruction]. Kyobu Geka 1999; 52(1):14-8.

79. Tsuchiya R, Asamura H, Kondo H, Goya T, Naruke T. Extended resection of the left atrium, great vessels, or both for lung cancer. Ann Thorac Surg 1994; 57(4):960-5.