

ACTIVE AND PASSIVE COPING UNDER DIFFERENT DEGREES OF STRESS: EFFECTS ON URINARY AND PLASMA CATECHOLAMINES AND ECG T-WAVE

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Cardiac sympathetic, urinary and plasma catecholamine effects of active and passive coping were investigated during both low and high stress. Stress intensity was manipulated by varying the work load on a bicycle ergometer.

As predicted, T-wave flattening of the ECG – an index of cardiac sympathetic activity – was significantly more pronounced during active coping than during passive coping. This effect did not depend upon the degree of stress. A significant increase in both adrenaline and noradrenaline levels depended upon the combined effects of active coping and high stress. Because high concentrations of circulating catecholamines are thought to be related to cardiovascular pathology, the results suggest that active coping during high stress might involve the highest cardiovascular risks.

1. Introduction

Although it is generally accepted that stress-induced adrenergic changes are important in the etiology of cardiovascular disease, the behavioral conditions related to these changes are still poorly defined. Factual or perceived control over an aversive event is one of the factors modulating the intensity of stress reactions (Averill, 1973). During control, as opposed to no control, both lower (Hokanson, De Good, Forrest and Brittain, 1971) and higher (Obrist, Light, McCubbin, Hutcheson and Hoffer, 1979) systolic blood-pressure has been reported. Obviously, specification of the conditions in which control is associated with an increase or a decrease in a particular physiological reaction system is necessary.

One influential approach studying these conditions has been reported by Obrist and coworkers. Reviewing an extensive series of experimental results Obrist (1976) concluded that two types of control over an aversive event, i.e.

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active and passive coping, are associated with different cardiovascular reaction patterns. During active coping, when subjects can escape or avoid the aversive event, relatively strong beta-adrenergically-mediated cardiac and vascular changes were observed. During passive coping, when subjects are helpless recipients of the aversive event, vagal influences dominate the heart (Obrist, Howard, Lawler, Galosy, Meyers and Gaebelein, 1974).

In a recent report (Light and Obrist, 1980) conditions related to sympathetic dominance on the cardiovascular system (CVS) were further specified; only control requiring a reasonable degree of effort gave rise to strong sympathetic effects on the heart. The cardiovascular reaction pattern found during active coping and stress is similar to the cardiovascular reaction pattern during the initial stages of fight-flight behavior (Obrist et al., 1974). Because no strenuous muscular activity was observed in the active coping situation, Obrist proposed that active coping occurs in a metabolically unjustified situation with a potential for cardiovascular disease.

In the present study, two questions related to Obrist's research were investigated. The first concerned the interaction between type of coping and degree of stress. Obrist et al. (1974) suggested that sympathetic influences on the heart can be evoked by stress and situations in which the organism engages in preparing or executing activities in order to cope with the stress. However, coping and stress have not been varied independently within one experimental design. In order to investigate whether coping or stress is the more effective variable causing the sympathetic effects on the heart, these variables were varied independently in the present study. It was hypothesized that active coping, as opposed to passive coping, results in cardiac sympathetic activation during both high and low stress.

The second question concerned the effects of coping and stress on plasma and urinary catecholamine levels. The emergency state of the fight-flight reaction is characterized by a wide-spread sympathetic activity, which also acts to release catecholamines from the medulla of the adrenal gland into the bloodstream (Cannon, 1915).

Because there is ample evidence that high levels of circulating catecholamines are related to cardiovascular pathology (Katz, 1977; Weick, Ritter and Ritter, 1980; Raab, Stark, MacMillan and Gisee, 1961; Rabinowitz, Elazar, Orenstein and Neufeld, 1978), the conditions in which coping is associated with an increase in circulating catecholamines should be specified. Although the effect of active versus passive coping on catecholamine excretion has not been investigated as yet, Lundberg and Frankenhaeuser (1978) showed that control versus no-control over noise intensity during low stress did not affect catecholamine excretion to a significant degree. Furthermore, Frankenhaeuser (1971) reported a non-linear relationship between cardiovascular changes and catecholamine excretion during physical exercise with no change in catecholamine excretion at low work-loads. In the present study it was there-

fore hypothesized that the fight-flight pattern occurs only if the organism actively tries to cope with high stress, whereas active coping during low stress is associated with increased cardiac sympathetic activity without a concomitant increase in catecholamine concentrations. Cardiac sympathetic activity was measured by *T*-wave flattening of the electrocardiogram (Punch, King and Matyas, 1976; Quazzi, Fiorentini, Polese, Magrini and Olivari, 1975). Urine and plasma samples were analyzed for catecholamines, i.e. adrenaline, noradrenaline, dopamine. Adrenaline concentrations were used as an index for adrenal medullary activity and noradrenaline as an index for sympathetic nervous system activity (Laverty, 1978). The physiological significance of plasma dopamine is still much disputed (Callingham and Barrand, 1976). Urinary dopamine is probably involved in natriuresis (Kuchel, Buu and Unger, 1978). No consistent relation between dopamine and stress or coping has been reported. Therefore no specific predictions concerning dopamine were formulated in the present study.

2. Method

2.1. Subjects

Twenty seven history students participated in the experiment. Their mean age was 22 years. Twenty five dutch guilders were paid to each subject. Because of cyclic fluctuations in catecholamine levels each experimental session was planned in the morning from 9 am to 12 am. Only male, non-smoking and physically untrained subjects participated. On the night before the experiment no alcohol was used and breakfast was consumed without coffee or tea.

2.2. Procedure

To obtain different degrees of stress, the same psychological stress was superimposed upon two levels of physical stress. The psychological stress consisted of a white noise of 100 dB. High and low workloads on a bicycle ergometer were used as the physical stressor. In order to vary type of control, subjects were asked to maintain a rhythm of exactly 70 cycles per minute on the bicycle ergometer. Pretrials have shown this to be a difficult task with no significant learning effect during the experimental period. In case of active coping each deviation from 70 cycles/min was followed by the noise. As soon as the correct rhythm was re-established the noise disappeared. Each subject of the passive coping group was exposed to the tape-recorded noise pattern of a subject from the experimental group. This yoked group could in no way escape or avoid the noise.

Subjects exercised twice during half an hour on the bicycle ergometer.

Workload was related to the amount of fat-free mass (FFM), estimated by measuring skin folds of the upper arm. Light exercise consisted of 0.5 Watt/kg FFM leading to an estimated oxygen consumption of 40% VO_2 max. Submaximal workloads were obtained using 2.0 Watt/kg FFM resulting in an estimated oxygen consumption of 60% VO_2 max. Each subject participated in both physical stress conditions. The order of low and high stress periods were systematically varied. The only task of each subject in both groups was to keep his rhythm at exactly 70 cycles/min. A green light indicated a correct rhythm, two red lights showed the rhythm too fast or too slow. Variations between 60 and 80 cycles/min on the bicycle ergometer did not affect work-load. During the experiment all subjects remained within this range. The total amount of time subjects deviated from the norm of 70 cycles/min was registered.

Each session started at 9 am. The first stress period was from 10.00 to 10.30 am, the second period was from 11.00 am to 11.30 am. Blood samples were taken immediately before and immediately after the stress periods, while sitting on the bicycle. Urine samples were obtained at 10, 11 and 12 am. To minimize the effects of needle insertion on catecholamine levels, at approximately 9.15 am a catheter was inserted. Only from subjects who did not object in any way to this procedure were blood samples taken.

Blood samples (5 ml) were collected in ice-chilled, heparinized tubes that contained 5.5 mg of disodium ethylenediaminetetraacetic (EDTA) and 6.0 mg of reduced glutathione (GSH) in a volume of 60 μl . Blood was then centrifuged and plasma was stored at -30°C . After a sample had been obtained, the catheter was rinsed with saline containing heparine (85 USP units/ml).

Immediately after arrival, the subjects emptied their bladders. Half way between two urine samples, 200 ml of mineral water was consumed. Urine samples were immediately acidified with 0.1 volume of 2 M perchloric acid. Five ml of the sample was stabilized with EDTA and GSH, and stored at -30°C .

Catecholamine levels were measured radioenzymatically (van der Gugten, Palkovits, Wijnen and Versteeg, 1976). Urine samples were diluted fifty times. Before and during the stress periods the three orthogonal electrocardiograms (X, Y, Z) using a modified Frank lead system were recorded on analog tape. Five minutes registration before each stress period was processed to obtain baseline values and twenty seconds during each minute of the stress periods were processed to obtain the experimental data. During each selected period the X, Y and Z lead was sampled at 2 μsec by a PDP-8E computer.

A detailed description of the computerized ECG analysis can be found elsewhere (Simoons, Boom and Smalenburg, 1975). For each sample period a single representative beat with a low noise level was obtained by averaging all detected beats, except those with an aberrant waveform. Each waveform analysis was also visualized on a scope and rejected if it was apparently non-realistic. The remaining averaged ECG complexes were quantified and for

the present purpose the area enveloped by the *T*-wave was selected for further analysis. Previous data had shown that the X-lead values were the most informative. Therefore only the X-lead results are reported.

After each stress period, subjects were asked whether the coping task was “easy”, “difficult” or “very difficult”, and whether the noise was “not irritating”, “irritating” or “very irritating”. In a post-experimental interview subjects were asked whether they had experienced control over noise presentations. The results of these questions were used to check the effectiveness of the manipulations.

3. Results

Seven subjects objected to needle insertion. The remaining subjects were equally divided over conditions.

The experimental questions showed that the experimental task was evaluated as difficult and the noise as irritating or very irritating. On both these verbal measurements the between-condition differences were not significant. The active coping (AC) group deviated on the average 229 sec from the norm of 70 cycles/min, the passive coping (PC) group deviated on the average 333 sec from the norm. This difference is highly significant ($p < 0.001$). No differences were found between high and low stress.

3.1. ECG *T*-wave area

Analysis of the ECG data showed that after several minutes a steady state was reached, i.e. after the first few minutes no significant trend for change could be observed in the data. Therefore the *T*-wave data from the last twenty minutes were averaged.

In table 1 differences from the preceding baseline values are reported.

Table 1
ECG *T*-wave area ^{a)}

	Low stress (LS)	Low stress vs. baseline	<i>N</i> ^{b)}	High stress (HS)	High stress vs. baseline	<i>N</i>	HS vs. LS
Active coping (AC)	- 71	$p < 0.001$	10	- 204	$p < 0.001$	10	$p < 0.001$
Passive coping (PC)	+ 19	n.s.	10	- 115	$p < 0.01$	10	$p < 0.001$
AC vs. PC		$p < 0.005$		$p < 0.01$			

^{a)} Data are presented as differences from baseline; between-conditions effects were tested with Student's *t*-test, one-tailed significance levels are reported.

^{b)} *N* = number of subjects.

Baselines from the first and the second stress period were not significantly different. Table 1 shows that during active coping the *T*-wave area is significantly smaller than during passive coping. This effect is apparent during both high and low stress.

During high stress the *T*-wave area is significantly smaller than during low stress. Finally, with the exception of PC during LS the *T*-wave data differ significantly from baseline.

3.2. Catecholamines

The catecholamine concentrations found during baseline periods are given in table 2.

No significant baseline differences existed for the plasma concentrations. Only one baseline was obtained for the urine samples. Apart from baseline values (table 2) catecholamine data are presented as differences from baseline. Results and levels of significance are given in tables 3 and 4. Significance levels are one-tailed, except for dopamine.

3.3. Noradrenaline

Table 3 shows that the urinary NA concentration is significantly increased during active coping and high stress only. According to table 4, plasma NA concentrations are higher during HS than during LS. The difference in mean NA between active and passive coping during high stress is in the predicted direction but fails to be significant (table 4). During low stress both the plasma and the urine concentrations are not different from baseline. During high stress the mean NA levels are significantly higher than during baseline periods (tables 3 and 4).

Table 2

Catecholamine concentrations during baseline conditions

		Urine ^{a)} (ng/min)	Plasma ^{b)} (pg/ml)
Adrenaline	Active coping	—	113
	Passive coping	—	85
Noradrenaline	Active coping	53	344
	Passive coping	37	356
Dopamine ^{c)}	Active coping	297	—
	Passive coping	242	—

^{a)} Urinary adrenaline values were unreliable, see text.

^{b)} Plasma values during the two baseline periods were not significantly different, therefore the average values of the baseline periods are given.

^{c)} Dopamine plasma values were generally below detection levels.

Table 3
Urinary catecholamines (ng/min)^{a)}

	Low stress (LS)		LS vs. baseline		<i>N</i> ^{b)}	High stress (HS)		HS vs. baseline		<i>N</i>	HS vs. LS	
	NA ^{c)}	DA ^{d)}	NA	DA		NA	DA	NA	DA		NA	DA
Active coping (AC)	+ 9	+ 129	ns	ns	10	+ 34	+ 85	$p < 0.005$	ns	10	$p < 0.005$	ns
Passive coping (PC)	+ 5	+ 86	ns	ns	9	+ 10	- 12	$p < 0.05$	ns	9	$p < 0.05$	ns
AC vs. PC	ns	ns				$p < 0.01$	ns					

^{a)} Data are presented as differences from baseline; between conditions effects were tested with Student's *t*-test, one-tailed significance levels are reported for NA and two-tailed significance levels for DA.

^{b)} *N* = number of subjects.

^{c)} NA = noradrenaline.

^{d)} DA = dopamine.

Table 4
Plasma catecholamines (pg/ml)^{a)}

	Low stress (LS)		LS vs. baseline		<i>N</i> ^{b)}	High stress (HS)		HS vs. baseline		<i>N</i>	HS vs. LS	
	NA ^{c)}	A ^{d)}	NA	A		NA	A	NA	A		NA	A
Active coping (AC)	+ 32	+ 64	ns	$p < 0.025$	9	+ 244	+ 142	$p < 0.005$	$p < 0.01$	9	$p < 0.005$	$p < 0.05$
Passive coping (PC)	+ 58	+ 81	ns	$p < 0.025$	9	+ 199	+ 81	$p < 0.005$	$p < 0.025$	9	$p < 0.005$	ns
AC vs. PC	ns	ns				ns	$p < 0.05$					

^{a)} Data are presented as differences from baseline; between-conditions effects were tested with Student's *t*-test, one-tailed significance levels are reported.

^{b)} *N* = number of subjects.

^{c)} NA = noradrenaline.

^{d)} A = adrenaline.

3.4. Adrenaline

Due to high dopamine concentrations in urine, the interference between adrenaline and dopamine in urine samples was of the same magnitude as the adrenaline concentration itself. Therefore the urine data, being unreliable, are not reported.

Table 4 shows that plasma adrenaline is significantly increased from baseline in all conditions. Type of coping is effective only during high stress; active coping is then associated with higher concentrations than passive coping.

3.5. Dopamine

Plasma dopamine concentrations are not reported because too many subjects remained below the detection threshold. The data in table 3 show that none of the conditions has a significant effect on urinary dopamine concentrations.

4. Discussion

The present experimental task was chosen because pretrials indicated that it was experienced as a difficult but certainly not impossible task. The results of the post-stress questions confirmed this. The postexperimental interview showed that all subjects in the active coping condition did perceive control over the noise while subjects in the passive coping condition did not. Therefore it is reasonable to assume that active and passive coping were established in a moderately difficult task situation.

Adrenaline concentrations increase with psychological stress (Frankenhaeuser, 1971). Thus the adrenaline increase from baseline in all conditions shows that noise has been an effective psychological stressor in this situation. Degree of stress was varied by imposing two levels of physical stress on the subjects via a bicycle ergometer; the reliability of this procedure is generally accepted.

The hypotheses formulated in the introduction are concerned with the effects of active versus passive coping on peripheral reactions during two degrees of stress. It was predicted that active coping results in higher sympathetic cardiac activity than passive coping whatever the degree of stress. The sympathetic effects were evaluated by *T*-wave changes.

Table 1 shows that the *T*-wave area is significantly smaller during AC than during PC. This supports the hypothesis.

A second hypothesis was based on the assumption that active coping is associated with a fight-flight like peripheral reaction pattern only if high stress is also present. The fight-flight pattern is characterized by a wide-spread

increase in sympathetic activity and increased adrenal medullary activity. If AC + HS results in a fight-flight like peripheral reaction, the plasma and urinary concentration of adrenaline and noradrenaline should be significantly higher than in any other condition. Support for this hypothesis comes from table 4 showing that the highest A concentrations are found in the AC-HS condition.

The hypothesis is also supported by the urinary NA results; the highest concentration is found in the AC-HS condition (table 3). The hypothesis is not supported by the plasma NA results, i.e. NA concentrations do not differ between active and passive coping during high stress (table 4). An explanation for this unexpected result can be suggested. Whereas urinary catecholamine values reflect an average activity over a relatively long period, plasma catecholamine levels reflect a much more momentaneous situation. The peripheral effects of active coping depend upon a continuous coping effort by the subjects (Light and Obrist, 1980). The fact that the active coping group deviated significantly less from the norm of 70 cycles/min than the passive coping group supports the idea that the effort invested during coping is at least one of the factors differentiating active from passive coping. Consequently, the difference between the plasma and the urine NA concentrations found might be attributed to a fading coping effort during the very last part of the experimental periods. Because only the total time that subjects deviated from the norm was registered, it was not possible to substantiate the idea of a fading coping effort during the last part of the experimental periods. As expected, none of the conditions affected dopamine concentrations to a significant degree.

In summary, in the present study it was shown that active coping, as opposed to passive coping, results in cardiac sympathetic activation (*T*-wave flattening) during both high and low stress. Elevated noradrenaline levels, indicating intense sympathetic discharges, and increased adrenaline levels, indicating increased adrenal-medullary activity, were obtained only if active coping was associated with a high degree of stress. Stress intensity was manipulated by varying the work-load on a bicycle ergometer. Consequently, generality of the results can only be established by further research using other types of stressors.

These results suggest that the contribution of active coping towards cardiovascular disease postulated by Obrist might depend upon or is increased by a stress-induced increase in circulating catecholamines. Because the present study investigated only acute reactions, the connection between coping and cardiovascular disease remains, however, a mere hypothesis. Future research should be directed to the chronic effects of coping and stress on the cardiovascular system. This research should also investigate whether catecholamine levels or duration and frequency of adrenal-medullary activation are related to cardiovascular disease.

References

- Averill, J.R. (1973). Personal control over aversive stimuli and its relationship to stress. *Psychological Bulletin*, 80, 286–303.
- Callingham, B.A. and Barrand M.A. (1976). Catecholamines in blood. *Journal of Pharmacy and Pharmacology*, 28, 356–360.
- Cannon, W.B. (1915). *Bodily Changes in Pain, Hunger, Fear and Rage*. Appleton-Century: New York.
- Frankenhaeuser, M. (1971). Behavior and circulating catecholamines. *Brain Research*, 31, 241–262.
- Hokanson, J.E., DeGood, D.E., Forrest, H.S. and Brittain, T.H. (1971). Availability of avoidance behaviors in modulating vascular stress responses. *Journal of Personality and Social Psychology*, 19, 60–68.
- Katz, A.M. (1977). *Physiology of the Heart*. Raven Press: New York, 368–370.
- Kuchel, O., Buu, N.T. and Unger, T. (1978). Dopamine-sodium relationship: Is dopamine a part of the endogenous natriuretic system? *Contributions to Nephrology*, 13, 27–36.
- Lavery, R. (1978). Catecholamines: Role in health and disease. *Drugs*, 16, 418–440.
- Light, K.C. and Obrist, P.A. (1980). Cardiovascular response to stress: Effects of opportunity to avoid, shock experience, and performance feedback. *Psychophysiology*, 17, 243–252.
- Lundberg, U. and Frankenhaeuser, M. (1978). Psychophysiological reactions to noise as modified by personal control over noise intensity. *Biological Psychology*, 6, 51–59.
- Obrist, P.A. (1976). The cardiovascular-behavioral interaction as it appears today. *Psychophysiology*, 13, 95–107.
- Obrist, P.A., Howard, J.L., Lawler, J.E., Galosy, R.A., Meyers, K.A. and Gaebelin, C.J. (1974). The cardiac somatic interaction. In: Obrist, P.A., Black, A.H., Brener, J. and DiCara, L.V. (Eds.). *Cardiovascular Psychophysiology*. Aldine Publishing Company: Chicago, 136–163.
- Obrist, P.A., Light, K.C., McCubbin, J.A., Hutcheson, J.S. and Hoffer, J.L. (1979). Pulse transit time: Relationship to blood pressure and myocardial performance. *Psychophysiology*, 16, 292–301.
- Punch, J.C., King, M.G. and Matyas, T.A. (1976). ECG T-wave amplitude, muscle tension and heart rate concomitants of conditioned suppression. *Physiological Psychology*, 4, 294–302.
- Quazzi, M., Fiorentini, C., Polese, A., Magrini, F. and Olivari, M.T. (1975). Stress-induced and sympathetically-mediated electrocardiographic and circulatory variations in the primary hyperkinetic heart syndrome. *Cardiovascular Research*, 9, 342–354.
- Raab, W., Stark, E., MacMillan, W.H. and Gisee, W.R. (1961). Sympathetic origin and anti-adrenergic prevention of stress induced myocardial lesions. *American Journal of Cardiology*, 8, 203–211.
- Rabinowitz, B., Elazar, E., Orenstein, A. and Neufeld, H.N. (1978). Plasma catecholamines and cyclic AMP levels in experimental atrial tachycardia and after coronary occlusion. In: Schwartz, P.J., Brown, A.M., Malliani, A. and Zanchetti, A. (Eds.). *Neural Mechanisms in Cardiac Arrhythmias*. Raven Press: New York, 301–304.
- Simoons, M.L., Boom, H.B.K. and Smallegange, E. (1975). On-line processing of orthogonal exercise electrocardiogram. *Computers and Biomedical Research*, 8, 105–117.
- Van der Gugten, J., Palkovits, M., Wijnen, H.J.L.M. and Versteeg, D.H.G. (1976). Regional distribution of adrenaline in rat brain. *Brain Research*, 107, 171–175.
- Weick, B.G., Ritter, S. and Ritter, R.C. (1980). Plasma catecholamines: Exaggerated elevation is associated with stress susceptibility. *Physiology and Behavior*, 24, 869–874.