

Influence of Surrounding Structures on Parallel Volume Determinations by Conductance Catheter

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When left ventricular blood volume is evaluated by the conductance catheter technique, an important fraction of the injected current "leaks" into the surrounding tissues giving rise to a parallel volume (V_p). To study the influence of the thoracic structures on V_p , 5 anesthetized dogs were studied during (a) closed thorax, (b) open thorax, (c) reclosed thorax without pericardium, (d) open thorax without pericardium, (e) replacing the pericardium with a nonconducting bag. Intraventricular volume and pressure, surface ECG, and V_p (determined by saline dilution technique) were monitored during each condition. A conductance diagram was used to represent the behavior of the structures within the thoracic cage, including cardiac muscle, pericardium, and also other structures (OS) such as lung, skeletal muscle, and bone tissues. Under conditions (a) and (b), V_p was not significantly different ($p < 0.005$); thus, changes in OS did not much modify V_p . When the pericardium was removed, the average V_p values for conditions (c) and (d) were smaller than those for (a) and (b) ($p < 0.005$). Apparently, because of good conductivity, the pericardium would favor the passage of current. Finally, when the fifth condition (e) was compared with (a) or (b), the differences were highly significant ($p < 0.005$), while the differences between (e) and either (c) or (d) were less significant and more variable ($p < 0.05$). Duncan's multiple comparisons by pairs confirmed the previous statements. The relative fraction of the total measured

volume through the surrounding tissues ($p\%$) systematically decreased as we moved from condition (a) to (e). Condition (e) decidedly forced a concentration of current lines through the myocardium. In conclusion, the pericardium is responsible for a significant fraction of the loss, which should be separated out from that due to the myocardium and OS. The latter would contribute to V_p with not more than about 10%.

Key words: saline dilution technique; impedance technique; left ventricular blood volume; pericardium; volume calibration.

INTRODUCTION

The accuracy of intraventricular conductance volumetry is strongly marred by current leaking through surrounding anatomical structures, which give rise to an undesirable phantom volume usually called the *parallel volume* (V_p). No studies, so far, have tried to quantify the main pathways and respective fractions being derived through them.

Baan *et al.* (1984) introduced the hypertonic saline procedure to estimate globally such parallel correcting V_p value. Steendijk *et al.* (1993a,b), in turn, analyzed the myocardial mass influence, having found resistivity changes under ischemic conditions. Volume changes in the adjacent cardiac cavities might affect V_p , especially those originated in the right ventricle. Burkhoff *et al.* (1985) reported a minimal influence of the right ventricle in the isolated ejecting left ventricle, all through the cardiac cycle, on volume measurements with the conductance catheter. Afterwards, Amirhamzeh *et al.* (1994) concluded that right ventricular volume has important influences on V_p in arrested hearts.

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In isolated heart experiments, Suga and Sagawa (1974) inserted a balloon in the left ventricle to cover the endocardium. The balloon was filled with a known volume of fluid with resistivity near blood resistivity. In turn, the preparation was submerged in a glass container filled with a solution of resistivity near myocardial resistivity (which they called “surrounding resistivity”). The latter was changed in steps. Their results showed that the parallel volume increased as the surrounding resistivity decreased, but they did not analyze the possible distribution in other anatomical structures.

Kun and Peura (1994) simulated the whole system and so modified blood and myocardial conductivities. Besides, they added the lungs as a third component. For large hearts (volumes greater than 35 mL), these authors reported a decrease of 7 mL in the parallel volume for every 100 mL of increase in ventricular volume, probably because of the smaller magnitude of the electrical field in surrounding structures.

There were also some attempts to evaluate the percentage of current lost when frequency was changed (McKay *et al.*, 1984). Within the 2–100 kHz range, blood resistivity is constant and about $150 \Omega \cdot \text{cm}$ (Schwan 1983), and the myocardial resistivity is about $400 \Omega \cdot \text{cm}$ at 20 kHz or more (1:2.7 ratio). However, at frequencies in the order of 2 kHz, the latter parameter lies above $1,000 \Omega \cdot \text{cm}$ (Zheng and Webster, 1984), meaning that leak through this tissue is smaller than expected (1:6.7 ratio). Gawne *et al.* (1987) made use precisely of this property (the higher the frequency, the lower the resistivity) to determine V_p . However, they noticed that the percentage differences of resistivity between the two frequencies were rather small. Recently, White *et al.* (1998) confirmed that the dual frequency technique can not be used as an alternative to the dilution technique (Baan *et al.*, 1984).

Herein, we evaluated V_p alterations under different experimental conditions trying to separate out the relative influences of cardiac tissue (CM), pericardium (PC), and the other (thoracic) structures (OS), such as the lungs, skeletal muscle and bone, the latter three lumped in one. The question is to determine the practical importance of the leakage through the different surrounding structures. In addition to a general analysis of the method, knowing the leakage distribution would improve the interpretation of the errors when the method is applied in different pathological situations that involve any of the considered structures. This is of particular importance in the operating room, since some potential influences on V_p (such as free fluid, atrial, vascular, and lung volumes) often undergo substantial changes during cardiac surgery (Amirhamzeh *et al.*, 1994).

METHODS

Experimental Protocol

Five mongrel dogs (between 18 and 28 kg of body weight) were used following the animal treatment recommendations of the American Physiological Society. Anesthesia was induced with morphine chlorohydrate (15 mg/kg, s.c.) followed by sodium pentobarbital (15 mg/kg, i.v.), thereafter maintaining it to effect with additional amounts as required. The animals were connected to a positive pressure electronically controlled respirator. Right femoral vein and artery were cannulated. The first one was used to administer drugs and physiological solution while the second was to measure intraventricular pressure (IVP) (with a micro-tip transducer, model SPC-360, Millar Instruments, Houston, Texas) and to collect blood samples. An eight-electrode conductance catheter was introduced via the left carotid artery. These signals were processed by a custom-made conductance system of 14 kHz (Herrera *et al.*, 1986) to finally derive intraventricular volume (IVV). Two main recording catheters (conductance and micro-tip pressure transducer) were positioned in the left ventricle. To assess the relative position of the catheter, intraventricular pressure versus conductance signals (PG loops), for all catheter sections, were simultaneously recorded when the catheter was slowly introduced into the cavity. The direction of rotation of the PG loops is a unequivocal indicator for the section position. When the PG loops rotate clockwise, the monitored section is still outside the ventricle but when they rotate counterclockwise, the section is inside the ventricle. If the PG loops look like an eight figure, the section is at the level of the aortic valve. Artifacts show up when the electrodes touch the endocardium or when the catheter bends. Catheter longitudinal movements are easily detected in the basal section PG loops. Sometimes, the catheter tip touches the cardiac apex or may even get stuck in it eliciting ectopic beats, which appear as bizarre conductance complexes or PG loops. Slightly and gently withdrawing the catheter usually takes care of such situation. Now and then, the catheter has to be partially or even fully removed from the cavity and, thereafter, carefully reinserted until satisfactory signals are obtained. Always, the final position of the catheter was verified at postmortem examination.

Hypertonic saline solution was injected with a Swan-Ganz catheter located either in the pulmonary artery or in the right atrium accessed to via the jugular vein. Besides, surface ECG, blood gases (with an AADE Gas Analyzer, model CCI-Q, Buenos Aires, Argentina), hematocrit, and rectal body temperature were constantly monitored. Blood

conductivity, σ_b , was determined by means of a conductivity cell immersed in a thermostatic bath, the latter to ensure temperature stability (Herrera *et al.*, 1999b).

The determination of V_p followed the well-known basic experimental protocol described by Baan *et al.* (1984) modifying it with the introduction of five different and sequential conditions:

- (a) Intact pericardium (+P) and closed thorax (CT)
- (b) Intact pericardium (+P) and open thorax (OT)
- (c) Heart without pericardium (−P) and reclosed thorax (RCT)
- (d) Heart without pericardium (−P) and open thorax (OT)
- (e) Replacing the pericardium with a nonconducting bag (BA) and reclosed thorax (RCT)

The first one, (a), acted as reference or control. Thoracotomy was performed by a sternal medial incision, while a plastic bag around the heart replaced the pericardium in condition (e).

Data Collection

All signals (ECG, G_t (intraventricular total conductance) and IVP) were digitized at a sampling rate of 200 Hz with a LAB PC+ acquisition board. The acquisition software was developed under LabVIEW (version 3.1.1, National Instruments) processing the data off-line in an IBM compatible PC. Heart rate (HR), left ventricular end-systolic pressure (LVESP), and left ventricular end-diastolic pressure (LVEDP) were obtained and used as control parameters. For the latter, the criterion was to choose as end-diastolic that pressure immediately preceding a minimum of 100 mmHg/s growth in dP/dt . The end-systolic points were determined by the maximum pressure/volume relationship during ejection of the left ventricle.

The conductance catheter method relates the time-varying intraventricular total conductance G_t and the blood ventricular volume G_b by a simple formula

$$V_b = (1/\alpha) \cdot (L^2/\sigma_b) \cdot (G_t - G_p) \quad (1)$$

where α is a dimensionless factor, σ_b is the blood conductivity, L is the interelectrode distance and G_p is the parallel conductance. Assuming ideal conditions, that is, uniform intracardiac electric field, the α -factor was assumed as equal to 1.0, as in previous experiments and reports (Baan *et al.*, 1984; Burkhoff *et al.*, 1985; Kass *et al.*, 1989), thus

$$V_b = [(L^2/\sigma_b) \cdot G_t] - [(L^2/\sigma_b) \cdot G_p] = V_t - V_p \quad (2)$$

where V_t and V_p are the total uncorrected intraventricular volume and the parallel volume, respectively.

Thereafter, a mean value of G_t ($G_{t\text{mean}}$) was calculated for each beat by means of

$$G_{t\text{mean}} = [(G_{es} + G_{ed})/2] \quad (3)$$

where G_{es} and G_{ed} are the end-systolic and end-diastolic intraventricular conductances, respectively. Then, the mean intraventricular total volume $V_{t\text{mean}}$ was calculated by

$$V_{t\text{mean}} = [(L^2/\sigma_b) \cdot G_{t\text{mean}}] \quad (4)$$

This procedure was done over 10–20 stable beats and, thereafter, an average of those mean values was calculated to finally get the uncorrected volume (Fig. 1(a)).

To determine an average V_p , we performed at least four saline dilution maneuvers in each experimental condition (see Experimental Protocol). Figure 1(a) displays the typical temporal course of the saline dilution maneuver. The upper channel is G_t and the lower one is IVP. For each maneuver, G_p was calculated following Baan's procedure (Baan *et al.*, 1984) including the conditions proposed by Herrera *et al.* (1999a,b), as illustrated in Fig. 1(b). It amounts to calculating, by extrapolation of the regression line, $G_{es} = f(G_{ed})$, its intersection with the identity line, $y = x$, which yields G_p . From the latter, V_p is obtained by means of a known equation,

$$V_p = (L^2/\sigma_b) \cdot G_p \quad (5)$$

The average of the four determinations, with their respective standard deviations SD, was used to assess the differences among the above-mentioned conditions. Finally, a single value of the actual intraventricular blood volume V_b was calculated for each situation by

$$V_b = V_{t\text{mean}} - V_p \quad (6)$$

Surgical Preparation

The thorax was opened by means of a medial sternotomy, always monitoring the signals from the five sections of the conductance catheter. Thereafter, the heart was uncovered by a pericardial incision along the basal–apical axis to form a temporary pericardial cradle held on each side with two Kocher hemostats to the sternal skin borders. While the heart was being held by the cradle, three acetate bands were passed between the ventricles and the

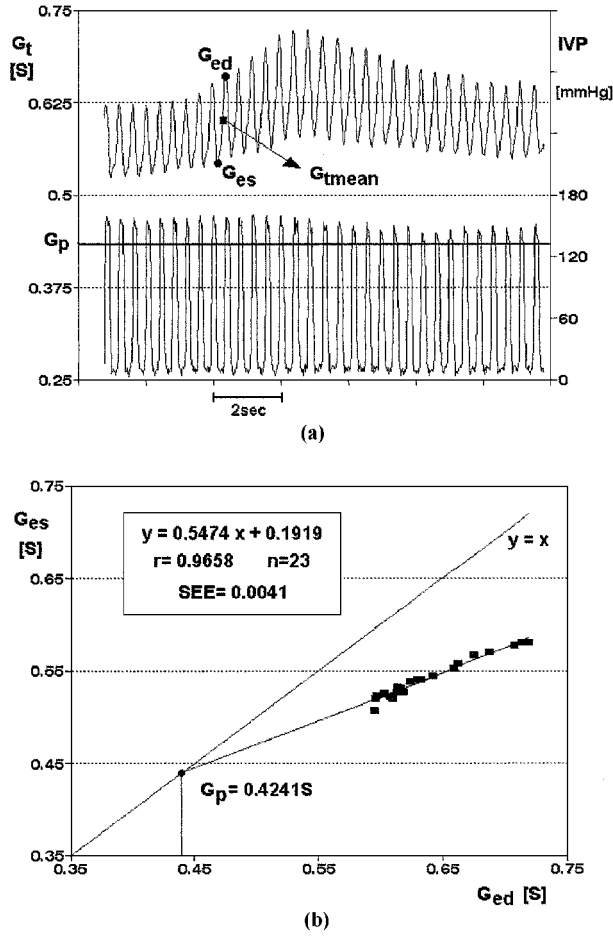


Figure 1. (a) Conductance G_t and intraventricular pressure IVP during a dilution maneuver injecting 1.5 mL of 6 M NaCl in the pulmonary artery. Upper channel calibration is on the left vertical axis and lower channel calibration on the right axis. Dog #003, body weight = 22 kg, $\sigma_b = 0.40$ S/m. $G_p = 0.4241$ S. G_{es} , G_{ed} and G_{tmean} are defined in Data Collection section. (b) Baan's procedure. End-systolic conductance G_{es} vs. end-diastolic conductance G_{ed} (experimental data) during the maneuver, the regression of this data and the intersection with the identity line ($y = x$) are represented. Linear regression equation and the correlation coefficient (r), number of data (n) and standard error of the estimate (SEE) are also shown.

pericardium to strap the former as a horse belly by its saddle. The acetate bands were sewn to the sternal borders and the hemostats removed while cutting the pericardium free, as necessary and carefully, so dropping it into the thoracic cavity.

One of the acetate bands had attached to it a many-folded plastic sheet lightly held on the two lateral extremes by cellophane tape. Removing the latter allowed the spreading of the plastic sheet and, thus, bringing out the sides so that a bag was formed around the heart. Only

the upper portion was partially exposed, which, in turn, was finally fully covered by another small piece of plastic. Ultimately, the thorax was reclosed by bringing together the two sternal sides holding them with three or four hemostats. This procedure is relatively simple and, if carefully done by a skillful operator, does not produce any damage neither to the heart nor to the great vessels. The phrenic nerve was preserved in all cases. However, animals were always connected to a mechanical respirator.

Conductance Diagram

In order to determine the relationship between the total conductance derived volume and the actual ventricular blood volume, a simple electric diagram is presented which includes lumped conductances for each of the impedance components in the thorax. A conductance catheter within an ideal ventricular cavity, where the electric current lines remain confined to its blood content alone, will produce a volume reading only dependent on the electrode geometry, the blood conductivity, and the cavity dimensions. Note that we assumed α -factor = 1 (homogeneous field). This situation can be represented by a single conductance G_b , the blood conductance. This is a leakless circuit and, thus, conductance G_b is proportional to the intraventricular blood volume, that is,

$$V_b = (L^2/\sigma_b) \cdot G_b \quad (7)$$

In the real system there are leaks, the measured volume V_t lumps the actual blood volume V_b in the cavity, and the phantom parallel volume V_p . A simplified electric diagram that represents this situation consists of two conductances: the blood conductance volume G_b within the ventricle and the conductance of the surrounding tissues G_p , both in a parallel circuit (Fig. 2(a)). In this diagram, the total conductance is calculated by

$$G_{tmean} = G_b + G_p \quad (8)$$

Therefore,

$$V_{tmean} = V_b + V_p = (L^2/\sigma_b) \cdot (G_b + G_p) \quad (9)$$

Since we are interested in the relative fractions of the total measured volume that represents the leak through the surrounding tissues, $p\%$ is defined as

$$p\% = 100 \cdot [V_p/(V_p + V_b)] = 100 \cdot (V_p/V_{tmean}) \quad (10)$$

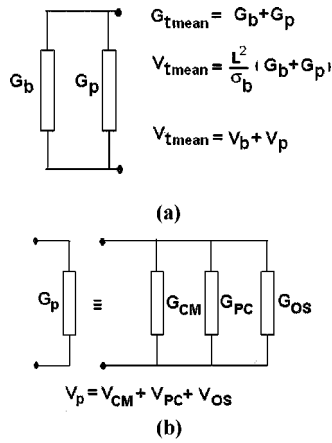


Figure 2. (a) Schematic diagram of the intact heart with leaks (+P at CT). G_b , left ventricle blood conductance. G_p , total parallel conductance. (b) The whole diagram for parallel conductance and its components. Each branch contributes with one specific parallel conductance, which is responsible for the corresponding parallel volume. G_{CM} , G_{PC} , G_{OS} , are partial conductances that represent myocardium, pericardium, and other structures, respectively (see text).

Besides, the parallel conductance G_p can be considered as the sum of the partial conductances contributed by the other thoracic structures, that is,

$$G_p = G_{CM} + G_{PC} + G_{OS} \quad (11)$$

representing, respectively, the myocardium (CM), the pericardium (PC), and the lungs and the rest of the tissues within the thoracic cage (OS). Therefore, the diagram for the whole leak-system can be depicted as shown in Fig. 2(b), where

$$V_p = V_{CM} + V_{PC} + V_{OS} \quad (12)$$

The latter is modified by the different situations described in the Experimental Protocol. With the intact animal (+P and CT), the total V_p is evaluated including all structures (G_{CM} , G_{PC} , and G_{OS}). When the chest is open (+P and OT), a redistribution of the current lines is likely to occur because of changes in G_{OS} . When the pericardium is removed (-P and RCT), the current lines previously derived through it are now bypassed through the myocardium and other structures (G_{CM} and G_{OS}). Without pericardium (-P at OT) and because of the open thorax, other changes are produced in G_{OS} ; this situation shows the effect of muscle and bone tissues. Finally, in the BA situation, the plastic bag (a good insulator) should concentrate most of the lines through the myocardium making $G_p = G_{CM}$.

All measurements were carried out during expiratory apnea.

RESULTS

Table 1 summarizes the hemodynamic parameters before (+P) and after removal of the pericardium (-P), at reclosed thorax (RCT) and at open thorax (OT) as well, and finally with the isolating bag (BA) and reclosed thorax (RCT). No significant changes were produced in HR and LVESP. The only parameter that showed significant changes with respect to the control condition (+P and CT) was LVEDP, especially with the plastic bag ($p < 0.005$). LVEDV and SV did not change significantly between +P and -P.

Table 2 displays V_p values and their standard deviations SD for each animal and in each condition. The last row presents the average V_p and the corresponding SD for each condition for all animals. The paired t test did not show significant differences between conditions (+P and CT) and (+P and OT) in four of the five animals. Similar results were found when we looked into conditions (-P and RCT) and (-P and OT).

The average parallel volume V_p with pericardium—in (+P and CT) and (+P and OT)—was always higher than without pericardium—in (-P and RCT) and (-P and OT)—it decreased from 18 to 27% (see Table 2). Condition (BA and RCT) produced a significant decrease ($p < 0.005$, paired t test) with respect to (+P and CT), showing an average fall of 40%. The same condition (BA and RCT), instead, with respect to (-P and RCT), displayed an average fall of 18% ($p < 0.05$, paired t test). Moreover, and accepting the risks of averaging out on an interindividual basis, the average V_p in Table 2 indicates an almost steady falling off of the phantom volume as we moved from condition (+P and CT) to condition (BA and RCT).

To obtain a stronger and better assessment of the differences, variance analysis was also applied. For a 0.01 level of significance, an F value higher than 4.89 is required (see any F -statistics table) after determining the proper degrees of freedom. Our five dogs yielded values between 15.9 and 95.8, that is, well above such limit, meaning highly significant differences for the average V_p . However, there is a lack of specificity in this statement. Thus, it is useful to carry out Duncan's multiple intervals test (Montgomery, 1989), which compares each experimental situation with respect to another one (Table 3). Only those comparisons considered more relevant were included in the table. For example, open thorax with intact

Table 1. Hemodynamic Parameters in Different Conditions

	+P		-P		BA
	CT	OT	RCT	OT	RCT
	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)
HR (beat/min)	100 (25)	104 (23)	106 (26)	99 (27)	113 (23)
LVESP (mmHg)	150 (15)	144 (23)	148 (16)	144 (19)	151 (17)
LVEDP (mmHg)	7 (2)	9* (4)	10* (3)	10* (4)	11** (5)
LVEDV (mL)	35 (21)	34 (18)	33 (16)	34 (17)	38 (23)
SV (mL)	14 (9)	12 (8)	14 (7)	14 (9)	16 (8)

Note: +P, intact pericardium; -P, without pericardium; CT, closed thorax; OT, open thorax; RCT, reclosed thorax; BA, with a nonconducting bag. Mean and standard deviations (SD) are given. Values of * $p < 0.05$ and ** $p < 0.005$ were obtained vs. the control condition +P at CT. HR, cardiac frequency; LVESP and LVEDP left ventricular end-systolic and end-diastolic pressures; LVEDV, left ventricular end-diastolic volume; SV, stroke volume.

pericardium was not compared with any of the other situations except the reference condition (a). Each cell shows whether the particular comparison described above in row 1 is significant or not (YES or NO). For example, in dog #1, parallel volume obtained with a closed thorax and an intact pericardium (+P and CT) was not significantly different from the value given by the open thorax and intact pericardium condition (+P and OT).

Leakage Through the Surrounding Structures $p\%$

The average percental loss ($p\%$) (Eq. 10) obtained with the conductance diagram is represented in Fig. 3 for the five animals and all conditions. Once more, and in agreement with Table 2, there is a clear and steady decrease from (+P and CT) to (BA and RCT). This loss is on the average lower without pericardium than with it (52 vs. 63%, respectively). However, the most striking

effect shows up when comparing the isolating bag condition against control (33 vs. 63%, average values).

DISCUSSION

The parallel conductance volume depends obviously on the structures surrounding the left ventricular cavity. Their temporal-spatial positions and the relationships between their conductivities and blood conductivity define the current line distribution in the thoracic cage. This topic is approached by our study applying controlled and specific conditions, which modify such distribution.

The first assumption, like other authors, is to consider that the electric field, in which these structures are, is homogeneous (α -factor = 1). Szwarc *et al.* (1995) corrected the conductance-derived volume applying V_p that does not vary between systole and diastole and then they applied an α -factor between 0.62 and 0.81 using the magnetic

Table 2. Parallel Conductance Volume (Mean and Standard Deviations), V_p and SD, under Different Experimental Conditions

Subject	+P		-P		BA
	CT	OT	RCT	OT	RCT
	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)
#001	40.4 (2.1)	40.9 (2.8)	37.0* (1.3)	32.8* (1.9)	14.8** (2.4)
#002	31.2 (1.0)	32.3 (2.1)	18.4** (0.2)	21.4** (0.5)	23.6** (1.4)
#003	31.1 (3.4)	32.5 (0.6)	26.0* (0.7)	32.9 (0.3)	26.0* (2.6)
#004	30.1 (0.6)	20.5** (0.8)	15.5** (1.6)	15.4** (2.5)	15.05* (0.9)
#005	24.3 (2.1)	24.4 (0.9)	17.5* (1.7)	21.1 (1.2)	14.6** (2.3)
Average V_p (mL)	31.4 (5.8)	30.1 (7.1)	22.9 (8.8)	24.7 (7.8)	18.8 (5.5)

Note: Values of * $p < 0.05$ and ** $p < 0.005$ were obtained vs. the control condition +P and CT. +P, pericardium intact; -P, without pericardium; CT, closed thorax; OT, open thorax; RCT, reclosed thorax; BA, with a nonconducting bag.

Table 3. Multiple Interval Duncan's Test

Subject	+P & CT vs. +P & OT	+P & CT vs. -P & RCT	+P & CT vs. -P & OT	+P & CT vs. BA	-P & RCT vs. -P & OT	-P & RCT vs. BA	-P & OT vs. BA
#001	NO	YES	YES	YES	NO	YES	YES
#002	NO	YES	YES	YES	NO	YES	NO
#003	NO	YES	NO	YES	SI	NO	YES
#004	YES	YES	YES	YES	NO	NO	NO
#005	NO	YES	YES (at $p < 0.05$)	YES	NO	NO	YES

Note: Results, "YES" or "NO" stands for the significance at the 0.01 level except for the marked cell. +P, intact pericardium; -P, without pericardium; CT, closed thorax; OT, open thorax; RCT, reclosed thorax; BA, with a nonconducting bag.

resonance imaging method as "gold standard." When we assume $\alpha = 1$, the values of V_{lmean} obtained are smaller than if we apply values of $\alpha < 1$ and, as consequence, the percentage losses $p\%$ (Eq. 10) obtained would be even higher.

On the other hand, the maneuvers (opening of the thorax or removal of the pericardium) may modify the control parameters. Table 1 indicates that, for example, LVEDP undergoes a significant increase, in agreement with previous reports (Applegate *et al.*, 1990). However, we believe it is more important to keep a constant LVEDV because the latter can seriously affect end-diastolic pressure (Applegate *et al.*, 1992). For example, without the pericardium, there is less constriction and the ventricles tend to a larger filling which, in turn, would change the leak relationship. In our experiments, neither LVEDV nor stroke volume SV showed significant variations.

The results show that opening of the thorax does not induce important changes in the current bypassed by

the surrounding structures (OS) and, hence, in V_p . Recent similar results were described by Szwarc and Ball (1998). They concluded that the fluctuations in conductance catheter-derived volume with ventilation are not caused by any significant changes in V_p . Experience taught that respiratory movements modify the measured volumes because they produce displacements of the catheter, eccentricity, torsion, or contact of the electrodes with the ventricular wall. To avoid these undesirable situations, the saline dilution maneuvers were performed in apnea.

Perhaps, it is better to say that changes in G_{OS} slightly modify the leakage effect of the myocardial and pericardial conductances (G_{MC} and G_{PC}). In fact, the open thorax should favor conduction through those structures (MC and PC). Finally, OS contributes to the parallel volume only with 10% or less of its value; this is shown in Fig. 3.

Amirhamzeh *et al.* (1994) added substances (such as saline, electrode gel, or ICD patches) between the pericardium and the myocardium. With saline, they observed an increment in the volume measured by conductance, thus, implying that there are current lines leaking out of the myocardium. The other structure that could modify V_p is the pericardium. Although many authors evaluated the mechanical characteristics of this membrane (Lee and Boughner, 1981; Lee *et al.*, 1987; Mirsky and Rankin, 1979; Wiegner and Bing, 1981), the authors of this paper did not find any work that estimates the pericardial conductive characteristics. Burkhoff *et al.* (1985) refer to the pericardial tissue as highly conductive (with a superficial conductivity higher than 2 S/m^2). We guess that surface and transmembrane conductivities should be distinguished and that both are relatively high as compared with blood conductivity. It is, thus, an open question still awaiting answer.

The average V_p values ($\approx 25 \text{ mL}$) obtained by Burkhoff were considerably lower than those reported from open thorax dogs with an intact pericardium (70–80 mL) (Kass *et al.*, 1989) and also from intact animals (Boltwood *et al.*, 1989). Burkhoff suggested a possible isolating effect of the pericardial balloon due to the

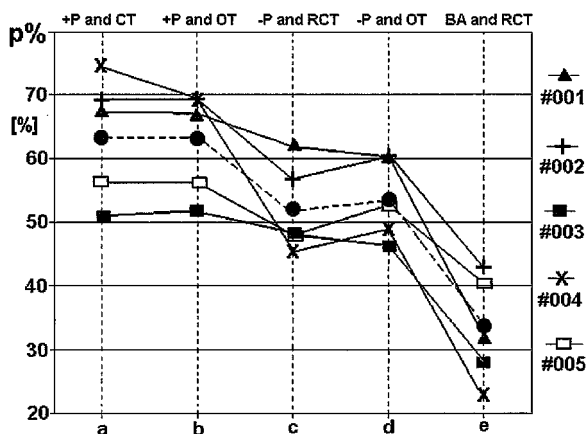


Figure 3. Average percentage loss ($p\%$) for each experimental condition and the averages over the 5 dogs (dash line). +P, pericardium intact; -P, without pericardium; CT, closed thorax; OT, open thorax; RCT, reclosed thorax; BA, with a nonconducting bag. Observe the monotonic decrease as the condition gets more stringent (conductively speaking).

formation of small air pockets between it and the myocardium. Our results suggest a short-circuiting effect of the intact pericardium. Even further, when the pericardium was removed, the current lines, those by-passed by the pericardium, are redistributed through the cardiac muscle ($-P$ at RCT) or through cardiac muscle and other structures ($-P$ at OT) thus leading to a smaller V_p . The conductance diagram has a similar behavior, this means that the losses ($p\%$) decrease and CM+OS are less conductive than the whole system. Hence, the pericardium must be considered a good conductor by-passing a significant number of current lines, without being very much affected neither by the closed nor by the open thorax. To our knowledge, the conductivity relationship between pericardium and myocardium has not either been compared so far.

The plastic bag, condition (BA and RCT), decidedly forced a concentration of current lines through the myocardium; hence, the parallel conductance G_p is equal to G_{CM} . However, muscle fibers display a complex orientation making extremely difficult an analytic solution for the current line distribution (Gawne *et al.*, 1987). Other authors estimated myocardial conductivity changes *in vivo* by means of an impedancimetric technique (Van Oosterom *et al.*, 1979), or modeled the system introducing the anisotropy concept (Steendijk *et al.*, 1993a). The former found an average transmural resistivity of the order of $410 \Omega \cdot \text{cm}$ for frequencies between 10 Hz and 5 kHz, independent on the cardiac cycle and uniform through the myocardial thickness. Besides, that resistivity increases considerably under ischemia or cell necrosis. In another paper Steendijk *et al.* (1993b) attempted to measure transversal and longitudinal myocardial resistivity within the 5–60 kHz range. Values reported in the literature for myocardial resistivity are between 390 and 705 $\Omega \cdot \text{cm}$ (Rush *et al.*, 1963; Schwan and Kay, 1956; Van Oosterom *et al.*, 1979). Here we intended to evaluate the overall myocardial conductance G_{CM} , which not only relates to its conductivity or resistivity but also to its geometry (dimensions). Our results show, in spite of the fact that the electrical field lines are limited to CM, a net decrease of the parallel volume under condition (BA and RCT), with respect to ($-P$ and RCT) and ($-P$ and OT) (see Table 3, and column 4). The current lines are mainly by-passed by the myocardium, which might lead to the conclusion that the bag redistributes the current through structures less conductive than in the normal situation with intact pericardium. Nevertheless, we also found that other anatomical thoracic structures had no significant effect under conditions (a), (b), (c), and (d); thus, the difference should be placed at the pericardium.

CONCLUSIONS

The pericardium has a higher conductivity and by-passes an important fraction of the electrical current lines in the thorax. Ventricular muscle alone (covered with an insulating plastic) is less conductive than the ventricular muscle with intact pericardium. Other anatomical structures (skeletal muscle, bone, and lung tissues) contribute to the parallel volume with no more than 10% of its value.

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