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Dynamic model of the influence of gravity, ventilation with positive and negative pressure on ventilation-perfusion ratio in lungs

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ABSTRACT

The concept of gravitational influence on ventilation and blood flow in lungs is well known. Dr. John West presented this concept in 1964. However, this concept does not fully explain some clinical features of pulmonary dysfunction. In addition, recent studies demonstrated a less important role of gravity in changing the regional distribution of blood flow (approximately 25%) than structural features of respiratory tract and blood vessels. We present a theoretical model designed to better understand the effects of different ventilation methods on pulmonary perfusion. The objective of this report is to discuss our theory with specialists in respiratory physiology and respiratory support, as well as to get your opinion. This is very important for us, since our theoretical model is based on a slightly different vision of perfusion and ventilation in lungs. We are particularly interested in your views on the issue of cuirass ventilation and combination of cuirass ventilation with positive pressure ventilation. We found no available literature data on such an approach to describing the relationship between ventilation and perfusion in lungs. We will be grateful for any information and especially your professional opinion. We are ready for cooperation in any form (from theoretical discussion to joint work). We are aware that certain our assumptions require verification. Additional research of cuirass ventilation and development of new types of ventilators are necessary. Perhaps, the combination of cuirass ventilation and positive pressure ventilation opens up great prospects in the treatment of severe pulmonary diseases. We hope that our dynamic model will be useful for understanding the processes of ventilation and perfusion in lungs and, possibly, for predicting the results of any methods of respiratory support.

Keywords: *respiratory physiology, respiratory support, perfusion and ventilation processes in lungs, cuirass ventilation, positive pressure ventilation, severe respiratory failure, Dr. John West's lung zones, dynamic model of ventilation-perfusion relationships in lungs, effects of various methods of respiratory support on ventilation -perfusion ratio, combination of cuirass ventilation with positive pressure ventilation.*

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Introduction

Most physicians are familiar with the concept of gravitational effects on ventilation and blood flow in lungs. West J.B. presented this concept in 1964. He was a young leader of respiratory research group at the medical school in London. Now, he is a distinguished scientist in respiratory physiology [1]. Modern textbooks on anesthesiology and intensive care detail the influence of gravity on regional ventilation and blood flow in lungs regarding vertical gradients, pleural pressure and pulmonary vascular pressure. However, this does not explain some clinical features of pulmonary dysfunction. In addition, recent studies revealed a less important role of gravity in changing the regional distribution of blood flow (approximately 25%) compared to structural features of respiratory tract and blood vessels [2].

Although ventilation-perfusion ratio has a 75% dependence on structural features of respiratory tract and blood vessels, 25% effect of gravity cannot be ignored, especially in patients with severe respiratory dysfunction. As stated above, the concept of pulmonary zones by West J.B. is well known and discussed in almost all textbooks on anesthesiology, intensive care and physiology. In classic version, West J.B. West distinguishes 3 zones in lungs. Four zones are described in some textbooks, but we will focus on the first version **(Fig. 1)**. Lungs are divided into 3 vertical zones depending on the relationship between alveolar pressure (PA), arterial pressure (Pa) and venous pressure (Pv).

We should emphasize several important features of this model before analysis of ventilation and perfusion in each zone. This model theoretically demonstrates ventilation-perfusion relationship in a healthy person in upright position at the end of expiration and during respiratory pause under spontaneous breathing.

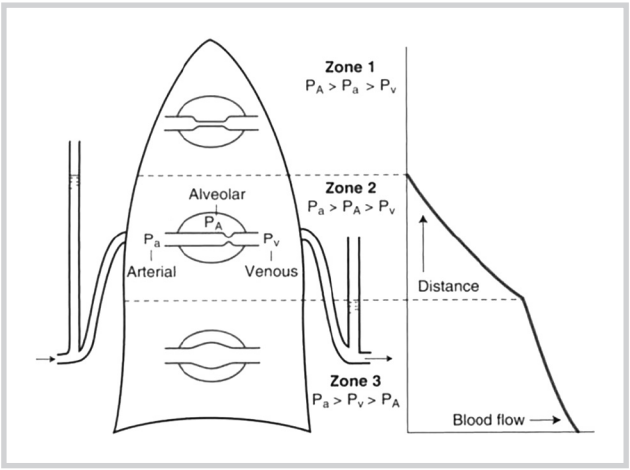

Fig. 1. **Three-zonal model of pulmonary blood flow distribution** [3].

Alveolar end-expiratory pressure is equal to atmospheric pressure (760±1.5 mm Hg in different parts of lungs). Gravity causes blood pressure gradient between the upper and lower parts of the lungs within 20 mm Hg in vertical position, mean pulmonary venous pressure ~5 mm Hg. Venous pressure decreases to –5 mm Hg at the apexes of the lungs and rises to 15 mm Hg at the base. Pulmonary artery pressure is usually 25—10 mm Hg (mean 15 mm Hg). Regional blood pressure is usually 5 mm Hg at the apexes of the lungs and 25 mm Hg in lower parts [2].

There is almost no zone 1 in lungs of healthy persons with normal blood pressure **(Fig. 2)**. This zone usually occurs under positive pressure ventilation or when blood pressure decreases due to bleeding or other causes. Under these conditions, pulmonary capillaries collapse completely under alveolar pressure, and this area becomes alveolar dead space. Zone 2 is a pulsed blood flow zone. In this area, arterial pressure increases until it exceeds alveolar pressure and capillary blood flow appears. Blood flow disappears as soon as pressure in arteries and capillaries falls below alveolar pressure. In this zone, venous pressure is always lower than alveolar pressure.

In zone 3, pressure in pulmonary arteries and capillaries always exceeds alveolar pressure. Therefore, blood flow is continuous throughout the cardiac cycle. In this zone, venous pressure is always higher than alveolar pressure.

We present distribution of blood flow in the lung at the end of normal exhalation and during respiratory pause under spontaneous breathing, i.e. when alveolar pressure is equal to atmospheric pressure [3]. Let us imagine in theory how this model might change if we consider pulmonary blood flow in all phases of spontaneous breathing cycle.

Zone 1 (no blood flow in all phases of respiratory cycle). Zone 1 coincides with classic zone 1 described by West J.B. There is no blood flow in this zone in all phases of respiratory cycle **(Fig. 1)**.

Normally, this zone does not exist. Indeed, blood pressure at the apex of the lung is +5 mm Hg and higher than alveolar pressure by 3.5 mm Hg even in the exhalation phase. However, this zone will appear as soon as blood pressure at the apex of the lung falls below alveolar pressure during all phases of respiratory cycle (due to blood pressure decrease or alveolar pressure increase).

Zone 2 (blood flow only in the inhalation phase). Zone 2 is included in a pulsed blood flow zone by West J.B. However, we believe there are several zones of pulsed blood flow **(Fig. 3)**.

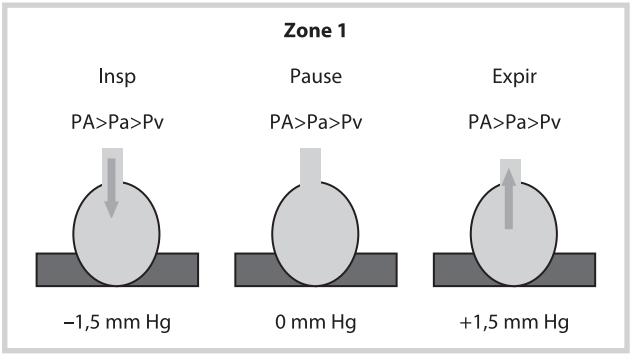

Fig. 2. **Zone 1.**

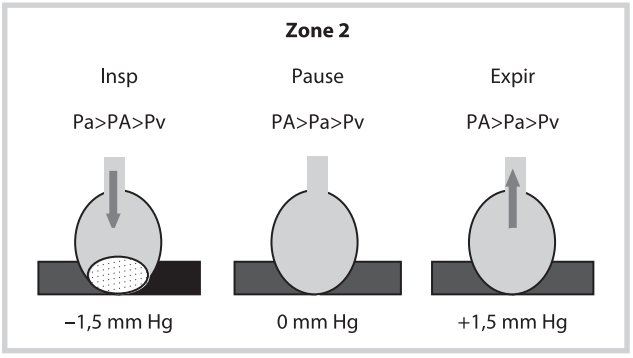

Fig. 3. **Zone 2.**

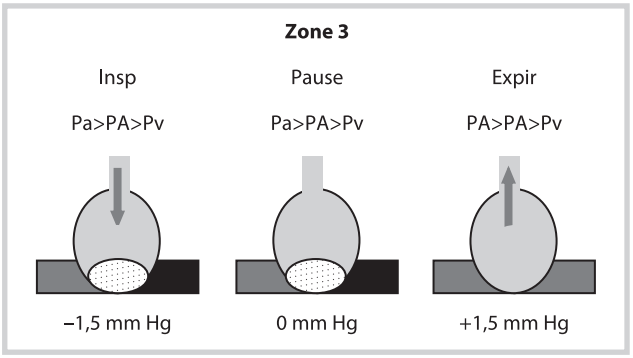

Fig. 4. **Zone 3.**

In zone 2, blood pressure is higher than alveolar pressure only in inspiratory phase, so blood flow is only possible in this phase. There will be no blood flow in this area during respiratory pause and exhalation.

Zone 3 (blood flow in the inhalation phase and respiratory pause). Zone 3 is also included in a pulsed blood flow zone by West J.B. Blood flow in this zone occurs during inhalation phase and respiratory pause, i.e. under negative or zero alveolar pressure **(Fig. 4)**.

Zone 4 (blood flow in all phases of respiratory cycle). Zone 4 is also included in a pulsed blood flow zone by West J.B. Blood flow in this zone occurs in all phases of respiratory cycle, since blood pressure exceeds alveolar pressure in exhalation phase **(Fig. 5)**. However, venous pressure is still lower than alveolar pressure in this zone. Therefore, there is no capillary plethora.

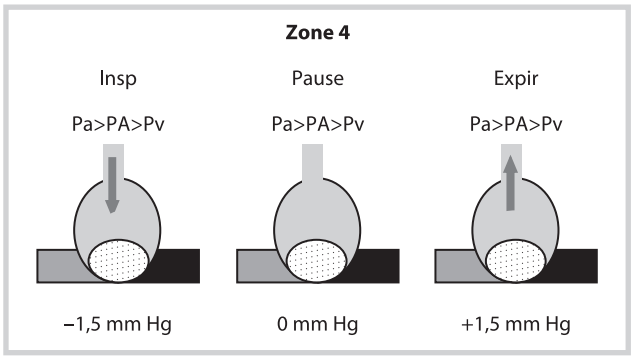

Fig. 5. **Zone 4.**

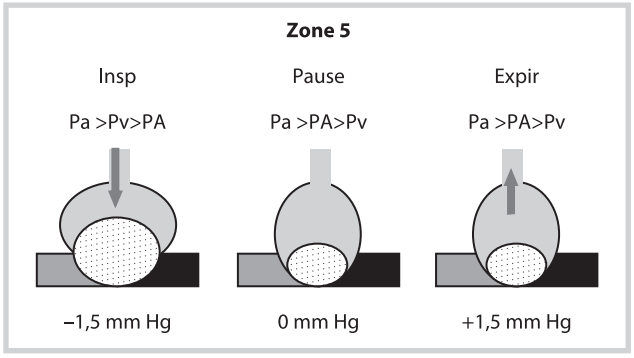

Fig. 6. **Zone 5.**

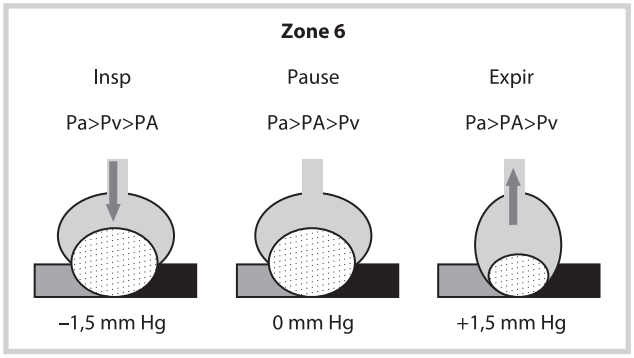

Fig. 7. **Zone 6.**

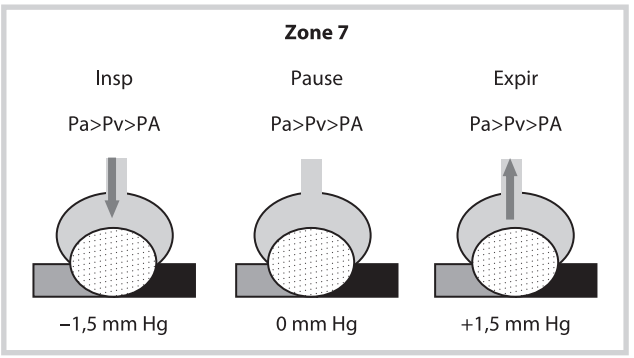

Fig. 8. **Zone 7.**

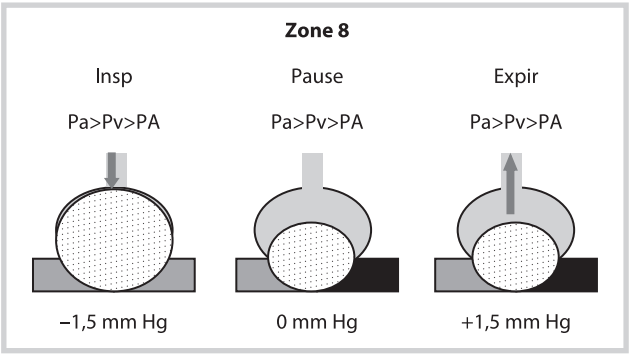

Fig. 9. **Zone 8.**

Zone 5 (blood flow in all phases of respiratory cycle with increased capillary filling in the inhalation phase). Zone 5 is in zone 3 by West J.B. However, venous pressure exceeds alveolar pressure only in inhalation phase **(Fig. 6)**. Capillary bed capacity increases at this moment and decreases again during respiratory pause and exhalation phase.

Zone 6 (blood flow in all phases of respiratory cycle with increased capillary filling in the inhalation phase and respiratory pause). Zone 6 is also located in zone 3 by West J.B., but here venous pressure exceeds alveolar pressure in inhalation phase and respiratory pause **(Fig. 7)**. Capillary bed capacity increases at this moment and decreases again during exhalation phase.

Zone 7 (blood flow with capillary plethora in all phases of respiratory cycle). Zone 7 completely coincides with zone 3 by West J.B. Venous pressure exceeds alveolar pressure in all phases of respiratory cycle. Throughput of capillary bed is always maximum in this zone **(Fig. 8)**.

Zone 8 (shunting in inspiratory phase). Capillary bed capacity is constantly maximum in zone 8, but arterial and venous pressure significantly exceed alveolar pressure in inhalation phase **(Fig. 9)**. Consequently, 100% shunting occurs in inspiratory phase.

Zone 9 (shunting in inhalation phase and respiratory pause). Capillary bed capacity is constantly maximum in zone 9, but arterial and venous pressure significantly exceed alveolar pressure in inhalation phase and respiratory pause **(Fig. 10)**. Therefore, 100% shunting develops in inhalation phase and respiratory pause.

Zone 10 (100% shunting in all phases of respiratory cycle). Zone 10 is a zone of 100% shunting in all phases of respiratory cycle **(Fig. 11)**.

Thus, if we consider the effect of gravity on ventilation / perfusion ratio taking into account phases of respiratory cycle, we get 10 zones. For better understanding, we placed them side by side to visualize transition of one zone to another and logic of our reasoning **(Fig. 12)**. Dynamic model describes ventilation-perfusion relationship from physiological dead space to 100% shunting. If pulmonary zones are located as shown in **Fig. 12**, this really reflects the effect of gravity on pulmonary circulation. However, 75% of pulmonary blood flow distribution depends on structural features of airways and blood vessels.

In our opinion, pulmonary blood flow distribution, independent of gravity, can also be described using the dynamic model. However, pulmonary blood flow zones will be chaotically arranged. The model shows how ventilation / perfusion ratio depends on arterial, venous and alveolar pressure. In principle,

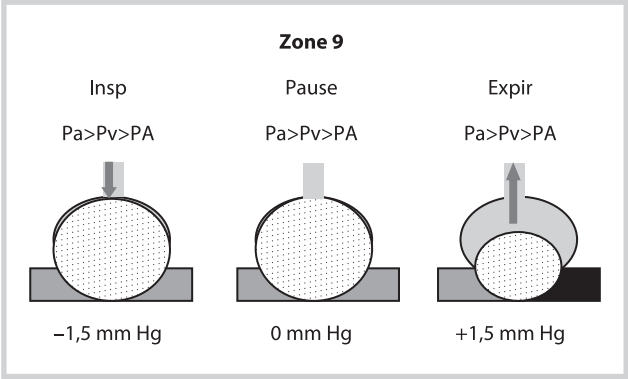

Fig. 10. **Zone 9.**

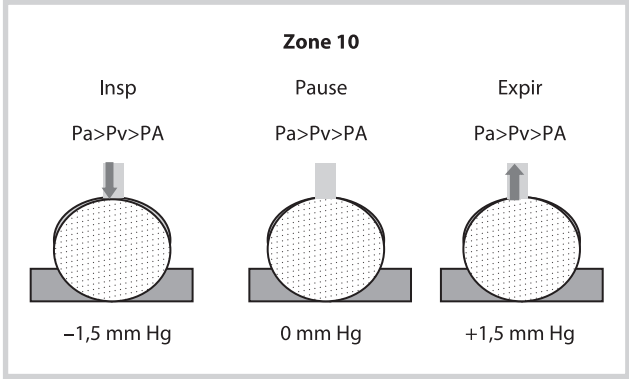

Fig. 11. **Zone 10.**



Fig. 12. **Effect of gravity on ventilation-perfusion ratio.**

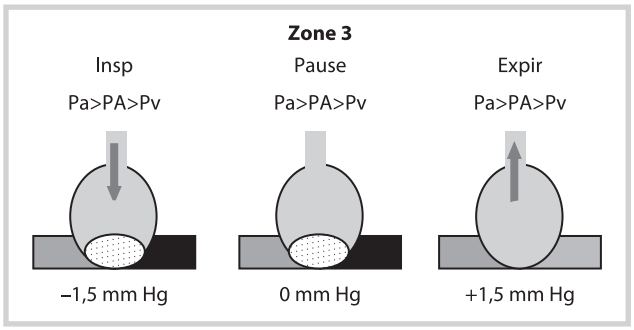

Fig. 13. **Zone 3.**

this model describes all possible options for ventilation and perfusion relationships. Final ventilation / perfusion ratio will depend on the number of different zones in lungs and different ventilation-perfusion parameters. Next, we will consider the effect of various methods of respiratory support on ventilation / perfusion ratio considering dynamic model.

Effect of positive end-expiratory pressure on ventilation / perfusion ratio in lungs under spontaneous breathing

Any positive end-expiratory pressure (PEEP) during spontaneous breathing (we do not consider indications, only the ef-



Fig. 14. **Effect of PEEP on ventilation-perfusion ratio in lungs under spontaneous breathing.**

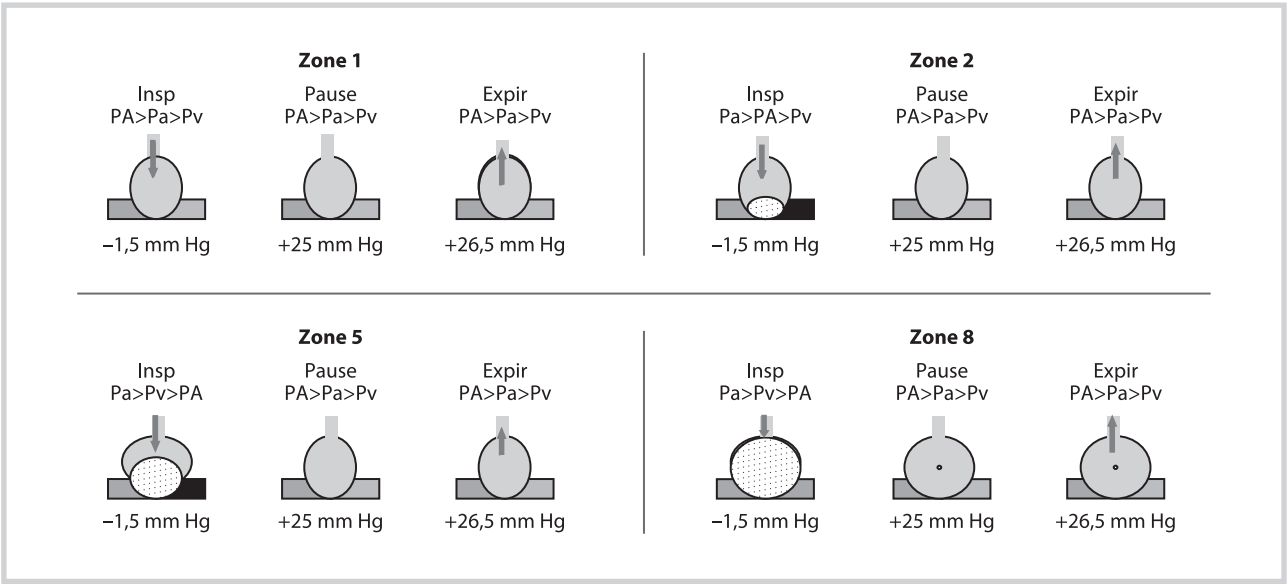

Fig. 15. **Lung zones at PEEP +25 mm Hg and spontaneous breathing.**

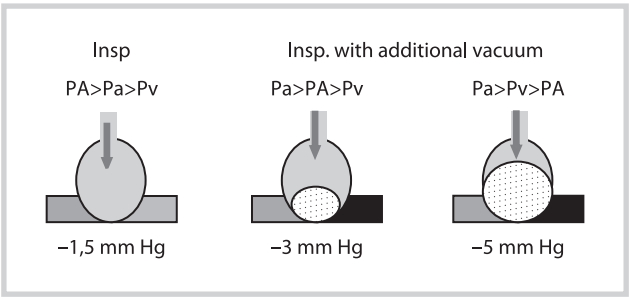

Fig. 16. **Effect of inspiratory vacuum on perfusion.**

fect on perfusion) affects perfusion in zones 3—10 **(Fig. 13)**. This is because PEEP under spontaneous breathing does not affect perfusion during inspiratory phase, and perfusion during respiratory pause begins only from zone 3.

PEEP increment will lead to progressive deterioration of perfusion during expiratory phase and respiratory pause in zones 3—10. First, perfusion decreases in expiratory phase and then in respiratory pause.

Let us consider the effect of PEEP (+3 mm Hg) on ventilation / perfusion ratio under spontaneous breathing using our model **(Fig. 14)**. The total number of perfusion sites in all ten zones is 24. PEEP under spontaneous breathing will worsen perfusion in 15 sites, i.e. all areas with perfusion during respiratory pause and expiratory phase (red shading). However, impaired perfusion will lead to better oxygenation due to stopping 100% shunting in zone 9 during respiratory pause and in zone 10 during respiratory pause and expiratory phase (3 areas are marked with red and green shading). Using this dynamic model, we can clearly demonstrate negative impact of PEEP on lung perfusion under spontaneous breathing. It is difficult to evaluate the result of oxygenation as a whole. In fact, perfusion decreased in 15 sites across all zones along with improved ventilation (red shading). However, oxygenation would clearly improve due to cessation of 100% shunting in 3 zones (red and green shading). Following our logic, if we theoretically make PEEP equal to arterial pressure in basal parts of the lung (+25 mm Hg), perfusion in all zones will remain only in inspiration phase, with the exception of zone 1 without baseline perfusion **(Fig. 15)**. Using a dynamic model, we can clearly demonstrate negative impact of PEEP on lung perfusion under spontaneous breathing. Thus, progressive PEEP increase under spontaneous breathing from 0 to +25 mm Hg will result a sequential reduction in the number of ventilation / perfusion zones from 10 to 4. However, oxygenation will occur in only two out of four remaining zones.

"New" method of assisted ventilation (additional vacuum during inhalation phase)

Now we can see what areas of lungs and phases of respiratory cycle are exposed to PEEP during spontaneous breathing. There is a question whether we can improve pulmonary perfusion under spontaneous breathing using theoretical model. We found no literature data about the method of assisted ventilation that we want to propose for better lung perfusion under spontaneous breathing. According to dynamic model, additional vacuum in inhalation phase will increase blood flow and capillary filling, for example at inspiratory vacuum increase from –1.5 to –5 mm Hg **(Fig. 16)**.

Here is the dynamic model if we increase inspiratory vacuum to –3 mm Hg **(Fig. 17)**. Additional vacuum in inhalation phase improves perfusion in 7 out of 24 sites in 7 zones (green shading). Theoretically, additional vacuum in inhalation phase should decrease alveolar dead space. Improved perfusion of the apex of the lung may be useful for the treatment of infectious processes (tuberculosis, etc.).

Technically, it is simple to create additional vacuum in inhalation phase for spontaneous breathing. You need to use a valve with pressure regulator (like PEEP valve, only in reverse). Additional research will be necessary to answer the question of effectiveness of higher inspiratory vacuum and to determine the correlation between blood flow and vacuum. Creation of additional vacuum in inhalation phase during mechanical ventilation is discussed below.

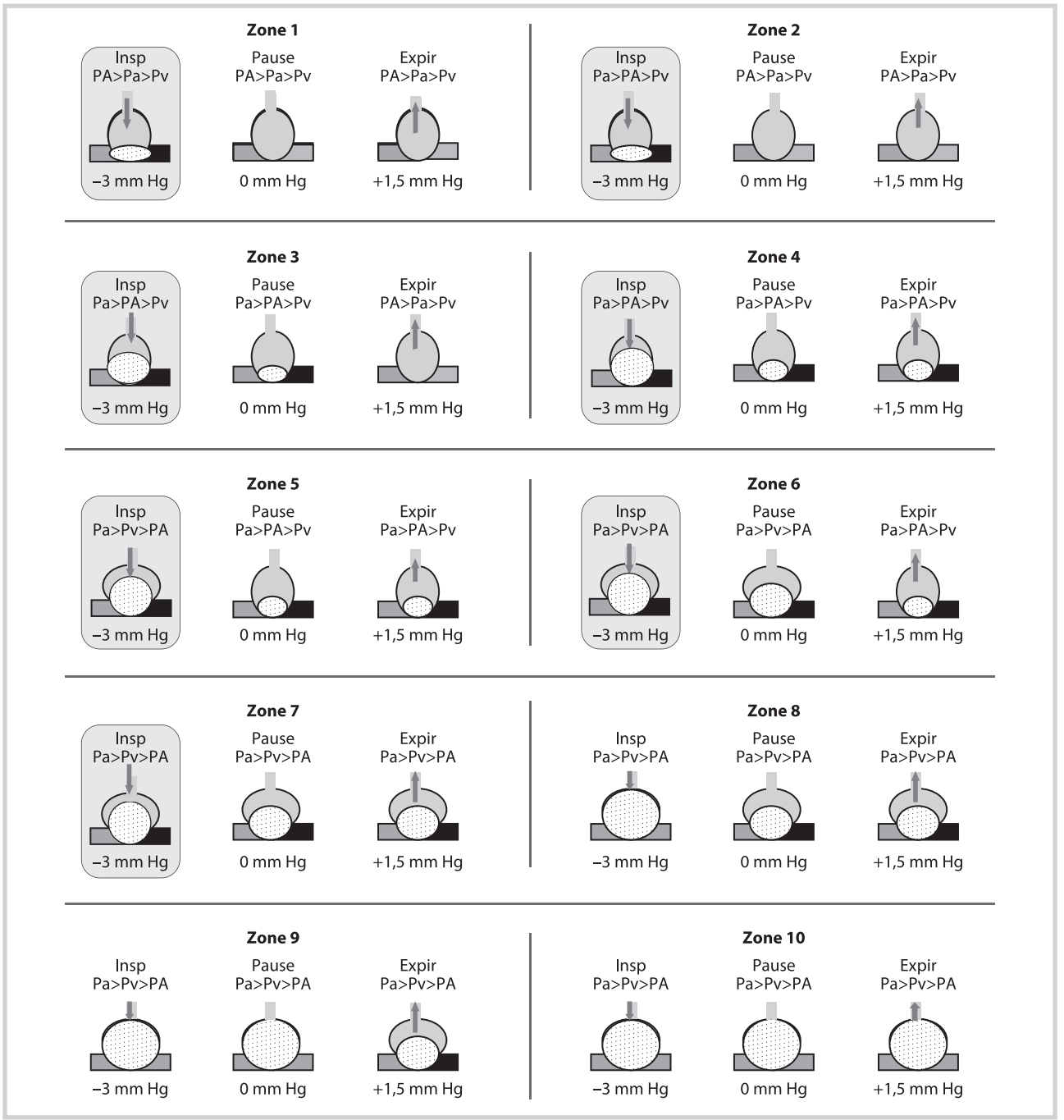

Fig. 17. **Effect of inspiratory underpressure –3 mm Hg on perfusion.**

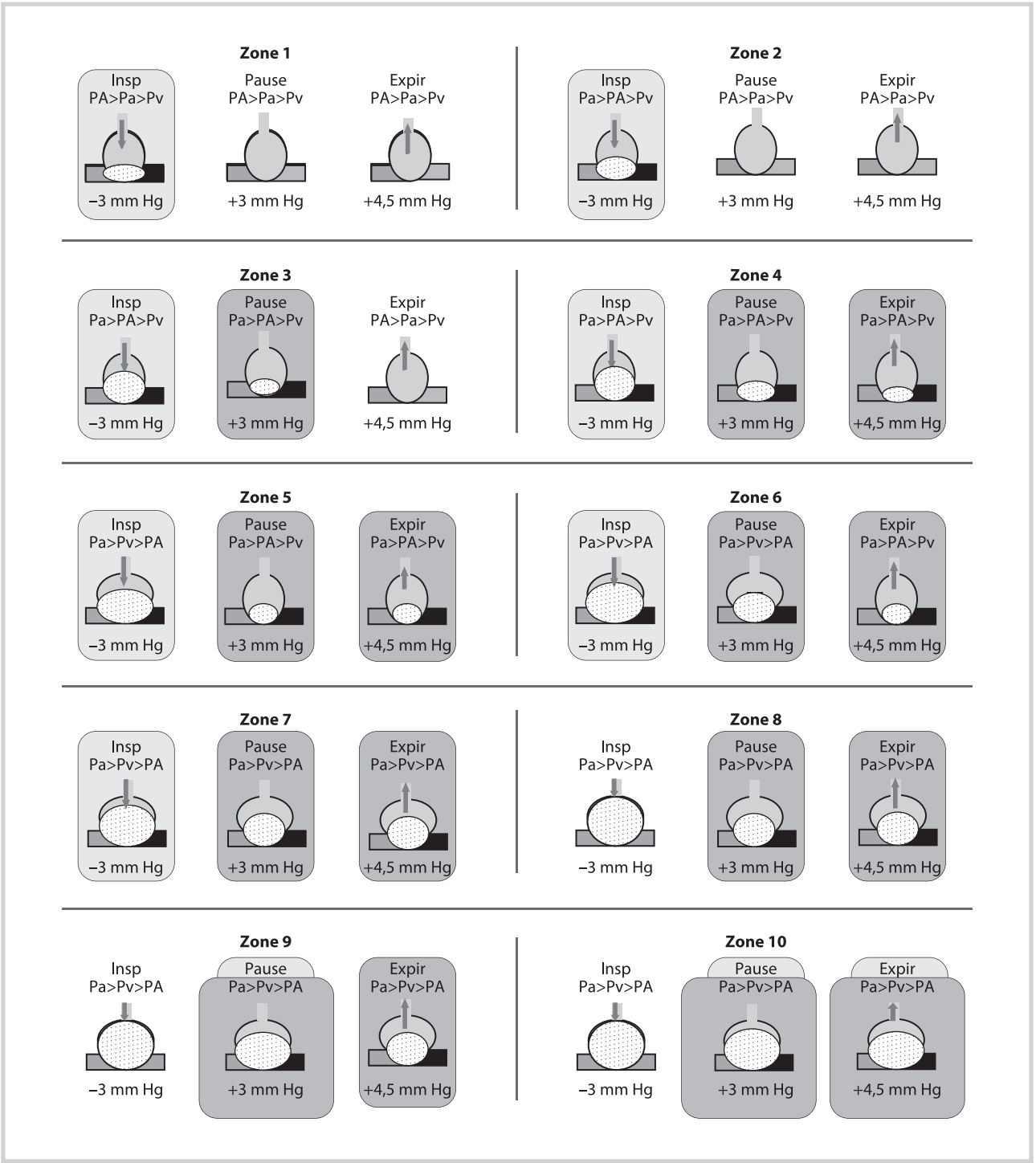

Fig. 18. **Dynamic model (PEEP +3 mm Hg) + (underpressure –3 mm Hg) under spontaneous breathing.**

Dynamic model of the influence of PEEP+additional inspiratory vacuum on ventilation / perfusion ratio under spontaneous breathing

Additional inspiratory vacuum under spontaneous breathing with PEEP will be followed by the following pattern. Dynamic model (PEEP +3 mm Hg)+(vacuum –3 mm Hg) under spontaneous breathing is presented in **Fig. 18**.

The total number of perfusion sites in all zones is 24. PEEP during spontaneous breathing will worsen perfusion in 15 sites, i.e. all sites perfused during pause and exhalation phase (red shading). However, impaired perfusion will improve oxygenation via decrease in 100% shunting in zone 9 during respiratory phase and zone 10 during respiratory pause and expiratory phase (3 areas are additionally highlighted in green shading). Additional vacuum will improve perfusion in 7 areas (green shading).

Now let us see what happens after combination of PEEP and additional vacuum. The total number of perfusion sites in all zones is 24. PEEP during spontaneous breathing will wors-

en perfusion in 15 sites. However, impaired perfusion will improve oxygenation via decrease in 100% shunting in 3 sites. Therefore, lower perfusion negatively affecting oxygenation will only occur in 12 sites. Nevertheless, additional vacuum will improve perfusion during inspiratory phase in 7 sites. Therefore, the overall ratio will be as follows: perfusion decrease in 12 sites, perfusion increase in 7 sites, reduced perfusion and improved oxygenation due to cessation of 100% shunting in 3 sites. It appears that ventilation / perfusion ratio will increase, but it is currently difficult to say what the effect on oxygenation will be. Experimental studies with additional vacuum in inhalation phase are needed to answer this question.

Effect of positive pressure ventilation on pulmonary perfusion according to dynamic model

Here we would like to estimate the effect of positive pressure ventilation on pulmonary perfusion according to dynamic model. For a better understanding, let us divide breathing cycle into phases. How will perfusion change if active inspiratory phase will be turned off? According to dynamic model, turning off the active inspiratory phase will deteriorate perfusion in 7 out of 24 sites (red shading). However, lower perfusion following turning off the active phase of inspiration will improve oxygenation due to stopping 100% shunt-

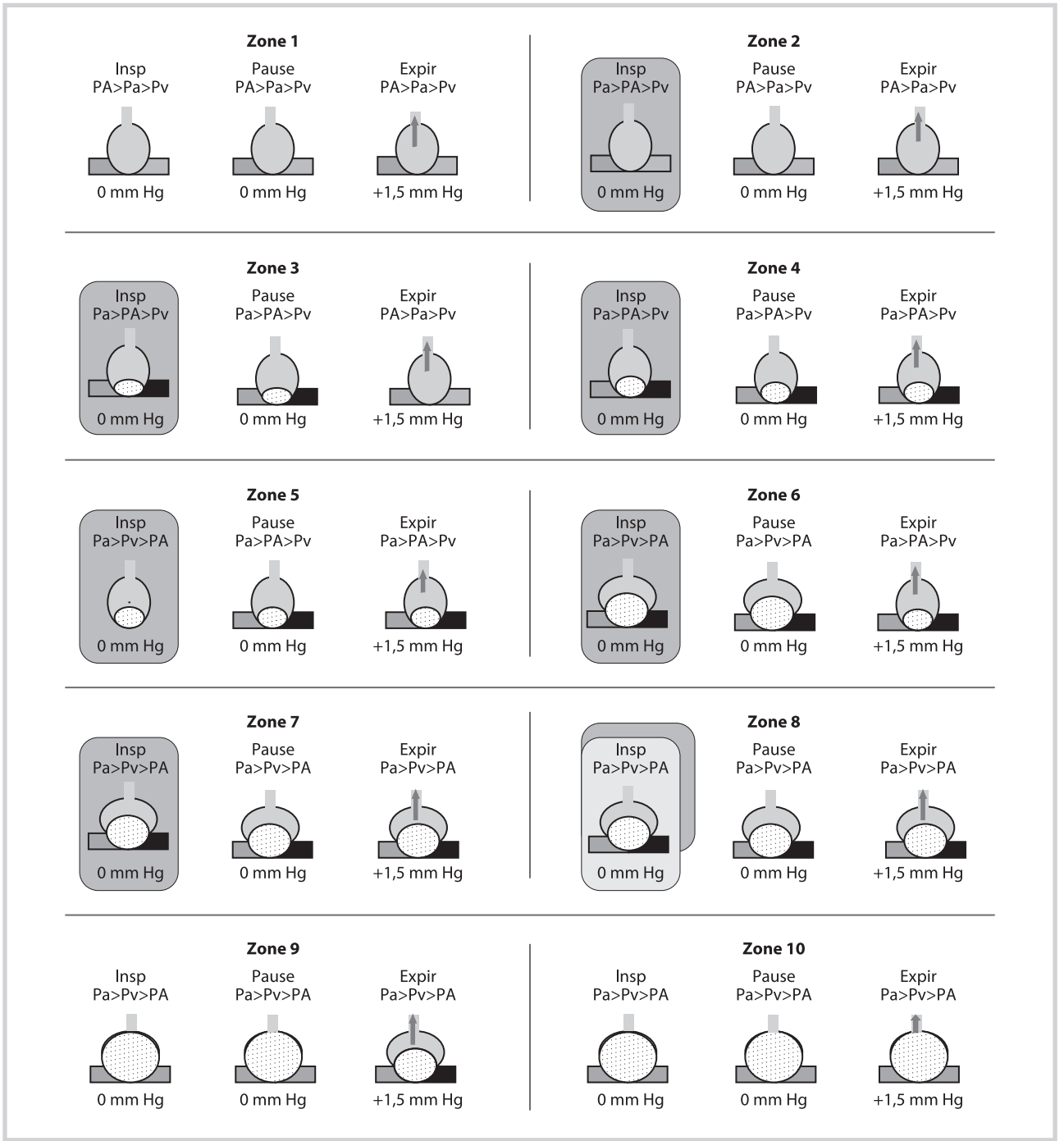

Fig. 19. **Active inspiratory phase is disabled.**

ing in 1 site of zone 8 (green shading) **(Fig. 19)**. Essentially, turning off the active inspiratory phase will lead to disappearance of zones 2, 5 and 8. Importantly, zones 2 and 5 improve oxygenation **(Fig. 20)**. Disappearance of zone 8 will decrease the shunting effect.

Positive pressure ventilation will have the same effects as turning off the active inspiratory phase, but to a greater extent depending on peak pressure **(Fig. 21)**. Positive pressure inspiration will deteriorate pulmonary perfusion in 8 out of 10 zones. Pulmonary perfusion will decrease in inspiratory phase and affect zones 3—10. In fact, this will lead to lower pulmonary perfusion in 8 out of 23 zones (red shading). However, shunting volume will decrease in zones 9 and 10 with 100% shunting in inspiratory phase. This process will improve oxygenation (zones are additionally highlighted with green shading).

Thus, ventilation / perfusion ratio will increase during inspiratory phase at positive pressure ventilation. Therefore, this will increase alveolar dead space. Theoretically, we can stop capillary blood flow in all zones during inhalation phase using peak pressure +25 mm Hg **(Fig. 22)**.

Effect of positive pressure ventilation and positive end-expiratory pressure on perfusion in lung regions according to dynamic model

This most common method of ventilation is the most destructive for capillary perfusion. Dynamic model for positive pressure ventilation+PEEP is presented in **Fig. 23**. This figure demonstrates the extent of capillary perfusion catastrophe following positive pressure ventilation+PEEP, i.e. the most common method in modern anesthesiology and intensive care. Negative effect on capillary blood flow will be observed in all 24 sites (red shading). However, ventilation / perfusion ratio will increase in 6 sites with 100% shunting (zones 8—10). This will positively affect oxygenation due to disappearance of 100% shunting (green shading) **(Fig. 24)**.

It is well known that modern methods of ventilation and especially mechanical ventilation with PEEP are not physiological. However, are there other more physiological methods of ventilation? Yes, they exist, or rather, they existed. These are cuirass ventilators. Considering modern electronics and materials, we think that they can be made compact and controlled. Let's look at how cuirass ventilation will affect perfusion in lungs.



Fig. 20. **Disappearance of zones 2, 5 and 8.**

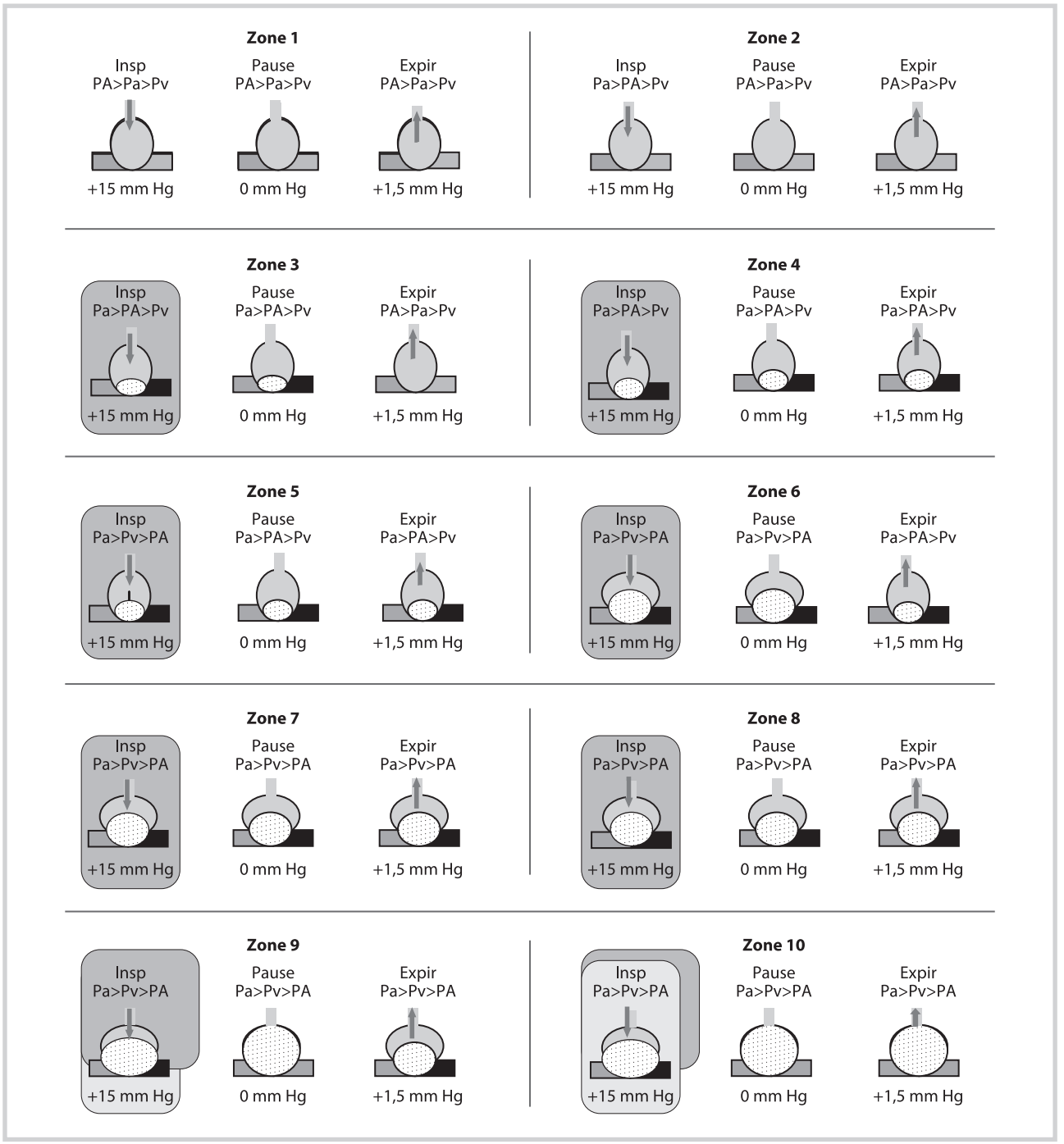

Fig. 21. **Effect of positive pressure inspiration on pulmonary perfusion.**

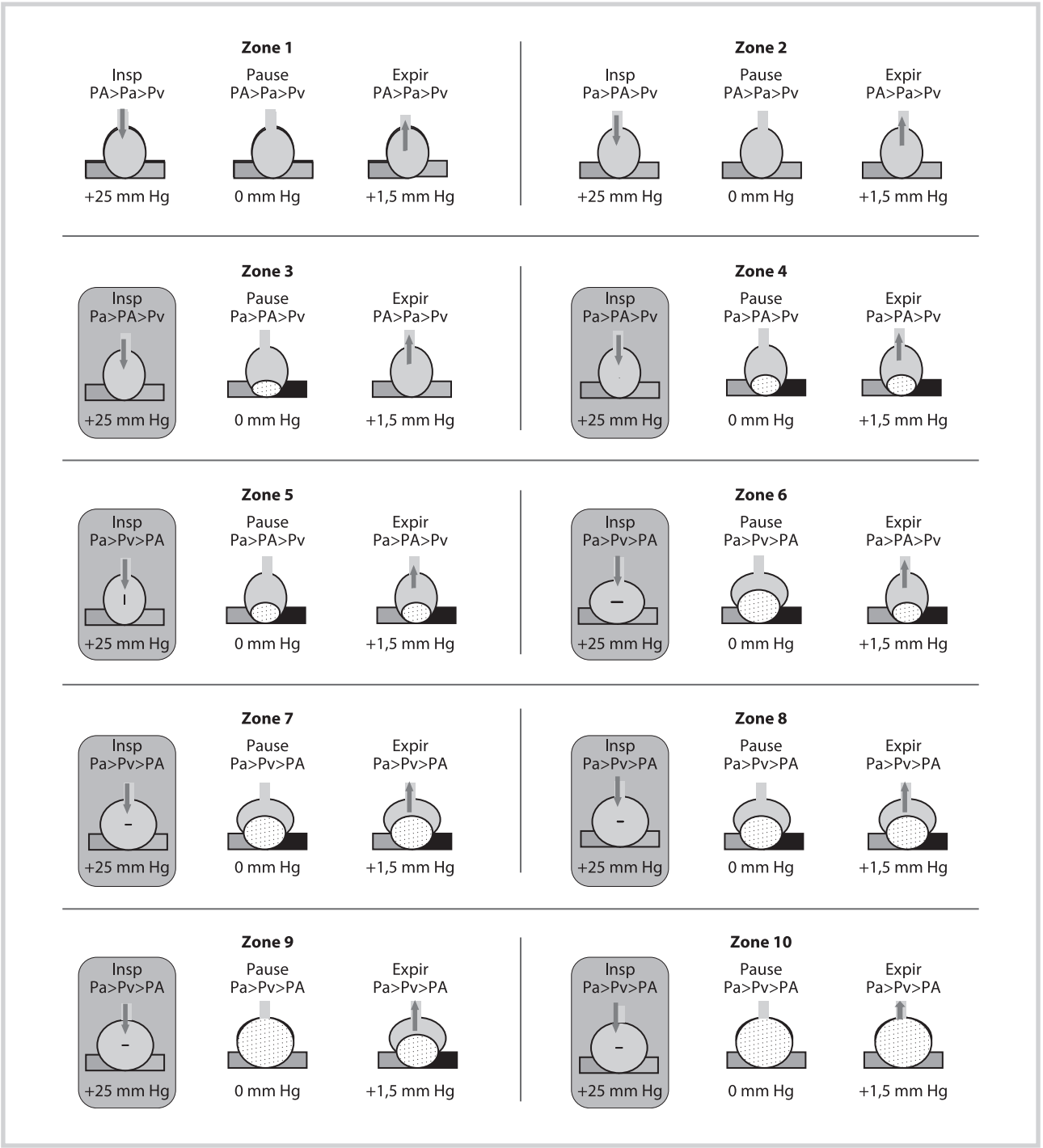

Fig. 22. **Peak pressure +25 mm Hg stops perfusion during inspiratory phase.**

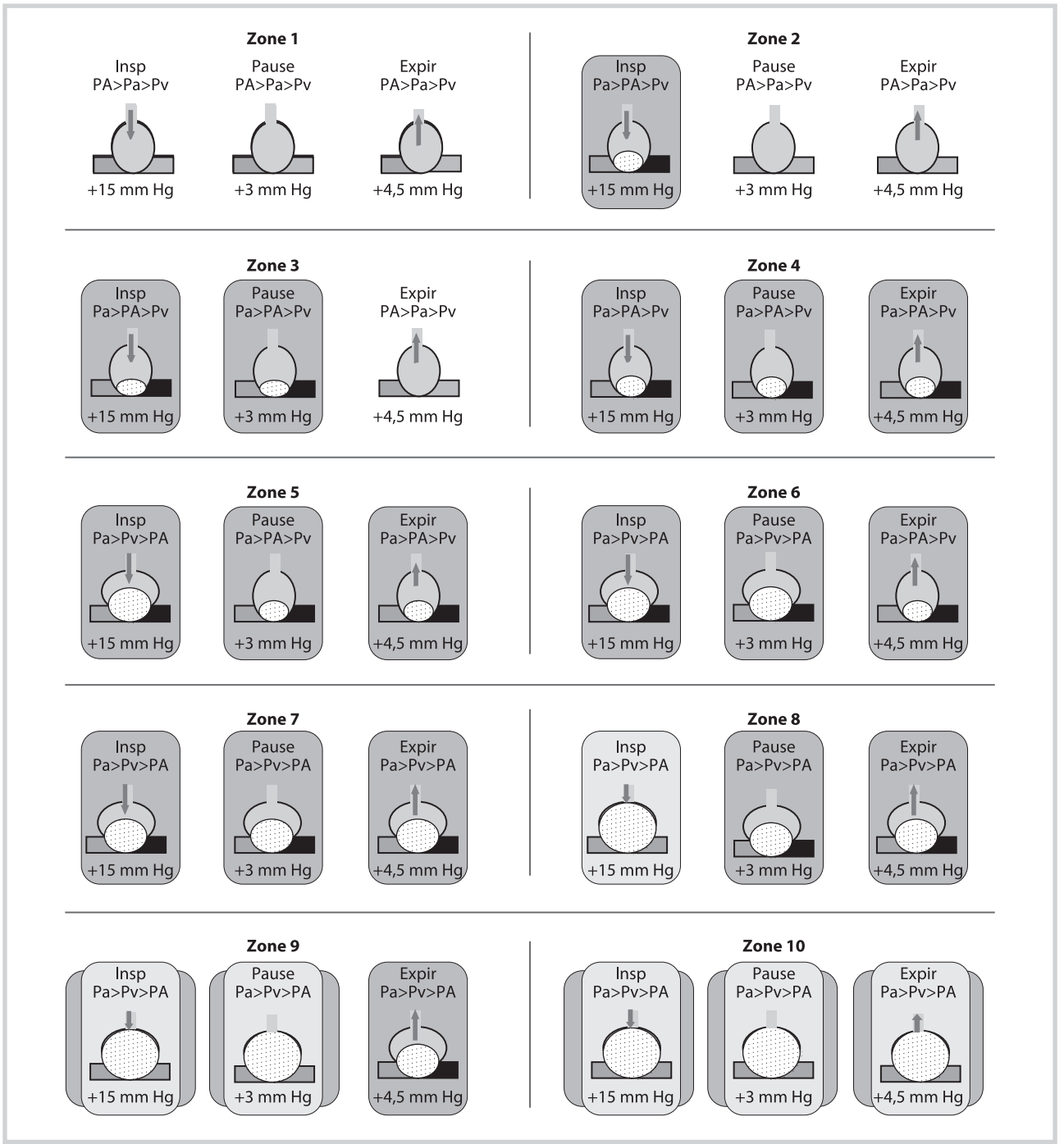

Fig. 23. **Capillary perfusion during positive pressure ventilation + PEEP.**

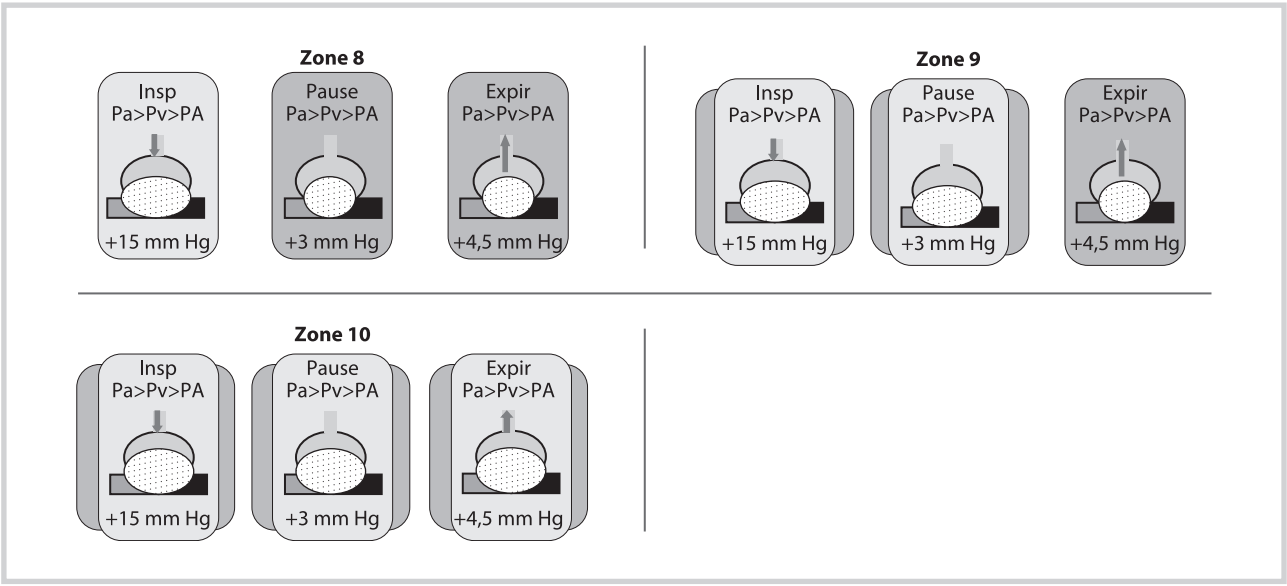

Fig. 24. **Increased ventilation-perfusion ratio in 6 sites of 100% bypass.**

Dynamic model for cuirass ventilation is shown in **Fig. 25**, and it does not differ from the model for spontaneous breathing. This is understandable, since cuirass ventilation without additional methods will not have a negative effect on lung perfusion, will not worsen or improve ventilation.

In case of additional inspiratory vacuum, we can improve perfusion in 7 areas during cuirass ventilation and spontaneous breathing (green shading) **(Fig. 26)**. There is no need for PEEP during cuirass ventilation because PEEP is designed to increase functional residual capacity (FRC). In case of cuirass ventilation, we can increase FRC by limiting chest size at the end of exhalation, i.e. stopping exhalation. With this method, the ventilated alveoli may remain open not because of additional pressure, but due to additional traction.

Combination of cuirass ventilation and positive pressure ventilation

We understand that cuirass ventilation has own drawbacks and difficulties, but it is more physiological for lungs. We also recognize few diseases potentially requiring "pure" cuirass ventilation. Therefore, we believe that combination of positive pressure ventilation and cuirass ventilation will open up enormous new possibilities for control of respiratory function.

Ventilation and perfusion parameters influenced by combination of positive pressure ventilation and cuirass ventilation

1. Preservation and control of negative pressure during inspiratory phase and blood flow to the lungs and heart.
2. Additional inspiratory vacuum will allow you to regulate negative alveolar pressure and control lung perfusion during inhalation phase.
3. One can control FRC by limiting end-tidal chest size and adding PEEP if necessary (PEEP will be significantly lower compared to appropriate value in positive pressure ventilation).
4. Tidal volume can also be controlled by vacuum in cuirass device and peak pressure in breathing circuit (peak pressure will be significantly lower compared to appropriate value in positive pressure ventilation).
5. Selecting the parameters of cuirass device (vacuum, negative pressure in respiratory circuit during inhalation phase, inhalation delay, exhalation phase delay, active exhalation, inhalation/exhalation ratio) and parameters of positive pressure of ventilator (peak pressure), PEEP, inspiratory/expiratory airflow ratio, oxygen concentration and nitric oxide can provide good ventilation with minimal impact on perfusion and reduce shunting.
6. Forced exhalation is possible (of course, this will negatively affect hemodynamics, but periodic use is acceptable for better mucus drainage).
7. Combination of cuirass ventilation with high-frequency ventilation may be useful.

Dynamic model of positive pressure ventilation combined with cuirass ventilation is presented in **Fig. 27**. You can see perfusion zones and lung zones influenced by parameters of cuirass ventilation device and positive pressure ventilation device. Alveolar pressure is similar to that in spontaneous breathing (initial model). In our opinion, control of these parameters combined with positive and negative pressure ventilation will allow respiratory support with minimal impact on pulmonary function and hemodynamics.

Parameters potentially influenced by combined positive and negative pressure ventilation

Vacuum in cuirass device and method of additional vacuum influence on perfusion sites in 7 zones of the lung during inspiratory phase (green shading). Peak circuit pressure affects all perfusion sites during inspiratory phase in all 10 lung zones (red shading). PEEP affects 20 perfusion sites in respiratory pause and expiratory phase in all 10 zones of the lung (blue shading). Positive pressure in cuirass device for active exhalation affects all perfusion sites during expiratory phase in all 10 zones of the lung (yellow shading).

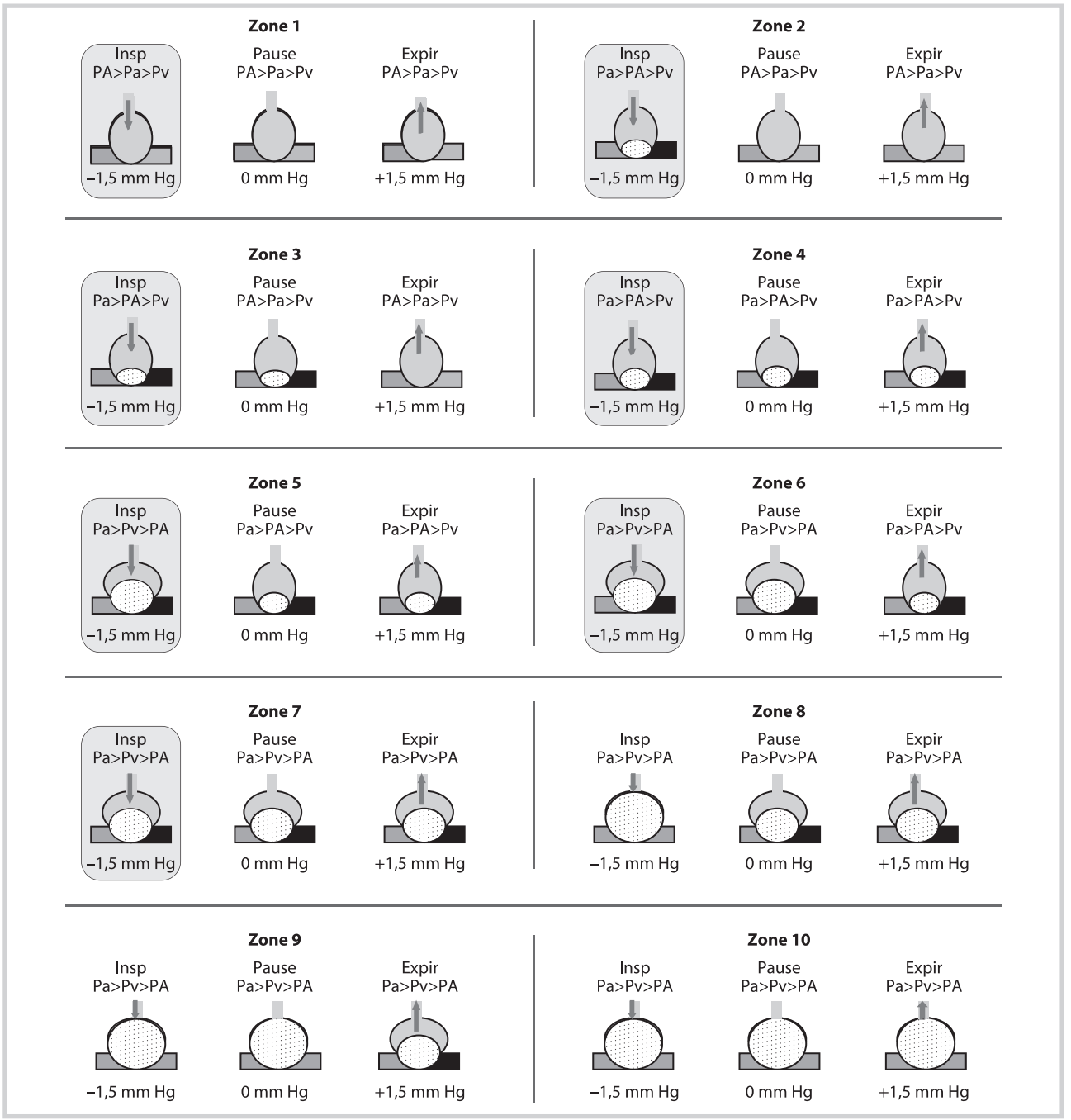

Fig. 25. **Effect of cuirass ventilation on lung perfusion.**

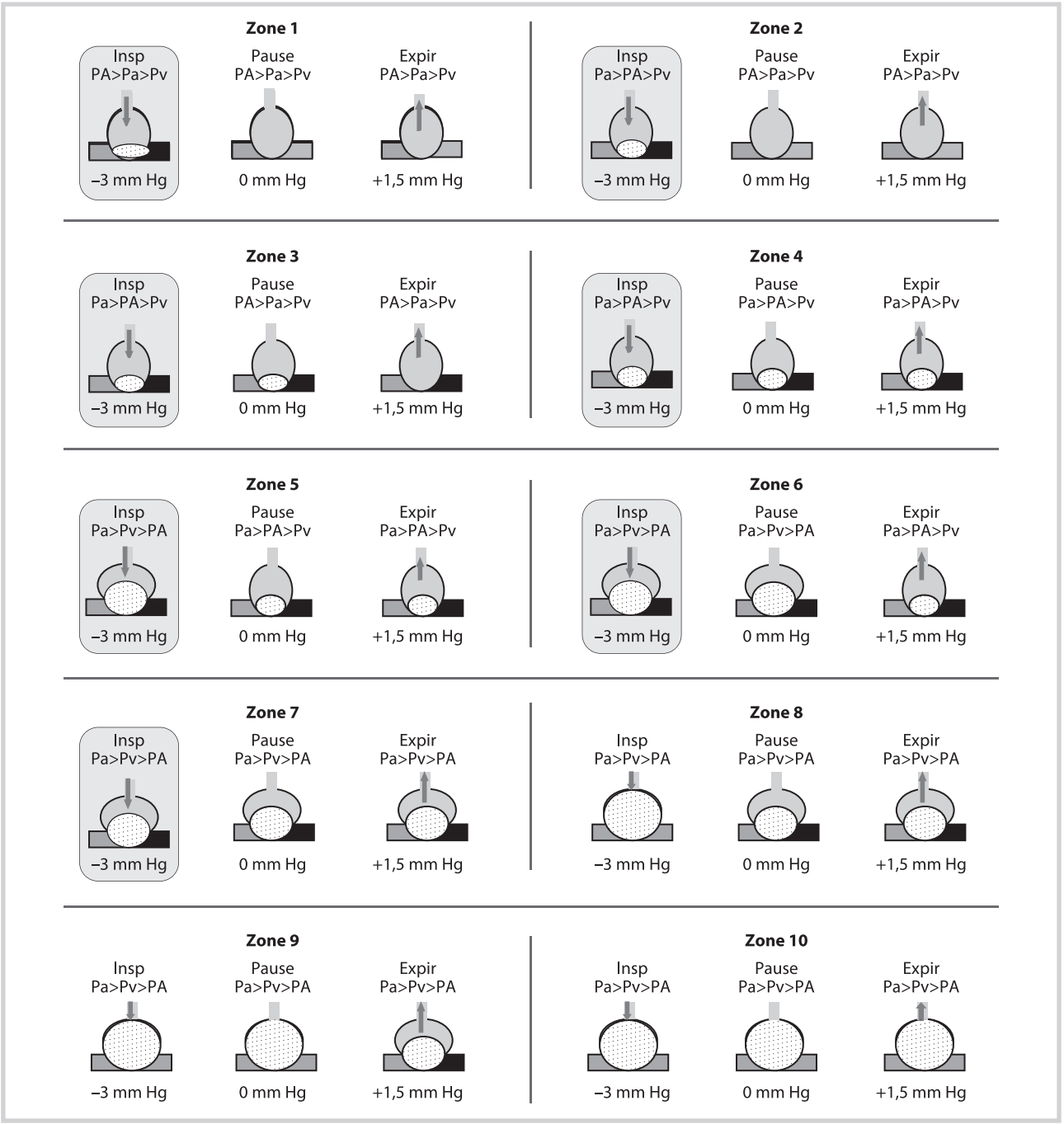

Fig. 26. **Effect of additional inspiratory underpressure on pulmonary perfusion.**



Fig. 27. **Perfusion zones and lung zones affected by cuirass and positive pressure ventilator parameters.**

Conclusion

Importantly, the proposed model is purely theoretical and intended for a better understanding of pulmonary ventilation and perfusion. Many of our assumptions need to be tested. Additional research of cuirass ventilation and development of new ventilators are needed. Currently, there are several devices for cuirass ventilation in the world. The medical company Hayek Medical releases the Hayek MRTX respirator for two-phase cuirass ventilation. The Dima Italia company produces the Pegaso Vent negative pressure ventilator and several devices for its use (Soft Cuirass, Poncho and Dima-Lung). In 2015, our colleagues from Germany published the results of positive and negative pressure ventilation in the treatment of a 75-year-old patient with acute respiratory distress syndrome. The result of combined respiratory support was positive [4].

Perhaps, combination of cuirass ventilation and positive pressure ventilation opens up great prospects in the treatment of severe pulmonary diseases. However, it is currently difficult to imagine the full extent of the work that needs to be done to confirm or refute the effectiveness of combination of these two types of respiratory support.

We hope that proposed dynamic model will be useful for understanding the processes of ventilation and perfusion in lungs and, possibly, for predicting the results of any methods of respiratory support.

Author contribution:
Concept and design of the study — Kolesnik A.I.
Collection and analysis of data — Kolesnik A.I.
Writing the text — Gritsan A.I., Kolesnik A.I., Bushueva L.V., Kokshin D.V.
Editing — Gritsan A.I.

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Comment on the article

"Dynamic model of the influence of gravity, ventilation with positive and negative pressure on ventilation-perfusion ratio in lungs"

One can congratulate the authors with this interesting report immersing the reader in fascinating and extremely significant problem of ventilation-perfusion (V/Q) imbalances and alternative options for their correction. The most significant result of original dynamic model seems to be educational. Indeed, the reader participates in a scrupulous analysis of V/Q disorders from the position of classical physiological ideas about West zones [1].

Considering obvious non-physiological nature of traditional ventilation with injecting gas into the airways, the authors hypothesize that ventilation with negative pressure in airways during inspiration is more physiological and probably more effective regarding gas exchange through alveolar-capillary membrane.

However, these postulates seem dubious from an applied point of view.

Firstly, the authors propose to create negative pressure in airways during inhalation through phase rarefaction of air around the chest wall using a cuirass respirator. In this case, air will flow into the lungs along pressure gradient created not by diaphragm contraction, but through raising the ribs and chest enlargement. Management of tidal volume in such a situation seems to be an unpromising objective both in presence and absence of spontaneous breathing.

Second, the idea about potential clinical benefit of negative pressure ventilation or its combination with increased end-expiratory pressure appears simplistic and mechanistic. The authors' assumption is based on modeling of V/Q imbalance in a healthy person under spontaneous breathing in upright body position. Under such conditions, the number of alveoli balanced in ventilation and perfusion is greater in negative inspiratory pressure compared to traditional ventilation. However, will this pattern persist in clinical conditions with numerous and heterogeneous V/Q disorders?

The need for mechanical respiratory support (MRS) may be due to various pathophysiological reasons, i.e. hemodynamic (pulmonary congestion or, conversely, depleted perfusion), respiratory (hypoventilation, bronchial obstruction, pulmonary infiltration) or their combination. Therefore, V/Q balance even in neighboring alveolar acini, including those localized in different gravitational zones, can differ significantly.

Thus, if we leave aside the rarer causes of MRS (pneumothorax, atelectasis, alveolitis obliterans), we can recognize significant diversity of V/Q disorders. How will the alternative model of pulmonary ventilation behave under such conditions? Will its hypothetical advantages remain? Apparently, extrapolation of the modeled prevalence of V/Q disorders into clinical practice will be difficult or even impossible.

Nevertheless, this report left a positive impression. Professional presentation and excellent illustrations make us think again about prevention of inevitable side effects of traditional ventilation.

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