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Enhancement of the Lead-Stimulus Inhibition Induced by Key-Pressing to S_1

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Abstract

The purpose of this study was to determine whether the lead-stimulus inhibition would be affected by the RT task in which subjects were required to respond to the lead-stimulus. Fourteen undergraduates were assigned into one of the following two groups. In the Group-T, subjects were required to press the key as they heared the lead-stimulus, tone of 70dB-, 1000Hz. In the Group-NT, subjects were required nothing. The results showed the mean eyeblink amplitude elicited by a 110-dB noise burst in the Group-T was significantly smaller at the SOA of 125-250msec than in Group-NT. These results can be interpreted by the limitted capacity model, as same as the phenomenon of psychological refractory period.

DESCRIPTORS: Lead-stimulation effects, Lead-stimulus inhibition, Prepulse inhibition, Starle reflex, Eyeblink reflex, RT task.

Introduction

The eyeblink reflex elicited by a strong noise burst (S_2) is inhibited if the S_2 is preceded by an extraneous lead-stimulus (S_1) with an SOA (Stimulus onset asynchrony) of around 100 msec. On the contrary, the effect turns into facilitatory if the SOA is prolonged beyond 400 msec or more with a variable SOA schedule (Graham. Putnam, & Leavitt, 1975: Yamada & Miyata, 1979).

The purpose of the present experiment was to determine whether these lead-stimulation effects would be enhanced if the task in which the reaction with the key to the lead stimulus was required. A theoretical concern behind this experiment was whether the occurrence of inhibition depends on some sort of the limitted capacity of the human acoustical information processing mechanism. As suggested by Braff, Stone, Callaway, Geyer, Glick and Bali (1978), the lead-stimulus inhibition would be the product of a certain processing to S_1 in the S_1-S_2 stimulus configurations. But in their notions, the contents of the processing was ambiguous in the general procedure of the lead-stimulus experiment. If the key-press task in which subjects were required to respond to S_1 as soon as possible, S_1 must be processed by the subject. In this experiment, we tried to examine the role of processing of S_1 in producing the lead-stimulus inhibition.

If the lead-stimulus inhibition would be caused from the subject's processing of S_1, the eyeblink reflex amplitude in the key-press group would be more clearly inhibited than that in a non-tasked control group.

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Method

Subjects

Fourteen undergraduates, seven males and seven females, served as subjects. Their mean age was 21.0 with a range of 19-22.

Apparatus

The testing room was an air-conditioned, sound-attenuating chamber of 215 × 135 × 177cm. The subjects, seated in a comfortable chair, received S_1 and S_2 binaurally through Pioneer SE-205 headphones. The S_1 was a 1000-Hz pure tone, generated by a Torio audio-generator AG-202A, and continued for 50mesc. The S_2 was a 50-msec burst of white noise, which was continuously recorded on a tape and fed into the audio-amplifier. Both stimuli were gated on and off with rise and fall times less than 1 msec, controlled by National reed relay NR-H-5V. The intensity level was calibrated on the C scale of the Rion NA-07 sound level meter at the headphones. Monaural intensity of the S_1 and S_2 were 70dB and 110dB, respectively. Stimulus presentation and timing were controlled by 6-channel digital preset timers made of IC circuits and paper tape reader.

Eyeblink reflexes were measured by an electrode hookup method recommended by our previous researches (Yamada, Yamasaki, & Miyata, 1979; Yamada, Yamasaki, Nakayama, & Miyata, 1980). That is, the vertical EOG was picked up from the left side of the subjects' face with the Nihon Koden Sintered Ag-AgCl miniature skin electrode, NT-212U. The positive electrode was placed just above the eyebrow; the negative one just below the cheekbone, beside the nose; the ground one on the fleshy part of the cheek. Attachment of the electrode was made with Nihon Koden electrode paste and collars. The electrodes were coupled to a San'ei Bio DC-amplifier Model 1117 and recorded by a San'ei pen-galvanometer (Rectigraph 8S) with a paper speed of 50mm/sec. The gain was arranged to give 40mm of trace deflection for 1mV.

Procedure

After resting about five minutes in the dark room, the subjects received the startle noises twice, and they were assigned to one of the following two groups, according to the blink amplitide measured in the pre-treatment. The mean blink amplitude for each group did not significantly differ ($F<1.0$). In the non-tasked group (named Group-NT), they were instructed to remain awake and to fixate a green light emitting diode (LED) which was one meter ahead them. In the task induced group (named Group-T), an additional instruction was given in which they were required to press the key by the dominant thum as fast as possible when they heard the tone (S_1).

After the three-minutes re-rest period, the subject received seven blocks of trials. In each block, there were one S_2-alone control and seven S_1-S_2 trials. In the S_1-S_2 trials, there were seven SOA conditions, i.e., 60-, 125-, 250-, 500-, 1000-, 2000-, and 4000-msec. The order of the SOA conditions were randomized. The inter-trial interval (ITI) varied randomly between 25 and 45 sec by 5-sec steps.

Analysis of responses

The eyeblink reflex to the startle stimulus was identified with the latency criterion, in which only the reflexes beginning in a window 20-120 msec after the startle stimulus onset were picked up. The peak amplitude was measured in micro volts and then transformed into a logarithmic scale as recommended by our previous reports (Yamada et al., 1979; 1980). If the lid was partially closed at the time of the S_2 onset, the trial was excluded. The mean amplitude across the trials was calculated for each subject. For the Group-T, the reaction time (RT), the time delay from the onset of the S_1 to the key-press, was measured in a msec scale with a digital counter.

Results and Discussion

The initial analysis was conduced to determine if the differential lead-stimulation effect has occured in the two groups. The ANOVA with Groups (T vs NT) and SOAs as the factors yielded a significant SOA main effect ($F_{(7,84)}=15.47, p<.001$), and a significant Group by SOA interaction ($F_{(7,84)}=5.99, p<.001$). Figure 1 shows these results; as can be seen, the interaction is due to the enhancement of inhibition effect at the SOAs of 125- and 250-msec in Group-T.

The second analysis was conduced to determine the lead-stimulation effects for each group with error terms obtained from the previous ANOVA, using a significance level of .05 for all comparisons.

In the Group-NT, both inhibitory and facilitatory effects were significant, i.e., the amplitude of the eyeblink reflex was significantly inhibited under the SOAs of 60- and 125-msec., and facilitated under SOAs of 2000- and 4000-msec.

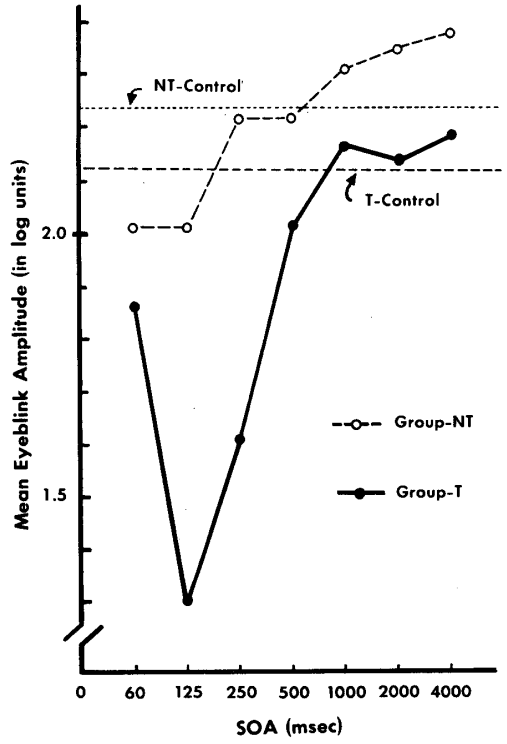

Fig. 1. The mean eyeblink amplitude for each group as a function of SOA (stimulus onset asynchrony).

On the other hand, Group-T showed only inhibitory effect, i.e., eyeblink amplitude was inhibited under shorter SOAs; 60-, 125-, and 250-msec SAO conditions. From these analysis, lead-stimulus inhibition was found in both groups but the lead-stimulus facilitation was found only in the Group-NT.

The last analysis was conduced to determine whether RT performance would be modified by the S_2 with the different intervals followed S_1, in the Group-T. The mean RTs varied around 360-390 msec but did not significantly differ among SOA conditions ($F<1.0$).

The results of this experiment showed that the lead-stimulus inhibition was enhanced and the facilitation disappeared if the information processing, key-pressing to the lead-stimulus, was demanded. This indicated that the inhibitory effect is produced by the processing of lead-stimulus (S_1), as expected initially. But the conclusion must be made after considering following problems.

First, the significant difference between two groups under short SOA conditions might be caused from the different arousal states during the experimental session. The arousal state in Group-T seems to be higher that that in Group-NT, because the key-pressing task was required in Group-T, but not in Group-NT. The theory of arousal insists that the reflex activity changes with an inverted-U function of arousal state (Ison & Hammond, 1971). If this arousal interpretation is valid, the arousal state of the subjects in Group-T would be extremely high on the arousal continum, because the overall blink amplitude is smaller than in Group-NT, although the difference was not significant. Though the enhancement of the inhibitory effect might be caused from the higher state of arousal, we do not have any interpretation to these phenomena by means of arousal.

Second interpretation refers to the muscle interference. The muscle reaction might interfere with the reflex activity in Group-T. In this group, the skeletal motor reaction initiated about

300mesc after the onset of S_1 could disturb the initiation of the reflex blink. Further experiments must be designed to examine these possibilities.

In any case, it is clear that the key-press task enhances the lead-stimulus inhibition at the SOA of around 125-250msec, where the key-press decision thought to be initiated. We regard this finding as the evidence supporting that the lead-stimulus inhibition is caused by the same or similar mechanism of information processing as that of the 'psychological refractory period effects', hypothesized by many authors (for example Smith, 1967). In the framework of information processing, we suppose that the inhibitory effect is produced as following manner.

At the SOAs of around 100-200 msec, reflex elicitation is interrupted by the processing of S_1, since the reflex eliciting stimulus has come into the startle center until the processing of S_1 does not terminated. Although the content of processing of S_1 is not identified so far, it might be some sort of the automatic detection of environmental changes. It may be the orienting reflex (OR) conceptualized by Sokolov (1963). As hypothesized by Stitt. Hoffman, and Marsh (1973), OR and startle reflexes share essential neural components, such an interpretation might be possible to approve.

Concerning with the facilitatory effect, additional evidence supporting Graham's hypotheses was obtained. According to Graham (1975), the reflex facilitation would depend on the 'physiological reboud' concomitant the termination of 'orienting-attentional process', which was initiated by S_1 onset in the temporal or event uncertain situation. In this experiment the orienting-attentional process was terminated before the onset of S_2 in the key-press group at the SOA of 2000- and 4000-msec, but not terminated in the Group-NT. Result showed that the facilitation disappeared in the Group-T as expected by Graham's rebound hypothesis.

On the analysis of this experiment, we would conclude that the lead-stimulus inhibition and facilitation were both related to the orienting mechanism in some part. Further researches concerning with OR and the lead-stimulation effects are needed.

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先行刺激に対するキー押し反応課題の付加による先行刺激抑制効果の強調

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邦文要約

110-dBの強音に対して誘発される驚愕性瞬目反射は，誘発刺激の開始に約100 m sec 先行して微弱な信号を付加することによって抑制される。これを先行刺激抑制効果と呼ぶ。さらに，先行刺激(S_1)と反射誘発刺激(S_2)の時間間隔(SOA)を1-2 sec と延長すると反射は促進する。先行刺激抑制効果は，S_1に対する受動的注意に伴う情報処理過程が驚愕反射の誘発機構に干渉することによって出現すると仮定できる。一方先行刺激促進効果は，S_2に対する能動的注意機構が誘発刺激の入力を促進するために出現すると仮定できる。

本研究では，先行刺激抑制効果の発現に関する上記の仮説を検討する事を目的として，S_1に対するキー押し反応の付加が先行刺激効果に及ぼす影響を検討した。14名の大学生被験者を，反射量でマッチングしたうえでT群とNT群とに無作為に振り分けた。T群の被験者には，S_1に対して利き手親指による迅速なキー押し反応を要求し，NT群では何等課題を課さなかった。S_1は70 dB，1000Hzの純音，S_2は110 dB の白色雑音であった。両刺激とも持続時間は50 msec であり，ヘッドフォンを介して両耳に呈示した。S_1とS_2とのSOAは60，125，250，500，1000，2000，4000 msec の7種で，これらのS_1-S_2条件にS_2単独呈示条件を加えた計8つの刺激条件を1ブロックとして7ブロック，計56試行呈示した。刺激条件の呈示順序は無作為とし，試行間間隔は25-45sec (平均35 sec)の無作為系列に従った。

平均瞬目反射量の結果は，(1)両群とも先行刺激抑制効果が有意であること，(2)125-250 msec のSOA条件ではT群の抑制効果が有意に強調されること，及び(3)T群の先行刺激促進効果が消失することを示し，上記仮説を支持した。

鍼刺激による痛み抑制効果と本先行刺激効果との関連は今後検討されねばならないであろう。