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Title	New measures of functional (but not perceptual) continuity in visual grouping
Author(s)	Elliott, Mark
Publication Date	2004
Publication Information	Seifert, D., Elliott, M. A., & Giersch, A. (2004). New measures of functional (but not perceptual) continuity in visual grouping. in A. M. Oliveira, M. Teixeira, G. F. Borges & M. J. Ferro. (Eds.). Fechner Day 2004. Proceedings of the Twentieth Annual Meeting of the International Society of Psychophysics. Coimbra, Portugal. The International Society for Psychophysics, (pp. 524-529).
Publisher	International Society for Psychophysics
Item record	<a href="http://hdl.handle.net/10379/1558">http://hdl.handle.net/10379/1558</a>

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## NEW MEASURES OF FUNCTIONAL (BUT NOT PERCEPTUAL) CONTINUITY IN VISUAL GROUPING

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### ABSTRACT

*The benzodiazepine, Lorazepam enhances the efficiency of inhibitory GABA<sub>A</sub> ( $\gamma$ -aminobutyric acid) synapses in the cortex, which stabilize postsynaptic, excitatory activity by synchronizing their own discharges at around 40 Hz. Lorazepam treatment also affects contour integration processes, suggesting GABA<sub>A</sub>-mediated synchronization may be of direct influence during visuo-perceptual organization. By adding contours orthogonal (but at varying distances) to the unspecified continuances formed by the collinear arrangement of cross elements that flicker asynchronously but in a regularly arranged 3 x 3 element matrix, we found priming by means of 4 synchronized crosses presented in square arrangement was substantially increased when orthogonal contours were placed directly adjacent to the virtual continuances between the crosses, but only following lorazepam administration. We conclude that GABA<sub>A</sub>-induced inhibition influences directly the coding of relations between spatially separate visual stimuli related to the Gestalt principle of good continuation.*

Lorazepam is a member of the benzodiazepine family of anxiolytics, which increase fixation of GABA exclusively on the receptor GABA<sub>A</sub>. The inhibitory neurotransmitter GABA is widespread in the cortex occupying receptors at around 40% of all synapses. In the visual cortex interneurons connected by synapses using GABA receptors are considered an important inhibitory mechanism by which neural-response selectivity to moving stimuli (orientation and direction selectivity) is achieved. Additionally, and in contrast to the effects of other benzodiazepines, Lorazepam has been shown to influence perceptual organization affecting both perceptual integration and segmentation processes. In particular it has been shown that Lorazepam brings about disruption in target detection and object recognition performance while facilitating of visual segmentation (see Giersch, 1999 for a general overview). GABA<sub>A</sub>ergic neurons appear to function, and indeed promote postsynaptic mechanisms to function within relatively well-

defined temporal constraints. Evidence from studies of hippocampal neurons on slice preparation suggest that GABA<sub>A</sub>ergic neurons tend to synchronize at between 33 and 50 Hz, a pattern of activity associated with visual perceptual organization and more particularly the binding together of simple visual features. Following GABA<sub>A</sub>ergic synchronization, postsynaptic excitatory neurons also synchronize with increased amplitudes but reduced frequencies, which appear to shift from around 40 Hz to between 10 and 30 Hz. The increase in amplitudes and accompanying reductions in frequency normally associated with GABA<sub>A</sub>ergic synchronization may be one mechanism by which low level bindings compete for access to subsequent perceptual-attentional mechanisms. It seems likely that Lorazepam-enhanced GABA<sub>A</sub>ergic inhibition attenuates the frequency shift in excitatory neural activity, thereby inhibiting synchronization at lower frequencies with the result that no single low-level grouping emerges as a clear candidate for subsequent detection or recognition.

On the basis of Elliott and Müller's (1998) Experiment (see below) it was proposed that early and local neural activity might become primed at 40 Hz by virtue of this temporal code being fed back from later mechanisms that respond at 40 Hz to the premask matrix as a whole. In spite of slowed target detection latencies and independent of the effects of sedation Elliott, Becker, Boucart and Müller, (2000) found priming effects to be significantly increased in Lorazepam relative to both control and Diazepam conditions. Diazepam was chosen as a control for Lorazepam because while it produces the same sedative effects it appears to exert no specific influence on perceptual organization (Beckers, Wagemans, Boucart & Giersch, 2001) and while it lowers the firing frequency of individual neurons it appears to have little influence on the generation of synchrony. Although the results of Elliott et al. (2000) are generally consistent with the fact that Lorazepam has been shown to enhance segmentation processes evidence for a mechanism common to both temporal and spatial segmentation remains to be revealed.

Concerning spatial segmentation, it is likely that the influence of Lorazepam is indirect. The effects of the drug on form perception appear to depend on the low-level, preattentive coding of the organization of line terminations. Concerning temporal segmentation, the possibility that Lorazepam comes to influence synchrony priming directly, at a level of simple feature coding, appears to contradict the summary hypothesis of Elliott et al. (2000), which held prime formation as a consequence of the modulation of local, bottom-up driven 10-Hz responses in early visual areas by the global frequency of premask-frame presentation fed back from later visual processing areas. Given the apparent contradiction between the temporal and spatial segmentation effects of Lorazepam, we asked whether or not these apparently different effects may be related or whether they are independent and arise through the widespread effect of the drug on the central nervous system? We expected that if the synchronous presentation induces low-level completion, priming effects observed in Lorazepam-treated subjects should be modified when the spatial properties of the prime are manipulated. In contrast, if the effects of synchronous presentation are mainly due to temporal segregation between the frames, the effects of Lorazepam should remain stable as the spatial properties of the prime are manipulated. In order to decide upon a method for determining the influence of low-level, simple feature coding upon prime formation, two possibilities were considered: On the one hand the potential for completion between premask crosses might be enhanced by means of increasing the physical specification of the cross – cross continuance. The specific aim of the experiment described here was to determine the extent to which priming performance was influenced by contours presented orthogonally and at varying distances to the unspecified continuance between premask crosses (see Figure 1). It was expected that

orthogonal contours terminating on the virtual continuance between synchronous premask crosses would assist boundary completion, with a commensurate increase in the magnitude of priming effects indicating that the prime comes to be formed as a function of computing the spatial relations between the synchronous premask elements.

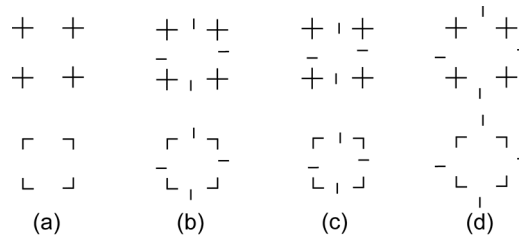


Figure 1: (a) no orthogonal contours are added to the unspecified continuance, (c) contours are placed directly, contours are placed overlapping (c) or in some distance (d) from the unspecified continuance.

## METHODS

12 paid volunteers (7 female), participated in the experimental study comprising three treatment sessions preceded by a practice session. Depending upon the session wise assignment, subjects were administered Lorazepam (0.038 mg/kg), Diazepam (0.3 mg/kg) or a placebo (lactose, 190 mg), using a double blind procedure. The time of administration was designed so that action peak of both drugs was achieved at the same time. Each experimental session consisted of 640 trials (40 trials per experimental condition), divided into sixteen 40-trial blocks separated a 5-second break. Each session had a duration time of approximately one hour. For each trial, subjects were presented with the 3 x 3 matrix of premask crosses, which flickered at 40 Hz for 600 ms. Upon termination, the premask matrix reduced to a target matrix of simple 90° corner junctions. Subjects had to discern at a distance of 57 cm (maintained via chin rest) the presence or absence of a Kanizsa-type square within the target matrix and produce a target-present/absent RT response as rapidly as possible. Fine scaled temporal resolution was achieved by presenting stimuli on a 6" Tektronix 608-oscilloscope monitor equipped with a very fast-decay P15 phosphor. The use of a P15 phosphor ensured that on-screen image persistence reduced to 10% of normal image intensity within 2.8 ms of image termination. The Interactive Electronic Systems point plotter buffer allowed pixels to be plotted at a rate of one pixel every microsecond.

Premask-matrix frame presentation frequency was fixed at 40 Hz, while the entire premask matrix, consisting of a sequence of four separate frames, was recycled at a rate of 10 repeats per second. Frames had constant exposure duration of 25 ms and an inter-frame interval of less than 1 ms. The continual recycling of the premask-frame sequence produced the phenomenal experience of a flickering matrix of 3 x 3 crosses. The display on a trial consisted of a premask matrix of crosses, composed of four repeatedly presented frames and presented for a brief period of time after which the premask crosses reduced to semi-static display of 90° corner junctions. On 50% of trials, the premask matrix included presentation of a figurally relevant 'synchronous premask' frame. The 'synchronous' premask was defined by four premask crosses (elements) presented in square arrangement at the premask-matrix location which could be subsequently occupied by the 4 target-matrix elements that defined a Kanizsa-type square target. Presentation of the synchronous premask was controlled for by presenting 4 elements in pseudo-random arrangement (that did not correspond to a square), henceforth referred to as the 'random premask condition'.

The matrix of corner junctions presented immediately after premask matrix presentation could, on 50% of trials, include presentation of a target Kanizsa-type square (Elliott & Müller, 1998, Exp. 2, see also Figure 2). Alternatively, the matrix of corner junctions could, on 50% of trials not include any combination of elements that grouped to form an illusory square (i.e. target-absent trials). In addition, on 75% of all trials the 4 premask-matrix frames were presented concurrent with a matrix of 12 contours. Using a within subjects repeated measures design, all subjects experienced each combination of the premask, target and contour condition on 40 occasions per experimental session.

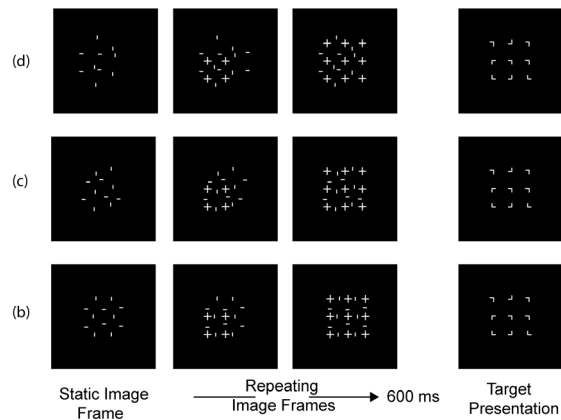


Figure 2: The cross matrix comprises four repeatedly presented premask frames each composed of a different spatial arrangement of 1 - 4 premask-matrix crosses. The sequence of premask frames was presented for 600 ms before presentation of a target matrix. Conditions (b), (c) and (d) correspond to the inducer conditions illustrated in Figure 1.

## RESULTS

Response errors accounted for 3.8% of all trials while trials that were allowed to time out without response accounted for .003% of all trials. Analysis failed to reveal evidence to suggest that the correct RT data may be contaminated by the outcome of a fast guess detection strategy (i.e. speed-accuracy trade offs). An omnibus ANOVA conducted on the mean correct RTs revealed a significant main effect of Target ( $F(1,11) = 35.78$ ,  $MSE = 58120.03$ ,  $p < .001$ ): Significantly faster RTs were found for target-present relative to the target-absent trials (mean RTs [and associated SE mean] in ms were 693 [27] vs. 813 [39] ms). A significant main effect of Synchrony was also revealed ( $F(1,11) = 12.35$ ,  $MSE = 1493.641$ ,  $p < .005$ ) based upon faster RTs following presentation of a synchronous premask relative to those following random-premask presentation (747 [32] vs. 758 [32] ms). A significant main effect of Treatment ( $F(2,22) = 21.95$ ,  $MSE = 132988.66$ ,  $p < .001$ ) was examined using Bonferroni-adjusted simple main effects analyses. These analyses revealed significant differences between Placebo relative to Diazepam ( $p < .001$ ; mean difference [and SE mean] was 177 [31] ms) and Placebo relative to Lorazepam treatment RTs ( $p < .001$ ; 237 [38] ms.). No significant differences were found between the Diazepam and Lorazepam treatment conditions ( $p > .17$ ; 60 [42] ms). A significant Treatment x Target interaction ( $F(2,22) = 10.05$ ,  $MSE = 20308.04$ ,  $p < 0,001$ ) was examined by means of Bonferroni-adjusted simple main effects analyses. Consistent with previous research a

significant Target x Synchrony interaction revealed priming to be specific to target presentation ( $F(1,11) = 40.08$ ,  $MSe = 861.68$ ,  $p < .001$ , the mean random-synchronous differences [and SE mean] were: 27 [4] ms and 4 [4] ms for the target present and absent trials, respectively). Of particular interest was the significant four-way interaction ( $F(3.32, 54.12) = 3.00$ ,  $MSe = 2723.54$ ,  $p < .05$ ) which emerged as a function of a very substantial (73 ms), target-specific synchrony-priming effect consequent upon the orthogonal contours terminating adjacent to the virtual continuance but only following Lorazepam administration (see Figure 3. The mean RTs [and 95% confidence intervals lower - upper bounds] were 732 [668 – 797] ms and 805 [734 – 877] ms for the synchronous and random RTs respectively. Note that, ordinarily and without either drug administration or the addition of orthogonal contours, comparable premask conditions produce synchrony priming effects of ~25 ms).

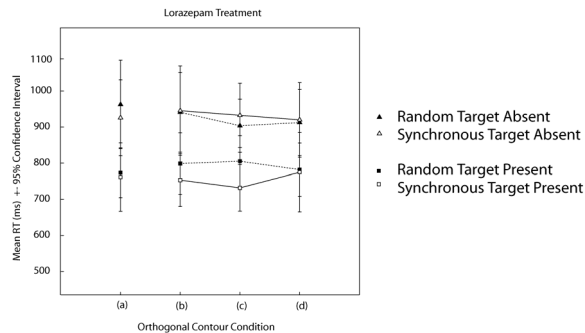


Figure 3 shows that, following Lorazepam administration there is an increased synchrony-priming effect on the target detection RTs when the orthogonal contours terminate directly on the unspecified continuance (condition (c)).

## DISCUSSION

The current study examined two contradictory hypotheses concerned with the effects of the benzodiazepine Lorazepam upon the coding of contours presented orthogonal to, but at varying distances from the unspecified continuance between the collinearly arranged premask crosses. Our predictions were that if synchrony-priming effects related to boundary completion an increase in the magnitude of priming effects would be expected to accompany presentation of orthogonal contours terminating on the virtual continuance between synchronous premask crosses. In addition, we expected enhanced priming effects to be of greatest magnitude when subjects had been administered with Lorazepam relative both to placebo and Diazepam treatment. Conversely, if priming were related to temporal but not to spatial segmentation we should expect priming to be enhanced following Lorazepam administration but to be invariant with variations in the termination of lines placed orthogonal to the unspecified continuance between the premask crosses. Evaluation of these hypotheses was undertaken by means of different drug administrations in combination with experimentation employing a variant of the premask paradigm introduced by Elliott and Müller (1998). Three findings were revealed: Firstly, the presence of synchrony-priming effects following placebo administration was revealed consistent with previously reported synchrony-priming effects in healthy volunteers. Secondly, subjects treated with Lorazepam were slower to detect targets than when treated with the placebo. This outcome is consistent with the idea that Lorazepam enhanced GABA<sub>A</sub> inhibition

facilitates spatial segmentation as a consequence of enhanced signaling of line terminations. Facilitation of spatial segmentation in this fashion is argued to have the contrary effect of disrupting perceptual integration and may come to inhibit detection of visual groupings. Thirdly, priming effects were substantially increased following Lorazepam administration when orthogonal contours were positioned such that they terminated adjacent to the unspecified continuance between the premask crosses. This result was confirmed statistically both by planned comparisons (between the synchronous and random target RTs) and, more importantly as a result of a more conservative and detailed breakdown of the significant 4- way interaction. From the latter analysis it was shown that the significant 4-way interaction was obtained as a function of significant (target-specific) priming effects following Lorazepam administration and consequent upon orthogonal contours positioned such that they terminated adjacent to the unspecified cross-cross continuance. The significance of target priming effects under this combination of treatment and contour conditions contrasts with (target-specific) random-synchronous comparisons under other treatment x contour combinations, which failed to achieve significance. These results offer strong evidence to consider the temporal segmentation of the synchronous premask from the premask matrix to occur relative to the coding of spatial continuity between the synchronous premask crosses. Moreover, given that this effect was not evident for the other contour conditions following Lorazepam administration, these results support the prediction that the synchrony priming effects relate to the coding of completions across the unspecified continuances between the premask crosses, while rejecting the hypothesis that priming effects relate solely to the temporal segmentation of the synchronous premask frame from the remainder of the premask matrix.

These results offer further evidence that Lorazepam enhanced GABA<sub>A</sub>-induced inhibition is important for the coding of spatially separate visual stimuli, these results show that Lorazepam-enhanced GABA<sub>A</sub> inhibition has a direct facilitatory influence upon the coding of stimulus-element relations by means of the generation of a 'virtual' contour between collinearly arranged cross elements. Given that line terminations are supposed to be coded by end-stopped cells, these results offer support for the idea that that Lorazepam has an indirect influence upon visual grouping by means of a direct enhancement of end-stopped, simple cell responses. The basis of our account is the suggestion that early visual-cortical mechanisms related to the signaling of line terminations are responsible for the coding of a virtual contour across the unspecified continuance between the premask crosses.

## REFERENCES

- Beckers, T., Wagemans, J., Boucart, M., & Giersch, A., (2001). Different effects of lorazepam and diazepam on perceptual integration. *Vision Research*, **41**, 2297-2303.
- Elliott, M. A. & Müller, H. J. (1998). Synchronous information presented in 40-Hz flicker enhances visual feature binding. *Psychological Science*, **9**, 277-283.
- Elliott, M. A., Becker, C., Boucart, M., & Müller, H. J. (2000). Enhanced GABA<sub>A</sub> inhibition enhances synchrony coding in human perception. *Neuroreport*, **11**, 3403-3407.
- Giersch A. (1999). A new Pharmacological tool to investigate integration processes. *Visual Cognition*, **6**, 267-297.