2017

ERPs in the context of the orienting reflex

Brett MacDonald

University of Wollongong

Recommended Citation


Research Online is the open access institutional repository for the University of Wollongong. For further information contact the UOW Library: research-pubs@uow.edu.au
ERPS IN THE CONTEXT OF THE ORIENTING REFLEX

A thesis submitted in fulfilment of the requirements for the award of the degree

Doctor of Philosophy

from

UNIVERSITY OF WOLLONGONG

by

Brett MacDonald

BA
BSc
B.Psyc. (Hons.)

School of Psychology

Faculty of Social Sciences

2017
Certification

I, Brett MacDonald, declare that this thesis, submitted in fulfilment of the requirements for the award of Doctor of Philosophy, in the School of Psychology, University of Wollongong, is wholly my own work unless otherwise referenced or acknowledged. The document has not been submitted for qualifications at any other academic institution.

Brett MacDonald

28th August 2017
**Acknowledgments**

On this journey Bob Barry has been my compass and guide. My critical thinking and approach to dealing with the diverse array of material has been experienced via Bob’s steady and considered input at the right times. On a personal level, I have enjoyed our chats and exchange of views.

Many thanks to Stuart Johnstone who modelled a gentle, good natured way of approaching research. His door, literally, is always open.

A very big thank you to Rebecca Bonfield, who spent many hours patiently and efficiently processing a wealth of peripheral vasoconstriction data.

A special thanks to Julita Purnomo, my partner, who supported me lovingly and eased many of the difficulties as they arose.

A vigorous handshake for the wonderful folk in the lab past and present, who in their own ways provided insights, skills and goodwill aplenty.
Published and Submitted Manuscripts used in this Thesis:


Submitted Manuscript:

Published Conference Abstracts:


**Note to reader:** Since each journal stipulates their referencing style, one style has been used uniformly through this work for consistency. Some changes have been made to the published manuscripts in this thesis for brevity and readability.
ABSTRACT

The early work of Sokolov established the phasic orienting reflex (OR) as primarily associated with changes in stimulus novelty – ‘newness’ *per se*. Novelty has been operationalised as reducing over re-presentations of the same innocuous stimulus (producing a lessening in response magnitude) and increasing for a changed stimulus. The OR is also sensitive to intensity and Significance (conferred by verbal instructions, “Significance” is capitalised in this thesis to distinguish it when it refers to a factor). Sokolov focused on stimulus-response patterns of autonomic measures. These measures were described as covarying, yet were found in subsequent Western work to fractionate. Only skin conductance response (SCR) demonstrated sensitivity to novelty, intensity, and Significance. Preliminary Process Theory (PPT) was proposed to accommodate these fractionating measures. PPT outlines sequential stages of processing leading to the generation of the OR. Previous research has linked various autonomic measures and EEG alpha desynchronisation to specific processing stages. Sokolov had contended that the phasic OR is linked with all the major systems of the body, so event related potentials (ERPs) should also be linked to OR-associated processing. Here, four studies employed innocuous auditory stimuli of moderate intensity, rise/fall times, and very long interstimulus intervals (ISIs). Single-trial autonomic and ERP time-locked data were collected concurrently, enabling direct comparisons of autonomic and central measures. Manipulation of novelty within subjects was common in all the studies. Temporal Principal Components Analysis decomposed ERPs into components that constituted central dependent variables. When an aspect of the stimulus-response pattern for a measure differed from SCR, that difference was statistically tested. Study 1 varied novelty within subjects solely; SCR, cardiac deceleration (ECR1), respiratory
pause (RP), and ERPs were dependent measures. Study 2 varied intensity between subjects; intensity-sensitive Peripheral Vasoconstriction (PVC) was included. Study 3 varied Significance within subjects, and Significance-sensitive cardiac acceleration (ECR2) replaced PVC. Summary Study 4 extracted data from the previous studies, combining it to increase power in an attempt to resolve cases of ‘no difference’ between a measure and SCR for habituation-based results; Processing Negativity (PN) was included to resolve inconsistent decrement findings. Elicitation of P3a prior to P3b and Novelty P3 (nP3) affirmed the independence and latency order of these Late Positive Complex (LPC) subcomponents. No single ERP matched the stimulus-response pattern of the SCR, which exhibited all aspects of the OR. The assessed measures were integrated provisionally into PPT. The data support ECR1, P1, N1-3, and PN as indexing stimulus registration; RP and nP3 as indexing novelty registration; PVC and P3b as indexing magnitude registration; and ECR2 and SW as matching the Response system. Finally, the LPC was assigned as the central analogue for the phasic OR. Further work is needed to explore dishabituation processes with respect to habituation criteria. Examination of the functional roles of the ERP components showing ambiguous results in the stages of processing may clarify their placement in PPT. Investigation into the neural substrates underlying stages of processing in PPT would be beneficial for PPT’s advancement. Finally, the expansion of PPT into other modalities and clinical applications is advocated.
Table of Contents

Certification ........................................................................................................ ii

Acknowledgements ........................................................................................ iii

Published and Submitted Manuscripts used in this Thesis ............................... iv

Published Conference Abstracts ........................................................................ v

Abstract ............................................................................................................ vi

Table of Contents ............................................................................................... viii

Abbreviations .................................................................................................... xvii

List of Tables ...................................................................................................... xx

List of Figures .................................................................................................... xxi

Chapter 1. General Introduction ........................................................................ 1

1.1 Sokolov and the OR ..................................................................................... 1

   1.1.1 Properties of the OR ............................................................................. 1

   1.1.2 Intensity and the OR ........................................................................... 2

   1.1.3 Sokolov’s Neuronal Model .................................................................. 3

   1.1.4 Significance and the OR ..................................................................... 3

1.2 Fractionation of measures linked to the unitary OR ................................ 4

1.3 Advent of PPT ............................................................................................ 5

1.4 ERPs and PPT ............................................................................................ 9

1.5 Research aims ............................................................................................ 16

Chapter 2. Study 1: Trial effects in single-trial ERP components and autonomic
responses at very long ISIs .................................................................................. 18
2.1 Abstract .................................................................................................................. 19

2.2 Introduction ........................................................................................................... 20

2.2.1 Response fractionation of the OR ......................................................... 20

2.2.2 ERPs in the OR context ........................................................................... 20

2.2.2.1 N1 ................................................................................................. 20

2.2.2.2 LPC ............................................................................................... 21

2.2.3 This study ........................................................................................................ 23

2.3 Method .................................................................................................................. 25

2.3.1 Participants ..................................................................................................... 25

2.3.2 Procedure ....................................................................................................... 25

2.3.3 Physiological recording ............................................................................. 26

2.3.3.1 Electrodermal activity ...................................................................... 26

2.3.3.2 Cardiac data ...................................................................................... 26

2.3.3.3 Respiration ......................................................................................... 27

2.3.3.4 Electroencephalogram ..................................................................... 27

2.3.4 Data extraction ............................................................................................. 27

2.3.4.1 SCRs ................................................................................................. 27

2.3.4.2 HR deceleration ............................................................................... 28

2.3.4.3 Respiratory pause .............................................................................. 28

2.3.4.4 ERPs ................................................................................................. 28

2.3.5 Statistical analysis ....................................................................................... 28

2.3.5.1 Congruence Coefficient ................................................................... 31

2.4 Results .................................................................................................................. 32
3.2.3 LPC ..........................................................66
3.2.4 This study ..................................................67
3.3 Methods ............................................................70
3.3.1 Participants ..................................................70
3.3.2 Procedure ..................................................70
3.3.3 Physiological recording ....................................71
3.3.3.1 Peripheral pulse amplitude .............................71
3.3.4 Data extraction ..............................................71
3.3.4.1 Peripheral vasoconstriction ............................71
3.3.5 Statistical analysis ..........................................72
3.4 Results ............................................................74
3.4.1 SCR ..........................................................74
3.4.2 HR ............................................................75
3.4.3 Respiratory pause ............................................77
3.4.4 Peripheral vasoconstriction ...............................78
3.4.5 ERP components ............................................79
3.4.5.1 Na (Frontal negative maximum) ......................81
3.4.5.2 P1 (Central positive maximum) .......................81
3.4.5.3 N1-1 (Central negative maximum) ....................83
3.4.5.4 PN (Frontal negative maximum) .......................83
3.4.5.5 P2 (Central positive maximum) .......................84
3.4.5.6 P3a (Central positive maximum) .....................84
3.4.5.7 P3b (Central positive maximum) .....................87
3.4.5.8 HabP3 (Parietal positive maximum on the first trial) .............87
3.4.5.9 IntP3 (Frontal maximum on the first trial) .........................88
3.4.5.10 Parietal SW (Parietal maximum) ..................................88
3.4.6 Correlations exploring patterns among measures .....................89
3.4.7 Examination of the proposed patterns ..................................90
3.4.8 Temporal comparison of PCA-derived components between studies ....92
3.5 Discussion .....................................................................................93
3.5.1 Autonomic response patterns .................................................94
3.5.2 ERP findings ............................................................................96
3.5.3 ERP component patterns .......................................................102
3.6 Conclusion ..................................................................................106
3.7 References ..................................................................................107

Chapter 4. Study 3: Significance and Novelty effects in single-trial ERP components and autonomic responses .........................................................114
4.1 Abstract .....................................................................................115
4.2 Introduction ................................................................................116
4.2.1 Background ...........................................................................116
4.2.2 Significance and the Voluntary OR .......................................117
4.2.3 Theoretical Background of PPT .............................................117
4.2.4 PPT in the context of other research fields .........................119
4.2.5 The present study .................................................................120
4.3 Methods ....................................................................................125
4.3.1 Participants .................................................................125
xii
4.3.2 Procedure........................................................................... 125
4.3.3 Physiological recording................................................... 126
4.3.4 Data extraction................................................................. 126
  4.3.4.1 Evoked cardiac response.............................................. 126
4.3.5 Statistical analysis............................................................ 127
4.4 Results................................................................................... 130
  4.4.1 SCR ............................................................................... 130
  4.4.2 ECRs ........................................................................... 131
  4.4.3 Respiratory pause............................................................ 133
  4.4.4 ERP components .............................................................. 134
    4.4.4.1 P1 (Central positive maximum)................................. 137
    4.4.4.2 N1-3 (Parietal negative maximum)............................ 138
    4.4.4.3 N1 (Central negative maximum)............................... 140
    4.4.4.4 PN (Frontal negative maximum)............................... 140
    4.4.4.5 P2 (Central positive maximum)................................. 141
    4.4.4.6 P3a (Central positive maximum)............................... 141
    4.4.4.7 P3b (Parietal positive maximum)............................... 142
    4.4.4.8 HabP3 (Frontal positive maximum)......................... 142
    4.4.4.9 FSW (Frontal negative maximum)............................ 143
    4.4.4.10 Classic SW (Parietal positive maximum)............... 144
  4.4.5 Skin conductance and heart rate levels (arousal and vigilance)............. 144
  4.4.6 Stimulus-response patterns of autonomic and ERP measures ............ 144
  4.4.7 Temporal comparison of PCA-derived ERP components between studies.
Chapter 5. Study 4: Integration of three investigations of Novelty, Intensity, and Significance in dishabituation paradigms: A study of the phasic Orienting Reflex.

5.1 Abstract ............................................................................................................ 168

5.2 Introduction ..................................................................................................... 169

5.2.1 Background ................................................................................................. 169

5.2.2 LPC as an OR index .................................................................................... 170

5.2.3 The present study ....................................................................................... 171

5.3 Methods ........................................................................................................... 172

5.3.1 Participants ................................................................................................ 172

5.3.2 Procedure .................................................................................................... 172

5.3.3 Physiological recording ............................................................................. 172

5.3.4 Data extraction ........................................................................................... 172

5.3.5 Statistical analysis ...................................................................................... 172

5.4 Results ............................................................................................................. 174

5.4.1 SCR ............................................................................................................. 174

5.4.2 Respiratory pause ....................................................................................... 174
5.4.3 ERP components ................................................................. 175
5.4.3.1 PN ................................................................................. 176
5.4.3.2 P3b ................................................................................ 176
5.4.3.3 Novelty P3 ..................................................................... 176
5.4.3.4 Classic SW ................................................................. 177
5.4.4 Novelty patterns for the autonomic and ERP measures examined here 177
5.4.5 Other results ........................................................................ 177
5.5 Discussion ................................................................................ 178
5.5.1 Autonomic findings................................................................. 179
5.5.2 ERP findings ........................................................................ 179
5.5.3 Stimulus-response patterns of the measures ............................. 181
5.5.4 LPC and the OR ................................................................. 182
5.5.5 Measure placement in PPT.................................................... 182
5.6 Conclusion ................................................................................. 183
5.7 References ................................................................................ 186

Chapter 6. General Discussion .......................................................... 191
6.1 Conclusions ............................................................................... 196
6.2 Further Research ................................................................. 196
6.2.1 The meeting of research areas and future directions .............. 198
6.3 References ............................................................................... 200

Full References ................................................................................ 201

Appendix A: Full List of Publications ........................................ 222
Appendix B: Ethics Application Approval ...................................... 223
## Abbreviations

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>AMLAB</td>
<td>Registered company name. Hardware and software for data acquisition and processing.</td>
</tr>
<tr>
<td>BPM</td>
<td>Beats Per Minute, unit of Heart Rate.</td>
</tr>
<tr>
<td>C</td>
<td>Count.</td>
</tr>
<tr>
<td>Classic SW</td>
<td>Slow Wave with positive parietal focus. Considered an OR index. The first component of the slow negative wave. Novelty dependent, possible sensitivity to intensity.</td>
</tr>
<tr>
<td>ECR</td>
<td>Evoked Cardiac Response: sum of two independent responses (initial heart rate deceleration and later heart rate acceleration).</td>
</tr>
<tr>
<td>ECR1</td>
<td>Small transient heart rate deceleration. Insensitive to novelty and intensity.</td>
</tr>
<tr>
<td>ECR2</td>
<td>Hypothetical construct estimated by Count - No Count, representing cognitive load.</td>
</tr>
<tr>
<td>EEG</td>
<td>Electroencephalogram: the recording of electrical brain wave patterns.</td>
</tr>
<tr>
<td>EKG</td>
<td>Electrocardiogram: shows electrical activity of the heart as a trace.</td>
</tr>
<tr>
<td>HEOG</td>
<td>Horizontal Electrooculogram.</td>
</tr>
<tr>
<td>HR</td>
<td>Heart Rate.</td>
</tr>
<tr>
<td>HRL</td>
<td>Heart Rate Level, a tonic measure of cardiac activity, representing prestimulus vigilance.</td>
</tr>
<tr>
<td>Hz</td>
<td>Hertz, unit of frequency.</td>
</tr>
<tr>
<td>IntP3</td>
<td>Intensity P3. Frontal distribution. Novelty and intensity dependent.</td>
</tr>
<tr>
<td>ISI</td>
<td>Interstimulus interval: time between the offset of one stimulus to the onset of the next stimulus.</td>
</tr>
<tr>
<td>LPC</td>
<td>Late Positive Complex, a broad composite waveform. Includes other</td>
</tr>
</tbody>
</table>
positive components not included in the P300.

ms  Milliseconds.
MANOVA  Multivariate Analysis of Variance.
Na  A negative middle latency response (MLR) approx. 25 ms susceptible to changes in frequency, location, and rarity. Automatic deviant detection response. Dominant at Fz.
NC  No Count.
N1  A composite waveform comprised of three ‘true’ components.
N1-1  Näätänen described as component 1 of N1 complex. In OR context, novelty independent but possibly intensity sensitive. Vertex topography. Possible transient detector.
N1-3  Näätänen described as component 3 of N1 complex. In OR context, novelty independent. Parieto-central/vertex topography. Possible transient detector.
OR  Orienting Reflex, evoked by stimulus novelty, modulated by intensity and Significance.
PCA  Principal Components Analysis, a technique to decompose a composite response into components.
PPT  Preliminary Process Theory, postulates different processing stages represented by different physiological measures. Proposed to address inconsistencies in Sokolov’s unitary concept of the OR.
PVC  Peripheral Vasocostriction, operationalised as the maximum relative decrease in pulse amplitude poststimulus. Insensitive to novelty but sensitive to intensity.
P1  Commonly showing a predominantly central distribution, triggered by stimulus onset. Possibly representing preattentive arousal.
P2  Vertex topography. Novelty and intensity independent.
P3  Composite waveform corresponding to the Late Positive Component or P300.
P3a  Anterior distribution. Novelty independent, possible intensity sensitivity.
(intensity deviation).

<table>
<thead>
<tr>
<th>Acronym</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>P3b</td>
<td>Parietal dominance. Novelty and intensity dependent.</td>
</tr>
<tr>
<td>RAAA</td>
<td>Revised Aligned-Artifact Average, a method of correcting EEG for EOG artifacts.</td>
</tr>
<tr>
<td>RP</td>
<td>Respiratory pause - increase in period of breath post stimulus. Novelty dependent, intensity independent.</td>
</tr>
<tr>
<td>SCL</td>
<td>Skin Conductance Level, a tonic measure of electrodermal activity, representing arousal.</td>
</tr>
<tr>
<td>SCR</td>
<td>Skin Conductance Response, a phasic measure of electrodermal activity. Also termed the Galvanic Skin Response. Index of the OR.</td>
</tr>
<tr>
<td>SNW/O</td>
<td>Slow Negative Wave and Orienting wave. The Orienting wave is elicited after a warning stimulus in a paired stimuli task, while the Slow Negative Wave is the identical wave in an unpaired task. Stimulus Significance appears a major eliciting factor.</td>
</tr>
<tr>
<td>VEOG</td>
<td>Vertical Electrooculogram.</td>
</tr>
</tbody>
</table>
List of Tables

Table 2.1 Trial effects and interaction tests
Table 2.2 Pearson product-moment correlation coefficients between the measures examined and the three patterns over trials
Table 3.1 Decrement, recovery, and intensity effects for ANS and CNS (ERP) measures
Table 3.2 Pearson product-moment correlation coefficients between the measures examined and the three patterns over groups and 12 trials
Table 3.3 Correlations between the 14 measures and 3 PCA-derived components. Bold represents the most extreme loading of each measure on a component
Table 4.1 Decrement, recovery, dishabituation and Significance effects for ANS and CNS (ERP) measures
Table 5.1 Decrement, Recovery, and Dishabituation for this study, along with Intensity and Significance from previous studies for selected ANS and CNS (ERP) measures
Table 5.2 Decrement, Recovery, and Dishabituation results from previous studies for measures not examined in this study; along with Intensity and Significance effects.
List of Figures

Figure 1.1. A schematic of PPT. Sequential and parallel processing of indifferent stimulus information leads to the generation of the OR from the interaction of novelty and stimulus magnitude processing. The dotted rectangles indicate measures indexing the processing of each of the modules. Physiological responses: HR decel, Heart rate deceleration; CVD, cephalic vasodilation; Resp, respiratory pause; EEG, EEG alpha desynchronisation; PVC, peripheral vasoconstriction; GSR, galvanic skin response (now SCR); HR accel, heart rate acceleration.

Figure 2.1. The square-root transformed phasic SCR as a function of trials. SE bars indicate the trial to trial variation, and the linear regression line and Coefficient of Determination for the first 10 trials are included.

Figure 2.2. Mean phasic HR response relative to the pre-stimulus level across trials. Time point variation is indicated by SE bars. Vertical scale: change in beats per minute; horizontal scale: s.

Figure 2.3. Respiratory Pause as a function of trials, with SE bars. The linear regression line and Coefficient of Determination over the first 10 trials are also indicated.

Figure 2.4. Randomly-selected examples of single trial ERPs at the midline sites for the 12 trials. Single trial data show clearly defined ERPs comparable to those from averaged data. Vertical scale: μV; horizontal scale: ms.

Figure 2.5. Averaged ERPs at the midline sites are presented for the 12 trials, with vertical eye artefacts before (VEOG uncorr) and after (VEOG corr) EOG correction. Vertical scale: μV; horizontal scale: ms; note different EOG scale.

Figure 2.6. The left panel shows the grand mean ERPs across subjects and trials at each
of the midline sites, with well-defined components labelled at Cz. The right panel
depicts the corresponding virtual ERPs derived from the sum of the first nine virtual
PCA components/subcomponents. The actual and virtual ERPs correspond very closely.
Vertical scale: μV; horizontal scale: ms.

Figure 2.7. Temporal PCA factor loadings plotted as a function of time, with
topographic head maps of the virtual temporal components displayed above.

Figure 2.8. The 9 identified PCA-derived components as a function of trial. Linear
regression lines and the Coefficient of Determination over the first 10 trials are also
indicated.

Figure 2.9. Topographic headmaps of the 9 identified components are shown for each
of the 12 trials.

Fig. 2.10. The scaled grand mean of the 16 “leave-one-out” factor loadings for the
HabP3 factor with the standard deviation of the mean at each time point displayed by
grey shading.

Figure 3.1. The square-root transformed phasic SCR as a function of trials and intensity
for condition. SE bars indicate the trial to trial variation, and the linear regression line
and Coefficient of Determination for the first 10 trials are included.

Figure 3.2. Panel A. Mean phasic HR response change relative to the pre-stimulus level
across time. Vertical scale: change in beats per minute; horizontal scale: s. Panel B.
Maximum HR deceleration over the 12 trials for both intensities. Vertical scale:
Maximum HR deceleration in beats per minute; horizontal scale: trials. Time point
variation is indicated by SE bars. Linear regression line and Coefficient of
Determination for the first 10 trials are included.
**Figure 3.3.** Respiratory Pause as a function of trials and intensity for both intensities, with SE bars. The linear regression line and Coefficient of Determination over the first 10 trials are also indicated.

**Figure 3.4.** Panel A: The mean phasic vasoconstriction as a function of poststimulus beats. Vertical scale: relative % constriction; horizontal scale: beats. Panel B: The mean phasic vasoconstriction over trials for condition. Vertical scale: relative % vasoconstriction; horizontal scale: trials. Beat point variation is indicated by SE bars. The linear regression line and Coefficient of Determination over the first 10 trials are indicated.

**Figure 3.5.** The left panel shows the grand mean ERPs across subjects and over intensities and across 12 trials at each of the midline sites, with well-defined components labelled at Fz and Cz. The right panel depicts the corresponding virtual ERPs derived from the sum of the first ten virtual PCA components/subcomponents over intensities and across 12 trials. The actual and virtual ERPs correspond very closely. Vertical scale: μV; horizontal scale: ms.

**Figure 3.6.** Rescaled factor loadings for the 10 PCA-derived ERPs. Each of the 10 factor loading peaks corresponds to the topographic grand mean headmap of that component along with its peak latency. Vertical scale: μV; horizontal scale: ms.

**Figure 3.7.** The headmaps of 10 PCA-derived ERPs from 19 sites are shown as a function of focal trials. Maximum averages at the midline for each component are marked by a grey ellipse.

**Figure 3.8.** The headmaps of 10 PCA-derived ERPs from 19 sites are shown as a function of pre-change and change trial. Maximum averages at the midline for each component are marked by a grey ellipse.
**Figure 3.9.** Pooled maximum amplitudes of the 10 PCA-derived ERPs at the sagittal regions over intensity and trials. Time point variation is indicated by SE bars. Coefficient of Determination for the first 10 trials is included.

**Figure 3.10.** Panel A: Scatterplot between mean z-scores for SCR and P3b. The open circles represent group 1 and filled circles group 2. The linear regression line and Coefficient of Determination of the total correlation over condition, and group are included. Panel B: Scatterplot between mean z-scores for HR and PN. The open circles represent group 1 and filled circles group 2. The linear regression line and Coefficient of Determination of the total correlation over condition, and group are included.

**Figure 3.11.** Topographic headmaps of PCA components from the present study and MacDonald & Barry (2014) are depicted. Latencies, % component variance, factor ranking, and Congruence coefficient are presented for comparison.

**Figure 4.1.** A schematic of PPT. Sequential and parallel processing of indifferent stimulus information leads to the generation of the OR from the interaction of novelty and stimulus magnitude processing. The smaller, lighter dashed rectangles indicate measures indexing the processing of each of the modules. The larger, bolder dashed line represents the moderation of the modules by Maltzman’s cortical set. Physiological responses: HR decel, Heart rate deceleration; CVD, cephalic vasodilation; Resp, respiratory pause; EEG, EEG alpha desynchronisation; PVC, peripheral vasoconstriction; GSR, galvanic skin response (now SCR); HR accel, heart rate acceleration.

**Figure 4.2.** The square-root transformed phasic SCR as a function of trials and levels of Significance: NC (No Count) and C (Count). SE bars indicate the trial to trial variation, and the linear regression line and Coefficient of Determination for the first 10 trials are
included.

**Figure 4.3.** Panel A. Mean phasic HR response for NC (No Count), C (Count), and estimated ECR2 (Count - No Count) relative to the pre-stimulus level across time. Vertical scale: change in beats per minute; horizontal scale: s. Panel B. Maximum HR deceleration over the 12 trials for both levels of Significance: NC (No Count) and C (Count). Vertical scale: Maximum HR deceleration in beats per minute; horizontal scale: trials. Panel C. Maximum HR acceleration over the 12 trials for levels of Significance NC: (No Count) and C (Count). Vertical scale: Maximum HR acceleration in beats per minute; horizontal scale: trials. Time point variation is indicated by SE bars. Linear regression line and Coefficient of Determination for the first 10 trials are included.

**Figure 4.4.** Respiratory Pause as a function of trials and levels of Significance: NC (No Count) and C (Count), with SE bars. The linear regression line and Coefficient of Determination over the first 10 trials are also indicated.

**Figure 4.5.** The left panel shows the grand mean ERPs across subjects, levels of Significance: NC (No Count) and C (Count), and across 12 trials at each of the midline sites, with well-defined components labelled at Cz. The right panel depicts the corresponding virtual ERPs derived from the sum of the first ten virtual PCA components/subcomponents over Significance and across 12 trials. The actual and virtual ERPs correspond very closely. Vertical scale: μV; horizontal scale: ms.

**Figure 4.6.** Rescaled factor loadings for the 10 PCA-derived ERPs. Each of the 10 factor loading peaks corresponds to the topographic grand mean headmap of that component along with its peak latency. Vertical scale: μV; horizontal scale: ms.

**Figure 4.7.** The headmaps of 10 PCA-derived ERPs from 19 sites over levels of
Significance: NC (No Count) and C (Count) are shown as a function of focal trials and overall topography. Maximum averages at the midline for each component are marked by a grey ellipse.

**Figure 4.8.** Pooled maximum amplitudes of the 10 PCA-derived ERPs at the sagittal regions over levels of Significance: NC (No Count) and C (Count) and trials. Time point variation is indicated by SE bars. Coefficient of Determination for the first 10 trials is included.

**Figure 4.9.** Topographic headmaps of PCA components from the present study and Chapter 2 are depicted. Latencies, % component variance, factor ranking, and Congruence coefficient are presented for comparison.

**Figure 5.1.** The Z-scores for autonomic and ERP components over 12 trials. SE bars indicate the trial to trial variation, and the linear regression line and Coefficient of Determination for the first 10 trials are included.

**Figure 5.2.** The restructured version of PPT including ERP components (in red). Measures displaying clear stimulus-response patterns are in bold script. Measures with ambiguous stimulus-response patterns are in smaller, non-bold script with a question mark attached.
Chapter 1. General Introduction

1.1 Sokolov and the OR

E. N. Sokolov continued the fruitful work of Pavlov on the Orienting Reflex (OR). Sokolov’s careful investigations into the factors governing OR elicitation created both interest and inspiration for western researchers of psychophysiology over many decades. Pavlov termed the OR, the “what-is-that?” reflex (Pavlov, 1927). This reflex, also known as the investigatory reflex, is sensitive to any perceivable environmental change in any modality. Since the Orienting Reflex (OR) is so acutely responsive to change, it provides the organism with a unique tool for survival. The reflex enhances the “preparedness of the whole body” and perception (Sokolov, 1963b, p 118). The OR is context and temporally relevant because it diminishes when a stimulus is repeated, i.e., as the novelty is reduced when the same stimulus is re-presented. If a different or altered stimulus is interpolated in a series of identical stimuli, the OR is reinstated (Pribram and McGuiness, 1975; Sokolov, 1963b), with the enhanced response proportional to the difference associated with the change (O’Gorman et al., 1970; Sokolov, 1960). The Cutaneo-galvanic Skin Reaction (GSR – now known as SCR) embodies all the characteristics of the phasic OR; Sokolov refers to the GSR as exemplifying the OR (Sokolov, 1963b). Traditionally, novelty has been recognised as the defining determinant of the OR, although stimulus Significance has been proposed as an essential eliciting factor (see Bernstein, 1969; Maltzman, 1979).

1.1.1 Properties of the OR

There are two important properties that characterise the phasic OR; the first is
non-specificity. Non-specificity refers to both the quality and intensity of the OR. The quality encompasses a diverse range of stimulus modalities in which the OR can be elicited. Sokolov (1963a) lists sound, light, electrical and thermal stimulation, and also increases and decreases in intensity; changes in all these will elicit the OR (Sokolov, 1960; 1963a). Selective inhibition, the second property, refers to the observed decrement in response attributed to one stimulus parameter; if a new stimulus is presented in which that aspect is changed, regardless of the aspect, the OR is elicited. Sokolov (1963a) indicated that reinstatement of the OR (recovery) was a more essential quality than extinction (response decrement), and that recovery differentiated the OR from the adaptation reflex.

1.1.2 Intensity and the OR

The intensity of a stimulus also affects the OR (Sokolov, 1963b). The association between intensity and the OR is less clear than for novelty. However, Sokolov comments, in regard to the orientation reflex, that there is a “direct dependence on the intensity of the stimulus” (Sokolov, 1963b, p 12). This relationship is supported by the linear region of the graph between 50 dB and 90 dB on the ‘J’ curve presented on page 63, presumably in the domain of low to medium intensities (p. 64). Further support for the relationship is found when it is stated that the OR is governed by the “law of intensity”, where a “stronger stimulus produces a stronger response” (p. 41). Specifically, Sokolov described the interrelationship between novelty and intensity as “the stronger the stimulus, the stronger the reaction and the more difficult the extinction” (p. 119). The dependency of the OR on intensity has been supported by subsequent researchers (Barry, 1977a, 1977b; Barry and James, 1981a; Uno and Grings, 1965). Stimulus parameters in the moderate range are required to render the stimuli innocuous; for example, a very small stimulus rise time may trigger the startle reflex.
1.1.3 Sokolov’s Neuronal Model

Sokolov proposed a cortical mechanism to address observations of decrement when a novel stimulus is re-presented (instances of habituation) or a different stimulus is presented after a series of identical stimuli. Two linked processes combined to generate the phasic OR: a neuronal model and the reticular formation (the amplifying mechanism). Stimulation triggers model formation in the cortex where aspects of the stimulus, via specific sense organs, are encoded and a representation (model) is developed and refined with stimulus repetition. When there is little coincidence between stimulus characteristics and the model, impulses linked to the discrepancy go directly to the amplifying system that generates the OR. The novelty of a stimulus was conceptualised as the difference between the current stimulus and the model, and the OR magnitude was suggested to be proportional to this discrepancy. The cortex also has inhibitory control preventing impulses reaching the amplifier system. The process of habituation of the OR results from the reducing or blocking of impulses from the cortex to the amplifying system (Sokolov, 1960).

1.1.4 Significance and the OR

Indifferent stimuli, i.e., those with no effects beyond their physical parameters alone, elicit the OR from changes in novelty, and reflect stimulus intensity. Significance is the third determinant of the OR and its influence extends beyond the physical nature of the stimulus. Sokolov indicates that verbal instructions to count may transform an indifferent stimulus into a signal stimulus, one with Significance (Sokolov, 1957, 1963b), yet the non-signal (indifferent) properties are preserved (Sokolov, 1963b). When a stimulus acquires signal Significance the response of the OR increases in
magnitude and extinction (habituation) is slower than that of an indifferent stimulus. While extinction from indifferent stimuli occurs after 3–12 re-presentations of the same stimuli, reduction related to signal stimuli occurs after dozens of re-presentations (Sokolov, 1963a, 1963b). The neuronal model, while generally accommodating physical discrepancies, lacks a mechanism for processing signal stimuli, nor does it handle empirical findings on the ‘missing stimulus effect’ (MSE); i.e., when a stimulus is omitted after a regular series of its presentation. The model would predict a strong OR at the time when the stimulus has been omitted; this has not always been found for all subjects (Barry, 1984d).

1.2 Fractionation of measures linked to the unitary OR

Sokolov nominated changes in the body movement, eyes, and head as components of the OR; autonomic changes in cardiovascular, respiratory, and GSR measures, as well as depression and enhancement of alpha rhythm, and evoked potentials (Sokolov, 1957, 1960, 1963a, 1963b). Sokolov considered the OR as a unitary phenomenon in which all the component responses covaried consistently over variable manipulations and experimental conditions i.e., showing the same directionally consistent stimulus-response patterns. Sokolov (1960, p. 191) describes the OR as a “unitary system” and later refers to the OR as “a complex combination of somatic and autonomic reactions...a complete functional system”. This has proven not to be the case (Barry 1977a, 1977b; Davis et al., 1955; Ginsberg and Furedy, 1974; Maltzman, 1979). Components of the OR display fractionation (failure to covary). The consequence of this apparent fractionation is that measures associated with the OR have differing stimulus-response patterns, i.e., reflect differing kinds of processing under different experimental conditions. Consequently, in a habituation protocol, the stimulus-response patterns of SCR and PVC would clearly differ (e.g., Barry, 1977a), yet from the unitary
OR perspective, stimuli eliciting both measures should have similar inputs into the neuronal model and similar outputs would be expected.

1.3 Advent of PPT

In the context of the OR components’ failure to demonstrate comparable stimulus-response patterns, Barry (1977a) implemented a seminal parametric study to examine the response characteristics of 5 variables employed by Sokolov and the additional evoked cardiac response (the variation of heart rate initiated at stimulus onset – heart rate deceleration [ECR1]). In order to examine habituation, subjects experienced 8 cycles of 20, 30, 40, and 50 dB tones at 1000 Hz and long ISIs; both novelty and intensity were varied within subjects. The dependent autonomic and central measures consisted of SCR, respiratory pause (RP), peripheral vasoconstriction (PVC) in the fingers, cephalic vasodilation in the temporal region of the head (CVD), EEG alpha band desynchronisation, and heart rate deceleration. Four patterns were found: ECR1 and CVD showed no trials or intensity effects, PVC failed to decrement but was sensitive to intensity, RP and alpha desynchronisation displayed trials effects but were insensitive to intensity, and SCR was both trials and intensity-dependent. This pattern of results demonstrated patent fractionation and proved consistent over subsequent studies (Barry, 1977b, 1979). The fundamental structure of PPT had taken form. A number of hypothetical stages/processes were proposed to accommodate these: Stimulus registration – ECR1 and CPV (sensitive to all stimuli regardless of novelty or intensity); magnitude (Intensity) registration – SCR and PVC (energy of the stimulus is reflected at this level of processing); Novelty registration – SCR, RP, and alpha desynchronisation (decrement reflected changes in novelty). A factor analysis confirmed the allocations of measures to these conceptual registration processes, with the inclusion of heart rate acceleration associated with a speeded motor response (Response system)
Figure 1.1. A schematic of PPT. Sequential and parallel processing of indifferent stimulus information leads to the generation of the OR from the interaction of novelty and stimulus magnitude processing. The dotted rectangles indicate measures indexing the processing of each of the modules. Physiological responses: HR decel, Heart rate deceleration; CVD, cephalic vasodilation; Resp, respiratory pause; EEG, EEG alpha desynchronisation; PVC, peripheral vasoconstriction; GSR, galvanic skin response (now SCR); HR accel, heart rate acceleration (Barry, 1979). At this stage of PPT development, SCR was grouped according to intensity registration.

Sokolov’s unitary concept was again tested but in a different modality. Thompson and Spencer (1966) had outlined a valuable set of criteria to differentiate habituation from decreased novelty and mere decrement (attributed to refractory cycle effects, adaptation, and fatigue). Barry and James (1981a) employed a visual dishabituation paradigm at very long ISIs, and manipulated novelty within subjects and stimulus magnitude between subjects. The stimuli consisted of either small or large white squares on a black background. Subjects were presented with 10 stimuli of the same magnitude, a change trial of a different magnitude (to test recovery), and five of
the original stimuli re-presented (to test dishabituation). The dependent variables included four that were used previously: SCR, ECR1, RP, and PVC; this set of measures was essential in examining the four-fold core pattern found previously. The results further established the fractionation of measures found in Barry (1977a, 1977b). Importantly the electrodermal responses demonstrated all the aspects of habituation as depicted by Thompson and Spencer (1966): decrement, recovery to change, and increased response to the original stimulus re-presentation, and intensity dependency – matching the expected phasic OR. The structure of PPT now outlined three stages of preliminary processing (Stimulus, Novelty, and Magnitude (intensity) registration) leading to the generation of the OR, indexed by SCR (as in Figure 1.1), with the additional Response system represented by HR acceleration. Dual-process theory proposed that two hypothetical processes interacted to generate the OR. The sensitisation (S) state component reflected stimulus energy and appeared relatively independent of the habituation component (H) that related to decrement rather than intensity (Thompson et al., 1979). Barry and James (1981b) suggested that PVC aligned with ‘S’ and RP aligned with ‘H’; the experimental findings affirmed these relationships. The reflexive, non-cortical mechanism of Dual-Process Theory (Barry, 1996, 2006, 2009) was dovetailed into the PPT structure. The structure of PPT followed the theoretical ordering of the processes such that stimulus registration, the encoding of stimulus properties, is a logical precursor to both novelty and intensity processing. Novelty and intensity processing were placed in parallel, since it made little sense to have the inputs and outputs of novelty and intensity processing in series. No mechanisms were proposed at this stage to account for the Significance effect. Sokolov’s neuronal model was capable of explaining only a single OR elicited from indifferent stimuli. The neuronal model was further inadequate at providing a plausible
account for the ‘missing stimulus effect’ (MSE) investigated in Barry (1984d). When a stimulus was omitted after a regular train of 20 innocuous auditory stimuli, less than half the subjects showed greater SCR activity in the MSE “window” compared to a control period. Maltzman’s involuntary/voluntary OR distinction was suggested as a suitable perspective to interpret these results (Barry, 1984d). The involuntary OR was seen as a response to indifferent stimuli, while the voluntary OR is generated when the attributes of the stimulus change are beyond the physical dimensions, and the OR is accordingly enhanced (Barry, 1984b, 1996). Instruction to silently count a series of items and report the total imparts Significance to a stimulus (Barry, 1982, 1984a, 1984b, 1984c, 2004; Lacey and Lacey, 1980; Maltzman, 1977, 1990; Sokolov, 1963a).

However, to encompass the different findings relating to experimenter instructions, a more plausible perspective posits Significance bifurcating into two dimensions: response requirements and prestimulus/poststimulus activity (Barry, 1982; 1984b; Barry and Mitchell, 1986; Tremayne and Barry, 2001). In this perspective, experimenter instructions induce a predisposing state (cortical set) that ‘steers’ attention to Significant stimuli, resulting in the voluntary OR. Directed attention manifests in prestimulus vigilance and/or poststimulus signal value, however some overlap may occur when stimulus durations are long (Barry, 1982). Barry (1988) re-analysed skin conductance responses (SCR) and heart rate (HR) data from Bernstein et al. (1985) from an OR perspective. Vigilance manipulation was reflected in both SCR and the biphasic HR (the brief initial HR deceleration and later HR acceleration). However, the difference in signal value was expressed in SCR alone. These findings indicate that some measures, commonly used in the OR context, are sensitive to the aspects of Significance. Consequently, the different cortical sets associated with prestimulus and poststimulus activity are reflected in the stimulus-response patterns of measures relevant to the task.
In both these aspects of Significance, Maltzman’s cortical sets provide the ‘steering’ mechanism, dynamic in the context of the instructions and experimental settings (Barry, 1984b, 1988; Maltzman, 1979, 1990). The voluntary OR, indexed by SCR, is elicited from vigilance or signal value manipulation, in contrast to the involuntary OR (indifferent stimuli). The voluntary OR has a slightly higher initial OR (Barry, 2004; Ben-Shakhar, 1980; Maltzman, 1990), is larger over trials (Barry, 2004; Iacono and Lykken, 1983; Maltzman, 1990; Steiner and Barry, 2011, 2014), with a slower response reduction with stimulus repetition (Barry, 2004; Ben-Shakhar, 1980; Iacono and Lykken, 1983, Maltzman, 1990). Barry (1982) included Maltzman’s cortical set into PPT as a modulating agent, and it functions there as a mechanism for directing attention. This inclusion addressed the fragility of the ‘missing-stimulus effect’ (different participants may develop different cortical sets) and the inadequacy of the neuronal model in accounting for Significance effects from the various experimental manipulations.

1.4 ERPs and PPT

Much of the research into the phasic OR used autonomic measures, and recorded individual trials at long ISIs to accommodate the slow resolution of their peak responses. Time-locked ERP responses, on the other hand, have very short latencies with poor signal/noise ratios that usually require averaging. The averaging procedure involves summing responses for the same trial position in a series of presentations over many trials (Ritter et al., 1968). This technique tends to obscure the initial responses in the initial blocks of trials and novelty effects are lessened – a considerable shortcoming from an OR perspective. The recognition of robust single-trial ERP responses, found at long ISIs, presented an alternative to the averaging procedure.
In their initial study into ERP and OR associations, Barry et al. (1992) investigated the N1 as a possible candidate for the OR in an auditory dishabituation paradigm at short ISIs. The stimulus parameters were moderate and conducive for OR elicitation. Novelty and Significance within subjects were manipulated by presenting 15 trains with 5 s inter-train intervals and, in the attend condition, participants were instructed to count high-pitched target tones. Within each train ten 60 dB tones at 1.1 s interstimulus intervals (ISIs) were administered: tones 1–7 were 500 Hz, tone 8 was 1000 Hz, tones 9 and 10 were 500 Hz. This configuration permitted an examination of ‘true’ habituation as outlined in Thompson and Spencer (1966). The N1 showed decrement over the 7 repetitions and recovery at change tone 8, but not dishabituation at tone 9, ruling it out as an OR index. Interestingly, over tones 1 to 7, a main effect of Significance was absent. Serious concerns about N1 decrement were raised, alluding to the possibility that the decrement was due to processes other than novelty reduction. Since decrement and recovery were noted for the N1, the N1 was aligned in PPT with novelty registration. The authors also surmised that the P300 may reflect a processing stage in PPT. Barry et al. (1993) addressed the possibility that the N1 results in Barry et al. (1992) were due to properties of the paradigm. The same paradigm was used as in Barry et al. (1992), but with SCR as the only dependent variable – the OR gold standard. Novel measurement procedures were needed to overcome the problem of small SCR responses at such short ISIs. The SCR exhibited all the aspects of habituation expected of the OR, so ruling out the possibility that paradigm aspects contributed to the N1 decrement and recovery without dishabituation. Budd et al. (1998) varied stimulus repetition and ISIs in an auditory dishabituation paradigm similar to that employed in Barry et al. (1992). Decrement, commonly found for the N1 at short ISIs, was explored, and examined to assess whether the decrement could be attributed to
habituation processes or the refractory period. The analyses included within subject factors of Range (early, middle, and late latency intervals) and Site (Fz, Cz, Pz, T3, and T4) to account for possible differing response patterns of the subcomponents of the N1. Two components were postulated as contributing to the decrement over the first two tones; a late component with widespread topography and Cz maximum that recovered between 3 and 10 s, and a frontal component recovering between 1 and 3 s. Generally, these results were best interpreted as refractory processing, and the longer latency N1 component was dissociated from the OR. These three pivotal studies demonstrated, from an OR perspective, the benefit of using the dishabituation protocol, SCR as the “gold standard” OR index, and a future requirement of a fine-grain examination of the component structure of ERP composite waveforms.

Rushby et al. (2005) extended the search for an ERP OR counterpart into the latency range of the Late Positive Complex (LPC, known previously as the Late Positive Component or P3[00]), as alluded to in Barry et al. (1992). The broad aim of the study was to examine trial effects of both temporal PCA-derived components and SCR in the context of the dishabituation paradigm and test whether the LPC or any of its subcomponents matched the stimulus-response pattern of the SCR. Novelty, Intensity and Significance (button press) effects, critical in attributing OR status to a measure, were assessed for the dependent variables. The procedure of averaging response means across corresponding trial positions in stimulus trains was adopted (as in Barry et al., 1992) for all measures. Since the long, fixed ISIs were 8 s, the ERPs and SCRs data could be concurrently collected. Importantly, SCR was characterised by decrement, recovery, and dishabituation, along with intensity and Significance sensitivity. The temporal PCA decomposed the LPC subcomponents in the ascending latency order of P3a, P3b, Novelty P3, and Classic SW. These subcomponents
displayed different stimulus-response profiles with respect to the variables manipulated. The authors concluded that “the LPC is an adequate EEG index of the OR” (p. 2363).

Barry and Rushby (2006) appraised the Go/NoGo task from an OR perspective, extending the interpretation of Significance into other protocols. Reports indicated NoGo responses include an anterior P3 component, while the Go responses include a more posterior P3 component. In addition, NoGo responses may be viewed as reflexive and indifferent, representing the involuntary OR; however, Go responses are related to Significance, requiring additional cortical processing, and represent the voluntary OR (Maltzman, 1990). Participants were presented with 15 target and 15 non-target tones randomly ordered at 1.1 s intervals. Sources of both the target and non-target responses were derived from low-resolution electromagnetic tomography (LORETA). Dominant sources of targets and non-target responses were identified on the first trial and these were followed over subsequent trials. Significant stimuli produced greater responses for SCR, supporting the relationship between non-target indifferent stimuli yielding an involuntary OR, and target Significant stimuli producing a voluntary OR. The P3a appeared to align with non-targets, while the P3b was elicited by targets, reflecting the involuntary and voluntary ORs respectively. The dominant sources of targets and non-targets, on the initial trials, showed considerable overlap, and the common sources decremented strongly; the decrement was suggestive of the Novelty P3 that diminishes over trials. Parallels between P3a and cardiac deceleration (involuntary processing) and P3b and cardiac acceleration (voluntary processing) were suggested as a focus in future research in terms of PPT processing stages.

Rushby and Barry (2007) explored the linkages between pre-stimulus Contingent Negative Variation (CNV) and the typical arousal index Skin Conductance Level (SCL), while the similarities between the LPC and SCR were also examined. The
passive dishabituation paradigm was the same structure used in Rushby et al. (2005), except Significance was omitted. From the PPT perspective, SCR showed the characteristic stimulus-response pattern: decrement, recovery at the change trial, and dishabituation when the original stimulus was re-presented; recovery was enhanced by an increase in intensity. The centro-parietal LPC mimicked SCR in regard to decrement, recovery, and dishabituation but an increase for recovery was only evident in the right hemisphere. These results placed the LPC as a possible central analogue to the phasic OR.

A fortuitous observation of robust single-trial waveforms for the placebo condition in Barry et al. (2008) prompted Rushby and Barry (2009) to investigate temporal PCA-derived ERPs as possible indices of the phasic OR. Very long ISIs (2 min) were employed and SCR served as the OR benchmark. The single-trial data in the placebo condition consisted of 12 innocuous significant tones at 80 dB forming a habituation series. Participants were instructed to alternately open and close their eyes for each stimulus in the series. Strong decrement in the SCR was observed, however only the Novelty P3, with global reductions over trials, matched SCR, consistent with a strong correlation between it and SCR. The composite LPC failed to show decrement, so in this study the LPC was dissociated from the OR. The Novelty P3 was nominated as a novelty index. Albeit decrement of centro-parietal LPC was absent, the centro-parietal P3b exhibited a small decrement; both these effects are consistent with the stimuli having some Significance. The latency order of P3a, P3b, and the Novelty P3 was consistent with Rushby et al. (2005).

In a unique study, Barry et al. (2011) sought to clarify the ERPs involved in OR habituation. Novelty was manipulated in this simple habituation paradigm providing single-trials data. Participants received 8 randomly presented innocuous monaural tones
with randomly varying ISIs ranging between 50 and 70 s. The dependent measures from the autonomic system were cardiac deceleration and SCR; along with PCA-decomposed N1 and LPC subcomponents, and horizontal eye movement in the direction of the ear stimulated as the behavioural representative of the OR. Behavioural orienting proved significant, observed as significant opposite EOG deflections towards the contralateral ears. The SCR was rescaled due to different SCLs for the right and left ear groups; these transformed SCRs decremented, along with Processing Negativity (PN) and Novelty P3; in contrast to heart rate deceleration, component 1 of the composite N1, and LPC. Component 3 of the composite N1, P3a, P3b, and the early and late SW displayed topographic or marginal reductions over trials, and were unlikely indices for the OR. Cardiac deceleration and component 1 of the N1 were cited as contenders for transient detection. Those measures yielding marginal or topographic reductions require further explorations to uncover the determining conditions that permit some topographic regions to reduce over trials and others to remain largely constant. The use of single-trial data derived from very long ISIs and implementation of temporal PCA analyses has been shown in this study to be efficacious in identifying novelty-sensitive components, however the identification of the N1 subcomponents requires replication from other data sets, especially when the component characteristics are estimated from their similarities to descriptions or generalisations drawn from other paradigms.

Underpinning the utilisation of single-trial data submitted to PCA is the availability of ‘clean’ waveforms, uncontaminated by sporadic ocular artifacts. Croft and Barry (2000) and Croft et al. (2005) outlined a procedure to account for and remove irrelevant eye movements in the EEG. This procedure optimises the influence of systematic variation in single-trial data, critical in extracting credible outcomes from PCA. In a comparison study, Croft et al. (2005) demonstrated that their technique was
superior to other extant correction techniques. The ocular rejection alternative, omission of the affected trial, or clustering consecutive trial responses into sequential groups (see Roth et al., 1984) becomes unviable when identification of changes in novelty in the first few trials is paramount.

MacDonald et al. (2012) exploited single-trial data to explore stimulus-response patterns of autonomic and central measures. The relationship between the autonomic (SCR, HR deceleration, and RP) measures and ERPs (N1 and LPC) was explored from a PPT perspective. An alternating series of 60 and 80 dB innocuous tones was presented to participants, who were randomly assigned to one of two counterbalanced groups based on the initial tone intensity. The SCR demonstrated both trials and intensity main effects, but both of these effects were absent from HR deceleration. A linear response decrease over trials marked decrement for RP, but no intensity effect was found. The N1 showed no main effect of trials but overall intensity sensitivity, however a reduction of midline responses in the left hemisphere over trials was found, suggesting the differential activity of N1 subcomponents. The LPC demonstrated no trials or intensity main effect, therefore it could not be considered an OR analogue. This investigation uncovered the presence of complex interactions for N1 and LPC, highlighting the need for EEG decomposition techniques such as PCA, to tease out subcomponents differentially sensitive to stimulus parameters, task demands, and paradigm characteristics.

Barry et al. (2013) revisited the issue of measure fractionation within and between autonomic and PCA-derived ERPs using single-trial data and these measures’ association with habituation of the phasic OR. This study followed the same paradigm used in MacDonald et al. (2012) except PVC was included. Novelty and intensity within subjects variation was examined but with a larger participant pool. Participants received
an alternating sequence of 60 and 80 dB tones in a habituation series. The study sought to affirm previous reports regarding SCR, HR deceleration, RP, and PVC (not included in MacDonald et al. 2012) and to examine autonomic and PCA-derived ERP matches for stages of processing in PPT. Rather than using peak-picking procedures that take considerable time, a novel, more convenient and efficient method was employed. Single-trial data were submitted to temporal PCA to produce virtual ERPs, free from the subjective evaluation of peaks commonly used. The comparison of the grand means from the raw data and the sum of virtual ERPs demonstrated a good fit at the midline sites. The predicted fractionation was found: HR deceleration showed no trials or intensity effects, PVC failed to decrement but was intensity dependent, RP decremented considerably but no intensity effect was evident, and SCR displayed a substantial trial and intensity main effect. The N1 and P3a showed no trials effect but intensity dependency, generally consistent with previous reports; these were tentatively placed as intensity representatives in PPT. The P3b proved to be intensity dependent but increased over trials. Surprisingly, Novelty P3 showed no trial effect but an inverse intensity effect i.e., greater response for the 60 dB tone. Both trial and intensity effects were absent for the SW. In light of some of these unusual findings, the authors postulated that the alternating series of different intensity tones may have reduced the novelty aspect of the stimulus sequence. A within subjects variation of novelty and between subjects variation of intensity was advocated to test these anomalous findings.

These studies underpinned and led to the present doctoral research programme.

1.5 Research aims

The broad aims of this doctoral thesis were to:

1. Explore the stimulus-response patterns of the autonomic and central measures
when novelty, intensity, and Significance are manipulated in auditory dishabituation paradigms at very long ISIs;

2. Match PCA-derived ERP components to existing autonomic measures and align the measure pairs to specific preliminary, OR, or post-OR processing in PPT;

3. Address the question of whether the LPC or any single PCA-derived ERP component can represent the phasic OR.

All stimuli are in the moderate range of intensity and all are considered innocuous.

Generally, the manipulation of the OR determinants follows a logical, methodical strategy across the four studies included here. Study 1 manipulates novelty within subjects. Study 2 manipulates novelty within subjects and intensity between subjects. Study 3 manipulates novelty and Significance within subjects. Study 4 re-examines ambiguous novelty results from these with a greater participant pool and attempts to place the matching measures into PPT. The same physical stimulus parameters, such as rise/fall time and duration, and ISI, are used in all the dishabituation paradigms. This structure permits replication in regard to novelty, since the stimuli are delivered under the same physical conditions, within-subjects novelty is varied in all the studies, and every study has an indifferent condition.
Chapter 2. Study 1: Trial effects in single-trial ERP components and autonomic responses at very long ISIs

Published as:

2.1 Abstract

Single-trial data from autonomic and ERP measures were used to capture the rapidly decreasing initial responses characteristic of the Orienting Reflex (OR) to repeated stimuli. Stimulus-response patterns were compared to determine central analogues of autonomic indices of processes leading to the OR, and the OR itself. Participants were presented with 12 indifferent tones in an auditory dishabituation paradigm. Principal Component Analysis (PCA) decomposed EOG-corrected ERP data. Response patterns of ERPs, cardiac, and respiratory responses were compared to the phasic skin conductance response (SCR). SCR decremented over trials, recovered on the change trial, and dishabituated to the representation of the standard, meeting the formal definition of habituation required of the OR. The evoked cardiac response showed no trial effects. Respiratory pause (RP) decreased linearly over trials, recovering marginally on the change trial. Nine identifiable ERP components were extracted: P1, N1-3, N1-1, Processing Negativity (PN), P2, P3a, P3b, a novelty-sensitive P3 component (labelled HabP3), and the Slow Wave (SW). P3b and SW showed decrement over trials, but with no recovery, HabP3 showed decrement and increased response on the change trial, while the P1, N1 subcomponents, P2 and P3a were insensitive to novelty. Stimulus-response patterns of the RP and HabP3 suggest sensitivity to novelty processing, while the P1, N1-3, N-1, PN, P2, P3a and cardiac deceleration appear to mark processing prior to novelty, such as stimulus transient detection (cardiac deceleration) and/or intensity processing. This study supports predictions of Preliminary Process Theory, demonstrating fractionation of 3 autonomic and 9 ERP components to Novelty, and disconfirming the unitary nature of the OR.
2.2 Introduction

2.2.1 Autonomics in the OR context

The General Introduction of Chapter 1 contains an account of the fractionating autonomic measures’ stimulus-response patterns developed over many studies. Specifically, SCR has reliably demonstrated decrement, recovery, and dishabituation. Heart rate deceleration and peripheral vasoconstriction have failed to decrement. Respiratory pause has shown decrement in various studies, however, recovery has only been found in a visual dishabituation paradigm (Barry and James, 1981a). Predictions about these measures have been made based on OR-styled paradigms in the moderate range of physical stimulus parameters and intensities.

2.2.2 ERPs in the OR context

If the PPT model of processing involved in OR elicitation and habituation is to have relevance beyond the autonomic data that generated it, parallels with ERP components should be apparent. In recent studies from our laboratory, some of the preliminary processes of PPT have been linked to ERP components. These studies used simple stimuli in the moderate range of intensity within either habituation or dishabituation paradigms, often with the inclusion of SCR as the OR benchmark.

2.2.2.1 N1

Rushby and Barry (2009), in a simple habituation study, presented 12 identical innocuous tones to subjects at very long ISIs, with ERPs and SCR measured. The vertex-dominant N1 found in the raw data and as a PCA-derived component failed to decrement, in contrast to the substantial decrement shown for SCR. Barry et al. (2011) also reported the lack of trial decrement for both the N1-3 and N1-1 subcomponents.
Lawrence and Barry (2009) manipulated intensity and cognitive load and found an N1 peak with fronto-central topography sensitive to intensity but unaffected by cognitive load.

2.2.2.2. LPC

Early parallels were drawn between the OR and the Late Positive Complex (LPC or P3[00]), an ERP complex sensitive to physical and contextual aspects of the stimulus (Donchin et al., 1984). Generally, the LPC has been interpreted as marking the updating of the stimulus context, and occurring on the first presentation of a stimulus regardless of relevance (Donchin, 1981). The LPC has 4 components separable using Principal Component Analysis (PCA): P3a, P3b, Novelty P3, and the Slow Wave (SW), each differing in topography and sensitivity to task demands (Barry et al., 2011; Barry et al., 2013; Donchin et al., 1984; Sutton and Ruchkin, 1984). Traditionally the OR has been examined employing autonomic measures such as heart rate and electrodermal activity, predominantly in habituation protocols at long ISIs, with single-trial data that captures the brief, transient responses characteristic of the phasic OR (McDonald et al., 1964; Siddle, 1985; Zimny and Schwabe, 1966). In contrast, ERP research on the OR has almost exclusively been conducted utilising variations of the oddball protocol (Friedman et al., 2001). In this protocol shorter ISIs necessitate signal averaging to reduce the inherent noise, but this averaging confounds identification of decrementing phasic responses. Except for the first in a sequence of stimuli and the role of ‘Significance’, oddball (classical, three-stimulus, and novelty) and habituation/dishabituation paradigms differ in stimulus context. This context is at least partially framed by the associations between stimulus events and provides a background for defining a component in terms of topography, morphology, and response to experimental variables in a specific paradigm (Donchin et al., 1978; Spencer et al.,
Consequently, the attributes of an ERP component/subcomponent will be ‘fixed’ by paradigm demands, although some ERPs appear to have equivalents in different paradigms (e.g., the oddball target P3b and the LPC subcomponent in a habituation paradigm sensitive to Significant stimuli; Barry and Rushby, 2006). The LPC subcomponents P3a and Novelty P3 have been identified in traditional and novelty oddballs respectively, the latter predominately to non-attended complex, environmental, auditory stimuli (Combs and Polich, 2006; Debener et al., 2005; Spencer et al., 2001). Spencer et al. (2001) employed classic and novelty oddballs under attend and ignore conditions, and reported a P3a, Novelty P3, and P3b subcomponent factors from a Spatio-Temporal PCA. The authors proposed that the P3a is probably a sum of the Novelty P3 and P3b, and claimed the P3a and Novelty P3 have never been recorded from the same subjects. In the oddball context, the P3a and Novelty P3 are often considered to be the same entity (Simons et al., 2001).

In some habituation/dishabituation studies employing simple, auditory stimuli, the P3a and what has been termed the ‘Novelty P3’ have emerged from PCA as independent components. The P3a appears equivalent to the fronto-central component elicited prior to the target P3b in an oddball task, but the ERP response to novel stimuli in a habituation task has consistently been elicited after the P3b, often displaying a posterior distribution (Barry et al., 2011; Rushby et al., 2005; Rushby and Barry, 2009). Acknowledging the role of stimulus context and paradigm differences, we will here label this ERP component as ‘HabP3’.

Rushby et al. (2005), using moderate stimulus parameters at long ISIs, reported decrement and recovery for P3b, Novelty P3 (the Novelty P3 has not always shown decrement; Barry et al., 2013), and early SW, but only decrement for the P3a. The early SW was the only LPC subcomponent to exhibit all the aspects of habituation. The P3a
and P3b reflected stimulus intensity, while the P3b and early SW were sensitive to Significance; but the ‘Novelty P3’ was insensitive to intensity and Significance.

2.2.3. This study

Previous studies generally support the notion that the LPC adequately represents the OR, yet no subcomponent reflects manipulations of novelty, intensity, and Significance demanded of an OR index. The N1 and HR deceleration appear to closely align with transient detection. The HabP3, usually displaying trial effects, mirrors the RP that indexes novelty. The P3b and SW associations are unclear, but appear to be linked with aspects of the voluntary OR.

In this dishabituation study using simple stimuli, we further explore linkages between central and autonomic measures to consolidate previous findings in relation to processes leading to the OR, and the OR itself. This study extends previous work by including RP, shown to be sensitive to novelty, and utilising PCA to search for possible ERP analogues for RP. Stimulus-response patterns are examined in single-trial autonomic and ERP data to assess similarities between the two systems from the perspective of PPT. It is predicted that the SCR will exhibit the typical OR response pattern: decrease with stimulus repetition, increase to the change stimulus, and relative increase to representation of the original stimulus. The phasic HR deceleration will remain relatively constant over all trials, marking occurrence of the stimulus event. In contrast, a substantial respiratory pause should occur at the first trial and decrease with subsequent stimulus presentations, show an increased response for the change stimulus, but no increased response for the dishabituation trial (Barry and James, 1981b).

The indifferent stimuli (without an effect over and above their physical parameters) in the moderate range of intensity will generate ERPs implicated in the
involuntary OR. The PCA will use a broad latency range to capture these. Expectations for many components are based largely on short interstimulus interval (ISI) paradigms outside the OR context. The P1, elicited by stimulus onset and reflecting preattentive arousal, is expected to be elicited by the first stimulus and may diminish over trials (Fruhstorfer et al., 1970; Gillette et al., 1997; Pratt et al., 2008), but an enhanced response is not foreseen for the change stimulus (Viswanathan and Jansen, 2010). The N1 at these long ISIs, and reflecting transient onsets/offsets (Barry, 2006; Näätänen, 1988; Näätänen and Picton, 1987), should be relatively insensitive to stimulus repetition (Rushby and Barry, 2009; Barry, 2009; Barry et al., 2011, 2013). The PN is expected to be elicited from the first stimulus, representing a reflexive attention switch to novel aspects of the stimulus (Barry et al., 2011), and exhibiting a large response at these longer ISIs (Picton et al., 1976), could show decrement with trials (Barry et al., 2011). The P2, sensitive to attention, should not show decrement over trials (Crowley and Colrain, 2004; Rushby and Barry, 2009). A P3a, responsive to salience (Goldstein et al., 2002) and functioning as an involuntary attention switch (Dien et al., 2004), should be insensitive to novelty at these longer ISIs and constant intensity (Rushby and Barry, 2009). A parietal P3b marking an attentional shift, particularly on the first stimulus (Ritter et al., 1968), and responsive to uncertainty (Sutton et al., 1965), could demonstrate some response decrement with trials (Rushby et al., 2005; Barry et al., 2011; but not Rushby and Barry, 2009; Barry et al., 2013), show some increased response to the change stimulus (Rushby et al., 2005), and remain unaffected by the representation of the original stimulus (Rushby et al., 2005). The ‘Novelty P3’/HabP3 subcomponent of the LPC has been isolated by PCA and is responsive to the initial presentation of a simple stimulus – the ‘newness’ of a tone (Barry, 2009, Barry et al., 2011). The HabP3 is expected to diminish over trials (Rushby et al., 2005; Barry and
Rushby, 2006; Barry et al., 2011; but not Barry et al., 2013), exhibit some recovery (Rushby et al., 2005), and some dishabituation (Rushby et al., 2005). The SW has been associated with novelty (Loveless and Sandford, 1974), so trial effects would be expected with stimulus repetition (Zimmer and Demmel, 2000; Rushby et al., 2005; but not Barry et al., 2011, 2013), with some response recovery, but no increase for the dishabituation trial (Rushby et al., 2005).

2.3 Methods

2.3.1 Participants

Sixteen university students participated in an experimental session as one means of fulfilling a course requirement (age 20–24, mean 21.4 years; 10 females; 13 right-handed). The procedure was explained and written consent was obtained in accordance with a protocol approved by the joint South East Sydney and Illawarra Area Health Service/University of Wollongong Human Research Ethics Committee, in line with the Declaration of Helsinki (WMO, 1996). Participants were required to complete a demographic and screening questionnaire, and only those with normal hearing participated. Individuals with a history of seizures, psychiatric illness or severe head injury were excluded, as were those currently taking psychoactive drugs.

2.3.2 Procedure

Participants were seated in a dimly-lit, sound attenuated, air-conditioned testing booth with a fixation cross displayed on a computer monitor placed at a distance of 1.5 m. Once comfortably seated, the participants were instructed that they would occasionally hear sounds over the headphones, but that there was no task in relation to them. They were asked to focus their eyes on the fixation cross presented on the monitor screen, try not to move or blink, and to stay relaxed. Prior to the presentation of tones,
participants engaged in an eye calibration task involving a series of vertical, horizontal, and blink eye movements (Croft and Barry, 2000).

Auditory stimuli were 1000 and 1500 Hz tones at 80 dB intensity, with a duration of 50 ms (15 ms rise/fall times) and a random, variable ISI of 50–70 s.

Participants received 10 tones at one frequency (either 1000 Hz or 1500 Hz), a change trial at the other frequency, and the original tone presented on trial 12. Zero, 1, or 2 tones were added randomly for each participant to reduce subject communication and expectation concerning the stimulus number. Participants were randomly assigned to one of two counterbalanced groups, based on the frequency of the first tone, so the different groups had complementary tones in the series. The first twelve tones were used for analysis.

2.3.3 Physiological recording

A digital signal-processing hardware and software package from Associative Measurement (AMLAB II) was used for the acquisition and storage of data.

2.3.3.1 Electrodermal activity

Skin conductance was recorded from silver-silver chloride (Ag/AgCl) electrodes, filled with electrode paste of 0.05 M NaCl in an inert ointment base, placed on the distal volar surface of digits II and III of the non-dominant hand. The electrode pair forming part of the input circuit was excited by a constant voltage of 0.5 V, and the current change representing conductance was recorded using a DC amplifier. Skin conductance was sampled continuously at 64 Hz.

2.3.3.2 Cardiac data

The electrocardiogram (EKG) was recorded from pre-jelled disposable Ag/AgCl
electrodes positioned at mid-sternum and over the third rib on the left mid-axillary line. The signal was amplified × 10,000, and sampled by a 16 bit A/D converter at 512 Hz.

2.3.3.3 Respiration

Respiratory activity was recorded using a piezoelectric respiration transducer mounted on a Velcro belt (Pneumotrace II, UFI). Respiration was sampled continuously at 64 Hz.

2.3.3.4 Electroencephalogram

EEG was recorded from 19 scalp sites using an electrode cap referenced to linked ears and grounded by the cap electrode located mid-way between Fpz and Fz. Vertical eye movement (VEOG) was monitored with tin cup electrodes placed 2 cm above and below the left eye. Horizontal eye movement (HEOG) was monitored with tin cup electrodes placed on the outer canthus of each eye. Impedance was less than 5 kΩ for cap electrodes and less than 3 kΩ for EOG and reference electrodes; care was taken to balance reference impedances. Scalp potentials were amplified × 20,000, and EOG × 5,000, with a bandpass down 3 dB at 0.01 and 30 Hz, and digitised at a rate of 512 Hz. The EEG data were EOG corrected using the RAAA EOG Correction Program (Croft and Barry, 2000; Croft et al., 2005).

2.3.4 Data extraction

2.3.4.1 SCR

The raw SCR traces were segmented offline in 8 s epochs commencing at stimulus onset. Each response was quantified for each subject, for each trial (1–12), as the difference between the value obtained at response onset within the 1–3 s post-stimulus interval (Barry, 1990) and the maximum value of the subsequent peak. These
data were then square-root transformed to reduce the skew typically associated with small SCRs (Barry and Sokolov, 1993).

2.3.4.2 HR deceleration

EKG was analysed using a locally produced R-wave peak detection program to compute R-R intervals in ms. Measures of cardiac activity were calculated in terms of mean values of HR for 0.5 s intervals relative to event onset (Velden and Wölk, 1987), with each epoch of data commencing 2 s before stimulus onset and ending 5 s after stimulus onset. Phasic responses were examined in terms of the change in HR from the immediately-prestimulus level at each trial.

2.3.4.3 Respiratory pause

The phasic change in respiration was quantified as the difference in time between the cycle containing the stimulus onset and the prestimulus cycle (inspiration-inspiration), divided by the duration of the prestimulus cycle.

2.3.4.4 ERPs

The continuous data were quantified offline using Neuroscan software (Compumedics, Version 4.3) for the 100 ms pre- to 1000 ms post-stimulus period, using the immediately-prestimulus 100 ms as baseline.

2.3.5 Statistical analysis

A single PCA was used to identify ERP components from single-trial data in the time range of 0–500 ms from 19 scalp locations. The full number of data files (16 subjects x 12 trials x 19 sites = 3648) were submitted to temporal PCA utilising Dien’s ERP PCA toolkit (v. 2.23; Dien, 2010) in Matlab. The PCA used a covariance matrix
and the number of components retained more variance than random noise (Parallel Test; Horn, 1965). Kaiser normalisation was employed along with Varimax rotation to maintain orthogonality and clarify interpretation. Virtual ERP component amplitudes based on factor scores were used for analysis.

For this within subject dishabituation study, virtual ERP component amplitudes at the site of maximum response and all autonomic measures were separately examined for response decrement in a repeated measures multivariate analysis of variance (MANOVA) with the factor Trials (for trials 1 to 10). Within Trials, linear trend was examined to assess decrement. Separate MANOVAs were conducted to examine change trial responses (recovery: trial 11 vs. 10) if decrement over trials was found. Similarly, responses for the representation trial (dishabituation: trial 12 vs. 10) were analysed only if recovery on the change trial was significant. The analysis of the HR response over a 2.5 s epoch also included a Time factor. Planned comparisons examined simple (linear, quadratic, and cubic) trends over Time to define the HR response. Each analysis of the component ERP measures from the nine central sites additionally included examination of topography, with coronal plane [left (F3, C3, P3), midline (Fz, Cz, Pz) and right (F4, C4, P4)] and sagittal plane [frontal (F3, Fz, F4), central (C3, Cz, C4) and parietal (P3, Pz, P4)] as repeated-measures factors for each component. Planned contrasts within the coronal plane compared the left (L) vs. right (R) regions, and the midline (M) vs. the mean of the left and right sites. Within the sagittal plane, frontal (F) vs. parietal (P) regions, and central (C) sites vs. the mean of the frontal and parietal sites, were analysed. These orthogonal planned contrasts provide optimal information on the topographic distribution of the amplitude of each component.

Separate repeated measures MANOVAs were conducted to examine differences between SCR and HR, SCR and RP, along with SCR and the virtual ERP components.
Specifically, the benchmark S-R pattern: decrement, recovery, and dishabituation is expected to be demonstrated by the phasic SCR. If a measure differed from the SCR pattern then the differing aspect of the pattern was tested with a repeated-measure MANOVA for that measure and SCR using Z-scores for each subject over the relevant trials, and the factor interaction: Measure $\times$ decrement/recovery/dishabituation was examined. This is subsequently termed the Interaction Test. If the test yielded a significant interaction, this confirmed that the measure’s pattern aspect differed from that same aspect of the SCR pattern; subsequent pattern testing was discontinued. If the interaction was non-significant, this was interpreted as that aspect of the pattern not differing significantly from SCR and subsequent testing ensued for the next aspect of the S-R pattern. For ERPs, this was conducted on a subject’s Z-scores representing amplitudes at the site of maximum response. Also, for negative components, the polarity was reversed to ensure decrement, recovery, and dishabituation were tested.

Since all contrasts were planned and there were no more of them than the degrees of freedom for effect, no Bonferroni-type adjustment to $\alpha$ was necessary (Tabachnick and Fidell, 1989). Also, Greenhouse-Geisser type correction was not necessary because single degree of freedom contrasts are not affected by the violations of sphericity assumptions common in repeated-measures analyses of physiological data (O’Brien and Kaiser, 1985). All tests reported have (1, 15) degrees of freedom, and effect sizes (partial $\eta^2$) are indicated. We report results from a large number of dependent measures, increasing the frequency of Type 1 errors. Since the probability of Type 1 error is the same for each measure ($p = .05$), 1 false positive may occur in 20 significant test results for a given measure when there is no true effect present. Another set of 20 significant results from another independent measure may also have the frequency of false positives as 1 in 20. For both measures together, the frequency may
be 2 false positives, however the probability of a false positive across both measures is 2 in 40, i.e., it remains at .05. Howell (1997) argues that alpha level adjustment cannot be used to control this frequency of Type 1 errors. Howell (2002) comments that \textit{a priori} tests are undertaken with per comparison error rate rather than family error rate (associated with post hoc tests). In some areas such as neuroimaging, genetic research, and evolutionary genomics research where 100s of tests may be carried out, there is no universally accepted method of dealing with multiple comparison testing (McDonald, 2014). McDonald (2014) points out the vagueness of the term ‘family’ in regard to test grouping. For example, a researcher decided to conduct 25 tests in a particular paradigm; if the experiment was performed 2 years later as a replication using 25 tests on the same variables, would the first experiment need to be reanalysed with adjusted familywise correction based on 50 tests? The cost of guarding against possible false positives by the Bonferroni correction is to increase the chance of false negatives, possibly missing out on finding a significant effect. Although this strategy may appear as a limitation, it could also be viewed as a reasonable balance of the likely occurrences of false positives and negatives in the context of expensively acquired and processed data. The reader should be mindful of the fact that no Bonferroni corrections have been made in the analyses performed in this thesis, consequentially significant results should be treated cautiously.

\textbf{2.3.5.1 Congruence Coefficient}

The Congruence Coefficient provides an index of similarity (Davenport, 1990) or matching (Nesselroade and Baltes, 1970) based on loadings of interpretable PCA components over time intervals (variables) in a latency range from different data sources. The form of the Congruence Coefficient is similar to Pearson’s product-moment correlation, both reflect the degree of relatedness, however, the former is based
on a ratio scale rather than an interval scale (Davenport, 1990). In an ERP context, the Congruence Coefficient reflects the stability and consistency of compared components over studies. If a covariance matrix is used in the PCA-decomposition, the factor loadings reflect ERP amplitudes over experimental conditions (Kayser and Tenke, 2003), latency, rise/fall times, but not topographical likeness (Barry et al., 2014). Lorenzo-Seva and ten Berge (2006) suggested that $r_c$ within the range of $0.85 - 0.94$ represents “fair similarity”, while $r_c > 0.95$ represents “good similarity”, i.e., an equivalence of the compared components.

2.4 Results

2.4.1 SCR

The mean SCR waveform showed onset latency of approx. 1.7 s and peak latency of approx. 3.8 s. The decreasing level of response across trials was characterised by a linear trend ($F = 32.32, p < .001, \eta^2_p = .683$), evident in Figure 2.1. The SCR showed both recovery to the change stimulus (11 > 10: $F = 7.74, p = .014, \eta^2_p = .340$), and dishabituation to representation of the original stimulus (12 > 10: $F = 10.65, p = .005, \eta^2_p = .415$); see Table 2.1 that summarises the trials, recovery, and dishabituation results for each measure, along with the measure vs. SCR Interaction Test for the three pattern aspects.
Figure 2.1. The square-root transformed phasic SCR as a function of trials. SE bars indicate the trial to trial variation, and the linear regression line and Coefficient of Determination for the first 10 trials are included.

Table 2.1 Trials effects and interaction tests.

<table>
<thead>
<tr>
<th></th>
<th>Decrement</th>
<th>Measure vs. SCR Decrement</th>
<th>Recovery</th>
<th>Measure vs. SCR Recovery</th>
<th>Dishabituation</th>
<th>Measure vs. SCR Dishabituation</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Autonemics</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR</td>
<td>—</td>
<td>*</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RP</td>
<td>*</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SCR</td>
<td>***</td>
<td>*</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>ERPs</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>P1</td>
<td>—</td>
<td>***</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>N1-3</td>
<td>—</td>
<td>***</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>N1-1</td>
<td>p = .088</td>
<td>*</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PN</td>
<td>—</td>
<td>**</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>P2</td>
<td>—</td>
<td>***</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>P3a</td>
<td>—</td>
<td>**</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>P3b</td>
<td>*</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HabP3</td>
<td>**</td>
<td>**</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Parietal SW</td>
<td>*</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* p < .05; ** p < .01; *** p < .001; — no significant result; grey shading represents measure-SCR interaction not tested.

2.4.2 HR

Figure 2.2 shows a substantial phasic heart rate deceleration, apparent within the first .5 s from stimulus onset, peaking within approx. 1.0 s, and returning to baseline within another 2 s. The evoked cardiac response was apparent in a significant quadratic trend ($F = 15.70, p = .001, \eta_p^2 = .511$). This showed no trials effect, or interaction with trial. The lack of trials effect was confirmed by comparison of HR and SCR over trials; this yielded a significant Measure (Heart Rate vs. Skin Conductance Response) \times Trial (linear trend over 1–10) interaction: $F = 17.27, p = .001, \eta_p^2 = .535$. 
2.4.3 Respiratory pause

A linear decrease over trials ($F = 8.14, p = .012, \eta^2 = .352$) was found, along with marginal recovery ($F = 4.45, p = .052, \eta^2 = .229$), but no significant dishabituation occurred. Figure 2.3 shows these effects. Since RP showed a marginal recovery, RP was tested against SCR for recovery, yielding a non-significant Measure (Respiratory Pause vs. Skin Conductance Response) × Trial (10 vs. 11) interaction, and indicating no significant difference between recovery in RP and SCR. RP was then tested against SCR for dishabituation and again yielded a non-significant interaction.

Figure 2.2. Mean phasic HR response relative to the pre-stimulus level across trials. Time point variation is indicated by SE bars. Vertical scale: change in beats per minute; horizontal scale: s.

Figure 2.3. Respiratory Pause as a function of trials, with SE bars. The linear regression line and Coefficient of Determination over the first 10 trials are also indicated.
2.4.4 ERPs

Figure 2.4 illustrates a random sample of single-trial responses for the 100 ms pre- to 500 ms post-stimulus epoch. A random participant was drawn for each trial in order; both trial and participant, once drawn, were not replaced. Figure 2.4 shows well-formed N1 and LPC components in the single-trial waveforms. Figure 2.5 demonstrates the averaged ERPs at the midline sites for each trial and removal of vertical eye

Figure 2.4. Randomly-selected examples of single trial ERPs at the midline sites for the 12 trials. Single trial data show clearly defined ERPs comparable to those from averaged data. Vertical scale: μV; horizontal scale: ms.
artifacts. The grand means at the midline sites are shown on the left of Figure 2.6, with the clearly defined components labelled at Cz.

Figure 2.5. Averaged ERPs at the midline sites are presented for the 12 trials, with vertical eye artifacts before (VEOG uncorr) and after (VEOG corr) EOG correction. Vertical scale: μV; horizontal scale: ms; note different EOG scale.
2.4.5 ERP PCA components

The PCA over the 0–500 ms latency range extracted 12 factors. The first nine factors, explaining 92.8 % of the total variance, were tentatively identified on the basis of peak latencies and topography: Factor 1 – Slow Wave (SW) at 428 ms (26.5% of total variance), Factor 2 – P3b at 309 ms (24.1% of total variance), Factor 3 – P2 at 223 ms (14.4% of total variance), Factor 4 – N1-1 at 125 ms (8.4% of total variance),

Figure 2.6. The left panel shows the grand mean ERPs across subjects and trials at each of the midline sites, with well-defined components labelled at Cz. The right panel depicts the corresponding virtual ERPs derived from the sum of the first nine virtual PCA components/subcomponents. The actual and virtual ERPs correspond very closely. Vertical scale: μV; horizontal scale: ms.
Factor 5 – Processing Negativity (PN) at 170 ms (7.9% of total variance), Factor 6 – N1-3 at 94 ms (4.6% of total variance), Factor 7 – P1 at 61 ms (2.7% of total variance), Factor 8 – HabP3 at 371 ms (2.2% of total variance), and Factor 9 – P3a at 264 ms.

Figure 2.7. Temporal PCA factor loadings plotted as a function of time, with topographic head maps of the virtual temporal components displayed above.

Figure 2.7 shows the rescaled factor loadings over time, and overall virtual topographies, for each of the 9 components. Virtual ERPs (the sum of these 9 components) are shown on the right of Figure 2.6; their good fit with the original ERPs on the left is readily apparent.

Figure 2.8 displays the mean PCA-derived components as a function of trials, while topography and amplitudes of each of the 9 components for the 12 trials are presented in Figure 2.9.
2.4.5.1 P1 (Cz)

P1 had a strong central (C > F/P: $F = 17.14, p < .001, \eta^2_p = .533$) and marginally frontal topography (F > P: $F = 3.36, p = .087, \eta^2_p = .183$), with a strong midline enhancement (M > L/R: $F = 28.38, p < .001, \eta^2_p = .654$) particularly in the frontal region (F > P × M > L/R: $F = 8.62, p = .010, \eta^2_p = .365$). The Cz activity showed no trial effects. This was confirmed by the comparison of P1 and SCR over trials, which yielded a significant Measure (P1 vs. Skin Conductance Response) × Trial (linear trend over 1–10) interaction: $F = 30.63, p < .001, \eta^2_p = .671$.

Figure 2.8. The 9 identified PCA-derived components as a function of trial. Linear regression lines and the Coefficient of Determination over the first 10 trials are also indicated.
2.4.5.2 N1-3 (Pz)

N1-3 negativity had a parieto-central topography ($F < P$: $F = 7.03$, $p < .018$, $\eta_p^2 = .319$; $C > F/P$: $F = 15.31$, $p = .001$, $\eta_p^2 = .505$) and marginal vertex enhancement ($C > F/P \times M > L/R$: $F = 4.22$, $p = .058$, $\eta_p^2 = .220$). No trials effects were evident at the Pz maximum. Comparison of N1-3 and SCR for trials showed a significant Measure (N1-3 vs. Skin Conductance Response) $\times$ Trial (linear trend over 1-10) interaction: $F = 30.25$, $p < .001$, $\eta_p^2 = .669$.

2.4.5.3 N1-1 (Cz)

As shown in Figure 2.9, N1-1 had a strong central ($C > F/P$: $F = 58.34$, $p < .001$, $\eta_p^2 = .795$) and midline topography ($M > L/R$: $F = 37.82$, $p < .001$, $\eta_p^2 = .716$), interacting to produce a distinct vertex maximum ($C > F/P \times M > L/R$: $F = 38.51$, $p < .001$, $\eta_p^2 = .720$). The vertex activity decremented marginally over trials ($F = 3.32$, $p = .088$, $\eta_p^2 = .181$) with no recovery on trial 11. A significant Measure (N1-1 vs. Skin Conductance Response) $\times$ Trial (linear trend over 1-10) interaction: $F = 5.88$, $p = .028$, $\eta_p^2 = .282$ marked the difference between N1-1 and SCR over trials.

2.4.5.4 PN (Fz)

PN had a frontal topography enhanced in the midline ($F > P \times M > L/P$: $F = 9.79$, $p = .007$, $\eta_p^2 = .395$). PN showed a maximum response at Fz that failed to decrement over trials. PN and SCR differed over trials, as indicated by a significant Measure (PN vs. Skin Conductance Response) $\times$ Trial (linear trend over 1–10) interaction: $F = 11.68$, $p < .004$, $\eta_p^2 = .438$.

2.4.5.5 P2 (Cz)

P2 had a central topography ($C > F/P$: $F = 16.38$, $p = .001$, $\eta_p^2 = .522$) and
responses in the frontal region were greater than in the parietal region (F > P: $F = 5.47$, $p = .034, \eta_p^2 = .267$), with a strong midline (M > L/R: $F = 18.95, p = .001, \eta_p^2 = .558$). These effects interacted to yield a frontally enhanced midline (F > P × M > L/R: $F = 5.62, p = .032, \eta_p^2 = .273$) with a distinct vertex positivity (C > F/P × M > L/R: $F = 9.85, p = .007, \eta_p^2 = .397$). The vertex activity showed no decrement over trials. Comparison of P2 and SCR over trials yielded a significant Measure (P2 vs. Skin Conductance Response) × Trial (linear trend over 1–10) interaction: $F = 21.06, p < .001, \eta_p^2 = .584$.

2.4.5.6 P3a (Cz)

P3a had a strong central (C > F/P: $F = 9.151, p = .009, \eta_p^2 = .379$) topography. The midline was greatest in the parietal sites (P > F × M > L/R: $F = 10.30, p = .006, \eta_p^2 = .407$). Activity at Cz failed to decrement over trials. A difference between P3a and SCR over trials was shown by a significant Measure (P3a vs. Skin Conductance Response) × Trial (linear trend over 1–10) interaction: $F = 14.89, p = .002, \eta_p^2 = .498$.

2.4.5.7 P3b (Pz)

P3b showed a parietal dominance (P > F: $F = 6.12, p = .026, \eta_p^2 = .290$) and marginal central elevation (C > F/P: $F = 4.52, p = .051, \eta_p^2 = .231$) with a strong midline (M > L/R: $F = 30.89, p < .001, \eta_p^2 = .673$). Responses at Pz decremented over trials ($F = 5.63, p = .031, \eta_p^2 = .273$) with no recovery on trial 11. Comparison of P3b and SCR for recovery yielded a significant Measure (P3b vs. Skin Conductance Response) × Trial (l0 vs. 11) interaction: $F = 5.78, p = .030, \eta_p^2 = .278$.

2.4.5.8 HabP3 (Fz)

HabP3 had a fronto-central topography (F > P: $F = 42.94, p < .001, \eta_p^2 = .741$; C
Figure 2.9. Topographic headmaps of the 9 identified components are shown for each of the 12 trials.

$F/P: F = 73.60, p < .001, \eta^2_p = .830)$. A vertex enhancement was evident ($C > F/P \times$
M > L/R: $F = 18.34, p = .001, \eta_p^2 = .550$). Fz activity diminished over trials ($F = 10.74, p = .005, \eta_p^2 = .417$), with pronounced recovery ($F = 15.19, p = .001, \eta_p^2 = .503$), but no dishabituation was found on trial 12. However, comparison of HabP3 and SCR for the dishabituation trial (12 vs. 10) showed no significant difference.

Figure. 2.10 The scaled grand mean of the 16 “leave-one-out” factor loadings for the HabP3 factor with the standard deviation of the mean at each time point displayed by grey shading.

For the HabP3 factor loading, the reliability “leave-one-out” analysis found that no congruence coefficient was less than .99, indicating that the HabP3s identified in each PCA can be considered equal (Lorenzo-Seva and ten Berge, 2006). Figure 2.10 shows the mean of the scaled factor loadings for the HabP3 from the 16 PCAs; note the variability is so small that it was necessary to use SD (rather than SE) error bars to see any sign of variability.

2.4.5.9 Parietal SW (Pz)

SW showed a negative frontal and positive parietal topography with a central reduction ($F < P$: $F = 49.15, p < .001, \eta_p^2 = .766$; $C < F/P$: $F = 4.61, p = .049, \eta_p^2 =$
There was negative dominance in the left hemisphere (L > R: $F = 6.54, p = .010, \eta_p^2 = .304$). Midline activity was greatest in the parietal region ($F < P \times M > L/R: F = 8.94, p = .009, \eta_p^2 = .374$). The Pz activity decremented over trials ($F = 8.60, p = .010, \eta_p^2 = .365$) with no recovery on trial 11. The parietal SW and SCR differed for recovery, shown by a significant Measure (parietal SW vs. Skin Conductance Response) $\times$ Trial (10 vs. 11) interaction: $F = 5.72, p = .030), \eta_p^2 = .276.$

2.4.6 Correlations exploring measure patterns

Three sets of correlations over 16 subjects and 12 trials (i.e., 192 corresponding pairs) were performed to explore the three patterns of relationships between the measures. The first pattern showed similarity between HR and P1, N1-3, N1-1, PN, P2, and P3a in failing to display a trial effect. The second pattern includes two ERP measures: P3b and parietal SW, which showed a trial effect but no recovery. The third pattern includes SCR, RP, and HabP3, which showed the three defining aspects of habituation. Table 2.2 presents the relationships between measures within each of the 3 patterns.

Table 2.2 Pearson product-moment correlation coefficients between the measures examined and the three patterns over trials.

<table>
<thead>
<tr>
<th>PATTERNS</th>
<th>SCR</th>
<th>HR</th>
<th>P3b</th>
</tr>
</thead>
<tbody>
<tr>
<td>RP</td>
<td>.109, $p = .067$</td>
<td></td>
<td></td>
</tr>
<tr>
<td>P1</td>
<td></td>
<td>.082</td>
<td></td>
</tr>
<tr>
<td>N1-3</td>
<td></td>
<td>.056</td>
<td></td>
</tr>
<tr>
<td>N1-1</td>
<td></td>
<td>-.007</td>
<td></td>
</tr>
<tr>
<td>PN</td>
<td></td>
<td>-.080</td>
<td></td>
</tr>
<tr>
<td>P2</td>
<td></td>
<td>.090</td>
<td></td>
</tr>
<tr>
<td>P3a</td>
<td></td>
<td>-.015</td>
<td></td>
</tr>
<tr>
<td>P3b</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HabP3</td>
<td>.160, $p = .013$</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Parietal SW</td>
<td></td>
<td>.284***</td>
<td></td>
</tr>
</tbody>
</table>

***$p < .001$. Tests are one-tailed. Grey shading represents correlations not examined.
2.5 Discussion

This study explored autonomic measures (HR deceleration, respiratory pause, and SCR) and ERP components in a dishabituation paradigm, permitting simultaneous measurements from different but related systems. Stimulus-response patterns of the measures were compared to those predicted by Preliminary Process Theory. Each measure was statistically compared to the relevant aspect of the S-R pattern of SCR (the gold-standard of the phasic OR). Novelty has been shown to be a major determinant of the phasic OR (Sokolov, 1963b) under different experimental conditions (Barry, 1996; Donchin et al., 1984; Zimmer and Demmel, 2000). The OR is often operationalised in terms of response decrement over trials, but the inclusion of a different stimulus on a change trial (to test recovery) and the representation of the former stimulus (to test dishabituation) are necessary to confirm that the response decrement reflects novelty reduction. The simple auditory stimuli at long ISIs allow clear ERP waveforms from single trials, whose “rapid changes in response amplitude are obscured” (Roth, 1973, p.136) by averaging group means over trials (see also Ritter et al., 1968).

The SCR to the moderate brief 80 dB tone exhibited a linear diminution over trials, increased to the change stimulus, and increased relative to the habituated level on the dishabituation trial. This characteristic S-R pattern identifies the SCR as the benchmark phasic OR (Barry, 1984b, 1996, 2006) and confirms this paradigm as suitable for exploring the phasic OR.

As predicted, the HR deceleration was apparent as a strong quadratic trend occurring immediately after stimulus onset, and reaching a maximum around 0.75 s. No systematic response decrement was found over trials, significantly different to that in SCR. The brief HR deceleration has been observed in other studies where novelty
(Barry et al., 2011), intensity (Lawrence and Barry, 2009; MacDonald et al., 2012; Barry et al., 2013) and cognitive load (Significance: Lawrence and Barry, 2010) have been manipulated with stimuli in the moderate range of intensity. The stability of this response over a variety of experimental conditions confirms the early HR deceleration as representing reflexive early stimulus processing in PPT prior to the elicitation of the OR, i.e., physical transient detection.

Respiratory pause is rarely measured, yet early studies demonstrated this measure’s sensitivity to stimulus repetition (novelty) (Barry, 1977a,b; Barry and James, 1981a,b), and this was recently confirmed in MacDonald et al. (2012) and Barry et al. (2013). The respiratory pause in this study decreased linearly with trials; for the change trial it showed marginal recovery that was found not to differ from recovery in SCR. RP showed no increased response on the dishabituation trial, but that also showed no significant difference from SCR’s dishabituation. This could indicate either that RP shows dishabituation, or that there is insufficient power to discriminate it from SCR in this regard; further research is necessary to clarify this issue. With this proviso, while previous studies involving the autonomic measures used here have demonstrated three S-R patterns: no trial effect (HR); trial and recovery effects (RP); and trial, recovery, and dishabituation effects (SCR), two patterns have emerged for the autonomics in this study, and they provide two patterns to seek analogues for in the ERPs.

Since the OR supposedly incorporates a diverse array of responses from different bodily systems, these different S-R patterns of the autonomic system to variations of novelty should be reflected in central measures. Previous investigations have aligned the LPC with SCR (Rushby et al., 2005; Steiner and Barry, 2011), subcomponents of the N1 with HR deceleration (Barry et al., 2011; Lawrence and Barry, 2009, 2010), P3a and P3b with HR deceleration and acceleration respectively.
(Lawrence and Barry, 2009; Rushby and Barry, 2009), and ‘Novelty P3’ with respiratory pause (suggested from a cluster of studies without concurrent RP measurements (e.g., Rushby and Barry, 2009).

In our study, PCA was used to extricate ERP components in the time range between 0 and 500 ms. The identification of each component was based on topography and latency, with the first 9 of the 12 factors tentatively labelled in temporal order: P1, N1-3, N1-1, PN, P2, P3a, P3b, HabP3, and early SW. Importantly, the PCA-derived components of the LPC displayed the same temporal order found previously in habituation studies: P3a, P3b, ‘Novelty P3’/HabP3 and SW (Barry, 2009; Barry et al., 2011; Barry et al., 2013; Rushby et al., 2005).

The P1 has been described as a fronto-central wave (Beer and Röder, 2004; Rushby and Barry, 2009) with small variable amplitude (Pratt et al., 2008). Consequently, the P1 has sometimes failed to emerge in PCAs at long ISIs, although the raw data indicate its presence (Roth et al., 1984; Rushby and Barry, 2009). Functionally, the P1 has links with early encoding of deviance and regularities (Grimm and Escera, 2012). The P1 in this study also had a fronto-central topography. Reported trial results have been scarce and inconsistent (Gillette et al., 1997; Sambeth et al., 2004). Trial effects were not found here and P1 differed from SCR over trials.

A typically central N1 is elicited strongly by a stimulus after a change from silence (Näätänen and Picton, 1987), especially at longer ISIs (Barry et al., 2011). A vertex-potent N1 emerged from the PCA with a latency of 125ms. This component, in terms of latency and topography, appears to align with the N1-1 found by Barry et al. (2011). No response decrement of the N1 was predicted, and no decrement was observed for either the N1-1 or N1-3 subcomponents, contrasting to the trials decrement
of the SCR.

The PN has generally-agreed frontal midline topography with a broad latency range (50–150 ms) (Näätänen, 1982, 1987; Picton et al., 1976). The PN has been identified as an ERP component by PCA previously and appears to represent reflexive attention-switching pertaining to novel stimuli, with the response decrement marking decreasing novelty over trials (Barry et al., 2011). The PCA in this investigation derived a similar factor with a frontal topography and latency 170 ms; no trial decrement was observed, and that differed significantly from the SCR pattern, suggesting engagement of an early attention-switching process with repeated stimuli.

The P2 in early studies was derived as the N1/P2 complex rather than a separate peak, but the use of PCA has isolated the central P2 (Roth et al., 1982). Functionally the P2 has been linked to difficulties in withdrawing attentional resources from a stimulus (Crowley and Colrain, 2004). Generally, the P2 has been reported as insensitive to stimulus repetition (Crowley and Colrain, 2004; Romero and Polich, 1996; Rushby and Barry, 2009). The P2 here had a fronto-central distribution with a distinct vertex topography, and activity at Cz was resistant to trial effects as expected. Comparison to SCR showed a significant difference over trials.

The central/parietal P3a has often been elicited by an infrequent distracter stimulus engaging focal attention in auditory oddball tasks (Polich, 2007), enhanced by the stimulus salience (Goldstein et al., 2002). The P3a at short ISIs has shown trials effects (as an overall decrement along with topographical interactions), intensity-dependent recovery, and dishabituation (Rushby et al., 2005). At longer ISIs trials effects have been absent (Rushby and Barry, 2009) or weak (Barry et al., 2011). In this paradigm, the P3a had a parieto-central topography with a strong vertex maximum, and
no trial decrement was found, differing significantly from the SCR trials response.

The P3b demonstrates a varying sensitivity to stimulus repetition, dependent on task requirements and paradigm conditions (Donchin et al., 1984). Rushby and Barry (2009) found only a weak linear trend for trials at very long ISIs. Barry et al. (2011), in a paradigm with no task requirement and long ISIs, found only topographical reductions without an overall decrement; while Rushby et al. (2005), with short ISIs with Significance (button press) and varying intensity, found overall decrement over trials, increased response on the change trial, but no dishabituation. The P3b in this study displayed a parietal dominance with a strong midline, and diminishing Pz activity over trials. No recovery was found from trial 10 to trial 11. Comparison between P3b and SCR on the recovery trial showed a significant difference. Although a trials effect was expected, the lack of recovery was not.

We identified a post-P3b HabP3 with a fronto-central topography. This component has a more anterior topography than previously found in a habituation paradigm (Barry et al., 2011) and dishabituation paradigm (Rushby et al., 2005). Our HabP3 displayed diminishing Fz activity over trials, with strong recovery consistent with a novelty response to the change, as predicted; along with no dishabituation. However, comparison between HabP3 and SCR found no significant difference for the dishabituation (12 > 10). Consequently, the HabP3 cannot be ruled out as a CNS analogue for the SCR.

Long ISIs not only produce a robust P3b, but a large SW that overlaps the preceding P3b and P3a (García-Larrea and Cézanne-Bert, 1998; Roth et al., 1984; Squires et al., 1975; Strüber and Polich, 2002), especially when attention is drawn to the stimulus (Rohrbaugh et al., 1978; Squires et al., 1975) or when a stimulus becomes
obtrusive to a participant (Roth et al., 1984). The classic SW with an anterior negativity and posterior positivity with peak latency approximately 450–650 ms has been identified as an ‘O wave’ component (Roth et al., 1984), the cortical equivalent of the OR (Loveless and Sandford, 1974). The SW component found here exhibited a frontal negativity and parietal positivity with a parietally enhanced midline, with positivity dominant in the right hemisphere. Decrement was evident in Pz activity, but no recovery was observed for the change stimulus. A significant difference was found between the parietal SW and SCR from trial 10 to trial 11. The influence of stimulus repetition on the SW has been inconsistent in a variety of paradigms, with reports of no trials effects (Roth et al., 1984), increased response over trials (Rohrbaugh et al., 1976; Simons et al., 1987), differential reductions of frontal and parietal components (Zimmer and Demmel, 2000), and reported decrement, recovery, and dishabituation (Rushby et al., 2005). Our SW demonstrated decrement as predicted, suggesting some linkage with the OR, yet no recovery occurred. The occurrence of a trial effect with no recovery weakens its association with novelty and suggests other conditions maybe more influential in its elicitation. The lack of recovery rules this SW out as a central index of the OR.

It should be noted that in oddball paradigms employing shorter ISIs, the latency ordering of the LPC components appears to be: P3a/Novelty P3, P3b, and classic SW (Spencer et al., 2001), contrasting to the habituation paradigm typical of OR investigations that orders the LPC components: P3a, P3b, HabP3 (previously referred to as ‘Novelty P3’), and classic SW. It is not clear what the relation is between Novelty P3 and HabP3 – whether they are the same component in different latency order, or different components. The presence of a P3a here and its supposed equivalence to the oddball Novelty P3 complicates this picture further.
This study examined 3 autonomic and 9 central measures (identified on the basis of latency and topography from a PCA) in a passive auditory dishabituation paradigm. The brief tones of moderate intensity at very long ISIs were conducive to OR elicitation, confirmed by the SCR’s S-R pattern. To our knowledge this is the first study to concurrently examine PCA-derived ERP components along with cardiac activity, respiratory pause, and skin conductance in a dishabituation paradigm, and to include a quantitative method for testing differences between trials effects in these measures (Interaction Test). From the autonomic measures, HR deceleration showed no trials effects, and RP demonstrated trial effects with recovery (its predicted non-dishabituation status could not be established). These two patterns support the core autonomic response fractionation that forms the basis of Preliminary Process Theory of the OR.

This is the first dishabituation study to incorporate statistical examination of different measures in comparison to SCR (the autonomic OR ‘yard-stick’). Three distinct patterns emerged for the measures with respect to the stimulus-response pattern demonstrated by SCR. The first pattern was demonstrated by HR deceleration, P1, N1-3, N1-1, PN, P2, and P3a, which failed to decrement over trials, differing significantly from the trials aspect of SCR. This places these measures as markers for transient detection or other processes prior to OR elicitation – the elicitation of these measures appears more dependent on physical aspects of the stimulus than novelty. The second pattern was demonstrated by the P3b and parietal SW, which exhibited trial decrement comparable to SCR, yet without an enhanced response on the change stimulus. The observed decrement with no recovery may relate more to a refractory period effect than to novelty per se. This second pattern was not matched by the autonomic measures examined. The third pattern of decrement, recovery, and dishabituation was
demonstrated by the HabP3 and SCR. Only HabP3 of the PCA-derived subcomponents of the LPC exhibited an S-R pattern analogous to the SCR. Previous habituation/dishabituation studies in our laboratory have not found recovery or dishabituation in HabP3. Here it failed to differ from SCR, and this might reflect a power issue rather than a real equivalence; further work is needed to resolve this question.

Fractionation was observed as two distinct S-R patterns for the autonomic measures: no decrement, and decrement-recovery-dishabituation. These patterns were somewhat matched by those of the ERP components. Three ERP patterns emerged: no decrement, decrement and no recovery, and decrement-recovery-dishabituation. The P3b and parietal SW both failed to show recovery for the change stimulus that has been demonstrated previously in another dishabituation study (Rushby et al., 2005), although in that study intensity was implicated in the increased response. Replication of this study at longer ISIs with the measures compared to SCR and the inclusion of intensity would clarify this second pattern.

Notwithstanding this question, we have demonstrated response fractionation in a range of autonomic measures associated with the OR, disconfirming the concept of the unitary OR and confirming the basis of Preliminary Process Theory. This alternative account of the processing involved in OR generation is the only extant theory that accommodates the observed response fractionation. We also demonstrate fractionation in the central correlates of the stimulus processing involved. Some parallels exist between PPT’s onset transient processing and the exogenous ERP components, and the later endogenous ERP components share some S-R patternings with later stages in PPT. The testing of observed pattern differences by assessing the interaction in pattern aspects between the measures and SCR, while posing some interesting questions, has
enhanced interpretation of the present results. Further exploration is recommended using simple stimuli in dishabituation paradigms with single-trial autonomic and central data concurrently collected. We also advocate further exploration of the use of PCA to ‘tease apart’ ERP components/subcomponents and widening that exploration to other OR determinants, such as intensity and significance.
2.6 References


Barry, R.J., James, A.L., 1981b. Fractionation of respiratory and vascular responses
with simple visual stimulation. Physiol. Psychol. 9, 96–101.


Lawrence, C.A., Barry, R.J., 2009. ERPs and the evoked cardiac response to auditory


Psychophysiology 24, 375–425.


Psychophysiology 10, 125–138.


Zimmer, H., Demmel, R., 2000. Habituation and laterality of orienting process as

Chapter 3. Study 2: Trials and intensity effects in single-trial ERP components and autonomic responses in a dishabituation paradigm with very long ISIs

Published as:

3.1 Abstract

The phasic orienting reflex (OR) was investigated using single-trial data collected concurrently from 4 autonomic measures and event-related potentials (ERPs). In an auditory dishabituation paradigm, twelve indifferent tones of two intensities (60 or 80 dB, intensity change on trial 11, counterbalanced between subjects) were presented at very long interstimulus intervals (ISIs). Novelty and intensity-based stimulus-response patterns were examined seeking ERP analogues of autonomic measures representing pre-OR and OR processing. Skin conductance response (SCR) represented the phasic OR index. EOG-corrected ERP data for 16 subjects were decomposed by a temporal Principal Components Analysis (PCA). SCR diminished over 10 standard trials, recovered on change trial 11, dishabituated to the re-presentation of the standard tone on trial 12, and showed intensity effects at the change – formal requirements for an OR index. The evoked cardiac response (HR) showed no trial or intensity effects. Respiratory pause (RP) decreased linearly over trials and showed recovery but no dishabituation or intensity effect. Peripheral vasoconstriction (PVC) failed to decrement but exhibited an intensity effect. Ten identifiable ERP components were extracted: Na, P1, N1-1, PN, P2, P3a, P3b, a novelty-sensitive HabP3, an intensity-sensitive IntP3, and the Slow Wave (SW). Pattern 1 showed no trial or intensity effects (HR, P1, PN, P2); Pattern 2 showed no trial effect but an intensity effect (PVC, Na, N1-1, P3a); and Pattern 3 demonstrated habituation and an intensity effect (SCR, RP, P3b, HabP3, IntP3, SW). The observed fractionation of autonomic and central measures is consistent with Preliminary Process Theory (PPT) rather than the notion of a unitary OR.
3.2 Introduction

3.2.1 Background

The General introduction of Chapter 1 has provided an account of stimulus-response patterns of autonomic and major PCA-derived components when novelty and intensity were varied. A brief summary of Chapter 2 is presented here as a background for Study 2, followed by relevant intensity related findings.

Study 1 (MacDonald and Barry, 2014) manipulated novelty in an auditory dishabituation paradigm at very long ISIs, and collected autonomic and EEG data simultaneously. The phasic skin conductance response (SCR), heart rate deceleration (ECR1), respiratory pause (RP), and 9 PCA-derived ERPs served as dependent measures. The measures were grouped into three patterns. Pattern 1 (no trial effect): ECR1, P1, N1-3, N1-1, PN, P2, and P3a; Pattern 2 (trial effect but no recovery): P3b and parietal SW; and Pattern 3 (decrement, recovery, and dishabituation): SCR and HabP3. Patent fractionation was evidenced by these measures when novelty was varied.

In Chapter 3 all central measures cited use linked ears or mastoids as reference unless otherwise stated.

3.2.2 N1

The topography of the N1 typifies the auditory evoked response (AER). Vaughan and Ritter (1970) proposed that “all AERs are maximum at or near the vertex” (p. 362), using tip of the nose as reference. The N1 has shown sensitivity to intensity manipulations in a variety of paradigms – e.g., it has been applied in the study of loudness dependence of auditory evoked potentials (LDAEP) in relation to serotonin levels; Nathan et al. (2006) – with louder stimuli evoking larger responses (MacDonald
et al., 2012; Näätänen and Picton, 1987; Squires et al., 1975; White and Yee, 2006).

### 3.2.3 LPC

Similarity between the antecedent conditions of the OR and LPC (Late Positive Complex, also known as the P3[00]) has long been recognised (Donchin et al., 1984). The LPC has been portrayed as a complex marking memory updating of the stimulus context (Donchin, 1981; Donchin and Coles, 1988), sensitivity to stimulus uncertainty (Sutton et al., 1965), degree of expectation (Donchin and Coles, 1988; Verleger, 1988), relevance (Donchin, 1981; Johnson, 1984), and stimulus parameters (Papanicolaou et al., 1985 — left ear reference; Polich et al., 1996). Subcomponents of the LPC were identified in two seminal oddball investigations according to their sensitivity to experimental variables. Squires et al. (1975) identified the P3a linked to intensity variation, while Courchesne et al. (1975) found what became known as the Novelty P3 associated with complex, unrecognisable stimuli; both subcomponents displayed a frontal topography. Subsequently, Principal Components Analysis has decomposed the LPC into at least four distinct subcomponents in habituation paradigms, in increasing latency order: P3a, P3b, Novelty P3, and the Slow Wave (SW), each differing in topography, eliciting conditions, and sensitivity to task demands (Barry et al., 2011, 2013; Roth et al., 1982; Sutton and Ruchkin, 1984; Study 1). The P3a has displayed trial decrement at short ISIs (Rushby et al., 2005) but no decrement at very long ISIs (Barry et al., 2013; Rushby and Barry, 2009; Study 1). An unpredictable change in intensity (Barry et al., 2013; Rushby et al., 2005; Squires et al., 1975) has been reported to elicit the P3a. However, Simons et al. (2001) replicated the oddball studies of Squires et al. (1975) and Courchesne et al. (1975) and concluded that the distinction between the Novelty P3 and P3a was not supported empirically. This has become the dominant view in the literature. Despite this, a component identified as the Novelty P3 in habituation...
studies has consistently decremented to re-presented stimuli (Barry and Rushby, 2006; Rushby et al., 2005; Rushby and Barry, 2009; Study 1) and shown a sensitivity to stimulus recognition and predictability independent of complexity (Courchesne et al., 1975); and no intensity sensitivity (contrasting with the P3a). While the P3a consistently exhibits a central focus (Barry et al., 2013; Study 1), often with a frontal enhancement (Rushby et al., 2005; Rushby and Barry, 2009), the Novelty P3 has been reported with a parietal (Rushby et al., 2005), fronto-central (Study 1), or non-specific (Rushby and Barry, 2009) dominance. Consequently, P3a and Novelty P3 may be considered separate PCA-derived ERP entities related to different aspects of the experimental context in OR research. However, in light of Simons et al. (2001), we now label this component from habituation paradigms as HabP3, and recognise that it may not be identical to Courchesne’s Novelty P3. The Introduction of Chapter 2 outlines intensity sensitivity of the P3b, Novelty P3, and SW at long ISIs.

3.2.4 This study

This study addresses and extends upon the recommendations of Barry et al. (2013) by manipulating novelty within subjects, and intensity between subjects, in a dishabituation rather than habituation paradigm. Some autonomic and central associations have already been proposed. The LPC as an OR marker has received substantial support (Donchin et al., 1984; Rushby et al., 2005; Ritter et al., 1968; Steiner and Barry, 2011), however no ERP component/subcomponent adequately meets the requirements of the OR when novelty, intensity, and Significance have been varied. Transient detection appears closely associated with the N1 and HR deceleration, while intensity-insensitive HabP3 and RP demonstrate trial effects, aligning with novelty processing. The P3b and SW may be related to the voluntary OR but no autonomic analogues are obvious.
The present study explores linkages between autonomic and central measures incorporating an intensity change. The auditory stimuli are simple tones of moderate intensity known to generate the OR. Temporal PCA-derived ERP components from single-trial data will be examined with respect to the stimulus-response patterns of SCR, HR, RP, and PVC. We intend to examine the degree of sensitivity of SCR, HR, RP, PVC, and the decomposed ERP components/subcomponents to novelty and intensity variation, thus clarifying the relationships between the various central and autonomic measures in preliminary processing prior to OR generation, and the OR itself. The response decrement of the SCR to variations in novelty for stimuli of moderate intensity has been well established (Barry, 1975; Barry et al., 2011; Blakeslee, 1979; Jackson, 1974; MacDonald et al., 2012; Sokolov, 1963b; Steiner and Barry, 2011, 2014). Within subjects’ responses are preferred for detecting differences between conditions due to less intrinsic error variance compared to between subjects responses. Consequently, our primary focus for detecting an intensity effect is an examination of the response difference between the pre-change trial (trial 10) and the intensity change trial (trial 11). The SCR should demonstrate the consistent OR response pattern: response decrement over repeated standard trials, enhanced response at the change stimulus (more so for the increase in intensity), and an increase to re-presentation of the standard stimulus. The phasic HR deceleration should show no systematic variation over trials or intensity. A strong respiratory pause is anticipated for the first stimulus and the response should decrement with trials (Barry et al., 2013; MacDonald et al., 2012; Study 1), demonstrate an intensity-independent recovery at the change stimulus (Barry and James, 1981a), along with no increased response for the dishabituation trial (Barry and James, 1981a; Study 1). Peripheral vasoconstriction has shown resistance to novelty reduction but sensitivity to intensity variation (Barry and James, 1981a, 1981b; Barry et al., 2013);
PVC should not decrement over trials but reflect changes in intensity. The temporal PCA will use an ample latency range to encompass ERPs reflecting aspects of the involuntary OR. The P1 has been linked to stimulus onset and preattentive arousal and should be evoked by the first stimulus with no diminution over trials (Gillette et al., 1997 — Fz reference; Pratt et al., 2008; Study 1), however some sensitivity to intensity change may be observed (White and Yee, 2006). The N1, sensitive to stimulus onsets/offsets (Näätänen, 1988; Näätänen and Picton, 1987), should not decrement over trials (Rushby and Barry, 2009; Barry et al., 2011, 2013), and increased intensity may yield greater responses (Näätänen and Picton, 1987; Polich et al., 1996). The novel aspects of the first stimulus should elicit the PN from reflexive attention switching (Barry et al., 2011; Study 1), and that may decrement over trials (Barry et al., 2011).

Some PN enhancement may be observed for the low-intensity tone (Näätänen, 1982). The P2 is not expected to show trial effects at these long ISIs (Crowley and Colrain, 2004; Rushby and Barry, 2009; Study 1) but should display an increased response for the 80 dB tone (Crowley and Colrain, 2004; Orlebeke et al., 1989; Roth et al., 1982). The P3a, associated with salience (Goldstein et al., 2002 — Cz reference), involuntary OR (Barry and Rushby, 2006), and attention switching (Dien et al., 2004; Pritchard, 1981) should be unresponsive to stimulus novelty at very long ISIs (Barry et al., 2011; Rushby and Barry, 2009; Study 1), but some sensitivity to changed intensity is expected (Rushby et al., 2005; Squires et al., 1975). The posterior P3b should show response reduction over trials but no overall recovery (Barry et al., 2011; Rushby et al., 2005; Study 1), however the P3b has sometimes shown resistance to decrement (Barry et al., 2013; Rushby and Barry, 2009). An intensity-dependent recovery is expected (Rushby et al., 2005). The HabP3 subcomponent is particularly sensitive to the first instance of a simple stimulus – the ‘newness’ per se (Barry et al., 2011; Rushby and Barry, 2009).
The HabP3 should decrement with trials (Barry et al., 2011; Rushby et al., 2005; Rushby and Barry, 2009; Study 1; but not Barry et al., 2013), show recovery independent of intensity (Rushby et al., 2005; Study 1), and possibly dishabituation (Rushby et al., 2005; Study 1). The SW has been associated with the OR (Loveless and Sandford, 1974), so consequently should demonstrate trial effects (Rushby et al., 2005; Zimmer and Demmel, 2000; Study 1; but not Barry et al., 2011, 2013), but no response recovery is expected at these very long ISIs (Study 1), nor intensity effects (Roth et al., 1982, 1984; Rushby et al., 2005). The present study consolidates and logically extends the investigation of novelty in the OR context in Study 1 and Barry et al. (2013) where novelty and intensity were manipulated in a habituation paradigm; as well as addressing the paucity of studies employing ‘genuine’ dishabituation paradigms. This rarely used auditory dishabituation paradigm incorporates the systematic variation of both novelty and intensity with autonomic (including the uncommonly examined RP and PVC) and central data collected concurrently. This unique opportunity permits the possible matching of autonomic and PCA-derived ERPs from the perspective of PPT.

3.3 Methods

3.3.1 Participants

Sixteen university students participated in an experimental session (age 18–25, mean 19.7 years; all female).

3.3.2 Procedure

To examine the influence of novelty and intensity, auditory stimuli of 60 dB and 80 dB tones at 1000 Hz, with a duration of 50 ms (15 ms rise/fall times) were presented at a random, variable ISI of 50–70 s. Novelty reduction is operationised by trial decrement, so participants received 10 tones at one intensity (either 60 dB or 80 dB), a
change trial at the other intensity, and the original tone was re-presented on trial 12. Zero, 1, or 2 standard tones were added randomly for each participant to reduce subject communication and expectation concerning the number of stimuli. Participants were randomly assigned to one of two counterbalanced groups based on the intensity of the first tone so the different groups had complementary tones in the series. The first twelve tones were used for analysis. The groups did not differ on age (mean 19.1, SD 1.6 vs. mean 20.2, SD 2.1 years).

3.3.3 Physiological recording

The details of the physiological recording of Electrodermal activity, Heart activity, Respiration, and the Electroencephalogram are identical to the descriptions in section 2.3.3. Peripheral pulse amplitude (PVC) has been included here as an intensity-sensitive autonomic dependent variable.

3.3.3.1 Peripheral pulse amplitude

Peripheral pulse amplitude response was recorded by means of a photoelectric transducer (Model 1020FC, UFI) attached to digit IV of the non-preferred hand. Pulse amplitude was sampled continuously at 512 Hz.

3.3.4 Data extraction

The criteria for data extraction for SCR, HR deceleration, Respiratory pause and ERPs are presented in section 2.3.4 of Chapter 2. Peripheral vasoconstriction is presented here.

3.3.4.1 Peripheral vasoconstriction

Analysis of PVC assessed the quadratic trend over beats that represents the
phasic constriction. The peripheral vasoconstriction was quantified as the maximum relative decrease in pulse amplitude between 4 and 13 beats after stimulus onset.

3.3.5 Statistical analysis

A single temporal PCA was used to identify ERP components from single-trial EOG-corrected data in the time range of -100–550 ms from 19 scalp locations across 16 subjects. The full number of data files (16 subjects × 12 trials × 19 sites = 3648) were submitted to temporal PCA decomposition utilising Dien's ERP PCA toolkit (v. 2.23; Dien, 2010) in Matlab. The temporal PCA used a covariance matrix and the number of components equalled the number of time points (332points). Kaiser normalisation was employed and all components were subjected to Varimax rotation to maintain orthogonality and clarify interpretation. Virtual ERP component amplitudes calculated from the product of factor loadings, factor scores, and standard deviations were used for analysis. Separate analysis of variance (MANOVA) of the identified component ERP measures from the nine central sites examined topography, with coronal plane [left (F3, C3, P3), midline (Fz, Cz, Pz) and right (F4, C4, P4)] and sagittal plane [frontal (F3, Fz, F4), central (C3, Cz, C4) and parietal (P3, Pz, P4)] as repeated-measures factors. Planned contrasts within the coronal plane compared the left (L) vs. right (R) regions, and the midline (M) vs. the mean of the left and right regions. Within the sagittal plane, frontal (F) vs. parietal (P) regions, and central (C) regions vs. The mean of the frontal and parietal regions was analysed. These orthogonal planned contrasts provide optimal information on the topographic distribution of the amplitude of each component.

For this dishabituation study, the pooled maximum amplitude across one of three medial regions (frontal, central or parietal regions) for each ERP was used for analysis. ERP maxima and all autonomic measures were separately examined for
response decrement. These measures were examined in repeated measures multivariate analysis of variance (MANOVA) with the factor Trials (for trials 1 to 10). Within Trials, linear trend was examined to assess decrement. Separate MANOVAs were conducted to examine change trial responses (recovery: trial 11 vs. 10) if decrement over trials was found. Similarly, responses for the re-presentation trial (dishabituation: trial 12 vs. 10) were analysed only if recovery on the change trial was significant. In addition, the within subject intensity effect for recovery (trial 11 vs. 10) was examined by testing the significance of the intensity × trial interaction (Barry and Furedy, 1993). A within subject intensity effect, consistent with the OR viewpoint, would be indicated by the magnitude of response from the 80 dB tone being significantly larger than that from the 60 dB tone. The analysis of the HR response over a 2.5 s epoch also included a Time factor with a planned comparison to examine the quadratic trend over Time that defines HR deceleration, and the PCV response was examined similarly for a quadratic trend from the 4th to the 11th post-stimulus beat.

Separate repeated measures MANOVAs were conducted to examine differences between HR/RP/PVC and SCR, along with SCR and the virtual ERP components (the Interaction Test). Specifically, the benchmark OR pattern: decrement, recovery, and dishabituation, plus the intensity × trial interaction at the change, is expected to be demonstrated by the phasic SCR. If a measure differed from the SCR pattern in its initial analyses, then the differing aspect of the pattern was tested with a repeated-measure MANOVA for that measure and SCR, using Z-scores for each subject over the relevant trials, and the Interaction Test would be applied: Measure × decrement/recovery/dishabituation or measure × intensity × trial interaction. If the test yielded a significant interaction, this confirmed that the measure’s pattern differed from that same aspect of the SCR pattern; subsequent pattern testing was discontinued. If the
interaction was non-significant, this was interpreted as that aspect of the pattern not differing significantly from SCR, and subsequent testing ensued for the next aspect of the stimulus-response pattern. For ERPs, this was conducted on a subject’s Z-scores representing the maximum mean amplitudes at either the frontal, central, or parietal regions for the component. Also, for negative components, the polarity was reversed to ensure decrement, recovery, and dishabituation was appropriately tested. Subsequently, if a specific response pattern across trials/intensity was observed for particular autonomic or central measures, then the relationship between these measures was examined by correlation.

To examine the stability of the OR-related components found here and in Study 1, Congruence Coefficients ($r_c$) were calculated from 0 to 500 ms for both studies between components in comparable latency ranges. The Congruence Coefficient captures information about latency, rise/fall times, and response magnitude rather than topographical similarities between PCA factors (Barry et al., 2014).

Since all contrasts were planned and there were no more of them than the degrees of freedom for effect, no Bonferroni-type adjustment to $\alpha$ was necessary (Tabachnick and Fidell, 1989). Also, Greenhouse-Geisser type correction was not necessary because single degree of freedom contrasts are not affected by the violations of sphericity assumptions common in repeated-measures analyses of physiological data (O’Brien and Kaiser, 1985). All tests reported have (1, 14) degrees of freedom, and effect sizes (partial $\eta^2$) are indicated.

3.4 Results

3.4.1 SCR

The mean SCR waveform displayed onset latency of approx. 1.8 s and peak
latency of approx. 3.5 s. Trial decrement was apparent as a linear trend \((F = 30.57, p < .001, \eta_p^2 = .686)\), evident in Figure 3.1. The SCR showed both recovery to the change stimulus \((11 > 10: F = 9.18, p = .009, \eta_p^2 = .396)\) and dishabituation \((12 > 10: F = 5.77, p = .031, \eta_p^2 = .292)\). An intensity effect was indicated by the intensity × trial interaction \((\text{Intensity} \times \text{Trial} (11 \text{ vs. } 10): F = 9.03, p = .009, \eta_p^2 = .392)\). See Table 3.1.

Table 3.1 summarises the trials, recovery, and dishabituation results for each measure together with enhanced amplitudes due to an intensity × trial (recovery) interaction to assess the intensity effect. Also included is the measure vs. SCR interaction test for the three pattern aspects and the measure × intensity × trial (recovery) to test differences for intensity at the change trial.

3.4.2 HR

Figure 3.2A shows a clear phasic heart rate deceleration, observed within the first 1 s from stimulus onset, with a peak latency of approx. .75 s, and returning to baseline within 3.50 s. A significant quadratic trend \((F = 10.85, p = .005, \eta_p^2 = .437)\)

![Figure 3.1](image.png)

Figure 3.1. The square-root transformed phasic SCR as a function of trials and intensity for condition. SE bars indicate the trial to trial variation, and the linear regression line and Coefficient of Determination for the first 10 trials are included.
Table 3.1. Decrement, recovery, dishabituation and intensity effects for ANS and CNS (ERP) measures.

<table>
<thead>
<tr>
<th>Autonemics</th>
<th>Decrement</th>
<th>Measure vs. SCR (Decrement)</th>
<th>Recovery</th>
<th>Measure vs. SCR (Recovery)</th>
<th>Dishabituation</th>
<th>Measure vs. SCR (Dishabituation)</th>
<th>Intensity × Trial (11 vs. 10) (Intensity)</th>
<th>Measure vs. SCR × Intensity × Trial (11 vs. 10) (Intensity)</th>
<th>S-R Pattern</th>
</tr>
</thead>
<tbody>
<tr>
<td>SCR</td>
<td>***</td>
<td>**</td>
<td>*</td>
<td>**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>3</td>
</tr>
<tr>
<td>HR</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>RP</td>
<td>*</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>3</td>
</tr>
<tr>
<td>PVC</td>
<td></td>
<td>**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2</td>
</tr>
<tr>
<td>Na</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2</td>
</tr>
<tr>
<td>P1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>*</td>
<td></td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>N1-1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2</td>
</tr>
<tr>
<td>PN</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>P2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>p = .064</td>
<td>1</td>
<td></td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>P3a</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>p = .066</td>
<td>1</td>
<td></td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>P3b</td>
<td>**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2</td>
</tr>
<tr>
<td>HapP3</td>
<td>*</td>
<td></td>
<td></td>
<td></td>
<td>p = .071</td>
<td>3</td>
<td></td>
<td></td>
<td>3</td>
</tr>
<tr>
<td>IndP3</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>*</td>
<td>3</td>
<td></td>
<td></td>
<td>3</td>
</tr>
<tr>
<td>Parietal SW</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>3</td>
</tr>
</tbody>
</table>

* p < .05; ** p < .01; *** p < .001; — no significant result; grey shading represents measure-SCR interaction not tested

marked the cardiac response over the first 2.5 s. No linear trend over trials 1–10 was found (Figure 3.2B). A marginal difference was noted between trials 10 and 11 for Intensity (60 dB vs. 80 dB) × Trial (11 vs. 10): F = 4.19, p = .060, \( \eta_p^2 = .230 \), with 60 dB stimuli giving a somewhat larger response. Comparison of HR and SCR over trials

Figure 3.2. Panel A. Mean phasic HR response change relative to the pre-stimulus level across time. Vertical scale: change in beats per minute; horizontal scale: s. Panel B. Maximum HR deceleration over the 12 trials for both intensities. Vertical scale: Maximum HR deceleration in beats per minute; horizontal scale: trials. Time point variation is indicated by SE bars. Linear

76
yielded a significant Measure (HR vs. SCR) × Trial (linear trend over 1–10) interaction: $F = 21.05, p < .001, \eta_p^2 = .601$; and a significant difference was revealed between HR and SCR with the intensity interaction (HR vs. SCR) × (60 vs. 80 dB) × Trial (11 vs. 10): $F = 15.00, p = .002, \eta_p^2 = .517$). These last tests confirm the lack of decrement over trials and intensity effect in HR compared with the SCR profiles. Table 3.1 shows the stimulus-response pattern of HR.

### 3.4.3 Respiratory pause

Respiratory pause showed a linear decrement with trials ($F = 5.90, p = .029, \eta_p^2 = .297$), and recovery ($F = 7.51, p = .016, \eta_p^2 = .349$), but no significant dishabituation. No intensity effect was found. Figure 3.3 shows these results. Since RP failed to show dishabituation, RP was tested against SCR for these trials, yielding a non-significant Measure (RP vs. SCR) × Trial (12 vs. 10) interaction, indicating that RP and SCR did not differ.

![Figure 3.3. Respiratory Pause as a function of trials and intensity for both intensities, with SE bars. The linear regression line and Coefficient of Determination over the first 10 trials are also indicated.](image)
not differ significantly on dishabituation. Also, no difference was detected between the RP and SCR for the (RP vs. SCR) \( \times \) Intensity (60 vs. 80 dB) \( \times \) Trial (11 vs. 10) interaction. The stimulus-response pattern of RP is depicted in Table 3.1.

### 3.4.4 Peripheral vasoconstriction

Figure 3.4A shows a vasoconstriction starting at approx. 4 beats after stimulus onset and reaching a maximum between the 8th and 9th beat. The peripheral vasoconstriction was characterised by a significant quadratic trend over beats 4 to 11 \( (F = 7.96, p = .014, \eta_p^2 = .363) \). There was no significant decrement over trials (Figure 3.4B). An intensity effect was apparent (Intensity (60 vs. 80 dB) \( \times \) Trial (11 vs. 10): \( F = 11.34, p = .005, \eta_p^2 = .448 \)), with the louder tones providing greater vasoconstriction.

The finding of no decrement over trials was confirmed by a significant Measure (PVC vs. SCR) \( \times \) Trial (linear trend over 1-10) interaction: \( F = 16.43, p = .001, \eta_p^2 = .540 \).

See Table 3.1 for the stimulus-response pattern for PVC.

---

![Figure 3.4. Panel A: The mean phasic vasoconstriction as a function of poststimulus beats. Vertical scale: relative % constriction; horizontal scale: beats. Panel B: The mean phasic vasoconstriction over trials for condition. Vertical scale: relative % vasoconstriction; horizontal scale: trials. Beat point variation is indicated by SE bars. The linear regression line and Coefficient of Determination over the first 10 trials are also indicated.](image)
3.4.5 ERP components

The P1, N1, P3 and SW can be seen as clear peaks in the raw, EOG-corrected data of Figure 3.5.

The first 10 factors from the PCA decomposition over the -100–550 ms latency range, explaining 89.7% of the total variance, were tentatively identified on the basis of peak latencies and topography: Factor 1 – P3b at 307 ms (37.7% of total variance), Factor 2 – Slow Wave (SW) at 512 ms (25.2% of total variance), Factor 3 – N1-1 at 121 ms (7.5% of total variance), Factor 4 – P2 at 203 ms (6.3% of total variance), Factor 5 – Processing Negativity (PN) at 162 ms (3.8% of total variance), Factor 7 – P1 at 64 ms (2.0% of total variance), Factor 9 – P3a at 236 ms (1.5% of total variance), and Factor 10 – HabP3 at 354 ms (1.0% of total variance). A novel component, Factor 6 at 391 ms (3.1% of total variance), because of its sensitivity to intensity (see later), was labelled IntP3. Another novel component at 29 ms (1.6% of total variance) – Factor 8 – was identified as Na, because it showed frontal negativity (McCallum et al., 1983) and peak latency between 20 and 30 ms (Althen et al., 2011).

Virtual ERPs (the sum of these 10 components) over condition for 12 trials are shown on the right of Figure 3.5; the virtual ERPs closely approximate the original ERP grand means at the midline. Correlations were performed between the virtual and raw ERP waveforms for each condition at each of the midline sites; the lowest correlation was $r = .966$, $p < .001$ (at Fz for the 60 dB tone) demonstrating the close match. The rescaled factor loadings over time along with grand mean topographies of these 10 components are presented in Figure 3.6. Figure 3.7 shows the topography of the ERP components/subcomponents across condition for the trials of interest in depicting habituation. In Figure 3.8 these ERP components are presented for trials 10 and 11 at
each intensity, indicating the topographic differences for intensity and recovery. Figure 3.9 depicts the 10 PCA-derived component amplitudes (pooled at the maximal sagittal region [frontal, central, or parietal]) as a function of trials at each condition.

Figure 3.5. The left panel shows the grand mean ERPs across subjects and over intensities and across 12 trials at each of the midline sites, with well-defined components labelled at Fz and Cz. The right panel depicts the corresponding virtual ERPs derived from the sum of the first ten virtual PCA components/subcomponents over intensities and across 12 trials. The actual and virtual ERPs correspond very closely. Vertical scale: μV; horizontal scale: ms.
3.4.5.1 Na (Frontal negative maximum)

Na showed a central reduction (C < F/P: $F = 7.34, p < .05, \eta_p^2 = .334$), with a midline enhancement (M > L/R: $F = 9.45, p = .008, \eta_p^2 = .403$). The frontal activity showed no trial effects. No intensity effect was found at the change trial (trial 11 vs. 10). Comparison of Na and SCR over trials yielded a significant Measure (Na vs. SCR) × Trial (linear trend over 1-10) interaction: $F = 27.39, p < .001, \eta_p^2 = .662$. No significant intensity difference between Na and SCR for trial 11 vs. 10 emerged.

3.4.5.2 P1 (Central positive maximum)

The P1 was greatest over the central sites (C > F/P: $F = 22.07, p < .001, \eta_p^2 = .612$) and displayed a strong midline enhancement (M > L/R: $F = 10.34, p = .006, \eta_p^2 = .425$). These effects interacted in an enhancement of amplitude at the vertex (C > F/P × M > L/R: $F = 9.53, p = .008, \eta_p^2 = .405$) and the midline activity was enhanced over the
Figure 3.7. The headmaps of 10 PCA-derived ERPs from 19 sites are shown as a function of focal trials. Maximum averages at the midline for each component are marked by a grey ellipse.
frontal region \((F > P \times M > L/R: F = 16.33, p = .001, \eta_p^2 = .538)\). The central activity showed no decrement over 10 trials. No intensity effect was found (trial 11 vs. 10). The lack of decrement was confirmed by the comparison of P1 and SCR over trials, yielding a significant Measure \((P1 \text{ vs. } SCR) \times \text{Trial (linear trend over 1–10)}\) interaction: \(F = 19.10, p = .001, \eta_p^2 = .577\). The comparison between P1 and SCR for trial 11 vs. 10 to assess the intensity difference found a significant Measure \((P1 \text{ vs. } SCR) \times \text{Intensity (60 vs. 80 dB)} \times \text{Trial (11 vs. 10)}\) interaction: \(F = 8.63, p = .011, \eta_p^2 = .381\).

### 3.4.5.3 N1-1 (Central negative maximum)

Figure 3.6 shows a centrally dominant N1-1 \((C > F/P: F = 19.57, p = .001, \eta_p^2 = .583)\) with a midline topography \((M > L/R: F = 8.20, p = .013, \eta_p^2 = .370)\), interacting in a distinct enhanced amplitude at the vertex \((C > F/P \times M > L/R: F = 5.42, p = .035, \eta_p^2 = .279)\). In addition, the difference between frontal and parietal activity was greater over the left hemisphere than the right hemisphere \((F < P \times L > R: F = 5.64, p = .032, \eta_p^2 = .287)\). No trial effect was found over the first 10 trials. No intensity effect emerged for the recovery trial (11 vs. 10). A significant Measure \((N1-1 \text{ vs. } SCR) \times \text{Trial (linear trend over 1–10)}\) interaction: \(F = 46.38, p < .001, \eta_p^2 = .768\) confirmed the difference between N1-1 and SCR over trials. Comparison of N1-1 and SCR for trial 11 vs. 10 yielded no significant difference for intensity, see Figure 3.9.

### 3.4.5.4 PN (Frontal negative maximum)

PN exhibited a frontal topography \((F > P: F = 10.10, p < .007, \eta_p^2 = .419)\), particularly over the midline \((F > P \times M > L/R: F = 21.73, p < .001, \eta_p^2 = .608)\). The frontal maximum failed to decrement over trials. A significant Measure \((PN \text{ vs. } SCR) \times \text{Trial (linear trend over and SCR over trials1–10)}\) interaction: \(F = 25.46, p < .001, \eta_p^2 = .645\) confirmed the difference between PN and SCR over trials. The comparison
between PN and SCR for trial 10 vs. 11 to examine intensity found a marginal Measure (PN vs. SCR) \times Intensity (60 vs. 80 dB) \times Trial (11 vs. 10) interaction: \( F = 4.05, p = .064, \eta_p^2 = .225 \), but as the change from a softer tone to a louder tone elicited a smaller PN response, this is likely to be a genuine difference.

3.4.5.5 P2 (Central positive maximum)

P2 displayed a fronto-central topography (F > P: \( F = 8.58, p = .011, \eta_p^2 = .380 \); C > F/P: \( F = 22.26, p < .001, \eta_p^2 = .614 \)) with a marginally enhanced midline (M > L/R: \( F = 3.88, p = .069, \eta_p^2 = .217 \)). These effects interacted, showing enhanced positive activity at the vertex (C > F/P \times M > L/R: \( F = 17.14, p = .001, \eta_p^2 = .550 \)). The central activity showed an increment over trials (\( F = 7.12, p = .018, \eta_p^2 = .337 \)). There was no intensity \times trial interaction for recovery to indicate an intensity effect. Comparison of trials for P2 and SCR yielded a significant Measure (P2 vs. SCR) \times Trial (linear trend over 1–10) interaction: \( F = 40.65, p < .001, \eta_p^2 = .743 \). The comparison between P2 and SCR for trial 11 vs. 10 found a marginal Measure (P2 vs. SCR) \times Intensity (60 vs. 80 dB) \times Trial (11 vs. 10) interaction: \( F = 4.17, p = .060, \eta_p^2 = .230 \), but since the louder tones produced a lesser P2 this marginal result is likely to represent a significant difference (see Figure 3.9).

3.4.5.6 P3a (Central positive maximum)

P3a showed a central topography (C > F/P: \( F = 6.52, p = .023, \eta_p^2 = .318 \)) and strong midline activity (M > L/R: \( F = 11.35, p = .005, \eta_p^2 = .448 \)). The sagittal and lateral main effects interacted indicating a prominent vertex peak (C > F/P \times M > L/R: \( F = 21.30, p < .001, \eta_p^2 = .603 \)). Responses were greater over the left than the right hemisphere (L > R: \( F = 6.13, p = .027, \eta_p^2 = .305 \), and the difference in activity between frontal and parietal regions was greater in the left hemisphere (F > P \times L > R: \( F = 6.12, \eta_p^2 = .305 \)).
Figure 3.8. The headmaps of 10 PCA-derived ERPs from 19 sites are shown as a function of pre-change and change trial. Maximum averages at the midline for each component are marked by a grey ellipse.
Figure 3.9. Pooled maximum amplitudes of the 10 PCA-derived ERPs at the sagittal regions over intensity and trials. Time point variation is indicated by SE bars. Coefficient of Determination for the first 10 trials is included.
$p = .027, \eta^2 = .304$). Central activity failed to decrement over the first 10 trials. No significant intensity effect emerged for recovery. The difference between P3a and SCR over the first 10 trials was indicated by a significant Measure (P3a vs. SCR) $\times$ Trial (linear trend over 1–10) interaction: $F = 22.16, p < .001, \eta^2 = .613$. The comparison between P3a and SCR found the measure $\times$ intensity $\times$ trial interaction for trial 11 vs. 10 to be non-significant.

### 3.4.5.7 P3b (Central positive maximum)

P3b displayed a parieto-central dominance ($P > F$: $F = 7.32, p = .017, \eta^2 = .343$; $C > F/P$: $F = 10.92, p = .005, \eta^2 = .438$), strong midline activity ($M > L/R$: $F = 26.43, p < .001, \eta^2 = .654$), and was enhanced over the left hemisphere ($L > R$: $F = 15.46, p = .002, \eta^2 = .525$). Averaged central responses decremented over trials ($F = 13.96, p = .002, \eta^2 = .500$). No significant recovery was detected for trial 11 vs. 10 ($F = 5.21, p = .056, \eta^2 = .427$) but an intensity effect was apparent as an interaction for the recovery trial (Intensity (60 vs. 80 dB) $\times$ Trial (11 vs. 10): $F = 8.95, p = .010, \eta^2 = .390$), see Figures 3.8 and 3.9. Comparison of P3b and SCR for recovery found no difference (P3b vs. SCR) $\times$ Trial (11 vs. 10) interaction: $F = 3.82, p = .092, \eta^2 = .353$); the subsequent comparison of P3b and SCR undertaken for the dishabituation trial (12 vs. 10) also resulted in no significant difference.

### 3.4.5.8 HabP3 (Parietal positive maximum on the first trial)

HabP3 exhibited a central reduction ($C < F/P$: $F = 19.46, p = .001, \eta^2 = .581$) that was larger over the right than the left hemisphere ($C < F/P \times L < R$: $F = 9.08, p = .009, \eta^2 = .393$). The midline activity showed the greatest amplitude at the parietal region ($F < P \times M > L/R$: $F = 5.16, p = .039, \eta^2 = .270$). Parietal activity diminished over trials ($F = 5.53, p = .034, \eta^2 = .283$), but recovery (trial 11 vs. 10) and intensity
effects were not observed. Comparison of HabP3 and SCR for the recovery showed no significant difference, but some difference was found for the dishabituation interaction (HabP3 vs. SCR) × Trial (12 vs. 10): $F = 3.82$, $p = .071$, $\eta^2_p = .215$). Since no intensity effect was noted, HabP3 and SCR were compared for an intensity effect; a marginal difference was detected: Measure (HabP3 vs. SCR) × Intensity (60 vs. 80 dB) × Trial (11 vs. 10) interaction: $F = 3.66$, $p = .077$, $\eta^2_p = .207$.

### 3.4.5.9 IntP3 (Frontal maximum on the first trial)

IntP3 displayed central (C < F/P: $F = 10.33$, $p = .006$, $\eta^2_p = .424$) and midline (M < L/R: $F = 4.52$, $p = .052$, $\eta^2_p = .244$) reductions. These effects interacted, indicating minimal positive activity at the vertex (C < F/P × M > L/R: $F = 18.13$, $p = .001$, $\eta^2_p = .564$). The difference between the parietal and frontal region was larger over the right than the left hemisphere (P > F × L < R: $F = 5.50$, $p = .034$, $\eta^2_p = .281$). Frontal activity diminished over trials ($F = 6.66$, $p = .022$, $\eta^2_p = .322$). No recovery was found but an intensity effect emerged (Intensity × Trial (11 vs. 10): $F = 7.77$, $p = .015$, $\eta^2_p = .357$), see Figures 3.8 and 3.9. A comparison of IntP3 and SCR for recovery (trial 11 vs. 10) showed no significant difference, but an (IntP3 vs. SCR) × Trial (12 vs. 10) interaction: $F = 5.34$, $p = .037$, $\eta^2_p = .276$ emerged for dishabituation.

### 3.4.5.10 Parietal SW (Parietal maximum)

SW displayed a negative frontal and positive parietal topography and a central reduction (F < P: $F = 64.07$, $p < .001$, $\eta^2_p = .821$; C < F/P: $F = 12.82$, $p = .003$, $\eta^2_p = .478$). Reduced activity was found over the midline (M < L/R: $F = 6.91$, $p = .020$, $\eta^2_p = .331$), and these last two effects interacted, producing minimal positive activity at the vertex (C < F/P × M > L/R: $F = 6.54$, $p = .023$, $\eta^2_p = .319$). Midline activity was greatest over the parietal region (F < P × M > L/R: $F = 8.94$, $p = .009$, $\eta^2_p = .374$).
Responses were greater over the right hemisphere than the left hemisphere ($L < R: F = 7.13, p = .018, \eta^2_p = .337$). The parietal activity diminished over trials ($F = 8.75, p = .010, \eta^2_p = .385$). Apparent recovery and intensity effects are seen in Figure 3.9, but neither of these effects reached significance. However, no difference was found between parietal SW and SCR for recovery and dishabituation, nor did these measures differ in their responses to intensity.

3.4.6 Correlations exploring patterns among measures

Three possible relationship patterns were identified (see Table 3.1) and examined with three groups of correlations, each group containing an autonomic measure. Correlations were calculated across subjects’ means over groups and 12 trials. Pattern 1 was characterised by no decrement or intensity effect, and represented by HR and P1, PN, and P2. Significant correlations for HR with both PN and P2 supported the inclusion of these measures. Pattern 2 also showed no decrement, but an intensity effect, and was represented by PVC, Na, N1-1, and P3a. Pattern 3 demonstrates the major defining aspects of OR habituation and an intensity effect; this group included SCR, RP, P3b, HabP3, IntP3, and SW. Table 3.2 displays the correlations between the patterns over trials (12) and intensity (2) were calculated using across-subjects mean for measures in the 3 patterns with their defining ANS measure (SCR, HR, and PVC). RP, P3b, HabP3, IntP3, and SW were shown to have an association with SCR. HR showed significant correlations to both PN and P2. PVC exhibited no significant correlation with any measure. Since these incorporate an unavoidable mix of within and between subject data that may lead to spurious correlations arising from differences in group means, scatterplots are presented of the highest significantly-correlated measures from each pattern to examine if group clustering of data occurred. Figure 3.10 shows no evidence of group clustering for SCR and P3b, or for HR and PN. That is, there is no
systematic pattern of group means around the trend line representing the total correlation.

Table 3.2. Pearson product-moment correlation coefficients between the measures examined and the three patterns over groups and 12 trials.

<table>
<thead>
<tr>
<th>PATTERNS</th>
<th>SCR</th>
<th>HR</th>
<th>PVC</th>
</tr>
</thead>
<tbody>
<tr>
<td>RP</td>
<td>.487**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Na</td>
<td></td>
<td>.144</td>
<td>.223</td>
</tr>
<tr>
<td>P1</td>
<td></td>
<td></td>
<td>.210</td>
</tr>
<tr>
<td>N1-1</td>
<td></td>
<td>.566**</td>
<td></td>
</tr>
<tr>
<td>PN</td>
<td></td>
<td>.511**</td>
<td></td>
</tr>
<tr>
<td>P2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>P3a</td>
<td></td>
<td>.182</td>
<td></td>
</tr>
<tr>
<td>P3b</td>
<td>.710***</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HabP3</td>
<td>.467*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IntP3</td>
<td>.495**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Parietal SW</td>
<td>.676***</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*p < .05; ** p < .01; *** p < .001. Tests are one-tailed. Grey shading represents correlations not examined.

3.4.7 Examination of the proposed patterns

In order to examine the summary-patterns and their inclusions, PCA was conducted using z-scores of the means from the 4 autonomic and 10 ERP measures over groups and 12 trials. Varimax rotation was employed to maintain orthogonality and facilitate interpretation. This PCA yielded 3 components with Eigenvalues greater than 1, and these 3 components explained 60.4% of the total variance. Table 3.3 depicts the loadings of each measure on the 3 components and the variance explained. Component 1 appears to capture aspects of novelty and intensity and corresponds to Pattern 3. While SCR, RP, P3b, and SW are common, HabP3 and IntP3 show a large negative loading on Component 2 that is difficult to interpret. Component 1 also includes PVC (possibly due to the intensity element common to SCR and PVC). Component 2 is maximally correlated with all the measures of Pattern 1, reflecting neither novelty nor intensity. Component 3 appears to align with intensity rather than novelty. The P3a and
Figure 3.10. Panel A: Scatterplot between mean z-scores for SCR and P3b. The open circles represent group 1 and filled circles group 2. The linear regression line and Coefficient of Determination of the total correlation over condition, and group are included. Panel B: Scatterplot between mean z-scores for HR and PN. The open circles represent group 1 and filled circles group 2. The linear regression line and Coefficient of Determination of the total correlation over condition, and group are included.

N1-1 load substantially on this component and fall within Pattern 2, but PVC has only some association. The Na has no strong positive correlation with any component, however a small positive loading is evident on Component 1.
3.4.8 Temporal comparison of PCA-derived components between studies

Figure 3.11 depicts the topographies of the ERP components in the latency range between 0 and 500 ms from the present and previous studies, showing the Congruence Component, $r_c$. Lorenzo-Seva and ten Berge (2006) extended the rule-of-thumb evaluation, suggesting that $r_c$ from .85 – .94 indicates “fair similarity” and $r_c > .95$ indicates “good similarity”, i.e., the components can be considered equal. Inspection of Figure 3.11 reveals equivalence of the PN, P3b, and SW over the studies, while P1, N1-1, and P2 demonstrate a close degree of similarity. The low coefficient of the P3a may be attributed to the large 28 ms difference between the two versions of the waveform, and the difference associated with intensity variation here. The P3bs and the SWs were found to be equivalent. Although the difference in SW latencies is quite large, both are broad, substantial ERPs extending over a considerable time range. The IntP3 had no counterpart in the previous study, however the very low coefficient for it and the HabP3 supports its distinction from the HabP3 and its identification as a separate entity.

Table 3.3 Correlations between the 14 measures and 3 PCA-derived components. Bold represents the most extreme loading of each measure on a component.
3.5 Discussion

This dishabituation paradigm employing moderate intensities and very long ISIs was well suited to examine autonomic measures traditionally related to the OR. Novelty and intensity were systematically varied. Few investigations have used the dishabituation paradigm to examine both novelty and intensity from the OR perspective. Central and autonomic single-trial responses were obtained concurrently to permit collection of data from possible rapidly decrementing phasic responses (Roth, 1973 — left ear reference) as distinct from responses compromised by averaging (Ritter et al., 1968). Investigation of stimulus-response patterns based on the current data was scrutinised from the perspective of Preliminary Process Theory. Further, particular stimulus-response patterns were examined within this study by correlation, and between studies (for PCA derived components/subcomponents) using the Congruence coefficient. The quantitative analyses of the patterns were performed by the Interaction Test where each measure is compared statistically to the stimulus-response pattern of...
SCR – the accepted ‘yardstick’ of the phasic OR. Habituation was operationalised by response decrement (to novelty reduction), increased response on the change trial (to test recovery), and a subsequent increased response with respect to the original stimulus (to test dishabituation). A within subject intensity effect was represented as a significant intensity × trial interaction between trials 10 and 11.

3.5.1. Autonomic response patterns

The SCR demonstrated the three aspects of OR habituation: decrement, recovery, and dishabituation; an intensity effect was also evident on recovery where 80 dB tones yielded larger responses. These results mirror findings from other rare auditory dishabituation studies involving novelty (Barry and James, 1981a; Rushby et al., 2005; Steiner and Barry, 2011, 2014; Study 1) and intensity (Rushby et al., 2005). Consequently, the SCR stimulus-response pattern was deemed an appropriate benchmark for examining the phasic OR.

The brief phasic HR deceleration was represented as a quadratic trend over time that reached the maximum response within .1 s from stimulus onset. The apparent HR deceleration failed to decrement and no significant intensity effect was found. Both these results differed significantly from the SCR pattern, using the Interaction Test. This phasic HR deceleration has consistently been reported in previous investigations with no trial effect (Barry, 1977a, 1977b; Barry et al., 2011; Barry and James, 1981a, 1981b; MacDonald et al., 2012; Study 1) and no intensity sensitivity (Barry, 1977a, 1977b; Barry and James, 1981a; Lawrence and Barry, 2009; MacDonald et al., 2012). Together, the findings here and in previous studies indicate that HR deceleration is independent of novelty and intensity variation and represents an early stage of stimulus processing, according to PPT, pre-OR elicitation, i.e., functioning in physical transient detection.
Respiratory pause has received little investigatory attention, yet early reports suggest that RP decrements over trials (Barry, 1977a, 1977b; Barry and James, 1981b) with no intensity effect (Barry, 1977a, 1977b). Later studies have confirmed this pattern (Barry et al., 2013; MacDonald et al., 2012). Barry and James (1981a), using a dishabituation paradigm, reported an enhanced response for the change trial but no dishabituation, with no intensity interaction between the pre-change and change trials. In Study 1, RP was quantitatively compared with SCR (showing all three aspects of habituation) and reported no difference between the measures for recovery and dishabituation. This study partially supports previous findings: RP demonstrated decrement with recovery; RP and SCR did not differ significantly for dishabituation and intensity. Considering previous reports, RP likely displayed no dishabituation and no intensity effect, and more statistical power may have revealed such a difference; hence placement in Pattern 3 is considered provisional.

Peripheral vasoconstriction, like RP, has not been the focus of recent investigations. Barry (1977a,b) reported no trial but an intensity effect for repetitive stimuli. Barry and James (1981a), in a dishabituation paradigm, presented small and large white squares on a black background, and also found no trial effect, however a clear main effect of stimulus magnitude and a magnitude by trial interaction between the trials 10 and 11 for recovery. Recently Barry et al. (2013) examined PVC in a habituation paradigm where a repetitive series of alternating tones was presented, using stimuli of the same intensity as used here. PVC was insensitive to stimulus repetition but sensitive to intensity. We report no trials effect over the 10 trials, and an intensity effect for recovery. The comparison between PVC and SCR verified the absence of decrement. The autonomic measures exhibited the predicted pattern of results (although some ambiguity rests on the finding in RP for the dishabituation trial). Three stimulus-
response patterns can be identified for these autonomic measures: 1. No trial and no intensity effect (HR); 2. No trial but an intensity effect (PVC); and 3. Trial decrement, recovery, and dishabituation effects plus an intensity effect (SCR, and RP – the latter with an unlikely dishabituation and tentative intensity effect; see Figure 3.3).

3.5.2. ERP findings

We sought to find matching stimulus-response patterns in PCA derived ERP components/subcomponents. The phasic OR has associations with different bodily systems and an integrated OR response involving the autonomic system should find parallels in central measures. Consequently, novelty and intensity manipulations are likely to uncover ERP counterparts to those autonomic measures – exhibiting the same stimulus-response patterns. To date, subcomponents of the N1 have been associated with HR deceleration (Barry et al., 2011; Lawrence and Barry, 2009, 2010; MacDonald et al., 2012; Study 1), the LPC with SCR (Rushby et al., 2005; Steiner and Barry, 2011), P3a and P3b with HR deceleration and acceleration respectively (Lawrence and Barry, 2009; Rushby and Barry, 2009), and the ‘Novelty P3’/Hab P3 with respiratory pause (Rushby and Barry, 2009; Study 1).

Temporal PCA between -100ms and 550ms allowed us to identify the first 10 ERP components based on polarity, topography, and latency when using a linked-ears reference. The components were labelled tentatively in temporal order: Na, P1, N1-1, PN, P2, P3a, P3b, HabP3, IntP3, and SW; notably the common LPC subcomponents followed the same temporal sequence that has been reported in recent OR research: P3a, P3b, ‘Novelty P3/Hab P3, and SW (Barry, 2009; Barry et al., 2011, 2013; MacDonald et al., 2012; Rushby et al., 2005; Study 1).

The Na emerged as an unexpected PCA-derived factor. To our knowledge no
PCA decomposition from a habituation/dishabituation study has identified and reported this component. Study 1 found a factor of comparable topography and latency when novelty was varied but that factor was not investigated due to the very short latency and because middle-latency responses are not commonly linked to the OR. Middle-latency responses typically are marked by an anterior distribution (Althen et al., 2011; McCallum et al., 1983 — non-cephalic reference; Sonnadara et al., 2006) and characteristically elicited by click stimuli of short duration at fast presentation rates (Althen et al., 2011; Borgmann et al., 2001). McCallum et al. (1983) explored selection aspects of stimulus properties as indexed by ERP components in a target localisation task. The PCA-derived frontal component labelled Na was described as having a stronger association with stimulus rarity than target properties; probably rarity can be interchanged with novelty. Intensity effects have been reported for the Na-Pa complex (Althen et al., 2011), yet saturation occurs around 60 dB (Borgmann et al., 2001). Althen et al. (2011) posited that the Na represented an early automatic deviant detection process, similar to the conclusion reached by Sonnadara et al. (2006) — Cz reference. The Na in this study showed an anterior focus and a latency of 29 ms, longer than usually found (Neves et al., 2007), but this may reflect the longer ISIs and greater intensity employed here. The Na failed to demonstrate trial effects and no intensity effect was found. Na and SCR differed for trial and intensity effects. This component needs further attention in future OR studies.

The P1 has previously shown a fronto-central distribution (Beer and Röder, 2004 — right ear reference; Rushby and Barry, 2009; Study 1) and has recently been derived from a temporal PCA in a dishabituation investigation. This component failed to demonstrate a trial effect and its stimulus-response pattern differed significantly from SCR (Study 1). No trial effects were anticipated (Gillette et al., 1997; Pratt et al., 2008;
Study 1 but an intensity effect was not ruled out (White and Yee, 2006). The P1 exhibited a vertex distribution with frontal enhancement consonant with previous accounts; no trial effect over the first 10 trials and no intensity interaction over trials 10 and 11 were found. When P1 was compared to SCR it was significantly different for trials 1 to 10 and intensity.

The composite N1 has shown a vertex dominance (Vaughan and Ritter, 1970; Rushby and Barry, 2009; Barry et al., 2013; Squires et al., 1975) mimicked in its PCA-derived counterpart (Rushby and Barry, 2009; Barry et al., 2013; Squires et al., 1975; Study 1). The N1 has been found to be insensitive to novelty at long ISIs (Barry et al., 2011, 2013; Rushby and Barry, 2009). Increases in peak N1 amplitudes of the raw ERPs have been related to increments in intensity (Lawrence and Barry, 2010; MacDonald et al., 2012; Squires et al., 1975) and the PCA-derived N1 components have also exhibited a comparable intensity sensitivity (Barry et al., 2013; Squires et al., 1975). In two studies (Barry et al., 2011; Study 1) the identified N1-1 subcomponent showed a vertex topography; but a clear novelty insensitivity was found only in Barry et al. (2011). The N1-1 in this investigation displayed a vertex topography and no trial effects; also, the intensity x trial interaction failed to reach significance. When the N1-1 was compared to SCR for decrement over the first 10 trials they were found to differ, however no significant difference emerged between the measures on the intensity test; consequently, the possibility of N1-1 having intensity sensitivity remains. Figure 3.9 indicates that increases in intensity elicited larger responses and decrements led to reduced responses, supporting the notion that the non-significant intensity effect may have resulted from too little power.

The PN is characterised by a frontal and midline distribution (Näätänen, 1982). In an OR context, the temporal PCA-derivative presents the same topography (Barry et
al., 2011; Study 1). In OR investigations, the PN has demonstrated decrement (Barry et al., 2011) and no decrement over trials (Study 1) at long ISIs. Barry et al. (2011) delivered tones in their last experimental session monaurally rather than binaurally (earlier in the session tones were delivered binaurally to the same subjects) to examine behavioural orienting operationalised as eye-movements left or right; the monaural tones may have attributed extra novelty to the stimuli. In Study 1 tones were presented binaurally to each subject throughout the session, but inspection of their Figure 7 shows a large response on the first stimulus presentation indicating the influence of novelty. This PN showed the typical fronto-midline topography. The frontal average response failed to decrement statistically, and no intensity effect was evident. Comparison between the PN and SCR disclosed a significant difference over trials and the intensity × trial interaction yielded a marginal difference. Inspection of Figure 3.8 shows the change to the 80 dB tone elicited a lesser response on trial 11. In contrast, the SCR demonstrated a greater response to the 80 dB on trial 11 consequently this was not considered a genuine intensity effect. Study 1 identified a PCA-derived PN that failed to decrement and differed from SCR over the first 10 trials, the authors speculate on the presence of an early attention-switching process. The PN here also appears linked to continuous attention switching that is triggered by each stimulus. The reason for a response decrease from trial 10 to trial 11 for both intensities is not clear.

The PCA-derived P2 component has previously demonstrated a central distribution (Orlebeke et al., 1989; Roth et al., 1982; Study 1) along with insensitivity to novelty (Crowley and Colrain, 2004; Rushby and Barry, 2009; Study 1). Some intensity sensitivity has been reported (Orlebeke et al., 1989; Roth et al., 1982). The P2 component in this study demonstrated a central distribution matching previous accounts, and displayed an increase in response over trials, consistent with Study 1. Contrary to
prediction, the manipulation of intensity did not produce an intensity enhanced response on trial 11; rather, both intensities showed decreased response. P2 and SCR differed significantly over trials 1 to 10, while a marginal difference was evident between the measures for intensity.

The P3a has consistently shown a central topography (Barry et al., 2011; Rushby and Barry, 2009; Rushby et al., 2005; Squires et al., 1975; Study 1). As a PCA-derived component trial effects have been observed at long ISIs, together with an intensity dependent recovery (Rushby et al., 2005), however at very long ISIs trial effects have been absent (Rushby and Barry, 2009; Barry et al., 2011; Study 1). Intensity increments as well as decrements can elicit a P3a (Rushby et al., 2005; Squires et al., 1975). Our P3a had a typical central-midline distribution. No trial effect was evident and P3a differed significantly from SCR. An intensity effect for recovery was not significant, however P3a statistically matched SCR. This is compatible with the P3a marking both stimulus intensity increments and decrements, see Figures 3.7 and 3.8.

The parietal P3b has emerged as a prominent PCA-derived component in OR related studies (Barry et al., 2013; Roth et al., 1982; Squires et al., 1975; Study 1). The topography of our P3b and its trial decrement were consonant with previous OR studies (Barry et al., 2011; Rushby et al., 2005; Study 1). As predicted, response recovery failed to reach significance, so dishabituation was not tested, however the predicted intensity effect was evident (see Figure 3.9). We are not aware of any other OR investigation at long ISIs that has reported this important result. Comparison with SCR for recovery and dishabituation yielded no differences; suggesting, together with the intensity effect, that the possibility remains for the P3b to be a central OR marker.

Few studies have addressed a temporal PCA-derived component’s response to
novelty in a dishabituation protocol using simple stimuli. The response to a change in a stimulus parameter (recovery), as distinct from the first response, is of critical importance from an OR perspective. In habituation investigations, a PCA-derived “novelty P3” has shown a variety of distributions and responses to stimulus repetition: an initial vertex maximum that decremented (Barry et al., 2011), indistinct topography reducing over trials (Rushby and Barry, 2009), and a parietal topography failing to decrement significantly (Barry et al., 2013). In Study 1 this is labelled HabP3 in recognition that it has not been conclusively identified as Courchesne’s novelty component. In that investigation, the HabP3 showed a fronto-central topography and the activity of Fz diminished over trials and displayed recovery. No difference was found between HabP3 and SCR; these two measures had the same stimulus-response pattern.

At shorter ISIs, Rushby et al. (2005) identified a temporal PCA-derived Novelty P3 independent of intensity but sensitive to novelty elicited after the P3b. This component showed a slight parietal topography, trial decrement, recovery, and dishabituation at the parietal sites. Our parietal HabP3 matched the “Novelty P3” of Rushby et al. (2005) and Barry et al. (2013) for topography and diminished over 10 trials as predicted, but the increased response for recovery was not significant, contrasting to Study 1 Also, in accordance with expectations, no intensity effect emerged – consistent with previous habituation investigations (Barry et al., 2013; Rushby et al., 2005). Rather, the 60 dB tone elicited the greater response. Comparison with SCR for recovery revealed no difference, and a marginal difference for dishabituation (i.e., dishabituation cannot be ruled out). The HabP3 was compared with SCR for intensity, and a marginal difference was found that was likely due to a lack of power. Trial decrement appears well established along with intensity insensitivity for this post-P3b component, but response to stimulus change and response re-establishment is not clear; exploring this aspect of
The IntP3 was a parietal positivity with a vertex negativity. The initial frontal positive activity decremented and showed no recovery (no difference was found between recovery for IntP3 and SCR), yet dishabituation differed from SCR. A clear intensity effect was evident at recovery (see Figure 3.8). The generation of this component resulted from both novelty and intensity manipulation at very long ISIs. We are not aware of any reports of a PCA-derived ERP component in this latency range sensitive to both novelty and intensity, consequently speculations are tentative, requiring support from comparable OR investigations in the future.

OR research from our laboratory has previously yielded the frontally negative and parietally positive classic SW (Barry et al., 2011, 2013; Rushby et al., 2005; Study 1). Generally, the SW has shown decrement as a main effect or a topographic interaction (Barry et al., 2011; Rushby et al., 2005; Zimmer and Demmel, 2000; Study 1), with no recovery (Study 1). Response enhancement from an intensity increment has not been a recognised aspect of the SW (Roth et al., 1982, 1984; Rushby et al., 2005). This classic SW displayed the expected decrement over trials and no recovery. A lack of intensity sensitivity was also observed. Interestingly, no difference was found between the SW and SCR for recovery, dishabituation, or intensity; these aspects would benefit greatly by increased subject numbers. Inspection of Figures 3.8 and 3.9 indicates the directional similarity of these OR and intensity aspects of the SW and SCR demonstrated by the Interaction Test. The SW cannot be ruled out as a central index for the OR by these data.

**3.5.3. ERP component patterns**

The ERP components/subcomponents can be tentatively matched with the
autonomic stimulus-response patterns noted above. Pattern 1 is characterised by insensitivity to both novelty and intensity: HR, P1, PN, and P2. The clustering of P1, PN, and P2 in the first stimulus-response pattern further supports the findings of Study 1. The first pattern appears to reflect an early stage of stimulus processing in the OR context, such as transient detection.

Pattern 2 shows no decrement but intensity sensitivity: PVC, Na, N1-1, and P3a. The manipulation of intensity has grouped both the N1-1 and P3a in the second stimulus-response pattern with PVC; the P3a as a PCA-derived component has an intensity linkage (Rushby et al., 2005; Squires et al., 1975), as does the N1 (Barry et al., 2013). Few studies have separated the composite N1 into separate subcomponents such as the N1-1 using temporal PCA. While Näätänen and Picton (1987) assert all N1 subcomponents have some intensity sensitivity, the relationship between the N1-1 and intensity requires further validation in the OR context.

Pattern 3 demonstrates all the defining aspects of habituation and intensity sensitivity represented consistently by SCR: SCR, RP, P3b, HabP3, IntP3, and SW. The correlation analysis supports the inclusion of P3b, IntP3, and SW in the third pattern hallmarked by an appreciable decrement and response recovery (intensity interaction and/or showing no significant difference from SCR). Interestingly, Study 1 reported RP decrement (also reported in MacDonald et al. 2012) and recovery, albeit marginally, and an association between the stimulus-response pattern of RP and SCR. It is not surprising that these measures cluster since decrement and recovery reflect changes in novelty. The finding of no difference for intensity and dishabituation trial response between RP and SCR in this study is unclear but may be due to lack of statistical power.

The placement of the Na in Pattern 2 and IntP3 in Pattern 3 are provisional;
future OR research employing temporal PCA decomposition and intensity variation may provide insight into the ‘reality’ of these components, optimally by replication. Further investigations concerning the variation of novelty and intensity in dishabituation protocols with greater N would clarify the placement of these ERP components in the stimulus-response patterns.

Inclusion of the measures into the 3 patterns was partially supported by correlation analysis (unaffected by group clustering), and a summarising PCA. SCR, RP, P3b, and SW were associated by correlation and inclusion in Component 1 (associated with novelty and intensity). PVC showed its maximal loading on Component 1, although some loading was noted on Component 3 (intensity related). The IntP3 and HabP3 also correlated significantly with SCR and had some loadings on Component 1, however their major loading (a large negative loading) was on Component 2 (unrelated to novelty and intensity); a finding difficult to conceptualise. The P2 and PN correlated significantly with HR and all four measures of Pattern 1 matched exactly the measures loading on Component 2. No significant correlation was found between PVC and Na/N1-1/P3a, however only N1-1 and P3a loaded substantially on Component 3. The 3 PCA-derived components appear to mimic 3 modules of processing in PPT: stimulus registration (sensitive to neither novelty nor intensity), magnitude register (sensitive to intensity but not novelty), and OR generation (sensitive to both novelty and intensity).

Congruence coefficients provided some insight into the between studies consistency of the various PCA-derived components elicited. It should be noted that the PCAs differed between the studies in epoch length. This would be expected to reduce the congruence coefficients somewhat, but despite this, very high congruence was obtained for the common components. Early latency ERPs relate strongly to physical
parameters of the eliciting stimulus while later components are characterised by more subjective variables. Consequently, the early latency ERPs may be considered ‘obligatory’ and so less variable in occurrence and response, and the later ERPs more prone to fluctuations of subjective variables such as attention (Donchin et al., 1978).

The P3b and SW exhibited the strongest congruence between studies, and the early ERP components the weaker. Squires et al. (1975) referred to the P3b-SW complex and the commonality of their eliciting conditions: probability and task variables; the extended duration of the P3b and SW, along with their overlap, contributes to the high degree of similarity over studies. Consequently, aspects of the OR paradigm appear to consistently ensure the strong presence of both the P3b and SW. The Na, P3a, and HabP3 show small variances. These ERPs produced significant topographic effects that support their identification. Some differences between the studies in regard to the topography of specific ERPs may be attributed to the variation of intensity in the present investigation. The Na appears related to intensity, while the IntP3 relates to novelty together with intensity, yet we are not aware of these ERP components being identified in previous habituation studies, so inclusion into these patterns remains provisional.

Rushby et al. (2005) manipulated novelty, intensity, and Significance in a dishabituation paradigm at long ISIs (8 s) with SCR serving as the OR ‘yardstick’. The between subjects “Significance” was operationalised by requiring a button press to the change tone. It would be beneficial for PPT development to vary the OR determinant “Significance” in a similar paradigm incorporating more than one autonomic measure. This could be done by a within subjects manipulation of Significance (e.g., counting the stimuli in one condition but not another) in a dishabituation paradigm at very long ISIs.
3.6 Conclusion

Four autonomic measures and 10 temporal PCA-derived ERP components/subcomponents were examined in an auditory dishabituation paradigm at long ISIs with novelty and intensity varied. The three stimulus-response patterns that emerged were based on the defining aspects of habituation and intensity using SCR as the OR ‘yardstick’. Quantitative comparisons between the measures and SCR, along with correlation considerations, placed autonomic and ERP measures into three patterns. Each of the stimulus-response patterns includes at least one autonomic measure. The respiratory response has close links with novelty and in view of the paucity of research into autonomic measures relevant to the OR, further examination into the measurement of the respiratory response to novelty is advocated. However, this is beyond the scope of the present paper.

This dishabituation study has manipulated intensity in the context of the OR, building on the investigation of Study 1 where novelty was the only OR determinant varied. The temporal PCA uncovered the Na and IntP3, both of which have not been identified previously and appear to have an association with intensity. Both the P3b and SW also were shown to have an association with intensity. It is important to note that the latency order of LPC subcomponents followed the sequence: P3a, P3b, HabP3, and SW that has been consistently reported previously. The three stimulus-response patterns grouped the measures representing aspects of the OR. Future investigations manipulating Significance would supplement the development of PPT, a theory that encompasses the fractionation of measures relating to the OR, fractionation that was clearly evident in this study, and contrary to the notion of a unitary OR.
3.7 References

http://dx.doi.org/10.1371/journal.pone.0028522.

Barry, R.J., 1975. Low-intensity auditory stimulation and the GSR orienting response.
Physiol. Psychol. 3, 98–100.

Barry, R.J., 1977a. Failure to find evidence of the unitary OR concept with indifferent
low intensity auditory stimuli. Physiol. Psychol. 5, 89–96.

Barry, R.J., 1977b. The effect of “significance” upon indices of Sokolov's orienting
response: a new conceptualisation to replace the OR. Physiol. Psychol. 5, 209–
214.

Barry, R.J., 2009. Habituation of the orienting reflex and the development of

Barry, R.J., Furedy, J.J., 1993. Stimulus intensity and novelty interact in elicitation of

Barry, R.J., James, A.L., 1981a. Fractionation of phasic responses in a dishabituation

Barry, R.J., James, A.L., 1981b. Fractionation of respiratory and vascular responses
with simple visual stimulation. Physiol. Psychol. 9, 96–101.

Barry, R.J., Rushby, J.A., 2006. An orienting reflex perspective on anteriorisation of the
P3 of the event-related potential. Exp. Brain Res. 173, 539–545.


Näätänen, R., Picton, T., 1987. The N1 wave of the human electric and magnetic
response to sound: a review and an analysis of the component structure.
Psychophysiology 24, 375–425.

that acutely enhancing serotonin with the selective serotonin reuptake inhibitor
citalopram modulates the loudness dependence of the auditory evoked potential
Exp. 21, 47–52.

latency response study of auditory evoked potentials amplitude and latencies
audiologically normal individuals. Braz. J. Otorhinolaryngol. 73, 69–74.


Orlebeke, J.F., Kok, A., Zeillemaker, C.W., 1989. Disinhibition and the processing of
451.

between stimulus intensity and the P300. Psychophysiology 22, 326–329.

Polich, J., Ellerson, P.C., Cohen, J., 1996. P300, stimulus intensity, modality, and

111


Roth, W., Dorato, K.H., Kopell, B.S., 1984. Intensity and task effects on evoked physiological response to noise bursts. Psychophysiology 21, 446–481.


Chapter 4. Study 3: Significance and Novelty effects in single-trial ERP components and autonomic responses

Published as:

4.1 Abstract

The phasic orienting reflex (OR) was investigated in two counterbalanced blocks of an auditory dishabituation paradigm differing in stimulus Significance (operationalised as tone counting). Twelve tones were presented at very long, randomly-varying interstimulus intervals (ISIs). Novelty and Significance were varied within subjects. Stimulus-response patterns were assessed to find ERP matches for autonomic measures. The phasic OR index was represented by the skin conductance response (SCR). SCR decremented over 10 standard trials, showed recovery on trial 11 (change trial), enhancement to re-presentation of the standard tone (trial 12: dishabituation), and a main effect of Significance over the first 10 trials – demonstrating the formal criteria for an OR index. The evoked cardiac response (HR) subcomponents ECR1 (deceleration) and ECR2 (acceleration) showed no trial effects, but ECR2 showed a Significance effect. Respiratory pause (RP) decreased linearly over trials, and showed recovery, but no dishabituation or Significance effect. Temporal PCA was applied to single-trial EOG-corrected data. Ten ERP components were extracted: P1, N1-3, N1-1, PN, P2, P3a, P3b, HabP3, a Frontal Slow Wave (FSW), and the Classic SW. The dependent measures showed four distinct patterns. Pattern 1: No trial or Significance effects (ECR1, P1, N1-3, P3a, FSW); Pattern 2: No trial effect but a Significance effect (ECR2, N1-1, P2); Pattern 3: Trial but not Significance effects (RP, PN, P3b, HabP3); Pattern 4: Both trial and Significance effects (SCR and Classic SW). The evidenced fractionation of autonomic and central measures is compatible with Preliminary Process Theory (PPT), contrary to the notion of a unitary OR.
4.2 Introduction

4.2.1 Background

The preceding Chapters have focused on the involuntary OR, an outcome from the presentation of indifferent stimuli. The present Chapter offers further detail to that found in the General Introduction in regard to Significance and the voluntary OR.

Study 1 (MacDonald and Barry, 2014) found 3 stimulus-response patterns when novelty was varied. The first pattern was characterised by no decrement: HR deceleration, P1, N1-3, N1-1, PN, P2, and P3a; the second pattern, represented by the P3b and parietal SW, showed decrement but not recovery, so the measures in these patterns were not related to novelty. The third pattern demonstrated all the aspects of ‘genuine’ habituation: SCR and HabP3.

Study 2 (MacDonald et al., 2015) extended the examination of stimulus-related patterns to both novelty and intensity. Two PCA-derived components were identified that were not found in Study 1. The Na failed to decrement but there was not a significant difference between it and SCR in regard to intensity. The IntP3 had a peak latency between the HabP3 and Parietal SW; this component showed both decrement and intensity effects. The N1-3 present in Study 1 was not identified. Three patterns emerged, Pattern 1 (no decrement or intensity effect): HR deceleration, P1, PN, and P2 – these measures demonstrated the same grouping based on no decrement as in Study 1, again clustered together showing no decrement. The Na, N1-1, and P3a from Pattern 1 in Study 1, were grouped in Pattern 2, along with PVC, all showing no decrement but intensity sensitivity. Pattern 3 showed all the habituation characteristics and an intensity effect: SCR, RP, P3b, HabP3, IntP3, and SW. These studies demonstrate that autonomic fractionation found in early studies is evident also in ERP measures in comparable
paradigms.

4.2.2 Significance and the Voluntary OR

The voluntary OR is generated when the attributes of the stimulus change beyond the physical dimensions (Barry, 1984c, 1996), and the OR is enhanced. Instruction to count a series of stimuli silently and report the total imparts Significance (Barry, 1984a, 1984b, 1984c, 2004; Lacey and Lacey, 1980; Maltzman, 1979, 1990; Sokolov, 1963a). Although counting may involve aspects of memory and the counting process, the general attentional effect is compatible with the original Sokolovian concept of Significance, including the instruction to mentally count stimuli (Sokolov, 1963b). The ongoing process of counting stimuli and maintaining the count in memory represents cognitive load (e.g., Barry and Tremayne, 1987). Experimenter instructions induce a predisposing state (cortical set) that ‘steers’ attention to Significant stimuli, resulting in the voluntary OR. Directed attention manifests in prestimulus vigilance and/or poststimulus signal value. Significant stimuli have shown a slightly higher initial OR (Barry, 2004; Ben-Shakhar, 1980; Maltzman, 1990), that remains larger over trials (Barry, 2004; Iacono and Lykken, 1983; Maltzman, 1990; Steiner and Barry, 2011, 2014), with a slower decrement to repetition (Barry, 2004; Ben-Shakhar, 1980; Iacono and Lykken, 1983; Maltzman, 1990). Figure 4.1 depicts the sequential and parallel processing in PPT, modulated by Maltzman’s cortical set (Barry, 1984a, 1988; Maltzman, 1979, 1990). The processing units are reflected in the various measures enclosed in the dashed boxes.

4.2.3 Theoretical background of PPT

Preliminary Process Theory development has been guided by the realist Popperian scientific framework. A cornerstone of this framework is formation of new
and testable hypotheses with close adherence to empirical data (Barry, 2006, 2009).

The structural presentation of PPT follows the practice of depicting complex processing as serial/sequential and parallel linked modules integrating a time dimension. This form is consistent with information theory representation (Shannon, 1948; Shannon and Weaver, 1949). In addition, the structural representation appears consistent with the organisation of cortical (Neisser, 1967; Tononi, 2004) and neuronal levels of brain structure and activity (Brennan et al., 2012; Dimitrov et al., 2011; Tononi, 2004).

The theoretical structure of PPT incorporates two major elements: Dual-process theory (utilisation of non-cortical mechanisms) in OR generation, and Maltzman’s cortical set to explain Significance effects. Importantly, in theory building, Dual-process theory was integrated into the PPT theoretical structure instead of Sokolov’s neuronal model and stimulus comparator mechanism since fractionating responses could be accommodated and it offered greater predictive power. The chief basis of Sokolov for the neuronal model, the so-called “missing-stimulus effect”, was earlier negated by Barry (1984) and Barry and O’Gorman (1987). The assimilation of cortical set – the steering mechanism – permitted greater flexibility in hypothesis formulation, and was an advance on Sokolovian theory, which had no mechanism at all for Significance effects. Preliminary process theory makes specific predictions. For example, Barry (1981) drew a distinction between two aspects of Significance: prestimulus ‘vigilance’ and poststimulus ‘signal value’. Vigilance referred to anticipatory activity in readiness for stimulus onset, while signal value related to events after initial stimulus processing. Based on the observations that Significance enhanced responses and the sequential nature of OR processing, it was hypothesised that variation in these two Significance aspects should have differential effects. Specifically, vigilance manipulation should
enhance both HR deceleration and SCR, and manipulation of signal value should only enhance SCR. These two predictions were confirmed (Barry, 1981). Interesting, Barry (2006), mindful of theorist’s bias, evaluated PPT using the theory criteria outlined in Dennis and Kintsch (2007). The form of the sequential theoretical structure offers distinct advantages e.g., depicting time-dependent networks of relationships. Näätänen (1990) has embedded attentional-trace theory in a sequential structure comprised of stages of processing similar to PPT, however Näätänen’s representation covered only ERP and not autonomic processing. Preliminary Process Theory has undergone critical evaluation in regard to its theoretical structure (see Barry, 1987a, 1987b).

4.2.4 PPT in the context of other research fields

The four core stages of processing: stimulus, novelty, magnitude registration, and OR generation, remains the solid foundation on which to proceed into subsequent theory development. This strategic backdrop integrates autonomic and central measures in stages of processing for the first time – consistent with the OR representing a general systems response.

The theoretical structure of PPT, in this thesis, depicts cognitive perceptual processes as a separate stage; a later version of PPT labelled this stage as the ‘Executive’ (Barry, 2009). Research focus into the executive is advancing with a recognition of the intrinsic value of sharing terms, concepts, and techniques from different allied research fields. For example, Information theory has adopted the terms entropy (Shannon, 1948) and free energy from thermodynamics (Collell and Fauquet, 2015) as has cognitive neuroscience in the context of brain activity (Friston and Stephan, 2007), while information theory was later termed information-processing in cognitive neuroscience (Luce, 2003; Thornton, 2013).
Information theory concepts have been subsumed into emerging information-processing models of the brain, establishing close relationships with cognitive science (Fan, 2014; Luce, 2003), neuroscience (Brennan et al., 2012; Fan, 2014; Jung et al., 2014), and providing potentially an avenue for future research in OR/PPT. The brain can be conceived as a probabilistic inference multilevel hierarchy, functioning to construct models of possible causes that predict sensory inputs (De Bruin and Michael, 2016). The claimed aim of this system is to minimise prediction error: the discrepancy between the predicted (from the model) and actual input (Clarke, 2013; De Bruin and Michael, 2016). The notion of discrepancy (difference) is reminiscent of Sokolov’s neuronal model (Sokolov, 1960). The prediction error carries information into the ensuing higher-processing with minimal redundancy (Clarke, 2013). The prediction error can be understood in terms of surprise (Clarke, 2013; Collell and Fauquet, 2015; Donchin, 1981; Hohwy, 2012, 2016), free energy (Collell and Fauquet, 2015), novelty (De Bruin and Michael, 2016; Hohwy, 2012), and information entropy (Clarke, 2013). The brain attempts to minimise the prediction error (Clarke, 2013; Hohwy, 2012, 2016), surprise (Clarke, 2013; Friston and Stephan, 2007; Hohwy, 2012), and free energy (Friston and Stephan, 2007; Hohwy, 2012) via active inference (behavioural intervention) and/or perceptual interference (altering the model) (De Bruin and Michael, 2016). The cognitive control processing of prediction error minimisation (PEM) lies in the executive stage of processing in PPT. In principle, the quantification of surprise (novelty) (Barceló and Cooper, 2016; Fan, 2014) holds some promise for PPT development.

4.2.5 The present study

This study follows the recommendations of Barry et al. (2013) and builds on Study 1 and Study 2 by systematically varying novelty and Significance within subjects
in a dishabituation paradigm. Both autonomic and central measures are compared when Significance is varied within subjects, an additional variable added to the previous novelty manipulations. Temporal PCA derived ERP components were sought to match and extend the autonomic measures already linked to preliminary and OR processing.

Figure 4.1. A schematic of PPT. Sequential and parallel processing of indifferent stimulus information leads to the generation of the OR from the interaction of novelty and stimulus magnitude processing. The smaller, lighter dashed rectangles indicate measures indexing the processing of each of the modules. The larger, bolder dashed line represents the moderation of the modules by Maltzman’s cortical set. Physiological responses: HR decel, Heart rate deceleration; CVD, cephalic vasodilation; Resp, respiratory pause; EEG, EEG alpha desynchronisation; PVC, peripheral vasoconstriction; GSR, galvanic skin response (now SCR); HR accel, heart rate acceleration.

Within subjects responses are more sensitive than between subjects responses for detecting condition differences, due to less intrinsic error variance. Significance was operationalised as the counting of tones; the main effect of Significance will be examined as the difference between Count and No Count responses over the first 10 trials. We predict SCR will demonstrate the OR response pattern: response decrement to
standard stimulus presentations over trials, response enhancement at the change stimulus, and an increase to re-presentation of the standard stimulus (dishabituation) (Barry and James, 1981a; Rushby et al., 2005; Steiner and Barry, 2011; Study 1; Study 2). The increased response to counted stimuli should be evident as a main effect of Significance over the first 10 trials (Barry, 1981, 1982, 1988, 2004; Barry and Rushby, 2006; Steiner and Barry, 2011). The general ECR, with no prepared motor response requirement, should be represented as a biphasic waveform (Barry, 1988, 1996; Lawrence and Barry, 2009, 2010). The initial phasic HR deceleration (ECR1) is an obligatory response to all stimuli and should show no systematic variation over trials (Barry, 1977b, 1982, 1984a, 1984d, 1986; Barry and James, 1981a; Barry et al., 2011; Study 1; Study 2) or Significance main effect (Barry, 1977b, 1981, 1982; Lawrence and Barry, 2009). The cardiac response to a Significant stimulus is largely acceleratory and is considered to reflect the obligatory ECR1 overlaid by a hypothetical ECR2. Because ECR2 cannot be seen separately from ECR1, it is estimated from the late acceleration. ECR2 should not decrement, but may show a slight increase over trials if cognitive load increases (Barry, 1984a, 1984d, 1996), and should reflect increased Significance from counting (Barry, 1984a, 1984d; Kaiser et al., 2001; Lawrence and Barry, 2009, 2010). A substantial respiratory pause should be evident for the first stimulus, show a linear decrement over trials (Barry, 1981, 1982; Barry and James, 1981a, 1981b; Barry et al., 2013; Study 1; Study 2), demonstrate recovery at the change stimulus (Barry and James, 1981a; Study 1; Study 2), but no increased response for the dishabituation trial (Barry and James, 1981a; Study 1; Study 2). Respiratory responses for counted stimuli are not expected to be enhanced (Barry, 1977b, 1981, 1982).

ERPs relevant to the phasic preliminary and OR processing as depicted by PPT should fall within the 600 ms latency range used previously for the temporal PCA. The
P1 should be observed clearly in the raw data, emerge as a PCA-derived component, and show no decrement (Rushby and Barry, 2009; Study 1; Study 2). No main effect of Significance is expected (Boutros and Belger, 1999; Courchesne et al., 1975; Picton and Hillyard, 1974; Rushby and Barry, 2009). The N1 is not expected to show a trials effect (Barry et al., 2011, 2013; Rushby and Barry, 2009; Study 1; Study 2) or a Significance effect (Lawrence and Barry, 2009; Näätänen, 1988) over the first 10 trials. The PN PCA-derivative exhibits a frontal topography with a narrow peak within the accepted PN latency range. Trial effects have been equivocal: decrement was found by Barry et al. (2011) but not Study 1 or Study 2. To our knowledge no investigations have varied novelty and Significance jointly to yield a PCA-derived PN, consequently there are no predictions in regard to trial effects and Significance. The P2 is not expected to decrement over trials at these very long ISIs (Crowley and Colrain, 2004; Romero and Polich, 1996; Rushby and Barry, 2009; Study 1; Study 2) and no increase in the Count condition is expected (Rushby and Barry, 2009; Becker and Shapiro, 1980; Crowley and Colrain, 2004; Squires et al., 1975).

In regard to the LPC subcomponents, the P3a should show no trials effects (Barry et al., 2011; Pritchard, 1981; Rushby and Barry, 2009; Steiner et al., 2014; Study 1; Study 2). At very long ISIs the P3a aligns more closely with physical parameters (Study 1; Study 2) than cognitive elements, consequently no Significance effect is expected. Our predictions for the posterior P3b are based on results from OR protocols that incorporate counting of repeated identical stimuli of a fixed number. Steiner et al. (2014), in an auditory dishabituation paradigm at long ISIs, found that counting enhanced the P3b. In that study no motor response was required and the P3b was derived from temporal PCA. Simple manipulation of Significance in the OR context is not common. Trial decrement has been shown under similar conditions (Barry et al.,
The P3b response should thus diminish over trials with no recovery and be enhanced for counted stimuli. The PCA-derived HabP3 subcomponent has proved sensitive to the first presentation of a simple stimulus – the ‘newness’ per se (Barry et al., 2011; Rushby and Barry, 2009; Study 1; Study 2). In habituation/dishabituation studies it has demonstrated a varied distribution. A parietally focused topography has been reported when intensity/Significance has been manipulated in conjunction with novelty (Barry et al., 2013; Rushby et al., 2005; Steiner et al., 2014; Study 2), this contrasts to the frontally focused topography found when novelty alone was varied (Barry et al., 2016; Study 1). Response decrement has been the defining feature of the HabP3 (Barry and Rushby, 2006; Barry et al., 2011; Rushby et al., 2005; Rushby and Barry, 2009; Steiner et al., 2014; Study 1; Study 2), but not Barry et al. (2013), along with some recovery (Rushby et al., 2005; Steiner et al., 2014; Study 1; Study 2), and possibly dishabituation (Rushby et al., 2005; Study 1). The observations of trial decrement and response recovery suggests novelty processing, indicating that novelty per se is the prime eliciting determinant. An effect of Significance has also been reported (Steiner et al., 2014) at long ISIs but the stimulus-response pattern of SCR was not available to affirm the Significance effect. Consequently, the HabP3 should show a trial decrement, possible recovery, but no main effect of Significance. The classic SW has been identified as the early component of the SW/O wave; the later broader component displays a general negative distribution (Rohrbaugh et al., 1978, 1984; Zimmer and Demmel, 2000). The SW has shown similarities to P3b, such as task and probability dependency, and overlaps the P3b in raw data (Donchin et al., 1978; Rohrbaugh et al., 1978; Squires et al., 1975). The classic SW has been associated with the OR (Loveless and Sandford, 1974; Rohrbaugh et al., 1984; Zimmer and Demmel, 2000). Therefore,
the SW should show a trial effect (Rushby et al., 2005; Zimmer, 2006; Zimmer and Demmel, 2000; Study 1; Study 2; but not Barry et al., 2011, 2013), but no response recovery (Study 1; Study 2). Greater SW responses are expected for counted stimuli with some right hemisphere enhancement (Rohrbaugh et al., 1984; Rushby et al., 2005; Zimmer and Demmel, 2000; Study 2).

The tonic measures of SCL and HRL have been included here to address the concern that some Significance effects may be attributed to non-specific state changes rather than stimulus-related Significance per se (Barry, 1982; O’Gorman, 1979). SCL has been used as a traditional arousal measure (Barry, 1982, 2004; Barry and Sokolov, 1993), whereas HRL has been suggested as a more appropriate measure of preparatory prestimulus vigilance (Barry, 1996, 2006; Tremayne and Barry, 1990, 2001).

4.3 Methods

4.3.1 Participants

Participants were selected as in previous Studies, except that 32 volunteers participated here (ages 18–60, mean 22.8 years; 25 female; 26 right-handed).

4.3.2 Procedure

In order to examine novelty and Significance variation, auditory stimuli of 80 dB tones at 1000 and 1500 Hz, with a duration of 50 ms (plus 15 ms rise/fall times) were presented at a randomly variable ISI of 50–70 s. Novelty reduction was operationalised by trial repetition. Participants received 10 tones at one frequency (standard), a change trial at the other frequency, and the original tone was re-presented on trial 12. For each participant zero, 1, or 2 standard tones were then randomly added to reduce the participants’ ability to communicate details of the tone sequence. The
standard/change frequencies were counterbalanced between participants. The procedure was identical to that in Study 1, except that each participant completed two tasks. In task 1 they either ignored or counted stimuli (No Count or Count), in task 2 the order was reversed. The initial condition in task 1 was counterbalanced between participants.

4.3.3 Physiological recording

The details of the physiological recording of Electrodermal activity, Heart activity, Respiration, and the Electroencephalogram are identical to the descriptions of section 2.3.3. PVC was not recorded.

4.3.4 Data extraction

The criteria for data extraction for SCR, Respiratory pause and ERPs are presented in section 2.3.4 of Chapter 2. This study includes heart rate acceleration (ECR2). This measure has been shown to be sensitive to Significance variation.

4.3.4.1 Evoked cardiac response

A locally produced R-wave peak detection program that computed the R-R intervals in ms was employed to analyse EKG data. Cardiac activity was calculated in terms of mean values of HR for 0.5 s intervals relative to event onset (Velden and Wölk, 1987). Each epoch of data commenced 2 s before stimulus onset and ended 5 s after stimulus onset. The phasic ECR consists of two additive components, an initial deceleration (ECR1) common to all stimuli, and an acceleratory ECR2 to significant stimuli. The ECR2 is reflected in the increased acceleration in the Count compared to No Count conditions. HR deceleration (ECR1) was defined as the maximum decrease in HR in the time range -.25–1.75 s relative to prestimulus HR over the 12 trials. The longer latency ECR2 was measured as the maximum increase in HR in the time range
2.25–4.75 s over the 12 trials.

4.3.5 Statistical analysis

Single-trial EOG-corrected data were submitted to a temporal PCA in the time range of -100–600 ms from 19 scalp locations across 32 subjects to derive and identify ERP components/subcomponents. A total of 14,592 cases (32 subjects ×12 trials× 2 conditions ×19 sites) were submitted to PCA decomposition utilising Dien’s ERP PCA toolkit (v. 2.23; Dien, 2010) in Matlab. The temporal PCA employed the covariance matrix; the number of ERP components obtained equalled the number of variables/time points (358 points). Kaiser normalisation was employed and Varimax rotation was applied to all components to maintain orthogonality and facilitate interpretation. Virtual ERP component amplitudes were calculated from the product of factor loadings, factor scores, and standard deviations and these were used for subsequent analysis. The virtual amplitudes for each identified component were subjected to a separate analysis of variance (MANOVA). Nine central sites represented topography, and the coronal plane [left (F3, C3, P3), midline (Fz, Cz, Pz) and right (F4, C4, P4)] and sagittal plane [frontal (F3, Fz, F4), central (C3, Cz, C4) and parietal (P3, Pz, P4)] were the repeated-measures factors. Planned contrasts within the coronal plane compared the left (L) vs. right (R) hemispheres, along with the midline (M) vs. the mean of the left and right hemispheres. In addition, the contrasts within the sagittal plane, frontal (F) vs. parietal (P) regions, and central (C) region vs. the mean of the frontal and parietal regions, were also analysed. These orthogonal planned contrasts and their interactions provide optimal, non-redundant information on the topographic distribution of the amplitude of each component.

The pooled maximum amplitude for each ERP component across one of three
medial regions (frontal, central or parietal regions) was used for the subsequent analysis. All autonomic measures (including HR deceleration and acceleration) and ERP component maxima were examined separately for response decrement and Significance. Repeated measures multivariate analysis of variance (MANOVA) was used to examine the autonomic and ERP measures with the factors Trials (for trials 1 to 10) and Significance (No Count vs. Count). Within Trials, decrement was examined by a linear trend. Responses for the change trial (recovery: trial 11 vs. 10) were investigated in separate MANOVAs if significant decrement over trials was found. Likewise, responses for the re-presentation trial (dishabituation: trial 12 vs. 10) were only examined if recovery on the change trial proved significant. Also, the within subjects Significance effect over the first 10 trials was examined for a main effect. A within subjects Significance effect from the OR perspective would be indicated by an increase in response magnitude for counted stimuli (Count) in comparison to indifferent stimuli (No Count). The analysis of the ECR over a 5 s epoch also included a Time factor with planned comparisons to examine the linear, quadratic, and cubic trends over Time that define ECR. ECR1 and ECR2 were analysed by separate repeated measures MANOVAs with the factors Trials (1–10) and Significance (Count/No Count); only the linear trend was examined to assess decrement. To test state differences between the levels of Significance, 2 separate one-way MANOVAs for SCL and HRL were employed.

Since counterbalancing was employed, possible carry-over effects may occur, and this was tested by separate MANOVAs for trials (1 to 10), recovery (trial 11 vs. 10, and dishabituation (trial 12 vs. 10) for SCR. Both a between subject factor Order (Count first/No Count first) representing order of presentation and the within-subject factor Significance (Count/No Count) were included in these analyses. Carry-over effects
would be indicated by a significant main effect or interaction involving Order.

The Interaction Test examined differences between SCR and RP/ECR1/ECR2, along with SCR and the virtual ERP components, with separate repeated measures MANOVAs using Z-scores for each subject’s measures. In the case of ERPs, a subject’s ERP Z-scores represented the maximum mean amplitudes at either the frontal, central, or parietal region for each component; these were used for the comparison analysis. The phasic SCR was expected to represent the benchmark OR pattern: decrement, recovery, and dishabituation, plus a Significance main effect over 10 trials. In order to capture the essence of the brief novelty influence, the measure comparison to SCR analysis is examined over the first five trials where decrement is expected to be most substantial (Barry, 2004; Barry and Rushby, 2006). If a difference was found between the measure and SCR in that pattern aspect for its initial analyses (e.g., no trial effect for ECR1 and trial effect for SCR), the difference in that pattern aspect was tested with a repeated-measure MANOVA for that measure and SCR, over the relevant trials. The factor interaction: Measure × decrement/recovery/dishabituation and/or measure × Significance was examined to test the observed differences. A significant interaction confirmed the difference between the measure and SCR for the pattern aspect; and subsequent pattern testing was not required. A non-significant interaction was interpreted as indicating that aspect of the pattern failed to differ significantly from SCR, consequently the next aspect of the stimulus response pattern was tested. The polarity was reversed for negative ERP components to ensure decrement, recovery, and dishabituation, were tested appropriately.

Congruence Coefficients \((r_c)\) were calculated for the common PCA derived components of the present study and those of Study 1 in the latency range from 0 to 500 ms. The Congruence Coefficient indicates the degree of similarity and stability across
experimental conditions based on latency, rise/fall times, and response magnitude rather than topographical similarities (Barry et al., 2014). No Bonferroni-type adjustment to $\alpha$ was necessary (Tabachnick and Fidell, 1989) since all contrasts were planned and the number of contrasts did not exceed the degrees of freedom for effect. Since single degree of freedom contrasts are unaffected by the violations of sphericity assumptions common in repeated-measures analyses of physiological data, Greenhouse-Geisser type correction was not necessary (O’Brien and Kaiser, 1985). All the reported tests have $(1, 31)$ degrees of freedom. The effect sizes ($\eta_p^2$) are suitably indicated.

**4.4 Results**

No major artifacts in the autonomic or EEG data rendered specific data unusable; consequently, data from all subjects were analysed appropriately.

**4.4.1 SCR**

The mean phasic SCR trace showed an onset latency of approx. 1.9 s and peak latency of approx. 4.1 s. Substantial response diminution over trials was apparent in a linear trend ($F = 100.45, p < .001, \eta_p^2 = .764$), evident in Figure 4.2, but no interaction with Significance was found. The SCR recovered to the change stimulus ($11 > 10: F = 14.24, p = .001, \eta_p^2 = .315$) and dishabituated to the re-presented original stimulus ($12 > 10: F = 10.96, p = .002, \eta_p^2 = .261$). The main effect of Significance was apparent over the first 10 trials ($F = 6.92, p = .013, \eta_p^2 = .182$). In the additional analyses checking for counterbalancing effects, no significant main effects of Order or any interactions involving Order were found. See Table 4.1 that summarises the Trials, Recovery, Dishabituation, and Significance results for each measure.
Figure 4.2. The square-root transformed phasic SCR as a function of trials and levels of 
Significance: NC (No Count) and C (Count). SE bars indicate the trial to trial variation, and the 
linear regression line and Coefficient of Determination for the first 10 trials are included.

4.4.2 ECRs

Figure 4.3A shows the ECRs (relative to pre-stimulus HR) for the No Count and 
Count conditions. Both conditions exhibit a brief simple cardiac deceleration, more 
prominent in the No Count responses (ECR1), with a peak latency of approximately .75 
s. The No Count response returns toward baseline, while the Count response overshoots 
the baseline with an additional rapid cardiac acceleration. The overall response profiles 
over the entire time interval can be accounted for by a marginal linear trend \((F = 3.60, p 
= .067, \eta_p^2 = .104)\) and a cubic trend \((F = 14.01, p = .001, \eta_p^2 = .331)\). The difference 
between conditions was indicated by a Significance by Time interaction (NC vs. C \(\times 
\) linear trend: \(F = 8.41, p = .007, \eta_p^2 = .213\), seen as a linear divergence of the condition 
responses. Count responses were generally larger (i.e., more positive) than No Count \((F 
= 5.98, p = .02, \eta_p^2 = .162)\). Subsequent analysis over the first 2.25 s revealed a 
significant quadratic trend \((F = 28.72, p < .001, \eta_p^2 = .481)\) that did not differ between
Table 4.1 Decrement, Recovery, Dishabituation and Significance effects for ANS and CNS (ERP) measures.

<table>
<thead>
<tr>
<th>Measure</th>
<th>Decrement</th>
<th>Recovery</th>
<th>Dishabituation</th>
<th>Significance</th>
<th>S-R pattern</th>
</tr>
</thead>
<tbody>
<tr>
<td>Autonomic</td>
<td>SCR</td>
<td>***</td>
<td>*</td>
<td></td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>ECR1</td>
<td>—</td>
<td>*</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>ECR2</td>
<td>—</td>
<td>**</td>
<td></td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>RP</td>
<td>**</td>
<td>**</td>
<td></td>
<td>3</td>
</tr>
<tr>
<td>ERP</td>
<td>P1</td>
<td>—</td>
<td>—</td>
<td>*</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>N1-3</td>
<td>—</td>
<td>—</td>
<td>**</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>N1-1</td>
<td>—</td>
<td>—</td>
<td></td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>PN</td>
<td>*</td>
<td>—</td>
<td>*</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>P2</td>
<td>—</td>
<td>*</td>
<td></td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>P3a</td>
<td>—</td>
<td>**</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>P3b</td>
<td>**</td>
<td>**</td>
<td>*</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>HabP3</td>
<td>**</td>
<td>—</td>
<td>**</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>FSW</td>
<td>—</td>
<td>—</td>
<td>*</td>
<td>1</td>
</tr>
</tbody>
</table>

* p < .05; ** p < .01; *** p < .001; — no significant result; grey shading represents measure-SCR interaction not tested

Placement of measures into the S-R patterns are determined on differences in responses according to aspects of habituation and Significance

conditions, representing the ECR1 component. The ERC2 is also included in Figure 4.3A, calculated as the difference between the Count and No Count responses. The ERC2 demonstrated no transient deceleration, but rapidly accelerated to reach peak latency approx. 3.75 s post-stimulus onset. Neither maximum HR deceleration (mean -1.39 BPM at .75 s) nor maximum acceleration (mean 1.82 BPM at 3.25 s) displayed response decrement (Figures 4.3B and C) or interacted with Significance. However, the difference between Count and No Count (ECR2), in contrast to ECR1, was represented by a main effect of Significance over the first 10 trials ($F = 8.00, p = .008, \eta^2_p = .205$) reflecting the additional acceleration to that in the No Count condition. Comparison of both components and SCR for trials yielded significant Measure (ECR1/ECR2 vs. SCR) × Trial (linear trend over 1-5) interactions: $F = 62.91, p < .001, \eta^2_p = .670$ and $F = 73.44, p < .001, \eta^2_p = .703$) respectively. The difference between ECR1 and SCR for Significance was indicated by the Measure by Significance interaction (Measure (ECR1 vs. SCR) × Significance (NC vs. C) interaction: $F = 6.76, p = .014, \eta^2_p = .179$). HR component effects are depicted in Table 4.1.
Figure 4.3. Panel A. Mean phasic HR response for NC (No Count), C (Count), and estimated ECR2 (Count - No Count) relative to the pre-stimulus level across time. Vertical scale: change in beats per minute; horizontal scale: s. Panel B. Maximum HR deceleration over the 12 trials for both levels of Significance: NC (No Count) and C (Count). Vertical scale: Maximum HR deceleration in beats per minute; horizontal scale: trials. Panel C. Maximum HR acceleration over the 12 trials for levels of Significance NC: (No Count) and C (Count). Vertical scale: Maximum HR acceleration in beats per minute; horizontal scale: trials. Time point variation is indicated by SE bars. Linear regression line and Coefficient of Determination for the first 10 trials are included.

4.4.3 Respiratory pause

Respiratory pause decreased linearly over trials ($F = 10.07, p = .003, \eta_p^2 = .245$),
and demonstrated recovery ($F = 5.38, p = .027, \eta^2_p = .148$), but dishabituation failed to reach significance. No main effect of Significance or interaction with trials 1–10 was found. These results are depicted in Figure 4.4. The lack of dishabituation in RP was tested against SCR over trials 10 and 12; the Measure (RP vs. SCR) × Trial (12 vs. 10) interaction was non-significant, indicating that RP and SCR did not differ on dishabituation. However, these measures differed for Significance: Measure (RP vs. SCR) × Significance (NC vs. C) interaction: $F = 4.96, p = .033, \eta^2_p = .138$. Table 4.1 shows these results.

![Figure 4.4](image)

Figure 4.4. Respiratory Pause as a function of trials and levels of Significance: NC (No Count) and C (Count), with SE bars. The linear regression line and Coefficient of Determination over the first 10 trials are also indicated.

### 4.4.4 ERP components

The distinct peaks of P1, N1, P2, P3, and SW are labelled in the raw EOG-corrected mean ERP data of Figure 4.5.

The first 11 factors from the PCA decomposition over the -100–600 ms latency range
Figure 4.5. The left panel shows the grand mean ERPs across subjects, levels of Significance: NC (No Count) and C (Count), and across 12 trials at each of the midline sites, with well-defined components labelled at Cz. The right panel depicts the corresponding virtual ERPs derived from the sum of the first ten virtual PCA components/subcomponents over Significance and across 12 trials. The actual and virtual ERPs correspond very closely. Vertical scale: μV; horizontal scale: ms.
each carried at least 2% of the variance and were examined, but one factor (Factor 8), a likely artifact of PCA processing at the end of the latency range, was excluded from analysis. The remaining ten components/subcomponents explained 87.1% of the variance and were tentatively identified with respect to their peak latencies and topography: Factor 1 – classic SW at 535 ms (32.8% of total variance), Factor 2 – P3b at 296 ms (21.1% of total variance), Factor 3 – P2 at 215 ms (9.2% of total variance), Factor 4 – N1-1 at 117 ms (6.8% of total variance), Factor 5 – Processing Negativity (PN) at 162 ms (6.0% of total variance), Factor 6 – N1-3 at 80 ms (2.5% of total variance), Factor 7 – HabP3 at 369 ms (2.4% of total variance), Factor 9 – FSW at 410 ms (2.2% of total variance), Factor 10 – P1 at 43 ms (2.1% of total variance), and Factor 11 – P3a at 252 ms (2.0% of total variance).

The Virtual ERPs consisted of the sum of these 10 identified ERP components over conditions for the 12 trials. Figure 4.5 shows the comparisons of the virtual and the actual ERPs. The grand means of the virtual ERPs mirror those of the raw ERPs at the midline regions Fz, Cz, and Pz.

The rescaled factor loadings of the 10 identified components/subcomponents are presented in Figure 4.6 over the time range. The grand headmaps for each component is placed above each maximum point of the corresponding peak, together with the peak latencies. The topography of the ERP component/subcomponents over the relevant trials and the overall mean for each condition are displayed in Figure 4.7. Table 4.1 summarises the Trials, Recovery, Dishabituation, and Significance results for each measure. Differences between SCR and measures are statistically tested when a non-significant aspect of a measure differs from that aspect of SCR, these results are included along with the stimulus-response pattern assigned to each measure. Figure 4.8 depicts the 10 PCA-derived component amplitudes (pooled at the maximal midline
region [frontal, central, or parietal]) as a function of trials 1 to 12 for each condition.

Figure 4.6. Rescaled factor loadings for the 10 PCA-derived ERPs. Each of the 10 factor loading peaks corresponds to the topographic grand mean headmap of that component along with its peak latency. Vertical scale: μV; horizontal scale: ms.

4.4.4.1 P1 (Central positive maximum)

The P1 amplitude was greatest over the central region (C > F/P: $F = 64.84$, $p < .001$, $\eta_p^2 = .677$), and midline activity was enhanced over the frontal region (F > P × M > L/R: $F = 6.76$, $p = .014$, $\eta_p^2 = .179$). No Trial or Significance main effects emerged, and there was no interaction between these variables. The lack of decrement was confirmed by the comparison of P1 and SCR yielding a significant Measure (P1 vs. SCR) × Trial (linear trend over 1–5) interaction: $F = 50.07$, $p < .001$, $\eta_p^2 = .618$. The comparison between P1 and SCR to assess Significance found a significant Measure (P1 vs. SCR) × Significance (NC vs. C) interaction: $F = 4.85$, $p = .035$, $\eta_p^2 = .135$. 

137
4.4.4.2 N1-3 (Parietal negative maximum)

The N1-3 had a parietal topography ($F < P$: $F = 16.03$, $p < .001$, $\eta^2_p = .341$) and

<table>
<thead>
<tr>
<th>Condition</th>
<th>Trial 1</th>
<th>Trial 10</th>
<th>Trial 11</th>
<th>Trial 12</th>
<th>Overall</th>
</tr>
</thead>
<tbody>
<tr>
<td>P1</td>
<td>NC</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>C</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>N1-3</td>
<td>NC</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>C</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>N1-1</td>
<td>NC</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>C</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PN</td>
<td>NC</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>C</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>P2</td>
<td>NC</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>C</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>P3a</td>
<td>NC</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>C</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>P3b</td>
<td>NC</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>C</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HabP3</td>
<td>NC</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>C</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>FSW</td>
<td>NC</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>C</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SW</td>
<td>NC</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>C</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Figure 4.7. The headmaps of 10 PCA-derived ERPs from 19 sites over levels of Significance: NC (No Count) and C (Count) are shown as a function of focal trials and overall topography. Maximum averages at the midline for each component are marked by a grey ellipse.
Figure 4.8. Pooled maximum amplitudes of the 10 PCA-derived ERPs at the sagittal regions over levels of Significance: NC (No Count) and C (Count) and trials. Time point variation is indicated by SE bars. Coefficient of Determination for the first 10 trials is included.
midline reduction (M < L/R: $F = 21.45, p < .001, \eta^2_p = .409$). The latter effect is greater in the parietal region (F < P × M < L/R: $F = 13.50, p = .001, \eta^2_p = .303$), with minimal negative activity at the vertex (C > F/P × M < L/R: $F = 22.50, p < .001, \eta^2_p = .421$). The difference in activity between the frontal and parietal regions was greater in the left hemisphere (F < P × L > R: $F = 9.98, p = .004, \eta^2_p = .243$). No trial or Significance main effect, or their interaction, was found. A significant Measure (N1-3 vs. SCR) × Trial (linear trend over 1-5) interaction: $F = 53.10, p < .001, \eta^2_p = .631$ confirmed these measures’ difference in decrement. Also, the difference in Significance was confirmed by a Measure (N1-3 vs. SCR) × Significance (NC vs. C) interaction: $F = 8.83, p = .006, \eta^2_p = .222$.

4.4.4.3 N1-1 (Central negative maximum)

Figure 4.7 displays the N1-1 with a strong central focus (C > F/P: $F = 155.21, p < .001, \eta^2_p = .834$) and a midline topography (M > L/R: $F = 120.71, p < .001, \eta^2_p = .796$); these effects interacted, reflecting an enhanced amplitude at the vertex (C > F/P × M > L/R: $F = 81.26, p < .001, \eta^2_p = .724$). In addition, the midline activity in the frontal region (F > P × M > L/R: $F = 22.43, p < .001, \eta^2_p = .420$), and the central enhancement was larger in the left than the right hemisphere (C > F/P × L > R: $F = 5.79, p = .022, \eta^2_p = .157$). No Trial or Significance main effect, or their interaction, was found over the first 10 trials. A significant Measure (N1-1 vs. SCR) × Trial (linear trend over 1–5) interaction: $F = 70.40, p < .001, \eta^2_p = .694$ confirmed the difference between N1-1 and SCR over the first 5 trials. No significant Measure by Significance interaction was found.

4.4.4.4 PN (Frontal negative maximum)

PN was shown to be frontally dominant (F > P: $F = 5.13, p = .031, \eta^2_p = .142$).
especially over the midline ($F > P \times M > L/R$: $F = 15.04, p = .001, \eta^2_p = .327$). The difference between the central and the mean of the frontal and parietal regions was greater in the left hemisphere ($C > F/P \times L > R$: $F = 11.11, p = .002, \eta^2_p = .264$). The frontal pooled maximum amplitude decremented over trials ($F = 5.33, p = .028, \eta^2_p = .147$), however no effect of Significance was apparent, nor interaction with Trial.

Recovery (trial 11 vs. 10) was not observed. Comparison of PN and SCR for recovery found a significant interaction: Measure (PN vs. SCR) $\times$ Trial (11 vs. 10): $F = 4.41, p = .044, \eta^2_p = .125$). A significant Measure (PN vs. SCR) $\times$ Significance (NC vs. C) interaction: $F = 4.97, p = .033, \eta^2_p = .138$ confirmed the difference between PN and SCR for Significance.

**4.4.4.5 P2 (Central positive maximum)**

P2 showed a fronto-central topography ($F > P$: $F = 30.34, p < .001, \eta^2_p = .494$; $C > F/P$: $F = 51.86, p < .001, \eta^2_p = .626$) with strong midline activity ($M > L/R$: $F = 63.19, p < .001, \eta^2_p = .670$). These effects interacted to produce maximum positive activity at the vertex ($C > F/P \times M > L/R$: $F = 66.61, p < .001, \eta^2_p = .682$) and frontal midline regions ($F > P \times M > L/R$: $F = 5.11, p = .031, \eta^2_p = .142$). In addition, responses were greater in the left hemisphere than the right hemisphere ($L > R$: $F = 9.64, p = .004, \eta^2_p = .237$, especially in the central region ($C > F/P \times L > R$: $F = 6.56, p = .015, \eta^2_p = .175$). The central activity failed to decrement over trials, however a Significance main effect was evident ($F = 4.36, p = .045, \eta^2_p = .123$) but there was no interaction with Trial. Comparison for P2 and SCR over trials yielded a significant Measure (P2 vs. SCR) $\times$ Trial (linear trend over 1–5) interaction: $F = 55.52, p < .001, \eta^2_p = .641$.

**4.4.4.6. P3a (Central positive maximum)**

P3a was characterised by elevated central ($C > F/P$: $F = 16.76, p < .001, \eta^2_p = .
midline activity (M > L/R: \( F = 10.61, p = .003, \eta^2 = .255 \)). These effects interacted to display maximum amplitude at the vertex (C > F/P × M > L/R: \( F = 7.92, p = .008, \eta^2 = .204 \)). The midline activity was also enhanced in the frontal region (F > P × M > L/R: \( F = 35.23, p < .001, \eta^2 = .532 \)). No central decrement was observed over the first 10 trials and no Significance main effect, or interaction with Trial, was found.

P3a differed significantly from SCR over the first 5 trials: Measure (P3a vs. SCR) × Trial (linear trend over 1-5) interaction: \( F = 42.05, p < .001, \eta^2 = .576 \). Comparison between P3a and SCR for Significance yielded a significant difference: Measure (P3a vs. SCR) × Significance (NC vs. C) interaction: \( F = 8.78, p = .006, \eta^2 = .221 \).

4.4.4.7 P3b (Parietal positive maximum)

P3b showed a typical parieto-central topography (P > F: \( F = 21.51, p < .001, \eta^2 = .410 \); C > F/P: \( F = 13.50, p = .001, \eta^2 = .303 \)), and strong midline activity (M > L/R: \( F = 79.36, p < .001, \eta^2 = .719 \)). The latter effect was prominent in the parietal region (P > F × M > L/R: \( F = 35.23, p < .001, \eta^2 = .532 \)). Parietal responses decremented over trials (\( F = 9.36, p = .005, \eta^2 = .232 \)) but no effect of Significance, or interaction with Trial, was observed. No significant recovery was found, see Figures 4.7 and 4.8.

Comparison of P3b and SCR for recovery found a significant Measure (P3b vs. SCR) × Trial (11 vs. 10) interaction: \( F = 15.23, p < .001, \eta^2 = .329 \). P3b and SCR also differed on their responses to Significance: Measure (P3b vs. SCR) × Significance (NC vs. C) interaction: \( F = 4.70, p = .038, \eta^2 = .132 \).

4.4.4.8 HabP3 (Frontal positive maximum)

HabP3 showed a frontal topography (F > P: \( F = 5.07, p = .032, \eta^2 = .141 \)) with a central reduction (C < F/P: \( F = 59.63, p < .001, \eta^2 = .658 \)), both these effects were greater for the No Count condition: Significance (NC > C) × Sagittal (F > P): \( F = 9.45,\)
A midline reduction was also evident (M < L/R: F = 5.06, p = .032, \( \eta_p^2 = .140 \)). The central and midline reductions interacted to indicate minimal positive activity at the vertex C > F/P × M < L/R: F = 34.54, p < .001, \( \eta_p^2 = .527 \)). Averaged frontal activity reduced over trials (F = 12.29, p = .001, \( \eta_p^2 = .284 \)), but no recovery (trial 11 vs. 10) was observed. Responses for the No Count condition were generally larger than for the Count condition (F = 4.71, p = .038, \( \eta_p^2 = .132 \)), in the opposite direction to expectations; no interaction of this effect with Trial was found. A significant difference was found when HabP3 and SCR were compared for the recovery: Measure (HabP3 vs. SCR) × Trial (11 vs. 10): F = 9.68, p = .004, \( \eta_p^2 = .238 \)). A significant difference was also found when HabP3 and SCR were compared for Significance: Measure (HabP3 vs. SCR) × Significance (NC vs. C) interaction: F = 10.63, p = .003, \( \eta_p^2 = .255 \).

4.4.4.9 FSW (Frontal negative maximum)

Topographically, FSW was dominant in the frontal region (F > P: F = 11.54, p = .002, \( \eta_p^2 = .271 \)), and exhibited enhanced midline activity in the frontal region (F > P × M > L/R: F = 6.18, p = .019, \( \eta_p^2 = .166 \)). A central reduction was greater in the right hemisphere than the left hemisphere (C < F/P × L < R: F = 6.34, p = .017, \( \eta_p^2 = .169 \)). The No Count condition showed a greater amplitude in the frontal region: Significance (NC > C) × Sagittal (F > P): F = 7.02, p = .013, \( \eta_p^2 = .185 \), particularly in the midline region: Significance (NC > C) × Sagittal (F > P) × Lateral (M > L/R: F = 4.86, p = .035, \( \eta_p^2 = .136 \). Frontal activity failed to decrement, and no main effect of Significance or interaction with Trial was noted. The FSW was compared to SCR over the first 5 trials for decrement, and the difference proved significant: Measure (FSW vs. SCR) × Trial (linear trend over 1–5) interaction: F = 86.31, p < .001, \( \eta_p^2 = .736 \). The comparison
between FSW and SCR for Significance showed some difference: Measure (FSW vs. SCR) × Significance (NC vs. C): $F = 3.36, p = .076, \eta^2_p = .098$.

### 4.4.4.10 Classic SW (Parietal positive maximum)

SW exhibited the typical negative frontal and positive parietal topography with a central reduction ($F < P: F= 114.13, p < .001, \eta^2_p = .786; C < F/P: F = 10.53, p = .003, \eta^2_p = .253$). Midline activity was greatest over the parietal region ($F < P \times M > L/R: F= 19.88, p < .001, \eta^2_p = .391$). Positive activity was greater over the right hemisphere than the left hemisphere ($L < R: F = 7.71, p = .009, \eta^2_p = .199$). Over trials 1–10, positive activity diminished ($F = 22.73, p < .001, \eta^2_p = .423$), but recovery was not apparent. A Significance main effect emerged ($F = 4.68, p = .038, \eta^2_p = .131$), but no interaction with Trial was found. Comparison of Parietal SW and SCR for recovery (trial 11 vs. 10) yielded no significant difference. Subsequent examination of a difference between SW and SCR for dishabituation (trial 12 vs. 10) also found no significant difference.

### 4.4.5 Skin conductance and heart rate levels (arousal and vigilance)

SCL failed to differ between the conditions NC (M = 9.11, SD = 4.48 µS) and C (M = 9.45, SD = 4.83 µS), $F < 1$, nor was a difference found for HRL between NC (M = 72.79, SD = 7.92 BPM) and C (M = 72.21, SD = 8.36 BPM), $F < 1$.

### 4.4.6 Stimulus-response patterns of autonomic and ERP measures

Four stimulus-response patterns emerged based on the observed aspects of trial decrement and Significance for each autonomic and ERP measure. Inclusion of a measure into a pattern was also supported by the Interaction Test when required. Pattern 1: no decrement or Significance effect, represented by ECR1, P1, N1-3, P3a, and FSW. Pattern 2: no decrement, but a Significance effect, represented by ECR2, N1-1, and P2.
Pattern 3: decrement but no Significance effect, grouped the measures RP, PN, HabP3, and P3b. Pattern 4 embodies the major defining aspects of the OR, both decrement and Significance effects; this group contains SCR and SW.

4.4.7 Temporal comparison of PCA-derived ERP components between studies

Figure 4.9 depicts the topographies of the common ERP components identified in the latency range between 0 and 500 ms of this study and Study 1. The Congruence Coefficient, $r_c$, provides a quantitative measure of component similarity to affirm the visual correspondence. Lorenzo-Seva and ten Berge (2006) suggested that $r_c$ from .85–.94 indicates “fair similarity” and $r_c > .95$ indicates “good similarity”, i.e., the components can be considered equivalent. Figure 4.9 reveals equivalence of the N1-1, PN, P2, P3b, and SW over the studies, while the HabP3 and P1 correspond closely.

Figure 4.9. Topographic headmaps of PCA components from the present study and Chapter 2 are depicted. Latencies, % component variance, factor ranking, and Congruence coefficient are presented for comparison.
4.5 Discussion

Examination of the effects of novelty and Significance follows on logically from our two previous investigations, where Study 1 varied novelty alone, and Study 2 varied novelty and intensity jointly. Since the paradigm structure is comparable to those studies, the present study serves as a replication of the previous studies in regard to novelty. The manipulation of Significance was operationalised here by a simple counting task. A within subjects Significance effect was represented as an increased response for counting vs. non-counting over trials 1 to 10.

4.5.1 Autonomic response patterns

The three defining aspects of OR habituation were demonstrated by SCR: decrement, recovery, and dishabituation; a main effect of Significance was also observed over the first 10 trials. These results are in accord with findings involving novelty from other rare auditory dishabituation studies (Barry, 1981, 1982, 2004; Steiner and Barry, 2011, 2014) and instructions to count (Steiner and Barry, 2014; Steiner et al., 2014). Therefore, the stimulus-response pattern of all the other measures can be compared to that of the exemplar of the phasic OR – SCR. Additional testing confirmed the absence of effects due to order of presentation (counted vs. not counted blocks) in the SCR data, indicating that we achieved an unbiased within subject testing of Significance in this study. The overall cardiac response was represented by both linear and cubic trends over time that described the biphasic waveform (brief deceleration followed by an extended cardiac acceleration). Further analysis over the first 2.25 s yielded a strong quadratic trend, confirming the presence of the cardiac deceleration ECR1. A Significance by Time interaction indicated that the HR increased more for the Count than the No Count condition over time in a linear fashion. Both the
ECR1 and the subsequent acceleratory component failed to decrement, however only the acceleration demonstrated a Significance effect. Both ECR deceleratory and acceleratory components differed markedly from SCR in regard to trials, and ECR1 differed for Significance (Interaction Test). The phasic ECR1 has consistently shown no trial effect (Barry, 1977a, 1977b; Barry et al., 2011; Barry and James, 1981a; Study 1; Study 2) and no Significance dependency (Barry, 1977a, 1977b, 1982; Lawrence and Barry, 2009). Likewise, the overall later acceleratory component (ECR2) failed to diminish over trials, but interestingly, it apparently increased with trials; although not significant, this is directionally consistent with increasing processing load (Barry, 1996; Barry and Tremayne, 1987). ECR2 showed a substantial main effect of Significance; this cognitive load finding is consistent with previous reports (Barry, 1984a, 1984d; Kaiser et al., 2001; Lawrence and Barry, 2009, 2010). These findings for HR deceleration support the notion of the ECR1 functioning according to PPT at an early stage of stimulus processing, pre-OR elicitation, i.e., marking the physical transient detection. The longer latency ECR2, on the other hand, appears to relate to cognitive processing, developing over trials and marking observable cognitive load variation.

Respiratory pause is not a commonly utilised measure in OR investigations, even though it has been shown to be susceptible to changes in novelty (Sokolov, 1963a). The RP decrement was represented in a linear trend over trials, consistent with earlier accounts (Barry, 1977a, 1977b, 1981, 1982; Barry and James, 1981a, 1981b; Barry et al., 2013; Study 1; Study 2). A clear recovery was also observed, establishing the selectivity of stimulus processing to novelty (Study 1; Study 2). Dishabituation was absent, yet no significant difference was found between RP and SCR; this is consonant with recent reports (Study 1; Study 2). No Significance effect was found here, compatible with expectations from early work (Barry, 1977b, 1981, 1982). Study 1
compared RP with SCR with respect to all three aspects of habituation and reported no significant differences between this measure and SCR for recovery and dishabituation; Study 2 found recovery and no difference between these measures for dishabituation. These dishabituation studies, including the present one, demonstrate a non-significant increase in RP response on the dishabituation trial. Dishabituation remains a possibility according to the Niewenhaus test, but this may be due to low statistical power or greater subject variability at the end of the experimental session (from tiredness or a loss of attentional focus) and needs further investigation. Considering RP’s strong association with novelty per se in the absence of Significance sensitivity, RP appears to index processing of stimulus ‘newness’, intermediate between stimulus registration and OR generation. The RP is placed in Pattern 3 based on the clear trial effect in the absence of a Significance effect.

The autonomic measures generally confirmed previous results. Four stimulus-response patterns can be identified for these autonomic measures: 1. No trial decrement and no Significance effect (ECR1); 2. No trial decrement but a Significance effect (ECR2); 3. Trial decrement but no Significance effect (RP); and 4. Trial decrement and Significance effect (SCR). These patterns provided the template for the ERPs examined.

4.5.2 ERP findings

Temporal PCA between -100 ms and 600 ms permitted identification and analysis of 10 ERP components based on polarity, topography, and latency. The components were labelled tentatively in temporal order: P1, N1-3, N1-1, PN, P2, P3a, P3b, HabP3, FSW, and classic SW. Importantly, the common LPC subcomponents were arranged in the same temporal order consistently reported in recent OR investigations: P3a, P3b, ‘Novelty P3’/HabP3, and classic SW (Barry et al., 2011; Barry et al., 2013;
To our knowledge the PCA derived FSW has not been identified previously in dishabituation studies but the latency of this component places it before the classic SW; this needs further investigation in future studies. The P1 has exhibited a fronto-central topography (Beer and Röder, 2004 — right ear reference; Rushby and Barry, 2009; Study 1). Temporal PCA-derived P1 has failed to demonstrate a trial effect (Study 1; Study 1). No trial effects were expected (Gillette et al., 1997; Pratt et al., 2008) and no Significance sensitivity was predicted. The P1 here showed a central distribution dominant frontally in accord with previous reports; no decrement or main effect of Significance over the first 10 trials was evident. When P1 was compared to SCR for trial and Significance both comparisons proved significant. Placement of the P1 in Pattern 1 was based on no observable trial or Significance effect.

The N1-3 in the present study exhibited a parietal topography. Trials and Significance effects were not observed. This component has been identified as a PCA-derived component in a number of studies (Barry et al., 2011; Study 1). Barry et al. (2011), in a unique monaural auditory habituation study, examined horizontal eye movement toward the ear of stimulation as a measure of behavioural orienting, and SCR served as the phasic physiological OR index. The topography of the N1-3 was similar but more anterior to the present N1-3; the latency in that study was 77 ms, compared to 80 ms in the present study. The N1-3 found in Study 1 showed a parieto-central topography similar to that found here, but with a latency of 94 ms. In both these previous studies the N1-3 was elicited prior to the N1-1 and demonstrated no decrement, consistent with the findings here. Näätänen and Picton (1987) had described this component as displaying a vertex topography, but more parietal and widespread than the N1-1. Their functional significance was linked to an alerting capacity of
sensory association after quiescence. Future investigations in OR-type paradigms may provide greater insight into this component. The lack of decrement and insensitivity to Significance warrants the inclusion of N1-3 into Pattern 1.

The composite N1 can be characterised by a vertex dominance (Vaughan and Ritter, 1970; Rushby and Barry, 2009; Barry et al., 2013; Squires et al., 1975), and is echoed in the PCA-derived counterpart (Rushby and Barry, 2009; Barry et al., 2013; Squires et al., 1975; Study 1; Study 2). The N1 has been reported as novelty independent at long ISIs (Barry et al., 2011; Barry et al., 2013; Rushby and Barry, 2009). The first “fronto-central” component identified by Näätänen and Picton (1987, p. 386) reflects closely the composite N1 in regard to topography and associations to eliciting conditions (Näätänen and Picton, 1987). Both the composite N1 and the N1-1 component appear more reliant on the physical aspects of the stimulus than subsequent processing (Näätänen and Picton, 1987). In three studies (Barry et al., 2011 Study 1; Study 2) the identified N1-1 subcomponent showed a vertex topography; but was clearly insensitive to novelty. There are few studies examining Significance for the N1-1. Rushby and Barry (2009), in a dishabituation protocol at very long ISIs, found no effect of instruction, but this did not involve a counting task. Consequently, no effect of trial or Significance was predicted here. Our N1-1 displayed a vertex distribution and no significant trial effect; also a main effect of Significance failed to reach significance. Comparison of N1-1 to SCR for decrement over the first 5 trials revealed a significant difference, yet these measures did not differ significantly on the Significance test; consequently, the possibility of N1-1 having some Significance dependency remains open. Figure 4.8 indicates that counted stimuli had a larger response than non-counted stimuli, however a sizeable variability appears to overshadow this difference. Since N1-1 decrement was not evident and the Significance effect failed to differ from that of the
SCR, these results led to the N1-1 tentatively being included in Pattern 2. This placement needs further consideration in future work.

The PN is typified by a frontal and midline distribution (Näätänen, 1982). The temporal PCA-derivative presents the same topography in the OR context (Barry et al., 2011; Study 1; Study 2). In the Introduction PN was shown to have exhibited variable patterns over stimulus repetition, and the present findings add to those of previous studies suggesting the PN represents an early reflexive attention-switching process. In this study the frontally-dominant PN demonstrated a trial but not a Significance effect. Thus, the PN was tentatively assigned to Pattern 3.

The PCA-derived P2 component has previously shown a strong central topography (Study 1; Study 2) and resistance to decrement (Crowley and Colrain, 2004; Rushby and Barry, 2009; Study 1; Study 2). No trial or Significance effect was anticipated. The P2 component in this study showed a fronto-central topography consistent with recent reports (Study 1; Study 2). Interestingly, the response increased non-significantly over trials, consonant with Study 1, and with Study 2 where the increase was significant. A main effect of Significance over the first 10 trials was clearly evident. The Significance effect for the P2 was not predicted and literature provides no clear foundation for this observation, especially when arousal is discounted. The absence of a decrementing trial effect and presence of a Significance effect places P2 in Pattern 2.

The P3a has consistently exhibited a central distribution (Barry et al., 2011; Rushby and Barry, 2009; Rushby et al., 2005; Squires et al., 1975; Study 1; Study 2). Rushby et al. (2005) found the PCA-derived P3a to decrement over trials at long ISIs, but trial effects have been conspicuously absent at very long ISIs (Rushby and Barry,
Although there are some reports of Significance sensitivity (Rushby and Barry, 2009; Steiner et al., 2014), no trial or Significance effect was predicted; both these expectations were confirmed, and hence P3a was assigned to Pattern 1.

The parieto-central P3b has emerged as a conspicuous PCA-derived component in OR studies (Barry et al., 2013; Rushby and Barry, 2009; Rushby et al., 2005; Study 1; Study 2). Our parieto-central P3b showed decrement but no recovery, confirming predictions. We also predicted a Significance effect, but this was not evident and quite surprising. Both Figures 4.7 and 4.8 depict an initial difference in Significance that remains over the first two trials. It is possible that after some initial processing the greater proportion of the cognitive load related to counting was treated at a later stage, perhaps reflected in the SW. The finding of a decrement in the absence of a Significance effect warrants the P3b inclusion in Pattern 3.

In recent times, our laboratory has sought the novelty ERP response in single-trial data. While the decrement over trials in the HabP3 has been consistently found, the response to Significance variation has received scant attention. We expected the characteristic decrement with a possible recovery, but a Significance effect was not predicted. The frontal HabP3 found here matched the HabP3 of Study 1 for topography and decrement, but not recovery (a suggestion of frontal recovery can be seen in Figure 4.7). No Significance effect emerged, as predicted. Comparisons with SCR for recovery and Significance both proved significantly different. Since the HabP3 showed decrement but no Significance effect, the HabP3 was grouped in Pattern 3.

The FSW was an unexpected discovery in the latency range between the HabP3 and SW. The latency of 410 ms places it at the beginning of the broad SW yet it has a
topography different to the later classic SW. The FSW matches neither of the O-wave components in regard to topography or latency (Loveless and Sandford, 1974; Rohrbaugh et al., 1984; Zimmer and Demmel, 2000). The FSW in this study exhibited a frontal midline topography that failed to decrement. The No Count responses were greater than Count. SWs have shown some enhancement to novelty and Significance (e.g., Rohrbaugh et al., 1978) but this FSW has shown neither. The validity of this component requires support from further investigations under similar conditions using PCA. The lack of a trial or Significance effect places this component in Pattern 1.

The frontally-negative and parietally-positive classic SW has been regularly reported from our laboratory in recent OR investigations utilising temporal PCA (Barry et al., 2011; Barry et al., 2013; Rushby et al., 2005; Study 1; Study 2). Such a SW occurring near the end of an epoch may simply be an outcome of the autocorrelation of EEG time-series data (Kayser and Tenke, 2003), but this component with similar topography has been reported at similar latencies over 150 ms before the end of longer PCA epochs (Rushby et al., 2005; Barry et al., 2011). Generally, the SW, in raw data and as a temporal PCA component, has demonstrated decrement as a main effect or a topographic interaction (Barry et al., 2011; Rushby et al., 2005; Zimmer and Demmel, 2000; Study 1; Study 2), with no recovery (Study 1; Study 2). Accordingly, decrement in the absence of recovery, and some effect of Significance, were predicted. Our PCA-derived SW demonstrated the decrement over trials as expected and no recovery. However, comparison of the SCR and SW indicated no difference for recovery or dishabituation, so neither recovery nor dishabituation can be definitively ruled out. The classic SW showed a main effect for Significance and also enhancement in the right hemisphere as predicted. Right hemisphere dominance has been linked to the orienting response (Maltzman, 1979; Rohrbaugh et al., 1984; Zimmer and Demmel, 2000). The
classic SW has been included in Pattern 4 based on decrement and Significance effects.

Both SCL and HRL were examined to address the possibility that Significance effects were produced by state changes. No significant state differences between conditions were found across trials 1–12. Therefore, non-specific arousal, as indexed by SCL, did not produce the Significance effects in this study, nor was there any evidence of prestimulus vigilance differences suggested by HRL. Hence the enhanced responses associated with counting can be solely and directly attributed to Significance.

4.5.3 ERP component patterns

The ERP components/subcomponents and autonomic measures have been grouped provisionally into four patterns. Stimulus-response patterns were used to decide on each measure’s inclusion in a pattern. Pattern 1 is characterised by insensitivity to both novelty and Significance: ECR1, P1, N1-3, P3a, and FSW. The independence from novelty has grouped ECR1, P1, and N1-3 together across three studies (here and Study 1; Study 2); but note N1-3 was not found in Study 2. The clustering of these measures in Pattern 1 supports the notion of an early stage of stimulus processing in the OR context, based on the variation of physical parameters, akin to transient detection.

Pattern 2 measures show no decrement over trials, but sensitivity to stimulus Significance: ECR2, N1-1, and P2. ECR2 has been linked previously to cognitive load (Lawrence and Barry, 2009, 2010) and the P3a has shown some sensitivity to executive processing (Rushby and Barry, 2009; Steiner et al., 2014). The Significance effect found for P2 was not anticipated, therefore inclusion of P2 in this pattern is tentative.

Pattern 3 measures demonstrate novelty but not Significance sensitivity: RP, PN, P3b, HabP3. These four measures have generally been reported as decrementing in OR-type paradigms where the same simple auditory stimulus was re-presented. In regard to
the PN, although decrement has been observed, Study 2 described a non-significant increase. Further comparable OR research using SCR as the OR benchmark is needed to confirm this pivotal aspect of habituation for the PN and address the paucity of these studies in the literature.

Pattern 4 is defined by the OR determinants of novelty and Significance, and inclusions in this pattern are: SCR and classic SW. Both the SCR and classic SW have close relationships to the phasic OR in previous literature.

The number of patterns equalled the combinations of novelty and Significance, and a separate autonomic measure was found to group into each of the four patterns. This configuration is consistent with the modules of processing proposed in PPT and the notion that autonomic measures serving as their indices have matching ERP counterparts.

Congruence coefficients confirmed the between-studies similarity and stability of the matched PCA-derived ERP components. Inspection of the topographies in Figure 4.9 also reveals close matches of the component pairs, and the latency order of the LPC subcomponents has been preserved: P3a, P3b, HabP3, and classic SW. Together these indicate stable ERP components in the OR in these two dishabituation studies.

4.6 Conclusion

We have extended the examination of the OR determinants investigated in Study 1 and Study 2 by varying novelty and Significance jointly. SCR showed all the defining aspects of the phasic OR examined here and was used as the OR ‘yardstick’ to assess each measure in relation to the OR. Tonic SCL and HRL data addressed the possible effects of arousal and vigilance. No differences in these tonic measures over conditions were found, demonstrating that the Significance effects found here can be attributed
directly to the Significance manipulation alone. The emergent four stimulus-response patterns were based on the defining aspects of OR habituation and Significance. Once again ECR1 has shown the critical aspects of early stimulus processing and four ERPs emerge as possible central matches, thus furthering the development of PPT. Novelty sensitivity was demonstrated by RP, PN, P3b, HabP3, and the classic SW. Significance effects were found in ECR2, P2, and classic SW. The lack of a Significance effect in the P3b and the possibility of that effect in N1-1 were both surprising results that require confirmation by replication, along with the anomalous Significance effect for P2.

The response pattern of the classic SW in this study and in Study 1 and Study 2 presents this LPC subcomponent as the most likely candidate for the central index of the phasic OR, consistent with early SW research. Importantly, the latency order of the LPC subcomponents has again been confirmed in the OR context, affirming the often disputed differentiation of the P3a and HabP3. These derived stimulus-response patterns contribute significantly to the ongoing development of PPT by introducing possible central matches for the autonomic measures, pending confirmatory findings. The present study has again found clear evidence of phasic measure fractionation, contrary to the unitary OR concept. The stimulus-response patterns identified confirm the patterns found in previous autonomic studies and strengthens the sequential and parallel processing model of the OR proposed in PPT. Inclusion of blood pressure as another cardiac measure in future OR investigations would further consolidate the understanding of PPT.
4.7 References

https://doi.org/10.1111/psyp.12814.


Barry, R.J., 1984c. The evoked cardiac response under processing load. Physiol. Psychol. 12, 35–40.

Barry, R.J., 1984d. Trial effects in the evoked cardiac response under processing load. Physiol. Psychol. 12, 315–318.

Barry, R.J., 1986. Heart rate deceleration to innocuous stimuli: an index of the orienting


De Bruin, L., Michael, J., 2017. Prediction error minimization: Implications for


Chapter 5. Study 4: Integration of three investigations of Novelty, Intensity, and Significance in dishabituation paradigms: A study of the phasic Orienting Reflex

Submitted as:

5.1 Abstract

Data from the three thesis studies examining autonomic and ERP measures in variants of a dishabituation paradigm were re-analysed to clarify ambiguous novelty results. The three studies manipulated 1. novelty, 2. novelty and intensity, and 3. novelty and Significance, in auditory dishabituation paradigms at very long interstimulus intervals (ISIs). The question of whether any single ERP matches SCR as the benchmark for the phasic Orienting Reflex (OR) was also addressed. Finally, the re-analysed measures of this study and measures from the previous studies were aligned with processes of Preliminary Process Theory (PPT). The SCR demonstrated all the aspects of habituation. The P3b and Classic SW showed decrement but no recovery, while Respiratory pause (RP) and Novelty P3 demonstrated novelty (decrement and recovery at the change trial), but dishabituation remained ambiguous. No decrement for the Processing Negativity (PN) was confirmed. Five autonomic and ERP groupings emerged and aligned with modules of processing in PPT: ECR1 (cardiac deceleration), P1, N1-3 and PN – Stimulus registration; RP and Novelty P3 – Novelty registration; Peripheral Vasoconstriction (PVC) and P3b – Intensity registration; and ECR2 (cardiac acceleration) and Classic SW – Response system. The SCR was confirmed to index the phasic OR. The pattern of results for the Late Positive Complex (LPC) components (P3a, P3b, Novelty P3 and Classic SW) suggests each is differentially sensitivity to selective determinants of the phasic OR, and consequently the summary LPC is presented as the most appropriate central index of the phasic OR.
5.2 Introduction

5.2.1 Background

The preceding Chapters have examined novelty, intensity, and Significance variation. The ensuing stimulus-response patterns were defined in accordance with what determinant was manipulated. Some clear linkages of measures’ stimulus-response patterns and stages of processing in PPT have emerged.

Study 1 (MacDonald and Barry, 2014) found 3 stimulus-response patterns when novelty was varied. The first pattern was characterised by no decrement: HR deceleration, P1, N1-3, N1-1, PN, P2, and P3a; the second pattern, represented by the P3b and parietal SW, showed decrement but not recovery, so the measures in this pattern were not related to novelty per se; the third pattern demonstrated all the aspects of ‘genuine’ habituation: SCR and HabP3. Study 2 (MacDonald et al., 2015) extended the examination of novelty to novelty and intensity. The temporal PCA found two ERP components not found in Study 1. The Na failed to decrement but there was not a significant difference between it and SCR in regard to intensity. The IntP3 had a peak latency between the HabP3 and Parietal SW, and this component showed both decrement and intensity effects. The N1-3 present in Study 1 was not identified. Three patterns emerged, Pattern 1 (no decrement or intensity effect): HR deceleration (ECR1), P1, PN, and P2 – these measures again clustered together, but Na, plus N1-1 and P3a from Pattern 1 in Study 1 were grouped in Pattern 2, along with PVC, all showing no decrement but intensity sensitivity. Pattern 3 showed all the habituation characteristics and an intensity effect: SCR, RP, P3b, HabP3, IntP3, and SW. These studies demonstrate that autonomic fractionation found in early studies is evident also in ERP measures in comparable paradigms.
Study 3 (MacDonald and Barry, 2017) manipulated novelty and Significance within subjects (subjects were instructed to count consecutive stimuli silently) in a dishabituation paradigm identical to that used in Study 1. Consequently, the study was a replication in regard to novelty, and data were collected from 32 rather than 16 participants. Since the focus was on Significance variation, the later cardiac acceleratory component of the HR response (ECR2) replaced PVC from Study 2. Cardiac acceleration has been shown to associate with Significance (Barry, 1984a; Lawrence and Barry, 2009, 2010); the other autonomic measures were the same as employed in the previous studies. Temporal PCA yielded 10 ERP components: P1, N1-3, N1-1, PN, P2, P3a, P3b, HabP3, Frontal SW (FSW), and the Classic SW. Four distinct patterns emerged: Pattern 1 (no trial or Significance effects): ECR1, P1, N1-3, P3a, and FSW; Pattern 2 (no trial effects but a Significance effect): ECR2, N1-1, and P2; Pattern 3 (trial but no Significance effects): RP, PN, P3b, and HabP3; Pattern 4 (trial and Significance effects): SCR and Classic SW. The manipulation of Significance has generated some re-organising of the 3-fold patterns in Study 1; however, some measures have shown consistent stimulus-response patterns over studies e.g., ECR1 and the P1. However, other measures were found to have ambiguous results. Specifically, measures such as N1-1 were found not to differ from SCR for intensity variation; the effects of these measures require resolution, optimally by employing greater N. Over the three studies, P3a, P3b, and HabP3 have been derived consistently, prior to the Classic SW. Barry et al. (2016) explored the response to novelty in habituation and oddball paradigms, and advocated the use of the term Novelty P3 (abbreviated as nP3) to replace HabP3; we adopt that nomenclature here.

5.2.2. LPC as an OR index

Many ERP components have been aligned with a limited set of OR criteria to
date. The N1 has been associated with the initial OR (Kenemans et al., 1989; Näätänen and Picton, 1987), the Mismatch Negativity (MMN/N2a) with change detection (Näätänen et al., 1978; Näätänen and Michie, 1979; Näätänen and Picton, 1987; Sokolov, 1990), N2b with the voluntary OR (Näätänen and Gaillard, 1983), along with the Processing Negativity (PN) (Barry et al., 2011; Sokolov, 1990), P2 (Rust, 1977; Walpurger et al., 2004), the P3a (Escera et al., 1998; Ford et al., 1976; Knight, 1996; Marinkovic et al., 2001; Squires et al., 1975), P3 (Ritter et al., 1968), the Novelty P3 (Courchesne et al., 1975), the O-wave (Slow Wave [SW] composite) (Loveless and Sandford, 1974; Rohrbaugh et al., 1984; Zimmer, 2002, 2006; Zimmer and Demmel, 2000;), and the Late Positive Complex (LPC) (Donchin et al., 1984; Rushby and Barry, 2007; Rushby et al., 2005). In these investigations only some of the requirements of the phasic OR are met, or met with limitations. The LPC appears the most likely candidate as a central index for the OR. The LPC subsumes the O-wave, which in turn encapsulates other distinct SWs. If an ERP composite can be shown to meet all the requirements of the OR and match SCR then it would be unlikely that any of its components would also meet all the OR criteria. Contrasting with SCR, no single ERP component has unequivocally fulfilled all the OR requirements to date. A series of experiments based on novelty manipulation in a dishabituation protocol and stepwise variation of the other key determinants appears optimal in examining the possible central analogues to the OR.

5.2.3. The present study

Our focus here was on sensitivity to novelty, particularly where the previous studies found no statistical difference between the novelty response of a measure and SCR – i.e., where the Interaction Test left an inconclusive result in regard to novelty sensitivity. The present study aimed to increase the power of such tests by combining
data from the previous three studies. Processing Negativity has also been examined due to some inconsistent decrement findings over the three studies. The resultant stimulus-response patterns based on novelty are then supplemented by, and integrated with, the intensity and Significance results reported previously, in order to align the measures to preliminary processing and the OR in PPT. In addition, the long standing question of whether the LPC is the most appropriate central measure to index the OR is addressed.

5.3 Methods

5.3.1 Participants

Sixty-four university students volunteered and participated in experimental sessions (ages 18 – 60, mean 22.4 years; 51 female; 51 right-handed).

5.3.2 Procedure

The procedures are the same as employed in previous studies of this thesis.

5.3.3 Physiological recording

The details of the physiological recording for the measures’ responses examined here are identical to those in section 2.3.3 of Chapter 2.

5.3.4 Data extraction

The criteria for data extraction for SCR, HR deceleration, HR acceleration, Respiratory pause, Peripheral vasoconstriction, and ERPs are presented in sections 2.3.4, 3.3.4, and 4.3.4.

5.3.5 Statistical analysis

Measures that previously failed to show significant differences between that
measure and SCR for decrement, recovery, or dishabituation, and PN (which showed inconsistent novelty effects) were analysed using the combined data from the three studies (see Table 5.1). Other results are taken directly from the relevant study (see Table 5.2).

The maximum response of each measure at each trial was used for analysis. For the present purposes Study 1 provided dishabituation profiles from 16 subjects’ data over the 12 trials with a frequency change at stimulus 11. The averaged repeated trials were counterbalanced across frequencies. Study 2 provided data from 16 subjects with a within subject change in intensity at stimulus 11. In this study the averaged repeated trials were counterbalanced across intensities. The within subjects data in Study 3 were averaged trials of 32 subjects and counterbalanced across frequency and Significance; as with Study 1 the “change” was one of frequency. Each of these studies thus provided data in response to 10 repeated stimuli, a change stimulus, and a re-presented standard stimulus; these novelty variations are examined here. To obviate between-study differences, the raw data were converted to within subject z-scores.

Repeated measures multivariate analysis of variance (MANOVA) was used to examine the autonomic and ERP measures with the factor Trials. Within Trials (for trials 1 to 10), decrement was examined by a linear trend. Responses for the change trial (recovery: trial 11 vs. 10) were investigated in separate MANOVAs if significant decrement over trials was found. Likewise, responses for the re-presentation trial (dishabituation: trial 12 vs. 10) were examined only if recovery on the change trial proved significant.

Differences between SCR and RP, PN, P3b, nP3, and Classic SW were examined with separate repeated measures MANOVAs. If a difference was found
between the measure and SCR in that pattern aspect for its initial analyses (e.g., no recovery effect for P3b and recovery effect for SCR), the difference in that pattern aspect was tested with a repeated-measure MANOVA for that measure and SCR, over the relevant trials. When a measure comparison to SCR involved decrement, the first 5 trials were used since the loss of novelty is most substantial over the initial trials (Barry, 2004; Barry and Rushby, 2006). Observed differences between the measure and SCR were tested by measure × decrement/recovery/dishabituation interactions; a difference was confirmed by a significant interaction, and consequently subsequent pattern testing was deemed unnecessary. An aspect of the pattern that failed to differ significantly from SCR was indicated by a non-significant interaction, and consequently the next aspect of the stimulus-response pattern was tested. Polarity was reversed for negative ERP components to ensure decrement, recovery, and dishabituation, were tested appropriately.

5.4 Results

No major artifacts in the autonomic or EEG data rendered specific data unusable; consequently, full data from all subjects could be analysed.

5.4.1 SCR

SCR showed substantial trial decrement apparent in a linear trend \( F = 319.66, p < .001, \eta_p^2 = .835 \), evident in Figure 5.1. The SCR recovered to the change stimulus (11 > 10: \( F = 36.76, p < .001, \eta_p^2 = .368 \)) and dishabituated to the re-presented original stimulus (12 > 10: \( F = 26.12, p < .001, \eta_p^2 = .293 \)).

5.4.2 Respiratory pause

Respiratory pause diminished linearly over trials \( F = 29.40, p < .001, \eta_p^2 = \)
Figure 5.1. The Z-scores for autonomic and ERP components over 12 trials. SE bars indicate the trial to trial variation, and the linear regression line and Coefficient of Determination for the first 10 trials are included.

.318), and showed recovery ($F = 16.23, p < .001, \eta_p^2 = .205$), but no significant dishabituation was observed. The results are shown in Figure 5.1. The absence of dishabituation in RP was tested against SCR for trials 10 and 12; the Measure (RP vs. SCR) × Trial (12 vs. 10) interaction resulted in a non-significant difference, indicating that RP and SCR did not differ on dishabituation. Table 5.1 shows these results.
5.4.3 ERP components

5.4.3.1 PN

The maximum amplitude reduced somewhat over trials ($F = 3.44, p = .068, \eta^2_p = .052$). Comparison of PN and SCR for decrement found a significant interaction: Measure (PN vs. SCR) × Trial (linear trend over 1-5): $F = 150.82, p < .001, \eta^2_p = .705$.

5.4.3.2 P3b

Responses diminished over trials ($F = 24.14, p < .001, \eta^2_p = .277$). No significant difference was observed between trials 10 and 11, see Figure 5.1. Comparison of P3b and SCR for recovery yielded a significant Measure (P3b vs. SCR) × Trial (11 vs. 10) interaction: $F = 19.59, p < .001, \eta^2_p = .237$.

5.4.3.3 Novelty P3

Novelty P3 reduced over trials ($F = 27.08, p < .001, \eta^2_p = .301$), and demonstrated recovery ($F = 6.48, p = .042, \eta^2_p = .093$). No dishabituation was evident. Novelty P3 (nP3) and SCR were compared for dishabituation, but no difference was found: the measure (Novelty P3 vs. SCR) × Trial (12 vs. 10) interaction was non-significant.

Table 5.1 Decrement, Recovery, and Dishabituation for this study, along with Intensity and Significance from previous studies for selected ANS and CNS (ERP) measures.

<table>
<thead>
<tr>
<th>Stimulus-response pattern for the current study</th>
<th>Stimulus-response pattern from previous studies</th>
</tr>
</thead>
<tbody>
<tr>
<td>Decrement</td>
<td>Measure vs. SCR (Decrement)</td>
</tr>
<tr>
<td>SCRs</td>
<td>****</td>
</tr>
<tr>
<td>PNs</td>
<td>****</td>
</tr>
<tr>
<td>ERP</td>
<td>nP3</td>
</tr>
<tr>
<td>Novelty P3</td>
<td>****</td>
</tr>
</tbody>
</table>

* $p < .1$, ** $p < .05$, *** $p < .01$, **** $p < .001$; — non-significant result; grey shading represents measure-SCR interaction not tested, double bordered cells represent ambiguous results.
5.4.3.4 Classic SW

Over trials 1–10, positive activity decremented \( F = 46.95, p < .001, \eta^2_p = .427 \), but recovery was not significant. Further, when the Classic SW and SCR were compared for recovery, a significant difference emerged: Measure (Classic SW vs. SCR) × Trial (11 vs. 10): \( F = 7.43, p = .008, \eta^2_p = .106 \).

5.4.4 Novelty patterns for the autonomic and ERP measures examined here

This study confirmed three novelty-based stimulus-response patterns over the autonomic and ERP measures. Pattern 1: no decrement was represented by PN. Pattern 2: decrement but no recovery was represented by P3b and Classic SW. Pattern 3: decrement and recovery; this clustered the measures SCR, RP and Novelty P3.

5.4.5 Other results

Table 5.2 includes all the remaining results from the three previous studies in regard to novelty, intensity, and Significance effects. For example, Table 5.2 indicates that ECR1 showed no decrement, intensity, or Significance effects, and differed significantly from SCR for each dependent variable in all three of the previous studies.

Table 5.2 Decrement, Recovery, and Dishabituation results from previous studies for measures not examined in this study; along with Intensity and Significance effects.

<table>
<thead>
<tr>
<th>Autonomics</th>
<th>Decrement (1, 2, 3)</th>
<th>Measure vs. SCR (Decrement)</th>
<th>Recovery</th>
<th>Measure vs. SCR (Recovery)</th>
<th>Dishabituation</th>
<th>Measure vs. SCR (Dishabituation)</th>
<th>Intensity (2)</th>
<th>Measure vs. SCR (Intensity)</th>
<th>Significance (3)</th>
<th>Measure vs. SCR (Significance)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PN</td>
<td>–2</td>
<td>***</td>
<td>–2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ECR</td>
<td>–(1, 2, 3)</td>
<td>**(1, 2, 3)</td>
<td>**2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ECR</td>
<td>–(3)</td>
<td></td>
<td>**2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ERPs</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ns</td>
<td>–2</td>
<td>***</td>
<td>–2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>P1</td>
<td>–(1, 2, 3)</td>
<td></td>
<td>**2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ns</td>
<td>–(3)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>P1–1</td>
<td>–(1, 2, 3)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>P2</td>
<td>–(1, 2, 3)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>P2a</td>
<td>–(1, 2, 3)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>*p &lt; .05, **p &lt; .01, ***p &lt; .001. — non-significant result; grey shading represents measure-SCR interaction not tested. The numbers 1, 2, and 3 correspond to MacDonald and Barry (2014) – novelty, MacDonald et al. (2015) – novelty and intensity, and MacDonald and Barry (2017) – novelty and Significance, respectively. * recorded in MacDonald and Barry (2015) only; **recorded in MacDonald et al. (2017) only; ***not recorded in MacDonald et al. (2015).</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
### 5.5 Discussion

This study consolidates the findings from Study 1, 2, and 3. Those investigations methodically varied the major determinants of the phasic OR: novelty, intensity, and significance; novelty was manipulated similarly in all the investigations. Since novelty has been shown to be the primary eliciting factor of the OR, our focus has been on novelty variation in dishabituation paradigms. Single-trial data captures the effects of rapidly diminishing novelty and concurrent measurement of autonomic and ERP measures permits matching for the relevant types of comparable stimulus processing based on the stimulus-response patterns – necessary for PPT development. In the previous and the current studies, the Interaction Test has provided a quantitative method of statistically exploring the difference between a measure and SCR when a stimulus-response pattern aspect differed in significance – i.e., it allowed resolution of whether a difference in significance level between effects in two measures indicates a significant difference. The combined larger pool of subjects has been used to help clarify ambiguous results found previously in the different studies. We have reported instances in these three studies where a measure has not differed from SCR, leaving open the possibility that although that aspect may not initially have reached significance, with greater power, significance may have resulted (e.g., RP failed to show dishabituation, while SCR exhibited clear dishabituation; however, no significant difference was found between RP and SCR for dishabituation). Consequently, we examined these novelty-based instances of ‘no differences’ with a greater number of subjects in an attempt to resolve the inconclusive findings. The Processing Negativity was included in this investigation due to differing novelty effects: no decrement (Study 1).
5.5.1 Autonomic findings

SCR demonstrated clear evidence of habituation (decrement, recovery, and dishabituation). Study 2 and Study 3 have reported SCR sensitivity to both intensity and Significance, respectively. Consequently, SCR fulfils all the requirements of the phasic OR, demonstrating sensitivity to novelty, intensity, and Significance.

Respiratory pause has previously been associated with stimulus novelty and the specific nature of the OR (recovery). In contrast to SCR, while RP demonstrated both decrement and recovery, dishabituation has not been significant in the previous three studies, nor here. Despite the increased power, no difference emerged here between RP and SCR for dishabituation, consistent with the three previous investigations, so dishabituation cannot be ruled out. However, Barry and James (1981a) also reported the lack of dishabituation in a rare, early visual dishabituation study, consequently it is unlikely that clear dishabituation is a typical aspect of the stimulus-response pattern for RP.

5.5.2 ERP findings

Processing negativity displayed non-significant decrement in Study 1, a non-significant increase in Study 2 and significant decrement but no recovery (that differed significantly from SCR) in Study 3. In the present study PN exhibited a marginal decrement that differed significantly from SCR. Barry et al. (2011) reported topographic reductions in PN but their habituation paradigm did not incorporate stimulus change. The presence of topographic reductions indicates some elements of the PN diminished while other elements were relatively active over trials. Barry et al. (2011) proposed that the PN in their study represented reflexive attentional switching, based on the notion of automatically focusing on attended stimuli. In this study, the slight reduction in frontal
activity was not inconsistent with the PN reduction of Barry et al. (2011), however, the present study indicates a relatively undiminished response over stimulus presentations. This finding may be parsimoniously explained by an initial alerting capacity associated with reflexive attentional switching that is partially present for subsequent presentations of stimuli. Accordingly, the PN appears dissociated from novelty per se. The PN has also shown no association with intensity (Study 2) or Significance (Study 3).

The P3b has previously shown decrement but no recovery (Studies 1 and 3). Steiner et al. (2014) and Study 2 found decrement but no recovery, but the increased response on trial 11 failed to differ from the comparable increase for SCR in Study 2. This study confirmed these previous reports of P3b decrement but no recovery (Studies 1 and 3) and clarified the findings of Study 2. The P3b has previously shown sensitivity to intensity (Study 2), but not Significance (Study 3).

The Novelty P3 has consistently demonstrated decrement (Studies 1, 2, and 3), however, recovery was reported only in Study 1. In Study 2 there was no statistical difference for recovery between nP3 and SCR, leaving open the possibility of recovery between nP3 and SCR, leaving open the possibility of recovery. In this summary study a robust decrement and substantial recovery were evident, presumably due to greater power. Dishabituation did not eventuate, however no difference was found between this measure and SCR, so dishabituation remains a possibility yet to be resolved. This outcome mirrors Study 1 and is consistent with Study 2 and Rushby et al. (2005), but inconsistent with Study 3 and Steiner et al. (2014). However, decrement and recovery demonstrates that the Novelty P3 reflects novelty per se. Novelty P3 has displayed no clear relationship with intensity or Significance.

The classic SW has previously demonstrated decrement (Studies 1, 2, and 3), but
recovery has not been a prominent feature (Studies 1, 2, and 3), while dishabituation has been less clear (Study 2). However, Rushby et al. (2005) reported the Classic SW demonstrating decrement, recovery, and topographic increase for dishabituation when novelty, intensity, and Significance were varied. The Classic SW examined here showed a substantial decrement but no recovery. An inconclusive intensity effect has been reported in Study 2 but a Significance effect has been reported in Study 3.

A greater sample size has clarified the lack of decrement for PN and endorsed the occurrence of recovery for RP and Novelty P3, and also the lack of recovery for P3b and Classic SW. However, dishabituation has not been resolved for RP or Novelty P3.

In terms of novelty, from the OR perspective, dishabituation appears to play a less important role than recovery. The possibility remains open that dishabituation is not solely a function of novelty, and that other processes are active, such as the arousal state of the subject (Steiner and Barry, 2014). Consequently, decrement and increased responding to the change stimulus jointly may be considered as clear manifestations of novelty per se, while decrement in the absence of recovery is associated with processes other than novelty.

5.5.3 Stimulus-response patterns for all the measures

Three stimulus-response patterns have emerged based on novelty. 1 (no decrement): PVC, ECR1, ECR2, Na, P1, N1-3, N1-1, PN, P2; 2 (decrement but no recovery): P3b and Classic SW; 3 (novelty [decrement and recovery]): SCR, RP and Novelty P3. These stimulus-response patterns found for the measures based on novelty may be expanded to encompass the findings from intensity and Significance variation. Within the data, we can recognise five patterns: Pattern 1 (no novelty, intensity or Significance effects): ECR1, P1, N1-3, and PN; Pattern 2 (novelty, but no intensity or
Significance effects): RP and Novelty P3; Pattern 3 (no novelty, intensity, but no
Significance effect): PVC and P3b [decrement but no recovery]; Pattern 4 (no novelty,
Significance but no intensity effect): ECR2 and Classic SW [decrement but no
recovery]; and Pattern 5 (novelty, intensity, and Significance): SCR.

The stimulus-response patterns of some measures remain unclear. Greater N
would clarify the following measures in regard to intensity effects: Na, N1-1 (also
Significance effects), P2, and P3a. In addition, recovery for IntP3 is inconclusive, and
Significance has not been examined for this measure or for Na.

5.5.4 LPC and the OR

While no single component of the LPC matches the SCR as a central measure of
the phasic OR, the subcomponents collectively appear to represent the OR, with each
assuming a different functional role. They can be linked to novelty (Novelty P3),
intensity (P3b and possibly the P3a), and Significance (Classic SW). Consequently, in
OR structured paradigms, consistent with Putman and Roth (1990), at least one LPC
component will be elicited by novelty variation, while the LPC should also be sensitive
to intensity and Significance manipulation. Rushby et al. (2005), when peak-picked data
and temporal PCA-derived components were both analysed, concluded that the
stimulus-response patterns of the LPC and SCR were identical. Rushby and Barry
(2007) reached a similar conclusion when they varied intensity in an auditory
dishabituation paradigm at long ISIs.

5.5.5 Measure placement in PPT

The various measures analysed in the previous and present studies can be placed
in an expanded version of the PPT schematic (Figure 5.2). Those measures with clear
stimulus-response patterns are in bold script, while other measures with ambiguous
sensitivity to a variable are presented in smaller, non-bold script with a question mark attached.

The transient ECR1, P1, and N1-3, have shown no tendency to decrement in any of the studies (see Table 5.2), and proved insensitive to both intensity and Significance, so these measures mark only the occurrence of a stimulus (stimulus registration); PN was added from this study. Respiratory pause and nP3 demonstrate strong decrement and recovery with no sensitivity to intensity or Significance, placing them as indices of the novelty register. The magnitude register is represented by both peripheral vasoconstriction (PVC) and P3b; both measures have shown sensitivity to intensity changes without novelty sensitivity. The later cardiac acceleration component (ECR2), Classic SW, and P2 have demonstrated no decrement but Significance sensitivity, and this suggests these measures as indices for the response system. Although a Significance main effect was noted in Study 2 for P2, the result was unexpected and lacked theoretical and prior experimental support. Further investigation using the same protocol and conditions may clarify this effect. The LPC is placed as the sole ERP counterpart to SCR representing the phasic OR.

5.6 Conclusion

We re-examined three auditory dishabituation investigations where novelty, intensity, and Significance were systematically varied (Studies 1, 2, and 3). In this study the autonomic and ERP measures that previously presented ambiguous results with respect to novelty were re-analysed with a greater N from the pooled data, using within subject z-scores. The SCR demonstrated decrement, recovery, and dishabituation. The data supported findings of no decrement for PN and recovery for RP and Novelty P3, and no recovery for P3b and Classic SW. Dishabituation for both the RP and Novelty
P3 remains unclear and requires resolution. These patterns over trials of the re-analysed

Figure 5.2. The restructured version of PPT including ERP components (in red). Measures displaying clear stimulus-response patterns are in bold script. Measures with ambiguous stimulus-response patterns are in smaller, non-bold script with a question mark attached.

measures were supplemented with the previous results for intensity and Significance, allowing an update of PPT. The re-analysed measures and previous findings were integrated into the schematic of Preliminary Process Theory primarily on the basis of novelty, but with intensity and Significance sensitivity added. Stimulus registration is indexed by ECR1, P1, N1-3, and PN; while novelty is represented by RP and nP3, and intensity by PVC and P3b. The ECR2 and Classic SW are indices of the Response system, and SCR and LPC represent the OR. The matched autonomic and ERP measures relate to 5 stages of processing: three for pre-OR, the OR itself, and post-OR.

No single PCA-derived ERP component matched the stimulus-response pattern of SCR, but it was noted that the LPC was composed of ERP components that
collectively met the requirements of the phasic OR, and this is included in the revamped PPT model.

Future investigations that jointly manipulate novelty and Significance (counting or actively responding to stimuli), along with intensity, in an auditory habituation paradigm and using a large subject pool are recommended. This would logically extend and partially replicate Rushby et al. (2005) by incorporating very long ISIs and clarify some of the ambiguous results of the four thesis studies. Since the OR is found across different stimulus modalities, extension into visual and other modalities with similar paradigms would be advantageous to strengthen the OR database and clarify the explanatory power of PPT.
5.7 References


Chapter 6. General Discussion

The doctoral research programme presented here focused on the relationship between ERP components and the OR. This main theme was outlined in three broad aims:

1. Explore the stimulus-response patterns of the autonomic and central measures when novelty, intensity, and Significance are manipulated in auditory dishabituation paradigms at very long ISIs;

2. Match PCA-derived ERP components to existing autonomic measures and align the measure pairs to specific pre-OR, OR, or post-OR processing in PPT;

3. Address the question of whether the LPC or any single PCA-derived ERP component can represent the phasic OR.

Although PPT provides some theoretical and structural guidelines upon which to interpret results, the research described in these four studies was primarily ‘data driven’. Many of the measures employed in the four studies have not been examined adequately at very long ISIs in auditory dishabituation protocols (e.g., PVC, PCA-derived P1, P2, and SW). Innocuous auditory stimuli and moderate stimulus parameters were used in all the studies to ensure the optimal conditions for phasic OR generation.

The broad aims outlined were addressed on an ‘evolving’ basis over the four studies. A measure’s stimulus-response pattern may fluctuate with respect to the determinants manipulated over studies. However, some measures have exhibited stable stimulus-response patterns in regard to novelty e.g., ECR1 and P1. Confirmation, with an appropriate degree of certainty, of any measure’s stimulus-response pattern may only be assessed after the final effects of the manipulated determinants are known. The
question of whether the LPC or any single PCA-derived ERP component adequately represents the phasic OR was discussed in Study 4 after all the measures’ stimulus-response patterns had been appropriately examined.

The determinants of the OR were introduced into the experimental structures in a stepwise fashion; concisely: Study 1 varied novelty within subjects, Study 2 varied novelty within subjects and intensity between subjects, while Study 3 varied novelty and Significance within subjects. Consequently, Study 3 replicated Study 1 regarding novelty as well as stimuli parameters used. Summary Study 4 re-examined ambiguous novelty-based findings from the previous studies, consolidated the findings of all the measures, and matched and placed ERP and autonomic measures into the structure of PPT.

Study 1 provided the foundation thesis investigation into novelty. The viability of this study was supported by MacDonald et al. (2012). MacDonald et al. (2012) used single-trial data to examine the relationship between the autonomic (SCR, HR deceleration, and RP) measures and peak-picked ERPs (N1 and LPC) in the OR context. Participants received an alternating sequence of 60 and 80 dB innocuous tones at very long ISIs. The SCR showed both trials and intensity main effects, however, HR deceleration failed to demonstrate either of these effects. Respiratory pause decremented over trials but an intensity effect was absent. The N1 showed no main effect of trials but an overall intensity effect; however, midline reduction in the left hemisphere over trials was reported. This three-way interaction suggested differential activity of N1 subcomponents. The LPC showed no trials or intensity effect, therefore it was not deemed a central analogue for the OR. The scope of investigation in MacDonald et al. (2012) was broadened for this thesis by the inclusion of the specific dishabituation structure, temporal PCA, and the Interaction Test. Temporal PCA was
employed to decompose the single-trial EOG-corrected raw data into ERP components – a considerable advancement on the time consuming, subjective method of peak-picking. The Interaction Test permitted apparent differences in stimulus-response patterns between SCR and other autonomic and ERP measures to be assessed quantitatively. The SCR was adopted as the standard comparison measure for the Interaction Test, using it as the phasic OR benchmark. This was confirmed as an appropriate choice through the thesis because SCR demonstrated significant decrement, recovery, and dishabituation in Study 1, novelty and intensity effects in Study 2, novelty and Significance effects in Study 3, and novelty effects in Study 4.

The autonomic measures HR deceleration (Studies 1, 2, and 3), HR acceleration (Study 3), Respiratory pause (Studies 1, 2, and 3), and Peripheral vasoconstriction (Study 2) affirmed earlier reports regarding their stimulus-response patterns. The HR deceleration consistently failed to decrement in the first three studies, and demonstrated intensity and Significance independence. Respiratory pause demonstrated novelty sensitivity, but dishabituation was found to be inconclusive (Study 4), even after utilising a large combined pool of participants. The findings for HR acceleration (no decrement but Significance effects) and Peripheral vasoconstriction (no decrement but intensity effects) confirmed their earlier reports.

Study 1 focused on novelty solely – the prime eliciting factor of the OR. The manipulation of novelty yielded 3 stimulus-response patterns. The first pattern was defined by no decrement: HR deceleration, P1, N1-3, N1-1, PN, P2, and P3a; the second pattern grouped P3b and parietal SW, both measures showing decrement but no recovery. Therefore, P3b and parietal SW were related to decrementing processes other than novelty. The third pattern included SCR and HabP3, both exhibiting all aspects of ‘genuine’ habituation. However, recovery and dishabituation for RP, and dishabituation
for the HabP3, required further resolution.

Study 2 extended Study 1 by jointly manipulating novelty and intensity. Intensity sensitive PVC supplemented the autonomic measures used in Study 1. Three patterns emerged, Pattern 1 (no decrement or intensity effect): HR deceleration, P1, PN, and P2; Pattern 2 (no decrement but intensity effects): PVC, Na, N1-1, and P3a; Pattern 3 (novelty and intensity effects): SCR, RP, P3b, HabP3, IntP3, and SW. Both N1-1 and P3a were re-grouped together according to intensity sensitivity; while RP, P3b, HabP3, and SW were similarly re-grouped on the same basis. Interestingly, the P3b and SW remained paired. Inclusion of some of the measures into the stimulus-response patterns was based on null results in the Interaction Test. Generally, the effects for these measures were directionally consistent for a significant effect e.g., RP appeared to dishabituate but random error may have prevented the possible effect reaching significance. Similarly, although the intensity effect was non-significant for P3a, the observed 80 dB response was greater than to 60 dB on the change trial, and no difference between P3a and SCR was apparent. Such results require further resolution in future studies.

Study 3 manipulated novelty and Significance within subjects jointly in the same paradigm used in Study 1. Heart rate acceleration (ECR2) replaced PVC. Four patterns were identified in the data, Pattern 1 (no trial or Significance effects): ECR1, P1, N1-3, P3a, and FSW; Pattern 2 (no trial effects but a Significance effect): ECR2, N1-1, and P2; Pattern 3 (trial but no Significance effects): RP, PN, P3b, and HabP3; and Pattern 4 (trial and Significance effects): SCR and Classic SW. The 3-fold pattern of Study 1 was expanded to incorporate measures showing either a Significance or trial effect, but not both.
Study 4 used a combined larger pool of participants to examine novelty-based instances of ‘no difference’ when a measure failed to differ in a stimulus-response pattern aspect from SCR. The Processing Negativity was included in this investigation due to differing novelty effects over Studies 1, 2, and 3. The utilisation of greater N substantiated no decrement for PN, recovery for RP and Novelty P3, no recovery for P3b and Classic SW, but no resolution was found with respect to dishabituation for both RP and Novelty P3. When the re-analysed results of Study 4 were combined with the intensity and Significance findings from the previous studies, 5 patterns resulted:
Pattern 1 (no novelty, intensity or Significance effects): ECR1, P1, N1-3, and PN; Pattern 2 (novelty, but no intensity or Significance effects): RP and Novelty P3; Pattern 3 (intensity, but no novelty or Significance effects): PVC and P3b [decrement but no recovery]; Pattern 4 (Significance, but no novelty or intensity effects): ECR2 and Classic SW [decrement but no recovery]; and Pattern 5 (novelty, intensity, and Significance): SCR. These patterns supported the matching of autonomic measures and ERP component measures (except in Pattern 5), and the matched pairs were included into the updated PPT. As a group, ECR1, P1, N1-3, and PN indexed Stimulus registration; RP and nP3 indexed novelty; PVC and P3b indexed intensity; ECR2 and Classic SW represented the Response system, and SCR exemplified the OR.

In each of the studies various ERP components were grouped with SCR, however, across the studies no single component was grouped invariably with SCR. Therefore, no single PCA-derived ERP component matched the stimulus-response pattern of SCR under all manipulations, but the LPC, composed of stable ERP subcomponents, collectively met the requirements of the phasic OR, and so the LPC was included in the updated PPT model.

Within the LPC composite, the ascending latency order of the main
subcomponents was invariant over studies: P3a, P3b, Novelty P3, and the Classic SW. Notably, the P3a and Novelty P3 were consistently derived as separate LPC subcomponents with distinct stimulus-response patterns.

6.1 Conclusions

This series of studies has consolidated past findings for autonomic measures relevant to the OR, including those largely used by Sokolov. The fractionation of measures has been convincingly demonstrated within and between autonomic and ERP measures. The presentation of auditory stimuli in the dishabituation paradigm at very long ISIs has provided robust single-trial data, which proved invaluable in capturing the responses to novelty in autonomic and especially EEG data over the first few trials. The SCR showed all the aspects of habituation as well as intensity and Significance effects. These consistent and robust results confirmed SCR’s use as the standard measure of comparison in the Interaction Test – so the qualitative descriptions of early PPT research progressed here to quantitative findings.

Study 4 revealed important autonomic and ERP matches that have been integrated to update PPT: ECR1 and P1, N1-3, and PN align with stimulus registration; RP and Novelty P3 represent novelty processing; PVC and P3b represent intensity processing; ECR2 and SW are grouped in the Response system; while LPC matches SCR as the phasic OR index.

6.2 Further Research

The auditory stimuli delivered at very long ISIs in these dishabituation paradigms allowed simultaneous recording of autonomic and EEG data, permitting the matching of ERP components with the autonomic measures.
Although all the studies statistically test the autonomic and ERP effects in multiple comparisons, Bonferroni corrections were not undertaken. Since all the tests were genuine *a priori* tests, this option reduces the chance of false negatives. If, however, more variables were introduced into future studies, techniques such as the Benjamini-Hochberg procedure may need to be considered.

Throughout these studies, SCR has remained the epitome of the phasic OR, no other measure has consistently matched the stimulus-response pattern of SCR. The question arises: Is the concept of the OR, as indexed solely be SCR, useful? If the OR demonstrated a unitary nature, then data from the various measures would seem redundant with only a vague promise of new information emerging. Rather than treating the OR as a ‘blanket’ term, PPT has opened up an array of investigatory possibilities by proposing distinct preliminary processing prior to OR generation. Consequently, the advent of PPT has redefined the OR and restricted the meaning of the term OR. Teasing out the characteristics of the preliminary processing, indexed by specific measures, remains a future challenge, while previous work provides a solid foundation of PPT.

In regard to dishabituation, Chapter 5 included sixty-four subjects, yet no dishabituation effects were found for the Novelty P3 or RP. I am not aware of any published paper reporting dishabituation effects at very long ISIs for either RP or Novelty P3. In this thesis, differences between SCR and RP/Novelty P3 were inconclusive for dishabituation, however SCR reliably exhibited dishabituation inferring the suitability of the paradigm for examining dishabituation. This suggests that both RP and Novelty P3 are not characterised by dishabituation and so not associated with OR generation. Steiner and Barry (2014) found that dishabituation was dependent on the current arousal level, and represented an interruption of the habituation processing. In Chapter 4, overall differences in prestimulus SCL and HRL were tested, but differences
between trials 10 and 12 were not examined; this would be worthy of future investigation into the occurrence and importance of dishabituation in the OR context.

The stimulus-response patterns for RP and Novelty P3, based on novelty, intensity, and Significance, are similar but not identical. Although both show strong, consistent novelty effects without dishabituation, no Significance effects are apparent. Taken together, these findings support the view that both RP and Novelty P3 index novelty registration rather than OR generation.

However, the stimulus-response patterns of some measures remain unclear. Greater N may clarify dishabituation for RP and Novelty P3, but recovery for IntP3 was inconclusive as were intensity effects for Na, N1-1 (also Significance effects), PN, P2, P3a, and SW. Significance has not been examined for Na. Although P2 showed a Significance effect, this finding was unsupported in the literature. The replication of these effects in future studies would consolidate the placement of these measures in PPT. Inclusion of other measures such as blood pressure or electromyogram (EMG) is recommended to expand the scope of PPT.

6.2.1 The meeting of research areas and future directions

The various stages of PPT, particularly the executive (cognitive perceptual processing) still lack neural mechanisms to underpin the observed effects. Substantial inroads have been gained in identifying neural populations and brain structures responsible for P3 generation. Nieuwenhuis and Aston-Jones (2005) propose that motivationally significant stimuli can initiate phasic activity in the Locus Coeruleus (LC) that elicits a P3 via norepinephrine efferents (LC-P3 hypothesis). Nieuwenhuis et al. (2011) expanded this mechanism to include autonomic responses; the parallel activation of the LC and sympathetic nervous system (autonomic responses) through
mediation from the paragigantocellularis (PGi) of the rostral ventrolateral medulla was suggested as a mechanism for generating the P3 and SNS components of the OR. Interestingly, the PGi receives input from the prefrontal and insula cortex. Barry and Rushby (2006) found associations between these cortical regions and an exponential P3 decrement, suggestive of the Novelty P3. Further application of LORETA examining the neural substrates underlying specific stages of processing in PPT would greatly enhance our understanding of the processing chain involved in elicitation of the OR. Future investigations into visual and other modalities would contribute to the generalisation of the results found here and strengthen the explanatory power of PPT. Finally, structural equation modelling may be undertaken with a sufficiently large data set, to investigate the extent of autonomic and ERP matchings in relation to the structure of PPT.
6.3 References


Full References


Barry, R.J., 1977b. The effect of “significance” upon indices of Sokolov’s orienting response: a new conceptualisation to replace the OR. Physiol. Psychol. 5, 209–214


Barry, R.J., 1984c. The evoked cardiac response under processing load. Physiol. Psychol. 12, 35–40.

Barry, R.J., 1984d. Trial effects in the evoked cardiac response under processing load. Physiol. Psychol. 12, 315–318.


Donchin, E., Coles, M.G.H., 1988. Is the P300 component a manifestation of context
Donchin, E., Heffley, E., Hillyard, S.A., Loveless, N., Maltzman, I., Öhman, A.,

endogenous components of the ERP. In: Callaway, E., Tueting, P., Koslow, H.

Edwards, D.C., 1974. Stimulus intensity and recency contrasts and orienting response

Psychophysiology 12, 12–14.

Involuntary Attention to Acoustic Novelty and Change. J. Cogn. Neurosci. 10,
590–604.


brain potential (ERP) sign of the brain's evaluation of novelty. Neurosci.


that acutely enhancing serotonin with the selective serotonin reuptake inhibitor
citalopram modulates the loudness dependence of the auditory evoked potential
Exp. 21, 47–52.


latency response study of auditory evoked potentials amplitude and latencies
audiologically normal individuals. Braz. J. Otorhinolaryngol. 73, 69–74.

Nesselroade and Baltes, J.R., Baltes, P.B., 1970. On a Dilemma of Comparative Factor
Meas. 30, 935–948.

Nieuwenhuis, S., Aston-Jones, G., Cohen, J.D., 2005. Decision Making, the P3, and the

relationship between the P3 and the autonomic components of the orienting
response. Psychophysiology 48, 162–175.


16, 253–262.

skin conductance component of the orienting reaction to an auditory stimulus.


Rankin, C.N., Abrams, T., Barry, R.J., Bhatnagar, S., Clayton, D.F., Colombo, J.,


Roth, W.T., Blowers, G.H., Doyle, C.M., Kopell, B.S., 1982. Auditory stimulus intensity effects on components of the late positive complex.


Berlin, Germany.


Appendix A: Full List of Publications


Appendix B: Ethics Application Approval

RENEWAL APPROVED
In reply please quote: HE02/076
Further Enquiries Phone: 4221 4457

27 April 2009

Professor Robert Barry
Department of Psychology
University of Wollongong

Dear Professor Barry,

I am pleased to advise that renewal of the following Human Research Ethics application has been approved. This certificate relates to the research protocol submitted in your original application and all approved amendments to date.

Ethics Number: HE02/076

Project Title: Integration of peripheral autonomic nervous system (ANS) and central nervous system (CNS) measures of brain function in a sequential processing approach to the psychophysiology of cognition

Name of Researchers: Professor Robert Barry, Mr Brett MacDonald

Approval Date: 24 April 2009

Expiry Date: 23 April 2010

Please remember that in addition to completing an annual report the Human Research Ethics Committee requires that researchers immediately report:

* proposed changes to the protocol including changes to investigators involved
* serious or unexpected adverse effects on participants
* unforeseen events that might affect continued ethical acceptability of the project.

You are also required to complete a monitoring report at the end of your project. This report will be sent out approximately 6 weeks prior to the date your ethics approval expires. The report must be completed, signed by the appropriate Head of Unit, and returned to the Research Services Office.

Yours Sincerely,

/s/ Professor Arthur Jenkins
Chairperson
Human Research Ethics Committee

cc:
Appendix C: Demographic Questionnaire

Subject No: 

Age: ________  Height: ________  
Sex: ________  Weight: ________  

Handedness: Left/Right (Please circle one)

Please indicate whether you have used any of the following substances in the past 12 hours:

Caffeine (eg. tea/coffee): Yes/No  When: ________  Qty: ________
Alcohol: Yes/No  When: ________  Qty: ________
Tobacco: Yes/No  When: ________  Qty: ________

Are you receiving treatment for any medical condition? Yes/No
If yes, please specify: ____________________________________________

Are you taking any medications? Yes/No
If yes, please specify: ____________________________________________

Are you pregnant? Yes/No

Have you received treatment for any of the following:
Heart Problems: Yes/No  Details: ________________________________
Circulation Problems: Yes/No  Details: __________________________
Nerve or Sensory Problems: Yes/No  Details: ____________________
Pain (Acute or Chronic): Yes/No  Details: ________________________

Are you experiencing any of these problems now? ____________________________

Have you ever suffered epileptic seizures? Yes/No
Have you ever suffered any serious head injuries or periods of unconsciousness? Yes/No
Have you ever sought treatment for any psychiatric illness? Yes/No