

Voluntary Control of Pulse Transmission Time to the Ear

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ABSTRACT

Two experiments involving voluntary control of pulse transmission time (PTT) to the ear were performed. In Experiment I (within-subject, 3 sessions), 12 male subjects attempting to control PTT with feedback showed significant bidirectional PTT changes in the target directions accompanied by parallel changes in pre-ejection period (PEP). There was no evidence of concomitant changes in respiration rate or general somatic activity. PTT control deteriorated across sessions.

In Experiment II (between-subjects, 3 sessions), 10 male subjects attempting to decrease PTT with feedback produced significant PTT decreases accompanied by PEP decreases. There was marginal evidence of increases in respiration rate but no changes in general somatic activity in this condition. Five subjects attempting to increase PTT with feedback and 5 subjects attempting bidirectional PTT control without feedback showed no significant changes in PTT or PEP.

The results from these experiments indicate that subjects demonstrate a modest degree of control over PTT to the ear when provided with feedback. This control of PTT is accompanied by parallel changes in PEP but is relatively free of somatic and respiratory concomitance.

DESCRIPTORS: Pulse transmission time, Pre-ejection period, Voluntary control, Biofeedback.

This report describes two experiments which examined characteristics of voluntary control of pulse transmission time (PTT) to the ear. Previous work with feedback of PTT has utilized the interval between the R-wave of the electrocardiogram and the upstroke of the radial pulse wave (e.g. Steptoe, 1976, 1977, 1978). PTT to any peripheral pulse site encompasses both an intracardiac component, essentially equal to pre-ejection period (PEP), and an arterial component. The PEP component is related to cardiac contractility and reflects beta-adrenergic cardiac influences (Ahmed, Levinson, Schwartz, & Ettinger, 1972; Newlin & Levenson, 1979); while the arterial component is related to arterial distensibility and mean arterial blood pressure (Gribbin, Steptoe, & Sleight, 1976).

Recent research in our laboratory (Newlin, 1979;

Newlin & Levenson, 1979) indicates that changes in PTT to the finger with biofeedback are a function of changes in both the PEP and the arterial component of PTT, rather than changes in the arterial component alone. These findings are seen as arguing against interpretation of changes in PTT as simply reflecting changes in mean arterial pressure (e.g. Steptoe, 1976, 1977). Obrist, Light, McCubbin, Hutcheson, and Hoffer (1979) have provided some confirmation of the relationship between PTT and mean arterial pressure, but interpret this as a secondary effect of changes in cardiac contractility acting to alter systolic blood pressure.

The present research is intended to provide additional information concerning the PEP and arterial components of PTT change with biofeedback. In these experiments, PTT to the ear rather than the finger is utilized to reduce the arterial component and thus increase the relative contribution of PEP. To the extent that voluntary control of PTT is a function of changes in PEP (and thus presumably of

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changes in cardiac sympathetic drive), it is hypothesized that concomitant changes in somatic and respiratory activity will be minimized. This prediction follows from the work of Obrist, Lawler, Howard, Smithson, Martin, and Manning (1974) indicating cardiac-somatic uncoupling under conditions of sympathetic cardiac activation. In addition, questions concerning the consistency across sessions of phenomena related to PTT control, and the necessity of biofeedback for PTT control are addressed.

Results from two experiments are reported. In the first experiment, subjects attempted bidirectional control of PTT to the ear with biofeedback. In the second experiment, subjects attempted unidirectional control of PTT with feedback, with a control group attempting bidirectional control without feedback. In both experiments PEP, heart rate, respiration, and somatic activity were monitored during attempted control of PTT.

EXPERIMENT I

Method

Subjects

Subjects were 12 male undergraduate and graduate students (median age = 23 yrs) who responded to a classified advertisement offering "\$2.25 per hour for subjects in a psychology experiment." Subjects who reported a history of any cardiovascular problems were excluded.

Apparatus

All recordings were made on a Grass Model 7 Polygraph. The electrocardiogram (EKG) was obtained with Beckman miniature electrodes in bipolar configuration. Phonocardiogram (PCG) was recorded with a low frequency microphone strapped at the apex with 10 Hz high pass filtering. The ear densitogram raw signal was transduced with a Hewlett-Packard #780-16 photoelectric earpiece attached to the pinna of the right ear. This signal was differentiated with a locally constructed circuit with a 100 Hz corner frequency. Respiration was measured using a mercury strain gauge stretched across the chest. General somatic activity (GA) was sensed with an electromagnetic coil attached to the suspension of the subject's chair which generated current with bodily movement. GA was amplified with a 7P5A Pre-amplifier and integrated with a 7P10B Integrator.

EKG, PCG, ear densitogram, respiration and GA signals were routed from Grass 7DAC Driver Amplifiers to analog-to-digital conversion channels on a PDP-11 mini-computer. The computer analyzed beat-by-beat cardiovascular measures on-line, and stored trial means for subsequent analysis.

The computer also controlled beat-by-beat feedback of the interval between the Q wave and the upstroke of the ear pulse wave. The feedback consisted of a light-emitting diode display placed 6 ft in front of the subject at

eye level. During feedback trials, the baseline mean PTT minus the mean of the three most recent PTT intervals was added to "100." Thus, decreased PTT was associated with numerical values above "100," and increased PTT with numbers below "100." Visual feedback was continuous during feedback trials, and the display was blank during baseline trials. A second display was used to instruct the subject as to direction of control to be attempted on a given trial, and to display "points" earned for performance (a cumulative sum of the mean msec change in PTT in the instructed direction).

Procedure

The subject was seated in a comfortable chair and recording devices were attached. He was given written instructions explaining operation of the feedback display and the instructions display. When the instructions display was blank (baseline) he was to rest and sit quietly. When the number "120" appeared on the instructions display, he was to "increase the strength of his heartbeat." To the extent the subject was successful at this task, his PTT would decrease relative to baseline and thus the feedback display would show numbers above "100." When the number "80" appeared on the instructions display, he was to "decrease the strength of his heartbeat." To the extent the subject was successful at this task, his PTT would increase relative to baseline and the feedback display would show numbers less than "100." The subject was informed at the start of the experiment that he would be paid ½ cent for each "point" earned. In addition, he was asked to "breathe normally and avoid excessive physical movement" while attempting to control the strength of his heartbeat.

Following a 10-min adaptation period during which subjects were asked to rest and sit quietly, the first baseline trial was initiated. The experiment consisted of an alternating series of 8 baseline trials of 60-beat duration (feedback display blank) and 8 feedback trials of 180-beat length (continuous, beat-by-beat feedback). The 4 increase and 4 decrease trials were in randomized order, with different orders between sessions. The number of points earned were displayed for 5 sec at the end of each feedback trial, and the display was blank for 10 sec prior to the next baseline trial.

The second and third sessions were scheduled within one week of the first session, usually one day apart. These sessions were identical to the first.

Data Quantification

Trial means for the following dependent measures were computed and stored on-line: cardiac interbeat interval (IBI), PTT (duration in msec from Q-wave to upstroke of ear pulse), PEP, respiratory period or intercycle interval (ICI), and GA (number of integrator resets per min multiplied by 100). A computerized method for quantification of PEP was used (see Newlin & Levenson, 1979 for a detailed description). In this method the interval between the initial wave of the second heart sound of the PCG and the most negative point (nadir) of the differentiated ear densitogram is subtracted from PTT to the ear to determine PEP. PTT to the ear is measured from the

apex of the Q-wave of the EKG to the upstroke¹ of the differentiated ear densitogram.

Trial means for physiological measures were submitted to a $3 \times 2 \times 4 \times 2$ repeated-measures analysis of variance (ANOVA) with Session, Direction (increase vs decrease), Trials (4 pairs of baseline and feedback trials), and Control (baseline vs attempted control) as within-subject factors. Experimental hypotheses were evaluated by *a priori* *t*-tests (Kirk, 1968).

Results²

Subjects were able to produce significant bidirectional changes in PTT in the target directions accompanied by parallel changes in PEP (Table 1). Examination of Table 1 will reveal the magnitude of changes in PTT and PEP to be quite comparable overall. The parallelism between PTT and PEP was further indicated by a progressive diminution of

¹In Experiment I, the upstroke was detected using a threshold technique, thus increasing the length of PTT to the ear and subsequently the length of PEP. We² later switched to a slope detecting technique which enables a closer determination of the onset of the upstroke, thus removing the unwanted time interval between the onset of the upstroke and the threshold point inherent in the earlier technique.

²The .05 level of significance was adopted for all statistical tests reported.

magnitude of change in both variables over the course of the 3 sessions (Table 2).

Specificity of PTT change was evaluated initially by examining the Direction \times Control interactions for IBI, ICI, and GA. Only in the case of IBI were any of these interactions significant with IBI increasing during PTT increase (Table 1). Since we were particularly interested in potential ICI and GA concomitants of PTT change, a more exacting test was made to determine whether the most successful PTT controllers differed from the least successful PTT controllers in ICI or GA. A Performance (6 best vs 6 poorest PTT controllers) \times Session \times Direction \times Trials \times Control ANOVA was computed revealing no differences. Performance \times Direction \times Control $F(1/10) < 1$ for ICI and GA.

These results are interpreted as preliminary evidence that normal subjects are able to produce small changes in PTT to the ear, and that these changes are associated with concomitant PEP changes but not with changes in ICI or GA. We performed a second experiment in order to: 1) replicate these results, 2) examine changes in performance across sessions when subjects attempted PTT control in only one direction, and 3) assess ability to control PTT without feedback. Results from Experiment I are discussed more fully with the discussion of the results from Experiment II.

TABLE 1
Overall data: Experiment I

Measures	Direction \times Control		PTT Decrease Trials			PTT Increase Trials		
			Means (msec)		Change $t(10)$	Means (msec)		Change $t(10)$
	$F(1/10)$	MS_e	Baseline	Feedback		Baseline	Feedback	
PTT	37.7*	18.7	228.4	225.7	-5.30*	227.2	228.9	3.34*
PEP	40.3*	30.2	140.9	136.8	-6.33*	139.9	141.5	2.47*
IBI	12.2*	2468.3	790.0	783.3	-1.20	789.3	810.5	3.62*

* $p < .05$.

TABLE 2
Sessions data: Experiment I

Measures	Session \times Direction \times Control		Changes from Baseline (msec)					
			Session 1		Session 2		Session 3	
	$F(2/20)$	MS_e	PTT Decrease	PTT Increase	PTT Decrease	PTT Increase	PTT Decrease	PTT Increase
PTT	8.3*	11.6	-4.8	+2.9	-2.0	+1.3	-1.3	+1.0
PEP	9.9*	24.8	-6.6	+4.2	-3.8	+1.1	-3.0	-0.2

* $p < .05$.

EXPERIMENT II

Method

A second experiment was performed to extend the findings of Experiment I. Twenty male undergraduate students participated in partial fulfillment of Psychology course requirements. The apparatus and procedure were identical to those of Experiment I except subjects were randomly assigned to one of three experimental conditions: 1) No feedback—5 subjects attempted to increase and decrease PTT without feedback during 3 sessions, 2) PTT decrease—10 subjects attempted to decrease PTT with beat-by-beat feedback during 3 sessions, 3) PTT increase—5 subjects attempted to increase PTT with beat-by-beat feedback during 3 sessions.

Results

No Feedback Group

Subjects not receiving feedback were unable to increase or decrease PTT. Nonsignificant Direction \times Control interactions were found for all dependent variables.

Feedback Groups

Data for subjects receiving feedback while attempting PTT decreases and increases were analyzed using $2 \times 3 \times 4 \times 2$ ANOVAs for each dependent measure with Direction (increase vs decrease) as a between-subjects factor and Session, Trials (4 pairs of baseline and feedback trials), and Control (baseline vs attempted control) as within-subject factors. An unweighted means solution was used to handle the unequal number of subjects in the PTT increase and decrease groups.

Results indicated that only subjects attempting to decrease PTT were successful and that their PTT decreases were accompanied by parallel decreases in PEP (Table 3). The parallelism between PTT and PEP replicated a finding of Experiment I; however, the diminution in the magnitude of changes in these variables across sessions found in the earlier study was not replicated, Direction \times Session \times Control $F(2/26) < 1$ for PTT, $F(2/26) = 1.4$ for PEP.

Specificity of the PTT change relative to IBI, ICI₂, and GA was evaluated in the same manner as in Experiment 1. The Direction \times Control interaction was only significant for IBI with IBI decreasing during PTT decreases (Table 3). The more exacting tests for ICI and GA were done comparing the 5 best and 5 worst PTT decreaseers revealing nonsignificant differences, Performance \times Control $F(1/8) = 4.9$ for ICI, $F(1/8) = 2.3$ for GA. However, it should be noted that this interaction for ICI was close to significance ($p = .055$), with the most successful PTT decreaseers decreasing ICI by 1098 msec from baseline.

DISCUSSION

Subjects in these experiments produced small PTT and PEP decreases with feedback but were relatively unsuccessful in producing increases in these variables. Since PEP decreases represent *increased* levels of cardiac contractility, these findings may not be encouraging for investigators wishing to utilize voluntary control of PTT to the ear with clinical populations. It should be noted, however, that our data were obtained using normotensive subjects with no history of coronary heart disease, thus it is possible that different results would be obtained with a clinical population such as hypertensives.

The deterioration of PTT control found in Experiment I was unexpected. We did not find this effect when subjects were trained to change PTT in only one direction in Experiment II. It appears likely that this deterioration is related to the bidirectional control of PTT attempted in Experiment I, but the basis of this effect is unclear. Results from Experiment II suggest that feedback is necessary for successful control of PTT to the ear; however, the small number of subjects ($N=5$) who attempted to control PTT without feedback, and the previous demonstration of control of PTT to the wrist without feedback (Steptoe, 1976) argue against a firm conclusion.

Since PTT and PEP changed in the same direction

TABLE 3
Overall data: Experiment II

Measures	Direction \times Control		PTT Decreasers			PTT Increaseers		
			Means (msec)		<i>t</i> (13)	Means (msec)		Change <i>t</i> (13)
	<i>F</i> (1/13)	<i>MS_e</i>	Baseline	Feedback		Baseline	Feedback	
PTT	12.6*	92.5	205.1	201.2	-3.85*	203.3	204.9	1.6
PEP	5.4*	125.9	103.5	100.0	-2.96*	105.8	106.4	.5
IBI	5.0*	14.252.5	828.0	803.1	-1.98*	863.0	880.1	1.4

* $p < .05$.

and by comparable magnitudes in both Experiments I and II, we conclude that changes in PTT to the ear in this biofeedback paradigm are accounted for primarily by changes in PEP. These results are more consistent in this respect than those previously reported for PTT to the finger (Newlin, 1979; Newlin & Levenson, 1979) and may be seen as indirect evidence that voluntary control of PTT to the ear results from changes in cardiac contractility and thus, reflects alterations in beta-adrenergic drive. Additional support for this conclusion can be obtained from our finding of no significant changes in respiration rate or somatic activity in Experiment I, and marginal respiratory change in Experiment II. These findings represent greater independence of cardiac activity from respiratory and somatic variation than found in our work with voluntary control of heart rate (Levenson, 1976, 1979; Newlin & Levenson, 1978) and PTT to the finger (Newlin,

1979), or Steptoe's (1976, 1978) work with control of PTT to the wrist. The cardiac independence in the present experiment is consistent with Obrist et al.'s (1974) work with acute stress in humans, insofar as cardiac functioning under beta-adrenergic activation (indicated in the present experiment by changes in PEP) was found to be relatively uncoupled from somatic and respiratory activity. Thus, the weight of the evidence suggests that voluntary control of PTT to the ear is largely effected by alterations in beta-adrenergic drive. However, some caution is warranted since certain hemodynamic factors (e.g. cardiac preload and afterload) can disrupt the normal relationship between PEP, cardiac contractility, and beta-adrenergic drive (Newlin & Levenson 1979). Further experimentation assessing the effects of beta-adrenergic blockade on voluntary control of PTT to the ear would provide more conclusive evidence as to the basis of such control.

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