Table III. Effect of histamine on guinea-pig mast cell damage induced by promethazine or chlorcyclizine

Histamine	Promethazine 0.1 m*M*			Chlorcyclizine 0.1 m*M*		
	Average	Range	% of control	Average	Range	% of control
0	3.6 (6)	0.3–10.5	14.3	1.4[a]	1.4–1.4	5.4
0.1 m*M*	5.1 (4)	0.9–10.7	20.3	2.4	1.3–3.5	9.3
0.25 m*M*	5.7 (4)	1.0–18.2	22.7			
0.5 m*M*	12.8 (4)	2.4–23.2	51.0	5.3	4.6–6.1	20.6
0.75 m*M*	14.0 (2)	7.0–17.0	55.7			
1 m*M*	24.2 (6)	15.6–29.6	96.4	25.9	23.1–28.8	100
Control[b]	25.1 (6)	15.2–38.6	100	25.7	24.5–26.9	100

The results are given as the mean count of mast cells in 30 microscopical fields. Values in brackets indicate the number of experiments performed. [a] Mean of 2 experiments. [b] Incubated in tyrode alone.

whether the site of this antagonism is at the granule, cell membrane or some other cell site. However, since the high concentration of histamine normally found within the mast cell granule does not prevent the demaging action of antihistamines, these drugs are probably disrupting the mast cells and releasing histamine by a mechanism other than a direct displacement of the amine from the granules; exogenous histamine would thus be antagonising the antihistamines at a different site of the cell. Antihistamines with different chemical structures showed different ratios antihistamine:histamine (1:1 for diphenhydramine, and 1:10 for promethazine and chlorcyclizine) necessary for a complete protection of the mast cells. Preliminary experiments showed that chlorpromazine (0.0001 *M*), which has a much lower antihistamine activity than promethazine, damaged 100% of guinea-pig mast cells, but a concentration of 0.005 *M* of histamine was necessary for a complete protection of the mast cells, 5 times that necessary for promethazine.

The fact that the inhibitory action of histamine was rather easily overcome – it was sufficient to raise the ratio diphenhydramine:histamine from 1:1 to 1.5:1 for the protection to disappear – suggests that the histamine added is not very firmly bound to the cell. Our results do not imply as yet the existence of a relationship between chemical structure or potency of the antihistamine and antagonism by histamine at the mast cell level. More experiments are being performed to verify any such relationship and the nature and specificity of this antagonism.

Resumen. Se demonstra la capacidad de la histamina para inhibir las alteraciones de los mastocitos de la rata y cobayo producidas por diversos antihistamínicos (difenhidramina, prometazina, clorciclizina, clorpromazina). Diferentes concentraciones de histamina fueron necesarias para antagonizar los distintos antihistamínicos. Se sugiere la existencia de un antagonismo antihistamínico – histamina a nivel mastocitario.

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Severe Alterations in Myelin Structure in Experimental Lymphogenous Encephalopathy

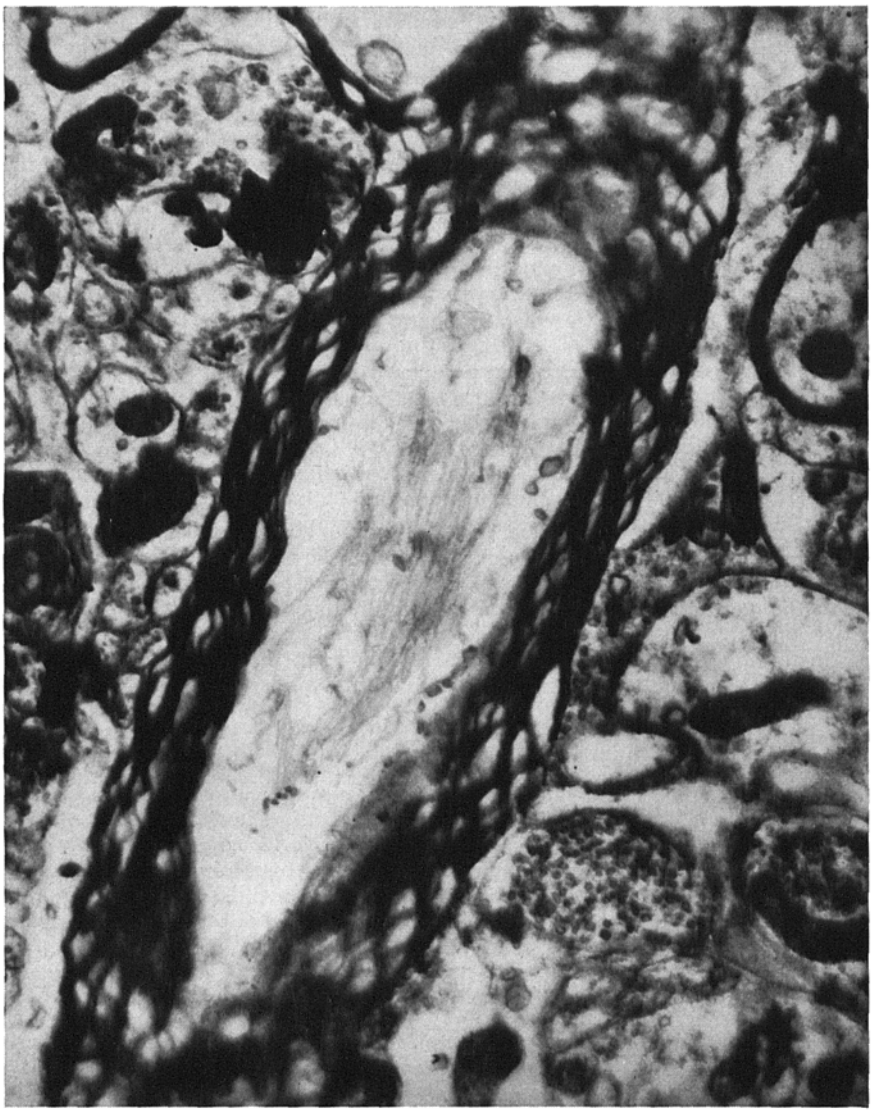
It has been reported in a series of papers that, in striking contrast to the text book opinion, lymphatics play a fundamental role in the fluid circulation of the brain[1-8]. A blockade of cervical lymph vessels and glands results in an experimental disease – lymphogenous encephalopathy – characterized by various neuropathological, biochemical and clinical signs.

Submicroscopic alterations in the grey substance of the brain are conspicuous 3 days after surgery; swelling of mitochondria, enlargement of the perivascular cisternae and the appearance of lysosomas. No alterations were found in the white matter at this time, however[9].

Further examinations of rats suffering from lymphogenous encephalopathy revealed that 7 days after surgery severe alterations in myelin structure became apparent.

Instead of geometrically regular concentric organization of the lamellae constituting the normal myelin

[1] M. Földi, E. Csanda, F. Obál, I. Madarász, G. Szeghy and Ö. T. Zoltán, Z. ges. exp. Med. *137*, 483 (1963).
[2] M. Földi, Arch. Kreislaufforsch. *41*, 186 (1963).
[3] M. Földi, E. Csanda, G. Szeghy and L. Varga, Klin. Wschr. *40*, 598 (1962).
[4] E. Csanda, Ö. T. Zoltán and M. Földi, Lancet, *1*, 832 (1963).
[5] F. Obál, I. Madarász, Ö. T. Zoltán, E. Csanda and M. Földi, Z. ges. exp. Med. *138*, 26 (1964).
[6] Ö. T. Zoltán, M. Földi, F. Obál and I. Madarász, Zh. éksp. teor. Fiz. *138*, 43 (1964).
[7] B. Csillik and M. Földi, *A nyirokpangás hisztokémiája és hisztofizikája* (Akadémiai Kiadó, Budapest, 1965).
[8] M. Földi, E. Csanda, B. Csillik, A. Jáki, I. Madarász, F. Obál and Ö. T. Zoltán, Angiologica *2*, 133 (1965).
[9] M. Földi, B. Csillik, F. Joó and Ö. T. Zoltán, Angiologica *4*, 50 (1967).



Dislocation of myelin lamellae in a thick neurofibre of the caudate nucleus 7 days after experimental cervical lymph blockage in the rat. × 32,000.

sheath, they became dislocated, resulting in a curly appearance (Figure).

It should be stressed that similar alterations were described by LEE and BAKAY[10] as a typical ultrastructural change for the œdematous CNS.

Zusammenfassung. Bei der durch Unterbindung der zervikalen Lymphbahnen herbeigeführten experimentellen lymphogenen Encephalopathie der Ratte konnte eine schwere Desintegration der Myelinstruktur – eine für ein Hirnoedem charakteristische Veränderung – nachgewiesen werden.

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[10] J. C. LEE and L. BAKAY, Archs Neurol., Chicago *13*, 48 (1965).

Mast Cells in Nerves Affected with Fowl Paralysis (Marek's Disease)

This communication records the occurrence and variation in numbers of endoneurial mast cells in the sciatic nerves of normal and fowl paralysis-affected Brown Leghorn hens. Fowl paralysis is a disease of the peripheral nerves of *Gallus domesticus*[1]. It is world-wide in distribution and causes considerable economic loss. WIGHT[2] suggested that the nerve lesions could be classified in 3 main histopathological types; the predominant feature of type I is neural infiltration by mature lymphocytes and some plasma cells, type II is characterized by interneurite oedema and a relatively sparse infiltration of mature lymphocytes and plasma cells, while the nerves in type III are massively invaded by primitive lymphoid cells which have many neoplastic characteristics.

[1] J. MAREK, Dt. tierärztl. Wschr. tierärztl. Rdsch. *15*, 417 (1907).
[2] P. A. L. WIGHT, J. comp. Path. Ther. *72*, 40 (1962).