The Influence of Pressure and Temperature on the Behavior of the Human Aorta and Carotid Arteries

José M. Atienza,[a] Gustavo V. Guinea,[a] Francisco J. Rojo,[a] Raúl J. Burgos,[b] Carlos García-Montero,[b] Francisco J. Goicolea,[b] Paloma Aragoncillo,[c] and Manuel Elicesa

[a]Departamento de Ciencia de Materiales, Universidad Politécnica de Madrid, Madrid, Spain
[b]Departamento de Cirugía Cardiaca, Hospital Universitario Puerta de Hierro, Madrid. Spain
[c]Departamento de Patología Cardiaca, Hospital Clínico Universitario San Carlos, Madrid, Spain

Introduction and objectives. The thermomechanical behavior of human arteries is still not well characterized despite its importance for understanding arterial physiology, and for evaluating and improving surgical procedures. The aim of this study was to provide, for the first time, experimental data illustrating how the mechanical responses of two types of human artery–the carotid artery and the aorta–are affected by changes in temperature.

Methods. The mechanical properties of the arteries were derived in vitro from internal pressure–external diameter curves measured at four different temperatures (ie, 17, 27, 37, and 42ºC). Coefficients of expansion and stiffness were obtained by thermomechanical analysis. The condition of the arterial wall was determined histologically.

Results. The aorta and the carotid artery became slightly more compliant as the temperature increased. In both vessels, the coefficient of expansion depended critically on internal pressure. At low pressures, the coefficient of expansion was negative (ie, the vessel contracted when heated), whereas close to a specific threshold pressure, which is different for each type of artery, the coefficient became positive.

Conclusions. The mechanical behavior of arteries is affected by the combination of internal pressure and temperature. Consequently, the effect of this combination should be taken into account in clinical situations involving a change in temperature. Moreover, the strength of the effect depends on the type of artery under study. As a result, more detailed experimental data focusing on vessels of clinical interest are required.

Key words: *Aorta. Carotid artery. Mechanical stress. Blood pressure. Temperature. Fundamental research.*

Influencia de la presión y la temperatura en el comportamiento de la aorta y las carótidas humanas

Introducción y objetivos. La respuesta termomecánica de las arterias humanas es poco conocida a pesar de su importancia para la comprensión de la fisiología arterial, y para la evaluación y mejora de los procedimientos quirúrgicos. El objetivo de este trabajo es aportar por vez primera datos experimentales que muestren cómo se ve afectada la respuesta mecánica de dos tipos de arterias humanas –aorta y carótida– por los cambios de temperatura.

Métodos. La respuesta mecánica de las arterias se ha obtenido in vitro a través de la medición de las curvas presión interior-diámetro exterior para 4 temperaturas (17, 27, 37 y 42 ºC). Se ha realizado un análisis termomecánico para obtener los coeficientes de dilatación y la rigidez del material. El estado de la pared arterial se ha evaluado mediante análisis histológico.

Resultados. Las arterias aorta y carótida aumentan ligeramente su flexibilidad con la temperatura. El coeficiente de dilatación de ambos vasos depende críticamente de la presión interior aplicada. A bajas presiones, el coeficiente de dilatación es negativo (el vaso se contrae cuando se calienta), mientras que por encima de cierta presión umbral –distinta para cada tipo de arteria– el coeficiente de dilatación se hace positivo.

Conclusiones. El efecto combinado de la presión interior y la temperatura afecta al comportamiento de las arterias y, por ello, debe ser tenido en cuenta al abordar situaciones clínicas que impliquen cambios de temperatura. La intensidad de este efecto depende del tipo de arteria estudiada, lo que requiere la obtención de datos más detallados, centrados en los vasos de interés clínico.

Palabras clave: *Aorta. Arterias carótidas. Tensión mecánica. Presión arterial. Temperatura. Investigación básica.*

INTRODUCTION

The mechanical properties of the arteries are of fundamental importance in the functions they perform as part of the cardiovascular system. Knowledge of these properties is vital if we are to understand the physiology

This work was funded by the Spanish Ministry of Science and Technology via project MAT 2005-6320, and by the Madrid Regional Government via the ESTRUMAT-CM program (reference MAT/77).

Correspondence: Dr. J.M. Atienza.
Departamento de Ciencia de Materiales. Escuela Técnica Superior de Ingenieros de Caminos. Universidad Politécnica de Madrid.
Profesor Aranguren, s/n. 28040 Madrid. España.
E-mail: jmatienza@mater.upm.es

Received May 3, 2006.
Accepted for publication November 2, 2006.

of the circulatory apparatus and to design therapies and techniques for repairing cardiovascular lesions and defects. Since the arteries are physiological conduits subject to internal pressure,[1] their mechanical properties are involved in the outcome of the vast majority of treatments for these vessels (the repair of arterial ruptures or dissections, endarterectomy, angioplasty, stents, by-passes, etc).

The thermomechanical response of human arteries remains little understood despite the increasing importance given to temperature in clinical procedures in recent years. Some types of heart surgery or surgical procedures involving the major arteries (aortic dissection, heart transplant, coronary artery surgery, valve surgery, repair of congenital heart defects, etc) are performed using systemic hypothermia; sometimes even profound hypothermia is induced (18-20ºC) with extracorporeal circulation.[2] The liquids used for the preservation of organs for transplantation, and in conventional surgery, are usually used at a temperature of 4-18ºC. Other therapies involve hyperthermal conditions, such as perfusion with warm solutions for the treatment of soft tissue tumors in the arms and legs, and in the sealing of coronary dissections with radiofrequency-heated balloons.[3-5]

The studies performed to date,[6-8] however, have not adequately taken into account the thermomechanical behavior of artery walls, which is very complex[9,10] (non-linear viscoelasticity, incompressibility, anisotropy, etc) and difficult to describe with simples indices. Although different variables have been proposed that explain certain arterial properties with differing degrees of success, all have their limitations. In addition, to simplify matters, they do not usually take into account the influence of thermal effects. Moreover, many of the experimental data available on the mechanical properties of arteries have been obtained in tests performed under physiological conditions (37ºC, 120-60 mm Hg). While such tests provide information within the context of the normal working environment, the small mechanical and thermal ranges involved allow no laws or models to be produced that predict what will happen in other situations. Numerical models have been shown useful in cardiovascular medicine for predicting the behavior of tissues and for the optimization of treatments.[9] However, to develop models of arterial thermomechanical behavior that can be applied to clinical situations, experimental data obtained under a wide range of conditions are required; such tests would also allow the responses of diseased vessels to be examined. To date, information of this type has been very scant, especially with respect to human vessels. The aim of the present work was therefore to examine the thermomechanical behavior of the human carotid artery and aorta over a wide range of pressures (0-200 mm Hg) and temperatures (17-42ºC) in order to predict repercussions at the clinical level. The study subjects had no apparent vascular disease and were middle aged (aorta donors) or of advanced age (carotid donors); the present comparisons should therefore be read with caution, and certainly, the results should not be used as absolute references. All work was performed at the Dept. of Materials Science of the *Universidad Politécnica de Madrid*, with the collaboration of the Cardiac Pathology Units of the *Puerta de Hierro* and *Clínico San Carlos de Madrid* hospitals.

METHODS

The Studied Arteries: the Aorta and Carotid Artery

The arteries studied were the human aorta and carotid artery. Carotid arteries (11 complete vessels) were obtained from eight individuals who had died of non-atherosclerotic causes. None had been smokers, and none had any history of high blood pressure, diabetes or hypercholesterolemia. The mean age of these donors was 81 (7) years. The donated arteries were therefore theoretically free of vascular disease, but generally came from patients of advanced age.

Four segments of ascending aorta were obtained from four cadavers, all heart donors (mean age 49 [10] years). Given this origin, all were considered theoretically healthy. The longest lengths of vessel possible were extracted in order to facilitate their use in pressurization tests. All vessel samples were obtained after acquiring the permissions required by current legislation.

The carotid artery segments analyzed were 55 (11) mm long and 9.3 mm in diameter; the ascending aorta segments were 63 (12) mm long and 20 (2) mm in diameter. Before extraction of the vessels, all were measured in order to proportionally reproduce their natural state of axial elongation during pressurization tests.

All samples were preserved at a temperature of 4-8ºC in cardioplegic solution[11-13] (mannitol 60 mmol/L, lactobionic acid 80 mmol/L, glutamic acid 20 mmol/L, sodium hydroxide 100 mmol/L, calcium chloride-2H_2O 0.25 mmol/L, potassium chloride 30 mmol/L, magnesium chloride-6H_2O 13 mmol/L, histidine 30 mmol/L, glutathione 3 mmol/L; osmolarity 345 mOsmol/kg). All tests were performed within 48 h of vessel extraction.

Histological Analysis

All samples were subjected to histological analysis in order to study the composition and structure of the mid-layer (the layer largely responsible for their mechanical behavior). The distribution of the elastic and muscular fibers were recorded, and the position of any possible atherosclerotic lesions noted. This analysis required the removal of a section from each sample vessel, which was

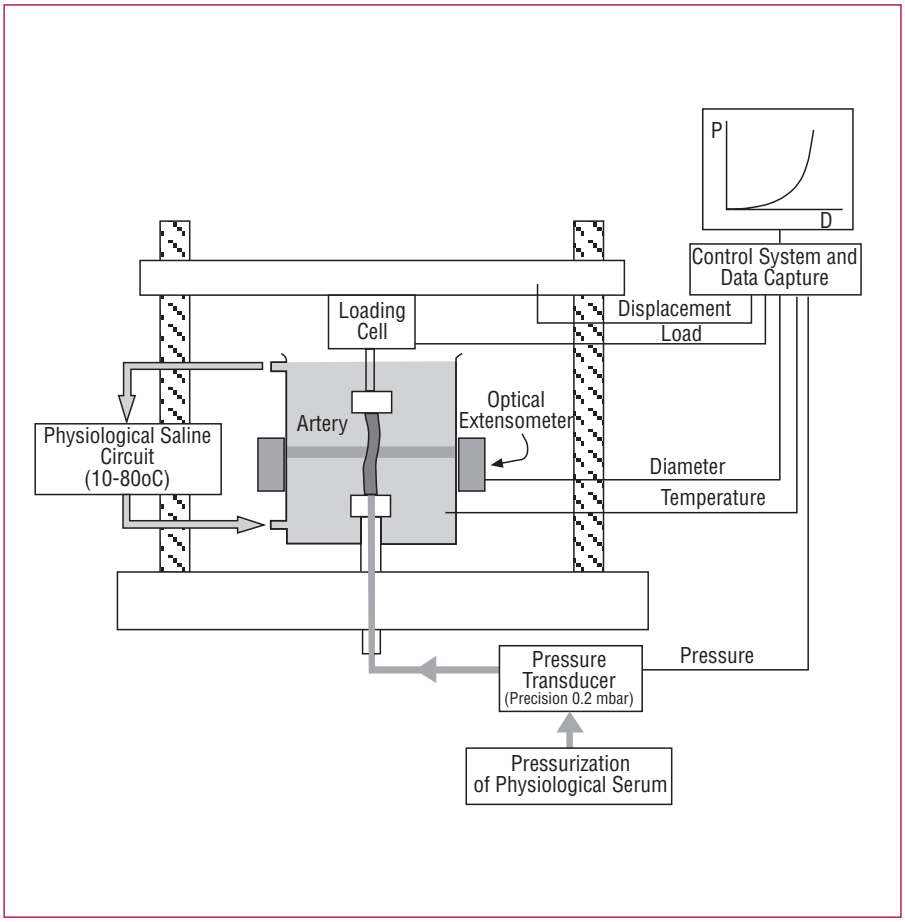

Figure 1. The experimental apparatus.

immediately fixed in buffered formol and later stained with hematoxylin and eosin plus orcein. The ratio of elastic/muscular fibers was recorded. Atherosclerotic lesions were graded according to the criteria of the International Committee of the American Heart Association.[14,15]

Pressurization Tests

The mechanical properties of the vessels were investigated using a pressurization test, and the internal pressure/external diameter curve at different temperatures plotted for each. The aim of this test was to reproduce in vitro the kinds of mechanical stress that might be experienced by the vessels in the living organism under different conditions. Figure 1 shows the apparatus used. All tests were performed inside a transparent, polymethacrylate cell; this allowed the test vessel to be seen at all times and permitted its dimensions to be recorded optically. The external diameter was measured at the center of the segment using a Keyence LS-7500 optical extensometer (precision 0.001 mm). During the test the vessels were submerged in PBS, the temperature of which was maintained within ±1°C by an Unitronic 6320200 thermostatically controlled water bath. The temperature of the vessel was measured using a type K thermocouple positioned at a distance of 5 mm.

The arteries were held in the clamps of an Instron 5866 mechanical test apparatus; this was used to obtain the proportional axial elongation recorded before removal. The precision of this axial extension was 0.0001 mm (measured using the transducer incorporated into the apparatus). The lower clamp allowed for the internal pressurization of the vessel with PBS (monitored using a Druck PMP 4000 transducer [precision 0.15 mm Hg]).

All tests were performed with the vessels in a passive state, ie, with no electrical or chemical activation of the vascular wall cells. The tests were performed at 17, 27, 37, and 42°C, and the internal pressure/external diameter ratio determined in each case.

The vessels were attached to the clamps by mechanical and chemical (cyanocrylate) means and first submerged in PBS at 17°C for 10 min. After thermal equilibration, axial elongation was performed so that their proportional length matched that of their in vivo state. The vessels were then conditioned to pressure changes by putting

them through 10 cycles of pressurization of between 0 and 200 mm Hg (increasing by 2 mm Hg/s) in order to obtain a stable behavior pattern and repeatable results. A new cycle following the same pattern was then undertaken and the internal pressure/external diameter ratio continuously monitored. At the end of this test phase the PBS was warmed to 27ºC (and successively to 37ºC and 42ºC) and the same steps repeated. The mean duration of a complete test (ie, covering all four temperatures) was 3 h. At the end of the test, a piece of each artery was fixed in formol for histological analysis (see above).

Thermomechanical Analysis

The internal pressure/external diameter curves for the different temperatures were used to determine the thermomechanical behavior of the arteries. This was characterized by the use of two indices: the coefficient of thermal expansion *(α)*, and the Hayashi stiffness parameter *(β)*.[16] Together, these provide an adequate idea of the influence of temperature on the behavior of the artery wall. The former provides information on the change in the dimensions of the artery with temperature, while the latter reflects the influence of temperature on arterial rigidity.

The coefficient of thermal expansion measures the change in dimensions experienced by a material with a change in temperature, and is defined as the relative variation of the diameter (ΔD/D) divided by the increase in temperature (ΔT)

$$\alpha=(\Delta D/D)/\Delta T \quad (1)$$

The integration of this equation provides:

$$D=D_{Ref}e^{\alpha(T-TRef)} \quad (2)$$

where D_{Ref} is the diameter at temperature $T_{Ref.}$ Equation (2) can then be transformed to provide:

$$D=D^{*}e^{\alpha T} \quad (3)$$

where $D^{*}=D_{Ref}e^{-\alpha TRef}$. The coefficient of thermal expansion can therefore be obtained simply by fitting experimental data into equation (3).

The rigidity of the walls of the studied arteries was characterized by the Hayashi stiffness parameter. Hayashi et al,[16] proposed an exponential equation for describing the relationship between arterial pressure and diameter (p-D):

$$\ln (p/p_s)=\beta(D/D_s-1)$$

where p_s is a reference pressure (normally 100 mm Hg)[8] and D_s is the diameter of the artery at pressure p_s (ln indicates naperian logarithm).

RESULTS

Histological Analysis

The ascending aorta and the carotid arteries are close to the heart; both are elastic arteries.[17-19] These become distended during the contraction of the ventricles, transforming the pulsating blood flow produced by the heart beat into a continuous capillary flow; their elastic fiber content is therefore high. The mid-layer of both these arteries is formed by concentric laminas of elastin alternating with smooth muscle and collagen fibers. The differences between the aorta and carotid arteries lie in the dimensions of this mid-layer and the percentage of elastic fibers. The histological analysis showed the aorta to have a significantly thicker mid-layer than the carotid artery (1.47 [0.04] mm compared to 0.22 [0.03] mm). In addition, the aorta was found to have a higher percentage of elastic fibers (50% compared to <35%); these values agree with those typically reported in the literature.[17-19]

No atherosclerotic lesions were detected. However, given the age of the donors (especially those who donated carotid arteries), age-related alterations were seen, characterized by a profound thickening of the intima associated with an increased number of smooth muscle cells surrounded by variable quantities of connective tissue (type IV lesions according to the American Heart Association). Type IV lesions are degenerative arterial lesions that appear with age[14,15]; this classification differentiates them from more complicated lesions associated with pathological processes such as fibroatheromas, calcifications, hemorrhages, and thromboses. Some 78% of the carotid arteries examined and 56% of the aortas were thus affected.

Pressurization Test

Figure 2 shows the internal pressure/external diameter curves for the two types of artery studied. These curves reflect the variation of the external diameter of the vessels with respect to the pressure exerted on their walls at different temperatures. The mean values for each type of artery are shown. Relative diameters were calculated by dividing the absolute diameter results by the reference diameter (37ºC and 0 mm Hg) for each type of artery. This allowed comparisons to be made between the two vessels.

The effect of temperature on arterial behavior is more easily seen, however, when each recorded diameter is divided by a reference diameter for each temperature (at 0 mm Hg). Figure 3 shows the result of this treatment, in which all the curves pass through the origin. The figure shows that the change in relative diameter of the vessels increases with temperature.

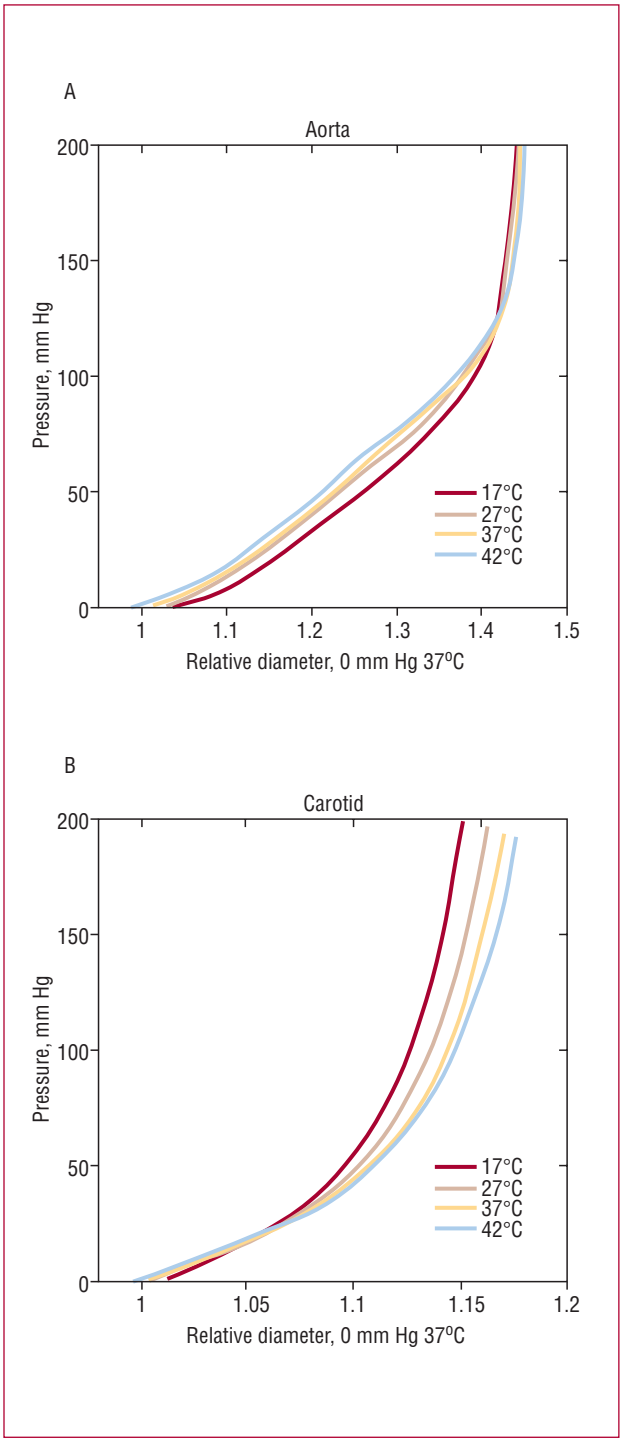

Figure 2. Internal pressure/external diameter curves for different temperatures: (A) aorta, (B) carotid artery. The reference diameter for each vessel was taken as that at 0 mm Hg and 37ºC.

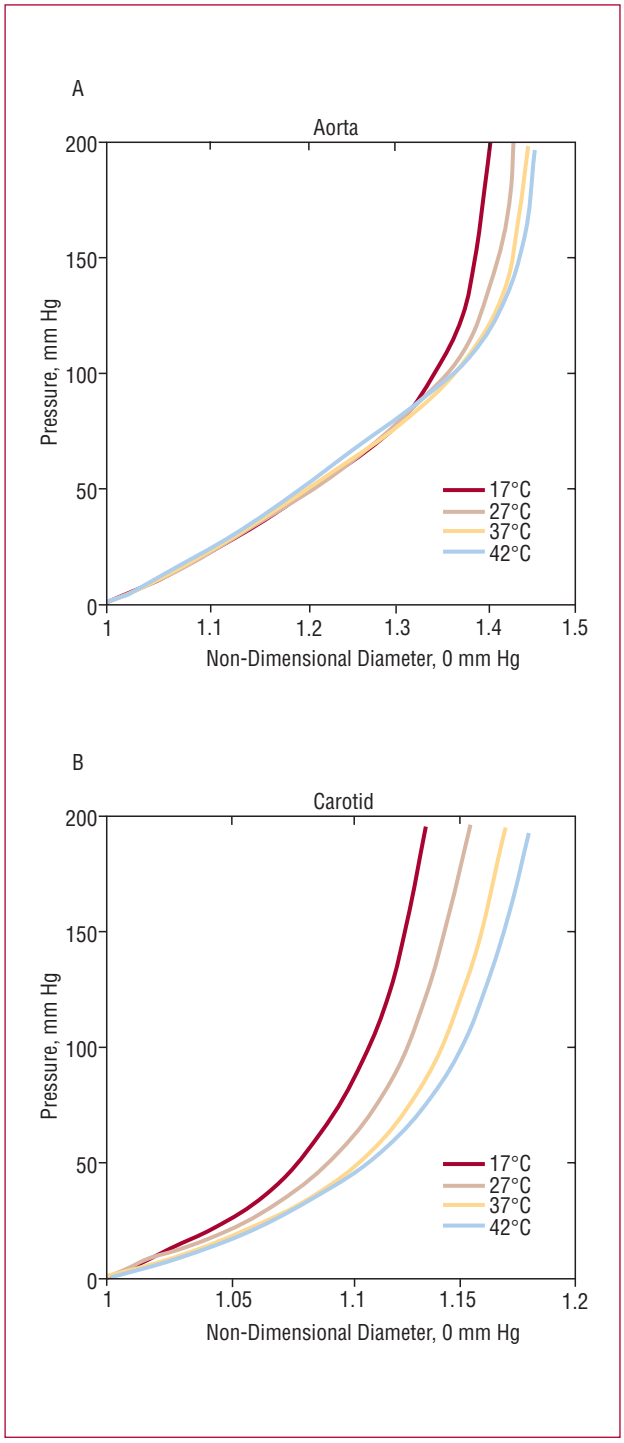

Figure 3. Non-dimensional internal pressure/external diameter curves for different temperatures: (A) aorta, (B) carotid artery. The reference diameter for each vessel was taken as that at 0 mm Hg at each temperature.

Thermomechanical Analysis

Coefficient of Thermal Expansion

Figure 4 shows the mean coefficient of thermal expansion for each artery, calculated by fitting the temperature-diameter (T, D) data pairs to equation (3) at each test pressure. In both vessels, the coefficient of thermal expansion is negative at low pressure but becomes positive above a certain threshold–approximately 20 mm Hg for the carotid artery and 150 mm Hg for the aorta.

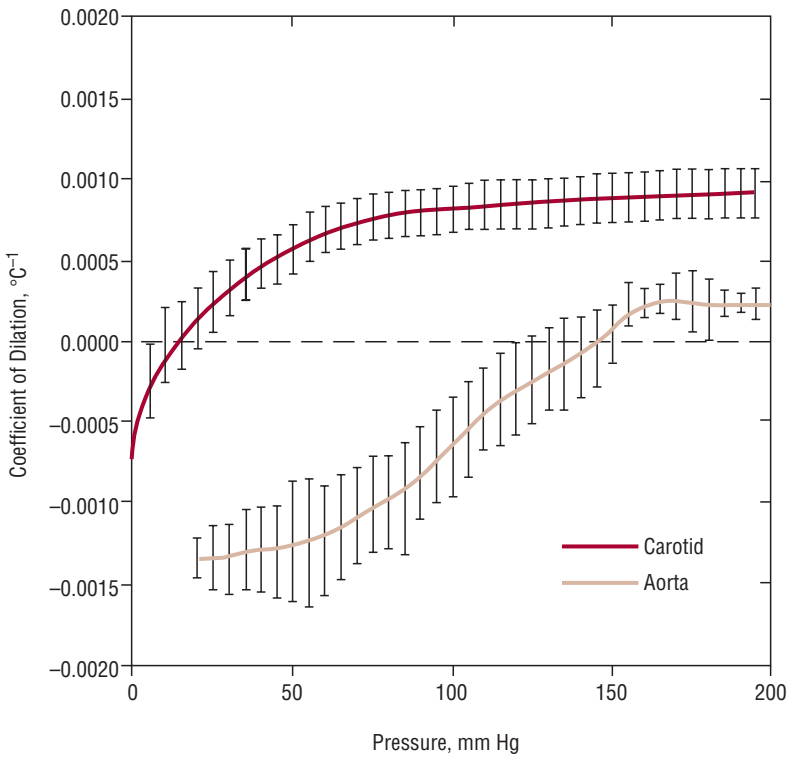

Figure 4. Variation of the coefficient of thermal expansion with internal pressure. Bars are standard errors of the mean.

The value of the coefficient of thermal expansion increases progressively with pressure, although the progressive decline in the slope of the curves in Figure 4 would appear to indicate the existence of a saturation point.

Although the trend of the curves was similar, the coefficient of thermal expansion of the aorta was always smaller than that of the carotid artery, and did not become positive until a higher pressure was reached.

The Hayashi Stiffness Parameter

The Hayashi stiffness parameter was obtained for each type of vessel by treating the internal pressure/external diameter curves with equation (4); Figure 5 shows the means and errors for both the aorta and carotid artery. For both vessels the value of the stiffness parameter decreased with temperature, although no significant differences were seen even between the two temperature extremes (17ºC and 42ºC). Figure 5 shows that the carotid arteries are more rigid than the aorta.

DISCUSSION

The first study in the literature on the effect of temperature on human blood vessels was that of Roy,[20] which was published in 1880. This author, who performed experiments on the arteries of humans, cows and sheep over the 16-54ºC interval, reported arterial walls to show the peculiar behavior of contracting when heated and expanding when cooled, ie, they have a negative coefficient of thermal expansion. In 1954, Lawton[21] obtained similar results with dog arteries. However, other authors report conflicting results. Dobrin et al[22] found no significant influence of temperature on dog arteries, Kang et al[4] reported the same for sheep arteries, and Herrera et al[23] obtained contradictory results for the coefficients of expansion of rat and pig arteries. Thus, the literature contains little information on the influence of temperature on the behavior of arteries, and what there is inconclusive, and sometimes even contradictory. This confusion may be due to cross-over effects produced by the combined action of the state of mechanical tension (largely a consequence of the internal pressure of the

vessel), the temperature, and the state of muscular activation of the vessels examined.

The present work analyzes the effect of temperature on two human arteries (the aorta and the carotid artery) without muscular activation, and the results show that it has an important influence on their behavior.

Increasing temperature led to the arteries becoming more deformable as temperature increased (Figure 3). Although the effect was similar in both types of artery, their mechanical behavior was somewhat different. In absolute terms, the diameter of the aorta grew more than that of the carotid (note the difference in the scales of Figures 2A and B), and showed a more notable initial elastic phase; this is due mainly to the presence of the elastic fibers, which are found in greater proportion in the aorta than in the carotid artery. The greater stiffness seen for the carotid arteries may also be due to the age of their donors; arteries may become more rigid with age.[6,8]

Figure 5 shows the Hayashi stiffness parameters for both arteries. Higher values indicate lower deformability; as might be expected from the above, the values for the carotid arteries were much higher. In agreement with that shown by the internal pressure/external diameter curves, the rigidity of the vessels decreased with increasing temperature. However, the differences were not statistically significant.

The two arteries behaved similarly in the pressurization test. When the pressure applied was low, the largest diameters were obtained at lower temperatures in both vessels (Figure 2). This confirms their possession of a negative coefficient of thermal expansion when not under load. However, when the pressure was increased to physiological levels the diameter of the vessels increased with temperature. Figure 4 shows the variation in the coefficient of thermal expansion with internal pressure for both types of artery. Despite the dispersion of the results, it is clear that for both vessels the coefficient of thermal expansion is negative at low pressures, gradually rising with pressure until it becomes positive.

This finding of a negative coefficient of thermal expansion at low temperature agrees with the results of Roy[20] for the human aorta. Curiously, the value deduced from his work (approximately -7×10^{-4}ºC^{-1}) is similar to those shown in Figure 4. Lawton[21] obtained similar results in mammalian arteries under no pressure, and explained this in terms of the enthropic origin of the elastic behavior of the artery wall (similar to that of elastomers), which is directly related to the elastic fiber content.

Recently, Herrera et al[23] have reported results for pig renal arteries and rat aortas. While the former dilated as temperature increased, the latter contracted. The authors suggest the explanation for these different behaviors to lie in the characteristics of the arteries studied. They postulate that the elastic fibers were behind the contraction seen in the aorta, and that the muscular component of the wall and the state of activation masked their effect in the more muscular renal arteries.

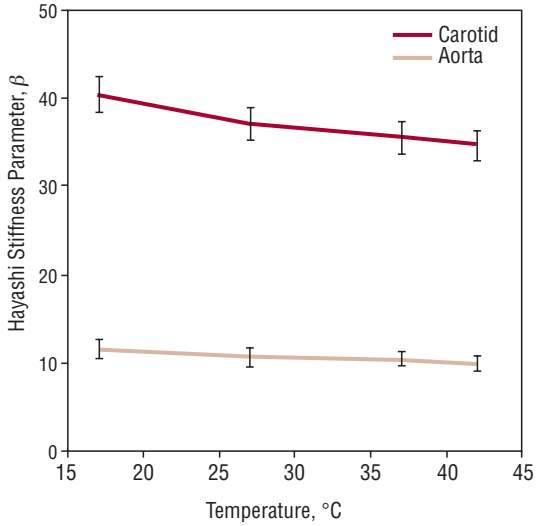

Figure 5. Variation of the Hayashi stiffness parameter with the temperature. Bars are standard errors of the mean.

The ascending aorta and carotid artery are both elastic arteries and in the present work were studied without muscular activation. In agreement with that expressed above, it might be expected that they should both show a negative coefficient of thermal expansion. However, and despite the fact that Figure 4 shows the most negative values to be seen in the aorta (which is much more elastic than the carotid artery), the coefficient of thermal expansion depends on their internal pressure. This dependency is complex and needs to be further studied. Recently, we have shown[24] that the axial lengthening to which arteries are subjected also affects their thermomechanical behavior; it should therefore be taken into account along with the effect of internal pressure.

Given that the coefficients of thermal expansion shown by the studied aortas and carotid arteries were very different (differences of more than 10^{-3}ºC^{-1} were recorded), variation might also be expected between vessels of other origin, when they come from donors of different age, or when subject to different pathological processes. This might become a problem when vessels with different coefficients of expansion

are in contact, eg, at anastamoses, or at the point of contact between an atherosclerotic plaque and the arterial wall. Changes in temperature could lead to high mechanical tensions developing that could result in the deterioration of the points of union, perhaps even to rupturing. In such situations a detailed study of the thermomechanical properties of the elements in contact is required.

CONCLUSIONS

This work is the first to provide experimental data on the thermomechanical behavior of the human aorta and carotid artery, and shows that the combined effect of internal pressure and temperature on the response of the artery is notable. The present tests were performed in vitro and over a wide range of pressures (0-200 mm Hg) and temperatures (17-42ºC). The results extend our knowledge of the mechanical properties of arteries and their physiology, and could be of use in the construction and testing of numerical models.

The rigidity of the vessels, reflected in the slope of the internal pressure/external diameter curves, decreases with increasing temperature. However, the differences (measured by the Hayashi stiffness parameter) were not significant. The greater stiffness of the carotid arteries is due to their lower content of elastic fibers.

Both types of vessel have a coefficient of thermal expansion dependent on the internal pressure to which they are subjected. This coefficient is negative (the vessels contract as the temperature increases) when the internal pressure is sufficiently low. However, after a threshold, it becomes positive, and reaches a saturation point at pressures higher than those experienced physiologically.

The thermomechanical behavior of arteries should be borne in mind when confronted with clinical situations in which, for example, different types are in contact (ie, at anastamoses), and in which changes in temperature could induce undesirable mechanical stress at the point of union. The intensity of this effect will depend upon the exact tissues/materials in contact; experimental data are therefore needed to characterize the behavior of those of clinical interest.

REFERENCES

1. Atienza JM, Guinea GV, Elices M. Colapso de conductos presurizados: Aplicación a los vasos sanguíneos. Anales de Mecánica de la Fractura. 2002;19:519-24.
2. Khatri P, Babyak M, Croughwell N, Davis R, White W, Newman F, et al. Temperature during coronary artery bypass surgery affects quality of life. Ann Thorac Surg. 2001;71:110-6.
3. Goicolea J, Aranda L, Alfonso F, Segovia RHJ, Rodríguez A, Macaya C. Mechanisms of radiofrequency thermal balloon angioplasty. A study with intracoronary ultrasound. J Am Coll Cardiol. 1995;25:143A-4A.
4. Kang T, Resar J, Humphrey J D. Heat-induced changes in the mechanical behavior of passive coronary arteries. J Biomech Eng. 1995;117:86-93.
5. Orihara K, Biro S, Hamasaki S, Eto H, Miyata M, Ikeda Y, et al. Hyperthermia at 43ºC for 2h inhibits the proliferation of vascular smooth muscle cells, but not endothelial cells. J Mol Cell Cardiol. 2002;34:1205-15.
6. Humphrey JD. Mechanics of arterial walls: Review and directions. Critical Rev Biomed Eng. 1995;23:1-162.
7. Schulze-Bauer CAJ, Holzapfel GA. Determination of constitutive equations for human arteries from clinical data. J Biomech. 2003;36:165-9.
8. Hayashi K. Experimental approaches on measuring the mechanical properties and constitutive laws of arterial walls. J Biomech Eng. 1993;115:481-8.
9. Goicolea JM. Factores biomecánicos y su influencia en la función cardiovascular. Rev Esp Cardiol. 2005;58:121-5.
10. Bia D, Aguirre I, Zócalo Y, Devera L, Fisher EC, Armentano R. Diferencias regionales en viscosidad, elasticidad y amortiguamiento parietal de arterias sistémicas: Análisis isopulsátil de la relación presión-diámetro arterial. Rev Esp Cardiol. 2005; 58:167-74.
11. Hernández A, Borrego JM, Gómez S, Gutiérrez E, Lage E, Hinojosa R, et al. Myocardial preservation using celsior: clinical results in high risk cardiac transplantation. Transplant Proc. 2005;37:1543-5.
12. Remadi JP, Baron O, Roussel JC, Habash OA, Treilhaud M, Despins P, et al. Myocardial preservation using Celsior solution in cardiac transplantation: Early results and 5-year follow-up of a multicenter prospective study of 70 cardiac transplantation. Thorac Surg. 2002;73:1495-9.
13. Menashé P, Termignon JL, Pradier F, Grousset C, Mouas C, Alberici G, et al. Experimental evaluation of celsior, a new heart preservation solution. Eur J Thorac Cardiovasc Surg. 1994;8:207-13.
14. Stary HC, Chandler AB, Glasgov S, Guyton JR, Insull W, Rosenfeld ME, et al. A definition of initial, fatty streak and intermediate lesions of atherosclerosis. A report from the committee on vascular lesions of the council of arteriosclerosis, American Heart Association. Circulation. 1994;89:2462-78.
15. Stary HC, Chandler AB, Dinsmore RE, Fuster V, Glasgov S, Insull W, et al. A definition of advanced types of atherosclerotic lesions and histological classification of atherosclerosis. Circulation. 1995;92:1355-74.
16. Hayashi K, Handa H, Nagasawa S, Okamura A, Moritake K. Stiffness and elastic behaviour of human intracraneal and extracraneal arteries. J Biomech. 1980;13:175-84.
17. Fawcet DW. Tratado de histología. 12ª ed. New York: Interamericana McGraw-Hill; 1995. p. 413-4.
18. Joung B, Heath JW Weather S. Histología funcional. 4ª ed. Madrid: Elsevier España; 2002. p. 147-,8.
19. Schoen FJ, Cotran RS. Los vasos sanguíneos. In: Kumar V, Cotran RS, Robins SL, editors. Patología humana. Madrid: Elsevier España; 2004. p. 325-60.
20. Roy CS. The elastic properties of the arterial wall. J Physiol. 1880;3:125-59.
21. Lawton RW. The thermoelastic behaviour of isolated aortic strips of the dog. Circ Res. 1954;3:120.
22. Dobrin PB, Canfield T. Identification of smooth muscle series elastic component in intact carotid arteries. Am J Physiol. 1977;232:H122-30.

23. Herrera B, Eisenberg G, Holberndt O, Desco MM, Rábano A, García-Barreno P, et al. Paradoxical effects of temperature on vascular tone. Cryobiology. 2000;41:43-50.
24. Atienza JM, Burgos R, Morán M, García-Montero C, Goicolea FJ, Acevedo MLE, et al. Experimental data on the thermo-mechanical behaviour of human aorta. In: Hamza MH, editor. Proceedings of the III International Conference on Biomechanics. Benidorm: Acta Press. 2005;485:15.