INTRODUCTION

It is widely accepted that in humans blood pressure is partially modulated by naturally occurring emotional responses. Potential candidate mechanisms include sympathetic output and systemic vascular resistance acting through α or β-adrenergic receptors. Moreover, clinicians are acutely aware of transient “white coat hypertension” elicited by the emotional state of the patient as a complicating factor in the diagnosis of chronic hypertension and the risks arising from treatment based on these blood pressure measurements. We sought out to first, determine if an emotionally driven increase in blood pressure is seen in dogs (pressor response), and second, to better understand the mechanism mediating this response.

METHODS

Dogs were instrumented with devices (DSI - St. Paul, MN) that would allow their unfettered movement and simultaneously record a dynamic electrocardiogram and arterial pressure. Throughout the study each dog was housed individually in isolated rooms with automated water and food sources, free from other dogs, and from unscheduled human interactions. Scheduled and controlled human interactions were either verbal from unscheduled human interactions. Scheduled and controlled human interactions were either verbal or physical only in the form of gentle restraint of the dog in lateral recumbancy. To test the effects of beta and alpha adrenergic mechanisms on the pressor responses, dogs were also tested 2 hours after pretreatment with either atenolol, prazosin or both. Systolic blood pressures were averaged following prazosin treatment resulted in a higher change in mean systolic pressure (23.4 mmHg) compared to physical restraint (10.2 mmHg) (fig. 6). Verbal interaction following atenolol treatment resulted in a higher change in mean systolic pressure (18.1 mmHg) compared to physical restraint (6.7 mmHg) (fig. 8).

RESULTS & DISCUSSION

There is a blood pressure response in dogs (pressor response) housed in isolation upon exposure to verbal or physical interaction (fig. 1). Verbal interaction resulted in a higher change in mean systolic pressure (32.9 mmHg) compared to physical restraint (7.3 mmHg) (fig. 2). 

Mandal attenuated the peak pressor response while prazosin delayed the development of a peak response (fig. 3+5). Verbal interaction following prazosin treatment resulted in a higher change in mean systolic pressure (23.4 mmHg) compared to physical restraint (10.2 mmHg) (fig. 6). Verbal interaction following atenolol treatment resulted in no significant difference between verbal (18.1 mmHg) and physical (7.3 mmHg) interaction (fig. 8).

We hypothesize that human interaction would elicit a pressor response in dogs and variation in the canine pressor response. Moreover, the interaction pressor response would be greater during physical restraint than during non-contact verbal-only interaction.

Specific Goals:
1. Quantify the blood pressure response associated with human interaction in normal dogs.
2. Compare the blood pressure response to verbal interaction to that of gentle physical restraint.
3. Determine whether the blood pressure response is affected by alpha, beta, or combined adrenergic blockade.

Drugs

<table>
<thead>
<tr>
<th>Drug Treatment</th>
<th>Description</th>
<th>Design</th>
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</thead>
<tbody>
<tr>
<td>Placebo</td>
<td>2 hours</td>
<td>Day 1</td>
</tr>
<tr>
<td>Atenolol 4 mg/kg PO</td>
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<td>Day 2</td>
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<tr>
<td>Prazosin 2 mg PO</td>
<td>2 hours</td>
<td>Day 3</td>
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CONCLUSION

“White-coat hypertension” elicited by the emotional state of the patient as a complicating factor in the diagnosis of chronic hypertension should be considered prior to the institution of antihypertensive therapy in dogs. This data is consistent with non-invasive human studies and warrants further study.

FINANCIAL SUPPORT

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REFERENCES