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The Cortical Destruction Necessary to Produce a Transfer of a Forced Practice Function

Patricia E. Barnett

CORTICAL DESTRUCTION - BARNETT

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A Thesis

Submitted in Partial Fulfillment of the

Requirements for the Degree of Master of Science

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This thesis, directed and approved by the candidate's committee, has been accepted by the Graduate Committee of the University of New Mexico in partial fulfillment of the requirements for the degree of

MASTER OF SCIENCE

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Introduction

One of the major problems of psychology concerns the possibility of a change in the nervous system due to practice. Although investigators have postulated many different types of change, no direct evidence of its nature exists. The nervous system itself tends to compound the problem. Within the complex of central tracts and centers and peripheral nerves, where does one look for a region which consistently represents a function and which can also be altered by practice? Investigation at the level of the spinal cord has often failed when the particular function is also represented in the cortex, and vice versa. What is needed is a highly localized function which is not so complex as to be partially represented at various levels of the nervous system and yet is not so simple that it is merely reflexive in nature and cannot be modified by practice. Such a function is handedness, which is localized in the contralateral frontal cortex (Peterson, 1934).

This region can be located by making a small cortical lesion to effect a transfer in handedness preference. (In rats, the handedness preference is defined by the number of times an animal completes hand to mouth feeding with a particular hand, either right or left.) The volume of a destruction necessary to produce such a transfer may be as little as 1.3 cubic millimeters (Peterson and Gucker, 1959).

The evidence indicates that the depth locus of the handedness function is probably in the fifth and sixth cortical layers (Peterson

and Farcarol, 1938). This conclusion, however, needs verification. The common areas in terms of the medial or frontal plane are not so finely delineated as the depth area, but effective lesions are known to cluster about the head of the caudate nucleus. Extensive lesions outside this area fail to produce transfer.

A method other than extirpation has also been used to localize the handedness function, in an attempt to leave the area functionally intact. Electrical stimulation is applied to the cortex to produce a movement of the contralateral arm. The stimulable area for arm movements is in the frontal cortex (Woolsey, 1958). This method provides information about the locus of the function without destroying tissue or producing transfer. Small destructions made in the electrical stimulation area, however, do not always produce transfers, indicating that the method is not very precise (Peterson and Gucker, 1959).

Because the fundamental problem under investigation concerns a change in cortical tissue as a function of practice, a method (such as electrical stimulation) which delineates without destroying should add a good deal of information. One factor operating against such a method is the spread of current over a field. Thus, stimulation at the dura may not follow a straight line to the fifth cortical layer. This would interfere with accurate determination by electrical stimulation. Obviously, the difficulty with the destruction method is that the tissue cannot be observed postoperationally.

If one wishes to study the neural equivalent of practice (in the handedness area) then one must change the animal's behavior by

means of practice. The method for achieving this is to force practice with the non-preferred hand. One way of doing the latter is to use an offset feeding dish, placed so that the rat can make complete reaches only with the non-preferred hand (Peterson, 1951). Unfortunately this method does not often produce transfer if the animal is given many pre-test reaches to establish handedness preference. Moreover, if the animal is given only a few test trials with the non-biased dish, the assertion that it is single-handed is less reliable.

Peterson (1951) however, reported another method which is superior to that of using the offset feeding dish. This involved binding the preferred arm so that it was held against the trunk of the animal. Thus, the animal could continue to feed from a non-biased dish, yet was forced to use the non-preferred hand for reaching. After a number of forced trials, the animals were unbound and observed for transfer. This method was highly efficient in producing transfers, but has been criticized as being injurious to the bound limb so that the resulting transfers might be due to injury. However, control animals which were bound but given no practice did not transfer, indicating that it was the forced practice which effected the change. It may be presumed that such practice produces a change somewhere in the rat.

The question now arises as to where the changes occur. A reasonable place to look for such changes would be the contralateral frontal cortex, if forced practice handedness is localized in the same way as natural handedness. If small destructions in this region

produce a retransfer to the originally preferred hand, and large destructions outside the area fail to be effective, then the question of the locus would be answered affirmatively. Thus, the present study is aimed at mapping the cortical region which controls forced practice of the handedness function, in the hope that such delineation may provide a specific region for the study of the neural equivalent of practice.

Method

General:

This study is based on the results of tests on forty-one animals from two laboratory strains (albino and hooded) which met the criterion of 90 or more single-handed reaches out of 100 trials in the Peterson (1931) food reaching situation.

These experimental animals had their preferred arms bound with adhesive tape. With the originally preferred hand thus immobilized, the animals were forced to reach with the non-preferred hand. Forced reaching continued until a transfer of preference occurred. The number of forced reaching trials to achieve transfer criterion varied from 600 to 1300 for different animals (see Table I). Transfer was checked by unbinding the originally preferred arm and observing the animals in a reaching situation. Those which transferred (90% single-handed reaches) were subjected to brain operation under ether anesthesia.

A 3/16 inch trephine was used to expose the frontal cortex at the angle of the midline and parietal sutures. This opening was contralateral to the hand preferred after forced reaching.

The cortical area to be altered operationally was located by electrical stimulation, resulting in a flexion of the contralateral arm. Following localization, an electrode was inserted in the cortex to produce a destruction of tissue.

Postoperationally, animals were given 100 reaches (in blocks of fifty) to check for Retransfer to the originally preferred hand.

Apparatus:

During the operation, animals were held by a modified stereotaxic instrument with two base plates. The uppermost of these plates had a raised V-block for holding the rat's head and a wire clamp for the animal's upper incisors. The lower of the two base plates had a steel arm which could be moved vertically so that the stimulating and destruction electrodes could be raised and lowered. In order to stimulate different areas of the cortex, the upper plate could be moved across the lower in a horizontal plane.

Electrical stimulation to produce an arm movement was performed with interrupted direct current at a 200 cps rate with a pulse duration of 0.5 milliseconds. The stimulation waveform was displayed on a Tektronix oscilloscope. Stimulation voltage was applied between a search electrode (0.6 millimeters in diameter) and a grounding electrode inserted at the region of the soft pad in the homolateralhind leg.

A thermocautery was used to make the larger destructions, while a unipolar electrode of approximately 100 microns in diameter and insulated to within 150 microns of the tip was used to produce smaller destructions. The insertion depth of the small electrode was measured by a GE depth gauge, reading in tenths of millimeters.

Procedure:

In fifteen animals, the transfer trials were followed by electrical stimulation and thermocauterization. A single-wire cautery, less than 1 millimeter in diameter (Peterson and Gucker, 1959) was used to produce destructions. The destructions varied in size from

Apparatus:

During the operation, animals were held by a padded U-shaped mechanical instrument with two base plates. The uppermost of these plates had a raised V-block for holding the rat's head and the other for the animal's upper incisors. The lower of the two bars had a metal arm which could be moved vertically so that the stimulating and destruction electrodes could be raised and lowered. The other plate could be moved in different areas of the coronal plane, the upper plate could be moved across the lower in a horizontal plane.

Electrical stimulation to produce an area was provided with interrupted direct current at a 200 cps rate with a pulse duration of 0.5 milliseconds. The stimulation wave-form was displayed on a Tektronix oscilloscope. Stimulation voltage was applied between a coated electrode (0.6 millimeters in diameter) and a grounding electrode inserted at the region of the scalp and the base of the left hind leg.

A thermocautery was used to make the lesions, while a unipolar electrode of approximately 300 microns in diameter and insulated to within 150 microns of the tip was used to produce small destructions. The insertion depth of the small electrode was measured by a CE depth gauge, reading in tenths of millimeters.

Procedure:

In fifteen animals, the stimulation trials were followed by electrical stimulation and thermocauterization. The single-wire coating, less than 1 millimeter in diameter (Peterson and Kaufman, 1953) was used to produce destructions. The destructions varied in size from

3.6 to 18.8 mm^3. The brains for these cases were prepared for histologic analysis by freezing, sectioning at 30 microns, and staining with thionin. Every other section through the damaged region was mounted and stained. Destruction areas were measured microscopically by a grid with minimum divisions of 210 microns on the side.

In twenty-one animals, electrical stimulation was followed by dessication with a Hyfrecator and a 100 micron electrode. The Hyfrecator setting for these destructions was 75 and the variac reading 100. Time was set at minimum, which was approximately 0.2 seconds duration. These settings were chosen because they produce a very small destruction.

Since it was difficult to see when the brain contact was made with this electrode, an auditory signal was used to indicate contact. This was triggered by an abrupt change in conductivity between the electrode and the dura. At the signal, the depth recorder was engaged and the electrode pierced the cortex to a predetermined level. Depth for these destructions was varied from 1.5 to 2.5 millimeters to provide destruction in different cortical layers.

Cases 16 through 32 had one electrode insertion to provide destruction. Rats 35 and 36 had two electrode insertions displaced approximately 1.5 millimeters in the area of the arm movement response. Rats 33 and 34 had three electrode insertions triangulated about the site of the positive stimulation area, and displaced from each other about 1.5 millimeters.

The brains from cases 17 through 32 were frozen, sectioned at

30 microns, and stained with thionin. Rats 33 through 36 has a brownish colored circle on the cortex which disintegrated when sliced. Since no prediction could be made for these animals unless the discolored area could be microscopically observed, cases 35 and 36 were not prepared by the freezing method, but were encased in thick celloidin. Slices were taken at 50 microns, and every fifth section through the damage region was kept for mounting and staining with thionin.

Five animals had trephine openings in the occipital area contralateral to the preferred hand. This opening was enlarged in a dorso-lateral direction by means of bone foreceps. No electrical stimulation was applied. Destructions were made with a looped wire thermocautery, heated to a cherry red glow. The areas destroyed by this procedure ranged from 33% to 50% of one hemisphere.

Results

The results for all animals are summarized in Table I. Seven animals, with thermocautery destructions ranging from 10.3 to 18.8 mm^3, retransferred to the originally preferred hand. One animal who failed to transfer (see No. 12) after 1300 forced reaches, was operated on the side contralateral to the originally preferred hand. 100% transfer resulted from a 12.6 mm^3 destruction for this animal.

Four animals with thermocautery destructions of from 51 to 9.8 mm^3 showed partial retransfer following the operation. The magnitude of this retransfer ranged from 15% to 83%.

Three animals, with thermocautery destructions, failed to retransfer. The destruction volumes for these animals were 3.6, 5.6, and 10.7 mm^3.

In order to determine if a common area existed for the retransfer cases, tissue sections which represented the site of greatest damage were selected for each animal. Cases which had destruction in the left hemisphere were transposed for ease of examination. Figure 1 shows the superimposed damage areas for retransfer cases. Cases were studied for common depth locus without regard for frontal dispersion, since previous studies established such variability as to cause a common area to vanish.

Figure 2 displays the superimposed areas of greatest damage for partial and non-retransferring cases.

Figure 3 was derived from an overlay of Figure 1 on Figure 2, and is that area in which at least part of each transfer case

destruction appears, and in which no partial or non-retransferring case is represented. This region is approximately 3 millimeters wide, and 2 millimeters deep at the points of greatest width and depth and does not involve cortical layers 1, 2, and 3.

Using the head of the caudate nucleus as a landmark for determining destruction spread along a frontal plane, it was found that no common area existed for the fifteen thermocautery animals. However, the limits for effective destruction were from one millimeter rostral to somewhat more than 2.3 millimeters caudal of the landmark.

Of the 21 animals with tissue destruction produced by dessication, eight retransferred following operational procedures. Four of these animals had dessication destructions varying in volume from 0.7 to 3.4 mm^3.

The other four who retransferred had both dessication destructions and thermocoagulated tissue, resulting from trephine heat. The volumes for these destructions ranged from 18.6 to 27.1 mm^3. It is immediately apparent that these destructions are of a magnitude and nature that adds no knowledge to the question under investigation. Therefore, no further analysis of these cases was made.

Animals 18, 22, 27, and 31 exhibited partial retransfer following dessication of cortical tissue. Percent retransfer for these animals ranged from 28 to 75% and the volumes of tissue damage from 0.6 to 1.0 mm^3. Nine animals failed to retransfer following dessication. Damage volumes for this group ranged from 0.1 to 2.1 mm^3.

Figure 4 was obtained by superimposing the individual areas

of destruction for animals who retransferred following dessication of cortical tissue. This figure is a spatial overlay of the lateral and depth planes. To determine the correspondence between the area defined by the thermocautery cases, Figure 4 was placed on Figure 3 and the region common to both figures presented in Figure 5. Thus Figure 5 represents the area common to all retransferring animals regardless of type of destruction. Figure 5 is somewhat smaller than Figure 3 in a lateral direction. (2.3 millimeters as compared to 3.1 millimeters in Figure 3.)

The dorso-ventral spread of the dessication destructions shows that no retransfer animal had a destruction nasal to the head of the caudate nucleus, and the caudal-most destruction is only 1.7 millimeters behind the caudate nucleus.

Of the five animals which had occipital destructions varying in size from 33% to 50% of one hemisphere, only one showed a minor (18%) retransfer. The destruction area for that animal (no.39) is shown in Figure 7. It is doubtful that this is a bona fide retransfer case, especially in the light of the other four occipital-temporal cases who failed to be influenced despite extensive injuries.

Damage areas for animals 37, 38, 40, and 41 are superimposed on figure 6. All these animals failed to retransfer. Thus, extensive lesions outside the critical area (as defined by these data) fail to produce a change in the handedness function.

Examination of the damage areas for all retransfer cases, despite operating method, shows that the cortical care which represents

forced practice is approximately the same as the cortical area which represents "natural" handedness (see Figure 5).

Discussion

In an effort to localize the region representing forced practice, both the volume and location of effective destructions were critically analyzed.

It was found that a volume of 0.7 mm^3 representing approximately 1000 cells (Tower, 1952) effected a retransfer in one case. This volume is smaller by 0.6 mm^3 than the least effective volume reported by Peterson and Gucker (1959) for ambidextrous animals.

It might be hypothesized that "forced practice" animals are more like ambidexters than single-handed animals, since both hemispheres have regions representing the handedness function. This hypothesis is supported by the fact that the least effective volumes reported for single-handed animals was 1.3 mm^3.

On the other hand, it may be that the limiting factor is not in the size of the destruction, but in the ability of the equipment accurately to place the electrode. If this is the case, then ambidexters and single-handers may not be so divided as it now appears.

Certainly the size of the destruction alone does not account for transfer or retransfer. Excluding those cases with trephine injury, and focusing on cases with frontal damage alone, it may be seen that a great deal of size overlap occurs. Table II shows the volumes of destructions for these animals, dividing the cases into retransferring, partial retransferring and non-retransferring categories. The volumes are reported in increasing magnitudes from the smallest to the largest. The area of overlap is enclosed by horizontal

lines. Inspection of this table supports the postulate that volume cannot be the critical variable in producing retransfers.

From Figure 5, showing the effective region common to all retransferring cases, the limits in the lateral plane are well defined. The problem, then, is to assure that a lesion will occur within the limits set by the data. This involves the equipment to be used.

Part of the problem lies in the stereotaxic instrument. If the two base plates of this equipment were marked off in 0.01 millimeter divisions, and a vernier control for the two transverse directions in the horizontal plane was attached, then post-stimulation operating would be more precise.

Of course there is always the problem of subdural spread of impulses when electrical stimulation is used to identify a region. At the surface of the tissue point X might be stimulated to produce a response. However, by the time the impulse reaches layer 5, the spread might be such that a circular area, say four times the diameter of the stimulating electrode might be involved. If only one peripheral arc of this circle really represented handedness, and if the destruction electrode was consistently inserted at the point on the dura where stimulation produced a response, then a change in handedness preference could not always be predicted for such a destruction. What is called for to correct for sub-dural or intra-cortical spread is a method of asserting just what this spread will be.

If one had the precise stereotaxic instrument indicated previously, it would be possible to maximize the choice of destruction region by

plotting stimulation responses as a function of two mutually orthogonal directions.

However, even if the region was so minutely defined at the surface, other problems arise. There are two major problems in trying to isolate the destruction to a very restricted area at a sub-dural level. One is the incompatibility of the strength of materials, with the desirability of producing as small a destruction as possible in the layers lying above the fifth and sixth layers. The other is the restriction of the destruction zone in a longitudinal direction (along the axis of the electrode at the fifth and sixth layers). A possibility that may be examined comes from recent developments in the field of metallurgy and the marriage of metallurgy and ceramics. Single crystalline iron with approximately ten times the strength of normal iron has been grown in the laboratory. One of these small "whisker" crystals with a thin plating of fused quartz as an insulator should allow a reduction of a full order of magnitude in the diameter of the destruction electrode. In turn, this would allow for the control of the longitudinal length of the destruction within a tolerance of 0.04 millimeters.

Thus, by the development of precision equipment it may be possible to localize the region of forced practice in handedness to within far smaller limits than were achieved in this study.

Summary and Conclusions

If one wishes to study the neural equivalent of learning as a function of practice, then one must locate a particular neural region which consistently represents a function and which may also be altered by practice. The handedness area of the frontal cortex has been located for a "natural" handedness preference, an area which also fulfills the second requirement. This study was done to ascertain whether or not this region could be so located and so altered when it represents a forced practice handedness function.

Forty-one rats were forced to reach with an originally non-preferred hand until this forcing resulted in a transfer of handedness preference.

Animals were then operated on and cortical lesions produced. If the animals demonstrated a retransfer to the originally preferred hand following the operation, the area of lesion would represent the forced handedness function. The brains of all animals were examined histologically and the destruction areas for non-retransferring cases subtracted from the areas for retransferring cases to delimit the critical area.

As hypothesized, the resulting area was in the same region as that which had been found to represent a "natural" handedness function in the rat. Thus, a particularized region for the study of the neural equivalent of learning as a function of practice is available for further investigation.

Within the limits of the data for this study, the volume of the

lesion was not as critical to retransferring as was the position of the lesion. Since one retransfer was effected by a lesion of 0.7 mm^3, it may be inferred that, with finely controlled equipment, the region may be accurately predicted within the confines of some 1000 cells.

REFERENCES

References

Earp, Kenneth R., Electrical dessication as a method for marking functional cortical areas. Unpublished thesis.

Harlow, H. F., & Woolsey, C.N., Biological and biochemical bases of behavior. Madison: University of Wisconsin Press, 1958. 63-82.

Peterson, Geo. M. Mechanisms of handedness in the rat. Comp. Psych. Monogr., 1934, 9, 1-67.

Peterson, Geo. M. and Fracarol, C. The relative influence of the handedness by the cerebral cortex. J. Comp. Neurol., 1938, 68, 173-190.

Peterson Geo. M. Change in handedness in the rat by local application of acetycholine to the cerebral cortex. J. Comp. & Physiol. Psychol., 1942, 36, 404-411.

Peterson, Geo. M., Transfer in handedness in the rat from forced practice. J. Comp. & Physiol Psychol., 1951, 44, 184-190.

Peterson, Geo. M., & McGibony, D. R., Reeducation of handedness in the rat following cerebral injuries. J. Comp. & Physiol. Psychol., 1944, 38, 191-196.

Peterson, Geo. M., & Gucker, Donald K., Factors influencing identification of the handedness area in the cerebral cortex of the rat. J. Comp. Physiol. Psychol, 1959, 52,

Tower, D.B. Structure & functional organization of the mammalian cerebral cortex: The correlation of neuron density with brain size. J. Comp. Neurol., 1954, 101, 19-46.

Wentworth, K. L. Some factors determining handedness in the white rat, Genet. psychol mono. 1942, 26, 55-117.

Wright, Sampson (ed), Applied Physiology, Oxford: Oxford University Press, 1956.

References

Barp, Kenneth P., Electrical deactivation as a method for marking functional cortical areas. Unpublished thesis.

Barlow, H. F., & Woolsey, C.N., Stimulated and Electrocortical Waves of Behavior. Wisconsin: University of Wisconsin Press, 1938. 63-12.

Peterson, Geo. N. Incidence of handedness in the rat. Comp. Psych. Monogr., 1934, 9, 1-67.

Peterson, Geo. N. and Fracarol, C. The relative facilitation of the handedness by the cerebral cortex. J. Comp. Neurol., 1938, 68, 173-190.

Peterson Geo. N. Change in handedness in the rat by local excitation of acetylcholine to the cerebral cortex. J. Comp. & Physiol. Psychol., 1942, 35, 409-431.

Peterson, Geo. N., Transfer in handedness in the rat from forced practice. J. Comp. & Physiol. Psychol., 1938, 44, 184-190.

Peterson, Geo. N., & McGlenn, R. E., Deprivation of handedness in the rat following cerebral injuries. J. Genet. & Physiol. Psychol., 1944, 38, 191-196.

Peterson, Geo. N., & Castar, Donald E., Factors influencing identification of the handedness area in the cerebral cortex of the rat. J. Comp. Physiol. Psychol., 1939, 32,

Tower, D.B. Structure & functional organization of the mammalian cerebral cortex: The correlation of neurone density with brain size. J. Comp. Neurol., 1934, 101, 19-46.

Wadsworth, K. L. Some factors determining handedness in the white rat, Genet. psychol. mono. 1940, 26, 55-117.

Wright, Sampson (ed), Applied Physiology, Oxford: Oxford University Press, 1956.

APPENDIX

TABLE I
Summary of Reaching Records and Results
According to Various Methods

Rat No.	Pref. Hand	Number of Reaches Forced	Pre-op R	Pre-op L	Post-op R	Post-op L	Destruction in mm^3
1	R	1000	1	99	98	2	12.3
2	L	1000	100	0	0	100	16.4
3	R	1000	0	100	42	58	8.8
4	L	1000	96	4	3	97	12.8
5	L	1000	96	4	0	100	11.2
6	R	1000	0	100	81	19	10.0
7	L	1000	100	0	98	2	10.7
8	L	800	93	7	6	94	10.3
9	L	1000	96	4	0	100	13.6
10	L	1000	100	0	100	0	3.6
11	L	1200	93	7	7	93	18.8
12	R	1300	69	31	0	100	12.6
13	L	1000	99	1	17	83	7.6
14	L	1000	92	8	85	15	5.1
15	L	1000	90	10	100	0	5.6
		Dessicator					
16	R	800	3	98	100	0	0.7
17	L	900	90	10	97	3	0.2
18	R	700	6	94	75	25	0.5

TABLE I (cont'd)
Summary of Reaching Records and Results
According to Various Methods

Rat No.	Pref. Hand	Number of Reaches					Destruction in mm^3
		Forced	Pre-op		Post-op		
			R	L	R	L	
19	L	900	99	1	91	9	2.0
20	R	600	0	100	1	99	0.5
21	R	900	0	100	50	50	0.6
22	R	700	1	99	94	6	3.4
23	R	700	0	100	100	0	2.8
24	L	900	99	1	100	0	0.6
25	R	900	100	0	99	9	2.1
26	R	900	7	93	28	72	0.4
27	R	1000	0	100	96	4	1.1
28	L	1000	100	0	100	0	0.4
29	L	900	99	1	91	9	0.3
30	L	600	98	2	97	3	0.1
31	R	1000	0	100	69	31	0.9
32	L	600	98	2	97	3	0.2
		Dessicator-Trephine Damage					
33	R	800	0	100	100	0	18.6
34	R	800	6	94	100	0	22.4
35	L	800	100	0	0	100	27.1
36	R	800	10	90	91	9	19.8

TABLE I (cont'd)
Summary of Reaching Records and Learning
According to Various Methods

Rat No.	Trial Hand	Trained	Number of Reaches Pre-op R	Pre-op L	Post-op R	Post-op L	Destruction $\frac{gm}{mm^3}$
19	L	900	99	1	91	9	2.0
20	R	600	0	100	1	99	0.5
21	R	900	0	100	50	50	0.5
22	R	700	1	99	94	6	3.4
23	R	703	0	100	100	0	2.8
24	L	900	99	1	100	0	0.6
25	R	909	100	0	99	9	2.1
26	R	909	7	93	28	72	0.4
27	R	1200	0	100	96	4	1.1
28	L	1000	100	0	100	0	0.4
29	L	900	99	1	91	9	0.3
30	L	600	98	2	97	3	0.1
31	R	1000	0	100	69	31	0.9
32	L	600	98	2	97	3	0.2
			Denervator-Training Damage				
33	R	800	0	100	100	0	18.6
34	R	800	6	94	100	0	22.4
35	L	800	100	0	0	100	27.1
36	R	800	10	90	91	9	19.8

TABLE I (cont'd)
Summary of Reaching Records and Results
According to Various Methods

Rat No.	Pref. Hand	Number of Reaches					Percent Hemisphere Destroyed
		Forced	Pre-op		Post-op		
			R	L	R	L	
37	L	600	92	8	100	0	36
38	L	1000	100	0	100	0	33
39	L	700	95	5	82	18	41
40	L	700	100	0	97	3	50
41	R	800	0	100	0	100	36

TABLE II
Volumes of Destruction for All Cases
(Excepting occipital and trephine damage)

Rat No.	Retransferring	Rat No.	Partial Retransferring	Rat No.	non-Retransferring
				30	0.1
				32	0.2
				17	0.2
				29	0.3
		26	0.4	28	0.4
		18	0.5	20	0.5
		21	0.6	24	0.6
16	0.7	31	0.9	19	2.0
27	1.1	14	5.1	25	2.0
23	2.8	13	7.6	10	3.6
22	3.4	3	8.8	15	5.6
8	10.3	6	10.0	7	10.7
5	11.2				
1	12.3				
12	12.6				
4	12.8				
9	13.6				
2	16.4				
11	18.8				

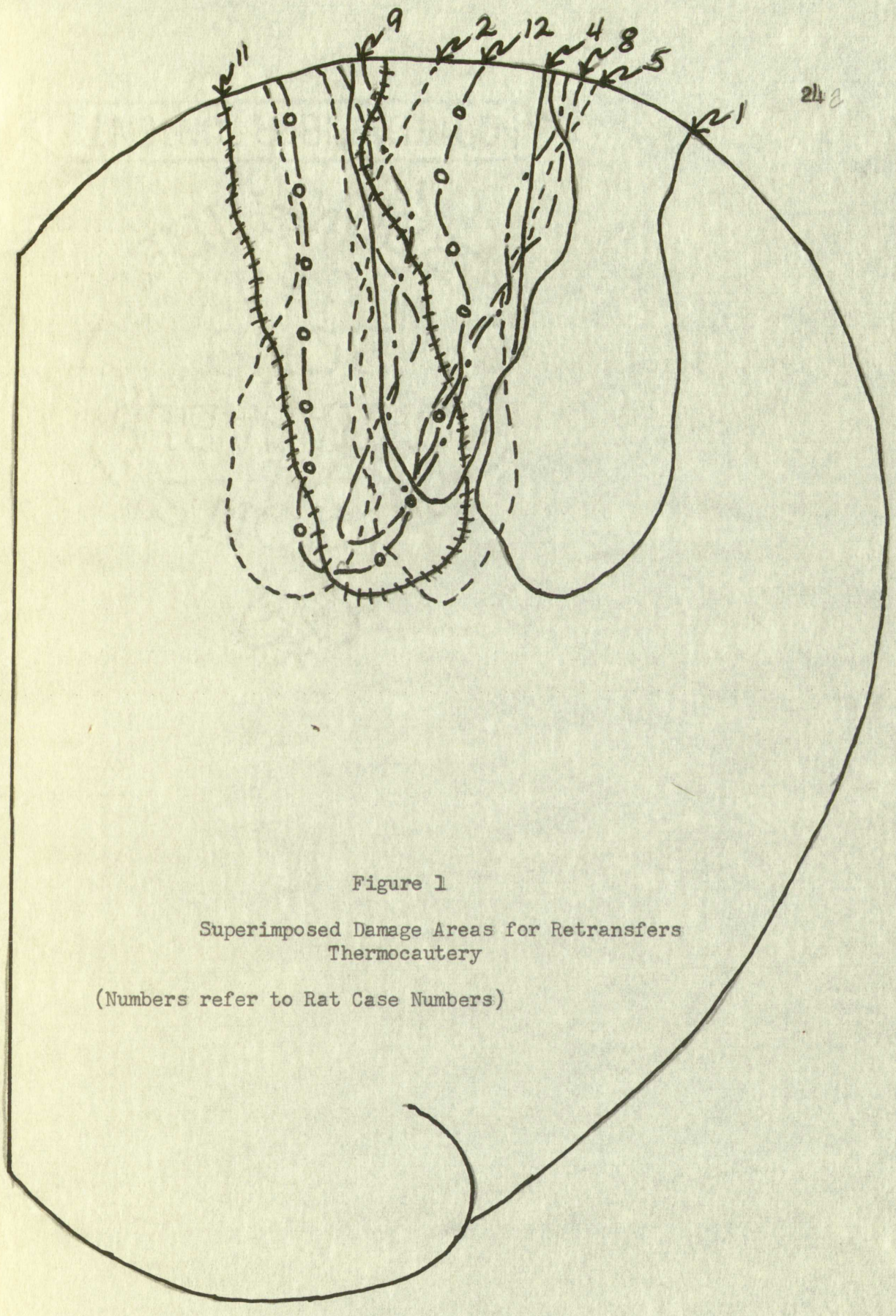
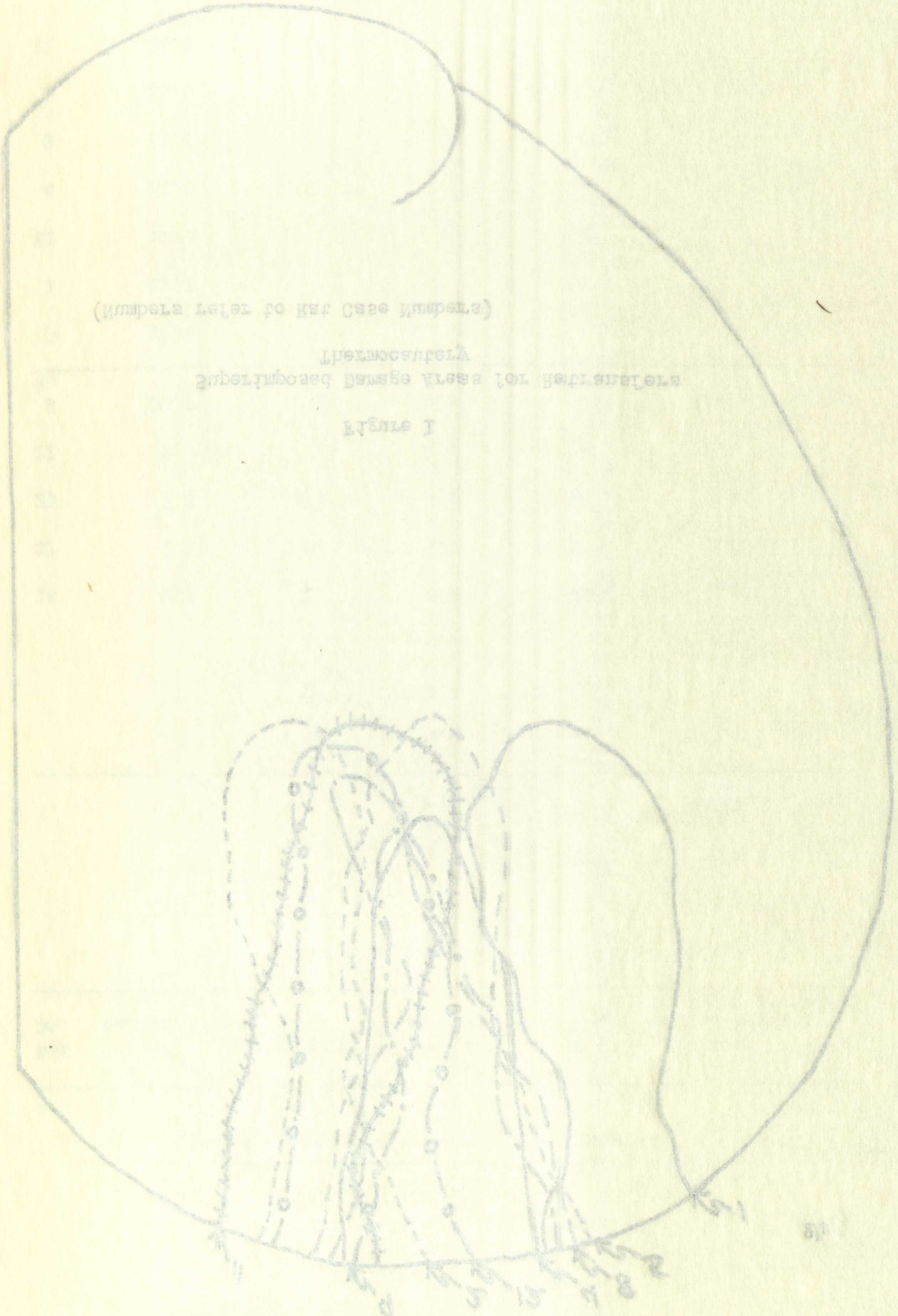


Figure 1

Superimposed Damage Areas for Retransfers
Thermocautery

(Numbers refer to Rat Case Numbers)



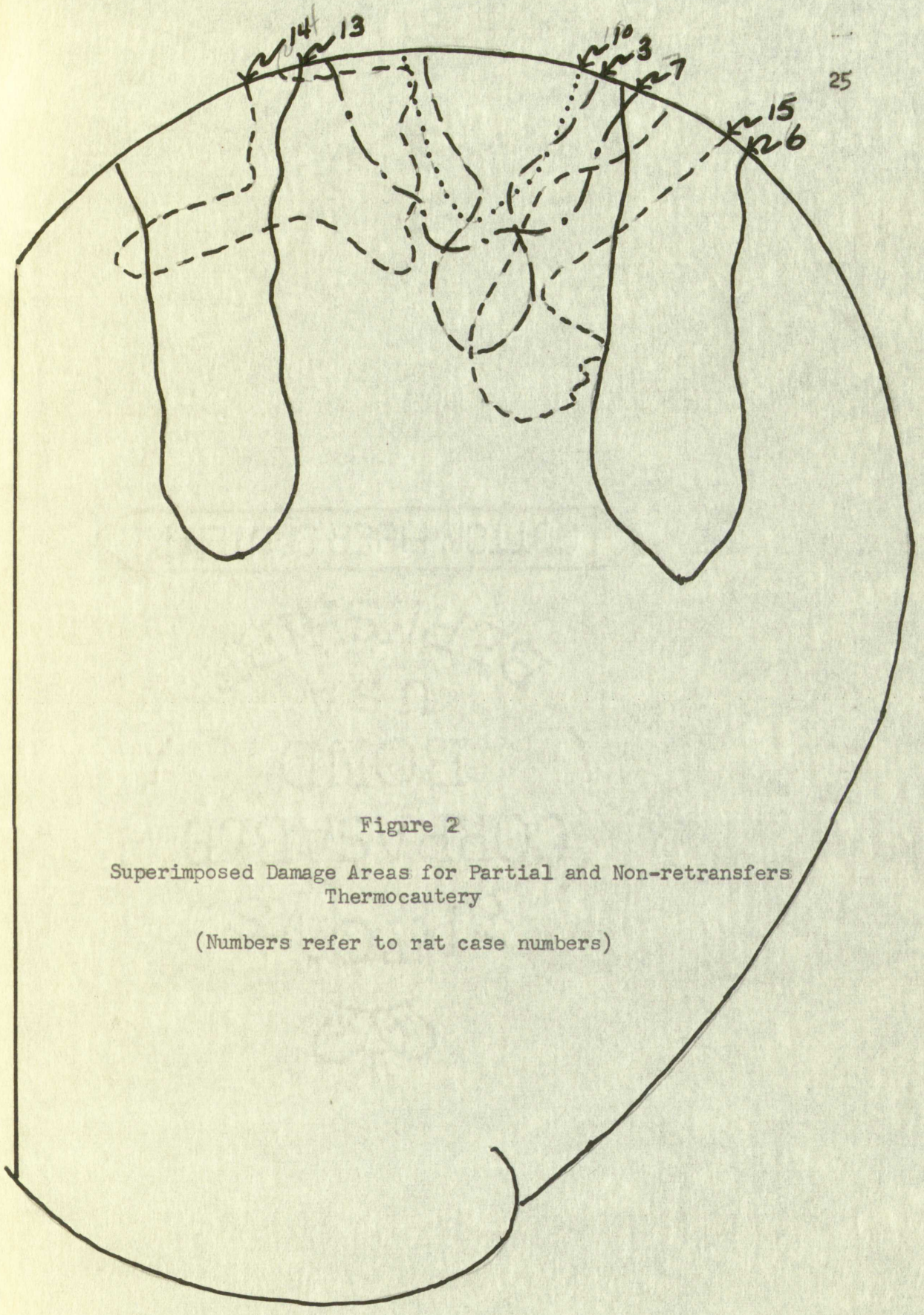
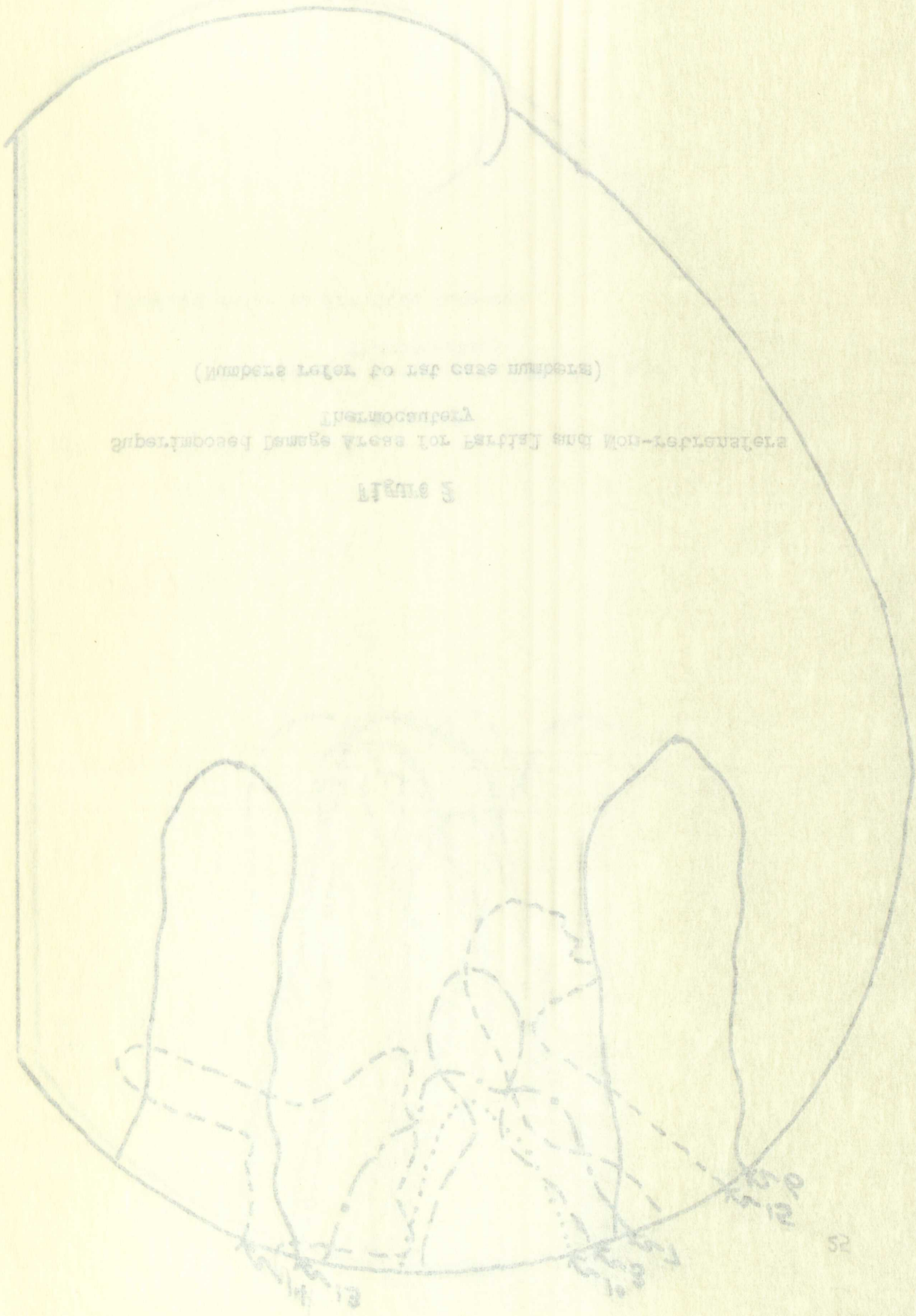


Figure 2

Superimposed Damage Areas for Partial and Non-retransfers Thermocautery

(Numbers refer to rat case numbers)



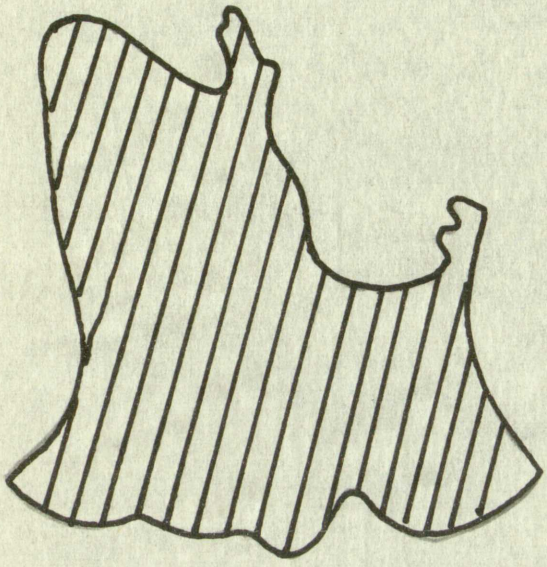


Figure 3

Critical Area for Retransferring Cases
Thermocautery

(Figure 1 minus Figure 2)

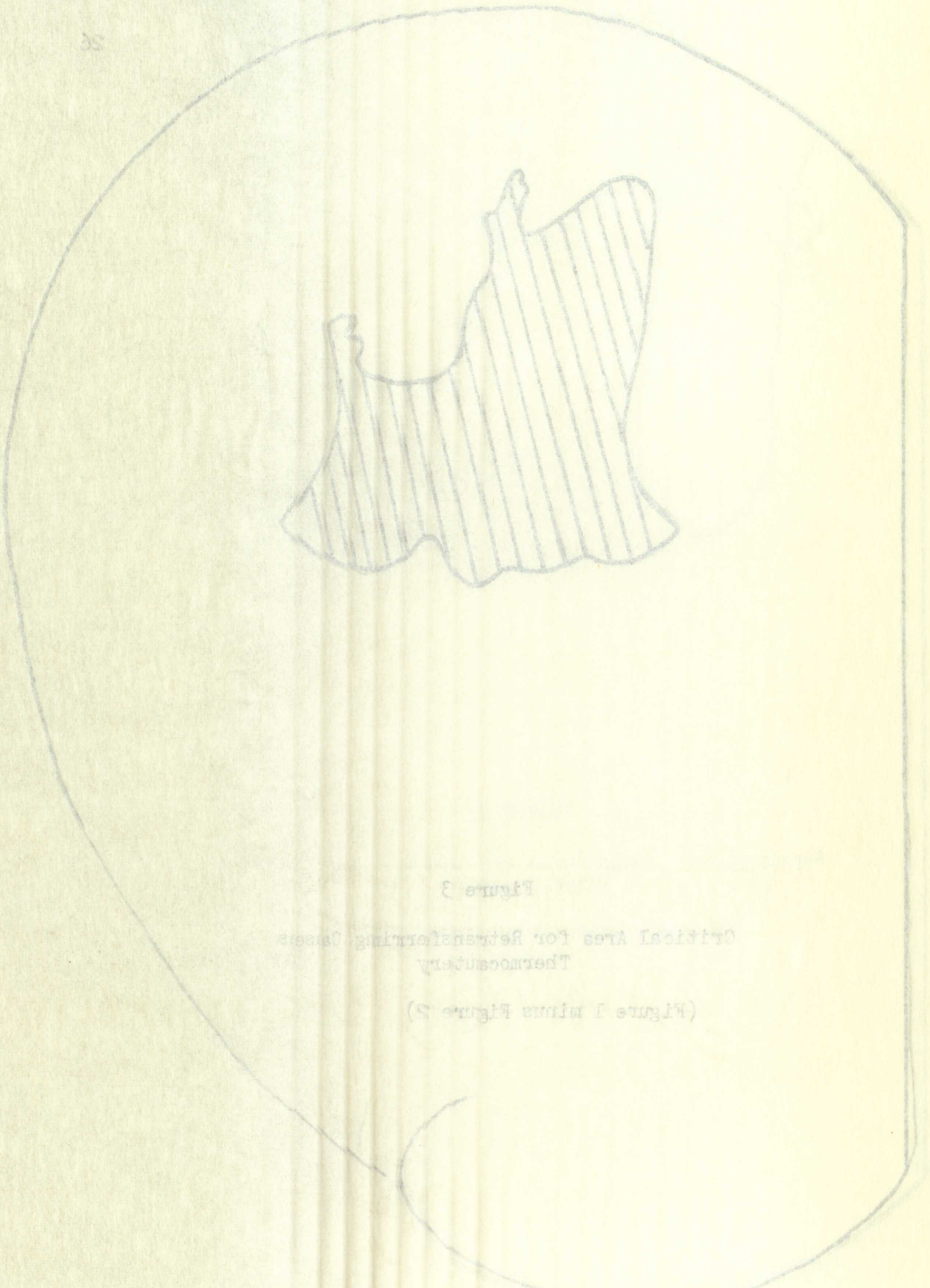


Figure 3

Critical Area for Retransferring Cases
Thermocautery

(Figure 1 minus Figure 2)

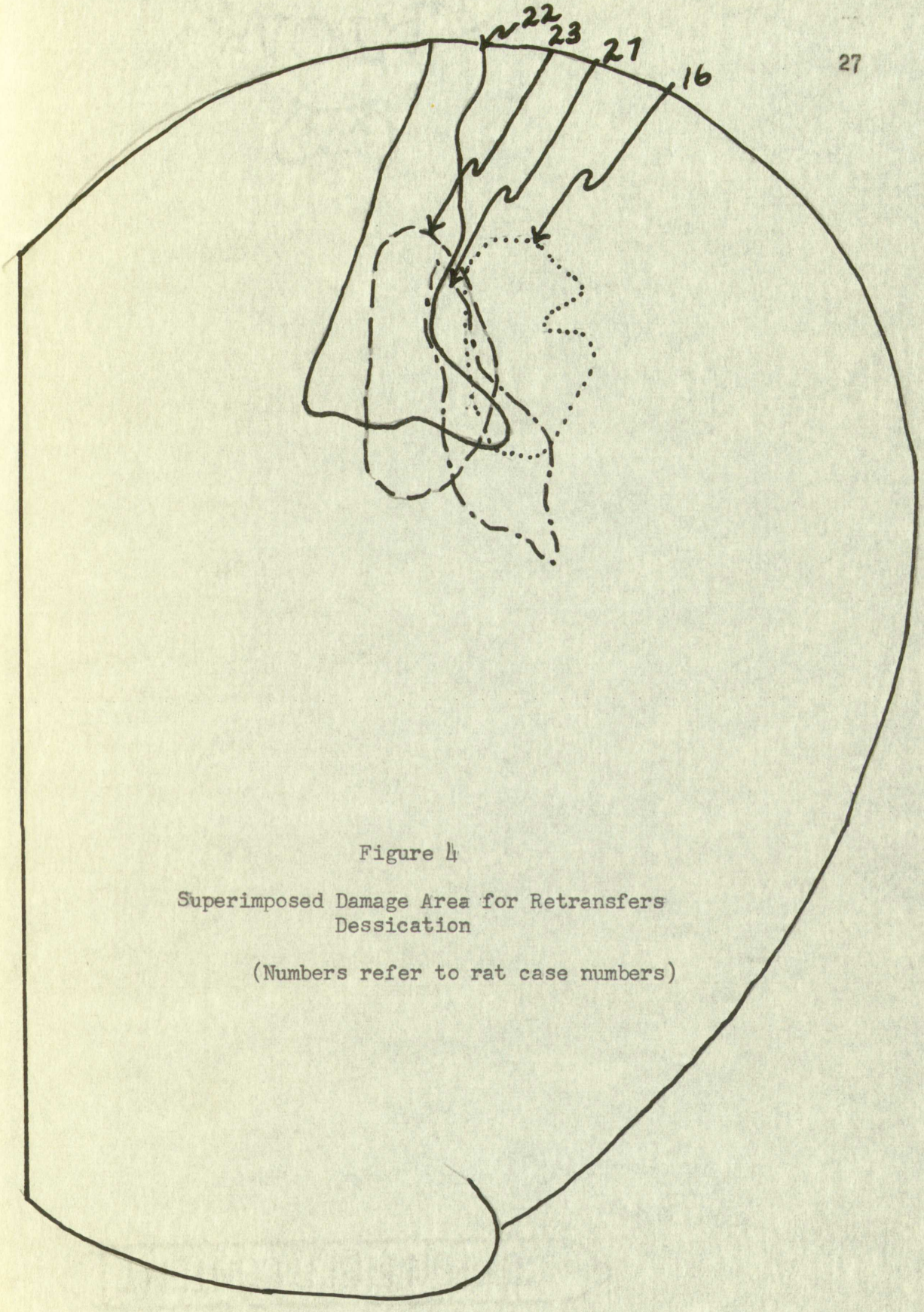


Figure 4

Superimposed Damage Area for Retransfers
Dessication

(Numbers refer to rat case numbers)

Figure 1

Superimposed Damage Area for Retransfers
Dessication

(Numbers refer to vet case numbers)

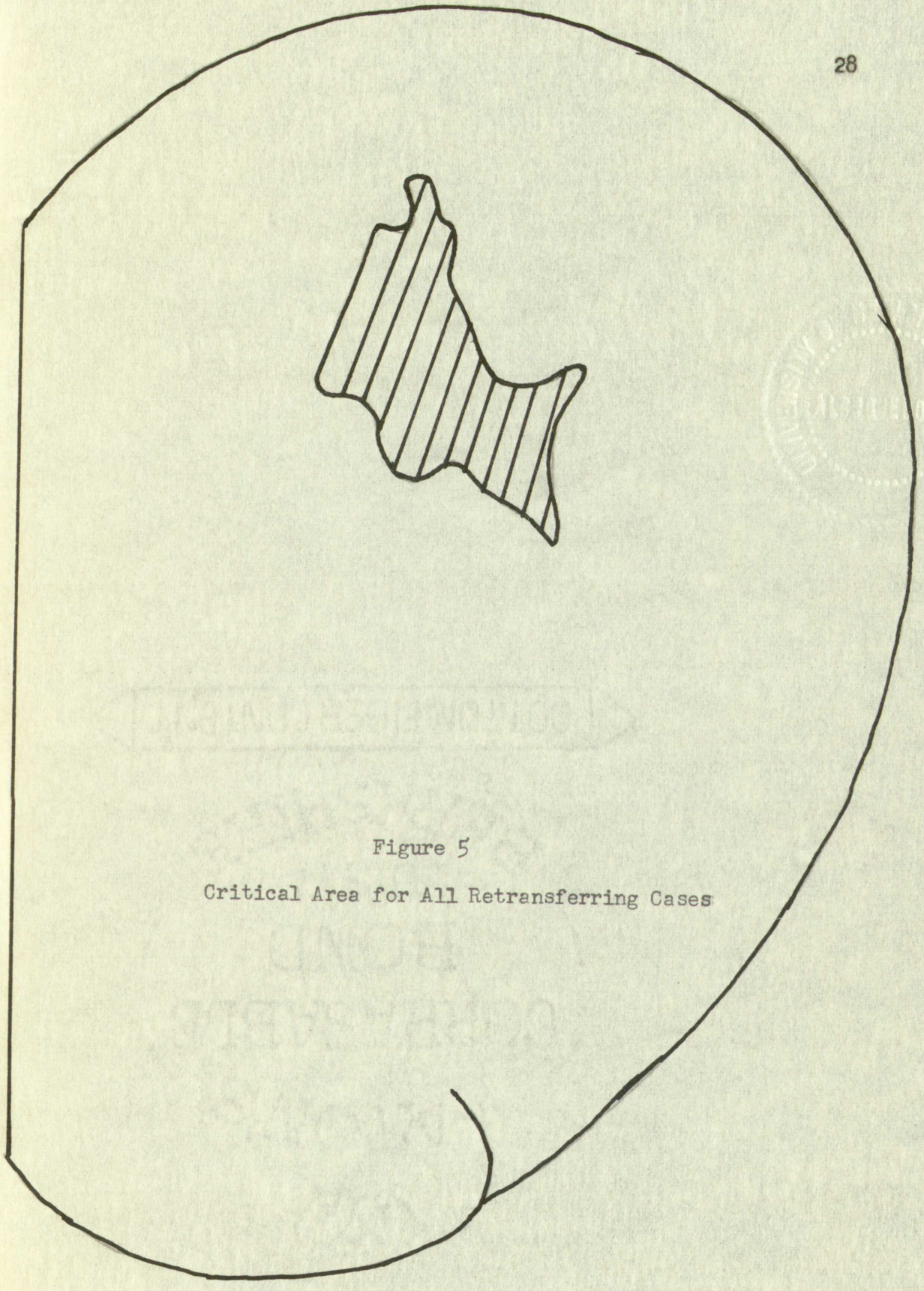
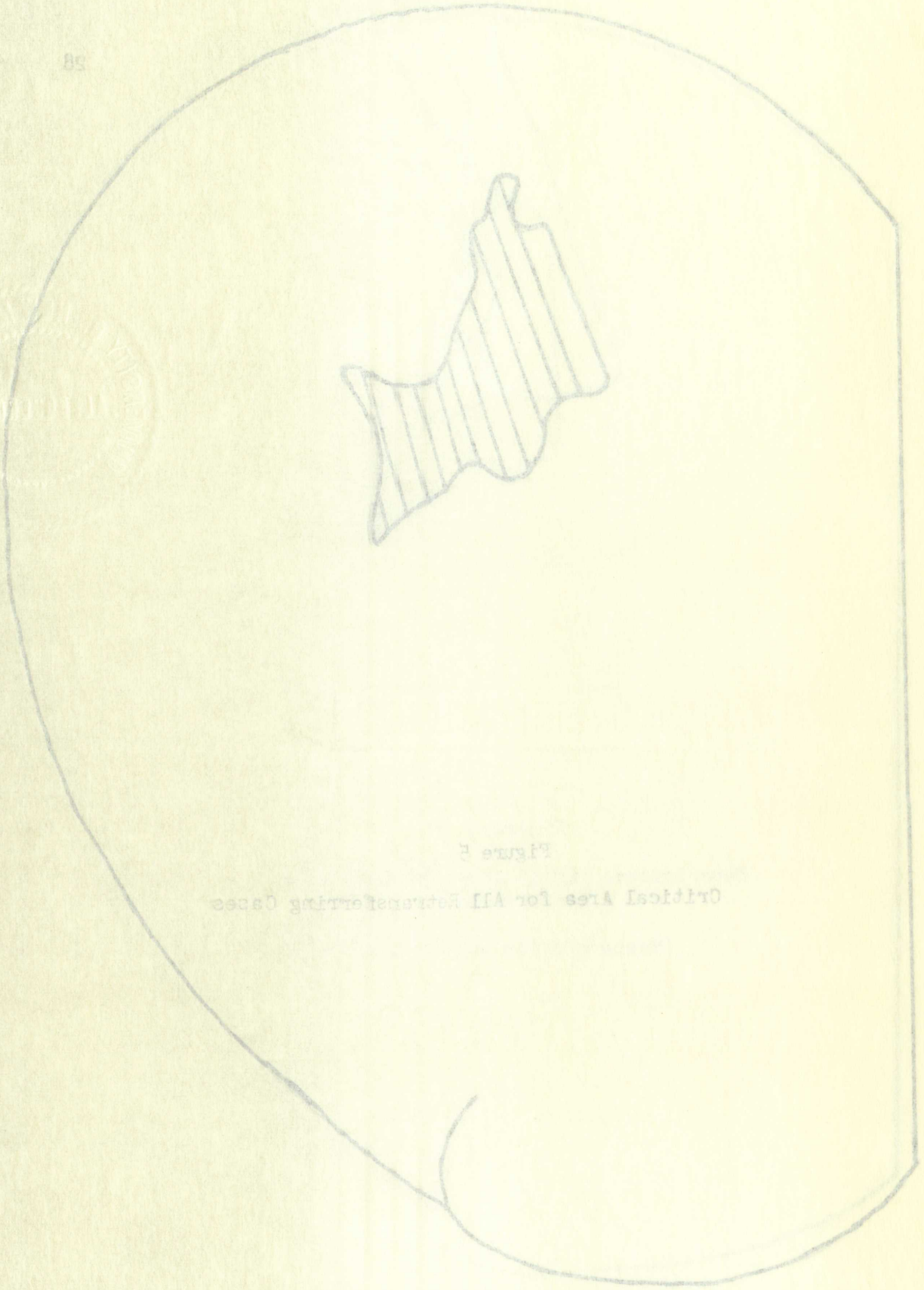


Figure 5

Critical Area for All Retransferring Cases



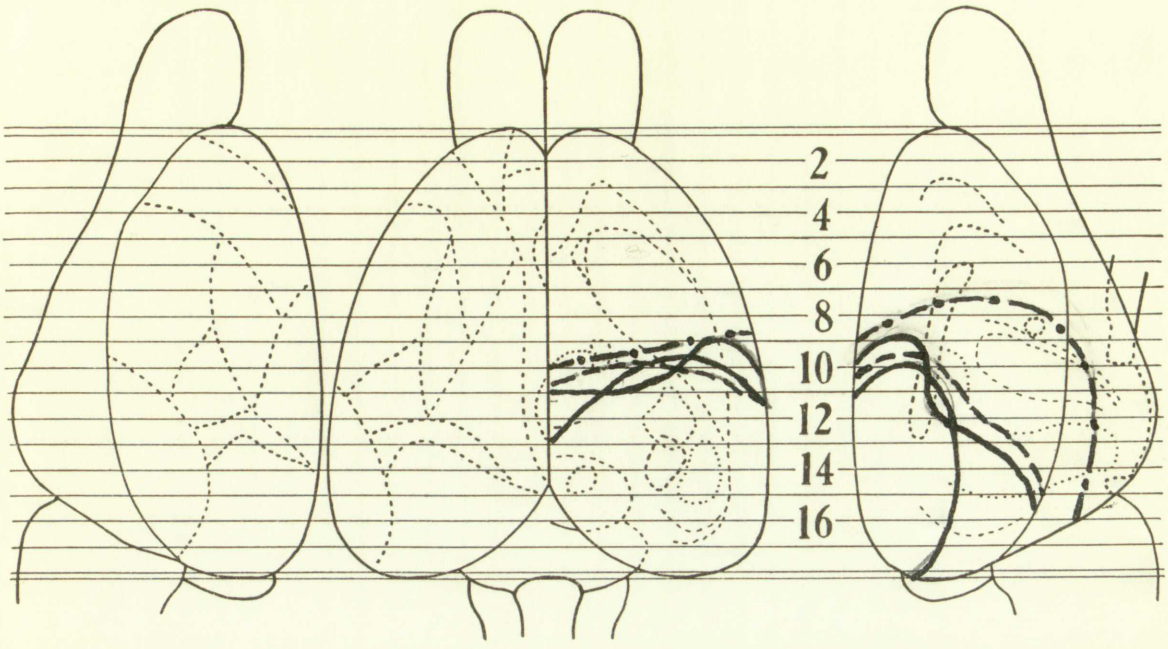


Figure 6. Destruction for Occipital Cases 37, 38, 40 and 41

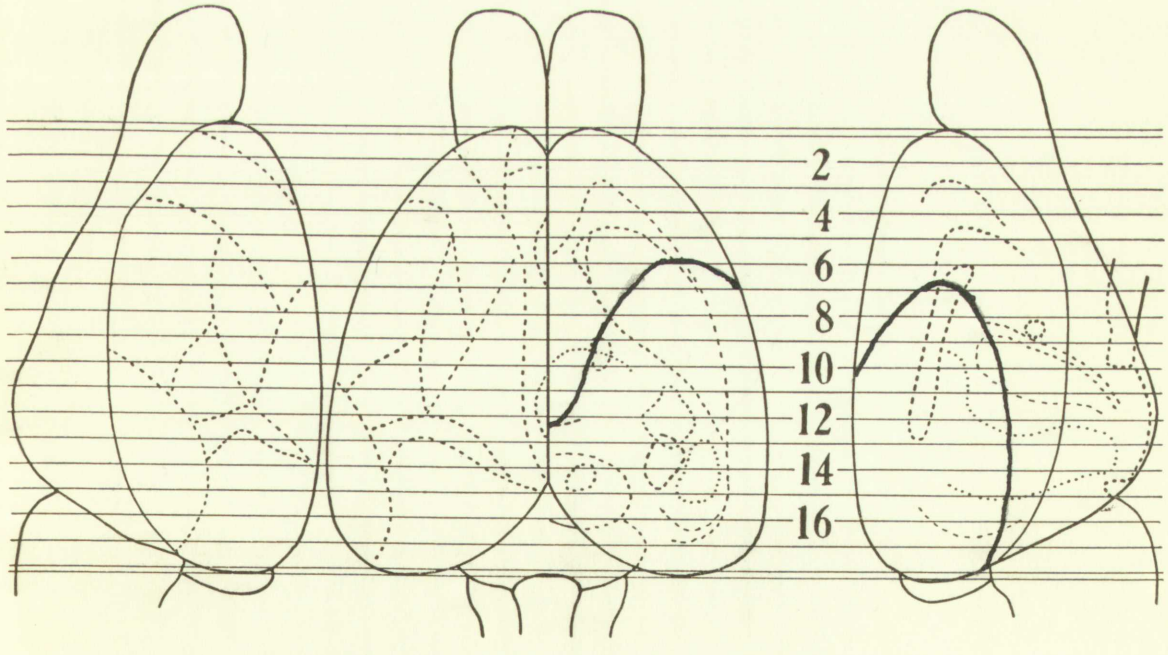


Figure 7. Destruction for Occipital Case 39

IMPORTANT!

Special care should be taken to prevent loss or damage of this volume. If lost or damaged, it must be paid for at the current rate of typing.