Topographical amnesia

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SUMMARY The ability to learn to criterion a visually-guided stylus maze was found impaired in patients with right posterior cerebral damage, not only in comparison with controls but also with other hemisphere-damaged groups. The contribution of the corresponding left sided area to this task is dubious, and certainly not substantial. This finding points to the independent organisation of long-term spatial memory in the right posterior cerebral cortex, an inference that was further supported by the study of two cases. The first was a female patient with right temporo-parietal softening (as suggested by clinical, EEG, and brain scan data) who showed topographical amnesia and inability to learn the visual maze over 275 trials. On an extensive battery of tests she was found free from disorders of space perception, and from verbal and visual memory impairment. The second was a patient presenting with severe global amnesia who, nevertheless, had no difficulty in route finding, and reached the criterion on the maze in 31 trials.

Defective route finding, in the absence of impaired consciousness or of global amnesia, is a symptom that deserves special consideration because it can provide hints on the way in which spatial functions are organised in the cerebral hemispheres. Attempts at elucidating its nature have, however, met with remarkable difficulty, because in most cases the concomitant presence of other spatial deficits makes it difficult to analyse the mechanism by which patients lose their bearings.

Brain (1941) listed four basic disorders that may render a patient unable to find his way about. Three of them are perceptual disorders that prevent the subject from adequately appreciating the spatial arrangement of the external world or from recognising its landmarks. These are: (1) loss of awareness of the absolute and relative position of objects, which, when not limited to one hemi-field, results in total failure to orientate towards a seen object; (2) inattention to the left half of external space following a right hemisphere lesion, with the consequent tendency to ignore left turns and to turn always to the right; and (3) visual object agnosia, which hampers recognition of surroundings. It must be said, however, that not every type of visual agnosia is accompanied by topographical disorientation, as shown by the perfectly retained topographical orientation reported in some patients with severe object agnosia of the associative

type (Hécaen and Ajuriaguerra, 1956; Rubens and Benson, 1971; Hécaen *et al.*, 1974). On the other hand, there is at least one case in the literature (Pallis, 1955) where the patient's inability to find his way about appeared to be dependent on the failure to single out the individual features of buildings and places. This patient showed also difficulty in recognising faces, and had dyschromatopsia, and may, therefore, be considered as an example of agnosia of the aperceptive type.

The fourth basic disorder pointed out by Brain (1941) as a possible source of defective route finding is loss of topographical memory. This deficit is described by Benton (1969) as a failure to retrieve former geographical knowledge and long-established visual memories concerning the spatial characteristics of familiar surroundings and routes. If buildings, squares, and streets, though well recognised individually, no longer represent topographical landmarks, it is understandable that the subject will be at a loss in finding his way about.

Defective topographical memory can thus be said to be present only when the patient loses his bearings in a well known environment and is not able to give a verbal or a graphic description of familiar routes or places. There are, however, other patients who lose their way only in new surroundings—for example, when they try to find their way through hospital wards (Scotti, 1968)—while they are well-orientated in an environment known before the onset of the disease, and show intact geographical knowledge. This raises the possibility that the deficit in these cases should not

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be regarded as a disorder of memory, and should be distinguished from that of patients with loss of long-established visuospatial memories. This is the opinion of Benton (1969) who distinguishes defective topographical memory from defective route finding, and interprets the latter as a 'failure to acquire an adequate appreciation of the basic spatial schema expressed in the route'. One wonders, however, whether both disabilities might not be more appropriately perceived as due to an amnesic disorder, one showing anterograde, and the other, retrograde amnesia. Provided that deficits of space perception and cognition can be positively excluded, the inability to form, store, and retrieve the spatial schema needed to learn a new route seems to be basically a failure in amnesic performance.

Because of the confusing array of spatial disorders which frequently accompany defective route finding, and because of the difficulty of examining topographical orientation in normal surroundings in patients with motor and language deficits, the analysis of single cases has been supplemented in recent years by quantitative testing of groups of patients with known hemispheric lesions. Among the large number of spatial tests available, three appear to be most directly related to the issue raised by topographical disorientation. The first is the locomotor maze test used originally by Semmes *et al.* (1955), and more recently by Hécaen *et al.* (1972) and by Ratcliff and Newcombe (1973). The patient is asked to walk along a specific path on the basis of information provided by maps, visually or tactually presented. This task has an important feature in common with the attempt to follow an itinerary in the real world—namely, it requires the subject to maintain his orientation despite the repeated shifts in body position that occur as he walks along the route. No demand is, however, made on the patient's memory, because all the information necessary to trace the path is provided. The testing situation is, therefore, more closely related to the task of a subject trying to orientate himself in an unfamiliar city by following a map, rather than to that of a subject recalling a route he has already travelled many times. It is noteworthy that some patients with route finding difficulty have been found able to perform the locomotor maze (Newcombe, 1974). When the test has been given to large groups of patients with localised hemisphere damage, a poor performance has been found to be associated with lesions in either parietal lobe (Semmes *et al.*, 1955), in either hemisphere, but especially the right (Hécaen *et al.*, 1972), and in both parietal lobes (Ratcliff and Newcombe, 1973).

A more direct approach to the relation of topographical memory to brain injury has been sought with two standardised procedures, one testing retro-

grade and the other anterograde amnesia. The first examined former geographical knowledge by means of verbal responses and pointing to a map (Benton *et al.*, 1974). When educational background was controlled for, brain-damaged patients performed less well than controls, but there was no significant difference between the two hemispheric groups. Patients with poor scores showed a shift in localisation of cities or states towards the side of the map corresponding to the side on the injured hemisphere. The test, therefore, appeared to be especially sensitive to mild hemi-inattention. Since in this study none of the patients was suffering from clinical signs of topographical amnesia, we do not know to what extent such a disorder would be reflected in the performance on this test.

Anterograde amnesia has been investigated with a stylus maze test, which requires the subject to discover and remember, through trial and error, the one correct path leading from one point to another of an array of boltheads or grooves. In absence of any other clue, errorless performance can be achieved only by memorising the spatial sequence of turns, a task found by Milner (1965) to be severely impaired in epileptics who had undergone ablation of the right hippocampal region. Patients with frontal (particularly right frontal) excisions also made many errors, but these seemed to be due mainly to failure to carry out the test instructions. In this study there were only a few patients with large right posterior cortical ablation and most of them had also undergone hippocampal excisions: their performance was remarkably poor, a finding subsequently confirmed in patients with longstanding missile injury (Newcombe and Russell, 1969; Ratcliff and Newcombe, 1973), and consistent with the clinical observation that topographical amnesia more frequently follows damage to the posterior portion of the right hemisphere (Hécaen, 1972). These data would suggest that the stylus maze is the test more suited to investigate the impairment of topographical memory in brain-damaged patients. It remains, however, to be demonstrated that patients unable to find their way in the real world fail in fact to learn the maze.

The purpose of the present research has been twofold. Firstly, on the basis of the results reported by the Montreal and the Oxford groups, it would appear that injury to the left retro-Rolandic area does not affect maze performance. Does this conclusion hold also for patients with more acute and less circumscribed damage, who frequently suffer from severe aphasia? Secondly, it was hoped, and actually happened, that during the period of this research a patient with clinical signs of topographical amnesia would be extensively studied. The investigation of this case and that of a patient with severe impairment in recalling

past and present events allowed us to provide evidence (a) that topographical amnesia is a specific amnesic disorder independent of impairment of spatial perception or of a general deficit in memory; (b) that it is faithfully reflected in maze performance; (c) that it is not necessarily present in cases with failure in long-term retention of most ongoing events.

Patients and methods

All the subjects were right handed patients admitted to the wards of the Neurological Department. Fifty of them suffered from disease not involving the brain and made up the control group. One hundred and fifty subjects showed definite neurological evidence indicating damage at the level of one cerebral hemisphere. In 51 patients the right hemisphere was injured and in 54 the left. The presence of visual field defect (VFD) was assessed in each brain-damaged patient by the confrontation method, supplemented, in any apparently negative case, by symmetrical double simultaneous stimulation in the upper and lower temporal quadrants. Thus the brain-damaged group was divided into four subgroups: left hemisphere-injured patients without VFD (LH-)=33; left hemisphere-injured patients with VFD (LH+)=21; right hemisphere-injured patients without VFD (RH-)=27; right hemisphere-injured patients with VFD (RH+)=24.

Aetiology was not represented differently across the brain-damaged groups. The most frequent diagnosis was vascular disease (70%), followed by brain tumour (21%). Age and years of schooling were controlled by means of the covariance analysis.

The Token test was given to all subjects. Twenty-three patients with left hemisphere disease were diagnosed as aphasic on the grounds of their performance on this test (De Renzi and Faglioni, 1975): 10 of them were LH- and 13 LH+.

TEST

The visual maze apparatus of the present research was identical to that used by Milner (1965); the correct path that the patient had to discover was, however, a little simpler, in that it had only 23 choice points instead of 28, and implied six turns instead of 11. It was also symmetrical with respect to the midline. Briefly, the patient had to discover the path, proceeding with a stylus from bolthead to bolthead, the only restriction being not to move diagonally and not to come back towards the bottom. If a wrong bolthead was tapped, this produced a loud click. Two separate electric error counters marked the wrong choices made in the left and right half of the maze. Practice was continued until the criterion of three successive errorless runs was attained. Training was

carried out for 25 trials; if the criterion was not reached, a second block of 25 trials was given the next day. A score of 50 was allowed if the patient failed to attain the criterion at the end of the second block. A practise test with four choice boltheads and two turns was given for demonstration at the beginning of the first session.

Results

Two statistics were used to evaluate the performance on the visual maze: trials to criterion and number of errors. A one-way covariance analysis (age and years of schooling being the covariates) was carried out to assess whether the performance of the five groups was different, and the *post hoc* Sheffè method was used to test pairwise comparisons between groups. Table 1 shows the mean number of trials to criterion of the five groups. The general analysis of covariance

Table 1 Mean trials to criterion corrected for age and years of schooling

Controls	LH-	LH+	RH-	RH+
13.41	15.98	21.31	20.47	31.76
<i>Fs of the multiple comparisons</i>				
	LH-	LH+	RH-	RH+
C	<1	2.18	2.12	13.06***
LH-		<1	<1	8.38***
LH+			<1	2.90*
RH-				3.90**

* = $P < 0.05$; ** = $P < 0.005$; *** = $P < 0.0001$.

yielded an $F: 14.27$ ($P < 0.001$ with 4148 d.f.). The outcome of the *post hoc* multiple comparisons is reported in the lower half of Table 1. The RH+ group was significantly inferior not only to the control group, but also to every other brain-damaged group. The difference between LH- patients and controls was minimal; that between LH+ and RH- patients and controls was more substantial, but failed to reach the 0.05 significance level with a two-tailed test. It may be argued, however, that a one-tailed test is more appropriate in this case, as there is no reason to expect an inferior performance on the part of controls. This would bring the difference between controls and LH+ and RH- to the 0.05 level. Eleven patients failed to reach criterion in 50 trials; eight of these belonged to the RH+ group, one to each of the other brain-damaged groups, and none to controls. This means that the inferiority of RH+ patients is likely to have been underestimated, as patients unable to reach the criterion received an arbitrary score of 50. The correlation between trials to criterion and Token test score were: -0.42 for controls; -0.40 for left brain-damaged patients; and -0.47 for right brain-damaged patients, all significant at the 0.01 level.

Table 2 indicates the mean number of errors made at the choice points over the learning trials. The first row reports total errors, while the second and the third rows give the errors made on the half of the maze homolateral and contralateral to the side of the lesion respectively. In control patients the left visual field was arbitrarily defined homolateral and the right field contralateral. Controls averaged 37.5 total errors. The means of the brain-damaged groups were higher, but, owing to the large variance, only RH+ patients were significantly impaired relative to controls ($F: 10.52, p < 0.001$). They also made significantly more errors with respect to the LH- and RH- group ($F: 4.35$ and $3.21, p < 0.01$ and 0.05 , respectively), but not with respect to the LH+ group. All other inter-group comparisons failed to reach the significance level, even when a one-tailed probability test was used to assess the difference.

Table 2 Mean errors made on the whole maze, on the ipsilateral, and on the contralateral half of the maze

	Controls	LH-	LH+	RH-	RH+
Whole maze	37.5	53.0	78.8	60.9	135.5
Ipsilateral maze	18.5	26.1	36.2	28.7	64.6
Contralateral maze	19.0	26.9	42.6	32.2	70.9

The same results obtained when the groups were compared with respect to errors made in the homolateral or in the contralateral field: again RH+ patients were significantly poorer than LH- and RH- patients, while no other comparison attained the significance level. Most importantly, no inter-group comparison was significant, when the difference between number of errors made on the contralateral and those made on the homolateral visual field was computed. This means that the inferior performance of RH+ patients cannot be attributed to an abnormal tendency to make more wrong choices in the contralateral (hemianopic) field.

In the course of this investigation a contrasting pattern of memory impairment was observed in two patients. One showed difficulty in route finding, but no disorder of general memory; the other suffered from global amnesia, but was perfectly able to find his way about. The results of their examination will be reported.

CASE REPORTS

MA, a 62 year old housewife, was apparently in good health until 15 January 1973, when she was found by her daughter lying unconscious on the floor. Admitted to a local hospital, she promptly regained consciousness, but presented with a left hemiparesis and was unable to utter a single word, although she could

write down her responses correctly and appeared to understand every question. Both the hemiparesis and the speech disorder resolved in eight days, leaving only a mild dysarthria. As soon as she got up and began walking, however, it was noticed that she mistook her bed and was unable to learn the way to the toilet. Discharged after 25 days with a diagnosis of cerebrovascular accident, she showed spatial disorientation in her own apartment, where she had been living for many years, and she lost her bearings whenever she went out alone.

She was readmitted to hospital on 10 May 1973 when neurological examination disclosed a minimal deficit of the left limbs, without asymmetry of deep reflexes and with the plantar responses in flexion. No deficit for pain, temperature, vibration, or two-point discrimination was apparent, but there were some errors in position sense in the fingers of the left hand and consistent tactile extinction over the left side of the body on double simultaneous stimulation. Cranial nerves showed no deficit. There was, however, a mild, but definite dysarthria, and the visual-field examination showed a left homonymous hemianopia with macular sparing. Routine laboratory tests were normal. The electroencephalogram showed right fronto-temporal slow waves in the delta range, and the brain scan revealed a moderate uptake of isotope in the right temporal region. Right carotid angiography and pneumoencephalography were unremarkable, except for a mild symmetric ventricular enlargement. The patient stayed in the hospital for one month and during this period was repeatedly interviewed: she was alert and cooperative, able to give details of her past history, and to memorise current events. She showed an appropriate concern about the future and about her relationship with her daughter. The range of her interests was rather narrow but had always been so. Occasionally she read magazines but for the most of the time remained quiet and reserved. During the early days of her hospitalisation she had trouble in finding where her bed and the toilet were, but this subsequently improved, and she managed to get her bearings in the ward. However, if carefully observed, it was apparent that she was not self-confident about the path and looked hesitantly to left and right in search of some familiar landmark. When taken for the first time to the testing room (which is in the corridor immediately outside the ward), on leaving the room at the end of the session she turned to the right instead of to the left and only realised that this was wrong when she found herself in the male ward. If she went downstairs to the ground floor, on her return to the first floor she was uncertain whether she had to turn to the right or to the left in order to come back to the female ward. These were all very simple routes; when shown more complex paths in the garden

outside the clinic, she always got lost. The general impression, at the end of her hospital stay, was that she could eventually learn to find her bearings, but only with great trouble and hesitation.

Neuropsychological examination

The patient was given the Wechsler Scale (Form 1) and achieved an IQ of 92, with a verbal quotient of 91 and a performance quotient of 93. On the Raven Coloured Matrices she scored 24 in 10 minutes (the mean score of 55 control patients, examined in this laboratory, is 24.2).

A comprehensive language examination failed to show any sign of oral or written language disorder. On formal tests of ideomotor, ideational, constructional, and oral apraxia the patient did as well as normal subjects examined in previous studies. On no occasion did she disclose any difficulty in recognising objects or realistic figures, and on a profile front-view face identification test (De Renzi *et al.*, 1968) she achieved a score of 8 out of 10 (normal mean: 7.80).

Space perception was assessed not only by asking the patient to copy drawings (which were well performed), but also by two more specific tests. The first (De Renzi *et al.*, 1971) required the patient to reproduce the orientation in space of a rod and yielded a mean error score (in degrees) of 8.9, which is greater than the normal mean (4.5), but no different from the mean of 9 obtained by a sample of right brain-damaged patients with VFD—that is, the group more similar to the patient from the standpoint of locus of lesion. The second task (De Renzi and Scotti, 1969) was a tactile shape discrimination test that involved reconstructing the shape of a block on the basis of changes of direction in space made by the right forefinger which was exploring it. The patient scored practically at the same level as normal controls (22 versus 24.9 out of a maximum score of 36), and much better than a right hemisphere group with VFD (15.3). Table 3 gives on the left the scores obtained by the patient on a series of memory tests, and on the right the means found in previous researches in a right

hemisphere group with VFD and in a control group. The first three tests are drawn from the Wechsler Memory Scale; test 4 involves learning a list of 10 words; in tests 5 and 6 the subject must learn to rearrange 10 meaningful and eight meaningless figures, respectively, in the order they have been shown to him (De Renzi *et al.*, 1977); test 7 is similar to Kimura's (1963) recurring nonsense figures test but uses faces instead of figures (De Renzi *et al.*, 1968). The last three tests have to do with spatial memory; test 8 uses the cube display devised by Corsi (Milner, 1971), and assesses spatial memory span, requiring the subject to point to the cubes previously tapped by the examiner; in test 9 the subject must learn over three consecutive trials the position occupied by six geometrical designs, laid down in two rows (De Renzi *et al.*, 1969); test 10 is the maze. Except for tests 4, 5, and 6, the higher the score, the better the performance. In none of these tests, except the maze, was the patient found to be impaired and in most of them she performed as well as normal subjects.

This behaviour contrasted sharply with that shown when she was asked to learn the visual maze. The patient was given 10 blocks of 25 trials over a period of eight days and never reached criterion. A final block of 25 trials was administered two months later, and the patient was again unable to reach criterion. The Figure shows the error curve plotted over 275 trials; each point of the curve represents 25 trials. Although there was an improvement through the blocks, the patient was still making approximately two errors per trial during the last 25 runs. As with normal subjects, the wrong choices progressively concentrated on the boltheads in the middle of the path, whereas, for instance, the last two turns had already been learned after the first 25 trials.

An opposite pattern of memory deficit was shown by a second patient (RA) who will be described more extensively in a subsequent paper. For the present purpose, it is sufficient to say that this 53 year old man, previously employed at a gasoline pump, presented to us with a history of severe amnesia for everyday events, dating back to an operation performed

Table 3 Mean scores of patients MA and RA, of RH+ and control patients on a series of memory tests

Test	MA	RA	RH+ patients	Controls
1. Digit forward	5	4	5.95	6.18
2. Paired-associate words (maximum score: 22.5)	11	2	—	12.5
3. Babcock story (two consecutive trials; maximum score for trial: 26)	14	0	—	11.12
	16	0	—	15.31
4. Ten words learning (trials to criterion)	10	> 40	—	8.35
5. Serial learning of realistic figures (trials to criterion)	7	21	16.2	9.9
6. Serial learning of random shapes (trials to criterion)	9	16	22.1	12.7
7. Recurring face recognition test (maximum score: 24)	18	11	7.3	—
8. Spatial memory span	4	4	4.20	5.92
9. Visual memory for position (maximum score: 18)	18	—	9.9	14
10. Visual maze (trials to criterion)	> 275	31	31.76	13.41

Figure Visual maze test—error curve plotted over 275 trials.

one year earlier for an aneurysm of the anterior communicating artery. He showed a remarkable forgetfulness for current events: for instance, he could not remember any circumstance of his disease nor the marriage of his son a few months after the operation, and was never able at the end of a testing session to recall any of the tests he had been given. His memory for remote events was also partially defective, as shown by his inability to give any detail on the death of Mussolini, on the way the Second World War had ended, or to recall the name of any past or present players of his favourite football team. The neurological examination was unremarkable and the Wechsler IQ was 96. The patient's verbal learning deficit was reflected in his extremely poor performance on verbal memory tests, as shown by the scores reported in Table 3. This failure contrasted with his behaviour on visual memory tests and on the visual maze, where the criterion was attained in 31 trials, a mildly poor performance in comparison with the control mean, but not when compared with that of RH+ patients. This finding was in agreement with his everyday topographical orientation which was adequate, as testified by his ability to walk alone in the large city (Bologna) where he had been living, without losing his bearings.

Discussion

The findings of the present research converge with

those of previous studies (Milner, 1965; Newcombe and Russell, 1969; Ratcliff and Newcombe, 1973) in showing that learning a visually-guided maze is a suitable task to bring out the specific role played by the right hemisphere in long-term spatial memory. On this test RH+ patients are severely impaired not only with respect to controls, but also in comparison with RH-, LH-, and LH+ patients. This is a different pattern of impairment from that observed on a spatial short-term memory task such as pointing to cubes previously tapped by the examiner, where patients with posterior damage to either hemisphere were equally impaired (De Renzi and Nichelli, 1975). One could, however, ask whether a minor responsibility in carrying out the maze test is also borne by another region of the brain, particularly the left posterior one. The data of Newcombe and Russell (1969) and of Ratcliff and Newcombe (1973) argue against this possibility. In our study LH+ as well as RH- patients required more trials to reach the criterion than controls, but the difference was significant only if a one-tailed probability test was used; moreover, if the error score was taken into consideration, only the RH+ group's inferiority was confirmed. On balance, the evidence pointing to the critical participation of other cerebral areas besides the right posterior one in carrying out this performance seems meagre and not safely distinguishable from an unspecific effect of brain damage.

Errors made by RH+ patients were not more

concentrated in the left than in the right half of the maze, as would have been expected if they were due to some sort of neglect or perceptual deficit in the visual field contralateral to the lesion. This is a relevant finding, because unilateral neglect, which is known to occur not rarely among patients with right sided lesions, could have hampered their ability to explore the left half of the maze, thus preventing a faithful retracing of the path. Also subtle space perceptual deficits have been advocated (Newcombe and Russell, 1969) as a possible source of the maze learning difficulty of patients with right parietal lesions on the grounds of their performance on Raven Matrices, block design, and cube counting. Although it is true that right parietal damage can result in space perceptual as well as space memory disorders, it is more difficult to accept the view that the elementary changes in direction (turning to left, up, and down) required by the maze task are actually taxing the patients' ability to detect orientation and, consequently, bear an important responsibility for their learning failure.

Further support for the position that the retro-Rolandic area of the right hemisphere plays a critical role in topographical memory is provided by the study of patient MA. On all the available evidence she suffered from a right parieto-temporal softening. Her space exploration and perception were found to be normal on a series of tests and her verbal, as well as visual memory compared well with that of the controls. Her failure in route finding cannot, therefore, be traced to the derangement of other basic functions, and represents a discrete memory deficit which is faithfully reflected in the persisting inability to learn the same path on the maze over 275 trials. This behaviour contrasted with her normal spatial span and, in conjunction with the findings of a previous study (De Renzi and Nichelli, 1975), where two patients with an exceedingly poor score on the cube-pointing test were found to perform well on the maze, extends to spatial memory the generality of the proposition already advanced for verbal memory, namely, that long-term and short-term mechanisms are independent. A countercheck that separate neural structures underlie verbal and spatial memory is provided by the amnesic patient who did not present route finding difficulty in the external world and passed the maze task fairly well. A similar dissociation has already been reported by Starr (1970) in a patient who had developed amnesia following herpes simplex encephalitis. This author preferred the distinction between verbal and motor memory which, in our opinion, is reductive with respect to the autonomy of spatial amnesia, hardly traceable to a defective storage or retrieval of tactile and kinesthetic cues. It is noteworthy that the opposite

pattern of a preserved verbal memory and a clinically evident deficit of topographical memory has never been described in patients with damage to the hippocampus or subcortical structures, albeit epileptics with right medial temporal-lobe ablation have been found impaired on the maze (Milner, 1965) as well as on other spatial memory tests (Milner, 1971). Topographical amnesia was present in some patients with bilateral hippocampal involvement but always in a setting of global amnesia. We too have recently observed a patient, suffering in all likelihood from bilateral softening of the hippocampus, who presented with a profound inability to learn new facts as well as new routes. Thus, the evidence currently available appears to indicate that extensive damage or removal of the medial parts of both temporal lobes may disrupt the acquisition of all types of memories—manual skills excluded (Corkin, 1968)—but that for an isolated, severe topographical amnesia to occur, injury to the right posterior neo-cortex is a necessary prerequisite.

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