Transient global amnesia

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Transient global amnesia (TGA) is a memory dysfunction characterized by a sudden onset of dense anterograde amnesia (AA) that gradually resolves across several hours. TGA is typically accompanied by repeated questions (concerning present circumstances) with retrograde amnesia for events preceding the attack, and it usually strikes individuals in their late 50s and early 60s. The etiology of TGA remains unclear, although it probably reflects a temporary disruption of the blood supply to the temporal lobe. The physical and psychological events preceding the attack are diverse, but they often include acute emotional and physical stressors. TGA appears to be benign, with low risk for recurrence or residual complications. While systematic memory research on TGA patients is rare, these individuals present a unique opportunity to study amnesic behaviors in otherwise normal individuals. A guide to conducting future research is provided.

Over the last 4 decades, a considerable literature has accumulated concerning transient global amnesia (TGA). Originally described by Hague (1954; see Haas, 1983), the term TGA was coined by Fisher and Adams (1958) to describe a reversible, short-term (several hour) inability to store and/or retrieve new information most commonly observed in older adults (50–70 years old). Its onset is sudden, and its resolution is gradual. A period of retrograde amnesia (RA) is usually observed, the duration of which is highly variable (hours to years) across individuals and shrinks during the recovery period. TGA victims often ask the same question(s) repeatedly and are generally confused regarding time, place, and circumstances. Despite the inability to retain new information, general intellectual function (reading, writing, speaking, calculation) and self-identity are preserved, and obvious neurological abnormalities are absent. Oddly, most TGA victims experience only one episode in a lifetime. TGA appears to be unassociated with any obvious physical or psychological disease process or residual disability, contrasting with other memory maladies, such as Alzheimer's dementia, Korsakoff's disease, drug addiction, or hysterical fugue states (Markowitsch, 1995).

The primary purpose of the present review is to acquaint behavioral researchers with a common malady that is relatively unknown among psychology professionals. Despite a large literature on TGA, the phenomenon is poorly understood, and no consensus has emerged on its psychological or physiological cause. The present literature summary will hopefully allow cognitive, clinical, and health

psychologists to participate in the problem-solving process. The literature will be cited in detail to allow interested readers to pursue specific issues with the aid of a complete reference base. However, to enhance the readability of the manuscript, extensive blocks of references will be placed in the notes at the end of the article.

The literature on TGA consists of both *empirical* (describing specific TGA episodes) and *interpretive* (focusing on a possible etiology; summarizing some dimension of the phenomenon) articles. Empirical articles are composed of case studies, detailing the TGA episode in one or more patients, and group summaries, in which only summary data from a number of TGA patients are presented. (Note that, in the References, case studies are indicated by an asterisk [*], and group summary studies are indicated by a diamond [♦]. Articles that include a group summary and description of the TGA episode for a subsample of patients are tagged with both symbols. Some of these articles are not specifically cited in the text.) The 150 case-study articles describe a total of 488 individual cases, with the majority of these articles (59%) describing only one case. The 51 group articles summarize a total of 2,585 TGA patients, with a mean of 51 patients per article.

DEFINING TGA

Since the literature on TGA has evolved slowly from diverse areas of medicine (anesthesiology, neurology, and radiology), a methodological anarchy exists in the way TGA is defined and reported. For the most part, researchers adhere to the definitional criteria provided by Caplan (1986b):

- (1) The attack onset should have been witnessed.
- (2) Dysfunction during the attack should have been limited to repetitive queries and amnesia.
- (3) There should have been no other major neurological signs or symptoms.
- (4) The memory loss should be transient, usually lasting hours or up to 1 day.

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Such a definition is designed to rule out other neurological dysfunctions or traumatic events that can cause amnesia (e.g., stroke). However, some claim that this criterion is too strict or premature in this nascent stage of research on TGA (Hodges & Warlow, 1990b; Meador, Adams, & Flanigin, 1986; Shuaib, 1986), and the fine points of the definitional criteria debate can be found in a series of letters and replies.¹ As Shuaib (1986) points out, although restrictions are needed on the definition of the phenomenon, such exclusionary criteria may work at cross-purposes to ultimately identifying the cause of TGA, given the current state of uncertainty in the field (see Jain et al., 1989). In short, no general paradigm or format has evolved on reporting TGA, as illustrated by these contrasting case reports:

Case 1

A 60-year-old civil engineer had no family history of nervous or mental disease ... On February 21, 1965, he had spent the day much as usual. In the morning he had been in the garden and had eaten a normal lunch at mid-day. After lunch he had gone out in his car and spent the next two hours cleaning it ... He then came indoors and his behaviour was perfectly normal in every way and he went to have a bath and change. He normally took his baths exceptionally hot and would soak in them for an hour or more. On this afternoon he emerged from the bathroom after half an hour, correctly dressed in ordinary clothes, but he looked puzzled and upset. His first words were "Am I going mad? I can't remember anything." He then repeatedly asked a number of questions such as "What day is it? What has happened? What have I been doing? How did this start?"

His wife replied to all these questions and he appeared to understand what she said, but he would ask the same question a few minutes later. From his behaviour he recognized his wife and knew his way around the house. He was brought a cup of tea which he drank normally, but then accused his wife of drinking his tea, apparently forgetting that he had himself drunk it. At this stage he was reminded that the car was still outside the garage, so he went out and put it into the garage without mishap. He spent the rest of the evening watching television and subsequently went to bed, sleeping well. During this time his speech was coherent, he knew where he was working and he knew all about his friends. At first he was bewildered and wept on two occasions, but later he became quiet and subdued.

On the following morning he awoke and said "What am I doing in bed at this time of day?" His wife tried to explain what had happened and then he said that he thought it was Sunday morning. Later on during the morning ... his memory for previous events returned as far as lunch time on the previous day. (Evans, 1966, pp. 542-543)

Case 2

The principal of a Technical College was seen five days after an episode of amnesia which had occurred on his 60th birthday. He had been chopping wood for two hours on a warm day when he suddenly lost his memory. He kept asking the same questions and repeatedly asked his wife where he was and what he was doing. His speech and behavior were otherwise normal. His wife called their family physician who confirmed the memory loss, which extended back

for several years, and toward the end of his visit the patient's memory began to return. The period of permanent amnesia was about two hours. (Heathfield, Croft, & Swash, 1973, p. 730)

DEMOGRAPHY OF TGA

Incidence

The estimate of the incidence of TGA in the general population is 5.3 per 100,000 people across six studies from four countries (United States, England, Spain, and Finland).² Among older adults (≥ 50 years), the incidences are much higher at 32 (Koski & Marttila, 1990) and 23.5 (Miller, Petersen, Metter, Millikan, & Yanagihara, 1987) per 100,000. Thus, it appears that the likelihood of experiencing a TGA in any given year is roughly 0.005% (1 in 20,000 people) in the general population and 0.028% (1 in 3,000) among older adults.

These numbers probably underestimate the actual incidence of TGA. Because there is minimal physical abnormality during the TGA episode and recovery is generally complete, there may be little motivation to receive medical assistance or even report the incident (Bender, 1956, 1960; Rollinson, 1978). As Bolwig (1968) cogently points out, TGA "is probably more common than would be assumed by the relatively few reports, as many patients would hesitate to consult a physician for symptoms that are likely to impress the relatives more than the patients themselves" (p. 105) (cf. Dinsdale, 1971). Under-reporting may also be motivated by relatives who interpret the symptoms as reflecting dementia or mental instability (Santoro, Casadei, & Venco, 1988). When a wife of a TGA victim heard him discussing a relative who had died a year ago as if she were alive, she "grabbed the telephone and hung it up in an effort to conceal the insanity from the rest of the world" (Patten, 1971, p. 690). Rollinson (1978) further notes that patients tend to be "the more prominent members of the community, e.g. physicians and relatives of physicians, perhaps suggesting that the occurrence of TGA in such a person is less likely to pass unnoticed" (p. 547) (cf. Hodges & Warlow, 1990b).³

The likelihood of different members in the same family experiencing a TGA is addressed in several reports (Corston & Godwin-Austen, 1982; Dupuis, Pierre, & Gonsette, 1987; Munro, 1982; Stracciari & Rebucci, 1986). In group studies, Ciucci, Stracciari, and Bianchedi (1988) found that 7% of their patients had other family members who had also experienced a TGA, whereas Hodges and Warlow (1990b) noted this rate to be 2% of their patients. While these familial reports flag a possible genetic component of TGA, it is also possible that families are more alerted to the phenomenon after TGA has occurred to one member.

Age

The distribution of patients' age during the TGA episode from case-study reports is presented in Figure 1, separately for males and females. Overall, the range is 6-84 years (interquartile range = 51-64 years), with a

Figure 1. Frequency distribution of patients' age during the TGA attack.

mean of 55.3 years ($SD = 13.9$ years), a median of 59 years, and a mode of 60 years (7% of the patients were 60). In the set of 25 group studies reporting mean age for their sample, the mean of the means is 61.0 years (median = 60.8, range = 55–67, $SD = 2.3$).

To compare this age distribution with the population in general, statistics from the U.S. Bureau of the Census (1994 resident population) were used to compute the percent of individuals in each age category presented in Figure 1 (using only individuals between the ages of 11 and 85 to compose a 100% baseline). When the overall population percentages are subtracted from the percent of TGA patients in each age category (see Figure 2), individuals under 50 are underrepresented as TGA patients, whereas people between 50 and 65 are overrepresented as TGA patients. More importantly, the sharp drop in TGA incidence after age 65 does not simply reflect a natural decline in the number of older adults. In short, the ages of 50–70 represent an elevated risk period for experiencing TGA.

Gender

Patients' gender is reported in all case studies (males = 280; females = 208) and in 37 group studies (males = 812; females = 976). Combined across both case and group studies, there were 1,092 males (48%) and 1,184 females (52%), and these percentages differ significantly

from a chance (50/50) split [$\chi^2(1) = 3.98, p < .05$]. While female outnumber male patients, this may be due to an overrepresentation of females in the older age range where TGA is most common. Comparing the genders on mean age during the TGA episode (see Figure 1), females were significantly older than males both in case studies (males = 53.1 years; females = 58.1 years) [$t(486) = 3.72, p < .01$] and in the eight group studies reporting such data (males = 58.7 years; females = 61.7 years) [$t(7) = 4.00, p < .01$]. This significant age difference may simply reflect that females live longer than males.

In summary, the likelihood of experiencing TGA in a given year is about 1 in 20,000 in the general population and 1 in 3,000 in older adults. The actual incidence of TGA is probably higher, with many cases unreported. Upper-class or well-educated individuals tend to be overrepresented among patients. Typical TGA patients are in their mid to late 50s or early 60s, with a slightly higher probability of being female than male. Females who experience TGA are also older than their male counterparts.

THE TGA EPISODE: GENERAL FRAMEWORK

The following section summarizes dimensions related to the onset and duration of the TGA experience, including events preceding the attack, when an attack is likely

Figure 2. Difference between population and TGA age distributions, with respect to the percent of individuals in each age grouping.

to occur, and the duration of the episode. Following this, the patient's experience during the attack is addressed, including physical and psychological symptoms, orientation, and RA.

Precipitating Events

One of the most enigmatic dimensions concerning TGA is its cause. In this predominantly medical literature, many investigators have searched for a specific biological trigger, an effort that has not proved successful. Most of the literature focuses on events that immediately precede the attack, and these proximal causes are reviewed first. A few reports also include experiences preceding the attack by days, weeks, or months, and these distal factors will be considered second.

Proximal factors. A wide variety of circumstances have been found to precede a TGA attack, and this information is provided in about a third (32%) of reports. The various preceding events are listed in the Appendix, along with the frequency and percent of cases for each. This information is presented in detail in the hopes that some readers may detect patterns that have previously gone unnoticed. TGA attacks are most frequently preceded by light to moderate physical exertion (about a third of the time), with the most notable activities being sexual intercourse (8%), driving (5%), and bathing (5%). TGA also follows heavy physical exertion, with farm work (7%) topping the list. General emotional stress also accounts for a fair num-

ber of cases (8%), as does mild head injury (5%) unaccompanied by concussion or loss of consciousness.

In an effort to give coherence to this diversity of "precipitating events," Caplan (1990) suggests that an abrupt change in physical activity, temperature, environmental conditions, or emotions may precipitate an attack (see Dinsmore & Callender, 1983; Frederiks, 1993), and Fisher and Adams (1964) note that nearly a third of their patients' attacks are immediately preceded by emotionally or physically stressful events (or both). Frederiks (1990) evaluated the consistency in the antecedent activities for 19 recurrent episodes experienced by 15 patients. He found that 11 preceding events were the same and 8 were different, suggesting an equal likelihood that multiple TGA attacks will be preceded by the same or different events. For a more thorough description and analysis of proximal precipitating events, see Fisher (1982).

Distal factors. Physical and psychological stressors in the weeks prior to TGA may also precipitate an attack. Although such events are rarely reported (less than 10% of case reports), they include (frequency in parenthesis): a trip for business or pleasure (17), a death or illness in the family (10), the end-of-year holiday season (10), hosting an upcoming party (5), impending or completed surgery (4), stress at work (4), severe depression (2), pregnancy (1), and financial difficulties (1). Laurent, Trillet, and Croisile (1990) discovered that a third to half of their patients had a profile of "psychological pressure" preceding the

attack, and about a third of the patients in other studies were under considerable stress prior to TGA (Miller, Petersen, Metter, et al., 1987; see also Guyotat & Courjan, 1956, cited in Laurent et al., 1990). Markowitsch (1983) further observed that a number of TGA patients appear to be currently (or previously) in responsible, high-stress positions (see Fisher, 1982).

As an example of the type of emotional stress that may precipitate TGA, Merriam, Wyszynski, and Betzler (1992) describe a woman who had a strong need to reunite with her family over Christmas because it was the first time they could get together since the recent death of her mother. Her supervisor would not allow sufficient time off, causing her to agonize over the prospect of being alone for the holidays. On the evening prior to her TGA, she received a note from her son pleading with her to join them, which she felt placed her in an "unbearable situation." Also, Stevens and Ammerman (1974) point out that their patient had been "agonizing over a complicated estimate on an extensive landscape project" (p. 594) prior to TGA. At any rate, as with the proximal factors, a more thorough exploration of the patient's personal stressors could help to shed light on the cause(s) of TGA.

Time of Onset

TGA is more likely to occur in the morning and in colder months. In the 78 cases where a starting hour was given (see Figure 3), TGA most often occurs between 7 a.m. and 12 noon (59%). In 72 other cases, the start time is

given generally as morning (61%), afternoon (25%), or evening (14%). These percentages parallel those in Figure 3, as well as others' reports of 60% (Mazzucchi, 1988) and 62% (Zinelli, Nasuto, & Miari, 1988) of TGAs occurring in the morning hours. The month of the TGA attack is identified in 85 cases (see Figure 4), and most (59%) occur during the colder autumn and winter months (October through March), with the heaviest preponderance in December (18%). This supports Mazzucchi's (1988) finding that 70% of TGA episodes occur in the autumn and winter months. While this trend may be related to cold weather, it may also be related to the preponderance of holidays (Thanksgiving, Christmas, and New Years) during this period and the stress accompanying such events (see earlier section on Precipitating Events).

Duration

Most case studies (95%) report TGA duration, and these data are summarized in Figure 5, with half-hour increments rounded down (e.g., 2½ h is treated as 2 h). The mean TGA duration is 8.2 h ($SD = 11.1$, range = 4–96), with a mode of 2 h and a median of 5 h. The period of 2–4 h accounts for more than a third (37%) of the attacks. In 10 group studies (619 patients) reporting mean TGA duration, the mean of the means is 7.2 h ($SD = 3.6$, range = 4.1–17), with a median of 6.5 h.

Several cautions are in order in evaluating these data. First, the precise time of onset is often ambiguous. Although TGA onset is believed to be sudden, the relatively

Figure 3. Frequency distribution of times of the day when TGA started.

Figure 4. Frequency distribution of months of the year when TGA occurred.

Figure 5. Frequency distribution of TGA duration.

subtle change in the patient's behavior (disorientation, inability to encode new information) may not be immediately apparent if the victim is engaged in routine (work, hobby) or isolated (watching television) activities. Another difficulty in evaluating the temporal extent of the TGA episode is that recovery is usually gradual making a precise termination point difficult to establish. A further complication is that some individuals wake up in the morning with TGA or go to bed prior to recovery (Fisher, 1982), and reported TGA duration may or may not include the sleep period. This distribution may also be truncated at each end because researchers sometimes exclude TGA episodes that are very short (< 1 h) and/or very long (> 24 h) (Laurent et al., 1990).

With respect to gender, the mean TGA duration is identical (8.2 h) for males and females. With respect to age, there is no significant correlation ($p > .05$) between age and TGA length either overall ($r = +.09$) or when the extremes of age (patients younger than 40) and TGA length (episodes longer than 20 h) are excluded from the analysis ($r = +.07$). Comparing older (≥ 60 years) with younger (< 59 years) patients, there is a nonsignificant trend for longer TGA episodes with older patients (8.9 h) than with younger patients (7.5 h) [$F(1,418) = 1.63, p > .05$].

In summary, the typical TGA episode lasts 2–4 h and occurs in the morning hours and during colder weather (winter). The length of attack does not differ as a function of age or gender of the patient. A wide variety of events may precede the TGA, although most appear to involve some emotional and/or physical stressor.

Nonmemory Symptoms

Although the memory-related symptoms are at the heart of the TGA experience, TGA patients do exhibit a variety of physical and psychological symptoms during the attack, which are usually described by physicians, relatives, and friends. Of the psychological symptoms described, 50% involve *confusion* (confused, bewildered, disoriented, dazed, puzzled, perplexed), 36% involve *arousal* (anxious, agitated, restless, astonished, euphoric, disturbed), and 14% involve *lethargy* (tired, quiet, strange, vegetative, apathetic). It is often reported that complex behaviors are carried out in a bland or blunted manner. For example, a man who insisted on playing with his band during TGA was reported (by his wife) as playing in a detached manner, as if simply going through the motions (Palmer, 1986). Laurent et al. (1990) suggest that this typical passive or quiet personality might reflect the patient's desire to keep others from discovering their temporary dysfunction. Some patients report feeling psychologically distant, like being a "long way off" (Munro, 1982) or "looking through the wrong end of a telescope" (Meador, Adams, & Flanigin, 1985). Any of these symptoms may reflect either the patients' reaction to their cognitive dysfunction or a changed physical state during TGA.

The physical symptoms experienced during TGA are more diverse and are reported in only a small percentage

of reports (16%). Of the 253 reported physical symptoms, headache accounts for nearly half (48%) (Laurent et al., 1990), followed by nausea/vomiting (14%) and dizziness/unsteadiness (14%), paleness (5%), tearfulness (4%), and neck pain (2%). As with the psychological symptoms, physical symptoms may be either a direct result of TGA or the patients' reaction to the episode.

ANTEROGRADE MEMORY PERFORMANCE

The primary defining feature of TGA is temporary anterograde amnesia (AA), or inability to encode new information. *Direct* measurements of AA are covered under the topics of working memory (less than 1 min) and episodic memory (2 min or longer). *Indirect* indications of memory dysfunction are reflected in repeated questions and behaviors, answers to physicians' orienting questions, and patients' self-reflections on their present circumstances.

A major difficulty in assessing anterograde memory dysfunction is that the TGA episode is transient with gradual resolution. Thus, as an evaluation is being performed, the episode is usually clearing up. This problem is illustrated by Kritchevsky, Squire, and Zouzounis (1988):

Patients were examined and tested between 2 and 11 hours after the onset of the TGA. All patients were amnesic at this time, although in three cases . . . family members stated that the memory problems had begun to improve by the time of testing. A fourth patient . . . recovered noticeably during the testing session. The fifth patient . . . developed memory problems in the hospital, was examined and tested beginning 2 h later, and did not exhibit signs of improvement until several hours after the completion of testing. (p. 213)

Thus, evaluations conducted during the episode provide a conservative account of the full extent of cognitive and memory impairment resulting from TGA (also see Kritchevsky & Squire, 1989).

Working Memory

With respect to working memory, the most commonly used test is digit span. The forward digit span is consistently in the normal range of 5–9 digits, with a mode across studies of 7.⁴ For backward digit span, performance is within the normal range (4–6 digits) in most (10 of 15) cases.⁵ Also consistently in the normal range are forward word span (Gordon & Marin, 1979) and serial subtractions (generally by 7s).⁶ Immediate recall of short (sub-span) lists of words is also normal (Cafarra, Moretti, Maz-zucchi, & Parma, 1981; Wilson, Koller, & Kelly, 1980), as is kinesthetic working memory (Cafarra et al., 1981; Gallassi, Lorusso, & Stracciari, 1986; Hodges & Ward, 1989).

Several dimensions of working memory do reflect deficiencies, or mixed performance. When the task becomes more complex or demanding, performance appears to suffer. Paired-associate learning appears to be impaired,

with the 17 TGA patients tested performing (on the average) at about 30% of the level of control subjects (Hodges & Ward, 1989; Kritchevsky & Squire, 1989; Kritchevsky et al., 1988). A more serious deficiency involves spatial ability. All 4 patients tested for immediate spatial recall showed very poor (or no) ability to reproduce a spatial arrangement (Cafarra et al., 1981; Hodges & Ward, 1989; Ponsford & Donnan, 1980). Auditory verbal processing (oral presentation of words) is also markedly impaired in the 3 patients evaluated (Berthier & Starkstein, 1990; Gallassi et al., 1986; Stracciari, Rebucci, & Gallassi, 1987). Finally, patients' ability to process visual (non-verbal) information is also mixed. When evaluating visual retention (Benton test), 2 of 3 patients were in the normal range (Araga, Fukada, Kagimoto, Inagawa, & Takahashi, 1989; Berthier & Starkstein, 1990; Cafarra et al., 1981). With figure copying (Rey complex figure test), some have found that TGA patients perform comparably to controls, whereas others have found impaired performance relative to controls.⁷

In summary, while working memory is generally intact, there may be some problem with complex material (story recall, paired associate learning), as well as spatial ability and visual memory. However, the limited number of patients evaluated on these dimensions limit the generality of such conclusions, and further research is strongly indicated.

Episodic Memory

The evidence for severe impairment of retention 2 min after input is absolutely monolithic, with most patients unable to recall even experiencing the earlier input task. Dismal levels of episodic memory have been confirmed with word lists in visual or auditory format, numbers and letters, faces, visual figures, objects, stories, paired-associate lists, and spatial location.⁸ Shuttleworth and Wise (1973) compared a variety of different materials (proprioceptive/kinesthetic, olfactory, auditory nonverbal, auditory verbal, visual verbal, and tactile) and failed to find evidence of memory in any dimension. Even providing associative and rehearsal strategies at input (Gordon & Marin, 1979) or cuing at recall (Caplan, 1985; Gordon & Marin, 1979; Patten, 1971; Stillhard, Landis, Schiess, Regard, & Sialer, 1990) fails to help.

Post-TGA memory gap. Following recovery from TGA, most patients do not recall any experiences from the period of AA, even though they are aware of the memory gap. Although concerned during the episode, patients are typically unconcerned after TGA about the memory gap and what it may portend (Frederiks, 1993). Patients occasionally recall fragments of experiences that had occurred during the amnesic period,⁹ although such memories usually have a dreamlike quality (Evans, 1966). These memory pockets may also be a result of rare occasions where AA is less "dense" (Okada, Ito, & Tsukamoto, 1987) or the TGA onset is gradual or "stuttering" (Laurent et al., 1990). However, such occasional reports

should be viewed skeptically for several reasons. Patients who hear witness' descriptions of their episodes may later mistakenly identify these "recollections" as their own (Brown & Murphy, 1989). Also, these experiences may have occurred when the episodes were close to resolution. In any case, these recollections appear to be rare exceptions, and the general rule is that the patient recalls nothing from the episode.

Repeated Questions

Aside from direct empirical measurement of anterograde memory during TGA, there are several indirect indications of impaired memory function—the most obvious of which is repeated questioning.

The patient would ask, in a very polite but stereotyped manner as if it were a new event, "Excuse me, I know that this is a silly question, but what am I doing here?" The examiner made exactly the same reply each time. "You are in this place because of some trouble with memory which you seem to have. You will be well very soon." This same conversation took place 15 to 20 times, with the same degree of surprise on the part of the patient. No recollection of it was ever noticed. (Goldenberg, Podreka, Pfaffelmeyer, Wessely, & Deecke, 1991, p. 573)

Repeated questions are asked by 40% of case-study patients and between 50% and 92% of patients in group summaries (50%, Kushner & Hauser, 1985; 61%, Caplan, 1985; 90%, Fisher, 1982; 92%, Hodges & Warlow, 1990b). Most patients ask the same question(s) 10–20 times, with the same stereotyped vocal pattern each time (Bender, 1956; Fisher, 1982), "as if a fragment of sound track is being repeatedly re-run" (Frederiks, 1993, p. 266). If several questions are asked, they are usually in the same order each time (Hodges & Ward, 1989). In 125 case reports where specific questions are described, 46% of the patients repeated one, 30% repeated two, 16% repeated three, and 8% repeated four through six questions.

The specific questions mainly concern present situation (51%), with the most typical being "what am I doing here?" (15%), "why am I here?" (6%), and "how did I get here?" (4%). The temporal dimension is the focus of 27% of questions ("what time/day/month/year is it?"), with spatial disorientation ("where am I?") involved in 19% of the queries. Finally, questions relating to personnel are relatively rare (2%). TGA patients, almost without exception, retain an awareness of who they are. Additional speculation on the neurological significance of repeated questioning can be found in Fisher (1982).

A few case reports (5%) describe repeated behaviors, such as wandering through the rooms of a familiar house, pacing back and forth, repeatedly calling someone, visiting a friend over and over, turning the TV on and off, getting in and out of a chair, and walking up and down stairs. Also noted are repeated "checking" behaviors involving such things as a dishwasher, car trunk, or closet. Similar to repeated questions, repeated behaviors probably reflect the patient's inability to keep track of recent

experiences. Interestingly, prospective memory may be more intact than retrospective memory, in that the patient can keep track of the goal but not whether they have accomplished it. A precise record of the nature and timing of such behaviors would be of considerable value in evaluating the distinction between prospective and retrospective memory function.

Physician's Exam

In addition to repeated questions and behaviors, disorientation during TGA also may be inferred from a physician's standard neurological examination (Amit, Shapira, Flusser, & Aker, 1986). None of 74 patients was correctly oriented to the present event or situation, and only 15% were correctly oriented for time (0% for time of day, 7% for day of week, 17% for date, 25% for month, and 18% for year). Patients are more aware of place, with 30% able to identify where they were. Orientation to person is the most successful. Patients always know who they are, and they recognize familiar others 63% of the time. Interestingly, recognition is higher for relatives (64%) than for acquaintances (30%), which may reflect the level of prior familiarity with the person. In a large group study, Hodges and Warlow (1990a) found percentages roughly similar to the present ones: 13% of patients were oriented to time, and 42% were oriented to place.

In summary, orientation improves across the domains of situation (0%), time (9%), place (30%), and person (57%). The relative degree of orientation parallels that found with repeated questions, with most questions about situation and fewest about personnel.

Self-Awareness

During TGA, some patients (18% of case reports) express concern over their dysfunction (Bender, 1956; Evans, 1966). While 5% of reports simply note that the patient is aware of a disability, the remainder provide quotes that reflect the patient's reflective position: 8% note a general functional problem ("what has happened to me?" "there's something wrong with me"), 3% comment on a general cognitive difficulty ("have I lost my mind?" "I'm disoriented"), and 3% mention a specific memory dysfunction ("I can't remember anything," "I have amnesia"). Of special note is the patient who commented that "my memory is a tape that is being erased as it is made." Memory comments are occasionally repeated (similar to questions), as illustrated by Fisher and Adams's (1964) patient who repeated "I can't remember today" about 30 times.

Patients' diverse comments on their present, amnesic situation have the potential to yield valuable information concerning the subjective experience of TGA, but they remain largely untapped by researchers. An exception is Stevens and Ammerman's (1974) description of an unusually reflective patient:

He states "everything evaporated into the distance. I knew what I had to do but knew that I couldn't think how to do it, I was conscious through the whole thing." He vainly struggled to reconstruct the events of the day, tried to think

of his employees, "it was like I knew them years ago but there was not a vivid present thing." ... To his wife he stated "Helen, you seem to be a million years away." He then states he felt he was in a setting "like I just returned from a long interval, like I had to get reacquainted again." (pp. 594–595)

This feeling of being distant or far away is similar to other patients' descriptions of being disoriented, dreaming, in a haze, in a fog, and everything being strange and far away. It could be of considerable value to systematically gather these metamemory and metacognitive evaluations through a structured questionnaire. To date, no case study has done this.

RETROGRADE MEMORY PERFORMANCE

Researchers with considerable experience evaluating TGA claim that all patients have some RA during TGA (Caplan, 1990; Markowitsch, 1983), and many researchers use it as a defining criterion. Although frequently described, haphazard reporting makes it difficult to determine how often RA actually accompanies TGA and the exact nature and duration of the dysfunction. While a few studies have attempted a formal, structured assessment of the duration of RA, many more have evaluated its existence in a more informal manner.

Formal Quantitative Assessment

A few investigations have utilized standardized techniques to evaluate the existence, and extent, of remote memory during the TGA episode. With the Famous Faces Test (Albert, Butters, & Levin, 1979; Sanders & Warrington, 1971), Hodges and Ward (1989) found that during TGA there is a "graded" RA, with name identification ability more impaired for the most recent decade. However, with the Famous Events Test, Hodges and Ward found that recognition is normal for all patients for all decades. Despite adequate recognition, dating these events is problematic. While 3 patients showed selectively poor performance for events from the most recent 2 decades (graded RA), 2 others performed more poorly on all decades (flat RA). Using a similar test of public events, Kritchewsky and Squire (1989) found degraded recall performance for the period of 25 years preceding the episode, along with a recognition deficit extending back 5 years. In contrast, a recognition test for single-season TV shows showed no diminished remote memory (Kritchewsky et al., 1988).

The Crovitz Test of Remote Autobiographical Memory (Crovitz & Schiffman, 1974) requires individuals to generate (free associate) personal events to common noun cue words (birds, tree, car). While control individuals produce 25% of their personal events from the most recent 5-year period, Hodges and Ward (1989) found that TGA patients produced only 4% of responses from this period during the episode. In contrast, Kritchewsky et al. (1988) failed to find any difference between pre- and post-TGA performance at any time period on this test. Using

a test constructed from the patients' own areas of interests and experiences, Kritchevsky and Squire (1989) found that the most severe performance decrement during TGA (compared with post-TGA recall) is for the year and a half preceding TGA.

In summary, although the amount of research is limited, there is a consistent evidence of a graded remote memory deficit during TGA, with remote memory difficulties more pronounced for recent events than for older events. Although there are some exceptions, this has been found across a variety of materials (famous faces, public events, personal experiences) and tests (recall, recognition, free association). The existence of graded RA has important implications for the question concerning the significance of flat versus graded RA in amnesics (Albert et al., 1979; Beatty, Salmon, Butters, Heindel, & Granholm, 1988; Kopelman, 1989; Squire, Haist, & Shimamura, 1989). Prior evidence for graded RA has been found mainly in amnesic patients with neurodegenerative diseases (Alzheimer, Korsakoff), making the interpretation of such selective impairment problematic: Did a gradual onset of the dysfunction result in progressively degraded encoding prior to the amnesia? Thus, the existence of graded RA in TGA patients suggests that graded RA is not a result of progressive encoding inadequacies but rather can be considered a retrieval phenomenon.

Informal Quantitative Assessment

Most of the information on the extent of RA has been derived from physicians' queries, errors in the patient's voluntary verbal reports, or comments by friends or relatives concerning patients' behaviors. For example, while nearly all patients remember their birth date, most are unable to recall their current age,¹⁰ or they claim to be several years younger than they actually are—a discrepancy that logically reflects RA. With voluntary reports, a particularly striking example of RA is the patient who asked what happened to the fingers on his left hand, not remembering that they had been traumatically amputated in a farm machinery accident 4 months earlier (Logan & Sherman, 1983).

The extent of RA has been assessed both during and after TGA, and these data are presented separately. A range of times is often given, such as Evans's (1966) patient whose RA "was judged to be several days, if not a few weeks" (p. 540). In such instances, the more conservative estimate (2 days) is used in the data summary.

During TGA. RA during TGA is reported in 39% of case reports, with 7% simply noting its existence and 32% providing a specific time period. In group summaries, RA is found in the majority of patients (66%, Hinge, Jensen, Kjaer, Marquardsen, & Olivarius, 1986; 78%, Hodges & Warlow, 1990b). Data from 160 case-study patients, summarized in Figure 6, reveal considerable vari-

Figure 6. Frequency distribution of retrograde amnesia period during TGA.

ability in the temporal extent of RA during TGA, with a median of 7 days and modes of 1 and 2 days (range = 0.5 h to 35 years) (note that these data summarize informal assessments). Since RA almost always shrinks during TGA, with more remote events recovering first (Bassior, Vogel, & Mitton, 1985; Benson & Geschwind, 1967; Fisher & Adams, 1964; Hodges & Ward, 1989), the farther into the TGA episode that RA is assessed, the shorter its duration. This makes the present summary of RA length during TGA conservative.

After TGA. While RA is common during TGA, some patients also exhibit a permanent, residual RA following the attack. RA always shrinks by the end of TGA, and it is not uncommon for RA to continue to shrink even after the episode is over, as illustrated by the following examples: a 2-month RA during TGA reduced to 8 h the following day, 2 h a few days later, and 1 h after 3 months (Jensen, 1980); a 2-month RA during TGA shrank to 1 month after 9 h, 15 h after 1 day, and was gone by 2 days (Regard & Landis, 1984); a 2-year RA during TGA decreased to less than 1 h after 12 h, and was gone by 3 days (Stracciari, Rebutti, & Gallassi, 1987).

A summary of the extent of final, or permanent, RA, which is provided in 21% of cases (see Figure 7), indicates a median and mode for post-TGA RA of 1 h (range = 0 h to 5 months) (see Fisher, 1982). Among group studies, the percent of patients exhibiting permanent RA varies considerably from 20% (Kritchevsky et al., 1988) to 44% (Fisher, 1982) to 78% (Hodges & Warlow, 1990b). The actual percentage of patients who experience per-

manent RA is unclear. Many studies do not provide this information, even when RA during TGA is reported. Others (perhaps mistakenly) assume that permanent RA is a necessary result of TGA (Rollinson, 1978) and therefore do not consider it necessary to comment on it.

Relationship of RA to AA. Are RA and AA related to each other (Kritchevsky et al., 1988; Laurent et al., 1990)? This issue has stirred considerable interest because, if true, it implies that the same structures mediate both AA and RA (Blomert & Sisler, 1974; Russel & Nathan, 1946; Shimamura & Squire, 1986). Kritchevsky et al. (1988) note that, among their 5 patients, those with less severe AA had shorter RA. The present data were examined for evidence of a relationship between the length of TGA and RA. Comparing short (5 h or less; 67 cases) versus long (6 h or more; 63 cases) TGA episodes, RA during TGA is greater than 2 days in 51% of short episodes and 62% of long episodes, but this difference is not statistically significant [$\chi^2(1) = 1.62, p > .05$]. Comparing short (43 cases) TGA episodes with long (41 cases) TGA episodes for RA following TGA, RA is greater than 1 h in 40% of short episodes and 49% of long episodes, a difference that is again nonsignificant [$\chi^2(1) = 3.05, p > .05$].

In general, it appears that a typical RA of 1 or 2 days during the TGA shrinks to about 1 h by the end of the attack (see Hodges & Ward, 1989). However, unlike the conservative measurements during TGA, evaluations after TGA are most likely liberal because many are done soon after recovery. Also, if patients are queried days or weeks following the attack, friends or relatives are likely

Figure 7. Frequency distribution of retrograde amnesia period after TGA.

to have discussed pre-TGA behaviors with the patients, contaminating the patient's own recollections. While there is a nonsignificant trend for longer (more serious?) TGA episodes to be associated with longer RA (see Vohanka & Zouhar, 1988), this potentially important question warrants further investigation using more precise measures of both the severity of AA and the length of RA (see Fisher & Adams, 1964; Whitty, 1977).

Qualitative Assessment

In addition to measuring the duration of RA, there are other alterations in the quality of the remote memories that deserve comment. For instance, dating remote events appears to be consistently problematic during TGA (Caffarra et al., 1981; Gallassi et al., 1986; Hodges & Ward, 1989; Stracciari, Rebutti, & Gallassi, 1987). Wood, McHenry, Roman-Campos, and Poser (1980) describe their patients' extensive (10-year) retrograde "amnesia" as consisting of numerous islands of intact memories detached from the temporal context, resulting in considerable confusion of event sequence. Anecdotal reports confirm this dating difficulty, as illustrated by patients who could, for example, describe a car but did not know whether he currently or previously owned it (Regard & Landis, 1984), remember his two daughters and their names but did not know who was older (Regard & Landis, 1984), not remember whether he had retired in 1969 or 1971 (Kritchevsky et al., 1988), remember the recent Presidents but got their order confused (Bender, 1960), and recall a helicopter landing but not whether it happened "a few minutes ago, a few hours ago or on a previous day" (Regard & Landis, 1984, p. 668).

There is also a consistent tendency during TGA for personal recollections of remote experiences to be "curiously empty and lacking in colour . . . as if reduced to the bare bones of memory" (Hodges & Ward, 1989, p. 600). In addition, confabulation rarely occurs during spontaneous recall, the physician's interview, or standardized tests.¹¹ In fact, Hodges and Ward (1989) note that their patients often begin confidently to respond to an autobiographical inquiry, and this quickly turns to surprise and frustration when an "unexpected void" is encountered.

On occasion, recall of remote events may have scattered lacunae rather than being completely missing (see Kritchevsky & Squire, 1989; Whitty, 1977). This "patchy" RA is illustrated by patients who had no memory of eating lunch just before the TGA and a "vague" and "incomplete" memory of the morning events preceding lunch (Kritchevsky & Squire, 1989), no memory for 2 days and poor memory for 2 weeks prior to the TGA (Meador, Loring, King, & Nichols, 1988), dense amnesia for 2 weeks and patchy amnesia for 2 months before TGA (Stillhard et al., 1990), and dense amnesia for 1 day and patchy for 1 week prior to the TGA (Wilson et al., 1980). In all cases, the memory void (dense period) is always immediately preceding TGA, while the period of degraded memory (patchy period) precedes the dense period.

General Knowledge

The general knowledge base of patients is usually unaffected during TGA. Often, such confirmation is global and nonspecific, such as a report that the patient has normal reading, writing, and language comprehension abilities (see Stillhard et al., 1990), left-right orientation (Amit et al., 1986; Gorelick, Amico, Ganellen, & Benevento, 1988; Halsey, 1967; Stillhard et al., 1990), and writing to dictation (Donaldson, 1985; Gorelick et al., 1988). More specific memory tests involving such things as simple analogies, proverb interpretation, or object naming also reveal preserved performance.¹² All patients provided their home address (17) or birth date (10) correctly, although 7 of the latter 10 underestimated their age. Remembering personally relevant numbers (social security, safe combination, telephone) is also problematic, with only 7 of 15 patients answering such queries correctly. In summary, general knowledge is usually preserved during TGA, with the only sign of dysfunction being with numerical information such as addresses, current age, and phone numbers.

Procedural Memory

While procedural memory also appears to be unaffected during TGA, this conclusion is mainly derived from reports of complex behaviors performed during TGA rather than systematic empirical evaluation. TGA episodes often occur while the patient is driving, and witnesses consistently report that this does not interfere in any obvious way with the control of the automobile. During TGA, patients are also able to complete a sporting event (Giroud, Guard, & Dumas, 1987; Posteraro, 1988), taxi an airplane for takeoff (Fisher, 1982), complete a bell-ringing musical performance (Hodges & Warlow, 1990b), and cook a meal (Evans, 1966) without any diminution of skill. One surgeon was in the midst of an operation when TGA occurred, and, despite his confused comments to his nurse, he finished the surgical incision "as well as he had closed up any gallbladder . . . in the last dozen years. Technically his hands had performed their job as well as ever" (Klawans, 1988, p. 14).

Sometimes patients revert to an earlier (outdated) version of a complex behavior sequence. For example, a retired music professor stricken with TGA in the middle of a piano recital and duet with his wife (Byer & Crowley, 1980) completed the performance successfully, but without recent modifications practiced many times prior to the recital. Similarly, a patient who was ballroom dancing when TGA occurred could only perform older dance routines (Hodges & Warlow, 1990b). Such cases could provide an indirect measure of the extent of RA, as reflected in the time span in which the recent updating occurred.

When automated behavior involves spatial directions, procedural behavior is often disrupted by TGA. During an attack, patients often get confused in a familiar area, or lost when following a familiar route, as illustrated by patients who had difficulty walking to a local bus stop

(Cunningham, 1968), got lost or were found wandering in a familiar area close to home (Amit et al., 1986; Bender, 1956; Palmer, 1986), drove an incorrect or unusual route home (Fisher & Adams, 1964; Hall, 1982; Martin, 1970), and got lost driving to work (Okura, Maeda, & Abe, 1985).

Given the growing interest in the area of procedural memory, it is surprising that only two studies have attempted an empirical evaluation. In one investigation on a single patient, Goldenberg et al. (1991) found that "in contrast to the absence of any learning effect across five trials of verbal learning, the patient markedly improved from the first to the second trial of a procedural learning test which assessed recognition of fragmented digits" (p. 1749), with performance increasing from 67% on Trial 1 to 100% on Trial 2. Unfortunately, there are only four or five sentences concerning the materials and outcome, making any interpretation difficult.

In another study of procedural memory, Kazui et al. (1995) primed 3 TGA patients with word solutions to Kanji letters and then later tested them twice, once during and once following TGA. The mean percents of primed Kanji solutions both during (41%) and after (41%) TGA were higher than the base rate in an unprimed control group (16%). Furthermore, a primed control group showed a solution rate (42%) comparable to the TGA patients' performance during (and after) the episode. Although Kazui et al. provide more detail than Goldenberg et al. (1991), there is insufficient detail on materials, procedures, or analysis to adequately assess the outcome.

In summary, procedural, or implicit, memory appears to be generally preserved during TGA, as is true in other forms of amnesia (Graf, Squire, & Mandler, 1984; Squire, Cohen, & Zouzonis, 1984). The disruption of the spatial component probably reflects an inability to monitor the current context rather than poor procedural function per se (see Working Memory section). The experimental exploration of procedural memory is an important, untapped dimension of TGA. Additional investigation of this topic with more patients and a more comprehensive description of the experimental methodology would be of considerable value (Goldenberg, 1995).

POST-TGA EFFECTS

This section addresses two important and frequently raised questions concerning TGA. The first is the likelihood of experiencing another TGA. Is this malady benign, or are people who have had a TGA at risk for another attack? The second question is whether there is any residual cognitive deficit after the attack, or whether recovery is complete following TGA.

Recurrence

Data on the likelihood of a repeat TGA have been gathered only sporadically, both retrospectively from patients and prospectively from visits to physicians or phone interviews. Combining retrospective and prospective reports

across both case and group studies,¹³ 13% of patients report one additional TGA, 2% claim two other attacks, and 2% report three or more additional TGA episodes. Thus, 17% of TGA patients will experience multiple episodes. This low recurrence rate adds to the puzzle of TGA. As Hodges (1992) notes, "the fact that most attacks are singular ... has been one of the facts most difficult to accommodate into any aetiological theory" (p. 238).

These overall recurrence data are problematic for several reasons. Since retrospective reports are usually unverified, one cannot be sure whether the prior experience is TGA or another memory dysfunction. Also, the follow-up period in prospective studies is often short, averaging 3 years or less in most (62%) studies. Finally, the prospective follow-up period varies considerably across studies, and patient to patient within studies, as the following sample of group studies illustrates: 7–210 months ($M = 67$ months) in Hinge et al. (1986); 1–150 months ($M = 73$ months) in Jensen and Olivarius (1981); 19–51 months ($M = 30$ months) in Matias-Guiú, Casquero, Alvarez, and Bonaventura (1987).

A recurrence statistic that controls for variations in follow-up period is *average annual rate*, or the percent of TGA patients who can expect a recurrence in any given year. This varies from 2% to 5% across 10 studies, with a median of 3%.¹⁴ Although this rate suggests that the chance that a TGA will recur in a given year is relatively small (1 in 33), it is much greater than the incidence of TGA in the general population of older adults (1 in 3,000; see the earlier section on Age). However, a higher report rate is to be expected among TGA patients, who are more sensitized to such an occurrence than are individuals in the general population.

The interval between successive attacks in multiple-TGA patients (71 cases; see Figure 8) suggests that most (56%) happen within 3 years, a figure close to the 24-month (Melo, Ferro, & Paiva, 1994), 29-month (Miller, Petersen, Metter, et al., 1987), 34-month (Frederiks, 1993; Gandolfo et al., 1992), and 36-month (Jacome, 1989) average interattack intervals reported in other studies. Aside from the possible increased risk of a recurrent TGA, the survival rate for TGA patients appears to be normal (Gandolfo et al., 1992).

Cognitive Functioning

Two common assumptions about TGA are that memory performance (1) returns to normal soon after the attack and (2) is not permanently altered by the experience. These assumptions have been challenged in a number of investigations. With respect to the first issue, 12 studies have traced the changes in 17 patients' cognitive functions across multiple testings following TGA (see Table 1). While some research indicates that spatial/performance and verbal abilities may recover as rapidly as 1 day later, most studies show that cognitive function usually takes several days to weeks to fully recover.¹⁵ This slow recovery contrasts with the impression of the patient, as well

Figure 8. Frequency distribution of years between multiple TGA episodes.

as their friends and family, that recovery is complete immediately after the episode (Hodges & Ward, 1989; Regard & Landis, 1984; Stracciari, Rebucci, & Gallassi, 1987). This slow recovery also parallels the continued shrinkage in RA for days following the end of the episode.

With respect to possible permanent cognitive dysfunctions resulting from TGA, there is considerable speculation that TGA results in a permanent, albeit slight, impairment of verbal abilities (Caplan, 1985; Hodges & Oxbury, 1990; Mazzucchi, Moretti, Caffarra, & Parma, 1980; Posteraro, 1988). Summarizing the 29 cases where individuals' verbal and performance I.Q. scores were measured after TGA (Wechsler Adult Intelligence Scale,

WAIS; Wechsler Bellevue Intelligence Scale) supports this speculation: Mean performance (116.8) is significantly higher than mean verbal score (110.9) [$t(28) = 2.80, p < .01$]. Matias-Guiu and Codina (1985) found a similar pattern among their 29 patients (mean performance = 100.4; mean verbal = 92.8), although no statistical test was performed.

In contrast, Hodges and Oxbury (1990) found no difference, and they, as well as others (Matias-Guiu, Bonaventura, Casquero, Calatayud, & Codina, 1987; Stracciari, Rebucci, & Gallassi, 1987), claim that any verbal deficit is due to testing too soon after the TGA, prior to full recovery. Comparing short (<6 months) versus long (9–35 months) TGA-to-test intervals, Gallassi et al. (1993) found comparable performance on verbal tasks at both intervals, but short-delay patients performed poorer than did long-delay patients on spatial learning and complex figure copying and recall. Finally, Faglioni, Scarpa, Colombo, Botti, and Grisanti (1992) also found no evidence of memory impairment in TGA patients relative to controls using a variety of different materials (story, paired-associate list, 10-word list), and they attribute the "permanent" verbal deficit found in some prior studies (Mathew & Meyer, 1974; Mazzucchi et al., 1980; Roman-Campos, Poser, & Wood, 1980; Steinmetz & Vroom, 1972) to contamination of the TGA sample with individuals with other neurological pathologies.

With respect to long-term remote memory, Hodges and Oxbury (1990) compared a large number of TGA patients with controls long after the TGA and found that,

Table 1
Retest Intervals for Cognitive Function in TGA Patients

Study	Retest Interval			
	Hours	Days	Weeks	Months
Cafarra et al. (1981)			1	6
Evans et al. (1993)		2		2
Gallassi et al. (1986)		1, 2, 3		1
Goldenberg et al. (1991)	12	10		1
Gorelick et al. (1988)			1	2
Hodges & Ward (1989)		1	1	1
Meador et al. (1988)				1
Ponsford & Donnan (1980)		1		2
Regard & Landis (1984)	9	1, 2, 3		1
Stillhard et al. (1990)	15	1½, 5	1½, 3	12
Stracciari, Rebucci, & Gallassi (1987)	12	1½, 3		1, 9
Wilson et al. (1980)		1, 2		

Note—All studies except Gorelick et al. (1988) include a test during TGA.

with the Famous Faces Test (Albert et al., 1979), the TGA patients scored more poorly than did normal controls in both naming and recognition, but not personal identification. Thus, although impaired in name memory, TGA patients could supply ancillary information (e.g., occupation) concerning the person. In contrast, on the Famous Events Test (Hodges & Ward, 1989), TGA patients performed comparably to controls on recognition but were significantly worse in dating when the events occurred. Thus, the event-dating difficulty found during TGA may have some permanent residual.

Another facet of this question is whether patients who experience multiple TGA episodes have greater cognitive dysfunction than those who have only one attack. Mathew and Meyer (1974) discovered that all who showed permanent memory impairment were multiple-episode TGA patients, whereas all who showed no memory impairment were single-episode patients. In addition, Gallassi, Stracciari, Morreale, Lorusso, and Ciucci (1988) note greater post-TGA impairments of memory and attention in multiple- compared with single-episode patients, but a later study by Gallassi et al. (1993) found lower performance for multiple-episode patients only for visual, perceptual-motor, and attentional tests, but not verbal tests. In short, there is strong evidence that multiple-episode patients are at greater risk of some degree of permanent cognitive impairment.

Overall, the data suggest that the recovery of memory function takes at least a week, and probably longer. Thus, testing patients too soon after the attack may give the misimpression of a permanent cognitive deficit. Any cognitive disability seems to be more likely with attention and spatial abilities and with multiple-episode patients. Of course, without a pre-TGA memory evaluation, one cannot rule out TGA as a symptom of a preexisting subclinical memory impairment (Cattaino, Pomes, Querin, & Cecotto, 1989). Although there are sufficient indications of a permanent memory dysfunction in some TGA patients (i.e., those with multiple episodes), a definitive answer awaits further investigation. Additional discussion of post-TGA memory function can be found in Columbo and Scarpa (1990).

ETIOLOGY OF TGA

Perhaps the most puzzling dimension of TGA is its cause. Despite an enormous amount of research, analysis, and conjecture, no single cause has been identified. Most of the literature on TGA is devoted to an extensive, almost tedious, rehashing of a few possible causes, a situation probably encouraged by this ambiguity. As Caplan (1985) cogently commented after a review of 485 patients, "Despite numerous case reports...TGA remains a disorder of uncertain cause. In fact, there may be a number of diverse causes which have in common only transient dysfunction of structures critical for memory function" (p. 216). The difficulty in identifying a cause

may be partially attributed to the loose and inconsistent manner in which TGA has been defined (Hodges & Ward, 1989) or to patients' RA for the physical and psychological conditions immediately preceding the episode (Rosenberg, 1979).

Early speculation on the etiology of TGA included dementia (Alzheimer's or Pick's disease), toxic-metabolic disorders (hypoglycemia, acute alcoholism, uremia), aphasia, psychogenic amnesia, and concussion (Cafarra et al., 1981), although these have now been uniformly discarded. Most current speculation centers on three possibilities: epilepsy, migraine, and ischemia (temporary blood flow reduction). Although evidence can be selected to support each, none clearly emerges as the sole and consistent cause of TGA.¹⁶ Given the technical density of the medical arguments and counterarguments in the TGA literature, each position will be briefly summarized, with the interested reader directed to more detailed discussions.

Epilepsy

TGA may reflect a temporary dysfunction of the electrical activity of the cortex, including the structures involved in memory formation and retrieval: the verte-brobasilar and posterior cerebral arterial systems that serve the hippocampus, fornix, amygdala, mamillary bodies, and the mesial and basilar portions of the temporal lobe. The most likely culprit is an epileptic seizure in the temporal lobe, which disrupts the normal consolidation and retrieval processes. Although the original reports on TGA by Fisher and Adams (1958, 1964) suggest epilepsy, support for this position has weakened as evidence has accumulated. An epileptic attack can produce a period of amnesia (Croft, Heathfield, & Swash, 1973; Gallassi & Morreale, 1990), although this is distinct from TGA by the absence of repetitive questioning, frequent episodes of short duration (30 min or less), partial (rather than global) AA, and cessation of the episode following the administration of antiepileptic medication (see Kapur, 1990, 1993).

Evaluation of epilepsy as a possible cause is made more difficult because "epileptic features" (Hodges & Warlow, 1990b) or a history of epilepsy (Erli, Franza, Viscardi, & Defanti, 1988; Gandolfo et al., 1992) are sometimes used as exclusionary criteria in TGA. Interestingly, Hodges and Warlow (1990b) excluded epileptics but found that 9% of patients developed epilepsy during the follow-up period. In addition, a patient was more likely to develop subsequent epilepsy if (1) he/she had had multiple episodes of TGA and (2) the TGA episode was short (< 1 h) rather than long.

While TGA patients are routinely evaluated for epilepsy (and other seizure disorders), only a miniscule number (less than 1%) have the disorder. While some post-TGA EEG analyses have yielded evidence of epileptic activity,¹⁷ most research finds credible evidence lacking.¹⁸ Primavera, Novello, and Stara (1993) further

caution that post-TGA irregularities in EEG may have been created by the TGA episode.

A potentially stronger test of the epileptic hypothesis comes from recordings done during TGA. These outcomes, unfortunately, are as ambiguous as post-TGA analyses. Among case-study reports, evidence of EEG abnormalities have been found about as often as not,¹⁹ although one study did record a seizure during TGA (Tassinari et al., 1991). Among group studies, the evidence is overwhelmingly negative. While one study found abnormal activity during TGA (Heathfield et al., 1973), most have failed to find such evidence.²⁰ Additional negative evidence comes from the serendipitous EEG recording (during routine medical tests) at the onset of a TGA attack (Cole, Gloor, & Kaplan, 1987), which revealed no difference in electrical activity before and after the onset of TGA.

The ambiguity of these data may be due, in part, to measurement difficulties. Many studies supporting the epileptogenic origin lack sufficient technical detail to support their claims (Gallassi & Morreale, 1990) or point to "abnormalities" that are found routinely in normal recordings (Pedley, 1983; Tharp, 1979). Fogelholm, Kivalo, and Bergstrom (1975) further note that EEG abnormalities become progressively more common with age, especially in the range typical of TGA patients (Miller, Yanagihara, Petersen, & Klass, 1987).

It should be emphasized that none who supports an epileptic etiology suggests that it is the exclusive cause of TGA, but rather it is one of several possible causes that accounts for only a small percentage of cases. Additional discussion of epilepsy as a cause of TGA can be found in Gallassi and Morreale (1990), and Stracciari, Ciucci, Bianchedi, and Rebucci (1990) provide a detailed description of how to differentiate TGA from transient epileptic amnesia.

Migraine

Another possible cause of TGA is a migraine attack, which disturbs the electrical activity and/or blood supply (vasodilation/vasoconstriction) in the brain, resulting in a temporary dysfunction of the structures involved with memory formation (Crowell, Stump, Biller, McHenry, & Toole, 1984; Kushner & Gzesh, 1990). Among the case studies, 11% of patients reported migraine, and, across 19 group studies, the percents of migraineurs varies considerably from 4% to 42%.²¹

Some research points to spreading depression of Leao as a neurological disturbance common to both migraine and TGA (Hinge et al., 1986; Hodges & Warlow, 1990a; Nichelli & Menabue, 1988; Olesen & Jorgensen, 1986). According to Olesen and Jorgensen (1986), when a highly emotional experience excites the hippocampus, glutamate is released, which then triggers a spreading neurological depression in the hippocampus, resulting in a temporary shutdown of normal memory activity.

While migraines are suggested as precipitating specific TGA episodes (Cochran, 1984; Dupuis et al., 1987; Jensen, 1980; Olivarius & Jensen, 1979; Santoro et al., 1988; Stracciari & Rebucci, 1986), few make the claim that TGA is caused solely by migraine. As with supporters of epilepsy, these researchers suggest that it is one of several possible causes. One problem with the migraine hypothesis is that the malady is most prevalent between the ages of 12 and 50, which is outside of the age range typical of TGA patients (Frederiks, 1993). For further discussion of the migraine hypothesis, see Caplan, Chedru, Lhermitte, and Mayman (1981), Crowell et al. (1984), Haas (1990), and Kushner and Gzesh (1990).

Ischemia

Perhaps the most viable explanation of TGA is a temporary vascular insufficiency of the temporal lobe structures involved in the formation and storage of new memories, probably in the medial-temporal branches of the basilar artery. Most researchers find it sufficient to advocate the nonspecific disruption of the blood supply (transient ischemic attack, or TIA) unrelated to a structural or functional abnormality (tumor, lesion, concussion, etc.).²²

Cerebral blood flow measurement during TGA, through doppler (rCBF) or tomographic (SPECT, PET) techniques, has consistently verified a reduced blood supply to the temporal lobe.²³ Out of 19 patients who have been measured in this manner, 5 showed bilateral reduction to the temporal lobe, and 7 showed a unilateral reduction (6 on the left and 1 on the right temporal lobe). There were also reductions to the thalamus (3 bilateral, 1 left, 1 right), frontal lobe (1 bilateral, 2 left, 1 right), hippocampus (2 bilateral, 1 right), and occipital lobe (1 bilateral). Thus, the most commonly observed blood supply reduction is to the left temporal lobe, with 11 of 19 patients evaluated exhibiting this pattern. A blood supply insufficiency has also been noted in most patients soon after the TGA episode (Crowell et al., 1984; Fayad, Kain, Hoffer, Smith, & Brass, 1990; Laloux, Brichant, Cauwe, & Decoster, 1992; Matsuda et al., 1993), although Croisile, Trillet, and Laurent (1990) found that this slowly returns to normal 40 days after TGA.

Others have speculated that some structural or functional brain pathology might underlie the temporary vascular insufficiency. This would suggest a higher incidence of stroke among TGA patients than among the general population, but data on this are ambiguous²⁴ (see Ghika & Bogousslavsky, 1990). Others try to document the presence of cerebrovascular risk factors (hypertension, hypercholesterolemia, smoking, etc.) in TGA patients, but large variations across studies in what is included in the set of risk factors make these data relatively uninterpretable (Frederiks, 1993).

On some occasions, a specific malady can be identified as the apparent cause of the temporary reduction in blood supply leading to TGA, and these include heart

dysfunctions of a structural (Crowell et al., 1984; Jackson, Boughner, Bolton, Hopkins, & Barnett, 1985; Shuping, Toole, & Alexander, 1980) or functional (Dugan, Nordgren, & O'Leary, 1981; Fisher & Adams, 1964; Greenlee, Crampton, & Miller, 1975) nature, as well as vascular difficulties, usually involving the carotid artery.²⁵

Everyday behaviors that cause a temporary reduction of cerebral blood supply have also been implicated in precipitating TGA: sudden neck extension or flexion (including whiplash) resulting in temporary arterial constriction (Ardila, 1989; Fogelholm et al., 1975; Hofstad & Gjerde, 1985; Muller & Mase, 1981); hypocapnia, or reduced oxygen supply from the lungs (Martin, 1970), from such activities as "singing lustily" in church (Evans, 1966); a sudden drop in blood pressure caused by rapidly standing from a seated position (Evans, 1966) or a period of quiescence following intense physical activity (Evans, 1966).

Further discussion of the ischemic hypothesis can be found in Ghika and Bogousslavsky (1990) and Scarpa and Columbo (1990).

Other Causes

In a few instances, a TGA can be traced to damage to brain structures due to stroke, cyst, tumor, hydrocephalus, neurosyphilis, and infection²⁶ (see Brillman, 1990). Various drugs have also been implicated in producing TGA, including scopolamine (Ardila & Moreno, 1991), digitalis (Greenlee et al., 1975), and alcohol (Hodges & Warlow, 1990b). Of special interest is clioquinol, a drug used to treat diarrhea (Cras & Martin, 1990; Kaeser, 1984; Mumenthaler, Kaeser, Meyer, & Hess, 1979). Parkin (1987) notes that serious flooding in Japan in 1966 led to the widespread distribution of clioquinol to combat dysentery. This resulted in over 100 reports of a malady similar to TGA, although Kaeser (1984) cautions that these episodes may differ from typical TGA on several dimensions (e.g., younger patients, longer episodes, insidious onset).

Finally, benzodiazepines have supposedly precipitated TGA attacks (Gilbert & Benson, 1972; Morris & Estes, 1987; Pande, 1987; Sandyk, 1985; cf. Trillet, Laurent, & Croisile, 1990). Interestingly, Merriam (1988; Merriam et al., 1992) suggests that emotionally stressful events, which often precede TGA attacks, could release a natural benzodiazepine receptor ligand, which temporarily interferes with hippocampal function. Frederiks (1993) cautions, however, against classifying these drug-induced amnesic episodes as TGA because the episode has a slow onset and recovery, with disturbed consciousness during the attack.

MANAGING THE TGA AND ITS AFTEREFFECTS

Medical assistance during a TGA episode is rare, and drugs designed to enhance memory function (physostigmine, vasopressin, and naloxone) have consistently failed

to alter the course of TGA (Croisile et al., 1990). Antiplatelet aggregation medications have been recommended as a precaution against vascular coagulation during the TGA (Jensen & Olivarius, 1980, 1981; Mathew & Meyer, 1974), and vasoactive drugs have been suggested as a prophylactic measure after the TGA episode (Erokhina, Stakhovskaya, & Sarycheva, 1988), but the efficacy of either treatment has not been evaluated.

Some physicians suggest modified behavioral routines for TGA patients as a precautionary measure. Cartlidge (1991) admonishes his TGA patients to refrain from driving for 6 months after the attack and reports that one English licensing authority (Swansea) puts a 1-year post-attack restriction on vocational drivers' licenses of TGA patients. Similarly, Palmer (1986) advises his TGA patients to refrain from complex business activities for 1 month after the attack, while Hodges and Warlow (1990b) suggest that TGA patients who have had either several TGA episodes or attacks of very short duration (less than 1 h) be advised against driving for 12 months after the episode due to an increased risk of developing epilepsy. Others suggest that TGA is benign except where other cerebrovascular risk factors are present, in which case more careful follow-up and therapy are indicated (Jensen & Olivarius, 1981).

Legal Implications

Although most patients carry out routine activities with little difficulty during TGA, some professionals raise a concern that TGA patients might engage in legally problematic actions. Rolak (1984) describes an altercation between a person experiencing a TGA and a park owner over fees and regulations: "The argument became so heated the owner brandished a knife and threatened the patient. The patient retrieved a rifle... and held the owner at bay until the police arrived.... The rifle was never fired and the patient was not charged with any crime." As expected, the next morning the patient had no memory of the conflict.

Such incidents are extremely rare, and most evidence suggests that patients are so distracted by their mnemonic dysfunction that they become reflective and confused rather than aggressive and hostile. Despite the absence of historical precedent, Stracciari, Rebucci, and Ciucci (1989) raise a flag concerning the medicolegal aspects of TGA, and Parwatikar (1990) provides a thorough overview of the possible legal implications for a patient accused of committing a crime during an episode (see Suranyi, 1983). At the very least, the status of TGA as a psychological dysfunction should be formally recognized by inclusion in the Diagnostic and Statistical Manual, or DSM-IV (Caine, 1993).

LOOKING FORWARD

TGA has considerable potential as a crucible within which to examine various dimensions of memory in temporarily dysfunctional individuals. Particularly exciting

is the prospect of using amnesic individuals as their own controls to examine both the loss and the subsequent recovery of various memory functions. A similar line of research involves the pharmacological induction of a temporary amnesic state by the use of drugs in the benzodiazepine family (Danion, Zimmerman, Willard-Schroeder, Grange, & Singer, 1989; Fang, Hinrichs, & Ghoneim, 1987; Polster, 1993; Polster, McCarthy, O'Sullivan, Gray, & Park, 1993). However, participants in such a drugged state are encumbered by a general diminution of cognitive skills, whereas patients experiencing TGA appear to be fully functional with the exception of the mnemonic deficit.

For this unique research opportunity to be fully realized, changes must be made in the way TGA episodes are recorded and evaluated. There is a serious lack of consistency in reporting TGA episodes with respect to demographic information, inclusion and exclusion criteria, physical and psychological precursors of the TGA episode, repeated questions during TGA, assessment of memory dysfunction, and the post-TGA follow-up. This inconsistency stems primarily from the nascent stage of research, with much of the data collected indirectly (by relatives) and after the attack (retrospectively) and guided by a medical focus on the biological etiology of the dysfunction. Researchers are often interested in a specific issue related to TGA (does it happen in children? is it related to migraine attacks? is it likely to recur?) and ignore a rich variety of behavioral and psychological data not relevant to their research question. Since most articles emanate from the medical domain with a focus on a possible etiology of TGA, there is relatively little attention to documenting the variety of behaviors preceding, during, and following the attack. Perhaps the most striking lacuna in the TGA literature is the paucity of detail on memory function, given that the disability is primarily mnemonic.

In short, there needs to be a paradigm shift away from an explanatory and toward a descriptive approach to TGA. A more complete and standardized set of guidelines for data collection, presented below, could help reframe this research within a psychological perspective.

Background History

More extensive background information is needed on each patient, including age, gender, occupation, socioeconomic status, educational background, ethnicity, and race. Does the patient have a history of hypertension, migraine, epilepsy, or head trauma, and was he/she taking any medication(s) at the time of the attack? A thorough record of both mundane (e.g., hot bath) and unusual (e.g., death of a friend) preceding events should be made. After recovery, the TGA patient should receive a questionnaire regarding current life stressors that existed preceding the attack, including physical health problems, emotional difficulties, family relationships, professional activities, sexual problems, and financial issues. Rela-

tives or close friends of the patient should also be given a questionnaire addressing the behavior and circumstances of the patient prior to TGA: Has the patient been under unusual stress? Have you noticed any recent changes in the patient's behavior? Did anything unusual happen to the patient just prior to TGA? Both the patient and the relative/friend questionnaires should have two sections, covering both distal (previous 3 months) and proximal (prior day) periods preceding the attack.

Exclusionary criteria. The exclusionary criteria for identifying TGA episodes (Caplan, 1985) usually involve such things as episode length restrictions, presence of RA and repeated questions, and witnesses being present. Even these simple inclusion/exclusion criteria are often adhered to in a selective or haphazard manner. For instance, in 29 studies that explicitly state their selection criteria (rather than simply indicating that they follow Caplan, 1985), there is only moderate compliance: 62% require that the attack be witnessed, 52% have the dysfunction limited to repeated questions and amnesia, and 69% require that the amnesia last between several hours to 1 day. In the future, these criteria should be noted but not used to eliminate cases from the database. Given our current lack of understanding about TGA, a better approach would be to loosen the restrictions but list the exceptions.

Documenting the Episode

A thorough record of the attack should be made, including when it started (day of the week, time of day, date, month), where the patient was when the attack began and for the short period prior to it, who was with the patient, and what the patient was doing at the onset of TGA, no matter how ordinary. In the absence of unusual or dramatic events prior to TGA, researchers frequently conclude that there were no special circumstances (Vohanka & Zouhar, 1988). In fact, less than a third of case studies report such information for just this reason. However, the key to understanding TGA may lie in a careful analysis of the collage of these preceding events and behaviors. Where possible, an audiotape or videotape recording of the episode would be useful to document the patient's affective, verbal, and physical behaviors. If the patient is examined by a physician/researcher, the time from the beginning of the attack to the beginning of the examination/testing should be noted, as well as the duration of the evaluation.

Anterograde amnesia. A standardized set of verbal and nonverbal instruments should be developed to evaluate the encoding/retrieval difficulties during the TGA. To document the time course of the gradual improvement in the amnesic state, a short battery of cognitive tests (e.g., forward and backward digit span; 10-word list tested after 1 min) should be readministered at regular (half-hour) intervals during the attack.

A complete log should be made of the frequency, timing, and content of repeated questions and behaviors. The cycle of repeated questions could provide a naturalistic avenue to evaluate the temporal extent of short-term

memory, with the time interval between cycles of repeated questions as an index. Furthermore, given that there is a gradual clearing of the period of AA, is there an increase in the interval between repeated questions over the course of TGA to reflect this gradual resolution? What point in the episode do the repeated questions (behaviors) cease?

The existence of repeated questions should not be used as an exclusionary criterion (Caplan, 1986b), because some patients may not vocalize their confusion. Also, while there are very few reports of repeated behaviors during TGA (5% of case studies), it is unclear whether such data are not recorded because of their apparently peripheral nature or whether such behaviors are actually this rare. In any case, such information could provide a better understanding of prospective behavior.

Retrograde amnesia. In the vast majority of the case studies, the extent of RA is determined informally through lacunae in the patients' recollections (i.e., they do not remember a wedding, death of a relative, birth of a grandchild, purchase of a car). To evaluate dense RA during TGA, standardized instruments should be routinely included (personal and cultural; Presidents test), as well as the question, "What was the last thing you remember prior to the attack?" This should be readministered at regular (half-hour) intervals during TGA to document the course of shrinking RA. While it is often reported that there was no residual RA after TGA, this is usually done through an informal query of the patient. Relative to the inordinately large period of RA during TGA (months to years), a shrinkage to an hour or less might be interpreted as a "complete" resolution by contrast. The evaluation of final RA should be delayed until at least 1 week following TGA, given that RA may continue to shrink following the episode. Finally, precautions should be taken to evaluate the extent to which the patient's pre-TGA memories have been implanted by others after the episode. Such an evaluation of RA and its posttrauma shrinkage with TGA patients could be especially enlightening, given that the cognitive confusions typical of head-trauma patients are not present in TGA patients.

Procedural memory. TGA presents a unique opportunity to evaluate implicit memory function in "amnesic" patients who will soon recover. While a few researchers have attempted such an evaluation (Ghidoni, Pattacini, & Previdi, 1988; Goldenberg et al., 1991), these have been neither extensive nor systematic. During the episode, patients should be administered a set of verbal (words, trivia information) or pictorial material. Following input, explicit (recognition) and implicit (fragment completion) tests should be given at regular intervals during and following the episode, with each test consisting of a mix of repeated and new items.

Procedural memory could also be studied through an analysis of the repeated questions asked during TGA. It is often reported that the patient repeats the same question(s) with the same inflection and in the same order (if more than one). With no memory of the prior question(s)

or answer(s), it is impressive that patients repeatedly duplicate the same query. A thorough analysis of this unique form of "self-priming" could provide a valuable tool to evaluate procedural memory function.

Metamemory. A rich, untapped, source of information concerning the psychological dimensions of TGA is the patients' perception of their current circumstances. A structured query concerning the patients' impressions of their cognitive functioning should be administered during TGA, including their perception of the present circumstances (what are you thinking about?), emotional reaction to their encoding failure (what are you feeling?), subjective impression of how their learning and retrieval are impaired (are you confused? does there appear to be a memory void? how familiar are your surroundings?), reactions to the typical time and space distortions, and impression of the quality of their remote memory (hazy? patchy? missing?). As illustrated by the rich detail provided by Stevens and Ammerman's (1974) patient (cited earlier), a considerable amount of untapped information may be available concerning the metacognitions of TGA patients.

GENERAL SUMMARY

TGA is a mnemonic dysfunction characterized by the sudden onset of a temporary inability to retain new information. From information on 3,037 patients described in 201 articles, this disability strikes individuals mainly in their 50s and 60s, and it affects women slightly more often than men. The modal TGA attack is 2 h, and it is most likely to occur during the morning hours and in winter (holiday) months. RA is typically experienced with TGA, extending back 1–2 days during the episode and back 1 h when the episode resolves. The patient retains self-orientation but is disoriented to space, time, and present situation, and he/she asks repeated questions that reflect this disorientation. Physical symptoms are rare and are usually restricted to headache, dizziness, and nausea. Psychological symptoms include confusion and arousal, along with lethargy. While retention of information past several minutes is universally absent during the attack, working memory is generally preserved, except for some difficulties with visual/spatial information. Both procedural and semantic memory are also preserved during TGA, although there is a problem with spatial orientation (getting lost) and temporal dating (confusing event order) preceding TGA.

Recovery from the period of AA is gradual, extending over several hours. About 1 in 6 patients experience another TGA episode. Complete recovery of cognitive function usually takes weeks, even though patients feel fully recovered immediately after the episode. There may be a slight, permanent erosion of verbal and spatial memory abilities following the TGA, although further research is needed. A diverse set of circumstances precedes TGA, but a common thread appears to be a moderate emotional

or physical stressor or an abrupt change in the psychological or physical environment. Although migraine and epilepsy have been cited as causes of TGA, the most likely explanation is a transient disruption in the posterior cerebral blood supply, probably in the medial-temporal branches of the basilar artery.

In the broad sense, TGA provides a natural laboratory to compare implicit and explicit memory in temporary amnesics (see Wilson et al., 1980). Martin (1970) has even suggested that, since the consequences of TGA are apparently benign, the phenomenon could be studied by having the patient duplicate the triggering event (a cold swim in the ocean) to see if a similar attack is elicited. Although this approach is ethically questionable, individuals have experienced a repeat TGA under similar conditions. For instance, Frederiks (1990) reports a man who experienced TGA on three separate occasions during exciting card games. In any case, TGA has the potential to provide unique insights in the areas of psychosomatics, emotion, cognition, geriatrics, neuropsychology, and memory. As Mazzucci (1988) cogently remarked, "the study of a so pure form of amnesia, which exhibits all the characteristics of an experiment conducted by nature, could provide a greater number of answers to general questions about memory" (p. 14).

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NOTES

1. Caplan (1983, 1986a, 1986b); Jain et al. (1989); Matias-Guiu and Codina (1986); Mazzucchi (1988); Meador et al. (1986); Stracciari et al. (1989).
2. 2.8 (Miller, Petersen, Metter, Millikan, & Yanagihara, 1984); 3.0 (Matias-Guiu, Blanquer, Falip, Oltra, & Martin, 1992); 3.4 (Hodges & Warlow, 1990a); 5.2 (Miller, Petersen, Metter, et al., 1992); 7.5 (Hodges & Warlow, 1990b); and 10 (Koski & Marttila, 1990).
3. TGA patient is a physician (Donaldson, 1985; Fisher & Adams, 1964; Klawans, 1988; Mazzucchi et al., 1980; Morris & Estes, 1987), or the physician was the son (Nishida et al., 1990; Tanabe et al., 1991), daughter (Bender, 1960; Gorelick et al., 1988), husband (Fisher & Adams, 1958; Mumenthaler et al., 1979), father (Mumenthaler et al., 1979), or other relative (Fisher & Adams, 1964; Whitty, 1977) of the TGA patient.
4. Cafarra et al. (1981); Donaldson (1985); Evans, Wilson, Wraight, & Hodges (1993); Fisher and Adams (1964); Gallassi et al. (1986); Goldenberg et al. (1991); Gordon and Marin (1979); Halsey (1967); Hodges and Ward (1989); Lin, Liu, Yeh, Wang, and Liu (1993); Meador et al. (1988); Merriam et al. (1992); Patten (1971); Ponsford and Donnan (1980); Regard and Landis (1984); Roman-Campos et al. (1980); Stracciari, Rebutti, & Gallassi (1987); Wilson et al. (1980).
5. Normal range (Cafarra et al., 1981; Donaldson, 1985; Evans et al., 1993; Fisher & Adams, 1964; Goldenberg et al., 1991; Hodges & Ward, 1989; Meador et al., 1988; Regard & Landis, 1984; Roman-Campos et al., 1980); below normal range (Gallassi et al., 1986; Halsey, 1967; Hodges & Ward, 1989; Patten, 1971; Stracciari, Rebutti, & Gallassi, 1987).
6. Cafarra et al. (1981); Dinsmore and Callender (1983); Fisher and Adams (1964); Gordon and Marin (1979); Gorelick et al. (1988); Roman-Campos et al. (1980).

7. Comparable to controls (Berthier & Starkstein, 1990; Goldenberg et al., 1991; Hodges & Ward, 1989; Meador et al., 1988; Regard & Landis, 1984; Stracciari, Rebutti, & Gallassi, 1987); impaired relative to controls (Kritchevsky & Squire, 1989; Kritchevsky et al., 1988).
8. Word lists (Cafarra et al., 1981; Gallassi et al., 1986; Goldenberg et al., 1991; Gordon & Marin, 1979; Regard & Landis, 1984; Stillhard et al., 1990; Stracciari, Rebutti, & Gallassi, 1987; Wilson et al., 1980); numbers and letters (Gordon & Marin, 1979); faces (Gorelick et al., 1988); visual figures (Goldenberg et al., 1991; Hodges & Ward, 1989; Kritchevsky & Squire, 1989; Kritchevsky et al., 1988; Meador et al., 1988; Regard & Landis, 1984; Stracciari, Rebutti, & Gallassi, 1987); objects (Gordon & Marin, 1979; Gorelick et al., 1988; Hodges & Ward, 1989; Roman-Campos et al., 1980; Shuttleworth & Wise, 1973); stories (Gallassi et al., 1986; Hodges & Ward, 1989; Kritchevsky & Squire, 1989; Kritchevsky et al., 1988); paired-associate lists (Hodges & Ward, 1989); spatial location (Gorelick et al., 1988).
9. Fisher and Adams (1964); Kritchevsky and Squire (1989); Kritchevsky et al. (1988); Man-Son-Hing (1968); Okada et al. (1987); Patten (1971); Paulson (1981); Shuttleworth and Morris (1966); Tanabe et al. (1991).
10. Cafarra et al., 1981; Cole et al. (1987); Cuningham (1968); Fisher and Adams (1964); Giang and Kido (1989); Patten (1971); Stracciari, Rebutti, & Gallassi (1987).
11. Bolwig (1968); Donaldson (1985); Gallassi et al. (1986); Hodges and Ward (1989); Markowitsch (1983); Patten (1971); Ponsford and Donnan (1980); Regard and Landis (1984); Stillhard et al. (1990); Tanabe et al. (1991).
12. Simple analogies (Gallassi et al., 1986; Stracciari, Rebutti, & Gallassi, 1987); proverb interpretation (Gordon & Marin, 1979; Gorelick et al., 1988); object naming (Amit et al., 1986; Donaldson, 1985; Gorelick et al., 1988; Kritchevsky & Squire, 1989; Regard & Landis, 1984); Stroop color-word naming (Regard & Landis, 1984); providing the rules of contract bridge (Gordon & Marin, 1979); WAIS subscales (Berthier & Starkstein, 1990; Ponsford & Donnan, 1980); successive category generation (Cafarra et al., 1981; Gordon & Marin, 1979; Wilson et al., 1980).
13. 3% (Muller, 1990); 4% (Hinge & Jensen, 1990); 5% (Columbo & Scarpa, 1988; Hinge et al., 1986); 6% (Fisher & Adams, 1964); 7% (Kushner & Hauser, 1985); 8% (Hodges & Warlow, 1990b); 10% (Guidotti, Anzalone, Morabito, & Landi, 1989); 12% (Muller, 1990); 13% (Fisher, 1982; Melo, Ferro, & Paiva, 1994); 14% (Mazzucchi et al., 1980); 15% (Matias-Guiu, Casquero et al., 1987); 16% (Rollinson, 1978); 18% (Accurti & Proli, 1988; Caplan, 1985; Shuping, Rollinson, & Toole, 1980); 19% (Gandolfo et al., 1992; Melo et al., 1992); 22% (Hinge et al., 1986); 23% (Muller & Mase, 1981); 24% (Miller, Petersen, Metter, Millikan, & Yanagihara, 1987); 25% (Cattaino, Querin, Pomes, & Piazza, 1984; Laurent et al., 1990; Nausieda & Sherman, 1979); 26% (Frederiks, 1990); 30% (Rowan & Protass, 1974); 37% (Fogelholm et al., 1975); 38% (Guyotat & Courjan, 1956, cited in Poser & Ziegler, 1960); 40% (Tukel, 1977); 42% (Heathfield et al., 1973); 57% (Mathew & Meyer, 1974).
14. 1.9% (Columbo & Scarpa, 1988); 2.5% (Hinge & Jensen, 1990; Muller, 1990); 2.7% (Gandolfo et al., 1992); 3.0% (Guidotti et al., 1989; Hodges & Warlow, 1990b); 3.3% (Hofstad & Gjerde, 1985); 3.4% (Miller, Petersen, Metter et al., 1987); 4.2% (Matias-Guiu, Casquero, Alvarez, & Bonaventura, 1987); 4.7% (Hinge et al., 1986).
15. Within one day (Hodges & Ward, 1989; Stillhard et al., 1990; Stracciari, Rebutti, & Gallassi, 1987; Wilson et al., 1980); several days to weeks (cf. Hodges & Ward, 1989; Gallassi et al., 1986; Ghidoni et al., 1988; Laine, Portin, & Koski, 1984; Mazzucchi et al., 1980; Mazzucchi & Parma, 1990; Ponsford & Donnan, 1980; Regard & Landis, 1984; Roman-Campos et al., 1980; Stillhard et al., 1990; Stracciari, Ciucci, & Bissi, 1987; Stracciari, Rebutti, & Gallassi, 1987; Zinelli et al., 1988).
16. Croisile et al. (1990); Frederiks (1990); Fujii, Sadoshima, and Fujishima (1990); Hinge and Jensen (1990); Melo et al. (1992); Palmer (1986).
17. Cantor (1971); Deisenhammer (1981); Evans (1966); Fisher (1982); Gilbert (1978); Greene and Bennett (1974); Heathfield et al. (1973); Hodges and Warlow (1990a, 1990b); Joynt, Satran, and Charlton (1973); Kennedy, Ratnam, Freeman, and Ziegler (1979); Mathew and Meyer (1974); Primavera et al. (1993); Rowan and Protass (1974); Steinmetz and Vroom (1972).
18. Case studies (Araga et al., 1989; Collins & Freeman, 1986; Croft et al., 1973; Shuttleworth & Morris, 1966); group studies (Cattaino et al., 1984; Ciucci et al., 1988; Fogelholm et al., 1975; Frederiks, 1990, 1993; Jacome, 1989, 1990; Jaffe & Bender, 1966; Jensen & Olivarius, 1981; Kushner & Hauser, 1985; Laloux et al., 1992; Lou, 1968; Man-Son-Hing, 1968; Mathew & Meyer, 1974; Miller, Yanagihara, & Klaus, 1986; Miller, Yanagihara, Petersen, & Klass, 1987; Nausieda, 1979; Shuping, Rollinson, & Toole, 1980).
19. EEG abnormalities found (Dugan et al., 1981; Tassinari et al., 1991; Tharp, 1969); EEG abnormalities not found (Bender, 1960; Cole et al., 1987; Collins & Freeman, 1986; Hodges & Ward, 1989).
20. Guidotti et al. (1989); Jaffe and Bender (1966); Man-Son-Hing (1968); Melo et al. (1992); Miller et al. (1986); Miller, Yanagihara, et al. (1987).
21. 4% (Columbo & Scarpa, 1988; Jacome, 1989; Zinelli et al., 1988); 6% (Kushner & Hauser, 1985); 8% (Accurti & Proli, 1988); 13% (Guidotti et al., 1989); 14% (Miller, Petersen, Metter, et al., 1987); 19% (Ciucci et al., 1988); 20% (Hinge et al., 1986; Matias-Guiu, Pico, Bonaventura, Martin, & Monasterio, 1989); 23% (Matias-Guiu, Colomer, Segura, & Codina, 1986); 25% (Melo et al., 1992); 30% (Hodges & Warlow, 1990b); 32% (Laine et al., 1984); 39% (Jensen & Olivarius, 1981); 42% (Crowell et al., 1984).
22. Ahmed (1978); Bender (1956, 1960); Bogousslavsky and Regli (1988); Bolwig (1968); Caplan (1985); Cattaino et al. (1984); Evans (1966); Fisher and Adams (1964); Fogelholm et al. (1975); Frederiks (1990, 1993); Fujii et al. (1990); Heathfield et al. (1973); Jensen and Olivarius (1980); Kushner and Hauser (1985); Ladurner, Skvarc, and Sager (1982); Longridge, Hachinski, and Barber (1979); Man-Son-Hing (1968); Markowitsch (1983); Martin (1970); Mathew and Meyer (1974); Matias-Guiu et al. (1986); Mazzucchi (1988); Milandre, Donnet, Rumeau, Ali Cherif, and Khalil (1990); Olivarius and Jensen (1979); Ponsford and Donnan (1980); Poser and Ziegler (1960); Rowan and Protass (1979); Shuping, Rollinson, and Toole (1980); Shuping, Toole, and Alexander (1980); Stevens and Ammerman (1974); Tharp (1969).
23. Baron et al. (1994); Croisile et al. (1990); Evans et al. (1993); Goldenberg (1995); Goldenberg et al. (1991); Hodges (1994); Kazui et al. (1995); Laloux et al. (1992); Lin et al. (1993); Matsuda et al. (1993); Sakashita, Sugimoto, Taki, and Matsuda (1993); Stillhard et al. (1990); Tanabe et al. (1991); Volpe, Herscovitch, Raichle, Hirst, & Gazzaniga (1983).
24. Low incidence (Crowell et al., 1984; Fisher & Adams, 1964; Frederiks, 1993; Hinge et al., 1986; Nausieda & Sherman, 1979; Shuping, Rollinson, & Toole, 1980); high incidence (Jensen & Olivarius, 1980; Mathew & Meyer, 1974).
25. Occlusion (Araga et al., 1989; Ladurner et al., 1982); aneurysm (Rosenberg, 1979); lesions (Lattera, Gebarski, & Sackellares, 1988; Poser & Ziegler, 1960; Robinson & Long, 1972; Rimpl & Rimpl, 1979); embolism (Kadota, Irino, Nishide, Kaneda, & Hashimoto, 1981; Milandre et al., 1990; Shuttleworth & Wise, 1973; Steinmetz & Vroom, 1972); hypertension (Ghidoni et al., 1988); platelet aggregation (Matias-Guiu et al., 1989); hematoma (Chatham & Brillman, 1985; Moonis et al., 1988); high cholesterol (Matias-Guiu, Davalos, Antem, & Codina, 1984); scleroderma (Nishida et al., 1990); hemorrhage (Bogousslavsky & Regli, 1988; Jacome & Yanez, 1988; Landi, Giusti, & Guidotti, 1982; Sandyk, 1984); high hematocrit levels (Matais-Guiu, Masague, & Codina, 1985); angiography (Caplan, 1985; Cochran, 1983; Cochran, Morrell, Huckman, & Cochran, 1982; Frank, 1981, as cited in Markowitsch, 1983; Giang & Kido, 1989; Haas, 1983; Juni et al., 1992; Minuk, Melancon, Tampieri, & Ethier, 1990; Pexman & Coates, 1983; Reichter, Belt, & Stevens, 1986; Shuttleworth & Wise, 1973; Wales & Nov, 1981).
26. Stroke (Bender, 1956; Bogousslavsky & Regli, 1988; Ghika & Bogousslavsky, 1990; Halsey, 1967; Heathfield et al., 1973; Logan & Sherman, 1983; Mathew & Meyer, 1974); cyst (Stracciari, Ciucci, & Bissi, 1987); tumor (Araga et al., 1989; Cattaino et al., 1989; Collins & Freeman, 1986; Findler, Feinsod, Lijovetzky, & Hadani, 1983; Hartley, Heilman, & Garcia-Bengochea, 1974; Lisak & Zimmerman, 1977; Matias-Guiu et al., 1986; Meador et al., 1985; Shuping, Toole, & Alexander,

1980); hydrocephalus (Giroud et al., 1987); neurosyphilis (Hall, 1982); infection (Nishida et al., 1990; Pommer, Pilz, & Harrer, 1983).

APPENDIX

Proximal Experiences Preceding TGA by Percent and Frequency (in Parentheses)

Routine Activities: 10% (62)

awakening in the morning (17); eating a meal (13); watching a movie (11); talking on the phone (5); watching television (2); sitting (2); sunbathing (2); conversing (2); attending church (1); praying on knees (1); standing up suddenly (1); discussing a manuscript (1); getting dressed (1); getting a haircut (1); riding a bus (1); riding a subway (1)

Emotional Stress: 15% (95)

general emotional stress (49); intense argument (5); exciting card game (4); arriving home from work (4); visiting a friend (4); sudden illness/injury to a friend or relative (4); visiting a cemetery (2); business meeting (2); watching a sporting event (2); testifying in court (2); public speech (2); getting robbed (2); attending a memorial service for a spouse (1); receiving unpleasant news (1); attending a party (1); tearful reunion with a child (1); business foreclosure (1); relative's funeral (1); death of spouse (1); getting fired (1); musical performance (1); harsh criticism from boss (1); obscene phone call (1); witness to a car accident (1); watching challenger explosion (1)

Light to Moderate Physical Stress: 41% (268)

sexual intercourse (52); driving (30); bathing in hot water (29); housecleaning (25); showering in hot water (20); gardening (20); at work (19); walking (18); shopping (9);

defecating (8); neck extension (6); head retroflexion (5); vertigo (3); working on a car (3); preparing dinner (2); playing the piano (2); fishing (2); whiplash (2); hyperventilation (2); preparing for a trip (2); singing (1); playing shuffleboard (1); cutting grass (1); hunting (1); carpentry work (1); horseback riding (1); inspecting real estate (1); coughing spell (1); push-starting a moped (1)

Heavy Physical Exertion: 19% (125)

general farm work (44); general physical exertion (32); swimming in cold water (13); athletic exercise (11); bicycling (11); chopping wood (8); vomiting (2); pitching hay (1); gymnastics (1); playing football (1); installing a fireplace (1)

Physical Pain: 10% (63)

nonconcussive mild head injury (35); muscular pain (8); migraine attack (7); headache (4); abdominal pain (3); chest pain (2); sinus pain (2); dental extraction (1); childbirth (1)

Medical Procedures: 5% (35)

angiography (17); general medical procedures (2); hospitalization (2); spinal anesthesia (2); trigeminal ganglion stimulation (2); pulmonary function test (2); treadmill test (2); bronchoscopy (1); EEG exam (1); cystoscopy (1); cervical manipulation (1); excretory urography (1); nasogastric tube insertion (1)

Drugs: 1% (8)

chloroquine (3); diazepam (1); morphine (1); lorazepam (1); triazolam (1); clioquinol (1)

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