Short term memory bowing effect is consistent with presentation rate dependent decay

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Abstract I reanalyze the free recall data of Murdock, *J Exp Psychol* 64(5):482–488 (1962) and Murdock and Okada, *J Verbal Learn and Verbal Behav* 86:263–267 (1970) which show the famous bowing effect in which initial and recent items are recalled better than intermediate items (primacy and recency effects). Recent item recall probabilities follow a logarithmic decay with time of recall consistent with the tagging/retagging theory. The slope of the decay increases with increasing presentation rate. The initial items, with an effectively low presentation rate, decay with the slowest logarithmic slope, explaining the primacy effect. The finding that presentation rate limits the duration of short term memory suggests a basis for memory loss in busy adults, for the importance of slow music practice, for long term memory deficiencies for people with attention deficits who may be artificially increasing the presentation rates of their surroundings. A well-defined, quantitative measure of the primacy effect is introduced.

Keywords Short term memory · Presentation rate · Free recall · Synapse · Endocytosis · Bowing effect · Primacy effect · Priming

Introduction

There exists a controversy over what causes loss of short term memory—decay, interference or distinctiveness (Oberauer and Lewandowsky 2008). A recent challenge to

existing theories is the linear relationship between response time and probability from 6 to 600 s in the word recall and recognition experiments of Rubin et al. (1999) and Anderson (1981) as found in Tarnow (2008). This linearity was accounted for using the tagging/retagging theory (Tarnow 2008, 2009), a phenomenological activation/decay theory.

In this paper I consider the challenge of the bowing effect in the classic Murdock free recall experiments (Murdock 1962) and Murdock and Okada (1970) in which intermediate memory items are recalled less well than initial (“primacy effect”) or final items (“recency effect”). Any theory of short term memory has to be able to explain the bowing effect but temporal distinctiveness fails (Brown et al. 2008). As presented so far, the tagging/retagging theory explains the “recency” effect, i.e. why the final items are recalled better than intermediate items, as a decay in time. I will organize the experimental data on the bowing effect to show that the data can be consolidated and that the tagging decay rate depends on the information presentation rate from which the primacy effect will follow.

The phenomenological tagging/retagging theory

The tagging/retagging theory (Tarnow 2008, 2009) describes the experimental data on memory decay from the experiments of Rubin et al. (1999) and Anderson (1981) which show a linear relationship between response time and response probability (Tarnow 2008). The tagging/retagging theory proposes that a short term memory is a tagged long term memory. When a word is read from a display, or remembered, the tagging level increases linearly in time to 100% according to equation 1:

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$$\text{Tagging level increase} = \text{stimulus time} / (\text{Tagging Time}) \quad (1)$$

where $0 < \text{stimulus time} < \text{Tagging Time}$ and the stimulus is either an external item or an internal item amplification. After the word is no longer displayed the tagging level decreases according to equation 2:

$$\text{Tagging level decay} = b - c \times \ln(\text{time after stimulus}) \quad (2)$$

The second equation, being the simplest fit of the decay data of Rubin et al. (1999) forms a straight line on a linlog plot where the time scale is logarithmic and the scale of the tagging level is linear. The tagging level is the probability that a word will be recalled during cued recall and the deviation of the tagging level from 100% is proportional to the subjects' response time delay because the word needs to be fully retagged. The proportionality constant, the Tagging Time, is related to the meaningfulness of the word—the more meaningful, the longer it takes to tag or retag the word. Words which lack meaning can take 0.3 s to tag and meaningful words can take 1.8 s to tag (Tarnow 2008).

In Tarnow (2009) a biochemical mechanism for the tagging/retagging model was suggested. It was proposed that reading a word causes prolonged neuron firing of action potentials which depletes the Readily Releasable Pool (RRP) of neurotransmitter vesicles at presynaptic terminals, exocytosis (see Fig. 1). Once the word is no longer read, the neuron firing stops. The pattern of depleted presynaptic terminals represents the long term memory trace of the word and the depletion itself (the tagging) of this trace is the short term memory. After the end of the neuron firing, synaptic endocytosis of the neurotransmitter causes the word to decay from short term memory. If the endocytosis is allowed to finish (the word is not read again and is not being actively remembered), the pattern of

exhausted postsynaptic terminals becomes invisible and, in our model, the short term memory of the word is gone. The long term memory remains as the pattern of the neuronal firing which is formed when a word is read.

The evidence for short term memory being related to exocytosis/endocytosis includes the shapes and slopes of the exocytosis and endocytosis curves (linear and logarithmic with time). The logarithmic decay for cued recall and recognition of words in the experiments by Rubin et al. (1999) has an associated slope of $-0.11/\text{s}$ and endocytosis of a variety of cell ensembles in mouse hippocampus (the hippocampus is thought to play an important role in memory) varies between -0.11 and $-0.14/\text{s}$ (Tarnow (2009) shows that experimental data, typically fitted to a sum of exponentials, is actually logarithmic). Exocytosis takes place in a quasi linear increase with a time. In one example (Dobrunz 2002) the associated time constant (in Eq. 1 lagged as tagging time) is about 1 s, similar to my finding of a tagging time of 0.3–1.8 s for words.

Results: the Murdock (1962) data and Murdock and Okada (1970) reanalyzed

The Murdock (1962) data can be downloaded from the Computational Memory Lab at University of Pennsylvania (<http://memory.psych.upenn.edu/DataArchive>). The data consists of six experiments in which lists of words, selected from 4,000 of the most common words in English, were read to groups of subjects to the accompaniment of a metronome (to fix the presentation rate) after which the subjects wrote down as many of the words as they could remember for a period of 1.5 min. The experiments are typically labeled M–N in which M is the number of list items and N is the number of seconds between item presentations. I calculated the average recall positions for the

Fig. 1 Stylized drawings of proposed short term memory. In the *left panel* is shown a neuron with synapses before tagging/exocytosis. In the *right panel* is shown the same neuron with expanded synapses after tagging/exocytosis (The synapses are shown as expanded since

the vesicles fuse with the presynaptic membrane increasing its surface area). In the *right panel* the neuron is part of a tagged long term memory trace

various items to estimate the exact time that an item was recalled (a 1 s average delay for each recall position was added to the Murdock (1962) data since it was not in the experimental data; the functional form of the data did not seem sensitive to the value of this parameter).

Murdock and Okada (1970) investigated inter-response times in single-trial free recall. Each of 72 subjects was given 20 test lists with 20-word lists presented visually at either 1 or 2 words per second. Spoken recall was tape-recorded and timed afterwards. The data can be also downloaded from the Computational Memory Lab at University of Pennsylvania (<http://memory.psych.upenn.edu/DataArchive>). The average inter-response times after the first few items were a function of the number of items left to be found in memory (Tarnow 2010b) and this function was used to get a second estimate of the recall times in the Murdock (1962) data (1 item left—8.8 s inter-response time, 2 items left—3.9 s, 3 items left—2.1 s, 4 items left—1.5 s, 5 items left—1.2 s, 6 or more items left 1 s). Both estimates of the recall times in Murdock (1962) gave similar qualitative results.

The recall probabilities from Murdock (1962) versus average recall time are displayed on lin-log plots in Fig. 2. In Fig. 2a we see the results from the three experiments with the 0.5 items/s presentation rate. Without the initial three items (right panel) all the remaining items lie on the same curve. They form a straight line, a logarithmic time dependence (see Eq. 1), just as is found in serial recall/recognition (Tarnow 2008). In Fig. 2b we see the results from the three experiments with a 1 item/s presentation time. The initial logarithmic time constant is the same for the three experiments with the 1 item/s presentation rate but higher than for the 0.5 item/s presentation rate. With the exceptions of the three initial items, items with the same presentation rate also lie on the same curve. At recall probabilities lower than 0.2–0.3, the logarithmic curve changes slope, giving it a “hockey stick” shape.

In Fig. 2c is displayed the data from Murdock and Okada (1970) in which the interitem presentation times were measured rather than estimated (the word articulation time was not measured and was fixed it at a reasonable value of 0.5 words/s—the shapes of the curves were not sensitive to the particular value). With the exception of the three initial items the remaining items seem to fit two smooth curves, one for each presentation rate (1 and 2 items/s). Again there is a change in the shape of the curve, this time for recall probabilities lower than 0.35.

In Fig. 3 is shown the decay of the so far removed first list items, second list items and third list items. The left panel shows the results from the 0.5 items/s presentation rate and the right panel shows the results from the 1 item/s presentation rate. The most recent list item which did not decay much is included as a guide to the eye. For the 0.5

Fig. 2 **a** Probability of recall of an item as a function of the difference in time between study time and actual recall time for the three conditions with presentation rate of 0.5 items/s. The time axis is logarithmic. The interrecall time was estimated at 1 s. The *left panel* includes all data points, the *right panel* includes all but the initial three data points. Note the common logarithmic dependence (a *straight line*) that appears without the initial three data points. The labels 10-2, 15-2 and 20-2 stand for 10, 15 and 20 words at a presentation rate of one every 2 s. **b** Probability of recall of an item as a function of the difference in time between study time and actual recall time for the three conditions with presentation rate of 1 item/s. The time axis is logarithmic. The interrecall time was guessed at 1 s. The *left panel* includes all data points, the *right panel* includes all but the initial three data points. Note the common logarithmic dependence (a *straight line*) that appears without the initial three data points. The labels 20-1, 30-1, 40-1 stand for 20, 30 and 40 words at a presentation rate of one every second. **c** Probability of recall of an item as a function of the difference in time between study time and actual recall time for the data in Murdock and Okada (1970). The *unfilled circles* correspond to the presentation rate of 1 item/s and the *filled circles* correspond to the presentation rate of 2 items/s. The time axis is logarithmic. The interrecall time was calculated from the data with the average word pronunciation length guessed at 0.5 s. The *left panel* includes all data points, the *right panel* includes all but the initial three data points. Note the approximate logarithmic dependence

items/s presentation rate the decay appears to be logarithmic across the three experiments; the fit is not as good for the 1 item/s presentation rate.

The slopes resulting from the Murdock (1962) data and the Murdock and Okada (1970) data show well defined trends, see Table 1. The slopes vary from small to large presentation rates. It also vary from the first item (least steep) to recent items (steeper). Since the first item did not have an item preceding it, it effectively has a lower presentation rate explaining the shallower slope. It is not clear why the Murdock (1962) data at a presentation rate of 1/s is different from the corresponding Murdock and Okada (1970) data. The slope of the logarithmic curve for a high statistic measurement by Rubin et al. (1999) calculated in Tarnow (2009) was $-0.11/s$ (see Fig. 4). The presentation rate was 0.17 items/s (5 s word display and 1 s null display). There is no primacy effect in this figure because the 47 min experiment had only one initial item which is buried in the statistics of the other items. The primacy effect at this low presentation rate may disappear altogether.

Our findings thus far enables us to explain the bowing effect as arising from presentation rate dependent decay. In Fig. 5 is shown results of a simulated 10 item free recall test. The upper panel in Fig. 5 shows the recency effect that comes about because of a single logarithmic decay (0.5 items/s). The middle panel in Fig. 5 shows what happens when the first three items decay with different rate constants (0.2, 0.3 and 0.4 items/s)—the primacy (and bowing) effect appears. The bottom panel in Fig. 5 shows the same bowing plotted on the more usual recall probability versus item number curve.

There is an important feature in Fig. 3 that seems to contradict our premise that decay and presentation rate explains all of the data. If decay and presentation rate did explain all of the data, then the first list items should fall on the same line for both presentation rates since the presentation rate is equally ill defined and their recall time is roughly the same. They do not. Rather, the probabilities of

the first list items seem to be a constant shift of about 0.22 down as the post-item presentation rate is increased from 0.5 to 1 per s (from left to right in Fig. 3). The tagging/retagging theory does have a potential explanation for this shift: there is a minimum time, the tagging time, needed to initially fully tag a short term memory (see Eq. 1). This tagging time ranges from 0.2 s (nonsense words) to 1.3 s

Fig. 3 Probability of recall for the initial three items (first items in *squares*, second items in *diamonds*, third items in *triangles*) in the three conditions with presentation rate of 0.5 items/s (*left panel*) and 1 item/s (*right panel*). Note the slower decay associated with the 0.5 item/s presentation rate and also associated with the item order (lowest for the first item)

Fig. 4 Tagging remaining averaged over recall and recognition memory items from Tarnow (2008). The curve represents a two parameter logarithmic fit, moving $t = 0$ s to $t = 0.5$ s to avoid a divergence

(unusual words) to 1.7 s (meaningful words) (Tarnow 2008). Since the presentation rate of 1/s affords only 1 s for the tagging, the tagging level of the initial word may not be 100% by the time the tagging is interrupted by the next word. I do not have a measure of the tagging time for the words in Murdock (1962) which were not unusual but samples of the 4,000 most common words in English. If the tagging time is similar to unusual words, the tagging level of the initial item would be only $1/1.3 = 77\%$ which is similar to $1 - 0.22 = 78\%$.

This introduces another contradiction: if the tagging level in the 1/s presentation rate experiments is limited to 78%, then no items should be remembered better than 78%. The last two items in the 20-1, 30-1 and 40-1 data are remembered better than that. The last item can be excluded since it is not interrupted in the tagging process (not immediately followed by another word) and thus can be fully tagged. The only remaining item that is remembered better than 78% is the second to last item which is remembered 82–89% of the time in the 20-1, 30-1 and 40-1 data. This item forces us to postulate a presentation rate

Table 1 Slopes of logarithmic fits to macroscopic data from Murdock (1962), Murdock and Okada (1970) and Rubin et al. (1999)

Presentation rate (items/s)	First item decay rate (items/s)	Second item decay rate (items/s)	Third item decay rate (items/s)	Recent items decay rate (items/s)
0.17 ^a (Rubin et al. 1999)				−0.11 ^a
0.5 (Murdock 1962)	−0.15	−0.21	−0.23	−0.29 (−0.42)
1 (Murdock 1962)	−0.23 (when corrected for incomplete tagging this decay rate may be −0.15)	−0.29	−0.36	−0.69 (−0.65)
1 (Murdock and Okada 1970)				−0.5
2 (Murdock and Okada 1970)				−0.6

^a A recognition and cued recall experiment. The recent item decay rate for the Murdock (1962) experiments displays two sets of numbers corresponding to the two assumptions of the interitem recall times—constant interval and a varying interval obtained from the Murdock and Okada (1970) data as described above

Fig. 5 *Top panel* probability of recall of an item as a function of the difference in time between study time and average recall time. Items decay with the same decay constant. *Middle panel* probability of recall of an item as a function of the difference in time between study time and actual recall time. Three initial items decay with different decay constants. *Bottom panel* results from *middle panel* re-graphed as a function of item number rather than recall time

dependence in the tagging process: as the presentation rate increases the tagging time decreases. This presents a Heisenbergesque principle: the higher the presentation rate the quicker words can be tagged but also the quicker the tagging disappears.

Fig. 6 **a** Difference in probability of recall from 20-2 base line data in which differences were calculated pairwise starting with the last presented item and then moving back to the second last presented item, etc. Item number 1 corresponds to the first presented item for the 10-2 and 15-2 data but the 11th and 6th items in the corresponding 20-2 data. **b** Difference in probability of recall from 40-1 base line data in which differences were calculated pairwise starting with the last presented item and then moving back to the second last presented item, etc. Item number 1 corresponds to the first presented item for the 20-1 and 30-1 data but the 21st and 11th items in the corresponding 40-1 data

Finally, consider the item-by-item differences between, for example, the 20-1 and 40-1 data, matching the twenty items from the 20-1 data with the last twenty items of the 40-1 data. We should find that they would fluctuate around zero for the last presented items, as the presentation rate becomes well defined, but show a large difference close to the initial 20-1 item when the presentation rate of the 20-1 data is not well defined. These differences are shown in Fig. 6 and seem to bear out our reasoning. The decay to the zero level fluctuations is faster for the faster presentation rate (Fig. 6b) so for the slower 0.5/s presentation rate the zero level fluctuations have not quite set in at the lower

item counts used (Fig. 6a). The careful reader will also note that the 1/s presentation rate data fluctuates not around 0 but rather around 0.04 (Fig. 6b). I do not have an explanation for that offset.

The shape of the item-by-item difference curves for similar presentation rates but different number of items suggests a well-defined quantitative measure of the primacy effect: perform memory tests using two item series, one short of length S and one long of length L . The primacy effect is the difference between the recall/recognition probability of item 1 of the short series and item $L-S+1$ of the long series. $L-S$ should be sufficiently large so that the differences in recall/recognition probabilities at the final item is 0. The primacy effect is a function of the presentation rate; it should go to zero as the presentation rate decreases. As long as the presentation interval allows for full tagging, this measure of the primacy effect is well defined. From Fig. 6a we see that the primacy effect is about 0.37 at a presentation rate of 0.5/s. In Fig. 6b we read off a primacy effect of about 0.33 to which we have to add the 0.22 that was missing (see above) resulting in 0.55. Thus as the presentation rate is increased the primacy effect increases. A well defined primacy measure is extremely important since it allows one to make well defined individual comparisons and may also help diagnose neurological diseases.

Proposed biochemistry mechanism for presentation rate dependency of short term memory decay: Ca^{2+} catalyzed endocytosis

If we speculate that short term memory is synaptic exo/endocytosis of neurotransmitters (Tarnow 2009), then what would explain the presentation rate dependency of the memory decay found? We have four findings:

1. Increased presentation rate increases the memory decay rate (Table 1).
2. Subsequent items decay faster (Table 1). Other than limiting the tagging time, the influence does not go the other way—if it did the last presented items would not decay with the same speed as the previous items (just like the first presented items decay with different speeds from the subsequent items).
3. The more previous items per unit time, the larger the total effect (Fig. 3) but with each previous item the effect per item diminishes. It diminishes the quickest for the fastest presentation rates (Fig. 6).

It would seem that something in the synaptic exo/endocytosis processes might be catalyzing the endocytosis. Ca^{2+} ions can catalyze endocytosis: Sankaranarayanan and Ryan (2001) showed that intracellular Ca^{2+} ions can

regulate the speed of endocytosis (see also Wu et al. 2007, 2009; Yao et al. 2009). They found that the higher the Ca^{2+} concentration the faster the endocytosis proceeded. In Fig. 7 we see that the decay rates almost double as the intracellular Ca^{2+} concentration is doubled (see Table 2). If one compares Tables 1 (macroscopic properties) and 2 (microscopic properties) one finds that the numerical values are in the same range. There is also evidence that the concentration of intracellular Ca^{2+} ions can increase after repeated exocytosis: As the number of action potentials increase, Regehr et al. (1994) found that the presynaptic residual free intracellular Ca^{2+} level in the mossy fiber in the guinea pig hippocampus increased (see also, for example, Hofer and Lefkimmatis 2007).

The biochemistry of calcium participation in exo/endocytosis is exceedingly complex and much of it unknown (Yao et al. 2009). Intracellular calcium levels may increase by action of the endoplasmic reticulum (Verkhratsky 2005) though why an increased presentation rate would have that effect is not clear. There is a hypothesis that Alzheimer's disease causes the endoplasmic reticulum to leak calcium

Fig. 7 Data from Fig. 4c in Sankaranarayanan and Ryan (2001) of fluorescence (arbitrary units) and time (seconds). For a $1\times$ intracellular concentration of Ca^{2+} (squares) the logarithmic fit decays with a -0.6 units/s slope, for a $2\times$ concentration of Ca^{2+} (diamonds) the slope is -1.0 units/s

Table 2 Slopes of logarithmic fits to microscopic data from Sankaranarayanan and Ryan (2001)

Intracellular Ca^{2+} concentration (mM)	Decay rate (items/s)
In vivo	-0.11 to -0.14
$1\times$ constant	-0.58 units
$2\times$ constant	-1.04 units

The intracellular Ca^{2+} concentration was manipulated via the extracellular Ca^{2+} concentration. The actual intracellular Ca^{2+} concentration is not known except that it was shown to be proportional to the extracellular Ca^{2+} concentration

leading to intracellular calcium overload (Sammels et al. 2010) which according to our speculation would lead to the characteristic quicker memory decay.

If our speculations are correct, there are many missing pieces in between the macroscopic memory effects and the synaptic biochemistry. How do previous items cause calcium catalysis for subsequent items? At what point in the cognitive network might such a communication occur? For example, if words are presented visually, would changing the font impact the primacy effect (for considerations of font impact on priming, see Reder et al. 2009). Is this presentation rate effect related to, or even the same thing, as priming? Do fluctuations in extracellular calcium levels impact memory? The parathyroid regulates blood calcium levels to within 1% and associated problems causes cognitive issues that are reversed upon parathyroidectomy (Roman et al. 2005). Other chemicals such as caffeine and the poisonous alkaloid Ryanodine affect intracellular calcium levels (Scott and Rusakov 2006)—do they have an effect on short term memory? One can also ask whether there are any collective effects of the neuronal ensemble that would change the conclusions.

How the tagging/retagging theory relates to other theories

There is a considerable literature that purports to show that decay theories are unlikely to be valid. Berman et al. (2009) argue that interference is much more important than is decay. Here they define interference broadly: “interfering” items do not have to be similar, just tax short term memory like in the case of the counting backwards of Peterson and Peterson (1959). They state that interference “can be produced by other verbalizable items that are not similar to the to-be-remembered material.” I believe it is important to better probe this ill-defined statement: narrowing down cognitive effects to particular points in the cognitive network will tell us more about what and why interference would occur if it does. These authors also include a condition for decay theories: there should be a proposed process for the memory decay—the tagging/re-tagging theory fulfills this condition.

Lewandowsky et al. (2008), question decay theories on the basis of incorrect quantitative predictions of decay rates (the theories giving incorrect predictions were SIMPLE, ACT-R, PM and Trace decay). This objection does not pertain to the tagging/retagging theory which is a phenomenological theory.

Lewandowsky et al. (2008) further argue that decay theorists can hide behind the concept of rehearsal and that rehearsal has to be carefully monitored. They state that “the notion of rehearsal is indispensable to decay models

because they otherwise could not account for any persistence of immediate memory beyond a few seconds.” It was argued elsewhere that rehearsal is an ill-defined concept (Tarnow 2010a) and consequently the tagging/retagging theory has no rehearsal built in but it nevertheless describes the experiments of Rubin et al. (1999) in which memories last a full 20 min.

Lewandowsky et al. (2008) also argue that the experiments of Berman et al. (2009) show that “information no longer relevant lingers undiminished over time unless cleared by intervening cognitive events.” The authors also argue that the small decreases in recall at different recall speeds, or equivalent, longer delays show that there is no decay. A small decay is nevertheless a decay and can be accommodated in the tagging/retagging theory, depending upon when the delay occurs and what the presentation rate is.

The serial order in a box (SOB—abbreviation from model authors) interference model is a connectionist model, applied to serial recall data, in which information is stored in an associative network. The encoding strength is a function of its novelty or dissimilarity compared to what is already in the network, this being derived from the “principle that memory avoids recording redundant information” (Oberauer and Lewandowsky 2008). But the novelty measure, is not well defined (Barrouillet and Camos 2009). In their own words: “feature overlap, (not similarity) causes interference” (Lewandowsky et al. 2009). There is only one memory “store” in the model (S. Farrell, private communication) which makes it intellectually consistent but which contrasts with biochemical findings that there is a difference between short and long term memory encoding—the protein synthesis required for the latter (Kandel 2001).

While the tagging/retagging theory attempts to anchor its generalizations in biochemistry, the interference model is constructed using theoretical properties and rules that do not relate to the underlying biochemistry. These properties include “energy-gated coding”, “primacy gradient”, “response selection mechanism”, “output interference”, “response suppression”, etc. (Lewandowsky and Farrell 2008). The tests of my theory are simpler and more well defined: it can be done both via macroscopic memory experiments and microscopic biochemistry experiments.

The linearity in response time and probability (Tarnow 2008) seems to rule out interference theories in which an item is either there or it is not and there is no direct relationship between item response time and response probability. It is suggested that one such model, SOB, would account for response times in serial recall experiments (Farrell and Lewandowsky 2004), but it has not been applied to experiments other than serial recall (S. Farrell, private communication).

It is a challenge to the interference theorists to predict what the fMRI scans (which show transient behavior in time) would look like with an interference theory. How would it be different from a time-based decay theory? And what is the time dependence once a new item enters and replaces in whole or in part the old item—is it immediate or is it a slow process? In the tagging/retagging theory the fMRI scans which apparently probe the glial cells rebuilding the neurons after firings should show a decay similar to the logarithmic time-decay discovered if this rebuilding is correlated with endocytosis.

The time-based resource-sharing model (Barrouillet et al. 2004) states that time-based forgetting occurs and can be slowed down with attention-based refreshing (rehearsal). The more attention is required to other tasks (the more the “cognitive load” increases) the less attention can be given to rehearsal which then decreases the memory span. This model does not seem to be all that different from interference theories (where interference is so broadly defined it might be defined as “cognitive load”).

Conclusion

In this paper I showed that the curious bowing effect (Murdock 1962; Murdock and Okada 1970), in which intermediate items are recalled less well than initial or final items, is a result of decay (recency effect) and a presentation rate dependence of this decay (primacy effect). Decay rates of recent items were found to be the same for equal presentation rates but faster presentation rates were associated with steeper decay. Initial items have an effectively lower presentation rate (no previous items) and fall on a separate set of decay curves that are less steep. A measure of the primacy effect was introduced that should be important for quantitative disease diagnoses. I also found indications that the higher the presentation rate the quicker words can be tagged, analogous to the priming concept. If this is valid, the Heisenbergesque principle holds: the higher the presentation rate the quicker items can be tagged but also the quicker the tagging disappears.

These findings should please the interference theorists: it describes a comparably slow decay for slow presentation rates (a little similar to the claim that memory does not decay with time) and a higher decay for higher presentation rates (similar to the claim that interference causes decay). It should also please Time-Based Resource-Sharing model theorists since a high presentation rate is similar to a high cognitive load which in both theories lead to memory impairment.

The physiological mechanism suggested earlier as responsible for the decay of short term memory, synaptic endocytosis (Tarnow 2009), might be catalyzed by changes

in the intra-cellular level of Ca^{2+} ions which is the mechanism suggested for the presentation rate dependency of the memory decay: a higher presentation rate may lead to a higher concentration of intracellular Ca^{2+} which leads to a quicker logarithmic endocytosis. I do not have a proposed mechanism for how the intracellular level is affected by previous item presentations.

Long term memories are probably more likely to form the longer short term memories last. Thus the importance of a decreased presentation rate on memory cannot be emphasized enough. It suggests a basis for memory loss in adults with a lot of things going on, a basis for the importance of slow music practice, a basis for long term memory deficiencies for people with attention deficits who may be artificially increasing the presentation rates of their surroundings (though it is not settled whether such memory impairments exist, see Castellanos and Tannock 2002). It also suggests that speed reading, in which the presentation rate is artificially increased, is likely to be ineffective.

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