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Algorithms and procedures to analyze physiological signals in psychophysiological research

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Algorithms and procedures to analyze physiological signals in psychophysiological research

by

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For the Major Program

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FREQUENTLY USED ABBREVIATIONS

ECR	Evoked cardiac response
EDA	Electrodermal activity
EKG	Electrocardiogram
HF	High frequency heart rate variability
HR	Heart rate
HRV	Heart rate variability
LF	Low frequency heart rate variability
M	Mean
mdn	Median
msec	Milliseconds
NS-SCR	Non-specific skin conductance response
rMSSD	Root-mean-square successive differences
OR	Orienting response
RSA	Respiratory sinus arrhythmia
SCL	Skin conductance level
SCR	Skin conductance response
SD, sd	Standard deviation
SDANN	Standard deviation of normal-normal intervals calculated over short periods
SDNN	Standard deviation of the normal-normal intervals
SDSD	Standard deviation of successive differences
ULF	Ultra low frequency heart rate variability
VLF	Very low frequency heart rate variability

ABSTRACT

This dissertation presents analytical techniques which allow more information to be derived from psychophysiological data than possible with traditional methods. The techniques include an implemented algorithm for chest strain-gauge respiration signal analysis and a permutation testing method for evaluating changes over time in physiological signals. These methods are applied to three data sets, each examining physiological correlates of emotional experience. In the first study physiological correlates of moods induced using music were identified, although respiration entrainment confounds the issue of whether mood or the music caused the observed patterns. The second study examined physiological responses while subjects watched an emotion-inducing movie under three emotion-regulation conditions; changes relating both to the movie scenes and condition were identified. Finally, the third study evaluated short term changes in heart rate while viewing words in terms of the type of word viewed and later word recall.

INTRODUCTION

Motivation

This dissertation was motivated by two factors: the acknowledgement that measuring the physiological concomitants of "emotion" requires measures typically multivariate, noisy, and exhibiting patterns of change over time; and the acknowledgement that current statistical analytical techniques in psychophysiological emotion research are generally inadequate to fully address these issues. Theoretical and empirical work on emotion has converged on the conception of "emotion" not as a state, but rather as a process extended in time, one which involves a large number of changes in multiple bodily parameters. Yet emotion research has historically focused on an extremely impoverished set of data; a narrow window into the many somatic state changes that in fact comprise emotional response.

Despite this theoretical framework considering emotion as a complex high-dimensional process, and the common practice of measuring multiple bodily parameters concurrently in research, typical statistical data analyses left much to be desired. A good part of this project was thus motivated by the desire to derive as much information as possible from psychophysiological data. Many existing analysis methods were ill-suited to detect effects that change over time, focusing instead on averaged responses which risk missing smaller or short-lived effects. It is now possible, due to increased computational power, to perform more complex, intensive, and multivariate analyses than were previously possible. These methods make it possible to derive more numerous and informative measures from raw physiological data, and to examine the data for interacting patterns of change. The methods described in this dissertation certainly do not exhaust the possibilities, but provide several more tools, which may lead to the development of even more robust and flexible techniques.

While this dissertation focuses on data analysis and statistical treatment, it is hoped that the issues addressed will in turn influence the ways in which data are initially collected. Psychophysiological parameters can now be recorded in parallel and with better fidelity than was previously possible. Good recordings of multiple systems at high sampling rates will provide the raw data necessary for ever more thorough treatment of emotion and its influence on cognition and behavior.

Organization of the Dissertation

This document consists of two broad sections tied together by new methods of analyzing physiological data, in the context of psychophysiological experiments aimed at understanding mood and emotion. The first section focuses on analytical methods while the second section discusses the application of these methods to three different psychophysiological data sets. Two analytical methods are presented: a respiration signal processing procedure and a method for evaluating the significance of curves describing changes over time in repeated measures data. The experiment data was analyzed using a variety of graphical and statistical techniques, notably permutation testing, allowing a more accurate and detailed interpretation than is possible with traditional parametric methods alone.

CHAPTER 1: BACKGROUND AND LITERATURE REVIEW

Introduction: Psychophysiology

The field of psychophysiology is concerned with the interaction of the mind, brain, and body. It intersects multiple disciplines, including neurology, neuroscience, physiology, and psychology. A concise definition of psychophysiology is provided by the Society for Psychophysiological Research's journal, *Psychophysiology*, which describes its focus as "covering research on the interrelationships between the physiological and psychological aspects of brain and behavior (Blackwell Publishing)." The most complete definition may be the one given in the *Handbook of Psychophysiology* (Cacioppo, Tassinary, & Berntson, 2000): "Thus, psychophysiology can be defined as the scientific study of social, psychological, and behavioral phenomena as related to and revealed through physiological principles and events in functional organisms."

Many physiological processes are measured in psychophysiological research, although only a tiny fraction of those monitored by the brain. Measures commonly used in psychological research include facial and peripheral muscle activity, heart rate, respiration rate, respiration volume, electrodermal activity, peripheral skin temperature, blood pressure, pupil diameter, eye movements, gastrointestinal activity, and hormone levels. Each measure provides insight into different cognitive and emotional, as well as physical and neurological, processes. This dissertation is primarily concerned with three systems: cardiovascular, respiratory, and electrodermal, which will now be discussed in detail.

Cardiovascular Psychophysiology

Monitoring heart activity has long been a standard component of psychophysiological experiments since it is easy to accurately and continuously determine the state of the heart by monitoring its electrical activity with an electrocardiogram (EKG). Examination of these recordings reveals that the normal heart rate is not completely regular, but rather varies at multiple frequencies. The study of the irregularities in the heart beat pattern, heart rate variability (HRV) analysis, revealed that physiological processes cause variations in the heart rate at specific frequencies, and analysis of these variations provides information about the underlying physiological process (reviewed in Berntson, et al., 1997; Brownley, Hurwitz, & Schneiderman, 2000; Porges & Byrne, 1992; Stein & Kleiger, 1999; Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996).

Biological Basis

The heart's sinoatrial node generates signals which trigger heart beats at a regular frequency even if the heart is isolated from external influences (Brownley, Hurwitz, & Schneiderman, 2000). The heart does not normally beat at this rate however, because it is influenced by autonomic nervous system activity, which causes constant fluctuation in the heart rate (Stein & Kleiger, 1999; Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996). In addition to direct neuronal control of the heart, the heart rate (HR) is influenced by substances circulating in the bloodstream, particularly epinephrine and norepinephrine released by the adrenals (Brownley, Hurwitz, & Schneiderman, 2000) and rennin-angiotensin system activity (Berntson, et al., 1997). At rest, parasympathetic nervous system activity has a stronger influence on the heart rate than sympathetic nervous system activity, but the rate of beating is the net result of the combination of neural and hormonal factors (Brownley, Hurwitz, & Schneiderman, 2000; Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996). HRV varies with age and gender; HRV generally decreases with increasing age and is lower in young females then males (Umetani, Singer, McCraty, & Atkinson, 1998).

HRV is a reflection of the tight neuronal control of the heart that is present in mammals. This central control, described in detail below, enables the HR to be rapidly increased or decreased in response to metabolic demands and changing conditions. Phylogenetically more primitive vertebrates, such as lizards and fish, lack this tight central control and must instead rely on the slower endocrine system to adjust HR (Porges, 2001; Porges, Riniolo, McBride, & Campbell, 2003). Hormonal changes by the endocrine system, also present in mammals, affect many organs throughout the body, a massive response not well suited to rapid or minor HR adjustments.

Central Control

The primary autonomic nervous system influence on the heart is that of the parasympathetic branch via the vagus nerve. The sympathetic nervous system also innervates the heart, however, through fibers arising from the spinal cord. These sympathetic fibers increase the heart's activity by increasing the sinoatrial node's rate of firing and the excitability of the atrioventricular node and ventricles (Aminoff & Daroff, 2003). The vagus (tenth cranial) nerve is the primary parasympathetic nerve, innervating many organs, including the heart, striated facial muscles, and viscera (Greenstein & Greenstein, 2000; Porges, 2003). About 75% of parasympathetic innervation of the viscera is done by

the vagus (Aminoff & Daroff, 2003). The vagus nerve is primarily responsible for slowing the resting heart rate; HR can be increased by either increasing sympathetic activity or reducing vagus activity (Brownley, Hurwitz, & Schneiderman, 2000). The vagus slows the HR primarily by sinoatrial node inhibition, but it also reduces atrioventricular contraction and increases the ventricular refractory period (Aminoff & Daroff, 2003).

The vagus is composed of two main branches, both of which project from the medulla: the dorsal (also called the motor efferents) which projects from the dorsal motor nucleus of the vagus, and the ventral (also called the parasympathetic efferents), which projects from the nucleus ambiguus (Greenstein & Greenstein, 2000; Porges, 2001). The ventral branch has myelinated axons and is the main inhibitory influence on the sinoatrial node. The unmyelinated dorsal branch primarily regulates organs below the diaphragm. Higher cortical areas, including the limbic system, can regulate heart and visceral activity through connections to the origins of the vagus in the medulla (Porges & Byrne, 1992). The central nucleus of the amygdala, for example, has direct inhibitory connections to the nucleus ambiguus, as do the frontal cortex corticobulbar pathways, which regulate several medullary nuclei, including the nucleus ambiguus (Porges, 1995; Porges, 2001). The dorsal motor nucleus of the vagus receives inputs from the hypothalamus, reticular formation, and nucleus of the solitary tract, areas involved in regulating autonomic functions, while the nucleus ambiguus receives inputs from the corticobulbar tract and reticular formation (Pritchard & Alloway, 1999).

The vagus carries not only signals from the brain to regulate heart and visceral activity, but also information about peripheral activity to the brain; vagal afferents outnumber efferents by at least 4:1 (Greenstein & Greenstein, 2000). Afferents terminate in the nucleus of the solitary tract in the medulla, which connects to the paraventricular nucleus of the hypothalamus and the pontine parabrachial nucleus, which in turn connects to the amygdala, hypothalamus, and bed nucleus of the stria terminalis, central structures involved in emotion, motivation, and learning (Pritchard & Alloway, 1999).

Frequencies of Heart Rate Variability

Due to the interaction of these influences on the heart, the heart continually varies its rate of beating. Specific heart rate variability (HRV) frequencies arise from different physiological processes. Most often, four bands of heart rate variability are described: high frequency (HF) at 0.15-0.4 Hz, low frequency (LF) at 0.05-0.15 Hz, very low frequency (VLF) at 0.003-0.05 Hz, and ultra low frequency (ULF) at less than 0.003 Hz (Berntson, et al., 1997; Stein & Kleiger, 1999).

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Of particular interest to psychophysiologists is HF variability, the range of HRV at frequencies corresponding to normal adult human resting respiration rates. Variation in the HF band is called respiratory sinus arrhythmia (RSA) and is mediated by the parasympathetic nervous system primarily through respiration-related activity in the vagus nerve (Berntson, et al., 1997; Stein & Kleiger, 1999). Fibers from the nucleus ambiguus coordinate respiratory and cardiac activity and participate in respiratory rhythm generation, so increases in ventral vagus activity increases RSA (Porges, 1995; Porges, 2001). A great deal of research has examined the relationship between the autonomic nervous system and RSA, with the consensus that RSA can serve as an index of changes in vagal heart control, though it is not a pure or absolute measure (reviewed in Berntson, Cacioppo, & Quigley, 1993; Stein & Kleiger, 1999).

Variation in the longer frequency bands (low, very low, and ultra low) is less useful in psychophysiology, but is potentially useful in other fields, such as in the study of chronic heart failure (reviewed in Guzzetti, Magatelli, Borroni, & Mezzetti, 2001). Low frequency HRV is also caused by a combination of sympathetic and parasympathetic nervous system activity, and is affected by changes in blood pressure (Berntson, et al., 1997; Stein & Kleiger, 1999). Very low frequency HRV is not well understood, but is thought to be caused by changes in the peripheral vasomotor or plasma rennin-angiotensin systems (Berntson, et al., 1997; Stein & Kleiger, 1999). Ultra low frequency HRV is related to slower physiological rhythms, such as thermoregulatory, neuroedndocrine, and circadian cycles (Berntson, et al., 1997; Stein & Kleiger, 1999).

Electrocardiograms

The heart's electrical activity produces a strong signal that can be easily recorded using an electrocardiogram (EKG). EKG signals are well understood; they have been recorded and studied since early in the 20th century (Brownley, Hurwitz, & Schneiderman, 2000). The waveforms present in EKG recordings correspond to different physical stages of the heart beat. A sample EKG signal appears in pane a of Figure 1.1, while a schematic beat with labeled waveforms is in pane b. The P wave is caused by the firing of the sinoatrial node and corresponds to the contraction of the atria. The QRS complex of waves is caused by electrical activity in the Purkinje plexus (electrical conduction fibers), and corresponds to the contraction of the ventricles. Finally, the T wave is caused by recovery of baseline electrical activity and corresponds to filling the atria with blood (Kapit & Elson, 1993).
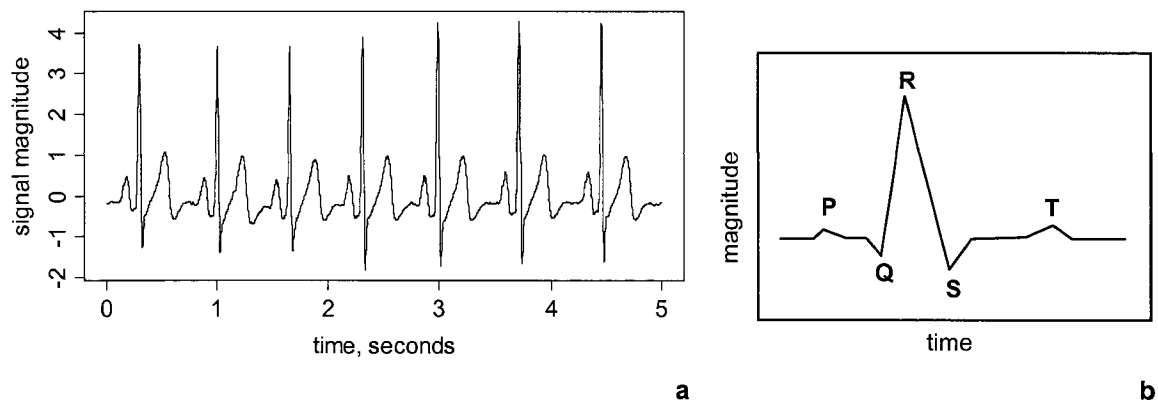
Figure 1.1. EKG heart beat signals. Pane a: actual signal. Pane b: schematic signal, with waveforms labeled.

Heart Rate Variability Analysis

Numerous methods have been developed for HRV quantification and interpretation (reviewed in Berntson, et al., 1997; Stein & Kleiger, 1999; Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996). In general, these analysis techniques fall into two categories: time and frequency domain measures. Additional methods focused on nonlinear properties (such as Poincaré plots) are being investigated but are not yet in wide use (Kamen, 1996). There is evidence that time and frequency domain measures produce comparable results in many cases; the choice of HRV measure in any particular situation depends on the specific research or clinical purpose of the analysis (Friedman, Allen, Christie, & Santucci, 2002; Grossman, van Beek, & Wientjes, 1990).

Time Domain Measures

Time domain HRV measures are suited to many psychophysiological experiments because they can be calculated over very short time periods and make few assumptions about the statistical structure of the variability. A critical consideration when using time domain HRV measures is that it is only valid to compare measures calculated from recordings of equal length. The recording length must be taken into account because the measures increase as recording length increases, due to increasing HRV over time because of factors such as cognition and diurnal cycles. Commonly used time domain measures are described below, but this is not an exhaustive list. Other time-domain measures (such as geometric methods) are more appropriate for clinical applications or require longer time periods than usually feasible in psychophysiological research.

Simple Measures

If short recordings of a fixed length are used, even very simple measures may produce interesting findings, such as when quantifying the cardiovascular component of orientating responses (e.g. Niepel, 2001; Stekelenburg & van Boxtel, 2002). With these very short (less than a minute) recordings, simple measures are the only option, since too few beats are present to quantify the other measures. Simple measures convert the EKG into a series of RR intervals then quantify characteristics of the series, such as the longest and shortest intervals, the difference in length between longest and shortest interval, or the mean RR interval length (which can be transformed to the mean heart rate) (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996).

Related to the simple measures are measures used to quantify variation in long (such as 24-hour) recordings by dividing the long recording into short segments (often 5 minutes in length) (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996). One example of this type of measure is the standard deviation of the mean heart rate in the short segments (SDANN), which is correlated to ULF power; another is the mean of SDNN in each the segment (SDNNIDX), which correlates with LF and VLF power (Stein & Kleiger, 1999).

Short-term Variability Measures

Short-term time domain variability measures are highly correlated (sometimes even mathematically related) both with themselves and with RSA. As with other time domain measures, short-term variability measures should only be compared for recordings of the same duration. One of the most common short-term variability measures is the standard deviation of the RR interval series, the standard deviation of the normal-normal intervals (SDNN) (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996). The square of SDNN (variance of the RR interval sequence) is directly related to the total power calculated by spectral analysis; both measures estimate overall HRV, the variability due to all cyclical components affecting the heart rate (Stein & Kleiger, 1999; Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996). A related measure is the standard deviation of successive differences (SDSD), the standard deviation of the difference in length between successive RR intervals (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996). Like

RSA and SDNN, SDSD measures short-term changes in heart rate which are related to changes in parasympathetic activity.

Three additional measures, pNN50, rMSSD, and MSD, estimate short term (high-frequency) variations in heart rate and are correlated with each other as well as with HF power calculated using spectral methods (Stein & Kleiger, 1999). rMSSD is the square root of the average squared successive differences in RR interval lengths, while pNN50 is the proportion of RR intervals more than 50 msec different in length from the preceding RR interval (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996), and MSD is the mean of the absolute values of successive HR differences (Friedman, Allen, Christie, & Santucci, 2002).

Peak-Valley RSA

Peak-valley RSA is a time domain method for RSA quantification. Peak-valley RSA is measured separately for each breath as the difference in time between the longest and shortest RR interval within the breath, with the additional requirement that the longest RR interval occurs after the shortest (Berntson, et al., 1997; Grossman, van Beek, & Wientjes, 1990; 1996). Two methods of implementing this definition frequently appear in the literature: the difference between the shortest RR interval occurring during inspiration and the longest RR interval occurring during expiration, or the difference between the shortest and longest RR intervals occurring within a single breath (but not requiring the longest to occur during expiration or the shortest during inspiration) (Grossman, van Beek, & Wientjes, 1990; Ritz, Thons, & Dahme, 2001). In both methods, a breath is assigned a difference of zero if the longest RR interval precedes the shortest.

When the first implementation method is used (dividing each breath into its inspiration and expiration), the times of inspiration and expiration are usually forwarded, due to reported phase lags between the onset of inspiration and onset of heart period shortening (Eckberg, 1983). Grossman, van Beek, and Wientjes (1990) recommend forwarding each window by 750 ms, while Uijtdehaage (1994) recommends the larger of 1000 ms or an average RR interval. The technique described by Grossman, van Beek, and Wientjes (1990) often appears in the literature (e.g. Palomba, Sarlo, Angrilli, Mini, & Stegagno, 2000; Reyes del Paso, Godoy, & Vila, 1992; Sarlo, Palomba, Angrilli, & Stegagno, 2002). In the second implementation method breaths are not divided into inspiratory and expiratory windows, so no window forwarding is required (e.g. Ritz, Thons, & Dahme, 2001).

Frequency Domain Measures

Spectral methods use power spectral density analysis methods to estimate the distribution of power (variance) explained by periodic oscillations of the heart rate at different frequencies (discussed more fully in Berntson, et al., 1997; Stein & Kleiger, 1999; Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996). These methods, often using Fast Fourier Transforms or autoregressive modeling, quantify the amount of signal variation in the frequency bands (HF, LF, VLF, ULF) previously described (Berntson, et al., 1997). In short recordings (2 - 5 minutes), only measurements of the LF and HF bands are recommended (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996). Spectral analyses of long recordings, similar to the time-domain measures SDANN and SDNNIDX, are also possible; one such measure is total power, an estimate of the total variation due to all causes over 24 hours (Stein & Kleiger, 1999).

Frequency domain measures are sensitive to nonstationarities (periods during which the heart rate changes unpredictably) in the heart signal (Berntson, et al., 1997). Nonstationarities are common in heart signals, since subjects continually move or engage in tasks or cognitions that affect heart rate. Various detrending methods (e.g. Tarvainen, Ranta-aho, & Karjalainen, 2002) have been proposed to remove these nonstationarities from the data and allow accurate spectral analysis, but recent work by Friedman, Allen et al. (2002) suggests that stationarity of the heart rate signal may not be of practical importance for analysis of short (3 to 7 minute) recordings.

Clinical Findings

Clinically, a reduced HRV is a warning sign of diabetic neuropathy or increased likelihood of complications and death after a heart attack (reviewed in Stein & Kleiger, 1999; Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996). While decreased HRV is consistently shown to be related to various cardiac disorders, it is not sufficient on its own to serve as a screening test (Stein & Kleiger, 1999). An abnormally low HRV is associated with a risk of death from cardiac events, but the mechanism is not understood (Huikuri, et al., 1999; Stein & Kleiger, 1999). Regular aerobic exercise, by contrast, is associated with an increase in HRV (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996), and HRV is increased in fit individuals (Stein & Kleiger, 1999), presumably due to increased vagal tone and decreased sympathetic activity (Kamen, 1996).

Relationships have also been found between HRV and psychological conditions. HRV changes associated with psychological manipulations, even relatively simple ones such as mental arithmetic, are variable and dependant on subject and protocol (Berntson & Cacioppo, 2004). Several consistent patterns have been found, however, including a reduction in HF HRV in subjects with anxiety and depression (Berntson & Cacioppo, 2004). It is possible that these findings are related to findings of abnormal HRV in cardiac patients, since anxiety can serve as a predictor of coronary heart disease, and anxiety and depression often occur together (Berntson & Cacioppo, 2004).

RSA analysis in particular has proven fruitful across many subject populations and experimental questions. For example, RSA predicted recovery from major depression (Rottenberg, Wilhelm, Gross, & Gotlib, 2002), and HRV characteristics distinguished depression due to bereavement from other major depression (O'Connor, Allen, & Kaszniak, 2002). Beauchaine (2001) reviewed studies using RSA as an index of vagal tone in terms of behavioral and emotional theories, while studies utilizing HRV and RSA measures of anxiety and panic attack subjects were reviewed by Friedman and Thayer (Friedman & Thayer, 1998).

Respiration Psychophysiology

Respiration is of interest to psychophysiologists because alterations in respiration are associated with changes in mood, and its changes can affect measures of other physiological activity (such as skin conductance and HRV). Respiration is a complex system, partly because it is under both conscious and unconscious control. People can alter their respiration pattern when desired (such as by breath-holding or when playing an instrument), but under resting conditions each individual's pattern of respiration has been found to be consistent, even over long periods (Benchetrit, et al., 1989; Shea & Guz, 1992; Shea, Walter, Murphy, & Guz, 1987). Causes of variation in individual respiration patterns has been described (Boiten, 1993; Shea, Benchetrit, Pham Dinh, Hamilton, & Guz, 1989; Shea & Guz, 1992; Shea, Walter, Murphy, & Guz, 1987). There is evidence that emotional states may be associated with alterations in respiratory pattern (e.g. Boiten, 1998). Interest in respiration as an independent source of data for psychophysiologists has been less strong than for other systems (reviewed in Harver & Lorig, 2000; Wientjes, 1992; Wientjes & Grossman, 1998).

Biological Basis

Respiration is the process of moving air in and out of the lungs in order to oxygenate the blood and remove carbon dioxide, processes which take place in the alveoli. Most (75%) of the air exchange is accomplished by diaphragm movements, the remainder by neck and abdominal muscles (Aminoff & Daroff, 2003). The diaphragm and intercostal muscles are controlled by a collection of nuclei in the medulla, the medullary respiratory center, via the phrenic nerve, which comes from cervical roots C3-C5 (Aminoff & Daroff, 2003; Pritchard & Alloway, 1999). The medulla is the primary generator of the respiratory rhythm, but its rhythm is modulated by inputs from the cortex and pons, as well as from chemoreceptors in the aorta and carotid arteries and pulmonary stretch receptors in airway smooth muscles (Aminoff & Daroff, 2003).

The medullary respiratory center is composed of multiple nuclei in the reticular formation, which can be divided into the dorsal and ventral nuclear groups (Greenstein & Greenstein, 2000). The dorsal group is primarily responsible for respiratory cycle timing and has inspiratory motor neurons located near the nucleus tractus solitarius, which has sensory inputs from the vagus and glossopharyngeal nerves (Aminoff & Daroff, 2003). The ventral group is composed of four nuclei centered around the nucleus ambiguus: the nucleus retroambigualis which innervates expiratory muscles, the nucleus paraambigualis, which innervates inspiratory muscles, the nucleus retrofacialis (Bötzinger complex) which inhibits inspiration, and the nucleus ambiguus, which innervates the larynx and pharynx via the glossopharyngeal and vagus nerves (Aminoff & Daroff, 2003).

Other brain areas also regulate respiration. Areas of the pons (nucleus parabrachialis lateralis and Kölliker-Fuse nucleus) are important for regulating respiratory rhythm (switching from inspiration to expiration) and tidal volume (Aminoff & Daroff, 2003). The midbrain also influences respiration, through projections from the periaqueductal gray matter, while the extrapyramidal system is especially important for voluntary control (Aminoff & Daroff, 2003). The caudal hypothalamus is involved in increasing respiration rate in response to stimuli such as exercise and threat, while the ventral supplemental and primary motor areas of the cortex control voluntary respiratory control, including for speech (Aminoff & Daroff, 2003). Limbic areas, including the cingulate gyrus, uncus fornix, and amygdala also participate in respiratory control; stimulation of these areas inhibits respiration (Aminoff & Daroff, 2003).

Measurement Devices

Respiration is one of the more difficult signals to record, primarily because it occurs without generating a strong electrical signal. Respiration measurement is also complicated by the ease of conscious control. Making subjects aware of their respiration, through directions or by the measurement device itself, can cause significant changes in the respiration depth and pattern (Askanazi, et al., 1980; Boiten, Frijda, & Wientjes, 1994; Gilbert, Auchincloss, Brodsky, & Boden, 1972). To minimize these changes respiration should be recorded using the least intrusive device possible. Respiration measurements are also susceptible to artifacts that may arise from activities such as talking, eating, shifts in posture, or physical activity.

When measuring airway resistance, tidal volume, or other physical lung function characteristics, complex invasive techniques, such as spirometry or body plethysmography, must be used. These methods, frequently used in medical settings, are extremely accurate, but somewhat limited in applicability to psychophysiological studies due to their tendency to disrupt normal respiration. Ritz et al (Ritz, et al., 2002) detail the use of these techniques in the context of psychophysiological experiments; they were also used in early psychophysiological research (Woodworth, 1938), with infant subjects (Korten & Haddad, 1989), and with mood induction procedures (Ritz, George, & Dahme, 2000).

Devices used to detect the flow of air due to breathing include spirometers, thermistors, and flowmeters. Spirometers measure the total volume of air forcefully expired, and are used to measure tidal volume and as an indirect measure of airway resistance (Harver & Lorig, 2000; Ritz, et al., 2002). Thermistors are attached near the nose or mouth and record the difference in temperature between inspired and expired air. They do not yield volume measurements but rather breath phase (inspiration or expiration). Their use is limited, however, because they require the subject to breathe via the measured airway (nose or mouth) exclusively and may have time lags in their recordings (Harver & Lorig, 2000). Thermistors have been used successfully in research when only an approximate measure of respiration phase is required (e.g. Chen, et al., 2000; Kira, et al., 2001; Yardley, Gresty, Bronstein, & Beyts, 1998). Flowmeters are used in conjunction with a face mask or mouth piece and nose clip, and record the amount of air moving through them during each breath. While they have been used in psychophysiological research (e.g. Masaoka & Homma, 2000), they are even more intrusive than thermistors, since a mouthpiece or airtight mask must be used, and so may cause alterations in the respiration pattern.

Rather than monitoring air movements, strain gauges and respiratory inductive plethysmography measure respiration by recording body movements. Strain gauges detect the change in tension in an elastic belt snugly fastened around the chest or abdomen, producing a continuous record of its circumference changes. Respiratory inductive plethysmography, by contrast, indirectly detects changes in body circumference by analyzing changes in electric current running through wire coiled into two bands, one each around the abdomen and chest (Harver & Lorig, 2000). If the output from the two bands used in respiratory inductive plethysmography, or from chest and abdominal strain gauges, is carefully calibrated against a spirometer, accurate measurements of breath volume can be obtained in addition to timing (Harver & Lorig, 2000). Frequent recalibration is necessary however, as accumulation of small movements in the body and bands will lead to ever more inaccurate recordings (Wientjes, 1992).

When only chest circumference is recorded by a strain gauge (rather than both the chest and abdomen simultaneously), it is not possible to obtain accurate measurements of respiration volume; only timing measures can be collected (Harver & Lorig, 2000). While using a single belt thus reduces the amount of data that can be recorded, the ease of measurement may outweigh the desire to record respiration volume in some cases. For example, the inspiration and expiration times can be recorded without the lengthy and potentially difficult calibration process that is required when using respiratory inductive plethysmography. This may be a significant advantage when the subject's breathing should be as natural as possible, such as in mood induction procedures.

Chest Strain Gauge Respiration Signals

A respiration signal derived from a strain gauge attached to a chest strap produces a single channel of data corresponding to the circumference of the chest over time. As the subject inhales the chest expands, increasing in circumference and causing a rise in the signal. Correspondingly, as the subject exhales the chest circumference decreases and the signal falls. The alternation of inhalations and exhalations results in an irregular sinusoidal pattern, in which the peaks are the end of inspiration (start of expiration) and the troughs are the end of expiration (start of inspiration). A representative raw respiration signal obtained from a chest belt strain gauge appears in Figure 1.2 (pane a). The absolute height of the troughs is fairly constant over the recording, while the height of the peaks varies. Differences in peak or trough height such as these are extremely common in strain gauge belt recordings and may indicate the depth of respiration, but may also be due to a shift from

chest to abdominal breathing, a change in posture, or some other factor, so no conclusions about the respiration volume or depth should be drawn.

In the broadest terms, a breath can be divided into four parts: inspiration, post-inspiratory pause, expiration, and post-expiratory pause. Figure 1.2 (pane b) illustrates these breath parts as recorded by a single chest strain gauge. Other methods of recording respiration, such as via a flowmeter, also produce a sinusoidal tracing, but the point between inspiration and expiration is the point of zero air flow, between the peak and trough, rather than the peak. Using Figure 1.2 pane b, inspiration time can be defined in several ways: from trough to peak, during active inspiration only (b1 to c), by including the post-inspiratory pause (if present) in the inspiration (b1 to d), or by including the previous post-expiratory pause (if present) in the inspiration (a1 to c). By convention, respiratory periods are defined trough-to-trough (one inspiration and the following expiration), rather than peak-to-peak (an expiration and the following inspiration) (Grossman, Karemaker, & Wieling, 1991; Wientjes, 1992).

Respiration Measures

A wide variety of measures exist to quantify respiration. These measures fall into three broad categories: volume and timing parameters, breathing curve morphology

a   b
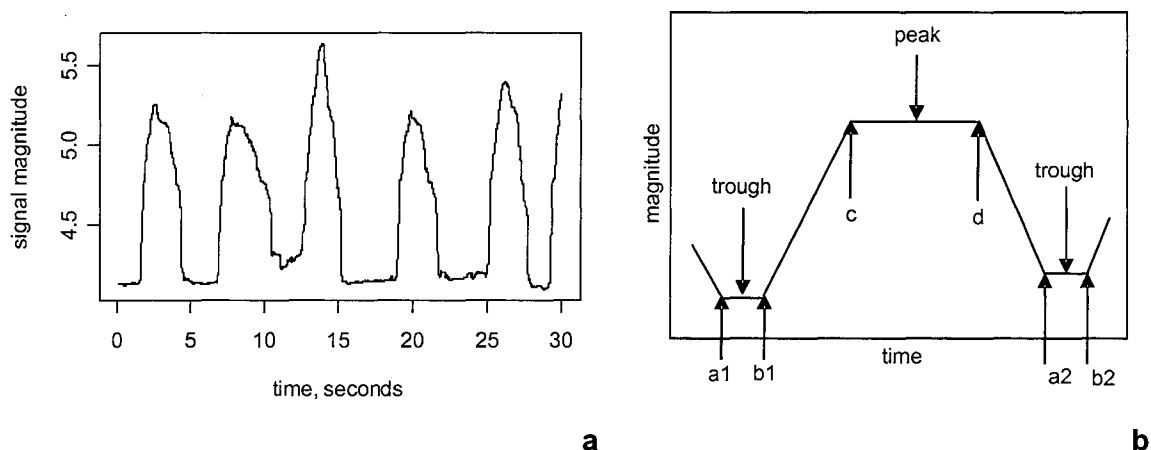
Figure 1.2. Strain gauge chest belt respiration signals. Pane a: actual signal, collected with a TSD201 Respiratory Effort Transducer (BIOPAC Systems, Inc. Santa Barbara, CA). The troughs correspond to the point of minimum chest circumference, between expiration and inspiration. Peaks correspond to the point of maximum chest circumference, between inspiration and expiration. Pane b: schematic signal, with peaks and troughs as labeled. A post-expiratory pause begins at a1 and ends at b1, as well as between a2 and b2. A post-inspiratory pause begins at c and ends at d.

descriptions, and gas exchange measures (Boiten, Frijda, & Wientjes, 1994). The best measures to use for any particular experiment depend on its goals and measurement devices; measures of expiratory CO_2 levels can not be obtained using inductive plethysmography, for example. Traditionally, respiration analysis focuses on volume and timing parameters, which are usually analyzed in the time domain using methods similar to time domain HRV analysis. Comparatively few studies examine breathing morphology or qualitative analyses such as frequency of sighing and yawning, or changes in respiration pattern over time. Generally, multiple respiration timing measures are quantified and analyzed when respiration is used in psychophysiology experiments. Single measures, such as respiration rate, are not sufficient to fully describe the relationship between respiration and metabolic needs, since multiple changes (such as an alteration in inspiration time or pauses) can cause identical changes in respiration rate (Wientjes, Grossman, & Gaillard, 1998).

Timing and Volume Measures

The fundamental respiration timing measure is total breath duration, T_{tot}, or respiration rate (RR), its equivalent when converted to respiratory cycles per minute. T_{tot} is the length of a complete respiratory cycle, which is composed an inspiration and the following expiration. The components of T_{tot}, the duration of inspiration (T_i) and expiration (T_e), may be analyzed separately or combined into the inspiratory duty cycle, the proportion of the breath duration taken up by inspiration (T_i/T_{tot}). Changes in the inspiratory duty cycle indicate changes in respiratory timing independently of the absolute amount of time spent in each phase (Wientjes, 1992). Additionally, the duration of the post-inspiratory (P_i) and post-expiratory (P_e) pauses may be measured (Wientjes, 1992; Wientjes, Grossman, & Gaillard, 1998).

Several volume measures are commonly reported. The fundamental respiration volume measure is tidal volume (V_T), the volume of air displaced in a complete respiratory cycle (Wientjes, 1992). Tidal volume is of interest on its own, but is also used in other measures, such as when defining sighs (Wilhelm, Trabert, & Roth, 2001) or calculating the inspiratory flow rate (V_T/T_i), an index of inspiratory drive intensity (Wientjes, 1992). Another common volume measure is minute ventilation (V_E or V_{MIN}), the total volume of air moved by breathing per minute (Wientjes, 1992).

Other Measures

Multiple studies have used novel methods of describing respiration morphology. For example, Weintraub et al. (2001) used a mixture of qualitative descriptions and quantitative values to describe periodic respiration in adults and infants, while Benchetrit and Shea quantified typical breath shape in twins (Benchetrit, et al., 1989; Shea, Benchetrit, Pham Dinh, Hamilton, & Guz, 1989). Statistical methods from HRV analyses have also been used with respiration data, including complex demodulation and rMSSD (e.g. Wilhelm, Trabert, & Roth, 2001).

Clinical Findings

Abnormal patterns of respiration, especially hyperventilation, have been associated with clinical conditions including mood disorders (anxiety, panic disorder, and depression), chronic pain, asthma, chronic fatigue syndrome, and functional cardiac disorder (reviewed in Wientjes, 1992; Wientjes & Grossman, 1998; Wilhelm, Gevirtz, & Roth, 2001). The finding of abnormal respiration patterns in people with certain mood disorders (e.g. panic disorder, depression, anxiety) confirms a physiological component that has long been reported subjectively. Efforts have begun to quantify patients' physiological parameters, in the hope of identifying physiological/respiratory subtypes of the conditions and so perhaps develop treatments for the physiological abnormalities/maladaptive behaviors (reviewed in Battaglia & Ogliari, 2005; Wilhelm & Roth, 2001).

Many abnormal respiration clinical findings are related to hyperventilation. Unlike hypoventilation, the onset of hyperventilation can be very rapid, and it may persist for lengthy periods. This difference is due to asymmetry in the physiological control mechanisms underlying respiration: increased ventilation is promptly induced if CO_2 builds up in the body, but a decrease in ventilation is only mildly stimulated if CO_2 drops (Wilhelm, Gevirtz, & Roth, 2001). This difference does not mean that a reduction in arterial CO_2 (hypocapnia) is harmless, however. Among other effects, hypocapnia increases blood pH, interferes with O_2 delivery to some brain regions, changes coronary artery tone, and changes levels of extracellular calcium and potassium levels, which affect neuronal excitability (Wilhelm, Gevirtz, & Roth, 2001). Subjectively, these changes are experienced as shortness of breath, faintness/dizziness, palpitations, and tingling in the extremities, symptoms often reported in clinical populations (Battaglia & Ogliari, 2005; Wilhelm, Gevirtz, & Roth, 2001).

Respiration has been found to be irregular (e.g. frequent sighing, tidal volume instability) in subjects with anxiety disorders, especially panic disorder (Wilhelm, Trabert, &

Roth, 2001); these respiration irregularities can lead to hyperventilation. Evidence for a relationship between these disorders and respiration patterns is strengthened by reports that therapy (biofeedback techniques such as monitoring respiration rate or pCO_2) aimed at correcting the abnormal respiration provides clinical improvement in the mood disorder (Wientjes, 1992; Wientjes & Grossman, 1998; Wilhelm, Gevirtz, & Roth, 2001). Anxiety may lead to an increase in respiration rate, as well as changes in its pattern (longer T_e), unrelated to metabolic demands (Masaoka & Homma, 2001). These respiration changes may be driven by the emotional response; Masaoka & Homma (2000) observed respiration-coordinated increases in amygdala activation during a study using anticipatory anxiety.

Electrodermal Psychophysiology

Changes in the electrical properties of the skin (electrodermal activity, EDA) have been studied since they were first observed in the second half of the 19th century (Dawson, Schell, & Filion, 2000). As EDA was studied it became apparent that some changes were due to mental, as opposed to strictly physical (such as temperature regulation or movement), processes. Further study divided EDA into two subtypes: tonic skin conductance level (SCL) and rapid fluctuations called skin conductance responses (SCRs). In brief, tonic SCL is a measure of general arousal which decreases with relaxation and increases with alertness (e.g. Alexander, et al., 2005; Nagai, Critchley, Featherstone, Trimble, & Dolan, 2004). SCRs, in contrast are produced in response to distinct stimuli, varying in size with stimulus characteristics such as novelty, emotional content, and significance (Dawson, Schell, & Filion, 2000).

Biological Basis

The EDA changes measured as SCL and SCRs derive from the physiological characteristics of the skin, especially sweat gland activity. The skin is a complex layered organ containing many specialized structures, including sensory receptors, hair follicles with associated sebaceous glands, blood vessels, and sweat glands. The skin's structure varies in thickness and structure with location; some areas of the skin are hairless, others have hair, and the density of sweat glands varies. In all cases, however, the skin is a poor conductor of electricity. Salty water, contained in sweat glands, conducts electricity well, thus EDA is related to sweat glands and sweating.

Sweating is most often associated with thermoregulation: increased sweating cools the body as moisture evaporates. Sweating is produced by stimuli besides thermoregulation, however, such as after eating spicy foods (gustatory sweating), or when a small area of the

skin is stimulated by heat or needle punctures (Boucsein, 1992). Of most interest to psychophysiology researchers is emotional sweating, sweating related to psychological or emotional states (sweaty palms when anxious, for instance). Different types of sweating are often observed on different areas of the body: thermoregulatory sweating on the torso, gustatory sweating on the face, and emotional sweating on the palms and feet. Some of this apparent specificity is due to differing sweat gland densities (sweat glands are especially dense on the palms and feet, so emotional sweating may be more apparent in these areas), but the specificity is also due to differences in enervation (e.g. gustatory sweating) (Boucsein, 1992; Dawson, Schell, & Filion, 2000).

Sweating is due to activity in the sweat glands, which produce the liquid (primarily, but not exclusively, salt water) that reaches the skin's surface. The amount of sweating is determined by the amount of sweat produced by the glands and the number of glands actively producing sweat, which in turn is determined by the sympathetic nervous system; autonomic nervous system fibers from the sympathetic branch innervate the secretory portions of sweat glands (Boucsein, 1992). Since salt water is a good conductor of electricity, the amount of sweat in the glands and the number of glands producing sweat can be estimated by measuring the level of skin conductance, which therefore is an index of changes in sympathetic nervous system activity (Dawson, Schell, & Filion, 2000).

Central Control

Multiple brain centers, at different levels of the central nervous system, are responsible for the sympathetic nervous system activity that control sweat gland enervation; the central control is complex, not due to any single mechanism but rather to multiple areas that act at least partially independently of each other (reviewed in Aminoff & Daroff, 2003; Boucsein, 1992; Dawson, Schell, & Filion, 2000). Efferents from the brain travel down the intermediolateral spinal column, synapsing in the paravertebral ganglia; lesions in the ganglia or spinal column can inhibit sweating in the corresponding dermatome (Aminoff & Daroff, 2003).

Several of the central structures which control sweating have been identified. The primary central control of sweating is the ipsilateral hypothalamus. The preoptic area of the anterior hypothalamus plays a large role in thermoregulatory sweating; increased output leads to increased sweat gland activity, as does amygdala activation (Aminoff & Daroff, 2003). Areas of the frontal cortex, reticular formation, and basal ganglia also influence sweating; stimulation of the reticular formation can trigger SCRs (Dawson, Schell, & Filion, 2000). Excitatory impulses from the premotor cortex travel through the pyramidal tract;

lesions of the premotor cortex can cause excessive sweating (Aminoff & Daroff, 2003; Dawson, Schell, & Filion, 2000). These different cortical structures may control the different methods of inducing EDA: the reticular formation controls changes due to muscle tone and movement; the hypothalamus thermoregulation; the amygdala affective processes; the premotor cortex fine motor control, and the prefrontal cortex orientation and attention (Dawson, Schell, & Filion, 2000).

Some of the research to identify the cortical structures regulating EDA was done using the lesion method. The consensus from early research was that left hemisphere lesions were not associated with EDA abnormalities, while right hemisphere and bilateral lesions were associated with abnormal EDA, generally a reduction or elimination in EDA (Tranel & Damasio, 1994). Additional recent research has both refined and muddied this view. Tranel and Damasio (1994) found that bilateral ventromedial frontal damage was associated with EDA impairment, and suggestions that anterior cingulated gyrus and right inferior parietal damage was associated with EDA impairment. Zahn, Grafman et al. (1999), in an extension of that study, also found abnormal EDA responses in subjects with frontal damage, but major deficits only to tasks that require effortful mental processing (EDA responses were more normal to stimuli provoking orienting responses). Zahn, Grafman et al. (1999) did not confirm the previous finding of ventromedial frontal damage associated with strong deficits in EDA, but did confirm the general observation that right hemisphere and bilateral lesions are more associated with deficits in EDA than left hemisphere lesions.

Measurement and Quantification

EDA measurement quantifies two broad types of activity: tonic and phasic. The tonic skin conductance level changes slowly, over the course of minutes and hours, and is related to the general level of arousal. The phasic component, skin conductance responses, is composed of rapid changes, on the scale of seconds. SCRs consist of a rapid increase in skin conductance followed by a gradual decrease, returning to the baseline SCL. SCRs are often associated with distinct stimuli, such as after a loud noise or following a deep breath. Figure 1.3 contains actual and schematic skin conductance recordings, illustrating SCRs superimposed on a changing SCL.

EDA quantification is made more complex by wide variation between people and numerous triggers. Absolute resting SCL varies between normal individuals, and within individuals associated with mental and physical state changes (Dawson, Schell, & Filion, 2000). EDA variability and magnitude is associated with age; age-related EDA reduction is not well understood; it may be due to changes in sweat gland structure or activity, or to
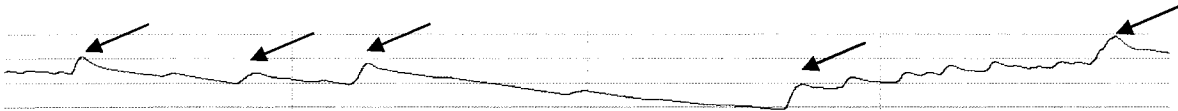
Figure 1.3. Skin conductance recording showing waves of SCRs (several marked with arrows) superimposed on the underlying SCL.

changes in the underlying innervation or central signals (Tranel & Damasio, 1994). There are also interactions between respiration and EDA; including respiration as a covariate when analyzing EDA may improve accuracy. Many normal subjects are EDA *nonresponders*; they do not produce SCRs or have typical SCL changes. Estimates of the proportion of nonresponders in normal populations vary, most ranging from 5 to 10%, a few sources estimating up to 25% (Boucsein, 1992; Dawson, Schell, & Filion, 2000; Tranel & Damasio, 1994).

EDA Recording

Recording techniques for EDA in psychophysiology experiments are well described and methodologies have been standardized (reviewed in Boucsein, 1992; Dawson, Schell, & Filion, 2000). In general, EDA is measured as skin conductance (as opposed to skin potential), with a pair of electrodes attached the palmar surface of the fingers or hands. These electrodes pass a small current with constant voltage through the skin; the amount of current passing between the electrodes is the skin conductance, usually reported in μS (Dawson, Schell, & Filion, 2000; Fowles, et al., 1981). The palms are used because palmar sweating is highly associated with mental processes but more convenient (e.g. less callusing, not covered by clothing) than the feet (Boucsein, 1992). EDA measurement is very sensitive to chemical and electrical properties of the skin, so it is important that the hands be clean and freshly washed (Dawson, Schell, & Filion, 2000). Recordings are made digitally, resulting in a single channel of data corresponding to the skin conductance over time, as in Figure 1.3 (pane b).

SCR Quantification

Although methodological differences still exist, there is a extensive literature detailing methods of measuring distinct SCRs (e.g. Alexander, et al., 2005; Boucsein, 1992; Lim, et al., 1999; Lim, et al., 1997). Most commonly, quantification methods measure the amplitude and/or duration (or components of the duration, such as *rise time*) of each SCR. SCR amplitude is measured as change from signal baseline (i.e. from the SCL), rather than from zero. Instead of amplitude and duration measurements researchers occasionally quantify

SCRs as the area under the curve. Boucsein (1992) discourages the use of area measures because the relationship between SCR amplitude and time course can confound the estimates.

In most people EDA is constantly occurring, producing small SCL fluctuations, so it can be difficult to determine which changes are large enough to be considered SCRs. Traditionally, fluctuations with amplitude changes greater than 0.5 μS are classified as SCRs (Dawson, Schell, & Filion, 2000). This amplitude was the smallest that could be reliably distinguished using older paper-based EDA recording devices. Digital techniques can easily distinguish much smaller amplitude responses, and some researchers are starting to use other minimum amplitudes, such as 0.02 μS (e.g. Naveteur, Buisine, & Gruzelier, 2005; O'Keeffe, Dockree, & Robertson, 2004; Papousek & Schulter, 2001). A few studies calculated minimum SCR amplitude thresholds for each subject, citing differences in individual EDA (e.g. Bensafi, Tsutsui, Khan, Levenson, & Sobel, 2004; May, Paulinyi, & Vassy, 2005), or used a threshold combining amplitude with other characteristics of the SCR, such as its slope (e.g. Papousek & Schulter, 2001).

SCL Quantification

In contrast to fairly distinct SCRs, SCL changes are relatively difficult to quantify, and a consensus about the best method does not exist at this time. Two measures for quantifying SCL are common in the literature: the rate (usually count per minute) of non-specific SCRs (NS-SCRs, NS.EDRs) and the SCL (Dawson, Schell, & Filion, 2000). NS-SCRs are SCRs not caused by a distinct stimulus, either external (such as a surprising photograph or noise) or internal (such as a deep breath or sudden movement). Boucsein (1992), after reviewing relevant research, concludes that NS-SCR rate and SCL are correlated, but not redundant, measures.

A large number of studies use NS-SCR rate to quantify tonic electrodermal activity, but the definition and quantification techniques vary. A typical method is that used by Sponheim, Iacono et al. (2003): NS-SCRs were defined as SCRs greater than 0.05 μS in magnitude occurring more than ten seconds after a stimulus; differences in NS-SCR rate were reported between schizophrenic patients, their first-degree relatives, and control subjects. Occasionally, aspects of the NS-SCRs (such as amplitude) are measured, instead of just the rate (e.g. Carrillo, et al., 2001; Schneider, Kuhl, & Walach, 2004).

As with NS-SCR rate, various methods have been used to quantify tonic SCL. Boucsein (1992) suggested quantifying SCL as the mean of the SCL at the onset of each NS-SCR (omitting overlapping NS-SCRs). A somewhat similar method was used by

Naveteur, Buisine et al. (2005) in a study of subjects with anxiety. They quantified changes in SCL over the course of the experiment as the skin conductance at the onset of each stimulus (i.e. before the subject could have started responding to the stimulus). Collet, Petit et al. (2005) quantified SCL in a study of EDA while driving. SCL was quantified by using the average SCL during a pre-task rest period as each subject's baseline, then dividing the average SCL measured during periods throughout the task by this value. These "normalized" values were compared between subjects. Additionally, they measured the mhomic perturbation duration, the length of time a clear response was present while driving. (Lim, et al., 1996) used a similar method, quantifying SCL as the mean skin conductance during discrete periods following each stimulus, while Goldstein, Jerram et al. (2005) averaged the skin conductance within each block of stimuli (both stimuli and intermediate periods).

Clinical Findings

Abnormal EDA has been reported in an assortment of clinical disorders, including schizophrenia, depression, psychopathy, and ADHD. It generally appears that abnormalities in EDA are related to deficits in cortical structures or processing, rather than in the sweat glands themselves. Abnormal EDA in schizophrenic subjects is reviewed by Dawson and Schell (2002), who report two classes of abnormal responding: heightened EDA (raised SCL accompanied by frequent SCRs, including SCRs to innocuous stimuli) and depressed EDA (low SCL accompanied by few SCRs or NS-SCRs). EDA hyperactivity during active psychosis was associated with a poor treatment outcome, and could predict imminent relapse when hyperactivity was found during remission. Abnormal EDA was also found in subjects at high risk of developing schizophrenia before symptom onset; the type of EDA abnormality was related to the type of schizophrenic disorder later developed (Dawson & Schell, 2002).

Tonic and phasic EDA responding is reduced in patients with major depression; the underlying physical cause appears to differ from that causing abnormal EDA responding in schizophrenics (Dawson, Schell, & Filion, 2000). Skin conductance is also related to pain; increasing as pain increases (Fujita, Fujii, Nakamura, Miyauchi, & Takagi, 2000). Several EDA abnormalities are associated with psychopathy, generally involving reduced skin conductance magnitude; skin conductance responses to emotional pictures, personally significant information, and aversive stimuli may all be muted (e.g. Benning, Patrick, & Iacono, 2005; Lorber, 2004; Verschuere, Crombez, De Clercq, & Koster, 2005). EDA abnormalities are present in ADHD subjects, as well, often suggesting dysregulation. These

abnormalities may include increased NS-SCR rate and SCL during states that should produce low arousal, though sometimes abnormally low SCL is reported (e.g. Hermens, Kohn, Clarke, Gordon, & Williams, 2005; Iaboni, Douglas, & Ditto, 1997; van Goozen, Matthys, Cohen-Kettenis, Buitelaar, & van Engeland, 2000).

CHAPTER 2: PERMUTATION TESTING

Abstract

This chapter reviews the use of permutation testing in statistical analysis and describes the ways in which permutation testing techniques are used to determine significance in later chapters of this dissertation.

General Characteristics and History

Permutation testing methods evaluate statistical significance by comparing the statistics describing the data with statistics obtained on reordered arrangements of the same data. The collected data itself is manipulated to provide a distribution from which significance is estimated, rather than on the theoretical population distribution, as is done in inferential statistics. In general, the proportion of reordered data sets that have a greater value on the statistic of interest as the actual data set is the statistic's significance. Therefore, permutation testing is not a different type of statistical test, but rather a different method for determining significance of test results (Byrne, 1993; Edgington, 1995). Randomization tests, the method used here, are a type of permutation test that rely on random assignment of subjects to conditions, with the null hypothesis that all assignments of subject to condition are equally likely (Byrne, 1993; Pesarin, 2001). Randomization and permutation tests differ from resampling methods such as the bootstrap and jackknife in that the data is not sampled with replacement.

Permutation testing to determine significance levels was developed by early eminent statisticians in the 1930s and 1940s, but its application was limited until powerful computers were widely available (Byrne, 1993; Hall, 2003). One of the first permutation tests described was Fisher's Exact Test, created by R. A. Fisher, still used to compare the frequency of binary values in independent groups (Petrie & Sabin, 2000). Permutation testing was used to verify the validity of the p-values produced by certain parametric methods (Edgington, 1995). Permutation testing produces equivalent p-values to parametric tests in many cases, particularly those when the parametric test assumptions (random sampling, homogenous variances, and properly distributed data) are met. Permutation testing may produce more valid p-values than parametric tests when these assumptions are not met, particularly with small sample sizes or outliers are present in the data (Byrne, 1993). The concept of permutation derives from the mathematical field of combinatorics, the study of collections of objects.

Although currently not in common usage in psychophysiology research, recent papers have urged the use of permutation testing in the cognitive sciences (e.g. Byrne, 1993; Hunter & May, 1993; Hunter & May, 2003). Permutation testing is especially valuable when the data does not follow a known distribution, when the variance in different groups is unequal, or when the subjects were not chosen from the population at random, all characteristics common in psychophysiological research (Byrne, 1993). Using parametric tests under these conditions may produce questionable results, but permutation tests will accurately estimate significance (Edgington, 1995). Permutation testing is valuable for cases when subjects were not selected at random from a population, because it evaluates the significance based on the experimental data, rather than on assumptions of the distribution of values in the population. Accordingly, however, it is not possible to extrapolate permutation testing results to a larger population (Hunter & May, 2003). This is not generally a concern in psychophysiology research, but becomes an issue when the research goal is to predict the behavior of large populations, such as in political surveying.

Randomization testing can be used to determine the significance of traditional statistics (such as a t value) but also of novel statistics which capture the relationship of interest, allowing researchers to use the most informative description of experimental results. For example, a mixture of techniques was used by (Prvan & Bowman, 2001), who utilized permutation testing to evaluate the time dependence of data described using Principal Components Analysis (PCA).

Repeated Measures Methods

Permutation testing has been previously used to evaluate significance in repeated measures data sets (generally produced by experiment designs in which multiple measurements are made on each subject). Edgington (1995) covers the use of permutation tests with repeated measures experiments. He does not explicitly discuss time series data (data in which measurements are made on each subject at specific time intervals), but does discuss experiments in which multiple treatments are applied to the each subject, so that every subject experiences every treatment. He then proposes a test to evaluate whether the different treatments produced different responses. The use of permutation testing with repeated measures data where the measurements are time series is explicitly addressed by Good (2001, section 9.1.2). Good describes testing subjects' response profiles for parallelism by calculating the difference between adjacent measurements, then evaluating the significance of the observed differences by permutation testing. In this test the group labels are permuted; the vector of differences for each subject is never separated. A chapter

on permutation testing of repeated measures data is also included in *Multivariate permutation tests* (Pesarin, 2001, chapter 11), which discusses applications to time series data. The article by Hunter and May (2003) also describes methods of using permutation testing with repeated measures data, suggesting that permutations be done within subjects in the case of testing treatment effects when each subject completed each treatment, but that vectors of data be permuted for multivariate designs.

Null Bands and Null Hypotheses

In permutation testing the null hypothesis of *exchangeability* is used (Buja & Rolke, 2005). In this context, *exchangeability* refers to the hypothesis that the data labels can be rearranged. For example, imagine a study comparing the rate of growth in boys and girls. This data would consist of a collection of vectors, each entry in which is the height of a particular child at a measurement time. Each vector of measurements would be labeled with the child's gender. In this case, the null hypothesis is that the vectors do not vary with gender; the gender labels can be exchanged. A null band outlines the area where the summary curves would be expected to be located if the null hypothesis were true. In this example, the vector of heights for each child could be plotted as a curve, with measurement time on the x-axis and height on the y-axis. Summary curves could then be constructed to display the characteristic of interest, such as median height at each measurement time, with separate curves for boys and girls. Null bands could be constructed for each summary curve (one for the boys, another for the girls), outlining where each curve would be expected to be found if the null hypothesis were true. If the summary curve is outside its null band, it suggests that the null hypothesis is not true for the area of the curve that is outside its band, and so that there is a relationship between height and gender. In this example the null bands would be calculated by permuting the gender labels many times (but not permuting the height vectors). For each permutation the summary curves would be calculated and their positions stored. The location of each null band would be determined by the desired proportion of the permuted curve locations, decided by the level of significance desired. For instance, the null band could be set at the most extreme value attained by a permuted summary curve, or at some percent of the permuted summary curve distribution. Null band width and location depends on the sample size and variability in the data; a data set with high variability or a small number of subjects will be wider than those for a set with lower variability or more subjects.

Papers using Repeated Measures and Permutation Testing

Several recent papers have utilized permutation testing to estimate the significance in repeated measures data. These papers are from a variety of fields and use a variety of measures. This is not an exhaustive list of papers, but rather a selection of papers whose analytical method is somewhat similar to the method developed for the data in this thesis, as will be described below.

In a pair of papers, Ventura, Cai, and Kass (Ventura, Cai, & Kass, 2005a; Ventura, Cai, & Kass, 2005b) describe a randomization testing-based method to evaluate the amount of synchronization in neuron firing rates. They are interested in the frequency at which neurons fire over time, measured during multiple trials. This data generates a curve for each neuron and trial, the height of the curve representing the neuron's firing rate at that time. Each neuron does not behave identically on each trial, but rather varies in firing rate. The amount of synchronization between a pair of neurons at each time point is estimated by ξ_δ. The significance of the ξ_δ measured at each time point is determined using pointwise null bands; if the true ξ_δ is within the null bands then there is no evidence for synchronization between the neurons. Ventura, Cai et al. (2005b) placed the null bands at the 0.025 and 0.975 quantiles (95% probability boundaries) of the ξ_δ curves resulting from 1,000 bootstrap samples. The bootstrap samples were taken from simulated data generated by simulating the firing rates of individual neurons with the same stochastic properties as observed in the actual neurons. The significance level of the observed ξ_δ was determined by measuring the amount the true ξ_δ curve extended beyond the null bands, and the location of the excursion indicated when the synchronization occurs. The precise significance level was determined by calculating the proportion of bootstrap sample ξ_δ curves with an excursion as far outside the null bands as observed in the true ξ_δ curve. Ventura, Cai et al. (2005a) ended the paper with a discussion of the test's statistical properties.

A different analysis method, involving permuting the collected data, was used by Schweder, Jorde et al. (2005) to study the genetic characteristics of BCB Bowhead whales. They are interested in the genetic similarity of the whales varying in age and catch time (due to whale migration, different populations are sampled when whales are caught at the same location at different times). Their measure is a_{ij}, an estimate of the genetic similarity of a pair of whales. a_{ij} was measured for all whale pairs, producing graphs of a_{ij} by time (age or days between catch times). If there is no relationship between genetic similarity and time the a_{ij} curve should be flat; deviations suggest that whales of particular ages or catch times are more (or less) similar than others. Schweder, Jorde et al. (2005) evaluated the significance

of a_{ij} deviations by what they termed "simultaneous confidence null-bands," null bands constructed by permuting the data labels (whale age or catch time) and recalculating the a_{ij} on each permuted data set. They calculated these bands at the 50%, 95%, and 99% quantiles to indicate significance level of excursions of the true a_{ij} curve outside the corresponding bands (excursions indicate that the genetic similarity of the whale pairs of particular ages/catch times is higher (or lower) than expected if genetic similarity was not related to age/catch time).

Patterson, Garway-Heath et al. (2005) examined a permutation testing method for repeated times series data in clinical applications. The goal was to monitor the progression of glaucoma using confocal scanning laser tomography images of the optic disc. Image analysis produced a topographic height value at each pixel of each image; decreasing height over time indicates progressing glaucoma severity. At each pixel, the change over time is described by calculating a least-squares regression line through the topographical heights; the statistic they used to describe this line is the slope of the regression line divided by its standard error. *Statistic image maps* were then constructed to summarize the changes at all pixels over time: each pixel of the statistic image map corresponds to a pixel in the original image; its shading corresponds to the calculated statistic for that pixel. Areas of glaucoma progression appear as dark clusters in the statistic image maps. Significance of these clusters was determined by two sets of permutation testing on the images for each person. First, a permutation test was performed for the statistic calculated at each pixel by permuting the image order (permuting each image's time label). Pixels with statistical significance at the 0.05 level or higher were passed to the second level of permutation testing. The statistic image map for each person at this level of testing contains only pixels found to be significant during the first round of testing (similar to thresholding the original image). Clusters of these thresholded pixels are tested for significance by permuting the pixels in each statistic image map, testing for cluster size at each permutation. Significantly large clusters are then flagged as areas of glaucoma progression. Patterson, Garway-Heath et al. (2005) tested this method on simulated and actual data, and compared it to the standard method for evaluating these images; they found that it was more accurate and had fewer false positives than the standard method.

Permutation Testing for Psychophysiological Data

This section discusses a permutation testing method used to evaluate the statistical significance of patterns seen in curves of physiological activity. This data consists of repeated physiological measurements; measurements of physiological activity at fixed time

points after an experimental stimulus. In some cases each subject was measured on one stimulus only, resulting in a single vector of data for each subject, each entry of which is the subject's physiological activity at that time. With this data parametric statistical methods, such as MANOVA, may be used. But in other experiments each subject was exposed to multiple stimuli of multiple types. This data is repeated on several layers: each subject's responses to an individual stimulus can be represented as a vector of values as before, but since each subject was exposed to multiple stimuli, the data for each subject is a collection of vectors, one vector for each stimulus. Each subject tends to have a unique pattern and level of physiological response, so it is expected that the responses be correlated within each vector, but also that all vectors for each subject be correlated. It is not possible to accurately model this multiply-layered repeated measures data structure (given the sample sizes and distributions) with standard parametric statistical testing. Instead of attempting to reduce the data to a form where parametric testing would be possible, a graphical permutation testing method was developed and used to evaluate significance.

This method consists of calculating null bands, which indicate where the summary curve is expected to lie if the null hypothesis is true; the null hypothesis is that there is no difference in response between groups. The summary curve is constructed using a smooth of the raw data. Since a large amount of raw data was available it was not necessary to perform a bootstrap analysis using simulated data; instead, many permutations of the actual data were created, and the summary curve saved for each permutation. Null bands were set at the 95% quantiles of the permuted summary curve locations.

This method is related to the reviewed permutation testing methods for repeated measures data, but distinct. In particular, this method is focused on evaluating differences in curves when the curves summarize physiological changes over time. This method is most similar to that described by Ventura, Cai, and Kass (Ventura, Cai, & Kass, 2005a; Ventura, Cai, & Kass, 2005b), in that pointwise null bands were calculated to evaluate significance of curves constructed from time series data. This method varies from that of Ventura, Cai, and Kass, however, in that the null bands were constructed using permutation testing, rather than from bootstrap samples, and the lowess, rather than ξ_δ, was used to describe the raw data. The application of the method is also distinct; as far as I am aware, this is the first use of permutation testing methods to evaluate curves resulting from physiological data.

Smoothing: Summary Curves

A lowess curve was used as the summary curve, the curve representing the pattern of physiological responses for all subjects in the particular group. Lowess (also called loess)

curves were chosen (as opposed to a measure such as the mean) because the lowess function generally creates a curve that is less sensitive to outliers and more appropriate for data sets with unequal variances at each time point (Diggle, 2002), a common (and sometimes dramatic) characteristic of this data. The lowess function calculates a locally-weighted least-squares line through the data, resulting in one value for each time point. The lowess curves were calculated using the R (R Development Core Team, 2005) lowess function (Cleveland, 1981), as implemented in the gplots package. This procedure is consistent with other descriptions of permutation testing for repeated measures data; smoothing of this type was mentioned for use with permutation testing and null bands in (Buja & Rolke, 2005).

Permuting

In all permutations the vector of measured physiological values for each person and stimulus was treated as an indivisible unit. In other words, suppose that the measured heart rate at ten time points is subject 1's response to stimulus a. These ten heart rates will always be together, and in the same order, although they may be recoded to seem to have resulted from a different subject or to a different stimulus during the permutations. These vectors are kept together to isolate the effect of interest: differences in response profiles between groups or stimuli. Likewise, in experiments where each subject viewed multiple stimuli of various types, and permutation testing is being done to evaluate significance of differences in summary curves for each stimuli type, the stimuli type labels are permuted within each subject individually (each subject will always have the same number of stimuli of each type, but the labels of the response profile vectors are permuted). Permuting responses within individuals avoids confounding variation due to differences between subjects with variation in subjects' responses to stimuli of the various types. Each permuted data set was created by randomly reassigning the label of interest (e.g. word category or recall accuracy) within each subject then calculating the summary lowess curves in the same manner as for the true data.

The total number of permutations possible in these data varies with experiment and hypothesis, but in all cases it is too large (on the order of millions) to calculate every permutation. Instead, a random subset of all possible permutations was used to determine significance. Statistical power is reduced when calculations are based on a random subset, rather than all possible permutations, but sufficient power generally remains when at least a thousand random permutations are selected; increasing the number of permutations increases the power (Edgington, 1995). In the analyses performed for chapter 4 1,500

random permutations were calculated, while 2,000 random permutations were calculated for the analyses in chapters 5 and 6.

Null Bands

The null bands for each curve were set at the pointwise 95% and 5% quantile of the summary curves calculated from the random data permutations. Given that the data is taken from psychophysiological experiments, and that the analysis is exploratory in nature, it was felt that 95% and 5% quantile curves would be an appropriate significance level. The process is illustrated in Figure 2.1. First, summary (lowess) curves are calculated to show the trends of interest in the data (pane a). Then the permutations are performed; each permutation consists of randomly relabeling the data and recalculating the summary curves. Lowess curves for two permutations are shown in panes b and c of Figure 2.1. After the necessary number of permutations has been completed the location of the null bands is determined by calculating the 95% and 5% quantile of all permutation lowess curves at each point (pane d).

Discussion

Additional details of this permutation testing method, as applied to the data in this thesis, are contained in the relevant chapters. In no cases were exact p-values calculated to describe the permutation testing results. This might be possible by using the proportion of permuted curves with the same number of points outside the null bands as the actual data
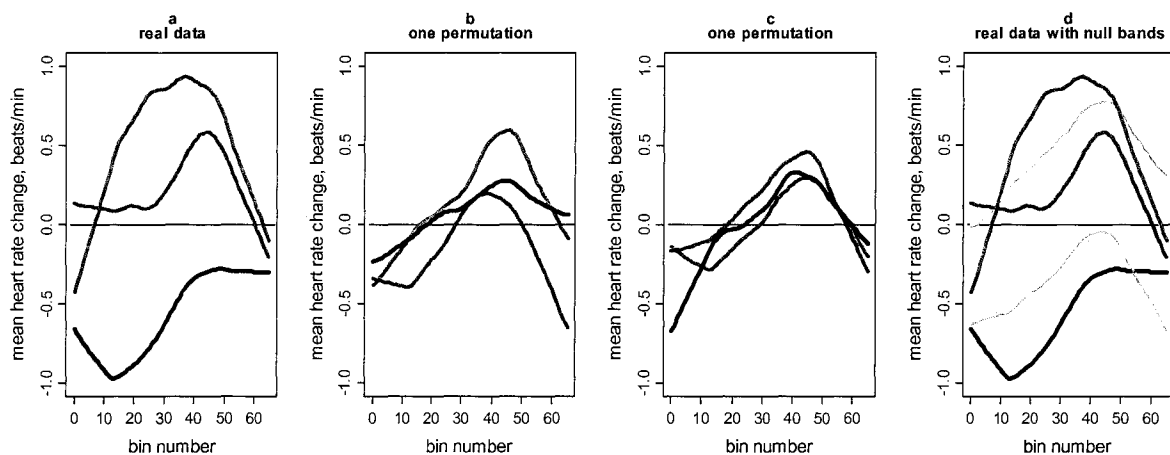
Figure 2.1. Illustration of permutation testing methodology. Pane a: lowess curves calculated on the real data. Panes b and c: lowess curves for two permutated data sets. Pane d: lowess curves for real data with null bands.

curve. Determining an actual p-value is complex in this case because of the large number of comparisons: the null bands were placed using the pointwise quantiles, not an overall measure. It isn't clear how null bands based on the overall significance level could be placed; a two-step process such as used in Patterson, Garway-Heath et al. (2005) may be helpful: the first step would be the same as the analysis done here. The additional second step would consist of additional permutations evaluating how often a summary curve has an area of excursion beyond the null bands as large as seen in the actual data.

In this case, instead of exact p-values the claim was made that, at the level of an exploratory study, these results were unlikely to have occurred if in the absence of actual differences between the groups. This claim is not as strong as would be possible if a p-value is calculated, but valuable in exploratory studies nonetheless, as demonstrated in later chapters, by providing evidence concerning whether curves are different or not.

CHAPTER 3: SIGNAL PROCESSING AND PUKA

Abstract

A common method of recording respiration in psychophysiology respiration is a single strain-gauge belt wrapped around the chest. A standard analytical method for measuring total breath length and its components from these signals is lacking, however. This chapter describes an algorithm which accurately identifies the breaths in these signals. The algorithm is implemented in puka, an open-source software program which also provides tools for time-domain EKG signal analysis through an interface to the PhysioToolkit.

Introduction

Researchers in multiple fields are interested in analyzing human respiration. For example, psychophysiologists study changes in respiration related to psychological processes and in respiratory sinus arrhythmia calculations (e.g. Palomba, Sarlo, Angrilli, Mini, & Stegagno, 2000; Sarlo, Palomba, Angrilli, & Stegagno, 2002), while physiologists examine cardiorespiratory reactions to changes in factors such as stress, hypertension, exercise, and posture (e.g. Buchholz, Schachinger, Wagner, Sharma, & Deter, 2003; Goodman, Martin, & Williams, 2002). Accurate techniques for measuring mechanical lung function (such as airway resistance and maximum airflow) are available and are used for clinical research and assessment of conditions such as asthma and chronic obstructive pulmonary disease, but these techniques are not suitable for all types of research because they require intrusive apparatus (such as a mouthpiece or nose clip) which interfere with natural breathing (Ritz, et al., 2002). A strain-gauge transducer attached to a belt wrapped around the chest is often used to measure respiration in psychophysiological research: it is easy to use, minimally intrusive, and widely commercially available (e.g. ADInstruments, accessed 21 July 2005; Bio-logic Systems Corp., accessed 21 July 2005; BIOPAC Systems Inc., accessed 21 July 2005; Grass-Telefactor, accessed 21 July 2005). Despite this, there is not a standard technique for measuring the components of respiration signals derived from a strain-gauge transducer. This chapter describes an algorithm, implemented in a program called puka, to reliably detect the breaths in strain-gauge belt recordings.

In contrast to the paucity of tools for respiration analysis, many excellent algorithms and programs exist for electrocardiogram (EKG) waveform detection. Heart rate variability (HRV) analysis is based on heart beat timing; the R wave (see pane b of Figure 1.1) is used

to locate each beat. HRV analysis accuracy depends on precisely locating the time of every normal beat (R wave). In addition to the implemented respiration analysis algorithm, puka performs EKG R wave detection and time-domain HRV analyses, including peak-valley RSA, by serving as a graphical interface to several PhysioToolkit analysis programs (Goldberger, et al., 2000).

Signal Analysis

Respiration Analysis Algorithm

This algorithm is designed to identify the critical parts of each breath (peak, trough, start and stop of the post-inspiratory pause, start and stop of the post-expiratory pause; as labeled in Figure 1.2, pane b) in a respiration signal recorded by a strain-gauge transducer. Once these critical points have been identified, statistics, such as mean respiration rate, can be calculated. The algorithm consists of three distinct steps. First, the peaks and troughs are identified. Second, the pause around each peak and trough is marked. Finally, the peak and trough markers are adjusted to the center of their pauses.

Step 1: Identify the Peaks and Troughs

The first step is to identify the approximate location of each breath using a peak-detection algorithm to mark the extreme points of the signal: the points of maximum expiration (troughs) and inspiration (peaks). The method described by Todd and Andrews (1999) works well on the respiration signals tested and was adapted for use in puka. Briefly, the algorithm first calculates a threshold value, then steps through the signal, testing the direction and magnitude at each point. The height of the signal at each point is compared to the height of the previously marked peak or trough, and a new peak or trough is marked at the extreme point if the height difference is greater than the threshold value. As implemented, the threshold is initialized to 0.1 times the difference between the 75th and 25th percentile of the signal's height; the percentage height in the threshold calculation can be adjusted so that more or fewer peaks are found. The algorithm alternates between identification of peaks and troughs, ensuring that each trough is followed by a peak and each peak by a trough. All breaths must be identified at this step; the implementation allows the user to verify the peak-detection algorithm's performance and to indicate if certain peaks/troughs should be omitted from analysis.

Step 2: Mark the Pauses around each Peak and Trough

The approximate location of each breath is determined from the peak and trough markers placed in Step 1. These markers are used as the starting point from which to examine the signal for a possible pause at that peak or trough. Since the respiration signal's shape can change quickly, the beginning and end of each pause is calculated independently. As implemented, all signals are resampled to 200 Hz for pause calculation; the values are returned at the original sampling frequency. For clarity, this section refers to the identification of the beginning of a post-inspiratory pause; the procedure for identifying the end of the post-inspiratory pause and post-expiratory pauses is analogous. In brief, the algorithm is initialized at a point (p) in the inspiration preceding the peak beyond the pause (Figure 3.1, pane a). p is then moved back towards the peak one point at a time until a stopping condition is met, which suggests that inspiration has ceased and the post-inspiratory pause has begun (Figure 3.1, panes b-f).

The stopping conditions are: the height of the curve at p is equal to or greater than the height at the peak marker (Figure 3.1, pane b); the signal's slope has moved in the incorrect direction (negative during inspiration, positive during expiration) for enough consecutive intervals and p is near the peak (pane c); the signal's slope has changed only a small amount for enough consecutive intervals and p is near the peak (pane d); p has reached the peak (pane e); and most of the remaining intervals have very small height differences (pane f).

The stopping rules require the use of three threshold values (α, β, and γ), which are calculated separately for every inspiration and expiration. The magnitude of different breaths in a strain gauge respiration signal often vary greatly, so the use of a single set of thresholds for the entire recording would result in too-large thresholds for low-magnitude breaths and too-small thresholds for high-magnitude breaths. The thresholds are defined as proportions of the height of the inspiration or expiration for which the pause is being calculated. Thresholds are used to initialize p (α), define "near" the peak's height (β), and define a "small" change in signal height (γ). Values for the thresholds and parameters were determined through trial and error. The stopping rules and thresholds are given in Listing 3.1.

Step 3: Center Peak and Trough Points

Finally, each peak and trough is moved to the center of its surrounding pause. This maximizes accuracy of later calculations, since the locations identified as peaks and troughs

in Step 1 are somewhat arbitrary. Centering the peaks and troughs ensures that the length of each breath will not include an unpredictable portion of each surrounding pause.

EKG Waveform Detection

Very accurate programs for EKG waveform detection are available; puka uses programs from the PhysioToolkit (Goldberger, et al., 2000 http://www.physionet.org/physiotools/), a set of programs implementing well-validated algorithms to identify and derive measurements from EKG waveforms. PhysioToolkit
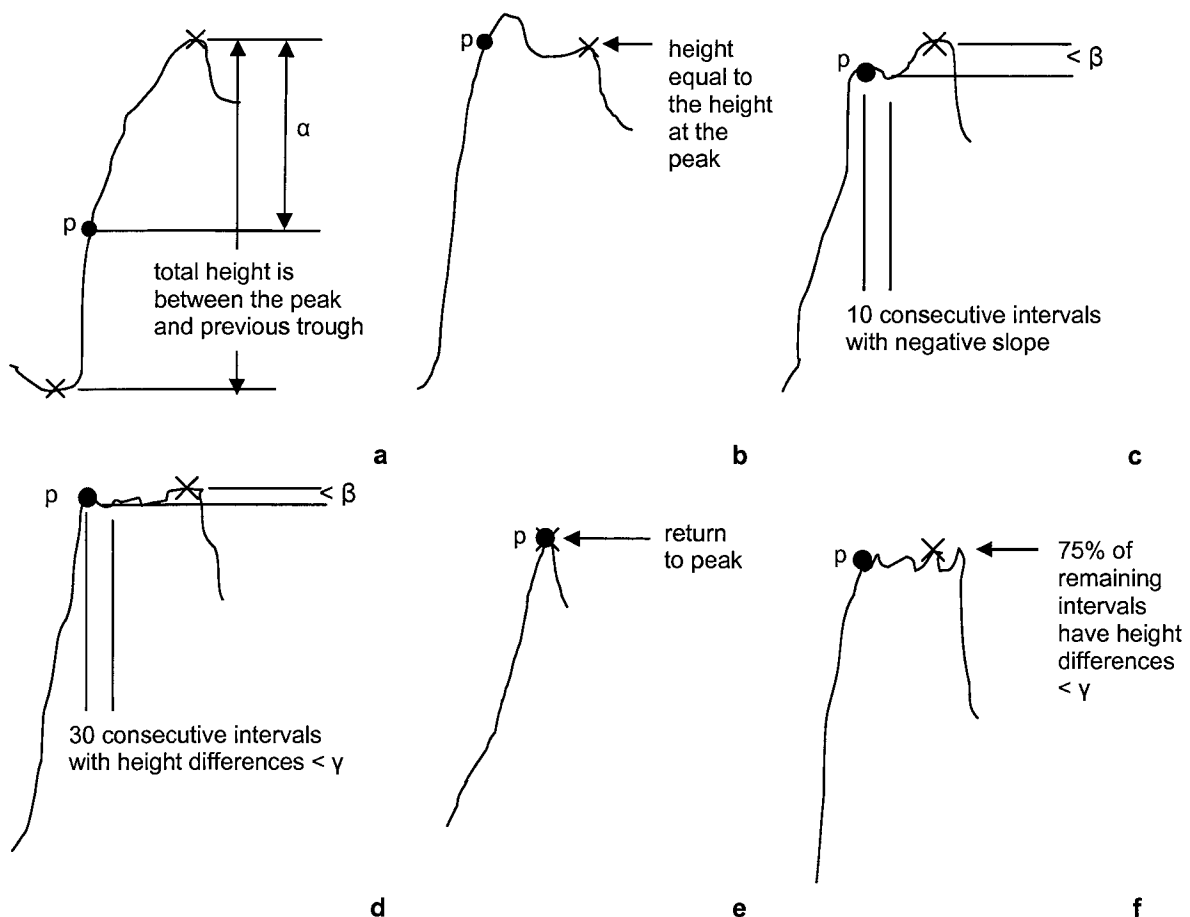
Figure 3.1. Illustration of the pause-detection procedure. In all panes the peak identified in Step 1 is marked by an x and the point (p) showing the beginning of the pause before the peak is a solid dot. Pane a: the first step in the algorithm, at which the point is initialized as the sample on the inspiration α below the peak's height. Pane b: pause end set where the point's height is equal to the peak's height. Pane c: pause end set to the first of the 10 intervals with negative slope. Pane d: pause end set at the center of a stretch of 30 small-slope intervals. Pane e: pause end reaches peak marker; no pause found. Pane f: pause end set at the sample where 75% of the intervals between p and the peak have height differences < γ.
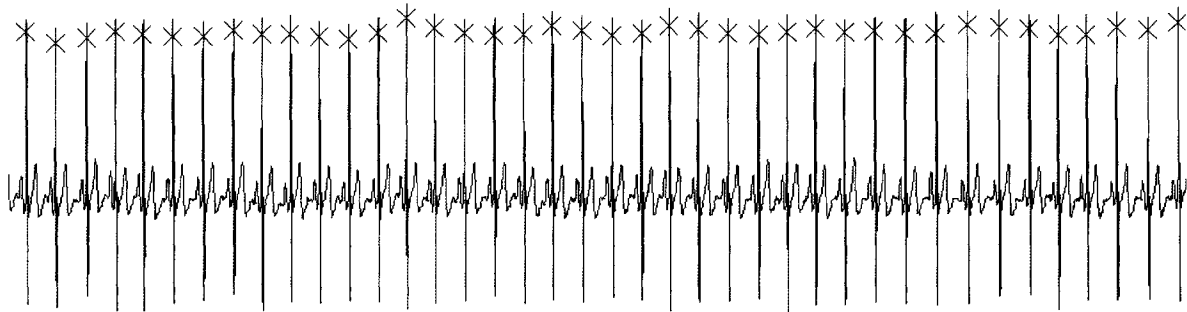
Figure 3.2. EKG signal plotted in puka, with detected R waves marked.

programs have been used in recent research (e.g. Bickel & Lai, 2001; Gerstenfeld, et al., 2003; Peng, et al., 1999) and are accepted as a standard, accurate tool for EKG analysis (Moody, Mark, & Goldberger, 2001). Puka uses the PhysioToolkit program *ecgpuwave* to annotate the components of the EKG waveforms when possible (*ecgpuwave* requires a sampling frequency above 500 Hz; if the sampling frequency is below this the PhysioToolkit *sqrs* program is used instead). *ecgpuwave* uses the Pan-Tompkins algorithm for QRS detection (Pan & Tompkins, 1985), and has been validated against annotated EKG recordings (Jane, Blasi, Garcia, & Laguna, 1997). The time of each normal R wave is extracted from the annotations by the PhysioToolkit *rdann* program. The output from *rdann* is read by puka and sent to MATLAB for plotting (such as shown in Figure 3.2) and statistical computations.

Implementation

As discussed, the respiration analysis algorithm and EKG waveform detection processes were implemented in puka, an open-source program available for download at PhysioNet (http://www.physionet.org/physiotools/puka/). Puka is written in Java and uses MATLAB (MathWorks, 2003) for signal processing, graphing, and statistical calculations. It also provides a graphical interface for several PhysioToolkit analysis programs (*ecgpuwave*, *sqrs*, *rdann*) for EKG R wave detection (Goldberger, et al., 2000). Each step of the algorithm is shown to the user for accuracy; respiration signals can be very irregular so manual intervention is often necessary.

HRV Figures and Calculations

Puka calculates several time-domain HRV measures using the results of the EKG waveform detection; all EKG signal processing is performed by the PhysioToolkit programs. The HRV values calculated by puka are displayed to the user to aid in confirming the

analysis validity. Partial beats (those that overlap the start or end of a clip) are properly weighted, so the calculated values are accurate even for short recordings. HRV measures can also be calculated in statistical packages using the R peak times calculated by puka.

Respiration Summary Measures

After the peaks, troughs, and pauses in the respiration signal have been identified statistics to describe the signal as a whole can be calculated. By convention, respiratory periods are defined trough-to-trough (an inspiration and the following expiration), rather than peak-to-peak (an expiration and the following inspiration) (Grossman, Karemaker, & Wieling, 1991). The mean and standard deviation of the breath length is therefore calculated in puka as the mean and standard deviation of the time between successive marked troughs.

The calculation of the mean and standard deviation of inspiratory and expiratory time can be performed in several different ways. If pauses are ignored, inspiratory time is the time between each trough and its following peak, while expiratory time the time between each peak and its following trough. If pauses are included in the calculations puka uses the time spent in active inspiration (end of post-expiratory pause to start of post-inspiratory pause) in the inspiratory time calculation and active expiration (end of post-inspiratory pause to start of post-expiratory pause) in the expiratory time calculation. This definition has been used in previous research describing the timing of the respiratory cycle (e.g. Boiten, 1993), and allows greater flexibility in later analyses. If desired, puka calculates the mean and standard deviation of the post-expiratory and/or post-inspiratory pause length. Puka also calculates the inspiratory duty cycle, the mean inspiration length divided by the mean breath length. This statistic, which can vary between zero (no inspirations) and one (no expirations), gives the proportion of respiration cycle spent in inspiration. As with the HRV measures, additional respiration statistics can be calculated from saved peak, trough, and pause times.

Peak-Valley RSA Calculation

The algorithm by which the peak-valley RSA is calculated in puka is consistent with that described in the literature (e.g. Grossman, van Beek, & Wientjes, 1990). Specifically, the RSA score for each breath is the difference, in msec, between the longest and shortest inter-beat (RR) interval occurring within the breath (first RR interval starts at the first beat after onset of inspiration). If the shortest RR interval does not occur before the longest within each breath, the breath is assigned a score of zero. A breath is defined trough-to-trough, with the first RR interval in the breath starting at the first R wave after the start of the breath,

and the last RR interval ending with the first R wave after the end of the breath. A RSA score is computed in this way for each breath in the clip; the mean for all breaths in a time period is the RSA measure.

Implementation Testing

HRV and RSA

The accuracy of puka's HRV calculations was validated using artificial EKG signals, paced breathing data, and hand calculations. Artificial EKG signals (at 60, 70, and 80 beats per minute) were generated using *ECGwaveGen* (Harriott, 2005). Puka correctly calculated the mean and standard deviation of the heart rate, as well as the minimum, maximum, and RR interval statistics of these signals. Also, statistical calculations on several actual signals were verified using a spreadsheet and calculator.

Respiration: Paced Breathing Study

A paced breathing study, in which subjects coordinated their respiration with a visual signal, was performed to verify the respiration analysis algorithm using actual data. The study included six patterns selected to span normal adult resting respiration rates of paced breathing, described in Table 3.1. Each trial of the paced breathing study consisted of one respiration pattern and lasted approximately two minutes. A computer program displayed "breathe in," "breathe out," or "hold breath" messages on a monitor located directly in front of the subject, and subjects were instructed to follow the cues as closely as possible. Each trial was separated by a resting period of at least thirty seconds in length, during which respiration was not controlled.

Table 3.1. Paced breathing patterns included in the validation experiment. All times are in seconds. The names reflect either the respiration rate or pause type; PE is the trial with post-expiratory pauses and PI is the trial with post-inspiratory pauses.

trial name	breaths/ minute	total breath length	inspiration length	expiration length	post-inspiratory pause length	post-expiratory pause length
8	8.0	7.5	3.75	3.75	0.0	0.0
10.5	10.5	5.7	2.85	2.85	0.0	0.0
13	13.0	4.6	2.30	2.30	0.0	0.0
18	18.0	3.3	1.65	1.65	0.0	0.0
PE	6.9	8.7	2.90	2.90	0.0	2.9
PI	8.7	6.9	2.30	2.30	2.3	0.0

Data were collected using a single strain gauge respiration belt (TSD201 Respiratory Effort Transducer manufactured by BIOPAC Systems, Inc., Santa Barbara, CA). The transducer consists of an elastic belt attached to a strain gauge, which generates an electric signal proportional to the amount of tension on the belt, which is caused by changes in chest circumference. In brief, the belt was snugly wrapped around the chest at the approximate height of the sternum. The subject was instructed to take several deep breaths while the respiration signal was observed on the monitor. The belt was adjusted, if necessary, to ensure that the signal rose with inspiration and fell with expiration without exceeding the range of the recorder.

Nine subjects, three female and six male, participated in the paced breathing task. All subjects completed the 8, 10.5, 13, and 18 trials; three male subjects completed at least one trial twice. If any trials were repeated the second trial was performed on a different day than the first. No subject ended a trial early, but several subjects were not able to complete all trials due to time constraints. Each subject was observed to insure that they closely matched their respirations to the timed instructions. All data were analyzed using the algorithms and methods described in this chapter, as implemented in puka.

Validation Results

The calculated mean respiration rate and mean total breath length for each trial closely matched the actual lengths for each trial, showing that the implemented algorithm was able to accurately detect each breath and calculate the summary statistics. The distribution of mean breath length for each subject on each trial is shown in the left pane of Figure 3.3, while the respiration rate is in the right pane. In each trial the calculated respiration rates are tightly grouped around the target value.

The calculated mean inspiration and expiration lengths were shorter than expected, except in the PE and PI trials. As can be seen in Figure 3.4 and Figure 3.5, the difference between the expected (solid dot) and actual mean is directly related to the rate of respiration: at faster respiration rates the difference between the actual and expected length is small, while at slower respiration rates the difference is large. The recorded inspiration and expiration times were shorter than expected on the 8, 10.5, 13, and 18 trials due to the detection of short post-inspiratory and post-expiratory pauses. The mean observed length was longer than expected on two trials: the inspiration time was longer than expected on the PE trial, and the expiration time was longer than expected on the PI trial. This is most likely due to a portion of the pause being assigned to the following breath part.

The detection of pauses when none were cued (no pauses were cued on the 8, 10.5, 13, or 18 trials) is clearly seen in Figure 3.5 and Table 3.2. By comparing the left pane of Figure 3.5 (post-inspiratory pause length) with the right (post-expiratory pause length), it can be seen that the range of pause lengths decreases as the respiration rate increases. Also, it is apparent that the post-expiratory pause length for the PE trial was more variable than the pause lengths for any other trial. This is due to a combination of subjects' difficulty in following the cues and misidentification of pauses.

To determine whether the discrepancy between the expected and calculated pause length was due to signal characteristics or a misinterpretation of the signal by the analysis program, the raw respiration signals, with all detected peaks, troughs, and pauses marked, were visually examined. It was apparent that while all subjects closely followed the cued signal, some subjects were more adept than others at precisely controlling their respiration. Many subjects exhibited a moderate post-inspiratory pause, suggesting that they inhaled maximally, and then held their breath until the cue to exhale appeared. Only a few subjects were able to match the cued signal precisely. For example, the 10.5 breaths/minute trial tracings of four subjects appear in Figure 3.6, in which it can be seen that only subject *d* followed the cues exactly; subjects *a*, *b*, and *c* exhibit post-inspiratory pauses.

Pause Detection

The pause-detection algorithm implemented in this program was able to detect the post-inspiratory and post-expiratory pauses with a reasonable degree of accuracy, although not perfectly. The longest post-inspiratory pauses were detected in the PI trial, while the longest post-expiratory pauses were detected in the PE trial (Figure 3.5). The detected length of the post-inspiratory pauses on the PI trial was close to the cued length, while the detected post-expiratory pause length on the PE trial varied substantially. The reason for this variation can be seen when the raw respiration tracings are examined with the detected peaks, troughs, and pauses marked (Figure 3.8). The effort of each subject to hold their breath after respiration is evident, but subjects varied in their ability to do this, and the signal is contaminated by heart-beat detection. The algorithm did a fairly good job at accurately marking the extent of the post-expiratory pauses for subject b, whose pauses were generally of a constant magnitude and with only moderate noise. The algorithm was less accurate at marking the pauses for subject c and d, however. In the case of subject c, the pause extents are not sharply defined on the raw signal, because the subject inhaled slightly at the beginning of most pauses. The tracing of subject d exhibits an irregular signal magnitude in

the pauses, and the algorithm mistakenly marked the point between the end of the pause and beginning of inspiration too near the trough.

Table 3.2. Mean calculated values over paced breathing study.

	8 bpm	10.5 bpm	13 bpm	18 bpm	PE	PI
total breath length, mean	7.40	5.60	4.60	3.30	8.60	6.90
total breath length, standard deviation	0.62	0.53	0.49	0.29	0.86	0.34
inspiratory time, mean	2.90	2.30	2.00	1.60	3.70	2.30
inspiratory time, standard deviation	0.57	0.40	0.30	0.19	0.84	0.34
expiratory time, mean	3.10	2.60	2.20	1.70	2.70	2.50
expiratory time, standard deviation	0.44	0.40	0.28	0.18	0.35	0.53
post-inspiratory pause length, mean	0.98	0.39	0.16	0.04	0.51	2.10
post-inspiratory pause length, standard deviation	0.54	0.34	0.19	0.09	0.32	0.55
post-expiratory pause length, mean	0.44	0.39	0.25	0.10	1.60	0.15
post-expiratory pause length, standard deviation	0.40	0.33	0.30	0.17	0.85	0.23
inspiratory duty time	0.39	0.40	0.44	0.46	0.43	0.32
respiration rate, mean	8.10	10.60	13.00	18.00	7.00	8.60
respiration rate, standard deviation	0.62	0.53	0.49	0.29	0.86	0.34
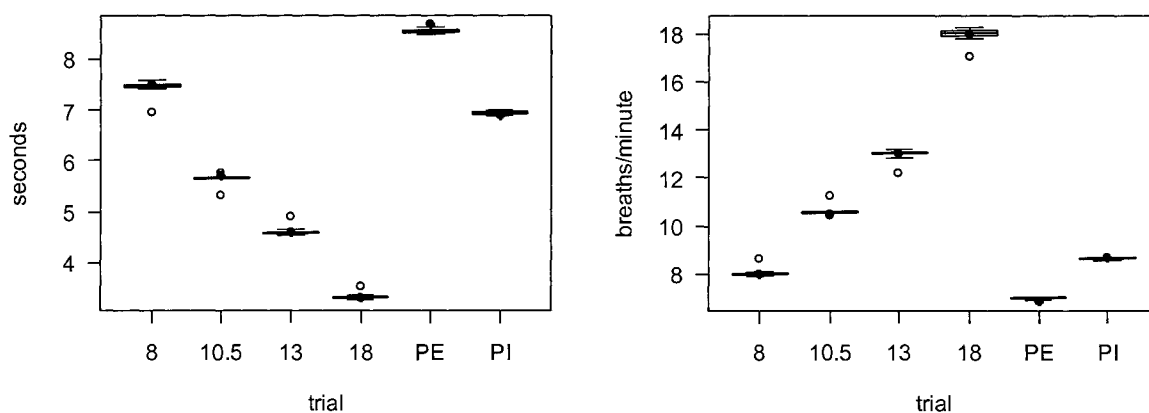
Figure 3.3. Mean total breath length in seconds (left) and mean respiration rate (right) as calculated for each trial. Solid dots indicate the cued value for each variable. Each boxplot indicates the spread of the data: median plotted as the center of the box, upper and lower quartile as the ends of the box, whiskers at the most extreme data point which is no more than 1.5 times the spread (box length) beyond the quartile, and open circles for outliers (data points beyond the whiskers).
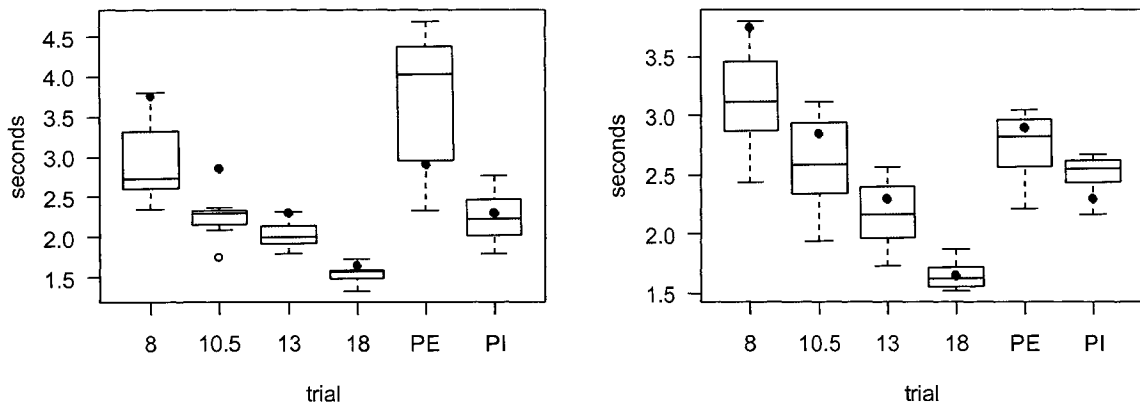
Figure 3.4. Mean length of inspiration (left) and expiration (right) as calculated for each trial. Solid dots indicate the expected (cued) value for each variable.
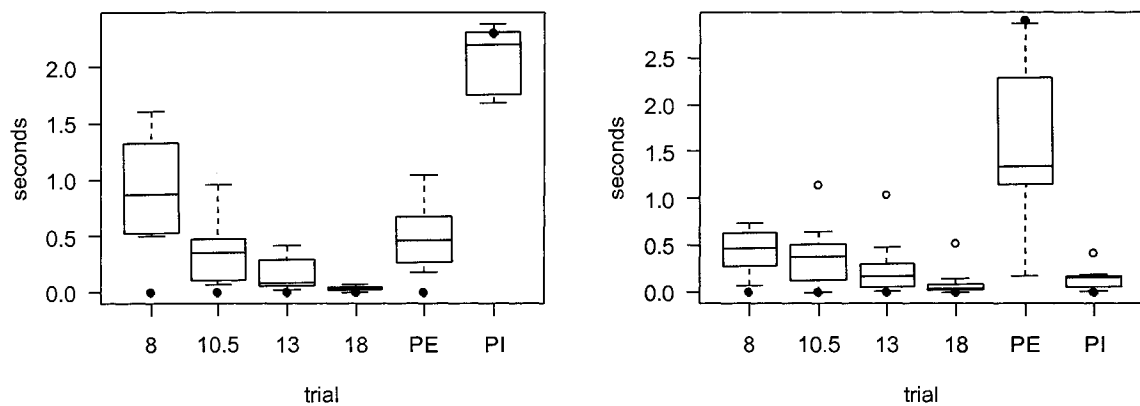
Figure 3.5. Mean post-inspiratory pause length (left) and mean post-expiratory pause length (right) as calculated for each trial. Solid dots indicate the cued value for each variable.
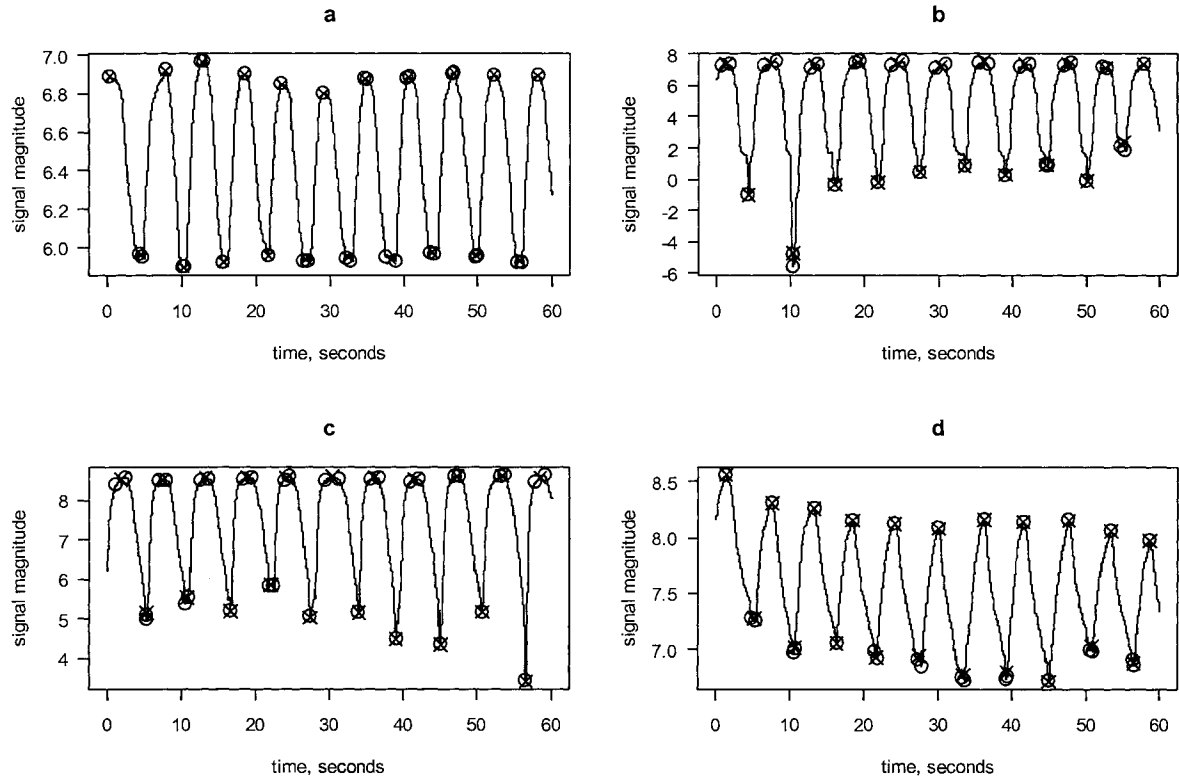
Figure 3.6. Four respiration tracings from the validation experiment, with identified peaks and troughs (x) and pauses (o) marked. All subjects were breathing at 10.5 breaths/minute. Subjects a, b, and c exhibit slight post-inspiratory pauses, while subject d did not.
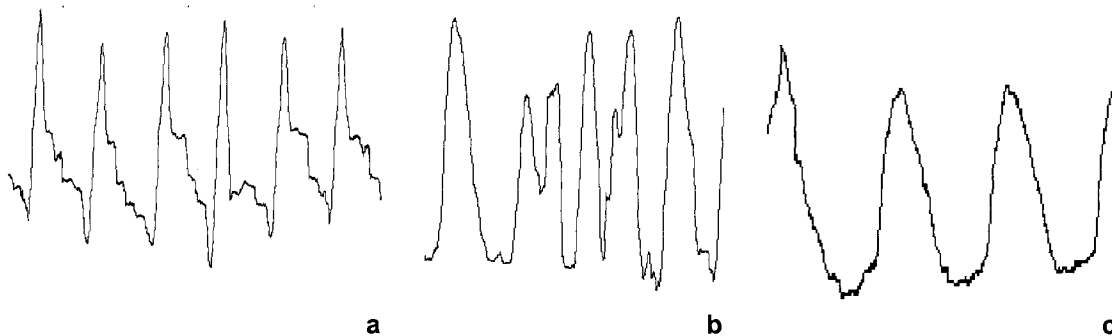
Figure 3.7. Sample ambiguous respiration signals. Irregular breaks in the exhalations (a), short exhalations during and after inspiration (b, second and fourth breaths), and gradual transitions between expiration and inspiration (c).
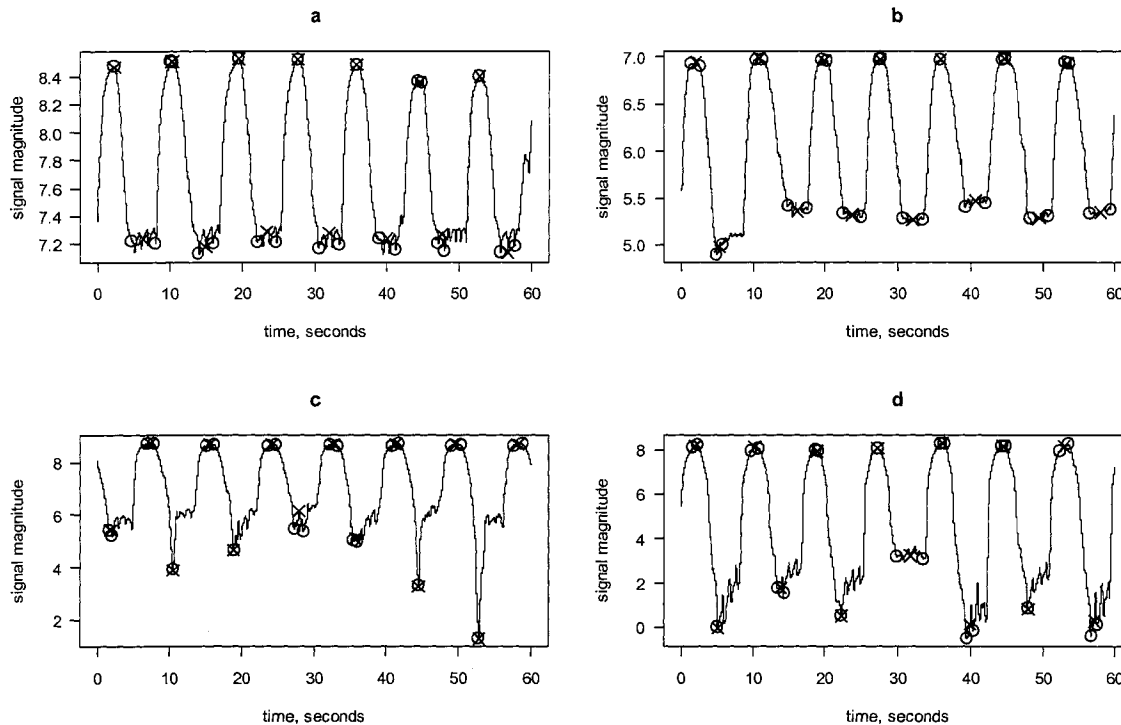
Figure 3.8. Respiration tracings with marked peaks and troughs, PE trial. Each tracing contains at least one post-expiratory pause for which the algorithm was not able to exactly mark the ends of the pause, primarily due to an increase in the signal magnitude during the pause, combined with heart-beat detection artifacts. Subjects c and d inspired slightly during most pauses, making the true extent of the pause ambiguous.

Discussion

The algorithm and program described in this paper accurately determines the location of breaths (extents of inspiration, expiration, and pauses) in respiration signals recorded using a single strain-gauge chest belt. The detection of pauses with this algorithm may have errors when the transition from inhalation to exhalation (or exhalation to inhalation) is smooth (resulting in a rounded cone shape) or the signal magnitude shifts during a pause, as shown in Figure 3.7. Part of the problem in analyzing these types of signals is that the literature lacks a clear definition of where the inspiration and expiration points in these cases are located. As implemented, the automatically detected pause points can be manually adjusted if needed, but this must be done with care, ensuring that consistent, reliable criteria are used.

This algorithm and program will be useful to other researchers analyzing respiration signals recorded from a single chest strain gauge belt. It is hoped that this work will spur increased interest in performing more detailed analyses of strain gauge respiration recordings. More research can be done to improve the algorithm's identification of pauses, particularly on signals that have shifting baselines during pauses. A greater consistency and precision in respiration signal definitions will also be helpful. Use of well-defined metrics will allow for increased consistency in the literature and facilitate comparisons between studies. Also, the ability to accurately locate the parts of each breath allows a more detailed analysis of the respiration signal than was previously possible.

At this peak marker:
- Calculate the parameter values for this inspiration:
  - t: absolute value of the difference between signal height at this peak and the signal height of the previous trough.
  - α: $t * 0.5$
  - β: $t * 0.08$
  - γ: $t * 0.0005$
- Initialize p to the sample (in the inspiration preceding this peak marker) closest to this peak marker such that the [absolute value of the difference between signal height at this p and at the peak marker] $> \alpha$

At each new p calculate:
- l: if [absolute value of the difference between signal height at p and at the previous p] $< \gamma$ then increment l else $l = 0$
- n: if [difference between signal height at p and at the previous p] < 0 then increment n else $n = 0$

Test these stopping conditions at each p:
- p at peak marker
- signal height at p same as height at peak marker
- $l > 30$ and either
  - difference between signal height at the peak marker and at $p < \beta$
  - or 50% of the intervals between p and the peak marker have height differences $< \gamma$
- $n > 10$ and either
  - difference between signal height at the peak marker and at $p < \beta$
  - or 50% of the intervals between p and the peak marker have height differences $< \gamma$
- 75% of the intervals between p and the peak marker have height differences $< \gamma$

If one of the stopping conditions is met, stop iterating and set the pause end to p. If none of the stopping conditions were met move p one sample closer to the peak marker and repeat.

Comments:
- The order in which the signal heights is subtracted to calculate n is set based on which pause is being determined. n is the number of adjacent intervals in which the slope is moving in the incorrect direction (for the pause before a peak the signal should be rising, so the height at the previous p is subtracted from the height at this p).
- If the $l > 30$ stopping condition is met, p needs to be moved 15 samples back from the peak marker (to the center of the stretch of 30 intervals with small slope).
- If the $n > 10$ stopping condition is met, p needs to be moved 10 samples back from the peak marker (to the first sample in the stretch of 10 intervals with incorrect slope).
- For some respiration signals including the final stopping condition (75% of the sampling intervals between p and the peak marker have height differences $< \gamma$) makes pauses too large. In *puka* there is an option to exclude this stopping condition when needed.

Listing 3.1. Description of the procedure to identify the beginning of a post-inspiratory pause. The procedure for identifying pauses around each trough is analogous. The signal has been resampled to 200 Hz before beginning the procedure.

CHAPTER 4: CARDIOVASCULAR AND RESPIRATORY RESPONSES DURING MUSICAL MOOD INDUCTION

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Abstract

Music is used to induce moods in experimental settings as well as for therapeutic purposes. Prior studies suggest that subjects listening to certain types of music experience strong moods and show physiological responses associated with the induced emotions. We hypothesized that cardiovascular and respiratory patterns could discriminate moods induced via music. 18 healthy subjects listened to twelve music clips, four each to induce happiness, sadness, and fear, while cardiovascular and respiratory responses were recorded using an electrocardiogram and chest strain-gauge belt. After each clip subjects completed a questionnaire. Subjects consistently reported experiencing the targeted mood, suggesting successful mood induction. Cardiovascular activity was measured by calculating time domain measures and heart rate changes during each clip. Respiratory activity was measured by total, inspiration, and expiration lengths as well as changes in mean respiration rate during each clip. Evaluation of individuals' patterns and mixed-model analyses were performed. Contrary to expectations, the time domain measures of subjects' cardiovascular responses did not vary significantly between the induced moods, although a heart rate deceleration was found during the sadness inductions and acceleration during the fear inductions. The time domain respiratory measures varied with clip type: the mean breath length was longest for the sad induction, intermediate during fear, and shortest during the happiness induction. However, analysis using normalized least mean squares adaptive filters to measure time correlation indicated that much of this difference may be attributable to entrainment of respiration to characteristics of the music which varied between the stimuli. Our findings point to the difficulty in detecting psychophysiological correlates of mood induction, and further suggest that part of this difficulty may arise from failure to differentiate it from tempo-related contributions when music is used as the inducer.

Introduction

A large literature, in both healthy and psychiatric individuals, has investigated the psychological, biological, and neural correlates of mood. Experiments in this literature have explored the effects of mood on overall health, immune system function, memory, attention, and perception (Cacioppo, Berntson, Larsen, Poehlmann, & Ito, 2000). However, in the context of a laboratory achieving successful emotional induction may be very difficult since induction techniques are limited by ethical and experimental feasibility. Musical mood induction is an attractive option to induce moods in experimental settings since subjects consistently report experiencing strong emotions in response to music (Juslin & Sloboda, 2001). Music has been used for mood induction in a wide variety of experiments, both alone and combined with other stimuli (for review, see Gerrards-Hesse, Spies, & Hesse, 1994). For example, music has been used in combination with reading self-referential statements (Mayer, Allen, & Beauregard, 1995; Richell & Anderson, 2004), with altered lighting (Davey, Startup, Zara, MacDonald, & Field, 2003), to study autobiographical recall (Setliff & Marmurek, 2002), salivary cortisol levels (Clark, Iversen, & Goodwin, 2001; Hucklebridge, et al., 2000), and emotional face judgments (Bouhuys, Bloem, & Groothuis, 1995). A growing literature has also investigated the changes in the brain that arise from inducing strong moods via music (reviewed in Lewis, 2002). For instance, music excerpts that were pleasurable for specific individuals were associated with reliable activation of emotion-related processing regions of the brain (Blood & Zatorre, 2001). These and other findings have supported that idea that music is processed in a special way by the brain (Peretz, 2001) and can tap powerfully into the neural circuitry that generates emotional responses.

It is generally accepted that large and reliable changes in physiological states are associated with emotional responses, regardless of the manner in which the emotional response was induced. There is consensus that such physiological changes are a reliable correlate of certain psychiatric disorders, including anxiety and panic disorders and depression (Berntson & Cacioppo, 2004; Berntson, Sarter, & Cacioppo, 1998; Grossman, 1983; Wientjes, 1992). However, whether specific physiological patterns for each unique normal emotional state exist is controversial (e.g. Collet, Vernet-Maury, Delhomme, & Dittmar, 1997; Hagemann, Waldstein, & Thayer, 2003; Levenson & Ekman, 2002). A meta-analysis and literature review by Cacioppo et al. (2000) highlighted the inconsistent results found in studies searching for distinct emotion-specific patterns of physiological activity, but indicated that autonomic activation may be greater in negative than positive valenced states.

Two psychophysiological measures thought to index emotional states are respiration and cardiovascular patterns.

Respiration patterns

A number of studies have suggested that the experience of emotional states is accompanied by respiratory changes (reviewed in Boiten, Frijda, & Wientjes, 1994; Ritz, 2004; Wientjes, 1992). One of the most well-established connections is between anxiety-related states and respiratory changes (e.g. Bass & Gardner, 1985; Grossman, 1983; Wientjes, 1992). Wientjes (1992) suggests that hyperventilation may be a normally occurring passive coping response in situations of pain, apprehension, anxiety, or fear. Stressful or effortful mental tasks also can increase respiration rate, and respiratory disregulation is associated with several diagnostic groups, including depression, panic disorder, and anxiety (Boiten, Frijda, & Wientjes, 1994; Wientjes, 1992). Evidence that voluntary alteration of respiration patterns can change subjective emotions (such as by reducing anxiety in a stressful situation) also suggests interactions between emotion and respiration (Bass & Gardner, 1985; Boiten, Frijda, & Wientjes, 1994; Grossman, 1983).

Other research has probed for specific respiratory patterns for basic emotions. Bloch, Lemeignan et al. (1991) quantitatively and qualitatively described unique patterns of respiration for each of six different emotion types (joy/laughter, sadness/crying, fear/anxiety, anger/aggression, erotic love, and tenderness) in trained actors. Particular patterns of respiration accompanied specific emotions; for instance fear/anxiety correlated with frequent pauses, increased respiratory rate, increased respiratory rate variability, and increased inspiration time relative to expiration time. Wientjes (1992) describes four breathing patterns associated with emotional states: rapid and shallow respiration in tense anticipation/anxiety, rapid and deep respiration in excitement/arousal/fear/anger/joy, slow and shallow respiration in passive grief/depression, and slow and deep respiration during sleep/deep relaxation. In an experiment using autobiographical recall mood induction Collet, Vernet-Maury et al. (1997) found significant differences in instantaneous respiratory frequency between emotional states: shortest mean breath lengths occurred during happiness, whereas the longest mean breath lengths were found in surprise, anger, disgust, and intermediate breath lengths occurred during fear and sadness. Boiten (1998) studied respiration changes during moods induced by emotional movie clips and found significantly shorter inspiratory duty cycle, shorter post-expiratory pause length, and greater total breath length variability for the positive films when compared to the negative films. These data indicate that respiratory

measures may provide a sensitive correlate of emotional experiences induced in a variety of ways.

Heart rate variability patterns

Heart rate variability may provide another measure of mood, although whether heart rate variability patterns are distinct for each emotional state is debated. A number of studies reported increased heart rate during anger, fear, and sadness (Collet, Vernet-Maury, Delhomme, & Dittmar, 1997; Levenson, 1992; Levenson, Ekman, & Friesen, 1990), while others reported increased heart rate during anger, fear, and sadness compared to happiness (Ekman, Levenson, & Friesen, 1983; Levenson & Ekman, 2002). Heart rate during disgust has been reported to be lower than during anger, fear, and sadness (Levenson, Ekman, & Friesen, 1990). Schwartz, Weinberger et al. (1981) found emotion-specific (happiness, sadness, anger, and fear) changes of diastolic and systolic blood pressure and heart rate while subjects performed autobiographical recall mood induction. Palomba et al. (2000) measured heart and respiration rate during viewing films designed to elicit either a threat/anxiety, disgust (surgery/mutilation), or neutral state, and reported an increase in respiration rate while viewing all films, an increase in heart rate during the threat/anxiety film, and a slight decrease during the disgust and neutral films.

Other researchers have not found evidence of differences in heart rate between specific emotions, but rather an increased heart rate across all emotions compared to a neutral state (e.g. Neumann & Waldstein, 2001; Prkachin, Williams-Avery, Zwaal, & Mills, 1999). Sinha, Lovallo et al. (1992) found changes in blood pressure and vascular resistance between emotional states but not in heart rate. Stemmler (1989) did not find respiration or heart rate differences between emotional conditions (fear, anger, happiness, control, induced by real-life task manipulation and autobiographical recall), although differences were reported in other psychophysiological measures (also Gendolla, Abele, & Krèusken, 2001).

Coordination of respiration with external signals

It is known that respiration is influenced by factors other than physiological requirements, in addition to factors that induce emotions. For example, respiration has been shown to coordinate to rocking frequency in newborns (Sammon & Darnall, 1994), steps while walking (Loring, Mead, & Waggener, 1990), passive leg movement (Gozal & Simakajornboon, 2000), and bicycle peddling (Kohl, Koller, & Jèager, 1981). This coordination may occur without conscious awareness (e.g. Haas, Distenfeld, & Axen, 1986;

Kohl, Koller, & Jèager, 1981). Haas et al. (1986) recorded subjects' respiration while they listened to a metronome and four musical pieces of varying rhythms and tempos, either with or without tapping to the perceived beat. Many subjects synchronized their respiration to musical rhythms without reporting a conscious effort at coordination, and more synchronization was found to pieces with simple, as opposed to complex, rhythmic structures.

Psychophysiological reactions to music

A subset of the literature examining physiological reactions while listening to music explicitly relates these reactions to those described in psychophysiological studies of specific emotions (reviewed in Krumhansl, 2002). Rickard (2004) found differences in skin conductance and "chills" but not heart rate or skin temperature between inductions. Nyklícek et al. (1997) measured a large number of measures of respiratory and cardiovascular activity while subjects listened to music chosen to induce specific emotional states (happiness, sadness, serenity, agitation) or neutral stimuli. The respiratory measures were found to best distinguish between the states (increase in happiness/agitation relative to sadness/serenity); few differences were found in the cardiovascular measures other than those attributable to respiratory effects. Similar results were reported by (Krumhansl, 1997): increased respiration rate during the clips chosen to induce happiness and fear compared to baseline and heart rate deceleration during the sadness induction.

The present study expands the literature of psychophysiological measurements of musically-induced emotions by examining individual changes in physiological activity during the stimuli and coordination of respiration with the music. The goal of the present study was to determine whether consistent cardiovascular and respiratory changes occur while subjects experience emotions induced by music. We chose our music stimuli in a pilot study based on its ability to reliably induce reports of strong happiness, sadness, and fear in the listeners. We hypothesized that (a) the induction of emotion would be associated with reliable changes in heart rate and respiration, and that (b) these changes in heart rate and respiration would differ systematically between the different induced moods. It was expected that changes would be consistent with those reported in previous studies: decreased respiration and heart rate during sadness compared to fear or happiness inductions, with the measures highest on the happiness inductions and intermediate during fear.

Materials and Methods

Participants

Eighteen subjects (10 females and 8 males) participated in the experiment. Subjects were screened to be neurologically and psychiatrically healthy, right-handed, with normal hearing (confirmed using audiometry), and without professional or college-level music experience. The subjects ranged in age from 31 to 74 years (M=50, Mdn=50); the distribution of ages was similar for the males (M=48, Mdn=48) and females (M=52, Mdn=51). Respiration recordings were not taken from five subjects (3 females and 2 males) due to a change in experimental protocol. Subjects provided informed consent prior to participation and were compensated for their time.

Stimuli

Music stimuli were selected from a large pool of potential stimuli using a pilot study. The chosen stimuli produced the most intense and specific reported experience of each target emotion: happiness, sadness, and fear (details are presented in Johnsen, 2004). The stimuli consisted of twelve music clips; four different clips were chosen to induce each target mood (fear, sadness, or happiness). Details of each stimulus appear in the Procedure section. The stimuli were short classical music selections taken from movie soundtracks ranging in length from 74 to 189 seconds (M=136 seconds). Stimuli of various lengths were used so that each clip could form a musically complete unit. The stimuli are labeled by a letter indicating the targeted mood (H=happiness, F=fear, S=sadness) and a number indicating its place in the presentation order (the presentation order was the same for all subjects). The music was selected based on how well it induced each specific mood; no effort was made to match tempo, mode, or pitch. The stimuli were presented via headphones at a loud, but comfortable, volume.

Procedure

After briefing the subject and obtaining informed consent, the electrodes and respiratory belt were placed. The subjects then completed inventories and tasks to allow time to acclimate to the laboratory setting prior to mood manipulation. Subjects completed various written measures, including inventories of current mood and previous musical experience. Participants then closed their eyes and rested for one minute, then rested for another minute as neutral auditory stimuli (tone sequences) were presented through the

same headphones used for music presentation. The subjects were given the following instructions:

"In this task, I will play for you some excerpts of background music, the sort you often hear in the background of TV shows and movies. As you listen to each clip, I would like you to continually rate the strength of the emotions you are feeling using this dial from "weak" to "strong," with "moderate" in between. You may move the dial as much or as little as you like based on your own responses to the music. There is no right or wrong answer to this task. What's most important is that your ratings are based on how you feel in response to the music."

Three sample clips were played, followed by the twelve experimental music stimuli (in the fixed randomized order). Physiological data was collected and subjects adjusted the dial to reflect the intensity of their emotional experience while listening to the stimuli. Following

Table 4.1. Stimuli details.

clip name	film name	track name	target mood	present-ation order	length (seconds)	dominant tempo (beats/minute)
H1	Charlie!	Cancan a Paris Boulevard	happiness	1	138	115
F2	Dangerous Liaisons	Tourvels Flight	fear	2	101	61
H3	A Midsummer Nights Sex Comedy	Vivace non troppo	happiness	3	122	124.5
F4	Crimson Tide	Alabama	fear	4	130	42.5
S5	Vertigo	Madeleine and Carlottas Portrait	sadness	5	99	65
H6	Gone with the Wind	Mammy	happiness	6	140	85.3
S7	Backdraft	Brothers	sadness	7	140	44
S8	Out of Africa	Alone on the Farm	sadness	8	149	68
F9	Vertigo	Vertigo Prelude and Rooftop	fear	9	100	60
H10	Dances with Wolves	The Buffalo Hunt	happiness	10	162	105
S11	Spartacus	Blue Shadows and Purple Hills	sadness	11	74	58
F12	Henry V	The Battle of Agincourt	fear	12	124	115.3

each clip participants completed a questionnaire that assessed current happiness, sadness, and fear on a Likert scale ranging from 0 "not at all (happy, sad, fearful)" to 9 "very (happy, sad, fearful)." Additionally, the questionnaire asked the subject to rate the level of "activation/energy" they experienced during the music (scale from 0 "none" to 9 "very much"), to select one word that best represented the emotion expressed by the music (regardless of their experience), and whether they had previously heard the music. After the final stimulus the recording equipment was removed, subjects were debriefed, thanked, and dismissed.

Physiological measures

Recording equipment

Respiratory activity was recorded with a TSD201 Respiratory Effort Transducer manufactured by BIOPAC Systems, Inc. (Santa Barbara, CA). The transducer consists of an elastic belt attached to a strain gauge which generates an electric signal proportional to the amount of tension on the belt, which in turn is caused by changes in chest circumference due to breathing. The belt was wrapped around the subject at the approximate height of the sternum and fastened to be snug but not uncomfortably tight. The subject was asked to take several deep breaths while the signal was examined to ensure that it rose and fell with respiration without exceeding maximum range. The EKG was taken with electrodes in the lead II configuration (an Ag-AgCl electrode placed over the right carotid artery in the neck and on the lower left flank; electrodes on the palms served as the ground for the entire system). Both signals were recorded at 1000 Hz using AcqKnowledge v. 3.7 (BIOPAC Systems Inc., 2003).

Derivation of measures

The time that each normal R wave, inspiration, expiration, and pause occurred was found using the computer program and methods described by Etzel et al. (2004; Etzel, Johnsen, Dickerson, & Adolphs, 2005) and visually verified for accuracy. The occurrence times were converted to RR interval, total breath length, inspiration length, and expiration length series for analysis. Statistical measures were derived from the physiological recordings of each clip for a sixty-five second period starting nine seconds after clip onset. The first nine seconds of each clip were omitted to allow orienting responses to pass and subjects to start experiencing the target mood. The sixty-five second analysis period was used since it is the longest length that could be derived from all the clips. All statistical

testing used a 0.05 significance level and was performed using R (R Development Core Team, 2005).

Two types of statistical analyses were performed. The first used two typical time-domain measures of heart rate variability (SDNN and SDSD) to summarize each subject's responses during the mood inductions and then compared these measures across conditions using mixed models[1]. SDNN is a measure of total heart rate variability, and is calculated by the standard deviation of the RR interval series (RR intervals are the amount of time between adjacent normal R waves in the EKG) (Berntson, et al., 1997; Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996). SDNNresp, the equivalent measure estimating total respiratory variability, was defined as the standard deviation of the breath length series. SDSD, the standard deviation of successive differences, is the standard deviation of the difference between adjacent entries in the RR interval series (Malik & Camm, 1995; Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996). SDSD is a measure of short-term heart rate variability (e.g. Hoyer, et al., 2002; Morrow, Andrews, Hickok, & Stern, 2000). The equivalent measure, SDSDresp, was calculated as the standard deviation of the difference in total breath length of adjacent breaths.

Respiratory sinus arrhythmia (RSA) was estimated using the peak-valley technique as defined by Ritz (Ritz, Thons, & Dahme, 2001). In brief, the peak-valley estimate of RSA is the mean time difference between the longest and shortest RR interval within each breath, with the requirement that the longest RR interval must occur after the shortest (Berntson, et al., 1997; Grossman, van Beek, & Wientjes, 1990; Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996). As calculated here, the RSA score for each breath is the difference (in ms) between the longest and shortest inter-beat (RR) interval occurring within the breath. If the shortest RR interval does not occur before the longest the breath is assigned a score of zero. Each breath was defined trough-to-trough, with the first RR interval in the breath starting at the first R wave after the start of the breath, and the last RR interval ending with the first R wave after the

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[1] Mixed model analysis was performed using the *lme* function in R (call: lme(fixed=meanRRLen~clip, data=mgd, random=~1|subID, na.action=na.omit)). The fixed effects were defined with the clip name as the primary covariate, the subject was used for the random effects, with the default general positive-definite symmetric covariance structure. Contrast tests were performed following significant clip effects using the *estimable* function to test for differences between clip types.

end of the breath. The mean of the RSA scores for all breaths in a clip was the clip's RSA estimate.

The second type of analysis was designed to identify changes in the physiological measures during each mood induction. This was done by calculating mean heart rate and mean respiration rate changes from baseline at regular intervals ("bins"). The mean heart rate change from baseline was calculated in one second intervals during the sixty-five second analysis period for each clip and subject[2], while the mean respiration rate was calculated for five second intervals[3]. The shape of these changes was summarized for each mood induction type using lowess curves and significance of differences between the curves was estimated using permutation analysis.

The pattern of mean heart and respiration rate changes after the stimuli was plotted using lowess curves to summarize responses within and between subjects. Lowess curves were calculated using the R *lowess* function (Cleveland, 1981). The lowess (also known as "loess") method calculates a locally-weighted least-squares line through the data. The resulting line is often similar to the mean but less sensitive to outliers and more appropriate for longitudinal data sets with unequal variances (Diggle, 2002). Statistical significance of the lowess curves describing the responses over time was assessed by calculating null bands by permutation testing for each curve (background information on permutation testing is available in Edgington, 1995; Good, 2001; Ludbrook & Dudley, 1998). Null bands indicate where the lowess curves describing the data fall under the null hypothesis (no relationship between the physiological changes and type of mood induction) (Buja & Rolke, 2005; Swayne, Cook, Buja, Hofmann, & Lang, 2005). If the true lowess line falls outside its null bands the line is considered unlikely to have occurred by chance. The null bands for each curve were set at the 95% and 5% quantile lines resulting from lowess curves for 1,500 permutations of the data set. Each permutated data set was created by randomly

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[2] The mean heart rate in the one second bin immediately preceding the start of the analysis period was used as the baseline; the mean heart rate in each of the following bins was subtracted from this baseline to obtain mean heart rate change. The mean heart rate in each bin was calculated by taking the weighted mean of all RR intervals overlapping the bin after the RR intervals had been converted to heart rate in beats/minute. At 1000 Hz, one second bins are 1000 samples long. The first and last sample number of each bin was identified, as well as that of the R wave immediately preceding the bin, all R waves in the bin, and the first R wave after the bin. The mean heart rate in the bin was calculated by taking the average of the heart rate at each of the 1000 samples contained in the bin.

[3] The mean respiration rate change in each bin was calculated in the same manner as the mean heart rate changes (described in footnote 2) except that the bins were enlarged to five seconds since mean breath length is greater than mean heart beat length.

reassigning the induction type label within each subject then calculating lowess curves in the same manner as for the true data.

Entrainment determination

The entrainment analysis was performed using normalized least mean squares adaptive filters to measure time correlation. This method involves using past values of the music clip as a reference signal to predict the respiration signal (the desired signal). As neither the music nor the respiration signals are stationary in time a linear adaptive filter was used for prediction; the predictor uses the previous 0.3 seconds (determined heuristically from a range of values between 0.1 and 2 seconds) of the music clip to predict the respiration signal[4]. The filter coefficients were updated using the normalized least mean squares algorithm (Manolakis, Ingle, & Kogon, 2000). If the music is correlated with the respiration signal in a statistical sense, then the estimator can predict the respiration signal with low mean squared error over intervals with a steady beat, implying that the music signal is influencing the subjects' respiratory response. Tracking is feasible only if the characteristics of the music signal are changing slowly in time relative to the adaptation time of the filter. The mean square error between the estimated respiration signal from the filter and the actual respiration was calculated. If the filtered signal is correlated with the music the mean squared error should be small. To estimate significance the filtering results using the actual clip (the music the subject was listening to at the time) for the reference signal are compared to the results from using the other clips as the reference signal for each respiration recording. All calculations to detect entrainment were performed in MATLAB (2003).

Results

Subjective results

Subjects reported experiencing the targeted mood at a stronger intensity than the other emotions following each induction. The mean rating given by the subjects on each clip to each question appears in Figure 4.1, with bars indicating standard error of the mean. The ratings were lower on the sad clips than the fear or happy ones, representing a more mixed reaction to the sad and fear clips than the happy ones. Nevertheless, the questionnaire

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[4] For this analysis, the music signal was downsampled and converted into a mono signal. The filter had a length of 1500 samples and a sampling rate of 1000 Hz (uses the last 1.5 sec of information). This sampling rate ensures that the system concentrates on lower frequency components of the music clip below 500 Hz.

results indicate that the music clips were effective at eliciting the targeted mood. Analysis of the dial and questionnaire data is presented elsewhere (Johnsen, 2004); this study did not attempt to relate the dial and physiological responses. Few subjects reported familiarity with the music and no subject was able to correctly identify the source of any clip.

Cardiovascular measures

Time-domain measures

The mean and standard deviation of each time-domain heart rate variability measure on each clip is listed in Table 4.2. Over all clips the mean RR interval length was 896.50 ms (SD=111.73), corresponding to a mean heart rate of 66.9 beats/min. The mean RR interval length varied from 886.37 (SD=116.95) ms on H1 to 909.10 (SD=109.46) msec on F9, a narrow range well within one standard deviation of the overall mean. Mixed model analysis was used to test whether the pattern of changes in the mean RR interval length was similar across subjects. The model returned a highly non-significant estimate (F(11)=0.72, p=0.71), indicating that mean RR interval length did not vary significantly with clip.
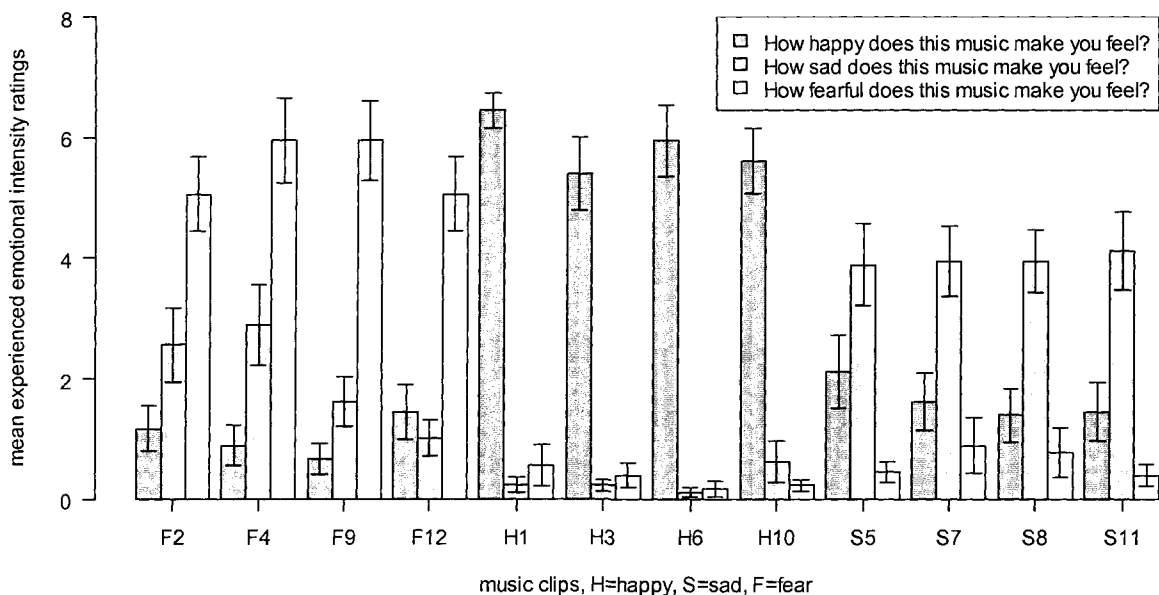
Figure 4.1. Mean ratings on each clip, bars indicate the standard error of the mean. The scale ranged from 0 ("none") to 9 ("very much"), N = 18.

The same analyses were performed on the SDNN (standard deviation of the RR interval series), SDSD (standard deviation of the differences between successive RR intervals), and RSA data (respiratory sinus arrhythmia). The mean and standard deviation of SDNN, SDSD, and RSA for each stimulus are included in Table 4.2. As with the RR interval lengths, the difference between the clips is well within one standard deviation of the mean for all measures. There is a trend towards a higher SDNN on the fear than the other stimuli, but this was not significant in the mixed model, although close to significance $(F(11)=1.82, p=0.054)$. The mean SDSD was similar on all clips; consistently the mixed model did not return a significant clip effect $(F(11)=1.37, p=0.19)$. The mean RSA was also similar on all clips, ranging from 26.37 to 47.98 msec with large standard deviations; the mixed model for RSA was not significant $(F(11)=0.83, p=0.606)$.

Activity during mood inductions

The lowess curves calculated for the subjects' mean heart rate changes during the happy (H1, H3, H6, and H10) sad (S5, S7, S8, and S11) and fear clips (F2, F4, F9, F12) appear in pane a of Figure 4.2. If the subjects' mean heart rate was constant throughout the induction the curves would remain at the zero line, representing no change in mean heart rate from baseline. The mean heart rate during the happiness inductions did remain near zero for thirty seconds, followed by a modest acceleration then deceleration back to zero. The mean heart rate during the fear inductions accelerated steadily the first half of the period, then returned to baseline, while during the sadness inductions the mean heart rate initially slowed, followed by a slow return to near baseline. The significance of these trends was assessed using null bands calculated from permutation testing, as described in Materials and Methods. The lowess curve for the happiness inductions (pane c of Figure 4.2) falls within the bands, indicating that the curve is not distinct from one that might have occurred by chance. The curve for the sadness induction (pane b), by contrast, falls below the bands until about 55 seconds, indicating a larger heart rate deceleration than would be expected if there is not an interaction between clip type and heart rate. Finally, the curve for the fear induction (pane d) rises above the null bands for about half the clip, indicating greater heart rate acceleration than expected by chance. It is not a contradiction to suggest that significant heart rate differences occurred during the clips while the time domain measures did not find significant differences since the two analyses detect different types of patterns.
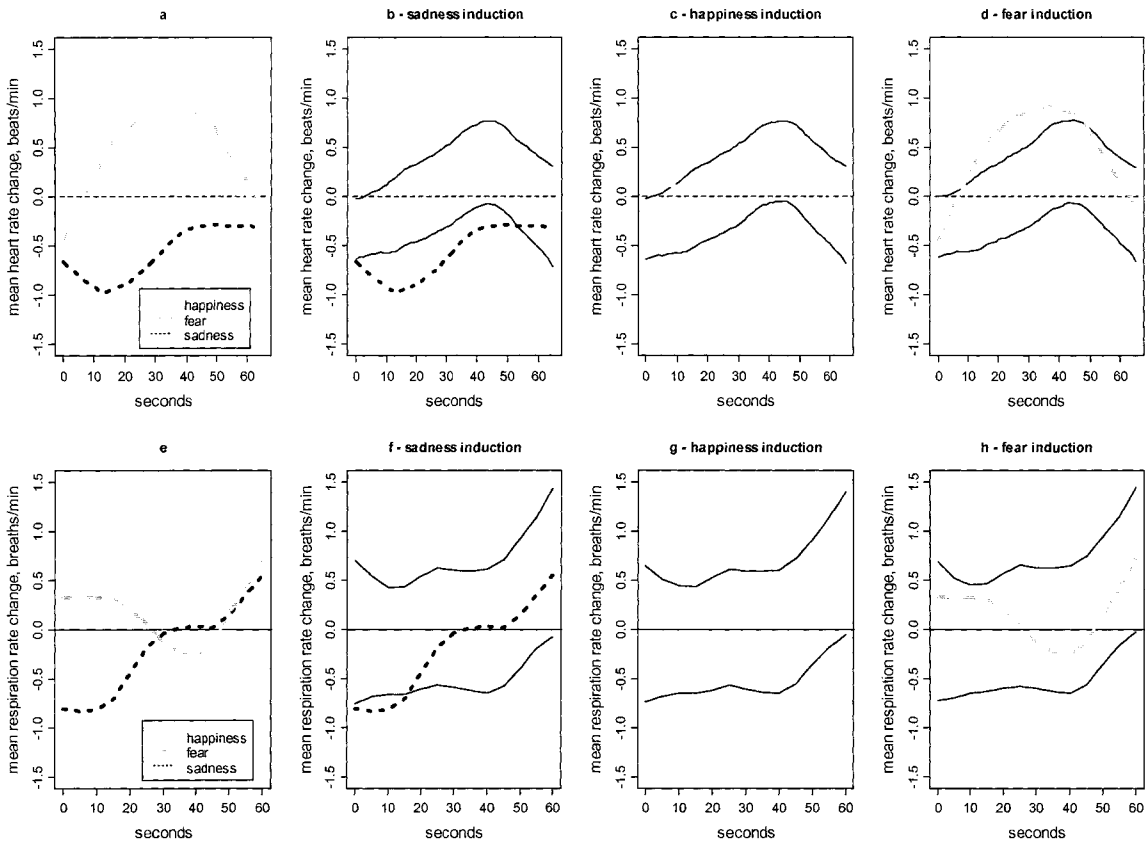
Figure 4.2. Lowess curves and null bands depicting the mean heart rate changes (panes a, b, c, d, N=18) and mean respiration rate changes (panes e, f, g, h, N=13) during the mood inductions. Mean heart rate changes are in beats/minute, mean respiration rate.

Table 4.2. Mean (standard deviation) of heart rate variability measures by clip. All values in milliseconds, N=18.

clip	RR interval length	SDNN	SDSD	RSA
F2	893.83 (119.64)	37.30 (41.86)	39.74 (58.06)	46.44 (75.22)
F4	902.91 (114.51)	45.55 (46.18)	52.51 (70.33)	39.70 (47.74)
F9	909.10 (109.46)	50.30 (46.02)	51.67 (73.83)	47.98 (74.43)
F12	893.27 (104.22)	40.21 (42.07)	42.99 (67.65)	39.16 (43.51)
H1	886.37 (116.95)	40.12 (46.48)	46.49 (76.55)	42.51 (67.55)
H3	893.73 (121.95)	42.23 (41.88)	47.92 (62.08)	43.09 (51.21)
H6	892.17 (117.28)	34.74 (34.09)	35.81 (49.29)	34.68 (50.87)
H10	896.79 (109.75)	40.51 (40.15)	38.52 (62.38)	41.62 (66.03)
S5	897.54 (112.74)	38.37 (34.60)	39.68 (55.43)	35.58 (30.49)
S7	899.00 (119.31)	43.48 (40.28)	42.37 (60.43)	26.37 (20.67)
S8	898.24 (114.53)	41.81 (41.68)	37.20 (60.13)	34.07 (36.10)
S11	895.42 (114.28)	37.36 (37.20)	36.98 (55.70)	30.63 (24.00)

Respiratory Measures

Time-domain measures

The mean and standard deviation of each respiratory measure for each clip is listed in Table 4.3. Averaging over all clips the mean breath length was 3447.03 (SD=380.06) ms, corresponding to 17.41 breaths/minute; mean SDNNresp 472.99 (SD=291.21) ms, mean SDSDresp 603.92 (SD=390.18) ms, mean inspiration length 1514.44 (SD=340.41) ms, mean expiration length 1930.69 (SD=367.52) ms, and mean inspiration duty cycle 0.44 (SD=0.08).

The mean breath lengths listed in Table 4.3 vary with clip type: the mean breath length is shortest on the happy clips (H1, H3, H6, and H10), longest on the sad clips (S5, S7, S8, and S11), and intermediate on the fear clips (F2, F4, F9, F12). The mixed model of mean breath length returned a significant clip effect estimate (F(11)=2.77, *p*=0.003), indicating that mean breath length varies significantly with clip. Further estimates (Table 4.4) were performed to determine the source of the variation. These estimates indicate that the mean breath length was significantly (*p*<0.0001) longer during the sadness than the happiness induction, and significantly (*p*=0.001) longer during the sadness than the fearful induction. There was not a significant difference (*p*=0.07) in mean breath lengths during the happiness and fearful inductions.

An interaction of clip type and mean expiration length is suggested by the figures in Figure 4.2. The differences were further examined by plotting the mean expiration length on each clip for each subject individually, indicating that for most subjects the mean expiration length was longer on the sad than the fear or happy clips (not shown). The mixed model of mean expiration length returned a significant effect of clip type (F(11)=2.44, p=0.01). The estimates (Table 4.5) indicate that the pattern of significant mean expiration length differences matches that of total breath length differences: expiration length was significantly (p<0.0001) longer during the sadness than the happiness inductions, and significantly (p=0.005) longer during the sadness than the fearful inductions. There was not a significant difference (p=0.09) between mean expiration length during the happiness and fearful inductions.

No relationships between mean inspiration length, SDNNresp, SDSDresp, or inspiratory duty cycle and mood induction type were found. The range of mean inspiration lengths for individual subjects tended to be narrow, with similar values on all clips. The mixed model of mean inspiration time (F(11)=0.37, p=0.96) did not show a significant effect of clip type. Despite the relatively constant mean SDNNresp and SDSDresp (Table 4.3)

across the inductions, the values for each individual subject varied a great deal between the clips (not shown). The pattern of variation varied for each subject however, so the mean was relatively constant. Mixed models of SDSDresp (F(11)=0.94, p=0.50) and SDNNresp (F(11)=0.94, p=0.50) did not find significant clip effects. The mean inspiration duty cycle for the individual clips was very similar, ranging from 0.41 to 0.47 (Table 4.3), well within one standard deviation of the overall mean of 0.44 (SD=0.08) and the mixed model (F(11)=0.91, p=0.53) did not find a significant interaction of clip type and mean inspiration duty cycle.

Table 4.3. Mean (standard deviation) of respiration measures by clip. All values in milliseconds, N = 13.

clip	breath length	SDNNresp	SDSDresp	inspiration time	expiration time	inspiration duty cycle
F2	3394.99 (324.42)	362.47 (125.16)	495.88 (221.85)	1519.61 (272.36)	1771.09 (317.34)	0.46 (0.08)
F4	3513.97 (394.74)	541.42 (293.54)	712.42 (466.75)	1516.71 (304.27)	1864.82 (291.52)	0.45 (0.08)
F9	3421.65 (312.94)	493.88 (281.11)	572.81 (289.34)	1434.12 (333.04)	1884.28 (403.84)	0.44 (0.10)
F12	3372.15 (387.01)	433.99 (407.68)	526.94 (510.12)	1545.81 (385.76)	1973.49 (337.54)	0.44 (0.08)
H1	3300.25 (330.18)	479.18 (290.96)	710.91 (498.16)	1447.52 (277.34)	2136.94 (347.16)	0.41 (0.07)
H3	3331.28 (283.00)	299.42 (124.18)	379.57 (133.55)	1629.52 (628.78)	1743.10 (328.27)	0.47 (0.11)
H6	3377.32 (376.98)	474.48 (260.62)	606.91 (427.03)	1605.48 (241.71)	1989.89 (386.97)	0.45 (0.07)
H10	3343.31 (308.35)	487.37 (194.80)	578.21 (322.11)	1472.96 (288.56)	2065.48 (306.95)	0.42 (0.06)
S5	3596.33 (386.98)	505.69 (322.87)	613.18 (369.33)	1484.06 (288.81)	1932.83 (384.34)	0.44 (0.08)
S7	3585.15 (339.25)	513.70 (255.59)	724.13 (416.66)	1508.93 (302.75)	1839.89 (311.25)	0.45 (0.08)
S8	3547.89 (388.94)	507.82 (243.48)	643.34 (273.21)	1519.97 (286.07)	2057.48 (443.31)	0.43 (0.06)
S11	3567.82 (613.89)	544.96 (492.99)	636.75 (522.96)	1481.08 (412.64)	1900.88 (440.14)	0.44 (0.11)

Table 4.4. Mixed model estimates for mean breath length.

Label	Estimate	Standard Error	t Value	DF	Pr > |t|
F - H	356.54	195.07	1.83	119	0.0701
F - S	-636.41	194.56	-3.27	119	0.0014
H - S	-992.95	189.34	-5.24	119	< 0.0001

Table 4.5. Mixed model estimates for expiration length.

Label	Estimate	Standard Error	t Value	DF	Pr > |t|
F - H	385.06	227.95	1.69	119	0.0938
F - S	-653.59	227.39	-2.87	119	0.0048
H - S	-1038.65	221.31	-4.69	119	< 0.0001

Activity during mood inductions

Lowess curves for the subjects' mean respiration rate changes during the happy, sad, and fear clips appear in Figure 4.2, pane e. The curve representing the changes during the fear and happiness inductions are similar, although the fear induction includes a decreased respiration rate followed by an increase during the last half of the clip. The curve for the sadness induction differs, showing an initial respiration rate decrease. As before, the significance of these trends was assessed using null bands calculated from permutation testing, which appear in panes f, g, and h of Figure 4.2. The null bands are relatively wide, encompassing nearly all of the true lowess curves, suggesting that differences in the pattern of respiration rate changes during the mood inductions are not significant. This conclusion does not suggest that conclusions drawn from the time-domain measures are incorrect; the two analyses capture different aspects of variability.

Entrainment

The correct music clip tended to predict the respiration signal for the corresponding case more accurately (in terms of mean squared error) than the other music clips used in this study. The prediction is most accurate during periods where the music has a fairly steady rhythm. Further analysis is necessary to determine which components of the music the subject may be responding to and the speed of the physiological response. The results are illustrated in Figure 4.3, which shows the respiration and error signals for one subject. In this figure the dark line is the estimation error that results when the clip the subject was listening to is used to train the filter, while the lighter lines show the estimation error when other clips were used. The error is much less when the clip the subject was listening to is used to train the adaptive filter, showing that for this subject during these clips there is a relationship between the music signal and the respiration signal. The mean squared error between the respiration signal and the filtered estimate of the respiration signal for each subject and clip appear in Table 4.6 and are plotted in Figure 4.4.

The mean squared errors vary both by subject (Table 4.6) and clip (Figure 4.4). Comparing the columns of Table 4.6 it can be seen that the errors tend to be smaller on all clips for some subjects (e.g., b, c, h, i, k), indicating that their respiration matched the music

more closely. Also, several clips did not predict any subjects' respiration signal well (e.g., S8 and H6), probably due to these clips' lack of a strong rhythm and/or frequent rhythm changes. Clips with the steadiest beats (e.g. H10, S11) had the lowest error for most subjects, suggesting that clips with a standard rhythm tend to result in more entrainment, or perhaps that these clips are better for predicting quasiperiodic signals (such as respiration) in general.
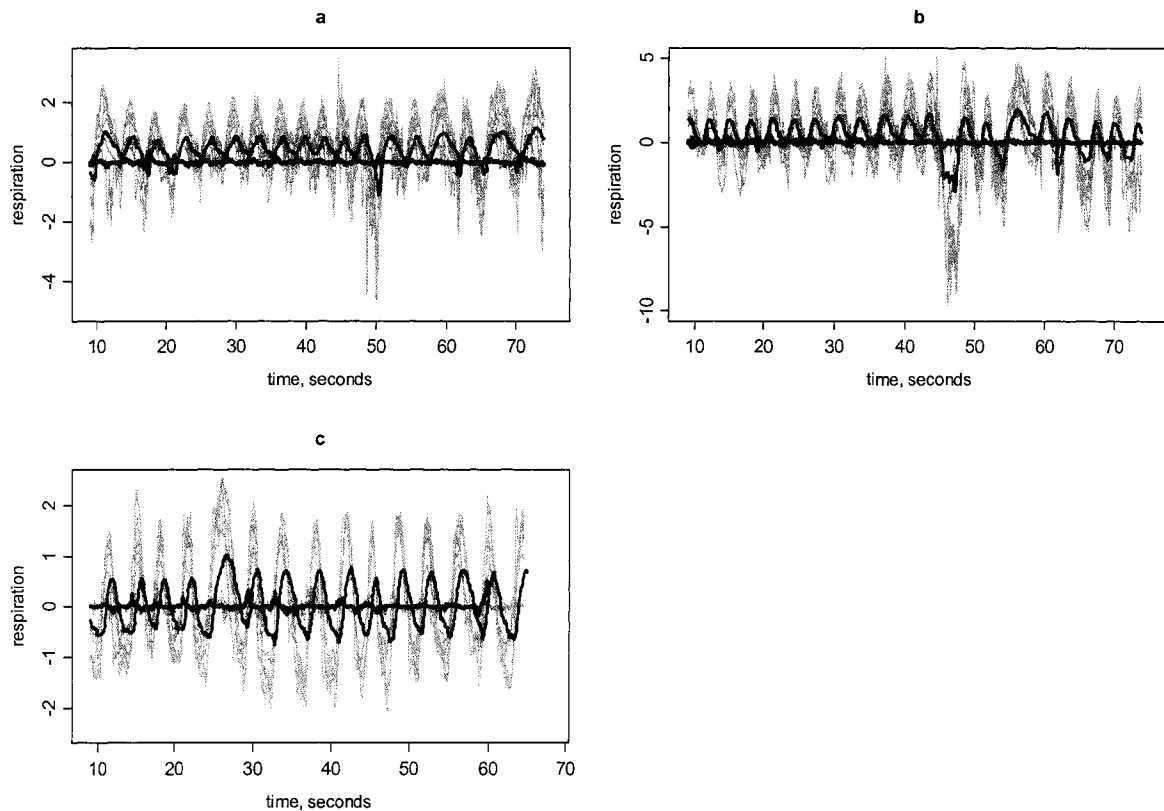
Figure 4.3. Representative signals to illustrate the results of the entrainment analysis. These three plots show the respiration for one subject during three different stimuli: F9 (pane a), H10 (pane b), and S11 (pane c). In each plot the blue line is respiration, black is signal error for the matching clip (F9 for pane a, H10 for pane b, S11 on pane c), and orange is the signal error when the other clips were used as predictors. The error when the clip the subject was listening to (black line) was used as the predictor is generally less than the error to the other clips, indicating higher correlation between the matching clip and the respiration signal than the other clips.

Table 4.6. Mean squared error estimates for each clip. The mean squared errors were estimated over the 65-second period starting 9 seconds after the onset trigger. Missing data indicated by NA.

Clip\Subject	a	b	c	d	e	f	g	h	i	j	k
H1	0.78	0.21	0.27	1.67	3.20	1.29	2.61	0.56	0.16	1.36	0.10
F2	8.95	0.09	0.07	1.01	NA	6.00	7.44	1.31	0.31	NA	0.21
H3	4.99	0.03	0.19	1.66	7.06	4.20	6.65	0.64	0.30	11.53	0.13
F4	11.68	0.03	0.26	1.75	8.10	3.21	9.41	0.86	0.09	2.84	0.09
S5	0.19	0.01	0.03	1.67	2.58	1.04	6.97	0.39	0.27	0.14	0.09
H6	15.55	9.58	2.39	11.17	7.43	4.90	13.72	5.91	2.81	16.11	0.15
S7	8.29	2.50	0.77	5.76	6.80	4.88	9.96	4.06	1.58	3.96	1.68
S8	12.69	14.96	2.93	12.57	7.13	8.54	16.19	10.69	2.81	7.99	9.19
F9	1.05	1.16	0.36	0.98	2.23	1.73	7.61	0.68	0.54	2.39	1.26
H10	0.24	0.00	0.13	0.09	0.99	0.20	1.54	0.07	0.02	0.30	0.01
S11	0.03	0.05	0.02	0.08	0.23	0.10	1.99	0.03	0.04	0.71	0.01
F12	1.82	0.19	0.11	3.82	3.64	0.97	14.86	0.32	0.39	5.16	0.07
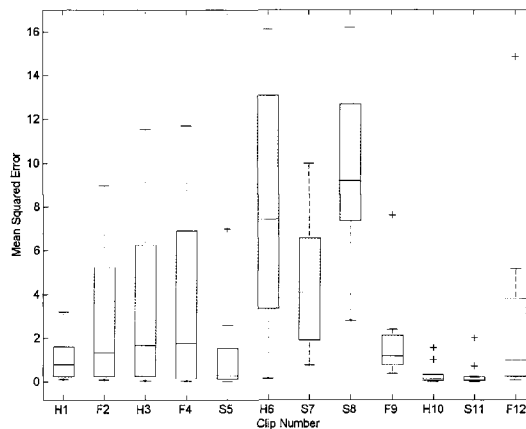
Figure 4.4. Boxplots of the mean squared error estimates for each clip.

Discussion

Based on subjects' reports, the musical mood induction used in this experiment was successful. The subjects reported experiencing moods while listening to the music; in many cases strong emotions. The subjects' median emotional intensity ratings indicate distinct emotional experiences: the median rating was 6 for the fear and happiness inductions and 4 for the sadness induction on a scale of 0 ("none") to 9 ("very much"). No differences were found on any of the traditional cardiovascular measures examined, but evidence was found that the heart rate decelerated during the sadness induction and accelerated during the fear inductions. Differences in total breath length and total expiration length were found in the

expected direction (slower respiration during the sadness than the fear or happiness inductions) on the time-domain measures but not when changes during the clips were examined.

Previous research and theories of the mechanisms of emotional experience suggest that measurable physiological changes accompany the experience of strong emotion, and that music is capable of inducing such strong emotion. We found modest evidence that the physiological changes occurred consistently with the mood inductions. Distinct differences in measures of respiratory and cardiovascular activity occurred between the mood inductions. Both the mean total breath length and expiration length had significant interactions with clip type: mean breath and inspiration lengths were longest for the sad induction, intermediate during fear, and shortest during the happiness induction. Despite this significant interaction it is not clear that the differences we found in respiratory activity were due to the induced emotion because the music used for the different emotion inductions varied greatly in dominant rhythm.

The music used to induce happiness had quick, toe-tapping rhythms and melodies, in stark contrast to the sadness-inducing music, dominated by long chords and much slower tempos. We chose the stimuli because they were the most consistently shown to induce emotion in the pilot study; no effort was made to match rhythms or tempos. This is consistent with the protocol used in previous musical mood induction experiments (e.g. Krumhansl, 1997; Mayer, Allen, & Beauregard, 1995; Nyklícek, Thayer, & Van Doornen, 1997). It is not surprising that our pilot study resulted in pieces with different tempos: musical theory and research suggests that tempo is a critical determinant of the mood associated with music. Mode and tempo are often cited as the characteristics most influential for emotional expressiveness in music (Dalla Bella, Peretz, Rousseau, & Gosselin, 2001; Peretz, Gagnon, & Bouchard, 1998). According to a review by Gabrielsson and Lindstrom (2001), fast tempos have been associated with expressions of activity/excitement, happiness/joy/pleasantness, potency, surprise, anger, and fear. Slow tempos are associated with expressions of calmness/serenity, dignity/solemnity, sadness, tenderness, boredom, and disgust. Further emphasizing the relationship between tempo and mood, Gottselig (2000) found that the ability of subjects to correctly identify the emotion expressed by music was related to their temporal auditory perception.

The short-term cyclic correlation analyses suggest that subjects' respiration became partly entrained to the music beats on many clips, indicating that the interaction of clip type and physiological measures may be due to differences in the tempo of the music used to induce each emotion rather than emotional experience itself. We do not believe that the

subjects were consciously attempting to match their breathing to the music's tempo; manipulating the rating dial and preparing for the post-clip questionnaires should have dominated the subjects' attention (subjects were not asked about their respiration, however). Instead, it is likely that the subjects unconsciously matched their breathing to dominant tempos. Past observations have identified a tendency of subjects to breathe with musical rhythms (Diserens, 1923); our finding of a relationship between respiration and the tempo of music is consistent with the more recent findings of Haas et al (1986). It would be interesting for future studies to examine music similarity and features (such as timbre) to check for frequency-based responses to the clips (Park, 2000), or evaluate the clips with acoustic similarity measures (Berenzweig, Logan, Ellis, & Whitman, 2003). It is also possible that the differences in heart rate found between the fear and sadness inductions were due to the driving of respiration by the music tempos, since respiratory changes can cause heart rate variability changes (reviewed in Boiten, 1996; Grossman, 1983). This finding, that subjects respond to aspects of the stimuli in addition to any induced mood, is not unique to music stimuli, but occurs with many types of stimuli.

The low magnitude of cardiovascular and respiratory differences between the mood inductions is surprising given the intensity of emotional experience reported by the subjects. There are several possible explanations for this finding. One possible explanation is that the subjects exaggerated their experienced mood intensity ratings. Previous work has shown that the efficacy of musical mood induction procedures is heavily dependent on subject instructions (Hermans, De Houwer, & Eelen, 1996; Lenton & Martin, 1991). Lenton and Martin (1991) compared the ability of music and "subliminal" music (silence) to induce moods under two sets of instructions (telling the subjects that they will be asked to rate their mood versus performing unspecified future "tasks") and found that instructions containing references to mood were necessary and sufficient for successful mood induction measurements. The instructions provided to the subjects in this study did not include guidance on how to change their mood as is sometimes done during musical mood induction (Clark, 1983; Clark & Teasdale, 1985; Sutherland, Newman, & Rachman, 1982), but did include directions to rate the "strength of the emotions you are feeling" continuously, implying that the subjects should feel emotion in response to the music. It is possible that the subjects responded to the questionnaire based more on the mood that they perceived the music expressing than the mood they actually felt. If this occurred and subjects only experienced minor emotions the physiological correlates may be too subtle to detect with our methodology.

Another possible explanation is that the subjects chosen for this experiment had muted cardiovascular responses due to age. The average age of the subjects was 50, the oldest 74. Many measures of cardiovascular activity are reduced in variability in older people (Antelmi, et al., 2004; Stein, Kleiger, & Rottman, 1997; Umetani, Singer, McCraty, & Atkinson, 1998). As a result, repeating the experiment with younger subjects may produce larger-magnitude findings of emotion-related cardiovascular change, although this would raise questions of the necessity of cardiovascular and respiratory responses for emotional experience.

It is also possible that significant differences were not found in the time-domain measures of cardiovascular activity due to the small number of participants and stimuli length in this experiment. Similar previous studies (Krumhansl, 1997; Nyklícek, Thayer, & Van Doornen, 1997), which did report significant time-domain measure differences, included more than twice the number of participants and longer recordings than this study. The effect size reported in those studies was very small, so a larger sample size than used here may be needed to obtain significant results. This study did find evidence of changes in mean heart rate during the mood inductions consistent with the differences in time-domain measures reported in those previous studies, however, so the results are not contradictory.

The findings of this study add to the understanding of physiological reactions to emotions induced by music. Time-domain cardiovascular differences were not found, in contrast to previous research (Krumhansl, 1997; Nyklícek, Thayer, & Van Doornen, 1997), although evidence for differences in heart rate within the clips between the inductions was identified. The entrainment of respiration to the music in this study, where conscious coordination should have been minimized by instruction and concurrent tasks, adds emphasis to previous reports that respiration can be driven by music (Diserens, 1923; Haas, Distenfeld, & Axen, 1986). The ability of many music tempos to drive respiration complicates the use of music for mood induction, and may make it impossible to separate physiological reactions to tempo from those due to the experienced mood. It remains to be seen whether entrainment is necessary for subjective reports of emotional experience, but clearly entrainment must be considered in studies utilizing musical mood induction and physiological measurements.

Acknowledgements

CHAPTER 5: PHYSIOLOGICAL RESPONSES WHILE VIEWING A COMPLEX EMOTIONAL VIDEO STIMULUS AND DURING SELF-REGULATION

Abstract

Many previous studies have attempted to relate patterns of physiological changes with emotional states, but the findings are not yet complete nor well understood. This chapter discusses changes in physiological state associated with specific scenes in a movie designed to induce emotion. The movie contained two distinct halves; the first half was upbeat and cheerful while the second was upsetting and sad. Within each half were scenes of particular intensity, including a wedding and the death of a young mother. Additionally, subjects viewed the movie in one of three conditions, consisting of instructions to watch the movie in a neutral way or to enhance or repress emotional experience. Distinct patterns of physiological responses were observed, both to particular movie scenes and associated with condition. In general, subjects in both the enhance and repress condition were more physiologically aroused than neutral condition subjects, perhaps related to task difficulty; in contrast to the findings in previous studies of emotional regulation using reappraisal methods. Changes in physiological state were most often observed at the wedding reception and death scenes. The changes at the death scene, though varying with condition, can be characterized by decreases in respiration and heart rate combined with increases in EDA, RSA and rMSSD. The wedding reception scene was characterized by increases in respiration and heart rate but a decrease in RSA; EDA increased in enhance and decreased in repress subjects.

Study Description

The data in this chapter was collected during an experiment designed and conducted by Unni Jensen, under the supervision of Ralph Adolphs, at the University of Iowa. The experiment was intended to investigate the influence of emotion regulation on recall accuracy. The protocol spanned two days: on the first day subjects viewed a short movie designed to induce emotion, and on the second day subjects completed an online test evaluating their memory for movie details. This study is an expansion of a pilot study also conducted by Unni Jensen (Jensen, Lewis, Tranel, & Adolphs, 2004). The pilot study used the same stimulus and memory test, but did not include physiological measurements. An interaction of memory and regulation was found in this study: subjects instructed to

suppress their emotional responses using reappraisal methods performed less well on the memory test. It was expected that the additional subjects tested here would also exhibit an interaction of memory and condition (as was observed in the pilot study), and that physiological responses during encoding would vary with condition, correlating the physiological responses with recall test performance. This chapter contains an analysis of the physiological data collected during the movie; analysis of the memory data was carried out by Unni Jensen and will be available elsewhere.

Subjects

The subjects in this study were females ranging in age from 26 to 55 years (M=41). The subjects provided informed consent and were reimbursed for their time. All subjects were without serious mental or physical conditions. Physiological data for 31 subjects is available; respiration data is missing for four subjects while nine subjects are missing skin conductance data due to equipment failure.

Each subject was randomly assigned to one of three conditions: "repress" (10 subjects) "enhance" (10 subjects), or "neutral" (11 subjects). These conditions differed only in the instructions given the subjects prior to movie viewing: in the neutral condition the subjects were asked to watch the movie "as if they were at home or in a movie theater." In the repress condition subjects were asked to watch the movie "as a movie critic," remembering that the movie is not real and that the characters are actors. In the enhance condition, subjects were to watch the movie "as if the depicted events were actually happening" to themselves or a family member.

These conditions are similar to those used in previous studies of physiological correlates of emotion regulation, allowing the results of this study to be compared with previous work. Using the emotional regulation process model proposed by Gross (Gross, 1998; Gross, 2002), the subjects were instructed to use cognitive change methods of emotion regulation, particularly reappraisal, to increase or decrease their responses. In particular, the instructions used parallel those used by Ochsner and colleagues (Ochsner, et al., 2004). Unlike some previous work (e.g. Demaree, et al., 2006; Gross, 1998) subjects were not instructed to attend to their facial expression, although demand characteristics to do so may have been present, since the experimenter remained in the room throughout recording.

Stimulus

A single movie was used as the stimulus in this study, created by editing scenes taken from "Steel Magnolias" (Ross, 1989) to create a coherent story 13 minutes 12 seconds in length. The emotional tone of the movie changes abruptly approximately halfway through the film (6:55): the first half of the movie is cheerful, composed of happy scenes and people (wedding, parties, a young child), while the second is sad (illness, death, funeral, crying people). In brief, the film starts with a scene of a happy couple conversing, then shifts to their wedding, then to the wedding reception. The wedding reception is full of happy people dancing to music; the couple leaves as the guests throw rice. The next scene is after some time has passed, and shows a Christmas party scene in which the father of the bride cheerfully announces that the bride is pregnant. The next scene shows the bride and groom with extended family members at their son's first birthday party. The tone of the movie shifts at the next scene, in which the wife, home alone taking care of the still-young son, falls ill and collapses. She is then depicted attached to life-support machines in the hospital; the groom signs a form and the machines are turned off. She dies, surrounded by sobbing family members. The scene shifts to the funeral; a somber scene with many crying people. The final scene is comparatively neutral, showing the bride's mother with the young son. The duration and timing of each scene is listed in Table 5.1.

Measures and Quantification

All physiological signals were recorded at 1,000 Hz using a Biopac MP150 system (Biopac Systems, Santa Barbara, CA). Heart rate was measured using two

Table 5.1. Timing and description of movie scenes.

Time	Scene Description
0 - 1:26	bride and groom conversing before their wedding, smiling
1:26 - 3:13	wedding: walking down the aisle, standing by the minister
3:13 - 4:46	wedding reception: dancing, music
4:46 - 5:26	newlyweds leave reception, guests throw rice
5:26 - 6:10	Christmas party, bride's father happily announces pregnancy
6:10 - 6:55	son's birthday party
6:55 - 8:03	bride collapses, son cries, groom arrives home frantic
8:03 - 8:41	bride on life support in hospital, relatives crying
8:41 - 9:04	groom signs form to disconnect life-support machines, bride dies
9:04 - 10:06	life-support machines stop, bride dies, relatives cry
10:06 - 10:32	bride's mother cries while driving to funeral
10:32 - 12:34	funeral, family and groom sad
12:34 - 13:12	son with bride's mother

electrocardiograph electrodes, one placed on the right side of the neck and the other on the left side of the torso below the rib cage. Skin conductance (electrodermal activity, EDA) was measured using a pair of Ag-AgCl electrodes, one placed on the thenar and one on the hypothenar surface of the left palm. Finger temperature was recorded with the TSD202B Digit Surface Probe, which was taped to the palm side base of the left index finger. Respiratory activity was recorded with a TSD201 Respiratory Effort Transducer, wrapped around the subject's chest at the approximate height of the sternum.

All signal processing was performed offline. Heart beats and breaths were derived using puka, as described in Chapter 3 of this thesis. Finger temperature and EDA were quantified using AcqKnowledge (BIOPAC Systems Inc., 2003). Finger temperature was taken as the mean temperature during the time of interest; no further processing was done. NS-SCRs were identified using a method adapted from option C in Biopac's *Application Note 216: Scoring Methods for Electrodermal Response Changes,* (BIOPAC Systems Inc., accessed 7 February 2006)[5]. Skin conductance level SCL was quantified as the skin conductance immediately prior to each NS-SCR. All statistical testing (0.05 significance level) and graphing were performed using R (R Development Core Team, 2005).

An additional measure, finger temperature, was included in the data for this chapter. Changes in finger temperature can be used to index sympathetically-modulated vasoconstrictor responses (Kistler, Mariauzouls, & von Berlepsch, 1998). Increases in sympathetic activity cause vasoconstriction (a reduction in skin blood volume) which leads to a decrease in finger temperature (Kistler, Mariauzouls, & von Berlepsch, 1998). Decreases in finger temperature have been observed in association with stimuli which increase sympathetic activity, such as stress, anxiety, deep breathing, and fear (reviewed in Levenson, 1992). For example, Kistler, Mariauzouls, & von Berlepsch (1998) monitored subjects' finger temperature while viewing scenes from Alfred Hitchcock's "Psycho." They found that finger temperature decreased throughout the upsetting, anxiety-provoking shower scene, reaching a nadir when the murder victim was shown dead. Increases in finger temperature have also been associated with general negative affect, particularly anger (Levenson, Carstensen, & Gottman, 1994).

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[5] First, the data was smoothed using the AcqKnowledge smoothing function (Transform → Smoothing, median value, value of 50). Second, the data was filtered using a high-pass filter (Transform → Digital filters → IIR → High Pass, set to 0.05). Finally, all peaks higher than a 0.05 µS threshold were identified (Transform → Math Functions → Threshold, lower at 0.05 µS, upper at 0.0501 µS). All peaks identified in this way were considered NS-SCRs.

Results: Differences between Conditions

This study used a lengthy (13 minutes 12 seconds) movie stimulus previously shown to be capable of evoking several strong emotions (happiness and sadness, data not shown). It was expected that the subjects would experience multiple moods while watching the movie, and that these moods would be accompanied by changes in the subjects' physical state, such as a reduction in heart and respiration rate during the sad portion of the movie. Also, women in different emotion-regulation conditions could have different reactions to scenes in the movie. The data was examined for changes over time by dividing the movie into one-minute bins. Each physiological measure was calculated for each bin and compared to identify changes over time. The movie is not exactly 13 minutes long, so the first twelve seconds were omitted from the analysis (the first bin starts twelve seconds after the start of the movie). This initial twelve second period is a relatively mood-neutral section of the movie, and may allow the subject to adjust to the stimulus.

The same analyses were performed for each measure on both the raw data and data change scores. Change scores were calculated for each subject by using the subject's measure on the first minute as baseline. This baseline was subtracted from the rest of the bins, converting the data to change from baseline. Mean, standard deviation, and median for each measure and minute is shown in tables. MANOVAs were performed for each measure to test for differences between conditions when possible. The data for each subject was plotted as curves, while lowess curves were calculated to summarize the data for each condition, and significance of differences between lowess curves was evaluated using null bands created by permutation testing, as described in Chapter 2. Vertical lines on the plots indicate the onset and end of the wedding reception and death scene, respectively.

Heart Rate Variability (HRV)

Mean Heart Rate (HR)

The mean, standard deviation, and median heart rate in each bin is listed in Table 5.2. The mean heart rate in each bin for each subject, separated by condition, is shown in panes a, b, and c of Figure 5.1. Most lines are fairly horizontal, reflecting that each subject has her own resting heart rate; changes due to the movie are superimposed upon this resting heart rate. It also appears that the range of subjects' heart rates is approximately the same in all three conditions. Three subjects in each condition have more variability than the others; these lines have more abrupt changes in direction. Heart rate change curves were calculated to make changes in each subjects' heart rate more apparent and reduce the

impact of different resting heart rates; these change scores are plotted in panes d, e, and f of Figure 5.1. The range of changes is about the same for subjects in all three conditions, though the HR of one enhance condition subject decelerated at the end of the clip more than the others. There is more variability in the mean HR in the enhance and repress conditions than the neutral, and the neutral condition's lowess curve (Figure 5.2 pane d) is closer to zero.

Figure 5.2 shows mean and lowess curves at each time point for both mean HR and the HR change scores. The lowess and mean curves are very similar for mean HR (panes a and b): HR is highest for the enhance condition subjects, intermediate for the repress, and lowest for the neutral. All three curves are within the null bands, however, suggesting that the HR difference between the groups is not larger than could have occurred by chance, given the present data and assuming no condition differences. Consistently, a MANOVA did not find a significant difference between the groups (condition: df=2, Wilks=0.28, F=1.27, p=0.25, residuals=28), although the intercept was significant (condition: df=1, Wilks=0.34, F=2.74, p=0.03, residuals=28).

More overlap is present in the change curves (Figure 5.2 panes c and d), and these mean and lowess curves differ. The enhance mean curve is lower than the lowess enhance curve, probably because the enhance mean is pulled down by the subject whose HR decelerated more than the others (lowess curves are less affected by outliers). The lowess curve for the neutral condition is nearest to zero during the first half of the clip, then drops below zero while the curves for the enhance and repress conditions rise. Portions of the curve for the neutral subjects are outside the null bands, suggesting that there may be a difference in HR changes between the neutral and other subjects. A difference in the responses for subjects in different conditions is also present at the very beginning of the movie: the enhance subjects experienced a rapid HR acceleration, while the neutral and repress subjects were near zero during the first two bins. This difference can be seen in the subjects' data in Figure 5.1 panes d, e, and f: the enhance subjects HR change curves begin above zero, while the neutral and repress subjects' curves are split between initial acceleration and deceleration. The lowess curve for the repress subjects' HR accelerated however, so that it is above the null bands by the 4th bin (pane d of Figure 5.2).
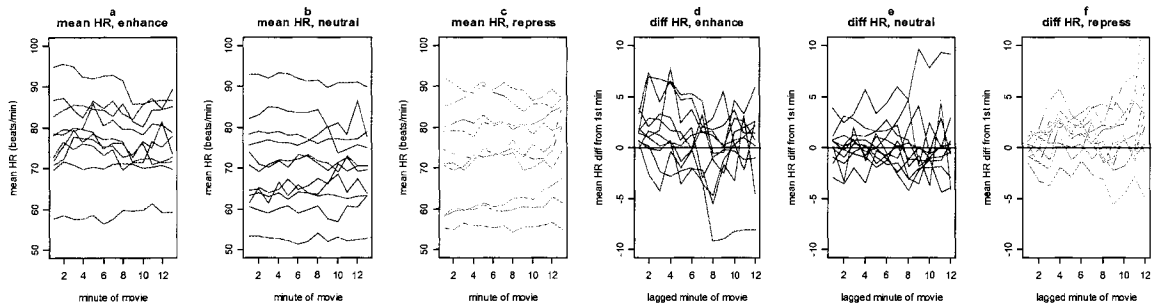
Figure 5.1. Actual HR (beats/minute) and HR change from baseline, by condition. Pane a: mean HR, enhance condition subjects. Pane b: mean HR, neutral condition subjects. Pane c: mean HR, repress condition subjects. Pane d: mean HR change, enhance condition subjects. Pane e: mean HR change, neutral condition subjects. Pane f: mean HR change, repress condition subjects.
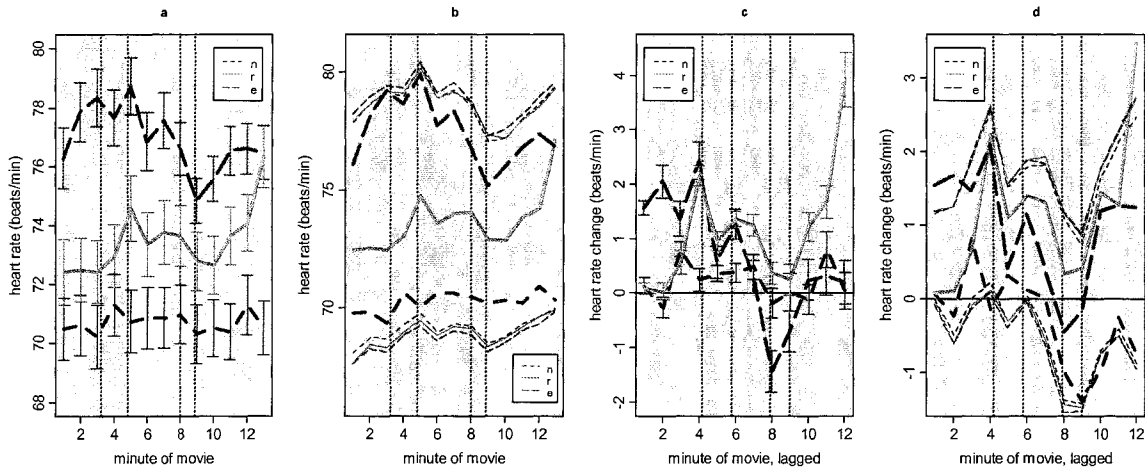
Figure 5.2. Means with bars indicating standard error of the mean and lowess curves with null bands. Panes a and b: mean HR (beats/minute). Panes c and d: mean HR change.

Table 5.2. HR (beats/minute) mean, median, and standard deviation.

min	all subjects mean (sd)	mdn	enhance mean (sd)	mdn	neutral mean (sd)	mdn	repress mean (sd)	mdn
1	72.96 (11.25)	72.52	76.28 (10.32)	75.25	70.46 (11.43)	70.54	72.38 (12.24)	72.65
2	73.53 (11.19)	73.43	77.84 (10.01)	77.34	70.58 (11.36)	68.96	72.46 (11.87)	72.32
3	73.52 (11.35)	74.60	78.33 (9.84)	79.15	70.19 (11.88)	69.99	72.39 (11.61)	71.54
4	73.86 (10.95)	75.86	77.65 (9.36)	78.47	71.27 (11.62)	71.64	72.93 (11.71)	72.62
5	74.52 (11.34)	75.50	78.71 (9.54)	78.70	70.70 (11.59)	70.13	74.53 (12.28)	75.00
6	73.57 (11.04)	73.38	76.85 (9.83)	76.23	70.82 (11.54)	72.07	73.33 (11.86)	73.35
7	73.93 (11.00)	74.47	77.55 (9.55)	76.74	70.83 (11.27)	72.79	73.73 (12.04)	73.86
8	73.63 (10.47)	74.84	76.59 (9.06)	76.33	70.94 (10.96)	70.94	73.61 (11.45)	75.82
9	72.54 (9.84)	72.66	74.84 (7.58)	73.17	70.27 (10.97)	69.44	72.74 (10.94)	72.99
10	72.81 (9.74)	72.31	75.57 (7.75)	76.36	70.47 (10.64)	70.98	72.63 (10.74)	73.83
11	73.39 (9.91)	72.98	76.53 (8.26)	75.86	70.34 (10.45)	72.49	73.59 (10.75)	73.11
12	73.88 (10.09)	74.02	76.59 (8.61)	77.89	71.25 (11.15)	70.51	74.05 (10.51)	74.71
13	74.30 (10.31)	74.50	76.48 (9.12)	75.77	70.50 (9.95)	70.48	76.30 (11.57)	80.24

rMSSD

rMSSD, a measure of short term HR variability, was calculated for each one-minute bin during the clip, as given in Table 5.3 and shown in Figure 5.3 and Figure 5.4. As with the mean HR, the baseline and amount of variability in rMSSD varied by subject: one neutral subject had a lower rMSSD than the other subjects, while another neutral subject had a very large increase in rMSSD at the 4th bin. Many subjects have a rMSSD increase near the 4th bin (wedding reception with dancing) and the 9th bin (when the life-support equipment is disconnected and the bride dies). The rMSSD lowess curves (Figure 5.4 pane b) are almost entirely within the null bands; only the peak for neutral subjects at the 3rd bin extends beyond the bands. Nevertheless, the curves for the neutral subjects are consistently above those for the repress, which in turn are above those for the enhance. A MANOVA did not find a significant difference between the groups (condition: df=2, Wilks= 0.33, F=1.04, p=0.44, residuals=28), nor a significant intercept (condition: df=1, Wilks=0.44, F=1.80, p=0.13, residuals=28).

rMSSD change curves are plotted in panes d, e, and f of Figure 5.3, and panes c and d of Figure 5.4. There is one subject in each condition that has larger variation in their rMSSD than the other subjects in these curves as well. There is a small increase in the rMSSD change scores at the 8th bin (9th minute, death scene); particularly in the enhance and repress subjects. This increase at the 8th bin is much more apparent in the mean scores (Figure 5.4 pane c), which also has an increase at the 3rd bin (4th minute, wedding reception scene). The peak at the 3rd bin is not visible in the lowess curves (pane d), suggesting that it may be primarily due to the subject in each condition that had large variability; these large-variability subjects each had an unusually large increase in rMSSD at the 3rd bin. In the lowess curves (pane d), the largest rMSSD increase is in the enhance subjects from the 6th until the 8th bin. This increase is outside the null bands, and the curve for the repress subjects is also near the bands at the 8th bin, while the neutral subjects' line is at the lower band at the 9th bin suggesting increased HRV in enhance and repress subjects compared to neutral at this time.
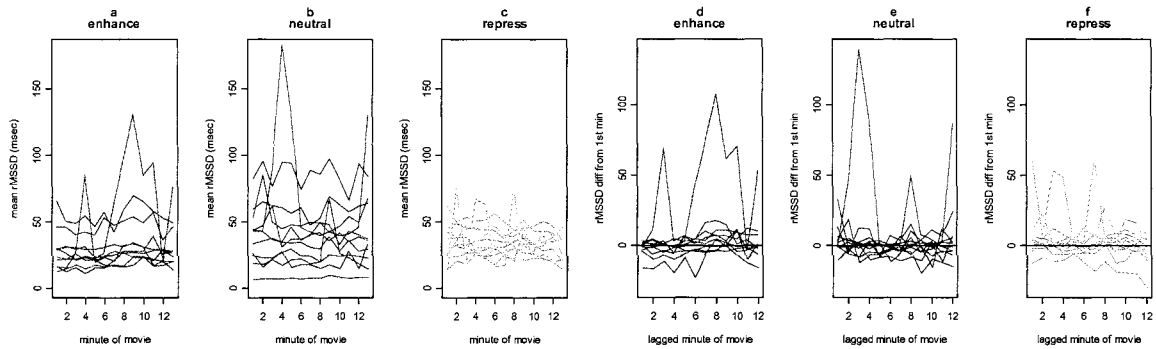
Figure 5.3. Actual rMSSD (msec) and rMSSD change from baseline, by condition. Pane a: mean rMSSD, enhance condition subjects. Pane b: mean rMSSD, neutral condition subjects. Pane c: mean rMSSD, repress condition subjects. Pane d: mean rMSSD change, enhance condition subjects. Pane e: mean rMSSD change, neutral condition subjects. Pane f: mean rMSSD change, repress condition subjects.
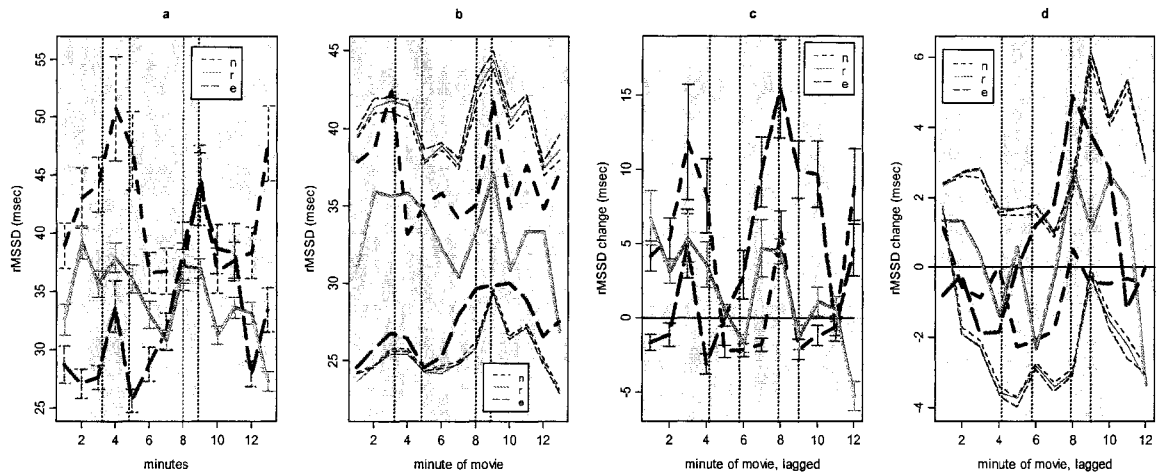
Figure 5.4. Means with bars indicating standard error of the mean and lowess curves with null bands. Panes a and b: mean rMSSD (msec). Panes c and d: mean rMSSD change.

Table 5.3. rMSSD (msec) mean, median, and standard deviation.

min	all subjects mean (sd)	mdn	enhance mean (sd)	mdn	neutral mean (sd)	mdn	repress mean (sd)	mdn
1	33.50 (17.55)	29.38	28.66 (15.95)	25.59	38.85 (20.99)	42.47	32.45 (14.84)	31.67
2	36.57 (21.06)	32.32	27.01 (12.76)	23.91	42.96 (28.65)	42.09	39.11 (15.68)	36.21
3	36.01 (18.80)	30.81	27.54 (10.24)	26.17	44.15 (26.20)	37.17	35.51 (12.23)	35.07
4	41.02 (32.90)	30.71	33.68 (22.09)	26.74	50.64 (49.76)	33.98	37.78 (13.59)	32.17
5	36.53 (24.82)	29.29	25.55 (10.07)	21.98	47.02 (36.79)	36.05	35.97 (13.38)	33.20
6	32.81 (15.83)	30.19	28.65 (14.67)	23.93	36.52 (19.44)	35.77	32.90 (12.80)	30.71
7	33.09 (16.44)	30.28	31.55 (15.80)	26.03	36.66 (21.95)	39.05	30.69 (9.57)	28.57
8	37.47 (20.61)	32.25	38.43 (24.82)	29.48	36.99 (22.34)	39.32	37.04 (15.61)	34.36
9	41.93 (24.55)	37.35	44.07 (34.29)	30.65	44.57 (25.24)	41.81	36.88 (9.16)	38.32
10	35.50 (17.75)	30.86	38.61 (21.19)	29.29	36.66 (21.20)	32.49	31.12 (8.22)	30.16
11	36.52 (17.79)	34.14	38.32 (24.82)	28.62	37.62 (17.07)	35.23	33.49 (10.01)	32.74
12	33.20 (16.90)	29.05	27.86 (10.56)	26.94	38.24 (24.55)	38.82	33.00 (10.39)	36.04
13	36.42 (25.36)	28.70	33.34 (18.84)	27.09	47.65 (35.96)	35.55	27.13 (9.56)	27.49

RSA

The mean, standard deviation, and median RSA in each one-minute bin during the movie is listed in Table 5.4 and plotted in Figure 5.5 and Figure 5.6. As expected due to the high correlation between rMSSD and RSA, mean RSA plots for each subject (Figure 5.5 pane a, b, and c) are similar to those for mean rMSSD (Figure 5.3 pane a, b, and c). Subjects vary in baseline RSA as well as in RSA variability. Overall, neutral subjects had the most variability, followed by enhance subjects, with repress subjects the least. Mean (pane a) and lowess (pane b) curves for RSA by condition are shown in Figure 5.6. There is a rapid increase in mean RSA at the 8th bin, a pattern also present in the lowess curves and null bands. Mean and lowess curves for the neutral and repress subjects are similar throughout the movie, while the curve for the enhance subjects is comparatively lower. This difference is outside the null bands for the first 6 bins of the movie, and again at the 11th, suggesting that enhance subjects had a lower RSA during these period than would be expected if there was no interaction between condition and RSA.

As with the other measures, change curves were calculated to minimize the influence of individual baselines on the patterns by subtracting each subject's RSA in the first bin from their RSA in the remaining bins. Differences between subjects in different conditions are still present in this data, but less pronounced than in the raw data. The ordering of these mean and null bands (Figure 5.6 panes c and d) is different than in the raw data: enhance subjects have a larger RSA difference than the repress and neutral subjects after the 4th bin, and this increase is marginally outside the null bands. The curves for the neutral and repress subjects are still similar, although the neutral subjects' curve approaches the lower null band at several points.

To summarize, the RSA level was relatively stable during the first half of the movie but rose during the second, with a spike at the 9th minute (death scene). RSA levels were similar for neutral and repress condition subjects; enhance subjects had the greatest RSA increase.

81
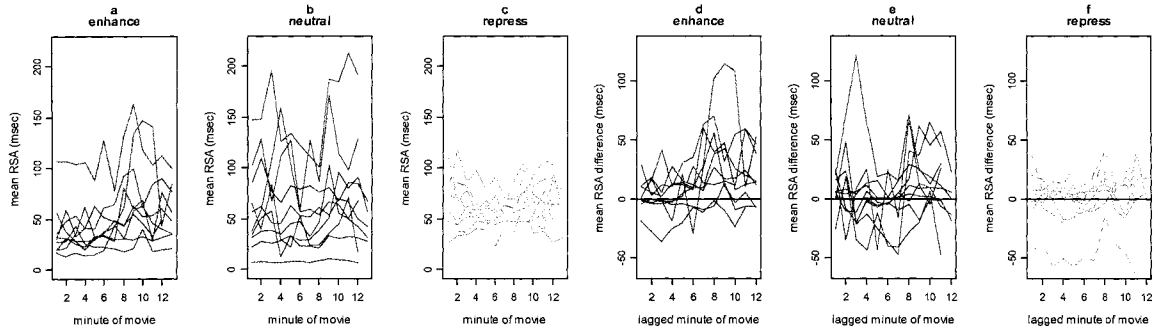
Figure 5.5. Actual RSA (msec) and RSA change from baseline, by condition. Pane a: mean RSA, enhance condition subjects. Pane b: mean RSA, neutral condition subjects. Pane c: mean RSA, repress condition subjects. Pane d: mean RSA change, enhance condition subjects. Pane e: mean RSA change, neutral condition subjects. Pane f: mean RSA change, repress condition subjects.
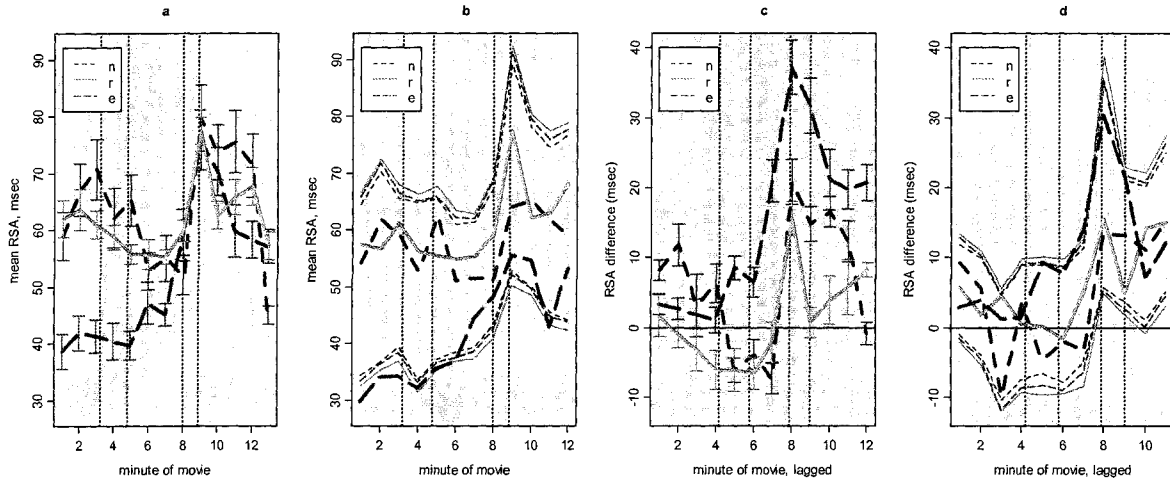
Figure 5.6. Means with bars indicating standard error of the mean and lowess curves with null bands. Panes a and b: mean RSA (msec). Panes c and d: mean RSA change.

Table 5.4. RSA (msec) mean, median, and standard deviation.

| min	all subjects		enhance		neutral		repress	
	mean (sd)	mdn	mean (sd)	mdn	mean (sd)	mdn	mean (sd)	mdn
1	52.95 (34.19)	40.85	38.55 (28.11)	29.84	58.82 (42.61)	47.13	61.81 (26.44)	61.05
2	57.54 (36.40)	46.71	41.84 (27.91)	31.01	66.95 (46.11)	54.28	63.44 (28.54)	57.12
3	57.89 (37.84)	51.72	41.22 (25.66)	33.10	70.58 (52.78)	62.13	60.79 (19.73)	59.41
4	53.73 (37.08)	45.10	40.38 (28.62)	28.22	61.93 (52.35)	40.11	58.50 (17.21)	54.12
5	53.80 (31.88)	44.99	39.60 (22.44)	32.53	65.10 (44.70)	53.94	55.65 (13.65)	54.07
6	51.66 (28.37)	49.85	47.10 (32.83)	36.26	52.63 (31.76)	52.85	55.58 (20.38)	52.83
7	51.70 (26.67)	49.07	45.08 (17.99)	44.45	54.89 (40.29)	41.98	55.18 (10.04)	53.45
8	56.60 (28.30)	56.82	59.53 (36.80)	44.75	51.50 (29.73)	49.78	59.67 (15.24)	59.07
9	77.61 (44.44)	66.88	75.74 (47.33)	59.78	79.50 (58.54)	60.47	77.36 (18.96)	77.86
10	69.15 (37.38)	62.82	70.20 (37.90)	62.46	73.57 (49.04)	65.70	62.43 (19.35)	60.40
11	67.24 (42.16)	59.02	59.66 (41.33)	45.89	75.36 (54.95)	68.85	65.64 (24.45)	62.46
12	65.75 (39.71)	60.41	58.22 (29.56)	48.87	71.12 (56.12)	58.70	67.50 (26.29)	64.79
13	53.49 (22.45)	54.76	57.04 (27.48)	52.17	44.90 (16.74)	42.72	57.11 (19.43)	59.89

Respiration Rate Variability

Mean Respiration Rate

The mean respiration rate in each one-minute bin is plotted in Figure 5.7 and Figure 5.8; the mean, median, and standard deviation of the respiration rate are listed in Table 5.5. Several patterns are visible in plots of subjects' respiration rate (panes a, b, and c of Figure 5.7): many subjects have an abrupt transitory decrease in respiration rate at the 6th bin, many subjects' respiration rate decreases after the 8th bin, and the amount of respiration rate variability varies by subject. The subjects' baseline respiration rate also varies, as expected. But while some subjects change their respiration rate only a moderate amount around their baseline, other subjects' respiration changes drastically, most commonly by decreasing in the second half of the movie. There is less variance among the repress condition subjects (lines are more tightly grouped together and more the same direction), and a higher proportion of the lines in the repress condition show a deceleration during the second part of the movie.

The overall mean curve (Figure 5.8 pane a) rises and falls several times in the first half of the movie, followed by a large decrease after the 7th bin and slight recovery at the final bin, with troughs at the 3rd and 6th minutes. This overall pattern is present in the curves for all three conditions, and the mean and lowess curves are similar. The neutral condition curve is closest to the overall curve, with the enhance curve above (faster respiration rate) and repress below (lower respiration rate). The enhance curve is at the null bands at several points (1st, 4th, 7th bin), as is the repress curve (8th, 9th bin), suggesting that the difference in respiration rate for those conditions at those points is slightly larger than expected. The MANOVA did not find a significant difference between the groups, however (condition: df=2, Wilks=0.22, F=1.25, p=0.29, residuals=24), although the intercept was significant (condition: df=1, Wilks=0.10, F=9.54, p<0.001, residuals=24).

Respiration change curves are plotted in panes d, e, and f of Figure 5.7, and pane c and d of Figure 5.8. The individual change curves (Figure 5.7) are rather similar to the raw scores: several peaks in the first half of the film followed by a general reduction. There appears to be more variation in the enhance subjects' respiration change curves throughout the recording, especially when compared to the repress subjects. The mean and lowess curves calculated for the change scores are similar to those for the raw scores, but the order of the curves is different: the enhance curve is now lowest and the repress highest; the neutral curve between them as before. The repress curve is mostly above zero for the first half of the movie (respiration rate increased from the first bin), while the neutral curve

straddles zero and the enhance curve is below (respiration rate decreased from the first bin). All curves are clearly below zero after the 7th bin, with the enhance curve outside the null band at the 9th bin. This graph suggests that the enhance subjects decreased their respiration rate more from baseline than subjects in the other conditions throughout the movie, but especially at the 8th and 9th bins (death scene).
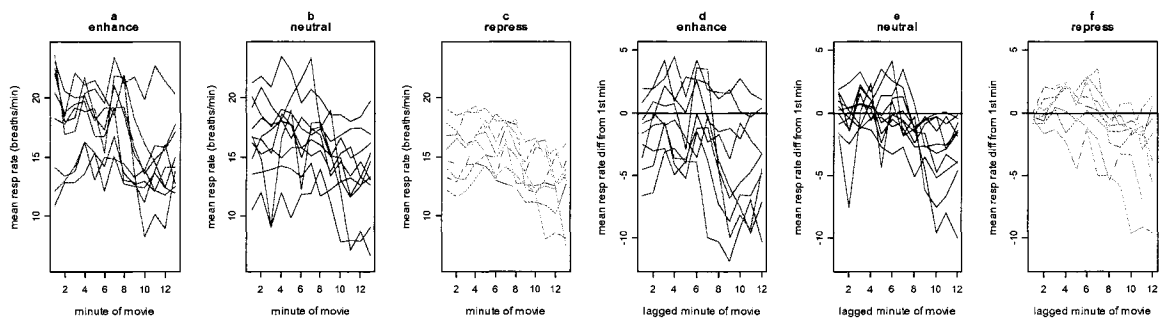
Figure 5.7. Actual respiration rate (breaths/minute) and respiration rate change from baseline, by condition. Pane a: mean respiration rate, enhance condition subjects. Pane b: mean respiration rate, neutral condition subjects. Pane c: mean respiration rate, repress condition subjects. Pane d: mean respiration rate change, enhance condition subjects. Pane e: mean respiration rate change, neutral condition subjects. Pane f: mean respiration rate change, repress condition subjects.
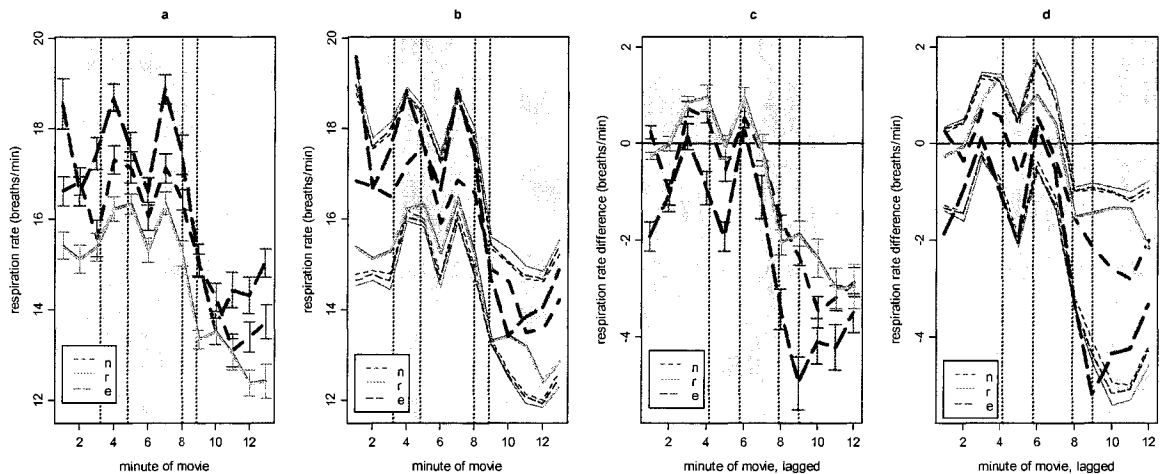
Figure 5.8. Means with bars indicating standard error of the mean and lowess curves with null bands. Panes a and b: mean respiration rate (breaths/minute). Panes c and d: mean respiration rate change.

Table 5.5. Respiration rate (breaths/minute) mean, median, and standard deviation.

min	all subjects mean (sd)	mdn	enhance mean (sd)	mdn	neutral mean (sd)	mdn	repress mean (sd)	mdn
1	16.88 (3.78)	16.63	18.53 (4.96)	20.41	16.59 (3.20)	16.60	15.38 (2.36)	15.15
2	16.23 (2.84)	16.69	16.60 (2.87)	17.73	16.81 (3.09)	16.58	15.09 (2.49)	15.41
3	16.12 (3.34)	16.03	17.44 (3.20)	18.27	15.55 (3.97)	16.48	15.34 (2.49)	15.01
4	17.43 (2.82)	17.96	18.67 (2.71)	19.67	17.29 (3.15)	18.02	16.20 (2.13)	16.51
5	17.03 (2.75)	17.37	17.60 (2.73)	18.10	17.11 (3.44)	17.64	16.31 (1.84)	16.22
6	16.00 (2.71)	15.72	16.60 (2.70)	16.74	16.04 (3.19)	15.75	15.28 (2.18)	15.02
7	17.46 (2.91)	17.37	18.86 (2.96)	19.12	17.10 (3.21)	17.04	16.32 (2.01)	16.96
8	16.38 (2.96)	16.85	17.41 (3.92)	18.71	16.40 (2.32)	17.11	15.21 (2.26)	15.29
9	14.50 (2.55)	13.62	15.09 (3.15)	13.62	14.93 (2.50)	14.99	13.31 (1.56)	13.15
10	13.79 (2.80)	13.59	13.56 (3.20)	13.59	14.24 (3.03)	14.73	13.48 (2.27)	12.28
11	13.50 (3.31)	13.32	14.43 (3.55)	13.80	13.08 (3.67)	13.25	12.99 (2.72)	12.86
12	13.38 (3.14)	12.65	14.30 (3.54)	14.13	13.37 (3.36)	13.21	12.35 (2.32)	12.30
13	13.75 (3.33)	13.78	15.02 (2.82)	14.08	13.70 (3.80)	13.93	12.39 (3.04)	13.20

rMSSDresp

rMSSDresp mean, standard deviation, and median is listed in Table 5.6 and shown in Figure 5.9 (panes a, b, and c) and Figure 5.10 (panes and b). Changes in rMSSDresp from baseline are shown in Figure 5.9 (panes d, e, and f and Figure 5.10 (panes c and d). rMSSDresp estimates short term variation in respiration rate and is calculated in the same way as rMSSD, but on the respiration instead of the heart rate series.

Raw rMSSDresp scores for each subject are shown separated by condition in Figure 5.9. There is a large amount of variability in these scores, even within individual subjects, which makes finding trends difficult. The rMSSDresp (Table 5.6) for all subjects combined has a relatively steady median during the movie, with the lowest value at the 7th minute (bride collapses and in hospital). The mean and lowess curves (Figure 5.10 panes a and b) for the enhance condition subjects peaks above that of the neutral and repress subjects at the 2nd and 5th minutes; the 2nd minute peak is well outside the null bands while the 5th minute peak is at the null bands. These times correspond to happy scenes in the movie; the enhance subjects may have been attempting to laugh or otherwise strongly react to the situations. During the second half of the movie the lowess and mean curves for all groups overlap. There is a noticeable drop in rMSSDresp in all groups at the 7th minute (bride collapses). The MANOVA did not find a significant difference between the groups (condition: df=2, Wilks=0.17, F=1.5, p=0.14, residuals=24), although the intercept was significant (condition: df=1, Wilks=0.24, F=3.42, p=0.02, residuals=24).

There are also large differences between subjects in rMSSDresp change scores (Figure 5.9 panes d, e, and f). Several subjects had their largest rMSSDresp in the first

minute of the movie, so their rMSSDresp was negative for the remainder of the recording; several other subjects had very large (more than 2000 msec) change scores. As with the raw scores, there was more variation in the enhance subjects' change scores, while repress subjects had more variation in the second half of the movie than the first. A drop in rMSSDresp is present at the 6th bin (7th minute, bride's collapse) in all conditions (Figure 5.10 panes c and d). The curves overlap during the remainder of the movie; only the initial high rMSSDresp (1st bin, 2nd minute) is outside the null bands, consistent with what was seen in the raw scores.
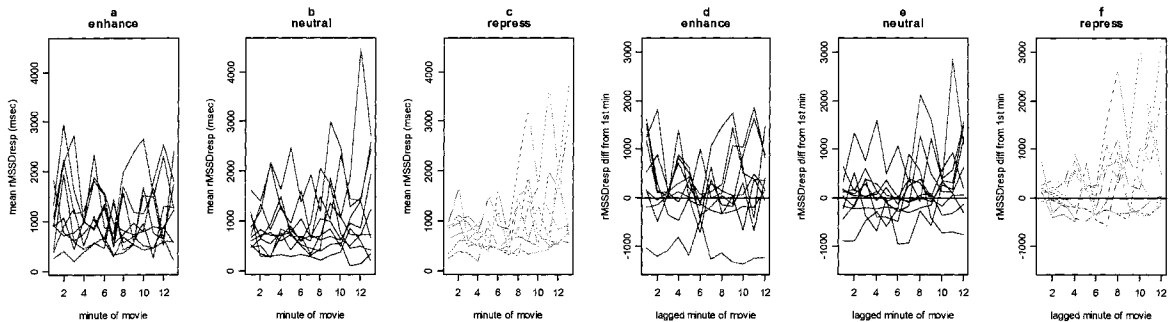
Figure 5.9. Actual rMSSDresp (msec) and rMSSDresp change from baseline, by condition. Pane a: mean rMSSDresp, enhance condition subjects. Pane b: mean rMSSDresp, neutral condition subjects. Pane c: mean rMSSDresp, repress condition subjects. Pane d: mean rMSSDresp change, enhance condition subjects. Pane e: mean rMSSDresp change, neutral condition subjects. Pane f: mean rMSSDresp change, repress condition subjects.
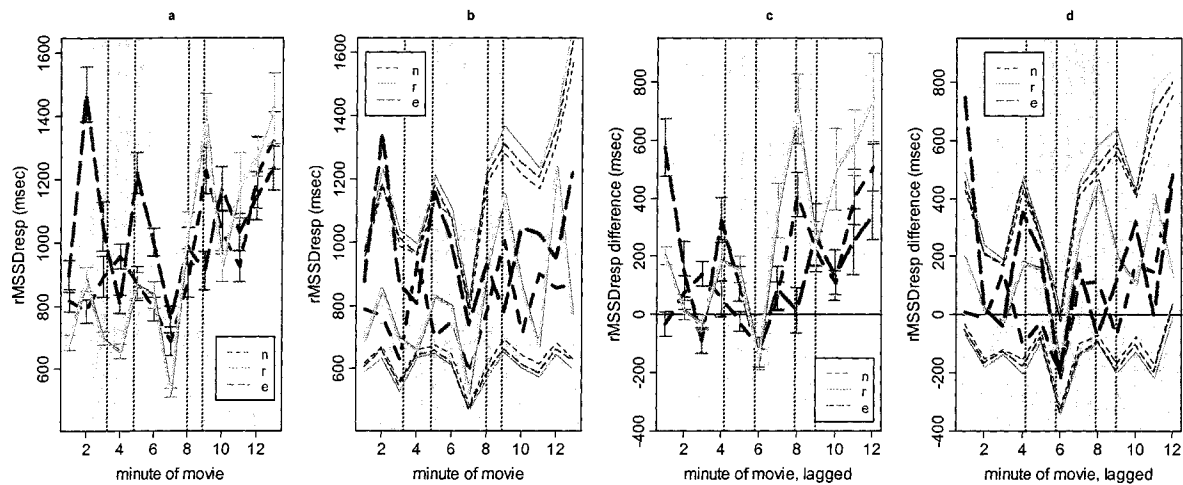
Figure 5.10. Means with bars indicating standard error of the mean and lowess curves with null bands. Panes a and b: mean rMSSDresp (msec). Panes c and d: mean rMSSDresp change.

Table 5.6. rMSSDresp (msec) mean, median, and standard deviation.

min	all subjects		enhance		neutral		repress	
	mean (sd)	mdn	mean (sd)	mdn	mean (sd)	mdn	mean (sd)	mdn
1	801.84 (390.42)	860.64	894.83 (496.50)	923.88	815.09 (377.31)	683.67	680.66 (270.63)	764.30
2	1039.47 (657.60)	817.47	1468.40 (875.70)	1077.66	780.13 (400.96)	673.63	881.10 (402.25)	846.35
3	884.22 (631.54)	699.54	1050.02 (778.86)	781.69	888.29 (712.91)	730.41	692.62 (239.52)	652.33
4	814.48 (350.57)	764.98	804.54 (288.94)	955.77	953.91 (432.79)	791.57	651.37 (248.88)	674.02
5	983.78 (575.73)	828.50	1219.68 (638.36)	1031.35	868.18 (595.56)	747.08	862.90 (449.52)	742.41
6	871.92 (427.74)	689.26	1000.84 (438.88)	888.93	788.41 (410.81)	646.47	831.29 (457.41)	687.26
7	660.78 (393.16)	588.05	757.24 (425.24)	619.48	685.17 (486.43)	626.69	521.78 (168.39)	513.01
8	959.70 (582.23)	735.75	986.80 (602.63)	735.75	875.48 (569.56)	691.77	1034.48 (639.94)	859.82
9	1167.18 (777.44)	960.65	910.72 (600.73)	695.20	1224.74 (797.08)	993.10	1383.75 (934.22)	1370.58
10	1043.95 (660.45)	798.27	1169.74 (698.53)	959.86	1029.06 (753.56)	682.14	921.06 (542.67)	709.49
11	1034.49 (750.18)	858.01	1030.88 (586.97)	889.01	922.28 (527.36)	905.02	1178.80 (1138.93)	621.50
12	1208.90 (912.12)	856.64	1146.13 (763.21)	872.03	1216.90 (1222.10)	787.67	1269.52 (703.46)	1237.65
13	1321.44 (952.53)	902.38	1234.42 (695.48)	1266.31	1318.75 (998.65)	976.44	1422.70 (1230.24)	850.73

Electrodermal Activity (EDA)

The number of NS-SCRs and mean SCL in each one-minute bin are shown in Figure 5.11, Figure 5.12, and Figure 5.13, and listed in Table 5.7 and Table 5.8. Difference scores were not calculated, unlike for the other measures, because the baseline values did not vary between subjects to the same degree. In general, there was more variation in NS-SCR rate than SCL for subjects in all conditions, and subjects in the neutral condition had reduced EDA compared to enhance and repress subjects, which were similar (Figure 5.11).

The mean, median, and lowess curves with null bands are plotted in Figure 5.12 for mean SCL and Figure 5.13 for NS-SCR count. Similar differences for both measures are present between the conditions. Subjects in the neutral condition had reduced mean SCL throughout the movie, slightly decreasing as the movie progressed (below the null bands after the 4th minute). The enhance and repress curves are near each other, crossing twice:

repress subjects have a higher SCL initially, then the enhance subjects are higher from the 3rd until the 5th minute, then the repress subjects are higher from the 7th minute until the end. The repress curve is just outside the null bands after the 9th minute (death and funeral), while the enhance curve is outside the null bands from the 4th until the 5th minute (wedding reception and leaving wedding). The MANOVA did not find a significant difference between the conditions (df=2, Wilks=0.07, F=1.86, p=0.10, residuals=19), nor a significant intercept (df=1, Wilks=0.36, F=1.19, p=0.41, residuals=19). The lack of significant intercept suggests that the curves did not significantly change height during the movie.

The patterns present in the NS-SCR count curves in Figure 5.13 are similar to those for mean SCL, but not identical. In particular, there is a large increase in the number of NS-SCRs in repress subjects at the 9th minute of the movie (death scene). This increase is outside the null bands, although the repress curve is otherwise within the null bands. The enhance curve has a spike at the 12th (end of funeral), and a relative increase at the 5th minute (leaving wedding reception) which approach the null bands. The curve for the neutral subjects is at the lower null band throughout the movie; paralleling SCL, the neutral subjects had a relatively lower NS-SCR count throughout the movie. The MANOVA did not find a significant difference between the conditions (df=2, Wilks=0.22, F=0.77, p=0.73, residuals=19), but the intercept was marginally significant (df=1, Wilks=0.18, F=3.09, p=0.06, residuals=19).

In general, EDA (both mean SCL and NS-SCR count) was reduced in neutral condition subjects compared to enhance and repress condition subjects. The repress subjects had an EDA increase at the 9th minute of the movie, corresponding to the death scene. The enhance condition subjects had an EDA increase during the early part of the movie, corresponding to the wedding reception scenes. These differences suggest that the enhance and repress subjects were more aroused than the neutral subjects, with the repress subjects becoming particularly aroused during the saddest part of the movie.
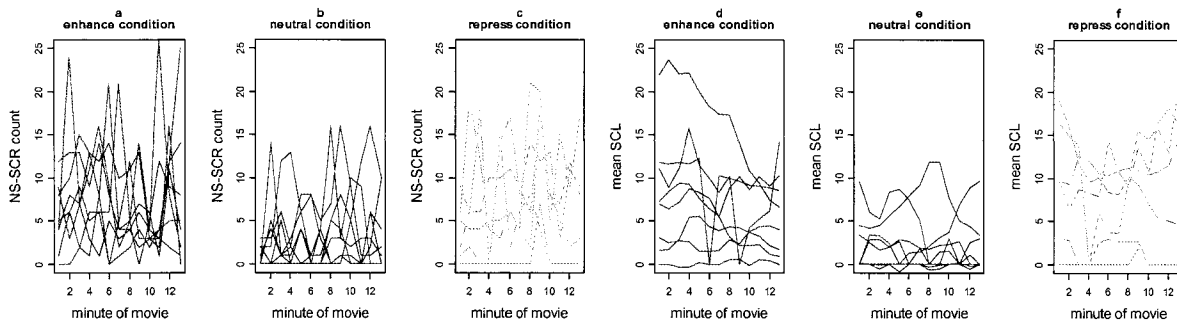
Figure 5.11. EDA measures, by condition. Pane a: mean NS-SCR count, enhance condition subjects. Pane b: mean NS-SCR count, neutral condition subjects. Pane c: mean NS-SCR count, repress condition subjects. Pane d: mean SCL, enhance condition subjects. Pane e: mean SCL, neutral condition subjects. Pane f: mean SCL, repress condition subjects.
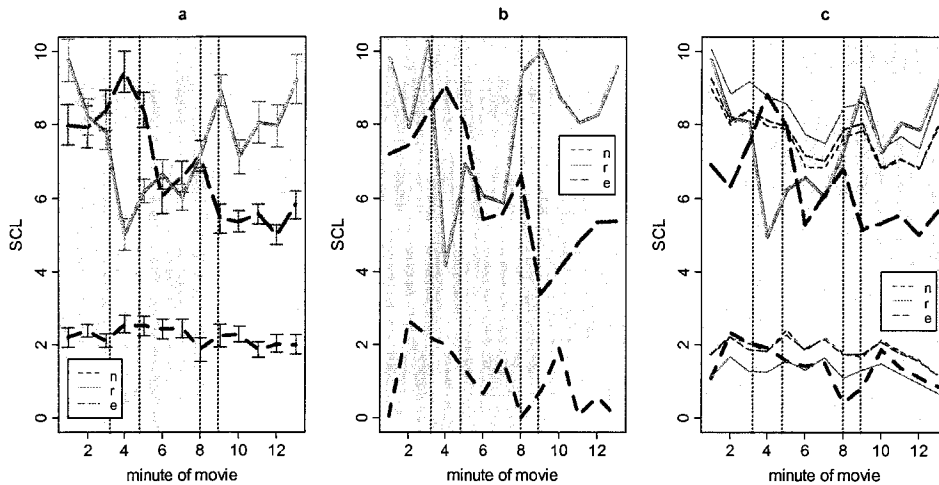
Figure 5.12. Pane a: mean SCL (µS), bars give standard error of the mean. Pane b: median SCL (µS). Pane c: SCL (µS), lowess curves with null bands.
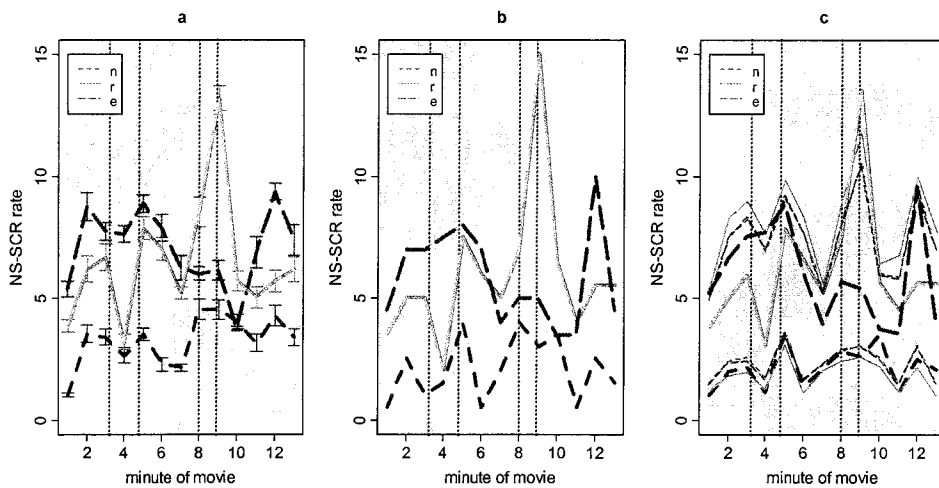
Figure 5.13. Pane a: mean NS-SCR count, bars give standard error of the mean. Pane b: median NS-SCR count. Pane c: NS-SCR count, lowess curves with null bands.

Table 5.7. SCL (µS) mean, median, and standard deviation.

min	all subjects mean (sd)	mdn	enhance mean (sd)	mdn	neutral mean (sd)	mdn	repress mean (sd)	mdn
1	6.36 (6.69)	3.90	7.99 (7.05)	7.20	2.18 (3.48)	0.03	9.75 (7.46)	9.80
2	5.98 (6.18)	3.79	7.93 (7.54)	7.45	2.35 (2.22)	2.64	8.21 (6.41)	7.91
3	5.94 (6.04)	3.87	8.39 (7.04)	8.32	2.09 (2.24)	2.22	7.80 (6.27)	10.27
4	5.72 (6.10)	4.05	9.44 (7.23)	9.09	2.54 (3.01)	1.96	4.98 (5.60)	4.12
5	5.64 (5.35)	5.75	8.40 (6.36)	8.01	2.49 (3.52)	1.29	6.17 (4.22)	6.92
6	4.87 (5.20)	3.38	6.03 (6.17)	5.42	2.40 (3.38)	0.65	6.62 (5.33)	6.03
7	4.90 (4.72)	3.74	6.58 (5.49)	5.56	2.41 (3.20)	1.60	5.99 (4.58)	5.84
8	5.22 (5.28)	2.97	7.14 (5.48)	6.60	1.84 (4.17)	0.00	7.17 (4.67)	9.41
9	5.21 (5.43)	2.53	5.42 (5.19)	3.36	2.22 (4.10)	0.68	8.91 (5.63)	10.01
10	4.70 (4.45)	3.76	5.35 (3.83)	4.04	2.26 (2.61)	1.87	7.11 (5.99)	8.74
11	4.88 (5.15)	3.82	5.55 (3.61)	4.79	1.82 (2.74)	0.04	8.05 (7.40)	8.03
12	4.71 (5.15)	3.43	4.98 (3.45)	5.34	2.00 (3.24)	0.55	7.95 (7.47)	8.27
13	5.34 (6.25)	3.17	5.81 (5.00)	5.36	1.97 (3.41)	0.00	9.23 (8.73)	9.58

Table 5.8. NS-SCR count mean, median, and standard deviation.

min	all subjects mean (sd)	mdn	enhance mean (sd)	mdn	neutral mean (sd)	mdn	repress mean (sd)	mdn
1	3.36 (3.54)	2.00	5.38 (4.07)	4.50	1.00 (1.20)	0.50	3.83 (3.43)	3.50
2	6.14 (6.42)	4.50	8.75 (7.34)	7.00	3.50 (4.66)	2.50	6.17 (6.71)	5.00
3	5.86 (5.36)	5.50	7.75 (4.62)	7.00	3.38 (4.10)	1.00	6.67 (7.12)	5.00
4	4.59 (4.76)	3.00	7.62 (4.57)	7.50	2.62 (4.34)	1.50	3.17 (4.02)	2.00
5	6.64 (5.12)	6.00	8.88 (4.88)	8.00	3.50 (3.16)	4.00	7.83 (6.11)	7.50
6	5.59 (6.06)	5.50	7.88 (7.04)	7.00	2.25 (3.58)	0.50	7.00 (6.20)	6.00
7	4.45 (4.66)	4.00	6.25 (6.54)	4.00	2.12 (2.10)	2.00	5.17 (3.25)	5.00
8	6.14 (5.57)	5.00	6.00 (3.55)	5.00	4.50 (5.37)	4.00	8.50 (7.84)	7.00
9	7.45 (6.43)	5.00	6.12 (5.06)	5.00	4.50 (5.26)	3.00	13.17 (6.59)	15.00
10	4.36 (3.54)	4.00	3.75 (1.28)	3.50	4.00 (3.93)	3.50	5.67 (5.09)	6.50
11	5.00 (6.43)	3.00	6.88 (8.43)	3.50	3.12 (4.73)	0.50	5.00 (5.62)	4.00
12	6.50 (5.35)	6.00	9.38 (4.41)	10.00	4.25 (5.31)	2.50	5.67 (5.61)	5.50
13	5.59 (6.57)	3.50	7.38 (8.42)	4.50	3.38 (4.31)	1.50	6.17 (6.55)	5.50

Skin Temperature

The mean, median, and standard deviation of finger temperature by condition are listed in Table 5.9 and plotted in Figure 5.14 (pane a, b, and c), and Figure 5.15 (pane a and b); change scores are shown in Figure 5.14 (pane d, e, and f) and Figure 5.15 (pane c and d). Finger temperature changes slowly compared to the data examined previously so the curves are relatively smooth, though subjects still vary in baseline. Most subjects' finger temperature stayed relatively constant or decreased, increasing in only a few subjects. Several neutral subjects had colder fingers than the other subjects. These two subjects drew the mean skin temperature curve for neutral subjects lower than both the repress and

enhance subjects (Figure 5.15 pane a), while the neutral lowess curve is between the enhance and repress curves (Figure 5.15 pane b). All lowess curves are within the null bands, suggesting that they do not vary from what would be expected if there was no condition effect. Consistently, a MANOVA did not find a significant difference between the groups (condition: df=2, Wilks=0.57, F=0.46, p=0.97, residuals=28), although the intercept was significant (condition: df=1, Wilks=0.31, F= 3.08, p=0.017, residuals=24).

Examining the finger temperature change curves makes it easier to look for condition-related trends in the data, since effects due to each subject's baseline are minimized. It can be seen in the temperature change curves for each subject (Figure 5.14 panes d, e, and f) that most neutral and repress subjects' finger temperature decreased substantially, but only three enhance subjects did; one neutral subjects' finger temperature increased substantially (this is the subject with the lowest baseline temperature, so may be a floor effect). The lowess curve (Figure 5.15 pane d) for the enhance subjects is at the zero line (no change) throughout the movie, while the curves for the neutral and repress subjects reduce substantially. The enhance curve is outside the null bands for the second half of the movie, so it differs from the neutral and repress curves more than would be expected without a condition effect.
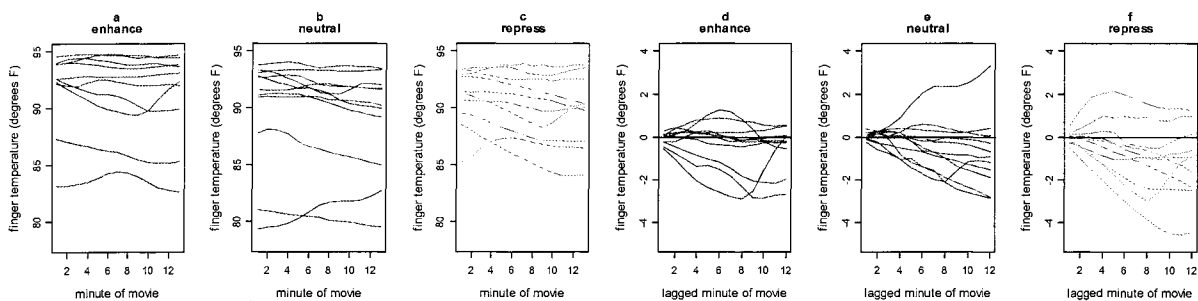
Figure 5.14. Actual finger temperature (degrees F) and finger temperature change from baseline, by condition. Pane a: mean finger temperature, enhance condition subjects. Pane b: mean finger temperature, neutral condition subjects. Pane c: mean finger temperature, repress condition subjects. Pane d: mean finger temperature change, enhance condition subjects. Pane e: mean finger temperature change, neutral condition subjects. Pane f: mean finger temperature change, repress condition subjects.
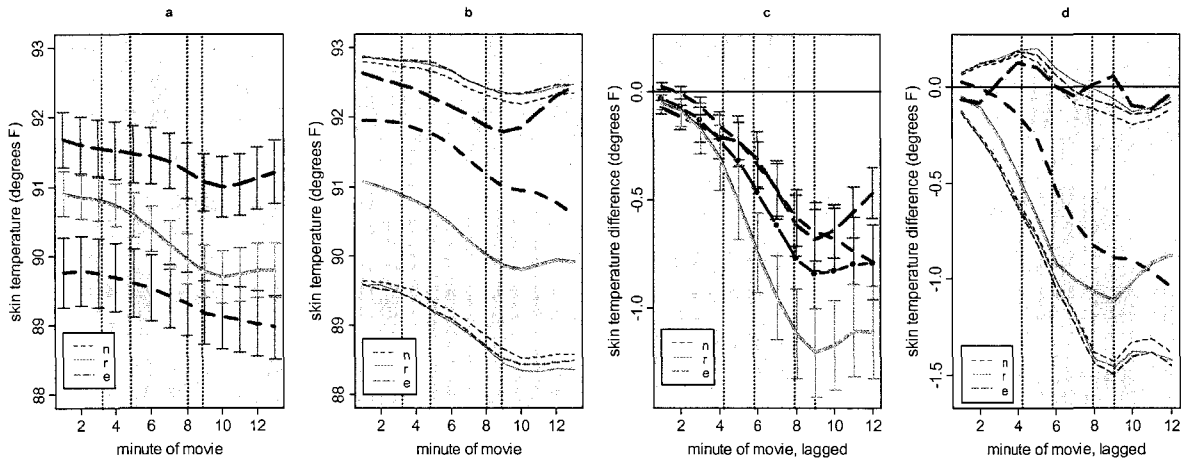
Figure 5.15. Means with bars indicating standard error of the mean and lowess curves with null bands. Panes a and b: mean finger temperature (degrees F). Panes c and d: mean finger temperature change.

Table 5.9. Finger temperature (degrees F) mean, median, and standard deviation.

min	all subjects mean (sd)	mdn	enhance mean (sd)	mdn	neutral mean (sd)	mdn	repress mean (sd)	mdn
1	90.74 (3.87)	92.33	91.68 (3.62)	92.60	89.75 (5.02)	91.58	90.90 (2.59)	91.33
2	90.71 (3.87)	91.99	91.60 (3.69)	92.43	89.78 (5.01)	91.54	90.84 (2.54)	91.19
3	90.67 (3.86)	91.85	91.56 (3.72)	92.37	89.74 (5.00)	91.54	90.81 (2.47)	91.11
4	90.61 (3.85)	91.62	91.52 (3.73)	92.58	89.68 (4.97)	91.62	90.72 (2.48)	90.96
5	90.52 (3.85)	91.73	91.47 (3.69)	92.63	89.59 (4.92)	91.73	90.59 (2.60)	90.89
6	90.42 (3.79)	91.76	91.44 (3.62)	92.65	89.52 (4.78)	91.76	90.38 (2.68)	90.76
7	90.28 (3.74)	91.39	91.36 (3.58)	92.58	89.41 (4.63)	91.39	90.15 (2.77)	90.55
8	90.13 (3.76)	91.19	91.22 (3.62)	92.48	89.30 (4.61)	91.19	89.94 (2.88)	90.39
9	89.97 (3.83)	90.80	91.06 (3.75)	92.41	89.17 (4.61)	90.66	89.77 (3.01)	90.26
10	89.90 (3.89)	90.64	90.99 (3.91)	92.44	89.10 (4.63)	90.64	89.69 (3.02)	90.16
11	89.91 (3.95)	90.77	91.03 (4.00)	92.47	89.07 (4.67)	90.53	89.72 (3.09)	90.08
12	89.94 (3.99)	90.60	91.12 (4.07)	92.53	89.02 (4.65)	90.40	89.79 (3.15)	90.09
13	89.95 (4.00)	90.37	91.21 (4.10)	92.73	88.96 (4.61)	90.22	89.78 (3.15)	90.18

Results: Changes Over Time

The previous permutation and MANOVA tests evaluated the physiological responses for differences between the conditions. An excursion of one of these lowess curves outside the null bands implied that the responses of subjects in that condition were different than expected for that interval; that there was an interaction between the physiological response and the emotion regulation condition. Another question can also be asked in relation to this data, namely, did the subjects' responses vary over time? Examination of the lowess curves

for each condition reveals many amplitude changes, but not whether the changes are larger than expected, given the range of values found for subjects in that condition.

The graphs in this section were constructed by permuting the time of each observation within each person, individually for each condition. These null bands indicate where the lowess curve for each conditions' subjects should lie if the curves do not systematically vary with time. Excursions of the curves beyond these bands indicates that the measure was higher (or lower) than expected at that time, given the variability in the measurements for subjects in that condition. The null bands vary in width for each condition because the number of subjects in each condition varies, and because the amount of variability is different for each condition and measure. The permutations were only done on the change curves (except for EDA, since change curves were not constructed for those measures) because these curves were the most informative in the previous analyses. Note that the lowess curves in these plots are the same as those seen previously; only the null bands changed.

Heart Rate Variability (HRV)

The null bands calculated by permuting each subject's data over time for the HRV measures are shown in Figure 5.16. The enhance condition subjects had a large decrease in HR and increase in both rMSSD and RSA at the 8th and 9th bin of the movie (death scene). The neutral condition subjects also had a HR reduction at the 9th bin, but the
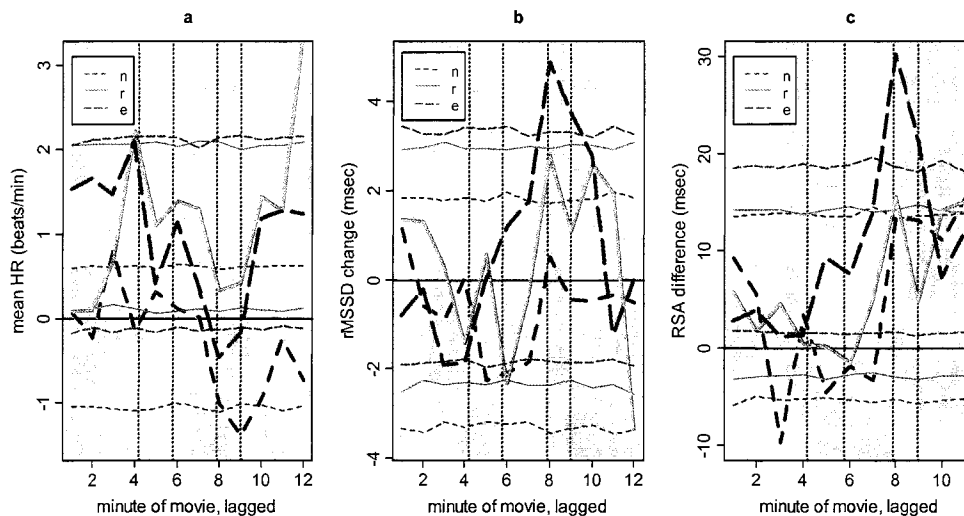
Figure 5.16. Null bands constructed for changes over time, HRV measures. Pane a: mean heart rate change. Pane b: rMSSD change. Pane c: RSA change.

increase in rMSSD and RSA did not exceed the bands. Neutral condition subjects had a HR increase and RSA decrease at the 3rd bin (dancing at reception). The curves for the repress condition subjects were mostly contained within the null bands, with only a few small excursions: HR increased at the 4th bin (leaving reception), rMSSD decreased at the 6th bin (bride's collapse), and RSA increased at the 8th bin (death scene).

Respiration Rate Variability

The null bands for the respiration data (Figure 5.17) reveal strong time-related trends in subjects' respiration. In all conditions respiration rate was increased during the first part of the movie (especially 3rd, 4th and 6th bin) and reduced during the second part of the movie (after the 8th bin). Subjects in all conditions also had a sharp decrease in rMSSDresp at the 6th bin (bride's collapse). This was the only excursion period for the enhance and neutral subjects, but the repress condition subjects had a modest excursion above the null bands at the 8th bin (death scene).

Electrodermal Activity and Skin Temperature

Several trends are present in the graphs examining the EDA data for changes over time (Figure 5.18, panes a and b). First, the line for neutral subjects is contained within the null bands for both the NS-SCR count and SCL (although it reaches the SCL bands at the
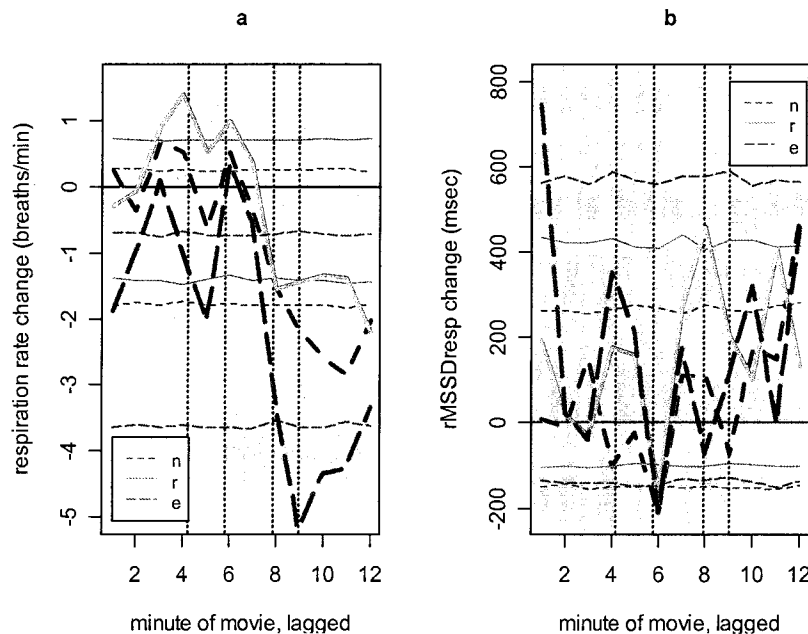
Figure 5.17. Null bands constructed for changes over time, respiration measures. Pane a: mean respiration rate change (breaths/minute). Pane b: rMSSDresp change (msec).
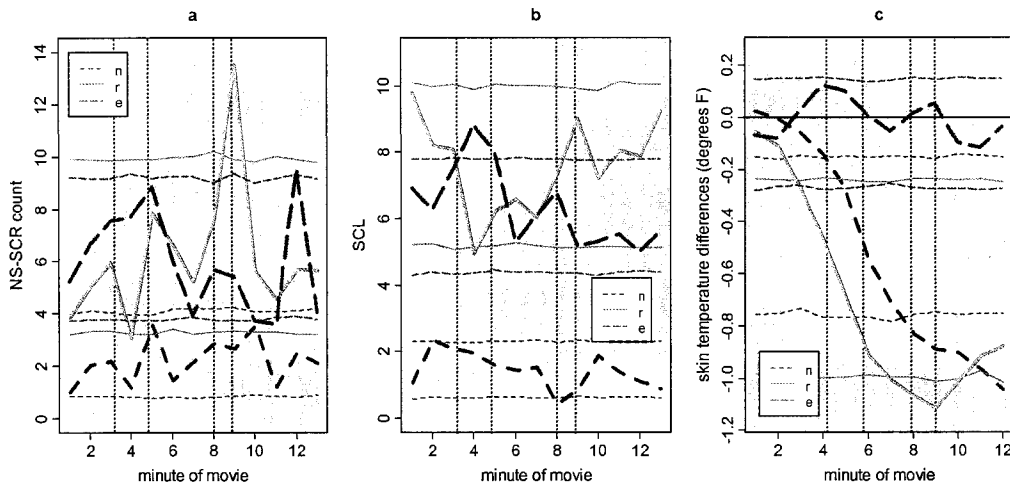
Figure 5.18. Null bands constructed for changes over time, electrodermal measures. Pane a: NS-SCR rate. Pane b: mean SCL. Pane c: skin temperature.

2nd and 8th minutes), suggesting that EDA did not substantially change during the course of the movie for neutral subjects. A strong peak in NS-SCR rate is present for repress subjects at the 9th minute (death scene), and a marginal decrease at the 4th minute (dancing/leaving wedding reception). In contrast, the SCL curve for repress subjects only reaches the null bands at the 4th minute. The NS-SCR count curve for the enhance subjects is primarily within the bands, reaching the lower band at the 10th and the upper at the 12th minute, while the SCL curve for the enhance subjects is clearly outside the curves at the 4th and 5th minutes.

The curves for changes over time in skin temperature (Figure 5.18, pane c) highlight differences between the conditions: the curve for the enhance subjects is completely within the bands while those for the neutral and enhance are initially above the bands then below, indicating a strong change in finger temperature over time, especially in the neutral subjects.

Discussion

These data and analyses suggest that physiological responses occurred in conjunction with particular scenes, and also that responses differed with condition. The design of the study allows the results to be interpreted in two ways: in terms of emotion regulation and in terms of emotional experience. Analysis in terms of emotion regulation is possible by comparing the physiological responses of subjects in the different conditions, while comparing the responses during the first and second halves of the movie within

subjects provides the opportunity to contrast the physiological states which occurred during induced happiness and sadness.

Condition-Related Findings

Emotion Regulation Techniques

In general, the emotion regulation literature has found that subjects attempting to regulate their emotions by suppressing outward signs of emotional state exhibit more physiological arousal (consistent with increased sympathetic activation) than subjects exposed to the same stimuli but not attempting to suppress their emotions. In contrast, subjects regulating their emotional responses using *reappraisal* methods (cognitively changing their interpretation of negative stimuli) do not exhibit increased physiological arousal (reviewed in Gross, 1998; Gross, 2002; Ochsner & Gross, 2005). The instructions in this study encouraged repress condition subjects to use reappraisal methods of emotion regulation; no reference to visible emotional response (as is done in studies of suppression) was given. According to emotion regulation theory, it would therefore be predicted that, in this experiment, repress condition subjects would exhibit physiological responses similar in magnitude to subjects in the neutral condition.

Few studies of emotion regulation include a condition comparable to the enhance condition used here (in which subjects were instructed to use reappraisal to increase, rather than decrease their emotional response). One example is Jackson et al. (2000); they did not measure cardiorespiratory or electrodermal activity, but found increased startle eyeblink magnitude and corrugator activity in enhance and reduced in suppress, relative to neutral trials. Another is Ochsner, et al. (2004), which used condition instructions similar to those used here but in a fMRI paradigm. Cardiorespiratory responses were measured by Demaree and colleagues (Demaree, et al., 2006), but the subjects were instructed to suppress or enhance facial signs of emotional response (disgust stimuli only), rather than to reappraise their responses; few condition-related HRV differences and no respiratory differences were reported.

Observed Patterns by Condition

Overall, the physiological responses of neutral subjects tended to be most consistent. Their EDA was comparatively reduced and constant, suggesting a fairly steady level of arousal. Neutral subject's HR was also comparatively level, fluctuating around zero during the first half of the movie then decreasing. Changes in neutral subjects' respiration

rate also fluctuated around zero during the first half of the movie, but increased more than expected at the 4th-5th minutes (wedding reception). This increase may be due to induced happiness or to entrainment of respiration to the prominent rhythm present during this scene. Another transitory respiration rate increase occurred during the 7th minute (bride's collapse), followed by a steady decrease in respiration rate until the end of the film. rMSSDresp dropped substantially during the 7th minute, but was consistent otherwise. The 7th minute was also marked by an increase in respiration rate, suggesting that subjects' breaths became quicker and more regular during this scene. Neutral subjects' HRV was fairly consistent throughout the film, but HRV differences (HR increase and RSA decrease) did occur at the 4th minute, (end of wedding reception). The respiration rate increased during this period; these HRV changes may be due to the respiration changes or to positive emotions. HR also decreased during the period from the 9th until the 11th minutes (death scene, driving to funeral), corresponding with the period in which respiration rate decreased. Finally, neutral subjects experienced a steady drop in finger temperature.

Subjects in the repress condition had physiological responses largely similar to those just described for the neutral subjects, with the notable exception of EDA. Repress condition subjects had higher EDA than neutral subjects throughout the movie, with a large increase in NS-SCR rate at the 9th minute (death scene). This sudden spike may indicate an increase in arousal and mental activity as subjects attempted to reappraise their responses during this emotional scene. Also, repress subjects had a marginal dip in EDA with an increase in respiration and HR at the 4th minute of the movie (end of reception), perhaps indicating sympathetic withdrawal or driving of responses by entrainment. Repress subjects largely were the same as neutral subjects on rMSSD, RSA (sharing the marginal peak at the 9th minute but lacking the dip at the 4th), respiration rate (sharing the increase/decrease pattern), and finger temperature (steady decrease). Like neutral subjects, repress subjects had a reduction in rMSSDresp at the 7th minute, although it was accompanied by a reduction in rMSSD not present in the neutral subjects.

Enhance condition subjects exhibited responses suggestive of an increase in arousal and physiological reactivity compared to neutral subjects. When averaged over the entire movie, enhance subjects had a higher NS-SCR rate than neutral subjects, and a marginally higher SCL. Additionally, enhance subjects failed to show the reduction in finger temperature during the second half of the movie that was seen in neutral and repress subjects; enhance subjects' finger temperature remained relatively constant. Enhance subjects exhibited the HR decrease at the 9th minute (death scene) seen in the neutral and repress subjects, but combined this with a larger increase in rMSSD and RSA. While

subjects in all conditions showed a reduction in respiration rate during the second half of the movie, this reduction was larger in the enhance subjects, especially during the death scene. Deep breathing may have caused the increase in NS-SCR rate, RSA, and rMSSD at the 12th minute of the movie, since deep breathing can serve to increase sympathetic activity.

Discussion

From the emotion regulation literature it was predicted that subjects' physiological responding would vary little between conditions. Instead, condition-related differences in responding were found: both enhance and repress condition subjects appeared to be more physiologically aroused than neutral condition subjects, with the enhance condition subjects differing more than the repress. This finding may be explained by the increased mental effort required to comply with the instructions for these conditions: subjects had to actively distance themselves from the situations portrayed (suppress condition) or imagine themselves experiencing the situations (enhance condition).

The presence of condition-related physiological differences in this study contradicts the results of Gross (1998). The addition of a condition in which subjects were instructed to suppress only their external reaction to the movie (paralleling his *suppress* condition) may help clarify this difference; it would be predicted that subjects in this new condition would exhibit more physiological arousal than subjects in the current experiment. Also, Gross used two short movies of medical procedures to elicit disgust, rather than the longer movie used here, which was intended to induce happiness and sadness. It may be that regulating responses to that type of disgust is cognitively different than regulating emotional responses to the more ordinary (e.g. wedding, illness, death) but nevertheless intense situations portrayed in the movie used in this experiment.

Emotion-Related Findings

Respiration

A relatively small literature has examined the specificity of respiratory patterns to emotional states (reviewed in Boiten, Frijda, & Wientjes, 1994; Cacioppo, Berntson, Larsen, Poehlmann, & Ito, 2000; Wientjes, 1992). Many reported relationships include patterns of changing respiration depth, which was not recorded here; including respiration depth (or a related measure, such as end-tidal CO_2) in this study would have allowed more detailed interpretation. In general, relationships have been reported between respiration and the amount of excitement and arousal, rather than with emotional valance. Increases in

respiration rate have been reported during states of excitement (such as anxiety, fear, stress, and tenseness), including participation in a stressful mental task, while decreased respiration rate has been reported during states of sadness, depression, and relaxation (Boiten, Frijda, & Wientjes, 1994). Additionally, increased respiratory irregularity has been associated with anxiety and emotional upset (Boiten, Frijda, & Wientjes, 1994).

These previous respiratory findings are consistent with what was found here. In particular, subjects in the arousal condition had a pronounced decrease in respiration rate (greater than that observed in the other conditions) during the second half of the film, particularly during the death scene. It has been reported that subjects voluntarily decrease respiration rate in response to a stressful situation; subjects in the enhance condition may have slowed their respiration to reduce anxiety they felt at the thought of a family member suffering in the way depicted in the film. Alternatively, they may have purposely decreased their respiration rate to depress their mood in an attempt to follow the instructions. Decreases in respiration rate were also observed in subjects in the repress and neutral conditions. These subjects may have slowed their respiration for the same reasons as the enhance subjects, or perhaps in association with a state of passive grief created by the film (Boiten, Frijda, & Wientjes, 1994). Yet another alternative explanation is simply that the subjects were highly aroused at the beginning of the experiment (due to tenseness at participating in an experiment) and became more relaxed (and so their respiration slowed) as the experiment progressed. This explanation would not account for the differences between conditions, however, or for the timing of the decrease (the respiration rate began to decrease during the death scene).

Many subjects had a respiration rate increase in during the wedding reception scene. This may be caused by positive affect, consistent with previous reports of increased respiration rate associated with happiness (Boiten, Frijda, & Wientjes, 1994), but may also be due to entrainment, since the music during the scene had a very strong, fast beat. Entrainment needs to be ruled out before the observed increase in respiration rate during this period can be accurately interpreted.

The amount of respiration irregularity has been associated with the level of anxiety: more irregularity associated with increasing anxiety (Boiten, Frijda, & Wientjes, 1994). In this data the measure of short-term respiration variability, rMSSDresp, does not fit that pattern. Instead, there was an abrupt, consistent drop in rMSSDresp during the scene in which the bride collapsed, the scene which marked the transition from an upbeat to sad movie and should have elicited tension or surprise. The respiration rate increased during this scene,

however, so it may be that the drop in rMSSDresp was caused by the increased respiration rate (consistent with an increase in tension) rather than a decrease in anxiety.

Heart Rate Variability

A number of studies have looked for different patterns of cardiovascular activity associated with emotional states (reviewed in Cacioppo, Berntson, Larsen, Poehlmann, & Ito, 2000; Levenson, 1992). The most consistent finding may be that HR is greater in emotional (happiness, sadness, anger, fear; disgust/blood may be an exception) than neutral states (e.g. Levenson, 1992; Palomba, Sarlo, Angrilli, Mini, & Stegagno, 2000; Prkachin, Williams-Avery, Zwaal, & Mills, 1999; Rainville, Bechara, Naqvi, & Damasio, in press; Sinha, Lovallo, & Parsons, 1992). Relatively few studies have examined HRV patterns associated with emotions other than fear (discussed in Levenson, 1992; Rainville, Bechara, Naqvi, & Damasio, in press).

These previous findings suggest that we should have expected to find increasing HR during the movie (since emotional intensity increases as the movie progresses), or that HR should have transitorily increased during especially intense scenes (such as the death or collapse scenes). In general, this is not what was found. There was a transitory HR increase around the time of the collapse scene in the enhance and repress condition subjects, but this increase was less than that observed during the wedding reception. The HR decreased in all conditions during the death scene, then increased again, remaining below baseline in the neutral subjects. These HR changes may have been driven by the concurrent respiration rate decrease during the second half of the movie. The amount of short-term HRV (rMSSD and RSA) was relatively constant in neutral and repress subjects, but increased sharply in enhance subjects at the death scene (it also increased in repress and neutral condition subjects, but to a lesser degree).

EDA

As reviewed in Chapter 1 of this dissertation, tonic EDA is used as a sign of general arousal and alertness, increasing with alertness and decreasing with relaxation. The EDA patterns observed in this study are consistent with that interpretation: EDA was increased in both enhance and repress condition subjects compared to neutral subjects; repress condition subjects had a large increase in EDA during the death scene, while enhance subjects had an EDA increase at the wedding reception scene. The increased EDA in the repress and enhance condition subjects relative to the neutral may be related to task difficulty; the instructions for these conditions required the subjects to actively consider and

manipulate their cognitions. The increased EDA at the death scene in the repress subjects is also consistent with this interpretation; this scene is highly emotional and would require effort to reappraise. It is surprising that the enhance condition subjects did not also exhibit an increase during this scene; they did show increased NS-SCR rate a few minutes later during the funeral scene, and had large increases in HRV during the death scene. The increased SCL in enhance subjects during the wedding reception may reflect a response to the physical activity depicted in the scene, or activation due to pleasant emotions evoked by the scene. Enhance subjects' HR was also at its maximum during this time period, consistent with increased arousal.

Skin Temperature

Previous studies of finger temperature (reviewed in the Measures and Quantification section of this chapter) suggest that decreases in finger temperature should correlated with movie scenes causing stress (such as the collapse and death scene), and perhaps be larger in enhance and repress than neutral subjects, since these subjects were given explicit instructions to regulate their responses. Also, decreases in finger temperature should have been associated with changes in other physiological responses that index sympathetic nervous system activity, such as increased EDA and HR. This expected pattern of skin temperature changes was not observed. Instead, finger temperature decreased throughout the film in most neutral and repress condition subjects; the decrease leveled off in several subjects towards the end of the film. The finger temperature was far more consistent in most enhance condition subjects, only two enhance condition subjects exhibited a pattern of decrease similar to that seen in the neutral subjects (see Figure 5.14).

It is not clear why skin temperature decreased steadily in repress and neutral condition subjects during the first (happy) part of the movie. Pronounced increases in EDA and HR, which may be associated with decreasing finger temperature, were not present during this period. The recovery in finger temperature seen in some subjects towards the end of the movie (during emotionally upsetting scenes) may reflect an increase in general negative affect. Enhance condition subjects had a distinctly different pattern of finger temperature changes, essentially remaining steady throughout the movie. This may reflect the effort required to follow the experimenter's instructions, or perhaps anger/negativity at personalizing the depicted scenes. Alternatively, perhaps these subjects had increased muscle tension (such as clenching their fists) that affected finger temperature.

Discussion

The movie was designed to induce two distinct emotions: happiness (first half) and sadness (second half). The valance and tone was not constant in either half; some scenes were especially intense. Many physiological changes occurred at either the death (8 - 10 minutes) or wedding reception scenes (3 - 5 minutes). The consistent drop in rMSSDresp spanned the bride's collapse scene (7 - 8 minutes), marking the transition from a happy to a sad movie (HR and respiratory rate increased during this period). The changes at the death scene, although varying with condition, can be characterized as decreases in respiration and heart rate combined with increases in EDA, RSA and rMSSD. The wedding reception scene was characterized by increases in respiration and heart rate, and a decrease in RSA; EDA increased in enhance and decreased in repress subjects.

While changes in physiological state were associated with particularly intense movie scenes, it is not possible to clearly match combinations of physiological measures to states of happiness or sadness. Part of this difficulty is due to the lack of a consensus in the literature as to the patterns of physiological response that are associated with particular emotional states, or even if unique collections of physiological response exist for each emotional state. One consistent finding in the literature, however, is that respiration rate is reduced during sadness compared to happiness inductions. This was also observed here: respiration rate was reduced during the second half of the movie, particularly in enhance condition subjects. In general, however (respiration rate and skin temperature are exceptions), distinct shifts in physiological state were associated with particular scenes, rather than between the film halves. This suggests that detailed analysis of the scenes provoking the largest transient changes may provide more consistent physiological response patterns, and highlights the likelihood of failing to find unique response patterns, even if they exist, when too large a segment of time is considered.

CHAPTER 6: TRANSIENT HEART RATE RESPONSES TO WORD VIEWING AND THEIR RELATIONSHIP TO RECALL

Abstract

This chapter expands the analysis of data from an experiment designed and conducted by Tony Buchanan at the University of Iowa described in *The influence of autonomic arousal and semantic relatedness on memory for emotional words* (Buchanan, Etzel, Adolphs, & Tranel, in press). The primary HR finding was replicated: recalled unpleasant words were associated with heart rate deceleration at encoding, while forgotten unpleasant words were associated with heart rate acceleration. Unexpectedly condition-related differences were found, and the marginal recall-related heart rate difference to taboo words was not replicated. While the overall heart rate response pattern is consistent with an evoked cardiac response, the presence of neither an evoked cardiac response nor a heart rate deceleration caused by an orienting response is sufficient to explain the recall or word type results.

Study Description

This chapter describes the findings from an experiment testing the influence of stress on memory for different types of words. The experiment was designed and conducted by Tony Buchanan at the University of Iowa and is described in *The influence of autonomic arousal and semantic relatedness on memory for emotional words* (Buchanan, Etzel, Adolphs, & Tranel, in press). In brief, each subject was presented 40 words, ten in each of four categories, on a computer monitor while their heart rates were recorded. Each subject completed several additional tasks, including one designed to cause stress, and was twice tested for their recall of the 40 words. This chapter describes the heart rate response patterns that occurred while the subjects were initially viewing the words, with special focus on relationships between heart rate changes, word type, and recall accuracy. The published analysis (Buchanan, Etzel, Adolphs, & Tranel, in press) includes data from the warm condition subjects. This chapter expands that treatment by considering the cold condition subjects as well. It was predicted that the same relationships between heart rate response, word type, and recall observed in the warm condition subjects would be found in the cold condition subjects.

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Stimuli and Protocol

The stimuli for this study consisted of words, which were presented individually to the subjects as slides on a computer screen. The words fell into four categories: taboo (t), school-related (s), unpleasant (u), and neutral (n). 80 words were selected, 20 in each of the four categories. Each subject was presented 40 words, ten of each type. The words presented to each subject were selected at random from the larger set, and the words were presented in random order.

The protocol consisted of several parts: viewing the words (encoding), a water task, a distractor task, and two (immediate and delayed) memory tests. All subjects completed all parts of the protocol, but the order varied. In all cases, the subjects completed a one hour distractor task between the immediate and delayed recall tests. In the *consolidation* version the water task came after the immediate recall test, before the distractor task, while in the *retrieval* version the water task came before the delayed recall test, after the distractor task. The water task, designed to induce stress, consisted of immersing one hand in water for three minutes. Subjects were assigned to either the *warm water* (room temperature water, control condition) or *cold pressor* (ice water, stress-inducing) version of the water test.

Subjects

49 subjects (23 females, 26 males) participated in the experiment, all normal undergraduate students at the University of Iowa. The number of subjects in each experiment and condition is shown in Figure 6.1. The subjects are split equally by gender and condition in the retrieval experiment, more males than females participated in the consolidation experiment, and more subjects of both genders were in the cold than the warm condition. The number of subjects in each condition and experiment is large enough to permit comparisons of gender, condition, and experiment effects.

Data Analysis Techniques

The mean heart rate change in consecutive 500 msec bins for five seconds following the presentation of each word was used as the measure of cardiovascular activity. The mean HR in the 500 msec bin immediately preceding the onset of the word stimulus was used as the baseline; the mean HR in each of the bins following the stimulus was subtracted from this baseline for HR change. The mean heart rate in each bin was calculated by taking the weighted mean of all RR intervals overlapping the bin after the RR intervals had been converted to heart rate in beats/minute. This procedure is similar to typical methods of
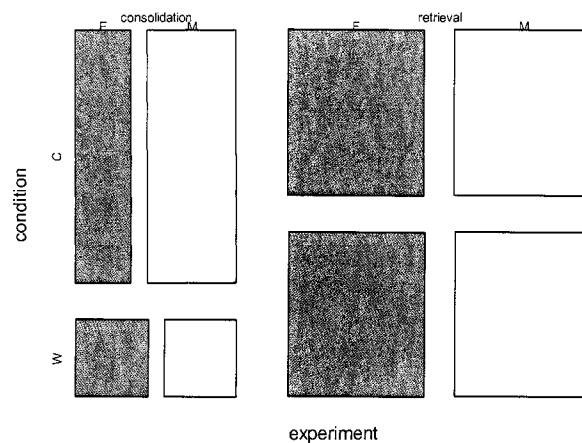
Figure 6.1. Proportion of subjects in each experiment and condition, by gender. 7 females were in the consolidation experiment, 5 in the cold condition and 2 in the warm. 10 males were in the consolidation condition, 8 in the cold condition and 2 in the warm. 16 males and 16 females were in the retrieval experiment, 8 of each gender in each condition.

quantifying the HR component of orienting responses (e.g. Binder, Barry, & Kaiser, 2005; Stekelenburg & van Boxtel, 2002) but rather than use the maximum change as the magnitude estimate HR change curves were calculated.

The pattern of HR changes in response to the stimuli was plotted using lowess curves to summarize responses within and between subjects, in the same manner as previously described. Significance was assessed by calculating null bands using permutation testing for each curve. The null bands for each curve were set at the 95% and 5% quantile lines resulting from lowess curves describing each of 2,000 permutations of the data set. Each permuted data set was created by randomly reassigning the label of interest (e.g. word category or recall accuracy) within each subject then calculating lowess curves in the same manner as for the true data. The null band width and location varies with both sample size and variability.

General Observations

To gain an impression of the overall heart rate changes the data was first examined by averaging over all data from all subjects. The mean, standard deviation, and median heart rate change at each time point for all data together is listed in Table 6.3 and plotted in Figure 6.2. The mean and median heart rate change show an initial deceleration followed by acceleration. The standard deviation is large and increases further over time, but is always larger than the mean and difference between the means. This pattern is most easily seen in
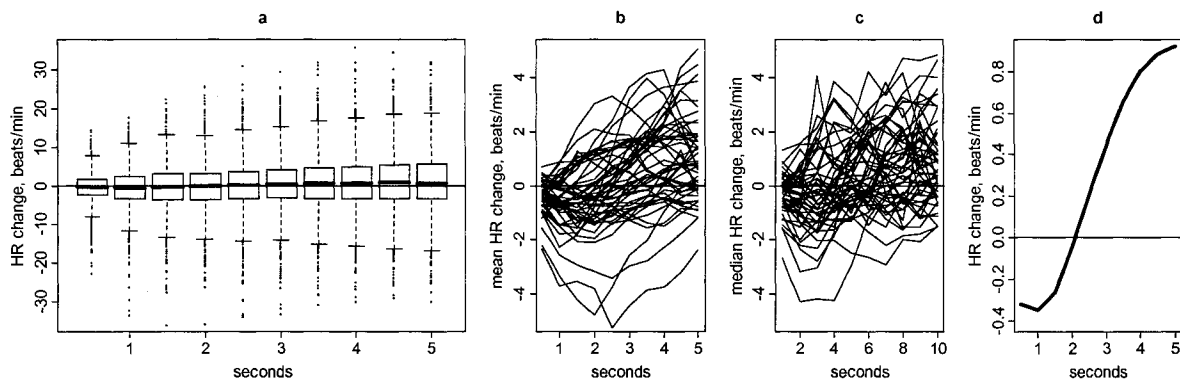
Figure 6.2. All subjects, all word types, conditions, and experiments. Pane a: boxplots of all data (one data point for each word for each subject). Pane b: mean heart rate change for each subject (averaged over all words). Pane c: median heart rate change lines for each subject. Pane d: lowess curve calculated over all data.

pane d of Figure 6.2: the lowess curve reflects an initial small heart rate deceleration, crosses zero at about two seconds, and then shows heart rate acceleration.

HR variability increases as time after stimulus onset increases. This trend can be seen in the boxplots in pane a of Figure 6.2 (the box and whisker width gradually increases) as well as the standard deviations in Table 6.3. The gradually increasing medians in this chart are consistent with the lowess curve in pane d. The overall amount of variation in each subjects' heart rate changes is high, as seen in the frequent direction changes in the lines in panes b and c of Figure 6.2.

Recall and Word Type

Subjects remembered words of different types at different rates, as shown in Table 6.1 and Figure 6.3. Taboo words were most often correctly recalled, followed by student, and unpleasant, with neutral words least often remembered. The proportion of words of each type remembered is similar at both immediate and delayed recall; subjects performed very similarly on the immediate and delayed recall tests.

Figure 6.4 shows the number of subjects remembering (pane a) and forgetting (pane b) various numbers of words at both immediate and delayed recall. The histogram in pane b is shifted to the right compared to the histogram in pane a: more subjects forgot words on both tests than remembered words on both tests. The histogram in pane c is the sum of the pane a and b histograms: it shows the number of words subjects performed identically on at both recall tests. Subjects changed their recall (i.e. remembered a word at immediate recall but forgot it at delayed recall) on nine or fewer (out of 40) words; one subject performed

identically at immediate and delayed recall. Since the results at immediate and delayed recall were so similar, the heart rate change curves should be very similar when calculated on immediate and delayed recall.

Recall, Gender, and Word Type

In addition to differences in recall rates due to word type, recall rates varied by gender, though gender differences are small in comparison to word type differences. The proportion of words correctly recalled at each time by gender and word type is shown in Figure 6.5 and listed in Table 6.2. Taboo words were most often remembered by both genders, followed by student, unpleasant, then neutral. Males more often correctly recalled the neutral and taboo words; the unpleasant and student words were remembered at about the same rate by both genders. The recall patterns were the same at delayed and immediate recall for both genders.
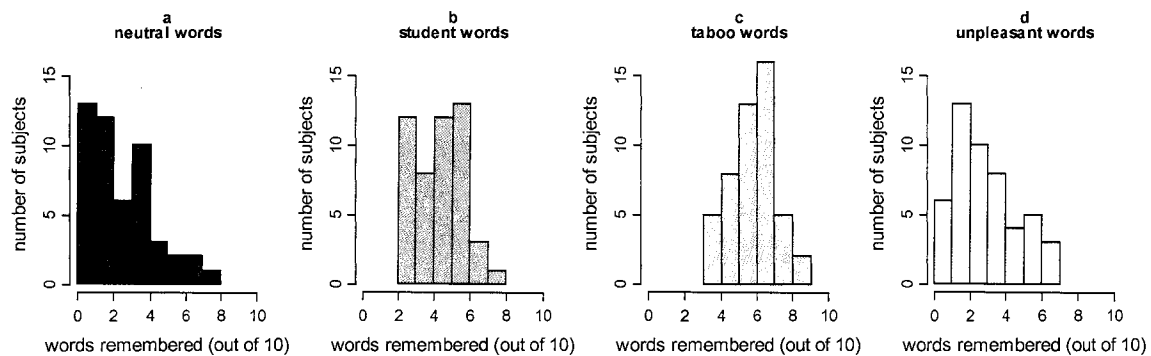
Figure 6.3. Frequency of remembered words at delayed recall by word type. Histograms indicate the number of subjects by number of words recalled. Pane a: neutral words, pane b: student-related words, pane c: taboo words, pane d: unpleasant words.

Table 6.1. Number of words correctly recalled or forgotten at immediate and delayed recall, by word type.

	immediate recall				delayed recall			
	forgotten		recalled		forgotten		recalled	
	#	proportion	#	proportion	#	proportion	#	proportion
neutral	332	0.68	158	0.32	348	0.71	142	0.29
student	233	0.48	257	0.52	257	0.52	233	0.48
taboo	159	0.32	331	0.68	183	0.37	307	0.63
unpleasant	299	0.61	191	0.39	328	0.67	162	0.33
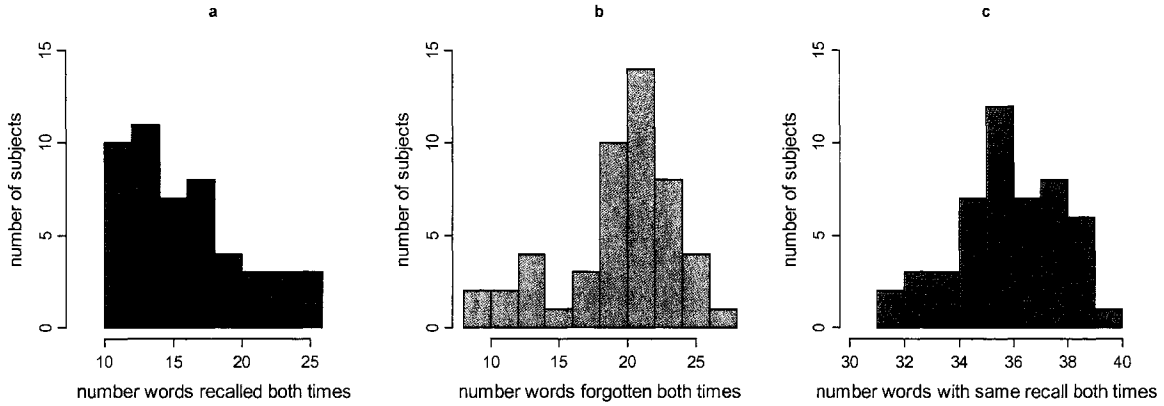
Figure 6.4. Frequency of performing identically at immediate and delayed recall. Pane a: number of subjects remembering words at both immediate and delayed recall, by number of words recalled. Pane b: number of subjects forgetting words at both immediate and delayed recall, by number of words forgotten. Pane c: number of subjects remembering or forgetting words at both immediate and delayed recall, by number of words (40 words viewed total).
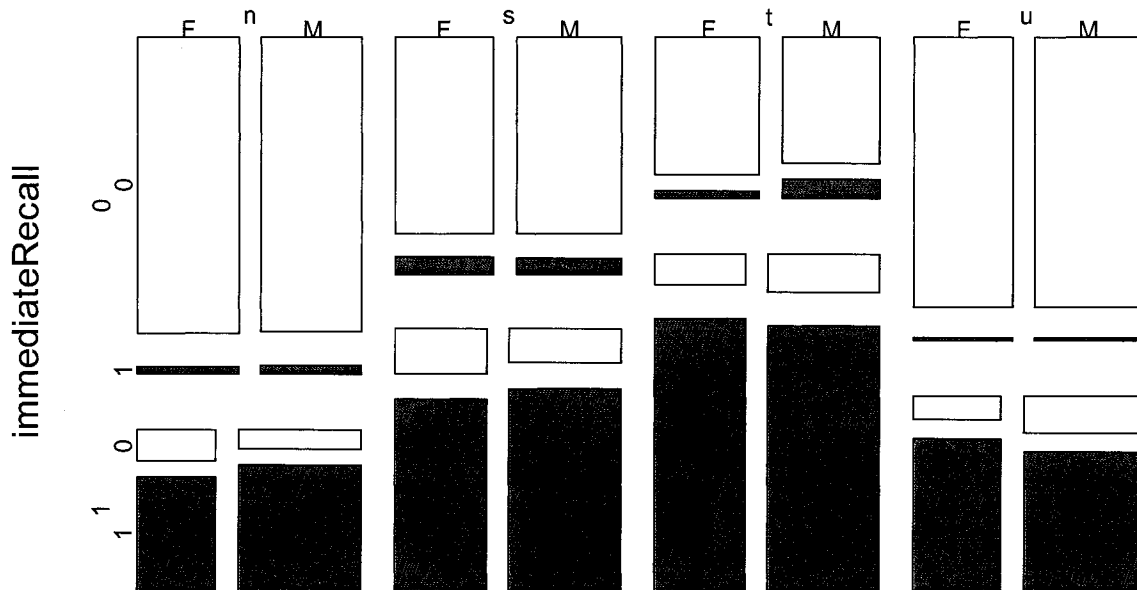
Figure 6.5. Proportion of words correctly (1) and incorrectly (0) recalled at delayed (inner y axis) and immediate recall (outer y axis), by word type (outer x axis) and gender (inner x axis).

Table 6.2. Number of words correctly recalled or forgotten at immediate and delayed recall, by word type and gender.

		immediate recall				delayed recall			
		forgotten		recalled		forgotten		recalled	
		#	prop.	#	prop.	#	prop.	#	prop.
males	neutral	164	0.63	96	0.37	171	0.66	89	0.34
	student	120	0.46	140	0.54	131	0.50	129	0.50
	taboo	77	0.30	183	0.70	90	0.35	170	0.65
	unpleasant	152	0.58	108	0.42	172	0.66	88	0.34
females	neutral	168	0.73	62	0.27	177	0.77	53	0.23
	student	113	0.49	117	0.51	126	0.55	104	0.45
	taboo	82	0.36	148	0.64	93	0.40	137	0.60
	unpleasant	147	0.64	83	0.36	156	0.68	74	0.32

Univariate Analyses

Univariate analyses were performed on the heart rate data to determine if the subjects' heart rate change patterns varied with any overall measure. For each univariate measure (gender, word type, condition, experiment, immediate recall, and delayed recall), the data were split on the values of that measure and heart rate change curves calculated for each section. On several measures (gender, condition, and experiment) each subject fell into only one category, allowing a MANOVA to be performed in addition to permutation testing. The MANOVA tests were performed on the averaged data for each subject (heart rate change in each bin averaged over all 40 words), so there was only one entry in the MANOVA test for each subject. This method of performing the test treats the mean heart rate change in each bin as a 10-element vector for each subject; the test determines if these profiles differ between groups.

Gender

Due to the emotionally-charged nature of the taboo stimuli it was predicted that male and female subjects may differ in their response to some stimuli, but no overall differences were found. The mean, standard deviation, and median change in each bin by gender is listed in Table 6.3 and plotted in Figure 6.6. The values and plots are similar, suggesting that males and females did not differ appreciably in heart rate changes when all word types, experiments, and conditions are combined. There is slightly more variation in the male than the female heart rate changes; the mean heart rate change curves for males (Figure 6.6, pane c) surround those for females, but this difference is not significant (pane d). Consistent with these observations, a MANOVA test did not find a significant difference between the
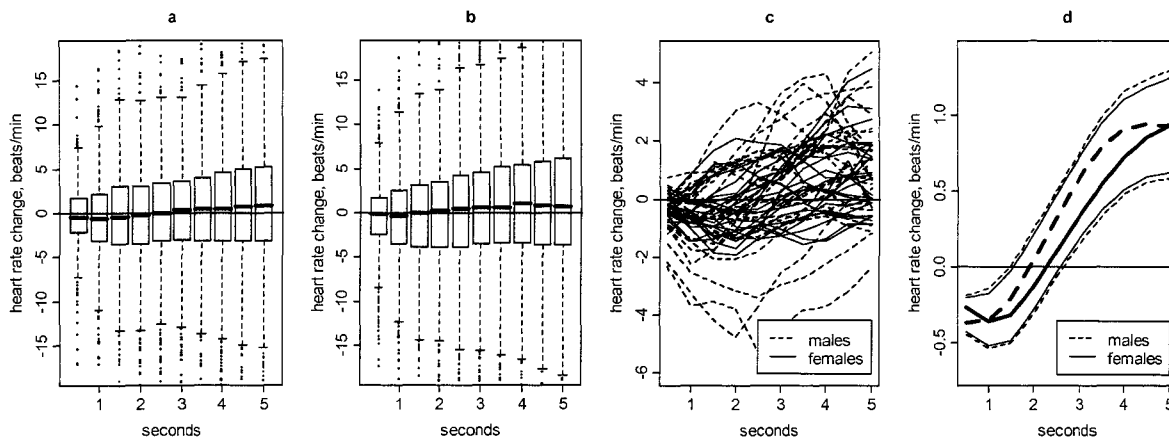
Figure 6.6. Mean HR changes by gender, all subjects combined. Pane a: boxplots of mean heart rate change in each bin, females. Pane b: boxplots of mean heart rate change in each bin, males. Pane c: mean heart rate change curves for each subject, colored by gender. Pane d: lowess curves and null bands for heart rate changes over time, by gender.

Table 6.3. Mean (standard deviation) and median of mean heart rate change in each 500 msec bin after the trigger, by gender.

bin	all subjects		males		females	
	mean (sd)	mdn	mean (sd)	mdn	mean (sd)	mdn
0	-0.38 (3.60)	-0.27	-0.46 (3.77)	-0.15	-0.29 (3.41)	-0.42
1	-0.59 (5.10)	-0.50	-0.67 (5.32)	-0.40	-0.50 (4.84)	-0.55
2	-0.50 (5.96)	-0.30	-0.50 (6.10)	-0.03	-0.50 (5.80)	-0.42
3	-0.36 (6.42)	-0.01	-0.28 (6.61)	0.28	-0.44 (6.20)	-0.28
4	-0.04 (6.73)	0.21	0.03 (7.16)	0.48	-0.11 (6.21)	0.06
5	0.34 (6.87)	0.44	0.48 (7.39)	0.59	0.19 (6.24)	0.32
6	0.59 (7.04)	0.58	0.82 (7.44)	0.60	0.34 (6.55)	0.53
7	0.83 (7.37)	0.71	1.07 (7.73)	1.09	0.55 (6.94)	0.54
8	0.99 (7.60)	0.85	1.16 (7.89)	0.90	0.80 (7.26)	0.78
9	0.95 (7.63)	0.77	1.07 (7.93)	0.74	0.82 (7.29)	0.80

genders (gender Df=1, Pillai=0.78, approx F=1.04, num Df=10, den Df=38, Pr(>F)=0.43; intercept Df=1, Pillai=0.39, approx F= 5.92, num Df=10, den Df=38, Pr(>F)<0.001; residuals=47). The intercept was highly significant, indicating that the curves are different than zero (a heart rate change is present).

Word Type

Heart rate changes while viewing words were similar for the different word types. The mean, standard deviation, and median heart rate change by word type are listed in Table 6.4 and shown in Figure 6.7. The null bands in pane d of Figure 6.7 overlap since each subject saw exactly ten words of each type. The median curves (pane c, calculated using all

ten words for the 49 subjects, 490 values per time point) are very similar, overlapping except for the curve for the neutral words around 3 seconds, which is slightly higher than the others. This difference is also present in the lowess curves (pane d); the n curve shows marginal heart rate acceleration between 2.5 and 3.5 seconds after stimulus onset. The general similarity in responses to words of different types can also be seen in individual subjects' responses (panes a and b); the approximate range and distribution of the responses is the same for all word types.

Figure 6.7. Curves showing the heart rate changes when observing words of each type. Pane a: mean heart rate change for n and s type words, averaged for each subject. Pane b: mean heart rate change for t and u type words, averaged for each subject. Pane c: median heart rate changes by word type. Pane d: lowess curves and null bands for heart rate change by word type.

Table 6.4. All subjects. Mean (standard deviation) and median of mean heart rate change in each 500 msec bin after the trigger, by word type.

bin	taboo mean (sd)	mdn	unpleasant mean (sd)	mdn	student mean (sd)	mdn	neutral mean (sd)	mdn
0	-0.43 (3.67)	-0.30	-0.27 (3.54)	-0.16	-0.58 (3.92)	-0.48	-0.25 (3.25)	-0.15
1	-0.79 (5.00)	-0.55	-0.55 (4.85)	-0.46	-0.61 (5.84)	-0.43	-0.40 (4.65)	-0.50
2	-0.83 (5.92)	-0.45	-0.54 (5.69)	-0.13	-0.44 (6.57)	-0.02	-0.18 (5.61)	-0.36
3	-0.71 (6.34)	-0.18	-0.38 (6.23)	-0.06	-0.37 (6.80)	-0.06	0.03 (6.31)	0.22
4	-0.18 (6.45)	-0.07	-0.03 (6.57)	0.10	-0.18 (6.99)	0.23	0.25 (6.89)	0.59
5	0.18 (6.59)	0.28	0.56 (6.75)	0.32	-0.14 (7.04)	0.04	0.77 (7.08)	0.81
6	0.52 (6.86)	0.53	0.87 (6.88)	0.58	0.11 (6.94)	0.25	0.88 (7.46)	0.90
7	0.78 (7.05)	0.55	1.14 (7.10)	0.81	0.43 (7.30)	0.41	0.96 (8.01)	0.93
8	0.91 (7.12)	0.70	1.30 (7.45)	1.00	0.74 (7.53)	0.93	1.01 (8.26)	0.85
9	0.79 (7.01)	0.50	1.25 (7.44)	1.06	0.92 (7.77)	0.98	0.85 (8.27)	0.42

Condition

Unexpectedly, small, but consistent, differences were found between subjects assigned to the warm and cold conditions. The mean, standard deviation, and median mean HR change in each bin by condition is given in Table 6.5; the data is plotted in Figure 6.8 and pane a of Figure 6.10. Subjects assigned to the cold water condition tended to have greater and earlier HR acceleration than subjects assigned to the warm water condition (lines outside the null bands in Figure 6.8 pane e). This difference is not large; boxplots of the distribution of the mean HR in each bin for both conditions (Figure 6.8, panes b and d) show that the interquartile ranges overlap. The MANOVA did not yield a significant condition effect (condition Df=1, Wilks=0.74, approx F=1.31, num Df=10, den Df=38, Pr(>F)=0.26), though the intercept was significant (intercept Df=1, Wilks=0.40, approx F=5.71, num Df=10, den Df=38, Pr(>F)<0.001). The difference in heart rate changes is not due to a difference in mean heart rate. Figure 6.9 shows the actual heart rate of the subjects at stimulus onset, separated by condition. It can be seen that the warm and cold condition subjects' heart rates are similar.

The presence of a difference in heart rate changes between subjects in the warm and cold water conditions is surprising because of the experiment design: the heart rate responses were recorded while subjects were viewing the word slides, before they had participated in the water portion of the experiment, and before they knew to which condition (warm or cold water) they were assigned. There is no reason that subjects assigned to the two conditions should have varied when initially viewing the word slides. This difference complicates the interpretation of later analyses; it may be difficult to separate experiment effects from other effects.

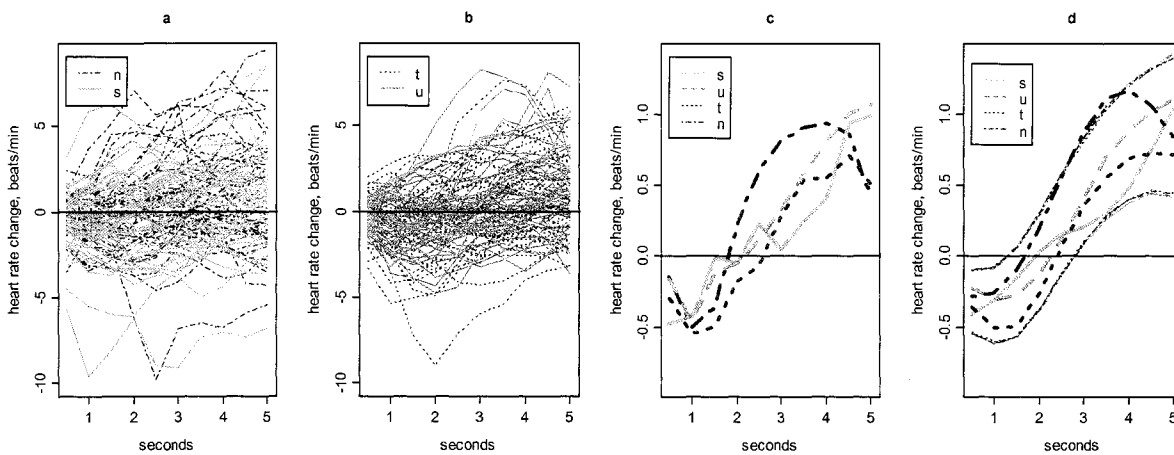Table 6.5. Mean (standard deviation) and median of mean heart rate change in each 500 msec bin after the trigger, by condition.

bin	warm only mean (sd)	mdn	cold only mean (sd)	mdn
0	-0.37 (3.11)	-0.30	-0.39 (3.91)	-0.22
1	-0.77 (4.41)	-0.65	-0.46 (5.53)	-0.36
2	-0.79 (4.93)	-0.51	-0.30 (6.57)	0.09
3	-0.73 (5.40)	-0.37	-0.10 (7.03)	0.30
4	-0.59 (5.93)	-0.26	0.34 (7.20)	0.75
5	-0.25 (6.20)	0.01	0.76 (7.27)	0.74
6	0.11 (6.50)	0.25	0.93 (7.38)	0.82
7	0.44 (6.93)	0.55	1.10 (7.66)	0.95
8	0.73 (7.20)	0.68	1.17 (7.86)	1.03
9	0.87 (7.28)	0.61	1.00 (7.87)	0.91
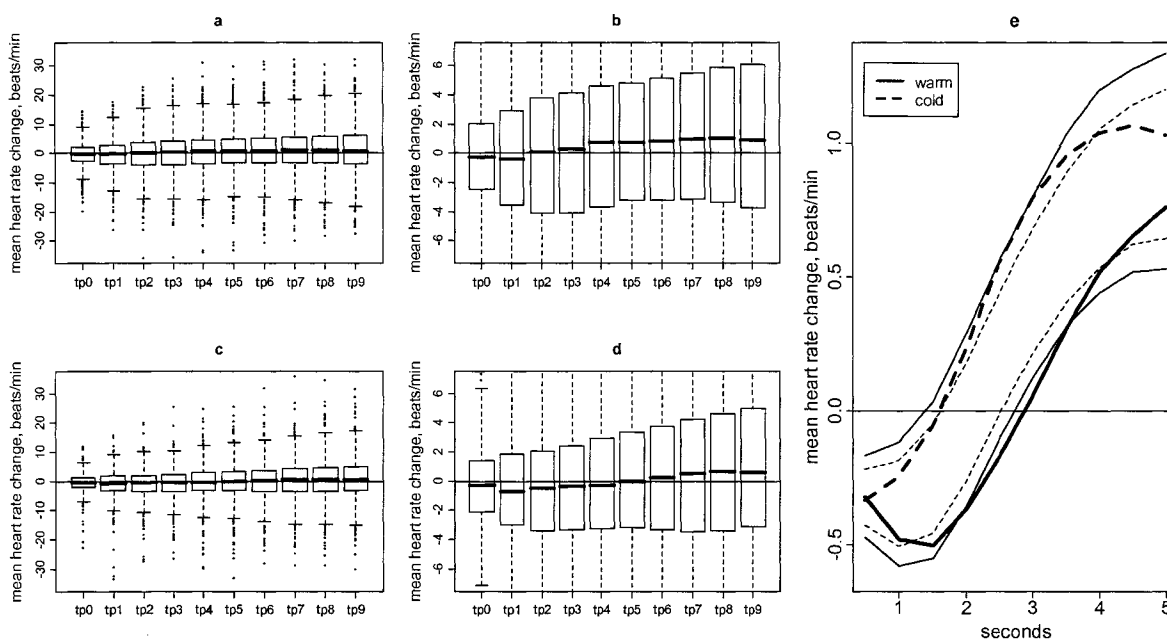
Figure 6.8. Data divided by condition, all subjects. Pane a: boxplots of heart rate responses in each bin, cold water condition subjects. Pane b: cold water condition subjects boxplots, zoomed view. Pane c: boxplots of heart rate responses in each bin, warm water condition subjects. Pane d: warm water condition subjects boxplots, zoomed view. Pane e: lowess curves for each condition with null bands.
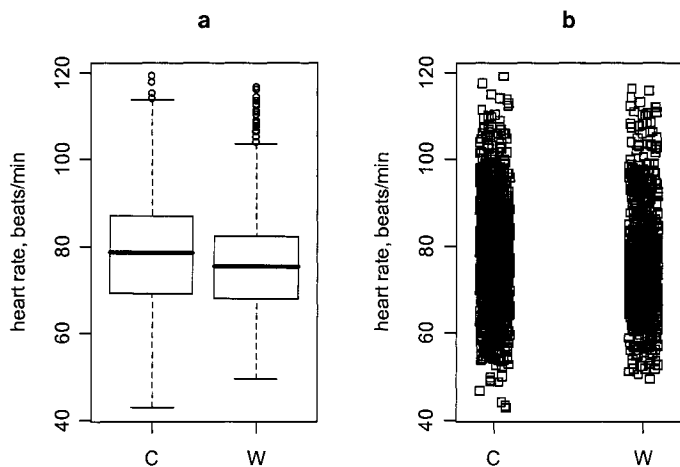
Figure 6.9. Actual heart rate (beats per minute), by condition.

Experiment

There is no evidence for a significant difference when the mean HR change in each bin is compared by experiment. The mean, standard deviation, and median in each bin by condition is listed in Table 6.6 and plotted in Figure 6.10, panes b and c. The mean and median increased slightly over time in both versions of the experiment, though by much less

than the standard deviation. The lowess curves (Figure 6.10, pane c) suggest that there is not a significant difference between the mean HR change in each bin in the consolidation and retrieval experiments. Consistently, the MANOVA did not find a significant experiment effect (experiment Df=1, Wilks=0.87, approx F=0.58, num Df=10, den Df=38, Pr(>F)=0.82) though the intercept was significant (intercept Df=1, Wilks=0.40, approx F=5.78, num Df=10, den Df=38, Pr(>F)<0.001).
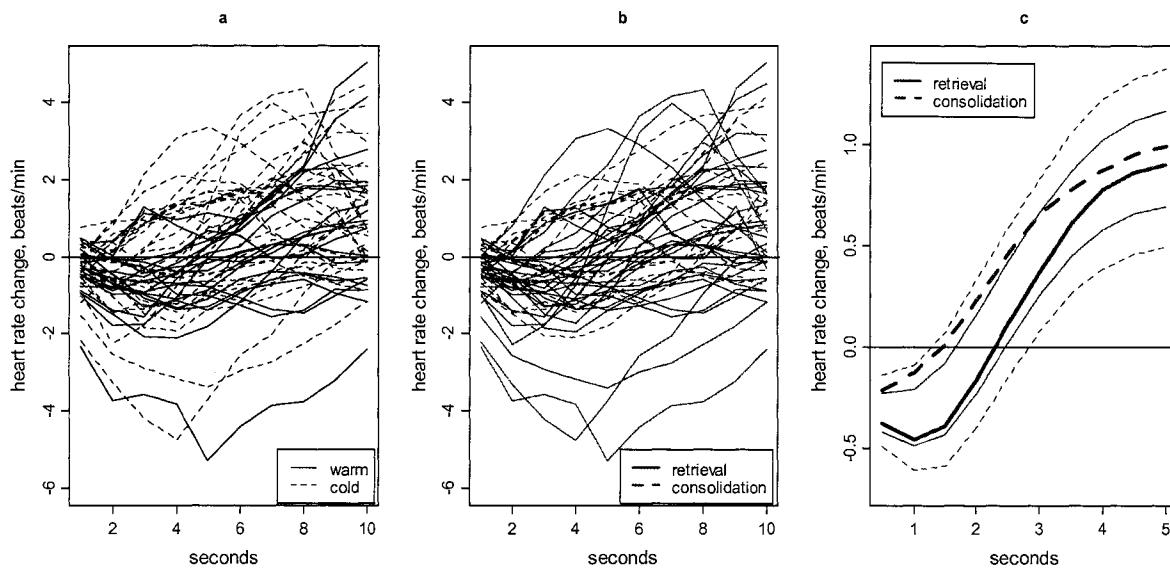
Figure 6.10. Curves showing HR changes by condition and experiment. Pane a: mean heart rate change lines for each subject, colored by condition. Pane b: mean heart rate change lines for each subject, colored by experiment. Pane c: lowess curves and null bands for heart rate changes by experiment.

Table 6.6. Mean (standard deviation) and median of mean heart rate change in each 500 msec bin after the trigger, by experiment.

bin	consolidation only		retrieval only	
	mean (sd)	mdn	mean (sd)	mdn
0	-0.22 (3.68)	-0.18	-0.47 (3.56)	-0.32
1	-0.23 (5.13)	-0.25	-0.78 (5.08)	-0.56
2	-0.01 (6.00)	0.14	-0.76 (5.92)	-0.44
3	0.14 (6.44)	0.28	-0.62 (6.40)	-0.17
4	0.48 (6.80)	0.48	-0.31 (6.67)	0.16
5	0.74 (7.15)	0.68	0.13 (6.71)	0.32
6	0.90 (7.35)	0.73	0.43 (6.87)	0.51
7	1.11 (7.73)	0.81	0.68 (7.18)	0.69
8	1.27 (7.97)	1.12	0.84 (7.39)	0.68
9	1.24 (7.98)	1.06	0.80 (7.44)	0.63

Recall

Subjects remembered an average of 20 words (half) at immediate recall, ranging from 13 to 30 words remembered (Figure 6.11 panes a and b). Therefore, at least ten words from each subject are included in each HR change curve when the curves were calculated based on accuracy at immediate recall (Table 6.7, Figure 6.11 panes c and d). The true lowess curves extend beyond the null bands for much of the time in pane d, suggesting that the HR accelerated somewhat more than expected while viewing words that were later forgotten, but decelerated more than expected when viewing words later remembered.

The mean, standard deviation, and median HR change in each bin is listed in Table 6.7 and plotted in Figure 6.12 for delayed recall performance. As expected given the large number of words either remembered at both delayed and immediate recall or forgotten at both delayed and immediate recall, the HR curves calculated for delayed recall are very similar to those calculated for immediate recall: the HR decelerated slightly more than expected while viewing words later remembered, and accelerated slightly more than expected while viewing words later forgotten (Figure 6.12, pane d), although the curves are closer to the null bands for delayed recall.
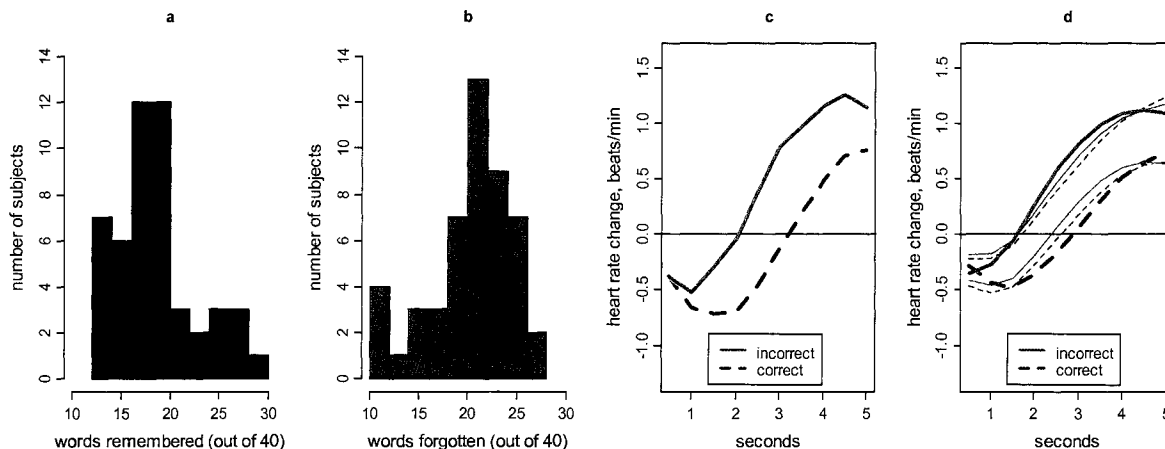
Figure 6.11. Immediate recall. Pane a: histogram showing the number of subjects by number of words correctly recalled. Pane b: histogram showing the number of subjects by number of words forgotten. Pane c: mean heart rate change in each bin recorded while viewing words later recalled or forgotten. Pane d: lowess curves and null bands for heart rate change in each bin recorded while viewing words later recalled or forgotten, null bands calculated by permuting immediate recall labels within each subject. Null bands vary due to the different number of words recalled and forgotten.
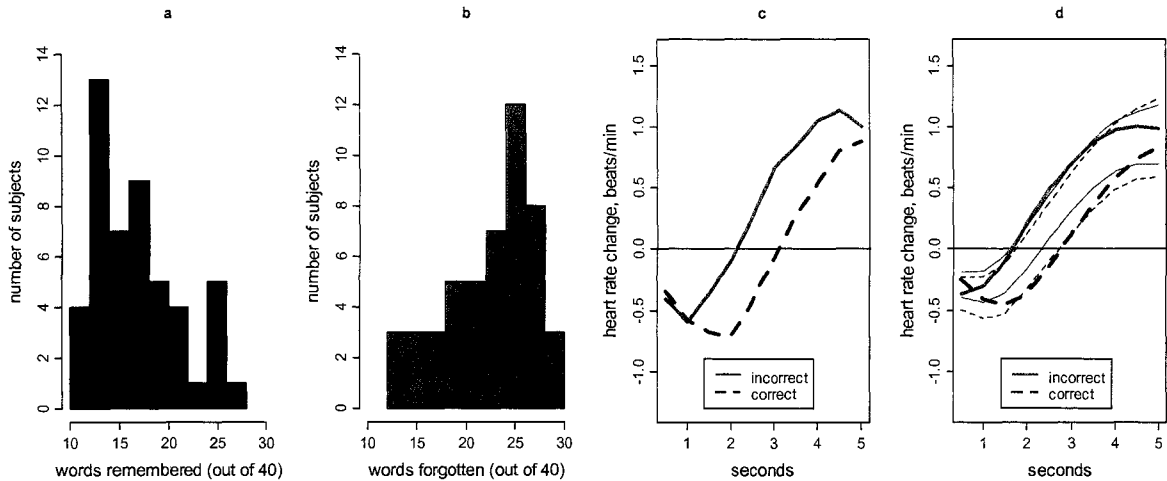
Figure 6.12. Delayed recall. Pane a: histogram showing the number of subjects by number of words correctly recalled. Pane b: histogram showing the number of subjects by number of words forgotten. Pane c: mean heart rate change in each bin recorded while viewing words later recalled or forgotten. Pane d: lowess curves and null bands for heart rate change in each bin recorded while viewing words later recalled or forgotten. Null bands vary due to the different number of words recalled and forgotten.

Table 6.7. Mean (standard deviation) and median of mean heart rate change in each 500 msec bin after the trigger, by recall.

| | immediate recall | | | | delayed recall | | | |
| | incorrect | | correct | | incorrect | | correct | |
bin	mean (sd)	mdn	mean (sd)	mdn	mean (sd)	mdn	mean (sd)	mdn
0	-0.39 (3.56)	-0.32	-0.38 (3.66)	-0.21	-0.41 (3.58)	-0.31	-0.34 (3.63)	-0.19
1	-0.53 (5.04)	-0.46	-0.65 (5.17)	-0.52	-0.60 (5.13)	-0.47	-0.58 (5.07)	-0.53
2	-0.30 (6.05)	0.07	-0.72 (5.85)	-0.52	-0.36 (6.08)	0.01	-0.68 (5.79)	-0.61
3	-0.05 (6.55)	0.31	-0.69 (6.27)	-0.36	-0.09 (6.57)	0.30	-0.72 (6.20)	-0.52
4	0.36 (6.80)	0.75	-0.47 (6.62)	-0.29	0.27 (6.80)	0.60	-0.44 (6.60)	-0.29
5	0.78 (6.93)	0.75	-0.13 (6.77)	-0.08	0.67 (6.90)	0.71	-0.08 (6.82)	-0.04
6	0.97 (7.21)	0.83	0.18 (6.83)	0.33	0.84 (7.10)	0.72	0.27 (6.96)	0.34
7	1.15 (7.68)	1.00	0.48 (7.01)	0.35	1.05 (7.50)	0.87	0.54 (7.20)	0.56
8	1.25 (7.96)	0.92	0.70 (7.18)	0.73	1.13 (7.72)	0.85	0.80 (7.43)	0.86
9	1.14 (7.89)	1.01	0.75 (7.34)	0.60	1.01 (7.70)	0.98	0.88 (7.54)	0.61

Discussion

The overall pattern of HR changes following stimulus onset (initial deceleration followed by acceleration) is seen when HR change curves are calculated based on each univariate measure. No differences were found in the curves calculated for gender or experiment, and only marginal differences seen in word type. Clear differences were seen in the condition and recall curves, however: cold condition subjects exhibited earlier HR

acceleration with less deceleration than the warm condition subjects. This finding is surprising; no condition-related differences were expected since the division of subjects into condition occurred after viewing the word slides. Differences were also found in the HR responses to words later recalled or forgotten: the acceleration was greater and earlier to words forgotten than remembered. This pattern was present both in the immediate and delayed recall data, but the relationship was stronger for immediate recall.

Bivariate Analyses

Heart rate responses may be determined by multiple factors, and these factors may interact to produce complex patterns. For example, males and females may have different HR patterns to words of different types. The data was not examined on all possible dimensions, because some pairings (e.g. condition and experiment) are not informative. Two methods of permuting the data were performed for each of these bivariate analyses, since multiple sources of variation are present. For example, when examining the HR changes by gender and word type, word type can be permuted within each subject of each gender (to indicate if HR change curves varied with word type for female subjects alone), or subject gender can be permuted within each word type (to indicate if HR change curves varies with gender for taboo words alone). The null bands generated by these two permutation tests highlight these different sources of variation in the data. The HR curves are identical in both cases, only the null bands change.
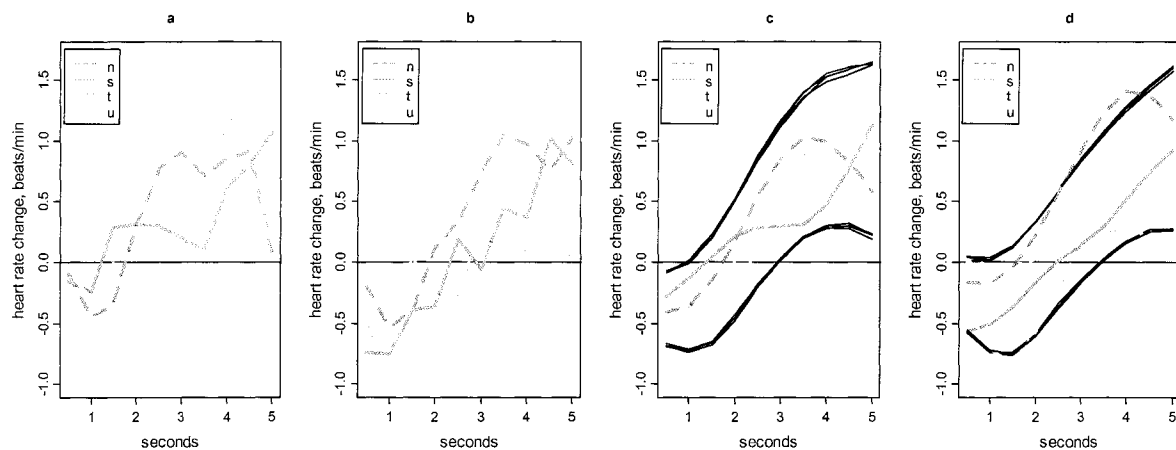
Figure 6.13. Heart rate change curves by word type after separating by gender. Pane a: median heart rate curves by word type for male subjects. Pane b: median heart rate curves by word type for female subjects. Null bands calculated by permuting word type after separating by gender. Pane c: lowess curves with null bands, male subjects. Pane d: lowess curves with null bands, female subjects.
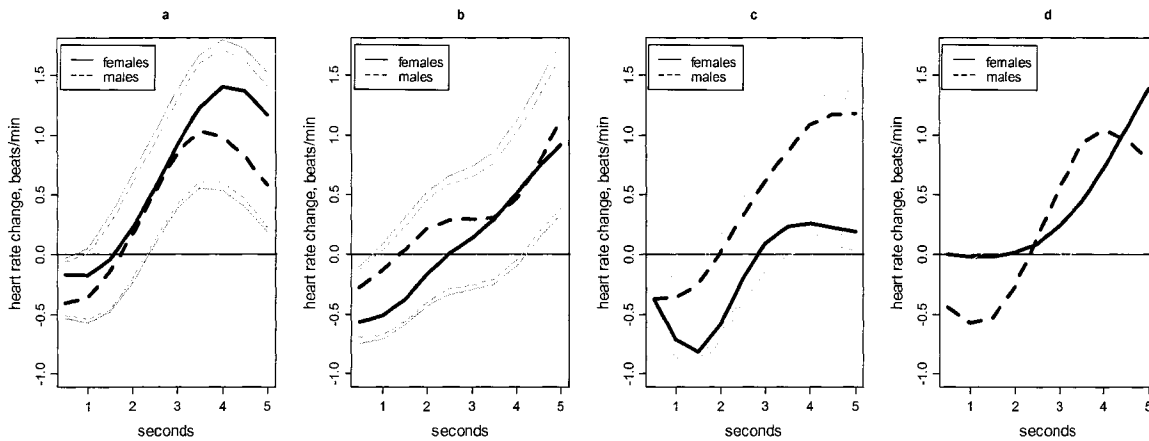
Figure 6.14. Heart rate change curves by gender after separating by word type. Null bands created by permuting gender within each word type. Pane a: neutral words. Pane b: student words. Pane c: taboo words. Pane d: unpleasant words.

Word Type and Gender

The mean, standard deviation, and median heart rate change in each bin by word type and gender is listed in Table 6.8; the data are plotted in Figure 6.13 and Figure 6.14. Nearly all (portions of the neutral and taboo curves exceed the bands for females, Figure 6.13, pane d) of the true curves are within the null bands, suggesting that heart rate does not vary systematically with word type within each gender or with gender within each word type.

Word Type and Condition

No differences were expected between the responses of cold and warm condition subjects, since the subjects were divided at random and the condition manipulation occurred after word viewing. Differences in HR changes associated with condition were found, however. The mean, standard deviation, and median HR change in each bin, separated by word type and condition, is listed in Table 6.9 and plotted in Figure 6.15 and Figure 6.17. The most striking differences are seen in to the unpleasant and taboo words; responses to the student words were similar between the two groups. The lowess (panes c and d) and median (panes a and b) curves in Figure 6.15 show that the HR decelerated more than expected to unpleasant words in the warm condition subjects, but accelerated more than expected in the cold condition subjects. Similarly, the HR accelerated more than expected during the taboo words in the warm water subjects, but decelerated more than expected in the cold condition subjects. These differences can also be seen when the null bands were

constructed by permuting condition label within each word type (Figure 6.17): the conditions varied more than expected during unpleasant and neutral words. These differences in HR responses during word viewing are not explained by differences in mean HR at stimulus onset; heart rates (Figure 6.16) did not vary with condition for any word type.

Table 6.8. Mean (standard deviation) and median of mean heart rate change in each 500 msec bin after the trigger, by word type and gender.

bin	males, taboo		males, unpleasant		males, school		males, neutral	
	mean (sd)	mdn	mean (sd)	mdn	mean (sd)	mdn	mean (sd)	mdn
0	-0.42 (3.70)	-0.09	-0.47 (3.49)	-0.36	-0.55 (4.40)	-0.15	-0.41 (3.41)	-0.09
1	-0.79 (5.31)	-0.28	-0.75 (4.79)	-0.71	-0.61 (6.39)	-0.24	-0.54 (4.66)	-0.45
2	-0.66 (5.99)	0.01	-0.78 (5.96)	-0.36	-0.31 (6.84)	0.28	-0.24 (5.55)	-0.34
3	-0.54 (6.50)	0.12	-0.37 (6.68)	0.30	-0.22 (6.77)	0.31	-0.00 (6.52)	0.31
4	-0.01 (6.93)	0.22	0.04 (7.12)	0.49	-0.20 (7.30)	0.29	0.28 (7.29)	0.77
5	0.25 (7.10)	0.10	0.88 (7.36)	0.55	-0.14 (7.69)	0.20	0.95 (7.37)	0.90
6	0.62 (7.33)	0.60	1.25 (7.37)	1.08	0.19 (7.45)	0.09	1.23 (7.60)	0.71
7	0.89 (7.49)	1.17	1.50 (7.51)	1.43	0.55 (7.78)	0.61	1.36 (8.13)	0.85
8	1.13 (7.50)	1.08	1.40 (7.77)	0.83	0.65 (7.99)	0.80	1.46 (8.29)	0.90
9	1.06 (7.32)	0.91	1.13 (7.66)	0.61	0.80 (8.16)	1.06	1.29 (8.57)	0.09

bin	females, taboo		females, unpleasant		females, school		females, neutral	
	mean (sd)	mdn	mean (sd)	mdn	mean (sd)	mdn	mean (sd)	mdn
0	-0.45 (3.65)	-0.52	-0.04 (3.60)	-0.07	-0.61 (3.30)	-0.74	-0.07 (3.06)	-0.19
1	-0.79 (4.63)	-1.00	-0.33 (4.92)	-0.39	-0.62 (5.16)	-0.75	-0.25 (4.64)	-0.53
2	-1.02 (5.84)	-0.83	-0.26 (5.37)	0.04	-0.58 (6.27)	-0.39	-0.12 (5.68)	-0.39
3	-0.91 (6.16)	-0.50	-0.39 (5.70)	-0.28	-0.54 (6.83)	-0.37	0.07 (6.07)	0.12
4	-0.38 (5.86)	-0.30	-0.12 (5.89)	-0.28	-0.17 (6.65)	0.19	0.22 (6.42)	0.33
5	0.10 (5.98)	0.36	0.21 (5.99)	0.18	-0.13 (6.23)	-0.06	0.57 (6.74)	0.75
6	0.40 (6.30)	0.40	0.44 (6.26)	0.10	0.02 (6.32)	0.44	0.49 (7.29)	1.04
7	0.65 (6.54)	0.06	0.73 (6.59)	0.54	0.30 (6.72)	0.37	0.52 (7.87)	0.97
8	0.66 (6.67)	0.34	1.18 (7.09)	1.06	0.84 (7.00)	1.02	0.50 (8.21)	0.76
9	0.47 (6.64)	-0.03	1.39 (7.21)	1.19	1.06 (7.33)	0.80	0.35 (7.90)	1.02
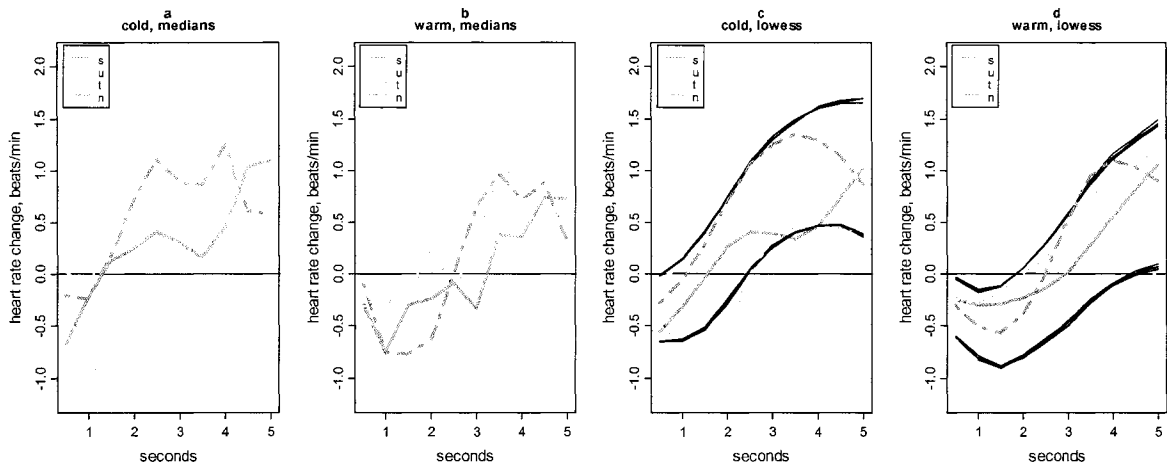
Figure 6.15. Heart rate change curves by word type after separating by condition. Pane a: median heart rate curves by word type for cold condition subjects. Pane b: median heart rate curves by word type for warm condition subjects. Pane c: lowess curves with null bands, cold condition subjects. Null bands calculated by permuting word type after separating by condition. Pane d: lowess curves with null bands, warm condition subjects. Null bands calculated by permuting word type after separating by condition.
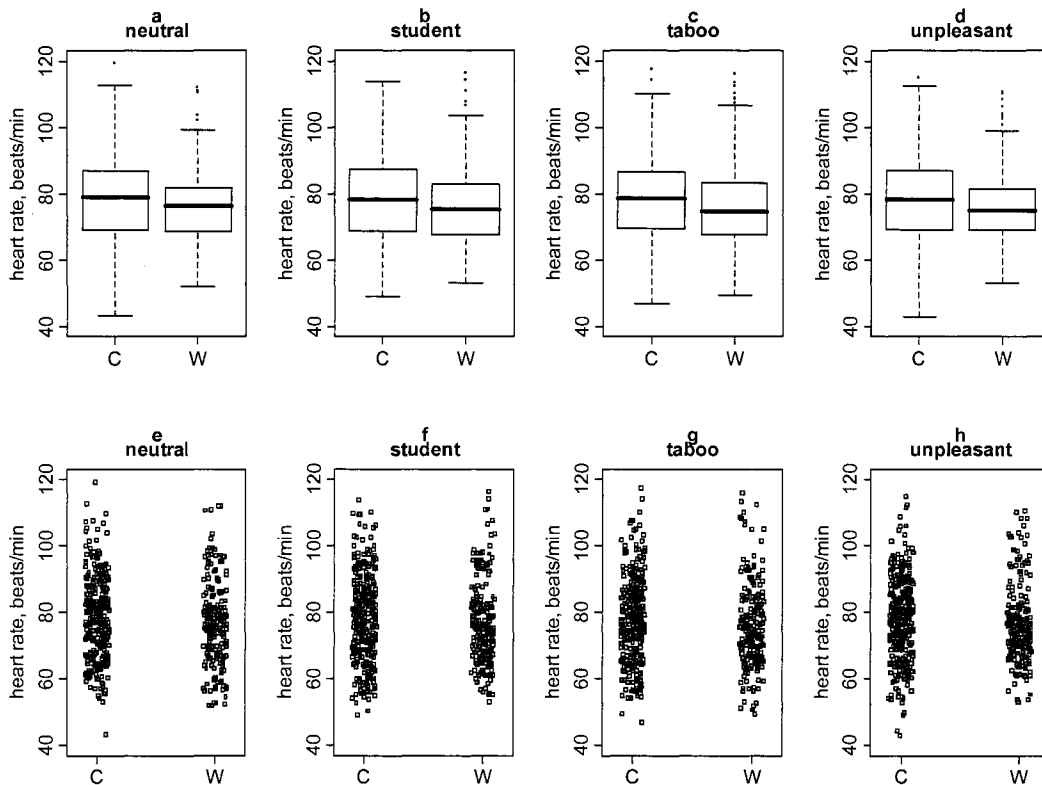
Figure 6.16. Mean heart rate at stimulus onset, by word type and condition. Panes a and e: neutral words. Panes b and f: student words. Panes c and g: taboo words. Panes d and h: unpleasant words.
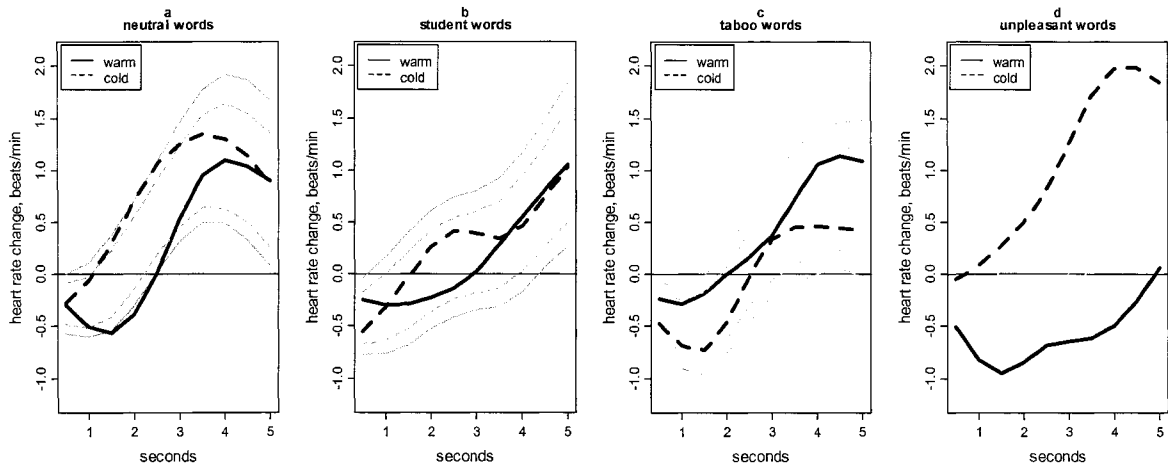
Figure 6.17. Heart rate change curves by condition after separating by word type. Null bands created by permuting condition within each word type. Pane a: neutral words. Pane b: student words. Pane c: taboo words. Pane d: unpleasant words.

Table 6.9. Mean (standard deviation) and median of mean heart rate change in each 500 msec bin after the trigger, by word type and condition.

bin	cold, taboo mean (sd)	mdn	cold, unpleasant mean (sd)	mdn	cold, student mean (sd)	mdn	cold, neutral mean (sd)	mdn
0	-0.53 (4.14)	-0.26	-0.15 (3.90)	0.03	-0.62 (3.92)	-0.67	-0.24 (3.66)	-0.20
1	-0.90 (5.54)	-0.93	-0.30 (5.37)	-0.18	-0.44 (5.90)	-0.22	-0.21 (5.29)	-0.24
2	-0.98 (6.67)	-0.85	-0.13 (6.24)	0.71	-0.17 (6.99)	0.13	0.08 (6.33)	0.17
3	-0.96 (6.94)	-0.40	0.12 (6.74)	0.86	-0.07 (7.45)	0.24	0.51 (6.93)	0.72
4	-0.26 (6.96)	0.04	0.51 (6.98)	1.18	0.29 (7.55)	0.41	0.84 (7.29)	1.11
5	0.22 (7.00)	0.10	1.22 (7.26)	1.43	0.27 (7.32)	0.30	1.32 (7.46)	0.90
6	0.51 (7.14)	0.38	1.66 (7.39)	1.97	0.38 (7.14)	0.16	1.17 (7.79)	0.86
7	0.71 (7.44)	0.19	1.91 (7.47)	2.01	0.57 (7.40)	0.45	1.19 (8.26)	1.25
8	0.74 (7.38)	0.06	2.00 (7.89)	1.92	0.90 (7.63)	1.04	1.03 (8.48)	0.61
9	0.59 (7.22)	0.11	1.48 (7.81)	1.51	1.08 (7.99)	1.09	0.86 (8.42)	0.58

bin	warm, taboo mean (sd)	mdn	warm, unpleasant mean (sd)	mdn	warm, student mean (sd)	mdn	warm, neutral mean (sd)	mdn
0	-0.28 (2.87)	-0.31	-0.44 (2.96)	-0.59	-0.50 (3.92)	-0.29	-0.27 (2.54)	-0.10
1	-0.64 (4.10)	-0.28	-0.91 (3.95)	-0.79	-0.86 (5.76)	-0.75	-0.67 (3.51)	-0.76
2	-0.62 (4.61)	-0.18	-1.13 (4.74)	-1.23	-0.84 (5.90)	-0.30	-0.57 (4.35)	-0.77
3	-0.35 (5.34)	0.25	-1.11 (5.34)	-0.87	-0.81 (5.72)	-0.24	-0.67 (5.21)	-0.64
4	-0.06 (5.64)	-0.16	-0.82 (5.85)	-0.46	-0.86 (6.05)	-0.09	-0.60 (6.17)	-0.01
5	0.13 (5.98)	0.32	-0.39 (5.82)	-0.54	-0.73 (6.57)	-0.33	-0.02 (6.41)	0.65
6	0.53 (6.46)	0.97	-0.27 (5.90)	-0.42	-0.28 (6.63)	0.39	0.46 (6.96)	0.97
7	0.87 (6.48)	1.02	0.01 (6.37)	0.00	0.23 (7.16)	0.36	0.64 (7.65)	0.72
8	1.17 (6.74)	1.19	0.28 (6.64)	-0.44	0.50 (7.41)	0.74	0.98 (7.94)	0.87
9	1.06 (6.70)	1.10	0.92 (6.89)	0.11	0.68 (7.47)	0.73	0.83 (8.06)	0.32

Word Type and Experiment

The mean, standard deviation, and median HR change in each bin, separated by word type and experiment, is listed in Table 6.10 and plotted in Figure 6.18 and Figure 6.19. There is more variation in the lowess curves, and wider null bands, in the consolidation experiment data compared to the retrieval subjects. This difference is primarily due to subject size: nearly twice as many subjects were assigned to the retrieval than the consolidation experiment ordering. When separated by experiment there are no differences in HR when words of different types were viewed. This result is consistent with what was observed when all subjects were combined (no clear difference in heart rate responses by word type, but marginal acceleration to neutral words around 3 seconds after stimulus onset). Consistently, no clear differences were observed between subjects in the retrieval and consolidation experiments when responses to words of each type were tested separately (Figure 6.19). These observations suggest that there was not a difference in HR responses between subjects assigned to the different experiment orderings.
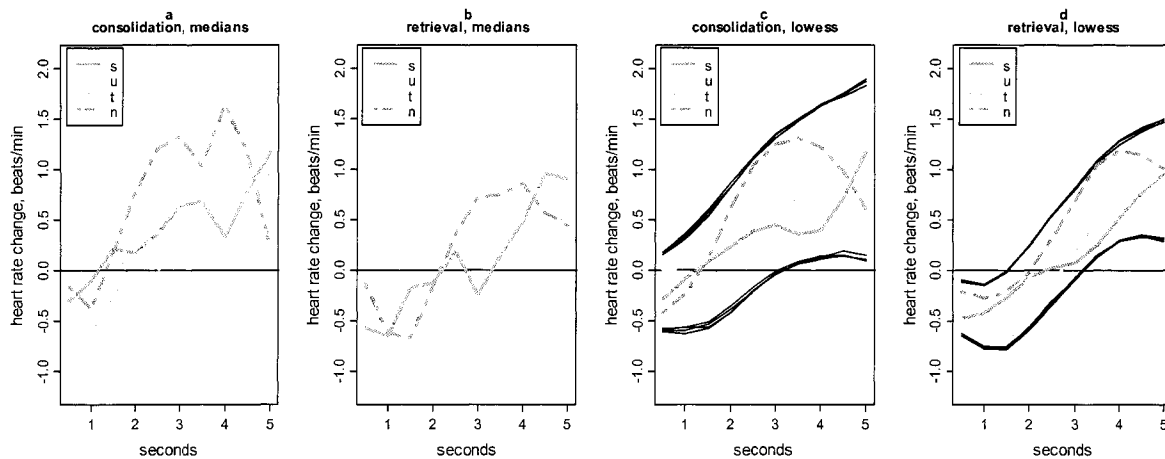
Figure 6.18. Heart rate change curves by word type after separating by experiment. Pane a: median heart rate curves by word type for consolidation experiment subjects. Pane b: median heart rate curves by word type for retrieval experiment subjects. Null bands calculated by permuting word type after separating by experiment. Pane c: lowess curves with null bands, consolidation experiment subjects. Pane d: lowess curves with null bands, retrieval experiment subjects.
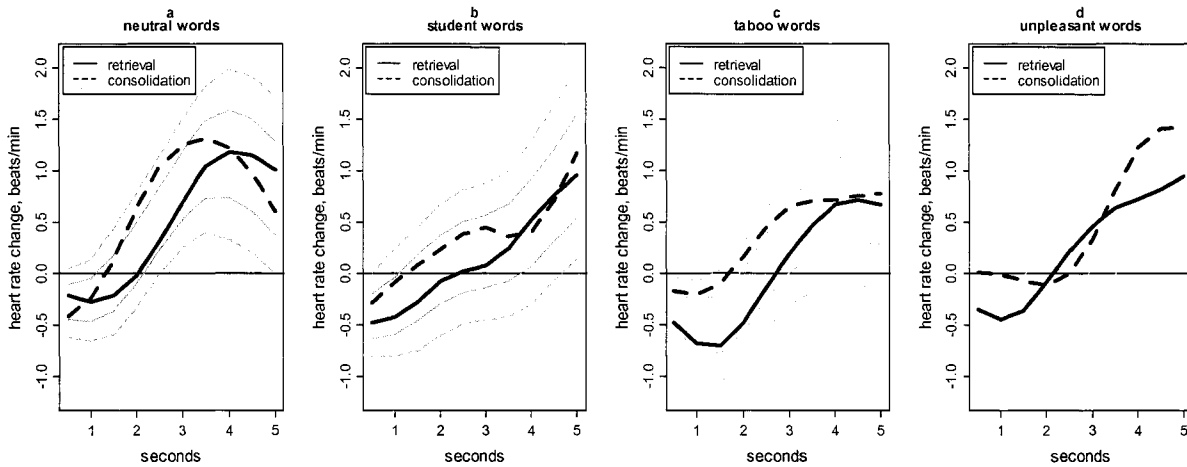
Figure 6.19. Heart rate change curves by experiment after separating by word type. Null bands created by permuting experiment within each word type. Pane a: neutral words. Pane b: student words. Pane c: taboo words. Pane d: unpleasant words.

Table 6.10. Mean (standard deviation) and median of mean heart rate change in each 500 msec bin after the trigger, by word type and experiment.

bin	consolidation, taboo mean (sd)	mdn	consolidation, unpleasant mean (sd)	mdn	consolidation, student mean (sd)	mdn	consolidation, neutral mean (sd)	mdn
0	-0.09 (3.65)	-0.09	-0.03 (3.46)	-0.06	-0.40 (3.95)	-0.31	-0.38 (3.68)	-0.16
1	-0.30 (4.87)	-0.64	-0.13 (4.98)	-0.01	-0.14 (5.75)	-0.11	-0.36 (4.92)	-0.38
2	-0.21 (5.86)	-0.27	-0.07 (5.87)	0.32	0.26 (6.50)	0.21	-0.01 (5.80)	0.17
3	-0.13 (6.33)	0.14	-0.11 (6.50)	-0.27	0.29 (6.58)	0.17	0.48 (6.39)	0.77
4	0.22 (6.76)	0.37	0.19 (6.87)	0.00	0.52 (6.61)	0.36	0.98 (6.97)	1.20
5	0.40 (7.01)	0.55	0.70 (7.24)	0.32	0.56 (6.97)	0.64	1.30 (7.41)	1.33
6	0.43 (7.09)	0.55	1.17 (7.52)	0.66	0.55 (6.94)	0.67	1.48 (7.83)	1.03
7	0.63 (7.21)	0.08	1.58 (7.82)	1.34	0.63 (7.67)	0.33	1.59 (8.18)	1.64
8	0.90 (7.16)	0.49	1.79 (8.20)	1.78	0.76 (8.05)	0.79	1.65 (8.44)	1.15
9	0.85 (7.18)	1.02	1.64 (8.10)	1.37	0.86 (8.10)	1.17	1.61 (8.51)	0.22

bin	retrieval, taboo mean (sd)	mdn	retrieval, unpleasant mean (sd)	mdn	retrieval, student mean (sd)	mdn	retrieval, neutral mean (sd)	mdn
0	-0.61 (3.68)	-0.39	-0.40 (3.59)	-0.25	-0.67 (3.91)	-0.58	-0.18 (3.00)	-0.14
1	-1.06 (5.05)	-0.55	-0.77 (4.77)	-0.51	-0.87 (5.88)	-0.65	-0.42 (4.51)	-0.62
2	-1.16 (5.93)	-0.52	-0.78 (5.59)	-0.43	-0.81 (6.59)	-0.17	-0.28 (5.51)	-0.67
3	-1.03 (6.33)	-0.42	-0.53 (6.09)	0.18	-0.72 (6.89)	-0.13	-0.21 (6.26)	-0.17
4	-0.39 (6.28)	-0.21	-0.15 (6.41)	0.12	-0.56 (7.17)	0.19	-0.13 (6.82)	0.35
5	0.06 (6.37)	0.10	0.49 (6.49)	0.32	-0.51 (7.06)	-0.25	0.49 (6.89)	0.73
6	0.56 (6.74)	0.51	0.71 (6.52)	0.55	-0.12 (6.94)	0.15	0.56 (7.25)	0.74
7	0.86 (6.98)	0.76	0.90 (6.68)	0.62	0.32 (7.10)	0.47	0.63 (7.92)	0.86
8	0.92 (7.11)	0.81	1.04 (7.02)	0.51	0.73 (7.26)	0.96	0.66 (8.15)	0.56
9	0.75 (6.93)	0.35	1.05 (7.08)	0.69	0.95 (7.61)	0.90	0.44 (8.12)	0.44

Word Type and Recall

The mean, standard deviation, and median HR change in each bin separated by word type and immediate recall is listed in Table 6.11 and plotted in Figure 6.20 and Figure 6.22. The most striking pattern is seen with unpleasant words: the heart decelerated more than expected when viewing unpleasant words later recalled correctly. Also, the heart tended to accelerate more than expected while viewing unpleasant words later forgotten. This effect is seen both when word type was permuted after separating the data by immediate recall (Figure 6.22 panes d and h) and when permuting immediate recall after separating the data by word type (Figure 6.20 pane d). There is also a trend towards a relationship between HR at viewing, word type, and immediate recall in the neutral and school words; no interaction is apparent for the taboo words. These graphs suggest that the subjects' HR changes while viewing the word slide stimuli may influence later recall for unpleasant words; the trends observed in the univariate recall data may be primarily due to the unpleasant and neutral words, not the student or taboo words.

The analysis and conclusions for heart rate changes by delayed recall and word type match those for heart rate changes by immediate recall and word type, due to the similarity between recall at both times. The mean, standard deviation, and median HR change in each bin are listed in Table 6.12 and plotted in Figure 6.21 and Figure 6.23. As with immediate recall, the clearest finding with delayed recall is in unpleasant words: the heart tended to decelerate when viewing words later remembered and accelerate when viewing words later forgotten. The patterns are not as strong in the delayed as in the immediate recall data.
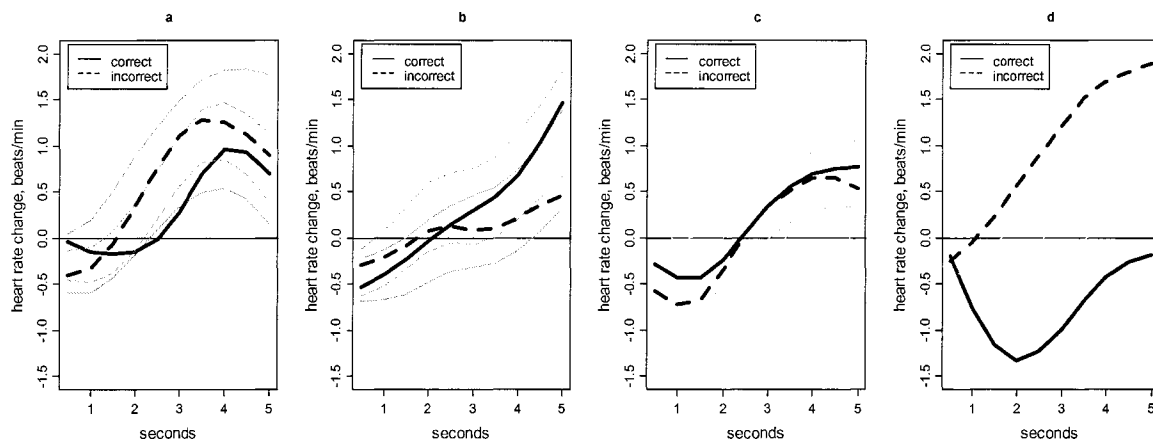
Figure 6.20. Null bands created by permuting immediate recall after separating the data by word type. Pane a: neutral words. Pane b: student words. Pane c: taboo words. Pane d: unpleasant words.
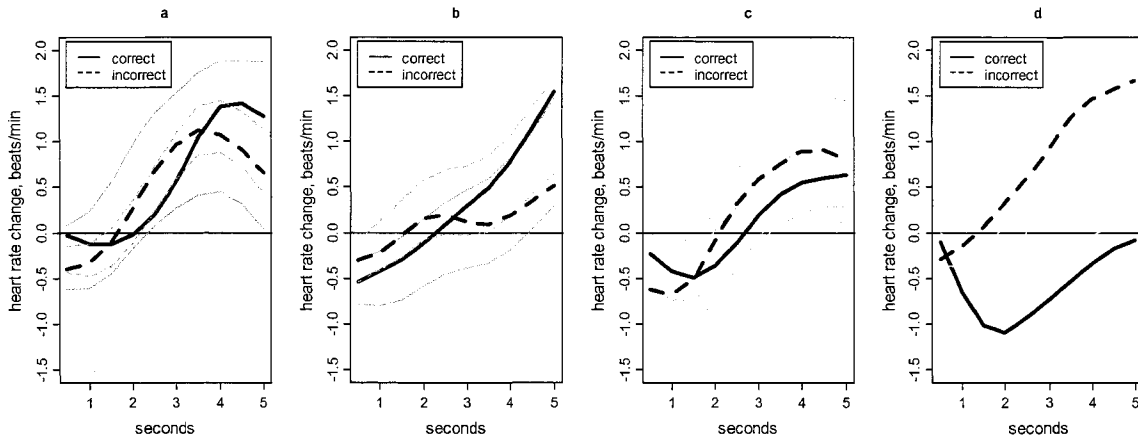
Figure 6.21. Null bands created by permuting delayed recall after separating the data by word type. Pane a: neutral words. Pane b: student words. Pane c: taboo words. Pane d: unpleasant words.
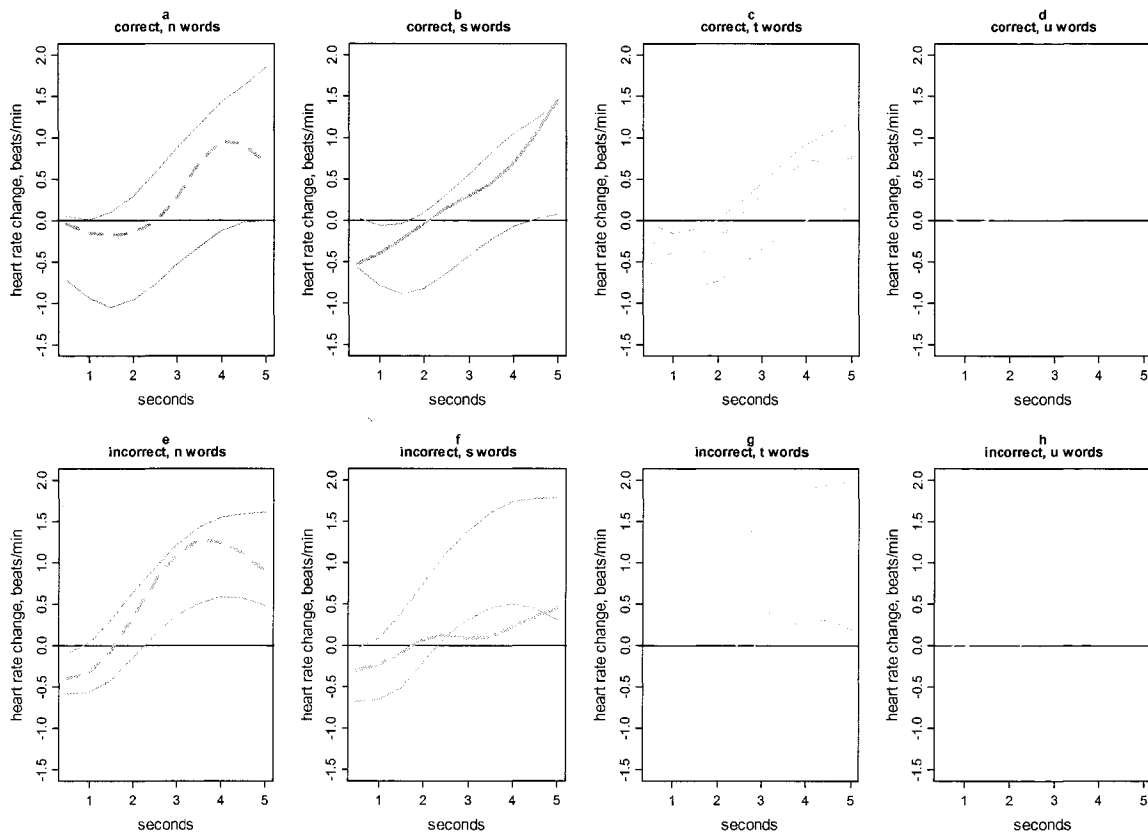
Figure 6.22. Null bands created by permuting word type after separating the data by accuracy at immediate recall. Pane a: correctly recalled neutral words. Pane b: correctly recalled student words. Pane c: correctly recalled taboo words. Pane e: correctly recalled unpleasant words. Pane e: forgotten neutral words. Pane f: forgotten student words. Pane g: forgotten taboo words. Pane h: forgotten unpleasant words.
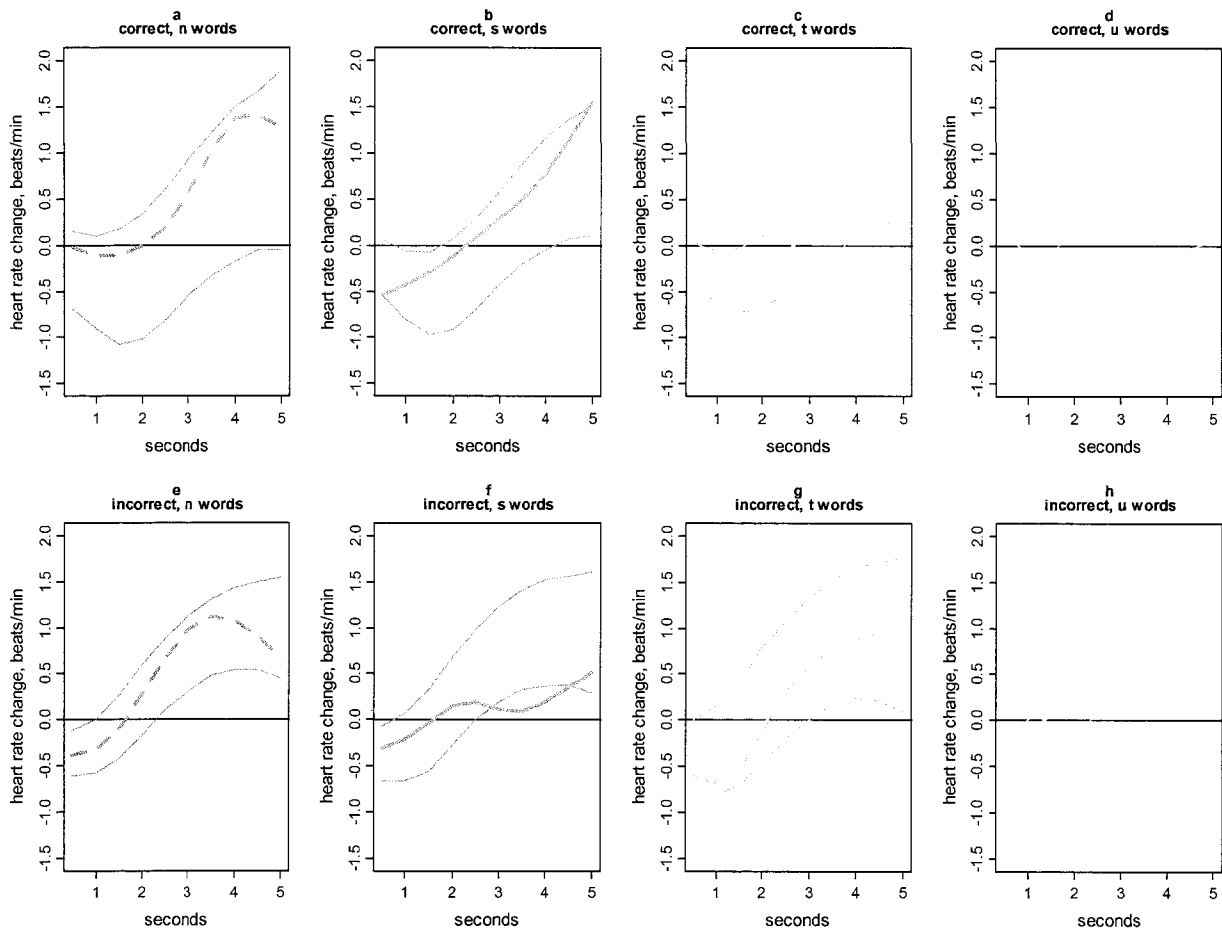
Figure 6.23. Null bands created by permuting word type after separating the data by accuracy at delayed recall. Pane a: correctly recalled neutral words. Pane b: correctly recalled student words. Pane c: correctly recalled taboo words. Pane e: correctly recalled unpleasant words. Pane e: forgotten neutral words. Pane f: forgotten student words. Pane g: forgotten taboo words. Pane h: forgotten unpleasant words.

Table 6.11. Mean (standard deviation) and median of mean heart rate change in each 500 msec bin after the trigger, by word type and immediate recall.

bin	correct, taboo		correct, unpleasant		correct, school		correct, neutral	
	mean (sd)	mdn	mean (sd)	mdn	mean (sd)	mdn	mean (sd)	mdn
0	-0.32 (3.53)	-0.18	-0.26 (3.72)	-0.28	-0.73 (4.08)	-0.58	-0.05 (3.07)	0.14
1	-0.61 (4.72)	-0.52	-0.98 (5.09)	-1.03	-0.72 (6.06)	-0.46	-0.25 (4.61)	-0.22
2	-0.53 (5.43)	-0.32	-1.48 (5.96)	-1.52	-0.53 (6.44)	-0.24	-0.48 (5.50)	-0.45
3	-0.41 (5.65)	-0.14	-1.65 (6.55)	-1.13	-0.43 (6.63)	-0.21	-0.56 (6.50)	0.02
4	0.01 (5.86)	-0.21	-1.54 (6.89)	-1.05	-0.20 (6.90)	0.00	-0.64 (7.19)	0.03
5	0.33 (6.18)	0.11	-1.11 (6.85)	-0.52	-0.01 (7.08)	-0.13	-0.11 (7.29)	0.54
6	0.63 (6.54)	0.54	-0.94 (6.79)	-0.27	0.29 (6.79)	0.55	0.43 (7.47)	0.66
7	0.89 (6.69)	0.31	-0.74 (6.94)	-0.01	0.74 (6.98)	0.88	0.66 (7.69)	0.59
8	1.08 (6.67)	0.79	-0.61 (7.17)	-0.51	1.08 (7.22)	1.32	0.88 (8.00)	1.00
9	1.05 (6.61)	0.78	-0.61 (7.46)	-0.88	1.37 (7.51)	1.21	0.75 (8.18)	0.32

bin	incorrect, taboo		incorrect, unpleasant		incorrect, school		incorrect, neutral	
	mean (sd)	mdn	mean (sd)	mdn	mean (sd)	mdn	mean (sd)	mdn
0	-0.65 (3.96)	-0.63	-0.27 (3.43)	-0.15	-0.41 (3.74)	-0.32	-0.35 (3.33)	-0.38
1	-1.18 (5.53)	-0.60	-0.27 (4.68)	-0.10	-0.49 (5.60)	-0.42	-0.47 (4.67)	-0.69
2	-1.46 (6.79)	-0.59	0.06 (5.43)	0.67	-0.34 (6.72)	0.13	-0.04 (5.66)	-0.35
3	-1.34 (7.56)	-0.24	0.43 (5.89)	0.85	-0.30 (6.99)	0.34	0.31 (6.20)	0.32
4	-0.57 (7.54)	0.49	0.92 (6.18)	1.22	-0.16 (7.11)	0.35	0.68 (6.71)	0.83
5	-0.14 (7.39)	0.40	1.63 (6.48)	1.33	-0.27 (7.00)	0.35	1.19 (6.94)	1.04
6	0.27 (7.49)	0.40	2.02 (6.70)	1.30	-0.09 (7.14)	0.06	1.09 (7.46)	1.04
7	0.54 (7.78)	0.96	2.34 (6.94)	1.56	0.10 (7.63)	0.02	1.11 (8.17)	0.99
8	0.57 (7.99)	0.50	2.52 (7.38)	1.57	0.37 (7.87)	0.57	1.07 (8.39)	0.81
9	0.23 (7.76)	-0.40	2.44 (7.20)	1.78	0.43 (8.04)	0.62	0.89 (8.32)	0.45

Table 6.12. Mean (standard deviation) and median of mean heart rate change in each 500 msec bin after the trigger, by word type and delayed recall.

	correct, taboo		correct, unpleasant		correct, school		correct, neutral	
bin	mean (sd)	mdn	mean (sd)	mdn	mean (sd)	mdn	mean (sd)	mdn
0	-0.27 (3.59)	-0.07	-0.14 (3.40)	-0.18	-0.78 (4.15)	-0.59	-0.01 (3.00)	0.09
1	-0.54 (4.69)	-0.47	-0.74 (4.67)	-1.03	-0.75 (5.99)	-0.53	-0.21 (4.63)	-0.22
2	-0.52 (5.53)	-0.51	-1.38 (5.61)	-1.51	-0.58 (6.40)	-0.23	-0.41 (5.50)	-0.63
3	-0.52 (5.91)	-0.63	-1.63 (6.20)	-1.10	-0.50 (6.39)	-0.31	-0.46 (6.48)	0.06
4	-0.14 (6.15)	-0.36	-1.47 (6.66)	-0.89	-0.17 (6.76)	-0.11	-0.34 (7.16)	0.17
5	0.15 (6.44)	0.04	-0.92 (6.84)	-0.27	0.02 (7.05)	-0.13	0.21 (7.18)	0.71
6	0.47 (6.73)	0.37	-0.75 (7.03)	-0.39	0.40 (6.86)	0.70	0.78 (7.46)	1.11
7	0.68 (6.89)	0.29	-0.60 (7.22)	-0.28	0.86 (7.19)	1.00	0.99 (7.76)	1.68
8	0.86 (7.06)	0.67	-0.34 (7.39)	-0.48	1.23 (7.50)	1.37	1.28 (8.08)	1.59
9	0.86 (6.97)	0.55	-0.31 (7.58)	-0.89	1.48 (7.81)	1.13	1.28 (8.15)	0.89

	incorrect, school		incorrect, neutral		incorrect, taboo		incorrect, unpleasant	
bin	mean (sd)	mdn	mean (sd)	mdn	mean (sd)	mdn	mean (sd)	mdn
0	-0.39 (3.70)	-0.27	-0.35 (3.34)	-0.31	-0.70 (3.81)	-0.63	-0.34 (3.61)	-0.16
1	-0.48 (5.70)	-0.42	-0.48 (4.66)	-0.65	-1.22 (5.46)	-0.60	-0.46 (4.94)	-0.25
2	-0.31 (6.73)	0.04	-0.09 (5.66)	-0.33	-1.36 (6.50)	-0.45	-0.12 (5.69)	0.55
3	-0.25 (7.15)	0.38	0.23 (6.23)	0.29	-1.03 (7.01)	-0.05	0.23 (6.16)	0.59
4	-0.20 (7.21)	0.37	0.49 (6.77)	0.81	-0.24 (6.93)	0.52	0.67 (6.41)	0.96
5	-0.28 (7.03)	0.35	1.00 (7.03)	0.86	0.24 (6.86)	0.61	1.30 (6.60)	1.02
6	-0.15 (7.01)	-0.01	0.92 (7.47)	0.79	0.58 (7.09)	0.78	1.67 (6.67)	1.03
7	0.04 (7.38)	-0.02	0.95 (8.13)	0.84	0.95 (7.34)	1.12	1.99 (6.89)	1.38
8	0.30 (7.55)	0.62	0.90 (8.34)	0.44	1.00 (7.24)	0.80	2.11 (7.35)	1.39
9	0.41 (7.72)	0.67	0.67 (8.32)	0.29	0.66 (7.09)	0.23	2.03 (7.27)	1.59

Discussion

Performing the bivariate analyses provided additional information about the relationships that were observed in the univariate analyses. No interaction between word type and gender or word type and experiment was found in the HR responses, consistent with the lack of overall differences seen in the univariate data. Interactions were observed between word type, recall and condition.

When the HR change curves were analyzed by recall it was found that the acceleration was greater and earlier to words forgotten than remembered, especially for immediate recall. An additional interaction was found between recall, word type, and HR response: the greater and earlier acceleration was most apparent for the unpleasant words, marginally present for the neutral words, and absent for the student and taboo words. Among the words later forgotten, the heart did not accelerate as much as expected to the

student words, while among the words later remembered the heart decelerated more than expected to the unpleasant words.

In the univariate data, it was found that cold condition subjects exhibited earlier HR acceleration with less deceleration than warm condition subjects. When the data was examined for interactions between word type and condition it could be seen that this pattern is only present in the HR change curves for neutral and unpleasant words; the HR change curves for the student and taboo words do not show condition-related differences.

Trivariate Analyses

Finally, a series of analyses were performed comparing the HR change curves when the data was split by three variables. As with the bivariate measures, the permutation tests were performed two ways for each variable combination. The number of subjects and words included in each group decreases as the number of variables included increases. These small sample sizes may increase the likelihood of encountering spurious results, so caution must be used in interpretation.

Word Type, Gender, and Recall

Curves were calculated for each word type by gender and recall (both immediate and delayed), as shown in Figure 6.24 and Figure 6.25 for immediate recall, and Figure 6.26 and Figure 6.27 for delayed recall. The mean, standard deviation, and median HR change in each bin is listed in Table 6.14 for immediate recall and in Table 6.13 for delayed recall. The results are very similar for immediate and delayed recall, and so are described together.

For female subjects, there is evidence for a relationship between HR response when viewing a word and recall, but only for unpleasant words. Panes a and b of Figure 6.24 (immediate) and Figure 6.26 (delayed) show lowess curves for female subjects for each word type after separating the data by recall. All curves are within the null bands except the curve for remembered neutral words (and a small portion of the remembered taboo word curve at delayed recall only). This curve represents the smallest number of subjects, however, only 27% of the neutral words were correctly recalled by female subjects at immediate recall (Table 6.2; 23% at delayed), so this finding may be primarily due to the small amount of data in this category. Together, these curves suggest that there was not a significant difference in female subjects' HR responses to words of different types when the responses were separated by recall accuracy. Figure 6.25 and Figure 6.27 contain the same lowess curves, calculated for each word type and recall, but with the null bands calculated by permuting immediate recall accuracy instead of word type. These curves

suggest that there was a difference in the HR response when females viewed unpleasant words related to whether the words were remembered at immediate recall. The curve for recalled unpleasant words suggests a deceleration compared to the curve for words later forgotten. Supporting the idea that the neutral correctly-recalled curve was primarily due to sample size, the curve is within the null bands when recall is permuted (pane a).

Male subjects exhibit the same immediate recall and HR response relationship to unpleasant words seen in the female subjects, as well as several relationships not found in the female subjects. The unpleasant word finding described in the females (acceleration associated with forgotten unpleasant words compared to remembered words) is more strongly apparent in the male subjects (Figure 6.25 and Figure 6.27, pane h). Additionally, male subjects showed more acceleration for forgotten neutral words compared to recalled neutral words (Figure 6.25 pane e; marginal for delayed recall, Figure 6.27 pane e). Significant differences are also found when the curves are compared by word type after separating by immediate recall (Figure 6.24, Figure 6.26, panes c and d). Specifically, among remembered words (pane c), unpleasant words had more HR deceleration than expected and taboo words (slightly) more acceleration. Among the forgotten words (pane d), there was more deceleration than expected to the taboo (immediate recall only) and student words and slightly more acceleration to the unpleasant words for a portion of the recording period.

These findings are consistent with what was observed previously looking at the measures univariately and bivariately. Particularly, these findings confirm that the relationship between HR response to unpleasant words and recall is present in both males and females. The marginal finding of a relationship between HR response to neutral words and recall (Figure 6.20 and Figure 6.21, pane a) is seen here in male, but not female subjects.

a
females, correct

b
females, incorrect

c
males, correct

d
males, incorrect

n
s
t
u

heart rate change, beats/min

seconds
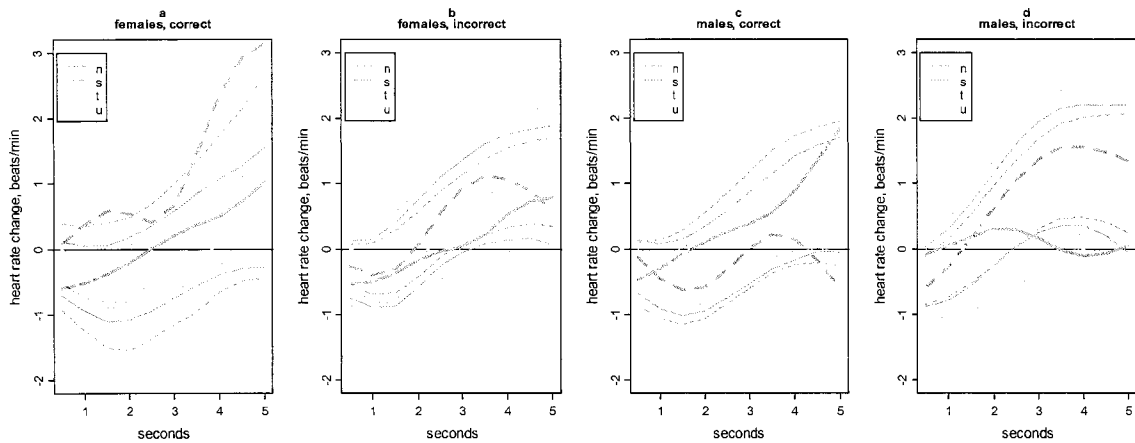
Figure 6.24. Heart rate change curves by gender, word type, and immediate recall. The null bands were calculated by permuting word type after separating the data by gender and immediate recall. Pane a: remembered words, female subjects. Pane b: forgotten words, female subjects. Pane c: remembered words, male subjects. Pane d: forgotten words, male subjects.

a
n, females

b
s, females

c
t, females

d
u, females

correct
incorrect

heart rate change, beats/min

seconds

e
n, males

f
s, males

g
t, males

h
u, males

correct
incorrect

heart rate change, beats/min

seconds
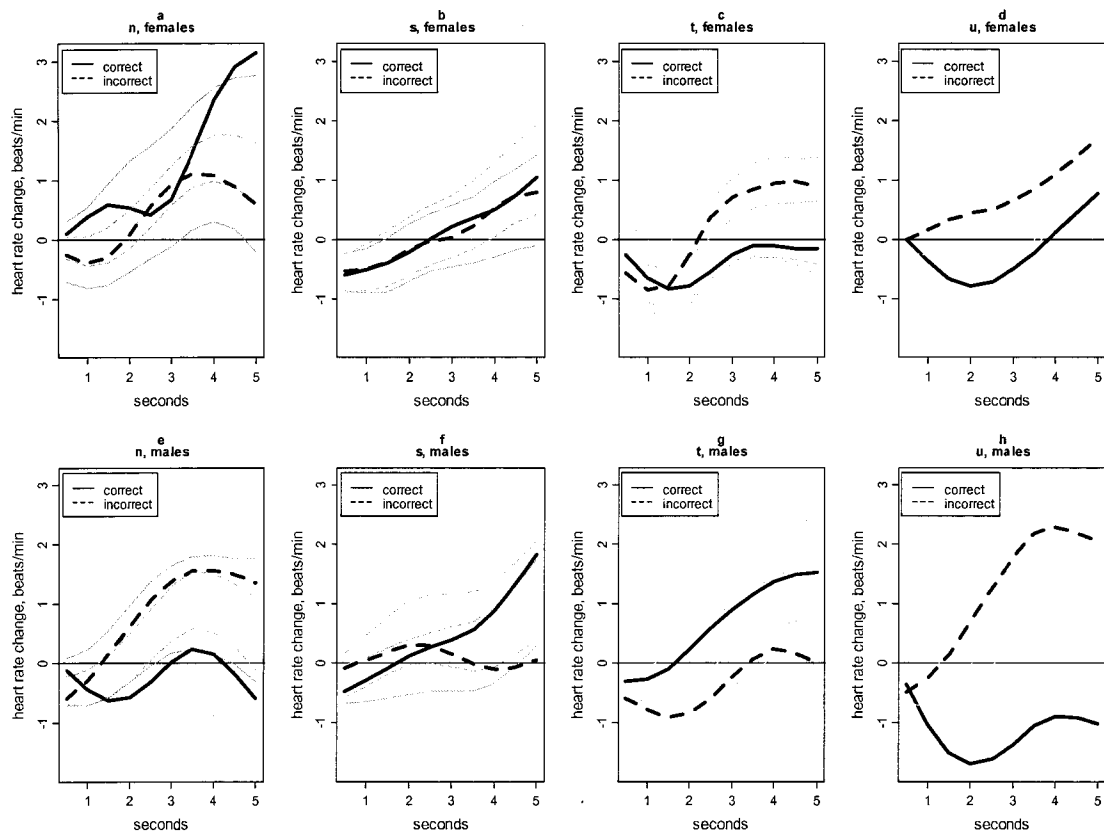
Figure 6.25. Heart rate change curves by gender, word type, and immediate recall. The null bands were calculated by permuting immediate recall after separating the data by word type and gender. Pane a: neutral words, female subjects. Pane b: student words, female subjects. Pane c: taboo words, female subjects. Pane d: unpleasant words, female subjects. Pane e: neutral words, male subjects. Pane f: student words, male subjects. Pane g: taboo words, male subjects. Pane h: unpleasant words, male subjects.
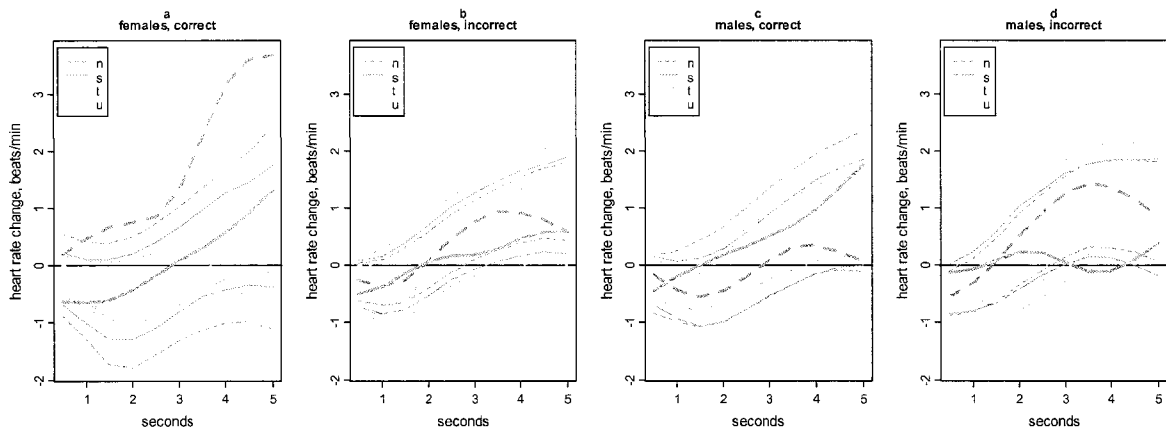
Figure 6.26. Heart rate change curves by gender, word type, and delayed recall. The null bands were calculated by permuting word type after separating the data by gender and delayed recall. Pane a: remembered words, female subjects. Pane b: forgotten words, female subjects. Pane c: remembered words, male subjects. Pane d: forgotten words, male subjects.
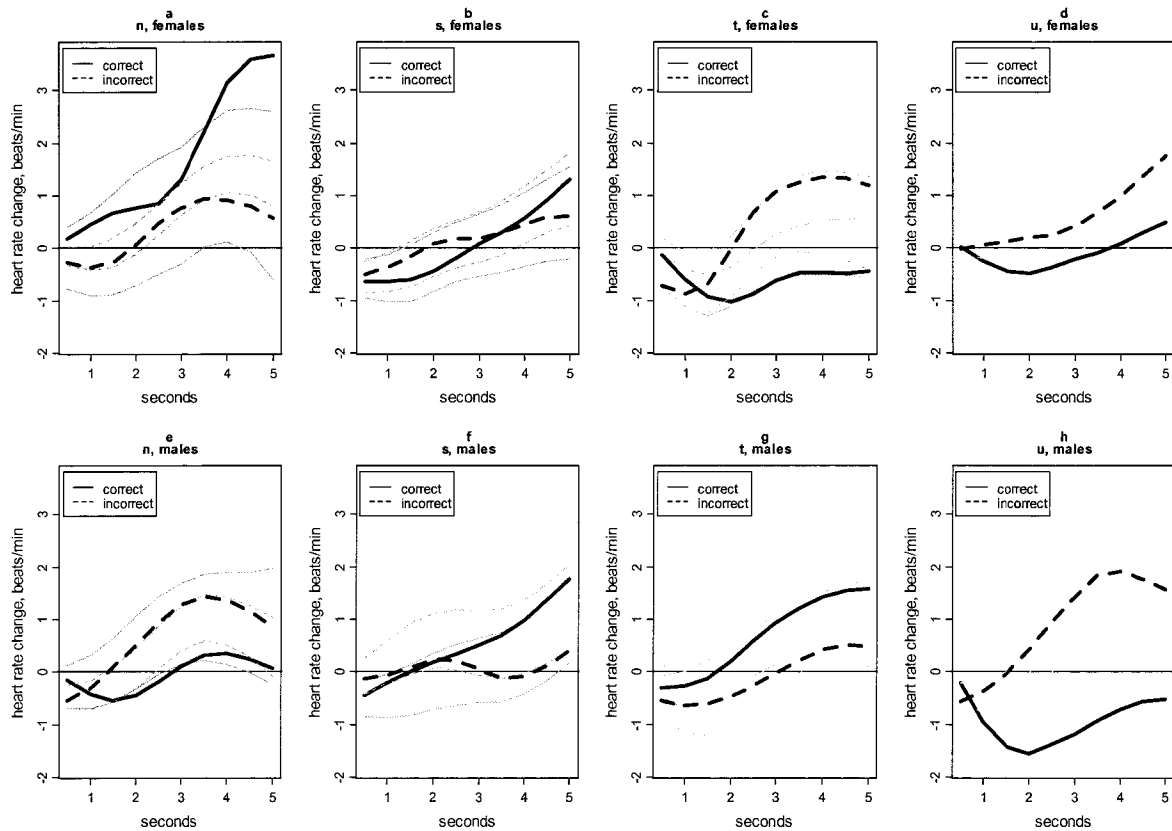
Figure 6.27. Heart rate change curves by gender, word type, and delayed recall. The null bands were calculated by permuting delayed recall after separating the data by word type and gender. Pane a: neutral words, female subjects. Pane b: student words, female subjects. Pane c: taboo words, female subjects. Pane d: unpleasant words, female subjects. Pane e: neutral words, male subjects. Pane f: student words, male subjects. Pane g: taboo words, male subjects. Pane h: unpleasant words, male subjects.

Table 6.13. Mean (standard deviation) and median of mean heart rate change in each 500 msec bin after the trigger, by word type, gender, and delayed recall.

bin	males correct, taboo		males correct, unpleasant		males correct, school		males correct, neutral	
	mean (sd)	mdn	mean (sd)	mdn	mean (sd)	mdn	mean (sd)	mdn
0	-0.30 (3.53)	-0.06	-0.21 (3.01)	-0.07	-0.75 (4.72)	-0.57	-0.12 (2.85)	0.11
1	-0.49 (4.76)	-0.29	-0.84 (3.95)	-1.21	-0.76 (6.84)	-0.23	-0.59 (4.19)	-0.32
2	-0.15 (5.53)	0.00	-1.62 (5.33)	-2.34	-0.48 (6.99)	0.33	-0.76 (4.82)	-0.91
3	0.00 (6.04)	0.26	-1.80 (6.05)	-1.24	-0.35 (6.65)	-0.05	-0.93 (6.36)	-1.01
4	0.42 (6.31)	0.91	-1.82 (6.36)	-1.54	-0.25 (7.23)	0.09	-0.88 (7.45)	-0.07
5	0.50 (6.74)	0.76	-1.06 (6.72)	-0.53	-0.05 (7.68)	0.20	-0.20 (7.38)	0.69
6	0.75 (6.97)	0.79	-0.76 (6.92)	-0.61	0.42 (7.27)	0.55	0.27 (7.52)	0.20
7	1.20 (7.06)	1.41	-0.72 (7.23)	-0.78	1.06 (7.73)	1.53	0.34 (7.80)	0.20
8	1.52 (7.37)	1.19	-0.58 (7.24)	-0.55	1.28 (8.06)	1.64	0.52 (7.86)	0.43
9	1.59 (7.27)	1.57	-0.66 (6.95)	-1.60	1.48 (8.21)	2.13	0.75 (8.06)	-0.35

bin	males incorrect, taboo		males incorrect, unpleasant		males incorrect, school		males incorrect, neutral	
	mean (sd)	mdn	mean (sd)	mdn	mean (sd)	mdn	mean (sd)	mdn
0	-0.63 (4.02)	-0.20	-0.61 (3.71)	-0.45	-0.34 (4.08)	0.17	-0.55 (3.67)	-0.31
1	-1.36 (6.20)	-0.28	-0.70 (5.17)	-0.36	-0.45 (5.94)	-0.37	-0.51 (4.90)	-0.59
2	-1.62 (6.71)	0.11	-0.35 (6.22)	0.65	-0.15 (6.71)	0.26	0.03 (5.89)	0.25
3	-1.56 (7.22)	0.01	0.36 (6.88)	1.23	-0.10 (6.92)	0.60	0.47 (6.57)	0.80
4	-0.82 (7.95)	-0.22	0.99 (7.32)	1.31	-0.15 (7.40)	0.32	0.89 (7.16)	1.07
5	-0.22 (7.75)	-0.50	1.87 (7.49)	1.45	-0.23 (7.73)	0.22	1.55 (7.32)	1.37
6	0.36 (7.98)	0.17	2.27 (7.40)	2.02	-0.03 (7.65)	-0.19	1.73 (7.62)	0.92
7	0.31 (8.24)	0.97	2.64 (7.42)	2.29	0.04 (7.83)	-0.16	1.89 (8.28)	1.21
8	0.40 (7.71)	0.51	2.42 (7.85)	1.20	0.03 (7.90)	0.55	1.94 (8.48)	1.10
9	0.07 (7.34)	-0.22	2.05 (7.86)	1.50	0.12 (8.08)	0.21	1.57 (8.84)	0.32

bin	females correct, taboo		females correct, unpleasant		females correct, school		females correct, neutral	
	mean (sd)	mdn	mean (sd)	mdn	mean (sd)	mdn	mean (sd)	mdn
0	-0.23 (3.67)	-0.18	-0.05 (3.84)	-0.32	-0.82 (3.34)	-0.63	0.17 (3.27)	-0.08
1	-0.59 (4.62)	-0.93	-0.61 (5.43)	-0.70	-0.75 (4.78)	-1.06	0.43 (5.27)	0.00
2	-0.97 (5.51)	-0.99	-1.11 (5.95)	-0.46	-0.72 (5.62)	-0.55	0.19 (6.49)	0.61
3	-1.17 (5.69)	-1.12	-1.43 (6.41)	-0.87	-0.68 (6.08)	-0.73	0.32 (6.68)	1.54
4	-0.85 (5.90)	-0.82	-1.05 (7.02)	-0.64	-0.07 (6.16)	-0.43	0.58 (6.61)	0.84
5	-0.29 (6.04)	-0.50	-0.76 (7.02)	-0.22	0.10 (6.22)	-0.30	0.92 (6.84)	1.31
6	0.13 (6.43)	-0.28	-0.74 (7.21)	-0.31	0.38 (6.34)	0.83	1.63 (7.35)	1.97
7	0.03 (6.64)	-0.50	-0.45 (7.25)	0.03	0.61 (6.49)	0.32	2.09 (7.66)	2.99
8	0.04 (6.58)	-0.25	-0.06 (7.60)	-0.15	1.16 (6.78)	1.16	2.56 (8.37)	3.49
9	-0.04 (6.50)	-0.51	0.11 (8.29)	-0.07	1.49 (7.32)	0.73	2.17 (8.32)	3.95

Table 6.13, continued.

bin	females incorrect, taboo mean (sd)	mdn	females incorrect,unpleasant mean (sd)	mdn	females incorrect, school mean (sd)	mdn	females incorrect, neutral mean (sd)	mdn
0	-0.77 (3.62)	-1.06	-0.04 (3.49)	0.01	-0.43 (3.27)	-0.81	-0.15 (3.00)	-0.34
1	-1.09 (4.67)	-1.02	-0.19 (4.67)	-0.09	-0.52 (5.47)	-0.53	-0.45 (4.43)	-0.69
2	-1.11 (6.32)	-0.59	0.13 (5.04)	0.35	-0.47 (6.78)	-0.08	-0.21 (5.44)	-0.49
3	-0.52 (6.81)	-0.20	0.10 (5.28)	0.04	-0.42 (7.42)	0.12	-0.01 (5.91)	-0.21
4	0.32 (5.77)	0.77	0.33 (5.23)	-0.04	-0.25 (7.05)	0.58	0.11 (6.37)	0.23
5	0.68 (5.87)	0.82	0.67 (5.41)	0.80	-0.33 (6.26)	0.43	0.46 (6.72)	0.71
6	0.80 (6.13)	1.29	1.00 (5.70)	0.55	-0.28 (6.31)	0.28	0.14 (7.26)	0.72
7	1.58 (6.33)	1.12	1.29 (6.20)	0.66	0.05 (6.92)	0.57	0.05 (7.89)	0.65
8	1.57 (6.74)	1.03	1.77 (6.78)	1.56	0.57 (7.19)	0.88	-0.12 (8.08)	-0.09
9	1.24 (6.82)	0.74	2.00 (6.58)	1.69	0.70 (7.35)	1.03	-0.20 (7.72)	0.26

Table 6.14. Mean (standard deviation) and median of mean heart rate change in each 500 msec bin after the trigger, by word type, gender, and immediate recall.

bin	males correct, taboo mean (sd)	mdn	males correct, unpleasant mean (sd)	mdn	males correct, school mean (sd)	mdn	males correct, neutral mean (sd)	mdn
0	-0.31 (3.50)	-0.07	-0.43 (3.71)	-0.18	-0.74 (4.56)	-0.31	-0.14 (3.02)	0.14
1	-0.58 (4.90)	-0.34	-1.19 (4.95)	-1.21	-0.84 (6.67)	-0.23	-0.57 (4.19)	-0.34
2	-0.21 (5.43)	0.01	-1.65 (6.16)	-1.75	-0.58 (6.91)	0.24	-0.82 (4.81)	-0.87
3	0.01 (5.66)	0.28	-1.62 (6.85)	-1.14	-0.45 (6.84)	-0.09	-0.95 (6.35)	-1.01
4	0.47 (5.88)	0.59	-1.71 (7.02)	-1.54	-0.40 (7.40)	0.03	-0.87 (7.36)	-0.37
5	0.64 (6.34)	0.73	-1.11 (7.03)	-0.54	-0.26 (7.72)	-0.11	-0.16 (7.26)	0.42
6	0.88 (6.69)	0.98	-0.97 (6.85)	0.00	0.28 (7.18)	0.34	0.26 (7.36)	0.36
7	1.33 (6.73)	1.44	-0.88 (7.18)	-0.27	0.96 (7.56)	1.29	0.23 (7.63)	-0.03
8	1.72 (6.74)	1.47	-0.99 (7.36)	-0.77	1.26 (7.83)	1.65	0.24 (7.67)	-0.84
9	1.75 (6.72)	1.59	-1.17 (7.31)	-1.70	1.50 (7.93)	2.15	0.18 (7.99)	-0.98

bin	males incorrect, taboo mean (sd)	mdn	males incorrect, unpleasant mean (sd)	mdn	males incorrect, school mean (sd)	mdn	males incorrect, neutral mean (sd)	mdn
0	-0.67 (4.15)	-0.30	-0.50 (3.33)	-0.38	-0.32 (4.22)	0.24	-0.56 (3.62)	-0.33
1	-1.30 (6.17)	-0.21	-0.44 (4.66)	-0.20	-0.33 (6.06)	-0.30	-0.52 (4.93)	-0.59
2	-1.71 (7.09)	0.21	-0.16 (5.75)	0.81	0.00 (6.76)	0.31	0.10 (5.93)	0.28
3	-1.85 (8.05)	-0.16	0.51 (6.43)	1.33	0.04 (6.71)	0.62	0.55 (6.57)	0.88
4	-1.14 (8.91)	-0.39	1.28 (6.96)	1.64	0.04 (7.22)	0.35	0.96 (7.19)	1.13
5	-0.69 (8.61)	-0.89	2.29 (7.27)	1.83	0.01 (7.69)	0.52	1.60 (7.38)	1.48
6	-0.02 (8.66)	-0.90	2.82 (7.35)	2.16	0.09 (7.79)	-0.04	1.79 (7.70)	1.18
7	-0.15 (9.00)	0.72	3.19 (7.31)	2.61	0.07 (8.05)	-0.23	2.02 (8.37)	1.38
8	-0.25 (8.95)	0.37	3.10 (7.62)	1.49	-0.05 (8.14)	0.27	2.17 (8.57)	1.34
9	-0.58 (8.40)	-0.51	2.77 (7.50)	2.23	-0.02 (8.37)	-0.25	1.93 (8.85)	0.78

Table 6.14, continued.

bin	females correct, taboo mean (sd)	mdn	females correct, unpleasant mean (sd)	mdn	females correct, school mean (sd)	mdn	females correct, neutral mean (sd)	mdn
0	-0.34 (3.58)	-0.36	-0.05 (3.75)	-0.28	-0.71 (3.42)	-0.67	0.09 (3.16)	0.15
1	-0.64 (4.50)	-0.80	-0.72 (5.28)	-0.74	-0.58 (5.26)	-0.92	0.26 (5.18)	0.61
2	-0.92 (5.43)	-0.83	-1.25 (5.72)	-0.61	-0.48 (5.87)	-0.51	0.04 (6.43)	0.63
3	-0.93 (5.60)	-0.92	-1.70 (6.17)	-1.08	-0.41 (6.39)	-0.36	0.05 (6.74)	1.56
4	-0.56 (5.81)	-0.64	-1.30 (6.75)	-0.66	0.03 (6.28)	-0.19	-0.28 (6.97)	0.32
5	-0.05 (5.97)	-0.34	-1.10 (6.65)	-0.52	0.28 (6.25)	-0.13	-0.03 (7.40)	0.75
6	0.32 (6.36)	0.19	-0.89 (6.75)	-0.31	0.30 (6.25)	0.74	0.68 (7.69)	1.18
7	0.36 (6.61)	-0.38	-0.57 (6.65)	-0.01	0.47 (6.24)	0.35	1.32 (7.79)	2.63
8	0.28 (6.52)	-0.19	-0.11 (6.93)	0.11	0.86 (6.43)	0.73	1.85 (8.47)	2.65
9	0.19 (6.40)	-0.20	0.13 (7.63)	0.16	1.21 (7.00)	0.74	1.62 (8.45)	3.51

bin	females incorrect, taboo mean (sd)	mdn	females incorrect, unpleasant mean (sd)	mdn	females incorrect, school mean (sd)	mdn	females incorrect, neutral mean (sd)	mdn
0	-0.64 (3.79)	-1.10	-0.04 (3.52)	0.09	-0.50 (3.17)	-0.80	-0.14 (3.02)	-0.53
1	-1.07 (4.89)	-1.15	-0.10 (4.71)	-0.05	-0.66 (5.08)	-0.74	-0.43 (4.42)	-0.76
2	-1.22 (6.54)	-0.84	0.29 (5.10)	0.60	-0.69 (6.69)	-0.17	-0.18 (5.40)	-0.67
3	-0.87 (7.10)	-0.28	0.34 (5.29)	0.23	-0.67 (7.29)	-0.46	0.08 (5.83)	-0.25
4	-0.04 (5.99)	0.55	0.55 (5.24)	0.05	-0.38 (7.02)	0.37	0.41 (6.21)	0.33
5	0.38 (6.03)	0.68	0.94 (5.48)	0.97	-0.56 (6.21)	0.06	0.79 (6.48)	0.75
6	0.55 (6.27)	0.97	1.20 (5.86)	0.65	-0.28 (6.40)	0.23	0.41 (7.16)	0.96
7	1.19 (6.43)	1.02	1.47 (6.46)	0.75	0.13 (7.20)	0.43	0.22 (7.90)	0.78
8	1.35 (6.93)	0.74	1.91 (7.10)	1.57	0.81 (7.58)	1.12	0.00 (8.08)	0.19
9	0.99 (7.07)	0.17	2.10 (6.89)	1.60	0.90 (7.69)	0.85	-0.12 (7.67)	0.16

Word Type, Condition, and Recall

Finally, HR change curves were calculated to describe the interactions of word type, condition, and recall. The mean, median, and standard deviation HR change at each time point is listed in Table 6.15 for immediate recall and Table 6.16 for delayed recall. The immediate recall data is graphed in Figure 6.28 and Figure 6.30, while the delayed recall data is graphed in Figure 6.29 and Figure 6.31. Figure 6.32 contains the mean HR at stimulus onset, by word type, condition, and immediate recall. In general, the same patterns were observed in the warm and cold condition subjects, but the results are not equivalent for both conditions. The differences are not due to a difference in mean HR between the conditions; the mean HR at stimulus onset were the same for the conditions (Figure 6.32).

Recall was permuted within word type and condition (Figure 6.30 and Figure 6.31) to check for differences in the pattern of interactions in HR, recall, and word type between the conditions. The same patterns were observed for warm and cold condition subjects, especially on the neutral and student words. At both immediate and delayed recall the

curves for words remembered and forgotten were within the null bands for neutral and student words, suggesting that the HR responses for these words were not related to recall, in warm or cold condition subjects. As discussed in Buchanan et al. (in press), there is a suggestion of a difference between HR responses to taboo words remembered or forgotten at immediate recall in warm condition subjects. This suggestion is not present in cold condition subjects, however; the curves for cold condition subjects are within the null bands but suggest an opposite trend to that observed in the warm condition subjects. The distinct difference in HR change curves to unpleasant words recalled or forgotten is present in both warm and cold condition subjects, but the difference is larger in cold than warm condition subjects (distinct deceleration to recalled words, acceleration to forgotten words).

Several additional trends are present in the graphs showing the results of permuting word type within each condition and recall group (Figure 6.28 and Figure 6.29), although the overall patterns are similar. A difference in HR change curves for remembered and forgotten taboo words is present at both immediate and delayed recall in the warm subjects; this difference is not present in the cold subjects, though they have a small split in the forgotten words. The difference in unpleasant words between the HR curve for recalled and forgotten words is more pronounced in the cold subjects, though present in recalled words in the warm condition subjects. Also, a distinct flattening of the HR changes is present in the cold condition subjects for forgotten student words. This trend is absent in the warm condition subjects. In both groups the neutral curve was within the bands nearly all of the time, for both recalled and forgotten words.
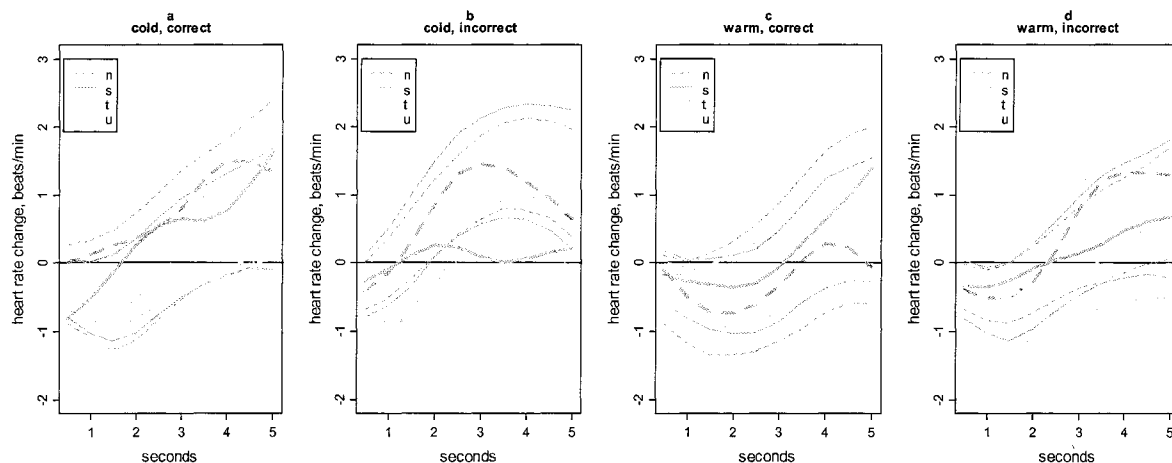
Figure 6.28. Heart rate change curves by condition, word type, and immediate recall. The null bands were calculated by permuting word type after separating the data by condition and immediate recall. Pane a: remembered words, cold condition subjects. Pane b: forgotten words, cold condition subjects. Pane c: remembered words, warm condition subjects. Pane d: forgotten words, warm condition subjects.
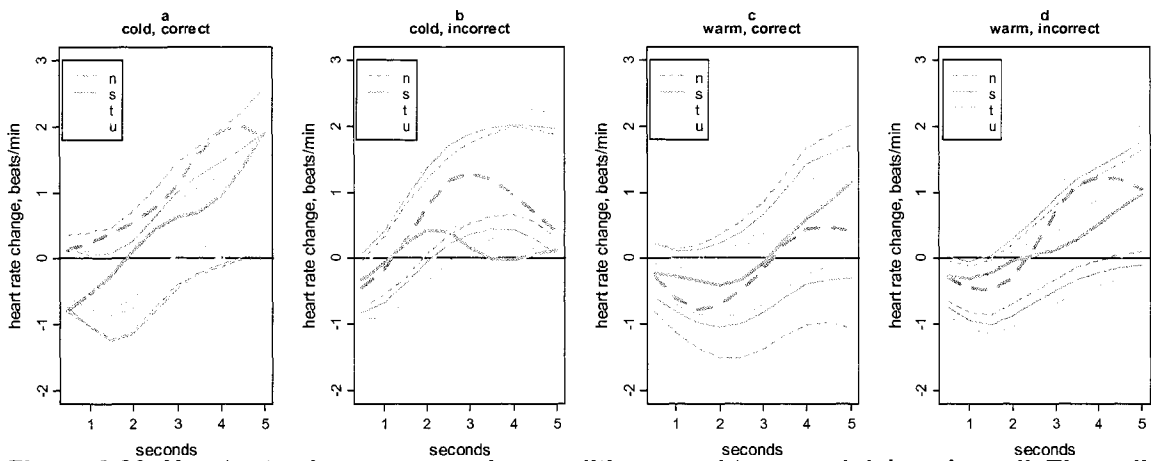
Figure 6.29. Heart rate change curves by condition, word type, and delayed recall. The null bands were calculated by permuting word type after separating the data by condition and delayed recall. Pane a: remembered words, cold condition subjects. Pane b: forgotten words, cold condition subjects. Pane c: remembered words, warm condition subjects. Pane d: forgotten words, warm condition subjects.
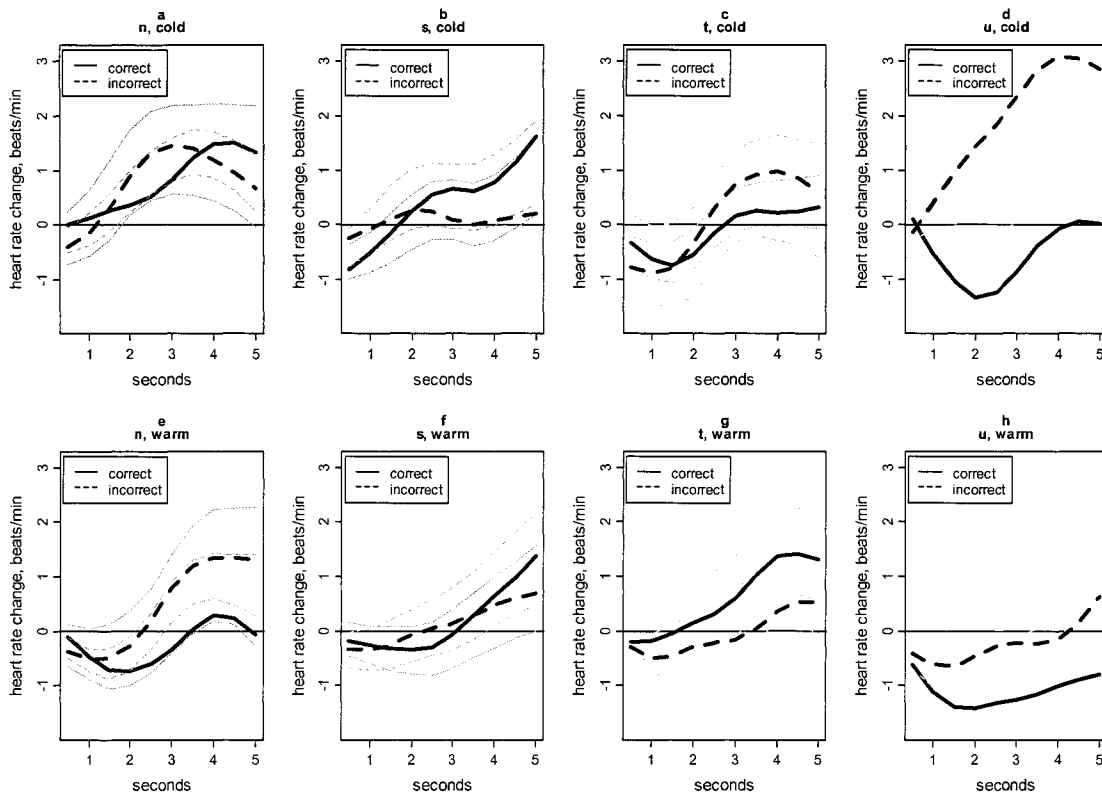
Figure 6.30. Heart rate change curves by condition, word type, and immediate recall. The null bands were calculated by permuting immediate recall after separating the data by word type and condition. Pane a: neutral words, cold condition subjects. Pane b: student words, cold condition subjects. Pane c: taboo words, cold condition subjects. Pane d: unpleasant words, cold condition subjects. Pane e: neutral words, warm condition subjects. Pane f: student words, warm condition subjects. Pane g: taboo words, warm condition subjects. Pane h: unpleasant words, warm condition subjects.
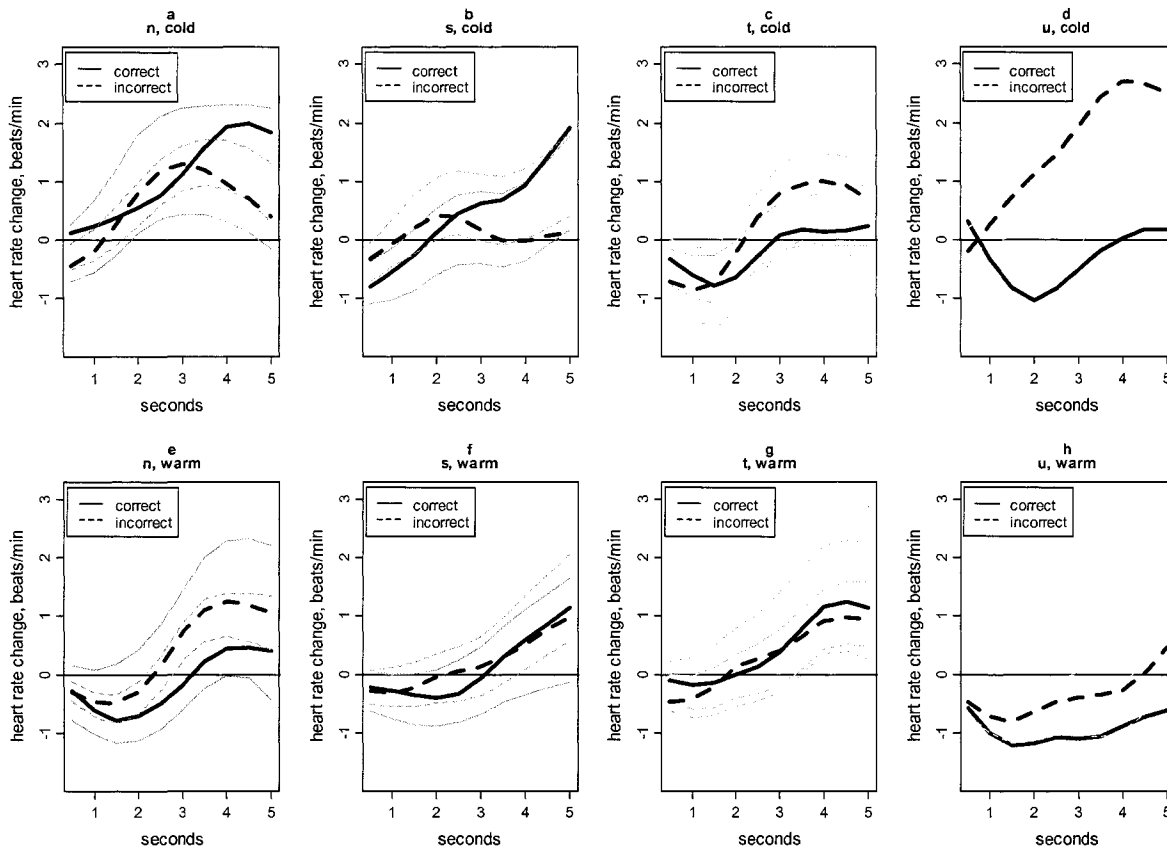
Figure 6.31. Heart rate change curves by condition, word type, and delayed recall. The null bands were calculated by permuting delayed recall after separating the data by word type and condition. Pane a: neutral words, cold condition subjects. Pane b: student words, cold condition subjects. Pane c: taboo words, cold condition subjects. Pane d: unpleasant words, cold condition subjects. Pane e: neutral words, warm condition subjects. Pane f: student words, warm condition subjects. Pane g: taboo words, warm condition subjects. Pane h: unpleasant words, warm condition subjects.
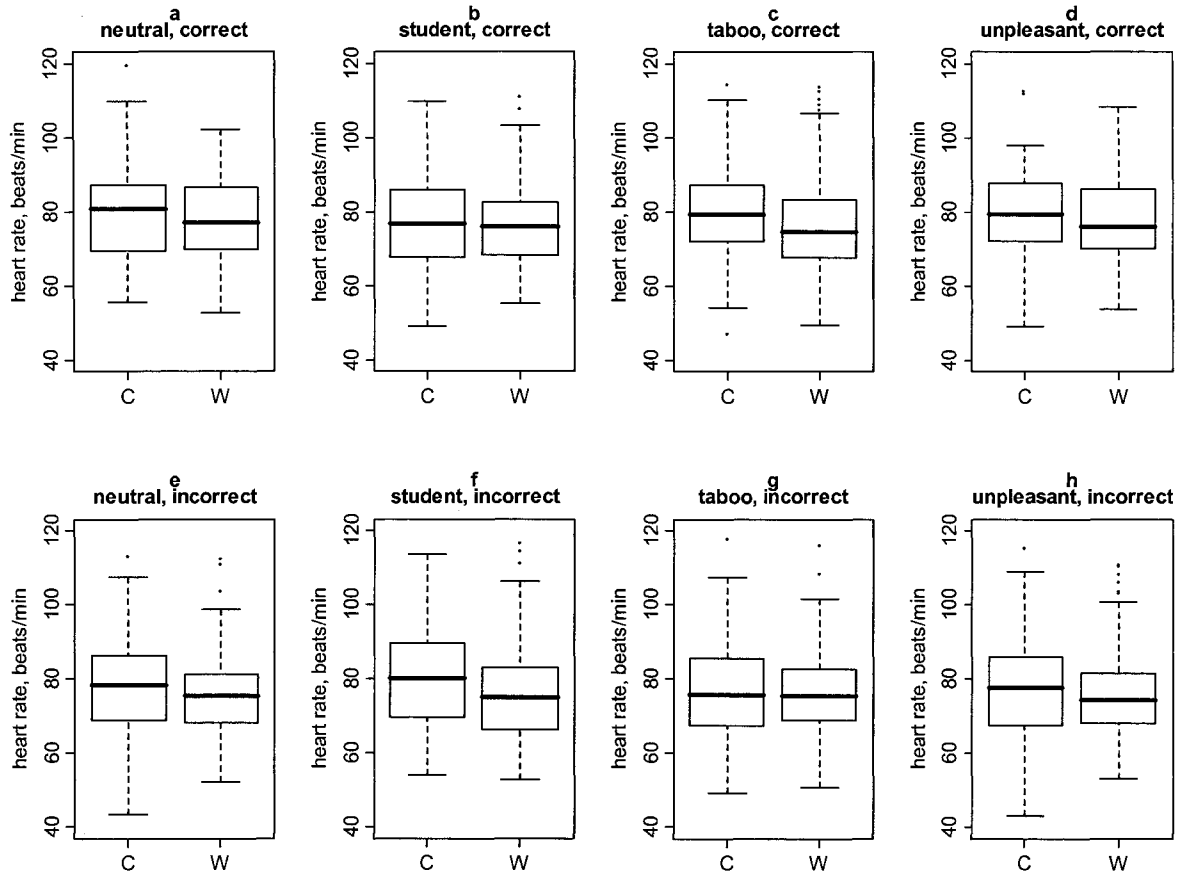
Figure 6.32. Mean heart rate at stimulus onset, by word type, condition, and immediate recall. Pane a: remembered neutral words. Pane b: remembered student words. Pane c: remembered taboo words. Pane d: remembered unpleasant words. Pane e: forgotten neutral words. Pane f: forgotten student words. Pane g: forgotten taboo words. Pane h: forgotten unpleasant words.

Table 6.15. Mean (standard deviation) and median of mean heart rate change in each 500 msec bin after the trigger, by word type, condition, and immediate recall.

| bin	warm correct, taboo mean (sd)	mdn	warm correct, unpleasant mean (sd)	mdn	warm correct, school mean (sd)	mdn	warm correct, neutral mean (sd)	mdn
0	-0.25 (2.56)	-0.31	-0.56 (3.21)	-0.65	-0.37 (3.99)	-0.36	-0.09 (2.47)	0.16
1	-0.48 (3.98)	-0.16	-0.94 (4.11)	-1.25	-0.67 (5.99)	-0.62	-0.51 (3.63)	-0.56
2	-0.39 (4.44)	-0.03	-1.33 (5.15)	-1.84	-0.62 (5.84)	-0.55	-0.81 (4.08)	-1.14
3	0.01 (5.08)	0.35	-1.36 (6.04)	-1.27	-0.60 (5.59)	-0.34	-1.48 (5.83)	-1.05
4	0.21 (5.38)	-0.25	-1.37 (6.80)	-0.67	-1.01 (6.17)	-0.48	-2.25 (7.30)	-1.10
5	0.39 (5.66)	0.45	-1.13 (6.46)	-0.59	-0.90 (6.77)	-0.63	-2.05 (7.21)	-0.36
6	0.82 (6.17)	1.28	-1.19 (6.28)	-0.61	-0.38 (6.76)	0.43	-1.63 (7.71)	-0.19
7	1.18 (6.11)	1.41	-0.95 (6.70)	-0.93	0.12 (7.15)	0.28	-1.17 (8.45)	0.12
8	1.66 (6.08)	1.52	-0.79 (6.95)	-0.67	0.44 (7.16)	0.80	-0.82 (8.68)	-0.24
9	1.55 (6.12)	1.68	-0.33 (7.29)	-0.92	0.51 (7.16)	1.02	-0.56 (8.81)	-0.79

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Table 6.15, continued.

	warm		warm		warm		warm	
	incorrect, taboo		incorrect, unpleasant		incorrect, school		incorrect, neutral	
bin	mean (sd)	mdn	mean (sd)	mdn	mean (sd)	mdn	mean (sd)	mdn
0	-0.35 (3.44)	-0.39	-0.36 (2.77)	-0.54	-0.65 (3.87)	-0.26	-0.36 (2.58)	-0.34
1	-1.00 (4.37)	-0.56	-0.89 (3.85)	-0.71	-1.08 (5.51)	-0.83	-0.75 (3.47)	-0.85
2	-1.09 (4.96)	-0.42	-0.99 (4.45)	-0.43	-1.09 (5.99)	0.11	-0.45 (4.48)	-0.64
3	-1.13 (5.81)	-0.33	-0.93 (4.81)	-0.44	-1.04 (5.88)	0.07	-0.28 (4.86)	-0.26
4	-0.64 (6.17)	0.22	-0.44 (5.09)	-0.28	-0.70 (5.95)	0.31	0.19 (5.40)	0.32
5	-0.42 (6.62)	0.19	0.13 (5.30)	-0.42	-0.52 (6.38)	0.25	0.95 (5.77)	0.75
6	-0.08 (7.03)	-0.50	0.36 (5.56)	-0.34	-0.16 (6.52)	0.21	1.46 (6.35)	1.17
7	0.23 (7.21)	0.49	0.68 (6.06)	0.20	0.35 (7.21)	0.57	1.51 (7.10)	0.91
8	0.13 (7.92)	0.59	1.03 (6.34)	0.00	0.56 (7.72)	0.71	1.84 (7.44)	1.38
9	0.03 (7.74)	0.35	1.79 (6.48)	1.12	0.87 (7.83)	0.53	1.51 (7.62)	0.86

	cold		cold		cold		cold	
	correct, taboo		correct, unpleasant		correct, school		correct, neutral	
bin	mean (sd)	mdn	mean (sd)	mdn	mean (sd)	mdn	mean (sd)	mdn
0	-0.38 (4.08)	-0.12	-0.04 (4.07)	0.02	-0.97 (4.13)	-0.93	-0.03 (3.44)	0.09
1	-0.70 (5.18)	-0.95	-1.02 (5.73)	-1.03	-0.76 (6.12)	-0.36	-0.06 (5.19)	0.22
2	-0.63 (6.04)	-0.82	-1.59 (6.53)	-0.93	-0.47 (6.86)	0.09	-0.25 (6.31)	0.18
3	-0.70 (6.00)	-0.71	-1.87 (6.93)	-0.95	-0.31 (7.29)	-0.05	0.09 (6.89)	0.75
4	-0.13 (6.18)	-0.21	-1.66 (6.98)	-1.73	0.36 (7.33)	0.28	0.48 (6.94)	0.58
5	0.29 (6.53)	0.04	-1.08 (7.16)	-0.49	0.61 (7.25)	0.25	1.24 (7.08)	0.74
6	0.50 (6.80)	0.09	-0.75 (7.17)	0.02	0.77 (6.74)	0.59	1.87 (6.99)	1.08
7	0.70 (7.07)	-0.15	-0.59 (7.14)	0.35	1.16 (6.85)	1.00	1.94 (6.86)	1.65
8	0.67 (7.04)	-0.16	-0.47 (7.36)	0.18	1.52 (7.25)	1.56	2.06 (7.32)	1.95
9	0.71 (6.94)	0.17	-0.81 (7.61)	-0.31	1.97 (7.72)	1.25	1.66 (7.63)	1.37

	cold		cold		cold		cold	
	incorrect, taboo		incorrect, unpleasant		incorrect, school		incorrect, neutral	
bin	mean (sd)	mdn	mean (sd)	mdn	mean (sd)	mdn	mean (sd)	mdn
0	-0.86 (4.27)	-0.76	-0.22 (3.80)	0.04	-0.25 (3.65)	-0.36	-0.34 (3.77)	-0.41
1	-1.31 (6.21)	-0.82	0.13 (5.11)	0.43	-0.09 (5.64)	0.03	-0.28 (5.35)	-0.51
2	-1.70 (7.81)	-1.04	0.75 (5.80)	1.44	0.17 (7.15)	0.17	0.24 (6.34)	0.13
3	-1.49 (8.57)	-0.24	1.31 (6.35)	1.92	0.20 (7.63)	0.55	0.71 (6.96)	0.65
4	-0.53 (8.36)	0.51	1.81 (6.65)	2.04	0.20 (7.81)	0.67	1.02 (7.47)	1.40
5	0.05 (7.90)	0.65	2.60 (6.98)	2.79	-0.09 (7.41)	0.35	1.35 (7.65)	1.23
6	0.51 (7.82)	1.16	3.11 (7.15)	3.21	-0.04 (7.55)	-0.01	0.84 (8.13)	0.69
7	0.75 (8.17)	1.12	3.42 (7.28)	2.95	-0.07 (7.93)	-0.09	0.83 (8.84)	1.21
8	0.87 (8.06)	0.37	3.49 (7.85)	2.95	0.23 (7.99)	0.55	0.53 (8.96)	-0.07
9	0.36 (7.81)	-0.48	2.87 (7.62)	2.57	0.12 (8.20)	0.85	0.48 (8.77)	0.17

Table 6.16. Mean (standard deviation) and median of mean heart rate change in each 500 msec bin after the trigger, by word type, condition, and delayed recall.

bin	warm correct, taboo		warm correct, unpleasant		warm correct, school		warm correct, neutral	
	mean (sd)	mdn	mean (sd)	mdn	mean (sd)	mdn	mean (sd)	mdn
0	-0.11 (2.56)	-0.05	-0.62 (2.98)	-0.66	-0.48 (4.09)	-0.46	-0.25 (2.57)	0.11
1	-0.38 (3.81)	-0.12	-0.94 (3.73)	-1.25	-0.69 (6.22)	-0.96	-0.62 (3.81)	-0.56
2	-0.45 (4.36)	-0.22	-1.56 (4.61)	-2.22	-0.64 (6.06)	-0.72	-0.87 (4.17)	-1.39
3	-0.17 (5.28)	0.15	-1.66 (5.30)	-1.23	-0.58 (5.66)	-0.37	-1.60 (6.01)	-1.21
4	-0.11 (5.83)	-0.32	-1.59 (6.26)	-0.64	-0.97 (6.38)	-0.95	-2.29 (7.51)	-0.02
5	0.03 (6.19)	0.05	-1.28 (6.22)	-0.59	-0.97 (6.98)	-0.72	-2.14 (7.43)	-0.06
6	0.49 (6.70)	0.88	-0.87 (6.39)	-0.61	-0.37 (6.90)	0.50	-1.73 (8.07)	0.09
7	0.79 (6.77)	0.90	-0.61 (6.88)	-0.72	0.20 (7.42)	0.28	-1.25 (8.86)	0.12
8	1.30 (6.99)	1.19	-0.31 (7.17)	-0.50	0.42 (7.42)	0.34	-0.82 (9.14)	0.31
9	1.21 (6.86)	1.10	0.01 (7.46)	-0.92	0.40 (7.45)	0.73	-0.20 (9.24)	-0.47

bin	warm incorrect, taboo		warm incorrect, unpleasant		warm incorrect, school		warm incorrect, neutral	
	mean (sd)	mdn	mean (sd)	mdn	mean (sd)	mdn	mean (sd)	mdn
0	-0.58 (3.34)	-0.54	-0.33 (2.95)	-0.54	-0.52 (3.79)	-0.17	-0.28 (2.54)	-0.14
1	-1.12 (4.58)	-0.52	-0.89 (4.09)	-0.71	-1.02 (5.35)	-0.67	-0.69 (3.41)	-0.83
2	-0.91 (5.04)	-0.13	-0.87 (4.82)	-0.48	-1.01 (5.78)	0.11	-0.45 (4.42)	-0.49
3	-0.67 (5.46)	0.27	-0.78 (5.36)	-0.58	-1.00 (5.79)	0.12	-0.31 (4.85)	-0.26
4	0.02 (5.34)	0.55	-0.37 (5.58)	-0.31	-0.77 (5.77)	0.31	0.04 (5.48)	0.32
5	0.30 (5.64)	0.56	0.14 (5.53)	-0.47	-0.51 (6.21)	0.25	0.78 (5.81)	0.75
6	0.60 (6.04)	1.03	0.07 (5.58)	-0.34	-0.19 (6.42)	0.14	1.28 (6.32)	1.06
7	1.03 (5.96)	1.15	0.38 (6.05)	0.17	0.25 (6.96)	0.57	1.36 (7.04)	0.91
8	0.97 (6.32)	1.27	0.64 (6.32)	-0.21	0.59 (7.43)	0.88	1.65 (7.35)	1.10
9	0.81 (6.43)	1.08	1.45 (6.50)	1.02	0.93 (7.51)	0.71	1.23 (7.56)	0.53

bin	cold correct, taboo		cold correct, unpleasant		cold correct, school		cold correct, neutral	
	mean (sd)	mdn	mean (sd)	mdn	mean (sd)	mdn	mean (sd)	mdn
0	-0.38 (4.17)	-0.12	0.27 (3.69)	0.12	-0.98 (4.19)	-0.76	0.13 (3.25)	0.08
1	-0.65 (5.24)	-0.91	-0.57 (5.35)	-1.02	-0.80 (5.86)	-0.24	0.05 (5.09)	0.05
2	-0.56 (6.24)	-0.82	-1.23 (6.36)	-1.13	-0.54 (6.64)	0.31	-0.11 (6.20)	0.16
3	-0.78 (6.31)	-1.21	-1.61 (6.91)	-1.02	-0.44 (6.86)	-0.13	0.27 (6.70)	0.75
4	-0.17 (6.39)	-0.38	-1.37 (7.02)	-1.35	0.38 (6.97)	0.28	0.90 (6.68)	0.96
5	0.23 (6.63)	0.04	-0.63 (7.33)	-0.10	0.69 (7.04)	0.25	1.70 (6.64)	0.89
6	0.46 (6.77)	-0.07	-0.64 (7.57)	-0.01	0.93 (6.80)	0.77	2.36 (6.62)	1.63
7	0.62 (6.99)	-0.21	-0.59 (7.52)	0.33	1.31 (7.02)	1.34	2.41 (6.65)	2.17
8	0.55 (7.11)	-0.12	-0.37 (7.61)	-0.21	1.79 (7.52)	1.67	2.59 (7.08)	2.35
9	0.61 (7.06)	0.11	-0.58 (7.71)	-0.61	2.22 (7.99)	1.58	2.22 (7.29)	2.19

Table 6.16, continued.

	cold		cold		cold		cold	
	incorrect, taboo		incorrect, unpleasant		incorrect, school		incorrect, neutral	
bin	mean (sd)	mdn	mean (sd)	mdn	mean (sd)	mdn	mean (sd)	mdn
0	-0.78 (4.10)	-0.76	-0.34 (3.98)	-0.01	-0.29 (3.63)	-0.60	-0.40 (3.82)	-0.41
1	-1.29 (5.99)	-0.95	-0.19 (5.39)	0.35	-0.11 (5.93)	-0.20	-0.33 (5.38)	-0.50
2	-1.65 (7.30)	-0.91	0.35 (6.14)	1.03	0.18 (7.31)	0.00	0.16 (6.39)	0.18
3	-1.26 (7.88)	-0.16	0.87 (6.55)	1.66	0.27 (7.95)	0.55	0.62 (7.04)	0.65
4	-0.41 (7.81)	0.51	1.32 (6.81)	1.77	0.20 (8.07)	0.87	0.82 (7.55)	1.23
5	0.19 (7.57)	0.65	2.02 (7.10)	2.25	-0.11 (7.57)	0.35	1.15 (7.82)	1.03
6	0.57 (7.72)	0.75	2.66 (7.10)	2.55	-0.12 (7.43)	-0.29	0.66 (8.20)	0.45
7	0.90 (8.13)	1.13	3.00 (7.20)	2.70	-0.11 (7.69)	-0.13	0.66 (8.82)	0.70
8	1.03 (7.80)	0.37	3.03 (7.81)	2.53	0.09 (7.65)	0.44	0.35 (8.95)	-0.22
9	0.56 (7.51)	0.11	2.38 (7.70)	2.22	0.04 (7.87)	0.67	0.27 (8.82)	-0.01

Discussion

This chapter contains an expansion and partial replication of the HR data analyses performed for *The influence of autonomic arousal and semantic relatedness on memory for emotional words* (Buchanan, Etzel, Adolphs, & Tranel, in press). The analysis was expanded by including subjects in both conditions; the published account only considered warm condition subjects. It was hoped that the larger number of subjects would provide more power to identify subtle trends, as well as provide a confirmation of the patterns found in the warm condition subjects. Since the condition manipulation was performed after word viewing, no differences were expected in HR responses between subjects in the two conditions. Condition-related differences in HR responses were identified, however. No HR differences were found in the curves calculated for gender or experiment, nor interactions between word type and gender or word type and experiment, as expected.

The previous analysis (Buchanan, Etzel, Adolphs, & Tranel, in press) had several main findings related to HR, the principal being that deceleration was greater for remembered than forgotten unpleasant and neutral words (strongest for unpleasant words). The opposite trend (greater deceleration for forgotten words) was present in the taboo words, though this trend was only suggested at immediate recall, and no relationship between HR and recall was observed in school words. When both remembered and forgotten words were analyzed together the HR change curves for neutral, school, and taboo words were very similar, but a pronounced deceleration was present for unpleasant words (Fig. 2 in paper, Figure 6.15 pane d in this chapter). Many, but not all, of the main findings from the previous analysis were found here.

Condition Effects

Unexpectedly, consistent differences in HR changes between warm and cold condition subjects were found. Over all words, cold condition subjects exhibited earlier acceleration with less deceleration than the warm condition subjects. When the data was examined for interactions between word type and condition it could be seen that differences were most present in the neutral and unpleasant word responses; the HR change curves for the student words do not show condition-related differences, while a marginal difference in the opposite direction (more deceleration in the cold condition subjects) is present in the taboo words. When the word type and recall data were examined for condition differences the same patterns were found in both groups, but the results are not equivalent for both conditions. In particular, the distinct difference in HR change curves to unpleasant words recalled or forgotten is present in both warm and cold condition subjects, but the difference is larger in cold than warm condition subjects (distinct deceleration to recalled words, acceleration to forgotten words). The curves for words remembered and forgotten were within the null bands for student and neutral words (neutral words somewhat marginal), suggesting that the heart rate responses for these words were not related to recall, in either warm or cold condition subjects. A marginal interaction of recall and HR was observed on taboo words in warm, but not cold, condition subjects.

There is no clear explanation for the finding of different HR responses in warm and cold condition subjects, since the condition manipulation was performed after the word encoding. The experimenter was aware of the condition assignment prior to the subjects' arrival, so it is possible that the experimenter's apprehension at asking the subject to perform the cold pressor task was conveyed to the subject. Experimenter apprehension could have caused anxiety, and so greater HRV, in the subjects. This possibility can not be ruled out in the present case; it would be advisable in the future for the experimenter to be kept blind to the subject's condition until after word viewing to eliminate this possibility. Alternatively, the cold condition subjects could have varied in baseline HR from the warm condition subjects, perhaps causing a difference in change patterns due to floor or ceiling effects. This was not the case in this data, however; subjects' mean HR did not vary with condition.

Recall Effects

When the HR change curves were analyzed by recall for all word types together, it was found that the deceleration was greater and more prolonged for words remembered than for those forgotten, especially at immediate recall. An interaction was found between

recall and word type as well: increased and prolonged deceleration was most apparent for unpleasant words, marginally present for neutral words, and absent for student and taboo words. Among words later forgotten the heart did not accelerate as much as expected to the student words, while among words later remembered the heart decelerated more than expected to the unpleasant words. Analyzing these patterns by gender confirms that the relationship between HR response to unpleasant words and recall is present in both males and females. The marginal finding of a relationship between HR response to neutral words and recall is present in male, but not female subjects.

Evoked Cardiac Responses and the HR Component of the Orienting Response

Distinct HR changes often occur in conjunction with stimuli provoking orienting responses, but there is disagreement as to a necessary HR component to orienting responses (OR), and, if it exists, the form of that component. The primary view appears to be that a transitory HR deceleration is part of the orienting response, although it may not show the same habituation effects as does SCR (e.g. Cook & Turpin, 1997; Niepel, 2001; Ohman, Hamm, & Hugdahl, 2000; Stekelenburg & van Boxtel, 2002). Others argue that HR changes are not part of the orienting response but rather reflect general stimulus recognition (e.g. Barry, 1996; Barry & Maltzman, 1985; Binder, Barry, & Kaiser, 2005).

When HR changes are considered an integral part of the OR, theory predicts that they take the form of a transitory HR deceleration (Ohman, Hamm, & Hugdahl, 2000). As the amount of stimulus complexity or interest increases the amount of HR deceleration increases (Ohman, Hamm, & Hugdahl, 2000). More intense stimuli (such as tones at an uncomfortably loud volume) produce HR acceleration, as part of the defense reflex; intermediate stimuli produce bi- or tri-phasic HR changes (reviewed in Cook & Turpin, 1997; Ohman, Hamm, & Hugdahl, 2000). OR studies using pictures as stimuli generally find HR deceleration, but highly aversive pictures (suggesting personal danger) produce HR acceleration, such as in phobic subjects exposed to pictures of the sources of their phobia (reviewed in Cook & Turpin, 1997). Abrupt, transitory increases in HR are also associated with startle stimuli (reviewed in Cook & Turpin, 1997; Ohman, Hamm, & Hugdahl, 2000).

The term *evoked cardiac response* (ECR) is used to describe transient HR changes occurring after brief stimuli (also called post-stimulus primary bradycardia) by those that question the inclusion of a HR deceleration in the OR (e.g. Binder, Barry, & Kaiser, 2005). Two forms of ECR are described, the first a deceleration (ECR1) and the second an acceleration (ECR2) (Kaiser, Beauvale, & Bener, 1997). As described by Barry (Barry, 1996; Kaiser, Beauvale, & Bener, 1997), ECR1 is a brief (seconds after stimulus onset) mild

(perhaps 1 beat per minute) heart rate deceleration seen after "irrelevant" stimuli; stimuli such as tones that do not require cognitive processing. ECR2s are produced following "relevant" stimuli, stimuli that require a cognitive task, such as tones that must be counted. ECR2 is dominated by a brief (seconds after stimulus onset) mild (perhaps 2 beats per minute) HR acceleration. ECR2 only occurs in conjunction with ECR1; their combination produces a biphasic HR curve, of which the ECR1 component reflects automatic processes which register the stimulus while the ECR2 component reflects cognitive processing, with more intensive processing producing greater acceleration (Barry, 1996; Kaiser, Barry, & Beauvale, 2001).

Overall HR Responses

The overall HR response pattern found in this study may be interpreted in terms of ECRs and the HR component of the OR, but neither framework fully explains the results. When averaged over all words, the HR change pattern is a small, transitory deceleration followed by a more prolonged acceleration (see Figure 6.2, pane d). This pattern is seen in both males and females and for words of each type independently. This curve is consistent with that expected for a joint ECR1-ECR2 response: the initial deceleration reflects stimulus occurrence while the acceleration reflects cognitive processing (in this case reading and attempting to encode the word). The overall response curve found here is somewhat similar in duration and magnitude to ECRs using relevant auditory stimuli (e.g. Kaiser, Barry, & Beauvale, 2001; Kaiser, Beauvale, & Bener, 1997). The overall HR response curve shape is somewhat more difficult to explain in the framework of OR, because the response was dominated by HR acceleration, not deceleration as would be predicted by a simple OR. The stimuli were not aversive nor unusually intense, and so should not have triggered acceleratory defense or startle reflexes.

The same overall HR response pattern was found to words of each type (small, transitory deceleration followed by a more prolonged acceleration, see Figure 6.7, pane d). HR acceleration was marginally earlier and greater than expected to neutral words; the HR response patterns did not vary for the other word types. Under ECR theory this finding may be explained by an increased cognitive load invoked in an effort to encode the neutral words (which were least often recalled, see Table 6.1). It may have been expected that a larger OR (and so increased or prolonged HR deceleration) would have been caused by the unpleasant or taboo words, since some of these words were more arousing, aversive, or surprising than the student or neutral words. HR deceleration differences to words of different type were not observed, however.

Recall-Related HR Responses

This study's recall findings are not easy to explain in either the OR or ECR framework. It may have been predicted that increased cognitive load (and therefore increased and earlier HR acceleration) at encoding would be associated with improved recall, since the additional cognitive processing could have resulted in improved encoding. Or, it could have been predicted that words causing an emotional reaction would provoke a larger OR (and so increased and prolonged HR deceleration) and so would be the most recalled. Neither pattern explains the results completely, although when a HR response pattern was associated with recall, HR deceleration was greater and more prolonged for words remembered than for those forgotten, while HR acceleration was greater and earlier for words forgotten.

The HR response curve for remembered unpleasant words takes the form of a pure transient HR deceleration, suggestive of a OR-related HR deceleration (such as to tones in Stekelenburg & van Boxtel, 2002). The HR response curve for correctly recalled words of other types does not take this form however; nor does the HR response curve for all correctly recalled words (Figure 6.11 and Figure 6.12, pane d). The HR response curves for all word types together do show that HR deceleration was greater and more prolonged for words remembered than for those forgotten, however, consistent with OR theory predictions.

The opposite pattern to that suggested by ECR theory was observed in recall of unpleasant (and marginally of neutral) words: increased and earlier HR acceleration was associated with forgotten words, while no consistent HR and recall relationships were observed to taboo and school words (see Figure 6.20 and Figure 6.21). Kaiser, Barry, & Beauvale (2001) warn that the ECR patterns "are related to brain function, not behavioural performance." Perhaps the strong recall-related HR pattern in unpleasant words was due to the emotional or arousing nature of the words, rather than to encoding efforts. This does not explain why the HR response pattern to recalled unpleasant words takes the form of an ECR1, however.

Conclusions

The analyses contained in this chapter replicate one of the primary HR findings from *The influence of autonomic arousal and semantic relatedness on memory for emotional words* (Buchanan, Etzel, Adolphs, & Tranel, in press): recall of unpleasant words was associated with a HR deceleration at encoding, while forgotten unpleasant words were associated with HR acceleration at encoding. Unexpected condition-related differences were also found, however, and the marginal recall-related HR difference to taboo words was not

replicated. The overall HR response pattern is consistent with an ECR, but relative deceleration was associated with recall, closer to OR theory predictions. The presence of neither an ECR nor an OR is sufficient to explain either the recall or word type results.

CONCLUSIONS

The work in this dissertation was centered on the goal of expanding the collection of statistical tools and techniques available to understand psychophysiological data, data collected from monitoring peripheral body activity in order to understand the brain. The body is an incredibly complex entity, composed of numerous systems (including the brain itself), which constantly change to maintain stability in the face of continually altering external and internal conditions. The central nervous system and brain monitors, interprets, and coordinates these changes.

Interactions between the brain and the remainder of the body occur to maintain functioning in the face of mental and physical challenges. Even a relatively simple physiological necessity, such as ingesting sufficient food for energy needs, requires considerable monitoring and cognitive control. Visceral activity is represented in the brain, and conscious sensations of hunger generated when a threshold is crossed. Humans are not aware of this monitoring; we do not contemplate every digestive process. Instead, central circuits which do monitor the body's energy balance and digestive processes trigger conscious thoughts and actions when the need is great enough. The process can work in the other direction as well; thoughts of an especially delicious meal can trigger salivation, even in the absence of food.

The brain monitors all bodily activities, not just those of physiological processes which maintain homeostasis but also those related to thoughts and emotions. Changes in emotional state are accompanied by changes in systems throughout the body, and bodily changes can alter emotional state. Due to this continual interaction, monitoring changes in peripheral activity can provide insight into the functioning of the brain and emotional state.

The research projects described in this dissertation attempt to get a window into the brain's activity by monitoring the same physiological systems that the brain monitors and controls. Only a tiny subset of the information and analysis methods are available to us, however, compared to the enormous amount of information the brain continually processes. Insight is possible, however, by carefully choosing the systems to monitor and using analysis methods which mimic what we know of the brain's activities.

We know that the brain continually monitors many physiological systems, and that it is capable of rapid adjustments in neuronal control. Short-term changes may therefore be especially informative. Also, many systems are controlled by the same networks of neuronal

inputs, and interactions/patterns of activity in multiple systems may provide more information than isolated measures of individual systems. The body interacts so much that looking at only one measure (like HR) is unlikely to provide much information as to the set of changes that accompany any particular state (emotion).

The methods described in this dissertation represent a small step towards the goal of understanding emotion and the larger issue of how the brain and body interact. The permutation testing methods developed here make it is possible to detect smaller and more transitory changes than are detectable using averaged values, as was demonstrated with the data from several different experiments. Much work remains to be done, but this work represents a concerted effort to derive even more information, using the best possible signal processing and analytical techniques, from the recorded signals taking us a little farther towards our goal.

FUTURE WORK

The techniques and analyses presented in the dissertation, far from exhausting the possibilities, create numerous avenues for future research. Additional work will expand the data analyses contained here, and answer remaining questions. Improved data collection methods and choice of systems to quantify may aid in understanding brain-body relationships, as will even more sophisticated and targeted data analysis techniques.

Expansion of Current Findings

Additional analysis of the emotional movie study discussed in Chapter 5 may improve understanding of the current findings. In particular, the respiration recorded during the wedding reception scene should be analyzed to determine whether subjects' respiration became entrained to the music accompanying this scene. Many subjects displayed a change in respiration and heart rate during this period, but it is not possible to determine whether these changes were due to entrainment or to happiness invoked by the movie; only further analysis will separate these two possibilities. Also, the overall analysis could be improved by determining time periods of the movie of particular interest (such as the death scene) then adjusting the bins used for analysis periods to correspond with these scenes of interest. The current analysis used 1-minute bins; in some cases an important scene overlapped several bins. Finally, a different method of quantifying SCL (such as the average skin conductance during a period) or changes over time (such as differences between adjacent bins instead of difference from baseline) may provide additional insights.

Additional analysis of the results from the taboo word study discussed in Chapter 6 may also prove fruitful. In particular, it may be beneficial to include both skin conductance and heart rate responses in the analysis. Performing such a multivariate analysis may make it possible to identify patterns of joint activity that are not present in either measure alone, and clarify whether orienting responses were present, and if so, to recall. Analysis of multiple systems at once also may be helpful because it is more similar to how the brain continually and simultaneously monitors, interprets, and coordinates the interactions of many body systems.

Physiological Measurement and Data Analysis

Additional work may also be performed to determine the optimal methods of physiological data collection as well as the systems chosen for analysis. The use of higher sampling rates and improved electrodes may result in a less noisy signal from which more

accurate measures may be obtained. Control of experiment environments may also help, such as ensuring that experimenters are blind to subject condition and that extraneous noise and distracting stimuli are kept to a minimum. The recording of additional physiological systems, such as gastric activity (electrogastrogram), peripheral muscle tension, or hormone levels may also provide information such that a fuller picture of the patterns of physiological activity can be made. More detailed and complete pictures of physiological activity may make it possible to identify a clear pattern of activity correlated with emotional states, the existence of which is now debated.

Another area of future work is in deriving more accurate measures of physiological activity. For example, including respiration when quantifying EDA may result in a purer index of sympathetic activity, while more sophisticated statistical techniques, such as template matching, may reduce error and noise when identifying SCRs. New HRV measures may be developed to capture certain types of variation.

There is also room for improvement in the permutation testing method introduced here. It would be valuable to fully evaluate the performance of the permutation testing methods, perhaps by comparing the findings of these methods with results obtained by statistical modeling. This may make it possible to estimate the minimum amount of difference necessary to produce a significant result, and to determine the best proportion of the permuted curves to use when calculating the null bands. Also, it should be possible to modify the technique to produce exact p-values, such as by calculating the proportion of permuted lowess curves outside the null bands at the number of places (or by the amount) observed in the actual data. These refinements may make it possible to adjust the method to take into account the multiple comparisons, ensuring that the error does not become inflated.

There is also ample room for improvement in the respiration signal processing algorithm presented in Chapter 3. While this algorithm reliably identifies breaths, its pause quantification performs poorly on some types of signals, requiring manual intervention. Performance may be improved by adjustments to the program, such as by changing the pause calculation thresholds depending on the characteristics of individual breaths, rather than applying the same thresholds to the entire signal. The development of formal criteria for determining when a fluctuation in the signal should be scored as a breath would also benefit many researchers; the determination currently depends on experience and individual judgment.

Another possible area of future research is that of developing methods to search for patterns in multiple measures simultaneously. The brain is capable of such complex

monitoring, and of interpreting the patterns in terms of mental and physiological state. Improved analytical techniques, perhaps using computational neural network classifiers, may enable us to do the same thing, eventually perhaps creating a method of classifying emotional states based on the combination of physiological patterns present at any particular time. Computational science is continually developing new methods of classifying large data sets; these methods should be applied to psychophysiological data analysis as they become available.

Additional Research Questions

The widespread use of auditory stimuli in psychophysiological research makes it important to fully understand entrainment, in order to avoid confounding of results. Entrainment was identified in the study using musical mood induction (Chapter 4), but this study was not designed for the evaluation of entrainment. Future work could investigate the characteristics of music (such as rhythm or dominant melody) that promote entrainment, and if it is possible to separate the characteristics of music that promote entrainment from those that induce mood. An easy method of quantifying the degree to which entrainment occurred would also be beneficial.

A few preliminary experiments have begun to identify specific changes in brain activity which correspond with changes in psychological measures. This type of work, monitoring brain and body activity simultaneously, holds promise for providing data that allows a complete understanding of brain-body interactions.

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