

A heuristic point of view on the breathing in the corona virus environment – A REVIEW

Emanuel Gluskin

Engineering faculty,

Ruppin Academic Center, Emek Hefer, 40250 Israel.

emanuel15@bezeqint.net

Abstract: It is a mini-review of a study related to the breathing of a human having the corona virus disease. The motivating arguments and two essential results of the "macroscopic" analysis are carefully discussed. The weak points of the formal constructions are stressed in order to encourage a teacher to suggest good topics (some of which are formulated here) for students' projects, which creates some pedagogical slant.

Keywords: Corona Virus Disease; Human behavior; Breathing (Respiration); Balance Equation; Medical Treatment.

1. Introduction

The intention of [1-3], completed by the present discussion, is to check whether or not it is possible to analyze something related to the corona virus disease [4-7] in simple equational terms. In the view of the problem of human behavior, we focus at the role of breathing (respiration) in the coronavirus environment.

Consider the breathing of an *already ill* man. There are breaths and exhalations, whose roles are very different, because the averaged spatial concentrations of the CVM (Corona Virus Molecules) -- those inside the thorax and those in the surrounding space -- are very different. The concentration inside is denoted as n , and that outside as n_o . Obviously,

$$n \gg n_o, \quad (1)$$

and we shall even set $n_o = 0$, so that the expression for the flow related to the breath, including n_o as a factor, disappears from the balance equation ((9) below) for CVM.

When analyzing the breathing, one should be careful with the use of the concept of the lung's volume (V). Human's interaction with the present in the air CVM is, first of all, via lung's surface that has a very complicated form. It may be even said that the lung fills a significant part of the internal space (volume) of the thorax *with its surface*, and one, motivated by the known from topology concept of Jordan' curve, can speak about "Jordan surface". The structure of the lung is even so complicated, that complete breath requires some diffusion [8] of the air into the twists (convolutions, slots) of the lung. If, furthermore, the CVM, absorbed by the surface of the lung, bother the oxygen molecules to be absorbed, then the theory of percolation [9] that considers the physics of the very long paths, should be involved in order to define the time delays of the propagations of the molecules of the two group. Contrary to diffusion, percolation is a threshold process (of the type of phase transition) which would mean here that the absorption of the oxygen molecules by the lung can be interrupted, meaning very quick death. However, with our macroscopic outlook, we cannot analyze here any such "theory in depth".

In terms of some averaged spatial distribution, the connection between the total number N of the CVM in the thorax, and the spatial density n can be taken as

$$n = \frac{N}{V}. \quad (2)$$

Because of the breathing, the volume oscillates:

$$V(t) = V_o + \varepsilon(t) \quad (3)$$

where we can set $\varepsilon(t) = \varepsilon_o \sin(2\pi At)$, assuming that the oscillations are significantly smaller than the average volume, $\varepsilon_o \ll V_o$. One can estimate ε_o as $V_o / 4$. The frequency of the breathing A is the main parameter here.

We can find $n(t)$ from (2) after finding $N(t)$. The latter is done if we write the balance equation for $N(t)$, which together with (2) represents a coherent, easily solvable, system of equations.

Despite the simple mutual connection, the physical roles of N and n are distinct. While the balance equation must be written for N , n is a much more suitable parameter for the physical and visuality considerations, and any thinkable measurement here is directly in terms of n . It is like the fact that humidity of the breathing can be measured via the exhalation, not via the never known whole amount of the water in the thorax.

2. The problem of the size

Obviously (see Fig.1):

$$\frac{dN}{dt} = -(\text{the flow out}) + (\text{the source inside}), \quad (4)$$

while both of the terms in the right-hand side are directly proportional to n . The first term is

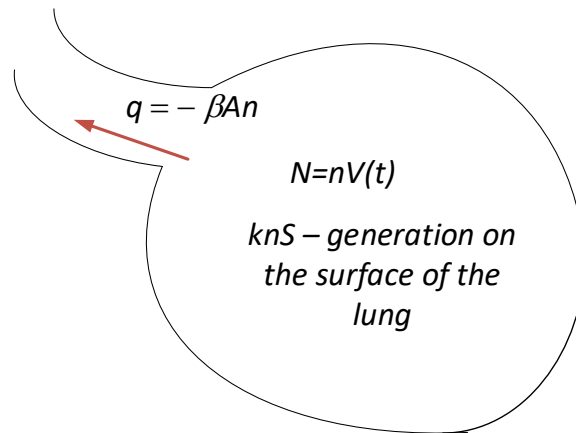
$$q_{out} = -\beta An \quad (5)$$

with a constant β such that the physical dimension of βA is that of velocity. Regarding q_{out} , it was already said that because of the relative smallness of the outside concentration n_o , this term is much more significant than the input breath-flow $q_{in} = +\beta A n_o$. That the velocity of each of the flows is directly proportional to the frequency of breathing A , is obvious.

As for the source-term in (4), we take it as

$$kSn \tag{6}$$

with a constant k . The direct proportionality here to the surface area S of the lung is natural, because the reproduction of the CVM – the essence of the illness -- takes place just on the surface.



$$dN/dt = knS - \beta An$$

$$A > kS/\beta$$

Fig 1. An illustration of the main equational terms; "q" here is " q_{out} "

As regards factor n in (6), -- we know from the theory of the ideal gas, that the gas pressure p is the transfer of the impulse \vec{p} of the molecules to the walls of the cavity (with the reflection, $\vec{p} \rightarrow -\vec{p}$, i.e. the change of the impulse is $\Delta\vec{p} = -2\vec{p}$), which is a direct interaction with the wall. This interaction results in the well-known formula for the pressure p of the gas

$$p = nkT \quad (7)$$

with the direct proportionality to n . Since in our case also, there is a direct interaction of the CVM with the surface of the lung, we use n in (6) by analogy. However, there is an argument, as follows, which leads to another dependence on n

Consider a small cube, whose volume, *in terms of* n , is just n , which touches the surface of the lung with its side. Obviously, the value of cube's side (i.e. of the touch), the square, is

$$(n^{1/3})^2 = n^{2/3}. \quad (8)$$

If to assume that the area of the touch is important, one could expect that instead of n in (6), $n^{2/3}$ should appear. This mathematical complication is put aside. A student project can be to return to the Boltzmann's theory of gases, and check whether or not for any form of the wall some geometrical averaging returns us to n

Using (5) and (6) in (4), we obtain

$$\frac{dN}{dt} = -\beta An + kSn = -Pn \quad (9)$$

with

$$P = \beta A - kS.$$

Now, using (2) and (3), and using that, because of the smallness of $\varepsilon(t)$,

$$n = \frac{N}{V(t)} = \frac{N}{V_o + \varepsilon(t)} \approx \frac{N}{V_o} \left(1 - \frac{\varepsilon(t)}{V_o}\right) \quad (10)$$

with the error of order

$$\left(\frac{\varepsilon(t)}{V_o}\right)^2,$$

we obtain (9) as

$$\frac{dN}{dt} \approx -\frac{P}{V_o} \left(1 - \frac{\varepsilon(t)}{V_o}\right) N(t). \quad (11)$$

This linear time-variant equation is easily solved by separating the variables:

$$N(t) = K \exp \left\{ -\frac{P}{V_o} \int_0^t \left[1 - \frac{\varepsilon(\lambda)}{V_o}\right] d\lambda \right\} \quad (12)$$

with a constant K . Considering again the smallness and the oscillatory nature of $\varepsilon(t)$, we observe from (12) that $N(t)$ is mainly proportional to the factor

$$e^{-\frac{P}{V_o} t}.$$

This factor obviously requires $P \geq 0$, that is

$$A \geq \frac{kS}{\beta}. \quad (13)$$

The opposite inequality would result in the tragedy of $N \rightarrow \infty$, as $t \rightarrow \infty$; thus (13) can be named *the survival condition*.

Inequality (13) is an important result of our research, and it deserves thorough heuristic discussion as regards theoretical applications and instructive conclusions.

3. The discussion of (13)

1. Physical weakness, expressed in a noticeable decrease in A , compared to the usual frequency of breathing, is absolutely unacceptable for one having the CV disease. Being born in Russia, I saw many very small apartments, so that the air in the room was too close (stuffy). Obviously, there was a shortage of oxygen, but since we exhalate about 16% of the oxygen that was taken with the previous breath, some oxygen always remains in the air, and in order to take it, the people simply (inevitably) have to develop some strong breathing, which means, in particular, a sufficiently high A . I would say that the possession (development) of a high A is more important than having fresh air with a lot of oxygen, since the Russian guys grown up in such poor conditions earn many Olympic medals, i.e. can be very healthy ... To our point, it is suggested to critically consider the hurry in giving oxygen to an ill man, because just some shortage of oxygen could cause (force) the patient to intensively (quickly) breath – which should cause better cleaning the lung from the CVM, according to (13). A strong patient may be even given air supply with an intentionally reduced percentage of the oxygen (just add some N_2 to the air, while keeping its pressure) for the stimulation purpose. Of course, this has to be done under careful individual treatment and continuous observation, for not to danger the life, but we do have to distinct between the necessity to save one in a critical state, and the necessity to strengthen him -- for him not to return to the hospital with the same trouble.
2. The death of the huge-size animals, like dinosaurs, during the "Ice Period" is explained by the cold. We can assume, however, that the cold was not the only cause. The viruses in the air, and the insufficiently high A of the breathing of the giants (consider (13) for a very large S), which could not free the animals' lungs from the virus, could also cause the death. The nature of the environment should be carefully discussed here. High humidity

contributes to spreading and holding viruses in the air. The cold generally reduces the humidity, but not sufficiently everywhere, and there could be some, -- optimal for a pandemic -- conditions when both the cold and the humidity are significant, bringing together the harm.

3. Generally, (13) shows advantage of the small-size creatures, because small S makes it easy to realize the inequality. Thus, a (healthy) child, bird, or a mouse have good chances not to become ill with the coronavirus. As the matter of fact, already such an average-size creature as the dog demonstrates a relatively quick breathing and does not become ill with the corona virus. The tendency of some mothers to feed their children very well -- for them to become tall and wide -- which means a large S -- should *not* be encouraged. The discoveries of the armors of knight had shown that -- contrary to one's romantic opinion -- the knights were, as a rule, *lower* (shorter) than we are. At the same time, sport and the associated development of the respiration are, of course, very helpful. Similarly to the fact that our eye is most sensitive (adjusted) just to the green light that is in the maximal of the sunlight spectrum, our size and weight are also made optimal by the nature.

4. Do can we cause an antagonism between the CVM, allowing them to kill each other?

This is a topic for further development, which requires, in particular, the equational outlook to be continued. The initial idea was to use that the CVM are "blind", assuming that the molecules can attack each other, just as they can attack the lungs' cells. If this would be so, then it would be possible, as was assumed, to insert, just before the artificial ventilation of the lungs, many other CVM, causing all of the CVM present in the thorax -- *because of their high concentration* -- to *collide*, attack and kill each other. By controlling (measuring) $n(t)$ that then should reduce in time, the moment to start the ventilation should be determined.

This idea means, in fact, some very primitive imagining CVM as billiard balls that can collide with each other just as they can collide with the wall, and was immediately found to be too simple for the tough reality. A physician (see the Acknowledgements) explained me that the lung's surface is covered by some protein

layer (itself very complicated and many-functional) which covers (catches) the coming CVM whose subsequent self-reproduction is then both due to the molecules themselves and the material of the layer. Thus, the collisions of CVM with the lung surface and their mutual collisions are (would be) not at all the same.

It became clear that in order to allow effective antagonism between CVM, some of them (those that we specially insert into the thorax) have to receive – by some special treatment -- a cover that would allow the CVM already present in the thorax to become "glued" to the inserted CVM. A preliminary laboratory experiment in which this cover could be sprayed onto a dense ensemble of CVM, is required.

Finding such covering material is a matter of biology, meanwhile we continue with the equational side in this direction.

5. The linear time-dependent equation

If we consider collisions of similar molecules belonging to an ensemble with the spatial concentration n , then the probability of the binary collisions is $n \cdot n$, and if these collisions result in a *decrease* of the concentration of CVM because of the "killing" by the molecules each other, then a nonlinear term of the type

$$-\gamma n^2$$

with a positive constant γ , arises in the balance equation. However, if we actually generate an air pulse with the specially covered molecules, then this pulse *is a known function*. Denoting it as $n^*(t)$, we would have in the balance equation not the above nonlinear term, but the *linear time-variant* term

$$-\gamma n^*(t) \cdot n.$$

For this situation, the ballast equation becomes (n_o is still ignored, and P is as in the above):

$$\frac{dN}{dt} = -Pn - \gamma n^*(t) \cdot n, \quad (14)$$

or

$$\frac{dN}{dt} = -[P + \gamma n^*(t)] \cdot n \quad (15)$$

Using (3), we obtain from (15):

$$\frac{d[(V_o + \varepsilon(t)) \cdot n]}{dt} = -[P + \gamma n^*(t)] \cdot n \quad (16)$$

or

$$(V_o + \varepsilon(t)) \frac{dn}{dt} + n \frac{d\varepsilon}{dt} = -[P + \gamma n^*(t)] \cdot n, \quad (17)$$

which yields

$$\frac{dn}{n} = - \frac{\frac{d\varepsilon}{dt} + P + \gamma n^*(t)}{(V_o + \varepsilon(t))} dt, \quad (18)$$

and using (10)

$$d(\ln n) \approx - \frac{1}{V_o} \left(1 - \frac{\varepsilon(t)}{V_o}\right) \left(\frac{d\varepsilon}{dt} + P + \gamma n^*(t)\right) dt. \quad (19)$$

Finally,

$$n(t) \approx K \exp \left\{ - \frac{1}{V_o} \int_0^t \left[\frac{d\varepsilon}{dt} - \frac{\varepsilon}{V_o} \frac{d\varepsilon}{dt} + P + \gamma n^*(t) - P \frac{\varepsilon(t)}{V_o} - \frac{\varepsilon(t)}{V_o} \gamma n^*(t) \right] dt \right\}$$

(20)

The degree of the exponent includes the terms:

$$\frac{-P}{V_o}t, \frac{\varepsilon(t) - \varepsilon(0)}{V_o}, \frac{P[\varepsilon(t) - \varepsilon(0)]}{V_o^2}, \frac{\varepsilon^2(t) - \varepsilon^2(0)}{2V_o}, -\frac{\gamma}{V_o} \int_0^t n^*(t) dt, \frac{\gamma}{V_o^2} \int_0^t \varepsilon(t) n^*(t) dt \quad (21)$$

Since $n^*(t)$ is *compactly supported* (nonzero only in a finite interval), and $\varepsilon(t)$ finite, if P is nonzero, then $\frac{-P}{V_o}t$ is the only expression here that infinitely increases in time, and all the others are not so important. Thus as $t \rightarrow \infty$ we remain with the result of $n(t)$ and $N(t)$ be directly proportional to $e^{-\frac{P}{V_o}t}$.

However, at the limiting (formally permitted) "balanced" case of $P = 0$, the value of

$$\frac{\gamma}{V_o^2} \int_0^t \varepsilon(t) n^*(t) dt \quad (22)$$

and all the other nonzero terms become important as defining the stationary, almost constant $n(t)$ and $N(t)$.

Denoting the integral value of the pulse $n^*(t)$ as N^* , we have, for the time when the pulse is already finished

$$-\frac{\gamma}{V_o} \int_0^t n^*(t) dt \equiv -\frac{\gamma}{V_o} N^*.$$

The situation of (22) is also simple, since the inserted in the thorax pulse $n^*(t)$ should be naturally correlated with $\varepsilon(t)$ -- it is most simple to insert the covered molecules with the breath. Thus, we can take the compactly supported $n^*(t)$ directly proportional to $\varepsilon(t)$ at some half period of the $\varepsilon(t)$ -- this time-interval is physically suitable.

Calculation of all of the terms in (21) is very easy. On the physical side, the measurement of the *established* value of n also should be relatively easy.

6. Some research targets

1. To carefully consider the points of Section 2, and to formulate relevant students' research projects.
2. To develop methods for an easy measurement of n (perhaps, in the exhaled air).
3. To develop the cover material for the CVM, considered for a use in Section 4.
4. To develop a method for creation ensembles of CVM with prescribed n for preliminary experiments with the cover.
5. To enhance (improve) the theory of Section 5. The immediate improvement is taking one more term in the expansion of $1/V$, that is, using

$$\frac{1}{V_o + \varepsilon(t)} \approx \frac{1}{V_o} \left[1 - \frac{\varepsilon(t)}{V_o} + \frac{\varepsilon^2(t)}{V_o^2} \right] . \quad (23)$$

As is shown in the next section, this correction leads to a small addition to the degree in the main factor

$$e^{-\frac{P}{V_o} t} .$$

6. To try to connect our results with the theory of diffusion regarding the processes in the thorax.
7. The same regarding the theory of percolation.
8. To support the choice of n , and not $n^{2/3}$, in (6).

7. The use of (23)

Let us rewrite (19), using (23)

$$d(\ln n) \approx -\frac{1}{V_o} \left[1 - \frac{\varepsilon(t)}{V_o} + \frac{\varepsilon^2(t)}{V_o^2} \right] \left[\frac{d\varepsilon}{dt} + P + \gamma n^*(t) \right] dt \quad (24)$$

The most essential term, appearing here after integration, still is $-\frac{P}{V_o}t$. However,

there is now one more term that also, though slower, infinitely increases with time.

Integrating

$$\begin{aligned} \int_0^t \left[-\frac{1}{V_o} P \frac{\varepsilon^2(t)}{V_o^2} \right] dt &= -\frac{P}{V_o^3} \int_0^t \varepsilon^2(t) dt = \\ &= -\frac{P \varepsilon_o^2}{V_o^3} \int_0^t \sin^2(2\pi A t) dt = -\frac{P \varepsilon_o^2}{2V_o^3} \int_0^t (1 - \cos(4\pi A t)) dt \end{aligned}$$

and using that the average of $\cos(4\pi A t)$ is zero, we obtain the here important part as

$$-\frac{P \varepsilon_o^2}{2V_o^3} t.$$

Together with $-\frac{P}{V_o}t$ in the degree, the essential factor in $n(t)$ and $N(t)$ now is

$$e^{-\frac{P}{V_o} \left(1 + \frac{\varepsilon_o^2}{2V_o^2} \right) t}. \quad (25)$$

Even though $\varepsilon_o < V_o$, the added degree need not be not ignorable; it may be several percentages. The condition $P > 0$ remains the main one.

8. Conclusions

The topic of breathing of a human, having the corona virus disease, is heuristically interesting, and our simple analytical tools and physical arguments are useful. Even though we have considered the process of the breathing very "macroscopically", some unexpected logical and physical connections are revealed on the way of the research. It is important that such results should be relevant for any mutation of the CVM. The frequency of breathing is a threshold parameter for survival. For large creatures, it must be correspondingly large, which hardly takes place in reality. The idea to cause an antagonism between the corona virus molecules for them to kill each other, presents, at this stage, just a line of thought, but I find also it fruitful. There is the hope that researches, -- even professional biologists and physicians, -- will find helpful for them suggestions in the discussion.

Acknowledgements

I am grateful to MD Eitan Gluskin for explaining me (Section 6) the role of the lung's cover in the CV disease.

References

1. Emanuel Gluskin, "Some simple equations for the analysis of the CoronaVirus Disease", *Universal Journal of Mathematics and Mathematical Sciences*, vol. 13, no 2, 2020, pp. 97-104
(<http://dx.doi.org/10.17654/UM013020097>)
2. Emanuel Gluskin, "A way to fight corona virus", viXra:2010.0235 (Oct. 2020)
3. Emanuel Gluskin, "A comment on the breathing in the Corona Virus environment", *Circuit Theory and Applications*, DOI: 10.1002/cta.2934.

3. S. Mangiarotti, M. Peyre, Y. Zhang, M. Huc, F. Roger, and Y. Kerr, "Chaos theory applied to the outbreak of Covid-19: An ancillary approach to decision-making in pandemic context," *Epidemiology & Infection*, vol. 148, no. 10.1017/S0950268820000990, pp. 1–29, 2020
4. S. Mangiarotti, M. Peyre, and M. Huc, "A chaotic model for the epidemic of Ebola virus disease in West Africa (2013–2016)," *Chaos: An Interdisciplinary Journal of Nonlinear Science*, vol. 26, no. 11, p. 113112, 2016
5. Seyed M. Moghadas, Meagan C. Fitzpatrick, Pratha Sah, Abhishek Pandey, Affan Shoukat, Burton H. Singer, and Alison P. Galvani, "The implications of silent transmission for the control of COVID-19 outbreaks" *Proceedings of national Academy of Science* (PNAS_ July 28, 2020 117 (30) 17513-17515; first published July 6, 2020; <https://doi.org/10.1073/pnas.2008373117>
6. Cao, X. "COVID-19: immunopathology and its implications for therapy". *Nat Rev Immunol* **20**, 269–270 (2020). <https://doi.org/10.1038/s41577-020-0308-3>
7. Gbenemene Kpae, "Impact of Covid-19 on Society, Economics and Education of Nigeria", *International Journal of English Literature and Social Sciences*, 5(5) Sep-Oct 2020; Available online: <https://ijels.com/> ISSN: 2456-7620 <https://dx.doi.org/10.22161/ijels.55.66>
- [8] L.D. Landau and E.M. Lifshitz, "*Fluid Mechanics*", Pergamon, New York, 1959.
- [9] A.L. Efros, "*Physics and Geometry of Disorder: Percolation Theory*" (Science for Everyone)", Nauka, Moscow, 1982.