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Why do we yawn?*

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ABSTRACT

The biomedical hypothesis proposed here is that the immediate trigger for a yawn is a restricted collapse of a few alveoli in the lungs. The extent of this alveolar collapse may be too small for it to be detected by current X-ray technology, but this technology is continually improving and may soon be good enough to test the hypothesis. In support of the hypothesis, it is shown that yawning can be inhibited by deep breaths of air, nitrogen or carbogen, thus showing that yawning is not triggered by lack of oxygen or by excess carbon dioxide, leaving alveolar collapse as the most likely possibility. A more extensive form of alveolar collapse is termed atelectasis and this involves a serious state of hypoxia which, if deepened or prolonged, can be fatal. Therefore, if the hypothesis is correct, yawning may prevent the development of atelectasis and save lives. This paper is not concerned with other indirect ways in which yawning may be induced, nor with the mechanism and neural circuitry of the yawn, nor with social aspects of yawning, only with the immediate trigger. My aim is to get better evidence for the hypothesis put forward here and also to study the behaviour of the pulmonary alveoli in normal respiration.

Keywords: Yawn; Alveolar Collapse; Shallow breathing; Surfactant

1. INTRODUCTION

A yawn comprises an initial deep intake of breath which is then held for several seconds, after which there is a slower passive expiration. Over two or three millennia, this behaviour has intrigued and puzzled most people. Why do we yawn? A recent publication provides a good summary of current research and opinions on yawning [1]. However, in spite of much research over many years, there is no agreement as to the immediate stimulus triggering a yawn. This is partly because a yawn is commonly associated with a variety of conditions such as tiredness, boredom, sleepiness, loss of attention, fatigue, hunger, malaise and, most surprisingly, observing other people yawning (contagious yawning). Some other occasions when yawning occurs will be mentioned in the discussion. This suggests that there may be several triggering stimuli. In spite of such views, I have made an assumption that there is a common trigger for the yawn in most of these conditions in spite of their apparent differences. A particular problem in the study of yawning is that, unlike many other reflexes, a yawn is not easily and reliably triggered. An exception to this statement is contagious yawning and also yawning by thinking about it and taking a deep breath. However, it is not clear that such "copy" yawns are identical with the naturally occurring yawn. One way around this difficulty is to choose a period of the day when yawns occur reliably. Two such periods are the period of about an hour just before sleep and the hour just after waking [2,3]. I have found the latter period to be the more reliable and all my experiments have been based on this condition. I wish to put forward as a possible biomedical hypothesis that the trigger for a yawn is a collapse of some pulmonary alveoli.

2. METHODS

Yawning is often accompanied by stretching of limbs and other parts of the body (pandiculation). I have not made any detailed observations in this area, primarily because of the difficulty of quantifying this activity. With regard to yawns, the suggestion was made by Cahill [4] that an induced yawn should be used to alleviate

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atelectasis (alveolar collapse) in patients unable to take deep breaths because of post-operative pain. There is reason to think that this procedure would be helpful and therefore the relationship between atelectasis and yawning might be a causal one. One way to test this idea is to study the effect of deep breaths on yawning.

After some preliminary investigations I realized that a suitable choice of control yawns would be those occurring immediately after rising from bed in the morning, I am awoken each morning by an alarm clock at 7.00 am. I normally get up immediately and count the number of yawns between 7.00 am and 7.15 am. During this time I am performing various toilet functions. If I depart from this procedure to a significant degree I do not use these data. On certain days I commence my awake session by taking five deep breaths of either room air, nitrogen, or carbogen (5% carbon dioxide, 95% oxygen), holding my breath for about 5 seconds. I then count the number of yawns in the following 15 minutes. The gases, except the room air, were contained in Douglas bags. Supplies of nitrogen and carbogen were obtained from Coregas, Villawood, NSW, Australia. I report a large variance in the occurrence of yawns but at present I do not have a good explanation for this variability.

Statistical comparisons between groups were made using the Mann-Whitney test, accepting significance at $P \leq 0.05$.

3. RESULTS

3.1. Experiment 1. Choice of Control Yawns

I found that I yawn an average of about 3 times in the 15 minutes from 7.00 to 7.15 am. In **Table 1** are shown the results from two periods obtained in this way, one from 9 successive days, the other from 12 days. The number of yawns per day varies from 0 to 7. There was no significant difference between the two periods (Mann-Whitney; $P = 0.2243$). On other occasions (see Experiments 2 - 4, 6) the control yawn counts were similar and not significantly different from one another, so this procedure provides a satisfactory basis for control responses.

3.2. Experiment 2. Effect of Deep Breaths on Yawning

There are several possible causes of yawning. The most likely are: lack of oxygen, excess of carbon dioxide and collapse of the lung alveoli. As explained in the Methods section, I investigated these possibilities by taking deep breaths. In this experiment immediately on rising I took 5 deep breaths (of air), holding my breath for about 5 seconds on each occasion. I then counted the number of yawns in the following 15 minutes. On alternate days I began with deep breathing and on the other days I made no deep breaths. The results are shown in **Table 2**. The days are consecutive but on some days no observations were made for various reasons, such as a common cold, an interrupted sleep or absence from home. It is clear that the deep breathing almost abolishes yawning, with a highly significant difference between the control and experimental observations (Mann-Whitney: $P = 0.0002$). On a few occasions in both the control and the experimental data I noticed very weak yawns

Table 1. Number of yawns in morning period.

Day	1	2	3	4	5	6	7	8	9	10	11	12	Mean	Mean of all
	3	4	2	6	0	2	1	3	1				2.44	2.90
	3	3	2	3	4	2	3	4	1	7	4	3	3.25	

Two separate periods, one of 9 days, one of 12 days, in which the number of yawns between 7.00 am and 7.15 am was observed. The distributions are not significantly different (Mann-Whitney: $P = 0.2243$). Thus, the procedure is a suitable one for control responses; see also the other Tables.

Table 2. Number of yawns in morning period: Effect of deep breaths on yawning.

(a)

Day	1	2	3	4	5	6	7	8	9	10	17	18	19	20	21	22	23
C	4		2		5		1		2		(2)	6		6		1	
E		0		0		(3)		0		(1)			(1)		0		0

(b)

Day	24	25	26	27	28	29	31	32	33	34	35	36	Mean	*P*
C	4		5		0		1		2		2		2.80	0.0002 MW
E		1		0		1		1		0		0	0.39	

C: control (spontaneous). E: experimental (each session commenced with 5 deep breaths (of air), each held for about 5 seconds). MW: Mann-Whitney test. Note, although the days are consecutive, days are omitted when no observations could be made on those days. The figures in brackets are 'sighs' (mot full yawns) and are divided by 2 for mathematical and statistical purposes.

(figures in brackets), which I call "sighs". For all the mathematical and statistical calculations I divided these figures by 2. I did not study the number of conditioning breaths taken in any detail. I think 4 breaths might have a weaker effect than 5 breaths and that 6 breaths may not be any stronger than 5 breaths.

3.3. Experiment 3. Could Yawning Be Due to Hypoxia

The experiment described in Experiment 2 was repeated but, instead of taking deep breaths of air, these breaths were of pure nitrogen. If yawning is triggered by hypoxia, it should not be relieved by nitrogen, indeed it might be intensified. The results, as shown in **Table 3**, are similar to those in **Table 2** and indicate a virtual abolition of yawning. There is a highly significant difference between the control and experimental data (Mann-Whitney: $P < 0.0001$). There is no support here for the idea that hypoxia might cause yawning.

3.4. Experiment 4. Could Yawning Be Due to Hypercapnia

Could yawning be caused by accumulation of carbon dioxide in the lungs or tissues?

Experiment 4 is similar to Experiment 3 except that, instead of taking deep breaths of nitrogen, I took deep breaths of carbogen (in this case 5% carbon dioxide, 95% oxygen). The results are shown in **Table 4**. Again, the effect of the deep breaths is to almost abolish the yawning. The difference between the controls and the experimental data is statistically highly significant (Mann-Whitney: $P < 0.0001$). This result means that the possibility that yawning is due to an accumulation of carbon dioxide in the lungs or elsewhere can be discounted.

3.5. Experiment 5. Is Morning Yawning Dependent on the Transition from Sleep to Wakefulness?

There has been considerable interest in what changes occur in the metabolism of the nervous system in relation to yawning on awakening, and on the corresponding changes on falling asleep. This topic was recently reviewed by Giganti and colleagues [3]. However, I accidentally discovered that the subject is more complicated than a simple transit between sleeping and waking. On one occasion I found that, after waking but remaining in

Table 3. Effect of deep breaths of nitrogen on yawning.

(a)

Day	1	2	3	4	5	6	7	8	9	10	11	13	14	15	16	17	18	19
C	4		3		5		1		6		1		0		4		3	
E		0		0		0		0		0		0		0		0		0

(b)

Day	27	28	29	30	31	34	35	36	37	38	39	40	Mean	*P*
C	2		5		3		4		1		3		3.00	<0.0001 MW
E		0		0		0		0		0		0	0	

C: control, spontaneous yawns, unconditioned. E: experimental, session commencing with 5 deep breaths of nitrogen, each held for about 5 seconds. Note, although the days are consecutive, days are omitted when no observations could be made on those days. MW: Mann-Whitney test.

Table 4. Effect of deep breaths of carbogen on yawning.

(a)

Day	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
C	4		4		0		3		5		3		5		6		5	
E		0		1		0		0		1		0		0		1		1

(b)

Day	20	32	34	35	36	37	38	39	40	41	42	43	Mean	*P*
C	7		3		5		4		6		4		4,267	< 0.0001 MW
E		0		0		0		0		0		0	0.267	

C: control, spontaneous yawns, unconditioned. E: experimental, session commencing with 5 deep breaths of carbogen, each held for about 5 seconds. Note, although the days are consecutive, days are omitted when no observations could be made on those days. MW: Mann-Whitney test.

bed for more than 15 minutes, I did not yawn. Since I occasionally do not yawn in the first quarter-hour after waking and arising (see, e.g. all the previous Tables), it was necessary to check the reproducibility of this result. **Table 5** shows that absence of yawning on wakening occurs regularly if one remains in bed. It is possible that the horizontal position in some way inhibits or prevents yawning. Therefore it seemed worthwhile to remain in bed but to alter the position of the body.

3.6. Experiment 6. Effect of Sitting in Bed in the Morning

This experiment was like Experiment 5 but I remained in bed, sitting upright, for 15 minutes. On alternate days I rose and counted my yawns in 15 minutes as in Exprriment 1. The results are shown in **Table 6**.

There was no significant difference between the two sets of values. Thus, it appears that the vertical position of the chest facilitates the occurrence of yawns. The reason for this will be taken up in the Discussion.

4. DISCUSSION

4.1. Evidence for the Hypothesis

The results presented here are from a single person and are put forward as a hypothesis, not an unshakeable conclusion. It is essential that these or similar experiments be repeated in many more people in order to reach firm conclusions. Nevertheless, my results are in good agreement with previous work. For example, Cahill [4] recommended yawning to prevent the development of atelectasis in patients experiencing respiratory disorders or post-operative complications. It has also been suggested that other phenomena related to yawning, such as spontaneous deep breaths and inspiration-augmenting reflexes act to prevent or reduce atelectasis [5-8]. The use of Hyperinflation Therapy to prevent or treat atelectasis is a further indication that yawning is acting in a similar way. Reluctance to see that atalectasis could be the trigger for yawning probably stems from the belief that atelectasis is pathological. I am proposing here that mild atelectasis may be a normal feature of regular breathing.

4.2. Cause of Alveolar Collapse

Previous research has provided evidence that yawning is not triggered by either lack of oxygen in the blood or brain or an excess of carbon dioxide [9]. The experiments I have performed on myself support this conclusion. Therefore, the most likely explanation for my results is that yawning is due to a mild atelectasis, a collapse of a few alveoli in the lungs. This pulmonary collapse probably results from shallow breathing because it has been shown that low lung volumes and low transpulmonary pressures lead to alveolar collapse over time [10,11]. Shallow breathing, in turn, is due to a marked relaxation of the whole body such as may occur in all the states mentioned in the introduction. Such relaxation can occur even when the person is attending to a situation, such as a lecture, or when driving, or watching television. This conclusion is in disagreement with previous views. For example, yawning during these conditions is often regarded as due to boredom or loss of attention. I am suggesting that the cause is a mild atelectasis due to

Table 5. Number of yawns while lying in bed, awake, for 15 minutes before rising.

Day	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	Mean
	0	2	0	0	0	0	0	0	0	0	0	0	0	0	0	0.13

Table 6. Number of yawns while remaining in bed awake but sitting upright, for 15 minutes before rising.

(a)

Day	1	2	3	4	5	6	7	8	9	11	12	13	14	15	16	17	18
C	1		4		1		3		1		2		3		1		3
E		0		1		2		0		5		0		3		0	

(b)

Day	19	20	21	22	23	24	25	26	27	29	30	31	32	Mean	*P*
C		2		1		4		3		5		5		2.60	0.3719
E	4		7		0		2		0		5		4	2.20	MW

C: control (awake, as in **Table 1**). E: experimental (awake, sitting in bed). MW: Mann-Whitney test. Note, although the days are consecutive, days are omitted when no observations could be made on those days.

shallow breathing.

4.3. Significance of Yawning

What is the usefulness of yawning? It follows from the argument put forward in the above paragraphs that yawning should relieve atelectasis. The mild atelectasis I am proposing as a trigger for yawning is probably not a cause of serious changes in oxygen or carbon dioxide levels but could be if not relieved by yawning or deep breathing. Furthermore, yawning or deep breathing is more efficient at preventing atelectasis than in re-opening collapsed alveoli [10]. Also, it has been shown previously that installation of surfactant to reduce atelectasis attenuates bacterial growth and translocation, and prevents pneumonia [12]. So, it would seem that yawning could act similarly. Therefore yawning must be seen as an important reflex.

Clinical atelectasis occurs when the patient has difficulty breathing, for example, after a chest or abdominal operation and in other conditions such as in premature infants who may have inadequate surfactant. Surfactant reduces surface tension and enables the alveoli to remain patent. Hyperinflation therapy is widely used in such states and is very effective. There is an important connection between hyperinflation and surfactant because it is well established that stretching the alveoli releases surfactant. It has also been shown that surfactant is released from its cellular source, the alveolar epithelial type II (AEII) cells by direct stretch of these cells [13]. Furthermore, it has been shown that a variable stretch pattern is more effective in causing release of surfactant than a monotonous pattern [14]. Thus, the irregular pattern of yawning may very well have an unanticipated benefit in surfactant release.

4.4. Detection of Alveolar Collapse

The standard technique to detect atelectasis is chest X-ray but this technique is at present not sensitive enough to detect the degree of atelectasis that I am postulating. Nevertheless, considerable advances have recently been made in 3D X-ray microscopy and I understand it may soon be possible to reach a level of sensitivity to provide the evidence for my hypothesis. Later in the Discussion I mention the fact that alveoli in the basal regions of the lung are more likely to collapse than those in the apical regions. Thus, initial attempts to detect small areas of atelectasis should concentrate on basal regions. Meanwhile, probably the best indicator of alveoli collapse is the reduction in lung compliance. Mead & Collier [10], working with dogs, found that anaesthesia reduced lung compliance and this was reversed by hyperinflation, a result confirmed in humans by Bendixen and colleagues [15]. Furthermore, the post-mortem appearance of the dog lungs showed a clear atelectasis. I have not been able to measure lung compliance in myself at the time of my experiments. Pulmonary compliance must be distinguished from changes in compliance in the tracheobronchial tree, for example due to bronchospasm, bronchial oedema, secretions, ciliary action and airway narrowing [16]. However, if these factors can be controlled or eliminated, it would be a good project to try to relate yawning to lung compliance and, at a later time if the technology can be developed, to alveoIar collapse.

4.5. Central Nervous System Circuitry Associated with Yawning

In any experiment, it is always necessary to determine the immediate trigger for an effect. For yawning I am postulating that the immediate trigger is a collapse of some alveoli. This is just a hypothesis and, although plausible, more direct evidence must be sought. It is therefore important to be aware that there are many regions of the central nervous system that relate to yawning. The main regions are shown in the diagram in **Figure 1**. It is well accepted that there is a region in the brain stem that could be regarded as a "yawn centre" [17]. It follows from this that this region might be stimulated electrically or pharmacologically to produce a yawn. I am not concerned with this mode of action, only with what is the immediate trigger for a yawn in normal circumstances. I am also not concerned with the circuit involved in the execution of the yawn or with other secondary features of the yawn. It is possible that a more remote cause may lead to a collapse of alveoli and this then causes the yawn. I am not seeking to rule out such remote causes. Indeed, any activity or lack of activity that leads to shallow breathing will, I believe, cause yawning.

4.6. Clinical and Other Conditions Associated with Yawning

It is interesting that yawning commences in the human foetus at about the 13th week of gestation but decreases after about the 30th week [18]. This is the canalicular period during which there is a development of the alveoli, so yawning is closely related to alveolar development. It would not be expected that hypoxia or hypercapnia would be factors in this process. Of course, there is no air in the lungs in the foetus but there is movement of amniotic fluid through the mouth and into the airway during a yawn [19], so it can be presumed that yawning would inflate the alveoli via amniotic fluid. Surfactant is not generated until about the end of this stage. Therefore the yawns can be regarded as maintaining the patency of the alveoli during their development. The need for yawning is obviously reduced when surfactant appears. Thus, my hypothesis can be seen as applicable from the earliest

possible moment in life.

It is also clear from the early development of yawning that the higher neural circuitry shown in **Figure 1** is not needed for yawning.

It has been observed that paratroopers yawn before making their jump and musicians yawn before going on stage [20]. I suggest that in any tense state there is shallow breathing and this leads to a mild alveolar collapse.

Yawning is associated with many diseases, e.g. lesions of the brain stem, tumours, encephalitis lethargica, diencephalic seizures and chorea [21]. But, more significantly, in several conditions, excessive yawning is associated with atelectasis, e.g. asthma, cystic fibrosis and pulmonary embolism.

The so-called "contagious" yawning has aroused much interest and speculation as to its significance. However, the simple explanation is that this is merely an example of what we have all been doing since childhood, namely, copying what our parents, siblings and neighbours do. That is how we learn. This explanation was pointed out many years ago by Barbizet [21]. In any case, it is likely that the cerebral cortex is involved (**Figure 1**). The person who makes the first yawn probably has some slight atelectasis. However, in a group of people not everyone mimics the yawning, so it is possible that mimicking occurs only in those with some degree of alveolar collapse. According to the society in which we live, yawning may be acceptable or disrespectful.

Another way in which yawning is mimicked is among non-human primates who seem to use it as a threat [22]. Almost certainly there would be no atelectasis in this case. This yawn would be initiated from higher brain centres and the pathway would pass to the "yawn centre" to execute the yawn. Yawning has obviously evolved early in biological history and it is clear that, like many other reflexes, it could be used for functions other the innate purpose. The recent book on yawning edited by Walusinski [1] summarises these various adaptations

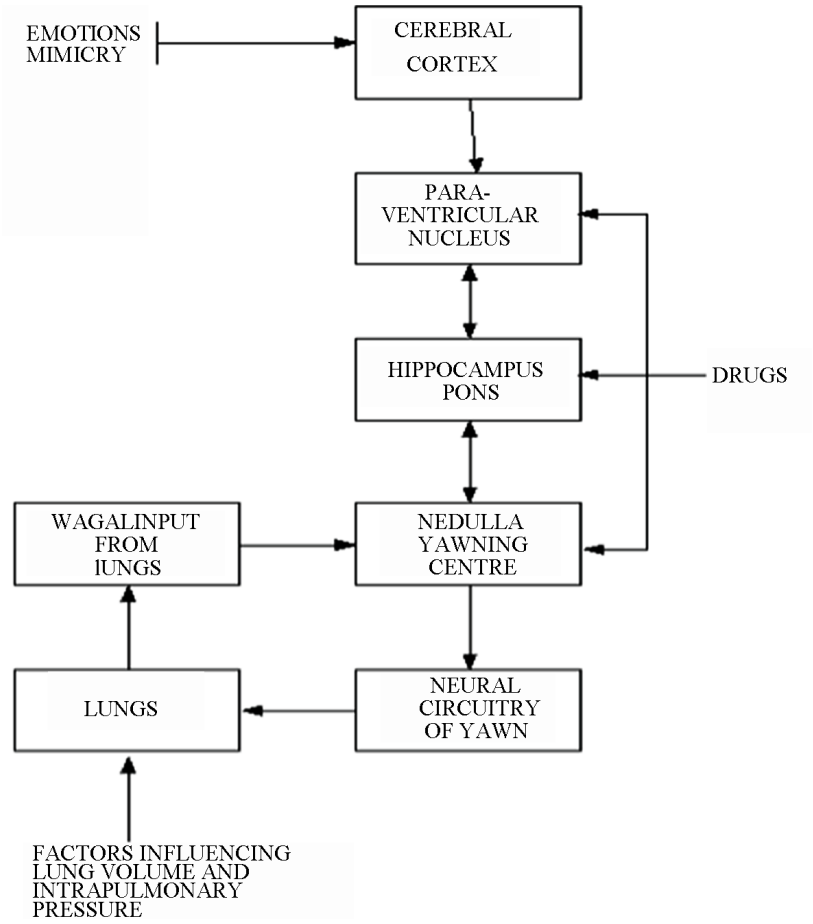

Figure 1. Diagram to show some parts of the brain associated with yawning, The straight lines between the boxes indicate functional connections but do not represent exact neural pathways. The present paper is concerned primarily with the circuitry at the base of the diagram involving the lungs and medulla. Details of the pharmacology of the higher centres may be found in the papers of Argiolas & Melis [30] and Collins & Eguibar [31].

very well.

4.7. Triggering Signal from Collapsed Alveoli

It is uncertain what signal comes from the collapsed alveoli to initiate the yawn. I assume that signalling nerves run in the vagus and there is a possibility that the relevant nerves may be those originating from the J-receptors [23]. These receptors lie in the interstitium of the alveoli and respond to conditions causing a decrease in oxygenation such as pulmonary congestion and oedema. Their excitation leads to an increase in ventilation [24]. However, it is possible that some lung stretch receptors may also be involved because in conditions of atelectasis they become very sensitive, responding to very small increases of lung volume; this sensitization was only seen when there was a decrease in lung compliance [25].

4.8. Effect of Sleep and Posture on Yawning

Experiments 5 and 6 demonstrate that the occurrence of yawning on awakening is not solely due to the transition from sleep to wakefulness because it did not happen if I remained lying in bed. If I remained in bed but sitting upright yawning occurred in the same way as in Experiment 1, *i.e.* walking around, washing, shaving, etc. These experiments suggest that posture affects the degree of alveolar collapse, increasing collapse when the upper part of the body is vertical. It is well known that when the body is horizontal the lungs experience the same intrapleural pressure at all regions but when the chest is vertical, the gravitational effect increases the intrapleural pressure in the basal regions relative to the apex [26]. This would tend to increase the likelihood of alveolar collapse in the basal regions. However, the relevance of gravitation on ventilation has been questioned in recent years and it has been suggested that, in clinical conditions, a more important factor may be the asymmetric branching of the airways [27]. Nevertheless, in the absence of lung disorders, gravity may provide the simplest explanation the postural effect.

4.9. Interpretation of "Alveolar Collapse"

I have been using the term "alveolar collapse" because that is the term in general use. However, recent studies in normal conscious humans demonstrate that going from deflation to inflation is accomplished mainly by the recruitment of alveoli, that is, the number of open alveoli increases, while the volume and size of all alveoli remains relatively unchanged [28,29]. If this is correct, we can rephrase "alveolar collapse" as "closed alveoli". It is not known how "closed" alveoli become open but this might be a function of surfactant. If inflation caused secretion of surfactant at the mouth of a closed alveolus this might cause it to open.

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