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Prestimulus Response in the Sympathetic/Parasympathetic Nervous System

by

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Introduction

Background

Vassy conducted what he conceived of as a classical, but remote, conditioning experiment in the 1960's (Vassy, 1978). In that experiment, a sender, who was sensorially isolated from a receiver, was shown a randomly determined light flash as a signal to "transmit" a telepathic warning message (i.e., a putative conditioned stimulus) to the receiver that he/she was about to experience a mild electric shock (i.e., unconditioned stimulus) in their left hand, while their skin conductance was monitored continuously in their right hand. The stimuli timing was such that if there was a conditioned response to the telepathic conditioning stimulus, it would appear before the unconditioned stimulus and a few seconds before the well-known unconditioned response (Woodworth & Schlosberg, 1961). Using manual and graphical methods of analysis, Vassy analyzed 10 sender-receiver sessions and found that six pairs were individually significant at the $p < 0.01$ level. While impressive, these results were obtained with visual meter readings and not with state of the art equipment and techniques. The experiment was repeated in 2002 with 50 sender/receiver pairs and showed significant evidence ($p < 0.01$) in the first of three series of what appears to be a telepathically conditioned response. Subsequent series, however, with some changes of equipment and protocol did not produce significant effects (Vassy, 2003). The relevance of this study is that another interpretation of such significant effects might be that receivers have been responding in advance (i.e., anticipation) to future electroshock stimuli.

Radin and Bierman began investigating, and observing significant anticipatory differential orienting effects in skin conductance responses prior to emotional and neutral photographic stimuli (Bierman & Radin, 1997; Radin, 1997a, 1997b, 2004). Radin coined the term presentiment to describe this type of pre-orienting effect.

More recently, significant anticipatory effects were observed not only with skin conductance measures but also with electroencephalogram and electrocardiogram measures (McCraty, Atkinson, & Bradley, 2004a, 2004b).

We believe there may be a complication that might muddy the interpretation of all of the presentiment results—photographic, cognitive stimuli can elicit idiosyncratic responses. For instance, a photographic stimulus that has been previously rated as having a low average affectivity may have, for some individual participants, a large affectivity, and vice versa. This reduces the contrast between arousing and calming presentations and constitutes an unwanted source of variance in these designs. This is particularly a problem in heart rate measures (Öhman, Hamm, & Hugdahl, 2000).

May and Spottiswoode adopted a different approach to remedy this the problem of increased variance. They replaced the emotional visual stimuli with acoustic startle stimuli and vastly simplified the analysis. Their dependent variable was the difference of proportions of prestimulus intervals that contained non-specific skin conductance responses (ns-SCR) prior to acoustic stimuli as compared to prior to silent controls. The null hypothesis was that these proportions should be equal. The first 105 participants of 125 reported later (Spottiswoode & May, 2003) were considered a pilot study. After trying a number of different approaches and parameters, they found mean proportions of 0.099 and 0.064 before acoustic stimuli and silent controls, respectively. Instead of an

expected ratio of 1.0, they found a ratio of 1.53 ($Z = 2.84$, effect size = 0.086 ± 0.030 , $p = 0.002$).

In a 100-participant formal follow-on study (May & Spottiswoode, In preparation) they reported proportions of 0.162 and 0.087 prior to acoustic and control stimuli, respectively, for a ratio of 1.87 ($Z = 5.08$, effect size = 0.162 ± 0.032 , $p = 1.79 \times 10^{-7}$).

To complement and extend the successful skin conductance results, we have conducted a study to examine changes in instantaneous heart rate prior to randomly-timed acoustic startle stimuli compared to changes before silent controls. There is a substantial literature on the balance between the sympathetic nervous system (SNS) and the parasympathetic nervous system (PNS) in the regulation of heart beat. It appears that heart rate responses to stimuli may be driven more, but not exclusively, by the PNS. Since skin conductance is governed by the SNS, this study will investigate the “other half” of the autonomic nervous system (i.e., the possible role of the PNS in prestimulus responses).

Anticipation of stimuli produces an orienting response as a deceleration in instantaneous heart rate. Yet, a post stimulus response might be orienting or defensive depending upon individual differences and stimulus intensity (Brownley, Hurwitz, & Schneiderman, 2000). In any event, it is clear that if there is a prestimulus anomalous anticipatory effect in the instantaneous heart rate, we can expect it to be an orienting response and be represented by a deceleration of the heart rate relative to controls. A substantial added benefit of looking for prestimulus response in heart rate is that by definition there are no “zombies—non-responders” as there were in the skin conductance studies. Therefore, we can immediately realize a 40% increase in participant inclusion over those studies.

The results of previous anticipatory studies imply a particular mechanism; individuals somehow appear to be able to respond psychophysiologicaly, in advance, of cognitive arousing or acoustic startle stimuli. However, there is an alternative interpretation that involves an anomalous anticipatory effect by the experimenter in such a way to mimic physiological responses.

Decision Augmentation Theory—DAT (May, Utts, & Spottiswoode, 1995) holds that individuals may use psi-mediated anticipation to bias their general decision making toward favorable outcomes. As applied to prestimulus response studies, the model questions who is actually playing the role of the participant. Under a pure physiological interpretation, the participant is the individual connected to the measurement hardware and who is responding, in advance to future startle or cognitive stimuli.

In the DAT interpretation, the experimenter uses his/her own psi to initiate a run such that otherwise random data from the experiment participant appear in appropriate pre-stimulus and pre-control bins to mimic physiological responses. The protocol described below will allow us to determine which model is a better fit to the data.

Method

In this section, we provide the details of the methodology for a search for prestimulus response in the human autonomic nervous system.

Hardware

Most all of the hardware necessary for this study was present from other projects; however, we did need to obtain a bio-amplifier, special leads, and a large number of pregelled standard ECG electrodes.

Software

In the past, we have used Microsoft's Visual Basic to collect physiology data and Research System's IDL language for analysis computation. This project, however, represents a major conceptual and software shift. We now use Mathworks Matlab to perform both functions.

Protocol

The following protocol was developed during an extended pilot phase and was used for the remainder of the study.

Stimuli

Rather than the proposed 24 different acoustic stimuli, we used only 1-second, 95 dB bursts of white noise for the stimuli. Controls are simple data markers in the record without any concomitant sound.

Electrode Placement

Because we were only interested in beat-to-beat timing, we initially placed one electrode on the right wrist (positive input), one on the left wrist just below the palm (instrument ground), and one adjacent to the ground on the left wrist closer to the heart (negative input). This electrode placement, however, lead to significant problems due to motion-induced muscle artifact. Sadly this initial mistake, under which significant amounts of data were collected had to be abandoned and the study restarted.

We visited with the technical people at a San Francisco Bay Area organization called Heart Math who showed us an acceptable electrode location that was mostly artifact-free and allowed for proper participant modesty. The positive electrode is position just under the rib cage on the left side; the reference electrode is positioned 2-3 cm below on the same side; and, the negative electrode is positioned just under the rib cage on the far right side of the participant.

Conditions

At the start of each run, a pre-randomized list was consulted to determine whether 16 stimuli (condition A) or 48 stimuli (condition B) would be used in the analysis.

Session Timing

We adopted a 1-minute cool-down period before the random inter stimulus interval (ISI) of 30 ± 10 seconds began. Stimuli timing within this ISI was determined by a true random number generator; however, stimulus type was determined at the start of the session and counter-balanced in sets of 16 for eight controls and eight acoustic stimuli. This assured that in condition A, there would be equal numbers of acoustic and control

stimuli. Stimulus duration was one second. The entire session lasted no more than about 45 minutes.

Feedback

At the conclusion of each session the participant was shown a complete analysis of the session.

Prestimulus Region

The prestimulus region was 3.5 seconds but it begins 1.2 s before the stimulus onset and extends by 3.5 s to 4.7 s before stimulus onset. This timing avoids a potential problem which arises because of the way heart rate is computed. The rate between any two adjacent R-waves is assumed to be constant and is defined as:

$$Pulse\ Rate = \frac{60}{\Delta t},$$

where Δt is the time in seconds between the R-waves. However, it is likely that a stimulus will occur between two beats and thus the rate *before* the stimulus will include some *post stimulus* information. Only counting heart rate data no closer to the stimulus onset than 1.2 s avoids leakage from the post stimulus region.

Analysis Details

ECG and stimulus marker data were collected at a rate of 500 samples per second and stored automatically. Using newly developed Matlab code, the raw ECG was converted to instantaneous heart rate in beats per minute. Figure 1 shows a segment of ECG data for two consecutive beats. The R-wave has been accentuated by hardware filtering.

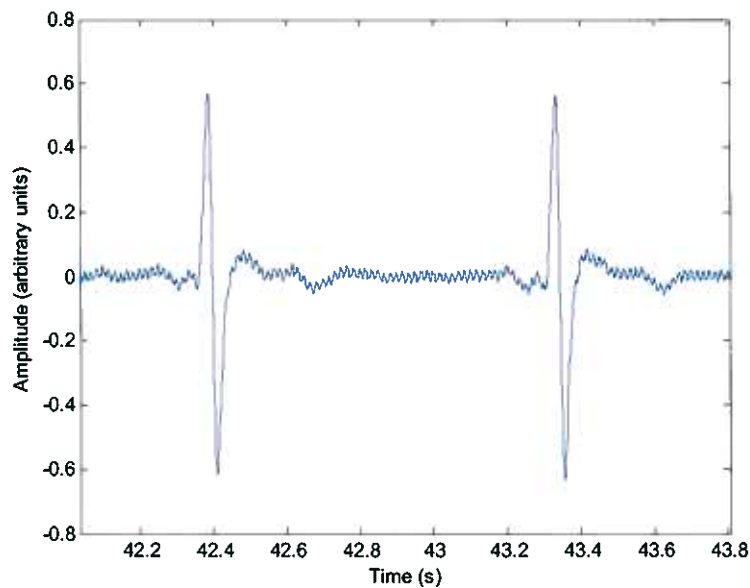


Figure 1. Sample of filtered (10-40 Hz) ECG.

The Matlab code senses where the positive peaks are and computes the difference in seconds and converts to beats per minute. Figure 2 shows a complete session of the resulting beats per minute for one participant in the formal study.

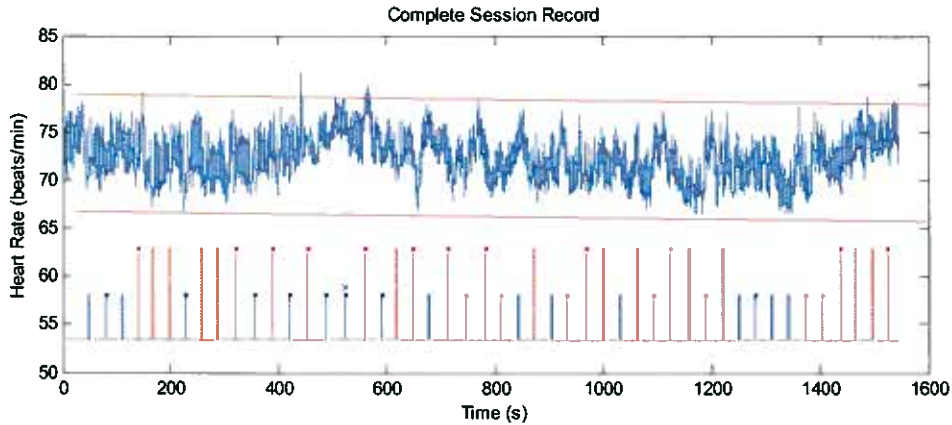


Figure 2. Beats per minute for a complete session. Long (red) markers indicate acoustic stimuli and short (blue) ones indicate controls.

By inspection, this participant produced a mean of about 75 beats per minute.

We computed regression lines, which are relatively insensitive to short anomalies. We then computed from the total standard deviation for the beats per minute data two threshold lines as:

$$T_{up/down} = (a \pm n\sigma) + b \times time.$$

The constants a and b are the intercept and slope, respectively, resulting from the regression analysis and σ is the standard deviation of the beats per minute data.

These regression lines are shown as the two horizontal red lines corresponding to the 2.5758 sigma level computed from the beats per minute session variance. Note that this corresponds to single-tailed p-value of 0.01. This means that given the variance for the sessions only a small number of beats per minute will exceed this limit due to statistical variation, alone. Thus, we used this notion as a form of artifact rejection. Note, for example, the blue 'x' above the control stimulus at approximately 550 seconds. This means that the heart rate data exceeded the predefined limit within the prestimulus region and thus this single stimulus was not used in the analysis. This served as an effective objective artifact rejection technique.

Epoch Analysis

The heart rate data from -4.7 seconds to $+15$ seconds surrounding each stimulus was obtained down-sampled to 50 samples per second and normalized with the rate at -4.7 seconds (i.e., clamped). For 48 stimuli, and two-dimensional array with 48 rows and 985 columns was created with the first 24 being for acoustic stimuli and the remainder for controls. Separate column averages were computed for each stimulus type. Figure 3 shows the best result for a single run from the formal study.

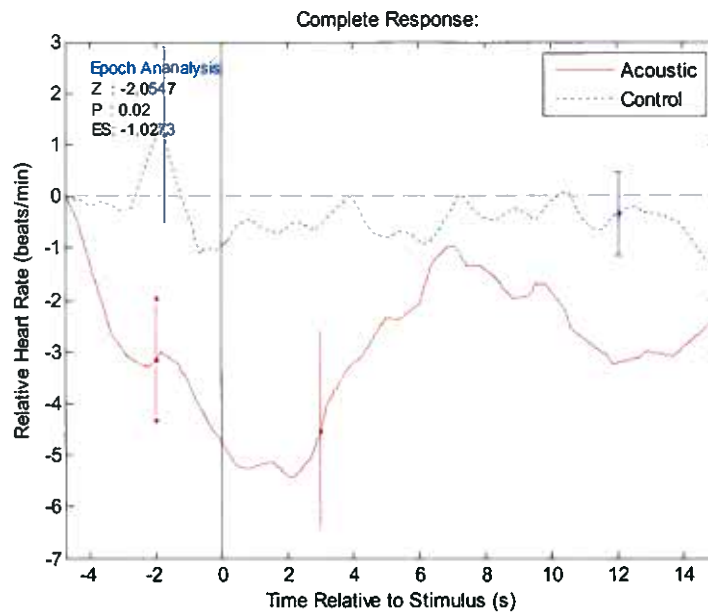


Figure 3. Epoch analysis pre- and post stimulus for a single participant.

The red trace corresponds to changes in heart rate prior to and after an acoustic stimulus and the dashed blue trace corresponds to changes in heart rate prior to and after a silent control. These data represent a near text book example of what we were looking for. The control is statistically zero throughout the region yet the stimulus data show a strong post stimulus drop of over 5 beats per second 2 seconds after the onset of the stimulus but also a substantial anticipatory drop in the prestimulus region. This might correspond to the expect orienting effect described above.

Statistical Assessment

Random permutation analysis was used to produce a statistical estimate of the prestimulus area between the two stimulus types. To accomplish this, we randomly ordered the rows in the 2-dimension array discussed above and ignored the actual stimuli types and computed the prestimulus area difference for this first permutation assuming the first half of the rows are for “stimuli” and the second half are for “controls.” We repeated this process 20,000 times to create a distribution of areas under the null hypothesis of no differential effect in the prestimulus region. Because of the central limit theorem this distribution is normal. The mean and standard deviation of this distribution was used to compute a z-score for the actual area observed such as that shown in Figure 3. In this case, $z = -2.05$ corresponding to a acoustic stimulus effect size of -1.03 . Because this was an example from Condition A of only 8 stimuli and 4 were artifact rejected, $n = 4$ stimuli corresponding to that particularly large effect size.

Results

We have collected data with 34 participants in the formal study. We analyzed the data with regard to condition A of 8 acoustic stimuli (n= 16 participants) and with regard to condition B of 24 acoustic stimuli (n = 18 participants).

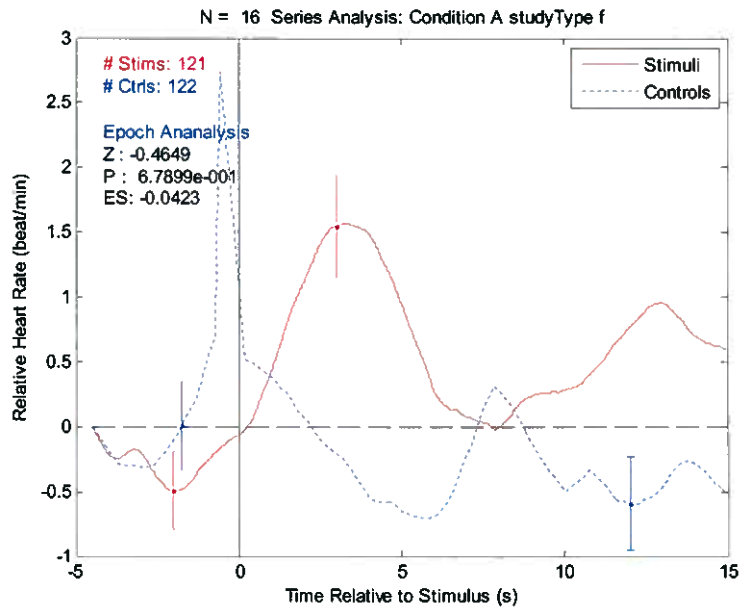


Figure 4. Pre- and post stimulus epoch analysis for Condition A of 16 Stimuli per Participant

The solid red and dashed blue traces represent the relative heart rate averages for acoustic and control stimuli, respectively. In condition A, we accumulated a total of 121 acoustic stimuli and 122 controls. The epoch analysis shows a slight decrease of heart rate prior to controls in the prestimulus region ($z = -0.46$, $p = 0.68$, $ES = -0.043$).

Figure 5 shows the results of the epoch analysis for condition B of 24 acoustic stimuli.

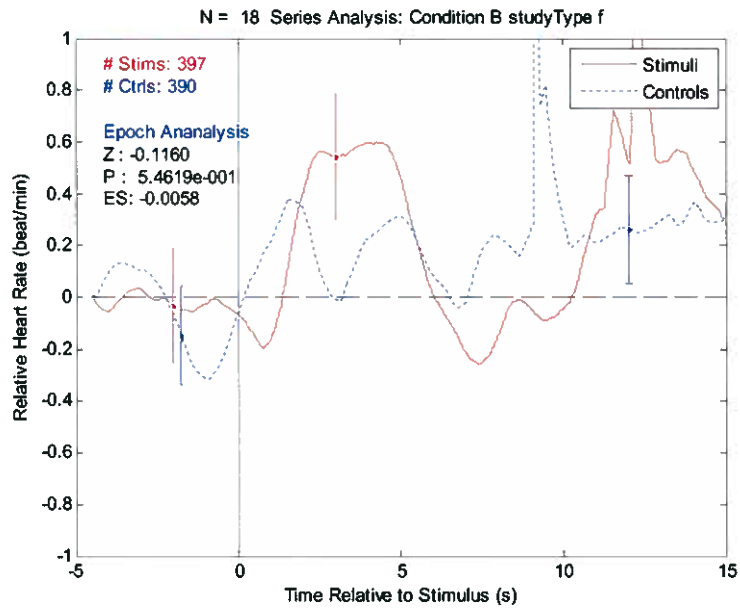


Figure 5. Pre- and post stimulus epoch analysis for Condition B of 24 Stimuli per Participant

We accumulated 397 acoustic and 390 control stimuli for the total 18 participants. Here we see that the effect size is essentially zero ($z = -0.12$, $p = 0.54$, $ES = -0.006$).

For completion we show in Figure 6 the result of the epoch analysis for the combined conditions.

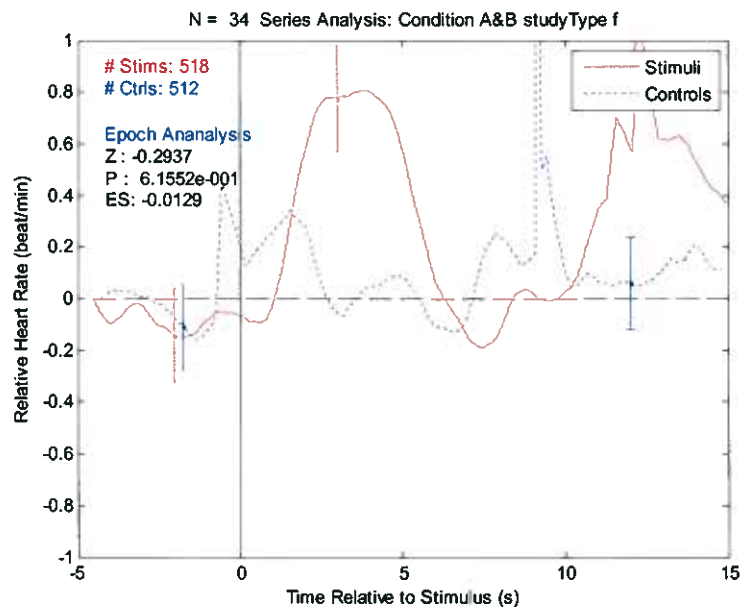


Figure 6. Pre- and post stimulus epoch analysis for Conditions A and B

The epoch analysis show essentially no effect ($z = -0.29$, $p = 0.6$, $ES = -0.01$).

Discussion

These results are quite disappointing. We used a very few participants in our exploratory data from which we formed our proposal, and we found an effect size of 0.24 in what we now call condition B of 24 acoustic stimuli. Figure 4 and the associated analysis of condition B in the formal studies shows an effect size 40 times smaller of 0.006. Thus the initial power calculations for the number of required participants was substantially incorrect.

In condition A of 16 acoustic stimuli, we found an effect size of 0.04.

In consulting with Professor (statistics) Jessica Utts, we decided to close the study because of the gross under assessment of the expected effect size. For example, if we had finished the study with the proposed total of 125 participants in condition A, we would have ended up with a z-score given by:

$$z = ES \times \sqrt{n} = 0.04 \times \sqrt{125} = 0.45.$$

If the DAT hypothesis were correct the z-score in condition B would be less by a factor of square root of 3 or 0.26.

For reasons we will discuss below, there appears not to be any prestimulus response effect in this study and thus are unable to conclude from these data whether or not Decision Augmentation Theory plays a role in prestimulus response heart rate studies.

Considerations for the Results of this Effort

It is difficult to ascribe a meaning to a null result; however, we do consider a number of potential explanations.

- Contrary to expectations, heart rate may not be subject to prestimulus response effects.
- This particular study was plagued with difficulties from its inception. Some of these issues arose because we were not well trained in heart-rate measures and analyses. The result was that we had to restart the study learning as we went. This had two important side effects. The first is that it sharply reduced the available participant pool from which we could draw, and secondly and most importantly it had a demoralizing effect on the researchers.

This last point requires further discussion. It is a well established effect that set and setting play an important role in experimental psychology and perhaps a determining role in parapsychological experiments. One of the strongest effects in the PSI literature is the so-called sheep/goat effect which may be a strong manifestation of this effect

Because of the frustrating beginning to the study and because our team is strongly accustomed to obtaining positive results, we all became discouraged and less attentive to this study. Perhaps at a minimum this contributed to the null result or, at worst, maybe "caused" the null result.

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