Systolic axial artery length reduction: an overlooked phenomenon in vivo

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Tozzi, P., D. Hayoz, C. Oedman, I. Mallabiabarrena, and L. K. Von Segesser. Systolic axial artery length reduction: an overlooked phenomenon in vivo. Am J Physiol Heart Circ Physiol 280: H2300-H2305, 2001.-To demonstrate axial artery motion during the cardiac cycle, the common carotid arteries (CCA) of 10 pigs were exposed and equipped with piezoelectric crystals sutured onto the artery as axial position detectors. An echo-tracking system was used to simultaneously measure the CCA diameter. For each animal, data for pressure, length, and diameter were collected at a frequency of 457 Hz. At a mean pulse pressure of 33 ± 8 mmHg, the mean systolodiastolic length difference was 0.3 \pm 0.01 mm for a mean arterial segment of 11.35 ± 1.25 mm. Systolic and diastolic diameters were 4.1 \pm 0.3 and 3.9 \pm 0.2 mm, respectively. The examined CCA segment displayed a mean axial systolic shortening of 2.7%. This study clearly demonstrates, for the first time, that the length of a segment of the CCA changes during the cardiac cycle and that this movement is inversely correlated with pulse pressure. It is also apparent that the segmental axial strain is significantly smaller than the diameter variation during the cardiac cycle and that the impact of the axial strain for compliance computation should be further evaluated.

compliance; vascular ultrasound; sonography; arterial wall

THE MECHANICAL PROPERTIES of the arterial wall have been extensively described for decades by Lawton (8), Patel et al. (10), and Dobrin (4), to name a few authors. Measurements obtained with electromechanical gauges applied to exposed vessels or with roentgenographic and ultrasonic methods have shown that arterial dimension changes occur mostly in radial and circumferential directions. Therefore, it has been assumed that artery length changes were negligible over a wide range of pressure levels (5). Considering only the two-dimensional geometry changes may have consequences on the computation of arterial compliance due to the anisotropy of the mechanical properties of arteries. In clinical practice, cross-sectional compliance is commonly used, assuming that there is no significant axial vessel movement due to pulse pressure; therefore, vessel volume changes are mostly due to changes in vessel diameter.

By characterizing the axial arterial movement, we can precisely define the physiological arterial response to different hemodynamic environments and estimate its contribution for the assessment of arterial compliance. Furthermore, axial movement can also play an important role in the understanding of the mechanical properties of vascular anastomoses and in the prediction of their long-term outcome.

MATERIALS AND METHODS

Instrumentation. Axial length measurements were obtained with small piezoelectric crystals that transmit and receive short ultrasonic pulses. The crystals were sutured onto the common carotid arteries (CCA) 10–15 mm apart. Under electric stimulation, one crystal produces a sound wave that is detected by a second crystal, inducing an electrical response. A simple calculation (distance = velocity × time) yields the distance between the crystals. The sound velocity in the carotid artery wall of a pig at 38°C is 1.18 mm/µs (12). The system setting used had the following characteristics: sampling rate, 457 Hz; transmit pulse, 357 m/s; and sampling time, 5 s. This technique has been extensively validated previously (6, 12).

Arterial pressure was obtained using a high-fidelity pressure probe (model MPC-500, Millar Mikro-Tip) with a pressure range of -50 to 300 mmHg and a sensitivity of 5 μ V·V⁻¹·mmHg⁻¹.

A high-resolution echo-tracking system (NIUS 02) was used to measure the arterial diameter between crystals. Figure 1 illustrates the method of the A-mode echo tracking for arterial diameter determination. Extensive description of the device and validation of the technique have been reported previously (7).

The experiment was performed on 10 domestic pigs 45–65 kg in weight. All animals have received human care in compliance with the *Guide for the Care and Use of Laboratory Animals* published by the National Institutes of Health (NIH publication 85-23, Revised 1985). For each animal, we calculated blood viscosity with a Wells-Brookfield viscometer and hematocrit.

Surgical technique. Pigs were given 15 mg/kg ketamine, 0.5 mg azaperon, and 2 mg atropine. General anesthesia was induced with thiopental sodium (solution of 25 mg/ml, 15–25 ml/h) or 1.5% fluothane. Electrocardiograms (ECG), O_2 saturation, and Pco_2 were continuously monitored. Pigs were laid on their back with a neck extension of 160°. Both carotid

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Fig. 1. Schematic illustration of the arterial diameter determination by an A-mode echo-tracking system (NIUS 2). The cursors automatically focus on the echogenic interfaces delineating the wall lumen. IMT, intima-media thickness.

arteries were exposed. The adventitia was left in place, and we carefully avoided isolating the carotid artery from the jugular vein and vagus nerve. The pressure probe was placed in one carotid artery while up to six piezoelectric crystals were sutured with a polypropylene 6-0 suture on the wall of the other carotid artery (Fig. 2). The distance between piezoelectric crystals was between 9 and 15 mm. To generate detailed cross-sectional artery profiles, we used a high resolution echo-tracking system (NIUS 02), as shown in Fig. 2. Finally, the pressure probe, ECG, crystals, and NIUS 02 were connected to our measurement system. The artery was irrigated with 0.9% NaCl solution at 37° every 10 min to prevent desiccation and to control temperature. During data acquisition, contact with the animal was prohibited. Data were also acquired in two animals during apnea to assess the influence of ventilation.

Data collection. After 15 min of stabilization, data collection was carried out for a period of 5 s without interruption four times per minute for no less than 2 h for each animal. Segmental CCA length, diameter, and blood pressure were captured at 457 Hz. To avoid blood mass and pulse wave interference, the transmitter and receiver functions of the piezoelectric crystals were switched alternatively. To avoid paradoxical effect due to possible axial curvature of the vessel, in three animals, the axial length on three different sites (a, b, and c) of the vessel circumference were measured and compared as a percentage of axial deformation (ΔL) , as shown in Fig. 3. In three animals, data acquisition was carried out after wound closure.

RESULTS

We collected, from each animal, a total of 1.2×10^6 simultaneous data for pressure, diameter, and distance between crystals. The systolic piezoelectric distance was in the range of 8.59-14.9 mm (mean $11.05 \pm$ 1.06 mm); the diastolic piezoelectric distance was between 8.81 and 15.07 mm (mean 11.35 ± 1.25 mm). The systolodiastolic piezoelectric displacement ranged between 0.11 and 0.65 mm (mean 0.30 ± 0.01 mm). The percentage of CCA axial deformation (ΔL) comprised between 1 and 5% of the considered length (mean 2.7%). No differences in ΔL were noted when switching receiving and transmitting piezoelectric crystals. The mean carotid systolic diameter was 4.1 \pm 0.3 mm, and the mean diastolic diameter was 3.9 ± 0.2 mm (mean 4.0 \pm 0.2 mm). The mean variation in vessel diameter during the cardiac cycle was 0.2 ± 0.02 mm



Fig. 2. The carotid arteries of a pig were exposed. Three piezoelectric crystals were sutured onto the right carotid artery, and the echo-tracking system (NIUS 02) was placed on the carotid artery between the 2 crystals to calculate carotid diameter and cross-sectional compliance. A high-fidelity pressure probe (yellow catheter on the *right top* part of the incision) was inserted into the left carotid artery.



Fig. 3. Drawing of a carotid artery. Three pairs of crystals (solid cubes) measuring carotid axial length at three different sites of the vessel circumference (*a*, *b*, and *c*) are shown. The systolodiastolic vessel deformation (ΔL) in each considered site ($a\Delta L$, $b\Delta L$, and $c\Delta L$) were compared with those measured in the absence of bending of the vessel.

(4.8% of mean diameter). Mean diastolic pressure was 62 ± 13.5 mmHg, mean systolic pressure was 95 ± 15.8 mmHg, pulse pressure was between 20 and 40 mmHg (mean 33 ± 8 mmHg), and mean heart rate was 88 ± 18 beats/min. The hematocrit was between 45 and 49%, and the blood viscosity was 0.004 Pas.

The results are summarized in Table 1.

In three animals, we measured the possible curvature effect (ΔL) in three different sites of the vessel circumference, as shown in Fig. 3. The results are reported in Table 2.

Figure 4 shows the simultaneous axial, radial, and pressure measurements in one animal as a function of time.

In Fig. 5A, the inverse correlation between the crystal distance and blood pressure is shown. Increasing blood pressure causes vessel length reduction, whereas in the cross-sectional direction it increases the CCA diameter (Fig. 5B). During systole, vessel diameter increases up to 4%, whereas the examined vessel length decreases up to 2%.

Table 1. Characteristics of arterialgeometric parameters

			Distance
	Pressure	Diameter	Crystals
Systole	95 ± 15.8	4.1 ± 0.3	11.05 ± 1.06
Diastole	62 ± 13.5	3.9 ± 0.2	11.35 ± 1.25
Systolodiastolic changes			
(means \pm SD)	33 ± 8	$0.2 \pm 0.02^{*}$	$0.30\pm0.01^*$
Relative variation	34.7%	4.8%	2.7%

Values are means \pm SD; n = 10 pigs. Arterial blood pressure (in mmHg), carotid diameter (in mm), and axial displacement (distance, in mm) of piezoelectric crystals fixed on the carotid artery in 10 pigs recorded simultaneously during the cardiac cycle. *P < 0.001.

Table 2. Distance between crystals at three sitesof vessel circumference

ΔL	Pig 1	Pig 2	Pig 3
Site a Site b Site c	$\begin{array}{c} 2.12\pm 0.2\\ 2.12\pm 0.2\\ 2.13\pm 0.1\end{array}$	$3.42 \pm 0.2 \\ 3.42 \pm 0.1 \\ 3.42 \pm 0.2$	$\begin{array}{c} 2.14 \pm 0.3 \\ 2.13 \pm 0.2 \\ 2.14 \pm 0.1 \end{array}$

Values are means \pm SD. Measurements of axial length at three different sites (*a*, *b*, and *c*) of the vessel circumference. Axial deformation (ΔL , in %) is given for each animal.

DISCUSSION

This study clearly demonstrates a substantial axial strain in exposed carotid segments during the cardiac cycle. This systolic axial length reduction appears to result mainly from the elastic recoil of the arterial wall constituents to the cross-sectional wall expansion. Therefore, the notion that artery length changes during the cardiac cycle is negligible deserves reappraisal. Furthermore, it is clear from the data provided in the study that the segmental axial strain is definitely smaller than the diameter variation during the cardiac cycle and that the impact of this axial strain for arterial compliance calculation should be further evaluated.

The limitations of the study reside in part in the fact that the data presented are valid only for the segment of the CCA examined, which corresponds to the distance between the two crystals. Considering this arterial segment, one can deduce by simple mathematical computation that the volumetric changes of the segment are smaller than usually thought because the volume gained in the cross-sectional direction is reduced in the longitudinal axis. Therefore, the principal determinant of the elastic property of the vessel wall appears to reside in its cross-sectional variation and is only marginally affected by axial deformation.

The surgical procedure may also have modified the genuine elastic properties of the vessel wall. However, careful attention was paid not to severe the adventitia at the level of the sutures. Assessment of the arterial diameter by echo tracking before the suturing of the crystals did not change from that observed after crystal fixation, supporting the fact that significant vascular wall reaction was absent.

Another limitation of the experiment is that a single element of the arterial impedance was considered (compliance); no specific information on resistance and wave reflection is provided. However, the alternate switching of the transmitter and receiver functions addresses to some extent the effect of wave reflections on this segment of the CCA, as discussed below.

Previous studies have reported arterial length changes during the cardiac cycle; however, no clear relationship with pulse pressure was provided. Lawton (8) reported a 1% increase in length of the thoracic aorta and a 1% decrease in length of the abdominal aorta during cardiac cycles without identifying any relationship with pulse pressure. Patel and Fry (10) reported that the ascending aorta and pulmonary ar-



Fig. 4. Simultaneous recordings of diameter, length, and intraarterial blood pressure changes over time in one animal.

teries could modify their length up to 11%, but this axial movement resulted from gross motion of the heart. In vivo, there are at least two structures that provide arterial fixation to the surrounding structures: the perivascular connective tissues and the arterial side branches. The fixation of arterial side branches and the presence of periadventitial connective tissues cause tethering. Patel and Fry (11) suggested that perivascular traction is distributed over the length of the arterial tree. It is minimal at the aortic root and increases with the distance from the aortic valve.

Moreover, arteries are extended in the longitudinal direction. This is reflected by the observation that excising arteries causes them to retract. Dobrin (5) reported that the interaction between pressure and traction stresses keeps the length of artery nearly constant. All these statements are based on experimental data obtained with electromechanical gauges applied to exposed vessels or with roentgenographic and ultrasonic methods (4, 5).

Recent developments in investigational tools gave us the opportunity to evaluate more precisely the longitudinal properties of arteries. Piezoelectric crystals have an axial resolution of 15 μ m, which is much higher than that of any other equivalent tool (6). Longitudinal displacement of piezoelectric crystals placed 9–15 mm apart is between 0.11 and 0.65 mm. Because the distance between the two piezoelectric crystals could not be kept constant due to technical constraints, the results are expressed as relative changes of the distance between crystals: the displacement varies from 1.14 to 5.8%. The behavior of the vessel did not change significantly during the whole experiment. To exclude interference of pulse waves and blood mass (viscosity and hematocrit) on the ultrasonic waves during measurements, we switched the receiver and transmitter functions of the piezoelectric crystals in all feasible combinations. The results obtained under these conditions did not reveal any significant difference in ΔL . Therefore, blood mass and pulse wave reflections can be considered to have no significant influence on the measurements of this segment of CCA.

Our conclusions hold true provided that the examined arterial segment is straight. Indeed, if the vessel shows a significant curvature, then the slugging of the convex part over the concave part of the vessel wall should occur. We tested this possibility by measuring axial length at three different sites of the vessel circumference and comparing their axial deformation $(a\Delta L \text{ versus } b\Delta L, a\Delta L \text{ versus } c\Delta L$, and $b\Delta L \text{ versus}$ $c\Delta L$), as shown in Fig. 3 and Table 2. No differences were observed between the sites, implying that the examined vessel segment was straight.



Fig. 5. A: pressure-length changes; B: pressure-carotid diameter changes during one cardiac cycle in one animal. Arrows describe the temporal relationship between blood pressure and dimension changes.

Surgical exposure of the vessel can affect wall properties. Arndt and Kober (1) used roentgenographic methods to measure arteries in animals and humans. They reported diameter oscillations of $\sim 15\%$ and noted that this was larger than values reported by other investigators for exposed vessels. They suggested that surgical exposure of vessels might have increased stiffness. However, Busse et al. (3) compared pulse-wave velocity measurements and found that exposure did not alter wall stiffness. In our surgical procedure the adventitia were left in place, and we carefully avoided isolating the carotid artery from the jugular vein and vagus nerve. In three animals, data acquisition was carried out after wound closure to reproduce the physiological environment as accurately as possible. We used thiopental sodium in six animals and fluothane in four animals to minimize the vasoactive effect of the anesthetic drug. No differences were noted between the two groups.

Axial movement is inversely correlated to pulse pressure (Fig. 5A). This was a characteristic feature observed in all animals in the study. The slope in the diastolic pressure range was less pronounced than the systolic slope, which most likely results from the hysteresis due to viscoelastic properties of the material in the axial direction. Similar observations have been reported in the radial direction (4). Whether integrity of the endothelial function plays a role in the behavior of the axial wall motion and more specifically on the importance of this hysteresis remains to be determined (see Ref. 2). It is well known that the carotid artery diameter is directly correlated to blood pressure (1, 3), and this was also confirmed in our study. Figure 5*B* shows the relationship between carotid diameter and arterial pressure.

In general, it is assumed that axial strain can be neglected compared with circumferential strain. Our findings confirm that the axial strain is indeed lower than the radial strain. On the average, an axial length modification of 2.7% was found for a diameter variation of 4.8%. It is also well known that the mechanical properties of the arterial are anisotropic, although for deformations that occur in vivo, the elasticity of arteries can be considered isotropic (13). However, the canine carotid artery and femoral arteries are stiffer in the circumferential than in the longitudinal direction, whereas the reverse is true for canine and bovine aorta. This may be due to the differences in wall architecture and load bearing of the wall constituents in each direction. Thus arteries in vivo undergo unequal deformations in each direction, and this differentially stretches and stiffens all constituents.

Our data suggest that when arterial pressure increases, a segment of the carotid artery dilates and shortens. This phenomenon, measured locally in a segment of CCA, should not be extended to other parts of the vascular tree before it has been tested elsewhere. We suggest qualifying this arterial segmental motion as axial systolic shortening. It represents the capacity of the vessel wall to passively adapt to axial stress, and it should clearly be differentiated from an active contraction. These findings contrast with the description of the arterial wall movement observed by Lichtenstein et al. (9). In an elegant experimental study performed in vitro on rat CCA, the authors reported an increase in carotid length in response to pressure. This axial length increase was more important in normotensive than in spontaneously hypertensive rats. However, the measurements were determined by video microscopy and computer-assisted image analysis. Therefore, due to the major differences in experimental conditions, the results cannot be appropriately compared.

The role of the axial movement in the determination of volumetric compliance measurement is not clear. Arterial compliance (C) is expressed as the ratio between vessel volume variation (ΔV) during the cardiac cycle and pulse pressure (ΔP) as follows: $C = \Delta V/\Delta P$. In clinical practice, a high-resolution echo-tracking system allows us to precisely measure local cross-sectional compliance considering only vessel diameter variations. Instead of measuring volumetric changes (Δm^3), only cross-sectional area (Δm^2) is measured. Crosssectional compliance (CC) is thus defined as the ratio between variations in arterial cross-sectional area (ΔA) and blood pressure (ΔP), i.e., CC = $\Delta A/\Delta P$ [CC is expressed in $\mu m^2/mmHg$ or m^2/kPa (5)].

According to our data, it would not appear appropriate to neglect the axial movement in the computation of segmental arterial compliance because it overestimates the volumetric elastic properties of the vessel. If similar axial movement occurs in the human carotid artery, then reappraisal of local arterial compliance measurement would appear necessary. Volumetric compliance assessment based on a method taking into account the postulate of mass continuity may represent an interesting option. It is based on measuring the ratio of a blood flow gradient through an arterial segment $(Q_{in} - Q_{out})$, where Q_{in} and Q_{out} are the blood flows into and out of the arterial segment) to the derivative of blood pressure over a given time. However, for practical purposes, cross-sectional distensibility of the vessel provides a valuable information on the elasticity of the vessel wall, and, when the distensibility is related to wall thickness, the elastic modulus of wall material can be assessed.

In conclusion, we provide new in vivo data on the mechanical properties of longitudinal segments of conduit arteries. With the use of high-resolution investigational tools, we show that the axial deformation of a 1-cm-long segment of pig CCA is consequential, although twice smaller than the diameter changes observed during the cardiac cycle. We also demonstrate that, during systole, the vessel shortens and dilates. We suggest qualifying this arterial motion as axial systolic shortening because active contraction appears to not be required. Finally, the impact of this axial strain in compliance calculation should be further evaluated.

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