British Journal of Psychiatry (1986), **148**, 463–465

Brief Reports

Late-Onset Folie Simultanée in a Pair of Monozygotic Twins

A case-report is presented of folie simultanée in a pair of female monozygotic twins with onset at the remarkable age of 81. The twins demonstrated no evidence of dementia or other psychopathology apart from delusions. Folie simultanée in monozygotic twins provides a model example of the potential role of genotype-environment correlation in the etiology of psychiatric illness.

Although rarely observed, cases of *folie à deux* in twins have played an important role in speculations on the aetiology of this intriguing condition (Gralnick, 1942; Adler & Magruder, 1946; Oatman, 1942; Rioux, 1963). In an early review of the syndrome, Tuke (1888) described a separate sub-type of 'double insanity' for twins, as follows:

> It really seemed as if there was some sort of sympathy between them, or it would perhaps be more correct to say that their constitutions, having originated and been built up at the same time, and under precisely the same conditions, were so nearly identical, that the existing causes of insanity produced when operating on them, the same results.
>
> (Quoted in Enoch & Trethowan, 1979)

Although *folie á deux* is a far from common syndrome, its occurrence in the agèd is rarer still. In a thorough review, McNiel *et al* (1972) found only 17 cases in the English literature where one or both of the patients was over the age of 65 at evaluation: one of these was a pair of twins.

Because of its rarity, we here present a case of the *folie simultanée* sub-form of *folie à deux,* occurring in a pair of female monozygotic twins, with onset at the age of 81.

Case Report

The twins were born on September 1902 in India, and each weighed about 2 kilograms. Twin A was older by 10 minutes.

Their childhood were described as 'happy'. They were repeatedly mistaken for one another by their teachers, friends and parents, and both twins felt that as children they were 'like two peas in a pod'. Twin B was more tomboyish and tended to be the leader of the pair. They both completed school in India, went to Europe for 'finishing school' and then returned to India.

Twin A married at the age of 28 to a man in banking. The marriage, which was described as happy, produced one boy and one girl who are both still living. The couple emigrated to Ireland in 1949. The girl developed psychiatric problems, the nature of which are uncertain. The husband died in 1976, at which time twin A moved in with her widowed twin sister.

Twin B taught in a school for some time, nursed her dying parents then came to Ireland in 1949, at the same time as her married twin sister. A few years later she met a man 12 years her senior, and married him at the age of 56. They had no children, and the husband died in the early 1970s.

Between 1976 and 1983, the twins lived together in their own house and were popular and well-liked by their neighbours, who reported that they seemed 'perfectly normal'. In 1983, because of increasing physical incapacity, the twins moved into an old people's home, where they shared an apartment. The twins denied experiencing any symptoms indicative of anxiety disorder, affective illness or psychosis before 1983. Excessive alcohol intake was also denied.

In May 1984, twin B heard dogs yelping outside her window at night. She brought this to the attention of twin A. After hearing the sound for several nights, they both became convinced that the dogs were being tortured by being tied to the wheels of moving cars. Twin A called the local police and the animal protection society but obtained no satisfactory response.

Two months later, the twins were both walking down a corridor in the home when they heard a fire alarm ring. 'Behind' the sound of the fire alarm, they both 'heard' a woman screaming: twin A said that she heard her yelling 'Take it away! Please don't hurt me!'. That afternoon, a woman in the home was found dead and, contrary to local custom, was buried the next day. Both twins became convinced that she had been murdered and that the fire alarm had been sounded to cover up her screams. They decided that a conspiracy existed between the staff of the home, and relatives of certain of the occupants to murder the occupants for their inheritance. When twin A attended the funeral, she was sure that she was being watched by 'four lads on motorcycles'.

Over the next few weeks, the twins realised that their knowledge of the plot put their lives in danger. They decided that their phone was bugged and their food was being poisoned. They felt that they were being tortured by static electricity which they could 'sense' in their beds at night. They began sleeping in chairs and/or wearing their coats to bed in an effort to protect themselves from the 'electricity'. When asked how they came to this series of realisations, twin B replied 'We figured it out together'. In early September 1984, they moved out of the original home. They then lived in a series of other establishments: each time they would stay for several days and then accuse

the staff of poisoning them. This behaviour, plus repeated calls to the police to request protection, led to their admission to hospital on 18 September 1984.

At admission, their appearance matched their stated age, and was neat and tidy. Their speech was well organised and their general demeanour normal. They were very similar in physical appearance, voice, mannerisms and general personality, although twin A was somewhat more kyphotic and twin B slightly heavier. Their affect was well modulated, without evidence of depression or euphoria. When they were discussing subjects other than their psychosis, no psychopathology was evident in either twin. They described the psychosis as noted above, and the descriptions obtained separately from the two twins agreed on all main facts. Apart from the misperceptions noted above, the twins reported no hallucinations; and apart from the 'electricity' which both twins insisted they 'sensed' rather than 'felt with the skin', the twins denied any passivity or other Schneiderian symptoms. Both had insight that others perceived their beliefs as aberrant. Neither twin felt that their delusions could possibly be products of their imagination, nor did they feel that one of them had experienced those things first and then had to convince the other of their reality.

On routine cognitive and neuropsychological testing (including Raven's Progressive Matrices, Benton Visual Retention Test, and specific tests of short-term memory) both twins performed in the 'normal'—'high normal' range fo their age. No evidence of a dementing process was evident in either twin.

Hospital treatment started with modest doses of thioridazine (75–150 mg/day), which had no significant effect on their delusional beliefs: they continued to feel that their beds were 'electrified' and their food poisoned. The nursing staff felt that twin B seemed more often to be the leader in their delusional beliefs, but not always. By the time of their discharge to relatives in December 1984, twin A was marginally the more adamant in her delusional beliefs, although both twins continued to insist that their food was being poisoned.

HLA typing showed that the twins had identical alleles at both the A and B loci.

Discussion

There is little doubt that twins A and B were monozygotic and presented with *folie simultanèe*. Given that both members of the pair stated that they were mistaken for each other in childhood by family and friends, that they were 'as alike as two peas in a pod', and that they were identical on HLA typing, the probability of monozygosity is in excess of 95% (Cederlof *et al*, 1961; Torgersen, 1979). It was not possible to identify either twin as the 'dominant' member of the pair who had developed the delusions first and then convinced the other of their veracity. As far as we could tell, the twins truly developed their psychosis together.

This case fits the pattern of late-onset *folie à deux* described by McNiel *et al* (1972). It is typical of such cases that our patients were sisters who had lived together for years prior to the onset of the illness; and compared with cases of earlier onset, *folie à deux* in the elderly more often presents as *folie simultanée* (McNiel *et al*, 1972). Furthermore, McNiel *et al* (1972) found that organic brain deficits rarely play an important role in late-onset *folie à deux*.

The twins were older than any of the sibling pairs reported by McNiel *et al* (1972). The most similar case in the literature appears to be one of female twins aged 66 described by Gralnick (1942), in which zygosity was uncertain, the disorder was clearly of the *folie communiquée* type, and the dominant twin first developed psychotic symptoms at the age of 49. Thus the present case appears to represent the oldest age of onset of *folie à deux* in a twin pair yet reported, by over 30 years.

Although no firm conclusions can be reached on the basis of a single case, these twins may provide a model example of the action of 'genotype–environment correlation' in Man (Eaves, 1976). These twins had lived together for many years and had come to depend greatly on each other. In their later years, each twin played a dominant role in the social environment of the co-twin. Since they were genetically identical, any genetically influenced tendency toward delusional thinking in one twin would tend to be reinforced by the other; so while neither twin may have been sufficiently predisposed to become delusional on their own, the two together created a 'positive feedback loop' and developed a shared psychosis. Consistent with these speculations is the repeated observation that *folie à deux* occurs much more commonly among genetically related individuals (Gralnick, 1942; Rioux, 1963) and that in unrelated pairs with *folie à deux*, both members commonly have a familial loading for psychosis (Scharfetter, 1972). In a social species such as Man, where blood relatives usually form a major part of an individual's social environment, genotype–environment correlation probably plays a significant role in preventing or precipitating psychiatric illness.

Acknowledgements

This study was supported in part by the Scottish Rite Schizophrenia Research Program, Northern Masonic Jurisdication, USA, and the Department of Mental Health and Mental Retardation of the Commonwealth of Virginia. The neuropsychological testing of the twins was performed by Dr Michael Gallagher. Dr Ruth Loane reviewed the manuscript.

References

ADLER, A. & MAGRUDER, W. W. (1946) Folie à deux in identical twins treated with electroshock therapy. *Journal of Nervous and Mental Disease*, **103**, 181–186.

CEDERLOF, R., FRIBERG, L., JONSSON, E. & KAIJ, L. (1961) Studies on similarity diagnosis in twins with the aid of mailed questionnaires. *Acta Geneticae Medicae et Gemellologiae*, **11**, 338–362.

EAVES, L. J. (1976) A model for sibling effects in man. *Heredity*, **36**, 205–214.

ENOCH, M. D. & TRETHOWAN, W. H. (1979) *Uncommon Psychiatric Syndromes*. Bristol: Wright.

GRALNICK, A. (1942) Folie à deux—The psychosis of association. *Psychiatric Quarterly* **16**, 230–263.

MCNIEL, J. N., VERWOERDT, A. & PEAK, D. (1972) Folie à deux in the aged: Review and case report of role reversal. *Journal of the American Geriatrics Society*, **20**, 316–323.

OATMAN, J. G. (1942) Folie à deux: Report of a case in identical twins. *American Journal of Psychiatry*, **98**, 842–845.

RIOUX, B. (1963) A review of folie à deux, the psychosis of association. *Psychiatric Quarterly*, **37**, 405–428.

SCHARFETTER, C. (1972) Studies of heredity in symbiotic psychoses. *International Journal of Mental Health*, **1**, 116–123.

TORGERSEN, S. (1979) The determination of twin zygosity by means of mailed questionnaire. *Acta Geneticae Medicae et Gemellologiae*, **28**, 225–236.

TUKE, D. H. (1888) Folie à deux. *Brain*, **10**, 408–421.

*Kenneth S. Kendler, MD, *Associate Professor, Departments of Psychiatry and Human Genetics, Medical College of Virginia/Virginia Commonwealth University, P.O. Box 710, Richmond, Virginia 23298, USA.*

Gillian Robinson, BSoc Sci, *Research Assistant, Medico-Social Research Board, Dublin, Ireland.*

Mary McGuire, MB, BCh, MRCPsych, *Consultant Psychiatrist, St Patrick's Hospital, Castlerea, Ireland.*

Mary P. Spellman, MB, BCh, MRCPsych, *Medical Superintendent, St Patrick's Hospital, Castlerea, Ireland.*

*Correspondence

(Accepted 23 May 1985)

British Journal of Psychiatry (1986), **148**, 465–467

Evolution of Variants of the Munchausen Syndrome

With the identification of basic criteria by DSM-III (1980) for Munchausen Syndrome (Asher, 1951), it could have been possible to predict that adaptive variations existed and would emerge continuously. By their very nature, fictitious illnesses must be adaptive to changing circumstances, or they would be too obvious.

The Ganser Syndrome (Ganser, 1898), the Gaslight Phenomenon (Smith & Sinanan, 1972), Munchausen Syndrome (Asher, 1951), Feigned Bereavement (Snowden *et al*, 1978), Artefactual Illness to Attract Medical Attention (Carney, 1980), The Munchausen Syndrome as a Psychiatric Condition (Cheng & Hummel, 1978) and The Extended Munchausen Syndrome (Black, 1981) are in reality descriptions of fictitious illnesses responding to and identified by different circumstances.

The case reported here provides evidence that the basic criteria for fictitious illnesses need reconsideration. Furthermore, the probable range of variations in the presentation of Munchausen Syndrome is likely to develop in parallel with the evolution of medical and social services. In the example below, the patient transferred the pathology of the Munchausen Syndrome away from hospital to community services, and in so doing, endangered the health of the paramedical staff involved.

Case report

Mrs A was referred by her family doctor at the suggestion of the police, following her presentation of a self-forged prescription for Pethidine to a new chemist.

She stated that when she was pregnant in 1974, seven years before, she developed a tubercular gland in her neck, requiring an operation, followed by daily intra-muscular injections of Streptomycin, which were administered by two Public Health Nurses (PHN) for over one year; that she failed to recover completely, and that a visiting London Professor of Tropical Medicine discovered that she was suffering from Kala-Azar. He prescribed injections to be administered frequently by the PHNs for over one and a half years. Failure to respond resulted in the