was found that the rat blood pressure gave quite an amount of depressor artifacts.

Results and discussion. From Figure 1, it is evident that reduction of temperature from 37 °C to 20 °C increased the time for complete block of the neuromuscular junction at various doses of the snake venom (0.25, 1.25, 2.5, 6.25, 12.5, and 25 µg/ml). This is in agreement with the work of Patel and Excell[5], who have suggested involvement of at least two different types of mechanisms.

Post-tetanic potentiation was found to be depressed from the control values (considered as 100%) to 17.21% ± 7.84 (Mean ± SE; $p < 0.01$) and 2.07% ± 2.07 ($p < 0.01$) at 12 and 17 min respectively. The frequency of electrical pulses for post-tetanic potentiation being 20 Hz and repeated at intervals of 5 min after addition of 12.5 µg/ml snake venom to the bath. There was no post-tetanic potentiation at end of 22 min. Post-tetanic potentiation has been found to be a phenomenon concerned with presynaptic nerve terminals specifically dependent on the movement of Ca^+ and associated with an intracellular accumulation of Ca^+ during tetanus[9,10].

In Figure 2, the uptake of Ca^+ is reduced to less than half at the end of 32 min ($p < 0.01$) and the uptake of Ca^+ falls to about $^1/_4$th the control values after 180 min ($p < 0.001$). Ca^+ is essential for the release of Ach from presynaptic nerve terminals[11]. It was found that, in the present series of experiments, the Ach released from nerve terminals fell from the control values 78.10 ± 7.90 ng of the base to 26.28 ± 3.73 ng ($p < 0.001$) immediately after the complete block. The values fell to 15.43 ± 1.57 ng ($p < 0.001$) 180 min after the block.

From the present investigation it can be concluded that *D. jamesoni* venom has a multiple mode of action and one of the possible ways the venom acts is by affecting the release of Ach from presynaptic nerve terminals by restricting the uptake of Ca^+ necessary for the release of the neurotransmitter.

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The Influence of Salt Intake on Glomerular Count in Compensatory Kidney Hypertrophy in Rats of Different Ages

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Summary. Drinking saline instead of water elevated the glomerular count in hypertrophied kidneys of rats uninephrectomized as adults. No changes occurred in glomerular concentration in kideny tissue indicating a more marked increase of other kidney structures. This procedure was ineffective in immature animals.

The glomerular count was found to increase during postnatal kidney growth up to the age of 100 days[1,2], and even more so in kidneys undergoing compensatory hypertrophy induced by uninephrectomy before the 50th day of age[2]. At this period of development, a higher sensitivity of rats has been reported to hypertensogenic action of chronically increased salt intake[3]. The kidney plays a critical role in the pathogenesis of hypertension from the point of view of maintaining the water and salt balance[4] as evidenced, among other things, by the exaggerated hypertensive response to chronic excess salt ingestion in unilaterally nephrectomized rats[5]. Because of this, the influence of chronically increased salt intake on the number and 'concentration' of glomeruli was investigated in the normal and compensatory growing kidneys.

Methods. Male Wistar strain rats, maintained on a balanced pellet diet containing 1% NaCl and tap water ad libitum, were unilaterally nephrectomized at ages 18 and 80 days. 1 week after surgery, during which the young animals were left in the nest with the mother, an aliquot of these groups was exposed to a high salt intake (1% saline as the only drinking fluid) and the rest were left on the original dietary regime ('normal' salt intake). Intact animals of the same ages and maintained either on high or normal salt intake served as controls. At ages 55 and 117 days the animals were killed, the kidneys weighed and glomerular count determined by the method of Damadian et al.[6], as modified by Bonvalet et al.[2]. In order to compare the kidney weights in young and adult animals with different body weights at the end of the experiment, organ weight was expressed as: (mg organ wt./g body wt.^{2,3}) × 0.1, since in Wistar rats this expression does not change in the body weight range 30–680 g[5]. It corresponds basically to the formula used

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Table I. Relative kidney weights (mg/100 cm² body surface area) in normal and uninephrectomized animals reared under conditions of normal or high salt intake either from the 25th or the 87th day of life for 30 days

Group	Intact		Uninephrectomised	
	W	S	W	S
Young	254.3 ± 7.4 (5)	258.7 ± 5.8 (5)	371.4 ± 8.9 (5)	401.0 ± 10.1 (6)
Adult	249.7 ± 5.8 (5)	274.7 ± 23.5 (6)	333.6 ± 6.8 (5)	345.4 ± 11.4 (6)

Young, animals uninephrectomized on the 18th day of life. Adult, animals uninephrectomized on the 80th day of life. W, animals on a normal salt intake; S, animals on a high salt intake; number of animals is given in bracketts. Means ± SE; 100 cm² body surface area = (body weight in g)$^{2/3} \times 10$.

Table II. Glomerular count (NG) and glomerular concentration (GC) in 1 mg kidney mass in kidneys of normal and uninephrectomized animals reared under conditions of normal or high salt intake, either from the 25th or 87th day of age for 30 days

Group	Age (days)	Regime	NG		GC	
			Intact	Uninephrectomized	Intact	Uninephrectomized
Young	18	W	18,312 ± 711 (5)		55.7 ± 2.25 (5)	
	55	W	29,533 ± 677 (5)	31,617 ± 369 (5)	33.9 ± 1.52 (5)	24.7 ± 0.74 (5)
		S	28,854 ± 631 (5)	30,743 ± 690 (6)	32.7 ± 1.11 (5)	23.9 ± 1.20 (6)
Adult	80	W	31,563 ± 679 (5)			31.3 ± 1.41 (5)
	117	W	34,083 ± 767 (5)	32,722 ± 370 (6)	26.3 ± 1.06 (5)	18.7 ± 0.49 (6)
		S	31,200 ± 1018 (5)	35,833 ± 739 (6)	21.2 ± 1.80 (5)	19.8 ± 1.02 (6)

For symbols see Table I.

in the rat for calculating different parameters per 100 cm² of body surface area[7].

The results were statistically evaluated by the F-test for contrast and Student's *t*-test.

Results and discussion. In animals uninephrectomized when young, the compensatory kidney growth was more pronounced and was stimulated by increased salt intake (Table I). In this age group, the hypertrophied kidneys were heavier by 10% than in adult animals ($p < 0.01$) and drinking saline resulted in an additional increase in kidney weight (by 8%, $p < 0.05$).

In intact animals maintained on a normal salt intake, the number of glomeruli in the kidneys increased approximately by 90% between the 18th and 117th day of age (Table II). This was accompanied by decreased glomerular concentration in the kidneys, indicating intensive growth of non-glomerular structures (NGS). These changes are in agreement with literary data[1,2]. Unilateral nephrectomy increased the growth rate of NGS in the remaining kidney to a comparable degree in both young and adult animals, as evidenced by a roughly 30% decrease in glomerular concentration ($p < 0.01$). In rats uninephrectomized when young, the age dependent increase in glomerular count was more pronounced and the final values in hypertrophied kidneys were 7% higher than in normal animals ($p < 0.05$). This is in agreement with the findings of BONVALET et al.[2], the increase observed by us, however, was substantially lower. The sex of experimental animals might account for this difference, since former experiments[2] were carried out on females.

Increased salt intake did not influence either the glomerular count or glomerular concentration in both normally growing and hypertrophying kidneys in young animals. In rats exposed to a high salt intake when adult, however, both these parameters changed. The age dependent 9% increase in glomerular count observed in intact animals between the 80th and 117th day of age ($p < 0.05$) was inhibited and a 24% decrease in glomerular concentration ($p < 0.005$) indicated that kidney growth was due to a more pronounced growth of NGS. Compensatory growth counteracted this inhibition. Glomerular counts in the hypertrophied kidneys exceeded values found in both intact saline drinking controls ($p < 0.01$) and uninephrectomized animals on a normal salt intake ($p < 0.01$). Despite this the glomerular concentration remained unchanged, indicating that there is a more pronounced increase of NGS similar to that in normally growing kidneys.

Thus it appears that when salt intake is chronically increased, the kidneys of adult, but not young rats, are capable of changing their growth characteristics. This might play a role in the higher sensitivity of immature rats to the hypertensogenic action of salt.

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