

# Loops, Kinks and Anomalies of Vertebral Arteries

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## Introduction

The diagnostic procedures, indications for surgery and operative techniques for carotid artery occlusive diseases are well established, but still there is a lot of controversy regarding surgery of vertebral arteries (VAs), especially kinked VAs. Even more controversy exists about indications for surgical repair of different anomalies of VAs. The role of VAs in supplying blood to the brain and the value of surgical repair of stenotic lesions of the VAs are still underestimated by neurologists and most vascular surgeons.

For a long time reconstructive surgery of the VAs was performed occasionally by a few vascular surgeons. In the 1980s and 1990s experience with VA surgery developed in several vascular surgery centers. In recent years most vascular surgery centers have begun to operate on VAs. Neurologists and angiologists have also begun to appreciate the relationship between stenotic lesions of the VAs and symptoms of ischemia in vertebrobasilar territory, as well as effectiveness of surgical correction of the VAs in these cases. With improved surgical techniques, VA surgery is no longer considered too difficult or dangerous. Those vascular surgeons, who have gained their personal experience with considerable number of operations on kinked VAs and know the late results of these operations, have no doubts about the need for correction of kinked symptomatic VAs. Our extensive experience in vertebral artery surgery, exceeding 2251 operations, among them - 1059 performed for VA kinking, convinced us of the effectiveness of these operations. Moreover, such a big amount of performed operations for VA kinking demonstrates: 1) the widespread existence of this pathology between population; 2) good results achieved by surgical repair, otherwise, the neurologists would not send the patients for surgical treatment.

## Historical hints

Cate and Scott were pioneers in VA surgery<sup>1</sup>. In 1953 they performed the first direct operation on the VA: transsubclavian endarterectomy. In 1958 Crawford and De Bakey<sup>2</sup> were the first to publish their experience in VA surgery. The results of anatomic studies on cadavers made by Hutchinson and Yates in 1956<sup>3</sup> provided great impetus for the development of VA surgery. They found that approximately 70% of patients who had died due to vertebrobasilar stroke, had atherosclerotic stenotic or occlusive lesions in their VAs, most of which were at their origins at the subclavian arteries. Hemodynamic and angiographic studies showed, that such lesions cause a decreased blood flow in the vertebrobasilar territory of the brain and therefore, the neurological symptoms of vertebrobasilar insufficiency (VBI), described by Denny-Brown in 1953<sup>4</sup>. Contorni in 1960<sup>5</sup> described the subclavian steal syndrome, as including symptoms of VBI, associated with a reversed flow in the VA, supplying the blood to the arm, where the proximal part of the subclavian artery is occluded. He proposed a surgical repair in these cases to restore normal blood flow in the subclavian artery, which in turn, reverses to normal the direction of blood flow in the VA. Powers et al in 1961, 1963<sup>6,7</sup> described the lateral and posterior branching of the VA from the subclavian artery and successfully corrected this pathology by excision of the scalenus anterior muscle, ligation and transection of the thyrocervical trunk, and

desympatization of the VA. Hardin and Poser in 1963<sup>8</sup> were the first to describe the extravasal compression of the first portion of the VA by the fascial bands. They proved the hemodynamic significance of this pathology by angiography: the first portion of the VA was stenosed or even occluded while rotating the head to the opposite side. At operation they found the fibrous bands crossing and compressing the first portion of the VA. Their surgical procedure included scalenotomy, transection of fibrous bands, crossing the VA, transection of the thyrocervical trunk, and fixation of the subclavian artery, using the thyrocervical stump, to the scalenus anterior muscle stump on the first rib. They used the same procedure for the straightening of the kinked VAs.

The anatomic studies of subclavian artery and its branches made by Daseler and Anson in 1959<sup>9</sup> showed that anomalies of vertebral arteries are often encountered. In 2,5% of autopsies they have found the left vertebral artery originating from the aortic arch. The right vertebral artery originates from the aortic arch in 0,1% of angiographically studied patients (Pauliukas et al, 1993<sup>10</sup>). Normally the VAs enter the canalis transversarius at the C6 transverse process. The VA enters the canalis transversarius at the C5 transverse process in 6,6% of population<sup>9</sup>. The incidence of this anomaly is the same for both left and right VAs<sup>9</sup>. The VA enters canalis transversarius at the C4 level in 0,5% of the population<sup>9</sup>. Anatomic studies by Stopford in 1916<sup>11</sup> revealed an incidence of 15% of one hypoplastic VA. The hypoplasia of the right VA is more common (ratio 2:1) in comparison to the left VA<sup>10</sup>. Hypoplasia of both VAs is encountered in 0,3% of angiographically studied patients<sup>10</sup>. Aplasia of one VA is encountered with the same frequency, in 0,3% of angiographically studied patients (Berk, 1961)<sup>12</sup>. We have described a case of a 37 year old male, having VBI, with congenital aplasia of both vertebral arteries and having compensating anomaly: his basilar artery was a continuation of the left occipital artery<sup>10</sup>.

Husni in 1966<sup>13, 14</sup> described the entrapment of the VA between the scalenus anterior and longus colli muscles just below its entrance into canalis transversarius at the C6 transverse process which they were able to demonstrate angiographically, with the head rotated to the opposite side. The procedure performed by them was scalenectomy and partial excision of the longus colli muscle. VBI symptoms cleared in 19 patients from 20 operated. An angiography was repeated one week and one year after operation. No compression of the operated VA was observed, while turning the head on either side. Kojima et al in 1985<sup>15</sup> also operated the patient suffering vertigo episodes, while turning the head to the right. Angiography in this position showed the right VA compressed at the entrance into the bony canal. At operation they have found right VA compressed between longus colli and scalenus anterior muscles. Scalenectomy and partial excision of longus colli muscle cured the patient. The same observations and angiographic documentation of the entrapment of the VA between longus colli and scalenus anterior muscles independently were made by myself from 1983 in many cases.

I was very happy later, after finding Husni's papers on this subject, because it is very difficult to convince others to believe in this pathology. My experience with this pathology is published in 1993, 1996 years<sup>10, 16</sup>. Hurvitz in 1976<sup>17</sup> described the extraluminal compressions of the VAs in cases of VBI and successfully cured these patients by surgical decompression and desympatization of the VAs. He found that sympathetic trunk or its branches were often the causes of VA compression. Intermittent occlusion of VA caused by external fibrous band was described by Mapstone and Spetzler in 1982<sup>18</sup>. Extravasal strangulation of VA by sympathetic trunk and its branches is widely described in my papers, as well<sup>10,16</sup>. The hemodynamic and clinical importance of VA kinks was appreciated and

surgically managed by Rainer et al, 1970<sup>19</sup> ; Cormier and Laurian, 1976<sup>20</sup> ; Barkauskas, 1977<sup>21</sup> ; Imparato et al, 1967, 1981<sup>22,23</sup> ; Neugebauer and Stimmell, 1983<sup>24</sup> ; Thevenet and Ruotolo, 1984<sup>25</sup> ; Agrifoglio, 1984<sup>26</sup> ; Nishijima et al, 1989<sup>27,28</sup> ; Koskas et al, 1994<sup>29</sup>.

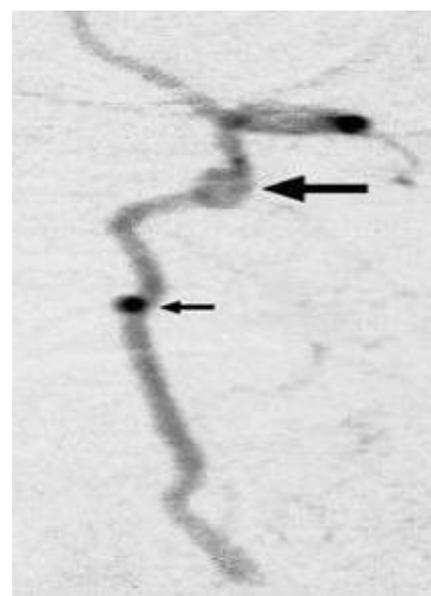
During the last two decades there was a dramatic increase in the number of papers on tactics and techniques in vertebral artery surgery: Cormier, 1980<sup>30</sup> ; Diaz et al, 1982<sup>31</sup> ; Berguer, 1983, 1992, 1993, 1996<sup>32-35</sup> ; Diaz et al, 1984<sup>36</sup> ; Imparato, 1985<sup>37</sup> ; Benedetti-Valentini et al, 1985<sup>38</sup> ; Senter et al, 1985<sup>39</sup> ; Magometschnigg et al, 1990<sup>40</sup> ; Branchereau and Magnan, 1990<sup>41</sup> ; Deriu et al, 1991<sup>42</sup> ; Giangola et al, 1991<sup>43</sup> ; Habozit, 1991<sup>44</sup> ; Ogawa et al, 1991<sup>45</sup> ; Deruty et al, 1991<sup>46</sup> ; Comerota and Maurer, 1992<sup>47</sup> ; Archie, 1992<sup>48</sup> ; Van Schill et al, 1992<sup>49</sup> ; Miyamoto et al, 1992<sup>50</sup> ; Pauliukas et al, 1993, 1996<sup>10,16</sup> ; Kieffer et al, 1996<sup>51</sup>.

## Etiology

Most of the loops of VAs are congenital, because they are encountered in the childhood, as well. The same definition should be applied to the coils, although some of them could be acquired due to elongation of the first segment of the VA. Elongation of the artery is a result of arterial hypertension and loss of elasticity by the arterial wall in the aged. Relative elongation of VA results from the shortening of the neck due to osteochondrosis in the senile population. Kinks are mostly acquired, due to elongation of the artery, although some of them are congenital: kinked VAs due to extravasal compression by crossing sympathetic trunk or its branches, by muscle tendons in cases of high VA entrance into canalis transversarius, by sympathetic nerves or fascial bands in some cases of VA branching from the aortic arch. All stated above is applied to the first segment of the vertebral artery. In the second VA segment kinks and loops are rare. Nevertheless, when the VA enters canalis transversarius high in the neck, it can have the loop at the level of the 4-th or higher vertebra (Fig. 1).



**Figure 1**



**Figure 2**

**Fig.1.** Left VA enters canalis transversarius at C-4 transverse process. VA has a loop below the entrance into it (arrow).

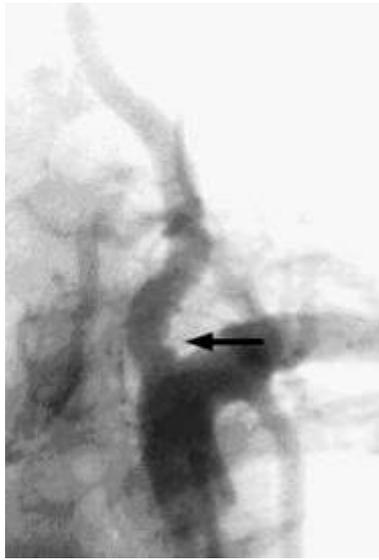
**Fig.2.** Left VA has a loop between the transverse processes of the 3-rd and 2-nd vertebrae (small arrow) and the second loop between the transverse processes of the 2-nd and 1-st vertebrae (large arrow).

Kinks of the VA in the V2 segment are mostly due to compression of the VA by osteophyte, or because of deformation of the vertebral column in cervical osteochondrosis. Loops are encountered in cases of VA herniation from the canalis transversarius. In the third segment of the VA, rarely encountered kinks are due to elongation of the artery in the aged, or due to strangulation of it by the ventral branch of the second cervical nerve. Loops in this segment are very rare and we believe they are congenital. Figure 2 illustrates rarely encountered loops in the second and third segments of the VA in the same patient. Kinks in the fourth VA segment and even of the basilar artery are also seldom encountered and can be the cause of VBI. Congenital redundancy together with the aging elongation of these arteries are the causes of this intracranial pathology.

## **Pathophysiology**

At the site of the kink the lumen of VA is stenosed due to protrusion of the arterial wall duplicature into the lumen. The degree of stenosis can differ from minimal to complete vessel occlusion and depends generally on the angle of the kink: the smaller the angle, the greater the vessel stenosis. The degree of lumen narrowing and its hemodynamic importance is best assessed by doppler flow studies. All hydraulic physics are in force, in the blood flow through the blood vessels. That's, why we can appreciate the degree of vessel stenosis from the linear blood flow velocity. The greater the vessel stenosis, the higher the linear blood flow velocity at the site of stenosis in comparison with normal vessel lumen. Angiography is a gold standard in appreciating atherosclerotic vessel stenosis, however it can be misleading in cases of kinking. Extravasal compression or strangulation of the artery also causes stenosis due to deformation and narrowing of its lumen. Some of VA kinks are due to fixation and strangulation of the VA by the sympathetic trunk or its branches, so it sometimes difficult to separate kinks from extravasal compressions or strangulations of the VAs. Figure 3 and case 1 illustrates this statement.

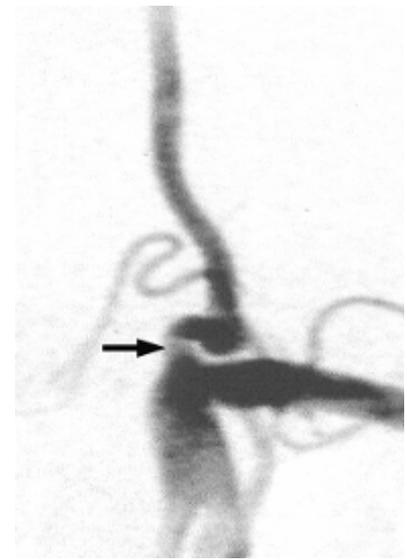
*Case 1. Patient have had long lasting VBI with severe vertigo episodes, which became incapacitating during the last 8 years. On angiograms, performed in another institution vertebral arteries were considered to be normal. After 4 years he was admitted to our hospital with intensive vertigo episode. Dopler and duplex blood flow studies showed a hemodynamically significant kink of the left VA. Patient had his old angiograms. On the left VA angiogram (Fig.3 A) a small notch was seen, creating suspicion that the VA is strangulated with sympathetic nerve. Angiography was repeated in our institution. Fig. 3 B, C shows angiographic findings in the same artery after 4 years. Total loop was formed around the sympathetic nerve by the left VA. At operation findings confirmed preoperative diagnosis: left VA was strangulated by sympathetic trunk with severe VA kinking (Fig.3 D). VA was pulled out from behind the sympathetic nerves, resected and reimplemented in front of nerves back into enlarged its orifice (Fig. 3 E). After operation all symptoms of VBI cleared and the patient is symptom free for 3 years.*



**Figure 3 A**



**Figure 3 B**

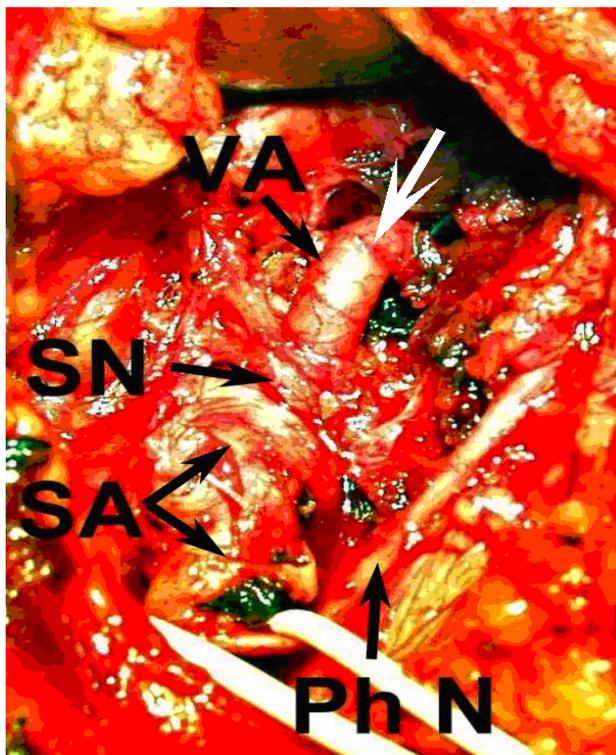


**Figure 3 C**

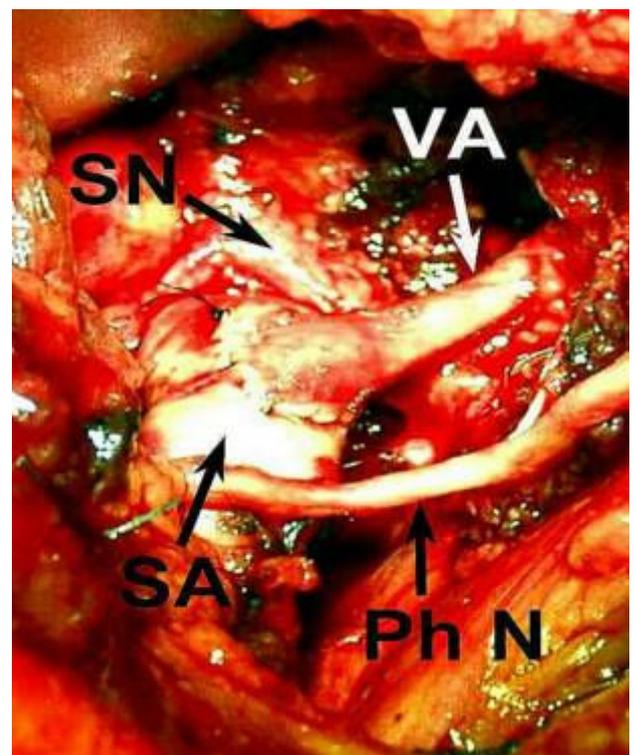
**Fig. 3 A.** Left VA angiogram. A small notch is seen at the site of VA strangulation by the sympathetic nerve (arrow).

**Fig. 3 B.** Left anterior VA angiogram 4 years later. Kinked VA at the site of strangulation by the sympathetic nerve (arrow).

**Fig. 3 C.** Oblique view of the left VA 4 years later. Arrow points to the site of strangulation by the sympathetic nerve.



**Figure 3 D**



**Figure 3 E**

**Fig. 3 D.** Findings at the operation: origin of the VA was found crossed and strangulated by thick sympathetic nerve. White arrow points to the kink of vertebral artery.

**Fig. 3 E.** Vertebral artery was pulled out from behind the sympathetic nerve, resected and reimplemented into its enlarged orifice in front of sympathetic nerve. Note the conus shaped origin of the vertebral artery.

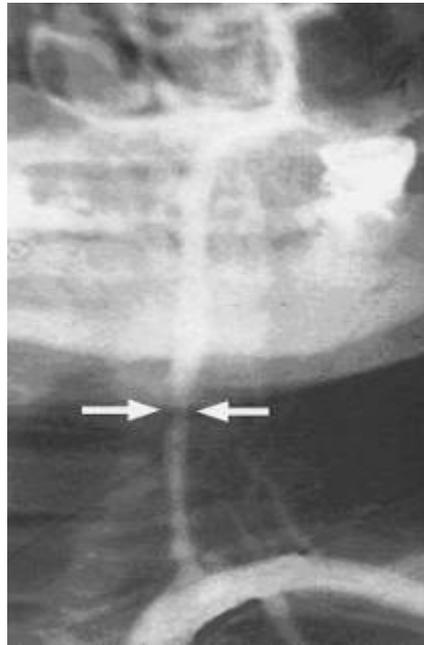
SA – subclavian artery, VA – vertebral artery, SN – sympathetic nerve, PhN – phrenic nerve.

That's, why author believes, that all kinds of VA stenoses, except of atherosclerotic ones, must be discussed in this chapter. VA kinks at the ostium, in cases of its posterior branching from the subclavian artery. In case of lateral branching of the VA, the VA originates from the subclavian artery more laterally, than normally, and in this case usually it originates in the same sagittal plane as thyrocervical trunk. VA orifice is situated in this case dorsally to the thyrocervical trunk and has at the same time posterior branching, as well, and kinking at the ostium (Fig. 4). Scalenus anterior muscle and the branches of thyrocervical trunk fixes firmly the position of subclavian artery and dorsal position of the orifice of the VA. In some cases VA originates even more laterally, lateral to the thyrocervical trunk. In these cases VA orifice is also rotated dorsally by scalenus anterior muscle, because VA is situated under this muscle. Scaleneotomy with ligation of thyrocervical trunk is curable in these cases, since the vertebral artery comes into normal position after this procedure. Only in few cases light rotation of the subclavian artery and fixation of it by the stump of the thyrocervical trunk to the stump of the scalenus anterior muscle on the first rib is desirable. In cases of extravasal compression and strangulation of VAs, especially with sympathetic trunk or its branches, an arterial spasm plays a major role in narrowing the arterial lumen and causing VBI. In these cases, at operation VA is typically found very spasmophylic and falls into spasm during mobilisation. Freeing the VA from compression and irritation and moistening it by papaverine solution, ceases the spasm. Sometimes additional desympatization of the VA is needed to bring the artery into normal status. Patients, having extravasal compressions or strangulations of the VAs can be asymptomatic for long years, provided the VA is not spasmophylic. They become symptomatic, when the VA becomes spasmophylic. The attack of VBI lasts as long, as spasm of VA lasts. The antispasmodic drugs, like papaverin, are most helpful in these cases. Case 2 illustrates this statement.

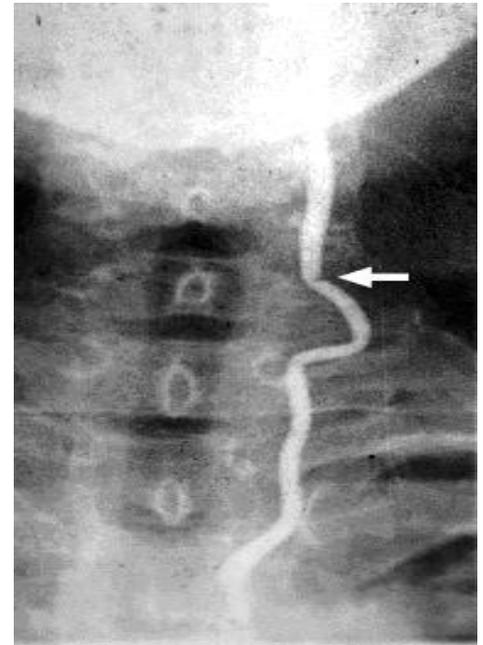
**Case 2.** *A 34 year old female presented with symptoms of chronic VBI since childhood. During the last 5 years, frequent episodes of vertigo had occurred, lasting for hours and even days. She also had tinnitus in the left ear. The CW-doppler study revealed high pitched signal (high linear flow velocity, what means high grade VA stenosis) at the entrance of the left VA into the canalis transversarius at 6-th transverse process, confirmed by angiography (Fig.5). On angiograms the right VA appeared normal. The circle of Willis was totally disconnected posteriorly. The compression of the VA between longus colli and scalenus anterior muscle tendons was diagnosed and patient was operated. Preoperative diagnosis was confirmed at the operation. The left VA was found entrapped between tendons of above mentioned muscles. It entered canalis transversarius normally, at the 6-th transverse process, however the tendons of scalenus anterior and longus colli muscles conjugated more caudad than normally. The extracanal part of the VA was highly spasmophylic and fell into deep spasm during it mobilisation, became less than 2 mm in diameter. Scaleneotomy, partial excision of lateral border of longus colli muscle, desympatization of the VA and moistening of it with 2% papaverine solution, was carried out. VA relaxed from the spasm and became absolutely normal, 4 mm in diameter, with normal blood flow velocity. No anastomosis was required in this case. Patient is symptom-free for 15 years postoperatively.*



**Figure 4**



**Figure 5**



**Figure 6**

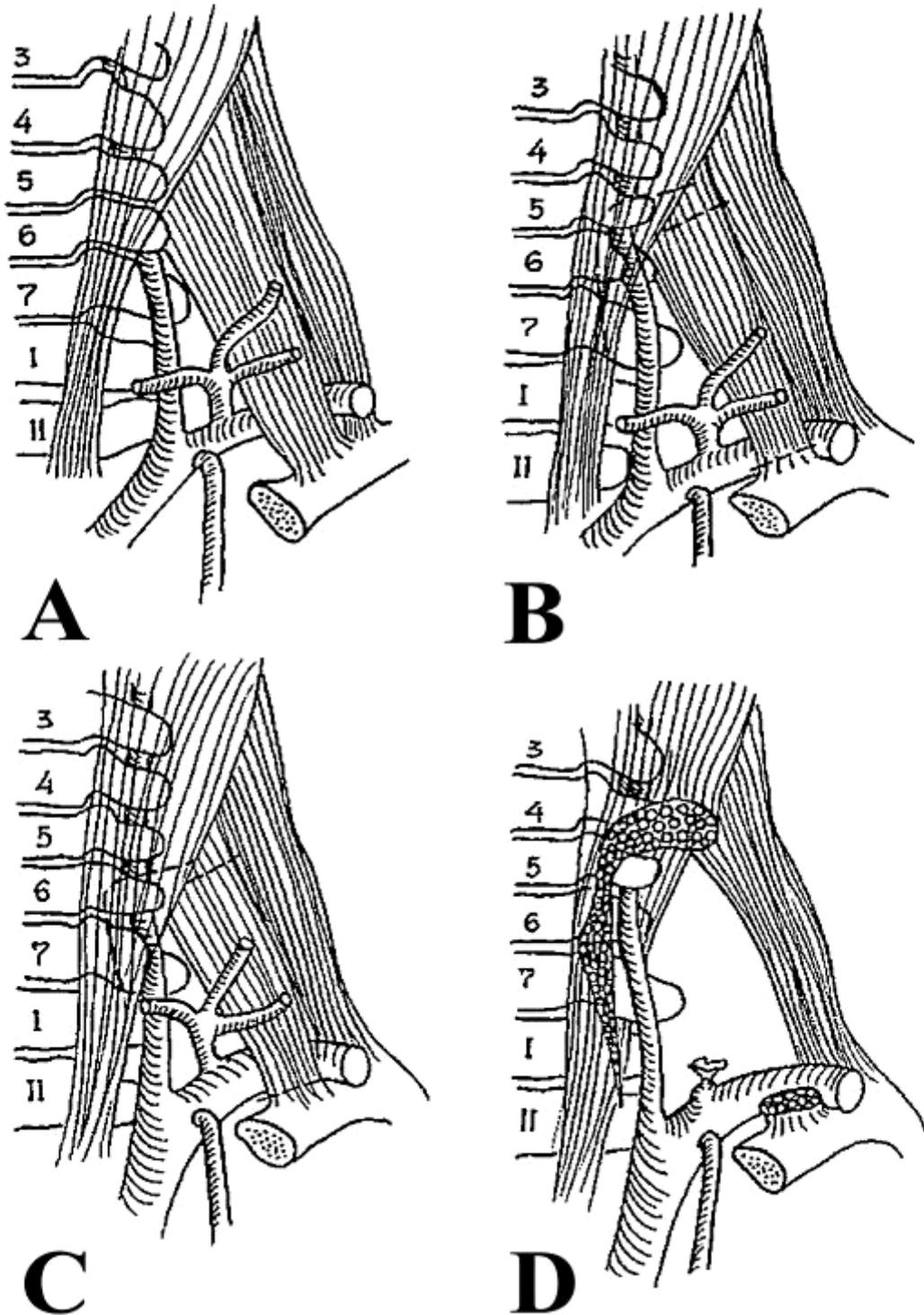
**Fig. 4.** Lateral branching of the right VA. VA originates at the same sagittal plane as thyreocervical trunk. Its orifice is located on the posterior (dorsal) surface of the subclavian artery (arrows). VA kinks at the orifice.

**Fig .5.** Left VA is compressed between scalenus anterior and longus colli muscle tendons (arrows). Note the spasmophylic narrowed extracanal part of the VA and normal lumen of the VA in canalis transversarius.

**Fig. 6.** Left VA originates from the aortic arch and enters canalis transversarius at the 5-th cervical vertebra. Longus colli muscle compresses the VA against the C-6 transverse process (arrow).

When the VA enters canalis transversarius at the 5-th transverse process or higher, it is always entrapped at the entrance into transverse process between longus colli muscle tendon and underlying transverse process (Fig. 6). Fig. 6 illustrates the compression of the VA in case of its high entrance into the canalis transversarius. This pathology can be asymptomatic in patients with normal circle of Willis and even in patients with posteriorly disconnected circle of Willis, provided the VA is not spasmophylic. The momentary compressions of the VA during head movements usually are asymptomatic. When VA becomes spasmophylic, the clinical picture changes dramatically and symptoms of VBI appear. VA originating from aortic arch is more prone to extravasal strangulation by sympathetic trunk, because it has a long course in the mediastinum and often intercrosses with sympathetic trunk or its fibres. On the other hand, VA, originating from the aortic arch, often enters canalis transversarius higher than normally and has problems with entrapment between longus colli muscle tendon and transverse processes of cervical vertebrae (Fig.8).

In Fig. 7 is presented drawing, illustrating the compression of the VA in both cases (high entry of VA into canalis transversarius and abnormal course of longus colli and scalenus muscles) and the typical surgical procedure performed in such cases.



**Figure 7**

**A** – normal relationships between scalenus anterior, longus colli muscles and vertebral artery.

**B** – VA enters canalis transversarius at C-5 transverse process and is compressed by longus colli muscle tendon against C-6 transverse process.

**C** – longus colli and scalenus anterior muscles conjugate lower than normally and entrap the VA in cases of normal entering of it into the C-6 transverse process.

**D** – typical surgical procedure, performed in these cases: total excision of scalenus anterior muscle and partial excision of longus colli muscle.



**Figure 8**

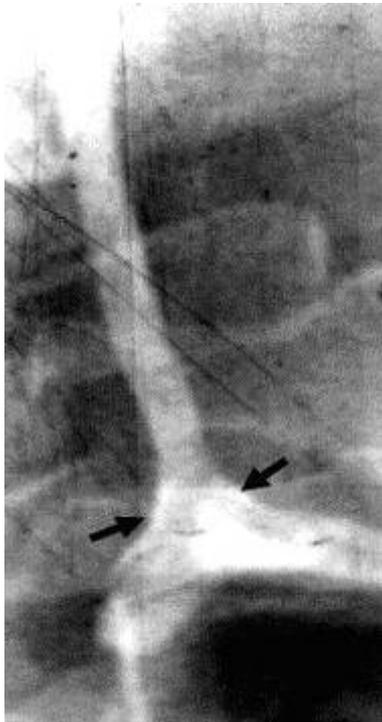
**Fig. 8.** Left VA originates from the aortic arch, is strangulated by the sympathetic trunk ( black arrows ) , enters canalis transversarius at C-5 transverse process and is compressed at the entrance into canalis transversarius by longus colli muscle tendon ( white arrow ).

The right VA of this patient also originated from the aortic arch.

In cases of loops and coils of the VA, the most important mechanism of blood flow impairment is also the kink of the artery, which usually appears at some point of loop or coil. When there is no kink in the loop or coil, the blood flow diminishes only by 5% due to kinetic energy losses in the loop, provided the loop has regular circle shape (Stehbens, 1969)<sup>52</sup>. In reality the loops and coils seldom have regular circle shape. In most cases they are elliptical or irregular, that's why angulations and kinks of different degree appear at the sites of the loops and coils. Moreover, the shape of loops and coils usually is variable. Therefore, the degree of stenosis at the site of loop or coil varies from time to time. This explains the typical clinical manifestations of the loops, coils and kinks: symptoms of VBI may be absent or light for periods of time and then reappear with full expression. Contrary to the loops of internal carotid arteries, the degenerative changes in the arterial wall at the sites of the VA loop poles are seldom expressed (Pauliukas et al, 1989<sup>53</sup>; Matskevitchus and Pauliukas, 1990<sup>54</sup>). So, the main pathogenetic mechanism of brain ischemia and VBI in VA kinking, extravasal compression, loops and coils is hypoperfusion.

The role of the circle of Willis should be stressed in redistributing the blood to all brain territories. Normal circle of Willis can redistribute the blood and compensate the blood flow to the V/B region even in cases of occlusion of both vertebral arteries in their proximal parts. Such patients have mild chronic VBI symptoms, however they don't have frank neurological deficit. On the other hand, vertebral arteries through the circle of Willis can compensate occlusion of both internal carotid arteries. Most neurologists and vascular surgeons underestimate the role of VAs in supplying the blood to the brain, especially in terms of their capability to supply blood flow to the anterior circulation through the circle of Willis. The capability of the circle of Willis to redistribute the blood flow to all brain territories is illustrated in Fig. 9 and case 3.

**Case 3.** 52 year old man with asymptomatic occlusion of both internal carotid arteries developed hemispheric symptoms when the left dominant VA became tightly stenosed at the orifice, because the blood supply in such cases is the lowest in farthest, the anterior circulation. Reconstruction of the orifice of the left VA, using internal shunt in the VA (Fig. 9 A) made patient symptom free. Postoperative angiography showed the blood supplied to all 6 main cerebral arteries from operated left VA (Fig. 9 B). Patient remains symptoms free for 7 years.



**Figure 9 A**



**Figure 9 B**

**Fig. 9 A.** Left VA, reimplanted into the subclavian artery due to tight atherosclerotic ostial stenosis. Note the conus shaped VA orifice ( arrows ). Right vertebral and both internal carotid arteries are occluded.

**Fig. 9 B.** Reconstructed left VA supplies the blood to all 6 brain arteries: both middle, both anterior and both posterior cerebral arteries .

Improved carotid territory circulation by vertebral artery reconstructions was also documented by Archie, 1992<sup>48</sup>. Patients with kinks, coils, loops or extravasal compressions of VAs, having symptoms of VBI, as a rule, have abnormal circle of Willis, otherwise they would compensate the shortage of blood supply in V/B region through the circle of Willis. Half of the population has abnormal circle of Willis ( Alpers et al, 1959<sup>55</sup>; Riggs and Rupp, 1963<sup>56</sup>; Fisher, 1965<sup>57</sup> ). The most often encountered abnormality is absence of one or both posterior communicating arteries of the circle of Willis. People with normal circle of Willis, having kinks of VAs, usually are asymptomatic and they don't come to neurologists and don't reach the vascular surgeons. Every open minded vascular surgeon, having enough personal experience in cerebrovascular revascularisation, who analyzes angiograms and compares them with the clinical symptoms is familiar with this fact. Kinked vertebral arteries are often seen on angiograms, performed on patients, having atherosclerotic lesions of carotid arteries, who are free from VBI symptoms. These, accidentally found kinked VAs in asymptomatic patients, mislead some vascular surgeons and neurologists in appreciating the hemodynamic and clinical importance of VA kinking.

Another point is, that vertebral arteries are two, and they conjugate to form the basilar artery. If both VAs has normal diameter, the kink or extravasal strangulation of one VA can be compensated by contralateral VA. However, in cases with totally disconnected posteriorly circle of Willis, most of such patients will be symptomatic. When one of VA is hypoplastic, the kink or extravasal compression of another VA can be asymptomatic if the circle of Willis is normal, or highly symptomatic in patients with one or both posterior communicating arteries absent.

## **Clinical manifestations**

Hypoperfusion can cause symptoms from the entire V/B region. Since the most sensible to ischemia are vestibular nuclei ( Nagashima C, et al, 1970<sup>58</sup> ) the first, most common, symptoms are dizziness, disturbances of equilibrium, nausea, nystagmus. Nystagmus, especially micronystagmus, visible through the Frenzel,s glasses is one of the earliest symptoms of VBI <sup>58</sup>. In more serious cases vertigo episodes appear and are very characteristic for VA kinking. Vertigo is not common and characteristic for atherosclerotic VA lesions. The next most characteristic symptom is visual disturbances due to hypoperfusion in the area of posterior cerebral arteries: occipital cerebral ( visual) cortex , which is the farthest point in V/B blood supply. Patients experience different visual defects: from localized haze up to homonymous hemianopsia or total cortical blindness in case of bilateral occipital brain lesion. Oculomotor nuclei are the next ones, most sensible to ischemia in the brain stem. Blurring of the vision, or frank diplopia occurs due to ishemia to these nuclei. Ataxia, a common symptom, is related to hypoperfusion of the cerebellum. Common symptoms are headache ( mostly occipital ) and hearing disturbances ( tinnitus and hypoacusicis ). Sensor and motor disturbances manifest in pronounced brain stem ischemia. As stated above, the characteristic feature of the kinks, coils, loops and extravasal compressions of the VAs is fluctuation of clinical expression of VBI symptoms. Symptoms may be absent, mild or intensive. The degree of expression of the symptoms depends upon the degree of the kink, or degree of the spasm, in cases of extravasal compression of VA. The patient can be asymptomatic for weeks, months or even years and then suddenly becomes symptomatic. Periods of aggravation of symptoms alternate with periods of alleviation. Usually patients have light symptoms of VBI even in the periods of alleviation. Contrary, patients with atherosclerotic lesions of VAs, as a rule, have stable VBI symptoms. Patients with congenitally kinked VAs have symptoms of VBI from the childhood. The most common being nausea, vomiting, especially while riding by car, boat. Such children can't endure swinging. They complain of headache, mostly occipital. Mental tiredness is characteristic. Vertigo episodes appear in more serious cases. Correction of VA pathology cures all symptoms in these children.

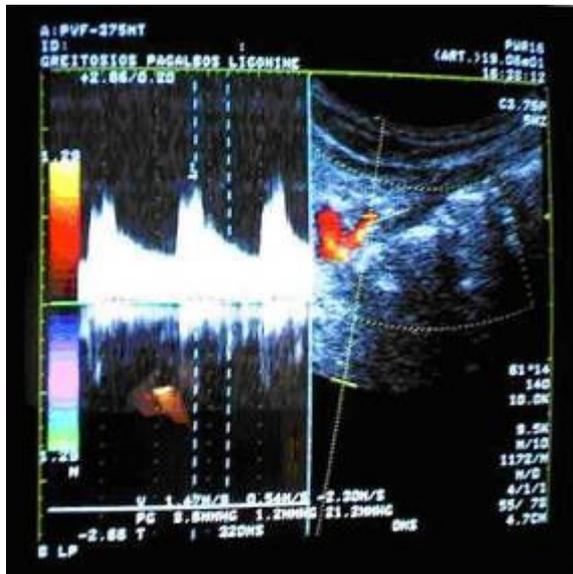
Table 1 presents the incidence of VBI symptoms encountered by us in 862 patients, operated for VA kinking.

**TABLE 1.** *Incidence of symptoms in 862 patients, operated for VA kinking*

Symptoms	Number of patients	Percentage
Dizziness	846	98,1
Visual disturbances	725	84,1
Ataxia	667	77,4
Headache ( mostly occipital )	621	72,0
Nausea / vomiting	482	55,9
Vertigo episodes	457	53,0
Hearing disturbances	425	49,3
Sensory disturbances	386	44,8
Memory deterioration	372	43,1
Episodic or continuous arterial hypertension	318	36,9
Vegetative and vasomotor paroxysms	299	34,7
Syncopal episodes	238	27,6
Drop attacks	173	20,1
Motor disturbances	153	17,7
Dysarthria	141	16,4
Temporal epilepsy	62	7,2
Transient global amnesia	32	3,7

## Diagnostic evaluation

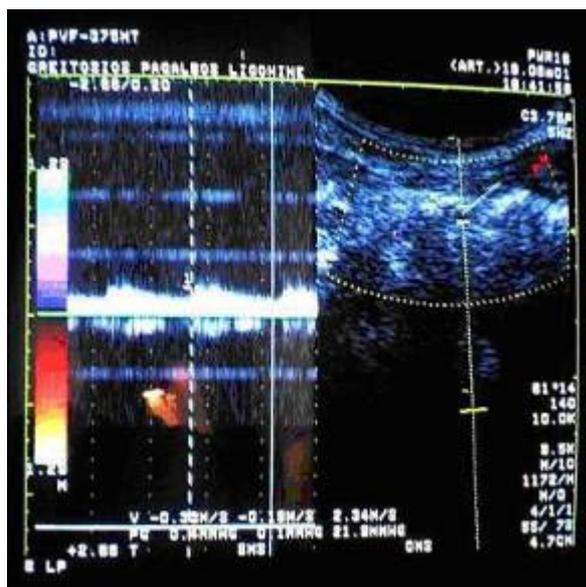
The diagnosis of VBI is a clinical challenge, because most of its manifestations are subjective and difficult to quantify. Otoneurological evaluation is helpful in differentiating VBI from other neurological diseases. If the diagnosis of VBI is suspected or established, doppler flow studies should be carried out. Simple, inexpensive continuous wave doppler is still very informative method in assessing the blood flow features and changes in the vessel. Large personal experience acquired and knowledge of fluid physics, enables examiner to diagnose arterial stenoses very precisely. I still use Parks Electronics doppler model 909 for this purpose. Continuous wave doppler is more sensitive to blood flow disturbances in the vessels when the blood flow velocity is assessed by acoustic signal. It is superior to duplex or even color doppler in cases of extravasal strangulations or compressions of VAs, because theoretically one hear every erythrocyte moving along the vessel. The signals from all erythrocytes merge in the ear into the sound of real blood flow in the vessel. Duplex scanners and color dopplers use spectral analysis of the doppler signals. The signals are packed into pieces of different frequency and displayed on the screen in grey scale or are color coded. So, a lot of information about blood flow is lost. Multiple samples should be obtained along the vessel with the duplex scanner in order to obtain more exact data about blood flow changes in the artery. Blood flow velocities and character of blood flow ( normal, poststenotic, prestenotic, stenotic ) must be obtained in the V2 segment of the VA in canalis transversarius as well. At the site of stenosis systolic and diastolic blood flow velocities increase (Fig. 10 A ).



**Figure 10 A**

**Fig. 10 A.** Doppler sample is placed at the site of the kink of the VA. Note high systolic and diastolic blood flow velocities and turbulence.

Distal to stenosis turbulence and poststenotic blood flow features appear ( Fig. 10 B ).



**Figure 10 B**

**Fig. 10 B.** Low poststenotic turbulent blood flow in canalis transversarius.

Continuous wave doppler enables examiner to hear specific blood flow features in lateral and posterior branching of the VA from subclavian artery and even the movements of arterial wall at the kink. However, spectral analysis of doppler signal is more objective and easier to analyse. Visualisation of artery itself, adds an information, what is very important in carotid artery disease, evaluating and appreciating the plaque structure. Irrespective of the method used to evaluate the blood flow in the artery, the blood flow velocities and shape of blood flow should be assessed along the artery by multiple samples. In our institution the accuracy of duplex scanning in evaluation of the VA pathology is over 80% ( depends on the experience of the examiner ). Normal linear blood flow values in the orifice of the VA ,

according to our data are following: systolic,  $83 \pm 14$  cm/sec; and diastolic,  $18 \pm 3$  cm/sec. The normal linear blood flow values in extracranial ( V1 ) and osseous ( V2 ) portions of the VAs are following: systolic,  $65 \pm 11$  cm/sec; and diastolic,  $21 \pm 4$  cm/sec. Hemodynamically important stenoses of the proximal part of the VAs are considered when the systolic blood flow velocity exceeds 120 cm/sec. Hemodynamic consequences of kink or extravasal compression of the VA are the same, as in atherosclerotic stenosis. Fig.10 illustrates the analogical changes in blood flow velocities in kinked, as in atherosclerotically stenosed VAs. In extravasal strangulations of the VA systolic blood flow velocity exceeding 100 cm/sec can be of diagnostic importance, because arterial spasm plays a major role, not the stenosis, in these cases. Compression of common carotid artery during doppler flow studies enhances the blood flow in the VAs, if that carotid artery is connected with V/B region by posterior communicating artery.

Transcranial doppler ( TCD ) studies add an information about the integrity of the circle of Willis. The presence of the posterior communicating arteries of the circle of Willis and the direction of blood flow in these arteries can be detected. The blood flow velocities in basilar and posterior cerebral arteries are obtained by this method as well. Poststenotic blood flow character and low values of blood flow velocities in the intracranial arteries indicate the more proximal stenosis of VAs. In some cases even reversed blood flow in basilar artery, confirmed by angiography, can be obtained during TCD examination. Monitoring of the blood flow in the middle cerebral artery is widely used by vascular surgeons in carotid artery surgery, while clamping the internal carotid artery. TCD is very useful in monitoring blood flow in the posterior cerebral arteries while clamping the only VA ( when contralateral VA is occluded, hypoplastic or aplastic ) and circle of Willis is posteriorly disconnected or both internal carotid arteries are occluded.

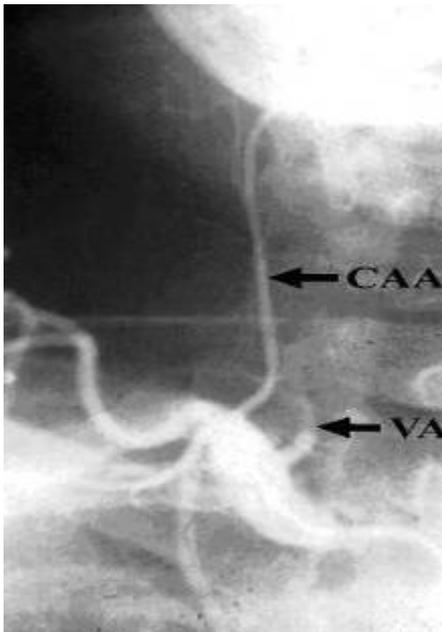
Doppler, duplex and TCD studies are noninvasive and allows examiner to repeat them as many times, as needed in different head and neck positions, if required.

Electroencephalography is of little value as diagnostic technique with regard to changes in brain blood flow, but it reflects the functional status of the brain and enables to detect infarction or even hypoperfusion areas of the brain, brain tumors. It is most valuable in diagnosing localized or generalized epileptic activity of the brain. Computed tomography ( CT ) and magnetic resonance imaging ( MRI ) are both very valuable in differentiating brain infarction from hemorrhagic stroke, brain tumors and other brain diseases. MRI is more advanced and sensitive method of imaging than CT and enables the detection of very small, sometimes clinically silent areas of brain infarction. VA kinking causes brain hypoperfusion, therefore brain infarction results only in long-lasting severe hypoperfusion episodes. In VA kinking VBI symptoms arise due to brain hypoperfusion, not to the infarcted area, that's why CT or MRI brain scans are typically normal in these clinical cases.

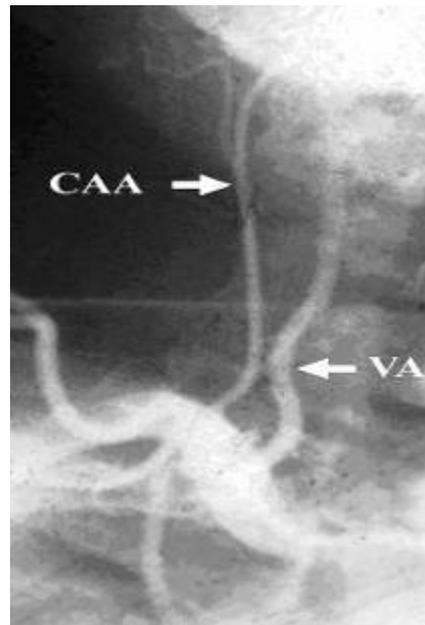
**Angiography ( ANG )** is still a gold standard in evaluating the lesions of the arteries, supplying the blood to the brain. Four vessel biplane digital subtraction ANG must be performed in all cases in order not to skip another pathology in arteries supplying the blood to the brain. CT angiography as an adjunctive method for visualisation of intracranial arteries is used by us in cases when the integrity of the circle of Willis must be assessed. Magnetic resonance angiography ( MRA ) can be also used for this purpose as well, as for visualisation of extracranial arteries. However, selective, contrast enhanced, digital subtraction ANG is superior to MRA, especially for visualisation of such fine pathology like extravasal compression or strangulation of the VA. Angiographic evaluation should

visualise the subclavian arteries, orifices of the VAs, VAs from the origin to their joining with the basilar artery, the basilar artery and all intracranial arteries of V/B region. The left VA can be absent on the left subclavian angiogram, because in 3% of population it originates from the aortic arch. Aortic arch angiogram must be performed in these cases. To obtain good visualisation of the entire left VA, its branches, basilar artery and its branches, selective ANG of the left VA should be performed afterwards. The same is applied to the right VA, when it originates from the aortic arch ( in 0,1% of population<sup>10</sup> ). When posterior originating of the VA from the subclavian artery is suspected by the doppler and duplex scanner , oblique and cranio-caudad projection of the VA should be added. In all angiograms secondary signs of slowed VA blood flow, such as delayed filling of VA with contrast in comparison with other branches of subclavian artery should be appreciated. Bosniak was the first<sup>59</sup>, who appreciated the importance of secondary angiographic signs in evaluating the brachiocephalic arteries. Normally, the blood flow in the VA is a lot faster than in other subclavian artery branches. The VA is already filled with contrast, when other subclavian artery branches begin to fill. Hypertrophied collaterals ( ascendens cervical artery ), shunting the blood to the distal VA, indicate the long lasting stenosis in the proximal VA.

Fig. 11 A illustrates the delayed filling of the right VA and hypertrophied ascendens cervical artery in case of hemodynamically significant posterior originating of the VA from subclavian artery. Otherwise, the right VA ( Fig.11 B ) has normal appearance and pathology would be skipped.



**Figure 11 A**



**Figure 11 B**

**Fig. 11 A.** Cinematographic angiography of the VAs. Posterior originating of the VA from subclavian artery with hemodynamically important kink at the VA ostium. Only experienced eye can see the posterior (dorsal) position of the VA orifice. Note very slow blood flow in the VA. Muscular branches of subclavian artery are already filled with contrast, meanwhile VA only begins to fill with contrast. Cervical ascendens artery serves as collateral for kinked VA.

**Fig. 11 B.** VA looks normal in this late angiogram, except posterior origin from the subclavian artery, which can be easily skipped viewing angiogram. Only hypertrophied cervical ascendens artery ( Bosniak's collateral ) shows slowed insufficient blood flow in the VA.

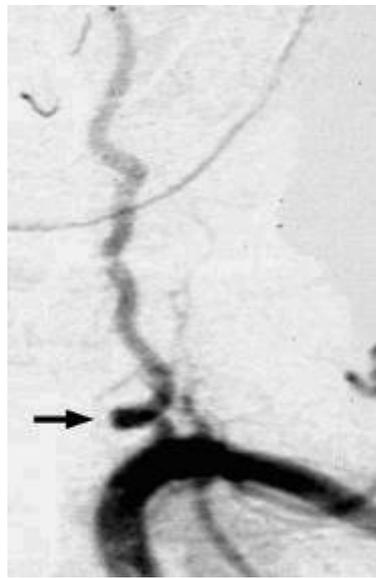
VA – vertebral artery, CAA – cervical ascendens artery.

The proximal extracanal ( V1 ) part of the VA is the most common site of kinks, loops, coils and extravasal compressions. Atherosclerosis affects the VA orifice and its proximal part most often, as well. When the doppler flow studies indicate the stenotic lesion of the VA and ordinary ANG does not show the lesion, dynamic ANG, using cinematographic angiography in special positions of the head and neck, in which the doppler registers the stenosis should be obtained. The pathology of the VA is best assessed by this way, because the kink or extravasal compression is best delineated by the flowing blood. We have had the patients in whom despite the doppler flow studies, indicating the stenotic lesion of the VA, the first time performed ANG was normal. Due to expressed VBI symptoms patients returned to our institution. Doppler flow studies repeatedly indicated kinked VA. The second time ANG revealed kink or even loop of the VA. ( Fig. 3).

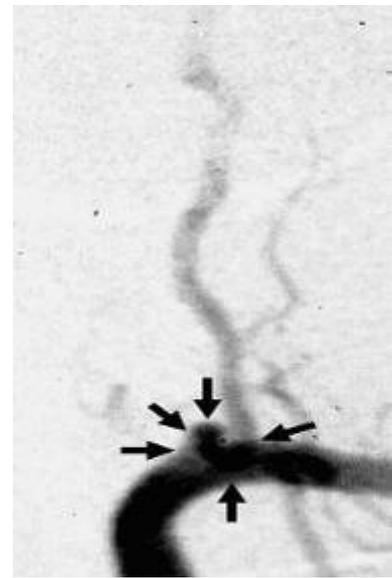
Fig. 12 illustrates angiograms of various types of VA kinks, which were symptomatic and surgical correction of them cured the patients.



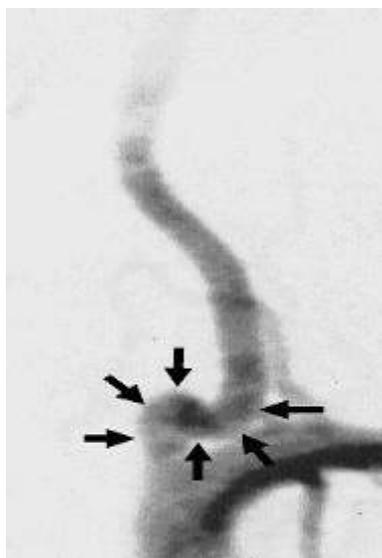
**Figure 12 A**



**Figure 12 B**



**Figure 12 C**



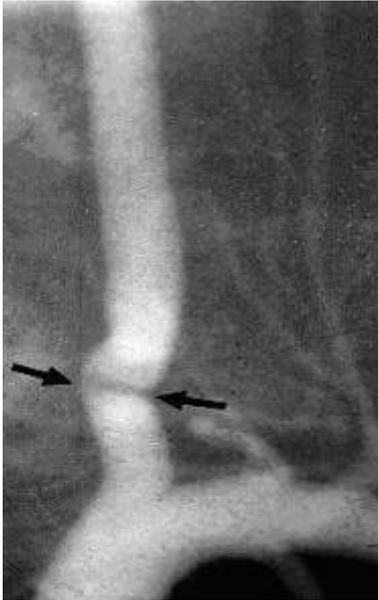
**Figure 12 D**



**Figure 12 E**

**Figures 12 A – 12 E. Different types of VA loops. Arrows point to the loop of VA.**

Fig. 13 illustrates the extravasal strangulation of the VA by the sympathetic trunk.



**Figure 13 A**



**Figure 13 B**

**Fig. 13.** Angiogram of the left VA. A – antero – posterior view, B – oblique view. Note tightly strangulated VA by the sympathetic trunk. Arrows point to the site of strangulation.

We have had the patients in whom at the first time taken angiograms of the VA, they appeared normal, despite the fact, that doppler and duplex studies indicated extravasal strangulation of the VA. Repeated ANG showed marked strangulation or compression of the VA like illustrated in Fig.14.



**Figure 14 A**



**Figure 14 B**

**Fig. 14 A.** Left VA on angiogram has normal appearance despite the doppler flow studies, indicating tight VA stenosis due to strangulation in its proximal part.

**Fig. 14 B.** Repeated angiogram of the same artery showed tight strangulation of the VA by sympathetic nerve (arrows).



**Figure 15 A**



**Figure 15 B**

**Fig. 15 A, 15 B.** Two other types of VA strangulation by the sympathetic nerve ( arrows point to the site of strangulation ).

Figure 15 illustrates other types of extravasal strangulation of the VAs. Operation cured these patients as well.

### **Indications for surgical treatment**

The indications for surgical repair of stenotic lesions of the VAs, in cases of hypoperfusion of V/B region, are still controversial. Berguer<sup>35</sup> proposes following indications: a 75% or greater stenosis of both VAs, if they are both of equivalent size. If one VA is hypoplastic, a 75% stenosis of the dominant VA is required. These anatomic requirements are applied also to the kinks and extrinsic compressions of the VAs during head rotation/extension, that cause symptoms during angiography. The same strict definition of indications for VA surgery are proposed by Deriu et al<sup>42</sup>, Giangola et al<sup>43</sup>, Imparato<sup>60</sup>.

Our definition of indications for VA surgery differs from these. We believe, that the key to deciding on surgical repair, lies in the posterior communicating arteries of the circle of Willis. In patients with normal circle of Willis, stenosis or even occlusion of one VA will be asymptomatic. They will survive even with extracranial occlusion of both VAs. The opposite, will be in patients with absent posterior communicating arteries. Even one stenosed VA, can be the cause of VBI, despite a normal contralateral VA. The same point of view is supported by Benedetti-Valentini et al<sup>38</sup>, Comerota et al<sup>47</sup>, Nagashima et al<sup>58</sup> and many others. Even one kinked VA can be the cause of VBI. Extrinsic compression of the VA can be the cause of VBI, if the contralateral VA is abnormal and one or both posterior communicating arteries are absent. In cases of extravasal compression of the VAs, spasm of the VA plays a major role (Fig.5). This can be revealed by duplex scans during the V/B TIA, related to the spasm. On duplex scan images, the artery will show a normal lumen, except at the site of extravasal compression, in an asymptomatic period. Extravasal compression of the VA will be seen during dynamic ANG, and in some cases, vasospasm of the compressed VA is also seen on angiograms ( Fig.5). In aged patients with multiple lesions of carotid and vertebral arteries, the decision, concerning which artery to repair,

depends on the presenting symptoms ( hemispheric or vertebrobasilar ) and presence or absence of posterior communicating arteries of the circle of Willis. If the patient has VBI symptoms and both posterior communicating arteries are absent on angiogram, the VA must be repaired. When posterior communicating arteries are functioning, repair of a tightly stenosed internal carotid artery clears the VBI symptoms. Hence, in patients with normal circle of Willis, hemodynamically significant stenoses of internal carotid arteries should be repaired first. On the other hand, when internal carotid arteries are occluded or stenosed intracranially, repair of the tightly stenosed VA clears hemispheric symptoms ( Fig. 9 ). The same data are presented by Archie <sup>48</sup>.

Extensive experience, knowledge of brain hemodynamics, appreciation of the role of the circle of Willis, and long-term follow up data enable the physician to make the right decision for each patient, including the cases with kinked or extrinsically compressed VAs.

Following recommendations can be given for the beginners in VA surgery. At first, the diagnosis of VBI must be established and all the other neurological diseases, capable to mimic VBI, must be ruled out. Then, the doppler and duplex studies must be appreciated. Angiograms, as the most reliable diagnostic data, must be evaluated. The decision as to operate the patient, is made by relying on the patients clinical symptoms, doppler blood flow studies, duplex or color doppler data and, of course, on angiographic data. All these data are compiled together and compared with each other. Experienced and open minded neurologist must work in the vascular surgery team, which operates VAs. Nevertheless, vascular surgeon, operating VAs, must have good knowledge of neurology, otherwise, he will not be able to consult the patients with VBI and to make decisions, as to operate the patient with kinked, or strangulated VA. Large personal experience of vascular surgeon and knowing of the long-term results of operated by him patients is needed to make the right decision in each case. The landmarks in making decision for operative treatment of the kinked VAs are: established diagnosis of VBI; doppler flow studies indicating hemodynamically significant VA stenotic lesion ( systolic blood flow exceeding 120 cm/sec at the site of the kink and poststenotic blood flow in the V2-V4 segments of the VA ); angiographically confirmed the doppler and duplex data and visualised kink. Once again, all other neurological diseases, which can mimic the VBI should be ruled out. Extravasal compressions and strangulations of the VAs are even more challenging and demanding than kinks in the decision making, as to operate or not. Operation is easy to perform in these cases, however the decision making is difficult. One must keep in mind, that only good late surgical results ensures the stream of patients from the neurologists with symptoms of VBI to vascular surgeon.

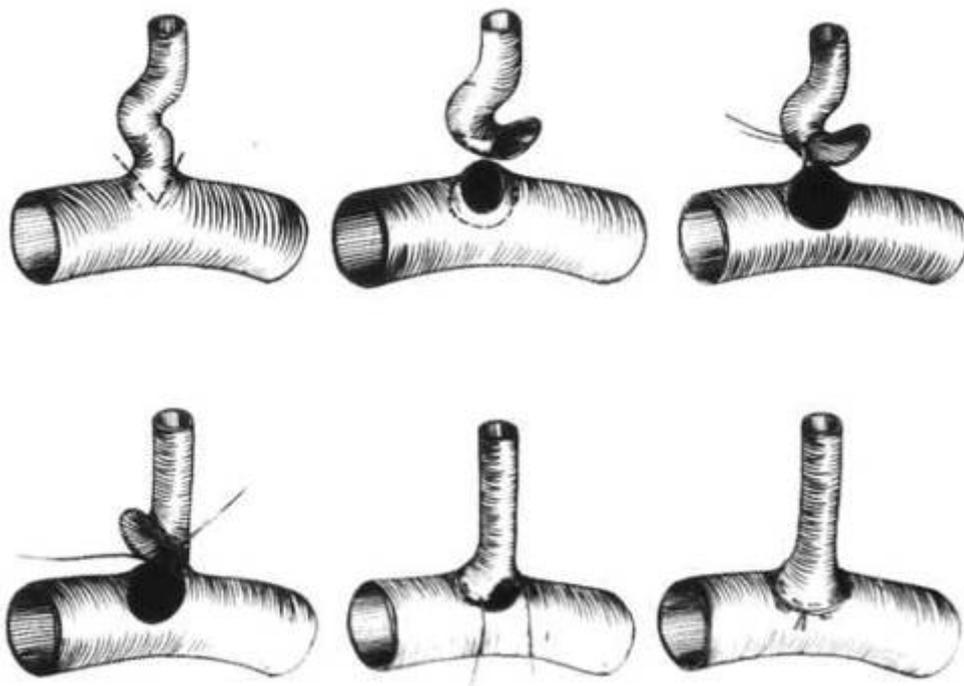
## **Surgical management**

General anaesthesia is preferred, although cervical plexus blockade can be used successfully. The patient is placed in a supine position with the neck slightly hyperextended; this is accomplished by placing a folded sheet under the shoulder on the side of operation with the head rotated to the opposite side. The rotation and hyperextension of the neck is accomplished after intubation. A transverse incision 10 cm long, ( 1 - 2 cm above the medial half of the clavicle along the skin crease ) is carried out. This incision is adequate for exposing the VA up to the 5-th transverse process. In cases of high entry of the VA into the C4 or C3 transverse processes, a vertical incision of reversed "T" configuration is added as high, as needed. The platysma is transected. The supraclavicular nerves in the lateral corner of the wound are preserved. Medial head of the sternocleidomastoid muscle should be left intact, if good postoperative cosmetical results are desired. The omohyoid muscle is freed

and pulled upward, or downward, or divided. The prescalene fat pad is divided along the scalenus anterior muscle and its medial and lateral flaps are retracted by traction sutures. The transverse colli artery must be anticipated, ligated and divided. The phrenic nerve is isolated, encircled with the tape and mobilised from its origin at C5 cervical nerve to below its crossing the subclavian artery. The medial and lateral edges of the anterior scalene muscle are mobilised, a right angle haemostat is gently passed behind the scalenus anterior muscle at the level of the subclavian artery. The muscle is divided with scalpel, as the right angle hemostat pulls the muscle away from the subclavian artery. The anterior scalene muscle is excised to the level of C6 vertebra. All the branches of the thyreocervical trunk are ligated and divided. The internal mammary artery in most cases can be preserved for future mammaro-coronary anastomosis. The vertebral vein is mobilised, ligating all its tributaries up to its entrance into the bony canal, then ligated and excised. This manoeuvre facilitates the mobilisation of the VA close to the entrance into the bony canal. The VA is inspected, encircled with the tape and mobilised to its entrance into the bony canal, irrespective how high VA enters it. The longus colli muscle tendon is clearly seen crossing the C6 transverse process. If the VA enters canalis transversarius higher than C6 transverse process, it is always compressed by this tendon against the transverse process of the C6 vertebra. If the course of the longus colli muscle is more lateral and lower than normally, its tendon conjugates with the medial edge of the scalenus anterior muscle somewhat lower and the VA is compressed between these two muscles even in cases of normal entry of the VA into canalis transversarius ( Fig.5, Fig.7 ). Our method is to always totally excise the scalenus anterior muscle and partially the longus colli muscle ( lateral part ), to the level of C6 transverse process, completely exposing the entrance of the VA into the bony canal in all operations for VA kinks and anomalies. If the VA enters the bony canal at C5 or higher level, it is mobilised until its entrance into the bony canal, excising all muscle tissue ( intercrossing longus colli, scalenus anterior, longus capitis fibres ) in front of the VA and freeing pathway for vertebral artery. If the space between the two adjacent transverse processes is narrow, the unroofing of the transverse process just above the entrance of the VA into canalis transversarius may be necessary to avoid compression of the VA between two adjacent transverse processes while turning the head. Fig.7 illustrates the surgical technique for correction of VA compression by longus colli muscle at C6 and higher level. If the proximal VA is normal, the surgical correction is complete. If there is extra length with kinking of the VA, resection of the proximal VA part with reimplantation back into its enlarged orifice is carried out. In cases of extravasal strangulation of VAs by sympathetic trunk or its branches, VA is divided at its orifice, pulled from behind the sympathetic nerves and reimplanted back into its enlarged orifice in front of sympathetic nerves ( Fig. 3 D, 3 E ). If the VA has no extra length, it is reimplanted into the position of thyreocervical trunk, because subclavian artery at this point is situated higher in the neck. If there is still VA too short for spatulation, all branches of the subclavian artery are ligated and divided, including mammary artery. This manoeuvre relaxes subclavian artery, it can be pulled up and reimplantation of the VA can be successfully accomplished almost in all cases, including atherosclerotic ones. In some cases of VA kinking in hypertensive patients subclavian artery is considerably elongated, so that VA orifice is at the C6 transverse process. In such cases, resection of subclavian artery distal to the VA is needed in order to bring it back into normal position, otherwise it is impossible to straighten the VA even if its all extracanal part is resected. To leave very short, 3-5 mm long segment of the VA is a bad decision, because in the future subclavian artery elongates, rises up, and such short segment of the VA kinks very sharply. Such short segment of the VA can not acquire "S" shape. When the cause of

extravasal compression are fascial bands or small branches of sympathetic trunk, these structures can be divided without any sequelae and division and reimplantation of the VA is not needed. My policy is always to discuss with the patient before the operation the possible Horner's syndrome. If the patient is worried by this phenomenon, I always reimplant the VA and don't disturb the sympathetics. Almost in all cases of VA extrinsic compressions the VA falls into spasm. Moistening with 2% papaverine solution relaxes the spasm of the VA and greatly enhances the blood flow through it. In very resistant cases of the spasm, desympatization of the VA ceases the spasm.

The important points of VA reimplantation are the following: the VA must have enough length for spatulation and widening of its orifice by incising its posterior wall; the hole made in the subclavian artery must be round or oval and its perimeter must match that of widened spatulated orifice of the VA; the posterior border of the hole must be at the same level as the posterior wall of the reimplanted VA, otherwise, there will be the kink at the VA ostium; reimplantation is started from the posterior wall, using a parachute technique. Our technique of reimplantation is depicted in Fig. 16.



**Figure 16**

Reimplantation of the VA into subclavian artery is preferred for following reasons: 1) it avoids clamping of two arteries (vertebral and common carotid ) supplying the blood to the brain; 2) after the reconstruction, the blood flow is physiological; 3) in cases of VA kinking often all brachiocephalic arteries are redundant, the common carotid artery is very mobile, and it is very difficult to appropriately adjust the length of the VA, when transposing it into common carotid artery. In hypertensive patients ,whose arteries tend to elongate, a common follow up finding, is a kink of the VA, transposed to the common carotid artery. We use transposition of the VA into common carotid artery exclusively in atherosclerotic cases, when subclavian artery is unsuitable for reimplantation: stenosed or heavily calcified. All patients in our series with anomalies and kinks of VAs tolerated VA clamping and we do not use the TCD monitoring for these patients. In overall our series of 2251 operated VAs only 5 patients did not tolerate the clamping of the VA. In 3 cases both internal carotid

arteries were occluded and the only one still patent and tightly stenosed VA was operated. In 2 other cases patients did not tolerate the clamping of the only VA despite the both functioning internal carotid arteries. The circle of Willis was completely disconnected posteriorly in these patients. We successfully have used Senters technique<sup>39</sup> of using an internal shunt for reconstruction of the VA at any level in these cases.

We don't use routine intraoperative angiography in VA surgery, because it is expensive and time consuming. We use intraoperative doppler flow studies instead, in the operated arteries by continuous wave doppler with pencil-type probe. Intraoperative dopplerometry enables surgeon to assess the blood flow velocity along the vessel, as well as the vessel diameter (the smaller the diameter, the greater the blood flow velocity and vice versa). So, the stenosed segments of the vessel (e.g. anastomosis) are recognised very easy and, if needed, are corrected. The superiority of the doppler against the ANG is, that the doppler allows examiner to assess the blood flow through the operated vessel. Anyway, one of control methods should be used in reconstructive vascular surgery after the completion of the reconstructive procedure to be sure that correction of pathology is optimal.

## Our experience

From May, 1975 to July, 2001, 2251 VA operations on 2187 patients were performed. Among them - 103 distal bypasses to the V3 VA segment. Distribution of operations according to the pathology is given in table 2.

**TABLE 2.** *Distribution of operations according to the type of pathology and sex*

Sex	Atherosclerosis	Kinks and coils	Anomalies	Osteochondrosis	Total
Male	431	294	162	28	915 (40,6%)
Female	342	765	193	36	1336 (59,4%)
Total	773 (34,3 %)	1059 (47,1%)	355 (15,8%)	64 (2,8%)	2251 (100%)

Almost half of operations, performed by us, were due to VA kinking. VA anomalies constituted 15,8 % of all VA operations.

Neurological indications for VA surgery are presented in table 3 (64 cases due to osteochondrosis are not included).

**TABLE 3.** *Indications for surgery in 2187 operation according to VA pathology*

Indications	<u>Atherosclerosis</u>		<u>Kinks and coils</u>		<u>Anomalies</u>	
	Number	Percentage	Number	Percentage	Number	Percentage
VB TIAs	171	22,1	329	31,1	39	11,0
Chronic VBI and TIAs	255	33,0	338	31,9	210	59,1
VB RIND	242	31,3	294	27,7	54	15,2
VB stroke (per history)	64	8,3	96	9,1	51	14,4
Acute VB stroke	11	1,4	2	0,2	1	0,3
Hemispheric stroke	21	2,7	-	-	-	-
Hemispheric TIAs	9	1,2	-	-	-	-
Total	773	100	1059	100	355	100

Types of surgical repair of the Vas, used by us, are presented in table 4.

**TABLE 4.** *Types of surgical repair of vertebral arteries ( 2251 operations )*

Surgical technique	Number of operations	Thrombosis of reconstructed VA	Died
Transsubclavian endarterectomy	14	1	1
VA endarterectomy with vein patch angioplasty	55	1	2
VA reimplantation into the subclavian artery	876	1	2
VA transposition to the site of thyrocervical trunk	872	2	3
VA replacement by autologous vein	14	1	1
VA transposition to the common carotid artery	21	0	1
VA resection with end-to-end anastomosis	16	1	1
VA transposition from the aortic arch to the subclavian artery	53	0	0
Straightening of the VA by resection of the distal subclavian artery	32	0	0
Simultaneous VA and subclavian artery reconstructions	46	0	0
Simultaneous VA and common carotid artery reconstructions	15	0	0
Simultaneous VA and internal carotid artery reconstructions	18	0	1
Scalenectomy and subclavian artery fixation ( Powers procedure)	116	0	1
Reconstruction of the distal VA ( V3 segment )	103	2	0
Total	2251	9 (0,4%)	13 (0,58%)

Transsubclavian endarterectomy for atherosclerotic ostial VA stenosis and VA resection with end-to -end anastomosis for VA kinking were used in early years of VA surgery in the eighth decade and are no longer used by us. Transposition of the VA to the common carotid artery we use only when subclavian artery is not suitable for reimplantation. Most of the deaths occurred in the first years of VA surgery. In the last 1000 VA operations there were 3 deaths. In VA anomalies group we have had one, unrelated to surgery death. In VA kinking group we have had 3 deaths (mortality rate 0,3%). Other 9 deaths occurred in atherosclerotic patients (mortality rate 1,2 % ). Other nonmortal complications are presented in the table 5.

**TABLE 5.** *Perioperative surgical complications in 2251 VA operations*

Type of complication	Number of operations	Percentage
Horner's syndrome	139	6,2
Temporary	130	5,8
Permanent	9	0,4
Phrenic nerve palsy	78	3,5
Temporary	76	3,4
Permanent	2	0,1
Brachial plexalgia	33	1,7
Inferior laryngeal nerve palsy	5	0,2
Wound hemathoma	9	0,4
Lymphocele	3	0,1

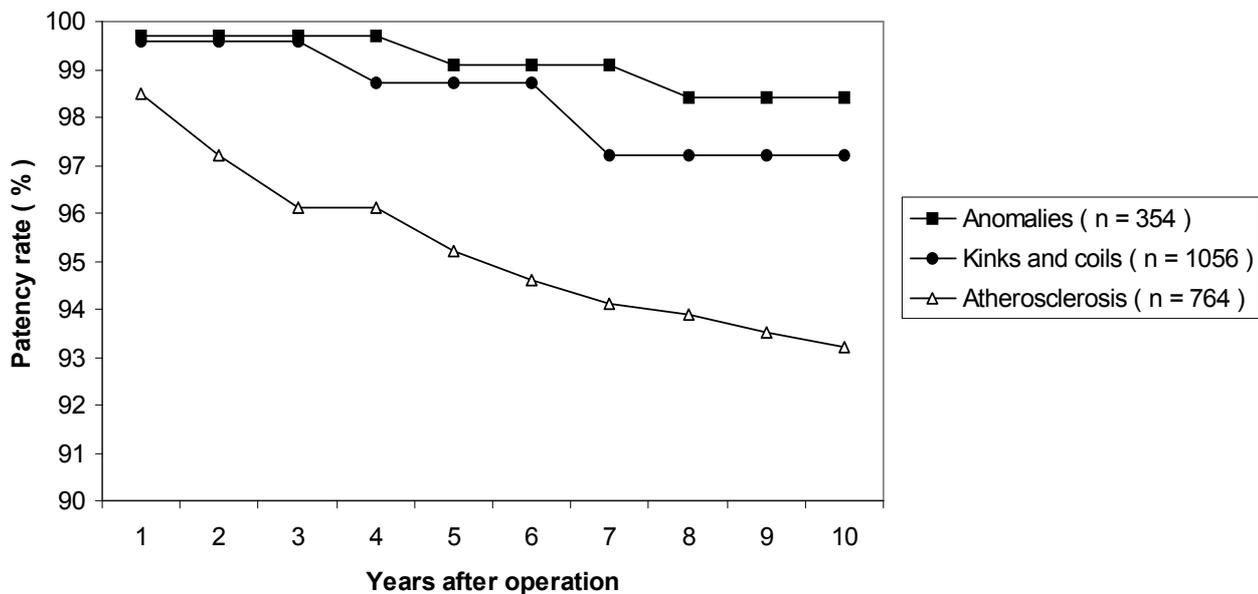
Neurological results of VA surgical repair were assessed after 6 months and later, on a yearly basis, after the operation. At the same patients visits patency and blood flow in the operated VA was assessed by duplex scanner. Interpretation of the results earlier than 6 months after the operation is not reliable. Postoperative results after 6 months are presented in the table 6 (mortal 13 cases are excluded).

**TABLE 6.** *Immediate ( after 6 months ) postoperative results in 2238 VA operations*

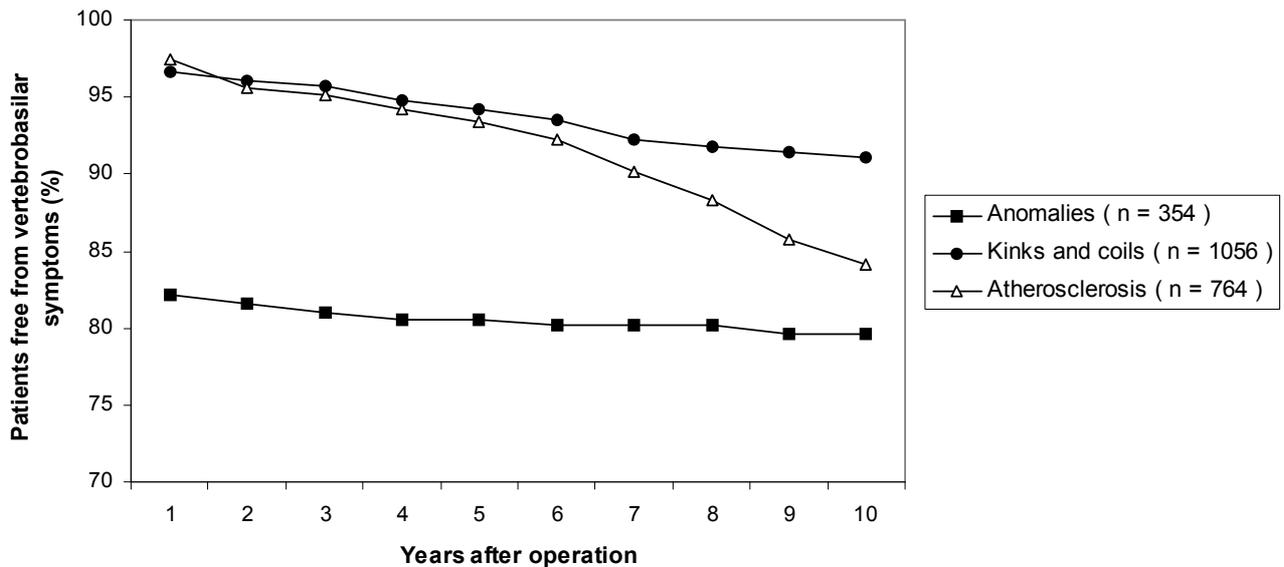
Result	Atherosclerosis ( n= 764 )	Kinks and coils ( n= 1056 )	Anomalies ( n= 354 )
Cured	468 ( 61,3% )	909 ( 86,1% )	272 ( 76,8% )
Improved	277 ( 36,3% )	109 ( 10,3% )	19 ( 5,4% )
Unchanged	17 ( 2,2% )	34 ( 3,2% )	61 ( 17,2 )
Worsened	2 ( 0,3% )	4 ( 0,4% )	2 ( 0,6% )

Relatively high percentage ( 17,2% ) of patients with unchanged neurological status after repair of VA anomalies is mostly due to failure to diagnose other neurological diseases mimicking VBI in these patients in early series of this surgery. The most difficult thing in these patients is to establish the correct diagnosis and the relation of symptoms to the anomalous VA. If the diagnosis is correct and surgical repair of anomalous VA is adequate, the results are rewarding.

**Long term results are presented in Figures 17 and 18.**



**Figure 17.** Cumulative patency rates of operated vertebral arteries in all 3 groups of VA pathology



**Figure 18.** Cumulative freedom from vertebrobasilar symptoms in all 3 groups of VA pathology.

Vertebral arteries have a high patency rate due to high blood flow velocity in these arteries. Most VA occlusions are due to the technical faults, while performing the anastomosis. Long term results are good in all 3 groups of patients. Immediate postoperative results are worst in the VA anomalies group, however they are stable during the time. Surgical correction of VA kinking gives good long-term results and should be used by vascular surgeons, familiar with VA surgery techniques.

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