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RESEARCH ARTICLE

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Cortical regions contributing to the anterior commissure in man

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Abstract The human anterior commissure is believed, by extrapolation from data obtained in macaque monkeys, to convey axons from the temporal and orbitofrontal cortex. Reports of interhemispheric transfer and sexual dimorphism related to the anterior commissure, however, make more precise data on the human anterior commissure desirable. We investigated the connectivity of the human anterior commissure in six adults (male and female) that had circumscribed hemispheric lesions in temporal, frontal, parietal or occipital cortices or in infrapallidal white matter using the Nauta for anterogradely degenerating axons. Axons originating in the inferior part of temporal or occipital lobes, occipital convexity and possibly central fissure and prefrontal convexity were found to cross the midsagittal plane in the anterior commissure. The largest contigent of commissural axons originated in the inferior part of the temporal lobe; it displayed a roughly topographic organization, preferentially running through the inferior part of the commissure. The inferior temporal contigent seemed to reach homotopic and heterotopic targets in the opposite hemisphere. Among the latter were the amygdala and possibly the orbitofrontal cortex. The present data suggest that the human anterior commissure conveys axons from much larger territories than expected from work on non-human primates. Similarly to the human and non-human primate corpus callosum, the anterior commissure is roughly topographically organized and participates in heterotopic connectivity.

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Introduction

The human anterior commissure is a compact fibre bundle that crosses the midsagittal plane below and posterior to the rostrum of the corpus callosum. Within the hemisphere, the anterior commissure forms two limbs. A relatively detailed description of their trajectories was obtained with the fibre dissection technique (Klinger and Gloor 1960). The posterior limb, which carries the major part of the commissural fibres, travels within the basal parts of the putamen and the caudate nucleus and below the anterior border of the globus pallidus. After emerging at the lateral side of the putamen, the compact bundle fans out into the white matter of the temporal lobe; few fibres have been followed into the occipital lobe. The anterior limb, which is much smaller and varies considerably in size between subjects, includes small bundles of fibres that leave the main bulk of the commissure at the level of the anterior perforated space.

Relatively little is known about the regions of origin of human anterior commissural axons, which are presumed to be similar to those found in non-human primates. However, several interesting findings make human data desirable.

Intriguing results were reported concerning the size of the human anterior commissure; it was claimed to differ between the sexes, but the nature of the difference remains controversial. The surface of the mediosagittally cut anterior commissure was reported to be larger in men (Demeter et al. 1988) or in women (Allen and Gorski 1991). Furthermore, the anterior commissure was proposed to display variation related to sexual preference, being found larger in homosexual than in heterosexual men (Allen and Gorski 1992). Differences in commissural size are difficult to interpret, since they could arise from differences in size of the extracellular matrix, proportion of glial cells, number of axons or axonal diameters (for discussion see,

e.g. Clarke et al. 1989). It is often assumed that the two latter factors play a crucial role and that differences in commissural size reflect differences in connectivity.

Reports on patients with callosotomy that spared the anterior commissure indicate that a certain degree of visual, auditory and olfactory interhemispheric transfer is possible through the anterior commissure (Risse et al. 1978; Trevarthen 1990; Berlucchi et al. 1995). In callosal agenesis, an enlarged anterior commissure was reported in association with normal visual and tactile interhemispheric transfer, whereas an absent anterior commissure was associated with a lack of interhemispheric transfer (Fischer et al.1992). When the anterior commissure had a normal size in cases of callosal agenesis, it appeared to have only a limited capacity for visual interhemispheric transfer (Martin 1985; Karnath et al. 1991).

We investigated the connectivity of the human anterior commissure in cases with circumscribed hemispheric lesions using the Nauta method for anterogradely degenerating axons. Preliminary results were published in abstract form (Di Virgilio and Clarke 1996).

Materials and methods

The contribution of different cortical regions to the anterior commissure was studied in six human brains with relatively circumscribed hemispheric infarctions (Table 1, Fig. 1). All brains were fixed within 24 h after death by immersion in 10% formalin solution. They were examined for presence of lesions macroscopically on about 2-cm-thick slices, and microscopically on histological sections stained for Nissl, myelin or anterogradely degenerating axons. In previous studies (Clarke and Miklossy 1990; Clarke 1994; Di Virgilio and Clarke 1997), this approach allowed the identification of all lesions, even very small ones in the white or grey matter. In the present cases, particular attention was devoted to regions suspected of sending axons to the anterior commissure, such as the inferior and lateral temporal and frontal cortices, as well as the white matter of the temporal and frontal lobes. This detailed examination revealed no other lesions than those listed in Table 1 and those shown in Figs. 1 and 4. Lesions were dated using classical neuropathological stains (haematoxylin and eosin; Prussian blue). The location of lesions was assessed according to the Talairach and Tournoux atlas (1988), which corrects for the relatively large individual differences in size and shape of human brains. Talairach and Tournoux (1988) introduced a proportional grid and a coordinate system anchored in the forebrain commissures. According to this system, three planes are placed within the brain, corresponding to a horizontal plane through the anterior and posterior commissures and two coronal planes through the anterior and posterior commissures, respectively. The hemispheres are subdivided into 12 horizontal, 11 coronal, and 8 sagittal slices. The site and extent of each lesion as apparent on histological sections were determined within the corresponding horizontal, coronal and parasagittal planes of the proportional grid and represented on the standard brain (Fig. 4).

Axons originating in the lesion were detected with the Nauta stain for anterogradely degenerating axons. We used a modification described by Albrecht and Fernstrom (1959), which yielded repeatedly good results for human tissue in previous studies (Clarke and Miklossy 1990; Clarke 1994; Di Virgilio and Clarke 1997). Serial 40-µm-thick coronal sections were cut frozen through all lesions, the region of anterior commissure and other selected regions. As part of other studies (Clarke and Miklossy 1990; Clarke 1994; Di Virgilio and Clarke 1997; and unpublished), both hemispheres were cut into serial sections to their whole extent in case 1, to 70% in case 2, to 50% in cases 3 and 5, and to 40% in cases 4 and 6. Every 400 µm, one or several sections were stained with a modification of the Nauta method for anterogradely degenerating axons (Albrecht and Fernstrom 1959; Fig. 2) and adjacent sections with cresyl violet for cell bodies and for myelin (Schroeder 1939; Loyez 1910 as adapted for frozen sections by Clarke and Nussbaumer 1987).

Numerous technical trials in our laboratory confirmed that the method originally described by Albrech and Fernstrom (1959) is best adapted for human tissue. This method is a selective silver impregnation in which it is possible to suppress normal fibre staining with a passage through a solution of potassium permanganate; the time spent in this solution is roughly proportional to the degree of normal fibre suppression. Briefly, frozen, 40-µm-thick sections are stored in 10% formalin at room temperature and then processed floating as follows (all solutions are aqueous): three rinses in distilled water; 24–48 h in 0.5% oxalic acid and 0.5% hydroquinone; three rinses in distilled water; 10 min in 0.05% chromic acid; three rinses in distilled water; 5 min in 4% hydrobromic acid; three rinses in distilled water; 15 min in 1% phosphotungstic acid; three rinses in distilled water; 0.5–4 min in 0.05% potassium permanganate; 5 min in 0.5% oxalic acid and 0.5% hydroquinone; three rinses in distilled water; 30 min in 1.5% silver nitrate; three rinses in distilled water; 3 min in Laidlaw's ammoniacal silver carbonate solution; 5 min in 0.003% formalin, 0.0003% citric acid and 0.1% ethanol; one rinse in distilled water; 2 min in 1% sodium thiosulphate; and two rinses in distilled water. The sections are then mounted on gelatinized slides, dehydrated in alcohol, cleared in xylol and coverslipped.

The Albrecht and Fernstrom (1959) modification of the Nauta method was developed for human material; it stains reliably de-

Table 1 Cases included in the study

Case	Age at death (years)	Sex	Cause of death	Lesion[a]	Age of lesion	Degenerating fibres in anterior commissure
1	69	M	Pulmonary embolism	R inf. temporal and thalamic	1–3 months[b]	+++
2	74	F	Respiratory failure	L inf. occipital	1–3 months[b]	++
3	78	F	Cardia failure	R inf. occipito- temporal	4 weeks[a]	++
4	70	M	Cardiac failure	L central fissure, R prefrontal and occipital convexity	1–3 months[b]	+
5	92	F	Cardiac failure	R occipital convexity	1–3 months[b]	+
6	69	M	Acute sepsis	L inferopallidal	3–6 weeks[b]	–

[a] Clinically recorded; clinical and histological dating matches
[b] Clinically not recorded; dating based on histological examination

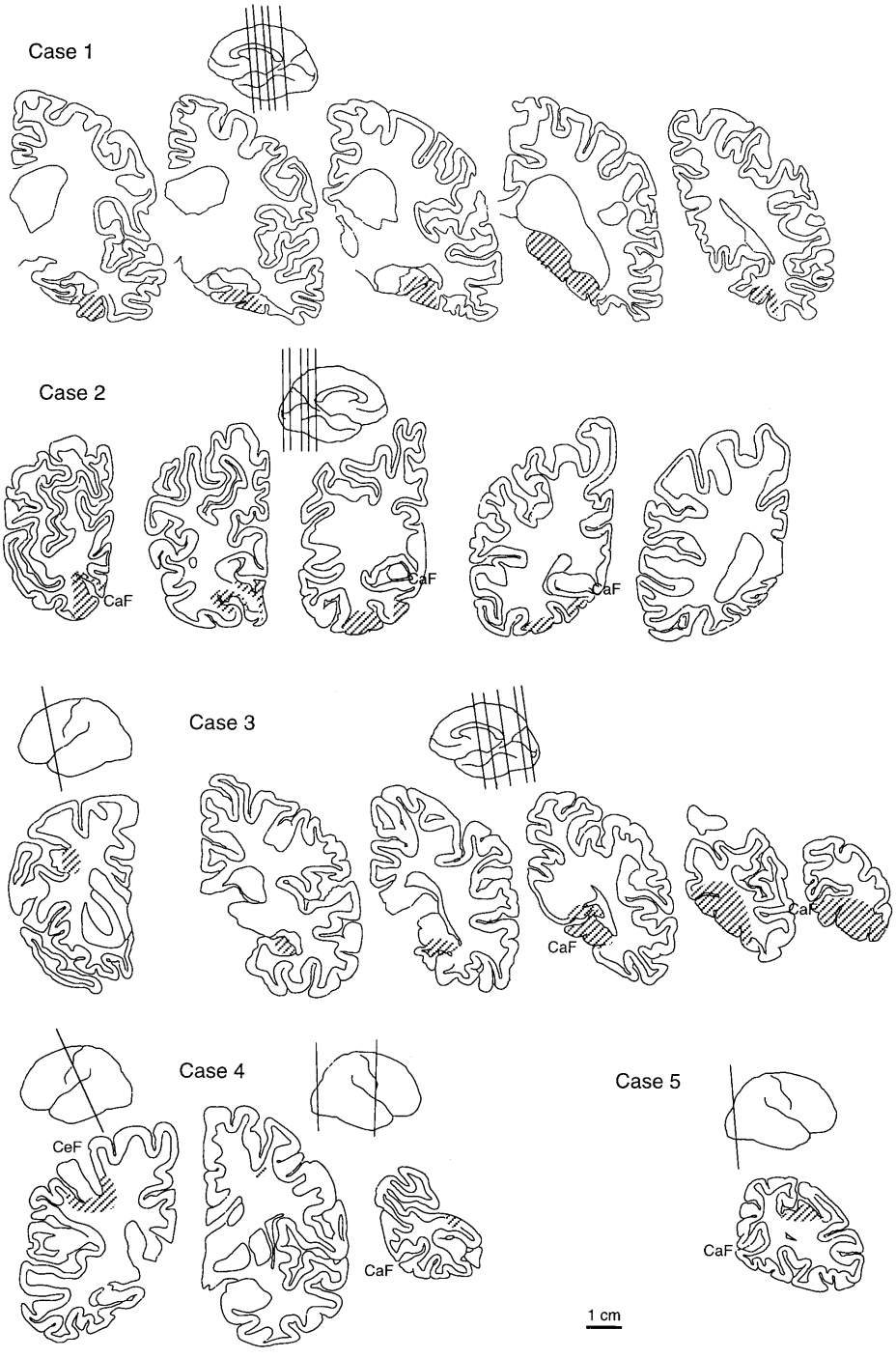

Fig. 1 Lesions of cases 1–5 (*hatched*) in coronal sections (*posterior view*). In cases 1 and 5 the lesion was limited to the right hemisphere, in case 2 to the left hemisphere, and in cases 3 and 4 the lesion was bilateral. Case 1 had an additional right posterior thalamic lesion (not shown here) (*CaF* calcarine fissure, *CeF* central fissure)

generating fibres, but it leaves a few normal fibres stained as well. Degenerating axon segments can be recognized by morphological criteria, which are: (1) irregular contours; (2) great changes in diameter; and (3) interruptions. To avoid false positives, we counted as degenerating only segments that displayed all three criteria (and had at least two interruptions). Note that only axon segments running parallel to the plane of section were likely to be thus detected; hence the counts shown in Fig. 3 underestimate the numbers of degenerating axons.

The distribution of axon segments within the anterior commissure was charted using a ×40 oil immersion objective. Five to ten coronal equally anteroposteriorly spaced sections through the anterior commissure were chosen in each case. In each section, the number of degenerating axon segments within a 300×300-µm counting grid was determined. To avoid double counting, degenerating axon segments were counted when they intersected two of the grid sides (generally the upper side and the side nearest to the midsagittal plane) as well as the included corner, but not when they intersected the other two sides and three corners. For a given section, the grid was placed at the uppermost part of the commissure and then moved downwards by 300 µm for the next count; in this way the whole extent of the commissure was covered.

In addition, two normal brains (from 46- and 52-year-old men respectively) were cut and processed in the same way as the six described above.

Results

Degenerating axons segments were found in the white matter around the lesions and in the surrounding grey matter (e.g. Fig. 2C) in all the cases with lesions, confirming that all six cases were well suited to tracing studies.

Sites of lesions and presence of degenerating axons in the anterior commissure

Anterior commissure was analysed in serial coronal sections in the six brains with lesions and the two without lesions. Degenerating axon segments were found in cases 1–5, but not in case 6 or in the two normal specimens.

Case 1 had a lesion of the anterior part of the fusiform and parahippocampal gyri and the hippocampal formation as well as a right posterior thalamic lesion. The anterior commissure of this case contained the highest densities of degenerating axons. The degenerating fibres were found throughout the anterior commissure, as well as in the posterior and anterior limb on the right side (Figs. 2A,B, 3). Case 2 had a lesion of the posterior part of the left lingual gyrus and the adjacent part of the posterior fusiform gyrus; the anterior commissure of this case contained a relatively high density of degenerating axons. Case 3 had two lesions, a relatively large one in the right lingual and parahippocampal gyri as well as the hippocampal formation and a small second one in the posterior part of the left inferior frontal sulcus. The anterior commissure of case 3 contained a similar density of degenerating axons to that in case 2. Case 4 had three small lesions of the convexity, one located in the right lateral occipital sulcus, the second one in the posterior part of the superior frontal sulcus, and the third one in the left central fissure; the anterior commissure contained degenerating axons, but their density was low. Case 5 had one small lesion in the right superolateral occipital cortex; the anterior commissure contained degenerating axons, but in relatively small numbers.

In case 6, no degenerating axons were found in the anterior commissure. This negative result was not due to failure of staining, since numerous degenerating axons were found around the lesion, including the lower pallidum (Fig. 2C). This case was also interesting from the point of view of judging the effective size of lesions. As assessed with haematoxylin-eosin and Prussian blue stains, the lesion was near, but not encroaching upon, the posterior limb of the anterior commissure. The absence of degenerating axons within the anterior commissure confirmed the reliability of neuropathological lesion assessment.

Topographic arrangement of inferior temporal axons within the anterior commissure

The inferior temporal cortex appeared in the present study to contribute the densest contingent of anterior commissure fibres. We studied the distribution of axons originating in the inferior temporal cortex within the anterior commissure and its posterior limb of case 1 (Fig. 3). In the midsagittal plane, inferior temporal axons were found within the whole extent of the anterior commissure (compare counts A and B in Fig. 3). Higher densities of axons were found in the inferior than in the superior part of the anterior commissure, suggesting an, at least partially, topographic arrangement of cortical fibres in the mediosagittal plane. No distinct topographic arrangement was found in the more lateral parts of the anterior commissure. The posterior limb was analysed at several locations of its trajectory under the lenticular nucleus (Fig. 3, bottom). Degenerating axons were found throughout this posterior limb, with no indication of a particular topographic arrangement.

The majority of the degenerating axons within the anterior commissure and its posterior limb seemed to run in parallel to the bulk of the commissural fibres (Fig. 2A). Occasional fibres, however, were observed to run obliquely in the direction of the other axons (Fig. 2B). The oblique trajectories were reminiscent of those found in cat auditory callosal pathway, which is known to have only a very rough topographic arrangement (Clarke et al. 1995).

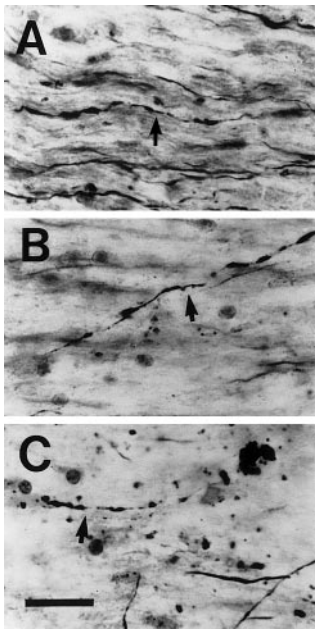

Fig. 2A–C Photomicrographs from Nauta-stained sections. Some of the degenerating axons are indicated by *arrows*. *Bar* 20 µm. **A** Anterior commissure of case 1, left of the mediosagittal plane (i.e. contralateral to the lesion). Degenerating axons were intermingled among and often parallel to normal ones. **B** Anterior commissure of case 1, right of the mediosagittal plane. Occasionally, degenerating axons had an oblique trajectory within the commissural bundle. **C** Lower left pallidum of case 6, near the lesion. Numerous degenerating axons were present, indicating clearly that the lesion caused anterograde degeneration; no degenerating axons were found within the anterior commissure of this case

Heterotopic commissural axons

Most degenerating axons stained after lesions in the inferior temporal and occipital cortex were found within the ipsilateral posterior limb, with only one degenerating fibre within the anterior limb. On the side opposite the lesion, degenerating axons were found in the posterior limb, but also, in cases 1 and 2, in small numbers in the anterior limb. The anterior limb is believed to convey axons from and to the anterior perforated space and possibly the orbitofrontal cortex (Klinger and Gloor 1960); if

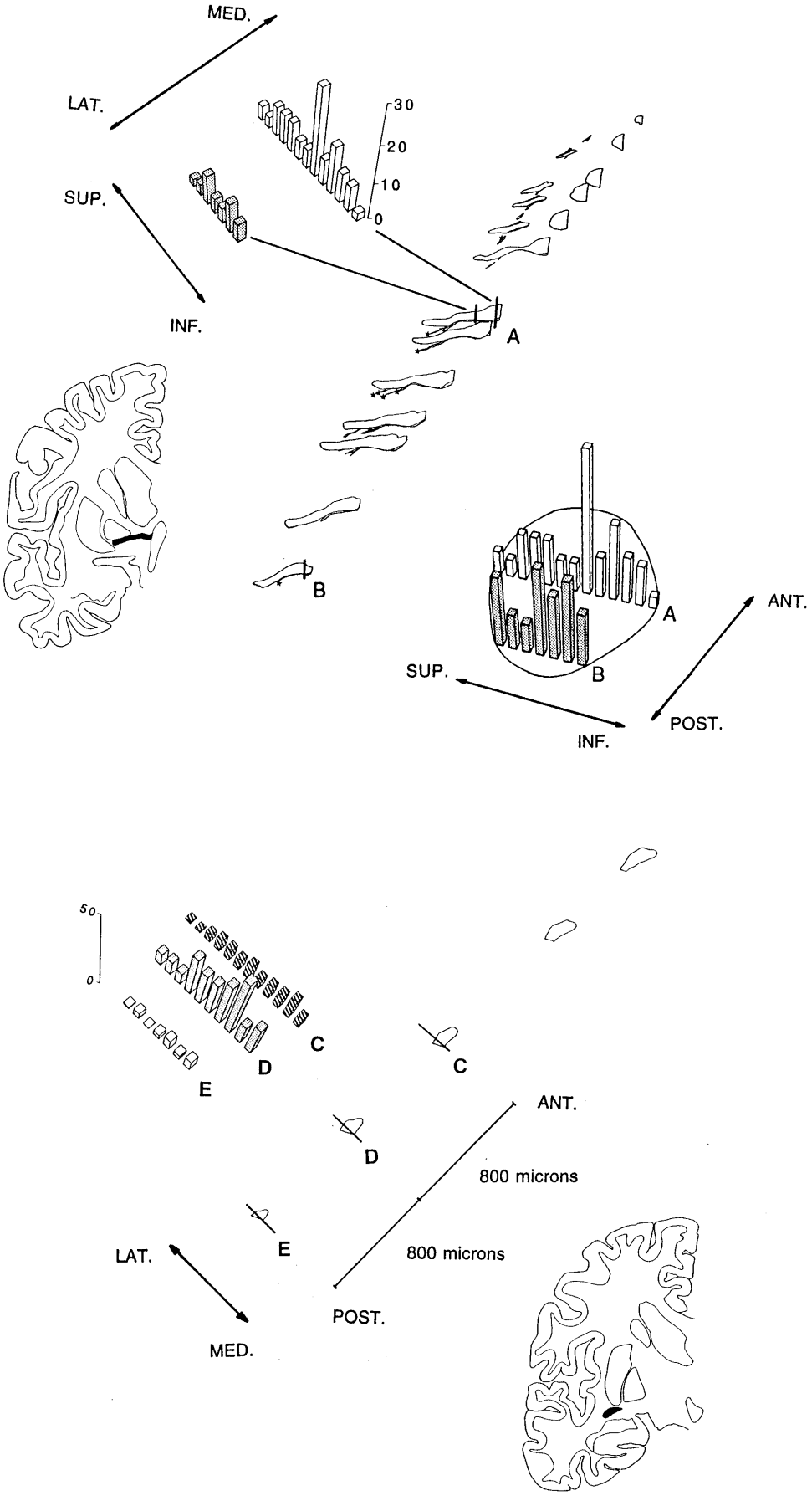

Fig. 3 Distribution of inferior temporal axons within the anterior commissure (*top*) and the posterior commissural limb (*bottom*) in case 1. The outlines of the anterior commissure and the posterior limb are represented in posterior view in serial coronal sections (caudalmost sections at *lower left*). Histograms represent densities found in 40-µm-thick coronal sections *along the indicated lines*; *on the y-axis* are indicated numbers of axons per 300×300-µm counting field. Intermediate sections were analysed in the same way but are not shown here

so, degenerating fibres observed within the anterior limb contralataral to the lesion may represent heterotopic connections, from the inferior temporal and occipital lobes to the anterior perforated space or the orbitofrontal cortex.

In case 1, we analysed one of the putative heterotopic target territories, namely the amygdala complex. Degenerating axons were present within the lateral part of the amygdala contralateral to the lesion, but in very low densities. No clear topographic arrangement was apparent.

Discussion

Regions contributing to the anterior commissure in man

Our results show that the inferior temporal cortex, inferior occipital cortex and locations on the upper part of the hemispheric convexity contribute axons to the anterior commissure. Such a widespread origin for the anterior commissure was not expected from work on non-human primates.

This unexpectedly wide origin for anterior commissural axons is a genuine characteristic of the human brain and is not due to artefacts of the technique. There are several reasons to exclude the latter. First, the Nauta method for anterogradely degenerating axons has been used successfully in several studies of human connectivity (Clarke and Miklossy 1990; Clarke 1994; Di Virgilio and Clarke 1997; Clarke et al. 1998). Second, the presence of other lesions than those listed in Table 1 and shown in Figs. 1 and 4 was excluded by minute examination of the brains. Third, cases without brain lesions showed no degenerating axons in the anterior commissure. Fourth, the location of lesions was critical; in case 6, with an inferopallidal lesion outside the posterior limb of the anterior commissure and signs of heavy anterograde degeneration in the white and grey matter around the lesion, no degenerating fibres were found in the anterior commissure.

As typical of infarctions, lesions in the present cases were not strictly limited to the cortical grey matter. A few millimetres (case 5) to about 1.5 cm (case 3) of underlying white matter were involved in lesions. The Nauta method also stains degenerating axons following a proximal interruption, and some of the degenerating axons observed in the anterior commissure could have been interrupted by the lesion, while their somata lay outside the lesion. Comparison of site of lesion, the degree of white matter involvement, and presumed axonal trajectories allows the evaluation of this problem. In case 1, axons that degenerated may have originated not only in the inferior temporal, but also partially in the infero-anterior occipital cortex. In case 2, the additional territory may include the occipital pole; in case 3 cortex next to the lesion; in case 4 possibly the medial portion of the precentral gyrus; and in case 5 cortex next to the lesion. Although the regions of origin of commissural fibres in the individual cases that we studied may be slightly larger than indicated by the extent of the lesions, the general conclusion appears to hold, namely that regions outside the inferior temporal and orbitofrontal cortex send axons to the anterior commissure.

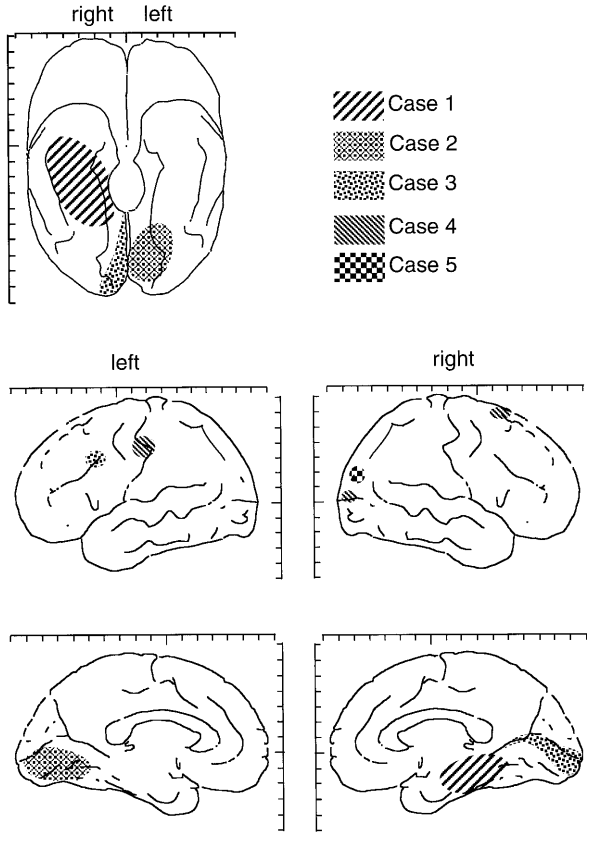

Fig. 4 Locations of lesions in cases with anterogradely degenerating axons within the anterior commissure, assessed by Talairach and Tournoux (1988) coordinates and thus allowing interindividual comparisons

Tract tracing studies in macaque monkeys showed that the anterior commissure receives fibres from the temporal lobe (temporal pole, superior and inferior temporal gyri and parahippocampal gyrus), the orbitofrontal cortex, the prepiriform cortex, and the amygdala. The heaviest projection derives from the rostral third of the temporal isocortex, with a less dense projection from the middle part and no projection from the caudal part of the temporal isocortex (Pandya et al. 1969; Zeki 1973; Cipolloni and Pandya 1985; Demeter et al. 1990). The macaque occipital cortex does not send axons to the anterior commissure (Rockland and Pandya 1986).

The origin of the human anterior commissure presents a more complex picture (Fig. 4). As in macaque monkeys, the human anterior commissure receives a substantial projection from the inferior temporal cortex. However, unlike in monkeys, it also receives a projection from the occipital cortex, both from the inferior part (case 2) and the convexity (case 5). Presently, we cannot affirm that the anterior commissure receives afferents from the central fissure and prefrontal cortex, since in both these cases additional lesions were present in regions known to project to the anterior commissure (cases 3 and 4). To facilitate comparison with other anatomical, activation or

neuropsychological studies, Fig. 4 indicates the Talairach coordinates of lesions that were associated with degenerating axons in the anterior commissure.

Topography within the anterior commissure

Studies using the Nauta method (Pandya et al. 1969) or radioactive amino acids (Cipolloni and Pandya 1985; Demeter et al. 1990) suggested a topographic arrangement of axons within the macaque anterior commissure; axons originating from the anterior parts of the supratemporal plane and the superior temporal gyrus were located in the ventral portion of the anterior commissure when crossing the midsagittal plane. The topographic arrangement within the human anterior commissure seems to be less clear. Axons from the inferotemporal cortex were found within the whole commissure, but they were more numerous in its inferior part. The mixing of commissural axons of different origin occurs at least partially before the axons join the posterior limb; we have found no specific inferotemporal compartments at any location of the posterior limb (representative counts are shown in Fig. 3). There are, however, signs of fibre mixing also in the midsagittal plane, as shown by the presence of obliquely running fibres (Fig. 2B).

Heterotopic connections from the inferotemporal cortex to the amygdala

Heterotopic interhemispheric connections appear to be a distinctive feature of the human cerebral cortex (Clarke 1998). For example, a monosynaptic interhemispheric projection links the right inferior temporal cortex to Wernicke's and Broca's areas (Di Virgilio and Clarke 1997). Here we found indications that the inferior temporal cortex also sends heterotopic interhemispheric connections to other locations. Small numbers of degenerating axons were found in the amygdala contralateral to the lesion. Furthermore, small numbers of degenerating axons travelled, after crossing the midsagittal plane, in the anterior limb, which is believed to convey fibres to territories others than the temporal cortex.

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