Patients with Alzheimer’s disease and dementia with Lewy bodies mistaken for Creutzfeldt-Jakob disease


Abstract

Objectives—To describe the clinical presentation of patients with Alzheimer’s disease (AD) or dementia with Lewy bodies (DLB) who were suspected of having Creutzfeldt-Jakob disease (CJD) and to investigate whether current clinical diagnostic criteria cover these atypical forms of AD and DLB.

Methods—Brains from necropsy were examined for the diagnosis of CJD at the German reference centre for spongiform encephalopathies. Symptoms and signs in patients with suspected CJD in whom necropsy showed AD (n=19) or DLB (n=12) were analysed. Their data were compared with a group of patients with CJD (n=25) to determine overlapping and discriminating clinical features. All patients were classified according to clinical diagnostic criteria for CJD, AD, and DLB.

Results—Demented patients were suspected of having CJD if disease was rapidly progressing and/or focal neurological signs appeared and/or an EEG showed sharp wave complexes. Myoclonus and limb rigidity were the most common neurological signs in all three dementias. DLB was not suspected in any patient, although patients with DLB showed parkinsonism (58%) and fluctuations (58%). Periodic sharp wave complexes (PSWCs) in EEG typical of CJD were found in five patients with AD and one patient with DLB. 1–4–3 Protein in CSF was detected in 20 patients with CJD, in two patients with AD, but not in any patient with DLB. Although most patients with DLB or AD met the clinical criteria for their respective diagnosis (74% and 90%), they also fulfilled criteria for CJD (42% and 58%).

Conclusions—In patients with rapidly progressive dementia and focal neurological signs, CJD should be the first line diagnosis. Facing the triad dementia, myoclonus, and rigidity, AD should be considered if the disease course is longer and DLB is the differential diagnosis if parkinsonism or fluctuations are present. Findings on EEG or CSF typical of CJD do not exclude AD or DLB.

Keywords: Creutzfeldt-Jakob disease; dementia with Lewy bodies; Alzheimer’s disease; diagnosis

Creutzfeldt-Jakob disease (CJD), Alzheimer’s disease (AD), and dementia with Lewy bodies (DLB) are human neurodegenerative disorders presenting with dementia as their principal sign. All three are of unknown origin and have a fatal outcome. Whereas CJD is a rare disease with an incidence of around one case/million/year, AD and DLB are the two most common causes of dementia in elderly people.

In most cases, patients with CJD have a rapidly progressive disease course and periodic sharp wave complexes (PSWCs) in the EEG. They usually develop neurological signs such as gait disturbances or myoclonus. Most patients become bedridden and die after a median disease duration of 7–8 years. Gener- ally, AD is of slow course (duration of illness 7–8 years) and neurological signs appear only in late stages of the disease. DLB was recently defined as a separate disease entity with the characteristics of dementia, parkinsonism, visual hallucinations, fluctuations in cognitive impairment, and neuroleptic hypersensitivity. Life expectancy in DLB of 5–8 years is shorter than in AD.

Studies on patients with DLB whose diagnoses were confirmed by necropsy focus on the clinical differentiation of DLB and AD or DLB and Parkinson’s disease. They have included only a few patients. There are case reports of necropsy confirmed patients with AD and patients with DLB in whom CJD had been clinically suspected, but no systematic study has yet been undertaken to differentiate those patients with a rapidly progressive disease course.

In our study we describe a larger number of patients suspected clinically of having CJD but in whom AD or DLB was shown at postmortem examination. Frequency of clinical signs and symptoms and the results of technical investigations are reported. Reliability of current clinical diagnostic criteria for CJD, DLB, and AD is evaluated, and a diagnostic pattern is proposed to facilitate clinical differential diagnosis.

Materials and methods

PATIENTS

Since 1993 brains from necropsies have been sent to the German reference centre for spongiform encephalopathies for the diagnosis of Creutzfeldt-Jakob disease or genetic prion disease. Most patients necropsied were examined as suspected CJD cases during their lifetime by a study physician of the German CJD surveillance unit.
We collected clinical data from patients in whom CJD was ruled out by neuropathology, and in whom AD or DLB were confirmed.

The control group of 25 patients with definite sporadic CJD was recruited from patients consecutively referred to the CJD surveillance unit in the same period as the patients with AD and those with DLB. Recruitment of patients with CJD ceased as soon as a representative group of patients was achieved that corresponded in clinical signs and symptoms to our previous findings.1 Patients with genetic15 or iatrogenic prion disease were excluded.

ANALYSIS OF CLINICAL DATA

Medical records of all patients were studied retrospectively. Each patient’s chart was analysed with a standardised checklist, which contained the following items:

(1) Reason for sending in material for prion disease analysis.

(2) Signs and symptoms typical for CJD were rated according to modified Masters criteria.1 15–17 Extrapyramidal signs were subdivided into limb rigidity, parkinsonism, and dystonic, choreatic, athetoid or ballistic movements. Visual disturbances included deteriorated or blurred vision, visual field restriction, and disturbed perception of structures and colours. Visual hallucinations were rated separately.

Clinical features of AD were rated according to NINCDS-ADRDA criteria.4 Clinical features of DLB were noted according to McKeith et al.5

(3) Technical investigations: All EEGs were initially reviewed by one of us (SP). The EEGs were additionally reviewed without knowledge of clinical information by a second observer (BJS).19 20 “CJD typical EEG” was diagnosed in EEG tracings fulfilling Steinhoff’s criteria. Testing for protein 14–3–3 and neuron specific enolase (NSE) in CSF was performed as previously described.14 A cut-off for CJD was defined as NSE concentration >35 ng/ml in CSF.18

CLINICAL CLASSIFICATION OF PATIENTS

Using the results from the above checklist, patients were classified according to international diagnostic criteria for CJD,16 AD,4 and DLB.5 Each patient was clinically classified for all three dementia entities. The categories “possible”, “probable”, and “other disease” (not fulfilling the criteria) were used to express the degree of certainty in the clinical diagnosis.

NEUROPATHOLOGICAL EVALUATION

Brains were fixed in 10% formalin for at least 14 days. Representative blocks were cut, decontaminated for 1 hour in concentrated formic acid,21 and embedded in paraffin. Whenever possible recommended regions according to the consensus criteria for diagnosing AD and DLB were used.5 22 Paraffin sections (2 µm) were stained with haematoxylin and eosin and with Bielschowsky methods.

Immunocytochemistry was performed with monoclonal antibodies against a synthetic pep-
Table 1
Clinical signs and symptoms at onset of disease

<table>
<thead>
<tr>
<th>Sign/symptom</th>
<th>CJD patients (%) (n=25)</th>
<th>AD patients (%) (n=19)</th>
<th>DLB patients (%) (n=12)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dementia only</td>
<td>25 (100)</td>
<td>19 (100)</td>
<td>12 (100)</td>
</tr>
<tr>
<td>Myoclonus</td>
<td>22 (88)</td>
<td>11 (58)</td>
<td>9 (75)</td>
</tr>
<tr>
<td>Extrapyramidal signs</td>
<td>21 (84)</td>
<td>10 (53)</td>
<td>11 (92)</td>
</tr>
<tr>
<td>Limb rigidity</td>
<td>17 (68)</td>
<td>10 (53)</td>
<td>11 (92)</td>
</tr>
<tr>
<td>Parkinsonism</td>
<td>2 (8)</td>
<td>2 (11)</td>
<td>7 (58)</td>
</tr>
<tr>
<td>Chorea or athetoid/dystonic signs</td>
<td>10 (40)</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Pyramidal signs</td>
<td>19 (76)</td>
<td>6 (32)</td>
<td>2 (17)</td>
</tr>
<tr>
<td>Cerebellar signs</td>
<td>23 (92)</td>
<td>7 (37)</td>
<td>—</td>
</tr>
<tr>
<td>Gait or postural ataxia</td>
<td>20 (80)</td>
<td>3 (16)</td>
<td>—</td>
</tr>
<tr>
<td>Limb ataxia</td>
<td>15 (60)</td>
<td>5 (26)</td>
<td>—</td>
</tr>
<tr>
<td>Visual disturbances</td>
<td>12 (44)</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Akinesia or mutism</td>
<td>16 (64)</td>
<td>4 (21)</td>
<td>1 (8)</td>
</tr>
<tr>
<td>Fluctuations</td>
<td>—</td>
<td>1 (5)</td>
<td>7 (58)</td>
</tr>
<tr>
<td>Visual hallucinations</td>
<td>9 (36)</td>
<td>2 (11)</td>
<td>7 (58)</td>
</tr>
<tr>
<td>Recurrent falls</td>
<td>4 (16)</td>
<td>6 (32)</td>
<td>6 (50)</td>
</tr>
<tr>
<td>Neuroleptic hypersensitivity</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Delusions</td>
<td>3 (12)</td>
<td>5 (26)</td>
<td>9 (75)</td>
</tr>
</tbody>
</table>

*Visual disturbances included deterioration of vision (n=2), blurred vision (n=3), visual field restriction (n=4), disturbed perception of structures and colours (n=3), and double vision (n=3).
†Neuroleptic medication was received by six AD patients, nine DLB patients, and one CJD patient without notice of hypersensitivity by treating physician.
— Signs not noted.

Table 2
Summary of clinical signs and symptoms during course of disease

<table>
<thead>
<tr>
<th>Sign/symptom</th>
<th>CJD patients (%) (n=25)</th>
<th>AD patients (%) (n=19)</th>
<th>DLB patients (%) (n=12)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dementia only</td>
<td>7 (28)</td>
<td>12 (63)</td>
<td>5 (42)</td>
</tr>
<tr>
<td>Myoclonus + neurological signs</td>
<td>4 (16)†</td>
<td>3 (16)†</td>
<td>2 (17)†</td>
</tr>
<tr>
<td>Gait disturbances</td>
<td>11 (44)</td>
<td>15 (79)</td>
<td>7 (58)</td>
</tr>
<tr>
<td>Visual disturbances</td>
<td>7 (27)</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Confusion</td>
<td>4 (16)</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Vertigo</td>
<td>4 (15)</td>
<td>1 (5)</td>
<td>—</td>
</tr>
<tr>
<td>Aphasia</td>
<td>2 (8)</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Behavioural changes</td>
<td>2 (8)</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Dysphoria</td>
<td>1 (4)</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Hallucinations</td>
<td>1 (4)</td>
<td>1 (5)</td>
<td>—</td>
</tr>
<tr>
<td>Parkinsonism</td>
<td>2 (8)</td>
<td>—</td>
<td>1 (8)</td>
</tr>
<tr>
<td>Limb paresis</td>
<td>1 (4)</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Myoclonus</td>
<td>1 (4)</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Epileptic seizures</td>
<td>—</td>
<td>3 (16)</td>
<td>—</td>
</tr>
<tr>
<td>Delusion</td>
<td>—</td>
<td>—</td>
<td>1 (8)</td>
</tr>
<tr>
<td>Aggressive behaviour</td>
<td>—</td>
<td>—</td>
<td>1 (8)</td>
</tr>
<tr>
<td>No data</td>
<td>—</td>
<td>—</td>
<td>3 (25)</td>
</tr>
</tbody>
</table>

*Multiple mentioning possible.
†Neurological signs in CJD patients were dystonia, gait disturbances, visual disturbances, and confusion; in AD patients they were myoclonus, epileptic seizures or gait disturbances; in DLB patients they were parkinsonism or visual hallucinations.
— Signs not noted.

Table 3
Results of technical investigations

<table>
<thead>
<tr>
<th>Investigation</th>
<th>CJD patients (%) (n=25)</th>
<th>AD patients (%) (n=19)</th>
<th>DLB patients (%) (n=12)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PSWCs in EEG</td>
<td>56% (14/25)</td>
<td>31% (5/16)</td>
<td>8% (1/12)</td>
</tr>
<tr>
<td>Protein 14-3-3 in CSF</td>
<td>87% (20/23)</td>
<td>18% (2/11)</td>
<td>0% (0/5)</td>
</tr>
<tr>
<td>Median NSE value in CSF (ng/ml)</td>
<td>62.5 (range: 15-210)</td>
<td>18% (2/11)</td>
<td>0% (0/5)</td>
</tr>
<tr>
<td>NSE raised in CSF</td>
<td>73% (16/22)</td>
<td>29% (2/7)</td>
<td>0% (0/5)</td>
</tr>
</tbody>
</table>

PSWCs=periodic sharp wave complexes; NSE=neuron specific enolase.

Patients with AD and LBD mistaken for CJD

The median duration of illness varied from 4 months (range 2–24 months) in patients with CJD to 18.5 months (range 1–60 months) in patients with DLB and 24 months (range 3–108 months) in patients with AD. A patient with AD was suspected of having CJD after 9 years of disease course when he developed rigidity and myoclonus.

REASONS FOR SUBMITTING BRAIN TISSUE FOR PRION DISEASE ANALYSIS

Patients with CJD (n=25)

In 20 patients the primary clinical diagnosis was CJD, the remaining five patients were referred as patients with suspected CJD with other differential diagnoses.

Patients with AD (n=19)

Nine patients had rapidly progressive dementia with or without focal neurological signs and the EEG showed sharp wave complexes; therefore CJD was clinically suspected. “Probable” CJD was diagnosed in five of these patients with AD as sharp wave complexes fulfilled criteria for periodicity. In 10 further patients CJD was considered in the differential diagnosis as the disease course was very rapid.

Patients with DLB (n=12)

One patient’s clinical diagnosis by the notifying hospital was CJD, because of rapidly progressing illness with dementia, parkinsonian syndromes, and myoclonus, but without specific EEG findings. In the remaining patients CJD was suspected because of rapid progression of disease, and/or the development of myoclonus. In two of these patients, EEG showed sharp wave complexes, one of those fulfilled criteria for periodicity; therefore in this patient CJD was clinically “probable”.

CLINICAL SIGNS AND SYMPTOMS AT ONSET OF DISEASE

The details are listed in table 1. Dementia was the first sign of disease in most patients with AD and those with DLB and in 44% of patients with CJD. Whereas patients with AD or with DLB often had dementia only, in many patients with CJD, dementia was accompanied by neurological signs or symptoms, or neurological signs preceded dementia.

CLINICAL SIGNS AND SYMPTOMS DURING THE COURSE OF DISEASE

All patients developed dementia. Myoclonus and extrapyramidal signs were especially common in patients with CJD and patients with DLB (>80% and >70% respectively) and in more than 50% of patients with AD (see table 2 for details). Extrapyramidal signs in patients with AD and with DLB were restricted to limb rigidity (>50% respectively) and parkinsonism (58% in patients with DLB, 8% in CJD, and 11% in AD). Patients with CJD additionally showed hyperkinetic signs (40%). Nearly all patients with CJD and 37% of patients with AD had cerebellar signs, not noted in DLB. Visual disturbances such as loss or deterioration of vision were noted in 11% in AD. Patients with CJD additionally showed myoclonus (58% in patients with DLB, 8% in CJD, and 11% in AD). Patients with CJD additionally showed hyperkinetic signs (40%).
tion of vision, visual field restriction, or disturbed perception of colours and structures were exclusively found in CJD (44% of patients).

Signs typical of DLB such as fluctuations of cognitive impairment were found in 58% of patients with DLB, but only in one patient with AD and no patients with CJD. Visual hallucinations were most common in DLB (58%), but were also present in CJD (36%) and AD (11%). Referring physicians did not report neuroleptic hypersensitivity in any patient, including nine patients with DLB (75%), six patients with AD (32%), and one patient with CJD (4%) who had received neuroleptic medication.

![EEG](http://jnnp.bmj.com/)

**Figure 1** CJD typical periodic sharp wave complexes in EEGs from (A) a histologically proved AD patient, aged 76 years after 4 months of disease (EEG curves by courtesy of Dr E Banzhaf). (B) A histologically proved patient with DLB, aged 77 years, after 36 months of disease (EEG curves by courtesy of Professor R Benecke).
Patients with AD and LBD mistaken for CJD

In 16% of patients with AD the diagnosis of “possible” CJD was made, in 26% “probable” CJD was diagnosed clinically; 74% of patients with AD fulfilled clinical criteria of “probable” AD; one patient with AD (5%) met the criteria for “probable” DBL (dementia, fluctuations, parkinsonism, and visual hallucinations); and one patient met the criteria for “possible” DBL (dementia and visual hallucinations). In another patient with AD, dementia had preceded the development of parkinsonism by 9 years, so that in this case parkinsonism was not rated for the diagnosis of DBL.

Patients with definite DBL

“Possible” CJD was diagnosed in 50% of patients with DBL, but only one patient with DBL had CJD typical PSWCs in EEG recordings and was therefore clinically “probable” CJD; 58% of patients with DBL met criteria for “probable” AD; 84% of patients with DBL fulfilled clinical criteria for “possible” or “probable” DBL (42% each, fig 2).

Discussion

Our series is the first to describe clinical findings on a large number of patients erroneously suspected of having Creutzfeldt-Jakob disease, who were histologically proved to have Alzheimer’s disease or dementia with Lewy bodies. We are aware of only three comparable series20–28 that report necropsy results of suspect patients with CJD who had other final diagnoses. Detailed clinical information was not given in these series. In all studies, AD was the most frequent alternative diagnosis; DBL was not mentioned, as it was only recently defined as a separate diagnostic entity.

As shown in clinicopathological studies,7 20–29 clinical diagnosis in patients with dementia has become increasingly accurate, ranging from a specificity of 95% in CJD28 and 90%–97% in DBL22 to 79%29 in AD. Thus, patients with typical clinical presentation seem to be sufficiently covered by current diagnostic criteria. However, differential diagnosis is a major challenge in atypical cases, such as patients with rapidly progressive AD or DBL or patients with slowly progressive CJD.

In our study, CJD was suspected in patients with AD or DBL if the disease course was rapid or patients developed focal neurological signs, especially myoclonus, or if EEG was considered to point to CJD.

In most patients with AD or with DBL, dementia was present already at the onset of disease, corresponding to the literature.4 17 By contrast, patients with CJD additionally had focal neurological signs such as gait or visual disturbances. During the course of disease dementia, myoclonus, and limb rigidity were the overlapping features of the three patient groups. Due to selection of “CJD-like” cases myoclonus was more common in DBL (75%) and AD (58%) than usually reported (19% in DBL19 and 10% in AD8). In accordance with the literature,15 extrapyramidal signs were the cardinal features in patients with DBL. Parkinsonism was rarely found in patients with AD.
and those with CJD, but was present in 58% of patients with DLB. Although described also in 6% of patients with DLB in the literature, we found choreoathetoid or dystonic movements exclusively in patients with CJD. Clinical signs particularly common in CJD were ataxia and visual disturbances. A differentiation of visual disturbances from visual hallucinations was especially helpful. Although visual disturbances were found exclusively in CJD, visual hallucinations were present in all three dementias. As there are reports from non-CJD dementia syndromes which show predominantly visual impairments, this should be further investigated.15

Although the clinical course was more rapid and myoclonus was more common than generally, our patients with DLB showed the cardinal features of DLB1 (fig 3). Neuroleptic hypersensitivity, a complication in DLB, was not described in any of the patients by the treating physicians. Looking for the clinical details in our patients with DLB, nine of them received neuroleptic medication (median 2 months before death). In six of them, further clinical deterioration and death (median 1 month later) could be consistent with neuroleptic hypersensitivity as described in the literature.14 In three patients with DLB, no hypersensitivity to neuroleptic drugs was found, even with hindsight. Both factors, the lack of hypersensitivity in some patients and no clear correlation of clinical deterioration with administration of neuroleptic drugs, could have contributed to missing the clinical diagnosis of DLB.

Using the diagnostic criteria for PSWCs proposed by Steinhoff et al,16 17 we found EEGs typical of CJD in 56% of patients with CJD, which is slightly less than previously reported.19 In five patients with AD and one patient with DLB, EEG was indistinguishable from typical CJD findings (fig 1A and B). In four additional patients with AD and two patients with DLB, EEG was considered by notifying hospitals to be compatible with CJD, but these EEGs did not correspond to the established criteria. The high frequency of false positive EEGs is most probably due to study design: demented patients are often referred due to “CJD-like” EEG. In the literature there are reports on periodic sharp wave complexes in single cases of AD11 12 and in two cases of DLB.13 14 “Pseudoperiodicity” in EEG is reported to be more often found in patients with DLB.35

Analysis of CSF16 yielded false positive results for 14–3–3 protein and NSE in one patient with AD and false positive 14–3–3 with normal NSE in another patient with AD. An explanation for the first patient could be an epileptic seizure 4 days before taking the CSF. In the five patients with DLB tested, 14–3–3 was negative and NSE was within the normal range. Negative findings for protein 14–3–3 and NSE in the CSF have also been found in five patients with DLB in another laboratory (Aksamit AJ, personal communication and Aksamit et al36).

According to Parchi et al,37 in sporadic CJD disease different subtypes exist. As described in detail by Zerr et al,38 the typical clinical phenotype with rapidly progressive dementia, myoclonus and typical EEG and CSF findings is present in about 70% of patients with CJD. In 10% of cases of CJD, disease duration is longer and patients often show extrapyramidal signs (MV2 subtype).39 In these patients EEG does not show CJD typical PSWCs and testing for protein 14–3–3 yields inconsistent results.16 The patients with MV2 and CJD could thus be missed or mistaken as a non-CJD neurodegenerative disorder, especially DLB. By contrast with AD or DLB, the patients with MV2 and CJD often exhibit ataxia as well as extrapyramidal features. Magnetic resonance imaging showing CJD typical findings in patients with MV2 could also help in the differential diagnosis. Due to lack of material no MR scans were described in this study.

Testing overlap in classification criteria (fig 2), we showed that “probable” CJD was misdiagnosed in six patients with the combination of limb rigidity, myoclonus, and false positive EEGs or CSF data. Classification of all patients according to AD criteria showed also that six patients with CJD and seven patients with DLB met criteria for “probable” AD. Comparable series on patients with DLB reported clinical criteria for “probable” AD in 15% and for “possible” AD in 50% of cases. In our study, DLB criteria specifically identified 84% of patients with DLB and falsely classified 16% of patients with CJD and 11% of patients with AD as having DLB.

Although our patients with DLB showed the major clinical signs of DLB (table 2), this disease was not mentioned as a clinical diagnosis in any of our patients. This accords with general findings that DLB escapes diagnosis in up to 66% of cases during a lifetime,4 probably because it is still quite unknown to the clinicians and specific technical examinations for DLB are lacking.

In summary, in patients with rapidly progressive dementia and focal neurological signs, CJD should be the primary diagnosis, especially if EEG shows periodic sharp wave complexes or if the 14–3–3 protein is detectable in CSF (fig 4). Ataxia, visual disturbances, and hyperkinetic limb movements are strong

Figure 3 Clinical overlap between CJD, AD, and DLB. *See materials and methods for definition.
hippocampal hyperintensities in protein 14–3–3 in CSF
PSWC in EEG
and/or slow progression
Duration >2 years
Fluctuations and/or visual hallucinations and/or parkinsonism
Neuropathology for definite diagnosis

Figure 4 Diagnostic pattern in the differential diagnosis of patients with the triad dementia, myoclonus, and rigidity.

hints for CJD. In a patient with the triad dementia, myoclonus, and rigidity with or without EEG changes, the history should be thoroughly reinvestigated to exclude the possibility that the current condition is the end stage of a dementia disorder with a longer duration. In the second case, AD should be considered. A suspicion of DLB should be raised, if an akinetic rigid parkinsonism or fluctuations in cognition are present.

This study was supported by a grant from the Bundesministerium für Bildung und Forschung, Bonn, Germany, to HAK and to SP (BMG Gz 325–4711–02/15). We thank all the physicians who referred suspect patients to the German reference centre for sporadic encephalopathies, for providing necropsy brains, and clinical data. We are especially obliged to Professor R Benecke, University of Rostock, Germany and to Dr B Banzhaf, Klinikum Münchberg GmbH, Germany, for kindly providing EEG curci. We thank Dr Otto Windl, Department of Neuropathology, University of Göttingen for analysis of the prion protein (PrP) gene (PrNP). We express our thanks to Drs Sabine Rümke, Stefan Grosse, Amelie Kordts, Martha Kemm, Maria Scherg, Volker Leist, Werner Schäfer, and Stefan Kropp, who worked as visiting physicians in the CJD surveillance unit. We are obliged to Monika Bodemer and Barbara Csicsery, for CSF analysis, Maia Schneider-Dominco, Chris Ernsting, and Cynthia Bunker for technical assistance, and Drs Markus Otto and Jens Willfang for their support.

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J Neurol Neurosurg Psychiatry 2001 71: 33-39
doi: 10.1136/jnnp.71.1.33

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