Laplace's Law: What It Is About, Where It Comes from, and How It Is Often Applied in Physiology

By Max E. Valentinuzzi and Alberto J. Kohen

Have you ever seen a birthday party balloon shaped as a bunny rabbit or a teddy bear or Mickey Mouse? Sure, you have. Did you know that Laplace's law governs the mechanical behavior at every single point over their respective surfaces? Ha-ha-ha… a healthy laugh comes out, 'cause it sounds like Alice's little fancy joke! Imagine… His Honor, Le Marquis de Laplace, playing with puppets!

There are two beautiful laws contributing to the mechanisms associated with intraventricular pressure buildup. Both are significant in cardiac physiology. The first one is physiological, born around 1914 after a series of experiments were carried out in London by Ernest Henry Starling (1866–1927) and his collaborators, while the second one is physical, found in a monumental opera, *La Mécanique Céleste*, authored by the French mathematician and astronomer Pierre Simon Laplace (1749–1827) and published in several volumes between 1790 and 1825 [1], [2].

Here, we want to deal with the latter, first saying what it is all about, and thereafter briefly searching in the regular physiology textbooks for its apparent background and/or origin, and finally mentioning its applications to better understand some pathophysiological behaviors. Another note will tackle its mathematical derivations, for there are several, including their historical chronology that brings to light the question of who first really found the law, while a final note will discuss a few epistemological puzzling aspects. Altogether, Laplace's law will be covered in three notes. Surely we can anticipate that Laplace was not the first who ever cared about the heart, vessels, and bubbles. Somehow, someone in the middle credited him with the law, which, incidentally, is not usually taught in regular physics courses. The subject Le Marquis did study was surface tension of liquids contained in capillaries.

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What Is Laplace's Law?

The relationship is often and traditionally found in physiology textbooks when referring to hollow organs such as the cardiac chambers, blood vessels, bladder, stomach, uterus, lungs, or the like. It states that the intracavitary pressure (P_{ic}), more precisely the difference with the external pressure (which frequently is ignored as being constant and is usually equal to the atmospheric pressure), is directly proportional to the wall stress (W_s) and inversely proportional to the equivalent radius (R_{eq}) or

$$P_{ic} = k\, W_s h / R_{eq} \qquad (1)$$

where k is a constant and h is the wall thickness. Another convenient way of expressing it is by stating that the product $P_{ic}R_{eq}$ is proportional to the $W_s h$ (wall stress and wall thickness) product. The equivalent radius is a concept trying to reduce any cavity shape into a sphere (quite a reduction, indeed, but models always simplify things out); hence, you as a reader must work out a procedure to do that and, obviously by and large, such a sphere has little in common with the original shape.

Another simpler form applies to spherical bubbles (typically, soap bubbles) with a wall of negligible thickness; in other words, it refers to a film under tension, the so-called surface tension (T), which is defined as the tangential force (tangent to the surface) applied perpendicularly to a unit length segment lying on the surface. However, if we consider a membrane separating two spaces of any shape with tension in it (as the bunny rabbit balloon mentioned earlier), its curved surface can always be decomposed into many elements (kind of caps resembling a parachute), each defined by two mutually perpendicular circumferences of respective radii, R_1 and R_2, which are called the *principal radii of curvature* (because they define the curved surface element) [3], [4]. Inside the balloon, the pressure P (actually, the difference between the inside and outside pressure, which most frequently is the atmospheric pressure, as stated earlier) is given by

$$P = T\left[\frac{1}{R_1} + \frac{1}{R_2}\right] \qquad (2)$$

where T is the already mentioned surface tension in dynes/cm and R is the radii expressed in centimeters. It is easily seen that pressure is measured in dynes/cm^2. However, (2) incorrectly assumes a uniform tension value all around the 360° of the studied point Q (Figure 1); more realistic is to consider tension T_1 and T_2, respectively, corresponding to each principal curvature radius, i.e.,

$$P = \frac{T_1}{R_1} + \frac{T_2}{R_2} \qquad (3)$$

and we will stick to the latter from now on.

When both the radii and tension values are equal, we have a sphere (such as a bubble), and the equation simplifies to

$$P = \frac{2T}{R}. \qquad (4)$$

Figure 1 graphically clarifies the conceptual framework from which the spherical and cylindrical cases are easily visualized

by stretching the parachute in the longitudinal AB direction (to become a cylinder) or equalizing both AB and CD arcs (which is the case of a sphere). Also observe that the tension T_2 along CD tends to zero as the radius R_2 tends to infinity, because the cavity is now a cylinder; hence, the equation becomes even simpler, or $P = T_1/R_1$, which is precisely the case of a blood vessel.

Thus, if the latter equation for the sphere is compared with (1) by equating both the right-hand terms (because the pressures inside are the same), it comes out that

$$k\, W_s\, h = 2T \tag{5}$$

or

$$W_s = \frac{2T}{kh} = \frac{KT}{h} = \frac{KF}{d.h}, \tag{6}$$

clearly showing that wall stress is proportional to the surface tension distributed over the wall thickness. The constant $K = 2/k$ can eventually be equated to one, while d is the unit length over the surface to which the force F acts perpendicularly and tangentially to the same surface. Observe that the product $d.h$ represents a small rectangular cross-sectional area over which a perpendicular force F is applied. It must be underlined that T is equal all around the surface point Q only when the cavity is a sphere. Besides, a uniform distribution in depth can be assumed over the wall thickness, h, at each level; the latter situation, however, is not met in practice, as tension shows an increasing gradient from the inner to the outer layers.

Regarding Principal Radii:

"At each point on a given two-dimensional surface, there are two principal radii of curvature. The principal directions corresponding to the principal radii of curvature are perpendicular to one another. In other words, the surface normal planes at the point and in the principal directions are perpendicular to one another, and both are perpendicular to the surface tangent plane at the point."

The latter short paragraph is a quote from [4]. The radii fully characterize the curved surface element.

To complement (6): Wall stress is an extremely important concept in cardiology. Excessive stress causes injury to the cardiac tissue and the latter, in turn, may end up in lower contractility and consequent insufficiency. One clinical strategy aims at reducing wall stress, which acts as an *internal load* to the heart. Remember also, this is *mechanical stress*, not related to *psychological stress*; the former refers to a possible physical rupture while the latter may be interpreted as an emotional rupture risk (the so-called nervous breakdown). Nonetheless, the latter can lead to the former. Thus, beware of the boss, spouse, or money issues!

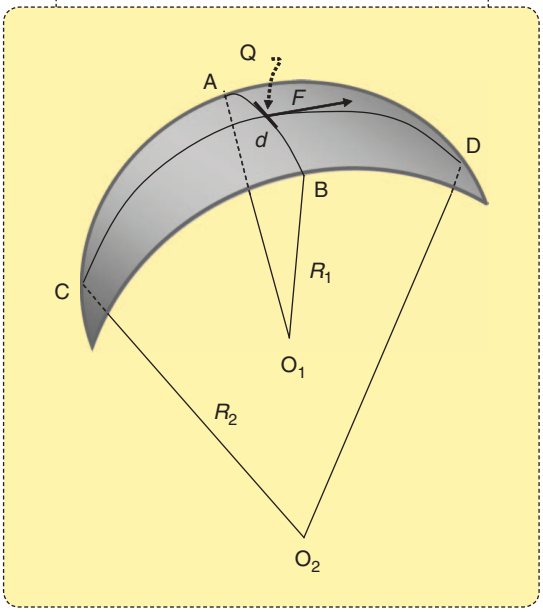

FIGURE 1 Parachute-like shape of a curved surface element cut from a complex irregular volume (e.g., a bunny rabbit balloon). The membrane thickness is negligible. There are two segment osculating curves, AB and CD, each part of a circumference and perpendicular to each other at point Q. Their respective radii (called *principal radii*) are R_1 and R_2 with centers O_1 and O_2. At point Q, force F applied perpendicularly to the length element d pulls tangentially along CD. Another similar force (not shown) does the same along curve AB. F/d is defined as the surface tension in the CD direction, while the other force divided by the perpendicular unit length segment d defines the surface tension in the AB direction. In fact, the tangentially applied forces should be named F_1 and F_2, respectively, to be precise. In the case of a sphere, both surface tensions are equal. In the case of a long cylinder (CD becomes a straight line with infinite radius R_2), surface tension is only radially defined, i.e., along the arc AB.

Where Does the Law Come from?

When one of these authors (Max E. Valentinuzzi) faced the law as a graduate student in the physiology courses, he tried to remember in which physics course he had seen it before as an engineering undergraduate; however, there was no memory of it. His colleague (Alberto J. Kohen), an active physics lecturer in engineering, did not recall it either. We both consulted with a couple of physicists and checked several physiology textbooks, and the results were disappointing. The physicists had no recollection, and the information found in the textbooks ranged from very poor (or even wrong) to right but incomplete or limited and far from being indisputably convincing, both historically and mathematically speaking. Let us review a few, after browsing original copies resting in my (Max E. Valentinuzzi) personal library over a period from 1914 to 1993.

The first mention was found in a Spanish translation of a rather old textbook published in 1926, which was used by Max E. Valentinuzzi's father when a medical student in Buenos Aires back in 1928 (Gley [5]). Laplace and the *Mécanique Céleste* are briefly referred to but not in regard to any of the expressions described herein. The author vaguely refers to the respiratory gas exchange studies that Laplace apparently carried out with the chemist Lavoisier. Neither the section was devoted to the cardiovascular system nor the thoracic cage or the lungs (seen as compliant cavities) recalls such law.

Almost 40 years later, a small but outstanding book by the famous Alan C. Burton presented the law as in (2), discussing and obtaining excellent and useful consequences applicable to circulatory physiology. However, no lead was given regarding its mathematical background, and the concept of wall stress was lacking [6]. Close in time, the traditional 19th edition of Howell-Fulton [7] textbook explicitly refers to the law when dealing with vessels and the heart. It mentions

other authors too, and quite interesting numerical examples and illustrative graphs are given, but the mathematical expression is presented again in the simplified and incorrect form of (2), without any derivation [7]. Soon thereafter, we found that the textbook by David F. Horrobin [8]. Several times the law is mentioned along with its applications in cardiovascular, respiratory, and bladder physiology, but once more no good mathematics is given, and even a historical error shows up when Laplace appears as "having studied the relationship between a vessel radius, tension in its wall, and the pressure across it." No, to the best of our knowledge, he never did that. In turn, Mountcastle, in 1968, came out with another well-known textbook, where the law is barely and poorly referred to [9]. No other comment appears as pertinent in it.

Skipping over a decade, we found two of the mostly used textbooks in medical schools—that of William F. Ganong's and Guyton's [10], [11]; however, the information in them regarding the said law is extremely poor. By the same token, the Spanish edition of a huge treatise by Lloyd H. Smith and Samuel O. Thier, barely and even almost as passing, mentions the law and the simple equation (2) form, adding also a few other minor comments [12]. Very meager in this respect is the discussion offered by the traditional Best and Taylor in another Spanish edition based on Brobeck's version [13]. In Argentina, the traditional Houssay's physiology, in its 6th edition, gives a good discussion of all cardiac biomechanics based on the modern concepts of the pressure–volume diagrams [14]. Laplace's law is given in a simplified form, where two semispheres are the reference shapes when trying to pull them apart by the pressure P inside. Such pressure can be expressed as a force F over the sphere cross-sectional equator area, i.e.,

$$P = F/\pi R^2, \tag{7}$$

and the tendency to separate both halves apart must be counteracted by the surface tension T generated by the same force, but applied perpendicularly to the equator circumference, i.e.,

$$T = F/2\pi R. \tag{8}$$

Solving the two equations above for F and equating both results produces $P\pi R^2 = T2\pi R$, from which $P = 2T/R$ is easily obtained, identical to (4). However, although its mathematics and physics are correct, they are restricted in reach, lacking generality.

To finish this quick tour along time through several common textbooks, let us go back to 1914, almost a 100 years ago, to browse Leonard Landois' Spanish version of his physiology treatise [15]. It does not mention the law. Thus, the conclusion is that Laplace's law has been referred to frequently but poorly in the regular physiology textbooks, and even errors are detected. No good and convincing mathematical derivation is given, and no clue seems to be found regarding its first inclusion in physiology—indeed disappointing.

Thereafter, we established a search in the regular and more specialized scientific physiology literature, where better and more detailed information could be found.

The first application of this law in physiology we could find, and to the best of our knowledge, was that of Robert H. Woods [16], in Dublin, Ireland, back in 1892. In fact, later on, this author published a letter seriously questioning some concepts previously made public by Samways [17]. Let us reproduce almost verbatim that letter because it is enlightening indeed. It indicates two sources for the same article, while it clearly shows that a complete understanding of the subject had not been yet reached. The equation given seems to include the factor t for both thickness and wall stress, although the author only mentions the former, and the equation is not fully correct, as already explained earlier.

> In the *British Medical Journal* of 23 January 1897, there appears an article by Dr. D.W. Samways, in which he details a series of experiments with two bags of different capacities distended under the same pressure, and applies the conclusions drawn to the working heart. In the year 1892, I read before the Royal Academy of Medicine, in Ireland, and published both in its *Transactions* and in the *Journal of Anatomy and Physiology* [16], a paper on *A Few Applications of a Physical Theorem to Membranes in the Human Body in a State of Tension*, a copy of which I now enclose, where the same experiment was described in the same connection, with this difference, that my paper treats the subject mathematically. Both Dr. Samways and I have shown that a small contracting sac has a mechanical advantage over a large one, but he pushes the matter to an absurdity when he contends that the left auricle by its contraction sends blood into the contracting ventricle. There are no valves between the auricle and the lungs, and therefore raising the intraauricular pressure during contraction of the ventricle could only result in the blood following the path of least resistance, namely, back into the pulmonary veins. To add another reason may seem "wasteful and ridiculous excess," but it will be found that the mean radius of curvature of the contracted auricle is nothing like small enough to enable it to raise the pressure of its contents to anything approaching that of the ventricle when the relative thicknesses of the walls are taken into account. By taking the formula given in my paper (p. 367),
>
> $$P = t(1/r_1 + 1/r),$$
>
> where P is the pressure of blood, t the thickness of the wall at any point, and r_1 and r, the maximum and minimum radii of curvature at that point, and applying it to the contracted auricle and dilated ventricle in question, its value will be found to be greater for the ventricle than for the auricle.
>
> *Robert H. Woods, M.B., F.R.C.S.*
> *(Dublin, 30 January)*

Máximo Valentinuzzi, Sr., retook the subject many years later in regard to the human uterus, having carried out multiple experiments in vitro and in balloon models [18], [19]. For historical reasons, he called it as *Barrau–de Snoo's law*, instead of Laplace's law, since Karl de Snoo, in Utrecht, The Netherlands, had also studied the laboring uterus in

The Netherlands in 1936, while Barrau, a mathematician, verified and apparently simplified the calculations [20]. These latter authors stated the law by saying that "tension on a given surface point of the uterus equals the product of the intrauterine pressure and the curvature radius at that point," which is similar to the statement given earlier after (1) but simpler; however, they did not seem to have recognized Laplace's long-standing priority. Nonetheless, first, de Snoo and afterward Valentinuzzi repeating it, a geometrical and ingenious demonstration was offered [18]–[20]. It will be developed in the second note of this subject when dealing with its mathematical foundations.

In a short communication, Martin and Haines [21], from the University of Oklahoma, performed an experimental study of the left ventricular thickness and radii in calf, dog, rabbit, guinea pig, hog, and rat with the purpose of checking how well the numerical values followed Laplace's surface tension law. Assuming blood pressure to be constant (= C), they rearranged the equation as in (2), but replacing T by t and defining the latter as ventricular thickness, i.e., quite similar to what Woods presented in 1892 [17]. Their results gave an average C of 0.737 with a coefficient of variation of 22%, whereas the heart weight varied 767 times. No units are given. Thus, this article does not add any significant information to our search.

Another experimental set of measurements was run by Valentinuzzi et al. in 1987 [22]. Burton's predictions were found to be correct, i.e., the left ventricular wall is thicker around the middle region and toward the base. However, it was also found that the law held only for hearts in systole and when the papillary muscles were included as part of the wall thickness. The study included canine, cow, chicken, and human hearts.

Concluding the second part of this section, the specialized literature displays a good deal of experimental tests, and a mathematical demonstration was located (which is left for our second note dealing with Laplace's law). Probably, there are more reports testing it experimentally, but we think it would not add anything of significance.

How Can the Law Be Applied in Pathophysiology?

The simplified version of the law, as in (4) for a sphere or as in its cylindrical version, is good enough to make qualitative predictions giving the basis for the many good physiological discussions found in the textbooks. Let us go back first to our birthday balloon; when air is blown in, a certain volume needs to build up before a pressure gauge would detect a small pressure value, in turn developing some counteracting surface tension. As more air gets in, the pressure inside increases, and the surface tension also increases. When the balloon becomes too big, simple finger touching signals a large surface tension and, therefore, a possible rupture risk. All these phenomena are static in nature.

William Ganong in his well-used textbook [10], or in earlier editions such as the 7th edition of 1975, explains the case of a blood vessel using the equation $P = T_1/R_1$ (see above): the smaller the vessel's radius, the smaller the wall tension to balance the distending pressure. Moreover, when pressure in a small vessel is reduced, a point is reached where blood flow becomes zero, although the pressure is not zero. The latter is called *critical closing pressure* (see previous paragraph when a balloon is inflated and compare both statements, as the latter phenomenon describes its counterpart). Also notice that surface tension (in a thin film) appears as loosely and confusedly mixed with the concept of wall stress (where thickness plays a role). A few numerical values illustrate this (given in the table below).

Vessel	P (mmHg) (dyn/cm^2)	R (cm)	$PR = T$ (dyn/cm)	Wall thickness, h (mm)
Aorta	120		200,000 C	
	160,000	1.25	170,00 G	2.00
Vena cava	8		16,500 C	
	11,000	1.50	21,000 G	1.50

1 mmHg = 1,333 dyn/cm^2. The fourth column was calculated with the simplest equation (2) in its cylindrical form for vessels. For the aorta, we have taken the systolic pressure; for the vena cava, 8 mmHg was accepted. Check the figures of this table with others reported in the literature (as a little exercise). *C* means computed with $PR = T$; *G* means data from Ganong's textbook.

An aneurysm (literally meaning widening, from Greek) is a sac formed by the dilatation of the walls of an artery or vein. The elastic properties change dramatically so that the walls become more compliant and, thus, mechanically weaker. Since surface tension in a cylinder appears only tangentially and along the circumferential cross section, as the vessel weakens, a dissecting aneurysm (wall rupture) only occurs longitudinally, because the pull is perpendicular to the vessel direction. The law predicts such behavior, and clinical practice demonstrates it.

Consider now any of the cardiac chambers, say the left ventricle. A dilated heart shows a larger equivalent radius, while its wall becomes thinner than normal. Thus, the myocardium must develop a greater tension during contraction to sustain the required pressure, and in the end, poor ejection of blood may occur. Conversely, if hypertrophy takes place (say because of hypertension or excess physical training), the ventricular wall gets thicker, partially compensating for the necessary increase in wall stress. We do not enter into the etiology of these cardiac pathologies; we underline only the law that explains the behavior.

Another example: anatomical observations in normal ventricles showed that the radius at the basal region is larger than at the apical zone, thus approximating the left ventricle with two superimposed spheres, an upper and a lower one, as Valentinuzzi et al. did [22]. Since the pressure inside is the same, Laplace's law clearly explains why the wall is thicker in the former than in the latter. Besides, the right ventricle develops a much lower pressure than the left, and as a consequence, its wall is thinner.

The law also finds a place in respiratory physiology. Alveolar surface tension is controlled by a substance called *surfactant*, and T is inversely proportional to its concentration. Surfactant molecules are spread apart as alveolar size increases during inspiration (concentration

gets down) and move closer together at expiration (concentration goes up), thus adjusting T during breathing. Were this not the case, the alveoli would collapse at expiration because of the too high surface tension. Another interesting and significant phenomenon is that a small alveolus connected to a larger one empties its air content in the latter, simply because its surface tension surpasses that of the bigger alveolus. Atelectasis and hyaline membrane are the two classical examples to illustrate the importance of alveolar surface tension in the respiratory act, both directly linked to Laplace's law.

Micturition is a physiological function usually studied by cystometry, which relates intravesical volume with intravesical pressure; hence, a nice situation to look at through the law. A normal cystometry shows a middle long flat portion indicating volume increase due to vesical filling but constant pressure, because the radius of the cavity increases with surface tension, thus compensating for the enlargement. The micturition reflex is triggered at a critical high volume and surface tension.

The pregnant uterus appears as another example where the law is very handy. It shows a behavior similar to that of the bladder because surface, or better, wall stress accommodates to the slowly increasing volume [18]–[20].

Conclusions

We have presented the law as is currently found in the regular physiology literature and recognize certain flaws that seem to project from decade to decade. Its mathematical derivation is specifically never given and even one equation often cited is incorrect, as it assumes the same surface tension on both principal circumferences characterizing the curved surface element. A couple of publications in this respect by de Snoo and by Valentinuzzi, Sr. seem to be an exception. Its historical origin appears confusing at best, and proper definition of the important concept of wall stress is usually not clearly given. The next note on the subject will offer the several demonstrations we have found, which simultaneously indicate a historical enlightening chronology.

Pierre Simon de Laplace: A nobleman carrying the title of Marquis was born at Beaumont-en-Auge, France, in 1749, and died in Paris, in 1827. The solar system for him originated in a rotating nebulous formation. He heavily contributed to probability theory. The Laplacian operator,

$$\Delta = \nabla^2 = \frac{\partial^2}{\partial x^2} + \frac{\partial^2}{\partial y^2} + \frac{\partial^2}{\partial z^2},$$

also called *nabla squared*, remembers him. The celestial mechanics (*La Mécanique Céleste*) mentioned earlier was, without doubt, his opera magna written over a long span of time. In it, several subjects were dealt with, going well beyond the field of astronomy. Its reading is rather difficult, because the author was not a didactic writer or too clear in his style. In theoretical physics, the theory of capillary attraction is due to Laplace, who accepted the idea that the phenomenon was due to a force of attraction. The part that deals with the action of a solid on a liquid and the mutual action of two liquids was ultimately completed by Gauss. In 1862, Lord Kelvin (Sir William Thomson) showed that if we assume the molecular constitution of matter, the laws of capillary attraction can be deduced from the Newtonian law of gravitation.

Laplace was vain and selfish, a fact not denied even by his warmest admirers; his conduct to the benefactors of his youth and his political friends was ungrateful and contemptible, while his appropriation of the results of those who were comparatively unknown seems to be well established. Of those whom he treated, three rose to distinction (Legendre and Fourier, in France, and Young, in England). It should also be added that toward the close of his life, and especially to the work of his pupils, Laplace was both generous and appreciative [2].

Antoine Laurent de Lavoisier (1743–1794), founder of modern chemistry, tragically ended his life in the guillotine during the terror period of the French Revolution.

Leonard Landois was a German physiologist (1837–1902) who produced the first faithful blood pressure record by just letting an arterial blood jet to sprinkle the surface of a rotating drum. Its dicrotic notch was clearly depicted.

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Biomedical Microsystems

Edited By Paul King

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This book describes the history, basics, and recent important studies of biomedical microsystems. The content is composed of introductory lectures in this field.

Chapters 1–4 of the book are well organized. It starts by detailing the history, benefits, and basic design rules of microsystems. The following chapters describe materials and fabrication techniques as well as important theories in designing microsystems. Most of the important topics needed to understand current trends in biomedical microsystems are included.

This book gives a quick snapshot of a booming research area of biomedical microsystems.

Remarks on the microfluidics chapter: 1) a more detailed description of the Navier–Stokes equations is necessary to understand their importance. As the Reynolds number is one of the most important parameters in designing microfluidic systems, it should be discussed in more detail. The practical cases of Couette and Poiseuille flow as in the book are very important. 2) Another chapter discussing capillarity and contact angles is also suggested.

Chapters 5–9 provide a good overview of recent studies in biomedical microsystems. Reading Chapter 6, "Sensing and Detection Methods," before Chapter 5, "Lab-on-a-Chip," is suggested.

Overall, *Biomedical Microsystems* is an interesting book that gives a quick snapshot of a booming research area of biomedical microsystems. Depending upon the targeted research communities ranging from clinical practitioners to biomedical engineers, future expansions may include biology- or clinical-driven microsystems design with a translational impact on the society.

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