Respiratory Blood Pressure Fluctuations Observed During Polygraph Examinations

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Abstract

The purpose of this paper is to propose possible explanations for what is truly being recorded on the cardiograph waveform during normal and deep breathing patterns. The basic principles underlying cardiovascular recordings monitored during a polygraph examination and what may be affecting the waveform as a result of the breathing patterns will be explored. The term "vagus roll," widely employed by the polygraph community to describe the undulating cardiovascular waveform, is physiologically meaningless and not used by the broader physiologic or medical scientific community. In lieu of this polygraph manufactured term, it is suggested the fundamentally more accurate and reflective expression, Respiratory Blood Pressure Fluctuation (RBPF) be used to describe the cardiovascular waveform undulations.

Introduction

The cardiovascular recordings observed during a psychophysiological detection of deception (polygraph) examination reflect a familiar pattern when the subject engages in deep breathing. Polygraph examiners often use the expression "vagus roll" to describe the phenomenon of a cyclical rising and falling of the cardiograph waveform synchronous with respiratory activity (Krapohl & Sturm, 2002; DoDPI, 2006). Explanations have been posited that include physical movement associated with respiration imparted onto the blood pressure cuff and neuronal influence mediated through the parasympathetic nervous system (Krapohl & Sturm, 2002).

A recent internet search for "vagus roll" did not reveal any use of the expression except by the polygraph community. To conform to the physiologic scientific community, respiratory blood pressure fluctuation (RBPF) is an expression which more aptly describes the origin of the cardiovascular oscillations observed during the polygraph examination.

The cardiovascular circulation is a closed system consisting of the heart muscle, arteries, capillaries, and veins. The purpose of the cardiovascular system is to transport nutrients, hormones, enzymes and oxygen to body tissues, and remove metabolic wastes and carbon dioxide.

In polygraphy, heart and blood vessel hemodynamics provide significant diagnostic data that can be used to assess subject veracity. Presently, and historically, cardiovascular recordings have been obtained with a partially inflated sphygmomanometer (blood pressure cuff). One of the diagnostic features often discussed in the polygraph literature is baseline arousal. Baseline arousal is a rise in the pulse waveform from a pre-stimulus level. Previous investigators have reported the primary cause of baseline arousal is an increase in blood pressure. (Geddes & Newberg, 1977; Handler, Geddes, & Reicherter, 2006). This paper will focus on the undulations of the cardiograph baseline observed during eupneic (normal) and deep or exaggerated breathing cycles and discuss possible underlying causes.

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Background

Blood pressure is a measurement of force per unit of area exerted on a blood vessel wall and is typically expressed in units of millimeters of mercury (mmHg) (Marieb, 1999). Since blood pressure in the vascular system oscillates between contracting and relaxing cardiac cycles, the pressure recordings are expressed in systolic (contracting) and diastolic (relaxing) values.

Due to the pressure oscillations between the systolic and diastolic phases of the cardiac cycle, the mean arterial pressure (MAP) may provide enhanced criteria signal value but has not been used by current polygraph scoring systems. Mean arterial pressure is not merely the average between the systolic and diastolic values because the diastolic phase of the cardiac cycle lasts about twice as long as the systolic phase. Since systolic phase is about one-third of the cardiac cycle, the mean arterial pressure can be calculated by multiplying the pulse pressure (systole-diastole) by one-third and adding that value to the diastolic pressure. For instance, a blood pressure of 120/80 would have a mean arterial pressure of 93 mmHg. Calculation: 120-80 = 40; 40 x 1/3 = 13; 13+80 = 93.

Blood flow rate is a function of pressure differentials and resistance factors which follow the principles of Ohm's law, (**I = V/R**). The **I** symbol of the Ohm's equation in hemodynamic measurements becomes the flow rate which can be represented by the symbol **F** (flow rate) or **V** (velocity). The systemic blood pressure source is generated by the contraction of the left ventricle. The power or force component in the Ohm's equation, represented by **V** (voltage) can be replaced by **P** (pressure). Change in pressure (delta P or P1-P2) is actually the more precise value but for the sake of the fundamental concept, **P** can be used. The symbol **R** (resistance) in hemodynamics is primarily determined by total vessel length with vessel diameter and viscosity contributing to the overall **R** value. With Ohm's law modified to: F = P/R, hemodynamic measurements as they apply to polygraph recording can be evaluated and contribute to the criteria for decision making.

Hemodynamic recordings are based on measurements as a function of time. The one-minute time unit is the customary time bar for this parameter. Cardiac output volume is calculated by multiplying the stroke volume (SV) or ejection volume (EV) of one cardiac cycle **x** the number of cycles generated in one minute. For example, a stroke volume of 80 ml **x** 75 cycles per minute would generate a volume of 5.6 L.

The cardiac volume output can be altered by a change in stroke volume capacity, heart rate or both. Stroke volume capacity can vary according to venous blood volume availability and heart muscle contractile strength. According to the Frank-Starling Law of the Heart principle, when venous return increases, the heart chambers respond by stretching to increase the end diastolic volume (EDV) or preload. On the next cardiac cycle, the systolic contractile force will eject a larger stroke volume which, in turn, increases the systemic pressure. If the EDV continues to increase, the stroke volume will usually continue to increase for several more cycles until the heart reaches a maximum cardiac output efficiency. Total cardiac output is a function of the Frank-Starling mechanism and the sympathetic nervous system influence on cardiac contractile cells. Since there is always some blood volume left in the ventricles after systole, the final stroke volume can be calculated by subtracting the end systolic volume from the end diastolic volume. SV = EDV-ESV.

Venous return is affected primarily by the respiratory pump and the muscular pump. During inspiration, the contraction of the diaphragm will cause an increase in abdominal pressure, and simultaneously decrease thoracic pressure. This combination of cavity pressure changes will act as respiratory pump and will cause more blood to return to the heart and stimulate the Frank-Starling mechanism. Within limits, the greater the inspiration dynamic, the greater the effect will be on the cardiovascular response.

The muscular pump coupled with the respiratory pump, also aids in venous return by squeezing certain veins strategically positioned between the muscles. Exercise such as walking and running compress the veins in the legs which greatly aid the

cardiovascular system return of blood to the heart.

The Valsalva maneuver can significantly alter blood flow dynamics and raise blood pressure. Compressing the abdominal muscles while exhaling through a partially closed glottis (opening of the voice box) can prevent cerebral blood loss and pooling in the legs. Combat pilots employ this maneuver to prevent black-outs from negative G forces. Variations of this activity can be used as a polygraph countermeasure.

Bainbridge (1915) observed that right atrial distention produced an increase in heart rate. He found the reflex arc responsible for tachycardia was mediated through an increase in the sympathetic effect and a decrease in the parasympathetic effect (Brownly, Hurwitz & Schneiderman, 2000).

Respiratory Sinus Arrhythmia (RSA) is a phenomenon that was first described by Ludwig in 1847 (Porges, McCabe & Yongue, 1982). The mechanisms responsible for RSA include CNS influence from the cardiac and respiratory centers, afferent feedback from stretch receptors in the lung (Hering-Breuer reflex), ventilation dynamics and barorecpe-tors in the aortic and carotid sinuses. (Porges, McCabe & Yongue, 1982)

In summary, there are several physiological mechanisms which affect cardiovascular dynamics. Cardiac output and blood pressure can be influenced by central and peripheral sensory receptors. Vasomotor function such as venous return mechanisms and arterial resistance factors can have a major influence on cardiovascular changes during the polygraph examination experience.

Observation

During polygraph examinations, the cardiovascular waveform normally maintains a relatively stable baseline. There are times, however, when the waveform undulates. As discussed above, this undulating pattern has been erroneously referred to as a "vagus roll."

Figures 1 and 2 show examples of a respiratory effect on the cardiovascular channel. Both figures are from the same subject and the sensors were not moved between chart recordings. While collecting the data shown in Figure 1, the subject was instructed to answer "yes" or "no" to the test questions. Note the peaks of the respiration lead the peaks of the cardiograph waveform by approximately two seconds. Figure 2 is a screen shot of data collected using a "Silent Answer Test." The undulations of the cardiograph waveform have decreased markedly during the more typical 14-16 eupneic breathing cycles.

Figure 3 is from a different subject in a laboratory setting. This subject was instructed to sit quietly and not attempt to control breathing cycling. The blood pressure cuff was placed in contact with the subject's chest during the polygraph recordings. A matched peak-to-peak synchrony between the respiration and blood pressure waveform cycles can be observed. Note the difference in the peak-to-peak timing between the two waveforms when comparing Figure 1 and Figure 3.

Discussion

There appears to be a difference in undulating waveforms produced when the cuff is in contact with the subject and when the cuff is not in contact. Respiratory influenced undulations can be caused by cuff to subject contact but also can occur when there is no such contact. The former results in a waveform in which the peak-to-peak synchrony is very closely matched in time. The latter produces a waveform in which the peaks are more delayed. Differentiating between the causes of the undulations then becomes a matter of comparing the peaks of the waveforms.

During breathing, vasoconstrictor neurons are activated in the inspiratory phase leading to rhythmic vasoconstriction of blood vessels controlling blood pressure (Janig, 2006). Bursts of sympathetic activity in human muscle vasoconstrictor neurons are generated by pulsatile unloading of baroreceptors (Janig 2006). Increased vasomotor constriction results in increased blood pressure. Increasing the depth of breathing can exacerbate this phenomenon because of increased effect on the baroreceptors.

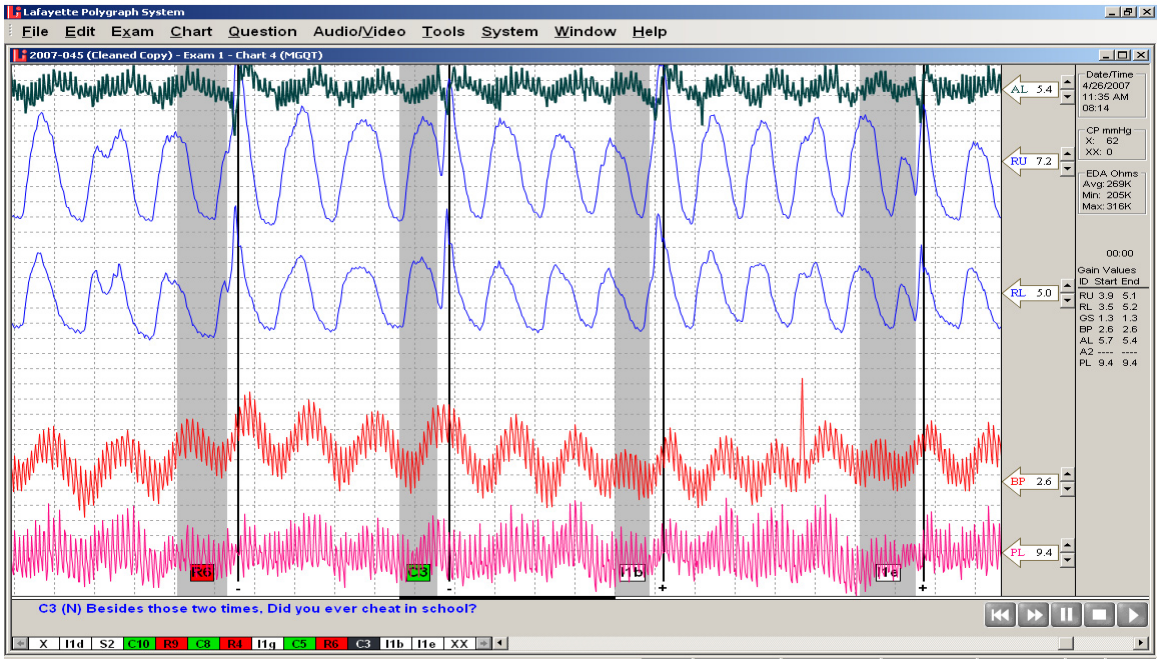

Figure 1. The respiratory effect on the cardiovascular channel (the second waveform from the bottom) can be observed. For the purpose of this figure, the respiration channel sensitivities were adjusted to match the settings of those in Figure 2 for comparison. The rise in relative blood pressure follows behind the inspiration cycle. Also, a decrease in finger blood pulse amplitude (bottom waveform) can be seen just prior to the rise in the blood pressure. Note: The EDA channel has been hidden to allow a more clear view of the data of interest.

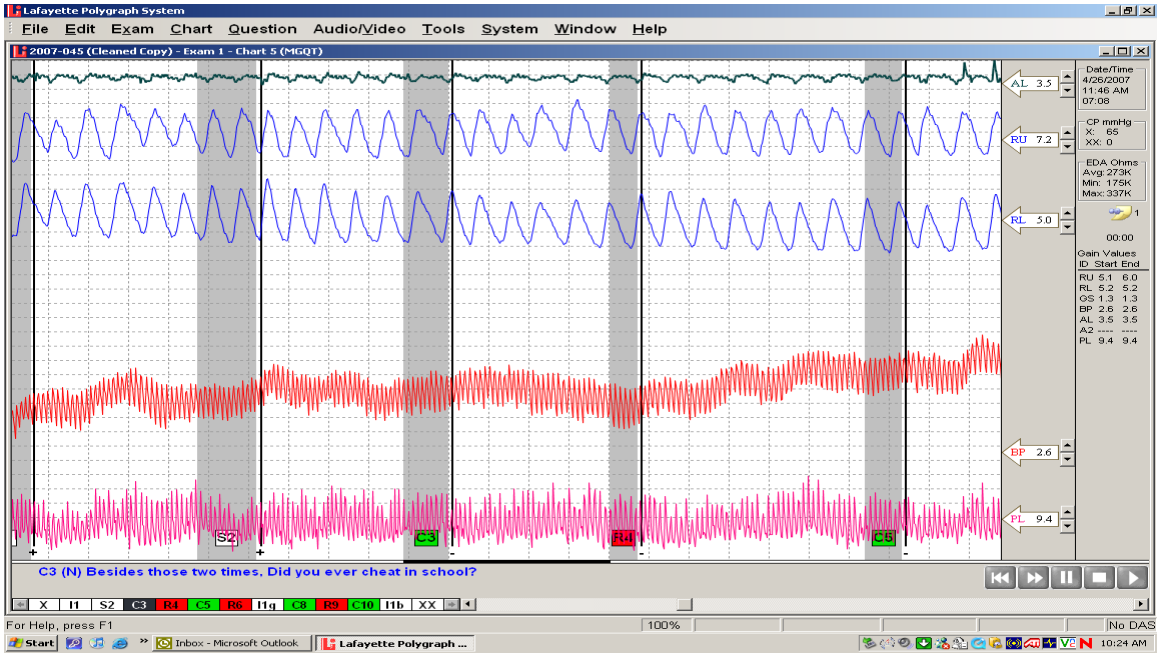

Figure 2. The same examinee during a Silent Answer Test administered shortly after the waveforms shown in Figure 1 were collected. The components are in the same location and the examinee is in the same body position. The respiratory effect on the cardiovascular channel has been reduced significantly. Note the difference in the respiration rate and depth. The respiration channel sensitivities are the same for Figures 1 & 2. Note: The EDA channel has been hidden to allow a more clear view of the data of interest.

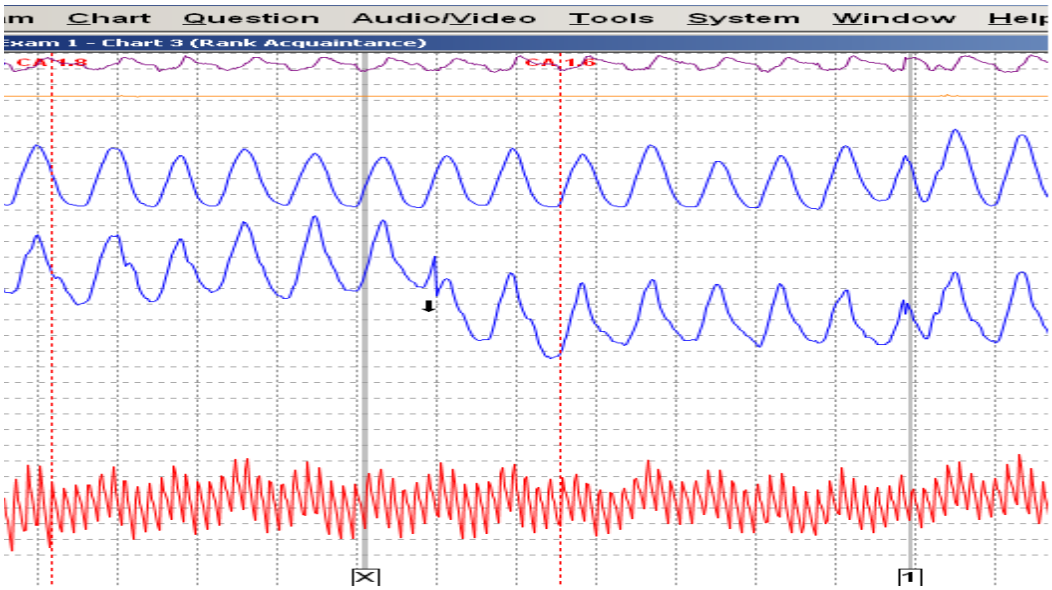

Figure 3. Data collected while purposely placing the blood pressure cuff in contact with the subject's upper body. This allowed the movement associated with breathing to be imparted onto the blood pressure cuff. Note the matching (in time) of respiration and blood pressure waveform peaks.

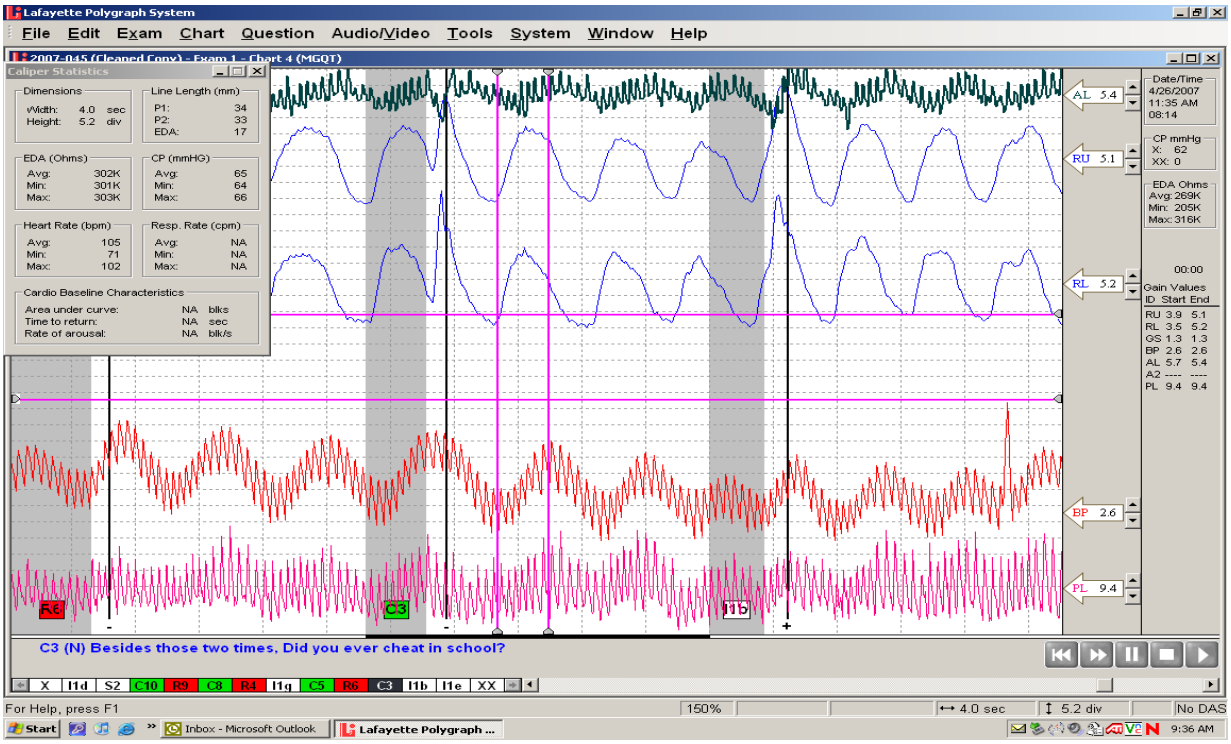

Figure 4. Measurement of the heart rate during inspiration. RSA can be observed in the measurements taken using the calipers. During the 4.0 second time period of inspiration, the average heart rate value (expressed in beats per minute) was 105 BPM.

Additionally, deep breathing results in negative pressure in the venae cavae resulting in increased blood flow. Increased blood flow results in a larger "pre load" (the amount of blood returning to the right side of the heart) or end diastolic volume which leads to increased blood pressure. RSA has been shown to be directly affected by consciously controlled deep respiration. Increased RSA results in increased heart rate during the inspiration cycle. Figure 4 provides examples of RSA measured using a modern polygraph instrument (Lafayette Instrument Company, Lafayette, In.). Using the calipers provided in the software and fixing them at four seconds, we were able to measure the heart rate during inspiration and expiration. Note the heart rate is greater during inspiration than it was during expiration. Increased heart rate results in increased cardiac output which, in turn, results in increased blood pressure. When an examinee engages in a deliberate pattern of deep and slow breathing we can expect to see a cyclic waveform in the cardiovascular channel. The synchronous rise and fall of the relative blood pressure we may observe is quite possibly a result of any combination of these physiological factors discussed earlier.

The cause of the respiratory/heart rate interaction is the interconnection between the cardiac and respiratory centers in the medulla. Even at rest, there is an increase in heart rate during inspiration and a decrease during expiration (Geddes, 1998). With slow, deep breathing these events are more prominent in the blood pressure record. During polygraph examinations in which the examinee breathing is eupneic, the cardiovascular channel often shows a slight undulation that follows normal breathing. Deep, slow breathing merely increases the variation in rate and pressure.

Conclusion

It is not our intention to suggest that vagal tone does not play a part in the undulating blood pressure wave form. It is suggested, however, the scientifically baseless term "vagus roll" be replaced by a more universally accepted and understood expression such as RBPF to describe the undulating cardiograph waveform.

Polygraph professionals should not use terminology that is in conflict with conventional scientific terminology and understandings. We should invite outside scientists to join us in our efforts to define and understand human physiologic responses in polygraphy. Collaboration with outside experts from sister disciplines can help our field avoid "jargonization" of concepts which have established terms in mainstream science.

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