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# ORGANIZATION OF THE SUBCORTICAL SYSTEM GOVERNING DEFENCE AND FLIGHT REACTIONS IN THE CAT

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Earlier studies carried out in this laboratory by means of electrical stimulation in the brains of freely moving cats have indicated that a subcortical system governing elementary affective reactions extends from forebrain into brain stem. This system reaches from dorsomedial parts of the amygdala along the stria terminalis to the level of the anterior commissure (Fernandez de Molina & Hunsperger, 1959) and from thence into the preoptic area, the 'intermediate' zone of the hypothalamus (Hess & Brügger, 1943; Hunsperger, 1956), and the central grey matter of the mid-brain (Hunsperger, 1956).

These investigations also showed that the reactions obtained from the forebrain and the brain stem were similar but not identical. They were all associated with pupillo-dilatation and pilo-erection, but the defence pattern obtained from amygdala and stria terminalis was characterized by growling and growling–hissing, whereas the responses from the brain stem comprised two patterns, a defence pattern characterized by hissing and an escape pattern. The experiments made it clear that the brain-stem field comprises: (*a*) two 'inner' zones related to the hissing pattern and situated in the 'perifornical' region of the hypothalamus and in the central grey matter of the mid-brain respectively, and (*b*) one common 'outer' zone related to the escape pattern (Hunsperger, 1956).

By combining stimulation with coagulation in the active field at brain-stem levels Hunsperger (1956) demonstrated that hissing could not be elicited from the hypothalamus after bilateral coagulation of the mid-brain 'hissing zone', but could be elicited from the mid-brain after bilateral coagulation of the 'hissing zone' in the hypothalamus. Flight elicited from the hypothalamus was suppressed by coagulation of the mid-brain 'hissing zone'; it could, however, still be obtained by increasing the strength of stimulation. Bilateral coagulation in the active field at the level of the caudal hypothalamus did not affect the hissing produced from the rostral hypothalamus. It follows from this that the central grey matter

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of the mid-brain is not simply traversed by a discharge path originating in the hypothalamus, but constitutes a self-reliant station within the neurone paths controlling defensive behaviour. Further support for this interpretation was the observation that after destruction of the 'hissing zone' in the mid-brain the cats, when confronted with a dog, no longer hissed nor attacked; animals, however, with coagulation of the 'hissing zone' in the hypothalamus still responded with a complete defence pattern.

Changes in the emotional behaviour of the cat have also been reported after surgical ablation of the amygdala involving the pyriform cortex. Results obtained vary between production of savageness (Bard & Mountcastle, 1948; Bard, 1950) and greater docility (Schreiner & Kling, 1953). Recently Wood (1958) after bilateral electrolytic lesions in restricted parts of the amygdala produced increased aggressive behaviour in cats.

The purpose of this paper was to investigate whether the growling and growling-hissing patterns produced by electrical stimulation of dorsomedial parts of the amygdala also depend on the integrity of the 'hissing zones' situated in the central grey matter of the mid-brain and in the hypothalamus. Unilateral and bilateral coagulation was therefore effected in these zones and the responses elicited from the amygdala were tested before and after. We also studied the relative contribution of the amygdala to the hissing and to the flight reactions evoked from the hypothalamus, and compared the responses produced from the 'inner' and 'outer' zones of the hypothalamus before and after coagulation in the amygdala. The effect of amygdala coagulation on the spontaneous behaviour of the animals was also studied. Finally we interrupted the route connecting the active area in the amygdala with the active zones in the hypothalamus by placing lesions in the stria terminalis bed, and compared the stimulation responses evoked from the amygdala before and after interruption. The responses obtained from the hypothalamus were only tested after interruption.

The Hess–Wyss technique of previous studies was employed. Two preliminary reports have been published (Fernandez de Molina & Hunsperger, 1958; Hunsperger & Fernandez de Molina, 1958), the matter dealt with was included in a review (Hunsperger, 1959).

## METHODS

Stimulation was combined with coagulation in 23 of the 47 cats in which Fernandez de Molina & Hunsperger (1959) studied the central representations of affective reactions in amygdala and related structures. The Hess–Wyss technique (Hess, 1932, 1957; Wyss, 1945, 1950) was used. All surgery was carried out under $N_2O$ anaesthesia combined with local infiltration of procaine. Although the effect of the local anaesthetic, which contained no adrenaline, would be expected to wear off after 30 min, none of the animals showed any signs of discomfort, such as scratching of the small opening in the skin or rubbing its head against a wall, and they remained fully responsive to fondling throughout.

The stimulation parameters in forebrain structures were described by Fernandez de Molina & Hunsperger (1959); the brain-stem parameters correspond to those outlined by Hunsperger (1956).

Lesions were placed by means of high-frequency coagulation (Wyss, 1945, 1957). For coagulation in the 'hissing zones' of the brain stem and in the stria terminalis bed a unipolar electrode arrangement was used, the indifferent electrode, connected to the metal frame screwed on the skull, being earthed. The needles, 0·3 mm in diameter, had a bared tip of 1·5 mm for coagulation in the grey matter of the hypothalamus; 1·0 mm for coagulation in the central grey matter of the mid-brain, and in the stria terminalis bed and surrounding structures. The electrode tips had an active surface of 1·4 and 1·0 mm² respectively. Table 1 gives a survey of the average size of the lesions in the structures listed. These lesions were obtained with optimal current strength (cf. Hunsperger & Wyss, 1953) and represent the minimal sizes producing decisive effects.

TABLE 1

| Structure | Active surface of electrode (mm²) | Duration of coagulation (sec) | mA | V | Size of lesion | |
|---|---|---|---|---|---|---|
| | | | | | Diameter (mm) | Length (mm) |
| Grey matter of the hypothalamus | 1·4 | 15 | 38 | 24 | 3·3 | 4·6 |
| Central grey matter of the mid-brain | 1·0 | 15 | 35 | 24 | 3·2 | 4·3 |
| Structures within and outside the stria terminalis bed | 1·0 | 10 | 23 | 23 | 2·2 | 3·5 |

For coagulation of the amygdala a bipolar arrangement was used, with one needle earthed. The coagulation needles, which had a diameter of 0·8 mm, were lowered into the brain by means of a modified Hess frame carrying on each side two needle holders 3 mm apart and distant 9 mm from the midline. The needles were inserted in the brain 1·5 and 4·5 mm behind the coronal suture until their tips touched the bone under the pyriform cortex. They were then fixed on the holders by tightening clamp screws. In order to coagulate the amygdala and to spare the pyriform cortex (Pl. 1, fig. *C*), the needle shafts were bared between 2 and 5 mm from the tips, each electrode having an active surface of 7·5 mm². A current of 145 mA and a voltage of 45 V applied for 15 sec between the electrodes produced a lesion measuring on an average 7·3 mm craniocaudally, 6 mm transversely and 8 mm dorsoventrally.

*Procedure.* When testing the responses elicited from the amygdala following coagulation in the 'hissing zones' of the hypothalamus or of the mid-brain we proceeded as follows: one set of three electrodes, 1·5 mm distant from one another, was placed in the active area of the amygdala on one side; a second and a third set were symmetrically placed in the 'hissing zones' either in the hypothalamus or in the mid-brain. Electrical stimulation was first carried out on all electrodes inserted. The electrode that yielded the response pattern from the amygdala with lowest threshold was selected as the test needle; the needles placed in the brain stem that produced the hissing pattern with low threshold were chosen for coagulation. Coagulation in the brain stem was first carried out on one side, usually with two needles, either ipsilaterally or contralaterally to the side of stimulation in the amygdala. The response was re-tested, following which coagulation was carried out in the brain stem on the side that was still intact. A third and final check in the amygdala completed the experiment. Conversely, when testing the effect of unilateral and bilateral coagulation of the amygdala on the response patterns elicited from the hypothalamus, a similar two-stage procedure was followed; no exploratory stimulation, however, was carried out with the

large-sized electrodes used for amygdala coagulation. Coagulation within the stria terminalis bed, carried out on one side only, was combined with bilateral stimulation of the amygdala and of the ipsilateral hypothalamus. It should be noted that we used non-stimulating high-frequency coagulation in the waking animal and were thus able to test the response patterns immediately after coagulation.

In this series of 23 animals, 7 had lesions in the central grey matter of the mid-brain, and 7 in the hypothalamus. In 3 of these latter 7, lesions serving as controls were placed in areas situated rostrally or medially of the 'hissing zone' in the hypothalamus. In 3 cats both amygdalae were coagulated and in 6 the lesions were placed in the stria terminalis bed or in neighbouring structures.

In the animals with bilateral coagulation of one of the 'hissing zones' in the brain stem or with bilateral coagulation of the amygdala, the effect of coagulation on the spontaneous behaviour and on the aggressive reactions when provoked by unoperated cage mates was observed over a period of three weeks. In the two groups with coagulation in the central grey matter of the mid-brain or in the amygdalae a second stimulation was carried out in the amygdala or hypothalamus 2 weeks later, on the side that had not been previously explored.

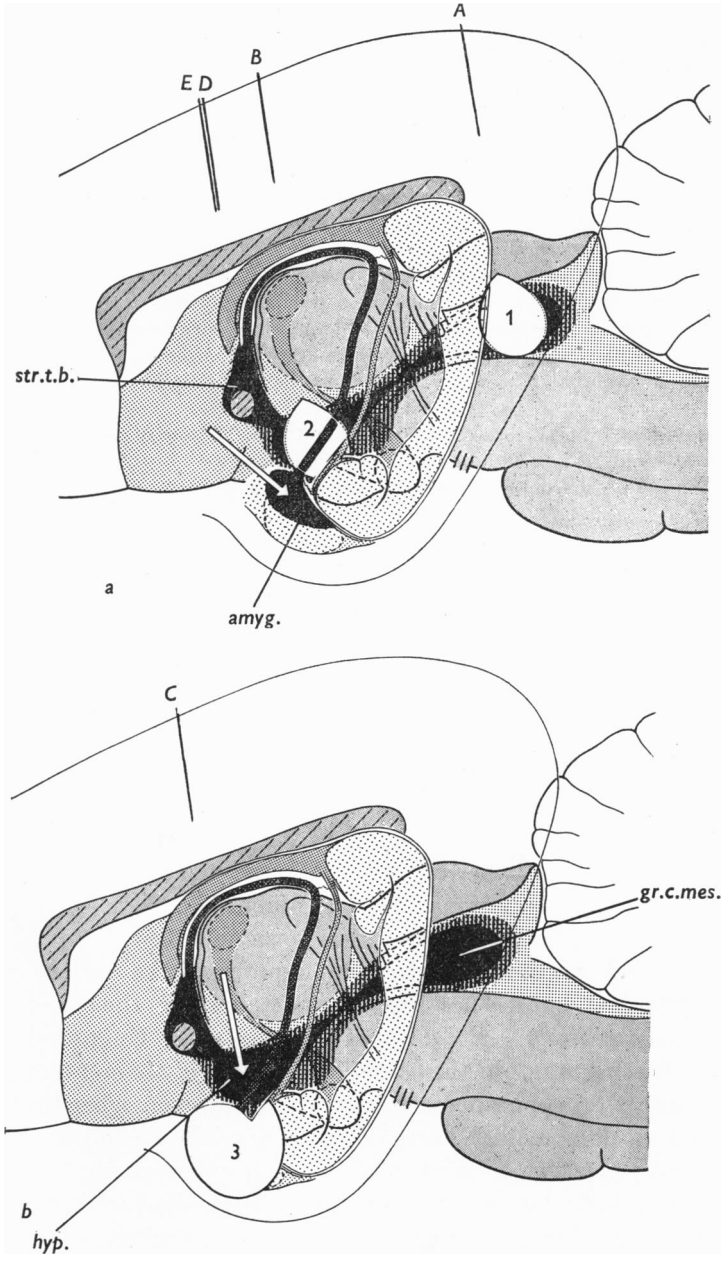
All effects observed were recorded by protocol and film. The site of the tips of the stimulating electrodes and the site and dimensions of the lesions were histologically examined in serial sections cut in a frontal plane and alternately stained by Heidenhain and Nissl techniques.

## RESULTS

### *Effects of coagulation on the affective reactions produced by electrical stimulation*

Coagulation in the brain stem and forebrain of the zones mediating the hissing or growling-hissing reactions was combined with electrical stimulation at various levels of the whole reactive field. Consistent results were obtained. Thus the growling and growling-hissing patterns elicited from the amygdala were abolished by coagulation of the 'hissing zone' at either mid-brain or hypothalamic level (Text-fig. 1*a*); while conversely the hissing and escape patterns elicited from the hypothalamus were not altered by bilateral coagulation of the amygdalae (Text-fig. 1*b*). A coagulation carried out at the level of the stria terminalis bed, interrupting the route between forebrain and brain stem, suppressed the pattern obtained from the amygdala, but did not affect the patterns from the hypothalamus.

*Coagulation within the active areas of the brain stem.* Unilateral coagulation of the 'hissing zone' in the *central grey matter of the mid-brain* had no noticeable effect on spontaneous behaviour. However, when stimulation was applied to dorsomedial parts of the amygdala the marked pupillo-dilatation, pilo-erection on back and tail, retraction of ears, lowering of the head and slight hunching of the back, associated with growling or growling leading to hissing, could no longer be produced when the electrical stimulation was applied on the ipsilateral side. The reactions elicited were slight pupillo-dilatation and alertness of the animal. Normal growling and growling-hissing patterns were observed in those experiments in




**Text-fig 1. For legend see opposite page.**

which stimulation was applied to the contralateral amygdala. A bilateral coagulation at this mid-brain level reduced the spontaneous activity and inquisitive interest of the animals (see below) besides, as was to be foreseen, suppressing the amygdala pattern.

The site and dimensions of the bilateral lesions in the 'hissing zone' of the central grey matter of the mid-brain that prevented the development of the amygdala pattern are shown in Pl. 1*A*, illustrating one case typical for five others. The lesions extend from the level of the posterior commissure to the level of the trochlear nucleus. In one of the six animals pilo-erection unaccompanied by excitement of the cat was still elicited. In two others pilo-erection was produced by increasing the strength of stimulation, and in one of these, with doubled stimulus intensity, the animal again emitted low growls without assuming a defence posture. In a seventh cat stimulation of the amygdala produced 'unpremeditated' flight—that is, flight not preceded by searching for an outlet. This reaction, rare from the forebrain, was not suppressed by bilateral coagulation in the 'hissing zone' of the mid-brain.

Lesions in the 'hissing zone' of the *hypothalamus* had similar effects on the growling-hissing pattern from the amygdala. A unilateral coagulation suppressed the defence pattern from the amygdala of the same side, but had no effect when carried out on the opposite side. Bilateral coagulation caused little impairment of the spontaneous activity of the animal but abolished the electrically evoked amygdala pattern. Effective bilateral coagulation within the 'hissing zone' of the hypothalamus is illustrated in Pl. 1*B*. These lesions comprise the rostral portion of the 'perifornical' region. In none of the four animals was pilo-erection observed after bilateral coagulation. Increase in stimulus strength did not produce any signs of emotional excitement.

Bilateral lesions placed headward of the 'hissing zone' in the hypothalamus, damaging this field but slightly, had no effect on the growling and growling-hissing patterns from the amygdala (two cases). In a third

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Legend to Text-fig. 1

Text-fig. 1. Schematic illustration of site of stimulation (arrow) and coagulation (numbered white fields) in the structures governing defensive behaviour in forebrain and brain stem. In both figures the sagittal plane through forebrain has been superimposed on the sagittal plane through brain stem. (*a*) Stimulation at the level of the amygdala, coagulation at the level of the central grey matter of the mid-brain (1) or of the hypothalamus (2). (*b*) Stimulation at the level of the hypothalamus, coagulation of the amygdala (3).

*A–E* planes of frontal sections illustrated in Pl. 1. (Figure based on illustration of active field in Fernandez de Molina & Hunsperger, 1959; Text-fig. 2.) *amyg.*, amygdala; *gr.c.mes.*, central grey matter of the mid-brain; *hyp.*, hypothalamus; *str.t.b.*, stria terminalis bed.

animal a lesion involving medial parts of the rostral hypothalamus bilaterally, and impinging on the 'hissing zones' on both sides, prevented the development of the 'unpremeditated' flight response elicited from the amygdala. When stimulation strength was increased the animal again showed excitement accompanied by growls.

Concomitant effects occasionally obtained from the amygdala, such as turning of eyes and head to the side opposite to stimulation, and sniffing, were not abolished by bilateral coagulation within either of the 'hissing zones' of the brain stem.

*Coagulation of the amygdala.* Unilateral and bilateral coagulation of the amygdala did not reduce spontaneous activity and inquisitive interest of the animal. The electrically produced hissing pattern obtained from the 'inner' zone and the escape pattern evoked from the 'outer' zone in the hypothalamus remained unaffected. Both patterns were obtained with the same stimulation strength as before coagulation and the latency for hissing and flight remained unaltered. Plate 1*C* shows the site and dimensions of bilateral lesions in the amygdala that destroyed the whole active area—central, medial, and basal nuclei—damaged the lateral nucleus and spared most of the pyriform cortex.

*Coagulation within the stria terminalis bed and surrounding structures.* Interruption of the route connecting the active area in the amygdala with the active zones of the hypothalamus was carried out on one side only, in order to compare the patterns produced by ipsilateral and contralateral amygdala stimulation. The growling and growling-hissing patterns from the amygdala of the same side were suppressed, they could still be obtained from the amygdala of the opposite side. Increased stimulation strength on the ipsilateral side after coagulation merely produced pupillodilatation and alertness. The defence posture with accompanying piloerection and growls was never evoked. Plate 1*D* shows the site of a lesion in the stria terminalis bed that prevented the development of the defence pattern from the amygdala on the same side. In contrast to the suppressing effect of lesions involving the stria terminalis bed on the responses produced from the ipsilateral amygdala, no influence on the hissing pattern elicited from the ipsilateral hypothalamus was noticed.

The growling and growling-hissing patterns obtained from the amygdala were not altered after lesions had been placed in structures adjacent to the stria terminalis bed. Both descending columns of the fornix at the level above the anterior commissure, including precommissural fornix fibres, or rostral portions of the septum, or anteromedial divisions of the anterior thalamic nuclei were destroyed. Plate 1*E* shows the sites of bilateral lesions that interrupted both fornices and the precommissural fornix fibres, leaving the stria terminalis bed intact.

*Effects of coagulation on spontaneous behaviour*

Changes in the emotional behaviour of the animals were only observed after bilateral lesions within the active field of the brain stem, their degree varying according to the level of destruction. Bilateral coagulation within the 'hissing zone' in the mid-brain produced marked and long-lasting changes, whereas bilateral coagulation within the 'hissing zone' of the hypothalamus only gave rise to slight and short-lasting changes. As these findings confirm previous results (Hunsperger, 1956) they will be but briefly described. The animals after coagulation in the mid-brain neither hissed nor attacked when provoked by their unoperated cage mates; previously aggressive cats were now easy to handle. At the same time the cats showed a lack of spontaneous activity and inquisitive interest; a deficit that lasted approximately 2 weeks. Recovery of spontaneous activity and inquisitive interest was not parallelled by recovery of aggressive behaviour. The animals after coagulation in the 'hissing zone' of the hypothalamus, in spite of a transient reduction in spontaneous activity and inquisitive interest, showed no impairment of aggressive behaviour. Although the general state of these animals was less affected, two of the cats died in the night following coagulation.

Bilateral coagulation of the amygdala in these waking cats, impinging only slightly on the pyriform cortex, was without noticeable effect on the emotional behaviour of the animals and no reduction of spontaneous activity and inquisitive interest was seen. Thus one cat, placid before operation, remained docile immediately after the intervention and during the whole survival period of 3 weeks; the two others, hostile when approached by hand or by other animals, also showed no changes in their disposition. Alterations in feeding habits, exaggerated oral tendencies (Klüver & Bucy, 1939), or abnormal sexual behaviour (Schreiner & Kling, 1953; Green, Clemente & De Groot, 1957) were never observed.

*Electrical stimulation in chronic preparations*

Two weeks after bilateral coagulation within the 'hissing zone' in the central grey matter of the mid-brain, stimulation was applied to the amygdala that had not been previously explored. Histological examination showed that the electrodes had been successfully placed in the centre of the active area in one of 3 cats. In this preparation the growling-hissing pattern developed at the usual stimulus strength. The cat, however, manifested less excitement compared with the reactions seen upon stimulation in the intact animal. In the other two preparations the needles were placed at the border of the active area. In the first cat 'unpremeditated'

flight was obtained, in the second pupillo-dilatation and pilo-erection, but only with intensities exceeding the usual values.

In the series of animals with coagulation of both amygdalae electrodes were placed 2 weeks later in the 'hissing' and 'flight' zones of the hypothalamus in one cat. The hissing and escape patterns were obtained at the usual critical intensities; they developed in the same manner as in acute preparations and in intact animals.

## DISCUSSION

Our data indicate that the growling and growling-hissing patterns produced by electrical stimulation of dorsomedial portions of the amygdala arise because of activation of the zones controlling the hissing pattern situated in the hypothalamus and in the central grey matter of the mid-brain respectively. Although coagulation of one of these brain stem zones in the acute state suppresses the affective patterns obtained from the amygdala, concomitant effects, such as turning of the eyes and head to the side opposite stimulation—a reaction recalling the 'attention response' of Kaada (Kaada, Anderson & Jansen, 1954; Ursin & Kaada, 1960*a*)—remain unaffected. Since unilateral coagulation never produced drowsiness, the suppressing effect cannot be attributed to generalized reduction of the state of wakefulness. These observations indicate that lesions in the brain stem regions governing the hissing pattern suppress those aspects of the affective responses obtained from the amygdala, which are specifically concerned with defence, but leave the motor elements of turning unaffected. They also support the suggestion of Fernandez de Molina & Hunsperger (1959) that the turning movements that sometimes accompany the affective patterns should be considered independent factors. It is interesting to note that even extensive bilateral lesions in the brain stem failed to suppress the turning of eyes and head to the opposite side on stimulation of the amygdala (Ursin & Kaada, 1960*b*).

Our experiments also show that the growling-hissing pattern obtained from the amygdala is suppressed by an ipsilateral lesion placed at mid-brain or hypothalamic levels. The hissing pattern obtained from the hypothalamus is only suppressed by a bilateral coagulation in the mid-brain field (Hunsperger, 1956). An explanation of this fact might be that electrical stimulation at the level of the amygdala is associated with ipsilateral activation of the responsive fields in the hypothalamus and the mid-brain, whereas electrical stimulation at the level of the hypothalamus causes a bilateral spread of activation, although the pathways conducting such activation are so far unknown. An investigation dealing with spread of activation according to the level stimulated is in progress and further

discussion will be postponed (T. L. Brown and R. W. Hunsperger, to be published).

Recovery of the electrically produced affective patterns occurs 2 weeks subsequent to coagulation in the central grey matter of the mid-brain, not only from the amygdala, but also from the hypothalamus (Hunsperger, 1956). It is not likely that the suppressing effect in the acute state is due to the development of oedema, because the test stimulations were carried out immediately after coagulation. It would seem, therefore, that recovery of the patterns produced by electrical stimulation in the chronic state may be due to a reorganization of the remaining parts of the system. However, the fact that 11 such animals out of 12 failed to react when confronted with a dog or cat or when handled suggests that this reorganization of the remaining parts of the system does not provide a mechanism which is efficient enough to respond to natural stimuli.

Results also indicate that threshold, latency, and character of the hissing and flight patterns elicited from the respective zones in the hypothalamus in acute and chronic preparations are not noticeably affected by coagulation of both amygdalae that spared most of the pyriform cortex. Furthermore, the individual disposition of the animals (savageness, docility) remained unaffected. All these observations are not in accord with the hypothesis of Bard & Mountcastle (1948; see also Bard, 1950) that the amygdala acts as a funnel through which inhibitory mechanisms originating in the cortex may exert a suppressing action on brain-stem mechanisms yielding affective reactions. Nor do our results lend support to the conclusion of Schreiner & Kling (1953) that the shift towards greater placidity observed in their cats after surgical ablation of the amygdala—including the pyriform cortex—may be due to the destruction of the amygdala. Our negative findings with regard to abnormal sexual behaviour after bilateral coagulation of the amygdala are in agreement with results obtained by Green *et al.* (1957). For further references and discussion of discrepancies in results of previous authors with regard to effect of bilateral amygdalectomy on the emotional behaviour of the animal, see the review of Hunsperger (1959).

Recently, Wood (1958) observed increased aggressive behaviour (snarling, biting, clawing on even gentle handling) in half of his cats with bilateral electrolytic lesions in either the basal or the central nucleus of the amygdala. This author pointed out that it seemed hard to understand that the structures, which upon electrical stimulation gave rise to growling and hissing, should also exert a suppressing action on brain-stem mechanisms for the same behaviour, and tentatively suggested that the lateral nuclei of the amygdala may exert an inhibitory action on the basal and central nuclei. This apparent contradiction can be resolved, however, by assuming

that his electrolytic lesions, which did not destroy the whole active area in the amygdala, were irritative and led to chronic excitation. The observation of Green *et al.* (1957) that bilateral electrolytic lesions in the amygdala, pyriform cortex, and neighbouring parts of the hippocampus produced increased aggressive behaviour only in those cats developing seizures, would support this point of view.

From the evidence presented in this paper and previous communications (Fernandez de Molina & Hunsperger, 1959; Hunsperger, 1956) we conclude that the basal, medial, and central nuclei of the amygdala are a part of the neural system governing defensive behaviour in forebrain and brain stem. This system is organized at three levels of progressively increasing importance—amygdala, hypothalamus, and central grey matter of the mid-brain. Thus the growling and growling-hissing patterns obtained by electrical stimulation from the amygdala probably develop through activity of the 'hissing zone' of the hypothalamus, and activity of the hypothalamus is maintained in turn by the presence of the central grey matter of the mid-brain. Such a hypothesis of a segmental and hierarchical arrangement of the system in the central nervous system governing defensive behaviour would explain why coagulation at the level of the mid-brain in the acute state is able to suppress the responses yielded at the level of the amygdala, although the interposed 'hissing zone' of the hypothalamus remains undestroyed. The suppressing effect of coagulations at the level of the hypothalamus would then be due to elimination of a relay station connecting the active area of the amygdala with the 'hissing zone' of the mid-brain. Our assumption is further supported by the finding that acute interruption of the stria terminalis prevents the development of the reactions from the amygdala, but leaves those produced from the hypothalamus unchanged.

The question might be asked whether we are not dealing with a discharge path originating at amygdala or even higher levels and descending via hypothalamus and central grey matter of the mid-brain to effector structures situated more caudally in the brain stem. Decortication (Dusser de Barenne, 1920; Schaltenbrand & Cobb, 1930; Bard & Rioch, 1937; Rioch & Brenner 1938) and transection of the brain stem at the level of the anterior hypothalamus (Bard, 1928, 1934) in cats, however, do not impair rage reactions to 'nociceptive' stimuli. A transection separating mesencephalon from diencephalon in the chronic cat affects the posture of the defence pattern, but leaves hissing, growling, and pilo-erection unimpaired (Keller, 1932; Bard & Macht, 1958). Even less complete patterns are obtained after transection at mid-collicular levels (Woodworth & Sherrington, 1904; Bazett & Penfield, 1922) or upper pons (Bard & Macht, 1958). These findings show that rage reactions are displayed as long as mid-brain

structures are preserved. Our investigations indicate that these structures are located in the central grey matter of the mid-brain.

Previous experiments (Hunsperger, 1956), as mentioned above (p. 200), have shown that bilateral coagulation within the system governing defensive behaviour at the level of the caudal hypothalamus does not affect the responses obtained by electrical stimulation either from the rostral hypothalamus or from the central grey matter of the mid-brain. This would also tend to prove that the suppressing effect of lesions in the central grey matter of the mid-brain on the responses produced from the amygdala is not simply due to interruption of a discharge path originating at higher levels. Although Zbrozany (1960), studying defence reactions in the cat by means of combined stimulation and coagulation experiments, concluded that the stria terminalis constitutes the activating route from hypothalamus to amygdala, additional experiments are necessary to further our knowledge of the direction of conduction within the activating routes at forebrain and brain stem levels. Our knowledge of the efferent routes to effector structures, routes originating possibly at various levels of this system, also requires further investigation.

SUMMARY

1. High-frequency coagulation was carried out in subcortical structures governing defensive behaviour in forebrain and brain stem in freely-moving cats, and was combined with electrical stimulation at various levels of the reactive field.

2. The growling and growling-hissing responses elicited by electrical stimulation of the amygdala were abolished by ipsilateral coagulation of either of the 'hissing zones' situated in the central grey matter of the mid-brain and in the hypothalamus respectively; these responses were not affected by contralateral lesions.

3. The hissing and flight reactions elicited from the hypothalamus remained unaltered after bilateral coagulation of the amygdala that left the pyriform cortex intact.

4. A unilateral coagulation carried out at the level of the stria terminalis bed, interrupting the route between forebrain and brain-stem active areas, suppressed the affective pattern obtained from the ipsilateral amygdala, but did not abolish the pattern produced from the ipsilateral hypothalamus.

5. Changes in the emotional behaviour of the animals, which varied in degree according to the level of destruction, were only seen after bilateral lesions. Bilateral lesions in the 'hissing zone' of the mid-brain produced transient reduction of spontaneous activity and permanent impairment of aggressive reactions. Bilateral coagulation of the 'hissing zone' of the hypothalamus only resulted in transient reduction of spontaneous activity

and did not affect aggressive behaviour. Bilateral amygdalectomy did not affect the mechanisms subserving spontaneous activity and defensive behaviour.

6. From the evidence furnished by this study and previous data it is concluded that the subcortical system governing defensive behaviour is organized in forebrain and brain stem at three levels of progressively increasing importance. These stations are situated in amygdala, hypothalamus, and central grey matter of the mid-brain.

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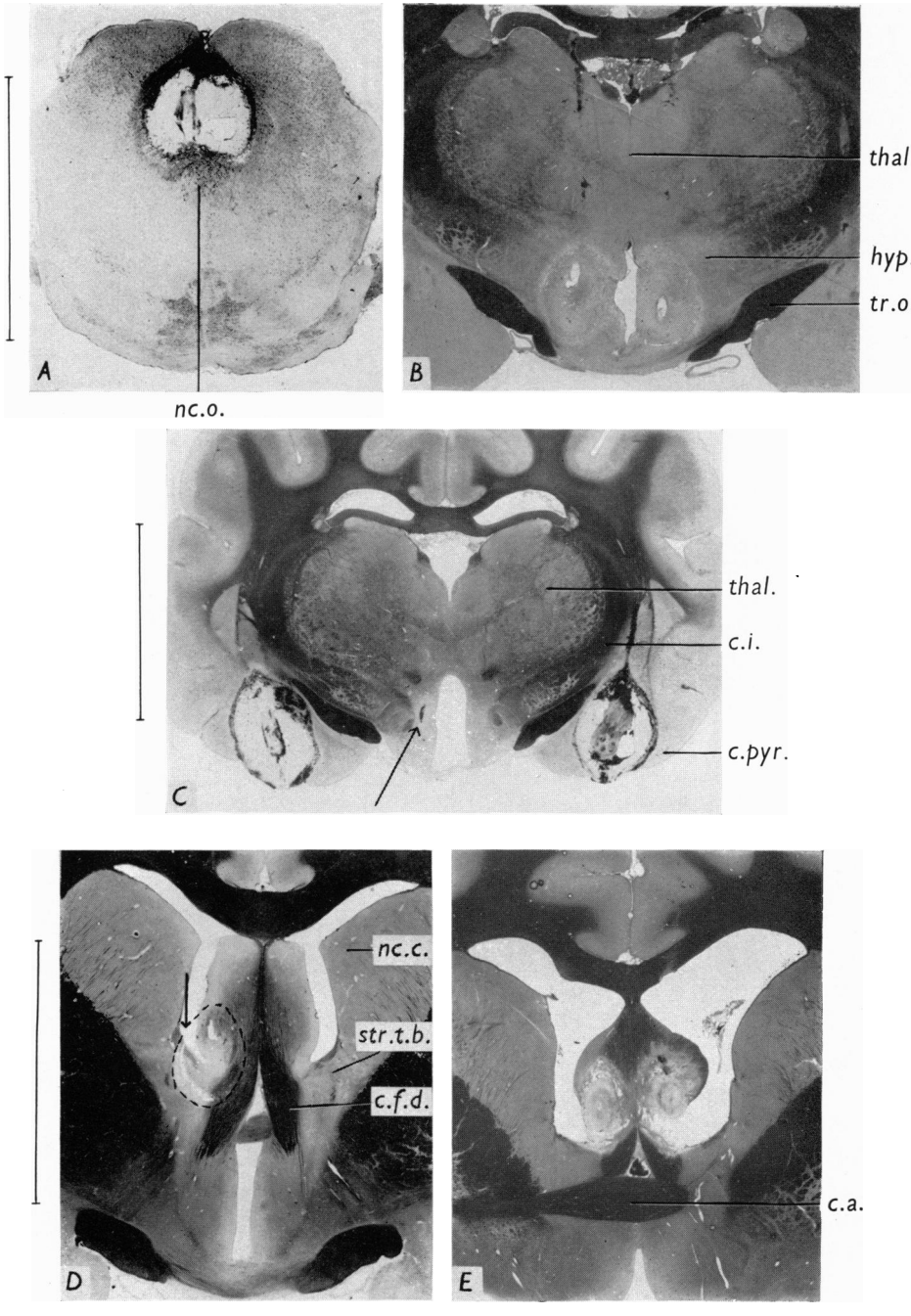
A
nc.o.
B
thal.
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C
thal.
c.i.
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D
nc.c.
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c.f.d.
E
c.a.

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## EXPLANATION OF PLATE

Photomicrographs of frontal sections showing site and dimensions of lesions. Figs. *A–E* correspond to the frontal planes indicated in Text-fig. 1. Calibration, 10 mm.

*A*, Bilateral coagulation of the 'hissing zone' in the central grey matter of the mid-brain that prevented the development of the growling-hissing pattern from the amygdala. Nissl stain (chronic expt. 109).

*B*, Bilateral coagulation of the 'hissing zone' in the hypothalamus resulting in similar impairment. Heidenhain stain (acute expt. 112).

*C*, Bilateral coagulation of the amygdala, leaving most of the pyriform cortex intact. The lesions did not affect the response patterns produced from the hypothalamus. Arrow indicates site of stimulating electrode in the 'hissing zone' of the hypothalamus. Heidenhain stain (chronic expt. 118).

*D*, Unilateral coagulation in the stria terminalis bed that prevented the development of the growling-hissing pattern produced from the amygdala on the same side. Note: at arrow the lesion severs the stria terminalis at its entrance to the bed (acute expt. 131).

*E*, Bilateral coagulation severing both fornices but leaving the stria terminalis bed undestroyed. Lesions without effect on the growling-hissing pattern from the amygdala (acute expt. 133).

*c.a.*, anterior commissure; *c.f.d.*, descending column of the fornix; *c.i.*, internal capsule; *c.pyr.*, pyriform cortex; *hyp.*, hypothalamus; *nc.c.*, caudate nucleus; *nc.o.*, oculomotor nucleus, *str.t.b.*, stria terminalis bed; *thal.*, thalamus; *tr.o.*, optic tract.