



# Left ventricular structural and functional abnormalities in dogs with hyperadrenocorticism



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## KEYWORDS

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**Abstract Objectives:** Hyperadrenocorticism has been reported to cause left ventricular (LV) structural and functional abnormalities in human patients. The purpose of the present study was to assess the incidence and features of LV structural and functional changes in dogs with hyperadrenocorticism.

**Animals:** Twenty-two client-owned dogs with pituitary-dependent hyperadrenocorticism ( $n = 15$ ) and cortisol-secreting adrenocortical tumors ( $n = 7$ ) and 6 control dogs were enrolled in this study.

**Methods:** Echocardiographic examinations were performed and non-invasive measurements of systolic blood pressure (SBP) were obtained.

**Results:** The normalized LV wall thickness and LV mass index of the affected dogs differed significantly from those of control dogs. Using a published reference value for M-mode measurements, 15 of the 22 dogs (68%) were found to have increased LV wall thickness. Eleven of the 15 (73%) dogs with increased LV wall thickness were normotensive, and no significant correlation between LV wall thickness and SBP was found.

**Conclusions:** Regardless of the presence of systemic hypertension, hyperadrenocorticism should be included in the differential diagnosis of underlying disorders that may cause LV hypertrophy in dogs.

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### Abbreviations

ACEi	angiotensin converting enzyme inhibitor
ACTH	adrenocorticotrophic hormone
A wave	late diastolic wave of transmitral flow
CVHD	chronic valvular heart disease
E wave	early diastolic wave of transmitral flow
IVSd	interventricular septal wall thickness in end-diastole
IVSdN	normalized interventricular septal wall thickness in end-diastole
IVSs	interventricular septal wall thickness in end-systole
IVSsN	normalized interventricular septal wall thickness in end-systole
LA	left atrium
LA/Ao	left atrium to aorta ratio
LV	left ventricle
LVH	left ventricular hypertrophy
LVIDd	left ventricular end-diastolic diameter
LVIDdN	normalized left ventricular end-diastolic diameter
LVIDs	left ventricular end-systolic diameter
LVIDsN	normalized left ventricular end-systolic diameter
LVM	left ventricular mass
LVMI	left ventricular mass index
LVPWd	left ventricular posterior wall thickness in end-diastole
LVPWdN	normalized left ventricular posterior wall thickness in end-diastole
LVPWs	left ventricular posterior wall thickness in end-systole
LVPWsN	normalized left ventricular posterior wall thickness in end-systole
PH	pulmonary hypertension
SBP	systolic blood pressure
SHT	systemic hypertension
2DE	two-dimensional echocardiography

## Introduction

Hyperadrenocorticism is a common endocrine disorder in middle-aged to elderly dogs.<sup>1,2</sup> Prolonged excessive secretion of cortisol leads to various clinical complications including pyelonephritis, cystic calculi, glomerulonephropathy, pancreatitis, diabetes mellitus, pulmonary thromboembolism, and pituitary macrotumor syndrome.<sup>1,2</sup> Systemic hypertension (SHT) is also a common complication in both humans and dogs with hyperadrenocorticism.<sup>3,4</sup> An increased risk of cardiovascular events has been reported in human patients with excessive cortisol secretions.<sup>4</sup> Left ventricular (LV) structural changes, mainly increased LV wall thickness, have been observed echocardiographically in people with hyperadrenocorticism.<sup>5–8</sup>

The purpose of this study was to evaluate LV structure and function in dogs with hyperadrenocorticism using transthoracic echocardiography.

## Animals, materials and methods

### Animals and inclusion criteria

Data from dogs diagnosed with hyperadrenocorticism at Azabu University Veterinary Teaching Hospital was prospectively evaluated between March 2010 and July 2011.

Dogs with previously diagnosed with hyperadrenocorticism at the teaching hospital that came in during the study period for recheck evaluations were also included. All dogs with hyperadrenocorticism presented to the hospital underwent echocardiographic examination. Diagnosis of hyperadrenocorticism was based on physical examination, clinical signs, adrenal gland ultrasonography (>6 mm of maximum transverse diameter), biochemical profile, adrenocorticotrophic hormone (ACTH) stimulation testing (post-ACTH serum cortisol concentration >20 µg/dL) and low-dose dexamethasone suppression testing (serum cortisol concentration >1 µg/dL, 8 h after

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