Auditory hallucinations are a frequent symptom in schizophrenia. While functional imaging studies have suggested the association of certain patterns of brain activity with sub-syndromes or single symptoms (e.g., positive symptoms such as hallucinations), there has been only limited evidence from structural imaging or post-mortem studies. In this study, we investigated the relation of local brain structural deficits to severity of auditory hallucinations, particularly in perisylvian areas previously reported to be involved in auditory hallucinations. In order to overcome certain limitations of conventional volumetric methods, we used deformation-based morphometry (DBM), a novel automated whole-brain morphometric technique, to assess local gray and white matter deficits in structural magnetic resonance images of 85 schizophrenia patients. We found severity of auditory hallucinations to be significantly correlated \( (P < 0.001) \) with volume loss in the left transverse temporal gyrus of Heschl (primary auditory cortex) and left (inferior) supramarginal gyrus, as well as middle/inferior right prefrontal gyri. This demonstrates a pattern of distributed structural abnormalities specific for auditory hallucinations and suggests hallucination-specific alterations in areas of a frontotemporal network for processing auditory information and language.

Keywords: auditory, cortex, hallucination, morphometry, prefrontal, schizophrenia

Introduction

Schizophrenia is a complex and heterogeneous psychotic disorder with a wide range of symptoms including delusions, hallucinations, formal thought disorder, altered affect and cognitive functioning. Previous neuroimaging studies have suggested certain patterns of brain activity to be associated with sub-syndromes (Liddle et al., 1992; Schröder et al., 1996) or single symptoms. Positive symptoms such as auditory hallucinations have been associated with superior temporal cortical dysfunction (e.g., Dierks et al., 1999; Lennox et al., 2000). However, it has been difficult to establish structural correlates of these functional findings (Weiss and Heckers, 1999).

Auditory hallucinations are probably one of the most frequent and most challenging symptoms in schizophrenia (David and Busatto, 1998). During the course of illness about two-thirds of patients will experience this symptom (David, 1994). Most often these are auditory–verbal hallucinations (i.e., ‘hearing voices’, often conversing or commenting on the patient), but might include simple noise, sounds or music as well. While much research has focused on neuropsychological approaches (David, 1994), we still know little about the underlying neurobiology and the mechanisms leading to the occurrence of this symptom. The functional neuroanatomy of auditory–verbal hallucinations has recently been addressed in functional neuroimaging studies. These have revealed transient brain activation accompanying auditory hallucinations (Silbersweig et al., 1995; Weiss and Heckers, 1999). Activated areas primarily included language-related areas such as superior temporal cortical regions (Lennox et al., 2000), including the primary auditory cortex in one study (Dierks et al., 1999), Broca’s area (McGuire et al., 1993), as well as the basal ganglia and anterior cingulate (Silbersweig et al., 1995). Electrophysiological studies have also linked the superior temporal (Ishii et al., 2000) or temporal/parietal region (Line et al., 1998) to auditory hallucinations in schizophrenia.

While many magnetic resonance imaging (MRI) studies have shown volumetric changes in cortical and subcortical areas in the schizophrenic brain (Wright et al., 2000; Shenton et al., 2001), there are only few studies on correlations with specific symptoms. Two groups found evidence for smaller posterior superior temporal cortex in patients with formal thought disorder (Shenton et al., 1992; Menon et al., 1995). Some preliminary evidence also links the superior temporal cortex (Barta et al., 1990) and the anterior cingulate (Noga et al., 1995) to hallucinations. However, these MRI studies were limited in resolution of studied MR images, as well as restricted to the analysis of single pre-defined regions of interest.

In this study, we tried to find evidence for regionally specific alterations accompanying auditory hallucinations in schizophrenia. Based on the functional studies (Line et al., 1998; Dierks et al., 1999; Lennox et al., 2000) and a previous structural MRI study (Barta et al., 1990), we set up a specific regional hypothesis for the perisylvian areas, i.e. the superior temporal cortical areas extending along the Sylvian fissure, as well as Broca’s area. We applied deformation-based morphometry (DBM), a novel fully automated whole-brain morphometric technique (Gaser et al., 1999), to high-resolution MR images to test the hypothesis that severity of auditory hallucinations might be related to focal brain shrinkage in perisylvian areas. The DBM method might overcome limitations of conventional volumetric assessments of brain structure, assessing differences on the voxel level over the entire brain and minimizing user bias (Gaser et al., 2001). We have previously applied this method to detect structural changes in patients with schizophrenia (Gaser et al., 1999; Volz et al., 2000) and compared the method to conventional volumetry (Gaser et al., 2001).

Methods

Subjects

We studied 85 right-handed patients with schizophrenia (33 females, 52 males; mean age 36.2 years, SD ±10.9), who were all on stable neuroleptic medication. Analyses comparing this population to healthy controls were reported earlier (Gaser et al., 1999). Patients were...
reliability in trained raters. The study was approved by the ethical
advantage of applying a well-established and validated rating system,
scores is displayed in Figure 1. Using these single SAPS items has the
maximum of ongoing auditory hallucinations in the period of at least 1
hallucinations are often transient, but brain structure is relatively
irrespective of the presence of auditory hallucinations. Thus, the
typo included both patients with and without a history of auditory
hallucinations. Most of the patients studied here enrolled in other
studies or research protocols as well and therefore multiple (up to
three) SANS and SAPS scores over a (variable) period of up to several
weeks before or after scanning MR scanning were available. As auditory
hallucinations are often transient, but brain structure is relatively
stable, we decided in these cases to take the maximum score of patients
achieved in a SANS/SAPS interview, as the potential error from under-
estimation hallucinations was expected to be much higher than
overestimating the score value. This means that the score reflected the
maximum of ongoing auditory hallucinations in the period of at least 1
week before the scanning session. Of the 85 patients, 56 patients did
not shown any hallucinations and their pooled auditory hallucination
(AH) score was zero. Twenty-nine patients had scores between 1 and
15 (the maximum possible value). The distribution of the pooled AH
scores is displayed in Figure 1. Using these single SAPS items has the
advantage of applying a well-established and validated rating system,
which is used in many studies with schizophrenia patients and has good
reliability in trained raters. The study was approved by the ethical
review board of the University of Jena and, after an explanation of the
scanning protocol, all participants gave written informed consent.

Data Acquisition and Image Processing

High-resolution MRI was performed on a 1.5 T Philips Gyroscan ACSII
system. We acquired 256 contiguous sagittal slices of 1 mm thickness
using a \( T_1 \)-weighted sequence \((T_E = 13 \text{ ms}, T_R = 5 \text{ ms}, \alpha = 25^\circ; \text{field-of-view} = 256 \text{ mm})\) with a matrix size of 256\(^2\), resulting in an isotropic voxel size of 1 mm\(^3\). For morphometric analysis of the data, we applied our DBM approach (Gaser et al., 1999, 2001). This method is based on nonlinear image registration, commonly used for the spatial normalization of functional imaging data across subjects. The DBM method analyses deformations used to normalize images onto a standard template brain by introduction of local deformations \("warp\)\)s\) to the object brains, in order to infer on structural differ-
cences. We have recently validated our DBM approach in a direct
comparison to semi-automated volumetric measurement in a group of
schizophrenia patients (Gaser et al., 2001). DBM has several advan-
tages over conventional volumetric techniques (i.e. tracing of
regions), including elimination of user-bias, minimization of partial
volume effects and the opportunity to study the entire brain rather
than pre-defined regions.

We used SPM99 spatial normalization routines (Ashburner and
Friston, 1999) with an algorithm based on image intensity differences
rather than landmarks or surfaces. First, we applied an affine trans-
formation to normalize all images to the same stereotactic space
(Talairach and Tournoux, 1988) of the template image (single subject
brain as provided in the SPM package). This step corrects for the
global differences of individual brains, such as brain size, as well as
orientation in space, using transformations such as scaling, rotation,
translation, or shearing. Hence, this step does not introduce changes
to single structures, but applies changes to all voxels of the brain
uniformly. It preserves the individual anatomy and does not introduce
a bias for specific regions. The spatially normalized images were then
resized to an isotropic voxel size of 2 mm. The second step of the
normalization procedure accounts for the remaining local anatomical
differences. This nonlinear registration is based on a regularized mini-
mization of the residual squared intensity difference between an
image and a template image, while simultaneously maximizing the
smoothness of the deformations. The coefficients of a linear combina-
tion of \(11 \times 13 \times 10\) three-dimensional discrete cosine transform basis
functions were estimated to perform this nonlinear registration
(Ashburner and Friston, 1999). Hence, in difference to the linear
normalization routine, the nonlinear normalization changes local
anatomical features rather than global parameters such as brain width.
This includes changes of single structures such as gyri, for which local
deformations are introduced to achieve similarity between the brain
being normalized and the template brain.

The deformations applied to accomplish this nonlinear normaliza-
tion were then used for computation of brain morphometric differ-
ences: we obtained a deformation field for each subject with a
specific three-dimensional displacement vector in every voxel. This
displacement vector defines the transformations required to map the
voxel of one brain onto its corresponding position in another brain.
Therefore it includes information of positional differences as well as
changes in local volume. Rather than using the entire information, we
extracted the local Jacobian determinant, a variable commonly used in
continuum mechanics (Gurtin, 1987), from the deformation fields.
This restricts information on morphometric differences to the volume
information, i.e. local shrinkage or expansion, yielding more precise
maps of volumetric differences than analysis of the entire deformation
field, as shown previously (Gaser et al., 2001).

Statistical Analysis

For our main statistical analysis we considered a general linear model
for the volume change in each voxel, testing the linear relationship
between the hallucination score and volume decrease of the assessed
differences. A general linear model matrix was set up, in which the
hallucination score was entered as a parameter. Furthermore, SANS
total score, SAPS total score without auditory hallucination sub-items
and gender were defined as confounding variables to eliminate their
possible effects. Hence, this analysis equals a multiple regression

![Figure 1](https://academic.oup.com/cercor/article-abstract/14/1/91/433501)
model which tests for correlations between the hallucination score and volume changes in each voxel, while eliminating the effects of the potentially confounding variables mentioned above. In contrast to simple correlations, the variance introduced by the above confounds can thus be eliminated. We have preferred this statistical approach rather than a simple dichotomization, since it avoids certain problems associated with a group analysis (e.g. choice of cut-off values for dividing groups into ‘hallucinating’ and ‘non-hallucinating’ patients, sensitivity to false categorization of single patients, etc.).

Perisylvian areas (i.e. Broca’s region and the superior temporal cortex, including the superior temporal gyrus with planum polare and temporale, extending up to the temporoparietal junction) were included in our hypothesis and therefore assessed without correction for multiple comparisons. For other cortical areas, we performed an exploratory analysis. Thus, we defined an overall threshold of significance of $P < 0.001$. As a result, we obtain statistical parametric maps for the entire brain showing voxels with a significant correlation to the hallucination score. To further minimize false positive errors, we applied a spatial threshold criterion: we only report clusters consisting of at least $k = 55$ voxels, corresponding to an extent threshold of $P = 0.01$.

Additionally, we performed supplementary analyses to further support our findings. These analyses were variations of the general linear model described above, the main difference being that we compared groups of subjects, rather than performing a correlation. Thus, each subject was assigned to a group, while the confounding variables as described above were also entered to correct for variance related to these. For the first additional analysis, we divided the patient group in those patients with versus those without hallucinations. For this group analysis, patients with a score of zero ($n = 56$) made up the ‘non-hallucinating’ group, whereas those with a score of 1 and above ($n = 29$) made up the second group of hallucinating patients. In a further variation of this analysis, we then re-grouped the patients with those of a score value of either zero or one being classified as ‘non-hallucinating’ ($n = 67$) and defining those with a score value of greater 1 ($n = 18$) as ‘hallucinating’. In these supplementary analyses, the difference of the Jacobian determinant between the groups (rather than a correlation) was computed in each voxel, thus yielding maps to describe voxels in which the hallucinating patients showed focal volume loss compared to non-hallucinating subjects. While this approach was considered to be less stringent than the correlation analysis, it parallels more closely most previous morphometric studies. The second supplementary analysis was computed to confirm that effects were indeed related to hallucinations rather than other aspects of the disease. For this purpose, we included a sample of 75 healthy controls in the group of non-hallucinating subjects. These controls, taken from the previous study (Gaser et al., 1999), were matched for sex and age to the schizophrenia group (22 women, 53 men; mean age 31.2 ± 9 years). They were all screened thoroughly before scanning to exclude any psychiatric or neurological history, developmental abnormality, history of head trauma, or major medical condition. They all scored zero on the auditory hallucination scale. We used the same statistical model of our main analysis to compute the correlation between hallucination scores and local volume decrease. We included hallucination scores as regressor variable and SANS, SAPS without auditory hallucinations, and gender as confounding variables. Thus, the second analysis equals our main analysis, but additionally includes a control group with zero values on the hallucination scores (SANS and SAPS values all being zero). Both supplementary analyses were performed to confirm results found in the main analysis.

Finally, we calculated correlations between the auditory hallucination score and certain selected demographic and psychopathological variables. This included testing relations with age, gender, the SAPS total score without auditory hallucinations, the SAPS formal thought disorder sub-items, the SAPS delusion sub-items and the SANS total score. In schizophrenia, certain symptoms are often found together with other specific psychopathological variables. For example, positive symptoms (such as those mentioned above) often occur together. These correlations were computed to show that auditory hallucinations could be well isolated from the complex psychopathology of the disease and had no substantial overlap in variance with the other main positive symptoms.

### Results

DBM analysis revealed three cortical regions showing a significant ($P < 0.001$) correlation of local volume decrease with severity of auditory hallucinations in the main analysis (Table 1 and Figs 2 and 3): a right prefrontal region comprising parts of the middle and inferior frontal gyri (including parts of the right hemisphere homologue of Broca’s area), the left transverse temporal gyrus (Heschl’s gyrus, including the primary auditory cortex) and the inferior part of the left supramarginal gyrus (posterior to the planum temporale). While the latter two regions were included in the previous anatomical hypothesis, the prefrontal finding includes only part of the homologue of Broca’s area and is hence based on the exploratory analysis of the entire brain.

Post-hoc inspection of secondary auditory areas surrounding Heschl’s gyrus, which were implicated in an earlier study (Barta et al., 1990), showed effects appearing only at a lower level of significance ($P < 0.01$).

The supplementary analyses confirmed the main finding. The three clusters were found for both group analyses of hallucinating versus non-hallucinating patients (irrespective of the chosen cut-off used to divide the sample) and for the supplementary correlational analysis including healthy control subject. In all analyses the three regions were significant ($P < 0.001$), while the location of the maximum voxels and the extent of the clusters varied slightly from the main analysis. The only significant correlation between auditory hallucination score and the selected demographic and psychopathological variables was found for SAPS delusion sub-items (correlation coefficient $r = 0.366$, $P = 0.0003$). All other correlations were not significant ($P < 0.01$). In particular, there was no significant correlation of auditory hallucination score with age of subjects ($r = 0.04$), gender ($r = 0.052$), SANS total score ($r = 0.114$), SAPS formal thought disorder sub-items ($r =

### Table 1

| $x$ | $y$ | $z$ | $t$-score | Effect size $d$ (95% confidence interval) | Region (Brodmann’s area) |
|-----|-----|-----|-----------|----------------------------------------|--------------------------|
| 44  | 40  | 14  | 4.15*     | 0.927 (0.497–1.357)                     | Right middle/inferior frontal gyrus (45/46) |
| −42 | −16 | 10  | 4.10*     | 0.916 (0.486–1.346)                     | Left transverse temporal gyrus (Heschl’s) (41) |
| −44 | −50 | 32  | 3.90*     | 0.871 (0.441–1.301)                     | Left inferior supramarginal gyrus (40) |

* $P < 0.001$; spatial extent $k = 55$ voxels. Coordinates (given in millimetres) refer to the template space and correspond approximately to the space of the Talairach atlas. The effect size of $d$ indicates to what extent the volume decrease is related to hallucination score.
The main finding of our study is that structural changes associated with auditory hallucinations include the gyrus of Heschl, an area mostly coinciding with the primary auditory cortex (Liegeois-Chauvel et al., 1991). Previous studies on temporal lobe pathology in positive symptoms have suggested that changes in the posterior portions of the superior temporal gyrus (STG) to be relevant for formal thought disorder (Shenton et al., 1992; Menon et al., 1995). Another study has suggested a relation to STG morphology and auditory hallucinations (Barta et al., 1990), but since a coarse resolution (including gaps between MRI slices) was used, no precise localization within STG cortical fields was possible. Our results show that in auditory hallucinations, the preferentially affected area appears to be located in the primary auditory cortex, rather than the secondary or association areas of the posterior planum temporale. Thus, these two major symptoms of schizophrenia both appear related to localized shrinkage in the left superior temporal cortex, but with slightly different anatomical localization. However, the difference in localization of changes might be relative, since other areas around the gyrus of Heschl’s appeared at lower thresholds on post-hoc inspection and the previous studies on formal thought disorder (Shenton et al., 1992; Menon et al., 1995) did not include specific delineation of the gyrus of Heschl. Recent cognitive studies, however, give further support for the assumption of regional separation. These studies have shown distinct profiles of cognitive dysfunction to be associated with particular positive symptoms (Kerns and Berenbaum, 2002). Formal thought disorders and auditory hallucinations appear to be associated with different profiles of cognitive deficits, for example in word production (Kerns et al., 1999).

The cross-sectional design of our study does not allow inference on the timing of these alterations and possible changes during the course of the disorder. Recent studies in first-episode schizophrenia, however, underline that STG pathology is often present at the onset of schizophrenia (Hirayasu et al., 2000) and it is therefore unlikely to be simply an effect of medication or hospitalization, even though some brain regions might show progressive reduction in some patients on follow-up after several years (Mathalon et al., 2001).

Another area of volume loss was located in the left supramarginal gyrus. Temporoparietal and inferior parietal cortical areas have been difficult to measure in conventional morphometric approaches. However, these areas are central for the processing of language. The supramarginal gyrus in particular is a key module of the auditory/phonological loop, a circuitry mediating the short-term storage and processing of auditory information and especially language (Paulesu et al., 1993). Although the phenomenology of auditory hallucinations can be heterogeneous, many (if not most) patients present with verbal hallucinations, such as voices conversing or commenting on the patient (David and Busatto, 1998). This second cluster in our results is therefore suggestive of a pathology involved in higher aspects of language. Also, schizophrenia patients with auditory hallucinations show selective deficits in speech perception (Hoffman et al., 1999), underlining the intimate relation to disturbed anatomical substrates of speech. Both temporal and temporoparietal clusters show an overlap with the functional neuroanatomy of physiological processing of auditory information and language (Silbersweig and Stern, 1998).

Beside these two areas, which were included in our anatomical hypothesis, we found an effect in the right prefrontal cortex, a major target of previous imaging studies in schizophrenia. Although the maximum voxels in this cluster were in fact more significant than in the STG clusters, this finding can only be reported as a trend, as in the absence of a previous anatomical hypothesis it was encountered at a threshold uncorrected for multiple comparisons. Nevertheless, this finding deserves attention in the light of studies emphasizing fronto-temporal interplay of regions in volitional (and involitional)
auditory perception (Silbersweig and Stern, 1998). While several studies indicate prefrontal cortical abnormalities in schizophrenia (Wible et al., 2001), there are relatively few studies relating specific symptoms to this region. This study provides a first link of auditory hallucinations in schizophrenia to a discrete prefrontal structural change. Interestingly, this alteration partially includes the right hemisphere homologue of Broca’s area. As with temporal cortical changes, deficits in frontal gray matter are also detectable in first-onset patients (Hirayasu et al., 2001). Further studies might be warranted to investigate the relation of prefrontal to temporal function in auditory hallucinations.

Our results implicate that auditory hallucinations are not associated with a single regional deficit. Rather, several nodes of a more complex circuitry might be involved. Changes in primary auditory cortex and temporoparietal areas might be at the core of this abnormality, and together with prefrontal deficits this could result in deficient frontotemporal interaction. Recent functional imaging studies have elucidated the function of frontotemporal networks on conscious volitional auditory perception and sensory awareness (Frith, 1996; Silbersweig and Stern, 1998). These results emphasize an interaction between auditory temporal areas and the prefrontal cortices, which constantly modulate the superior temporal areas, therefore enabling facilitation or inhibition of the processing of sensory information. In schizophrenia, this frontotemporal network appears to be affected, either through local structural deficits or due to compromised connectivity of regions, which might lead to the emergence of positive symptoms such as auditory hallucinations. In neuropsychological terms, dysfunction of this network could be understood as a failure to inhibit and attribute internal speech, as suggested previously in theories relating to ‘inner speech’ (McGuire et al., 1995; Johns and McGuire, 1999).

So far, there are few data on the relation of specific symptoms to regional neuropathology in schizophrenia. Our method only allows inference on volume changes. Thus, it does not disclose the type of microscopic pathology underlying these changes. While alterations on the cellular level might be held responsible at least for part of the effects, there might be other factors, such as changes in local blood flow or blood volume, that contribute to the MRI volumetric structural measurements.

Another general problem in assessing the relation of symptoms to structural changes is the transient nature of most symptoms, particularly hallucinations. Similar to post-mortem studies, it is difficult to obtain valid scores of a person’s lifetime experience of auditory hallucinations. For our study psychopathology scores were chosen to cover at least 1 week before scanning. Our results therefore also rely on the assumption of a correlation between the stability of auditory hallucinations (possibly a persistent predisposition for experiencing them) and underlying brain structure.

In conclusion, our results can be interpreted as a symptom-specific distributed structural deficit, comprising multiple nodes of a more complex frontotemporal circuitry. The particular anatomical distribution of changes is associated with the emergence of the specific symptom of auditory hallucinations. Arising from both prefrontal and superior temporal cortical pathology, this abnormality predisposes patients to transient auditory hallucinations with its most pertinent characteristics: its involitional nature (inability to suppress these perceptions), the predominantly verbal phenomenology of ‘voices’ and, finally, the co-occurrence with other positive symptoms arising from prefrontal and/or superior temporal pathology in schizophrenia. Novel morphometric techniques might open further avenues to segregate localized abnormalities and specific symptoms or syndromes in schizophrenia.

Notes
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