Computer Science and Artificial Intelligence Laboratory

Technical Report

MIT-CSAIL-TR-2007-033 June 12, 2007

The Psychophysiology of Risk Processing and Decision Making at a Regional Stock Exchange

John C. Perry

massachusetts institute of technology, cambridge, ma 02139 usa — www.csail.mit.edu

The Psychophysiology of Risk Processing and Decision Making at a Regional Stock Exchange

by

John Christian Perry

B.S., University of Utah (1996)
S.M., Massachusetts Institute of Technology (2001)
S.M., Massachusetts Institute of Technology (2001)

Submitted to the
Department of Electrical Engineering and Computer Science
in partial fulfillment of the requirements for the degree of

Doctor of Philosophy in Electrical Engineering and Computer Science

at the

MASSACHUSETTS INSTITUTE OF TECHNOLOGY

June 2007

© John Christian Perry, MMVII. All rights reserved.

The author hereby grants to MIT permission to reproduce and distribute publicly paper and electronic copies of this thesis document in whole or in part.

Author .....
Department of Electrical Engineering and Computer Science
May 23, 2007

Certified by .....
Andrew W. Lo
Harris & Harris Group Professor, Sloan School of Management
Thesis Supervisor

Certified by .....
Peter Szolovits
Professor of Computer Science
Professor of Health Sciences and Technology
Thesis Supervisor

Accepted by .....
Arthur C. Smith
Chairman, Department Committee on Graduate Students

The Psychophysiology of Risk Processing and Decision Making at a Regional Stock Exchange

by

John Christian Perry

Submitted to the Department of Electrical Engineering and Computer Science
on May 23, 2007, in partial fulfillment of the
requirements for the degree of
Doctor of Philosophy in Electrical Engineering and Computer Science

Abstract

A longstanding controversy in philosophy is whether decision-making is governed by reason or emotion. I study the role of physiological responses in the decision-making process within the realm of financial markets, where both the environment and decisions—trades—are measurable.

In an experiment performed on a regional stock exchange, my collaborators and I record six different types of physiological signals—skin conductance/galvanic skin response (SCR/GSR), blood volume pulse (BVP), electrocardiogram (ECG), electroencephalogram (EEG), electromyogram (EMG), and temperature (Temp)—of monetarily motivated professionals making high pressure decisions. From these signals I estimate underlying physiological features, such as heart rate, changes in body temperature, and amplitude of SCR, which are proxy for affect. Simultaneously, we record real-time market information which the specialists process and which serves as the basis for their decisions, as well as recording their decisions and outcomes.

In a sample of eight market-makers, I find statistically significant differences in mean skin conductance response and cardiovascular variables during transient market events relative to no-market-event control intervals. In addition, I find a strong relationship between trading decisions and physiological responses. Using regression, I demonstrate that heart rate variability can statistically significantly improve predictions of trading decisions, although not by much.

Thesis Supervisor: Andrew W. Lo
Title: Harris & Harris Group Professor, Sloan School of Management

Thesis Supervisor: Peter Szolovits
Title: Professor of Computer Science
Professor of Health Sciences and Technology

Acknowledgments

> "It is a truth universally acknowledged, that a single man in possession of a good fortune, must be in want of a wife." –Opening line of *Pride and Prejudice* by Jane Austen

I begin with a quote from *Pride and Prejudice*, my wife's favorite book, because my wife represents everything wonderful that has happened to me since my arrival at MIT. We met on MIT's campus near the Kresge BBQ pits, dated on campus, and even currently live on campus.

The next best thing to happen was a product of marriage: our son, Ethan.

In contrast to these happy events, there have been some somber moments as well. My two grandmothers passed away during my tenure here. I miss them, but know that they have gone to a far better place. In addition, my first academic advisor, Al Drake, passed away. I am grateful for my time with him.

One of the few books I have been able to squeeze in amidst the piles of textbooks and academic papers was *The Autobiography of Benjamin Franklin.* I quote from the autobiography: "We sometimes disputed, and very fond we were of Argument... Which disputacious Turn, by the way, is apt to become a very bad Habit, making People often extreamly disagreable in Company... I had caught it by reading my Father's Books of Dispute about Religion. Persons of good Sense, I have since observ'd, seldom fall into it, except Lawyers, *University Men*, and Men of all Sorts that have been bred in Edinborough." (emphasis added) I find that, in some regards, life has not changed much, that some University Men who for whatever reason sour and spoil conversation where we should have had occassion for friendship. However, those men have not been my advisors and close associates.

It goes without saying that I have many friends who deserve much credit. But, I will say it anyway. First and foremost, I would like to thank my wife, Elisa, and her family who made the latter half of the PhD so much more enjoyable than the first half. Second, I would like to thank my mom and dad. They have provided so much support. They were especially supportive when I made the decision to finish the PhD, a decision that was not lightly made.

I owe a great deal to my advisors, Peter Szolovits and Andrew Lo. I cannot imagine working with a better pair. In addition, I have learned much from David Staelin, Lucila Ohno-Machado, Fred Bowman, and Whitney Newey. My favorite Staelinism is that Masters students learn to answer tough questions, but PhD students learn to *ask* tough questions. I hope that I have learned to ask the right questions. Keith Herring helped with the SOON algorithm.

I have enjoyed the company of many people within the Computer Science and Artificial Intelligence Laboratory: Tom Lasko, Choong-Hyun Lee, Caleb Hug, Mark Finlayson, Fern Deoliveira, and Sungho Jo, most of whom hail from the Clinical Decision Making Group. My interactions with the Laboratory for Financial Engineering have been limited to Svetlana Sussman and Dmitry Repin, a former post doc and part mastermind behind the experiment. Finally, Mike Coughlin at the Boston Stock Exchange was gracious to provide much needed assistance.

This research has been generously funded by the National Library of Medicine.

Thank you!

Contents

List of Figures

List of Tables

Chapter 1

Introduction

There is a longstanding debate regarding the relationship of emotion to decision-making. Some argue that rational decisions are emotionless. Advocates of this theory espouse clear thinking, where clear means divorced from emotion. Certainly, there is enough anecdotal evidence that too much emotion is harmful. But others question whether no emotion is beneficial when making decisions (Pixley, 2002).

Research in the cognitive sciences and financial economics suggest an important link between rationality in decision making and emotion (Koenigs and Tranel, 2007; van 't Wout et al., 2006; Shiv et al., 2005; Bechara, 2004; Lerner et al., 2004; Bechara et al., 2003; Sanfey et al., 2003; Loewenstein, 2000; Peters and Slovic, 2000; Keltner and Gross, 1999; Lo, 1999; Elster, 1998; Damasio, 1994; Grossberg and Gutowski, 1987), implying that the two notions are not contradictory but complementary. Moreover, other fields have long established the beneficial role of emotion. For instance, neurobiologists have shown that suppressing the emotional response impairs memorization and that memorization improves when facts are learned in connection with an emotion, although under extreme conditions emotion can impair memory (Bechara et al., 2000; McEwen, 2000; Richards and Gross, 2000; Easterbrook, 1959).

In this study, my collaborators and I have verified the link between the emotional response and decision making experimentally by measuring real-time psychophysiological characteristics, which act as a proxy for emotion, of professional securities traders during real trading sessions. I use the phrases *psychophysiological response*, or more simply physiological response, and *emotional response* interchangeably, although the psychophysi-

ological literature tends to favor the later. Using ambulatory biofeedback equipment, several physiological variables are measured without disrupting the trader's normal work flow. Simultaneously, financial pricing data are captured from which market events are detected. By matching these events with the traders' psychophysiological responses, I am able to link financial risk measures and emotional states. For instance, in a sample of eight traders, I have found statistically significant differences in mean skin conductance response and cardiovascular variables during transient market events relative to no-market-event control intervals.

In studying the link between emotion and decision making under uncertainty, professional securities traders are ideal subjects for several reasons. Because the basic functions of securities trading involve frequent decisions concerning risk/reward trade-offs, traders are almost continuously engaged in the activity that I wish to study. This allows us to conduct our study in vivo and with minimal interference to, and, therefore, minimal contamination of, the subjects' natural motives and behavior. Traders are highly trained and compensated professionals. Therefore, they are likely to be among the most rational decision makers in the general population, hence, making ideal subjects for examining the role of emotion in rational decision-making processes. Finally, due to the real-time nature of professional trading operations, it is possible to construct accurate real-time records of the environment in which traders make their decisions, namely, the market prices of the securities that are traded. Given these records, it is possible to identify market events in parallel with physiological characteristics.

To measure the emotional responses of the subjects, I focus on indirect manifestations through the responses of the autonomic nervous system (Cacioppo et al., 2000). Autonomic nervous system (ANS) responses have two desirable characteristics. First, they can be measured non-invasively and, consequently, do not interfere with the subject's cognition. Second, they occur on the time scale of seconds, which is essential for real-time investigation.

This research extends a proof-of-concept (Lo and Repin, 2002) that showed significant differences in physiological responses during market events. The purpose and design of this (current) research has been to provide insight into the role the emotional response plays in risk processing and decision making, which data was not available from the first experiment.

Related work has studied levels of autonomic activity as a function of task complexity or mental strain. For example, some experiments have considered the link between autonomic

activity and driving conditions and road familiarity for drivers (Healey, 2000; Aronsson and Rissler, 1998; Brown and Huffman, 1972), and the stage of flight (take-off, steady flight, landing) in a jet fighter and its flight simulator (Ylnen et al., 1997; Veltman and Gaillard, 1996; Lindholm and Cheatham, 1983). These studies report relationships between autonomic activity and task complexity. (Picard, 1997) cites findings that indicate that emotions play an essential role in decision making, perception, learning, and more—that is, they influence the very mechanisms of rational thinking. However, the book stops short of providing any new empirical data. Perhaps the most influential set of experiments in this field was conducted in the broad context of an investigation of the role of emotion in decision-making processes (Damasio, 1994). In one of these experiments, skin conductance responses (SCRs) were measured in subjects involved in a gambling task (Bechara et al., 1997). The results indicated that the anticipation of the more risky outcomes led to more SCRs than of the less risky ones. The brain circuitry involved in anticipating monetary rewards has also been localized (Breiter et al., 2001). Other experiments support the significance of autonomic responses during risk-taking and reward-related behavior. They provide more details on brain activation correlates of peripheral autonomic responses and also claim the possibility of discriminating the activity patterns related to changing versus continuing the behavior based on the immediate gain/loss history in a gambling task.

Human computer interaction (HCI) studies have attempted to identify patterns of autonomic activity as a function of emotion. (Peter and Herbon, 2006) surveys several HCI studies including (Ekman et al., 1983), which was one of the first studies to show that different physiological variables—heart rate acceleration, hand temperature, and skin conductance—could be used to differentiate emotions. Other studies, such as (Elton, 2006; Kim et al., 2004; Cowie et al., 2001; Healey, 2000), have attempted to classify specific emotions based on physiological data. Typically, these studies collect data in the lab and then train classifiers based on self-reported emotion labels. Such studies are plagued by a number of problems inherent to self-reporting techniques. However, if emotion recognition proves itself, I can envision a future research agenda studying specific emotions and the decision-making process.

1.1 Road Map

The purpose of this experiment has been to study the relationship between emotional responses and the decision making within financial markets, although it should be possible to extrapolate any findings outside of financial markets. To better understand the relationship between emotional forces and decision making, I use proxies for the emotional response in the form of psychophysiological variables. Professional securities traders are the subjects.

Chapter 2 summarizes research and theories from several fields—Philosophy, Economics, Neuroscience, Artificial Intelligence, Psychology, and Psychophysiology—that relate to the decision-making process.

Chapter 3 describes the physiological variables, or signals, used in this study. The chapter describes the brain's influence on, the characteristics of, and features of each physiological signal. Based on psychophysiological research, I implement about two dozen meaningful, time-based features. I compute each of these features for the different physiological signals (ECG, BVP, SCR, EMG, and Temperature). In addition, I explore frequency-based features, such as periodograms and spectrograms. (Frequency-based features have not received as much attention in the literature. Since I find nothing interesting, I would propose no further investigation of the frequency-based features.)

Chapter 4 describes the experiment that we performed on the Boston Stock Exchange. The Exchange's specialists served as subjects. Financial data, which aims to capture the specialists' external environment, and physiological data, which aims to capture the specialists' internal environment, were collected. I cleansed and preprocessed the data. I synchronized the financial and physiological time series, which were recorded on separate computer systems.

I identify a variety of events from the financial data. I have classified these events as

- **Market events** Market events can be one of six variations: price deviation, spread deviation, return deviation, price trend reversal, spread trend reversal, and volatility.
- **Alerts** Alerts indicate that trades or quotes have taken place on other exchanges.
- **Trading events** Trading events are manually executed trades by the traders (in contrast to automated trades, which are higher in volume because they are executed by the exchange).

- **Profit and loss events** Profit and loss events are instants when the value of the portfolio has achieved large profits or losses or instants at the close of a high volatility or low volatility 60-second interval.

For each of these events, I test for differences in mean values of the feature vector calculated during an interval of, say, 10 seconds immediately following (the "reaction" period) or immediately preceding (the "anticipation" period) an event versus a feature vector calculated during a random, no-event interval. I show that there are, indeed, statistically significant differences in autonomic responses before and after events, as compared to a no-event controls. Some types of events are more significant than others.

Of all the events examined in this experiment, the Trading events may be the most interesting because they represent decisions, the result of an individual acting on information garnered from the environment. As I show in this paper, the specialists experience emotional responses during Trading events. The ensuing question is whether their emotions influence their decisions, for better or worse. To couch the question in the language of probability:

$$\mathrm{P}(trade|market, physiology) = \mathrm{P}(trade|market)?$$

More generally, is $\mathrm{P}(decision|input, emotion) = \mathrm{P}(decision|input)$? In English: does the distribution of decisions vary with emotion? For example, if a specialist takes a large position, is the decision to cover the position correlated or preceded with an emotional response? Does the specialist make the same decision in every situation, regardless of their internal milieu? I attempt to identify types of scenarios that are common enough to be able to test for significance. For instance, it is likely that these situations could be large positions, large gains and losses, or heavy concentration on a security.

In order to get at the answers behind the questions, I build models. One model uses principal components analysis to reduce the dimensionality of the physiological feature space. If the features proposed by the field of psychophysiology are meaningful—and this thesis seems to confirm that they are—then it may be possible to identify which combination of features best captures the emotional response. While many qualitative models of affect exist, a quantitative model could, quite possibly, be the first of a kind. The second model reduces the dimensionality of the input space by sequentially deleting the variable that contributes the least to trade predictions. Chapter 5 describes all these results.

I end with discussion and contributions (Chapter 6).

Chapter 2

Review

Decision making involves the synthesis of a variety of kinds of information: multimodal sensory inputs, autonomic and emotional responses, past associations, and future goals. These inputs must be integrated with information about uncertainty, timing, cost-benefit, and risk and then applied to select appropriate actions. In addition, decisions must be completed rapidly and must retain some degree of flexibility. Many fields of research have theorized, experimented, and modeled rationality and decision-making: Philosophy (2.1), Economics (2.2), Neuroscience (2.3), Artificial Intelligence (2.4), Psychology (2.5), and Psychophysiology (2.6). I briefly highlight contributions from each of these fields.

2.1 Philosophy

Most of the great classical philosophers—Plato, Aristotle, Spinoza, Descartes, Hobbes, Hume—had recognizable theories of emotion, conceived as responses to certain sorts of events of concern to a subject, triggering bodily changes and typically motivating characteristic behavior.

Some of the ancient philosophers, such as the Epicureans and the Stoics, believed that emotions are irrational. According to (de Sousa, Spring 2003), "the Stoics adapted and made their own the Socratic hypothesis that virtue is nothing else than knowledge, adding the idea that emotions are essentially irrational beliefs."

2.2 Economics

Economics, a field which grew out of philosophy, is the study of human behavior and choice under resource constraints, incentives, and/or uncertainty. Traditionally, economists used the idea of rationality to describe behavior, where rationality is the concept that individuals make decisions by maximizing some target function—a utility function—given all of the knowledge they currently possess.

There has been much ink spilt in economics debating whether Man is rational, bounded rational[1] (Kahneman, 2002; Hanoch, 2002), or even irrational, where irrational can mean emotional. For instance, Herbert Simon offered a model of decision-making that places rationality at the apex and emotions at the nadir. He hypothesized that analytical decisions are rational and logical, that intuitive and judgmental decisions are non-rational, and that emotional decisions are irrational. Moreover, decisions that can be expressed are logical, whereas those that cannot be expressed are non-logical (more on tacit knowledge below). Ironically, some, such as Kahneman and Tversky[2], have suggested that psychological elements—such as emotion—make individuals more efficient at decision-making, where efficient need not be optimal; hence, the phrase *bounded rationality*.

In finance, this debate usually centers on the Efficient Markets Hypothesis which asserts that asset prices "fully reflect all available information" (Samuelson, 1965). The underlying assumption of the efficient markets hypothesis[3] is that investors have rational expectations; that is to say that investors make decisions using the rules of probability and statistics. The market, speaking collectively of the investor population and not individually, is correct in that any informational advantages that might exist among any members of the investment community are eventually traded away. Certainly, there may be individuals who overreact or underreact, which may still meet rational expectations by correctly doing their math.

Critics of the efficient markets hypothesis argue that investors are often, if not always,

[1]The term *bounded rationality* was coined by 1978 Nobel Prize winner Herbert Simon. Simon pointed out that bounded rational individuals are unable to compute an optimal solution within a reasonable time frame and thus *satisfice*, or choose actions that are satisfactory but suboptimal.

[2]Daniel Kahneman shared half of the 2002 Nobel Prize in Economics for "decision-making under uncertainty." Amos Tversky may have shared part of the prize but had passed away.

[3]The efficient markets hypothesis is a modern incarnation of a much older idea. For instance, Francis Galton's *Vox populi* precedes the efficient markets hypothesis by more than half a century. Galton described a weight-judging competition at the West of England Annual Fat Stock Show at Plymouth (1906). The competition was to guess the weight of a fat ox which had been slaughtered and dressed. The average of 787 guesses originating from the crowd were more accurate than most of the individual guesses from the crowd as well as being more accurate than any guesses from the so-called cattle experts.

irrational, exhibiting predictable and financially ruinous biases. Behavioral finance is the branch of finance devoted to the study of these biases. It often applies lessons from psychology to financial decision making. Most of behavioral finance focuses on cognitive biases. By way of contrast, this experiment focuses on emotional responses.

In economics, rationality is defined in terms of utility, where utility is a map from choices to a measure of happiness or satisfaction. Maximizing a utility function is to choose the choice that maximizes happiness. The modern history of the utility function begins with Daniel Bernoulli, who first proposed expected utility as a solution to the St. Petersburg paradox. The St. Petersburg paradox presents two choices: $40 or a lottery ticket that pays according to the outcomes of one or more fair coin tosses: heads you get $2 and the game ends, tails you get another toss and the game repeats, but now if the second toss lands heads up you get $4, and so on. If the nth toss is the first to land heads up, you get 2^n dollars. The game continues, however long it takes, until the coin lands heads up. The expected value of this lottery is infinite:

$$\begin{aligned} \text{Expected value} &= (0.5 * 2) + (0.25 * 4) + (0.125 * 8)... \\ &= 1 + 1 + 1... \end{aligned}$$

Since most people are willing to pay only $40 to play a game whose expected value is infinite, Bernoulli realized that a theory based on expected value fails to explain human behavior. Economists now explain this behavior by assuming that the desirability of money does not increase linearly, but rather grows more and more slowly as the total amount at stake increases.

Although expected utility solved the St. Petersburg paradox, subsequent paradoxes, such as the Allais paradox and the Ellsberg paradox, now plague expected utility theory. In Ellsberg's paradox (Ellsberg, 1961) an urn contains 90 balls. Of these, 30 are blue, and 60 are either red or yellow. You are then offered a choice between a lottery that pays $100 if a blue ball is drawn (a 1/3 probability) and one that pays $100 if a red ball is drawn. The probability of a red draw is unspecified or ambiguous—it is a choice between an event with a known probability and an event with an unknown probability. Under these circumstances, individuals typically choose the first lottery, which wins if a blue ball is drawn. According to expected utility theory individuals could only do so if they believe that there are fewer

than 30 red balls in the urn or, equivalently, that there are more than 30 yellow balls. Then (before any balls are actually drawn, but with the same urn standing in front of you) you are asked to choose again, this time between a lottery that pays $100 if either blue or yellow is drawn and one that pays $100 if either red or yellow is drawn. The likelihood of winning is now clear in the second case (a 2/3 probability of winning $100) but unclear in the first case (a probability between 1/3 and 1). Individuals typically choose the second lottery. The first lottery seems less attractive, because there might be too few yellow balls. What's the paradox? If expected utility theory is correct, you cannot think that there are too few and too many yellow balls in the urn at the same time.

Given the problems with expected utility theory, alternatives have been proposed, such as Prospect Theory (discussed later in Section 5.4). Neuroscience, discussed next, is another field studying utility, but from a different approach.

2.3 Neuroscience

Neuroscience addresses the issue of decision-making and risk processing from a neurological perspective. It views the brain and the nervous system as circuits that determine behavior by processing stimuli and the evoking bodily responses. Neuroscience deals with the nervous system's anatomical, physiological, and biochemical structures. I include a brief summary of such structures before continuing the discussion of rationality.

The brain is divided into the hindbrain, the midbrain, and the forebrain. The hindbrain (rhombencephalon) is located in the bottom portion of the brain and is an extension of the spinal cord. It functions to serve automatic activities such as breathing. The midbrain (mesencephalon) lies between the hindbrain and the forebrain and plays a role in emotional feelings.[4] The forebrain (prosencephalon) is the largest part of the brain and is the most highly developed portion of the brain, responsible for cognitive and mental processes like thinking and language.

The major components of the forebrain are the cerebrum, the limbic system, the corpus callosum, the thalamus, and the hypothalamus. The cerebrum is the largest part of the forebrain and is responsible for complex mental activities. Its outer surface is the cerebral cortex. The cerebrum contains four major lobes: (1) the frontal lobe that controls voluntary

[4]Walter Rudolf Hess received the Nobel Prize in 1949 for mapping various functions of the midbrain.

movement and includes the motor cortex; (2) the parietal lobe that contains the primary somatosensory area that manages skin senses; (3) the occipital lobe that contains the visual cortex; and (4) the temporal lobe that contains the auditory cortex. The limbic system relates structures such as the amygdala and hippocampus that control emotion, motivation, and memory. The corpus callosum is a communication network that connects the right and left hemispheres. The thalamus relays and translates information from all of the senses, except smell, to the higher levels in the brain. The hypothalamus helps to regulate basic biological drives by controlling autonomic functions.

Research on the neural basis of emotions has been dominated by the limbic system theory, which was initially proposed by Paul MacLean. MacLean added the amygdala, septum and prefrontal cortex to the *Papez circuit*, which is a pathway involved in the cortical control of emotion and which also plays a role in storing memory.

By understanding the structures just described, cognitive neuroscientists seek to explain a global theory of choice that is not plagued with paradoxes like expected utility theory (Glimcher and Rustichini, 2004). For example, decision-makers may be subject to physiological processes, which processes economists have overlooked and which may account for unexplained differences. The primary methods used to study decision making in neuroscience are tasks for patients with brain lesions, experiments such as gambling that pit risk vs. reward, neuroimaging, and models of decision making. The following paragraphs briefly survey each method.

Subjects with brain lesions Studying patients with brain lesions, Antonio Damasio has amassed an impressive body of neurological evidence suggesting that emotions do, indeed, aid in everyday reasoning. His patient Elliot is the poster child who was unable to make decisions after losing parts of his frontal lobes (Damasio, 1994). Frontal lobes are part of the brain's structures that control emotions. Following treatment, Elliot still appeared intelligent, but struggled with making certain kids of decisions and planning work. Damasio wrote that "reduced emotion and feeling might play a role in Elliot's decision-making failures."[5] These observations and others like them have led Damasio to propose that the inability of patients with brain lesions to make advantageous decisions under some circumstances is caused by damage to structures that mediate emotion and that store and

[5]While Damasio points the causal direction from reduced emotion to poor decision-making, others, such as (Minsky, 2006), reverse the direction, meaning that the inability to make decisions reduces emotions.

signal the value of future consequences of an action.

Gambling experiments Experiments that pit risk vs. reward, as in the Ellsberg paradox, have confirmed the importance of the frontal lobes for decision-making. (Bechara et al., 1994) uses a gambling experiment, called the Iowa gambling task, to study decision making. The gambling task requires participants to repeatedly choose from four decks of cards with the goal of winning as much play money as possible. Each card is associated with a win, and some cards also carry losses. Overall, choosing from two of the decks results in larger wins but even larger losses, whereas choosing from the other two results in small wins but even smaller losses. As participants progress through the 100 trials, they gradually learn to avoid the riskier decks and choose more often from the lower stakes, overall advantageous decks. The study reported that participants with brain lesions performed quite differently, persistently choosing more often from the riskier (ultimately disadvantageous) decks. (Koenigs and Tranel, 2007) use the Ultimatum Game to study decision making. Ultimatum is a game in which two players are given one chance to split a sum of money. The first player offers a portion of the money to the second player. The second player can accept the offer (in which case both players split the money as proposed) or reject the offer (in which case both players get nothing). Expected utility theory would predict that any proposal should be accepted. However, experiments show that low offers are consistently rejected. The rejection rate of players with brain lesions is even higher than of the control group, which suggests that emotion regulation processes are a critical component of normal decision making.

Neuroimaging Neuroimaging, such as functional magnetic resonance imaging, is a recent technique used in studying decision making in the face of uncertainty. Several fMRI studies show that different regions of the brain—including the frontal lobes—are activated when contemplating risk and reward (Tom et al., 2007; Hampton and O'doherty, 2007; Phan et al., 2002; O'Doherty et al., 2001). (Sanfey et al., 2003) studied the fMRI of subjects playing the Ultimatum Game. Players who refused offers experienced activation of brain circuits associated with emotional arousal. Once again, expected utility theory fails to explain observed behavior in which aroused neural circuits are associated with decisions to reject utility-enhancing offers.

Models of decision making Models of decision making range from the oversimplified to the overly complex. An example of a simple model is: (1) identify options, (2) evaluate options, and (3) choose option. More complicated models include neurological components, such as models that differentiate between the processing speed of the conscious, explicit reasoning homonid brain and the unconscious mid-brain. This model might postulate that the decision to duck at a 100mph-speed baseball before the conscious brain has had a chance to reason originates in the unconscious brain. Or it might postulate that decisions in complex matters, such as choosing between different houses or different cars, should be left to unconscious thought (Dijksterhuis et al., 2006). See (Fellows, 2004) for a review of all these methods.

2.4 Artificial Intelligence

Artificial Intelligence (AI) is a field that attempts to implement human-level intelligence in computers. Expert systems are one of the tools which AI researchers have used to mimic the intelligence of the humans. A subfield[6] of AI, expert systems aim to capture the intelligence of subject matter experts in a format amenable to computational reasoning. The two main components of an expert system are the knowledge base and the reasoning (inference) engine.

An expert system is only useful if knowledge can be explicitly codified or, in other words, if enough knowledge can be codified to make a program useful. AI researchers have spilt much ink debating just how much knowledge can be codified. For example, (Clancey, 1989) has criticized approaches to expert system development based the assumption that expertise can be captured in overt knowledge: "Knowledge can be represented, but it cannot be exhaustively inventoried by statements of belief or scripts for behaving. Knowledge is a capacity to behave adaptively within an environment; it cannot be reduced to representations of behavior or the environment." Explicit, declarative knowledge may be contrasted with implicit knowledge. Implicit knowledge, or, to use a phrase popularized by Michael Polanyi,

[6]Another subfield of AI is knowledge discovery and data mining. Data mining attempts to model intelligence in a very different fashion than expert systems—by learning from data. Data miners are also interested in rational decision making. The August 2006 Poll at one of the premier data mining internet portals, www.KDNuggets.com, asked the question: Can computers make better decisions by mining historical data than humans who rely on their intuition? Over half of the respondents said usually, unless the conditions change significantly compared to historical data. (See http://www.kdnuggets.com/polls/2006/computers_make_better_decisions.htm)

tacit knowledge, is knowledge that is not readily verbalized, but which may be gained by training or personal experience. As (Polanyi, 1966) wrote, "we can know more than we can tell." For instance, it is much easier to act out habits such as riding a bike than to explain how to ride a bike. In medicine, clinicians are often able to diagnosis patients, but are not able to provide explicit reasons why. In sports and the arts, a performer's tacit knowledge is sometimes called talent (although the word *talent* has nuances that also refer to explicitly developed aptitudes).

The reasoning engine's ability to manage the *frame problem*[7] also weighs on the usefulness of an expert system. The frame problem is the problem of knowing which components of state, or frame, change from one point in time to the next point in time. Given that the number of consequences of any one strategy is possibly infinite, unless some drastic pre-selection can be effected among the alternatives, the evaluation will never terminate. The following illustration should make this problem a bit clearer.

> "Suppose that I wish to arrange an appointment with my doctor, and he suggests two alternative days—say, next Monday or next Tuesday. If I am a rational agent (according to the instrumental view of rationality), I will calculate the expected utility of going to the doctor's on each of these days and choose that which has the higher. Before I can do this, I need to assign a conditional probability $Pr(\omega|x)$ to each outcome, which ω is the outcome and x is the action. Before I can do *that*, however, I must first of all list all the possible outcomes. And, to borrow a phrase, there's the rub; for who knows what 'what dreams may come, when we have shuffled off this mortal coil'—or even what might ensue from arranging to see the doctor on Monday? If I have already agreed to take my kids out to the zoo on Monday, then arranging the appointment for Monday would mean having to reschedule the trip to the zoo. Rescheduling the trip to the zoo would be one outcome of arranging the appointment for Monday, but why stop there? Why not also consider the possible consequences of *that*? My kids might get annoyed with me; I might tell them off for being intransigent; this might lead them to be more flexible, which might help them to be happier later in life.. and so on, *ad infinitum*. Since the relation *being an outcome of* is transitive, it follows

[7]The phrase *frame problem* was coined by John McCarthy in 1969 while developing a temporal reasoning formalism. However, the term is now used in a very general context.

> that all these are also outcomes of arranging the appointment for Monday. So even for a simple decision like arranging an appointment with the doctor, the set of possible outcomes for each action is in principle unbounded. Therefore, listing the possible outcomes of any given action is a potentially endless task. Yet, if I am to make a decision, I must stop listing outcomes at some point." (Evans, 2002)

(LeDoux, 1996) describes a similar example using a confrontation with a bobcat.

2.5 Psychology

Psychology is the study of the mental processes and behavior. Traditionally, it has studied the mind whereas neuroscience has studied the brain, a philosophy of mind known as *dualism*.

An example of a classic psychological hypothesis is that positive affect produces better decisions. In one such study, carried out in a hospital setting, the researchers gave a small gift of candy to one group of physicians in order to induce positive affect and gave nothing to the remaining (control) group of physicians. The physicians then had to read a description of a patient and think aloud (which was recorded and rated by outside coders) as they tried to determine the correct diagnosis. It was found that while the positive affect-induced doctors considered as many diagnoses as the control group of doctors, the positive affect-induced doctors came to the correct solution significantly earlier than control group, and were less likely to incorrectly anchor on an incorrect hypothesis. This study suggests that positive affect can facilitate the thorough, efficient, and flexible use of new information, which increases decision effectiveness. Positive affect, which psychologists postulate is associated with increased levels of dopamine, has also been linked to deeper analytic processing and efficiency in decision making. When quick answers are needed, people in positive moods can respond with an appropriate decision making strategy; if the task requires deeper, more analytic processing, people who are in a good mood recognize this necessity and can do so as well.

To be fair, other psychological studies have concluded just the opposite—that negative affect leads to more effective decision making. One set of studies shows that negative affect leads to more concentrated, detailed, and analytic processing while positive affect can lead

to the opposite. For instance, (Isen, 2001) reviews classical psychological research aimed at answering the same question posed in this paper—what is the role of emotion, or the emotional response in decision-making? The approaches of traditional psychologists are markedly different than approaches of research that blur the boundaries of psychology with economics, neuroscience, and artificial intelligence.

Cognitive psychology has developed theories in parallel to that of Artificial Intelligence with regards to tacit knowledge. This area of research, called *implicit* and *explicit learning*, centers on two debates.

The first debate is whether information is stored in memory in an abstract form or a specific form. The debate between the abstract form and the specific form (also known as exemplar or instance based theories) takes place in the areas of concept representation, syllogistic reasoning, as well as implicit and explicit learning.

The second debate is how much, if any, knowledge results from unconscious processes.

Implicit learning has been characterized as a passive process, where individuals are exposed to information, and acquire knowledge of that information simply through that exposure. Explicit learning, on the other hand, is characterized as an active process where individuals seek out the structure of any information that is presented to them. Some psychologists suggest that much of the information learned during the normal course of life is learned implicitly, not explicitly. They cite activities such as language learning, bicycle riding, and other complex activities, as examples of implicit learning. These are activities that people can do, but that they cannot explain how they do.

(Reber, 1993) studied participants who saw thousands of trials. It should come as no surprise that repetition and immersion were a key to learning. However, a surprising conclusion was that individuals can learn (to utilize) complex structural relationships in data in a completely non-reflective manner. The implications of this conclusion are that if a doctor sees only three patients each day or a trader makes only three trades each day, it could take a long time to acquire expert knowledge. This conclusion also implies that after extensive immersion doctors and traders may be able to diagnose a disease or to sense when a market is likely to fall with relatively few data points—though neither may be able to verbalize why. (It is currently presumed that much of the excessive trading is irrational noise trading. However, this conclusion seems to suggest an explanation of the high level of trading in financial markets. Trading at frequencies higher than what is demanded by

asset portfolio models may generate information, accelerate learning, create commitments and enhance social capital, all of which sustain traders‘ long term survival in the market.)

Psychologists hold different views about human behavior than economists. Psychologists contend that economists' models bear little relation to actual behavior. This view is supported by a large body of psychological research that shows that emotional state can significantly affect decision making. Economists, on the other hand, argue that psychological studies have no theoretical basis and offer little empirical evidence about people's decision-making processes.

That said, some economic psychologists are attempting to bridge the divide between the two domains. (Ackert et al., 2003) argue that emotion has important, and possibly beneficial, influences on financial behavior. The discussion focuses particularly on three aspects of emotion and financial decision making: emotional disposition and stock market pricing, the feeling of regret, and investors' emotional response to information. (Kiev, 1998) has studied and provided counseling for traders. He draws comparisons of traders to Olympic athletes, elite military troops, and performing artists. (Steenbarger, 2002) has also studied traders. In his book, he points out that high-frequency, short-term traders have a very different basis for making decisions than long-term, buy-and-hold traders, otherwise known as asset managers. For instance, short-term traders experience more emotional responses in the trenches vs. the long-term asset managers who experience the markets from more distant vantage points, thus shielding themselves from the minute-to-minute ebbs and flows in the market.

Steenbarger writes about his own trading experiences and those of individuals he has mentored:

> "I have found that if I am loose during the day, I am most likely to be flexible in my thinking and quick in responding. It is when I am tense and take things far too seriously that I am apt to freeze up and compound a situation that has gone sour. One of my trading colleagues, Henry Carstens, frequently marvels over what a great 'game' trading is. It is not surprising to me that he is successful: He plays with trading ideas as spiritedly as he plays with his little son, Everett. Another trading colleague consistently describes trading as a battlefield. It is not surprising that he seems to experience post-traumatic stress with each loss, setting him-and his equity curve-back for considerable periods. Many successful

> traders learnt to keep loose at the trading station in the same way that boxers or ball players stay loose before and during their contests: with uplifting music, banter with friends, and pregame warmup drills. I have recently begun using biofeedback for this purpose...
>
> "Chronic stress is experienced as dis-stress. Anxiety, depression, and anger are common consequences of viewing the world through the lenses of threat. These emotional reactions, in turn, produce typical behavioral consequences, such as indecision, lack of self confidence, impulsivity, and interpersonal conflict. We know from cognitive neuroscience research that high levels of distress shift regional cerebral blood flow away from the frontal cortex–our executive center of judging, planning, and reasoning–and toward motor regions. This is why it is so difficult for people under chronic stress to calmly work out their problems. Their perceptions of threat create physical and emotional arousal, which in turn make it difficult to access the cognitive capacities most needed at those times. Every trader knows how easy it can be to abandon a well thought out trading plan in the heat of adverse market activity!"

As Steenbarger points out, chronic stress can impact an individual's physiology, thus confounding the signal in the emotional response.

2.6 Psychophysiology

Psychophysiology is a cross of psychology and cognitive neuroscience. For the purposes of this paper, psychophysiology deals with innervations beginning in the brain and terminating in the body. I begin by describing a neural model of an emotional response. Based on the model, I identify several physiological features that aim to capture the physiology of the emotional response. Conditions of high motivation accompanied by frustration, failure, or extreme danger are seen as stressful and are accompanied by (negative) emotional responses. On the other hand, conditions of high motivation accompanied by fulfillment and success are seen as non-stressful and are accompanied by (positive) emotional responses.

The nervous system is the body's system that receives and interprets stimuli and transmits impulses to other organs. The cornerstone of the nervous system is the brain. The function of the brain is to receive input from the sensory organs and process these in-

puts. The brain may then formulate a response that is executed by the organs. Scientists have divided the brain into three main parts: brain stem, cerebellum, and cerebrum (telencephalon). The brain stem is responsible for visceral functions, such as heart rate and breathing rate, and serves as a relay station for motor reflexes. It also plays a role in the selection of important information—a cognitive process called *attention*—and in concentration. The cerebellum coordinates the somatic muscle system. The cerebrum is further divided into four lobes: frontal lobe, temporal lobe, parietal lobe, and occipital lobe. It has been hypothesized that the frontal lobe is the lobe most affiliated with thought, problem solving, and emotions.

In addition to the brain, the nervous system's other components include the spinal cord, nerves, ganglia, and the related parts on other organs. The *central nervous system*[8], the subdivision of the nervous system devoted to information processing, consists of the brain and spinal cord. The *peripheral nervous systems* consists of the nervous system that excludes the central nervous system. The peripheral nervous system is further subdivided into the somatic (or skeletal) nervous system and the autonomic nervous system. The somatic nervous system consists of voluntarily controlled bodily functions, such as skeletal muscle. The autonomic nervous system (ANS)[9] consists of involuntarily controlled bodily functions, such as heart muscle, and regulates a wide range of cardiovascular, gastrointestinal, electrodermal, respiratory, endocrine, and exocrine organs. The autonomic nervous system is further subdivided into three complementary systems called the *sympathetic nervous system*, or visceral nervous system, and the *parasympathetic nervous system*, and the *enteric nervous system*. The sympathetic nervous system is synonymous with energy-consuming activities and is also called the "fight-or-flight" system. In contrast, the parasympathetic nervous system opposes the sympathetic nervous system and is synonymous with energy-producing and energy-conserving activities such as salivation, digestion, peristalsis, and bladder contraction and hence is called the "rest-and-digest" system. The enteric nervous system, which can actually function independently of the central nervous system, is a collection of neurons in the gastrointestinal tract that constitutes the "brain of the gut" (Goyal and Hirano, 1996).

The peripheral nervous system functions as the reflex arc. The reflex arc consists of

[8]The first record of the central nervous system was postulated by Galen, a second century Greek physician. He modeled nerves emanating from the brain and distributing "spirit" to the body parts.

[9]John Newport Langley coined the term *autonomic nervous system* at the turn of the 20$^{\text{th}}$century.

(1) sensory organs, which sense the environment, (2) sensory nerves, which communicate messages to the brain, (3) the central nervous system, which acts as the controller, (4) motor nerves, which communicate messages from the brain, and (5) effector organs, which carry out messages from the brain.

The most popular theory hypothesizes that the objective of the sympathetic and parasympathetic nervous systems is to achieve an equilibrium state called *homeostasis*[10]. To achieve homeostasis and regulate the stress response, the sympathetic nervous system controls and stimulates the adrenal gland to manufacture three hormones: cortisol, adrenaline, and norepinephrine.

Cortisol is a corticoid (also called corticosteroid or, in its synthetic form, *hydrocortisone*), which is a steroid hormone manufactured in the adrenal cortex. More specifically, cortisol is a glucocorticoid, so named because of its ability to promote conversion of protein and lipids to usable carbohydrates. The production of cortisol is controlled by a feedback loop. The hypothalamus, a section of the mammalian brain located, not surprisingly, below the thalamus, responds to stress in all its varieties, e.g., emotional, physical, intellectual, etc. The hypothalamus acts as a transducer by linking the nervous system, which uses neurons to relay messages, to the endocrine system, which uses hormones[11] to relay messages. The hypothalamus releases hormones that control a variety of autonomic functions as well as hormones that control the production of yet more hormones in different glands, such as the pituitary gland. For example, corticotropin-releasing hormone is one such hormone that stimulates the pituitary gland to produce adrenocorticotropic hormone (ACTH). ACTH, in turn, stimulates the adrenal cortex to manufacture cortisol. Thus, when stressed the body produces cortisol. The loop is closed because the hypothalamus responds to cortisol levels. This negative feedback loop is called the hypothalamus-pituitary-adrenocortical axis.[12] Cortisol sharpens the memory, increases blood pressure, and increases blood sugar levels (gluconeogenesis), which acts to mobilize energy supplies.[13] Cortisol concentrations

[10]Walter Cannon, author of the Cannon-Bard theory (described later), coined the terms *homeostasis* and *fight or flight*.

[11]The word *hormone* is derived from the Greek word *hormodezein* which means "to arouse."

[12]Hans Selye, the "father" of stress research, defined stress as the presence of hypothalamus-pituitary-adrenocortical activation.

[13]Cortisol produces elevated levels of sugar in the blood (*hyperglycemia*). If prolonged for weeks, insulin, the hormone manufactured by the pancreas to control sugar metabolism, levels will rise. If this situation continues for a long time, sustained elevations of cortisol will lead the body down the path to type 2 diabetes. In the cardiovascular system, the elevation of ANS activity, combined with hyperglycemia and too much insulin (*hyperinsulinemi*) promote both hypertension and harmful metabolic conditions, such as rising blood

follow a circadian cycle and are highest in the early morning and lowest during sleep.

Adrenaline (also called adrenalin™, adrenin, epinephrine, or epinephrin) is a hormone secreted by the adrenal medulla.[14] Compared to cortisol, adrenaline works much faster. Adrenaline increases heart rate and stroke volume, constricts blood vessels in the skin and gastrointestinal tract (vasoconstriction) but dilates blood vessels in the muscles, brain, heart, and lung (vasodilation), increases breathing rate, dilates bronchiole, dilates pupils, contracts muscles, and elevates metabolism. These physiological changes can produce goose bumps (contracted surface muscles), increase blood pressure, and lower temperature on the surface of the skin. High concentrations of adrenaline and other catecholamines at neuroreceptor (adrenergic receptors) sites enable spontaneous and intuitive behavior, thus bypassing the normal circuitry through the brain. Adrenaline is considered a neurotransmitter because it relays signals between neurons and other cells.

Norepinephrine, or noradrenaline, is another hormone (and neurotransmitter) released during stressful events. In conjunction with adrenaline, norepinephrine increases heart rate, increases skeletal muscle readiness, causes vasoconstriction, and decreases appetite.

According to Table 2.1, the hormones of the sympathetic nervous system cause the body to tense, breathe faster, and produce more sweat. Increased levels of hormone ultimately produce a state of anxiety, which manifests itself as difficulty in breathing, muscle tension and cramps, noise sensitivity, severe hand trembling, and loss of appetite.

Dopamine is a hormone often cited for playing a minor role in the emotional response of decision-making. Dopamine[15] is an intermediate in the synthesis of adrenaline and is

cholesterol. In the immune system, which is controlled by the nervous system and by hormones, chronic stress suppresses the ability of the immune system to do its job. In the digestive system, the elevated levels of stress hormones can make the digestive cells in the stomach and intestines extra-sensitive to pain. Consequently, normal contractions and movements can become excruciating. Constipation or diarrhea may follow. This addendum is not exhaustive and does not include magnification effects caused by elevated stress hormones, such as worse ulcers. One possible remedy is Prozac and similar antidepressants, which may actually work on the gut, not the brain. Drugs known as selective serotonin reuptake inhibitors (SSRI) enhance levels of serotonin, which, scientists assume, helps improve mood. Ninety-five percent of the serotonin in the body actually lies within the digestive system. So, it is possible that these drugs boost serotonin in the gut, not the brain.

[14]This research is based on several Nobel Prizes. For instance, one of the first Nobel Prizes was awarded in 1906 to Camillo Golgi and Santiago Ramn y Cajal "for research on the nervous system." Others have been recognized for their contributions to the understanding of the chemicals of the sympathetic nervous systems. For instance, in 1950 Edward Calvin Kendall, Tadeus Reichstein, and Philip Showalter Hench were recognized "for the discovery of the hormones of the adrenal cortex, their structure and function." Sir Bernard Katz (Katz, 1966), Ulf von Euler, and Julius Axelrod's work on nerve, muscle, and synapse was recognized in 1970. In 1971 Earl W. Sutherland, Jr. was recognized "for discovery of the action of hormones, especially epinephrine, via second messengers." Other Nobel Prizes recognized contributions from Roger W. Sperry (1981) "for research on the cerebral hemispheres."

[15]Dopamine was discovered by Arvid Carlsson and Jils-Ake Hillarp in Sweden in 1952. Arvid Carlsson

Structure	Sympathetic Stimulation	Parasympathetic Stimulation
Muscles	Muscles contracted	Muscles relaxed
Iris (eye muscle)	Pupil dilation	Pupil constriction (miosis)
Heart	Heart rate increased	Heart rate decreased
Heart	Force of contraction increased	Force of contraction decreased
Heart	Stroke volume increased	Stroke volume decreased
Blood vessels (skin)	Vasoconstriction	Vasodilation
Blood vessels (brain)	Vasodilation	Vasoconstriction
Lung	Breathing rate increased	Breathing rate decreased
Lung	Bronchiole dilation	Bronchiole constriction
Stomach	Peristalsis reduced	Gastric juice secreted; motility increased
Metabolism	Metabolism increased	Metabolism decreased
Small Intestine	Motility reduced	Digestion increased
Large Intestine	Motility reduced	Secretions and motility increased
Liver	Increased conversion of glycogen to glucose	-
Kidney	Decreased urine secretion	Increased urine secretion
Oral/Nasal Mucosa	Mucus production reduced	Mucus production increased
Salivary Glands	-	Salivation increased
Apocrine Sweat Glands	Sweating increased	-
Eccrine Sweat Glands	Sweating increased	-
Lacrimal Glands	-	Lacrimation increased
Bladder Wall	Relaxed; sphincter closed	Wall contracted; sphincter relaxed
Pituitary gland	ACTH secreted	-
Adrenal medulla	Norepinephrine and epinephrine secreted	-
Adrenal cortex	Cortisol secreted	-

Table 2.1: Characteristics of the autonomic nervous system (Brunton et al., 2005)

capable of producing some of the same reactions, such as increased heart rate. However, dopamine is better known for its role when processing rewards. It appears that dopamine neurons encode the prediction error of rewarding outcomes. If concentrations of dopamine increase following an unexpected success, then the physiological response should follow.

There are several hypotheses regarding which components of the nervous system contribute to emotions and emotional decision-making. Recall Damasio's hypothesis which postulates that the orbitofrontal, amygdala, and peripheral nervous system—structures that mediate emotion—mediate decision making. Damasio's colleagues, (Bechara et al., 1995), postulate that the hippocampus and amygdala are critical to decision making.

Other hypotheses have implicated the *limbic system*, a term that refers to structures in the brain involved with emotion and memory. The limbic system is poorly defined and, consequently, the term is used inconsistently in the literature; (LeDoux, 1996) even goes so far as to suggest that the concept of the limbic system is not even valid. That said, the traditional limbic system includes the amygdala (or reptilian brain), hypothalamus, mammillary body (used to form memory), and the orbitofrontal cortex. Since decision-making and memory are processed by overlapping neurological structures, there may be similarities between the two.

The problem of isolating the components of the nervous system that govern decision-making is difficult because the nervous system responds to so many inputs. (It is this many-to-one mapping that makes emotion recognition difficult as well since various emotions—fear, anger, and sadness—may all produce identical emotional responses.) Consider how the death of a spouse or winning the lottery the day before could have a profound impact on hormone levels. This study did not account for any such inputs.

Given this background material, the next chapter describes how to measure emotional responses. The most direct way to measure the status of the ANS would be to measure adrenaline via blood or saliva in a chemical analyzer, but the analyzer is not a real-time solution. Instead, physiological signals, which I describe next, can be measured in real-time. Moreover, the instruments are non-invasive and minimally distractive. Thus, I am able to establish real-time associations between the ANS and both environmental and decision-making variables.

---

won a share of the 2000 Nobel Prize for showing that dopamine is not just a precursor of noradrenaline and adrenaline, but a neurotransmitter as well.

Chapter 3

Transcribing the Emotional Response

> "If an individual is placed in circuit with a delicate galvanometer and made to laugh, to feel sad, or is suddenly surprised, there will be movements in the instrument indicating the passage of small electric currents. Such interesting scientific facts as these, and many others to be mentioned later, make it clearly evident that emotions are something more than mere states of mind." –Eastman (1914)

Documenting a relationship between emotional responses and the decision-making process requires measuring the emotional response. The first record of electronically measuring the emotional response is from the 1880s. William James and Carl Lange independently hypothesized that the physiological response *causes* the emotion. "Our natural way of thinking about ... emotions is that the mental perception of some facts excites the mental affection called the emotion, and that this latter state of mind gives rise to the bodily expression. My theory, on the contrary, is that the bodily changes follow directly the perception of the exciting fact, and that our feeling of the same changes as they occur *is* the emotion." (James, 1992) This theory is known as the James-Lange theory. In contrast, the Cannon-Bard theory, named after Walter Cannon and Philip Bard, turns the James-Lange theory on its head. Cannon postulated that the physiological response *follows* the emotion. In neurobiological terms, the thalamus signals to the amygdala (emotional response) and to the hypothalamus (physiological response). In order for the emotion to precede the physiological response, either the signal's path must be sequential or the amygdala must react

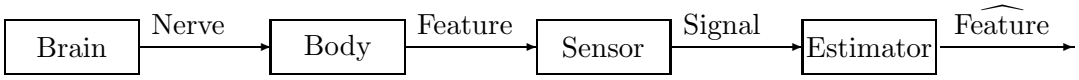

Figure 3-1: Structure of estimation and detection problem

faster than the hypothalamus. A third theory, proposed by Schacter and Singer, postulates that emotion is the cognitive interpretation of a physiological response. A fourth theory is Zajonc's theory, which is a variation on Schacter's theory. Zajonc argues that in most circumstances the emotional response occurs too rapidly to be caused by cognition, but that those processes run in parallel.

Although they dispute causation, the common thread woven among these theories is that the emotional and physiological response are correlated.

The problem of determining what takes place in the brain looks like a general detection and estimation problem. In this particular problem, the underlying signal is the emotional response generated in the brain, played out in the body, and measured by sensors. Thus, the goal is to estimate features that act as proxy for emotion (see Figure 3-1).

The estimated features come from an estimator. A signal, or time series, feeds the estimator. The six physiological signals employed in the study are skin conductance/galvanic skin response (SCR/GSR), blood volume pulse (BVP), electrocardiogram (ECG), electroencephalogram (EEG), electromyogram (EMG), and temperature (Temp).

A sensor is a specific kind of transducer that converts a physical property to an electric signal. More generally, the word *sensor* refers not only the device's capability to transduce but also to amplify, record, and quantify. The choice of sensors was driven by the goal of this research, which was to measure emotional response amidst real-time decision making. Consequently, the design of the experiment and its goal determined the kinds of sensors employed. Desirable attributes of a sensor are: (1) minimally intrusive, or non-invasive (which are less accurate than invasive sensors); (2) real-time, or on-line, as opposed to delayed, or off-line; and (3) most likely to minimize as opposed to maximize the energy extracted from the system. Most sensors are continuous and analog, but since the advent of the computer, more and more sensors sample and digitize, thus discretizing in both time and magnitude. (Note the semantics: I use the word *magnitude* to describe an absolute value and the word *amplitude* to describe the relative value between two points, i.e., the trough to peak height of a waveform.) Common physiological sensors measure weight, distance or

BVP	Blood Volume Pulse
ECG	Electrocardiogram
EEG	Electroencephalogram
EMG	Electromyogram
HR	Heart Rate
HRV	Heart Rate Variability
RMS	Root Mean Square
Std	Standard Deviation
SCR	Skin Conductance Response
Temp	Temperature

Table 3.1: List of abbreviations

length, pressure, voltage, volume, and their accompanying rates, such as volume/minute, distance/second.

The SCR, ECG, EEG, and EMG record electrical potentials across cellular membranes. The cycle of depolarization and repolarization is called an *action potential*.[1] Sensors that measure electrical potential have at least two leads. The BVP and temperature are non-electrical signals. The BVP sensor also uses two leads—a phototransmitter and a photodetector—in one self-contained unit. The temperature sensor requires a single lead. Table 3.2 lists several characteristics of the physiological signals.

The following sections describe six physiological signals employed in the study: While an entire book could be, and has been, devoted to each physiological signal, I will briefly summarize each physiological signal and highlight why it is pertinent to this study. I will describe the physiology behind each signal, how the signal is recorded and measured, the signal's characteristics, and why each signal is a proxy for affect.

[1]Although the signal generated within a neuron is electric, the communication between neurons takes a chemical form. The signal, called an action potential, is generated within the body of a neuron when it becomes stimulated. The electrical impulse travels along an axon until it reaches the terminal, the point of contact with a dendrite, which is a pathway leading to another neuron. At the point of contact there is a gap called a *synapse*. The arrival of the action potential (electrical impulse) releases a small quality of chemical substances (neurotransmitters), which travel across the synapse and attach themselves to the receptors, highly specialized molecules on the other side of the gap. The neurotransmitters are then broken down in the synapse with the help of special enzymes (or reabsorbed in some cases) and the process continues. The first models of actions potentials were published by Alan L. Hodgkin and Andrew F. Huxley in 1952, for which they and Sir John Carew Eccles received the 1963 Nobel Prize. Their work had built on the work of Sir Charles Scott Sherrington and Edgar Douglas Adrian "for work on the function of neurons, including the fact that stronger stimuli result in a higher frequency of nerve impulses" for which they were awarded the Nobel Prize in 1932.

Physiological Signal	*Method*	*Signal Characteristics*	*Sampling Frequency*	*Range*
BVP	non-invasive	cyclic (75bpm)	32 Hz	0–100 %
ECG	non-invasive	cyclic (75bpm)	256 Hz	0–50 μV
EEG	non-invasive	stochastic	256 Hz	10 μV
EMG	non-invasive	stochastic spikes	32 Hz	0–200 μV
SCR	non-invasive	baseline + phasic	32 Hz	0–10 μMhos
Temp	non-invasive	stable	32 Hz	75–100 °F

Table 3.2: Characteristics of physiological signals

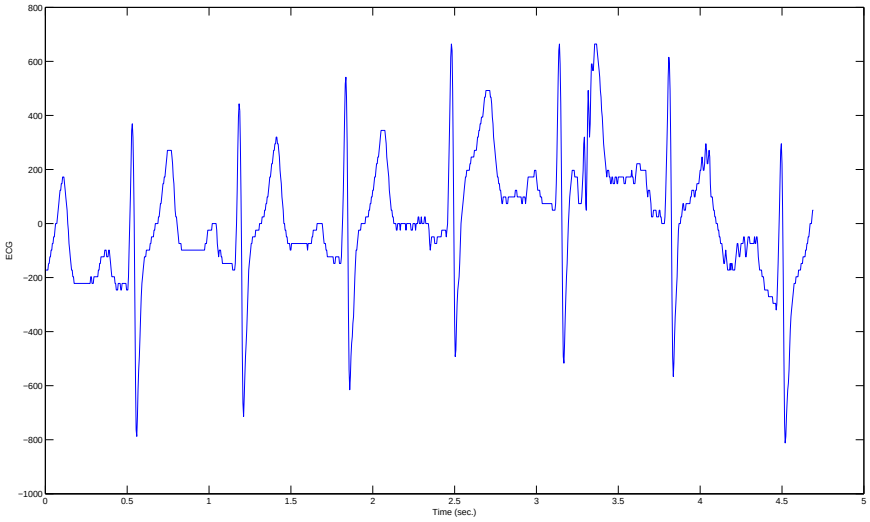

Figure 3-2: Electrocardiogram (ECG)

3.1 Electrocardiogram (ECG)

The electrocardiogram (ECG)[2] is a measurement of the heart's electrical activity. The ECG represents the electrical depolarization and repolarization of the heart. The anatomical explanation of the cardiac cycle is that the heart is composed of four chambers—two atria and two ventricles. The two atria contract first, followed by the two ventricles contracting. The contraction period is called *systole* and the relaxation period is called *diastole*. The cycle then repeats itself. See Figure 3-2 for a sample from one of the individuals.

To record an ECG, the skin is cleansed with alcohol and then smothered in gel to improve conductivity. The electrocardiograph uses three leads, or electrodes. The negative

[2]Willem Einthoven, a Dutch physician, invented the *electrokardiogramm* (EKG) in 1903 for which he received the Nobel Prize.

electrode is placed to the right of the sternum near the base of the clavicle. The positive electrode is placed on one of the ribs below the left armpit. The ground node is placed on one of the ribs below the right armpit.

The relationship between the heart and emotional response may be confounded by exercise or motion, artifacts (missed, spurious, and ectopic beats), respiration (inhalation quickens heart rate and exhalation slows heart rate), EMG noise, power line interference (60 Hz), electrode noise, etc. Attempts were made to mitigate as many of these confounding factors as reasonable. For instance, all the sensors, including the ECG sensor, have a built-in notch filter at 60 Hz. Natural environmental constraints restricted the individuals to sedentary positions, which eliminated several confounding sources, such as exercise.

3.1.1 Signal Characteristics

The ECG is cyclic. See Figure 3-2. The average periodicity of the cardiac cycle is 0.8 sec (1.25 Hz or 75 beats per minute). The ECG is characterized by three waves, each identified by a letter, and three intervals, which are identified by pairs of letters. The P-wave (90 msec.) represents the depolarization of the atria. The QRS-complex (80 msec.) represents the depolarization of the ventricles. The atria repolarize at the same time that the ventricles depolarize. The atrial repolarization is, however, obscured by the much larger QRS-complex. The third wave, the T-wave, represents the repolarization of the ventricles. The P-R interval, the Q-T interval, and the S-T interval are the most interesting intervals within of the cardiac cycle. The R-R interval is usually inverted to determine the instantaneous heart rate.

The QRS complex is approximately ten times larger in amplitude than the P-wave. It is for this reason that the R-R interval is used to calculate the heart rate.

3.1.2 Neural Control

The relationship between heart and emotion is long-established.[3] It is common knowledge that the heart rate increases during excitement and decreases during boredom. The heart's activity reflects many underlying psychological and physiological states.

[3]In 1628 William Harvey wrote, "For every affection of the mind that is attended with either pain or pleasure, hope or fear, is the cause of an agitation whose influence extends to the heart." While the quote is the earliest reference I found, it would be presumptuous to imply that Mr. Harvey was the first person to recognize the relationship between the brain and the heart.

Heart activity is affected by both the sympathetic and parasympathetic nervous systems, though not symmetrically. Heart rate variability (HRV), or the change in heart rate through time, is caused by the push and pull of the sympathetic and parasympathetic nervous system. As seen in Table 2.1, the sympathetic nervous system causes the heart rate to increase. In contrast, the parasympathetic nervous system causes the heart rate to decrease. Under normal conditions, an individual experiences periodic variation in HRV, a phenomenon known as respiratory sinus arrhythmia (RSA). During extreme (emotional) stress, the tug-of-war between the sympathetic and parasympathetic nervous systems, or *sympathovagal balance*, produces irregularly spaced, skipped, or added heartbeats. (See (Clifford et al., 2006) for a detailed discussion of the morphological changes that occur during HRV.)

HRV is not entirely understood. It appears to indicate both dynamic and cumulative load, where load can be physical or mental exertion. As a *dynamic*, or short-term, marker of load, HRV appears to be sensitive and responsive to acute stress. Under laboratory conditions, mental load—including making complex decisions, and public speech tasks—have been shown to lower HRV. As a *cumulative*, or long-term, marker of load, HRV has been shown to decline with the aging process. Although the resting heart rate does not change significantly with advancing age, there is a decline in HRV, which has been attributed to a decrease in efferent vagal tone and reduced beta-adrenergic responsiveness. In contrast, regular physical activity has been shown to raise HRV, presumably by increasing vagal tone.

Several studies have suggested a link between negative emotions (such as anxiety and hostility) and reduced HRV. (Kawachi et al., 1995) reported a cross-sectional association between anxiety and reduced HRV (as assessed by two time-based features) in 581 men. (Offerhaus, 1980) observed lower HRV in individuals who were "highly anxious" according to the Minnesota Multiphasic Personality Inventory. (Yeragani et al., 1990, 1993) have reported a reduction in HRV (using both time-based and frequency-based features) among panic disorder patients.

3.1.3 Features

The features identified below are all time-based features. Frequency-based features that are calculated over short time intervals are not accurate. Since the goal is to identify features that are useful for measuring emotional response, which occurs within short time

intervals, on the order of 10 sec, frequency-based features would have been inaccurate. I did not employ non-linear or chaotic analysis, as they usually require long-term monitoring of signals.

The following lists the four features of the ECG signal:

Heart Rate is the frequency of contraction of the cardiac muscle and is measured as the inverse of the heart cycle duration, most often measured by the ECG's R-R interval. Two types of mechanisms affect heart rate: intrinsic and extrinsic. Intrinsic mechanisms include stretching the sinoatrial node and warming the heart (the warmer the heart, the slower it wants to pump). Extrinsic mechanisms include emotions. Heart rate and heart rate acceleration have been used as proxy for physical activity and emotions, such as fear and anger (Levenson, 1992).

I used an SQRS-based algorithm to calculate the mean heart rate over different intervals (Pino et al., 2005; Goldberger et al., 2000).

Heart Rate Variability (HRV) is the standard deviation of the duration of the inter-beat intervals. See Figure 3-3 for a tachogram, which shows HRV in one of the traders.

There are several ways to calculate HRV. At least 26 different metrics have been used in the literature (Electrophysiology, 1996). Examples include: the standard deviation of the inter-beat intervals during a short-term window; the range, or difference between the shortest R-R interval and the longest (called the MAX-MIN, or peak-valley quantification of HRV); the standard deviations of the normal mean R-R interval obtained from successive 5-minute periods over 24-hour Holter recordings (called the SDANN index); the number of instances per hour in which two consecutive R-R intervals differ by more than 50 msec over 24-hours (called the pNN50 index); the root-mean square of the difference of successive R-R intervals (the rMSSD index); and the base of the triangular area under the main peak of the R-R interval frequency distribution diagram obtained from 24-hour recording.

Preliminary analysis included the range of the inter-beat intervals. However, the final analysis did not include this feature since it did not provide additional information that the standard deviation did not already provide.

Frequency-based statistics, such as power in certain bands, may also be used to estimate HRV. The power spectral density of an R-R tachogram[4] may show two dominant

[4]The signal is now unevenly sampled.

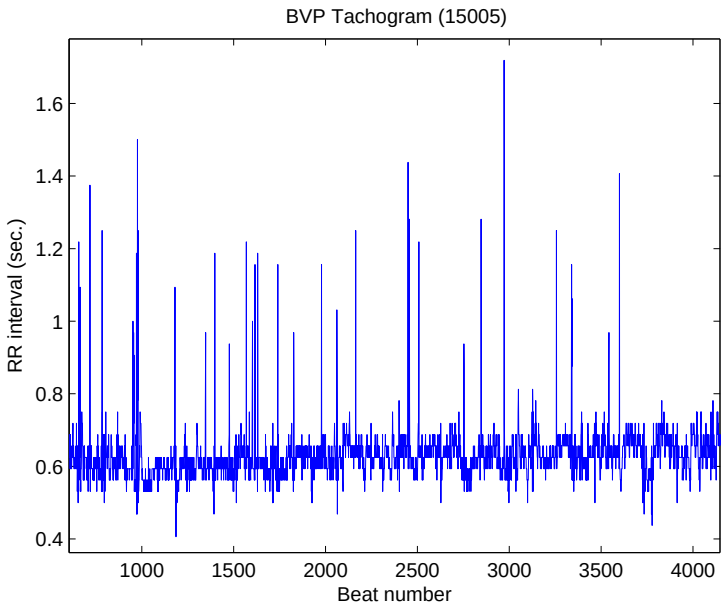

Figure 3-3: Tachogram

peaks: one in the low frequency region (0.015–0.15 Hz), which represents the sympathetic nervous system, and one in the high frequency region (0.15–0.40 Hz), which represents the parasympathetic nervous system. An increase in sympathetic response or a decrease in parasympathetic response will increase HRV in the low frequency region, decrease HRV in the high frequency region. Thus, one possible HRV statistic is the low/high frequency-ratio. The European and North American Task Force on standards in HRV suggests that the shortest interval over which HRV should be calculated is five minutes.

Heart Rate Acceleration (RMS) is the rate of shortening the inter-beat intervals. It is a measure of intensity, attention, and cognitive processing. Researchers have studied heart rate deceleration primarily in the performance of athletes, such as archers, golfers, rifle shooters, and tennis players (Jennings and Wood, 1977).

Heart Rate Acceleration I is computed as the root-mean-square (RMS) of the inter-beat intervals.

Heart Rate Acceleration (Std) is the standard deviation of the difference in inter-beat intervals.

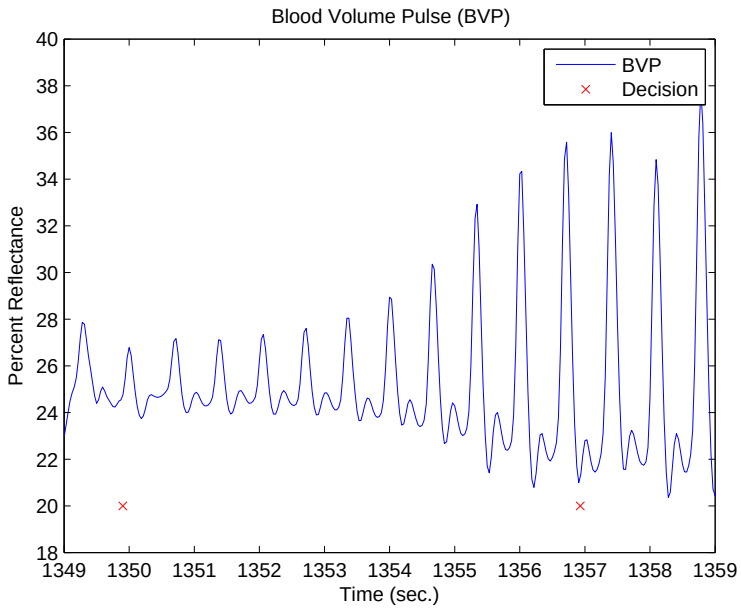

Figure 3-4: Blood Volume Pulse (BVP)

3.2 Blood Volume Pulse (BVP)

The blood volume pulse (BVP), or plethysmogram, is a measurement of, not surprisingly, blood volume. Blood volume is a function of the vessel's diameter and blood pressure. The vessel diameter is controlled by vasomotor activity. The blood pressure is controlled by the contraction and relaxation of the heart. When the ventricles contract, blood pressure rises. When the ventricles relax, blood pressure drops.

The changes in volume are measured by a plethysmograph[5]. A photoplethysmograph measures changes in volume with light. This device emits infra-red light and measures the amount of reflected light with a photosensor. As the heart forces blood through the vessels, the vessels engorge, which changes the amount of light reflected. The other options of measuring changes in blood volume are hydraulic and electrical sensors.

To record the BVP, the photoplethysmograph is attached directly to the tip of the thumb. Because the sensor is a single self-contained unit, attaches to a finger, and does not use gel, the BVP is much easier to use than an ECG though it is more susceptible to noise.

Several factors can affect the accuracy of the BVP: fluorescent light, posture, and motion artifacts. For example, fluorescent light (60 Hz) can affect the BVP if the photosensor is

[5]The word *plethysmos* is of Greek orgin and means "an enlargement."

not lightproof. Posture changes, such as lowering the finger, can decrease the amplitude, an artifact that is difficult to discriminate from signal. While these factors affect the accuracy of the BVP, the factors may actually provide insight into behavior. For instance, if a trader lowers his finger after every big trade, the true value of the BVP may be unknown, but the BVP recording still carries information.

Other factors can lower the signal-to-noise ratio, where the signal represents the neural control of the BVP and the noise is everything else. For instance, hot, ambient temperatures, which have nothing to do with processing risk, can increase blood flow in the skin.

3.2.1 Signal Characteristics

The plethysmogram is cyclic. See Figure 3-4. Its periodicity is virtually identical to that of the ECG (see Figure 3-2). The rising waveform depicts the systolic inflow, and the apex represents the peak volume systole. The falling waveform depicts the diastolic outflow, and the trough represents diastole. At the trough, inflow and outflow are equal. The amplitude measured from trough to peak is called the pulse volume. The cycle then repeats itself.

Some waveforms include a notch on the falling portion curve. This notch is called the dicrotic notch, which marks the end point of the volume systole. It is produced by the closure of the aortic valve.

Unlike the other physiological signals, the BVP does not have an absolute scale for comparison. Thus, the magnitude of the BVP from one individual should not be compared to the magnitude from another individual.

3.2.2 Neural Control

The plethysmographic response is a product of the sympathetic nervous system; no parasympathetic fibers have been found. The sympathetic nervous system innervates the blood vessels in the palms of the hands and soles of the feet to constrict. In contrast, the sympathetic nervous system innervates the blood vessels in the other parts of the body to dilate. For instance, blushing is the dilation of blood vessels in the face.

In comparison to the changes in the SCR and the heart rate that accompany arousal, the plethysmographic response persists much longer. Also, the plethysmogram tends to be more sensitive to nervous system innervations than the SCR.

3.2.3 Features

The following lists the six features of the BVP signal:

Heart rate has already been described. See Section 3.1 for details.

Heart Rate Variability (Std) is the standard deviation of the duration of the inter-beat intervals. It is the same as its ECG counterpart, except that it is calculated from the BVP.

Heart Rate Variability (Range) is the range in duration of the inter-beat intervals.

Heart Rate Acceleration (RMS) is the same as its ECG counterpart, except that it is calculated from the BVP. It was necessary to duplicate features in the BVP because of the missing ECG signal in one of the individuals (15008).

Heart Rate Acceleration (Std) is the same as its ECG counterpart, except that it is calculated from the BVP.

BVP Ratio is the ratio of the average local BVP amplitude to the average of a larger window. Vasoconstriction measures the narrowing of the lumen of blood vessels in the skin of the digits; vasodilation measures the widening of the vessels. The muscles and nerves that control the size of the blood vessels are called the vasomotor response. The vasomotor response includes sympathetic vasoconstrictors and parasympathetic vasodilators. As explained in section 2.6, adrenaline causes vasoconstriction, though adrenaline is just one of many vasoconstrictors. Other (confounding) vasoconstrictors include cold temperatures, caffeine, antihistamines, decongestants, and norepinephrine. In addition, physiological research has shown that vasoconstriction may follow a single deep breath.

Vasoconstriction and vasodilation are measured by the amplitude (trough-to-peak) of the BVP. Vasoconstriction produces smaller amplitudes, and vasodilation produces larger amplitudes. To normalize the feature, it is necessary to divide the average BVP amplitude during an (event) interval by the average BVP amplitude during the entire interval.

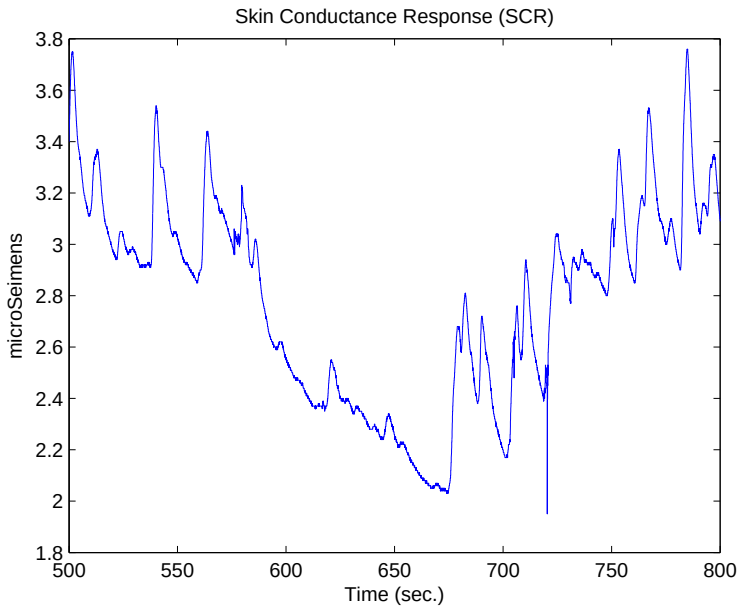

Figure 3-5: Skin Conductance Response (SCR)

3.3 Skin Conductance Response (SCR)

The skin conductance response (SCR)[6] is a measurement of the electrical conductivity on the skin.

The physiological origin of the SCR is unclear. Initially, researchers believed the SCR was caused by the vascular system, or changes in blood flow. While there might be a vascular component to the SCR, the increase in conductance is currently thought to be due to an increase in the permeability of the cutaneous membrane as a result of innervations by the sympathetic nervous system. One hypothesis is that the increase in permeability is caused by sweat from the eccrine sweat glands. Eccrine sweat glands, which are distributed over the entire body surface are more abundant on the palms of the hands, soles of the feet, and on the forehead[7]. Myoepithelial cells, which contain smooth-visceral-muscle-like organs, contract to squeeze the sweaty fluid through thin ducts in the skin. Myoepithelial "muscles" are innervated by the sympathetic nervous system; the muscle-like organs also

[6]Fere, a French neurologist (1888), and Tarchanoff, a Russian physiologist (1890), wrote the initial papers on the subject of the psycho-galvanometer.

[7]The other type of sweat glands that appear in the body are apocrine sweat glands. These glands are mainly present in the armpits and are responsible for the odor associated with sweat. Stress stimulates the apocrine gland to squeeze the sweat out of the gland. It is not a pleasant thought, as (Scripture, 1908) explained, "some people have objected to the sweat glands being dignified into organs of the emotions. But dignity does not exist for science."

contract in response to adrenaline. However, this sweating response is about one second slower than the SCR. An alternative hypothesis is that the increase is caused by changes in electrical potential across neurons connected to the sensorimotor strip of the cortex. The cortex also mediates capillaries, which can affect conductance. The consensus is that the SCR is associated with activation of (not necessary moisture from) the sweat glands by the postganglionic sympathetic fibers but that the perspiration does not produce the characteristic increase in skin conductance by acting as an electrolytic conductor.

To record the SCR, two electrodes are applied to the palms of the hands. K-Y Jelly is used as a contact between the electrode and the skin because of its low-conductivity. Because the hands have a particularly large number of eccrine sweat glands as well as nerve endings from the sensorimotor strip of the cortex, the SCR is most often measured across the palms. The SCR is measured by the voltage drop between the two electrodes. There are two ways to measure the SCR. If the electrodes actively apply a current, the measurement is called *exosomatic*. Otherwise, the electrodes are passive and do not apply a current, which is called *endosomatic*.

Several factors can affect the accuracy of the SCR. As usual, one primary suspect is electrode noise. Temperature is another factor that can change skin conductance as conductance decreases by about 3% for a drop in temperature of 1 °C.

3.3.1 Signal Characteristics

The SCR is a signal with two components: a tonic baseline level and a short-term phasic response which is superimposed on the baseline level. See Figure 3-5. Depending on the intensity of the arousal, the response appears 1 to 4 seconds after stimulation. The response rises to a maximum after 2 to 10 seconds before subsiding at a slower rate. Technically, the skin conductance response refers to the phasic component. This phasic response has many names including the galvanic skin response (GSR), galvanic skin reflex (GSR), psychogalvanic reflex (PGR), skin potential response (SPR), skin resistance response (SRR), Tarchanoff reflex, electrodermal response (EDR), or electrodermal activity (EDA). The baseline and phasic components together may be called an electrodermogram (EDG), which would be a term more consistent with the other physiological signal terms used in this paper, but this term is rarely used in the literature. SCRs that occur as the product of stimulation are called *specific*, whereas SCRs that occur without stimulation are called *nonspecific*.

One of the challenges of using the SCR can be determining whether the signal indicates the presence of a response or not. Rather than manually identify responses, I use a modified slope-detection algorithm to automate the identification process. It can be especially difficult if responses occur during responses. If a second response occurs during recovery of the first response, the responses are called type 2. If a second response occurs between onset and peak of the first response, the responses are called type 3. If no second response occurs, the response is type 1. There are three different methods to measure the additional response.

3.3.2 Neural Control

The SCR is mediated by the sympathetic nervous system often as a result of stimulation, such as a pinprick, threat, images, mental effort, emotions, arousal, or attention. It appears that stimuli which are more salient to the individual are more likely to generate a response. Attention-grabbing stimuli and demanding tasks evoke increased SCR responses. The response appears as an increase in electrical conductance (a decrease in resistance) of the skin. The earliest study relating the SCR to an emotional response was performed by Carl Gustav Jung, a Swiss psychologist. Dr. Jung's 1906 experiment consisted of reading words to subjects who were wired to a galvanometer. If a word was emotionally charged, there was a change in resistance. However, without amplification the galvanometer was difficult to use. Thus it was not used much until the development of valve amplifiers in the 1930s.

The SCR is one of the most sensitive non-invasive indicators of autonomic nervous system activity. The sympathetic nervous system innervates the eccrine sweat (sudomotor) glands; the parasympathetic nervous system does not affect the SCR. Skin conductivity varies linearly with intensity, novelty, and significance (Lang, 1995). Stress may cause a rise in conductivity. However, not all forms of stress cause a rise in conductivity, a contradiction called by (Apter, 2006) the *paradoxical arousal*. The SCR is essentially involuntary, although people can learn to control it somewhat via biofeedback, where biofeedback is the technique of becoming self-aware of involuntarily controlled bodily functions in order to self-regulate or manipulate them. As a sensitive detector of emotion, the SCR has served as one of the components in the polygraph[8], in addition to blood pressure, heart rate, and respiration.

[8]There is a rich and long history of SCR and the lie detector. Inspired by Dr. Jung, Volney Mathison was a pioneer in the lie detector field and invented the modern device used to measure SCR today. Another pioneer was William Marsten, the Harvard psychologist who many consider to be the father of the modern

(Critchley, 2002) reviews the brain mechanisms involved in generating the SCR.

3.3.3 Features

The following lists the five features of the SCR signal:

SCR Count is the number of SCR responses occurring within an (event) interval. I used a slope-detector algorithm to calculate the SCR response.

SCR Amplitude is the amplitude (trough-to-peak) of the SCR.

SCR Duration or, to be more specific, the rise time is the average time interval between onset of response and peak of response within an (event) interval. Because the full recovery time of the SCR can be long, overlapping SCRs may occur during the decay, which complicates the estimation of the entire SCR duration. Instead, the rise time is a better feature than the duration. (In addition to SCR Mean Duration, the preliminary analysis included two other SCR features: Min and Max duration. The min and max durations are the minimum and maximum time intervals between onset and peak of the response within an (event) interval. However, the final analysis did not include these features since they did not provide any unique insights.)

SCR Area is a function of the SCR amplitude and SCR duration. The SCR area is the triangular area from onset to peak, or, mathematically, $\frac{1}{2}$*SCR Amplitude*SCR Duration.

SCR Δ or first-order difference is the change in magnitude of the SCR.

3.4 Electromyogram (EMG)

The electromyogram (EMG)[9] is a measurement of muscle activity. The EMG is the one physiological signal that measures the activity of the somatic (or skeletal) nervous system. The EMG is the spatially weighted sum of electrical activity of all the muscles within range

polygraph. It is rather comical that Dr. Marsten created the popular character Wonder Woman. Wonder Woman's signature weapon was the Lasso of Truth. This magic rope was a rudimentary version of the lie detector as it could make all who were lassoed tell the truth.

[9]Hans Piper invented the EMG in 1907.

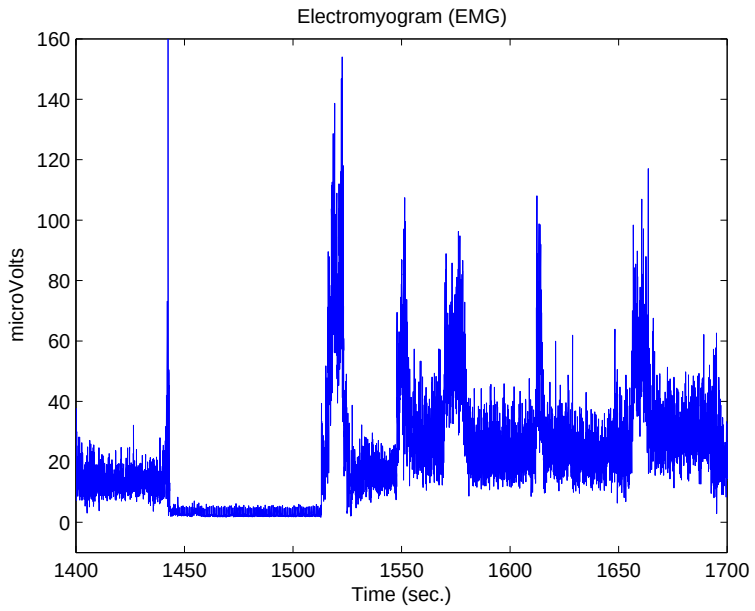

Figure 3-6: Electromyogram (EMG)

of the sensor. The EMG may be measured subcutaneously or on the surface of the skin. I employed the surface method.

Muscle is controlled by a motor unit. When resting, normal muscle is electrically silent, but generates electrical current when active, such as during contraction or stimulation. Stronger contractions generate stronger action potentials. A fraction of the action potential traverses through the body to the surface of the skin.

To record the EMG, the skin is cleansed from dirt and oil. A triode electrode is attached to the skin over the muscle of interest. Gel is used to improve conductivity. The sensor is secured with adhesive tape. The two muscles measured were the trapezius (back) and the flexor digitorum superficialis (forearm). The trapezius contracts when individuals tense. The flexor digitorum detects finger and arm movements. The signal was sampled at 32 Hz.

The EMG is the noisiest of the signals under study in this paper. In addition to the usual culprits of power line interference (60 Hz) and electrode noise, the orientation of the muscles relative to the electrodes can alter the measurements. As explained above, the EMG measures the activity of several muscles, which may include more than just the muscles of interest. Technically, the EMG does not measure motion, though motion may be the result of muscle contraction. Moreover, motion can generate noise in the signal.

3.4.1 Signal Characteristics

The amplitude of the EMG indicates the force of contraction (larger muscles can produce larger contractions) and the number of motor neurons. The amplitude may be diminished if the muscles are deep in the body and the signal has to traverse other tissue to reach the body surface. The amplitude ranges from 0 to 1000 μV. See Figure 3-6.

The EMG signal experiences bursts during muscle activity. The instantaneous value of the EMG contains no information and is never used. The signal has interesting frequency content from 0 to 500 Hz.

3.4.2 Neural Control

There are two types of muscles: skeletal and smooth. Skeletal muscles are controlled by conscious (voluntary) cognitive processes. Smooth muscles are controlled by unconscious (involuntary) cognitive processes. In both skeletal and smooth muscles, the controlling process sends a binary signal: contract or no-contract. If a muscle contracts, an opposing muscle, called the antagonist, must contract to restore the body to its original position. Because muscles are pitted against one another, the surface EMG is often unable to differentiate the electrical activities of the agonist from the antagonist. When the two opposing muscle groups push and pull with equal force, there is no movement, but the tension is still measurable. Since I am not aiming to measure the agonist or the antagonist muscle, but simply to measure whether any muscle is being contracted, a surface EMG meets the criteria.

3.4.3 Features

The following lists the two features of the EMG signal:

Amplitude is the amplitude (trough-to-peak) of the EMG. (Sykes and Brown, 2003) showed that individuals in a state of arousal depress gamepad buttons with more pressure. This result demonstrates the physiological manifestations of emotional energy. The amplitude of the EMG is a measure of muscle tension and relaxation—the greater the amplitude, the greater the tension.

Preliminary analysis included the EMG energy, $\sum_i (x[i])^2$. However, the final analysis did not include this feature since it did not provide any information not already provided

by the amplitude.

Count of zero-crossings is the number of directional changes in the EMG. This count is a measure of jitter. (Perhaps more common measures of jitter are frequency-based features, such as moments of the spectrum and average magnitude over a frequency range. But the short 10-sec. window precludes the use of frequency-based features.)

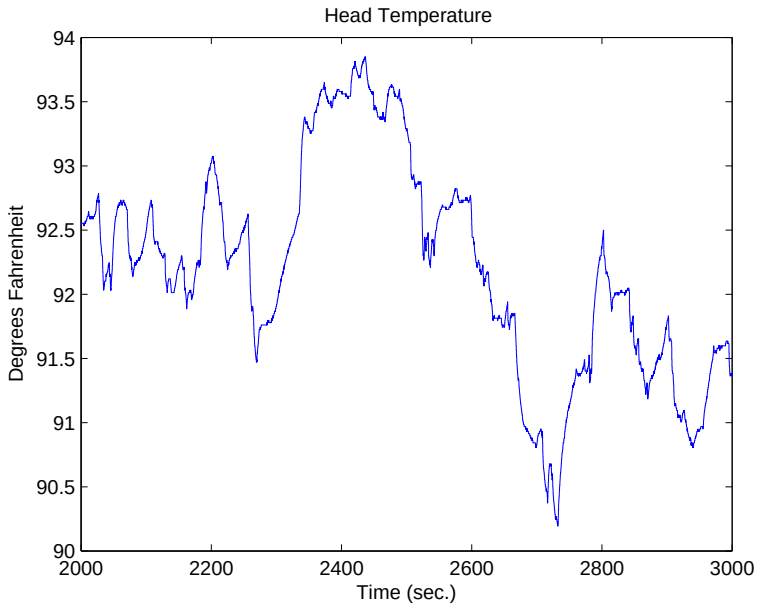

Figure 3-7: Finger Temperature (Temp)

3.5 Temperature (Temp)

Temperature is a measurement of hotness or coldness. According to Maxwell, temperature is the "thermal state considered with reference to communicate heat to other bodies." Heat may be, in the words of Maxwell, "communicated" to other bodies, such as a sensor, via conduction, convection, or radiation (conduction is the most common).

To record the temperature, a thermometer is attached directly to the skin. The thermometer is secured with adhesive tape. Two thermometers measured the temperature on a finger and the forehead. The finger thermometer was placed on the tip (distal phalanx) of one of the fingers, usually the index finger. The distal phalanx is more reactive than the other phalanges. The forehead thermometer was placed above the left eye, half way between the eyebrow and hairline, which is the location of the vein connected to the brain.

3.5.1 Signal Characteristics

Temperature hovers around a precise value. See Figure 3-7. Core body temperature is 98.6 °F (37 °C), although the extremities may be colder. The precise value can change slowly over time, although from day to day it is remarkably fixed.

3.5.2 Neural Control

Temperature in the skin varies due to changes in blood flow caused by vascular resistance or arterial blood pressure, both of which are regulated by the autonomic nervous system. Several studies have related the temperature of different parts of the body to cognitive and emotional stimuli (Shusterman and Barnea, 1995; Lombard, 1879). Forehead temperature, a measure of brain temperature, increases while experiencing negative emotions; cooling enhances positive affect, while warming depresses it (McIntosh et al., 1997). In contrast, finger temperature increases while experiencing positive emotions, and decreases with threatening and unpleasant tasks (Rimm-Kaufman and Kagan, 1996).

The primary noise factor that affects temperature recordings is the temperature of the ambient environment. Since the experiment took place inside a temperature-controlled building, all the individuals shared the same baseline environment.

3.5.3 Features

The following lists the two feature of the Temperature signal:

Number of Temperature jumps is the count of temperature jumps exceeding 0.1 °F.

Range is the difference between max and min values within an (event) interval.

3.6 Electroencephalogram (EEG)

The electroencephalogram (EEG)[10] is a measurement of the electrical activity of the brain. The nerve cells of the brain generate electrical impulses. The impulses are controlled by neural activity.

[10]Hans Berger invented the EEG in 1929.

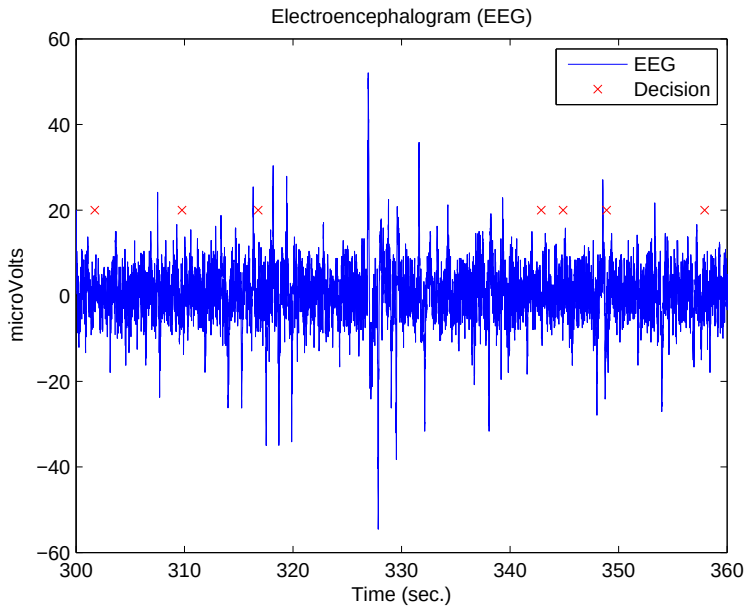

Figure 3-8: Electroencephalogram (EEG)

To record an EEG with an electroencephalograph[11], electrodes are placed in pairs on the scalp. Each pair of electrodes transmits a signal to one of several recording channels of the electroencephalograph. This signal consists of the difference in the voltage between the pair. The rhythmic fluctuation of this potential difference is shown as peaks and troughs on a line graph by the recording channel. The EEG of a normal adult in a fully conscious but relaxed state is made up of regularly recurring oscillating waves known as alpha waves. When a person is excited or startled, the alpha waves are replaced by low-voltage, rapid, irregular waves. During sleep, the brain waves become extremely slow. Such is also the case when a person is in a deep coma. Other abnormal conditions are associated with particular EEG patterns. Irregular slow waves known as delta waves, for example, arise from the vicinity of a localized area of brain damage.

3.6.1 Signal Characteristics

The EEG, the biosignal of science fiction, does not have much structure in the time domain. See Figure 3-8. Analysis of EEG takes place in the frequency domain. Several frequency bands are named. These bands are: Delta (1–4 Hz), Theta (4–8 Hz), Alpha (8–12 Hz), Beta (12–30 Hz), Gamma (30–70 Hz), High gamma (80–150 Hz).

[11] *encephalo* stems from the Greek word *en-kephale*, which means "in-head."

3.6.2 Neural Control

The EEG is a crude measure of neural activity in the brain. The initial research of the EEG showed that increased EEG activity is correlated with increased cortical activity, such as mathematical exercises. Researchers postulate that the gamma band is affiliated with perception and consciousness. (Mller et al., 2000) demonstrated that when subjects attended to a certain stimulus or perceived an object, spectral power in the gamma band was increased as compared to when the same stimulus was ignored or not perceived. Thus, the gamma band provides an indication of whether the subject "sees" something. In the specific case of specialists, it seems natural to postulate that gamma band activity might increase when a specialist sees something, such as a familiar pattern or situation. However, the effectiveness of the EEG as a research tool is limited because it records only a small sample of electrical activity from the brain. Many of the complex functions of the brain, such as those that underlie emotions and thought, cannot be measured by EEG patterns. That said, electroencephalography has proved useful as a diagnostic aid in a few select cases such as serious head injuries, brain tumors, cerebral infections, epilepsy, and various degenerative diseases of the nervous system.

3.6.3 Features

In this paper, I analyze the EEG separately from the other psychophysiological signals and focus exclusively on frequency-based features.

3.7 Other Physiological Signals

There are other physiological signals besides those detailed above. These include the electrooculogram, microphone, and electrogastrogram None of the following physiological signals were employed in this study. I have included them here for reference with the intent that future studies consider them.

3.7.1 Electrooculogram

An electrooculogram (EOG) is a measurement of eye movements. There are two primary eye movements related to the ANS: the blink rate and the widening of the eyes. The normal, resting blink rate of a human being is 20 closures per minute, with the average

blink lasting one quarter of a second. Significantly faster blink rates may reflect emotional stress. People blink faster when excited because eyelid movements reflect bodily arousal levels established by the brain stem's reticular activating system (Veltman and Gaillard, 1996). The widening of the eyes is another eye-based involuntary response, often performed in situations of intense emotion, such as anger, surprise, and fear.

3.7.2 Microphone

A microphone measures of sound. The specific sound of interest is speech (Borchert and Dusterhoft, 2005; Fernandez, 2004). The ANS can impact speech since the vocal tract is affected by respiration, muscles, salivation, heart rate, blood pressure, etc. (see Table 2.1). When stimulated by the sympathetic nervous system, speech will be faster, louder, more precisely enunciated with strong high frequency energy, higher-pitched, and wider in pitch range. In contrast, when the parasympathetic nervous system is more active, the resulting speech is typically slower, lower-pitched, more slurred, and with little high frequency energy (Johnstone et al., 2005). Thus, the speech features are rate, pitch, harmonics, and energy. A future study could include speech recordings, or, if that is not permitted, rough voice pitch recordings.

3.7.3 Electrogastrogram

An electrogastrogram (EGG) is the measurement of gastric myoelectrical activity from electrodes placed on the surface of the epigastrium. The ANS is partly responsible for moving contents through the digestive tract. The sympathetic nervous system reduces the movement, which explains, in part, why individuals under intense pressure may not be hungry until afterwards the episode has passed. A possible study could analyze food consumption volume and rate in conjunction with market and performance data. I would suspect that when traders lose significant amounts of capital, consumption is significantly less than when traders do not lose much capital.

Chapter 4

Experiment

This study was conducted on the Boston Stock Exchange. The stock exchange provides a liquid, open market for buying and selling the shares of publicly owned companies. The majority of the exchange's membership is composed of broker-dealer organizations which represent investors in the market place, buying and selling shares on behalf of their clients. The Boston Stock Exchange has approximately 200 members and trades about $800 million per day. In addition to the Boston Stock Exchange (BSE), the other U.S. Stock Exchanges that make up the National Market System are: New York (NYSE), American (AMEX), Chicago (CHX), National (NSX, formerly Cincinnati), Pacific (bought by NYSE in 2006), and Philadelphia (PHLX). By way of comparison, the dollar volume of NYSE, which is the largest exchange in the world, exceeds $10 trillion per day. Stock exchanges in the U.S. are regulated by the Securities and Exchange Commission (SEC).

The Boston Stock Exchange guarantees execution for small trades, where a small trade is defined to be less than 1,300 shares. These trades are executed automatically. This research does not analyze any of the automatic trades. Instead, when analyzing trading decisions, the focus is the manual trades, i.e. trades that require decisions from the trader. Occasionally, the specialist may intervene on a trade marked for automatic execution, in which case it is then treated as a manual trade.

4.1 Subjects

The sample of subjects consisted of eight members of the Boston Stock Exchange. These members, better known as *specialists*, buy and sell multiple stocks and carry responsibility

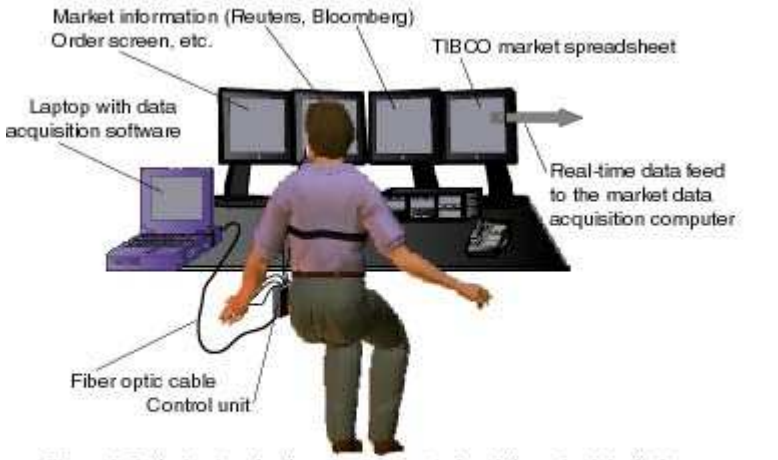

Figure 1. Typical set-up for the measurement of real-time physiological responses of financial traders during live trading sessions. Real-time market data used by the traders are recorded synchronously and subsequently analyzed together with the physiological response data.

Figure 4-1: Typical setup for trader

for maintaining a stable market in the basket of stocks they trade. Throughout this paper I use the labels *15001*, *15002*, ... *15008* to identify the specialists.

Each session started just before 9:30am Eastern Time, which is when the exchange opens for trading. The sessions lasted roughly 100 min. During the session, the specialists faced decisions on which stocks to monitor and which stocks to trade. Table 4.1 profiles the specialists. Figure 4-1 shows the setup of the experiment.

The specialists monitored anywhere from 14 to 48 securities with an average of about 20. The number of securities traded was half as many as the number monitored. The specialists who monitored larger numbers of equities did not spend an equal amount of time monitoring each equity, ranging from literally one second to almost one hour. The analysis below omits equities whose total monitoring time was less than 30 seconds and whose contribution to profit and loss was less than $5.

The computer terminals on the exchange allowed a single security to be monitored at a time. Thus, to monitor multiple securities a specialist would have to switch contexts, or manually time-slice. Figures 4-2 to 4-9 show the frequency with which the specialists changed context. 15003 stands out because he switched contexts almost twice as often as anyone else.

Figures 4-10 to 4-15 are graphical views of the same data from Table 4.1. The number

	Subjects							
	15001	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*
Gender	M	M	M	M	M	M	M	F
Start Time	9:27	9:24	9:31	9:27	9:19	9:20	9:24	9:07
Duration (min.)	104	108	103	106	111	111	107	105
# Equities Monitored	21	48	14	16	17	14	18	31
# Equities Traded	15	47	10	13	10	14	14	13
# Commands Issued	770	980	2570	1940	880	1400	1050	470
# Context Switches	280	80	1310	880	560	180	200	280
# Screen Updates	14000	14000	38000	27000	13000	21000	34000	14000
# (Manual) Buys	42	157	41	83	24	141	71	30
# (Manual) Sells	59	262	63	105	15	115	63	26
Profit & Loss ($)	-700	3,200	900	6,800	700	3,900	4,200	-700
Capital ($1,000s)								
Mean	-40	380	-10	520	4	-60	-40	-3
Max	90	1,140	110	1,040	25	230	320	4
Min	-170	-50	-80	-50	-63	-410	-900	-65
ROI (%)	-0.4	0.3	0.8	0.7	1.1	1.0	0.5	-1.1

Table 4.1: Summary profile of all specialists

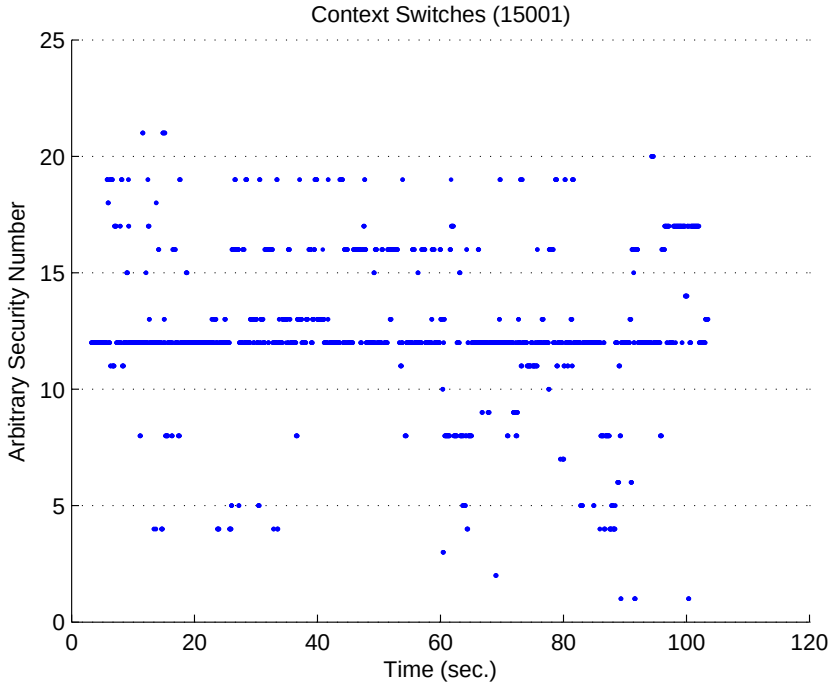

Figure 4-2: Switching contexts between different securities (15001)

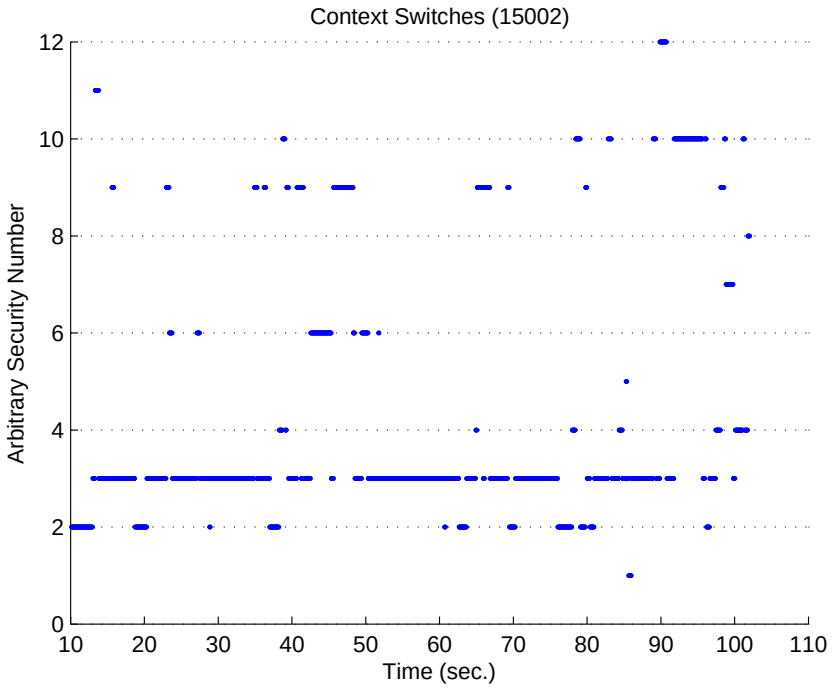

Figure 4-3: Switching contexts between different securities (15002)

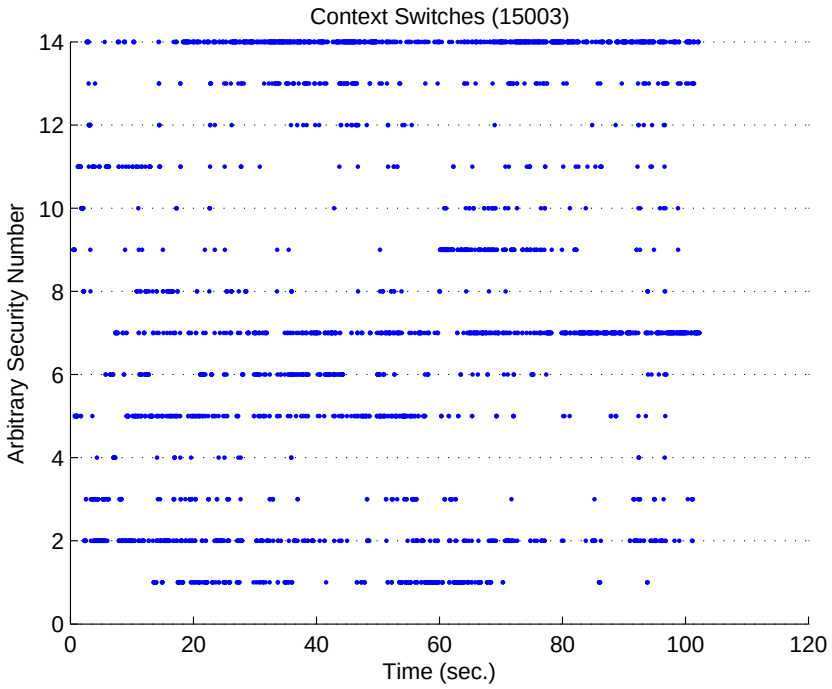

Figure 4-4: Switching contexts between different securities (15003)

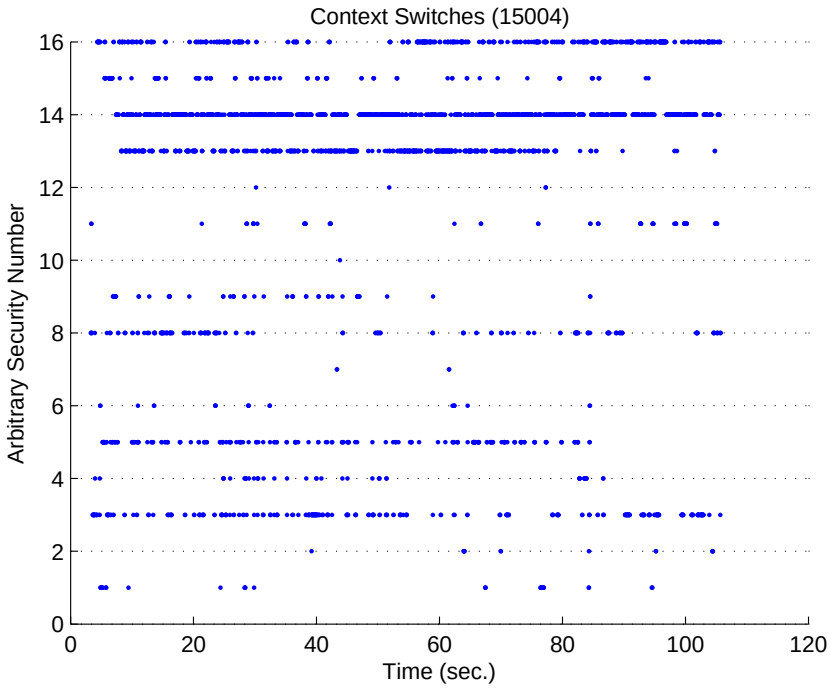

Figure 4-5: Switching contexts between different securities (15004)

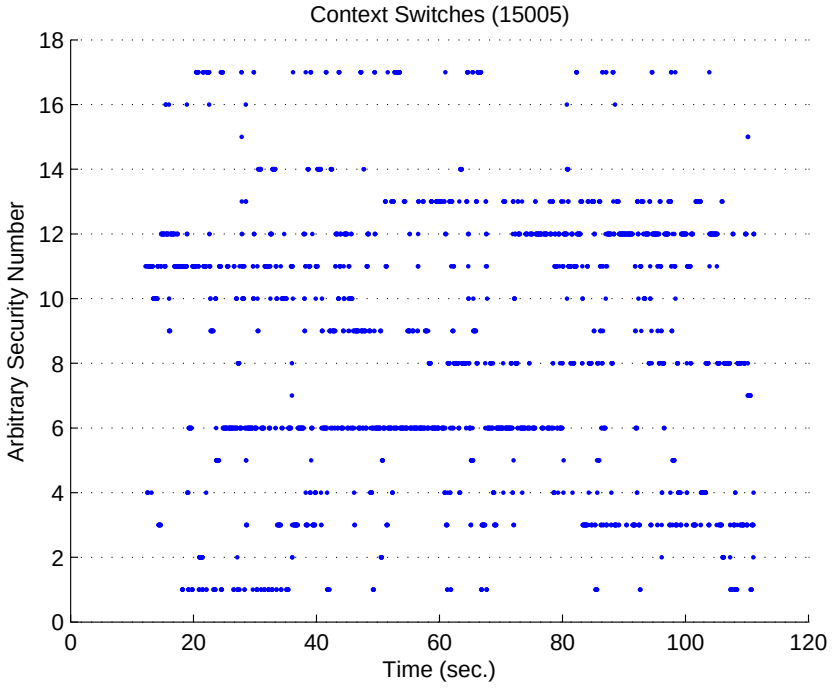

Figure 4-6: Switching contexts between different securities (15005)

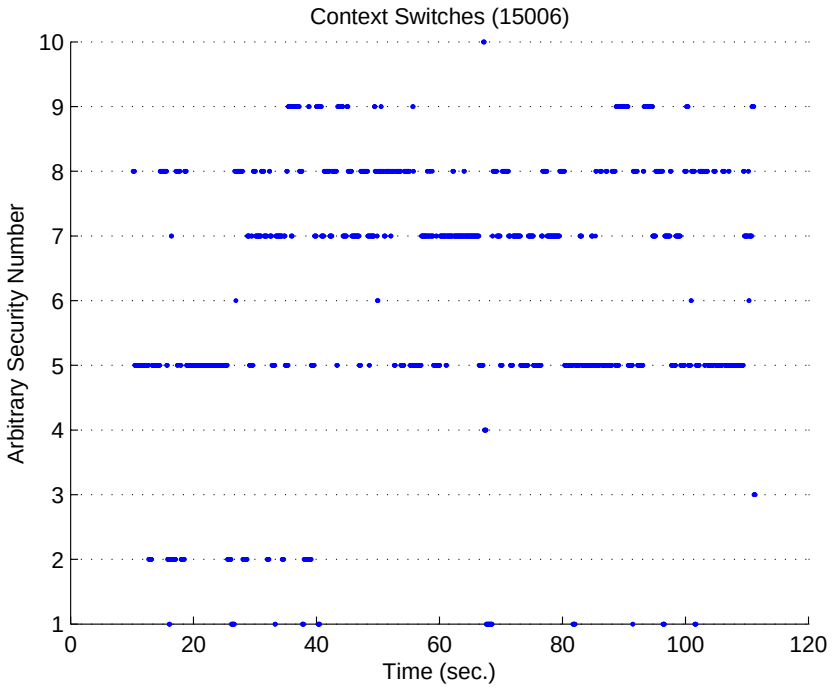

Figure 4-7: Switching contexts between different securities (15006)

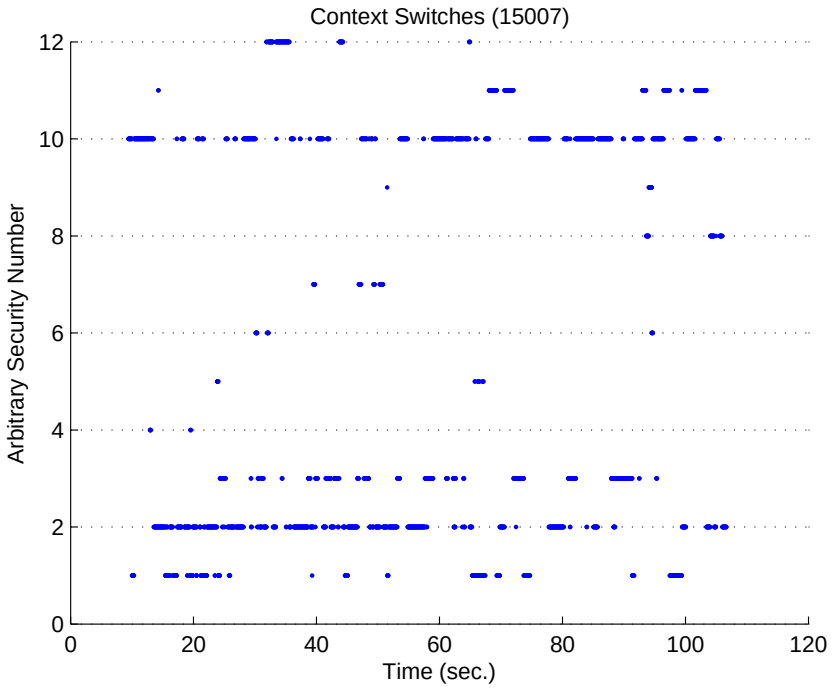

Figure 4-8: Switching contexts between different securities (15007)

of screen updates is a metric that attempts to capture the volume of information presented to a specialist. See Figure 4-10. 15003, the same trader who switched contexts the most, also experienced the greatest number of screen updates. Figure 4-11 plots the number of screen updates vs. the number of context switches. Since Table 4.1 is normalized to roughly 100 min., the highest update frequency is about six updates per second.

Table 4.1 includes data that will be discussed later, such as Total Profit and Loss and Capital. Profit and loss (P&L) is the amount of money the trader has gained or lost. Capital is the amount of money the trader has invested in the market. The return on investment (ROI) is the ratio of profit or loss relative to the maximum capital invested or borrowed. Figure 4-12 shows the number of Buy and Sell trades for each specialist. Figure 4-13 shows the profit and loss for each specialist. Figure 4-14 shows a scatterplot of Figure 4-12 and 4-13. Figure 4-15 shows a scatterplot of the profit and loss and mean capital.

4.2 Financial Data Collection

For each session, real-time market data for securities actively traded or monitored by the subject were recorded. The software that captured the information was a screen-scraping process that did not interfere with the workflow of the specialists. The specialists were able to personally configure their screens, allowing them to only view the information they wanted to see. Unfortunately, if the data did not appear on the screen, it was not recorded. The result is that some data is sometimes missing. For instance, for subject 15002, there is limited information about his positions since he suppressed that information. (The analysis below only uses the information that was recorded, as there is no way to impute the data.) Quotes were inconsistently captured by the log files of the specialists, so an independent data provider (the TAQ database) provided the information.

For each security, four time series were monitored: (1) the bid price P_t^b; (2) the ask price P_t^a; (3) the bid-ask spread $X_t \equiv P_t^b - P_t^a$; and (4) the net return $R_t \equiv (X_t - X_{t-1})/X_{t-1}$. Table 4-16 and Figure 4-17 display examples of the real-time market data, where Table 4-16 shows how the data is literally presented to the specialist on their computer terminals and Figure 4-17 shows the data collected over a 60-min. interval. In Figure 4-17, which did occur during the study, IBM's price dropped about 1%, erasing $1.25 billion of market capitalization in three to five minutes.

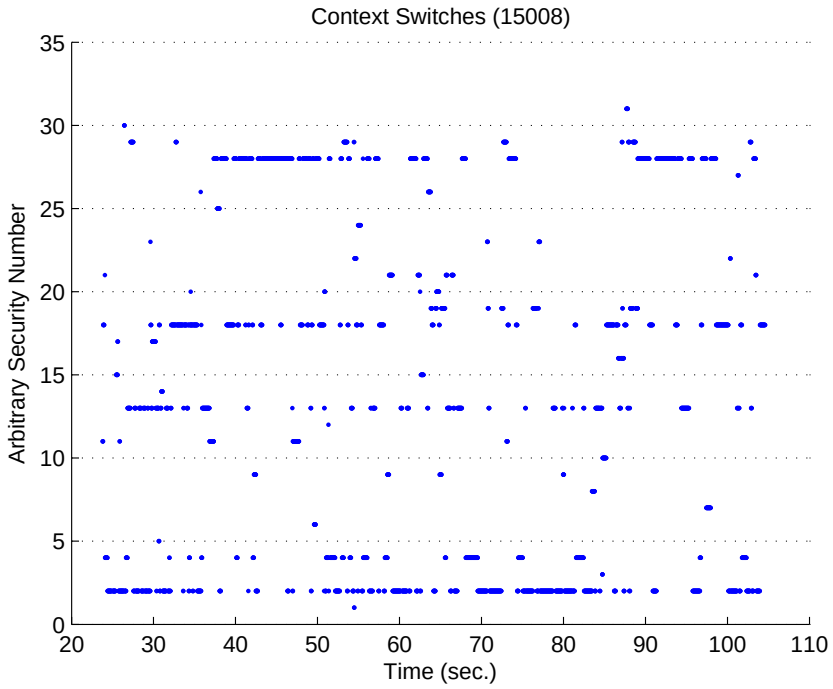

Figure 4-9: Switching contexts between different securities (15008)

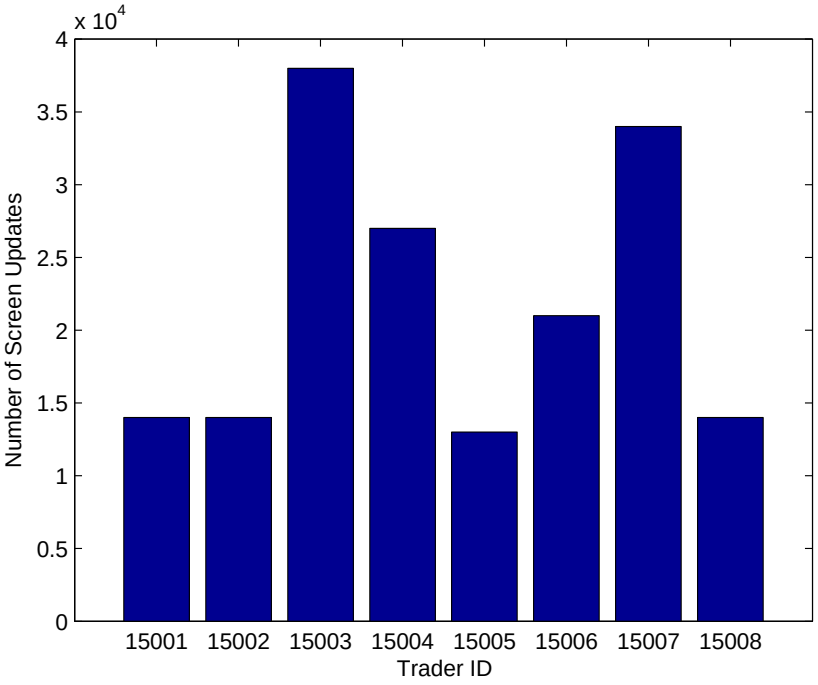

Figure 4-10: Number of screen updates

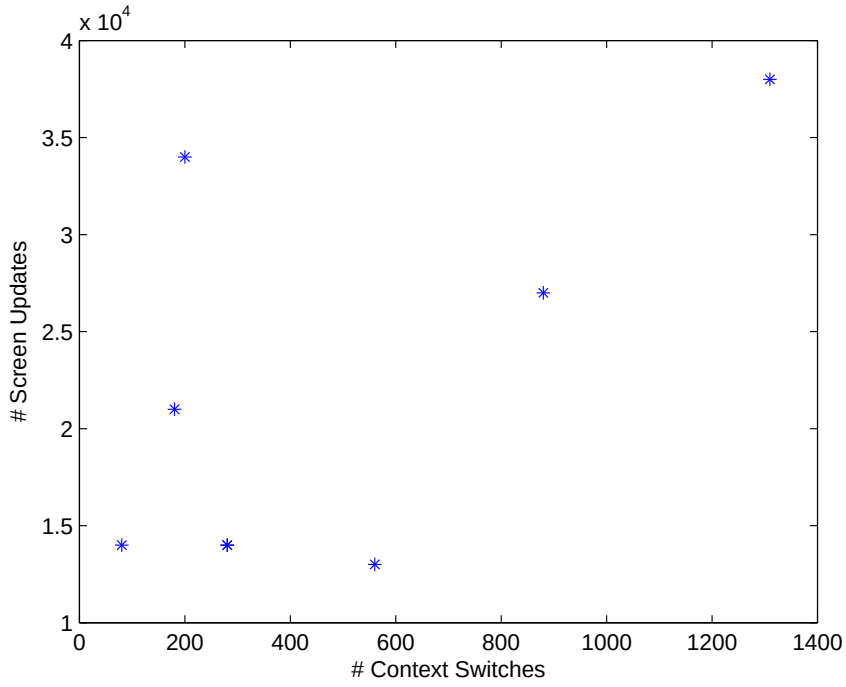

Figure 4-11: Number of screen updates vs. number of context switches

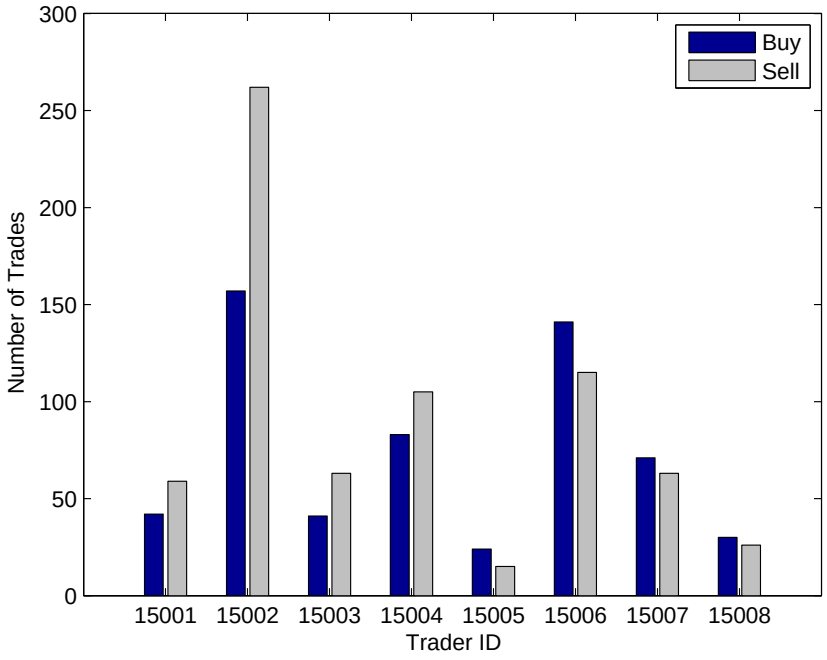

Figure 4-12: Number of trades

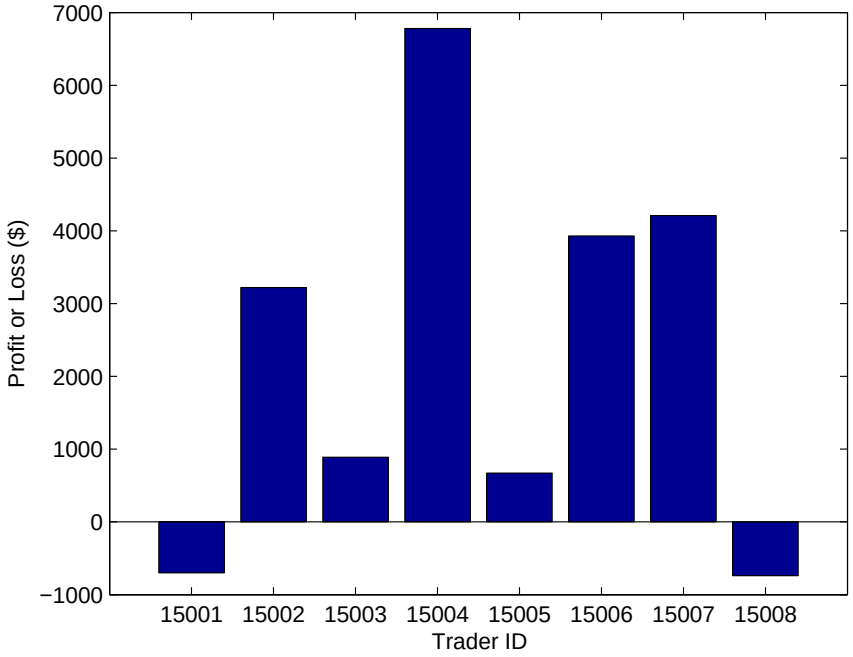

Figure 4-13: Profit and Loss

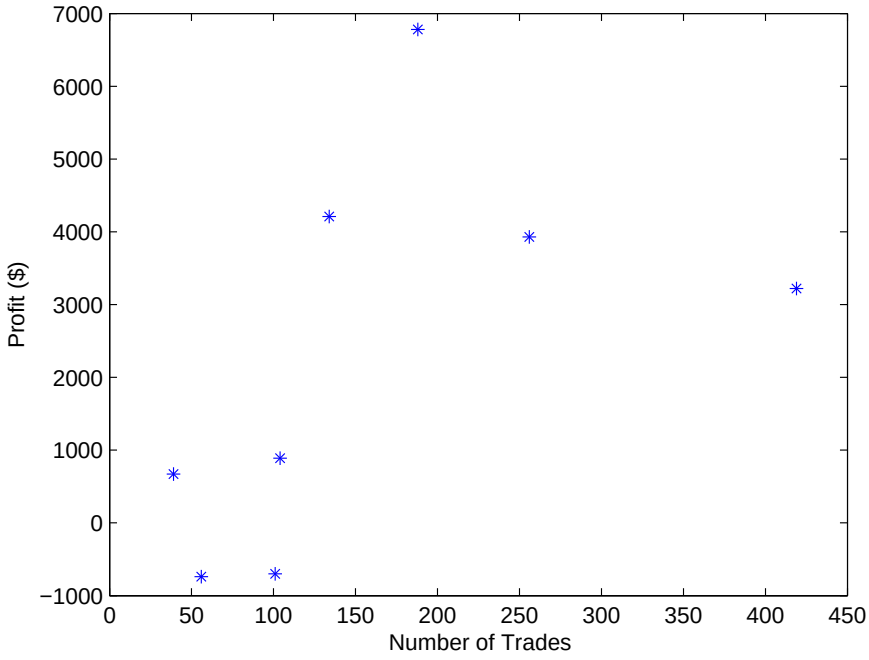

Figure 4-14: Profit and Loss vs. Number of Trades

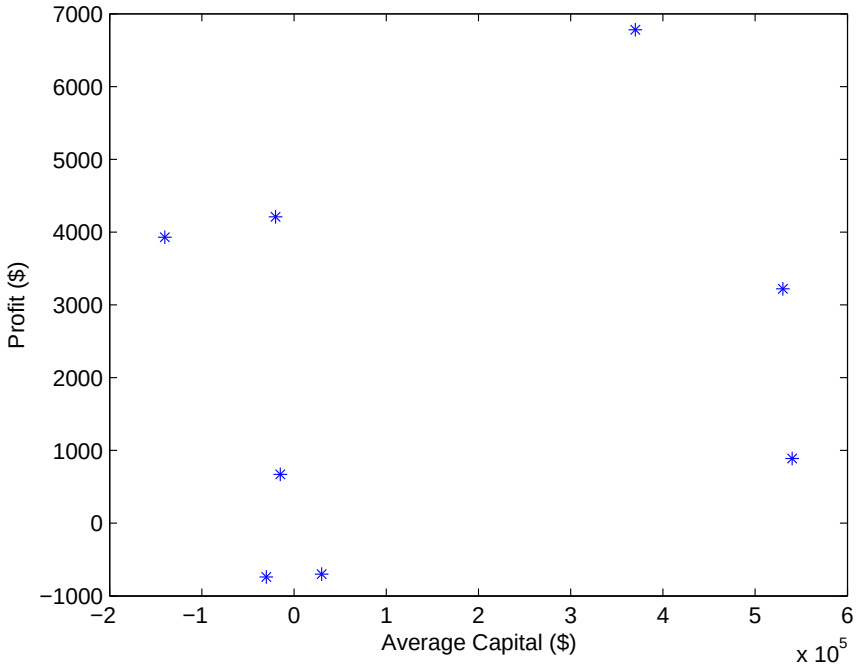

Figure 4-15: Profit and Loss vs. Average Capital Invested

```
IBM
  Bid     Offer              Time  Vol    Last
81        81.25  23x5    10:27:22
81.06     81.42   1x1    10:27:30
81.11     81.44  25x35   10:26:56
81.12   > 81.14   1x1    10:27:24  2.1M+ 81.13
81.13 <   81.21   8x6    10:27:24
                           :00:00
81.12     81.15  10x10   10:27:16
80.50     82.14   7x1    10:27:06

<> Indicate best Bid and Offer, respectively
```

Figure 4-16: Example of real-time market data presentation (IBM)

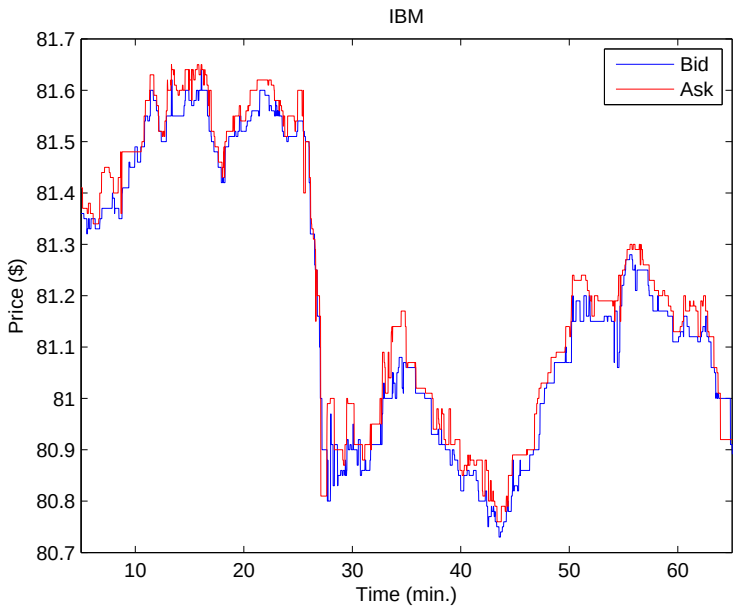

Figure 4-17: Example of real-time market data (IBM) recorded over 60 min.

Market makers on exchanges other than the Boston Stock Exchange may also quote prices for securities. Securities regulations require that transactions take place at the National Best Bid and Offer price (NBBO) (Schwartz and Francioni, 2004). The best bid is the highest bid and the best offer is the lowest offer. Along with price, size also matters when determining which exchange has the best bid or offer. Given the best quotes from all the market makers, I calculated the best bid and ask prices for all the securities.

In addition to the quote information, we also collected position and trade information for each security that was actively traded. (Although a specialist often holds inventory of securities which are not actively traded or even monitored and, without question, these securities impact the total profit and loss for each specialist, I chose to omit the contributions to the profit and loss in order to focus on those fluctuations in securities that held the attention of the specialist.) The validity of this assumption seems psychologically tenable. But the assumption may be questioned if the specialist is holding, say, a very large position. No specialist held any such large positions during the experiment.

From the position and trade information it is possible to calculate both profit and loss (P&L) and amount of capital invested. P&L calculations require historical information of average transaction price. In the few cases where the average transaction price is not available the P&L is a relative number, where relative means that the P&L is calculated

just for the day (as if the opening position was flat). For instance, if a specialist is short 100 shares of IBM at the open and the transaction price of shorting these 100 shares is unknown, buys 500 shares, and then sells 300 shares, the relative P&L is calculated on the 300 shares bought and sold. In contrast, the absolute P&L would be calculated on 400 shares.

The amount of capital invested is the dollar amount invested in the market. It is the difference between the value of the long and short positions. For instance, if a specialist is long $1,000,000 and short $250,000 then the amount of capital invested in the market is $750,000. Table 4.1 lists the average, min, and max capital invested by each trader during the experiment, including ROI. It should be obvious that gains generated by smaller amounts of capital are better than gains generated by larger amounts of capital. Thus, while 15004 earned a profit of $6,800, the trader required $520,000 of capital on average and $1,040,000 of capital in the extreme. In contrast, 15006 managed to earn $3,900 from a more modest $-60,000 of average capital, generating a 1% return.

About 20 percent of the securities have unknown opening positions. According to the literature on missing data, there are multiple types of missingness. This missingness is not *missing completely at random (MCAR)* because the missingness is not randomly distributed across all observations: securities with missing data were those that were not aggressively traded or monitored. Thus, this missingness is *missing at random (MAR)*. However, rather than impute opening positions—a piece of information that was critical to the analysis—we did not compute P&L on any security until a position was provided. It is unknown whether the missingness introduces bias into the analysis below. Table 4.1 lists the relative P&L for all of the traders. Figure 4-12 shows the number of trades.

Any non-zero position in a security generates a running profit and loss. I call the sum of all the securities' profits and losses the overall P&L or total P&L. The analysis below will sometimes differentiate between single security P&L and overall P&L. The reason for this is that there may be moments when overall P&L may be steady, whereas two of the underlying securities may be gaining and losing in the same proportions. It seems to hold, as discussed later in the section on Prospect Theory, that individuals process—and react to—gains and losses, whether those gains and losses belong to the overall portfolio or to its components. After all, how many times have I fretted over a single losing trade when my overall portfolio was actually winning? When analyzing P&L, I indicate whether the

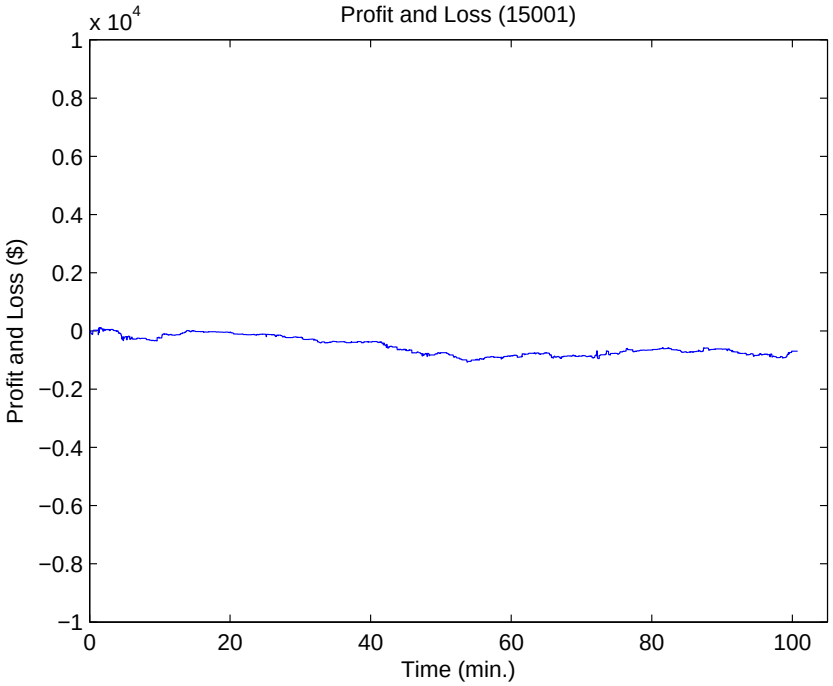

Figure 4-18: Profit and Loss (15001)

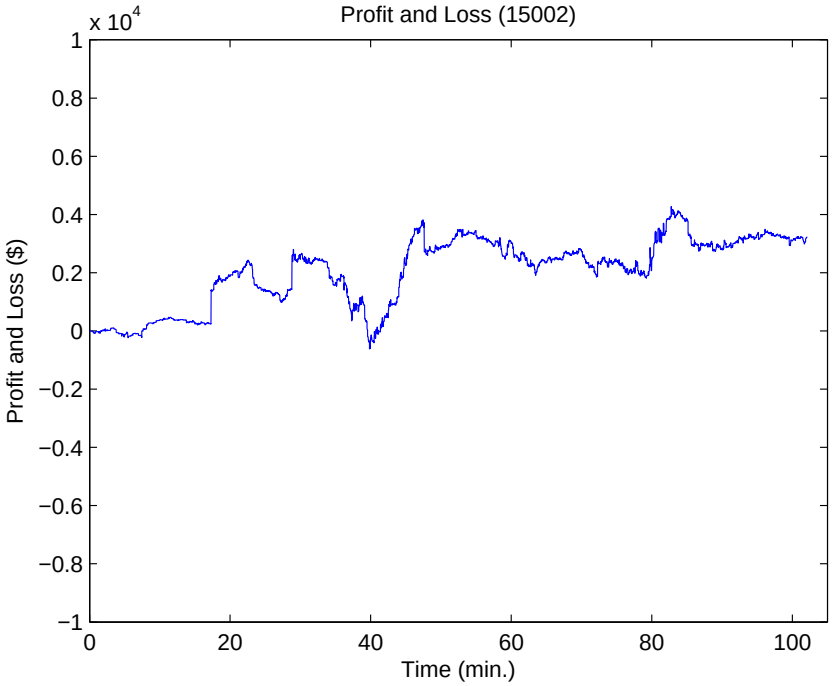

Figure 4-19: Profit and Loss (15002)

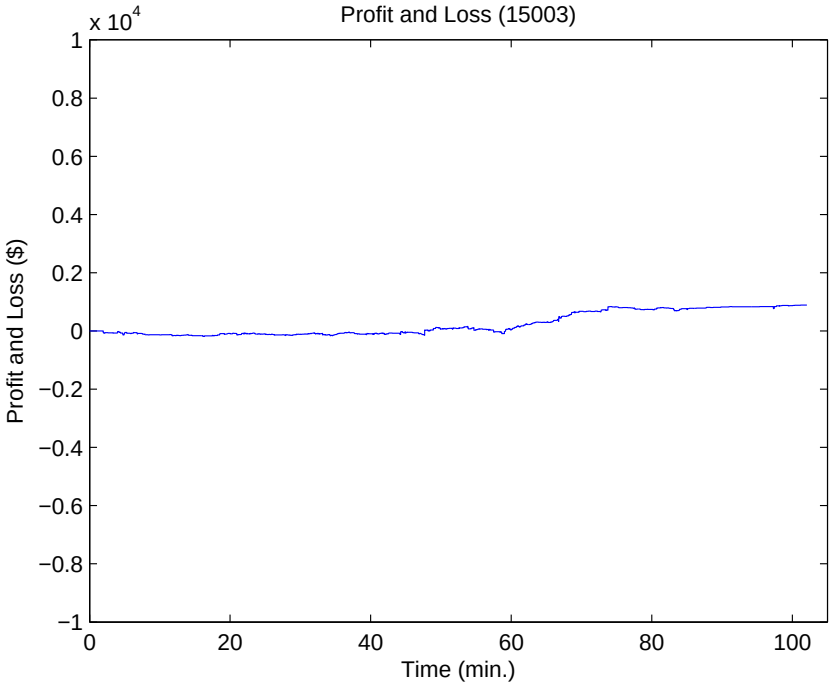

Figure 4-20: Profit and Loss (15003)

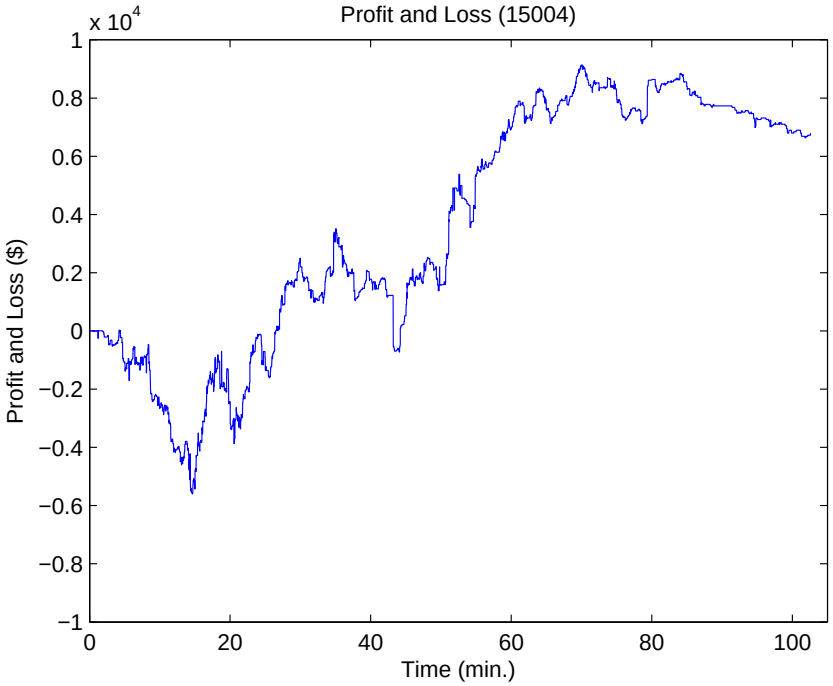

Figure 4-21: Profit and Loss (15004)

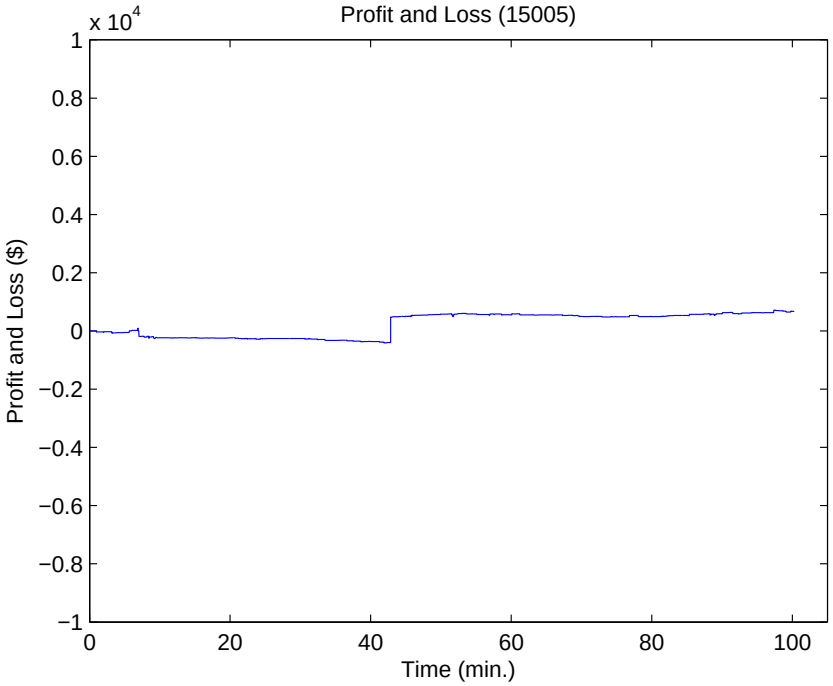

Figure 4-22: Profit and Loss (15005)

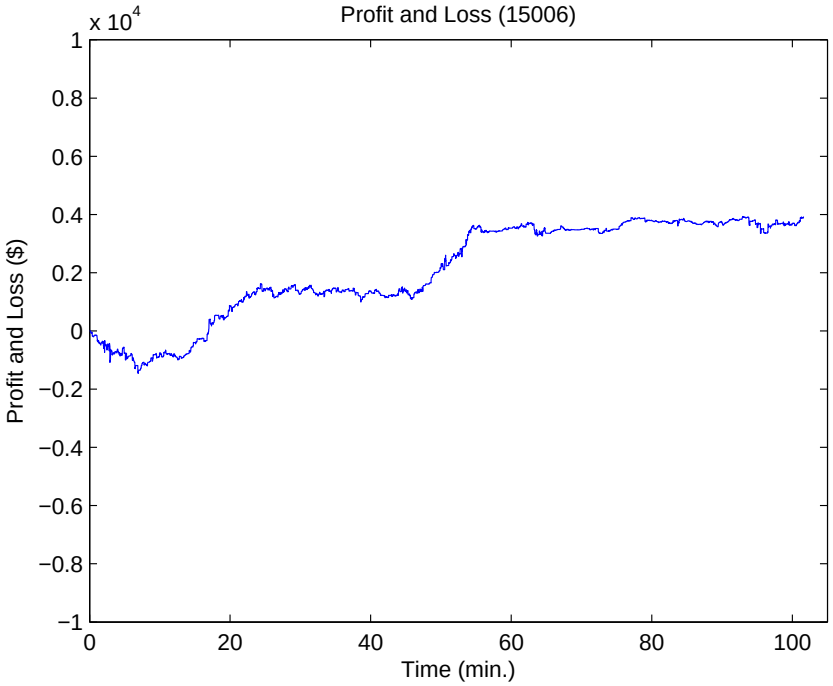

Figure 4-23: Profit and Loss (15006)

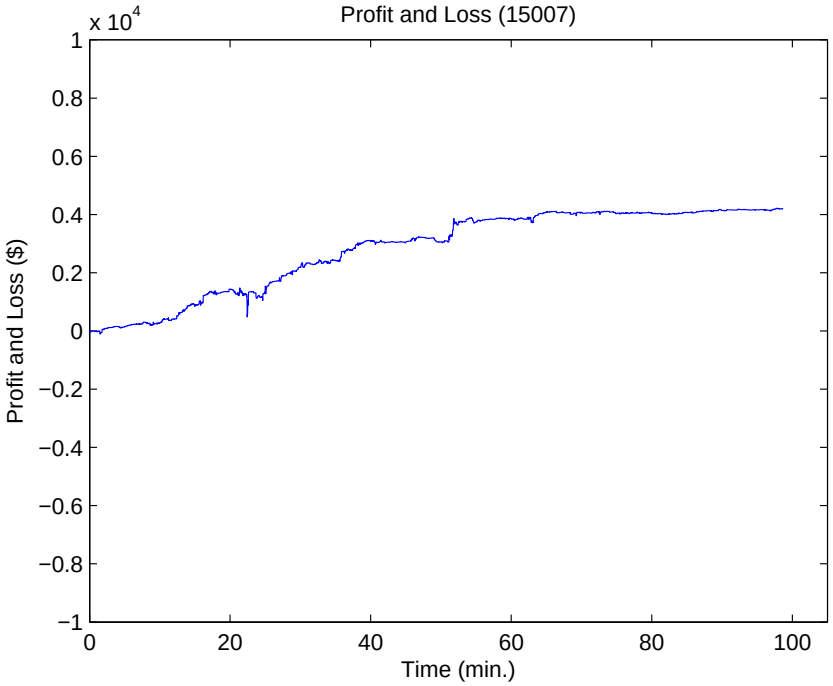

Figure 4-24: Profit and Loss (15007)

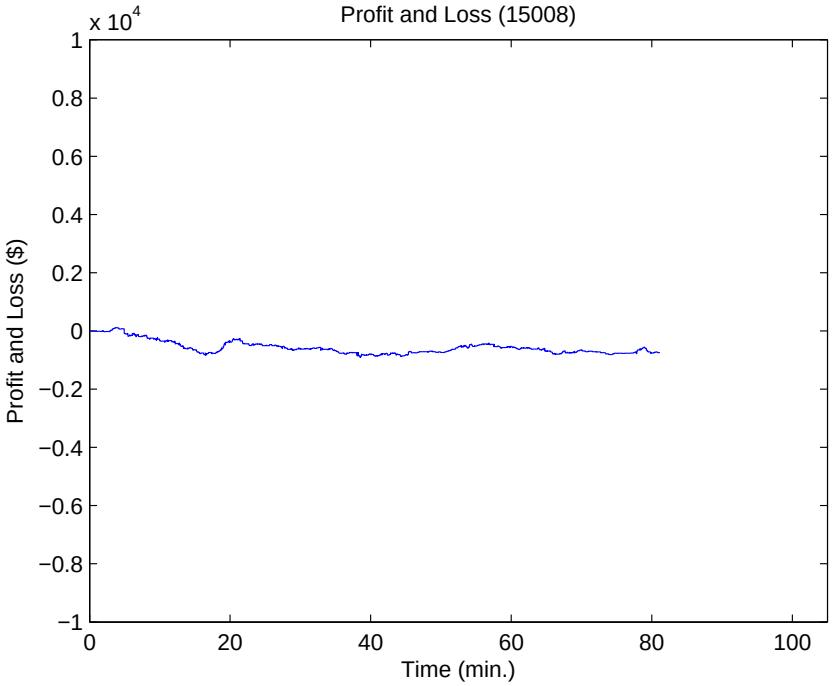

Figure 4-25: Profit and Loss (15008)

analysis uses overall P&L or single security P&L.

Although the financial data originated on the exchange, the data is plagued with errors. There are problems with quotes, trades, and time-stamps. For instance, 15004 sold 50 shares at a price of $39.98 at 9:31:01am, but the best bid and offer that were posted for that moment were $38.80 and $45.39, respectively. Two seconds later, the best bid and offer were $39.90 and $45.00. Thirty-three seconds later, the best bid and offer were $39.95 and $40.00. While the exchanges are supposedly synchronized to the same clock, it appears that there are minor differences, especially during the volatile opening of the market. When data errors, such as these, were discovered, they were manually corrected. In addition there are multiple instances of incorrect (inverted) bid-ask spreads (see Figure 4-17 which shows an inverted bid-ask spread between the $27^{\text{th}}$and 28^{th}minutes). I used filters similar to those described by (Blume and Goldstein, 1997) to eliminate quote data—but not trade data—which appeared to be erroneous. Since (Blume and Goldstein, 1997) is a decade old, I used different parameters. For instance, instead of using a threshold of 20% for valid spreads, I used a spread of $1.25 to eliminate quotes. In addition, I attempted to correct inverted bid-ask spreads by a simple algorithm. See `fixbidask.m` in Appendix D for details. (Brownlees and Gallo, 2006; Falkenberry, 2002) discuss similar problems with Trade and Quote (TAQ) data.

4.3 Financial Data Feature Extraction

From the market data, I identified three classes of market events: deviations (DEV), trend-reversals (TRV), and volatility (VOL) events. These events are often cited by traders as significant developments that require heightened attention, potentially signaling a shift in market dynamics and risk exposures.

Deviation and trend-reversal events were defined to last 10 seconds. The 10-sec interval length was chosen because the slowest varying psychophysiological response lasts 10 seconds. Volatility events were defined to last 5 minutes because volatility requires more than 10 data points.

Of the six types of events, three were deviations, two were trend-reversals, and one was a volatility event type:

1. mid-price deviations

2. spread deviations
3. return deviations
4. mid-price trend-reversals
5. spread trend-reversals
6. maximum mid-price volatility

Deviations of a time series $\{Z_t\}$ were defined as those instants t when the time series deviated from the series mean by a certain threshold, where the threshold was defined as a multiple k of the standard deviation σ_z of the time series. Positive deviation events were defined at those instants t where $Z_t > \bar{Z} + k\sigma_z$, and negative deviations were defined at those instants t where $Z_t < \bar{Z} - k\sigma_z$. The series mean, $\bar{Z}$, was calculated over the length of the interval, which was defined to be 10 sec. The value of the multiplier k varied from series to series. After ordering all of a subject's time series by the absolute value of k, the top 20 instants from all the market data for each subject were selected as deviation events. This procedure was done for each of the three deviation events using different Zs.

Trend-reversal events were defined as instants when a time series $\{Z_t\}$ intersected its 5-min moving-average, $\mathrm{MA}_{5-min}(Z_t)$. Positive and negative trend-reversals events were defined at those instants t where $Z_t > (1+\delta)\mathrm{MA}_{5-min}(Z_t)$ and $Z_t < (1-\delta)\mathrm{MA}_{5-min}(Z_t)$, respectively. As with the case of the deviation events, the δ values varied for each time series. After ordering all of a subject's time series by the absolute value of δ, the top 20 instants for each subject were selected as trend-reversal events. This procedure was done for both of the two trend-reversal events. The reason a return trend-reversal event was not defined was due to the sampling frequency of the time series data. By sampling the pricing data every second, most observations did not vary from one observation to the next. Hence, most of the return values were zero, making it difficult to define trends in returns.

By definition, the volatility event was calculated over a 5-min. interval. The volatility event was based on the following statistic, which we call *maximum volatility* because it is the difference between the maximum and minimum prices:

$$\sigma_j = \frac{\max_{t_j-300<\tau\leq t_j} P_\tau - \min_{t_j-300<\tau\leq t_j} P_\tau}{\frac{1}{2}(\max_{t_j-300<\tau\leq t_j} P_\tau + \min_{t_j-300<\tau\leq t_j} P_\tau)}$$

where P is the mid-price, j is an index of the interval, and t_j is the end time (in sec.) of the j^{th} interval. Positive and negative volatility events were defined at those instants when

$\sigma_j > (1+\eta)\sigma_{j-1}$ and $\sigma_j < (1-\eta)\sigma_{j-1}$, respectively. Again, the η values varied from series to series. After ordering all of a subject's time series by the absolute value of η, the top 5 instants for each subject were selected as volatility events. Since a 100-min time series contains 20 5-min. intervals, I chose 5 intervals because I did not want more than 25% of the time series to be labeled as volatile.

With these definitions and calibrations in hand, I implemented an automatic procedure for detecting deviations, trend-reversals, and volatility events for all the relevant time series in each session.

4.4 Physiological Data Collection

In addition to the market data, eight physiological variables were simultaneously monitored in real time during the duration of each session while the subjects sat at their trading consoles:

1. SCR
2. BVP
3. ECG
4. EEG
5. EMG-Arm
6. EMG-Back
7. Temp-Finger
8. Temp-Head

Because two variables were the same type, only six types of sensors were used.

The benefits of acquiring real-time physiological measurements in vivo must be balanced against the cost of measurement error, spurious signals, and other statistical artifacts, which, if ignored, can obscure and confound any genuine signals in the data. A ProComp+ and ProComp Infiniti data-acquisition unit and Biograph biofeedback software from Thought Technology (Tho) were used to measure and record physiological data for all subjects. The eight sensors were connected to the small, battery-powered control unit, which was placed on each subject's belt. A fiber optic cable connected the unit to a laptop computer equipped with real-time data acquisition software. Each sensor was equipped with a built-in notch filter at 60 Hz for automatic elimination of external power line noise, and standard AgCl

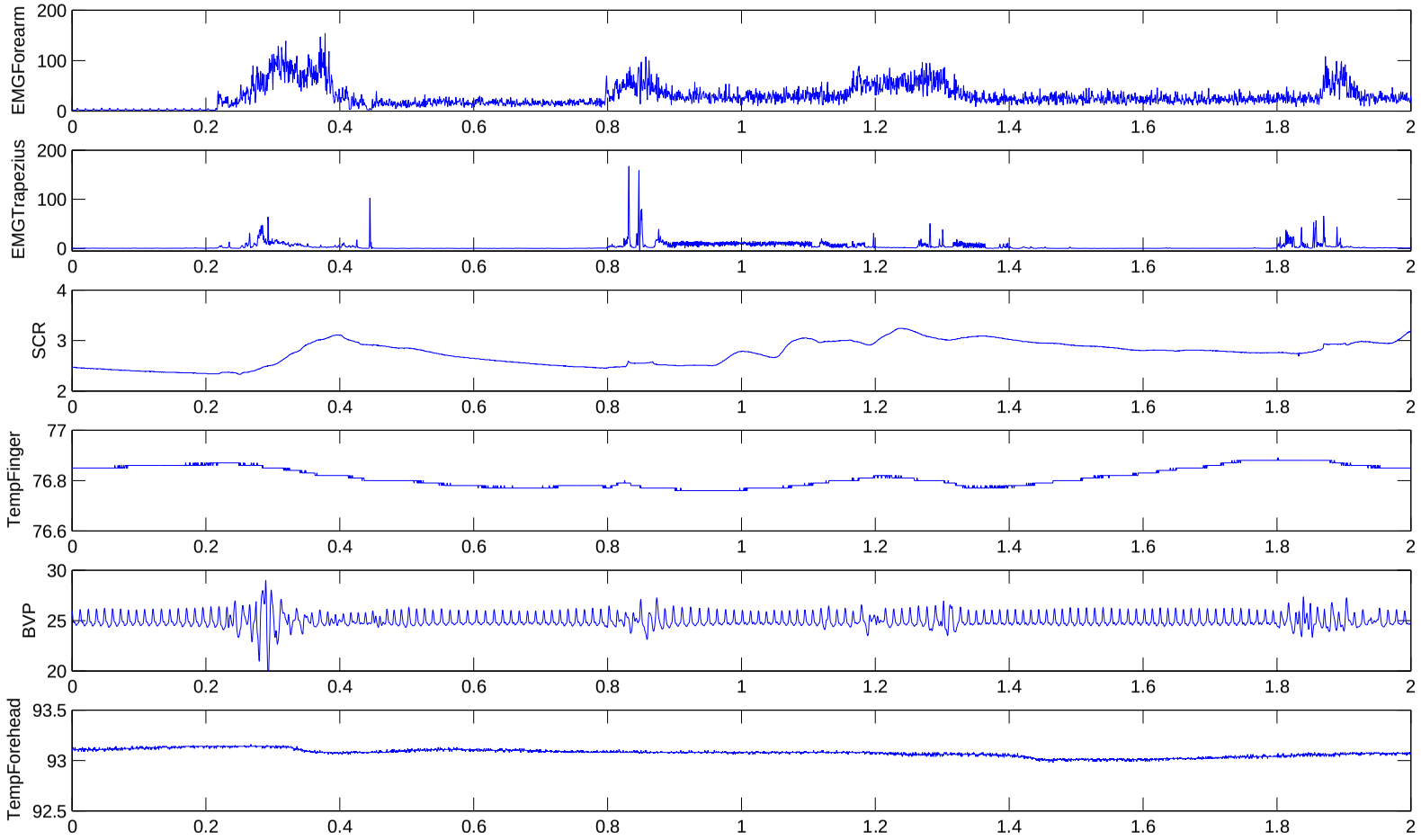

Figure 4-26: Recordings of 32 hz physiological signals (15001)

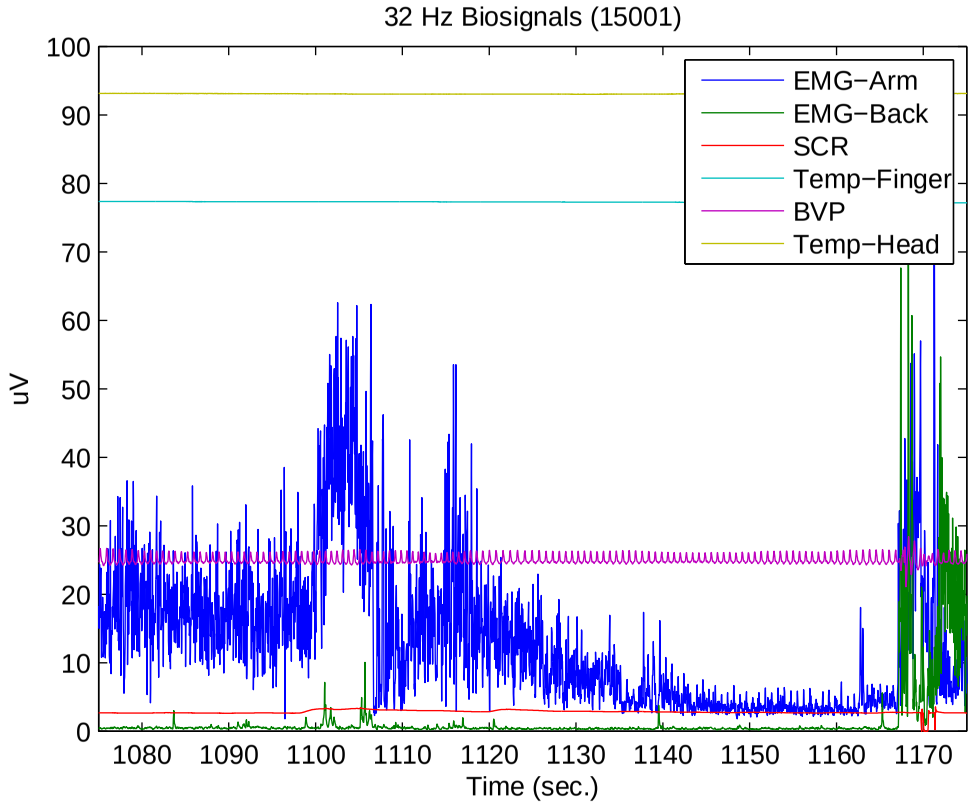

Figure 4-27: Recordings of 32 hz physiological signals on same scale (15001)

triode and single electrodes were used for SCR and EMG sensors, respectively. The sampling rate for ECG and EEG was 256 Hz. For the other signals, the sampling rate was 32 Hz.

The procedure of outfitting each subject required approximately five minutes and was performed prior to the opening bell. The SCR, BVP, EMG-Arm, and Temp-Finger were recorded on the subject's non-dominant arm. Subjects indicated that the presence of the sensors, wires, and a control unit did not compromise or influence their trading in any significant manner, and that their workflow was not impaired in any way. This was verified not only by the subjects but also by their supervisors. Given the magnitudes of the financial transactions that were being processed, and the economic and legal responsibilities that the subjects and their firms bore, even the slightest interference with the subjects' workflow or performance standards would have caused the supervisors or the subjects to terminate the sessions immediately. None of the sessions were terminated prematurely.

At the start of each session, a common time-marker was set in the biofeedback unit and in the subject's trading console. Because the biofeedback unit and the trading console used different clocks, periodic markers were also set. I used these markers to synchronize the physiological and financial time series data.

4.4.1 Statistical Signal Processing

Figures 4-26 and 4-27 show physiological data spanning one to two minutes for a single trader. The physiological data for the other traders is similar. Table 4.2 shows summary statistics for the physiological data.

Correlation Table 4.3 shows correlations between physiological signals and Table 4.4 shows the correlations when averaged over all traders.

Table 4.5 shows the autocorrelation[1] for the SCR of 15001. This SCR is very autocorrelated. Table 4.6 shows the autocorrelation of the first difference for the SCR of the same trader. The difference is not autocorrelated.

Figure 4-28 is the graphical form of the autocorrelation tables (not included) for the

[1]By way of reference, the autocorrelation R_{xx} is

$$R_{xx}(m) = \mathrm{E}[x_{n+m}x_n] = \mathrm{E}[x_n x_{n-m}]$$

where x_n is a stationary random processes and E[] is the expected value operator. If R_{xx} is normalized so that $R_{xx}(0) = 1$ (the autocorrelation at lag zero is unity), the results are called the autocorrelation coefficients.

		Min	Q_1	Q_2	*Mean*	Q_3	*Max*	*Range*	*Std*	*Kurtosis*	*Skewness*
15001	EMG-Arm	-1	2.74	10.61	14.59	20.60	203	204	14.56	7.67	1.83
	EMG-Back	-12	0.41	1.40	8.77	9.78	284	296	15.07	15.03	2.77
	SCR	0	2.04	2.51	2.39	2.86	4.55	4.55	0.68	3.05	-0.56
	BVP	0	23.68	24.63	25.05	25.68	100	100	4.52	15.94	2.06
	Temp-Head	92.5	93.02	93.12	93.11	93.20	93	0.99	0.16	3.66	-0.27
15002	EMG-Arm	0	2.02	3.52	4.08	5.23	56.0	56.0	3.33	9.73	1.73
	EMG-Back	0.4	0.83	2.17	3.27	4.45	67	66.46	3.54	18.80	2.98
	SCR	0	6.62	7.26	7.14	7.91	12.45	12.45	1.48	9.44	-1.56
	BVP	1.4	33.67	37.17	37.03	40.28	69.68	68.25	4.59	3.09	-0.03
	Temp-Head	85	88.45	89.28	89.11	90.09	92.01	7.06	1.41	3.10	-0.61
15003	EMG-Arm	0	10.35	18.69	21.3	30.90	191	192	13.54	3.58	0.77
	EMG-Back	-9	0.47	0.98	10.9	12.42	212	222	18.56	5.51	1.82
	SCR	0	30.00	30.00	28.9	30.0	30.0	30.0	5.09	28.06	-5.06
	BVP	0	17.99	21.61	25.79	33.0	100	100	10.40	2.84	0.91
	Temp-Head	91	92.72	93.24	93.0	93.45	94.06	2.91	0.50	3.45	-1.00
15004	EMG-Arm	0.1	3.7	5.6	6.1	7.9	42.4	42.3	3.6	7.1	1.4
	EMG-Back	0.2	0.6	0.9	1.4	1.4	124.3	124.1	2.6	613.7	20.1
	SCR	0	6.4	8.4	11.6	15.5	30.0	30.0	7.8	2.8	1.1
	BVP	0	35.2	36.8	37.0	38.8	74.6	74.6	3.5	11.0	0.0
	Temp-Head	87.8	89.7	91.0	90.9	92.0	93.8	6.1	1.4	2.1	-0.1
15005	EMG-Arm	1	2.48	4.87	9.33	9.01	412	411	16.78	111	7.88
	EMG-Back	-12	0.57	0.83	3.61	1.40	252	264	10.15	71.14	6.67
	SCR	0	6.88	7.66	7.42	8.29	12.76	12.76	1.68	9.69	-1.99
	BVP	0	22.41	23.97	24.98	26.66	100	100	6.13	16.73	2.09
	Temp-Head	91	91.93	92.16	92.11	92.33	92.74	1.99	0.32	3.81	-0.81
15006	EMG-Arm	0	0.98	1.86	2.47	3.52	140	140	2.05	145	4.17
	EMG-Back	0	0.52	0.83	1.67	2.02	266	266	4.76	1460	35.10
	SCR	0	3.66	4.14	4.13	4.67	9.22	9.22	1.06	7.00	-0.62
	BVP	0	35.62	36.72	37.07	38.48	100	100	4.47	33.17	0.72
	Temp-Head	85	88.88	90.28	89.84	91.13	92.46	7.47	1.67	2.84	-0.86
15007	EMG-Arm	-12	2.23	2.74	5.35	3.99	412	424	9.99	83.88	6.80
	EMG-Back	-2	14.39	30.33	39.52	57.40	412	414	33.64	5.23	1.31
	SCR	0	6.39	7.51	7.55	8.77	15.42	15.42	1.81	4.06	-0.00
	BVP	0	21.30	23.79	25.47	28.54	100	100	8.16	11.35	1.48
	Temp-Head	93	94.89	95.05	94.95	95.13	95.33	2.54	0.32	14.38	-3.01
15008	EMG-Arm	1	1.24	1.55	4.13	3.73	91.82	91	5.58	9.86	2.46
	EMG-Back	-12	0.36	1.24	4.84	5.02	412	424	9.19	180	7.42
	SCR	2	2.34	3.21	3.15	3.73	6.45	4.81	0.85	2.36	0.20
	BVP	0	23.79	24.57	24.86	25.76	100	100	2.45	203	6.53
	Temp-Head	75	94.94	95.23	95.04	95.80	96.11	20.65	2.08	65.35	-7.56

Table 4.2: Summary statistics of physiological data for all traders

15001	*EMG-Arm*	*EMG-Back*	*SCR*	*BVP*	*Temp-Head*
EMG-Arm	1.00	-0.12	0.23	-0.01	-0.30
EMG-Back	-0.12	1.00	0.15	-0.00	0.08
SCR	0.23	0.15	1.00	-0.01	-0.05
BVP	-0.01	-0.00	-0.01	1.00	-0.00
Temp-Head	-0.30	0.08	-0.05	-0.00	1.00
15002	*EMG-Arm*	*EMG-Back*	*SCR*	*BVP*	*Temp-Head*
EMG-Arm	1.00	0.07	0.25	0.00	0.25
EMG-Back	0.07	1.00	-0.09	-0.01	-0.06
SCR	0.25	-0.09	1.00	-0.00	0.33
BVP	0.00	-0.01	-0.00	1.00	-0.00
Temp-Head	0.25	-0.06	0.33	-0.00	1.00
15003	*EMG-Arm*	*EMG-Back*	*SCR*	*BVP*	*Temp-Head*
EMG-Arm	1.00	0.44	-0.08	-0.04	0.04
EMG-Back	0.44	1.00	-0.12	-0.00	0.27
SCR	-0.08	-0.12	1.00	-0.00	0.20
BVP	-0.04	-0.00	-0.00	1.00	-0.00
Temp-Head	0.04	0.27	0.20	-0.00	1.00
15004	*EMG-Arm*	*EMG-Back*	*SCR*	*BVP*	*Temp-Head*
EMG-Arm	1.00	0.07	-0.01	-0.04	-0.14
EMG-Back	0.07	1.00	0.08	0.03	-0.09
SCR	-0.01	0.08	1.00	0.00	-0.61
BVP	-0.04	0.03	0.00	1.00	0.00
Temp-Head	-0.14	-0.09	-0.61	0.00	1.00
15005	*EMG-Arm*	*EMG-Back*	*SCR*	*BVP*	*Temp-Head*
EMG-Arm	1.00	0.17	-0.16	-0.01	-0.20
EMG-Back	0.17	1.00	-0.19	0.01	-0.12
SCR	-0.16	-0.19	1.00	-0.01	0.43
BVP	-0.01	0.01	-0.01	1.00	-0.01
Temp-Head	-0.20	-0.12	0.43	-0.01	1.00
15006	*EMG-Arm*	*EMG-Back*	*SCR*	*BVP*	*Temp-Head*
EMG-Arm	1.00	0.16	0.08	-0.04	-0.06
EMG-Back	0.16	1.00	0.01	-0.01	0.04
SCR	0.08	0.01	1.00	-0.01	0.22
BVP	-0.04	-0.01	-0.01	1.00	-0.00
Temp-Head	-0.06	0.04	0.22	-0.00	1.00
15007	*EMG-Arm*	*EMG-Back*	*SCR*	*BVP*	*Temp-Head*
EMG-Arm	1.00	0.26	-0.08	0.04	-0.05
EMG-Back	0.26	1.00	-0.05	0.02	-0.19
SCR	-0.08	-0.05	1.00	0.00	-0.36
BVP	0.04	0.02	0.00	1.00	-0.05
Temp-Head	-0.05	-0.19	-0.36	-0.05	1.00
15008	*EMG-Arm*	*EMG-Back*	*SCR*	*BVP*	*Temp-Head*
EMG-Arm	1.00	0.31	0.17	0.01	-0.22
EMG-Back	0.31	1.00	0.08	0.02	-0.10
SCR	0.17	0.08	1.00	-0.00	0.03
BVP	0.01	0.02	-0.00	1.00	-0.01
Temp-Head	-0.22	-0.10	0.03	-0.01	1.00

Table 4.3: Correlations of physiological data

	EMG-Arm	*EMG-Back*	*SCR*	*BVP*	*Temp-Head*
EMG-Arm	1.00				
EMG-Back	0.17	1.00			
SCR	0.05	-0.02	1.00		
BVP	-0.01	0.01	-0.00	1.00	
Temp-Head	-0.08	-0.02	0.03	-0.01	1.00

Table 4.4: Average correlation of physiological data for all traders

Lag	*Autocorrelation*
0	1.0000
1	0.9998
2	0.9994
3	0.9993
4	0.9991
5	0.9989
6	0.9988
7	0.9986
8	0.9985
9	0.9984
10	0.9982

Table 4.5: Autocorrelation of SCR (15001)

other 32 Hz physiological signals, BVP, EMG, and Temp. Figure 4-29 is the partial autocorrelation for BVP, EMG, SCR, and Temp.

Spectral Analysis Spectral analysis is a frequency-based method that describes the distribution (over frequency) of the power contained in a signal, based on a finite set of data. I use spectral techniques to analyze the EEG.

The power spectral density of a stationary random process, x_n, is mathematically related to the correlation sequence by the discrete-time Fourier transform (DTFT). In terms of

Lag	*Autocorrelation*
0	1.0000
1	0.1528
2	-0.3220
3	-0.0237
4	0.0397
5	-0.0267
6	-0.0607
7	-0.0303
8	0.0693
9	0.0838
10	0.0396

Table 4.6: Autocorrelation of changes in SCR (15001)

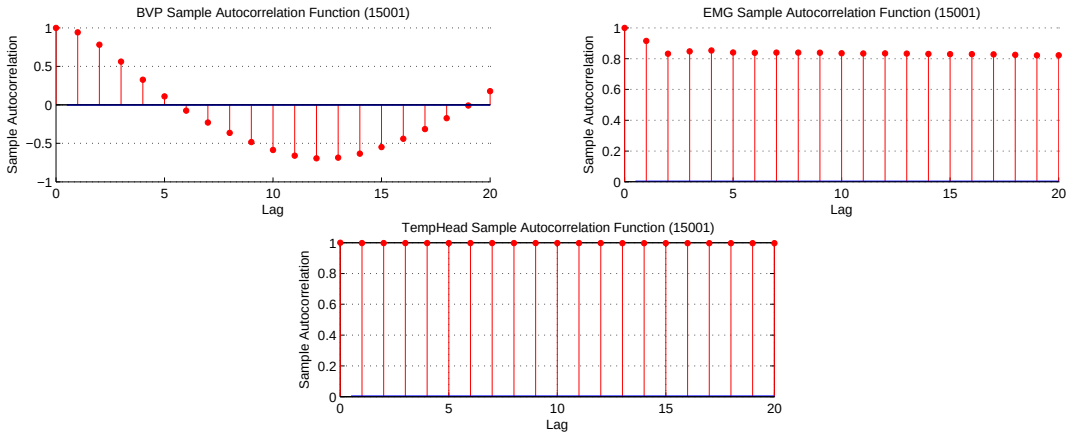

Figure 4-28: Autocorrelations of BVP, EMG, and Temp (15001).

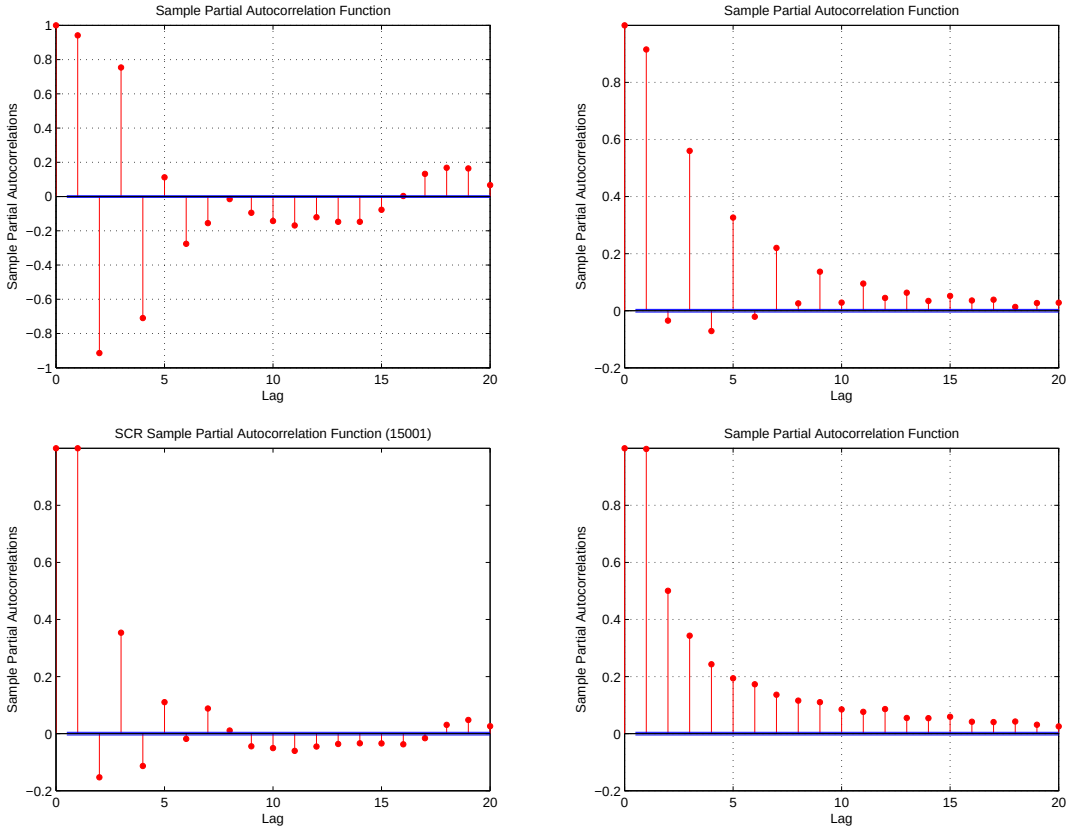

Figure 4-29: Partial autocorrelations of BVP, EMG, SCR, and Temp (15001).

normalized frequency, the power spectral density, P_{xx}, is

$$P_{xx}(\omega) = \frac{1}{2\pi} \sum_m R_{xx}(m) e^{-j\omega m}$$

where $\omega = 2\pi f$ and the units of $P_{xx}(\omega)$ are watts/radian/sample or simply watts/radian.

There are three kinds of methods which are used to estimate the power spectral density (PSD): (1) Nonparametric methods, (2) Parametric methods, and (3) Subspace methods. I use the nonparametric method. Nonparametric methods are those in which the PSD is estimated directly from the signal itself. The simplest such method is the *periodogram*. (An improved version of the periodogram is Welch's method and a more modern nonparametric technique is the multitaper method.) See (Mat, 2006) for details.

Using spectral techniques, it is possible to view gamma band activity of the EEG. The gamma band is the frequency band often associated with attention and cognitive processing.

The periodogram of each trader's EEG appears in Figures 4-30 and 4-31. Every periodogram shows gamma band activity with the peak at the center of the gamma band (40 Hz). While the periodogram shows the spectral composition of the entire signal, the spectrogram shows the spectral decomposition as it changes in time. Figure 4-32 displays the spectrogram of the gamma band. To clean the signal, I replace the ten extreme high and low values of the power spectral density signal with a local average computed from one percent of the extreme values. See `surfeeg.m` in Appendix D for source code. The figure is the sum of all the spectral components in the gamma band. The figure also includes the market's opening bell ('o') and all the trades ('*').

The EEG appears to change composition when the market opens. In almost every specialist, the spectrum transitions from before vs. after the market open. The spectrogram shows that some of the specialists maintain a uniform mental state throughout—15007—and some do not—15005. However, there does not appear to be any obvious relationship between gamma band activity and trading activity.

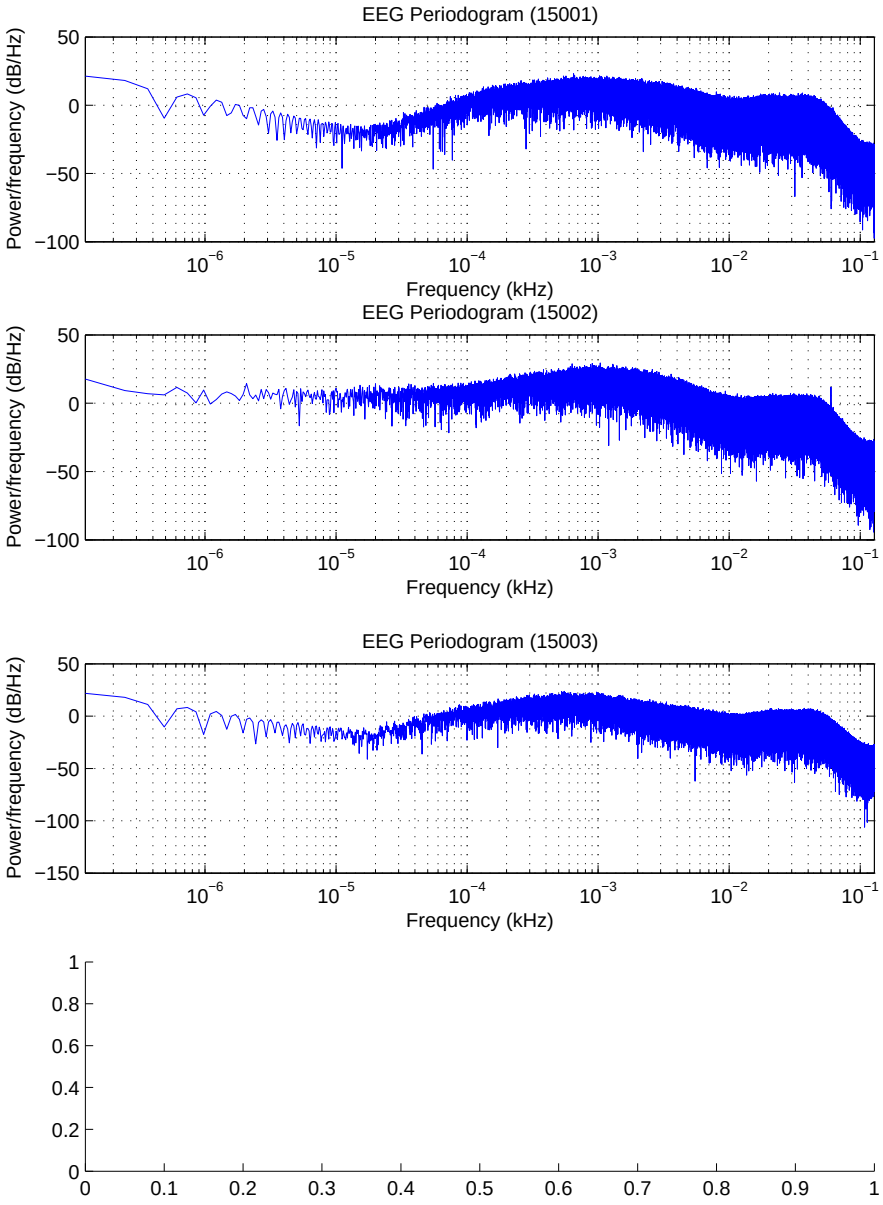

Figure 4-30: Power spectral density estimate via periodogram of EEG on log plot (15001–4). Note the peak near 40 Hz.

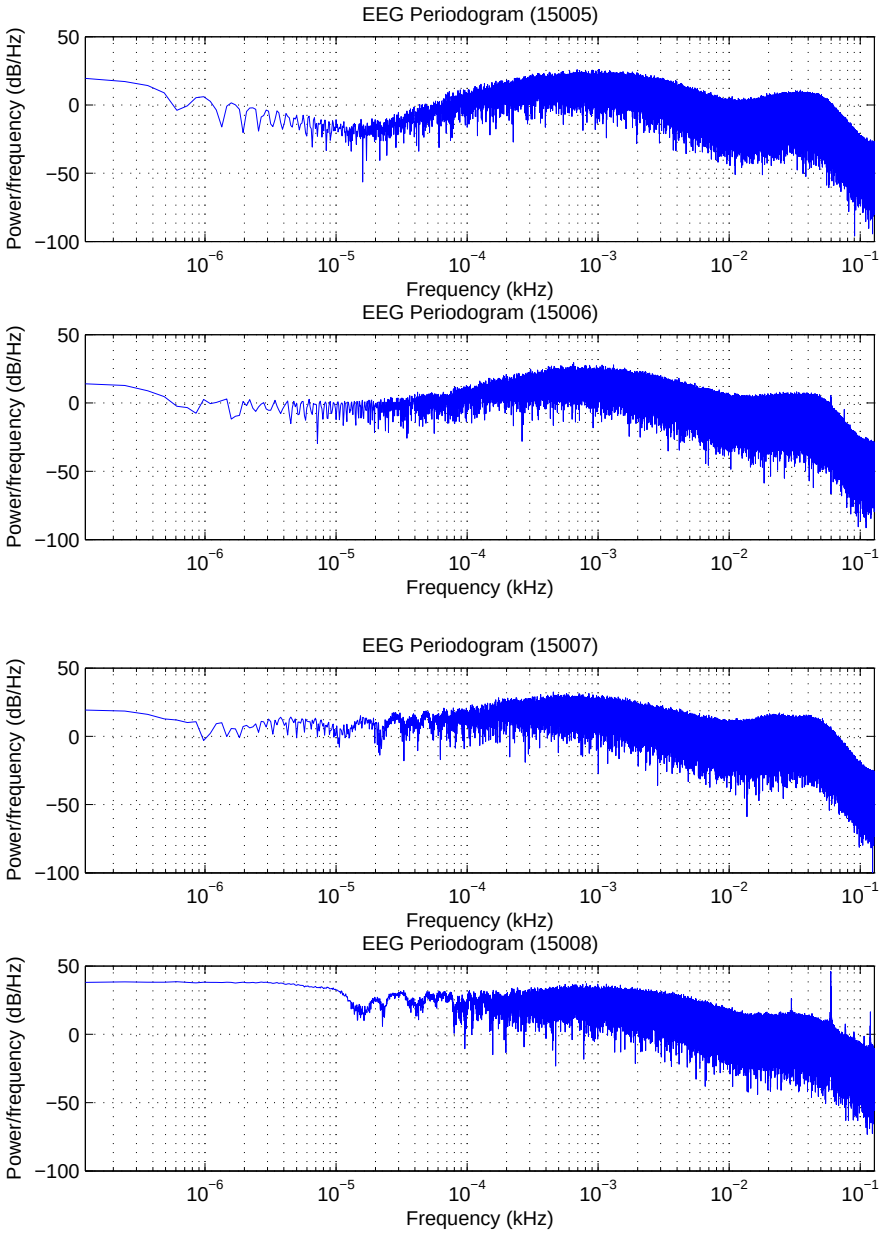

Figure 4-31: Power spectral density estimate via periodogram of EEG on log plot (15005–8). Note the peak near 40 Hz.

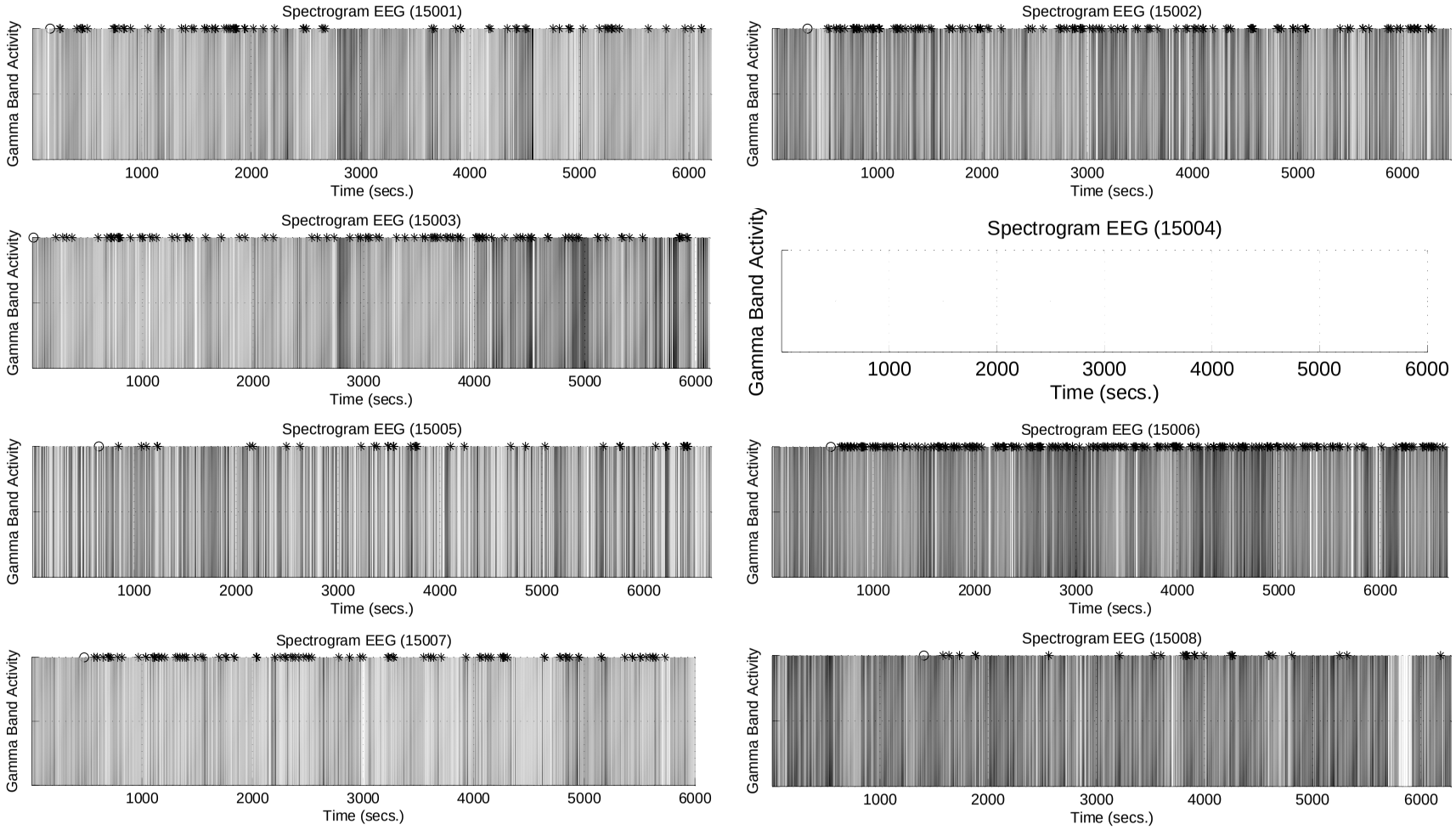

Figure 4-32: Spectrogram of EEG Gamma Band with marks for trades (*) and the open (o)

4.5 Physiological Data Feature Extraction

In order to extract meaningful features from the physiological data, it was necessary to address the problems with the data. The two broad categories of problems with the data were (1) missing data and (2) noisy data.

Of the 64 physiological signals collected (8 specialists with 8 signals each), 7 are missing. (Missing financial data is described earlier in this paper.) The finger temperature is missing in individuals 15002, 15004, 15006, and 15007. Individuals 15002, 15004, and 15006 were measured with the same recording instrument, though that cannot explain the missing data in 15007. The error was not noticed until it was too late to correct. Because half of the total finger temperature data is missing, I have not analyzed the finger temperature signal. In addition, the ECG is missing in individuals 15004 and 15008. (15008 is the lone female of the group.) Finally, the SCR is partially missing in individual 15003.

The other problem with the data set is noise. Given that the physiological data were collected while individuals performed their routine work, it should come as no surprise that the data are noisy. For instance, the EMG signal is full of spikes, which are abrupt changes on the order of 10 to 20 standard deviations. These spikes indicate a displacement of the sensor—not a meaningful emotional response. As another example, the temperature signal may oscillate by 0.01 °F, which is the resolution of the thermometer.

In order to improve the quality of the data, the SCR, ECG, BVP, and temperature features were computed using non-recursive (moving-average) filters. For instance, a 1-sec moving average applied to the temperature signal eliminated the aforementioned oscillations. Other non-recursive filters were also applied to remove noise.

After preprocessing the physiological signals, features can be extracted. The features, selected because they reflect the effect of the ANS on physiology (as described in Chapter 3), are:

1. time of onset of SCR response
2. time of peak of SCR response
3. amplitude of SCR response
4. instantaneous heart rate (HR)
5. instantaneous heart rate (BVP)
6. amplitude of BVP

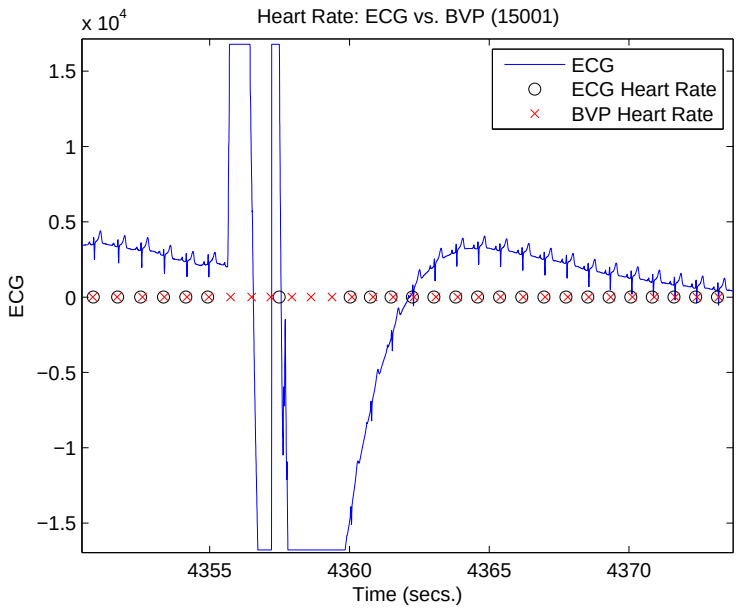

Figure 4-33: ECG vs. BVP (15001)

7. volatility of BVP
8. amplitude of EMG-Arm
9. time of EMG-Arm zero-crossing
10. amplitude of EMG-Back
11. time of EMG-Back zero-crossing
12. temperature change (from the 10-sec lag)
13. amplitude of temperature

Two of the physiological signals measure the heart rate—ECG and BVP. While both signals are noisy, the BVP exhibits fewer recording problems than the ECG. Figure 4-33 shows why the BVP-based estimate of the heart rate is more accurate than the ECG-based estimate. The figure marks the heart beats as calculated from the ECG and the BVP, superimposed on the raw ECG. The two heart rates are almost aligned, other than the five seconds when the ECG recording is missing. The BVP-based and ECG-based HR align within 0.1 sec. more than 90% of the time. That figure of merit make actually be higher since it does not accurately account for all missing ECG data. Rather than discard the ECG entirely, I included the ECG where possible.

For each market event of each individual, I constructed physiological feature vectors.

Since the market events were interval-based, I aggregated the feature vectors listed in the paragraph above over the duration of the event window (either 10 sec. or 5 min.):

1. number of SCR responses
2. average SCR amplitude
3. average HR of ECG
4. average ratio of the BVP amplitude to local baseline
5. average ratio of the BVP amplitude to global baseline
6. number of head temperature changes exceeding 0.1 °F
7. average duration (or rise time) of SCR
8. area from onset to peak of SCR
9. average change (or slope) of SCR
10. heart rate variability of ECG
11. heart rate acceleration (based on root-mean-square) of ECG
12. heart rate acceleration (based on standard deviation) of ECG
13. range of head temperature
14. average amplitude of EMG-Arm
15. number of zero-crossings of EMG-Arm
16. average amplitude of EMG-Back
17. number of zero-crossings of EMG-Back
18. average HR of BVP
19. heart rate variability (based on standard deviation) of BVP
20. heart rate variability (based on range) of BVP
21. heart rate acceleration (based on root-mean-square) of BVP
22. heart rate acceleration (based on standard deviation) of BVP
23. heart rate volatility of BVP

where the local baseline for the BVP signal is the average level during the event window, and the global baseline is the average over the entire recording session for each trader.

Table 4.7 shows the feature vector of the entire experiment for each individual.

Figures 4-34 to 4-36 show distributions of each feature for 15001.

	Totals							
Physiological Features	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*
# of SCR responses	249	706	-	1200	1360	1210	1960	130
Ave. SCR amplitude	0.33	0.77	-	1.01	0.74	0.78	1.13	0.28
ECG HR	81.75	72.66	73.11	-	98.12	93.56	88.27	-
Local BVP ratio	0.77	0.65	2.29	0.56	1.16	0.46	1.81	0.38
Global BVP ratio	0.77	0.65	2.29	0.56	1.16	0.46	1.81	0.38
# of Temp jumps	0	390	210	540	230	580	50	70
Ave. SCR duration	2.01	1.60	-	0.96	1.17	0.97	0.82	1.79
SCR area	90	600	-	510	770	480	950	40
Ave. SCR Δ	0	0	-	0	0	0	0	0
ECG HRV (Std)	21.28	8.71	43.89	-	30.80	12.43	18.06	-
ECG HR Accel. (RMS)	84.5	73.2	85.3	-	102.8	94.4	90.1	-
ECG HR Accel. (Std)	27.18	7.25	55.07	-	34.75	12.17	20.16	-
Range of Temp	0.8	7.1	2.5	6.1	1.3	6.5	0.9	1.7
Ave. EMG-Arm amplitude	15.6	4.3	22.1	6.0	7.1	2.4	17.3	2.3
EMG-Arm zero-crossings	5350	334	8145	1110	1610	66	2510	516
Ave. EMG-Back amplitude	20.2	2.4	20.5	1.2	7.3	1.6	39.2	15.1
EMG-Back zero-crossings	2040	210	3680	220	180	130	5370	0
BVP HR	80	72	95	75	91	88	85	71
BVP HRV (Std)	9.42	9.81	7.32	17.46	8.44	17.85	13.52	11.96
BVP HRV (Range)	130	100	130	140	110	130	140	122
BVP HR Accel. (RMS)	81.0	72.3	95.5	76.5	91.6	89.3	85.8	72.5
BVP HR Accel. (Std)	10.1	11.0	7.9	23.3	8.1	20.8	18.3	13.9
BVP Volatility	3.09	4.01	10.28	2.69	4.68	2.37	6.68	1.48

Table 4.7: Statistics for physiological features over entire duration.

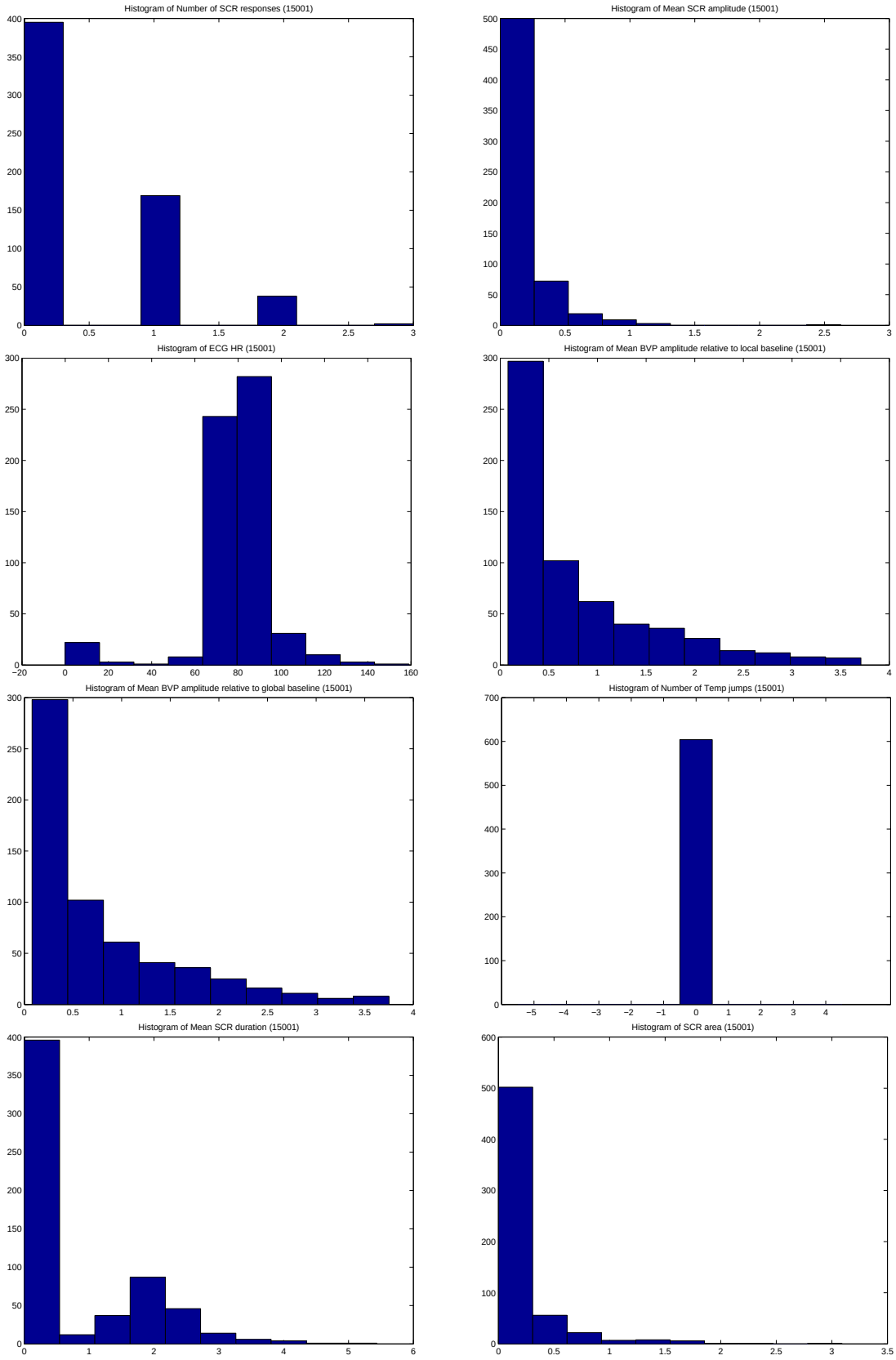

Figure 4-34: Histogram of physiological features, part 1 (15001)

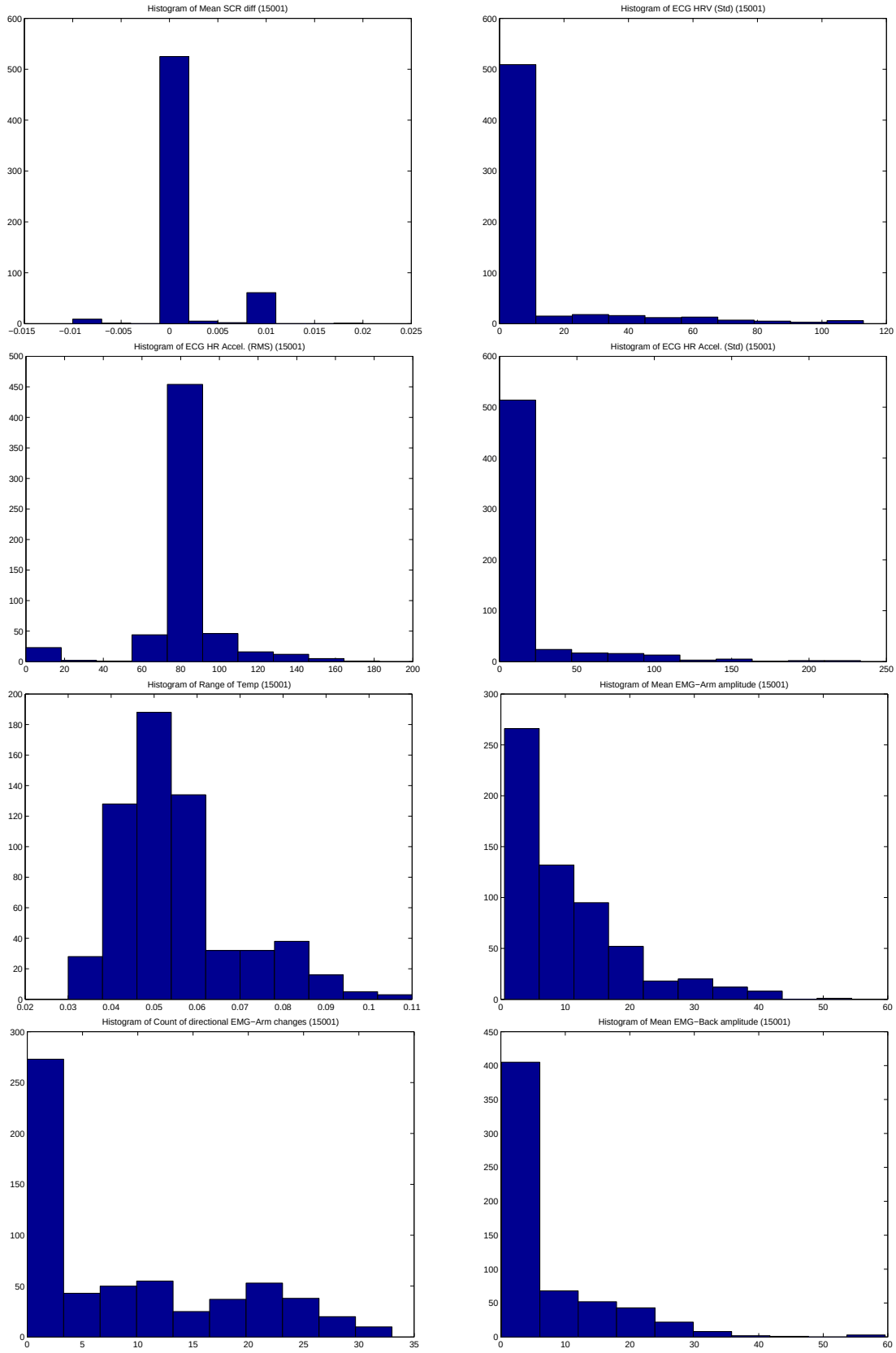

Figure 4-35: Histogram of physiological features, part 2 (15001)

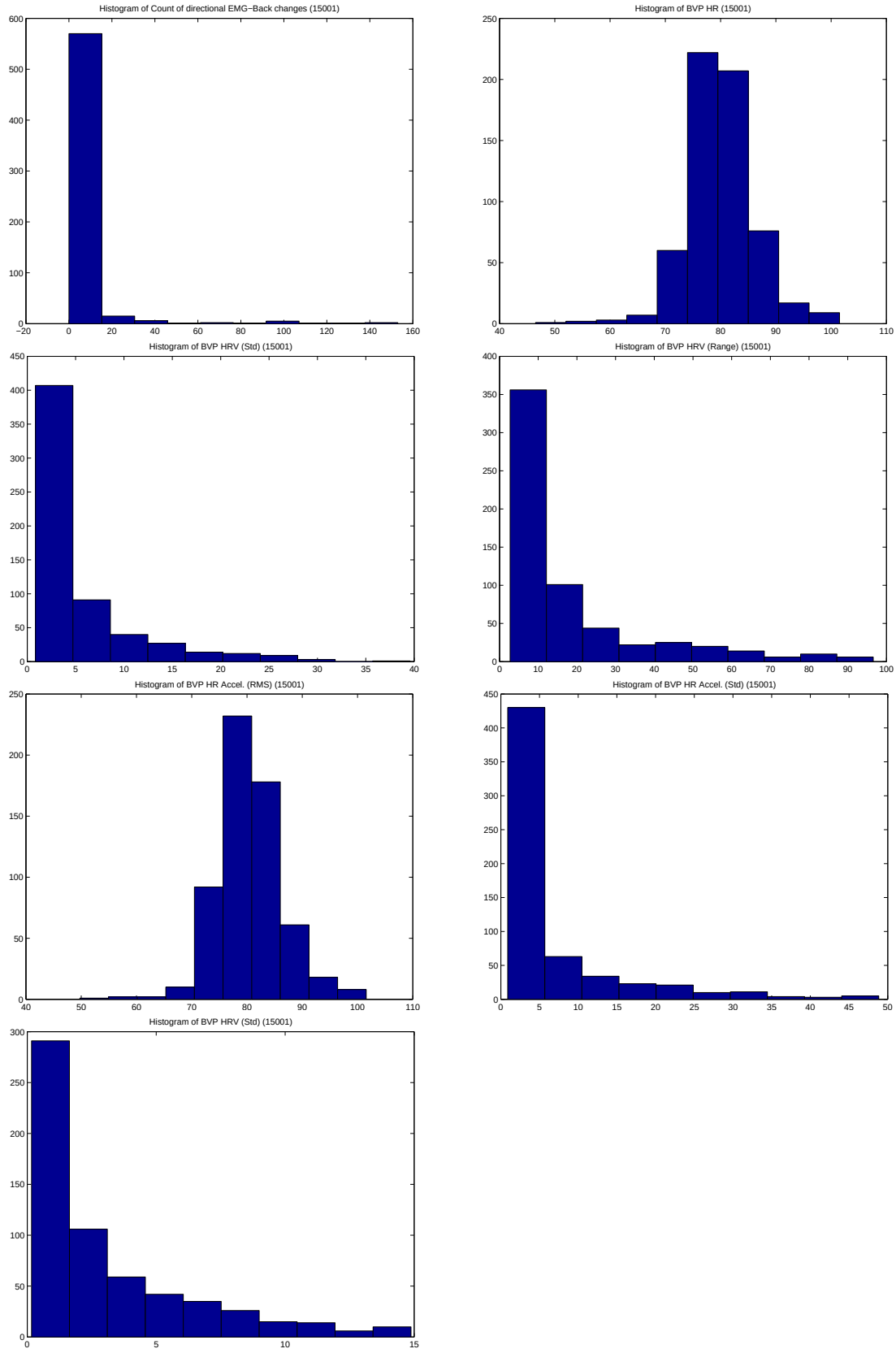

Figure 4-36: Histogram of physiological features, part 3 (15001)

Chapter 5

Results

This chapter describes my analysis and the results. The null hypothesis of the analyses is that individuals should demonstrate uniform emotional responses regardless of events. This chapter tests intervals before and after market events (which are described in Section 4.3), before and after decision events (trades), before and after certain alerts, and before and after significant P&L events. The chapter closes with a model of affect.

5.1 Market Events

For deviation and trend-reversal market events, I constructed pre- and post-event feature vectors, where the pre-event was the 10-sec interval prior to the event and the post-event was the 10-sec interval following the event.

Control feature vectors were constructed by applying the same feature-extraction process to randomly selected windows containing no market events of any kind. One set of control feature vectors was constructed for deviation and trend-reversal events (10-sec intervals), and one set was constructed for volatility events (5-min intervals). The control feature vectors for the deviation and trend-reversal events were the same. The pre- and post-event feature vectors were not compared head-to-head, but against the control feature vector.

The motivation for the post-event feature vector was to capture the individual's reaction to the event, with the pre-event feature vector as a benchmark from which to measure the magnitude of the reaction. A comparison between the pre-event feature vector and a control feature vector may provide an indication of a specialist's anticipation of the event. Latencies of the autonomic responses reported in previous studies were on a time scale of

1 to 10 seconds (Cacioppo et al., 2000). Hence, 10-sec event windows were judged to be long enough for event-related autonomic responses to occur and, at the same time, short enough to minimize the likelihood of overlaps with other events or anomalies. Five-min intervals were used for volatility events because 10-sec intervals were simply insufficient for meaningful volatility calculations. For all of the financial time series used in this study, and for most financial time series in general, there are few volatility events that occur in any 10-sec interval, except, of course, under extreme conditions, such as the stock market crash of October 19, 1987. No such conditions prevailed during any of the sessions.

The statistical analysis of the physiological and market data was motivated by several objectives: (1) to identify particular classes of events with statistically significant differences in autonomic responses before or after an event, as compared to the no-event control, (2) to identify particular physiological variables that demonstrate significant mean response levels immediately following the event or during the event-anticipation period, as compared to the no-event control, (3) to determine whether there are differences in autonomic responses before vs. after an event, (4) to check whether the positive results are random, and (5) to investigate the effect of the 10 second interval length.

Market Events To address the first objective, a two-sided t test was performed for the pooled sample of each component of each feature vector and the corresponding control vector to test the null hypothesis that the feature vectors were statistically indistinguishable from the control feature vectors. Actually, two separate t tests were performed. One test compared the 10-sec intervals preceding each market event to 10-sec intervals which contained no events. The second test compared the 10-sec intervals following each market event to the same no-event control.

The t statistics corresponding to the two-sided hypothesis test in Table 5.1 are highlighted if significant at the 5% level. I felt that the 5% level provided sufficient evidence. I felt that the Bonferroni correction was overly conservative for this research since the tests were not independent and since I am looking for yet-to-be-discovered associations. The t statistics are based on the t distribution with the appropriate degrees of freedom for each entry. The left panel, labeled "Pre-event Interval," lists the t statistics when comparing the features as calculated prior to the event versus the no-event period features. Analogously, the right panel, labeled "Post-event Interval," lists the t statistics of the features as

	Market Events											
	Pre-event Interval						Post-event Interval					
Physiological Features	*DEV Price*	*DEV Spread*	*DEV Return*	*TRV Price*	*TRV Spread*	*VOL Max*	*DEV Price*	*DEV Spread*	*DEV Return*	*TRV Price*	*TRV Spread*	*VOL Max*
# of SCR responses	0.33	1.18	0.21	-0.11	0.07	-0.47	0.07	0.17	1.60	-0.61	-0.45	-0.31
Ave. SCR amplitude	**-2.00**	1.40	0.82	0.88	0.62	-1.74	1.96	0.61	1.52	1.08	1.41	-1.27
ECG HR	-1.25	-0.29	-0.05	0.07	0.31	-0.03	-1.25	-0.64	0.03	-0.00	0.45	0.08
Local BVP ratio	1.91	1.77	1.63	**-3.01**	0.59	0.08	**-2.02**	1.60	1.52	**-3.14**	0.60	-0.01
Global BVP ratio	1.93	1.79	1.65	**-3.06**	0.57	0.08	**-2.06**	1.63	1.53	**-3.20**	0.58	-0.02
# of Temp jumps	-1.70	0	0	1.12	0.11	0.06	-0.91	-0.89	-0.22	1.12	0.68	-0.45
Ave. SCR duration	0.25	-0.72	-1.35	-1.17	-0.23	0.26	0.00	-0.99	-1.17	-1.51	0.47	0.15
SCR area	1.91	1.06	0.82	1.51	0.55	-0.93	1.74	0.49	**-1.98**	1.03	0.05	-0.52
Ave. SCR Δ	-1.49	-1.43	-0.04	1.07	1.01	0.30	-1.44	**1.98**	-1.00	1.67	-0.41	0.59
ECG HRV (Std)	0.49	1.48	1.77	0.79	1.34	-0.74	0.42	0.61	1.33	0.35	1.61	-0.19
ECG HR Accel. (RMS)	-1.14	-0.15	0.11	0.14	0.43	-0.14	-1.15	-0.56	0.14	0.05	0.62	0.02
ECG HR Accel. (Std)	0.54	0.75	**-2.66**	0.98	0.80	-0.90	0.55	0.39	1.05	0.47	1.18	-0.22
Range of Temp	-1.02	1.17	0.74	1.93	0.84	-0.62	-0.75	1.21	0.42	1.81	0.99	-0.19
Ave. EMG-Arm amplitude	-1.54	-1.07	0.67	-0.21	0.92	0.02	-1.35	-1.09	0.29	-0.66	0.88	0.47
EMG-Arm zero-crossings	-1.96	-0.99	-0.10	-0.12	0.08	-0.27	-1.86	-1.03	-0.06	-0.49	-0.01	0.05
Ave. EMG-Back amplitude	**2.87**	0.12	1.07	-0.57	0.32	0.31	**2.78**	0.91	1.59	-0.53	-0.46	0.11
EMG-Back zero-crossings	**3.13**	-0.93	-1.35	1.72	1.58	0.68	**3.11**	-0.74	-0.94	1.87	1.52	1.24
BVP HR	**2.19**	-1.54	-1.76	**2.82**	0.55	0.16	**2.42**	-1.47	-1.24	**2.55**	0.11	0.22
BVP HRV (Std)	0.56	**-2.18**	**-2.15**	1.28	1.56	-0.56	0.54	0.77	1.58	0.97	0.77	-0.84
BVP HRV (Range)	1.03	**-1.99**	1.89	1.05	1.67	-0.60	0.94	0.52	1.71	0.76	0.87	-1.21
BVP HR Accel. (RMS)	**2.25**	-1.38	-1.54	**2.76**	0.76	0.12	**2.49**	-1.47	-1.11	**2.55**	0.24	0.17
BVP HR Accel. (Std)	0.85	1.77	1.71	0.99	1.35	-0.61	1.06	0.68	1.66	0.56	0.76	-0.71
BVP Volatility	1.63	1.82	1.45	**-3.08**	0.44	0.12	1.75	1.64	1.41	**-3.19**	0.46	0.02

Bolded statistics are significant at the 5% level.

The left panel contains t statistics for pre-event features and the right panel contains t statistics for post-event features, both tested against the same set of control features. See Table 3.1 for abbreviations.

Table 5.1: t Statistics for two-sided hypothesis tests of the difference in mean autonomic responses during pre- and post-event intervals versus no-event intervals (controls) for each of the components of the physiological features (rows) for market events.

calculated following the event versus the no-event period features.

Based on the t statistics in the table, there are two event types that show significant differences in autonomic response: Price deviations and price trend-reversals. In both cases, the number of SCR responses and the average heart rate are statistically significant. For those same two events, the two BVP amplitude-related features are even more significant—at levels less than 0.1%. No other event types show any statistical significance. Both the pre-event interval and the post-event interval identify the same type of market events.

Physiological Variables To address the second objective, I use the same t tests as above. The null hypothesis of the t tests is that both pre- and post-event feature vectors are statistically indistinguishable from the control feature vector. As seen in Table 5.1, the two sets of t tests produce very similar results, implying that none of the physiological variables are predictors of anticipatory emotional response. Of course, the similarity may reflect the definition imposed on the problem—that an event occurs instantaneously. Instead, traders may react over the pre- and post-event window, as they follow the market. Future work may redefine the duration of an event.

Pre vs. Post Having tested the pre- and post-event feature vectors against a random, control vector, I now test the pre- and post-event feature vectors directly against each other. Table 5.2 shows the results of this head-to-head test. Few features and events are statistically significant, confirming the null hypothesis that there are few differences between anticipation and reaction. These results in combination with the results in Table 5.1 suggest that market events may belong to a long-running, overarching process.

Note that the head-to-head analysis does not control for overlapping events. For instance, if there is a TRV Price event during a DEV Spread event, the pre- and post- DEV Spread events could be statistically different as a result of the TRV Price event.

Random Events Since there appears to be statistically significant differences between features, I now test whether the results are random. The argument could be made that since there are about 20 features and 8 traders, 5% of the t statistics, or about one per trader, should be significant. Accordingly, I select 20 random times and label them as events. The corresponding feature vectors are tested against a control feature vector, which is also randomly selected, under the single constraint that the control vector contains no

	Market Events (Head to Head)					
Physiological Features	*DEV Price*	*DEV Spread*	*DEV Return*	*TRV Price*	*TRV Spread*	*VOL Max*
# of SCR responses	0.26	1.05	-1.46	0.49	0.51	-0.18
Ave. SCR amplitude	0.12	0.69	-1.50	-0.16	-0.68	-1.23
ECG HR	0.00	0.34	-0.08	0.07	-0.14	-0.11
Local BVP ratio	-0.13	0.16	0.10	-0.13	-0.02	0.09
Global BVP ratio	-0.14	0.15	0.10	-0.14	-0.02	0.09
# of Temp jumps	-0.79	0.89	0.22	0	-0.57	0.52
Ave. SCR duration	0.27	0.23	-0.09	0.32	-0.67	0.13
SCR area	0.23	0.46	-1.11	0.63	0.46	-0.42
Ave. SCR Δ	-0.05	1.15	0.53	-0.85	1.05	-0.25
ECG HRV (Std)	0.07	0.89	0.49	0.48	-0.19	-0.53
ECG HRV (Range)	0.05	1.02	0.96	0.85	-0.13	-0.89
ECG HR Accel. (RMS)	0.01	0.41	-0.04	0.09	-0.18	-0.16
ECG HR Accel. (Std)	-0.01	0.39	1.56	0.55	-0.29	-0.64
Range of Temp	-0.31	0.03	0.34	0.01	-0.18	-0.44
Ave. EMG-Armamplitude	-0.23	-0.01	0.37	0.49	0.05	-0.50
EMG-Armzero-crossings	-0.11	0.06	-0.04	0.38	0.09	-0.33
Ave. EMG-Back amplitude	-0.13	-0.75	-0.49	-0.04	0.73	0.20
EMG-Back zero-crossings	-0.05	-0.19	-0.48	-0.66	1.00	-1.00
BVP HR	0.19	-0.12	-0.55	-0.24	0.45	-0.06
BVP HRV (Std)	0.02	1.39	0.54	0.29	0.81	0.24
BVP HRV (Range)	0.08	1.46	0.17	0.27	0.79	0.64
BVP HR Accel. (RMS)	0.20	0.02	-0.48	-0.19	0.55	-0.05
BVP HR Accel. (Std)	-0.20	1.08	0.04	0.38	0.61	0.08
BVP Volatility	-0.13	0.16	0.03	-0.11	-0.02	0.09

Bolded statistics are significant at the 5% level.

The panel contains t statistics for pre-event features tested against post-event features. See Table 3.1 for abbreviations.

Table 5.2: t Statistics for two-sided hypothesis tests of the difference in mean autonomic responses during pre- and post-event intervals for each of the components of the physiological features (rows) for market events.

random events.

Table C.1 contains the t statistics. Two physiological features stand out: the temperature features of 15002 and the BVP features of 15005. The origin of this significance is unknown, prompting more careful scrutiny of drawing conclusions regarding these features for these particular traders. Otherwise, the lack of significant t statistics for each trader—half of the traders have *no* significant t statistics—should confirm that the results above are not mere coincidences.

Interval Length I conclude this section with a discussion of interval lengths. Throughout this paper, the length of the feature windows is 10 seconds unless explicitly stated. The 10-sec interval length was chosen because the slowest varying psychophysiological response lasts 10 seconds. While a psychophysiological response may occur within 10 seconds of an event, research is not conclusive addressing the duration of the response. Thus, the body may respond to an event within 10 seconds, but the body may remain stimulated for a duration that exceeds 10 seconds, which should be measurable.

Table 5.3 shows the results of varying the interval length from 10 to 100 seconds in 10-sec increments. As the interval increases, the total number of statistically significant variables for market events also increases. In contrast, for random events the number of significant variables decreases. Because the construction of the statistical tests requires that the control intervals contain no events, the interval length cannot increase beyond 100 seconds in a 6,000-sec (100-min) study. (If there are 20 events, a 100-sec interval would require 4000 seconds for the pre- and post- feature vectors, leaving the remaining 2000 seconds for the no-event control vector.) The tests are not pooled (as above), but are individual in order to generate eight times as much data.

Figure 5-1 shows how the number of significant variables increases with interval length. The counts come from Tables C.2 to C.11 which display t statistics for DEV Price for all 10 intervals. This data is summarized in column two of Table 5.3. I have omitted the figures and tables for the other columns in Table 5.3.

5.2 Trading

Up to this point, the data analysis parallels the analysis in (Lo and Repin, 2002). Having confirmed the findings from that study, I devote the remainder of this paper to new tests and

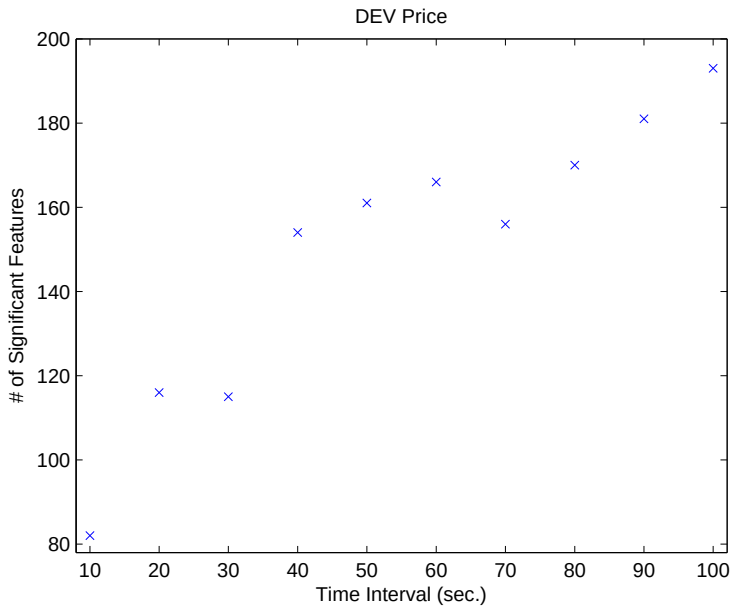

Figure 5-1: Number of significant variables vs. the interval length over which the variables are computed

new results. What sets this experiment apart from any previous study is the uniqueness of the data set. Physiological data by itself is interesting. Market data by itself is interesting. Trading activity and the specialist's book by itself is interesting (and perhaps the most difficult data to obtain). However, the conjunction of all three data sets is much more interesting than any of the data sets by itself—the whole is greater than the sum of the parts.

The analysis of the trading activity focuses on correlating the emotional response with trading activity. Generally speaking, trades represent decisions, the result of an individual acting on information garnered from the environment. The objectives are to identify: (1) whether trades generate statistically significant differences in autonomic responses before or after a trade, as compared to the no-trade control; (2) particular physiological variables that demonstrate significant mean response levels during the preceding or trailing interval following a trade as compared to the no-trade control interval, (3) whether buy or sell trades exhibit different emotional, or physiological, signatures, (4) whether winning or losing trades exhibit different responses, (5) whether large trades exhibit differences in responses, and (6) whether automatic trades generate differences in mean response levels.

Interval Sec.	*DEV Price*	*DEV Spread*	*DEV Return*	*TRV Price*	*TRV Spread*	*Random*
10	82	29	35	72	19	19
20	116	34	47	98	52	15
30	115	45	38	90	39	13
40	154	88	54	100	64	24
50	161	76	80	120	64	26
60	166	72	87	128	63	23
70	156	79	94	130	51	16
80	170	85	107	137	57	14
90	181	85	136	158	59	14
100	193	92	122	159	62	28

Table 5.3: # of significant physiological features for different intervals

Trades The first objective is to test whether there are differences in autonomic responses before or after a trade. The idea is to test whether there is a differences in the physiological signature of anticipating a decision and/or differences in the physiological signature for having made a decision. The implication, as explained in Chapter 2, is that the brain triggers these changes. To address the objective, a two-sided t test was performed for each component of each trader's feature vector. The feature vector was oriented around trade events. The null hypothesis was that the feature vectors would be statistically indistinguishable from the control feature vectors. Two separate t tests were performed. One test compared the 10-sec intervals preceding each trade to 10-sec intervals which contained no trades. The second test compared the 10-sec intervals following each trade to the same no-trade control.

The t statistics corresponding to the two-sided hypothesis test in Table 5.4 are highlighted if significant at the 5% level. The t statistics are based on the t distribution with the appropriate degrees of freedom for each entry. The left panel, labeled "Pre-event Interval," lists the t statistics that result from the comparison between the pre-trade period features to the no-trade period features. Analogously, the right panel, labeled "Post-event Interval," lists the t statistics of the post-trade period features when compared to the no-trade period features.

Physiological Variables To address the second objective, I identified the physiological features in Table 5.4 that were significant at the 5% level. Each of the traders shows different significant features in varying intensities. The number of SCR responses and the

	Trades															
	Pre-event Interval								Post-event Interval							
Physiological Features	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*
# of SCR responses	**2.57**	**-2.92**	0.58	0.52	-1.48	-0.54	0.68	-0.70	-1.00	**-4.88**	0.14	0.10	-0.71	1.11	-0.54	-0.53
Ave. SCR amplitude	**2.13**	-0.77	-1.22	-1.23	-0.52	**-2.57**	1.45	-1.26	-0.89	-0.10	-1.32	-0.88	-0.46	**-2.38**	0.05	-1.25
ECG HR	0.19	1.02	0.56	-	0.66	0.91	0.14	-	0.81	**-2.28**	0.18	-	**-2.20**	1.13	0.03	-
Local BVP ratio	**-3.59**	-0.31	-0.76	**-2.99**	1.38	**-2.56**	**-3.90**	-0.77	**-4.10**	-0.22	-0.21	**-2.64**	**-2.05**	**-3.57**	**-2.42**	-0.42
Global BVP ratio	**-3.59**	-0.32	-0.73	**-2.98**	1.46	**-2.57**	**-3.98**	-0.78	**-4.09**	-0.23	-0.16	**-2.62**	**-2.02**	**-3.55**	**-2.42**	-0.42
# of Temp jumps	-	**-3.99**	0.15	0.37	0.25	0.90	0.40	0.35	-	**-3.81**	0.15	0.89	-0.24	1.64	**-2.07**	0
Ave. SCR duration	**2.91**	-1.10	-0.11	0.75	0.33	0.79	**2.30**	-1.54	-1.40	-0.05	-0.58	**-2.49**	1.23	0.34	**2.41**	-1.73
SCR area	-1.62	-0.40	-0.72	-0.83	-0.67	0.65	0.64	-1.21	-0.73	0.19	-1.09	-0.12	1.12	1.29	0.76	-1.50
Ave. SCR Δ	-1.12	1.82	-	1.53	1.66	**2.37**	0.88	-1.39	-1.26	1.82	-	0.88	-0.35	**3.09**	0.39	-1.38
ECG HRV (Std)	0.59	0.43	-0.12	-	-0.07	-0.97	**-1.99**	-	**-2.04**	1.28	-0.34	-	**-2.48**	0.86	1.76	-
ECG HR Accel. (RMS)	0.29	1.14	0.38	-	0.50	0.48	0.70	-	1.13	**-2.41**	-0.01	-	**-2.31**	1.11	0.49	-
ECG HR Accel. (Std)	0.80	0.56	-1.69	-	-0.42	-0.85	**-2.59**	-	**-2.15**	1.45	-1.04	-	**-2.31**	0.93	1.58	-
Range of Temp	1.61	**-2.38**	0.18	0.03	1.37	0.61	0.72	-1.83	**-2.00**	**-2.47**	-0.18	0.35	0.34	1.14	1.87	-1.94
Ave. EMG-Arm amplitude	**5.01**	-0.20	-1.61	**2.10**	-0.30	-1.68	**-2.13**	**3.67**	**3.15**	-0.20	-1.11	**2.82**	1.32	-1.45	**-1.99**	-1.72
EMG-Arm zero-crossings	**5.53**	**-5.63**	**2.90**	-0.80	0.34	1.28	**-2.52**	**3.13**	**4.04**	**-5.03**	**2.66**	-1.01	**-2.19**	-0.27	1.51	-1.28
Ave. EMG-Back amplitude	-1.05	**-1.99**	**2.73**	**-2.02**	-0.18	0.52	1.21	-0.77	-0.37	**-2.11**	**3.01**	**-2.10**	0.48	1.94	-0.39	0.63
EMG-Back zero-crossings	-0.69	0.46	1.93	-0.49	1.00	-1.71	-1.64	-	-1.15	0.02	1.82	0.61	1.00	-1.09	-1.47	-
BVP HR	**1.97**	0.19	0.35	0.35	-0.23	-0.70	**3.75**	-0.15	-1.69	1.47	0.73	0.38	-0.00	**2.38**	**2.14**	-0.99
BVP HRV (Std)	**2.80**	**-2.88**	1.72	0.88	0.26	0.61	**-2.27**	-0.24	0.39	**-3.38**	1.78	-0.33	1.96	1.73	1.90	0.26
BVP HRV (Range)	**3.21**	**-2.96**	1.60	0.57	0.11	0.25	**-2.15**	-0.65	0.11	**-3.63**	1.65	-0.48	**-2.07**	0.86	1.69	-0.15
BVP HR Accel. (RMS)	**2.28**	0.61	0.46	0.50	-0.21	-0.60	**3.43**	-0.09	-1.65	1.92	0.76	0.24	0.06	**2.23**	-1.79	-0.85
BVP HR Accel. (Std)	**2.91**	**-3.54**	1.44	0.64	-0.25	0.75	**-2.04**	-0.87	-0.28	**-3.91**	0.75	-0.46	1.89	1.87	1.89	0.27
BVP Volatility	**-3.59**	-0.66	-1.01	**-3.94**	1.45	**-2.49**	**-3.24**	-0.18	**-4.07**	-0.39	-0.43	**-3.70**	**-2.00**	**-3.46**	**-1.98**	-0.32

Bolded statistics are significant at the 5% level.

The left panel contains t statistics for pre-event features and the right panel contains t statistics for post-event features, both tested against the same set of control features. See Table 3.1 for abbreviations.

Table 5.4: t Statistics for two-sided hypothesis tests of the difference in mean autonomic responses during pre- and post-event intervals versus no-event intervals (controls) for each of the components of the physiological features (rows) for trades.

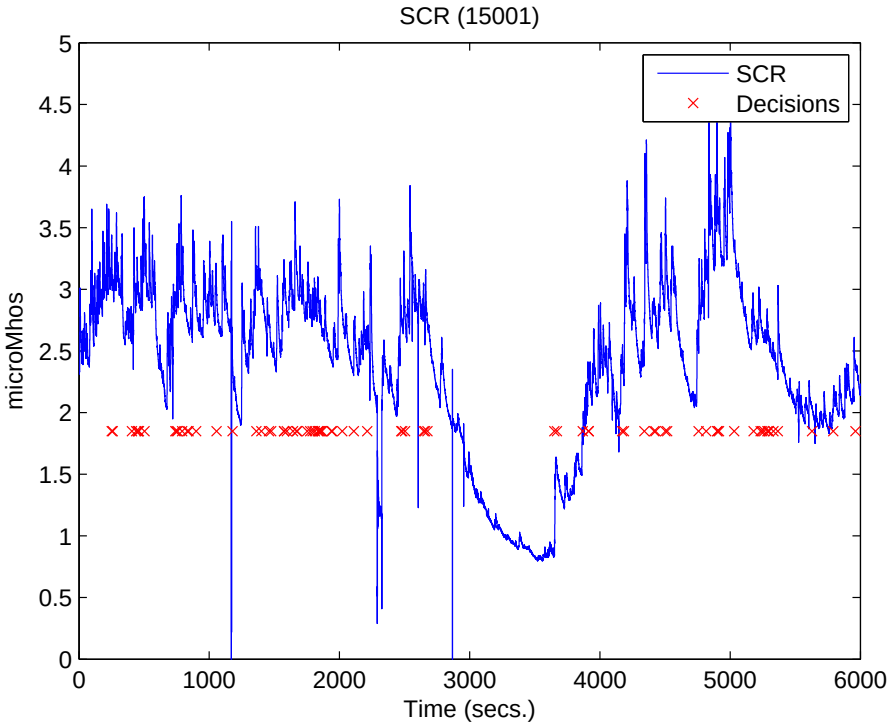

Figure 5-2: SCR and decisions (15001)

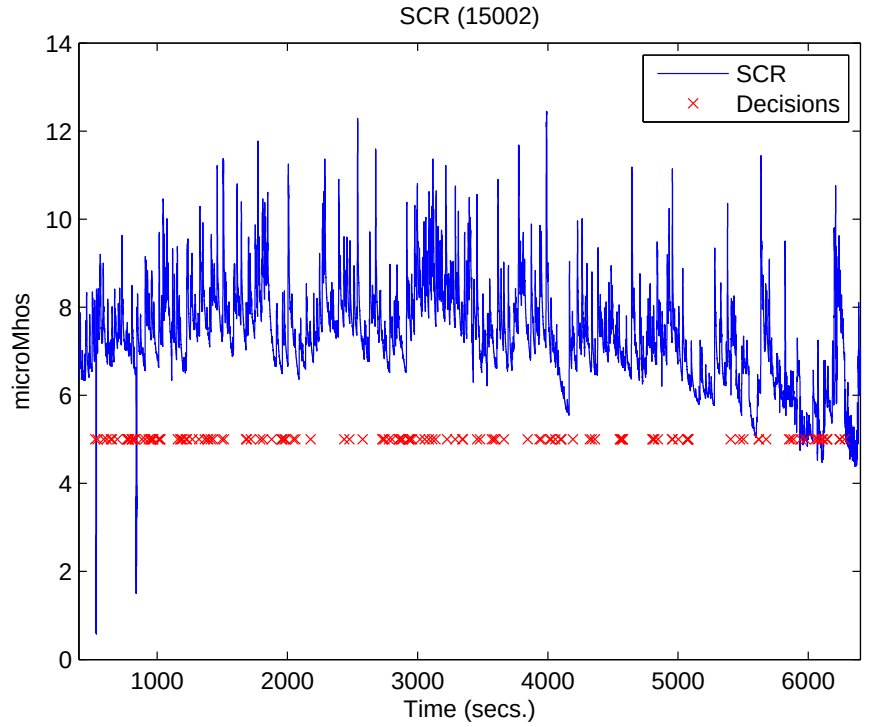

Figure 5-3: SCR and decisions (15002)

average heart rate are statistically significant. Figures 5-2 and 5-3 show the SCR and trading decisions for two specialists. It is possible to visually see the differences in the SCR while trading vs. not trading in 15001, but not in 15002. See Appendix A for a laboratory-based experiment that shows a similar result to Figure 5-2. The two BVP amplitude-related features were even more significant—at levels less than 0.1%. No other event types or features show any statistical significance. Amazingly, both the pre-event interval and the post-event interval identify the same physiological features as being significant, which implies that the trades may belong to a long-term process.

The results in Figure 5-2 confirm the results from 15001's spectrogram in Figure 4-32. A careful glance reveals that the EEG changes (darkens) around 3000 and 4500 sec. These moments align with drops in SCR magnitude. The instant 15001 begins trading again around 3800 sec. is the instant that the EEG transitions—see the change in color tone in the EEG spectral composition. It seems likely that 15001 mentally checked out and backed off from the markets during this period.

In a later section, I bifurcate the trades in Table 5.4 into Buy trades and Sell trades. In order to draw comparisons between Buy and Sell trades, I now report separate statistical tests for Buy trades and Sell trades. 15001 and 15007 show significant differences of means in BVP features when buying as compared to non-buying intervals. 15002, 15005, and 15008 show significant differences in means of cardiovascular and SCR features following a buy trade as compared to non-buying intervals. 15004 experiences differences of means in BVP features when selling as compared to non-selling intervals. 15005 and 15008 experience differences of means in muscle tension prior to selling but not after, which either may signal true tension or may reflect the muscle activity in the other, mouse-wielding arm. Overall, it appears that the BVP provides the most information of all the physiological signals. The detailed results appear in Tables C.12 and C.13.

Buy vs. Sell Trades To address the third objective, I performed a two-sided t test that compared each component of each trader's feature vector for buy trades against sell trades. The construction of the test parallels the construction of the previous tests: the null hypothesis is that the feature vectors of a buy trade are statistically indistinguishable from the feature vectors of a sell trade. Table C.14 compares the 10-sec intervals following a buy trade to 10-sec interval following a sell trade.

Winning vs. Losing Trades The fourth objective tested for differences in winning or losing trades relative to non-trading event intervals. It is one thing to put on a losing trade; it is quite another to put on a winning trade. A definition of a winning or losing trade depends on the lifetime of the trade, making it somewhat of a moving target. The easy case is when a specialist opens and some time later closes the position. However, this case rarely occurs. More often what happens is that the specialist goes long and then piles on to that position. One possible way around this problem is to argue that back-to-back buys are equivalent to selling the outstanding shares and purchasing the sum total. However, I question whether that is what takes places psychologically when adding to positions. So, instead, I have chosen to classify trades by their status after two arbitrary intervals: instant and 10 seconds.

Winning trades are trades that make money. For example, if a specialist sells short at \$81.14 and the best offer one tick later is \$81.13, then the trade is an instant winning trade. Losing trades are trades that lose more money than the bid-ask spread. For example, if a specialist buys at \$81.13 when the best bid is \$81.11 and the best bid 10 seconds later is \$81.10, then the trade is a 10-second losing trade. However, if the best bid 10 seconds later had been \$81.12, then the trade would be neither a winning nor a losing trade.

I performed two-sided t tests that compared each component of each trader's feature vector against a control, no-event vector for each of the three intervals. Tables C.15 to C.18 show the results of the winning and losing trade analysis.

Table C.15 shows the results of instantaneous winning trades. Table C.16 shows the results of instantaneous losing trades. 15007 is the only trader to exhibit differences during both instant winning and losing trades. 15002, in contrast, only exhibits differences prior to instant winning trades. One possible explanation for this behavior stems from how a market-maker operates. A specialist may sometimes find that a trade that can be flipped for a quick profit. Not every specialist is so lucky, as 15005 and 15008 can testify since neither of them had a free lunch.

Table C.17 shows the test results of 10-second winning trades. Table C.18 shows the test results of 10-second losing trades. 15001 and 15006 exhibit strong differences for trades that went net positive 10 seconds after the trade was made. Not only was the BVP statistically significant, but also the EMG-Arm. This result seems to suggest that the EMG-Arm is experiencing joyous tension or, perhaps, is wildly pumping the non-dominant fist. There

was relatively little activity for a 10-sec losing trade.

Large Trades Stratifying trades according to the dollar size of the trade is another way to classify trades besides classifying trades according to whether they won or lost money. Table C.19 shows results of testing for differences in means of physiological features for the 20 largest trades of each trader. Other than 15007, who appears to exhibit anticipation, no other specialists seem to be moved by large trades. While not shown, the histogram of the size of the trades shadows the amount of capital employed by each specialist (see Table 4.1). Thus, 15008 trades much smaller sizes than 15004.

Automatic Trades The sixth objective concerns not manual trades but automatic trades. If trading, be it manual or auto, generates differences in mean autonomic responses, one possible conclusion is that the physiological response captures outcomes–not necessarily decisions.

Table C.20 and C.21 contain the results from the tests. Statistically significant differences in means prior to an automatic trade should be viewed with caution. For instance, 15002 exhibits difference in cardiovascular and skin conductance response variables prior to both auto buy and auto sell trades. However, 15002 does not exhibit differences after the auto buy trades. Other specialists exhibit differences as well, with much more activity preceding and following auto sell trades.

Unfortunately, the trading environment is sufficiently complex that multiple events can happen at the same time. Since auto trades are so intertwined with manual trades and are more numerous, it is hard to separate the one from the other. Consequently, I imagine that some of the significance owes itself to overlaps with manual trades.

Other The above analysis show that there are different signatures of winning and of losing trades. However, the analysis says nothing about whether there are differences between the winning and losing trades themselves. One way to test for such differences is to compare the physiological features following winning trades to physiological features following losing trades. To define winning and losing trades requires a time interval. Subsequently, Tables C.22 and C.23contain results from two-sided t tests that compare instantaneous and 10-sec winning and losing trades, respectively. There are almost no differences for instant trades. However, there are some differences as the time interval increases.

5.3 Alerts

The volume of information processed by the specialists can, at times, be overwhelming. Table 4.1 lists a metric that attempts to capture the volume of information presented to a specialist. The metric is called *# Screen Updates*. Alerts belong to this category.

There are four different kinds of alerts: trade alerts, quote alerts, stop trade alerts, and stop quote alerts. A trade alert indicates that a trade on another market has taken place at the same price as the specialist's best order (highest bid or lowest ask price). A quote alert indicates that a quote on another market has taken place at the same price as the specialist's best order. A stop trade alert indicates that a trade on another market has taken place at the same price as the specialist's stop order. A stop quote alert indicates that a quote on another market has taken place at the same price as the specialist's stop order. I chose to analyze the trade alerts and quote alerts, but not the two stop alerts because the sample size was too small.

The objective of analyzing the alerts is to determine whether the traders exhibit statistically different emotional responses after the alerts, though I also include t statistics for emotional responses before the alerts as well. (The analysis parallels the analysis for both market events and trades.) Post alert statistics may provide information regarding risk processing. If the trader realizes that another exchange completed a trade, the physiological response may capture the trader's reaction.

To test the hypotheses, I identified all of the alerts. I computed feature vectors corresponding to each alert as well as a control vector from an identical number of randomly chosen, non-overlapping intervals. A two-sided t test was performed for each component of each feature vector and the corresponding control vector. The null hypothesis was that the feature vectors should be statistically indistinguishable from the control feature vectors. Table C.24 compares the 10-sec intervals preceding and following a trade alert to 10-sec intervals which contain no such events. Table C.25 compares the 10-sec intervals preceding and following a quote alert to 10-sec intervals which contain no such events.

Some of the t statistics in Table C.24 are large, such as -8.97. These extreme values are a product of sparse data. For instance, there are only two trade alerts for 15008; 15005 does not even have a trade event.

Even though there appear to be differences in emotional response both before and after

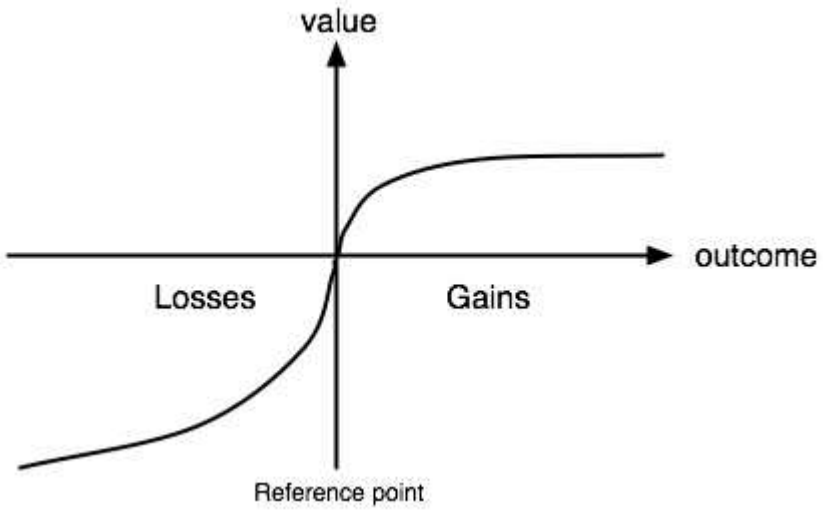

Figure 5-4: Prospect Theory value function.

the alerts, I hesitate to draw any conclusions. First, the data is sparse. Second, the alerts only appear if the trader is actively working an order. Thus, the alerts are correlated in some temporal dimension with trades. As was seen in Section 5.2, trading generates differences in means of physiological features. It may be that the alerts fall within the same window.

5.4 Profit and Loss

According to the most widely cited article in *Econometrica*, individuals evaluate gains and losses differently (Kahneman and Tversky, 1979). I perform statistical tests to evaluate the validity of this theory, called Prospect Theory. Prospect theory aims to explain anomalies predicted by expected utility theory. For instance, prospect theory offers an explanation of why individuals are more excited about earning their first million dollars than their tenth. It also attempts to explain why individuals are risk averse.

Given the same magnitude of gain or loss, prospect theory postulates that individuals feel the impact of the loss more than the impact of the gain. Figure 5-4 shows the *s*-shaped value function. The value function shows that small gains and losses generate disproportionate value, i.e., feelings of joy or pain. As the magnitude of the gain increases, the incremental enjoyment from the gain lessens (the curve flattens). As the magnitude of the loss increases, the incremental pain from the loss also lessens, but at a much slower rate (the curve flattens out more slowly). The magnitude of large gains is smaller in absolute

value than the magnitude of large losses—indicating that individuals feel loss more than they feel gains.

I aim to test the validity of four aspects of prospect theory. First, I test the null hypothesis that there is no difference in emotional response during intervals of P&L volatility relative to intervals of no volatility. Second, I test for differences in emotional responses during intervals of gains and losses relative to intervals of no gains or losses. Third, I test whether losses are felt more than gains. Fourth, I test whether anticipating or reacting to gains and losses have similar emotional response signatures.

I will next explain the construction of the first hypothesis test. The second test parallels the construction of the first. The description and results of the third and fourth tests appear below.

To test the first hypothesis, I identified one-minute intervals with (1) low P&L volatility as measured by each of the securities monitored, (2) high P&L volatility as measured by each of the securities monitored, (3) low P&L volatility of the overall P&L, and (4) high P&L volatility of the overall P&L. As explained in Section 4.2, profit and loss typically refers to profit and loss aggregated over all securities and includes both realized and unrealized gains and losses. While this profit and loss is important, specialists often track a handful of securities. For this reason, I computed feature vectors from each of the securities monitored, choosing volatility events from across all securities. A control vector was computed from an identical number of randomly chosen, non-overlapping intervals. A two-sided t test was performed for each component of each feature vector and the corresponding control vector. The null hypothesis was that the feature vectors should be statistically indistinguishable from the control feature vectors.

Four separate tests were performed. Table C.26 compares the 10-sec intervals preceding and following a one-minute interval of low P&L volatility, where the volatility is measured by a single security, to an equivalent number of 10-sec intervals which contain no such events. Table C.27 compares the 10-sec intervals preceding and following a one-minute interval of high P&L volatility, where the volatility is measured by a single security, to an equivalent number of 10-sec intervals which contain no such events. See Figure 5-5 for an example of high volatility events. Table C.28 compares the 10-sec intervals preceding and following a one-minute interval of low overall P&L volatility to an equivalent number of 10-sec intervals which contain no such events. Table C.29 compares the 10-sec intervals

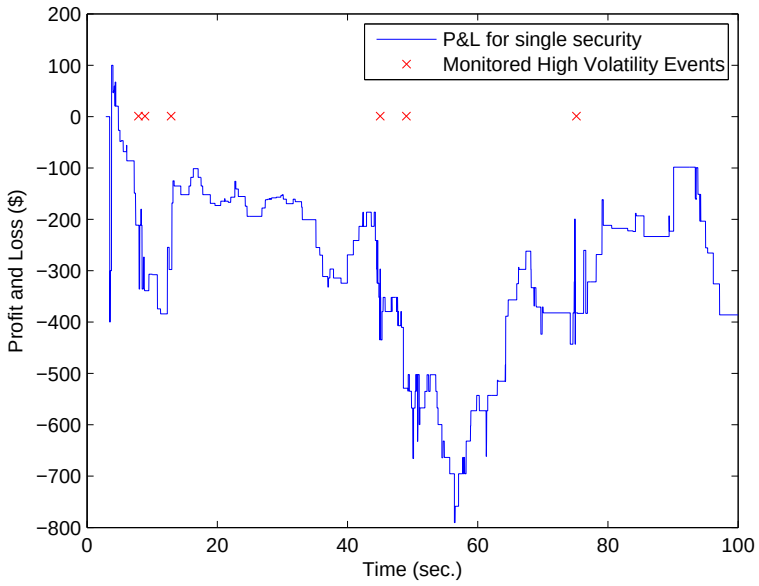

Figure 5-5: Profit and Loss (in $) of a single security. (High volatility events are marked when security is actively being monitored.)

preceding and following a one-minute interval of high overall P&L volatility to an equivalent number of 10-sec intervals which contain no such events.

Comparing and contrasting these tables, it appears that the significance of high volatility events of a single security P&L is not directly correlated with the significance for high volatility events of overall security P&L. For instance, 15006 has no significant physiological features for single security P&L events, but the BVP is significant for overall P&L events.

Table C.30 is the pooled version of Table C.29. None of the physiological variables are statistically significant.

The second set of tests closely parallels the first set of tests. However, in lieu of measuring periods of volatility, I measure periods of large profits and large losses. To calculate gains and losses requires both a time horizon and a gain or loss threshold (in either units of dollars or percent return). I chose an arbitrary time horizon of four minutes, meaning that the profit or loss had to fall above or below the threshold within four minutes. The choice of a threshold—how much rise in value is a gain event?—can be independent of other variables, say $100, or dependent on other variables, such as capital invested or total profit. I chose to use an independent value. I explain why. Since it does not seem fair to use an independent threshold for traders who can invest with different levels of capital, I look

at how a threshold customized for every trader might look. For example, an independent threshold could be a percentage of each trader's average capital. Figure 5-6 shows graphs of varying such a threshold from one basis point to 400 basis points of average capital. The number of profit and loss events exceeding the threshold monotonically decreases as a function of increasing threshold. These graphs show that a threshold equal to one basis point (0.01%) of the average capital is sufficiently small such that *all* of the traders have multiple profit-and-loss events. In cases where the average capital is negative, I treat the average capital value as positive. (Fortunately, the average capital is never zero as that would pose a problem.) However, one basis point of, say, $60,000 only amounts to $6—hardly an exciting or threatening gain or loss. Therefore, I select the top gains and losses exceeding a threshold of $100.

Using four minute time horizons and $100 thresholds, I identified (1) large gains in single security P&L, where the securities were actively monitored (2) large losses in single security P&L, where the securities were actively monitored (3) large gains in overall P&L, and (4) large losses in overall P&L.

A two-sided t test was performed for each component of each feature vector and a corresponding control vector, which control vector was computed from an identical number of randomly chosen, non-overlapping intervals. The null hypothesis was that the feature vectors should be indistinguishable from the control feature vectors. Four separate tests were performed.

Table C.31 compares features from 10-sec intervals preceding and following a large gain in single security P&L to an equivalent number of no-event, control features. 15007 exhibits significant differences in most of the BVP features. None of the other traders appear to feel the gain.

Table C.32 compares features from the 10-sec intervals preceding and following an interval of large loss in single security P&L to an equivalent number of no-event, control features. 15003 exhibits significant BVP differences. 15007 exhibits significant differences in the SCR features.

Table C.33 compares the features from 10-sec intervals preceding and following a large gain in the pooled, overall P&L to an equivalent number of no-event, control features. 15003 experiences differences in cardiovascular variables.

Table C.34 compares the features from 10-sec intervals preceding and following an in-

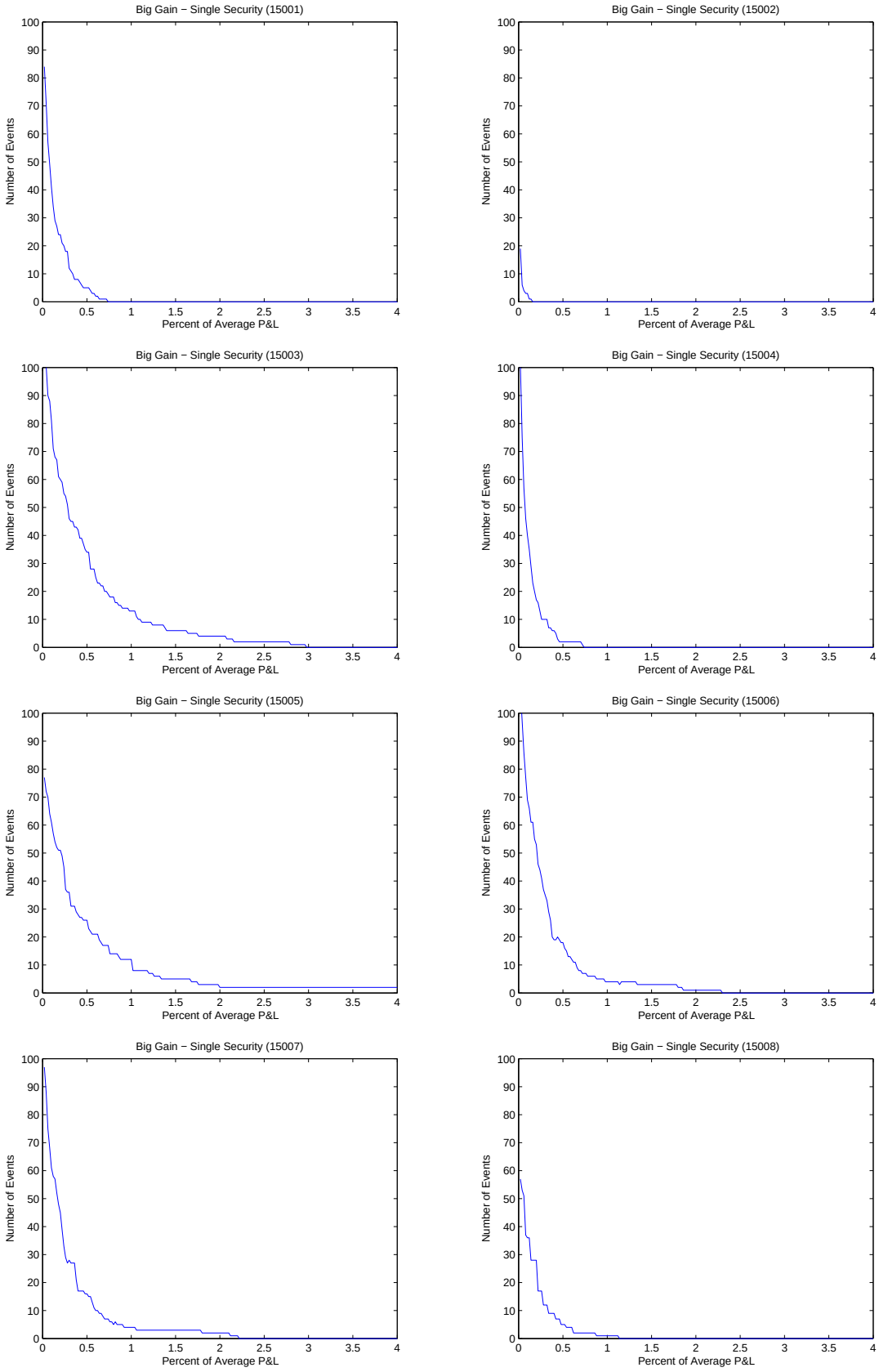

Figure 5-6: Number of events varies as threshold of profit and loss changes

terval of large loss in the pooled, overall P&L to an equivalent number of no-event, control features. None of the other traders appear to feel the pooled losses.

Figures 5-7 to 5-14 show overall profit and loss events corresponding to Tables C.33 and C.34. The figures show that the variation in the number of events varies from trader to trader. For example, 15005 experiences just one big loss and only three big gains, most of the gain coming from a single trade.

For the third of the four prospect theory tests, I test whether losses are felt more than gains. Specifically, I test whether there are statistical differences in means of emotional responses prior to or following intervals of large gains versus intervals of large losses. The null hypothesis is that there is no difference. Using the single security profit and loss events (the same events found in Tables C.31 and C.32), a two-sided t test is performed in a head-to-head comparison. Note that this test is different than the tests in Tables C.22 and C.23, which coupled the trade events directly to profit and loss. Now, the profit and loss are decoupled from the trade—the profit and loss is the only factor. Table C.35 shows the results. The left panel compares features prior to large gains against features prior to large losses. The right panel compares features following large gains against features following large losses. In both cases, few of the features appear to be significant.

A similar test can be performed for the overall profit and loss events (the same events found in TablesC.33 and C.34). The results of the test, found in Table C.38, confirms that there are few significant features. Based on these results, one possible conclusion is that losses are not felt differently than gains.

The fourth and final test is to determine whether anticipating or reacting to gains or losses have similar emotional response signatures. Once again, I use the same profit and loss events as above, but now compare pre-event to post-event feature vectors for both gains and losses. Table C.36 shows the results for single security gain events. Table C.37 shows the results for single security loss events. Table C.39 shows the results for overall gain events. Table C.40 shows the results for overall loss events.

From these tests, it appears that 15004 is the only trader to exhibit any differences before and after gains. It may be mere coincidence, but 15004 is the biggest money maker of the group. 15006 exhibits a different SCR signature preceding overall P&L events than following.

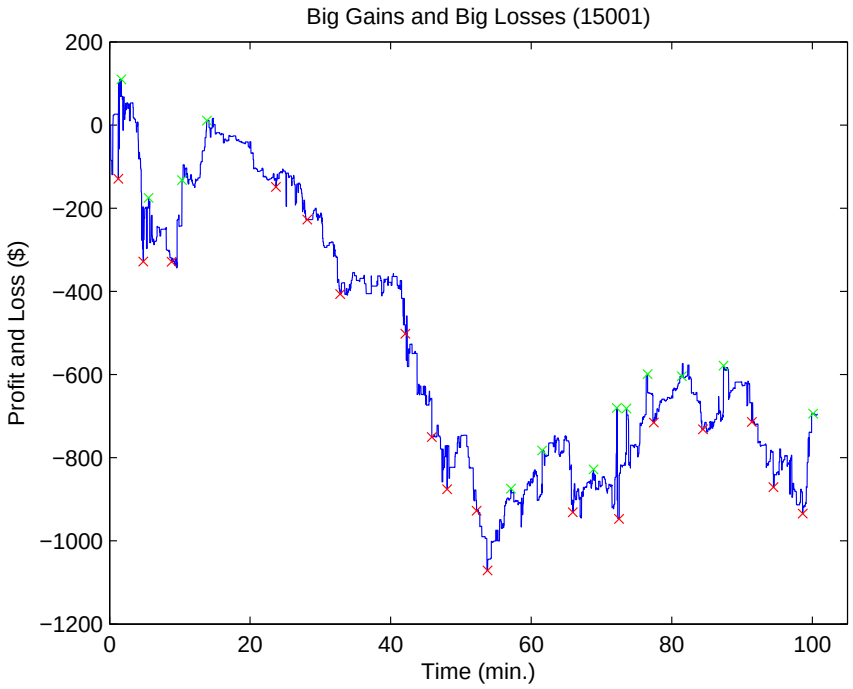

Figure 5-7: Overall Profit and Loss events of big gains (green) and big losses (red) (15001).

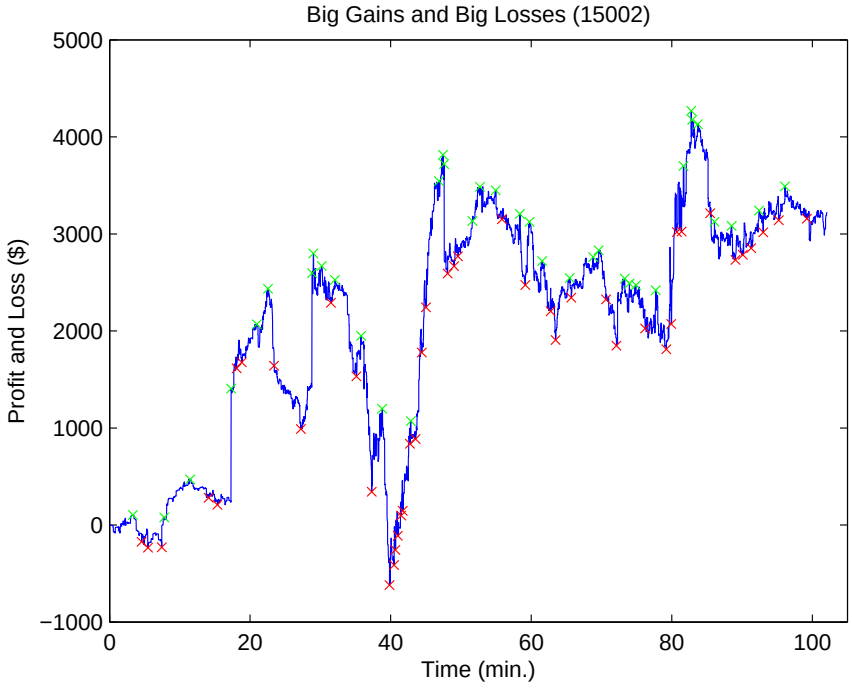

Figure 5-8: Overall Profit and Loss events of big gains (green) and big losses (red) (15002).

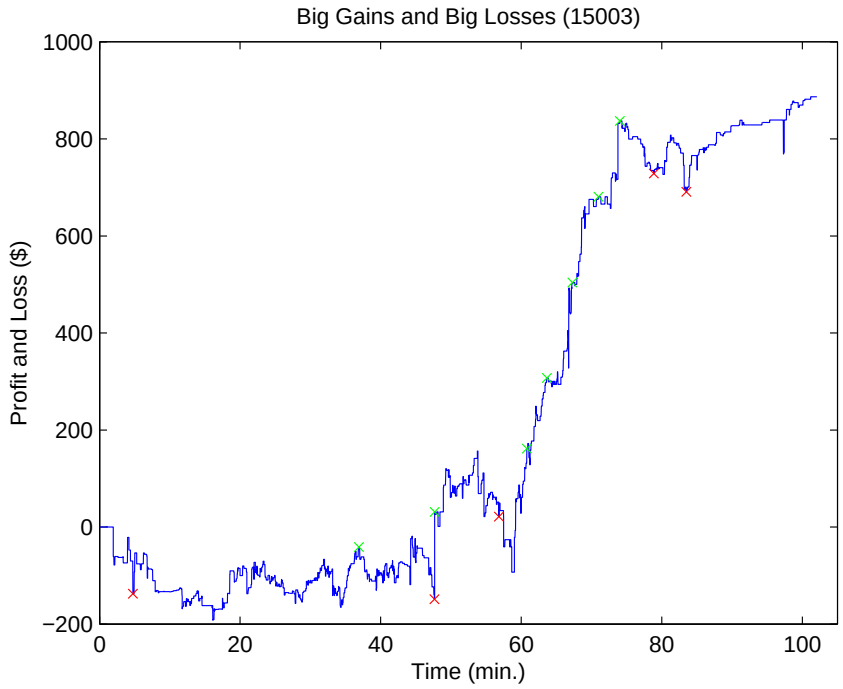

Figure 5-9: Overall Profit and Loss events of big gains (green) and big losses (red) (15003).

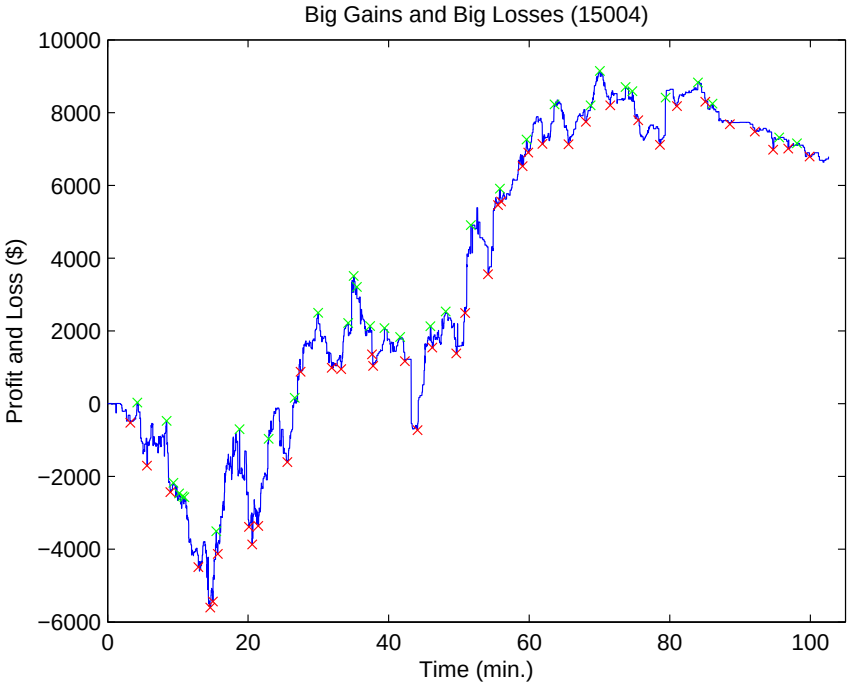

Figure 5-10: Overall Profit and Loss events of big gains (green) and big losses (red) (15004).

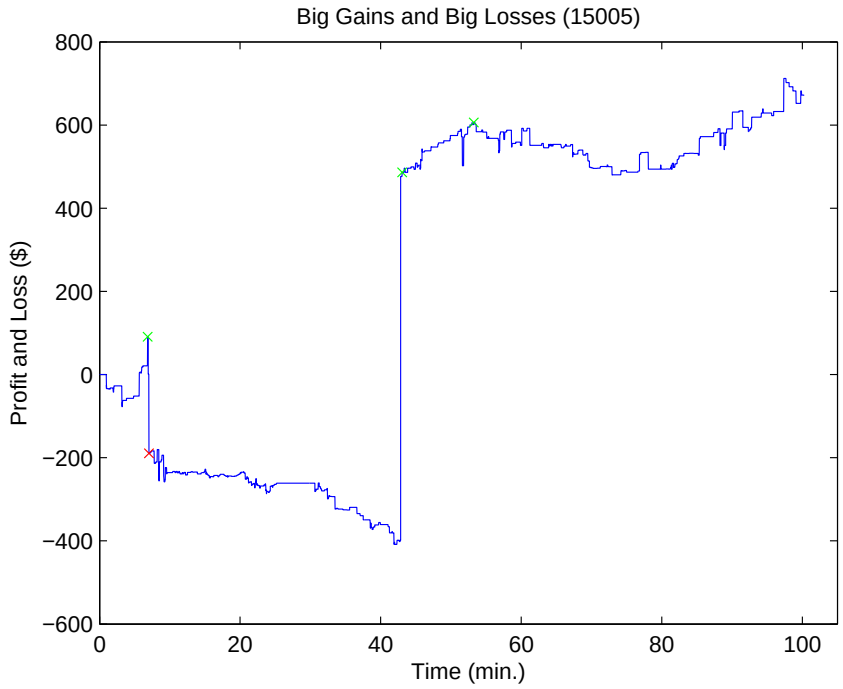

Figure 5-11: Overall Profit and Loss events of big gains (green) and big losses (red) (15005).

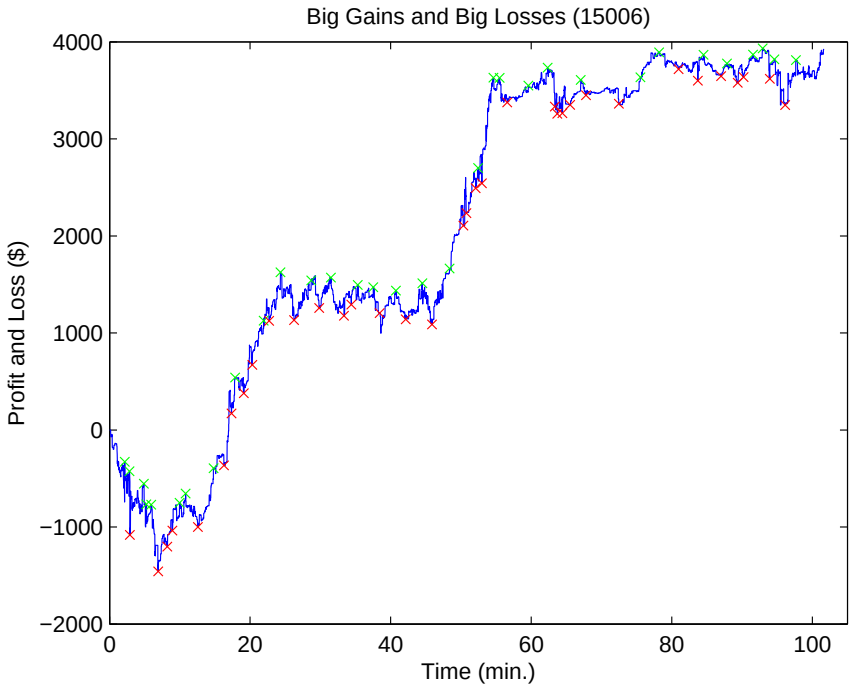

Figure 5-12: Overall Profit and Loss events of big gains (green) and big losses (red) (15006).

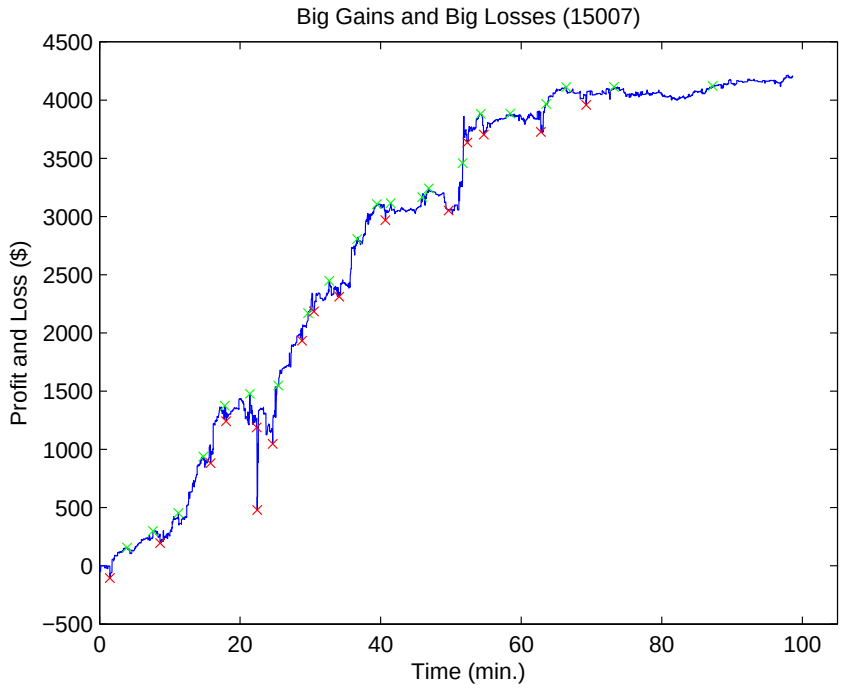

Figure 5-13: Overall Profit and Loss events of big gains (green) and big losses (red) (15007).

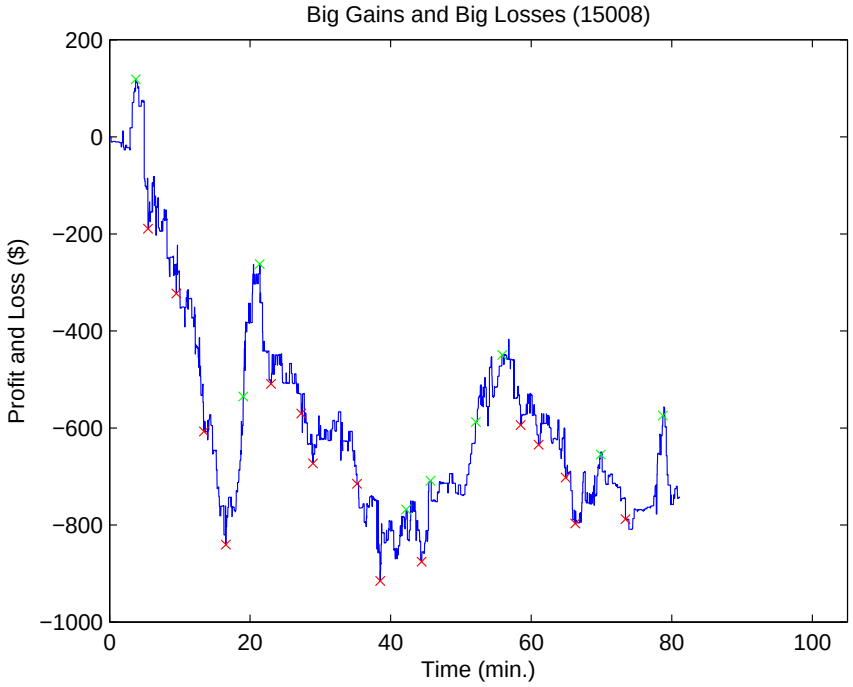

Figure 5-14: Overall Profit and Loss events of big gains (green) and big losses (red) (15008).

5.5 Model of Affect

This section is titled *Model of Affect*, though it will be quite unlike the usual *qualitative* models of affect proposed by the emotion theory community (Cacioppo and Gardner, 1999). Instead I present a *quantitative* model of affect. Before doing so, I will highlight two or three models of affect which, by and large, are qualitative.

Qualitative models of affect rely on self-reported emotional states, such as "I feel happy" or "I am disappointed." These labels may be dichotomous ("I am sad" or "I am not sad") or ordinal ("On a scale of 1 to 10, how sad do you feel?"). In contrast, a quantitative model would not rely on self-reported emotions, but use physiological features as proxy for emotion. For example, a quantitative model of affect might define happiness as a process whose manifestation can be measured by parameters such as heart rate, amplitude of the BVP, etc. Qualitative, self-reporting measures, which are currently the standard, could be replaced by more objective measures of affect. This thesis contributes novel research to the body of work that aims to define a quantitative model of affect.

Qualitative models of affect may be classified as either *categorical* or *dimensional*. Categorical models of affect, such as those advocated by Descartes, posit that emotions are discrete and that emotions belong to one of a small set of base clusters (Ortony et al., 1988). Among emotion theorists, the set of universal categories varies in both content and length, though most authors include *happiness*, *sadness*, *fear*, and *anger*.

Dimensional models of affect posit that emotions can be decomposed into a continuous space along multiple axes or features or dimensions—not a discrete, finite set. (Schlosberg, 1953) proposed a 3-dimensional model: one dimension accounting for pleasantness or *valence* (happy vs. sad), one dimension accounting for level of activation or *arousal* (calm. vs. excited), and one dimension accounting for level of attention or *stance*. See Figure 5-15. It is not uncommon for the last axis to be omitted, resulting in a model with two axes: valence and arousal. In machine-learning terminology, I like to think of the categorical model as classification using a nearest-neighbor algorithm, where the distances between clusters is unknown but the distances within clusters is known. I like to think of the dimensional model as regression.

Throughout much of this document, I have avoided using the word *emotion* and have used the phrase *emotional response* where possible because I prefer to sidestep philosophical

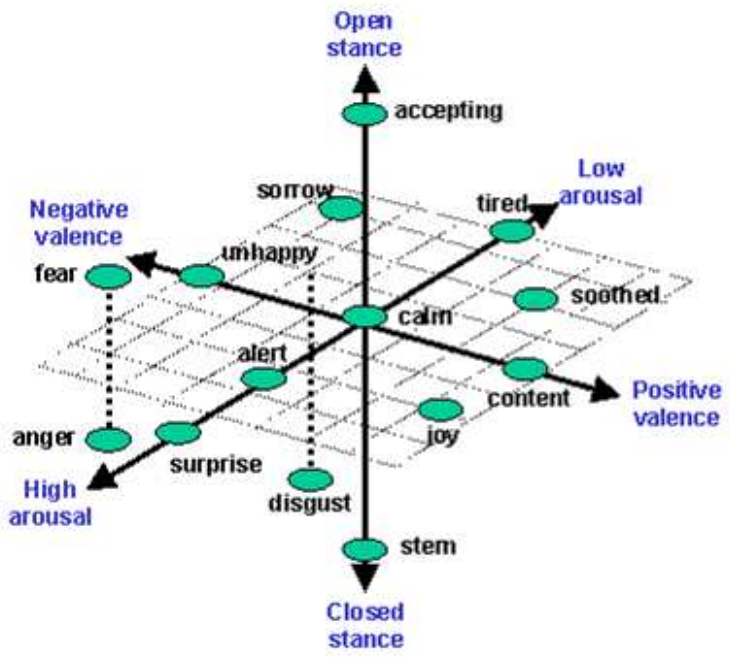

Figure 5-15: Axes of emotion

debates. This paper makes no claims or contributions relative to the theories of emotion, identifying or recognizing emotions, or correlating specific emotions with other phenomena. What I do contribute is an analysis of the components of the emotional response. The analysis is an effort to identify the quantitative features, or axes, of emotion, without assigning labels, such as fear, anger, or sadness, to such axes.

The analysis is based on principal components analysis (PCA). I use the terms PCA and eigenanalysis interchangeably. The latter term focuses attention on the fact that not just the eigenvectors (principal components) are important here, but also the eigenvalues.

Using the same physiological features as above, I calculated feature vectors for every 10 sec for each trader. After normalizing the feature vectors, I computed the principal components of this vector.

Figure 5-16 shows the scree plots, which plots the eigenvalues, sorted from large to small, as a function of the eigenvalue, or component, index. Based on the knee in the scree plots, it seems that most of the specialists have at least two significant eigenvalues. In addition, the first two eigenvalues are greater than two, which indicates their importance in eigenanalysis.

In order to determine whether one trader's eigenvector aligned with a second trader's eigenvectors, I computed the dot products of the first two eigenvectors of each trader.

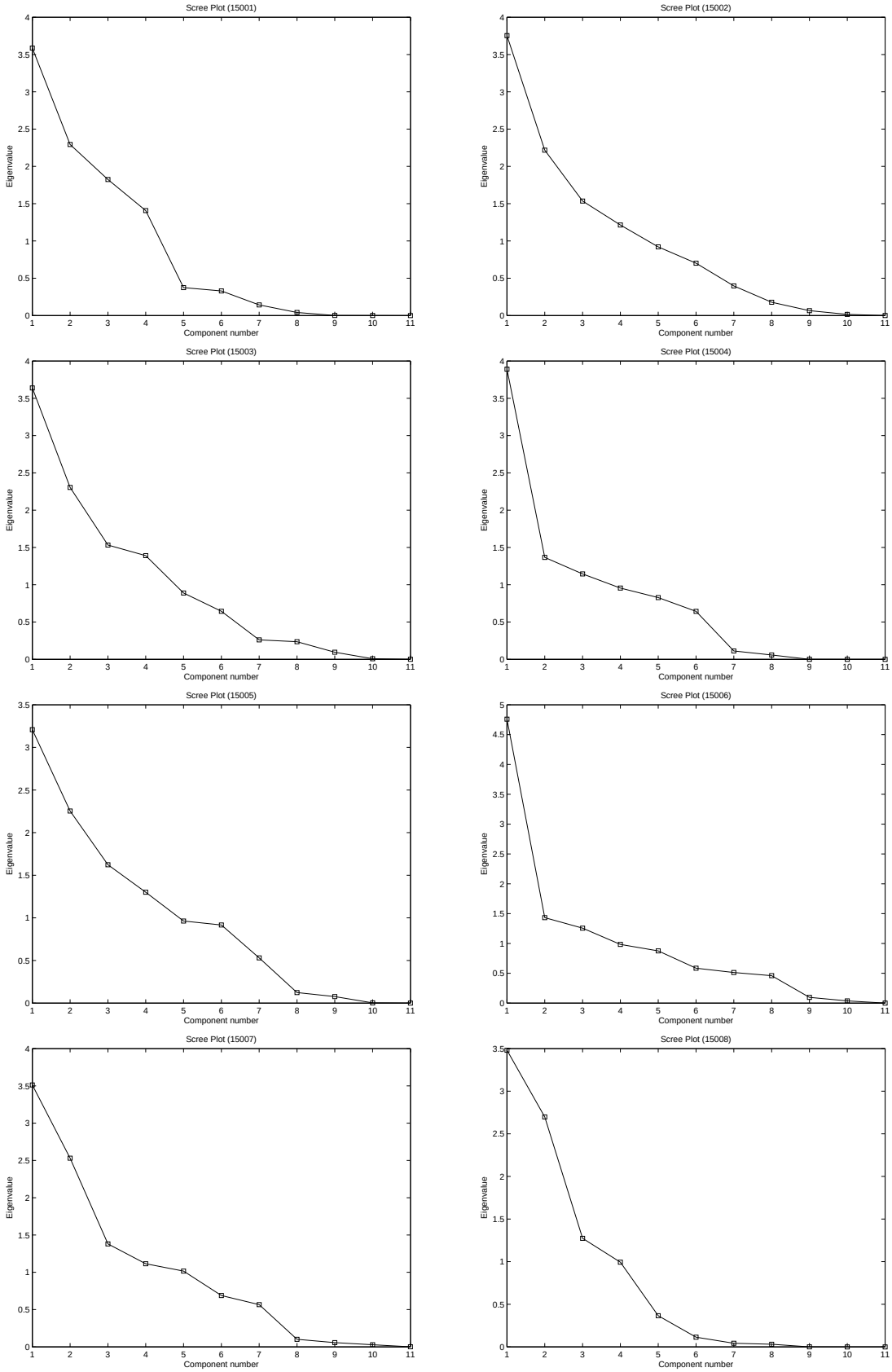

Figure 5-16: Scree plots

	15001	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*
15001	1.00	0.98	0.97	0.83	0.93	0.90	0.84	0.85
15002	0.98	1.00	0.98	0.90	0.92	0.87	0.95	0.79
15003	0.97	0.98	1.00	0.90	0.92	0.82	0.90	0.70
15004	0.83	0.90	0.90	1.00	0.78	0.96	0.91	0.97
15005	0.93	0.92	0.92	0.78	1.00	0.89	0.94	0.77
15006	0.90	0.87	0.82	0.96	0.89	1.00	0.96	0.94
15007	0.84	0.95	0.90	0.91	0.94	0.96	1.00	0.86
15008	0.85	0.79	0.70	0.97	0.77	0.94	0.86	1.00

Table 5.5: Maximum dot product of first two eigenvectors for all traders.

Table 5.5 shows the maximum dot product of these four dot products. For example, the value in the cell of 15001 vs. 15002 is 0.98, which is the maximum of the following four dot products: (1) the dot product of the first eigenvector of 15001 and the first eigenvector of 15002, (2) the dot product of the first eigenvector of 15001 and the second eigenvector of 15002, (3) the dot product of the second eigenvector of 15001 and the first eigenvector of 15002, and (4) the dot product of the second eigenvector of 15001 and the second eigenvector of 15002. By way of reminder, the dot product of a normalized vector with itself is one; the dot product of orthogonal vectors is zero. Because eigenvectors of the different traders may not have the same eigenvector ordering, it is necessary to test more than just the first eigenvector. In this case, I have chosen the first two eigenvectors. The table shows that all the traders have physiological eigenvectors that align around 85% or higher.

5.6 Predictions Using SOON

The sections above describe statistical tests aimed at testing specific time intervals, such as trade events. The preceding section described an eigenanalysis of the physiological signals. This section builds on those analyses. I now describe using principal components to build a predictive model for not just specific time intervals but for the entire signal.

Principal components analysis (PCA) is one of many techniques aimed to reducing the dimensionality of data. SOON is an algorithm, similar to PCA, that transforms data into a lower dimension. It is a second-order method for blind source separation of noisy, instantaneous linear mixture where the signal order and noise covariance are unknown Herring (2006). With the assistance of Mr. Herring, I used the SOON algorithm to build two models: one to predict physiological variables from financial variables and one to predict

financial variables from physiological variables.

The first objective of the analysis was to investigate whether the financial data could predict physiological changes, such as increased heart rate or lower skin temperature. The inputs to the SOON algorithm were the financial variables in Table 5.6. To compute the variables, I used a 2 sec. interval in order to provide as large a training set as possible. The physiological variables, which align with the financial variables, were the same features from Section 4.5, only calculated over a 2 sec. interval.

The results of the predictive model were rather disappointing. It appears that the reduced feature space is sufficiently noisy so as to provide no predictive power.

The second objective was to investigate whether the physiological features could predict any of the financial variables. This model to look for anticipation was as unsuccessful as the previous SOON-based model.

5.7 Predictions Using Logistic Regression

The grand challenge of finance, and not just this thesis, is determining under what conditions (price, risk-level, and perhaps time) an investor will buy or sell. The ability to predict such behavior has important and wonderfully rich ramifications. The analyses in this paper have flirted with such predictions. Given my interest in the information contained in the emotional response, this final analysis investigates whether the emotional response is related to trading decisions or, in a stronger sense, can improve predictions of trading decisions. Probabilistically,

$$\mathrm{P}(trade|market, physiology) = \mathrm{P}(trade|market)?$$

In order to determine whether the emotional response contains information, I build models with only financial data and models with both financial and physiological data. I then compare the accuracy of the financial-only model against the financial-physiological model.

I build different models for predicting buy decisions and for predicting sell decisions since those decisions entail processing different financial data. The response variable in both models is binary—either there was a trade or there was no trade. The explanatory variables originate from the same feature set.

Group	*Variable*	*Data Type**
Market Event	DEV Price	B
	DEV Spread	B
	DEV Return	B
	TRV Price	B
	TRV Spread	B
P&L	Single Security Gain	B
	Single Security Loss	B
	Total Gain	B
	Total Loss	B
Alerts	Trades	B
	Quotes	B
Buy Trades	Buys	B
	.. within last min.	S
	.. within last 10 min.	S
	.. since the open	S
Sell Trades	Sells	B
	.. within last min.	S
	.. within last 10 min.	S
	.. since the open	S
Buy Size	Dollars bought	N
	.. within last min.	S
	.. within last 10 min.	S
	.. since the open	S
Sell Size	Dollars sold	N
	.. within last min.	S
	.. within last 10 min.	S
	.. since the open	S
Auto Trades	Trades	B
Clickstream	Count	B
	.. within last min.	S
	.. within last 10 min.	S
	.. since the open	S
Context Switches	Count	B
	.. within last min.	S
	.. within last 10 min.	S
	.. since the open	S
Screen updates	Count	N
	.. within last min.	S
	.. within last 10 min.	S
	.. since the open	S
P&L	Dollar Change	N
	.. within last min.	S
	.. within last 10 min.	S
	.. since the open	S
	Rate of gain	N
	Rate of loss	N
	Volatility within last min.	N

*Data Type: 'B' = Boolean, 'N' = Number, 'S' = Windowed Sum.

Table 5.6: PCA Variables

The first step in model building is to identify variables. The physiological variables are the same that appear elsewhere in this paper. The financial variables appear in Table 5.7. I chose these variables based on my own experience, and this list is not exhaustive. There are limits to the number of variables that can be evaluated. In an ideal world, there is more than enough data to evaluate multiple variables. However, in the real world, data is often limited. In this particular case, the number of positive responses, i.e. trades, is limited. For instance, building a model for sell trades for 15005 has only 15 positive responses. Since the number of variables must be less than the number of positive responses, not even all of the raw physiological variables can be used, let alone the financial variables.

As already observed, the physiological variables are computed over 10 secs. intervals. I use the same 10-sec time interval for financial variables in order to align variables. Consequently, the response variable is 1 if a trade takes place anywhere within the 10 sec. window; otherwise, it is 0.

By way of contrast, PCA (see Section 5.6) starts with *all* the variables and reduces its dimensionality—independent of the response variable. The learning method in this section reduces the dimensionality by sequentially removing variables from the model. However, in this case, the decision to reduce the dimension incorporates the response variable through prediction accuracies. Thus, it should come as no surprise that the analyses produce different results, and different input variable combinations.

The second step in model building is variable selection. To select variables, I used both forward and backward selection techniques. Forward selection is a technique that adds one variable at a time to the model until there are no variables left. Backward selection is a technique that includes all of the variables in the initial model and then deletes variables one at a time until there are no variables left. The decision of which variable to add or delete at each step is a function of a measure that compares all the models with or without the variable. The traditional measure for evaluating two logistic regression models is the difference in the models' deviances. As an example, Table 5.7 lists the difference in deviance, ΔDeviance, for the first step of the forward selection algorithm. The results show that Monitoring within last min. has the largest difference in deviance for any of the single-variable models, followed by Manual Trades Count Sells within last min. Consequently, Monitoring within last min. is the variable added first to the model. The next step in the forward selection algorithm starts with this variable and then determines which is the next

Group	*Variable*	*Data Type**	*ΔDeviance (15001)*
Price	Bid (Normalized)	N	0
	Ask (Normalized)	N	0
	Return of Mid-Price	N	11
	.. within last min.	N	1
Monitoring	within last min.	M	61
	secs within last min.	N	29
Position	Position	N	17
	Sign of Position	M	27
Buy Trades (Auto)	Count within last min.	S	3
	Size of Trade ($) within last min.	S	1
Sell Trades (Auto)	Count within last min.	S	8
	Size of Trade ($) within last min.	S	0
Manual Trades	Count..		
	.. Buys within last min.	M	8
	.. Sells within last min.	M	29
P&L	Overall P&L	N	15
	Single Security P&L	N	0
P&L	Dollar Change	N	2
	Dollar Change within last min.	N	0
	Volatility within last min.	N	18

*Data Type: 'M' = Multinomial, 'N' = Number, 'S' = Windowed Sum.

Table 5.7: Financial variable selection using forward selection algorithm and deviance as measure for logistic regression.

best variable to add.

As common as the difference in deviance is for evaluating logistic regression models, the deviance may not be used to evaluate goodness-of-fit for models with a binary response (Collett, 2002). A less commonly used though equally valid evaluation measure is the area under the curve (AUC), where the curve is the receiver operating characteristic (ROC) curve (Lasko et al., 2005). (The AUC can range from 0 to 1, where a value of 1 means the model perfectly predicts the response variable.) As an example of its use in backward selection, Figures 5-17 to 5-20 show the AUC measure for both Buy and Sell Trade models for each trader. The x-axis lists the number of variables included in the model, and the y-axis lists the AUC. These graphs show that when the model contains few variables, the AUC is lower than when it contains several variables. However, if the model contains all of the variables, the AUC tends to be lower. The AUC measure allows model comparisons. At each step in the backward selection algorithm, the algorithm computes the AUC for each model with one of the outstanding variables omitted. The largest AUC determines which omitted variable to permanently delete.

Number of Variables in Model	*AUC*	*p-value*
1	0.713	< 0.001
2	0.766	0.605
3	0.770	0.888
4	0.771	1.000

Table 5.8: p-values for comparing AUCs of models of buy trades with financial variables with 1–4 variables against the model with 4 variables (15006).

I use two-fold cross validation on the training set to train and evaluate models. Half of the training data is used to learn the parameters of the model and the other half is used to evaluate the model. The training set contains 75% of the data.

Once the variables have been ordered by predictive ability, the challenge is to pick the smallest subset of the variables that captures the greatest amount on information. To identify how many variables to include, I check for statistical differences in the AUC. (By way of contrast, the backward selection algorithm merely uses the differences in magnitude of the AUC—not its distribution.) If the p-value (two-sided) for the difference between the AUCs of the model, m_j, with the largest AUC and any of the models with fewer number of variables, $m_i, i < j$, is less than 0.10, then I choose the model with $i + 1$ variables. The idea behind the cut-off number of $i + 1$ is that the $i + 1$ variable is sufficiently similar to the model with j variables. For example, Table 5.8 shows that p-values for comparing AUCs of models of buy trades with 1–4 variables against the model of buy trades with 4 variables for 15006. The comparison of a model against itself produces a p-value equal to one. (See the panel for 15006 in Figure 5-17). Based on this table, a model with one variable is statistically different than the model with four variables with p-value < 0.001. Thus, the number of selected variables is two.

One interesting observation in Figures 5-17, 5-18, 5-19, and 5-20 is an occasional jump in the AUC from the deletion of one variable. For example, in panel 15001 of Figure 5-18, the AUC with nine variables is 0.880 while the AUC with eight variables is 0.929. In panel 15005 of Figure 5-17, the AUC with eight variables is 0.830 while the AUC with seven variables is 0.912. In panel 15007 of Figure 5-17, the AUC with 16 variables is 0.766 while the AUC with 17 variables is 0.592. For models of Buy Trades, the variable affiliated with this jump is a buy-trade variable. For models of Sell Trades, the variable affiliated with this jump is a sell-trade variable.

The backwards elimination algorithm orders the variables for each of the buy and sell

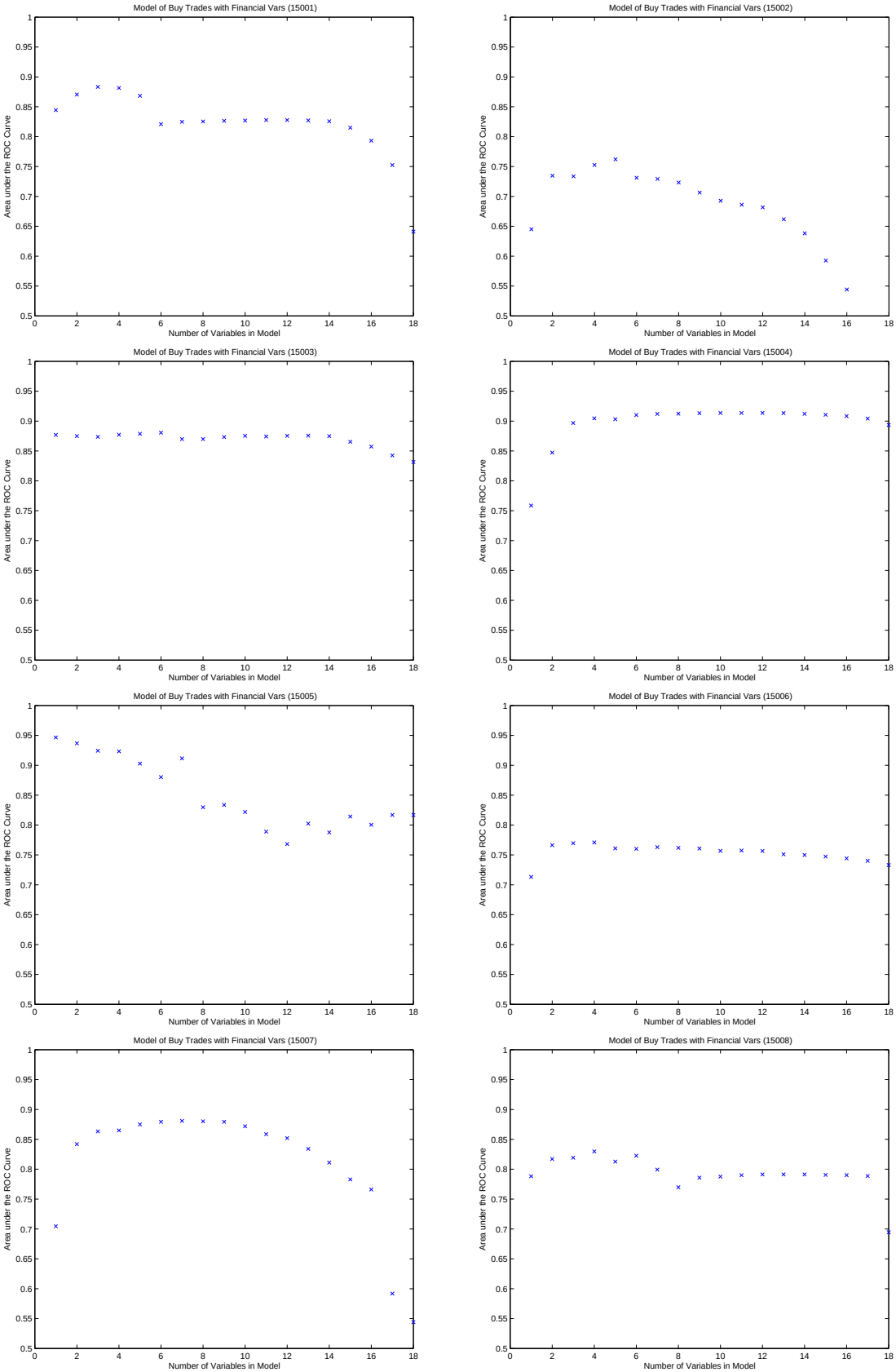

Figure 5-17: Area under the ROC curve of models vs. number of financial variables to predict buy trades

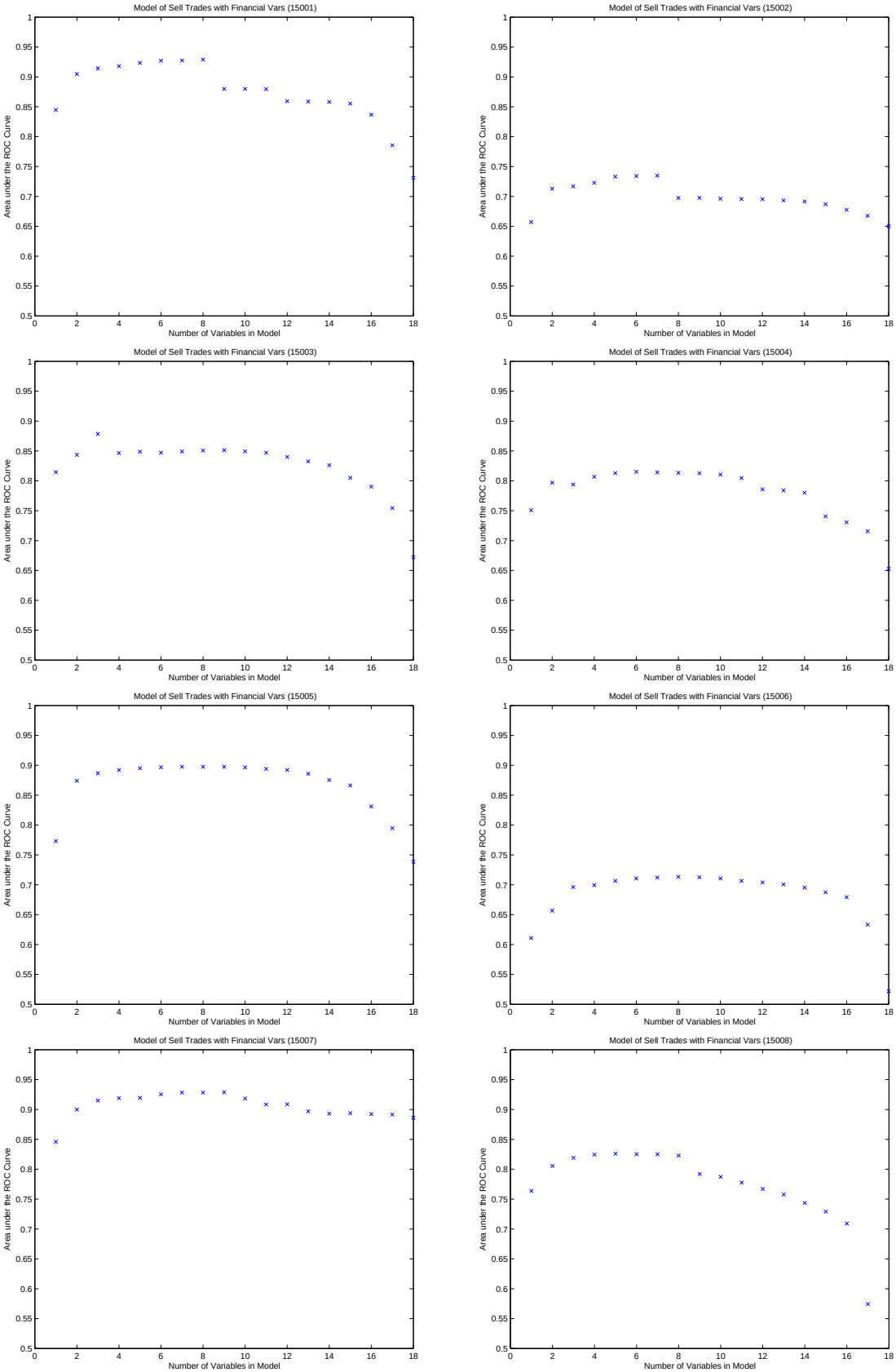

Figure 5-18: Area under the ROC curve of models vs. number of financial variables to predict sell trades

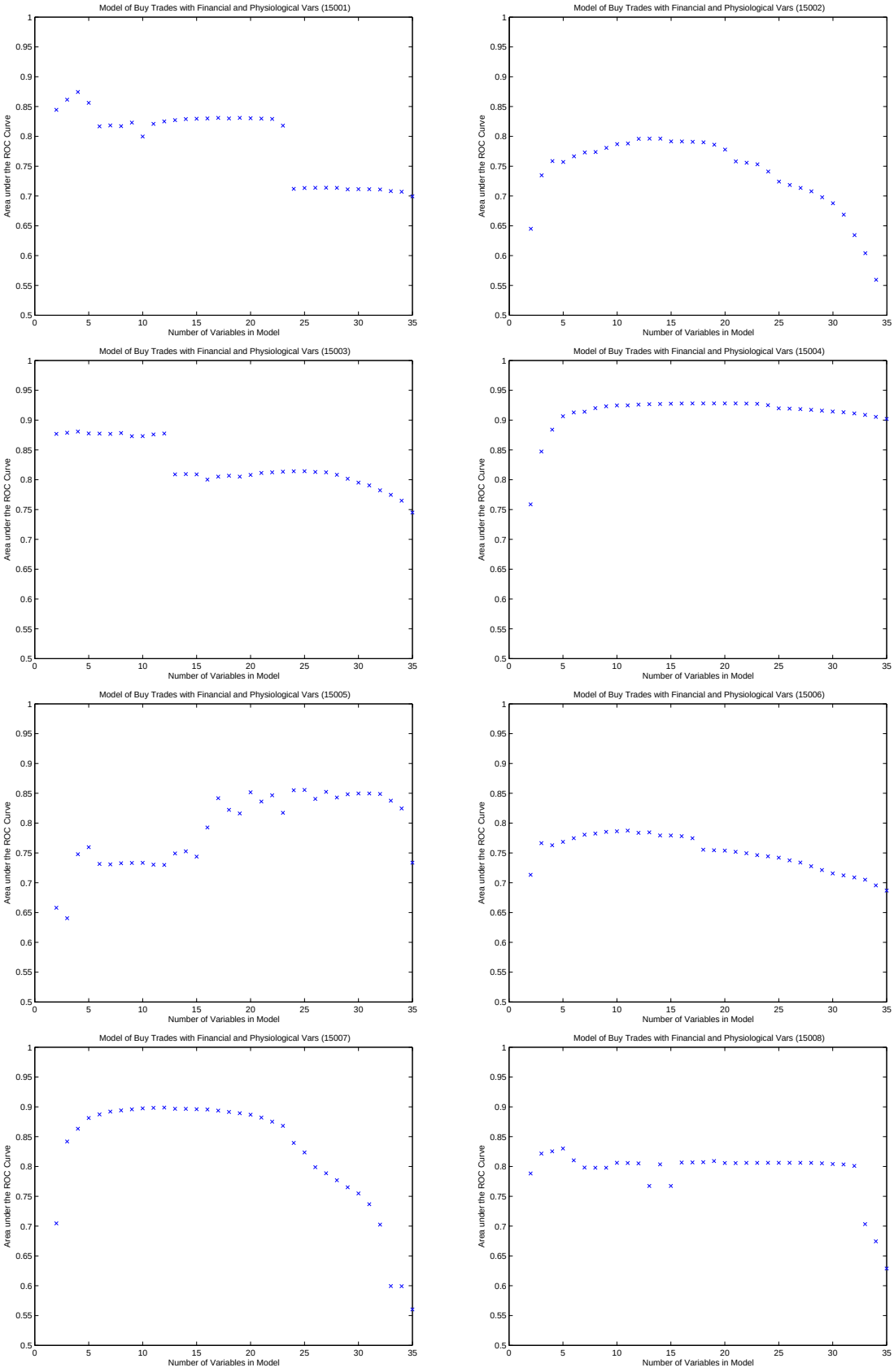

Figure 5-19: Area under the ROC curve of models vs. number of financial and physiological variables to predict buy trades

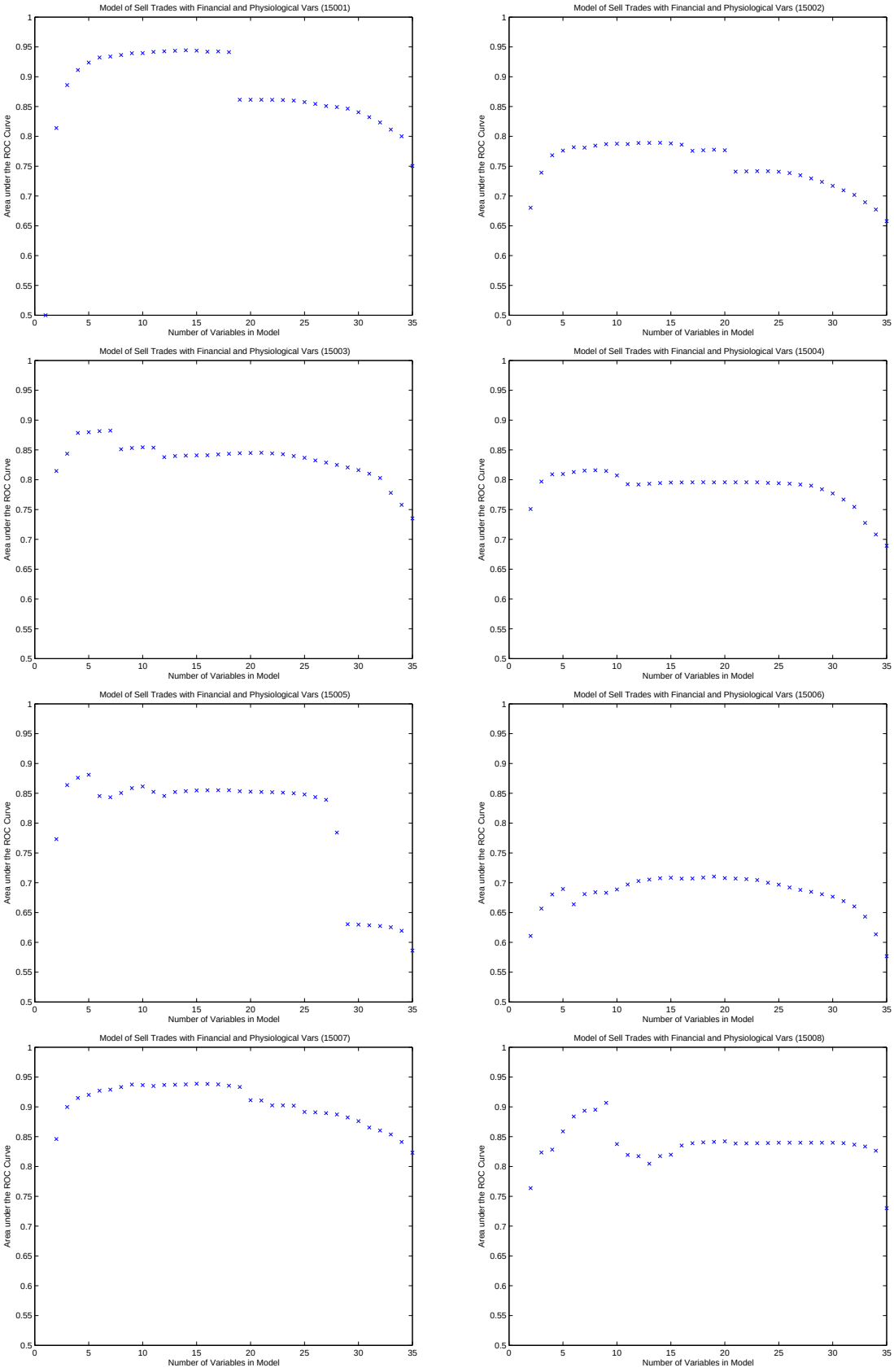

Figure 5-20: Area under the ROC curve of models vs. number of financial and physiological variables to predict sell trades

models for each trader. The variable eliminated at each step is the "worst" variable, in the sense that removing the variable from the model improves the prediction by the greatest amount at that particular step. Averaging the order of each variable across traders for both buy and sell trades provides a rough estimate of the predictive ability of the variables. The ordering, however, says nothing about where the cutoff variable is.

For buy trades, the variable that was eliminated last and, hence, on average was the most important was: seconds monitored within the last minute. This finding should not come as a surprise! By itself, seconds monitored is not all that informative in explaining buy trades. However, in conjunction with other variables, it helps build a more accurate predictor. Consider Figure 5-17's panel 15004 of the buy trades, in which the area under the ROC curve for one variable in the model—seconds monitoring within the last minute—is 0.76. What this means is that 76% of the time a buy trade is made, the stock is being monitored; and the remaining 24% of the time a buy trade is made, the stock is *not* being monitored. Adding a second variable to the model improves the AUC to 0.85.

Averaging across traders, the best five variables of buy trades are:

1. Monitored within the last minute
2. Sign of position
3. Ask price
4. Seconds monitored within the last minute
5. Position size

where monitored within the last minute is binary (+1 if monitored and 0 otherwise); sign of position is ternary (-1 if short, 0 if flat, and +1 if long).

Averaging across traders, the best five variables of sell trades are:

1. Monitored within the last minute
2. Sign of position
3. Seconds monitored within the last minute
4. Return of mid-price
5. Bid price

The preceding analysis considered each trader separately. Now, I perform the same analysis with pooled data. The backward selection algorithm selects the following six variables for the buy-trade response variable, listed in order of importance:

1. Number of buy trades within the last minute (the autoregressive variable)

2. Monitored within the last minute

3. Sign of Position

4. Overall P&L 5. Number of auto sell trades in the last minute

6. Seconds monitored within the last minute

After adding all the physiological variables to the six financial variables, backward elimination indicates that two physiological variables statistically significantly improve the AUC:

1. Heart rate variability from BVP

2. Heart rate acceleration from BVP

Although the improvement is minor, it is statistically significant.

Having identified the variables for the financial model and for the financial-physiological model, I now compare the models. Using 1000 bootstrap samples by sampling from all of the training data (more than 50,000 cases), I learn the parameters for the buy-trade models with only the selected financial variables and with both the selected financial-physiological variables. If the bootstrap sample contains no positive response variables, I discard the sample. I evaluate each learned model by predicting buy trades with all of the test data. From the predictions, I compute the AUC.

Figure 5-21 shows the distribution of the AUC for the buy-trade models with financial and with financial-physiological variables. Note the scale of the x-axis.

Repeating the same pooled data analysis as above but for the sell trade response variable produces nearly the same six variables, in order of importance:

1. Number of sell trades within the last minute (the autoregressive variable)

2. Monitored within the last minute

3. Sign of Position

4. Overall P&L 5. Number of auto sell trades in the last minute

6. Number of buy trades within the last minute

Essentially, the only difference between the buy trade and sell trade variables is the sixth variable.

After adding the physiological variables to the six financial variables and using backwards elimination, one physiological variable shows a statistically significant improvement the AUC:

1. Heart rate variability from BVP

Bootstrapping from all of the training data, I learn parameters for a sell-trade model

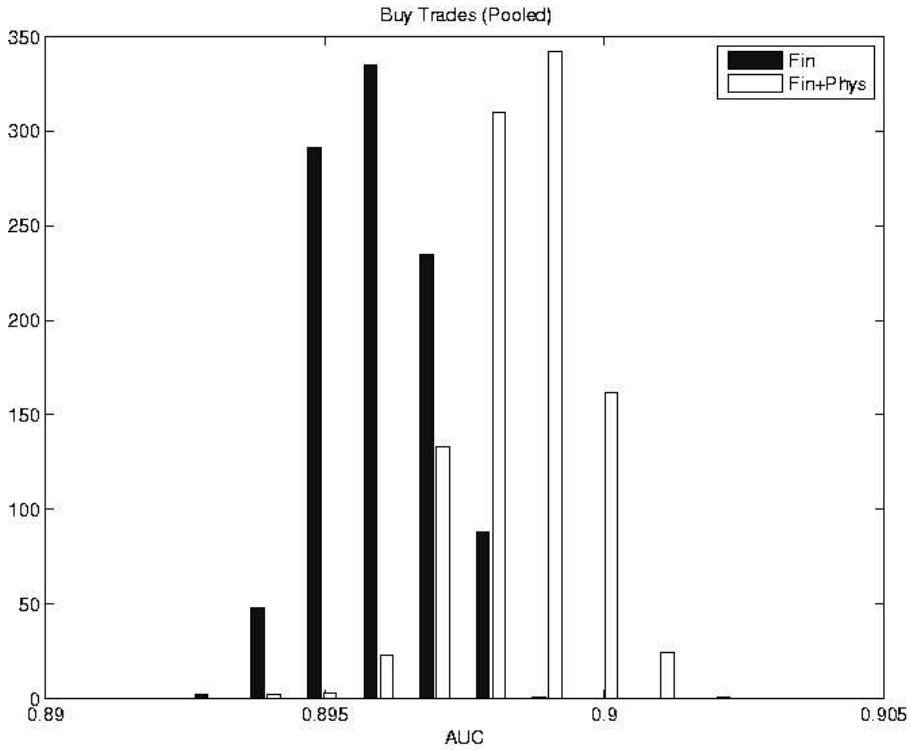

Figure 5-21: Histogram of area under the ROC curve when predicting the buy trades for a pooled sample.

with only the selected financial variables and with both the selected financial-physiological variables. Figure 5-22 shows the distribution of the AUC for the sell-trade models with financial and with financial-physiological variables. Note the scale of the x-axis.

The Kolmogorov-Smirnov test rejects the null hypothesis that the financial-only predictions and the financial-physiological predictions are drawn from the same continuous distribution. Thus, the difference between the financial-only and the financial-physiological model is statistically significant (p-value = 0), which means that the physiological response does contain information. HRV can improve predictions of trades for all eight specialists. However, the improvement is so small—the AUC is better by 0.001—that the difference does not have much effect.

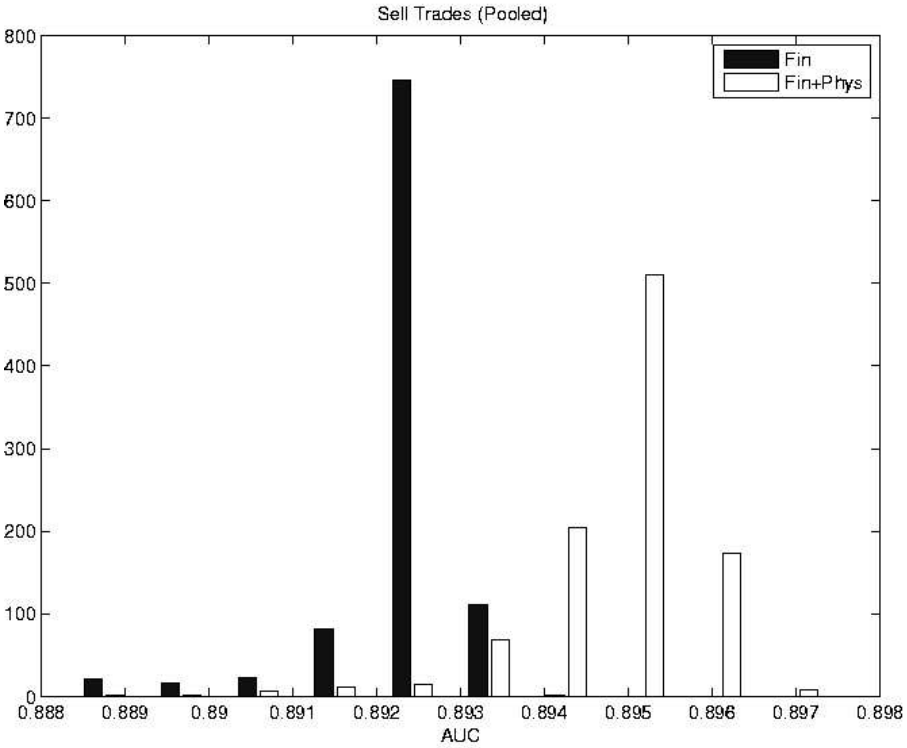

Figure 5-22: Histogram of area under the ROC curve when predicting the sell trades for a pooled sample.

Chapter 6

Discussion

The aforementioned results suggest that emotional responses are associated with real-time processing of (financial) risks. This notion runs contrary to conventional beliefs regarding rational (financial) decision-making. It has been hypothesized that rationality entails acting without emotion and that professional traders—of all people—should be the least likely to exhibit emotion. Yet, there are statistically significant differences in emotional responses related to changes in cognitive inputs.

By capturing the relationship between cognitive inputs and physiological outputs, these findings may be viewed more broadly as a study of cognitive-emotional interactions, which is sometimes called *intuition*. Intuition, not to be confused with *premonition*, is the act of arriving at a conclusion without the use of reasoning. For example, it is possible, though unlikely, that a first-year medical student could accurately diagnose primary HIV because it masquerades as mononucleosis or the flu (Schacker et al., 1996). But a clinician who practices in, say, an inner-city hospital, may have a much better feel, an *intuitive* feel, for proper diagnosis because the clinician has seen repeated, though few, cases over the years. While the word *intuitive* may used to describe both clinicians, I prefer to describe the novice as being lucky (less palatable definitions of intuition are clairvoyance or gut-feeling) and the expert as being intuitive. For the experienced clinician, intuition is subconscious pattern matching. For some, intuition is a rational process, while for others it is not (Redelmeier et al., 1993).

Because intuition is the act of arriving at a conclusion, intuition is also used to describe decision making. This association is logical, given that making decisions, or choosing ac-

tions, is a natural consequence of processing information.[1] Individuals often make intuitive decisions when the need for a decision is too complex or does not allow sufficient time to obtain a detailed analysis of the situation. Usually, the individual cannot explain either the process by which the decision was reached or the reasons for selecting it. Yet, individuals exhibit confidence in the decision because it is based on implicit knowledge and experience. Man's sophisticated pattern-matching skills have been a challenge for even the best computers to mimic. In contrast to intuitive decision-making, analytical decision-making (1) explicitly defines goals and alternatives, (2) calculates the rewards of pursuing the alternatives, and (3) evaluates which alternatives are closest to the goals.

Cognitive science research suggests that there is a continuum of decision-making styles involving an intimate combination of intuitive and analytic techniques. Chess is the most cited example of this phenomenon. Good players intuitively see moves within a few seconds, though they may spend more time analyzing the consequence of such a move. Under time constraints (deadlines), mistakes are more frequent. Experts are able to make decisions instantaneously when compared to novices, who may work through the same task in a conscious and analytical fashion.

The most successful traders seem to trade based on their intuition, often without the ability to articulate a precise algorithm for making these complex decisions (Steenbarger, 2002; Schwager, 1992). Their intuitive trading "rules" are based on associations and relations that are formed in the subconscious. The results established here seem to confirm that decisions based on intuition require not only cognitive but also emotional mechanisms. We may conjecture that such emotional mechanisms are partly responsible for the ability to form intuitive judgments and for those judgments to be incorporated into a rational decision-making process.

The intellectual decision-making giants, such as Kahneman, Tversky, Simon, and Raiffa, have addressed the rational decision-making process. If I were to try and summarize their work, and where this experiment fits in to their work, I would like to first back up and partition decision-making into two parts. One part would include decisions where both the distributions of outcomes and the utility function are known and computable. The other part would include decisions where the distributions of outcomes and/or the utility

[1]It is unfortunate that the Myers-Briggs personality test uses the word *intuition* (vs. sensing) only to refer to how individuals process information. To describe the decision making process, Myers-Briggs defines the axis using the words *thinking* and *feeling*, where *feeling* is supposedly independent of intuition.

function are *not* known or *not* computable, at least at the moment of decision making. In a sense, both parts qualify as making decisions in the face of uncertainty. After all, if there is no uncertainty and the computation tractable, the decision should be trivial. With that framework in place, I see textbook decision analysis as addressing the first of these two parts. See, for example, Howard Raiffa's work. However, in the second of the partitions, there cannot be a decision formulated from the outcomes and utility function because at least one of them is unknown or uncomputable, at least within the time constraints for which a decision must be made. In this situation, Simon says that people move from a state of indecision to a state of decision by satisficing. Satisficing means make decisions that are satisfactory with regards to those pieces of the decision which are uncomputable. Satisfactory is, in most cases, suboptimal. Kahneman and Tversky attacked the problem of unknown distributions and utility functions. According to their research, psychological elements, such as emotion and cognitive biases, help decision-makers formulate outcome distributions and utility functions.

Now, if the utility function is known for an uncertain situation of the more complicated type (as just described), then the psychological elements—emotion—may serve to influence the perception of outcome distributions. Of course, that scenario would play nicely into the experiment described in this paper.

While the analysis above seems to be rather convincing—there appears to be a different physiological signature during epochs of trading—some may argue that the findings may be undermined by the complexity of the trading environment. For example, a market event, a trade alert, a trade decision, and a P&L event could all take place at the exact moment. It would be virtually impossible to separate each event and partition a certain percentage of cognitive processing and physiological response to each event.

These results may be less than noteworthy if the experiment had been conducted inside a controlled environment, such as laboratory. But this experiment was conducted on a regional stock exchange with a group of highly experienced traders. Because of the competitive nature of capital markets, only the fittest traders survive. Given this survivorship bias, we may conclude that learning how to manage the emotional response is critical to survival. (Kiev, 1998), who mentored traders at SAC Capital Partners, one of the most successful hedge funds of the past decade, points out, "How you handle the seesawing effects of the marketplace and the emotions they provoke often turns out to be a more significant factor

in your trading success than the trading method you have chosen."

6.1 Future Work

There are several possibilities for future work.

Perhaps the single most important data missing from the experiment was a measurement of the baseline emotional response of the traders, i.e., what is their emotional response outside of the trading pit when making decisions?

Future work should include more data! The data collected only allows for short-term performance analysis. Long-term outcomes of buy-and-hold strategies are not known because I have no such data.

As already mentioned above, events may coincide and/or be correlated. For example, P&L events are intertwined with trading decisions, making it virtually impossible to separate the two. Yet, it is the feedback from the trade decisions that makes the trading floor such a rich environment to study. Perhaps a future study will have enough data so as to be able to identify non-overlapping events, thereby isolating the event of interest.

Perhaps a future study could compare the emotional response of high-frequency traders to the response of low-frequency traders, otherwise known as buy-and-hold asset managers. I would postulate that the time frame under which their (investment) decisions are made—short-term vs. long-term—influences whether the decisions are made implicitly or explicitly.

To further refine the hypothesis regarding decision-making in the face of uncertainty and its relationship with emotion, any number of possible studies could be conducted. One of the advantages of studying traders is that the feedback is almost immediate. In contrast, feedback from clinical decisions may take years to play out. Consequently, I would choose a domain with fast feedback, such as measuring emotional responses of air traffic controllers, emergency-room clinicians, test takers, or video game players (Green and Bavelier, 2003; Sykes and Brown, 2003).

As mentioned early in this paper, neurobiologists have shown that suppressing the emotional response impairs memorization and that memorization is improved when facts are learned in connection with an emotion. A future study may attempt to suppress the emotional response as much as possible and then measure whether people demonstrate any difference in their ability to acquire intuitive knowledge or make decisions.

Atropine is a possible drug candidate for blocking the ANS. Atropine is a competitive antagonist of the muscarinic acetylcholine receptors. (Acetylcholine is the main neurotransmitter used by the parasympathetic nervous system.) Consequently, atropine lowers the "rest and digest" activity of all muscles and glands regulated by the parasympathetic nervous system.

Another future, drug-based study may investigate the performance of memory enhancers. (Yesavage et al., 2002) discovered that donepezil, a drug approved by the FDA to slow the memory loss of Alzheimer's patients, improves the memory of the normal population. In the study, pilots were trained in a flight simulator to perform specific maneuvers and to respond to emergencies that developed during their mock flight, after giving half the pilots donepezil and half a placebo. One month later the pilots were retested. Those who had taken the donepezil remembered their training better, as shown by improved performance. Other memory enhancers include "smart drugs", or nootropes, such as MEM 1414. MEM 1414 is a promising chemical currently in clinical trials which shows promise to enhance memory.[2] Perhaps, kindred drugs will be discovered that enhance decision making.

This study did not test personalities using frameworks such as Jung-Myers-Briggs, the Big Five, or Beck Depression Inventory to determine whether any of the subjects experience chronic stress or display negative affect (classical psychological information).

Psychologists have found that individuals whose large emotional response shoots up and then goes away quickly are happier, more relaxed, and have better relationships with other people when compared to individuals with chronically increased stress. Future studies may analyze whether traders with sluggish responses underperform.

As explained in Section 4.4.1, there does not appear to be any obvious relationship between gamma band activity and trading activity. If gamma band activity truly is related to attention, one possible conclusion is that the specialists are not paying attention, a conclusion that does not seem very plausible. Future work may investigate what part of the brain wave is more closely correlated with trading.

If I repeat this research project, I would use a subset of the sensors. Not all of the sensors provided interesting data. For example, I would use the SCR, BVP, EMG-Arm, and possibly EEG. The ECG may provide more detailed information about heart rate

[2]MEM 1414 is a product of the work of Eric R. Kandel, who won the year 2000 Nobel Prize for his research on learning and memory in the sea slug Aplysia.

activity than BVP, but the BVP is much easier to record in real world settings.

Future studies that would be peripherally related might use functional magnetic resonance imaging—instead of psychophysiological responses—to study the anticipation and experience of monetary gains and losses. Rather than study random subjects, as was done by (Breiter et al., 2001), I would suggest studying experienced traders and/or comparing inexperienced vs. experienced traders fMRI profiles.

6.2 Contributions

First and foremost, I have completed a detailed analysis of a unique data set, collected on the floor of one of a regional stock exchange.

In this research, I have confirmed the results of a pilot study (Lo and Repin, 2002), verifying the existence of statistically significant differences in mean autonomic responses during transient market events relative to no-market-event control intervals. In addition, I have shown a strong link between autonomic responses and trading decisions.

I have demonstrated that physiological responses—heart rate variability—contains sufficient information to statistically significantly improve predictions of trading decisions, although not necessarily enough to mean much.

Finally, I have paved the way for future research in this area showing, in part, what works and what does not. Hopefully, future work will improve upon these findings.

Appendix A

Lab Experiment

This section includes a result from one experiment performed in the MIT Sloan Trading Lab.

The lab experiment consists of a series of three trading games. Each trading game mimics, or rather, is designed to mimic financial markets. In the game, only one security may be bought and sold. At the start of a game, each participant is allocated the same amount of money. The participant who ends the game with the most money wins. The game is a two-periods. Each period lasts about five minutes. Information about the security's value is gradually released to the market over the course of the game. The game was developed by the Laboratory for Financial Engineering.

Each experiment includes one practice and two trials of the game. The practice game is to help the participants learn to use the software. The game does not count, and the results are not tabulated. Participants in the game were mostly PhD students from MIT and Harvard.

Figure A-1 shows the SCR for one of the participants. The baseline of the SCR is higher when the market is open than when the market is closed. The first 10 minutes were spent adjusting the equipment and letting the participant acclimate. The practice trial, which is not marked on the figure, occurred sometime between minutes 10 and 20. There was a small break followed by the first trial, which began at minute 25. Each period lasted 5 minutes and the trial ended at minute 35. Towards the end of the first period of the first trial, the SCR began to slowly drop, but as soon as the second period started, the SCR fired up to even higher levels. Following the first trial, there was a 10-minute break during

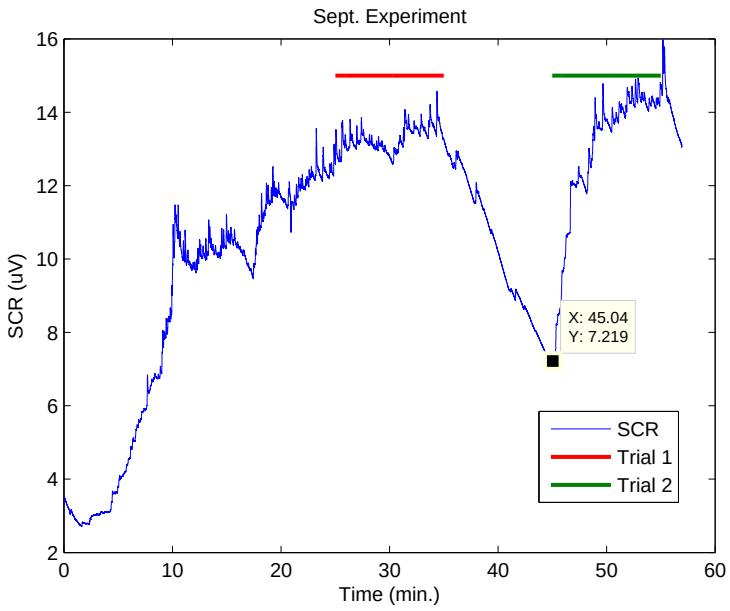

Figure A-1: Experiment showing how the SCR baseline changes during risk processing.

which results were publicly tabulated. The second trial began at minute 45 and lasted 10 minutes. As soon as the trial ended, the SCR started returning to lower levels.

Appendix B

Setup Protocol

1. Prepare sensors by attaching electrodes.
2. Prepare sites:
    - Wipe non-dominant palm with prep pad
    - Wipe forehead with prep pad and NuPrep
3. Attach unit to individual's belt
4. Arm
    - Attach EMG to forearm muscle
    - Attach BVP to top of thumb and secure with tape
    - Attach Temp to top of first (index) finger and secure with tape
    - Wait to attach SCR
5. Head
    - Attach EMG to trapezius muscle
    - Attach EKG
    - Attach EEG
    - Attach Temp to left temple and secure with tape
6. Attach SCR to lower palm of non-dominant hand
7. Begin collecting data

Appendix C

Tables

	Random Events															
	Pre-event Interval								Post-event Interval							
Physiological Features	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*
# of SCR responses	**2.06**	-0.59	-1.34	-0.36	0.66	0.58	-0.29	0	-0.56	0.85	0	-1.54	0.58	0.62	-0.41	0
Ave. SCR amplitude	-0.92	-0.14	-1.00	-0.19	-0.85	-1.06	-0.83	-0.77	-0.08	0.54	0.88	0.30	0.16	0.89	0.60	-1.14
ECG HR	0.28	0.02	0.37	-	-1.60	1.51	-0.19	-	-0.27	0.40	1.72	-	-1.46	1.76	-0.13	-
Local BVP ratio	-0.44	1.10	0.34	-1.19	**-2.52**	0.48	-0.19	0.81	-0.57	1.80	-0.19	-1.69	1.87	0.85	-0.11	0.55
Global BVP ratio	-0.44	1.12	0.35	-1.17	**-2.57**	0.46	-0.24	0.81	-0.58	1.81	-0.16	-1.70	1.86	0.86	-0.13	0.56
# of Temp jumps	-	**-2.04**	-0.65	1.04	0.95	0	0	0.47	-	**-2.39**	-0.33	0	0.95	-1.00	0	-0.41
Ave. SCR duration	-1.20	-0.62	-0.83	-0.38	-1.42	-0.52	-0.48	-0.24	0.32	0.91	-0.17	-0.25	-0.96	1.95	0.17	-0.95
SCR area	-1.03	-0.51	-1.28	-1.60	-0.05	-1.77	-0.73	-0.88	0.06	-0.25	0.84	-1.78	0.14	0.47	0.15	-1.42
Ave. SCR Δ	-0.86	-1.00	-	0.74	-1.36	**2.83**	-0.29	0.43	0.99	-1.00	-	0.40	-1.60	-1.33	0.26	1.83
ECG HRV (Std)	-1.15	-0.37	-0.05	-	-0.64	0.10	-0.94	-	-1.77	-0.79	0.14	-	0.15	-0.99	-1.03	-
ECG HR Accel. (RMS)	-0.01	0.01	0.29	-	-1.45	1.48	-0.32	-	-0.80	0.27	1.42	-	-1.15	1.44	-0.41	-
ECG HR Accel. (Std)	-1.53	-0.26	0.18	-	-0.46	0.27	-1.00	-	-1.73	-0.97	0.14	-	0.54	-0.28	-1.14	-
Range of Temp	-1.07	**-2.28**	-0.63	-0.72	-0.98	0.22	0.69	0.35	-0.76	1.76	0.62	-0.13	-0.28	0.52	0.98	-1.14
Ave. EMG-Arm amplitude	-0.73	1.19	0.97	-1.25	-0.48	-0.43	-0.05	0.18	0.22	1.54	1.89	-1.17	0.69	0.16	1.84	0.58
EMG-Arm zero-crossings	-0.05	1.41	0.69	-1.32	-0.44	-1.52	0.50	0.50	0.70	**-2.33**	1.47	-1.35	0.81	-0.59	1.76	0.52
Ave. EMG-Back amplitude	-0.88	0.27	0.28	-1.06	0.03	0.01	0.20	0.24	-0.68	0.37	0.14	-0.96	0.30	0.22	0.64	0.27
EMG-Back zero-crossings	1.39	0.70	-1.19	-	-	1.33	-1.00	-	1.39	0.83	-1.11	-1.37	-	1.33	-1.00	-
BVP HR	0.01	0.23	-0.16	0.53	**2.57**	0.45	0.95	0.76	-0.43	0.19	-0.37	0.29	**2.13**	-0.64	-0.11	0.41
BVP HRV (Std)	-0.65	-0.50	0.05	-0.82	**2.23**	0.96	0.46	1.04	1.13	0.59	0.59	-1.17	-0.61	0.56	0.74	1.29
BVP HRV (Range)	-0.75	-0.39	0.43	-0.12	**2.32**	1.09	0.36	0.90	1.42	0.43	0.41	-0.81	-0.57	0.28	0.72	1.25
BVP HR Accel. (RMS)	0.04	0.18	-0.16	0.40	**2.65**	0.71	1.11	0.92	-0.33	0.28	-0.35	0.03	**2.14**	-0.56	0.03	0.62
BVP HR Accel. (Std)	-0.47	0.13	0.11	-0.72	-1.81	0.42	0.40	0.62	1.24	0.73	0.27	-1.46	0.33	0.21	0.96	0.92
BVP Volatility	-0.48	0.90	0.39	-1.19	**-2.55**	0.06	-0.45	0.70	-0.61	1.57	-0.14	-1.80	1.86	0.81	-0.56	0.36

Bolded statistics are significant at the 5% level.

The left panel contains t statistics for pre-event features and the right panel contains t statistics for post-event features, both tested against the same set of control features. See Table 3.1 for abbreviations.

Table C.1: t Statistics for two-sided hypothesis tests of the difference in mean autonomic responses during pre- and post-event intervals versus no-event intervals (controls) for each of the components of the physiological features (rows) for random events.

	DEV Price (10-sec Interval)															
	Pre-event Interval								Post-event Interval							
Physiological Features	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*
# of SCR responses	-0.52	-1.04	1.67	-0.32	-1.19	0.52	-0.52	-0.72	-0.28	-0.92	1.67	-0.11	-1.77	-0.22	-0.21	-1.05
Ave. SCR amplitude	-1.44	-0.87	1.16	0.45	-2.00	**-2.06**	**-2.25**	-1.01	-1.18	-0.82	1.16	0.34	-1.94	1.67	**-2.03**	-1.10
ECG HR	-0.18	**2.12**	-1.94	-	-1.69	-0.32	-1.47	-	-0.27	-1.98	-1.85	-	-1.45	-0.70	-1.81	-
Local BVP ratio	0.23	-1.12	-1.54	-1.80	**-3.48**	0.09	**-2.33**	1.12	0.52	-1.00	-1.81	-1.76	**-3.39**	-0.07	**-2.54**	1.41
Global BVP ratio	0.22	-1.13	-1.46	-1.84	**-3.49**	0.06	**-2.42**	1.12	0.53	-1.02	-1.70	-1.83	**-3.40**	-0.07	**-2.62**	1.42
# of Temp jumps	-	-0.35	0.97	-1.44	**2.71**	-	0.47	**2.28**	-	0.65	**-2.14**	-1.44	-1.98	1.00	0.47	**2.99**
Ave. SCR duration	-1.17	0.05	1.81	-0.84	**2.46**	-1.61	-1.84	-0.80	-1.18	0.05	1.81	-1.12	**2.45**	-1.92	**2.32**	-1.11
SCR area	-1.20	-0.99	1.17	0.88	**2.63**	1.19	0.76	-0.93	-0.76	-0.85	1.17	0.86	**2.77**	0.39	0.56	-0.99
Ave. SCR Δ	0.68	1.06	-	0.09	-1.28	-0.13	-1.55	-0.45	0.18	1.06	-	0.24	-1.35	-0.18	-1.39	-0.00
ECG HRV (Std)	-0.01	-0.06	**-2.28**	-	0.93	-1.01	1.13	-	0.28	0.49	**-2.22**	-	0.99	-1.47	1.15	-
ECG HR Accel. (RMS)	0.01	**2.10**	-1.12	-	-0.98	-0.43	-0.97	-	0.01	-1.94	-1.06	-	-0.81	-0.91	-1.24	-
ECG HR Accel. (Std)	-0.06	-0.34	1.63	-	0.88	-1.02	1.33	-	0.51	0.27	1.64	-	0.84	-1.51	1.37	-
Range of Temp	0.64	-1.35	0.62	0.09	**2.98**	1.03	-0.54	-1.74	0.61	-1.23	1.13	-0.03	**2.30**	1.95	-0.38	**2.39**
Ave. EMG-Arm amplitude	**2.54**	0.82	-1.73	0.93	**2.56**	-2.01	**-2.69**	-0.33	**2.33**	0.70	-1.84	1.30	**2.37**	**2.37**	**-2.88**	-0.35
EMG-Arm zero-crossings	**2.36**	**-2.37**	**2.09**	0.29	-1.28	1.37	1.86	-0.13	**2.25**	**-2.37**	-1.93	0.54	-1.33	1.37	2.01	-0.36
Ave. EMG-Back amplitude	-0.65	0.45	**9.26**	-1.36	0.06	1.52	-0.07	2.01	-0.54	0.33	**9.57**	-0.94	0.01	1.48	0.26	1.86
EMG-Back zero-crossings	-0.25	-0.85	-1.49	**4.69**	-0.94	**3.59**	**3.51**	-	-0.60	0	-1.11	**4.98**	-0.38	**3.32**	**3.49**	-
BVP HR	0.36	**2.52**	1.53	**4.27**	**3.73**	-0.32	**3.37**	**-2.48**	0.13	**2.38**	0.74	**5.33**	**3.49**	0.28	**3.50**	**-2.20**
BVP HRV (Std)	-0.48	0.90	**-3.48**	0.67	**2.09**	1.24	0.83	-1.71	-0.01	1.17	**-3.09**	0.81	**2.10**	0.78	1.05	**2.13**
BVP HRV (Range)	-0.38	1.07	**-3.46**	0.73	**2.08**	1.45	0.57	-1.13	0.07	1.27	**-3.04**	0.75	-1.95	1.04	0.89	-1.70
BVP HR Accel. (RMS)	0.34	**2.52**	1.62	**4.45**	**3.81**	-0.08	**3.33**	**-2.11**	0.17	**2.35**	0.83	**5.40**	**3.54**	0.45	**3.47**	1.78
BVP HR Accel. (Std)	-0.44	0.69	**-2.71**	0.90	-1.68	1.02	1.25	-1.38	0.33	0.78	**-2.41**	1.45	-1.38	0.42	1.73	-1.79
BVP Volatility	0.23	-1.14	-1.24	-1.84	**-3.54**	-0.11	**-2.46**	**-2.94**	0.54	-0.98	-1.49	-1.88	**-3.42**	-0.04	**-2.70**	**-2.83**

Bolded statistics are significant at the 5% level.

The left panel contains t statistics for pre-event features and the right panel contains t statistics for post-event features, both tested against the same set of control features. See Table 3.1 for abbreviations.

Table C.2: t Statistics for two-sided hypothesis tests of the difference in mean autonomic responses during pre- and post-event intervals versus no-event intervals (controls) for each of the components of the physiological features (rows) for DEV Price events.

	DEV Price (20-sec Interval)															
	Pre-event Interval								Post-event Interval							
Physiological Features	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*
# of SCR responses	**2.40**	-0.68	1.42	0.71	0.78	0.40	-0.88	0	**2.23**	-0.76	1.42	0.71	-0.12	0.08	-1.20	-0.64
Ave. SCR amplitude	-1.95	-0.04	1.06	**-2.48**	-1.23	**-2.86**	1.88	-1.19	-1.45	0.33	1.06	**-2.41**	-1.30	**-2.66**	1.23	-1.37
ECG HR	-1.38	-1.95	**2.65**	-	**4.42**	0.09	**2.40**	-	-1.40	**2.04**	**2.44**	-	**3.90**	0.23	**2.98**	-
Local BVP ratio	0.56	**3.14**	-1.59	0.00	**-3.88**	0.61	**-2.75**	0.34	1.26	**2.97**	-1.83	-0.37	**-3.54**	0.41	**-3.09**	1.06
Global BVP ratio	0.54	**3.12**	-1.53	0.00	**-3.98**	0.62	**-2.84**	0.34	1.24	**2.96**	-1.72	-0.38	**-3.62**	0.42	**-3.18**	1.06
# of Temp jumps	-	-1.34	0.21	-1.24	**2.73**	-1.00	-0.78	-1.95	-	0	1.41	-1.24	-1.51	0.59	-0.78	**3.69**
Ave. SCR duration	**2.11**	-0.13	1.44	-1.13	0.21	-0.79	-0.14	-1.08	-1.90	-0.10	1.44	-1.36	0.30	-1.44	0.42	-1.62
SCR area	-1.89	0.39	1.04	**-2.83**	-0.02	**-2.15**	0.66	-1.39	-1.11	0.57	1.04	**-2.63**	-0.39	1.87	0.05	-1.50
Ave. SCR Δ	0.82	1.00	-	**5.13**	-1.45	1.10	**2.41**	-1.05	0.82	1.00	-	**4.94**	-1.57	0.56	-1.77	-1.21
ECG HRV (Std)	1.75	1.45	**-3.32**	-	-1.29	-1.55	0.67	-	**-2.24**	**-2.17**	**-3.00**	-	-1.36	**2.47**	0.10	-
ECG HR Accel. (RMS)	-0.73	-1.79	-1.75	-	**3.71**	-0.35	-1.88	-	-0.67	-1.84	-1.61	-	**3.37**	-0.41	**2.43**	-
ECG HR Accel. (Std)	1.50	1.50	**-3.81**	-	-1.19	-1.24	0.98	-	**-2.10**	**-2.26**	**-3.38**	-	-1.38	**2.26**	0.39	-
Range of Temp	0.73	-1.46	1.46	-0.21	**3.03**	**-2.56**	-1.36	-1.84	1.66	-1.00	1.58	-0.06	-1.79	**-2.63**	-0.61	**2.78**
Ave. EMG-Arm amplitude	-2.03	1.34	**3.45**	**-2.95**	**2.13**	-1.57	**-3.00**	0.60	-1.71	1.06	**3.56**	**-3.19**	-1.78	**2.10**	**-2.95**	0.33
EMG-Arm zero-crossings	**3.06**	**-2.06**	**4.89**	**-2.73**	**2.35**	**-2.35**	**-2.08**	0.61	**3.13**	**-2.31**	**4.82**	**-2.71**	-1.75	1.23	1.57	0.60
Ave. EMG-Back amplitude	0.15	-1.10	**12.13**	0.90	-1.87	1.73	-0.75	1.52	0.35	-1.05	**11.85**	1.04	**2.03**	1.14	-0.76	1.58
EMG-Back zero-crossings	-1.66	0.19	0.55	**7.14**	-0.77	**3.23**	**3.11**	-	-1.53	1.10	0.73	**6.91**	0.11	**3.16**	**3.08**	-
BVP HR	-1.61	**2.54**	-0.48	**5.24**	**5.45**	0.64	**2.99**	**-3.01**	-1.94	**2.52**	-1.71	**6.68**	**4.54**	1.17	**3.05**	**-2.44**
BVP HRV (Std)	-0.13	1.17	**-3.97**	1.15	-1.63	0.63	0.80	**2.41**	0.48	1.54	**-3.79**	1.58	-1.57	0.82	0.30	**2.18**
BVP HRV (Range)	-0.29	1.22	**-3.02**	0.99	-1.61	-0.22	0.87	-1.54	0.27	1.43	**-3.03**	0.90	-1.47	0.65	0.47	-1.41
BVP HR Accel. (RMS)	-1.71	**2.34**	-0.34	**5.60**	**5.52**	0.97	**3.01**	**-2.82**	-2.01	**2.30**	-1.55	**6.64**	**4.61**	1.54	**3.15**	**-2.15**
BVP HR Accel. (Std)	-0.42	0.83	**-3.00**	1.69	-1.80	0.50	0.73	-1.85	0.52	0.97	**-2.66**	**-2.29**	-1.17	0.68	0.53	-1.54
BVP Volatility	0.56	**2.89**	-1.41	-0.87	**-3.96**	0.19	**-2.55**	**-2.05**	1.25	**2.70**	-1.62	-1.38	**-3.57**	0.19	**-2.92**	**-2.61**

Bolded statistics are significant at the 5% level.

The left panel contains t statistics for pre-event features and the right panel contains t statistics for post-event features, both tested against the same set of control features. See Table 3.1 for abbreviations.

Table C.3: t Statistics for two-sided hypothesis tests of the difference in mean autonomic responses during pre- and post-event intervals versus no-event intervals (controls) for each of the components of the physiological features (rows) for DEV Price events.

	DEV Price (30-sec Interval)															
	Pre-event Interval								Post-event Interval							
Physiological Features	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*
# of SCR responses	-0.60	-0.38	**-2.15**	1.23	1.56	0.12	0.33	-0.59	-0.56	-0.76	**-2.15**	0.92	1.16	0	-0.57	-1.24
Ave. SCR amplitude	0.17	1.52	**-2.03**	1.45	-0.65	**-2.38**	**-2.89**	-1.44	0.64	**-2.17**	**-2.03**	1.08	-0.37	1.92	**-2.21**	-1.19
ECG HR	-0.69	**2.55**	-1.69	-	**4.04**	1.36	-1.23	-	-0.71	**3.07**	-1.40	-	**3.17**	1.60	-1.66	-
Local BVP ratio	0.08	**3.34**	-1.37	**2.61**	**-3.72**	0.58	**-3.31**	-0.25	0.92	**3.36**	-1.64	**3.35**	**-3.46**	0.73	**-3.67**	0.81
Global BVP ratio	0.08	**3.34**	-1.31	**2.62**	**-3.69**	0.58	**-3.37**	-0.25	0.92	**3.37**	-1.55	**3.37**	**-3.43**	0.72	**-3.72**	0.81
# of Temp jumps	-	-1.30	0.83	**2.40**	**2.58**	-1.45	0.47	-1.34	-	0	**-2.07**	**2.40**	-1.46	0.47	0.23	**3.01**
Ave. SCR duration	-1.29	1.69	**-2.82**	1.65	0.05	-1.02	-0.30	-1.19	-0.72	1.73	**-2.82**	**-2.09**	0.58	-1.59	0.82	-1.52
SCR area	0.06	1.77	1.90	**-2.60**	1.05	1.43	**-2.36**	-1.92	0.67	1.81	1.90	**-2.14**	1.12	1.38	1.43	-1.59
Ave. SCR Δ	0.99	-1.00	-	-1.92	-1.32	0.87	-1.56	-1.53	0.34	-1.00	-	-1.63	**2.11**	0.26	-0.83	-1.61
ECG HRV (Std)	1.29	1.70	**-3.13**	-	-0.60	-1.05	1.61	-	**-2.66**	1.85	**-2.74**	-	-0.06	-1.64	0.74	-
ECG HR Accel. (RMS)	-0.03	-1.52	-0.58	-	**2.86**	0.85	-0.60	-	0.26	-1.90	-0.39	-	**2.12**	0.97	-1.08	-
ECG HR Accel. (Std)	0.88	1.80	**-3.39**	-	-0.74	-1.22	1.76	-	**-2.48**	1.86	**-2.99**	-	-0.37	**2.03**	0.83	-
Range of Temp	0.72	-1.74	1.40	**2.03**	-0.43	**-2.18**	-1.48	-1.05	**-2.35**	-0.99	1.46	-1.76	0.22	**-2.24**	-0.76	**2.09**
Ave. EMG-Arm amplitude	-1.70	0.78	**2.68**	**-3.58**	-0.81	-0.51	**-2.20**	-0.82	-1.36	0.25	**2.92**	**-3.57**	-0.35	-0.91	**-2.06**	-1.12
EMG-Arm zero-crossings	-1.62	1.74	**3.15**	**-2.29**	-1.46	1.51	1.56	-0.22	-1.76	**-2.12**	**3.36**	1.96	-0.48	1.31	0.91	-0.11
Ave. EMG-Back amplitude	-0.71	0.33	**11.91**	-1.82	**3.11**	1.05	-1.11	0.48	-0.48	0.70	**11.93**	-1.08	**2.78**	0.57	-1.26	1.21
EMG-Back zero-crossings	-1.23	-1.32	0.48	**11.13**	**2.81**	**3.91**	**3.55**	-	-1.34	-1.09	0.68	**11.37**	**2.14**	**4.04**	**3.55**	-
BVP HR	-1.81	**4.00**	0.28	**4.79**	**8.01**	0.77	**4.59**	**-2.19**	**2.23**	**4.47**	-0.72	**6.85**	**6.53**	0.96	**4.36**	1.64
BVP HRV (Std)	-0.23	1.77	**-3.05**	0.24	-1.06	0.90	1.34	**2.49**	0.95	**-2.15**	**-3.05**	0.66	-0.44	1.58	0.19	**2.22**
BVP HRV (Range)	-0.02	1.47	**-2.79**	0.86	**2.08**	0.54	0.63	-1.79	0.93	1.89	**-2.69**	0.54	-1.47	1.32	-0.51	-1.59
BVP HR Accel. (RMS)	-1.86	**3.55**	0.62	**5.66**	**7.80**	1.06	**4.44**	2.00	**2.15**	**3.99**	-0.36	**7.30**	**6.39**	1.36	**4.37**	1.36
BVP HR Accel. (Std)	-0.86	1.11	**-2.13**	0.51	**2.36**	0.61	1.56	**2.36**	0.66	1.49	1.98	1.20	-1.18	1.35	0.71	-1.91
BVP Volatility	0.10	**3.10**	-1.24	**2.83**	**-3.65**	0.08	**-3.22**	1.51	0.94	**3.05**	-1.49	**3.91**	**-3.38**	0.43	**-3.55**	**-2.56**

Bolded statistics are significant at the 5% level.

The left panel contains t statistics for pre-event features and the right panel contains t statistics for post-event features, both tested against the same set of control features. See Table 3.1 for abbreviations.

Table C.4: t Statistics for two-sided hypothesis tests of the difference in mean autonomic responses during pre- and post-event intervals versus no-event intervals (controls) for each of the components of the physiological features (rows) for DEV Price events.

	DEV Price (40-sec Interval)															
	Pre-event Interval								Post-event Interval							
Physiological Features	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*
# of SCR responses	-1.98	-0.45	**-2.10**	**-2.70**	0.97	1.14	-1.35	-1.36	-1.79	-0.93	**-2.10**	**-2.52**	0.63	0.49	**2.19**	**2.09**
Ave. SCR amplitude	-0.12	0.15	1.34	**-2.06**	-1.51	**-5.19**	**-2.70**	**2.38**	-0.17	1.08	1.34	1.32	-1.16	**-4.65**	1.54	**2.29**
ECG HR	-0.03	**2.53**	**3.01**	-	-1.65	1.29	**4.86**	-	-0.12	**3.25**	**2.64**	-	-0.96	1.87	**5.55**	-
Local BVP ratio	-0.53	**2.22**	**2.86**	-1.75	**-3.61**	**-2.45**	**-5.08**	-0.16	0.35	**2.47**	**3.30**	**2.56**	**-3.35**	1.50	**-5.72**	0.88
Global BVP ratio	-0.54	**2.21**	**2.76**	-1.77	**-3.59**	**-2.46**	**-5.24**	-0.16	0.35	**2.46**	**3.17**	**2.60**	**-3.33**	1.50	**-5.86**	0.88
# of Temp jumps	-	-0.55	1.78	-1.23	**2.26**	-1.99	-1.09	**2.60**	-	1.18	**-3.66**	-1.23	-1.23	-1.23	-1.36	**4.34**
Ave. SCR duration	-1.40	1.19	**-2.39**	-1.07	-1.43	-0.42	-0.15	**2.75**	-1.71	1.05	**-2.39**	-1.26	-0.48	-0.95	1.24	**3.28**
SCR area	-1.10	0.64	1.84	**-2.88**	0.59	**-3.71**	1.38	-1.80	-0.28	0.80	1.84	**-2.33**	0.75	**-3.38**	0.51	-1.61
Ave. SCR Δ	0.62	1.12	-	**3.56**	-1.27	0.61	**2.14**	**2.23**	-0.15	1.12	-	**2.70**	**2.19**	-0.08	-1.09	-1.83
ECG HRV (Std)	1.72	1.60	**-4.52**	-	0.49	**2.22**	1.62	-	**-3.83**	1.78	**-4.19**	-	1.29	-1.94	0.34	-
ECG HR Accel. (RMS)	0.83	-1.90	-0.90	-	-1.02	0.54	**3.06**	-	1.42	**2.50**	-0.72	-	-0.34	1.03	**3.77**	-
ECG HR Accel. (Std)	1.17	1.45	**-5.34**	-	0.40	**2.20**	1.90	-	**-3.79**	1.50	**-4.87**	-	0.93	-1.94	0.61	-
Range of Temp	-0.05	-1.87	**-2.57**	-1.35	0.16	**-2.14**	**2.90**	0.12	1.16	-0.65	**-2.69**	-1.02	0.84	**-2.26**	**2.37**	-0.91
Ave. EMG-Arm amplitude	**2.11**	1.72	**3.46**	**-6.65**	-0.37	-1.40	**-2.71**	-0.24	-1.80	1.16	**3.95**	**-6.49**	0.42	-1.83	**-2.27**	-0.50
EMG-Arm zero-crossings	-2.02	**-3.26**	**4.14**	**-5.08**	0.06	1.21	1.66	-0.14	**2.24**	**-4.05**	**4.28**	**-4.47**	1.12	0.78	0.63	0.05
Ave. EMG-Back amplitude	-0.78	0.24	**11.64**	-1.08	-0.29	**-2.63**	**2.34**	1.08	-0.54	0.70	**12.31**	0.04	0.05	1.20	**2.43**	**-2.54**
EMG-Back zero-crossings	-0.50	-0.77	**2.12**	**8.11**	**2.97**	**4.04**	**3.55**	-	-0.90	0.53	-1.09	**8.48**	**2.15**	**4.13**	**3.55**	-
BVP HR	-1.51	**4.37**	-0.55	**5.56**	**6.88**	-0.28	**7.06**	**-3.19**	-1.62	**5.03**	-1.59	**8.09**	**5.67**	0.27	**6.69**	**-2.80**
BVP HRV (Std)	1.03	**-2.05**	**-3.66**	0.66	-0.62	**-3.47**	1.69	**3.54**	1.86	**-2.53**	**-3.87**	1.74	-0.09	**-3.27**	0.23	**3.33**
BVP HRV (Range)	0.99	1.37	**-3.11**	1.19	-1.89	**-2.19**	1.49	**2.21**	**-2.11**	2.00	**-3.12**	0.88	-1.25	1.89	0.19	**2.17**
BVP HR Accel. (RMS)	-1.37	**3.90**	-0.27	**6.43**	**6.76**	0.31	**7.34**	**-2.93**	-1.40	**4.54**	-1.26	**8.52**	**5.60**	0.90	**7.16**	**-2.44**
BVP HR Accel. (Std)	0.72	1.26	**-3.50**	1.03	**2.09**	**-2.54**	1.90	**2.94**	1.80	1.87	**-3.35**	**-2.65**	-1.10	**-2.75**	0.71	**2.59**
BVP Volatility	-0.55	**2.16**	**2.58**	**2.97**	**-3.57**	1.85	**-4.88**	1.63	0.33	**2.26**	**3.01**	**4.30**	**-3.31**	1.25	**-5.33**	**-2.56**

Bolded statistics are significant at the 5% level.

The left panel contains t statistics for pre-event features and the right panel contains t statistics for post-event features, both tested against the same set of control features. See Table 3.1 for abbreviations.

Table C.5: t Statistics for two-sided hypothesis tests of the difference in mean autonomic responses during pre- and post-event intervals versus no-event intervals (controls) for each of the components of the physiological features (rows) for DEV Price events.

	DEV Price (50-sec Interval)															
	Pre-event Interval								**Post-event Interval**							
Physiological Features	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*
# of SCR responses	-1.67	-1.30	**-2.78**	1.83	0.86	0.71	-0.53	-0.55	-1.81	-2.02	**-2.78**	**-2.07**	0.41	-0.15	-1.45	-1.04
Ave. SCR amplitude	0.37	-0.32	1.72	**-3.61**	-0.94	**-3.95**	**-2.19**	-1.03	0.49	0.93	1.72	**-3.17**	-0.78	**-3.60**	1.19	-0.52
ECG HR	-0.68	**3.70**	**2.85**	-	-1.48	0.35	**2.65**	-	-0.91	**4.74**	**2.32**	-	-0.67	1.24	**3.36**	-
Local BVP ratio	1.18	**3.21**	**2.66**	**2.13**	**-4.27**	**-2.11**	**-3.27**	1.20	**-2.84**	**3.70**	**3.34**	**2.81**	**-3.61**	0.93	**-3.56**	**-2.21**
Global BVP ratio	1.18	**3.24**	**2.50**	**2.15**	**-4.26**	**-2.11**	**-3.38**	1.20	**-2.84**	**3.73**	**3.17**	**2.85**	**-3.59**	0.93	**-3.66**	**-2.20**
# of Temp jumps	-	**3.02**	0.37	**2.08**	-2.02	-0.82	-1.00	**2.33**	-	-0.86	**-2.60**	-1.79	-0.73	-0.26	-1.18	**4.10**
Ave. SCR duration	-1.75	**-2.87**	**-2.99**	-1.73	-1.07	-0.36	-1.61	-1.65	**2.22**	**-2.42**	**-2.99**	**2.17**	-0.13	-0.43	-0.35	-1.59
SCR area	-0.51	0.64	1.97	**-4.52**	0.66	**-3.29**	0.95	-1.48	0.08	0.57	1.97	**-4.03**	0.70	**-2.66**	0.06	-0.68
Ave. SCR Δ	1.41	-	-	**4.92**	-1.40	1.72	-1.12	-1.74	1.06	-	-	**3.52**	**2.63**	0.77	0.55	-1.16
ECG HRV (Std)	1.29	1.94	**-4.09**	-	1.13	**3.37**	**-3.64**	-	**-3.09**	**-2.30**	**-3.68**	-	1.88	**2.63**	1.95	-
ECG HR Accel. (RMS)	-0.03	**3.45**	-0.90	-	-0.58	-0.53	-0.97	-	0.20	**4.41**	-0.58	-	0.20	0.30	-1.80	-
ECG HR Accel. (Std)	1.03	1.95	**-4.83**	-	0.99	**3.49**	**-3.71**	-	**-3.16**	1.94	**-4.15**	-	1.48	**2.43**	**-2.14**	-
Range of Temp	0.88	**2.25**	-0.30	-0.67	-0.21	0.83	**2.29**	-0.69	**-2.35**	-0.87	0.63	-0.50	0.70	1.20	-2.02	**2.04**
Ave. EMG-Arm amplitude	**3.54**	**-2.36**	**2.26**	**-5.84**	-1.62	-1.38	0.88	0.60	**3.38**	1.89	**2.69**	**-6.15**	-0.43	**2.03**	0.22	0.22
EMG-Arm zero-crossings	**3.58**	**-2.45**	**2.72**	**-3.97**	-1.32	1.54	0.91	0.39	**3.85**	**-3.39**	**3.07**	**-3.90**	0.28	0.87	-0.05	0.47
Ave. EMG-Back amplitude	0.36	-0.10	**8.15**	-1.66	-0.95	1.17	**3.38**	0.86	0.55	0.46	**8.79**	-0.22	-0.23	0.15	**2.80**	**-2.25**
EMG-Back zero-crossings	-1.82	-1.17	**2.70**	**19.32**	**3.11**	**4.16**	**2.82**	-	-1.78	-0.62	-1.45	**19.49**	**2.06**	**4.19**	**2.81**	-
BVP HR	**3.62**	**5.40**	0.11	**5.65**	**6.51**	-0.67	**6.33**	**-2.71**	**3.57**	**6.44**	-0.53	**8.71**	**5.31**	0.64	**5.86**	**-2.16**
BVP HRV (Std)	-0.24	1.40	**-3.18**	-0.37	-1.66	**-2.93**	**-2.76**	**2.36**	1.12	1.94	**-3.68**	1.05	-1.37	**-2.22**	0.91	**2.23**
BVP HRV (Range)	-0.70	0.35	**-2.14**	0.90	**4.03**	1.58	**-2.64**	-1.47	0.38	1.05	**-2.41**	0.36	**4.28**	1.12	1.17	-1.40
BVP HR Accel. (RMS)	**3.65**	**5.02**	0.28	**6.73**	**6.50**	-0.05	**6.05**	**-2.58**	**3.53**	**5.96**	-0.33	**9.27**	**5.33**	1.26	**5.80**	1.95
BVP HR Accel. (Std)	-0.75	0.31	**-2.82**	0.29	**2.59**	1.56	**-2.59**	-2.02	0.81	0.91	**-2.83**	**-2.24**	-1.83	1.23	0.98	-1.41
BVP Volatility	1.15	**3.01**	**2.30**	**2.65**	**-4.21**	1.69	**-3.20**	**-2.49**	**-2.82**	**3.26**	**2.95**	**3.80**	**-3.57**	0.81	**-3.40**	**-3.22**

Bolded statistics are significant at the 5% level.

The left panel contains t statistics for pre-event features and the right panel contains t statistics for post-event features, both tested against the same set of control features. See Table 3.1 for abbreviations.

Table C.6: t Statistics for two-sided hypothesis tests of the difference in mean autonomic responses during pre- and post-event intervals versus no-event intervals (controls) for each of the components of the physiological features (rows) for DEV Price events.

	DEV Price (60-sec Interval)															
	Pre-event Interval								Post-event Interval							
Physiological Features	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*
# of SCR responses	-0.59	1.19	**-2.39**	1.55	0.83	1.25	-0.57	-0.57	-0.76	0.57	**-2.39**	1.89	0.18	0.14	-2.00	-0.98
Ave. SCR amplitude	-0.12	-1.01	**-2.06**	**-3.09**	-1.34	**-5.07**	**-3.20**	**2.57**	0.40	0.20	**-2.06**	**-2.67**	-1.13	**-4.49**	**-2.51**	-1.94
ECG HR	0.08	**2.37**	-1.73	-	-1.91	1.05	**3.66**	-	0.10	**3.50**	-1.53	-	-1.10	1.90	**4.52**	-
Local BVP ratio	-0.39	**3.48**	**2.93**	**3.14**	**-3.88**	1.94	**-4.08**	0.87	0.68	**4.32**	**3.85**	**3.93**	**-3.35**	1.12	**-4.17**	1.66
Global BVP ratio	-0.39	**3.51**	**2.81**	**3.16**	**-3.83**	1.94	**-4.21**	0.89	0.68	**4.35**	**3.72**	**3.95**	**-3.30**	1.12	**-4.30**	1.66
# of Temp jumps	-	**3.05**	0	**2.12**	**2.97**	-0.41	0	**2.28**	-	-0.79	**-2.07**	-1.56	-1.57	1.05	-0.19	**4.05**
Ave. SCR duration	-0.20	0.63	**-3.35**	0.50	**2.04**	**2.15**	-1.53	**2.83**	-0.61	0.41	**-3.35**	-0.21	-0.72	**2.05**	-0.04	**2.53**
SCR area	0.07	1.10	**-2.03**	**-2.90**	0.60	**-3.39**	1.71	**2.35**	0.92	0.76	**-2.03**	**-2.63**	0.72	**-2.43**	0.96	-1.65
Ave. SCR Δ	1.61	0.91	-	**4.04**	-1.28	1.52	**2.57**	-1.20	1.51	0.91	-	**2.46**	-1.68	0.59	-0.53	-0.77
ECG HRV (Std)	0.85	1.08	**-3.76**	-	0.56	**3.32**	**-3.82**	-	**-2.45**	1.28	**-3.42**	-	1.25	**2.38**	1.74	-
ECG HR Accel. (RMS)	0.54	-2.01	-0.59	-	-1.12	0.16	-1.90	-	0.87	**3.05**	-0.48	-	-0.36	0.96	**2.89**	-
ECG HR Accel. (Std)	0.69	1.25	**-4.92**	-	0.32	**3.62**	**-3.92**	-	**-2.51**	1.28	**-4.13**	-	0.68	**2.54**	1.91	-
Range of Temp	0.64	**2.47**	0.83	**2.50**	-0.31	1.30	-1.58	-0.73	**-2.73**	-0.59	1.66	**2.35**	1.22	**-2.24**	-1.55	**2.20**
Ave. EMG-Arm amplitude	**2.36**	0.89	**2.84**	**-5.75**	-0.52	-1.62	**-3.27**	0.15	**2.22**	0.45	**2.85**	**-6.64**	0.47	**2.24**	**-2.32**	-0.47
EMG-Arm zero-crossings	**2.35**	**-2.56**	**2.76**	**-3.82**	-0.41	0.73	1.77	0.43	**2.73**	**-3.12**	**3.11**	**-3.98**	0.98	0.25	0.20	0.10
Ave. EMG-Back amplitude	-0.63	-0.05	**8.67**	-1.45	-0.02	1.54	**3.80**	0.56	-0.49	0.59	**9.93**	0.35	0.57	0.60	**2.73**	**-2.09**
EMG-Back zero-crossings	-1.16	-1.24	0.82	**8.70**	**3.22**	**4.13**	**3.54**	-	-1.28	-0.91	0.85	**9.05**	**2.06**	**4.22**	**3.54**	-
BVP HR	-1.62	**4.57**	0.68	**5.95**	**6.77**	0.13	**6.86**	**-3.43**	-1.58	**5.85**	-0.03	**9.82**	**5.51**	0.80	**6.53**	**-2.94**
BVP HRV (Std)	-1.11	1.46	**-2.92**	-0.95	-1.57	**-3.81**	**-4.69**	**2.82**	0.49	**-2.05**	**-3.17**	1.58	-1.21	**-3.34**	**-2.45**	**2.79**
BVP HRV (Range)	-0.36	0.72	**-2.92**	0.13	**3.78**	1.93	**-3.82**	**2.11**	0.92	1.48	**-3.13**	-0.39	**3.30**	1.86	**-2.20**	**2.04**
BVP HR Accel. (RMS)	-1.64	**3.79**	0.99	**6.96**	**6.77**	0.80	**6.37**	**-3.30**	-1.52	**4.88**	0.42	**9.91**	**5.54**	1.51	**6.27**	**-2.72**
BVP HR Accel. (Std)	-1.69	1.14	**-2.63**	-0.58	**3.11**	**-2.20**	**-3.97**	**2.74**	0.30	1.77	**-2.65**	**-2.36**	**2.09**	**-2.21**	1.99	-2.01
BVP Volatility	-0.40	**3.32**	**2.75**	**3.33**	**-3.95**	1.48	**-3.80**	**-2.03**	0.69	**3.80**	**3.64**	**4.45**	**-3.40**	0.95	**-3.81**	**-2.45**

Bolded statistics are significant at the 5% level.

The left panel contains t statistics for pre-event features and the right panel contains t statistics for post-event features, both tested against the same set of control features. See Table 3.1 for abbreviations.

Table C.7: t Statistics for two-sided hypothesis tests of the difference in mean autonomic responses during pre- and post-event intervals versus no-event intervals (controls) for each of the components of the physiological features (rows) for DEV Price events.

	DEV Price (70-sec Interval)															
	Pre-event Interval								**Post-event Interval**							
Physiological Features	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*
# of SCR responses	-1.40	-0.80	**-2.03**	**-2.26**	**-2.32**	0.91	-0.08	-1.25	-1.92	-1.75	**-2.03**	**-2.86**	1.26	0.39	-1.18	-1.63
Ave. SCR amplitude	-0.84	0.52	1.52	**-3.90**	-0.18	**-4.57**	**-4.00**	**2.72**	-0.07	1.98	1.52	**-2.52**	-0.15	**-4.24**	**-3.22**	-1.68
ECG HR	-1.14	**3.77**	**2.87**	-	-1.00	1.04	-0.86	-	-0.63	**5.35**	**2.62**	-	0.05	**-2.29**	-1.47	-
Local BVP ratio	-0.26	**2.44**	**3.50**	-0.41	**-3.68**	0.23	**-2.23**	0.52	0.68	**3.48**	**4.92**	-1.25	**-3.06**	0.59	**-2.29**	1.95
Global BVP ratio	-0.27	**2.47**	**3.35**	-0.43	**-3.63**	0.23	**-2.30**	0.52	0.68	**3.52**	**4.74**	-1.29	**-3.02**	0.59	**-2.36**	1.96
# of Temp jumps	-	-1.75	-0.21	-1.36	-1.58	-0.35	-0.18	-1.60	-	0.66	**-2.16**	-0.26	-0.50	1.23	-0.18	**3.32**
Ave. SCR duration	-1.55	**-2.24**	**-3.28**	-0.40	-0.64	**2.33**	-0.59	**4.85**	-1.71	**-2.08**	**-3.28**	-1.69	0.25	**2.35**	0.88	**3.84**
SCR area	-1.33	1.68	1.59	**-4.74**	1.64	**-3.27**	**-3.18**	**2.22**	-0.35	1.36	1.59	**-3.86**	1.54	**-2.96**	**-2.64**	-0.96
Ave. SCR Δ	0.44	1.00	-	**4.57**	-0.82	1.06	**2.60**	**3.62**	0.43	1.00	-	**2.60**	**3.03**	-0.08	-0.28	**2.93**
ECG HRV (Std)	-1.15	0.95	**-4.58**	-	1.55	**3.91**	**-3.40**	-	1.84	**-2.23**	**-4.12**	-	**-2.46**	**2.53**	1.92	-
ECG HR Accel. (RMS)	-1.70	**3.66**	-1.50	-	-0.05	-0.11	0.22	-	-0.01	**5.06**	-1.35	-	0.90	1.21	-0.48	-
ECG HR Accel. (Std)	-1.25	1.44	**-4.89**	-	1.44	**3.84**	**-3.69**	-	1.97	**-2.05**	**-3.85**	-	1.77	**2.47**	2.00	-
Range of Temp	0.19	-1.98	-0.09	**2.36**	0.20	1.33	**2.71**	0.28	**-2.72**	0.21	1.33	**2.20**	1.50	**-2.48**	**2.40**	-0.82
Ave. EMG-Arm amplitude	**2.75**	0.30	**3.75**	**-4.12**	-0.10	**2.21**	**-4.31**	0.22	**2.69**	-0.05	**3.64**	**-5.05**	1.18	**2.54**	**-2.87**	-0.65
EMG-Arm zero-crossings	**2.29**	**-3.33**	**3.38**	**-3.45**	-0.48	0.69	**-2.88**	0.76	**2.88**	**-4.54**	**3.63**	**-3.71**	1.90	0.41	1.61	-0.05
Ave. EMG-Back amplitude	0.87	-1.97	**8.21**	-0.35	0.31	0.09	-1.91	-0.02	0.73	-1.29	**9.98**	1.06	0.97	0.02	-1.11	1.93
EMG-Back zero-crossings	-1.61	-1.45	0.58	**24.10**	**3.36**	**4.14**	**3.40**	-	-1.61	-1.45	0.58	**20.16**	**2.06**	**4.26**	**3.39**	-
BVP HR	-1.28	**4.32**	1.19	**4.82**	**7.07**	0.84	**4.67**	**-3.16**	-1.30	**6.07**	0.15	**7.55**	**5.70**	1.24	**4.28**	**-2.42**
BVP HRV (Std)	-0.70	0.75	**-5.02**	**2.10**	-0.62	**-2.60**	**-4.06**	**2.87**	0.82	1.95	**-6.10**	0.53	-0.37	**-2.81**	1.50	**2.80**
BVP HRV (Range)	-1.53	-0.61	**-3.28**	-1.41	**4.05**	1.10	**-3.01**	-1.55	-0.02	0.84	**-3.84**	-1.73	**3.52**	1.35	0.04	-1.45
BVP HR Accel. (RMS)	-1.32	**4.09**	1.37	**5.84**	**7.07**	1.49	**4.34**	**-3.02**	-1.24	**5.51**	0.36	**8.10**	**5.74**	**-2.08**	**4.17**	**-2.12**
BVP HR Accel. (Std)	-1.17	-0.31	**-5.05**	-1.36	**2.16**	1.59	**-3.83**	**2.49**	0.35	0.81	**-5.12**	1.77	-1.40	**-2.30**	1.56	-1.64
BVP Volatility	-0.29	**2.31**	**3.15**	-1.10	**-3.58**	-0.35	**-2.19**	1.98	0.67	**2.97**	**4.50**	**2.45**	**-2.98**	0.25	**-2.23**	**-2.91**

Bolded statistics are significant at the 5% level.

The left panel contains t statistics for pre-event features and the right panel contains t statistics for post-event features, both tested against the same set of control features. See Table 3.1 for abbreviations.

Table C.8: t Statistics for two-sided hypothesis tests of the difference in mean autonomic responses during pre- and post-event intervals versus no-event intervals (controls) for each of the components of the physiological features (rows) for DEV Price events.

	DEV Price (80-sec Interval)															
	Pre-event Interval								Post-event Interval							
Physiological Features	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*
# of SCR responses	-1.23	-0.96	**-2.87**	1.51	1.71	1.43	0.37	-1.38	-1.81	**2.12**	**-2.87**	**-2.19**	1.12	0.81	-1.12	-1.55
Ave. SCR amplitude	0.62	0.56	**-2.30**	**-2.96**	-0.69	**-4.41**	**-3.08**	**3.18**	1.06	1.57	**-2.30**	**-2.13**	-1.00	**-4.10**	**-2.43**	-2.00
ECG HR	-1.37	**4.11**	-1.71	-	**3.91**	1.99	**2.20**	-	-0.50	**5.85**	-1.56	-	**2.20**	**-2.98**	**2.83**	-
Local BVP ratio	**-2.08**	**2.41**	**4.05**	-1.80	**-4.80**	1.59	**-2.98**	1.03	**-2.67**	**3.82**	**5.67**	**2.88**	**-3.96**	1.68	**-3.05**	**-2.16**
Global BVP ratio	**-2.07**	**2.45**	**3.82**	-1.86	**-4.77**	1.58	**-3.08**	1.03	**-2.66**	**3.87**	**5.40**	**2.95**	**-3.93**	1.68	**-3.15**	**-2.16**
# of Temp jumps	-	**3.43**	0.47	-1.27	**2.88**	-0.41	0.76	-1.18	-	0.19	**-2.70**	-0.70	-1.76	0.20	0.91	**2.94**
Ave. SCR duration	-1.10	1.78	**-3.60**	0.13	-1.27	**2.63**	**2.39**	**3.74**	-0.68	1.76	**-3.60**	-1.28	-0.28	**2.82**	-0.18	**3.28**
SCR area	-1.01	1.44	**-2.33**	**-2.95**	1.44	**-2.94**	1.50	**3.20**	0.50	0.84	**-2.33**	**-2.58**	1.31	**-2.47**	1.36	-1.89
Ave. SCR Δ	1.45	1.35	-	**3.10**	-1.14	1.28	**2.98**	-1.64	1.21	1.35	-	-0.94	-1.69	0.12	-0.36	-1.06
ECG HRV (Std)	1.09	0.69	**-5.34**	-	0.62	**2.16**	**-3.54**	-	**-2.85**	1.69	**-5.05**	-	1.99	-1.06	1.85	-
ECG HR Accel. (RMS)	-0.29	**3.97**	0.45	-	**2.31**	1.08	-1.03	-	0.83	**5.52**	0.49	-	-0.72	**-2.11**	-1.76	-
ECG HR Accel. (Std)	0.52	0.87	**-5.61**	-	0.62	**2.23**	**-3.78**	-	**-3.14**	1.17	**-4.59**	-	1.10	-1.33	1.97	-
Range of Temp	-1.94	**2.76**	0.58	**4.12**	-1.87	1.86	-1.12	-0.56	1.50	0.08	1.64	**3.69**	0.84	**-2.78**	-0.98	**2.05**
Ave. EMG-Arm amplitude	**2.86**	-0.13	**3.80**	**-4.27**	-0.64	**2.33**	**-3.67**	-0.75	**3.04**	-0.57	**3.34**	**-5.37**	0.40	**2.78**	**-2.24**	-1.72
EMG-Arm zero-crossings	**2.73**	**-2.03**	**3.76**	**-2.88**	-0.30	0	**-2.50**	-0.40	**3.67**	**-2.79**	**3.92**	**-3.13**	1.21	0	1.07	-1.36
Ave. EMG-Back amplitude	1.22	-0.88	**7.97**	-1.99	-0.65	0.88	**2.49**	0.33	0.52	-0.05	**10.01**	0.95	0.38	0.79	-1.53	2.00
EMG-Back zero-crossings	-1.11	-1.45	0.13	**25.12**	**3.40**	**4.17**	**3.54**	-	-1.08	-1.45	0.28	**24.22**	**2.06**	**4.26**	**3.53**	-
BVP HR	-2.03	**4.44**	0.43	**5.52**	**8.31**	0.75	**5.17**	**-2.92**	-2.01	**6.56**	-0.31	**9.02**	**6.78**	1.08	**4.83**	1.83
BVP HRV (Std)	**2.65**	0.88	**-3.59**	-1.24	-0.76	**-3.19**	**-3.98**	**2.29**	0.36	**-2.77**	**-3.93**	1.66	-0.88	**-3.32**	1.63	**2.22**
BVP HRV (Range)	**2.18**	-0.62	**-3.28**	0.05	**4.22**	0.98	**-3.25**	-1.57	-0.43	1.24	**-3.61**	-0.57	**4.36**	1.24	0.65	-1.46
BVP HR Accel. (RMS)	**2.11**	**4.39**	0.69	**6.64**	**8.24**	1.48	**4.91**	**-2.82**	-1.95	**6.09**	0.04	**9.61**	**6.79**	1.97	**4.76**	1.55
BVP HR Accel. (Std)	**4.25**	-0.55	**-3.21**	-0.50	**2.80**	1.84	**-3.56**	**2.85**	-1.88	0.93	**-3.22**	**-3.22**	**2.25**	**-2.51**	1.67	-1.69
BVP Volatility	**-2.06**	**2.09**	**3.86**	**2.56**	**-4.85**	0.99	**-2.99**	**-2.09**	**-2.68**	**3.06**	**5.43**	**4.09**	**-3.97**	1.35	**-2.99**	**-2.74**

Bolded statistics are significant at the 5% level.

The left panel contains t statistics for pre-event features and the right panel contains t statistics for post-event features, both tested against the same set of control features. See Table 3.1 for abbreviations.

Table C.9: t Statistics for two-sided hypothesis tests of the difference in mean autonomic responses during pre- and post-event intervals versus no-event intervals (controls) for each of the components of the physiological features (rows) for DEV Price events.

	DEV Price (90-sec Interval)															
	Pre-event Interval								**Post-event Interval**							
Physiological Features	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*
# of SCR responses	-1.69	**2.22**	**-2.20**	1.47	1.70	0.58	0.16	**2.50**	**2.59**	**4.27**	**-2.20**	**-2.38**	0.69	0.13	-1.73	**2.47**
Ave. SCR amplitude	-0.73	-0.86	1.95	**-4.03**	0.49	**-3.77**	**-4.33**	**2.77**	0.43	0.33	1.95	**-3.08**	0.09	**-3.65**	**-3.75**	-1.52
ECG HR	**3.97**	**4.95**	**3.26**	-	**2.83**	1.65	-1.86	-	**2.65**	**6.94**	**3.15**	-	-1.31	**-2.62**	**2.61**	-
Local BVP ratio	**-3.00**	**2.89**	-0.96	-1.36	**-4.07**	1.99	**-2.93**	-0.07	**-2.99**	**4.51**	**2.08**	**2.56**	**-3.25**	**-2.22**	**-2.97**	1.55
Global BVP ratio	**-2.99**	**2.91**	-0.80	-1.37	**-4.02**	1.99	**-3.05**	-0.07	**-2.98**	**4.54**	-1.92	**2.58**	**-3.21**	**-2.22**	**-3.08**	1.55
# of Temp jumps	-	**4.55**	0.48	-1.74	**2.83**	-0.90	0.45	-1.61	-	-0.17	**-2.40**	-1.57	-1.52	0	0.66	**3.39**
Ave. SCR duration	-1.80	1.81	**-3.04**	1.35	-1.49	**2.57**	-1.13	**3.41**	-0.93	1.75	**-3.04**	0.27	-0.38	**2.61**	1.50	**2.91**
SCR area	**3.28**	0.35	1.58	**-4.33**	**-2.32**	**-2.79**	**-3.06**	**2.77**	-1.63	-0.04	1.58	**-3.82**	**-2.13**	**-2.55**	**-2.96**	-1.56
Ave. SCR Δ	1.59	1.00	-	**5.06**	-0.81	0.84	**2.50**	**2.35**	1.61	1.00	-	**2.38**	**4.27**	-0.22	0.74	-2.00
ECG HRV (Std)	1.31	0.22	**-5.72**	-	0.51	**3.17**	**-4.23**	-	**-2.86**	1.42	**-5.86**	-	1.80	**2.26**	**-2.50**	-
ECG HR Accel. (RMS)	**2.98**	**4.61**	-0.91	-	-1.67	0.59	-0.47	-	-1.31	**6.29**	-0.83	-	-0.23	1.60	-1.32	-
ECG HR Accel. (Std)	0.54	0.58	**-6.54**	-	0.62	**3.15**	**-4.28**	-	**-2.57**	0.90	**-5.95**	-	0.92	**2.22**	**-2.50**	-
Range of Temp	-0.89	**4.29**	1.02	**2.10**	0.00	**-2.48**	-1.82	0.09	1.73	-0.44	**-2.25**	-1.26	1.90	**-3.71**	-1.92	-1.30
Ave. EMG-Arm amplitude	**2.70**	-0.60	**5.13**	**-5.54**	-0.09	-0.84	**-4.63**	-0.45	**3.36**	-0.90	**4.53**	**-7.17**	0.87	-1.52	1.85	-1.32
EMG-Arm zero-crossings	**2.44**	**-2.54**	**3.64**	**-3.80**	0.56	0.48	**-3.56**	0.13	**3.93**	**-2.97**	**3.76**	**-4.04**	2.02	0.65	1.47	-0.66
Ave. EMG-Back amplitude	**-2.90**	**2.37**	**8.72**	-0.89	1.16	**-2.17**	**2.83**	-0.59	1.63	-1.55	**11.73**	1.55	**-2.06**	2.01	-1.80	1.79
EMG-Back zero-crossings	-1.58	-1.45	-0.89	**11.23**	**3.37**	**4.24**	**3.14**	-	-1.56	-1.44	-0.41	**13.40**	**2.06**	**4.23**	**3.11**	-
BVP HR	**3.05**	**5.25**	0.30	**6.73**	**7.32**	-0.20	**5.43**	**-3.22**	**2.89**	**8.22**	-0.62	**12.09**	**5.97**	0.19	**5.02**	**-2.15**
BVP HRV (Std)	-1.77	0.24	**-4.01**	-0.20	0.95	**-4.07**	**-4.09**	**3.30**	-0.13	**-2.45**	**-4.55**	**-3.31**	0.70	**-4.17**	1.45	**3.19**
BVP HRV (Range)	-1.82	-0.80	**-3.00**	1.64	**3.97**	**-2.39**	**-2.83**	-1.27	-1.03	1.14	**-3.42**	0.77	**4.47**	**-2.40**	0.68	-1.14
BVP HR Accel. (RMS)	**3.13**	**5.58**	0.55	**7.86**	**7.24**	0.60	**5.16**	**-3.08**	**2.89**	**7.92**	-0.33	**12.35**	**5.93**	1.15	**4.96**	1.80
BVP HR Accel. (Std)	**2.86**	-0.46	**-2.98**	0.56	-1.61	**-2.22**	**-3.83**	**3.51**	-1.28	1.20	**-2.98**	**-4.83**	-1.58	**-2.87**	1.67	-1.90
BVP Volatility	**-3.01**	**2.73**	-0.67	**2.78**	**-4.03**	1.37	**-3.02**	1.38	**-2.99**	**3.96**	-1.74	**4.48**	**-3.20**	1.81	**-2.99**	**-2.37**

Bolded statistics are significant at the 5% level.

The left panel contains t statistics for pre-event features and the right panel contains t statistics for post-event features, both tested against the same set of control features. See Table 3.1 for abbreviations.

Table C.10: t Statistics for two-sided hypothesis tests of the difference in mean autonomic responses during pre- and post-event intervals versus no-event intervals (controls) for each of the components of the physiological features (rows) for DEV Price events.

	DEV Price (100-sec Interval)															
	Pre-event Interval								**Post-event Interval**							
Physiological Features	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*
# of SCR responses	-1.52	-0.79	**-2.35**	1.57	1.88	1.15	0.46	**2.48**	**2.76**	**2.47**	**-2.35**	**-2.60**	0.99	0.83	-1.39	**2.21**
Ave. SCR amplitude	0.84	0.04	**-2.22**	**-2.84**	0.44	**-4.93**	**-3.76**	**4.82**	1.61	1.53	**-2.22**	1.71	-0.02	**-4.83**	**-3.31**	**3.68**
ECG HR	**4.08**	**3.64**	**2.28**	-	**2.69**	1.87	-1.13	-	**2.52**	**6.07**	**2.03**	-	-1.13	**-2.75**	-1.79	-
Local BVP ratio	**-3.53**	**2.81**	**3.52**	**2.13**	**-4.61**	1.77	**-2.38**	0.17	**-3.50**	**5.36**	**5.09**	**3.35**	**-3.71**	**-2.44**	**-2.49**	**-2.29**
Global BVP ratio	**-3.52**	**2.83**	**3.36**	**2.14**	**-4.55**	1.77	**-2.47**	0.16	**-3.49**	**5.39**	**4.93**	**3.37**	**-3.66**	**-2.44**	**-2.59**	**-2.28**
# of Temp jumps	-	**3.55**	1.17	-1.38	**2.99**	-1.09	1.13	-0.81	-	-0.07	**-3.07**	-1.06	-1.29	-0.26	1.21	**2.52**
Ave. SCR duration	-1.39	2.01	**-3.64**	0.48	-1.77	**3.08**	-1.95	**4.49**	-0.46	1.72	**-3.64**	-0.64	-0.66	**3.07**	0.12	**4.11**
SCR area	**2.13**	1.55	1.82	**-4.61**	**-2.05**	**-3.57**	**-2.25**	**3.81**	-0.55	1.40	1.82	**-3.60**	2.00	**-3.49**	**-2.30**	**3.05**
Ave. SCR Δ	1.50	1.00	-	**5.94**	-1.00	0.01	**2.29**	-1.54	1.63	1.00	-	**2.55**	**4.52**	-1.13	0.66	-0.99
ECG HRV (Std)	1.95	0.45	**-5.05**	-	0.47	**2.79**	**-4.81**	-	**-3.05**	1.80	**-5.67**	-	1.85	-1.39	**-3.33**	-
ECG HR Accel. (RMS)	-1.91	**3.35**	-0.28	-	-1.67	0.95	0.34	-	-0.25	**5.44**	0.02	-	-0.20	1.88	-0.36	-
ECG HR Accel. (Std)	1.42	0.83	**-5.01**	-	0.56	**3.14**	**-4.66**	-	**-2.97**	1.20	**-5.03**	-	0.82	-1.52	**-3.19**	-
Range of Temp	-0.51	**4.08**	1.40	**3.47**	-0.72	1.76	-0.81	0.61	**-3.28**	0.00	**-2.72**	**2.37**	1.31	**-3.02**	-1.39	-0.61
Ave. EMG-Arm amplitude	**4.24**	0.26	**5.90**	**-4.13**	-1.05	-1.70	**-4.64**	-0.74	**5.29**	0.20	**5.53**	**-5.50**	-0.07	**2.07**	1.14	-1.36
EMG-Arm zero-crossings	**4.26**	**-2.61**	**4.63**	**-3.22**	-0.57	1.91	**-3.42**	-0.24	**6.28**	**-2.86**	**4.81**	**-3.56**	0.91	**-2.06**	1.55	-0.76
Ave. EMG-Back amplitude	**-2.91**	-1.73	**10.23**	-1.57	0.41	1.97	**4.47**	0.43	1.47	-0.64	**15.70**	1.42	1.28	**-2.05**	**2.94**	**-2.55**
EMG-Back zero-crossings	-1.32	-0.31	0.48	**10.59**	**3.38**	**4.28**	**3.55**	-	-1.22	0.30	0.69	**14.31**	**2.06**	**4.18**	**3.53**	-
BVP HR	**3.01**	**3.27**	0.16	**7.48**	**7.72**	-0.32	**6.04**	**-3.25**	**2.73**	**6.76**	-0.85	**13.11**	**6.18**	-0.21	**5.69**	1.98
BVP HRV (Std)	-0.25	0.42	**-3.79**	-0.80	-1.57	**-4.39**	**-4.65**	**3.61**	0.29	**-3.06**	**-4.10**	**-3.12**	**2.29**	**-4.75**	1.74	**3.52**
BVP HRV (Range)	-0.47	-0.84	**-3.30**	0.48	**6.05**	1.93	**-3.43**	**2.77**	-0.40	1.36	**-3.59**	-0.67	**6.99**	**-2.14**	1.30	**2.60**
BVP HR Accel. (RMS)	**3.17**	**3.57**	0.66	**8.58**	**7.68**	0.48	**5.67**	**-3.10**	**2.83**	**6.50**	-0.26	**12.96**	**6.20**	0.75	**5.50**	1.55
BVP HR Accel. (Std)	-1.06	-0.41	**-3.26**	-0.06	**3.62**	**-2.83**	**-4.24**	**3.87**	-0.34	1.52	**-3.25**	**-4.62**	**3.72**	**-3.68**	2.00	**2.06**
BVP Volatility	**-3.54**	**2.81**	**3.14**	**3.61**	**-4.56**	1.20	**-2.32**	1.43	**-3.49**	**4.96**	**4.66**	**5.44**	**-3.66**	**-2.05**	**-2.41**	**-3.06**

Bolded statistics are significant at the 5% level.

The left panel contains t statistics for pre-event features and the right panel contains t statistics for post-event features, both tested against the same set of control features. See Table 3.1 for abbreviations.

Table C.11: t Statistics for two-sided hypothesis tests of the difference in mean autonomic responses during pre- and post-event intervals versus no-event intervals (controls) for each of the components of the physiological features (rows) for DEV Price events.

	Buy Trades															
	Pre-event Interval								Post-event Interval							
Physiological Features	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*
# of SCR responses	**2.48**	1.04	-0.51	0.10	**3.56**	-0.99	-0.63	-1.36	-0.45	**-2.73**	-0.28	0.36	**2.47**	0.07	-1.91	-1.62
Ave. SCR amplitude	**2.42**	0.44	-1.71	-0.44	0.18	1.57	0.62	**2.12**	-0.90	-0.29	-1.73	-0.08	0.75	1.05	0.08	**2.42**
ECG HR	0.34	1.32	0.63	-	-0.28	0.41	-1.31	-	0.16	**-2.15**	0.31	-	**-2.03**	0.87	-0.27	-
Local BVP ratio	**-3.13**	-1.46	0.78	-0.15	1.08	1.42	**-2.70**	0.50	**-3.10**	-1.40	0.57	-1.31	1.06	1.60	**-2.06**	0.52
Global BVP ratio	**-3.12**	-1.51	0.73	-0.16	1.06	1.45	**-2.79**	0.48	**-3.10**	-1.40	0.55	-1.31	1.05	1.59	**-2.11**	0.51
# of Temp jumps	-	1.81	0.71	1.00	0.94	0.72	-1.32	-0.46	-	1.69	1.22	0	0	1.29	-0.39	0.59
Ave. SCR duration	**3.18**	-0.37	-0.08	0.64	0.03	0.58	-1.78	**2.95**	-1.01	-0.18	-0.24	**-2.36**	0.32	0.02	-1.77	**2.93**
SCR area	-1.58	0.10	-1.51	-0.01	-0.27	-0.50	-0.48	-1.48	-0.62	0.12	-1.44	1.42	1.23	0.31	-0.83	-1.75
Ave. SCR Δ	-0.95	1.09	-	1.51	1.01	-0.74	-0.86	**2.02**	-0.73	1.09	-	0.98	0.96	-1.68	-1.36	**2.33**
ECG HRV (Std)	0.74	1.61	0.42	-	-0.71	-0.31	0.74	-	1.69	1.48	1.28	-	1.76	**-2.07**	1.49	-
ECG HR Accel. (RMS)	0.56	1.50	0.62	-	-0.39	0.22	-0.80	-	0.73	**-2.26**	0.52	-	**-2.02**	1.13	0.17	-
ECG HR Accel. (Std)	1.12	1.41	0.02	-	-0.93	-0.13	1.42	-	1.75	1.39	0.74	-	1.78	1.95	1.49	-
Range of Temp	-0.20	-0.09	0.49	0.70	0.96	-1.49	-0.36	-1.49	0.67	0.38	0.18	-0.32	-0.07	-1.22	0.84	-1.35
Ave. EMG-Arm amplitude	**3.73**	-0.35	-1.47	-0.57	0.68	-1.24	0.53	-1.43	-1.76	-0.18	-1.10	-1.14	**-2.03**	-1.90	1.40	-0.41
EMG-Arm zero-crossings	**3.95**	**-3.00**	**3.20**	0.10	0.57	1.75	0.27	-1.27	**2.17**	**-2.80**	**2.58**	0.25	**-2.39**	-0.23	0.68	-0.47
Ave. EMG-Back amplitude	-0.56	1.45	-1.56	1.12	0.04	0.39	0.37	-0.38	-0.52	1.89	-1.73	1.47	0.07	1.28	0.35	-0.43
EMG-Back zero-crossings	-1.39	0.25	1.32	-0.37	-	-0.65	-0.10	-	-1.83	**-2.00**	1.38	-0.14	-	-0.20	0.01	-
BVP HR	-1.87	1.39	-0.44	0.73	0.36	0.17	**2.45**	-1.71	**3.08**	**-2.00**	0.10	0.90	1.01	-0.96	-1.47	**2.29**
BVP HRV (Std)	-1.76	0.87	0.05	-0.07	-0.29	0.68	1.11	-0.38	1.14	0.73	0.16	-1.80	0.89	1.40	0.71	-0.73
BVP HRV (Range)	-1.94	0.64	-0.23	0.19	0.04	0.60	1.32	-0.62	0.60	0.75	0.05	-1.45	1.34	1.19	0.86	-1.19
BVP HR Accel. (RMS)	**2.05**	1.43	-0.33	0.64	0.36	0.34	**2.37**	-1.67	**3.03**	**-2.03**	0.09	0.33	1.04	-0.78	-1.43	**2.24**
BVP HR Accel. (Std)	**2.07**	0.54	0.00	-0.91	-0.84	0.65	0.38	-0.43	0.27	0.31	-0.14	**2.00**	0.21	1.74	0.37	-0.21
BVP Volatility	**-3.12**	-1.80	0.62	0.44	1.05	1.44	**-2.54**	0.57	**-3.09**	-1.46	0.41	-0.05	1.02	1.43	1.97	0.41

Bolded statistics are significant at the 5% level.

The left panel contains t statistics for pre-event features and the right panel contains t statistics for post-event features, both tested against the same set of control features. See Table 3.1 for abbreviations.

Table C.12: t Statistics for two-sided hypothesis tests of the difference in mean autonomic responses during pre- and post-event intervals versus no-event intervals (controls) for each of the components of the physiological features (rows) for buy trades.

	Sell Trades															
	Pre-event Interval								Post-event Interval							
Physiological Features	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*
# of SCR responses	-0.88	1.45	0.78	0.05	-1.42	-0.93	0.52	-1.32	-0.44	**-3.11**	0	-0.73	-1.09	0.58	0.17	-0.81
Ave. SCR amplitude	-1.24	-0.98	-0.27	-0.23	-1.51	0.36	1.42	-1.91	-0.22	0.63	-0.22	-0.12	-1.50	0.94	-0.19	-1.10
ECG HR	0.45	-0.75	1.78	-	1.32	1.30	1.40	-	1.20	0.78	1.52	-	1.70	1.05	0.31	-
Local BVP ratio	1.74	0.96	-1.04	**-2.28**	-1.25	0.92	1.62	-0.56	**-2.25**	1.05	-0.15	**-2.91**	-0.49	**-2.36**	0.36	-0.30
Global BVP ratio	1.75	0.95	-1.05	**-2.26**	-1.21	0.91	1.63	-0.56	**-2.24**	0.99	-0.13	**-2.88**	-0.48	**-2.35**	0.31	-0.30
# of Temp jumps	-	**-3.76**	0	**2.28**	0.36	0	-0.31	1.42	-	**-3.55**	-0.37	-0.60	0.74	0.72	1.17	0
Ave. SCR duration	-0.81	-1.46	-0.36	0.70	-0.32	0.09	-1.03	-1.80	-0.50	0.09	-0.83	1.48	0.96	-0.00	-1.21	-0.99
SCR area	-0.02	-0.68	0.40	-0.57	-1.68	0.60	0.94	-1.86	0.32	0.28	-0.75	-0.55	-0.84	0.77	1.22	-1.10
Ave. SCR Δ	-1.52	1.21	-	0.75	0.14	-0.56	0.63	-0.30	-1.85	1.21	-	0.46	-0.44	-0.17	0.51	0.33
ECG HRV (Std)	-0.72	-0.54	0.78	-	-0.62	-0.39	**-2.01**	-	0.30	0.88	-0.08	-	0.60	-0.27	1.10	-
ECG HR Accel. (RMS)	0.25	-0.59	1.68	-	0.87	1.12	1.70	-	1.19	0.92	1.22	-	1.50	0.96	0.56	-
ECG HR Accel. (Std)	-1.13	-0.19	-0.39	-	-0.82	-0.49	**-2.24**	-	-0.02	1.04	-0.22	-	0.45	-0.11	0.91	-
Range of Temp	0.70	**-2.98**	0.68	-1.70	0.52	-0.24	0.82	-0.86	0.52	**-2.75**	0.57	-0.46	0.44	0.36	1.06	-1.24
Ave. EMG-Arm amplitude	-1.98	0.32	-1.04	-1.66	**2.32**	-0.27	**-2.02**	**3.03**	-1.15	0.17	-0.74	**2.12**	-1.18	0.86	-0.11	-1.15
EMG-Arm zero-crossings	**2.48**	**-3.13**	-1.69	-0.59	**2.22**	0.84	1.43	**2.60**	-1.96	**-2.72**	-1.94	-1.00	-0.82	0.67	-0.93	-0.83
Ave. EMG-Back amplitude	-0.10	0.70	-1.04	1.86	-1.84	0.15	0.09	-1.37	0.74	0.62	-1.33	1.92	-0.78	1.46	**2.06**	1.19
EMG-Back zero-crossings	0.08	1.23	0.89	-0.34	1.42	-1.87	-1.39	-	0.36	-0.39	0.49	1.03	1.42	-1.24	-1.24	-
BVP HR	-0.64	-1.52	1.05	-1.59	**-2.09**	0.47	-1.70	-0.51	0.42	0.22	0.93	-1.67	1.73	-1.20	-0.50	-1.35
BVP HRV (Std)	-1.95	0.65	1.20	1.31	-0.42	-0.52	0.89	-1.08	0.40	1.68	1.63	1.42	0.99	0.54	0.66	0.06
BVP HRV (Range)	**2.20**	1.12	1.38	0.77	-1.19	-0.32	0.82	-1.62	0.29	**-2.12**	1.37	0.90	0.73	0.07	0.55	-0.22
BVP HR Accel. (RMS)	-0.88	-1.40	1.17	-1.31	**-2.07**	0.40	-1.53	-0.52	0.45	0.41	1.04	-1.35	1.75	-1.18	-0.29	-1.23
BVP HR Accel. (Std)	-1.83	1.16	1.21	1.31	-1.61	-0.84	0.74	-1.70	0.06	**-2.02**	0.75	0.91	0.78	-0.16	0.43	-0.20
BVP Volatility	1.80	0.84	-1.18	**-2.64**	-1.15	1.05	1.40	-0.08	**-2.28**	0.98	-0.22	**-2.86**	-0.44	**-2.57**	0.39	-0.19

Bolded statistics are significant at the 5% level.

The left panel contains t statistics for pre-event features and the right panel contains t statistics for post-event features, both tested against the same set of control features. See Table 3.1 for abbreviations.

Table C.13: t Statistics for two-sided hypothesis tests of the difference in mean autonomic responses during pre- and post-event intervals versus no-event intervals (controls) for each of the components of the physiological features (rows) for sell trades.

	Buy vs. Sell Trades															
	Pre-event Interval								Post-event Interval							
Physiological Features	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*
# of SCR responses	-0.06	**-4.79**	-0.84	-0.49	-0.96	1.77	**-2.31**	-0.94	0.76	**-4.00**	-0.62	-0.05	-0.22	1.40	1.00	**2.36**
Ave. SCR amplitude	-0.70	1.24	-0.20	0.05	-1.52	1.70	0.05	-1.13	-0.04	-0.16	-1.34	0.30	-0.99	0.95	-0.34	**2.25**
ECG HR	1.16	1.90	0.67	-	1.23	1.30	**2.23**	-	-1.49	**-2.11**	0.57	-	0.89	0.68	-1.06	-
Local BVP ratio	-0.39	1.40	-0.44	1.55	1.59	**-3.62**	1.57	0.07	0.98	0.29	0.57	-0.38	1.62	**-2.87**	**-2.25**	0.49
Global BVP ratio	-0.39	1.33	-0.47	1.58	1.59	**-3.63**	1.57	0.08	0.98	0.27	0.53	-0.36	1.63	**-2.87**	**-2.36**	0.49
# of Temp jumps	-	**-2.01**	0.20	1.14	0.85	1.05	1.19	-1.47	-	1.28	1.45	0.29	-0.30	1.29	0.34	0
Ave. SCR duration	-1.00	0.83	-1.13	1.48	-0.45	-0.81	-0.40	-0.67	-0.11	-0.17	-0.79	**-2.29**	-0.68	-1.02	-0.63	**2.39**
SCR area	-0.12	1.78	-0.37	-0.52	-0.32	0.91	1.93	-1.31	0.19	0.97	-0.72	1.23	0.03	0.45	0.45	-1.74
Ave. SCR Δ	-1.47	0.41	-	-0.13	-0.83	**2.26**	1.29	0.00	0.00	0.33	-	0.39	-1.31	**3.31**	-0.02	-1.70
ECG HRV (Std)	-0.65	1.72	-0.37	-	0.45	0.79	-0.58	-	0.37	1.11	1.36	-	0.59	**-2.05**	0.93	-
ECG HR Accel. (RMS)	1.04	**-2.06**	0.42	-	1.07	1.35	**2.00**	-	-1.45	**-2.19**	0.74	-	0.82	0.93	-0.68	-
ECG HR Accel. (Std)	-0.85	1.27	-0.06	-	0.46	1.11	-0.54	-	0.23	0.76	1.18	-	0.71	**-2.11**	0.93	-
Range of Temp	1.09	**-2.30**	0.72	0.12	0.69	1.48	0.46	-1.70	1.98	1.53	0.01	-0.56	-0.12	-0.18	0.11	-1.17
Ave. EMG-Arm amplitude	-0.26	0.57	0.17	-1.90	0.28	0.41	-0.24	-1.09	-0.11	0.28	0.42	**2.17**	**-2.62**	-1.33	0.56	-1.39
EMG-Arm zero-crossings	-0.29	**-3.39**	0.05	-1.25	0.54	0.79	-1.05	-0.91	-0.18	**-3.22**	0.19	-1.11	**-2.35**	0	0.32	-1.15
Ave. EMG-Back amplitude	-0.73	0.80	-1.85	1.72	0.08	**-2.25**	-1.39	0.73	0.57	1.49	-0.85	1.85	0.15	**-2.27**	0.57	-0.77
EMG-Back zero-crossings	**-2.03**	-0.29	1.51	0.39	0.88	-0.25	-1.73	-	-1.05	1.56	1.07	0.39	1.00	0.43	-0.13	-
BVP HR	0.95	0.83	1.47	-0.08	1.11	**2.28**	-0.75	**2.55**	-1.57	1.34	0.51	1.18	0.38	**2.02**	-1.13	**2.12**
BVP HRV (Std)	1.05	**-3.10**	**-3.34**	0.20	**-2.10**	1.85	1.02	0.45	**-2.49**	1.95	1.19	-1.31	**-2.31**	**-1.99**	1.40	-0.70
BVP HRV (Range)	0.66	**-3.44**	**-3.11**	-0.01	**-2.17**	0.73	1.12	0.12	**-2.11**	**-2.15**	1.21	-0.75	**-2.43**	1.13	1.69	-1.27
BVP HR Accel. (RMS)	1.20	1.24	1.79	-0.00	1.19	**2.03**	-0.56	**2.47**	-1.27	1.65	0.55	0.74	0.47	-1.74	-0.94	**2.18**
BVP HR Accel. (Std)	0.62	**-3.06**	**-3.01**	0.02	**-2.75**	1.78	1.25	0.23	**-2.14**	1.49	1.34	-1.02	**-2.54**	**-2.60**	1.51	-0.58
BVP Volatility	-0.40	1.31	-0.74	1.42	1.52	**-3.82**	1.68	0.08	0.92	0.32	0.29	0.30	1.52	**-2.82**	**-2.19**	0.50

Bolded statistics are significant at the 5% level.

The left panel contains t statistics for pre-event features and the right panel contains t statistics for post-event features, for Buy vs. Sell Trades. See Table 3.1 for abbreviations.

Table C.14: t Statistics for two-sided hypothesis tests of the difference in mean autonomic responses during pre- and post-event intervals for each of the components of the physiological features (rows) for buy vs. sell trades.

	Instant Winning Trades															
	Pre-event Interval								Post-event Interval							
Physiological Features	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*
# of SCR responses	-2.00	-2.50	0.86	0.29	-	-0.13	-0.96	-	-0.71	0	0	0.64	-	-0.54	-0.64	-
Ave. SCR amplitude	-1.49	-0.62	0.25	1.19	-	0.47	-1.11	-	-0.37	0.87	0.00	0.75	-	-0.59	-0.97	-
ECG HR	1.12	-0.79	0.88	-	-	0.75	-0.45	-	1.16	0.92	-1.35	-	-	-0.23	0.55	-
Local BVP ratio	0.55	-0.16	0.10	-0.89	-	0.11	**-2.33**	-	0.52	-1.16	0.56	-0.82	-	-0.85	1.47	-
Global BVP ratio	0.56	-0.08	0.12	-0.90	-	0.08	**-2.38**	-	0.52	-1.18	0.57	-0.82	-	-0.87	1.45	-
# of Temp jumps	-	-2.00	-0.51	-1.00	-	-1.00	-0.88	-	-	-2.00	0	0	-	-1.00	0	-
Ave. SCR duration	-1.34	-1.52	0.83	-0.67	-	-0.08	-0.32	-	-0.46	0.29	0.15	0.15	-	-0.03	-0.04	-
SCR area	-1.13	-1.14	0.64	1.32	-	-0.08	-0.80	-	-0.16	0.92	-0.01	1.36	-	-1.47	-0.55	-
Ave. SCR Δ	-2.00	-	-	1.30	-	-1.99	-0.66	-	-1.00	-	-	1.76	-	-1.80	-1.13	-
ECG HRV (Std)	1.05	-0.59	1.77	-	-	-0.97	0.59	-	1.06	-0.64	-0.75	-	-	-0.42	**-2.07**	-
ECG HR Accel. (RMS)	1.17	-0.85	1.08	-	-	0.73	-0.06	-	1.21	0.88	-1.30	-	-	-0.25	1.14	-
ECG HR Accel. (Std)	1.02	-1.30	2.17	-	-	-1.23	0.85	-	1.04	-1.18	1.21	-	-	-0.68	1.93	-
Range of Temp	1.00	-0.11	-1.20	-0.24	-	-0.73	-1.19	-	1.00	-1.11	-0.88	-0.47	-	-1.05	-0.26	-
Ave. EMG-Arm amplitude	-1.75	-0.86	0.74	0.53	-	-0.90	-1.74	-	-1.83	-0.54	1.18	-0.05	-	-1.88	**2.48**	-
EMG-Arm zero-crossings	-1.50	-2.00	0.33	-0.39	-	-	**2.41**	-	-1.90	-	1.77	-0.78	-	**2.16**	**3.04**	-
Ave. EMG-Back amplitude	-0.57	-0.28	-0.90	0.19	-	0.18	**2.35**	-	-0.64	0.44	-0.89	-0.21	-	-0.36	**2.53**	-
EMG-Back zero-crossings	-1.00	1.00	-0.84	-	-	1.00	-0.59	-	-	1.00	-0.63	-	-	1.00	-0.41	-
BVP HR	-1.02	-0.41	1.47	0.59	-	0.64	-0.60	-	-1.01	1.24	1.28	0.59	-	0.91	-1.43	-
BVP HRV (Std)	-0.70	**2.83**	1.20	0.36	-	-0.40	-1.53	-	0.09	-0.89	0.66	0.30	-	-0.92	-1.13	-
BVP HRV (Range)	-0.78	**4.23**	1.18	0.62	-	-0.47	-0.93	-	-0.05	-1.56	0.27	0.72	-	-1.05	-0.82	-
BVP HR Accel. (RMS)	-1.07	-0.66	1.50	0.63	-	0.67	-0.91	-	-1.01	1.19	1.30	0.56	-	0.79	-1.51	-
BVP HR Accel. (Std)	-0.57	**4.58**	0.85	0.15	-	0.34	-1.12	-	-0.54	-1.80	0.34	-0.24	-	-0.95	-1.06	-
BVP Volatility	0.61	0.06	0.06	-1.05	-	-0.31	**-2.19**	-	0.56	-1.14	0.52	0.04	-	-1.04	1.37	-

Bolded statistics are significant at the 5% level.

The left panel contains t statistics for pre-event features and the right panel contains t statistics for post-event features, both tested against the same set of control features. See Table 3.1 for abbreviations.

Table C.15: t Statistics for two-sided hypothesis tests of the difference in mean autonomic responses during pre- and post-event intervals versus no-event intervals (controls) for each of the components of the physiological features (rows) for instant winning trades.

	Instant Losing Trades															
	Pre-event Interval								Post-event Interval							
Physiological Features	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*
# of SCR responses	-	1.26	1.00	0.30	-	1.69	0.54	-	-	1.12	-0.63	0.52	-	0.89	0.83	-
Ave. SCR amplitude	-	-1.00	1.00	-0.80	-	1.09	-0.84	-	-	-0.64	0.44	-0.73	-	-1.33	-0.46	-
ECG HR	-	-0.74	1.21	-	-	-0.27	0.30	-	-	0.09	0.91	-	-	-0.05	0.66	-
Local BVP ratio	-	0.13	-0.46	0.41	-	1.74	1.07	-	-	-0.11	-0.32	1.30	-	1.70	0.97	-
Global BVP ratio	-	0.08	-0.48	0.39	-	1.73	1.10	-	-	-0.10	-0.33	1.30	-	1.69	0.96	-
# of Temp jumps	-	0	0	0.61	-	-	-0.36	-	-	0	-0.62	-1.00	-	-	0.86	-
Ave. SCR duration	-	0.12	1.00	1.18	-	1.68	-1.49	-	-	0.43	-0.37	0.92	-	-0.81	**2.50**	-
SCR area	-	-0.09	1.00	-1.25	-	1.64	-1.36	-	-	-0.66	-0.68	0.21	-	-0.90	-0.96	-
Ave. SCR Δ	-	-	-	0.70	-	-0.91	**-2.66**	-	-	-	-	0.04	-	0.20	**-2.42**	-
ECG HRV (Std)	-	-0.94	0.63	-	-	-0.04	1.99	-	-	0.70	0.90	-	-	-0.09	1.81	-
ECG HR Accel. (RMS)	-	-0.77	1.15	-	-	-0.27	0.86	-	-	0.11	0.93	-	-	-0.08	1.08	-
ECG HR Accel. (Std)	-	-0.48	1.43	-	-	0.14	**-2.54**	-	-	1.11	0.71	-	-	-0.39	1.92	-
Range of Temp	-	0.36	0.55	0.21	-	1.51	0.43	-	-	0.37	0.08	1.35	-	1.69	1.13	-
Ave. EMG-Arm amplitude	-	-0.32	-0.09	-1.85	-	-0.28	1.56	-	-	-0.31	0.22	-1.71	-	-0.84	**-2.19**	-
EMG-Arm zero-crossings	-	-1.00	0.99	-1.29	-	-	1.42	-	-	-1.00	1.05	-1.51	-	-1.48	1.98	-
Ave. EMG-Back amplitude	-	-1.16	0.51	0.51	-	1.12	-0.47	-	-	-0.94	0.01	-0.12	-	1.45	0.37	-
EMG-Back zero-crossings	-	-	-	-0.78	-	-	-1.42	-	-	-	-	0	-	-	-1.44	-
BVP HR	-	-0.05	0.59	-0.00	-	-1.03	**2.28**	-	-	0.18	-0.25	-0.39	-	-0.86	**2.16**	-
BVP HRV (Std)	-	-1.41	0.90	-0.03	-	0.73	1.24	-	-	0.37	1.25	0.00	-	0.65	1.43	-
BVP HRV (Range)	-	-0.92	0.90	0.65	-	0.60	1.15	-	-	0.32	1.31	0.14	-	0.49	1.27	-
BVP HR Accel. (RMS)	-	-0.14	0.94	-0.10	-	-0.98	**2.16**	-	-	0.19	0.49	-0.39	-	-0.83	-1.99	-
BVP HR Accel. (Std)	-	-0.78	1.00	-0.15	-	0.45	0.57	-	-	-0.01	1.17	-0.24	-	0.76	1.18	-
BVP Volatility	-	0.46	-0.82	0.39	-	1.49	0.90	-	-	0.18	-0.70	0.75	-	1.62	0.69	-

Bolded statistics are significant at the 5% level.

The left panel contains t statistics for pre-event features and the right panel contains t statistics for post-event features, both tested against the same set of control features. See Table 3.1 for abbreviations.

Table C.16: t Statistics for two-sided hypothesis tests of the difference in mean autonomic responses during pre- and post-event intervals versus no-event intervals (controls) for each of the components of the physiological features (rows) for instant losing trades.

	10-sec Winning Trades															
	Pre-event Interval								Post-event Interval							
Physiological Features	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*
# of SCR responses	-0.50	-1.12	0.42	1.79	**3.48**	-0.97	0.27	0.52	0.50	1.90	-0.24	0.86	1.34	0	1.00	0.85
Ave. SCR amplitude	-0.22	0.61	-1.19	1.72	-1.00	0.29	0.14	-0.58	0.09	1.40	-1.37	**-2.45**	-1.20	0.27	-0.18	-0.80
ECG HR	0.64	0.39	1.87	-	-0.45	0.41	-1.52	-	1.09	1.13	0.79	-	0.11	1.18	0.17	-
Local BVP ratio	**-2.75**	0.57	-0.79	1.49	0.68	**-2.52**	1.90	-0.85	**-3.17**	0.08	-0.24	0.39	1.21	**-2.52**	1.78	-0.14
Global BVP ratio	**-2.75**	0.56	-0.85	1.47	0.68	**-2.51**	1.92	-0.84	**-3.16**	0.06	-0.31	0.35	1.21	**-2.50**	1.75	-0.13
# of Temp jumps	-	-0.28	-0.59	0	-1.26	-0.73	-1.71	0.61	-	0.82	0.31	0	0.63	-1.16	-1.38	1.53
Ave. SCR duration	-0.81	0.45	-0.27	0.01	-0.76	0.34	0.61	-0.72	0.35	1.79	-1.05	1.43	-2.16	0.02	-1.04	-0.94
SCR area	0.49	0.45	0.03	1.16	-1.22	-0.48	-0.07	-0.27	0.27	0.96	-0.76	**-2.48**	-0.99	0.02	0.58	-0.20
Ave. SCR Δ	-0.22	-	-	-0.07	-0.59	**2.42**	0.12	-0.34	-1.17	-	-	-0.97	-1.21	**2.89**	0.80	0.00
ECG HRV (Std)	0.25	1.00	0.10	-	-0.37	**1.98**	0.03	-	1.03	0.71	-1.18	-	0.97	-0.30	**-2.64**	-
ECG HR Accel. (RMS)	0.61	0.62	1.60	-	-0.38	0.09	-1.26	-	1.19	1.20	0.36	-	0.29	1.18	0.59	-
ECG HR Accel. (Std)	0.08	0.92	-0.81	-	-0.36	-0.94	0.77	-	1.02	0.86	-0.81	-	1.00	0.51	**-2.49**	-
Range of Temp	0.87	1.90	-0.79	-0.54	1.19	-1.01	-1.10	0.44	1.13	**-2.16**	-0.25	-0.72	1.22	0.01	-0.80	1.54
Ave. EMG-Arm amplitude	**6.27**	1.29	1.62	1.17	0.33	0.15	0.05	-1.05	**4.21**	1.28	**-2.45**	0.43	0.62	-0.17	1.24	-1.64
EMG-Arm zero-crossings	**7.30**	0.23	0.98	0.91	0.63	**-3.01**	0.34	-0.72	**5.00**	1.58	1.71	0.16	0.73	0.33	0.92	-1.37
Ave. EMG-Back amplitude	0.63	-0.70	1.86	1.03	-1.73	1.03	-0.25	**2.32**	1.61	0.17	1.52	1.12	-0.93	**-2.65**	0.12	-1.57
EMG-Back zero-crossings	**2.12**	-0.37	1.22	0.88	-	-1.58	-0.97	-	**2.45**	-0.20	1.31	0.71	-	-1.19	-0.68	-
BVP HR	-1.27	0.26	0.21	-0.87	-1.05	-0.72	-1.90	-0.18	-1.46	0.97	0.71	1.04	-1.16	-0.91	-1.60	0.80
BVP HRV (Std)	**2.68**	-1.63	1.18	1.53	-0.42	-0.13	0.19	0.38	-0.23	-0.98	1.70	0.81	0.73	0.94	1.15	0.57
BVP HRV (Range)	**2.88**	-1.53	1.43	1.75	-0.44	-0.85	0.45	0.39	-0.34	-0.90	1.59	1.13	0.44	0.54	1.22	0.92
BVP HR Accel. (RMS)	-1.58	0.10	0.42	-0.50	-1.04	-0.62	-1.93	-0.02	-1.46	0.88	1.02	1.24	-1.12	-0.74	-1.41	0.87
BVP HR Accel. (Std)	**2.25**	**2.03**	1.28	1.15	-0.29	0.16	0.13	0.32	-0.12	-1.20	1.19	0.50	0.67	0.89	0.94	0.58
BVP Volatility	**-2.76**	0.67	-1.10	**-2.27**	0.72	**-2.21**	1.66	-0.48	**-3.21**	0.07	-0.65	0.99	1.21	**-2.21**	1.73	0.18

Bolded statistics are significant at the 5% level.

The left panel contains t statistics for pre-event features and the right panel contains t statistics for post-event features, both tested against the same set of control features. See Table 3.1 for abbreviations.

Table C.17: t Statistics for two-sided hypothesis tests of the difference in mean autonomic responses during pre- and post-event intervals versus no-event intervals (controls) for each of the components of the physiological features (rows) for 10-sec winning trades.

	10-sec Losing Trades															
	Pre-event Interval								Post-event Interval							
Physiological Features	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*
# of SCR responses	0.71	-0.33	0.97	0.66	0.45	-0.99	-0.72	0	2.00	0.17	0.20	0.15	1.00	-1.11	-1.24	0.71
Ave. SCR amplitude	0.14	1.55	1.31	0.58	-0.93	-0.35	-0.19	0.43	1.89	2.03	-0.02	0.48	1.04	-1.31	-0.59	0.49
ECG HR	1.09	0.80	-0.79	-	-0.54	1.39	-0.15	-	0.13	-0.17	-0.96	-	-0.14	1.05	-0.55	-
Local BVP ratio	-0.02	0.58	0.50	1.12	-0.35	0.53	0.59	1.10	-0.03	1.24	0.75	1.05	-0.18	1.58	1.08	0.08
Global BVP ratio	-0.02	0.57	0.53	1.09	-0.35	0.55	0.61	1.10	-0.03	1.25	0.79	1.08	-0.18	1.58	1.06	0.08
# of Temp jumps	-	0.66	0.45	1.46	-1.00	-1.44	-0.75	-	-	-1.07	0	0.59	-	-0.58	0.46	-
Ave. SCR duration	0.20	0.84	-0.43	-0.45	0.38	-0.29	0.06	-0.06	1.99	1.72	-0.12	-0.56	0.71	-1.37	-0.19	-0.11
SCR area	-0.25	0.97	1.27	0.23	-0.41	-1.33	-0.64	0.39	1.83	1.71	-0.17	0.74	1.63	-1.78	-0.30	-0.20
Ave. SCR Δ	0.71	-	-	0.90	1.25	-0.95	**-2.01**	0	2.00	-	-	1.75	0.24	-0.21	-0.11	0
ECG HRV (Std)	-0.88	0.06	-0.36	-	-0.98	-0.07	1.41	-	**-3.17**	-1.04	-0.60	-	-0.89	-0.91	0.28	-
ECG HR Accel. (RMS)	1.13	0.79	-0.71	-	-0.66	1.33	0.26	-	0.16	-0.20	-0.89	-	-0.41	0.68	-0.32	-
ECG HR Accel. (Std)	**-3.76**	-0.23	-1.05	-	-1.01	0.28	1.97	-	**-3.41**	-1.07	-1.07	-	-0.92	-0.71	0.55	-
Range of Temp	-1.51	-0.05	0.36	-0.71	0.34	0.72	0.22	0.13	0.45	-0.22	-0.97	-1.56	-0.34	0.49	0.91	-0.33
Ave. EMG-Arm amplitude	-0.99	0.31	0.04	1.72	0.44	-1.60	0.97	0.51	0.46	0.09	-0.05	1.47	0.76	-1.22	0.86	-0.85
EMG-Arm zero-crossings	-1.00	1.27	0.41	1.48	0	-0.75	0.72	-1.00	-	0.69	0.21	0.99	1.00	0.38	0.19	-1.00
Ave. EMG-Back amplitude	0.42	-1.56	-0.50	1.93	-1.18	-1.06	-0.42	-0.27	0.62	-1.53	-0.70	1.35	-0.91	0.55	-0.27	-0.61
EMG-Back zero-crossings	-	1.00	-1.00	-1.43	-	0.34	-0.43	-	-	1.00	-	-0.57	-	-0.30	-0.26	-
BVP HR	-1.28	0.82	0.58	0.56	0.58	1.26	**2.21**	-0.60	-0.31	-0.22	1.07	0.73	0.47	0.92	-1.13	-0.80
BVP HRV (Std)	-0.85	1.32	-0.55	1.70	-0.28	-0.36	1.57	1.09	-0.75	0.60	1.22	1.94	2.50	-0.52	1.49	-0.55
BVP HRV (Range)	-0.75	1.84	-0.37	1.84	-0.16	-0.12	1.74	0.90	-0.76	0.85	1.21	1.81	1.73	-0.92	1.49	-0.63
BVP HR Accel. (RMS)	-1.28	0.93	0.55	0.90	0.58	1.28	-1.97	-0.56	-1.09	-0.17	1.08	1.12	0.47	0.96	-0.90	-0.84
BVP HR Accel. (Std)	-0.98	1.56	0.33	1.79	-1.48	-0.85	1.43	1.13	-0.89	0.85	1.20	1.96	0.66	-1.27	**-2.00**	-0.18
BVP Volatility	-0.01	0.55	0.58	0.97	-0.34	0.56	0.15	1.13	-0.06	1.33	0.83	0.51	-0.18	1.53	0.67	0.06

Bolded statistics are significant at the 5% level.

The left panel contains t statistics for pre-event features and the right panel contains t statistics for post-event features, both tested against the same set of control features. See Table 3.1 for abbreviations.

Table C.18: t Statistics for two-sided hypothesis tests of the difference in mean autonomic responses during pre- and post-event intervals versus no-event intervals (controls) for each of the components of the physiological features (rows) for 10-sec losing trades.

	Large Trades															
	Pre-event Interval								Post-event Interval							
Physiological Features	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*
# of SCR responses	-0.23	0.59	0.21	0.10	-1.57	-0.28	1.10	-0.99	1.07	0.27	0.50	0.47	-0.94	-0.78	0.96	-1.17
Ave. SCR amplitude	0.06	1.51	-0.33	-0.69	-0.84	0.09	-0.88	0.15	0.54	1.35	-0.20	-0.39	-0.39	0.01	-0.99	-0.20
ECG HR	-0.71	-0.77	1.22	-	-1.47	0.69	-0.08	-	-0.77	0.53	0.90	-	-0.65	1.03	-0.28	-
Local BVP ratio	1.25	0.45	-0.14	-1.64	1.59	1.11	0.50	0.64	1.26	0.14	0.20	-0.16	1.14	1.03	1.14	0.70
Global BVP ratio	1.26	0.40	-0.18	-1.66	1.57	1.12	0.51	0.67	1.26	0.13	0.19	-0.17	1.11	1.01	1.10	0.73
# of Temp jumps	-	1.65	0	1.45	1.38	-1.00	-1.04	0.78	-	0.68	0.35	**-2.52**	0	0.59	0	0.78
Ave. SCR duration	-0.03	1.50	0.34	0.21	0.96	-0.41	-0.71	-1.40	0.83	0.85	0.55	0.01	1.50	-1.16	0.24	-1.67
SCR area	-0.30	1.73	-0.71	-0.16	-0.99	0.20	-0.81	0.72	-0.04	1.48	0.81	0.78	0.61	-0.06	-0.03	-0.01
Ave. SCR Δ	-0.47	-1.00	-	**-2.36**	0.61	-0.05	**-2.46**	-1.00	1.00	-1.48	-	1.51	-1.08	-0.29	**-2.82**	1.00
ECG HRV (Std)	0.77	-1.11	0.31	-	-1.65	-0.17	1.47	-	0.45	0.86	-0.35	-	-0.42	0.31	1.03	-
ECG HR Accel. (RMS)	-0.27	-0.79	1.02	-	-1.50	0.64	0.48	-	-0.48	0.55	0.64	-	-0.60	1.05	0.19	-
ECG HR Accel. (Std)	0.64	0.55	-1.17	-	-1.85	-0.31	1.65	-	0.46	1.19	-0.47	-	-0.57	0.17	1.02	-
Range of Temp	0.26	2.00	-0.78	-0.95	**-2.84**	1.09	-0.55	1.02	1.00	0.53	-1.04	-0.09	0.04	**-2.43**	1.21	0.85
Ave. EMG-Arm amplitude	-1.53	0.98	1.44	-0.41	0.64	-0.70	0.66	0.29	-0.76	1.69	0.48	-0.10	1.36	-0.30	0.48	0.49
EMG-Arm zero-crossings	**2.30**	0.99	1.52	-0.40	0.36	-0.26	0.07	0.54	-1.60	-0.07	0.21	0.95	1.22	-0.48	0.04	0.38
Ave. EMG-Back amplitude	-0.25	**-2.24**	1.52	0.45	-0.84	-0.64	-0.39	1.03	0.46	0.71	1.03	1.74	-1.29	-1.18	-0.54	0.83
EMG-Back zero-crossings	0.24	-1.00	1.51	-0.89	1.45	**2.39**	0.53	-	0.34	-1.00	1.51	-0.14	1.45	**2.39**	0.82	-
BVP HR	-1.44	-1.02	-0.55	0.04	-0.92	1.20	**2.47**	-0.44	-1.53	0.81	-0.43	**2.22**	-1.00	0.49	-1.09	-0.83
BVP HRV (Std)	-0.70	-0.36	1.03	0.48	0.04	-0.75	**-2.11**	-0.32	1.03	-0.03	0.90	0.16	**-2.31**	-0.04	0.85	-0.76
BVP HRV (Range)	-0.91	-0.41	0.79	0.82	-0.20	-0.35	2.00	-0.54	0.85	0.00	0.59	0.20	**-2.16**	0.22	0.77	-1.06
BVP HR Accel. (RMS)	-1.49	-0.95	-0.44	0.03	-0.89	1.22	**2.19**	-0.40	-1.49	0.73	-0.27	**2.54**	-0.93	0.54	-1.03	-0.87
BVP HR Accel. (Std)	-0.49	0.04	1.21	0.40	-0.17	-0.84	1.78	-0.37	1.05	-0.20	0.68	0.11	1.62	-0.11	0.67	-0.50
BVP Volatility	1.30	0.45	-0.58	-1.65	1.50	1.40	-0.01	0.75	1.30	0.18	-0.21	-0.23	1.02	1.22	1.08	0.76

Bolded statistics are significant at the 5% level.

The left panel contains t statistics for pre-event features and the right panel contains t statistics for post-event features, both tested against the same set of control features. See Table 3.1 for abbreviations.

Table C.19: t Statistics for two-sided hypothesis tests of the difference in mean autonomic responses during pre- and post-event intervals versus no-event intervals (controls) for each of the components of the physiological features (rows) for large trades.

	Auto Buy Trades															
	Pre-event Interval								Post-event Interval							
Physiological Features	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*
# of SCR responses	-0.12	**-3.16**	0.50	0.78	1.18	-0.99	-0.27	-1.89	1.73	0.96	-0.26	-0.81	1.25	-1.36	0.47	**2.53**
Ave. SCR amplitude	-0.38	**-2.77**	1.46	-0.13	-0.69	0.39	0.11	-1.52	-0.25	**-2.36**	-0.09	1.55	-0.06	1.26	0.69	**2.24**
ECG HR	1.28	**-2.17**	1.29	-	-0.04	1.84	-1.51	-	0.37	-0.30	1.98	-	0.09	0.60	-0.15	-
Local BVP ratio	-0.05	-1.04	-0.12	-0.12	**-2.08**	**-2.16**	0.34	0.97	-0.08	-1.51	0.13	0.81	1.98	0.70	**-1.98**	-0.17
Global BVP ratio	-0.05	-1.05	-0.16	-0.18	**-2.04**	**-2.14**	0.40	0.97	-0.07	-1.52	0.12	0.82	1.96	0.69	**-2.03**	-0.18
# of Temp jumps	-	0.48	0.22	**-2.23**	-0.85	1.09	**-2.39**	**-2.53**	-	1.52	0.22	1.81	-0.85	1.32	**-2.39**	**-2.53**
Ave. SCR duration	-0.05	**-1.98**	0.44	-0.10	-0.62	1.11	0.70	-1.92	0.20	-0.04	0.41	-1.19	-0.19	0.93	0.19	**2.49**
SCR area	0.13	**-3.17**	1.19	0.61	0.39	-0.17	0.42	-1.29	0.63	1.36	0.44	0.71	0.87	0.62	1.47	-2.06
Ave. SCR Δ	0.82	-1.03	-	**2.70**	-0.93	-1.78	1.00	1.41	0.88	-0.76	-	**2.60**	0.22	-0.57	-0.76	-
ECG HRV (Std)	**-3.06**	1.36	-0.62	-	-0.26	-0.02	-1.48	-	1.75	1.34	0.82	-	0.04	-0.84	-0.01	-
ECG HR Accel. (RMS)	1.80	**-2.21**	0.96	-	-0.17	1.67	-1.65	-	0.84	-0.12	1.98	-	0.02	0.26	-0.24	-
ECG HR Accel. (Std)	**-3.14**	1.08	-1.18	-	-0.30	0.02	-1.43	-	1.93	1.92	0.36	-	-0.27	-1.21	-0.21	-
Range of Temp	0.38	1.32	1.47	1.73	-1.91	-1.72	**-2.68**	0.24	0.37	1.04	0.75	0.62	-1.57	-1.42	1.36	0.45
Ave. EMG-Arm amplitude	1.64	-1.50	0.78	1.05	1.05	-1.36	-1.49	-0.83	**-2.26**	-1.24	1.42	0.16	1.88	-1.92	1.11	-1.14
EMG-Arm zero-crossings	1.74	1.76	0.60	1.29	0.90	**-2.38**	-1.04	-0.98	1.64	**-2.02**	1.52	0.58	1.08	1.58	1.72	-0.91
Ave. EMG-Back amplitude	0.69	0.74	0.06	1.75	1.93	0.03	0.98	-0.74	0.30	1.16	0.03	1.26	0.96	-0.45	**-2.93**	-0.92
EMG-Back zero-crossings	-0.92	1.42	0.91	-0.08	1.00	0.57	**-3.22**	-	-1.29	1.11	0.68	-0.05	1.00	0.88	**-3.37**	-
BVP HR	0.54	0.88	0.22	-1.69	0.02	0.31	0.10	0.12	-0.12	-1.79	0.82	**2.59**	0.34	0.49	-0.66	0.51
BVP HRV (Std)	1.08	1.55	0.81	0.68	-0.26	0.28	-0.80	0.11	0.97	**-3.08**	1.62	1.10	0.33	0.12	1.40	-1.07
BVP HRV (Range)	1.14	1.75	0.67	1.00	-0.10	-0.45	-1.05	0.27	1.06	**-2.95**	1.82	0.92	0.46	-0.56	1.19	-0.73
BVP HR Accel. (RMS)	0.65	1.11	0.31	-1.78	0.01	0.47	-0.04	0.17	-0.02	-1.33	0.94	**2.60**	0.34	0.61	-0.46	0.26
BVP HR Accel. (Std)	0.89	1.51	0.72	0.68	0.16	0.50	-0.75	0.30	0.83	**-3.62**	1.95	0.90	0.62	-0.58	1.30	-0.68
BVP Volatility	-0.08	-1.07	-0.29	-0.25	**-2.03**	**-2.30**	0.93	0.91	-0.11	-1.41	0.04	0.98	1.91	1.07	**-2.22**	-0.29

Bolded statistics are significant at the 5% level.

The left panel contains t statistics for pre-event features and the right panel contains t statistics for post-event features, both tested against the same set of control features. See Table 3.1 for abbreviations.

Table C.20: t Statistics for two-sided hypothesis tests of the difference in mean autonomic responses during pre- and post-event intervals versus no-event intervals (controls) for each of the components of the physiological features (rows) for auto buy trades.

	Auto Sell Trades															
	Pre-event Interval								Post-event Interval							
Physiological Features	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*
# of SCR responses	-1.03	**-3.17**	-1.51	**-2.79**	1.22	0.66	0.84	0.25	-1.97	0.89	-1.64	1.25	1.00	0.48	0.66	1.14
Ave. SCR amplitude	-0.49	**-3.23**	-1.01	1.22	0.78	1.34	-0.43	-0.63	-1.38	**-3.21**	-1.07	-0.16	0.89	1.58	-1.53	-0.62
ECG HR	-1.23	1.26	0.00	-	0.63	0.88	-1.09	-	-0.48	-0.10	-0.44	-	0.13	1.54	-1.82	-
Local BVP ratio	**-1.99**	-0.14	**-2.72**	1.96	1.49	1.54	**-2.98**	1.94	**-2.05**	-0.21	**-2.68**	-0.30	1.21	0.56	**-2.76**	-0.12
Global BVP ratio	**-2.00**	-0.14	**-2.74**	1.97	1.49	1.52	**-3.03**	1.94	**-2.04**	-0.17	**-2.71**	-0.29	1.20	0.56	**-2.89**	-0.12
# of Temp jumps	-	0.49	-0.48	-1.38	-0.71	1.56	0	-0.76	-	-1.14	0	0	0	**-2.77**	0.19	0
Ave. SCR duration	-1.01	**-2.39**	-1.35	0.09	-0.41	0.83	**2.62**	-0.80	-1.98	1.89	-1.43	0.37	0.20	1.65	-1.39	-0.38
SCR area	-0.17	**-3.02**	-1.49	1.44	1.03	0.85	-0.53	-0.62	-0.85	**-3.05**	-1.12	0.33	0.96	0.94	-1.14	-0.79
Ave. SCR Δ	-1.12	-0.82	-	**2.41**	**3.40**	-1.20	1.14	-1.34	-0.17	-1.08	-	-0.11	-2.14	-0.14	1.43	0.27
ECG HRV (Std)	1.32	**-2.31**	0.32	-	1.63	1.06	-0.00	-	1.13	**-2.85**	-1.00	-	0.68	0.90	-1.19	-
ECG HR Accel. (RMS)	-0.40	1.56	0.08	-	0.82	1.02	-0.89	-	0.03	0.40	-0.55	-	0.29	1.58	-1.79	-
ECG HR Accel. (Std)	1.17	1.90	0.83	-	1.66	1.75	-0.26	-	1.30	**-2.58**	0.64	-	0.83	1.60	-1.39	-
Range of Temp	**-2.21**	0.30	-0.36	-0.78	-2.10	**-2.98**	-0.52	-1.22	1.53	0.42	0.34	-1.70	-1.19	**-2.56**	-0.16	-0.17
Ave. EMG-Arm amplitude	1.06	-0.49	-1.64	**-2.34**	1.25	-1.87	1.93	0.68	1.16	-0.08	-1.14	1.00	0.87	**2.21**	-0.90	0.41
EMG-Arm zero-crossings	0.36	-0.33	-0.56	1.84	**-2.83**	0.86	1.05	0.81	0.77	-0.98	-0.28	0.50	**-2.83**	0.45	-1.06	0.64
Ave. EMG-Back amplitude	0.60	-0.26	-1.37	**-2.22**	1.13	0.33	**-3.92**	1.96	0.16	-0.76	-1.34	1.90	1.16	0.21	1.93	0.45
EMG-Back zero-crossings	-1.30	1.16	-	0.36	-	-1.70	**-2.61**	-	-1.74	**-2.27**	-	0.14	-	**2.31**	**-2.57**	-
BVP HR	-1.31	0.52	-0.39	-0.81	-2.27	0.04	**3.38**	0.36	-1.88	-1.42	-0.74	0.54	-1.71	0.19	-1.88	1.56
BVP HRV (Std)	1.57	0.57	-1.07	**-3.06**	1.32	0.18	**-3.26**	0.01	1.77	**-2.73**	-0.22	1.65	1.02	0.68	1.93	0.21
BVP HRV (Range)	1.47	0.62	-1.01	**-3.28**	0.24	0.03	**-2.42**	-0.21	1.63	**-2.59**	-1.00	**-2.20**	0.63	0.74	1.43	0.48
BVP HR Accel. (RMS)	-1.15	0.59	-0.45	-0.31	-2.23	0.09	**2.99**	0.38	-1.72	-1.07	-0.72	0.86	-1.69	0.31	-1.62	1.53
BVP HR Accel. (Std)	1.33	0.43	-0.55	**-2.73**	-1.05	0.46	1.52	0.08	1.73	**-2.24**	-1.33	0.97	2.25	0.50	0.53	0.70
BVP Volatility	1.96	-0.11	**-2.65**	1.57	1.50	1.23	**-2.73**	**-2.15**	**-2.03**	-0.17	**-2.60**	-0.96	1.14	0.38	**-2.68**	0.05

Bolded statistics are significant at the 5% level.

The left panel contains t statistics for pre-event features and the right panel contains t statistics for post-event features, both tested against the same set of control features. See Table 3.1 for abbreviations.

Table C.21: t Statistics for two-sided hypothesis tests of the difference in mean autonomic responses during pre- and post-event intervals versus no-event intervals (controls) for each of the components of the physiological features (rows) for auto sell trades.

	Instant Winning vs. Losing Trades															
	Pre-event Interval								Post-event Interval							
Physiological Features	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*
# of SCR responses	-	0.34	-1.17	0.64	-	0.60	-1.02	-	-	1.00	-1.43	0.71	-	-0.11	**2.29**	-
Ave. SCR amplitude	-	0.39	-1.17	1.04	-	-1.01	0.12	-	-	1.15	-1.51	1.97	-	-1.72	0.20	-
ECG HR	-	0.84	-0.09	-	-	-0.51	-1.84	-	-	1.87	-1.13	-	-	-1.40	-1.89	-
Local BVP ratio	-	-0.50	2.13	1.25	-	1.18	0.56	-	-	-1.20	1.99	-0.19	-	0.27	1.00	-
Global BVP ratio	-	-0.53	2.14	1.27	-	1.18	0.59	-	-	-1.32	1.97	-0.19	-	0.26	1.02	-
# of Temp jumps	-	-0.20	1.59	-	-	-	-0.19	-	-	-1.00	**-2.16**	1.00	-	-	-0.59	-
Ave. SCR duration	-	0.29	-1.17	0.91	-	-1.07	-0.03	-	-	1.28	-1.53	0.99	-	0.17	1.60	-
SCR area	-	0.36	-1.17	**-2.30**	-	-0.52	0.33	-	-	1.15	-1.51	**-2.86**	-	-1.50	0.23	-
Ave. SCR Δ	-	-	-	0.74	-	1.04	-0.21	-	-	-	-	1.27	-	1.01	-1.63	-
ECG HRV (Std)	-	1.28	-0.40	-	-	-0.70	-1.42	-	-	-0.59	-1.61	-	-	-0.69	-0.49	-
ECG HR Accel. (RMS)	-	0.85	-0.08	-	-	-0.61	-1.90	-	-	1.91	-1.30	-	-	-1.47	-1.93	-
ECG HR Accel. (Std)	-	0.84	-0.57	-	-	-0.47	-1.42	-	-	-0.82	-0.57	-	-	0.38	-0.91	-
Range of Temp	-	-0.93	0.92	1.97	-	1.13	0.13	-	-	-1.36	0.57	0.39	-	0.11	-0.50	-
Ave. EMG-Arm amplitude	-	-0.28	-0.70	-0.40	-	-0.29	0.89	-	-	0.04	-0.64	-0.08	-	0.04	-0.55	-
EMG-Arm zero-crossings	-	0.66	-0.16	-0.39	-	-0.52	0.28	-	-	1.00	-0.86	0	-	-1.59	-1.37	-
Ave. EMG-Back amplitude	-	1.02	-0.10	-0.39	-	0.35	-0.43	-	-	**-3.17**	0.20	-0.54	-	-0.93	**2.30**	-
EMG-Back zero-crossings	-	-	0.86	-1.23	-	1.63	-1.14	-	-	-	0.70	-	-	1.56	-1.40	-
BVP HR	-	0.59	-0.49	0.06	-	-0.30	**2.15**	-	-	1.47	-0.81	1.50	-	0.46	**2.92**	-
BVP HRV (Std)	-	1.54	-0.04	-0.46	-	0.12	0.40	-	-	0.10	-0.82	0.23	-	-1.32	-0.28	-
BVP HRV (Range)	-	1.83	-0.73	-0.63	-	-0.14	0.10	-	-	0.32	-0.91	0.82	-	-1.59	-0.75	-
BVP HR Accel. (RMS)	-	0.72	-0.50	-0.06	-	-0.31	**2.05**	-	-	1.59	-0.83	1.53	-	0.18	**2.89**	-
BVP HR Accel. (Std)	-	1.19	-0.12	-0.87	-	-0.40	0.49	-	-	0.10	-0.58	-0.43	-	**2.09**	-0.75	-
BVP Volatility	-	-0.47	2.09	0.75	-	1.36	0.35	-	-	-1.21	1.87	0.69	-	0.38	0.89	-

Bolded statistics are significant at the 5% level.

The left panel contains t statistics for pre-event features and the right panel contains t statistics for post-event features, for testing Instant Winning vs. Losing Trades. See Table 3.1 for abbreviations.

Table C.22: t Statistics for two-sided hypothesis tests of the difference in mean autonomic responses during pre- and post-event intervals for each of the components of the physiological features (rows) for instant winning vs. losing trades.

	10-sec Winning vs. Losing Trades															
	Pre-event Interval								Post-event Interval							
Physiological Features	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*
# of SCR responses	1.15	0.17	-1.04	-0.17	0.27	-0.77	-0.08	-0.25	0.50	**-2.80**	-1.61	0	0.88	-0.85	0.93	-1.00
Ave. SCR amplitude	1.05	0.37	-1.78	-0.92	0.62	-0.14	-1.15	-0.50	0.30	0.59	-1.01	0.75	-0.97	0.33	-1.08	-1.26
ECG HR	1.89	-0.17	**2.36**	-	0.31	-0.03	-0.47	-	1.40	1.13	-1.58	-	-0.10	0.96	0.14	-
Local BVP ratio	0.44	1.02	1.73	0.77	0.72	**-2.28**	1.46	-0.48	1.67	-0.09	0.50	-0.14	0.98	**-2.30**	1.59	0.03
Global BVP ratio	0.44	0.99	1.77	0.78	0.71	**-2.28**	1.45	-0.49	1.66	-0.13	0.48	-0.16	0.99	**-2.30**	1.58	0.03
# of Temp jumps	-	-1.38	-0.51	-0.52	1.46	0.01	0.84	-	-	0.82	0.62	-1.79	1.26	0	0.35	-
Ave. SCR duration	1.24	0.26	-0.64	-0.24	0.39	-1.98	-1.57	-0.73	0.27	1.33	-1.68	1.46	-0.48	-1.04	-1.29	-1.05
SCR area	0.91	-0.04	-1.64	-0.57	0.98	-1.09	-1.14	-0.94	0.34	-0.30	-1.21	0.75	0.50	-0.62	-0.50	-1.26
Ave. SCR Δ	0.39	-	-	1.46	1.25	-0.28	-0.05	-0.74	-1.44	-	-	-1.31	-0.39	**2.67**	0.27	0
ECG HRV (Std)	0.74	-1.36	-1.84	-	-0.82	0.15	0.74	-	1.37	-0.60	-1.94	-	1.12	1.74	**-2.88**	-
ECG HR Accel. (RMS)	1.60	-0.20	**2.34**	-	-0.13	0.04	-0.08	-	1.54	1.09	-1.72	-	0.19	1.33	0.88	-
ECG HR Accel. (Std)	0.66	-1.39	-1.86	-	-0.98	0.43	0.80	-	1.04	-1.03	-1.38	-	1.03	**-2.37**	**-2.64**	-
Range of Temp	1.22	-0.13	-1.68	0.37	-0.24	0.36	1.54	-1.72	0.81	**-2.09**	0.15	-0.45	0.24	-0.04	1.33	-0.83
Ave. EMG-Arm amplitude	1.38	1.40	-0.80	-0.13	1.21	-1.58	1.50	-0.05	-1.79	1.23	0.89	-0.74	-0.05	-1.28	1.89	-1.43
EMG-Arm zero-crossings	1.43	1.08	0.51	-0.17	1.07	0.54	0.96	0.06	**2.82**	1.81	0.82	-0.65	0.87	-0.73	1.35	-1.42
Ave. EMG-Back amplitude	-0.05	0.57	-0.60	0.39	0.65	**-2.00**	-0.04	-0.47	**-2.13**	**-2.03**	0.57	-0.42	0.07	**-2.45**	0.18	-0.93
EMG-Back zero-crossings	0.53	1.01	0.85	0.31	-	-1.43	0.00	-	-0.35	-0.69	1.16	0.95	-	**2.29**	-1.27	-
BVP HR	0.84	-0.27	0.60	-0.75	0.75	-0.99	-1.51	-0.88	-1.09	1.19	1.26	0.85	-1.09	-0.66	**2.44**	-0.36
BVP HRV (Std)	-0.22	-0.12	0.84	0.44	2.20	0.50	-0.11	-1.78	**-2.37**	0.42	0.58	-0.65	**-3.00**	1.42	0.95	-1.65
BVP HRV (Range)	-0.12	0.06	0.80	0.13	**-2.79**	-0.00	-0.38	-1.95	**-2.43**	0.56	0.32	-0.47	**-3.26**	1.49	0.68	-1.55
BVP HR Accel. (RMS)	0.81	-0.26	0.69	-0.59	0.81	-0.93	-1.48	-0.98	-0.84	1.22	1.36	0.63	-1.03	-0.42	**2.30**	-0.63
BVP HR Accel. (Std)	-0.60	0.31	0.78	0.11	1.65	0.49	0.49	-1.50	**-2.04**	0.49	0.63	-1.20	**-2.39**	1.81	0.88	-1.47
BVP Volatility	0.41	1.10	1.69	0.36	0.72	**-2.18**	1.00	-0.58	1.66	-0.19	0.35	0.36	0.97	**-2.22**	1.22	0.04

Bolded statistics are significant at the 5% level.

The left panel contains t statistics for pre-event features and the right panel contains t statistics for post-event features, for testing 10-sec Winning vs. Losing Trades. See Table 3.1 for abbreviations.

Table C.23: t Statistics for two-sided hypothesis tests of the difference in mean autonomic responses during pre- and post-event intervals for each of the components of the physiological features (rows) for 10-sec winning vs. losing trades.

	Trade Alerts															
	Pre-event Interval								Post-event Interval							
Physiological Features	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*
# of SCR responses	0	0	1.00	-0.16	-	0.58	0.66	-	-0.83	0.43	1.00	0.82	-	0.04	1.20	-1.00
Ave. SCR amplitude	1.05	0.14	1.00	-1.09	-	-0.07	1.51	-	0.34	0.84	1.00	0.52	-	-0.84	1.24	-1.00
ECG HR	-0.19	**-2.05**	0.32	-	-	-0.29	0.84	-	-1.21	1.35	-0.95	-	-	-0.36	-0.17	-
Local BVP ratio	1.55	0.35	1.42	0.42	-	0.76	0.07	-1.64	1.63	0.92	0.38	0.92	-	0.23	0.70	-2.59
Global BVP ratio	1.55	0.36	1.43	0.40	-	0.79	0.08	-1.65	1.63	0.89	0.41	0.93	-	0.27	0.70	-2.62
# of Temp jumps	-	-1.21	-0.63	0.55	-	-0.45	0	-	-	-1.75	0	**-2.68**	-	-1.01	0.65	-
Ave. SCR duration	0.48	0.98	1.00	**-1.99**	-	-0.26	0.73	-	0.41	1.34	1.00	**-2.02**	-	**2.19**	-1.06	-1.00
SCR area	1.06	0.13	1.00	0.73	-	-0.06	1.23	-	-0.44	1.35	1.00	1.29	-	-1.44	1.36	-1.00
Ave. SCR Δ	0	1.00	-	1.20	-	-1.23	-1.31	-	-1.19	1.00	-	1.47	-	0.38	-1.72	-1.00
ECG HRV (Std)	0.52	1.39	-0.63	-	-	**2.45**	0.77	-	1.58	0.66	-0.75	-	-	**2.54**	0.12	-
ECG HR Accel. (RMS)	-0.09	**-2.05**	0.16	-	-	-0.88	0.85	-	-0.93	1.35	-1.00	-	-	-0.94	-0.03	-
ECG HR Accel. (Std)	0.50	1.19	-0.56	-	-	**2.29**	0.85	-	1.58	0.71	-1.25	-	-	**2.33**	0.38	-
Range of Temp	0.74	-1.38	-0.85	1.16	-	-1.10	-0.32	0.71	1.12	-0.19	0.81	**-2.28**	-	0.00	0.55	-0.45
Ave. EMG-Arm amplitude	-0.82	-0.61	-0.20	-0.33	-	0.88	1.01	**-8.97**	0.60	-1.25	0.74	0.71	-	0.80	1.42	-1.32
EMG-Arm zero-crossings	-0.71	0.17	-2.09	0.55	-	-1.37	1.44	-	-0.54	0	**3.15**	1.07	-	-0.59	1.26	-
Ave. EMG-Back amplitude	1.40	-1.39	0.79	1.19	-	-1.09	1.78	**7.60**	1.83	-1.33	1.00	1.40	-	-1.03	**-2.41**	-2.32
EMG-Back zero-crossings	-0.41	-1.47	0.30	-0.90	-	-1.22	-0.80	-	-0.84	-1.49	-0.72	0	-	-1.75	-1.13	-
BVP HR	-0.93	1.49	-0.15	-0.08	-	-0.33	-0.92	-0.47	-1.48	1.44	-1.04	-0.09	-	-0.04	-0.89	-0.50
BVP HRV (Std)	-0.13	0.67	1.87	0.77	-	0.27	1.68	3.31	1.82	0.24	**-2.89**	1.84	-	-0.64	1.41	3.15
BVP HRV (Range)	-0.06	0.44	2.17	0.94	-	-0.29	1.65	**-6.02**	**-2.51**	0.11	**-4.37**	1.86	-	-1.00	1.19	4.19
BVP HR Accel. (RMS)	-0.99	1.54	-0.09	-0.05	-	-0.23	-0.76	-0.36	-1.47	1.52	-0.98	0.16	-	-0.11	-0.72	-0.39
BVP HR Accel. (Std)	-0.12	0.33	0.74	1.29	-	-0.09	1.83	**-5.04**	1.64	0.15	1.85	**-2.18**	-	-0.91	1.41	3.42
BVP Volatility	1.58	0.05	1.33	-0.15	-	0.54	0.14	-1.66	1.64	0.62	0.33	0.77	-	0.28	0.85	-2.50

Bolded statistics are significant at the 5% level.

The left panel contains t statistics for pre-event features and the right panel contains t statistics for post-event features, both tested against the same set of control features. See Table 3.1 for abbreviations.

Table C.24: t Statistics for two-sided hypothesis tests of the difference in mean autonomic responses during pre- and post-event intervals versus no-event intervals (controls) for each of the components of the physiological features (rows) for trade alerts.

	Quote Alerts															
	Pre-event Interval								Post-event Interval							
Physiological Features	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*
# of SCR responses	0.94	0.59	-0.40	-0.35	0.47	1.73	-0.44	-0.60	0.76	0	-0.47	-0.87	0.91	1.22	0.25	-1.60
Ave. SCR amplitude	**-2.27**	0.72	1.69	0.10	-0.55	1.68	-0.78	-0.94	0.15	-0.61	1.27	0.29	0.29	**-2.53**	-0.98	-1.54
ECG HR	0.98	-1.32	-1.22	-	-0.59	1.51	-1.82	-	1.30	**2.48**	0.08	-	0.89	0.99	**2.69**	-
Local BVP ratio	**-2.47**	-0.72	1.70	-0.39	**-2.77**	**-2.73**	1.30	-0.57	**-2.48**	-0.65	**-2.60**	0.35	**-2.30**	**-3.50**	**-2.22**	-0.97
Global BVP ratio	**-2.46**	-0.76	1.77	-0.39	**-2.77**	**-2.72**	1.38	-0.57	**-2.48**	-0.65	**-2.63**	0.32	**-2.30**	**-3.44**	**-2.35**	-0.97
# of Temp jumps	-	-1.23	**-2.11**	0.63	-0.52	1.43	1.13	0.60	-	-0.72	1.30	1.57	1.73	0.34	0	0.60
Ave. SCR duration	1.58	0.68	0.53	-1.72	0.56	-1.74	-0.77	-0.83	0.73	-0.49	-0.55	-1.72	1.60	-1.42	**2.24**	-1.85
SCR area	**-2.34**	1.00	1.51	-0.93	0.55	1.72	0.28	-1.05	0.07	0.22	0.54	-1.09	0.84	**-2.57**	-0.49	-1.74
Ave. SCR Δ	0.74	-	-	-1.28	**-2.87**	1.47	0.71	1.41	0.98	-	-	0.24	1.82	1.05	0.35	1.56
ECG HRV (Std)	0.99	-0.42	-0.50	-	-0.24	0.71	0.01	-	1.57	-0.77	0.33	-	1.08	1.66	-0.93	-
ECG HR Accel. (RMS)	1.12	-1.31	-1.17	-	-0.56	1.44	-1.40	-	1.47	**2.42**	0.15	-	0.91	1.25	**2.35**	-
ECG HR Accel. (Std)	1.14	-0.31	-1.43	-	-0.40	0.44	-0.22	-	1.25	0.27	0.60	-	0.50	1.46	-1.25	-
Range of Temp	0.94	0.45	1.26	**-2.98**	**2.42**	0.86	0.06	1.03	1.05	0.92	1.00	**-2.45**	-0.57	1.61	-1.02	0.57
Ave. EMG-Arm amplitude	-0.40	-0.00	0.33	-1.46	-1.40	1.91	-0.78	-0.55	0.14	-0.07	-0.50	-0.85	0.13	**-2.24**	0.13	-0.41
EMG-Arm zero-crossings	-0.91	0	-0.47	-0.74	-0.80	-0.90	-1.45	0.38	-0.17	1.10	-0.05	0.04	0.16	1.25	-0.64	0.66
Ave. EMG-Back amplitude	1.35	-0.36	**-2.35**	-1.58	1.12	1.75	**2.23**	0.78	1.11	-0.35	1.49	-1.01	1.66	**-2.78**	-1.88	-0.26
EMG-Back zero-crossings	0.85	-1.82	-1.66	0.56	-	-1.93	-1.69	-	1.07	**2.56**	-1.77	0	-	**2.58**	-1.78	-
BVP HR	-0.36	-1.70	0.69	0.94	**2.88**	-0.82	-1.67	1.49	0.24	**2.84**	0.40	1.32	-1.81	-1.72	**3.06**	1.72
BVP HRV (Std)	1.24	1.14	-0.58	-1.04	-0.41	**-2.16**	-0.42	-0.43	1.71	0.03	-1.09	-1.11	1.35	**-2.23**	0.93	-0.99
BVP HRV (Range)	1.05	1.17	-0.26	-0.53	-0.42	1.53	-0.58	0.29	1.60	-0.28	-1.05	-0.83	1.23	**-2.00**	0.44	-0.42
BVP HR Accel. (RMS)	-0.22	-1.60	0.67	0.73	**2.73**	-0.49	-1.74	1.37	0.38	**2.82**	0.32	1.17	-1.62	-1.48	**3.00**	1.57
BVP HR Accel. (Std)	1.68	1.11	-0.17	-1.78	-1.01	1.43	-0.54	-0.14	**-2.08**	-0.16	-0.36	-1.36	1.57	1.84	0.41	-0.54
BVP Volatility	**-2.45**	-0.47	1.64	0.35	**-2.72**	**-2.47**	1.38	0.14	**-2.52**	-0.50	**-2.52**	0.75	2.16	**-3.34**	**-2.02**	-0.21

Bolded statistics are significant at the 5% level.

The left panel contains t statistics for pre-event features and the right panel contains t statistics for post-event features, both tested against the same set of control features. See Table 3.1 for abbreviations.

Table C.25: t Statistics for two-sided hypothesis tests of the difference in mean autonomic responses during pre- and post-event intervals versus no-event intervals (controls) for each of the components of the physiological features (rows) for quote alerts.

	Low Volatility Events of Single Security P&L															
	Pre-event Interval								Post-event Interval							
Physiological Features	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*
# of SCR responses	-0.42	1.66	-1.66	**-2.12**	-0.53	-0.19	-0.87	-0.92	-0.84	0.94	**2.10**	**-2.67**	-1.93	-0.19	-0.67	-0.32
Ave. SCR amplitude	-0.50	0.32	-1.38	0.75	0.06	-0.24	0.67	-1.32	-1.17	-1.36	-1.87	0.29	-0.98	-1.58	0.09	-0.91
ECG HR	1.33	0.12	**-2.52**	-	0.47	**2.18**	-1.44	-	1.39	-1.24	2.03	-	0.96	-0.39	-1.02	-
Local BVP ratio	0.00	**2.21**	0.75	0.38	0.05	0.22	0.35	-1.35	-0.50	-0.80	0.97	0.05	0.66	0.67	-0.03	-1.10
Global BVP ratio	-0.00	**2.18**	0.67	0.37	0.04	0.19	0.36	-1.35	-0.51	-0.74	0.88	0.04	0.68	0.69	0.05	-1.10
# of Temp jumps	-	**-2.48**	0.36	1.47	0	-	-0.59	0.42	-	1.29	0	1.47	0.81	1.00	1.00	1.47
Ave. SCR duration	-0.42	-0.26	-1.81	0.76	0.14	0.33	0.65	-1.20	-0.97	-1.22	**3.00**	-0.01	-1.75	0.45	0.60	-0.68
SCR area	-0.52	0.49	-1.19	0.39	-0.04	-0.71	1.02	-1.16	-1.44	-1.37	**2.23**	1.66	-1.79	-1.20	-0.54	-0.92
Ave. SCR Δ	-0.45	1.00	-	-0.15	-0.34	-0.17	**2.25**	-1.00	0.00	1.00	-	-1.35	0.13	-0.29	0.36	0
ECG HRV (Std)	-0.38	1.58	0.80	-	1.40	-1.20	**2.70**	-	1.00	0.29	0.27	-	0.99	-0.62	-1.73	-
ECG HR Accel. (RMS)	0.96	0.18	**-2.19**	-	0.71	**2.28**	-1.85	-	1.39	-1.23	1.72	-	1.01	-0.44	-1.27	-
ECG HR Accel. (Std)	0.01	1.17	1.15	-	1.44	-0.88	**2.51**	-	0.86	-0.06	-0.17	-	0.93	0.17	-1.70	-
Range of Temp	0.29	1.57	0.40	1.46	0.47	-0.25	-1.30	1.63	-0.91	**-2.27**	0.74	0.65	1.67	-0.72	-0.19	1.35
Ave. EMG-Arm amplitude	1.18	0.48	-0.19	**-3.64**	-0.26	0.32	0.10	-0.35	1.23	0.36	-0.09	**-2.08**	-0.25	1.07	-0.63	-0.24
EMG-Arm zero-crossings	0.87	1.24	0.39	**-2.83**	-0.66	-1.47	0.43	0	**-2.13**	1.36	1.31	0.94	0.78	-1.87	0.24	-0.71
Ave. EMG-Back amplitude	0.34	1.24	**-2.16**	-0.36	1.93	-0.08	0.86	-0.27	0.69	1.37	1.59	-0.06	1.80	-0.08	-0.03	0.36
EMG-Back zero-crossings	0.17	-0.84	1.00	-	-	1.00	1.00	-	1.21	1.00	1.00	-	-	1.00	1.00	-
BVP HR	1.32	-0.94	**2.60**	-0.69	-0.91	-0.36	0.18	0.79	0.21	-1.24	**2.84**	-1.55	0.20	-0.38	-0.38	0.39
BVP HRV (Std)	1.60	**-2.42**	-0.45	1.01	0.85	-0.37	0.22	-0.07	-0.43	-0.38	-0.40	0.17	-0.46	-0.23	0.23	0.32
BVP HRV (Range)	1.94	**-2.23**	-0.14	0.72	0.94	-0.52	0.34	0.25	-0.58	-0.48	-0.03	-0.24	-0.58	-0.43	0.37	0.34
BVP HR Accel. (RMS)	1.45	-0.74	**2.66**	-0.49	-0.90	-0.52	0.29	0.76	0.20	-1.35	**2.85**	-1.43	0.16	-0.44	-0.30	0.38
BVP HR Accel. (Std)	1.03	**-2.63**	-0.15	0.93	0.48	0.16	0.03	-0.01	-0.58	-0.37	0.29	0.64	-0.61	-0.23	-0.07	0.09
BVP Volatility	-0.04	**2.28**	0.78	-0.11	0.20	0.10	0.40	-1.40	-0.56	-0.47	0.96	-0.06	0.70	0.61	0.10	-1.24

Bolded statistics are significant at the 5% level.

The left panel contains t statistics for pre-event features and the right panel contains t statistics for post-event features, both tested against the same set of control features. See Table 3.1 for abbreviations.

Table C.26: t Statistics for two-sided hypothesis tests of the difference in mean autonomic responses during pre- and post-event intervals versus no-event intervals (controls) for each of the components of the physiological features (rows) for low volatility events of single security P&L.

	High Volatility Events of Single Security P&L															
	Pre-event Interval								Post-event Interval							
Physiological Features	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*
# of SCR responses	-0.66	0.89	-0.66	-1.13	0.51	-1.90	-0.45	0	0	1.21	0.59	0.44	-0.42	-0.66	-1.59	0
Ave. SCR amplitude	-0.08	0.06	1.00	0.37	1.57	-0.30	-0.44	0.76	-0.54	0.24	1.04	1.34	-0.39	0.08	-0.76	0.36
ECG HR	**2.74**	-0.13	1.07	-	-0.04	-0.15	-1.39	-	-0.43	0.75	1.60	-	-0.76	-1.37	-1.79	-
Local BVP ratio	-1.10	-0.10	0.34	**2.27**	-0.92	0.84	**-2.45**	-0.55	-1.03	-0.86	1.22	0.86	-0.69	1.03	**-2.47**	0.90
Global BVP ratio	-1.10	-0.12	0.29	**2.26**	-0.98	0.86	**-2.47**	-0.55	-1.03	-0.86	1.15	0.89	-0.73	1.03	**-2.51**	0.90
# of Temp jumps	-	0.48	0.39	-0.59	-0.36	1.47	-0.59	0.89	-	**-2.07**	-1.09	-0.59	1.09	-	0	1.47
Ave. SCR duration	-0.16	-0.84	0.54	-1.03	1.01	-0.59	-1.53	0.29	-0.12	0.07	0.80	0.90	1.90	-0.22	-0.14	0.14
SCR area	-0.00	-0.54	1.00	-0.33	1.13	-1.31	-1.00	0.88	-0.57	-0.05	1.26	1.08	-0.62	-0.58	-0.95	0.66
Ave. SCR Δ	0	0.40	-	-1.51	0.82	-0.08	-0.88	-1.00	1.00	0.40	-	-1.34	1.16	0.44	0.48	1.47
ECG HRV (Std)	-1.87	-1.07	0.30	-	0.35	-1.20	-0.38	-	-1.82	0.38	0.84	-	-0.81	-1.47	-0.65	-
ECG HR Accel. (RMS)	**2.84**	-0.40	1.02	-	-0.02	-0.24	-1.23	-	-0.80	0.76	1.64	-	-0.86	-1.60	-1.57	-
ECG HR Accel. (Std)	-1.79	-1.21	-0.62	-	0.21	-1.40	-0.58	-	-1.84	-1.84	0.14	-	-0.64	-1.26	-0.94	-
Range of Temp	-0.85	0.16	-1.15	1.00	0.43	0.77	-1.47	0.95	-1.54	0.96	-1.38	0.82	0.68	1.11	**2.24**	0.44
Ave. EMG-Arm amplitude	-0.76	-0.32	-0.45	0.05	0.71	0.18	-1.69	-0.56	-0.10	0.07	0.70	**-2.07**	-0.52	0.43	**2.74**	-0.23
EMG-Arm zero-crossings	-0.36	0.43	-0.88	-0.64	0.53	1.74	-1.38	0.83	-0.27	1.07	-0.57	0.82	-0.74	1.74	**3.22**	0.24
Ave. EMG-Back amplitude	-0.43	0.21	0.43	-0.61	-1.19	1.26	-0.76	-1.42	-0.23	-0.32	0.20	**-2.28**	-1.79	1.95	-2.02	0.69
EMG-Back zero-crossings	-1.45	1.06	1.35	1.60	-	-1.42	1.00	-	-2.00	-0.10	1.35	1.49	-	-1.00	1.00	-
BVP HR	-1.80	0.01	0.58	0.66	0.19	0.07	-1.38	-0.19	-1.62	0.11	1.94	-1.61	0.07	0.18	-1.87	-1.98
BVP HRV (Std)	0.26	0.00	0.94	-1.16	-1.09	-0.78	0.16	-1.33	0.09	0.62	0.15	-0.29	-0.63	-1.00	-1.48	0.62
BVP HRV (Range)	-0.01	-0.41	0.63	-0.97	-1.53	-0.76	0.15	-1.06	-0.17	0.49	-0.27	-0.64	-0.84	-0.59	-1.40	0.46
BVP HR Accel. (RMS)	-1.83	0.05	0.63	0.42	0.14	-0.07	-1.43	-0.34	-1.66	0.21	1.88	-1.68	0.04	-0.01	**2.19**	-1.91
BVP HR Accel. (Std)	0.20	0.01	0.17	-0.91	-0.91	-0.24	-0.57	-1.48	0.03	0.37	-0.57	0.14	-1.06	-0.40	-1.92	0.51
BVP Volatility	-1.13	-0.34	0.28	-1.78	-0.89	1.43	**-2.33**	-0.68	-1.05	-1.17	1.11	0.33	-0.60	0.98	**-2.21**	0.24

Bolded statistics are significant at the 5% level.

The left panel contains t statistics for pre-event features and the right panel contains t statistics for post-event features, both tested against the same set of control features. See Table 3.1 for abbreviations.

Table C.27: t Statistics for two-sided hypothesis tests of the difference in mean autonomic responses during pre- and post-event intervals versus no-event intervals (controls) for each of the components of the physiological features (rows) for high volatility events of single security P&L.

	Low Volatility Events of Overall P&L															
	Pre-event Interval								Post-event Interval							
Physiological Features	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*
# of SCR responses	0.29	-1.25	0.89	-1.20	0.13	-1.85	-0.22	-0.36	-0.95	-1.85	0.55	-0.10	1.19	0.25	0.72	0
Ave. SCR amplitude	-0.93	-0.01	0.55	-0.08	0.68	0.10	1.25	-1.39	-0.67	-0.93	1.38	-1.21	0.02	0.09	1.90	-0.89
ECG HR	-1.14	0.13	-0.04	-	1.15	**2.17**	1.82	-	-1.34	0.49	0.13	-	1.35	-1.67	1.48	-
Local BVP ratio	-1.34	1.94	0.47	0.58	0.74	-1.10	-1.84	0.64	-1.42	1.77	0.20	-0.04	0.26	-0.14	-0.78	0.46
Global BVP ratio	-1.35	1.93	0.47	0.59	0.79	-1.12	-1.89	0.64	-1.43	1.75	0.20	-0.04	0.23	-0.17	-0.80	0.47
# of Temp jumps	-	0	0.81	-1.06	0.89	-1.00	0	0.48	-	0	-0.73	0	-1.12	-1.00	1.00	0
Ave. SCR duration	-0.56	-0.45	-0.30	1.62	0.53	1.34	-1.73	-0.99	-0.65	-1.13	0.25	**-2.50**	-1.96	1.82	-1.09	-0.76
SCR area	-1.23	0.00	0.70	-0.27	0.39	-1.48	0.59	-1.05	-0.50	-0.43	1.11	-0.66	-0.36	-0.32	1.70	-0.67
Ave. SCR Δ	1.20	-	-	-0.76	0.20	-0.92	-1.88	-0.45	0.75	-	-	-0.72	0.47	-0.18	-0.04	1.34
ECG HRV (Std)	-0.05	0.67	0.88	-	0.81	-1.37	1.42	-	-1.24	1.06	0.09	-	0.33	1.02	1.06	-
ECG HR Accel. (RMS)	-0.96	0.42	0.14	-	1.14	**2.35**	1.80	-	-1.35	0.76	0.09	-	1.17	-1.65	1.51	-
ECG HR Accel. (Std)	-0.48	0.83	1.36	-	0.81	-1.32	1.24	-	-1.58	0.98	0.24	-	0.39	1.19	1.15	-
Range of Temp	-1.84	-0.06	-0.89	-0.19	-0.57	0.16	1.06	0.24	-0.65	0.00	-1.24	-0.08	0.19	0.19	1.01	-0.43
Ave. EMG-Arm amplitude	0.73	-0.76	0.61	0.50	0.84	-1.32	-0.04	1.33	-1.01	-0.46	-0.34	0.99	**-2.35**	-0.96	0.58	1.76
EMG-Arm zero-crossings	0.69	0.64	1.98	0.27	0.65	-0.45	0.32	1.43	-0.95	-0.68	0.38	0.17	**-2.38**	-0.59	1.51	1.79
Ave. EMG-Back amplitude	0.64	-0.42	1.01	-0.83	1.71	-1.08	0.41	-0.89	0.67	0.00	1.31	-1.78	1.01	-0.65	1.24	-1.36
EMG-Back zero-crossings	1.00	-	-0.71	-1.00	-1.00	1.00	-1.38	-	1.00	-	-0.54	-1.00	-1.00	1.00	-1.46	-
BVP HR	-0.61	-0.93	-1.46	0.92	-0.43	1.01	1.35	0.32	-0.60	-0.84	-1.75	0.16	1.06	0.20	1.12	1.47
BVP HRV (Std)	0.59	-0.11	-0.10	-0.29	0.07	-1.85	-0.46	-0.87	-0.30	0.91	-0.32	1.31	-0.72	-0.27	-0.02	0.31
BVP HRV (Range)	0.86	-0.08	-0.36	-0.57	0.28	-1.42	-0.49	-0.47	-0.27	0.86	-0.31	1.29	-0.27	0.29	-0.09	0.41
BVP HR Accel. (RMS)	-0.51	-0.90	-1.45	0.81	-0.43	0.78	1.24	0.12	-0.60	-0.68	-1.76	0.40	1.03	0.13	1.13	1.34
BVP HR Accel. (Std)	0.60	0.54	-0.44	0.21	0.83	**2.80**	-1.20	-0.55	-0.13	1.15	-0.11	1.27	-0.12	-0.87	-0.11	0.50
BVP Volatility	-1.37	1.97	0.36	-0.03	0.76	-0.58	-1.91	0.81	-1.45	1.61	0.16	-0.59	0.30	0.04	-0.61	1.16

Bolded statistics are significant at the 5% level.

The left panel contains t statistics for pre-event features and the right panel contains t statistics for post-event features, both tested against the same set of control features. See Table 3.1 for abbreviations.

Table C.28: t Statistics for two-sided hypothesis tests of the difference in mean autonomic responses during pre- and post-event intervals versus no-event intervals (controls) for each of the components of the physiological features (rows) for low volatility events of overall P&L.

	High Volatility Events of Overall P&L															
	Pre-event Interval								Post-event Interval							
Physiological Features	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*
# of SCR responses	-1.26	-0.71	1.38	0	-1.16	0.72	-0.93	**-2.07**	-0.25	0.48	-0.56	0.12	0.89	1.19	-1.92	1.12
Ave. SCR amplitude	-0.95	0.96	1.47	0.52	0.35	1.13	0.19	0.93	0.10	0.57	1.37	1.34	-0.52	1.63	-0.16	-0.71
ECG HR	-0.04	-0.78	-0.37	-	0.37	-0.18	-1.14	-	0.53	-1.11	1.20	-	0.61	-0.88	-1.09	-
Local BVP ratio	1.47	-0.15	-0.19	-0.90	0.30	**-2.61**	1.39	1.16	1.33	0.53	-0.25	-0.94	0.03	**-2.48**	1.44	1.45
Global BVP ratio	1.46	-0.20	-0.19	-0.90	0.25	**-2.60**	1.40	1.16	1.33	0.46	-0.26	-0.96	-0.07	**-2.47**	1.47	1.45
# of Temp jumps	-	-0.89	0.71	-0.59	1.56	1.47	1.00	-1.00	-	0.39	0	-1.47	0	1.00	0	-1.87
Ave. SCR duration	**2.10**	0.04	1.46	**2.33**	-0.52	-0.30	-0.69	0.71	-0.25	0.02	-0.70	-0.53	-0.83	-0.05	0.51	-0.34
SCR area	-0.43	1.34	1.36	1.09	-0.13	0.99	0.46	0.02	0.52	1.55	1.22	1.40	-0.02	1.19	0.05	-1.13
Ave. SCR Δ	-0.59	1.02	-	-1.14	1.19	-1.14	0.47	0.81	-0.28	1.02	-	0.57	1.63	-0.42	1.36	1.41
ECG HRV (Std)	-0.73	0.72	-0.01	-	0.91	1.83	-1.87	-	-1.03	0.67	1.56	-	1.25	1.73	-1.42	-
ECG HR Accel. (RMS)	-0.18	-0.41	-0.32	-	0.51	0.03	-1.33	-	0.04	-0.70	1.32	-	0.76	-0.73	-1.23	-
ECG HR Accel. (Std)	-0.72	0.99	0.77	-	0.88	1.26	-1.79	-	-1.10	1.06	0.34	-	1.14	**-2.34**	-1.42	-
Range of Temp	0.50	0.48	-0.39	0.02	0.80	0.41	-0.50	-0.67	0.26	1.20	0.59	-0.52	-0.38	-0.34	-1.64	-1.48
Ave. EMG-Arm amplitude	-1.53	1.42	-0.71	1.97	-0.23	0.00	-0.84	0.21	-1.51	0.97	-0.17	0.86	0.02	-0.31	-1.06	1.07
EMG-Arm zero-crossings	**2.16**	0.19	-1.56	1.17	-0.32	0.39	-0.39	0.66	**2.23**	1.94	-1.32	0.48	-0.30	1.38	-1.00	1.11
Ave. EMG-Back amplitude	-0.15	-0.45	1.08	1.48	-0.15	**-2.21**	0.14	-0.45	1.37	0.32	0.91	-0.85	-0.12	**-2.90**	-0.44	0.86
EMG-Back zero-crossings	-1.09	-1.24	1.00	0.44	-	-1.38	0.01	-	-0.35	-1.80	-0.47	0.72	-	-1.38	0.02	-
BVP HR	-1.74	-1.32	-0.89	-0.16	-0.29	**2.15**	-0.66	-0.18	-1.42	-1.97	-0.11	-0.49	-0.16	-1.48	-0.57	0.57
BVP HRV (Std)	-0.82	-0.26	-1.10	-1.65	0.30	**-2.99**	-0.28	-0.09	-0.07	0.40	0.86	-1.24	0.58	**-2.67**	-1.39	-0.76
BVP HRV (Range)	-0.87	-0.38	-1.24	-1.73	0.31	**-2.52**	-0.31	0.13	-0.29	0.20	0.64	-1.48	0.50	**-2.80**	-1.33	-0.18
BVP HR Accel. (RMS)	-1.79	-1.39	-1.00	-0.69	-0.27	-1.80	-0.73	-0.24	-1.43	-1.86	-0.08	-0.89	-0.14	-1.22	-0.77	0.44
BVP HR Accel. (Std)	-1.12	-0.00	-1.41	-1.97	0.22	**-2.64**	-0.49	0.10	-0.84	0.65	0.23	-0.97	0.28	1.96	-1.10	-0.16
BVP Volatility	1.51	-0.13	-0.08	-0.25	0.22	**-2.31**	1.40	0.95	1.39	0.43	-0.51	-0.81	-0.02	**-2.45**	1.41	1.50

Bolded statistics are significant at the 5% level.

The left panel contains t statistics for pre-event features and the right panel contains t statistics for post-event features, both tested against the same set of control features. See Table 3.1 for abbreviations.

Table C.29: t Statistics for two-sided hypothesis tests of the difference in mean autonomic responses during pre- and post-event intervals versus no-event intervals (controls) for each of the components of the physiological features (rows) for high volatility events of overall P&L.

	High Volatility Events of Overall P&L	
	Pre-event Interval	**Post-event Interval**
Physiological Features	*Pooled*	*Pooled*
# of SCR responses	0.32	-0.15
Ave. SCR amplitude	1.22	0.65
ECG HR	-0.07	0.18
Local BVP ratio	0.46	0.20
Global BVP ratio	0.45	0.20
# of Temp jumps	1.04	0.13
Ave. SCR duration	-0.28	0.24
SCR area	1.58	0.54
Ave. SCR Δ	1.25	0.70
ECG HRV (Std)	0.36	0.52
ECG HR Accel. (RMS)	-0.06	0.17
ECG HR Accel. (Std)	0.85	0.04
Range of Temp	1.22	0.29
Ave. EMG-Arm amplitude	-0.51	0.36
EMG-Arm zero-crossings	-1.11	-0.94
Ave. EMG-Back amplitude	-0.61	-0.29
EMG-Back zero-crossings	0.70	0.87
BVP HR	-1.60	-0.69
BVP HRV (Std)	0.67	1.10
BVP HRV (Range)	0.65	1.49
BVP HR Accel. (RMS)	-1.53	-0.54
BVP HR Accel. (Std)	0.22	1.08
BVP Volatility	0.36	-0.09

Bolded statistics are significant at the 5% level.

The left panel contains t statistics for pre-event features and the right panel contains t statistics for post-event features, both tested against the same set of control features. See Table 3.1 for abbreviations.

Table C.30: Pooled t Statistics for two-sided hypothesis tests of the difference in mean autonomic responses during pre- and post-event intervals versus no-event intervals (controls) for each of the components of the physiological features (rows) for high volatility events of overall P&L.

	Gain Event of Single Security P&L															
	Pre-event Interval								Post-event Interval							
Physiological Features	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*
# of SCR responses	0	-0.26	-	1.39	0.45	0.92	-1.24	0.39	0.49	0.56	-	1.11	-1.41	0.88	-1.51	-1.15
Ave. SCR amplitude	-1.03	-1.38	-	0.43	0.90	0.44	-1.15	1.14	0.54	0.06	-	0.51	0.47	0.52	-1.10	0.57
ECG HR	-1.76	0.47	0.29	-	0.61	0.29	-1.97	-	-1.70	-0.03	0.38	-	-2.32	0.16	**2.44**	-
Local BVP ratio	1.36	-0.57	0.43	0.41	-0.06	1.26	**-2.14**	-1.16	0.88	0.15	1.42	-0.15	0.15	1.48	**-2.92**	-1.15
Global BVP ratio	1.34	-0.53	0.41	0.39	-0.07	1.33	**-2.16**	-1.16	0.86	0.09	1.39	-0.13	0.16	1.42	**-2.96**	-1.15
# of Temp jumps	-	1.29	0	1.00	1.00	-1.00	-1.03	0.60	-	0	0.61	0	0	0	-0.58	1.48
Ave. SCR duration	-1.56	-0.94	-	**2.00**	0.62	1.28	0.39	0.68	-0.73	-0.16	-	**2.12**	0.77	1.05	0.66	-0.61
SCR area	-0.73	-1.34	-	0.82	0.95	1.33	-0.66	1.33	1.15	0.31	-	1.17	0.68	0.86	-0.55	0.87
Ave. SCR Δ	-0.94	1.00	-	-0.58	0.92	-1.24	0.21	-0.45	-1.69	1.00	-	**2.45**	-1.07	-1.07	0.50	0.56
ECG HRV (Std)	-0.93	0.31	-0.13	-	0.86	-0.31	-0.08	-	-1.02	-0.14	-1.10	-	-0.60	-0.39	-0.82	-
ECG HR Accel. (RMS)	-1.74	0.48	0.24	-	0.72	0.23	-1.59	-	-1.77	-0.05	0.13	-	-1.44	-0.03	**2.05**	-
ECG HR Accel. (Std)	-0.97	-0.02	-0.23	-	0.78	0.01	-0.47	-	-0.89	-0.74	-0.67	-	-0.66	-0.34	-0.95	-
Range of Temp	-0.11	0.83	0.70	-0.48	1.67	-0.91	-1.53	-0.38	0.55	1.37	1.02	-0.52	-0.14	0.83	-0.92	-0.39
Ave. EMG-Arm amplitude	-0.38	-0.88	0.60	0.78	-0.39	1.69	0.12	-0.36	-0.48	-0.24	0.29	-0.21	-1.48	**-3.01**	-0.46	-1.23
EMG-Arm zero-crossings	-0.72	0.36	-1.08	1.63	-	0.39	-0.76	0	-0.75	-0.18	-1.04	0.64	-1.80	1.18	-1.06	-0.81
Ave. EMG-Back amplitude	-0.50	-0.12	1.32	-0.12	3.05	1.26	-0.55	0.22	0.46	-0.23	1.25	0.24	-0.98	1.49	-0.24	0.05
EMG-Back zero-crossings	-1.61	1.00	1.24	-0.44	-	0.94	1.47	-	-1.65	1.00	1.24	-0.14	-	0.72	1.47	-
BVP HR	-1.30	0.06	-0.41	-0.96	-0.06	-0.28	**2.12**	-1.25	-0.80	0.22	-0.61	-0.96	-0.44	-1.42	**2.80**	-0.21
BVP HRV (Std)	1.36	0.81	1.28	**-2.00**	**8.51**	0.46	0.99	0.02	1.56	-0.23	0.60	-0.32	-1.03	1.59	0.06	-0.35
BVP HRV (Range)	1.44	0.72	1.03	1.84	**6.54**	0.23	0.51	0.08	1.62	-0.14	0.86	-0.46	-1.54	1.54	-0.24	-0.25
BVP HR Accel. (RMS)	-1.23	0.21	-0.38	-0.55	-0.07	-0.23	**2.07**	-1.12	-0.73	0.17	-0.59	-1.06	-0.44	-1.28	**2.98**	-0.20
BVP HR Accel. (Std)	1.54	0.89	0.20	1.79	-3.42	0.87	0.73	-0.34	1.63	-0.26	0.51	-0.42	-1.07	**-2.27**	-0.11	-0.83
BVP Volatility	1.36	-0.67	0.18	0.45	-0.06	1.51	**-2.32**	-1.53	0.87	0.07	1.20	0.30	0.20	1.41	**-3.11**	-1.18

Bolded statistics are significant at the 5% level.

The left panel contains t statistics for pre-event features and the right panel contains t statistics for post-event features, both tested against the same set of control features. See Table 3.1 for abbreviations.

Table C.31: t Statistics for two-sided hypothesis tests of the difference in mean autonomic responses during pre- and post-event intervals versus no-event intervals (controls) for each of the components of the physiological features (rows) for gain event of single security P&L.

	Loss Event of Single Security P&L															
	Pre-event Interval								Post-event Interval							
Physiological Features	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*
# of SCR responses	0.21	-0.71	0.45	1.36	3.00	-1.49	**3.13**	0	0.43	-1.13	0	-0.05	1.00	-1.18	**2.35**	1.01
Ave. SCR amplitude	1.96	-0.85	-0.46	0.21	-0.33	-0.00	**2.27**	-1.27	1.43	-1.79	-0.84	0.24	-1.48	0.05	**2.22**	0.93
ECG HR	-1.43	-0.80	0.21	-	-2.17	0.54	-1.76	-	-1.70	-1.47	-0.55	-	-0.99	0.48	-1.09	-
Local BVP ratio	-0.26	-0.94	**-2.40**	0.74	0.64	-1.73	1.38	-0.50	-0.37	-0.83	**-2.90**	-0.53	-0.19	-1.20	1.81	0.19
Global BVP ratio	-0.27	-0.99	**-2.41**	0.76	0.62	-1.74	1.40	-0.53	-0.38	-0.84	**-2.84**	-0.54	-0.18	-1.22	1.88	0.20
# of Temp jumps	-	0.83	-0.63	1.45	0	-1.38	-1.00	1.46	-	-0.48	0	0.23	0	-0.39	-1.44	-0.47
Ave. SCR duration	1.07	-0.10	0.18	-0.38	-0.49	-0.31	1.84	-0.74	0.94	-1.05	-0.14	-1.14	-0.48	-0.26	1.95	1.20
SCR area	1.52	-0.55	0.23	0.04	-0.20	-1.00	**2.25**	-1.42	0.87	-1.89	-0.88	-1.11	-1.74	-0.42	**2.56**	1.15
Ave. SCR Δ	-0.77	1.00	-	0.75	3.13	0.61	-0.19	1.18	0.34	1.00	-	1.15	1.27	0.79	0.05	0.99
ECG HRV (Std)	1.27	1.38	-0.18	-	**4.81**	-1.10	-0.93	-	1.31	0.18	-0.59	-	-2.18	-0.05	-0.30	-
ECG HR Accel. (RMS)	-0.80	-0.72	0.21	-	-2.18	0.22	-1.42	-	-0.97	-1.41	-0.56	-	-1.04	0.52	-0.79	-
ECG HR Accel. (Std)	0.95	1.71	2.43	-	-0.18	-0.39	-1.09	-	0.97	0.12	1.01	-	-1.81	0.20	-0.38	-
Range of Temp	-0.27	0.51	0.46	1.50	0.11	0.64	-1.04	-0.15	0.59	-0.53	-0.53	0.52	1.05	0.62	**2.44**	-0.74
Ave. EMG-Arm amplitude	0.26	0.71	1.02	**-2.20**	-1.13	**2.24**	-1.90	-0.74	0.75	0.61	0.90	1.30	-1.93	-1.69	-0.67	-1.04
EMG-Arm zero-crossings	0.11	-0.28	1.51	1.85	-1.00	-1.31	-1.96	-0.92	0.38	-0.51	1.92	1.08	-1.67	**2.05**	-1.36	-0.56
Ave. EMG-Back amplitude	1.03	0.24	1.25	-0.00	-0.19	-0.36	-1.66	-0.53	-0.61	0.21	1.49	-0.15	-0.51	-0.52	-1.13	-0.43
EMG-Back zero-crossings	-0.11	-1.00	1.00	-0.59	-	-0.58	1.00	-	-0.45	-1.06	1.00	0.65	-	-1.07	1.00	-
BVP HR	0.01	-0.64	**-2.44**	**2.00**	-2.09	**-2.54**	-1.35	-0.92	0.02	-1.70	1.92	-0.31	-1.09	1.70	-1.63	-0.13
BVP HRV (Std)	0.91	-0.01	0.50	1.46	-0.97	-0.79	-1.39	0.51	0.68	0.40	0.25	1.02	-1.39	0.06	-0.04	1.48
BVP HRV (Range)	1.11	-0.11	0.47	1.00	-0.66	-0.53	-1.28	0.79	0.63	0.33	0.00	1.11	-2.40	0.04	-0.14	1.26
BVP HR Accel. (RMS)	0.08	-0.64	**-2.43**	-1.77	-2.11	**-2.61**	-1.60	-0.85	0.08	-1.56	1.92	-0.13	-1.11	1.84	-1.70	0.07
BVP HR Accel. (Std)	0.47	-0.48	0.96	1.12	-1.31	-0.75	-1.33	0.81	0.49	0.26	0.20	0.25	-0.33	0.06	0.32	1.23
BVP Volatility	-0.25	-0.93	**-2.50**	0.84	0.71	-1.73	1.79	-0.57	-0.40	-0.68	**-2.83**	-0.22	-0.16	-1.52	1.79	0.32

Bolded statistics are significant at the 5% level.

The left panel contains t statistics for pre-event features and the right panel contains t statistics for post-event features, both tested against the same set of control features. See Table 3.1 for abbreviations.

Table C.32: t Statistics for two-sided hypothesis tests of the difference in mean autonomic responses during pre- and post-event intervals versus no-event intervals (controls) for each of the components of the physiological features (rows) for loss event of single security P&L.

	Gain Event of Overall P&L															
	Pre-event Interval								Post-event Interval							
Physiological Features	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*
# of SCR responses	0.95	-0.25	-1.51	0.35	0.59	0.15	-0.09	**-2.53**	-1.02	-0.65	-1.00	**-2.83**	-0.85	0.43	-1.41	0
Ave. SCR amplitude	1.45	-1.16	-1.22	-0.31	0.33	0.64	0.08	**-2.19**	-0.52	-0.02	-1.00	-0.04	-1.08	0.82	-0.83	0.70
ECG HR	-0.08	-0.82	1.60	-	1.28	0.21	**2.62**	-	-0.52	0.16	**-2.87**	-	0.67	0.69	**2.24**	-
Local BVP ratio	1.41	1.94	1.36	-1.56	-0.53	1.01	1.39	1.16	1.03	1.51	**-2.31**	-0.06	-0.03	0.10	0.82	0.22
Global BVP ratio	1.41	1.98	1.30	-1.58	-0.55	1.02	1.48	1.16	1.01	1.51	**-2.29**	-0.09	-0.03	-0.02	0.89	0.25
# of Temp jumps	-	0.80	-1.11	-1.19	0	-0.58	-0.59	0	-	0.80	-0.60	-1.72	-1.04	-0.58	1.00	1.00
Ave. SCR duration	1.38	-0.65	-1.42	0.39	-1.78	-1.32	-0.52	**-2.16**	-0.75	-0.24	-1.00	-0.13	-1.21	-1.74	0.46	0.40
SCR area	1.77	-0.73	-1.12	0.66	0.30	0.21	0.40	1.82	-0.37	0.06	-1.00	0.58	-1.95	0.99	-0.42	0.32
Ave. SCR Δ	0.39	-	-	0.39	1.06	1.55	-0.17	1.00	-0.70	-	-	-0.50	-0.44	-0.30	-0.27	1.34
ECG HRV (Std)	1.76	-0.24	-1.01	-	1.59	-0.43	-1.67	-	1.30	0.46	-1.03	-	0.69	-0.68	**2.08**	-
ECG HR Accel. (RMS)	0.25	-0.80	1.22	-	1.36	0.04	**2.38**	-	-0.23	0.18	**-2.73**	-	0.70	0.56	**2.22**	-
ECG HR Accel. (Std)	1.68	-1.03	**2.31**	-	1.64	-0.19	-1.38	-	1.48	-1.03	0.93	-	0.54	-0.16	-1.73	-
Range of Temp	1.06	1.66	-0.41	-0.50	0.13	1.02	0.09	-0.28	0.11	1.63	-0.34	0.35	0.43	1.11	0.35	0.61
Ave. EMG-Arm amplitude	**-2.06**	-0.81	**-2.36**	-0.08	0.87	-0.19	0.55	-0.03	1.71	0.55	1.22	0.69	-0.16	-0.19	0.28	-0.18
EMG-Arm zero-crossings	1.97	-1.84	0.97	-0.35	1.14	-0.91	0.47	0.26	1.44	-1.26	0.63	1.28	0	0	0.19	0.13
Ave. EMG-Back amplitude	0.12	-1.56	1.66	-0.38	-1.58	0.24	0.62	0.95	-0.44	-1.21	1.67	0.98	-1.84	-0.55	-0.59	0.55
EMG-Back zero-crossings	-0.64	-	1.44	-0.40	-	0	-	-	1.46	-1.00	1.44	-0.88	-	-0.74	-	-
BVP HR	-0.37	-0.71	-0.97	-0.24	-0.19	0.69	-1.14	-1.45	-0.19	0.20	-0.99	-1.35	-0.43	0.28	-0.20	-2.00
BVP HRV (Std)	**-2.31**	-0.26	0.20	0.10	0.93	0.01	0.41	-0.91	1.03	0.27	-0.02	0.38	0.49	-0.08	0.02	-0.27
BVP HRV (Range)	**-2.44**	-0.57	-0.25	-0.11	0.37	0.02	0.13	-0.85	0.98	-0.01	0.03	0.47	-0.03	-0.34	0.01	-0.18
BVP HR Accel. (RMS)	-0.22	-0.69	-0.94	-0.21	-0.16	0.81	-1.09	-1.46	-0.09	0.20	-0.97	-1.35	-0.40	0.34	-0.19	-1.84
BVP HR Accel. (Std)	**-2.35**	-0.56	-0.88	0.18	1.42	-0.04	0.50	-0.69	0.48	0.07	-1.55	0.19	0.71	-0.48	0.04	-0.24
BVP Volatility	1.35	2.02	1.10	-1.84	-0.46	1.20	1.78	1.18	0.95	1.64	2.05	-0.24	0.08	0.06	1.24	0.39

Bolded statistics are significant at the 5% level.

The left panel contains t statistics for pre-event features and the right panel contains t statistics for post-event features, both tested against the same set of control features. See Table 3.1 for abbreviations.

Table C.33: t Statistics for two-sided hypothesis tests of the difference in mean autonomic responses during pre- and post-event intervals versus no-event intervals (controls) for each of the components of the physiological features (rows) for gain event of overall P&L.

	Loss Event of Overall P&L															
	Pre-event Interval								Post-event Interval							
Physiological Features	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*
# of SCR responses	-0.61	0.86	0.44	1.49	-0.65	0.52	-0.19	-1.05	-0.78	1.19	-0.34	0.92	-0.87	1.86	-0.47	-1.84
Ave. SCR amplitude	0.81	1.84	0.83	-1.45	-2.01	-0.61	-1.28	-1.11	0.65	1.81	0.35	-1.98	**2.79**	0.97	-1.08	-1.51
ECG HR	-0.86	0.89	-0.28	-	0.24	1.27	-1.22	-	-0.66	1.32	-0.50	-	0.14	0.80	-0.46	-
Local BVP ratio	0.64	-0.20	1.91	-0.12	0.17	1.39	0.00	-0.11	0.15	-1.39	1.78	0.06	0.33	0.38	0.57	-0.44
Global BVP ratio	0.65	-0.21	1.91	-0.14	0.09	1.33	-0.01	-0.11	0.14	-1.35	1.77	0.05	0.27	0.28	0.43	-0.44
# of Temp jumps	-	1.84	**-3.29**	-0.35	-0.65	-	1.04	0	-	0	**-4.58**	-0.74	-1.41	1.44	1.04	-0.59
Ave. SCR duration	0.04	**-2.22**	0.19	1.67	-1.76	0.19	-0.80	-1.19	-0.35	**-2.67**	-0.57	0.13	-1.51	1.71	-1.21	-1.42
SCR area	0.33	1.67	1.06	0.27	**3.67**	-1.02	-0.81	-1.06	-0.30	1.28	-0.50	-0.57	**2.71**	0.78	-0.69	-1.13
Ave. SCR Δ	-0.85	0.96	-	-0.33	-1.05	0.21	-0.30	0	0.84	0.96	-	0.52	-0.96	-0.43	-0.48	1.77
ECG HRV (Std)	-0.11	0.28	-0.20	-	1.18	0.41	-1.80	-	-1.89	0.13	-0.42	-	0.94	1.42	-0.27	-
ECG HR Accel. (RMS)	-0.87	0.87	-0.32	-	0.52	1.17	-1.35	-	-0.97	1.27	-0.63	-	0.42	1.03	-0.41	-
ECG HR Accel. (Std)	-0.39	0.61	-0.95	-	1.13	0.91	-1.76	-	-1.96	0.79	-1.14	-	0.98	1.30	-0.24	-
Range of Temp	-0.63	0.21	1.80	-0.99	0.73	-0.28	1.36	-0.56	0.74	-0.24	2.00	-1.34	-0.84	0.38	0.96	-1.12
Ave. EMG-Arm amplitude	-0.94	-0.19	-1.35	0.99	0.89	-0.36	-0.11	-1.41	-1.05	-0.56	**2.56**	0.46	0.82	0.96	0.79	-0.84
EMG-Arm zero-crossings	-1.47	1.00	-0.04	0.33	1.11	-0.50	-0.93	-0.09	-1.38	0.64	-1.00	-0.15	0.95	1.12	0.15	-0.26
Ave. EMG-Back amplitude	0.33	0.87	-0.33	0.99	1.72	**-2.46**	1.52	-0.37	-0.45	0.11	-0.71	0.38	1.58	1.84	1.82	-1.18
EMG-Back zero-crossings	-1.79	-1.00	-1.00	0.86	-	1.44	-1.00	-	-1.93	-1.00	-1.00	1.70	-	1.44	-1.00	-
BVP HR	-1.30	-0.37	1.55	0.99	-0.61	-0.20	0.14	0.03	-0.83	-0.01	0.88	0.93	-0.62	-0.24	-0.38	-0.60
BVP HRV (Std)	-1.20	1.25	0.65	-1.08	1.27	0.51	-0.33	-1.27	-0.72	2.03	1.78	-1.15	0.46	1.14	-0.24	-1.43
BVP HRV (Range)	-0.70	1.24	0.51	-0.95	0.79	0.10	-0.47	-0.84	-0.90	**-2.17**	1.70	-1.09	0.03	0.63	-0.30	-0.88
BVP HR Accel. (RMS)	-1.53	-0.16	1.56	0.81	-0.60	-0.07	0.18	-0.09	-0.87	0.26	0.90	0.74	-0.62	-0.08	-0.39	-0.74
BVP HR Accel. (Std)	-1.49	1.45	1.95	-0.58	1.49	0.07	-0.93	-1.29	-1.49	**-2.09**	1.20	-1.00	1.66	0.08	-0.63	-1.12
BVP Volatility	0.64	-0.34	2.01	-0.04	0.01	1.05	-0.20	0.12	0.12	-1.49	1.82	0.32	0.17	0.11	0.41	-0.34

Bolded statistics are significant at the 5% level.

The left panel contains t statistics for pre-event features and the right panel contains t statistics for post-event features, both tested against the same set of control features. See Table 3.1 for abbreviations.

Table C.34: t Statistics for two-sided hypothesis tests of the difference in mean autonomic responses during pre- and post-event intervals versus no-event intervals (controls) for each of the components of the physiological features (rows) for loss event of overall P&L.

	Gain vs. Loss Events of Single Security P&L															
	Pre-event Interval								Post-event Interval							
Physiological Features	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*
# of SCR responses	0.99	-0.48	-	-1.14	-	0.58	0.68	0.24	-0.23	-0.16	-0.73	0.55	-	-0.65	-0.06	-1.62
Ave. SCR amplitude	-1.21	-1.70	-	0.37	-	**-2.02**	0.47	0.71	0.39	0.71	-0.73	-0.91	-	0.90	1.13	-1.66
ECG HR	-0.92	-0.43	0.35	-	-	-0.13	0.40	-	-0.97	-0.50	-0.81	-	-	-0.81	0.49	-
Local BVP ratio	0.20	-0.41	-0.72	**3.69**	-	1.00	0.83	0.86	0.12	0.37	0.16	0.15	-	0.77	0.86	1.07
Global BVP ratio	0.18	-0.32	-0.75	**3.72**	-	1.02	0.85	0.85	0.11	0.30	0.17	0.15	-	0.77	0.86	1.07
# of Temp jumps	-	0.78	2.10	-0.20	-	0.13	-0.88	-0.29	-	0.43	0.42	0.57	-	0.51	0.74	1.18
Ave. SCR duration	-0.22	-1.04	-	-0.94	-	0.80	0.55	0.29	0.15	0.20	-0.73	-1.45	-	-0.76	-1.94	-1.82
SCR area	-1.10	**2.09**	-	0.06	-	1.52	0.19	0.88	0.72	0.76	-0.73	-0.50	-	0.33	0.75	**2.32**
Ave. SCR Δ	1.10	-	-	1.21	-	-1.88	-0.45	-1.21	-0.85	-	-	**2.44**	-	-0.75	-0.16	-0.66
ECG HRV (Std)	-1.33	-1.82	-0.07	-	-	-0.21	0.86	-	-1.39	-0.71	-0.83	-	-	-1.88	0.15	-
ECG HR Accel. (RMS)	-1.19	-0.48	0.27	-	-	-0.15	0.45	-	-1.38	-0.54	-0.94	-	-	-1.24	0.39	-
ECG HR Accel. (Std)	-1.57	-0.55	-0.64	-	-	-0.21	1.33	-	-1.27	-0.39	-1.68	-	-	-1.38	0.37	-
Range of Temp	-1.32	-0.18	0.76	-1.97	-	0.79	-1.00	-0.67	0.69	0.24	0.67	0.85	-	-0.08	0.46	-0.17
Ave. EMG-Arm amplitude	1.19	-1.22	1.63	-0.55	-	1.07	**-2.12**	0.54	0.74	-0.33	0.19	-0.54	-	1.02	-0.26	-0.19
EMG-Arm zero-crossings	0.84	-0.68	1.48	-0.89	-	1.20	1.52	1.11	0.77	-1.22	-1.12	0.40	-	1.33	-0.03	-0.09
Ave. EMG-Back amplitude	-1.26	-0.81	-0.21	-1.48	-	1.23	1.35	0.65	-0.22	-1.36	-1.01	-0.01	-	0.81	0.62	0.47
EMG-Back zero-crossings	0.24	1.00	1.39	0.69	-	0.47	-	-	-0.24	1.48	1.39	-0.82	-	-0.13	-	-
BVP HR	0.47	-1.04	-0.32	0.98	-	-1.43	-0.17	**3.01**	0.44	0.08	0.05	-1.96	-	-0.41	1.05	0.02
BVP HRV (Std)	1.00	0.11	-0.13	-1.43	-	1.29	1.83	0.12	0.96	-1.51	-0.58	-0.42	-	0.60	-0.16	0.35
BVP HRV (Range)	0.83	0.28	0.06	-1.21	-	1.05	1.77	-0.45	0.89	-1.27	-0.07	-1.04	-	0.70	0.02	0.57
BVP HR Accel. (RMS)	0.62	-1.01	-0.34	0.73	-	-1.37	0.23	**2.78**	0.51	-0.17	0.03	**2.14**	-	-0.33	1.13	0.05
BVP HR Accel. (Std)	1.19	0.67	-0.71	-1.26	-	1.22	1.67	-0.22	0.91	-1.81	-0.56	-0.24	-	0.46	-0.28	0.14
BVP Volatility	0.24	-0.32	-0.72	**3.92**	-	0.87	0.79	0.80	0.15	0.48	0.17	0.06	-	0.93	1.18	0.86

Bolded statistics are significant at the 5% level.

The left panel contains t statistics for pre-event features and the right panel contains t statistics for post-event features. See Table 3.1 for abbreviations.

Table C.35: t Statistics for two-sided hypothesis tests of the difference in mean autonomic responses during pre- and post-event intervals for each of the components of the physiological features (rows) for gain vs. loss events of single security P&L.

	Gain Events of Single Security P&L (Head to Head)							
Physiological Features	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*
# of SCR responses	-0.52	-0.91	-	0.30	2.24	0.07	0.63	1.63
Ave. SCR amplitude	-1.61	-1.48	-	-0.07	0.82	-0.06	-0.11	1.57
ECG HR	-0.24	0.59	-0.06	-	1.48	0.11	0.51	-
Local BVP ratio	0.36	-0.96	-0.78	0.55	-0.18	-0.28	-0.14	-0.07
Global BVP ratio	0.36	-0.82	-0.76	0.52	-0.18	-0.15	-0.15	-0.07
# of Temp jumps	-	1.29	-0.61	1.00	1.00	-1.00	-0.46	-1.00
Ave. SCR duration	-1.01	-0.89	-	0.02	-0.71	0.35	-0.41	1.63
SCR area	**2.06**	-1.58	-	-0.26	1.78	0.31	-0.14	1.86
Ave. SCR Δ	0.39	-	-	1.92	1.41	-0.27	-0.37	-0.80
ECG HRV (Std)	0.06	0.37	0.88	-	0.93	0.12	0.71	-
ECG HR Accel. (RMS)	-0.16	0.62	0.21	-	1.54	0.23	0.53	-
ECG HR Accel. (Std)	-0.18	0.70	0.41	-	0.92	0.36	0.45	-
Range of Temp	-0.68	-0.43	-0.44	-0.03	0.79	**2.23**	-0.47	0.00
Ave. EMG-Arm amplitude	0.15	-0.62	0.21	0.98	1.23	-1.22	0.65	0.83
EMG-Arm zero-crossings	0.05	0.70	0.04	0.95	1.80	-0.70	0.44	0.79
Ave. EMG-Back amplitude	-0.85	0.09	1.28	-0.31	1.02	-0.45	-0.28	0.26
EMG-Back zero-crossings	0.21	-	-	-0.29	-	0.20	-	-
BVP HR	-0.48	-0.22	0.24	0.01	0.29	1.18	0.39	-1.37
BVP HRV (Std)	-0.53	1.15	1.13	**-2.36**	-1.20	-1.13	0.95	0.43
BVP HRV (Range)	-0.48	1.00	0.26	**-2.33**	-1.36	-1.25	0.75	0.39
BVP HR Accel. (RMS)	-0.48	0.01	0.25	0.53	0.29	1.09	0.57	-1.28
BVP HR Accel. (Std)	-0.33	1.35	-0.51	**-2.25**	-0.04	-1.41	0.86	0.48
BVP Volatility	0.37	-1.01	-0.81	0.16	-0.20	0.09	-0.20	-0.35

Bolded statistics are significant at the 5% level.

The panel contains t statistics for pre-event features tested against post-event features. See Table 3.1 for abbreviations.

Table C.36: t Statistics for two-sided hypothesis tests of the difference in mean autonomic responses during pre- and post-event intervals for each of the components of the physiological features (rows) for gain events of single security P&L.

	Loss Events of Single Security P&L (Head to Head)							
Physiological Features	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*
# of SCR responses	-0.41	-0.18	-	**-2.37**	-	-1.15	0.10	0
Ave. SCR amplitude	0.25	0.30	-	-0.03	-	-0.14	1.16	-0.82
ECG HR	-0.14	0.10	-0.74	-	-	0.08	0.04	-
Local BVP ratio	0.03	0.05	0.26	1.13	-	-0.85	0.02	-0.53
Global BVP ratio	0.02	0.02	0.31	1.13	-	-0.84	0.00	-0.57
# of Temp jumps	-	0.43	-1.26	1.27	-	-0.46	0.59	0.59
Ave. SCR duration	-0.42	0.00	-	0.98	-	-0.83	-1.85	-0.24
SCR area	0.45	0.84	-	1.56	-	-1.11	0.79	-1.09
Ave. SCR Δ	-1.26	-	-	-0.73	-	-0.42	1.00	-0.23
ECG HRV (Std)	-0.33	0.96	-0.37	-	-	-0.35	-0.30	-
ECG HR Accel. (RMS)	-0.25	0.14	-0.72	-	-	-0.02	-0.08	-
ECG HR Accel. (Std)	-0.07	0.58	-0.03	-	-	0.09	-0.40	-
Range of Temp	-0.46	-0.00	-0.36	1.45	-	0.09	0.83	-0.17
Ave. EMG-Arm amplitude	-0.11	0.22	-0.10	1.30	-	-1.02	-1.17	0.39
EMG-Arm zero-crossings	0.03	-0.28	-1.10	1.69	-	0.84	-0.82	-0.40
Ave. EMG-Back amplitude	0.87	-0.28	-0.57	0.15	-	0.50	-0.96	-0.29
EMG-Back zero-crossings	0.61	-0.88	-0.34	-0.34	-	-0.56	-	-
BVP HR	-0.21	0.52	0.62	-1.82	-	0.82	-0.06	0.51
BVP HRV (Std)	0.17	-0.59	-0.59	0.65	-	-0.79	-0.88	-0.56
BVP HRV (Range)	0.38	-0.64	-0.03	0.31	-	-0.34	-0.80	0.14
BVP HR Accel. (RMS)	-0.23	0.44	0.61	-1.82	-	0.75	-0.24	0.35
BVP HR Accel. (Std)	-0.11	-1.31	0.38	0.67	-	-0.75	-1.06	-0.26
BVP Volatility	0.04	0.09	0.31	0.82	-	-0.56	0.20	-0.66

Bolded statistics are significant at the 5% level.

The panel contains t statistics for pre-event features tested against post-event features. See Table 3.1 for abbreviations.

Table C.37: t Statistics for two-sided hypothesis tests of the difference in mean autonomic responses during pre- and post-event intervals for each of the components of the physiological features (rows) for loss events of single security P&L.

	Gain vs. Loss Events of Overall P&L															
	Pre-event Interval								Post-event Interval							
Physiological Features	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*
# of SCR responses	-0.15	-0.96	1.81	-1.77	-	1.26	0.79	-0.37	-1.61	1.16	0.24	0.99	-	-0.58	1.41	-0.18
Ave. SCR amplitude	-0.74	-0.78	1.78	0.84	-	**-2.81**	0.94	0.31	-1.42	0.09	-0.75	0.35	-	-0.40	**-2.51**	0.74
ECG HR	-0.74	0.35	0.76	-	-	1.02	1.00	-	-1.50	-1.60	1.90	-	-	0.21	-0.30	-
Local BVP ratio	0.87	0.57	-0.57	-0.67	-	1.31	0.00	1.78	0.89	0.90	-0.08	**-2.17**	-	0.54	-0.13	-0.67
Global BVP ratio	0.87	0.63	-0.58	-0.69	-	1.37	0.01	1.78	0.88	0.90	-0.14	**-2.18**	-	0.52	-0.12	-0.67
# of Temp jumps	-	0.47	0.24	-0.45	-	1.05	1.68	-1.10	-	-0.02	-1.29	-1.46	-	0.06	-0.35	0.15
Ave. SCR duration	-0.32	-0.58	1.97	**2.32**	-	1.54	0.60	0.14	-1.36	0.49	-0.07	-0.73	-	**2.01**	-2.03	0.47
SCR area	-0.80	-1.51	1.97	-0.71	-	**-2.86**	1.41	0.55	-1.97	-0.26	-0.78	0.55	-	-0.05	1.81	0.66
Ave. SCR Δ	-0.43	-0.92	-	-0.03	-	0.48	-0.12	-1.31	-1.13	-1.16	-	**2.77**	-	0.50	-1.33	-0.62
ECG HRV (Std)	-0.23	1.29	0.46	-	-	1.19	1.49	-	-1.41	-1.03	1.28	-	-	1.00	-0.64	-
ECG HR Accel. (RMS)	-0.69	0.42	0.75	-	-	1.20	1.20	-	-1.64	-1.66	2.04	-	-	0.35	-0.40	-
ECG HR Accel. (Std)	-0.16	0.70	-1.46	-	-	1.30	1.83	-	-1.35	-1.12	**-2.29**	-	-	0.83	-0.49	-
Range of Temp	-0.50	-0.21	0.99	-0.56	-	0.66	0.11	-1.45	0.78	-0.10	-0.66	1.55	-	-0.36	0.03	0.29
Ave. EMG-Arm amplitude	0.95	**-2.34**	1.39	**2.13**	-	1.78	0.11	0.51	-1.31	1.78	**-2.71**	0.45	-	0.07	0.41	0.17
EMG-Arm zero-crossings	0.40	0.38	1.33	-0.71	-	0.97	0.18	0.45	-0.71	-0.38	1.33	0.55	-	0.75	0.29	0.38
Ave. EMG-Back amplitude	0.28	-0.97	0.73	-0.43	-	0.75	-0.63	0.84	0.03	-1.32	1.64	1.46	-	0.13	1.01	0.20
EMG-Back zero-crossings	0.61	-0.78	-	0.16	-	-1.05	0.80	-	-0.93	0.86	-	-0.20	-	-0.88	0.40	-
BVP HR	0.13	0.53	0.72	0.79	-	-1.26	-0.05	-0.98	0.81	-0.38	-0.77	-1.50	-	-0.72	0.85	-0.19
BVP HRV (Std)	1.90	-0.69	**-4.05**	0.63	-	0.65	-1.03	0.90	-0.75	-1.69	0.04	1.71	-	0.61	1.01	0.01
BVP HRV (Range)	1.80	-0.89	**-3.63**	0.95	-	0.29	-0.90	0.92	-0.74	-1.80	0.38	1.36	-	0.64	1.32	0.07
BVP HR Accel. (RMS)	0.42	0.48	0.82	0.96	-	-1.25	-0.24	-0.83	0.75	-0.66	-0.77	-1.16	-	-0.67	1.06	-0.23
BVP HR Accel. (Std)	1.83	-1.00	1.99	0.86	-	0.98	-0.65	1.22	-0.36	-1.45	0.49	1.49	-	0.58	1.28	-0.18
BVP Volatility	0.92	0.57	-0.60	-0.60	-	1.66	0.10	1.68	0.97	1.25	-0.12	1.99	-	0.96	0.17	-0.67

Bolded statistics are significant at the 5% level.

The left panel contains t statistics for pre-event features and the right panel contains t statistics for post-event features for testing Gain vs. Loss Events of Overall P&L. See Table 3.1 for abbreviations.

Table C.38: t Statistics for two-sided hypothesis tests of the difference in mean autonomic responses during pre- and post-event intervals for each of the components of the physiological features (rows) for gain vs. loss events of overall P&L.

	Gain Events of Overall P&L (Head to Head)							
Physiological Features	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*
# of SCR responses	1.54	0	1.00	**2.45**	-	0	-0.40	1.25
Ave. SCR amplitude	0.18	0.11	1.00	-0.06	-	1.02	-0.98	0.15
ECG HR	0.82	1.02	0.61	-	-	0.06	0.28	-
Local BVP ratio	0.15	-0.05	0.04	**2.47**	-	-0.30	0.49	1.15
Global BVP ratio	0.16	-0.05	0.05	**2.48**	-	-0.26	0.47	1.15
# of Temp jumps	-	0.25	0.61	1.03	-	0	1.45	-0.60
Ave. SCR duration	0.78	0.50	1.00	-0.21	-	1.26	1.38	0.86
SCR area	0.74	0.55	1.00	-0.87	-	0.43	-0.17	0.24
Ave. SCR Δ	0.48	1.00	-	1.68	-	1.33	0.70	-1.34
ECG HRV (Std)	0.83	1.66	0.72	-	-	0.12	0.67	-
ECG HR Accel. (RMS)	0.88	1.18	0.59	-	-	0.10	0.45	-
ECG HR Accel. (Std)	0.52	1.53	-0.99	-	-	0.48	0.79	-
Range of Temp	-0.42	-0.09	0.17	-0.75	-	-0.06	-0.46	-1.31
Ave. EMG-Arm amplitude	0.89	0.43	-0.20	-1.56	-	0.23	-0.60	0.16
EMG-Arm zero-crossings	0.23	0.26	-0.42	-1.19	-	-0.91	-0.89	0.24
Ave. EMG-Back amplitude	0.64	0.60	0.29	**2.14**	-	-0.07	-1.50	0.62
EMG-Back zero-crossings	0.98	-1.05	-	0.71	-	0.82	0.55	-
BVP HR	0.04	0.39	**-2.39**	1.56	-	0.80	-1.51	0.21
BVP HRV (Std)	1.48	0.81	1.50	-1.01	-	-0.27	-1.82	0.33
BVP HRV (Range)	1.45	0.85	1.37	-0.60	-	0.05	**2.10**	0.32
BVP HR Accel. (RMS)	0.16	0.52	**-2.42**	1.43	-	0.77	-1.83	0.28
BVP HR Accel. (Std)	1.38	0.19	1.59	-0.55	-	0.14	-1.80	0.63
BVP Volatility	0.18	-0.20	0.09	**2.39**	-	-0.05	0.29	1.17

Bolded statistics are significant at the 5% level.

The panel contains t statistics for pre-event features tested against post-event features. See Table 3.1 for abbreviations.

Table C.39: t Statistics for two-sided hypothesis tests of the difference in mean autonomic responses during pre- and post-event intervals for each of the components of the physiological features (rows) for gain events of overall P&L.

	Loss Events of Overall P&L (Head to Head)							
Physiological Features	*15001*	*15002*	*15003*	*15004*	*15005*	*15006*	*15007*	*15008*
# of SCR responses	0.30	**-2.12**	-1.21	0.32	-	-1.76	0.11	**-2.27**
Ave. SCR amplitude	-0.17	1.01	-1.14	-0.79	-	**2.23**	0.99	0.29
ECG HR	0.42	-0.99	1.38	-	-	-0.79	-0.97	-
Local BVP ratio	0.22	0.30	0.61	0.06	-	-0.88	0.23	0.25
Global BVP ratio	0.22	0.25	0.61	0.06	-	-0.91	0.22	0.26
# of Temp jumps	-	-0.24	-1.00	0	-	-1.00	-0.59	0.59
Ave. SCR duration	-0.14	1.70	-0.72	0.91	-	**2.31**	-1.90	1.01
SCR area	-0.10	1.80	-1.51	0.36	-	**2.72**	0.62	0.15
Ave. SCR Δ	-0.25	0.69	-	-0.72	-	0.84	-0.73	-1.47
ECG HRV (Std)	-0.18	0.37	1.23	-	-	-0.33	-1.35	-
ECG HR Accel. (RMS)	0.33	-0.98	1.55	-	-	-0.83	-1.05	-
ECG HR Accel. (Std)	-0.58	0.96	2.17	-	-	-0.24	-1.35	-
Range of Temp	0.88	0.02	-1.58	1.39	-	-1.18	-0.55	-0.20
Ave. EMG-Arm amplitude	-1.41	-0.15	0.34	0.95	-	-1.47	-0.27	-0.15
EMG-Arm zero-crossings	-0.94	-0.53	-0.25	0.05	-	-1.51	-0.74	0.28
Ave. EMG-Back amplitude	0.49	0.15	1.37	-0.51	-	-0.68	0.10	0.44
EMG-Back zero-crossings	-0.44	0.79	-	0.59	-	1.00	0.09	-
BVP HR	0.74	-0.53	0.81	-0.61	-	1.24	-0.59	1.25
BVP HRV (Std)	-1.56	-0.04	-1.99	-0.00	-	-0.41	0.31	-0.51
BVP HRV (Range)	-1.42	0.12	-1.56	-0.29	-	0.36	0.24	-0.43
BVP HR Accel. (RMS)	0.54	-0.62	0.74	-0.66	-	1.20	-0.51	1.12
BVP HR Accel. (Std)	-1.49	-0.11	-0.75	0.04	-	-0.42	0.22	-0.58
BVP Volatility	0.27	0.44	0.70	-0.18	-	-0.43	0.23	0.24

Bolded statistics are significant at the 5% level.

The panel contains t statistics for pre-event features tested against post-event features. See Table 3.1 for abbreviations.

Table C.40: t Statistics for two-sided hypothesis tests of the difference in mean autonomic responses during pre- and post-event intervals for each of the components of the physiological features (rows) for loss events of overall P&L.

Appendix D

Source Code

```
function fixbidask(tickerlist)
% fixbidask
%  tickerlist is a comma-delimited list of quoted strings, ie who('*')
%
%  fixes the inverted bid-ask spreads from tickers in tickerlist
%  also, updates the unrealizedpl and pl (profit and loss) columns

setglobal

for i = 1:length(tickerlist)
  qq = eval(char(tickerlist(i)));

% $$$   % key
% $$$   index = qq(:,1);
% $$$   bid = qq(:,2);
% $$$   ask = qq(:,3);
% $$$   position = qq(:,5);
% $$$   avetransactionprice = qq(:,6);
% $$$   realizedpl = qq(:,9);
% $$$   unrealizedpl = qq(:,10);
% $$$   pl = qq(:,11);

  spread = qq(qq(:,2) > qq(:,3),1);
  while( length(spread) > 0)
    idx = find(qq(:,1) == spread(1));
    while( qq(idx,2) > qq(idx,3))
      if( qq(idx,2) > qq(idx-1,2))
        % fix bid
        qq(idx,2) = qq(idx-1,2);
      end
      if( qq(idx,3) < qq(idx-1,3))
```

```
        % fix ask
        qq(idx,3) = qq(idx-1,3);
      end
      if( size(qq,2) >= 11 & qq(idx,6) > 0)
        % update unrealizedpl
        if( qq(idx,5) < 0)
          qq(idx,10) = qq(idx,5) * (qq(idx,3) - qq(idx,6));
        else
          qq(idx,10) = qq(idx,5) * (qq(idx,2) - qq(idx,6));
        end
        % update pl
        qq(idx,11) = qq(idx,9) + qq(idx,10);
      end
      idx = idx + 1;
    end
    spread = qq(qq(:,2) > qq(:,3),1);
  end
  eval([char(tickerlist(i)), ' = qq;']);
end

function surfeeg(id, data256, trades, start)
% surfeeg
%   removes outliers and then plots surf spectrogram of EEG
%   data256(:,2) is EEG
%   trades are indices of trades
%   start is index of start

    all = 0;  % plot all freqs (1) or just the gamma band freqs (0)

    window = 256*8; % number of time slices/segments*?
    noverlap = 128; % number of overlapping samples between time slices
    nfft = 256*2;   % number of frequency slices*2
    fs = 256;       % sampling frequency
    % spectrogram(data256(:,2),length(data256)/2560,256,[],256,'yaxis');
    % spectrogram(data256(:,2),[],256,[],256,'yaxis');
    [S,F,T,P] = spectrogram(data256(start-256*90:end,2),window, ...
                            noverlap,nfft,fs,'yaxis');
    if( all == 1)
      Px = conv2(P, ones(11,1));  % boxcar = 11
      Px = Px(6:end-5,:);         % readjust for boxcar
    else
      Px = P;
    end
    Pxx = zeros(size(Px));      % preallocate
    % adjust spectrogram for outliers
    for n = 1:size(Px,1)
      Prow = sort(Px(n,:));
      onepercent = ceil(0.01*length(Prow));
```

```
  % replace 1% highest extremes with an upperbound
  ub = mean(Prow(end-11-1-onepercent:end-1-onepercent);
  Px(n,:) = min(ub, Px(n,:));
  % skip the 10 lowest extremes and find average of next lowest 1%
  lb = mean(Prow(11:11+onepercent));
  % subtract lowerbound from signal
  Pxx(n,:) = max(eps, Px(n,:)-lb);
end

if( all == 1)
  x = Pxx;
else
  % pull out just the gamma band = 30-50 Hz
  x = sum(Pxx(30 <= F & F <= 50, :));
  x = repmat(x, 2, 1);
  F = 1:2;
end
h = figure;
surf(T,F,10*log10(x),'EdgeColor','none');
colormap(gray);
axis xy;
axis tight;
view(0,90);
title(sprintf('Spectrogram EEG (1500%d)', id))
xlabel('Time (secs.)')
if( all == 1)
  ylabel('Frequency (Hz)');
else
  ylabel('Gamma Band Activity')
  set(gca,'YTickLabel','');
  set(gca,'OuterPosition',[0 0.7 1 0.3]);
end

hold on
% plot trades
plot(trades/256, 2*ones(size(trades)), 'k*');

% plot start
plot(start/256, 2, 'ko');

saveas(h, sprintf('eeggba1500%d', id), 'epsc');
```

Appendix E

Physiological Extremes

Most of the financial and physiological data analysis in this document first identifies time points based on financial data and then assesses what is happening physiologically. In this section, I reverse the process. I first identify interesting physiological values and then assess what is happening financially.

The figures in this section show the time points of the five most extreme values from each of the physiological features as described in Section 4.5 for 15001. It is interesting to see that some the extreme values (denoted by the 'x') align with swings in P&L. Not surprisingly, the ticker that is monitored the most—#9—has the most extreme values. I include figures for all of the tickers monitored, even though not all of the tickers align with extreme physiological events.

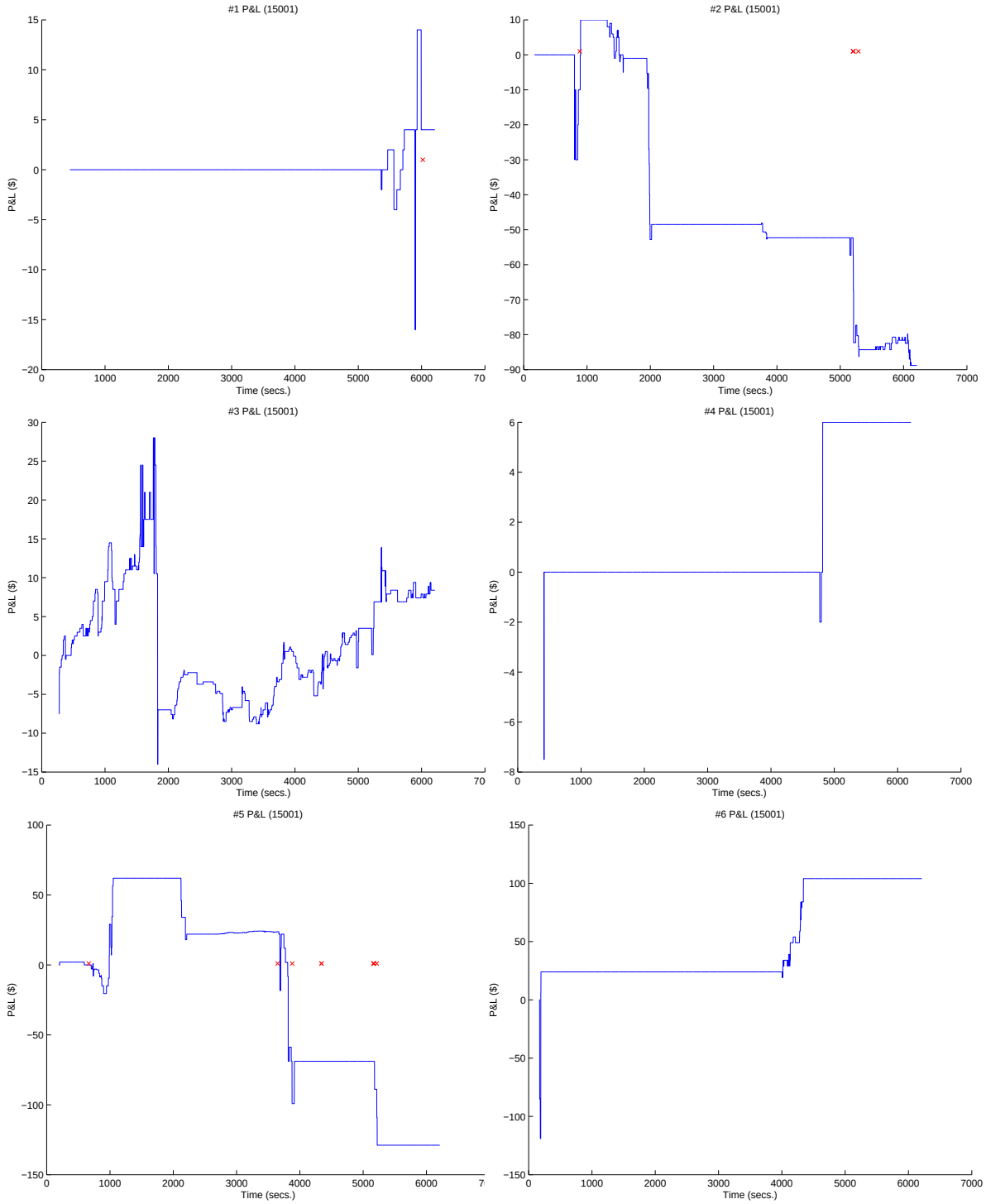

Figure E-1: Time points of extreme physiological events mapped onto P&L of a single security.

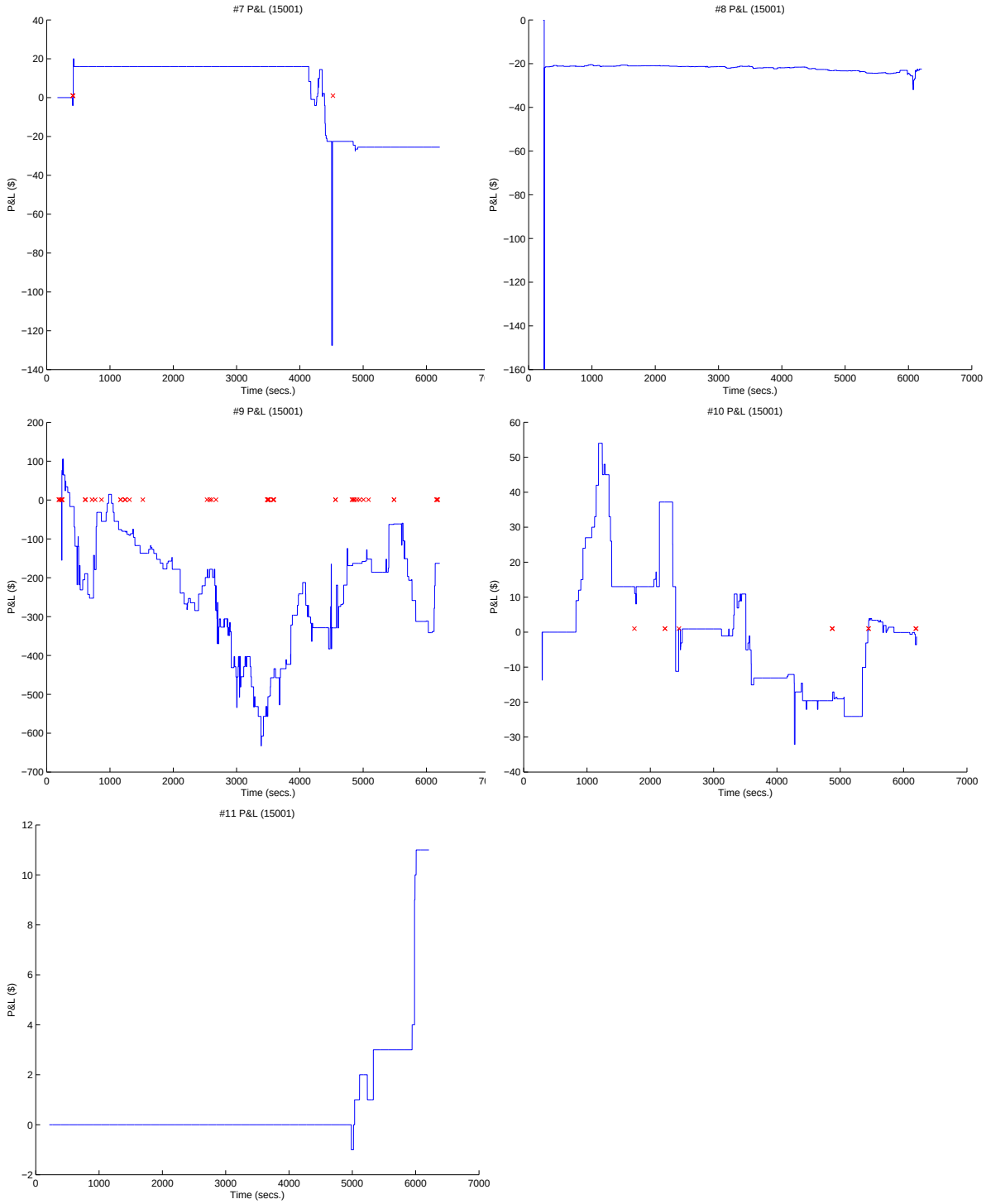

Figure E-2: Time points of extreme physiological events mapped onto P&L of a single security.

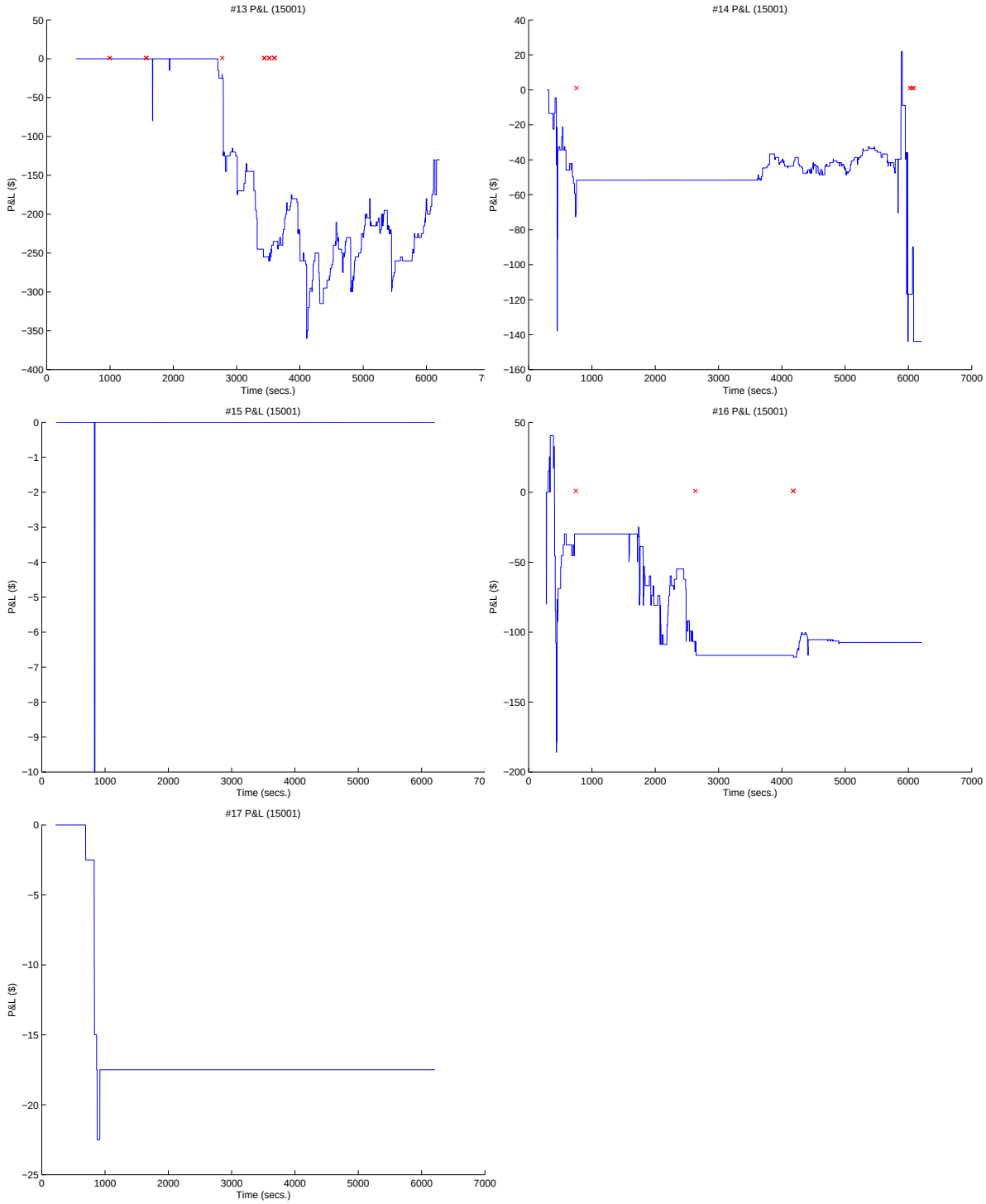

Figure E-3: Time points of extreme physiological events mapped onto P&L of a single security.

Bibliography

Lucy F. Ackert, Bryan K. Church, and Richard Deaves. Emotion and financial markets. *Economic Review*, FRB Atlanta:33–41, 2003.

Michael J. Apter. *Reversal Theory : The Dynamics of Motivation, Emotion and Personality.* Oneworld Publications, 2006.

G. Aronsson and A. Rissler. Psychophysiological stress reactions in female and male urban bus drivers. *J Occup Health Psychol*, 3(2):122–129, Apr 1998.

A. Bechara, A. R. Damasio, H. Damasio, and S. W. Anderson. Insensitivity to future consequences following damage to human prefrontal cortex. *Cognition*, 50(1-3):7–15, 1994.

A. Bechara, H. Damasio, and A. R. Damasio. Emotion, decision making and the orbitofrontal cortex. *Cereb Cortex*, 10(3):295–307, Mar 2000.

A. Bechara, H. Damasio, D. Tranel, and A. R. Damasio. Deciding advantageously before knowing the advantageous strategy. *Science*, 275(5304):1293–1295, Feb 1997.

A. Bechara, D. Tranel, H. Damasio, R. Adolphs, C. Rockland, and A. R. Damasio. Double dissociation of conditioning and declarative knowledge relative to the amygdala and hippocampus in humans. *Science*, 269(5227):1115–1118, Aug 1995.

Antoine Bechara. The role of emotion in decision-making: evidence from neurological patients with orbitofrontal damage. *Brain Cogn*, 55(1):30–40, Jun 2004.

Antoine Bechara, Hanna Damasio, and Antonio R Damasio. Role of the amygdala in decision-making. *Ann N Y Acad Sci*, 985:356–369, Apr 2003.

Marshall E. Blume and Michael A. Goldstein. Quotes, order flow, and price discovery. *The Journal of Finance*, 52(1):221–244, Mar 1997.

Martin Borchert and Antje Dusterhoft. Emotions in speech - experiments with prosody and quality features in speech for use in categorical and dimensional emotion recognition environments. In *Proceedings of 2005 IEEE International Conference on Natural Language Processing and Knowledge Engineering, 2005. IEEE NLP-KE '05.*, pages 147–151, 2005.

H. C. Breiter, I. Aharon, D. Kahneman, A. Dale, and P. Shizgal. Functional imaging of neural responses to expectancy and experience of monetary gains and losses. *Neuron*, 30 (2):619–639, May 2001.

J. D. Brown and W. J. Huffman. Psychophysiological measures of drivers under the actual driving conditions. *Journal of Safety Research*, 4:172–178, 1972.

C. T. Brownlees and G. M. Gallo. Financial econometric analysis at ultra-high frequency: Data handling concerns. *Comput. Stat. Data Anal.*, 51(4):2232–2245, 2006. ISSN 0167-9473.

Laurence Brunton, John Laz, and Keith Parker, editors. *Goodman & Gilman's The Pharmacological Basis of Therapeutics*. McGraw-Hill Professional, 2005.

J. T. Cacioppo and W. L. Gardner. Emotion. *Annu Rev Psychol*, 50:191–214, 1999.

John T. Cacioppo, Louis G. Tassinary, and Gary G. Berntson, editors. *Handbook of Psychophysiology*. Cambridge University Press, 2nd edition, 2000.

William J. Clancey. The knowledge level reinterpreted: Modeling how systems interact. *Machine Learning*, 4:285–291, 1989.

Gari D. Clifford, Francisco Azuaje, and Patrick McSharry. *Advanced Methods and Tools for ECG Data Analysis*. Artech House Publishers, 2006.

David Collett. *Modelling Binary Data*. Chapman and Hall/CRC, 2002.

R. Cowie, E. Douglas-Cowie, N. Tsapatsoulis, G. Votsis, S. Kollias, W. Fellenz, and J.G. Taylor. Emotion recognition in human-computer interaction. *Signal Processing Magazine, IEEE*, 18:32–90, Jan 2001.

Hugo D Critchley. Electrodermal responses: what happens in the brain. *Neuroscientist*, 8 (2):132–142, Apr 2002.

Antonio Damasio. *Descartes' Error : Emotion, Reason, and the Human Brain*. Harper Perennial, 1994.

Ronald de Sousa. Emotion. In Edward N. Zalta, editor, *The Stanford Encyclopedia of Philosophy*. Spring 2003.

Ap Dijksterhuis, Maarten W Bos, Loran F Nordgren, and Rick B van Baaren. On making the right choice: the deliberation-without-attention effect. *Science*, 311(5763):1005–1007, Feb 2006.

J. A. Easterbrook. The effect of emotion on cue utilization and the organization of behavior. *Psychol Rev*, 66(3):183–201, May 1959.

Fred W. Eastman. The physics of the emotions. *Harper's Magazine*, 128(300):297, January 1914.

Paul Ekman, Robert W. Levenson, and Wallace V. Friesen. Autonomic nervous system activity distinguishes among emotions. *Science*, 221:1208–1210, 1983.

Task Force of the European Society of Cardiology the North American Society of Pacing Electrophysiology. Heart rate variability: standards of measurement, physiological interpretation and clinical use. *Circulation*, 93(5):1043–1065, Mar 1996.

Daniel Ellsberg. Risk, ambiguity, and the savage axioms. *Quarterly Journal of Economics*, 75:643–669, 1961.

J. Elster. Emotions and economic theory. *Journal of Economic Literature*, 36:47–74, 1998.

Catherine Elton. Measuring emotion at the symphony. *Boston Globe*, April 5 2006.

Dylan Evans. The search hypothesis of emotion. *Br J Philos Sci*, 53(4):497–509, 2002.

Thomas Neal Falkenberry. High frequency data filtering, 2002.

Lesley K Fellows. The cognitive neuroscience of human decision making: a review and conceptual framework. *Behav Cogn Neurosci Rev*, 3(3):159–172, Sep 2004.

Raul Fernandez. *A computational model for the automatic recognition of affect in speech*. PhD thesis, Massachusetts Institute of Technology, 2004.

Paul W Glimcher and Aldo Rustichini. Neuroeconomics: the consilience of brain and decision. *Science*, 306(5695):447–452, Oct 2004.

A. L. Goldberger, L. A. N. Amaral, L. Glass, J. M. Hausdorff, P. Ch. Ivanov, R. G. Mark, J. E. Mietus, G. B. Moody, C.-K. Peng, and H. E. Stanley. PhysioBank, PhysioToolkit, and PhysioNet: Components of a new research resource for complex physiologic signals. *Circulation*, 101(23):e215–e220, June 13 2000.

R. K. Goyal and I. Hirano. The enteric nervous system. *N Engl J Med*, 334(17):1106–1115, Apr 1996.

C. Shawn Green and Daphne Bavelier. Action video game modifies visual selective attention. *Nature*, 423(6939):534–537, May 2003.

S. Grossberg and W. Gutowski. Neural dynamics of decision making under risk: Affective balance and cognitiveemotional interactions. *Psychological Review*, 94:300–318, 1987.

Alan N Hampton and John P O'doherty. Decoding the neural substrates of reward-related decision making with functional mri. *Proc Natl Acad Sci U S A*, 104(4):1377–1382, Jan 2007.

Yanic Hanoch. "neither an angel nor an ant": Emotion as an aid to bounded rationality. *Journal of Economic Psychology*, 23:1–25, 2002.

Jennifer A. Healey. *Wearable and Automotive Systems for Affect Recognition from Physiology*. PhD thesis, Massachusetts Institute of Technology, May 2000.

Keith Herring. Blind separation of noisy multivariate data using second-order statistics. Master's thesis, Massachusetts Institute of Technology, 2006.

Alice M. Isen. An influence of positive affect on decision making in complex situations: Theoretical issues with practical implications. *Journal of Consumer Psychology*, 11(2): 75–85, 2001.

William James. *William James : Writings 1878-1899*. Library of America, 1992.

J. R. Jennings and C. C. Wood. Cardiac cycle time effects on performance, phasic cardiac responses, and their intercorrelation in choice reaction time. *Psychophysiology*, 14(3): 297–307, May 1977.

Tom Johnstone, Carien M van Reekum, Kathryn Hird, Kim Kirsner, and Klaus R Scherer. Affective speech elicited with a computer game. *Emotion*, 5(4):513–518, Dec 2005.

Daniel Kahneman. Maps of bounded rationality: Psychology for behavioral economics. web, Dec 2002. Revised version of lecture delivered when receiving the Nobel Prize in Economic Sciences.

Daniel Kahneman and Amos Tversky. Prospect theory: An analysis of decision making under risk. *Econometrica*, 47:263–291, 1979.

Bernard Katz. *Nerve, Muscle, and Synapse*. Mcgraw-Hill Book Company, 1966.

I. Kawachi, D. Sparrow, P. S. Vokonas, and S. T. Weiss. Decreased heart rate variability in men with phobic anxiety (data from the normative aging study). *Am J Cardiol*, 75(14): 882–885, May 1995.

Dacher Keltner and James J. Gross. Functional accounts of emotions. *Cognition and Emotion*, 13:467–480, 1999.

Ari Kiev. *Trading to Win*. John Wiley & Sons, Inc., 1998.

K. H. Kim, S. W. Bang, and S. R. Kim. Emotion recognition system using short-term monitoring of physiological signals. *Med Biol Eng Comput*, 42(3):419–427, May 2004.

Michael Koenigs and Daniel Tranel. Irrational economic decision-making after ventromedial prefrontal damage: evidence from the ultimatum game. *J Neurosci*, 27(4):951–956, Jan 2007.

P. J. Lang. The emotion probe. Studies of motivation and attention. *American Psychologist*, 50(5):372–385, May 1995.

Thomas A Lasko, Jui G Bhagwat, Kelly H Zou, and Lucila Ohno-Machado. The use of receiver operating characteristic curves in biomedical informatics. *J Biomed Inform*, 38 (5):404–415, Oct 2005.

Joseph LeDoux. *The Emotional Brain*. Simon and Schuster, 1996.

Jennifer S. Lerner, Deborah A. Small, and George Loewenstein. Heart strings and purse strings: Carry-over effects of emotions on economic transactions. *Psychological Science*, 15:337–341, 2004.

Robert W. Levenson. Autonomic nervous system differences among emotions. *Psychological Science*, 3(1):23–27, Jan. 1992.

E. Lindholm and C. M. Cheatham. Autonomic activity and workload during learning of a simulated aircraft carrier landing task. *Aviat Space Environ Med*, 54(5):435–439, May 1983.

Andrew Lo. The three ps of total risk management. *Financial Analysts Journal*, 55:12–20, 1999.

Andrew W. Lo and Dmitry V. Repin. The psychophysiology of real-time financial risk processing. *Journal of Cognitive Neuroscience*, 14:323–339, 2002.

G. Loewenstein. Emotions in economic theory and economic behavior. *American Economic Review*, 90:426–443, 2000.

J. S. Lombard. *Experimental Researches on the Regional Temperature of the Head under Conditions of Rest, Intellectual Activity, and Emotion*. H. K. Lewis, London, 1879.

On-line User's Manual. The MathWorks, Inc., Natick, MA, 2006.

Patrick M. McCaney. Emotional response modeling in financial markets: Boston stock exchange analysis. Master's thesis, Massachusetts Institute of Technology, 2004.

B. S. McEwen. The neurobiology of stress: from serendipity to clinical relevance. *Brain Res*, 886(1-2):172–189, Dec 2000.

D. N. McIntosh, R. B. Zajonc, P.S. Vig, and S. W. Emerick. Facial movement, breathing, temperature, and affect: Implication of the vascular theory of emotional efference. *Cognition and Emotion*, 11:171–195, 1997.

Marvin Minsky. *The Emotional Machine: Commonsense Thinking, Artificial Intelligence, and the Future of the Human Mind*. Simon & Schuster, New York, 2006.

M. M. Mller, T. Gruber, and A. Keil. Modulation of induced gamma band activity in the human eeg by attention and visual information processing. *Int J Psychophysiol*, 38(3): 283–299, Dec 2000.

J. O'Doherty, M. L. Kringelbach, E. T. Rolls, J. Hornak, and C. Andrews. Abstract reward and punishment representations in the human orbitofrontal cortex. *Nat Neurosci*, 4(1): 95–102, Jan 2001.

R.E. Offerhaus. *The Study of Heart Rate Variability*, chapter Heart rate variability in psychiatry, pages 225–238. Oxford University Press, 1980.

Andrew Ortony, Gerald Clore, and Allan Collins. *The Cognitive Structure of Emotions*. Cambridge University Press, 1988.

Christian Peter and Antje Herbon. Emotion representation and physiology assignments in digital systems. *Interact. Comput.*, 18(2):139–170, 2006. ISSN 0953-5438.

E. Peters and P. Slovic. The springs of action: Affective and analytical information processing in choice. *Personality and Social Psychology Bulletin*, 26:1465–1475, 2000.

K. Luan Phan, Tor Wager, Stephan F Taylor, and Israel Liberzon. Functional neuroanatomy of emotion: a meta-analysis of emotion activation studies in pet and fmri. *Neuroimage*, 16(2):331–348, Jun 2002.

Rosalind W. Picard. *Affective computing*. MIT Press, 1997.

Esteban Pino, Lucila Ohno-Machado, Eduardo Wiechmann, and Dorothy Curtis. Real-time ecg algorithms for ambulatory patient monitoring. *AMIA*, 2005.

Jocelyn Pixley. Finance organizations, decisions and emotions. *Br J Sociol*, 53(1):41–65, Mar 2002.

Michael Polanyi. *The Tacit Dimension*. Doubleday, 1966.

Arthur S. Reber. *Implicit Learning and Tacit Knowledge*. Clarendon Press, 1993.

D. A. Redelmeier, P. Rozin, and D. Kahneman. Understanding patients' decisions. Cognitive and emotional perspectives. *JAMA*, 270(1):72–76, Jul 1993.

J. M. Richards and J. J. Gross. Emotion regulation and memory: the cognitive costs of keeping one's cool. *J Pers Soc Psychol*, 79(3):410–424, Sep 2000.

S. E. Rimm-Kaufman and J. Kagan. The psychological significance of changes in skin temperature. *Motivation and Emotion*, 20:63–78, 1996.

Paul Samuelson. Proof that properly anticipated prices fluctuate randomly. *Industrial Management Review*, 6:41–49, 1965.

Alan G Sanfey, James K Rilling, Jessica A Aronson, Leigh E Nystrom, and Jonathan D Cohen. The neural basis of economic decision-making in the ultimatum game. *Science*, 300(5626):1755–1758, Jun 2003.

T. Schacker, A. C. Collier, J. Hughes, T. Shea, and L. Corey. Clinical and epidemiologic features of primary HIV infection. *Ann Intern Med*, 125(4):257–264, Aug 1996.

Harold Schlosberg. Three dimensions of emotion. *The Psychological Review*, 61(2):81–88, 1953.

Jack D. Schwager. *The New Market Wizards: Conversations with America's Top Traders*. HarperBusiness, 1992.

Robert A. Schwartz and Reto Francioni. *Equity Markets in Action*. John Wiley and Sons, Inc., 2004.

E. W. Scripture. Detection of the emotions by the galvanometer. *Journal of the American Medical Association*, 50:1164, 1908.

Baba Shiv, George Loewenstein, Antoine Bechara, Hanna Damasio, and Antonio R Damasio. Investment behavior and the negative side of emotion. *Psychol Sci*, 16(6):435–439, Jun 2005.

V. Shusterman and O. Barnea. Spectral characteristics of skin temperature indicate peripheral stress-response. *Biofeedback Self Regul*, 20(4):357–367, Dec 1995.

Brett N. Steenbarger. *The Psychology of Trading*. Wiley, 1st edition, Dec. 2002.

Jonathan Sykes and Simon Brown. Affective gaming: measuring emotion through the gamepad. In *CHI '03: CHI '03 extended abstracts on Human factors in computing systems*, pages 732–733, New York, NY, USA, 2003. ACM Press. ISBN 1-58113-637-4.

ProComp Software User's Manual. Thought Technology, Montreal, Quebec, Canada.

Sabrina M Tom, Craig R Fox, Christopher Trepel, and Russell A Poldrack. The neural basis of loss aversion in decision-making under risk. *Science*, 315(5811):515–518, Jan 2007.

Mascha van 't Wout, Ren S Kahn, Alan G Sanfey, and Andr Aleman. Affective state and decision-making in the ultimatum game. *Exp Brain Res*, 169(4):564–568, Mar 2006.

J. A. Veltman and A. W. Gaillard. Physiological indices of workload in a simulated flight task. *Biol Psychol*, 42(3):323–342, Feb 1996.

V. K. Yeragani, R. Balon, R. Pohl, C. Ramesh, D. Glitz, P. Weinberg, and B. Merlos. Decreased r-r variance in panic disorder patients. *Acta Psychiatr Scand*, 81(6):554–559, Jun 1990.

V. K. Yeragani, R. Pohl, R. Berger, R. Balon, C. Ramesh, D. Glitz, K. Srinivasan, and P. Weinberg. Decreased heart rate variability in panic disorder patients: a study of power-spectral analysis of heart rate. *Psychiatry Res*, 46(1):89–103, Jan 1993.

J. A. Yesavage, M. S. Mumenthaler, J. L. Taylor, L. Friedman, R. O'Hara, J. Sheikh, J. Tinklenberg, and P. J. Whitehouse. Donepezil and flight simulator performance: effects on retention of complex skills. *Neurology*, 59(1):123–125, Jul 2002.

H. Ylnen, H. Lyytinen, T. Leino, J. Leppluoto, and P. Kuronen. Heart rate responses to real and simulated BA Hawk MK 51 flight. *Aviat Space Environ Med*, 68(7):601–605, Jul 1997.