Sexual Behavior in the Human Male (Saunders, Philadelphia, 1948), p. 161.
7. W. H. Masters and V. E. Johnson, *Human Sexual Response* (Little, Brown, Boston, 1966), p. 296.
8. ______, *The Pleasure Bond* (Little, Brown, Boston, 1975), pp. 247–248.
9. A. Comfort, *The Joy of Sex* (Crown, New York, 1972), pp. 51–52.
10. Sonagrams A and B were made from the soundtracks of two films, *The Squeeze Technique* and *Effective Sexual Functioning*, respectively. These films are used in sexual education and therapy. A tape used in private sexual counseling provided the material for sonagram C. The partners in all three cases were married and were aware they were being filmed or recorded. The first filmstrip is distributed by the Multi Media Resource Center, San Francisco, Calif., and the second was contributed by the Center for Marital and Sexual Studies, Long Beach, Calif.
11. N. Bolwig, *Behaviour* **14**, 136 (1959).
12. G. S. Saayman, *Folia Primatol.* **12**, 81 (1970).
13. K. R. L. Hall, *Proc. Zool. Soc. London* **139**, 283 (1962); *ibid.*, p. 181.
14. ______ and I. DeVore, in *Primate Behavior*, I. DeVore, Ed. (Holt, Rinehart & Winston, New York, 1965), p. 53.
15. W. J. Hamilton, III, and P. C. Arrowood, personal observations.
16. Adult males, $N = 5$; adult females, $N = 5$; juvenile males, $N = 6$. An adult male is here defined as a male larger than a female.
17. A female chacma baboon in another troop in the Okavango Swamp, Botswana, was recently observed to give copulatory calls when defecating, and, on one occasion, a female gave a copulatory call when a high-ranking male walked nearby. Hall (*13*) also reported that females sometimes gave copulatory calls while defecating. For males, recent observations in Botswana (S. K. Smith, personal communication) suggest that only the more dominant adult males vocalize when copulating.
18. Durations of female calls in seconds: Heady, 6.85 (S.D. = 0.32, $N = 2$); Lightning Tail, 4.47 (S.D. = 0.94, $N = 6$); Zebra, 4.07 (S.D. = 1.19, $N = 4$); Prudence, 3.14 (S.D. = 0.51, $N = 4$); Dawn, 1.94 (S.D. = 0.16, $N = 6$).
19. Duration (in seconds) of female calls over all 22 matings with adult males: 3.68 (S.D. = 0.44). Duration of female calls over seven matings with juvenile males: 3.68 (S.D. = 0.89). Durations of individual female calls during matings with juvenile males: Lightning Tail, 4.65 (S.D. = 0.94, $N = 3$); Heady, 3.42 (S.D. = 1.84, $N = 3$); Dawn, 1.55 ($N = 1$).
20. L. P. Stoltz and G. S. Saayman, *Ann. Transvaal Mus.* **26**, 99 (1970).
21. C. R. Cox and B. J. LeBoeuf, *Am. Nat.* **111**, 317 (1977).
22. C. Darwin, *The Origin of Species* (New American Library of World Literature, New York reprint. ed., 1958).
23. G. Hausfater, *Contrib. Primatol.* **7**, 1 (1975).
24. T. E. Rowell, in *Social Communication Among Primates*, S. Altmann, Ed. (Univ. of Chicago Press, Chicago, 1967).
25. We used a tape recorder (Uher 4000 Report-L), microphone (Uher M514), and 60-cm parabola to record baboon vocalizations and the same equipment, without the parabola, to record the gibbon and human vocalizations. Sonagrams were made with a sonagraph (Kay Elemetrics model 6061B) on narrow band filter. (For the human sounds, the wide-band filter was used.) An accessory unit, a 6076C scale magnifier, allowed us to expand the width of these low frequency sounds over the entire 10-cm width of the sonagram paper, thus providing a clearer picture of the complex structure of these sounds.

26. We thank C. D. Busse and Drs. A. B. Clark, G. Hausfater, G. W. Salt, W. A. Mason, and B. S. Orlove for critical reviews of the manuscript; the Anthropology Department, University of California, Davis, for the use of their sound spectrograph; V. Hildebrand for numerous helpful suggestions; and Multi Media Resource Center and the Center for Marital and Sexual Studies for the use of their filmstrips.

17 June 1977; revised 20 December 1977

Commissural Transmission: Maturational Changes in Humans

Abstract. *Latency differences between ipsilateral and contralateral somatosensory evoked potentials show maturational trends in keeping with the myelogenic timetable and development of the corpus callosum. The distribution and modality-specific projection of early ipsilateral activity suggests an origin in the contralateral posterior-parietal area.*

In humans and other mammals, the commissures of the forebrain are poorly established at birth (*1*). Their growth parallels that of the neocortex, building up as medullary sheaths (myelin) are deposited. Culmination of the myelogenic cycle in a given pathway has been taken as an index of its functional maturity (*2*). The corpus callosum and other association bundles as well as the intracortical plexus of the parietal lobe are among the last cortical zones to begin and complete the process of myelination.

The evoked potential has been widely used to track the development of interhemispheric communication in animals. Recordings obtained directly from the pial surface in a number of species (*3*) show that the latency of the transcallosal response decreases as a function of age. Detailed electron microscopic examination of the corpus callosum has revealed a strong inverse relationship between the emergence of myelinated callosal axons and the latency of the interhemispheric evoked potential (*4*).

Topological studies of human evoked potentials to sensory stimulation have shown that the ipsilateral response following unilateral stimulation is consistently slower and of smaller magnitude than the contralateral response (*5*). The latency difference between selected components of ipsilateral and contralateral responses has been interpreted as representing the commissural delay (*6*). In the visual modality, a stimulus falling on the left hemiretinae projects exclusively to the right occipital lobe and one on the right hemiretinae projects to the left lobe. Thus commissural transmission can be directly inferred from ipsi-contralateral differences. In this respect, significantly longer latencies and lower amplitudes have been observed over the homolateral cortex after hemifield stimulation (*7*).

Since strict fixation is required for unilateral visual activation, this modality is not suitable for testing young children and infants. Behavioral determinants such as dichotic listening or tachistoscopic reaction time have provided unreliable and often contradictory results regarding commissural transfer (*8*).

I have studied the somatic afferent system in order to derive an objective measure of commissural transmission in humans irrespective of age. Differences found in the latency, distribution, and development of ipsilateral and contralateral responses indicate that the evoked potential recorded from the scalp's surface can provide valuable information regarding myelogenesis and maturation of the corpus callosum.

Sixty healthy subjects ranging in age from 2 years to adulthood (*9*) received tactile stimulation of the left or right index finger (counterbalanced). The stimulus was produced by an Optacon reading aid for the blind. The Optacon consists of a 24 by 6 array of probes covering an area of 28 mm by 14 mm; it generates a 250-Hz vibrotactile pulse. The intensity of the stimulus was a 65-μm indentation for a duration of 50 msec. Cortical potentials evoked by 25 stimuli delivered at a rate of one every 2 seconds were automatically summated with a special-purpose (Nicolet 1070) or general-purpose (PDP 8/E) computer. Bioelectric activity was recorded bilaterally from symmetrical placements 6 to 7 cm lateral to the midline in the interaural plane and 1 to 3 cm posterior to this point. In several subjects, recordings were obtained from multiple parasagittal locations. The vertex (Cz) served as a common reference. Preamplifiers (Grass P511) set for a bandpass of 3 to 100 Hz amplified the signal. The average evoked potential waveform was read out, on line (Hewlett-Packard XY plotter), and the latency of the initial positive-negative-positive complex was digitized.

Representative ipsilateral and contralateral evoked potentials for several age groups are presented in Fig. 1. The peaks of interest, P1, N1, and P2, are identified (top tracings). These peaks were selected for their high within-and-between subject reliability. Since they constitute the earliest deflections of the response, their accurate recognition across age and between ipsilateral and contralateral potentials was simplified. Repeated measurements taken within a session or over the course of 2 years in several adults showed a high degree of replicability. Notice the reduced amplitude and prolonged latency of the early components of the ipsilateral response (Fig. 1). With increasing age there is a progressive decline in the ipsi-contralateral difference. Prior to 3.5 years of age, reliable ipsilateral activity could not be obtained. Figure 2 shows the mean

0036-8075/78/0623-1409$00.50/0 Copyright © 1978 AAAS

commissural transmission time (ipsilateral-contralateral latency difference) as a function of age. Analyses of variance performed on the individual components (P1, N1, and P2) showed significant differences ($F = 14.0$, $P < .001$; $F = 10.05$, $P < .001$; and $F = 6.44$, $P < .05$, respectively) (*10*).

While these findings are compatible with the notion of callosal maturation, the origin of ipsilateral activity remains open to question. Despite bilateral representation in the somatosensory system, the ipsilateral response is mediated largely by the contralateral koniocortex via the corpus callosum (*11*). In the cat, section of the corpus callosum or cooling of the opposite hemisphere led to the immediate disappearance of ipsilateral potentials, although all extralemniscal pathways remained intact. Moreover, destruction of cortical areas SI and SII eliminated short-latency evoked potentials in the somesthetic sector of the corpus callosum to direct (callosal) or peripheral stimulation (*12*). That surgical removal of or damage to the contralateral primary receiving area in humans also completely eliminates all cortical evoked activity supports the view that the current fields set up in ipsilateral and extracallosal pathways contribute little to the surface response (*13*).

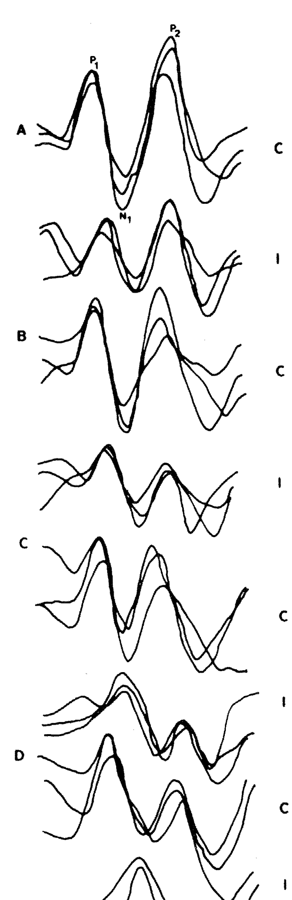
Thus, if ipsilateral activity depends on output from primary and adjacent association cortices, transcallosal potentials would be expected to exhibit a homotopical projection (*12, 14*). In several subjects, recordings were obtained from an array of electrodes placed 1.5 cm apart traversing the sensory hand area of the postcentral gyri (superficial determination). Early activity, particularly P1 and N1, was confined exclusively to the homologous site of maximum amplitude, in contrast to the later waves, which were diffusely represented (Fig. 3). This differential distribution of the various components further strengthens the supposition that early ipsilateral potentials are indeed conveyed commissurally.

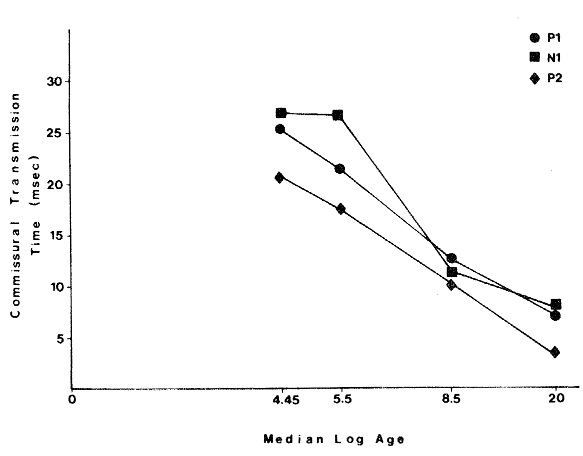
Even more compelling are the striking maturational changes seen in commissural transmission time (Fig. 2), which conform to the known myelogenic cycle of the corpus callosum (*2*). Correlational analysis of the ipsi-contralateral latency difference with age yielded significant ($P < .001$) values for each peak (P1, $r = -.66$; N1, $r = -.67$; P2, $r = -.58$) in the 3.75- to 13-year age range but nonsignificant coefficients from 10 years through adulthood. This indicates that commissural transmission attains symptote by the end of the first decade of life and agrees with previous histochemical observations (*2*).

This study supports the contention that ipsi-contralateral differences reflect somatosensory commissural transmission and that the developmental time course of the corpus callosum can be studied in humans (in vivo). This technique may prove useful in assessing demyelinating disease and cases in which maturational delay is suspected.

A. Salamy

Langley Porter Neuropsychiatric Institute, University of California, and Brain-Behavior Research Center, Sonoma State Hospital, Eldridge, California 95431





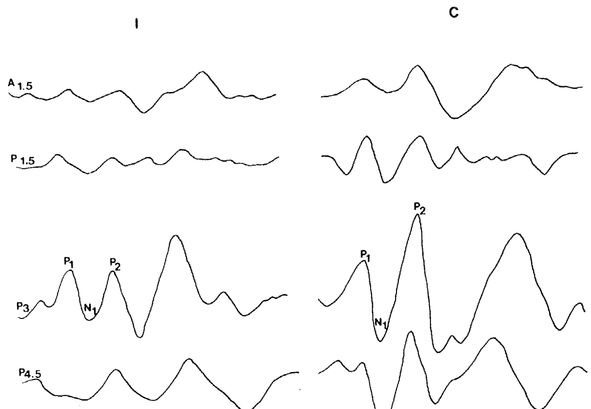


Fig. 1 (left). Somatosensory evoked potentials to tactile stimulation: (A) 10 years to adulthood; (B) 7 to 10 years; (C) 5 to 7 years; (D) 3.75 to 5 years. Each trace represents an average of 25 responses recorded from the contralateral (*C*) and ipsilateral (*I*) hemispheres of a different subject. Note the longer latency and lower amplitude of the ipsilateral response. Also notice the progressive decrease in ipsilateral latency (all peaks) with increasing age despite stable contralateral responses. Fig. 2 (top right). Mean commissural transmission time (ipsi-contralateral latency difference) for three components (*P1, N1, P2*) as a function of age. Fig. 3 (bottom right). Distribution of contralateral (*C*) and ipsilateral (*I*) potentials in an adult subject. Note that *P1* and *N1* of the ipsilateral response are present only at the P_3 (3 cm posterior to interaural plane) location which roughly corresponds to the homolateral sensory receiving area. Late components of unknown origin are bilaterally represented particularly in the posterior quadrant of each hemisphere. The designations *A* and *P* with subscripts refer to the distances in centimeters either anterior or posterior to the interaural plane.

References and Notes

1. W. Hewitt, *J. Anat.* **96**, 355 (1962); P. Flechsig, *Lancet* **1901-II**, 1027 (1901); M. Geschwind, *Brain* **88**, 237 (1965).
2. P. Yakovlev and A. Lecours, in *Regional Development of the Brain in Early Life*, A. Minkowski, Ed. (Blackwell, Oxford, 1967), p. 3; A. Lecours, in *Foundations of Language Development*, E. H. Lenneberg and E. Lenneberg, Eds. (Academic, New York, 1975), vol. 1, p. 121.
3. B. Grafstein, *J. Neurophysiol.* **26**, 79 (1963); P. J. Savarie and M. J. Hoska, *Dev. Psychobiol.* **6**, 301 (1973); M. E. Molliver, *Progr. Brain Res.* **26**, 78 (1967).
4. J. Seggie and M. Berry, *Exp. Neurol.* **35**, 215 (1972).
5. R. Goff, B. S. Rosner, T. Allison, *Electroencephalogr. Clin. Neurophysiol.* **14**, 697 (1962); T. Allison, Y. Matsumiya, G. D. Goff, W. R. Goff, *ibid.* **42**, 185 (1977); G. D. Goff, Y. Matsumiya, T. Allison, W. R. Goff, *ibid.*, p. 57; T. Schenkenberg, thesis, University of Utah (1970).
6. W. B. Hardin and V. F. Castellucci, *Electroencephalogr. Clin. Neurophysiol.* **28**, 448 (1970); R. Cohen, *ibid.* **29**, 612 (1970); J. Cernacek and F. Podivinsky, *Cerebral Interhemispheric Relations* (Bratislava, 1972), p. 69.
7. J. L. Andreassi, H. Okamura, M. Stern, *Psychophysiology* **12**, 541 (1975).
8. R. A. Filbey and M. S. Gazzaniga, *Psychonom. Sci.* **12**, 355 (1969); M. Amadeo, R. A. Roemer, C. Shagass, *Biol. Psychiatry* **12**, 289 (1977); M. White, *Br. J. Psychol.* **63**, 497 (1972); P. Satz, R. Goebel, H. Van Der Vlugt, *Brain Lang.* **2**, 171

(1975); S. Blumstein, H. Goodgrass, V. Tartler, *ibid.*, p. 226; R. J. Porter and C. I. Berlin, *ibid.*, p. 186.

9. Statistical analyses were computed only on those subjects above the age of 3 years 9 months, who displayed reliable ipsilateral responses. This included 15 adults 13 to 40 years old, 11 children 7 to 10 years old, 12 children 5 to 7 years old, and 7 children 3.75 to 5 years old. In addition 7 children 2 to 3.75 years old were tested. All subjects were right-handed. A pilot series conducted on several adults showed no systematic difference in commissural transmission between the left- and the right-handed or between left and right finger stimulation within a given subject.
10. Separate analyses of variance computed for the ipsilateral and contralateral responses revealed that only the ipsilateral response (all components) varied with age. Thus the ipsi-contralateral differences could not be attributed to changes occurring in the contralateral response (Fig. 1). Others have also reported little difference in the latency of early contralateral activity to shock stimulation between 4 years of age and adulthood. Changes in body length seemed to account for the relative consistency across age [see J. E. Desmedt, E. Brunko, J. Debecker, *Electroencephalogr. Clin. Neurophysiol.* **40**, 43 (1976); P. Laget, J. Raimbault, A. M. D'Allest, R. Flores-Gueuara, J. Mariani, *ibid.*, p. 499].
11. D. L. Robinson, *Exp. Brain Res.* **18**, 131 (1973).
12. G. M. Innocenti, T. Manzoni, G. Spidalieri, *ibid.* **19**, 447 (1974).
13. P. E. Stohr and S. Goldring, *J. Neurosurg.* **31**, 117 (1969); P. D. Williamson, W. R. Goff, T. Allison, *Electroencephalogr. Clin. Neurophysiol.* **28**, 556 (1970); W. T. Liberson, *Am. J. Phys. Med.* **45**, 135 (1966).
14. K. A. Hossmann, in *Cerebral Localization*, K. J. Zulch, O. Creutzfeldt, G. C. Gailraith, Eds. (Springer-Verlag, Berlin, 1975), p. 150; N. Nakahama, *Int. Rev. Neurobiol.* **3**, 187 (1961).
15. I thank T. Mendelson and A. Amochaev for their technical assistance in collecting the data. The Montessori School of San Francisco and the Sonoma Valley High School and Alta Mira Junior High School of Sonoma County provided the subjects.

1 November 1977; revised 8 February 1978

Lateralization of Norepinephrine in Human Thalamus

Abstract. *Norepinephrine has a strongly lateralized distribution in the human thalamus. In the pulvinar region the left hemisphere is rich in norepinephrine, whereas in the somatosensory input area the right hemisphere has a higher concentration of this catecholamine. Such naturally occurring left-right differences in concentration of a neurotransmitter represent a new aspect of hemispheric specialization.*

In this report we provide evidence of a significant, naturally occurring lateralization in the distribution of a neurotransmitter system in the human brain. These findings add a new dimension to laterality concepts—that of left-right (L-R) variations in the "chemical neuroanatomy" of the central nervous system—the significance of which we think might be correlated in the future with functional and behavioral effects. The introduction to a recent symposium on hemispheric specialization and lateralization in the nervous system suggests that, "the lateralization problem impinges upon the entire spectrum of brain-behavioral research from the synapse to the sentence" (*1*). Here we provide strong evidence of the chemical laterality.

Recently developed assay procedures enable us to analyze rapidly and accurately the concentration of norepinephrine (NE), epinephrine, dopamine, and ascorbic acid in brain tissue samples weighing between 0.5 and 50 mg. Using small punches from coronal slices we initiated a detailed mapping of these compounds in selected areas of human brain. Occasional bilateral punching, designed to check the precision of the assays and our neuroanatomical expertise (or lack thereof), gave analyses with surprising L-R differences in certain areas. These differences far exceeded the estimates of uncertainties in the assays or punching techniques. The clinical histories did not provide any evidence of hemineurological dysfunction which might account for such results. Accordingly, we began studies of tissue samples from accurately placed bilaterally symmetric punches. The data from five brains are summarized herein and primarily concern the L-R distribution of NE in the thalamus, an area in which we have been particularly interested because of the possible involvement of catecholamines in somatosensory pathways.

Brains were obtained after routine autopsy with the time between death and autopsy varying from 9 to 16 hours (average, 12 hours). Immediately upon removal the whole brains were placed in a −70°C freezer and kept at this temperature until ready for sectioning. The temperature of the specimen was then allowed to rise until slicing could be accomplished; however, the brain remained in the frozen state at all times. These procedures are qualitatively similar to those used by other investigators in postmortem neurochemical studies (*2*). Coronal slices 3 to 4 mm in thickness were cut, placed on a glass plate over Dry Ice, and photographed in color. Samples were removed from the desired areas with a sharpened punch. These samples routinely weighed 30 to 40 mg and were about 3 to 5 mm in diameter. The slice was rephotographed after punching and anatomical assignments were confirmed from the photographs. Standard whole brain and special thalamic atlases were used as required (*3*). Tissue punches were immediately returned to the −70°C environment until analyzed. Catecholamine (*4*) and ascorbic acid (*5*) concentrations were determined by liquid chromatographic procedures with electrochemical detection. The analyses were conducted blind, in that the analyst did not know the side or exact location from which a given sample was obtained.

The results showing thalamic cate-

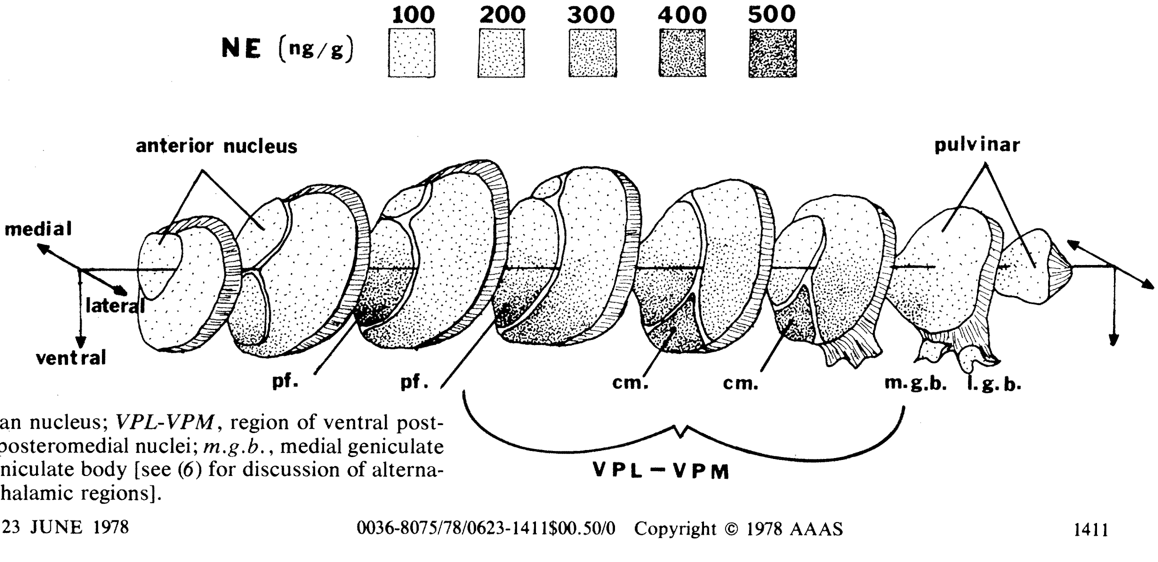


Fig. 1. Regional norepinephrine (NE) distribution in human thalamus. The NE values were averaged over both thalami from five separate brains, but are represented here on a single (left) thalamus. Although precise anatomical localization is not intended, general subdivisions and nuclei of interest are designated on the diagram: *pf*, parafascicular; *cm*, centromedian nucleus; *VPL-VPM*, region of ventral posterolateral and ventral posteromedial nuclei; *m.g.b.*, medial geniculate body; *l.g.b.*, lateral geniculate body [see (*6*) for discussion of alternative nomenclature of thalamic regions].

0036-8075/78/0623-1411$00.50/0