



Pulmonary Surfactant Prevent Formation of Blocking Liquid Columns

Maaïke De Vries*

Department of Epidemiology, University Medical Center Groningen, University of Groningen, the Netherlands, Germany

Abstract

Pulmonary surfactant consists of about phospholipids, amphipathic molecules characterized by having a polar head which is hydrophilic, whereas the fatty acids, at the other end of the molecule, are decidedly hydrophobic. Because the molecule is partly explicitly hydrophobic it cannot be completely surrounded by water molecules, the fatty acids must stay away from the water and they will if they adjoin the fatty acids of other phospholipid molecules. This is probably the reason that the molecules, as they are synthesized, form typical arrangements in the moist cytoplasm. They are known as lamellar bodies and consist of concentric bilayer shells, layered one outside the other.

Keywords: Bilayer shells; Air-liquid interface; Plasma proteins; Alveolar expansion; Amphipathic molecule; Pre-mature infants;

Introduction

Between the phospholipid molecules, composing the bilayers of the lamellar bodies, are also the surfactant associated proteins, nominated surfactant protein. In the last few years they have been the focus of an enormous investigative activity [1]. However, their function is still not completely clear, although two of them, known to be essential since they promote rapid adsorption, a fast formation of a phospholipid monolayer at an air-liquid interface. Clark strongly supports the functional importance [2]. When the gene for this apoprotein was disrupted in mice, lamellar bodies did not form in a normal manner in the fetal cells and, in spite of postnatal respiratory efforts, the lungs did not become expanded and the neonates succumbed. There are also reports that surfactant proteins counteract the surfactant inhibiting effect of plasma proteins and that helps combat airway infection. There are several excellent reviews on the molecular biology, structure and function of the apoproteins. The equivalent surface tension will be very much affected by an expansion of the surface area at which a monolayer has formed [3].

Methodology

As the meniscus of the airway is moving in the direction of the alveoli during the initial aeration there is a continuous expansion of the conducting airway's air-liquid interface. The absorbance of amphipathic molecules to this expanding surface area may not be quick enough to keep up the number of molecules that would be present at the surface under equivalent conditions [4]. The intermolecular distance would then be greater, resulting in a higher value of surface tension. Once the alveolus becomes aerated the surface area is vastly increased and when breathing has been established there will be a regular oscillation of the surface area, a compression during expiration and an expansion during inspiration [5]. This would mean that for the alveolus to remain expanded during expiration, it has to be surrounded by a pressure which becomes more negative as the alveolar radius diminishes. However, it is well known that during expiration trans-mural pressure is diminishing. Furthermore, the alveoli will not all be of the same size, and those that are smallest are at the greatest risk of collapsing since they require to be surrounded by a greater negative pressure to remain expanded [6]. Thirty-five years ago there were several established methods to measure surface tension, but Clements saw the need for a method to evaluate how surface tension was affected when a film, formed at the air-liquid interface, was rhythmically compressed and expanded [7].

Discussion

Using the modified wilhelm balance he demonstrated very conclusively that lung lavage fluid, when spread over the surface of a Langmuir trough, will show a much lower value of surface tension when the surface area is compressed than when it is expanded. From this observation he drew the conclusion that when the surface area is being compressed, as in alveoli during expiration, surface tension will be lowered, a phenomenon he suggested would be an anti-atelectasis factor [8]. The value might then diminish during expiration, such that the need for a negative intra-pleural pressure to maintain alveolar expansion would gradually be reduced. When the alveolar surface area is expanding, as it will be during inspiration, surface tension increases and if augmented more than the radius, the value will increase [9]. The reason that amphipathic molecules cause surface tension to change when surface area is compressed or expanded has been the subject of many studies. A current report gives an excellent review of the most recent concepts [10]. The amphipathic molecule is characterized by having a polar head which is hydrophilic while the opposite end is hydrophobic, and for that reason will be attracted to air. The major component of pulmonary surfactant is more than half of that phospholipid has two acids constituting the molecule's hydrophobic end. Palmitic acid is a chain which is straight since it has no double bonds [11]. For this reason, molecules can be very tightly packed, and when the surface area is compressed, forcing the molecules to come even closer together, this is mechanically resisted. Clements used the wilhelm balance for an evaluation of pulmonary surfactant as shown in (Figure 1). A chemical analysis showed him that a major component consisted, and when he studied that phospholipid alone, dissolved in hexane, he found, after the hexane had evaporated, that the monolayer would exert very high surface pressure just as pulmonary surfactant would. Based on that observation Clements hypothesized that, since it had been shown that premature infants developing the respiratory distress syndrome

*Corresponding author: Maaïke De Vries, Department of Epidemiology, University Medical Center Groningen, University of Groningen, the Netherlands, Germany, E-mail: Maaïke@gmail.com

Received: 28-Aug-2023, Manuscript No. JRM-23-115396; **Editor assigned:** 30-Aug-2023, PreQC No. JRM-23-115396 (PQ); **Reviewed:** 14-Sep-2023, QC No. JRM-23-115396; **Revised:** 20-Sep-2023, Manuscript No. JRM-23-115396 (R); **Published:** 27-Sep-2023, DOI: 10.4172/jrm.1000179

Citation: Vries MD (2023) Pulmonary Surfactant Prevent Formation of Blocking Liquid Columns. J Respir Med 5: 179.

Copyright: © 2023 Vries MD. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.



Figure 1: Wilhelm balance for an evaluation of pulmonary surfactant.



Figure 2: Clinical trial not to be effective treatment.

had a surfactant deficiency, it might be possible to prevent and treat by supplying to the airways. He organized a clinical trial which was carried out in Singapore. Turned out not to be an effective treatment probably because its absorbance is extremely slow as shown in (Figure 2). Nevertheless, the concept to supply surfactant the premature infant was missing appeared to be correct [12]. If instilled into the airways before the infant takes its first breath, the surfactant might prevent a condition which largely develops as a vicious circle. The surfactant instillation might preclude from ever being induced. Furthermore, there is the possibility that the monolayer is not only at the surface of the bubble, but is also extruded into the vertical capillary where it would line its inner wall [13]. It is a distinct possibility that surfactant is indeed extruded into the vertical capillary, but if that happens it is likely that the wall of the capillary eventually becomes saturated, and that would impede further extrusion of the bubble monolayer. That assumption was supported by experiences with the hypo-phase exchange [14]. An active preparation of pulmonary surfactant was studied, and when the recording showed that an active surfactant monolayer had formed and was lining the bubble, the liquid surrounding it was replaced with saline solution. This could be done repeatedly without a significant change of the surface tension recording.

Conclusion

If there had been a continued loss of the monolayer to the capillary the tracing would indicate a loss of activity since surfactant molecules from the hypo-phase could no longer be replaced. To avoid the possibility of a monolayer loss to the capillary was made a modification of the pulsating bubble surfact-meter which allows him to study a captive bubble. He has also developed an interesting technique to evaluate the surface tension existing inside the airways.

Acknowledgement

None

Conflict of Interest

None

References

- Gergianaki I, Bortoluzzi A, Bertsias G (2018) Update on the epidemiology, risk factors, and disease outcomes of systemic lupus erythematosus. *Best Pract Res Clin Rheumatol* EU 32:188-205.]
- Cunningham AA, Daszak P, Wood JLN (2017) One Health, emerging infectious diseases and wildlife: two decades of progress? *Phil Trans UK* 372:1-8.
- Sue LJ (2004) Zoonotic poxvirus infections in humans. *Curr Opin Infect Dis* MN 17:81-90.
- Pisarski K (2019) The global burden of disease of zoonotic parasitic diseases: top 5 contenders for priority consideration. *Trop Med Infect Dis* EU 4:1-44.
- Kahn LH (2006) Confronting zoonoses, linking human and veterinary medicine. *Emerg Infect Dis* US 12:556-561.
- Bidaisee S, Macpherson CNL (2014) Zoonoses and one health: a review of the literature. *J Parasitol* 2014:1-8.
- Cooper GS, Parks CG (2004) Occupational and environmental exposures as risk factors for systemic lupus erythematosus. *Curr Rheumatol Rep* EU 6:367-374.
- Parks CG, Santos ASE, Barbhaiya M, Costenbader KH (2017) Understanding the role of environmental factors in the development of systemic lupus erythematosus. *Best Pract Res Clin Rheumatol* EU 31:306-320.
- Barbhaiya M, Costenbader KH (2016) Environmental exposures and the development of systemic lupus erythematosus. *Curr Opin Rheumatol* US 28:497-505.
- Cohen SP, Mao J (2014) Neuropathic pain: mechanisms and their clinical implications. *BMJ* UK 348:1-6.
- Mello RD, Dickenson AH (2008) Spinal cord mechanisms of pain. *BJA* US 101:8-16.
- Bliddal H, Rosetzky A, Schlichting P, Weidner MS, Andersen LA, et al (2000) A randomized, placebo-controlled, cross-over study of ginger extracts and ibuprofen in osteoarthritis. *Osteoarthr Cartil* EU 8:9-12.
- Maroon JC, Bost JW, Borden MK, Lorenz KM, Ross NA, et al. (2006) Natural anti-inflammatory agents for pain relief in athletes. *Neurosurg Focus* US 21:1-13.
- Birnesser H, Oberbaum M, Klein P, Weiser M (2004) The Homeopathic Preparation Traumeel® S Compared With NSAIDs For Symptomatic Treatment Of Epicondylitis. *J Musculoskelet Res* EU 8:119-128.