Figure 1 shows a plot of the decay of IPSP amplitude following presentation of trains of 20 stimuli at different frequencies. The amplitude of test responses had an average period of decay of 60 sec to the baseline value, and the variation of the period was not considerable over the range 0.5–2.0 pulses/sec (40–70 sec). In figure 2, evidence is presented that the length of the train is important in governing the decay time of the response at tetanic frequency. The graph on the left shows the decay time course of the amplitude of responses after trains of different length presented at non-tetanic frequency. Points at time zero show the facilitated amplitude of the response at the end of the train. Full facilitation of 2.5 times the original response amplitude is partially developed after 10 pulses and more or less fully developed after 20–50 pulses. There is little variation in the timecourse of the decay process over this range of train lengths. The amplitude of potentials is back to normal after 40–60 sec, as shown by test responses evoked at 10 sec intervals after the train ends. There is no further increase in response amplitude during the period after the train. In the right-hand graph, the same range of train lengths was presented at tetanic frequency. Final responses in the train are much smaller due to tetanic fusion of the potentials. After 10 pulses, the amplitude and decay timecourse are those of simple facilitation. After 20 pulses, the timecourse is doubled and amplitude increased. After 40 and 60 pulses, a maximum potentiation and decay timecourse of 4 min are achieved. These are greater than those of facilitation and depend on tetanic stimulation for their appearance. In the 2 synapses described, both facilitation and PTP can be seen. In other cells, however, where little or no facilitation could be seen on sub-tetanic stimulation, potentiation of post-synaptic potentials could still be generated at higher frequency.

This long-lasting variety of plasticity of synaptic transmission resembles the post-tetanic potentiation observed widely in animal preparations[11]. In *Helix*, it does not appear after stimulation below the tetanic range, it also depends critically on the length of the priming train. On high-frequency stimulation of the synapse, the magnitude and duration of the potentiation developed as the train length was increased. This supports the view that PTP arises from a cumulative factor causing its increase with successive pulses[12].

1 M.G. Larrabee and D.W. Bronk, J. Neurophysiol. *10*, 139 (1947).
2 T.P. Feng, Chin. J. Physiol. *16*, 341 (1941).
3 P.B. Farel, Brain Res. *66*, 113 (1974).
4 J.C. Eccles and A.R. McIntyre, Nature, Lond. *149*, 274 (1951).
5 J.I. Hubbard, J. Physiol., Lond. *169*, 641 (1963).
6 R.I. Birks, J. Physiol., Lond. *271*, 847 (1977).
7 W.T. Schlapfer, J.P. Tremblay, P.B.J. Woodson and S.H. Barondes, Brain Res. *109*, 1 (1976).
8 J.P. Tremblay, P.B.J. Woodson, W.T. Schlapfer and S.H. Barondes, Brain Res. *109*, 61 (1976).
9 L.W. Haynes and G.A. Kerkut, Comp. Biochem. Physiol. *59A*, 311 (1978).
10 G.A. Kerkut, J.D.C. Lambert, R.J. Gayton, J.E. Loker and R.J. Walker, Comp. Biochem. Physiol. *50A*, 1 (1975).
11 J.R. Hughes, Physiol. Rev. *38*, 91 (1958).
12 J. Rosenthal, J. Physiol., Lond. *203*, 121 (1969).

Extracellular fluid distribution in salt-hypertensive monkeys

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Summary. In salt-hypertensive monkeys, a decrease in plasma volume was found, inversely related to blood pressure level and not accompanied by a corresponding change in interstitial fluid volume.

Most cases of essential hypertension were found to be associated with a decrease in plasma volume (PV), inversely related to arterial blood pressure (BP) level and not accompanied by a concomitant decrease of interstitial fluid volume (IFV). This was evidenced by the decreased PV/IFV ratio, indicating that the distribution of extracellular fluid volume (ECFV) between the extra- and intravascular compartment is altered[1].

Such a situation was not observed in animal experiments, however. In rats with established 1-kidney renal hypertension, reverse changes were found, the PV being expanded and PV/IFV ratio increased[2]. Thus the only common feature with essential hypertension was an abnormality in ECFV partition.

The differences in PV and PV/IFV ratio may be due to different mechanisms involved in the pathogenesis of renal and essential hypertension, including species differences. We therefore measured the changes of PV and IFV in relation to BP levels in monkeys exposed to a chronically increased salt intake. This regime was found to cause hypertension in baboons[3], and a high salt intake was suggested to play a role in the pathogenesis of essential hypertension in man[4].

Material and methods. Measurements were carried out in monkeys (*Maccacus rhesus*) of both sexes aged 5–7 years, of 7 kg mean b.wt, exposed to a high-salt regime (about 1.5 g NaCl/kg b.wt/24 h[3]) for a period of 3 years. Animals of comparable age and weight, receiving about 0.06 g NaCl/kg b.wt/24 h, served as controls. All measurements were performed under pentobarbital anaesthesia (30 mg/kg).

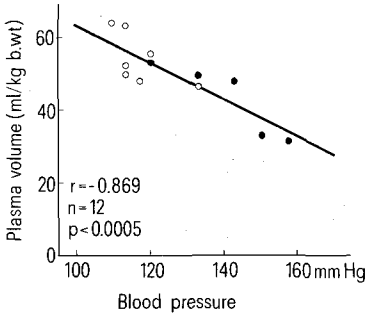
PV was estimated by dilution method, using Evans blue (1% solution w/v, 0.8 g/kg b.wt) injected into the exposed femoral vein. Plasma dye concentration was measured after a 10-min equilibration period against standard samples prepared in appropriately diluted blood plasma of the same

Blood pressure and extracellular fluid compartments in control and experimental monkeys

		Controls (n = 7)	Experimental (n = 5)
BP	S	133.6 ± 4.18	158.0 ± 5.83*
	D	82.9 ± 1.84	106.0 ± 8.12*
	M	116.6 ± 3.13	140.0 ± 6.50*
PV		54.2 ± 2.54	42.7 ± 4.22**
Hct		37.1 ± 1.63	42.4 ± 1.60
ECFV		176.3 ± 5.69	146.4 ± 11.54**
IFV		122.1 ± 5.59	103.7 ± 9.89
PV/IFV		0.451 ± 0.0314	0.419 ± 0.0632

BP, blood pressure in mm Hg: S, systolic, D, diastolic, M, mean; PV, ECFV, IFV, volumes of blood plasma, extracellular and interstitial fluid respectively (ml/kg b.wt); Hct, haematocrit. Means ± SEM. n, number of values. *$p < 0.01$; **$p < 0.05$.

animal, obtained before dye injection and used for haematocrit determination. ECFV was estimated as sodium ferrocyanide space using a single injection technique[5] (10% Na-ferrocyanide solution w/v, 1 g/kg b.wt) from plasma concentrations found 80, 100, 120 and 140 min after ferrocyanide administration. The exact amount of both indicators administered was estimated by weighing the syringe before and after injection. IFV was calculated as the difference between PV and ECFV. Systolic and diastolic BP were measured immediately after awakening from anaesthesia in unrestrained animals by Korotkoff's method. Mean BP was calculated as previously described[3]. Results were statistically evaluated using Student's t-test. Coefficients of linear correlations were calculated in the usual manner.



Correlation between blood pressure and plasma volume in control (open circles) and salt-fed (closed circles) monkeys. r, coefficient of linear correlation.

Results and discussion. In salt-fed animals, the mean values of systolic, diastolic and mean arterial BP were significantly elevated (table), demonstrating the hypertensogenic effect of increased salt intake in *Maccacus rhesus*, previously shown in *Pappio hamadryas*[1]. The BP increase was associated with a decrease of ECFV in both its compartments. However, only the PV reduction accompanied by a slight increase in haematocrit ($0.05 > p < 0.1$) was significant. Since there was no correlation between PV and IFV, the ECFV decrease apparently resulted from a disproportionate reduction of both these compartments. A slight though nonsignificant decrease of PV/IFV ratio indicates that the PV reduction was predominant. The PV decrease was inversely related to the BP level, as may be deduced from the negative correlation between PV and mean BP values ($r = -0.899$, $n = 5$, $p < 0.05$). This was also the case when control and experimental animals were pooled (figure). With IFV, no such correlation was found.

It is concluded that in salt-hypertensive monkeys the PV. but not IFV, decreases inversely to BP levels. This resembles the situation observed in essential hypertension[1], and thus supports the view that high salt intake may play a role in the pathogenesis of this disorder[4].

1 R.C. Tarazi, Circ. Res., Suppl. II, *8*, 11 (1976).
2 J. Lucas and M.A. Floyer, Clin. Sci. mol. Med. *47*, 1 (1974).
3 G.M. Cherkovich, K. Čapek, Z. Jefremova, I. Pohlová and J. Jelínek, J. appl. Physiol. *40*, 601 (1976).
4 L.K. Dahl, Am. J. Cardiol. *8*, 571 (1961).
5 C.R. Kleeman, F.H. Epstein, M.E. Rubini and E. Lamdin, Am. J. Physiol. *182*, 548 (1955).

Spider odor receptor: Electrophysiological proof

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Summary. By means of electrophysiological single-cell recordings, it was shown that the tarsal organ of *Cupiennius salei* is an olfactory chemoreceptor. One cell type in the organ of males responds to the odor of living females.

It has been shown several times that spiders perceive odorous substances[1]. In the hunting spider *Cupiennius salei*, behavioral reactions to fatty acids, terpineole, and tobacco smoke were observed[2]. The female of *Cyrtophora cicatrosa* most probably emits a sexual pheromone on its web to attract conspecific males[3].

The receptors transmitting such odorous stimuli in spiders are still unknown. Earlier authors[2,4,5] mistook the slit sense organs for olfactory receptors; in fact the slit sense organs are mechanoreceptors[6] and not chemoreceptors[7] as erroneously stated by Keller[2]. The so-called tarsal organ is widely regarded as a possible chemoreceptor[8]. It consists of a cuticular cup lying beneath the cuticular surface in a capsule open to the outside. According to electron microscopical investigations, this organ consists of 6–7 sensilla in the web spider *Araneus diadematus*[9]. These are innervated by 3 (rarely 4) sensory cells, each with several inbranched dendrites. The presence of cuticular pores[9] and behavioral experiments[8] suggest an olfactory or also hygroreceptive function of the tarsal organ. Electrophysiological proof for this suggestion is so far lacking. It is given in the present study.

Contrary to statements in the literature[2], *Cupiennius salei* is provided with tarsal organs. As in other spiders, one of

Reactions to 8 different stimuli in 20 recordings from the tarsal organ of *Cupiennius salei* which are representative for the total of 200 recordings

	Recordings from females										Recordings from males									
Formic acid	+	0	0	0	0	+	0	0	+	0	0	+	0	+	0	0	0	0	+	0
Valerian acid	+	0	0	0	0	+	0	0	+	0	0	+	0	+	0	0	0	0	+	0
Caproic acid	+	0	0	0	0	+	0	0	+	0	0	+	0	+	0	0	0	0	+	0
t-2-Hexenale	+	0	0	0	0	+	0	0	+	0	0	+	0	+	0	0	0	0	+	0
Hexanone	+	0	0	0	0	+	0	0	+	0	0	+	0	+	0	0	0	0	+	0
Tobacco smoke	0	+	+	+	0	0	0	+	+	+	+	0	0	+	0	0	+	+	0	+
Cupiennius female	0	0	0	0	0	0	0	0	0	0	0	0	+	0	+	0	0	0	+	0
Propylamine	−	0	0	0	+	−	+	0	−	0	0	−	0	−	0	+	0	+	−	0

+, Increase of the spontaneous cell activity of at least 10 imp/sec; −, decrease of the spontaneous cell activity to 0 imp/sec; 0, no reaction.