~The Valsalva Wave~

The Changing Landscape of Heart Rate Variability Biofeedback

Stephen Elliott – President & Life Scientist, COHERENCE

Dee Edmonson, RN, BCIAC-EEG, Neurologics
What Is The “Valsalva Wave”?

The Valsalva Wave is the “breathing induced” component of circulation.

- thoracic pump
- heart beat
- vascular action
The question behind the effort (circa 2004):

What is heart rate variability and how does it relate to blood flow and pressure?
There is an arterial phenomenon known as the “respiratory arterial pressure wave” which rises with exhalation and falls with inhalation.

There is also a recognized but little understood venous phenomenon known as the “venous wave”.

In biofeedback circles, there is also a nascent discussion of “blood volume”.

However, these phenomena have not been seen as being “connected”.

“Valsalva Wave” is a name I’ve proposed for the wholistic arterial and venous phenomena that occurs as a function of respiration.
About Stephen:

Stephen Elliott is the founder and president of COHERENCE LLC, Allen, TX. He is a long-term student/teacher of Eastern yogic and martial arts and an avid life sciences researcher. Stephen is an avid inventor with ~40 patents issued or pending in life sciences and telecommunications systems. Stephen is the developer of Coherent Breathing® and the theorist and proponent of the “respiratory arterial pressure wave” theory of heart rate variability and cardiopulmonary resonance.

About Dee:

Integrative neurotherapist Dee Edmonson, RN, BCIAC-EEG, is the founder and director of Neurologics, Plano, Texas, specializing in the treatment of traumatic brain injury, attentional disorders, addiction, and stress/anxiety, with special emphasis on restoration of autonomic balance via breathing. Dee has pioneered the clinical application of Coherent Breathing®.

Dee also directs neurotherapy services Plano Neurotherapy, Plano, Texas.

Stephen and Dee are the authors of *The New Science of Breath* (2005) and *Coherent Breathing - The Definitive Method* (2008).
Please note that the information presented here is correct to the best of our knowledge and consistent with the “state of this art”.

Please consider it a work in progress.
Agenda

- The heart beat & heart rate variability (HRV)
- The Thoracic Pump
- Autonomic nervous system governance of blood flow and pressure
- Valsalva Wave – The Biometric
- Clinical experience and client observations
- Study: HRV Amplitude vs. blood pressure (if we have time)
Part I
~The Heart Beat~
The “beat” itself is generated by the Sinoatrial Node.
Heart Rate Variability:
“Variation in heart rate for any reason.”

Breathing Induced Heart Rate Variability:
“Variation in heart rate as a consequence of respiration.”

We also know it as “Respiratory Sinus Arrhythmia”. 
Heart Rate Variability: “Variation in heart rate for any reason”.

What are some of the reasons that the heart rate varies?
Heart Rate Variability

- “Breathing induced heart rate variability” is variation in heart rate as a consequence of respiration.

- It is the same as the phenomenon that goes by the name “Respiratory Sinus Arrhythmia” or “RSA”.

- RSA refers to the “sinusoidal” variation in heart rate that tends to occur with breathing.
Heart rate *tends* to increase during inhalation and decrease during exhalation.
The contemporary understanding is that the heart, as well as other body functions are managed via the complex interplay between sympathetic and parasympathetic systems.

- Sympathetic action causes the heart beat rate to increase.

- Parasympathetic action ("vagal braking") causes the heart beat rate to decrease.
Vagus nerve endings secrete acetylcholine activating cholinergic receptors, inhibiting cardiac activity.

Sympathetic nerve endings secrete nor-epinephrine activating adrenergic receptors, stimulating cardiac activity.

In the absence of ANS interaction, the heart rate is constant.

It is the interplay between sympathetic and parasympathetic systems that results in breathing induced HRV (RSA).

If the vagus nerve is sectioned, breathing induced HRV stops and the heart beats at a constant rate only moderated by sympathetic action.
When we breathe synchronously, RSA becomes synchronous, heart rate phase locking with respiration.
The contemporary understanding is that “RSA” is the autonomic response to:

- Baroreceptor action in response to changes in blood flow and pressure
- Mechanoreceptors of the chest (Brown, Gerbarg, Muskin, 2009)

Whatever the stimulus, RSA ends if the vagus nerve is sectioned.
If the ANS is responding to changes in blood flow and pressure, then, flow and pressure must be changing with respiration...

And there is some understanding that it does, but it’s sketchy.
Heart rate responding to changes in blood pressure during a Valsalva maneuver. Baroreceptor action is thought to be primary mechanism behind the response.
Valsalva Wave of the Valsalva Maneuver

Blood volume measured at the tip of the thumb during a Valsalva maneuver. This demonstrates volume as opposed to pressure.

Hold nose and blow

Blood volume in thumb falls

Momentary rise above normal.

Blood volume in thumb rises

Blood volume returns to normal level.

Inhale and hold for a few seconds.

Average or “DC” blood volume
Blood Volume of A Yawn

Blood volume at quiescent level.

Yawn starts

Average blood volume.

Yawn ends.
The Valsalva Maneuver

- Today, the Valsalva Maneuver is frequently used in cardiology to assess autonomic and cardiac responsiveness.

- It is a means of "forcing" blood pressure to both increase and decrease such that the response can be observed.

- The general response that is anticipated is that heart beat increases as blood pressure falls and heart beat decreases as blood pressure rises....consistent with RSA.
Let’s explore *why* heart beat rate changes with respiration....
~The Thoracic Pump~
The Thoracic Cavity

A sealed chamber bounded on 3 sides by the rib cage and the diaphragm at the bottom.

Below the diaphragm is the abdominal cavity.
The heart and lungs reside inside.
Pressure in the thoracic cavity varies with diaphragm position which can vary by up to 10 cm.
Boyle’s Law: Absolute pressure and volume of a gas are inversely proportional:

- As volume increases, pressure decreases
- As volume decreases, pressure increases
### A 10X Relationship?

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Vagus nerve endings secrete acetylcholine activating cholinergic receptors, inhibiting cardiac activity yet stimulating gastrointestinal activity.

Sympathetic nerve endings secrete noradrenaline activating adrenergic receptors, stimulating cardiac activity; yet inhibiting gastrointestinal activity.

Upward movement of the diaphragm emphasizes sympathetic function.

Downward movement of the diaphragm emphasizes parasympathetic function.
The diaphragm is a key determinant of both thoracic and abdominal function and status.
The Thoracic Cavity

Pulmonary circulation holds \(~450\text{ml}\) of blood at nominal atmospheric pressure. (Neutral diaphragm position.)

However it can hold as much as \(~900\text{ml}\) and as little as \(~200\text{ml}\).

The pulmonary circulation has a compliance equal to that of the entire arterial tree.

How much it holds is a function of thoracic pressure.

Thoracic pressure is a function of diaphragm position.

Pulmonary capillary bed

Vena Cava

Aorta

anatomy is simplified for purposes of illustration
Blood Flow

anatomy is simplified for purposes of illustration

So, if we observe blood flow at the Vena Cava during respiration what will we see?

And, if we observe blood flow at the Aorta during respiration what will we see?
And Heart Rate?

And heart rate...
During inhalation?
During exhalation?
Why Does Heart Rate Change?

Why does heart rate change during respiration?

- The simple answers....

1. When this much blood (the extreme case) flows into the aorta rapidly, if heart rate did not decrease, blood pressure would rise too much.

2. When the lungs are storing this much blood, if heart rate did not increase, blood pressure would decrease too much.
This supports the theory that “breathing induced HRV” is an outcome of autonomic nervous system regulation of blood flow and pressure.

[Again, this is consistent with what we know about Respiratory Sinus Arrhythmia.]

If this is so, we can expect to see that changes in blood flow and pressure precede changes in heart rate....

And if we look, this is what we see...
We see that changes in the blood wave lead changes in heart rate (at resonance).
~ANS Governance of Blood Flow & Pressure ~
Copyrighted graphic that requires permission to reprint.

(See Coherent Breathing – The Definitive Method)
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(See *Coherent Breathing – The Definitive Method*)

**Effects of respiration on volumes and pressures**
(reprinted with permission of *The American Journal of Medicine*)
Four Factors

There are 4 major mechanisms that result in circulation:

1) The heart beat
2) Respiration (the thoracic pump)
3) Vascular relaxation and constriction
4) Gravity

The ANS employs them in unison to govern blood flow and pressure.
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The Mechanics

Negative thoracic pressure, increased heart rate, decreased heart output, and arterial constriction facilitate venous blood flow during inhalation. The pulmonary tree fills. The arterial tree empties.

Positive thoracic pressure, decreased heart rate, increased heart output, and arterial expansion facilitate increased blood flow during exhalation. The pulmonary tree empties. The arterial tree fills.
76 BPM
87 BPM
89 BPM (4 beats)
85 BPM
15 RPM
5 RPM
40 beats
56 BPM
96 BPM
56 BPM
56 BPM

Variation in HRV With Breathing Frequency

\[ \Delta 7 \text{ beats (decrease in sympathetic emphasis)} \]

\[ \Delta 29 \text{ beats (decrease in parasympathetic emphasis - 4X the decrease in sympathetic emphasis)} \]

Autonomic Balance
Theoretical RAPWave and HRV at 5 & 15 RPM

- **HRV**
  - 96 BPM
  - 81 mmHg
  - 89 BPM (4 beats)

- **RAPW**
  - 56 BPM
  - 79 mmHg
  - 85 BPM
  - 76 BPM

- 20 mmHg
- 2 mmHg
- 4 beats
- 79 mmHg
- 76 BPM
A View of This Process Measured At Left Thumb

Red = Blood

Cardiac Systole

Cardiac Diastole
Under typical breathing circumstances, the heart beat is the only significant variation. The heart beat is nominally 40mmHg. During deep breathing, the respiratory component may rise and fall by 20mmHg. I believe that at resonance, it may be 30mmHg or more.

The pulse “normally” contributes to about 33% of blood pressure (40mmHg) at the brachial artery. During very shallow breathing, the respiratory component is effectively zero – the remainder (80mmHg) falls to the vascular system and gravity.
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The End of Part I
Thank You!