J Clin Monit Comput (2013) 27:47–53
DOI 10.1007/s10877-012-9391-8

ORIGINAL RESEARCH

Pulmonary artery pulsatility is the main cause of cardiogenic oscillations

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Received: 12 July 2012 / Accepted: 9 August 2012 / Published online: 22 August 2012

Abstract The genesis of cardiogenic oscillations, i.e. the small waves in airway pressure (COS_{paw}) and flow (COS_{flow}) signals recorded at the airway opening is under debate. We hypothesized that these waves are originated from cyclic changes in pulmonary artery (PA) pressure and flow but not from the physical transmission of heartbeats onto the lungs. The aim of this study was to test this hypothesis. In 10 anesthetized pigs, COS were evaluated during expiratory breathholds at baseline with intact chest and during open chest conditions at: (1) close contact between heart and lungs; (2) no heart–lungs contact by lifting the heart apex outside the thoracic cavity; (3) PA clamping at the main trunk during 10 s; and (4) during manual massage after cardiac arrest maintaining the heart apex outside the thorax, with and without PA clamping. Baseline COS_{paw} and COS_{flow} amplitude were 0.70 ± 0.08 cmH_2O and 0.51 ± 0.06 L/min, respectively. Both COS amplitude decreased during open chest conditions in step 1 and 2 ($p < 0.05$). However, COS_{paw} and COS_{flow} amplitude did not depend on whether the heart was in contact or isolated from the surrounding lung parenchyma. COS_{paw} and COS_{flow} disappeared when pulmonary blood flow was stopped after clamping PA in all animals. Manual heart massages reproduced COS but they disappeared when PA was clamped during this maneuver. The transmission of PA pulsatilty across the lungs generates COS_{paw} and COS_{flow} measured at the airway opening. This information has potential applications for respiratory monitoring.

Keywords Cardiogenic oscillations · PEEP · Pulmonary blood flow · Ventilation · Heart-lungs interaction

1 Introduction

Cardiogenic oscillations (COS) are small waves produced by heartbeats superimposed on gas (COS_{gas}), pressure

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(COS_{paw}) and flow (COS_{flow}) signals recorded at the airway opening. Such mechanical waves, represented by COS_{paw} and COS_{flow}, have important clinical implications. For example, they participate in the process of gas mixing within lungs [1–5], they can be used to assess lung mechanics [6, 7], they can auto-trigger assisted breaths during apnea [8] or they can differentiate patients with central and obstructive sleep apneas [9].

The genesis of COS has been a matter of debate in the past and results from studies are contradictory. On the one hand, several authors concluded that the main cause of COS is the direct physical transfer of heartbeats onto the lungs because both organs are in close contact each other [3, 10–14]. On the other hand, variations in thoracic blood volume during the cardiac cycle or the transmission of pulmonary artery (PA) pulse waves throughout the airways have been related to COS by other researchers [15–18].

We have recently studied the origin of COS in patients undergoing cardiopulmonary bypass, a model that allows to independently manipulate the factors related to COS origin [18]. We observed that COS_{paw} and COS_{flow} amplitude was not related to the physical contact between heart and lungs but was directly proportional to the increment in pulmonary blood flow. Despite that we have demonstrated that PA pulsatility causes COS in humans, there is still some contradictory information against this theory. In this regards, Fukuchi et al. [4] showed in dogs that COS in nitrogen signal obtained in the right middle lobe persisted when its PA branch was blocked by inflating the balloon of a Swan-Ganz catheter. These data moved us to conduct the present experimental study performing extreme physiological maneuvers ethically impossible to do in humans to provide further evidence about the origin of COS.

Therefore, the main objective of this study was to test the hypothesis that PA pulsatility is the main cause involved in the origin of COS_{paw} and COS_{flow}. In an experimental model we studied how COS_{paw} and COS_{flow} were affected by the following maneuvers: (1) interrupting PA blood flow by clamping the main PA artery, (2) totally isolating the heart from lungs lifting the heart apex outside the thorax and (3) performing manual massages after cardiac arrest with the heart apex outside the thorax, with and without PA clamping.

2 Methods

The protocol was approved by the local Ethics Committee for animal experimental research of the Fundación Jiménez Díaz, Madrid, Spain. We studied ten pigs (weight 27 ± 3 kg, length 125 ± 5 cm) anesthetized with a continuous i.v. infusion of propofol 100–150 μg kg min and remifentanyl 1 μg kg min. Saline solution was continuously infused i.v at a rate of 5 mL kg h. The trachea was intubated by a cuffed endotracheal tube and the lungs were mechanically ventilated by a Servo*i* (Maquet Critical Care, Solna, Sweden) using a constant flow mode with a tidal volume of 7 mL/kg, respiratory rate of 25 bpm, I:E of 1:2, FiO_2 of 50 % without positive end-expiratory pressure.

Standard monitoring included ECG, pulse oxymetry, rectal temperature and capnography (NICO; Philips Respironics, Wallingford, CT, USA). Invasive systemic arterial pressure was recorded by a femoral arterial catheter (PV2015L20, Pulsion, Germany). A pulmonary artery catheter was placed through the right jugular vein (Edwards Life-sciences, Irvine, CA, USA). The hemodynamic parameters studied were heart rate (HR), mean arterial pressure (MAP), central venous pressure (CVP), mean pulmonary artery pressure (MPAP), cardiac output (CO) and stroke volume (SV = CO/HR) determined by transpulmonary thermodilution (PiCCO2, Pulsion, Germany). Thermodilution was performed by triplicate before each protocol step.

All described physiologic parameters were recorded continuously and stored in a customized data acquisition system programmed in LabView (National Instruments, Austin TX) during each of the protocol steps.

2.1 Measurement of COS_{paw} and COS_{flow}

COS were recorded during the protocol performing expiratory breath holds maneuvers by pressing the expiratory hold button of the ventilator for 15 s [18]. We used a dedicated sensor system for measuring airway pressure relative to ambient pressure (HCLA Series, Miniature amplified Low pressure sensors, range ±75 mbar), differential pressure across a Fleisch pneumotachograph. This sensor was placed at the airway opening between the Y piece and the endotracheal tube to assess flow (HCLA Series, Miniature amplified Low pressure sensors, range ±12.5 mbar), and barometric pressure to convert to standard conditions (HCA-BARO Series, Sensor Technics, range 600–1100 mbar)(CSEM, Lanquart, Switzerland). Data from sensors were acquired with a microprocessor and converted into a data stream out of a USB port. The device samples data at 200 Hz for Paw and differential pressure.

2.2 Protocol

After anesthesia induction and animal instrumentation we allowed 30 min for stabilization. Baseline data were recorded at the end of this period in a closed chest condition. Then, the chest was opened by medial sternotomy and the heart was exposed by a sternal retractor after

performing a pericardiotomy. In this condition we studied COS in the following sequential steps:

1. *Close contact* between heart and lungs confirmed by visual inspection.
2. *No contact* between heart and lungs: the heart was embraced by a soft bandage and then the apex was gently lifted out the thoracic cavity for 15 s.
3. *Pulmonary artery clamping:* in order to test the role of PA pulse pressure and flow in the origin of COS we clamped the artery for 10 s. This maneuver was performed in two different conditions: a) with close contact between heart and lungs as in point 1 and, b) without contact between heart and lungs by lifting the heart out the thoracic cavity as in point 2.
4. *Manual cardiac massages:* at the end of the protocol the animal was sacrificed using i.v. potassium chloride. Immediately after cardiac arrest, cardiac massage was performed with the heart lifted out the thoracic cavity (point 2), with and without clamping the PA.

At baseline, steps 1, 2 and 3 the breath hold maneuvers were done by triplicate but at step 4 such maneuver was performed only once. Between each protocol step we introduced at least 5 min of baseline ventilation.

2.3 Data analysis

Raw data of physiologic parameters including ECG, PA pressure, airway flow and pressure were continuously recorded and analyzed off-line. A dedicated software written in Matlab® (Mathworks, Natick, MA, USA) identified COS and ECG signals for their analysis. COS were defined as the small amplitude waves of higher frequency appearing in the pressure and the flow signals between two R waves. Fifteen to twenty COS were analyzed per protocol step depending on the HR. As steps were repeated 3 times in each protocol condition, we analyzed at least 45 COS per step in each animal. The software automatically calculated COS_{paw} and COS_{flow} amplitude detecting the nadir-to-peak distance of each oscillation corresponding to one cardiac cycle.

We analyzed the hemodynamic data only at baseline (closed chest) and in steps 1 and 2. In steps 3 and 4 we only could describe the presence or absence of COS.

2.4 Statistical analysis

The statistical analysis was performed using the program SPSS (SPSS Inc, Illinois, USA). For comparison of variables between baseline measurements and data from protocol steps 1 and 2 a repeated-measurements analysis of variance was used. If the analysis of variance (F statistic) was significant, the Student–Newman–Keuls post-test was applied. Values are presented as mean ± SD and level of significance was established at $p < 0.05$.

3 Results

No animal showed COS_{paw} and COS_{flow} after clamping the PA (Fig. 1). PA pressure signal disappeared during PA clamping maneuver which coincided with an instantaneous disappearance of COS in the pressure and flow signals. This effect was observed when PA clamping was performed with and without contact between heart and lungs (step 3a and 3b, respectively). When PA clamping was released, COS_{paw} and COS_{flow} reappeared immediately.

After the administration of i.v. potassium chloride at the end of the experiment, COS_{paw} and COS_{flow} were reproduced during manual cardiac massage without any contact with the lung parenchyma (step 2, Fig. 2). These artificial COS immediately disappeared when PA was clamped during cardiac massage.

The results observed during baseline and protocol steps 1 and 2 with an open chest conditions are presented in Table 1. Compared to baseline values, COS_{paw} amplitude decreased to 50 % and COS_{flow} amplitude decreased to 37 % when heart and lungs were in close contact. When COS amplitude was compared between baseline and no contact, COS_{paw} decreased to 59 % and COS_{flow} decreased to 43 % in step 2 (all $p < 0.05$). We found a small but significant difference in COS_{paw} amplitude of 18 % between close and no contact.

Hemodynamic data during closed and open chest conditions steps 1 and 2 are presented in Table 2. In general, the hemodynamic state remained stable during baseline measurements and protocol steps 1 and 2. We found a small but significant decrement in SV when no contact was compared with baseline. MPAP was 19 % higher in close contact and 12 % higher in no contact when compared with baseline ($p < 0.05$). We found no correlation between SV and COS amplitude in any protocol step.

4 Discussion

In this study we provided stronger evidence supporting that COS_{paw} and COS_{flow} are mainly produced by transmission of pulmonary artery pulsatility. Interrupting pulmonary blood flow after clamping the pulmonary artery eliminated COS_{paw} and COS_{flow} irrespective of the degree of contact between the beating heart and surrounding lungs. Additionally, manual heart massage during cardiac arrest reproduced artificial COS even when heart was lifted and maintained outside the thorax but disappeared after clamping the pulmonary artery. On the contrary, although

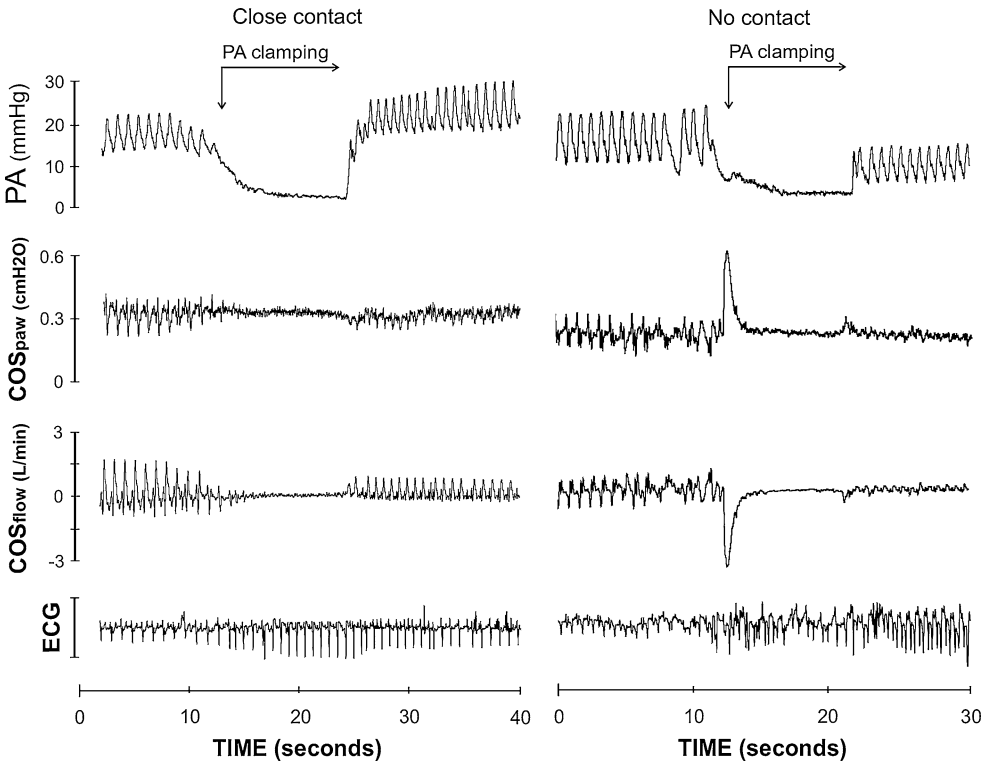

Fig. 1 Effect of pulmonary artery clamping on cardiogenic oscillations. Two examples of the effect of pulmonary artery clamping on cardiogenic oscillations during close and no contact conditions. Immediately after clamping the *PA* pulmonary artery pulses, COS_{paw} and COS_{flow} disappear. After releasing the clamp PA pulsatility and COS reappear concomitantly. Note the corresponding reduction in amplitude of both PA pulsatility and COS after clamp release in the no contact condition

there was a small decrease in COS_{paw} amplitude during no contact, the physical transmission of heartbeats onto the lungs has a minor contribution in COS_{paw} and no contribution in COS_{flow} origin as both COS persisted after total isolation between heart and lung parenchyma (step 2). These results support our previous findings in human patients and add new evidence regarding the origin of COS answering questions ethically impossible to formulate in humans.

Cyclic changes in intra-thoracic blood volume have been related to COS. This has been explained by a temporal imbalance between the amount of blood ejected outside the thorax by the left ventricle and the inflow received by the right ventricle [11, 16]. However, as the lungs are the main organs within the chest cavity, the pulsatile nature of pulmonary blood flow can be enough to explain the cyclical changes in intra-thoracic volume irrespective of any imbalance between left and right heart volumes. Many authors have demonstrated this pulmonary pulsatility at the pulmonary capillary level [19–21] and similar pulsations have been seen during whole plethysmography [22] or during the recording of nitrous oxide uptake [23]. Furthermore, Montmerle and Linnarsson [16] described how in healthy volunteers COS_{flow} amplitude increased in response to sudden increases in intra-thoracic blood volume after inflation of an anti-gravity suit. They concluded that changes in intra-thoracic volume during the cardiac cycle participate in the genesis of COS_{flow}.

Our previous findings in humans demonstrated a close relationship between COS and the amount of pulmonary blood flow [18]. COS_{flow} and COS_{paw} doubled their amplitudes when pulmonary blood flow was restored from very low to normal values during cardiopulmonary bypass weaning. Data were obtained with a beating heart under minimal heart-lung contact conditions with the sternal retractor in place. The present results support these findings obtained in humans. We observed COS_{paw} and COS_{flow} in presence of a normal pulmonary blood flow and pressure but with the beating heart totally isolated from the lungs as in step 2.

The right heart not only creates the pulmonary blood flow but also produces a pulsatile wave that travels along the pulmonary vascular tree [19, 24, 25]. As any mechanical wave, the transmission of this pulsatile wave can occur through different media such as the gas within airways. These transmitted waves can therefore be collected at the airway opening as pressure or flow waveforms with the use of sensors with sufficient sensitivity.

Dahlstrom et al. [15] supported this explanation in an isolated human lung preparation. They reproduced COS in the nitrogram (COS_{N2}) artificially created in the pulmonary vasculature in the absence of any cardiac activity. These data are similar to the ones we observed during the cardiac massage phase of our protocol (Fig. 2) and confirm that the transmission of a mechanical wave, such as the PA pulse pressure wave, is an important factor in the origin of COS_{paw} and COS_{flow}.

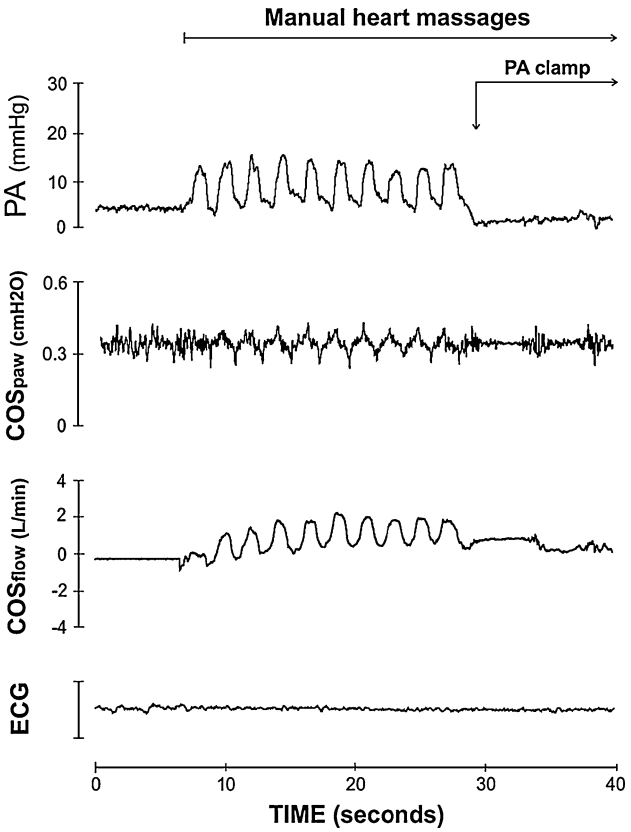

Fig. 2 Effect of manual heart massage and PA clamping on cardiogenic oscillations during cardiac arrest. Of notice is the increase in amplitude in COS_{flow} as compared with all other conditions. COS disappear immediately after PA clamping (*arrow*). *PA* pulmonary arterial pressure, COS_{paw} cardiogenic oscillations in airway pressure and COS_{flow} cardiogenic oscillations in airway flow. Electrocardiogram (ECG) shows a ventricular fibrillation during this protocol step

Table 1 COS amplitude during protocol steps

Parameter	Closed-chest	Open-chest	
	Baseline	Close contact	No contact
COS_{paw} amplitude (cmH_2O)	0.70 ± 0.08	0.35 ± 0.03*	0.29 ± 0.06*†
COS_{flow} amplitude (L/min)	0.51 ± 0.06	0.32 ± 0.05*	0.29 ± 0.04*

Baseline measurements were obtained during closed-chest condition. After opening the chest, measurements were performed with and without contact between heart and lungs (protocol steps 1 and 2 respectively)

COS_{paw} cardiogenic oscillations in airway pressure signal, COS_{flow} cardiogenic oscillations in airway flow signal

* Compared to baseline; $p < 0.05$. † Compared close contact versus no contact; $p < 0.05$

The above data are in opposition to the ones reported by Fukuchi et al. [3] who showed that COS_{N2} persisted from the right middle lobe when its PA branch was blocked by inflating the balloon of a Swan-Ganz catheter. It is however not easy to fully evaluate Fukuchi's findings because they performed measurements in only one animal, did not provide any additional hemodynamic data and did not confirm that the vascular occlusion and N_2 sampling corresponded to the same lobe. Furthermore, an important difference between Fukuchi's findings and the data presented here is that we have studied COS as pure mechanical waves (COS_{paw} and COS_{flow}) while Fukuchi's analyzed COS_{N2}. COS_{N2} not only depends on the molecular N_2 transport of the mechanical wave but also on N_2 concentration stratification among acini due to gravity and lung asymmetry. Thus, possible explanations for their opposing findings could be related to: (1) the pulsatility from right upper and lower lobes made a churning effect on the middle lobe (the one studied), transporting N_2 molecules in direction to the sampling catheter. (2) The bronchial vessels, supplied by the aorta, which run alongside the bronchi, caused a churning effect on the right middle lobe [26]. (3) the bronchio-pulmonary anastomoses found at the alveolar level could maintain pulsatility in the right middle lobe distal to the vascular obstruction in the studied dogs [26, 27]. Therefore, COS_{N2} could be observed despite blood flow interruption in this lobe. In our study pulmonary artery was clamped at its origin thus, pulsatility was stopped in the entire lung parenchyma.

Table 2 Hemodynamic data

Parameter	Closed-chest	Open-chest	
	Baseline	Close contact	No contact
CO (l/min)	2.4 ± 0.3	2.3 ± 0.2	2.3 ± 0.1
SV (ml)	40 ± 6	37 ± 8	36 ± 7*
HR (bpm)	62 ± 9	61 ± 7	66 ± 8†
MAP (mmHg)	83 ± 16	80 ± 16	70 ± 15*†
MPAP (mmHg)	21 ± 3	26 ± 3*	24 ± 4*
CVP (mmHg)	10 ± 2	10 ± 1	11 ± 2

Baseline measurements were obtained during closed-chest condition. After opening the chest, measurements were perfomed with and without contact between heart and lungs (protocol steps 1 and 2 respectively)

CO Cardiac output, *SV* stroke volume, *HR* heart rate, *MAP* mean systemic arterial pressure, *MPAP* mean pulmonary arterial pressure and *CVP* central venous pressure

* Compared to baseline; $p < 0.05$. † Compared close contact versus no contact; $p < 0.05$

4.1 Limitations

We acknowledge that the results obtained in this experimental setting with an open chest and an open pericardium are not easy to extrapolate to humans with an intact thorax. Despite this non-physiological settings can affect COS

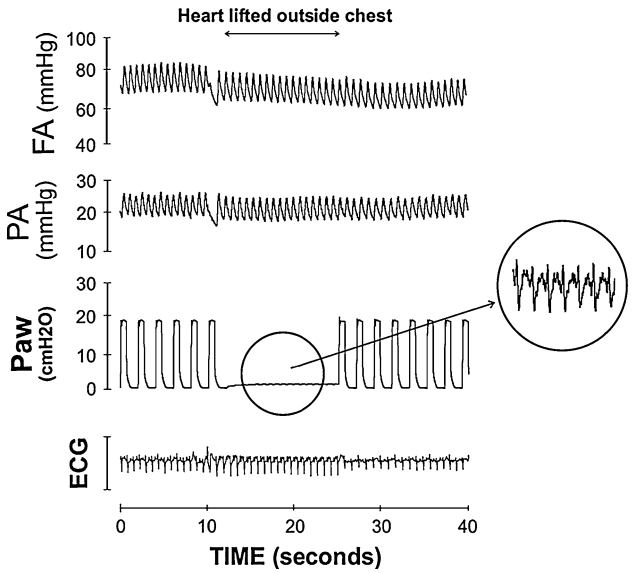

Fig. 3 Femoral and pulmonary arterial pressures during open-chest and no contact condition (step 2). The figure illustrates the minimal impact on systemic arterial and pulmonary artery pressure amplitude during this stressful protocol condition, when the heart was lifted out of the thoracic cavity and during an expiratory breath hold. *FA* systemic femoral artery pressure, *PA* pulmonary arterial pressure and *Paw* airway pressure. *Circle* shows the zoom view of cardiogenic oscillations in the airway pressure signal. Despite this aggressive maneuver, electrocardiogram (ECG) confirmed heart activity while PA and FA showed pulmonary and systemic arterial pressures values within a normal range

amplitude in many complex ways, we believe that the information derived from the extreme maneuvers performed during the presented protocol confirm our hypothesis.

One limitation of our study is that we were unable to acquire thermodilution CO and derived SV data during the 15 s breath-hold maneuver. This means that CO and SV were calculated beat-by-beat using the contour analysis of systemic arterial pressure waveforms (PiCCO2, Pulsion, Germany); a measurement that cannot provide accurate reliable information during these extreme protocol maneuvers. Therefore, the hemodynamic data collected immediately before breath-holds (Table 2) could not represent the real hemodynamic status during this particular moment. We could observe that during the end-expiratory breath-hold continuous pulse contour cardiac output did not change nor did the hemodynamic status *per se*, as witnessed by stable values of invasive systemic and pulmonary arterial pressures. Furthermore, this limitation may have been of relevance only for the data collected in step 2 as lifting the heart outside the chest could be associated with a more important hemodynamic effect. However, we did not observe any major hemodynamic impairment during this maneuver in the studied animals. Figure 3 illustrates an example of how continuous systemic and pulmonary pressure behaved during step 2 in one of the animals.

4.2 Conclusions

The reported data in this study confirm and reinforce that the genesis of COS_{paw} and COS_{flow} is mainly related to the transmission of pulmonary pulsatility i.e. the cyclic changes in pulmonary blood flow and pressure induced by the right heart activity. Our results, obtained in an open chest condition, suggest a minor contribution of the physical transfer of the heart motion to the surrounding lung parenchyma to the origin of COS_{paw} and COS_{flow}.

The fact that COS represent the pulmonary artery pulse wave transmission may have interesting implications in lung monitoring and cardiopulmonary interactions. These implication must be analyzed in future studies.

Acknowledgments We would like to thank Dr. Carlos Castilla head of the experimental laboratory of the Fundación Jiménez Díaz for his highly professional assistance to conduct the experiments and invaluable help in animal preparation and management and Mrs. Pilar Manzano for her skillful assistance. This study was supported by a grant of the "Fondo de Investigaciones Sanitarias" FIS-PI07136.

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