

10 Late paraphrenia

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Historical context • *Psychiatric signs and symptoms* • *Classification*
• *Prevalence and incidence* • *Risk factors* • *Divisions* • *Management*
• *Conclusion*

Historical context

The term 'paranoid' has its origin in the ancient Greek words 'paranoia' and 'paranoeo', which were commonly used to express the idea of madness or being out of one's mind. In the 18th century the term was incorporated into medical terminology to describe a number of mental disorders that included mood disorders and dementia. By the end of the 19th century, paranoia was part of the medical vocabulary, although controversy surrounded its use and definition.

Kraepelin introduced the term 'paraphrenia', and in 1913, described it as a condition which occurred in patients with well-preserved personalities, no disturbance of will and good affective response. He identified four forms:

- (a) Systematica: an insidious development of delusions of persecution and exaltation.
- (b) Expansiva: exuberant ideas of grandiosity and mild excitement.
- (c) Confabulaloria: falsifications of memories.
- (d) Phantastica: extraordinary, incoherent and changeable delusional ideas.

The first systematic description of paranoid features with onset in later life was published under the title of *Involuntional Paranoia* (Kleist, 1913). Kleist speculated on the possible contribution of organic factors to the development of psychotic symptoms, and concluded that involuntional paranoia was unlikely to be caused by a primary degenerative or vascular dementia. He remarked that the clinical features were very similar to those described by Kraepelin as paraphrenia.

In 1952, Roth & Morrisey described a group of elderly patients with a well-organised system of paranoid delusions in whom signs of organic dementia, sustained confusion or affective disorder were absent. The authors emphasised that the disorder developed in the setting of a well-preserved intellect and personality, was often primary in character, and was usually associated with hallucinations, passivity phenomena and other volitional disturbances.

A few years later, Fish (1960) challenged the use of the term late paraphrenia. He believed that the condition Roth had described represented nothing more than a senile form of schizophrenia. Fish advocated the use of Leonhard's confusing classification of schizophrenia (Fish, 1958) and, by an inexplicable paradox, chose to allocate his patients into six different subtypes of the very diagnosis he had just rejected: affect-laden, hypochondriacal, fantastic, incoherent, phonemic and confabulatory.

Since the early 1960s, discussions about the nature of late paraphrenia and related disorders have focused on two conflicting views:

- (a) Late paraphrenia is nothing more than the expression of schizophrenia in the elderly (Fish, 1960; Gold, 1984; Grahame, 1984).
- (b) The paranoid symptoms of late life are genetically different from schizophrenia and arise from the complex interaction of various pathogenic factors associated with old age (Funding, 1961; Post, 1966; Herbert & Jacobson, 1967; Almeida *et al*, 1992).

Psychiatric signs and symptoms

Delusions and hallucinations

Delusions and hallucinations usually dominate the clinical picture. Kay & Roth (1961) offered a vivid description:

“Neighbours, landlords, or relatives are implicated in plots to be rid of the patients, or to annoy or interfere with them through jealousy or simply for amusement... Patients feel drugged, hypnotised, have their thoughts read, their minds or bodies worked upon by rays, machines or electricity, complain that they are spied upon, can get no privacy in thought or act... (auditory hallucinations) consist of threatening, accusing, commanding or cajoling voices, jeering commentaries, screams, shouts for help, obscene words and songs, music, loud bangs, rappings, shots or explosions... their thoughts are repeated aloud... God, spirits, distant or deceased relatives, or most often, jealous, hostile neighbours are held responsible (for these phenomena)”.

A wide range of delusions have been observed in late-onset paranoid states (Box 10.1). Persecutory delusions are possibly the most consistent and frequent; they are found in around 90% of patients (Almeida *et al*, 1995a) (Table 10.1). Auditory hallucinations are detected in about three-quarters of patients. Visual, olfactory, tactile and somatic hallucinations are less common, but not rare. Visual hallucinations, for instance, have been observed in up to 60% of patients. Other frequent symptoms include first rank symptoms of Schneider (Post, 1966; Holden, 1987). A similar type and frequency distribution of psychotic symptoms has been found in schizophrenia of early onset (Andreasen, 1990a), which seems to

Box 10.1 Types of delusions found in late paraphrenia

Persecution
Reference
Misidentification
Control
Hypochondriasis
Grandiosity
Gender
Religion

indicate that late paraphrenia and schizophrenia are the same condition. However, there is more to schizophrenia than delusions and hallucinations.

Other schizophrenic symptoms

ICD-10 (World Health Organization, 1992) and DSM-IV (American Psychiatric Association, 1994) include thought disorder, catatonic symptoms and negative symptoms as important clinical features of schizophrenia. Thought disorder is present in up to 60% of early-onset people with schizophrenia (Wing *et al*, 1974; Andreasen, 1990*b*; Menezes, 1992), and catatonic symptoms are observed in up to a quarter of patients (Creer & Wing, 1975; Andreasen, 1990*b*). In addition, typical negative symptoms such as lack of speech inflection, reduced speech output, reduced facial expression, reduced gestures and affective blunting are found in up to 85% of early-onset people with schizophrenia (Andreasen *et al*, 1990).

These findings contrast with late paraphrenia. Thought disorder, catatonic symptoms and negative symptoms are extremely uncommon

Table 10.1 Psychiatric symptoms in late paraphrenia

	%
Persecutory delusions	83
Auditory hallucinations	77
First rank symptoms	46
Delusions of reference	33
Delusions about mind reading	17
Visual hallucinations	13
Somatic/tactile hallucinations	12
Somatic delusions	9
Grandiose delusions	6
Thought broadcast	4
Olfactory hallucinations	4
Delusions of sin or guilt	2

in late-onset cases. Even in the few instances when such symptoms are reported, they are not described as severe (Kay & Roth, 1961; Pearlson *et al*, 1989; Almeida *et al*, 1995a). By emphasising the similarities between the delusions and hallucinations of late paraphrenia and schizophrenia, sight may be lost of important psychopathological differences. In fact, the two disorders show very different symptomatological profiles.

Other psychiatric symptoms

There are few references to other psychiatric symptoms in the late paraphrenia literature. Almeida *et al* (1995a) found irritability, social unease, loss of interest and concentration, self-neglect and obsessive features associated with the typical psychotic symptoms of late paraphrenia.

Classification

The classification of the paranoid states of late life has been the subject of an intense, and sometimes emotive, controversy (Levy *et al*, 1987; Grahame, 1988). ICD-10 (Boxes 10.2 and 10.3) and DSM-III-R (American Psychiatric Association, 1987) introduced further problems by not including a diagnosis of late paraphrenia. In ICD-10 some people with late paraphrenia are diagnosed with schizophrenia, while others are

Box 10.2 Summary of ICD-10 diagnostic guidelines for schizophrenia (World Health Organization, 1992)

The presence for one month or more, of at least one very clear symptom (and usually two or more if less clear cut) belonging to (a) to (d) or symptoms from at least two groups of (e) to (h)

- (a) thought echo, insertion, withdrawal or broadcasting
- (b) delusions of passivity, delusional perception
- (c) hallucinatory voices giving a running commentary, discussing the patient or coming from some part of the body
- (d) persistent delusions of other kinds such as superhuman abilities
- (e) persistent hallucinations in any modality
- (f) thought disorder
- (g) catatonic behaviour
- (h) negative symptoms such as marked apathy, paucity of speech, blunting of emotion, lowering social performance
- (i) a significant and consistent change in quality of personal behaviour with loss of interest and social withdrawal

Box 10.3 Summary of ICD-10 diagnostic guidelines for delusional disorder (World Health Organization, 1992)

Delusions are the most conspicuous or only clinical characteristic, present for at least three months
 Depression may be present but only intermittently
 No evidence of brain disease
 No or only occasional auditory hallucinations
 No history of schizophrenic symptoms

classified as delusional disorder, schizoaffective disorder, or other types of psychoses (Rabins *et al.*, 1984; Quintal *et al.*, 1991).

There is little evidence (Quintal *et al.*, 1991; Howard *et al.*, 1994; Almeida *et al.*, 1995a) to suggest that people with late paraphrenia are better classified under the headings 'persistent delusional disorder' or 'schizophrenia' (Grahame, 1984; Harris & Jeste, 1988; Jeste, 1993). The use of the diagnosis of delusional disorder seems inappropriate because most people with late paraphrenia display symptoms such as prominent hallucinations. Similarly, there are limitations to the use of the diagnosis of schizophrenia:

- (a) There is poor agreement on how to define it.
- (b) The diagnosis of schizophrenia fails to take into account the associated factors of late paraphrenia.
- (c) It is unlikely that the neurodevelopmental basis postulated for schizophrenia (Weinberger, 1987; Bloom, 1993) plays an important pathogenic role in late-onset cases.

Prevalence and incidence

People with late paraphrenia represent approximately 10% of the elderly population of psychiatric hospitals. The reported prevalence of the disorder among the elderly living in the community ranges from 0.1 to 4% and its incidence has been estimated to be between 10–26 per 100 000 per year. On average only 1.5% of those diagnosed with schizophrenia have an onset of illness after the age of 60, in contrast to 23 and 16% of patients with other paranoid states and reactive psychoses (Almeida *et al.*, 1992).

Risk factors

It is difficult to think about lung cancer without immediately associating it with smoking. Similarly, a number of risk factors are important in late paraphrenia (Box 10.4).

Box 10.4 Factors associated with late paraphrenia

Female gender
 Hearing loss
 Visual impairment
 Social isolation
 Brain disease
 Not marrying (may be a weaker factor)
 Family history

Gender

Late paraphrenia is more common in women than men. Ratios have been reported between three to one, and 20 to one (Kay *et al*, 1964; Herbert & Jacobson, 1967). Compared to men the onset of schizophrenia in women is delayed by three to four years (Bland, 1977; Keith *et al*, 1991; Häfner *et al*, 1993). The later preponderance of women is unlikely to be due merely to some protective factor delaying the onset of illness in this population. Indeed, Castle & Murray (1991) showed that the incidence of schizophrenic symptoms in women after the age of 55 clearly exceeds the frequency expected if the incidence curve had been simply shifted to the right. In other words, early (with a predominance of men) and very late-onset cases (with a clear predominance of women) may represent distinct diseases.

Theories to explain the later onset of schizophrenia in women include:

- (a) There is a separate disorder genetically related to mood disorders, rather than schizophrenia (Castle & Murray, 1991), which explains why these patients show a better prognosis and response to treatment (Hogarty *et al*, 1974).
- (b) The condition is associated with a concurrent decline of oestrogen levels, and a relative excess of dopamine D₂ receptors (Pearlson & Rabins, 1988; Seeman & Lang, 1990).
- (c) Psychosocial factors, such as better use of coping behaviour strategies and social support schemes, delay the onset of schizophrenic symptoms among women (Riecher *et al*, 1990).

Sensory deficits

Hearing impairment has been experimentally (Zimbardo *et al*, 1981) and clinically (Moore, 1981) associated with the development of paranoid symptoms. Almeida *et al* (1995*b*) estimated that the risk of hearing impairment among people with late paraphrenia was four times greater than that of age- and gender-matched controls. Cooper *et al* (1974) showed that this hearing loss is usually caused by conductive, rather than

degenerative, mechanisms. Late paraphrenia was associated with deafness of early onset, long duration and profound auditory loss (Cooper, 1976). Unfortunately, interpreting these associations is far from simple because most elderly individuals with hearing problems do not become psychotic (Corbin & Eastwood, 1986). Moreover, hearing impairment in the elderly has been more frequently associated with depression and decreased self-sufficiency in daily living activities (Carabellese *et al*, 1993) than with paranoid symptoms.

Cooper & Porter (1976) suggested that hearing deficits may have a pathogenic role by reinforcing a pre-existing tendency to social isolation, withdrawal and suspiciousness. However, it is not suspiciousness but auditory hallucinations that have been most frequently connected to hearing impairment (Corbin & Eastwood, 1986). The symptomatological relief reported by some of these patients after the fitting of a hearing aid (Khan *et al*, 1988; Almeida *et al*, 1993) highlights the contribution made by deafness to the production of auditory hallucinations. David & Lucas (1992) propose a cognitive model in which auditory hallucinations result from a failure to recognise internal auditory input at the auditory analysis system level. This model suggests that a defective auditory analysis system might lead to impaired discrimination of spoken material. This has been confirmed by the preliminary work of Stein & Thienhaus (1993).

The diagnosis of late paraphrenia has also been associated with visual impairment (Kay & Roth, 1961; Post, 1966; Herbert & Jacobson, 1967). Major ocular pathology (predominantly cataracts) was found in over half of people with late paraphrenia, significantly more than in elderly depressive controls (Cooper & Porter, 1976). However, the mechanisms by which visual impairment could produce psychotic symptoms are still unclear.

Social isolation

Kay & Roth (1961) pointed out that people with late paraphrenia were socially isolated, and that 40% of them lived alone. A more systematic assessment of social isolation was reported by Almeida *et al* (1995*b*) who evaluated the frequency of contacts with friends during the six months prior to the interview. They found that 79% of people with late paraphrenia were socially isolated, compared with 18% of age- and gender-matched controls. The role of social isolation in late paraphrenia is uncertain, but there have been suggestions that deafness, deviant personality, and few surviving relatives may contribute (Kay & Roth, 1961; Post, 1966; Herbert & Jacobson, 1967).

Brain disease

Evidence for an association between late paraphrenia and brain dysfunction comes from clinical, neurological, psychological, imaging and pathological studies.

Clinical

The concept of late paraphrenia emerged as an attempt to address the contribution of brain disease to the late-onset psychotic states (Roth & Morrisey, 1952). Kay & Roth (1961) found that 21% of their patients showed evidence of organic disease, although organic factors had bearing on the later course of the illness in only 5% of subjects.

Neurological

Almeida *et al* (1995*b*) found two main types of neurological signs in late paraphrenia: (a) abnormal and dyskinetic movements, which were associated with the use of neuroleptics; and (b) neurological soft signs.

Neurological soft signs do not have the same localising power as hard signs (Cadet *et al*, 1986), although their presence can often be related to impaired performance on cognitive tests (Förstl *et al*, 1992*a*; Liddle *et al*, 1993), and may indicate the presence of brain disease or dysfunction (Förstl *et al*, 1992*a*; Manschreck, 1986).

Psychological

Naguib & Levy (1987) found deficits in a group of people with late paraphrenia on the Mental Test Score and a mild deterioration on scores at 3.7 year follow-up, although they remained above the cut-off point for dementia (Hymas *et al*, 1989). Almeida *et al* (1995*c*) found that performance on tests assessing memory was fairly well preserved, although executive functions (frontal tests) and general mental skills (Wechsler, 1981; Roth *et al*, 1988) were worse than matched controls.

Imaging

A number of computerised tomography and magnetic resonance imaging studies have looked at the association between late paraphrenia and brain pathology (Almeida *et al*, 1992) (see Chapter 12). Their results can be summarised by three main findings:

- (a) Late life psychosis is often associated with cerebrovascular pathology, either in the form of white matter changes or infarcts.
- (b) People with late paraphrenia with first rank symptoms of Schneider, have more cortical atrophy than patients without first rank symptoms.
- (c) Patients with late life psychosis show mild ventricular enlargement compared to controls. Ventricular enlargement has been observed in late paraphrenia and late-onset schizophrenia (Naguib & Levy, 1987; Rabins *et al*, 1987; Burns *et al*, 1989; Krull *et al*, 1991; Howard *et al*, 1992).

Pathological

Blessed *et al* (1968) published the only available quantitative neuropathological study of late paraphrenia. They examined the brains of five people with late paraphrenia and found that the mean number of plaque counts was much lower than in people with dementia, and not significantly different from those observed in elderly with depression or controls.

Marriage

Some reports suggested that people with late paraphrenia were more likely to be unmarried than elderly people with depression or normal age matched controls (Post, 1966; Herbert & Jacobson, 1967). Furthermore, married patients with late paraphrenia have a lower fertility rate than control groups (Kay & Roth, 1961; Herbert & Jacobson, 1967). More recent investigations suggest that these associations are not as consistent as female gender, hearing impairment, social isolation and brain damage (Rabins *et al*, 1984; Pearlson *et al*, 1989; Almeida *et al*, 1995*b*).

Family history

A family history of schizophreniform disorder is another factor frequently associated with the diagnosis of late paraphrenia. Kay (1972) reported that the risk of developing schizophrenia among first degree relatives of people with late paraphrenia was 3.4%, a rate intermediate between that described for the general population (less than 1%) and for the families of young people with schizophrenia (5.8%). Naguib *et al* (1987) used the human leucocytic antigen system to look at the association between late paraphrenia and genetic markers. There was no obvious relationship between the disorder and the antigens investigated although the authors proposed the HLA-B37 as a possible marker candidate for late paraphrenia. More recently Howard *et al* (1995) reported no difference in ApoE4 frequency rates between people with late paraphrenia and controls.

Divisions

A number of attempts have been made to subdivide the paranoid states of late life. Kay & Roth (1961) allocated patients to three different groups according to their symptoms and aetiology. Other subdivisions have included those of Post (1966, 1980) and Holden (1987). Howard *et al* (1994) divided a group of 101 people with late paraphrenia using ICD-10 criteria and a cluster analysis of symptoms. They found poor agreement between the two groupings (Table 10.2).

More recently, Almeida *et al* (1995*d*) divided people with late paraphrenia into two groups by a cluster analysis of cognitive test scores.

Table 10.2 Divisions of patients with late paraphrenia

	%
<i>ICD-10 diagnoses</i>	
Paranoid schizophrenia	61
Delusional disorder	31
Schizoaffective disorder	8
<i>Cluster analysis of symptoms</i>	
Schneider's first rank symptoms	35
Prominent auditory hallucinations, few delusional experiences	27
Older age at onset, marked delusions, but few hallucinations	38

These groups need to be tested against clinical outcomes, functional neuroimaging and neuropathology:

- (a) A more 'functional' group which showed cognitive deficits restricted to executive functions and a high prevalence of positive psychotic symptoms, including first rank symptoms of Schneider.
- (b) A more 'organic' group which exhibited generalised cognitive decline and a relatively high frequency of neurological signs.

Management

Patients with late paraphrenia rarely seek help spontaneously, but may request action from their relatives, police or other authorities against their persecutors. Many are maintained in the community with support from families, friends and social workers, but the effectiveness of this form of management has not been evaluated. Doctors tend to see these patients only when these measures fail and behaviour becomes disruptive.

These patients rarely agree to attend a psychiatric appointment in an out-patient clinic, and a home visit is often necessary. A successful first medical contact may prove crucial in overall treatment. The psychiatrist should try to establish a good rapport with the patient, and be sympathetic to his or her complaints. They must also ensure that late paraphrenia is the correct diagnosis (investigate cognitive decline, neurological signs or symptoms, associated affective symptoms, etc.), and further interviews should be organised to establish the patient's good compliance with the therapeutic strategy. Antipsychotic medication can be rationalised for the patient as a way of lessening distress. Unfortunately, compulsory admission may be necessary with non-compliant subjects. In these cases the medical team should try to regain confidence in order to guarantee an effective future management.

Box 10.5 Management

Home visit
Establish rapport
Exclude other illnesses
Develop a therapeutic alliance
Reduce isolation
Antipsychotics
Compulsory admission sometimes necessary

Antipsychotics

The use of antipsychotics for the treatment of late paraphrenia is widely accepted, although no controlled trials have been reported. The risk of side-effects (mainly extrapyramidal) favours the use of oral medication, although Howard & Levy (1992) have suggested that depot intramuscular medicine may improve compliance and reduce the required antipsychotic dose. Post (1966) looked at patients who had received 10–30 mg of trifluoperazine or 40–500 mg of thioridazine per day. Nine per cent showed no response, 31% showed some signs of improvement and 60% presented a complete response to treatment. Less optimistic results were reported by Rabins *et al* (1984) and Pearlson *et al* (1989), who found that 15 and 24%, respectively, failed to improve after neuroleptic treatment. Howard & Levy (1992) reported that after at least three months of treatment, 42% of patients showed no response, 31% improved partially and 27% presented symptomatological remission. Several factors may be involved with failure to respond to the medication side-effects, and the presence of concomitant organic factors.

Outcome

Post (1966) suggested a number of good prognostic features: immediate response to treatment; development of insight; younger age; and being married. Psychological, social and occupational support are also important for a good outcome. Early interactions are important. Untreated patients may pursue an unremitting chronic course and once the symptoms have fully developed they are unlikely to change substantially until the patient's death.

Conclusion

The aetiology of late paraphrenia is complex, and includes the interaction of ageing, female gender, social isolation, hearing impairment, subtle

brain lesions and cognitive decline. There is evidence that the division of late paraphrenia into delusional disorder and schizophrenia is unsatisfactory. The term 'late paraphrenia' may still be the best option available. However, increasing evidence from neuroimaging and neuropsychological studies may help subdivide late paraphrenia into a functional group and an organic group. Few studies have looked at treatment for this condition but there is evidence that neuroleptics benefit a majority of patients. Attempts should always be made to form a therapeutic alliance with the patient. A multi-disciplinary approach offers the best hope for successful treatment and outcome.

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