(95 min) and so was not delayed by more than about 15 min; the failure to observe the earlier signs of poisoning suggests that this stage was actually reached during anoxia. Moreover, aldrin- or isodrin-treated insects actually recovered and behaved normally between the times of removal from nitrogen and the times of appearance of first signs of poisoning (that is, between 60 and 133 min). One conceivable explanation was that anoxia had delayed penetration of compounds III and IV through the insect cuticle considerably more than that of compounds V and VI. However, gas-chromatographic analysis of tissue extracts showed that following a dose of 1·1 μg of endrin in air, 0·14 μg of the unchanged insecticide was recovered from the tissues within the hour, and following a dose of 1·1 μg of isodrin in nitrogen, 0·13 μg of unchanged insecticide was recovered after 1 h. This shows that the delays indicated in Table 1 are not due to delayed penetration of the insecticide through the cuticle.

Table 1. Appearance of Signs of Poisoning in Adult Houseflies Exposed to Insecticides in Air or in Oxygen-free Nitrogen

	Insects dosed and kept in air at 25° C		Insects dosed and kept in nitrogen for 60 min and then observed in air at 25° C	
Compound	Time (min.) after treatment of first positive signs of poisoning, 'flight convulsions', etc.	Time (min) after treatment of 'knockdown' effects	Time (min) after treatment of first positive signs of poisoning, 'flight convulsions', etc.	Time (min) after treatment of 'knockdown' effects
Aldrin (III)	70	120	133	176
Isodrin (IV)	80	135	136	175
Dieldrin (V)	45	78	—	95
Endrin (VI)	45	80	—	90

These results show that aldrin and isodrin possess lower intrinsic toxicities than the corresponding epoxides. The higher polarity of the epoxides (as indicated by their higher R_F values under conditions of reversed-phase paper chromatography) may favour a more rapid penetration of these insecticides to the sites of action *in vivo*, possibly by permitting a more favourable partition between aqueous and lipid phases. Relevant is the observation that isodrin (0·37 μg), injected in 1 μl. of liquid paraffin, produced no signs of poisoning for 5 h in air, while with this dose of endrin signs of poisoning appeared in 2 h. This suggests that the more polar epoxide was released more rapidly into the hæmolymph from the injected oil. The insecticidal properties of aldrin or isodrin seem unlikely to be due entirely to epoxidation since the related dihydrocompounds (VII or VIII), which do not undergo epoxidation *in vivo*, are moderately toxic on their own account.

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Probable Mechanism regulating Water Economy of *Rhodnius prolixus*

Fifth-stage larvæ of *Rhodnius prolixus* decapitated immediately (10–20 min) after feeding have been shown[1] to excrete less urine than normal controls. These results apparently contradict the report of Maddrell that "ligating the fed insect anterior to the ganglionic mass has no effect on urine production".

To decide between these alternatives the amount of urine produced by three groups of animals was measured: (*a*) with all the abdominal nerves (originating at the large used ganglionic mass situated in the mesothorax) sectioned (the leg nerves were not sectioned); (*b*) with the connectives between the prothoracic ganglion and the mesothoracic fused ganglionic mass sectioned (these animals lose control over the two pairs of posterior legs); (*c*) controls operated without neurotomy.

20 h after the operation all animals were fed with oxalic (1 part per thousand) blood; their ani were previously plugged to avoid loss of excreted urine. 3–5 h after feeding they were dissected; the success of the operation was verified and the amount of urine produced was determined. The weight of the rectal contents was measured by difference with non-fed controls.

The values given in Table 1 confirm the earlier results obtained by decapitation in showing inactivity of the Malpighian tubes in all operated animals if operation is performed before feeding.

Table 1

	Weight of rectum (mg)		Weight of rectum contents (mg)	Weight of animals (mg)
Non-fed controls	3	(n = 5)*		40
Fed controls	14	(n = 12)	11	197
Group (*a*)	4·8	(n = 18)	1·8	155
Group (*b*)	4·9	(n = 7)	1·9	250

* n = No. of animals

The absence of diuretic hormone in operated animals (judged from the amount of urine accumulated in the rectum) may be attributed, according to preliminary observations, as follows: In animals of group (*a*), to the interruption of the neurosecretory pathway caused by sectioning of the axons of the neurosecretory cells (responsible for the production of the diuretic hormone) in the fused ganglionic mass (the absence of neurosecretion in these cells resembles that described by Scharrer[3] after section of the n.c. allatum). In animals of group (*b*), to the interruption of the central nervous efference (efference which is due, probably, to dilatation of the gut[4]).

Furthermore, unpublished results show in normal animals the existence of a peripheral inhibition of central origin which is exerted on the abdominal wall and which can be demonstrated on sectioning the abdominal nerves on one side. This inhibition, probably related to that found by Dethier[5] in *Phormia*, could serve as a feedback mechanism (via the hydrostatic pressure in the gut) for the regulation of the time of ingestion in *Rhodnius*.

The apparent contradiction with Maddrell's results may be due to the fact that already during ingestion there is liberation of some diuretic hormone since urine production starts in the first 15 min from the beginning of feeding.

It remains to be decided whether in the input of the regulating circuit there operate signals of abdomen volume apart from those signals which arrive through the prothoracic-mesothoracic connective.

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Mating Behaviour of *Anopheles stephensi*

The successful eradication of screw worm fly from Curaçao and from the southern United States has created a great interest in the possibility of insect eradication by release of sterile males[1]. Even where complete eradication cannot be achieved, the method can still be used for limiting the population density of harmful species to a level where it ceases to be of importance. If this can be done for a certain length of time in the case of vectors of diseases, it is possible that the disease-cycle may be completely interrupted and the disease itself eradicated.

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