Arterial properties of the carotid and femoral artery in endurance-trained and paraplegic subjects

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As shown in animal experiments, changes in arterial pressure and blood flow initiate structural and functional arterial adaptations (15). The latter is associated with alterations in local wall shear stress, which has been shown to be one primary stimulus for vascular adaptations. Thus a chronic increase in arterial blood flow volume leads to an outward vascular remodeling (20), whereas a decrease causes an inward remodeling of the arterial wall. According to the minimum-cost theory, arterial inward and outward remodeling result to keep the wall shear stress constant (14).

Chronically increased or decreased physical activity in humans leads to changes in arterial blood flow volume, mainly in arteries supplying the working or inactive musculature, respectively. Conducting musculature arteries like the common femoral artery (CFA) have seldom been the object of studies, although they are important for the propagation of blood flow (23). In athletes, a larger luminal size of the CFA (11), but not a higher elasticity, has been reported, whereas in the immobilized upper limb compared with the mobile limb a parallel reduction in elasticity and luminal size of the radial artery has been shown (6). However, so far, no study in humans has assessed the local shear rate in relation to or together with arterial structure and elasticity, although local shear rate is one primary determinant of these parameters. This comprehensive view seems to be of particular interest because it is still not known whether enlarged arteries in highly endurance-trained subjects still maintain a constant wall shear rate independent of the vessel diameter. In addition, nothing so far is known about the shear rate in hypotrophied human arteries. Thus we chose physically inactive paraplegic subjects as a model for long-term physical inactivity and known marked smaller luminal size of the CFA (10, 12). However, the paraplegic model may, at least in part, express a nonphysiological regulation of the vessel diameter due to the disturbed vascular innervation and neurohumoral changes, and it cannot be used without restrictions to explain the physiological adaptations of a reduced blood flow vol-
volume caused by immobilization or an extremely sedentary lifestyle.

The changes in common carotid artery (CCA) blood flow during physical exercise represent only about a 30–40% increase compared with the values recorded during resting conditions (8). Thus the changes in local blood flow during exercise may not be high enough to result in structural changes of the CCA. On the contrary, it is conceivable that a higher CCA elasticity due to increased basal NO production (13) or a reduced resting heart rate (25) may occur in physically active subjects. Nothing so far is known about the structure of, function of, or blood flow in the CCA of extremely inactive subjects such as paraplegic subjects. If any of the effects of different levels of physical activity are visible on the resting arterial properties of the CCA, this should probably be observed in highly endurance-trained athletes and paraplegic subjects, with strongly increased or decreased physical activity over several years, respectively. Abergel et al. (1) found a higher intima-media thickness (IMT) of the CCA in professional road cyclists. However, an increased IMT of the CCA is known to be associated with hypertension (27) or reduced wall shear stress (7). In contrast, endurance training is not known to be associated with manifest hypertension, and, during physical exercise, blood flow volume and consequently wall shear stress are increased in the CCA and are unlikely to increase IMT. The combined assessment of the arterial properties of the CCA in subjects in whom a wide spectrum of physical activity is covered might add information to or help to clarify some of the above-named discrepancies.

Thus in this study, we set out to examine the local properties of the CFA, a conducting muscular-type artery directly involved in exercise-induced changes in local blood flow volume, and the CCA, an elastic-type artery with minor changes in arterial blood flow during exercise or inactivity, by means of noninvasive ultrasound with the subjects at rest. This was done to evaluate the relationship of the local arterial wall thickness and diameter, compliance, and local wall shear rate of these different types of arteries to long-term extremely increased or chronically decreased physical activity in humans.

METHODS

A total of 51 male subjects were included in the study. The 21 highly endurance-trained athletes, consisting of 14 cyclists, 4 middle- or long-distance runners, and 3 triathletes, were examined in the sports medicine clinic. All of the athletes had competed in international competitions and participated in systemic endurance training for at least 14 yr. The peak oxygen consumption, the decisive criterion for endurance performance ability, was determined by different ergonomic methods (treadmill and bicycle ergometry) and was >65 ml·kg⁻¹·min⁻¹ in all the athletes. All the athletes had been training for over 30 h/wk. The 10 paraplegic subjects were examined in the Swiss Paraplegic Center (Nottwil, Switzerland). The average amount of time since the accident causing paralysis was 11.4 yr. The lesion level was below T₄ in all paraplegic subjects. The average weekly activity did not exceed 1 h of vigorous or endurance-type wheelchair exercise. The control group consisted of 20 physically inactive students who were doing no more than 30 min of vigorous or endurance-type physical activity per week. Their peak oxygen uptake was determined by bicycle ergometry (beginning at 50 W, with an increment of 50 W every 3 min until subjective exhaustion). On average, they achieved a peak oxygen uptake of 42.3 ± 7.1 ml·kg⁻¹·min⁻¹, which was thus within the normal range of untrained subjects of that age.

No signs of cardiovascular disease were apparent in any of the groups after the interview, echocardiography, or electrocardiogram (ECG). In addition, plaques, as a sign of atherosclerotic vessel changes in the carotid arteries, were ruled out with transcutaneous ultrasound.

All participants were informed about the purpose of the study before the examination and gave their written consent. Before the examination of the paraplegic subjects, the protocol was approved by the Institutional Review Board of the Swiss Paraplegic Center. The examination of the remaining subjects was not formally approved by the Ethics Committee of the Freiburg University hospital, but it complied completely with the principles of the Declarations of Helsinki.

Ultrasound examination. The ultrasound examination was performed with a high-resolution ultrasound scanner (model SSA-380A, Toshiba) with a digital beam former and a linear 10 MHz-transducer. Both examinations, in Freiburg and Nottwil, were performed with the same ultrasound machine and by the same investigator. All measurements were done between 9:00 and 12:00 in the morning. All subjects were explicitly required not to participate in any exercise or other form of exertion on the day of and the day before the examination. All subjects emptied their bladders before the examination to avoid a possible variance of the vegetative tone and its effects on the functional vessel properties (35).

For the examination of the right CCA, the subject’s neck was turned slightly to the left. The transducer was positioned on the right side of the neck without compromising the internal jugular vein. Special attention was given to the position of the internal jugular vein (between the transducer and the CCA). To measure the maximal diastolic and systolic diameter, the CCA was shown in the longitudinal plane with an optimal picture of the close and far vessel wall. The diameters were measured in the ultrasound M mode operating at a speed of 25 mm/s, and the cursor was set perpendicular to the vessel wall. The diastolic diameter was determined as the smallest lumen diameter directly after the R peak in the ECG in the prejection phase. The systolic diameter was measured when the parallel registered ECG was at the top of the T wave. The IMT of the far arterial wall was measured from the start of the intimal layer (the start of the first echogenic zone) to the start of the adventitia (the start of the echogenic layer on the far side of the transducer).

A pulsed-wave Doppler was used to measure the blood flow properties. In this study, uniform insonification was chosen. The angle of incidence was uniformly 60°, and the vessel wall area was adjusted parallel to the transducer. The range-gate length spanned the lumen of the artery. The pulsed-wave Doppler was kept continuously in the correct position by controlling the sample volume at the position with the duplex capability of the ultrasound system.

The CCA measurements were performed 2–3 cm proximal to the carotid bifurcation to reduce variation due to the measurement site and to avoid possible reverse effects of the vessel expansion in the bifurcation area on the blood flow properties of the CCA. The measurement of the above-mentioned parameters in the CFA were executed in a similar...
fashion ~3 cm proximal to the bifurcation in the artery curvature after the artery leaves the pelvic region.

The mean value from three consecutive measurements was used for the statistical evaluation. The error of these consecutive measurements (S) was computed according to the method recommended by Sachs

\[
S = \sqrt{\frac{n \sum_{i=1}^{n} \sum_{j=1}^{m} (x_{ij} - \bar{x})^2}{n(m-1)}}
\]

where \(x_{ij}\) is the \(j\)th measurement of the \(i\)th examination, \(\bar{x}\) is the mean of the \(i\)th examination, \(m\) is the number of measurements, and \(n\) is the number of examinations (26). S was 0.16 mm for the diastolic diameter of the CCA and 0.15 mm for the CFA, 0.08 mm for the CCA IMT and 0.06 mm for the CFA.

The maximum blood flow speed measured by pulsed Doppler from three consecutive measurements resulted in an S of 4.6 cm/s for CCA and 3.8 cm/s for CFA, S for the average flow speed during one cardiac cycle was 2.7 and 2.2 cm/s for CCA and CFA, respectively.

Calculated parameters. The regional compliance coefficient was calculated as the change in cross-sectional area relative to pulse pressure (24) as

Compliance coefficient (mm²/kPa)

\[
\text{Compliance coefficient} = \frac{1}{\text{PP}} \cdot \frac{D_{\text{dia}} - D_{\text{syst}}}{PP} \quad (1)
\]

where \(D_{\text{dia}}\) is diastolic diameter, \(D_{\text{syst}}\) is systolic diameter, and PP is pulse pressure.

The regional shear rate was calculated with the following equations (7)

Peak shear rate (s⁻¹)

\[
\text{Peak shear rate} = 4 \cdot \frac{\text{maximum blood flow velocity}}{D_{\text{syst}}} \quad (2)
\]

and

Mean shear rate (1/s) = \(4 \cdot \frac{\text{mean blood flow velocity}}{D_{\text{dia}}} \quad (3)

Blood pressure was measured on the right upper arm with an oscillometer. The average was taken from two to four blood pressure values measured during the examination.

Statistics. All data were calculated using the software program Software Package for the Social Sciences for Windows (SPSS version 7.5.2). The arithmetic mean and the SD were used for the descriptive statistics. Groups were compared by the nonparametric Mann-Whitney U-test due to the small group size. To avoid multiple testing errors, we applied Holm’s correction (9).

The relationships between parameters were illustrated by Spearman’s correlation coefficient (r). A P value of <0.05 was considered significant and of < 0.01 highly significant.

Table 1. Anthropometric data, resting heart rate, and diastolic and systolic blood pressure of the endurance-trained athletes, sedentary subjects, and paraplegic subjects

<table>
<thead>
<tr>
<th></th>
<th>Age, yr</th>
<th>BMI, kg/m²</th>
<th>HR, beats/min</th>
<th>RR_{diastolic}, mmHg</th>
<th>RR_{systolic}, mmHg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Athletes</td>
<td>25.9 ± 4.4</td>
<td>21.8 ± 1.6</td>
<td>49.8 ± 9.1</td>
<td>73.8 ± 6.6</td>
<td>125.8 ± 9.0</td>
</tr>
<tr>
<td>Sedentary</td>
<td>24.6 ± 2.3</td>
<td>22.9 ± 3.1</td>
<td>65.5 ± 9.9</td>
<td>76.5 ± 8.0</td>
<td>124.4 ± 10.0</td>
</tr>
<tr>
<td>Paraplegic subjects</td>
<td>38.5 ± 6.8</td>
<td>24.1 ± 1.7</td>
<td>70.4 ± 12.0</td>
<td>80.1 ± 9.5</td>
<td>129.0 ± 7.3</td>
</tr>
</tbody>
</table>

Values are means ± SD. BMI, body mass index; HR, heart rate; RR, blood pressure; RR_{diastolic}, diastolic blood pressure; RR_{systolic}, systolic blood pressure. *P < 0.05 vs. athletes. †P < 0.01 vs. athletes and sedentary subjects. ‡P < 0.01 vs. athletes.

RESULTS

Age was significantly higher in the paraplegic subjects. The athletes had a significantly lower body mass index (BMI) than the paraplegic subjects. However, the absolute differences in BMI were small. As expected, the athletes had the lowest resting heart rate and the paraplegic subjects the highest. Diastolic and systolic blood pressures did not differ significantly among the groups (Table 1).

The diastolic and systolic diameters of the CCA were not significantly different among the groups examined, with the exception of the significantly larger systolic diameter in athletes compared with the paraplegic subjects (Fig. 1A). The CFA diameter was largest by far in the athletes and smallest in the paraplegic subjects, measuring only ~60% of the athletes’ value (Fig. 1B).

The IMT of the CCA and CFA did not differ significantly among the groups (Fig. 1C).

The compliance coefficient of the CCA, as a measure of the volume storage capacity of an artery normalized for blood pressure, was distinctly higher in the athletes than in the sedentary subjects (+23.1%; P < 0.05). In contrast, a marked lower compliance of the CCA was found in the paraplegic subjects than in the sedentary subjects (−37.5%; P < 0.01). In the CFA region the compliance of the athletes was twice as high as in the sedentary subjects (P < 0.01) and 3.5 times higher than in the paraplegic subjects (Fig. 2).

Compliance and diastolic diameter in all groups correlated moderately positively in the CFA (r = 0.62, P < 0.01), whereas no significant correlation was found in the CCA (Fig. 3).

The maximum blood flow velocity in the CCA was higher in the athletes [111 ± 22 (SD) cm/s] than in the sedentary subjects (98 ± 17 cm/s) and lowest in the paraplegic subjects (77 ± 19 cm/s) (P < 0.05 among all groups). The mean blood flow velocity in the CCA did not differ significantly among the groups (athletes 21 ± 3 cm/s, sedentary subjects 24 ± 4 cm/s, paraplegic subjects 20 ± 3 cm/s). The maximum blood flow velocity in the CFA was surprisingly high in the paraplegic subjects (92 ± 22 cm/s), almost reaching the athletes’ level (98 ± 20 cm/s; not significant). The values for the sedentary subjects (76 ± 18 cm/s) were significantly lower than in the other groups (P < 0.05). The mean blood flow velocity of the CFA was highest in the paraplegic subjects (14 ± 4 cm/s), almost double the sedentary values (7 ± 3 cm/s) and still 52.7% higher.
than in the athletes (9 ± 4 cm/s) (P < 0.01 vs. athletes and sedentary subjects).

The athletes showed the highest peak shear rate in the CCA, followed by the sedentary subjects and paraplegic subjects (Fig. 4A). The mean shear rates of the sedentary subjects and paraplegic subjects were significantly different between (Fig. 4B). In the CFA, the mean and peak shear rate of the paraplegic subjects was approximately double that of the other groups. The CFA peak shear rates of the sedentary subjects and athletes were similar because the markedly higher maximum blood flow velocity was related to a higher diastolic diameter.

**DISCUSSION**

The results of this study indicate a combined dimensional and functional adaptation of the CFA, a muscular distributing artery, in leg-trained endurance athletes with a markedly larger diameter associated with higher compliance compared with untrained persons and paraplegic subjects. The shear rates in the athletes and sedentary subjects, however, were similar. In contrast, the paraplegic subjects showed a smaller luminal size of the CFA with lower compliance and a markedly higher shear rate than the trained and untrained group, respectively, indicating an at least partially disturbed response of the CFA wall to changes in blood flow. The CCA did not show any structural differences among groups. However, the compliance and shear rate of the CCA in athletes were significantly higher than in sedentary subjects and both parameters were lowest in the paraplegic subjects compared with the other groups, suggesting a predominantly functional adaptation to changes in blood flow of the CCA. The possible reasons for the group differences will be discussed separately for arterial structure, elasticity, and local shear rate below.

**Arterial structure.** Langille and O’Donnell (17) and Masuda et al. (20) found that a chronic increase or decrease in regional blood flow volume induces an increase or reduction in the diameter of arteries in animals. Our study confirms these fundamental results in humans. The CFA of the athletes, which is
chronically exposed to an increased blood flow because of the endurance training, showed a 17.3% larger diastolic diameter than in the sedentary subjects (Fig. 1B). This finding has also been observed by Wijnen et al. (34). They assessed an 11–12% larger diameter of the CFA in cyclists compared with the sedentary subjects. On the other hand, the paralyzed limbs of the paraplegic subjects with a chronic reduction in blood flow due to forced immobilization had a 28.8% smaller diameter than did the controls. This result is in accordance with observations made by Hopman et al. (10) as well as Huonker et al. (12), who observed a 30–40% smaller diameter in paraplegic subjects than in able-bodied persons.

A conceivably smaller diameter of the CCA as a result of systemic forced long-term inactivity due to paraplegia or larger diameter due to long-lasting intense endurance training was not observed in our study (Fig. 1A). This can be explained by the minor blood flow volume change in the CCA during physical exercise compared with the CFA during leg exercise. Hellström et al. (8) calculated the maximum change in blood flow volume to the head during exercise to be ~30–40% higher than the resting values, whereas during leg exercise there is a ~20- to 30-fold increase in blood flow volume to the femoral artery. If any blood flow-induced change in the diastolic luminal diameter had occurred in the CCA due to an increase or decrease in physical activity, we should have seen it in our study subjects representing the extremes of long-term physical activity or inactivity, respectively.

Concerning the IMT of the CCA, we found the same values for all groups examined (Fig. 1C). With respect to the athlete group, this result contradicts that of Abergel et al. (1). They reported a 13% increase in the IMT of the CCA in professional cyclists compared with normal subjects. However, in their study, several athletes showed a considerable increase in left ventricular wall thickness, above the accepted training-induced upper limit of physiological myocardial hypertrophy (22), which may be due to hypertension in these athletes. A relationship between left ventricular and CCA IMT has been shown in subjects suffering from hypertension (27). This may explain the discrepancy to our data; none of our athletes had a left ventricular wall thickness above the physiological limit.
thickness exceeding 12 mm (data not shown) and/or an increased blood pressure. In addition, our results are supported by Segal et al. (32). They observed no increase in wall thickness after endurance training in the celiac arteries of rats; the probably minor increase in blood flow during exercise in this arterial region may be the common reason for the unchanged CCA IMT in humans. On the other hand, the similarity of the CCA IMT in the paraplegic subjects with the sedentary subjects and athletes suggests a minor decrease in blood flow volume in the CCA in the state of extreme long-term inactivity and thus no visible difference in the CCA IMT in paraplegic subjects.

The IMT of the CFA did not show any group differences (Fig. 1C). Although, according to the Laplace formula, in which vessel diameter is related to vessel wall thickness to compensate for the change in wall tension, we did not find a significant difference between the groups. This may be due to the small group size. However, it would be physiologically meaningful, and in the above-mentioned study Segal et al. (32) observed an increase in the arterial wall thickness of the femoral arteries in trained rats that were exposed to a strong increase in blood flow during exercise. On the other hand, the presumably higher wall tension in the larger arteries of the athletes may be compensated by a structural adaptation of the adventitia, which was not investigated in our study.

Arterial function. Arterial compliance is a measure of the ability of the wall to store volume energy and is therefore important under pulsatile flow conditions. Differences in the compliance of the vessel wall were observed in the CFA among the groups (Fig. 3) that correlated moderately with the vessel dimension changes ($r = 0.62$; Fig. 4). Vascular compliance of the CFA was lowest in the paraplegic subjects, who also had the lowest size of the lumen; on average, a nearly 29% smaller diastolic diameter was associated with 42% lower compliance compared with the sedentary subjects. On the other hand, in the endurance-trained athletes, a ~17% larger lumen diameter of the CFA was accompanied by ~100% higher compliance than in the sedentary subjects.

Compared with the sedentary subjects, the compliance coefficient of the CCA was 34.6% lower in the paraplegic subjects and 23.1% higher in the athletes, although no differences in lumen diameter could be observed between the groups. These results suggest that the CCA and CFA may have undergone structural adaptations not visible in transcutaneous ultrasound scans and/or changes in the regulation of the smooth muscle tone due to the level of physical activity, which influence the functional properties of the arterial wall more markedly than the structure. Several mechanisms may be responsible for the higher vessel wall compliance of both the CCA and CFA in athletes compared with the other groups. These comprise a reduced sensitivity to the vasoconstrictive effect of norepinephrine through an endothelium cell $\alpha_2$-adrenergic-receptor mechanism (3) as well as a decreased basal tone of the smooth muscle cells in the medial layer (18) as a result of endurance training. On the other hand, the lower compliance in the paraplegic subjects examined with a lesion level below $T_4$ may be caused by a possible spillover of epinephrine and norepinephrine plasma levels (30), which may be one reason for the reduced vascular compliance in the CCA and CFA. Beside neurohumoral effects, the different resting heart rates of the groups examined may influence arterial compliance. The 24% lower resting heart rate of the athletes compared with sedentary subjects, which is a known effect of increased vagal tone due to endurance training, may allow a more complete restoration of the arterial lumen diameter during the diastolic phase of the heart cycle (25). As a result, the local buffering capacity of the CCA and CFA may be increased. The moderately lower resting heart rate of the sedentary subjects compared with the paraplegic subjects (5 beats/min) may have only a marginal effect. In addition to the neurohumoral and heart rate-associated effects, it has been shown in animal experiments that endurance training increases the proportion of elastin fibers in the arterial vessel wall and reduces the calcium content (21), therefore maintaining or even increasing vessel wall elasticity. On the other hand, animal experiments have shown that a reduction in blood flow volume in the CCA of 70% results in a 30% reduction in the elastin content of the vessel wall (16). To a lesser degree, this is conceivable in paraplegic subjects. However, with the noninvasive ultrasound method used in our study, it is not possible to decide definitively which of the most important of the above-named reasons are responsible for the observed differences in arterial compliance among the groups examined.

Shear rate. The shear stress hypothesis is often mentioned to explain the acute and chronic changes in arterial vessel wall properties. On the basis of the studies by Langille and O’Donnell (17) and Masuda et al. (20), it can be assumed that the human arterial system strives to maintain wall shear stress by adapting the vessel diameter to changes in blood flow conditions. These hypotheses are based on the minimum-work theory, which emphasizes a constant average wall shear stress, independent of the vessel diameter, within a fluid transport system (14). Training leads to an increase in the regional blood flow volume to the activated muscles and causes endothelium-mediated dilatation (2) in the muscular-type distributing arteries. This obviously counteracts the effect of norepinephrine on the $\alpha_2$-adrenergic receptors of the vascular endothelial cells (33). Prostacyclin and nitric oxide (NO) are examples of mediators for the endothelium-mediated regulation of shear stress via the smooth muscle cells (4). It has been shown that the basal NO production can be increased with physical training resulting in a better dilative capacity as well as in arteries not directly involved in physical exercise (13).

In the present study, the blood flow velocity, a deciding factor for the wall shear rate (28) in the CCA, was highest in the endurance athletes (Fig. 4A). The calculated peak shear rates in the athletes and sedentary subjects were within the same range as the sedentary
subjects in a previous study (~630/s) (7). In a comparison with our data and those of Gnasso et al. (7), the paraplegic subjects showed a ~25% lower peak shear rate in the CCA. The reduced tone of the smooth muscle cells in the CCA as a reaction to the increased local and basal NO production in the endurance athletes could explain the improved compliance. Conversely, a decreased basal and local NO production in the paraplegic subjects as a result of a diminished shear rate in addition to the other factors mentioned may also be a conceivable cause for the decreased compliance.

The larger CFA diameter in the athletes, a structural adaptation caused by chronic endurance training with an increased shear rate during exercise because of the increase in blood flow, is suggested to be a physiological adaptation of the artery to maintain the wall shear rate constant. Because reference values for the shear rate of the CFA in athletes do not exist in the literature, it is only possible to take the CFA shear rate of the sedentary subjects for comparison. Thus similar CFA shear rates of sedentary subjects and athletes (Fig. 4B) strongly suggest that the athletes’ CFA wall responds regularly to changes in regional blood flow and gives support to the hypothesis of constant shear stress regulation in humans. On the other hand, the almost doubled peak and mean shear rate in the paraplegic subjects indicates an at least partial misregulation of the CFA diameter in response to the regional blood flow. It is unclear which mechanism is responsible. From animal experiments, it is known that shortly after sympathectomy the loss of sympathetically mediated vascular tone results in a more distensible artery (19), which counteracts the constrictive stimulus of the reduced shear stress, independent of the change in blood flow. In addition, the smooth muscle cells also are subject to a reduced stimulus to constrict. In the long term, chronic sympathectomy on the aortic wall in rabbits and rats led to a marked increase in the collagen content of the vessel wall (5), which suggests a consecutive stiffening of the wall and a change in circumferential wall stress (15). Although the blood pressure values measured at the upper arm were similar among the examined groups (Table 1), the arm-to-leg pressure relationship of paraplegic subjects might not be similar to the arm-to-leg pressure relationship of normal individuals. Thus, taking all the different influencing factors into consideration, the arterial inward remodeling in the paraplegic subjects can be partially attributed to being physiological as well as nonphysiological. On the other hand, the immobilization of an extremity that is not paralyzed allows for a rather moncausal explanation of the effects of muscular inactivity on the structural and functional properties of muscular-type arteries. Giannattasio et al. (6) found a reduction in lumen size and elasticity in the radial artery of the immobilized upper limb compared with the mobile limb. In contrast to our study, the shear rate was not assessed, which might have shown a physiological response of the arterial wall to changes in local blood flow in these subjects in contrast to the paraplegic subjects in our study.

**Limitations.** It was not possible to form completely matched age groups for this study because we were not able to recruit enough paraplegic subjects of a similar age to the other groups. However, taking a publication by Sandgren et al. (29) into consideration, the age differences here are unimportant. In their study, the diastolic diameter of the CFA increases with age. This leads to the conclusion that, matched with the paraplegic subjects in age, the sedentary subjects and endurance athletes would have even larger diastolic CFA diameters and the group differences would be reinforced. The diastolic CCA diameter in the paraplegic subjects is in fact smaller than in a healthy collective [data from our laboratory’s study (31)]; however, the absolute diastolic-systolic diameter change in the paraplegic subjects is considerably reduced compared with healthy, similarly aged persons. The comparison between groups of dimensional and functional properties of the CCA and CFA in this paper is subject to a slight limitation due to the age differences. In addition, the disturbed sympathetic innervation in the vascular wall of paraplegic subjects may have an impact on the structure and function of the arterial wall as well as a reduced blood flow volume caused by immobilization or an extremely sedentary lifestyle.

In summary, by means of noninvasive ultrasound, this study shows that in the CFA, the major supplying artery during leg exercise, lumen diameter (but not IMT) and compliance are associated positively with the level of physical activity. The major group differences in compliance compared with the diameter indicate that the impact of exercise-induced blood flow changes on the CFA wall appear to affect the elasticity more strongly than the structure. Future research in humans in this field should take both structural and functional arterial properties into consideration. Similar shear rates in sedentary subjects and athletes suggest a normal response of the arterial wall to changes in blood flow and support the hypothesis of constant shear stress regulation in humans. In the paraplegic subjects, the lower elasticity and at least doubled shear rates of the CFA may be partially attributed to a nonphysiological response of the CFA wall to changes in blood flow as a consequence of chronic disturbed sympathetic innervation of the vascular wall. In the CCA, with much lower exercise-induced changes in local blood flow than in the CFA, the results suggest that long-term marked differences in the physical activity level may have no influence on the arterial structure (at least not in the young to middle-age range). The higher compliance of the CCA in the endurance-trained athletes (associated with a lower resting heart rate and/or higher peak wall shear rate) and the lower compliance of the CCA in paraplegic subjects, both compared with the sedentary subjects, may be predominantly caused by an altered tone of the vascular smooth muscle cells. These findings indicate that the level of physical activity and thus the associated exercise-induced blood flow changes (even they are low
in the CCA compared with the CFA) are sufficient to change the ability of the arterial wall to store volume energy and are therefore important under pulsatile flow conditions.

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