4

Memory

- Memory can be FRACTIONATED into several different types.
- ICONIC MEMORY is a buffer store lasting about half a second, and SHORT-TERM STORE is a limited capacity memory lasting about 15 seconds and holding 6 to 8 items.
- Recall from LONG-TERM STORE not only depends on the DEPTH OF PROCESSING during ENCODING, but is also affected by INTERFERENCE during retrieval.
- AMNESIA as a result of head injury can either be due to failure of encoding or consolidation (as in ANTEROGRADE AMNESIA or the RETROGRADE AMNESIA in the minutes before injury), or due to a deficit of retrieval (as in the longer-term RETROGRADE AMNESIA before injury).
- The memory deficit in KORSAKOFF'S SYNDROME is best explained as a deficit of retrieval rather than encoding, because learning does occur, albeit without conscious awareness.

Without memory we could have no conscious sense of past (or future) and would be consigned to a continuously changing but incomprehensible present, a situation in which some patients with brain lesions seem to find themselves. Memory is not a single process and much research has concerned the FRACTIONATION of memory to reveal the functional components. Different types of information are stored and retrieved differently. Most studies consider the idiosyncratic knowledge called EPISODIC MEMORY, the episodes of personal experience recalled in a specific context (what you had for breakfast today, or about your schooldays); SEMANTIC MEMORY is a more general knowledge about the world (that Bombay is south-west of Delhi, that Incan terns are black, or that the plural of mouse is mice), is independent of the context in which it was acquired (when did you learn that mice is the plural of mouse?), and which is similar to the semantic system of language (and can be damaged specifically in the condition of ANOMIC APHASIA — see Chapter 23). Semantic and episodic memory are both DECLARATIVE MEMORIES, capable of conscious recall, and are distinct from PROCEDURAL MEMORY, the knowledge of motor skills and conditioning, which are automatic and unconscious.

Consider what episodic memory must do. Fleeting experiences must be grasped, and held until placed in a more durable store than

ephemeral electrical circuits (for memories can resist electroconvulsive shocks and general anaesthesia), and put where they can subsequently be found. Recall means finding the site of storage, extracting the memory, converting it to electrical events, and eventually outputting it. Failure at any stage means memory loss or AMNESIA for the event.

Many events are fleeting; indeed speech is so spread out in time that it has disappeared physically before the message is complete. To remember such events they must first be placed in a BUFFER STORE to await proper processing. This is best shown in the visual system. A very fast display is shown in a TACHISTOSCOPE. A subject looks at a screen with a central dot. The dot disappears, and a 3 × 3 array of letters appears for 50 milliseconds. The letters disappear and the subject has to name them; subjects are correct for four or five of the nine letters. Did the subject never see the remaining letters, or have they already been forgotten in the time it took to recall the other letters? Using a PROBE TECHNIQUE, we find that the letters were actually seen and then forgotten. After the letters are shown, a blank screen appears for t milliseconds, and then a high, medium or low pitched tone sound (the PROBE) indicates whether the top, middle or bottom row of letters should be reported. Subjects are 100% correct if the probe is presented immediately the stimuli have disappeared from the screen (Fig. 4.1). All the letters must therefore already be in memory, because the subject does not know which row is to be recalled until the stimuli have disappeared. The amount of forgetting depends upon t. The experiment shows the existence of ICONIC MEMORY, in which forgetting occurs by spontaneous decay, analogous to radioactive decay, with nothing able to prevent it. The half-life of iconic memory is about 450 milliseconds. Information still present after then survives because it has passed into the next stage. ECHOIC MEMORY is the auditory equivalent of iconic memory and has a similar half-life. Iconic memory is a mere copy of the sensory input but lasts longer than the input. However, sensory inputs need to be processed, or perceived, and information extracted. This requires a longer lasting buffer, a SHORT-TERM STORE or WORKING MEMORY, in which they are manipulated, processed, and kept while required.

Short-term store is easily demonstrated experimentally. A subject hears 15 one-syllable words read aloud, one per second, and then immediately recalls the words in any order. Words are recalled differently at different positions in the list (first, second, ... last), producing a SERIAL POSITION CURVE (Fig. 4.2), which has three portions: a PRIMACY EFFECT, words at the beginning being remembered well, a RECENCY EFFECT, words at the end being remembered well, and a flat portion in the middle, without a specific name, where words are remembered less well. If the experiment is repeated with the minor variation that there is a 30 second delay before recall starts in which the subject has to do a DISTRACTER TASK (e.g. counting aloud, backwards

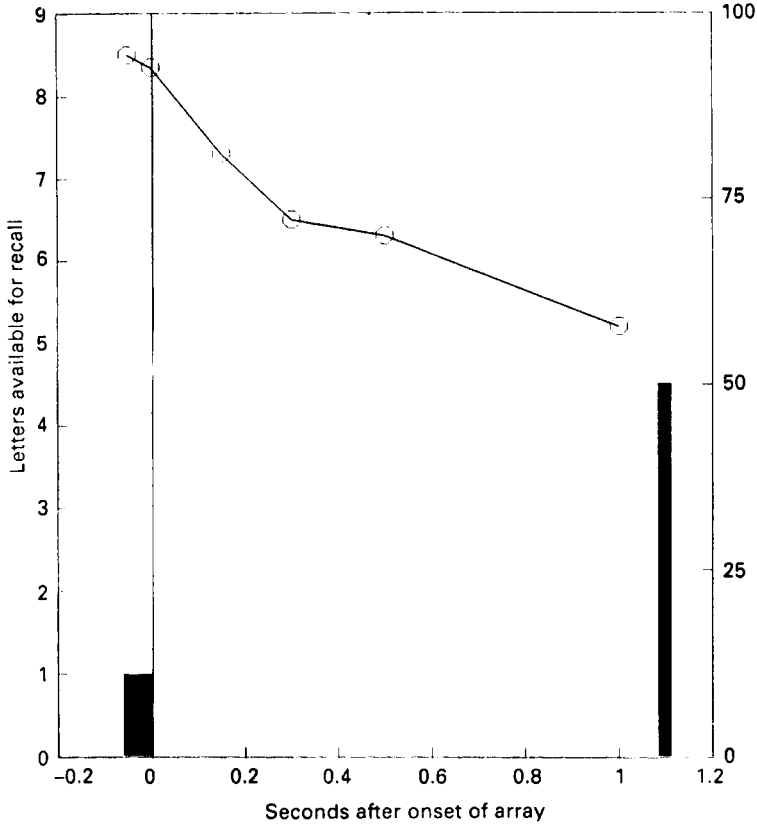

Fig. 4.1 Decay of iconic memory. A 3 × 3 array of letters was displayed for 50 milliseconds (shown by the small bar at the left). The dots show the numbers of letters available for recall when a probe was presented 0, 150, 300, 500 and 1000 milliseconds after the onset of the array. The bar at the right shows the number of letters recalled when the subjects are asked to recall the entire array. Adapted from Sperling G (1960), The information available in brief visual presentations, *Psychological Monographs*, **74**, (11).

n threes from a hundred), then the serial position curve shows no ·ecency effect (Fig. 4.2). In the first experiment, the subject has been ·emembering some words by repeating them (REHEARSAL) in the same ·ay that one remembers a telephone number for a minute or so. Rehearsal is disrupted in the second experiment and memory is mpaired for the last few words. The recency effect is due to the short-·erm store, which is of LIMITED CAPACITY, and is disrupted by overwriting with other information (as in distraction). The information does not decay spontaneously but is disrupted by new information. It is like a juggler who can keep four balls in the air; if the action is interfered with, by using the hands for something else, then the balls are dropped and lost. Short-term store is important for processing language because

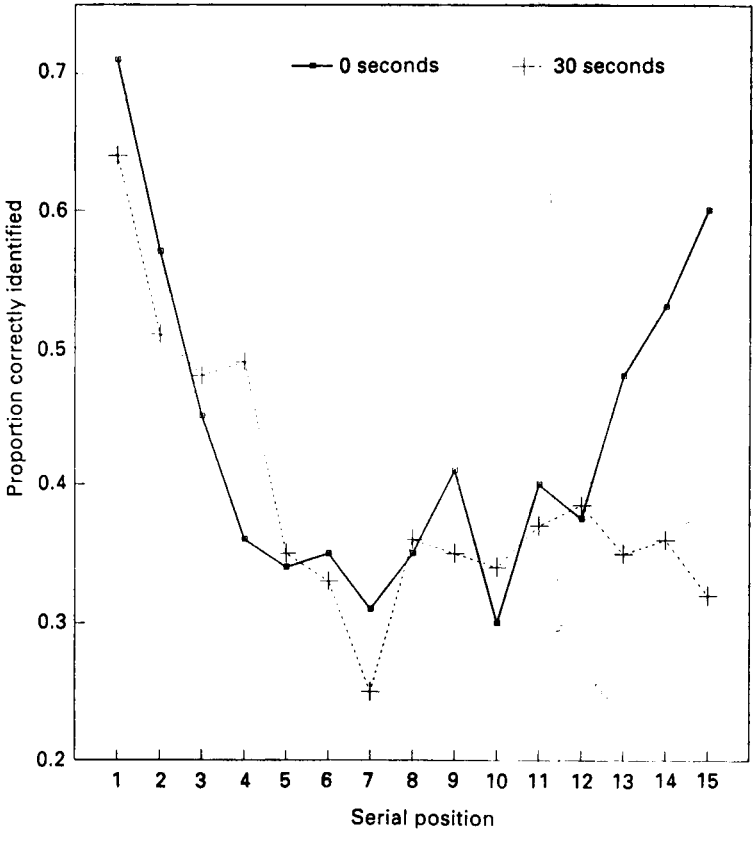

Fig. 4.2 The probability of a word being correctly recalled in a serial position experiment, according to its original position in the list. After reading out loud each of the 15 words, subjects carried out 0 or 30 seconds of a distracter task before recalling the words. Adapted from Glanzer M and Cunitz A R (1966). Two storage mechanisms in free recall. *Journal of Verbal Learning and Verbal Behaviour*, **5**, 351–60.

oken words extend over time, and grammar requires words to be lated to words spoken after a substantial delay (e.g. in '*the boy*, hose sister had long plaits and liked chasing kittens, *played* football', oy' must be held in working memory until 'played' appears).

If the recency effect reflects the short-term store, then the other arts of Figure 4.2 must be due to other, more durable memory, nown as SECONDARY MEMORY, or LONG-TERM STORE whose properties can e explored by other variations of the serial position experiment. If vords are read once every two seconds rather than once a second hen memory is improved (Fig. 4.3), and if more items are read then erformance on each item is worsened (Fig. 4.3). Greater resources mprove recall, implying words are actively put in memory. This is onfirmed if subjects are not told an experiment is on memory, but

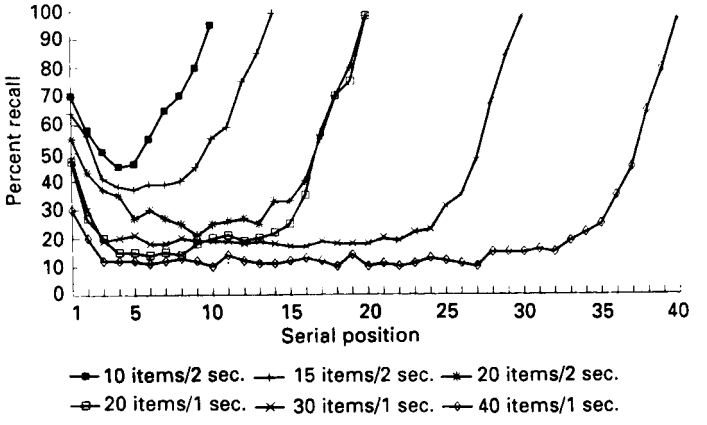

Fig. 4.3 Recall in a serial position experiment where lists are of different length, and items are presented either for one or two seconds. Words were read aloud and recall commenced immediately the list had finished. Adapted from Murdock B B (1962), The serial effect of free recall, *J Exp Psychol*, **64**, 482–8.

stead just to listen to words, and at the end are suddenly told to call them; INCIDENTAL LEARNING is less good than INTENTIONAL LEARNING, owing that information is active'y *put* into memory. Other experients confirm that recall from long-term memory is principally pendent on the DEPTH OF PROCESSING at the time of learning.

The primacy effect is particularly interesting. Consider a subject ho comes to the memory laboratory and carries out an experiment the first time, obtaining a typical serial position curve like that in gure 4.2. The experiment is then immediately repeated with different ords and a similar curve obtained except that the primacy effect is minished. And a third repetition gives a further diminution of the imacy effect. Why should this be? Think about the subject hearing e first word of the first experiment. They have a whole second to peat the word to themself and they learn it well. The second word pears and now two words need remembering, so each is worked at ss. This continues until fairly soon only the most recent three or ur words can be rehearsed. Because first words are processed more ey are remembered better. But that cannot explain why the primacy fect diminishes on each repetition (so-called PROACTIVE INTERFERENCE, which previous learning disrupts future learning). The problem is ot in LAYING DOWN or ENCODING the memory but in its RETRIEVAL. The rst word of the first experiment is highly distinctive and is easily etrieved from memory. The first word of the second experiment is ess distinctive, because already 15 other, potentially confusable, words are already in memory; and so on for the third list. Information nust not only be actively put into memory but also must be retrieved, nd INTERFERENCE between words prevents retrieval.

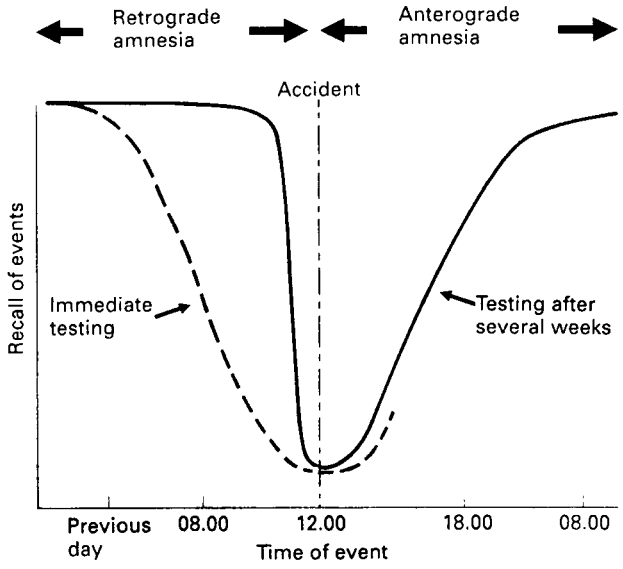

Fig. 4.4 A diagrammatic summary of memory loss, as shown by failure to recall events, in relation to the time of the event before or after an accident, for patients tested either immediately after or several weeks after an accident which occurred at midday. See text for further details.

Consider another experiment. Control subjects hear list A of ten adjectives and when asked to recall are correct on 4.5 items. Other subjects hear list A, but before recalling it, listen to another list B of ten items, and only then are asked to recall list A. Success on list A depends upon how similar B is to A. If B is 3-digit numbers, then on average 3.7 items of A are remembered, and if B is nonsense syllables hen 2.6 items are recalled from A. Unrelated adjectives in B reduce ecall of A to 2.2 items, antonyms of the items in A reduce recall to .8 items, and if B consists of synonyms of A then only 1.3 items are ecalled. Since B is heard *after* A is learnt, any effects of B must be pon retrieval rather than encoding (RETROACTIVE INTERFERENCE, in which revious learning is disrupted by subsequent learning). Successful iemory therefore not only requires encoding, but also retrieval; herwise it is like a card index in which the cards are in random der and although everything is there, nothing can be found.

Memory defects are common and illuminate underlying processes. TRAUMATIC AMNESIA occurring after head injury is common, particularly unconsciousness is prolonged. A motorist may have an accident at iidday, just before lunch, be concussed for a few minutes, regain onsciousness, and be seen half an hour later in a hospital accident and mergency department. Nothing about the accident and subsequent vents is remembered. Next morning there may still be no recall of n ambulance or of the accident, but there are vague memories of mergency treatment and clear memories of a hospital dinner the revious night (Fig. 4.4). ANTEROGRADE OR POST-TRAUMATIC AMNESIA (PTA) s for events *after* the accident and is presumably due to concussion disrupting cerebral functioning. Of greater interest is that even next

ıorning the patient may not remember events before the accident, ıch as getting up, having breakfast, and setting out on the journey. ETROGRADE AMNESIA (RA) extends backwards *before* the accident. Since ıemories of those events must have been encoded (normally people *ın* remember the previous morning), RA must be a DEFICIT OF RETRIEVAL. hat can be confirmed a month or two after the accident when the ıorning of the accident is remembered, showing that information ad been present but not retrievable during the period of RA. The nly exception is for the few minutes before the accident itself, ıemories of which never return, suggesting a failure of CONSOLIDATION, vhich is putting information into a durable form that can withstand oncussion. That is confirmed in studies of patients receiving electro-onvulsive shocks (ECS) to treat depression, to whom a word is said 0, 30 or 60 seconds before the shock; on regaining consciousness hey recall words given 60 seconds before the shock but not words ;iven 10 seconds before the sł ock, suggesting that consolidation takes ıbout half a minute.

A rarer problem occurs in KORSAKOFF'S SYNDROME, the eventual ɔutcome of WERNICKE'S ENCEPHALOPATHY, which occurs mainly in chronic ılcoholism and is due to deficiency of vitamin B_6, thiamine. The ɔredominant symptoms are a profound amnesia and CONFABULATION, a 'abrication of plausible stories to conceal the memory deficit. The ɔatient shows a complete inability to create new memories with ıothing being recalled since the condition developed; a doctor or nurse is not recognized despite daily visiting for years. There is good recall of events from earlier in life, before the condition, and as a result they often believe that a different prime minister is in office, or even that a previous monarch is on the throne. If patients with Korsakoff's syndrome are tested on a serial position task then their results at first glance suggest that only the short-term store is present, showing just a recency effect (Fig. 4.5), and middle part completely missing, which has been interpreted as a pure deficiency of encoding in secondary memory. However Figure 4.5 shows some hints of a primacy effect in the amnesics and this suggests a different explanation involving recall deficits. If *some* items can be recalled from long-term store then there must be encoding and therefore other information might also have been stored but cannot be retrieved. That is supported by another experiment. Memory was tested indirectly by asking patients to name partial or fragmented stimuli (words or pictures; Fig. 4.6), the same objects or words being repeated with increasingly more intact forms until correct recognition occurred. To the subject this is not a memory experiment at all; however if the same words or objects are presented repeatedly then SAVINGS, needing successively less of a stimulus to make a correct response, demonstrate storage *and* retrieval of information. Korsakoff's patients never realize that they have seen the stimuli before but nonetheless they do show savings, and therefore

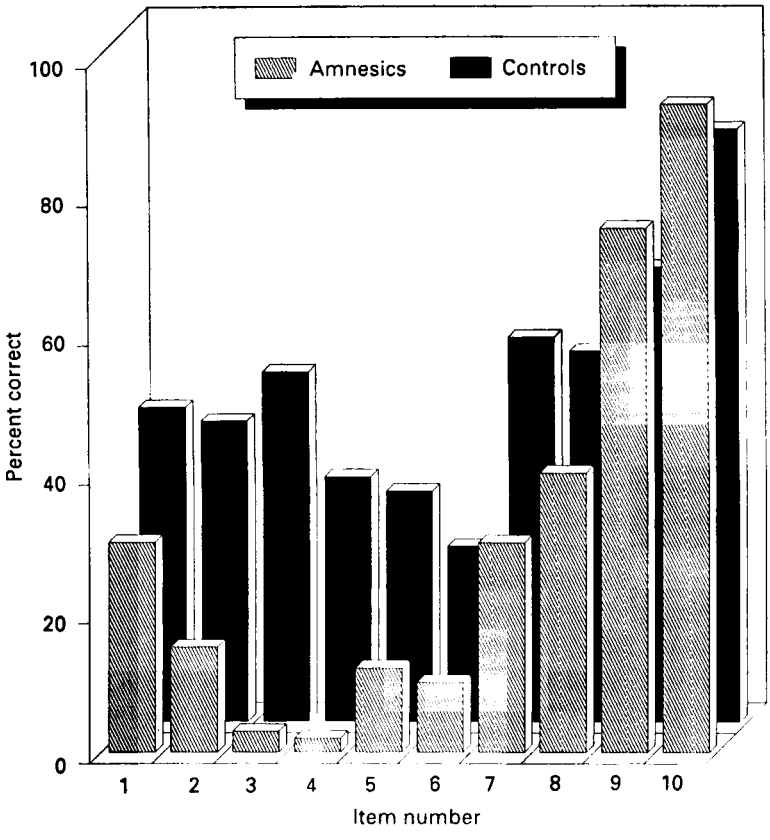

Fig. 4.5 Recall in a serial position experiment by amnesic patients with Korsakoff's syndrome, and by controls. Subjects were asked to remember a list of ten nouns which they read aloud from a card, one word being presented every three seconds. Adapted from Baddeley A D and Warrington E K (1970), Amnesia and the distinction between long- and short-term memory, *Journal of Verbal Learning and Verbal Behaviour*, **9**, 176–89.

must have adequate encoding and their deficit is one of retrieval, a deficit that is circumvented by providing 'hints' in the form of fragments. Such recognition without awareness in amnesia had first been described in CLAPAREDE'S PARADOX; the neurologist pricked the patient with a pin, left the room, returned a few minutes later and when the pin was produced the patient showed fear and moved away, but was unable to produce any conscious reason for the action.

An extremely rare form of amnesia was first described in a patient, now much investigated, called HM, who had severe, intractable epilepsy, particularly involving the temporal lobes. Since unilateral temporal lobectomy was known to benefit such patients, in 1953 it was decided to remove *both* temporal lobes. Postoperatively, HM had a profound anterograde amnesia, which still continues, along with

Fig. 4.6 Fragmented stimuli used to demonstrate savings in amnesic patients. Subjects are presented with progressively less fragmented versions of the stimulus until they can correctly recognize it. Reproduced with permission of authors and publishers from Warrington E K and Weiskrantz L (1968), New method of testing long-term retention with special reference to amnesic patients, *Nature*, **217**, 972–4; copyright 1968, MacMillan Magazines Ltd.

some retrograde amnesia, but no deficiency of short-term memory or of procedural memories (so that he can learn a complex motor task such as mirror-drawing, improving each day but never being aware of seeing the task before). Patients such as HM with the AMNESIC SYNDROME, which also occurs with encephalitis, cerebral anoxia and tumour, appear similar to Korsakoff's syndrome in their symptoms, although they do not confabulate. However careful testing suggests the two syndromes are different. When stimuli are presented very many times, both Korsakoff's and temporal lobe amnesics can remember at the same level as controls. Testing a day or a week later shows that Korsakoff's and controls have similar rates of forgetting,

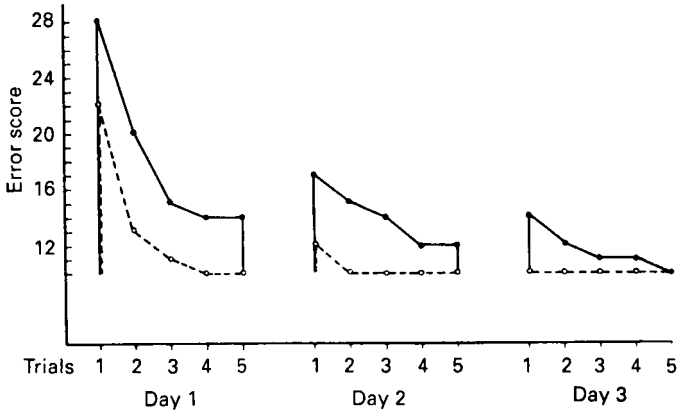

Fig. 4.7 Experiment using the fragmented stimuli of Figure 4.6, which shows that amnesics (solid points) are capable of learning to recognize the fragmented stimuli in Figure 4.6, albeit somewhat more slowly than control subjects (open points). Reproduced with permission of authors and publishers from Warrington E K and Weiskrantz L (1968), New method of testing long-term retention with special reference to amnesic patients. *Nature*, **217**, 972–4; copyright 1968, MacMillan Magazines Ltd.

but that temporal lobe amnesics forget much more quickly, which suggests different functional deficits.

This summary of memory deficits should not ignore the commonest cause, ALZHEIMER'S DEMENTIA; diffuse cerebral damage particularly affects the hippocampus and temporal lobes, and severe anterograde and retrograde amnesia occur. The neurochemistry of Alzheimer's shows a depletion of cholinergic neurones, which may be responsible for memory loss. Cholinergic blockade can produce an amnesia, first recognized by anaesthetists using hyoscine as a 'pre-med' with patients often not even remembering being taken to theatre. The effect has been confirmed experimentally. Attempts to improve memory in demented patients by improving cholinergic transmission, either with choline precursors or with cholinesterase inhibitors, have not as yet been successful.

The neuro-anatomy of amnesia is confused. Korsakoff's syndrome produces lesions of the mamillary bodies and the anterior thalamus, whereas the critical lesion in the amnesic syndrome involves the hippocampus. Since both are parts of the limbic system, it is suggested that the circuit from hippocampus to mamillary bodies is involved in retrieval. However, lesions of the fornix, which connects these areas, do not always produce amnesia. More confusingly still, in animals, lesions of hippocampus or mamillary bodies do not produce amnesia, although recent work finds that damage to hippocampus *and* amygdala results in amnesia in animals.

This account of memory deficits ends with an extremely rare syndrome which at first sight seems not to be a problem (and indeed seems advantageous), the HYPERMNESIC SYNDROME, in which patients are unable to forget. The Russian neuropsychologist A R Luria (1902–1977) described a patient, S, who automatically and uncontrollably used MNEMONICS (or memory strategies) to remember. In 1934, S was shown the following random formula:

$$N.\sqrt{d^2 \times \frac{85}{vx}}.\sqrt[3]{\frac{276^2.86x}{n^2v.\pi 264}}n^2b = sv\frac{1624}{32^2}.r^2s.$$

He spent six or seven minutes remembering it, composing a short story to help, the beginning of which was:

> Neiman (N) came out and jabbed at the ground with his cane (.). He looked up at a tall tree which resembled the square-root sign ($\sqrt{}$) and thought to himself: 'No wonder the tree has withered and begun to expose its roots. After all, it was here when I built these two houses' (d^2) ['dom' is the Russian for house]...

This ran for a further hundred and fifty lines. Fifteen years later, in 1949, and completely unprompted, S recalled both the story and the formula perfectly. Such mnemonics can be helpful in normal memory since they organize and structure information (and many medical students have remembered the cranial nerves by recalling that '*O*n *O*ld *O*lympus' *T*owering *T*ops *A* *F*inn *A*nd *G*erman *P*icked *S*ome *H*ops' in which the initial letters correspond to the nerves, with the P standing for 'pneumogastric', the old name for the vagus nerve). Sadly, S derived little pleasure from his ability, which was automatic, and he found it to be a liability. The implication seems to be, as Freud had suggested, that it is necessary to forget in order to live happily.

This chapter has discussed the functional organization of memory but has hardly considered its structural organization. Where and how are memories stored? It is probably fair to say that at present we have little idea, but that a Nobel prize could await the person who unravels the mechanism. Several biochemical mechanisms have been suggested of which one of particular interest is LONG-TERM POTENTIATION. A brief, tetanic electrical stimulus (several seconds at 100 Hz) to the hippocampus results in an enhanced synaptic sensitivity, which can appear within ten minutes of stimulation, the mechanism being due to high intra-cellular calcium levels that irreversibly induce a protease whose action induces glutamate receptors and increases numbers of synaptic receptors. Blocking of glutamate transmission by 2-APV (2-amino-5-phophonovalerate) has since been shown to inhibit the learning of new material in rats. The cerebral localization of memory has been studied in 'model systems', and for low level tasks such as eye-lid conditioning, seems in part to be mediated by cerebellar

mechanisms; a lesion to the middle cerebellar peduncle both abolishes and prevents re-acquisition of a conditioned eye-lid response on the contralateral side. The localization of higher memories, such as of visual discriminations, is far more diffuse, and studies of cerebral metabolism using isotopically-labelled 2-deoxyglucose during learning suggest that increased activity occurs throughout much of the cerebral cortex, limbic system, thalamus and cerebellum, although cingulate gyrus and hippocampus do seem to be especially involved. Such studies reinforce the suggestion that a specific memory for a specific event is not stored at a single cerebral location (as it might be in a computer), but rather is distributed throughout much of the brain, so that the entire integrated ensemble holds the memory (and hence memories in general are relatively resistant to localized cerebral damage). Recent analysis of such PARALLEL DISTRIBUTED PROCESSING (PDP) shows that it has many of the properties of memory, with an ability to learn complex events, based on only a few training trials, and without prior theoretical analysis, and that the memories so formed are like human memory in age and undergo 'graceful degradation', rather than sudden catastrophic failure.