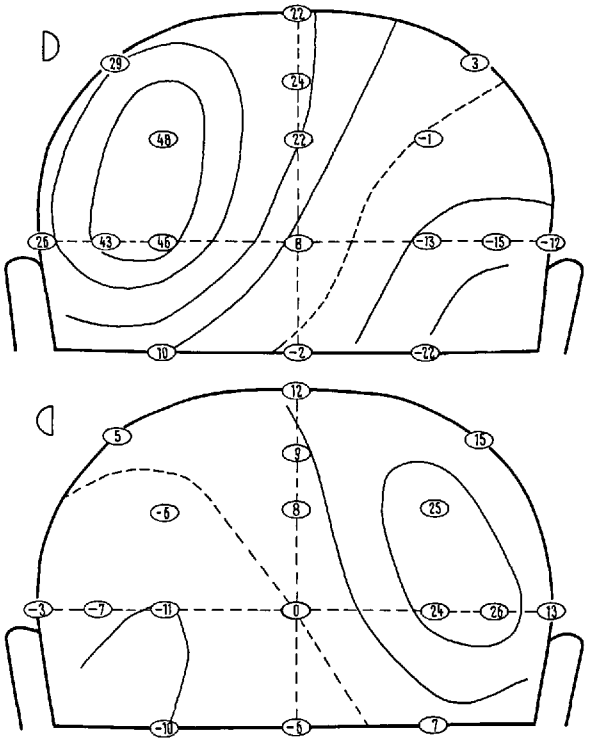


Fig. 2. Posterior scalp view of the P_{1a} potential distributions from stimulation of the right visual field (upper plot) or left visual field (lower). Electrode location at the bottom midline is the inion. Moving forward are electrodes at 10% distance (O_z), 20%, 30% (P_z), and 50% (C_z). Moving left horizontally from O_z are electrodes at 15% distance, 30% (T_5), and 50% (T_3). Also shown in the upper left quadrants are electrodes P_3 and C_3. Directly below the first electrode to the left of O_z is one on line horizontally with the inion. Similar positions are recorded on the right hemisphere (even-numbered). Potentials are microvolts × 10.

ponent preceding P_{1a} or the negative component following P_{1a}. The preceding negative component is very small, however, and difficult to measure reliably in the present averages.

Clinical studies on hemianopic patients support the interpretation that these localized evoked potentials result from stimulation of visual half-fields[2,4,5]. Research is continuing on the contour mapping of these potential distributions, and to determine the possible cortical sources for these potentials.

Conclusions. We conclude that under photopic experimental conditions it is possible in normal subjects to achieve sharply differentiated evoked potentials over the 2 cerebral hemispheres with lateral half-field stimulation. This finding has implications both for the understanding of normal visual brain functioning and for the clinical problem of hemianopia[6].

Zusammenfassung. Als Reaktion auf Reizung der lateralen Gesichtsfeldhälfte wurden evozierte Potentiale der Hirnrinde des Menschen registriert. Reizung der einen Gesichtshälfte erzeugte frühzeitig ein Maximum der Potentiale der gegenseitigen Hemisphäre sowie Phasenumkehr der Potentiale der gleichseitigen Hemisphäre.

W. R. Biersdorf and Z. Nakamura

Department of Ophthalmology, and Institute for Research in Vision, Ohio State University, Columbus (Ohio 43212, USA), 5 October 1970.

[4] K. A. Kooi, A. M. Güvener and B. K. Bagchi, Neurology *15*, 841 (1965).
[5] D. Regan and J. R. Heron, J. Neurol. Neurosurg. Psychiat. *32*, 479 (1969).
[6] Supported in part by U.S. Public Health Service grant No. EY00454. The authors wish to thank William Bell for technical assistance.

Photolysis and Birefringence of Frog Rods

When an action potential is transmitted through nerve fibres they exhibit an almost synchronous change in birefringence[1]. It is, therefore, of interest to inquire whether photoreceptors may not also manifest changes in birefringence following the absorption of light.

The effect of light on the properties of frog rods[2] can be studied in more detail with spectral lights if the time-course of events is tracked than was done in Schmidt's pioneering study[3]. 3 distinct processes can be distinguished and related to the photolysis of rhodopsin if the photometric density of the material is studied under similar conditions.

Single dark-adapted frog rods were examined with a polarizing microscope. Their nominal retardance was measured with a Brace-Köhler compensator by the method of Bear and Schmitt[4]. The densitometric data were obtained by passing light through a small number of randomly orientated rods, collecting it on a photoelectron multiplier and displaying the resultant photocurrent on a cathode-ray oscillograph. Data from 3 experiments are shown by the open symbols in the Figure. The measuring and bleaching lights were the same, $\lambda = 506$ nm, and the intensity equalled $3.03 \times 10^{18}\, q_{506}/m^2/s$. These data follow an exponential (half-time = 16 sec).

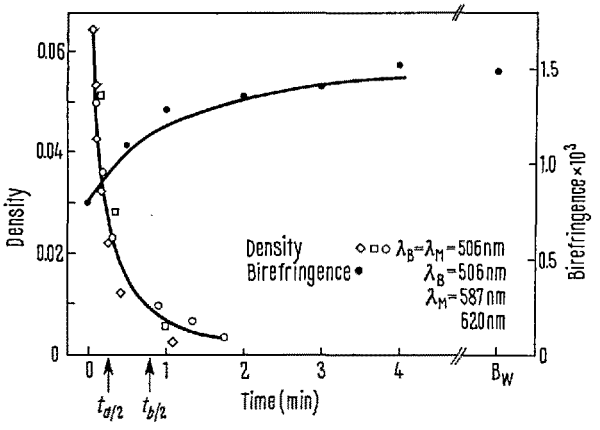
If the monochromatic light used in the polarizing microscope is absorbed by rhodopsin then the nominal retardance measures essentially the dichroism due to the orientation of the unbleached rhodopsin molecules[3,5]. But this is impossible with light of long wavelengths, which are not appreciably absorbed by rhodopsin. The filled circles in the Figure show average results for the retardance (per unit path-length) measured with $\lambda = 589$ nm and $\lambda = 620$ nm. The active light was the same as that used in the densitometric data, namely $\lambda = 506$ nm. Similar sets of data can be obtained with measuring lights of $\lambda = 567$ nm and even $\lambda = 506$ nm if a correction is applied for the contribution due to dichroism.

The results shown with the filled circles may hence represent a change in the birefringence of the rod: they follow an exponential (half-time = 46 sec); the final value (B_w) for the bleached material lies within the

[1] L. B. Cohen, R. D. Keynes and B. Hille, Nature, Lond. *218*, 438 (1968).
[2] R. A. Weale, J. Physiol., Lond. *204*, 123P (1969).
[3] W. J. Schmidt, Pubbl. Staz. zool. Napoli *23*, 158 (1951).
[4] R. S. Bear and F. O. Schmitt, J. opt. Soc. Am. *26*, 363 (1936).
[5] E. J. Denton, Proc. R. Soc. B. *150*, 78 (1959).

quantitative brackets given by SCHMIDT[3]. The change occurs only during light exposure. Data obtained with other measuring wavelengths show that the results for the earlier, fast (dichroic) component have the same photo-sensitivity as rhodopsin and agree spectrally with its extinction coefficient. The retardance spectrum of the slower (birefringence) function is independent of the measuring wavelength.

Measurements with $\lambda = 470$ nm revealed dichroism attributable to a photoproduct, i.e. the third component mentioned at the beginning. It disappeared with a half-time of several minutes[6]. Hence the reaction with the time-constant of 46 sec is unlikely to be a property of rhodopsin or of any of its photoproducts, although additional measurements show that the photolysis of rhodopsin causes it. The chain of events following the absorption of light by rhodopsin provides a clock for timing the occurrence of events connected with it: as the disruption of the rhodopsin complex in the rod proceeds very slowly and is unlikely to have passed within a few minutes of photolysis the stage at which both metarhodopsin I and II are in equilibrium, the rod material is probably altered soon after photic absorption. The aforementioned changes in the birefringence of nerve fibres during the passage of an action potential[1] are faster than those reported here; but those in the rods are much larger, owing to the favourable observing conditions. It remains to be seen if they relate to the visual process, and what happens following a flash. Further details will be published elsewhere.



Comparison between the time courses of photolysis (left scale) and a change in birefringence (right scale) of isolated frog rods as a function of exposure time (abscissa) to light of 3.03×10^{18} q_{506} nm/m²/s. The half-times of the 2 changes are indicated by the arrows and marked $t_{d/2}$ (density change) and $t_{b/2}$ (change in birefringence) respectively. The wavelength of the bleaching light is given by λ_B, that of the measuring light by λ_M. Birefringence is expressed as Retardance/path length.

Zusammenfassung. In distalen Stäbchen der Froschretina entwickelt sich nach Belichtung eine Änderung der Doppelbrechung als Ausdruck des photochemischen Exitationsprozesses. Letzterer wird in Analogie mit der nach Transmission eines Aktionspotentials auftretenden Änderung der Transmission des Nerven gesetzt.

R. A. WEALE

University of London, Institute of Ophthalmology, London W.C. 1 (England), 9 September 1970.

[6] C. BAUMANN, Pflügers Arch. ges. Physiol. *298*, 144 (1967).

[7] I thank Dr. R. W. SMITH, Imperial College, London (England), for help with some of the underlying theory and Miss G. M. VILLERMET for technical assistance.

Chronic Effects of Nicotine on Gastric Secretion in Vagotomized Rats

Tobacco smoking has been incriminated as an etiologic agent in peptic ulcer formation in man[1]. Furthermore, the mortality from duodenal ulcer disease is greater for smokers than for non-smokers[2]. We have recently shown that nicotine when given chronically (2 weeks) to rats, produces an increase in basal gastric acid and pepsin outputs[3]. The precise mechanism of this nicotine effect is not clear but may involve central stimulation. Since central stimuli usually act via the vagus nerve, we investigated the effect of chronic nicotine exposure on gastric secretion in vagotomized rats.

Materials and methods. 24 male Sprague-Dawley rats[4] weighing 326.6 ± 3.6 g were used. They were housed as described previously[5] and randomly divided into 2 groups of 12 rats each; one group was vagotomized, whereas the second was sham operated. Bi-lateral abdominal vagotomy was performed under pentobarbital[6] anesthesia (50 mg/kg) by LAMBERT's method[7]. Control rats were sham operated; sham operation consisted of an upper abdominal laparotomy under pentobarbital anesthesia. From the first post-operative day, 6 vagotomized and 6 sham operated rats were injected s.c. daily for 14 days with nicotine (2000 µg base/2.0 ml/kg); control vagotomized and sham operated rats received nicotine control saline (2.0 ml 0.85 g/100 ml w/v NaCl/kg). All rats survived the operation and after 14 days of injections they were isolated from food for 40 h as described previously[5]. Basal, unstimulated gastric secretion was collected following pylorus ligation. Preparation of nicotine and control injectables, details of animal care and housing, and techniques of gastric juice collection and analysis have been presented previously[3,5].

Results. Gastric secretory data are presented in the Figure. In sham operated rats chronic nicotine injections significantly increased basal gastric juice volume ($P < 0.025$), acid output ($P < 0.05$) and pepsin output ($P < 0.01$). Following vagotomy however, there were no differences between chronic nicotine-, or chronic nicotine control-injected rats.

[1] R. DOLL, F. A. JONES and F. PYGOTT, Lancet *1*, 657 (1958).
[2] R. DOLL, Scott. med. J. *9*, 183 (1964).
[3] J. H. THOMPSON, C. A. SPEZIA and M. ANGULO, Experientia *26*, 615 (1970).
[4] Charles River Breeding Laboratories, Breeding Shed 1, 251 Ballardvale Rd. Nt. Wilmington, Mass., USA 01887.
[5] J. H. THOMPSON, C. A. SPEZIA and M. ANGULO, Res. Comm. Chem. Path. Pharmac. *1*, 230 (1970).
[6] Nembutal sodium, Abbott Pharmaceuticals Inc. North Chicago, Ill., USA 60064.
[7] R. LAMBERT, *Surgery of the Digestive System in the Rat* (Charles C. Thomas, Springfield, Ill., USA 1965), chapt. 38, p. 477.