Age and hemisphere effects on dendritic structure

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Summary
The dendritic structures of 187 small supragranular pyramidal neurons of the posterior superior temporal gyrus were studied with rapid Golgi impregnations in post-mortem samples from 10 men aged 21–71 years. The number of primary basilar dendritic branches, the total number of basilar dendritic endings, the total basilar dendritic length, the total number of visible basilar dendritic spines and the cell soma sizes were all positively inter-correlated and all features were correlated with age \( r = -0.77, -0.88, -0.82, -0.72, -0.86 \), respectively; all \( P < 0.05 \). These neuronal measures all correlated with brain weight \( r = 0.79*, 0.65*, 0.51, 0.45, 0.55 \), respectively; *denotes \( P < 0.05 \). A first principal component derived from the inter-correlations of the neuronal features plus brain weight correlated almost perfectly with age \( r = -0.93 \). The neuronal features differed between the right and left hemispheres (Wilks’ Lambda = 0.91, \( P < 0.01 \)). Post hoc tests showed that the dendritic trees of the right hemisphere were longer \( (P = 0.002) \), more branched \( (P = 0.008) \) and possessed more dendritic spines \( (P = 0.0009; \text{Sheffe’s tests}) \). In conclusion, there are hemispheric differences in the dendritic structure of the small pyramidal neurons of presumptive human speech cortex and its right hemisphere analogue. Generalized neuronal atrophy is highly correlated with both brain weight and age, and is a candidate process to explain the decline in cognition with age.

Keywords: ageing; cerebral lateralization; dendrites; cerebral cortex; language

Introduction
This study examined the basilar dendritic architecture of the small supragranular pyramidal neurons of the posterior superior temporal gyrus in 10 men aged 21–71 years. From these data we were able to address two questions. (i) How does the dendritic architecture of this neuronal population change with age? (ii) Are there hemispheric differences in dendrite structure between presumptive human language cortex and its contralateral analogue?

Age, cognition and dendrites
The motivation for examining the relationship between dendritic architecture and age is to understand the physical bases for the decline in cognitive capacities that occurs with ageing. This decline has been repeatedly documented in numerous studies and these studies have been reviewed (e.g. Albert and Moss, 1988; Zec, 1995). Briefly, decline in cognition with age often start from the fourth decade onwards and affects numerous domains; memory decline has been the most consistently demonstrated (Perlmutter et al., 1987; Bamford and Caine, 1988; Light and Albetson, 1989). General intelligence also declines with age (Cunningham, 1987). Intelligence has been shown to decline with age, even when tested longitudinally, thus eliminating an explanation based solely on cohort effects (Schae and Strother, 1968; Schae and Hertzog, 1983); while some changes in IQ began to appear in the subjects' fifties, most significant changes did not appear until the subjects' sixties.

Reasoning ability, which is related to intelligence, has also been shown to decline with ageing. Inter-individual variability in reasoning ability has been argued to be a result of variation in working memory ability (Kyllonen and Christal, 1990). However, in ageing the decline in reasoning ability cannot be explained by changes in working memory ability or vocabulary ability alone (Gilinsky and Judd, 1994). The central executive component of working memory also declines with age, as assessed by dual task performance (Crossley and Hiscock, 1992). In this task subjects were asked to perform finger tapping and reading, and the older subjects showed a much greater decrement in performance than young subjects when trying to do the tasks concurrently. On the Stroop interference task, aged subjects do not show declines in colour naming or word reading alone but do

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show a gradually increasing interference effect with increasing age (Houx et al., 1993).

Language task performance may also decline with age. Performance on picture-naming tasks decreases with age but some of this change is secondary to medication and illness effects (Goulet et al., 1994). There is also a decline in access to semantic material that occurs with age. There is a relatively poor use of context for sentence comprehension (Wingfield et al., 1994) and a greater difficulty in understanding sentences as the presentation rate is increased (Wingfield et al., 1992).

While a general, but not uniform, decline in cognitive capacity with age has been repeatedly demonstrated, the physical basis for the decline has not been demonstrated. The evidence on cerebral cortical neuron number and age is mixed. Some studies, based on measures of neuronal density estimates derived from profile counts, suggest a decrease in cerebral cortical neuron number with age (Henderson et al., 1980; Coleman and Flood, 1987). Other studies argue that while neurons shrink they do not decline numerically (Terry et al., 1987). Stereological estimates, which are less subject to biases than profile counts, have suggested both a decline in neurons with age (Pakkenberg et al., 1989) and no change with age (Haug, 1987). In summary, the data on cerebral cortical neuron number changes with age are conflicting, but it does not appear that a decrease in cerebral cortical neuron number is a sufficient explanation for the cognitive changes that occur with ageing.

A few studies have examined the effect of age on the dendritic architecture of cerebral cortical neurons. The Scheibels, based on qualitative studies, suggested that there was an age-related decline in dendritic extent (Scheibel and Scheibel, 1975; Scheibel et al., 1976, 1977; Scheibel, 1979a, b). Buell and Coleman (1981) reported a growth of the dendrites with age in layer II parahippocampal neurons. The results of Coleman and Flood (1987), from frontal isocortex, suggested an age-related decline in dendritic extent. Nakamura et al. (1985), looking at neurons of the frontal motor cortex, also reported an age-related decline.

The most comprehensive study of the effect of age on cerebral cortex pyramidal neurons is that of Jacobs and Scheibel (1993). They studied the supragranular pyramidal neurons of Wernicke’s area (a cerebral cortical region relevant for language comprehension) in 20 subjects ranging in age from 18 to 79 years. They found an age-related decrease in total dendritic length ($r = -0.44$) and in mean dendritic length ($r = -0.69$). Mean dendritic length refers to the length of individual dendritic segments (a segment of a dendrite is a portion of a dendrite that is demarcated by branch points or a branch point and either the cell soma or the end of the dendrite). Dendritic spines were not analysed. No study has reported a quantitative analysis of the change in dendritic spine number with normal ageing.

It is probable that an analysis of dendritic spine counts would show age-related declines, as studies estimating synaptic density have shown an age-related decline and dendritic spines are the site of most synaptic contacts. Masliah et al. (1993) assessed synaptic density in the frontal cortex of 25 individuals with an age range of 16–98 years. A significant inverse relationship was found between age and synaptic density ($r = -0.7$). This study estimated synaptic density from synaptophysin immunocytochemistry. Other studies, using other techniques, generally show a decline of synaptic density with age, although there is some regional heterogeneity (Huttenlocher, 1979; Gibson, 1983; Adams, 1987; Bertoni-Freddari et al., 1989).

Thus, while cognitive capabilities are known to decline with age, the neural basis for the decline remains undetermined. A decrease in cortical neuron number seems an incomplete explanation. Qualitative and quantitative studies of dendritic architecture and synapse density estimates imply an age-related decline. Only one study has examined these changes in human posterior superior temporal cortex, and no study has simultaneously characterized the dendritic structure and dendritic spine number on neurons in this region.

**Hemispheric specialization and dendrites**

The motivation behind looking for differences in the dendritic architecture of the two cerebral hemispheres is to understand the physical basis for hemispheric specialization better. It is well recognized that areas of the cerebral cortex are functionally specific. In many of these cases the functional specificity can be partially understood as deriving from input-output connectivity, e.g. the occipital cortex receives relatively direct projections from the eyes, but not the ears.

There are cerebral cortical areas whose functional definition cannot be deduced from their anatomical connectivity; this is especially true in the circumstance where one hemisphere is functionally dominant. The most dramatic example of lateralized functional specialization is language. For most individuals, regardless of handedness, the left hemisphere is dominant for the semantic aspects of language, with posterior areas generally relevant for language comprehension and anterior regions for language expression (Caplan, 1992).

How is it that the left posterior temporal region should be relevant for semantic comprehension while the same region of the right hemisphere is not, even though both regions seem to receive similar input projections and send their output to similar areas?

One possibility is that functional competence for language is simply a quantitative matter, and the left hemisphere speech area is larger (Geschwind and Levitsky, 1968; Witelson and Kigar, 1992; Foundas et al., 1995). However, since the right temporal lobe of some tall men may be greater in size than the left temporal region of small women, a mass action basis for left hemisphere language dominance appears unsatisfactory as a complete explanation.

A second hypothesis for left hemisphere language specialization is that the input–output connections of the two posterior temporal regions, left and right, are different. Unfortunately, the fine anatomical tract tracing studies that can be performed in animals cannot currently be performed.
for ethical reasons, in human subjects. Thus, while this hypothesis has no positive support, the existing evidence is inadequate to disprove it.

A third possible explanation for left hemispheric language dominance is that there are hemispheric differences in the intrinsic circuitry of the language dominant posterior—superior temporal cortex. In investigations of the cytoarchitectonic zones of the posterior temporal lobe no cytoarchitectonic field unique to the left hemisphere has been reported (Galaburda and Sanides, 1980; Seldon, 1981a).

Is it possible that proportions of different neuronal subtypes might vary between the same region of the two hemispheres and that this could have functional consequences? Rosen et al. (1993) looked at the numbers and percentages of parvalbumin and vasoactive intestinal peptide positive neurons in asymmetrical regions of rat cortex and found qualitative differences between regions, not just quantitative ones. No corresponding evidence is available for laterality effects in human posterior temporal cortex.

In a subtler vein, the local connectivity of temporal lobe neurons in the two hemispheres might be different. Seldon performed extensive studies on the auditory cortices of a small number of older human subjects (Seldon, 1981a, b, 1982, 1985). In his analysis he analysed the distance between neuronal columns and compared this distance with various measures of tangential dendritic extent. Seldon reported a greater ‘tangential’ radius for pyramidal neurons of the left hemisphere but this was smaller, in relation to the inter-columnar distance, than it was for the right hemisphere; this allowed him to conclude that the neurons of the left hemisphere were less entangled than neurons of the right.

Jacobs et al. (1993) performed a study on the dendritic architecture of Wernicke’s area in the left and right hemisphere. They evaluated the basilar dendrites of the small supragranular pyramidal neurons. Major measures were the total dendritic length, dendritic segment count and the mean dendritic segment length. The left hemisphere values were greater than those in the right for all measures when data from all subjects were grouped. At the individual level only twelve out of twenty subjects showed a significant left to right advantage for total dendritic length and 13 out of 20 for dendritic segment counts.

In summary, the basis for functional specification of cerebral cortex can, for primary sensory and motor cortices, be explained on input and output connections. However, for association cortex this explanation becomes increasingly incomplete. By the time we consider cortex which is dominant for language function, this explanation is inadequate. Although limited, there is more information on the neuroanatomical features of language-specialized cortex than other lateralized, functionally specialized, cortical regions. So far no consistent differences, other than volume, have been found between language competent and analogous language incompetent areas of the left and right hemisphere. There are indications of lateralizing differences in the dendritic architecture of the neurons of language cortex.

**Methods**

**Subjects**

All 10 subjects were men and they ranged in age from 21 to 71 years. Brain tissue was taken at the time of routine clinical post-mortem examination by collaborating pathologists at the Walter Reed Army Medical Center (Washington, DC) and the Wilford Hall Medical Center (Lackland AFB, Tex., USA). In none of the subjects was there a known history of a primary neurological illness. One subject had died instantly of a self-inflicted gunshot wound to the head, remote from the posterior temporal lobes. Clinical data on the subjects were limited and did not permit firm conclusions regarding the subjects’ handedness or multilingual status. Although all subjects had undergone testing with various versions of the Armed Services Vocational Aptitude Battery (ASVAB; Anonymous, 1984), the archives could only be accessed for the four youngest subjects. This precluded efforts to correlate dendritic measures with life experiences or cognitive ability.

Gross inspection and routine histopathological studies showed no evidence of tissue injury in the posterior temporal cortex of any of the subjects. A modified silver stain (Davenport stain) was performed on celloidin-embedded sections taken from blocks adjacent to those used for the Golgi studies (Clark, 1973). Neurofibrillary tangles and senile plaques were not noted for any of the specimens. Post-mortem intervals ranged from <1 h to 24 h. Removal of the brain area for analysis was based on standard instructions. The pathologists performing the autopsy removed, bilaterally, a 1 cm block of the superior temporal gyrus, immediately posterior to Heschl’s transverse gyrus. The tissue block was taken perpendicular to the long axis of the temporal lobe. It was wrapped in gauze, placed in 10% neutral buffered formalin and shipped to the authors.

**Histological processing**

The brain tissue remained in 10% neutral buffered formalin for several weeks prior to being processed for rapid Golgi impregnation. Processing consisted of removing the blocks from formalin and cutting an ~4 mm thick portion that was oriented so as to give a good perpendicular section through the cerebral cortex. Next, the tissue was suspended by a thread in a freshly made solution of 0.25% osmium tetroxide/3.5% potassium dichromate for 6 days.

After a brief rinsing in old 0.75% silver nitrate the tissue was suspended in a fresh solution of 0.75% silver nitrate for a period of 24–48 h. After the silver bath, 120 μm sections were cut on a sliding microtome using the paraffin shell technique (Millhouse, 1981). Sections were collected in ethanol, passed through α-terpineol and toluene and mounted under a coverslip with Histomount (National Diagnostics, Atlanta, Ga., USA).

**Cell selection and tracing**

The goal was to locate 10 neurons for each hemisphere of each subject. In two cases this was not possible because
of the poor quality of the impregnation. Slides were coded so that the subject’s age was not known at the time of cell selection.

Cells were selected for tracing if they were small pyramidal cells of the supragranular layers. Specific efforts were made to avoid the very small pyramids near the layer I–II border and the larger pyramids of deep layer III. The neurons had to be well-impregnated, not obscured by adjacent cells or debris and possessing at least three primary basilar dendrites. The apical dendritic dendrite only had to be present to a length sufficient to allow identification of the cell as a pyramidal neuron.

Cells were only selected if they gave the qualitative appearance of a full basal dendritic arbour. In general, the first ten cells meeting these criteria were selected. For some brains all available sections had to be searched, for others ten well-impregnated cells with full arbours could be found on just two sections. Most cells had dendritic trees large enough that there were at least some cut dendrites distally. No effort was made to try and locate these cut tips in adjacent sections.

Cells meeting the selection criteria had their positions recorded and were then traced using a Neurolucida Tracing System (MicroBrightField, Colchester, Vt., US) interfaced with a Nikon Optiphot microscope. Tracing was performed at a magnification of X400. The cell soma was outlined and then the dendritic tree traced. Although the portions of the axon and apical dendrite present were traced, these cellular components were too fragmentary to be included in the quantitative analyses. As the dendritic tree was being traced the locations of all visible dendritic spines, or portions thereof, were marked, but no delineation based on spine size or shape was made. Thus spines projecting more directly towards or away from the examiner, and not visually separable from the dendrite, could not be counted; therefore, the spine count is an underestimation of the true number of dendritic spines.

This tracing system allows for the computation of a number of quantitative measures. We evaluated five parameters reflecting the extent and complexity of the basal dendrites, namely: cell soma size; the number of primary basal dendrites; the total number of dendritic endings (a measure of branching), the total dendritic length and the number of visible dendritic spines. Primary basal dendrites were defined as those which left the cell body directly, excluding the apical dendrite.

Statistics
Statistical analyses were made using the Statistica computer program (StatSoft, Tulsa, Okla., US). Analyses included multivariate analyses of variance, principle components analyses and descriptive statistics.

Results
A total of 187 neurons were traced. The general statistics for all the neurons together are shown in Table 1. In all, $-0.5 \text{m}$ of basilar dendrites were traced. None of the measures showed any significant correlation with post-mortem delay ($r$ ranged from $-0.04$ to $+0.34$). Precise timing of the duration of the agonal period was not possible from a retrospective review of the charts. However, in three cases it was known that death was near instantaneous (two gunshot wounds and one exsanguination after surgery), whereas for the other seven there was probably at least some agonal interval (sepsis and cancer were the causes of death). When these two groups were compared on the anatomical measures, and after controlling for age, there were no significant differences between them, suggesting that the agonal period before death did not affect the results.

All the primary variables were inter-correlated with each other and with brain weight. The inter-correlations are shown in Table 2. A principle components analysis extracted a single general factor that explained 74% of the shared variance (eigenvalue 4.46; factor loadings’ range 0.75–0.98).

Ageing
All of the primary variables were negatively correlated with age (see Table 2 and Fig. 1). For computing these correlations the mean value of each measure for each subject was used. In addition, factor scores for each case were computed using factor score coefficients from the principle components analysis. This ‘neuronal size’ factor was inversely correlated with age ($r = -0.93$).

Hemisphere
The statistics for the dendritic measures, subdivided by hemisphere, are shown in Table 1. A one way MANOVA was conducted with hemisphere as the independent variable and the five measured parameters (cell soma size; the number of primary basal dendrites; the total number of dendritic endings/branches, the total dendritic length and the number of visible dendritic spines) as the dependent variables. This analysis showed a significant hemisphere effect (Wilks’ Lambda = 0.91; $P = 0.003$). In post hoc Sheffe testing there were no significant hemispheric effects for soma size or the number of primary basal dendrites but the right hemisphere contained longer dendrites ($P = 0.002$), more dendritic branch endings ($P = 0.008$) and more dendritic spines ($P = 0.0009$). For the nine subjects who had neurons traced in both hemispheres these features favoured the right hemisphere in seven.

Discussion
In this study we examined the basilar dendritic architecture of 187 small pyramidal neurons in 10 men aged 21–71 years. The neurons sampled came from the supragranular layers of the superior temporal gyrus posterior to Heschl’s gyrus. In the left hemisphere this zone would fall in Wernicke’s area, a cerebral cortical region relevant for language comprehension.
Table 1  Descriptive statistics for total neuron population

<table>
<thead>
<tr>
<th></th>
<th>Both hemispheres (mean±SD)</th>
<th>Left hemisphere (mean±SD)</th>
<th>Right hemisphere (mean±SD)</th>
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<tr>
<td>Primary basal dendrites</td>
<td>4.5±1.1</td>
<td>4.38±1.1</td>
<td>4.66±1.1</td>
</tr>
<tr>
<td>Basal dendritic length (μm)</td>
<td>2488.9±1670.8</td>
<td>2238.5±1135.2</td>
<td>2758.8±1154.5</td>
</tr>
<tr>
<td>Endings</td>
<td>24.9±7.1</td>
<td>23.5±7.2</td>
<td>26.3±6.9</td>
</tr>
<tr>
<td>Spines</td>
<td>606.6±3508</td>
<td>525.6±338.8</td>
<td>693.9±344.2</td>
</tr>
<tr>
<td>Soma size (μm²)</td>
<td>226.4±70.2</td>
<td>229.3±72.2</td>
<td>223.2±68.4</td>
</tr>
<tr>
<td>Brain weight (g)</td>
<td>1304±143</td>
<td>1304±143</td>
<td>1304±143</td>
</tr>
</tbody>
</table>

Table 2  Inter-correlations of neuronal measures and correlation with age

<table>
<thead>
<tr>
<th></th>
<th>Primary b.d.</th>
<th>Endings</th>
<th>Length</th>
<th>Spines</th>
<th>Soma</th>
<th>Brain weight</th>
</tr>
</thead>
<tbody>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Endings</td>
<td>0.74</td>
<td>0.92</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Length</td>
<td>0.60</td>
<td>0.84</td>
<td>0.97</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Spines</td>
<td>0.57</td>
<td>0.86</td>
<td>0.74</td>
<td>0.57</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Soma</td>
<td>0.53</td>
<td>0.65</td>
<td>0.51</td>
<td>0.45</td>
<td>0.55</td>
<td></td>
</tr>
<tr>
<td>Brain weight</td>
<td>-0.77*</td>
<td>-0.88*</td>
<td>-0.82*</td>
<td>-0.72*</td>
<td>-0.86*</td>
<td>-0.79*</td>
</tr>
</tbody>
</table>

b.d. = basal dendrites; *P < 0.05

Fig. 1  The relationships between age (years) and dendritic length (left ordinate), and age and dendritic spine number (right ordinate). The mean values for each subject (both hemispheres combined) are plotted with the two regression lines. All the other neuronal measures showed similar relationships to age.

The motivation for the investigation was to examine the relationship between dendritic architecture and both age and hemispheric localization.

Technical considerations

The rapid Golgi techniques are mercurial but have the advantage of being able to be employed on aldehyde fixed tissue, despite prolonged periods of fixation. Changes in dendritic architecture can occur with post-mortem delay but these changes are recognizable (Williams et al., 1978; Buell, 1982). In the present study neurons were only selected for tracing if the impregnation characteristics were good, even though this resulted in an incomplete collection in one subject and the abandonment of collecting neurons from the right hemisphere of another subject. There was no relationship between any of the anatomical measures and the post-mortem interval.

Whether the rapid Golgi technique impregnates a random population of neurons has never been conclusively
established. Where estimates have been made, they suggest that the cell population impregnated by the Golgi techniques is random (Smit and Colon, 1969; Shimon and Tsuji, 1987), although impregnated cells may underestimate dendritic extent. These concerns are not germane to the present investigation since all subjects were submitted to the same technique. Other techniques, such as post-mortem intracellular tracer injection, are available for studying dendritic architecture but they do not offer the rapid Golgi technique’s ease, efficiency, economy or flexibility (Einstein, 1988). These other techniques are also susceptible to post-mortem changes. Thus, while not without its challenges, the Golgi technique appears to provide a random selection of neurons. With the application of standard selection criteria, the risk of selecting neurons showing autolytic changes can be reduced. In the present study there is no indication that the technique used for delineating neuronal structure would have introduced any systematic bias between subjects.

Ageing
Our result that dendrites atrophy with age is consistent with prior investigations (Scheibel et al., 1976; Nakamura et al., 1985; Jacobs and Scheibel, 1993). The decline in the number of dendritic synapses suggests that there is a loss of synapses with ageing too. This finding agrees with a prior report (Huttenlocher, 1979; Gibson, 1983; Adams, 1987; Bertoni-Freddari et al., 1989; Masliah et al., 1993). To our knowledge, this is the first study where synapses were quantified simultaneously with dendritic length, as a measure of excitatory synapse number to show that both measures vary together with age.

Is the dendritic atrophy a linear process beginning at the age of 20 years? While the linear regression is highly significant, visual inspection of Fig. 1 suggests that the dendritic measures may remain stable until ~50 years of age, and then start to decline. This would be consistent with other data suggesting that cerebral atrophy begins around the start of the sixth decade (Miller et al., 1980) and that fluid intelligence begins its decline simultaneously (Schaie and Hertzog, 1983). However, it should be noted that others have not noted this pattern of change in dendrites with age (Jacobs and Scheibel, 1993) or cerebral atrophy (Ho et al., 1980). Further studies on additional subjects will be necessary to evaluate this issue conclusively.

It is known that in Alzheimer’s disease cerebral cortical tissue synapses and dendritic spines are lost and that these measures provide some of the strongest correlations with dementia severity (Dekosky and Scheff, 1990; Terry et al., 1991; Anderson, 1995). Therefore, it is tempting to suggest that a synapse-/dendritic spine-loss might also provide the physical substrate for a cognitive decline with normal ageing, and that Alzheimer’s disease might be merely an exaggeration of the normal changes which occur with ageing. While such a circumstance may be true there are significant differences between the cerebral pathology of Alzheimer’s disease and normal ageing. While the number of dendritic spines per neuron decreases in both ageing and Alzheimer’s disease, this spine-loss occurs without a decrease in dendritic length in Alzheimer’s disease (Anderson, 1995), whereas the spine loss parallels dendritic shortening in normal ageing. This pathological difference would imply that the pathogenesis of spine loss in normal ageing is different from that which occurs in Alzheimer’s disease.

Even if dendritic spine-loss were the ultimate explanation for the cognitive changes of ageing, the high degree of inter-correlation among all the cellular and dendritic measures provokes a chicken and egg puzzle. Does the disappearance of synapses lead to decreased neuronal activity and consequently a generalized neuronal atrophy, or is there some sort of neuronal fatigue that leads to the loss of a neuron’s ability to keep up a complex dendritic arbor with, as a consequence, a secondary loss of spines and synapses? Intermediate hypotheses are also possible, and the correlative nature of the present results does not allow for the evaluation of these competing hypotheses.

Hemisphere
In this study we found a difference between the left and right hemispheres in the dendritic architecture of supragranular small pyramidal cells of the posterior superior temporal gyrus. Seldon’s published data (Seldon, 1981a, 1982) do not allow conversion to the measures used in the present study, therefore, a direct comparison is not possible.

Jacobs and Scheibel (1993) used similar technical procedures and measured some of the same variables. The mean number of primary basal dendrite branches (4.0 in men) and the total dendritic length (2263 μm in men) in the Jacobs and Scheibel study are similar to those of the present report (Table 1), suggesting that a similar neuronal population was analysed in both studies. It is of note therefore that important differences were found in the results. While both investigations measured total dendritic length, the present study found total dendritic length greatest on the right in seven out of nine subjects with significant differences determined from a multivariate analysis. This same measure was greatest on the right in only four out of ten of the male subjects of Jacobs and Scheibel (1993).

A significant drawback of this study and all studies of ‘language’ cortex defined by anatomical landmarks is that such cortex may not actually be ‘language’ cortex. Functional mapping studies, such as those using intracranial electrical stimulation (Ojemann et al., 1989), have revealed significant variation in the location of ‘language’ cortex. A case in point is another Golgi-based study by Ojemann et al. (1989) concerning dendritic studies in a person who had undergone electrical brain activity mapping. In this subject, the anatomically defined Wernicke’s area did not produce naming errors during electrical stimulation whereas a slightly distant temporal area did. To advance in our efforts to define the structurally unique characteristics of cerebral cortex
specialized for language clearly and confidently, it would be preferable to combine anatomical techniques with some method of functional localization.

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