

Acute vascular and hemodynamic effects of cranio-sacral therapy in healthy subjects

Robinson Ramírez Vélez¹, María del Pilar Espinosa², Carlos Alejandro López-Albán³, Ricardo Antonio Agredo-Zúñiga⁴

1. Grupo GICAEDS. Facultad de Cultura Física, Recreación y Deportes, D.C, Colombia

2. Fundación FUNDALINFA, Santiago de Cali, Colombia

3. Centro para la Investigación en Salud y Rendimiento Humano ZOE, Santiago de Cali, Colombia

4. Programa de Fisioterapia, Universidad de San Buenaventura, Seccional Cartagena, Cartagena de Indias, Colombia

Abstract

Introduction: Craniosacral therapy (CST) has been shown to be effective in treating several neurological conditions affecting the central nervous system. There are no reports in the conventional medical literature the CST interventions on vascular response and hemodynamic parameters in humans. **Material and Methods:** We conducted an experimental study in 8 healthy subjects (4 men and 4 women; mean age, 22.5 ± 1.70 years) were enrolled. The intervention consisted of a single 50 min session of the 10 step protocol of CST intervention as previously described by Upledger. Were measured before and after in carotid artery (arterial compliance, systolic and diastolic arterial diameter, pressure-strain elasticity modulus, systolic/diastolic ratio, resistance index, and pulsatility index) by an ultrasound with an echo-tracking system and the hemodynamic parameters (blood pressure and heart rate). **Results:** After intervention with TCS, we found lower values of blood pressure (systolic and mean), $P < 0.01$. Also found statistically significant changes in plethysmographic parameters: systolic diameter and pressure-strain elasticity modulus after intervention, $P < 0.01$. No differences in flow velocity, systolic/diastolic ratio, arterial compliance, resistance index, pulsatility and vascular function. **Conclusion:** Applying of craniosacral therapy modifies the physiology of the vascular system and hemodynamic parameters in healthy subjects.

Key words: Craniosacral therapy, Complementary medicine, Clinical benefit, Vascular function, Hemodynamics.

Artículo de investigación

Introduction

Craniosacral Therapy (CST) is a very popular non-pharmacological method used for the treatment and prevention of migraines, although at present there is limited evidence for safety and efficacy.¹ CST identifies restrictions in the craniosacral system, including the skeletal system, the membranes and the cerebrospinal fluid (CSF) surrounding the brain and spinal cord. This manipulation by gentle patting, normalizes the rate and fluid from the central nervous system (CNS), through the release of restrictions in peri-spinal tissue like fascias and the connective tissue in general.¹ The manual palpation and manipulation of this system, theoretically affect sensory, motor, cognitive and emotional processes which are controlled by the CNS.^{1,2} Often, CST has been used as a complementary treatment for physical therapists,

osteopaths, chiropractors and alternative therapists in order to reduce conventional drug use for pain in a variety of pathological conditions³⁻⁵. Current scientific evidence reports the use of CST in patients with migraine, either alone or in combination with standard medical care⁵⁻⁸.

Several authors who have used CST suggest significant benefits, including: decrease in the intensity of pain associated with migraine, decrease in the use of drugs and improvement in physical symptoms^{5,1,9}. However, these studies lack standardization in the diagnosis of the headache or migraine and lack documentation of criteria with scientific validity such as the co-morbidities of the intervened population, the number of treatments applied, psychosocial conditions, strategies for coping with pain, previous trauma, clinical monitoring, changes in quality of life, or these were not properly reported.

Additionally, these studies did not include control groups or randomization, which partially invalidate its clinical efficacy.

CST also lacks studies evaluating the physiological effects that may occur during its application. Also, the biochemical, biomechanical and metabolic mechanisms have not yet been elucidated¹. Since its creation in the early 80's, it has been postulated that some of the physiological effects of CST are due to changes in the cardiovascular system, influenced by the movement and the increase in CSF cycles and changes in the acid/base balance, which increase cell metabolism, decreasing the impulses in the cranial rhythm and restricting respiratory functions observed in the spinal cord^{1, 2}. Other empirical approaches suggest that due to pulsations of the spinal and epidural arteries, and the cerebral vascular plexus, a state of stasis appears in the cortico-spinal fluid and it is present frequently in diseases and symptoms related to the CNS⁷. Stasis of the CSF acts on the cardiovascular system triggering hemodynamic, respiratory, endocrine and neurological effects⁸.

Thus, the objective of this study is to evaluate the acute physiological effects of CST on the cardiovascular system, which has been suggested to be one of the main systems affected by this type of therapy. This could partly explain the effectiveness reported empirically in the treatment of headaches with a vascular origin.

Patients and Methods

Patients. An experimental study was designed with 8 healthy individuals (4 men and 4 women) from a higher education level institution in the city of Cali-Colombia. The selection was performed after inviting subjects to participate and intention sampling. Subjects with the following characteristics, identified through a survey and/or medical exam were excluded: cigarette smoking, recent major surgeries or trauma, known endocrine disease, multiorgan or systemic autoimmune disease, significant respiratory or systemic disease, significant cardiac dysfunction, systemic infectious disease or a recent musculoskeletal disorder (<1 month). All participants gave written informed consent and the Ethics Committee of the Academic Center approved the study, under the ethical considerations recognized by the Helsinki Declaration and current Colombian legislation (Resolution 8430 of 1993 of the Ministry of Health).

Clinical and anthropometric evaluation. The following data was obtained from each participant: i) family and personal history of cardiovascular risk, ii) basic anthropometric values (weight and height) using standardized techniques, and iii) blood pressure measurement - always measured by the same person and with the same instrument (Dinamap® oscillometric method). Measurements were performed on the right arm with the participant lying comfortably, the cuff placed at heart level, adjusting the size of the diameter of the arm for each subject. Three measurements were averaged per participant. With these results we calculated the mean arterial pressure (MAP) by the equation: diastolic pressure + [systolic pressure - diastolic pressure] / 3].

Arterial physiological measurements by plethysmography. A SIEMENS SG-60® ultrasound scanner was employed with an Echo-Tracking® application. Measurements were carried out in a scale of grays from the right common carotid artery to its bifurcation and the internal carotid artery. A longitudinal assessment was performed and the thickness of the intima and media were measured at two levels: the carotid bulb and the internal carotid artery 10 mm from the bifurcation. Color Doppler was used to better define the vascular lumen while the participant on supine and with an ambient temperature of 22-25 °C, according to the procedure previously described by Bots et al¹. and Hulthe et al². The carotid artery was chosen since it is an important body segment, close to CST manipulation, and because it is a site associated with headaches of vascular origin¹. The participant remained in supine decubitus at least 5 min before the exploration (Figure 1). The 7 Mhz lineal transducer was positioned so that the carotid artery could be observed longitudinally maximizing the echoes from the interphase between the media and adventitia. When this line of division was captured sharply, the two available tracers were placed on interphase points that were diametrically opposed. Once placed, these tracers were displaced simultaneously with the arterial wall, which allowed the recording of the distance between both in function of time. The ECG was monitored during the exploration so that the system could detect the onset of the systolic (Vfs) and diastolic (Vfd) flow velocities. With this technique the following were obtained: a) graphic recording of the pulse waveform; b) maximum or systolic arterial diameter (Ds) corresponding to the highest point of each pulse, at which moment the vessel is subjected to the highest pressure (systolic blood pressure [SBP]); c) minimum or diastolic arterial diameter (Dd), corresponding to the

lowest point, at which moment the vessel is subjected to the least pressure (diastolic blood pressure [DBP]). All measurements were performed by the same operator with > 3 years experience in the technique (Figure 1), 2 min before (baseline values) and immediately after intervention with CST. With the collected data, the program implemented a series of equations to calculate the three parameters related to arterial elasticity:

- Arterial distensibility (D_a) (mm^2/kPa) = $\pi (D_s^2 - D_d^2) / 4(\text{SBP} - \text{DBP})$
- Elastic Modulus (E_p) (kPa) = $(\text{SBP} - \text{DBP}) D_d / (D_s - D_d)$
- Arterial rigidity index (β index) = $\ln (\text{SBP} - \text{DBP}) / [(D_s - D_d) / D_d]$

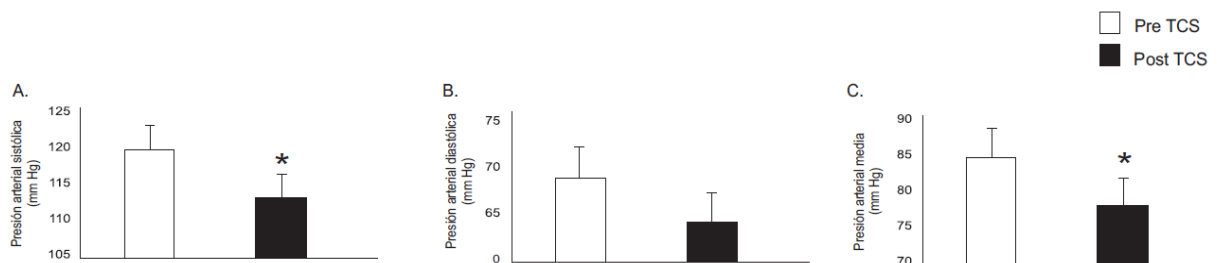
Additionally, the most common indices of the waves, representing the vascular flow velocity qualitatively, were calculated.

- Pulsatility index (IP) = $(S-D)/C$
- Systole/diastole relationship (S/D) = S/D
- Resistance index (IR) = $(S-D)/S$

Where S is the peak of maximum velocity at the end of systole, D is the peak of maximum velocity at the end of diastole and C is the average of velocity during the cardiac cycle.

Hemodynamic measurements. Monitoring of the electrocardiogram, heart rate and blood pressure 2 min

Figure 1. Assessment of vascular function by Doppler. A) Preoperative Doppler Record. B) Doppler signal record (note the passage of blood in the common carotid artery). C) Echo-tracking® Application by high resolution plethysmography.



prior to the implementation of the protocol, during and immediately after each intervention was performed.

Intervention. Participants after refraining from drinking alcohol or performing any physical activity were placed in a room with controlled temperature and humidity, with no visual or acoustic stimuli. The cranio-sacral therapy protocol was applied in this study following the 10-step sequence from TC v-2 Upledger Institute: Subject supine. Stations to listen (heels, back of the feet, thighs, EIAS, ribs, shoulders, 3 escuchas by the cranial vault). 1. Still point. 2. L5-S1 Decompression, Liberation of the sacroiliac joints, dural tube traction, 3. Diaphragms: a. pelvic, b. respiratory, c. estrada toracica, d. Hyoid e. Cranial Base. 4. Rolling / sliding dural tube. 5. Frontal Rise ("Lift") (Vertical membranous system). 6. Parietal rise ("Lift") (Vertical membranous system). 7. Spheno-

basilar compression / decompression. 8. Temporal Techniques (horizontal membranous system). 9. ATM compression / decompression 10. "Still Point" (CV-4). Stations to listen. An expert physiotherapist trained on CST (> 3 years) applied this protocol.

Analysis plan. The statistical analysis was performed with SPSS, version 19. The results are expressed as mean $\pm$ standard deviation. A factorial ANOVA was used to test the changes before and after the intervention in each variable, after CST. For all measurements, a statistical significance value of $p < 0.05$, was considered.

Results

The sample included 8 participants aged between 18 and 25 years. The average age was 22.5 ± 1.7 years. Table

1 summarizes the anthropometric and clinical data. All results were within the normal age range.

After CST lower blood pressure values (systolic and mean), $p < 0.01$, were evidenced (Figure 2). Statistically significant changes in the following plethysmographic parameters were shown after CST: systolic diameter and Ca, $p < 0.01$ (Figure 3). No differences in flow velocity, the systole/diastole relationship, elastic modulus, resistance, pulsatility and vascular stiffness indices (Figure 3) were found.

Discussion

Upledger and other pioneers who followed his observations and investigations popularized cranio-sacral therapy in the 80s 1, 2. Although movement of CSF is unlikely, it has been postulated that application of pressure on the cranio-sacral system results in its transmission through the system 5. In practice, CST stimulates some sensory nerves and the autonomic nervous system and thus induces blood circulation recovery. 7, 9

Table 1. Clinical characteristics (n = 8)

	(Media $\pm$ DE)
Age, (years)	22,5 $\pm$ 1,7
Weight, (kg)	60,3 $\pm$ 8,2
Height, (cm)	168,9 $\pm$ 6,1
Body mass index, (kg/m ²)	22,2 $\pm$ 1,9
Systolic Blood Pressure, (mm Hg)	123,0 $\pm$ 14,0
Diastolic Blood Pressure, (mm Hg)	69,5 $\pm$ 8,4
Average blood pressure, (mm Hg)	82,2 $\pm$ 10,8
Heart rate, (bpm)	70,9 $\pm$ 70,9

Figure 2. Craniosacral therapy effects on the hemodynamic response in healthy subjects.
Data presented as mean $\pm$ SD.

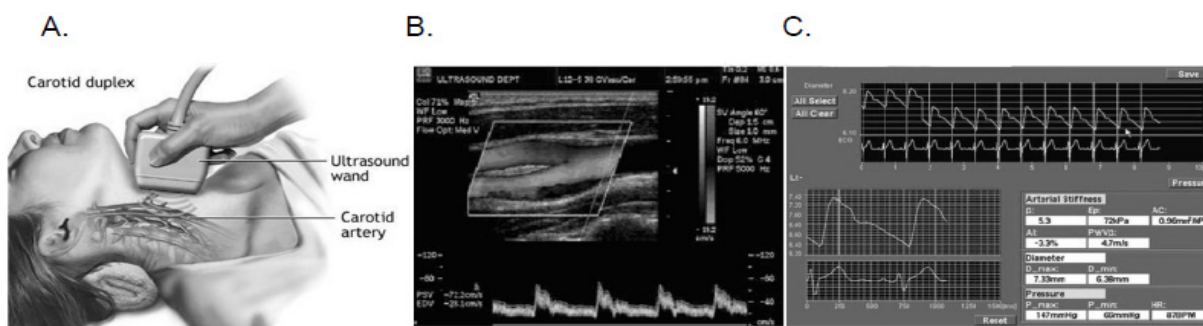
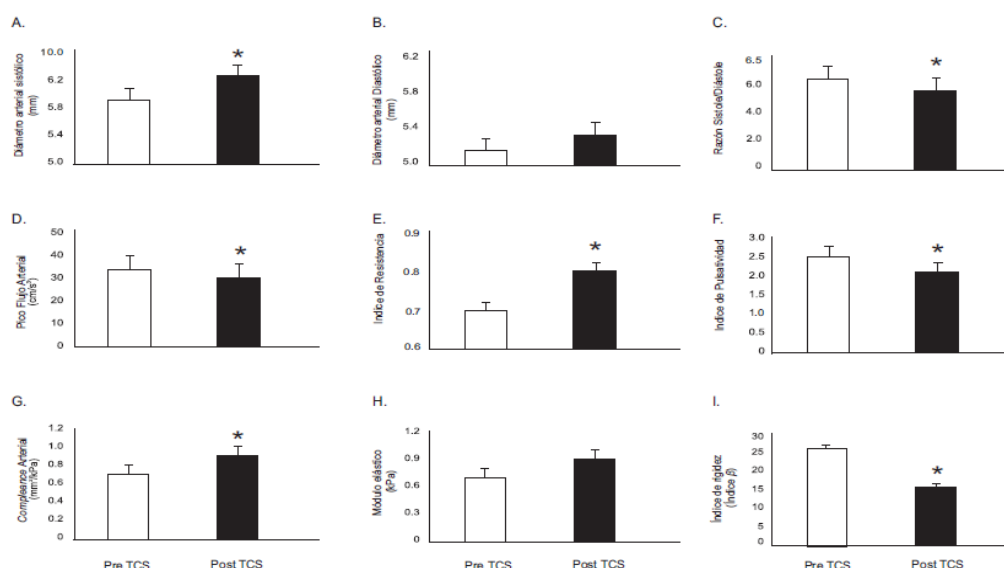


Figure 3. Craniosacral therapy effects on the acute vascular response and vascular parameters in healthy subjects.

Data presented as mean $\pm$ SD.



The main findings of this study were the following. First, a session of 50 min of CST with the 10-step protocol (TCS-v2) can change arterial compliance, arterial diameter and systolic blood pressure. To our knowledge, there are no reports in the literature about the effect of CST on the vascular response and hemodynamic parameters in humans. Several studies have reported that arteries of medium and large caliber, mainly in the cardiothoracic region of healthy middle-aged adults, appear to be less rigid than in adults with alterations of the musculoskeletal, vascular, lymphatic and endocrine systems.^{1,2} It has been suggested that the mechanisms underlying the increase in systolic arterial diameter induced by CST are based on activation of thin nerve fibers that release vasoactive neuropeptides and nitric oxide (NO), resulting in vasodilation and increased blood flow.¹ The resulting decrease in vascular tone (vasodilation) and blood pressure may directly and indirectly contribute to the regulation of brain activity and induce hemodynamic changes. These changes have been confirmed in this study, as well as a significant decrease in vascular rigidity. However, further studies are needed to elucidate the origin of these findings.

Secondly, the direct assessment of the dynamic compliance of the carotid artery revealed that CST could modify it approximately 29%. We can only speculate on the mechanism by which CST improves arterial compliance. This physiological variable is mainly determined by the elastic and intrinsic properties of the artery.¹ The arterial wall elements which determine arterial compliance are elastin and collagen (the structural determinant) and the vasoconstrictor tone exerted by smooth muscle cells (the functional determinant).¹ Since biochemical changes in the composition of elastin and collagen in the arterial wall are produced in the long term, it is unlikely that the short-term change of CST modify arterial distensibility by this mechanism. Nevertheless, it is possible that increased pulse pressure and mechanical distension during CST "the collagen stretch", change the crosslinking of collagen.¹ This would increase arterial compliance, a mechanism known as mechanotransduction.¹ This vascular variable may also be modified short-term, acutely, through modulation of the adrenergic sympathetic tone of the smooth muscle cells in the vessel walls. It is possible that CST increases arterial compliance by reducing chronic inhibitory

influence exerted by the adrenergic sympathetic tone, either directly or by increasing the sympathetic inhibitory effect of NO¹. Future studies will be needed to determine the physiological mechanisms by which CST generates changes on arterial distensibility.

Another finding was a significant decrease in blood pressure, approximately 7%. The mechanism by which CST can achieve this effect is not clear yet, but it has been suggested that CST can modify the neurotransmitter center in the central nervous system^{10, 19}. In our study, we hypothesized CST induces vascular and hemodynamic effects. The arteries (large and medium) have two types of innervation: i) sensitive afferent fibers and ii) autonomic sympathetic ones. Both type of fibers penetrate into the adventitia and media and the vascular smooth muscle of arteries. The axon reflex induced by CST induces vasodilation in arterioles, possibly by releasing NO in the axon terminal¹ or perhaps by blocking the sympathetic system by stimulation of the central nervous system². However, this effect was not determined.

Overall, our results have a number of potentially important clinical implications, since a reduction in arterial compliance is believed to contribute significantly to the pathophysiology of cardiovascular disease related to age (for example: migraine of vascular origin)¹. However, the reduced elasticity of arteries is the mechanism which predominates in reducing cardiovagal baroreflex sensitivity¹. This contributes to the onset of ventricular tachyarrhythmias, ischemic cerebral disease and sudden cardiovascular death with advancing age¹. It's possible that a greater compliance in the carotid artery (and perhaps the ascending aorta) is associated with increased baroreflex sensitivity¹⁴. Moreover, a reduction in arterial compliance related to age can affect cardiac function by increasing vascular impedance and this is related to cerebrovascular disease¹⁴. This in turn decreases left ventricular performance¹ and increases blood pressure, significant risk factors for cardiovascular and renal disease¹⁹⁻²².

Thus, our findings indicate that the vascular system is modified by the application of CST, and the temporality or intensity between the physiological processes that determine the vascular morphology and physiology are altered due to the endothelial reactivity of vessels, their tone and the peripheral vascular resistance. However, our results should be interpreted with caution due to the

limitations of the study. First, few subjects were included with a limited range of age. Secondly, the feeding patterns were not taken into account and they can modulate vascular and metabolic responses. Third, the inability to control the plethysmographic parameters during the CST application was not evaluated. Therefore, any changes that might have occurred during the intervention were not recorded. We emphasize though that if our hypothesis was the ability of CST to generate changes in vascular morphology physiology and hemodynamics, this limitation does not seem relevant.

Conclusion

This study is one of the first experimental research studies using plethysmographic techniques designed to examine changes in the local vascular morphology and physiology in large vessels. Our observations may provide a starting point for further clinical studies focusing in the field of rehabilitation and treatment of musculoskeletal and vascular disorders with the application of CST. In fact, some benefits in movement restricting conditions associated with the presence of vascular migraines have been shown with the use of CST^{1,2}. Future studies should explore these and other physiological and metabolic changes with more participants in order to validate the observations described here.

Ethical Responsibilities

Protection of people and animals

The authors declare that procedures were performed according to the ethical standards of the committee on responsible human research and in accordance with the World Medical Association and the Declaration of Helsinki.

Data Confidentiality

The authors declare they have followed their workplace protocols on data publication and all patients have received sufficient information and have given their written informed consent to participate in the study.

Right to privacy and informed consent

The authors have obtained the informed consent of patients and/or subjects. These documents are in possession of the main author.

Conflicts of interest

The authors declare no conflicts of interest

References

1. Harrison RE, Page JS. Multipractitioner Upledger CranioSacral Therapy: descriptive outcome study 2007-2008. *J Altern Complement Med*. 2011;17:13-7.
2. Upledger JE, Vredevoogt J. *Craniosacral therapy*. Eastland Press, Seattle (1983).
3. Downey PA, Barbano T, Kapur-Wadhwa R, Sciote JJ, Siegel MI, Mooney MP. Craniosacral therapy: the effects of cranial manipulation on intracranial pressure and cranial bone movement. *J Orthop Sports Phys Ther*. 2006;36:845-853.
4. Hanten WP, Olson SL, Hodson JL, Irnler VL, Knab VM, Magee JL. The effectiveness of CV4 and resting position techniques on subjects with tension type headaches. *J Manual & Manip Therap*. 1999;2:64-70.
5. Raviv G, Shefi S, Nizani D, Achiron A. Effect of craniosacral therapy on lower urinary tract signs and symptoms in multiple sclerosis. *Complement Ther Clin Pract*. 2009;15:72-5.
6. Ferguson A. A review of the physiology of cranial osteopathy. *J Osteopathic Med* 2003; 6:74-88.
7. Green C, Martin CW, Bassett K, Kazanjian A. A systematic review and critical appraisal of the scientific evidence on craniosacral therapy. Vancouver: B. C. Office of Health Technology Assessment, Center for Health Services and Policy Research, University of British Columbia; 1999:54.
8. Greenman P. Advanced technique for craniosacral therapy. In *Physical Medicine and Rehabilitation* Edited by: Tomsaki MA. Philadelphia, Hanly & Belfus; 2000:125-139.
9. Mann JD, Faurot KR, Wilkinson L, Curtis P, Coeytaux RR, Suchindran C, Gaylord SA. Craniosacral therapy for migraine: protocol development for an exploratory controlled clinical trial. *BMC Complement Altern Med*. 2008;8:28.
10. Bots ML, Evans G, Riley W. Carotid intima-media thickness measurements in intervention studies design options, progression rates, and sample size considerations: a point of view. *Stroke* 2003;34:2985-94.
11. Hulthe J, Wikstrand J, Emanuelsson H. Atherosclerotic changes in the carotid artery bulb as measured by B-mode ultrasound are associated with the extent of coronary atherosclerosis. *Stroke*. 1997;28:1189-94.
12. Kurth T, Mohamed S, Maillard P, Zhu YC, Chabriat H, Mazoyer B, et al. Headache, migraine, and structural brain lesions and function: population based Epidemiology of Vascular Ageing-MRI study. *BMJ*. 2011;18;342:c7357.
13. Rubin MJ, Etchison MR, Condra KA, Franklin Jr TD, Snoddy AM. Acute effects of ultrasound on skeletal muscle oxygen tension, blood flow and capillary density. *Ultrasound Med Biol*. 1990;16:271-7.
14. Robinson SE, Buono MJ. Effect of continuous-wave ultrasound on blood flow in skeletal muscle. *Phys Ther*. 1995;75:145-9.
15. Higashi Y, Yoshizumi M. New methods to evaluate endothelial function: method for assessing endothelial function in humans using a strain-gauge plethysmography: Nitric oxidedependent and -independent vasodilation. *J Pharmacol Sci*. 2003;93:399-04.
16. Nichols WW, O'Rourke MF. *McDonald's Blood Flow in Arteries. Theoretical, Experimental and Clinical Principles*, Fourth Edition. 4th ed. London, UK: Arnold; 1998.
17. Tanaka H, Dinunno FA, Monahan KD, Clevenger CM, DeSouza CA, Seals DR. Aging, habitual exercise, and dynamic arterial compliance. *Circulation*. 2000;102:1270-5.
18. Bruel A, Ortoft G, Oxlund H. Inhibition of cross-links in collagen is associated with reduced stiffness of the aorta in young rats. *Atherosclerosis*. 1998;140:135-45.
19. Hoffman BD, Grashoff C, Schwartz MA. Dynamic molecular processes mediate cellular mechanotransduction. *Nature*. 2011;475:316-23.
20. Boutouyrie P, Lacolley P, Girerd X, Beck L, Safar M, Laurent S. Sympathetic activation decreases medium-sized arterial compliance in humans. *Am J Physiol* 1994;267:H1368-76.
21. Barenbrock M, Spieker C, Witta J, Evers S, Hoeks AP, Rahn KH, et al: Reduced distensibility of the common carotid artery in patients treated with ergotamine. *Hypertension* 1996;28:115-9.
22. Zanzinger J. Role of nitric oxide in the neural control of cardiovascular function. *Cardiovasc*

- Res. 1999;43:639-49.
23. Sumner DS, Zierler RE. Vascular physiology. In: Rutherford RB, Editor. Vascular Surgery, 6th Ed., 2005, Elsevier Saunders, p. 75-122.
24. Toda N, Okamura T. The pharmacology of nitric oxide in the peripheral nervous system of blood vessels. *Pharmacol Rev.* 2005;55:271-324.
25. Castro-Sánchez AM, Matarán-Peñarocha GA, Sánchez-Labraca N, Quesada-Rubio JM, Granero-Molina J, Moreno-Lorenzo C. A randomized controlled trial investigating the effects of craniosacral therapy on pain and heart rate variability in fibromyalgia patients. *Clin Rehabil.* 2011;25:25-35.
26. Rowe JW. Clinical consequences of age-related impairments in vascular compliance. *Am J Cardiol.* 1987;60:68G-71G.
27. Billman GE, Schwartz PJ, Stone HL. Baroreceptor reflex control of heart rate: sudden cardiac death. *Circulation.* 1982;66:874-79.
28. Lakatta EG. Cardiovascular regulatory mechanisms in advancing age. *Physiol Rev.* 1993;73:413-67.
29. Curtis P, Gaylord SA, Park J, Faurot KR, Coble R, Suchindran C, et al. Credibility of low-strength static magnet therapy as an attention control intervention for a randomized controlled study of CranioSacral therapy for migraine headaches. *J Altern Complement Med.* 2011;17:711-21.
30. Jäkel A, von Hauenschild P. A systematic review to evaluate the clinical benefits of craniosacral therapy. *Complement Ther Med.* 2012;20:456-65.

Corresponding author

Robinson Ramírez-Vélez
Facultad de Cultura Física, Deporte y Recreación. Universidad Santo Tomás
Carrera 9 N° 51-23. Bogotá, D.C, Colombia
Teléfono: +55(1) 587 8797 ext. 1811
Fax: +55(1) 8578850
E-mail: robin640@hotmail.com

Received: Junio 18 de 2013

Accepted: Julio 10 de 2013



UNIVERSIDAD CES

Un Compromiso con la Excelencia

Resolución del Ministerio de Educación Nacional No. 1371 del 22 de marzo de 2007