Autonomic Nervous System
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Social Psychophysiology for Social and Personality Psychology
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Overview

The autonomic nervous system (ANS) functions to mobilize energy and deliver oxygenated-blood to the body. The ANS changes during sleeping and waking states, postural changes, and physical movement. Moreover, ANS changes occur during mental states such as stress, emotion, cognitive, and motivation. This document provides an overview of some of the more commonly used measures in stress studies that can be obtained relatively non-invasively. We describe the process of obtaining ANS responses including technological requirements for lab and field experiments, and the specifics of obtaining, scoring, quantifying, analyzing, and interpreting ANS responses.

To begin we include a table with some of the most commonly measured peripheral physiological responses obtained in studies examining acute and chronic stress. We include the abbreviation, definition, and the equipment needed to measure the response. Below we offer more context to obtain these measures.

Table 1. Descriptions of commonly used ANS measures and sources of the responses

Common abbreviations of ANS measures and their unit of measurement	**Definition**	**Equipment to source the measure**
HR (bpm)	Heart rate typically reported in beats per minute.	Pulse meter *or* Electrocardiograph
IBI (ms)	Interbeat interval reported in milliseconds. IBI is the preferred measure of chronotropic cardiac activity	Electrocardiograph
SV (ml)	Stroke volume measured in milliliters. It represents that amount of blood ejected on each heart beat.	Electrocardiograph *and* Impedance cardiograph *or* Phonocardiogram

CO (L)	Cardiac output measured in liters. Calculated as: HR x SV = CO Represents the amount of blood ejected from the heart during one minute	Electrocardiograph *and* Impedance cardiograph *or* Phonocardiogram
PEP (ms) (aka VC)	Pre-ejection period (also referred to as ventricle contractility) is a time-based measure that is determined as the time from the left ventricle contracting to the opening of the aortic valve. These two time points are often referred to as from point Q (on an ECG trace) to point B (on a dz/dt waveform) Ventricle contractility is a different score that is calculated from two different PEP measures and multiplied by -1 so that increases in VC represent increases in SNS. $PEP_{task} - PEP_{baseline} = PEP_{change}$ PEP_{change} x -1 = VC	Electrocardiograph *and* Impedance cardiograph *or* Phonocardiogram
LVET (ms)	Left ventricular ejection time is a time based measure determined from the B point on the dz/dt wave to the x point on the dz/dt wave	Impedance cardiograph
HI (ohm/sec2)	Heather Index is a measure of aortic contractility. It is derived as the ratio of dZ/dt max to Q-Z interval (electromechanical time interval). This index has been shown to be especially sensitive to changes in cardiac contractility	Electrocardiograph *and* Impedance cardiograph
EMS (ms)	Electrical mechanical systole is the total time from the left ventricle contracting to the aortic valve closing. Determined from the Q point on the ECG to the X point on the dz/dt or by adding PEP + LVET.	Electrocardiograph
T wave amplitude (volt)	T-wave amplitude is the change in amplitude of the T-wave between tasks. Increases in T wave are thought to be related to more SNS	Electrocardiograph

	activity.	
SBP (mmHg)	Systolic blood pressure measured in millimeters of mercury. SBP refers to the maximal blood pressure and occurs when the ventricles of the heart contract.	Blood pressure monitor
DBP (mmHg)	Diastolic blood pressure measured in millimeters of mercury. DBP refers to the minimal blood pressure and occurs when the ventricles are most relaxed.	Blood pressure monitor
PP (mmHg)	Pulse pressure measured in millimeters of mercury. Represents the difference between maximum blood pressure and minimum blood pressure. Calculated as: SBP-DBP = PP	Blood pressure monitor
MAP (mmHg)	Mean arterial pressure in millimeters of mercury. MAP refers to a type of average of blood pressure, but in this case SBP and DBP are not weighted equally, DBP is weighted more. One formula used to calculate MAP is: 1/3(SBP – DBP) + DBP = MAP	Blood pressure monitor
TPR (resistance units)	Total peripheral resistance is a measure of the overall resistance in the vasculature, specifically the arterioles. Along with cardiac output, TPR is the determinant of BP. Because TPR is difficult to measure directly it is derived using the following formula: (CO/MAP) x C = TPR C=constant, typically 80	Electrocardiograph *and* Impedance cardiograph *and* Blood pressure
RSA (ms)	Respiratory sinus arrhythmia (aka high frequency heart rate variability, HF HRV) is a type of heart rate variability in which spectral analysis is used to derive the high frequency component of the IBI cycle (.12 to .40 Hz).	Electrocardiograph
SDNN	Standard deviation of normal to normal heart beats. A measure of heart rate variability defined as the standard deviation of Interbeat intervals.	Electrocardiograph[a] Blood pressure Photoplethysomograph

RMSSD	Root mean square of successive differences. A measure of heart rate variability calculated as the square root of the mean squared difference of successive normal to normal heart beats.	Electrocardiograph[a] Blood pressure Photoplethysomograph
RR	Respiration rate refers to the number of breaths per minute that occur in the high frequency range (typically 12 to 20 breaths per minute).	Respiration band *or* Impedance cardiograph
RA or RD	Respiration amplitude or depth refers to the difference in chest circumference during inhalation compared to exhalation.	Respiration band *or* Impedance cardiograph
SCR (μS)	Skin conductance reported in microSiemens. Indicates the amount of activity in the eccrine glands and is tied to a specific event or stimulus.	Skin conductance
NS-SCR	Non-specific skin conductance responses are reported in terms of the number of these events per minute (typically between 1 and 3)	Skin conductance
SCL(μS)	Skin conductance reported in microSiemens. Indicates the amount of activity in the eccrine glands and, unlike SCR, is a time based measure of overall level of SC.	Skin conductance
FPT(ms)	Finger pulse transit time reported in milliseconds is determined by the time between the left ventricle contracting (Q-wave on ECG) and the height of a pulse wave form at the finger. Shorter FPT indicates that the pulse detected at the finger relative to the heart contracting traveled faster than longer FPT. FPT is inversely (though not perfectly) related to blood pressure.	Electrocardiograph *and* Photoplethysmograph attached at finger
FA	Finger pulse amplitude refers to height or amplitude of the pulse waveform detected at	Photoplethysmograph attached

	the finger. Typically examined as changes (reactivity) from one task (e.g., baseline) to another. Increases in FA indicate more local dilation of the vessels in the finger, whereas decreases in FA indicate local constriction.	at finger
EPT	Ear pulse transit time, similar to FPT, is a time based measure determined from the left ventricle contracting (Q wave on ECG) to the height of the pulse wave at the ear	Electrocardiograph *and* Photoplethysmograph attached at finger

Note. [a] The preferred source for the measure.

Which measure should I obtain? The question of what type of equipment one should obtain needs to be guided by what types of research questions will be explored. Researchers should not find themselves in the position of developing research questions around the equipment they have, but rather, the research questions should dictate the physiological equipment they obtain. Here, general guidelines for setting up a lab are described, delaying discussion of specific measures until the following section.

In order to set up a social psychophysiology lab one should consider a couple of issues up front. Generally, as for all human behavioral science laboratories, noise and comfort are important. Laboratories should be climate controlled, allowing a comfortable range of ambient temperatures. They also should be free of distractions in the form of sights and sounds from both lab equipment and personnel and the larger external environments (e.g., buildings and surroundings) in which they exist. For experiments in which participants are sitting, be sure that the participant's chair is comfortable and has arms on which participants can rest hands and arms comfortably especially if there are sensors applied to them. Some blood pressure monitors, for example, require the sensor to be placed on the wrist (e.g., tonometric technology) and even slight changes in arm elevation can influence the readings.

Regarding the lab space, a private room to apply sensors to participants is ideal. In some cases, the application of sensors requires participants to partially disrobe—exposing torsos, unbuckling pants—that can best be accomplished with privacy. This room can double as the testing room in which the participant remains for the study or a separate one, but a room in which two people – the experimenter and the participant—can move about without interference from equipment, furniture and each other. Separate or not, the *testing* room, where the experiment takes place, should be relatively comfortable and quiet because for most studies one needs to obtain quiet resting baseline data prior to beginning the formal experiment.

For most experiments, baseline data are typically obtained while participants are seated. However, if for some reason, participants will be standing or supine, during the formal experiment, one would want these positions replicated during the baseline period. If an experiment necessitates a reclining position—for example, a peripheral physiological study to complement a neuroimaging study—then obtaining a reclining baseline to compare responses is necessary. This is because body posture can not only influence blood flow through the heart, but might also influence emotional and mental states (Harmon-Jones, et al., 2009; Mendes & Barrett, 2010). For example, Harmon-Jones and colleagues recently reported that participants who were

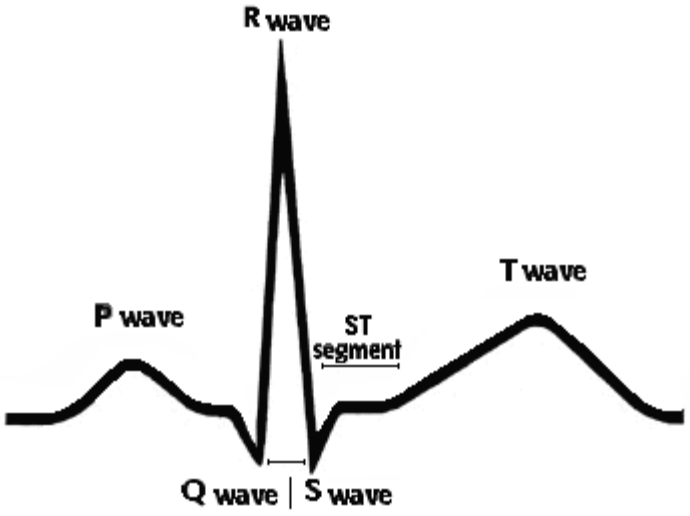
reclined showed less shifts in EEG left frontal asymmetry during an anger evocation compared to those sitting upright.

In the next section, we turn to the topic of equipment and various ANS measures that can be collected. We describe various ways to collect, edit, and quantify these responses. For organizational purposes the various measures are separated into cardiac responses (electrocardiography and impedance cardiography), hemodynamic responses (blood pressure), and peripheral responses (skin conductance). At the end of each of these sections we review social psychological research that has capitalized on these measures. For a summary of the measures, definitions, and common modes of collection see Table 1.

CARDIOVASCULAR (CV) MEASURES

In simplest terms, the cardiovascular (CV) system consists of the heart and pathways (vessels) through which oxygenated blood is delivered to the periphery and deoxygenated blood returns to the heart. Importantly to social psychologists, this system is responsive to affective states, motivation, attention, and reflexes. Additionally, CV responses have been linked to vulnerabilities in physical and mental illness. In this section we review several methods that examine changes in the cardiac cycle: electrocardiogram, respiration, and impedance cardiography.

Electrocardiograph and Respiration



The heart produces an electrical signal that can be measured via an electrocardiogram (ECG). A normal ECG recording is composed of various inflections (i.e., changes in direction or slope) referred to as P, Q, R, S, and T waves (Figure 1). Each heart cycle begins with an electrical impulse from the sinoatrial node (not detected on the ECG wave), which results in a depolarization of the atria (P-wave). The QRS complex represents the depolarization of the ventricles and the T inflection indicates repolarization (or recovery) of the ventricles. These inflections in combination can be used to determine a variety of chronotropic, (i.e., time-based) measures such as the time of one complete heart cycle, known as heart period (or interbeat interval [IBI]). This measure is the inverse of heart rate (beats per minute) though heart period is the preferred metric because of its statistical properties (see Berntson, Cacioppo, & Quigley, 1993).

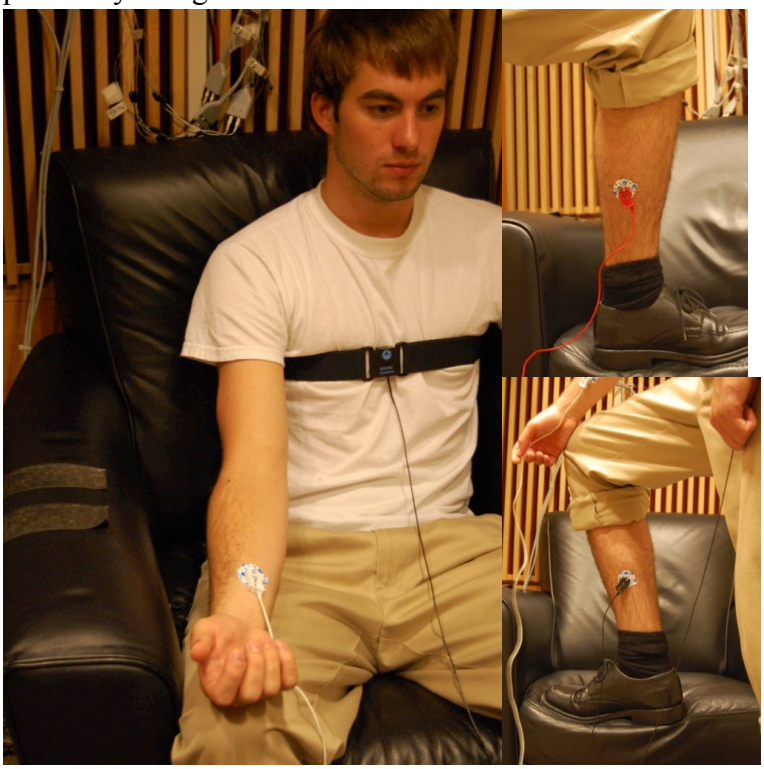
Equipment. There are many equipment options available for ECG recording and it is preferable to record the ECG waveform rather than obtaining a summarized data point like heart rate (HR). Though not completely uninformative, simply collecting HR and mean inter-beat interval (time between the R-points) limits the researcher's ability to calculate more sophisticated measures (see below and Table 1). In other words, it is relatively easy to collect the full ECG waveform and the benefits outweigh the costs.

Preparation and recording. ECG waveforms can be collected from several spot lead configurations of sensors (e.g., 35 mm electrodes) on the limbs. Described below are three standard configurations in which placement results in an upward deflection of the Q-R complex:

Lead I: Electrodes are attached just above the right and left wrists on the inside of the arms. The left arm has the positively charged lead.

Lead II: Electrodes are attached on the right arm and left ankle. The ankle has the positively charged lead.

Lead III: Electrodes are attached on the left wrist and left ankle. The ankle has the positively charged lead.



Lead placements can be adjusted so that the sensors are placed on the torso rather than the limbs. For example, a modified Lead II configuration places the right lead below the sternum and the left lead on the left side of the torso below the ribcage. Torso placement might be preferable over limb placement if there is anticipated movement of the limbs or for younger participants (i.e., babies and toddlers). In Figure 2, the participant has an ECG placement in a standard lead II configuration – right arm, left leg, and a ground wire attached to the right leg. He is also wearing a respiration band to track rate and depth of this breathing. This configuration is appropriate for obtaining heart rate variability (see more details below). Note that in this case we did not shave his legs to apply sensors. Though it would be preferable to have hairless skin, the ECG wave is especially strong and can be reliably obtained even with interference from hair.

The experimenter should apply sensors to participants. This is done to insure proper and consistent placement across participants. Preparing the site for ECG placement can include a gentle abrasion of the skin and a subsequent application of a thin layer of conductance gel, but in many cases a clean signal can be obtained without either given the relatively strong electrical signal of the heart.

Several factors can interfere with an ECG recording. First, excessive hair, either on the ankles or chest, can make recording difficult if using adhesive sensors. Shaving participants' ankles or torso is optimal, but not always possible. Either adjusting the sensor location or using additional medical tape to secure the sensor might reduce noise. Another potential problem is participant's skin type or changes in skin temperature during the course of the experiment, which can result in the sensors slipping or losing the pulse connection. Skin that is especially oily or prone to sweat might require additional taping of disposable sensors. Good lab practice includes taping the sensors with medical tape, and this is especially true in summer months or for longer studies, when the risk of warmer skin temperature is greater.

Collecting the signal: sampling speed, filters and amplification. Typically equipment (hardware) for collecting ANS responses allows for a specification of *sampling rate*. This is the number of samples per second that the computer records. Choosing sampling rate should be determined by the measure being collected—with slow moving waves one can have a slower sampling rate, but with faster moving signals one needs a faster sampling rate. For example, skin temperature changes slowly and thus can be sampled at a lower sampling rate like 200 Hz – or 200 samples per second. In contrast a quick moving signal like ECG should be sampled at a higher sampling speed (e.g., 1000 Hz) so that all the possible inflections and deflections are properly traced.

Typical equipment also allows for a selection of filters. The function of filters is to remove or reduce portions of the signal that are irrelevant for the signal of interest. There are four common types of filters: low pass, high pass, bandpass and notch filters. A low pass filter allows frequencies *below* a set frequency value to pass, whereas a *high pass* filter does the opposite – frequencies *above* a set frequency are allowed to pass. For example, the alpha rhythm from EEG ranges from 8 – 12 Hz. To insure that only signals in this frequency range are collected an experimenter could set a low pass filter to 12 and a high pass filter to 8, leaving only the desired frequency range for collection. A bandpass filter is simply a filter with low and high pass settings. A notch filter is a filter that attenuates a small range of frequencies. The most common notch filters are ones that attenuate AC current. In the US, AC current is set at 60 Hz, so a 60 Hz notch filter is often used.

The ECG waveform needs to be appropriately amplified. Amplification is the process of adjusting the strength of the signal so that a specific intensity of the signal is obtained. For most physiological signals discussed in this chapter the optimal amplification for the signal is 1 volt. Because the intensity of any waveform may change during the course of an experiment, one should amplify the signal at the beginning of the experiment, prior to baseline. This is accomplished by adjusting the *gain* on the amplifiers. If post acquisition software will be used that allows adjusting the amplification post-acquisition (e.g., Acknowledge software, produced by Biopac, allows this option), collect a small amount of data prior to the beginning of baseline or use the first minute of the data collection (presumably a baseline period) to determine how much the signal needs to be amplified and then amplify the entire signal by that amount.

Editing and quantification: ECG and HRV. Editing an ECG waveform is typically done off-line—that is once the session is complete. The primary concerns when editing an ECG

waveform are the removal of artifacts and the proper identification of the R inflection. Another critical point on the ECG waveform is the Q inflection—or the point at which the left ventricle of the heart contracts. The Q inflection, along with the B inflection from the $\Delta z/\Delta t$ impedance wave (see impedance cardiography section), is critical for the calculation of pre-ejection period (PEP), which is one of the purest measures of sympathetic nervous system activation.

Collection of the ECG trace also allows for the estimate of heart rate variability. HRV is influenced by a number of factors, but by deconstructing the variability one can isolate heart period changes due primarily to parasympathetic control, sympathetic control, or a combination of both. Of particular interest to psychophysiologists is high-frequency (HF) HRV because changes in variability in this range are believed to be due primarily to control of the vagus nerve and thus primarily an index of parasympathetic control. There are several measures of HRV estimates, time-domain, frequency domain, and non-linear measures (a full committee report by the Society for Psychophysiological Research is available for more details, Berntson, Bigger, & Eckberg, 1997). Here we briefly review some of the estimates and what is needed to calculate these measures.

One of the simpler measures of HRV is based on time-domain estimates, for example RMSSD (root mean square of successive R-R differences), which is calculated as the standard deviation of the beat-to-beat intervals. A popular frequency-domain technique to estimate HRV involves decomposing heart period variance into different frequency bands using Fourier transformations. For example, the HF band (high frequency band) ranges from .15 to .4 Hz (cycles per second) and is thought to represent primarily vagal influence and as such parasympathetic activity. Lower frequency bands (< .15) have also been identified and in these frequency domains the influence can be either sympathetic or parasympathetic.

Respiration can influence heart rate and heart rate variability. In Figure 2, the participant is wearing a respiration band on his chest to monitor both his respiration rate and the depth of his breath. Collecting respiration parameters are especially important if one wants to measure indicators of heart rate variability (HRV) because respiration can directly influence HRV – during inspiration the influence of the vagus nerve on the heart is removed and the heart rate accelerates; during expiration, the vagus nerve is applied and heart rate decelerates. One commonly debated measurement issue in HRV research concerns the importance of controlling for respiration rate and depth in HRV analysis. For a thorough understanding of the complexities of this issue, see Denver, Reed, and Porges (2007) for justification that respiration frequency need not be included in estimates of RSA/HRV and Grossman and Taylor (2007) for a discussion of why respiration measures are important.

Respiration can be measured a number of ways. One option is to use a strain gauge that measures pressure during inspiration and expiration, and rate and depth of breaths can be extracted. With a single strain gauge the recommended placement is high on the torso immediately under the arms (and above the breasts). This placement will allow for measurement of upper respiration, but not lower abdominal respiration, which may be important if the research focuses on deep breathing found in meditation or other focused breathing domains. In this case, two strain gauges can be used to provide both upper and lower respiration. Another option is to use impedance cardiography (see below), which can extract respiration rate and depth.

Applications of heart rate variability in social psychology. Initially, heart rate variability was believed to be a measurement artifact or nuisance, but further exploration into spontaneous changes in the timing of the heart cycle proved to be psychologically and physiologically meaningful. Though there are still disagreements on the specifics related to

measurement, quantification, and psychological meaningfulness of vagal tone and cardiac vagal reactivity (see *Biological Psychology, 2007, vol 74*), these measures might prove to be especially important for social psychologists interested in emotion and/or mental effort.

Though most work has focused on resting/baseline RSA (a type of HF heart rate variability) and its links to dispositions and responses to social and emotional situations, there is also a growing literature on vagal reactivity – focusing on RSA changes – and vagal rebound. Vagal rebound is the extent to which RSA responses return to or even over-shoot baseline levels after some suppression of the vagal brake. Below we describe some literature exploring these various components of HRV.

One theory that has received much attention in terms of the inferences one can draw from heart rate variability is Porges' polyvagal theory (e.g., Porges, 2007). In this theory, Porges argues that vagal regulation stemming from the nucleus ambiguous and enervation from cranial nerve X acts on the vagus nerve to modulate heart period. The polyvagal theory further specifies that primates uniquely have vagal nerve modulation (but see Grossman & Taylor, 2007), which has evolved as part of the social engagement system. One of the primary postulates of polyvagal theory is that social factors (affiliation, social engagement) or personality factors (optimism, bonding, compassion) can modulate vagal activity. Specifically, Porges argues that higher RSA (high cardiac vagal tone) can be used as an index of adaptive emotional regulation and responsiveness to the social environment. Similarly, cardiac vagal reactivity might also index appropriate social engagement in that increased vagal reactivity might be associated with calmness, equanimity, and a lack of distress.

Adding some complexity to these effects, however, is the nature of the social context. Indeed, in highly stressful situations or tasks that require mental attention or effort then one should expect a withdrawal of the vagal brake (resulting in lower RSA). Indeed, cognitive psychophysiologists have used decreases in RSA as an index of attention or mental effort (Tattersall & Hockey, 1995). In one study, relying on this type of interpretation for HRV reactivity, Croizet, et al (2004) examined changes in RSA during a stereotype threat paradigm. They found that participants assigned to a stereotype threat prime had greater decreases in RSA and poorer performance than those in the control condition and that RSA changes mediated the relationship between stereotype threat and performance.

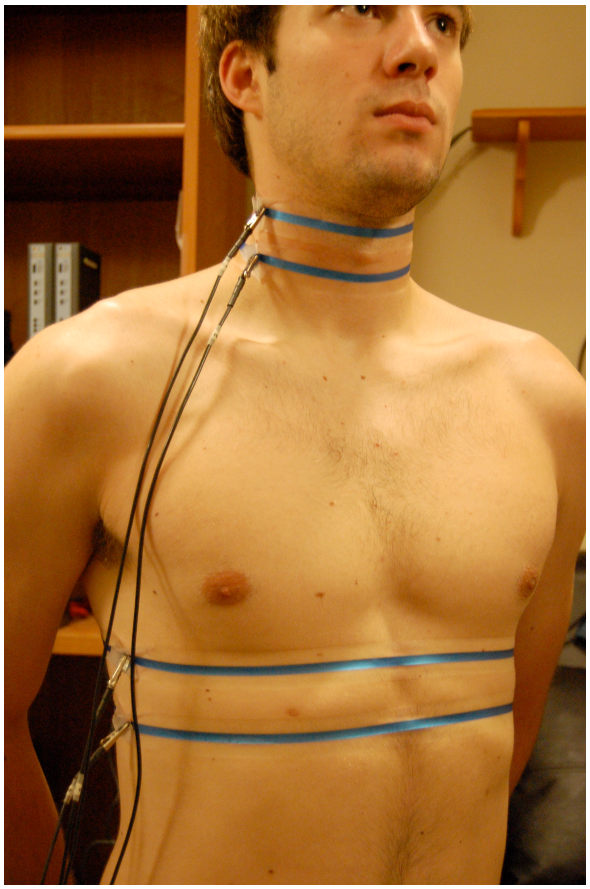
Applications of cardiac vagal tone and vagal reactivity are increasing in personality and social psychology. Some applications have focused on the extent to which dispositional emotional styles are linked with cardiac vagal tone (Demaree & Everhart, 2004; Oveis, et al., 2009; Sloan, et al., 2001). For example, individuals with greater hostile tendencies have lower cardiac vagal tone at baseline, during an emotional induction task, and at recovery than those low in hostility (Demaree & Everhart, 2004; Sloan, et al., 2001). Similarly, but on the brighter side, Oveis and colleagues found that those higher in optimism had higher vagal tone. Accumulating evidence suggests that vagal tone might be a reasonable physiological response to index general positive and negative affect.

Social psychologists have examined changes in RSA (RSA reactivity), relying on the inference that greater decreases in RSA are associated with distress and negative affect, to examine why implicit goal setting might result in improved performance. In previous studies, participants who exaggerated reports of their GPA tended to improve more than those who did not exaggerate (Willard & Gramzow &, 2008). However an open question was if exaggeration was benign and serving a type of implicit goal setting or was exaggeration a form of anxious repression. To examine this question, participants first reported their GPA and course grades in

private and then met with an experimenter to review their academic history (Gramzow, Willard, & Mendes, 2008). During this interview participant's ECG and respiration was recorded and RSA responses were calculated. Participants who exaggerated their GPA showed greater RSA increases from baseline to the interview, suggesting that participants who exaggerated their GPA were not necessarily anxious about exaggerating their achievements. Additionally, those who had greater increases in RSA when discussing their GPA tended to improve their GPA in a subsequent semester. Converging evidence from nonverbal behavior coded during the interview suggested that exaggerators appeared composed rather than anxious supporting the interpretation that higher RSA while discussing one's GPA was associated with equanimity rather than anxiety.

Impedance Cardiography

Impedance cardiography is a non-invasive technique to estimate blood flow changes in the heart. This technique allows for estimates of how much blood is ejected during each heart cycle (stroke volume; SV), and various changes in the cardiac cycle such as the timing of the aortic valves opening and closing.

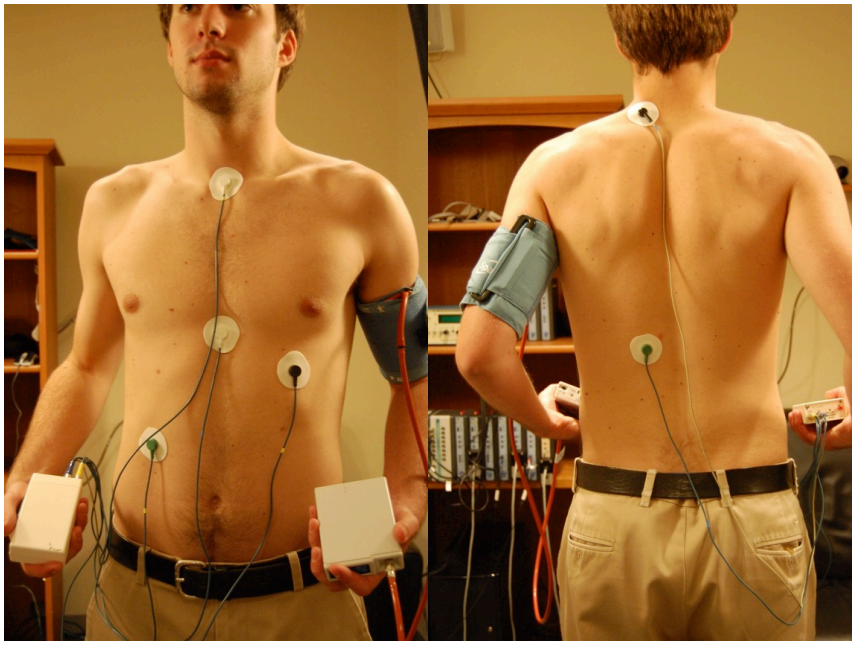


Impedance cardiography requires the use of either spot or band electrodes placed on the torso. In the figure to the left, the participant is wearing band electrodes that completely encircle his neck and torso. Two bands are placed around his neck and two around his torso, with the upper torso band placed directly on his xiphisternal junction (i.e., right under the sternum), and the lower torso band placed 3 cm below the upper band. Similarly the lower neck band is placed low on the neck and the upper neck band is placed 3 cm above that. Impedance cardiography employs an output of frequency modulated electrical current (ranging from .1 to 4 mAmps) to the two outer sensors (upper neck and lower torso), and the inner sensors detect the impedance (i.e., AC resistance) to the incoming current. Impedance values represent global blood flow in the thoracic cavity (typically referred to as Z_0 or basal impedance). As the blood volume increases the impedance decreases. The first derivative of the waveform, $\Delta z/\Delta t$, is the change in basal impedance over the change in time, which provides a waveform that allows for an estimate of the total amount of blood volume ejected from the heart on a single beat (i.e., stroke volume).

An impedance spot electrode placement is shown below using an ambulatory impedance machine (VU-AMS). This 6 spot sensor configuration allows for collection of both ECG waveform and $\Delta z/\Delta t$ waveform. The specific placement of these sensors is:

1. ECG: over the jugular notch of the sternum, between the collar bones
2. ECG: under the left breast, 4 cm under the nipple, between two ribs rather than on a rib
3. ECG: at the right lateral side, between the two lowest ribs

4. ICG: over the xiphoid process of the sternum
5. ICG: at the base of the neck (vertebrae C3/C4) and at least 3 cm above electrode 1
6. ICG: below the line connecting the tips of the shoulder blades (vertebrae T8/T9) and at least 3 cm below electrode 4



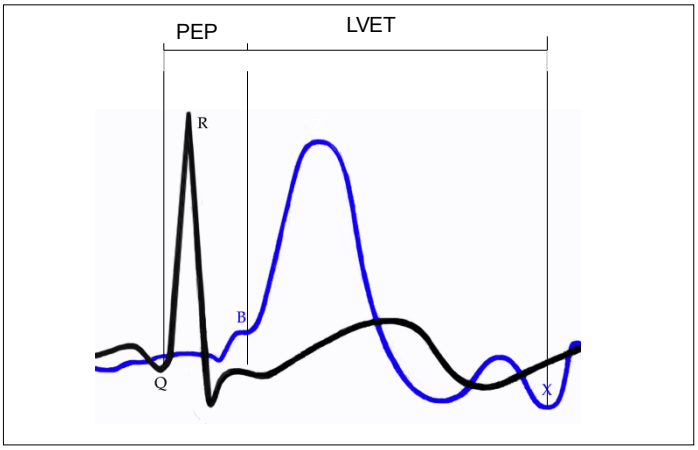
The combination of impedance and electrocardiograph produce several cardiac indicators. Figure 5 shows the $\Delta z/\Delta t$ waveform superimposed on an ECG waveform, which show several critical inflection points. For example, a chronotropic (time-based) measure of ventricular contractile force is *preejection period* (PEP). This is the time from the left ventricle contracting (Q inflection on the ECG wave) to the aortic valve opening (B inflection on the $\Delta z/\Delta t$ waveform). Preejection period is considered one of the purest measures of sympathetic activation. The shorter the time between the left ventricle contracting and the aortic valve opening indicates greater sympathetic nervous system activation. *Left-ventricular ejection time (LVET)* is also a time-based measure determined from the B and X inflections on the $\Delta z/\Delta t$ waveform. *Stroke volume*, the amount of blood ejected from the heart on any given cardiac cycle, is a volume based measure that requires the identification of the B and X- inflections on the $\Delta z/\Delta t$ waveform to determine the time when blood is being ejected into the aorta, along with the maximum point of the $\Delta z/\Delta t$ waveform on the given cardiac cycle labeled Z^2. The area under this curve is then estimated to determine stroke volume (see formulas below).

Stroke volume provides an estimate of the amount of blood ejected at each beat. The overall indication of how much blood is being pumped out of the the heart at any given time is expressed as *cardiac output* (CO) in liters per minute. Cardiac output is simply the product of SV and HR: (SV x HR)/1000. The metric for stroke volume is in milliliters, so the product is divided by 1000 to convert to CO which is reported in liters. Because CO is a combination of both heart speed and blood volume pumped by the heart, it is believed to be a measure of cardiac efficiency.

When using impedance cardiography in a laboratory setting, it is important to instruct participants to wear comfortable, two-piece clothing to the experiment. As the bands (or spots) require placement on the torso, directly on the skin, participants are required to lift their shirts to expose their torso. Additionally, placement of the neck sensors might be impeded by clothing that is snug at the neck. A well-equipped lab will keep loose shirts and pants for participants who arrive in clothing that would make attachment of the bands difficult.

Collecting impedance data. Similar to ECG a common sampling rate for impedance cardiography is 1000 Hz. A low pass filter set is typically set at 50 Hz. Also, the $\Delta z/\Delta t$ waveform

should be amplified to 1 volt. Note that by adjusting the amplifier in this way, the amplitude of the Z-point (a major component of SV/CO) will compromise individual differences in SV and CO, however the important point is that because of the variability in sensor placement of the bands/electrodes and individual differences in body morphology, it is highly questionable whether any single (raw) score of SV or CO is valid. Any minor variation in placement of the sensors can dramatically change the amplitude of the Δz/Δt waveform. Because of this and related problems, researchers typically examine *changes* (or reactivity) in SV and CO. This is also important in longitudinal studies. Unless there is assurance that the band electrodes were placed exactly in the same location on the body at every assessment, comparing SV or CO over time using a single time point might be highly questionable. However, the chronotropic components of the cardiac cycles that rely only on the x-axis points (PEP, LVET, etc) are valid as single or repeated point time estimates because they do not consider the y-axis in their calculations and thus the amplitude of the waveform is irrelevant.



Editing and quantification. Probably one of the greatest challenges for researchers interested in impedance cardiography is how to edit and summarize the data. There are important choices to be made regarding how the data are summarized for editing. One option uses ensembled waveform averages. This method determines the composite or average waveform across some specified time period (typically between 30 s and 5 minutes). By "ensemble averaging" the waveforms over time, random noise and movement are removed and a more representative cardiac cycle can be obtained. Another option is to determine blood volume changes on a cycle-to-cycle basis (see SPR committee guideline paper, Sherwood, et al., 1990).

In addition to how the data are averaged, there are also several formulas that can be used to estimate stoke volume. The Kubicek equation estimates SV from the derivative of the impedance signal and blood resistivity:

$SV = \rho \times L^2 / Z_0^2 \times \Delta Z / \Delta t_{max} \times LVET$

Where: ρ = 135 (blood resistivity)
L = distance between electrodes
$\Delta Z / \Delta t_{max}$ = peak amplitude of $\Delta Z / \Delta t$
LVET = left ventricle ejection time (time in ms between B and X)

More recently, other equations have been offered that might be superior to the Kubicek equation. For example, the Sramek-Bernstein estimates SV from the volume of electrically participating tissue scaled according to body surface:

$SV = \delta (VEPT) / Z_0 \times \Delta Z / \Delta t_{max} \times LVET$

Where: $\delta (VEPT) = weight_{max} / weight_{ideal} \times (0.17H)^3 / 4.25$
Participant's height, weight, and ideal weight are needed

Regardless of the equation used, one of the most critical decisions involved in scoring impedance data is accurately identifying the *B* and *X* points on the Δz/Δt waveform. Though tremendously time and labor intensive the technique that assures most accuracy is visual

detection of these points. Specifically *B* should be placed at the beginning of the longest uphill slope before the Z-point and X is typically the lowest point after the Z-point.

Applications of impedance cardiography. Cardiovascular responses have been used extensively in the areas of motivation, emotion, and stress. Interests in these measures are further fueled by the possibility that certain patterns or response profiles of CV responses repeatedly experienced over time might be linked to health outcomes. For example, early work linking type A personality and coronary heart disease examined CV responses as one of the likely mechanisms through which physical health was affected. Specifically, it was theorized that excessive CV responses would create tears in endothelial lining resulting in greater calcifications and plaque build-up that could possibly initiate ischemic events or strokes. Primarily, CV responses in this context included heart rate (heart period) and blood pressure responses.

A combination of cardiovascular and blood pressure responses is used in research attempting to index challenge and threat states. Though not without its critics (Wright & Kirby, 2003; see also Blascovich, et al., 2003), this theory attempts to differentiate motivational states using various CV measures, such as PEP, cardiac output, and total peripheral resistance (see below). This theory argues that in *motivated performance situations,* tasks that are active rather than passive and require some cognitive or behavioral responses, profiles of CV reactivity can differentiate approach from defeat orientation (Mendes, Major, McCoy, & Blascovich, 2008). Early work showed that task appraisals in which participants reported having greater resources relative to how demanding they perceived the tasks were associated with greater cardiac responses (shorter PEP [indicating greater ventricle contractility], increased HR and CO, and decreases in vascular resistance—lower TPR). This pattern of CV reactivity was believed to be a marker of the psychological state of *challenge*. In contrast, appraisals that showed greater perceived demands relative to resources to cope were associated with comparatively less CO and higher TPR (Tomaka, et al,. 1993). This profile of CV responses was thought to index *threat* states. Challenge and threat theory has been tested in a variety of social domains. For example, these indexes have been explored within dyadic social interaction when one member of the dyad is stigmatized. Stigmas were operationalized as physical stigmas (e.g., birthmarks), stigmas resulting from group membership (e.g., race/ethnicity), or socially constructed stigmas (e.g., accents, SES). Across more than a dozen studies, participants who interacted with stigmatized partners were more likely to exhibit threat (i.e., lower CO and higher TPR) than those interacting with non-stigmatized partners. If the results were found only with physiological responses they would have still been intriguing, but in many cases the physiological responses also correlated with other automatic or less consciously controlled responses such as cognitive performance, emotional states, and non-verbal behavior such as freezing, orientation away from the partner, and closed posture (Mendes, et al, 2008; Mendes, Blascovich, Hunter, Lickel, & Jost, 2007; Mendes, Blascovich, Lickel, & Hunter, 2002). Also of interest was the lack of correlations between participants' CV responses and their self-reported task appraisals and partner ratings. In contrast with the CV responses, self-reported partner ratings often showed a preference for stigmatized compared to non-stigmatized partners, suggesting that deliberate and consciously controlled measures might be more vulnerable to attempts to correct for racial bias (Blascovich, Mendes, & Seery, 2002; Mendes & Koslov, 2010).

In the personality domain, these measures have been used to assess individual's reactions to stressful situations. For example, individuals who score higher on belief in a just world scales (e.g., hard work is rewarded) tend to exhibit greater increases in cardiac and decreases in TPR

during stressful tasks than those who score lower on these scales who exhibited lower CO and higher TPR – consistent with threat profiles (Tomaka & Blascovich, 1994). Self-perceptions in the form of level and stability of self-esteem have been explored with these methods as well (Seery, et al., 2004). For participants with high and stable self-esteem, positive performance feedback resulted in more challenge responses than those with high and unstable self-esteem.

Loneliness appears to result in these profile patterns of CO and TPR as well (Cacioppo et al., 2002; Hawkley, et al., 2003). Cacioppo and colleagues have shown in various settings that individuals reporting higher levels of loneliness are more likely to exhibit lower CO and higher TPR than individuals reporting lower levels of loneliness. This effect has been found in both lab based settings in response to social evaluation, and field studies using ambulatory impedance and blood pressure devices. In the field study, due to lack of ability in determining whether individuals were actually in a *motivated performance situations*, the authors interpreted these profiles as indicating passive versus active coping styles (Sherwood, Dolan, & Light, 1990), with lonely individuals adopting more passive coping styles within the context of their day.

BLOOD PRESSURE

Blood pressure, measured in millimeters of mercury pressure (mmHg), refers to the amount of pressure on the vessel walls during the cardiac cycle. Distinctions are made between systolic blood pressure (SBP) and diastolic blood pressure (DBP), which represent peak pressure compared to lowest pressure in the arteries, respectively. Though correlated, these measures may provide unique information and are thus typically both obtained. For example, during stressful or emotionally provocative situations increases in SBP compared to DBP have been identified as part of an adaptive defense patterning (see Brownley et al., 2000). Systolic blood pressure responses have also been linked specifically to effort expenditure (Wright & Kirby, 2001). Health consequences have been associated with higher SBP and not necessarily higher DBP. For example, Chobanian, et al. (2003) reported that elevated SBP, and not necessarily DBP, predicted the development of coronary heart disease.

Although SBP and DBP are often presented separately, one will also find instances in which researchers combine the two in some meaningful way. For example, pulse pressure (PP) is calculated by subtracting DBP from SBP (PP= SBP – DBP). At rest, average pulse pressure is approximately 40 mmHg. During exercise SBP typically increases more so than DBP. Extremes in PP in both directions can indicate abnormalities. When PP is too high this is likely due to artery stiffness, leaky aortic valves, or hyperthyroidism and has been linked to cardiovascular complication (Blacher, et al. 2000). Low PP values, typically influenced by low stroke volume, can indicate abnormalities such as congestive heart failure. Another type of averaging is mean arterial pressure (MAP), which is calculated as a type of average (though not an exact mathematical average because DBP is weighted more given its longer time course within a given cardiac cycle), for example: $MAP \approx [(2 \times DBP) + SBP] / 3$.

Mean Arterial Pressure is often used in combination with CO to determine *total peripheral resistance* (TPR), using the formula: $TPR = (MAP / CO) \times 80$. Changes in TPR can be construed as an estimate of the amount of constriction versus dilation occurring in the blood vessels – specifically the arterioles. When the arterioles constrict less blood can flow to the periphery and this is indicated by an increase in TPR. In contrast, when arterioles expand, or dilate, this allows more blood flow and is indicated by decreases in TPR. Physiologically blood pressure is determined by TPR and CO, but in terms of measuring these parameters, technology is currently superior at measuring BP and CO, and so TPR is calculated from these measures.