The ‘exact’ origin of the pyramidal tract

A quantitative study in the cat

BY H. van CREVEL and W. J. C. VERHAART

Institute of Neurological Sciences, Leiden University

INTRODUCTION

In 1851, Türck described secondary degeneration of the medullary pyramid in cases of cerebral lesions. Since then, many studies have been devoted to the origin of the pyramidal tract. Though the techniques have been improved, most of these studies were based on the method inaugurated by Türck: tracing secondary degeneration in pathological (or experimental) material. In this way, it was established that most pyramidal fibres arise in the cerebral cortex, and, more specifically, in the pre- and post-central regions. Fibres from other cortical regions have also been described (see Literature).

However, there is no agreement on the question: how many, and of what size, are the fibres from these different cortical regions, that is: on the question of the ‘exact’ origin of the pyramidal tract. Häggqvist, in 1937, concluded from the number of normal fibres in the pyramid after lesions of the precentral area had been made, that five-sixths of the fibres in the pyramid do not belong to the pyramidal tract (monkey). It is noteworthy that he observed that these ‘non-pyramidal’ (normal) fibres were thin. Lassek (1942a) found more than half of the pyramidal fibres intact after large lesions of the pre- and post-central cortex in the monkey. The parietal lobe contributed few if any fibres to the pyramid. Later, he stated that about one-third of the fibres in the pyramid were normal after a lesion of all cortex in front of the central sulcus; again, these were the thin fibres (1952). He concluded that these fibres must arise elsewhere, but did not state from what region. However, Morin, Poursines & Maffre (1951) were able to determine the cortical origin of 98% of the pyramidal fibres in the dog. Maffre (1953) concluded that 80–85% originate from the posterior sigmoid gyrus (the precentral area in the dog) and the remainder from the cortex immediately before and behind that area. She made no attempt to differentiate between the various fibre sizes in the pyramid.

Nevertheless, the view that the origin of a large proportion of the pyramidal fibres is unknown has become widely accepted. Tower (1949) states that only 50% of the pyramidal fibres can be accounted for as arising in the precentral area and the parietal lobe. In her opinion, the cortical origin of the remainder has yet to be found. Brodal (1958) adopts the same view, also citing Lassek’s work. In a recent review by Bucy (1957), it is again stated that the origin of approximately 50% of the pyramidal fibres is unknown. However, the evidence from nearly all work on the subject in various species points to the pre- and postcentral areas as the origin of the pyramidal tract, with perhaps smaller contributions from frontal and parietal areas. Most workers have not been able to find any fibres from...
the temporal and occipital lobes (see Literature). The problem then arises: in what cortical region do the unaccountable 50% originate?

The present work is a quantitative study of this problem in the cat. Its central theme is the view that the confusion mentioned above is due to a lack of knowledge of secondary degeneration. More specifically, it is maintained that the notion that some 50% of the fibres in the pyramid arise outside the pre- and postcentral cortex is a misconception, caused by neglect of the time factor in secondary degeneration. In a previous study (van Crevel & Verhaart, 1963a), the rate of secondary degeneration in the pyramidal tract of the cat was studied. It was found that this rate is determined by the size of the fibres, thick fibres degenerating faster than thin ones.* As the pyramidal tract consists mainly of thin fibres, many of these fibres remain 'normal' for a long time after severance. The confusion appears to be caused by these thin 'normal' fibres. They were seen not to be degenerated—therefore they were thought not to be severed. The present study makes use of the aforementioned data on the degeneration rate, and is based on the following line of reasoning.

If ablation of the entire (iso)cortex (from which all pyramidal fibres arise, see Literature) results in degeneration of $c \%$ of the fibres of a certain size after a certain time, and ablation of area $A$ in degeneration of $a \%$ of the same size after the same time, then $a/c \times 100 \%$ of the fibres of that size must arise from $A$. When ablations of various areas, together comprising the whole cortex, using different degeneration times, are worked out in this way, there are two ways of checking the results. First, the proportion $a/c$ of a given area must be equal for different degeneration times. Secondly, the percentages of fibres of a certain size from the various areas must add up to 100. It is to be expected that these requirements will be met only in a crude way, owing to the lack of precision of the lesions, the flaws in our method of counting and measuring, and to ever-present individual variation. In principle, however, the opportunity to check the conclusions afforded by the method described seems to promise a final settlement of the problem. A synthesis in quantitative terms can then be reached.

The cerebral cortex of the cat was subdivided into six regions, and a number of ablations of each region was performed (see Material). Some of the experimental animals were killed after 3 weeks, others after 16 weeks. For each fibre size, the number of degenerated fibres in the pyramid ($a$) was determined (see Methods). This number was compared with the corresponding number in cases of total de- cortication with the same degeneration time ($c$). Thus, the number of severed fibres could be calculated from the number of degenerated fibres. In this way, all pyramidal fibres could be accounted for, and the results could be roughly checked as described above. It was found that in the cat nearly all pyramidal fibres originate in the anterior third of the cerebral cortex, most of them in the pre- and postcentral regions.

* 'Degeneration rate' is defined as the relation between the number of degenerated fibres and the degeneration time (i.e. the time-interval between severance of the fibres and death of the specimen).
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LITERATURE

To substantiate the assumptions made in the Introduction, the relevant literature on the origin of the longitudinal fibres in the medullary pyramid will be reviewed in the following order:

(1) Do all descending fibres arise in the cerebral cortex?
(2) Do ascending fibres exist?
(3) Do contralaterally descending fibres exist?
(4) Does intermingling with fibres from other tracts occur?
(5) From what cortical regions do the fibres originate?

(1) Descending fibres. The cortical origin of the pyramidal fibres in man was established during the last century. Türek (1851) stated that when degeneration in the pyramid was found in cases of lesions in the lentiform nucleus, the cortex was involved as well. Charcot & Flechsig (as cited by Nathan & Smith, 1955) found degeneration in cases of lesions of the central cortical area, but not in cases involving the lentiform and caudate nuclei. Since then, only Swank (1986) has contended that fibres arise from the lentiform nucleus (in the rabbit); but his lesions involved the cortex as well. Later, the cortical origin of all pyramidal fibres was confirmed in man (Lassek & Evans, 1945) and in primates (Mettler, 1944). Lewandowsky (1904) had studied the problem in the cat, and he also found that all pyramidal fibres arise in the cerebral cortex. The same holds for the dog’s pyramid (Morin et al. 1951). Thus, it seems well established that no descending fibres in the pyramid spring from the basal ganglia.

(2) Ascending fibres. Although in older studies no degeneration could be found rostral to transection of the pyramidal tract in experimental animals (Lassek, 1942b; Tower, 1940), ascending fibres have been claimed more recently by Brodal & Walberg in the cat (1952). The latter authors used the Glees method. Their assertion has been questioned by Glees & Nauta (1955) in a critical review of the silver methods. Moreover, ascending fibres in the pyramidal tract of the cat could not be found with the Häggqvist method (van Beusekom, 1955; van Crevel, 1958). Both Patton & Amassian (1955), and Landau (1956a) are of the opinion that the so-called ascending impulses in the pyramidal tract (Brodal & Kaada, 1958) were led off from the medial lemniscus. Although the matter has not been settled conclusively, it seems clear that if ascending fibres do occur in the pyramid at all, their number is negligible.

(3) Contralaterally descending fibres. Fibres crossing in the corpus callosum and descending in the contralateral pyramid have been described in the cat (Walberg & Brodal, 1954); Kennard & Watts (1934) did not find them in the monkey. Other authors have described fibres crossing at pontine levels and descending contralaterally (cf. Verhaart & Kramer, 1952). In the cat, a few degenerated fibres are found occasionally in the pyramid opposite to a hemidecortication, but their number is far less than 1% of the total (van Crevel, 1958).

(4) Intermingling of fibres. The medullary pyramid is one of the best demarcated tracts in the central nervous system. In the cat it is limited on its whole dorsal border by the medial lemniscus (Glees, Liddell & Phillips, 1951; Verhaart, 1954).
This boundary can be located easily in normal preparations stained with the Häggqvist method. However, some lemniscal fibres are always found in the dorsal part of the pyramid. Occasionally, other non-corticospinal fibres are seen in the pyramid. They are external arcuate fibres on their way from the nucleus of the anterior funiculus to the cerebellum (Busch, 1961). Sometimes they run in a longitudinal direction, but usually they run transversely. The number of 'foreign' fibres in the pyramid of the cat may amount to some 2–8%—this is in accordance with the data on the dog (Maffre, 1958).

(5) The cortical regions. Before the turn of the century, the cortical origin of the pyramidal tract was thought to be widespread, including at least the pre- and postcentral gyri in man (Flechsig, 1876; Gowers, 1885; Déjerine, 1901), and the sylvian and coronal gyri in carnivores (Loewenthal, 1886). Although the electrical excitability of part of the cortex had been discovered, and Betz had described the cells named after him, neither this 'motor' cortex nor the area gigantopyramidalis (studied already by Lewis in 1878) had been strictly correlated with the origin of the pyramidal tract. Further studies on the motor cortex and the effects of its ablation in apes (Grünbaum & Sherrington, 1901) led Campbell to define the motor area anatomically as the area containing Betz cells (1905). In the cat, this cyto-architectonic area consisted of the sylvian gyrus, according to Campbell; Brodmann (1905, 1906) found it to be larger. Inspired by the harmonizing discoveries of anatomy and physiology, Holmes & May performed their work on 'the exact origin of the pyramidal tracts in man and other mammals' (1909). Using the method of retrograde cell degeneration, they concluded that the pyramidal tract arises from the Betz cells only, a conception still found in recent textbooks. In the opinion of these authors, the region of origin corresponded well with the motor area as defined by the aforementioned physiologists and anatomists. In the cat, it comprised chiefly the posterior sylvian gyrus; the anterior sylvian and coronal gyri were assumed to be the origin of corticobulbar fibres. It is now generally recognized that the method of retrograde cell degeneration furnishes positive evidence only, and that no conclusions can be derived from absence of chromatolysis. Moreover, counts revealed later that the Betz cells can be the origin of only 2–3% of the pyramidal fibres (Lassek, 1940, 1941, 1943).

Meanwhile, other workers found the origin of the pyramidal tract to be larger than the precentral gyrus, both in frontal and in occipital directions (von Monakow, cat and dog, 1914, man, 1915; Minkowski, monkey, 1924). Fibres from the (frontal) gyrus proreus were demonstrated in the cat by McKibben & Wheelis (1982). Hoff (1935) and Kennard (1935), working in the same laboratory, showed pyramidal fibres originating from area 6 in the monkey, using the bouton and Marchi methods respectively. Their results could not be confirmed by Verhaart & Kennard (1940). However, corticospinal fibres from area 6 were also found by Minckler, Klemme & Minckler in man (1944) and by Mettler in the monkey (1947). By comparison of fibre counts of the cerebral peduncle and pyramid, Verhaart established that thin fibres from Arnold's bundle run in the pyramid (man, 1947; monkey, 1948a; ape, 1948b). Corticospinal fibres from the postcentral gyrus were demonstrated in the monkey by the method of retrograde cell degeneration by Levin & Bradford in 1938. Peele (1942) traced degenerated fibres from lesions in the parietal lobe to the
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cord in the monkey, and his pupils Gobbel & Liles did the same in the cat (1945).

Walberg & Brodal, using the Glees method, found corticospinal fibres from the occipital and temporal cortex in the cat (1954). This observation has not been made before, and requires confirmation. Manghi (1954), using forty cats, described the origin of the pyramidal tract as follows: areas 4, 1, 3 and 6, and the gyrus proreus and ectosylvius anterior, in order of importance. Fibres from the parietal areas 5 and 7 (gyrus lateralis and suprasylvius medius) were doubtful; no fibres from temporal and occipital areas could be found. Chambers & Liu (1957) state that the corticospinal system of the cat originates from the sigmoid, coronal and anterior ectosylvian gyri. They found no fibres from parietal, occipital and temporal regions, nor from the gyrus proreus. The results of the two last-mentioned investigations agree well with those of antidromic stimulation of the pyramid in the cat (Woolsey & Chang, 1948; Lance & Manning, 1954; Porter, 1955). This may be coincidence: Landau (1956b) has observed that the method is suitable to detect large fibres only. According to him, the more widespread origin found by other workers is due to diffuse recording of antidromic potentials and to orthodromic potentials from the medial lemniscus.

Summarizing, we may conclude that:

1. All descending fibres in the pyramid originate from the cerebral cortex.
2. Ascending pyramidal fibres are non-existent or negligible in number.
3. The same holds for contralaterally descending fibres.
4. Some 'foreign' fibres run in the pyramid, their number being some 2–3%.
5. Many pyramidal fibres arise in the pre- and postcentral regions, while fibres from the frontal and parietal regions have also been reported; but fibres from the temporal and occipital lobes have not been found by most workers.

METHODS

Staining. The Hagqvist modification of the Alzheimer–Mann methylblue-eosin stain was used, both for fibre analysis of the sections of the medullary pyramid and for the serial sections of the cortical lesions. This technique stains myelin sheath and nucleolus a bright red, axons, cells and glial tissue blue. Degenerated fibres can be distinguished as follows. In thick fibres, the axon swells and becomes hyperchromatic or metachromatic (red); in later stages it is pale, granular or partly resorbed. In thin fibres, a blue axon is lacking and the whole fibre shows a vicious red hue. In all fibres, the myelin sheath is swollen, cloudy and irregular. Artefacts are rare and can be distinguished by the use of serial sections or, in experimental material with strictly unilateral lesions, by comparison with the contralateral side of the section. The technique has been recently described by Busch (1961) (see van Crevel & Verhaart, 1963a).

The 'indirect' method. The number of degenerated fibres of each size in the pyramids of the experimental animals had to be determined. However, since degenerated fibres vanish sooner or later by resorption, to count those still visible is misleading. Moreover, degenerated fibres are swollen; and so, to measure them is also of little value. This difficulty was overcome by Verhaart (1947). Instead of the
degenerated fibres, the non-degenerated fibres in the degenerating pyramid are counted and measured. The same is done in the contralateral, normal pyramid of the same specimen. Both sets of data are then compared. In this ‘indirect’ way, the number and size of the degenerated fibres are found. The method is based on the assumption that number and pattern of the pyramidal fibres of both sides in a normal specimen are the same. This has been tested and confirmed for the cat by the present authors (1963a).

Fibre analysis was performed as follows. The (normal) fibres were counted and measured directly from the microscope, with the aid of a net-ocular micrometer. Then the whole surface area was determined and the total number of (normal) fibres calculated by multiplication. Ten fields under oil immersion were taken as samples. The fibres were measured and counted simultaneously with the aid of a differential counting machine. They were allocated to the following size groups: 0–2, >2–4, >4–6 and >6μ. Fibres on the outer lines of the square of the net-ocular were counted at two of the four sides only. The surface was measured microscopically, using the net-ocular.

Obviously, the method is subject to many errors. Apart from the imperfections of the histological material, the methods of fibre measuring and counting, of surface measuring, and of judging degeneration are inexact. This is the more unsatisfactory because the errors cannot be determined statistically. Fortunately, the errors are not correlated and tend to reach a constant level when work of this kind has been done for some time. The over-all error is estimated at some 10% for thick fibres, and, since they are more numerous in the pyramid, at rather less for thin fibres.

Operations were performed under intraperitoneally administered hexobarbitone anaesthesia. The various cortical regions (see Material) were removed by suction. After a degeneration time of 3 or 16 weeks, the animals were killed in deep anaesthesia by perfusion with a 10% formalin solution through the aorta. Transverse sections from the medulla at a mid-olivary level were stained with the Håggqvist method and used for fibre analysis. The hemispheres containing the lesions were cut serially at 6μ in a frontal plane, and every twentieth section was mounted and also stained with the Håggqvist method. Microscopical control of the lesions revealed that here is another important source of error (see Material).

MATERIAL

Subdivision of the cortex. The cytoarchitectonic maps of the cerebral cortex produced so far are unsatisfactory. The existing maps were made with the use of arbitrary or even irrelevant criteria (Le Gros Clark, 1952), and of methods that fail to show the total structure of cells and fibres (Sholl, 1956), while most workers paid no heed to individual variation (Lashley & Clark, 1946). Moreover, Walshe (1942) showed that the Betz cells do not constitute a specific morphological or functional entity, but merge gradually into the simple giant cells and large pyramidal cells. Therefore, subdivisions of the cerebral cortex with the purpose of establishing the origin of the pyramidal tract can claim no exact anatomical basis. Subdivisions on a physiological basis are similarly inadequate, in view of the ‘sensorimotor’ pro-
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Properties of the central cerebral cortex (Albe-Fessard, 1957). After consulting the anatomical work of Lewis (1878), Brodmann (1905, 1906), Campbell (1905) and Winkler & Potter (1914), and the physiological publications on the motor and sensory cortex by McKibben & Wheelis (1932), Ward & Clark (1935), and Garol (1942a, b), we subdivided the isocortex into six regions, as shown in Text-fig. 1.

They will be called frontal, precentral, postcentral, parietal, temporal and occipital regions. The terms 'precentral' and 'postcentral' are used for lack of better names: we realize that these regions are not strictly comparable to the pre- and postcentral gyri in primates and man, as supposed by Campbell (1905). The precentral region comprises the gyrus sigmoideus, except the medial strip of the gyrus sigmoideus anterior, up to the sulcus posterocruziatus or its usual location; the postcentral region comprises the coronal gyrus with the strip behind the sulcus postcruciatus and the gyrus compositus rostrally.

Description of the lesions. All lesions were unilateral and left-sided. In addition to the lesions of the six regions described above, separate ablations were performed on the motor foreleg and hindleg areas (Garol, 1942a), together constituting the precentral region. Degeneration times of 3 and 16 weeks were used only. All experiments are found in Table 1. Since the neurological motor signs (placing and hopping reactions, etc.) proved to be a reliable indication of damage to the sigmoid gyrus or its white matter, they will be mentioned with the microscopic description of the lesions. These signs were seen only contralaterally. The following description
of the lesions was made from serial sections, in which both grey and white matter were inspected.

**H 3175 (frontal ablation).** The gyrus proreus and a strip of the gyrus diagonalis have been removed. The white matter of the fronto-medial gyrus sigmoideus anterior is destroyed. A caudal extension of the lesion tapers into the white matter latero-ventral of the nucleus caudatus (Text-fig. 2).

**Table 1. Partial cortical ablations**

<table>
<thead>
<tr>
<th>Cat no.</th>
<th>Ablation</th>
<th>Degeneration time (weeks)</th>
</tr>
</thead>
<tbody>
<tr>
<td>H 3175</td>
<td>Frontal</td>
<td>3</td>
</tr>
<tr>
<td>H 3469</td>
<td>Frontal</td>
<td>3</td>
</tr>
<tr>
<td>H 3516</td>
<td>Frontal</td>
<td>3</td>
</tr>
<tr>
<td>H 4302</td>
<td>Frontal</td>
<td>3</td>
</tr>
<tr>
<td>H 3471</td>
<td>Frontal</td>
<td>16</td>
</tr>
<tr>
<td>H 3472</td>
<td>Precentral</td>
<td>3</td>
</tr>
<tr>
<td>H 3473</td>
<td>Precentral</td>
<td>16</td>
</tr>
<tr>
<td>H 3532</td>
<td>Precentral</td>
<td>16</td>
</tr>
<tr>
<td>H 3606</td>
<td>Sigm. post.</td>
<td>3</td>
</tr>
<tr>
<td>H 3607</td>
<td>Sigm. ant.</td>
<td>3</td>
</tr>
<tr>
<td>H 3529</td>
<td>Postcentral</td>
<td>3</td>
</tr>
<tr>
<td>H 3492</td>
<td>Postcentral</td>
<td>3</td>
</tr>
<tr>
<td>H 3530</td>
<td>Postcentral</td>
<td>16</td>
</tr>
<tr>
<td>H 3475</td>
<td>Postcentral</td>
<td>16</td>
</tr>
<tr>
<td>H 3493</td>
<td>Parietal</td>
<td>3</td>
</tr>
<tr>
<td>H 3494</td>
<td>Parietal</td>
<td>16</td>
</tr>
<tr>
<td>H 3511</td>
<td>Temporal</td>
<td>3</td>
</tr>
<tr>
<td>H 4060</td>
<td>Temporal</td>
<td>3</td>
</tr>
<tr>
<td>H 3512</td>
<td>Temporal</td>
<td>16</td>
</tr>
<tr>
<td>H 3531</td>
<td>Occipital</td>
<td>3</td>
</tr>
<tr>
<td>H 3517</td>
<td>Occipital</td>
<td>16</td>
</tr>
</tbody>
</table>

**H 3469 (frontal ablation).** Motor symptoms were present in both contralateral limbs. The gyrus proreus and medial gyrus sigmoideus anterior have been removed. A deep softening in the gyrus sigmoideus posterior is found.

**H 3516 (frontal ablation).** Motor symptoms were unmistakable. The gyrus proreus and rostral gyrus diagonalis have been removed. Frontally the white matter of the gyrus sigmoideus anterior has been undermined. Caudally a small infarct is located in the area of white matter containing fibres from the gyrus sigmoideus.

**H 4302 (frontal ablation).** No motor symptoms were observed. The basal part of the gyrus proreus has been removed. Neither the gyrus sigmoideus anterior nor the gyrus diagonalis has been damaged or undermined.

**H 3471 (frontal ablation).** Slight motor symptoms vanished quickly (traumatic oedema?). The gyrus proreus has been removed. The medial part of the gyrus sigmoideus anterior (area 6) has been destroyed or its white matter has been interrupted. The gyrus compositus and diagonalis have been spared (Text-fig. 2).

**H 3472 (precentral ablation).** Motor symptoms were conspicuous. The gyrus sigmoideus has been removed, the depth of the coronal sulcus included. The lesion has pierced part of the white matter containing fibres from the frontal region. Possibly some fibres from the postcentral region have been severed by extensions from the lesion (Text-fig. 2).
Text-fig. 2. Ablations of the cases represented in Tables 2 and 3; ablated tissue black, undermined or damaged tissue stippled.

H 3473 (precentral ablation). Motor symptoms were conspicuous. The gyrus sigmoideus has been removed, the most medial part in the caudal depth of the sulcus cruciatus excepted. This part was spared in all lesions; it is not known whether it belongs to the motor cortex. The gyrus proreus has not been damaged (Text-fig. 2).

H 3532 (precentral ablation). Motor symptoms were marked. The gyrus sigmoideus has been removed almost completely (see H 3473). The medial gyrus sigmoideus anterior has not escaped destruction but the gyrus proreus is intact. Some
fibres from the postcentral region may have been divided by an extension of the lesion into the white matter medial to the coronal sulcus (Text-fig. 2).

H 3606 (partial precentral ablation: hindleg area). Motor symptoms were most pronounced in the hindleg. The gyrus sigmoideus posterior has been removed completely; most of the grey matter of the gyrus sigmoideus anterior is intact, but the transition of that gyrus into the gyrus sigmoideus posterior has been extirpated, while some fibres from the dorso-caudal part of the gyrus coronalis may have been interrupted.

H 3607 (partial precentral ablation: foreleg area). Motor symptoms were striking in foreleg, only slight in hindleg. The gyrus sigmoideus anterior and its transition into the gyrus sigmoideus posterior have been removed. Part of the latter has been damaged. Fibres from the dorsal part of the gyrus coronalis may have been interrupted. The lesions of H 3606 and H 3607 are overlapping.

H 3529 (postcentral ablation). Motor symptoms almost disappeared in the course of 2 weeks. The lesion covers the gyrus coronalis and a caudal strip of the gyrus sigmoideus posterior. Hemorrhages and infarcts extend into the lateral part of the internal capsule. Many fibres from the gyrus sigmoideus must have been interrupted.

H 3492 (postcentral ablation). Motor symptoms persisted. The extent of the lesion is similar to that of H 3529.

H 3530 (postcentral ablation). Motor symptoms were definitely present. The lesion again is roughly similar to that of H 3529. The slips into the internal capsule are even deeper.

H 3475 (postcentral ablation). No distinct motor symptoms could be established. The gyrus coronalis and caudal strip of the gyrus sigmoideus posterior have been removed superficially, the transition of gyrus coronalis into gyrus ectosylvius anterior has been damaged. There are no extensions of the lesion into the internal capsule (Text-fig. 2).

H 3493 (parietal ablation). No motor symptoms were noticed. The gyrus suprasylvius medius and the rostral half of the gyrus lateralis have been removed, with small slips into the gyrus ectosylvius medius and lateralis posterior.

H 3494 (parietal ablation). No motor symptoms were present. The lesion is similar to that of H 3498 but deeper. Fibres from the occipital region are interrupted.

H 3511 (temporal ablation). Motor symptoms were absent. The lesion occupies the gyrus sylvius, the gyrus ectosylvius posterior and medius and part of the gyrus ectosylvius anterior, the gyrus suprasylvius posterior and the inferior part of the gyrus lateralis posterior. The basal cortex has been spared. The lesion reaches into the lateral ventricle, and approaches the lateral part of the internal capsule so closely that indirect damage during the operation is not out of the question.

H 4060 (temporal ablation). Motor symptoms were absent. The lesion, not examined microscopically, is similar to that of H 3511.

H 3512 (temporal ablation). Motor symptoms were absent. The cortex displays a highly abnormal pattern of gyri and sulci, but the lesion is similar to that of H 3511. Even deeper extensions round the internal capsule have interrupted fibres from the occipital region.

H 3531 (occipital ablation). The caudal half of the gyrus lateralis and upper half
of the gyrus lateralis posterior have been removed. In the underlying white matter areas of infarction are seen.

H 3517 (occipital ablation). This animal was soporose during the last part of the post-operative period, and suffered from convulsions. At autopsy it appeared that the cortex surrounding the lesion had herniated. The cortex has been transformed into glial tissue with small cysts. Part of the parietal and even the temporal cortex is damaged, and the entire occipital region is destroyed.

At autopsy all experiments of Table 1 (except H 3517) seemed successful, though we realized that fibres from other regions might have been interrupted in some cases. Earlier experiments had facilitated localization and indicated how herniation of surrounding cortex may be prevented. Histological examination, however, revealed that extensions from the primary lesion into the white matter are prevalent when the suction technique is used. The majority of these secondary lesions are minute and of no consequence, but some occupy essential areas of white matter. From the description of the lesions it will be clear that many of the ablations were failures, judged from the exacting aim of the series that together they should cover the entire cortex, without overlapping.

RESULTS

Inspection of the pyramids confirmed the conclusion derived from histological examination of the lesions. Several ablations of the frontal and postcentral regions had damaged the white matter to such an extent that heavy degeneration of the pyramid had resulted. Of the frontal ablations, H 3469 and H 3516 showed a very pronounced degeneration in the pyramid, comparable in degree to that following ablation of the gyrus sigmoideus. The pyramids of postcentral ablations H 3529, H 3492 and H 3530 showed the same aspect (cf. description of the lesions). Although fibre analysis was performed in these cases, the results were not taken into account, except to verify the qualitative observations. The pyramid of H 4302 (frontal ablation) showed hardly any degeneration and was not used for fibre analysis, but only to specify the conclusions drawn from the other frontal ablations. The pyramids of cases with parietal, temporal, and occipital ablations showed little or no degeneration, while the lesions were in any case not too small; therefore these cases were all regarded as usable.

Frontal, precentral and postcentral ablations

H 3175 and H 3471 had ablations limited to the frontal region (the medial part of the gyrus sigmoideus included). In the former, killed 3 weeks after the operation, degeneration in the pyramid is unmistakable, although only a small number of fibres of various sizes are affected. (In these, as in all other pyramids examined, the degeneration is distributed evenly throughout the pyramid.) The pyramid of the latter, killed after 16 weeks, contains a few large degenerated fibres; but careful examination reveals the thin fibres to be less numerous than in the normal pyramid. Evidently, a number of thin fibres have been cleared away after degenerating (Pl. 1, fig. 1). The degeneration resulting from a lesion of the gyrus proreus only (H 4302, 3 weeks) is very slight. Apparently, the pyramidal fibres from the frontal region originate mainly in the medial part of the gyrus sigmoideus anterior.
In precentral ablation H 3472 (3 weeks), marked degeneration of the pyramid is present. However, it seems to be less massive than that found 3 weeks after hemidecortication. In the pyramids of H 3473 and H 3532 (same lesion, 16 weeks) very many fibres are degenerated (Pl. 1, fig. 2), but not nearly so many as 16 weeks after a hemidecortication. Degeneration in the pyramids of H 3606 and H 3607, with overlapping lesions of the hindleg and foreleg motor area (3 weeks), is almost as heavy as that found in H 3472. It is noteworthy that in these two cats, symptoms were seen chiefly in the hindleg and foreleg respectively.

The only case with a lesion more or less restricted to the postcentral area is H 3475 (16 weeks). Under low-power magnification the pyramid looks almost normal, with only mild gliosis as a sign of degeneration. Under oil-immersion magnification, however, it is seen that the thin fibres are reduced in number, although the large fibres seem hardly affected (Pl. 1, fig. 8).

Quantitative analysis. Fibre analysis was performed (on both pyramids) in cases with lesions of the frontal, precentral and postcentral regions. The results confirmed those obtained from histological examination of the lesions: large numbers of fibres were found to be degenerated in cases of frontal and postcentral ablations damaging the internal capsule. The degeneration following the ablations of the motor foreleg and hindleg areas was also abundant, indicating even more overlap of the lesions than had been suspected. Because the data about these cases are not relevant to our quantitative conclusions, they will not be reported here. The results of counts in cases with ablations more or less restricted to the frontal, pre- or postcentral regions are shown in Table 2. They are presented as percentages of degenerated fibres.

Table 2. Percentages of 'degenerated' fibres in the pyramid after partial cortical ablations

<table>
<thead>
<tr>
<th>Cat no.</th>
<th>Ablation</th>
<th>Time (weeks)</th>
<th>0-2 μ</th>
<th>&gt;2-4 μ</th>
<th>&gt;4-6 μ</th>
<th>&gt;6 μ</th>
</tr>
</thead>
<tbody>
<tr>
<td>H 3175</td>
<td>Frontal</td>
<td>3</td>
<td>9-7</td>
<td>8-6</td>
<td>12-5</td>
<td>20-3</td>
</tr>
<tr>
<td>H 3471</td>
<td>Frontal</td>
<td>16</td>
<td>12-6</td>
<td>8-7</td>
<td>8-1</td>
<td>4-2</td>
</tr>
<tr>
<td>H 3472</td>
<td>Precentral</td>
<td>3</td>
<td>26-4</td>
<td>39-7</td>
<td>64-2</td>
<td>84-7</td>
</tr>
<tr>
<td>H 3473</td>
<td>Precentral</td>
<td>16</td>
<td>59-6</td>
<td>59-8</td>
<td>51-9</td>
<td>78-7</td>
</tr>
<tr>
<td>H 3532</td>
<td>Precentral</td>
<td>16</td>
<td>70-3</td>
<td>67-9</td>
<td>55-8</td>
<td>70-2</td>
</tr>
<tr>
<td>H 3475</td>
<td>Postcentral</td>
<td>16</td>
<td>27-9</td>
<td>35-9</td>
<td>36-6</td>
<td>5-9</td>
</tr>
</tbody>
</table>

To calculate the percentages of severed fibres, these data were multiplied by 100/c (where c = the percentage of degenerated fibres of the size in question, after hemidecortication with the same degeneration time). The values of c were derived from the previous study. For the first three size groups it was calculated from the regression equations; for group 4 it was found by approximation (see van Crevel & Verhaart, 1963a). The conversion factor 100/c for 3 weeks was computed as 2.86, 1.61, 1.22 and 1.09 for the four size groups respectively; for 16 weeks its values were 1.05, 1.02, 1.00 and 1.00. Multiplication by the appropriate factor resulted in the data of Table 3.

These data can be summed up as follows (see the approximate values in Table 3). In the cases of frontal ablations 10–15% of the pyramidal fibres were severed.
Evidently the lesion of case H 3175 had undermined more white matter of the gyrus sigmoideus anterior than that of H 3471. In the latter, mainly fibres thinner than 6 μ were interrupted, while in the former a considerable number of larger fibres were also severed. The ablations of the precentral region have divided the majority of the fibres of all sizes. Although large fibres have suffered more severely than thin fibres, this difference is not striking. About 80% of the large fibres (>6 μ) have been severed, as opposed to some 65% of the other ones. The fibres interrupted by the lesion of the postcentral region are nearly all under 6 μ in diameter. In all, some 30% of the pyramidal fibres have been severed by this ablation.

From all that has been said about the lesions, added to what we know about the technique of fibre analysis, it will be clear that we can expect errors of more than 10%. For that reason, the two ways of checking, as described in the Introduction, are applied only tentatively. The first check is made by comparing the results derived from cases with similar lesions but different degeneration times. It will be admitted that the three cases with precentral ablations (two with degeneration times of 16 weeks, one with a degeneration time of 3 weeks) yielded similar results. The second verification consists of adding the percentages of fibres from the various regions per size group: together they should be 100. For the thin fibres this sum exceeds 100 by 5–15%, while for the large fibres (>6 μ) the total is less than 100. Of course this difference may be caused by chance errors in counting and sizing, magnified by the conversion of the data (especially in the experiments with degeneration times of 3 weeks). However, an alternative explanation lies in the anatomical structure of the cortex. On the one hand, damage to some of the surrounding cortex can hardly be avoided in ablations of the precentral region, resulting in additional degeneration of fibres which are mainly thin. Similarly, lesions of the ‘postcentral’ region include the posterior strip of the gyrus sigmoideus posterior. On the other hand, the area containing Betz cells has probably never been completely removed, because of the depth, medially, of the sulcus cruciatus, so that possibly some large fibres were not severed. Thus, the deviations could be

<table>
<thead>
<tr>
<th>Cat no.</th>
<th>Ablation</th>
<th>Time (weeks)</th>
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<th>&gt;2–4 μ</th>
<th>&gt;4–6 μ</th>
<th>&gt;6 μ</th>
</tr>
</thead>
<tbody>
<tr>
<td>H 3175</td>
<td>Frontal</td>
<td>3</td>
<td>22.9</td>
<td>13.8</td>
<td>15.3</td>
<td>22.1</td>
</tr>
<tr>
<td>H 3471</td>
<td>Frontal</td>
<td>16</td>
<td>13.2</td>
<td>8.9</td>
<td>8.1</td>
<td>4.2</td>
</tr>
<tr>
<td>H 3472</td>
<td>Precentral</td>
<td>3</td>
<td>62.3</td>
<td>69.9</td>
<td>78.3</td>
<td>92.3</td>
</tr>
<tr>
<td>H 3473</td>
<td>Precentral</td>
<td>16</td>
<td>62.6</td>
<td>61.0</td>
<td>51.9</td>
<td>78.7</td>
</tr>
<tr>
<td>H 3532</td>
<td>Precentral</td>
<td>16</td>
<td>73.8</td>
<td>69.3</td>
<td>55.3</td>
<td>70.2</td>
</tr>
<tr>
<td>H 3475</td>
<td>Postcentral</td>
<td>16</td>
<td>29.3</td>
<td>36.6</td>
<td>36.6</td>
<td>5.9</td>
</tr>
</tbody>
</table>

Approximate values of these percentages:

<table>
<thead>
<tr>
<th>Cat no.</th>
<th>Ablation</th>
<th>Time (weeks)</th>
<th>0–2 μ</th>
<th>&gt;2–4 μ</th>
<th>&gt;4–6 μ</th>
<th>&gt;6 μ</th>
</tr>
</thead>
<tbody>
<tr>
<td>H 3175</td>
<td>Frontal</td>
<td>3</td>
<td>25</td>
<td>15</td>
<td>15</td>
<td>20</td>
</tr>
<tr>
<td>H 3471</td>
<td>Frontal</td>
<td>16</td>
<td>15</td>
<td>10</td>
<td>10</td>
<td>5</td>
</tr>
<tr>
<td>H 3472</td>
<td>Precentral</td>
<td>3</td>
<td>60</td>
<td>65</td>
<td>80</td>
<td>90</td>
</tr>
<tr>
<td>H 3473</td>
<td>Precentral</td>
<td>16</td>
<td>65</td>
<td>60</td>
<td>50</td>
<td>80</td>
</tr>
<tr>
<td>H 3532</td>
<td>Precentral</td>
<td>16</td>
<td>75</td>
<td>70</td>
<td>55</td>
<td>70</td>
</tr>
<tr>
<td>H 3475</td>
<td>Postcentral</td>
<td>16</td>
<td>30</td>
<td>35</td>
<td>35</td>
<td>5</td>
</tr>
</tbody>
</table>
explained by overlapping of the lesions and sparing of part of the precentral region together.

Conclusions based on this limited material should be formulated as follows. The gyrus proraeus and the medial part of the gyrus sigmoideus anterior together contribute a small number (some 10\%) of the fibres to the pyramid. Probably most of these fibres originate in the medial part of the gyrus sigmoideus anterior. Some 65\% of the fibres thinner than 6\(\mu\) and at least 80\% of those larger than 6\(\mu\) originate in the remainder of the gyrus sigmoideus, its most caudal strip excepted. The coronal gyrus and caudal strip of the gyrus sigmoideus (behind the sulcus postcruciatus), together with the gyrus diagonalis and gyrus compositus anterior, send a considerable number of thin fibres to the pyramid (about 30\%), but only few large fibres over 6\(\mu\) in diameter.

**Parietal, temporal and occipital ablations**

No complete fibre counts were performed in this group, because degeneration in the pyramid was either absent or insignificant.

The pyramid of H 3493 (parietal ablation, 3 weeks) shows an extremely small number of degenerated fibres. Though few in number, the large ones are clearly visible, while thin degenerated fibres can hardly be found. The pyramid of H 3494 (same lesion, 16 weeks) looks perfectly normal.

The pyramids of H 3511 and H 4060 (temporal ablations, 3 weeks) show an aspect similar to that of H 3493. The pyramid of H 3512 (same lesion, 16 weeks) looks normal.

In the pyramids of H 3531 and H 3517 (occipital ablations, 3 and 16 weeks respectively) a very small number of degenerated fibres is again seen. In both, the picture looks like that in the cases of parietal and temporal ablations with a 3 weeks’ degeneration time.

When these cases are considered together, it appears that 3 weeks after parietal, temporal, or occipital ablations some degenerated fibres may be found in the pyramid. These were not visible when the degeneration time had been 16 weeks, possibly because they had been resorbed completely. H 3517 (16 weeks) forms the exception to this rule; but it will be remembered that in this case the cortex surrounding the lesion had herniated extensively and that the animal had displayed signs of increased intracranial pressure. However, none of these cases is clear-cut. The parietal lesions encroach rostrally on the gyrus sigmoideus posterior, while the temporo-occipital lesions have only just spared the internal capsule. Therefore, some indirect traumatic effect seems not inconceivable, especially because mainly large fibres were involved. In any case, the number of degenerated fibres resulting from all these lesions together was estimated as well under 1\% of the total number.

Therefore, the conclusion was drawn that few if any fibres of the pyramidal tract originate in the parietal, temporal and occipital regions.

**DISCUSSION**

**The method.** Making perfectly circumscribed cortical ablations is difficult, though this is realized only when the lesions are examined histologically with a method showing not only cells but also the white matter. This circumstance has limited our
The ‘exact’ origin of the pyramidal tract

material, and final or detailed conclusions are as yet impossible. However, because the data supplement each other, an impression can be gained about the exactitude of the results. The final error in quantitative data of one experiment may amount to 10 or 20%; but comparison of various experiments and the simultaneous use of ordinary microscopic examination will yield more reliable results. Degeneration of some 5% of the fibres in degeneration times of about 3 weeks can be easily established by inspection but not by counting (because non-degenerated fibres have to be counted, see Methods). On the other hand, a considerable number of thin fibres may have vanished completely after degeneration times of 16 weeks or more—this is very difficult to observe but will manifest itself in counts. Estimating and comparing the degree of degeneration in areas of a different fibre pattern, especially when different degeneration times are used, is impossible without fibre analysis. Therefore, this latter method should be used in problems of that kind, and the material should contain experiments with both short and long degeneration times.

The results. McKibben & Wheelis (1932), confirmed by Manghi (1954), concluded that pyramidal fibres arise from the gyrus proreus; but Chambers & Liu (1957) found no degeneration in the pyramid in a case with ablation of that gyrus. From our material it was concluded that some 10% of the pyramidal fibres originate in the frontal region. This region includes the medial third of the gyrus sigmoideus anterior (see Brodmann, 1905; Campbell, 1905), and probably the majority of the fibres from the frontal region arise from that gyrus.

It could be established with certainty that not only most of the large fibres, but also the majority of the thin fibres of the pyramid arise in the sigmoid gyrus. The proportion of thin fibres may easily be underestimated (as has often been done) because of the difference in degeneration rate of thick and thin fibres. Studies with antidromic stimulation have also stressed the component of large fibres arising in this area, but Landau (1956b) has pointed out that this method is suitable only for large fibres. A relatively larger contribution to the pyramid could be established only for fibres over 6μ in diameter, and these constitute only 2% of the total number.

The gyrus coronalis of the cat, called postcentral region so far, is not strictly homologous to areas 3, 2 and 1 in primates, because it is also the motor face area (Garol, 1942a). In the face area of higher animals few or no Betz cells are found (von Economo & Koskinas, 1925, as cited by Walshe, 1942; Lassek, 1940), and in the cat they may be smaller than in the sigmoid gyrus. (This is indicated by the fact that Brodmann includes the gyrus coronalis in his area gigantopyramidalis, while Campbell does not.) This lack of giant cells may be correlated with our finding that chiefly thin fibres (<6μ) originate from the gyrus coronalis. Thus it completes the view generally held that, other things being equal, long fibres are also thick (Schwalbe, 1882; Szentágothai, 1941; Thiel, 1957), and that the leg area, containing most giant cells, sends relatively more thick fibres to the pyramid than the arm area, which contains fewer and smaller Betz cells (Häggqvist, 1987; Lassek, 1940, 1941, 1948; Lance & Manning, 1954). The relationship between giant cells in the motor area and large fibres in the pyramid is further substantiated by the lack of both at birth (Conel, 1939, 1941, 1947; Lassek, 1942c; Verhaart, 1950). The fact that the face area also sends fibres to the pyramid is not very surprising. Sherrington (1889) pointed out that degeneration resulting from discrete lesions of the arm
or leg area is not confined to the cervical or lumbar segments of the cord. This has been confirmed by Glees, Cole, Liddell & Phillips (1950), who, like Walsh (1951), consider it to be of great physiological importance. Fibres from the leg and arm areas have also been shown to terminate in the brain stem (Kuypers, 1958), and fibres from the face area to the cord seem to fit into this organization.

It has been stated already that our experiments furnish little or no evidence for the existence of pyramidal fibres from parietal, temporal, or occipital regions: the same conclusion was reached by Manghi (1954) and Chambers & Liu (1957). Parietospinal fibres in the cat were postulated only by Gobbel & Liles (1945); but their lesions encroached on the occipital part of the gyrus sigmoideus posterior. Pyramidal fibres from the temporal and occipital lobes have been described by Walberg & Brodal (1954); but their result could not be confirmed by the aforementioned authors. Moreover, the fibres from other regions have now been shown to make up about 100% of the pyramidal fibres (the surplus of thin fibres was attributed to overlap of the lesions). Therefore, it may be concluded that a contribution to the pyramidal from parietal, temporal and occipital regions cannot be quantitatively important.

The time factor. Attempts at exact, quantitative localization of the origin of the pyramidal tract were made by Haggqvist (1987) and Lassek (1942a, 1952). Examining the pyramids after ablations of the pre- and postcentral regions in monkeys, they were struck by the large number of normal fibres. Since their work the view tends to prevail that the origin of approximately 50% of the pyramidal fibres is unknown and has yet to be found (see Introduction, Tower, 1949; Brodal, 1953; Bucy, 1957). This is probably a misconception due to neglect of the time factor in secondary degeneration.

This view is supported by a quantitative study of the subject in the dog by Maffre (1953). Using the Weigert method, she was able to account for approximately all pyramidal fibres—but she used very long degeneration times. In the present study in the cat, both short and long degeneration times were used, and, after the time factor had been taken into account, the results of both could be shown to yield similar results. The data from Maffre’s study and the present work cannot be directly compared, because the area gigantopyramidalis in the dog is situated more posteriorly than in the cat (Brodman, 1906), and because no differentiation in fibre sizes was made by Maffre. Nevertheless, both studies indicate that the vast majority (over 90%) of the pyramidal fibres originate from the cortical motor area as defined physiologically.

Recently, the present view has been confirmed for the monkey (Macaca rhesus) in a very thorough study by Russell & De Myer (1961), who used silver impregnations. Their material comprises seventy animals with partial and complete unilateral cortical ablations. After quantitative analysis of the secondary degeneration in the pyramids, they conclude that in the monkey complete fibre degeneration takes 6–12 months. Virtually all pyramidal axons could be accounted for. From area 4 arose 31%, from area 6, 29%; and from the parietal lobe, 40%; while the occipital lobe sent no fibres at all to the pyramid, the temporal and frontal poles could not be completely excluded. It seems evident that in this respect considerable quantitative differences exist between carnivores and the monkey.
COMMENT

Knowledge of the process of secondary degeneration is essential for the correct anatomical interpretation of data from degeneration studies. More specifically, the degeneration rate should be known. This rate is determined by many factors (reviewed by van Crevel, 1958). Within one animal species, the most important factor is the size of the fibres in question. In the present study, it was demonstrated how information about the degeneration rate can be applied to a neuro-anatomical problem. Therefore, it seems appropriate to summarize here the findings on this subject, obtained from experiments on two different brain tracts (van Crevel & Verhaart, 1968a, b). Stress will be laid on the application of these findings in neuro-anatomy.

If degeneration rate is defined as the relation between the number of degenerated fibres and the degeneration time, then large fibres degenerate at a higher rate than thin ones. However, a large degenerated fibre remains visible as such for a long time after severance, while a thin one is rapidly resorbed. Therefore, the severed fibres of all sizes cannot be seen as degenerated at the same time. Degeneration is always partial, and when the last fibres are degenerating, the first have already been resorbed. (The terms ‘large’ and ‘thin’ are used in a comparative sense: transitions are gradual.)

Not even the fibres of one size degenerate simultaneously: they follow an exponential law, the number degenerating per time unit being proportional to the number present. This implies that most fibres of any size degenerate in an early stage; later fewer and fewer fibres break down. This, and the rapid resorption of thin fibres, cause degeneration of thin fibres to be most conspicuous a few weeks after severance. At later stages, it can only be established by very careful inspection or by counts. However, degeneration of large fibres, which are slowly resorbed, remains manifest for a long time. Thus, the behaviour of large fibres during degeneration corresponds to what is often taken for granted with respect to fibres of any size.

An exact analysis of tracts of composite origin by degeneration studies should be based on quantitative methods. Two principles should be remembered. First, for reasons explained above (see Methods), information should be gained from counting and measuring not degenerated fibres, but normal ones. The contralateral, normal tract should be used as a paired control (the ‘indirect’ method). Therefore, no quantitative data can be obtained from cases with bilateral lesions. Secondly, the degeneration rate should be taken into account. Preferably, two degeneration times should be used, e.g. 3 and 16 weeks. In early stages many severed fibres have not yet degenerated and quantitative differences will often be statistically insignificant. On the other hand, at late stages degeneration of a moderate but significant number of thin fibres may pass completely unnoticed (without counts), because the degenerated fibres are no longer visible as such.

When the long time needed for total degeneration of thin fibres is ignored, conclusions about the origin of thin ‘normal’ fibres in degeneration experiments are apt to be erroneous. This has been demonstrated above for the thin fibres in the pyramidal tract. A similar problem exists in isolation experiments of the cord. Thin
for neuro-anatomy

Finally, the caused misconception, gyrus coronalis between the cortex and criticism. 

The cortex was subdivided into six regions, and a number of ablations of each region was performed. Some of the experimental animals were killed after 3 weeks, some after 16 weeks. For each fibre size, the number of degenerated fibres in Häggqvist-stained sections of the pyramid was determined (see Methods). This number was compared with the corresponding number in cases of total decortication with the same degeneration time. In this way, the number of severed fibres could be calculated.

When the inevitable imperfections of the lesions and the inherent errors of the histological methods are recalled, it is evident that the results can never be truly 'exact'. Nevertheless, the following conclusions, checked in various ways, could be drawn from our limited material:

1. Nearly all fibres of the pyramid of the cat originate in the anterior third of the cerebral cortex (frontal, pre- and postcentral regions). A contribution from the parietal, temporal and occipital lobes is extremely small or non-existent.
2. In the gyrus proreus and medial third of the gyrus sigmoideus anterior together (the 'frontal' region) originate some 10% of the pyramidal fibres; most of them are thinner than 6 μ. Only few of them arise in the gyrus proreus.
3. The remainder of the gyrus sigmoideus, the strip behind the sulcus postcruciatus excepted (the 'precentral' region), is the origin of two-thirds of the fibres thinner than 6 μ and of at least 80% of those larger than 6 μ.
4. The rest of the fibres of the pyramid, most of them of small size, arise in the gyrus coronalis and its transition into the gyrus ectosylvius anterior, in the strip between the sulcus postcruciatus and ansatus, and in the gyrus diagonalis (the 'postcentral' region).

Further, it was concluded that the notion that at least 50% of the fibres of the pyramid arise from an unknown origin outside the pre- and postcentral cortex is a misconception, caused by neglect of the time factor in secondary degeneration. Finally, the significance of accurate data on the process of secondary degeneration for neuro-anatomy is stressed, and some of the relevant data are considered in the light of their application in neuro-anatomy.

The authors are deeply indebted to the late Dr D. A. Sholl for statistical advice and criticism.
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EXPLANATION OF PLATE

Pyramids 16 weeks after various partial cortical ablations (right) and the normal sides for comparison (left). Most degenerated fibres have been resorbed. Haggqvist stain, × 550.

Fig. 1. Frontal ablation: slight loss of thin fibres. H 3471.

Fig. 2. Precentral ablation: severe loss of fibres of all sizes. H 3473.

Fig. 3. Postcentral ablation: moderate loss of thin fibres. H 3475.