Barbiturate automatism—myth or malady?

R. C. B. AITKEN M.D., M.R.C.P.(Ed.), D.P.M.

Senior Lecturer, University Department of Psychiatry, Royal Edinburgh Hospital, Edinburgh, 10

Summary

Patients admitted to the Regional Poisoning Treatment Centre at the Royal Infirmary, Edinburgh, were assessed to identify cases of drug *automatism* and those who denied the act of selfpoisoning.

Only two out of 994 instances of poisoning could be attributed to barbiturate automatism. The case histories of these patients are reported.

Twenty-nine patients on thirty-one admissions denied the act of self-poisoning and clinical data on this sample are reviewed.

It is suggested that there is insufficient evidence for accepting barbiturate automatism as a clinical entity and that the failure of these patients to remember the ingestion of more than a therapeutic dose is the result of psychogenic defence mechanisms.

It is concluded that the use of the term *automatism* contributes nothing to the management of patients poisoned with these drugs.

Introduction

The term *automatism* was 'borrowed' in 1934 by Richards, a lecturer in Forensic Medicine, to explain three cases of poisoning by barbiturates. Each patient on recovering consciousness claimed to remember taking only one, or at the most, two doses though obviously more must have been taken. Richards suggested that 'the knowledge of the need for another tablet persists while the memory is so affected by the drug that the patient does not realize that he has already satisfied the need and automatically repeats the dose at intervals'.

In England and Wales in 1964, 506 people were certified by doctors or after corners' inquests as having died of *accidental* barbiturate poisoning (Registrar General's Statistical Review of England and Wales for the year 1964); in the same year in the United States, 478 died similarly (McCarthy, 1967). In both groups 99% of the patients were over the age of 15 years. It is difficult to conceive

*Formerly Registrar, Regional Poisoning Treatment Centre, Royal Infirmary, Edinburgh.

A. T. PROUDFOOT* B.Sc., M.B., M.R.C.P.(Ed.) Senior Registrar in Medicine, Royal Infirmary, Edinburgh, 3

how *accidental* poisoning with barbiturates was possible, unless some phenomenon such as drug automatism existed or the assessment of motivation was wrong.

Long (1960) reported that he could find no observation in the literature which substantiated the existence of barbiturate automatism as a clinical entity. His opinion was that 'to affirm the soundness of Richards' conclusions on the meagre evidence of his three cases is an appeal to credulity which ought not to be attempted in an intelligent society'. Litman *et al.* (1963) also failed to confirm the hypothesis of automatism as a cause of suicide or serious, non-fatal poisoning by barbiturates. They pointed out that the patients who described behaviour somewhat resembling automatism had usually ingested relatively non-lethal amounts of drugs.

On the other hand, Ettlinger & Flordh (1955) found that 28% of 500 cases of attempted suicide were due to 'serial consumption' of drugs, a phenomenon which appears identical with automatism. Jansson (1961) found that no less than 25% of 488 cases of attempted suicide by various drugs were due to automatism. Lieberman (1963) added further support for the existence of barbiturate automatism.

Despite the inherent weakness of the original description and the fact that only extremes of opinion seem to be held regarding the role of automatism in barbiturate overdosage and death, the phenomenon continues to be widely quoted (Backett, 1965; Goodman & Gilman, 1965; Leading Article, 1965; Meyler, 1966; Berger, 1967; Today's Drugs, 1968).

This report is an attempt to elucidate whether barbiturate automatism is a myth or a malady by assessing for that purpose poisoned patients admitted to the Royal Infirmary, Edinburgh during 1967.

Selection of cases

The organization and admission policy of the Regional Poisoning Treatment Centre has already

been described (Kessel, 1965). There has been a substantial increase in the number of patients admitted in recent years (Aitken & Carstairs, 1968), though the number poisoned by barbiturates has remained remarkably constant (Aitken, Buglass & Kreitman, 1969).

Of 994 admissions in 1967, 295 were on account of barbiturate poisoning. The vast majority of the patients were seen by at least one of the authors, whose interest in identifying cases of automatism was appreciated by their colleagues. The psychiatrist indicated on a code-sheet whether the patient denied the act of self-poisoning or claimed it was due to automatism.

Sixty-six of all admissions (7%) were identified supposedly as having denied the act and nineteen (2%) as having claimed automatism. The clinical records of these eighty-five cases were scrutinized by both authors and it was discovered that fifty-four had been miscoded. This was mainly for one of three reasons:

- (1) The *motive* of attempted suicide rather than the act was denied (thirty-eight admissions).
- (2) Probably no excess of drug other than alcohol had been taken (eleven admissions).
- (3) The possibility of the poisoning being *accidental* could not be excluded (five admissions).

The authors agreed that the act of self-poisoning was denied despite evidence to the contrary on thirty-one admissions by twenty-nine patients (twelve males, seventeen females); each of these admissions was then allotted to one of three categories:

- (1) *Partial denial*: the patient admitted having taken some drug but less than the likely amount after assessment of circumstantial, clinical and biochemical evidence (fifteen admissions).
- (2) Complete denial: the patient denied the ingestion of poison, despite definite evidence to the contrary (fourteen admissions).
- (3) Automatism: the patient denied taking more than a therapeutic dose though he accepted that he was poisoned and that a greater amount of drug must have been taken by himself (two admissions).

Case reports of two patients claiming automatism Case A

Female: aged 38 years. The patient, a nurse, living in a hospital, was admitted after being found unrousable in bed. She was unconscious but responded to minimally painful stimulation. No other abnormality was found on examination apart from mild hypotension. She regained consciousness after 2 hr and recovery was uneventful. The blood barbiturate level on admission was 1.1 mg/100 ml and there was an iron-deficiency anaemia (haemoglobin 9.2 g/ 100 ml).

In explanation of the overdose she said: 'I must have taken repeated doses thinking I had not already taken them'. On specific questioning she admitted knowledge of the concept of barbiturate automatism.

The patient was second in a sibship of five and after leaving school at the age of 14 was in unskilled employment until she married at the age of 25. Her husband was a merchant seaman and their 8year courtship was stormy because of her attraction to a man she met while doing voluntary work in hospital. Sexual adjustment with her husband was initially satisfactory but she did not conceive. After 3 years the marriage deteriorated and she took an overdose of barbiturate at age 31 after discovering her husband in adultery. They separated and were divorced 3 years later. During this time she trained as a nurse.

The patient never felt close to her family and though she lived with her parents while her husband was at sea she felt inhibited and unable to express her feelings in their company. There was considerable disturbance in relationships among members of the family but no history of overt psychiatric disorder.

During the week prior to her admission a young married patient to whom she had become particularly attached died and she said that an uncle had also died. The latter statement was retracted when it was found to be untrue and the patient admitted that she had used it to explain her distress at the younger man's death. She also said that she had again become preoccupied by the death of a young brother 12 years previously; she had felt responsible and had been blamed for his death by her mother as she had both bought the bicycle he was riding and had sent him on the errand on which he died.

The psychiatric diagnosis made later on the day of admission was mild depression in an anxious, histrionic personality, who was often dependent on barbiturates at times of stress. She was discharged to outpatient supervision but defaulted after two visits.

Six weeks later she was again admitted with mild barbiturate poisoning which she attempted to explain on the same basis. Later, however, she remembered having taken further doses of barbiturates on this occasion. After a period of psychiatric inpatient care, she was much improved in mood and was also in closer rapport with her family.

Case B

Male: aged 44 years. This patient was first admitted with barbiturate poisoning in 1965 following desertion by his wife after 21 years of marriage. While unconscious he sustained a full-thickness burn of the lower leg which resulted in chronic osteomyelitis. Despite the fact that he was soon fully mobile and despite every encouragement by a medical social worker to help him overcome his disability and obtain employment he did not work after this incident. He lived in lodgings, drawing sickness benefit and though he had six children he had lost contact with them. The youngest two were in the care of the local authority. In the past he had held many unskilled jobs but none for more than 3 years. In 1957 he had a partial gastrectomy and was drinking heavily before his first overdose.

In the week prior to his admission in 1967 he had been staying with his sister and her family in Glasgow; he enjoyed this visit as he had felt miserable and particularly lonely for several weeks beforehand. On the day following his return to his lodgings in Edinburgh, he was admitted drowsy with a blood barbiturate level of 1.4 mg/100 ml.

When he recovered he said that he could not remember taking more than his usual dose of pentobarbitone though he admitted 'I must have taken them'. He denied any suicidal ideation with the reply 'I have too much to live for'. He volunteered that he was overcome by loneliness after returning to his lodgings, and when his attention was directed to his social difficulties he continued 'I couldn't say I did it intentionally—but it could have been because I was so low'.

It was considered that he had an inadequate psychopathic personality with mild depression. His distress was attributed to chronic physical ill-health and interpersonal problems due to his dependency needs.

He failed to attend the psychiatric follow-up clinic after two visits but continued to contact the medical social worker, to whom he reported that he felt as lonely as ever, and still unable to work.

Denial of self-poisoning act

Those who believe in the existence of drug automatism as a clinical entity may consider that we have excluded further cases from identification because of prejudice when reading case-sheets. If this were so, it is likely that such cases would have been misclassified into the other 'denial' categories, rather than that our system of detection of cases omitted to find them altogether. Indeed the case histories of the two patients whose poisoning might have been due to barbiturate automatism seemed to have much in common with those of the patients who denied the act of self-poisoning. Hence the sample of twenty-nine patients who on thirty-one occasions denied having ingested an overdose of drugs was compared with the whole group of patients reviewed by the psychiatrists at the Regional Poisoning Treatment Centre in the same year with a view

to identifying any features specific to denial. No distinction was made between those classified as 'partial denial' and those as 'complete denial' as no difference in their clinical features was apparent.

Ecological data

Age: the average age of the patients who denied the act of self-poisoning was greater than that of the whole group. Fifty-eight per cent were over 40 years in contrast to only 34% of the whole group ($\chi^2 =$ 9.31; d.f.=1; P<0.01).

Sex: the male-female ratio in the sample was 1:1.4 which was similar to the whole group.

Social class: the distribution of occupation in the categories of the Registrar General's classification was the same as that of the whole group; 50% belonged to Classes IV and V.

Civil state: the civil state of the patients who denied having taken an overdose did not differ significantly from those in the whole group. Forty-eight per cent were married (39% of the whole group), and 17% widowed, separated, divorced or cohabitating (23% of the whole group).

Past psychiatric history

Previous episodes of self-poisoning: 49% of the patients who denied the act of self-poisoning had been admitted to the hospital before because of drug overdosage compared with 36% of the whole group of admission. This difference was not significant.

Previous psychiatric treatment: 32% of the sample and 38% of the whole group had received previously in-patient psychiatric care. The difference was not significant.

Danger to life

Method of poisoning: 48% of the patients who denied self-poisoning had ingested barbiturates compared with 30% of all admissions ($\chi^2 = 4.48$; d.f.=1; P<0.05).

Depth of coma on admission: 26% of the sample made no response or only minimal response to painful stimuli (Grades 3 and 4; Matthew & Lawson, 1966) compared with only 12% of the patients in the whole group ($\chi^2 = 6.86$; d.f.=1; P<0.01).

Psychiatric morbidity

Diagnosis: the distribution of psychiatric diagnoses in the patients who denied taking an overdose was similar to that in the whole group; 58% showed evidence of some form of personality disorder psychoneurosis, sociopathy, drug or alcohol addiction. Affective disorder was present in 32%. Though 10% of the patients in the sample were epileptic compared with 5% of the whole group the difference was not significant. Seven per cent of the patients who denied self-poisoning were classified as 'no psychiatric abnormality' which is similar to the proportion in the whole group and reflects the threshold of rating of psychiatric morbidity currently in use in the hospital.

Further management: 23% of the sample were transferred to psychiatric in-patient care; this proportion did not differ significantly from that in the whole group (28%). Thirty-nine per cent of the sample and 33% of the whole group were offered a follow-up appointment at a psychiatric outpatient clinic.

Conclusion

Those who deny the act of self-poisoning despite evidence to the contrary are more likely to be older and more of them to be more deeply unconscious due to barbiturates. The distribution of their psychiatric diagnoses is unlikely to differ from those who admit the act. More of them will have attempted suicide before. The same proportion will have been treated previously as in-patients at a psychiatric hospital, and require transfer on this occasion.

Discussion

In drug automatism the patient admits to having ingested at least one therapeutic dose, but is unable to remember taking more. He accepts that his clinical condition on admission to hospital was due to poisoning, that more drug must have been taken. and that it must have been taken by himself. One is left with the picture of a semi-drugged person reaching for sleeping tablets from the bedside table. quite unaware of the potential disastrous consequence of his actions. Indeed such a scene was included in a film shown at the exhibition Medicine with Care organized by the Pharmaceutical Society of Great Britain in 1967. The danger of keeping barbiturates near the bedside has been emphasized by several authors (Backett, 1965; McCarthy, 1967; Today's Drugs, 1968). The concept of automatism is firmly entrenched in the minds of the medical profession and general public; it provides a credible explanation of drug overdosage which is socially acceptable.

In the present study only two out of 994 instances of adult poisoning could be even remotely attributed to drug automatism. This very low incidence is in marked contrast to the figures of 28% and 25%reported by Scandinavian observers (Ettlinger & Flordh, 1955; Jansson, 1961). The explanation of this difference may lie partly in the definition of the term 'drug automatism'. Jansson (1962) defines his automatism cases as 'cases of poisoning where the person involved gradually has consumed an overdose of hypnotics in order to get to sleep without any intention to commit suicide'. This definition differs in two important respects from the original description by Richards (1934), used in the present study. Firstly, it does not necessarily imply that the patient has amnesia for the ingestion of subsequent doses of the drug, and secondly it introduces the motive for having ingested the overdose. The fact that by Jansson's definition the patients did not intend suicide does not make their behaviour *automatism*. It would be more accurate to describe it by the term 'self-poisoning', introduced by Kessel (1965) because of the difficulty in interpreting motive, and which is so seldom 'attempted suicide'.

There is no doubt that small doses of hypnotic drugs such as barbiturates can lead to clouding of consciousness. When this mental state is attributable to organic illness it is usually associated with impaired grasp of activity in the environment, liability to perceptual disturbance, and lack of constructive behaviour; there is also a tendency to vagueness of thought and incoherence of speech with fluctuating disorientation due to patchy diffuse amnesia. Despite this the patient with drug automatism is considered to have been capable of sufficient purposive behaviour to have ingested repeated small doses of drugs, and not to have revealed any other abnormality of cognitive function. The feasibility of such an explanation must be open to considerable doubt.

The question at issue is whether the ingestion of subsequent quantities of drugs is not remembered because it was not registered due to clouding of consciousness, or is not recalled because of psychogenic defences of repression or suppression. In both our patients, the only abnormality of thinking was a highly specific amnesia for the crucial events of overdosage. This suggests to us that such amnesia is unlikely to be due to pharmacological effects of drugs, but to pre-existing psychopathology.

One could readily understand the development of psychogenic amnesia in both our cases. Not only had Case A heard of barbiturate automatism but admission of deliberate self-poisoning would have threatened her esteem in particular among colleagues. Case B had unequivocal evidence of personality disorder. The psychiatric morbidity of these cases did not differ from many of the patients who denied the ingestion of their overdose or from many in the whole group of poisoned patients.

Should we have misclassified into the 'denial' categories further cases of drug automatism attributable only to sedation, evidence of less psychiatric morbidity amongst them would be expected. As at least as many of those who denied the act of self-poisoning had pre-existing psychopathology, this explanation does not seem tenable.

Self-poisoning can be regarded as a plea for help (Kessel, 1965) and this applied to our patients who claimed automatism; indeed in Case A the response to the first plea was inadequate and a further admission with self-poisoning was necessary before a period of in-patient psychiatric treatment was arranged. A diagnosis of barbiturate automatism, like that of hysteria, suggests that the underlying psychopathology need not be explored further, the term being sufficient explanation for the behaviour. In our experience it is abundantly clear that this is not the case. Moreover, Jansson (1962) found no significant difference in the incidence of repetition of overdose between those cases thought to be due to automatism and those classified as serious attempts at suicide. We are therefore of the opinion that the term automatism should be abandoned, and the concept discarded as an explanation of accidental barbiturate poisoning.

In appraising the present status of drug automatism we feel that it must be easier to describe a new syndrome than to prove that a rare condition diagnosed retrospectively does not exist. Let us rest the case that barbiturate automatism is a myth and not a malady with a quotation from Slater (1965) writing about *hysteria*:

'The malady of the wandering womb began as a myth, and a myth it yet survives. But, like all unwarranted beliefs which still attract credence, it is dangerous. The diagnosis of (barbiturate automatism) is a disguise for ignorance and a fertile source of clinical error. It is in fact not only a delusion but also a snare.'

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