demonstrated by Prussian blue reaction. Phagocytic cells were counted with the method used for autoradiographic examinations.

The results (Fig. 5) indicate that r.p. macrophages play the decisive part in iron phagocytosis while SchS reticulum cells, although of paramount importance in antigen capture, have only slight phagocytic activity. Stimulation and alteration of kinetics of phagocytosis by immunization is also evident. The stimulatory effect, which was exerted chiefly on macrophages, may have been due either to antibodies[20] or to the BSA itself[21].

In summary, the antigen retention observed in chicken spleen at 2 and 4 days after antigen administration is due to phagocytic activity of r.p. macrophages and phagocytosis unrelated binding of antigen by SchS reticulum cells. The latter probably bind the antigen in a complexed form with antibodies,[7] similarly to the follicular dendritic reticulum cells[22,23]. The high antigen content of SchS indicates that antigen leaves the circulation here to reach white pulp lymphoid structures. This suggests that SchS functions as a kind of shunt, limiting the access of antigen to splenic white pulp after the onset of antibody response.

We thank Mrs I. Marosvölgyi and Mr B. Jankó for technical assistance.

Z. A. Nagy
E. Horváth

Veterinary Medical Research Institute,
Hungarian Academy of Sciences

Zsuzsanna Urbán

Department of Biology,
Semmelweis University of Medicine

Received July 17; revised November 30, 1972.

1 Nossal, G. J. V., and Ada, G. L., *Antigens, Lymphoid Cells and the Immune Response*, 107 (Academic Press, London and New York, 1971).
2 Wolfe, H. R., Norton, S., Springer, E., Goodman, M., and Herrick, C. A., *J. Immunol.*, **64**, 179 (1950).
3 Rosenquist, G. L., and Wolfe, H. R., *Immunology*, **5**, 211 (1962).
4 Keily, D., and Abramoff, P., *J. Immunol.*, **102**, 1058 (1969).
5 Payne, L. N., in *Physiology and Biochemistry of the Domestic Fowl* (edit. by Bell, D. J., and Freeman, B. M.) (Academic Press, London and New York, 1971).
6 White, R. G., French, V. I., and Stark, J. M., in *Germinal Centers in Immune Responses* (edit. by Cottier, H., Odartchenko, N., Schindler, R., and Congdon, C. C.), 131 (Springer-Verlag, Berlin, Heidelberg, New York, 1967).
7 White, R. G., French, V. I., and Stark, J. M., *J. Med. Microbiol.*, **3**, 65 (1970).
8 Nagy, Z. A., and Fehér, Gy., *Z. Immun.-Forsch.*, **143**, 323 (1972).
9 Blazkovec, A. A., and Wolfe, H. R., *Inter. Arch. Allerg.*, **26**, 80 (1965).
10 Cooper, M. D., Peterson, R. D. A., and Good, R. A., *Nature*, **205**, 143 (1965).
11 Cooper, M. D., Peterson, R. D. A., South, M. A., and Good, R. A., *J. Exp. Med.*, **123**, 75 (1966).
12 Clawson, C. C., Cooper, M. D., and Good, R. A., *Lab. Invest.*, **16**, 407 (1967).
13 Nagy, Z. A., and Fehér, Gy., *Z. Immun.-Forsch.*, **143**, 223 (1972).
14 Fehér, Gy., and Nagy, Z. A., *Z. Immun.-Forsch.*, **143**, 245 (1972).
15 Jankovic, B. D., and Isakovic, K., *Inter. Arch. Allerg.*, **24**, 278 (1964).
16 Nossal, G. J. V., Austin, C. M., Pye, J., and Mitchell, J., *Inter. Arch. Allerg.*, **29**, 368 (1966).
17 Nossal, G. J. V., Ada, G. L., and Austin, C. M., *Austral. J. Exp. Biol. Med. Sci.*, **42**, 311 (1964).
18 Nossal, G. J. V., Abbot, A., and Mitchell, J., *J. Exp. Med.*, **127**, 263 (1968).
19 Nossal, G. J. V., Abbot, A., Mitchell, J., and Lummus, Z., *J. Exp. Med.*, **127**, 277 (1968).
20 Rabinovitch, M., *J. Immunol.*, **99**, 232 (1967).
21 Cohn, Z. A., and Parks, E., *J. Exp. Med.*, **125**, 213 (1967).
22 Ada, G. L., Nossal, G. J. V., and Austin, C. M., *Austral. J. Exp. Biol. Med. Sci.*, **42**, 331 (1964).
23 Herd, Z. L., and Ada, G. L., *Austral. J. Exp. Biol. Med. Sci.*, **47**, 73 (1969).

Vasoconstriction by Noradrenaline in the Brain

In view of the present controversy, recently voiced in your journal[1-3] and elsewhere[4,5], concerning a significant vasoconstrictor action of noradrenaline (NA) applied microiontophoretically in the brain, it seems worth pointing out that the required experiments, that is direct applications of NA to cerebral vessels in cats, have been performed by Lassen *et al.*[6]. According to their observations, cortical arterioles are rather insensitive to noradrenaline—in contrast to arterioles in the cremasteric muscle, tested as a control by the same technique—but are readily dilated by acid solutions. Thus, the release of NA from more-or-less acid solutions would be more likely to cause a local vasodilatation than vasoconstriction. Whether this contributes significantly to the excitation apparently caused by strongly acid solutions[7,8] remains to be established.

K. Krnjević

Department of Research in Anaesthesia,
McGill University,
McIntyre Medical Science Building,
3655 *Drummond Street,*
Montreal 109

Received October 23, 1972.

1 Boakes, R. J., Bradley, P. B., Candy, J. M., and Dray, A., *Nature New Biology*, **239**, 151 (1972).
2 Stone, T. W., *Nature New Biology*, **239**, 152 (1972).
3 Szabadi, E., and Bradshaw, C. M., *Nature New Biology*, **239**, 152 (1972).
4 Stone, T. W., *J. Pharm. Pharmac.*, **24**, 318 (1972).
5 Jordan, L. M., Lake, N., and Phillis, J. W., *J. Pharm. Pharmac.*, **24**, 739 (1972).
6 Lassen, N. A., Wahl, M., Deetjen, P., Thurau, K., and Ingvar, D. H., in *Brain and Blood Flow* (edit. by Russell, R.), 174 (Pitman, London, 1971).
7 Fredrickson, R. C. A., Jordan, L. M., and Phillis, J. W., *Brain Res.*, **35**, 556 (1971).
8 Jordan, L. M., Frederickson, R. C. A., Phillis, J. W., and Lake, N., *Brain Res.*, **40**, 552 (1972).

Noradrenaline Artefacts

Szabadi and Bradshaw[1] have made a serious criticism of my suggestion that excitatory responses to iontophoretic noradrenaline could be mediated by constriction of small blood vessels in the brain[2]. The pharmacology of the intracranial vasculature is not as clearcut as these authors would suggest, however, and there are several reports of constrictor effects of 5-hydroxytryptamine (5-HT) on cerebral arteries[3,4]. Several of the early studies on cerebral vascular 5-HT actions could not take count of the fact that 5-HT has a dual action on blood flow to the head: flow to areas supplied by the external carotid artery is indeed increased, but that to areas supplied by the internal carotid artery is reduced. Net flow is unchanged[9]. It is perhaps also relevant that LSD-25, which may antagonize some central 5-HT excitations[5,6], can also antagonize the cerebral vasoconstrictor action of this amine[3].

The situation is similarly unclear for sympathomimetic amines. Thus, reports have been made of a β-adrenergic vasoconstrictor influence on brain blood vessels[7,8], which would explain the similarity of action of isoprenaline and noradrenaline.

T. W. Stone

Physiology Department,
University of Aberdeen,
Marischal College,
Aberdeen AB9 1AS

Received November 13, 1972.