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Temporal twilight zone and beyond: Timing mechanisms in consciously delayed actions

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Abstract: Precise timing is essential for many kinds of human behavior. When a fastest response is not required, movements are initiated at the appropriate time requiring an anticipatory temporal component. Temporal mechanisms for movements with such an anticipatory component are not yet sufficiently understood; in particular, it is not known whether on the operational level for delayed movements distinct time windows are used or whether anticipatory control is characterized by continuous temporal processing. With a modified reaction-time paradigm, we asked participants to act with predefined time delays between 400 and 5000 ms; after each individual trial, a numerical feedback was provided which allowed correction of the response time for each next trial. Visual stimuli (Experiment 1) and auditory stimuli (Experiment 2) were used. In the statistical analyses, piecewise linear models and exponential decay models for the response variability of different delay times were compared. These analyses favored piecewise linear models; a decreasing variability with increasing delay of voluntary controlled actions was observed up to ~1 s, followed by close to constant variability beyond this delay. We suggest that precise temporal control of voluntary delayed movements is reached only after a "temporal twilight zone" of ~1 s, which apparently marks a temporal border between two different timing mechanisms.

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Temporal control in voluntarily delayed actions

Many human behaviors are dependent on accurate and precise processing of temporal information. Instead of being initiated as fast as possible, some behavioral processes require delaying the action for a certain time period. For instance, because greater distances are involved in tennis, players have time to monitor the direction and speed of the ball, and hit the ball at the appropriate time. Under such conditions, some anticipatory temporal processing is required for the intended actions. To predict when events will happen and to process efficiently temporal patterns of stimuli belong without question to the most basic functions of the brain (e.g., Buzsáki & Llinás, 2017; Ivry, 1996; Meck & Ivry, 2016; Repp & Su, 2013). The research presented here is focused on such event timing using a modified reaction-time paradigm. In a typical reaction-time experiment, participants are instructed to respond as quickly and accurately as possible, either in a simple or choice reaction-time paradigm (Niemi & Näätänen, 1981). This paradigm can be modified if participants are instructed to delay the response by a specified interval of hesitation. The present investigation, thus, focuses on action as a voluntary process embedded in a dynamic and anticipatory temporality aiming to shed some new light on the temporal constraints of behavioral control.

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Characterizing the internal clock

Central to the study of event timing has been the question of whether there exists an internal "clock" that measures temporal intervals (or regulates the onset of movements) required for the motor events. Evidence in support of the internal "clock" hypothesis comes from various sources, and a single timing mechanism has been suggested; that is, that temporal variability can be explained by a single timing mechanism (e.g., Matell & Meck, 2000; Treisman, Faulkner, & Naish, 1992). However, that perceptual processes and motor acts are controlled by different discrete time windows has also been conceptualized (Bao, 2017; Bao, Szymaszek, et al., 2013; 2014, 2015; Bao, Yang, Lin, & Pöppel, 2016; Fingelkurts & Fingelkurts, 2006; Pöppel, 1970, 1997, 2009; Pöppel & Logothetis, 1986; van Rullen & Koch, 2003; Wang, Lin, Zhou, Pöppel, & Bao, 2016); empirical evidence has suggested indeed such distinct timing mechanisms with different durations.

For instance, in time reproduction tasks, participants are presented with an auditory or a visual stimulus of a specific duration, then asked to interrupt a second sound or light once it has reached the same duration; short durations of 2 to 3 s are reproduced almost accurately whereas longer intervals are systematically underestimated (e.g., Pöppel, 1972; Ulbrich, Churan, Fink, & Wittmann, 2007). Specific insight into a time window of some 3 s comes also from neuropsychological studies with autistic children (Szelag, Kowalska, Galkowski, & Pöppel, 2004) or patients with aphasia (Szelag, von Steinbüchel, & Pöppel, 1997) in which some underlying temporal mechanisms have been disclosed. In temporal reproduction tasks, subjects with autism tend to reproduce any interval close to 3 s; this observation allows a view into the eigen operations of the temporal machinery, suggesting that in such patients, an endogenous temporal process presumably implemented by relaxation oscillations (Pöppel, 1972) can no longer be modulated by external temporal stimuli. On the contrary, some patients with Broca's aphasia have apparently lost their time window of some 3 s as an implicit neural basis of cognitive processing, and they adopt a new explicit strategy to deal with temporal information (Szelag et al., 1997).

Further and rather different paradigms also support the existence of a discrete 3-s time window. Experiments on sensorimotor synchronization have shown that up to a 2- to 3-s stimulus occurrence can be anticipated with high precision, but not beyond that (Mates, Müller, Radil, & Pöppel, 1994). Miyake, Onishi, and Pöppel (2004) showed

that a secondary working-memory task affected the accuracy of synchronization only with interstimulus intervals above this temporal interval; below this interval, the memory task had no influence on performance. Neuroimaging studies have shown that different regions are involved in temporal perception, which include areas in the posterior parietal lobe, frontal lobe, insular cortex, basal ganglia, and cerebellum (e.g., Coull, Cheng, & Meck, 2011; Wiener, Turkeltaub, & Coslett, 2010), although it still has to be clarified whether results of these neural activities support discrete timing mechanisms.

Modeling temporal variations

The basic hypothesis of the present study is that the variability of responses for different delay intervals will reflect specific timing mechanisms. By systematically varying the delay time of responses, the temporal acuity can be compared across different intervals. It is assumed that potentially distinct mechanisms do not produce the same pattern for response variability. If the argument of discrete temporal processing is correct, and different timing mechanisms are operating for motor control in different delay intervals, then the transition from one mechanism to the other one has to be detectable, which could indicate the boundary between different statistical models have to be considered.

We consider eight such distinct models with respect to response variability (Figure 1). The constant model (Figure 1A) predicts performance variability to remain a constant proportion for the target duration over the entire temporal range, which represents the scalar property (i.e., "Weber's Law") applied to timing. Although the scalar property is suggested by pacemaker/accumulator models, a simplified version of the accumulator system whose variance is based solely on the Poisson process would show an exponential decrease in performance variability with an increasing number of pacemaker counts (Gibbon, 1992); data following this pattern would support an exponential decay model (Figure 1B). If a single regression line through all data points with a positive slope could explain the data, the ideal model should be a linear increase model (Figure 1C) with greater temporal variability for longer intervals than for shorter intervals. Interestingly, in contrast, Lewis and Miall (2009) discovered a linear steady decrease of the coefficient of variation (CV) in a temporal reproduction task when durations increased from 68 ms to 16.7 min, which leads to our fourth hypothesis: the linear decrease



Figure 1. Eight statistical models proposed to fit the response variability as a function of delay intervals. The lines represent potential regression lines. If the data for the different delay intervals are projected onto the ordinate, they would give rise to different response distributions indicated by the shaded sketched histograms. A constant function (Model A) would be reflected in a unimodal distribution, a step function (Model E) in a bimodal distribution, or linear decrease or increase functions (Models C and D) in rectangular distributions.

model (Figure 1D). If there is a break point in the duration range, monotonically increasing or decreasing models no longer would make an accurate prediction; the step model (Figure 1E) would apply, if such a sudden change in relative variability would be observed. The quadratic, U-shaped model (Figure 1F) has previously been found for temporal reproduction tasks, with a minimum CV observed at 1000 ms (Bangert, Reuter-Lorenz, & Seidler, 2011). A review by Gibbon, Malapani, Dale, and Gallistel (1997) has evaluated a number of studies and has identified that CVs increased up to 100 ms, stayed constant until 1500 ms, and then increased again for longer durations. As no delay interval in our study was less than 100 ms, we excluded this part in Figure 1G. Finally, a piecewise linear decreasing model (Figure 1H) would show declining variability up to a break point, and a constant value beyond. In this study, we measured the response variability over different time intervals to test which model can provide the best fit to the experimental data.

Sensory modalities and temporal processing

Furthermore, timing might be of a general nature and be independent of the sensory modality, or timing might be part of a specific neural processing within a sensory modality (Buonomano, 2000; Burr, Tozzi, & Morrone, 2007). The auditory modality might have a special status, as compared to other sensory modalities with respect to the encoding of temporal information (e.g., Guttman, Gilroy, & Blake, 2005; for a review, see Ivry & Schlerf, 2008). An example of modality-specific timing mechanisms comes from tapping studies; it has been shown that visual pacing was generally more variable than tapping in the context of auditory stimuli (Jäncke, Loose, Lutz, Specht, & Shah, 2000). When judging the duration of a visual stimulus, an increase in the difference threshold was observed on trials in which repetitive transcranial magnetic stimulation (rTMS) was applied over the middle temporal visual area (V5/MT) whereas no change in performance was found when participants judged the duration of a tone (Bueti, Bahrami, & Walsh, 2008). When compared with networks recruited for an auditory tapping task and following a continuation task without stimuli, the visual-specific activity remained high in area V5/MT after the information was terminated (Jantzen, Steinberg, & Kelso, 2005). In contrast, evidence supporting a unified timing mechanism can also be found in some studies. For example, a recent study has observed that there is an "across-senses" (i.e., acrossmodalities) effect of perceptual modality (visual vs. auditory) in a rate perception task (Levitan, Ban, Stiles, & Shimojo, 2015). In addition, cross-modal effects have been well-illustrated on different percepts. Temporal processing in one modality may be affected by the signals from other modalities (Burr, Banks, & Morrone, 2009; Van Wassenhove, Buonomano, Shimojo, & Shams, 2008; Vicario, Rappo, Pepi, & Oliveri, 2009). On the basis of this unclear situation, we applied visual and auditory stimuli to examine whether the action mode is modality-specific or whether some differences can be observed.

Taken together, the two experiments reported here are designed to answer the following three questions: First, how does response variability evolve for different delay intervals? Second, is there a break point in response 20460260, 2020, 6, Downloaded from https://onlinelibarg.wiley.com/doi/10.1002/pchj.389 by Cochrane Germany, Wiley Online Libargy on [3108/2023]. See the Terms and Conditions (https://onlinelibargy.wiley.com/ems-and-conditions) on Wiley Online Libargy for rules of use; OA articles are governed by the applicable Creative Commons License

variability indicating perhaps different timing mechanisms? Third, does response variability reflect modality-specific timing mechanisms or a common timing mechanism?

Methods

Experiment 1

Participants

Twenty right-handed students from Peking University (7 males, 18–26 years old) participated in the experiment. All participants had normal or corrected-to-normal vision; they had given written informed consent before the experiments and received moderate rewards for their participation. The experiments had been approved by the Committee for Protecting Human and Animal Subjects in the School of Psychological and Cognitive Sciences at Peking University and were in accordance with the Declaration of Helsinki.

Apparatus

Visual stimuli were generated using MATLAB 7.13 (MathWorks, Natick, MA, USA) in conjunction with the Psychophysics Toolbox (Brainard, 1997) and displayed on a 19-in CRT monitor (1024*768 resolution, 100 Hz refresh rate). Responses were collected on a keyboard.

Materials and procedure

The experiment was conducted in a dimly lit room. Participants were seated 57 cm in front of the computer screen with their heads staying on an adjustable chin rest. Each trial began with a central fixation cross (white, 0.8°) on a black background for 1000 ms. Then a white solid dot

 (0.8°) serving as the target was presented for 100 ms centered on the screen (see Figure 2). Participants were required to respond to the target as fast as possible or with predefined delays between 400 and 5000 ms. Ten delayed intervals were tested in separate blocks (400, 600, 800, 1000, 1500, 2000, 2500, 3000, 4000, 5000 ms). The goal of the participants was to estimate passage of the time interval from the onset of the target, and to press a key as closely as possible with the requested delay time. For instance, when the delay interval was 400 ms, the participant's task was to press the key when he or she estimated that precisely 400 ms had elapsed since the appearance of the target. The key-press response may be either shorter or longer than the target delay time. In either case, feedback was displayed on the screen, indicating the participant's response time of the trial. Moreover, participants were asked not to do any mental counting, or to produce any movements beyond those required to give responses after a certain delay. For each delay time, 40 trials were sequentially employed, and the intertrial interval was 1.5 s. There were 11 blocks in total: 1 with the fastest reaction and the remaining randomly ordered 10 blocks with various delays. Each block began with the indication of the required target response time by a number on the screen (e.g., "If a target appears, please wait for 400 ms, and press the SPACE-BAR.") Before the experimental session, participants completed 20 practice trials to familiarize themselves with the experimental procedure. The procedure in the training phase was equivalent to that of the formal experiment, except that only two delay intervals were retained, which were randomly selected from the 10 intervals and assigned to each participant.



Figure 2. Basic trial sequences in Experiments 1 and 2. Participants were required to respond to the target as fast as possible or with predefined delays (400, 600, 800, 1000, 1500, 2000, 2500, 3000, 4000, 5000 ms).

Data analysis

For each delay condition, a variety of summary statistics were used to measure the dispersion or variability of the corresponding data set. Four measures are considered here: the coefficient of variation (standard deviation of response times divided by the mean; CV), the coefficient of dispersion (mean absolute deviation from the median divided by median; CD), the relative mean difference (mean of all possible absolute values of differences between two response values divided by mean; RMD), and the coefficient of quartile deviation (difference between the first and third quartiles divided by the sum of them; CQ). They differ in the weight placed on observations in the tails. All of them are measures of response variability/relative accuracy, where lower values indicate a better ability to consistently generate a movement with a particular delay, thereby allowing for comparisons across different delay conditions. These variability indices were calculated by averaging estimates within each participant and then across participants.

A flexible approach to model the nonlinear form of trends is the piecewise linear. This approach breaks up the linear trend into separate linear segments or pieces of different slopes, which are tied together by turning points. We estimate a piecewise linear regression (Figure 3, solid lines) to fit the four variability indices at group level with two line segments connected at the turning point:

$$f(x) = \begin{cases} \beta_0 + \beta_1 x & \text{for } x \le \alpha \\ \beta_0 + \beta_1 x + \beta_2 (x - \alpha) & \text{for } x > \alpha \end{cases}$$

One equation gives the values of f(x) when x is less than or equal to α , and the other equation gives the values of f (x) when x is greater than α . An exponential decay equation models many physical and biological processes. It is used whenever the rate at which something happens is proportional to the amount which is left. We also estimate an exponential decay function (Figure 3, dashed lines) to fit the same data sets for comparison:

$$f(x) = \alpha + (\beta - a)e^{-\frac{3}{2}}$$

where α is the upper limit (attained at x = 0), β is the lower limit reached for x going to infinity, and $\lambda > 0$ is determining the steepness of the decay. The function is monotonically decreasing.

To identify the best-fitting model, we use residual standard deviation (RSD) to compare the piecewise linear models with exponential decay models because there is no formal test available for the direct comparison of a piecewise regression model to an exponential decay model (Ryan & Porth, 2007). RSD is a measure of the distance between observed and predicted values based on the model fit, and it can be calculated as the square root of the quotient of the sum of squared errors in the fitted mode divided by the residual degree of freedom (*df*). The residual *df* equals to (n-4) for our piecewise linear model with four parameters whereas that for the three-parameter exponential decay model is (n-3). The model with the lowest residual *SE* is considered the best fit. All the calculations were conducted in R (Version 3.1.3) with add-on packages "drc" (Ritz & Streibig, 2005) and "segmented" (Muggeo, 2008).

Results

The mean response times in all 11 experimental conditions are summarized in Table 1, showing that participants were very fast to adjust their response time to the predefined delay time. The estimates of each of the four variability indices (CV, CD, RMD, and CQ) across different delay conditions are shown in Figure 3A; as can be seen, all of them show a downward trend, with the maximum value observed at the first delay interval (400 ms). The variability of response decreased over the tested temporal range with a rather steep slope until a break point of ~880 ms. The average response time of the fastest reaction was 225.39 ms (marked with an asterisk in Figure 3A).

Results from the curve fits are compared in Table 2. For example, the RSD of piecewise linear model fitted into CVs is 0.554 on 6 df, and that of the exponential decay model (7 df) is 1.112. This means that on average, using the piecewise linear model to predict CV from reported CV results in an error of about 0.554 whereas the error generated from the exponential decay model is 1.112. Based on comparisons of the model RSD and the visual fit, the piecewise linear model is clearly the better fit. Responses variability drops sharply from the first until the fourth delay intervals (400-1000 ms), and then smoothly decreases and remains at low values up to the longest delay employed (5000 ms). The estimated turning point is also identified in each fit: 947.70 ms (±52.64 ms) for CV, 879.90 ms (±37.98 ms) for CD, 884.60 (±43.50 ms) for RMD, and $837.00 \ (\pm 21.94 \text{ ms})$ for CQ. All of them fall into the range of 800-1000 ms.



Figure 3. Response variability observed with different statistical indicators: the coefficient of variation (CV), the coefficient of dispersion (CD), the relative mean difference (RMD), and the coefficient of quartile deviation (CQ). Experiment 1, visual stimuli (A); Experiment 2, auditory stimuli (B). All data were fitted with an exponential decay model (dashed lines) and a piecewise linear model (solid lines). The vertical lines crossing the abscissa in A and B represent the break points of the piecewise linear function for the different statistical indicators. The fastest reaction times (225.39 ms for visual stimuli, 181.57 ms for auditory stimuli) are represented bv asterisks.

Experiment 2

To test whether the findings previously obtained in the visual modality can be replicated in a different modality, Experiment 2 employed auditory stimuli, and used a design similar to that in Experiment 1.

Participants

A new sample of 20 students from Peking University participated in the experiment (10 females; 19–28 years old, mean = 23.2, standard deviation = 2.65). All of them were right-handed and had normal hearing ability. Participants

Mean Response Tin	nes to Visual T	Fargets for Each	h Delay Interval (r	ms) in Experimer	t 1						
					Targe	et delay interval	(ms)				
	ASAP	400	600	800	1000	1500	2000	2500	3000	4000	5000
Response 2	225.39	431.89	619.70	796.55	1003.76	1475.65	1992.02 (22.74)	2484.76 (25-14)	3032.98	4012.84 (33-12)	4965.18
control ((±0.0)	(70.1)	(00.1)	(=)	(10.01)	(10.11)	(1.1.77)	(11.07)	((((21.00)	(10.00)
Note. SEs are indicate	ed in parenthese	es.									
Table 3											
Mean Response Tin	nes to Auditory	y Targets for Ea	ach Delay Interval	l (ms) in Experin	lent 2						
					Ta	rget delay interv	val (ms)				
	ASAP	400	600	800	1000	1500	2000	2500	3000	4000	5000
Response times	181.57	468.59	635.27	847.16	1025.30	1517.27	2032.99	2487.44	3004.92	4025.43	5014.20

(43.53)

30.94)

26.10)

21.48)

80)

29.

(19.63)

82)

(13.

(11.85)

(8.74)

(8.35)

(9.15)

Vote. SEs are indicated in parentheses

Model SEs for the Exponential Decay and Piecewise Linear Models Fitted to Response Variability in Experiment 1

	Residual	SD (RSD)
	Exponential decay model	Piecewise Linear model
Coefficient of variation	1.11	0.55
Coefficient of dispersion	1.79	0.74
Relative mean difference	1.06	0.50
Coefficient of quartile deviation	1.13	0.32

were paid for their participation. This study was approved by the departmental ethical committee of Peking University. Subjects took part in the experiment after giving informed written consent.

Apparatus, stimuli, and procedure

The same experimental setup and procedure were used as those in Experiment 1, with the following exceptions. Pure sinusoidal tones of 441 Hz served as stimuli. They were presented binaurally via stereo headphones at an intensity of \sim 70 dB(A) SPL (a-weighted decibels sound pressure level) as measured at the participant's ears. The duration was 100 ms. Before the presentation of the auditory stimuli, a steady white fixation cross was presented in the middle of the screen that was replaced by a black blank screen when a response was required.

Design, and data analysis

Details were equivalent to those of Experiment 1. The four measures of response variability/relative accuracy (CV, SD, RMD, and CQ) were applied to participants' data. Then, we estimated a piecewise linear regression and exponential decay function to fit the four variability indices at the group level.

Results

Response times with *SEs* for each experimental condition, averaged across all participants, are shown in Table 3. Overall, participants quickly adjusted their response time to the predefined delay time, and the better response times were actually observed for the longer delay times. Response variability observed in four different measures (CV, SD, RMD, and CQ) are shown in Figure 3B. Results from the curve fits are compared in Table 4, showing that the piecewise linear model fits the data better than the exponential decay model. The pattern observed in

Table 1

Table 4
Model SEs for the Exponential Decay and Piecewise Linear Models Fitted
to Response Variability in Experiment 2

	Residual	SD (RSD)
	Exponential decay model	Piecewise Linear model
Coefficient of variation	1.08	0.77
Coefficient of dispersion	1.23	0.94
Relative mean difference	0.81	0.61
Coefficient of quartile deviation	0.71	0.61

Experiment 1 was also observed here: decreasing variability with increasing requested delay interval, and greater variability for shorter delays compared to longer delays. The estimated turning point was 1074.5 ms (averaged across the four variability measures). We compared distributions of subjects' variabilities in visual and auditory conditions, at the 800-ms and 1000-ms delay intervals; no difference was observed as indicated by nonsignificant two-sample Kolmogorov-Smirnov test ps > 0.8. Thus, it can be inferred that the turning points defined nearby are not significantly different between visual and auditory conditions. We also fitted individual participants' data and evaluated the reliability of the effect across participants. Outputs from two representative individuals are shown in Figure 4 (4A for Experiment 1 and 4B for Experiment 2) to demonstrate the model fittings at the individual level.

Discussion

Our three research questions can be answered as follows: First, response variability decreases as delay intervals to respond become longer in the time domain from 400 to 5000 ms. Second, a break point in response variability is observed at ~1 s, indicating different timing mechanisms before and after this break point. Third, response variability does not reflect modality-specific timing mechanisms because the break point is not significantly different for visual or auditory stimuli; this may suggest a common timing mechanism for the visual and auditory modality, although the lack of significant differences between visual and auditory processing cannot be used as a convincing argument for one underlying mechanism only; on a general level, note that the absence of evidence does not prove the evidence of absence. Further arguments (discussed later), however, indeed favor a common mechanism for both modalities.

Decreasing response variability across delay intervals

With respect to the first question, results of both experiments show that response variability decreases as delay intervals become longer. Szelag, Rymarczyk, and Pöppel (2001) also observed that the variability of responses was larger for short delays, as compared to longer delays. In other experiments, it has been reported that performance accuracy at longer delays can be higher than that at shorter ones (White, 2001). Taking such observations together with the findings presented in our experiments, we infer that a more precise action mode is switched on when some time has been allowed to pass. Better temporal control can apparently be achieved with delayed responses after a critical interval. The higher variability for shorter delay times is at odds with some previous reports of a constant coefficient of variation (CV) across different durations, which are taken to support the concept of a scalar property in timing tasks (Gibbon, 1991; Gibbon et al., 1997; Piras & Coull, 2011).

What might be a reason for the different results? Possibly, this is due to different experimental paradigms. Gibbon et al. (1997), for instance, identified patterns of increasing response variability for durations up to 100 ms, stable CVs from 100 ms to 1500 ms, and increasing CVs for durations of 1500 ms and longer. Grondin (2010a) found a smaller CV for 200 ms as compared to 1000 ms, regardless of the number or range of comparison intervals tested. The quadratic pattern of CVs from the study by Bangert et al. (2011) was found when adopting five intervals (300 ms, 650 ms, 1000 ms, 1350 ms, and 1700 ms) in temporal reproduction tasks. As the method in studies of time estimation is a factor influencing perceived duration (Bueti, Walsh, Frith, & Rees, 2008; Pöppel, 1972; Ulbrich et al., 2007), the differences between these results may be caused by different task requirements; different neural processes might be selected when translating sensory durations into motor programs. In temporal reproduction or production experiments, participants encode a duration, and then have to transform the duration into a motor program to produce or reproduce the duration (Grondin, 2010b). The experimental paradigm employed in the experiments reported here corresponds to the reaction-time paradigm (Niemi & Näätänen, 1981) in which one has to respond to a signal with predefined



Figure 4. Individual model fittings with an exponential decay model (dashed lines) and a piecewise linear model (solid lines). Shown are one representative participant's results in Experiment 1, visual stimuli (A), and another's results in Experiment 2, auditory stimuli (B).

delays. It is apparent that the results reported here, together with observations of previous studies, cannot be reconciled with only one functional mechanism for temporal processing and motor planning. Instead, the hypothesis of multiple clock mechanisms which is supported by a number of behavioral studies and with brain imaging research has to be favored for different sub- and suprasecond intervals (Bao et al., 2015; Bao et al., 2016; Pöppel, 1997, 2009; Wang, Lin, et al., 2016; Wiener et al., 2010; Wittmann, 2009).

Another possible interpretation of the results might be that the higher variability for shorter target intervals could be indicative of some compensatory mechanism, when accurate motor responses need to be achieved in the presence of an unstable sensory representation of temporal information. The variability might be attributed to fluctuations of attention during the passage of time or other cognitive malfunctions resulting in poor neural efficiency; thus, the higher variability may reflect such a compensatory mechanism when an accurate response is demanded for short intervals. This argument implies that the sensory representation of temporal information is more stable for longer delay intervals. When the required delay interval is long enough, movements are guided by a more stable temporal representation and, thus, guarantee an accurate outcome. Whether the attentional machinery is indeed more vulnerable for shorter temporal intervals remains an open question. Research on spatial attention and the "eccentricity effect" of attentional control throughout the visual field using the paradigm of "inhibition of return" (Bao & Pöppel, 2007; Bao, Wang, et al., 2013; Lei, Bao, Wang, & Gutyrchik, 2012) does not favor this hypothesis; this research supports the concept of a stable and highly efficient time window in the domain of some 3 s as an operative platform being used as a unifying principle (Pöppel & Bao, 2012).

Common timing mechanism across visual and auditory modalities

With respect to the third question when comparing the variances in the visual and auditory modality, no significant differences were observed. As in the first experiment where a decreasing variability with increasing delay intervals was found, the same was observed in the second experiment, although the break points were slightly, but not significantly, different in the two experiments. The decreasing variability for both the visual and the auditory tasks suggests a processing mechanism which is shared by the two modalities, at least with respect to some aspects of processing. Block, Hancock, and Zakay (2010) conducted a meta-analysis of 117 experiments showing that stimulus modality (auditory or visual) did not affect duration judgments. Boltz (2005) found that whether participants retrospectively or prospectively judged durations of naturalistic events in the auditory, visual, or audiovisual modality, no differences in either accuracy or bias were found. Interval durations themselves, however, may have some modulating effects, and one cannot exclude them in principle; for instance, different processes implicated in durations for less than ~1 s and longer durations could be suspected. The observation that the break point for visual stimuli is shorter than that for auditory stimuli (although not being significantly different) might result perhaps from differences in the central representation of temporal durations in the auditory and visual pathways. In particular, the "perceptual

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center" (Vos, Mates, & van Kruysbergen, 1995) within the two modalities might not correspond to each other, resulting in differences in central processing. It could well be, or at least cannot be excluded, that on the level of observations which are based on the real time of stimulus onset, the perceptual centers of stimuli themselves in the two modalities in fact might be more or even less similar than indicated by the physical measurements. Thus, a correspondence of the break points on the neural processing level might be shadowed. However, further research is needed to answer to this question.

Two temporal mechanisms in action mode with a break point at 1 s

The aforementioned argument implies the existence of a break point which is indeed suggested by the statistical analyses using four different measures (Figure 3) and which answers the second question. The statistical analyses lead to the conclusion that the piecewise linear function is the best explanation of the data. The decreasing variability with a break point argues against the other seven statistical models which have been indicated to be theoretically possible (Figure 1). The observation that the critical break point is close to 1 s invites to reflect in more detail a potential time window of this duration. A "temporal marker" occurring near 800 to 1100 ms has also been observed in some previous research in which a distinction between intervals above and below ~1 s has been emphasized (Lewis & Miall, 2003; Penney & Vaitilingam, 2008). Contrary to the argument that this interval reflects the border between automatic motor timing and cognitively mediated timing (Lewis & Miall, 2003), we suggest that the interval reflects the boundary between two different mechanisms that are both involved in consciously controlled actions. Our findings suggest different timing mechanisms in conscious control, and a temporal marker at 1 s reflects the boundary between these two mechanisms.

But why is it ~1 s? From previous neuropsychological research by Rubia, Schuri, von Cramon, and Pöppel (1997), it can be derived that the time window of 1 s is controlled by neural mechanisms located (at least partially) in the basal ganglia. This time window as a conventional temporal marker is most likely learned, as a reliable external physical or internal physiological unit that would match 1 s does not exist, and thus could be a sufficient temporal basis for a cognitive copy. This is very different for circadian rhythms

which correspond to the geophysical cycle of day and night (Aschoff, 1965: Bao et al., 2015). The neural mechanism for the cognitive generation of 1 s is suspected to be the consequence of two temporal eigen operations with different frequency. This hypothesis is motivated by observations with patients who have suffered local brain injuries. Some patients with injuries disrupting the connection between different areas show a unique pattern of counting in steps of 1 s; some patients count approximately twice as fast and others twice as slow in a simple counting task. A disruption in the interneuronal communication in the posterior part of the supralenticular white matter has been shown to be responsible for this counting deficit affecting a corticostrio-thalamic pathway. This disruption or disconnection can be considered as the reason for the accelerated or decelerated counting.

This phenomenon leads to the hypothesis that the learned temporal unit of 1 s is the consequence of the interaction between two oscillatory processes with approximately double and half period of the intended period of 1 s. If the two oscillatory processes are interconnected, phase coupling and thus precise control of the period length of 1 s can theoretically be achieved. If the output of 1 s is too long or too short, a precise phase coupling between the low- and high-frequency processes would be violated. As the high-frequency oscillation is embedded in the lowfrequency oscillation, a violation of phase coupling could be used in a feedback loop to readjust proper phase coupling. To initiate counting, however, a cortically controlled command has to be given, which is implemented with respect to precise timing at a lower neural level.

This consciously controlled and explicit counting interrupts an implicit temporal process which is suspected to be in operation at all times. Experimental evidence with a large number of different paradigms has proven the existence of a time window of \sim 3 s being neurally implemented presumably by relaxation oscillations (e.g., Bao, 2017; Bao et al., 2015; Mates et al., 1994; Pöppel, 1972, 1997, 2009; Szelag et al., 2004; Wang, Lin, et al., 2016). This time window represents a logistical basis of cognitive processing and, as such, has itself no conscious temporal representation. When asked to count, attention is drawn to time itself, and the implicit temporal segmentation of some 3 s is interrupted. Thus, we are dealing with both an implicit time window which has a common anthropological basis (Bao & Pöppel, 2012) and a learned time window of 1 s which is represented on an explicit or conscious level. In the counting task, the 1 s time window is overriding the otherwise continuous low-frequency oscillation of the 3-s time window. From an ecological perspective, however, note that counting is a rather rare event unless certain professional activities require explicit temporal control.

What could be the reason(s) for a disbalance in the processing systems up to the break point of 1 s? We submit the general hypothesis that the higher variability in responding for shorter delay times, and the gradual decrease of variability up to the break point of about 1 s, is the expression of an initial disbalance in the sensory and motor processing systems, modulated by a compensatory mechanism to return to a state of neural equilibrium within a given time, in the case of the observed results within \sim 1 s. It appears that between fastest reactions and optimal actions, our behavior is characterized by a "temporal twilight zone." The twilight zone is an operationally defined limit of applicability of stable temporal control. Furthermore, after the equilibrium has been reached and the disbalance between the sensory and motor processing systems has been removed (i.e., the "temporal twilight zone" has been left), the compensatory mechanism will be switched off, and reliable conscious control for actions has been gained.

Such a mechanism to return to equilibrium within a limited time has recently been described on the neural level using mismatch negativity (MMN) as an indicator in an experimental oddball paradigm. A phenomenon dubbed the "rubberband effect" indicates an anticipatory control in temporal processing (Wang et al., 2016). It was observed that the higher the amplitude of MMN, the steeper the slope to return to equilibrium; this return does not follow an exponential decay as one might have expected, but an anticipatory control mechanism appears to force the system to return to equilibrium within a given time. Presumably, such a hypothetical compensatory mechanism may also operate within a time window of ~1 s independent of sensory modality.

Taken together, we suggest that a "temporal marker" of 1 s reflects two different mechanisms, which are predominantly involved in the processing of temporal information in motor responses. Future studies should consider more experimental trials with a randomized design instead of a block design. This methodological modification may delineate potentially distinct learning curves between the two timing mechanisms, and thus bolster the hypothesis of the transition point derived from the current study. More knowledge about the functional transition will allow further study of each temporal epoch in isolation, and help to minimize the confusion that may otherwise be caused by differences between timing mechanisms that have not been identified as separate. It is crucial to consider such key points in the theoretical framework of psychological time models. Moreover, diagnostic and therapeutic tools may be developed to treat disturbed temporal mechanisms in patients with neurological malfunctions of the brain.

Disclosure of conflict of interest

All authors have no significant competing financial, professional, or personal interests that might have influenced the performance or presentation of the work described in this article.

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