References and Notes

1. W. E. Garrey, *J. Cellular Comp. Physiol.* **1**, 209 (1932); P. Heinbecker, *Am. J. Physiol.* **117**, 686 (1936); C. L. Prosser, *J. Cellular Comp. Physiol.* **21**, 295 (1943).
2. B. F. Hoffman and P. F. Cranefield, *Electrophysiology of the Heart* (McGraw-Hill, New York, 1960).
3. T. H. Bullock and C. A. Terzuolo, *J. Physiol. (London)* **138**, 341 (1957); S. Hagiwara and T. H. Bullock, *J. Cellular Comp. Physiol.* **50**, 25 (1957).
4. A portion of the work reported here was carried out at the Marine Biological Laboratory, Woods Hole, Mass.

3 April 1962

Repeated Homing Exhibited by a Female Pallid Bat

Abstract. A pallid bat (*Antrozous pallidus*) returned home from eight consecutive releases from six distinctly different directions and from distances ranging from 21 to 68 miles. This performance indicates that chance alone cannot be a major factor in homing, and that certain abilities possessed by this bat, and not simply randomness, must have been in operation.

For several years bats have been known to return to the point of capture after having been released elsewhere—an action usually reported as demonstrating homing ability and often interpreted as being the result of a "homing instinct." Several hundred bats, involving both Old World and New World species, have been marked in various ways and transported for distances up to 500 miles. Individuals have returned from distances as great as 450 miles (*1*), and the time of recovery has been as long as 5 years (*2*).

The majority of these experiments have been concerned with determining the percentage of bats returning to the point of original capture. Typical homing experiments, though the results are highly variable, usually show very low percentages of return, especially when the release points are several miles from the point of origin. These low percentages of return have been used to support the theory that so-called "homing ability" is simply the result of random dispersal from a release point, with individuals scattering in all directions, and with a few arriving home as a result of chance selection of the proper return route.

Little evidence in the literature can be used as an effective argument against randomness as an explanation for homing. Obviously, many other factors could contribute to low percentages of return, but at present it appears difficult to eliminate chance as one alternative. Rapid return flights, such as those reported by Cockrum (*3*) and by Mueller and Emlen (*4*), as spectacular as they may appear, could still be explained on the basis of chance since in both cases the rapid returns were accompanied by relatively low percentages of return. Even the extreme distance of return (450 miles) recorded by Smith and Goodpaster (*1*) with *Eptesicus fuscus*, which greatly lowered the probability of return by chance, did not eliminate the effect of randomness since the percentage of return was correspondingly low.

During the summer of 1960, we conducted a series of homing experiments with pallid bats (*Antrozous pallidus*) from a roost near St. David, Cochise County, in southeastern Arizona. During the analysis of results we found an interesting occurrence which casts serious doubt on the idea that mere chance is a major explanation of homing. While only a low percentage of pallid bats returned from any given experiment, it became evident that several individuals showed unusual consistency in their returns. The most spectacular of this group was a female (band number 10–51606) whose travels are summarized in Table 1 and Fig. 1. This female returned from eight consecutive releases from at least six distinctly different directions and from distances ranging from 21 to 68 miles. In the course of this study (between 28 May and 1 October) she had traveled a total of more than 450 miles.

Table 1. Summary of the homing of a female pallid bat. The recovery dates indicate only maximum possible time of return and should not be interpreted as actual "return dates." All releases and recoveries were made during the summer of 1960.

Release date	Release point	Recovery date
28 May	Sonoita	3 July
3 July	Mescal	17 July
17 July	Tucson	23 July
23 July	Nogales	6 Aug.
6 Aug.	near Willcox	27 Aug.
27 Aug.	Douglas	11 Sept.
11 Sept.	Duval Mine	17 Sept.
17 Sept.	Rodeo, N.M.	1 Oct.
1 Oct.	Mammoth	None

In all probability, the majority of these return trips were made from regions outside of the home range and therefore represent homing from unfamiliar territory. While no direct proof is available for this conclusion, our reasoning includes indirect evidence obtained both from experimentally determined flight speed and from an estimate of population home range based on banding records of members of this species (*5*). In no case do our data suggest the likelihood of an area of familiar territory which would approach a 50-mile radius in all directions—the approximate distance from which most of these returns were made. It is possible that the area of familiar territory might

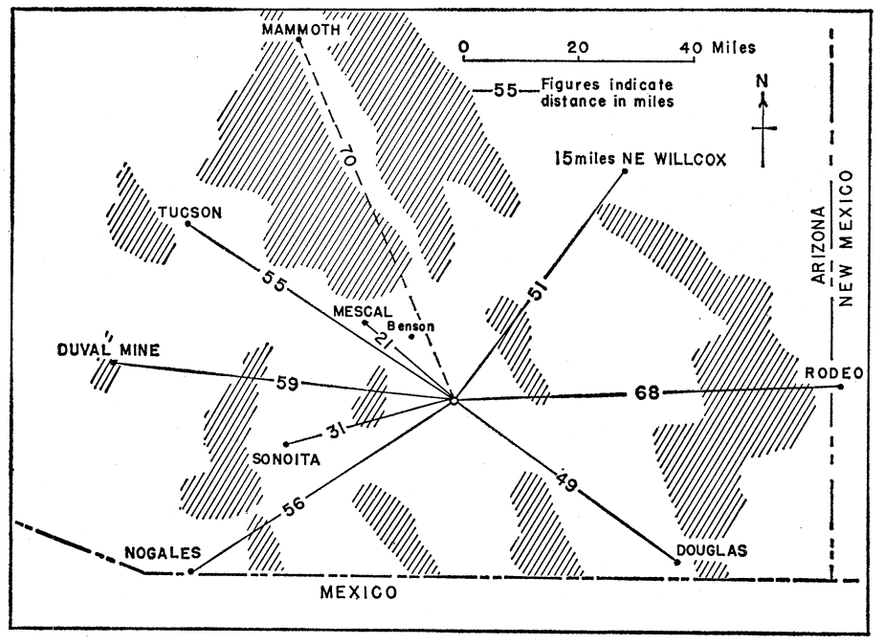

Fig. 1. Southeastern Arizona, showing spatial relationships of release points. The straight lines do not suggest actual routes taken on return trips. The bat was not recovered after its release at Mammoth. [Drafted by T. Shaman]

be considerably extended in one direction due to the use, by this population, of a migratory route to a winter roost. However, this certainly could not explain the successful return of this bat from the diversity of directions demonstrated in this study.

The failure of this individual to be recovered following her last release at Mammoth on 1 October was originally thought to be due to the fact that the bridge from which she was taken is used as a roost only during the warmer months of the year and was abandoned by all pallid bats some time between the period of 1 through 8 October. However, during repeated checks at this same bridge during the following summer we failed to recover this individual even though several other pallid bats, which had been used in the 1960 experiments, were in continuous residence throughout the summer.

From these results, it does not seem reasonable that chance alone would have permitted this bat to show such a consistency of return. Surely some abilities possessed by this bat, and not simply randomness, must have been in operation. The problem of low percentage of return, however, still remains and must be taken into account by any explanation of the mechanism of bat homing. Did this particular bat possess or develop abilities which are unique and which are absent in most other members of the same species? Individual variation is, of course, to be expected among the members of any species. Ordinarily, however, one would not expect variations in the presence of any aptitude or ability to range from extremely high values in certain individuals to near zero in others. Obviously, much additional evidence is needed to answer this and many other questions concerning homing in bats (*6*).

RUSSELL DAVIS
E. LENDELL COCKRUM
Department of Zoology,
University of Arizona, Tucson

References and Notes

1. E. Smith and W. Goodpaster, *Science* **127**, 644 (1958).
2. E. L. Cockrum, *J. Mammal.* **30**, 433 (1949).
3. ———, *ibid.* **34**, 48 (1956).
4. H. C. Mueller and J. T. Emlen, *Science* **126**, 307 (1957).
5. Papers concerning these data (speed of flight and population home range) are in preparation. We hope that more direct evidence can be obtained with a subminiature radio transmitter being developed by Howard Baldwin of the University of Arizona.
6. This report is the result of activities supported in part by grants from the National Science Foundation (G-5209) and the National Institutes of Health (E-3147).

12 February 1962

Chronic Effect of Tetanus Toxin Applied Locally to the Cerebral Cortex of the Dog

Abstract. Tetanus toxin injected into the cortex produces convulsions, neurological disability, and a strychnine-like discharging focus, appearing after a minimal latency of 2 days and lasting more than a month. Such a focus, apparently caused by a blockade of inhibitory synapses, does not demonstrate any major damage in microscopic studies.

The present study was undertaken in order to find an effective method of producing a chronic "epileptogenic" focus in the gray substance without significant structural changes. The purpose of this preliminary communication is to show how this has been accomplished with tetanus toxin in the cerebral cortex of the dog.

The acute action of tetanus toxin upon the spinal cord was recently studied (*1*, *2*). Its chronic effect upon the iris (*3*) and the pathogenesis of "local" or peripheral tetanus is still a matter of controversy (*4*). To our knowledge, the chronic action of tetanus toxin applied locally to the cerebral cortex has heretofore never been studied.

Under sterile conditions, 50 to 150 μg of tetanus toxin was injected immediately under the pia mater in 66 dogs. The dry toxin ($LD_{50} = 1.8$ μg) was kept in a vacuum container and placed in a refrigerator. A 0.1-percent solution in saline was prepared immediately before its use.

Thirty-four animals (group I) showed major convulsions. These appeared between the second and seventh day after the operation and lasted until the animals were sacrificed, or died, usually as the result of recurrent convulsions. In three dogs which were allowed to survive for more than a month, the convulsions diminished and finally disappeared by the end of the second month. Between convulsions, the animals showed disturbances of motor performance and behavior. The most significant findings observed were diminished strength, awkwardness of movements, and a brisk withdrawal response of the limbs on superficial stimulation, apparently leading to "steppage" during gait. These effects were contralateral to the side of injection.

Five animals (group II) showed minor motor disturbances but convulsions were not recorded, although they could have occurred and escaped observation. Twenty dogs (group III) showed no convulsions and no motor disturbances. This group includes three controls injected with inactivated toxin and those who received smaller amounts of tetanus toxin (see below). Seven animals (group IV) died within the first 2 days of the operation; unobserved convulsions might possibly have been the cause of death in these cases.

The spontaneous electrical activity of the brain was recorded with a Grass polygraph in intact nonanesthetized and in curarized animals. Needle electrodes inserted through the skin and small (0.2 to 0.5 mm diameter) silver ball electrodes applied to the surface of the exposed cortex were employed. Electrodes implanted at the original operation were used in a few circumstances for recordings in unrestrained animals. The usual technique was bipolar recordings with a 2- to 3-mm interelectrode distance. Unipolar recording and bipolar recording with different sized silver balls, leading the smaller to grid one, were employed to ascertain the polarity of the electrical events.

The common finding was the recording of a triphasic spike with an average total duration of 250 to 300 msec and an average peak to peak amplitude of 0.2 mv when recorded from the skin and 1.5 mv when recorded from the cortical surface. The outstanding event was a negative component, of 100-msec duration, which was preceded by a sharp positive deflection and followed by a variable, usually smoother positive wave (Fig. 1). Such spikes would appear at random but more frequently they were observed at slightly irregular intervals, of about 1 second duration, with random short silent periods (Fig. 1) or in bursts of three to ten spikes. Any one of these three patterns of activity could be alternately observed in the same experiment. These "tetanic" spikes were similar to those observed after the application of strychnine to the otherwise normal cortex.

The records showed that these spikes occurred in a small area about 5 mm in diameter, centered at the site of the previous injection of tetanus toxin. Occasional spikes or slightly abnormal cortical activities were sometimes recorded in the adjacent area. The remaining ipsilateral cortical areas as well as the cortex of the opposite hemisphere were normal. Abnormal activity transiently spread to adjacent previously normal areas during convulsive discharges.

Such abnormal electrical activity was more remarkable in animals that demonstrated convulsions (group I). It was also seen in animals showing mild neurological abnormalities but no convul-