Table II

Action of Insulin on the Glucose Uptake and Oxigen Consumption of the Spinal Cord. Buffer Gey and Gey. Not anesthetised

	Mean	σ	t	P	No. experiments
Glucose uptake mg × 100 mg of tissue					
Insulin 10^{-1}	0·328	0·027	1·867	$0{\cdot}05 < P < 0{\cdot}1$	14
Control . .	0·304	0·033			14
Oxygen consumption: microlitres × 100 mg of tissue × h					
Insulin 10^{-1}	12·3	1·06	1·1211	$0{\cdot}2 < P < 0{\cdot}3$	14
Control . .	12·7	0·65			14

In Table III the results are obtained by exactly following RAFAELSEN's technique: that is to say, after previously anesthetising the rat with a mixture of 50% CO_2/O_2. In this kind of test, the spinal cord appears sensitive to the action of insulin because the difference in glucose consumption of the tissue with and without insulin is significant. There is, however, no significant difference in the oxygen consumption.

Table III

Action of Insulin on the Glucose Uptake and oxigen Consumption of the spinal Cord. Buffer Gey and Gey. Anesthetised with a CO_2/O_2 mixture at 50%

	Mean	σ	t	P	No. experiments
Glucose uptake: mg × 100 mg of tissue					
Insulin 10^{-1}	0·485	0·021	3·8693	$< 0{\cdot}01$	9
Control . .	0·290	0·029			9
Oxygen consumption: microlitres × 100 mg of tissue × h					
Insulin 10^{-1}	17·1	2·07	0·5263	$0{\cdot}6 < P < 0{\cdot}7$	8
Control . .	16·6	1·38			8

Discussion. We were able to confirm RAFAELSEN's results, although our own data give them a different interpretation, because we observed that the spinal cord is *only sensitive* to insulin when the rat has been previously anesthetised with CO_2. The basal glucose-uptake is the same in the experiments but in the test made without anesthesia, no effect is produced when adding this hormone. Similar results have obtained URELES and MURRAY[6], because they have found that r. c. b. uptake of I^{131} triiodothyronine increases either with marked CO_2 retention (patients) or when the blood has been bubbled with CO_2 into the flask *in vitro*.

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Résumé

La moelle épinière ne paraît pas sensible à l'insuline *in vitro* si elle provient d'un animal décapité alors qu'elle l'est de façon significative ($P < 0{,}01$) si l'animal était anesthésié avec un mélange de CO_2/O_2 à 50%.

[6] A. URELES and M. MURRAY, cited by M. W. HAMOLSKY *et al.*, J. clin. Endocrin. Met. *19*, 103 (1959).

Ion Adsorption and Excitation II

It has been shown in a previous communication[1] that the internal and external ion concentrations of nerve can be related to the net quantity of ions fluxing during the passage of a single impulse by the expression $f = A\,r\,(C_i - C_0)$. It was also deduced that the ions involved in these fluxes form a monolayer at the nerve surface and that they are not hydrated. This was taken as an indication that excitation may be accompanied by an adsorption-desorption process.

The quantity Ar has been defined as that volume of the nerve substance in which the ionic concentration corresponds to that of the nerve interior or to that of the bathing solution depending on the presence or absence of the flux quantity.

The advantage of this formulation is that by summating the flux values over all such unit volumes available, the gross ion concentration difference between the axoplasm and the bathing fluid can be expressed in terms of the flux quantity characteristic of a single discharge.

Since Ar can also be regarded as a sort of 'extended surface', being in fact only one half of an ion thick and situated presumably at the functional surface of the nerve, this surface may be considered as having the same ionic composition as the nerve interior. This suggests that the forces which operate in maintaining the ionic composition of the nerve interior are identical with those which maintain the ionic composition of the nerve surface. Conveniently these forces may be characterized as adsorption forces, opposite and equal in magnitude to the potentials as given for each ionic species by the relation $E = kT/e \ln (C_i/C_0)$.

It follows from such a view that since the surface monolayers of the major monovalent ions can be regarded as spatially superimposed, the resting potential of the cell may be assumed to correspond to the algebraic sum of the potentials due to each of the major ions represented at the surface. This leads to the expression

$$R.P. = kT/e \ln(K_i/K_0 \times Na_i/Na_0 \times Cl_0/Cl_i).$$

Taking the external concentrations of K and Na ions as $K_0 = 22$ mM/kg and $Na_0 = 440$ mM/kg[2] and the internal ones as $K_i = 345{\cdot}3$ mM/kg and $Na_i = 62{\cdot}5$ mM/kg[1], and using the value of 59 mV for the corrected resting potential of the squid giant axon[3], one obtains from the above formula the ratio $Cl_0/Cl_i = 4{\cdot}5$.

According to the data of BEAR and SCHMITT[4] this ratio is about 4, the agreement between this value and the one calculated above suggesting that the assumption involved in the formulation is essentially correct.

I wish to thank Dr. B. W. ZWEIFACH for his kind advice and interest.

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Résumé

Une façon simple de formuler le potentiel de repos de l'axone géant du Calmar est présentée. L'équation est

[1] E. ASCHHEIM, Science *129*, 779 (1959).
[2] A. L. HODGKIN, Biol. Rev. Cambridge Phil. Soc. *26*, 339 (1951).
[3] A. L. HODGKIN and B. KATZ, J. Physiol. *108*, 37 (1949).
[4] R. S. BEAR and F. O. SCHMITT, J. cell. comp. Physiol. *14*, 205 (1939).

fondée sur une démonstration antérieure prouvant que pendant l'excitation les cations importants forment une seule couche à la surface fonctionnelle du nerf. La formule est employée pour calculer la distribution du chlorure. Le résultat est en accord avec les valeurs expérimentales publiées.

Effects of Chronic Lesions of the Peduncles on Cerebellum Cholinesterase Activity, in the Albino Rat

In a previous paper[1], the high true cholinesterase activity of cerebellum – predominantly localized in cerebellar cortex – has been related to cerebello-petal tracts, with the tentative purpose of applying available information on the ACh system in cerebellum to a cholinergic mechanism of afferent fibres. The hypothesis was supported by: (a) the differential distribution of true cholinesterase over the structurally uniform cerebellar cortex; (b) the sensitivity of some intracortical neurons to acetylcholine; (c) the presence of cholineacetylase in some cerebellar afferent systems.

The present paper concerns the fate of cerebellum cholinesterase after unilateral lesions involving, more or less extensively, cerebellar peduncles.

Adult albino rats of the Wistar strain were used throughout. Surgical procedure was performed aseptically, under nembutal anesthesia. Peduncles were approached from the IV ventricle, the extent and the location of the lesion being reconstructed on necroscopic examination. In a few animals only the opening of the IV ventricle was performed (sham operation).

Estimations of enzyme activity were carried out on the 10th day following the operation, by the Warburg manometric method, as previously described[1]. The two halves of the cerebellum of operated animals were tested separately, while cerebella of controls were tested *in toto*, having previously stated the same level of activity in the two halves. All estimations were carried out in duplicate, using acetyl-β-methylcholine and butyrylcholine as 'specific' substrates for true (ChEI) and pseudo-(ChEII) cholinesterase, respectively. Fresh solutions of substrates were used in each experiment, the final concentrations being acetyl-β-methylcholine 0·03 *M* and butyrylcholine 0·01 *M*. Results are expressed as μl of CO_2 evolved/h/g of wet weight of tissue.

The Table gives the results – both as absolute values and as per cent fall in activity – of seven animals out of a total of twenty operated. Results were consistent for similar types of lesions, and only the typical examples have been reported so as to summarize the course of events during progressive change of location and extent of peduncular lesion. Values refer to ChEI, no significant variation having ever been found in ChEII activity.

Unilateral lesions of cerebellar peduncles are followed by a fall in cerebellum ChEI content, the value depending on the extent and location of the lesion and reaching its maximum when all three peduncles are severed. Maximal drops in activity range around 60% in the ipsilateral side and 40% in the controlateral one, each peduncle contributing to a certain extent, as roughly indicated by results of partial lesions.

[1] L. Sperti, S. Sperti, and P. Zatti, Arch. ital. Biol. sper. in press.

Table

True cholinesterase activity of rat cerebellum after unilateral lesions involving cerebellar peduncles

	Homolateral half		Controlateral half		Control (*in toto*)
	Q MeCh	%	*Q* MeCh	%	*Q* MeCh
Sham operation	1014		1008		994
A	869	– 13	988	– 1	995
B	785	– 24	1044	0	1030
C	874	– 22	1024	– 8	1119
D	468	– 52	860	– 12	977
E	598	– 42	880	– 15	1030
F	487	– 50	817	– 16	976
G	388	– 63	635	– 39	1042

ICP = inferior cerebellar peduncle; MCP = middle cerebellar peduncle; SCP = superior cerebellar peduncle

Q MeCh = μl CO_2/g wet weight of tissue/h with acetyl-β-methylcholine as substrate; % = fall in activity as per cent of the control tested in the same experiment, which was of the same litter of the operated animal

Figures for sham operation are the mean of three experiments. Drawings roughly indicate location and extent of lesions

The disappearence of ChEI – in cerebellum predominantly localized in the cortex[1–4] – is clearly related to the degeneration of afferent fibres (partially crossed), the efferent ones – almost entirely nuclear in origin – being out of question.

As to the behaviour of ChEI in Wallerian degeneration, afferent tracts to cerebellum seem then to resemble pre-

[2] A. S. V. Burgen and L. M. Chipman, J. Physiol. *114*, 296 (1951).

[3] S. C. Shen, P. Greenfield, and E. J. Boell, J. comp. Neurol. *102*, 717 (1955).

[4] G. B. Koelle, J. comp. Neurol. *100*, 211 (1954).