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Accelerated Time Experience after Left Frontal Cortex Lesion

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Abstract

We report a patient, BW, with an accelerated time experience as a result of a glioblastoma in his left superior prefrontal cortex. He experienced external events as seeming to occur at a much faster rate than before the lesion, but he showed normal awareness of time, place, and identity. He displayed behavioral slowness, some aphasia, right hemispatial neglect, and other neuropsychological problems typical of left frontal damage. When asked to produce 60 s durations, BW's mean production was extremely long (286.0 s). This finding is consistent with the notion that subjective duration had decreased relative to objective duration. Several explanations for the findings are discussed, including the possibility that the rate of an internal pacemaker was greatly slowed.

Introduction

Psychological time involves several aspects: simultaneity and successiveness, movement timing, duration experience, and temporal orientation (Block, 1979, 1990a; Nichelli, 1993). According to the contemporary psychological view, these different aspects of psychological time are subserved by various attentional, memory, and other cognitive processes (Block, 1990b). Explaining behavioral and cognitive timing may also require assuming an internal-clock mechanism. Scalar timing theory proposes that this timing mechanism consists of a pacemaker (which produces pulses), a switch, and an accumulator, with a comparison made between pulse totals in working memory and in reference memory (for a review, see Church, 1989).

Although scalar timing theory proposes a single mechanism, several partially dissociable brain systems may be involved in different temporal experiences, judgments, and behaviors. A recent review of neuropsychological, psychopharmacological, and cognitive neuroscience research concluded that anatomically and functionally separate, yet interconnected, brain areas subserve the functioning of these different systems (Block, 1996). Studies of neuropsychological patients showing alterations in time-related judgments and behaviors have provided crucial evidence on the role of various cortical and subcortical structures in the experience of time, although the functioning and interrelationships of these structures are still poorly understood.

The dorsolateral prefrontal cortex may subserve the temporal organization of ongoing behavior, including timing short durations (experiencing duration in passing), temporally ordering events, and encoding the relative recency of events. Häfner (1953) reported two patients in whom fragment wounds had damaged the frontal cortex. One of them reported disordered feeling of time and was not able to know the time of day without a wristwatch. The other also reported a disordered experience of time, mainly an inability of time ordering or scheduling. The dorsolateral prefrontal cortex may contain specialized neural circuits that are part of an internal-clock mechanism which is necessary for judgment of durations in the range of seconds to minutes (for relevant discussion, see Church, 1989; Block, 1996; Block and Zakay, 1996). Patients with lesions of the dorsolateral prefrontal cortex, specifically in and around Brodmann's area 46, are impaired in judging which of two remembered events occurred more recently (Milner *et al.*, 1985, 1990, 1991) and in organizing temporal sequences of behavior (Fuster, 1985, 1993, 1995). Frontal patients also show decreased accuracy in discriminating short durations. Nichelli *et al.* (1995) interpreted this finding in terms of an impaired reference memory

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system for time intervals. In short, the dorsolateral prefrontal cortex seems to be crucially involved in temporal order judgment and sequential behavior, as well as in short duration experience, although its exact role in these phenomena remains unclear (Nichelli, 1993).

The long-term encoding of temporal information requires intact functioning of the hippocampus and probably other medial temporal lobe structures, apparently working in conjunction with information supplied by the prefrontal cortex. Because medial temporal lobe structures are involved in long-term memory for events, they are also involved in temporal sequencing of memory. Temporal lobe patients, especially those with damage to the hippocampus, apparently cannot encode new long-term memories for events, including information about the duration of past events, as well as details concerning the current context (time and place). They show abnormally short reproductions of durations greater than ~5–15 s (Richards, 1973; Williams *et al.*, 1989).

Temporal orientation relies partly on semantic memory areas, especially in the posterior frontal cortex, the inferior parietal cortex, and the temporal cortex of the left cerebral hemisphere. Subcortical structures may also be involved in temporal orientation. Some patients who had received bilateral lesions of the dorsomedial nucleus of the thalamus displayed an impairment of temporal orientation that is sometimes called chronotaraxis. Chronotaraxis may appear as an inability to know the season, the date, the day of week, or the time of day. Spiegel, Wycis, and their colleagues (Spiegel *et al.*, 1955, 1956; Spiegel and Wycis, 1967, 1968) found in 19 of 30 thalamotomy patients a more or less pronounced chronotaraxis, in four patients impaired orientation in time and place, and in seven no such disorders. In most patients, the chronotaraxis lasted only a few days or weeks, but in one case it lasted 6 months. Spiegel *et al.* (1956) maintained that the chronotaraxis was not simply a result of diffuse cerebral impairment, although they acknowledged that most patients showed disturbances of memory for past as well as recent events. The prefrontal cortex is the primary projection area of the dorsomedial nucleus of the thalamus, and the prefrontal cortex also projects back to thalamic nuclei (van Buren and Borke, 1972; Markowitsch and Pritzel, 1979; Markowitsch *et al.*, 1980; Llinás and Pare, 1991). Markowitsch (1992) suggested that all temporal experience may be mediated by the dorsomedial nucleus of the thalamus.

Van der Horst (1928) has been credited as the first person to characterize Korsakoff's syndrome as involving a loss of 'the temporal signs of the events' (Markowitsch, 1992, p. 89). Van der Horst (1932) also claimed that 'the actual comprehension of time and time as a principle of order are disturbed' (p. 83). Some patients described in older case studies also reported disorders of time consciousness (Grünthal, 1932; Becker and Sternbach, 1953). One of Becker and Sternbach's patients reported a feeling of time shrinkage, whereas another reported that time had widened into endlessness. Williams and Zangwill (1950) reported a Korsakoff's patient who experienced a minute as being a quarter of an hour and who described very recent events as if they happened in the distant past. However, Benton *et al.* (1964) found that temporal orientation (knowing the date and time of day) and duration estimation (producing a 60 s duration) are not correlated, implying that partially independent processes are involved.

The cerebellum has also been implicated in aspects of timing and perhaps also time perception (see Nichelli, 1993; Block, 1996). Lateral cerebellar damage produces deficits in timing movement execution and perhaps also in performing temporally predictive computations in various behavioral, perceptual, and cognitive situations (Ivry and Keele, 1989). Cortical structures (e.g. the supplementary motor cortex) subserve the timing of more novel movements, such as producing a rhythm.

Several neuropsychological patients have reported a striking disorder of temporal experience. Häfner (1953) recalled the initial observation of this phenomenon: in 1919, Klien had described an 8-year-old boy suffering from a fever who experienced a perception of accelerated motion of events. Payk (1977) reviewed various case studies of the *Zeitrafferphänomen – accelerated time phenomenon.* (A *Zeitraffer* is a mechanical apparatus used to accelerate apparent motion in a film, as in time-lapse cinematography, so common translations of *Zeitrafferphänomen* or *Zeitraffer-Erlebnis* usually include the ideas of accelerated motion of events or accelerated experience of time.) In five such Zeitraffer patients (six including the patient in Hoff and Pötzl, 1938) an accelerated time experience resulted from right hemisphere parietal–occipital damage (Hoff and Pötzl, 1934; Pichler, 1943; Wagner, 1943; Pötzl, 1951; Gloning *et al.*, 1955), whereas in another patient it resulted from left hemisphere parietal–occipital damage (Ahrens, 1943). In three patients, it was associated with diencephalic damage (Becker and Sternbach, 1953; Häfner, 1953, 1954; Gloning and Weingarten, 1954); in two cases, with encephalitis; and in one case, with *petit mal* epilepsy. Several patients apparently only reported an accelerated motion phenomenon for visual stimuli, perhaps restricted further to stimuli in the periphery of the visual field (e.g. the classic cases reported by Hoff and Pötzl, 1938, 1988). However, several Zeitraffer patients have noticed and reported that stimuli in modalities other than vision (such as speech and music) also seemed to be accelerated, sometimes to the point of incomprehensibility (e.g. Pötzl, 1951).

We describe here a patient who also experienced accelerated time. Unlike most Zeitraffer patients, who had right hemisphere parietal–occipital lobe damage, the present patient had damage in the left hemisphere prefrontal cortex, which resulted from a glioblastoma. To the best of our knowledge, the present study is the first report of an accelerated time experience resulting from a lesion in the prefrontal cortex, and it includes the first data on duration estimation by a Zeitraffer patient.

Case report

BW, a right-handed, 66-year-old retired clerk with no history of neurological disease, noticed while driving an automobile that external objects seemed to be rushing toward him at an incredible rate. Because of the apparent rushing of events he could not react quickly to them, and he had driven through a red traffic light. Regardless of whether he accelerated or decelerated, the velocity of the car seemed excessive. He stopped his car, unable to cope with the situation.

Upon his admission to the Heinrich-Heine-Universität Medical-Neurological-Radiological Hospital 2 days later, BW reported that time seemed to be passing very quickly. He described what he experienced as an 'accelerated motion' of events, like in a time-lapse film. He complained that he could no longer tolerate watching television, because the progression of events was too quick for him to follow. Because of the apparent high rate of events, he said that he tried to think more slowly. He also described the feeling that life had begun to pass too quickly for him.

BW's relatives reported that although he formerly was energetic, after the onset of his neurological symptoms he mainly sat apathetically in his armchair and slept more than usual. Consistent with this, BW reported that he became fatigued more easily than he used to be. His relatives also noticed that his movements seemed relatively slow and clumsy, that he seemed indifferent to others, and that he was reluctant to communicate. BW gave up hobbies like reading the newspaper and tending his garden. He also seemed to lose interest in his family life and social relationships, although formerly he had been a very sociable person. BW reported no problem with spatial orientation or long-term memory, and his relatives observed that he could find his way home just as before.

Methods and results

Computerized tomography (CT)

A CT scan was performed during the acute stage, within a few days after BW's admission to the hospital (see Fig. 1).

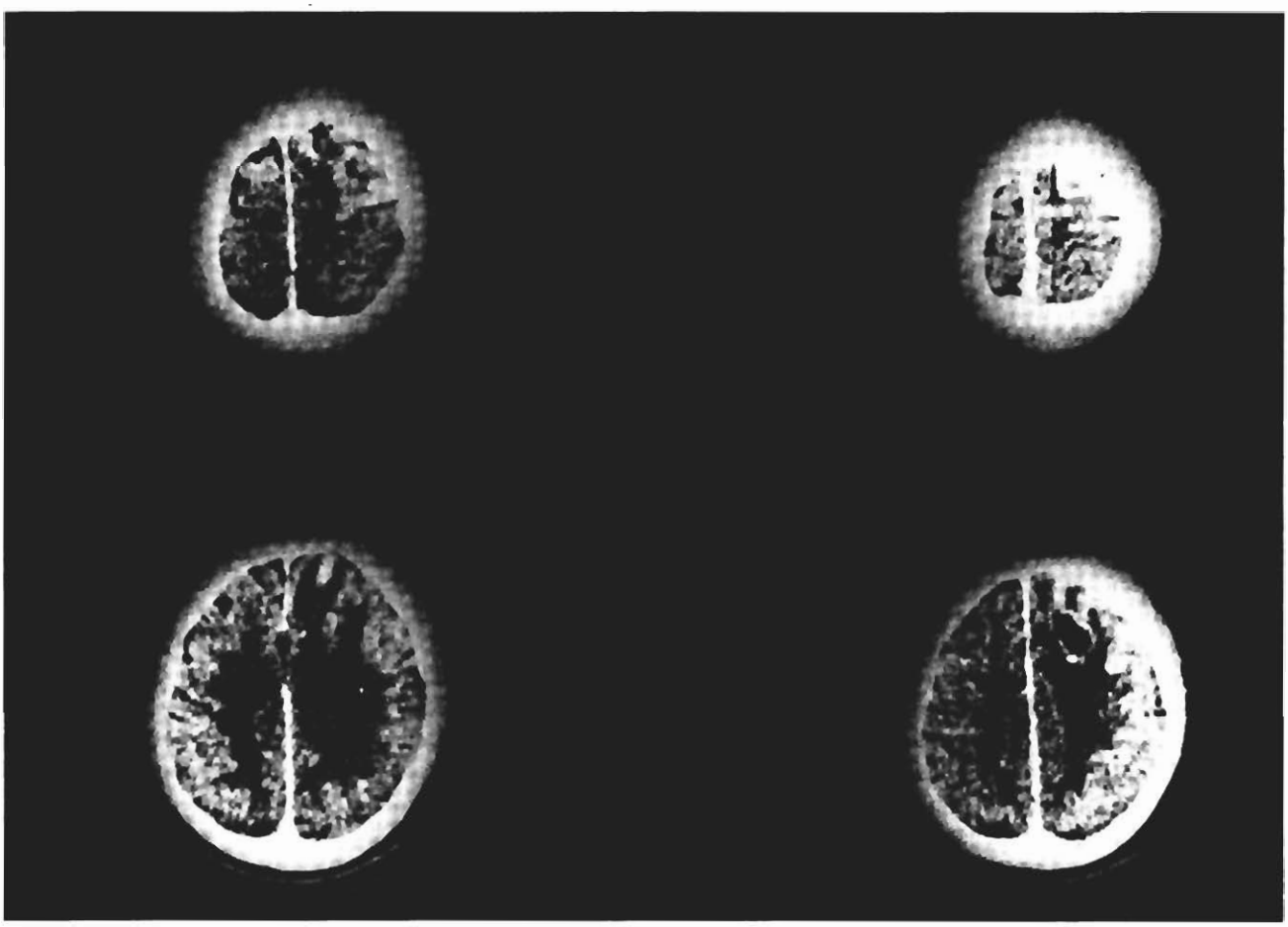

Fig. 1. A series of CT slices showing BW's left prefrontal cortical cystic lesion and surrounding edema. In all of them (according to standard radiological convention), the left hemisphere is at the right, and the right hemisphere is at the left. All scans were at 0° from the canthomeatal line. From right to left and top to bottom, the four scans are the 2nd, 3rd, 4th, and 5th sections from the vertex, as shown in Matsui and Hirano's (1978) atlas (see pp 140–7). The lesion is most clearly seen in the 4th section (lower right of figure).

It showed a left prefrontal cystic lesion, with ring-like contrast medium enhancement, surrounded by extensive perifocal edema. The focal lesion was near the posterior part of Brodmann's area 9, anterior part of area 8, and superior part of area 46, centered ~1 cm below the cortical surface. Although the edema affected most of the superior left-frontal lobe, it did not extend to the primary motor area. A biopsy revealed a high-grade glioblastoma in the left prefrontal cortex.

General neuropsychology

Shortly after admission to the hospital, various clinical observations and experimental tests were conducted. BW was alert and gave accurate information about himself, the current date, and the current place. His responses to environmental stimuli and his movements were appropriate, although slow, but he appeared somewhat apathetic. He complained of a frontal headache. In spite of the problems he was having, his overall demeanor was surprisingly positive.

BW's spontaneous speech displayed motor, or expressive, aphasia (i.e. speaking in a telegraphic manner, with hesitations, word omissions, and some substitutions). His performance on the Aachen Aphasia Test was pathological, showing evidence of motor aphasia. When asked to repeat sentences spoken by the examiner, BW's repetitions were slow, but accurate. He showed some anomia; for example, he sometimes could not name objects that he was using properly, such as a pencil.

BW displayed some agraphia characterized by micrographia, paragraphia, and a disturbed writing pattern (see Fig. 2A). His writing style and fluency were different than previously. However, he showed little or no dyscalculia (see Fig. 2B). On a subtraction task, he needed several attempts to write the correct result, and in the second step he made a mistake. He performed a multiplication task correctly. BW's drawings of a bicycle and a house (see Fig. 2C) were incomplete, especially on the right side. He was later able to draw a more complete house, but still with some difficulty. BW's drawing of a clockface also revealed right hemispatial neglect (see Fig. 2D): he placed the digits mainly on the left side. His attempts to improve the drawing were rather frustrating, with evidence of perseveration. BW also complained about micropsia and macropsia. In short, BW showed mild visuospatial, or constructional, apraxia with right hemispatial neglect (Heilman *et al.*, 1985). Several brain areas have been implicated in the neuropathology of neglect, including the dorsolateral frontal lobe (Heilman and Valenstein, 1972).

On the Trail Making Test (see Strub and Black, 1988), BW performed within the normal range. BW's working memory was tested with a standard digit span test, and the results showed normal immediate recall of verbal information. In addition, BW was asked to listen to three simple sentences and immediately repeat them. One of the

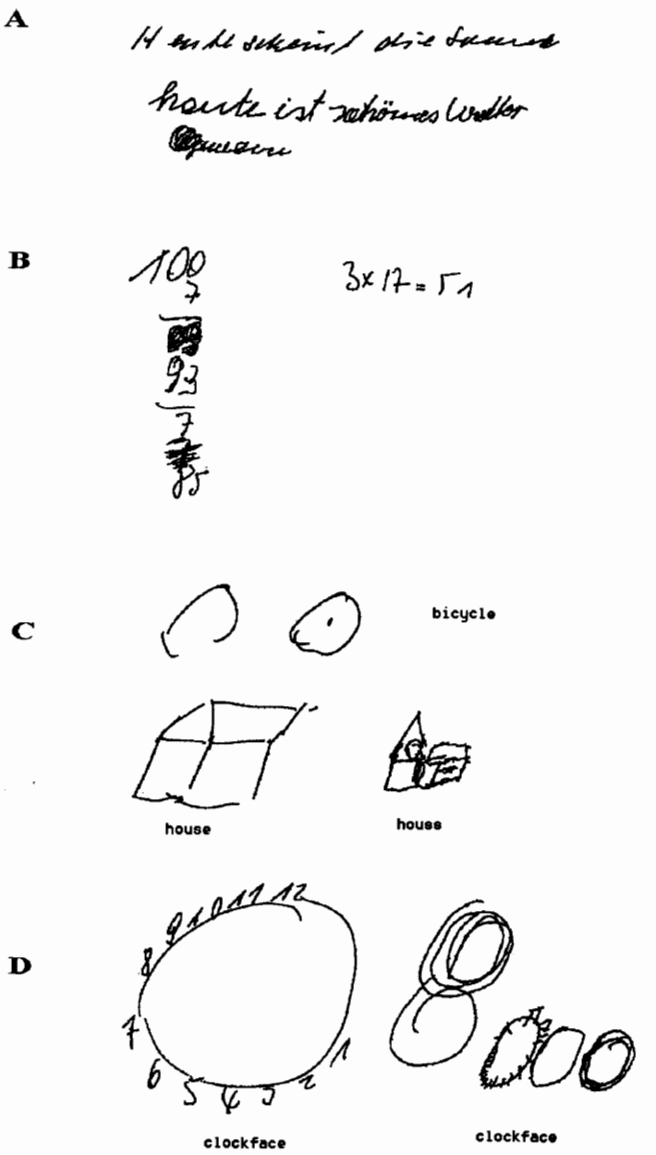

Fig. 2. (A) Examples of BW's handwriting (*Heute scheint die Sonne* = The sun is shining today; *heute ist schönes Wetter gewesen* = The weather is beautiful today). (B) Examples of BW's performance on arithmetic tasks. (C) BW's drawings of a bicycle and a house, illustrating constructional apraxia. (D) BW's drawings of a clockface, illustrating right hemispatial neglect.

sentences was meaningful, whereas the other two were not. His immediate recall of the sentences was very good. After recalling the sentences, BW also correctly indicated that two of the sentences had nonsensical content.

Motor examination

BW showed a normal reflex level, with no bilateral reflex differences, an absent Babinski sign, and a present palmomental reflex. He had normal force production in all tested muscles. However, BW showed latent hemiparesis of the right-sided extremities; this was not obvious on

examination of his muscle strength, but was revealed on some provocation tests. He also showed some diffuse, ill-defined impairment of the somatosensory functioning of the right side of his body. He showed diminished swinging of his right arm while walking. There was also some anxiety-bound walking imbalance. In addition, his right hand displayed bradykinesis (fairly slow tremors prominent in the distal upper extremities) and dysdiadochokinesis (sloppiness in the execution of rapid alternating movements), as well as slowing of fractionated finger movements. BW also showed some disturbance of alternating proximal arm movements (Freund and Hummelsheim, 1985).

BW's ability to reproduce rhythms demonstrated by the examiner was also tested. In this test, the examiner tapped a rhythm bimanually (for example, left, left, right, right, right), and BW was asked to reproduce the same rhythmic pattern, also bimanually. His reproductions showed marked deficits, mainly because he added too many strokes with both hands (see Fig. 3). This finding is consistent with prefrontal cortex damage (Halsband *et al.*, 1993). Most of BW's motor deficits resolved after treatment with corticosteroids. However, his neuropsychological deficits remained.

Transcranial Magnetic Stimulation

Transcranial Magnetic Stimulation revealed normal central motor latencies and magnetic evoked-potential amplitudes after stimulation of the motor cortex and registration of EMG responses in the Flexores digitorum I and Tibiales anteriores muscles. These findings suggest that there was no impairment of functioning in the pyramidal tract.

Electroencephalography

A standard electroencephalogram (EEG) was obtained, using the international 10–20 system of electrode placement over the entire cranium. All recording sites yielded normal EEG patterns, except for a focus in the left frontal-temporal region, which showed high-amplitude delta activity ($\sim$1–2 Hz). This focal slowing seen on the EEG is typically seen in cases of cortical tumors (Malloy and Nadeau, 1986). There was no accompanying epileptiform activity.

Temporal orientation

Although BW seemed oriented to person, time, and place, a temporal orientation test (Benton *et al.*, 1964) was administered in order to quantify any possible deficit. This asks the person to state the date, the day of week, and the present time of day. BW's only mistake on this test was that he stated a time of day that was $\sim$60 min earlier than the actual time. Thus, his score was 98 out of 100, where the mean for normal hospitalized patients was 99.4 (Benton *et al.*, 1964).

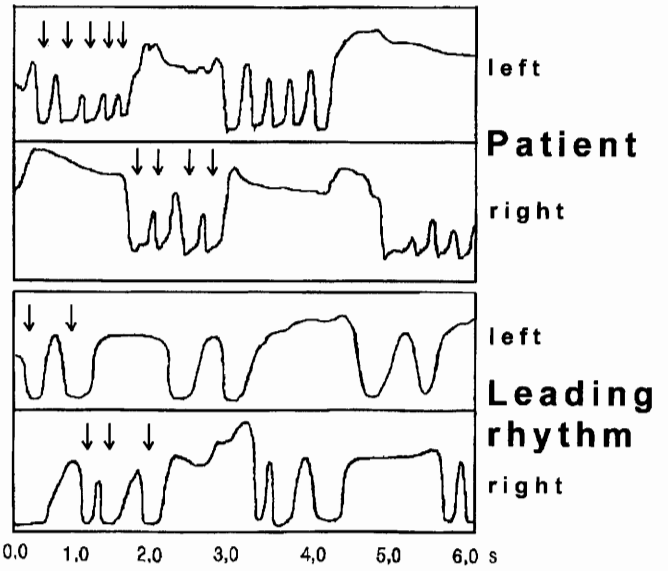

Fig. 3. An example of BW's performance on a rhythm reproduction task: A 6-s segment of a recording made by using an optoelectronic tracking system (Selspot II) with a light-emitting diode attached to each index finger of the patient (BW) and to each index finger of the experimenter ('leading rhythm'). The figure shows movements of the left and right index fingers of the patient (two upper traces) while attempting to reproduce a rhythm demonstrated by the experimenter (two lower traces). Two taps of the left index finger should have been followed by three taps of the right index finger, with this rhythm repeating about once every 2 s. BW produced too many taps with both fingers. The finger strokes (taps) are indicated by arrows for the first cycle only.

Duration estimation

A bedside test of subjective duration, using the method of production, was also administered (see Benton *et al.*, 1964). On each of 5 days, the experimenter asked BW to produce 60 s durations. It was explained that the experimenter would say *start*, and BW was asked to say *stop* when he thought that 60 s had elapsed since the *start* signal. Each of five trials per day was separated from the others by an interval of several minutes. All of BW's productions were much longer than 60 s; his overall mean production was 286.0 s (SE = 19.9 s). The overall mean error in his productions (the absolute value of the difference between the produced duration and the target duration) was therefore 226.0 s. Inspection of means (and standard errors) revealed no significant trend in his mean production across trials or across days. A duration judgment ratio was calculated by dividing the duration requested (60 s) by the duration produced (286.0 s). This ratio was 0.24 (SE = 0.02), and the 95% confidence interval associated with it was from 0.19 to 0.30.

BW's mean error of 226.0 s on the production of a 60 s duration contrasts markedly with Benton *et al.*'s (1964) hospitalized control subjects, who showed a mean error of 24.2 s, as well as with Benton *et al.*'s brain-damaged subjects, who showed errors ranging from 21 s to 48 s. A recent meta-analytic accumulation of results of nine

experiments on duration production by normal (non-brain-damaged) elderly subjects revealed that they gave an overall mean duration judgment ratio of 1.35, with a 95% confidence interval from 1.15 to 1.56 (Block and Zakay, 1994). BW's mean duration judgment ratio of 0.21 is well outside this confidence range. Stated in terms of actual produced duration, we can be 95% confident that an experiment in which normal elderly participants are asked to produce a 60 s duration will yield a mean production between 38.4 s and 52.3 s, with a mean of 45.4 s. BW made a mean production (286.0 s) far outside this 95% confidence range. Thus, BW's duration experience was $\sim$16% of that of normal elderly people, which is a duration-compression ratio of $\sim$6.3 : 1. This is approximately the rate of a modern video cassette recorder played on fast forward.

A related question concerns whether the lesion in BW's frontal cortex affected the variability of his duration productions. Intrasubject variability of duration judgment may be assessed by calculating the coefficient of variation (CV), which is the standard deviation divided by the mean duration estimate (see Block and Zakay, 1994). The CV of BW's duration productions was calculated separately for each of the 5 days (0.24, 0.20, 0.23, 0.24, and 0.28, respectively), then the five CVs were averaged. His overall mean CV was 0.24.

Although there are no normative data on intrasubject variability of duration production with which to compare his mean CV of 0.24, intrasubject CVs for duration judgments in the general range of 0.20 to 0.30 are not uncommon. Thus, although BW's mean production was abnormal, the variability of his productions was fairly normal.

Discussion

The person studied here, BW, had a glioblastoma, with surrounding edema, centered in Brodmann's areas 8, 9, and 46 of his left hemisphere superior prefrontal cortex. He also showed several classic symptoms of frontal lobe damage, such as some aphasia and anomia, along with a general slowness of his behavior (Malloy and Nadeau, 1986; Strub, 1989). In spite of BW's frontal impairments, in many ways he appeared relatively normal. He was as well oriented to date and place as is the typical normal person, except that his judgment of time of day was in error by about an hour. BW's pattern of little or no disturbance of temporal orientation (orientation to place and date) contrasted greatly with his severe distortion of the subjective rate of flow of time. BW's greatly altered duration experience was revealed in his duration judgment performance: When asked to produce a 60 s duration, his mean production was 286.0 s. The possibility that this may be a non-specific effect of brain damage is weakened by Benton *et al.*'s (1964) finding that 60 patients with various kinds of damage to the cerebral hemispheres gave productions far different from BW's.

This pattern of findings concerning BW's duration judgment and temporal orientation fits well with the notion that duration experience and temporal orientation are separate aspects of psychological time that reflect partially independent processes (Benton *et al.*, 1964; Block, 1996). Thus, these aspects appear to be dissociable – subserved by somewhat different brain areas and processes. BW's relatively normal temporal orientation was not related to the other temporal phenomena that he displayed, such as his subjective experience of a fast rate of external events and his greatly lengthened duration productions (i.e. greatly shortened duration experience).

Possible explanations

Along with findings of earlier case reports, the present findings may be interpreted in several, possibly interrelated ways. All of these are necessarily speculative. Nevertheless, the explanations offered here all relate to current hypotheses about the processing of time-related information in the brain.

Visual system pathways

Some previous patients who experienced an accelerated-time phenomenon mentioned it only for visually perceived objects. The lesion in these cases was usually near the parietal–occipital junction in the right hemisphere. One possible explanation is that, in these patients, the accelerated-motion phenomenon resulted from damage to part of an interconnected neural network that analyses visual motion information. Such an explanation, however, cannot be offered for the rather common report that the Zeitraffer experience extends also to stimuli in modalities other than vision, such as audition (e.g. speech and music), as in the case reported by Pötzl (1951). It is possible that other Zeitraffer patients also experienced it in other modalities, but either they or the researcher chose to mention the phenomenon only in its most striking modality, vision. In addition, there were no conspicuous moving stimuli during the bedside duration productions made by BW, which revealed a greatly shortened duration experience.

Synchronized firing in neural networks

Research has revealed synchronous oscillations (neural firing patterns) in various parts of the brain, which may play a role in the integration of specialized analysing mechanisms, such as for motion, form, and color (for reviews, see Pöppel and Schwender, 1993; Singer, 1993). These oscillatory responses are in the gamma range, between about 40 and 60 Hz. Synchronous oscillations have been found in the thalamus and in the frontal cortex. Perhaps BW's accelerated time experience occurred as a result of a slower rate of these oscillations, much like a time-lapse camera recording only one frame every few

seconds. Although this hypothesis does not encounter the same problems as the visual system explanation, perhaps that is attributable to its lack of specificity.

Attentional resources

Another possibility is that BW's lesion affected a cortical area involved in determining the amount of available attentional resources. One could assume a model in which decreased attentional resources do not allow an attentional gate to open as widely, thereby allowing fewer time-encoding pulses produced by a pacemaker to pass through to a counter (Block and Zakay, 1996). External events would not be associated with the usual large number of pulses, and therefore they would seem accelerated. Produced durations would be lengthened, because the production is terminated only when the accumulated pulse count nearly matches the average pulse count (for the target duration) stored in reference memory. Earlier findings that dorsomedial thalamic damage also may produce an accelerated-time phenomenon (Hoff and Pötzl, 1934, 1938) are consistent with this explanation, in that the thalamus is known to be involved in attention. Decreased attentional resources also provide a possible explanation for BW's slow, lethargic behavior. However, BW had no apparent difficulty sustaining a normal conversation, an observation that weakens this explanation.

Pacemaker mechanism

As noted earlier, scalar-timing theories usually propose that animal and human timing are subserved by an internal-clock mechanism. Certain drugs, for example, are thought to shorten duration productions because they accelerate the rate of the pacemaker, and others are thought to lengthen duration productions because they decelerate the rate of the pacemaker. In the present case, BW's productions of 60 s durations were greatly lengthened. One possible explanation, therefore, is that the pacemaker component of his internal clock was now producing pulses at a considerably decreased rate (see Nichelli, 1993). This explanation can handle the duration production data and possibly also the characteristics of the Zeitraffer experience.

Conclusion

Damage to part of BW's superior prefrontal cortex of the left hemisphere resulted in an accelerated-time (Zeitraffer) experience. To our knowledge, this case is novel in that it is the first report of this kind of experience resulting from a prefrontal cortex lesion. In addition, we obtained the first duration judgment evidence, which clearly shows that the Zeitraffer experience is accompanied by greatly lengthened duration productions (i.e. greatly shortened duration experience). It is unclear how best to explain the phenomenon. Of the explanations we have considered here, the hypothesis that the rate of an internal pacemaker was greatly slowed by the damage to BW's prefrontal cortex perhaps best accounts for all the available evidence.

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Accelerated time experience after left frontal cortex lesion

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Abstract

We report a patient, BW, with an accelerated time experience as a result of a glioblastoma in his left superior prefrontal cortex. He experienced external events as seeming to occur at a much faster rate than before the lesion, but he showed normal awareness of time, place, and identity. He displayed behavioral slowness, some aphasia, right hemispatial neglect, and other neuropsychological problems typical of left frontal damage. When asked to produce 60 s durations, BW's mean production was extremely long (286.0 s). This finding is consistent with the notion that subjective duration had decreased relative to objective duration. Several explanations for the findings are discussed, including the possibility that the rate of an internal pacemaker was greatly slowed.

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Primary diagnosis of interest

Accelerated-time experience caused by glioblastoma in left prefrontal cortex

Author's designation of case

BW

Key theoretical issue

- Accelerated-time experience in a patient with left prefrontal cortex damage is accompanied by greatly lengthened production of duration

Key words: time perception; accelerated-time (Zeitraffer) experience; prefrontal cortex

Scan, EEG and other related measures

CT, EEG

Standardized assessment

Benton *et al.*'s temporal orientation test, including duration production, Trail Making Test, digit span test, Aachen Aphasia Test

Other assessment

Cerebral biopsy, Transcranial Magnetic Stimulation, test of rhythm reproduction, drawing tests, motor examination

Lesion location

- CT: left hemisphere superior prefrontal cortex

Lesion type

Glioblastoma

Language

English