

amine seem to be mediated by the local release of catecholamine in the vessel walls, for the pressor response is: (a) prevented by cocaine; (b) abolished by pretreatment of the animal with reserpine; (c) then restored by an infusion of noradrenaline<sup>4</sup>. Thus, for equipressor effects, there are receptors which are activated by 5-HT and not by tryptamine and vice versa. The antagonism by 1-benzyl-2-methyl-5-methoxytryptamine of the pressor response to 5-HT, but not to tryptamine, may be explained, as suggested by Woolley and Shaw, by supposing that the two amines act on different receptors.

In a much simpler system, the isolated rat stomach strip, 1-benzyl-2-methyl-5-methoxytryptamine, does not antagonize the direct actions of 5-HT or tryptamine<sup>5</sup>; but brom-LSD and LSD, both of which are good general antagonists of indoles, appear to antagonize 5-HT to a greater extent than tryptamine<sup>6</sup>. This difference disappears if the preparation is pretreated with an amine oxidase inhibitor<sup>4</sup>. In the same tissue, inhibition of amine oxidase selectively potentiates the response to tryptamine without altering that to 5-HT<sup>7</sup>. Here, then, tryptamine is acting on two receptors (smooth muscle and amine oxidase), whereas 5-HT, perhaps because of a diffusion barrier, is acting on only one of them (smooth muscle); the effects of amine oxidase inhibitors and brom-LSD can be explained on this basis.

To say that, in some sites, 5-HT and tryptamine may act on different receptors (including active spots on amine oxidase, chemoreceptors and different types of chromaffin cells) does not exclude the possibility that these substances act mainly at receptors common to both drugs peripherally and centrally. To explain the main actions we described, we thought it unnecessary to postulate different types of receptors for tryptamine and 5-HT in the central nervous system.

J. R. VANE

Department of Pharmacology,  
Royal College of Surgeons,  
Queen Square, London, W.C.1.

H. O. J. COLLIER  
S. J. CORNE

Department of Pharmacological Research,  
Parke, Davis and Co., Hounslow, Middlesex.

E. MARLEY  
P. B. BRADLEY

Department of Experimental Psychiatry,  
Medical School, University of Birmingham,  
Birmingham.

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<sup>2</sup> Ginzel, K. H., *5-Hydroxytryptamine*, 181 (Pergamon Press, London, 1958).

<sup>3</sup> Reid, G., and Rand, M., *Nature*, **169**, 801 (1952).

<sup>4</sup> Vane, J. R. (unpublished results).

<sup>5</sup> Barlow, R. B., and Khan, I., *Brit. J. Pharmacol.*, **14**, 99 (1959).

<sup>6</sup> Barlow, R. B., and Khan, I., *Brit. J. Pharmacol.*, **14**, 265 (1959).

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## ANATOMY

### Centrifugal Fibres to the Retina in the Pigeon

DESPITE the description of centrifugal fibres in the retina of the bird by Cajal, Dogiel and others<sup>1,2</sup>, and the finding of Wallenberg<sup>3</sup> of Marchi degeneration passing from the brain-stem to the retina, there is still considerable doubt as to the existence of a centrifugal fibre system in the visual system.

In a previous investigation of the avian visual pathway indirect evidence was obtained for such a centrifugal projection originating in the nucleus isthmo-opticus<sup>4</sup>. The relevant findings are that this nucleus undergoes complete cell loss following removal of the eye, and that the nature and time-course of the fibre degeneration in the isthmo-optic tract are strikingly different from that in all other components of the visual pathway. To prove that this nucleus is the site of origin of centrifugal fibres it must be demonstrated that localized lesions involving it result in fibre degeneration which can be traced through the associated pathway to terminate in the retina. Such degenerating fibres have now been traced, by the paraffin Nauta method, through the isthmo-optic tract into the dorsal part of the optic tract. From here the fibres can be clearly followed into the central region of the optic chiasma and thence into the contralateral optic nerve. In sections of the eye heavy degeneration is found in the optic nerve head, from which it spreads out in the optic nerve layer to all parts of the retina as recorded in the Marchi experiments of Wallenberg<sup>3</sup>. Occasional degenerating fibres can be traced through the ganglion cell layer and across the internal plexiform layer to terminate around cells on the inner aspect of the bipolar cell layer. The form of the axon terminals closely resembles those described by Cajal around the amacrine cells. The ipsilateral optic nerve and retina were completely free of degeneration in all experiments.

W. M. COWAN  
T. P. S. POWELL

Department of Human Anatomy,  
Oxford.

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## PATHOLOGY

### Experimental Production of Spermatic Granuloma in Rats

SPERMATOZOAL invasion of the epididymis is a recognized phenomenon in man, but when reviewing the literature on the subject it was found that no detailed systematic experimental study of the cellular reactions to spermatozoa in the interstitial tissues of the epididymis in animals has been made, nor has there been any such correlation with human lesions of this type.

Russell and Friedman<sup>1</sup> severed the vas deferens in rats, transplanted it to either the scrotal or abdominal walls, or to the retroperitoneum. 'Granulomatous' lesions developed at the resected ends, and no such lesions were found when the testes were removed. No histological descriptions were given other than the mention of pools of sperm surrounded by a wall of histiocytes (macrophages) nor did they state the time-interval between resection and killing of the animals. However, the legends of two of their photographs (very low power fields) carry the times 2½ and 3 weeks. The purpose of this preliminary communication is to describe the experimental production of spermatic granuloma by a variety of techniques with the histological changes found at different intervals post-operatively and to attempt to relate the findings to human material available to me.