

The syndrome of Karl Ludwig Kahlbaum

MP BARNES, M SAUNDERS, TJ WALLS, I SAUNDERS, CA KIRK

From the Department of Neurology, Middlesbrough General Hospital, Middlesbrough, UK

SUMMARY Karl Ludwig Kahlbaum was the first to describe catatonia in 1868. There has been a tendency to consider catatonia as a psychiatric disease despite many case reports demonstrating a wide range of medical and neurological as well as psychiatric causes. We present our accumulated experience of the catatonic syndrome. Most cases (36%) were associated with affective illness but five cases (20%) had a defined organic disorder. A significant minority had no identifiable cause and there was only one case of schizophrenia. The idiopathic and affective groups had a high incidence of recurrent catatonic episodes and many had a family history of a similar problem. The prognosis was excellent, except for the few patients who presented with the acute and rapidly progressive form of the syndrome which led to acute renal failure.

Karl Ludwig Kahlbaum first described catatonia during a lecture in Innsbruck in 1868 and published his work on the subject 6 years later in a small monograph entitled "Die Katatonie oder das Spannungsirresein".^{1,2} He stressed that catatonia is strongly associated with affective illness, both depression and mania. He also gave examples of other diseases that are associated with catatonia; alcoholism, epilepsy, malaria, syphilis and tuberculosis. Although some of his cases eventually became "demented", he stressed that catatonia should not always be seen as a degenerating mental condition but in many cases there was a good prognosis for recovery. However, in 1893 Emil Kraepelin incorporated catatonia as a subtype of dementia praecox³ which was later termed schizophrenia by Eugene Bleuler.⁴ The work of Kraepelin and Bleuler has had much influence in the field of psychiatry and consequently there has been a tendency to consider catatonia as a purely psychiatric condition commonly associated with schizophrenia. In recent years there have appeared many reports of an association between catatonia and a wide variety of medical, neurological and psychiatric conditions.⁵

The aim of this paper is to present our experience of catatonia in a neurological unit over a 12 year period. Some of our cases illustrate new features of the catatonic syndrome.

Case reports

A total of 25 cases of catatonia have been seen by the Department of Neurology at Middlesbrough General Hospital during the period 1972 to 1984. The series may under-represent cases with a psychiatric cause although we believe that most instances of catatonia have been referred to the Department because of the known interest of one of us (MS) in the disorder. Our minimum definition of catatonia is that the patient must have exhibited at least one motor sign (cataplexy, posturing or waxy flexibility) in combination with at least one sign of psycho-social withdrawal or excitement and/or bizarre repetitious movements (mutism, negativism, impulsiveness, grimacing, stereotypies, mannerisms, command automatism, echopraxia/echolalia or verbigeration). Most patients were also stuporose on presentation although we do not consider that stupor is a necessary prerequisite for the diagnosis of catatonia. Every patient had been seen by a neurologist (MS) and also by a consultant psychiatrist. Every effort has been made to make either a psychiatric or organic diagnosis. Investigations have included CT scanning, EEG, examination of cerebro-spinal fluid and appropriate screening tests for the known metabolic and systemic causes of the syndrome.⁵

In 15 cases we have defined the likely cause of the syndrome. Nine patients (36%) had an affective illness. Five patients (20%) had a probable organic cause and in one case (4%) the patient was found to have schizophrenia. In 10 cases (40%) no organic nor psychiatric cause could be determined despite intensive investigation.

Psychiatric causes

Table 1 summarises the cases associated with a defined psychiatric condition. The single case of catatonia associated with previously known schizophrenia was in a 26-year-old male. The catatonic episode responded to electroconvulsive therapy (ECT) but the long-standing schizophreniform illness continued unchanged. In the other nine cases catatonia

Address for reprint requests: Dr M Saunders, Department of Neurology, Middlesbrough General Hospital, Ayresome Green Lane, Middlesbrough, Cleveland, TS5 5AZ, UK

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Table 1 *Psychiatric causes*

Patient	Sex	Age at onset of catatonia (yr)	Diagnosis	Number of catatonic episodes	Family history	Comments	Outcome
1 MT	M	26	Chronic schizophrenia	1	None		Catatonia responded to ECT but schizophrenic illness continues
2 WC	M	60	Recurrent catatonia associated with depression	3	Twin brother (case 3) had one episode of acute catatonia	Died by accidental drowning (not known to be depressed at the time)	Full recovery with ECT
3 GC	M	63	Acute catatonia associated with depression	1	Twin brother of case 2		Died
4 ML	F	21	Recurrent catatonia associated with depression	3	None	Also has generalised epilepsy	Episodes responded to ECT
5 FS	F	25	Recurrent catatonia associated with depression	6	Aunt, sister and cousin (case 6) have had catatonia associated with depressive illness sister died from "acute lethal" catatonia		5 episodes responded to ECT Died in acute, 6th, episode from renal failure
6 DF	F	17	Recurrent catatonia associated with depression	5	Cousin of case 5		Full recovery with ECT
7 HH	M	35	Recurrent catatonia associated with depression	2	Aunt (case 8) has similar problem. Sister had catatonia episode of uncertain cause		Recovery with ECT
8 DW	F	25	Recurrent catatonia associated with depression	5?6	Aunt of case 7		Recovery with ECT
9 VP	F	26	Acute catatonia secondary to puerperal depressive illness	1	None		Spontaneous recovery
10 SS	F	13	Recurrent catatonia associated with depression	2	Grandfather, great-grandfather and greatuncle all had similar condition		Spontaneous recoveries

was associated with depression. Seven of the nine patients gave a family history of similar problems. Also seven of the patients had recurrent episodes of catatonia and on each occasion associated with the onset of a further period of depression. The two patients who only had a single catatonic illness both presented acutely to another hospital and one died from the effects of acute renal failure. A third patient also died in a psychiatric institute from acute renal failure during the sixth episode of catatonia. The patients who did not present with the acute and rapidly progressive form of the syndrome all had an excellent prognosis, making a full recovery either spontaneously or with ECT.

Organic causes

Table 2 illustrates the five patients who were felt to have an organic cause for their catatonic illness. The precise cause in case 5 remains obscure although it is a reasonable assumption that there was some peri-operative interference, either mechanical or vascular, with brain stem or mid brain region. Four of the five cases suffered from short-lived catatonia of no real significance from the point of view of their neurological management and their recovery was complete and spontaneous. The exception to this was the patient who later developed clinically definite multiple sclerosis who made only a slow recovery with a combination of ECT and phenothiazine therapy.

Table 2

Patient	Sex	Age at onset of catatonia (yr)	Diagnosis	Number of catatonic episodes	Family history	Comments	Outcome
1 JK	F	22	Encephalitis	1	None		Spontaneous recovery
2 PL	F	20	Encephalitis	1	None		Spontaneous recovery
3 SP	F	20	Tuberculous meningitis	1	None		Spontaneous recovery
4 JE	F	29	Multiple sclerosis	2	None	2 episodes of catatonia as first manifestations of clinically definite multiple sclerosis	Slow recovery with ECT and phenothiazines
5 AP	M	24	Post-operative catatonia	1	None	Catatonia occurred post-operatively after removal of cerebellar haemangioblastoma	Spontaneous recovery

*The syndrome of Karl Ludwig Kahlbaum*Table 3 *Idiopathic cases*

Patient	Sex	Age at onset of catatonia (yr)	Diagnosis	Number of catatonic episodes	Family history	Comments	Outcome
1 JA	F	39	Idiopathic familial catatonia	2	Son and daughter (cases 2 & 3) have same condition		Slow improvement with chlorpromazine (ECT not used)
2 DA	M	19	Idiopathic familial catatonia	1	Mother (case 1) & sister (case 3)	In cases 2 and 3 the catatonia was associated with occasional generalised tonic/clonic convulsions. Seizure activity responded to anticonvulsant therapy but not the catatonia	Recovered after 4 ECT sessions over 10 days
3 LA	F	11	Idiopathic familial catatonia	1	Mother (case 1) & brother (case 2)		Recovered after 10 ECT sessions
4 KW	F	24	Idiopathic catatonia	3	None	Has generalised epilepsy. One catatonic episode followed a generalised seizure during pregnancy	All episodes responsive to ECT
5 JS	F	25	Idiopathic catatonia	1	None	Has generalised epilepsy. Dialysed whilst catatonic because of acute renal failure but made complete recovery	Catatonia responded to ECT
6 GS	F	37	Idiopathic/recurrent catatonia familial	8	Daughter (case 7) and brother had similar problem		Episodes responded to ECT
7 CS	F	20	Idiopathic/recurrent catatonia familial	6	Mother (case 6) and uncle		Spontaneous recoveries
8 EJ	F	42	Idiopathic/recurrent catatonia	3	Brother died from "acute lethal" catatonia		Spontaneous recoveries
9 MP	F	41	Idiopathic recurrent catatonia	8	None		Spontaneous recoveries
10 LS	F	51	Idiopathic acute	1	None		Died in acute renal failure

Idiopathic cases

Table 3 illustrates the 10 patients in whom no organic nor psychiatric cause could be determined. It is of interest that there was a marked familial tendency to develop catatonia among this group of patients, six of whom gave a family history of a similar disorder. In four cases the patient suffered from both generalised epilepsy and a recurrent catatonia and in case 4 a fit appeared to trigger one of the catatonic episodes. In cases 2 and 3 the catatonia was associated with occasional generalised tonic/clonic convulsions. The seizure activity responded to anticonvulsant medication but the catatonia only responded to ECT. Once again the acute and rapidly progressing form of the syndrome carried a poor prognosis and one of the two patients who had this presentation died in a psychiatric hospital from acute renal failure whilst the other recovered following dialysis. The remainder of the group had an excellent prognosis, either recovering spontaneously or following ECT. Nine of the 10 cases in this group were female.

Discussion

Our series has emphasised that catatonia is a non-specific entity that has a wide range of organic and psychiatric causes. There has been undue emphasis on the association with schizophrenia which in our experience is an unusual cause. The commonest defined association is affective illness but full neu-

rological and general medical investigation remains necessary to exclude the organic diseases that can underlie the syndrome. Gelenberg⁵ listed many of the causes that had been reported up to 1976 but more recently several more associations have been published. We have reviewed the current literature and Table 4 lists the known causes of the syndrome at the present time.

Our series illustrates that despite full investigation there remains a significant minority of patients who have no defined organic or psychiatric disease. We wish to draw attention to this interesting group of patients. We are not aware of any previous series that has shown such a definite familial tendency to recurrent episodes of catatonia. It is also of interest that four of this group had generalised epilepsy, both during catatonic periods and during periods of normal behaviour. Although this association was described by Kahlbaum in his original monograph^{1,2} it has received scant attention in the modern literature. Thompson and Greenhouse^{4,3} described three cases of catatonia occurring during "petit mal status". Kramer^{4,4} proposed epilepsy in a case of menstrually related catatonic stupor which eventually responded to phenytoin. Shah and Kaplan^{4,5} illustrated an episode of catatonia in a 10-year-old girl whose EEG

Table 4 Causes of catatonia

1. <i>Metabolic disorders</i> —Diabetic keto-acidosis ⁶
—Homocystinuria ⁷
—Hypercalcaemia ⁸
—Acute intermittent porphyria ⁹
—Hereditary coproporphyria ¹⁰
2. <i>Systemic Disorders</i> —Hepatic failure ¹¹
—Renal failure ¹²
3. <i>Toxic agents and drugs</i>
— <i>Central Nervous System Depressants</i> :—alcohol; ¹³ anticonvulsants; ¹⁴ disulphuram; ¹⁵ glutethamide withdrawal; ¹⁶ morphine; ¹⁷ neuroleptics. ^{18–20}
— <i>Central Nervous System Stimulants</i> :—amphetamines/cannabis; ²¹ mescaline; ²² methyl phenidate; ²³ phenylcyclidine. ⁵
— <i>Other Drugs or Toxic Agents</i> :—aspirin; ²⁴ levodopa; ²⁵ fluorinated hydrocarbons; ²⁶ coal gas; ²⁴ steroids. ²⁷
4. <i>Neurological Disorders</i>
— <i>Cerebrovascular Disease</i> :—subarachnoid haemorrhage; ^{28–30} cerebral infarct; ^{31–32} cortical venous thrombosis; ³³ thrombotic thrombocytopenic purpura. ³⁴
— <i>Cerebral Tumours</i> :—various sites. ^{35–39}
— <i>Degenerative conditions</i> Kraepelin's dementia; ⁴⁰ Parkinsonism. ^{41–42}
— <i>Epilepsy</i> . ^{43–45}
— <i>Neurological Infections</i> :—chicken pox; ⁴⁶ encephalitis lethargica; ⁴⁷ herpes encephalitis; ⁴⁸ hydatid disease; ⁴⁹ malaria; ⁵⁰ post immunisation encephalopathy; ⁵¹ subacute sclerosing panencephalitis; ⁵² syphilis; ^{1–2,24} tuberculosis; ^{1–2} typhoid. ⁵³
— <i>Neurological Trauma</i> :—post head injury; ⁵⁴ subdural haematoma. ⁵⁵
— <i>Other Neurological Lesions</i> :—Cerebral lupus erythematosus; ⁵⁶ frontal lobe atrophy; ⁵⁷ hydrocephalus; ⁵⁸ multiple sclerosis; ⁵⁹ narcolepsy; ⁶⁰ tuberose sclerosis ⁶¹
5. <i>Psychiatric Disorders</i> :—schizophrenia; ^{3–4} affective illness; ^{62–63} dissociative states ⁵

showed “high voltage bursts of sharp waves, spikes, polyspikes and diffuse slowing bilaterally”. She responded dramatically to intravenous phenytoin. In one of our patients the catatonia appeared to be triggered by a generalised convulsion but in no case did treatment of the epilepsy affect the catatonic state.

A familial tendency to catatonia is also apparent in patients who have a depressive illness. We are not aware of any similar description in the literature. These families appear to have recurrent, but irregular, periods of depression each associated with catatonia. These people differ from those with “periodic catatonia” as described by Rolv Gjessing²² in that his patients had a more regular and predictable pattern of recurrence without any family history.

In our organic group the association with tuberculous meningitis is most unusual and this appears to be the first definite case described. Kahlbaum^{1–2} does mention tuberculosis in some of his cases but this may have been a secondary phenomenon due to long periods of debilitation. Multiple sclerosis also appears to be a rare cause of catatonia. The only other report⁵⁹ is that of a 29 year old woman who presented with drawn, retarded and cataleptic and who later developed symptoms and signs diagnostic of multiple sclerosis.

In terms of prognosis our patients appear to divide into 2 groups. The 4 patients who presented with an acute and rapidly progressive form of catatonia had a high mortality and 3 of them died in acute renal failure. This acute form of the illness, often associated with the name of Stauder⁶⁶ and termed acute lethal catatonia, requires intensive treatment, particularly correction of dehydration, maintenance of renal function, withdrawal of neuroleptics, prevention of respi-

ratory and cardiovascular complications and cooling to prevent death from hyperthermia.⁶⁷ There is a degree of clinical overlap between acute lethal catatonia and the neuroleptic malignant syndrome which was originally described by Delay and Deniker.⁶⁸ This syndrome represents an idiosyncratic response to antipsychotic agents particularly the butyrophenones and phenothiazines. It is usually characterised by akinesia, rigidity, hyperthermia, autonomic instability and often stupor and coma. Other catatonic features can be found in the early stages. The pathogenesis is thought to be related to dopamine receptor blockade.⁶⁹ Successful treatment of the syndrome with levodopa/carbidopa or bromocriptine in combination with dantrolene^{67–70} gives rise to optimism for the treatment of acute lethal catatonia. If the acute illness is treated quickly then the long term prognosis ought to be good and no patient ought to succumb to the effects of acute renal failure.

The patients who did not present acutely all had an excellent outcome with the exception of a single patient with schizophrenia. In most cases the recovery was spontaneous or occurred after a course of ECT. Our patients tended to improve after a single shock and usually only required four to six shocks over a 1 to 2 week period to come completely out of the catatonia. We would agree with Waziri⁶⁵ that ECT is the “safest and most effective treatment in the majority of catatonic states.” ECT need not be restricted to the affective or idiopathic groups but can be used when an organic cause is found for the syndrome. Breakey and Kala⁵³ found that all 12 patients with typhoid catatonia made a rapid recovery with ECT, requiring only three or four sessions for remission. We would also recommend that ECT be used in the treatment of

acute lethal catatonia. Although we have no personal experience of the use of ECT in this variety of catatonia we see no reason why recovery should not be as rapid as in the less acute condition. Rapid employment of ECT may reduce the mortality and morbidity at present associated with acute catatonia.

The historical association of catatonia with chronic schizophrenia has often engendered a feeling of therapeutic nihilism. Perhaps a new name for the syndrome will act as a reminder that there are many treatable causes. We suggest that catatonia be known as Kahlbaum's syndrome to honour the man who so accurately described the disorder over a century ago.

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M P Barnes, M Saunders, T J Walls, I Saunders and C A Kirk

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