In Figure F, in response to the shock stimuli applied to IP, there occurred an excitatory postsynaptic potential (EPSP) with notches on its summit (the uppermost traces of Figure F). With a microelectrode just outside the cell soma, these notches were found to be due to field potentials occurring around the impaled cell (the middle traces). As difference between the intracellular and extracellular records the net EPSP was reproduced in the lowermost traces of F and found to be of simple shape, as those seen in cat's motoneurones[10]. As measured by the time intervals between the shock artifact and the foot of the EPSPs, the latency of the EPSPs was as short as 0.7 to 1.0 msec. Hence these EPSPs should have been induced monosynaptically. As shown in Figure G similar EPSPs were generated by stimulating VL with a latency of 1.0 to 1.2 msec. Concerning the fibre connection responsible for these VL-induced EPSPs, the following observation revealed a marked occlusion phenomenon between the VL and IP stimulation. As shown in Figure J, when the time interval for stimulating these two loci successively was relatively long, the respective EPSPs summated on each other without significant change in their size (controls in Figures H and I). However, when the interval was shortened to less than 1.0 msec, the response to the second stimulus decreased markedly, down to one third in the case of Figure K. Such a reduction of the succeeding EPSP as this was seen with either sequence of VL-IP or IP-VL stimulation. This occlusion phenomenon could be explained by assuming that the axons from IP innervate RN and VL commonly, and that stimulation at VL causes impulses to fire back to RN. In some cases shock stimuli were applied to AC. With relatively weak intensity, the AC stimulation did not induce postsynaptic potentials in the RN neurones, but it suppressed by about 20% the EPSP induced by excitation of IP. The suppression followed the AC stimulation immediately and lasted for several ten-milliseconds. The AC stimulation produces inhibitory postsynaptic potentials monosynaptically in IP neurones[11], and hence decreases the excitability of IP neurones. With relatively strong AC stimulation EPSPs were produced in the RN neurones. However, these EPSPs occurred with a latency comparable to those produced by the direct IP stimulation, and hence appeared to be caused by current spreading to IP from AC.

In Figure L EPSPs were generated in response to the shock stimuli applied to SM with a latency of about 1.2 msec. With a distance of 40 mm from SM to RN, it is likely that these EPSPs also are induced monosynaptically.

In conclusion, the RN neurones receive two monosynaptic excitatory connections, the one from IP and the other from SM. The former involves VL as the common target and is under inhibitory control by AC.

Résumé. Chez le chat anesthésié au Nembutal ou au chloralose, les réponses évoquées dans les neurones du noyau rouge par les stimulations antidromiques et orthodromiques ont été étudiées au moyen de microélectrodes intracellulaires.

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Inulin Clearance and Renal Lymph

In calculating the clearance of any substance excreted in the urine from the customary formula, relatively large errors occur whenever not all of the substance is carried away in the urine and *via* the renal vein. The error is the greater the more of the substance has undergone decomposition, been stored up by the kidney, and carried off in the renal lymph. Some workers[1-3] see the possibility that clearances calculated from the usual formula do not yield true values of the quantity of plasma cleared, because they fail to take into account the amount of substance transported in the lymph. Evidence has recently been produced that the amounts of *para*-amino-hippuric acid and inulin transported by the renal lymphatics are not large enough to invalidate the clearance values corresponding to the renal plasma flow and the glomerular filtration rate respectively[4-5].

As intravenous inulin is known to appear in readily measurable concentrations in the renal lymph[4,6-8] experiments were carried out to examine its influence on interpretation of clearance data obtained from the general formula.

Methods. From dogs urine was collected, after laparotomy, from the left ureter and lymph from one of the hilar lymph vessels in the left kidney. The clearance periods and the lymph collection periods were of the same duration. Inulin was determined by the procedure of LITTLE[9]. Inulin clearance was calculated from the well-known quotient

$$\frac{\text{amount of inulin excreted in the urine per min (mg/min)}}{\text{concentration of inulin in the plasma (mg/ml)}}$$

and

$$\frac{\text{amount of inulin in the renal lymph per min (mg/min)}}{\text{concentration of inulin in the plasma (mg/ml)}}$$

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Table I

No. of experiments	Weight (kg)	Period	Serum	Urine			Renal lymph		
			Inulin mg/100 ml	Flow ml/min	Inulin mg/100 ml	Inulin clearance ml/min	Flow ml/min	Inulin mg/100 ml	Inulin clearance ml/min
1	22.0	1	47.0	4.8	186.0	19.0	–	–	–
		2		5.1	165.0	17.9	0.02	25.0	0.01
		3	31.0	4.6	152.0	22.5	–	–	–
		4		5.7	144.0	26.5	0.02	25.0	0.02
2	29.0	1	16.5	5.7	160.0	55.3	0.10	16.0	0.09
		2	16.0	7.1	110.0	48.8	0.10	17.5	0.11
		3	17.5	8.0	114.0	52.1	0.09	17.5	0.09
		4	18.0	8.1	105.0	47.2	0.08	16.0	0.07
3	19.0	1	52.0	3.9	203.0	15.2	0.05	62.0	0.06
		2	49.5	4.0	203.0	16.4	0.05	64.0	0.06
		3	47.5	4.1	197.0	17.0	0.07	52.0	0.08
		4	46.0	3.7	208.0	16.7	0.08	51.0	0.09
Mean	23.3	–	35.8	5.4	162.2	29.5	0.07	34.6	0.07

Since the volume of lymph obtained from one of the hilar lymph vessels does not represent the total lymph volume, in this paper 'lymph inulin-clearance' is understood to mean inulin clearance of one hilar lymphatic.

Results and comments. With free urine flow, the concentrations of inulin in the lymph came on the whole quite near to those in the serum. Owing to this, the amounts of inulin transported by the lymph were but minute fractions of the inulin excreted in the urine (Table I).

In mechanical anuria the situation was found to be about the same (Table II). Accordingly, the amount of inulin transported by the renal lymphatics depends on the volume of lymph flow.

The total amount of lymph produced by a kidney is not known. The outflow from one lymphatic is less, and its tenfold – based on the assumption that the kidney contains ten efferent lymphatics – is much more, than the total production. It has been shown[10] that on cannulization of more than one efferent lymphatic, the outflow does not increase in proportion to the number of lymphatics cannulated. The 'surgical trauma' constituted by the preparation of the renal lymphatic may give rise to lymphangiospasm[11], which in turn impedes renal outflow. The values obtained in the present experiments for lymph flow are considerably lower than those stated by BULL and METAXAS[4]. The probable explanation for the discrepancy is that when these workers collected the lymph, the venous pressure in the kidney was raised due to shunt between the renal and the jugular vein. A rise in venous pressure considerably increases renal lymph flow[11–13]. The present experimental results appear to support the opinion of BULL and METAXAS[4] that under normal conditions the amount of the clearance substance in the lymph is without influence on its clearance.

Table II

No. of experiments	Weight kg	Serum	Renal lymph		
		Inulin mg/100 ml	Flow ml/min	Inulin mg/100 ml	Inulin clearance ml/min
3	23.0	3.2	0.04	4.5	0.06
4	14.0	6.2	0.02	2.5	0.01
5	18.0	10.0	0.03	10.8	0.03
6	25.0	5.9	0.05	–	–
8/a	20.0	5.4	0.04	4.4	0.03
8/b	20.0	4.7	0.04	3.8	0.03
9	24.5	5.0	0.05	2.1	0.02
10	33.0	13.0	0.05	6.3	0.03
11/a	15.5	7.4	0.13	5.7	0.10
11/b	15.5	5.9	0.13	7.2	0.16
12	16.5	7.4	0.04	11.3	0.06
13	25.5	3.7	0.02	3.6	0.02
Mean	20.8	6.5	0.05	5.2	0.05

The regression of the 'inulin clearance of the lymph', referred to lymph flow, is linear; the regression coefficient is 1.004. In oliguria, evaluation of any renal clearance is problematic. Difficulties may arise when it is associated with a considerable renal lymph flow. This is to be expected on occlusion of the ureter[14], particularly when the preceding minute diuresis has been high[11], on a rise in pressure within the renal vein[11–13], and during transfusion following the bleeding of the animal[2].

Zusammenfassung. Bei normaler Diurese ist die durch die renalen Lymphgefässe abgeführte Inulinmenge nur unbedeutend; sie beeinflusst die nach der üblichen Formel errechnete Inulinclearance nur unwesentlich.

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