Effect of autonomic blockade on the hemodynamic findings in acute cardiac tamponade

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FRIEDMAN, Howard S., FOUAD LAJAM, QAMAR ZAMAN, JOSEPH A. GOMES, JAIME CALDERON, NINO D. MARINO, HEMAL A. FERNANDO, AND SOO-SANG CHOE. Effect of autonomic blockade on the hemodynamic findings in acute cardiac tamponade. Am. J. Physiol. 232(1): H5–H11, 1977 or Am. J. Physiol.; Heart Circ. Physiol. 1(1): H5–H11, 1977.—Twenty-three closed-chest, α-chloralose-anesthetized, volume-expanded, alpha- and beta-adrenergic-blockaded dogs with rate fixed by atrial pacing had 20–90 ml of saline at 37°C infused into the pericardial sac a) with vagus intact, b) after vagotomy, and c) with vagus intact but with systolic pressure augmented with a balloon. A significant reduction in left ventricular (LV) systolic pressure (SP), and cardiac output (CO) occurred at a pericardial volume of 30–60 ml, when LV end-diastolic pressure and right atrial RA] pressures were not increased. Whereas the percentage decline of CO, LVSP, maximum negative and maximum positive dP/dt was greater in group A (vagus intact) than in group B (vagus cut), significant residual depressed performance was demonstrated only in group B. In four paced, atropinized, beta-blockaded dogs, response to tamponade was similar to that in intact dogs; vagotomy at 90 ml in these dogs resulted in a fall in CO, a rise of LVSP and a significant elevation in LVED and RA pressures. Thus, in the early phases of cardiac tamponade a sympathetic neurohumoral response supports cardiac performance while the vagus nerve exerts a myocardial protective effect. Vagal afferents appear to modulate this response.

vagus nerve; beta-adrenergic blockade; phenoxybenzamine; alpha-adrenergic blockade; propranolol; atropine; cardiopulmonary afferents; maximum positive dP/dt; maximum negative dP/dt


METHODS

Twenty-three mongrel dogs ranging in weight from 17.6 to 24.6 kg were studied 2–4 days after thoracotomy to insert a polyethylene catheter into the pericardial sac, with pentobarbital sodium, 30 mg/kg, as the anesthesia. These closed-chest dogs were anesthetized with a chloralose, 80 mg/kg, administered 1.5–2 h before the experiment. No additional anesthetic was administered prior to performing the experiment or during the experiment. All dogs were intubated and ventilated with a Harvard volume respirator (Harvard Apparatus Co., Inc., Millis, Mass.) set at a rate of 12 beats/min and a tidal volume of 12–15 ml/kg. Using cutdown techniques and under fluoroscopy we positioned a polyurethane catheter in the right atrium and another in the ascending aorta. A Statham SF1 catheter-tipped micromanometer (Statham Laboratories, Inc., Hato Rey, Puerto Rico) was placed in the left ventricle. A bipolar electrode catheter was positioned against the right atrial wall. Right-sided catheters were passed via the femoral veins, whereas left-sided catheters were passed via the femoral arteries. Pericardial pressures were measured before and after each pericardial infusion with the same Statham 23Db strain-gauge manometer used to measure right atrial pressure. Left ventricular pressures were measured by a catheter-tipped micromanometer and a Statham 23Db strain-gauge manometer that measured pressures obtained through the fluid-filled lumen of the SF1 catheter and that was used to calibrate the SF1 catheter in vivo. In addition, six dogs had a balloon-tipped catheter positioned in the descending aorta just distal to the left subclavian artery. Six dogs were studied after alpha- and beta-adrenergic blockade; six after alpha and beta blockade and bilateral sectioning of the cervical vago sympathetic trunk; seven after alpha and beta blockade and aortic...
pressure augmentation with a balloon; and four after both cholinergic blockade and beta-adrenergic blockade. Alpha-adrenergic blockade was produced with phenoxycyanamine HCl (generously supplied by Smith Kline & French Laboratories, Philadelphia), 5–10 mg/kg, intravenously infused over 15 min in 300 ml of normal saline. All dogs subjected to alpha blockade received an additional 300 ml of saline intravenously to expand blood volume. Beta-adrenergic blockade was produced with propranolol HCl (generously supplied by Ayerst Laboratories, Inc., New York), 1–2 mg/kg in 20 ml of normal saline, intravenously administered as a bolus over 20–30 s. Cholinergic blockade was produced with atropine, 0.4 mg/kg, intravenously administered as a bolus injection. After blockade, heart rate was held constant in all dogs by atrial pacing at the rate of 125 beats/min, with a Medronic model 5837 pulse generator (Medronic, Inc., Minneapolis) set at approximately twice the diastolic threshold. With heart rate held constant, tests for adrenergic blockade were performed with phencyclidine, 150 μg/kg, and isoproterenol, 1 μg/kg. Blockade was considered accomplished when these agonists failed to augment peak positive dP/dt or peak left ventricular systolic pressure by more than 5%. In six dogs after alpha- and beta-adrenergic blockade, an intra-aortic balloon was expanded in the descending aorta just distal to the left subclavian artery with 5–10 ml of radiographic contrast (Renografin 76%) until maximal elevation of the aortic systolic pressure had occurred. A minimum of 15 min was allowed after production of the desired experimental conditions to ensure that a hemodynamic steady-state was present. Continuous hemodynamic and electrocardiogram recordings were made on a multi-channel, oscillographic photographic recorder (model DR-8, Electronics for Medicine, White Plains, N.Y.) at paper speeds of 25–100 mm/s as 30 ml of normal saline at approximately 37°C was intermittently infused over a 15- to 30-s period at 5- to 10-min intervals. Recordings for measurement were obtained after the hemodynamic parameters had stabilized. After 90 ml of saline were infused and hemodynamic data were obtained, the fluid was rapidly removed. Five minutes after the removal of the entire volume of infused fluid, hemodynamic measurements were repeated. Additionally, for the purpose of determining the role of the vagal afferents in cardiac tamponade, four atropinized and beta-blocked dogs had hemodynamic determinations made immediately before and 15–30 s after bilateral vagotomy when 90 ml of fluid were present in the pericardial sac and the heart rate was fixed at about 125 beats/min by atrial pacing.

Measurements. All pressure measurements were made over an entire respiratory cycle (12 beats) and the mean value of such a determination used. The first derivative of the left ventricular pressure pulse (dP/dt) obtained with the SF-1 catheter-tipped micromanometer was computed continuously by a resistance-capacitance differentiating circuit and subsequently converted into millimeters of mercury per second. Cardiac output was determined in duplicate by the dye-dilution technique, with indocyanine green USP (Westcott & Dunning, Inc., Baltimore) injected as a bolus into the right atrium and sampled in the ascending aorta. A Harvard dual infusion-withdrawal pump (Harvard Apparatus Co.) was used to withdraw blood at a constant rate of 50 ml/min with the concentration of the dye determined by a Gilford model 103IR cuvette densitometer (Gilford Instrument Laboratories, Inc., Oberlin, Ohio). After each determination the blood was returned to the dog.

To determine the relationship of the findings observed in the blocked dog to those found in the intact animal, the data obtained from the present study were compared to data from 10 dogs prepared and studied in the same manner but with the autonomic nervous system intact. Comparison of the changes in variables was done by the Student t test for paired data and the changes between groups by the Student t test for unpaired data (P < .05 was considered significant) (22). Values given are means plus/minus the standard error of the mean.

RESULTS

A representative series of pressure recordings obtained during control periods and with increasing degrees of cardiac tamponade in an alpha- and beta-adrenergic blockaded dog with the vagus nerves intact and aortic pressure augmented with balloon is shown in Fig. 1. The hemodynamic effects of cardiac tamponade in dogs subjected to autonomic blockade are summarized in Table 1 and Figs. 2 and 3.

Alpha- and beta-adrenergic blockade with vagus nerves intact. With adrenergic blockade and vagus nerves intact, an infusion of only 30 ml into the pericardial sac produced a decline of left ventricular systolic pressure by an average 21 ± 2 mmHg, P < .001, cardiac output by 222 ± 69 ml, P < .025, maximum positive dP/dt by 371 ± 96 mmHg/s, P < .025, and maximum negative dP/dt by 796 ± 131 mmHg/s, P < .005, at a time when right atrial and left ventricular end-diastolic pressures were not increased. Another 60–90 ml produced marked decline in left ventricular systolic pressure, cardiac output and maximum positive and negative dP/dt. Left ventricular end-diastolic pressure failed to rise even after 90 ml and, in fact, was lower by 2 ± 4 mmHg, P < .005, than control after 60 ml had been infused. Five minutes after removal of infused fluid, cardiac performance had returned to control values. Alpha- and beta-adrenergic blockade with vagi cut. With adrenergic blockade and vagotomy an infusion of 30 ml into the pericardial sac resulted in a decline of left ventricular systolic pressure by 11 ± 3 mmHg, P < .025, and cardiac output by 218 ± 78 ml, P < .05. Peak negative dP/dt was significantly reduced after 60 ml had been infused, declining by 712 ± 141 mmHg/s, P < .005. Peak positive dP/dt was not significantly reduced until 90 ml had been infused, when it had declined by 682 ± 190 mmHg/s, P < .025. Right atrial and left ventricular end-diastolic pressures did not change significantly. Five minutes after evacuation of the infused pericardial fluid, left ventricular systolic pressure was still lower than control by 17 ± 3 mmHg, P < .01, cardiac output by 258 ± 95 ml, P < .05, and peak negative dP/dt by 585 ± 256 mmHg/s, P < .05.
FIG. 1. Representative response of \( \frac{dP}{dt} \), left ventricular (LV), right atrial (RA), and pericardial pressure to increasing (30-90 ml) degrees of cardiac tamponade and at 5 min after removal of fluid (post) in an alpha- and beta-blockaded dog with balloon. Abbreviations: PP, pericardial pressure; EDP, end-diastolic pressure. Interval between time lines is 1 s. Upper traces recorded at 25-100 mm/s, lower trace (pericardial pressure) at 10 mm/s.

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<th>TABLE 1. Effect of cardiac tamponade in dogs subjected to autonomic blockade</th>
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Values represent means ± 1 SE. Abbreviations are: LV, left ventricle; Posttamp, 5 min after relief of tamponade. *Values are significantly different (P < .05 to < .001) compared to control.
FIG. 2. Comparative changes of right atrial (RA) and left ventricular end-diastolic pressures (LVEDP) with increasing degrees of cardiac tamponade (30-90 ml) when compared to control (C). Mean values are shown. n, number of dogs. P value indicates significance of change when compared to control.

FIG. 3. Comparative percent changes of cardiac output (A), peak positive dP/dt (B), left ventricular systolic pressure (C), and peak negative dP/dt (D) with increasing degrees of cardiac tamponade (30-90 ml) when compared to control, C. Mean values are shown. n, number of dogs. P value indicates significance of change when compared to control.
Alpha- and beta-adrenergic blockade with vagus nerves intact and aortic pressure augmentation. With adrenergic blockade, vagus nerves intact, and systemic pressure augmentation with an intra-aortic balloon, the infusion of 30 ml into the pericardial sac produced a decline of cardiac output by 262 ± 83 ml, \( P < .05 \). However, left ventricular systolic pressure and peak positive \( dP/dt \) were not significantly reduced until 60 ml had been infused. Peak negative \( dP/dt \) did not change significantly even after 90 ml. Left ventricular end-diastolic pressure declined and was lower than control by 2.2 ± 0.6 mmHg, \( P < .02 \), after 90 ml had been infused; right atrial pressure did not change significantly after these infusions. Five minutes after evacuation of the fluid infused into pericardial sac, all hemodynamic determinations were not significantly different from control values.

Cholinergic-beta-adrenergic blockade with vagus nerves intact. Cardiac tamponade was produced in four dogs with both cholinergic and beta-adrenergic blockade. These dogs showed the characteristic increment of both left ventricular end-diastolic and right atrial pressure with increasing pericardial fluid. After 60 ml, right atrial pressure had increased from 3 ± 1.3 to 7 ± 2 mmHg, \( P < .01 \), and peak positive \( dP/dt \) had declined from 1,753 ± 100 to 1,496 ± 67 mmHg/s, \( P < .05 \). Vagotomy after the pericardial sac contained 90 ml elevated left ventricular end-diastolic pressure from 11 ± 3.9 to 16 ± 3.6 mmHg, \( P < .025 \), and right atrial pressure from 10 ± 3.9 to 12 ± 3.8 mmHg, \( P < .05 \); also, left ventricular systolic pressure increased from 160 ± 8 to 172 ± 25 mmHg and cardiac output declined from 1.6 ± 0.2 to 1.3 ± 0.3 liter/min. A representative pressure recording demonstrating the effect of vagotomy is shown in Fig. 4.

Comparative findings. The comparative hemodynamic changes in dogs with adrenergic blockade are shown in Table 1. The percentage decline of left ventricular systolic pressure, cardiac output, peak positive \( dP/dt \) and peak negative \( dP/dt \) was most marked in the dogs having adrenergic blockade, intact vagus nerves, and no aortic pressure augmentation. Moreover, the decline of left ventricular systolic pressure was greater in dogs with intact vagus nerves and no aortic pressure support than in the vagotomized dogs: \( \Delta 21 ± 5 \) (intact) vs. \( \Delta 11 ± 8 \) mmHg, \( P < .05 \), at 30 ml and \( \Delta 47 ± 10 \) (intact) vs. \( \Delta 28 ± 11 \) mmHg, \( P < .025 \) at 90 ml. This difference does not appear to be explained by the difference of initial values, which was not significant, \( P > .05 \). The presence of aortic pressure augmentation obscured the decline of left ventricular systolic pressure and peak positive \( dP/dt \) and completely masked the effect of cardiac tamponade on negative \( dP/dt \). It did not significantly affect cardiac output or diastolic pressure changes. Of the three completely adrenergic-blockaded groups, only those dogs vagotomized before cardiac tamponade showed residual depressed performance during the recovery period.

The hemodynamic findings in the four groups with autonomic blockade are compared to the intact dog in Figs. 2 and 3. Figure 2, A shows the characteristic progressive increment of right atrial pressure with increasing pericardial fluid in both intact and cholinergic-beta-blockaded dogs. The three groups with alpha blockade show a blunted response of right atrial pressure, with a significant increment present only after 90 ml in dogs with alpha and beta blockade and vagus nerves intact. Similarly, Fig. 2, B shows the blunted response of left ventricular end-diastolic pressure in the alpha-blockaded groups. By contrast, cholinergic-beta-blockaded dogs showed progressive elevation of left ventricular end-diastolic pressure from 7 ± 1.5 to 11 ± 3.9 mmHg and the intact dogs showed a significant elevation of left ventricular end-diastolic pressure at 90 ml.

Figure 3, A and B demonstrate the importance of intact beta receptors in cardiac tamponade. Whereas a significant reduction of cardiac output and peak positive \( dP/dt \) were not observed even after 90 ml in the intact dog, all other dogs showed a significant reduction of cardiac output after 30-60 ml and peak positive \( dP/dt \) after 30-90 ml. Figure 3, C shows the steep decline of left ventricular systolic pressure in dogs with alpha blockade when contrasted with intact or cholinergic-beta-blockaded dogs.

**FIG. 4.** Representative response of \( dP/dt \), left ventricular (LV), right atrial (RA), and pericardial (P) pressures to vagotomy in cholinergic-beta-adrenergic blockaded dogs. *Left panel*, after 90 ml infused into pericardial sac before vagotomy; *right panel* shows immediate effect of vagotomy. Heart rate is held constant by atrial pacing. Interval between time lines is 1 s. Upper traces recorded at paper speeds of 25-100 mm/s; lower traces (pericardial pressure) recorded at a paper speed of 10 mm/s. Abbreviations: PP, pericardial pressure; EDP, left ventricular end-diastolic pressure.
The importance of systolic pressure as a determinant of peak negative dP/dt is shown in Fig. 3, D. The intracardiac balloon-supported dogs and cholinergic-beta-blockaded dogs failed to show a significant decline in this variable even after 90 ml; both groups had a mean systolic pressure greater than 100 mmHg at this degree of cardiac tamponade. By contrast, the intact dogs had a significant decline of peak negative dP/dt at 90 ml despite having a left ventricular systolic pressure greater than 140 mmHg at that point.

DISCUSSION

By producing autonomic blockade the characteristic inverse relationship between ventricular diastolic pressure and cardiac performance found in acute cardiac tamponade could be dissociated. The rapid infusion of as little as 30-60 ml of fluid into the pericardial sac of the blocked dog produced a significant fall in cardiac output, left ventricular systolic pressure and peak positive dP/dt. The relatively early precipitous decline in cardiac performance was not accompanied by a concomitant increment of right and left ventricular end-diastolic pressures characteristically found in the intact dog. Vagotomy or systolic pressure augmentation failed to alter the pattern produced by the presence of adrenergic blockade. However, with cholinergic-beta-adrenergic blockade and the alpha-adrenergic receptors left intact, cardiac tamponade produced findings similar to those observed in the intact animal. Accordingly, the curvilinear volume-pressure relationships observed in the early phases of cardiac tamponade are not only dependent on the compliance characteristics of the myocardium and the pericardium (20), but also are dependent on an intact alpha-adrenergic autonomic nervous system.

The blunted increment of ventricular diastolic pressure that occurred with alpha-adrenergic blockade resembles findings previously reported in acute experimental cardiac tamponade with hypovolemia (5). It is possible that the expanded venous capacitance bed that resulted from sympathetic blockade may have produced a relatively hypovolemic state despite the comparatively large intravascular volumes infused prior to induction of cardiac tamponade. However, the finding of control right atrial and left ventricular end-diastolic pressures falling within the limits found in the intact dog, and the finding of a significant decline in left ventricular end-diastolic pressure in alpha- and beta-blockaded dogs after a pericardial infusion of 60-90 ml would suggest that other factors in addition to relative hypovolemia were operative.

There are two possibilities that could explain this seemingly paradoxical decline in left ventricular end-diastolic pressure with increased pericardial fluid. 1) The ventricular wall and/or pericardium became more compliant with increased pericardial fluid. 2) Part of the ventricular end-diastolic volume was displaced in a retrograde fashion into venous bed consequent to the failure of the venous bed to constrict appropriately (2). Reduction of venous tone would, thereby, remove a countervailing effect to the increasing compressive forces around the ventricle.

The first possibility seems unlikely. Although alpha-adrenergic receptors have been described in the left ventricle and may augment the contractile state when activated (19), previous studies have failed to demonstrate a change in ventricular compliance with direct sympathetic ventricular stimulation (15). Thus, it would appear that the blunted response of ventricular diastolic pressures in cardiac tamponade in the blockaded animal could not be explained by ventricular compliance changes resulting from alpha blockade. Moreover, it would seem inconceivable that increased pericardial volume per se could lead to an increased distensibility of ventricles, especially when myocardial ischemia, which accompanies cardiac tamponade (7, 11, 17, 24), leads to decreased compliance (14).

It would appear, therefore, that the rise of right atrial and left ventricular pressures in the early phases of cardiac tamponade, one of the cardinal findings in cardiac tamponade in the intact animal, requires a reactive venous-capacitance system to augment venous return and prevent ventricular volumes from being displaced retrogradely when external compression of the heart occurs. Such a mechanism would maintain end-diastolic dimensions and, when absent, no doubt would contribute to the relatively early decline in cardiac performance, as demonstrated in the present study.

It has previously been shown in the intact dog that ventricular performance curves, which take into account transmural ventricular pressure (10) and maximum dP/dt (9, 24) or normalized maximum dP/dt (7), do not change in the early phases of cardiac tamponade. Such findings suggest that in cardiac tamponade the contractile state of the left ventricle is maintained or augmented in the face of reduced left ventricular end-diastolic dimensions and myocardial ischemia. In the present study all dogs subjected to beta-adrenergic blockade had a significant decline in peak positive dP/dt at pericardial volumes found not to affect this parameter in the intact dog. Moreover, our findings are in accord with those of Pegram et al. (18). These investigators found that beta-adrenergic blockade significantly reduced the decline of the end-systolic diameter of the left ventricle in cardiac tamponade in closed-chest dogs. Thus, it would appear that a neurohumoral sympathetic response maintains cardiac performance by augmenting the contractile state, thereby compensating, in part, for the reduction of end-diastolic dimensions.

Another parameter that responded in a different fashion in the blockaded dog from that observed in the intact dog was maximum negative dP/dt. In the intact, anesthetized dog, maximal rate of pressure fall during ventricular relaxation was one of the earliest variables to fall significantly with cardiac tamponade (9). This change was presumed to be due to the pericardial fluid impeding ventricular relaxation and/or to the effects of the myocardial ischemia (14) resulting from tamponade. Whereas in the alpha and beta-blockaded dogs, both with the vagus nerve cut and intact, negative dP/dt was significantly reduced after 60-90 ml, in the dogs with systolic pressure augmentation this parameter failed to show a significant change, even after 90 ml of pericardial fluid. This finding is consistent with previous studies in both the anesthetized (3) and conscious dog (12)
that have demonstrated that peak systolic pressure is the major determinant of this parameter.

Finally, the present study suggests that the vagus nerve may play an important role in the early phases of tamponade. First, a role of the vagal efferents is suggested by the exaggerated depression of cardiac performance observed in the alpha- and beta-adrenergic blockaded dogs not subjected to vagotomy. However, only the adrenergic blockaded dogs that were vagotomized showed residual depressed performance after relief of tamponade. Thus, it would appear that the presence of the vagus nerve has a myocardial protective effect. Such an effect might be related to an improved oxygen supply-demand balance produced by the vagus nerve. The data from this study and the previously reported bradycardic-hypotensive effect observed in the late phases of tamponade in the intact dog (8, 9) would suggest that depression of cardiac performance by the vagus nerve may counteract the reduction of coronary blood flow produced by cardiac tamponade. A cholinergic coronary vasodilatory effect (4) could also play a role by promoting an increased delivery of oxygen to the myocardium. Because nearly maximum dilatation of the coronary arteriolar bed (1), previous studies have demonstrated a relatively minor role for sinoaortic receptors, especially under conditions of the present study in which carotid baroreceptors were left intact (6). This study would support, therefore, the hypothesis that vagal afferents may be modulating alpha-adrenergic efferents in acute cardiac tamponade.

Thus, the neurohumoral sympathetic response in tamponade sustains cardiac performance, not only by increasing heart rate, as previously shown (9, 11), and contractile state, but also by augmenting venous return. The vagus nerve appears to exert a protective effect in the early phases of cardiac tamponade, probably by improving myocardial oxygen demand-supply relations whereas in the late phases it is responsible for the bradycardic-hypotensive reflex of cardiac tamponade (8, 9).

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REFERENCE


