Challenges in teaching the mechanics of breathing to medical and graduate students

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West JB. Challenges in teaching the mechanics of breathing to medical and graduate students. *Adv Physiol Educ* 32: 177–184, 2008; doi:10.1152/advan.90146.2008.—The mechanics of breathing has always been a difficult topic for some medical and graduate students. The subject is very quantitative and contains a number of concepts that some students have trouble with, including physical principles such as pressure, flow, volume, resistance, elasticity, and compliance. Apparently, present-day students find the subject more difficult than students of 20 years ago. A possible reason for this is that the teaching of elementary physics in high school and college is now given less emphasis, whereas other topics, such as molecular biology, receive a great deal of attention. Another factor may be that many of us grew up building radios and other such devices, whereas modern students tend to plug in an electronic unit with little idea of its function. Some examples of misconceptions of present-day students who have taken our course are given. To help the weaker students, we now include a primer at the beginning of our handout for the course that covers simple physical principles. Examples of some of the most difficult concepts for students are given.

pressure; flow; resistance; elasticity; compliance

**When I was invited** to take part in this refresher course on the teaching of respiratory physiology, I requested the topic of mechanics of breathing because it is one of the sections that students find most difficult. It is interesting that, apparently, the present-day student finds the subject more challenging than was the case, say, 20 years ago, despite the fact that the actual material that we teach has not changed much. One likely reason for this is that the teaching of applied mathematics and physics in high school and college has been deemphasized in favor of other topics, such as molecular biology. Certainly, in my last year of high school (admittedly 60 years ago), we had a very strong grounding in physics, physical chemistry, and mathematics. In fact, the five subjects that I took in my final year of school were mathematics 1 and 2, physics, chemistry, and English. Looking back, it seems incredulous that there was no biology at all (in fact there was not even any organic chemistry), and no one today would regard that selection of subjects as balanced. However, we certainly learned a great deal of physics, and, in my final year, I wrote a description of the mechanism of the cyclotron, a particle accelerator developed at the University of California (Berkeley, CA) for our school science journal. All of this is rather anecdotal, and I do not have any metrics to conclude that the teaching of physics in high school and college has been downgraded, but this certainly is a general impression among educators.

Another reason why I grew up with an understanding of physics is that we made radios and similar electronic devices. I started, like most boys, with a crystal set but progressed to radios using thermionic valves (this was long before transistors). Do young people today do this? I think not. Rather they tinker with their computer, iPod, or whatever, and, if something goes wrong, a plug-in electronic component is replaced. There is little understanding of the inner workings of the device. Largely gone are the days of ham radios, although I still have my call sign, KA6EYI, and many of us have nostalgic memories of those wonderful Heathkits, where the assembly necessitated knowing what you were doing and, particularly, avoiding the high voltage hazards of a linear amplifier. If you Google Heathkit now you come up with the Heathkit Virtual Museum!

It might be interesting to illustrate some of the misconceptions of our students in this year’s course, and here are a couple of examples. The first involves a demonstration on mechanics that we show on the first day of teaching respiratory physiology. In fact, this is the first day of the whole physiology course because we choose to teach respiration before the other organ systems. We have three 50-min lectures in the morning and early afternoon (normally we avoid more than 2 lectures a day, but on the first day it is not possible to have small-group discussions or laboratories). The first lecture is on structure-function relationships in the lung and is an introduction to the anatomy of the lung as it relates to function. The second lecture is on ventilation, and the third is on respiratory mechanics and in particular statics, that is, pressures and volumes of the lung and chest wall.

After the third lecture, we have a 50-min demonstration in which a rabbit lung is set up in a Lucite box and ventilated by intermittently reducing the pressure in the box by means of a pump and rotating valve. The trachea of the lung is cannulated and connected to a small spirometer, as shown in Fig. 1. A water manometer measures the pressure in the box, and, by means of a video camera, the lung, water manometer, and spirometer are displayed on a large screen at the front of the lecture theater. The water manometer has one limb connected to the box and the other open to the atmosphere, and of course when the pressure in the box is reduced by the pump, the water column in the limb connected to the box rises. Now here is the punch line. I was very surprised when a student came up to me after the demonstration and said that he could not understand why the levels in the water manometer changed when the pressure in the box was reduced. Eventually, someone came to his rescue, pointing out that he uses the same principle to suck soda out of a can with a straw. However, to me, this was a vivid example of unfamiliarity with very basic physical principles.

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The next example is actually from the mechanics of the pulmonary circulation, but, again, it demonstrates an important misconception that frequently surfaces. Figure 2 shows a multiple-choice question about what happens when the blood flow to one lobe of the lung is completely obstructed. The first choice, choice A, “Vascular resistance of the nonoccluded lung is decreased,” is a true statement. The decrease in resistance is caused by recruitment and distension of pulmonary capillaries when all the blood flow is forced through the nonoccluded lung and the capillary pressure therefore rises. Leaving choice B for the moment, choice C, “Alveolar P\textsubscript{O}2 in the occluded lobe is increased,” is also a true statement because since the ventilation-to-perfusion ratio of the lobe rises, the P\textsubscript{O}2 increases. Choice D, “If total ventilation and blood flow remain constant, arterial P\textsubscript{O}2 will fall,” is also true. This is a more difficult concept, but it tests the student’s knowledge that whenever ventilation-perfusion inequality is introduced, the net result must be a fall in arterial P\textsubscript{O}2, with all other things being equal. Choice E, “After 3 days, the compliance of the occluded lobe will be reduced,” is a true statement because alveolar surfactant turns over rapidly and cannot be replenished in the occluded lobe because there is no blood flow. The result is a reduction in compliance of the lobe.

Now let’s turn to choice B, “Vascular resistance of the overall lung is decreased.” Surely most people would see intuitively that this is a false statement, because if you block a major conduit in the lung, the resistance to blood flow must increase. But, in fact, a substantial number of students do not see this. They argue that because the vascular resistance of the nonoccluded lung is decreased (choice A), the resistance of the overall system is decreased. To me, this is an example of change in a simple physical system that should be intuitive. For example, all the students would accept the fact that if you are trying to drive into a major city and one of the three freeways available is completely blocked by an accident, the resistance to the flow of traffic into the city is increased.

Because so many students have difficulty with relatively simple physical principles, we now add a primer at the front of the handout that is given out to all students and that includes the schedule of lectures, etc. This primer is shown in the APPENDIX. Incidentally, students occasionally raise questions about the first diagram (Fig. 13 in the APPENDIX), which simply shows that if a fluid (liquid or gas) flows along a tube, the pressure at the upstream end must be greater than the pressure at the downstream end. Sometimes a student will ask whether the difference in pressure is responsible for the flow or whether the flow is responsible for the difference in pressure. The answer is that this is not a useful question. The two must coexist. In fact, the concept is very important in understanding air flow in the lung under certain conditions because if you know that there is no flow, you can be certain that there is no pressure difference between the two ends of the tube.
It might be useful to briefly describe the respiratory section of our physiology course. I should start by saying that we only have 8 days to teach the whole of respiratory physiology. The section consists of twelve 50-min lectures, supplemented by two laboratories and three discussion groups. In the laboratories, which are really partly demonstrations, the students rotate through three stations on each of two afternoons so that they see six situations where respiratory physiology is being used. The six topics of the laboratories are spirometry (including flow-volume curves), measurement of the diffusing capacity of the lung for carbon monoxide, control of ventilation, exercise, expired gas patterns (including a single-breath nitrogen washout), and cardiac output measured by a rebreathing technique. The demonstration to the whole class of measuring a pressure-volume curve on an isolated rabbit lung was referred to above. The three discussion groups involve breaking the class into groups of ~12 students and assigning to each student a simple problem to present to the rest of the group. A faculty facilitator sits at the back and intervenes if a student is going off the rails. There is also a clinical correlation session at the end of the 8 days where we present two patients, one with obstructive lung disease and the other with restrictive lung disease, to demonstrate how respiratory physiology is useful in the clinical setting.

It might be helpful to add an example of the questions that are assigned to students in the small-group discussion sessions. One of these is shown in Fig. 3. This is one of the questions assigned to one of the students in this year’s course. I leave it to the reader to go through the calculations. For choice e in Fig. 3, the answer to the second part is 80 Torr, and the key assumption is that the patient’s carbon dioxide production is normal. The answer to choice f in Fig. 3 is hypoventilation.

I will not go through the content of the two lectures that are given on respiratory mechanics in detail, because these follow chapter 7 in my book, *Respiratory Physiology: the Essentials* (1). However, it might be useful to highlight some of the concepts that students find particularly difficult.

The diagram shown in Fig. 4 shows what happens when the diaphragm contracts, and students are sometimes confused by the fact that the lateral dimension of the rib cage increases. Their argument is that if the tension in the diaphragm rises, this should pull the attachments to the rib cage together. Indeed, this is a reasonable expectation. Perhaps the simplest way to explain the result is to say that when the diaphragm descends, it increases the pressure in the abdominal cavity, which is like a fluid-filled balloon, and this is responsible for splaying the rib cage.

The diagram in Fig. 5 uses the same preparation as Fig. 1 but now shows the static pressure-volume curve of the lung. An important concept here is that for a given lung volume, the difference of pressure between the inside and outside of the lung is fixed. Suppose the pressure in the jar is reduced to 10 cmH₂O below atmospheric (~10 cmH₂O) and the lung therefore expands. Now if we put a stopper in the airway of the lung and allow the pressure in the jar to rise to atmospheric pressure, what is the pressure inside the lung? The answer is +10 cmH₂O. Furthermore, if we leave the stopper in the airway and reduce the pressure outside the lung to ~15 cmH₂O, what is the pressure inside the lung now? The answer is ~5 cmH₂O. I go through these pressure changes on the blackboard while...

A 17-year-old female high school student was brought unconscious to the emergency room by her friends at 2:00 a.m. Her breath smelled heavily of alcohol. The friends disappeared before any information could be obtained. The patient has blue lips and fingernail beds and is barely breathing. Her tidal volume is 325 ml and respiratory rate is 15 breaths per minute. Arterial blood gases are:

\[ P_{O_2} = 50 \text{ Torr} \quad P_{CO_2} = 80 \text{ Torr} \quad pH = 7.20 \]

a. What would normal total ventilation be for this patient assuming 15 breaths per minute and a normal tidal volume of 500 ml?
b. If anatomical deadspace were 150 ml, what would normal alveolar ventilation be in this patient?
c. What is her actual total ventilation?
d. Assuming anatomical deadspace is 150 ml, what is her actual alveolar ventilation, in (i) liters/min and (ii) as a percentage of normal?
e. Alveolar PCO₂ is normally 40 Torr. What would you expect alveolar PCO₂ to be in this patient? Explain your answer. What key assumptions must you make to come up with this prediction?
f. What is likely wrong with this patient?

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Fig. 3. Example of a question with several parts assigned to a student in one of the early small-group discussion sessions. This is testing material from the lecture on ventilation.
interacting with the class. The bottom line is that for a given lung volume, the transpulmonary pressure is fixed.

The diagram shown in Fig. 6 is about the behavior of surfactant, which is always a difficult topic for some students. In fact, many students cannot grasp the concept of surface tension. Here it is useful to compare the surface of a liquid with a thin sheet of rubber under stretch. The discussion of surface tension in the alveolar-lining layer raises a number of difficult issues. One is what is the mechanism by which the surface tension is so greatly reduced when the area of the surface is reduced. We explain this by saying that apparently the surfactant molecules are hydrophobic at one end and hydrophilic at the other, and they align themselves in surface of the fluid. When this occurs, their intermolecular repulsive forces oppose the normal attracting forces between the liquid surface molecules that are responsible for surface tension. The reduction in surface tension is greater when the film is compressed because the molecules of surfactant are then crowded closer together and repel each other more. Another difficult point is why does surfactant exhibit such marked hysteresis, as shown in Fig. 6. We explain this by saying that when the surface is compressed the molecules are lined up like soldiers on parade, but when the surface is expanded the orientation of the surfactant molecules becomes random. This change in configuration of the molecules requires work that is reflected in the area of the hysteresis curve shown in Fig. 6B.

Figure 7 shows relaxation pressure-volume curves for both the lung and chest wall. Figure 7 illustrates some difficult concepts, and probably some courses will omit it. However, once grasped, it encapsulates a lot of useful information on the pressure-volume behavior of the lung and chest wall. The key to understanding Fig. 7 is to take the three lines one by one. Start with the central line, that is, the lung plus chest wall, and point out that the relaxation pressure at the functional residual capacity (FRC) has to be zero (atmospheric pressure) because the FRC is the resting equilibrium volume of the lung plus chest wall. Then, go on to the righthand curve, which is the same as that shown in Fig. 5 (without the hysteresis) except that it shows inflation by positive pressure rather than negative pressure. The only really difficult line is the lefthand line, the line for the chest wall alone. This makes the point that at lung volumes above ~70% of the vital capacity, the chest wall no longer tends to spring out but instead to spring in. Another important point is that the relaxation pressure of the lung plus chest wall is simply the sum of the relaxation pressures of the two components.

Now let’s turn briefly to dynamics, which has to do with pressures and flows. I do not spend a lot of time on the diagrams shown in Fig. 8 because laminar and turbulent flow are dealt with extensively by the cardiovascular section later in the course. However, it is important for students to be familiar with these two patterns. Also, we use Fig. 8 to introduce the Poiseuille equation for laminar flow with its important radius to the fourth power term, and we also introduce the Reynolds number. The diagram shown in Fig. 9 is very important. For example, students can easily become confused in the early
stages about what happens to alveolar pressure during inspiration. The best way to tackle the information shown Fig. 9 is to start with volume data (top), which are simple, and then go to the flow data (middle bottom), which follows directly from the volume. Then, move to the alveolar pressure tracing (Fig. 9, bottom), which would mirror the flow if airway resistance was constant (which it nearly is during normal breathing), and finally end up with the intrapleural pressure data (Fig. 9, middle top). This last is always confusing to students at first until they recognize that intrapleural pressure during inspiration falls because of two factors. One is the increase in elastic recoil of the lung, and the other is the fall in alveolar pressure that is necessary for flow to occur.

Finally, I spend a good deal of time on dynamic compression of the airways, partly because it is initially confusing to some students but also because of its critical importance in patients with lung disease. I take it slowly. The information shown in Fig. 10 is relatively easy to understand, and, in fact, we measure flow-volume curves on some students in one of our six laboratory sessions referred to above. The graph shown in Fig. 11 is always confusing until the student realizes that these curves do not represent three separate inspirations and expirations. In contrast, they are derived from a series of forced expirations, such as shown in Fig. 10, and in each of these the intrapleural pressure is read off at particular lung volumes. (Of course Fig. 11 shows inspiration as well as
expiration, whereas Fig. 10 depicts only expiration.) I go through the diagrams shown in Fig. 12 in detail because this is the nub for understanding dynamic compression of the airways. Incidentally, Fig. 12 is something of an oversimplification, but for my money this is completely justified for students at this stage.

I hope that this rather rambling series of anecdotes will be helpful to people who teach this difficult subject. In conclusion, we should have no doubts about the fundamental importance of respiratory mechanics, both in medical practice and simply in understanding how the lung works.

APPENDIX: SOME ELEMENTARY PHYSICS FOR THE ORGAN PHYSIOLOGY COURSE

Flow, Pressure, and Resistance

A fluid is something that flows and includes liquids and gases. Many of the same principles apply to both blood flow in arteries and air flow in the lung.

If a liquid flows along a tube, there must be a pressure difference \( (P_1 - P_2) \) along the tube (Fig. 13). It is not useful to discuss whether the pressure difference causes the flow or the flow causes the pressure difference. They simply coexist and are the result of viscous losses within the tube. The larger the pressure difference, the higher the flow.

Laminar and turbulent flow. If a fluid flows through a long, smooth-walled, narrow tube at a low flow rate, flow is likely to be “laminar.” Here, the particles of fluid travel parallel to the sides of the tube. This is often the case for blood flowing in veins. Flow is
proportional to the pressure difference (Fig. 14). The viscosity of the fluid is an important determinant of the relationship between pressure drop and flow.

At higher flow rates, the fluid particles swirl around in the tube. This is called turbulent flow (Figure 14B) and occurs in the trachea during exercise. Now, pressure is not proportional to flow but, approximately, to its square. In addition, the viscosity of the fluid becomes relatively unimportant, but an increase in fluid density increases the pressure drop for a given flow.

Bernoulli's principle. If fluid is flowing in a tube that is constricted in the middle, the pressure in the fluid flowing through the constriction is less than upstream or downstream (Fig. 15). The reason is that the fluid has to accelerate to get through the constriction, and, therefore, its kinetic energy increases. Consequently, its potential energy (which is related to its pressure) falls. This effect is important in some diseases of arteries.

Resistance. Resistance \( R \) is defined as the pressure drop divided by the flow. That is, \( R = (P_1 - P_2) / \text{flow} \). From this, it can be seen that for laminar flow, the resistance is constant irrespective of the flow. However, with turbulent flow, the calculated resistance increases with flow rate.

There is an analogy between flow resistance and electrical resistance. In the latter, the resistance of a piece of wire is given by the difference in volts (\( \Delta V \)) (or electromotive force) between the two ends divided by the current, that is, \( R = \Delta V / \text{current} \). An important difference between biological and electrical systems is that in electrical resistors, the resistance is independent of applied voltage or current, whereas in many biological systems the vascular resistance alters with the flow. This is partly because flow is not laminar but also because the geometry of the tubes changes as pressure is increased. For example, the diameter of the tube may increase when the pressure rises, and this may decrease its resistance. This occurs in the pulmonary circulation.

Force, Extension, and Elasticity

Suppose a coiled spring is extended by attaching weights to the bottom (Fig. 16). Typically, the length is proportional to the applied force. (The force in Newtons is the mass in kilograms multiplied by the gravitational constant \( g \)). If the weights are taken off, the spring returns to its original length. Elasticity is the property of a body to return to return to its original size after being distorted by a force. (In everyday speech, “elasticity” is often used for “stretchability,” but in fact a steel ball is technically more elastic than a rubber ball.)
Compliance ($C$) is the change in length ($\Delta L$) divided by the change in force ($\Delta F$), that is, $C = \Delta L/\Delta F$. (This fits with common usage, where we talk of something or someone being compliant if it/they are easily influenced.)

Other structures, such as balloons or lungs, also exhibit elasticity. If we inflate a balloon by increasing the pressure within it, the volume change ($\Delta V$) per unit pressure change ($\Delta P$) is referred to as the compliance (Figure 17), that is, $C = \Delta V/\Delta P$. However, while in the case of the spring, the compliance is typically constant, the pressure-volume curve of a balloon may be nonlinear with decreasing compliance at high volumes. This is frequently the case with biological structures such as the lung or blood vessels. The compliance of the lung is frequently altered by disease.

**Law of Laplace.** In the balloon shown in Fig. 17, raising the pressure increases the tension (force) in the wall. Laplace’s law states that the tension ($T$) is given by pressure ($P$) multiplied by the radius ($r$) of the balloon: $T = P \times r$. We use Laplace’s law to understand the pressures needed to expand the alveoli in the lung.

**Wall stress.** The tension in the wall of the balloon results in a stress that, if high enough, can break the balloon. Wall stress ($S$) is given by the tension divided by the wall thickness ($t$), that is, $S = T/t$. This means that wall stress is $T = (P \times r)/t$. Wall stress is important in heart disease and in many other physiological conditions.

**REFERENCE**