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A comparison of age-related changes in axial prestretch in human carotid arteries and in human abdominal aorta

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Abstract. It is known that large arteries in situ are subjected to significant axial prestretch. This prestretch plays an important physiological role in optimizing the biomechanical response of an artery. It is also known that the prestretch declines with age. However, a detailed description of age-related changes in prestretch is available only for the abdominal aorta and for the femoropliteal artery. Our study presents results of measurements of axial prestretch in 229 left common carotid arteries excised in autopsies. It was found that the prestretch of the carotid artery correlates significantly with age (R = –0.783, *p–value* < 0.001). A linear regression model was used to fit the observations. Simultaneously with the measurement of the prestretch in the carotid artery, the axial prestretch was also measured in abdominal aorta. By comparing data obtained from these locations, it was concluded that the axial prestretch in the carotid artery is greater than in the abdominal aorta, and that atherosclerosis develops more rapidly in the abdominal aorta than in the carotid artery. Histological sections obtained from 8 carotid arteries and aortas suggest that the medial layer of the left common carotid artery is significantly thinner than aortic media (median/IQR: 0.343/0.086 mm vs. 0.482/0.172 mm, p = 0.02 in Wilcoxon signed-rank test) and simultaneously that carotid media contains a lower number of elastic membranes (median/IQR: 26.5/11.8 vs. 31.5/11.8, p = 0.07 in the Wilcoxon signed-rank test). This could be a reason for the different extent of the prestretch observed in aorta and in carotid artery. Our data sample also contains 5 measurements of the axial prestretch in abdominal aortas suffering from an aneurysm. It was found that aneurysmatic aortas also exhibit axial retraction when excised from in situ position. To the best of our knowledge, this is the first time that detailed data characterizing axial prestretch of the human left common carotid artery has been presented.

Keywords: Abdominal aorta, age, autopsy, biomechanics, carotid artery, prestretch, retraction.

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1. Introduction

It is well known that large arteries, e.g. aorta and carotids, and also transitional arteries (transitioning from elastic type to muscular type), e.g. iliac, femoral and popliteal arteries, are significantly longitudinally prestretched in their in situ positions (Horný et al., 2011, 2014a; Learoyd and Taylor, 1966; Dobrin et al., 1970; Bergel 1961; Han and Fung, 1995; Kamenskiy et al., 2014). Biomechanical analyses show that axial prestrain and prestress play important physiological roles: they minimize the variation of the axial wall stress and strain within the cardiac cycle, and enhance the circumferential distensibility of the artery wall (Horný et al., 2014b, 2016; Schulze-Bauer et al., 2003; Van Loon et al., 1977; Dobrin et al., 1970). It is also clear nowadays that axial prestress is not negligible in mechanobiological processes, e.g. in the adaptation response to elevated blood pressure, endothelial damage, and of course in response to changes in axial loading (Jackson et al., 2002; Lawrence and Gooch, 2009; Lee et al., 2008, 2010; Han and Jiang, 2012; Davis et al., 2005). Sufficient axial tension is also necessary to prevent an artery from buckling, which can cause the artery to deflect perpendicular to its longitudinal axis and to become tortuous (Han 2007, 2008).

Axial prestretch declines in ageing (Horný et al., 2011, 2014a; Learoyd and Taylor, 1966). On occasion, it may change from pretension to pre-compression (a segment of an artery elongates when excised; Schulze-Bauer et al., 2003; Kamenskiy et al., 2015). From humans, and also from animal models, it is known that the extent of the prestretch depends on the location in the arterial tree (Learoyd and Taylor, 1966; Han and Fung, 1995). However, the only parts of the human arterial tree for which a detailed description of age-related changes in axial prestretch is available are the abdominal aorta (Horný et al., 2011, 2013, 2014a) and the femoropliteal artery (Kamenskiy et al., 2015).

Our main goal is to present how ageing changes the axial prestretch of the human left common carotid artery (LCCA), and to compare these changes with the prestretch observed in the infrarenal part of the human abdominal aorta (AA). Conclusions that will be presented may be summarized as: (1) the prestretch of LCCA declines significantly with age, and even pre-compressed arteries can be observed, (2) left common carotid artery exhibits higher axial prestretch than infrarenal aorta, (3) a statistical analysis showed that although atherosclerosis is frequently developed in both AA and LCCA, data suggests that atherosclerosis is not a factor capable of explaining the ageing-induced decrease of the prestretch, either in AA or in LCCA.

2. Methods

Data describing the in situ and excised lengths of LCCA and of the infrarenal part of AA, their circumferences, and also the age and the degree of atherosclerosis in both sites, were collected during regular autopsies of Caucasian cadavers of known age and known time of the death, conducted in the last two years by T.A. and M.K. in the Královské Vinohrady University Hospital in Prague, and in the Regional Hospital in Liberec. The post-mortem usage of human tissue was approved by the Ethics Committee of the Third Faculty of Medicine of Charles University in Prague. No putrefied bodies were involved. The degree of atherosclerosis (ATH) was examined by an experienced pathologist, and was quantified on a scale from 0 to 4 according to morphological features: 0 – intact artery and fatty streaks; 1 – fibro-fatty plaques; 2 – advanced plaques; 3 – calcified plaques; 4 – ruptured plaques (Kumar et al.,

2010). Axial prestretch, λ_z, was defined as the ratio of in situ length, l, to ex situ length, L, $\lambda_z = l/L$. Upper indices C and A are used to distinguish between LCCA and AA. The measured circumference of a cut arterial ring was divided by $2*\pi$ to obtain the average radius r. Eight representatives of AA and LCCA were used in histological analysis. Five-micrometer sections were stained by Orcein to highlight elastic membranes in the medial layer of the artery wall in transmission light microscopy.

2.1 Regression, correlation and statistics

The linear correlation coefficient R was used to quantify the dependence between age and prestretch. Hypothesis H_0: $R = 0$ was tested with $T = R\sqrt{((n-2)/(1-R^2))}$ statistics against H_A: $R \lessgtr 0$. Based on the least squares method, the collected data was fitted with the linear regression model $y = b_1x+b_0$, where y denotes dependent variable and x denotes independent variable, and b_i (i = 0, 1) are regression coefficients. The confidence interval for b_i was obtained as $b_i \pm t_{1-\alpha/2}(n-2)s(b_i)$, where $t_{1-\alpha/2}(n-2)$ is a quantile of the *Student T* distribution with $n-2$ degrees of freedom at a confidence level of $1-\alpha$, and $s(b_i)$ is the estimated standard error of b_i. To estimate how the variability of the observations affects the regression model predictions, the prediction intervals (PI) were also computed. In the linear regression model, PI can be obtained as $\pm t_{1-\alpha/2}(n-2)S_e\sqrt{(1+1/n+(x-x_m)^2/S^2_{total})}$. Here $t_{1-\alpha/2}(n-2)$ is again the quantile of the *Student T* distribution with $n-2$ degrees of freedom at a confidence level of $1-\alpha$, S_e is the residual standard deviation, x_m is the arithmetic mean of the independent variable, and S^2_{total} is the total sum of squares.

The data was ordered with respect to ATH. In order to reveal whether ATH disease has the same age-related distribution in LCCA and in AA, subgroups of the same degree of ATH in LCCA and in AA were compared by the *Kolmogorov-Smirnov test* (H_0: two samples have the same distribution function, against H_A: the distributions are different). To reveal whether atherosclerosis, which is well-known to be correlated with age, is a factor capable of explaining changes in the prestretch, data was divided into intervals where correlation between age and prestretch was proved to be non-significant. Subsequently, the *Kruskal-Wallis H test* was used to test the hypothesis that subgroups corresponding the degree of ATH have the same mean value of the prestretch. When only two subgroups were compared, the *Mann-Whitney U test* was used.

The paired *Wilcoxon signed-rank test* was conducted to reveal whether significant differences of the axial prestretch between LCCA and AA really do exist. The axial prestretch in abdominal aorta and in carotid artery were also compared in age-related change independent subintervals again by means of the *Wilcoxon signed-rank test* and also by the *Kolmogorov-Smirnov test*. In the entire study, a significance level of $\alpha = 0.05$ was used. If not otherwise specified, two-tailed tests are considered.

3. Results and Discussion

In the last two years, axial prestretch of LCCA and AA was measured in 258 individuals. 17 individuals (6.6% of 258) had tortuous LCCA. 5 individuals (1.9%) had aneurysm in AA, and 7 cadavers (2.7%) had tortuous abdominal aorta. One cadaver (0.4%) had totally mineralized aorta, and one had an aorto-iliac prosthesis. The total number of individuals with a straight infrarenal segment of aorta and a straight

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carotid artery was 229. This sample was used in the regression and statistical analysis of the axial prestretch. However, in two cases there was missing data describing the radius of LCCA. Thus the number of specimens used in the analysis of the radius was 227.

Table 1. Data summary. Indices C and A are used to denote LCCA and AA, respectively. * indicates mode.

	N	Mean Median	SD IQR	Min – Max
Age [years]	229	58.4 63	15.5 20	6 – 88
λ_z^C [-]	229	1.147 1.129	0.1065 0.1431	0.9888 – 1.493
λ_z^A [-]	229	1.080 1.058	0.07927 0.08547	0.9714 – 1.405
r^C [cm]	227	0.2959 0.3023	0.04104 0.04775	0.1751 – 0.4138
ATH^C [-]	229	1.310 1 2*	1.049 2	1 – 4
ATH^A [-]	229	2.917 4 4*	1.356 2	1 – 4

Table 2. Regression equations. $\alpha = 0.05$.

	$y = b_1x + b_0$	$\pm t_{1-\alpha/2}(n-2)S_e\sqrt{(1+1/n+(x-x_m)^2/S^2_{total})}$	b_1 confidence limits	b_0 confidence limits
$y = \lambda_z^C$ $x = Age$	$y = -5.380\cdot10^{-3}x+1.461$	$\pm0.1308\sqrt{(1.004+1.824\cdot10^{-5}(x-58.39)^2)}$	$-5.939\cdot10^{-3}$ $-4.821\cdot10^{-3}$	1.427 1.494
$y = r^C$ $x = Age$	$y = 1.266\cdot10^{-3}x+0.2222$	$\pm7.118\cdot10^{-2}\sqrt{(1.004+1.841\cdot10^{-5}(x-58.27)^2)}$	$9.609\cdot10^{-4}$ $1.572\cdot10^{-3}$	0.2037 0.2406
$y = \lambda_z^A-1$ $x = \lambda_z^C-1$	$y = 0.5255x+3.033\cdot10^{-3}$	$\pm0.1108\sqrt{(1.004+0.3865(x-0.1465)^2)}$	0.4566 0.5944	$-9.440\cdot10^{-3}$ $1.551\cdot10^{-2}$

Table 1 summarizes descriptive statistics of the data sample. Parameters of the linear regression equations are listed in Table 2. Regression equations for $\lambda_z^C = \lambda_z^C(Age)$, $r^C = r^C(Age)$, and $\lambda_z^A = \lambda_z^A(\lambda_z^C)$ are presented here, including the 95% prediction intervals for future observation and confidence intervals for regression parameters. Figures 1, 2, and 3 show predictions given by the regression equations and the observed data. Correlation coefficient R suggests that there is significant dependence between λ_z^C and *Age* ($R = -0.783$, $p < 0.001$, Figure 1), r^C and *Age* ($R = 0.478$, $p < 0.001$, Figure 2), and λ_z^A and λ_z^C ($R = 0.706$, $p < 0.001$, Figure 3). However, in the case of the relationship between λ_z^A and λ_z^C, we should not interpret the results presented in Figure 3 as a direct physical dependence between λ_z^A and λ_z^C, but rather as a consequence of the fact that both λ_z^A and λ_z^C depend on age. To emphasize this fact, points in the figure are colored to indicate the age, unlike Figures 1 and 2 where data is colored to indicate the degree of atherosclerosis.

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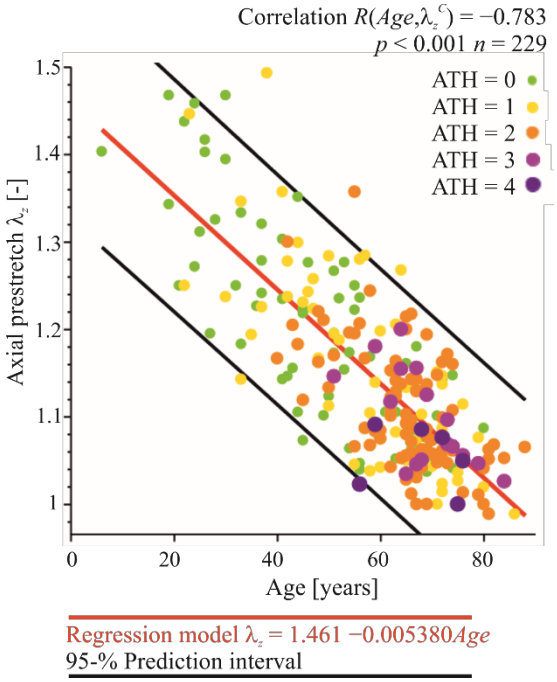

Figure 1. Axial prestretch in LCCA and age of autopsied individuals.

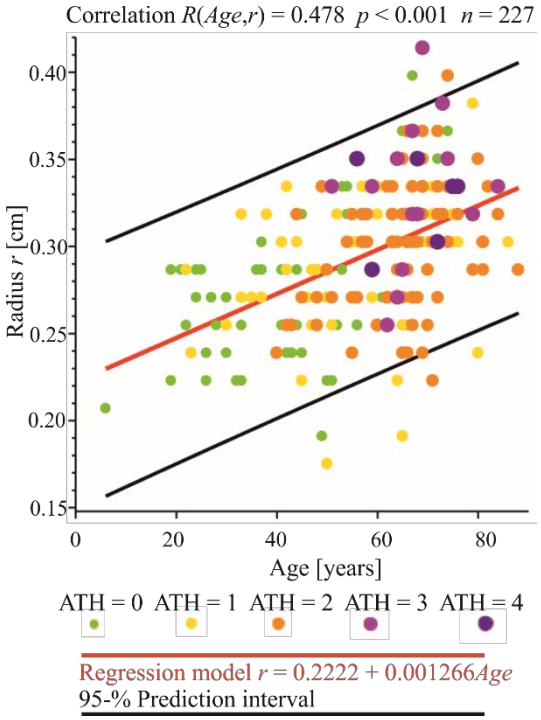

Figure 2. Radius of LCCA and age of autopsied individuals. The radius was computed from a strip cut in the circumferential direction.

The correlation between age and axial prestretch of LCCA did not reach as high a value as for the prestretch of AA. Horný et al. (2014a,b) found $R(ln(\lambda_z^A),ln(Age)) = -0.903$ for the sample of 365 cadavers, but the present sample of LCCA shows $R = -0.783$. In addition, the character of the dependence of the axial prestretch on age differs. The age-related decrease in prestretch in AA is best-fitted with the power law (Horný et al., 2011, 2014a). However, our attempts to fit the data in Figure 1 with an exponential function, a power function and a logarithmic function did not lead to a significant increase in the explained variability. In regard to the dependence between λ_z^A and age, we did not find

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any significant difference in comparison with results presented in Horný et al. (2014a,b). Thus a figure displaying $\lambda_z^A = \lambda_z^A(Age)$ is omitted, but for the sake of completeness we note that $\lambda_z^A = 2.149 \cdot Age^{-0.1718}$, $R(ln(Age), ln(\lambda_z^A)) = -0.851$, $p < 0.001$.

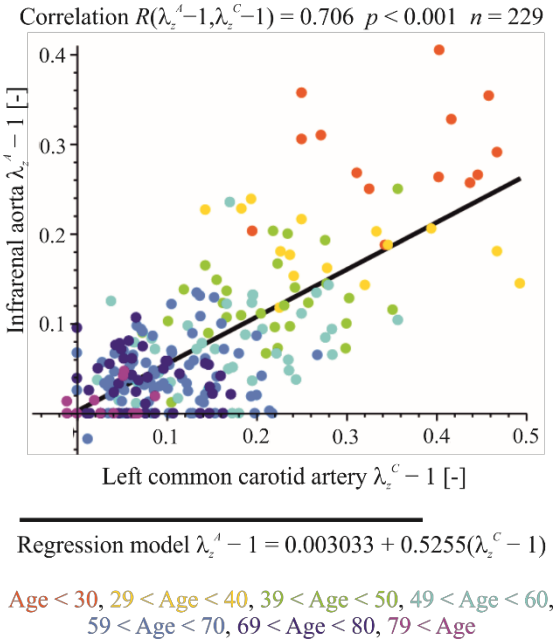

Figure 3. Mutual dependence between the prestretch measured in the infrarenal aorta and in the left common carotid artery.

Figure 3 shows that there are two basic differences between the axial prestretching of LCCA and the axial prestretching of AA. The first difference is that LCCA is prestretched to a greater extent than AA. This is clear from the slope of the regression line; *arctan*(0.5255) ≐ 27.7° with 95% confidence limits *arctan*(0.4566) ≐ 24.5° and *arctan*(0.5944) ≐ 30.7° (Table 1). However, the confidence interval for b_0 suggests that the hypothesis $b_0 = 0$ cannot be rejected at $\alpha = 0.05$. It should be noted that the fact that the axial prestretch of LCCA is greater than the prestretch in AA is not in accordance with the results known from the literature. Learoyd and Taylor (1966) and also Bergel (1961) reported these prestretches to be approximately equal. However, they used a significantly smaller number of samples. Learoyd and Taylor (1966) compared young and old human arteries, but did not disclose the exact number of observations. Instead, they stated that the number was at least five. Bergel (1961), in Table 1, presents mean values suggesting that the retraction of AA ($n = 17$) and LCCA ($n = 29$) is the same, but this result was obtained for a different species (dog), and the age of the animals is not documented.

Axial prestretch in LCCA and AA was also compared by means of paired statistical tests employing the whole data sample as well as data from subintervals where the correlation with age was not significant. A Wilcoxon signed-rank test conducted with the entire data sample showed that there is strong statistical evidence against the hypothesis that $|\lambda_{zi}^C - \lambda_{zi}^A|$ are symmetrically distributed around zero ($p < 0.001$), and the same result was obtained when a comparison was conducted with groups selected from age-independent intervals. See Figure 4 for details. A difference between LCCA and AA was also confirmed by a Kolmogorov-Smirnov test, by means of which the hypothesis that λ_z^A and λ_z^C has the same distribution function was rejected ($p < 0.001$).

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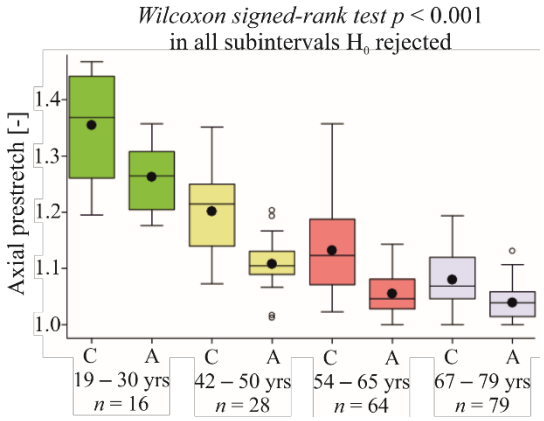

Figure 4. Comparison between axial prestretch in LCCA (columns denoted simply as C) and AA (denoted as A) in subintervals where correlation with age is not significant. Arithmetic means are indicated with black filled circles, whereas black empty circles show outliers.

The second difference is that the AA sample showed 34 (14.9% out of 229) individuals where $\lambda_z = 1$, but in LCCA only 5 (2.2%) such individuals were found. This high relative frequency of $\lambda_z^A = 1$ is the source of a nonlinearity found in $\lambda_z^A = \lambda_z^A(Age)$, see Horný et al. (2011, 2014a). Here we have to note that the measurements were conducted with a millimeter rule, so the resolution is approximately $\Delta\lambda_z = 0.01$. This value is obtained as the ratio of ΔL (= 1 mm) to mean ex situ length L (L = 79.1 mm in AA, and L = 88.7 mm for LCCA in the present sample; in the case of AA, further details can be found in Horný et al. (2012a). When we merge data given by $\lambda_z = 1.0 \pm 0.01$, it is found that the interval [0.99,1.01] contains 45 samples for AA, and 11 samples for LCCA. This still suggests that there is a difference between AA and LCCA, which can be expressed phenomenologically as: It is more likely that in ageing AA will approach the non-prestretched state than that this state will be reached by LCCA.

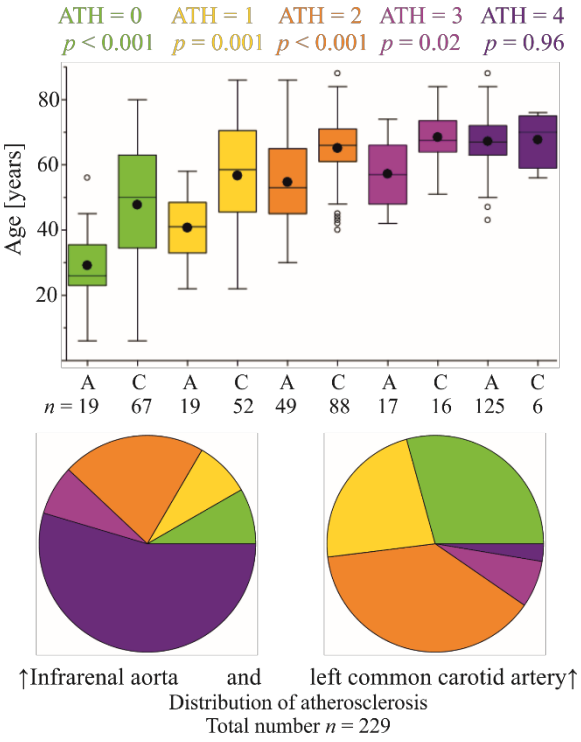

Figure 5. Comparison of distributions of atherosclerotic changes in AA and in LCCA as such (lower panels), and the age distribution in the groups of the same ATH (upper panel). In the upper panel, black filled circles indicate the arithmetic mean, and black empty circles are outliers. p values were obtained in a Kolmogorov-Smirnov test.

A similar difference is also revealed when the data is compared by means of the age distribution in groups with the same ATH. This is depicted in Figure 5. A greater number of individuals showed the

worst case of ATH in AA (ATH = 4 for 125 individuals) than in LCCA (ATH = 4 for 6 individuals). Moreover, as Figure 5 shows (upper panel), the Kolmogorov-Smirnov test confirmed that the age of the individuals is distributed in a different manner in AA and in LCCA, when the same degree of ATH is considered. This suggests that atherosclerotic changes occur earlier in AA than in LCCA.

From Figure 5, one might deduce that ATH plays a significant role in the age-related decrease of the prestretch. However, Horný et al. (2014a) showed that specifically in abdominal aorta there is no statistical evidence that supports this hypothesis unambiguously. They conducted an analysis of the variance of the prestretch divided into groups of the same ATH in age-adjusted data samples. Their results were not consistent. Two ageing intervals showed significant differences of the prestretch between the ATH groups. However, in the 5 remaining ageing intervals, statistical evidences against null hypothesis were not found. In other words, ATH was not capable to sort data to groups with significantly different mean values. Here we employed the Kruskal-Wallis test, non-parametric equivalent of ANOVA, to test this hypothesis in AA and LCCA. H_0 states that medians of the axial prestretch sorted into groups of the same ATH are equal. This was again conducted with data divided into intervals where the correlation with age is not significant. The results are presented in Figure 6.

The hypothesis that the medians of the prestretch are equal was not rejected in any subinterval in AA or in LCCA. In Figure 6, numerals in brackets indicate compared groups. The number of observations in each group is written on the horizontal axis, where the correlation with age and the p value of the hypothesis $R = 0$ are also shown. The groups that were compared consisted of at least four measurements. It is recommended to conduct a Kruskal-Wallis test with at least five observations in each group. We should therefore note that the results obtained in [74;88] years for LCCA, and in [23;33], [34;47], and [48;57] years for AA should not be considered as conclusive. However, for LCCA in the interval [74;88], the groups with ATH = 1 and 2 provide a sufficient number of observations to be compared by means of the Mann-Whitney test for two independent samples. The same applies for AA and intervals [34;47], [48;57] with groups of ATH = 1 and 2, and 2 and 4, respectively. Nevertheless, the Mann-Whitney test did not reveal significant differences in any of these groups. In this situation, we conclude that the data presented here indicates that atherosclerosis should not be considered as a factor significantly affecting the age-related decrease in axial prestretch.

Mechanical experiments conducted with genetically modified animals (Carta et al., 2009; Wagenseil et al., 2005) and with elastase treated arteries (Dobrin et al., 1984, 1990) have shown that elastic membranes forming the medial layer of the artery wall are crucial for bearing the prestretch and axial loading. Thus one can expect that if LCCA and AA differ with reference to the extent of the prestretch, they should also differ in the number of elastic membranes and in the thickness of the media. To evaluate this hypothesis, transmission light microscopy digital images of histological sections stained with Orcein obtained from the AA and LCCA of 8 donors were analyzed. In each image, the thickness of the media and a number of elastic membranes were determined three times and averaged. Data samples of 8 averages for LCCA and AA were compared by means of a paired Wilcoxon signed-rank test. It was found that AA media is significantly thicker than LCCA media (medians, 482 vs. 343 μm, $p = 0.04$) and AA consist of a greater number of elastic membranes than LCCA, although this difference did not reach statistical significance (medians, 31.5 vs. 26.5, $p = 0.07$ for a one-tailed test).

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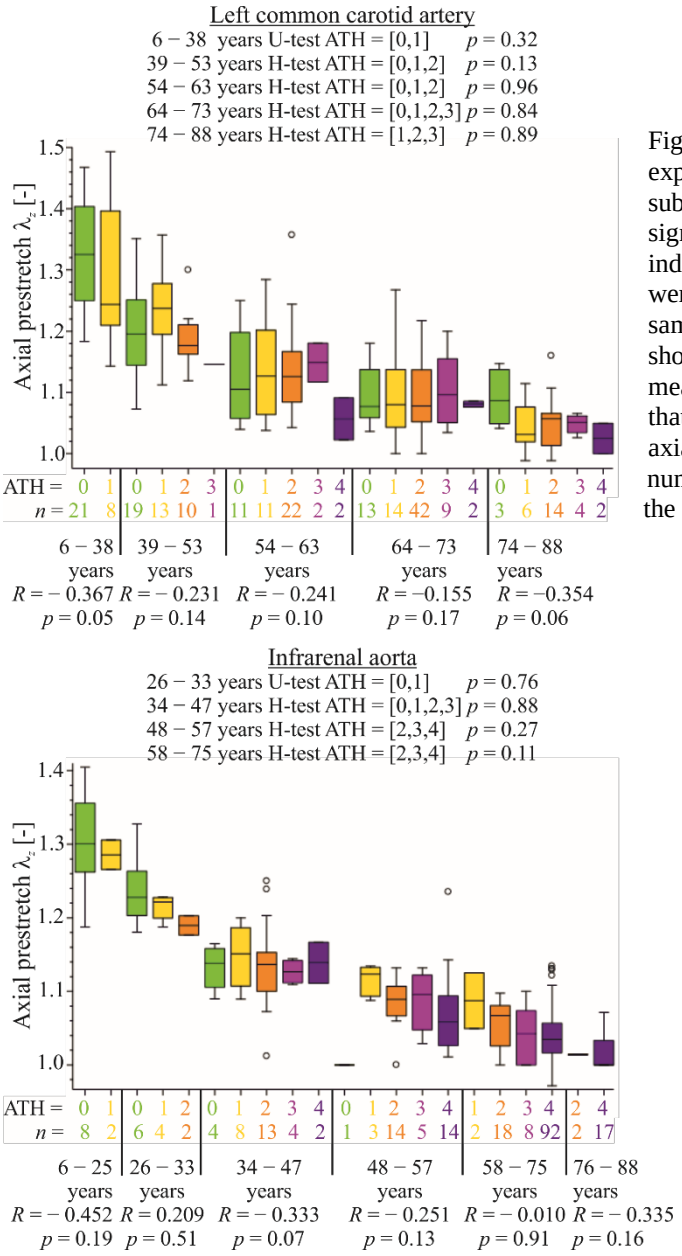

Figure 6. Atherosclerosis is not a factor capable of explaining the variance in the axial prestretch in subintervals where the correlation with age is not significant. A U-test (Mann-Whitney for two independent samples) and an H-test (Kruskal-Wallis) were used to test the hypothesis that subgroups of the same ATH have the same mean value. All tests showed that the hypothesis cannot be rejected. This means that ATH does not arrange arteries into groups that would differ significantly with reference to the axial prestretch. Compared groups are indicated by numerals in brackets. The colors of the boxes indicate the degree of ATH.

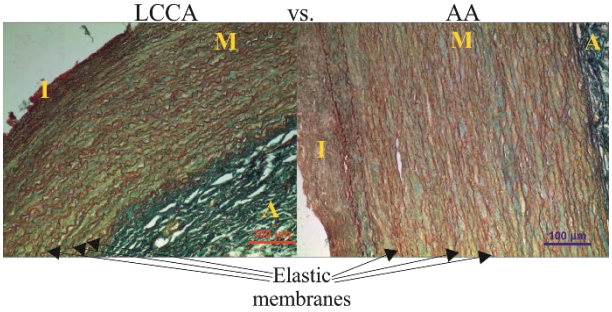

Figure 7. Histological sections stained with Orcein. I, M, and A indicate the tunica intima, tunica media, and tunica adventitia of the wall. Brown-red color corresponds to elastin in elastic membranes, and green-blue corresponds to collagen. Scale bar 100 μm.

It is worth noting that we found 17 individuals with buckled LCCA, 5 individuals with an aneurysm in AA, and 7 with buckled AA. In these individuals, the in situ and ex situ lengths were also measured. However, they were not included in the analysis of the axial prestretch because it is not clear how the

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prestretch is distributed along the length of these segments. It is reasonable to expect uniform distribution of the deformation along a sample with a uniform geometry. However, the spatial distribution of a deformation field in complex geometries has to be measured by more advanced methods (e.g. digital image correlation, Horný et al. 2012b), which are not available in our autopsy room. Nevertheless, we feel that this data could be interesting for those who conduct computational analyses of strain and stress fields in aneurysms and tortuous arteries.

Table 3. Data for tortuous and aneurysmatic arteries. A strip used to measure the circumference of AA was cut from a non-aneurysmatic part of AA. L, and l were obtained as lengths of end-to-end line segments.

	Age [years]	l in situ [mm]	L ex situ [mm]	$\lambda_z = l/L$ [-]	LCCA r [cm]
Tortuous LCCA	64	77	73	1.055	0.350
	58	91	79	1.152	0.286
	65	90	75	1.200	0.255
	61	75	65	1.154	0.271
	81	85	85	1.000	0.334
	81	102	103	0.990	0.302
	70	102	89	1.146	0.286
	75	110	113	0.973	0.302
	81	84	73	1.151	0.286
	80	75	77	0.974	0.366
	89	90	80	1.125	0.318
	69	95	89	1.067	0.239
	89	116	108	1.074	0.286
	71	91	87	1.046	0.302
	81	130	110	1.182	0.334
	88	103	92	1.120	0.318
	71	85	83	1.024	0.350
Mean	74.9	94.2	87.1	1.084	0.305
SD	9.8	14.9	14.1	0.075	0.035
Tortuous AA					
	64	102	99	1.030	0.350
	67	89	82	1.085	0.366
	75	98	97	1.010	0.350
	70	98	98	1.000	0.398
	64	76	76	1.000	0.302
	70	115	113	1.018	0.271
	88	78	71	1.099	0.318
Mean	71.1	93.7	90.9	1.035	0.336
SD	8.4	13.8	14.9	0.041	0.043
Aneurysm of AA					
	64	98.0	80.0	1.225	0.318
	68	93.0	90.0	1.033	0.318
	41	94.0	84.0	1.119	0.302
	73	61.0	58.0	1.052	0.382
	66	80.0	75.0	1.067	0.207
Mean	62.4	85.2	77.4	1.099	0.306
SD	12.4	15.1	12.2	0.077	0.063

Data is presented in Table 3. Here end-to-end distance refers to the length of a straight line from one end to the other. It is interesting that the data suggests that segments of aneurysmatic aortas could be axially prestretched. However, we have to mention that it is not possible using our method to say to what

extent the aneurysm is prestretched. We can only say that when an aneurysm that does not extend through the entire length of AA occurs in AA, measurements of the in situ and ex situ length imply that there is some average axial prestretch defined for the total length of the sample.

Finally, to be sure that our results are not biased by post mortal changes a correlation between the prestretch and the length of the post mortem interval (PMI) was investigated. PMI is the time interval between the death and the autopsy during which axial prestretch is measured. Descriptive statistics of our sample are as follows: mean/median 45.6/39, SD/IQR 30.0/40, lower/upper quartile 23/63, min/max 4/168 (all in hours). The correlation coefficients are $R(\mathrm{PMI},\lambda_z^C) = -0.006$ ($p = 0.92$) and $R(\mathrm{PMI},\lambda_z^A) = -0.062$ ($p = 0.63$). Correlation coefficients were also computed for age-adjusted intervals. None of these values reached statistical significance. This is consistent with our previous results, published in Horný et al. (2014a), where a detailed analysis of the effect of PMI in 365 autopsied aortas can be found. We attribute this relative independence of the prestretch on PMI to the fact that elastin, the main component of elastic membranes, is a highly stable protein resistant to autolytic processes in the order of magnitude of several days. Interestingly, a recent study by Hartung et al. (2015) has shown that arteries excised from corpses in a high grade of putrefaction also exhibit significant axial prestretch.

4. Conclusions

In conclusion, measurements of the in situ and ex situ lengths of the human left common carotid artery have shown that this artery is significantly axially prestretched, and that the specific extent of the prestretch depends considerably on the age of the individual. The relationship between age and prestretch was found to be well-fitted by a decreasing linear function. The comparison with the infrarenal aorta showed that the prestretch is greater in the carotid artery than in the abdominal aorta, and that these sites also differ in the manner in which they are afflicted by atherosclerosis in ageing. However, a comparison of the subgroups arranged with reference to the degree of atherosclerotic disease has shown that atherosclerosis cannot explain the variability remaining after age adjustment. This is not so surprising, when one considers that atherosclerosis predominantly affects the intimal layer of the artery wall. It seems that the ageing-induced decrease in prestretch is a consequence of a damage cumulated in elastic membranes due to concurrent action of the elastocalcinosis, elastolytic activity of cells, and fatigue failure rather than due to atherosclerosis (Horný et al., 2014a; Fritze et al., 2012; Greenwald, 2007; Persy and D'Haese, 2009; Atkinson, 2008).

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Conflict of interest

The authors declare that they have no conflict of interest.

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