J. Anat. (1975), **120**, 3, pp. 439–484
With 38 *figures*
Printed in Great Britain

Corticobulbar fibres in the North American opossum (*Didelphis marsupialis virginiana*) with notes on the Tasmanian brush-tailed possum (*Trichosurus vulpecula*) and other marsupials

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(*Accepted* 12 *February* 1975)

INTRODUCTION

Previous reports from our laboratory have described cortical projections to brain stem sensory nuclei (Martin & West, 1967) and to certain areas of the midbrain (Martin, 1968) in the North American opossum. However, the origin of many pathways is still in doubt. Since submission of our earlier papers describing results obtained by the Nauta technique, the more sensitive Fink–Heimer method has become available (Fink & Heimer, 1967) and a large number of brains with cortical lesions have been processed by variants of that method. Several of those brains provide evidence for connexions not previously recognized and because of the increased size of the collection, as well as new information relative to the size of somatosensory cortical representation (Pubols, Pubols, Dipette & Sheely, 1975), additional insight has been gained into the origin of both the projections described previously and those seen more recently. Also, autoradiographic fibre tracing methods have come into common use and have added a technique to our armamentarium which does not depend on a pathological process, but rather on the normally occurring phenomenon of axon transport. Because of the possibility that some degenerating fibres might not impregnate after the survival times employed, the autoradiographic method has been used in an attempt to cover that eventuality, as well as to verify the connexions demonstrated by degeneration methods (see Cowan *et al.* 1972).

We have also reported previously on corticobulbar connexions in the brush-tailed 'possum' or phalanger (Martin, Megirian & Roebuck, 1971; Martin & Megirian, 1972), a species chosen for study because, unlike the generalized American opossum, it is specialized for arboreal life. However, we have since accumulated material which has allowed us to detail the origin of these connexions more precisely and, in addition, we have gained some information on the same connexions in the macropod Tasmanian potoroo and the carnivorous Tasmanian native cat. The results obtained from these Tasmanian species (particularly the brush-tailed possum) are described briefly herein so that comparisons can be made with the more extensively

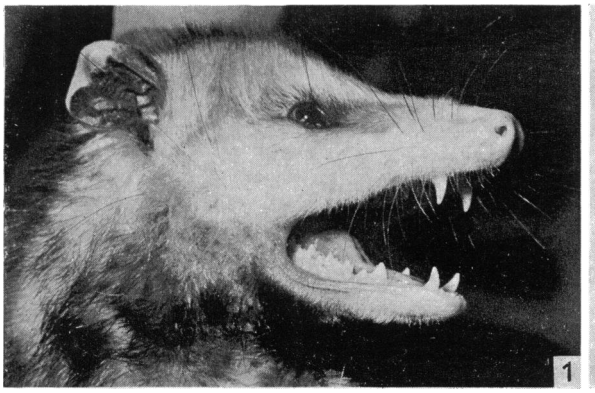
studied North American opossum. It is our belief that comparison between such widely divergent species will reveal patterns of cortical connexions which are common to marsupials and will highlight those which reflect species specialization.

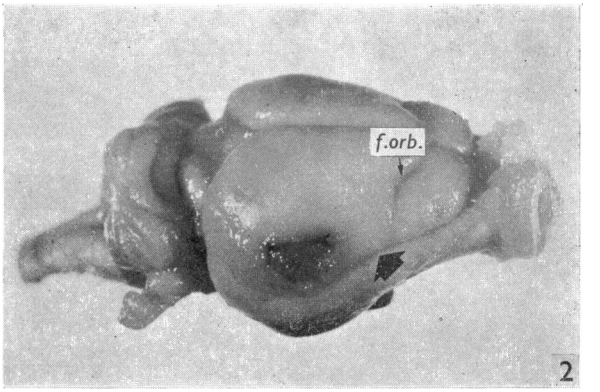
MATERIALS AND METHODS

The present report is based primarily on the results obtained from 65 opossum brains with lesions of functionally (Lende, 1963*a*, *b*; Pubols *et al.* 1975, see Fig. 3, present paper) and anatomically (Gray, 1924; Walsh & Ebner, 1970) distinct neocortical areas. The lesions were made either by electrocautery or by gentle aspiration and the brains were processed by either the Nauta–Gygax (1954) or the Fink–Heimer (1967) techniques for degenerating axons and their terminal endings. In a number of cases lesions were made in different areas on both sides so that the degeneration could be compared in brains subjected to the same technical variables. In most of the brains every fifth section was stained for Nissl substance in order to identify both the extent of the lesion and the regions containing axonal degeneration. The Nissl stained material was also used to map the extent of thalamic retrograde degeneration and/or gliosis, which, together with other criteria, was used as a control for the lesion placement. In selected cases appropriate sections were traced on Bristol board by means of a projector and the degeneration plotted on the drawing from the microscope. In general, brains from animals which were maintained post-operatively for 10–14 days showed the best impregnation of degenerating corticobulbar fibres, although in several cases time intervals of 2–7 days were employed to control for the possibility that some degenerating fibres might not impregnate at longer survival times. Terminal degeneration in the present study was defined in its usual light microscopic sense, i.e. fragmented axons which have left their main trajectory and either course in an obviously random fashion or present as dense argyrophilic spheroids.

For the autoradiographic part of the study 12 adult opossums were used. One or more injections of tritiated leucine were made in specific cortical areas (0·4 μl, concentrated at 50 μCi/μl), using a Hamilton syringe attached to a microdrive. Each injection was made over a period of approximately $\frac{1}{2}$ hour and the needle was kept in the injection site for at least 10 minutes before withdrawal. The animals were maintained for 1–10 days before being killed by intracardiac perfusion with a formol saline solution. Frozen sections of the brain were cut, mounted, coated with NTB-2 Kodak liquid emulsion diluted with an equal quantity of distilled water, and then stored under refrigeration in light-tight containers for 2–6 weeks. After the exposure period the slides were developed, stained with thionin and coverslipped. The sections were examined by both light and dark field microscopy and search was made for the presence of areas of silver grains which were above background density.

Comparisons with other marsupials were made from the brains of 53 brush-tailed possums (*Trichosurus vulpecula*), 11 potoroo (*Potorous apicalis*) and 14 Tasmanian native cats (*Dasyurus viverrinus*) which had been subjected to neocortical lesions at the University of Tasmania. The animals were allowed to survive for 7–14 days before being killed by perfusion with a formol saline solution and the removed brains and spinal cords were shipped in the appropriate fixative to The Ohio State University for processing. In most cases variations of the Fink–Heimer method (1967) were employed and every fifth section stained for Nissl substance.





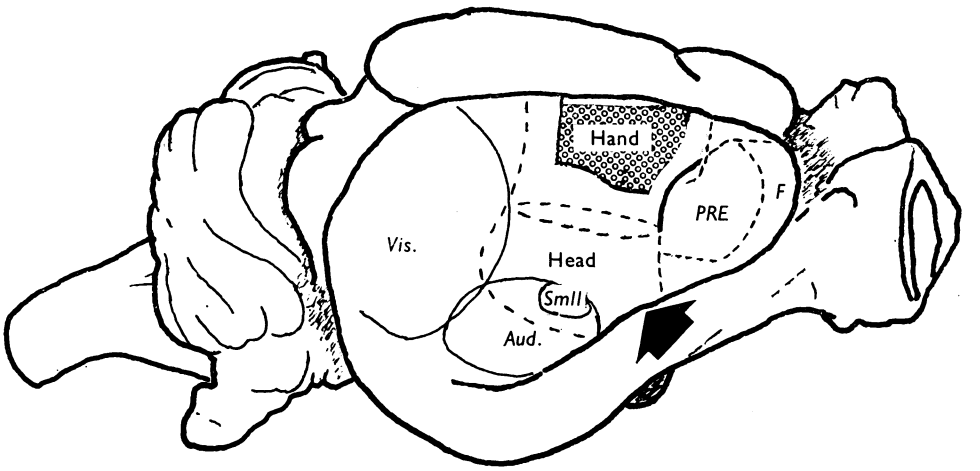


Fig. 1. The American opossum.

Fig. 2. Lateral view of an opossum brain with a large auditory lesion (P-299). The orbital (*f.orb.*) and rhinal (arrow) fissures are indicated.

Fig. 3. Drawing of an opossum brain. The somatosensory–motor area for the forelimb (Hand) and head are outlined (broken lines) as revealed by surface recordings and stimulation techniques (Lende, 1963*a*, *b*). The smaller region (small circles) represents the somatosensory forelimb representation as mapped with microelectrodes (Pubols *et al.* 1975). The secondary somatosensory area (*SmII*) as well as the auditory (*Aud.*) and visual (*Vis.*) regions are also outlined (from Lende, 1963*a*). For additional reference the rhinal fissure is shown by the arrow and the preorbital (*PRE*) and frontal (*F*) cortices are outlined as reconstructed by Walsh & Ebner (1970).

RESULTS

Since the American opossum (Fig. 1) provides the basis for this study, the results obtained in that species will be described first. Observations from experiments designed to elucidate the projections from specific cortical areas will be dealt with first, followed by a description of the results obtained from several brains subjected to unilateral removal of all neocortex.

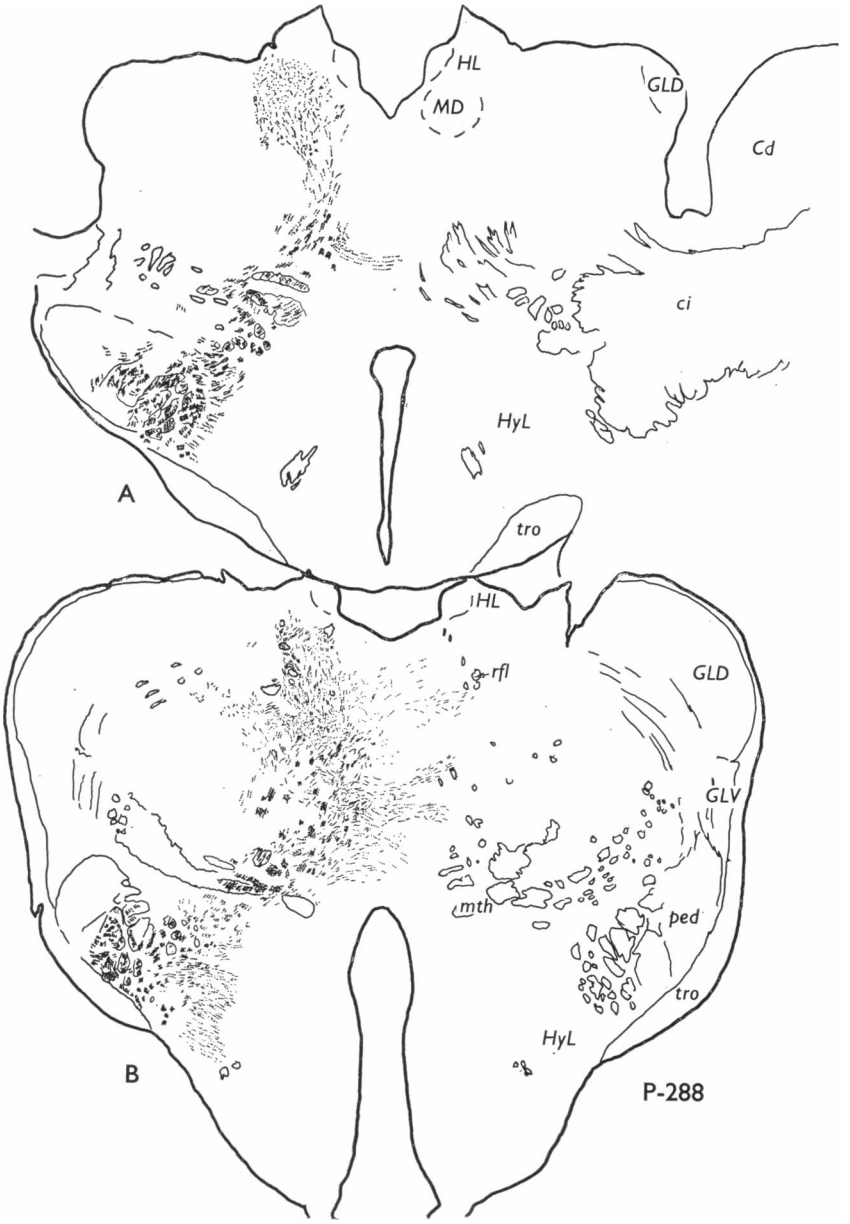


Fig. 4. Drawings of two sections through the thalamus (rostral A, caudal B) of an animal with a lesion of the preorbital area (see insert, Fig. 5). Axonal degeneration is plotted on the reader's left where it is illustrated either by broken, wavy lines (fibres of passage) or dots (fibres cut in cross section and/or terminals). Several nuclei and fibre bundles are labelled on the reader's right. For abbreviations in this and succeeding figures see p. 484.

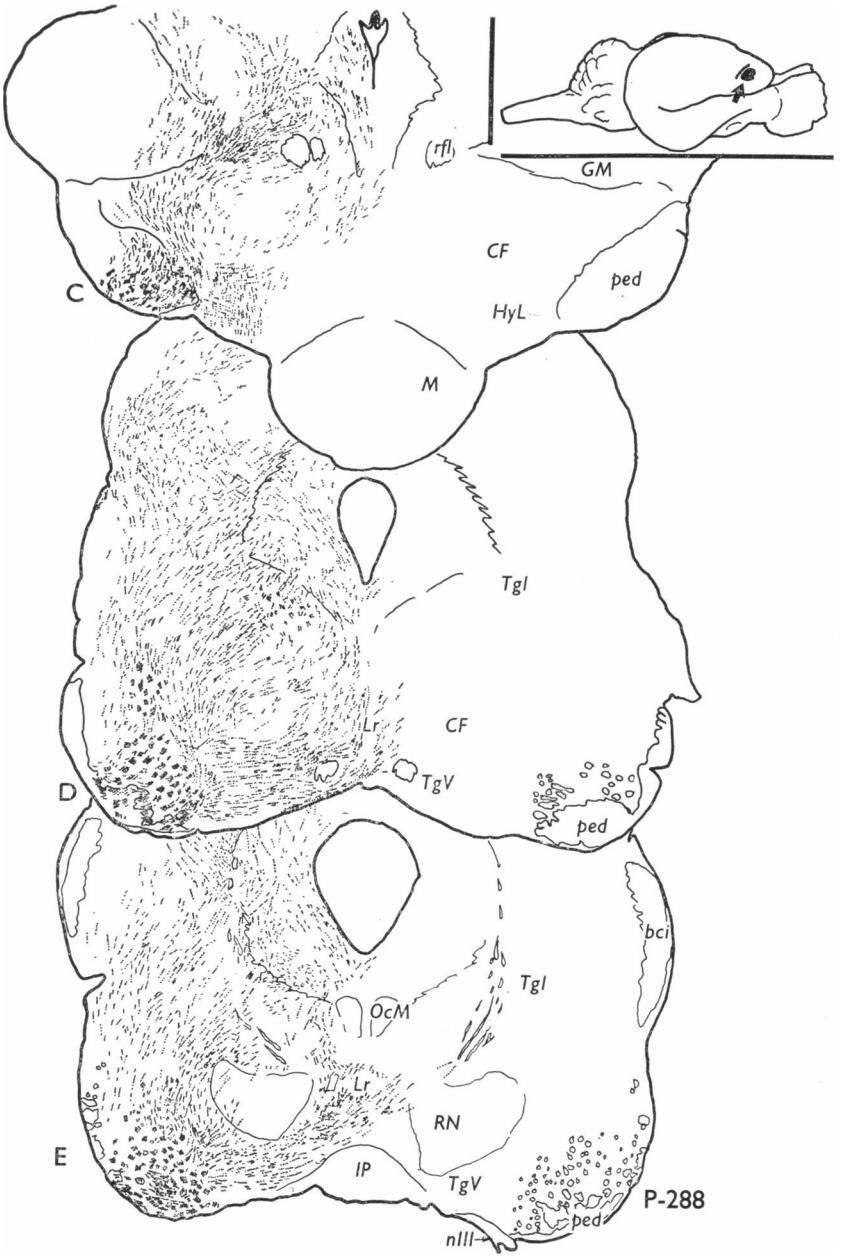


Fig. 5. Drawings of three sections through the thalamic–midbrain junction (C) and midbrain (D, E). The preorbital lesion is indicated by the arrow in the insert (upper right) and axonal degeneration is illustrated as described for Fig. 4. Several nuclei and fibre tracts are labelled on the right.

Projections of the cortex rostral to the orbital fissure (terminology of Elliot Smith, 1902)

As reported previously (Martin, 1968) lesions rostral to the orbital fissure (the fissure labelled in Figs. 2 and 23) produced axonal degeneration within the dorsomedial nucleus of the thalamus (particularly laterally), as well as within the parafascicular complex, the nuclei reuniens, paracentralis and ventromedialis, the lateral hypothalamus and certain basal forebrain areas (see Fig. 4 for an illustration of some of these). That at least a portion of the degeneration within the thalamus was orthograde in character was indicated by the presence of abundant silver grains over the same thalamic nuclei in the autoradiographic experiments.

A large part of the cortex rostral to the orbital sulcus has been referred to as the preorbital area (Gray, 1924; Tobias & Ebner, 1973, see Fig. 3). In brains with destruction of that area (insert, Fig. 5), degenerating fascicles were located within approximately the medial half of the basis pedunculi (Figs. 4, 5). Fragmented axons left that bundle and coursed medially into the medial substantia nigra and ventral tegmental area (Fig. 5D, E) where they interlaced with degenerating fibres which had descended to the same areas within the medial forebrain bundle. Many such fibres continued through the ventral tegmental area into both the nucleus linearis (some crossing to the opposite side) and the periaqueductal grey (Fig. 5D, E). Bundles of degenerating axons curved dorsally and laterally from the basis pedunculi (tending to circumvent the red nucleus, Fig. 5E) and were distributed to more dorsal and caudal midbrain targets (Fig. 5D, E). Degenerating axons arborized in a terminal fashion within the ventral tegmental area, the linear nucleus, the ventral and lateral periaqueductal grey, the dorsal and lateral tegmentum, the cuneiform area and the superior colliculus (Fig. 5D, E). Degenerating axons in the superior colliculus were abundant after large lesions and, for the most part, they were distributed deep to the stratum opticum. Although most degenerating fibres skirted the red nucleus (medially and laterally), some passed through it and terminal debris could not be excluded. Several brains were available with lesions rostral to that shown in Fig. 5, although in most of them there was some involvement of the preorbital area. In each case a similar pattern of degeneration was present within the ventral and medial midbrain, although the amount of axonal debris in the dorsolateral tegmentum and superior colliculus varied considerably. A distinct ventral area of the periaqueductal grey contained relatively dense terminal degeneration in most cases. This area has been referred to as the ventral nucleus of the periaqueductal grey (Martin, 1968; Oswaldo-Cruz & Rocha-Miranda, 1968).

In all cases degenerating fascicles left the 'pyramidal' tract and terminated within the basilar pons (particularly in the dorsomedial part of the medial nucleus, King, Martin & Biggert, 1968; Yuen, Dom & Martin, 1974), as well as within the superior central and reticulotegmental nuclei. Most of the terminals within the reticulotegmental nucleus were located just medial and dorsal to the medical lemniscus. More caudally a few degenerating fibres either entered the pontine raphe or trickled into the adjacent reticular formation. In brains with relatively large lesions, fragmented axons could be followed through the pyramidal decussation, but they were sparse and therefore difficult to trace with certainty at spinal levels.

Two brains were available with ^{3}H leucine placements just rostral to the orbital

sulcus and in each silver grains were abundant over the expected thalamic nuclei, but sparse at midbrain levels (24 hour survival). Silver grains were not numerous over neurons in the deeper cortical layers in either case, perhaps accounting for the relatively meagre brainstem labelling. In spite of this problem, evidence for a projection to the ventral tegmental area and linear nucleus was present.

Projections of the limb motor–sensory cortex of the opossum

The lesion on the right side of P-156 (insert, Fig. 8) was grossly limited to the forelimb motor–sensory area demarcated by Lende (1963*a*, *b*), and the somatosensory forelimb field defined by Pubols *et al.* (1975). Although there was some white matter undercutting, owing to the thinness of the cortex and its unique blood supply (Wislocki & Campbell, 1937), the case provided valuable information concerning forelimb cortical projections and was the basis for the following description. There was a small face area lesion on the left side of this brain (see Lende, 1963*a*, *b*; Pubols *et al.* 1975; Fig. 3, present account) which allowed the observer to compare the distribution of degeneration on the two sides. The forelimb cortical damage resulted in degeneration of axons within the rostral part of the ventral thalamus (cerebellar targets, Walsh & Ebner, 1973; Martin, King & Dom, 1974) and, more caudally, within the forelimb somatosensory thalamus (Erickson *et al.* 1964; Pubols & Pubols, 1966; Sousa, Oswaldo-Cruz & Gattass, 1971; Hazlett, Dom & Martin, 1972; Walsh & Ebner, 1973) where it was more extensive (left side of Fig. 6A, B). As was to be expected after a 2 week survival time, both orthograde degeneration and 'dust' which we believe is entitled to be designated 'retrograde dust'* were present, as well as cell changes with accompanying gliosis (Nissl stained material). The apparent sparing of the face cortex was verified by the absence of conspicuous terminal degeneration within the corresponding area of the ventral thalamus (compare the left side of Fig. 6B with Erickson, Jane, Waite & Diamond, 1964; Pubols & Pubols, 1966; Sousa *et al.* 1971). There was terminal debris, however, within the small hindlimb area of the ventral thalamus, probably as a result of fibre undercutting by the lesion. Further description of thalamic degeneration is not germane to this report.

At midbrain levels degenerating axons left the basis pedunculi (not shown in Fig. 7) and, together with similar fibres that descended through the thalamus, were distributed to the ipsilateral red nucleus and the midbrain tegmentum (left side, Fig. 7C). Such fibres were particularly numerous within the lateral tegmentum and the area defined as the intercollicular nucleus by Olszewski & Baxter (1954) and Mehler (1969). Many of the fibres to the intercollicular area took a curvilinear route to their destination and coursed through the periaqueductal grey, where terminal debris was also present. Degenerating axons were distributed to the caudal pretectal nucleus and the ventrolateral superior colliculus (left side, Fig. 7C) and more caudally to the nucleus of the inferior colliculus, pars lateralis (Oswaldo-Cruz & Rocha-Miranda, 1968; see left side of Fig. 7D in the present account). The latter area appeared comparable to the external nucleus of the inferior colliculus described for the cat (see Rockel & Jones, 1973*a*, *b* for review). Many of the fibres in the ipsilateral inferior colliculus were terminal, but a considerable number crossed the midline and were distributed within the contralateral external nucleus.

* Some investigators feel that the so-called retrograde dust results from an anterograde reaction (e.g. Kalil, 1974).

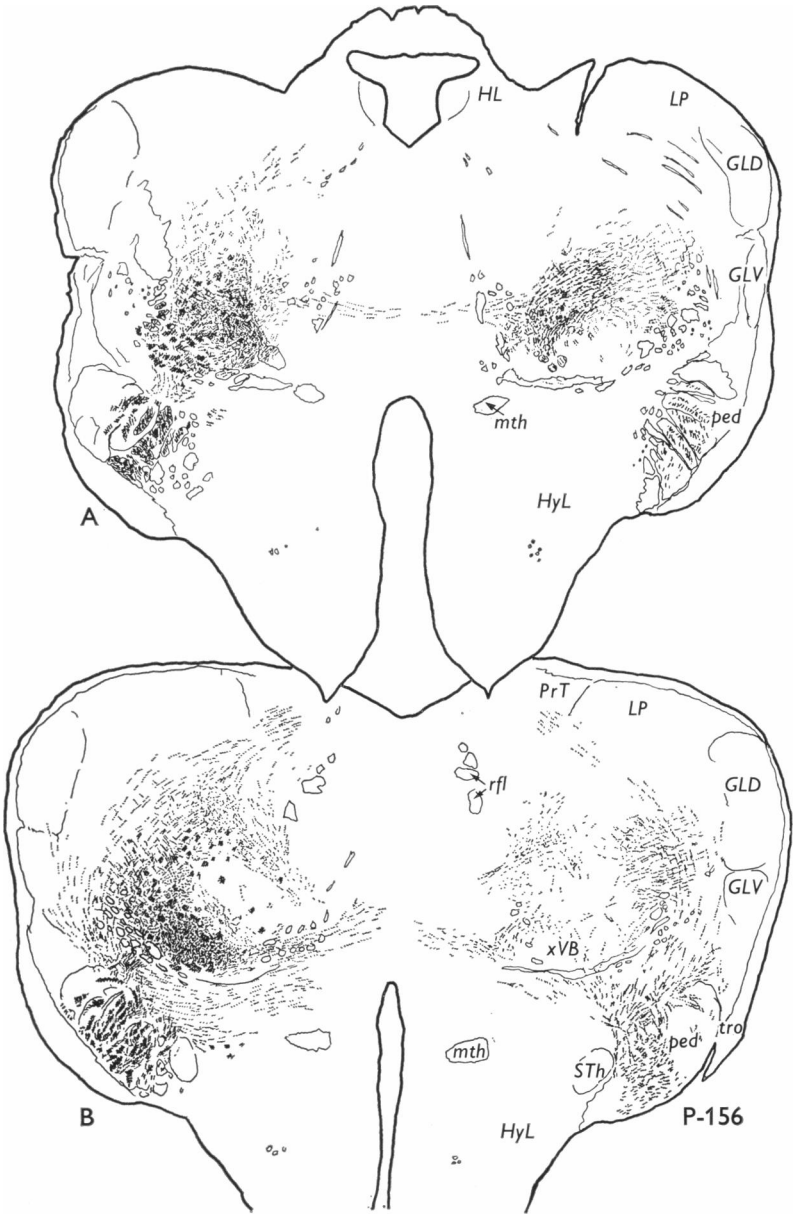


Fig. 6. Drawings of two sections through the thalamus (rostral A, caudal B) from the brain drawn in the insert of Fig. 8. The degeneration produced by the forelimb area lesion is on the reader's left, whereas that elicited by the face cortical damage is to the right. Degenerating axons are illustrated as in Fig. 4 and various structures are labelled for reference on the right.

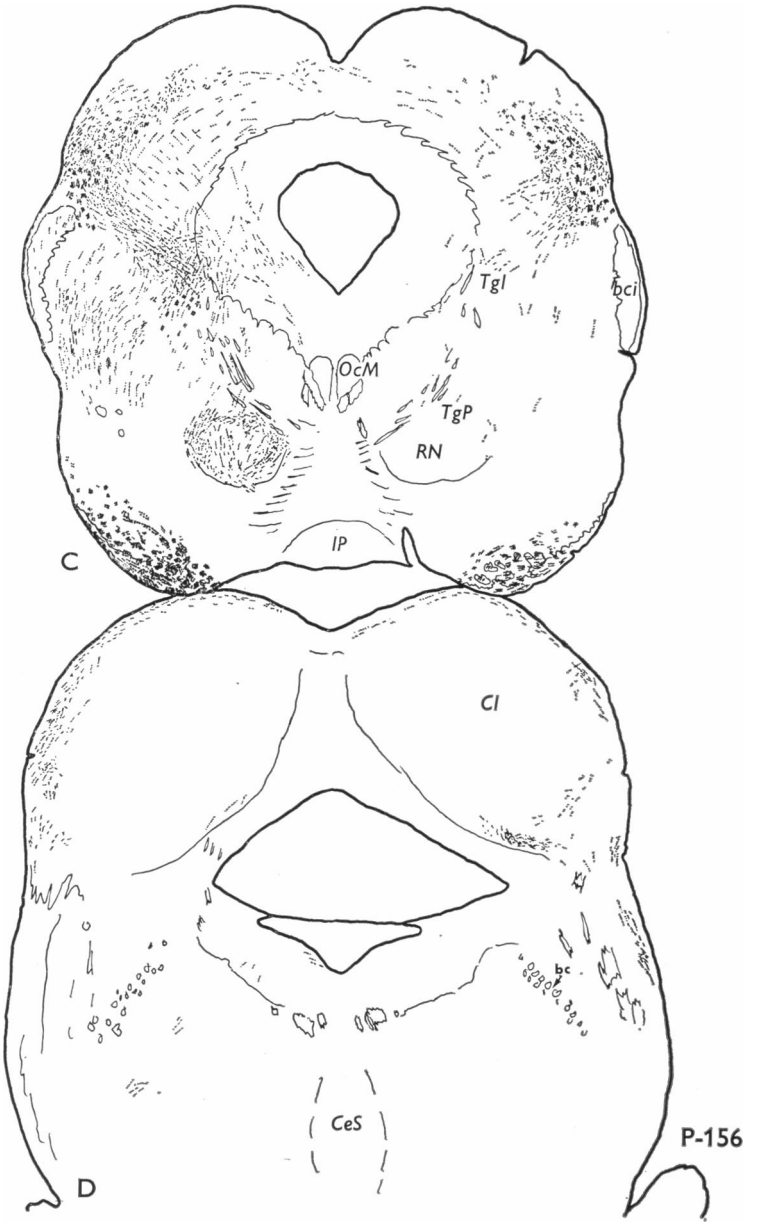


Fig. 7. Drawings of sections through the mesencephalon (rostral C, caudal D) of the brain shown in the insert of Fig. 8. Forelimb area fibre degeneration is plotted on the reader's left, whereas that elicited by face damage is similarly illustrated on the right. Degenerating axons are drawn as described for Fig. 4 and various structures are labelled on the right.

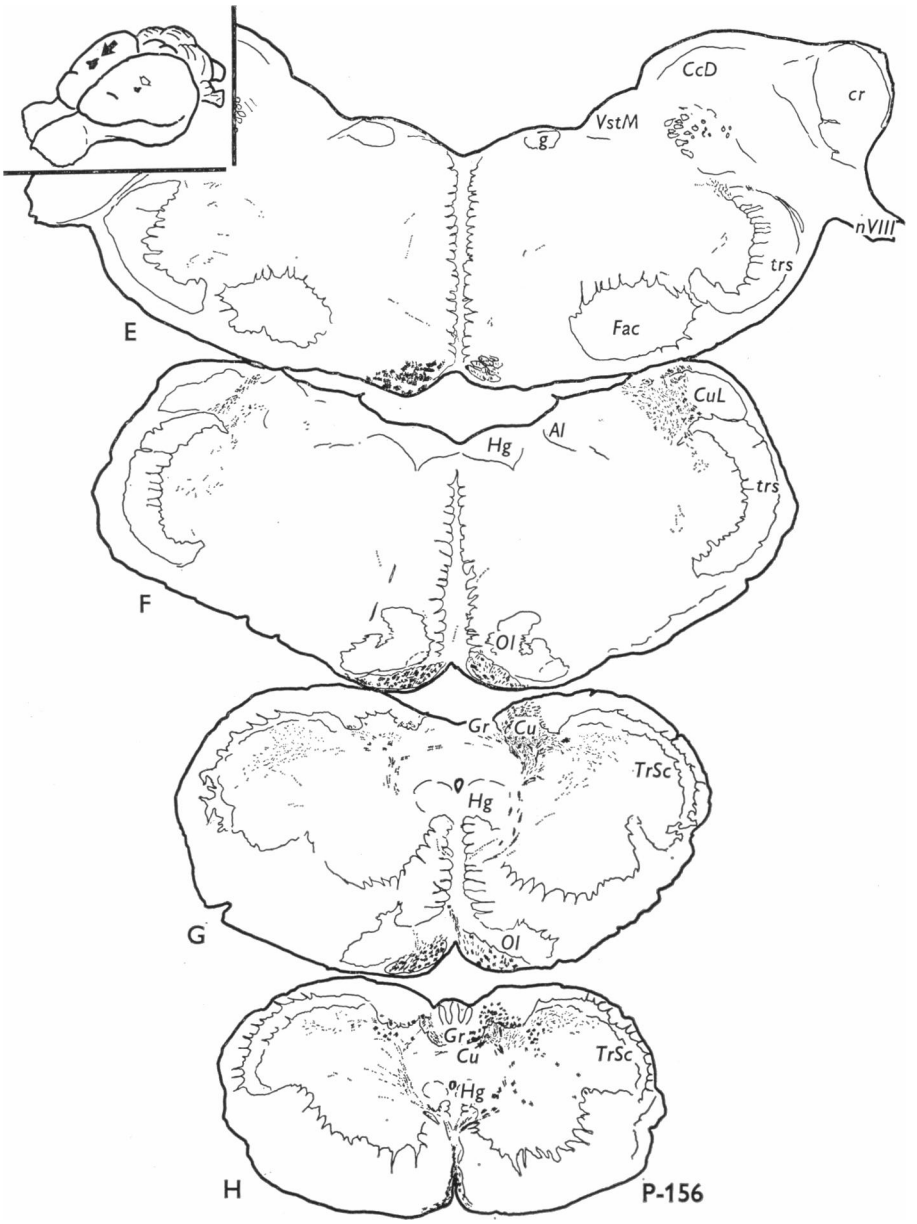


Fig. 8. Drawings of sections through the caudal pons (E) and medulla (F–H) of the brain shown in the insert (upper left). The lesions are indicated in the insert by arrows and degenerating axons are illustrated as in Fig. 4. The right side of the brainstem sections is to the reader's left (side of forelimb area lesion, black arrow in insert).

Except for those that were distributed within the basilar pons (particularly extensive within the caudal part of the lateral nucleus, see Yuen *et al.* 1974), few degenerating axons left the pyramidal tract at pontine levels (left side, Fig. 8E). At medullary levels rostral to the motor decussation (Figs. 8F, G), degenerating pyramidal fibres went to that portion of the medial accessory olivary nucleus described previously as the dorsolateral division of subnucleus c and part of subnucleus b (modified for the opossum from the terminology employed for the monkey by Bowman & Sladek, 1973; see Martin *et al.* 1975). Although a few degenerating fibres left the pyramidal tract rostral to its decussation and coursed randomly within both Roller's nucleus and the reticular formation, most of them participated in the decussation (Fig. 8H) and were distributed to the contralateral nucleus cuneatus (particularly its hilum, reader's right, Fig. 8F, G, H). In this case (unlike that illustrated in Fig. 9), some degeneration was also present within the adjacent dorsomedial part of the caudal spinal trigeminal nucleus (right side of Fig. 8F, G, H). Degeneration within the nucleus gracilis was sparse and difficult to distinguish from the coarse and obviously non-cortical debris present as a result of some unknown but apparently common neuropathy of the fasciculus gracilis. Although degenerating fibres coursed through the nucleus cuneatus to the adjacent fasciculus of the same name (descending caudally in that position as the dorsal corticospinal tract), many curved rostrally as recurrent fascicles to be distributed to portions of the nucleus cuneatus at more rostral levels. Some of the latter fibres cut through the accessory cuneate nucleus and a few of them could be followed as far rostrally as the motor facial nucleus where they occupied a position just dorsal to the spinal trigeminal tract (right side, Fig. 8E). A small group of degenerating fibres could be followed lateral to the spinal trigeminal tract at that level. Degenerating fibres crossed beneath the gracile nuclei (through the commissural part of dorsal vagal nucleus and the commissure of Cajal) from the side opposite the lesion to the ipsilateral side where it was difficult to discern their distribution because of the presence of degeneration produced by the small face lesion. However, even in brains containing only forelimb area lesions the contralateral targets of these fibres escaped detection. In the latter brains, additional degeneration (uncrossed pyramidal fibres) could be traced through the ipsilateral nucleus cuneatus to the adjacent fasciculus, but terminal fields were sparse. Although the lesion in P-302 (Fig. 9, insert) was larger than that in P-156, the distribution of degeneration was essentially the same (Fig. 9A–D). It should be noticed, however, that no degenerating axons terminated within the spinal trigeminal nuclei. Essentially the same pattern of degeneration was present in two brains with lesions limited *mainly* to the postorbital area as described by Walsh & Ebner (1970) (one example seen in Fig. 23), although at thalamic levels degeneration was not present within the hindlimb area of the ventral complex (dorsolateral extreme, Erickson *et al.* 1964; Pubols & Pubols, 1966; Sousa *et al.* 1971; Hazlett *et al.* 1972).

Several attempts were made to place lesions which were restricted to the hindlimb motor–sensory cortex (Lende, 1963*a*, *b*), but in each brain there was some extension of the lesion on to the lateral surface (e.g. left side of insert, Fig. 12). As was expected, degeneration was extensive in the hindlimb area of the ventral thalamic complex, but with some involvement of the forelimb region. Although the distribution of degeneration in the midbrain (Figs. 10, 11) was similar to that

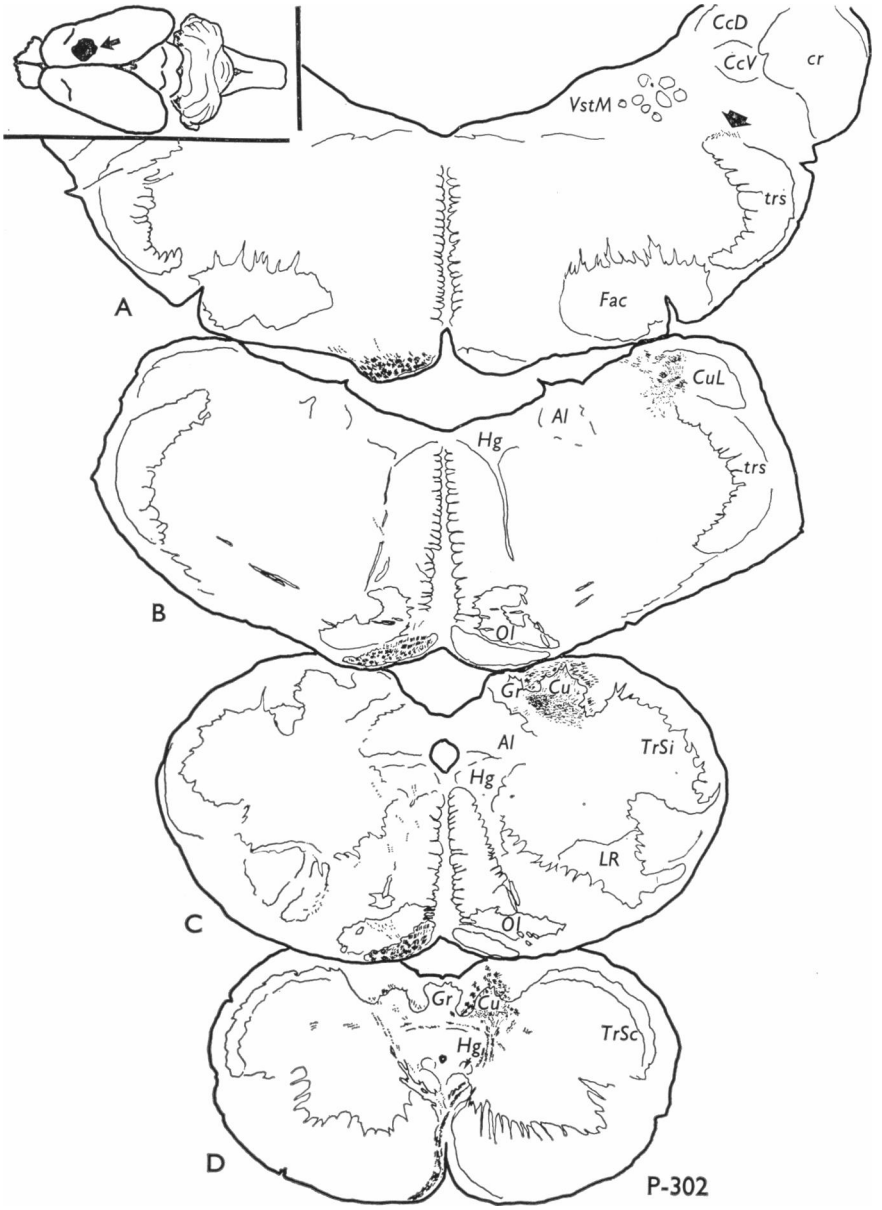


Fig. 9. Drawings of sections through the caudal pons (A) and medulla (B–D) of the brain shown in the insert (upper left). Degenerating axons are illustrated as in Fig. 4 and several structures are labelled on the reader's right.

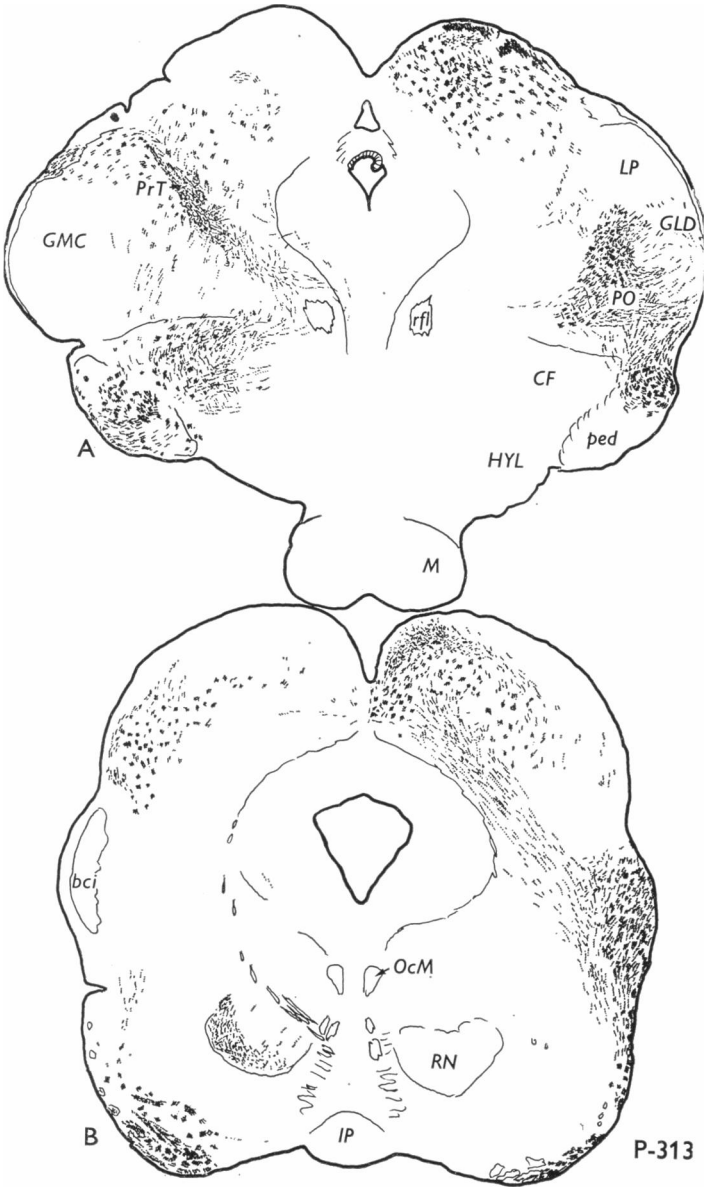


Fig. 10. Drawings of sections through the thalamic–mesencephalic junction (A) and mesencephalon (B) from the brain illustrated in the insert of Fig. 12. The degeneration produced by the hindlimb cortical damage (black arrow, insert Fig. 12) is on the reader's left, whereas that elicited by the 'auditory' destruction (open arrow, Fig. 12) is on the right. Degenerating axons are drawn as in Fig. 4 and relevant structures are labelled on both sides of the sections.

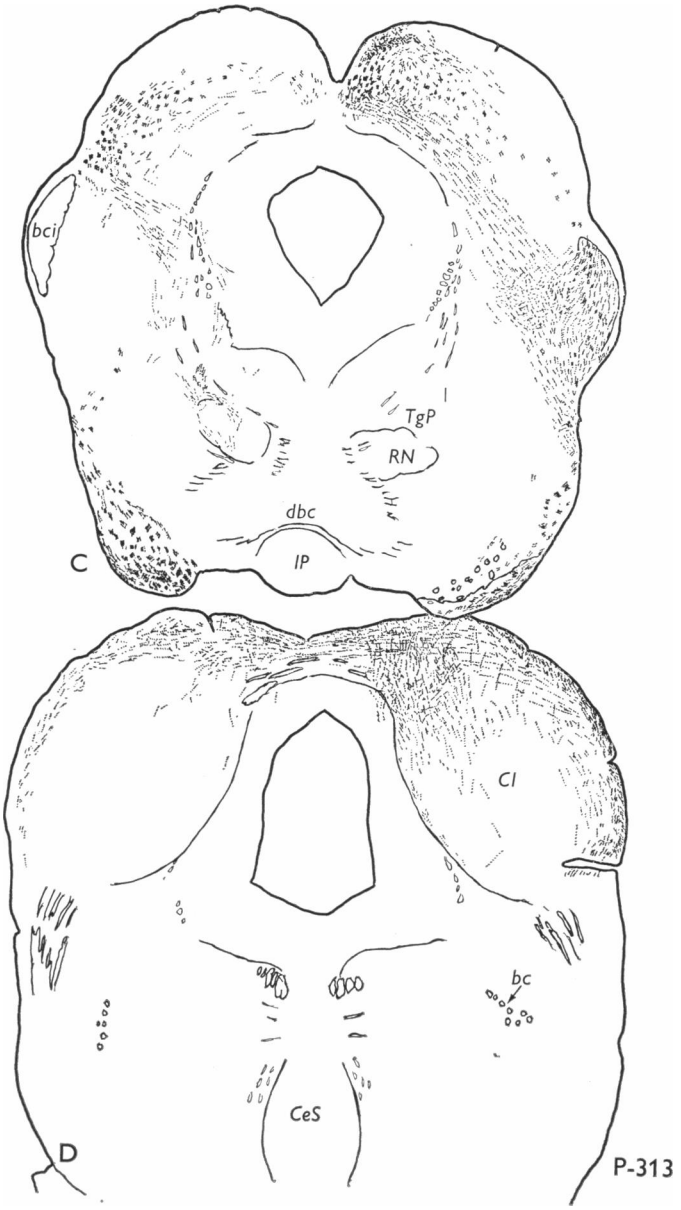


Fig. 11. Drawings of sections through the mesencephalon (rostral C, caudal D) of the brain shown in the insert of Fig. 12. The degeneration produced by the hindlimb lesion is on the reader's left, whereas that elicited by the 'auditory' damage is to the right. Degenerating axons are illustrated as in Fig. 4 and structures are labelled on the right hand side of the sections.

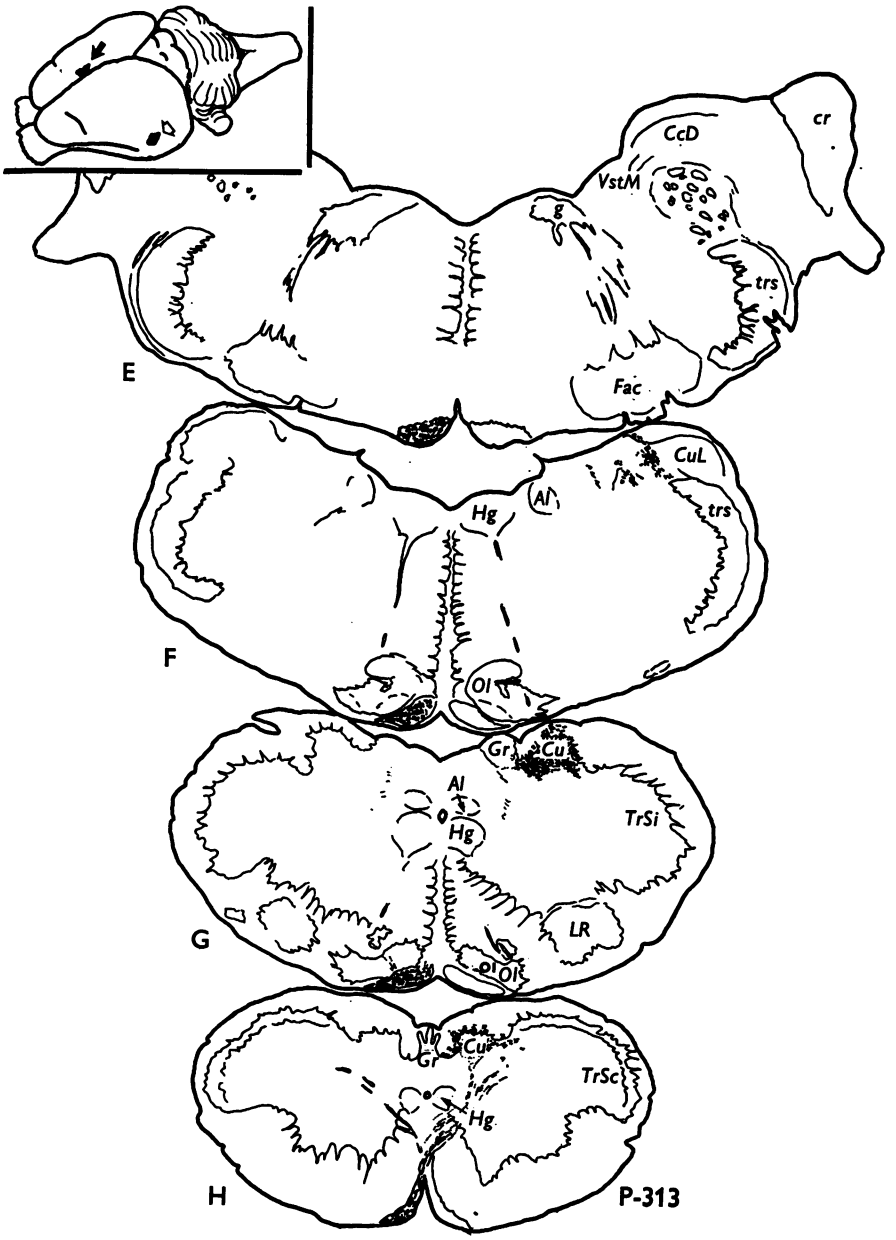


Fig. 12. Drawings of sections through the caudal pons (E) and medulla (F–H) of the brain shown in the insert (upper left). The right side of the sections is to the reader's left. Degenerating axons are illustrated as in Fig. 4 and structures are labelled on the reader's right.

described for the forelimb case, there was a *tendency* for it to avoid the medial and dorsal parts of the red nucleus rostrally (left side, Fig. 10B) and marked avoidance of its medial area caudally (left side of Fig. 11C). At pontine levels, no degeneration was present outside the basilar pons and pyramidal tract (Fig. 12E). Although degenerating axons could be traced caudally through the pyramidal decussation and into the spinal cord and a few entered the ipsilateral inferior olive and lateral reticular nuclei, unequivocal medullary terminal degeneration was present only within the nucleus cuneatus (Fig. 12G, H).

In one case (P-139, not shown) the lesion was restricted to the caudal 'parietal' cortex (Walsh & Ebner, 1970) outside of the limb somatosensory area as mapped by microelectrode techniques (Pubols *et al.* 1975). At thalamic levels degenerating axons were distributed to the posterior thalamic nucleus (as defined by Oswaldo-Cruz & Rocha-Miranda, 1968) and, more caudally, to the pretectal complex, the lateral superior colliculus and the basilar pons. Degeneration was not discernible caudal to the basilar pons.

Nine brains in our collection had leucine placements in forelimb motor–sensory areas (e.g. Figs. 24, 25). In each brain silver grains filled the appropriate thalamic nuclei after processing by routine autoradiographic procedures, but they were much less numerous within the brainstem (1–10 days survival, 2–6 weeks exposure time). However, the number of grains was clearly above background in the red nucleus, the nucleus intercolliculus, the basilar pons and the nucleus cuneatus. In each brain, grains were present over the nucleus gracilis, but they were only barely above background level. Most of the placements spilled over slightly on to the face area, possibly accounting for the finding of silver grain over the dorsomedial part of the caudal spinal trigeminal nucleus. It should be noted that all of the grains in a particular nucleus may not be in terminals since they were also found in the basis pedunculi and pyramidal tract at all survival times.

Projections of the face motor–sensory cortex of the opossum

Lesions of the face motor–sensory cortex as defined by Lende (1963*a*, *b*, Fig. 3, present study) produced extensive degeneration within both the face area of the ventral thalamic nucleus (Erickson *et al.* 1964; Pubols & Pubols, 1966; Sousa *et al.* 1971) and the sensory trigeminal nuclei of the brainstem. If the lesion was limited to the somatosensory face region defined by microelectrode methods (Pubols *et al.* 1975), *terminal* degeneration was not present in either the limb area of the ventral thalamic complex or the dorsal column nuclei.

The pattern of degeneration produced by the face lesion on the left cortex of P-155 (right insert, Fig. 14) was representative of that seen in several cases and provided the basis for the following description. This brain served an additional purpose in that it had a small forelimb lesion on the opposite side (left insert, Fig. 14) and the degeneration produced by the two lesions could be compared in sections subjected to similar technique variables. As mentioned above, degeneration was extensive in the face area of the ventral thalamic nucleus (as well as within other adjacent thalamic nuclei) and it traversed both the rostral cerebellar (motor) and the caudal somatosensory areas (see review by Walsh & Ebner, 1973 and Martin *et al.* 1974).

At rostral midbrain levels numerous degenerating fibres swept dorsally from the

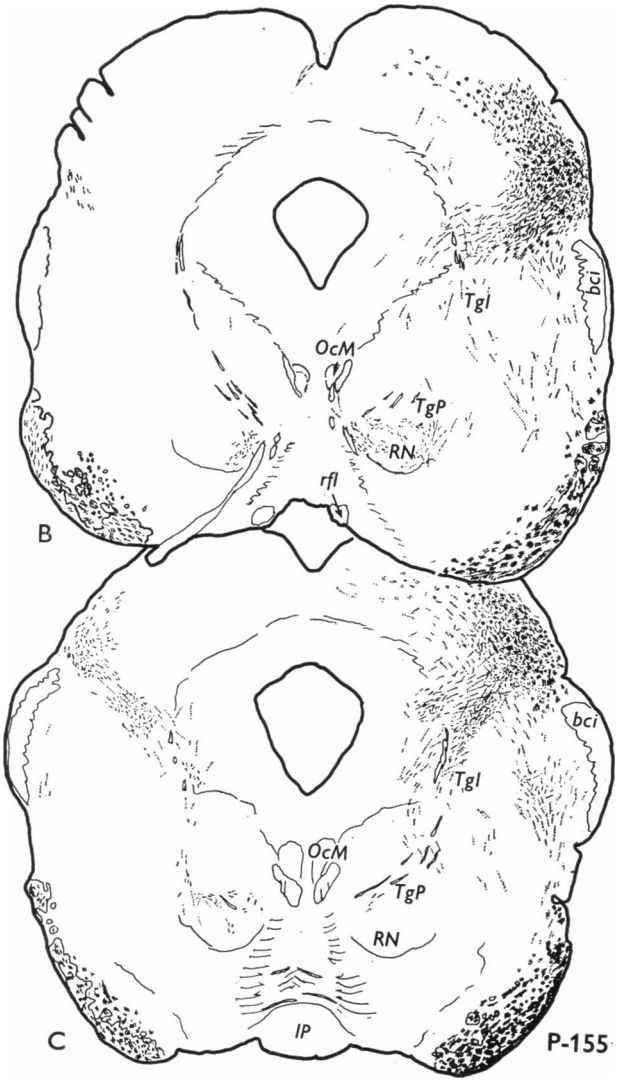


Fig. 13. Drawings of sections through the mesencephalon (rostral B, caudal C) of the brain shown in the inserts of Fig. 14. The forelimb area degeneration is on the reader's left, whereas that from the larger face damage is on the reader's right. Degenerating axons are illustrated as in Fig. 4 and various structures are labelled on the reader's right.

basis pedunculi and were distributed to the caudal pretectal nucleus and the superior colliculus. Degeneration was particularly extensive within the ventrolateral superior colliculus (right side, Fig. 13B, C). Degenerating fibres also left medially from the basis pedunculi, distributing to the fields of Forel, the red nucleus (minimal) and the adjacent tegmentum (right side of Fig. 13B, C). More caudally degenerating axons continued to be distributed to the superior colliculus and terminal fields were present within the intercollicular nucleus, the lateral periaqueductal grey and the external

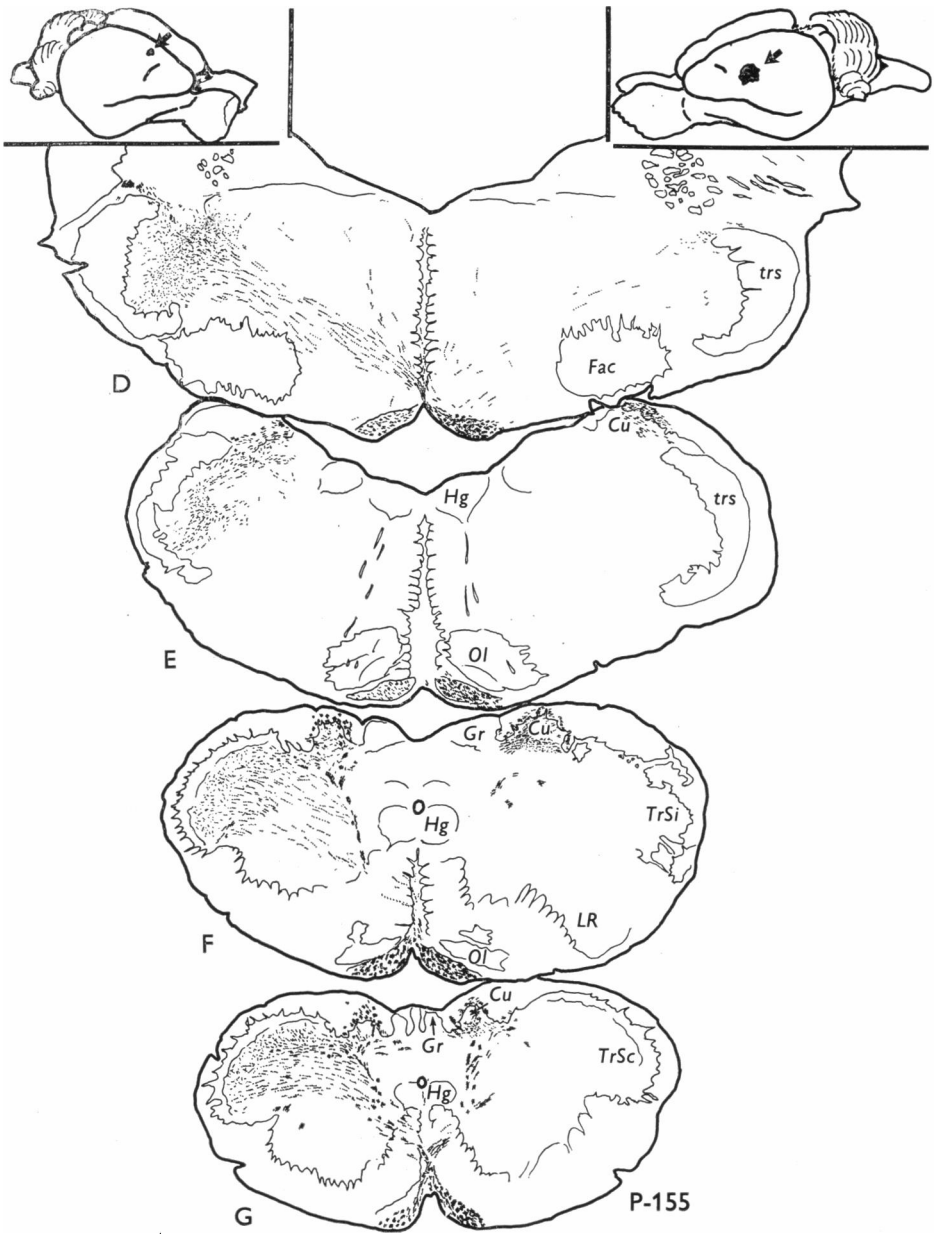


Fig. 14. Drawings of sections through the caudal pons (D) and medulla (E–G) of the brain shown in the inserts. The right side of the brainstem sections is on the reader's left. Degenerating axons are illustrated as in Fig. 4 and structures are labelled on the reader's right.

nucleus of the inferior colliculus, as the latter region has been labelled in the cat (Rockel & Jones, 1973*a*, *b*). The gentle curve of degenerating axons from the basis pedunculi to the intercollicular area was similar to that seen in the forelimb cases (Fig. 13B, C).

Degenerating pyramidal fibres continued caudally and were distributed to the basilar pons (see Yuen *et al.* 1974 for details). However, unlike those brains with limb motor–sensory lesions, numerous degenerating axons left the pyramidal tract between the basilar pons and the inferior olivary nucleus (Fig. 14D). Most of them either passed dorsally into the large cell portion of the raphe (where some continued into the periventricular grey and curved laterally beneath its pial surface) or passed through the tegmentum of the opposite side on their way to the parvocellular reticular formation and the adjacent oral portion of the spinal trigeminal nucleus (Fig. 14D). Degenerating axons also ramified profusely within the chief sensory trigeminal nucleus (some gaining access to it by curving out of the periventricular grey) as well as among the neurons intercalated between the exiting motor trigeminal rootlets and within the nucleus of the pontobulbar body (as that nucleus has been termed in the opossum by Oswaldo-Cruz & Rocha-Miranda, 1968). Although relatively few in number, degenerating axons left the pyramidal tract and were distributed to medial areas of the pontine reticular formation.

At medullary levels, degenerating axons were still extensive within the lateral reticular formation, where terminal debris extended into central areas of the interpolar spinal trigeminal nucleus (left side of Fig. 14E). At the pyramidal decussation, a few degenerating axons coursed *through* the nucleus cuneatus to the adjacent fasciculus (contributing a few fibres to the spinal cord), although few if any were terminal. In contrast, numerous degenerating fibres swept laterally and ended within the caudal spinal trigeminal nucleus (left side of Fig. 14F, G). Although an occasional degenerating axon was present within the pars gelatinosa, most appeared to follow its inner border and were distributed to magnocellular and reticular divisions. Many of the degenerating fibres looping back from the decussation supplied more rostral levels of the spinal trigeminal nucleus and some continued into the pons where they lay dorsal to the spinal trigeminal tract (left side, Fig. 14D). A few degenerating axons passed into the medullary raphe before turning laterally beneath the central canal, whereas others could be traced to the portion of the inferior olivary nucleus described previously, as well as to more dorsally situated reticular areas. Although degeneration was present within the ipsilateral lateral reticular nucleus it was extremely slight.

Several specimens were available with either relatively large lesions of forelimb cortex with minimal involvement of the dorsal face area, or with small ablations restricted to the latter region (e.g. see the left side of P-156, Fig. 8 for an example of the latter). In such brains the distribution of degeneration was essentially the same as reported above, although it was less abundant. However, in each case terminal degeneration in the caudal division of the spinal trigeminal nucleus was limited to its dorsal one third to half (left side, Fig. 8G, H). In one specimen (P-309, Figs. 15–17) the lesion on one side (insert, Fig. 17) was located ventrally and caudally within the face area (*SmI*), as defined by Lende (1963*a*) (perhaps also including his S-II), but with only minimal involvement of the comparable area, as outlined by Pubols *et al.*

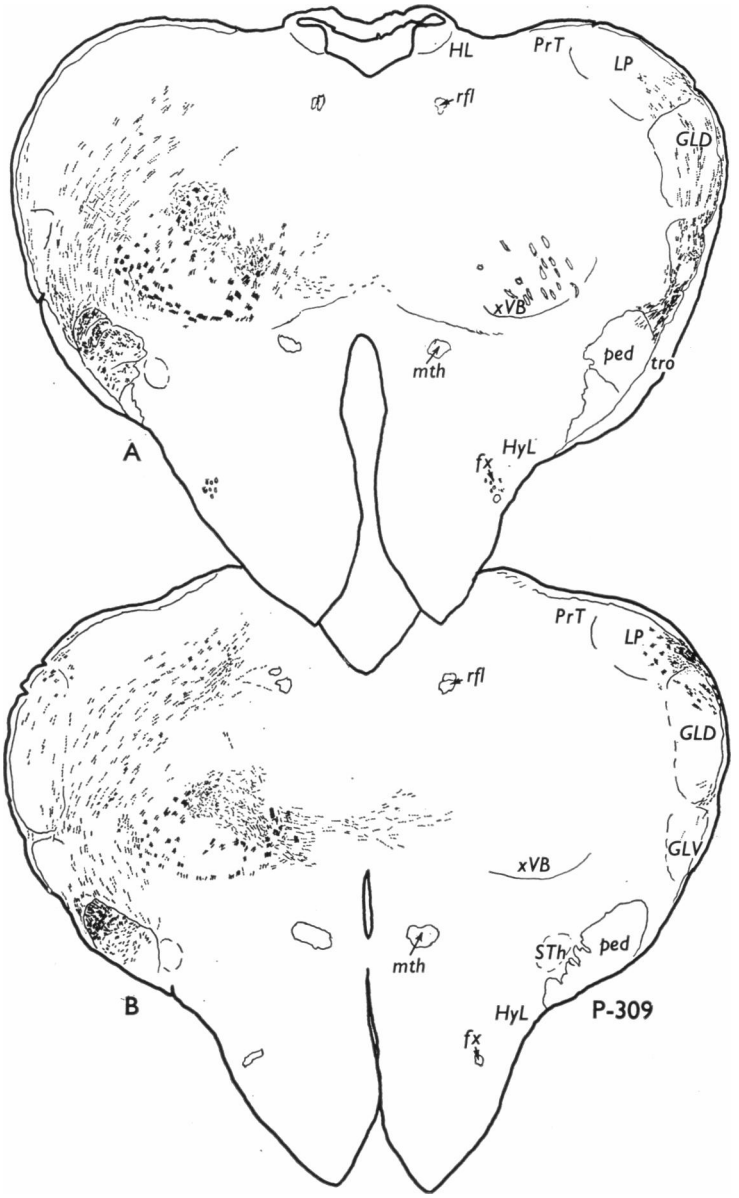


Fig. 15. Drawings of sections through the thalamus (rostral A, caudal B) of the brain shown in the insert of Fig. 17. The 'face' area degeneration is on the reader's left, whereas that from the visual cortical lesion is on the right. Degenerating axons are illustrated as in Fig. 4 and structures are labelled on the right.

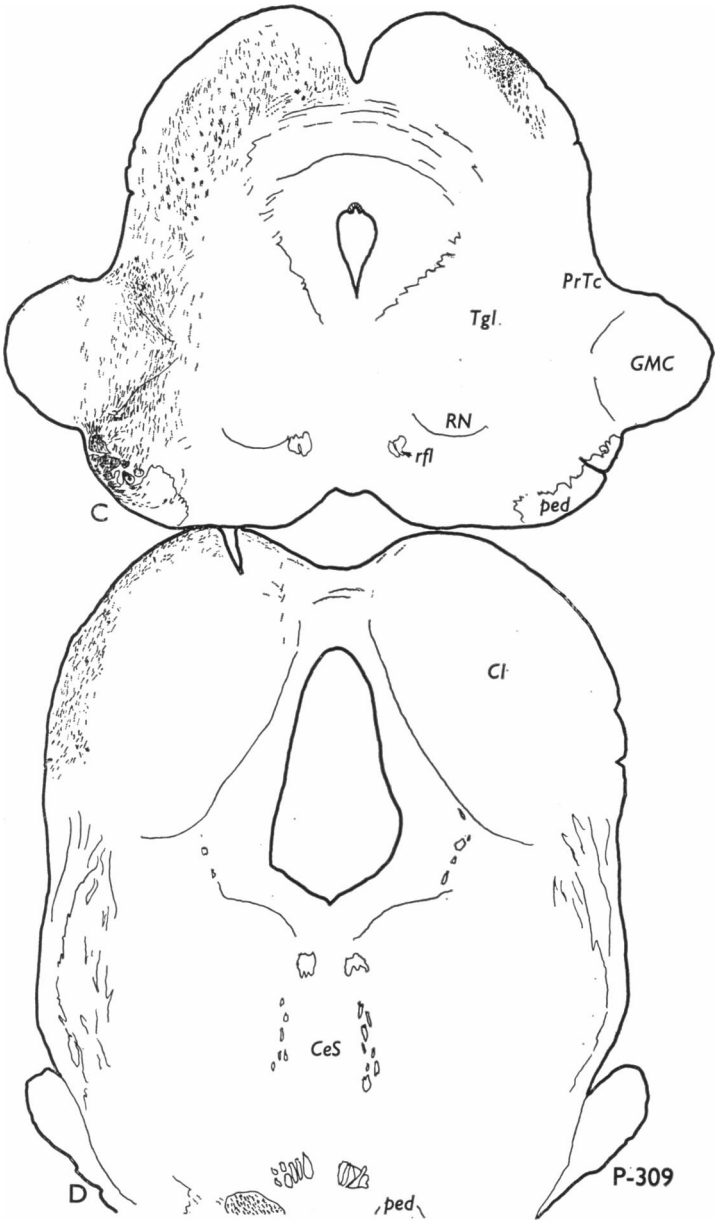


Fig. 16. Drawings through the mesencephalon (C) and mesencephalon–pons (D) from the brain shown in the insert of Fig. 17. Facial area degeneration is on the reader's left, whereas that resulting from the visual damage is on the right. Degenerating axons are illustrated as in Fig. 4 and various structures are labelled on the right.

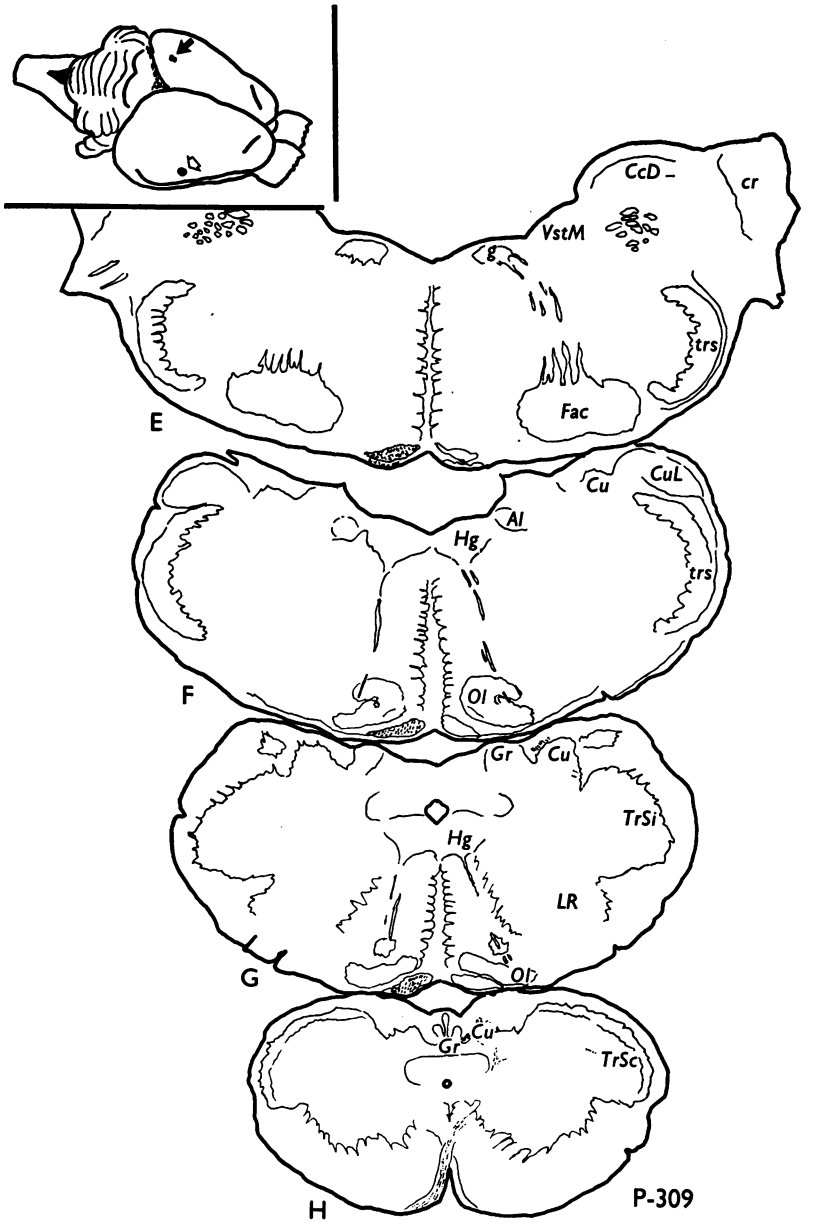


Fig. 17. Drawings through the caudal pons (E) and medulla (F–H) of the brain shown in the insert (upper left). The right side of the brain is on the reader's left. Degenerating axons are drawn as in Fig. 4 and various landmarks are labelled on the reader's right.

(1975). Thalamic degeneration was present in portions of the face area (left side, Fig. 15A, B) and in the midbrain degeneration was confined to the regions described previously (left side, Fig. 16). Although degenerating axons could be followed throughout the pyramidal tract and into the spinal cord, terminal degeneration was *not* present within trigeminal sensory nuclei (Fig. 17E–H). In still another brain, the damage was restricted to the ventral extreme of the face area. Axonal degeneration was present in the appropriate face area of the thalamus, and although minimal, was in the expected region of the midbrain. Degenerating fibres could be followed throughout the pyramidal tract (and into the cord) and some terminal debris was present in those areas of the brainstem which contained degeneration after larger face area ablations.

Autoradiographic results were available from two brains with ^{3}H leucine placements limited to face motor–sensory cortex (one shown in Fig. 26). In both cases silver grains were extensive over the expected areas of the thalamus; but, as in the forelimb cases, they were only sparsely distributed over the brainstem. However, grain counts above background were present in the ventrolateral superior colliculus, the nucleus intercollicularis, the basilar pons, the parvocellular reticular formation (particularly on the side opposite the placement) and in the contralateral sensory trigeminal nuclei. Grains were relatively abundant over the caudal spinal trigeminal nucleus (where some extended into the pars gelatinosa), although they were also noted in the hilum of the nucleus cuneatus. As in the forelimb cases, however, silver grains were located over the pyramidal tract (24 hours) and the grains in the cuneate hilum most likely reflected the presence of the labelled amino acid in recurrent fascicles.

Projections of opossum auditory cortex

Several brains with damage to auditory cortex (Lende, 1963*a*, see Figs. 2, 3 of present account) were present in our collection and the reader is referred to the right side of Figs. 10–12 for a plotting of the degeneration obtained in one specimen. In all such cases orthograde thalamic degeneration was present within the ventral and dorsal lateral 'geniculate' nuclei, the lateral posterior nucleus, the posterior nucleus (Oswaldo-Cruz & Rocha-Miranda, 1968) and within certain areas of the medial geniculate complex. 'Retrograde dust' was also present within the dorsal lateral geniculate and lateral posterior nuclei. Of course, the degeneration in the lateral geniculate nuclei suggested visual fibre undercutting. At superior collicular levels, all such brains contained anterograde degeneration in the lateral basis pedunculi (right side, Fig. 10A) as well as within fascicles coursing through the deep layers of the superior colliculus (right side, Fig. 10A, B). As might be expected from the visual undercutting, degenerating axons entered the medial stratum opticum and were distributed to that layer as well as to the adjacent stratum griseum superficialis and the stratum zonale (right side, Figs. 10B, 11C). Terminal degeneration was also present in both the parabigeminal area and the intercollicular nucleus at such levels. Degenerating axons within the brachium of the inferior colliculus, as well as many of those within the deeper layers of the superior colliculus (right side, Figs. 10B, 11C), could be followed to the level of the inferior colliculus (right side, Fig. 11D). Terminal debris was present within the external nucleus of the inferior colliculus, the

dorsomedial sector of its central nucleus and the pericentral nucleus (terminology used for the cat by Rockel & Jones, 1973*a*, *b*) and degenerating axons could be followed across the commissure of the inferior colliculus. A few impregnated axons could be traced into the pontine portion of the pyramidal tract from which they were distributed to ventral and dorsolateral areas of the basilar pons (see Yuen *et al.* 1974, for details of their pontine targets), but none could be traced more caudally (note pyramid, right side of Fig. 12). It is possible that much of the pontine degeneration (particularly ventrally) was the result of damage to fibres from the visual belt. Because of technical problems, and the relative inaccessibility of auditory cortex, autoradiographic evidence concerning its projections was limited to brains with ^{3}H leucine placements, which included both dorsal auditory and adjacent peristriate areas. In such cases silver grains were obvious in the expected areas of the thalamus (24 hours), but were barely above background level in the brainstem.

Projections of opossum visual cortex

In brains with damage to the visual cortex as defined by Lende (1963*a*) (compare Fig. 3 with insert, Fig. 17, present account) degenerating axons were present within the lateral geniculate and lateral posterior nuclei of the thalamus (right side, Fig. 15A, B), as well as within the superficial layers of the superior colliculus (right side, Fig. 16C). As would be expected, marked retrograde changes were present within the dorsal lateral geniculate nucleus, and the position of the orthograde degeneration within the ventral lateral geniculate nucleus and the superficial superior colliculus was highly dependent upon the location of the lesion. Although some brains showed degeneration within the basis pedunculi and pontine pyramidal tract (as well as the basilar pons, see Yuen *et al.* 1974), those with damage limited to the central striate area (left side of insert, Fig. 17) showed brainstem degeneration only within limited areas of the superior colliculus (right side of Fig. 16C). None showed axonal debris caudal to the basilar pons (right side, Fig. 17).

In four cases ^{3}H leucine was placed within the visual area. In each brain it was difficult to determine whether or not the injection was limited to either the striate or peristriate cortex because of the difficulty in drawing the appropriate boundary in the opossum and the tendency for the amino acid to spread. However, there was autoradiographic evidence for projections to the dorsal and ventral lateral geniculate nuclei, the lateral posterior thalamic nucleus and the superficial layers of the superior colliculus. Silver grains were also present over fibres in the optic tract and in fibre bundles within the posterior thalamic nucleus (1–3 day survival). In such brains the distribution of silver grains over the superior colliculus was consistent with the presence of axonal degeneration after visual cortical lesions.

Total corticobulbar projections in the opossum

Brains subjected to almost complete removal of neocortex on one side (e.g. insert, Fig. 18) provided information relative to the totality of corticobulbar projections. At midbrain levels, degenerating axons were present within all of the areas which showed degeneration after small lesions. At low power they were particularly obvious in a zone just beneath the surface of the superior colliculus (outer stratum zonale), in the stratum opticum and in bundles coursing within the deeper layers of the superior

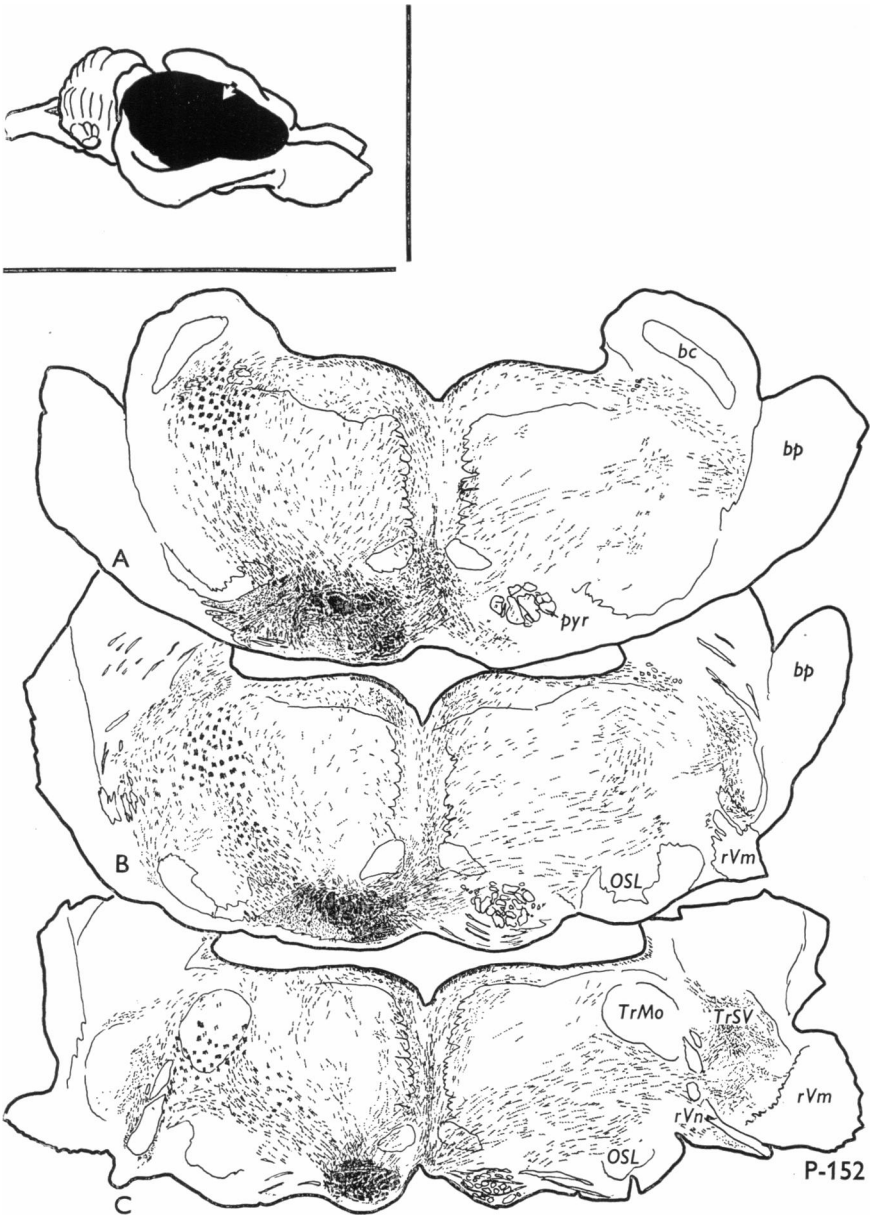


Fig. 18. Drawings of sections through the pons (A–C) of the brain shown in the insert (upper left). The lesion is essentially a decortication (arrow) and the degeneration is plotted as in Fig. 4. Relevant structures are labelled on the reader's right.

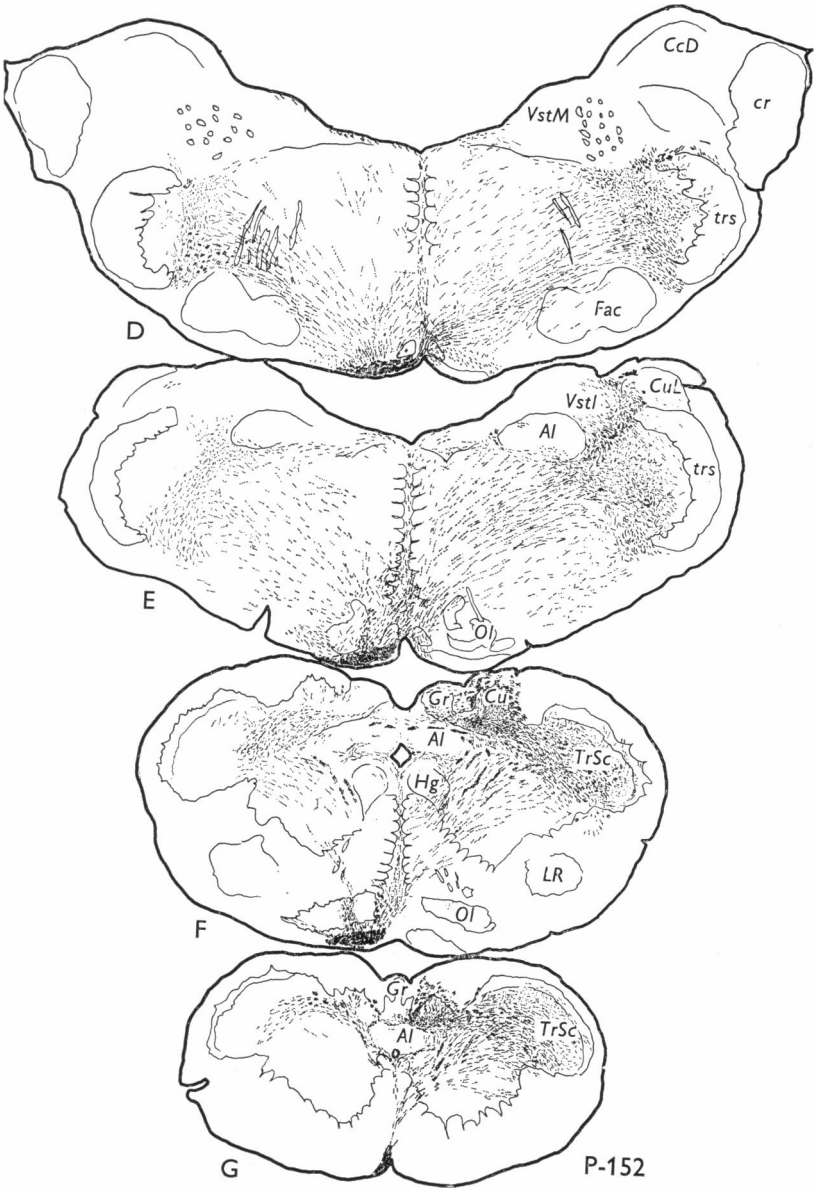


Fig. 19. Drawings of sections through the caudal pons (D) and medulla (E–G) of the brain shown in the insert of Fig. 18. Degenerating axons are illustrated as in Fig. 4 and certain nuclei and fibre tracts are labelled on the reader's right.

colliculus. Degenerating corticorubral fascicles were most numerous within the ventral part of the red nucleus rostrally (rostral two thirds) and in its lateral part caudally, and least numerous within its caudal, medial third (see King, Martin & Conner, 1972). As would be expected from the previous descriptions, degenerating axons were extremely dense and interlaced in the intercollicular area, and fragmented axons could be traced across the midline in the periaqueductal grey and the nucleus linearis, as well as within the commissures of the superior and inferior colliculi. In spite of the size of the lesion, few degenerating fibres were located within the area of the inferior colliculus which received input from the cochlear nuclei (unpublished results), although they were numerous in the other subdivisions.

More caudally, degenerating axons issued from the pyramidal tract and ended extensively in all areas showing degeneration after small lesions. Bundles of degenerating axons left the basis pedunculi at midbrain levels and descended into the pons on the side of their origin (left side, Fig. 18A–C), comprising an ipsilateral corticobulbar tract similar to that described for the goat by Bagley (see Haartsen & Verhaart, 1967). Although they were not distinguished readily in brains with small lesions, it could be concluded from our material that they arose within face motor–sensory cortex. Such fibres descended through and medial to the motor trigeminal nucleus and were joined by others which left the pyramidal tract at pontine levels. A large number of degenerating pyramidal fibres coursed dorsally within the midline (many ending within the reticulotegmental nucleus and the nucleus raphe magnus) and then turned laterally within the periventricular grey (Fig. 18A–C). Although the degenerating axons in the periventricular grey were difficult to trace to their termination, some of them appeared to gain access to the chief sensory trigeminal nucleus. Bilateral degeneration was present among the neurons intercalated between the emerging motor trigeminal rootlets (Fig. 18C) and in the nucleus of the pontobulbar body, although it was most extensive on the side opposite the lesion. Degenerating axons left the pontine pyramidal tract in large numbers, and coursed to both sides of the brainstem. It is obvious from Fig. 19, however, that in the caudal pons they were distributed most heavily to the contralateral side, particularly to the parvocellular reticular formation. At the same levels, degenerating recurrent bundles could be identified and they contributed to the degeneration just described (Fig. 19D). A few degenerating axons were present in ventral and lateral parts of the nucleus of the tractus solitarius (Fig. 19E).

In the medulla degenerating axons coursed within the midline and some eventually passed laterally beneath the central canal where they could be followed laterally through the nucleus intercalatus (Figs. 19F, 29). Degenerating axons terminated within the ipsilateral inferior olivary (parts of subnuclei b, c, Figs. 19F, 27, 28) and lateral reticular nuclei and bilaterally within the nucleus of the tractus solitarius. The degeneration within the lateral reticular nucleus was limited to its ventral external and parvocellular divisions. Terminal debris was extremely dense within the contralateral nucleus cuneatus (particularly its hilum and lateral sector, Figs. 19F, G, 30) and in the caudal division of the spinal trigeminal complex (Fig. 19F, G). Although degenerating axons could be traced into the base of the nucleus gracilis (Figs. 19G, 31), the bulk of the nucleus contained few, if any, degenerating axons which could be identified with certainty as cortical in origin. It

should be apparent from Fig. 19 that the major distribution of cortical fibres to the caudal trigeminal and dorsal column nuclei, as well as to the parvocellular reticular formation was contralateral.

Although degenerating axons coursed through the motor trigeminal (Fig. 18C) and facial (Fig. 19D) nuclei, no evidence of terminal debris was present. Degenerating fibres were also present within and around the hypoglossal nucleus (Fig. 19F), although their terminal character could be questioned. It should be noted, however, that available Golgi preparations revealed dendritic spread of cranial nerve motor neurons well beyond their nuclear 'confines', suggesting the possibility that cortical fibres make synaptic contact with distal dendrites even though they do not enter the nuclei proper.

In most hemi-decorticate cases a few degenerating axons were present within (as well as dorsal to) the pyramidal tract contralateral to the lesion, although the route they took across the midline was not clear. In sections caudal to the pyramidal decussation small bundles of degenerating axons re-crossed beneath the nucleus gracilis (through the commissural nucleus of the vagus and the commissure of Cajal, Figs. 19G, 31) and subsequently descended. In one specimen (P-358) these double crossed fibres could be observed in the first cervical segment where they passed to the dorsomedial sector of the caudal spinal trigeminal nucleus. Degeneration of these same fascicles was present in cases with large limb and face lesions.

Corticobulbar projections in the brush-tailed possum with notes on other marsupials

Material available in our laboratory from the brush-tailed possum of Tasmania suggested that corticobulbar fibres were distributed to all areas just described for the opossum (see also Martin *et al.* 1971; Martin & Megirian, 1972), although even casual inspection revealed that such fibres were more numerous in the brush-tailed possum. The projections of the cortex rostral to the orbital sulcus were similar to those arising from the comparable area in the opossum and will not be detailed here (see Martin *et al.* 1971; Martin & Megirian, 1972).

The distribution of thalamic degeneration after limb area lesions suggested that most of the fibres which were distributed to the somatosensory portion of the ventral thalamic complex (Rockel, Heath & Jones, 1972) arose somewhat caudally within the limb cortex, suggesting that overlap of somatic sensory and motor representation within the cortex was not complete (compare Abbie, 1940, with Adey & Kerr, 1954). Although the projections of the limb motor–sensory area were generally comparable to those described for the opossum, there was more evidence that the fibres to the nucleus intercollicularis and external nucleus of the inferior colliculus arose from caudal limb areas and there was a much greater distribution of fibres to the nucleus gracilis, including Bischoff's nucleus (Figs. 22B, C, 32, 33, 34). The latter observation is in accord with the relatively large hindlimb somatosensory representation in the brush-tailed possum cortex (compare Adey & Kerr, 1954, with Lende, 1963*a*). As in the opossum, little degeneration was present in either the parvocellular reticular formation or the adjacent trigeminal sensory nuclei (see F-112, Fig. 20) after lesions limited to the limb motor–sensory cortex (area medial to sulcus Zeta of Ziehen, 1897, Fig. 38, present study). In all such brains, however, degeneration was abundant in the dorsal column nuclei (F-112, F-41, Fig. 21). In several brains with particularly large,

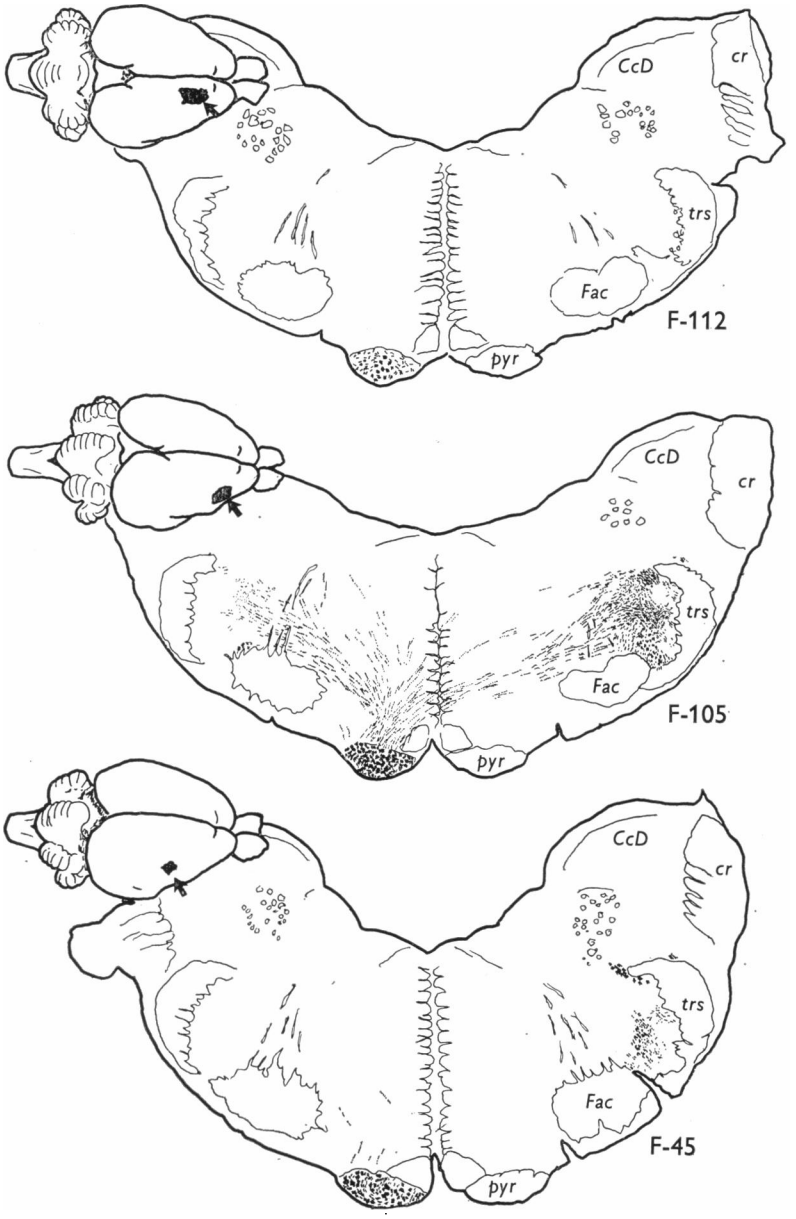


Fig. 20. Generally comparable sections through the pons of three separate brush-tailed possum brains are illustrated. The lesion in each is indicated by the arrow and the degeneration is illustrated as in Fig. 4. Comparable structures are labelled on the reader's right in each section.

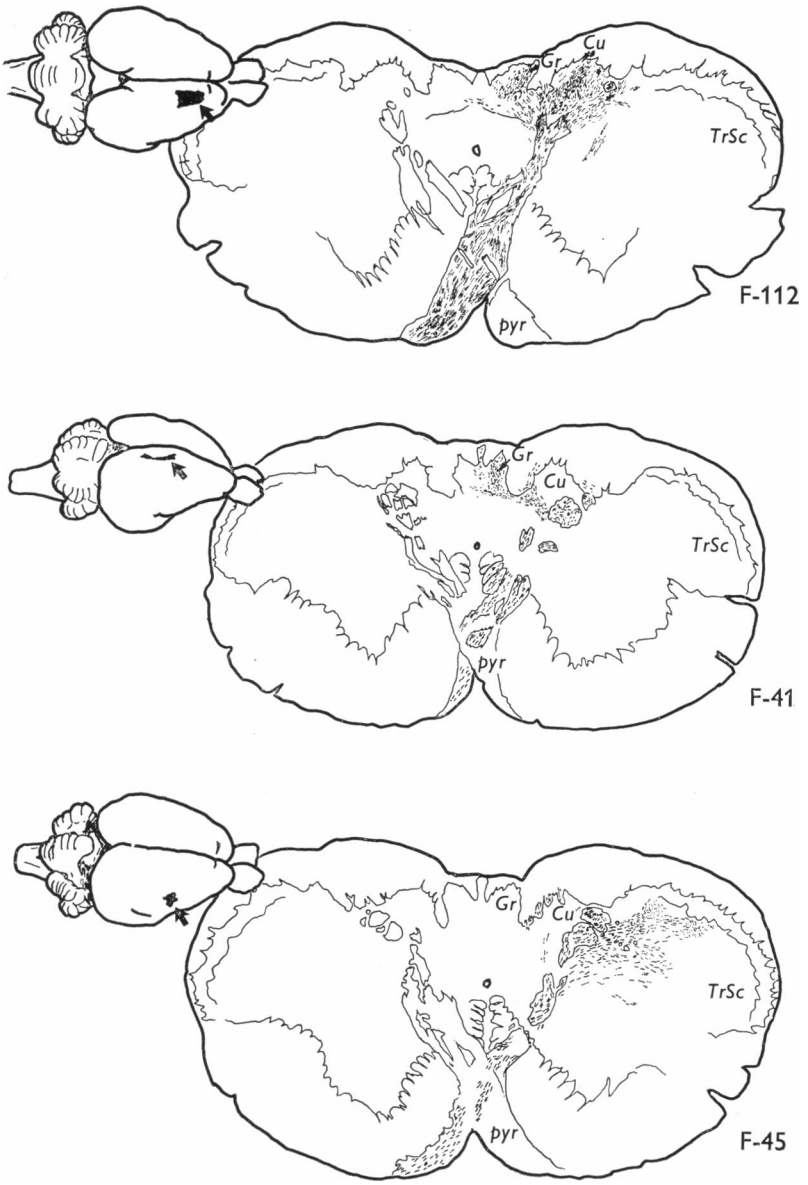


Fig. 21. Generally comparable sections through the caudal medulla are illustrated from three separate brush-tailed possum brains. The lesion is indicated by an arrow on the drawing of each brain and the axonal degeneration is illustrated as in Fig. 4. The same structures are labelled on the reader's right in each section.

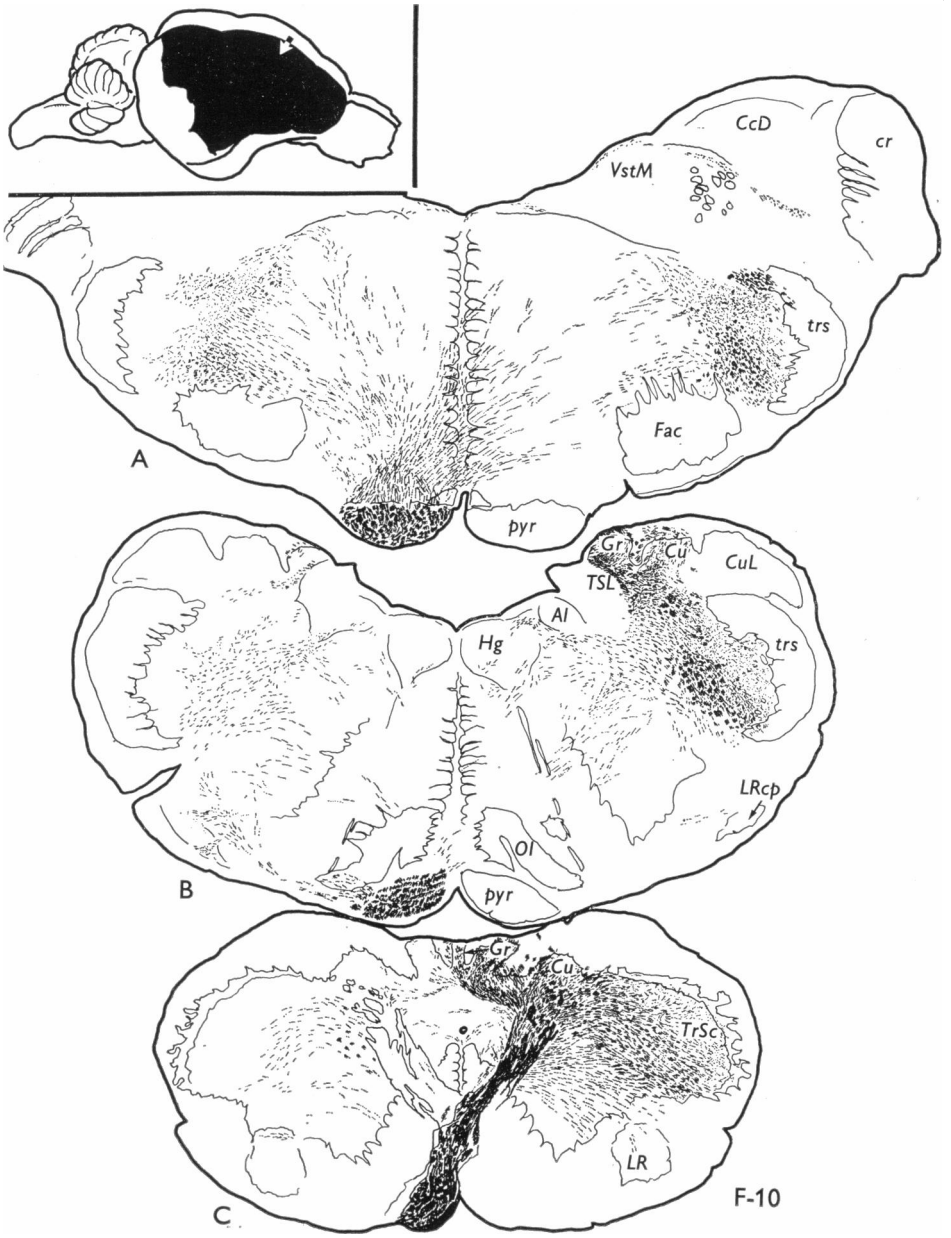
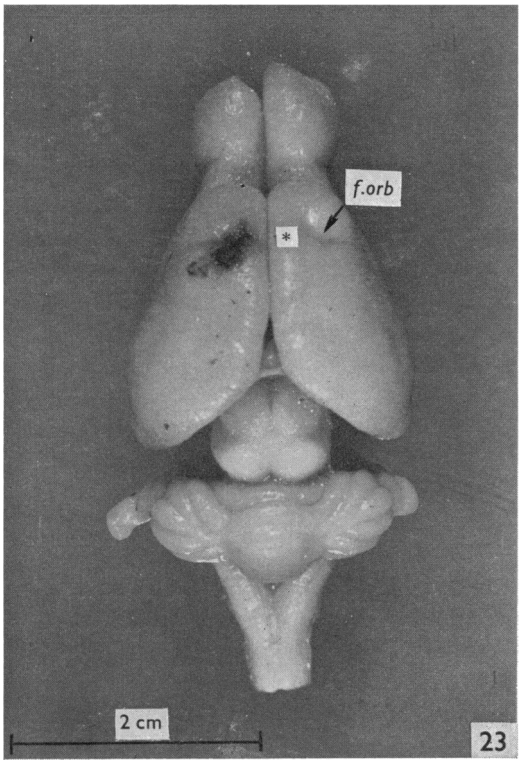


Fig. 22. Drawings of sections through the caudal pons (A) and medulla (B–C) of the brush-tailed possum brain drawn in the insert (upper left). Degenerating axons are drawn as in Fig. 4 and certain structures are labelled on the reader's right in each section.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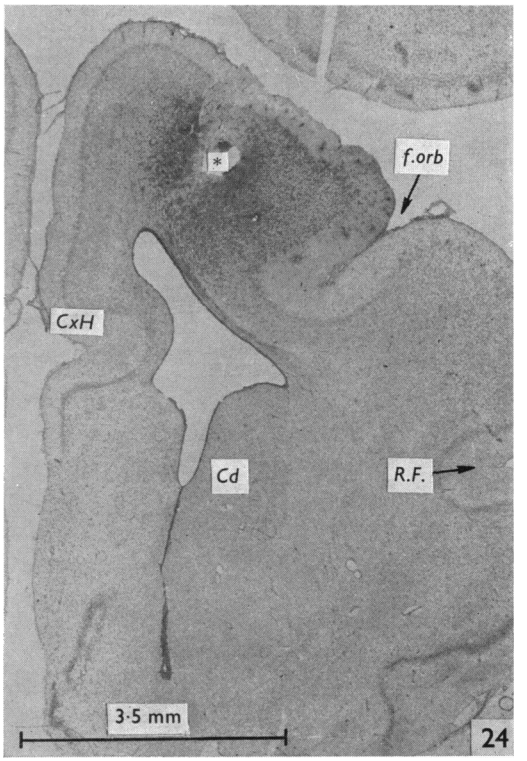
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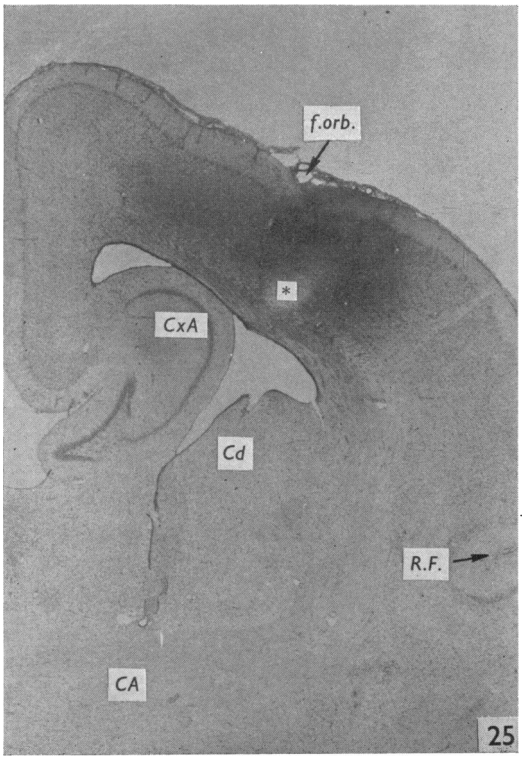
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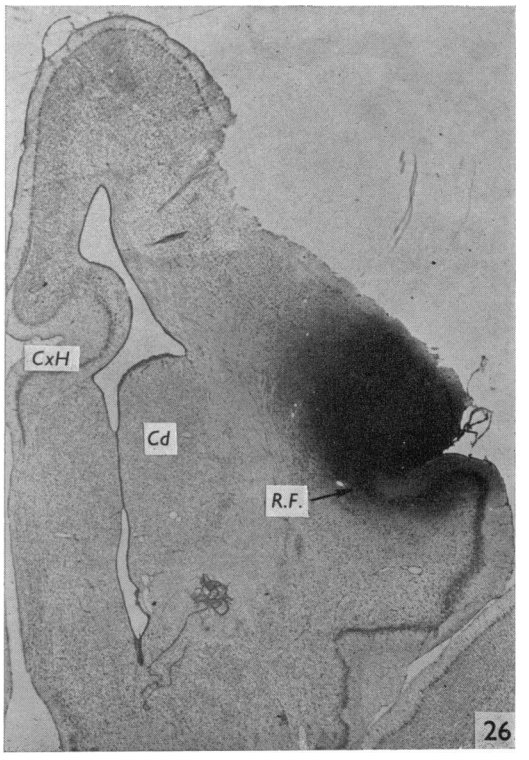
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caudally extending 'limb' lesions (e.g. Figs. 35, 36), degenerating axons were distributed to the medial reticular formation (nucleus gigantocellularis, paragigantocellularis, paramedian area, nucleus interfascicularis hypoglossi), the inferior olivary nucleus, the lateral reticular nucleus (ventral external arcuate nucleus and parvocellular part) and to the dorsomedial extreme of the spinal trigeminal nuclei (the latter by way of recurrent fascicles).

Haight & Weller (1973) state that the forelimb and hindlimb areas are often separated by a small dimple, referred to by them as the interbrachial sulcus (see also Abbie, 1940; Adey & Kerr, 1954). Comparison of brains with lesions generally limited to either forelimb (F-112) or hindlimb (F-41) motor–sensory areas suggested that the projections to the dorsal column nuclei were somatotopically organized (Fig. 21). It is possible that the degeneration in the lateral part of the nucleus gracilis in F-112 was a result of hindlimb fibre undercutting, since the lesion extended well into the white matter. Although some degeneration was present in the dorsal column nuclei of all brains with limb cortical lesions, it was relatively slight in those with lesions restricted to the rostral limb area, and extensive when more caudal cortex was involved. The degeneration present within the caudal brainstem was plotted for the brush-tailed possum in Fig. 22 after removal of all limb (and face) cortex on one side, serving as a comparison with that present after a comparable operation in the North American opossum (Fig. 19).

The face area of the brush-tailed possum brain was separated from the forelimb region by the jugular furrow (Haight & Weller, 1973), a sulcus comparable to that indicated by the Greek letter Psi by Ziehen (1897) (see Fig. 38, present study). As would be expected from the results obtained in the North American opossum, fibres from the face sensory area (Adey & Kerr, 1954) were distributed heavily to the parvocellular reticular formation and the trigeminal sensory complex, but not to the dorsal column nuclei (see F-105, F-45, Figs. 20, 21). However, in the brush-tailed possum, evidence was available that the fibres to the parvocellular reticular formation arose mainly within the rostral face cortices, whereas most of those to the trigeminal sensory nuclei took origin more caudally (compare F-105 with F-45, Fig. 20). The results obtained from additional brains with lesions of presumptive auditory and visual cortices were similar to those reported previously (Martin *et al.* 1971; Martin & Megirian, 1972) and, for brevity, the reader is referred to those earlier accounts.

Material available from the Tasmanian potoroo indicated that the targets of corticobulbar fibres were comparable to those in the species just described. As in the

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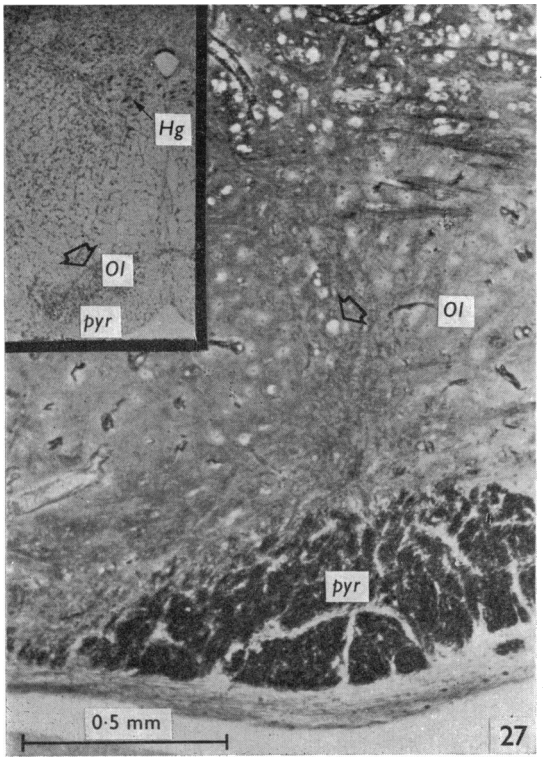
Fig. 23. Dorsal view of an opossum brain with a postorbital, parietal lesion on the left and the orbital 'fissure' labelled on the right (*f. orb*). The asterisk on the right shows the location of the ^{3}H leucine placement in the case illustrated in Fig. 24.

Fig. 24. Nissl stained frozen section through the location of the syringe needle (asterisk) in one of the opossum brains prepared by the autoradiographic technique (24 hour survival time, 2 week exposure). The placement is limited to forelimb cortex.

Fig. 25. Nissl stained frozen section through the location of the syringe needle (asterisk) in one of the opossum brains prepared for autoradiography. The leucine placement in this case overlapped both forelimb and face cortex (24 hour survival time, 4 week exposure).

Fig. 26. Nissl stained section through a ^{3}H leucine placement in an opossum brain processed by the autoradiographic technique The placement in this case is in the face motor–sensory cortex with some spill-over into the pyriform area (24 hour survival, 6 week exposure).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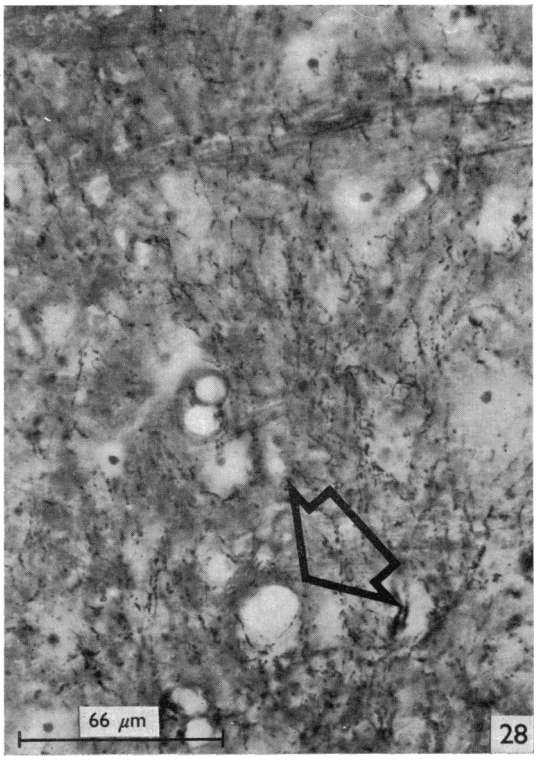
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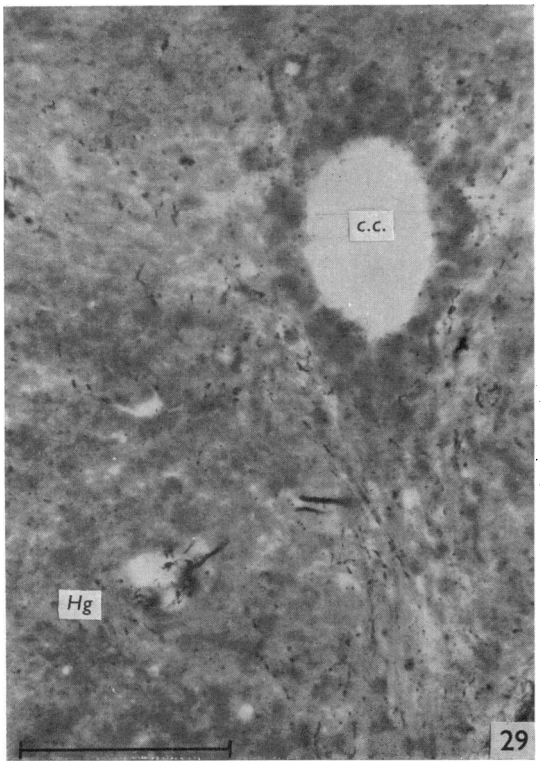
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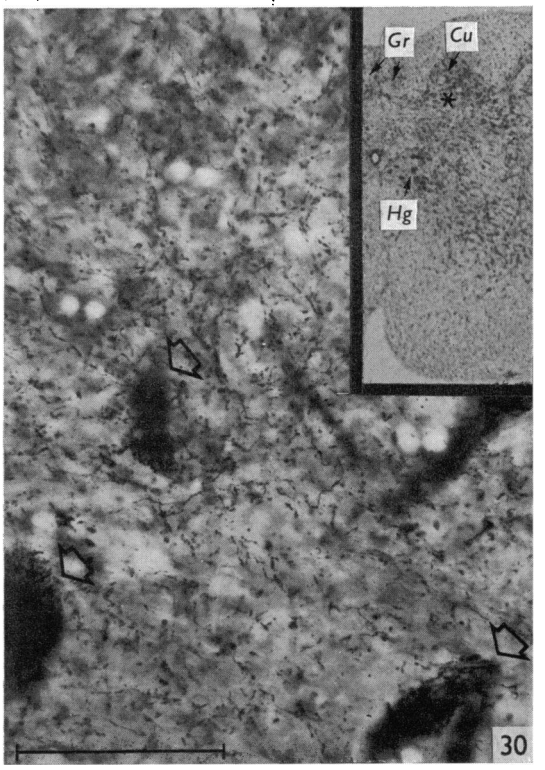
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brush-tailed possum (but not the North American opossum) there was a considerable distribution of cortical fibres to the nucleus gracilis. Unlike the brush-tailed possum, however, few if any such fibres could be traced into Bischoff's nucleus, even after removal of all the neocortex. Although it was apparent from our collection that the projections of cortex rostral to the orbital sulcus were similar to those described for the above species, and that the fibres to the dorsal column and sensory trigeminal nuclei could be fractionated by different lesions, the material must be treated conservatively because of its relatively limited nature and the absence of physiologically defined cortical maps.

The results obtained from the Tasmanian native cat brains in our collection showed that corticobulbar fibres enjoyed the same general distribution as that described for the other species and that, in general, they were organized in a comparable fashion. Evidence was present that the fibres to the parvocellular reticular formation and trigeminal sensory nuclei came from spatially co-extensive areas (similar to the situation in the opossum). However, in most brains, considerable 'extraneous' degeneration was present, which by its character and distribution was obviously not experimentally induced by cortical damage. This degeneration was either a reflexion of the age of the animals available or a predilection to some central neuropathy. Whatever its etiology, such extraneous degeneration made it difficult to differentiate with certainty between degeneration caused by the cortical damage and that resulting from some unknown pathology. For this reason, details in this species must await further study, either by degeneration methods in 'clean' animals, or by autoradiographic methods.

DISCUSSION

The present study reports evidence for corticobulbar connexions not previously described in the opossum. These include projections of the limb and face motor–sensory cortex to the external nucleus of the inferior colliculus, fibres from the face motor–sensory area to the nucleus of the pontobulbar body (including the neurons

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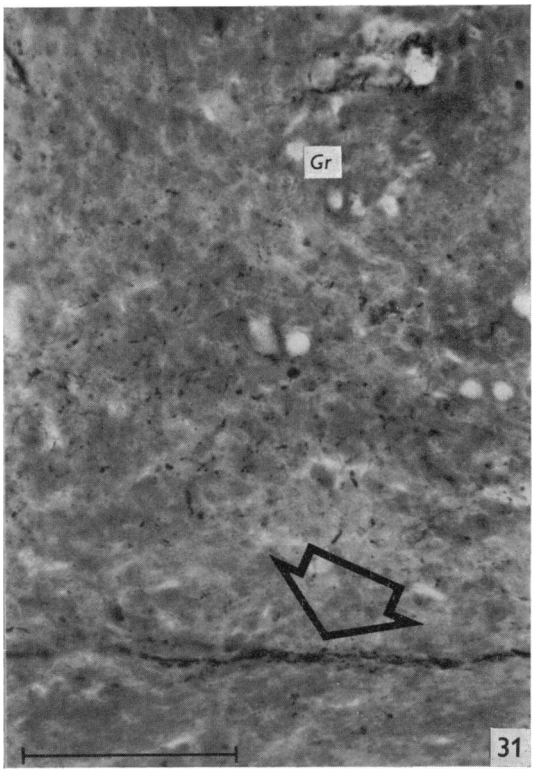
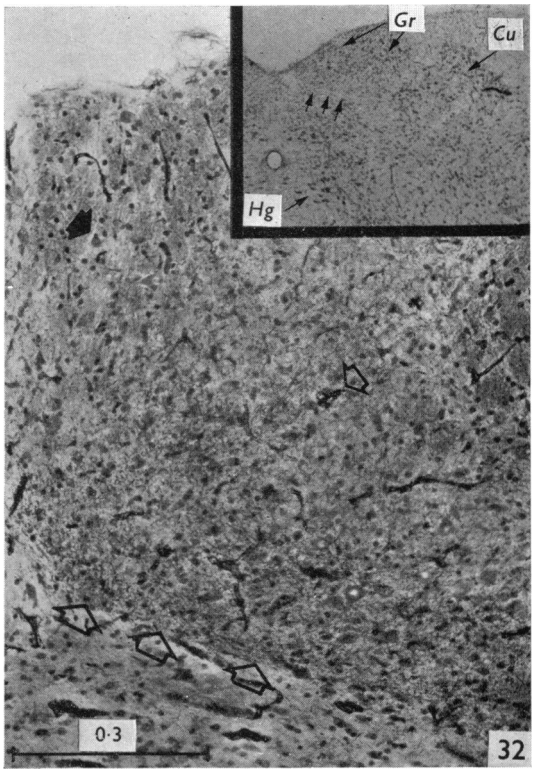
Fig. 27. Photomicrograph of the degenerating pyramidal tract (*pyr*) in the brain of an opossum subjected to decortication on the same side (Fink-Heimer technique). The arrow indicates a blood vessel within the inferior olivary nucleus (*OI*) which is similarly demarcated at higher power in Fig. 28. The insert is a photomicrograph of a Nissl stained section taken at the same level. The arrow in the insert points to the area of the inferior olivary nucleus containing cortical fibres.

Fig. 28. High power photomicrograph of axonal degeneration in the inferior olivary nucleus of the opossum subsequent to unilateral decortication. The arrow points to the blood vessel similarly indicated in Fig. 27.

Fig. 29. High power photomicrograph of the thin degenerating axons which curve laterally beneath the central canal (*c.c.*) and dorsal to the hypoglossal nucleus (*Hg*). Such fibres are readily observed at the level shown in the insert of Fig. 30 in opossum brains subjected to decortication. Fink–Heimer technique.

Fig. 30. Degenerating axons in the hilum of the nucleus cuneatus (area of the asterisk in the insert) from a brain previously decorticated. The arrows indicate bundles of pyramidal fibres on their way to either the fasciculus cuneatus (2 arrows on the left) or the recurrent bundles (lower right). Fink–Heimer technique. The insert is from an adjacent Nissl stained section which shows the nucleus gracilis (*Gr*), the nucleus cuneatus (*Cu*) and the hypoglossal nucleus (*Hg*) for reference.


Gr
31


Gr
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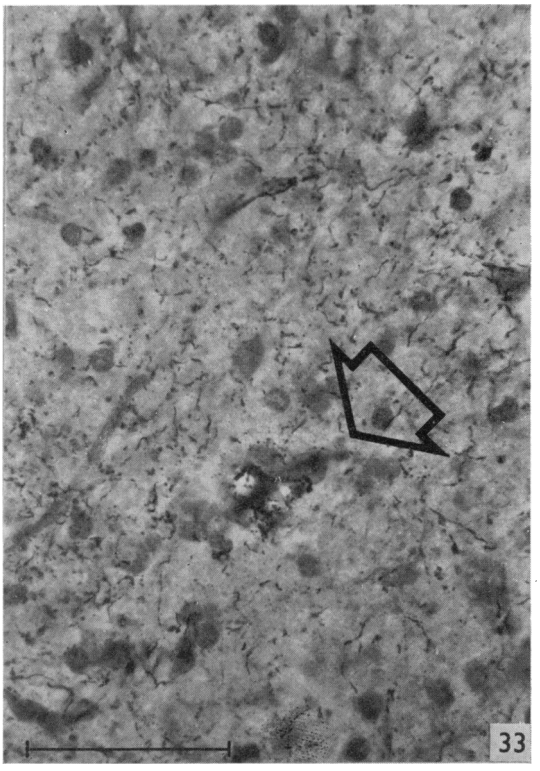
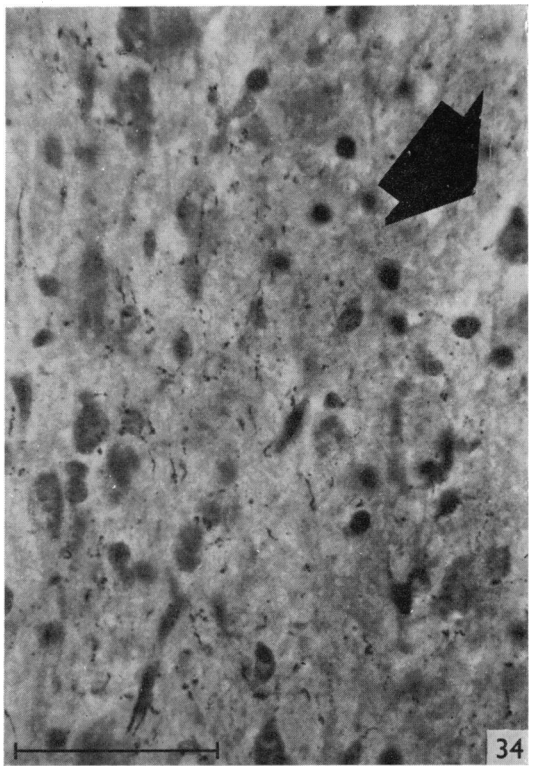
32


33


34

around the emerging motor trigeminal fascicles, see Oswaldo-Cruz & Rocha-Miranda, 1968) and motor–sensory cortical relays to the ipsilateral inferior olivary and lateral reticular nuclei. In addition, attention has been directed to the projections of limb and face motor–sensory areas to a region referred to as the nucleus intercollicularis by Olszewski & Baxter (1954).

Projections from the limb and face motor–sensory cortices appear to 'converge' on the intercollicular nucleus of the midbrain (part of the lateral tegmental area of our previous report, Martin, 1968) as well as on the external nucleus of the inferior colliculus. Previous studies from our laboratory (Hazlett *et al.* 1972) as well as from others (Mehler, 1969; Jane & Schroeder, 1971; Schroeder & Jane, 1971; RoBards, Watkins & Masterton, 1974) have shown that spinal and dorsal column fibres terminate within these same regions. On the basis of its decided somatosensory overlap the nucleus intercollicularis has been considered to be a somatosensory tectum (RoBards *et al.* 1974). Degenerating axon terminals are present in these same areas after lesions of the auditory cortex (as delineated by Lende, 1963*a*). However, a conclusion of auditory, somesthetic interaction at that level is premature, since Lende's mapping revealed a secondary somesthetic area which overlaps the auditory representation, and the entire region needs to be re-studied by microelectrode techniques.

Although the opossum nucleus of the pontobulbar body (Oswaldo-Cruz & Rocha-Miranda, 1968) may be comparable to the primate processus tegmentosus lateralis (see Mehler, 1969), such an homology is not certain. Whatever the case, in the opossum this group of neurons receives fibres from the red nucleus (Martin & Dom, 1970*a*, *b*; Martin *et al.* 1974), the spinal cord (Mehler, 1969 and unpublished results from our collection), the fastigial nucleus (Martin, 1973) and the cerebral cortex (present account). Although degeneration within the nucleus of the pontobulbar body was seen first in decorticate brains, evidence for its existence was also present in brains with lesions of face motor–sensory cortex. Further study of the nucleus of the pontobulbar body is needed to assess the potential overlap of its multiple inputs and its efferent connexions. It is our belief that it relays to the cerebellum, but this is by no means certain.

In a previous description of opossum corticobulbar fibres (as demonstrated by the Nauta–Gygax method, Martin & West, 1967) we had no evidence for projections to

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Fig. 31. Photomicrograph of the degenerating pyramidal fibres 'recrossing' (arrow) beneath the nucleus gracilis (*Gr*) at the level shown in the insert of Fig. 30 (opossum brain). This is from a decorticate case so that some axonal debris is present in the hilum of the nucleus gracilis above the arrow. Fink–Heimer technique.

Fig. 32. Photomicrograph of the nucleus gracilis in a decorticate brush-tailed possum brain processed by the Nauta–Gygax method. The dark arrow points to a neuron similarly indicated in Fig. 34, whereas the upper right arrow demarcates a blood vessel also referred to in Fig. 33 from the same section. The arrows on the bottom of the figure outline the border indicated in like manner by the small black arrows in the insert. The insert shows a Nissl stained section at the same level in which the nucleus gracilis (*Gr*), cuneatus (*Cu*) and hypoglossi (*Hg*) are shown for reference.

Fig. 33. High power view of the axonal degeneration in that portion of the nucleus gracilis indicated by the upper right hand arrow in Fig. 32. Nauta–Gygax technique.

Fig. 34. High power photomicrograph of the degeneration in that portion of the nucleus gracilis indicated by the arrow in the upper left of Fig. 32. Nauta–Gygax technique.

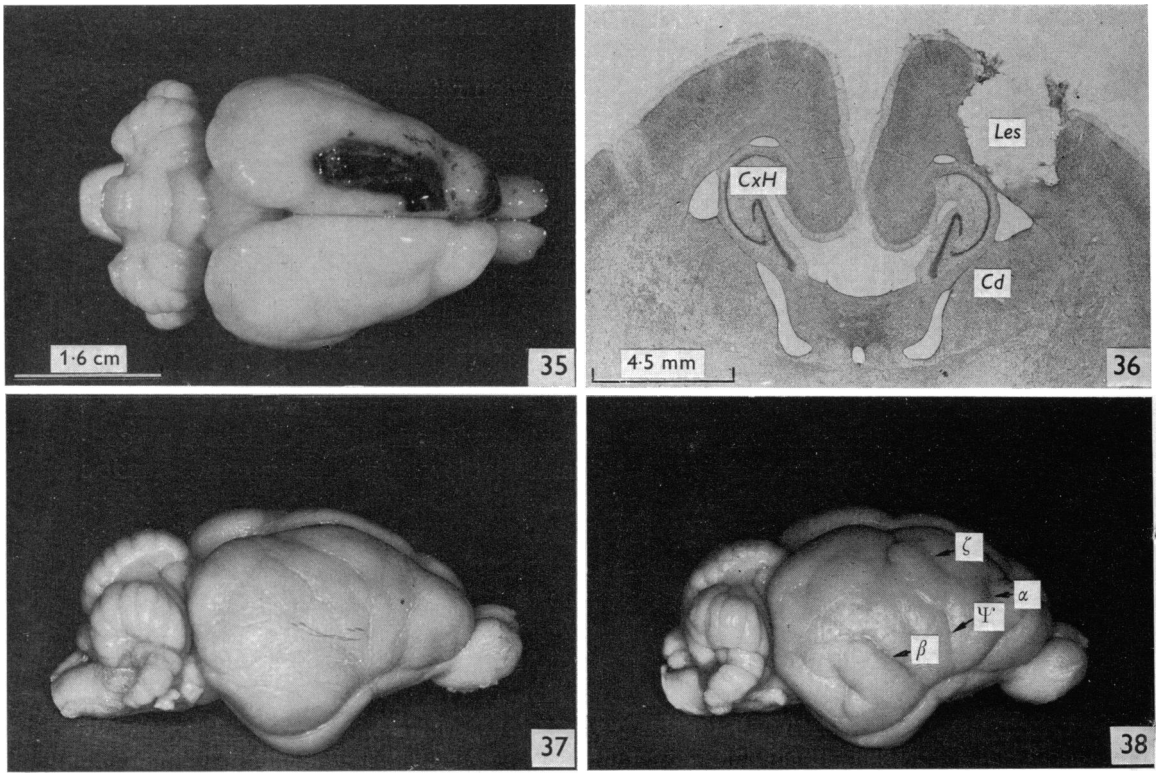

Fig. 35. Photograph of a brush-tailed possum brain (F-115) with a large forelimb motor–sensory cortical lesion. Since the damage extended into the white matter, hindlimb fibres were also interrupted.

Fig. 36. Photomicrograph of a Nissl stained section through the deepest extent of the lesion (*LES*) in F-115.

Fig. 37. Lateral view of a brush-tailed possum brain showing relatively little sulcal formation.

Fig. 38. Lateral view of another brush-tailed possum brain showing the most obvious sulcal pattern present in our collection. The arrows point to the sulci referred to by the indicated Greek letters (after Ziehen, 1897).

either the inferior olivary or lateral reticular nuclei. Since that time such fibres have been observed to degenerate after somatic motor–sensory cortical lesions in the brush-tailed possum (Martin *et al.* 1971) and have been similarly revealed in the opossum by the Fink–Heimer method. Although in our material their laterality and distribution differed from that described for the cat (the inferior olivary nucleus, Sousa-Pinto & Brodal, 1969; the lateral reticular nucleus, Brodal, Marsala & Brodal, 1967), it is premature to insist that there are true species differences.

In the present study attempts were made to limit lesions to functionally distinct areas as defined by physiological methods (Lende, 1963*a*, *b*; Pubols *et al.* 1975). However, as reported by others (Pubols, 1968), it is difficult, if not impossible, to avoid white matter undercutting, perhaps because of the opossum's unique

vascularity (Wislocki & Campbell, 1937). For that reason we examined all sections through the lesion site and plotted the degeneration within the thalamus as well as the brainstem as an additional control for the extent of cortical damage. Since maps of the opossum thalamus are available (Erickson *et al.* 1964; Pubols & Pubols, 1966; Hazlett *et al.* 1972; Walsh & Ebner, 1973) this new information provides insight into the origin of both the new connexions described herein and those seen (at least in part) in previous studies (Martin & West, 1967; Martin, 1968). Because of unavoidable fibre undercutting in some brains (see Yuen *et al.* 1974, for a discussion of the problem), the autoradiographic material was also helpful for establishing the origin of certain pathways, as well as for providing a control for the presence of axons which might not have been impregnated at the survival times chosen in the degeneration experiments.

From our study, and from that of Pubols (1968), it appears that the preorbital area (Gray, 1924; Walsh & Ebner, 1970; see Fig. 3, present study) is reciprocally connected with the dorsomedial nucleus of the thalamus. This conclusion is supported by the presence of axonal degeneration in the preorbital cortex after lesions of the dorsomedial thalamic nucleus (Tobias & Ebner, 1973). It should be noted, however, that damage to the dorsomedial thalamus does not elicit degeneration in the frontal pole of the opossum cortex (frontal area of Gray, 1924), suggesting that the preorbital area may be its sole 'prefrontal' homologue (compare Tobias & Ebner, 1973, with Leonard, 1969). In any case, the location of degeneration within the thalamus and brainstem after preorbital lesions is generally comparable to that reported after destruction of both the dorsomedial and ventrolateral 'prefrontal' sectors in the rat (see Leonard, 1969, for details). Although our material suggests that the projections of opossum 'frontal' cortices (Gray, 1924) may also be similar to those arising within certain 'frontal' areas in placental brains (Leonard, 1969), its connexions with the dorsal thalamus have not been fully elucidated and in most of our cases there was some involvement of the preorbital area. It appears from the present study (as well as Martin, 1968) that the cortex rostral to the orbital sulcus in the opossum is the main source of corticobulbar projections to the ventral tegmental area, the nucleus linearis, the dorsomedial extreme of the medial pontine nucleus (see also Martin & King, 1968; Yuen *et al.* 1974), the superior central nucleus and the rostroventral part of the reticulotegmental nucleus.

Although the opossum limb somatosensory area overlaps the comparable motor representation almost completely (Lende, 1963*a*, *b*), microelectrode techniques reveal that there is a small area of motor cortex just caudal to the orbital sulcus (postorbital area, Walsh & Ebner, 1970) which does not respond to cutaneous stimuli (Pubols *et al.* 1975, see Fig. 3, present study). The functional overlap in the parietal cortex (as that area is defined by Gray, 1924; Walsh & Ebner, 1970) is reflected in the fact that it receives input from both the cerebellar and somatosensory targets of the ventral thalamic nucleus (Killackey & Ebner, 1973) and projects to the red nucleus and midbrain tegmentum (Martin, 1968; King *et al.* 1972), as well as to the nucleus cuneatus (Martin & West, 1967, present study) and the dorsomedial grey of the cervical cord (laminae III through VI; Martin & Fisher, 1968). The present study provides evidence for an additional *small* projection to the 'hilum' of the nucleus gracilis (decorticate brains processed by the Fink–Heimer method and

autoradiography) and clearly reveals that the cortical input to the pontine and medullary raphe, the parvocellular reticular formation and the trigeminal sensory nuclei does not originate in the forelimb area as defined by Pubols *et al.* (1975). The presence of degeneration in such areas in the 'forelimb' cases reported previously (Martin & West, 1967) was a result of face area contamination. In that study we used the map of Lende (1963*a*, *b*) as the guide for making our lesions and, as has been shown by more precise methods (Pubols *et al.* 1975), the forelimb representation is considerably smaller than previously recognized (Fig. 3, present study). Brains with lesions restricted to limb motor–sensory cortex (according to the map of Pubols *et al.* 1975, and verified by a lack of degeneration within the face area of the thalamus) exhibit brainstem degeneration within the superior colliculus, the red nucleus, the midbrain tegmentum (including the intercollicular nucleus), the external nucleus of the inferior colliculus, the basilar pons, the paramedian reticular formation, the inferior olivary and lateral reticular nuclei, the dorsal column nuclei (mainly non-primary dorsal column receiving areas as in the cat; Kuypers & Tuerk, 1964) and the cervical spinal cord (Martin & Fisher, 1968). Review of the literature indicates that the projections from the highly 'amalgamated' motor–sensory limb cortex of the opossum are remarkably comparable to the combined projections of the spatially separate somatic motor (*MsI*) and sensory (*SmI*) limb cortices in the rhesus monkey and chimpanzee (Kuypers, 1958, 1960).

In contrast to primates, however, the opossum does not have direct cortical projections to the lumbosacral cord (Bautista & Matzke, 1965; Martin & Fisher, 1968), and its nucleus gracilis (Hamilton & Johnson, 1973) as well as the hindlimb areas of its somatosensory thalamus (Pubols & Pubols, 1966; Sousa *et al.* 1971; Hazlett *et al.* 1972) and cortex (Pubols *et al.* 1975) are exceedingly small. In such a brain it is not surprising that the projections of the hindlimb cortex to both the nucleus gracilis and the hindlimb thalamus are meagre. Although a somatotopic organization of corticorubral projections *may* exist in the opossum, it is not nearly as apparent as that reported for the cat and monkey (see King *et al.* 1972, and Martin *et al.* 1974, for discussion). As indicated previously (Martin & Dom, 1970*a*), the absence of direct cortical projections caudal to rostral thoracic cord segments means that cortical orchestration of hindlimb function must involve relays within the brainstem (red nucleus, paramedian reticular formation) and/or spinal cord.

The opossum motor–sensory face cortex gives rise to projections to the pontine and medullary raphe, the perihypoglossal region, the nucleus of the tractus solitarius, the spinal trigeminal nuclei (with the possible exception of a small dorsomedial part of the subnucleus caudalis) and the parvocellular reticular formation. No terminal degeneration is seen in the dorsal column nuclei when the lesion is restricted to the face area (Lende, 1963*a*, *b*; Pubols *et al.* 1975); this is supported by the lack of terminal debris in the limb somatosensory thalamus (present study). Although silver grains are present over the hilum of the dorsal column nuclei after placements limited to the face area, it is likely that they reflect the presence of labelled amino acid in recurrent fascicles rather than in terminals.

Review of the literature reveals that the projections of the opossum face cortex (motor–sensory amalgam, Lende, 1963*a*, *b* – at least in part) are markedly similar to the combined projections of the spatially separate *MsI* and *SmI* face cortices in primates

(Kuypers, 1958, 1960). In primates, however, fibres to the parvocellular reticular formation arise mainly from the motor face area (*MsI*), whereas most of those to the sensory trigeminal nuclei take origin more caudally from the face sensory cortex (*SmI*). Although no similar fractionation of these connexions was observed in the opossum (perhaps only reflecting an extremely small area which is exclusively 'motor' in the opossum), suggestion for such an organization was obtained in the brush-tailed possum (see below). It should also be noted that our material provides evidence for some organization to corticosensory trigeminal fibres, suggesting, for example, that fibres to dorsal regions of the subnucleus caudalis arise from mandibular areas of the cortex. The reader is referred to Matano *et al.* (1972) and Wold & Brodal (1973) for recent descriptions of corticosensory trigeminal connexions in the rat and cat.

In general the projections of opossum auditory cortex are similar to those described previously (Martin, 1968). In the present communication, however, the terminology for the inferior collicular subgroups conforms to that used for the cat (Rockel & Jones, 1973*a*, *b*) and degeneration within the nucelus intercollicularis is reported after auditory area destruction. In the light of the potential overlap of auditory and somatosensory inputs within the intercollicular nucleus and certain areas of the inferior colliculus, it is interesting to note that Lende (1963*a*) reported evidence for auditory and *SmI* overlap at the cortical level, as well as the presence of an *SmII* area well within the auditory region (Fig. 3, present study). However, further study of this area using single cell and unit recording is imperative if definitive conclusions are to be reached.

Not surprisingly, both degeneration and autoradiographic experiments provide evidence for highly ordered projections of visual cortex to the superficial layers of the superior colliculus (see also Martin, 1968) as well as to the lateral geniculate and lateral posterior nuclei of the dorsal thalamus. Although visual cortex projects to the basilar pons in the opossum (Martin & King, 1968; Yuen *et al.* 1974), it is apparent from the present study that there is a small cortical area in the centre of the visual representation that neither projects to the basilar pons nor gives rise to fibres within the cerebral peduncle.

The results described herein verify the corticobulbar connexions reported previously for the brush-tailed possum (Martin *et al.* 1971; Martin & Megirian, 1972), but provide more detail concerning their origin. First, it appears that the greatest number of fibres to the dorsal column nuclei arise from parietal portions of the limb representation and that, in part at least, they are somatotopically organized. Secondly, the limb motor–sensory cortex in the brush-tailed possum projects few fibres to the parvocellular reticular formation, the pontine and medullary raphe or the spinal trigeminal nuclei. Material accumulated since our earlier report clearly reveals that the degeneration present in the parvocellular reticular formation after lesions thought to be limited to limb cortex (see Fig. 3, Martin *et al.* 1971) was a result of face area undercutting. Thirdly, the present results reveal that fibres to the parvocellular reticular formation arise mainly in rostral (motor?) face cortices. Caudal face regions project mainly to trigeminal relay nuclei at pontine and medullary levels. Such results are remarkably consistent with those reported in primate studies (see Kuypers, 1958, 1960) in which it is stated that cortical projections to the

'lateral tegmentum' (and cranial nerve motor nuclei) arise within motor face areas, whereas those to the trigeminal sensory nuclei arise mainly within the face sensory region. Although in marsupials few cortical fibres show evidence of termination within the *confines* of cranial nerve motor nuclei, the rostral (motor?) face cortex discharges strongly into the parvocellular reticular formation (lateral tegmentum), providing at least indirect cortical control. This would appear to be particularly true of the facial nucleus (see Dom *et al.* 1973).

There is a tendency for mammalian neurologists to consider the marsupials a singular group (often thought to be 'represented' by the North American opossum), remotely different from the better known and more commonly studied placental species. Although the marsupial radiation does not show the extreme variation seen in placental mammals, it includes a large number of strikingly different species which show considerable central nervous system diversity (e.g. the difference in the length of the corticospinal tracts – the American opossum, Bautista & Matzke, 1965; Martin & Fisher, 1968; the brush-tailed possum, Martin, Megirian & Roebuck, 1970; Rees & Hore, 1970; the quokka wallaby, Watson, 1971; the kangaroo, Watson, 1972; the Tasmanian potoroo, Martin, Megirian & Conner, 1972). Experimental data reveals that, in spite of such variability, their often 'strange' appearance, and certain of their well known 'non-placental' features (e.g. lack of a corpus callosum, extensive overlap of cortical somatosensory and motor representation and limited corticospinal tracts), marsupial central nervous system connectivity is markedly similar to that of placental mammals (somatosensory connexions, Mehler, 1969; Hazlett *et al.* 1972; Watson & Symons, 1972; brainstem efferent pathways, Martin, 1969; Martin & Dom, 1970*a*, *b*; Beran & Martin, 1971; Warner & Watson, 1972; and cortical efferent projections as summarized in the present study and seen by Watson in the wallaby and kangaroo, personal communication). In fact, certain connexions either have been reported for the first time in a marsupial (e.g. non-dorsal column spinal afferents to dorsal column nuclei, Hazlett *et al.* 1972; rubral projections to certain brainstem areas, Martin & Dom, 1970*b*) and subsequently verified in the cat (non-primary afferents to dorsal column nuclei, Rustioni, 1973; rubrobulbar projections, Edwards, 1972) or reported for a marsupial and the cat at about the same time (cerebello-pontine and cerebello-olivary connexions; the opossum, Martin, 1973; Dom *et al.* 1973; Yuen *et al.* 1974; the cat, Brodal, Destombes, Lacerda & Angaut, 1972; Graybiel, Nauta, Lasek & Nauta, 1973). Although there are differences between and within both the marsupial and placental radiations, the theme is apparent and increasingly predictable.

SUMMARY

Corticobulbar projections have been studied in the American opossum by both degeneration and autoradiographic methods and, for the most part, the results confirm our earlier observations (Martin & West, 1967; Martin, 1968). However, we have obtained evidence for certain connexions not previously described and have delineated the origin(s) of several connexions more precisely by paying particular attention to the degeneration present at thalamic levels in all cases and by the use of autoradiography. When our results are collated and correlated with new somato-

sensory cortical maps arrived at by microelectrode techniques (Pubols *et al.* 1975), it is obvious that corticolbulbar connexions in the North American opossum are remarkably similar to those in the monkey and differ mainly in quantity, relative origins and distribution and in the fact that some of them arise from spatially co-extensive motor–sensory areas (Lende, 1963*a*, *b*).

In the light of our findings on the American opossum we have examined a large collection of brush-tailed possum material (as well as some from the potoroo and Tasmanian native cat) and have been able to extend our previous findings (Martin *et al.* 1971; Martin & Megirian, 1972) to a more precise evaluation of the origin of projections from the limb, face motor–sensory cortex. Differences between these representatatives of the marsupial radiation, as well as features which are common to all, are described.

This investigation was supported by U.S.P.H.S. Grants NS-07410 to Dr George F. Martin and NS-07195 to Dr David Megirian.

The authors wish to thank Mrs Mary Ann Jarrell and Ms Kit James for technical help and Ms Malinda Amspaugh for typing the manuscript. In addition, we are grateful to Mr Gabriel Palkuti for photographic assistance and to Drs John Haight, Benjamin Pubols and Lee Weller for reviewing the manuscript.

The cooperation and encouragement of the National Parks and Wildlife Service of the State of Tasmania is gratefully acknowledged.

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ABBREVIATIONS

AI	dorsal vagal nucleus
bc	brachium conjunctivum
bci	brachium of the inferior colliculus
bp	brachium pontis
CA	anterior commissure
c.c.	central canal
CcD	dorsal cochlear nucleus
CeS	superior central nucleus
CcV	ventral cochlear nucleus
Cd	caudate nucleus
CF	fields of Forel
ci	internal capsule
CI	nuclei of inferior colliculus
cr	restiform body
Cu	nucleus cuneatus
CuL	lateral cuneate nucleus
CxH	anterior hippocampus
dbc	decussation of brachium conjunctivum
Fac	motor facial nucleus
f.orb.	orbital sulcus (fissure)
Fx	fornix
g	genu of facial nerve
GLD	dorsal lateral geniculate nucleus
GLV	ventral lateral geniculate nucleus
GM	medial geniculate nucleus
GMC	central medial geniculate nucleus
Gr	nucleus gracilis
Hg	hypoglossal nucleus
HL	lateral habenular nucleus
HyL	lateral hypothalamic area
IP	interpeduncular nucleus
LES	lesion
LP	lateral posterior nucleus of thalamus
Lr	linear nucleus
LR	lateral reticular nucleus
LRcp	ventral external arcuate nucleus (parvocellular lateral reticular nucleus)
M	mamillary nuclei
MD	medial dorsal thalamic nucleus
mth	mamillothalamic tract
nIII	oculomotor nerve
nVIII	cochlear nerve
OcM	oculomotor nucleus
OI	inferior olivary nucleus
OSL	superior olivary nucleus
ped	cerebral peduncle
Prt	pretectal complex
Prtc	caudal pretectal nucleus
pyr	pyramidal tract
R.F.	rhinal fissure
rfl	fasciculus retroflexus
RN	red nucleus
rVm	major (sensory) root of trigeminal nerve
rVn	minor (motor) root of trigeminal nerve
Sth	subthalamic nucleus
tgl	lateral tegmental area
tgP	deep tegmental area
TgV	ventral tegmental area
TrMo	motor trigeminal nucleus
tro	optic tract
trs	spinal trigeminal tract
TrSc	caudal spinal trigeminal nucleus
TrSi	interpolar spinal trigeminal nucleus
tsL	lateral nucleus of tractus solitarius
VstL	lateral vestibular nucleus
VstM	medial vestibular nucleus
xVB	ventral basal thalamic nucleus