Animal-Assisted Therapy in Patients Hospitalized With Heart Failure

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CE 1.0 Hour

Notice to CE enrollees:

A closed-book, multiple-choice examination following this article tests your understanding of the following objectives:

1. Identify the physiological findings associated with advanced heart failure.
2. Discuss the overall effects of animal-assisted therapy on cardiopulmonary pressures, neurohormonal levels, and anxiety in advanced heart failure patients who participated in this study.
3. Describe the indications for further research in animal-assisted therapy that this study identifies.

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EBR Evidence-Based Review on pp 587-588.

Background Animal-assisted therapy improves physiological and psychosocial variables in healthy and hypertensive patients.
Objectives To determine whether a 12-minute hospital visit with a therapy dog improves hemodynamic measures, lowers neurohormone levels, and decreases state anxiety in patients with advanced heart failure.
Methods A 3-group randomized repeated-measures experimental design was used in 76 adults. Longitudinal analysis was used to model differences among the 3 groups at 3 times. One group received a 12-minute visit from a volunteer with a therapy dog; another group, a 12-minute visit from a volunteer; and the control group, usual care. Data were collected at baseline, at 8 minutes, and at 16 minutes.
Results Compared with controls, the volunteer-dog group had significantly greater decreases in systolic pulmonary artery pressure during (-4.32 mm Hg, $P = .03$) and after (-5.78 mm Hg, $P = .001$) and in pulmonary capillary wedge pressure during (-2.74 mm Hg, $P = .01$) and after (-4.31 mm Hg, $P = .001$) the intervention. Compared with the volunteer-only group, the volunteer-dog group had significantly greater decreases in epinephrine levels during (-15.86 pg/mL, $P = .04$) and after (-17.54 pg/mL, $P = .04$) and in norepinephrine levels during (-232.36 pg/mL, $P = .02$) and after (-240.14 pg/mL, $P = .02$) the intervention. After the intervention, the volunteer-dog group had the greatest decrease from baseline in state anxiety sum score compared with the volunteer-only (-6.65 units, $P = .002$) and the control groups (-9.13 units, $P < .001$).
Conclusions Animal-assisted therapy improves cardiopulmonary pressures, neurohormone levels, and anxiety in patients hospitalized with heart failure. (*American Journal of Critical Care.* 2007;16:575-588)

Heart failure is among the most common diagnoses in hospitalized adults in the United States. It is responsible for nearly 1 million hospitalizations annually, with estimated related healthcare costs of $27.9 billion.[1] Hospitalization for heart failure is associated with a poor prognosis for patients, and readmission rates within 6 months are close to 50%.[2]

Heart failure induces a number of neurohormonal changes, including activation of the sympathetic nervous system, activation of the renin-angiotensin system, and reduction in activity of the parasympathetic nervous system.[3] Increased levels of catecholamines, such as epinephrine and norepinephrine, are hallmarks of the deleterious neuroendocrine cascade that occurs in patients with advanced heart failure. Stress-induced increases in the epinephrine level may facilitate release of norepinephrine.[4]

Although these changes may result in a short-term increase in cardiac output toward normal, chronic neurohormonal activation is harmful and contributes to progression of heart failure.[5-7] Chronic neurohormonal activation has led to the use of medications such as neurohormonal antagonists, including angiotensin-converting enzyme inhibitors, aldosterone antagonists, and β-adrenergic receptor antagonists, to treat heart failure.[8]

Dog ownership is a significant, independent predictor of survival 1 year after a myocardial infarction.

Although advances in medication therapy have improved outcomes of patients with heart failure, medication regimens have an unintended consequence of making polypharmacy a central component of the management of heart failure. Little is known about the hemodynamic and neurohormonal effects of adding adjunctive and complementary therapies to pharmacological management of advanced heart failure. Animal-assisted therapy (AAT) is an adjunctive therapy that could benefit patients with heart failure.

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Review of the Literature

In AAT, the bond between humans and animals is an integral part of a patient's treatment.[9] Physiological variables change during short-term (2-12 minutes) interactions with animals and with pet ownership.[10-13] In several studies[10,12-14] with participants with normal or high blood pressure, interaction with animals yielded decreases in blood pressure and heart rate and an increase in peripheral skin temperature. Psychosocial and emotional benefits also have been the focus of studies of brief exposures of AAT of 10 to 30 minutes.[15,16] Psychosocial benefits include decreases in anxiety, isolation, and fear of procedures and improvements in social interaction, social support, communication, sensory stimulation, and happiness.[15-21]

In one study,[22] patients who were pet owners with long-term animal exposure had lower blood pressure, heart rate, and plasma renin activity in response to mental stressors (mathematical subtraction, speech) than did patients who were not pet owners. In patients who survived myocardial infarction, the risk for cardiovascular disease, morbidity, and mortality 1 year after the infarction was lower in those who were pet owners than in those who were not.[23-25] In the Cardiac Arrhythmia Suppression Trial, dog ownership was a significant independent predictor of survival in patients 1 year after acute myocardial infarction.[23] These data support the hypothesis that excess activity of the sympathetic nervous system due to both physiological and psychological stress can be reduced by AAT.

Patients with advanced heart failure are threatened by many physiological and psychological stressors.[26,27] Physiological stressors include the hallmark activation of the neuroendocrine cascade, most likely triggered by excitation of the sympathetic nervous system.[3,28] Chronic neurohormonal activation leads to ventricular remodeling.[27] Added to the physiological stress of heart failure is the psychological stress of living with a chronic, life-threatening illness and frequent hospitalizations. The presence of animals, and interaction with animals, decreases physiological indices such as heart rate and blood pressure and improves psychosocial variables (eg, reduces anxiety) in both patients and healthy persons.[15,22,29,30] In a patient with heart failure, the presence of a nonthreatening stimulus such as a dog could relax the patient by lowering the patient's state of arousal and reduce neurohormonal activation caused by overactivity of the sympathetic nervous system.[31-33]

Some patients may be indifferent to animals or may perceive animals as a source of stress.[34] In a study[35] of 58 hospitalized geriatric psychiatry patients who were randomly assigned to a pet therapy group or an exercise control group for 1 hour per day for 5 consecutive days, neither intervention resulted in significant differences in behavior or affect.[35] Similarly, in 33 male college students given mental stress tests with and without a dog present, no significant differences in dependent measures such as blood pressure and heart rate occurred between the 2 groups.[36,37]

Possible hazards associated with AAT include zoonotic infections (ie, infections that can be passed from animals to humans).[34] Effective strategies to prevent transmission of zoonotic infections include good hand washing and developing guidelines that include criteria for patient and animal suitability, infection control practices, and institutional policies.[34,38]

After the initiation of an AAT program, no zoonotic infections were reported in 3281 dog visits to 1690 hospitalized patients during a 5-year period.[39] Similarly, after the introduction of an AAT program, a children's hospital reported no increase in the rate of zoonotic infections or any other adverse incidences in the first 2-year period.[40]

Despite the applicability of AAT to hospitalized patients with heart failure, no randomized controlled trials of the effects of this therapy have been done. The purpose of this study was to determine if AAT could reduce the manifestations of physiological and psychological stress in patients with advanced heart failure. Specifically, we tested whether hospitalized patients with advanced heart failure who received AAT had improved hemodynamic measures, lower neurohormone levels, and decreased anxiety compared with patients visited by a volunteer only and a control group of patients who received usual care at rest.

Methods

Design

A 3-group (volunteer-dog team, volunteer only, and control) randomized, repeated-measures experimental design was used to determine the effect of AAT on multiple dependent variables. Dependent variables were (1) blood pressure, (2) heart rate, (3) pulmonary artery pressure (PAP), (4) pulmonary capillary wedge pressure (PCWP), (5) right atrial pressure (RAP), (6) cardiac index, (7) systemic vascular resistance (SVR), (8) epinephrine level, (9) norepinephrine level, and (10) state anxiety.

To control for the effect of the volunteer part of the team, we designed the study to include a group visited by a volunteer only and a control group. The experimental group received the AAT, which consisted of a 12-minute visit from a volunteer and dog per standard AAT protocol (see "Data Collection Procedures" section). The AAT protocol has been used at the University of California–Los Angeles Medical Center since 1994.[41] The patients in the volunteer-only group received a 12-minute visit from a volunteer who was unknown to the patients. The patients in the control group received usual care (at rest).

Sample and Setting

After approval from the institutional review board, 76 patients with a diagnosis of advanced heart failure admitted to the cardiac care unit or the cardiac observation unit were recruited for the study. Patients who met the selection criteria, agreed to participate, and signed the informed consent were randomized into 1 of 3 groups (volunteer and dog visit, visit by volunteer only, control). Volunteers also signed an informed consent as requested by the institutional review board.

> The presence of and interaction with animals decreases heart rate and blood pressure and improves anxiety.

Criteria for selection of participants included (1) advanced heart failure (including systolic and diastolic left ventricular dysfunction) requiring medical management with an indwelling pulmonary artery catheter; (2) age between 18 and 80 years; (3) ability to read, write, and speak English; (4) mental status alert and oriented to person, place, and time; and (5) SVR greater than 1200 dyne · sec · cm^{-5} at least once within 12 hours from the start of data collection. Exclusion criteria included (1) SVR less than 1200 dyne · sec · cm^{-5}; (2) allergies to dogs; (3) immunosuppression, defined as a white blood cell count of less than 4500 cells/μL; (4) infection, indicated by an elevated white blood cell count greater than 11 000 cells/μL; (5) body temperature greater than 38°C; and (6) decreased level of consciousness.

Data Collection Procedures

Patients were randomly assigned to 1 of 3 groups by using a table of random numbers. Group assignment determined the type of visit. Patients randomly assigned to the experimental group received a visit from a volunteer and a dog. The type of dog breed was not controlled for; 14 dogs of 10 various breeds were used. The 14 dogs included 1 extra-large dog, 6 large dogs, 5 medium dogs, and 2 small dogs. Each visit was conducted according to the guidelines taught during the volunteer and dog orientation: (1) volunteer introduces self and dog, (2) patient washes his or her hands before the visit, (3) dog lies on the bed with its head within 0.6 m (2 ft) of the patient's head on a clean sheet used as a barrier to the patient's bed, (4) patients may pet the dog and talk to the dog and volunteer, and (5) patient washes his or her hands after the visit. No attempt was made to control the content of the conversation during the visit. The visit lasted for 12 minutes. After the visit, an instant self-developing photograph was taken of the patient with the dog and given to the patient.

Patients randomly assigned to the volunteer-only group received a 12-minute visit from a volunteer. The volunteer introduced himself or herself, sat in a chair approximately 1.2 m (4 ft) from the patient's head, and let the patient know that the visit would last for 12 minutes if the patient was up to it. No attempt was made to control for the volunteer's usual conversation during the visit. No patient requested to end any earlier than 12 minutes. Patients randomly assigned to the control group were asked to lie quietly without talking unless they had a specific need or request. For all groups, a sign was placed on the patient's door or curtain asking everyone to please not interrupt the visit. Nurses assigned to patients to provide care were asked not to interrupt during the 12-minute interaction and data collection, unless an emergency occurred. Volunteers participating in the volunteer-dog teams were used for the volunteer-only group as much as possible to minimize any influence of a volunteer's personality on the results.

> The volunteer-dog team visit resulted in decreases in pulmonary artery and wedge pressures and state anxiety levels.

For all groups, data were collected at baseline immediately before the visit, 8 minutes after the intervention started, and at 16 minutes, which was 4 minutes after the end of the visit. These times were chosen because they most likely correspond to the maximal relaxation effect for a patient[18,30] and thus were times when a difference from baseline levels was most likely to be detected. For all data collection, patients were recumbent with the head of bed elevated 45° from horizontal.

The physiological variables (blood pressure, heart rate, PAP, PCWP, RAP, cardiac index, SVR, epinephrine level, and norepinephrine level) were assessed at all 3 times for all groups. In order to maximize a "steady state" period, neither dosages of intravenous medications nor patients' positions were changed in the 15 minutes before data collection or during data collection. Blood samples for measurement of plasma levels of epinephrine and norepinephrine were obtained at all 3 times for all groups. Anxiety was measured twice for all groups: at baseline and at 16 minutes. Only 2 measures of anxiety were used to avoid sensitizing patients to the instrument.

All data were collected by the coinvestigator (K.M.C.) or research assistants, who were critical care nurses with expertise in all areas of hemodynamic monitoring. Data collectors did not speak to the patients during the measurement of outcome variables and the intervention. Interrater reliability ($r = 0.94$) was established among research assistants by providing instruction that included training on how to measure all the dependent variables and completion of a test after the training on accurate measurement of hemodynamic variables. The entire data collection time per patient was approximately 1 hour.

Measures

Hemodynamic Parameters

Heart rate is defined as the number of beats per minute and was measured by using a bedside monitor (model M1176A, Phillips Medical Systems, Andover, Massachusetts). Blood pressure was measured noninvasively and automatically by using a bedside monitor (model M1006B, Phillips Medical Systems). The reliability and validity of the noninvasive automatic measurements of blood pressure have been established, and the accuracy of these measurements compared with intra-arterial measurements is high, with observed mean errors of +0.8% for systolic blood pressure and +1.7% for diastolic blood pressure.[42]

Cardiac index is a measure of flow. It is calculated as cardiac output in liters per minute divided by body surface area in meters squared. Cardiac output was measured by using the thermodilution technique: 3 injections with 10 mL of iced injectate per injection. Results were averaged to obtain cardiac output in liters per minute. SVR is total peripheral resistance of the vascular system and reflects the degree of vascular constriction. SVR was calculated from cardiac output by using the following formula:

([mean arterial pressure - RAP] x 80) divided by the cardiac output. Reliability of measures was ensured by leveling, zeroing, and calibrating instruments according to the manufacturers' specifications at the start of the study period. Accuracy of cardiac output obtained via the thermodilution method compared with output obtained via the direct Fick and dye dilution methods has been established ($r = 0.91$-0.97).[43]

Neurohormone Levels

Plasma levels of catecholamines (epinephrine and norepinephrine) were measured by using standard laboratory procedures (high-pressure liquid chromatography) and electromechanical detection. Because position can affect catecholamine levels, all patients were supine with the head of the bed elevated 45° when blood samples were obtained, and all had been in that position for 15 minutes before the first blood sample was taken. For measurements of catecholamine levels, 7 mL of blood was withdrawn from the proximal port of the pulmonary artery catheter after in-line withdrawal of an appropriate amount of blood for discard. Blood samples were placed in chilled heparinized tubes, put on ice, and immediately transported to the laboratory for processing. Reliability and validity of the measurement method have been well established. The interassay coefficient of variation was 8% for epinephrine and 5% for norepinephrine.[44]

Anxiety

The Spielberger State-Trait Anxiety Inventory, form Y-I, was used to measure state anxiety, defined as a transitory emotional state or condition of the human individual, characterized by subjective, consciously perceived feelings of tension and apprehension and heightened arousal of the autonomic nervous system.[45] The State Anxiety Inventory is a self-report questionnaire that asks participants about their feelings of anxiety "right now." It consists of 20 statements such as "I feel at ease" and "I feel worried." Our patients rated themselves on a 4-point scale of increasing intensity. After reverse-coding necessary items, we computed the scale score as the sum of the 20 items. The instrument has well-established reliability (Cronbach α .83-.92), validity (content, concurrent, and construct), and sensitivity for distinguishing changes in anxiety during periods as short as 10 to 30 minutes.[45]

Data Analysis

Descriptive statistics were used for all dependent variables. Means and standard deviations were calculated for continuous variables. Differences among the groups at baseline were examined by using 1-way analysis of variance. Frequencies and percentages were determined for categorical variables. The differences among the groups at baseline were determined by using the χ^2 statistic.

For each dependent variable, a mixed model was used to test for differences among the 3 groups at the 3 time points. All mixed model analyses were performed by using SAS statistical software (SAS Institute Inc, Cary, North Carolina). In each model, adjusted differences in the dependent variable from baseline to during the intervention (baseline-during differences) and from baseline to after the intervention (baseline-after differences) were estimated for all pairs of groups. For example, to compare the volunteer and dog team with the control group in the differences between baseline and during the intervention measurements, the estimated differences from baseline to during were calculated for each group and then subtracted by using the Estimate function in Proc Mixed. *P* values for these comparisons were computed. Each model was adjusted for the baseline measure of its dependent variable, age, sex, New York Heart Association classification, ejection fraction, dog ownership (never, in the past, present), having a live-in partner or a spouse, and current smoking status.

> Animal-assisted therapy reduced epinephrine and norepinephrine levels, suggesting changes in activation of the autonomic nervous system.

Results

Characteristics of the Sample

The 76 patients who completed the study had advanced heart failure as indicated by depressed left ventricular ejection fraction (mean, 23.4%; SD, 10.80%), impaired functional ability (New York Heart Association functional class III and IV, n = 72), vasoconstriction (SVR, mean, 1372 dyne · sec · cm^{-5}; SD, 365), and elevated plasma level of norepinephrine (mean, 1008 pg/mL; SD, 704; to convert to picomoles per liter, multiply by 5.911). Mean age of patients in the sample was 57 years (SD, 12.28). A total of 75% were men.

Heart failure was most often due to nonischemic cardiomyopathy (44 patients, 58%). Medications affecting cardiopulmonary status were not controlled for; however, intravenous infusions of drugs were not manipulated and oral medications were not given 1 hour before or during the intervention. The number and types of medications used did not differ significantly between groups; patients were given diuretics (83%), vasoactive medications (82%), inotropic agents (55%), angiotensin-converting enzyme

Table 1
Comparison of baseline demographic and clinical characteristics of the sample between volunteer-dog, volunteer-only, and control groups

Variable	Groups: Volunteer-dog (n = 26)	Volunteer-only (n = 25)	Control (n = 25)	*P*
	Mean (SD)	Mean (SD)	Mean (SD)	
Left ventricular ejection fraction, %	23.73 (12.26)	22.14 (9.76)	24.34 (11.04)	.77
Heart rate, beats/min	86.77 (18.83)	83.08 (13.87)	81.68 (15.45)	.50
Systolic blood pressure, mm Hg	98.85 (11.89)	100.68 (15.86)	99.56 (11.44)	.88
Diastolic blood pressure, mm Hg	60.69 (12.43)	61.68 (10.05)	59.04 (9.98)	.69
Mean arterial pressure, mm Hg	73.10 (10.43)	72.36 (11.06)	72.32 (9.18)	.95
Systolic pulmonary artery pressure, mm Hg	44.12 (14.23)	39.12 (10.54)	38.20 (11.19)	.18
Diastolic pulmonary artery pressure, mm Hg	20.96 (6.37)	19.52 (5.09)	19.64 (5.79)	.61
Pulmonary capillary wedge pressure, mm Hg	21.11 (6.51)	17.14 (5.79)	16.36 (4.74)	.02
Pulmonary capillary wedge pressure, mm Hg (without 2 outliers)	19.65 (5.11)	17.14 (5.79)	16.36 (4.74)	.15
Right atrial pressure, mm Hg	8.62 (5.12)	7.16 (3.88)	6.72 (3.63)	.26
Cardiac output, L/min	3.82 (0.92)	4.10 (0.79)	4.04 (0.66)	.41
Cardiac index[a]	2.02 (0.49)	2.14 (0.42)	2.13 (0.35)	.52
Systemic vascular resistance, dyne · sec · cm^{-5}	1428.62 (422.90)	1361.36 (394.22)	1348.96 (287.45)	.71
Plasma norepinephrine, pg/mL[b]	1098.23 (733.93)	927.65 (475.46)	923.43 (651.43)	.58
Plasma epinephrine, pg/mL[c]	84.45 (59.54)	58.27 (42.21)	52.68 (44.79)	.10
Anxiety, sum score in units	44.35 (15.32)	37.83 (12.21)	40.04 (12.70)	.23
Age, y	56.27 (14.36)	56.12 (12.56)	58.52 (9.73)	.74
	No. (%)	No. (%)	No. (%)	*P*
Men	17 (65)	22 (88)	18 (72)	.16
New York Heart Association class IV	17 (65)	18 (72)	15 (60)	.67
20% Left ventricular ejection fraction	8 (31)	8 (32)	7 (28)	.95
Cause of heart failure Ischemic	8 (31)	12 (48)	12 (48)	.35
Current smoker	4 (15)	3 (12)	1 (4)	.40
Living with a spouse (or significant other)	20(77)	15 (60)	17 (68)	.43
Current dog owner	19 (73)	14 (56)	11 (44)	.10
Previous dog owner	24 (92)	24 (96)	22 (88)	.11

[a] Calculated as cardiac output in liters divided by body surface area in meters squared.
[b] To convert to picomoles per liter, multiply by 5.911.
[c] To convert to picomoles per liter, multiply by 5.459.

inhibitors (62%), and β-blockers (38%). A total of 58% of the patients were current dog owners. The only difference among the groups at baseline was PCWP. It was significantly higher in the patients who received a visit from the volunteer-dog team than in the other 2 groups ($P = .02$, Table 1).

Mixed Models

Adjusted mean baseline-during differences for the 3 groups are presented in Table 2. Several differences in dependent variables were detected between the volunteer-dog group and the control group. Compared with the control group, patients in the volunteer-dog group had significantly greater decreases in systolic PAP (mean difference, -4.32 mm Hg; $P = .03$) and PCWP (mean difference, -2.74 mm Hg; $P = .01$).

Similar baseline-during differences were detected between the volunteer-dog group and the volunteer-only group (Table 2). Compared with the volunteer-only group, the volunteer-dog group had

Table 2
Differences between groups in decreases in each dependent variable from baseline to during the intervention

Variable	Groups: Volunteer-dog vs control[a]	Volunteer-dog vs volunteer-only[b]	Volunteer-only vs control[c]
	Adjusted mean difference (SD), *P*		
Heart rate, beats/min	-1.35 (1.84) .47	-3.55 (1.84) .06	+0.79 (1.35) .56
Respiratory rate, respirations/min	-0.19 (0.64) .76	-1.03 (0.64) .11	+0.36 (0.59) .54
Systolic blood pressure, mm Hg	+1.85 (2.30) .42	-0.71 (2.30) .76	-0.04 (2.25) .99
Diastolic blood pressure, mm Hg	-0.42 (2.88) .84	+0.18 (2.88) .95	-0.88 (2.82) .31
Mean arterial pressure, mm Hg	-1.23 (2.27) .59	-0.69 (2.27) .76	-0.59 (2.19) .79
Systolic pulmonary artery pressure, mm Hg	-4.32 (1.99)[d] .03	-5.48 (1.99)[d] .01	+0.13 (1.47) .93
Diastolic pulmonary artery pressure, mm Hg	-0.58 (1.39) .67	-2.78 (1.39)[d] .05	+1.19 (0.98) .23
Pulmonary capillary wedge pressure, mm Hg	-2.74 (1.04)[d] .01	-3.30 (1.04)[d] .003	+0.21 (0.97) .83
Pulmonary capillary wedge pressure, mm Hg (without 2 outliers)	-3.11 (0.06)[d] .005	-3.70 (1.06)[d] .001	+0.58 (0.99) .56
Right atrial pressure, mm Hg	-0.27 (0.70) .70	-1.87 (0.70)[d] .01	+0.91 (0.57) .11
Cardiac output, L/min	+0.14 (0.17) .41	+0.16 (0.17) .36	-0.32 (0.20) .11
Cardiac index[e]	+0.11 (0.08) .19	+0.09 (0.08) .27	-0.11 (0.10) .27
Systemic vascular resistance, dyne · sec · cm^{-5}	+11.9 (85.3) .89	-18.28 (85.27) .83	+87.34 (91.90) .35
Norepinephrine, pg/mL[f]	-47.44 (96.61) .63	-232.36 (95)[d] .02	-7.78 (93) .93
Epinephrine, pg/mL[g]	-2.80 (8.18) .73	-15.86 (7.82)[d] .04	-1.68 (9.85) .87

[a] Adjusted mean difference = mean decreases in the volunteer-dog group minus mean decreases in the control group. Negative values indicate greater decreases in the volunteer-dog group; positive values indicate greater decreases in the control group.
[b] Adjusted mean difference = mean decreases in the volunteer-dog group minus mean decreases in the volunteer-only group. Negative values indicate greater decreases in the volunteer-dog group; positive values indicate greater decreases in the volunteer-only group.
[c] Adjusted mean difference = mean decreases in the volunteer-only group minus mean decreases in the control group. Negative values indicate greater decreases in the volunteer-only group; positive values indicate greater decreases in the control group.
[d] $P \leq .05$.
[e] Calculated as cardiac output in liters divided by body surface area in meters squared.
[f] To convert to picomoles per liter, multiply by 5.911.
[g] To convert to picomoles per liter, multiply by 5.459.

significantly greater decreases in systolic PAP (mean difference, -5.48 mm Hg; *P* = .01), PCWP (mean difference, -3.30 mm Hg; *P* = .003), epinephrine levels (mean difference, -15.86 pg/mL; *P* = .04; to convert to picomoles per liter, multiply by 5.459), and norepinephrine levels (mean difference, -232.36 pg/mL; *P* = .02). In addition, patients in the volunteer-dog group had greater decreases in RAP (mean difference, -1.87 mm Hg; *P* = .01) and diastolic PAP (mean difference, -2.78 mm Hg; *P* = .05).

Adjusted mean baseline-after differences for the 3 groups are presented in Table 3. Two of the mean baseline-during differences between the volunteer-dog group and the control group also were found for mean baseline-after differences. Compared with the control group, the volunteer-dog group had significantly greater decreases in systolic PAP (mean difference, -5.78 mm Hg; *P* = .001) and PCWP (mean difference, -4.31 mm Hg; *P* = .001). Decreases in epinephrine and norepinephrine were similar for the 2 groups. However, compared with the control group, the volunteer-dog group had greater reductions in state anxiety (mean difference, -9.13 units; *P* < .001).

Most of the baseline-during differences between the volunteer-dog group and the volunteer-only group also were found for baseline-after differences (Table 3). Compared with the volunteer-only group, the volunteer-dog group had significantly greater decreases in systolic PAP (mean difference, -5.34 mm Hg; *P* = .002), PCWP (mean difference, -3.10 mm Hg; *P* = .02); epinephrine levels (mean difference, -17.54 pg/mL; *P* = .04), and norepinephrine levels (mean difference, -240.29 pg/mL; *P* = .02). However, decreases in RAP and diastolic PAP in the volunteer-dog group were not significantly greater than those in the volunteer group. In addition, the

Table 3
Differences between groups in decreases in each dependent variable from baseline to after the intervention

Variable	Groups: Volunteer-dog vs control[a]			Volunteer-dog vs volunteer-only[b]			Volunteer-only vs control[c]		
	Adjusted mean difference (SD), *P*								
Heart rate, beats/min	-1.44	(1.49)	.34	-2.76	(1.49)	.07	+1.32	(1.50)	.38
Respiratory rate, respirations/min	-0.39	(0.67)	.56	-0.67	(0.66)	.32	-0.27	(0.67)	.68
Systolic blood pressure, mm Hg	-3.35	(2.46)	.18	-0.75	(2.46)	.76	-2.60	(2.49)	.30
Diastolic blood pressure, mm Hg	-4.38	(2.53)	.09	-2.70	(2.53)	.29	-1.68	(2.56)	.51
Mean arterial pressure, mm Hg	-3.36	(2.25)	.14	-1.28	(2.25)	.57	-2.08	(2.27)	.36
Systolic pulmonary artery pressure, mm Hg	-5.78	(1.73)[d]	.001	-5.34	(1.72)[d]	.002	-0.44	(1.74)	.80
Diastolic pulmonary artery pressure, mm Hg	-0.68	(1.38)	.63	-1.59	(1.38)	.25	+0.92	(1.39)	.51
Pulmonary capillary wedge pressure, mm Hg	-4.31	(1.30)[d]	.001	-3.10	(1.28)[d]	.02	-1.22	(1.25)	.34
Pulmonary capillary wedge pressure, mm Hg (without 2 outliers)	-4.29	(1.36)[d]	.003	-3.07	(1.34)[d]	.03	-1.22	(1.27)	.34
Right atrial pressure, mm Hg	+0.44	(0.55)	.43	-0.96	(0.55)	.09	+1.40	(0.56)[d]	.01
Cardiac output, L/min	+0.06	(0.18)	.74	-0.16	(0.18)	.38	+0.22	(0.18)	.23
Cardiac index[e]	+0.10	(0.10)	.31	-0.02	(0.10)	.86	+0.12	(0.10)	.24
Systemic vascular resistance, dyne · sec · cm^{-5}	-73.30	(78.02)	.35	+69.06	(78.02)	.38	-142.36	(78.79)	.08
Norepinephrine, pg/mL[f]	-182.31	(102)	.08	-240.14	(100)[d]	.02	+57.83	(101)	.57
Epinephrine, pg/mL[g]	-12.56	(8.66)	.15	-17.54	(8.28)[d]	.04	+4.98	(8.46)	.56
Anxiety, sum score in units	-9.13	(2.10)[d]	<.001	-6.65	(2.13)[d]	.002	-2.48	(2.17)	.25

[a] Adjusted mean difference = mean decreases in the volunteer-dog group minus mean decreases in the control group. Negative values indicate greater decreases in the volunteer-dog group; positive values indicate greater decreases in the control group.
[b] Adjusted mean difference = mean decreases in the volunteer-dog group minus mean decreases in the volunteer-only group. Negative values indicate greater decreases in the volunteer-dog group; positive values indicate greater decreases in the volunteer-only group.
[c] Adjusted mean difference = mean decreases in the volunteer-only group minus mean decreases in the control group. Negative values indicate greater decreases in the volunteer-only group; positive values indicate greater decreases in the control group.
[d] $P \le .05$.
[e] Calculated as cardiac output in liters divided by body surface area in meters squared.
[f] To convert to picomoles per liter, multiply by 5.911.
[g] To convert to picomoles per liter, multiply by 5.459.

volunteer-dog group had significantly greater decreases in state anxiety (mean difference, -6.65 units, $P = .002$) than did the volunteer-only group.

Because PCWP was the only variable at baseline that differed significantly between the 3 groups, we explored this variable further. Using the box-plot method, we calculated the distance 1.5 times the interquartile range (ie, the distance between the 75th and 25th percentiles of the data). Any value more than this distance away from the median in either direction was considered an outlier. The volunteer-dog group had 2 outlier values. After eliminating these outlier values, we repeated the analysis for differences in PCWP between groups. The results showed similar differences between groups in PCWP with and without the outliers (Tables 2 and 3).

Therefore, the baseline-during and baseline-after values for systolic PAP and PCWP decreased significantly more in the volunteer-dog group than in the volunteer-only and control groups. Similarly, baseline-during and baseline-after values for epinephrine and norepinephrine levels decreased significantly more in the volunteer-dog group than in the volunteer-only group. The baseline-after decrease in state anxiety was greater in the volunteer-dog group than in the other 2 groups.

Discussion

In this study, patients hospitalized with advanced heart failure who received a visit from a volunteer and dog had lower cardiopulmonary pressures, neurohormone levels, and anxiety levels than did patients visited by a volunteer only and patients given usual care at rest (control group). Heart rate, blood pressure, cardiac index, and SVR were not significantly affected by the intervention.

In previous studies,[10,22,46] the presence of a dog, pet ownership, and AAT resulted in lower blood pressures in hypertensive and normotensive pet owners and in participants who did not own a pet. The mean blood pressure in these studies[10,22,46] ranged from a systolic pressure of 116 to 182 mm Hg and a diastolic pressure of 81 to 120 mm Hg. Several investigators[25,31,33] postulated that changes in blood pressure in pet owners were due to a decrease in sympathetic tone; however, the investigators did not measure neuroendocrine markers such as catecholamine levels.

The patients in our study did not have decreases in blood pressure similar to those reported in previous studies. One explanation is that changes in blood pressure in our patients may have been blunted by severe preexisting cardiac dysfunction. In addition, several of our patients received medications that could affect blood pressure response. A total of 62 patients (82%) were receiving vasoactive medications and 42 (55%) were receiving inotropic medications. Mean baseline blood pressures in our patients with advanced heart failure were low (mean systolic pressure, 99 mm Hg; mean diastolic pressure, 60 mm Hg) compared with normal and hypertensive populations in other animal interaction studies (mean systolic pressure, 144 mm Hg; mean diastolic pressure, 86 mm Hg).[10,11,13,14,22]

Despite evidence that AAT can lower blood pressure, the hemodynamic and neuroendocrine effects of this therapy are relatively unknown. To date, our study is the only experimental research in which the hemodynamic and neuroendocrine responses to AAT were examined. Hemodynamic effects of AAT included significant decreases in systolic PAP and PCWP during and after the intervention in patients visited by the volunteer and dog compared with the patients in the other 2 groups. Diastolic PAP and RAP were significantly lower during the intervention in the volunteer-dog group than in the volunteer-only group. Previous research[47,48] showed that the simple task of talking about a neutral topic can elevate cardiovascular reactivity, as indicated by increases in systolic blood pressure, diastolic blood pressure, and heart rate in patients with cardiovascular disorders.

Nemens and Woods[49] examined normal fluctuations in baseline hemodynamic pressures in a sample of cardiovascular patients and noted fluctuations of 5 mm Hg in systolic PAP. In our study, the mean decreases in systolic PAP from baseline to after the AAT intervention were approximately 6 mm Hg greater in the volunteer-dog group than in the control group. Therefore, the reduction in systolic PAP in response to AAT in the hospitalized patients with heart failure in our group was clinically relevant.

Hemodynamic changes also were associated with changes in plasma levels of epinephrine and norepinephrine. The decreases in plasma levels of epinephrine and norepinephrine during and after the intervention were significantly greater in the volunteer-dog group than in the volunteer-only group.

Plasma levels of catecholamines reflect the balance between the release of the hormones from sympathetic nerve terminals and their reuptake and degradation.[50] Increased levels of catecholamines, such as epinephrine and norepinephrine, are hallmarks of the deleterious neuroendocrine cascade that occurs in patients with advanced heart failure. The inotropic effects of endogenous catecholamines are blunted in heart failure, and catecholamines have direct deleterious effects on myocardial cells, left ventricular function, and mortality.[51,52] Furthermore, when a person perceives fear or loss of control, brain neuropeptides activate catecholamines such as epinephrine and norepinephrine, which affect heart rate and blood pressure.[53]

In our study, the decreases in neurohormone levels during and after AAT suggest changes in the activation of the autonomic nervous system. AAT may affect neurohormone levels by altering the response of the autonomic nervous system to stimuli that are perceived as pleasantly meaningful and stimuli that the individual wishes to interact with from the environment.[54] Thus, patients receiving AAT may have been focused on the dog and not on other environmental stimuli, such as the volunteer, the nurse who collected data, and other sensory stimuli present at the time of the intervention.

In our study, the decrease in anxiety after the intervention was significantly greater in the volunteer-dog group than in the volunteer-only and the control groups. Similar findings have been observed in hospitalized psychiatric patients and in college students.[15,17] For example, Barker and Dawson[15] investigated the effects of AAT on 230 hospitalized psychiatric patients and compared a single 30-minute AAT session with a 30-minute therapeutic recreation session. A significant decrease in anxiety occurred in patients with mood disorders, psychotic disorders, and other disorders who received AAT sessions.

The significant decrease in anxiety in the volunteer-dog group suggests that AAT may provide patients a meaningful stimulus and modulate anxiety-producing situations that encompass hospitalization, severe illness, and uncertainly of outcomes. AAT may be effective because pets are a source of social support and may buffer a person's reactivity to mental stressors.[20,22,23] AAT can provide a sense of comfort and safety during hospitalization, and it diverts attention away from the immediate stressors to a more pleasurable and calming interaction.[15]

After the intervention, the volunteer-only group had a significantly greater decrease in state anxiety than did the control group, who received usual care at rest. However, AAT resulted in the greatest decrease in state anxiety scores and was more effective than a visit from a volunteer only or leaving a patient at rest.

Limitations

The study has several limitations. Although PCWP was the only variable that differed significantly between the 3 groups at baseline, the volunteer-dog group had higher observed means than did the other 2 groups for most of the dependent variables at baseline. Data collection occurred during a short period (12 minutes), and patients in the volunteer-dog group with higher baseline measures might have been treated more aggressively than patients in the other 2 groups. Although no medication changes occurred 1 hour before and during the intervention, the greatest decreases in PAP, PCWP, and neurohormones could have been influenced by ongoing intravenous drug infusions at the time. Thus, the study results should be viewed cautiously, and replication is needed.

The lack of changes in other hemodynamic parameters such as cardiac index and SVR may be due to the short exposure (12 minutes) of the patients to the AAT. It is not known whether longer exposures may result in additional hemodynamic benefits. In addition, fluid limitations precluded more frequent measurements of cardiac index. Finally, the study was conducted at a single facility.

Conclusions

Our findings add to knowledge on AAT and indicate a new focus for study of interactions between physiological and psychological effects and therapeutic benefits of AAT. Although this preliminary study provides evidence of improved cardiopulmonary pressures, neurohormone levels, and anxiety in patients with heart failure who have AAT, a larger study is needed to sufficiently define the immediate and long-term improvement in these variables. Healthcare professionals can use AAT as an effective adjunctive treatment.

Further research is needed to evaluate the potential effect of AAT for achieving better management of symptoms (eg, dyspnea), improving satisfaction of patients, and decreasing length of stay in the hospital. Other areas of investigation are needed to determine whether this safe, inexpensive adjunctive therapy may contribute to the long-term treatment of patients with advanced heart failure by decreasing morbidity and mortality, decreasing depression, and improving the quality of life in domains such as functional status and social support.

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