rhythm, so that the peak appears just before food presentation⁶. This shift could explain the high morning levels observed in the group fed 5 g/day at 09.00 h (i.e., time and quantity restricted). However, age-paired animals, fed ad libitum from 09.00 to 17.00 h (that is, time, but not quantity restricted) showed significantly lower hormone levels $(a < 0.05)^{14}$. Thus, the difference observed can, in all likelihood, be related to the limited food intake.

A more efficient adaptation of younger rats to fasting and refeeding has been reported for other physiological parameters, e.g. protein turn-over. Starvation decreases hepatic RNA and protein, early, at any age, but muscular and cardiac protein and RNA loss occurs to a greater extent in younger rats¹⁵. In fact, protein content decreases because of diminished synthesis, as well as increased catabolism¹ The subsequent increase in amino acid availability fits well with the present data, concerning higher corticosterone levels in younger rats, because of the well known role of this hormone in gluconeogenesis⁸. After refeeding, the incorporation of ¹⁴C-leucine into protein recovers more efficiently using ribosomes from younger animals¹⁶. As a matter of fact, corticosterone plays an essential role in the 'enzyme overshoot' observed in the refed rats¹⁷, related to the increased efficiency of food utilization after weight

The high corticosterone levels observed under fasting conditions have been ascribed to a diminished rate of disappearence of the hormone without changes in the secretion rate¹⁹. The latter possibility has been partly overcome by the observation of changes in CRF and ACTH levels in starving rats⁵. Such an activation of the pituitary-adrenal axis could be related to serotonergic mechanisms: brain serotonin (5-HT) turnover was reported to increase under fasting conditions²⁰. It should be recalled that a stimulatory role has been claimed for 5-HT in the control of the adrenocortical function²¹. The higher levels of corticosterone found in younger rats could be related to immature feed-back mechanisms; according to Goldman et al.²², the control of the acute activation of the pituitary-adrenal system is fully mature in weanling rats, but the negative feedback mechanism continues to increase in effectiveness between weaning age and adulthood.

- Present address: Pharmacologie Biochimique, CHU Cochin, 24, rue du Fb StJacques, F-75014 Paris (France).
- G.G. Slater, Endocrinology 70, 18 (1962).
- R. Boulouard, Fedn Proc. 22, 750 (1963).
- D. Bellamy, R.A. Leonard, K. Dulieu and A. Stevenson, Gen. comp. Endocr. 10, 119 (1968).
- I. Chowers, R. Einat and S. Feldman, Acta endocr. 61, 687
- K. Inoue, K. Takahashi and Y. Takahashi, Folia endocr. jap. 52, 898 (1976).
- J.P. Henry, in: Catecholamines and stress, p.359. Eds E. Usdin, R. Kvetnansky and I.J. Kopin. Elsevier-North Holland, New York 1980.
- J.C. Edozien, N. Niehaus, M.H. Mar, T. Makovi and B.R. Seitzer, J. Nutr. 108, 1767 (1978).
- P. De Moor, O. Steeno, M. Raskin and A. Hendrikx, Acta endocr. 33, 297 (1960).
- O.H. Lowry, N.J. Rosebrough, A.L. Farr and R.J. Randall, J. biol. Chem. 193, 265 (1951).
- R. R. Sokal and F. J. Rohlf, Biometry, Freeman, San Francisco
- S. Dechezleprêtre and P. Lechat, Archs Sci. Biol. 25, 247 12 (1971).
- D.R. Usher, I. Lieblich and R.A. Siegel, Neuroendocrinology *16*, 156 (1974).
- M.A. Ventura, Doctoral thesis, University of Paris VI, Paris 1981.
- M.N. Goodman and N.B. Ruderman, Am. J. Physiol. 239, E269 (1980).
- K. Nakano and H. Sidransky, J. Nutr. 108, 399 (1978).
- C.D. Berdanier and D. Shubeck, J. Nutr. 109, 1766 (1979).
- A. Ozelci, D.R. Romsos and G.A. Leveille, J. Nutr. 108, 1724 (1978).
- J.P. Fromweiler, C. Mialhe-Voloss and F. Stutinsky, J. Physiol., Paris 60, 99 (1966).
 G. Curzon and P.J. Knott, Br. J. Pharmac. 50, 197 (1974).
- R. W. Fuller, Neuroendocrinology 32, 118 (1981).
- L. Goldman, C. Winget, G.W. Hollingshead and S. Levine, Neuroendocrinology 12, 199 (1973).

DISPUTANDUM

Tissue channels, prelymphatics and lymphatics

Introduction

by M. Földi

D-7821 Feldberg (Federal Republic of Germany)

In a paper entitled 'Lymphatic drainage of the brain', published in 1968 with my associates in this journal (Földi, 1968), we expressed the view that - in spite of the fact that there are no lymph vessels in the brain substance - the cervical lymphatic tissue plays a role of paramount importance in the drainage of cerebral interstitial fluid. The 'perivascular spaces', described by His in 1865, have been shown to be long 'prelymphatic tissue channels' connecting the neuropil with the cervical lymphatic system.

These studies have recently been confirmed by Cserr (1980). Obviously, a blockage of a pathway key to performing a physiological task must have pathophysiological consequences. We have described lymphostatic encephalopathy as a result of cervical lymphatic blockage. This part of our studies has yet to arouse interest amoung others and awaits confirmation.

An analogous system of short tissue channels connects blood capillaries and initial lymphatics within those tissues which possess the latter. They, evidently, play an important role in microcirculation.

The reader will find pertinent studies on these topics written by two eminent scientists, the physiologist A. Hauck and the electron microscopist J.R. Casley-Smith, and a comment by A. Silberberg, who is a chemist.

Cserr, H.F., 1980. Convection of brain interstitial fluid. Adv. physiol. Sci. 7, 337.

Földi, M., 1968. Lymphatic drainage of the brain. Experientia 24, 1283-1287.