Transient global amnesia and functional retrograde amnesia: contrasting examples of episodic memory loss

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SUMMARY

We studied 11 patients with transient global amnesia (TGA) and ten patients with functional retrograde amnesia (FRA). Patients with TGA had a uniform clinical picture: a severe, relatively isolated amnesic syndrome that started suddenly, persisted for 4–12 h, and then gradually improved to essentially normal over the next 12–24 h. During the episode, the patients had severe anterograde amnesia for verbal and non-verbal material and retrograde amnesia that typically covered at least two decades. Thirty hours to 42 days after the episode, the patients had recovered completely and performed normally on tests of anterograde and retrograde amnesia. By contrast, patients with FRA had a sudden onset of memory problems that were characterized by severe retrograde amnesia without associated anterograde amnesia and with a clinical presentation that otherwise varied considerably. The episodes persisted from several weeks to more than two years, and some of the patients had not recovered at the time of our last contact with them. The uniform clinical picture of TGA and the variable clinical picture of FRA presumably reflect their respective neurologic ('organic') and psychogenic ('non-organic') aetiologies.

1. TRANSIENT GLOBAL AMNESIA

Transient global amnesia (TGA) is a well-defined neurological disorder characterized by a temporary, relatively isolated amnesic syndrome. TGA was described independently by Bender (1956) and Guyotat & Courjon (1956), and also by Fisher & Adams (1958), who named the disorder. Reviews by Caplan (1985), Kritchevsky (1989), Hodges (1991) and Frederiks (1993) have summarized the neurological and neuropsychological features of this condition.

The patient with TGA has sudden onset of severe memory impairment, including both anterograde and retrograde amnesia, which lasts for at least several hours and resolves gradually over several hours to a day (Fisher & Adams 1964; Kritchevsky 1987). Most episodes last 2–12 h (Caplan 1985; Miller *et al.* 1987). Clinical examination during TGA suggests a relatively isolated amnesic syndrome (Donaldson 1985; Gordon & Marin 1979; Patten 1971; Shuttleworth & Wise 1973). The patient's neurologic examination is otherwise normal, without evidence of visual, motor or somatosensory system dysfunction. The patient does not lose personal identity. The TGA patient is often aware that something is the matter, and may complain spontaneously of memory impairment.

TGA generally occurs in persons over the age of 50 years (Caplan 1985; Fisher 1982; Hodges & Warlow 1990; Miller *et al.* 1987; Zorzon *et al.* 1995) and has an estimated incidence in persons older than 50 years of 23.5–32 per 100 000 per year (Koski & Marttila 1990; Miller *et al.* 1987). About one-third of TGA attacks are precipitated by physical or psychological stress (Fisher 1982; Miller *et al.* 1987), though the majority of episodes have no clear precipitating factor. Most patients with TGA have only a single attack. The recurrence rate is 2.5–5% per year for at least five years after the initial episode (Hinge *et al.* 1986; Miller *et al.* 1987; Nausieda & Sherman 1979; Shuping *et al.* 1980; Zorzon *et al.* 1995).

After TGA, patients remain unable to recall the period of TGA and, occasionally, they exhibit a period of permanent retrograde amnesia covering several hours to several days before the onset of TGA (Kritchevsky 1989). There is little evidence that TGA patients have an increased incidence of additional permanent memory impairment or other cognitive deficits following their attack. On the one hand, 27 patients who were tested neuropsychologically during and after TGA dramatically improved their test scores after TGA (Evans *et al.* 1993; Goldenberg *et al.* 1991; patient 2 of Hodges 1994; Hodges & Ward 1989; case 3 of Kazui *et al.* 1995; Kritchevsky & Squire 1989; Kritchevsky *et al.* 1988; Lin *et al.* 1993; Meador *et al.* 1988; Regard & Landis 1984; Stillhard *et al.* 1990; Stracciari *et al.* 1987; Wilson *et al.* 1980). On the other hand, mild verbal memory problems may have persisted in three patients (Gallassi *et al.* 1986; patient 1 of Hodges 1994; and case 4 of Kazui *et al.* 1995) who were studied 25 h to eight

Table 1. *Characteristics of 11 patients with transient global amnesia*[a]

age (yrs)	65 (56–77)
education (yrs)	13 (7–16)
gender	7 men, 4 women
testing (h)	6.4 (2–11)

[a]The values for age and education are means and ranges. The value for testing indicates how long after the onset of transient global amnesia formal testing began.

weeks after TGA. Additionally, comparisons of patients 1–39 months after TGA with age- and IQ-matched control subjects found that the after-TGA patients were mildly impaired on some tests of memory and attention and intact on several other tests (Gallassi *et al*. 1993; Hodges & Oxbury 1990). One complication of these between-group studies is that any finding of mild impairment in patients after TGA might reflect an impairment that was already present in the patients even before TGA.

(*a*) *Neuropsychological findings*

Thirty patients have been studied with formal neuropsychological tests during and after an episode of TGA (Evans *et al*. 1993; Gallassi *et al*. 1986; Goldenberg *et al*. 1991; Hodges 1994; Hodges & Ward 1989; Kazui *et al*. 1995; Kritchevsky & Squire 1989; Kritchevsky *et al*. 1988; Lin *et al*. 1993; Meador *et al*. 1988; Regard & Landis 1984; Stillhard *et al*. 1990; Stracciari *et al*. 1987; Wilson *et al*. 1980). Of these patients, many were presented as individual case reports, and had been studied with varied tests of anterograde and, sometimes, retrograde amnesia.

Table 1 describes 11 patients whom we have systematically tested during an episode of TGA. All patients were tested between 2 and 11 h after the onset of TGA and again 30 h to 42 days after the onset of TGA when the clinical signs of TGA were no longer apparent. All patients were amnesic during the initial testing, although in four of the 11 cases family members stated that the memory problems had begun to improve by the time testing began. Three other patients noticeably improved during the testing session itself. The results summarized in this section have been reported in more detail elsewhere (Kritchevsky 1989; Kritchevsky & Squire 1989; Kritchevsky *et al*. 1988). For comparison purposes, we also tested ten neurologically intact subjects on the tests of anterograde amnesia. These subjects averaged 69 years of age (65 for the TGA patients) and 12 years of education (13 for the TGA patients).

We assessed anterograde amnesia for verbal material with a story recall test and non-verbal material with a diagram recall test. Two forms of each of these tests were employed, one during TGA and the other after TGA. We administered the two forms in one order to six of the patients and in the opposite order to the remaining five patients. For the story recall test, subjects were read a short prose passage (Gilbert *et al*. 1968) with the instruction, 'When I am finished I want you to tell me as much of it as you can remember'.

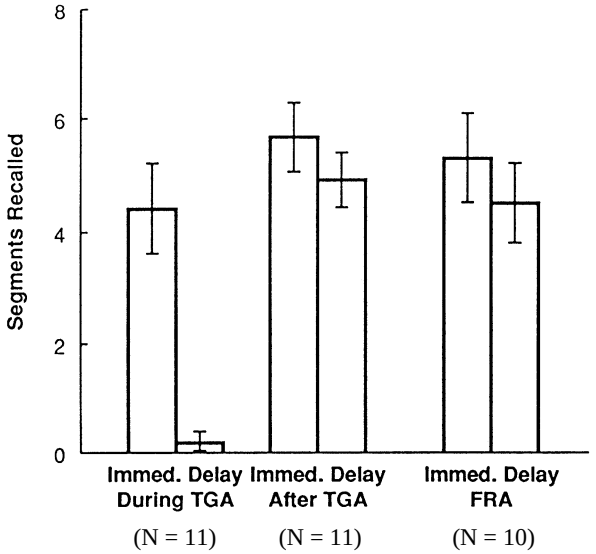

Figure 1. Story recall by patients during and after transient global amnesia (TGA) and by patients during functional retrograde amnesia (FRA). Recall was tested immediately after presentation of the story (Immed.) and again after a delay of 10–20 min (Delay). Brackets show standard errors of the mean.

Immediately thereafter, and again after a delay of 10–20 min, subjects attempted to recall the passage. The score was the number of story segments correctly recalled (maximum score = 19 or 21 segments). For the diagram recall test, subjects were asked to copy either the Rey-Osterrieth (Osterrieth 1944) or the Taylor (Milner & Teuber 1968) figure. After a 10–20 min delay, and without forewarning, we asked them to draw the diagram from memory. The maximum score was 36 points.

All 11 patients had severe anterograde amnesia for both verbal and non-verbal material. Delayed recall of verbal material on the story recall test was impaired during TGA, both in comparison with performance after TGA ($t_{10} = 8.9$, $p < 0.01$; figure 1) and also in comparison with the performance of control subjects ($t_{19} = 9.6$, $p < 0.01$). Performance after TGA was not noticeably different from the performance of control subjects ($p > 0.1$).

The ability to draw a diagram from memory was also markedly impaired during TGA both in comparison with performance after TGA ($t_{10} = 7.0$, $p < 0.01$; figure 2) and in comparison with the performance of control subjects ($t_{19} = 4.0$, $p < 0.01$). Again, performance after TGA was not noticeably different from the performance of control subjects ($p > 0.1$).

Several additional points about anterograde amnesia and TGA deserve mention. First, the test scores of the TGA patients were similar to scores previously obtained by a group of well-studied patients with chronic amnesia (Squire & Shimamura 1986). Those patients required supervisory care because of the severity of their memory impairment. Second, there appeared to be a correlation between severity of anterograde amnesia and time since onset of TGA (Kritchevsky & Squire 1989). The severity of the amnesia was less as time passed. Third, there was no

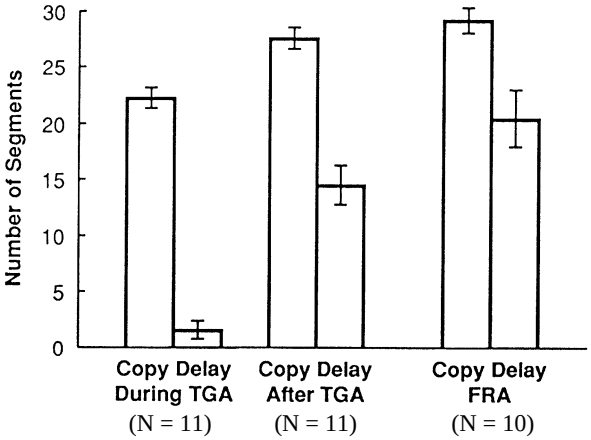

Figure 2. Copy and recall of a diagram by patients during and after transient global amnesia (TGA) and by patients during functional retrograde amnesia (FRA). Reconstruction of the figure was attempted 10–20 min after copying it. Brackets show standard errors of the mean.

evidence for material-specific, or partial amnesia. None of the patients had a noticeable disparity between the degree of verbal and nonverbal memory impairment. Finally, the findings from our 11 TGA patients are in agreement with other reports in the literature (Evans *et al*. 1993; Gallassi *et al*. 1986; Goldenberg *et al*. 1991; Hodges 1994; Hodges & Ward 1989; Kazui *et al*. 1995; Lin *et al*. 1993; Meador *et al*. 1988; Regard & Landis 1984; Stillhard *et al*. 1990; Stracciari *et al*. 1987; Wilson *et al*. 1980).

Retrograde amnesia was assessed with two tests, a test of public events and a test of autobiographical memory. Memory for public events was assessed in six of the patients with recall and recognition tests, which consisted of questions about public events that had occurred from 1950–85 (Squire *et al*. 1989). We presented the recall test orally. We then presented the recognition test in a four-alternative, multiple-choice format to be completed by the subject. Two alternate forms of the recall and recognition tests were available. Each form consisted of 9–15 items from each of the four decades covered by the tests. During TGA we gave three patients one form of the recall and recognition tests, and three the other form. After TGA we administered both forms to all patients. The tests were given in 1987.

We assessed retrograde amnesia for autobiographical memory in four of the 11 patients. A list of ten common nouns (e.g. bird, clock and ship) was presented one word at a time with the instruction to recall a specific personal event from any time in the past that involved the stimulus word (Crovitz & Schiffman 1974). Subjects were asked to describe the memory in the greatest detail possible, and then to date it as accurately as possible. When recall was not clearly specific to time and place, the examiner probed to elicit the most specific memory possible. Probing consisted of encouraging subjects to be more specific about what had already been stated, or suggesting examples of specific responses, so that subjects would better understand what was being requested. Responses were scored on a 0 to 3 scale, both before and after probing, with 3 representing a well-formed episodic memory (Zola-

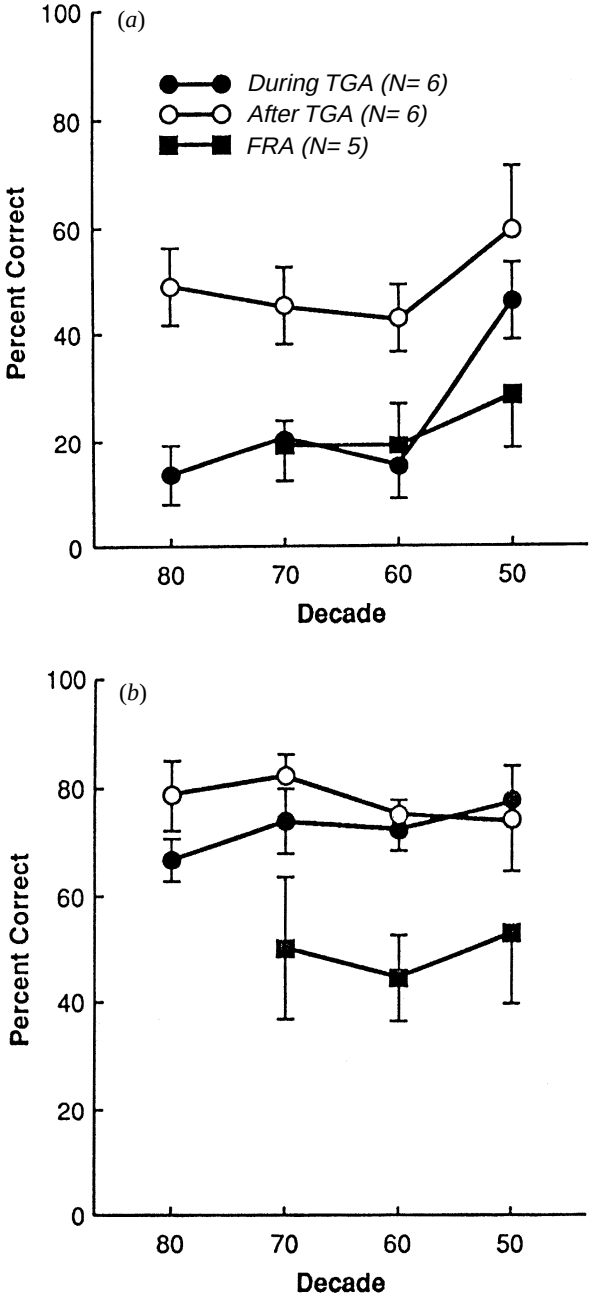

Figure 3. Performance of six patients during and after transient global amnesia (TGA) and five patients during functional retrograde amnesia (FRA) on recall and recognition of public events. Patients with TGA were tested in 1987 and were asked questions about public events that had occurred from 1950 to 1985. Patients with FRA were tested in 1982–85 and were asked questions about public events that had occurred from 1950–79 (*a*). Patients then attempted to recognize the correct answers to the same questions on a four-alternative, multiple-choice test (*b*). Only four of the five patients with FRA were administered the recall test. Brackets show standard errors of the mean.

Morgan *et al*. 1983). The maximum score was 30 points. Two different lists of words were used, one during TGA and one after TGA. The lists were presented in one order for two patients, and in the opposite order for two other patients.

Performance on the public events recall test is shown in figure 3*a*. The patients exhibited a temporally graded

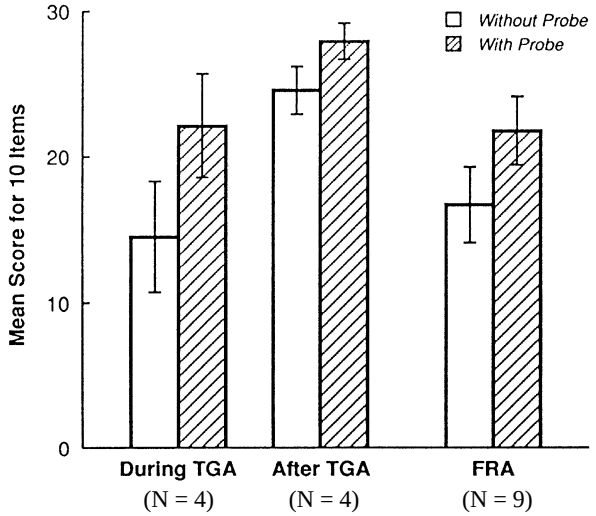

Figure 4. Performance of four patients during and after transient global amnesia (TGA) and nine patients during functional retrograde amnesia (FRA) on a ten-item test of past autobiographical memory. Patients were asked to recollect ten distinct episodes in response to ten cue words. Responses were scored (0 to 3) both before (without probe) and after (with probe) attempts by the examiner to elicit more specific recollections. Brackets show standard errors of the mean.

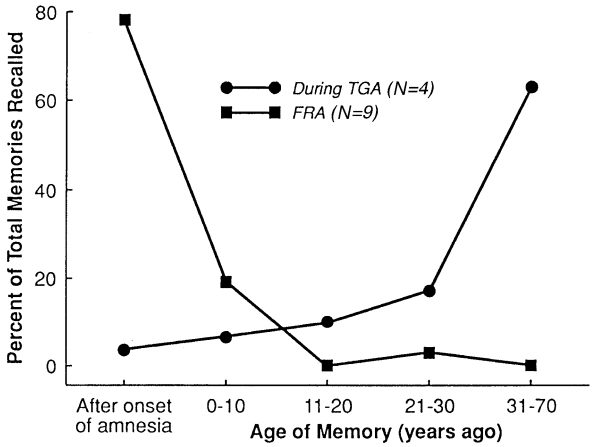

Figure 5. Percentage of memories recalled for the indicated past time periods for four patients during transient global amnesia (TGA) and for nine patients during functional retrograde amnesia (FRA). The analysis was based only on those responses given a maximum score of 3 on the ten-item test of past autobiographical memory.

retrograde amnesia covering at least 20 years prior to TGA onset. Recall during TGA was impaired in comparison with recall after TGA for the 1980s ($t_5 = 3.9$, $p < 0.05$), the 1970s ($t_5 = 4.3$, $p < 0.01$) and the 1960s ($t_5 = 4.6$, $p < 0.01$). Recall for the 1950s was similar during and after TGA ($p > 0.1$). Performance on the recognition test is shown in figure 3*b*. Recognition during TGA was impaired in comparison with recognition after TGA for the 1980s ($t_5 = 2.5$, $p = 0.05$), but not for the other time periods ($p > 0.1$).

Figure 4 shows performance on the test of past autobiographical memory. Patients performed more poorly during TGA than after TGA. When the examiner probed for more detailed and specific recall, performance improved. Without probes, performance was impaired during TGA compared with after TGA ($t_3 = 4.1$, $p < 0.05$), but with probes, performance during TGA was not measurably affected ($t_3 = 2.2$, $p > 0.1$). Thus, during TGA patients had difficulty recollecting full and detailed autobiographical memories unless they had the benefit of probing by the examiner. Indeed, performance during TGA may have been impaired even in the with-probe condition, if a 'ceiling effect' were operating in the after-TGA condition.

Examination of the dates of well-formed memories indicated that during TGA (figure 5), approximately 90% of memories were drawn from a time period greater than ten years before the episode of TGA. This distribution of memories is abnormal, in that normal aged subjects typically draw more than 30% of memories from the most recent ten years (MacKinnon & Squire 1989).

Other investigators have also found neuropsychological evidence of retrograde amnesia in TGA patients. Hodges & Ward (1989), found a temporally graded retrograde amnesia on a famous faces recall test in three of five patients studied during TGA. These three patients performed normally on the test after recovery from TGA. Three patients were also given a test of past autobiographical memory similar to the test we described above. The scores of two of these patients were the same as the scores of our patients during TGA. The third patient performed essentially normally. Evans *et al.* (1993) also found a temporally graded retrograde amnesia in one TGA patient who was studied with the Autobiographical Memory Interview (Kopelman *et al.* 1989) and with a famous faces recall test.

One final point deserves emphasis. The 11 TGA patients we studied had some difficulty copying a diagram. Their copy scores were lower during TGA than after recovery from TGA (22.1 versus 27.6, maximum = 36; $t_{10} = 4.2$, $p < 0.01$) and were lower than the average score obtained by the control subjects (26.7; $t_{19} = 3.1$, $p < 0.01$). This finding provides evidence for cognitive impairment during TGA that is separate from, and in addition to, the amnesic syndrome.

(*b*) *Neuroanatomy and aetiology of TGA*

The neuroanatomical substrate of TGA is not known. However, because patients with TGA have severe anterograde amnesia for verbal and non-verbal material during the episode, they are likely to be suffering from dysfunction of bilateral medial temporal or medial diencephalic structures important for memory. Moreover, because patients with TGA have extensive retrograde amnesia, the dysfunction must involve more than just the CA1 region of the hippocampus if medial temporal lobe structures are involved. (Rempel-Clower *et al.* 1996).

The aetiology of TGA is also unknown. TGA has a time-course similar to the time-course of a transient

ischaemic attack, and TGA occurs most frequently in patients who have increased risk for cerebral vascular disease because of their age. Nonetheless, the preponderance of evidence suggests that TGA patients do not have an increased incidence of atherosclerotic risk factors and that patients with TGA are not at increased risk for having transient ischaemic attacks or strokes subsequent to the TGA (Hodges & Warlow 1990; Zorzon *et al*. 1995). It has been proposed that TGA may be due to a reversible, 'benign', migraine-like ischaemic phenomenon of bilateral medial temporal or medial diencephalic structures important for memory (Caplan 1985; see discussion in Kritchevsky 1989).

2. FUNCTIONAL RETROGRADE AMNESIA

Functional retrograde amnesia (FRA), also known as 'hysterical amnesia', 'psychogenic amnesia' or 'generalized dissociative amnesia' (American Psychiatric Association 1994) is a well-established psychiatric condition (see Campodonico & Rediess 1996; Kopelman *et al*. 1994; Pratt 1977; Schacter & Kihlstrom 1989). FRA is the memory disorder most often popularized in literature and film, and is the syndrome that lay persons typically seem to regard as 'amnesia'. The typical patient with FRA is said to have the sudden onset of severe retrograde amnesia without clinically significant anterograde amnesia. The retrograde amnesia may affect most of the memories that occurred prior to the onset of memory problems, or may affect only the patient's memories for a 'localized' or 'limited' time period before the onset of the amnesia. Episodes of FRA are considered to be associated usually with severe psychological stress. Finally, many patients with FRA improve over days, weeks or months, with or without psychological treatment (Kaszniak *et al*. 1988; case M.M. of Lucchelli *et al*. 1995; Schacter *et al*. 1982), but other patients exhibit persisting FRA (De Renzi *et al*. 1995; Kopelman *et al*. 1994). The incidence of FRA is not known but it would appear to be much less common than TGA.

Since 1982, we have systematically studied ten patients with FRA (table 2). A more complete report describing neurological and neuropsychological findings will appear elsewhere. The patients were significantly younger than the patients whom we studied with TGA, but the two patient groups had similar educational backgrounds and a similar distribution of gender. Nine of the FRA patients complained of loss of all memories from before the onset of amnesia. The remaining patient complained of loss of all memories within the three-year time period immediately preceding the onset of amnesia. All patients reported normal memory for events that had occurred since the onset of amnesia.

Eight of the ten patients with FRA had an abnormal premorbid psychological status. For example, patients had histories that included severe alcohol abuse, previous conversion symptoms, and diagnoses of anxiety disorder, paranoid schizophrenia, depression and antisocial personality disorder. Of the other two patients, one had no known premorbid psychological condition, and the other's premorbid psychological status could not be determined. One or more potential precipitating factors were present in eight of the ten patients, such as intoxication with alcohol, mild closed head injury, or involvement in illegal or criminal activity.

Table 2. *Characteristics of 10 patients with functional retrograde amnesia*[a]

age (yrs)	37 (28–54)
education (yrs)	14 (12–18)
gender	8 men, 2 women
testing	7 at 1–9 days after onset and 3 at 2–9 months after onset

[a]The values for age and education are the means and ranges. The values for testing indicate how long after the onset of functional retrograde amnesia the patients were tested.

Seven of the ten patients had neurobehavioural abnormalities in addition to loss of memory for premorbid facts and events. These abnormalities included depression, bilateral leg weakness that had been present for ten months and was thought to be psychogenic, left-sided weakness that started at the same time as the FRA and was thought to be psychogenic, anomia, loss of the ability to read and write, and loss of the ability to use common devices such as a telephone and a microwave oven. One patient reported that he had to relearn the English language during the first week of FRA by reading the dictionary.

Only one of the ten patients fully recovered from FRA. Another patient's FRA had decreased and involved only the eight-month period before onset of amnesia at our last report of her. Five patients had significant and persistent FRA when we last saw them between ten days and one year following the onset of amnesia. Two additional patients had persistent retrograde amnesia 23 months and 30 months after the onset of amnesia, respectively, and had established new personalities at the time of onset of FRA. Neither of these patients had any other history to support a diagnosis of multiple personality disorder. Finally, one of the patients being described here admitted to us nine days after the onset of amnesia that he had malingered the amnesia in order to obtain admittance to the hospital.

(*a*) *Neuropsychological findings*

All ten patients were tested while they had severe retrograde amnesia. Follow-up testing was not possible, either because patients could not be located or because they exhibited no significant recovery from retrograde amnesia. Seven patients were tested between one and nine days after the onset of amnesia, and three patients were tested between two and nine months after the onset of amnesia.

All patients performed normally on the tests of anterograde amnesia for verbal and non-verbal material (figures 1 and 2). This finding was consistent with their own self-reports, as well as with the bedside

neurobehavioural findings of severe retrograde amnesia in the absence of anterograde amnesia.

Figure 3 shows the performance of five FRA patients who were administered remote memory tests for public events that had occurred from 1950–79. These patients were tested between 1982 and 1985. FRA patients had severely impaired recall for the entire time period covered by the test (figure 3*a*). As a group, they performed similarly to the patients who were tested during an episode of TGA. Of note, one of the FRA patients was unable to recall any of the items from this test, but none of the TGA patients performed so poorly.

Patients with FRA also appeared to be markedly impaired on the multiple-choice test for public events, in which the correct answer had to be selected from among four alternatives (figure 3*b*; chance = 25%). Their impairment was more severe than in the patients tested during TGA. Thus, patients with FRA had impaired recall for public events that was similar to the impaired recall of patients with TGA. In contrast, patients with FRA performed worse than TGA patients on the recognition test of public events.

Figure 4 shows the performance of nine patients with FRA on the test of past autobiographical memory. Like the patients tested during TGA, the patients with FRA were mildly impaired in their ability to recollect well-formed autobiographical memories unless they had the benefit of probing by the examiner. Yet, despite the fact that patients with TGA and FRA obtained similar scores, there was a striking difference in the time periods from which the two patient groups drew their memories (figure 5). Seventy-eight per cent of the memories reported by FRA patients concerned events that occurred after the onset of amnesia. Nineteen per cent of memories concerned events that occurred 0–10 years before the onset of amnesia, and only 3% of memories referred to events from more than ten years before the onset of FRA. The opposite pattern was observed in TGA. Only 3.5% of the memories reported during TGA concerned events that occurred after TGA onset. Six and one-half per cent covered events that had occurred 0–10 years before TGA, and most of the memories (90%) were drawn from more than ten years before the onset of TGA.

The neuropsychological findings for our ten FRA patients agree generally with the findings of four other patients who have been tested for anterograde and retrograde amnesia during FRA (Campodonico & Rediess 1996; patient K of Treadway *et al*. 1992; Kopelman *et al*. 1994; Schacter *et al*. 1982) and with the findings of three other patients who may have had FRA (the patient reported by De Renzi *et al*. 1995; patient F of Treadway *et al*. 1992; case M.M. of Lucchelli *et al*. 1995). Thus, these seven patients also performed normally on tests of anterograde amnesia, but had severe retrograde amnesia. Several points can be made about these patients. The patient described by Schacter *et al*. (1982) in their seminal, quantitative study of FRA performed similarly to our patients on the same test of past autobiographical memory, in that he drew most of his memories from the period after the onset of FRA. Their patient, however, exhibited an 'island' of preserved memories from a period about one year before the onset of amnesia. For one of the patients (Kopelman *et al*. 1994), malingering was thought to be an important factor. The patient reported by Campodonico & Rediess (1996) reported loss of all past memories, including memory of how to sew, drive and cook. Patients K (age 53 years) and F (age 39 years) of Treadway *et al*. (1992) developed severe localized retrograde amnesia covering 39-year and 16-year periods before the onset of amnesia, respectively. They had no impairment of earlier memories. The patients reported by Lucchelli *et al*. (1995) and De Renzi *et al*. (1995) each developed severe retrograde amnesia minutes to hours following motor vehicle accidents in which they suffered, at worst, minor head injury without loss of consciousness.

(*b*) *Neuroanatomy and aetiology of FRA*

Our ten FRA patients all had persistent, severe retrograde amnesia in the absence of anterograde amnesia, and we are unaware of any 'organic' brain lesion that can produce this clinical picture. We do not believe that our patients had focal retrograde amnesia (FoRA), which is characterized by minimal anterograde amnesia with severe retrograde amnesia for one to many decades before the onset of amnesia (Kapur 1993). FoRA is a neurological condition possibly resulting from damage to bilateral anterior and inferior temporal lobes (Kapur *et al*. 1992, 1994; Markowitsch *et al*. 1993).

Our findings agree with the clinical impression that FRA is a 'non-organic' or 'psychogenic' condition. The question often arises whether a patient with FRA has a conversion-like symptom (a deficit caused by some 'involuntary' or 'unconscious' process), or is malingering (intentionally feigning the deficit). One of our ten patients was certainly malingering. It is difficult to rule out entirely the possibility that additional patients also were intentionally faking their memory problems. There were no striking neuropsychological findings to distinguish our malingerer from the other nine patients, though he was the only patient who was entirely without premorbid psychological precipitating factors.

3. CONCLUSIONS

Our data indicate that patients with TGA have a uniform, consistent clinical picture. They have a severe relatively isolated amnesic syndrome that begins suddenly, persists for 4–12 h, and then gradually improves clinically to essentially normal over the next 12–24 h. During the episode, neuropsychological testing reveals that the patients have severe anterograde amnesia for verbal and non-verbal material. They have retrograde amnesia that typically covers at least two decades. Neuropsychological testing after the episode reveals normal performance on tests of anterograde and retrograde amnesia. The uniform clinical picture of TGA is consistent with its neurologic (organic) aetiology.

In contrast, patients with FRA have a sudden onset of severe retrograde amnesia without associated anterograde amnesia. FRA patients often have an

abnormal premorbid psychological status. Factors such as mild head injury, alcohol abuse, psychological trauma and recent criminal activity frequently may be associated with the onset of FRA. Examination of the patient with FRA often reveals overt depression, psychogenic sensory or motor impairment, or additional cognitive impairment such as forgetting how to use familiar devices such as a telephone, forgetting how to read and write, or forgetting the English language. Our findings show that patients with FRA do not necessarily have a benign course and often have persistent symptoms many months or years after the onset of the episode. The variable clinical picture of FRA reflects its psychogenic (non-organic) aetiology and is consistent with the idea that each patient adopts his or her own internally consistent pattern of memory impairment.

We thank Barbara Reader for assistance in the preparation of the manuscript. This work was supported by the Medical Research Service of the Department of Veterans Affairs and National Institute of Mental Health Grant MH 24600.

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