Neurogenic hypertensive dogs respond somewhat differently from normal ones. The pressor action is reduced and often reversed as in cats. The period of apnea is exaggerated as well as the recovery period with the forced respiration. Except for the respiratory effects the vascular responses in neurogenic hypertensive dogs are not unlike those of other pressor amines (Page and McCubbin, 1951).

**SUMMARY**

1. Natural serotonin, synthetic 5- and 7-hydroxytryptamine, 5-hydroxytryptamine-creatinine complex and tryptamine have been studied in dogs, cats and perfused organ preparations. In dogs they are primarily pressor with an initial depressor component suggesting the von Bezold effect. In cats serotonin and 5-hydroxytryptamine are primarily depressor.

2. Assayed in dogs, 5-hydroxytryptamine is about equal in pressor action to natural serotonin, three times more active than 7-hydroxytryptamine and twenty times as active as tryptamine. While epinephrine and norepinephrine are much more active as pressor agents, their vasomotor action is not proportionally as great as measured in the vessel of the perfused rabbit's ear. Tachyphylaxis has not been observed.

3. In perfused dog's legs and rabbit's ears, the tryptamines are vasoconstrictor with a reflex vasodilator component when the leg maintains nervous connection with the body. Severing vasomotor nerves in the perfused dog's kidney occurs but not to the degree elicited by norepinephrine.

4. The depressor action, in cats, in intact, and in cord-transected dogs is chiefly of vagal origin and is abolished by atropine or vagotomy. The respiratory phenomena of apnea followed by tachypnea are not significantly influenced.

5. Carotid sinus resection, spinal cord transection with or without removal, and partial autonomic blockade with penta- or hexamethonium iodide or tetraethylammonium chloride augment the pressor responses. Peripheral blockade of norepinephrine and epinephrine by benzodioxane and Priscoline does not block their action. 1-Hydrazino-2-phthalazine accentuates the subsequent depressor phase at the expense of the pressor.

6. Both "surgical" Pituitrinin certain doses and 6-amino-2-methyl-2-heptanol hydrochloride tend to augment the pressor action of the tryptamines especially in cord-transected dogs just as the latter enhances the action of norepinephrine. In contrast, large doses of Pituitrin completely block both the vascular and respiratory actions of 5-hydroxytryptamine-creatinine complex.

7. Cardiac output measured from satisfactory pressure-pulse tracings of a cord-transected dog shows increased cardiac output during the pressor phase and some decrease in calculated peripheral resistance.

8. The pressor response resulting from electrical stimulation of the central ends of the cut vagus nerves, in dogs with the spinal cord cut and treated with both ganglionic and peripheral blocking agents, is blocked by 1-hydrazino-2-phthalazine but failure to respond is not associated with inability of the dog to respond to 5-hydroxytryptamine.

9. The response of the tryptamines in neurogenic hypertensive dog's arterial