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THE EFFECTS OF DORSAL HIPPOCAMPAL LESIONS ON THE ENTRY INTO HIBERNATION IN THE BAT

BY

Gerald R. Conover

A dissertation submitted in partial fulfillment
of the requirements for the degree
of Doctor of Philosophy in
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ABSTRACT

THE EFFECTS OF DORSAL HIPPOCAMPAL LESIONS ON THE ENTRY INTO HIBERNATION IN THE BAT

by

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Recently, the attention of investigators has been focused on the neural mechanisms involved in hibernation. The hypothalamus has been shown to be involved in the entry into hibernation. It has also been reported that the dorsal hippocampus has a unique EEG pattern of activity during the entry into hibernation. The purpose of this study was to determine what influence the dorsal hippocampus has on the entry into hibernation.

The hibernator chosen as the experimental animal was the little brown bat, Myotis lucifugus lucifugus. The findings from 52 animals are reported in this study. Electrolytic lesions were placed in the forebrains of 30 animals. Twenty of the animals had sizable damage to the dorsal hippocampus. On the day following the lesions, the responses of the animals when placed in the cold were recorded. The parameters recorded during the experiments were the body temperatures and heart rates of the animals.

A normal pattern for the entry into hibernation was established in 17 normal animals. Five sham operated animals

and ten animals with lesions of brain areas other than the dorsal hippocampus had entry into hibernation patterns similar to the patterns of the normal animals. Nine of the 20 animals with dorsal hippocampal lesions had delayed entry into hibernation patterns as compared to the patterns of the control animals. Eleven of the animals with dorsal hippocampal lesions did not have delayed entry into hibernation patterns.

Suggestions are made, based on the observations of this study and other studies, as to why these latter animals did not have delayed entry into hibernation patterns.

The possible pathways and mechanisms by which the dorsal hippocampus influences the entry into hibernation are discussed. Future areas of investigation are also discussed.

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INTRODUCTION

There are a number of mammals that enter a unique physiological state termed hibernation. Hibernation is characterized by a drop in an animal's body temperature in a cold environment (usually 0°-10°C.) from warm-blooded levels (32°-39°C.) to a temperature near that of the environment. At the same time the animal's heart rate and metabolic rate also decline to very low levels.

This remarkable ability of a small number of mammals to drastically lower their body temperature is not restricted to any one zoological group. Instead, there are a few species which hibernate in several orders. The orders which contain hibernators include Insectivora, Primates, Chiroptera and Rodentia (Lyman, '62). In some species hibernation can only occur during the winter months; in other species it can occur in any season (Lyman, '61). Hibernators may wait in the cold for weeks, days, hours, or minutes before lowering their body temperature (Lyman, '54; Lyman, '61). It may take minutes or days for the body temperature to reach its lowest level (Strumwasser, '59; Lyman, '61). The mammals can be as physically different from one another as European hedgehogs, dwarf lemurs, little brown bats or American woodchucks, but they all have in common the unique ability of being able to hibernate (Johnson, '31). Figures 1, 2 and 3 demonstrate the entry into hibernation of three well-known hibernators: the cave bat, the little brown bat and the California ground squirrel.

Hibernation was formerly thought to be similar to the lethargic state of cold-blooded animals (Rasmussen, '16). Animals which hibernate were thought to possess imperfect temperature regulating mechanisms. Thus, in the cold, it was not surprising that their body temperatures would fall to the level of the ambient air. However, it is now known that all hibernators are capable of maintaining a high body temperature in a cold environment (Pearson, '47; Lyman, '62; Herreid and Schmidt-Nielsen, '66; Stones and Wiebers, '65). Also, investigators have shown that the declining body temperature of an animal entering hibernation, as well as the low body temperature during hibernation, are regulated conditions involving both the processes of heat production and of heat loss (Strumwasser, '59; Lyman, '61; Henshaw, '68). Finally, it is now recognized that all mammals in hibernation can endogenously produce heat and return to the warm-blooded condition.

Animals capable of hibernating have been designated as heterotherms because of their ability to regulate their body temperatures at both warm-blooded and cold-blooded levels. The majority of mammals are able to regulate their body temperatures at high levels only. These latter animals have been designated as homeotherms. The terms heterotherm and homeotherm imply that the animals in these two groups have a different temperature regulating mechanism. The temperature regulating mechanism of the heterotherm is most obviously different from that of the homeotherm in its ability to lower the body temperature to hibernating levels. Today

investigators are beginning to consider the temperature regulating mechanisms of heterotherms as being quite similar in many ways to those of homeotherms. The current reconsideration of the heterotherm-homeotherm relationship is due to the accumulation of experimental data clarifying the physiology of temperature regulation in these two groups.

It is well known that blood temperature-sensitive centers in the preoptic-anterior hypothalamic area of homeotherms function to regulate the body temperature at a fairly constant level (Teague and Ranson, '36; Magoun et al., '38; McCrum, '53; Freeman and Davis, '59; Hammel et al., '60; Andersson et al., '63; Eisenman and Jackson, '67; Squires and Jacobson, '68). Heating or electrical stimulation of this area elicits a coordinated response of the heat loss mechanisms, i.e. panting, sweating, cutaneous vasodilatation and inhibition of shivering. Cooling of this same area elicits coordinated heat production responses, i.e. shivering, release of thyroxin and cutaneous vasoconstriction. Thermal stimulation of other parts of the central nervous system does not elicit coordinated temperature regulation responses.

The thermal-sensitive centers have been analogized to a thermostat because their activity tends to balance the body temperature at a constant level or set point (Brobeck, '60). The setting of the physiological thermostat is higher or lower in such conditions as fever, exercise, sleep and wakefulness and under emotional stress. Fever is a dramatic example of the setting of the physiological thermostat at a higher level. Fox and Macpherson ('54) did comparative studies on

temperature regulation in febrile patients and in healthy subjects under thermal stress. Both quantitatively and qualitatively the thermoregulatory events were found to be the same with the exception that they all occurred at a higher temperature in the febrile patients. Thus the body is balanced at a new and higher temperature in the febrile patients, the physiological thermostat being reset at a higher set point but otherwise functioning normally. Similar phenomena have been observed in experimental animals following the administration of fever-producing agents (pyrogens). The heating of the preoptic-anterior hypothalamic area in un-anesthetized dogs has been shown to prevent the febrile response to pyrogens or to bring the fever temperature down to normal (Andersen et al., '60). This experiment indicates that the action of the particular pyrogens employed is to elevate the setting of the physiological thermostat.

A phenomenon analogous to fever may occur in mammals capable of hibernating. A number of investigators have postulated that the setting of the physiological thermostat of an animal entering hibernation is gradually lowered from a high level to a level just above that of the cold air of the environment (Lyman, '58; Strumwasser, '59; Koski, '68, Mrosovsky, '68). Hibernation would then be a condition in which there is a radical change in the sensitivity of the preoptic-anterior hypothalamic neurons which regulate body temperature.

In summary, it has been postulated that heterotherms

and homeotherms possess similar temperature regulating centers and that heterotherms, under certain conditions, are uniquely able to utilize these centers to lower their body temperature to levels near the freezing point of water.

Lesions of the hypothalamus in different species of homeotherms have produced alterations in temperature regulation, feeding and alertness. Lesions in the preoptic-anterior hypothalamic area interrupt normal regulation of temperature when the animal is heat or cold stressed (Andersson et al., '65; Keller, '63). Lesions in the posterior hypothalamus also interrupt temperature regulation and cause somnolence (Ranson, '39; Nauta, '46). The destruction of the ventromedial nuclei leads to overeating (hyperphagia) and rapid weight gain, whereas damage to the lateral hypothalamus causes an indifference to food (aphagia) and starvation (Anand and Brobeck, '51; Teitelbaum and Stellar , '54). Satinoff ('67a) destroyed corresponding areas in the hypothalamus of hibernators (two species of ground squirrels) and reported results such as impairment of temperature regulation, somnolence, hyperphagia and aphagia. Satinoff concluded from her study that "it appears that corresponding areas in the brains of homeotherms and hibernators subserve the same functions."

Satinoff ('67b) also reported an interesting observation from a later experiment on the effects of hypothalamic lesions on ground squirrels. Following preoptic-anterior hypothalamic and ventromedial hypothalamic nuclei lesions, the squirrels were placed in a 6°C. cold room and their

brain temperatures recorded. One of the animals which had become tremendously obese following ventromedial nuclei lesions was extremely slow in lowering his brain temperature to hibernation levels when compared to the normal and other lesioned animals. Koski ('68) tested the ability of unfed little brown bats to enter hibernation following various hypothalamic lesions. Preoptic-anterior hypothalamic and lateral hypothalamic area lesions produced animals which became hypothermic on exposure to a cold stress. Bilateral lesions just lateral to the ventromedial hypothalamic nuclei considerably slowed the little brown bat's entry into hibernation. These experiments indicate definite hypothalamic involvement in the entry into hibernation, at least in the species investigated.

The observations of one investigator suggest that the hypothalamus may not be the only brain center involved in the entry into hibernation. Strumwasser ('59) recorded the brain temperature and shivering activity of California ground squirrels during the entry into hibernation. He also recorded the electrical activity of many areas of the forebrain and brain stem at the same time. The chronic muscle leads showed that the ground squirrel intermittently shivers during the entry into hibernation. The intermittent short bursts of shivering create minute plateaus in the declining brain temperature. Strumwasser was able to correlate the intermittent shivering and resultant brain temperature plateaus with definite constant changes in the EEG activity

of three brain areas. These brain areas are the lateral motor cortex, basolateral amygdala and dorsal hippocampus.

Strumwasser's EEG records show that during a brain temperature plateau there is a grouping of a 9-10 cps rhythm in the motor cortex growing in amplitude and continuity until a peak amplitude is reached where shivering takes place. The 10 cps rhythm in the motor cortex continues throughout shivering and for several seconds after shivering. A constant finding seen after shivering as the motor cortex activity dies is the appearance of a high amplitude 5 cps rhythm in the amygdala during which an identical rhythm in the dorsal hippocampus breaks out. The hippocampal rhythm appears in the midst of the amygdaloid burst which outlasts the hippocampal activity. The motor cortex activity-shivering relation and the amygdaloid-hippocampal activity pattern were found to be unique to the state of entering hibernation. This activity pattern was observed hundreds of times by Strumwasser. Strumwasser suggests that the observed specific EEG patterns during entry into hibernation may indicate a "hierarchy of control systems exerting their influence on the fundamental system responsible for shivering; that is, these higher systems are determining the initation and termination of shivering and its integration into the coordinated pattern responsible for the form of the curve of the temperature drop."

The hippocampus in homeotherms has not been shown to be involved in temperature regulation. However, a number of

investigators have demonstrated that the hippocampus is anatomically connected to brain areas involved in temperature regulation. Humphrey ('36) has described in the free tailed bat, Tadarida mexicana, fornix fibers distributing to the preoptic-anterior hypothalamic area. A less known pathway of the hippocampus is the temporo-ammonic tract. A projection from the hippocampus by way of the temporo-ammonic (or ammonic-temporal) tract to the entorhinal area and from there via the striae medullaris to the midbrain tegmentum has been found in the phalanger (Adey et al., '58). Since the reticular formation of the midbrain has been implicated as an area which regulates the sensitivity of the hypothalamic temperature regulating centers (Euler and Söderburg, '57 and Squires and Jacobson, '68) hippocampal activity could theoretically influence the temperature regulation centers through similar circuits in a hibernator.

The hippocampus is also functionally connected to the hypothalamus and brain stem reticular formation. Hippocampal activity appears to be related in some way to TSH and ACTH release from the pituitary (Schizume, et al., '62; Knigge, '60). Recently investigators have made suggestions based on experimental evidence that the hippocampus exerts an inhibitory influence on the brain stem reticular formation (Korányi, et al., '67; Redding, '67).

I chose to investigate the influence of a brain center other than the hypothalamus on the entry into hibernation. The neural center investigated was the dorsal hippocampus.

(The term hippocampus is used in this study to refer to Ammon's horn and the dentate gyrus.) This neural center was focused on for the following reasons.

(1) The reasonable hypothesis, supported by recent experimental data, that hibernators utilize temperature-regulating centers in the preoptic-anterior hypothalamic area to lower their body temperatures to hibernating levels.

(2) Strumwasser ('59) has described a specific EEG activity in the dorsal hippocampus which is only present during the entry into hibernation.

(3) The hippocampus is connected to areas involved in temperature regulation.

(4) The size and location of the dorsal hippocampus lends itself to experimental investigation.

The classic method for demonstrating the involvement of various neural areas in a given function is to destroy the areas and observe the results. This was the method I chose for my experiments.

The little brown bat, Myotis lucifugus lucifugus, was selected as the experimental animal for this study. The little brown bat was selected for three reasons.

(1) The little brown bat is a hibernator.

(2) The bat, upon exposure to a cold environment, immediately or within hours begins to enter hibernation, a characteristic which eliminates the necessity of long term recording apparatus.

(3) The little brown bat was available in quantity within a reasonable distance and is easy to maintain in the laboratory.

MATERIALS AND METHODS

I. Experimental Animals

The little brown bat, Myotis lucifugus lucifugus, was the experimental animal chosen for this study. The bats were collected from their winter habitats in Indiana and the Upper Penninsula of Michigan in January and April of 1969, respectively. All the animals were stored in a high humidity refrigerator at $5^{o} \pm 2^{o}C$. where they hibernated until used. The bats were kept in the refrigerator in circular metabolic cages (9" high, 8" in diam.) which contained drinking water.

The findings from 52 bats are reported in this study. The majority of the experimental animals were males (N = 48). A few of the control (N = 2) and lesioned (N = 2) animals were females. The bats were weighed as they were taken from the refrigerator for the experiments. The body weights of the bats ranged from 5.2 gm to 7.6 gm. The bats which had body weights of 6 gm or more (N = 35) were not fed at anytime. These bats constituted the unfed group of experimental animals. The unfed bats, following a brief warming up period, were immediately subjected to the experimental routine. The bats weighing less than 6 gm (N = 17) were fed mealworms. The purpose of the feeding was to increase their body weights to 6 gm or more. These bats constituted the fed group of experimental animals. The fattening period lasted from two to four days following the removal of the animal from the refrigerator. The feeding period was terminated at least ten hours prior to the beginning of the experimental routine.

During the feeding period the bats were kept in a 32°C. incubator. Drinking water was available at all times to both the fed and unfed animals.

II. Experimental Routine

The experimental routine was divided into two parts. The first part consisted of preparing the animals for testing. The second part was the testing of the entry into hibernation. Both parts of the experimental routine were initiated at standardized times. The preparation of the animals began between the hours of 9:00 A.M. and 11:00 A.M. The testing period started the following day between the hours of 7:00 P.M. and 9:00 P.M. The purpose of the definite starting times was to give each animal approximately an equal recovery time from the anesthetic and surgical procedures before testing. The standardized starting times also eliminated the possibility that diurnal activity rhythms might affect entry into hibernation.

1. Preparation Procedures

The bats were divided into four groups according to their pretesting preparation. The groups were 1) normal control (N = 17); 2) sham operated control (N = 5); 3) lesioned control (N = 10); and 4) lesioned (N = 20) animals. The preparation procedures were performed using clean surgical techniques under ether anesthesia. All the animals had a segment of a .034 x .050" (inside x outside diameters) polyethylene tube (Clay-Adams Inc., N.Y.) implanted in the subcutaneous tissue of their backs. The end of the subcutaneous segment of the

tube was closed and lay between the scapulae. The free segment of the tube emerged at the base of the animal's tail. This segment was open at the end. The tube was secured in position by sutures. The polyethylene tube served as a receptacle for a thermocouple (fig. 4a). The tube implantation was the only preparation procedure performed on the normal control animals. The rest of the animals were placed in a small animal stereotaxic instrument (David Kopf Instruments, Tujunga, Cal.). The stereotaxic instrument was modified to accommodate the bat's head by using phone tip plugs for ear bars and a ⊔-shaped thin metal wire for a incisor bar. The snout was secured to the incisor bar by a thin strip of masking tape (fig. 5). The desired lesion coordinates had been determined previously by: 1) gross dissection and measurement, and 2) placing lesions at various coordinates in discarded animals. A midline incision was made in the bat's scalp and the skin, connective tissue and muscle were reflected away from the midline. The skull and dura mater over the desired lesion sites were pieced with a sharp surgical needle. An electrode was lowered to the appropriate depth and lesions produced. The electrode was held by a microelectrode holder which in turn was secured to the stereotaxic instrument by a universal holder (David Kopf Instruments, Tujunga, Cal.) (fig. 6). The electrode was constructed of tungsten wire (0.003 inch diameter) insulated with Insl-X (The Insl-X Products corporation, Yonkers, N.Y.) except for its tip. The lesions were produced by passing a 2.5 to

3.5 ma anodal direct constant current through the electrode for 10 to 20 seconds. The circuit was completed by an inactive electrode in the skin of the animal's neck. The current was delivered by a constant current stimulator (Electronics for Life Sciences, Rockville, Md.) (fig. 6). The sham operated animals did not receive current. The animals referred to as lesioned animals were found to have sizable damage to the dorsal hippocampus. The surgical wound areas were closed with suture and swabbed with betadine. All the animals received a 0.5 cc intraperitoneal injection of normal saline following the preparation procedures. The prepared animals were placed in a 32°C. incubator until testing.

2. Testing Procedure

The test period began the day following the preparation procedures. The prepared animal was removed from the incubator and placed in a restraining device made from a plastic 30 cc syringe tube. A 22 gauge, 2" steel syringe needle was placed against each wing and a thermocouple was inserted into the implanted polyethylene tube. The syringe needles were connected to leads and served as EKG electrodes (fig. 4). Electrode paste was used to insure electrical contact between the EKG electrodes and the wings. The thermocouple was of copper/constantan (Science Products Corporation, Dover, N.J.) and had been previously calibrated to a polygraph channel to record temperatures from 0° to 40°C. The syringe tube containing the bat was suspended from the roof of a 17" x 9" x 5" open bottom plastic box so that the bat was in an upside-down position

which is the normal hibernating position for these animals (fig. 7). The apparatus containing the bat was then placed in a 32°C. incubator for two hours. This two hour period allowed the animal to become accustomed to the apparatus. The animals appeared to readily adapt to the apparatus even before being placed in the incubator.

The test period was initiated following the two hour incubation period by removing the apparatus containing the bat from the warm environment (32°C. incubator) and placing it in a cold environment (refrigerator). The leads from the thermocouple and EKG electrodes were connected to channels of a Grass model 5 polygraph (Grass Instrument Company, Quincy, Mass.). The thermocouple reference junction was immersed in a 0°C. distilled water/ice bath kept in the refrigerator (fig. 8). A probe of a YSI thermometer (Yellow Springs Instrument Co., Yellow Springs, Ohio) was placed in the immediate area of the bat to record the refrigerator temperature. The heart rate and temperature of the bat were recorded on polygraph paper from the moment of initial exposure to the cold until hibernation had been attained. The temperature of the refrigerator was read from the dial of the YSI thermometer at five minute intervals and written down on the polygraph paper.

Following a recording session, the bats were kept in the refrigerator connected to the recording equipment for 8 to 10 hours. After this period, new recordings were made to see if the bats were alive and still hibernating. All the

animals were alive and still hibernating with the exception of one of the animals in the lesioned group which died during the second recording session. Following the second recording period the bats were removed from the refrigerator, were sacrificed and their brains removed and stored in 10% formalin. The brains of the animals in the lesioned and lesioned control groups were sectioned at 20 micra and stained with cresyl violet and eosin.

III. Analysis of Data

The first step in the analysis of the data was to prepare graphs from the polygraph records. The heart beat spikes were counted at the beginning of each five minute interval of time and expressed in beats per minute. The temperature of the animal was also determined at the beginning of each five minute interval. The temperature of the refrigerator had been written down on the polygraph paper at the beginnings of the five minute intervals during the recording session. Thus the data will be expressed on graphs as animal temperature in degrees centigrade, heart rate in beats per minute and refrigerator temperature at the beginnings of five minute intervals of time. Zero time on the graphs represents the moment of the animal's initial exposure to the cold. An animal is considered to be in hibernation when his temperature is within three degrees of the refrigerator temperature. The recording sessions at times were terminated some time after the animal had attained hibernation. The records were kept going because of fluctuations in the

animal's heart rate. Occasionally near the end, the data of these records will be expressed at the beginnings of ten minute intervals or longer.

IV. Method of Representing the Lesions

An atlas composed of photographs of the brain of the little brown bat, Myotis lucifugus lucifugus, in transverse section is presented in the Appendix. The sections were cut at 15 micra and stained with Lillie's variant ('60) of the Weil-Weigert stain for myelin. The photographs in the atlas are of sections at 225 micra intervals. The transverse planes of the various sections are indicated on the parasagittal section of the atlas. The solid lines on the parasagittal picture (A,B,C,E and G) indicate the transverse levels which are used to represent the lesions. The dashed lines on the parasagittal picture (D,F and H) indicate the transverse levels which were not used to present the lesions.

The slides (levels A,B,C,E and G) from which the brain atlas photographs were made, were projected by a Promar macroprojector (Ernst Leitz GMBH Wetzlar, Germany) onto drawing paper and traced. Small tracings of entire projected sections were made as well as larger tracings of the dorsal area of the brain. The larger tracings represent enlarged dorsal portions of the small tracings. These tracings are included on the pages preceding the appropriate brain atlas picture.

The slides of the brains with lesions were projected onto the previously described tracings of the atlas slide sections and the lesioned areas darkened in. Care was taken

to match the approximate levels of the projected lesioned sections with the levels of the traced slide sections. The tracings with the lesions drawn in are presented with the graphs which show how the animal performed during the testing period. The tracings are also lettered in the upper left hand corner so that the atlas section (A,B,C,E or G) level they represent may be determined.

There are also two sets of tracings with large areas darkened in. These sets of tracings will be used to compare as a group the locations of the lesions of the lesioned control animals and lesioned animals.

RESULTS

The data reported here were gathered from 52 bats. The animals were separated into four groups: (1) a normal control group (N=17); (2) a sham operated control group (N=5); (3) a lesioned control group (N=10); and (4) a lesioned group (N=20). The differences in the treatment of each group have been enumerated above (see page 11). The data for each group of animals will be discussed separately.

I. The Normal Control Group

Homeotherms, when exposed to a cold stress, are able to make certain physiological adjustments which serve to maintain their body temperature at a high level. The maintenance of a high body temperature in a cold environment will be referred to as thermoregulation. It has been reported that a bat in the cold may or may not thermoregulate before it begins the entry into hibernation (Burbank and Young, '34; Reeder and Cowles, '51; Herreid and Schmidt-Nielsen, '66; Hanuš, '59; Koski, '68). I found this to be true in my experiments. Twelve of the 17 normal control bats immediately began to enter hibernation when exposed to the cold. The remaining five normal control animals thermoregulated prior to beginning the entry into hibernation.

1. Animals that did not thermoregulate prior to the entry into hibernation.

Twelve of the 17 normal control animals did not thermoregulate prior to the entry into hibernation. The graphs of the body temperature and heart rate responses of these animals

when they were placed in the cold are presented in figures 9-13. The temperature curves of these animals are characterized by an immediate and steady decline. The temperature decline begins at the moment of initial exposure to the cold and continues steadily until the animal is in hibernation. (An animal is considered to be in hibernation when its body temperature is within three degrees of the refrigerator temperature.). Some of the animals have brief plateaus in their temperature curves at lower levels but there are no plateaus at higher levels.

The heart rate curves for these animals show one of two patterns which will be referred to as patterns A and B. In pattern A the heart rate curve is characterized by plateaus and peaks in its high segment. The peaks and plateaus are followed by sharp declines in the heart rate. This pattern appears in figures 9, 10 and 11. In pattern B the heart rate curve does not show plateaus and peaks in its high segment. Instead the heart rate falls in a linear fashion to the levels characteristic of hibernation. This pattern appears in figures 12 and 13.

2. Animals that thermoregulated prior to the entry into hibernation.

Five of the 17 normal control animals thermoregulated prior to the beginning of the entry into hibernation. The graphs of the body temperature and heart rate responses of these animals when they were placed in the cold are presented in figures 14-17. For these animals it is essential to

distinguish between the thermoregulatory and the entry into hibernation phases of the temperature curves. The beginning and end of the thermoregulatory phase of each temperature curve are indicated by arrows 1 and 2 in figures 14-17. The end of the thermoregulatory phase is signalled by a sharp decline in the heart rate (indicated by an S in figures 14-17). The body temperature and heart rate then progressively fall to hibernation levels.

The duration of the thermoregulatory phase of the temperature curve varies considerably from animal to animal. The range is from 60 to 350 minutes for the normal animals in this study. During thermoregulation the body temperature is not maintained at a constant temperature. Instead, there are small fluctuations which give the entire curve a saw-toothed appearance. The most striking characteristic of this phase of the temperature curve is the mean body temperature which is maintained. The mean body temperature during the thermoregulatory phase calculated for the five animals as a group is 31°C. (The lowest individual mean temperature is 28°C. and the highest is 34°C.). It is also interesting to notice that the lowest point to which the temperature falls prior to or during the thermoregulatory phase is 25°C. The body temperature does not fall below this point without continuing to decline directly to hibernation levels.

The heart rate during the thermoregulatory phase of the temperature curve is usually between 600 and 700 beats per minute. A sudden sharp decline in the heart rate signals the

end of the thermoregulatory phase and the beginning of the entry into hibernation.

In summary, certain characteristics of the temperature and heart rate curves of the normal control animals during the thermoregulatory phase may be enumerated.

(1) The duration of the thermoregulatory phase varies considerably from animal to animal, the range in this study being 60 to 350 minutes.

(2) The mean body temperature maintained during the thermoregulatory phase by all the animals as a group is 31°C.

(3) The body temperature during thermoregulation is not maintained at a constant level but instead has small fluctuations.

(4) The lowest temperature recorded prior to or during the thermoregulatory phase is 25°C.

(5) The heart rate during thermoregulation is usually between 600 and 700 beats per minute.

(6) A sudden sharp decline in the heart rate signals the end of the thermoregulatory phase and the beginning of the entry into hibernation.

II. The Sham Operated Group

There are five animals in this group. It should be recalled that these animals were treated the same as the lesioned animals except that electrolytic lesions were not produced. The temperature and heart rate responses of these animals when they were placed in the cold are presented in figures 18 and 19.

Examination of figures 18 and 19 reveals that the sham operated control animals have temperature curves which are similar to the curves of the normal control animals which did not thermoregulate. The heart rate curves of

four of the sham operated control animals are similar to the pattern A curves described above for the normal control animals. One sham operated control animal (animal 32u, fig. 19) has a heart rate curve corresponding to the B pattern described above.

III. The Lesioned Control Group

There are ten animals in this group. It should be recalled that these animals had electrolytic lesions placed in brain areas other than the hippocampus. (There was minor damage to the hippocampus in some of these animals.) Areas which were damaged in the lesioned control group include the neocortex, piriform lobe, septum, dorsal thalamus, habenular nuclei, caudate nuclei and olfactory bulbs. The graphs of the body temperature and heart rate responses of these animals when they were placed in the cold are presented in figures 20-23. Accompanying the graphs are tracings of brain sections showing the location of the lesions for each animal.

The temperature curves of the lesioned control group are similar to those of the normal control and sham operated control groups. Nine of the ten lesioned control animals did not thermoregulate. The heart rate curves of these nine animals again fall into the two patterns described above for the normal control animals. There was one lesioned control animal which did thermoregulate (animal 23u, fig. 21). This thermoregulation was similar to the thermoregulation of the normal control animals.

IV. The Lesioned Group

There are 20 animals in this group. It should be recalled that to be included in this group an animal had to have sizable damage to the hippocampus. The lesioned group can be separated according to their temperature curves into three subgroups: (1) animals with temperature curves that deviate from the curves of the control animals; (2) animals with temperature curves similar to the curves of the control animals that thermoregulated; and (3) animals with temperature curves similar to the curves of the control animals that did not thermoregulate.

1. Animals with temperature curves that deviate from the curves of the control animals.

Of the 20 animals in the lesioned group, nine have temperature curves that deviate significantly from those of the control animals. The graphs of the body temperature and heart rate responses of these animals when they were placed in the cold are presented in figures 24-30. Accompanying the graphs are tracings of brain sections showing the location of the lesions for each animal.

Since the animals of this subgroup will have their temperature curves compared to the temperature curves of the control animals, some of the characteristics of the latter's curves should be restated here.

(1) The temperature curves of the control animals can be divided into two phases: a thermoregulatory phase during which the body is maintained at a high temperature, and an entry

into hibernation phase during which the body temperature is declining toward the refrigerator temperature.

(2) The majority of the control animals begin the entry into hibernation phase as soon as they are placed in the cold; other animals show a thermoregulatory phase before beginning the entry into hibernation.

(3) There are two heart rate patterns associated with the control animals that did not thermoregulate: these are designated as patterns A and B.

(4) During the entry into hibernation phase of the temperature curve there are no plateaus at high body temperatures but there are plateaus at lower temperatures.

(5) For the group of normal control animals with a thermoregulatory phase in their curves, the mean body temperature maintained during the thermoregulation is 31°C., the lowest individual mean being 28°C. and the highest 34°C.

(6) During the thermoregulatory phase the body temperature is not maintained at a constant level but instead has small fluctuations.

(7) The body temperature prior to or during the thermoregulatory phase of the temperature curve does not fall below 25°C. without the animal continuing to lower its body temperature directly to hibernating levels.

The temperature curve of animal 40 (fig. 24) can be considered to be the prototype of the temperature curves of this entire subgroup. Examination of the graph of the response of this animal to the cold reveals that it differs from the response of the control animals in several ways.

(1) There is no clear demarcation between a thermoregulatory phase and an entry into hibernation phase of the temperature curve. Instead the body temperature falls very slowly toward hibernation levels.

(2) There is a sudden sharp drop in the heart rate at the ten minute recording (indicated by S), which resembles the sharp declines of the pattern A heart rate curves of the control animals.

(3) The body temperature falls below 25°C. after the 135 minute recording without continuing a direct and steady decline into hibernation.

(4) There are plateaus and peaks throughout the entire curve.

(5) About 350 minutes after the beginning of the testing there is a reversal of the declining temperature and the temperature returns to 29°C., the typical thermoregulatory level. This thermoregulatory level is not maintained. Instead a second temperature decline is immediately innitiated.

It is interesting to notice that the body temperature had fallen from 29°C. to 15°C. in 285 minutes prior to the temperature reversal. The same temperature range (29° to 15°C.) is spanned in only 40 minutes during the second temperature decline after the reversal. This second temperature decline closely resembles the entry into hibernation phase of the temperature curves of the control animals. However, once a body temperature of 15°C. has been reached, the lesioned animal again seems to experience difficulty in entering hibernation. The time it takes this animal to lower its temperature from 15°C. to hibernation levels is longer than normal.

An examination of the brain section tracings accompanying the graph of this animal's response reveals that the lesions involve the dorsal hippocampus bilaterally, causing particular damage to the dentate gyrus on both sides. The

dorsal thalamus is also slightly damaged.

The response of animal 38u (fig. 25) to the cold also deviates considerably from the control pattern. Upon exposure to the cold this animal's body temperature falls precipitously to a level below 25°C. However, unlike the control animals, this animal does not continue the temperature decline but maintains a mean body temperature of 22°C. for 115 minutes. Following this long temperature plateau, the temperature curve resembles the normal entry into hibernation. The heart rate of this animal is lower than the rate observed for the control animals during the thermoregulatory phase.

The lesion in this animal again is bilateral and in the dorsal hippocampus. The dentate gyrus is particularly involved. The dorsal thalamus is also damaged.

Animal 43 (fig. 26) is comparable to animal 38u just described. The temperature curve ischaracterized by large fluctuations about a mean temperature which is below 25°C. This mean temperature is 22°C. and is maintained for 155 minutes. Following the plateau the temperature curve appears to have a normal entry into hibernation phase.

The lesion in this animal involves the dorsal hippocampus on both sides. There is some damage to the dorsal thalamus.

The temperature curve of animal 9u (fig. 27) has thermoregulatory and entry into hibernation phases similar

to those of the control animals. However, the temperature is allowed to fall to 20°C. before the thermoregulatory phase is initiated. As was stated above, the control animals never let their body temperatures fall below 25°C. without continuing the decline into hibernation.

The lesion in this animal involves the dorsal hippocampus bilaterally, but with the major involvement on one side only. There is again some damage to the dorsal thalamus.

The temperature curve of animal 14 (fig. 28) is distinctly different from the control curves in the dramatic reversal which appears in the falling body temperature and heart rate about 50 minutes after exposure to the cold. The initial 50 minutes of this animal's response seems to reflect a normal entry into hibernation. However, after this time the process is reversed and the animal returns its body temperature and heart rate to thermoregulatory levels. Another decline in the temperature and heart rate then occurs almost immediately which leads this time to hibernation.

This animal's lesions are in the dorsal hippocampus. The involvement is bilateral.

Animal 24u (fig. 29) also has a reversal in its heart rate and body temperature curves after an apparently normal entry into hibernation. This reversal is not as dramatic as that of the preceeding animal. The heart rate and temperature do not return to thermoregulatory levels.

The lesion in this animal involves the dorsal hippocampus on one side only. There is also some damage to the dorsal thalamus.

Figure 30 contains the graphs of three lesioned animals whose temperature curves deviate from those of the control animals less than the curves of the lesioned animals just described. All three animals have plateaus and peaks in their temperature curves at high levels. The duration of these plateaus and peaks is brief when compared to the thermoregulatory phases of the normal animals. The maximum duration of the plateaus and peaks is 30 minutes (animal 11u). Also, animals 23 and 11u allow their temperatures to decline below 25°C. without immediately continuing the temperature descent to hibernation levels. Following the initial plateaus and peaks, the temperature curves of these animals are similar to the entry into hibernation phase of the curves of the control animals.

The lesions in these animals may be seen on the brain section tracings accompanying figure 30.

2. Animals with temperature curves similar to the curves of the control animals that thermoregulated.

Four of the 20 lesioned animals are in this subgroup. The graphs of the body temperature and heart rate responses of these animals when they were placed in the cold are presented in figures 31-33. Accompanying the graphs are tracings of brain sections showing the location of the lesions for each animal.

The temperature curves of these animals are in general similar to those of the control animals that thermoregulated.

However, there are some deviations of the temperature curves of animals 44 and 41 (fig. 32). Animals 41 and 44 maintain the thermoregulatory phases of their temperature curves near the minimum time observed in the control animals, 30 and 40 minutes. Animal 41 has a mean temperature which is low for thermoregulation (26°C.).

The brain tracings accompanying the graphs of these animals show involvement of the dorsal hippocampus. There is also involvement of the dorsal thalamus.

3. Animals with temperature curves similar to the curves of the control animals that did not thermoregulate.

Seven of the 20 lesioned animals are in this subgroup. The graphs of the body temperature and heart rate responses of these animals when they were placed in the cold are presented in figures 34-36. Accompanying the graphs are tracings of brain sections showing the location of the lesions for each animal.

The temperature curves of these animals are similar to the temperature curves of the control animals that did not thermoregulate. All the heart rate curves are similar to the B pattern described above for the control animals. The heart rate curves fall in a linear fashion to the levels characteristic of hibernation. There are no plateaus or peaks in their high segments.

The tracings of the brain sections which accompany the graphs of these animals show involvement of the dorsal hippocampus in each case.

V. Summary of the Data.

Table I on the following page summarizes the data.

ANIMALS	BODY TEMPERATURE CURVES	No.	HEART RATE CURVES	No.
Fed Control	No Thermoregulation	5 (56%)	Pattern A	5 (56%)
			Pattern B	0 (0%)
N=9	Thermoregulation	4 (44%)	Thermoregulation	4 (44%)
Fed Lesioned	Disrupted Curves	3 (37%)	?	3 (37%)
	Immediate Entry Into Hibernation	1 (13%)	Pattern B	1 (13%)
N=8	Normal Thermoregulation	4 (50%)	Normal Thermoregulation	4 (50%)
Unfed Control	No Thermoregulation	21 (91%)	Pattern A	9 (39%)
			Pattern B	12 (52%)
N=23	Thermoregulation	2 (9%)	Thermoregulation	2 (9%)
Unfed Lesioned	Disrupted Curves	6 (50%)	?	6 (50%)
	Immediate Entry Into Hibernation	6 (50%)	Pattern B	6 (50%)
N=12	Normal Thermoregulation	0 (0%)	Normal Thermoregulation	0 (0%)

Table I. A summary of the data.

DISCUSSION

The bats of the temperate climates are faced with a life-threatening dilemma with the onset of the cold winter months. They have a large surface area compared to their small body mass. Therefore it is metabolically expensive for these animals to maintain a high body temperature in a cold environment. This problem is coupled with a very poor winter food supply. Hibernation is an effective method of solving this dilemma, for the low metabolic rate and associated decrease in energy expenditure during hibernation allow the animals to survive during the cold season.

Hanuš ('59) measured the oxygen consumption of four species of bats during thermoregulation and while in hibernation. Animals with body temperatures of 35°C. in a 10°C. environment consumed 11.5 ml O_2/gm/hr. In contrast, the oxygen consumption at a hibernating body temperature of 10.3°C. in the same environment was 0.1-0.2 ml O_2/gm/hr. Herreid and Schmidt-Nielsen ('66) have calculated that a bat which maintains a high body temperature in a 10°C. environment could live on one gram of fat only one day, whereas a hibernating animal could live on the same quantity of fat over 80 days.

Hibernation has one main disadvantage. Because all the life processes are slowed, an animal in hibernation can not immediately react to external stimuli in a coordinated manner. A number of species of bats must have a minimum body temperature of about 30°C. in order to be able to fly (Burbank and Young, '34; Reeder and Cowles, '51; Morrison, '59).

It takes the little brown bat 30 to 40 minutes to warm its body to 30°C. from a temperature of 3°C. (Menaker, '62). It is interesting to notice that in this study the mean body temperature for all the normal control bats during thermoregulation was 31°C. This is probably near the minimum temperature necessary for flight in this species. Thus thermoregulation in the normal bat seems to function to keep the animal in an active state.

The entry into hibernation may be thought of as a transitional stage between the more stable states of thermoregulation and hibernation. Once the entry into hibernation begins in a normal animal it proceeds quickly and directly to the state of hibernation. In the experiments reported here the entry phase in all the control animals was complete within 60 to 100 minutes after it had begun. Hanuš ('59) and Herreid and Schmidt-Nielsen ('66) have also emphasized the transient nature of the entry into hibernation in other species of bats. They did not observe any animals which maintained body temperatures intermediate between thermoregulatory and hibernation levels. The rapid, steady decline of the body temperature is the most efficient method of entering hibernation for a small animal with a large surface area such as the bat, for during this time the animal can neither fly nor is he functioning at the level of the lowest possible energy expenditure.

In the experiments reported here, lesions of the dorsal hippocampus were often found to disrupt the normal transit

nature of the entry into hibernation of the bat. The lesioned animals presented in figures 24-30 have body temperature curves which differ markedly from those of the control animals. The general impression obtained from a comparison of the graphs of these lesioned animals with the graphs of the control animals (figs. 9-23) is that the lesioned animals have difficulty entering hibernation.

Of all the lesioned animals, animal 40 (fig. 24) shows the most severe disruption of the entry into hibernation. In this animal it is difficult to distinguish between a thermoregulatory phase and an entry into hibernation phase of the temperature curve. Upon exposure to the cold the animal's body temperature immediately begins to fall very slowly. Upon falling below 25°C., the temperature which in the control animals always indicates that a rapid entry into hibernation will follow, the temperature of animal 40 continues its very slow decline. Furthermore, about 350 minutes after the beginning of testing, there is a reversal of the falling temperature. The body temperature returns to 29°C., the typical thermoregulatory level, then falls again, this time to hibernating levels. The second temperature decline resembles a normal entry into hibernation.

The temperature curve of animal 38u (fig. 25) also reflects a disruption of the entry into hibernation. Upon exposure to the cold this animal immediately begins to lower his body temperature in what appears to be an attempt to enter hibernation directly. The body temperature drops below

25°C., but, instead of continuing its fall, at about 22°C. levels off in a plateau. During this plateau the animal maintains a mean body temperature of 22°C. for 115 minutes. After the plateau the animal appears to enter hibernation normally.

Animal 43 (fig. 26) is very much like the two animals just described. The body temperature falls below 25°C. but there is no rapid progression into hibernation thereafter. Instead there are a series of large fluctuations about a mean temperature of 22°C. The temperature is maintained at a mean of 22°C. for 155 minutes before an apparently normal entry into hibernation occurs.

These three animals showed the most dramatic effects of the hippocampal lesions on the entry into hibernation. They will therefore serve as the reference animals for the following discussion. Afterwards the responses of the other lesioned animals will be analyzed.

It was stated above that the "general impression" obtained from examining the graphs of the animals with dorsal hippocampal lesions is that these animals have difficulty entering hibernation. It is possible to consider that, on the contrary, these animals have difficulty thermoregulating. The question is still open as to which phase of the temperature curve is disrupted by the lesions. Do the lesions disrupt thermoregulation or the entry into hibernation? Are the lesioned animals attempting to hibernate or thermoregulate?

An experiment by Strumwasser ('59) strongly supports

the idea that the dorsal hippocampal lesions disrupt the entry into hibernation. Strumwasser recorded the brain temperature and shivering activity of ground squirrels during the entry into hibernation. At the same time he also recorded the electrical activity of many areas of the forebrain and brain stem. Strumwasser observed that a unique motor cortex-amygdaloid-dorsal hippocampal activity pattern appears at the beginning of and throughout the entry into hibernation of the ground squirrel. The activity pattern is uniform and centered around shivering. The motor cortex activity appears just before and during shivering, while the amygdaloid-hippocampal activity appears just after the shivering and cortical activity have ceased. Strumwasser suggests that the activity in the amygdala and hippocampus reflects an inhibitory influence which these two structures may be exerting on the motor cortex. Such inhibition would serve to "shut off" the animal's shivering. Strumwasser supports this suggestion by showing that the amygdaloid-hippocampal pattern is associated with a decline in the brain temperature of the ground squirrel. Thus the amygdala and hippocampus may well be part of the neural machinery which is involved in an animal's temperature decline during the entry into hibernation. The damage to the hippocampus produced in the experiments reported here would thus have the effect of disrupting the normal pattern of entry into hibernation rather than thermoregulation. The disruption would be in the form of delaying the entry into hibernation.

There is also a clue in the data reported here which indicates that the animals with dorsal hippocampal lesions may have been attempting to enter hibernation rather than thermoregulate (refer to table I). Considering the fed animals only, five of the nine (56%) control animals did not thermoregulate but began the entry into hibernation as soon as they were placed in the cold. On the other hand, only one of the eight (13%) fed animals with hippocampal lesions entered hibernation right away. Considering the unfed animals, 21 of the 23 (91%) control animals entered hibernation immediately while six of 12 (50%) of the animals with hippocampal lesions entered hibernation right away. It seems, then, that the tendency of the majority of the control animals is to enter hibernation as soon as they are exposed to the cold. Assuming that the lesioned group was drawn from a normal population of animals, it is reasonable to think that the tendency of a greater number of these animals would be to immediately begin entry into hibernation when exposed to the cold. The irregular temperature curves in some of these animals would seem to reflect a disruption in the entry into hibernation and not a disruption in thermoregulation.

The examination of the heart rate curves gives yet another indication that the animals with hippocampal lesions attempt to enter hibernation as soon as they are placed in the cold. Investigators have noted that when animals such as the California ground squirrel, thirteen-lined ground squirrel and woodchuck begin the entry into hibernation, the heart

rate falls precipitously before the body temperature begins to decline (Strumwasser, '59; Lyman and O'Brien, '63; Lyman, '58). The striking decline in heart rate which signals the beginning of the entry into hibernation in the bat has been pointed out in this study (see page 20 and fig. 14). In the animals with dorsal hippocampal lesions the heart rate curves occasionally give an indication that some process has been initiated which should have led to hibernation but did not. An examination of the heart rate curve for animal 40 (fig. 24) reveals that after ten minutes in the cold there is a sharp decline in the heart rate (indicated by an S on the graph). The body temperature does not decline in turn, as usually happens in the control animals. The rate then rises back to a high level. This sharp decline in the heart rate suggests an early attempt on the part of the animal to enter hibernation, which is not successful. Animal 38u (fig. 25) has several sharp declines in heart rate which could indicate attempts to enter hibernation (indicated by Ss on the graph). The body temperature, in each instance, remains at a high level and the heart rate is again accelerated to a high level. Animal 43 (fig. 26) does not show any conspicuous depressions in the heart rate curve that are not followed by declines in the body temperature.

The question asked above was this: Do the dorsal hippocampal lesions disrupt thermoregulation or the entry into hibernation? The evidence points to the latter interpretation:

that the dorsal hippocampal lesions interfere with the mechanisms which influence the normal entry into hibernation.

The disrupted temperature curves of three lesioned animals were used as the basis for the above discussion. Six other animals with dorsal hippocampal lesions also had disrupted body temperature curves. These animals will be considered next.

Segments of the temperature curve of animal 9u (fig. 27) closely resemble the thermoregulatory and entry into hibernation phases of the curves of the control animals. The animal appears to have no difficulty in maintaining the body temperature and heart rate at thermoregulatory levels. However, when first placed in the cold the animal's temperature falls considerably below 25°C., the temperature which in the control animals always leads to an immediate entry into hibernation. This initial decline in the temperature is interpreted as an attempt to enter hibernation directly which is not carried through to completion. Instead the body temperature and heart rate return to thermoregulatory levels. An apparent normal entry into hibernation follows the thermoregulatory period. There are two sharp declines in the heart rate curve which seem to indicate unsuccessful attempts to enter hibernation. These are indicated by Ss on the graph (fig. 27).

Animal 14 (fig. 28) begins to enter hibernation in the normal manner when exposed to the cold. However, before the body temperature has reached hibernation levels a reversal

in the declining temperature occurs. The body temperature and heart rate return to the thermoregulatory level, then another entry into hibernation is initiated almost immediately. This second entry seems to proceed normally.

Animal 24u (fig. 29) is very much like animal 14 just described. Upon exposure to the cold, animal 24u appears to enter hibernation in the normal manner. However, the animal has difficulty completing the final stages of the entry into hibernation. A reversal of the falling temperature occurs about 140 minutes after the beginning of the test period. This reversal raises the body temperature from 9°C. to 16°C. Following this reversal the temperature falls to hibernation levels.

Animals 38, 23 and 11u (fig. 30) have less severe disruptions of the entry into hibernation than the above animals. These animals have plateaus and peaks in the upper segments of their temperature curves. Following these disruptions, the temperature curves seem to reflect a normal entry into hibernation.

In this study 20 animals with sizable dorsal hippocampal damage were tested. In nine of the 20 animals the lesions produced a disruption of the normal entry into hibernation. The nine animals showing such a disruption of the entry into hibernation have been discussed above. Four of the 20 animals with dorsal hippocampal lesions have body temperature curves like the curves of the control animals which thermoregulated.

A close examination of these animals may provide insight into the nature of the hippocampal influence on the entry into hibernation.

The four animals with dorsal hippocampal lesions which displayed apparently normal thermoregulation and entry into hibernation are animal 21u (fig. 31), animals 44 and 41 (fig. 32) and animal 39u (fig. 33). The initial response of these animals to the cold is an increase in heart rate and a slight fall in the body temperature. The falling temperature is checked and the body temperature is maintained at a high level above 25°C. until the initiation of the entry into hibernation. A normal entry into hibernation follows the thermoregulatory phase.

The damage to the dorsal hippocampus in the four animals under consideration here is neither more nor less extensive than the damage to this structure in the previously discussed lesioned animals. Yet the latter animals have disrupted entry into hibernation curves, while the former do not. An explanation of the normal entry into hibernation of these four animals can be suggested on the basis of a characteristic of the temperature curves of the lesioned animals with the disrupted entry into hibernation.

All the animals with disrupted temperature curves have a segment of their curve which resembles the normal entry into hibernation. For example, animal 40 (fig. 24), when placed in the cold, has a body temperature which falls very slowly toward hibernation levels. About 350 minutes after

the beginning of the test period there is a reversal of the declining temperature and the temperature returns to 29°C., the typical thermoregulatory level. This thermoregulatory level is not maintained. Instead a second temperature decline is immediately initiated. The body temperature had fallen from 29°C. to 15°C. in 285 minutes prior to the temperature reversal. The same temperature range (29°C. to 15°C.) is spanned in only 40 minutes during the second temperature decline. This segment of the temperature curve of animal 40 closely resembles the normal entry into hibernation.

Animal 9u (fig. 27) is another example of a lesioned animal in which a segment of the temperature curve reflects a normal entry into hibernation. Animal 9u initially lowers his temperature to 20°C. This temperature is one which always commits the control animals to continue the temperature decline directly to hibernation levels. However, the temperature of animal 9u does not continue to decline but instead is raised to thermoregulatory levels for a period. Following the thermoregulation period, the temperature is lowered to hibernation levels in the normal manner.

An examination of the graphs of the rest of the animals with disrupted temperature curves reveals some segment of their curves which resembles a normal entry into hibernation (figs. 25,26,28,29 and 30). This segment always follows an earlier apparent attempt to enter hibernation which was not successfully carried through to completion.

This constant appearance of an essentially normal segment in the curves suggests that the animals are making adjustments during the abnormal phase of the temperature curve, adjustments which allow the animals to enter hibernation in the normal manner in spite of the hippocampal damage. The temperature curve of animal 14 (fig. 28) seems to strongly support this suggestion.

The initial 50 minutes of the temperature curve of animal 14 (fig. 28) appears to reflect a normal entry into hibernation. However, after this time the process is reversed and the animal returns its body temperature and heart rate to thermoregulatory levels. Another decline in the body temperature and heart rate almost immediately occurs which leads to hibernation. The interesting aspect of this animal's graph is that an enormous amount of energy is exerted in raising the temperature to thermoregulatory levels, yet the temperature is immediately returned to hibernation levels. The animal seems to be making adjustments during the abnormal temperature rise so that the temperature can be returned immediately to hibernation levels. It is unlikely that the temperature rise represents a normal return to the alert warm-blooded condition because the alert state is not maintained for any length of time.

In returning to the temperature curves of the lesioned animals of figures 31-33 with the normal thermoregulation and entry into hibernation phases, the period prior to the normal

entry into hibernation must be considered. These animals, like the lesioned animals of figures 24-30, are able to enter hibernation normally after a period of time. It is suggested that during the time of thermoregulation the intact neural centers involved in the entry into hibernation adjust their level of activity so that a normal entry into hibernation can occur.

It is interesting that the four lesioned animals which displayed an apparently normal thermoregulation and entry into hibernation were fed animals (refer to table I). Eight lesioned animals were fed prior to testing and four of them (50%) thermoregulated. Nine control animals were fed and four of them (44%) thermoregulated. It is probable that the animals with hippocampal lesions that thermoregulated and entered hibernation normally are that percentage of a normal population of fed bats which will respond to the cold by thermoregulating. In the animals which thermoregulate the dorsal hippocampal lesions have no apparent effect on the entry into hibernation.

In summarizing the discussion of the lesioned animals thus far, the following statements may be made:

(1) dorsal hippocampal lesions in a number of animals (N=9 of 20) disrupt the temperature curves as compared to the curves of the control animals;

(2) this disruption is in the form of a delay of the entry into hibernation;

(3) the disruption appears to occur only when an animal immediately attempts to enter hibernation upon exposure to the cold;

(4) there are some animals (N=4 of 20) with dorsal hippocampal lesions which have temperature curves that closely resemble the curves of the control animals which thermoregulated;

(5) the lesions of the dorsal hippocampus have no effect on the temperature curves of these animals;

(6) it is suggested that during the thermoregulation in these animals, adjustments are being made which will allow the animals to enter hibernation normally after a period of time; and

(7) these lesioned animals are probably that portion of a given population of fed bats which thermoregulate on exposure to the cold.

The remaining seven animals of the lesioned group (figs. 34,35 and 36) have temperature curves which resemble the curves of the normal animals that immediately entered hibernation upon exposure to the cold. These lesioned animals display no tendency to thermoregulate, and the entry into hibernation appears normal. The heart rate curves of all these animals resemble the B pattern which was described for the control groups. The B pattern heart rate curve is characterized by an immediate and rapid decline, with no peaks or plateaus in the high segment of the curve.

There are two possible explanations for the failure of the dorsal hippocampal lesions to disrupt the entry into hibernation in these seven animals. The first explanation is that the animals could be entering a state of hypothermia

when exposed to the cold. They may be in such an exhausted physiological condition that they are unable to make even a token attempt to maintain a high body temperature. The heart rate curves of the control animals of figures 12,13, 19 (animal 32u), 22 (animal 16) and 23 and lesioned animals of figures 35 (animal 17u and 35u) and 36 (animal 14u) in particular seem to reflect an exhausted condition. These animals on exposure to the cold have very low heart rates of around 200 beats per minute or less. The heart rates drop directly to hibernation levels by a series of short declines. If the lesioned animals under consideration here are entering a state of hypothermia, then the hippocampal lesions would have no effect, for the animals are incapable of producing heat.

The second explanation is that rather than hypothermia, these animals are entering a "hibernation of desperation." Some of the pattern B heart rate curves of both the control and lesioned animals do not seem to reflect an exhausted physiological condition. The heart rate curves of these control and lesioned animals can be seen in figures 22 (animals 19u and 32), 34,35 (animal 37) and 36 (animals 37u and 15u). These animals, although displaying pattern B curves, initially have heart rates of 400 beats per minute or more. Their heart rate curves are characterized by sharp declines in their high segments. These sharp declines in heart rate suggest an active inhibition of the heart rather than a slowing of the heart by chilling. Thus an actual

entry into hibernation rather than hypothermia is indicated. Thus, in this interpretation the heart (and heat production processes) are considered to be inhibited by an active process. This response would then represent the ultimate in conservation of energy. This extreme reaction to the cold may be a reflection of an animals nutrient state.

A number of investigators have noted a relationship between food and hibernation. Obesity, hoarding and starving in different hibernators can significantly influence entry into hibernation (Pearson, '47; Lyman, '54; Bartholomew and Cade, '57; Stones and Wiebers, '66; Mrosovsky, '68). Bartholomew and Cade ('57) observed that the little pocket mouse enters the hibernating state whenever denied food. Stones and Wiebers ('66) reported that the little brown bat will thermoregulate for long periods of time if well fed.

The fed animals of this study were almost evenly divided between the control groups (N=9) and the lesioned group (N=8). Most of the fed animals did not display the pattern B heart rate curve but instead accelerated their heart rates in response to the cold stress (N=16 of 17). An improved nutrient condition does seem to increase an animals readiness to expend energy in response to a cold stress. In contrast, a poor nutrient state, may serve to highly activate the machinery of energy conservation, namely the centers which influence the heart rate and metabolic rate decline of the entry into hibernation. Thus an animal in a poor nutrient condition could be primed to immediately reduce his

metabolic requirements at the first opportunity, namely a low ambient temperature.

The priming of the centers responsible for influencing the temperature decline of the entry into hibernation could account for the absence of disrupted temperature curves in the seven lesioned animals which entered hibernation immediately upon being placed in the cold. The intact centers remaining outside the focus of the lesions could be in such a highly activated state that the absent influence of the destroyed areas is more than compensated for and the animal enters hibernation in the normal manner.

There is one fed member of the lesioned group (fig. 34) which neither thermoregulated nor has a disrupted temperature curve. However, the animal does display a typical pattern B heart rate curve. This was the only one of the fed animals (N=1 of 17) with the pattern B curve. It is possible that prior to feeding this animal was in such a poor nutrient condition that the feeding did not significantly alleviate this condition.

The above explanations as to the reasons why some of the animals with dorsal hippocampal lesions do not display disrupted temperature curves are admittedly tentative. There are probably many factors which influence an animal's response to the cold. It is significant, however, that a similar percentage of both the unfed control and unfed lesioned animals displayed pattern B heart rate curves. It is also interesting that of the 17 fed animals, only one animal has a pattern B

heart rate curve (refer to table I). This latter animal was the only animal of the fed lesioned animals which did not thermoregulate or display a disrupted temperature curve.

The brain tracings accompanying the graphs of the lesioned animals show that in all cases there was sizable damage to the dorsal hippocampus (figs. 24-36). Figure 37 consists of brain tracings on which is shown composite lesions for all the animals in the lesioned group. Figure 38 is a similar diagram showing composite lesions for all the animals in the lesioned control group. A comparison of the brain tracings of figures 37 and 38 reveals that the neural destruction in the lesioned group (fig. 37) as compared to that of the lesioned control group (fig. 38) is concentrated in the dorsal hippocampus. The areas damaged in the lesioned group other than the dorsal hippocampus were also damaged in the lesioned control group. Since the lesions of the lesioned control animals did not produce disrupted temperature curves, areas other than the dorsal hippocampus can be ruled out as factors influencing the entry into hibernation.

The role of the forebrain in temperature regulation exclusive of the preoptic and hypothalamic areas has not been conclusively determined. Bazett and Penfield ('22) and Pinkston et al. ('34) found that the destruction of large areas of the forebrain did not significantly disrupt body temperature regulation in homeotherms. Showers ('59) observed a temporary fall in body temperature following lesions of the cingulate gyrus in the monkey. The hippocampus has not been

implicated as a neural center involved in temperature regulation in homeotherms. However, this study strongly suggests a role for the dorsal hippocampus in the entry into hibernation. This neural center, then, in hibernators must be capable of exerting an influence on the basic temperature regulating mechanisms which are most highly organized at the hypothalamic level.

The pathway by which the dorsal hippocampus influences the temperature decline of the entry into hibernation is unknown. Some of the lesions of the lesioned control animals involved such structures as the fimbria, hippocampal commissure and fornix. These structures constitute a major efferent and afferent pathway of the hippocampus. However, the hippocampus has been shown in other species to have secondary pathways. Votaw ('59) in the monkey has shown that Ammon's horn, when stimulated, elicits somatic movement. This same motor movement can be produced after fornix section but not after separating the hippocampus from the subiculum (Votaw, '59). Blackstad ('58) and Lorente De Nó ('34) among others have described a temporoammonic tract which connects the entorhinal cortex to the dentate gyrus and Ammon's horn. There are also fairly direct connections between the hippocampus and amygdala which pass in both directions (Green and Adey, '56). These hippocampal connections have been described in mammals other than the bat. The hippocampal connections of the bat have not been extensively investigated, but connections similar to those enumerated above are probably present in this species.

An unexpected finding is that the animals with the most disrupted temperature curves have fairly specific bilateral involvement of the dentate gyrus (figs. 24,25 and 26). It is interesting that the dentate gyrus is intimately related to the development of the olfactory system while Ammon's horn is not (Breathnach and Golby, '54; Humphrey, '66). A functional difference between the dentate gyrus and Ammon's horn has not been reported. Whether the dentate gyrus is more directly involved in the entry into hibernation than Ammon's horn is an interesting problem for future investigation.

The mechanism by which the hippocampus influences the temperature decline of the entry into hibernation can not be ascertained from this study. The hippocampus must either directly or indirectly influence the sensitivity of the temperature regulating centers of the hypothalamus. An indirect influence would involve modifying the activity of an area implicated in setting the sensitivity of the physiological thermostat. This area which determines the setting of the physiological thermostat may be the reticular activating system of the brain stem.

Euler and Söderburg ('57) have suggested, based on experimental evidence, that the level of activity of the reticular activating system determines the setting of the physiological thermostat. The general activity of the reticular activating system is in turn dependent on the magnitude of the input to this system from various peripheral sources. This mechanism

of setting the thermostat would take into consideration the homeostasis of the body as a whole in determining the level of body temperature regulation. Heat loss processes have been shown to be associated with diminished activity in the reticular activating system while heat production processes are associated with increased activity (Euler and Söderburg, '57). The role of the dorsal hippocampus and possibly other centers in the entry into hibernation may be to diminish the activity in the reticular activating system. This diminished activity would result in a lowering of the setting of the physiological thermostat and thus a regulated decline in the body temperature to hibernation levels.

The mechanism by which the dorsal hippocampus influences the entry into hibernation has yet to be demonstrated. Future investigations could include studies of such areas as the hippocampus, entorhinal area, amygdala, motor cortex and brain stem reticular formation. In this study a rather limited extent of the dorsal hippocampus was damaged. A method of producing more extensive damage to the dorsal hippocampus should be devised in order to see if greater disruption of the temperature curves can be attained. Also, the entorhinal area should be investigated as a possible relay center for hippocampal impulses which influence the entry into hibernation. Strumwasser ('59) reported a unique EEG activity pattern in the dorsal hippocampus, basolateral amygdala and lateral motor cortex during the entry into hibernation of the ground squirrel. The basolateral

amygdala and lateral motor cortex are additional areas which could be investigated for a possible role in the entry into hibernation. Finally, the role of the brain stem reticular formation in temperature regulation in general and in the entry into hibernation specifically could be a fruitful avenue of research.

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ILLUSTRATIONS

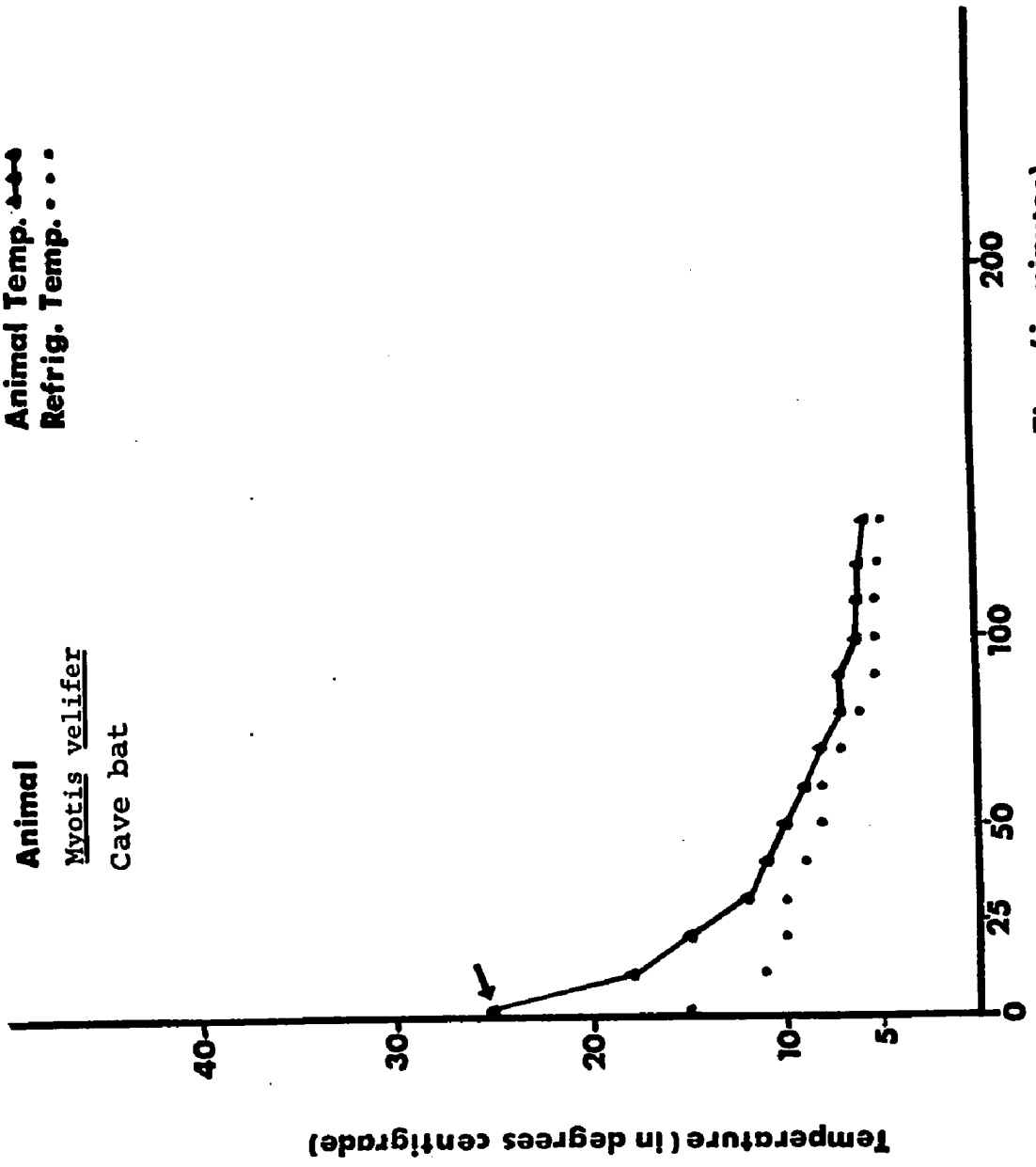


Fig. 1 (Adapted from Reeder and Cowles, '51) The arrow indicates the beginning of the entry into hiberation.

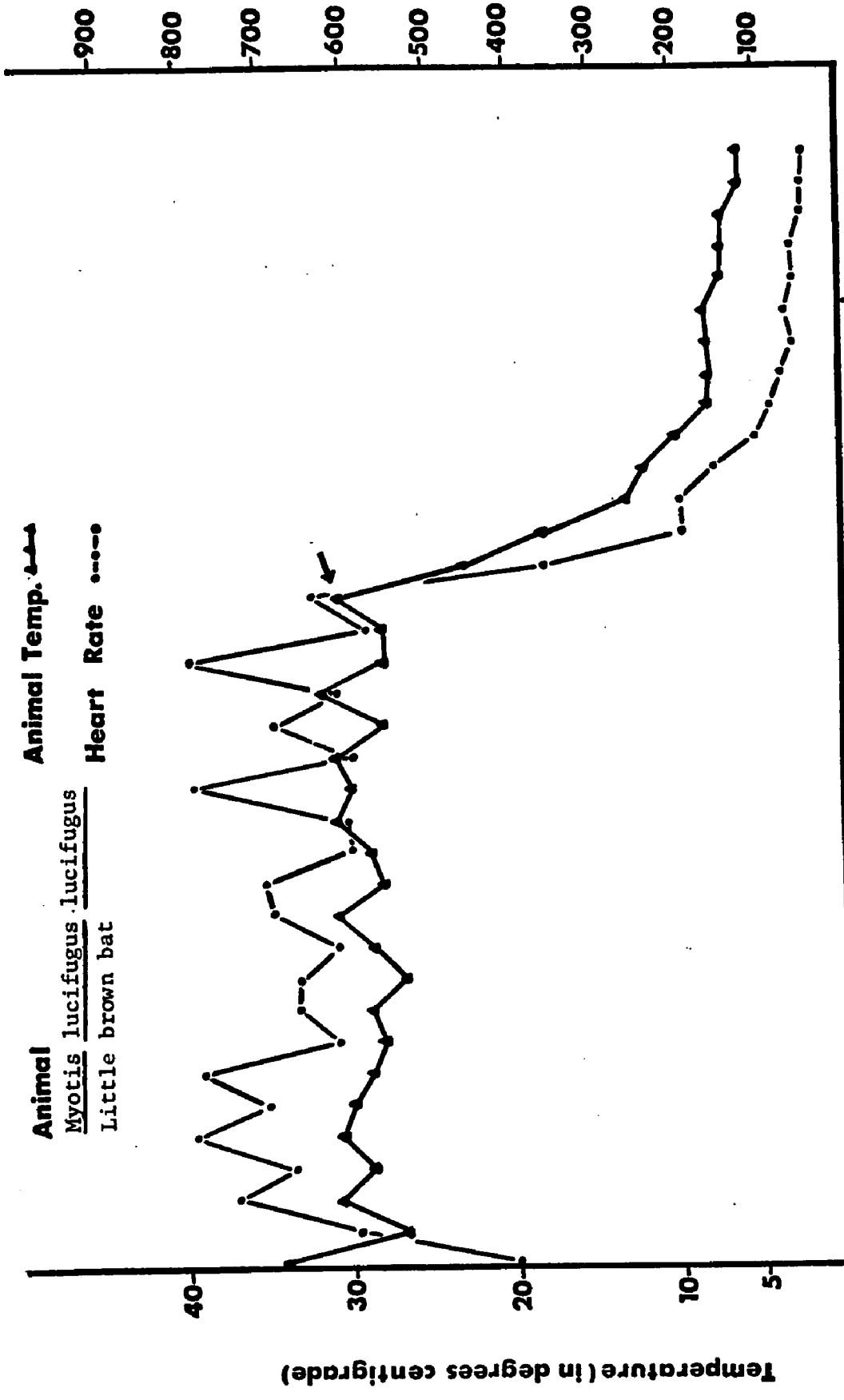


Fig. 2 (Adapted from Koski, '68) The arrow indicates the beginning of the entry into hibernation.

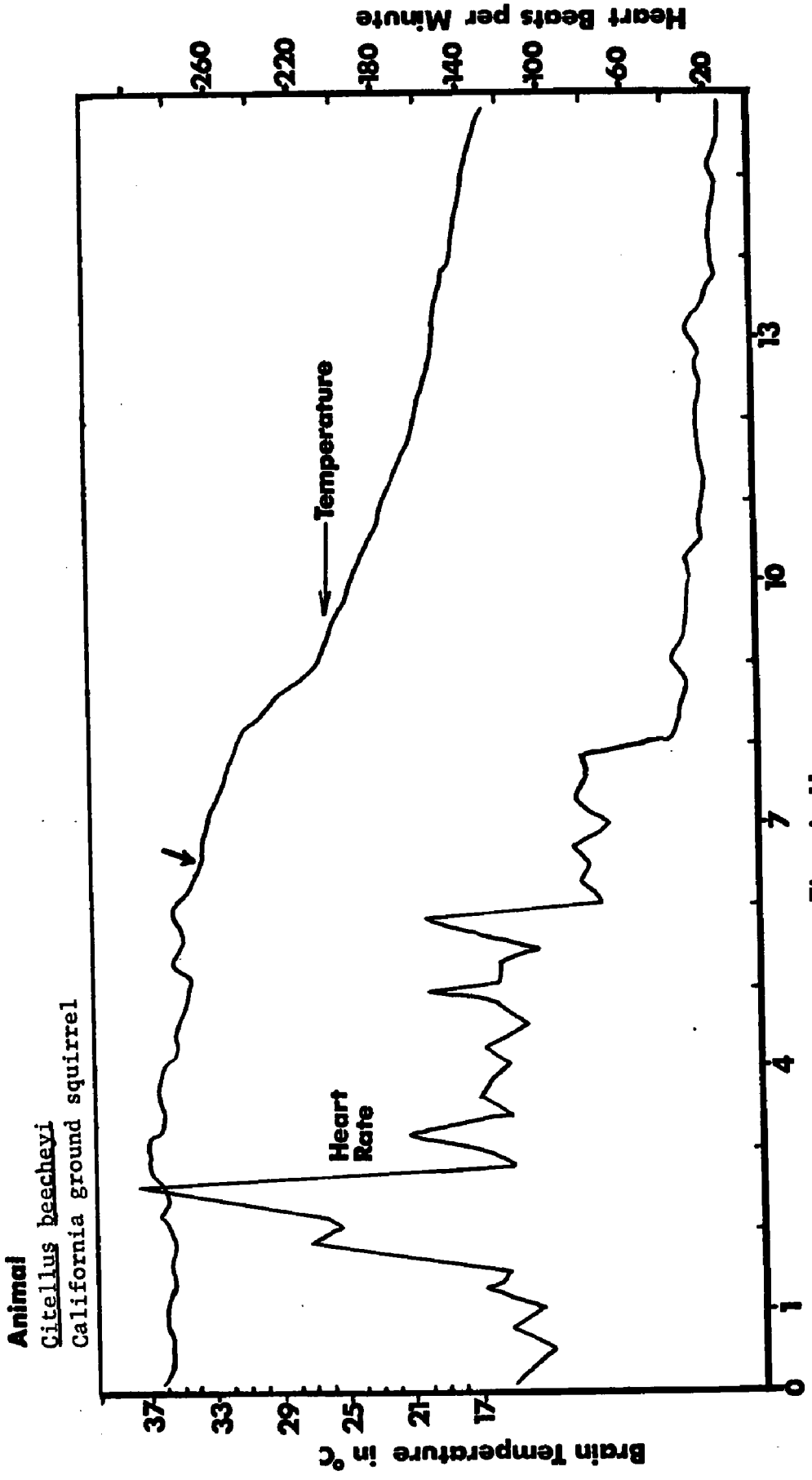


Fig.3 (Adapted from Strumwasser, '59) The arrow indicates the beginning of the entry into hibernation.

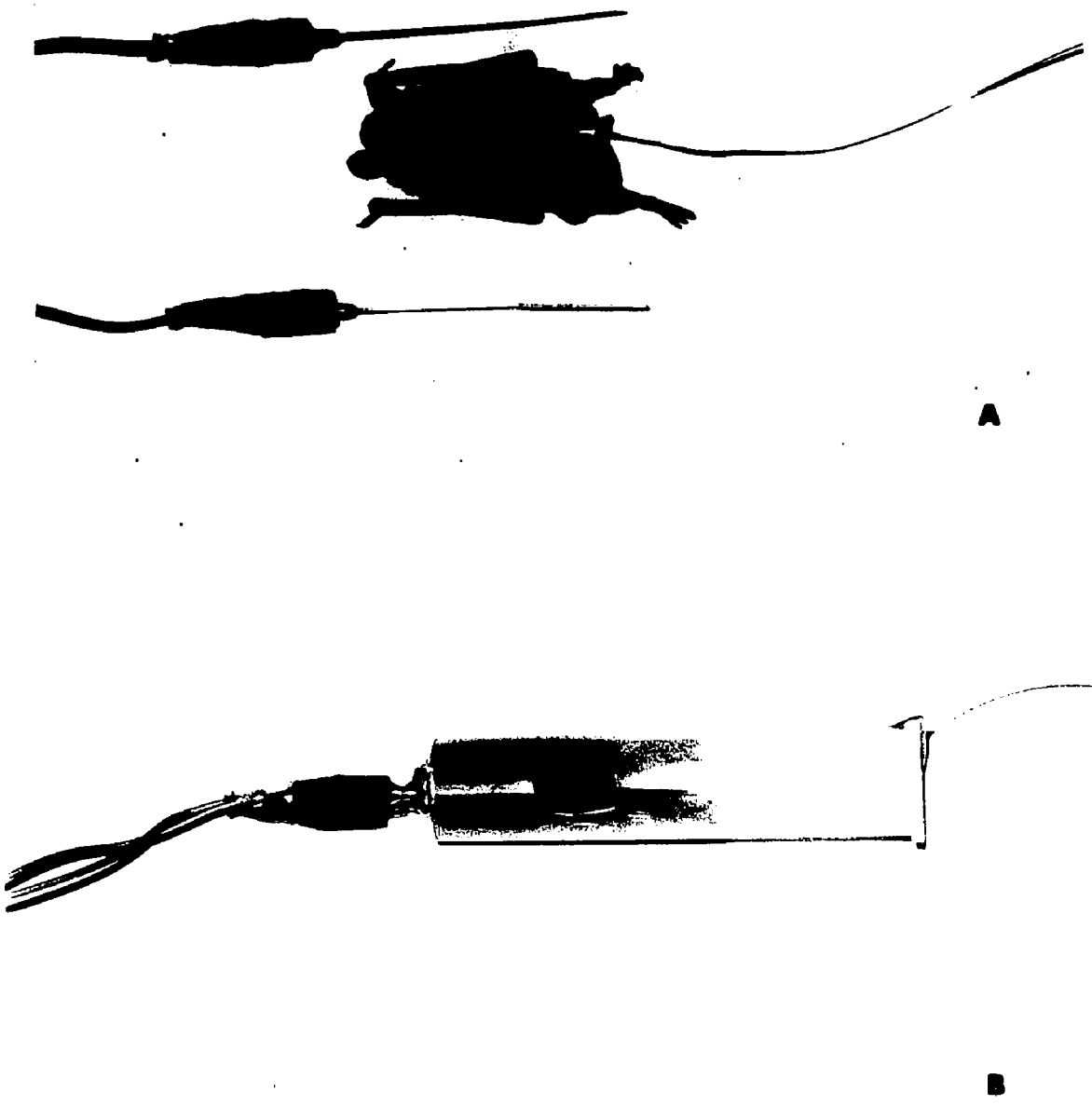


Fig. 4. A. Photograph shows the polyethylene tube with the thermocouple inserted and EKG electrodes on either side of the animal.
B. Photograph shows the animal in the syringe tube and an EKG electrode against a wing.

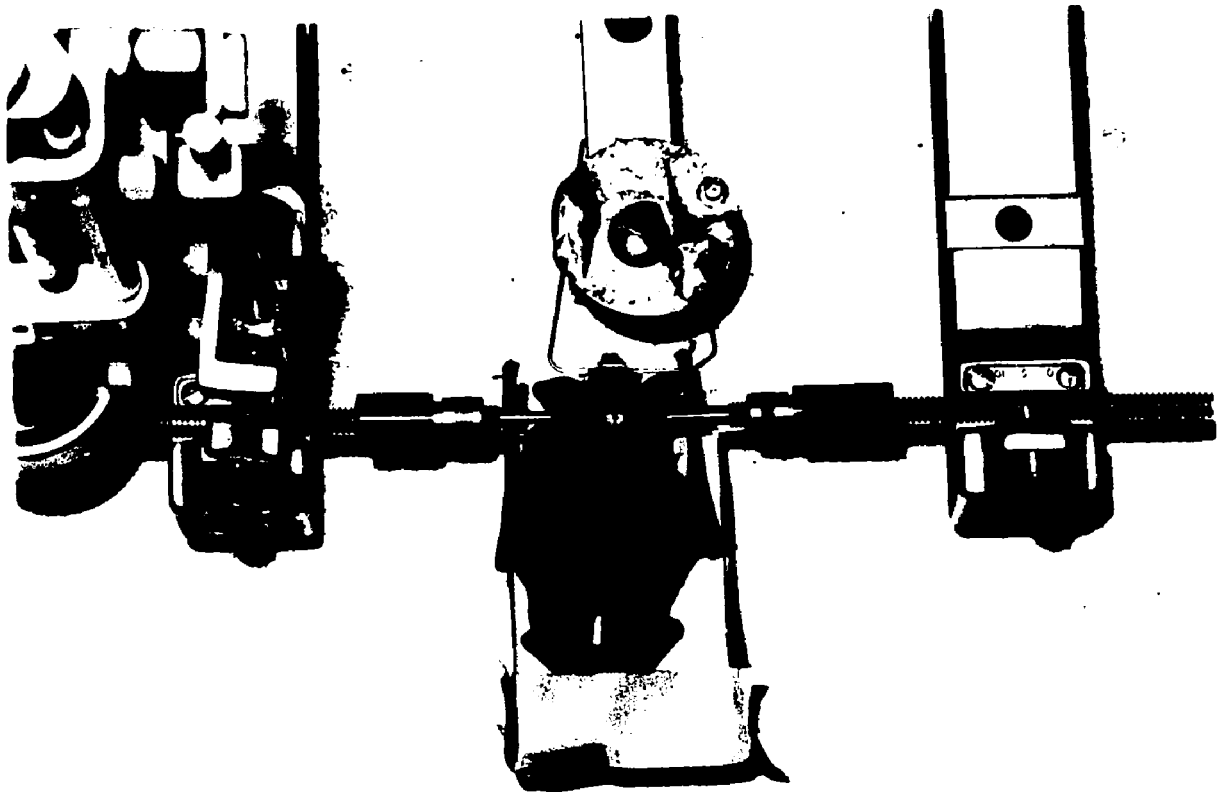


Fig. 5. Photograph shows an animal with skull exposed in the modified stereotaxic instrument.

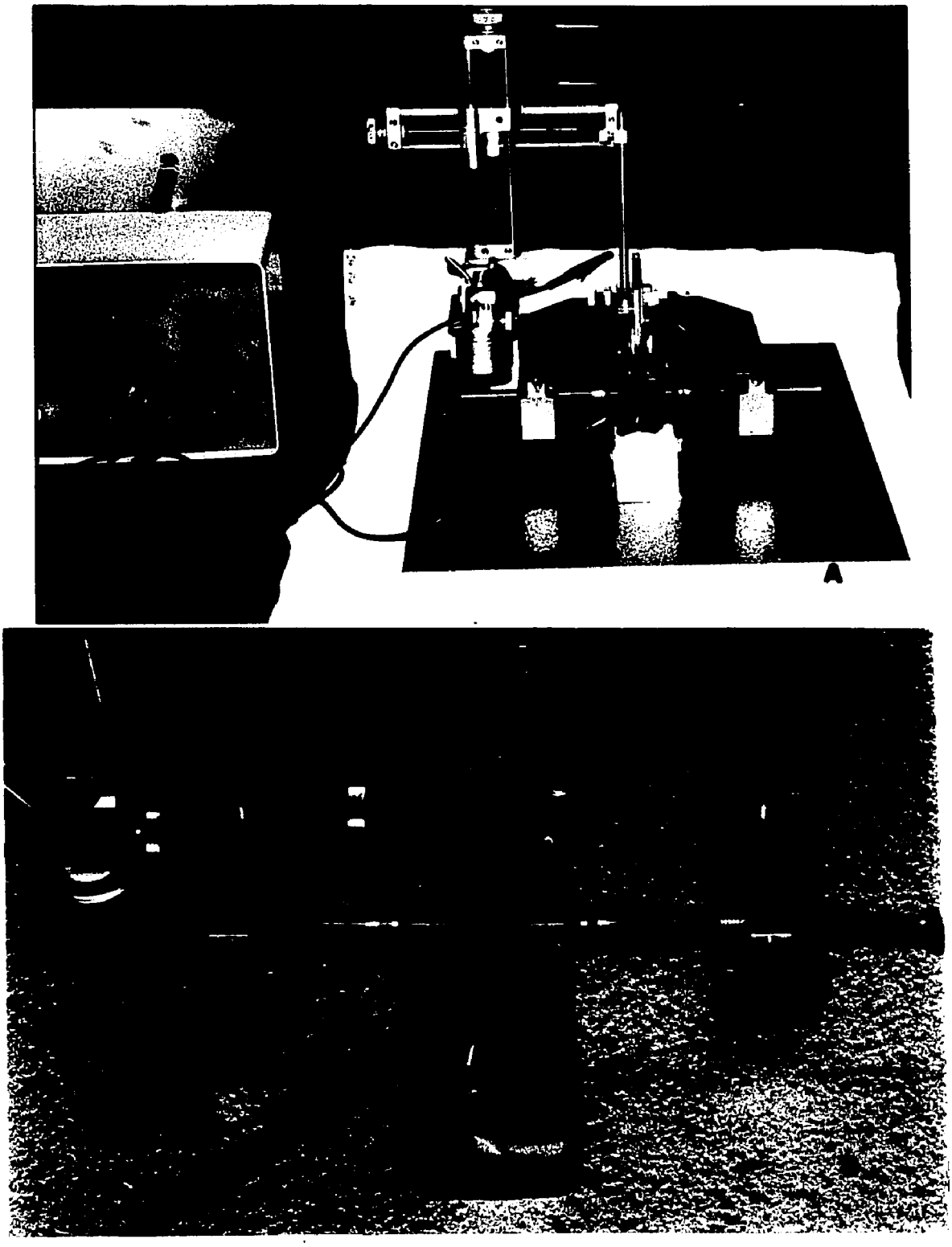


Fig. 6. A. Photograph shows the entire lesion set-up.
B. Photograph shows a close-up of the lesion set-up. The electode can be seen passing through a small hole in the skull.



Fig. 7. Photograph is a view looking up at an animal in the syringe tube which is suspended from a plastic box. The EKG electrodes are in place and the bat's face can be seen through the circular air hole.

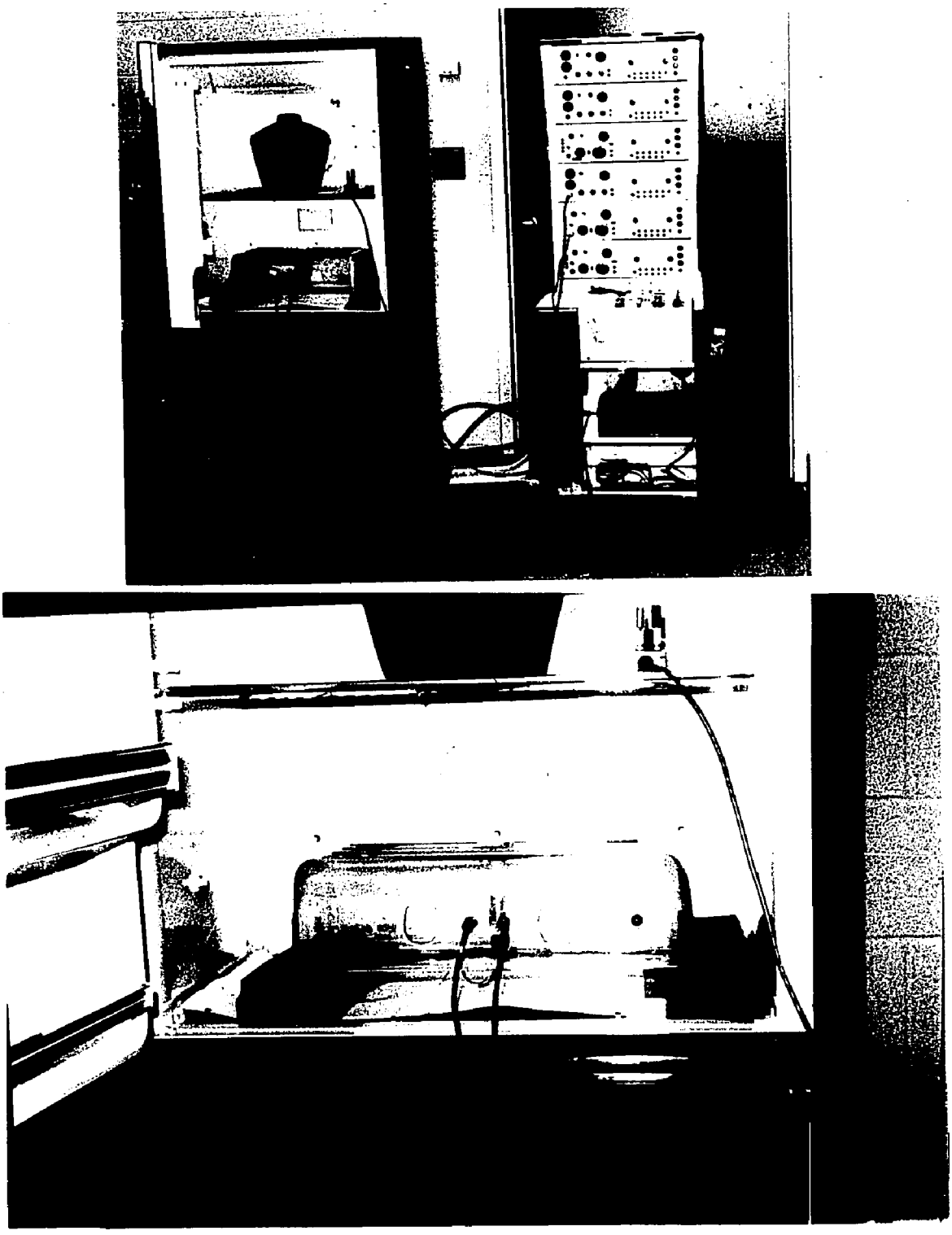


Fig. 8. A. Photograph shows the entire test set-up.
B. Photograph is a close-up of the test apparatus in the refrigerator. The syringe tube containing a bat can be seen through the semi-transparent plastic box.

Figures 9-17.

Graphs of the body temperature and heart rate of the normal control animals.

Arrows 1 and 2 in figures 14-17 indicate the beginning and end of the thermoregulatory phase of each temperature curve.

The Ss in figures 14-17 indicate sharp declines in heart rate which signal the end of the thermoregulatory phase.

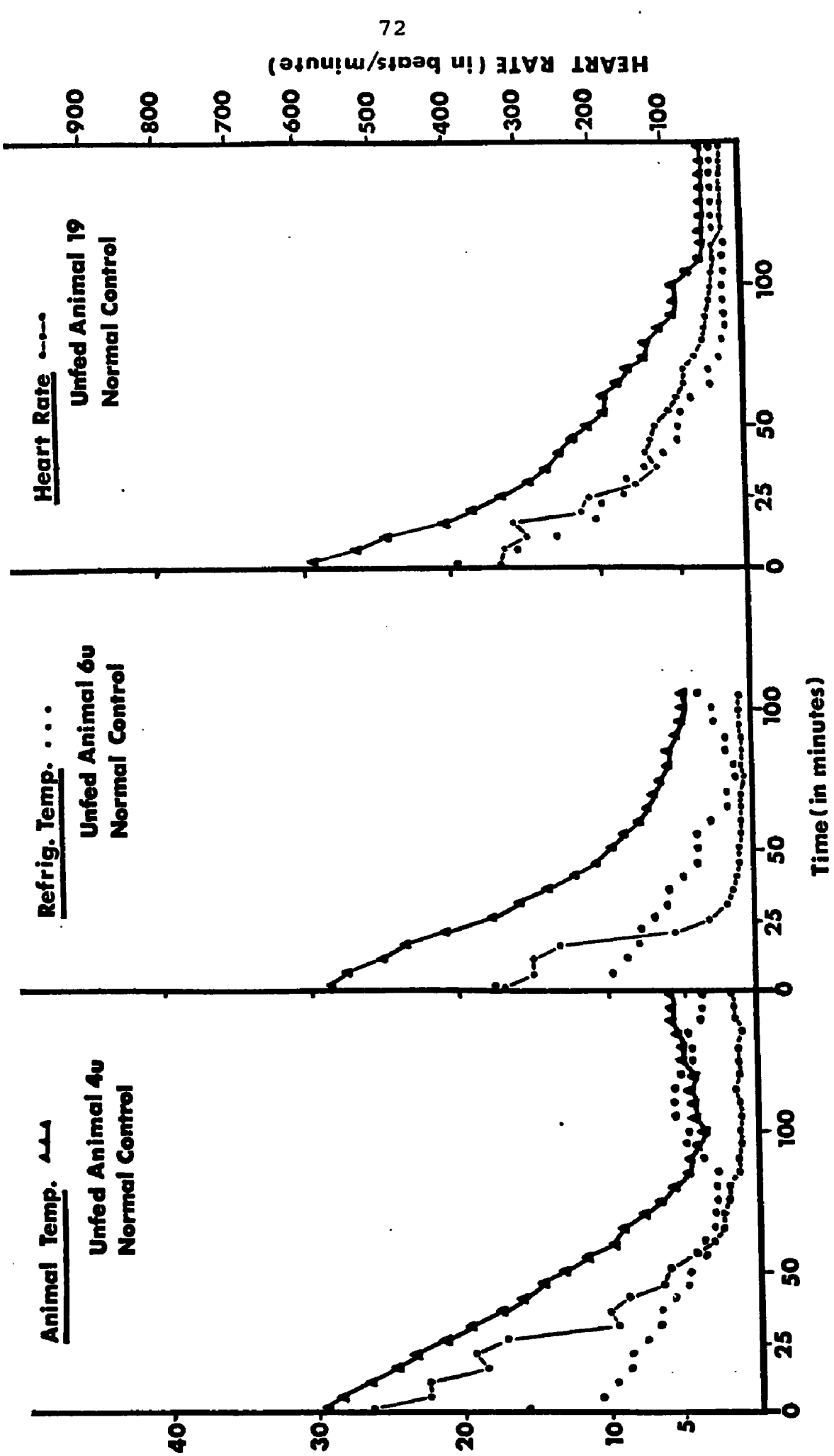

Animal Temp. ▲▲▲
Unfed Animal 4u
Normal Control
Refrig. Temp. • • •
Unfed Animal 6u
Normal Control
Heart Rate •—•
Unfed Animal 19
Normal Control
TEMPERATURE (in degrees centigrade)
HEART RATE (in beats/minute)
Time (in minutes)
40
30
20
10
5
900
800
700
600
500
400
300
200
100
0
25
50
100

Fig.9

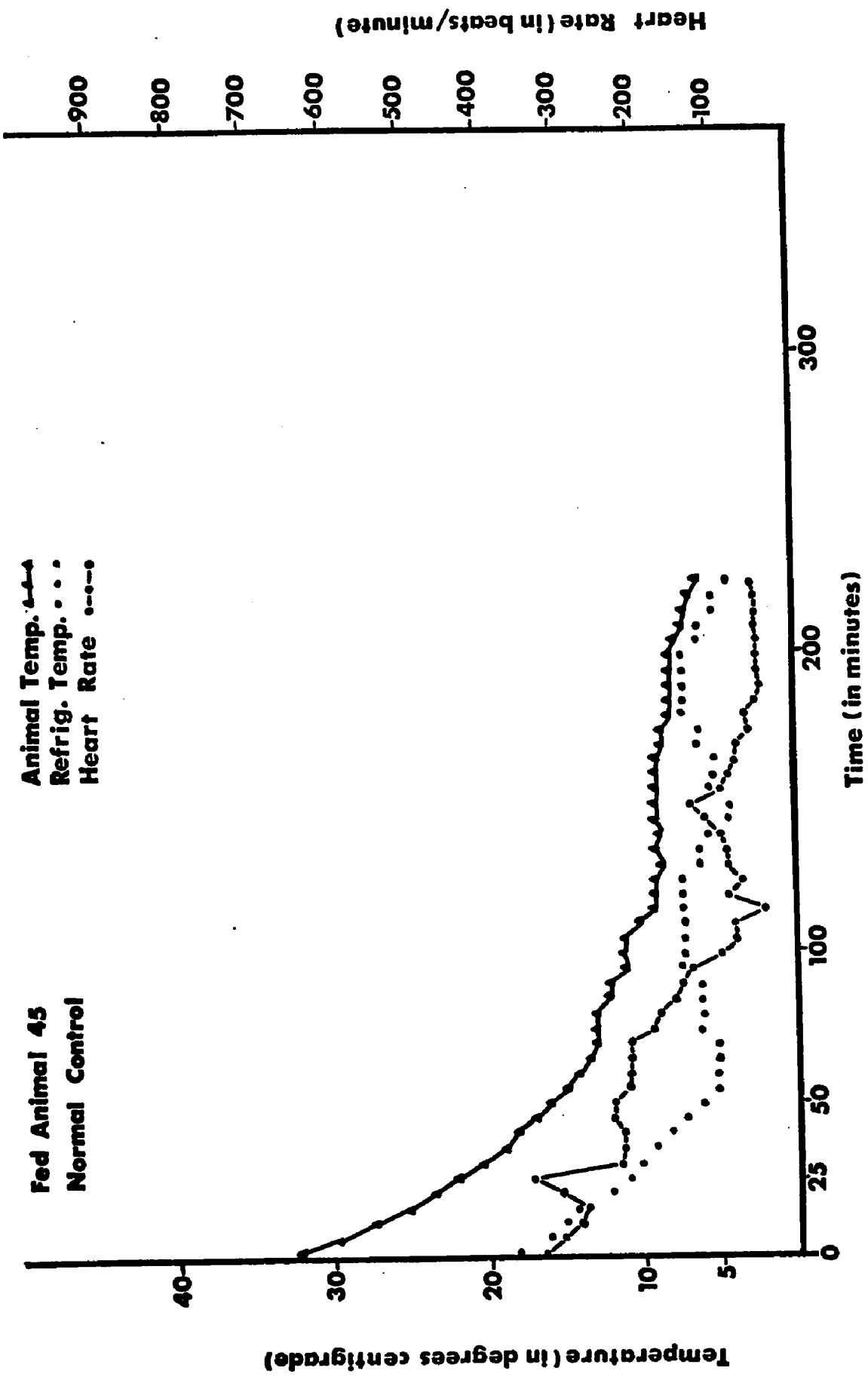

Heart Rate (in beats/minute)
900
800
700
600
500
400
300
200
100
Temperature (in degrees centigrade)
40
30
20
10
5
0
25
50
100
200
300
Time (in minutes)
Fed Animal 45
Normal Control
Animal Temp.
Refrig. Temp.
Heart Rate

Fig.10

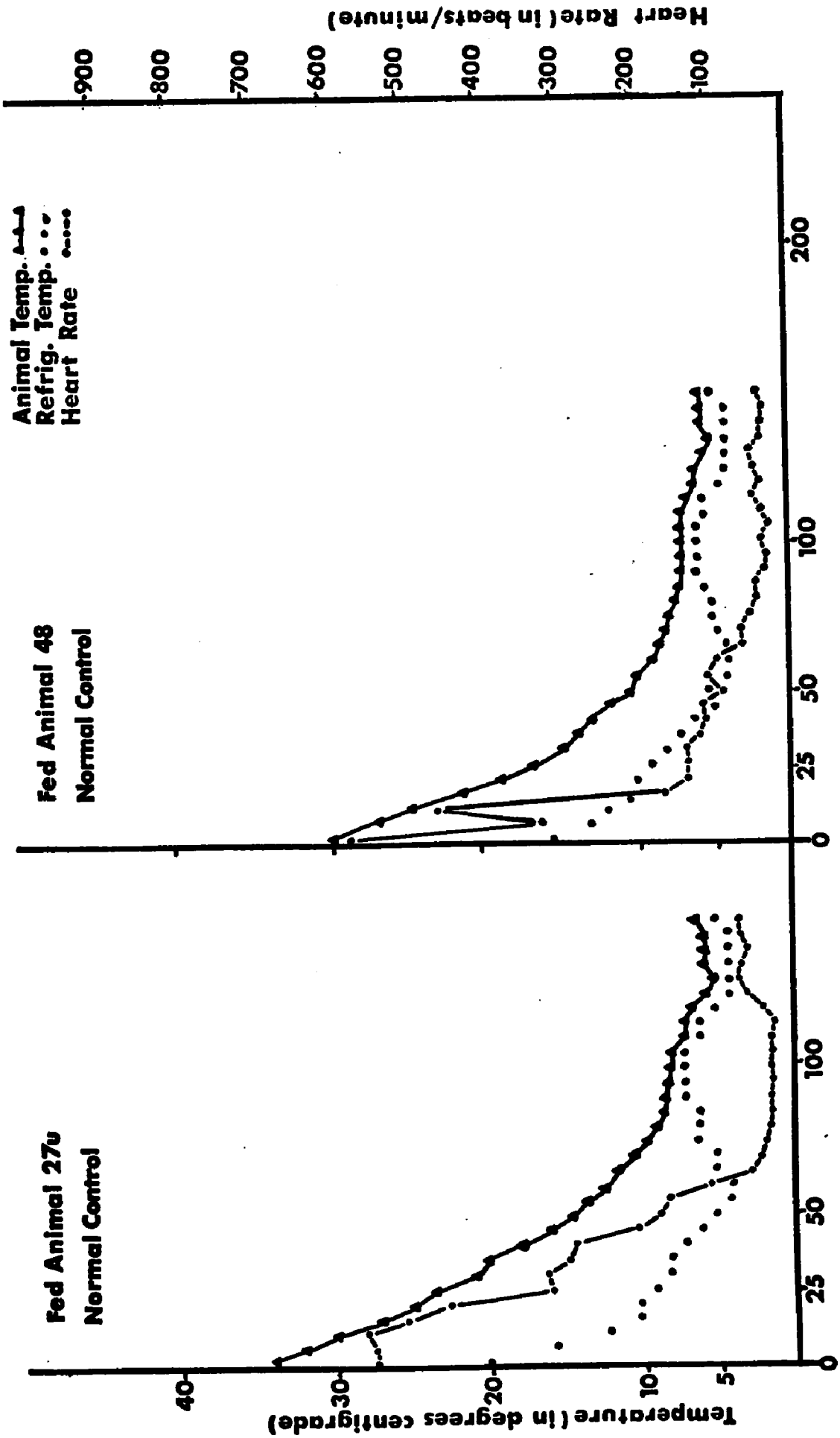

Fed Animal 27u
Normal Control
Fed Animal 48
Normal Control
Animal Temp.
Refrig. Temp.
Heart Rate
Temperature (in degrees centigrade)
Heart Rate (in beats/minute)
Time (in minutes)
0
25
50
100
200
5
10
20
30
40
100
200
300
400
500
600
700
800
900

Fig.11

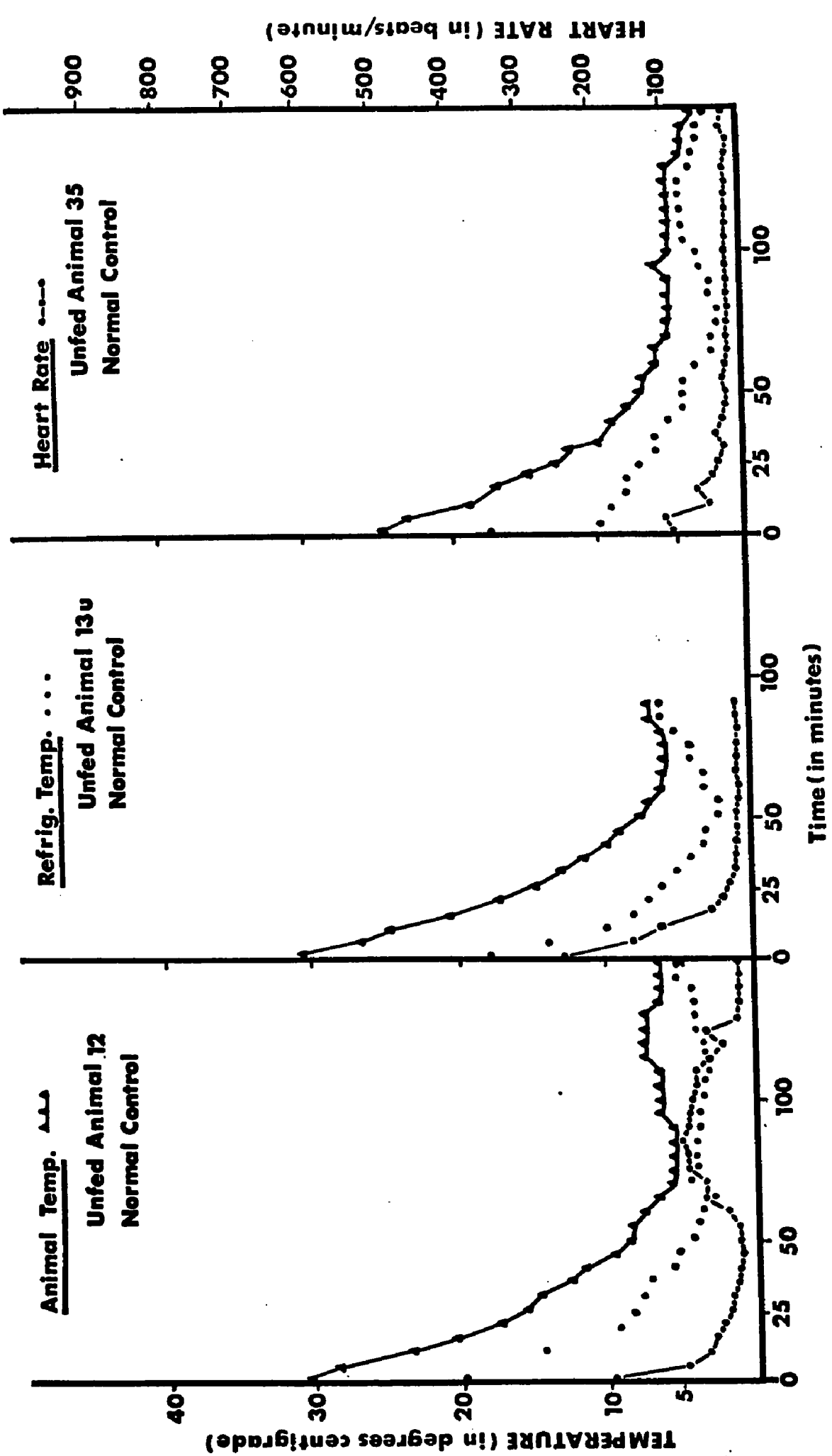

Animal Temp. ▲▲▲
Unfed Animal 12
Normal Control
Refrig. Temp. . . .
Unfed Animal 13u
Normal Control
Heart Rate •—•
Unfed Animal 35
Normal Control
TEMPERATURE (in degrees centigrade)
HEART RATE (in beats/minute)
Time (in minutes)
40
30
20
10
5
900
800
700
600
500
400
300
200
100
0
25
50
100

Fig.12

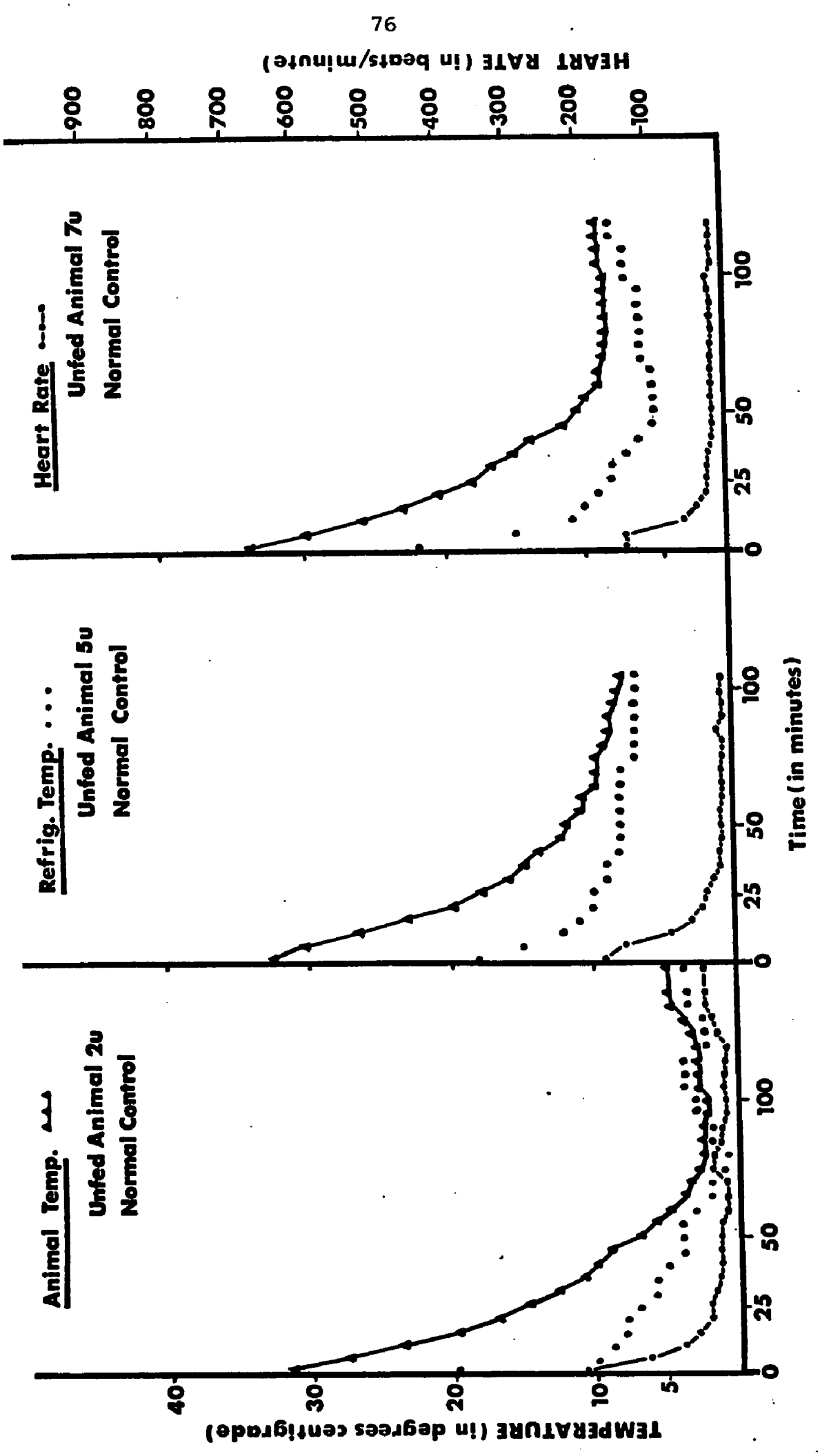

Animal Temp. ▲▲▲
Unfed Animal 2u
Normal Control
Refrig. Temp. • • •
Unfed Animal 5u
Normal Control
Heart Rate ▪—▪—▪
Unfed Animal 7u
Normal Control
TEMPERATURE (in degrees centigrade)
HEART RATE (in beats/minute)
Time (in minutes)
40
30
20
10
5
900
800
700
600
500
400
300
200
100
0
25
50
100

Fig.13

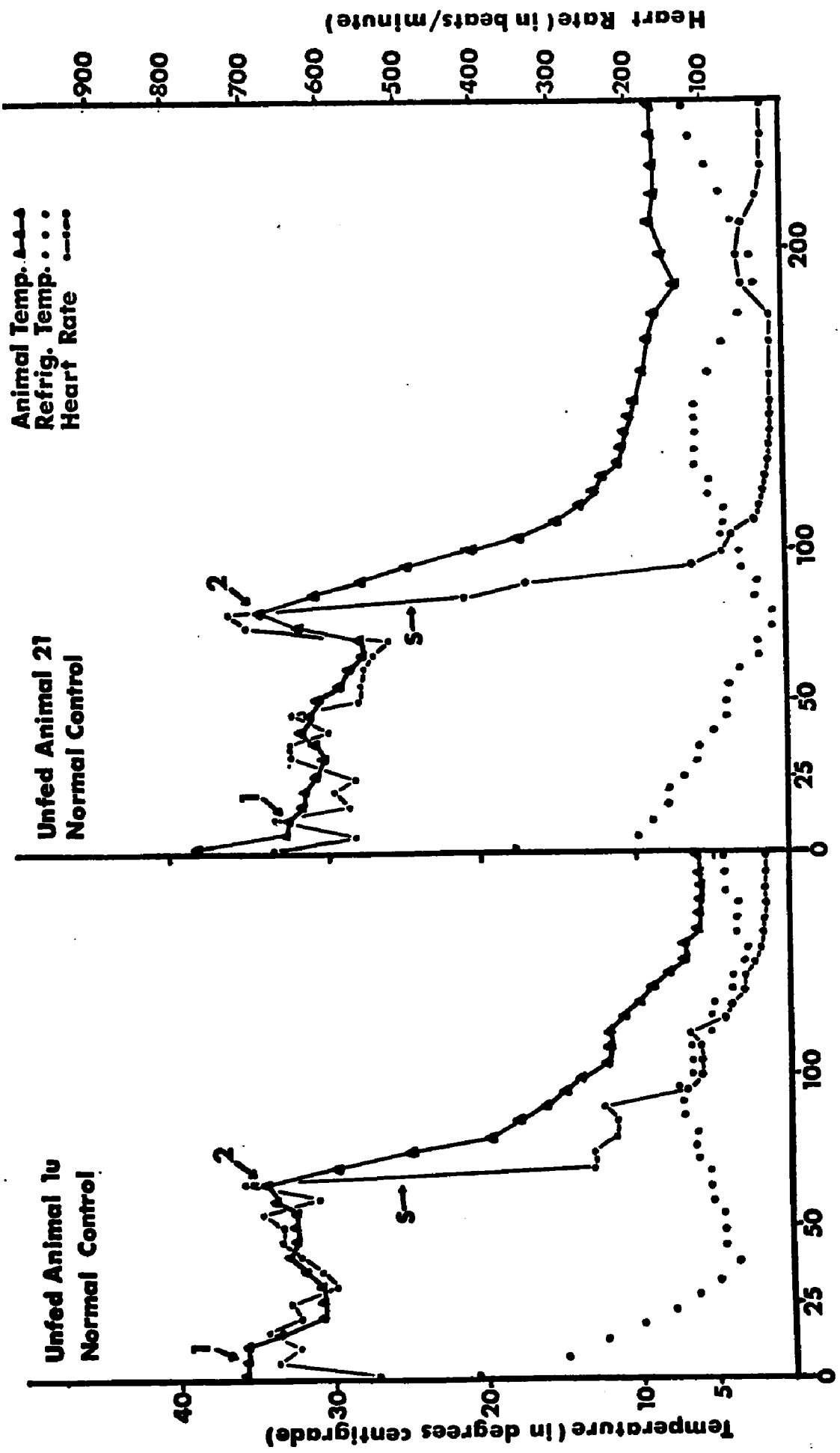

Unfed Animal 1u
Normal Control
Unfed Animal 21
Normal Control
Animal Temp.
Refrig. Temp.
Heart Rate
Temperature (in degrees centigrade)
Heart Rate (in beats/minute)
Time (in minutes)
1
2
S
0
25
50
100
200
5
10
20
30
40
100
200
300
400
500
600
700
800
900

Fig.14

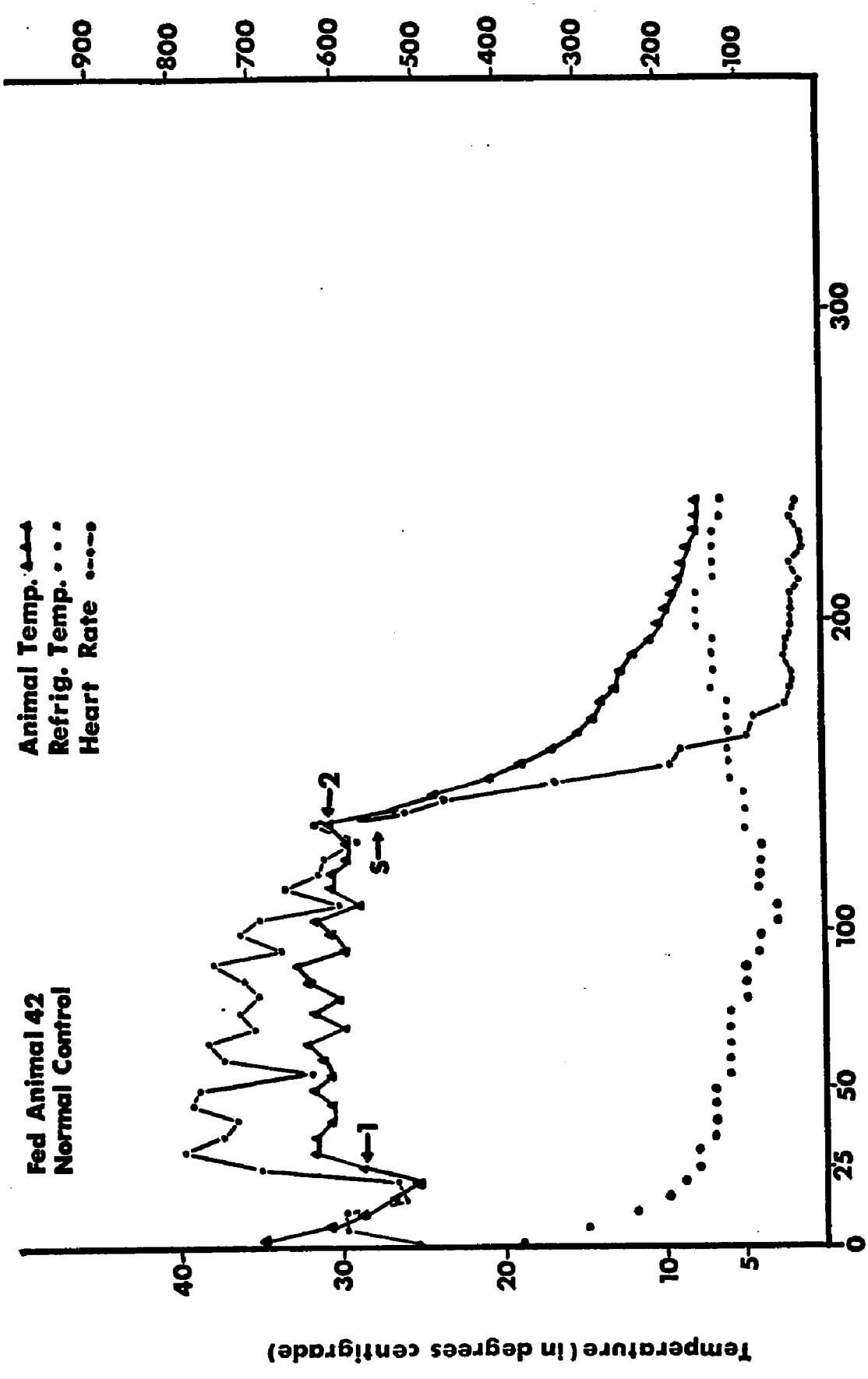

Fed Animal 42
Normal Control
Animal Temp.
Refrig. Temp.
Heart Rate
1
2
S
Temperature (in degrees centigrade)
Heart Rate (in beats/minute)
Time (in minutes)
40
30
20
10
5
900
800
700
600
500
400
300
200
100
0
25
50
100
200
300

Fig.15

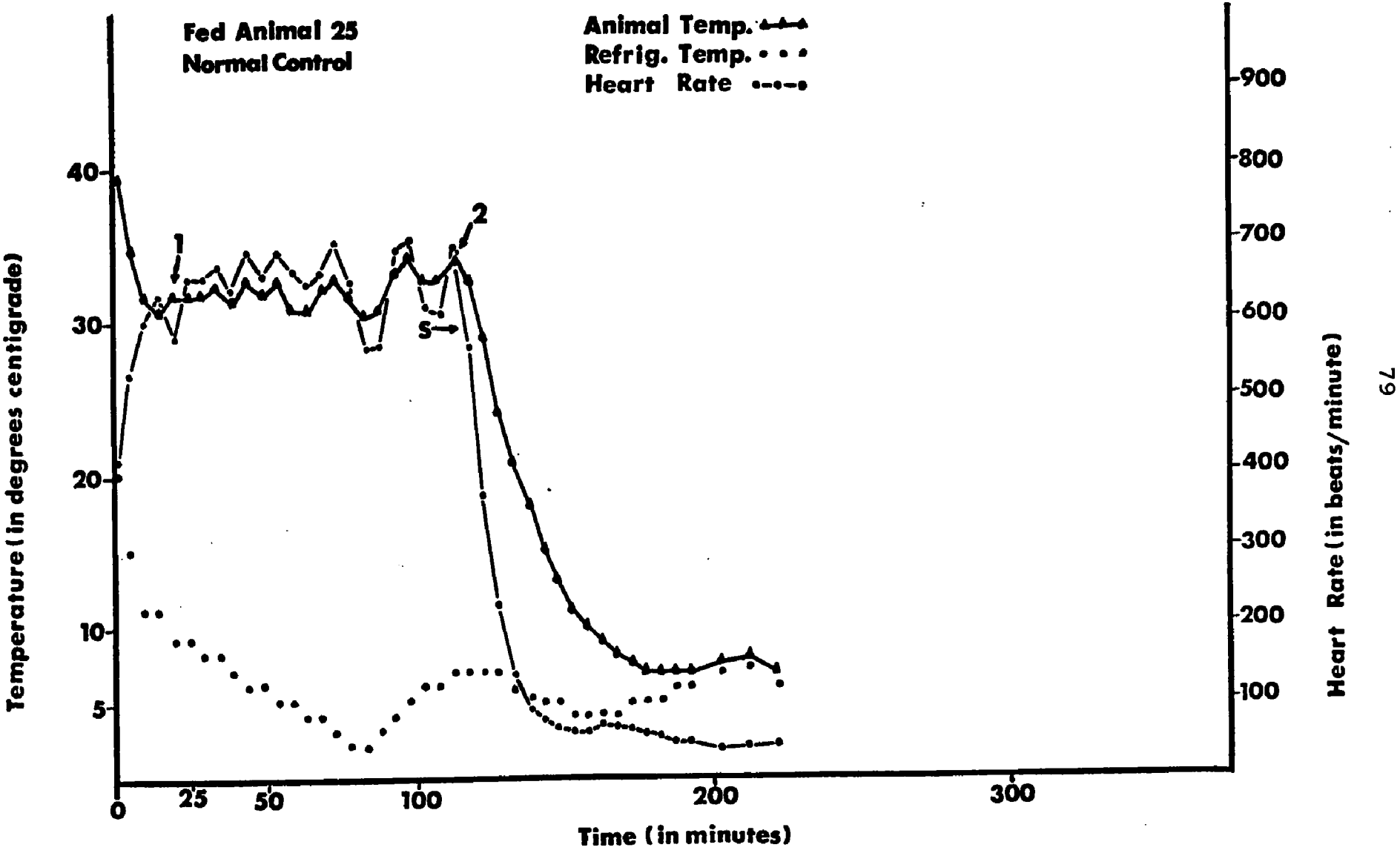

Fed Animal 25
Normal Control
Animal Temp.
Refrig. Temp.
Heart Rate
Temperature (in degrees centigrade)
Heart Rate (in beats/minute)
Time (in minutes)
40
30
20
10
5
900
800
700
600
500
400
300
200
100
0
25
50
100
200
300
1
2
S

Fig. 16

Fed Animal 46
Normal Control
Animal Temp.
Refrig. Temp.
Heart Rate
Temperature (in degrees centigrade)
Heart Rate (in beats/minute)
Time (in minutes)
1
2
S
0
25
50
100
200
300
400
5
10
20
30
40
100
200
300
400
500
600
700
800
900

Fig.17

Figures 18-19.

Graphs of the body temperature and heart rate of the sham operated control animals.

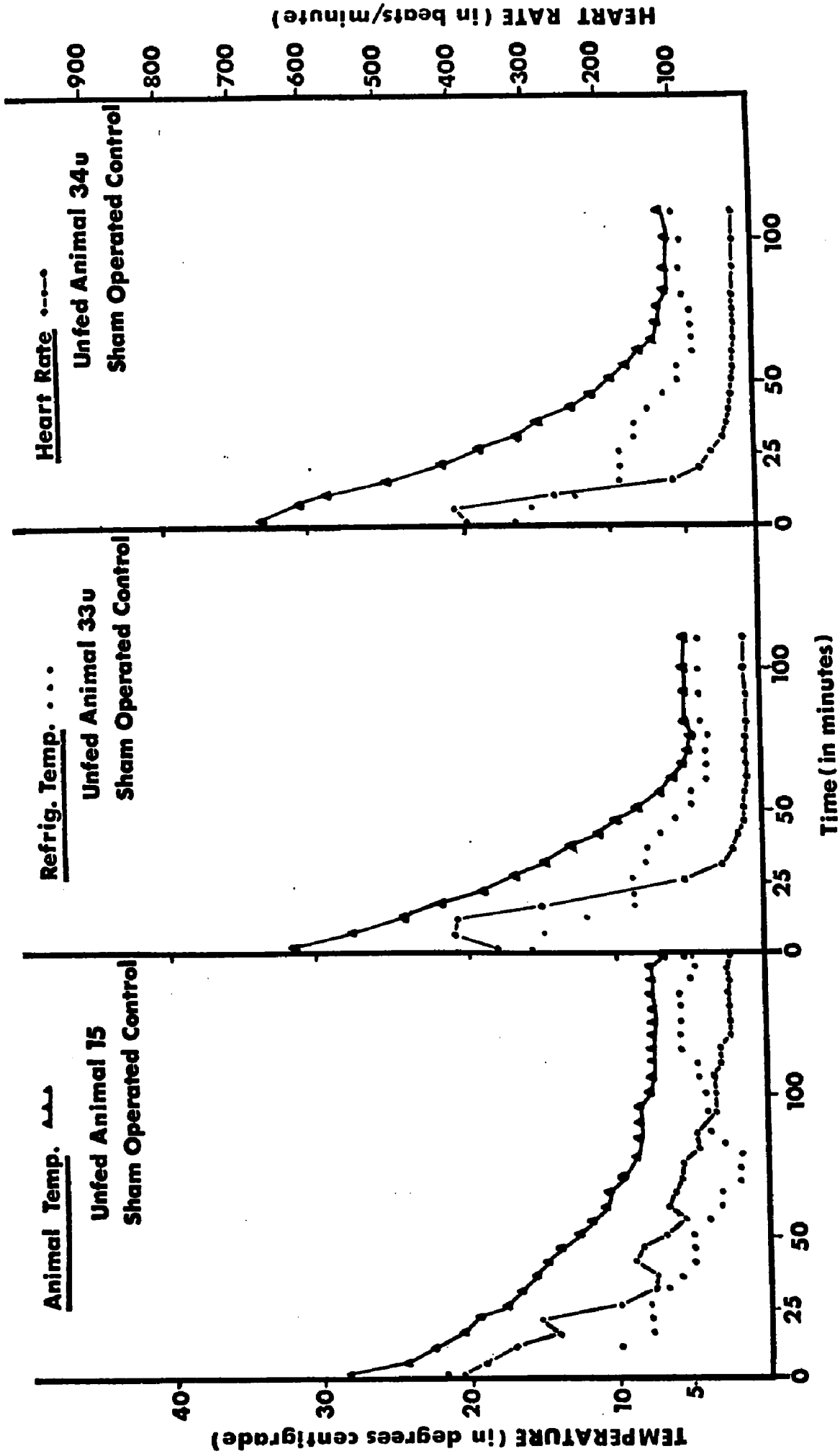

Animal Temp. ▲▲▲
Unfed Animal 15
Sham Operated Control
Refrig. Temp. . . .
Unfed Animal 33u
Sham Operated Control
Heart Rate •—•
Unfed Animal 34u
Sham Operated Control
TEMPERATURE (in degrees centigrade)
HEART RATE (in beats/minute)
Time (in minutes)
40
30
20
10
5
900
800
700
600
500
400
300
200
100
0
25
50
100

Fig.18

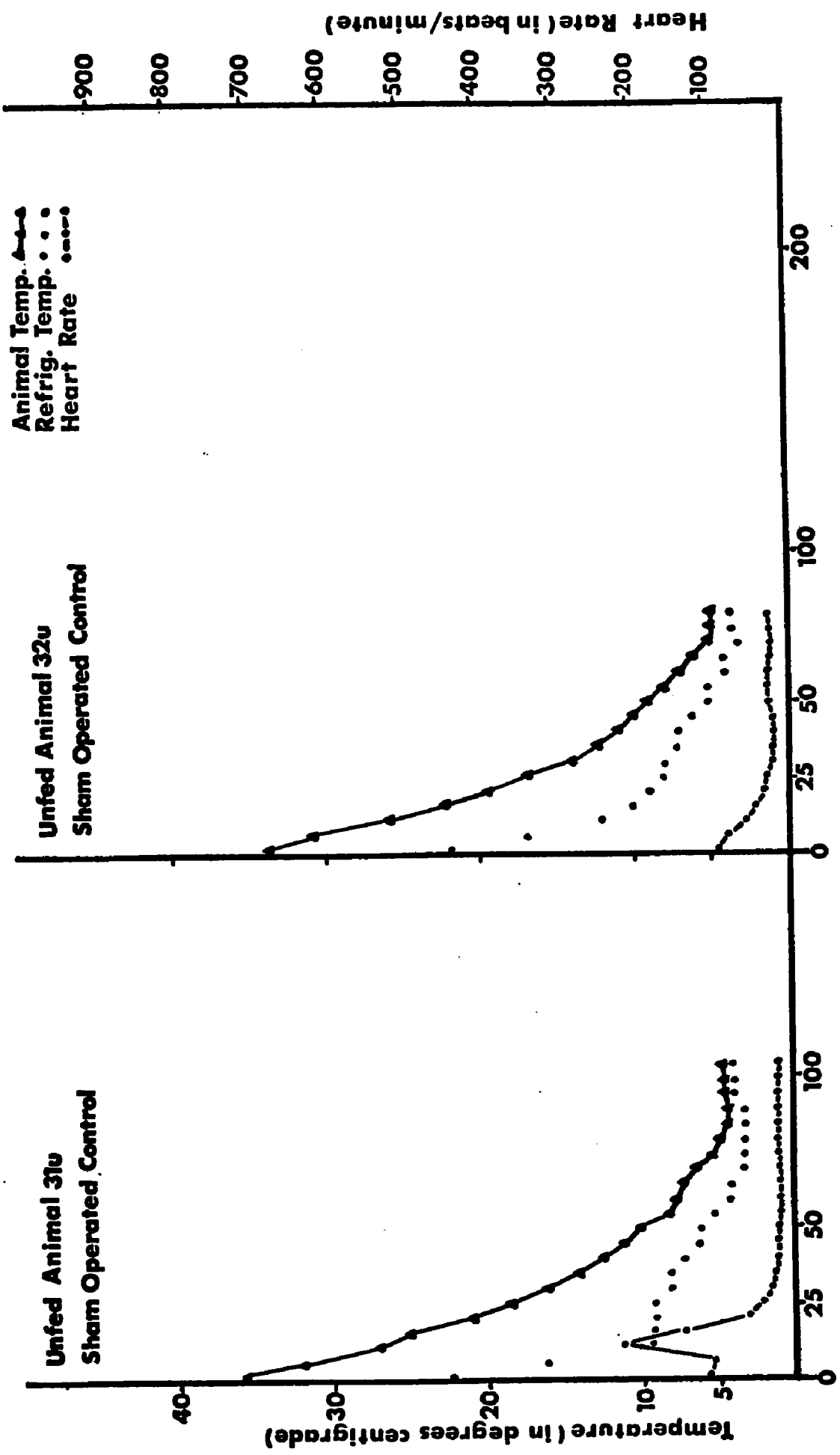
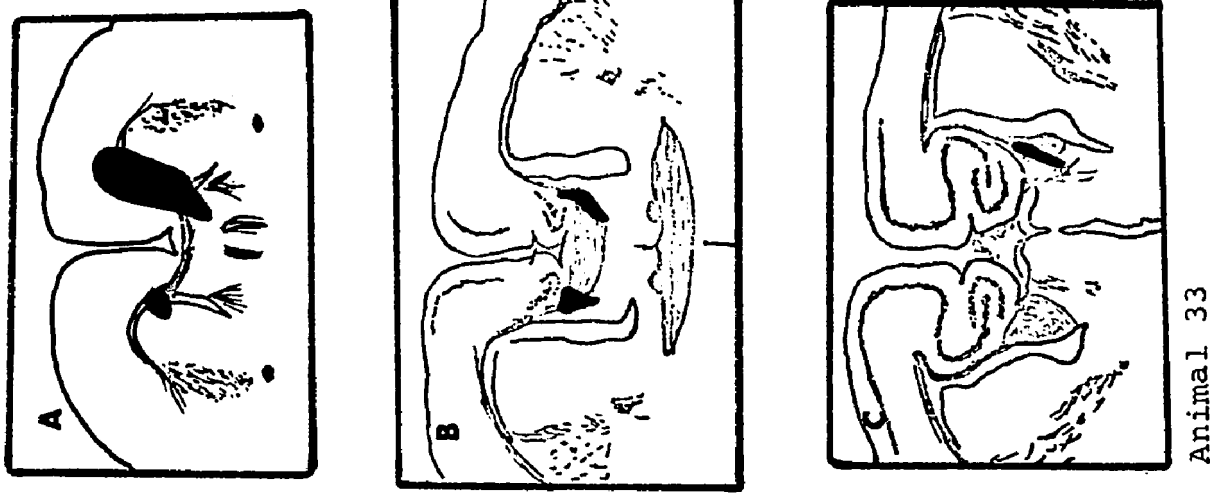

Unfed Animal 31u
Sham Operated Control
Unfed Animal 32u
Sham Operated Control
Animal Temp.
Refrig. Temp.
Heart Rate
Temperature (in degrees centigrade)
Heart Rate (in beats/minute)
Time (in minutes)
40
30
20
10
5
900
800
700
600
500
400
300
200
100
0
25
50
100
200

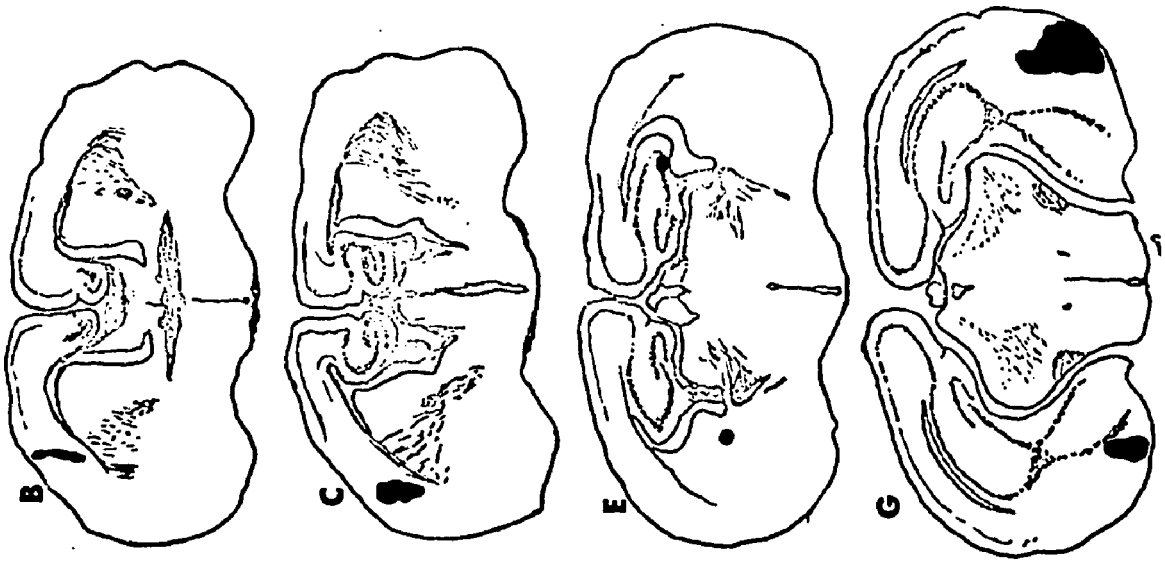
Fig.19

Figures 20-23.

Graphs of the body temperature and heart rate of the lesioned control animals.



Animal 33



Animal 20u

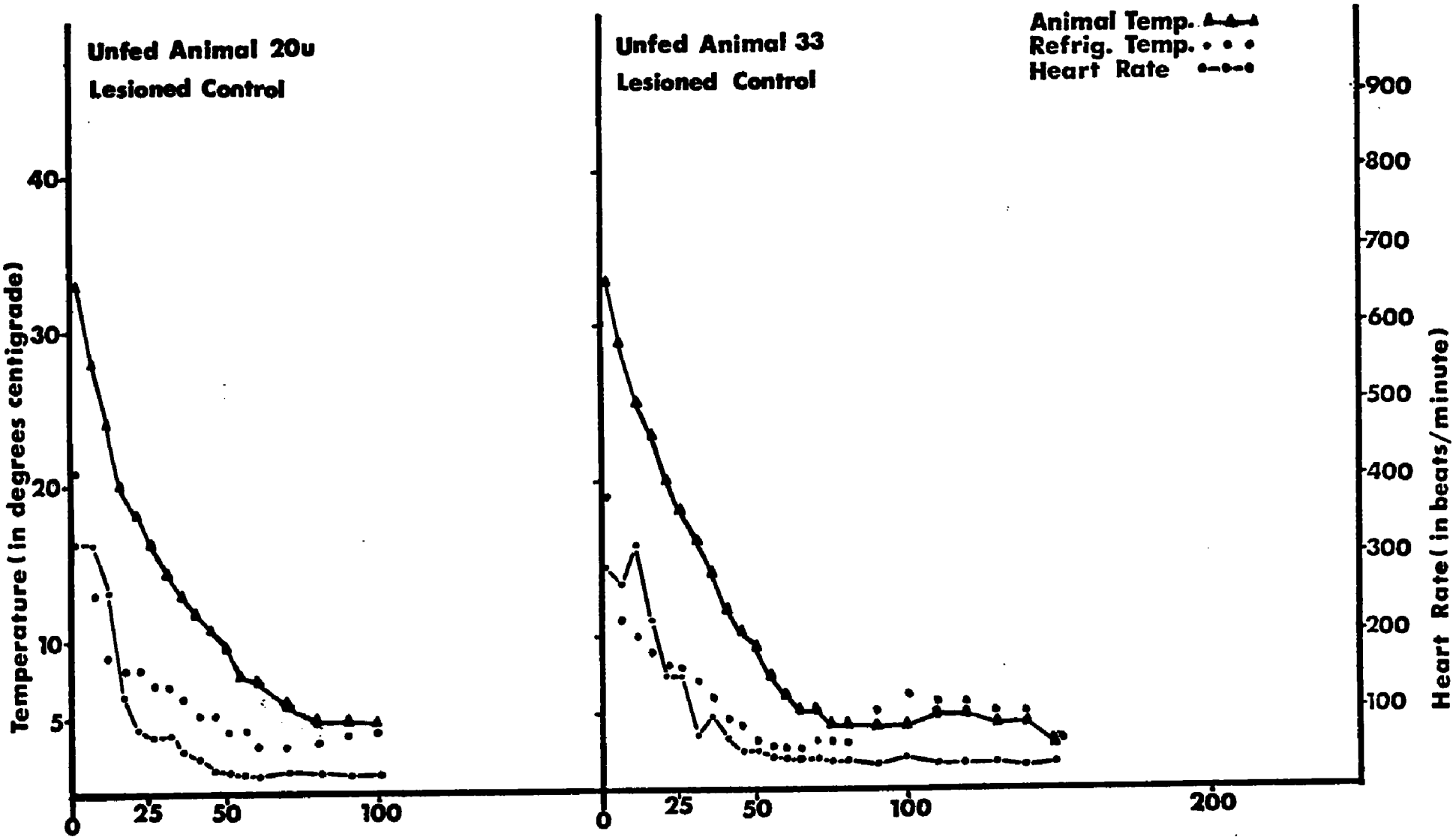
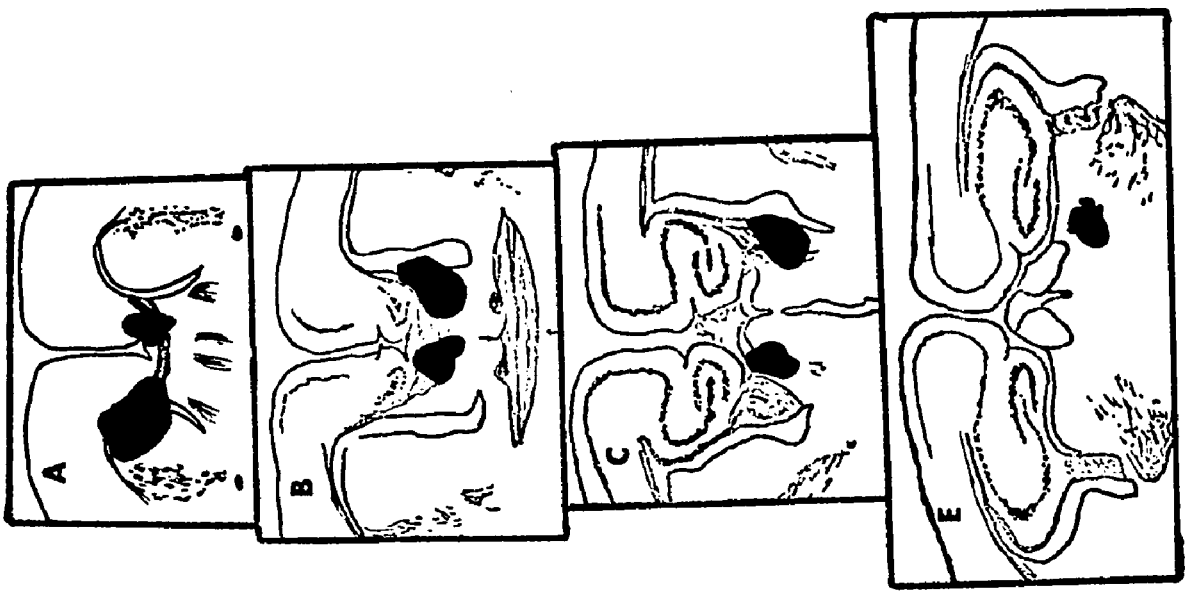
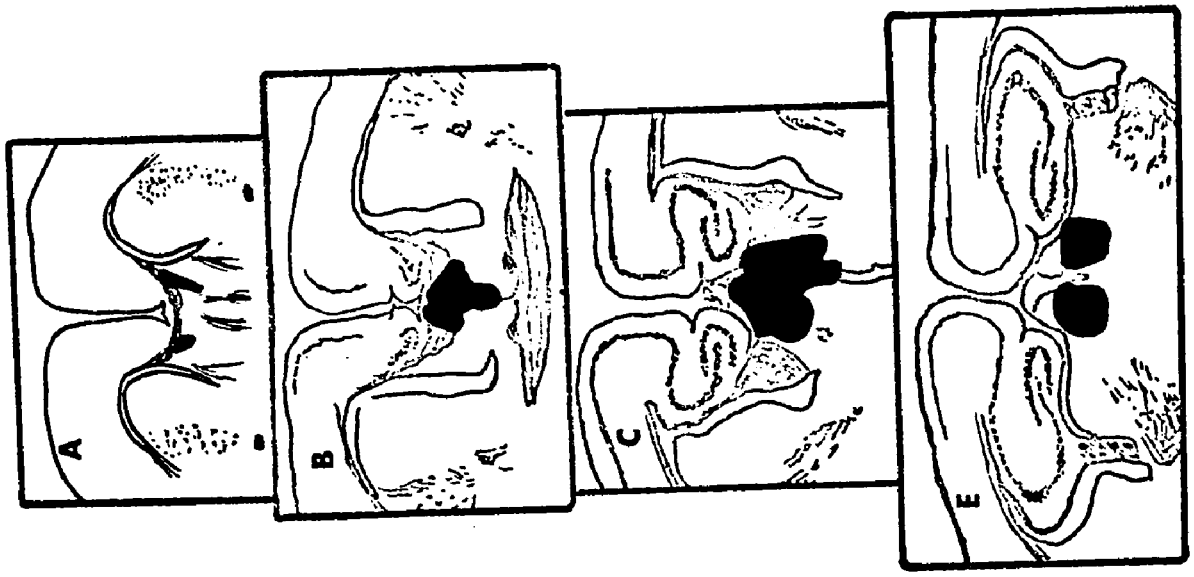

Unfed Animal 20u
Lesioned Control
Unfed Animal 33
Lesioned Control
Animal Temp.
Refrig. Temp.
Heart Rate
Temperature (in degrees centigrade)
Heart Rate (in beats/minute)
Time (in minutes)
40
30
20
10
5
900
800
700
600
500
400
300
200
100
0
25
50
100
200

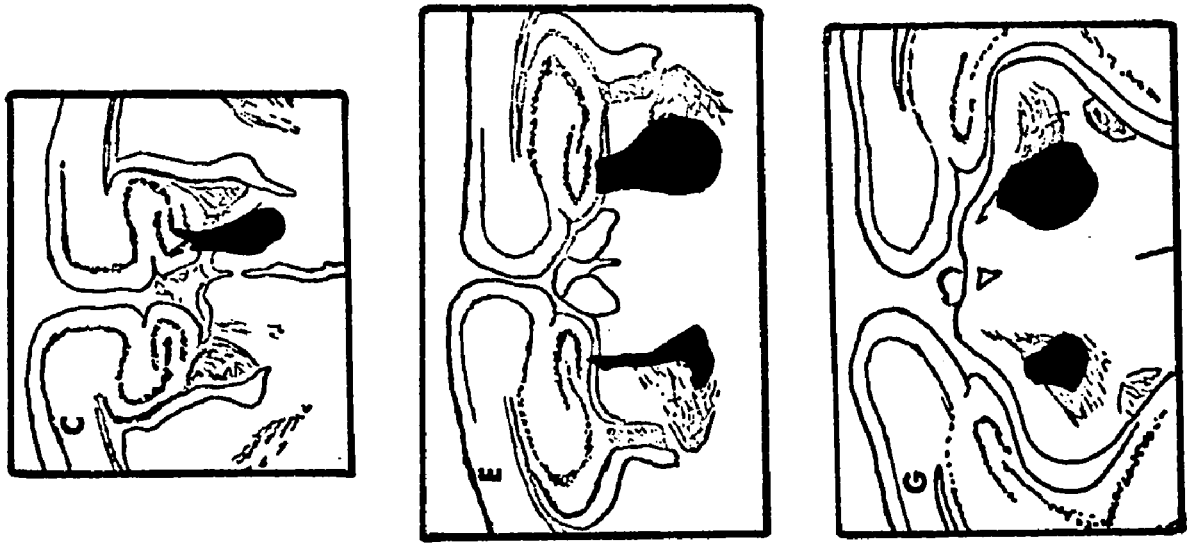
Fig. 20



Animal 42u



Animal 40u



Animal 23u

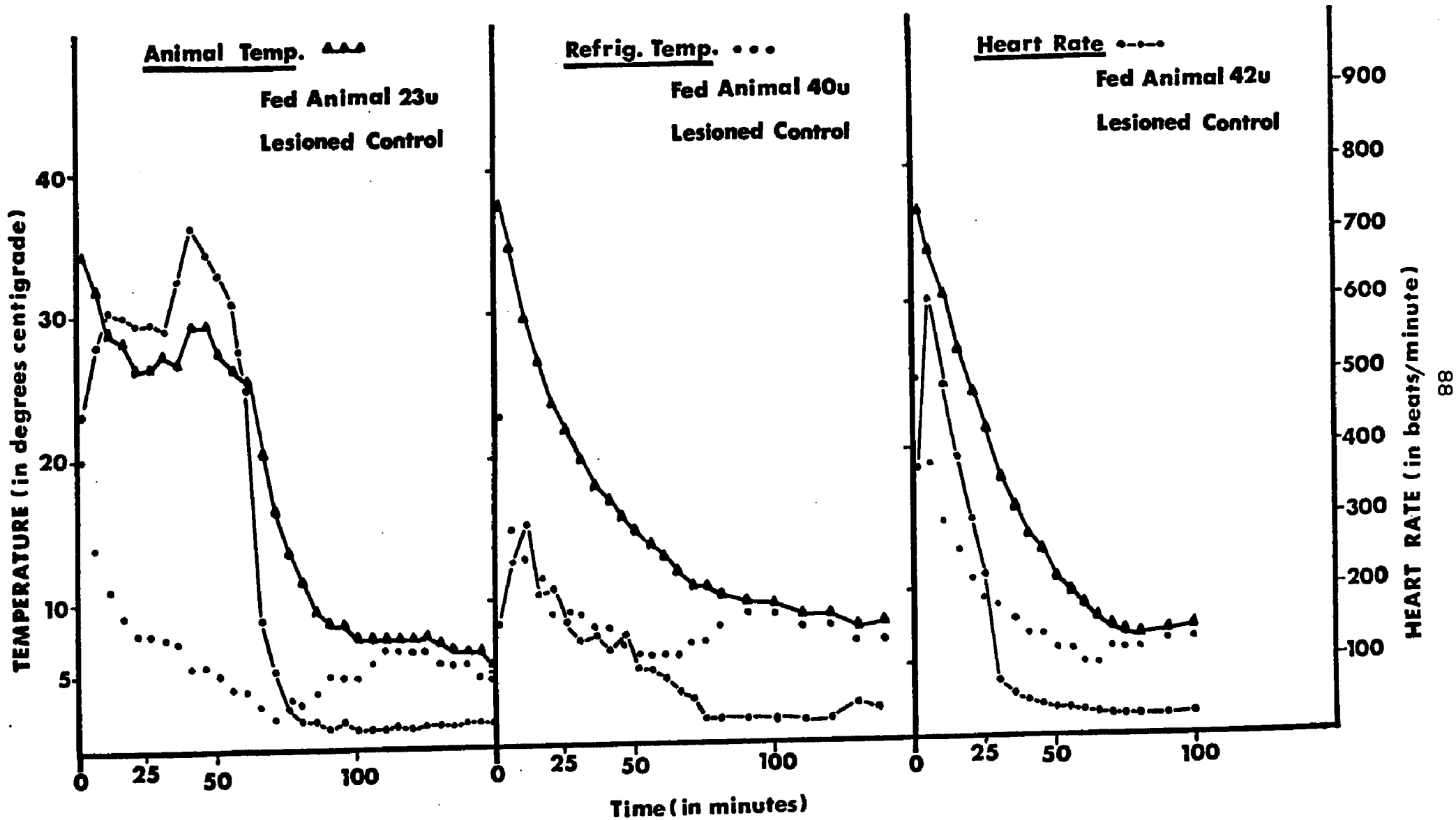
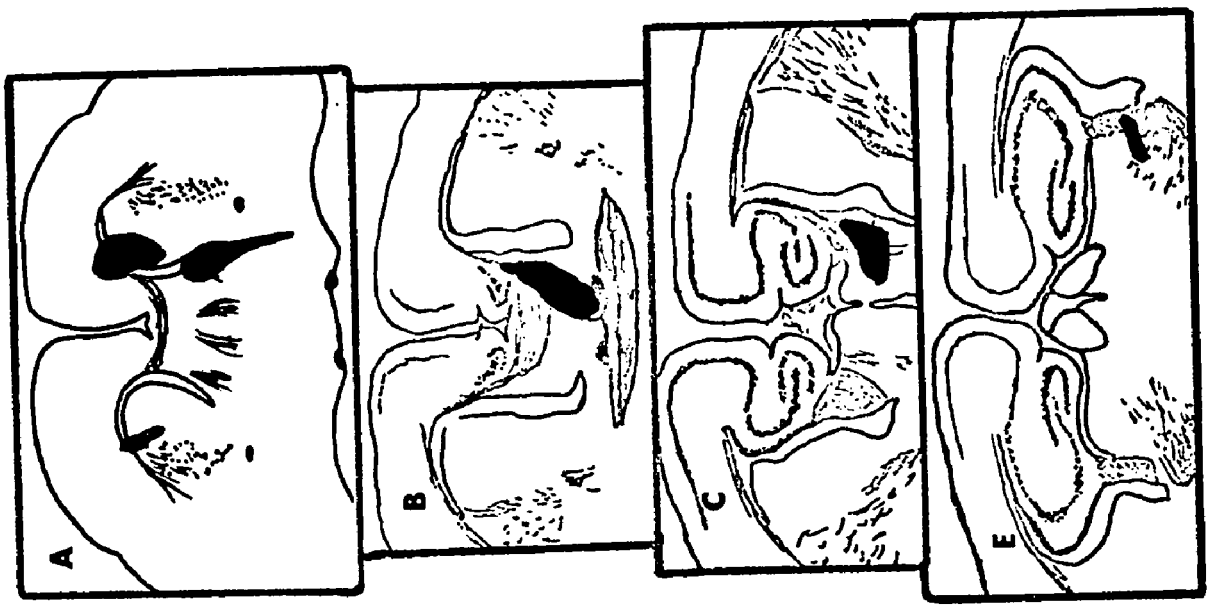
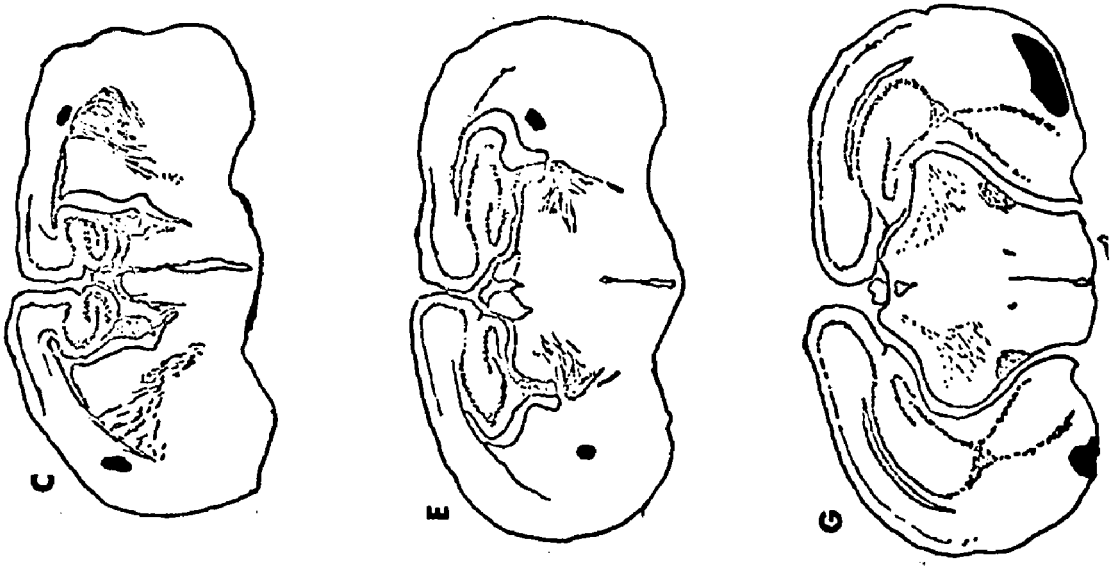

Animal Temp. ▲▲▲
Fed Animal 23u
Lesioned Control
Refrig. Temp. • • •
Fed Animal 40u
Lesioned Control
Heart Rate •-•-•
Fed Animal 42u
Lesioned Control
TEMPERATURE (in degrees centigrade)
HEART RATE (in beats/minute)
Time (in minutes)
40
30
20
10
5
900
800
700
600
500
400
300
200
100
0
25
50
100

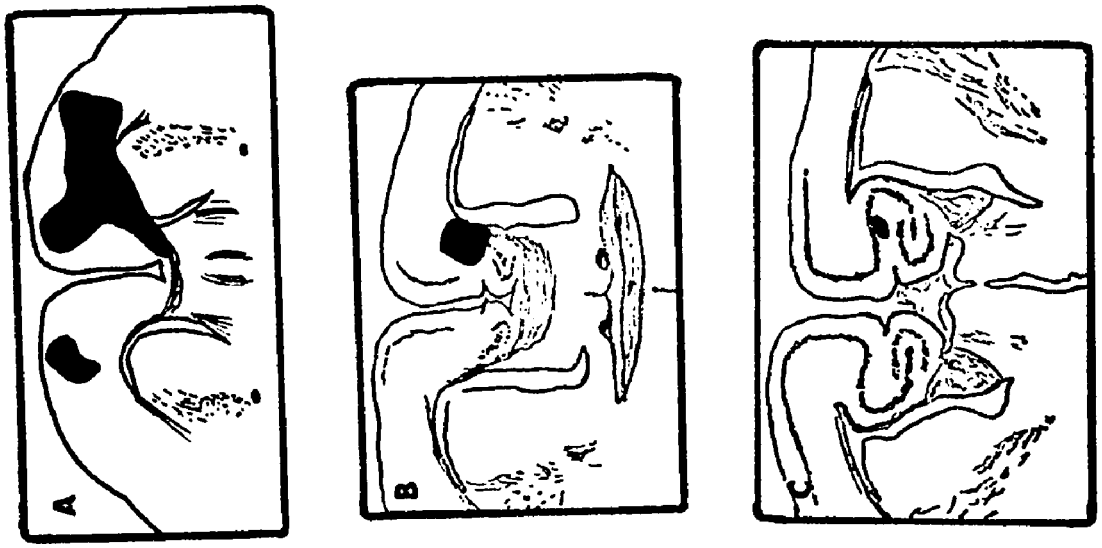
Fig. 21



Animal 32



Animal 19u



Animal 16

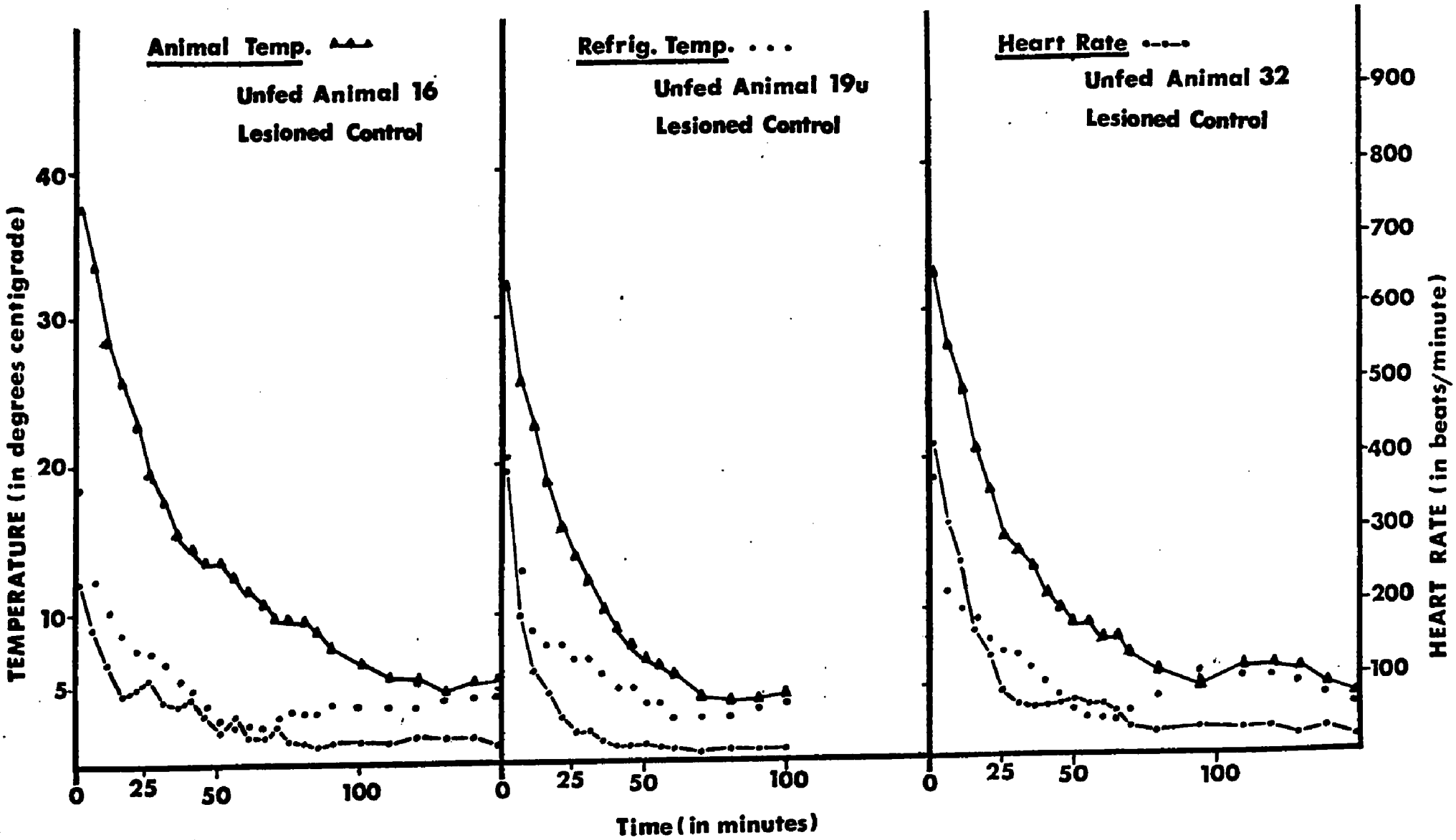
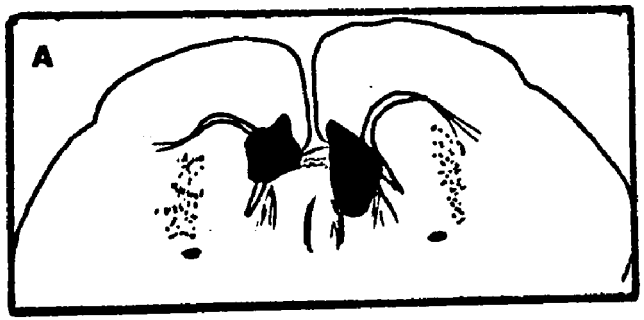

Animal Temp.
Unfed Animal 16
Lesioned Control
Refrig. Temp.
Unfed Animal 19u
Lesioned Control
Heart Rate
Unfed Animal 32
Lesioned Control
TEMPERATURE (in degrees centigrade)
HEART RATE (in beats/minute)
Time (in minutes)
40
30
20
10
5
900
800
700
600
500
400
300
200
100
0
25
50
100

Fig. 22



Animal 29

Animal 36u, lesions of olfactory bulbs not shown.

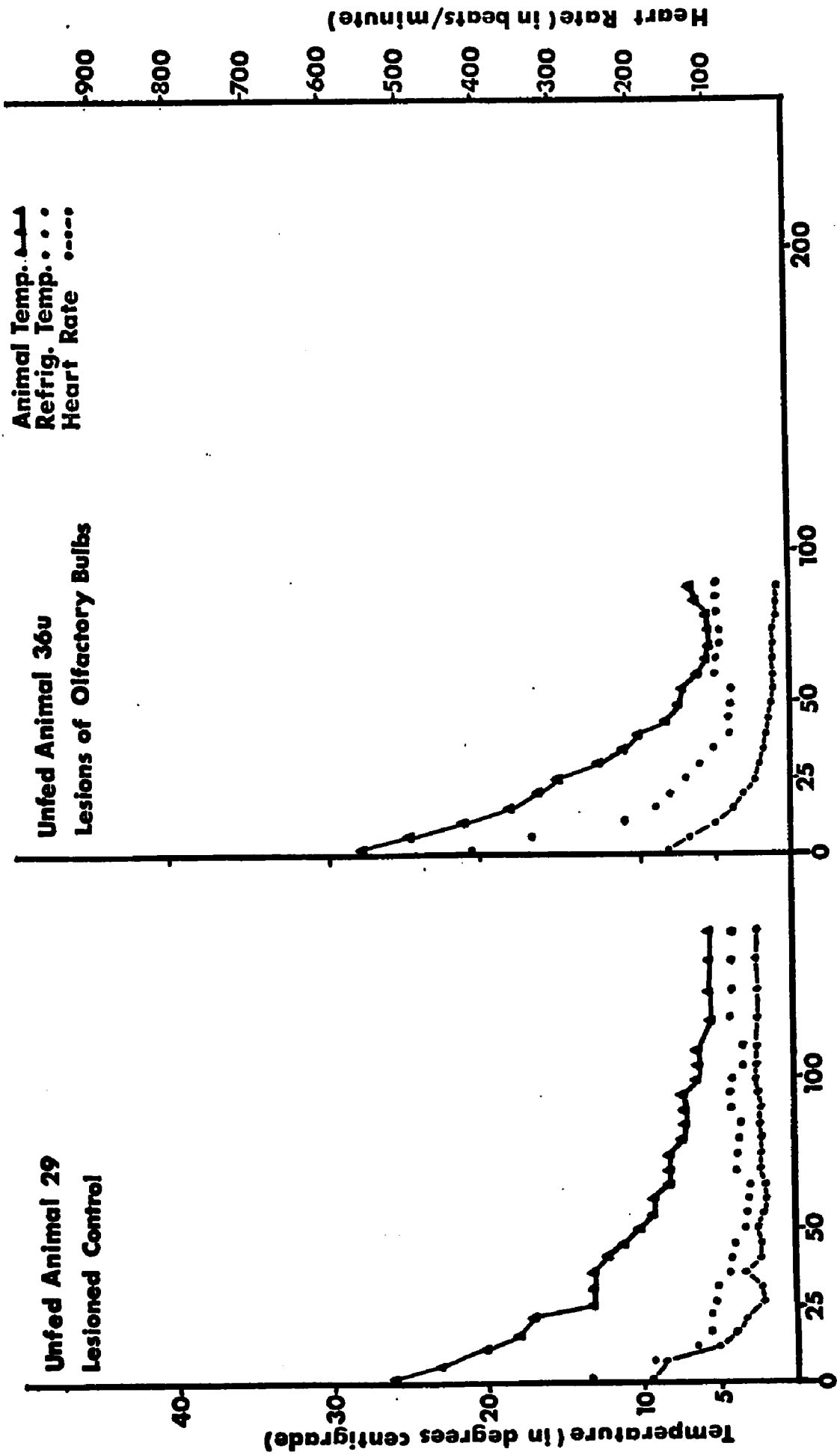
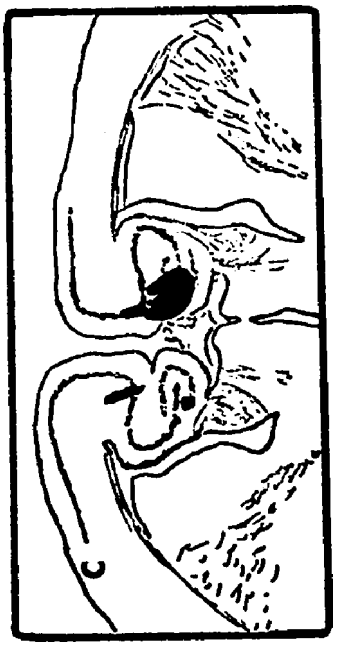

Unfed Animal 29
Lesioned Control
Unfed Animal 36u
Lesions of Olfactory Bulbs
Animal Temp.
Refrig. Temp.
Heart Rate
Temperature (in degrees centigrade)
Heart Rate (in beats/minute)
Time (in minutes)
0
25
50
100
200
5
10
20
30
40
100
200
300
400
500
600
700
800
900

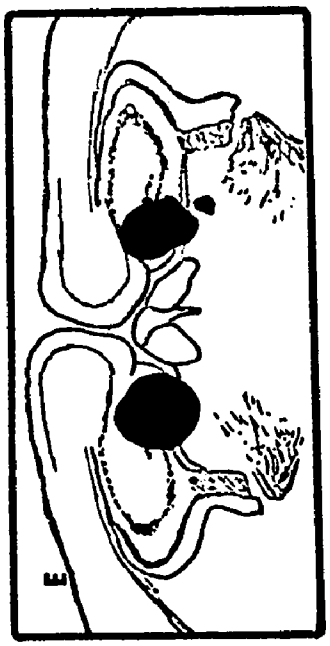
Fig.23

Figures 24-36.

Graphs of the body temperature and heart rate of the lesioned animals.

The Ss in figures 24, 25 and 27 indicate sharp declines in heart rate which suggest attempts to begin entry into hibernation.





Animal 40

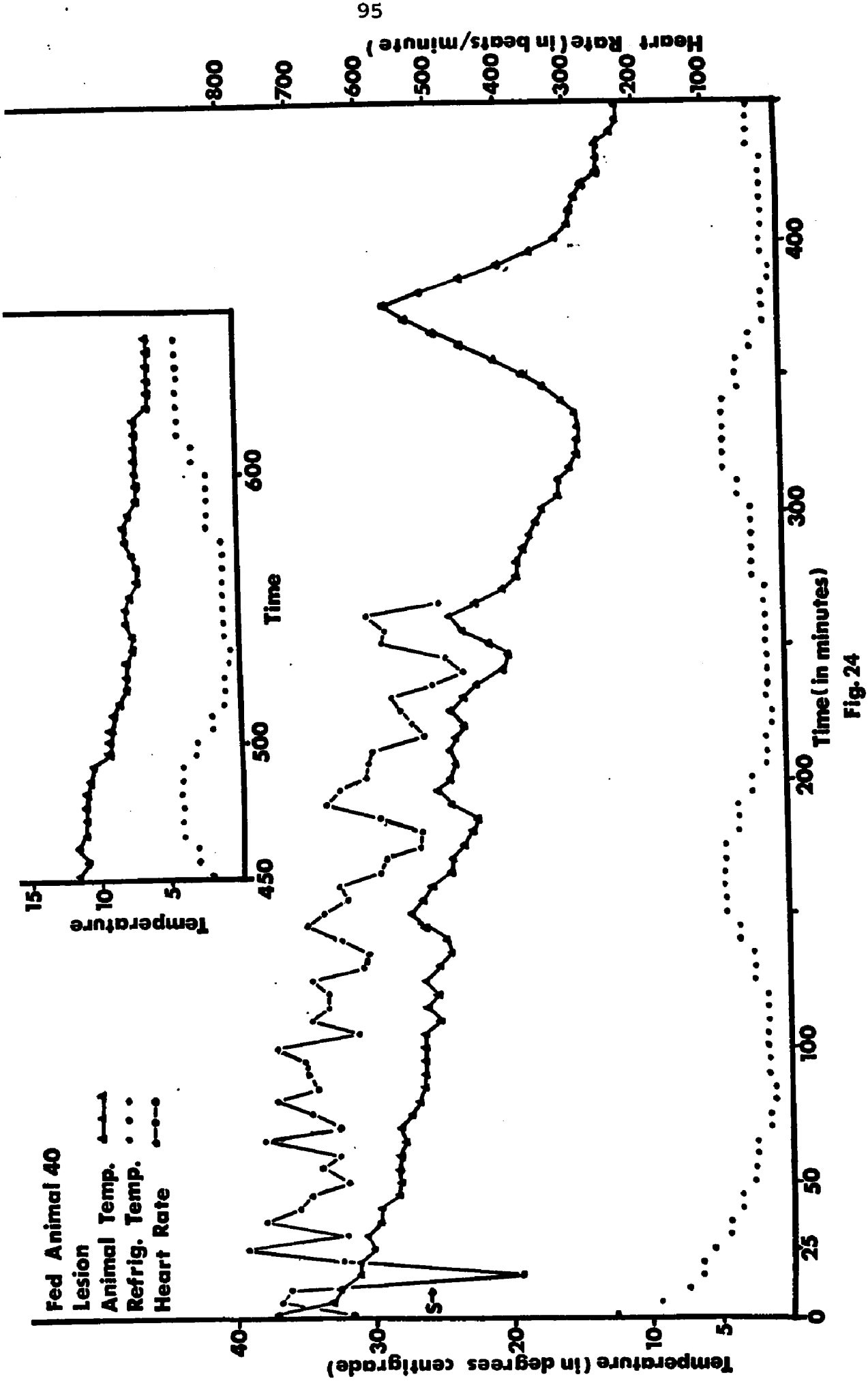
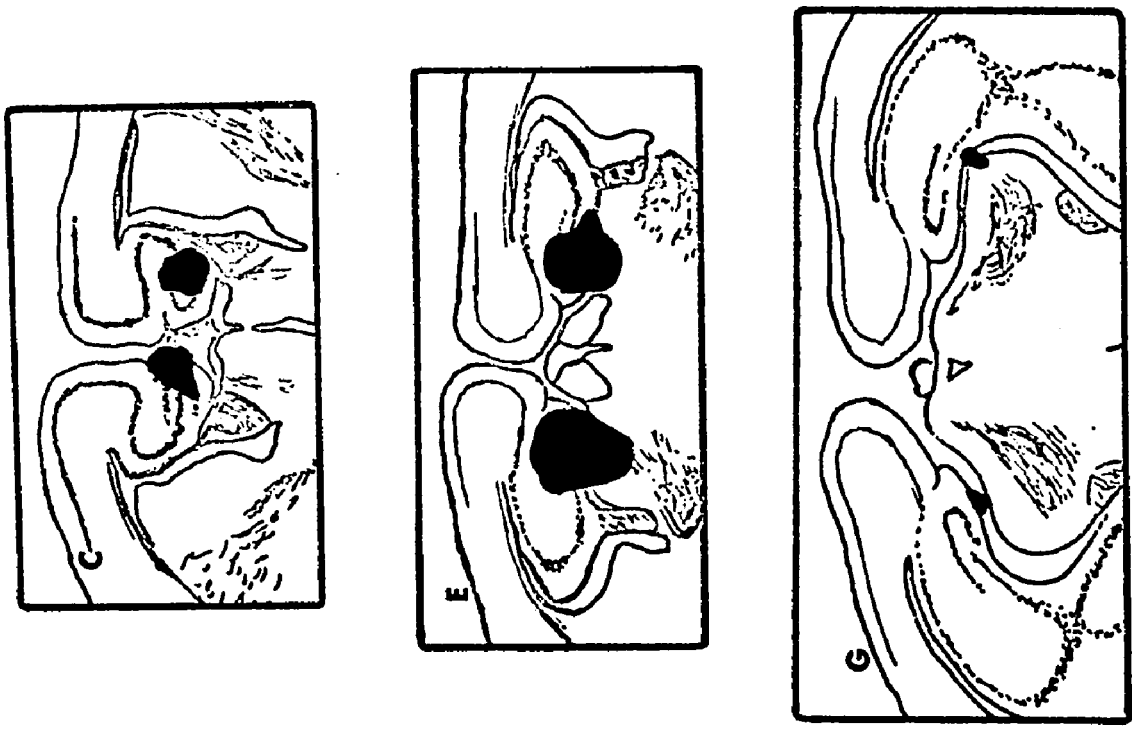

Fed Animal 40
Lesion
Animal Temp.
Refrig. Temp.
Heart Rate
Temperature (in degrees centigrade)
Heart Rate (in beats/minute)
Time (in minutes)
Temperature
Time

Fig. 24



Animal 38u

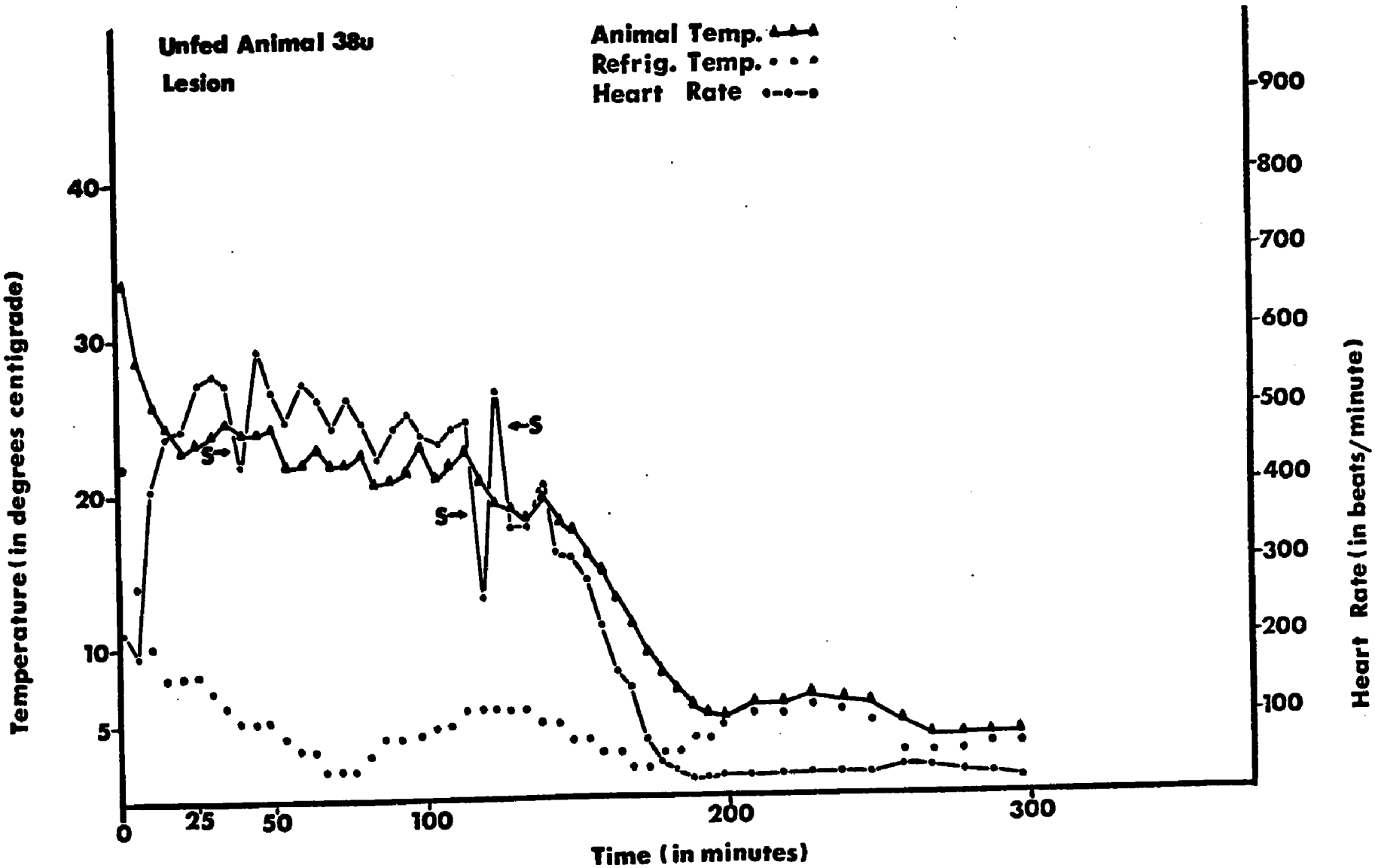
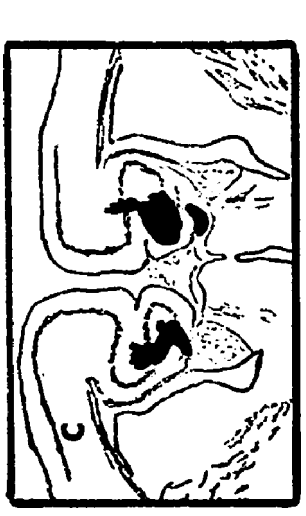
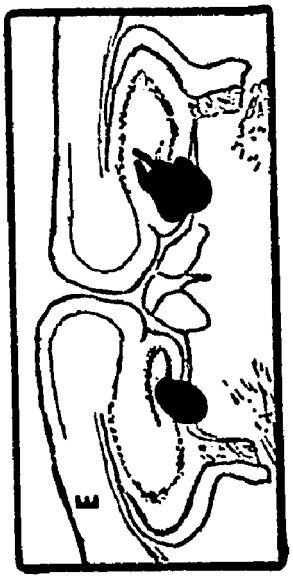

Unfed Animal 38u
Lesion
Animal Temp.
Refrig. Temp.
Heart Rate
Temperature (in degrees centigrade)
Heart Rate (in beats/minute)
Time (in minutes)
40
30
20
10
5
900
800
700
600
500
400
300
200
100
0
25
50
100
200
300
S
S
S

Fig. 25





Animal 43

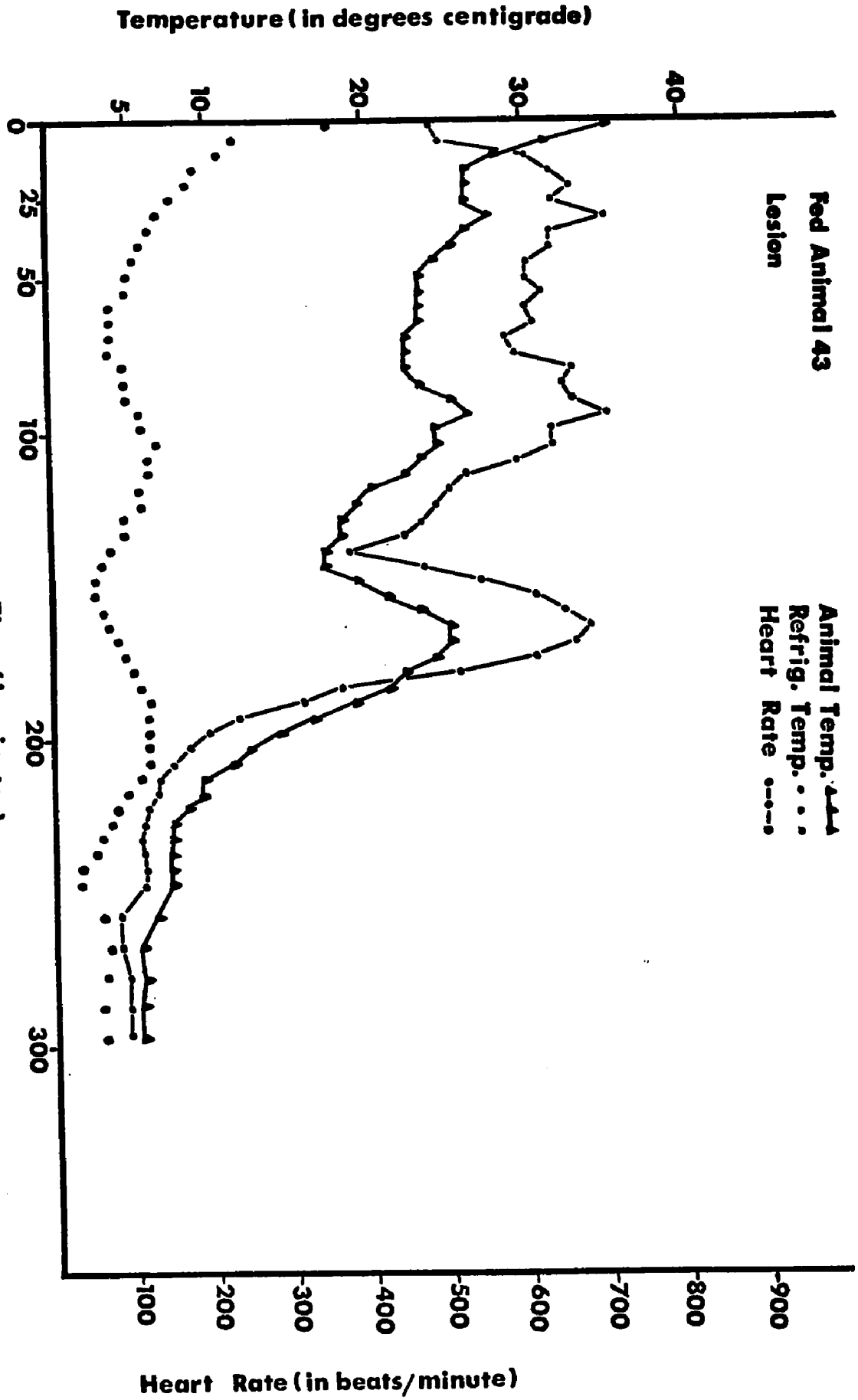
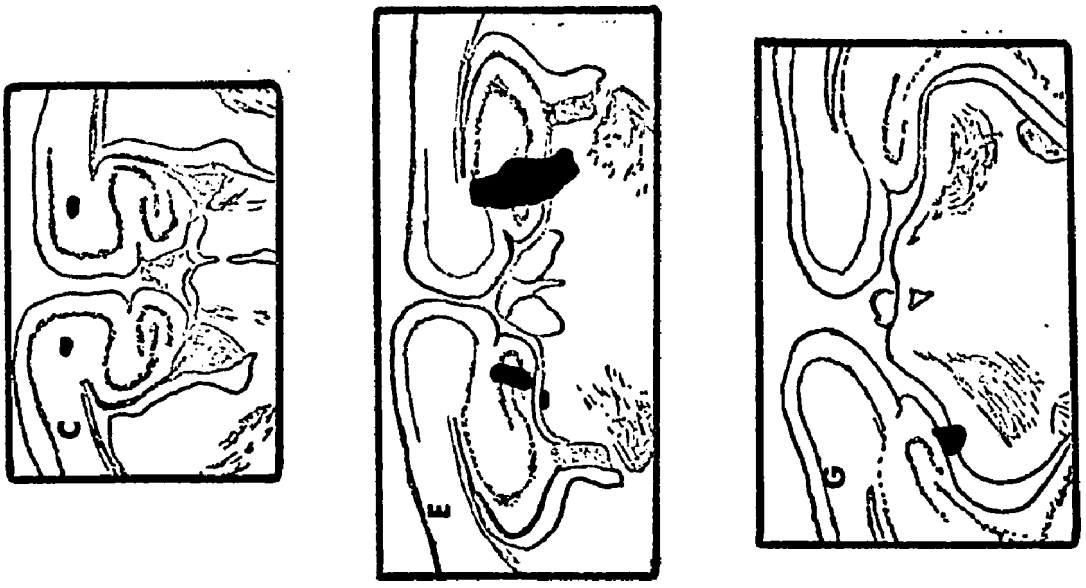

Temperature (in degrees centigrade)
5
10
20
30
40
Fed Animal 43
Lesion
Animal Temp.
Refrig. Temp.
Heart Rate
0
25
50
100
200
300
Time (in minutes)
100
200
300
400
500
600
700
800
900
Heart Rate (in beats/minute)

Fig. 26



Animal 9u

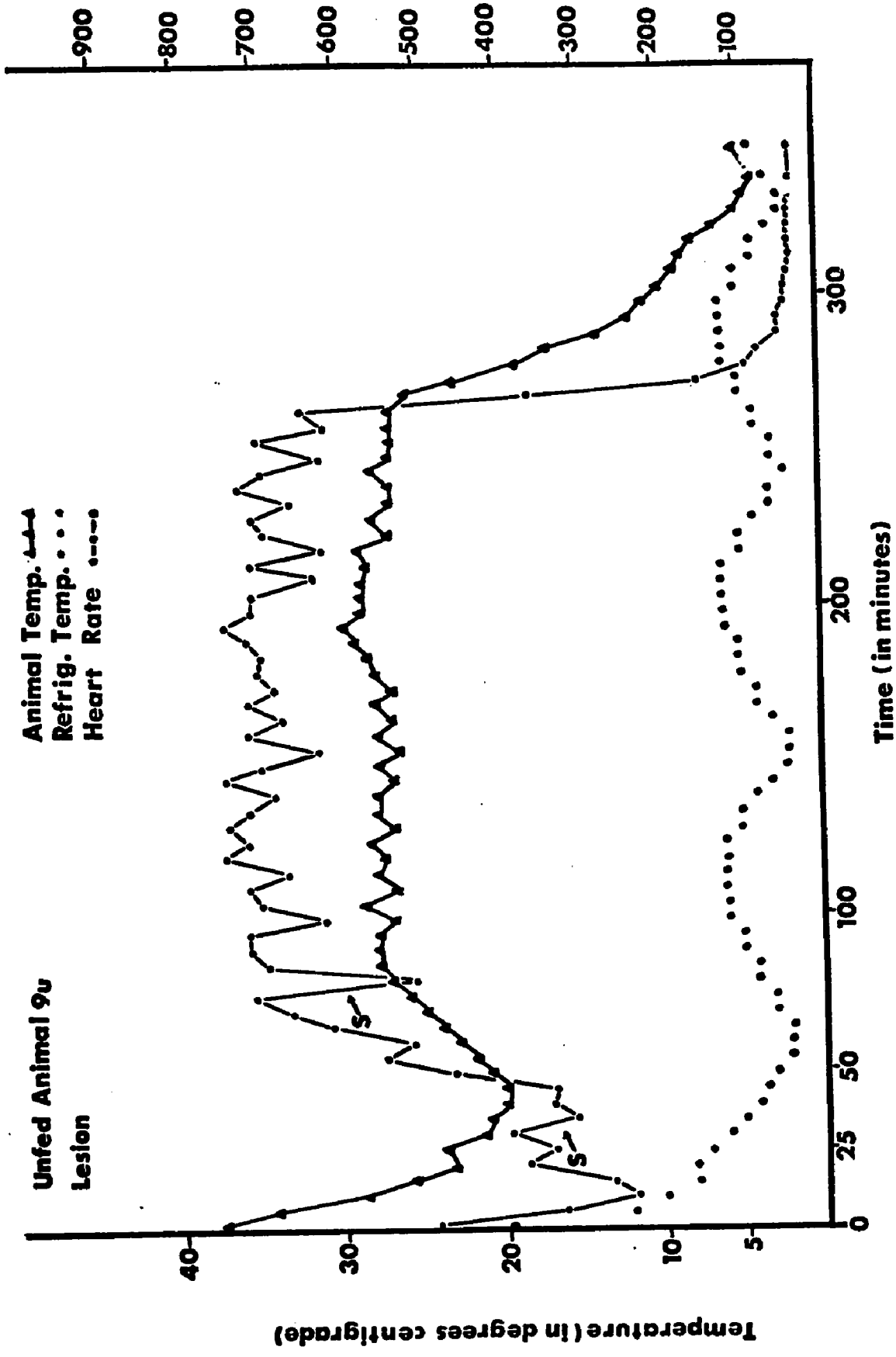
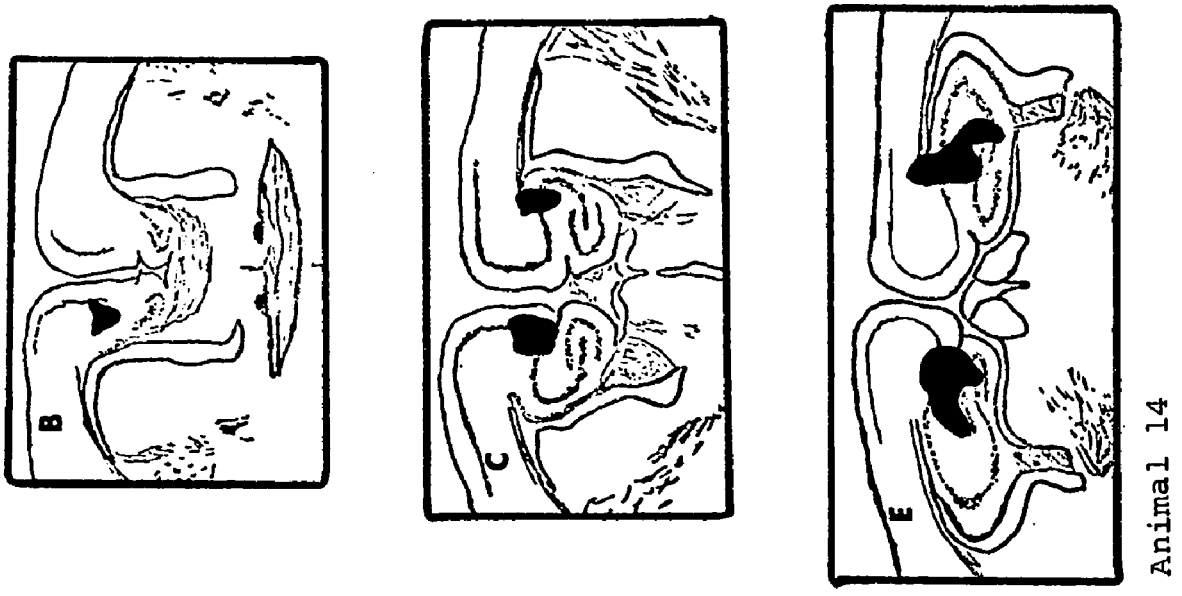

Unfed Animal 9u
Lesion
Animal Temp.
Refrig. Temp.
Heart Rate
Temperature (in degrees centigrade)
Heart Rate (in beats/minute)
Time (in minutes)
40
30
20
10
5
900
800
700
600
500
400
300
200
100
0
25
50
100
200
300
S
S

Fig.27



Animal 14

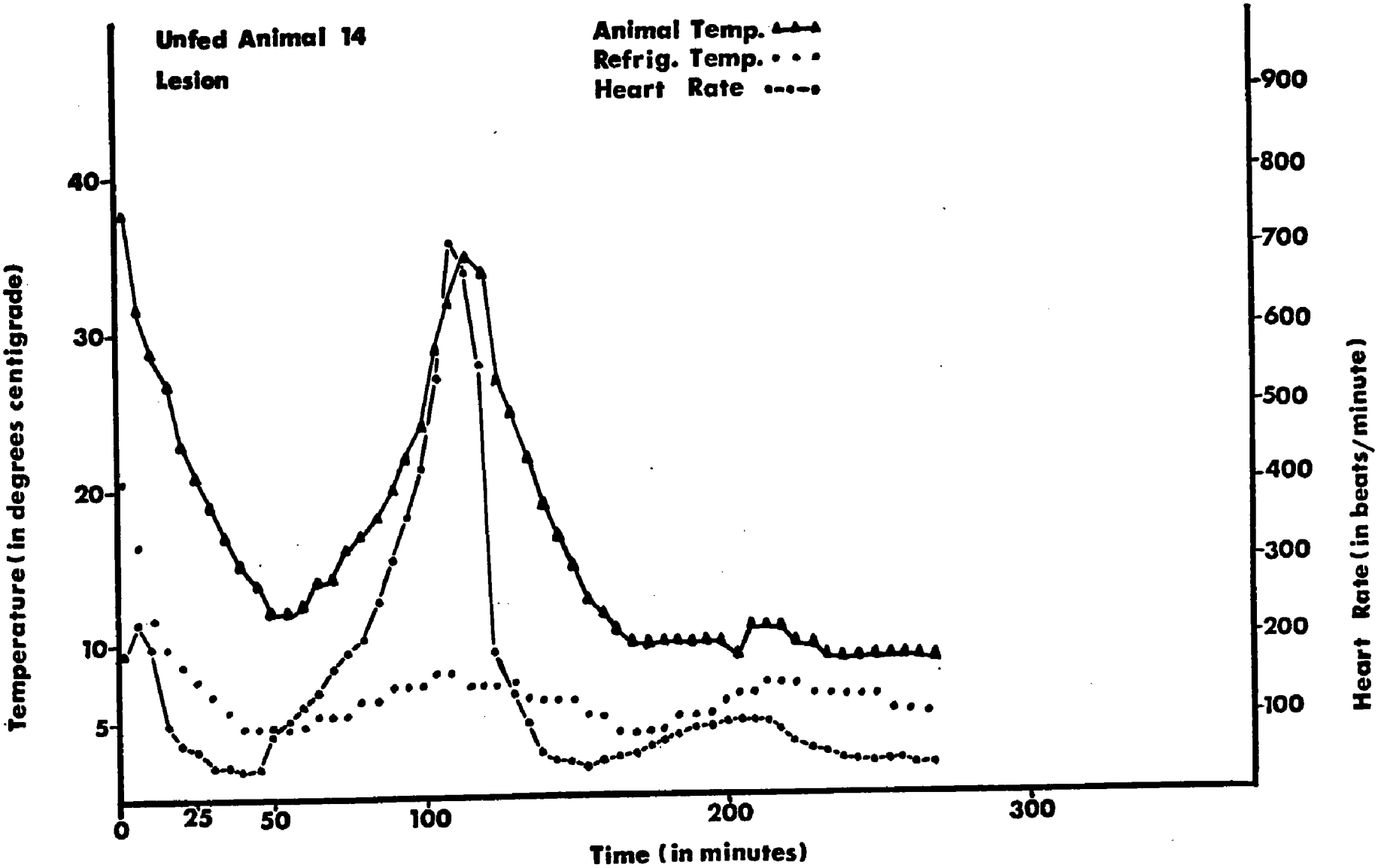
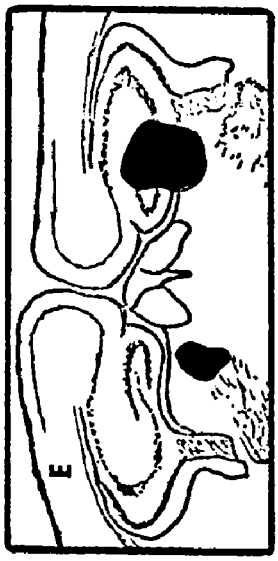
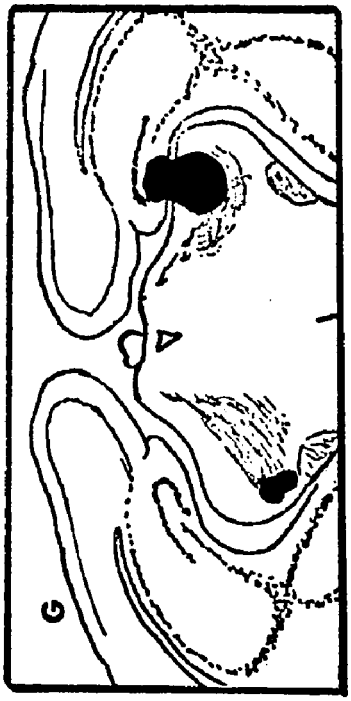

Unfed Animal 14
Lesion
Animal Temp.
Refrig. Temp.
Heart Rate
Temperature (in degrees centigrade)
40
30
20
10
5
0
25
50
100
200
300
Time (in minutes)
900
800
700
600
500
400
300
200
100
Heart Rate (in beats/minute)

Fig. 28





Animal 24u

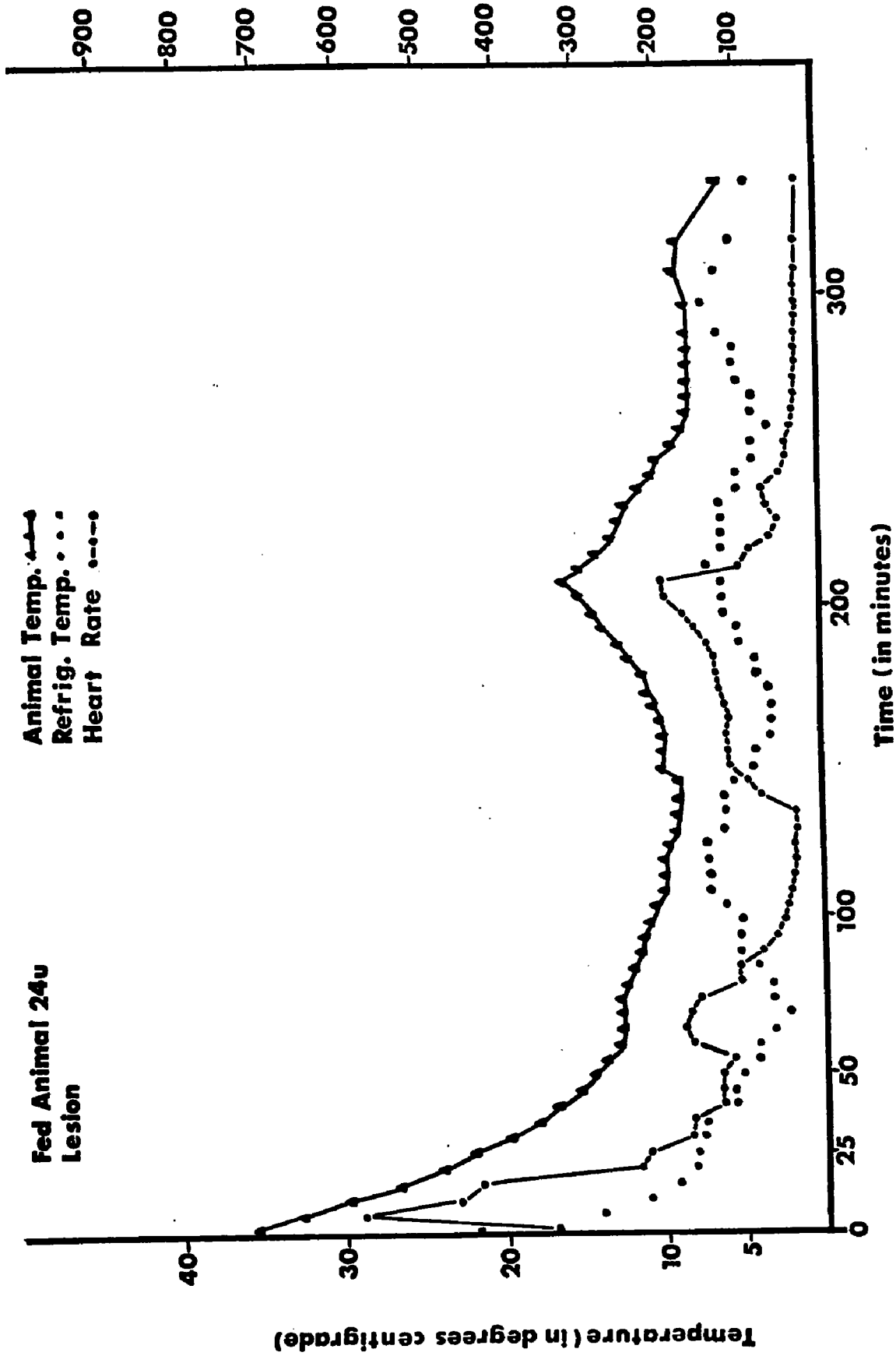
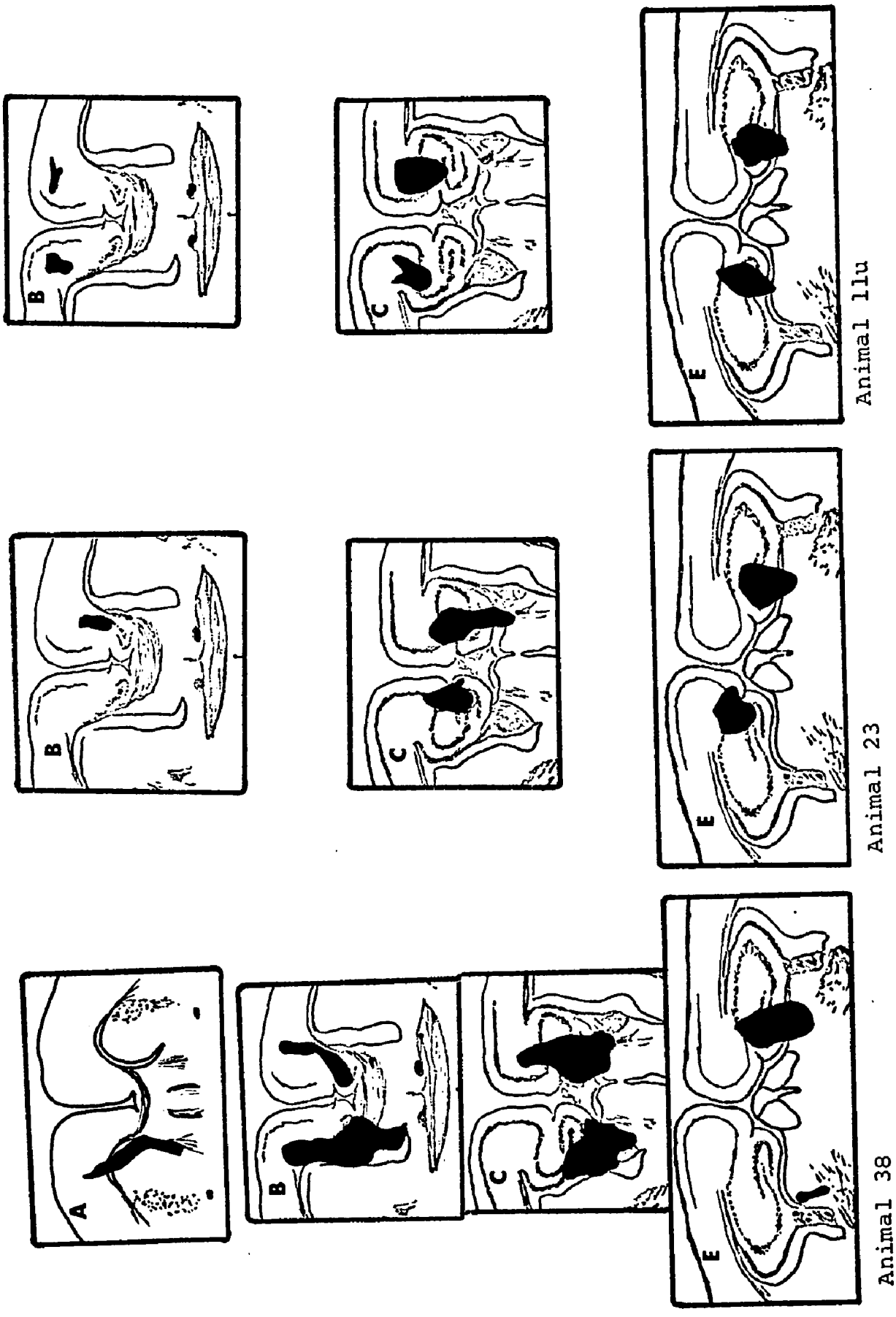

Fed Animal 24u
Lesion
Animal Temp.
Refrig. Temp.
Heart Rate
Temperature (in degrees centigrade)
Heart Rate (in beats/minute)
Time (in minutes)
40
30
20
10
5
900
800
700
600
500
400
300
200
100
0
25
50
100
200
300

Fig.29


A
B
C
E
Animal 38
Animal 23
Animal 11u

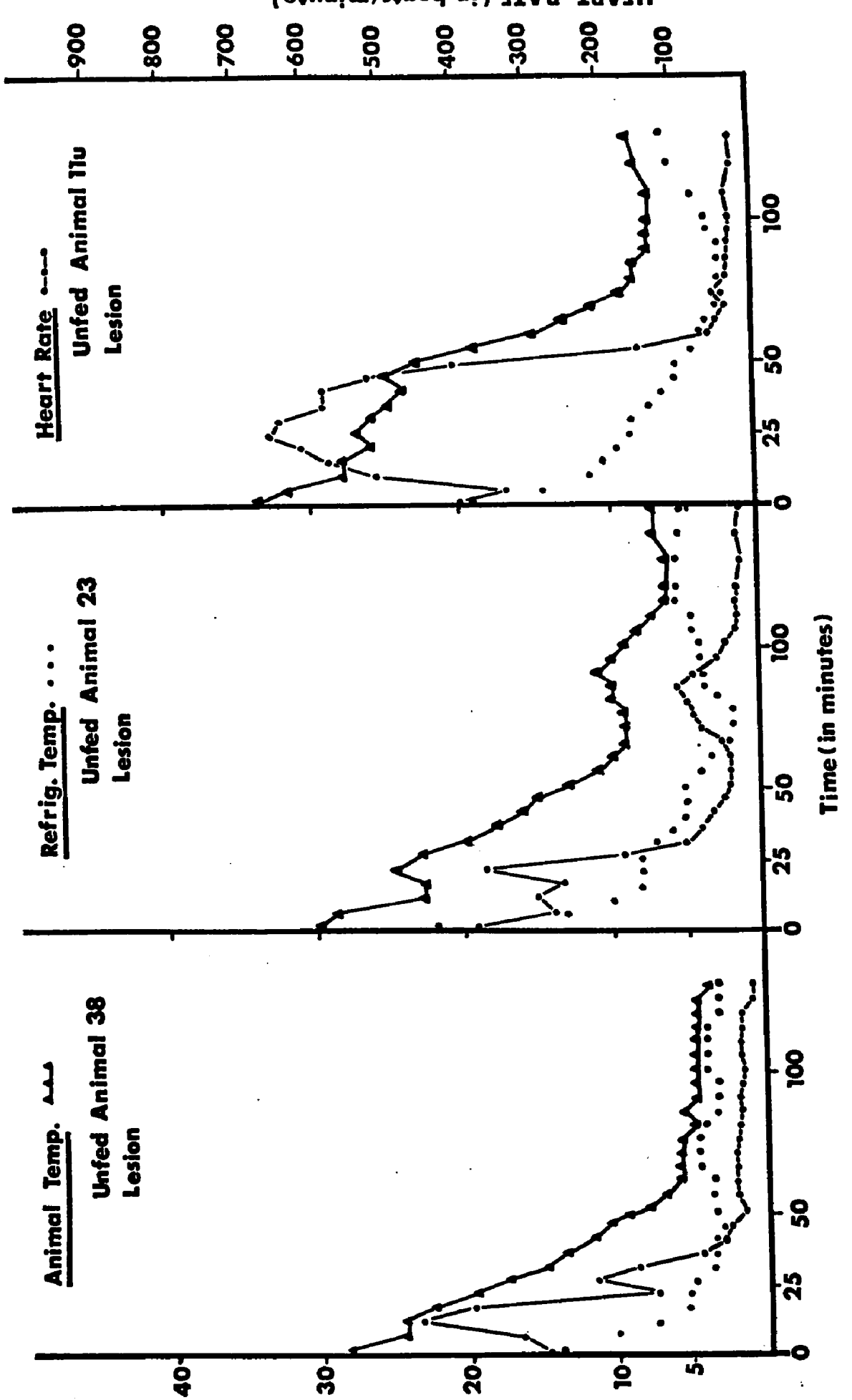
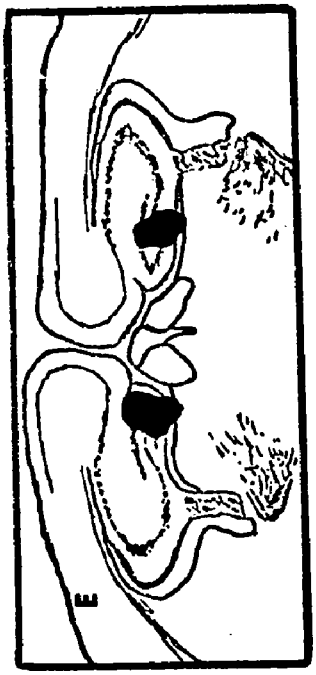

Animal Temp. ▲▲▲
Unfed Animal 38
Lesion
Refrig. Temp. . . .
Unfed Animal 23
Lesion
Heart Rate ----
Unfed Animal 11u
Lesion
TEMPERATURE (in degrees centigrade)
HEART RATE (in beats/minute)
Time (in minutes)
40
30
20
10
5
900
800
700
600
500
400
300
200
100
0
25
50
100

Fig.30



Animal 21u

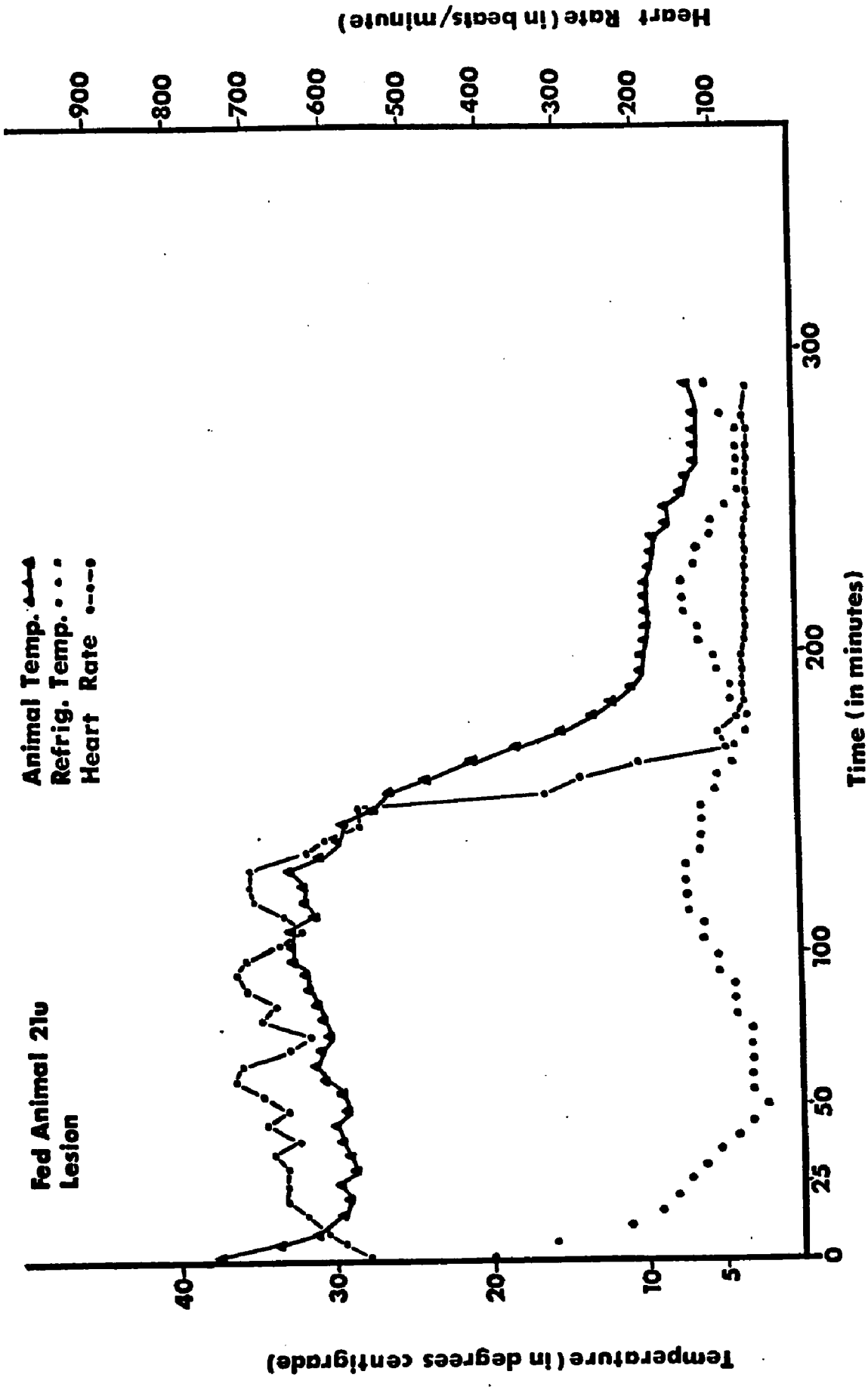
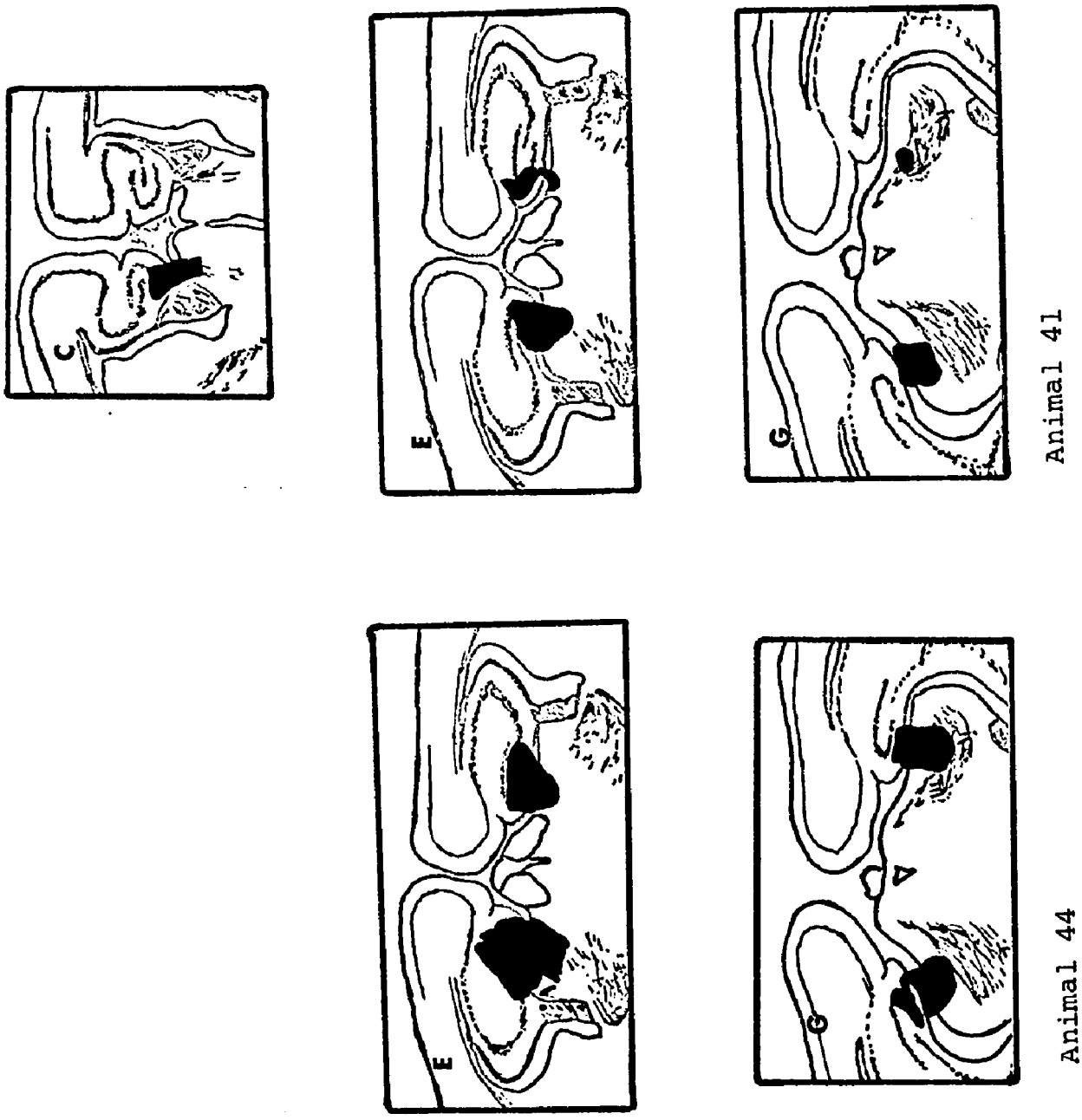

Red Animal 21u
Lesion
Animal Temp.
Refrig. Temp.
Heart Rate
Temperature (in degrees centigrade)
Heart Rate (in beats/minute)
Time (in minutes)
40
30
20
10
5
900
800
700
600
500
400
300
200
100
0
25
50
100
200
300

Fig.31


C
E
G
Animal 41
E
G
Animal 44

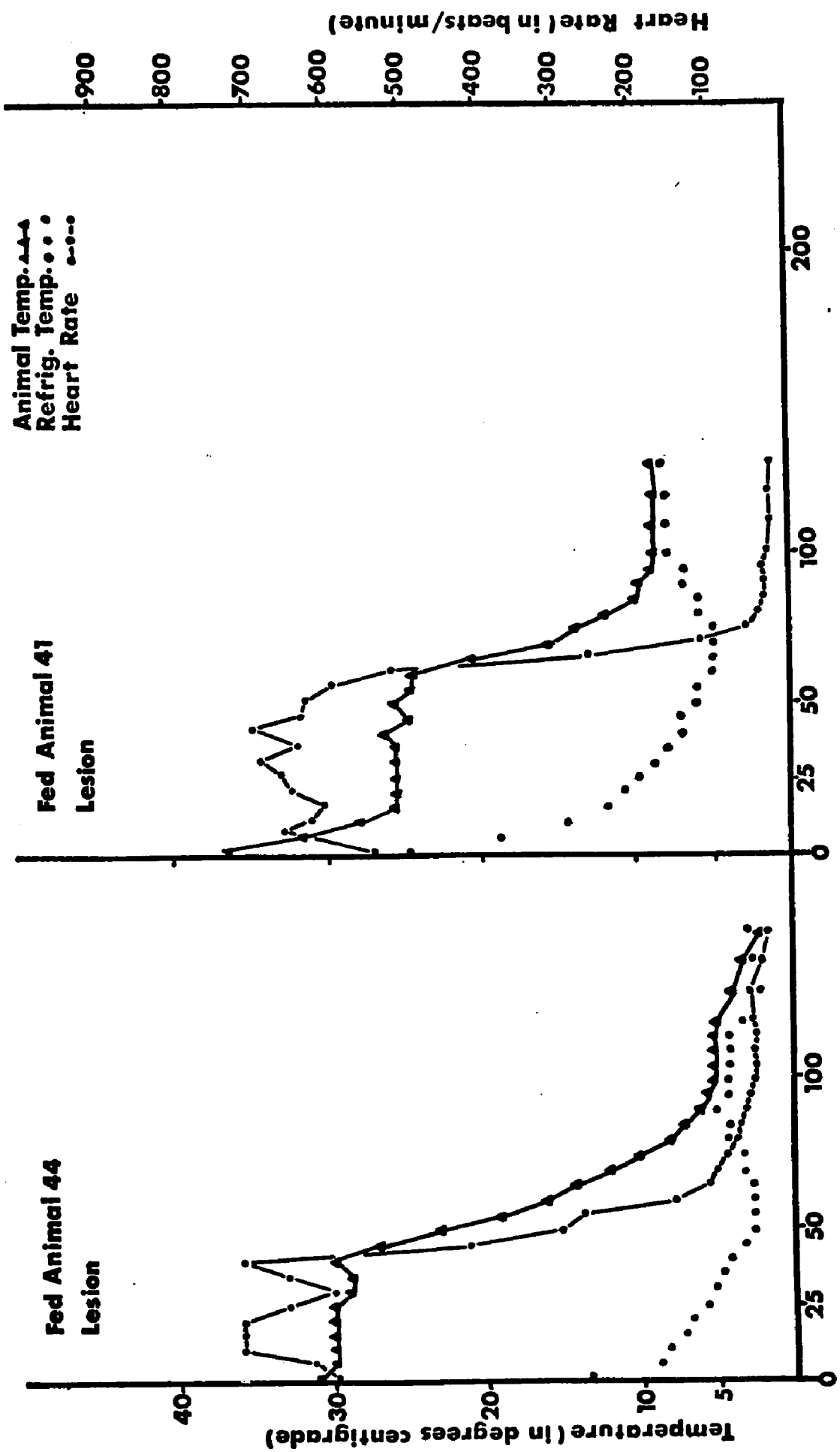
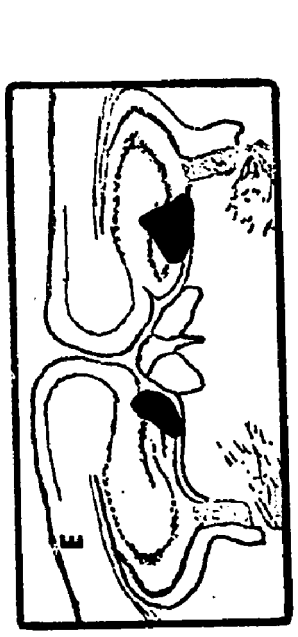
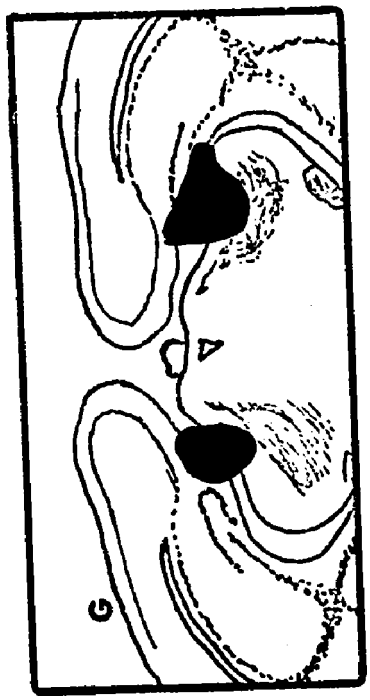

Fed Animal 44
Lesion
Fed Animal 41
Lesion
Animal Temp.
Refrig. Temp.
Heart Rate
Temperature (in degrees centigrade)
Heart Rate (in beats/minute)
Time (in minutes)
40
30
20
10
5
900
800
700
600
500
400
300
200
100
0
25
50
100
200

Fig.32





Animal 39u

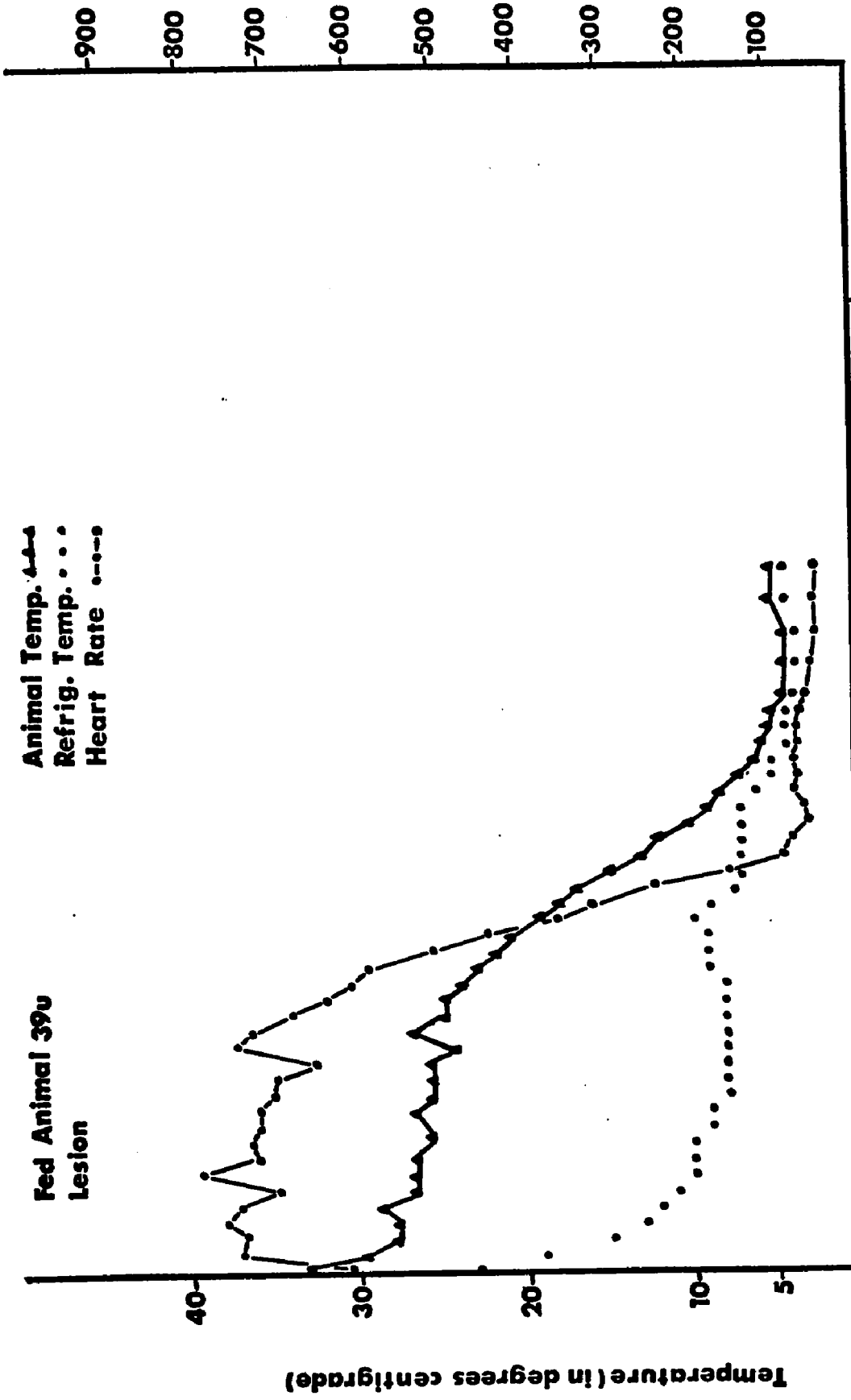
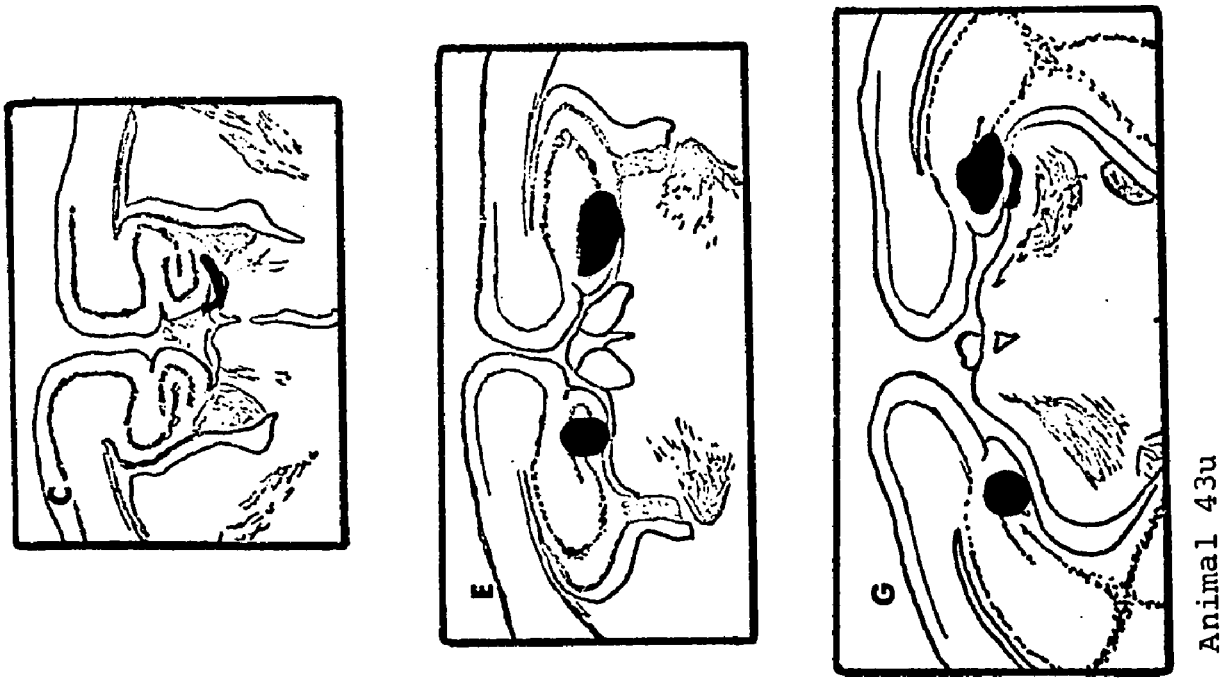

Fed Animal 39u
Lesion
Animal Temp.
Refrig. Temp.
Heart Rate
Temperature (in degrees centigrade)
Heart Rate (in beats/minute)
Time (in minutes)
40
30
20
10
5
900
800
700
600
500
400
300
200
100
0
25
50
100
200
300

Fig.33



Animal 43u

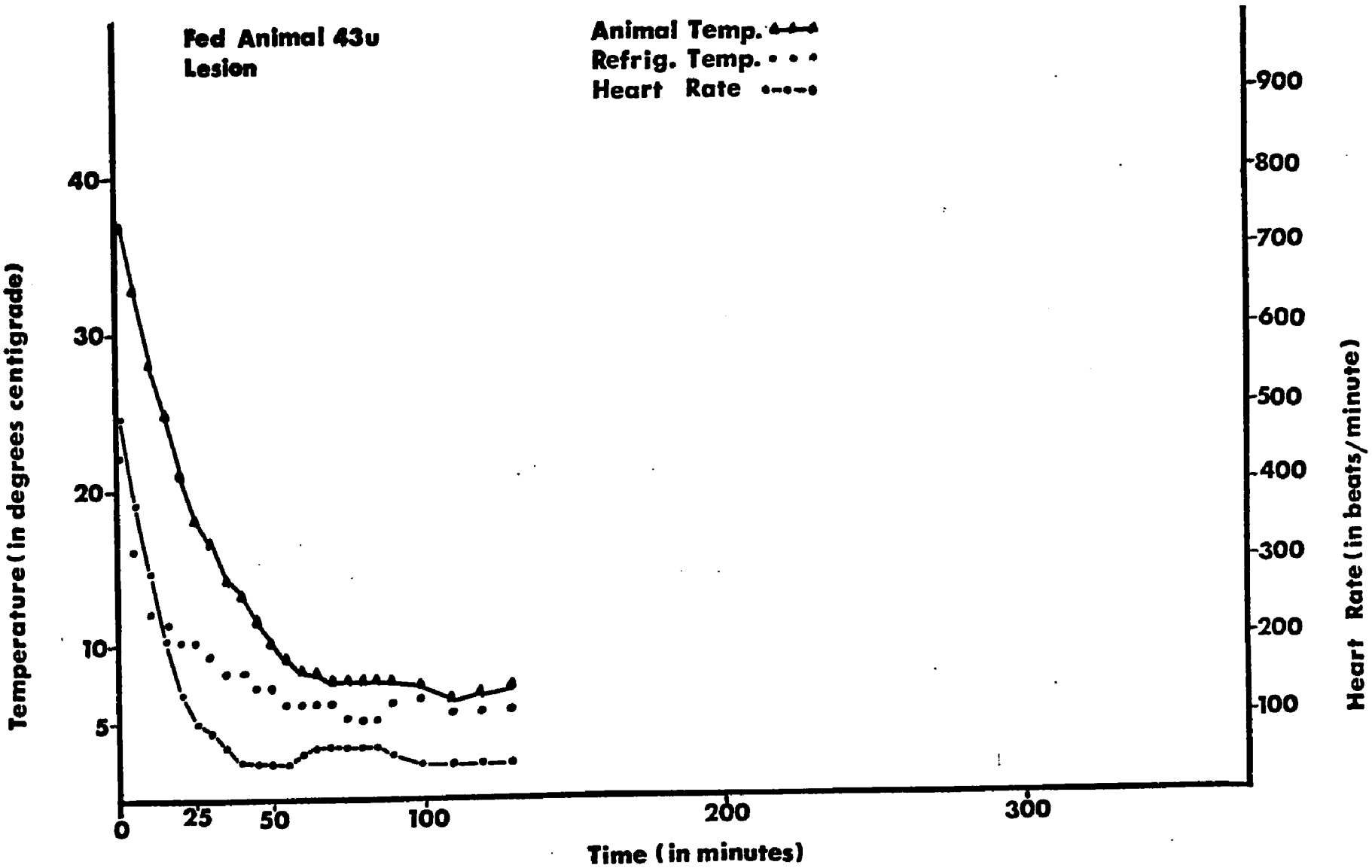
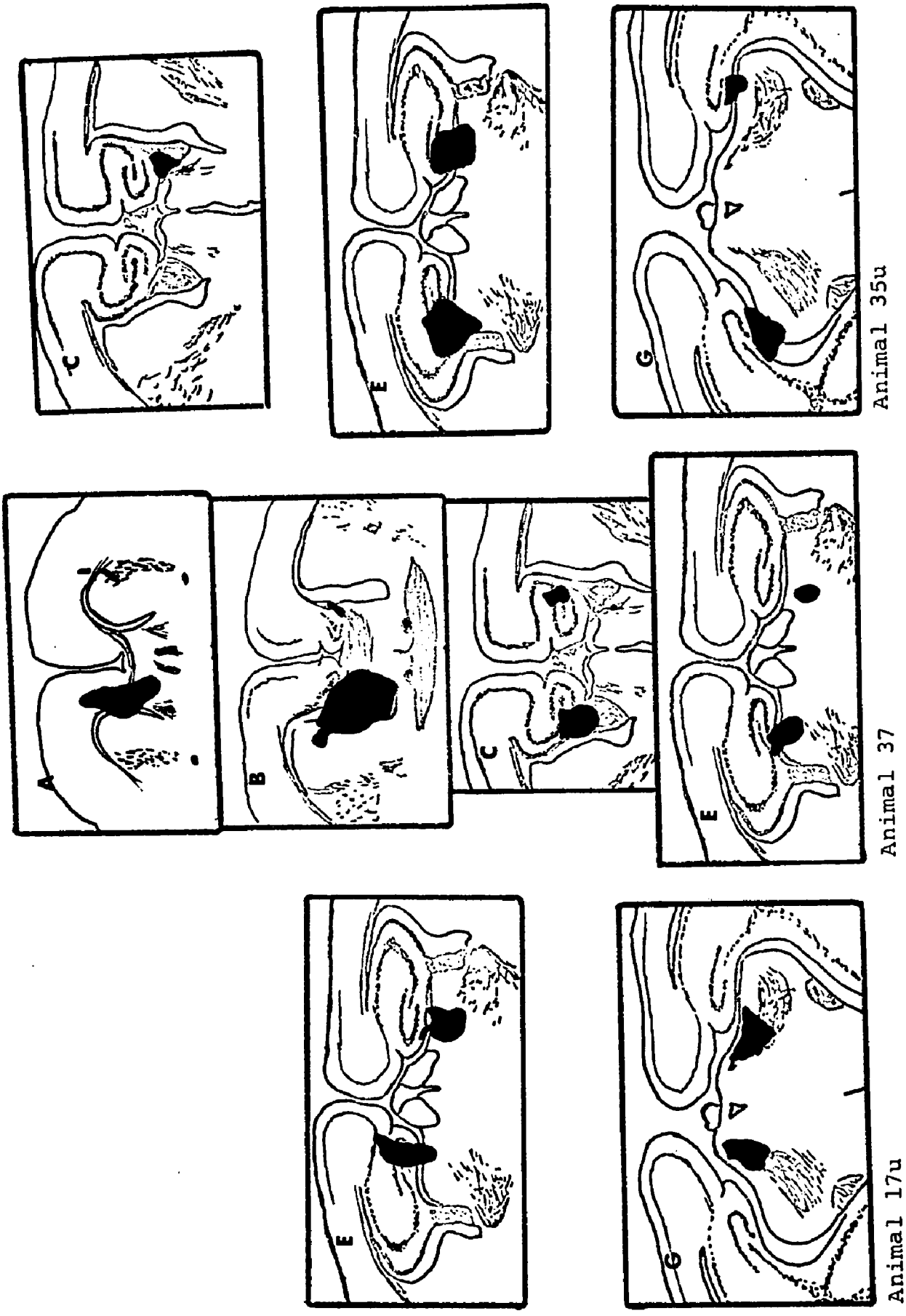

Fed Animal 43u
Lesion
Animal Temp.
Refrig. Temp.
Heart Rate
Temperature (in degrees centigrade)
40
30
20
10
5
0
25
50
100
200
300
Time (in minutes)
900
800
700
600
500
400
300
200
100
Heart Rate (in beats/minute)

Fig. 34


C
E
G
Animal 35u
A
B
C
E
Animal 37
E
G
Animal 17u

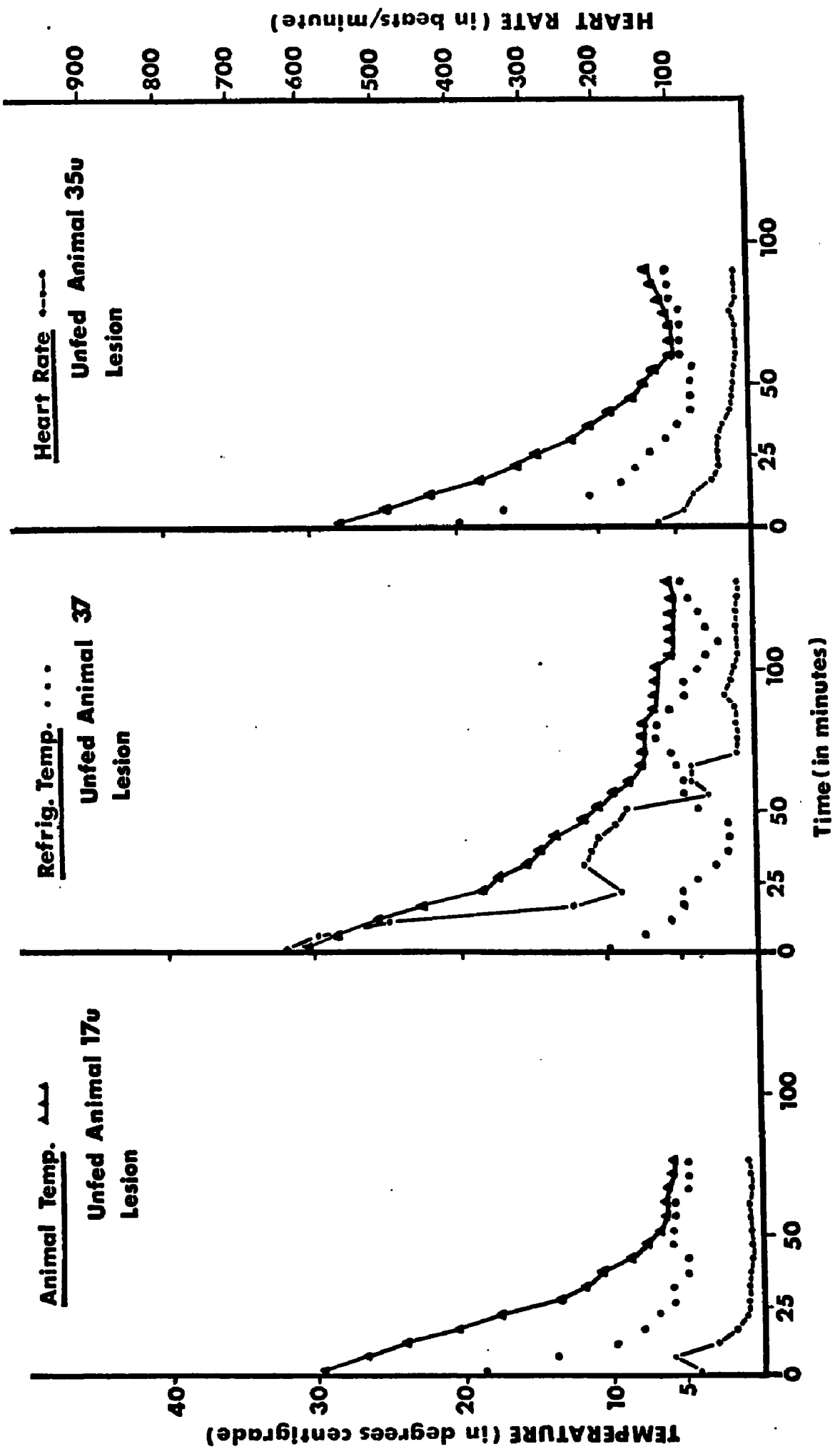
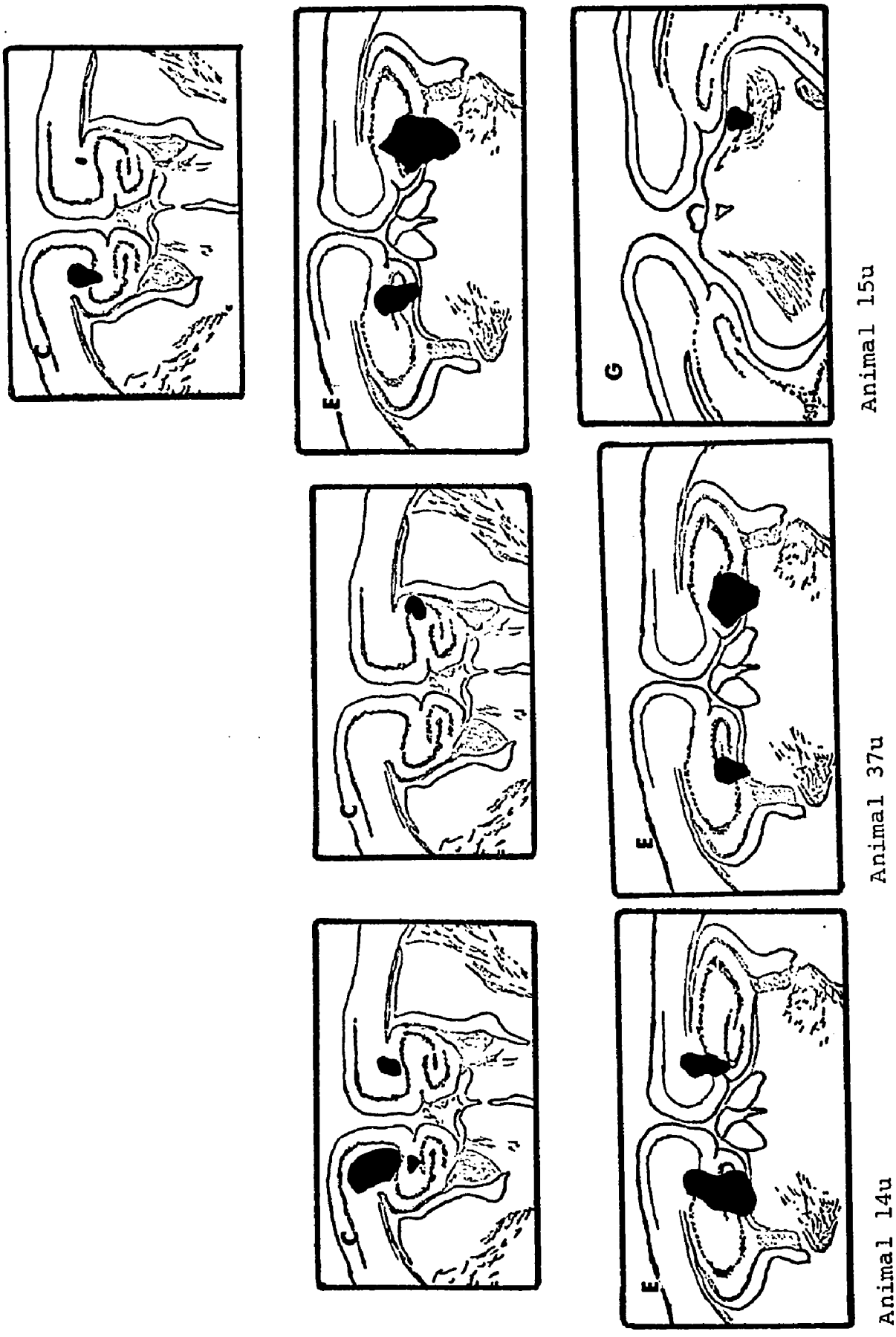

Animal Temp. ▲▲▲
Unfed Animal 17u
Lesion
Refrig. Temp. • • •
Unfed Animal 37
Lesion
Heart Rate •—•
Unfed Animal 35u
Lesion
TEMPERATURE (in degrees centigrade)
HEART RATE (in beats/minute)
Time (in minutes)

Fig.35


C
E
G
A
Animal 14u
Animal 37u
Animal 15u

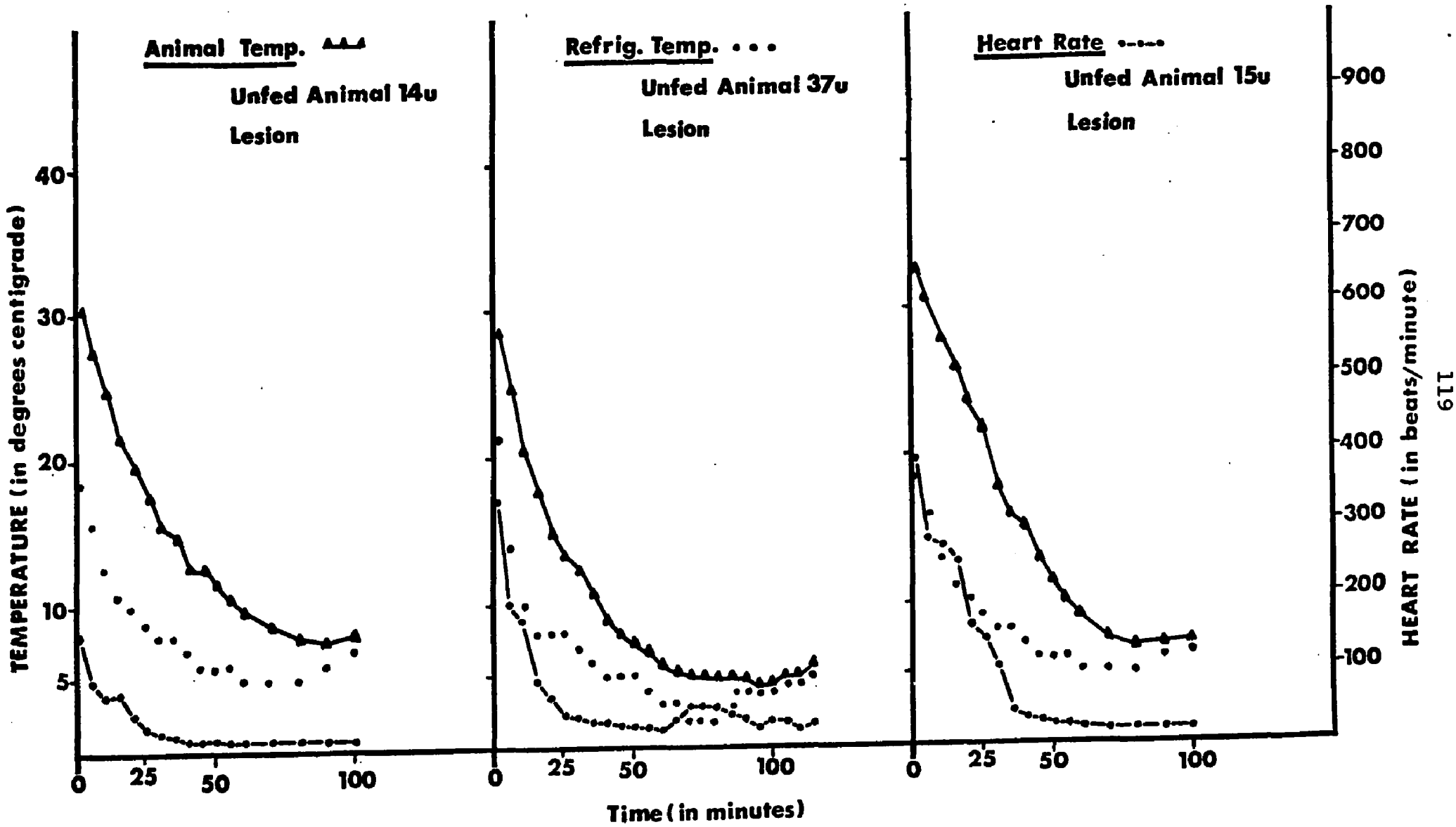

Animal Temp. ▲-▲-▲
Unfed Animal 14u
Lesion
Refrig. Temp. • • •
Unfed Animal 37u
Lesion
Heart Rate •-•-•
Unfed Animal 15u
Lesion
TEMPERATURE (in degrees centigrade)
HEART RATE (in beats/minute)
Time (in minutes)
40
30
20
10
5
900
800
700
600
500
400
300
200
100
0
25
50
100

Fig. 36

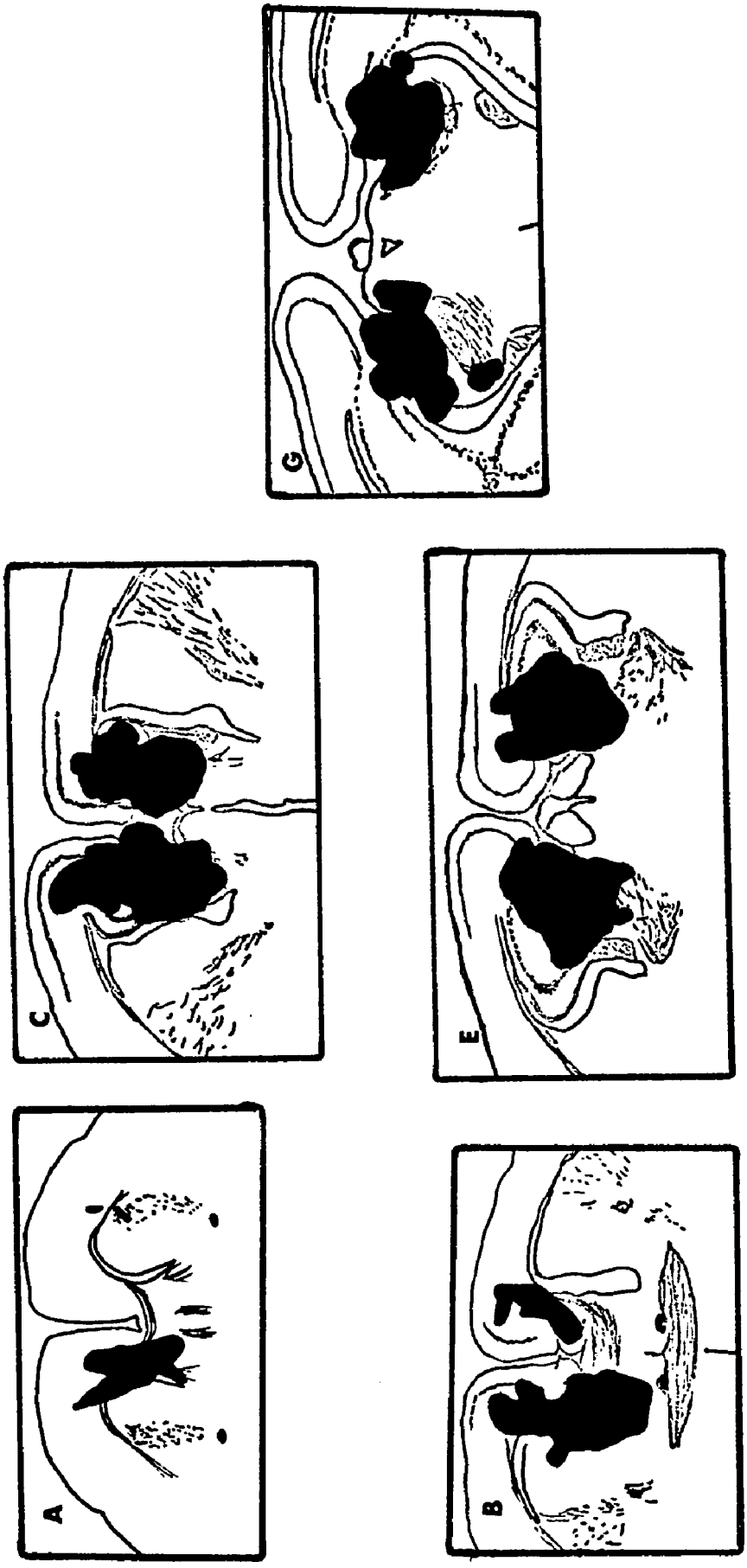


Fig.37 Brain tracings showing the location of the lesions of the lesioned group of animals.

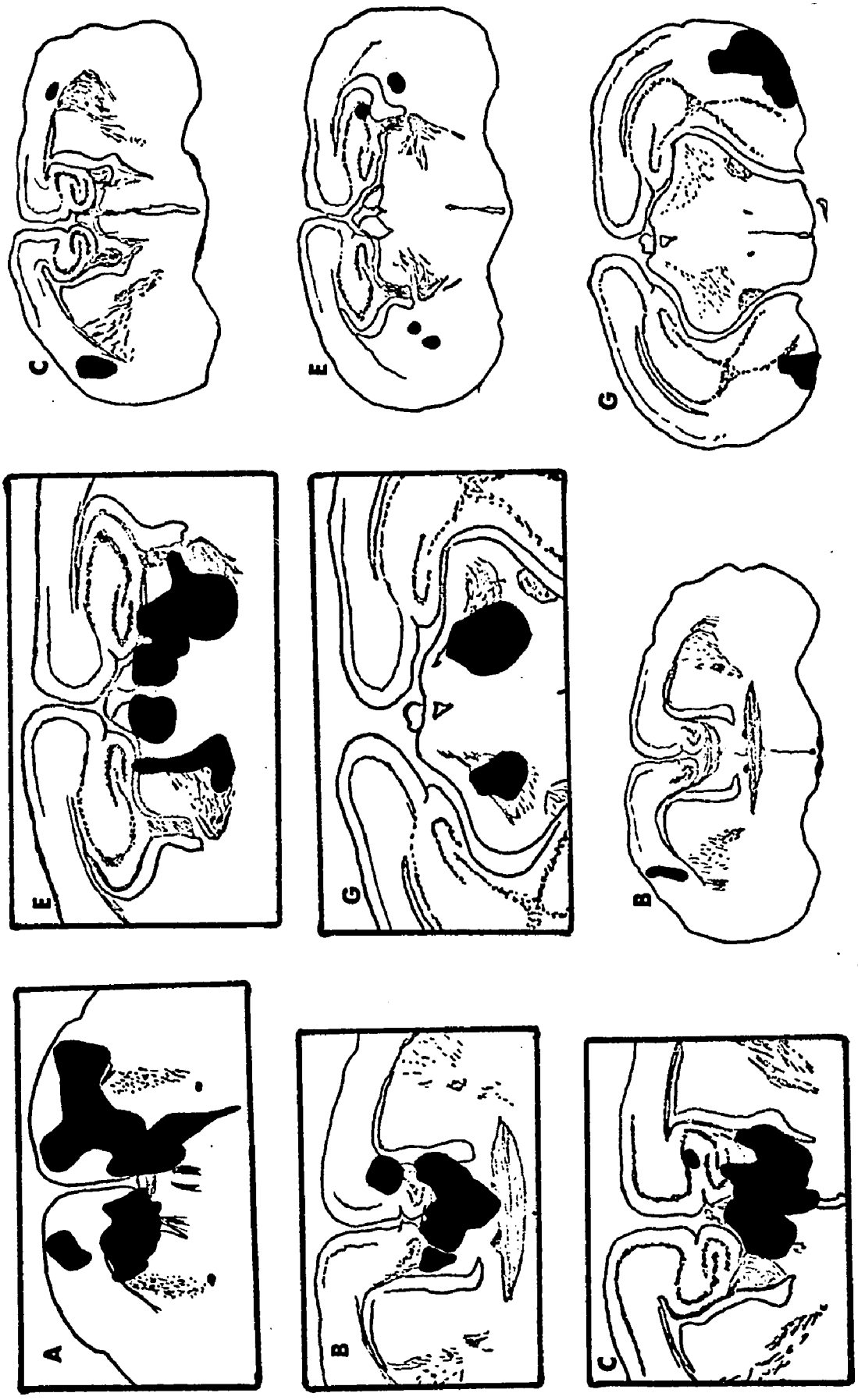


Fig.38 Brain tracings showing the location of the lesions of the lesioned control group of animals.

APPENDIX

An Atlas of the Brain of the Little Brown Bat, *Myotis lucifugus lucifugus*, in Transverse Section.[1]

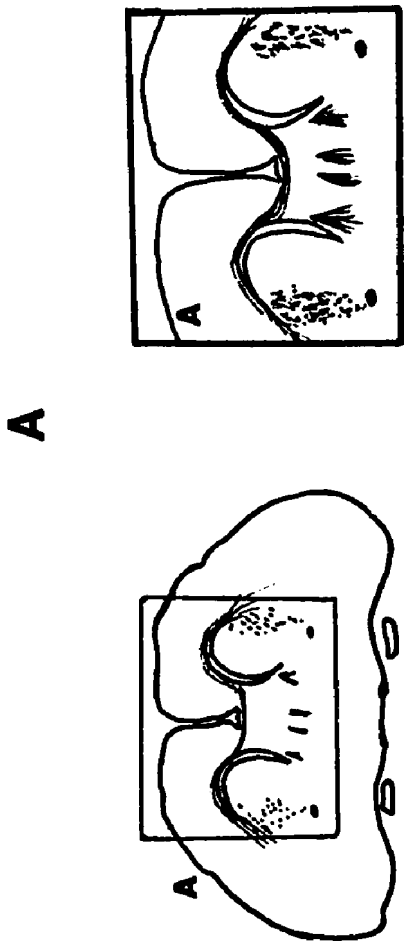
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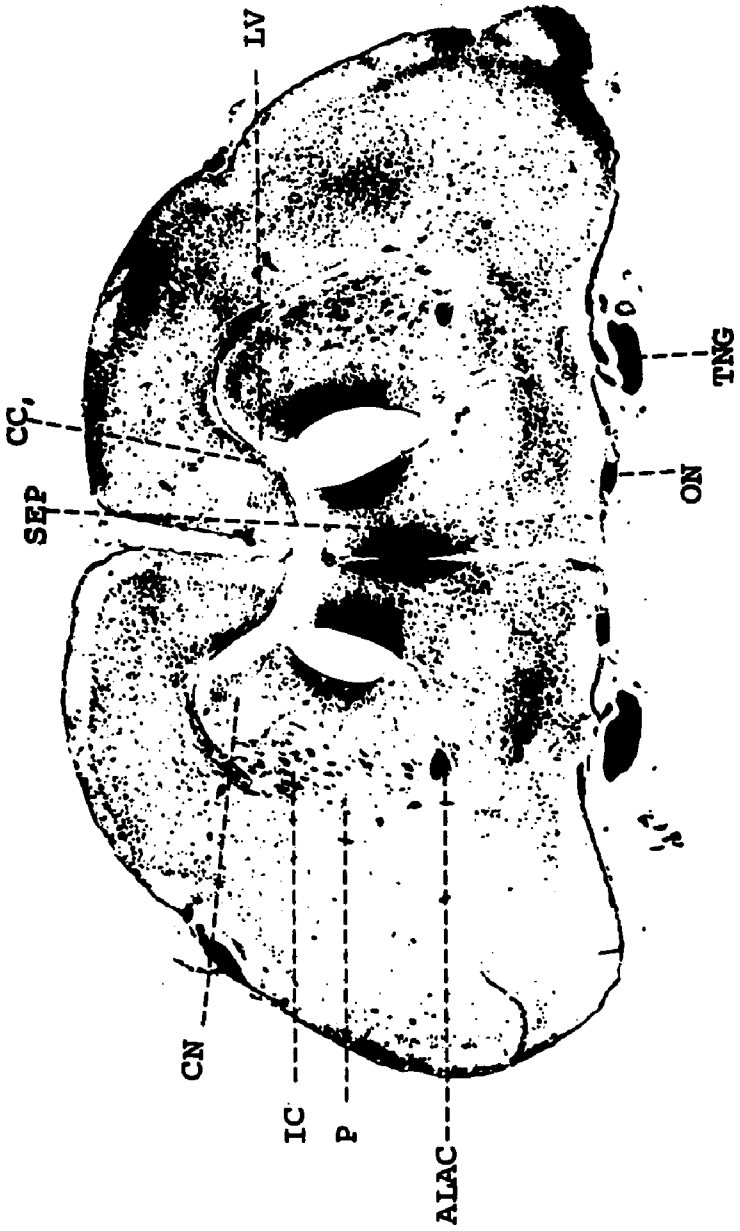
[1] I would like to thank Dr. James Koski, Jr. for allowing me to reproduce part of his atlas of the brain of the little brown bat, *Myotis lucifugus lucifugus*.

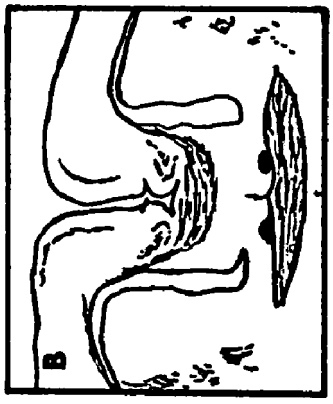
LIST OF ABBREVIATIONS

AC --- anterior commissure
ALAC --- anterior limb of the anterior commissure
AMY --- amygdala
CC_1 --- corpus callosum
CC_2 --- crus cerebri
CN --- caudate nucleus
DEN --- dentate gyrus
F --- fimbria
F_4 --- fornix
HAB --- habenular nucleus
HIP --- Ammon's horn
HIP + DEN --- hippocampus
HYP --- hypophysis
IC --- internal capsule
IFS --- infundibular stalk
LH --- lateral hypothalamus
LN --- lenticular nucleus
LPO --- lateral preoptic area
LV --- lateral ventricle
MH --- medial hypothalamus
MPO --- medial preoptic area
MT --- mammillothalamic tract
OC --- optic chiasm
ON --- optic nerve
OT --- optic tract
P --- putamen
PC --- posterior commissure
PG --- periventricular gray
PIN --- pineal gland
RF --- rhinal fissure
SC --- superior colliculus
SEP --- septal area
SM --- stria medullaris
TC --- tuber cinereum
THA --- thalamus
TNG --- trigeminal nerve and ganglion
TV --- third ventricle

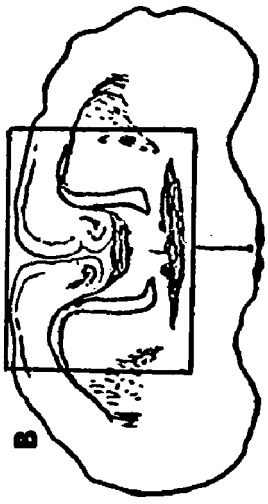


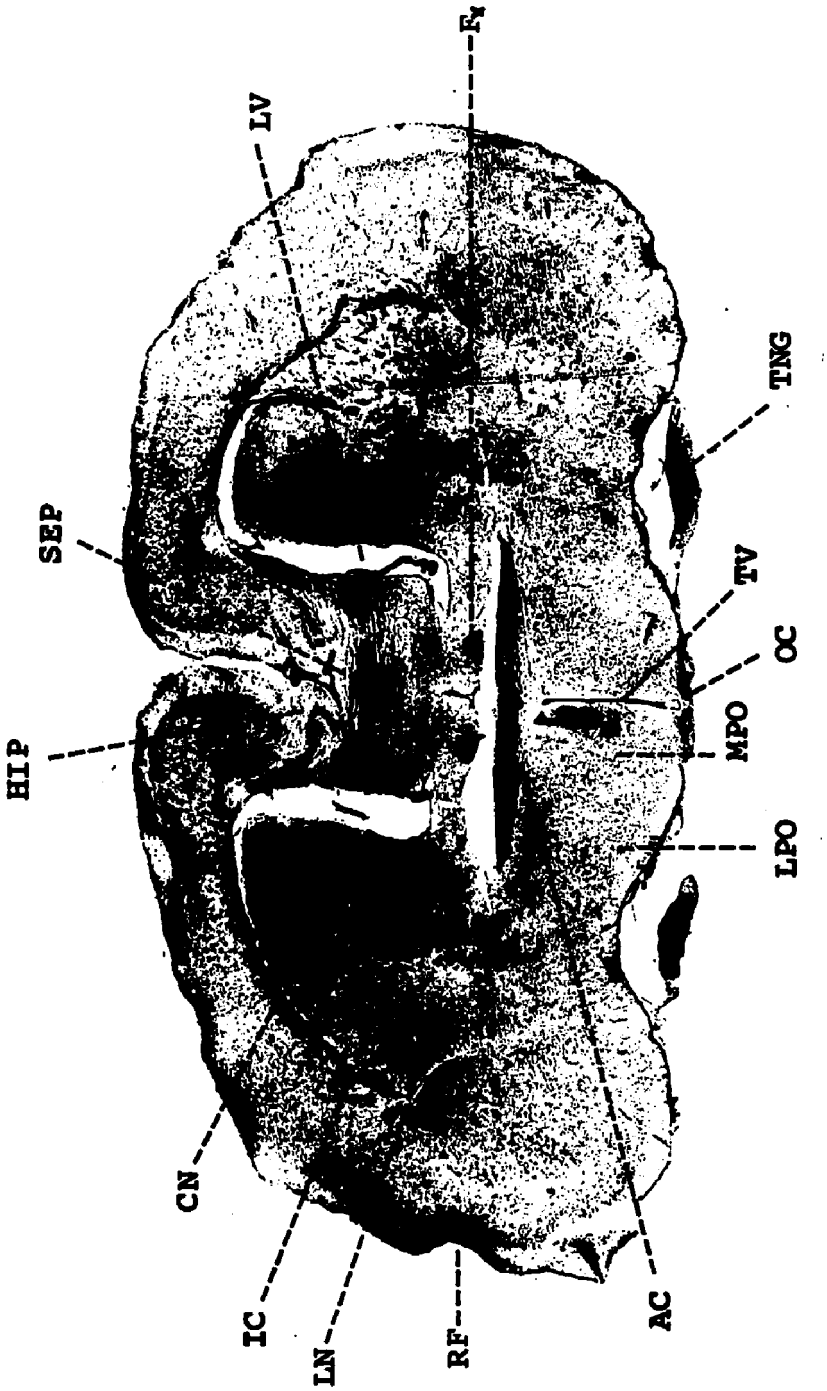

A
A


LV
CC
SEP
TNG
ON
CN
IC
P
ALAC



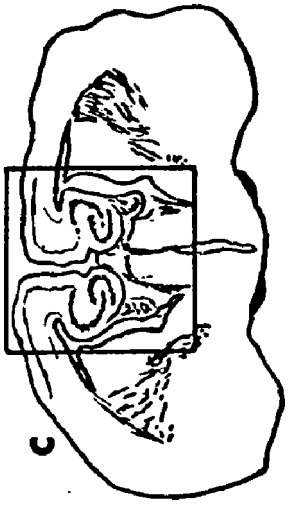
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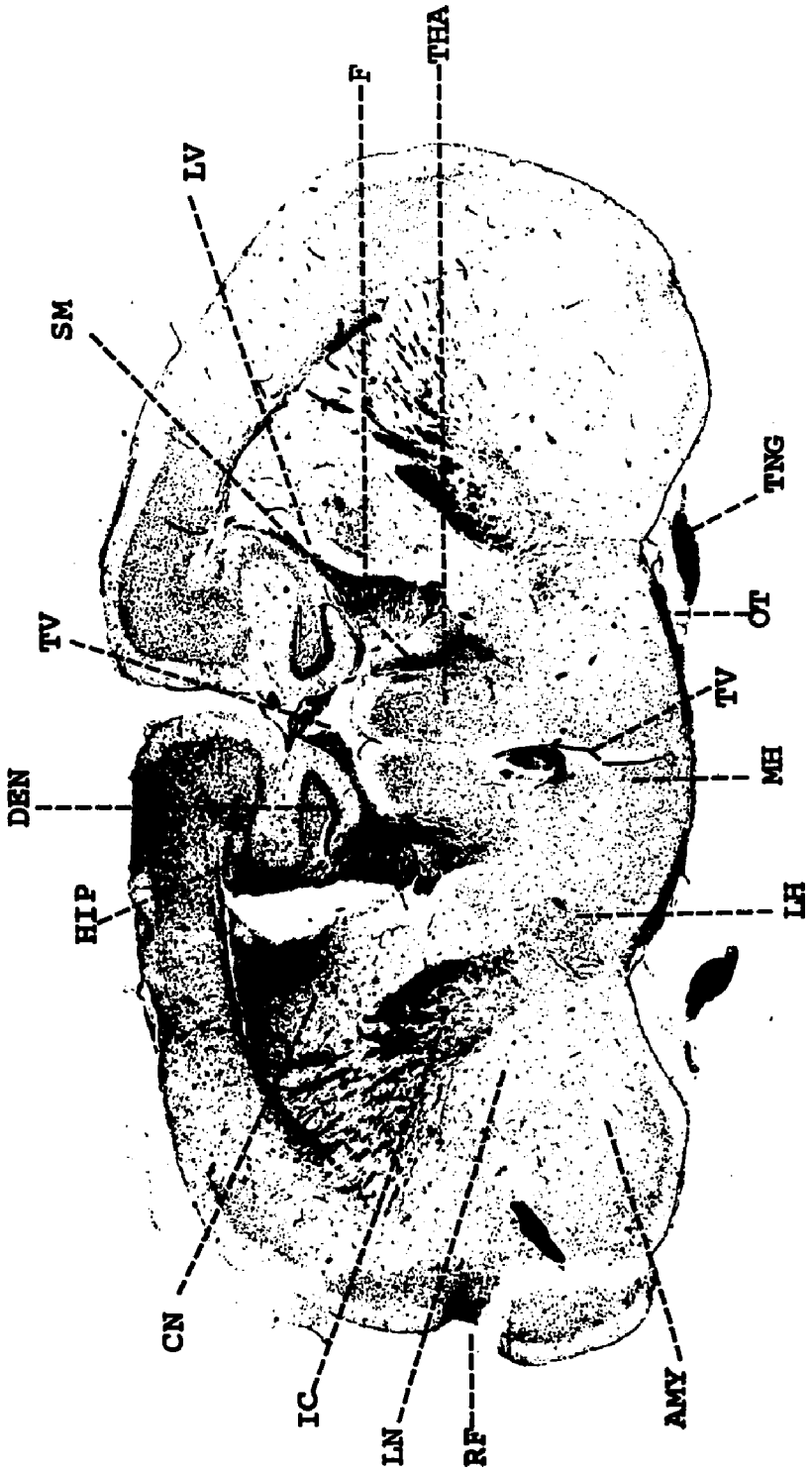


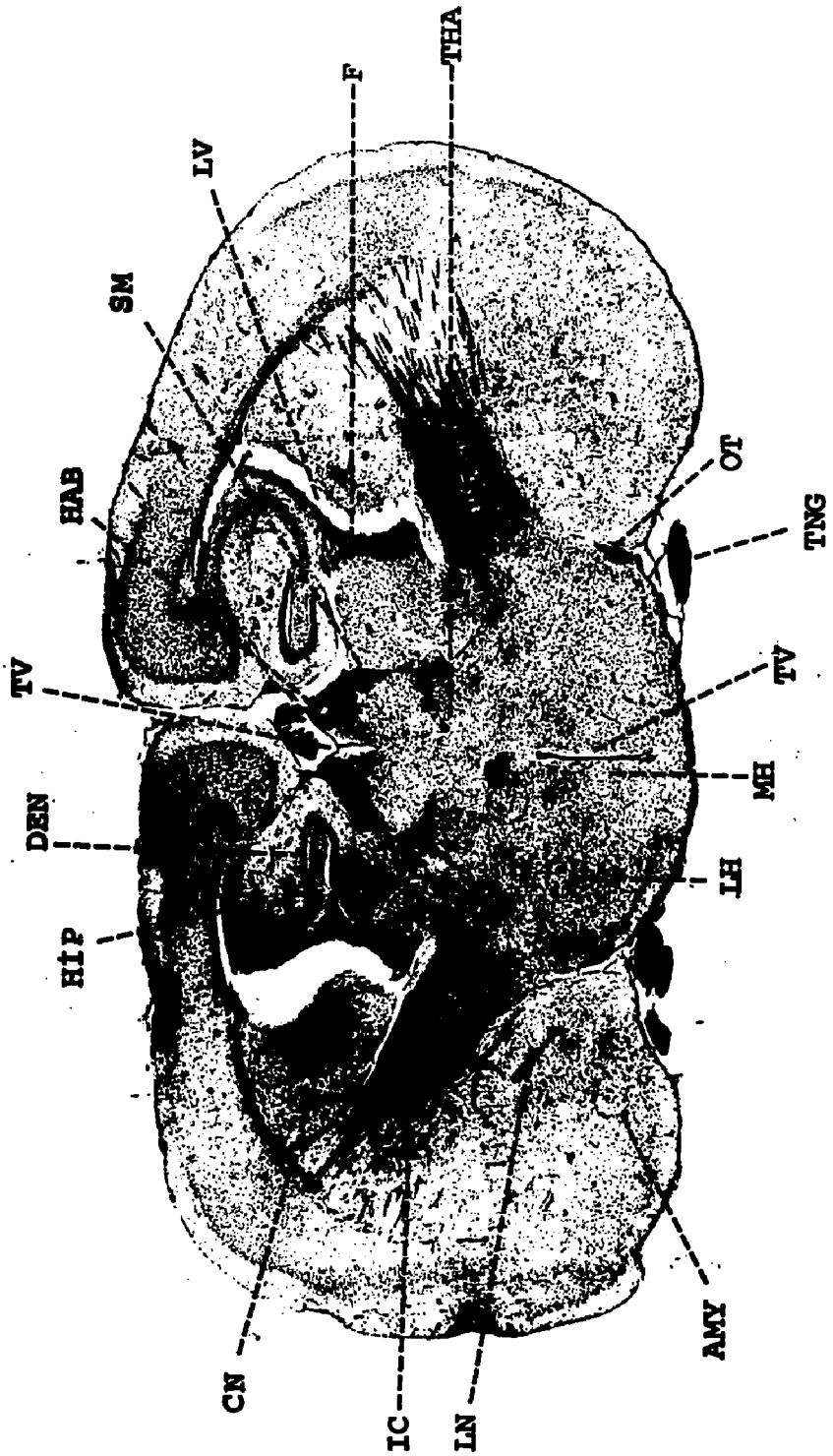

F4
LV
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OC
HIP
MPO
LPO
CN
IC
LN
RF
AC

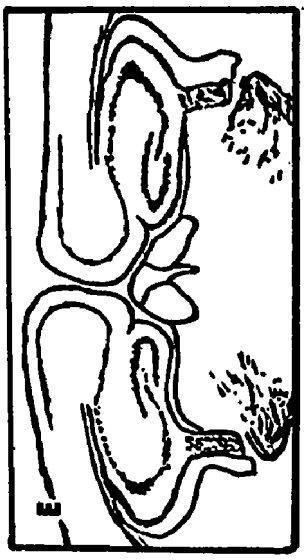


C

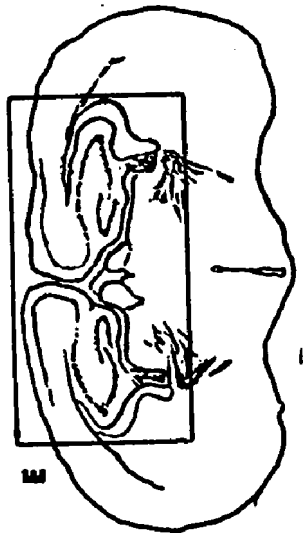


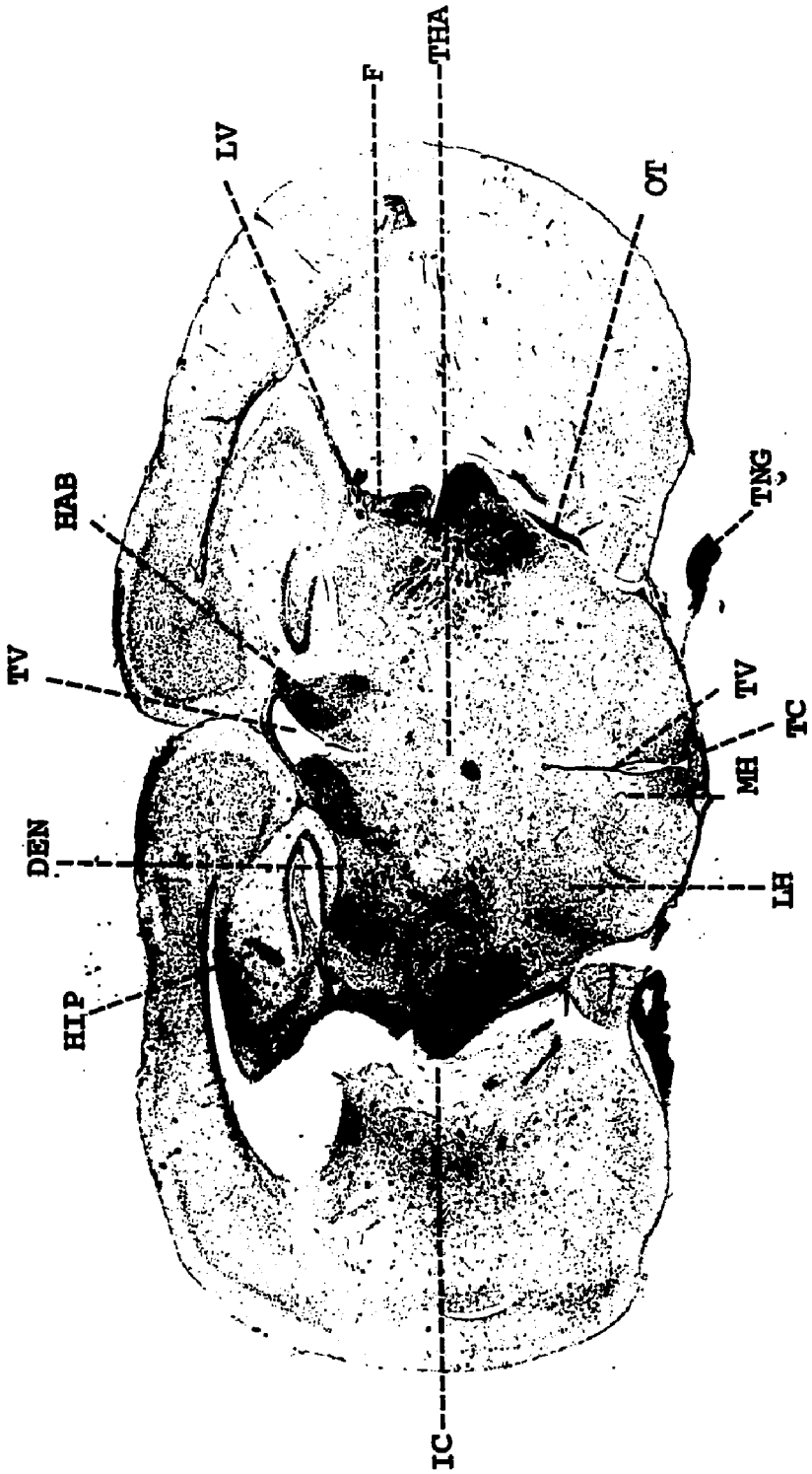

THA
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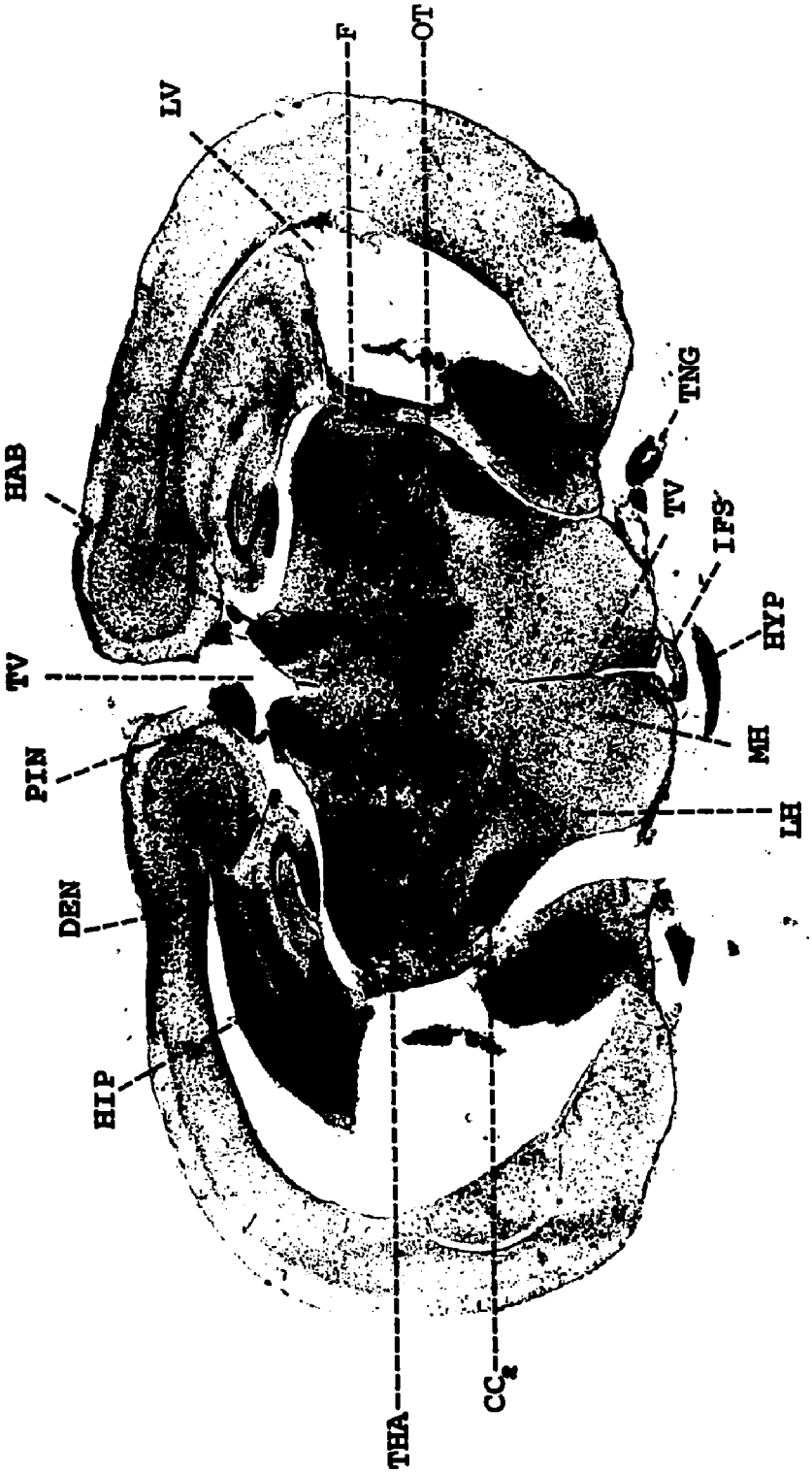

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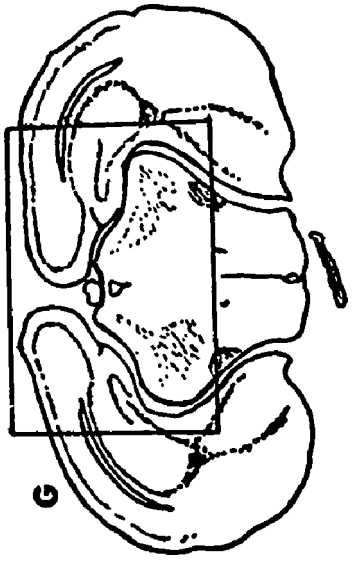


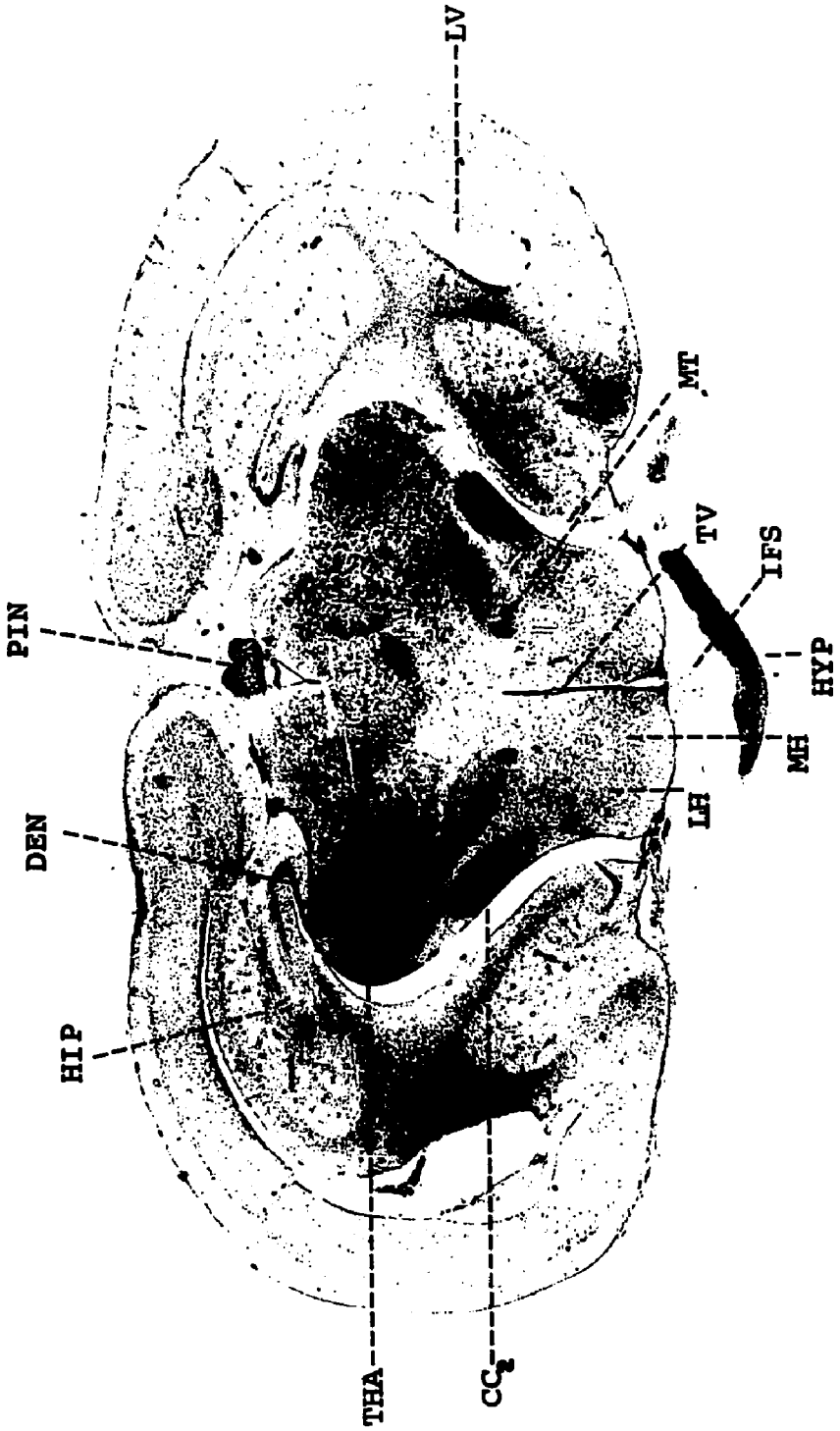

THA
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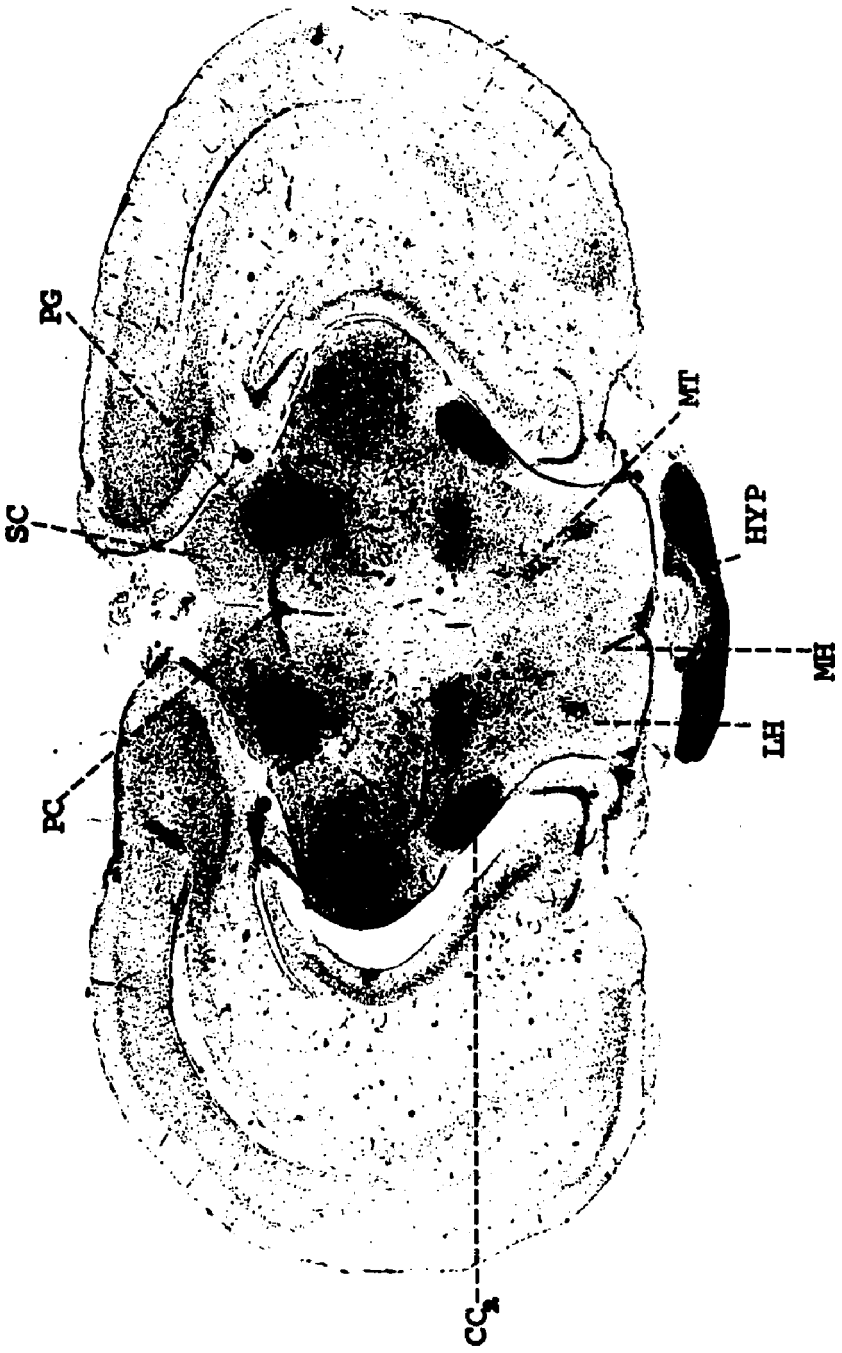

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HIP
THA
CC



G




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HIP
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MB
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PC
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