C mg/cm³	N	R	$M(x, y)$	$Po \pm s$	Oo
20	26	− 0,76	2,35/146,5	− 244 ± 40	227
55	17	− 0,93	2,61/223	− 469 ± 50	508
150	17	− 0,84	2,54/472	− 690 ±110	845

dit-on, que la vidange gastrique est inhibée par le glucose présent dans l'estomac, d'autant plus longuement que la concentration est élevée: celle-ci peut varier dans de larges limites de 50 à 250 mg/cm³ selon sans que la vitesse change[3].

Des travaux antérieurs nous ayant conduit à attribuer un rôle régulateur essentiel, et tout à fait indépendant de la vidange gastrique[4] aux modifications de la glycémie[5,6] nous nous sommes demandé comment se comporterait l'animal intact si l'on maintenait sa glycémie constante pendant qu'il absorbe des solutions de differentes concentrations, supprimant ainsi le deuxième mécanisme régulateur, celui de la glycémie, mais non le premier, celui de la vidange gastrique dont nous nous demandons qu'elle peut être l'importance.

Sur le cobaye, avec les techniques décrites[6–8], nous avons comparé trois solutions contenant 20, 55 et 150 mg/cm³, non point à une glycémie unique, mais en faisant un test d'ensemble sur les glycémies comprises entre 100 et 1000 mg %. Utilisant pour cela la corrélation négative qui, nous l'avons montré[7], existe entre les vitesses d'absorption et les glycémies, nous avons calculé, par la méthode des moindres carrés, puis comparé entre elles, les trois droites de régression représentant la fonction: vitesse d'absorption/log. des glycémies.

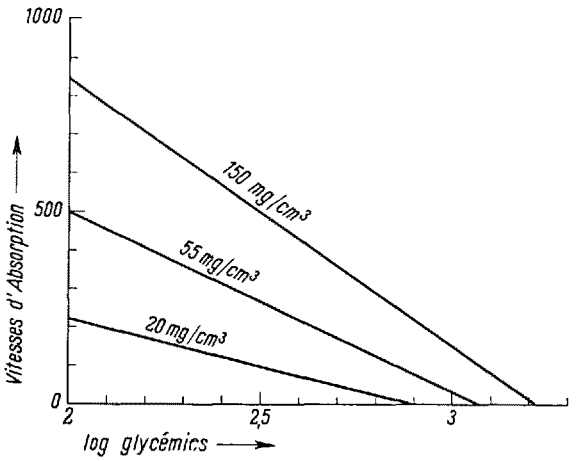
Pour chaque solution, nous avons donc mesuré le glucose absorbé, en 1 h, à des glycémies diverses obtenues par des injections isolées ou simultanées de glucose et d'insuline[7,8]; puis nous avons calculé les éléments du tableau, c'est à dire, avec les concentrations C et les effectifs N, les statistiques des droites de régression y en x: $M(x,y)$ coordonnées des centres de gravité, $Po \pm s$ pentes et écarts types, Oo ordonnées à l'origine.

Les droites ont été tracées dans la Figure (abscisses: log. des glycémies en mg %; ordonnées: vitesses en mg/h).

Les résultats peuvent être résumés ainsi:

1° les trois coefficients de corrélation sont significatifs au seuil de probabilité $1^0/_{00}$[7].

2° Les droites diffèrent à la fois par leur position dans le plan de Figure et par leur pente. Elles coupent l'axe des x en des régions voisines, mais les pentes croissant en valeur absolue avec les concentrations, elles s'écartent l'une de l'autre dans la région des faibles glycémies. Expérimentalement, cela signifie que les différences de vitesse sont d'autant moins marquées que les glycémies sont plus élevées, ce qui est le cas dans une épreuve d'absorption ordinaire. A ce facteur régulateur s'ajoute le fait que l'hyperglycémie est d'autant plus forte que la solution ingérée est plus concentrée. Maintenant si l'on se place à une glycémie normale (100 mg %) les vitesses, qui, dans la Figure, sont représentées par les ordonnées à l'origine, sont de 227, 508, 845 mg/h. Elles augmentent donc de 4 fois, et pour les deux dernières solutions qui, dans les expériences citées en [3] étaient absorbées à la même vitesse, nous trouvons une différence de 66%.



Il est possible qu'en augmentant les concentrations, les vitesses atteignent une limite. Mais pour les solutions utilisées, notre schéma expérimental montre que la seule présence de glucose dans l'estomac n'inhibe pas la vidange gastrique au point de rendre la vitesse d'absorption indépendante de la concentration. Ce sont les modifications de la glycémie qui jouent un rôle déterminant: ce sont elles qui ralentissent l'absorption et empêchent l'estomac de délivrer de nouvelles quantités de glucose. L'inhibition de la vidange gastrique apparaît ainsi comme la conséquence du défaut d'absorption et non comme sa cause.

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Summary

In intact guinea-pigs fed with glucose solutions of different concentrations, the main limiting factor of absorption is not the gastric emptying but the hyperglycaemia.

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[6] M. Lourau, C. R. Acad. Sci., Paris, *238*, 842 (1954).
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Response of Adrenal and Preputial Glands of Rats to Administration of 5-Hydroxytryptamine

It is well known that 5-hydroxytryptamine (5-HT) induces enlargement of the adrenal glands[1]. Hypertrophy of the adrenals and increased urinary output of 17-hydroxycorticoids after administration of reserpine, a 5-HT liberator, have been reported[2]. Moreover, it has been suggested that adrenal glucocorticoids may exert a functional control of the tissue level of 5-HT[3].

The relationship between 5-HT and the endocrine system has not been extensively studied, however. For

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Weight and Ascorbic Acid Content of Adrenal and Preputial Glands of Intact, Ovariectomized, Adrenalectomized and Ovariectomized-Adrenalectomized Female Rats, After a Single Injection of 5-HT.

	Body weight g	Adrenal gland weight mg	Adrenal ascorbic acid µg/100 mg tiss.	Preputial gland weight mg	Preputial ascorbic acid µg/100 mg tiss.
Intact					
Controls	162	50·3 ± 9·5	280·2 ± 43·9	52·1 ± 13·6	69·9 ± 15·4
Injected with 5 HT	161	54·3 ± 7·4	214·8 ± 33·4	65·6 ± 18·8	61 ± 24
% variation		+ 7·9	− 23·4	+ 25·9	− 12·8
		$p < 0·5$	$p < 0·01$	$p < 0·1$	$p < 0·5$
Ovariectomized					
Controls	153	50·2 ± 7·2	398·5 ± 39·2	47·1 ± 7·1	104·4 ± 20·4
Injected with 5 HT	154	48·7 ± 5	307·1 ± 66·6	51·7 ± 16·5	84·9 ± 20·4
% variation		− 3	− 23	+ 9·7	− 18·7
			$p < 0·001$	$p < 0·5$	$p < 0·1$
Adrenalectomized					
Controls	154			62·7 ± 9·8	92·6 ± 10·7
Injected with 5 HT	153			79·5 ± 23·5	75·7 ± 14
% variation				+ 26·7	− 18·3
				$p < 0·05$	$p < 0·01$
Ovariectomized-Adrenalectomized					
Controls	150			55·3 ± 18·6	114·3 ± 30·6
Injected with 5 HT	154			62 ± 18·7	89·6 ± 22·5
% variation				+ 12·1	− 21·7
				$p < 0·5$	$p < 0·05$

this purpose the effects of a single injection of 5-HT on the adrenal and preputial glands were examined, the latter being considered a target organ of corticotrophin action[4]. In order to evaluate this response, the endocrine sources of steroid hormones (viz. ovaries and adrenals) were removed.

Material and methods. Eighty albino female Wistar rats with a body weight of 150–160 g were employed. They were kept on Rockland diet and fed *ad libitum*. Four series were treated: intact, ovariectomized, adrenalectomized, and ovariectomized-adrenalectomized. The surgical operations were performed under ether anaesthesia four days before the beginning of the experiments. Adrenalectomized rats were maintained on drinking fluid containing 1% sodium chloride. Each series consisted of ten rats treated with 5-HT injected subcutaneously and ten controls. The dose was 10 mg/kg dissolved in saline[5]. The controls received saline only. All rats were sacrificed by decapitation 2 h after the injection. The adrenal and preputial glands were weighed and the percentage values of their ascorbic acid content determined[6].

Results. The data are summarized in the Table. The weight of the adrenals was unchanged after the 5-HT treatment. The adrenal ascorbic acid content was remarkably decreased, both in intact and ovariectomized rats (23·4% and 23·0% respectively). The increase in preputial gland weight was 25·7% in the series of intact rats, being almost unchanged in the adrenalectomized (26·7%). The increase was less, however, in ovariectomized and ovariectomized-adrenalectomized rats (9·7% and 12·1% respectively). The preputial ascorbic acid content was decreased in all series, most remarkably in those operated on.

Discussion. The adrenal ascorbic acid depletion observed in this experiment is in accordance with the results obtained by BERTELLI *et al.*[7], and seems to confirm previous reports concerning the effects of 5-HT treatment on the pituitary-adrenal axis, mediated by an ACTH-releasing effect[8]. The mediation by ACTH was also suggested by the observation that 5-HT induces no depletion of adrenal ascorbic acid in hypophysectomized rats[8] and by lacking eosinopenic response in adrenalectomized ones[9].

In the present experiment, a parallel depletion, although less marked, was found in the ascorbic acid content of the preputial glands after administration of 5-HT. This is in accordance with the results obtained by HESS *et al.*[10] concerning the depletion of preputial ascorbic acid induced by severe stress in intact rats, and by high doses of ACTH in hypophysectomized rats.

Moreover, an increase in perputial weight was observed in all series after injection of 5-HT. This increase was moderate in the ovariectomized animals (9·7%), but remarkable in the adrenalectomized (26·7%). Thus, through an uninhibited ACTH liberation, simple adrenalectomy may induce an increase in weight of the preputial glands (20·3% as compared with the controls of the intact series). The still higher increase in preputial weight of adrenalectomized rats injected with 5-HT (52·5%) also indicates an ACTH-releasing effect of this substance.

The ovariectomy, on the contrary, seems to inhibit the increase in preputial weight induced by 5-HT treatmens. We notify, however, that in another group of rats (not

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reported in the Table), the simple laparotomy induced a decrease of 14·3% in the weight of preputial glands.

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Zusammenfassung

An weiblichen intakten, adrenal- oder ovariektomierten und sowohl adrenal-, als auch ovariektomierten Ratten wurden 2 h nach Injektion von 5-Hydroxytryptamin die Gewichtsveränderungen und der Ascorbinsäuregehalt der Nebennieren und der Präputialdrüsen untersucht. Eine Abnahme des Ascorbinsäuregehaltes der Nebennieren und der Präputialdrüsen wurde festgestellt und eine Gewichtszunahme der Präputialdrüsen selber beobachtet. Die Ergebnisse stimmen mit den bekannten Befunden über eine ACTH-freisetzende Tätigkeit des 5-Hydroxytryptamins überein.

Identification of a Third Subdivision of the Dorsal Spino-Cerebellar Tract

Ascending tracts in the dorso-lateral funiculus (Flechsig's fasciculus) have been analyzed by recording the mass discharge from the dissected fasciculus and by intracellular recording from single fibres. Four types of ascending pathways have been differentiated[1].

A. Neurones monosynaptically activated by impulses in muscle spindle afferents (Ia and II).

B. Neurones monosynaptically activated by impulses in Golgi tendon organ afferents.

C. Neurones activated by impulses in group II and III muscle afferents, and also by impulses in skin and joint afferents. These excitatory actions are drawn from an extremely wide ipsilateral receptive field. On adequate stimulation, a discharge can be evoked by muscle stretch and also from large areas of skin (usually by pressure and pinching, but sometimes also by bending of hairs).

D. Neurones activated exclusively by ipsilateral low threshold cutaneous afferents from a very restricted receptive field, the discharge being adequately elicited by bending of hairs.

There was reason to assume that type A and B constitute the dorsal spino-cerebellar tract, because, on stimulation of group I muscle afferents, a potential can be recorded from the anterior cerebellar vermis, which corresponds to the discharge in these neurones[1]. Stimulation of group II and III muscle afferents, on the other hand, did not evoke a potential change in the cerebellar cortex; hence it could not be assumed that the axons of type C terminated in the cerebellar cortex.

In the present experiments, a more direct approach has been used for identification of axons belonging to the dorsal spino-cerebellar tract by investigating whether they can be activated antidromically from the cerebellar cortex.

Of 67 neurones belonging to type A and B, 50 could be activated antidromically from the anterior vermis. Those 17 axons which could not be activated antidromically may have terminated in the most rostral part of the vermis, not exposed for stimulation. None of 47 axons tested could be activated by stimulation of the dorso-lateral funiculus in the proximal L V segment, as would be expected since Clarke's column does not extend caudally below the L IV segment.

63 neurones belonging to type C were identified; 36 of them could be activated antidromically from the anterior cerebellar vermis. Stimulation of the dorso-lateral funiculus in upper L V failed to activate the axon of any type C neurone with connection to cerebellum. Hence it can be concluded that neurones of type C constitute a third subdivision of the dorsal spino-cerebellar tract.

However, it is noteworthy that the proportion of neurones not activated antidromically from the cerebellum is larger with type C than with A and B (43 resp. 25%). There is, indeed, evidence of another ascending system of type C because of 22 neurones, which could not be activated from cerebellum, 11 were activated on stimulation of the dorso-lateral funiculus in L V and consequently have their cell bodies below the caudal end of Clarke's column. No experiments have been made to find the termination of these axons.

None of the units of type D could be activated antidromically from the cerebellum, and with all of these neurones the axons could be stimulated in L V.

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Zusammenfassung

Aktionspotentiale einzelner Fasern des Funiculus dorsolateralis des Rückenmarks wurden registriert und dabei nach Axonen gesucht, die antidrom von der Kleinhirnrinde erregt werden konnten. Es gelang die Identifizierung einer dritten Unterabteilung der dorsalen spino-cerebellaren Bahn. Diese Neurone werden sowohl durch Muskelafferente der Gruppen II und III als auch durch Haut- und Gelenkafferente erregt. Zwei andere aufsteigende Bahnen, deren Zellkörper unterhalb des caudalen Endes der Clarkschen Säule liegen und deren Axonen sich in dem Funiculus dorsolateralis befinden, erreichen die Kleinhirnrinde nicht.

The Relationship Between the Flexion Reflex and Certain Ascending Spinal Pathways

In spinal cats, impulses in group II and III muscle afferents, in skin and in high threshold joint afferents give rise to the flexion reflex with excitation to flexor and inhibition to extensor motoneurones[1]. In decerebrate cats, these actions are markedly depressed or even absent because the interneurones mediating them are tonically inhibited from suprasegmental centres[2].

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