Left ventricular function after plication of the left ventricular free wall in dogs*

Estudo da função ventricular na técnica de plicatura da parede livre do ventrículo esquerdo em cães

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Abstract

Objective: We tested a new surgical technique, the plication of the left ventricular free wall, to reduce left ventricular area and volume and improve left ventricular systolic function, without using a cardiopulmonary bypass.

Methods: Dilated cardiomyopathy was induced in eight dogs by the injection of doxorubicin. Plication of the left ventricular free wall was performed in four dogs with induced cardiomyopathy and in five control dogs. Two dogs were not submitted to surgery. The other two dogs died during the induction phase. Cardiac output, 2-dimensional and M-mode echocardiography, arterial blood pressure and electrocardiography were recorded over a 180-day period. Ambulatory electrocardiography of 24 hours was performed during the first postoperative day.

Results: The cardiomyopathy-induced group had significant improvements in cardiac output, ejection fraction,

shortening fraction and reductions in the left ventricular end-systolic, end-diastolic area and volume after the surgery. One dog died. Electrocardiography and Holter revealed premature ventricular complexes, which improved spontaneously at the first week. The cardiomyopathy-induced dogs that did not undergo surgery deteriorated and died at about 40 days after the induction of cardiomyopathy. Hemodynamic values did not change in the normal dogs submitted to surgery.

Conclusion: The plication of the left ventricular free wall reduced the left ventricular area and volume and improved left ventricular systolic function in dogs with doxorubicininduced cardiomyopathy giving low morbidity and mortality.

Descriptors: Myocardial diseases, surgery. Cardiomyopathy, congestive, surgery. Heart ventricles, surgery. Doxorubicin. Dogs.

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Resumo

Objetivo: Avaliar os efeitos da técnica na função ventricular esquerda em cães hígidos e com cardiomiopatia dilatada induzida pela doxorrubicina.

Método: De 13 cães, oito receberam doxorrubicina até que a fração de encurtamento (FE) fosse menor que 20%. Destes, quatro animais e os cinco não induzidos foram submetidos à plicatura da parede livre do ventrículo esquerdo (PPLVE). Os demais cães não foram operados. Foram avaliados débito cardíaco (DC), pressão arterial, exame físico, eletrocardiografia, sistema "Holter" e ecocardiografia, por 180 dias.

Resultados: Houve redução do volume ventricular esquerdo. Os cães induzidos melhoraram após a operação e a fração de ejeção (FEj) retornou aos valores normais para a espécie. O DC e a FE aumentaram após a operação. Um cão foi a óbito. Nos cães não operados, a FE diminuiu e foram a óbito em torno de 40 dias após a indução; nos cães não induzidos, esta não se alterou. Houve extra-sístoles ventriculares, que se resolveram espontaneamente.

Conclusões: A PPLVE sem circulação extracorpórea reduz o volume ventricular esquerdo e melhora a função cardíaca dos cães com cardiomiopatia dilatada induzida pela doxorrubicina, demonstrando baixa morbidade e mortalidade tardia.

Descritores: Miocardiopatias, cirurgia. Miocardiopatia congestiva, cirurgia. Ventrículos cardíacos, cirurgia. Doxorrubicina. Cães.

INTRODUCTION

Ventricular remodeling, with changes in the geometry and increase in the stress on the ventricular wall and the consumption of oxygen by the myocardium, occurs as a result of the physiopathology and progression of cardiac insufficiency [1,2]. The definitive treatment is heart transplantation however this is not always possible due to the low numbers of donors and other limitations [3].

Recently, a new concept for the treatment of end-stage heart disease was introduced, to reduce the diameter of the remodeled left ventricle (LV) [4]. The operation, named partial ventriculectomy, is based on Laplace's law, in which a reduction of the left ventricle (LV) chamber diminishes the tension on the free wall of the LV and improves its function [4-6]. Although its clinical application is limited by the high rate of mortality during the first postoperative months and high late morbidity [7,8]. An alternative to the resection of a portion of the potentially viable myocardium was suggested, by means of plicature of the papillary muscles, after a small apical incision in the LV. This approach has given good preliminary results [9]. Another technique at LV reduction by plicature of akinetic areas after myocardial infarction induced in rats was described, with an initial improvement in LV function. However, this improvement was not maintained four weeks after the procedure [3]. An apparatus denominated as "myosplint" was also developed to change the shape of the LV, with a reduction of parietal stress and improvement in the LV function in dogs with cardiac insufficiency induced by high frequency pacemakers [2].

Recently, ANDRADE et al. [10] described a technique of plicature of the free wall of the LV (PLVFW) to reduce the LV in dogs, without the necessity of cardiopulmonary bypass (CPB).

The aim of this study was to evaluate the effects of this

technique on the ventricular function in dogs with dilated heart disease induced by doxorrubicin.

METHOD

Experimental Groups

Thirteen crossbreed dogs weighing on average 14.3 kg were utilized. Five were submitted to PLVFW without induction of cardiomyopathy (Group I) and eight received intravenous injections of 30 mg/m² of 0.2% doxorubicin (Adriblastina RD® -Pharmacia & Upjohn Ltda.) every 21 days until the shortened fraction of the LV was less than 20%, corresponding to a cumulative dose of 240 mg/m². Of these animals, four were submitted to PLVFW (Group II) and two were not operated (Group III). The remaining two dogs died during the induction process and could not be included in the study. The animals received care according to the "Guide for the Care and Use of Laboratory Animals" of the Institute of Laboratory Animal Resources, National Research Council, published by the National Academy Press, revised 1996 (http://www.nap.edu/readingroom/books/ labrats/chaps.html).

Clinical Evaluation

A physical examination, blood tests, serum biochemistry, computerized electrocardiogram (ECG) (TEB – ECGPC – Tecnologia Eletrônica Brasileira S.A.) and non-invasive mean blood pressure were performed one day before the operation and at one, two, seven, 15, 21, 30, 60, 90, 120, 150 and 180 days after the procedure. Continuous electrocardiography (Holter System; SpaceLabs – mod. 90208) was performed in the first 24 postoperative hours. Echocardiography (Pie Medical – mod. Pandion Vet) was performed in the same time intervals, except on the first postoperative day. The mean diameter and area of the LV at diastole and systole were obtained and the shortened fraction (EF) was calculated

using the following formula: EF = {[(DLVd-DLVs)/DLVd] x 100}, where DLVd = internal diameter of the LV at diastole and DLVs = internal diameter of the LV at systole. The left ventricle volumes at diastole (VLVd) and at systole (VLVs) were obtained using the formula { $V = 0.85 \times [(A)^2/L]$ } (where V = left ventricle volume, A = area of LV and L = long axis of the LV). The ejection fraction of the left ventricle (EjF) was obtained by the ratio between the diastolic and systolic volumes and the diastolic volume multiplied by 100 [11].

The cardiac output was measured by thermo-dilution [12] before and after the plicature, with the thorax closed.

Anesthetic induction and maintenance

Anesthesia was induced with propofol (Propofol® - Cristália Produtos Químicos e Farmacêuticos Ltda.) (8.0 \pm 2.0 mg/kg iv) and maintained with sevoflurane (Sevorane® - Abbott Laboratórios do Brasil Ltda.), diluted with $\rm O_2$ (3.5V%), in a semi-closed anesthetic circuit. After the opening of the thorax the breathing was controlled.

Operative technique

Left lateral thoracotomy in the sixth intercostal space was performed, followed by T-shaped pericardiotomy. Three anchoring U-shaped sutures were made using 2-0 polypropylene thread (Prolene 2-0 agulhado – Ethicon Johnson & Johnson Produtos Profissionais Ltda.), fixed with bovine pericardium strips (Braile Biomédica Indústria, Comércio e Representação S.A) on the free wall of the LV, stretching from the apex to the third dorsal, below the left ventricular marginal branch [10], as can be seen in Figure 1.

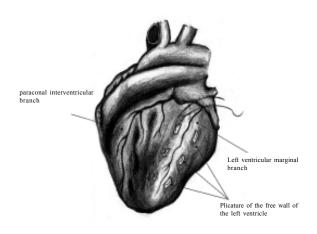


Fig. 1 – Technical design of the plicature of the free wall of the left ventricle. NB. The paraconal interventricular branch in the dog corresponds to the left anterior descending coronary artery

To avoid an excessive drop in the arterial pressure, the heart was partially dislocated, by lifting the tweezers fixed to the pericardium, only at the moment of making each stitch, repositioning it immediately after each stitch. The pericardium was not sutured and the thorax was closed in the normal manner.

Postoperative care

At the end of the operations, an intercostal block using bupivacaine (Neocaína® - Cristália Produtos Químicos Farmacêuticos Ltda.) (1.5 mg/kg) was performed. The dogs received tramadol (Tramal® - Searle/Monsanto do Brasil Ltda.) (1.5 mg/kg intramuscular at 8 hour intervals for three days), flunixim meglumine (Banamine® - Indústria Química e Farmacêutica Schering Plough S.A. – Divisão Veterinária) (1.1 mg/kg intramuscular at 24 hour intervals for three days) and ampicilina (Optacilin® - Byk Química e Farmacêutica Ltda) (22 mg/kg intramuscular at 24 hour intervals for seven days).

Statistical analysis

Analyses of repeated measurements over time were made for each of the evaluated variables, utilizing the mixed procedure of the SAS ® version 8.0 [13]. For the cardiac outflow, variance analysis with 2x2 factorial rearrangement was performed and for the variables of the Holter system, simple variance analysis was made. A significance level for a p-value of 0.05 was established.

RESULTS

Induction of dilated cardiomyopathy

All the animals in Groups II and III experienced a significant increase of the LV diameter at systole (mean 22.75 $\pm\,1.78$ mm to 30.84 ± 3.1 mm – p-value <0.05) and at diastole (mean 33.53 ± 1.31 mm to 37.9 ± 3.9 mm – p-value <0.01) after a cumulative dose 240 mg/m2 of doxorubicin. This condition reduced the EF from 31.67 ± 1.65 % to 17.50 ± 1.51 % (p-value <0.01) after induction. However, two dogs died due to congestive heart failure and ventricular arrhythmia during induction (cumulative dose 150 mg/m²).

Clinical evaluation

All the induced dogs demonstrated signs of intolerance to exertion and weak pulses, with a significant improvement after the operation. The operated dogs presented with a very fast rhythm in the immediate postoperative period. The dogs in Group III gradually deteriorated, with death due to acute pulmonary edema at 36 to 48 days after induction of cardiomyopathy.

A trans-operative ECG revealed ventricular extra-systoles during suturing of the myocardium, with one animal from

Group I presenting with paroxistic ventricular tachycardia which improved with lidocaine (Lidol® - Hipolabor Farmacêutica Ltda.). A postoperative ECG and the Holter monitoring demonstrated isolated ventricular extra-systoles in pairs in bigeminal pulses and occasional paroxistic ventricular tachycardia, which spontaneously improved 48 hours after the operation. There was also an increase in the amplitude of the T-wave and a reduction in the amplitude of the R-wave in the first postoperative week.

The DLVd and the DLVs reduced significantly 48 hours after the operation. In Group I, the EF did not alter greatly, in

Group II it increased significantly and in Group III it reduced drastically (Table 1, Figure 2). The increase in Group II was of about 38% in the immediate postoperative period (from 16.75% to 23.25%) and 114% on the 180th postoperative day (from 16.75% to 35.98%), but within the normal values from the 15th postoperative day.

The area and volume of the LV was significantly diminished in the operated groups and increased in Group III (Figures 3, 4 and 5). There was a significant increase of the EjF after the operation in Group II and gradual drop in Group III, with only slight changes in Group I (Table 2, Figure 6).

Table 1. Means, Standard errors (SE) and probability in respect to pre-operative (Pr>T) shortening fraction of the left ventricle (EF) of healthy dogs (Group I - n = 5), dogs with dilated heart disease induced by doxorubicin (Group II - n = 4) submitted to plicature of the free wall of the left ventricle and induced dogs which remained without operation (Group III - n = 2). P-values < 0.05 are highlighted.

Variable	time (days)	Group I			Group II			Group III		
		Mean	SE	Pr>T	Mean	SE	Pr>T	Mean	SE	Pr>T
	pre	33.20	1.68	-	16.75	1.88	-	19.00	2.66	-
	2	32.40	1.68	0.6868	23.25	1.88	0.0045	20.50	2.66	0.6326
	7	33.00	1.68	0.9197	24.75	1.88	0.0006	21.00	2.66	0.5242
	15	35.40	1.68	0.2694	29.31	2.12	0.0000	14.50	2.66	0.1544
EF (%)	21	33.20	1.68	1.0000	27.98	2.12	0.0000	10.50	2.66	0.0083
	30	32.60	1.68	0.7623	27.98	2.12	0.0000	15.50	2.66	0.2665
	60	36.40	1.68	0.0572	30.31	2.12	0.0000	-	-	-
	90	36.20	1.68	0.1336	27.98	2.12	0.0000	-	-	-
	120	36.00	1.68	0.1610	32.31	2.12	0.0000	-	-	-
	150	37.80	1.68	0.0229	34.98	2.12	0.0000	-	-	-
	180	37.20	1.68	0.0469	35.98	2.12	0.0000	-	-	-

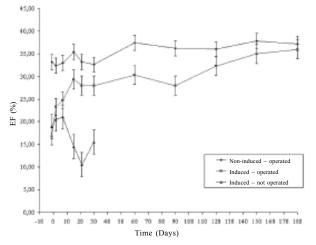


Fig. 2 – Left ventricular shortening fraction (FE) over time of healthy dogs (n = 5), dogs with dilated heart disease induced by doxorubicin (n = 4) submitted to plicature of the free wall of the left ventricle and induced dogs which remained without operation (n = 2)

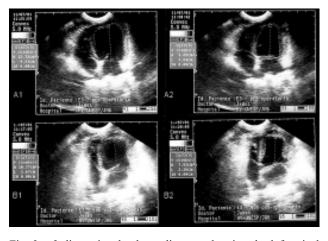


Fig. 3 – 2-dimensional echocardiogram showing the left apical view at diastole (1) and systole (2) of a dog with dilated heart disease induced by doxorubicin 24 hours before (A) and 48 hours after (B) plicature of the free wall of the left ventricle

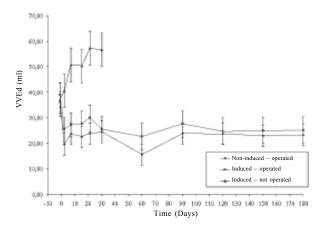


Fig. 4 - Volume of left ventricle at diastole (VVEd - mL), over time, of healthy dogs (n=5), dogs with dilated heart disease induced by doxorubicin (n = 4) submitted to plicature of the free wall of the left ventricle and induced dogs which remained without operations (n = 2)

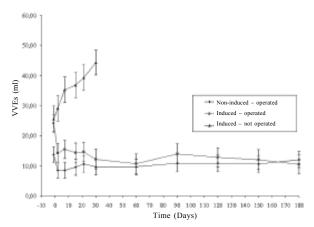


Fig. 5 – Volume of left ventricle at systole (VVEs – mL), over time, of healthy dogs (n=5), dogs with dilated heart disease induced by doxorubicin (n = 4) submitted to plicature of the free wall of the left ventricle and induced dogs which remained without operations (n = 2)

Table 2. Means, Standard errors (SE) and probability in respect to pre-operative (Pr>T) ejection fraction of the left ventricle (EjF) of healthy dogs (Group I - n = 5), dogs with dilated heart disease induced by doxorubicin (Group II - n = 4) submitted to plicature of the free wall of the left ventricle and induced dogs which remained without operation (Group III - n = 2). P-values < 0.05 are highlighted.

Variable	time	Group I			Group II			Group III		
	(days)	Mean	SE	Pr>T	Mean	SE	Pr>T	Mean	SE	Pr>T
	pre	59.40	3.73	-	37.50	4.18	-	31.00	5.91	-
	2	59.60	3.73	0.9674	44.25	4.18	0.2198	28.50	5.91	0.7467
	7	66.20	3.73	0.1676	43.75	4.18	0.2555	32.00	5.91	0.8972
	15	61.80	3.73	0.6241	46.84	4.79	0.1201	27.50	5.91	0.6512
EjF(%)	21	57.00	3.73	0.6241	53.50	4.79	0.0088	32.00	5.91	0.8972
	30	62.80	3.73	0.4879	53.50	4.79	0.0088	21.00	5.91	0.1989
	60	65.20	3.73	0.2383	53.50	4.79	0.0088	-	-	-
	90	59.20	3.73	0.9674	51.84	4.79	0.0183	-	-	-
	120	57.40	3.73	0.6829	53.50	4.79	0.0088	-	-	-
	150	57.20	3.73	0.6532	57.84	4.79	0.0010	-	-	-
	180	53.40	3.73	0.2227	60.17	4.79	0.0003	-	-	_

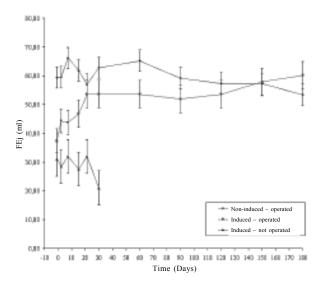


Fig. 6 – Ejection fraction (FEj) of the left ventricle, over time of healthy dogs (n=5), dogs with dilated heart disease induced by doxorubic (n = 4) submitted to plicature of the free wall of the left ventricle and induced dogs which remained without operations (n=2)

The cardiac output increased in Group I but without significance. In Group II however, this increase was significant (from $1,702 \pm 0.2$ vs. $2,315 \pm 0.11$ L/min – p-value < 0.1). The arterial pressure did not suffer alterations over the trial period.

COMMENTS

Induction of dilated cardiomyopathy by doxorrubicin was characterized by an increase in the DLVd and DLVs and reduced EF, in agreement to findings reported by TOYODA et al. [14] and MONNET & ORTON [15]. According to these authors the pharmacological therapeutic effects or operative techniques can be studied in experimental models of dilated cardiomyopathy induced by doxorrubicin, whose physiopathologic aspects are similar to idiopathic dilated cardiomyopathy.

Ventricular extra-systoles were observed during the suturing and in the first 48 postoperative hours. This arrhythmia was also observed by ANDRADE et al. [10] in 2001, and is possibly caused by mechanical irritation of the suturing process. The Holter system revealed ventricular extra-systoles and periods of paroxistic ventricular tachycardia, which disappeared after 48 hours, stressing the need of continuous electrocardiographic

monitoring in the first 48 postoperative hours.

The increase in the amplitude of the T-wave observed in this study may have been caused by myocardial hypoxia [10,16]. However, this alteration was temporary demonstrating that the PLVFW did not cause irreversible electrocardiographic alterations.

There was a reduction in the left ventricular volume at diastole and systole, which is in agreement to the findings of ANDRADE et al. [10]. In this study, the EjF increased by 60.4% 180 days after the operation (from 37.5 to 60.17%). In humans suffering from dilated cardiomyopathy submitted to partial ventriculectomy, a reduction in the DLVd and DLVs were observed. Also an increase of the EjF was seen, which according to BESTETTI et al. [17] was of about 65% (29.0% before the operation to 48.0% 12 months after), according to GRADINAC et al. [18] this was about 50.1% (23.9% vs. 40.7%), for MOREIRA et al. [19] it was an increase of 33.9% (17.7% vs. 23.7%), according to LUCCHESE et al. [20] it was 39.8% (22.1% vs. 30.9%) and according to POPOVIC et al. [21] it was 70.8% (24.0% vs. 41.0%).

In cases of partial ventriculectomy in humans, medical therapy is not discontinued and, normally, the operation is performed with inotropic support. In this study, the dogs with dilated cardiomyopathy induced by doxorrubicin did not receive any medication for congestive heart insufficiency, not even positive inotropic agents. The EjF of the induced dogs that did not undergo surgery, gradually dropped until the dogs died. Thus, the improvement in the operated dogs was attributed to PLVFW. However, the sample utilized here was small suggesting that further studies are necessary with a greater sample size.

MCCARTHY et al. [2] in 2001 observed an increase in the EjF from 19.0% to 36% in the immediate postoperative period that remained at 39% one month after the implantation of a myosplint, which is an apparatus with three polytetrafluoroethylene pins in the myocardium of dogs with cardiomyopathy induced by high-frequency pacemakers. However, this procedure is more onerous, aggressive and harder work than PLVFW.

According to NAIR et al. [9], a complication of partial ventriculectomy is the resection of a portion of the potentially viable myocardium. With PLVFW no area was removed and the blood supply of the pleated region was guaranteed by the maintenance of the left ventricular marginal branch.

In humans submitted to partial ventriculectomy, cardiac indices obtained from the outflow divided by the body surface area in m² were reported. According to

Gradinac et al. [18], the cardiac index increased from 2.3 to 2.8 L/min/m², while POPOVIC et al. [22] obtained an increase from 2.27 to 2.85 L/min/m² and KONERTZ et al. [23] reported an increase from 1.8 to 2.9 L/min/m². In this study, there was an increase in the outflow after PLVFW, which was significant in the dogs with cardiomyopathy induced by doxorrubicin.

CONCLUSIONS

Plicature of the left ventricle free wall without cardiopulmonary bypass reduced the area and volume of the left ventricle, which remained for a period of six months, both in healthy dogs as in those suffering from dilated cardiomyopathy induced by doxorrubicin. The surgical procedure was fast, with low morbidity and mortality.

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