Beat-To-Beat Cardiovascular Indices of Guilty Knowledge

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Abstract

This exploratory investigation described some beat-to-beat cardiovascular dynamics associated with concealed knowledge. The subjects were ten non-obese males. The paradigm was the Guilty Knowledge Test. Breathing pattern, skin conductance level (SCL), electrocardiogram, impedance cardiogram and fingertip pulse pressure were recorded for 30 sec. The SCL-based detection rates for lies, 62.5% and 67.5%, were well above the expected random guessing rate of 20%. Comparisons were made across twenty beats each for a pre-Lie true response, a Lie, and a post-Lie true response. The clearest differentiating signs of lying were (1) an immediate drop in heart rate in beats 1 through 8 associated with the Lie; (2) a decrease in myocardial contractility in beats 1 through 5 associated with pre-Lie; (3) an elevated cardiac output on beat 1 of post-Lie; and (4) the inverse rank orders of stroke volume and cardiac output versus total peripheral resistance and mean arterial pressure at beat 1 across the three conditions.

Keywords: cardiovascular, impedance cardiography, polygraph, lie detector, Guilty Knowledge Test, skin conductance

Introduction

The only practical, reliable, non-invasive tool available to the psycho-physiologist to monitor the physical function of the heart is thoracic electrical impedance plethysmography, known also as impedance cardiography (ZCG; (Miller & Horvath, 1978; Sherwood et al., 1990). The cardiac-somatic coupling hypothesis (Obrist, Webb, Sutterer, & Howard, 1970; Obrist, 1982) was instrumental in causing the psychophysiology research community to employ multivariate analysis techniques more widely in assessments of cardiovascular function. Obrist argued that it was virtually impossible to comprehend the meaning of heart rate change with respect to autonomic function unless one considered heart rate in the context of whole-body and individual-organ metabolic or related (such as heat loss) demands. Impedance cardiography enhances one's ability to acquire non-invasive, multivariate estimates of many whole-body cardiovascular parameters.

This exploratory investigation, conducted in the early 1990s, attempted to determine through a multivariate analysis of cardiovascular function what pattern of autonomic activity was associated reliably with the concealment of knowledge (Miller, 1994). The working assumption was that reliable, meaningful changes in the autonomic control of cardiovascular dynamics would be associated with the concealment of knowledge that occurred during the Guilty Knowledge Test paradigm.

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Declaration of Conflicting Interests. The author declares no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Methods

Experimental Design

The experiment was descriptive and correlational in nature, using the type of response (lie or truth) as the categorical criterion variable and using continuous physiological measures as the dependent measures. The single-factor design had three levels (pre-Lie, Lie, and post-Lie) with repeated-measures across and within these levels.

Subjects

The subjects were ten non-obese males ranging in age from 19 to 25 years (mean 21.8 years). Half were Caucasian and half were African American. I explained the investigation to each subject, and each subject provided informed consent using a form approved by the Human Use Committee of the DOD Polygraph Institute, Ft. McClellan, Alabama, where the data were collected. Each subject was paid a minimum of $30.00 for participating. Each was at least one hour post-prandial at the time of testing and none had consumed a high-fat meal within two hours of testing.

Procedure

Four trials of six questions each were used in the Guilty Knowledge Test paradigm (Furedy & Ben-Shakhar, 1991). The first trial used playing cards. The card selected came from the twenty 2- through 6-valued cards in one poker deck, allowing the selection of one of the five possible numbers, 2 through 6. The investigator was blind to the number selected. The second through fourth trials involved the subject's year, month and day of birth, respectively. After the card test, the subject wrote his birth year, month and day on a card, keeping the investigator blind to the information. The question topics and answers were heterogeneous across trials, reducing the likelihood that the subject would orient to a question-answer pair in one trial based upon the structure of a previous trial.

The year, month and date possibilities were narrowed to five choices each before questioning with the following procedure. Five-year periods (for example, 1970-74) were listed sequentially with 1-year lags for the period that included all possible birth years for the age range allowed for the subjects (18 to 35). Each period was printed on a new line on a single sheet of paper. Thus, the subject's year of birth appeared on five lines of the page. The subject selected any one of the five 5-year periods that included his birth year. Similar pages and selections were used for the birth month and day. The three sets of five possibilities each were given to the investigator. Thus, the investigator had five possible values each for year, month and day, without knowledge of the specific values.

Trials were presented in the order: card, year, month, and day. There were no practice trials. However, the first question in each 6-question set allowed the subject to become familiar with the question structure. The content of the question was outside of the set of five possibilities that included the true value. For example, if the subject indicated a birth year in the range 1970-1974, then the first question was "Is your year 1969?" The physiological responses to the first question in each trial were not used in the data set.

All questions were presented orally using the phrase, "Is your xxx nnn?" Where xxx was “number,” “year,” “month,” or “day” and nnn was a number. The subject immediately answered "No" to each question asked. One of the subject's six responses within a trial was always a lie: the "No" answer to the first of the six questions was always truthful, but one of the "No" answers to the subsequent five questions was a lie. The investigator attempted to identify the concealed number through a real-time review of the skin conductance response. The subject received feedback about the success or failure of his deception after the card trial and after the day trial. This feedback was based solely upon the subject's skin conductance response.

The intent of the protocol was to make the subject conceal information from the prying investigator. In addition to two instances of immediate feedback, two other motivational methods were used. First, the subjects sat face-to-face with the investigator. Generally, this confrontational seating arrangement is not used in field polygraph examinations. Second, the subject was offered a $5.00 bonus for each birthdate

question (year, month and day) for which the investigator failed to identify the lie. This carried the potential of being a 50% bonus for participating as a subject.

Primer on Cardiovascular Dynamics

For the reader who is not a physiologist, here is a brief explanation of the dynamics that I was able to examine non-invasively. The circulatory system delivers nutrients to body tissues and removes byproducts of cellular metabolism from body tissues. These functions are dependent upon the presence of arterial blood pressure, which oscillates as the pump, the heart, contracts (systole) and relaxes (diastole). I measured blood pressure with a small cuff placed on the fingertip and calculated a mean arterial pressure (MAP) from the pulsatile waveform, expressed in millimeters of the height of a column of Mercury (mmHg).

In turn, MAP is dependent upon the flow leaving the pump, called cardiac output (Q_dot), and the resistance to flow in the arterial system. The latter, called total peripheral resistance (TPR), is dependent upon wall friction in the arteries, the flexibility of the arterial walls, how many red blood cells are present in the fluid (hematocrit), the presence of laminar or turbulent flow, and the local regulation of pre-capillary sphincters. Reciprocal changes in Q_dot and TPR, controlled by local mechanisms and by the sympathetic and parasympathetic nervous systems (SNS, PNS), interact to maintain blood pressure within a relatively narrow range. This range assures blood flow to the brain within the fixed volume of the skull. This pressure control function is challenged especially by postural changes from lying to standing, by muscle blood flow demands in exercise, and by skin blood flow demands during heat stress. Biological pressure sensors located at strategic points within the arterial system, especially in the carotid arteries, provide the neural feedback needed for regulation.

Cardiac output is determined by the rate at which the pump contracts and the volume of blood ejected with each contraction. The rate was measured with electrocardiographic (ECG) electrodes placed upon the chest. For the beat-to-beat analysis, I examined the interbeat interval (IBI) in milliseconds per beat. In this article, I present the IBI as its reciprocal, heart rate (HR), expressed in beats per minute. The HR is under the control of both the SNS and the PNS, acting at the heart's pacemaker (the sino-atrial, or SA, node). Changes in HR are called cardiac chronotropy. I measured the volume of blood ejected at each beat with a device called an impedance cardiograph (ZCG). This device was perfected for NASA space flights by Dr. William Kubicek at the University of Minnesota (Kubicek, Patterson, & Witsoe, 1970; Kubicek, Witsoe, Patterson, & From, 1969). I pioneered the use of ZCG in psychophysiological research in an article extracted from my doctoral dissertation and published in the journal, Psychophysiology (Miller & Horvath, 1978). The device injected a 4 milliamp, 100 kHz signal through the thorax; this is a medically safe current that is not felt by the subject. The pulsatile change in impedance (Z) to the 4 ma current was proportional to the volume of blood ejected per beat, or the stroke volume (SV). Cardiac output (Q_dot) is calculated as the product of SV in milliliters per beat and HR in beats per minute, and is expressed as Liters per minute.

The combination of the ECG and ZCG waveforms allows an estimate of another variable, myocardial contractility, a primary contributor to SV. Contractility is a property of myocardial muscle fibers, determined in part by how much they are stretched; this is somewhat, but not exactly, like the tension that increases as you lengthen a spring. During the relaxed phase of a heart beat (diastole), blood is pushed into the left ventricle from the lungs. This push stretches the myocardial fibers of the left ventricle and they respond (systole) with proportional contractile force once they are stimulated by the heart's pacemaker. Like a spring, if the fibers are stretched too far, they begin to fail. This is called decompensation and occurs in congestive heart failure. Myocardial contractility is also affected by the SNS and PNS. Ideally, contractility is measured by placing a tension meter on a few fibers of myocardial muscle. Secondarily, it is measured as the increase in pressure that occurs between the time that the left ventricle is stimulated and the time that the aortic valve is forced open, allowing blood to flow

from the left ventricle into the aorta and the rest of the peripheral vasculature. The stimulation of the left ventricle is marked in the ECG waveform by the R wave. The opening of the aortic valve is marked in the first derivative of the ZCG waveform (dZ/dt) by a notch called the B wave. Thus, contractility may be estimated as the time, in milliseconds, between the ECG R wave and the ZCG B wave; I plotted the inverse of milliseconds here so that greater contractility is positive. Changes in myocardial contractility are called cardiac inotropy.

Instrumentation

A custom program running on one computer provided the investigator with the timing of questions and the random order of the numbers in each trial. The investigator entered the selected year, month, and day periods into the program just before the experimental procedure and the program created and cued the four randomized, 6-question trials. A second computer collected digitized physiological data and a manual event mark for each question within a trial. The event mark coincided with the last word spoken in each question, preceding immediately the subject's 1-word response of "No." A trial was 3.6 minutes long. The inter-stimulus interval was 36 seconds, of which 30 seconds were dedicated to physiological data collection and the other six to system adjustment, if required. Subjects were run at 0900, 1100 or 1400 hours.

Fingertip pulse pressure was recorded using the Ohmeda Model 2300 Finapres BP Monitor (Ohmeda Division of the BOC Group Inc., Englewood, Colorado), connected to a sensor placed on the middle phalanx of the second finger of the non-dominant hand. A 0-to-150 mmHg calibration was recorded at the beginning of each trial. Values recorded at the fingertip are usually aligned well with values acquired by standard auscultation of the brachial artery at the level of the heart.

The ECG was recorded using a SensorMedics R612 Dynograph and Type 9878 voltage coupler, with the "ECG" high pass filter, a 30 Hz low pass filter, and a 60 Hz notch filter (SensorMedics Corp., Anaheim, California). The ECG electrodes were attached approximately in the CR-5 locations (Simonson, 1971). This electrode configuration produced a lead-II-appearing waveform, i.e., with a large R wave.

Five channels of digitized physiological data and one manual reading were acquired following each stimulus. Each channel was digitized at 200 samples/sec. The impedance cardiogram (ZCG) was recorded as two channels of data, impedance change (dZ) and its first derivative (dZ/dt), using the IFM/Minnesota Model 304B Impedance Cardiograph (Instrumentation for Medicine, Inc., Greenwich, Connecticut). The baseline impedance of the thorax (Z0) was recorded manually. To acquire these signals, circumferential, aluminized Mylar tape electrodes (IFM T-8001, Instrumentation for Medicine, Inc., Greenwich, Connecticut) were placed around the neck and abdomen (Miller & Horvath, 1978). The inter-electrode distance (L) was estimated as the mean of the measured front and back inter-electrode distances. Calibration signals for dZ (0.1 ohm) and dZ/dt (1.0 ohm/sec) were recorded for each trial.

The subject's breathing pattern was recorded using the Lafayette Factfinder polygraph and Model 76477-G signal conditioner (Lafayette Instrument Co., Lafayette, Indiana), connected to a sensor that encircled the chest and responded to changes in chest circumference. Skin conductance level (SCL) was recorded using the same polygraph and Model 76483-G GSG signal conditioner, connected to stainless steel electrodes attached to the middle phalanges of the first and third fingers of the non-dominant hand. A novice and an expert scored the SCR data *post hoc*, without knowledge of the placement of the lie in the series of questions.

Data Reduction

The digitized data were collected using the AT-CODAS software (Release 5, DataQ_Instruments, Inc., Akron, Ohio) and 16-bit analog-to-digital conversion adapter in an 80386 IBM-clone personal computer. Each trial was recorded as a single CODAS binary data file. The data from the 36 seconds subsequent to the second through fifth number presentations within trials were extracted, using CODAS, as 6-channel text files for further spreadsheet processing.

The first cardiac cycle following the event mark was labeled cycle 0. The subsequent twenty cardiac cycles were characterized in the following manner:

Interbeat interval (IBI), presented here as its inverse, heart rate (HR)
Stroke volume (SV)
Single-beat cardiac output (Q_dot)
A correlate of myocardial contractility, the R-B interval (RB), presented here as its inverse (millisec^{-1})
Mean arterial pressure (MAP)
Total peripheral resistance (TPR)

The occurrence of the ECG R wave was identified by its greatest positive slope. This approach reduced the uncertainty associated with sampling-rate error in locating the peak of the R wave.

The dZ/dt B wave (aortic valve opening) was identified as the last sample preceding the greatest positive slope in the cardiac dZ/dt cycle. The dZ/dt Z wave was identified as the greatest value in the cardiac dZ/dt cycle, and the dZ/dt X wave as the lowest value. Stroke volume and Q_dot were calculated as in Miller and Horvath (1978). Contractility was estimated as the inverse of the R-B interval, as discussed by Siegel et al. (Kubicek et al., 1969). The MAP was calculated as the point two-thirds through the range from diastolic to systolic pressure. Total peripheral resistance was calculated as the quotient of Q_dot and MAP.

Calibration data were extracted from the CODAS ASCII files by a custom program and saved.

The calibration data were combined with the R wave location data by the custom software. This program found the dZ/dt B, Z, and X inflection points and the FinaPres low and high pressures, converted the raw data to physiological values, and stored IBI, contractility, SV, Q_dot, MAP, and TPR data. Another custom program organized the ZCG data by variable for spreadsheet input.

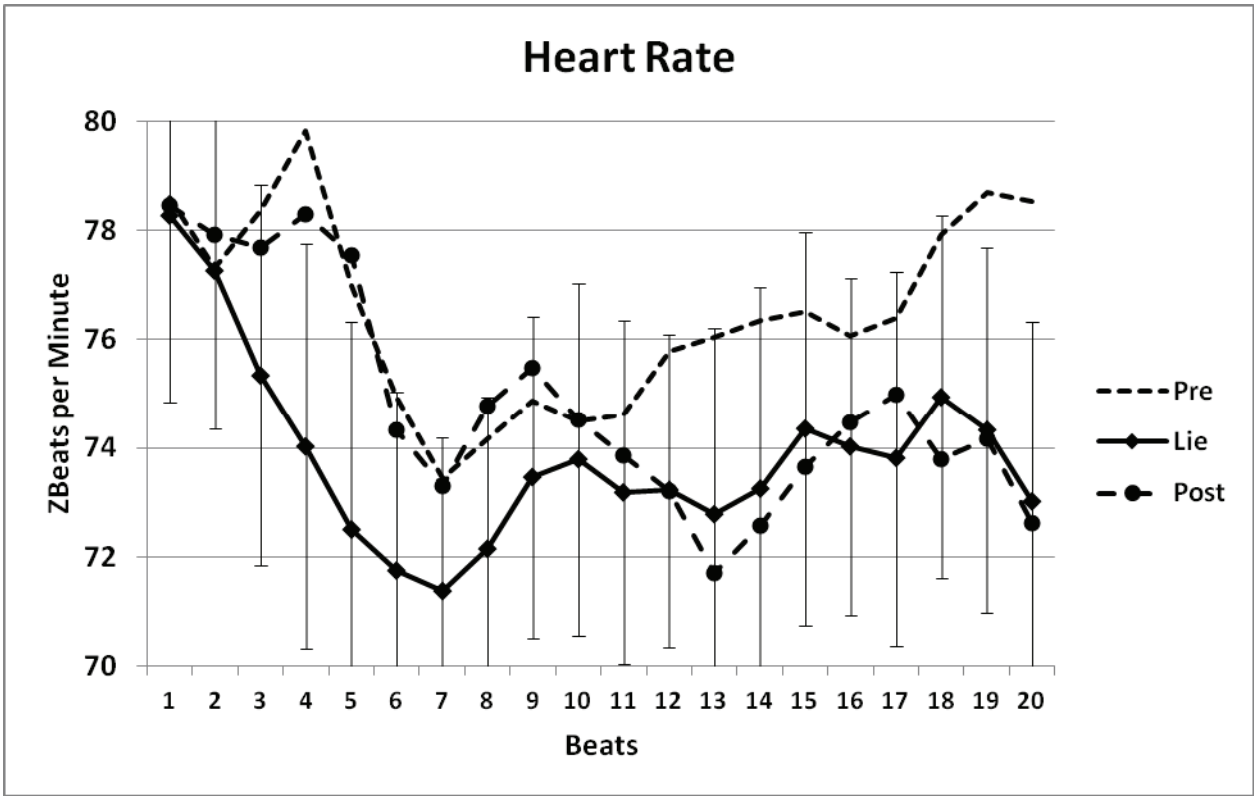
On about 60% of the trials, the lie occurred in response to question 3, 4 or 5. Within these trials, at least one pre-Lie true response and at least one post-Lie true response were available for analysis. The occurrence of trials with the Lie in answers 3 through 5 was distributed across subjects such that two subjects experienced one occurrence, four experienced two occurrences, and four experienced three occurrences. One of these trials per subject was selected for cardiovascular analysis: the trial with the most correctly interpreted SCR pattern. This selection biased the exploratory study slightly in favor of positive findings. Cardiovascular measures were extracted from each of three responses within each selected trial (pre-Lie truth, Lie, post-Lie truth). The pre- and post-Lie responses used were those immediately preceding and following the Lie.

Results

The novice identified 62.5% of the 40 lies correctly and the experienced scorer identified 67.5% of the lies correctly. For seven of the subjects, trials in which both scorers identified the lie correctly were available for cardiovascular analyses. For two subjects, the only trials available were trials in which only one scorer identified the lie correctly. For one subject, the only trial available was one in which neither scorer identified the lie correctly. Of the ten trials selected for analysis (one per subject), two were card trials, three were year trials, three were month trials, and two were day trials.

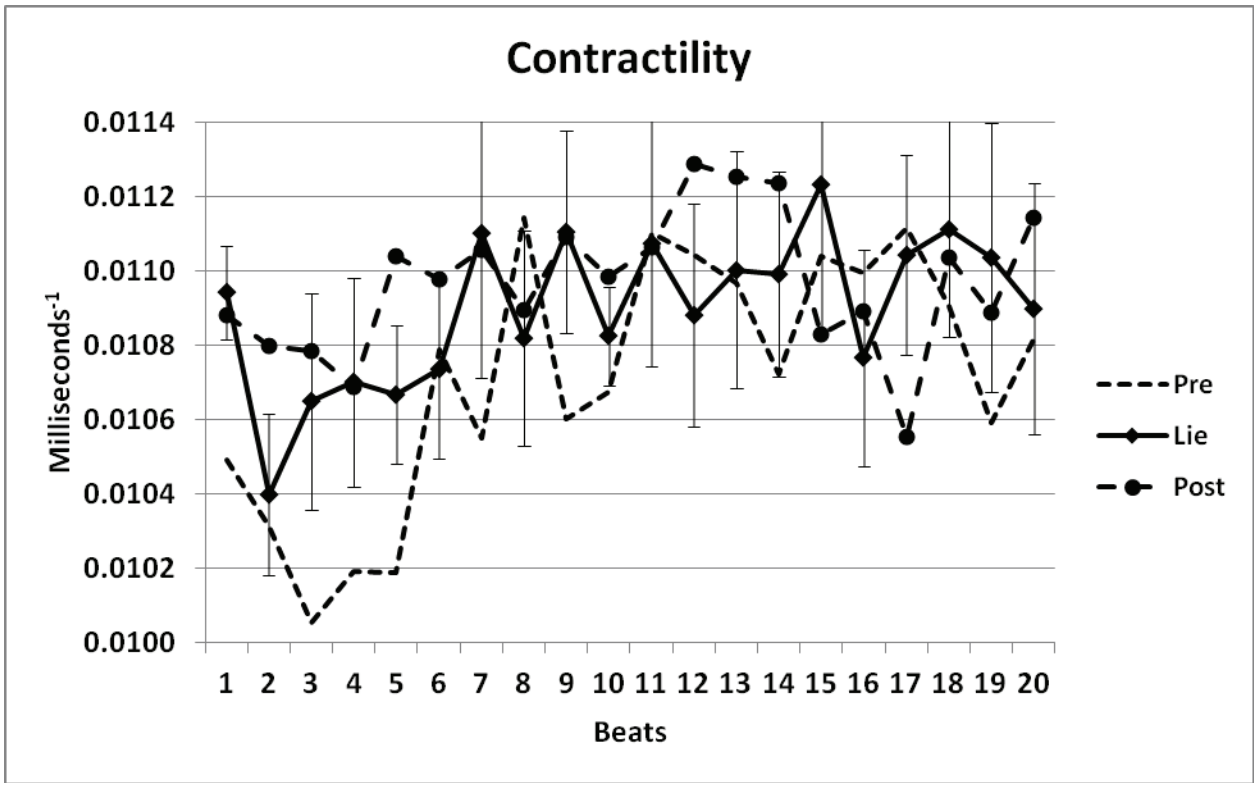
Heart Rate. The beat-to-beat patterns of HR are shown in Figure 1. During the first seven beats after the Lie, HR decreased about 6 beats/minute. There was a different pattern for pre- and post-Lie: a several-beat delay preceding the decline in heart rate. This difference was most pronounced at beats 4 and 5. Following both Lie and post-Lie, HR remained relatively stable across beats 10 through 20, in contrast to the slight increase in HR during this period following pre-Lie.

Figure 1. Mean heart rate (HR; beats per minute) and the standard error of the mean for the Lie response, n = 10, following the Lie and the true response immediately preceding and following the Lie (pre, post). HR was calculated as the inverse of beat-to-beat interbeat interval.



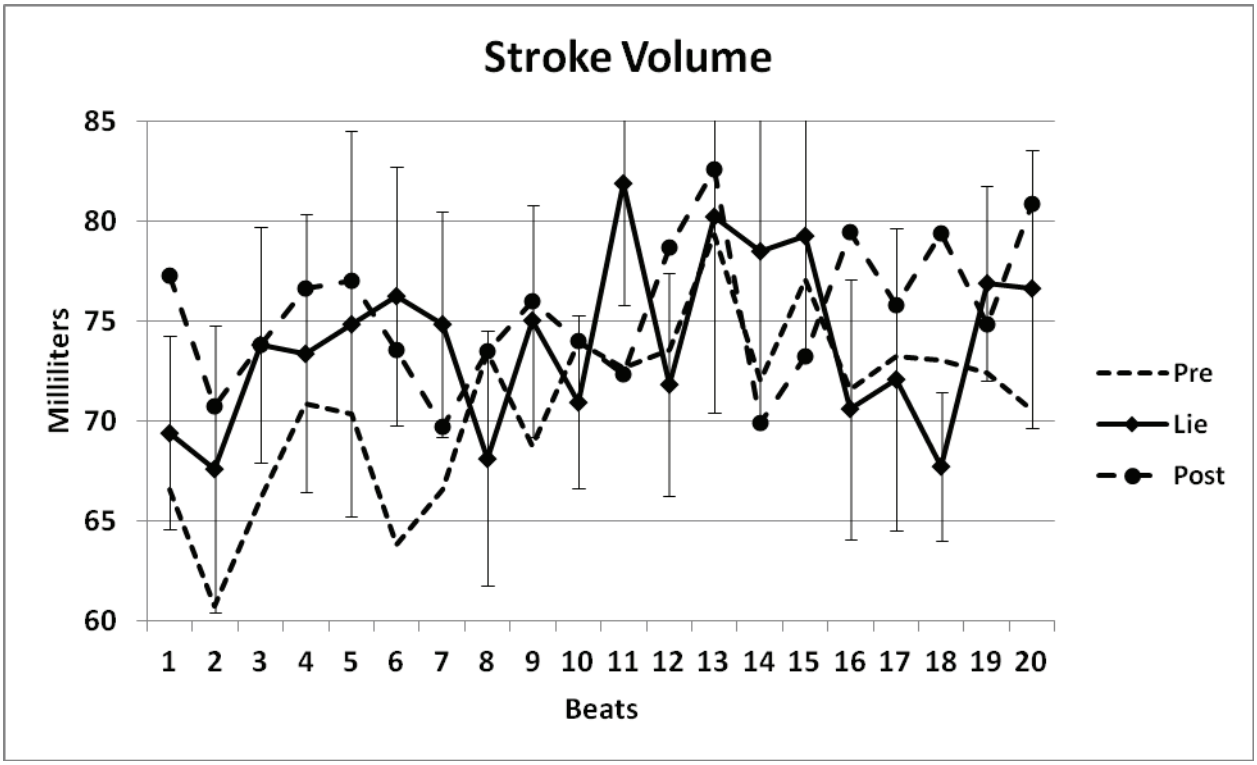
Contractility. The beat-to-beat patterns of contractility are shown in Figure 2. Note the sharply lower contractility at beat 1 for pre-Lie: contractility appears to been elevated in the first beat after the Lie and then to have remained elevated through post-Lie. Contractility remained sharply lower for pre-Lie during beats 3 through 5.

Figure 2. Mean contractility in milliseconds-1 and the standard error of the mean for the Lie response, n = 10, following the Lie and the true response immediately preceding and following the Lie (pre, post). Contractility was calculated as the inverse of beat-to-beat R-B interval.



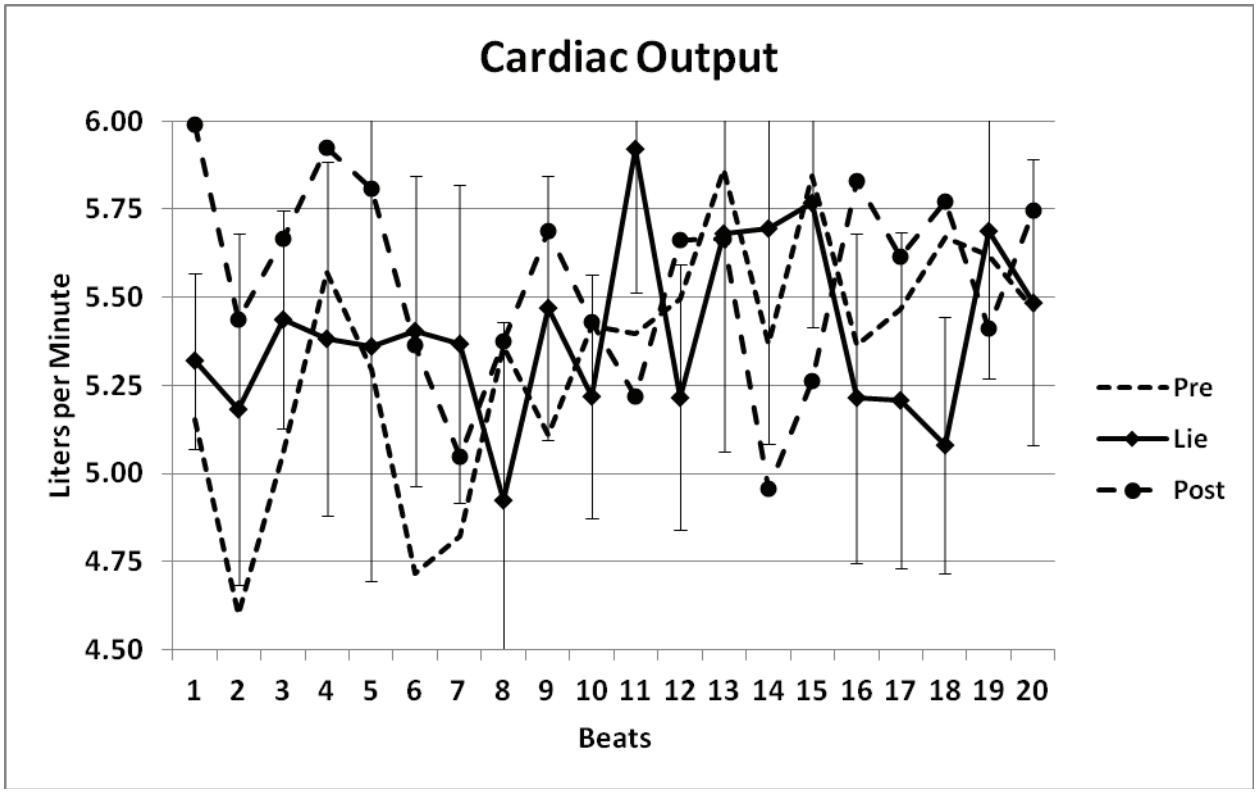
Stroke Volume (SV). The beat-to-beat patterns of SV are shown in Figure 3. Echoing contractility, SV was relatively low during the first several beats following pre-Lie, especially at beats 3 and 6. The two-beat elevation after beat 2 may be compensatory for the relatively low SV at beat 2.

Figure 3. Mean stroke volume (SV; milliliters per beat) and the standard error of the mean for the Lie response, n = 10, following the Lie and the true response immediately preceding and following the Lie (pre, post).



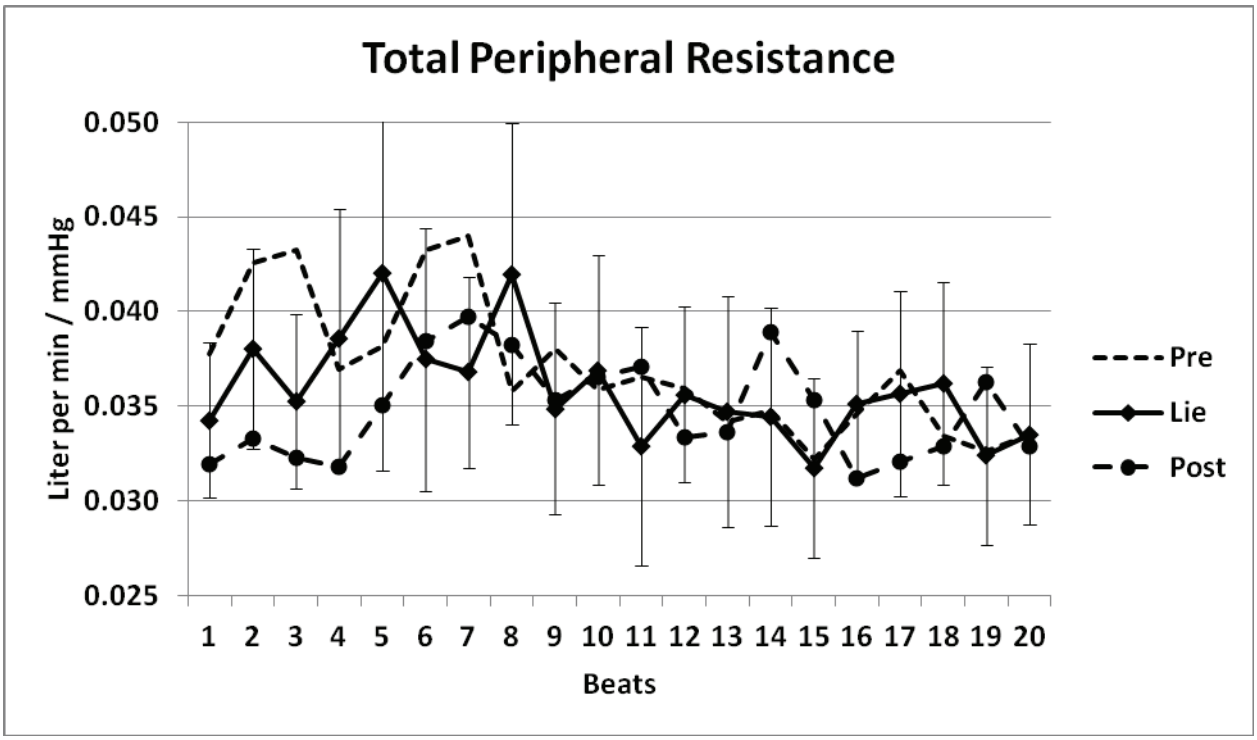
Cardiac Output (Q_dot). The beat-to-beat patterns of Q_dot are shown in Figure 4. Note the sharply elevated value at beat 1 for post-Lie. This echoes the SV pattern. The biphasic pattern across beats 1 through 9 for both true responses are due a similar, but more muted pattern in SV (Figure 3) and to the HR peak at beat 4 (Figure 1).

Figure 4. Mean cardiac output (Q_dot; Liters per minute) and the standard error of the mean for the Lie response, n = 10, following the Lie and the true response immediately preceding and following the Lie (pre, post).



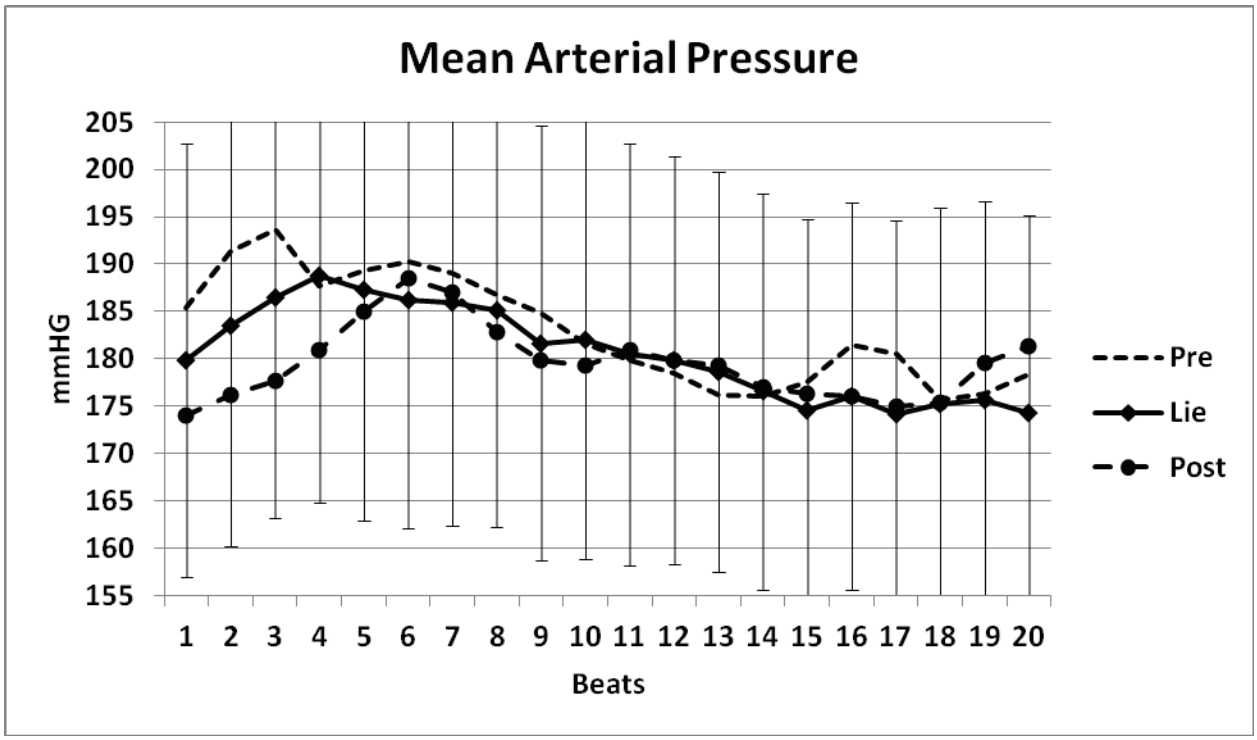
Total Peripheral Resistance (TPR). The beat-to-beat patterns of TPR are shown in Figure 5. This measure is back calculated from Q_dot and MAP. There was an early, small biphasic response for all three responses, but most noticeably at beats 3 and 7 for pre-Lie. Note that peaking was relatively delayed in the other two responses. The early pattern of TPR was the reciprocal of the early patterns for SV and Q.

Figure 5. Mean total peripheral resistance (TPR; Liters per minute of Q_dot divided by mmHG of MAP) and the standard error of the mean for the Lie response, n = 10, following the Lie and the true response immediately preceding and following the Lie (pre, post).



Mean Arterial Pressure (MAP). The beat-to-beat patterns of fingertip MAP are shown in Figure 6. Presumably, the high values recorded for MAP were caused by the occlusion of venous return by the upper arm cuff used with the Lafayette polygraph (Podlesny & Kircher, 1999). However, the relative beat-to-beat changes in MAP are useful data. Note the relative smoothness of the responses across the first 15 beats. Also note the orderly progression from pre-Lie, through Lie, to post-Lie: the initial value for MAP decreased about 5 mmHg for each response, and the peak subsequent peak was delayed one and then two beats, from beat 3 for pre-Lie, to beat 4 for Lie, and then to beat 6 for post-Lie.

Figure 6. Mean fingertip arterial pressure (MAP; mmHG) and the standard error of the mean for the Lie response, n = 10, following the Lie and the true response immediately preceding and following the Lie (pre, post).



Discussion

The overall SCR-based detection rates for lies, 62.5% and 67.5%, were not wonderful, but they were several times the expected random guessing rate of 20%. These detection rates suggested that different autonomic responses were associated with lies than with true responses in this experiment. In turn, this observation suggested that differences in cardiovascular dynamics between lies and true responses were likely to be discernible.

Cardiovascular Dynamics Associated with Lying

The error bars in the figures reveal the large degree of inter-individual variability

present in the cardiovascular data. This variability may be due to a number of factors, including gender and hostility (Davis, Matthews, & McGrath, 2000). The group means indicate, presumably, underlying patterns that may be present in most people. Thus, one may be able to pick out relative trends in an individual subject that are somewhat similar to the group patterns seen here. The clearest signs of lying in this database were (1) the immediate drop in HR in beats 1 through 8 associated with the Lie; (2) The decrease in contractility in beats 1 through 5 associated with pre-Lie; (3) the elevated Q-dot on beat 1 of post-Lie; and (4) the inverse rank orders of SV and Q_dot versus TPR and MAP at beat 1 across the three conditions.

As the subject formulates an answer in the GKT, I would not expect to see much difference in HR prior to subsequent truthful or untruthful responses. With a lie, the HR may drop immediately, while the drop may be delayed several beats with a truthful response. I would also expect a lower inotropic indication (contractility) during the second and third beats of a pre-lie response, compared to a lie.

I would expect cardiac output and total peripheral resistance to be bracketed by pre- and post-lie values at beat 1 when the response is given: cardiac output during a lie would be higher than during a pre-lie truthful response, but lower than during a post-lie truthful response. Conversely, total peripheral resistance during a lie would be lower than during a pre-lie truthful response, but higher than during a post-lie truthful response.

Finally, I would expect bracketing also for mean arterial pressure. It would be higher and rise more briefly during a pre-lie truthful response, and would be lower and rise across a longer period during a post-lie truthful response. The level and pattern for a lie would fall between these two observations.

General Cardiovascular Patterns

The first beat assessed here occurred at the end of the presentation of a question to the subject. Thus, the first through the 20th beats must be considered evoked responses. The initial values may have tonic components. The mean, resting HR at the first beat scored here was about 78 beats per minute. This is at the high end of the expected resting HR range, suggesting some tonic tension reflected chronotropically. The mean SV at beat 1 ranged from about 65 to 78 ml per beat, bracketing a typical value of 70 ml per beat. The mean Q_dot at beat 1 ranged from about 5.2 to 6.0 liters per minute, slightly above an expected value of about 5.25 liters per minute. The latter elevation was driven by the slightly elevated HR and not by SV. I have no normative data for MAP as recorded in this experiment.

The SV and Q_dot patterns showed a drop at beat 2 and then an immediate reversal. The drop at beat 2 was insignificant in the overall variability of the measure except for its repeatability across the three conditions. It appeared that this initial drop in SV and Q_dot was due to a rise in TPR and resulting myocardial afterload, and to a slight decrease in myocardial contractility. Myocardial afterload was probably exacerbated for this one beat by the positive intra-thoracic pressure associated with speaking the answer to the question posed by the investigator.

The initial values for HR were essentially the same across pre-Lie, Lie and post-Lie. However, the initial values of Q_dot and SV were rank-ordered similarly across those three conditions from highest to lowest, respectively. The rank order of the initial values of MAP and TPR was clearly the reciprocal of the order for Q_dot and SV. Thus, at the moment when each question ended, it appeared that MAP was more under the control of TPR than of Q_dot.

The MAP increased steadily after the question to a point of reversal. The increase from beat 1 to beat 2 was driven by the one-beat increase in TPR, mentioned above. The increase over the next two beats was driven by a combination of increasing TPR and/or HR and/or Q_dot. The reversal occurred later for post-Lie than for Lie, and later for Lie than for pre-Lie, at beats 3, 4 and 6, respectively. The beat 3 reversal of MAP in pre-Lie reflected a drop in TPR, while the continued increases in MAP for Lie and post-Lie were driven mainly

by increasing TPR. The beat 4 reversal for the Lie reflected sharply declining HR. The beat 6 reversal for post-Lie was driven by the beginning of a slow decline in TPR.

Lie vs. Post-Lie Patterns

The early slopes of several of the measures taken during the Lie were of interest when compared to post-Lie. Lie HR declined immediately, and to a lower value, instead of starting its decline at beat 4. However, Lie contractility reversed upward earlier, at beat 2 instead of beat 4, reaching about the same values at beat 7; and Lie SV presented its 1-beat drop, then rose through beat 6 instead of through beat 4, again to about the same levels. Thus, there was a more immediate, six beats per minute, cardiac chronotropic indication (HR) of relaxation with the Lie, but a two-beat longer, reciprocal excitation of cardiac inotropic responses (contractility) with the Lie, with the contractility increase beginning sooner with the Lie than for post-Lie.

Meanwhile, TPR reversed downward in the Lie condition at beat 4, rather than at beat 6, and peaked at a higher level of resistance. With the Lie, MAP rose more sharply and peaked earlier at about the same level as post-Lie, reversing downward at beat 4 instead of at beat 6. Thus, it appeared that there was a cardiac chronotropic release that occurred immediately with the Lie and that was delayed a few beats in post-Lie. Cardiac inotropic and TPR activations countered this release such that MAP increased across four to six beats to a level that was consistent across Lie and post-Lie, starting from a lower value in post Lie.

Pre-Lie Patterns

The pre-Lie patterns differed somewhat from Lie and post-Lie for all of the measures. Pre-Lie HR was similar to post-Lie, increasing to a peak at beat 4 and then reversing downward. At about beat 10, pre- and post-Lie diverged sharply. Pre-Lie HR increased to about the beat 1 level, while post-Lie HR remained depressed about five beats per minute.

Pre-Lie contractility was sharply lower than Lie and post-Lie during beats 1 through 5 and pre-Lie SV was lower during beats 1 through 6, but mainly at beast 2 and 6, with an upward oscillation between those two beats. The lower SV values and oscillation were reflected in Q_dot. Conversely, pre-Lie TPR was higher than Lie and post-Lie, and oscillatory from beats 1 through 7. Pre-Lie MAP began higher and reversed downward earlier than Lie and post-Lie, but was no different after beat 6.

Thus, at the beginning of the 20-beat epoch, pre-Lie cardiac chronotropy (HR) was relatively high, while inotropy (contractility) was relatively low. Inotropy increased at about beat 6 and there was an oscillation in TPR from beat 3 to 7. Both SV and Q_dot oscillated, also, while MAP increased smoothly to beat 3, and then began a downward slide.

Update

In a search of the literature conducted for the writing of this article, I did not find any attempts to replicate the findings noted in the original technical report (Meyerhoff, Saviolakis, Koening, & Yourick, 2001; Ryan, Pavlidis, Rohrbaugh, Marchak, & Kozel, 2003; Taylor et al., 2011). It remains to be seen whether the observations reported here are robust enough to enhance lie detection capabilities. To answer that question, this investigation should be replicated independently using the same kinds of concealed information and other kinds of concealed information. My expectations about dynamic patterns associated with lies and true responses, above, may be cast as testable hypotheses. In addition, investigations are needed to determine whether one or more measures of cardiovascular dynamics, combined with the SCR measure, will predict the occurrence of a lie at a statistically higher rate than SCR alone. If the pattern suggested here of tonic and evoked responses is consistent across experimental replications, an optimal linear model of the cardiovascular dynamics of lying might be generated through linear or non-linear combination rules.

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