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Tohoku Psychologica Folia
1984, **43** (1-4), 8-22

PHASIC HEART RATE CHANGE JUST BEFORE SHOCK PRESENTATION AND ITS POSITIVE RELATION TO PAIN

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The phasic heart rate (HR) changes in a foreperiod were investigated in nine male subjects for two days in an aversive situation with unavoidable electric stimulation. The experiment consisted of the shock trial (3. 6, and 9 or 8 mA) and the no-shock trial, either of which was informed to the subject (*S*) by a LED in front of him as soon as the trial started. Simultaneously the *S* was required either to keep his muscle tension of right forearm to be high (H-EMG condition) by pulling on an expander or to keep his tension of it to be relaxed (L-EMG condition). The results indicated that there occurred significant HR deceleration just before the shock presentation only in the L-EMG on Day 1, and on Day 2 besides this condition, in the H-EMG too, characterized by the increased metabolic activity. This result in the H-EMG seemed to support the existence of the central nervous function named "somatic-cardiac inhibitory effects" by Obrist (e.g. 1968). The magnitude of this deceleration, however, positively correlated with the magnitude of phasic acceleration immediately after the shock and with the values of pain estimation, in the higher intensity of shocks (6 and/or 9 mA) in both EMG conditions. This result disagreed with the Obrist's insistence (e.g. 1969) on the anticipatory deceleration connected with the attenuation of pain. Therefore, the somatic-cardiac inhibitory process was considered to represent a part of the "attention processes". The relationship among the magnitudes of anticipatory deceleration, of pain sensation and the degree of attention to a stimulus were discussed.

Key words: heart rate (HR), foreperiod, pain, shock, somatic activity, P.A.Obrist

There have been many studies which investigated phasic heart rate (HR) changes in a foreperiod with two-stimulus paradigm. The first stimulus was the warning stimulus (WS) and the second was either a non-aversive imperative stimulus (IS)

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demanding some reactive responses which was in the non-aversive situation (Chase et al ., 1968 ; Higgins, 1971 ; Hatayama et al ., 1981, 1982), or was usually an unavoidable aversive stimulus which was in the aversive situation (Obrist et al ., 1965 ; Obrist 1968 ; Obrist et al ., 1969 ; Niemalä, 1969 ; Jennings et al ., 1971).

In both situations, there was found a similar HR changing pattern in the foreperiod, that is, which consisted of initial small deceleration, successive acceleration and following deceleration immediately before the IS. The initial deceleration component did not always appear and was generally regarded as an orienting response (OR). Somsen et al . (1983) recently indicated that the HR changing pattern in the foreperiod except the initial deceleration was differentially affected by the various tasks which were systematically ranged from a disjunctive reaction time task in non-aversive situation to an unavoidable shock task in aversive situation. They reported that in the non-aversive condition there appeared apparent large initial acceleration followed by large deceleration, and in the unavoidable shock condition the pattern consisted mainly of large and sustained deceleration with superimposed small initial acceleration. These two components of this HR change were analyzed in some reports in terms of the functional or behavioral significance on the basis of the organism-environmental interactions.

In non-aversive situation, the acceleration component was considered not a defence or startle reflex but a reflection of an attention process (Higgins, 1971 ; Hatayama et al ., 1981) and was sometimes related to the efficiency of motor performance (e.g. Higgins, 1971). The second deceleration component was considered that it reflected an anticipation of a given IS, an enhanced sensitivity to the environmental inputs and a preparation for the response to be done, which processes were considered to be potentely related to an attention process. Lacey (e.g. Lacey & Lacey, 1974, 1978) viewed this component as a part of an attention process which facilitated the "sensory-intake" and the sensory-motor integration in the view points of visceral feedback afferentation.

In aversive situation, the acceleration component was often small, which some researchers considered to be related to an attention process (Niemelä, 1969 ; Jennings et al ., 1971), but others regarded as a respiratory artifact (Orbist et al ., 1965). The deceleration component was considered to be positively related to an attention process in non-aversive situation, too (Niemelä, 1969 ; Jennings et al ., 1971). But Obrist and his colleagues emphasized the "somatic-cardiac inhibitory" process rather than the attention process, especially in the aversive situation. Their model presented contrast to the Lacey's model in the non-aversive situation. Obrist stated that this component was previously conditioned response mediated by the anticipation, and had a large concomitance with a cessation of spontaneous somatic activities (e.g. Hastings & Obrist, 1967 ; Obrist, 1968). They hypothesized that this cardiac deceleration as well as decreases in somatic activity just before the shock was viewed as a peripheral manifestations of the inhibitory effects by the central inhibitory process.

If this central inhibitory process exists and have strong effects on cardiac activity, the deceleration component would appear just before the shock, although a somatic activity to a certain extent might be experimentally imposed. Concerning this point, Obrist et al. (1969) themselves imposed the task for the *S* to execute a gentle and continuous tapping in an aversive condition. They found deceleration component as predicted. But the experimentally imposed tapping task was regarded to be gentle and low in the viewpoint of the metabolic demands. If experimentally imposed somatic activity needs much more metabolic demands than the tapping task, will the "somatic-cardiac inhibitory effects" be still strong to overcome the HR acceleration supposed by the highly metabolic demands? In order to investigate this problem, we contrived to make the *S* continually pull on an expander, because this task was thought to demand much metabolic activity.

Obrist et al. (1968, 1969) presented one possible significance of this central inhibitory process to have relevance to coping responses with a pain. They suggested that the unavoidable effects might be the response to an unavoidable aversive situation, of which functions were to minimize the "stimulus-intake" and thereby to attenuate the noxiousness of an aversive stimulus. This deceleration component was considered, however, in non-aversive condition above mentioned, to reflect the attention process which enhanced the sensory-intake and facilitated sensory-motor integration. If this hypothesis in non-aversive situation can be applied to aversive situation, Obrist's inhibitory process is considered to be involved in the attention processes. Even Obrist referred to this inference as the "another possibility" of inhibitory mechanism (Obrist, 1968, p. 192). If this "another possibility" was true, it was inferred that the magnitude of the deceleration was positively related to the estimate of an aversive stimulus.

In sum, the purposes of this experiment were as follows;

(1) To examine whether the HR deceleration would appear immediately before the shock presentation in unavoidable shock condition or not, though the somatic activity such as a continuous expander-pulling was experimentally imposed, of which activity demanded much more metabolic activity than the tapping task used by Obrist et al. (1969).

(2) To examine whether the inhibitory process insisted by Obrist works to attenuate the noxiousnesss of electric shock or not, by investigating the relation between both the first acceleration and, in paticularly, the second deceleration components and the two pain responses described later.

Method

Subjects: Subjects (*S*s) were 9 male undergraduate students at the Tohoku University, ranging in age from 19 to 22. None of them reported any history of cardiovascular abnormality.

Apparatus: The instrumentation used in this experiment was the same as that of the previous study (Yamaguchi et al., 1982). Electromyogram (EMG) was recorded through electrodes placed above flexor capri radialis and flexor digitorum sublimis muscles of right forearm. Electrocardiogram (ECG) was recorded across the chest, and heart rate (HR) was instantaneously calculated from successive R-R wave intervals at the sampling time of 5 msec. Respiration was recorded from respiration strain gauge of rubber tube wound a belt around the abdomen.

Electrical stimulation (3, 6, and 9 or 8 mA) was delivered in a constant current (square-wave, 750 msec duration) through a concentric electrode recommended by Tursky et al. (1965). Only one subject was delivered 8 mA of shock intensity as substitute for 9 mA. The shock electrode was placed on the antebrachial-anterior surface pretreated between the two electrodes for EMG recording of right forearm. Electric stimulus was given on the shock trial (SH), but not given on the no-shock trial (NSH). At the time when each trial started, subject was informed whether it was the SH trial or the NSH one, by either of the two light emitting diodes (LEDs) lighted up (red or green) till the end of trial on the board in front of him. The colors of LEDs were counterbalanced through subjects.

S was required to keep the right-forearm muscle tention in a range of 260 μV to 610 μV in a high EMG condition (H-EMG), by pulling horizontally on an expander one side of which was fixed to a table in front of him and by using binary feedback signal (1 kHz, 59 dB) on the basis of his right-forearm muscle tention. But in a low EMG condition (L-EMG), *S* was required to keep the muscle tension relaxed by nothing to do (to be exactly in a range of 0 to 130 μV). The feedback signal was sounded when muscle tension level deviated from each the referred range concerning the EMG condition. These tasks of keeping the EMG level constant were very easy for all subjects after some training trials before the experiment.

Procedure: The experiment was repeated for two days almost in the same procedure. In a pre-experimental session, *S*s experienced all kinds of electric shocks in the same manner as in an experimental session. After the recording of the resting baseline of HR (5 min), the experimental session began, which consisted of 24 trials or more. *S*s were required to report through interphone the magnitude estimation of pain in the range from 0 (nothing to perceive) to 100 (pain tolerance) immediately after the end of every trial.

Presentation of electric stimulus: After each trial started, the computer waited subject's HRs being kept in a relatively low and constant range, using the same procedure as that in our previous report (Yamaguchi et al. 1982). After subject's HR level satisfied this range-condtion, the computer checked whether feedback tone was sounded or not. If the tone was not spontaneously sounded, the experimenter made subject sound it by the instruction through interphone to gain more strength of right forearm or be more relaxed, during short time. Actually, the spontaneously sounded tone was rare because the task of keeping EMG output constant was very easy. After

feedback tone sounded, which was identified with the warning stimulus (WS), R waves in ECG were counted by 8 beats in condition of keeping muscle tension within the referred range. If the muscle tension unexpectedly deviated from the referred range, that is, if feedback tone again sounded, the WS was reidentified with the latter feedback tone, and the counting of R waves recommenced at that time. This case seldom happened, but when it happened, the experimenter promptly instructed through interphone to sound feedback tone again in order to get subject to confirm this "intentional" tone as WS. After the completion of counting R waves by 8 beats, electric shock was triggered by the first R wave within the predetermined respiration cycle. After shock presentation, R waves were counted by 18 beats.

In this experiment the foreperiod was defined as the period from WS to the shock presentation, and the pre-foreperiod was defined before WS. The after-shock period consisted of 10 cardiac cycles after the shock. In the pre-foreperiod, the last 10 HRs were taken to calculate the mean HR just before the foreperiod (i.e. hereinafter referred to as initial level of HR).

The HR data analysis was done in the difference scores of HR from the initial level of HR for only 9 HRs in the foreperiod and only 10 HRs in the after-shock period. All statistical analysis was done for each subject's data in each condition, but not for each trial's data in each condition if not any provisoes. The condition of the respiratory cycle (inspiration or expiration) on the shock was combined with each other in the data analysis. The experimental conditions to be analyzed consisted of (1) SH or NSH condition, (2) L-EMG or H-EMG condition, and (3) First day (Day 1) or Second day (Day 2) condition.

Results

Initial level of HR in the pre-foreperiod: Table 1 showed the values of initial level of HR in each condition. On Day 1 there were two significant differences between the NSH and the SH in the L-EMG condition and between the L-EMG and

Table 1. Initial level of heart rate (bpm) in the pre-foreperiod. These values were the mean heart rates calculated from the last 10 heart rates in the pre-foreperiod. The *SD* was indicated in the parenthesis.

		Low EMG	High EMG	t score
DAY 1	No-shock	60.7(6.6)	61.8(7.8)	1.254
	Shock	62.1(6.3)	63.4(6.5)	2.630*
	t score	2.770*	1.719	
DAY 2	No-shock	65.6(7.8)	65.0(6.4)	0.682
	Shock	66.2(7.4)	66.2(6.7)	0.014
	t score	1.186	1.894	

*; $p<0.05$, $df=8$

Table 2. Mean length of the foreperiod by the time of duration (msec) and the number of heart beats in the foreperiod.

	Length of fore period.	Low-EMG		High-EMG	
		Duration	Beats	Duration	Beats
DAY 1	No-Shock	9931 msec (1461)	10.0 (0.7)	9718 msec (1578)	10.0 (0.8)
	Shock	11241 (3319)	11.2 (3.3)	10239 (1626)	10.6 (1.4)
DAY 2	No-Shock	9056 (1277)	9.8 (0.7)	9452 (1450)	10.3 (1.0)
	Shock	10142 (2261)	10.7 (1.9)	9851 (1444)	10.6 (2.1)

( ); *SD*

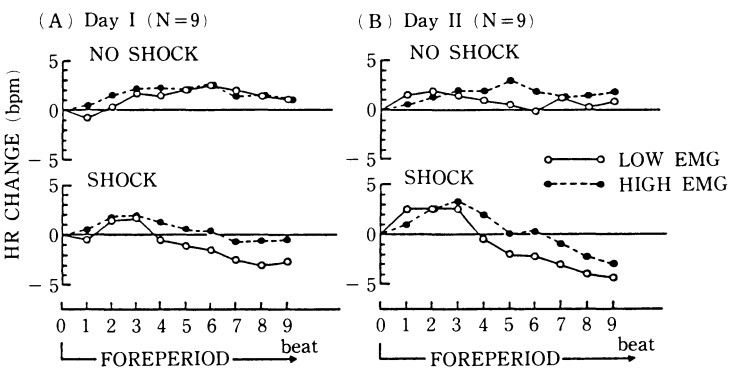

Fig. 1. Heart rate changing pattern in the foreperiod on Day 1 in (A) and on Day 2 in (B), which were represented by the difference scores from the initial level of heart rate.

the H-EMG in the SH condition. The higher initial level of HR might affect the magnitude of the HR changing pattern in the foreperiod. This implication was described in the part of the *HR changing pattern in the foreperiod* on Day1. On Day 2, there were no significant differences among conditions.

Mean length of foreperiod: Table 2 showed the mean duration (msec) and the mean heart beats during the foreperiod in each condition. The *t*-test indicated no significant differences among conditions.

HR changing pattern in the foreperiod: HR changes in the foreperiod were represented by difference scores (bpm) from the initial level of HR. These changes in 9 HRs during the foreperiod were presented in Fig. 1-(A) and -(B) for Day 1 and Day 2 respectively. The HR changing patterns during the foreperiod in the NSH did not contain a large deceleration component below the initial level of HR in both EMG conditions on both days. But, in the SH condition, except in the H-EMG on Day 1, there were the initial acceleration and the following deceleration components.

The HR changing patterns in the SH condition were further examined by two analyses each Day. At first, these patterns on Day 1 were evaluated by the one-way trend analyses, respectively in the both EMG conditions. In the L-EMG, the linear component had a significant F value of 30.83 ($df=(1,72)$, $p<.01$), and the cubic component added significantly predictability to that given by the linear trend ($F=6.21$, $df=(1,72)$, $p<.05$). This analysis indicated that HR changing pattern in the L-EMG primarily had a consistent deceleration, and secondarily might have an initial acceleration component. In the H-EMG, the F values of linear, quadratic, and cubic components were as follows: $F=6.77$, $p<.05$, $F=5.15$, $p<.05$, and $F=5.58$, $p<.05$, respectively, and all with $df=(1,72)$. These similar F values indicated that one notable component was not decided.

Further, in order to evaluate these patterns in the SH, compaired with them in the NSH condition, three HR scores (bpm) in the foreperiod were devised which scores were AF (the magnitude of initial acceleration in the foreperiod), D1 (the magnitude of type-1 deceleration just before the shock), and D2 (the magnitude of type-2 deceleration just before the shock), which were illustrated in Fig 2. The AF score was defined as the difference from the initial level of HR to the maximum HR (HR-max) in the foreperiod except the last two HRs (8th and 9th HRs). The D1 score was defined as the difference between the HR-max and the minimum HR (HR-min) within the last two HRs in the foreperiod. The D2 score was defined as the difference from the HR-min to the initial level of HR. The HR changes during the foreperiod in the SH were analysed on Day 1 in terms of these three HR scores (as shown in Table 3). For the AF score, there were not any significant differences among conditions. For the D1 score, there was a significant difference between the SH and the NSH in the L-EMG condition. The other combinations did not indicate any significant differences. For the D2 score, there were two significant differences between the SH and the NSH in the L-EMG, and between the L-EMG and the H-EMG in the SH. These D1 and D2 scores confirmed the existance of the deceleration component just before the shock in the L-EMG, as well as the trend analyses showed, but not in the H-EMG. The initial acceleration component was still equivocal in both EMG conditions in the SH in terms

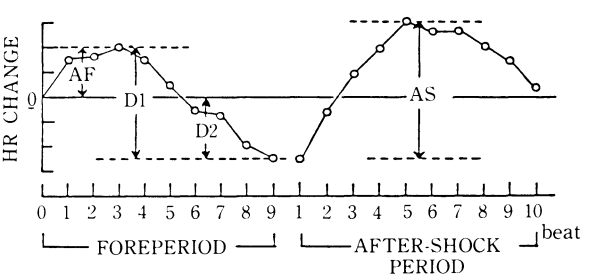

Fig. 2. Definition of three heart rate scores in the foreperiod and the AS score in the after-shock period. The AS score was one of the two pain scores which involved the magnitude estimate of the shock (E score) as another pain score.

Table 3. Values of the three heart rate scores in the foreperiod in each condition. The *SD* was indicated in the parenthesis.

〈AF score〉

		Low EMG	High EMG	*t* score
DAY 1	No-shock	4.0(2.7)	3.8(2.3)	0.188
	Shock	2.4(1.9)	3.2(2.7)	0.974
	t score	1.457	0.583	
DAY 2	No-shock	4.0(2.9)	3.9(3.3)	0.059
	Shock	3.8(2.4)	4.1(3.1)	0.259
	t socre	0.168	0.200	

〈D1 score〉

DAY 1	No- shock	3.3(2.1)	3.2(2.1)	0.174
	Shock	6.2(3.6)	4.6(2.1)	1.611
	t score	2.433*	1.277	
DAY 2	No-shock	4.5(5.4)	3.0(2.3)	0.932
	Shock	8.5(3.0)	7.2(3.9)	0.883
	t score	2.458*	3.167*	

〈D2 score〉

DAY 1	No-shock	−0.7(1.9)	−0.7(2.2)	0.043
	Shock	3.7(3.4)	1.4(2.5)	3.316*
	t score	3.197*	1.912+	
DAY 2	No-shock	0.5(4.1)	−0.9(3.1)	1.044
	Shock	4.7(2.3)	3.0(2.2)	1.558
	t score	3.168*	3.279*	

+ ; $p<0.1$, * ; $p<0.05$, $df=8$

of the AF score. There was the possibility in the SH with the L-EMG, however, that the D1 and D2 scores might be underestimated than the observed values, because the initial level of HR in this condition was significantly higher than in the NSH with the L-EMG condition (but only the mean-difference of 1.3 bpm ($SD=1.36$) between two conditions).

The HR changes in the foreperiod on Day 2 were presented in Fig . 1-(B). In the SH condition the one-way trend analysis was done in the same way as that in Day 1. In the L-EMG conditions the F values of linear, quadratic, and cubic components were as follows: $F=108.93$, $P<.01$, $F=6.54$, $p<.05$, and $F=19.05$, $p<.01$, respectively, and all with $df=(1,72)$. In H-EMG condition the F values of linear, quadratic, and cubic components were as follows: $F=30.26$, $p<.01$, $F=20.91$, $p<.01$, and $F=5.19$, $p<.05$, respectively. These values indicated that the HR deceleration component was primarily found notable in the foreperiod, and the initial acceleration component was

Table 4. Mean magnitude estimates of the electric shocks which intensities were 3, 6, and 9 (or 8) mA. These estimates were in the range from 0 (nothing to perceive) to 100 (pain tolerance).

		Low EMG	High EMG
DAY 1	3 mA	20.7(16.1)	27.0(17.4)
	6 mA	47.1(19.1)	45.3(18.8)
	9(8) mA	61.6(16.0)	67.5(15.8)
DAY 2	3 mA	20.4(17.6)	18.7(14.7)
	6 mA	47.8(12.2)	48.2(11.9)
	9(8) mA	65.4(7.8)	64.4(11.4)

( ); *SD*

secondarily found. Moreover, these HR changes on Day 2 were analysed in the same manner with three HR scores previously explained (as shown in the Table 3). For the AF score, there were not any significant differences among conditions. For the D1 and D2 scores, there were each two significant differences between the SH and the NSH in the L-EMG, as well as in the H-EMG. There were not any significant differences between two EMG conditions in the SH or in the NSH for the D1 and D2 scores.

The analyses by these HR scores and the trend analysis indicated that in the SH condition the pronounced HR deceleration just before shock was found in both EMG conditions especially on Day 2. These results were also emphasized by the fact that the initial levels of HR were not different among four conditions on Day 2.

But there were not significant differences in the initial HR acceleration component between the NSH and the SH conditions, and so, this component was better to be regarded as equivocal on both days, though the trend analyses showed the significant F values of the quadratic and/or the cubic components.

Magnitude estimation of the shock: Table 4 showed the results of the magnitude estimation of the shock in each condition. On Day 1, the estimates were higher in the H-EMG than in the L-EMG on 3 mA condition ($t=2.72$, $df=8$, $p<.05$) and on 9 mA ($t=3.15$, $df=8$, $p<.05$). The other differences were not significant between both EMG conditions on Day 1 and Day 2.

Relations between three HR scores in the foreperiod and two pain response scores: These three HR scores in the foreperiod were as the same as those previously shown in Fig. 2, except the points that these scores were calculated from each one trial's data by every condition. So, "The last two HRs in the foreperiod" explained in the aforementioned part of the definition of these scores, were the last two HRs immediatery before the shock and thereby were not necessarily 8th and 9th HRs. In addition to these HR scores, the magnitude of the HR acceleration just after the shock (AS score) and the estimates of the shock (E score) were considered as the two kinds of the pain response scores. The AS score was considered to reflect one of the phasic arousal responses elicited by the shock in the autonomic nervous system. The AS

score was defined as the difference (bpm) between the lower HR within two ones in which one was the last HR in the foreperiod and the other was the first HR in the after-shock period, and the highest HR in the after-shock period (as shown in Fig 2). The AS and the E scores were hereinafter referred as the two pain scores.

At first, Peason product-moment correlation coefficients were calculated between the two pain scores by every intensity of the shock in both EMG conditions, but the Day conditions were in the lump (Table 5). The results shown in Table 5 indicated that the significantly positive relations exsisted between these two pain scores in the higher intensity of 6 and 9 (or 8) mA in the both EMG conditions. In the following, it was examined whether any relations existed between three HR scores in the foreperiod and the two pain scores.

AF score & E or AS score: Table 6-(A) showed the correlation coefficients between AF and E. There existed a significantly negative correlaton in 6 mA in the H-EMG, but the other coefficients were almost near to 0. The Table 6-(B) showed the correlation coefficients between AF and AS, which indicated that significantly positive relation existed only in the 9 (or 8) mA in H-EMG condition. But the other coefficients were not significant. From these observations it should be considered that the relations were ambiguous between AF and each pain scores.

D1 score & E or AS score: The Table 7-(A) indicated that when the shock intensity was 3 or 6 mA there existed no significant correlations between D1 and E, but when shock intensity was raised up to 9 (or 8) mA the significantly positive correlations emerged in both EMG conditions. From the Table 7-(B), it was found that there was a significantly positive relation between D1 and AS in 9 (or 8) mA in the L-EMG. Furthermore, in the H-EMG there existed significantly positive relations in all shock-intensity conditions. These observations indicated that the larger deceleration just before the shock correspondent with the larger acceleration just after the shock in the higher intensity of the shock or in the H-EMG condition, and corresponded with the

Table 5. Pearson product-moment correlation coefficients between AS and E scores.

AS & E		Low EMG	High EMG
3 mA	r	0.265	−0.059
	t	1.599	−0.347
		NS	*NS*
6 mA	r	0.502	0.463
	t	3.383	3.045
		**	**
8 or 9 mA	r	0.591	0.445
	t	4.269	2.894
		**	**

($df = 34$), **: $p < .01$, *NS*; non significant

Table 6. Pearson product-moment correlation coefficients between AF and E scores in (A) and between AF and AS scores in (B).

(A) AF & E		Low EMG	High EMG	(B) AF & AS		Low EMG	High EMG
3 mA	r	0.097	−0.087	3 mA	r	0.072	0.235
	t	0.571	−0.509		t	0.423	1.410
		NS	*NS*			*NS*	*NS*
6 mA	r	−0.115	−0.332	6 mA	r	−0.001	0.015
	t	−0.672	−2.054		t	−0.003	0.086
		NS	*			*NS*	*NS*
8 or 9 mA	r	−0.001	0.292	8 or 9 mA	r	0.299	0.449
	t	−0.006	1.780		t	1.829	2.926
		NS	+			+	**

+: $p<.1$, *: $p<.05$, **: $p<.01$, ($df=34$)

Table 7. Pearson product-moment correlation coefficients between D1 and E scores in (A) and between D1 and AS scores in (B).

(A) D1 & E		Low EMG	High EMG	(B) D1 & AS		Low EMG	High EMG
3 mA	r	−0.056	−0.131	3 mA	r	0.215	0.331
	t	−0.326	−0.768		t	1.281	2.046
		NS	*NS*			*NS*	*
6 mA	r	0.212	−0.020	6 mA	r	0.312	0.331
	t	1.263	−0.116		t	1.914	2.044
		NS	*NS*			+	*
8 or 9 mA	r	0.350	0.477	8 or 9 mA	r	0.590	0.615
	r	2.178	3.168		t	4.256	4.550
		*	**			**	**

+: $p<.1$, *: $p<.05$, **: $p<.01$, ($df=34$)

Table 8. Pearson product-moment correlation coefficients between D2 and E scores in (A) and between D2 and AS scores in (B).

(A) D2 & E		Low EMG	High EMG	(B) D2 & AS		Low EMG	High EMG
3 mA	r	−0.232	−0.059	3 mA	r	0.254	0.127
	t	−1.392	−0.342		t	1.528	0.748
		NS	*NS*			*NS*	*NS*
6 mA	r	0.349	0.373	6 mA	r	0.357	0.381
	t	2.169	2.341		t	2.225	2.404
		*	*			*	*
8 or 9 mA	r	0.436	0.368	8 or 9 mA	r	0.361	0.379
	t	2.826	2.309		t	2.260	2.390
		**	*			*	*

+: $p<.1$, *: $p<.05$, **: $p<.01$, ($df=34$)

higher estimates in the highest intensity of the shock.

D2 score & E or AS score: The Table 8-(A) and 8-(B) showed that there were significantly positive relations between D2 and E as well as between D2 and AS in the higher shock intensities in both EMG conditions. These observations like in D1 score indicated that in the higher shock intensities in both EMG conditions the larger deceleration below the initial level of HR corresponded with the larger acceleration just after the shock, and with the higher estimates.

Discussion

On Day 1, the analysis by the three HR scores in the foreperiod (AF, D1 and D2 scores) and the trend analysis manifested the apparent deceleration component just before the shock presentation in the L-EMG condition, but not in the H-EMG condition. Furthremore, on Day 2, these two kinds of analyses confirmed the pronounced HR deceleration rather than on Day 1, in both EMG (L- and H-EMG) conditions. The initial acceleration component on both Days, however, was thought to be still better to regard it as equivocal because the AF score in the SH was not different from that in the NSH, though the trend analysis showed the significant F values of the quadratic and/or cubic components. These HR changing patterns almost consisted with the results shown in the unavoidable shock task by Somsen et al. (1983). In sum, in the two stimulus paradigm with the aversive condition there existed primarily the deceleration component just before the shock and secondarily, though equivocal, the initial acceleration component in the foreperiod.

In this experiment, subject had to keep the muscle tension of the right forearm in the range of 260 μV to 610 μV in the H-EMG condition. This task was considered to demand more metabolic activity, at least, than the gently tapping task used by Obrist et al. (1969). The fact that the significant HR deceleration was found in the H-EMG in the SH condition on Day 2 indicated that in spite of experimentally imposed somatic activity with highly metabolic demands, the "somatic-cardiac inhibitory effects" by the central nervous system were sufficiently strong to overcome the acceleration supposed by its peripherally metabolic demands.

In the next place, we investigated whether these inhibitory effects would attenuate the noxiousness of the aversive stimulus as suggested by Obrist et al. (1968, 1969) or not. In order to examine this suggestion, the correlation analyses were undertaken between the three HR scores (AF, D1, and D2) and the two pain scores (AS and E) which were highly correlated with each other in the higher shock intensities (6 and 9 (or 8) mA). The relations between the AF and each pain score were equivocal. The correlation analyses between D1 and the pain scores indicated that the positive relations existed between D1 and AS in the higher shock intensity and/or in the H-EMG condition, and between D1 and E in the highest intensity (9 or 8 mA) in both EMG conditions. The relations between D2 and the pain scores were also positive in

the higher intensities in both EMG conditions. Apparently, these results did not correspond with the suggestion by Obrist, or rather fitted the consideration in the non-aversive condition that the anticipatory deceleration was the reflection of the attention process which enhanced the "sensory intake". Namely, these results showed the possibility that the "attention hypothesis" considered in the non-aversive condition should be applied to in the aversive situation such as this experiment, too. If so, the "somatic-cardiac inhibitory" process was better to be considered the secondary one which was involved in the attention process, as inferred from the "another possibility" by Obrist et al. (1968, p. 192). In other words, the hightened attention might be related to the HR deceleration and the cessation of spontaneous somatic activity through the "somatic-cardiac inhibitory" process, and concurrently the hightened attention might facilitate the "sensory intake" to a shock, which resulted to the more pain.

However, it was questioned why the HR deceleration correlated with more pain was formed in the situation where the impendimg shock was anticipated. Further, it was questioned whether this anticipatory deceleration was always concomitant with more pain, or with a failure of coping responses. It might be possible to explain these problems by combination of the two hypotheses undermentioned.

At first, from the discussion up to this point, it was hypothesized that the larger magnitude of anticipatory deceleration was concomitant with the more hightened attention. This hypothesis simultaneously suggested that if subject predicted the time hard when an aversive stimulus came upon, the anticipatory phasic deceleration would be small or not be found. This inferrence was supported by Dean's report (1966) which indicated that the phasic deceleration did not occur under the condition of temporally uncertain presentation of a shock. This relation hypothesized between the magnitude of anticipatory deceleration and the degree of the attention to a pressing stimulus was illustrated in the Fig. 3 by a linear line of "a" and the axis of abscissa.

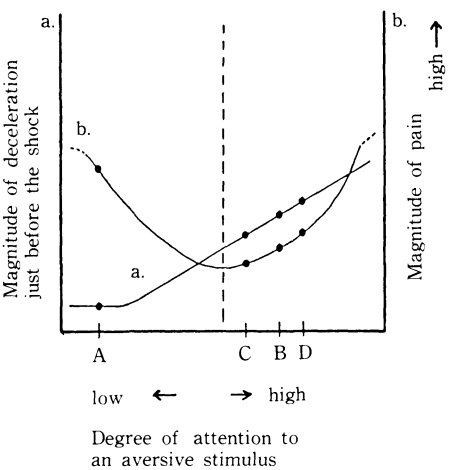

Fig. 3. Hypothetical relationship between the magnitude of deceleration just brfore the shock presentation and the degree of the attention to an aversive stimulaus (shown in "a"), and between the magnitude of pain and the degree of attention too (shown in "b").

As the second, it was hypothesized that the relation between the magnitude of pain and the degree of attention to an aversive stimulus was in the U-shped relationship (as shown in Fig .3). If our attention was focused on a painful region, we tended to perceive pain more intensely than we would normally (e.g. Melzack & Wall, 1982, p. 37). Moreover, Melzack said that "... the mere anticipation of pain is sufficient to raise ... the intensity of preceived pain". In contrast, it was also well known that the distraction of attention away from an aversive event produced the less pain. Namely, the hightened attention to the aversive event caused the more pain (as shown in the point of "D" in Fig .3) but the distraction from this event produced the less pain ("C" in Fig . 3). However, if a person had not paid attention to an impending aversive stimulus or if he could hardly predict the time when the shock was given, he would perceive the more pain, and be startled when the shock fell upon him ("A" in Fig . 3). Compared with this situation, if a certain degree of attention was possibe to be focused on the shock, the less startle and the less pain would be perceived ("B" in Fig . 3). This comparison of pain at the point of "A" with of "B" was investigated partly by our other experiment (Hatayama et al., 1984) which showed, in an aversive condition, that subjects reported more pain in the condition of very poor information about the time of the shock presentation than the in the condition with more precise information about it. From these considerations, it was hypothesized that the pain perception was related to the degree of the attention to the aversive stimulus by the U-shaped relationship.

Now, after the two hypotheses were combined on the basis of the continuum about the degree of the attention to an aversive event, the results of this experiment were probably more understandable. The experimental situations in this report were thought to be basically contained in the range of the right half continuum from the dashed line in Fig . 3, because the subjects in our experiment could apporoximately anticipate the time when the shock was given. In this right half range, the magnitude of the anticipatory deceleration was positively related to the magnitude of pain responses. This relation consisted with the results of this experiment. In contrast, in the left region from the dashed line the anticipatory deceleration was inferred to be positively related to the attenuation of pain.

At last, the relationship of the pain to the continuum of the degree of the attention to an aversive event well fits the facts that the pain caused by a suddenly getting stung by a bee is more severe than the pain by getting injected in a hospital, but when getting injected, it is less painful in the condition of distracting attention from the jnjection. The anticipatory phasic deceleration might not be found immediately before the sudden sting by a bee, but would be found larger in focussing on the injection, and moderate in the distraction. Hereafter, further efforts would be necessary to examine the relationship among the attention, the pain, and the anticipatory phasic deceleration on a concurrent comparison in the same experiment.

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(Received November 15, 1984)