time that direct contact with environmental lighting acted to synchronize the phase of the rhythms. This period of disorganization might render the already vulnerable neonate more susceptible to various insults that could influence survival.

STEVEN M. REPPERT
Laboratory of Developmental Chronobiology, Children's Service, Massachusetts General Hospital and *Harvard Medical School, Boston 02114*

WILLIAM J. SCHWARTZ
Neuroendocrine Research Laboratory, Neurology Service, Massachusetts General Hospital and *Harvard Medical School*

References and Notes

1. J. Aschoff, Ed., *Handbook of Behavioral Neurobiology*, vol. 4, *Biological Rhythms* (Plenum, New York, 1981).
2. For reviews, see R. Y. Moore, in *Neurosecretion and Brain Peptides*, J. B. Martin, S. Reichlin, K. L. Bick, Eds. (Raven, New York, 1981), p. 449; B. Rusak and I. Zucker, *Physiol. Rev.* **59**, 449 (1979).
3. W. J. Schwartz and H. Gainer, *Science* **197**, 1089 (1977); W. J. Schwartz, L. C. Davidsen, C. B. Smith, *J. Comp. Neurol.* **189**, 157 (1980).
4. S. I. Inouye and H. Kawamura, *Proc. Natl. Acad. Sci. U.S.A.* **76**, 5962 (1979); *J. Comp. Physiol.* **146**, 153 (1982).
5. R. Y. Moore and N. J. Lenn, *J. Comp. Neurol.* **146**, 1 (1972); A. E. Hendrickson, N. Wagonner, W. M. Cowan, *Z. Zellforsch.* **125**, 1 (1972).
6. For review, see F. C. Davis, in (*1*), p. 257.
7. T. Deguchi, *Proc. Natl. Acad. Sci. U.S.A.* **72**, 2914 (1975).
8. Time of birth in the rat appears to be gated to the photoperiod by a circadian mechanism (S. M. Reppert, unpublished observations); D. W. Lincoln and D. G. Porter, *Nature (London)* **260**, 780 (1976).
9. L. Sokoloff *et al.*, *J. Neurochem.* **28**, 897 (1977).
10. L. Sokoloff, in *Basic Neurochemistry*, G. J. Siegel, R. W. Albers, R. Katzman, B. W. Agranoff, Eds. (Little, Brown, Boston, 1976), p. 388.
11. D. H. Ingvar and N. A. Lassen, Eds., *Brain Work* (Academic Press, New York, 1975), p. 377; L. Sokoloff, *J. Neurochem.* **29**, 13 (1977).
12. Glucose utilization by the SCN has been studied by the deoxyglucose technique in the developing rat [J. L. Fuchs and R. Y. Moore, *Proc. Natl. Acad. Sci. U.S.A.* **77**, 1204 (1980)]. Although these investigators found a clear day-night variation of SCN glucose utilization during the first day of postnatal life, they were unable to detect a rhythm in the fetus (examined at day 20 of gestation).
13. Timed pregnant Sprague-Dawley rats (Zivic-Miller, Allison Park, Pa.) were shipped to us within the first 4 days after insemination; day 0 of gestation was designated as the day the rats showed a positive reaction for sperm. Each rat was housed singly in a clear plastic cage. Each cage was wired to a drinkometer relay so that the daily profiles of drinking behavior were monitored for each pregnant rat. The light portion of the lighting cycles provided 600 lux of light at the midcage level. Rat food and water were freely available, and the time of day of routine care was varied.
14. G. Tannenbaum and J. B. Martin, *Endocrinology* **98**, 562 (1976).
15. M. Reivich, J. Jehle, L. Sokoloff, S. S. Kety, *J. Appl. Physiol.* **27**, 296 (1969).
16. Maternal drinking behavior rapidly adjusted to the phase shifts and it was completely entrained to DL by day 9 of gestation.
17. Animals anesthetized with ether were blinded by bilateral orbital enucleation.
18. The rhythmic onset of drinking in the blind rats was delayed only about 1 hour when deoxyglucose was administered (3 weeks after they were blinded), compared to that in sighted animals maintained in LD throughout pregnancy.
19. The retinohypothalamic pathway does not innervate the SCN until postnatal day 3 to 4 [M. Felong, *Anat. Rec.* **184**, 400 (1976); B. Stanfield and W. M. Cowan, *Brain Res.* **104**, 129 (1976)].
20. We thank R. Coleman, H. Heath, and J. Swedlow for technical assistance and L. Sokoloff and the Laboratory of Cerebral Metabolism for use of their densitometer apparatus. Supported by the Wm. F. Milton Fund, by March of Dimes Basil O'Conner starter research grant 5-335 and by PHS grant HD 14427 (to S.M.R.). W.J.S. is supported by teacher-investigator award 1 KO7 NS 00672 from the National Institute of Neurological and Communicative Disorders and Stroke.

2 December 1982; revised 10 February 1983

Hormonal Control of Sexual Differentiation: Changes in Electric Organ Discharge Waveform

Abstract. *Males and females of some mormyrid electric fishes generate electrical pulses that differ in waveform and duration. For one such species, testosterone or dihydrotestosterone induces females and immature males to produce the mature male electric organ discharge which is two times the duration of the female or immature discharge. Estradiol has only a weak effect. For a second species where males and females have similar electric organ discharges, testosterone produces no effect. The data suggest that androgens affect the electric organ itself.*

Androgen- and estrogen-concentrating cells occur in gonadal tissues and in skeletal muscle and brain structures implicated in the control of vertebrate sexual behavior (*1*). Gonadal hormones can affect a variety of chemical and morphological processes ranging from acetylcholinesterase activity of nerve and muscle to dendritic growth of central neurons (*2*). We now propose that steroid hormones can modulate the development of action potentials of some electrically excitable membranes (*3, 4*).

For some mormyrid electric fishes from Africa, the electric organ discharges (EOD's) of males and females differ in waveform and duration (Fig. 1, d to f) (*5*). The electric organ, situated in the caudal peduncle (see Fig. 1, a and b), is derived from myomeres and includes four columns of serially stacked plates or "electrocytes" oriented in the transverse plane. For each electrocyte, an anterior or posterior face has rootlike extensions that join into a major stalk that is innervated by special spinal motoneurons, the "electromotoneurons" (*6, 7*). The EOD is elicited by a burst of three spikes in the electromotoneurons, which in turn are excited by a descending medullary volley (*7*). The membrane of each face and stalk produces an action

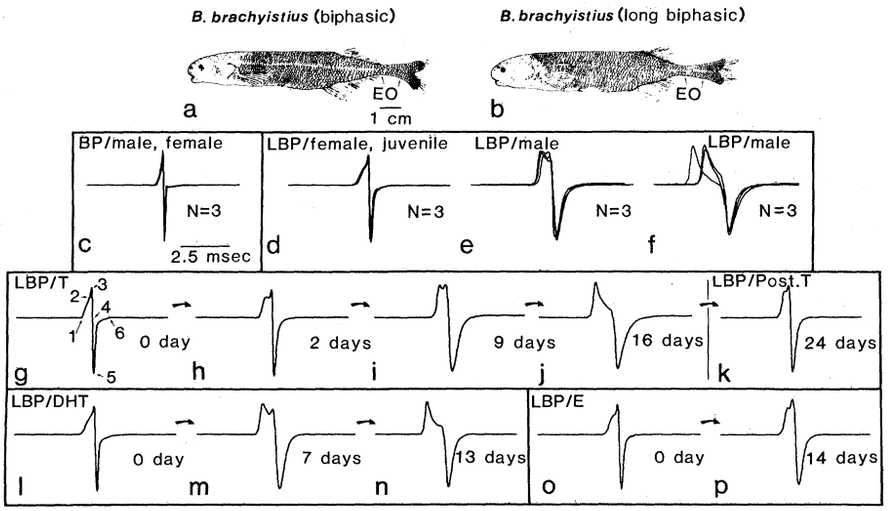

Fig. 1. (a and b) The two species of electric fish in this study were *Brienomyrus brachyistius* (biphasic) (BP) and *B. brachyistius* (long biphasic) (LBP); the electric organ (*EO*) is in the tail. (c to p) Oscilloscope records of electric organ discharge (EOD) waveforms scaled to the same peak-to-peak amplitude. (c) *B. brachyistius* (BP) males and females have similar EOD's (three superimposed individual EOD's). The EOD of juvenile and female *B. brachyistius* (LBP) are similar (d) but differ in form and duration from males (e and f). (g to j) Testosterone (*T*) added to the water of a *B. brachyistius* (LBP) female induces the male EOD for 16 days; the EOD reverts to a female type (k) after the fish is returned to freshwater for 24 days. (l to n) A pellet of DHT implanted in a gonadectomized female also induces a male EOD. (o and p) An estradiol (*E*) pellet implanted in an immature male has minimal effects on the EOD.

potential; their summed activity can predict the waveform and duration of the EOD (*8*).

Our report focuses on the mormyrids of the Ivindo River District of Gabon (0°30′N, 12°50′E), West Africa, where there are at least 23 species, most with distinctive EOD's (*5*). We describe the effects of gonadal steroids on the EOD's of two stream species, *Brienomyrus brachyistius* (biphasic) (BP) and *B. brachyistius* (long biphasic) (LBP). These species are sympatric and morphologically similar (Fig. 1, a and b), although they are electrically distinct, as indicated by the descriptor "BP" or "LBP" (*9*). The males and females of *B. brachyistius* (BP) had similar discharges in all of our recordings (mean duration, 0.412 msec, $N = 19$; mean peak frequency of the power spectrum, as determined by Fourier analysis, 1848 Hz) (Fig. 1c). In contrast, the average EOD duration for mature males of *B. brachyistius* (LBP) (Fig. 1, e and f) was 2.25 msec ($N = 6$) compared to 0.908 msec for females and immature specimens ($N = 25$) whose EOD waveforms are similar (Fig. 1d). Some males (Fig. 1e) have EOD durations intermediate between that of the fully adult male EOD (Fig. 1f) and that of the female (Fig. 1d). The differences in duration are matched by differences in the average peak power frequency for males [395 Hz ($N = 6$)] and females and immature specimens [925 Hz ($N = 25$)].

Experiments were conducted in Gabon during the October–November 1981 rainy season. *Brienomyrus brachyistius* (BP) (53 to 90 mm, total length) and *B. brachyistius* (LBP) (65 to 100 mm) were caught in local streams and maintained separately in 1.2 liters of aerated stream water (conductivity, 20 to 60 Kohm-cm;

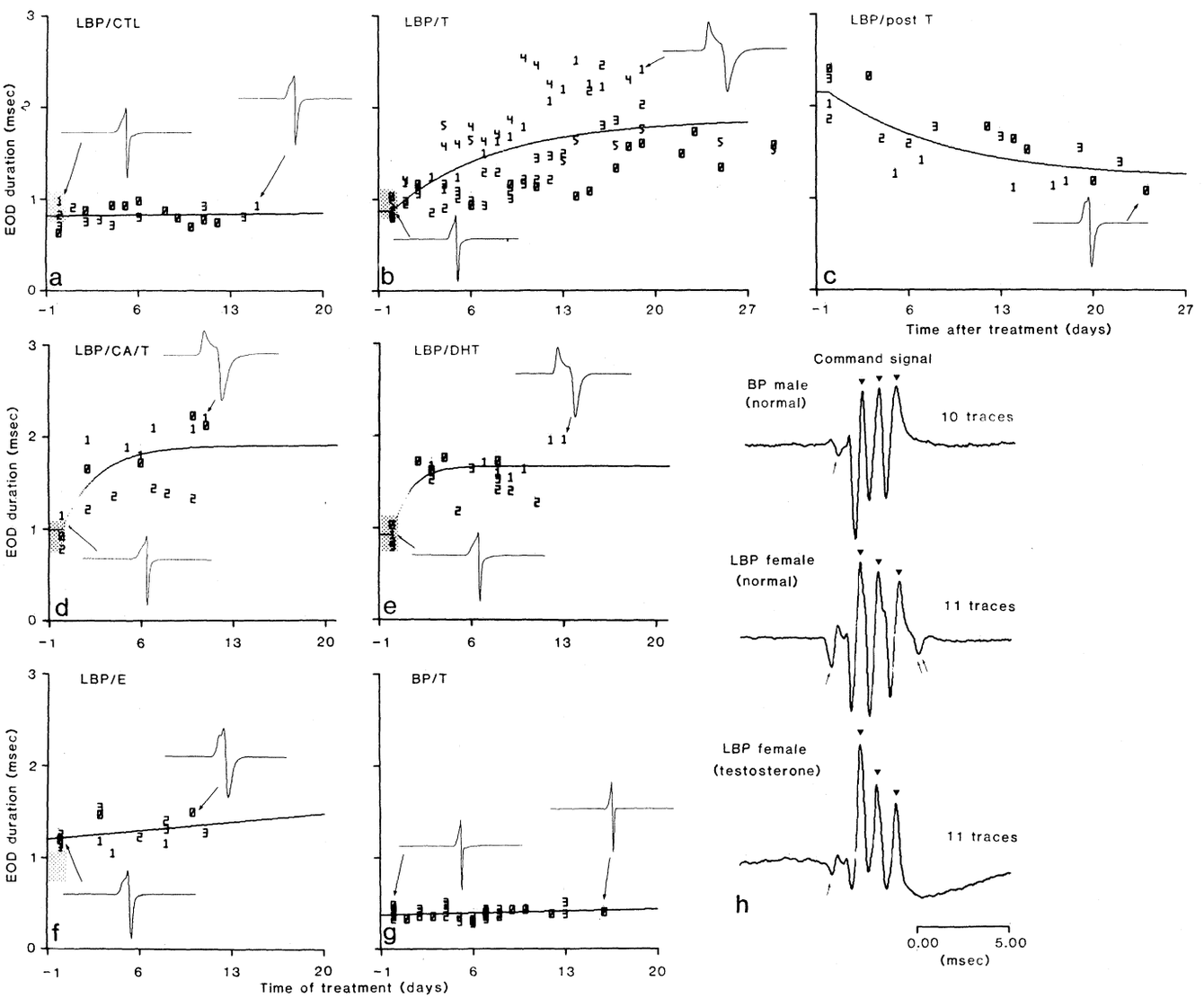

Fig. 2. (a to g) Plots of the time course of change in EOD duration during treatment periods. Representative EOD's are shown. The stippled area to the left of each plot is the range of EOD duration for immature and female *B. brachyistius* (LBP) for one standard deviation (0.161 msec) to either side of the population mean (0.908 msec, $N = 25$). Each symbol is for one individual except in (g) where "0" is for three specimens maintained in the same water. (c) Recovery of the same specimens illustrated during testosterone treatment in (b). Drawn lines represent a least-squares fit to the straight line (a, f, and g) or an exponential curve (b to e) (*15*). Changes in EOD duration were nonsignificant for the control (*CTL*) and estradiol (*E*)-treated *B. brachyistius* (LBP) (a and f) and testosterone (*T*)-treated *B. brachyistius* (BP) (g); but significant for *B. brachyistius* (LBP) during and after testosterone treatment (b and c), gonadectomized and treated with testosterone (d), or DHT (e); (h) oscilloscope records of the command signal of electromotoneurons that excite the electric organ. For a *B. brachyistius* (BP) male (upper record, ten averaged traces), *B. brachyistius* (LBP) normal female (middle record), and testosterone-treated female (lower record), the command has three major spikes; the temporal pattern is nearly identical in all three specimens. The initial (small arrow) and late (double arrow) negativities are, respectively, the medullary relay signal and an electrocyte stalk potential (*7*).

temperature, 22° to 24°C). Animals were held captive for at least 2 days before they were given hormones. At the end of each experiment the animals were killed by immersion in MS222, dissected for gonad examination, and identified as immature unidentified, immature male (IM) or female (IF), or mature male (M) or female (F) (*10*). All EOD's were recorded daily in fresh stream water with differential Ag-AgCl electrodes aligned parallel to a polyvinyl chloride tube that held the fish in a preferred orientation (*11*). After amplification, an EOD was hand-drawn from the oscilloscope, photographed, or tape-recorded (Nagra IV-SJ). After we returned from Gabon, representative EOD waveforms were digitized at a sampling rate of 40 KHz from the tape recordings and scaled to the same peak-to-peak amplitude (*12*). EOD duration was measured as the time interval between the first and last points on the waveform which depart from the baseline by more than 5 percent of the peak-to-peak amplitude (points 1 and 6, respectively, in Fig. 1g). All time measurements were corrected to 22°C by using a Q-10 factor of 1.75 (*13*). All EOD's are shown with the head positivity upward.

The initial experiments were done with *B. brachyistius* (LBP). For six specimens (two IF, three F, and one IM), 2.0 to 4.0 mg of 17α-methyltestosterone was dissolved in the 1.2 liters of stream water periodically at 24- to 48-hour intervals. Four specimens (two unidentified and two F) were maintained in stream water for similar time periods to control for the effects of captivity. The control group showed no significant change in EOD duration (slope of straight line fit in Fig. 2a is 0.002 msec/day, t-test of significance of regression coefficient, $t = .35$; $P > .6$, d.f. = 19) (*14*). In contrast, the testosterone-treated group showed a twofold increase in EOD duration (Fig. 2b shows best exponential curve fit instead of straight line for which the slope was 0.03 msec/day, $t = 5.46$, $P < .001$, d.f. = 77) (*15*).

The EOD waveform of *B. brachyistius* (LBP), as shown in Fig. 1g, is biphasic with an inflection point (point 2) on the rising positive phase (points 1 to 3), and a single smooth peak (point 5) on the negative phase (points 4 to 6). Testosterone appears to induce a shift in the amplitude of the waveform at the inflection point relative to that of the following positive peak: point 2 increases until it is equal in amplitude to point 3 (Fig. 1, h and i) and the EOD resembles that of a "transitional" male (Fig. 1e). After a few more days of testosterone, point 2 is very large and point 3 all but disappears (Fig. 1j) as the entire EOD slows to the longer, male EOD (Fig. 1f). When testosterone was removed by returning the fish to fresh water, the EOD reverted to the female-type waveform during 24 additional days (Figs. 1k and 2c) [see (*14, 15*) for results of statistics].

To show that the induced hormone changes were not dependent on intact gonads, we gonadectomized three fish (one IF, one F, and one IM) and treated them with testosterone in the same way. The effects on the EOD waveform were similar (Fig. 2d) (*14, 15*).

Since some vertebrates can convert testosterone to 17β-estradiol or another androgen, 5α-dihydrotestosterone (DHT) (*1*), it is possible that some of the testosterone effects were due to estrogen and were not androgen-specific. To examine this possibility, we treated animals as follows: three fish received a 2.0- to 3.0-mg pellet of DHT implanted within the ventrolateral body cavity (two IM and one F); one was gonadectomized and received a DHT implant (one IF, symbol 1, Fig. 2e); and four received a 2.0- to 3.0-mg estradiol pellet (three IM and one IF) (*16*). Only the fish that received DHT showed the marked change in EOD duration and waveform (Fig. 1, 1 to n, and Fig. 2e) (*14, 15*). The effects of estradiol were minimal (Fig. 1, o and p, and Fig. 2f). In our estradiol experiments, the initial EOD durations were already at the high end of the population range. The estradiol seemed to induce a slight increase in duration, but the effect was statistically nonsignificant for the entire treatment period (linear slope = .01 msec/day, $t = 1.26$, $P > .2$, d.f. = 12). Estradiol may therefore mimic some of testosterone's effects, as in mammalian sexual behavior (*17*), but this requires further study (*18*).

Thus, the observed changes in EOD waveforms and durations were (i) dependent on the addition of exogenous hormone and (ii) reversible (*4*). Also, the effect appeared to be specific to mormyrids with sex differences in their waveforms. Testosterone added to the water of *B. brachyistius* (BP) (three F and three IM) had no significant effect on the EOD (Fig. 2g, linear slope = .003 msec/day, $t = 1.45$, $.1 < P < .2$, d.f. = 40).

We considered two sites of action where gonadal hormones or their metabolites might induce EOD transformations: the peripheral electrocytes themselves or the central motor pathway controlling electric organ excitation. The former seemed more likely since the current generated by a single artificially stimulated electrocyte resembles that of the entire EOD waveform (*8*). Centrally mediated events control the synchrony and rate of electrocyte discharges (*7*). We recorded the command signal, arising from the spinal electromotoneurons that excite the electric organ, by inserting two 00-insect pins subdermally at either end of the organ in specimens whose EOD was blocked with intraperitoneal injections of Alloferin (0.1 μg), a curare-like drug. For *B. brachyistius* (BP) and for normal and testosterone-treated females of *B. brachyistius* (LBP), the command signal had three major spikes (large arrows, Fig. 2h) separated by about 1 msec. Thus, the central command is nearly identical for specimens with EOD's of differing form and duration. As in all other known mormyrids (*7*), the three spikes represent the synchronous firing of the electromotoneurons. The time interval between the spikes appears the same for all specimens, although the waveform varies with electrode placement. The general properties of the spinal command signal were unchanged in testosterone-treated females, as shown in Fig. 2h. The hormone effects thus appear to be distal to the command signal generator, at the level of the electric organ.

Gonadal hormones are known to affect the spontaneous activity and refractory period of central neurons (*19*). However, the effect here is on the waveform and duration of the electrocyte's discharge which depends on the functional properties of its component action potentials. There are different ways in which testosterone might exert this effect: (i) it may alter the physiology of the synapse between the electromotoneurons and the electrocyte's stalk, (ii) it may change the cable properties of the electrocyte, and (iii) it may change the distribution or magnitude of different ion channels in the electrocyte's excitable membranes (*20*). The last possibility is most intriguing, as it has already been shown for some amphibians and mammals that developmental changes in the waveform and duration of action potentials of motor or sensory neurons arise because of changes in specific ionic conductances (*21*).

ANDREW H. BASS*
CARL D. HOPKINS*
Department of Ecology and Behavioral Biology, University of Minnesota, Minneapolis 55455, and *Laboratoire de Physiologie Nerveuse, Centre National de la Recherche Scientifique, Gif-sur-Yvette, France*

References and Notes

1. D. Kelley and D. Pfaff, in *Biological Determinants of Sexual Behavior*, J. Hutchison, Ed. (Wiley, Chichester, England, 1978), pp. 225–254; A. P. Arnold, *Am. Sci.* **68** (No. 2), 165 (1980); C. W. Bardin and J. F. Catteral, *Science* **211**, 1285 (1981); B. S. McEwen, *ibid.*, p. 1303.
2. V. Luine, F. Nottebohm, C. Harding, B. McEwen, *Brain Res.* **192**, 89 (1980); T. DeVoogd and F. Nottebohm, *Science* **214**, 202 (1981).
3. In *Sternopygus*, a gymnotoid electric fish with a sex difference in its wavelike EOD [C. D. Hopkins, *Science* **176**, 1035 (1972)], DHT can lower the EOD frequency [J. H. Meyer and H. H. Zakon, *ibid.* **217**, 635 (1982)], an effect apparently mediated by the central nervous system [see (*7*)]. The effect we describe appears to be peripheral at the level of the electric organ.
4. Two types of evidence suggest there is a seasonal change in the male EOD: (i) These fish appear to breed during the rainy season (early October), which correlates with the appearance of the mature male EOD. (ii) The EOD's of captive, mature males revert to the female-type waveform but are reinduced by testosterone. We did not castrate males with fully transformed EOD's since the EOD's of captive males revert to the female form and we did not have enough males to define a possible difference between the "normal" reversible condition and castration.
5. C. Hopkins, *Behav. Ecol. Sociobiol.* 7, 1 (1980); P. Moller, *Oceanus* **23**, 44 (1980).
6. T. Szabo, *Z. Zellforsch. Mikrosk. Anat.* **49**, 33 (1958).
7. M. V. L. Bennett, in *Fish Physiology*, W. S. Hoar and D. J. Randall, Eds. (Academic Press, New York, 1971), vol. 5, pp. 347–491.
8. Microelectrode studies show that action potentials of the posterior and anterior faces underlie, respectively, the EOD's positive and negative phases (*7*). We believe the stalk's potential can account for the inflection of an EOD's positive phase (Fig. 1d).
9. Electrocyte morphology also differs in these fishes.
10. K. Lagler, in *Methods for Assessment of Fish Production in Fresh Waters*, W. E. Ricker, Ed. (Blackwell, Oxford, 1968), chapter 2.
11. Electrode orientation or proximity (within 15 cm) does not affect the EOD waveform; only the amplitude changes.
12. We used a PDP8/e with Lab8/e A/D converter sampling at 24.83 μsec per point.
13. The Q-10 value was determined by measuring the EOD duration of two specimens of another sympatric species, *B. brachyistius* (triphasic), over the range of 18° to 25.8°C.
14. All significance values are derived from a least-squares fit to a straight line, two-tailed *t*-test for significance of regression coefficient. Post-testosterone-treated group: linear slope = − .03 msec/day, $t = 3.91$, significant at $P < .001$, d.f. = 22. Gonadectomized, testosterone-treated group: linear slope = .08 msec/day, $t = 3.84$, significant at $P < .01$, d.f. = 16. DHT-treated group: linear slope = .05 msec/day, $t = 3.67$, significant at $P < .01$, d.f. = 20.
15. The data for Fig. 2, b to e, could be fit to the equation: $y = y_0 + y_f(1 - e^{-t/\tau})$. The time constant, τ, for each curve is Fig. 2b: 7.8 days, [standard error (S.E.) = 3.4; d.f. = 76]; Fig. 2c: 9.3 (S.E. = 6.5, d.f. = 18); Fig. 2d: 2.7 (S.E. = 1.9, d.f. = 15); and Fig. 2e: 1.4 (S.E. = 0.8, d.f. = 19).
16. In France, for a sham-operated fish (symbol 3, Fig. 2a) or fish treated with water-soluble cholesterol, there is no significant change in duration unlike additional testosterone-treated fish.
17. D. Pfaff, *J. Comp. Physiol. Psychol.* **73**, 349 (1970).
18. Increases in EOD duration are matched by decreases in the peak power frequency (PPW). The average maximum decrease in PPW for the testosterone-treated group was from 1054 to 414 Hz; for the gonadectomized and testosterone-treated group, 934 to 455 Hz; for DHT-treated group, 938 to 512 Hz; and for the estradiol-treated group, 800 to 622 Hz.
19. K. M. Kendrick and R. F. Drewett, *Science* **204**, 877 (1979); K. Yamada, *Brain Res.* **172**, 165 (1979).
20. Anatomical data for *B. brachyistius* (LBP) and *B. brachyistius* (triphasic) (which also has EOD sex difference under androgen control) suggest the electrocyte's anterior face is proliferated in males and androgen-treated females (A. H. Bass, J. Denizot, C. D. Hopkins, in preparation).
21. N. Spitzer, *Annu. Rev. Neurosci.* **2**, 363 (1979); K. D. Walton and B. Fulton, *Soc. Neurosci. Abstr.* **7**, 246 (1981).

22. We thank P. Posso, the Institute de Recherche Tropicale, M. Marchaterre, and C. Bell for field help; T. Szabo for the invitation to work in his laboratory; M. Boudinot for help with computer programs and curve fitting; D. Kelley, D. Crews, N. Stacey, and R. Peter for technical advice; M. Marchaterre and V. Wong for the fish portraits; C. Bell for suggesting the command signal experiments; R. Kado for the curve fitting; D. Rouilly for histology; and C. Bell, R. Kado, T. Szabo, P. Belbenoit, J. Serrier, G. Bass, L. Carlson, and T. Natoli for help with the manuscript. Supported by NIH grants to A.H.B. and C.D.H., a National Geographic Society grant to C.D.H., and a grant to T. Szabo from the Centre National de la Recherche Scientifique.

* Present address: Section of Neurobiology and Behavior, Cornell University, Ithaca, N.Y. 14853.

29 July 1982; revised 13 October 1982

Dopamine-β-Hydroxylase Activity and Homovanillic Acid in Spinal Fluid of Schizophrenics with Brain Atrophy

Abstract. *Schizophrenic patients with high ventricle brain ratios and cortical brain atrophy, as shown by computerized tomography, had decreased spinal fluid concentrations of homovanillic acid and dopamine-β-hydroxylase activity. These decreased cerebral spinal fluid concentrations in patients with brain atrophy support the proposal of disturbed noradrenaline and dopamine neurotransmission in a subgroup of schizophrenic patients.*

Brain atrophy has been detected in some schizophrenic patients by computerized tomography (CT) (*1–9*). Abnormalities in scans in schizophrenia have been associated with neuropsychological impairment (*2, 4–6*) and other nonpsychotic symptoms (*9*). Biochemical abnormalities in schizophrenics with brain atrophy have rarely been reported (*10*). Despite evidence of a dopamine disorder in schizophrenia (*11*), spinal fluid concentrations of the dopamine metabolite

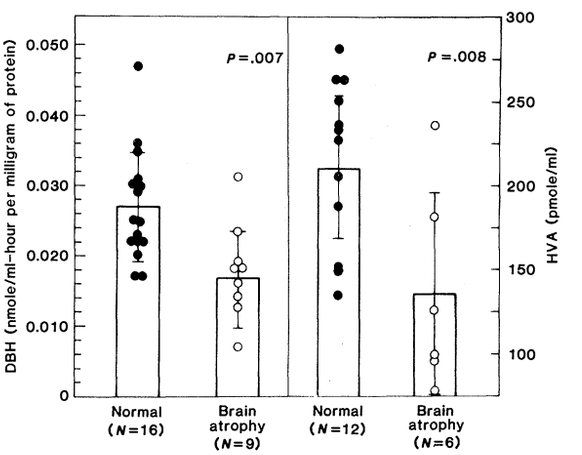

Fig. 1. Specific DBH activity and HVA content in the spinal fluid of patients with normal CT scans or brain atrophy. The probability given is that associated with the *t*-test for comparing two means (see Table 1). Error bars represent 1 standard deviation.

Table 1. Relation of normal and abnormal CT scans and VBR and the presence or absence of cortical atrophy to three variables in the spinal fluid of drug-free schizophrenic patients. The DBH activity and HVA concentrations were significantly lower in patients with brain atrophy. Values are mean + S.E.M. The probabilities are those associated with the *t* statistic for the difference between normal and abnormal. N.S., not significant.

Status	DBH					HVA		
	Patients (*N*)	Specific activity: nmole/ml-hour per mg protein	*P*	Activity: nmole/ml-hour	*P*	Patients (*N*)	pmole/ml	*P*
				CT scans				
Normal	16	0.027 ± .0020	.007	1.05 ± .170	.107	12	214 ± 13.5	.008
Abnormal	9	0.018 ± .0023		0.58 ± .006		6	138 ± 23.8	
				VBR				
Normal	20	0.026 ± .0017	.005	0.98 ± .139	.0056	16	191 ± 15.0	
Abnormal	5	0.014 ± .0021		0.49 ± .075		2	167 ± 67.2	
				Cortical atrophy				
Absent	17	0.025 ± .0022	N.S.	0.99 ± .171	.029	17	203 ± 14.5	.09
Present	7	0.019 ± .0024		0.60 ± .055		7	145 ± 23.3	