THE ROLF OF DRIVE REDUCTION IN THE CLASSICAL CONDITIONING OF AN AUTONOMICALLY MEDIATED RESPONSE

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APPROVED FOR PUBLIC RELEASE; DISTRIBUTION UNLIMITED. The Role of Drive Reduction in the Classical Conditioning

of an Autonomically Mediated Response

We propose to show that drive reduction can determine the form of a classically conditioned autonomic response, and that as a result there is no fixed relationship of anxiety and heart rate.

A number of investigators have found that the heart rate changes during anxious anticipation of an aversive stimulus, but the direction of change has not always been the same. Some have found the heart to accelerate during anxiety (1, 2, 5, 13, 17), while others have recorded a slow-down (5, 11, 13, 16, 19). A resolution of these discrepant results is offered in the present study.

The most extensive work on heart conditioning has been done recently by Notterman, Schoenfeld, and Bersh (10, 11, 12). They have shown that when anxiety is generated by a tone-shock conditioning procedure the heart slows in rate during the anxiety period between tone and shock. Zeaman, Deane, and Wegner (19) replicated this finding and made a detailed beat-by-beat analysis of the form of both the CR and UR. It is reported in the latter study that during the 6-sec. CS-US interval, the heart rate CR to the 1-sec. tone took the form of a slight initial rise followed by a deceleration of greater magnitude and duration. In contrast, the UR to the 6-sec. shock was a sharp elevation in rate reaching a peak in three seconds, and then a progressive drop in rate until the point of shock termination. The over-all picture was that of a slow-down CR and speedup UR. An attempt was made to relate the findings to several theoretical formulations of the mechanism of conditioning. One of these, the drive-reduction view. suggested that the predominantly decelerative form of the CR was due to the fact that the heart was decelerating at the time of maximal drive reduction (shock-off). A direct test of this view was proposed. It consisted of shortening the duration of the shock US to 2 sec., so that drive reduction would now occur during the acceleration stage of the UR. The predicted outcome was a change in the form of the CR from deceleration to acceleration. The present experiment reports the result.

METHOD

Apparatus

The apparatus has been described in detail in a previous report (19). Briefly, it consisted of an electrocardiograph to measure the heart rate, a 13 V.A.C. shock for the US, a tone (60 db, 512 c.p.s.) for the CS, and electronic timers for controlling the sequence of stimuli.

Subjects

The Ss were 16 students at the University of Connecticut, 10 males and 6 females. All were paid volunteers.

Procedure

The classical trace-conditioning procedure employed was very similar to that described in an earlier paper (19); the major change was the shortening of the duration of the shock from 6 sec. to 2 sec. Our technique was closely patterned after that of Notterman, Schoenfeld, and Bersh (11).

<u>Pre-conditioning</u>.— All Ss were given a series of pre-conditioning trials consisting of the presentation of a number of 1-sec. tones spaced at irregular intervals of one to two minutes. Ten of our Ss were given ten such pre-conditioning trials, and six were given twenty, before the start of the conditioning trials.

<u>Conditioning.-- A conditioning trial consisted of a l-sec.</u> tone followed in six seconds by a 2-sec. shock. All Ss were given ll conditioning trials separated by inter-trial intervals which varied irregularly between one and two minutes.

Cardiograph records were taken on each trial from about twenty seconds before the tone to about forty seconds after the tone. The Ss were instructed that they would receive a series of tones and a series of shocks but the order was not mentioned. The instructions were identical to those of Notterman, Schoenfeld, and Bersh (11).

RESULTS

Pre-conditioning

The response of the heart to tone alone in the pre-conditioning trials is represented by the dashed line in the lower half of Fig. 1. The response, an unconditioned one, is an immediate drop in rate of about 2 b/m, with partial recovery during the ten post-tone beats. The curve is an average of all Ss for all pre-conditioning trials. Since there is no appreciable adaptation of this response, the average curve of the group receiving only ten pre-conditioning trials differed only slightly from that of the group receiving twenty. For this reason, the results of both groups have been pooled.

Conditioning

The effect of tone-shock pairing on the heart's response to tone is shown by the solid curve in the lower half of Fig. 1. The major effect here, after a brief tone-UR dip, is a rise of about 4 b/m followed by a fall to a level below that of the pre-tone period. The last two post-tone beats are the next-to-last and last beats before shock. They have an average ordinal rank of 6.8 and 7.8 counting in the direction of tone to shock. The shock brings about a sharp elevation in rate of about $6\frac{1}{2}$ b/m. At the time of shockoff the heart is strongly accelerating. All points on the

Two 1-page tables giving the numerical values from which these and the subsequent figures were constructed may be found in the Appendix.

Fig. 1. The form of the heart rate response during pre-conditioning and conditioning (lower half). The top half represents the difference between the two lower curves.

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solid-line curve are the averages of all 16 Ss for conditioning trials 2 through 11.

A corrected measure of the effects of conditioning alone is presented in the top half of Fig. 1; it is the difference between the conditioning and pre-conditioning curves in the lower half of Fig. 1. This correction is based on the assumption that the tone UR, which remained constant during the pre-conditioning trials, continues to have a constant effect on heart rate during the conditioning trials. Thus, the tone UR can be subtracted out of the conditioning curve which presumably contains both the tone UR and CR. The result of this correction is to bring out the predominantly accelerative form of the CR. Of our 16 Ss, 6 had <u>uncorrected</u> CR's resembling closely the average corrected CR, that is, with no drop below pre-tone level for the next-to-last and last beats before shock. The negative values of the corrected curve signify only that the over-all level of heart rate is lower during the conditioning trials than the pre-conditioning trials, because of the Ss' progressive approach to a basal state during the course of the experimental session.

Having shown that the form of the CR is an acceleration with a maximum amplitude at the third post-tone beat, we show now that this CR has a gradual onset. The course of conditioning can be traced in Fig. 2. On each trial, the mean rates for all Ss of beats 3 and 4 are averaged and expressed as a difference from the mean rate during the ten pre-tone beats on that trial. The difference measure was chosen here merely to cancel out trial-by-trial changes in over-all level of heart rate. The solid line shows a typical negatively accelerated curve of conditioning. The negative values of the difference measure during the ten pre-conditioning trials indicate that the tone UR has depressed the rate below pre-tone level by approximately l_2^1 b/m. It can be seen from the flatness of the preconditioning curve that the tone UR showed no tendency to adapt out. For the sub-group which received twenty pre-conditioning trials, only the data from the second ten of the pre-conditioning trials are included in Fig. 2.

The next-to-last and last beats before shock were also averaged and expressed as a difference from the mean of the ten pre-tone beats on each trial. Considering the amount of pre-conditioning variability, we see no convincing evidence of a conditioned deceleration for this portion of the response.

DISCUSSION

The data we have presented clearly establish the fact that the heart shows a conditioned acceleration in anticipation of a short shock. This is to be contrasted with the previous finding of a conditioned deceleration when a long shock was used (11, 19). What the heart is doing at the time of shock-off seems to make the difference, Fig. 2 The course of conditioning for the accelerated and decelerated portions of the CR.



In the light of this finding, we were led to re-examine the data from our long-shock experiment. because we had observed there some evidence of a slight conditioned acceleration in the beginning of the CR, just before the large conditioned deceleration. The possibility was considered that some atypical Ss under the long-shock condition did not have a heart rate deceleration at the time of shock-off. Out of the group of 17 Ss we found 4 such cases. Their data were analyzed separately, and the result is shown in Fig. 3. The measure here is analogous to that in the top half of Fig. 1. Note that for these Ss the heart, at the time of shock-off, has just passed its peak of acceleration, and has not yet started its deceleration. Observe that, more importantly, the CR resembles closely that found in the present experiment with short shock. It must be emphasized that these Ss were selected only on the basis of the form of UR, not CR.

The remaining 13 Ss of our long-shock experiment were all decelerating at the time of shock termination. The form of their response is shown in Fig. 4. Here the reverse effect is clearly seen, a conditioned slow-down in rate. A comparison of the course of conditioning of these two contrasting forms of CR is depicted in Fig. 5. The two learning curves are similar in shape and magnitude, with the short shock yielding slightly better conditioning. We view each curve as representing the formation of a connection between the tone stimulus trace and the specific response occurring at the time of drive reduction (shock-off).

The results of both experiments taken together lead to the conclusion that the accelerated or decelerated form of the heart rate CR is not determined directly by the duration of the US, since both effects were obtained for different Ss under the same duration of shock, but rather that the particular phase of the UR occurring at the time of shock termination is selected for connection to the CS trace. The duration of US selects the type of CR only indirectly through its control of the UR.

Granting the above conclusion, it is not difficult to understand how previous investigators found sometimes one and sometimes another type of heart rate CR. A variety of types and durations of unconditioned stimuli have been used with no attention to the important factor of drive reduction which predicts the type of CR to expect. Earlier studies in cardiac conditioning have not been reported in sufficient detail to allow us to use them as precise tests of our conclusion. No obviously contrary cases have been reported.

We wish to consider briefly the relation of our results to theories of anxiety and the mechanism of conditioning. If one holds to the simple descriptive definition of anxiety as a state produced by some current stimulus which in the past has been followed by a noxious stimulus (4, 15), then the different heart rate CR's in both our short-shock and long-shock experiments can be Fig. 3 The form of the heart response for Ss whose hearts did not decelerate during a long shock.



Fig. 4 The form of the heart response for Ss whose hearts did decelerate during a long shock.



Fig. 5 The acquisition of the conditioned acceleration in the short-shock experiment compared with that of the conditioned deceleration in the long-shock experiment.



PHE-TONE--POST-TONE DIFFERENCES (B/H)

said to have occurred during anxiety. Thus the appropriate statement of the relationship of anxiety and heart rate is that the heart changes its rate during anxiety, but in a direction determined by factors other than anxiety.

If one views anxiety as an autonomically mediated response, conditioned like other viscero-vascular responses, then the mechanism by which heart rate is conditioned will also be the mechanism by which anxiety is learned. There are at least two schools of thought on this matter: one which holds that autonomic responses are conditioned according to a contiguity principle (3, 7, 9, 14, 18), and another which maintains that they are conditioned by drive reduction (6, 8). Since in the present experiment it was the shock termination (drive reduction) which proved to be the critical element in selecting the autonomic response to be conditioned, the results lend most comfort to the drive-reduction view.

SUMMARY

The heart rate response of human Ss was classically conditioned, using a l-sec. tone CS and a 2-sec. shock US separated by a 6-sec. delay interval. During the anxiety period between tone and shock, the conditioned cardiac response appeared as an acceleration in rate. This was contrasted with the conditioned deceleration found during anxiety when a long (6-sec.) shock was used. The difference in the form of the two CR's was attributed to the fact that at the time of shock termination the heart was accelerating in the short-shock condition and decelerating in the long-shock condition. The results are seen to favor the view that the mechanism of conditioning for this autonomically mediated response was that of drive reduction.

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APPENDIX

TABLE I

Mean heart rate (in beats per minute) during the pre-tone, tone, post-tone, shock, and post-shock periods for three groups of Ss. Group I (16 Ss) received a short (2 sec.) shock; Group II and III received a long (6 sec.) shock. The columns labelled 'difference' give the mean difference in rate between pre-conditioning and conditioning. The reported means are of all Ss under the given condition. Group II contains 4 Ss whose hearts were not decelerating at the time of shock termination. Group III contains 13 Ss whose hearts were decelerating at the time of shock termination. The post-tone beats labelled NI, and L are respectively the next-to-last and last beats before shock.

Ordinal Beat	Group I (Fig. 1)		Group II	Group III	
Number				F18. 3)	(r1g. 4)
	Pre-		Diff.:	Diff.:	Diff.:
	condit.	Condit.	PrecCond.	PrecCond.	PrecCond
Pre-tone 1	78.64	76.42	-2.22	-1.31	-3.07
2	78.98	76.39	-2.59	-1.72	-2.66
3	79.30	76.73	-2.57	-2.00	-2.61
4	79.36	75.79	-3.57	-2.73	-2.81
5	78.99	76.16	-2.83	-1.68	-2.54
6	78.98	75.98	-3.00	73	-2.56
7	79.30	75.66	-3.64	-2.00	-2.49
8	79.04	75.87	-3.17	-1.88	-2.52
9	78.62	75.89	-2.73	-2.02	-2.23
10	78.34	75.80	-2.54	-1.48	-2.89
Tone	76.61	74.66	-1.95	45	-2.32
Post-tone 1	76.50	76.37	13	•95	-1.73
2	76.84	77.68	.84	3.45	-2.07
3	77.31	78,78	1.47	3.58	-3.51
4	77.25	78.06	.81	3.56	-3.80
5	77.23	76.30	93	2.42	-4.39
6				(NL) .45	(NL) -4.25
(NL) 7	77.14	73.79	-3.35	(L) -1.38	(L) -4.96
(L) 8	77.12	73.24	-3.88		
Shock 1	76.80	74.93	-1.87	57	-2.31
2	77.15	78.62	1.47	4.69	32
3				7.98	•55
4				9.44	07
5				9.42	-1,32
6				9.37	-2.37
7				9.49	-2.58
Post-shock 1		79.36			
2		79.62			
3		78.96			
14		77.98			
5		77.35			
6		77.25			
7		77.98			
8		78.29			
9		78.64			
10		78.63			

TABLE II

Differences in heart rate (in b/m) between the mean rate of the ten pre-tone beats on each trial and the mean rate of the following posttone beats: beat 3 and 4 for the short-snock condition (Column 1), the next-to-last and last beat for the short-shock condition (Column 2), and the next-to-last and last beat for the long-shock condition (Column 3). The differences have been averaged over-all Ss and over blocks of three trials. The number indicating each three-trial block is the middle number of that block. The number of subjects is shown at the top of each column.

	1.	2.	3.
Rolling Averages of 3-Trial Blocks	Beat 3 and 4 <u>Short Shock</u> (Fig. 2 and 5) N=16	Next-to-last and last beat <u>Short Shock</u> (Fig. 2) N=16	Next-to-last and last beat Long Shock (Fig. 5) N=17
Pre-conditioning 2	-1.48	-2.1?	-1.65
3	-1.45	-1.10	-1.62
5	-1.77	98	-1.62
6	-1.05	76	-1.48
7	-1.23	-2.54	-1.73
8	-2.20	-2.21	-1.54
9 10	-1.89 69	-2.63 -1.14	-1.70 58
Conditioning			
2	1.05	-2.14	-2.34
3	1.25	-3.06	-3.18
4	1.70	-3.52	-3.35
5	2.11	-3.12	-4.57
6	2.49	-2.57	-4.71
7	2.70	-2.59	-4.02*
8	J.43	-2.50	-4.50**
ל חור	2.21	-2.22	-4.40
10	1.21	-6.66	

*Interpolated

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