Reflections on the Waterfalls in the Chest

John Butler, M.D.

Professor of Medicine, Division of Pulmonary and Critical Care Medicine, University of Washington, Seattle, Washington

My story begins long, long ago and far, far away, in a place called Birmingham in England. I had been chief resident at the university hospital, the Queen Elizabeth Hospital. Most English hospitals are named after queens or saints. The advantage of naming a hospital after a queen is that you can get her to open it and visit it occasionally—which increases its visibility and encourages donations. This is difficult with saints. The Queen Elizabeth Hospital was the teaching hospital for the University of Birmingham. After being subjected to a period of character formation as chief resident there, one was expected to join the Medical Professorial Unit. There was only one professor of medicine in those days: Professor, now Sir, Melville Arnott (Fig. 1). Actually the professorial unit was run by a triumvirate (Fig. 2). This picture is true; only the faces have been changed to protect the innocent. One of the seated members of the triumvirate was Peter Harris, who was senior lecturer in the Department of Medicine and is a contributor to this book. He is seated on the right, but has shaved and changed his clothes since that time.

When I followed the traditional path to the unit, Sir Melville thrust a paper labeled “The mechanics of breathing in man” by Otis, Fenn, and Rahn, into my hands and said, “You’d better do some research, Butler.” Because I had been in the army, I was good at swallowing things, and, after reading in this paper about measuring esophageal pressure changes as reflections of intrapleural pressure changes, I was not surprised to find I could easily swallow esophageal balloons. Otis
and his colleagues had used the esophageal balloon to measure the work of breathing and had found that the normal breathing rate was that at which the work was least (Fig. 3). So I decided to measure the work of breathing at different end-expiratory volumes throughout the vital capacity to see if it changed and at what chest volume it was least (Fig. 4).

I found that the volume at which the work was least was the end-expiratory volume or functional reduced capacity at which each subject normally breathed (Fig. 5). This was pleasing to me because it fulfilled my preconceptions. I wrote up the studies and, in the discussion, waxed rather too lyrical about the Wisdom of the Respiratory Apparatus and the evidence...
Because Sir Melville was the editor, my paper was published after I had excised the offending parts of the discussion. Flushed with success, I cast around for a “Big Problem” to solve. At this stage of knowledge of the mechanics of the chest, the Big Problem was obviously the lack of knowledge about its normal pressure-volume relationships (Fig. 7). The Relaxation Pressure curve, as carried out by Rahn and his colleagues, depended on complete muscle relaxation after inhalation or exhalation to different volumes; they themselves had pointed out that such complete relaxation was impossible. My brainstorm was to ensure complete relaxation by measuring these pressures and volumes in normal subjects whose mus-

---

**Figure 4.** Esophageal pressure-lung volume loops at different lung volumes (%VC). Total work on the lung is made up of elastic work (dotted) and frictional work (enclosed in loop). Breathing at 15 breaths/min with 0.5 L tidal volume. The zero pressure (vertical line) was not corrected for the small chest wall effect over this volume.

---

**Figure 5.** Work of lung movement (Fig. 4) in relation to lung volume at end exhalation. $S_1$ to $S_5$ = 5 normal subjects.

---

**TELEOLOGY**

• “Evidence of Purpose in Nature”

(Oxford English Dictionary)

---

Figure 6: Teleology.
Figure 7. Static pressure volume (compliance) relations of the thorax. The relaxation pressure ($P_r$) is made up of the lung ($P_L$) and chest wall ($P_{CW}$) pressure at each volume. $V_r$ is the relaxation volume when the system is in balance. The lung exerts no pressure at $V_o$, the chest wall at $V_w$. FRC = functional residual capacity; RV = residual volume (Rahn, Otis, Chadwick, and Fenn).}

...cles had been paralyzed with a relaxant. I could do this because I had become friendly with the surgeons and the anesthesiologists while I was chief medical resident.

Of course, the idea of obtaining a true value of the elastic properties of the lungs and chest wall by inducing paralysis proved to be extremely naive. Instead of the more compliant chest that I expected, the relaxation pressure curve in the paralyzed patient showed a loss of volume below that during voluntary apnea and a compliance that was only about half that obtained in the same patient during voluntary relaxation.

At this time, the Professorial Unit was visited by a friend of Sir Melville's, a famous researcher in physiology from America, Julius Comroe, Jr. (Fig. 8). Although I had no inkling at the time, J. C., Jr., was to change my life. I showed him my work, and he told me of work that he had recently carried out with an anesthesiologist called Nims. They had done exactly the same study as I had and found the same loss of volume and increase in rigidity. It was, of course, devastating to find that what I had imagined to be a most important contribution had already been contributed. This was the first of a series of Great Discoveries I was to make, only to find that they had already been discovered long before.

Nims and Comroe had attributed the increased rigidity in their anesthetized, paralyzed patients to a failure of the normal inspiratory muscle tone in the chest wall. On the other hand, it seemed to me, because I had used my favorite research instrument, the esophageal balloon, that it was the lungs rather than the chest wall that had changed. With trepidation I told J. C., Jr. (a la Jesus Christ, Jr.) about this. He pointed out that esophageal pressures were erroneous in the supine patient but, because my control measurements had been made in the same position, reassured me that there might be something in what I had found. Could I come over to Philadelphia to work in his lab and so arrive at the truth?

Figure 8. Julius Comroe, Jr. This and the sketch in Fig. 12 were made during his lectures, when the author should have been paying attention.
In those days it was not so difficult to get research grants (Fig. 9). I got a Rockefeller Traveling Fellowship. This success stunned my colleagues in the Professorial Unit. On arriving in Philadelphia, I found that Julius Comroe had fled to the west coast to become director of the Cardiovascular Research Institute in San Francisco. At his Graduate School of Medicine in Philadelphia I was, however, lucky enough to work with Arthur DuBois. Arthur was undoubtedly the person who gave the greatest help to my research career. With him and Colin Caro I studied another technique, suggested by Colin, for chronically reducing the functional residual capacity to a similar or greater extent than that consequent to muscle paralysis. This was strapping the chest and abdomen. Within a few minutes, the lung compliance invariably fell, sometimes to less than half normal, depending on the degree of constriction that was tolerated. Only Arthur could breathe with his lungs voluntarily held at a volume low enough to cause this compliance change without the aid of strapping. The low compliance, which persisted as long as the lungs were held in an expiratory position, reverted to normal immediately after a deep breath (Fig. 10). Normally the expiratory position is associated at first with a more positive intrapleural pressure and with narrower airways and blood vessels than those at the functional residual capacity. It seemed to us that this adaptation of the lungs by "shrinking" at low volumes was useful in maintaining the normal low intrapleural pressure and thus the

Figure 9. Availability of research fellowships in the 1950s, thought to be one of the first advertisements of NIH.

Figure 10. Top: Quasi-static pressure-volume relations of the lung (lung compliance slopes) with chest strapped and during first breath to total lung capacity after removal of strapping ("off" arrows). Note low compliance abolished by lung inflation. Bottom: Quasi-static conductance-volume relations under same conditions. Note increased conductance at each volume in the low compliance state.
normal airway caliber, airway resistance, and gradient for venous return (Fig. 11). Colin Caro and I wrote this up, aiming for the stars, that is, for the *Journal of Clinical Investigation*. After many months it bounced back rejected in part because two referees had found that the amount of teleology in the discussion section “did not befit good scientific work.”

We removed the offending passages, modified other parts as requested, and it became acceptable. I returned to England after this exciting glimpse of American research and, thanks to Sir Melville, obtained a job as first assistant in the Professoral Unit in Manchester. Julius Comroe (Fig. 12) wrote to me while I was there, asking me whether I would join him in San Francisco to exploit his body plethysmograph, about which I had learned so much from Arthur DuBois. This was an unexpected windfall, and as in many other stages of my career, I knew exactly what to do. I wrote and accepted his proposal. Later after I had thought about it a little, I wrote back that I regretted having to decline the position. I then thought about it for a month or two more and sent him a telegram saying that I would accept the position if the job were still open. It was. And so I moved to America.

After the windfall began the quest for waterfalls. I had learned the nitrous oxide technique for measuring instantaneous pulmonary capillary blood flow using the body plethysmograph while I was with Arthur DuBois in Philadelphia (Fig. 13). After the plethysmograph began to work in San Francisco, I pursued the idea that the maintenance of a low intrapleural pressure was important for regulating venous return. I had to assume that the pulmonary capillary flow reflected the rate of venous flow into the chest, since the right atrium, right ventricle, and pulmonary circulation in the normal person act as a conduit through the lungs to the left atrium. Together with Paul Vermeire, I studied the pulmonary capillary blood flow in a series of normal subjects when they changed their intrapleural pressure (reflected by esophageal pressure) during respiratory maneuvers and breath holds with open glottis (Fig. 14). We found that the pulmonary capillary blood flow and the venous return increased at the beginning of a slow inhalation but then failed to

---

**Figure 11.** Usefulness of increased recoil pressures in unexpanded regions of lung in keeping airways and blood vessels patent. *Left:* Normal lung. *Right:* Restricted lung. Hooks represent suspension of lung from chest wall and hilum.

**Figure 12.** Julius Comroe, Jr.
Reflections on the Waterfalls in the Chest

Figure 13. Nitrous oxide (N₂O) technique for measuring instantaneous pulmonary capillary blood flow in man. The subject in a closed box (plethysmograph) takes a breath of air containing a very soluble gas (N₂O) from a bag in the box. The N₂O is absorbed (disappears) into the pulmonary capillary blood as it pulses into the gas exchanging regions. This causes air to flow in through the flowmeter in the wall. The rate of this flow is proportional to pulmonary blood flow, which can be calculated from the alveolar N₂O concentration and solubility. The airflow trace after a breath of air must be subtracted from the N₂O tracing so that non-N₂O events can be excluded.

We stressed the system even more by slowly inhaling through a high resistance (Fig. 15). In spite of quite negative intrapleural pressures, pulmonary capillary blood flow did not increase. This was extraordinary because we had thought that the more negative the intrapleural pressures, the greater the increase in systemic venous return.

A few years previously Permutt and his colleagues in the U.S.¹⁶ and Bannister and Torrance in the U.K.¹ had described the alveolar “waterfall” or “sluice” effect. The delicate alveolar microvessels were assumed to partially collapse to form a waterfall at their distal ends when the alveolar pressure compressing them rose above the left atrial pressure within them. To me the waterfall concept has always been wonderfully evocative of the beauty of flow (Fig. 16). The rate at which water rise further in spite of a continued fall in intrapleural pressure. During exhalation venous return was lower but unchanging. We stressed the system even more by slowly inhaling through a high resistance (Fig. 15). In spite of quite negative intrapleural pressures, pulmonary capillary blood flow did not increase. This was extraordinary because we had thought that the more negative the intrapleural pressures, the greater the increase in systemic venous return.

Figure 14. Blood flow into the lungs (N₂O method), assumed to reflect systemic venous return. Right: Flow was higher in early inhalation to vital capacity than later, in spite of more negative intrapleural pressures later. There was no further change during slow exhalation. Left: Pleural pressure and lung volume during prolonged (10 to 15 s) apnea with the glottis open had no effect on blood flow. Blood flow was higher when functional residual capacity (FRC) was reached early, during inhalation, than when functional residual capacity was held during prolonged apnea.

Figure 15. Pulmonary capillary blood flow, reflecting systemic venous return at the same negative esophageal pressures during inhalation and the start of exhalation. More negative pressures did not increase blood flow.
falls over a cliff is not influenced by how far it falls. Only when the base of the falls is brought up above the level of the top of the cliff is the rate of flow diminished.\textsuperscript{10} To strengthen the analogy, the falling water can be thought of as enclosed in a flexible sheath with blood flow in a very thin elastic walled vessel. In reality, the flow need not plunge to a lower hydrostatic level. It is sufficient if the downstream pressure of a vessel, inclined in any direction, falls below the value at which the transmural pressure causes its walls to buckle inward (Fig. 17).\textsuperscript{10,11} This pressure and its locus are influenced by the rate of upstream inflow in relation to the downstream pressure; no more can be pulled through than is put in. The segment may be long if the collapsing transmural pressure continues downstream. Or it may be short if the transmural pressure is restored, as happens, for instance, where the veins enter the negative pressure domain in the chest. The compliance of a tube changes dramatically as the transmural pressure distorts the cross-sectional area of its lumen. Entering the partially collapsed segment it is high; upstream (fully distended) and in the partially collapsed segment (fully distorted) it is low. Complete stoppage of flow is prevented by the rigidity of the partially collapsed segment and the abrupt rise in transmural pressure that would occur as intravascular pressure upstream of a complete obstruction rose to input pressure. There is instability (“flitter”) when a flow-limiting segment forms, which may be audible as a venous hum.

After deep cogitation we surmised that there must be a flow-limiting or “waterfall” effect in the systemic veins as they enter the chest such that increasingly negative intrapleural pressure would not affect the rate of venous return. For several days, I was happy that we had finally made the Great Discovery. But then Paul found that the work of G. A. Brecher, which had been published 16 years previously,\textsuperscript{3} and then that of J. P. Holt, published 11 years before that.\textsuperscript{11} Both described flow limitation due to partial venous collapse where the veins enter the thorax. Brecher had applied increasingly negative pressures acutely to the downstream end of the vena cava and had shown that, after the pressure had reached $-15 \text{ cm H}_2\text{O}$, further decreases did not affect the rate of flow (Fig. 18). He showed

![Figure 16. Waterfall.](image)

![Figure 17. Venous caliber in relation to transmural pressure. The dramatic change in luminal area when transmural pressure is zero in this schematic causes the waterfall effect. Venous compliance (dotted) is highest at slightly positive transmural pressures (Hoffman and Spaan.\textsuperscript{10})](image)
Reflections on the Waterfalls in the Chest

Figure 18. Negative pressures applied to the downstream end of the superior vena cava (SVC) detached from the right atrium. At -6 cm the cumulative volume extracted increases rapidly during the depleting phase as the veins collapse. At -15 cm and more negative pressures the rates of outflow become the same, limited by the venous collapse (Brecher, Mixter, and Share.2)

that when the pressure falls there is first an increase of flow as the veins empty and partially collapse. This “depleting” phase is followed after 2 or 3 seconds (faster with more negative pressures) by a flow-limitation phase when the partial collapse of the veins prevents any further rise in flow rate. The reader can demonstrate this waterfall effect in the systemic veins by holding his wrist at a level below the chest and observing the distended veins on the back of the hand. Inspiratory efforts against a closed glottis (Mueller maneuver) do not alter their distension, showing that the veins are still emptying into the thorax at the same rate as they are filling on the back of the hand, in spite of very negative intrapleural pressures. However, if the upstream pressure is increased by raising the wrist above the level of the axilla, the veins collapse as blood flow into the thorax increases.

Very negative intrathoracic pressures are necessary during inhalation in severe lower airway obstruction such as asthma but are counterbalanced by very positive pressures during exhalation. Very negative intrathoracic pressures are present with upper airway obstruction during inspiration (snoring, adenoids, etc.), but these are not negated by high exhalation pressures. Were such pressures to cause a torrential inflow of systemic venous blood, the vessels in the chest and the pulmonary vasculature would overdistend and pulmonary edema could ensue. Indeed pulmonary edema has been described in such patients since the diphtheria days.22 Happily the systemic venous waterfall usually prevents such dramatic displacements of blood during sustained inspiratory efforts.

We pointed out this potentially important physiological role in the discussion section of the paper we sent to Circulation Research. Julius Comroe was the new editor of the journal. We got the reviews back in record time; J. C., Jr. regretted that the paper was “unacceptable in its present form” and suggested that we heed the suggestion of one of the referees that we omit our speculations about the “wisdom of the body” in pulmonary edema prevention. We did so but slipped in a little gas exchange teleology because we thought this would be too much for cardiologists to cavil about in a rereview—and indeed it was.21

After getting out of my depth in systemic waterfalls, I decided to continue the search downstream in the friendly flows of the pulmonary circulation.

The measurement of pulmonary venous pressure by passing catheters retrogradely up the pulmonary veins had proved an unrewarding technique. If the catheter is pushed to the wedged position, pulmonary arterial pressure is registered. As the catheter is unwedged and pulled toward the larger veins, the pressure falls, depending on how much the catheter blocks the flow. However, Caro and McDonald have shown that pressures can be measured without obstructing flow if thin catheters with a bell-shaped distension on one end and a point on the other are passed retrogradely out through the lung surface so that the bell-shaped end wedges
in a bifurcation of the vascular system (Fig. 19). We used this method to measure the pressures in the veins of the excised lung. Using several such catheters, we obtained upstream venous pressures in both small and large intrapulmonary veins. These were unaffected during constant flow when the downstream pressures in the left atrium or in the pulmonary veins in the intrapleural space were reduced below about 7 cmH₂O, even to very low pressures. We were able to find the site in the vein—the waterfall or partially collapsed segment—at which there was a sudden fall from upstream venous pressure to the low left atrial pressure. This was just before the veins passed out of the lungs (Fig. 20). When the lung surface was resected back 1 or 2 cm, the abrupt pressure change again occurred inside the new surface. We showed subsequently that we could make similar measurements in the living, open-chested animal and that the results were identical. We were reasonably certain that this discovery of a pulmonary venous waterfall would finally make us famous. However, it turned out that Duomarco and his colleagues had shown the pulmonary venous waterfall effect some fifteen years before. He had even drawn an elegant diagram of the venous narrowing and of the pressure relationships that must exist between the perivenous pressure within the lungs and that outside the lungs in the intrapleural space (Fig. 21). Another Great Discovery that wasn’t!

The position of this waterfall, just within the lungs, remains a puzzle. Although not as low as that around the arteries, the pressure around the veins within the lung has been measured and shown to be similar to that in the intrapleural space. Indeed Fung and his colleagues concluded that extra alveolar vessels in the lung “do not collapse” when subjected to alveolar pressures considerably higher than intravascular pressures. They used the term collapse, however, to signify a complete closure of a vessel, assessed using an elegant silicone elastomer cast technique. Part of the explanation for the position of the venous waterfall can likely be
Reflections on the Waterfalls in the Chest

Figure 21. Diagram of physical factors involved in pulmonary venous hemodynamics. X is the site of collapse. A is the low pressure domain (atmospheric or intrapleural), whereas B is the high pressure domain (intrapulmonary). H' relates intravenous to extrapulmonary pressure at the level of the vein, H to intrapulmonary pressure around the vein. L is the hydrostatic level of the surrounding pressure necessary to deform and collapse the vein when its intravascular distending pressure is \( \alpha \). \( \alpha \) is set by the rate of flow and the frictional resistance upstream of the vein. (From Duomarco, Rimini, and Giambruno.)

found in the work of Younes and others. They have shown that, in a system of collapsible tubes, the intravascular pressure fall toward their downstream ends is compounded by the narrowing it causes. This leads to a further rise in resistance. Also, the compliance of individual veins increases as they get bigger downstream; they have a poor intrinsic resistance to buckling and are less well tethered than are the arteries in the bronchovascular bundles. Presumably they partially collapse and a flow-limiting waterfall segment forms where they are particularly poorly supported just before they exit from the lungs. Although the intravascular pressure at the waterfall is hardly influenced by alveolar pressures up to 15 cmH₂O and the corresponding lung volume changes, it rises when these increase further. Thus near its edge the lung seems to be compressing rather than expanding the veins.

Another piece of this puzzle was put in place some years later by Grant Lee. Using dogs, he and his colleagues showed that the veins lying between the lung and the left atrium in the intrapleural space are easily distensible and, when distended, contain a volume that can be as large as the left ventricular stroke volume. Lee the cardiologist was interested in the damping of the flow pulse from the right ventricle by this distensible reservoir formed by the pulmonary veins. My interest in it, as a respiratory physiologist, was that it must also be a reservoir capable of holding the increase in flow from the partially collapsing intrapulmonary veins at the start of inspiration. This flow increment would otherwise distend the left atrium before the protective pulmonary venous waterfall formed to limit venous outflow from the lungs. Were it not for this waterfall effect, every time a person yawned, there would be an immediate and dramatic increase in cardiac output as pulmonary venous return to the left atrium rose in proportion to the fall in intrapleural pressure. Such a rise in output would be particularly inappropriate for the bored reader.

This paper went to the Journal of Applied Physiology. It was reviewed by two of my friends and finally accepted with these teleological warts untreated.

Are there, then, three waterfalls affecting blood flow in the chest (Fig. 22)? To my knowledge the alveolar waterfall has never been seen or shown by direct pressure measurements, as has the pulmonary venous waterfall. The many elegant studies of its formation have relied on raising pulmonary venous or left atrial pressure, downstream of both waterfalls, and showing an effect on upstream flow or pressure when the waterfall pressure was exceeded. So either, or both, could have caused the changes. However a micro-puncture study of the pressures in 50 µm venules showed no further fall as down-
stream venous pressure was lowered below 7 cmH₂O (alveolar pressure)² consistent with an effect downstream of the alveolar vessels.

The effect of the alveolar waterfall is to prevent more blood flowing through zone 2 of the lungs, when left atrial pressure falls, unless there is an augmented inflow (pulmonary arterial) pressure from a more vigorous right heart. In other words, although acute differences can still occur, it ensures the long-term matching of the stroke volumes of the right and left heart. But the pulmonary venous waterfall serves the same purpose. Must one postulate that there is an alveolar waterfall because pericapillary pressures in the alveoli should be so much higher than perivenous pressures in the extra-alveolar compartment? We now know that surface tension lowers pericapillary pressures, particularly around corner vessels, and that interstitial pressures measured in the extra-alveolar compartment depend on the circumstances and the location. Thus the vascular effect of the alveolar to extra alveolar interstitial pressure difference could have been over emphasized. This remains a fascinating field of study.

The natural waterfall does not have an opaque, plain vascular encasement. Thus, it can be seen to be beautiful. I think that one might be excused, however, for arguing that the waterfalls in the circulation are there for a beautiful physiological purpose. This is arrant teleology and unworthy of a scientist (Fig. 23). But this time I am not worried, for I know that my manuscript must be accepted.

Teleology is like a mistress; a person one cannot do without, but with whom one dare not be seen in public

Claude Bernard

References


