

# Elasticity of the carotid artery walls as a prognostic factor for the occurrence of restenosis after a surgery for internal carotid artery stenosis

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## Abstract

**Background:** Restoration of carotid artery patency is one of the most frequently performed operations in vascular surgery. One of the most important problems that occur both short- and long-term after carotid endarterectomy is recurrent stenosis. Despite advances in imaging studies and better knowledge of the mechanisms of atherogenesis, the mechanism of restenosis remains unclear. Patients with internal carotid artery atherosclerosis experience decreased elasticity of the intima–media complex, resulting in increased vessel wall stiffness. In the future, measurement of carotid artery elasticity may become a marker for the development of post-surgical stenosis occurring after endarterectomy of both the carotid artery and other peripheral vessels.

**Aim:** To assess the elasticity of carotid artery walls as a prognostic factor for the occurrence of restenosis after a surgery for common carotid artery stenosis.

**Methods:** Classic carotid artery endarterectomy was performed in 180 patients selected on the basis of standard, ultrasound-based recommendations. The phenomenon of restenosis was examined using ultrasound techniques at 3, 6, 9 and 12 months after the surgery. Measurements of carotid artery elasticity were performed using a Vascular Echo Doppler device, and patients were divided into two groups depending on the occurrence (or non-occurrence) of restenosis.

**Results:** Group I (without restenosis) included 156 (86.6%) patients, and Group II (with restenosis) included 24 (13.4%) patients. At 3 and 6 months after the surgery, an increase of the elasticity of vessel walls (coefficient  $\alpha$ ) was observed in both groups, but the differences in the elasticity of the carotid arteries were not significant. At 12 months after the surgery, all patients in Group II (with restenosis) had significantly increased coefficient  $\alpha$  values as compared to Group I patients ( $p < 0.05$ ).

**Conclusions:** A decrease in carotid artery wall elasticity as measured using coefficient  $\alpha$  may be associated with the process leading to the occurrence of restenosis after the surgery. Further research is required in order to confirm the conclusions presented in this paper and to explain potential mechanisms of this phenomenon.

**Key words:** elasticity, restenosis, endarterectomy

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## INTRODUCTION

Restoration of carotid artery patency is one of the most frequently performed operations in vascular surgery. One of the most important problems that occur both short- and long-term after carotid endarterectomy is recurrent stenosis. Studies indicate that the risk of restenosis is 10% during the first year, 3% during the second year, and 2% during the third year [1]. Remodelling of all 3 layers of the arterial wall, i.e. intima, media and adventitia, may occur following revas-

cularisation, as shown in clinical and experimental studies [2]. The pathogenesis of restenosis may involve such local factors as infection, inflammation, growth factors and cytokines, and the role of lymphocytes, macrophages and activated smooth muscle cells [3]. It seems that traditional risk factors for atherosclerosis do not play a key role in restenosis [4]. Despite advances in imaging studies and better knowledge of the mechanisms of atherogenesis, the mechanism of restenosis remains unclear. The basis for our study was an

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**Table 1.** Characteristics of two patient groups (mean values  $\pm$  standard deviation or numbers and percentages)

	Group I (no restenosis)	Group II (restenosis)
Number of patients	156 (86.6%)	24 (13.4%)
Age [years]	71 $\pm$ 7.2	66 $\pm$ 7.8
Women	48 (30.7%)	16 (66.6%)
Men	108 (69.2%)	8 (33.3%)
Ischaemic heart disease	82 (52.5%)	16 (66.6%)
Smoking	120 (76.9%)	16 (66.6%)
Hypertension	102 (65.3%)	14 (58.3%)

assumption that changes in the elasticity of carotid arteries may be a prognostic factor for recurrent stenosis. In studies on restenosis following coronary angioplasty, a relationship was found between vascular stiffness and restenosis [5, 6]. In patients with atherosclerotic lesions in the internal carotid artery, elasticity of the intima–media complex is reduced, leading to increased vessel wall stiffness.

The aim of the study was to evaluate the elasticity of carotid artery walls as a prognostic factor for the occurrence of restenosis after a surgery for internal carotid artery (ICA) stenosis.

## METHODS

### Study group

We prospectively evaluated 180 patients (68 women and 112 men, mean age  $67 \pm 8.9$  years) who underwent conventional carotid endarterectomy in the Department of Vascular Surgery and Angiology at Centre for Postgraduate Medical Education. Study group characteristics are shown in Table 1. The patients were divided in two groups. Group I included 156 patients (48 women and 108 men, mean age  $71 \pm 7.2$  years) (86.6%) without restenosis, and Group II included 24 patients (16 women and 8 men, mean age  $66 \pm 7.8$  years) (13.4%) with recurrent carotid artery stenosis following endarterectomy. Restenosis was diagnosed at mean  $252 \pm 37$  days after the surgery. The mean diameter of the evaluated vessel was  $6.7 \pm 1.9$  mm in patients without restenosis and  $6.3 \pm 1.2$  mm in patients with restenosis.

### Carotid endarterectomy

Carotid endarterectomy was performed using the standard technique with direct occlusion. A non-absorbable 6-0 monofilament was used for suturing at the site of arterial incision. Standard heparin doses were administered during the procedure. Following the surgery, patients were administered aspirin at the dose of 75 mg daily. Ultrasound monitoring of the anastomosis was used to exclude early restenosis related to surgical technique. Patients with symptomatic carotid artery atherosclerosis according to the NASCET study criteria and

the diameter of the carotid artery above 8 mm were eligible for the study [7].

Exclusion criteria included transient ischaemic attack or stroke during the surgery, atherosclerotic lesions in subclavian arteries precluding accurate blood pressure measurements in the brachial artery, uncontrolled hypertension, cardiac arrhythmia precluding pressure waveform recording and analysis, and diabetes. We also excluded patients with more than 50% contralateral carotid artery stenosis and patients with recurrent stenosis below 6 months. Patients operated using other techniques (patching or eversion) were also excluded.

### Measurements of carotid artery elasticity

Measurements of carotid artery elasticity were performed using a Vascular Echo Doppler (VED) device, designed and manufactured in the Department of Ultrasound Waves at the Polish Academy of Sciences. Measurements were performed in the A-mode at baseline and at 6 and 12 months after the surgery on both the operated and contralateral side. The device allows non-invasive evaluation of vascular haemodynamic parameters. It consists of a recording module connected to a computer. The transducer includes two probes, a 6.75 MHz pulsed wave Doppler probe that allows real-time measurements of the vessel diameter (precision  $7 \mu\text{m}$ ), and a 4.5 MHz continuous wave Doppler probe for flow measurements [8]. The VED device allows non-invasive measurements of carotid artery elasticity based on the correlation between artery cross-sectional area and blood pressure [9]. Based on this correlation, a logarithmic coefficient of the arterial wall stiffness ( $\alpha$ ) was calculated using the following formula:

$$\alpha = \frac{S_{\min}}{S_{\max} - S_{\min}} \ln\left(\frac{P_s}{P_d}\right) \quad \text{or} \quad \alpha = \frac{D_{\min}}{D_{\max} - D_{\min}} \ln\left(\frac{P_s}{P_d}\right)$$

where  $D_{\min}$  is the minimum artery diameter,  $D_{\max}$  is the maximum artery diameter, and  $P_d$  and  $P_s$  are corresponding diastolic and systolic pressure values.

Higher coefficient  $\alpha$  values indicate higher arterial wall stiffness, and lower values indicate that the vessel wall is more elastic.

### Identification of restenosis

The diagnosis of recurrent ICA stenosis was based on duplex Doppler examination using the Aloka  $\alpha$ -10 device with a linear 7.5 MHz probe. Similarly to other digital devices for vascular examinations, this device runs in B-mode and is equipped with all functions of colour-coded flow imaging. Examinations were performed using the criteria proposed by Grant et al. [10]. Recurrent stenosis was defined as at least 50% stenosis of the lumen of the ICA at the site of primary reconstruction [11]. Examinations were performed before the surgery and at 6 and 12 months after the surgery. Measurements were performed in a supine position after 10 min of rest. VED de-

**Table 2.** Descriptive statistics and tests of normal distribution

Variable	Median [interquartile range]	Min-max	Kolmogorov-Smirnov	
			Z (df)	p
$\alpha$ 1-O	4.16 [1.86]	2.15–9.65	0.19 (180)	< 0.001
$\alpha$ 2-O	6.23 [2.52]	3.58–11.50	0.16 (180)	< 0.001
$\alpha$ 3-O	5.48 [2.33]	3.13–10.58	0.13 (180)	< 0.001
$\alpha$ 1-NO	4.21 [1.31]	2.58–8.12	0.16 (180)	< 0.001
$\alpha$ 2-NO	5.23 [1.31]	3.58–9.21	0.11 (180)	< 0.001
$\alpha$ 3-NO	5.05 [1.56]	3.45–8.85	0.10 (180)	< 0.001

$\alpha$ 1-O and  $\alpha$ 1-NO — coefficient  $\alpha$  measured before the surgery;  $\alpha$ 2-O and  $\alpha$ 2-NO — coefficient  $\alpha$  measured at 6 months after the surgery;  $\alpha$ 3-O and  $\alpha$ 3-NO — coefficient  $\alpha$  measured at 1 year after the surgery; O — operated artery; NO — non-operated artery

vice transducer was placed over the common carotid artery (CCA) approximately 2–3 cm proximal to the bifurcation and 1 cm proximal to the atherosclerotic plaque. The right and left carotid artery was evaluated in all patients. Examinations were performed by the same physician using the same device.

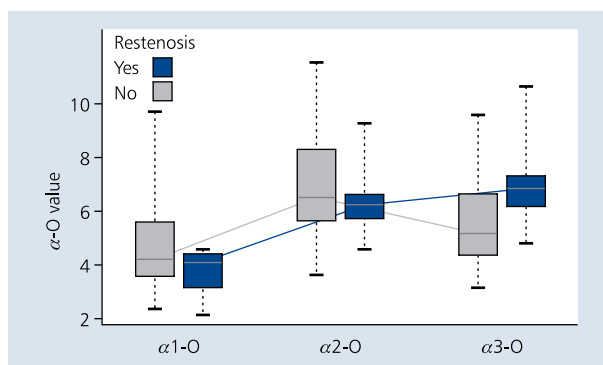
### Statistical analysis

Results were evaluated using a number of statistical methods that were adequate for the analysed data. First, descriptive statistics were used to characterise the study group. Then, normal distribution of the quantitative variables was verified using the Kolmogorov-Smirnov test. We tested preoperative coefficient  $\alpha$  values at the operated ( $\alpha$ -O) and non-operated ( $\alpha$ -NO) side as predictors of restenosis. The analysis had 3 aims: to check whether restenosis may be predicted based on coefficient  $\alpha$  values, what coefficient  $\alpha$  value is the best diagnostic cutoff for prediction of restenosis, and whether  $\alpha$ -O or  $\alpha$ -NO is a better predictor. Thus, we first used two separate univariate logistic regression models to evaluate how the likelihood of restenosis rose with increasing  $\alpha$ -O and  $\alpha$ -NO values, then we compared diagnostic parameters of both models at varying coefficient  $\alpha$  values using receiver operating characteristic (ROC) curves, and finally we compared the diagnostic values of both models using the DeLong method.

### RESULTS

Distributions of all analysed variables differed significantly from a normal distribution ( $p < 0.001$ ). For this reason, further analyses were performed with the use of non-parametric tests. Detailed description of distribution of  $\alpha$ -O and  $\alpha$ -NO values, along with tests for normal distribution, is shown in Table 2.

Baseline coefficient  $\alpha$  values were strongly associated with coefficient  $\alpha$  values at 1 year after the surgery, both on the operated ( $\rho = 0.72$ ,  $p < 0.001$ ) and non-operated side ( $\rho = 0.82$ ,  $p < 0.001$ ). The association between coefficient  $\alpha$  values on the operated and non-operated side was moderate, both at baseline ( $\rho = 0.50$ ,  $p < 0.001$ ) and at 1 year after the surgery ( $\rho = 0.54$ ,  $p < 0.001$ ).

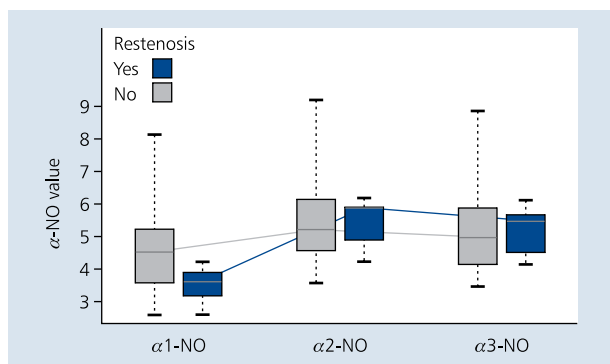


**Figure 1.** Coefficient  $\alpha$ -O values (in the operated artery) in relation to timing of the measurement and the occurrence of restenosis

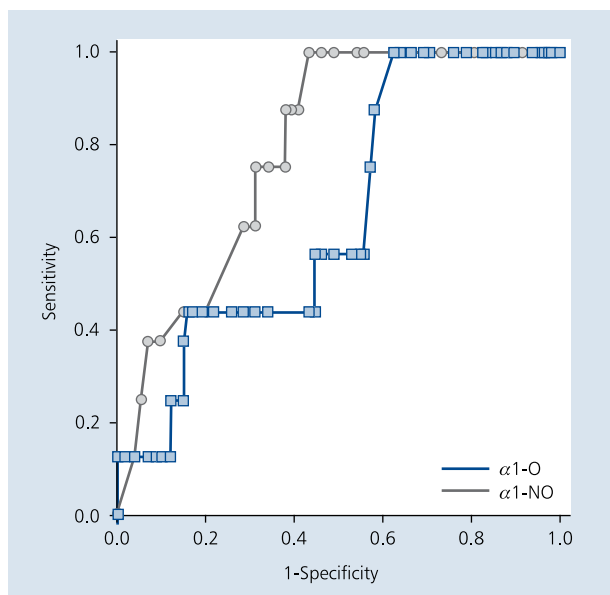
Median coefficient  $\alpha$  value on the operated and non-operated side was higher at 6 months after the surgery compared to baseline both in patients with restenosis and in those without restenosis. At 1 year after the surgery, median  $\alpha$ -NO decreased in both groups, while median  $\alpha$ -O increased further in the restenosis group and decreased in the non-restenosis group.

In patients with restenosis,  $\alpha$ -NO increased at a significantly higher rate during the first 6 months after the surgery compared to patients without restenosis ( $U = 64$ ,  $p < 0.001$ ), while no significant difference in the increase in  $\alpha$ -O was found between these two groups ( $U = 1916$ ,  $p = \text{NS}$ ). In patients with restenosis,  $\alpha$ -O was significantly higher at 12 months after the surgery compared to patients without restenosis ( $U = 56$ ,  $p < 0.001$ ), while  $\alpha$ -NO did not differ significantly between these two groups ( $U = 2490$ ,  $p = \text{NS}$ ) (Figs. 1, 2).

The risk of restenosis was significantly associated with both  $\alpha$ 1-O and  $\alpha$ 1-NO ( $p < 0.001$  for both), although more strongly with an increase in  $\alpha$ 1-NO compared to  $\alpha$ 1-O. The likelihood of restenosis was about 4-fold lower (reduced by 75% [52–86%]) with an increase in  $\alpha$ 1-NO by 1 unit, and

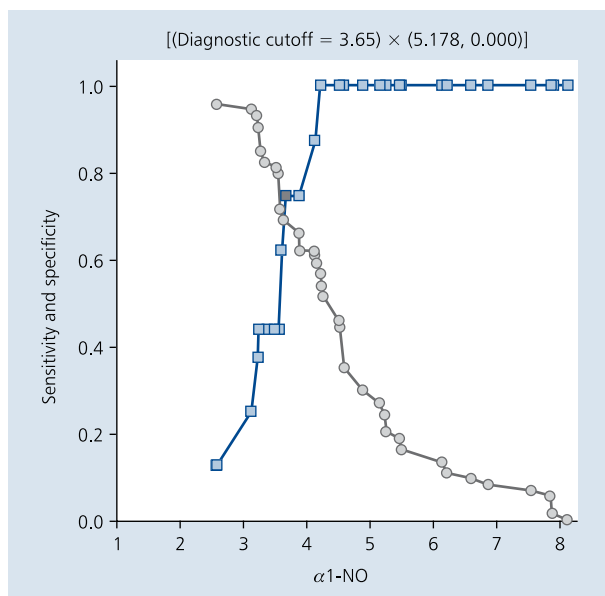


**Figure 2.** Coefficient  $\alpha$ -NO values (in the non-operated artery) in relation to timing of the measurement and the occurrence of restenosis

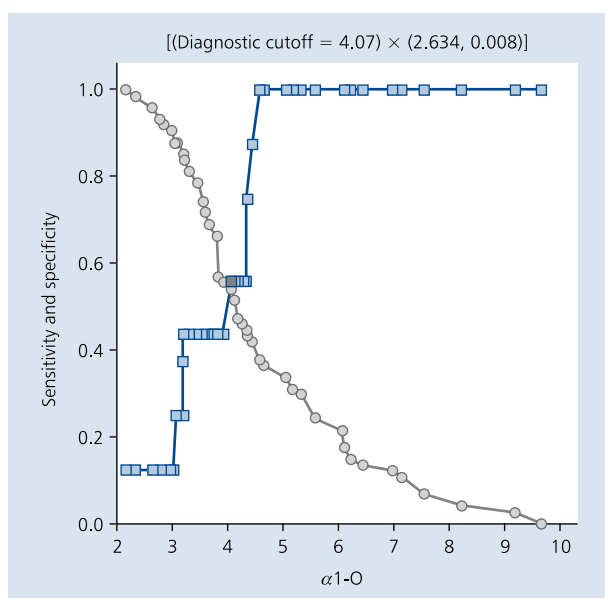


**Figure 3.** Comparison of the sensitivity and specificity of  $\alpha$ 1-NO (grey graph) and  $\alpha$ 1-O (blue graph) predictive models

about twofold lower (reduced by 46% [21–64%]) with an increase in  $\alpha$ 1-O by 1 unit.  $\alpha$ 1-NO was a better predictor of restenosis compared to  $\alpha$ 1-O, as the model for  $\alpha$ 1-NO was better fitted to data and showed better diagnostic parameters. Comparison of ROC curves (Fig. 3) using the DeLong method also indicated that diagnostic parameters of  $\alpha$ 1-NO were significantly better compared to  $\alpha$ 1-O ( $Z = 2.70$ ,  $p = 0.006$ ) (Fig. 3). This analysis indicated that the occurrence of restenosis was better predicted by  $\alpha$ 1-NO than by  $\alpha$ 1-O. The optimal diagnostic cutoff  $\alpha$ 1-NO value predicting restenosis may be identified as the intersection point of the two curves in Figure 4, showing the sensitivity and specificity of various  $\alpha$ 1-NO values. Thus, the optimal diagnostic cutoff  $\alpha$ 1-NO value is 3.65 (sensitivity 75%, specificity 69%, positive predictive value [PPV] 34%, negative predictive value [NPV] 95%).



**Figure 4.** Determination of the optimal diagnostic cutoff  $\alpha$ 1-NO value



**Figure 5.** Determination of the optimal diagnostic cutoff  $\alpha$ 1-O value

This is a better predictor of restenosis compared to  $\alpha$ 1-O, with the optimal diagnostic cutoff  $\alpha$ 1-O value of 4.07 (sensitivity 56%, specificity 55%, PPV 21%, NPV 85%) (Fig. 5).

Etiologic factors of recurrent carotid artery stenosis include female gender, diabetes, and smoking. Women were more prevalent among our patients with restenosis (67% vs. 31%). Multivariate analysis indicated that  $\alpha$ 1-NO was associated with restenosis, while gender and age were not. Logistic regression analysis, with age, gender and  $\alpha$ 1-NO as

predictors, showed that the likelihood of restenosis was significantly related only to  $\alpha$ 1-NO (OR 0.26,  $p < 0.001$ ), and it was not significantly related to age (OR 1.03,  $p > 0.05$ ) or gender (OR 0.69,  $p > 0.05$ ). The logit model showed good fitness to data, and the Hosmer-Lemeshow test was not significant ( $p > 0.5$ ).

## DISCUSSION

Our study indicates significant differences in the progression of CCA wall stiffness in patients after endarterectomy. In patients without restenosis, arterial stiffness increased during a 6-month period after the surgery, and in patients with restenosis, further progression of CCA wall stiffness was seen at 1 year after the surgery. When predicting restenosis, evaluation of elasticity of non-operated CCAs shows better diagnostic value compared to that of operated arteries.

Despite a large increase in the interest in mechanical properties of the arteries, invasive measurements of arterial stiffness, and Doppler measurements of vascular resistance, pulse wave velocity, and reflected arterial wave, no studies on non-invasive measurements of carotid artery elasticity in patients with restenosis following carotid endarterectomy are available in the literature.

Among the evaluated coefficients related to measurements of carotid artery elasticity ( $\alpha$ , compliance, and distensibility), stiffness coefficient  $a$  is characterised by the largest reproducibility and the smallest scatter compared to the mean values. This is related to the analytical form of the coefficient  $a$  which reflects a non-linear, logarithmic relation between transverse arterial dimensions and blood pressure. For this reason, coefficient  $a$  best characterised age- or disease-related changes in the arterial wall.

In 1835, Löbstein defined arteriosclerosis as the loss of vessel wall elasticity but did not explain reasoning leading to these conclusions. Features of arteriosclerosis include thickening and stiffening of the arterial wall. A gradual increase in the arterial wall thickness is an element of progressive arteriosclerosis which may be accompanied by atherosclerotic lesions. It is thought that progressive stiffening of the arterial wall may be associated with structural vascular changes, such as increasing diameter and wall thickness, and calcification associated with such risk factors as age, diabetes or arterial hypertension. Recently, a large group of specialists proposed a more convincing concept of factors increasing the risk of permanent arterial changes. According to the World Health Organisation definition, atherosclerosis is a net effect of various changes in the intima: accumulation of fatty bodies, neutral and acidic glycosaminoglycans, blood and its components, connective tissue, and calcium deposits, along with changes in the media. Atherosclerosis is a progressive disease that becomes irreversible at some quite early stage. In the early phase, it may manifest with local oedema, steatosis or mural microthrombus, progressing to yellow atheroma or greyish

sclerotic lesion (fibrous connective tissue). These processes result in a gradual loss of vessel wall elasticity due to fibrosis and loss of smooth muscle and elastic fibres in the media. Early atherosclerotic lesions may be reversible, so methods to detect them are searched for. Our initial findings indicate a large increase in stiffness of apparently healthy carotid arteries. This means that a change in mechanical properties of the arteries occurs also at sites other than those where disease manifests and subsequent perioperative trauma occurs. Histological and immunohistochemical studies of primary atherosclerotic plaques in patients with or without restenosis showed differences in their composition [12]. Restenosis is a result of excessive healing response within the vessel wall following revascularisation, involving migration of smooth muscle cells to the intima, their proliferation, and production of intercellular matrix, leading to formation of neointima [13, 14]. During this complex process, intraplaque bleeding, thrombosis, and other mitogenic factors may lead to expression of multiple growth factors and release of proinflammatory cytokines, resulting in smooth muscle cell proliferation and migration from the media [15].

Carotid endarterectomy is one of the most common procedures in vascular surgery. The annual number of these procedures in Poland is about 2500. Epidemiological studies indicate that the rate of recurrent ICA stenosis is 7–34% at 5 years. According to Frericks et al. [16], recurrent arterial lumen stenosis by more than 50% develops during the first 4 years after the procedure on average in about 10% of patients, with more than 70% lumen stenosis in half of these patients [17].

Management of patients with carotid artery restenosis is still controversial. Based in the available literature, it is difficult to provide an unequivocal assessment of the risk of complications, indications for intervention, ways to prevent recurrent stenosis, and the choice of the treatment method. Some authors suggest that restenosis has no effect on neurological complications, while others believe that the number of such complications increases with higher rates of recurrent stenoses. Reports have been published to suggest that the presence of haemodynamically significant restenosis does not affect the risk of stroke or death. Ganesan et al. [18] found that cerebral ischaemia was noted in 13.3% of patients with recurrent stenosis by less than 50% compared to 19.2% of patients with more than 50% stenosis, a nonsignificant difference. Based on these data, he concluded that there is no direct association between the degree of stenosis and the number of neurological complications [19]. Healy evaluated 301 patients after revascularisation of the ICA and found that among patients with restenosis by more than 50%, the degree of recurrent stenosis during 7 years of follow-up was 31%, and the cumulative regression rate was 10%. Similar values were given by Nicholls (22%) and AbuRahma (15%). Debatable observations were reported by Sanders et al. [20] who



found that if the degree of recurrent stenosis is more than 65% during the first 6 months after the procedure, it shows an increasing dynamics, and if it is less than 65%, regression is seen in 60% of patients. It is related to hyperplasia of intimal smooth muscle cells which undergo regression as a result of ongoing repair processes.

Studies indicate that gender plays a major role in recurrent stenosis. All authors highlight the fact that restenosis is seen more frequently in women compared to men. Similarly, the female-to-male ratio in our study was 2:1. The reason for this has not been clearly explained. Initial vessel size is likely an important factor, as arterial diameter in women is usually smaller than in men. Hormonal factors may also have some effect [20, 21].

The role of plaque morphology as a factor contributing to recurrent carotid artery stenosis seems underestimated in the literature. In his study, Madycki and Staszkiwicz [22] highlighted the effect of inflammatory factors on plaque stability and thus also on future carotid artery restenosis. A major mechanism of restenosis following endarterectomy is inflammation induced by surgical vessel trauma and proliferation of neointima [23, 24]. It is believed that proliferation is stimulated by inflammatory cells, i.e. monocytes/macrophages [25], as activated leukocytes (particularly monocytes and granulocytes), platelets, adhesion molecules, and monocyte and platelet aggregates are often found in blood [25, 26], along with increased interleukin-6 (IL-6) level [27]. On the exposed intima, monocytes undergo intense early accumulation, followed by transformation into activated macrophages [28]. Inflammatory mediators produced by these cells stimulate release of cytokines (tumour necrosis factor- $\alpha$ , IL-1, IL-2, IL-6, IL-8), growth factors (platelet-derived growth factor), vasculogenic factors (vascular endothelial growth factor), adhesion molecules, and free radicals [29]. Production of biologically active inflammation mediators stimulated migration and proliferation of smooth muscle cells, resulting in intimal hyperplasia and recurrent stenosis [30].

Physical properties of a vessel are related to its wall structure and changes in composition. The ability to increase vessel diameter in relation to pressure (pulsatile blood flow) may be considered a measure of arterial wall elasticity/stiffness. This relation is described by various parameters, for example compliance, distensibility, elasticity, and bulk modulus. Experimental studies identified functions that allow much more precise approximation of the relation between pressure and vessel diameter. One of such functions was developed by Polish scientists Powałowski and Peńsko. It is a logarithmic function describing the relation between changes in vessel diameter and pressure, yielding an index known as vessel stiffness coefficient  $\alpha$ . This function significantly limits the effect of blood pressure, particularly systolic blood pressure, on this index. Measurements of this parameter are made non-invasively using ultrasound Doppler techniques. A change

in vascular stiffness is considered a marker of atherosclerosis and coronary artery disease [31].

A study by Gatzka et al. [32] led to a concept of identifying patients at an increased risk of ischaemic heart disease by measuring central arterial compliance. A change in arterial elasticity may be an early indicator of potentially occurring atherosclerotic process. This process may be characterised by varying severity. Processes leading to formation of an atherosclerotic plaques include infiltration with lipoproteins (particularly oxidized low-density lipoproteins), various inflammatory cells (such as macrophages), or T cells [33]. Such changes are not seen in ultrasound imaging but may, for example, result in clinical manifestations, such as coronary syndromes with patients with advanced abdominal aortic atherosclerosis.

## CONCLUSIONS

Based on our findings, it may be concluded that significant differences in the progression of CCA wall stiffness exist in patients after endarterectomy. In patients without restenosis, arterial stiffness increases during 6 months after the surgery, and in patients with restenosis, CCA wall stiffness undergoes further progression during 1 year after the surgery. Evaluation of changes in the elasticity of CCAs in patients after endarterectomy has a predictive value for restenosis. When predicting restenosis, evaluation of elasticity of non-operated CCAs shows better diagnostic value compared to that of operated arteries. Confirmation of our conclusions and explaining potential mechanisms of this phenomenon requires further studies. If confirmed, our findings may significantly change the criteria of selecting patients for ICA endarterectomy and determine new indications for surgical treatment.

**Conflict of interest:** none declared

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# Elastyczność ścian tętnic szyjnych jako czynnik prognostyczny wystąpienia restenozy po operacji zwężenia tętnicy szyjnej wewnętrznej

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## Streszczenie

**Wstęp:** Udrożnienie tętnic szyjnych jest jednym z najczęściej wykonywanych zabiegów w chirurgii naczyniowej, a jednym z ważniejszych problemów wczesnych i odległych po endarterektomii tętnic szyjnych są ich nawrotowe zwężenia. Obecnie, mimo dobrze rozwiniętej diagnostyki obrazowej oraz poznania mechanizmów tworzenia zmian miażdżycowych, wciąż niejasny pozostaje mechanizm powstawania restenoz. U pacjentów z miażdżycą tętnicy szyjnej wewnętrznej stwierdza się spadek elastyczności kompleksu intima-media, a co się z tym wiąże większą sztywność ściany naczynia. Pomiar elastyczności tętnic szyjnych może się stać w przyszłości markerem narastania stenozy pooperacyjnej po endarterektomii tętnic szyjnych i pozostałych naczyń obwodowych.

**Cel:** Celem pracy była ocena elastyczności ścian tętnic szyjnych jako czynnika prognostycznego wystąpienia restenozy po operacjach zwężenia tętnicy szyjnej wspólnej.

**Metody:** U 180 pacjentów kwalifikowanych na podstawie klasycznych wskazań opartych na badaniu ultrasonograficznym (USG), wykonano klasyczną endarteriektomię tętnicy szyjnej. Zjawisko restenozy badano za pomocą USG 6 i 12 miesięcy od operacji. Badanie przeprowadzono zarówno na tętnicy planowanej do operacji, jak i tętnicy po przeciwnej stronie. Pomiar elastyczności tętnic szyjnych wykonywano za pomocą aparatu o nazwie Vascular Echo Doppler, losowo włączając chorych do dwóch grup wydzielonych pod kątem występowania restenozy lub jej braku.

**Wyniki:** Grupa I (bez restenozy) liczyła 156 (86,6%) pacjentów, a Grupa II (z restenozą) — 24 (13,4%) chorych. W okresie 6 miesięcy po operacji w obu grupach zaobserwowano wzrost wartości współczynnika elastyczności ścian naczyń (współczynnik  $\alpha$ ), jednak różnice dotyczące elastyczności tętnic szyjnych nie były znamienne statystycznie. W okresie 12 miesięcy po operacji u wszystkich pacjentów z Grupy II (z restenozą) stwierdzono istotny statystycznie wzrost wartości współczynnika  $\alpha$  w porównaniu z pacjentami z Grupy I (bez restenozy) ( $p < 0,001$ ). Mediana współczynnika elastyczności  $\alpha$  mierzonego na tętnicy operowanej ( $\alpha$ -O) i nieoperowanej ( $\alpha$ -NO) była wyższa po 6 miesiącach po operacji niż przed zabiegiem, zarówno u pacjentów z restenozą, jak i bez niej. Mediana współczynnika  $\alpha$ -NO następnie spadała po roku w obu grupach. Mediana współczynnika  $\alpha$ -O rosła dalej w grupie, w której wystąpiła restenoza i spadała w grupie, w której nie nastąpiła restenoza. Ryzyko wystąpienia restenozy było istotnie związane zarówno z wartością  $\alpha$ 1-O, jak i  $\alpha$ 1-NO ( $p < 0,001$  dla obu wskaźników), choć w większym stopniu ze wzrostem  $\alpha$ 1-NO niż  $\alpha$ 1-O. Ryzyko wystąpienia restenozy było ok. 4-krotnie większe [zwiększało się o 75% (52–86%)] wraz z większym  $\alpha$ 1-NO o jedną jednostkę, natomiast ok. 2-krotnie większe [zwiększało się o 46% (21–64%)] wraz ze wzrostem  $\alpha$ 1-O o jedną jednostkę.  $\alpha$ 1-NO było lepszym predyktorem — model był lepiej dopasowany do danych i charakteryzował się lepszymi parametrami diagnostycznymi wystąpienia restenozy niż  $\alpha$ 1-O.

**Wnioski:** Spadek elastyczności ścian tętnic szyjnych mierzony za pomocą współczynnika  $\alpha$  może mieć znaczenie prognostyczne w wystąpieniu restenozy po zabiegu chirurgicznym. Potwierdzenie wniosków przedstawionych w niniejszym opracowaniu i wyjaśnienie potencjalnych mechanizmów tego zjawiska wymaga przeprowadzenia dalszych badań.

**Słowa kluczowe:** elastyczność, restenoza, endarterektomia

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