Hypothetical Cause of Flat Chest Kitten Syndrome

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What this paper sets out to do is expand on a theory as to the probable cause of FCKS. It suggest that FCKS is related to a condition called atelectasis. In order to do the paper justice it is important to understand some basic principles of science and relate them to this hypothesis.

The Science

It is important to consider an aspect of physics then look at anatomy and physiology.

First Physics

Solids

When individual grains of sand or salt come together they are still individual grains of sand or salt. You can demonstrate this by simply lifting up a handful of sand or salt and allowing it to run through your fingers. It reverts back to individual grains

Liquids

H_2O otherwise known as water has unique properties associated with liquids and I will examine one property here. This property is called surface tension. If you examine water droplets carefully and look at the edges, they actually curve around. This is surface tension working on the water molecules. When droplets of water come together, they combine to become one large water droplet. It is unlikely that one can create the original water droplets from this large drop.

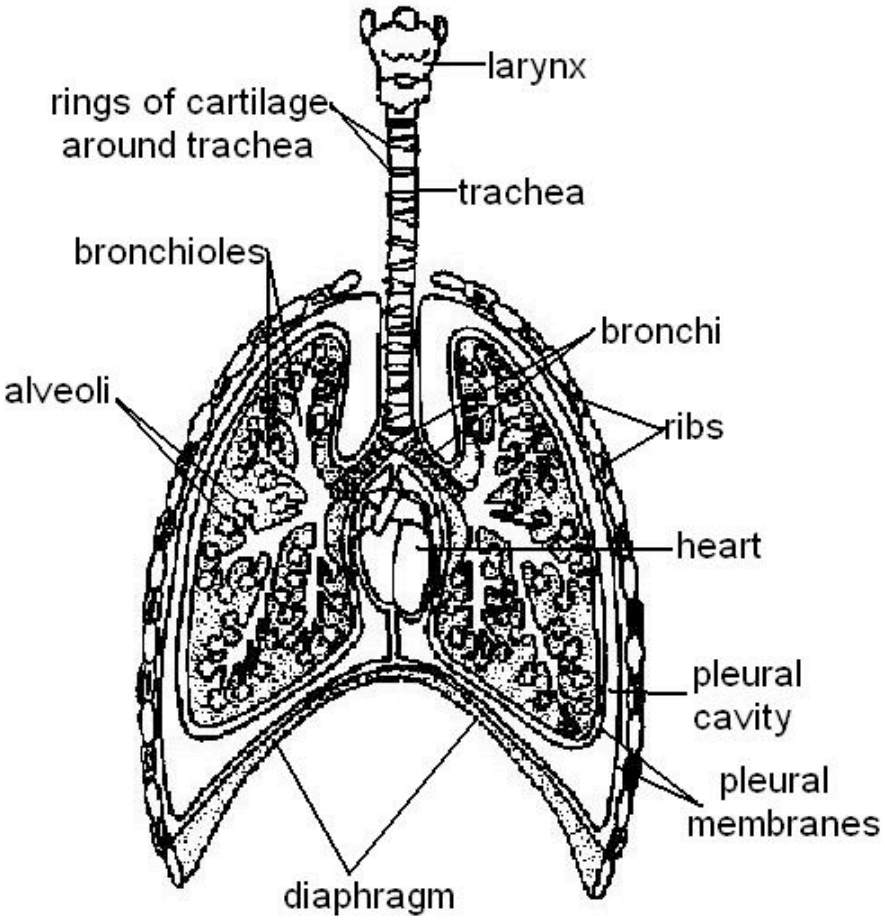
Many insects make use of this phenomenon like pond skaters that can literally walk on water. This surface tension also seems to draw any other water droplets to it that are close by, a bit like a magnet.

Now if one were to protect the droplets by coating them in a layer of oil then they would not combine but remain separate. The oily layer prevents surface tension working on the droplets. This is not the same as simply dropping oil into the water as this just sits on the surface of the water and does not actually coat the whole droplet

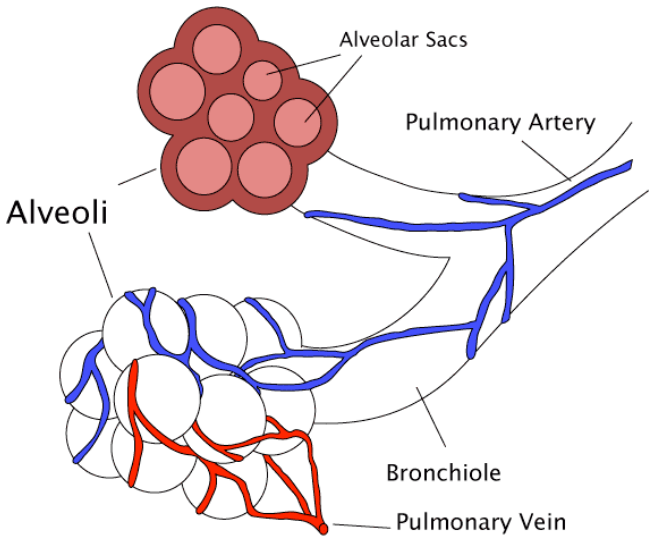
Now the anatomy and physiology.

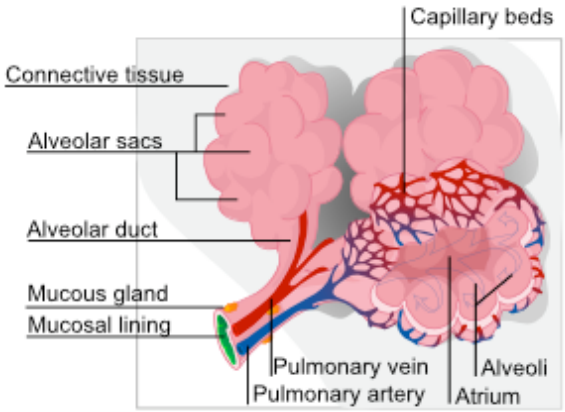
Let us start by looking at the respiratory system.

Diagram of respiratory system of a cat

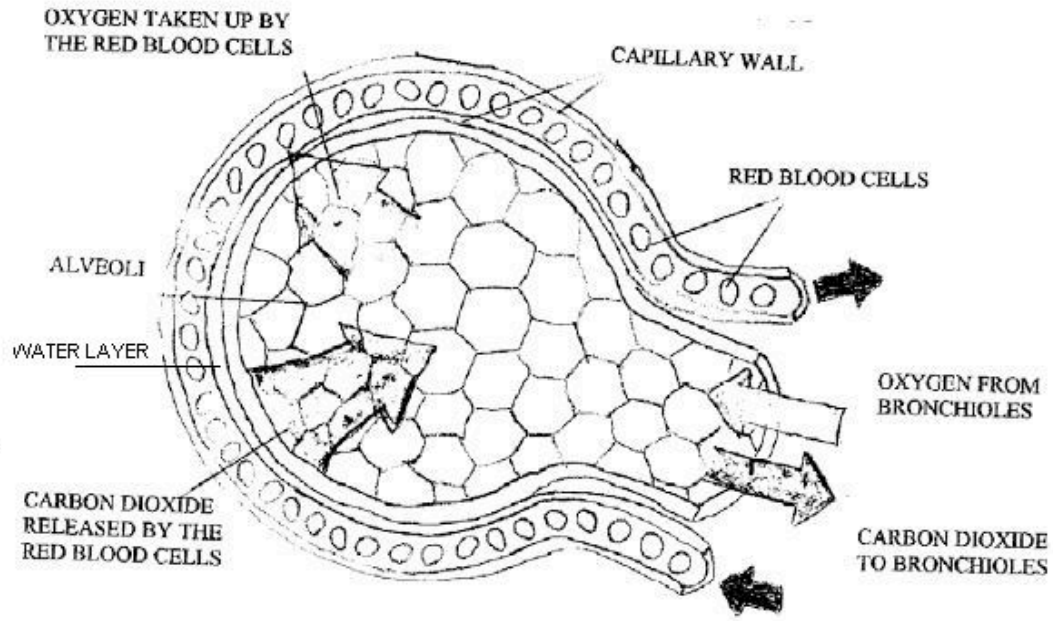


Breaking the anatomy of the lungs down in more detail





The main function of the lungs is to absorb oxygen into the bloodstream from the atmosphere and to expel carbon dioxide from the blood into the exhaled breath



Each alveolus is protected by a surface layer of water coated with a surfactant.

British Journal of Anaesthesia 1990; 65: 13–29

THE ROLE OF LUNG SURFACTANT

B. A. HILLS

Surfactant is essential for the wellbeing of the lung, a deficiency in quantity or composition causing atelectasis, alveolar flooding and generally compromising the spatial integrity of the mucosal surface needed for efficient gas exchange. The manner in which this delicate architecture can change has been well described in texts on the pathology of the respiratory distress syndrome (RDS) of the newborn [24, 76] and, more recently, in the adult form (ARDS) which is being diagnosed more frequently now that this specific disease entity has been recognized and better characterized clinically. The incidence of ARDS in the U.S.A. has been estimated [63] as 150000 cases per year, with a mean mortality rate of 50%. Surfactant is probably an important underlying factor in some cases of ventilator dependency, as the later discussion of its role in the work of breathing will imply.

There is also current research indicating that surfactant is present in its "active" state in many organs besides the lung and may provide the gastric mucosal barrier [50], the load-bearing lubricant in the joints [45], the release agent facilitating ventilation of the middle ear [12, 37] and the lubricant/release agent normally preventing premature rupture of the placental membrane [51]. A common metabolic pathway for the production of surfactant at different sites in the body could explain several interesting correlations between disease states—for example, why many patients with peptic ulcer also display impaired pulmonary function [56].

Despite the dominant role of surface factors in lung mechanics and alveolar flooding, surfactant is seldom mentioned in major reviews—especially of fluid balance in the lung [84, 85, 87]—probably because its inclusion invokes the relatively alien disciplines of physics and surface chemistry. However, it is surface activity which sets surfactants apart from other substances and so a little surface physics is essential for an in-depth understanding.

In this review we shall consider many basic aspects of the lung which, with few exceptions, have tended to be left in the "too-hard basket" for the past decade or two [42]. The more popular and pragmatic approach directed primarily to the problem of RDS has been to argue that, whatever its physical role, any deficiency in surfactant needs to be addressed by correction of the underlying metabolic problem or by supplementation with effective surfactant. Thus the major effort in the field today is either biochemical in nature or aimed at supplementation—either by promoting surfactant secretion with drugs such as ambroxol [60] or, more commonly, by administering exogenous surfactant to the neonate in various forms from various sources. The latest state of the art is contained in two volumes of collected papers published recently [20, 76]. The basic problem seems to be the compromise between the risk of transmitting pathogens and the loss in surface activity with processing of surfactant from natural sources. Although synthetic pathogen-free surfactant has so far defied dispersion in an aqueous medium while retaining surface activity of the "natural" material, that activity has been obtained in an ingenious formulation devised by Bangham and co-workers [7] in which the material is micronized into the air inspired by the patient. This has resulted in encouraging clinical results for premature babies [64], but such approaches return us to the basic question of what physical role surfactant is really playing at the alveolar surface.

KEY WORDS
Lung: surfactant.

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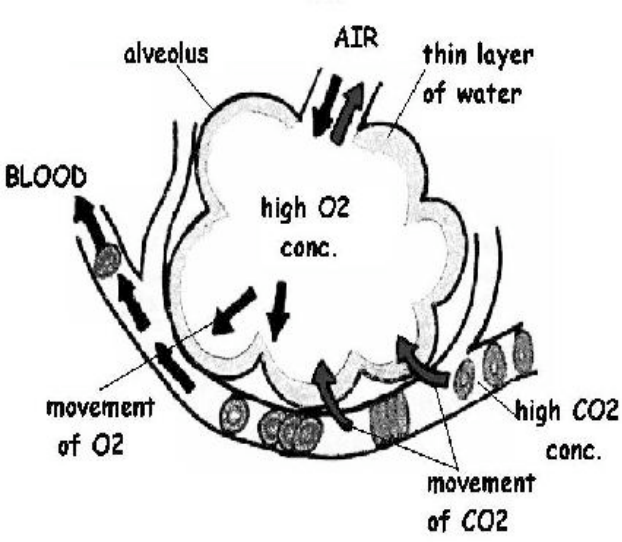
So now, examine respiration in more detail

Normal Healthy Kitten

At birth, the kittens are blind and deaf.

During the first three days of development, the alveoli express limited movement in terms of expansion and contraction during inhalation and exhalation. It is not required to fully expand and contract the lungs as the animal is still semi-conscious and the lungs themselves are still developing. In motion, the tips of the alveoli do not come into close proximity of their neighbours.

As the kitten develops, respiration increases and the expanded and contracted movement of the alveoli allow these to come into close contact with each other.



Remember the physics earlier and the properties of water and surface tension. On healthy kittens, the water cushion, protecting the alveoli is covered in surfactant and is not affected by this action. Gas exchange occurs and the kitten begins to develop normally.

For gas exchange to occur, the small air sacs within the lungs (alveoli) must remain open and filled with air. Alveoli are kept open by the elastic structure of the lung and by a thin layer of water coated with surfactant. Surfactant counters the natural tendency of the alveoli to close (collapse).

So can Surfactant Deficiency lead to FCKS?

The lining of the alveoli still has the water cushion but not the surfactant layer

Birth , day 1, day 2…..Respiration is slow see above

Day 3 respiration increases and the alveoli move extensively. On exhalation, they come very close to one another. The water cushion on adjacent alveoli is subject to surface tension movement. The water creates a larger single cushion exposing alveoli and drying them out. On inhalation, the dried out alveoli are no longer able to hold air, so they remain closed. The more alveoli that are closed, the less gas exchange occurs. The less gas exchange the more distressed the kitten becomes and the greater the rate of respiration. In humans, the medical condition is called atelectasis. Accordingly, atelectasis can decrease the level of oxygen in the blood. The body compensates for a small amount of atelectasis by constricting the blood vessels in the affected area. This constriction redirects blood flow to alveoli that are open so that gas exchange can occur.

So what causes the flat chest? With a large vacuum in the chest, following inhalation, there is an imbalance of pressure in the thoracic region. The ribs are trying to expand in reaction to the diaphragm moving. Atmospheric pressure is placed on rib cage. The pressure affects the weakest part of the chest wall and in the developing kitten that is the costochondral junction. This presents itself as a flattening of the rib cage along the junction and across the sternum. This is then the cause of flat chest in kittens with deficient surfactant covering alveoli.

So is this hypothesis founded; and could atelectasis be a related cause of FCKS?

There are three possible answers here.

1. This is complete hogwash. Ok surface tension and atelectasis is factual but really, this has no basis in the real presentation of FCKS. Well at least give this hypothesis a little house room before completely dismissing it.

2. OK so you have produced some evidence that may hold water..ish. But what about the use of splints on some kittens suffering from FCKS. Well there is the possibility that pressure exerted by the splint is sufficient t to break the surface tension of the larger water droplet allowing the droplet to split and cover alveoli letting them fill with air and allowing respiration to take place. It would be almost impossible to determine the exact external pressure necessary to break the water droplets into smaller parts to cover alveoli to allow respiration to take place and this splint is more a case of luck than judgement.

3. Ok so maybe this could be the situation. Now what? How can we prevent treat and possibly cure the condition if and the emphasis is on IF the condition is caused by this deficiency.

Prevention of surfactant deficiency..

When is surfactant produced in the embryo? Surfactant production in humans begins in Type II cells during the terminal sac stage of lung development. It is known as **Arachidonic acid** (AA, sometimes ARA) which is a polyunsaturated omega-6 fatty acid20:$4(\omega\text{-}6)$.

Arachidonic acid in the human body usually comes from dietary animal sources—meat, eggs, dairy—or is synthesized from linoleic acid.

Arachidonic acid is one of the essential fatty acids required by most mammals. Some mammals lack the ability to—or have a very limited capacity to—convert linoleic acid into arachidonic acid, making it an essential part of their diet. Since little or no arachidonic acid is found in common plants, such animals are obligate

carnivores; the cat is a common example, A commercial source of arachidonic acid has been derived, however, from the fungus *Mortierella alpina*.

This fatty acid is really Omega 6.

I feed my cat food with Omega 6. Of course, you do, but is it in the right form and at the right time to prevent deficiency? Many dieticians related Omega 3 and Omega 6 proportions to human dietary requirements. We are talking about cats and they have a different requirement.

Some cats may have a poor metabolism rate and require large amounts of arachidonic acid. In addition, a lot of processed food lacks omega 6 in a good form, remember the 1970s and taurine. Should we consider giving queens an omega 6 supplement at the right time of pregnancy? The supplement must be given in a form that the cat can digest and absorb.

Treatment

What about using an oxygen tent to substitute the deficient oxygen for 3 day old kittens?

Absorption Atelectasis

The atmosphere is composed of 78% nitrogen and 21% oxygen. Since oxygen is exchanged at the alveoli-capillary membrane, nitrogen is a major component for the alveoli's state of inflation. If a large volume of nitrogen in the lungs is replaced with oxygen, the oxygen may subsequently be absorbed into the blood reducing the volume of the alveoli, resulting in a form of alveolar collapse known as absorption atelectasis.[1]

Other treatment for affected kittens

Synthetic pulmonary surfactants

1. Exosurf - a mixture of DPPC with hexadeconal and tyloxapol added as spreading agents
2. Pumactant (Artificial Lung Expanding Compound or ALEC) - a mixture of DPPC and PG
3. KL-4 - composed of DPPC, palmitoyl-oleoyl phosphatidylglycerol, and palmitic acid, combined with a 21 amino acid synthetic peptide that mimics the structural characteristics of SP-B.
4. Venticute - DPPC, PG, palmitic acid and recombinant SP-C

Animal derived surfactants

1. Alveofact - extracted from cow lung lavage fluid
2. Curosurf - extracted from material derived from minced pig lung
3. Infasurf - extracted from calf lung lavage fluid
4. Survanta - extracted from minced cow lung with additional DPPC, palmitic acid and tripalmitin

Exosurf, Curosurf, Infasurf, and Survanta are the surfactants currently FDA approved for use in the U.S.[6]

1. White, Gary C. (2002). *Basic Clinical Lab Competencies for Respiratory Care, 4th ed.*. Delmar Cengage Learning. p. 230. ISBN 978-0766825321.