# Diaphragmatic, thoracic and abdominal breathing for sport: the effect on vagal activity, a case report

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***Abstract*—Breathing is important for any sport, particularly in aquatic sports. The autonomous response to diving is characterized by bradycardia mediated by the vagus nerve. Specific breathing techniques, particularly those involving deep and slow respiration tasks, could phasically and tonically stimulate the vagus nerve. This case report aimed to measure the effects of different breathing techniques (thoracic, diaphragmatic and spontaneous breathing) on autonomic vagal activity through simultaneous monitoring of ECG and ventilatory and thoraco-abdominal patterns. An elite male apneist was enrolled (50 years old, 186 cm, 80 kg) during spontaneous breathing (SB), diaphragmatic breathing (DB); thoracic breathing (TB) and diaphragmatic breathing combined with transverse abdominis activation (DTB) following a visual and acoustic timer to uniform the respiratory frequency at 6 breaths/minute. Compared to baseline, breathing exercises involving diaphragmatic recruitment (DB and DTB) increased the total power (index of overall autonomic activity and the plasticity of the cardiovascular system) by fourfold, while maintaining the mean heart rate nearly unchanged. However, the end-expiratory abdominal volume reduced during DB and DTB indicating a strong recruitment of the abdominal muscles. These results suggest that slow, deep and abdominal (and not just diaphragmatic) breathing enhanced vagal activity, independently of respiratory rate. We can speculate that a possible vagal stimulation occurs even when the diaphragm is stretched passively by the abdominal muscles. This mechanism would explain the vagal-related beneficial effects during exercise even in the absence of a direct increased diaphragmatic contribution. This hypothesis needs to be confirmed in a wider population.**

**Keywords—diaphragmatic breathing, thoracic breathing, opto-electronic plethysmography, vagus nerve, heart rate**

## I. INTRODUCTION

Breathing plays an important role in sports, particularly in aquatic sports. The most elite apneists can consciously suppress respiratory urges, while the autonomous response to diving is characterized by bradycardia mediated by the vagus nerve and by splenic and peripheral vasoconstriction mediated by the sympathetic nervous system[1]. Indeed, lower resting heart rate and high autonomic vagal activity are associated with superior exercise capacity[2].

Slow, deep and diaphragmatic breathing (DB) is renowned for its ability to enhance athletic performance through various aspects. Firstly, it facilitates the regulation of emotions and anxiety, thereby priming athletes for optimal performance. Notably, DB has been shown to improve executive functions, further augmenting athletes' cognitive abilities during competition[3]. Lastly, DB has been associated with beneficial effects on blood pressure and heart rate, promoting adaptability of the cardiovascular system and enabling it to respond effectively to the demands of physical exertion. [4], [5]. Since, specific breathing techniques, particularly those involving deep and slow respiration tasks, could phasically and tonically stimulate the vagus nerve[6], these results could be mediated by an enhancement of vagal activity induced by DB. However, there remains a gap in understanding the precise physiological pathways activated by DB.

Combining simultaneous measurements of cardiac and breathing functions might be useful in revealing the mechanism underlying the reported benefits of DB.

Thus, this case report aimed to measure the effects of different breathing techniques (thoracic, diaphragmatic and spontaneous breathing) on autonomic vagal activity in an elite apneist. A novel aspect of the experimental setting is the simultaneous monitoring of ECG and ventilatory and thoraco-abdominal patterns. ECG traces provide valuable insights into heart rate variability, a proxy of cardiovascular autonomic control. Meanwhile, the analysis of ventilatory and thoraco-abdominal patterns allows for the assessment of the quality of the performed breathing tasks.

## II. MATERIALS AND METHODS

### A. Subject

An elite male apneist was enrolled (50 years old, 186 cm, 80 kg). The participant had no chronic pathologies and was not under acute or chronic pharmacological treatment. He had stable sinus rhythm on ECG, no history of surgery in the last 12 months, and no recent infectious events (< 3 months). He gave his informed consent to the experimental session.

### B. The Equivital multi-parameter telemetric device

Continuous ECG was recorded through the Equivital multi-parameter telemetric device (Equivital EQ02, AD Instruments, Sydney, Australia). The device is a chest-worn sensor belt embedding textile-based electrodes. In addition to two channels of ECG, it also records breathing rate, body position, and movements through actigraphy. LabChart software was used to record all the signals.

### C. Opto-Electronic Plethysmography

Opto-electronic plethysmography (OEP System; BTS, Milan, Italy) is a motion analysis system based on infrared TV cameras. The output of a motion analysis system is the 3D coordinates of passive reflective markers [9]. We have used 89 markers placed on the chest wall according to precise anatomical reference points (Figure 1).

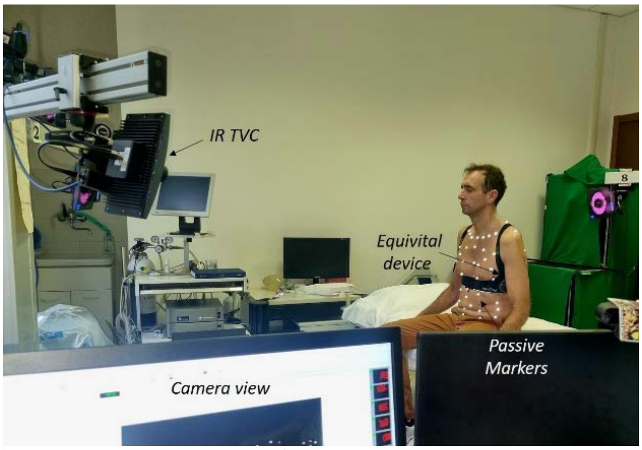

*Figure 1 Experimental set-up of the subject wearing the passive markers on his thorax and the belt for ECG monitoring. The subject was seated on the bed without back support with his arms relaxed aside. Photo published with the permission of the subject.*

The markers reflect the light emitted by the diodes and the system computes their centroids computed. The combination of Gauss's theorem and dedicated geometrical models provides the volumes enclosed by the markers. The Gauss's theorem states that the surface integral of a vector field over a closed surface equals the volume integral of the divergence over the region enclosed by the surface. The geometrical models define surface triangulation of the centroids, therefore identifying the closed surface. The accuracy of OEP in chest wall volume assessment was previously established[10].

Markers are put according to anatomical points, with the lower costal margin representing an important reference. The volumes of the pulmonary ribcage and the abdomen are accurately determined with OEP. The former is the volume enclosed between the clavicles and the xiphoidal process and it is under the action of ribcage muscles. The latter is the volume enclosed between the lower costal margin and the iliac crests. It is under the action of the diaphragm during inspiration and abdominal muscles during active expiration.

### D. Protocol of acquisition

Once the subject became familiar with the experimental set-up, a baseline of resting quiet breathing was acquired for 5 minutes and two slow vital capacities were asked at the end. Four breathing strategies were then performed:

- 90 seconds of quiet breathing followed by 5 minutes of spontaneous deep breathing (SB);
- 90 seconds of quiet breathing followed by 5 minutes of diaphragmatic breathing (DB);
- 90 seconds of quiet breathing followed by 5 minutes of thoracic breathing (TB);
- 90 seconds of quiet breathing followed by 5 minutes of diaphragmatic breathing combined with transverse abdominis activation (DTB);

Each task (SB, DB, TB and DTB) was performed following a visual and acoustic timer to uniform the respiratory frequency at 6 breaths/minute (5 seconds of inspiration and 5 seconds of expiration). All 4 respiratory tasks were followed by a 10-minute recovery period to wash out any effects of the breathing exercise. ECG was continuously monitored during both breathing tasks and recovery periods, and OEP acquisition was performed until the whole task.

The study was approved by the local Ethics Committee Milano Area 2 (approval code: 894_2022). Written informed consent for investigation was given by the athlete, and the study was conducted following the declaration of Helsinki.

### E. Cardiovascular autonomic control analyses

R-R time series segments were extracted from the ECG signal during: i) baseline; ii) SB iii) DB; iv) TB; v) DTB. For each segment, the mean HR and the total power of the heart rate variability spectrum was computed using a dedicated software (Heart Scope II, AMPS, ITA). The total power reflects overall autonomic activity and the plasticity of the cardiovascular system[7]. Moreover, the strength of the cardiorespiratory coupling, mediated by the vagus nerve, was evaluated during each respiratory exercise through the assessment of the respiratory sinus arrhythmia (RSA) [8]. This is a phenomenon characterized by heart rate (HR) fluctuations that are in phase with inhalation and exhalation and is mediated by a cardiorespiratory coupling pathway. Typically, HR increases during inspiration and decreases during expiration. For each respiratory task, the average value of the difference between the maximum HR rate during inspiration and minimum HR during expiration within the same respiratory cycle was derived (∑[HR max – HR min]/N° of respiratory cycles per task) [8].

### F. Respiratory pattern analyses

The breaths during the last 2 minutes of tasks were selected and the ventilatory pattern was calculated for each breath. The tidal volume (i.e.: the amplitude of the breath) was calculated on the chest wall, the pulmonary ribcage and the abdomen. The compartmental volumes were expressed as percentage values of the tidal volume. The duration of each breath was also computed and the respiratory rate was derived. Minute ventilation was calculated as the product of tidal volume and respiratory rate.

## III. RESULTS

### A. Baseline

At rest, before the breathing tasks, the participant presented a mean HR of 67 bpm and a total power of 1032 ms$^2$ (Figure 2). The tidal volume was 0.564 ± 0.08 L; the breathing frequency was 16.0 ± 1.7 breaths/min; the minute ventilation was 8.9 ± 1.1 L/min; the pulmonary ribcage contribution to tidal volume was 54.7 ± 1.8%, the abdominal contribution to tidal volume was 33.4 ± 1.9%.

The slow vital capacity was 5.28 L and the inspiratory capacity was 3.0 L.

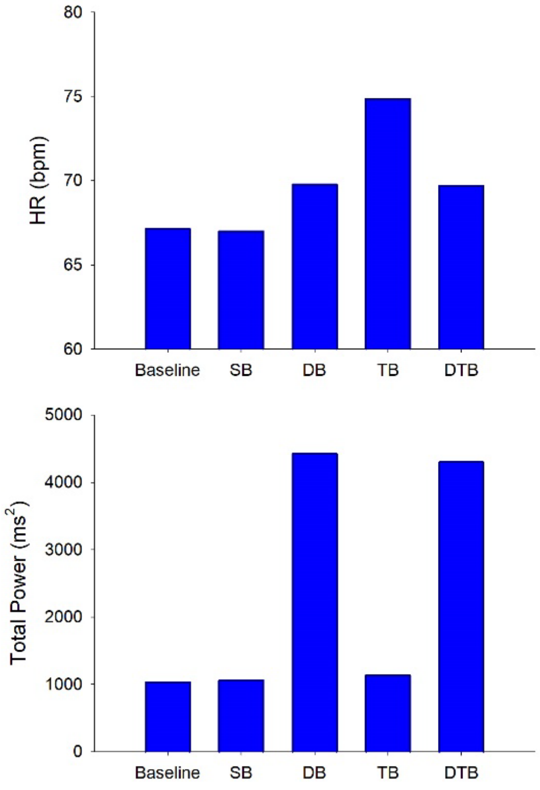

*Figure 2. Mean values of HR (top panel) and total power (bottom panel) during baseline, spontaneous deep breathing (SB), diaphragmatic breathing (DB), thoracic breathing (TB) and diaphragmatic breathing combined with transverse abdominis activation (DTB)*

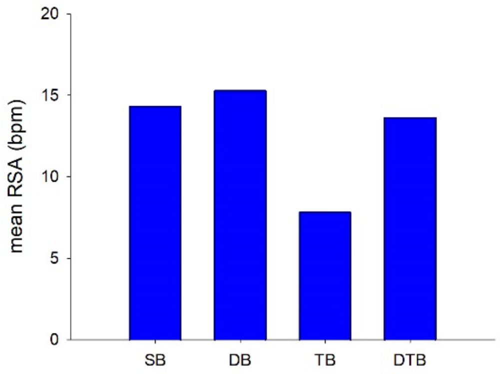

*Figure 3. Mean RSA magnitude during breathing tasks: spontaneous deep breathing (SB), diaphragmatic breathing (DB), thoracic breathing (TB) and diaphragmatic breathing combined with transverse abdominis activation (DTB)*

### B. Task 1: spontaneous deep breathing

During the SB task, the mean HR and the total power did not change with respect to the baseline condition. The mean RSA magnitude was 14.3 bpm (Figure 3).

The tidal volume was 1.22 ± 1.6 L; the breathing frequency was 5.8 ± 0.5 breaths/min; the minute ventilation was 7.2 ± 1.1 L/min; the pulmonary ribcage contribution to tidal volume was 54.9 ± 2.3%, the abdominal contribution to tidal volume was 31.6 ± 3.2% (Figure 4).

The subject maintained the thoraco-abdominal distribution of baseline condition, he doubled the tidal volume to normoventilate while following the imposed breathing frequency.

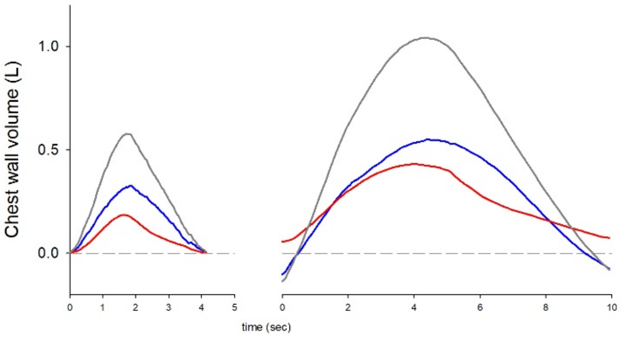

*Figure 4. Representative traces of chest wall (grey), pulmonary ribcage (blue), and abdominal (red) volume variations over time during spontaneous quiet breathing before (left) and during (right) spontaneous breathing following a breathing visual and acoustic timer (5 seconds inspiration and 5 seconds expiration)*

### C. Task 2: diaphragmatic breathing

When the subject was asked to perform the diaphragmatic breathing task, his mean HR increased by approximalty 3 bpm, while the total power quadrupled compared to baseline, reaching 4422 ms$^2$ (Figure 2). The mean RSA magnitude was 15.3 bpm (Figure 3). His tidal volume was 1.24 ± 1.4 L; the breathing frequency was 5.8 ± 0.4 breaths/min; the minute ventilation was 7.2 ± 0.7 L/min; the pulmonary ribcage contribution to tidal volume was 38.6 ± 4.9%, the abdominal contribution to tidal volume was 54.2 ± 5.4% (Figure 5).

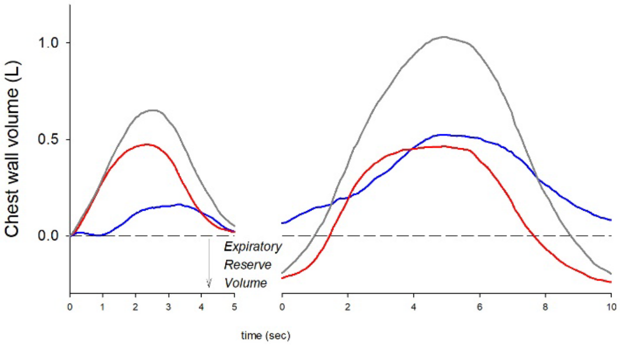

*Figure 5. Representative traces of chest wall (grey), pulmonary ribcage (blue), and abdominal (red) volume variations over time during spontaneous quiet breathing before (left) and (right) diaphragmatic breathing following*

*a breathing visual and acoustic timer (5 seconds inspiration and 5 seconds expiration)*

The subject reversed the thoraco-abdominal distribution of the baseline condition, by shifting breathing towards the abdomen. The operational volume of the abdomen was below the baseline condition, indicating the recruitment of the expiratory reserve volume and therefore contraction of the abdominal muscles (Figure 3). His tidal volume and minute ventilation were similar to the spontaneous breathing task.

### D. *Task 3: thoracic breathing*

The mean HR increased by approximatly 8 bpm and the and the total power was almost unchanged compared to baseline (1132 ms$^2$, Figure 2). The mean RSA magnitude was 7.8 bpm (Figure 3). The tidal volume was 3.3 ± 0.2 L; the breathing frequency was 5.8 ± 0.2 breaths/min; the minute ventilation was 19.3 ± 1.2 L/min; the pulmonary ribcage contribution to tidal volume was 47.7 ± 2.8%, the abdominal contribution to tidal volume was 30.6 ± 3.7% (Figure 4).

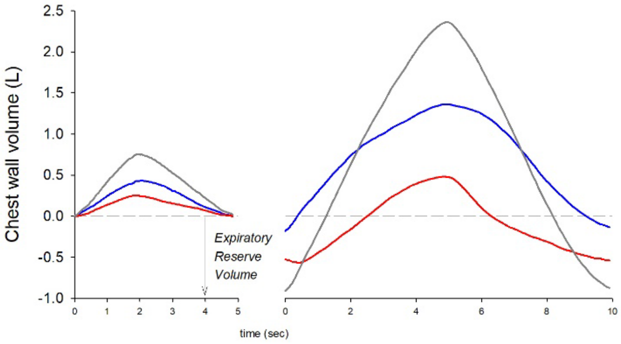

*Figure 6. Representative traces of chest wall (grey), pulmonary ribcage (blue), and abdominal (red) volume variations over time during spontaneous quiet breathing before (left) and during (right) thoracic breathing following a breathing visual and acoustic timer (5 seconds inspiration and 5 seconds expiration)*

The subject did not properly accomplish the requested task, as he maintained the thoraco-abdominal distribution of baseline condition, while he was supposed to increase the thoracic contribution. He also recruited the abdominal muscles as indicated by the operational abdominal volume below the baseline condition. Instead, he tremendously augmented the tidal volume (6 times compared to the baseline condition), approaching his inspiratory capacity and resulting in hyperventilation.

### F. *Diaphragmatic breathing combined with transverse abdominis activation*

When the subject was asked to perform the task with diaphragmatic breathing combined with transverse abdominis activation, his mean HR incresed by approximalty 3 bpm compared the baseline and the total power once again more than quadrupled with respect to the baseline, reaching 4298 ms$^2$ (Figure 2). The mean RSA magnitude of 13.6 bpm was comparable to that observed in the SB and DB tasks (Figure 3). His tidal volume was 1.67 ± 0.1 L; the breathing frequency was 5.8 ± 0.2 breaths/min; the minute ventilation was 9.7 ± 0.8 L/min; the pulmonary ribcage contribution to tidal volume was 6.6 ± 6.4%, the abdominal contribution to tidal volume was 84.7 ± 8.0% (Figure 5).

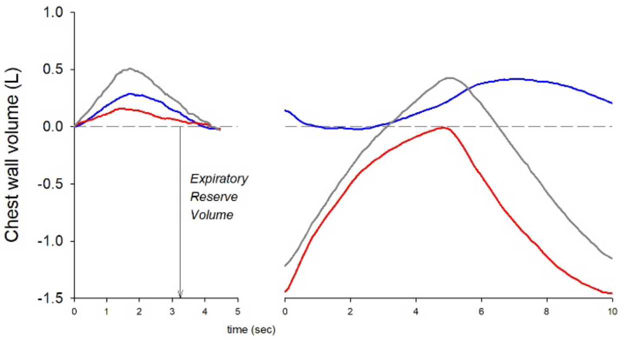

*Figure 7. Representative traces of chest wall (grey), pulmonary ribcage (blue), and abdominal (red) volume variations over time during spontaneous quiet breathing before (left) and during (right) diaphragmatic breathing combined with transverse abdominis activation following a breathing visual and acoustic timer (5 seconds inspiration and 5 seconds expiration). Note a slight inspiratory paradoxical indrawing of the pulmonary ribcage due to the increased abdominal contribution.*

The subject almost entirely shifted his breathing towards the abdomen. The abdominal tidal volume occurred exclusively below the baseline condition, indicating that abdominal breathing was accomplished within the expiratory reserve volume. He properly performed the task, as the transverse abdominis was activated during the whole period. His tidal volume and minute ventilation were slightly higher than the spontaneous and diaphragmatic breathing tasks, but not as high as during the thoracic task.

## DISCUSSION AND CONCLUSION

Compared to baseline, breathing exercises involving diaphragmatic recruitment (DB and DTB) were able to increase the total power by fourfold, while maintaining mean heart rate nearly unchanged. In contrast, this effect on heart rate variability was not observed during spontaneous deep breathing and thoracic breathing, even if the latest was performed suboptimally.

This suggests a potential influence on heart rate variability mediated by diaphragmatic engagement, independently of respiratory rate, which was kept constant at 6 breaths/minute in all exercises. Given that the increase in overall heart rate variability is associated with vagus nerve activity and considering the presence of vagal afferents innervating the diaphragm and coordinating cardiorespiratory coupling, it is plausible that diaphragmatic recruitment stimulated vagal activity, leading to heightened heart rate variability. Such an increase in heart rate variability is considered to be cardioprotective.

However, the end-expiratory abdominal volume strongly reduced in both diaphragmatic exercises that were accomplished by recruiting the expiratory reserve volume. Although no direct measurement of abdominal muslces contraction was assessed, the shifting of the abdominal operational volume indicate a strong recruitment of the abdominal muscles.

It has been observed that independently on the workload, the abdominal muscles contribute since the very beginning of exercise, whereas the diaphragm does not increment its contribution during exercise and it acts as a flow generator. Thus, the contraction of the abdominal muscles during exercise have different implications: 1) stabilize the chest wall, 2) recruit the expiratory reserve volume to meet the increased ventilatory demand and 3) stretch the diaphragm[11].

We can speculate that a possible vagal stimulation occurs even when the diaphragm is stretched passively by the abdominal muscles, as evidenced by the two diaphragmatic tasks. This mechanism would explain the vagal-related beneficial effects during exercise even in absence of a direct increased diaphragmatic contribution.

Notably, this study represents the first attempt to integrate two non-invasive methodologies – the assessment of cardiovascular autonomic control via heart rate variability analysis and the evaluation of ventilatory patterns through opto-electronic plethysmography – to investigate the physiological mechanisms triggered by different breathing techniques. The simultaneous monitoring of cardiac function and thoraco-abdominal volume was crucial to better understand the muscular strategy adopted by the subject and the consequent results on autonomic nervous system activity.

This case report shows that slow, deep and abdominal (and not just diaphragmatic) breathing enhanced the vagal activity. It opens to the hypotesis of a new mechanism of vagal stimulation during exercise mediated by the abdominal muscles. This speculation needs to be confirmed with dedicated study in a wider population.

## ACKNOWLEDGMENT

The authors are deeply thankful to Mark for his enthusiasm in participating in the acquisition while encouraging and supporting the project.

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