Renin Depletion in the Dog[1] (35065)

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(Introduced by J. P. Gilmore)

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The administration of DOCA and salt is known to deplete renin stores in the rat; however, the authors are unable to find any published regimen which depletes renal renin stores in the dog. We are presenting renal vein renin assays demonstrating the efficacy of DOCA and salt in canine renin depletion.

Methods. Four dogs weighing between 13 and 25 kg were used in this study. Three received 12.5 mg of DOCA intramuscularly and 6 g of sodium chloride by mouth daily for 14 days. The fourth dog was not pretreated with DOCA and salt and served as a control. Each dog was stimulated to release renin as follows:

Under pentobarbital anesthesia (25 mg/kg) a lower midline incision was made and a snare was passed about the abdominal aorta above the renal arteries. A catheter was placed in a radial artery to measure systemic arterial pressure above the snare and a similar catheter was placed in a femoral artery to measure arterial pressure below the snare. After blood pressure stabilized, a control blood sample was obtained from the left renal vein. Renal arterial pressure was then reduced to one half of the systemic pressure by constricting the aortic snare. The amount of aortic constriction was increased during the ensuring time to maintain renal arterial pressure relatively constant at one half of the systemic pressure. Twenty and 40 min after renal artery constriction, blood samples were again obtained from the left renal vein. Renal vein renin activity was determined by Laragh's modification of Pickens incubation technique and the Boucher bioassay (1, 2).

TABLE I. Renal Vein Renin (ng of Angiotensin II/100 ml/hr).

		Postocclusion	
	Control	20 min	40 min
Normal dog	472	1350	1800
Renin depleted			
Dog 1	23	83	69
2	0	47	55
3	135	161	150

Results. The results of the direct renin assays are shown in Table I. Resting renal vein renin activity in the untreated dog was 472 ng of angiotensin II/100 ml/hr. Following stimulation it rose to 1350 at 20 min and 1800 ng of angiotension II/100 ml/hr, at 40 min. Pretreated dogs demonstrated lower resting renin levels and failed to demonstrate the marked rise in renal vein renin during renal artery hypotension. In another study, one of the authors (E.D.V.) has used ureteral occlusion as a stimulus for renin release and has demonstrated the effectiveness of this method of renin depletion (3).

Discussion. The authors appreciate the paucity of control data, however our values are in agreement with the usual pre- and post stimulatory renin levels in the dog (3).

The dosage of DOCA and salt described above are well tolerated by the average size dog without marked weight gain or hypertension. In other studies of renin depleted dogs (3, 4) we have used 25 mg of DOCA and 12 g of sodium chloride/day. These dogs averaged about 20 kg and demonstrated a slight weight gain of about 5% over the 2-week

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period. Several of these dogs also demonstrated a very small increase in resting arterial pressure. The dosage of 25 mg of DOCA and 12 g of salt/day was also well tolerated by these dogs, but in smaller dogs (less than 10 kg) this dosage has been occasionally associated with vomiting, dehydration, and potassium depletion. The smaller dosage (12.5 mg of DOCA, 6 g of NaCl) has been extremely well tolerated by our animals, and, as demonstrated above, is an effective method for renal renin depletion.

Prior to aortic constriction, radial artery pressure in the control dog was 100 mm Hg. Following suprarenal constriction it gradually rose to between 110 and 115 mm Hg. The depleted dogs had a mean systemic pressure (as measured in the radial artery) of 133 mm Hg which increased to 140 mm Hg following constriction. Although the renin depleted dogs had a slightly higher systemic pressure, they did not demonstrate an appreciable change in pressure above the aortic constriction during our short period of study. Since only one control dog was used, no valid conclusions can be reached with this data.

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