the observed tissue distribution of ^{14}C even during the first 10 min after injection of the chyle lipoproteins cannot be assessed from our data[21].

^{14}C Distribution in alloxan diabetic rats

10 min		2 h	
Insulin maintained	Insulin deficient	Insulin maintained	Insulin deficient
% ^{14}C removed from plasma found in abdominal fat and skeletal muscle:			
Fat			
13.5 ± 1.8	5.3 ± 0.6	14.3 ± 1.2	3.5 ± 0.4
Muscle			
9.6 ± 1.7	19.1 ± 2.3	7.7 ± 0.4	20.8 ± 2.0
% of injected ^{14}C in expired CO_2:			
0.1 ± 0	0.3 ± 0	2.6 ± 0.5	19.3 ± 1.2

Values are means ± S.E.

Résumé. Chez des rats diabétiques, l'arrêt du traitement insulinique est suivi (a) d'une augmentation de la proportion des acides gras triglycériques du chyle marqués du ^{14}C prelevée dans le sang par les muscles du squelette après injection des triglycériques, (b) d'une diminution de la proportion prelevée par les depots gras abdominaux, et (c) d'une augmentation de leur oxydation en CO_2 expiré.

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The Effect of Chlorpromazine on the Adrenal Gland in Rats with Brain Stem Lesions

It is well known that the central nervous system plays an important role in the control of pituitary ACTH secretion. Several data have been published indicating that this control is exercised through the hypothalamus[1] and that the mesencephalic reticular formation (MRF) plays also a role in this mechanism[2,3].

Since it has also been held that chlorpromazine (CPZ) has an inhibiting action on the MRF[4,5] we have tried in the present work to establish the action of lesions in the MRF and simultaneous administration of CPZ, on the pituitary-adrenal axis, using the ascorbic acid depletion of the adrenals as a functional test[6].

Four groups of white male rats weighing between 180 and 220 g were used (Table). The first group included absolute controls and was injected with saline s.c. during 15 days. Those of the second group received CPZ s.c. in daily doses of 5 mg/kg during 15 days. On those of the third and fourth groups, electrolytic lesions were performed bilaterally in the MRF with a stereotaxic apparatus[2] under pentobarbital anaesthesia and using an intensity of 3 mA for 10 sec. After these localized lesions no changes in the behaviour and sleep-wakefulness cycles of the animals were detected. Two weeks after this operation the animals of the third group started receiving daily injections of saline, while those of the fourth group received daily injections of CPZ 5 mg/kg during the same period.

All animals receiving CPZ, in which indifference to the environment and to the observer was apparent, were fed through a gastric catheter to avoid the side effects and unspecific stress of starvation.

24 h after the last injection all the animals were killed by bleeding under pentobarbital anaesthesia, and the adrenal glands removed and weighed in a torsion balance. After this, the right glands were fixed in formalin-calcium for histological study (results will be published elsewhere), and the left ones were used for ascorbic acid determination[7].

In groups III and IV the actual site and size of central nervous system lesions were established through the study of serial sections. Lesions were found in the periaqueductal grey matter between the nuclei of the III and IV cranial nerves and its adjoining reticular formation.

As seen in the Table, body weight of the animals in the 4 groups did not vary significantly, indicating that the experimental procedures did not modify their nutritional state.

No significant differences in adrenal weight were detected between the animals of the 4 groups. Nevertheless, adrenal ascorbic acid content showed meaningful variations; thus, CPZ administration (group II) produced a significant increase of it, while after MRF lesions (group III) a highly significant depletion of the ascorbic acid content of adrenal glands was observed.

In a control group (not included in the Table), lesions performed in other areas of the mesencephalon including the medial and lateral lemnisci were not effective in modifying the ascorbic acid content of the adrenals.

The administration of CPZ during a period of 15 days was incapable of restoring the ascorbic acid depletion produced by MRF lesions in group IV.

Our results indicate that the lesions at the MRF level and the administration of CPZ have both a considerable

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Action of MRF lesions and CPZ administration on ascorbic acid content in adrenals of rats

	No. of animals	Initial weight (g ± s.e.)	Final weight (g ± s.e.)	Adrenals (mg ± s.e.)	Ascorbic acid content (mg% ± s.e.)
Control group	33	201 ± 2.2	210 ± 3.3	40.0 ± 4.5	375 ± 9.8
CPZ	20	200 ± 2.0	206 ± 3.5	49.5 ± 2.4	416 ± 13.5[a]
MRF lesions	20	222 ± 5.7	247 ± 7.8	46.0 ± 1.8	271 ± 22.0[a]
MRF lesion + CPZ	14	219 ± 5.4	244 ± 8.2	42.0 ± 1.6	288 ± 21.8[a]

s.e., standard error; [a], $P < 0.01$.

influence on adrenal function, although apparently acting in opposite directions.

The changes observed on the ascorbic acid content of the adrenals after the MRF lesions cannot be related to unspecific stress because animals of groups III and IV were sacrificed 30 days after performing the lesions. Further, lesions in other areas of the mesencephalon close to the MRF did not modify the ascorbic acid content of the adrenals, tending to corroborate the role of this area in the control of pituitary ACTH secretion, as had been shown in previous works[2,3].

The fact that CPZ administration during a long period of time was unable to increase the ascorbic acid in adrenals depleted as a result of strictly localized MRF lesions tends to indicate that the action of CPZ on adrenal function is not a direct one but conveyed through MRF inhibition.

Resumen. Lesiones de la formación reticulada mesencefálica (FRM) provocan una caída significativa del ácido ascórbico supprarrenal (AAS). La clorpromazine (CPZ) provoca un aumento significativo del AAS. La administración de CPZ a ratas con lesión en FRM es incapaz de restablecer los niveles de AAS.

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Isotope Scanning of Subdural Effusions in Infancy

Subdural effusion in infancy is infrequent; its diagnosis is, however, important since lack of adequate treatment may cause neurological complications and mental retardation[1]. Subdural effusion may be caused by natal or postnatal trauma[2], meningitis[3,4], diarrhoea[5], malnutrition[6] or excessive CSF-taps[5], or without specific cause[2].

The principal clinical method of detection of subdural effusion is subdural puncture at the extreme lateral angle of the fontanel. Although positive subdural puncture is diagnostic it provides little information on the size, exact location and shape of the effusion, and of its connections, if any, to the contralateral side. Radiological examination, with air injected into the effusion space, provides additional information on the cavity[7]. On the other hand, fluid mixes with fluid more readily than air. Following this principle, investigations were carried out to develop a new method for the determination of the space of subdural effusion, using radioisotopes.

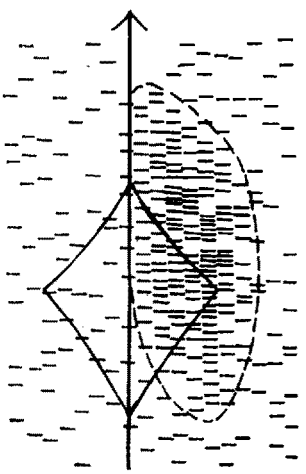

Fig. 1. The scan of the right-side effusion space. 1 μC I^{131}-Hippuran, with subdural puncture 13 ml, 'isotope dilution volume' 39 ml (see text, case 1).

Method. The isotope used was I^{131}-Hippuran, absorbed from the cerebrospinal fluid and eliminated from the organism definitely more rapidly than radio-iodinated human serum albumin. The dose used, 1–4 μC, is absolutely safe[8], and considerably smaller than that used in isotope ventriculography[9].

The isotope scanning of subdural effusion was performed as follows. When subdural effusion was suspected,

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