Gravity, the Belly, and the Diaphragm

You Can’t Ignore Physics

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Using a radiologic technique, the position and pattern of movement of the diaphragm have been evaluated in three adult volunteers, both awake and anesthetized, during spontaneous ventilation and with muscle paralysis and mechanical ventilation. Studies were made with the subjects in supine and left lateral decubitus positions with tidal and large-volume breaths. Positive end-expiratory pressure (PEEP) was added in studies of two subjects. During spontaneous ventilation awake or anesthetized, because of regional mechanical advantages, the dependent part of the diaphragm had the greatest displacement despite the higher intraabdominal pressure in this region. Paralysis, awake or anesthetized, caused a cephalad shift of the end-expiratory position of the diaphragm that was disproportionately large in dependent regions. It also reversed the pattern of diaphragmatic displacement. The passive diaphragm was displaced preferentially in nondependent zones where abdominal pressure is least. Consequently, PEEP could not restore the diaphragm to its awake functional residual capacity position, and large breaths also could not duplicate the pattern of displacement achieved spontaneously.

ABOUT one original research article per month continues to use the findings of the 1974 study cited above1 in its rationale for the design of investigations affecting anesthesia or intensive care. I gather that makes it a classic.

I arrived in Toronto, Ontario, Canada, in 1972, fresh from 2 stimulating years in the Harvard University anesthesiology residency program at the Massachusetts General Hospital (Boston, Massachusetts) to round out my anesthesiology training with a year of research at the Hospital for Sick Children. I found myself the research fellow of an incredibly innovative, productive respiratory physiologist, Charlie Bryan, M.B., B.S., Ph.D., F.R.C.P.C. (then Associate Professor in the Department of Anesthesia, University of Toronto, Toronto, Ontario, Canada, and Director of Respiratory Research in the Research Institute at the Hospital for Sick Children). I soon learned that his anesthesia credentials apparently originated from his ability to operate the hyperbaric chamber at the hospital next door. Lungs were his fascination, as they were mine.

Only later would I learn Charlie’s background. Charlie’s route to respiratory physiology expertise was typically unorthodox. Early in his career as a General Practitioner in rural Britain, he had an emergency nighttime summons to a farmhouse in a bitter storm to treat a baby that had been crying inconsolably for hours. Charlie’s examination revealed no cause until he stripped off the baby’s brand new vest to examine the chest. The cure was instantaneous as the staple on the vest’s price tag stopped poking into the baby’s back. The next morning, Charlie went down to London, where the New Zealand navy rejected him, but he was accepted into the Canadian Air Force. The management of high-altitude accidental decompression was a hot topic at the Institute for Aviation Medicine, and soon, Charlie was sent to McGill University in Montreal to obtain a Ph.D. in 1964 using the exciting new radioactive xenon techniques to study the distribution of ventilation and perfusion in the lung with David Bates, C.M., M.D., F.R.C.P., F.R.C.P.C.,
Fig. 1. Model of a fluid-filled container with two flexible walls (A, left). Depending on the characteristics of the fluid and the container, an inflection point will develop along the vertical flexible wall, above which pressure will be negative and below which fluid pressure is increasingly positive. In A, right, the resultant shapes of diaphragm and abdomen reflect the balance of forces across these structures in an upright posture. (B) Supine, the “flexible walls” change orientation with respect to gravity. Below the anterior abdominal wall, pressure is now positive or negative, depending on body habitus, whereas pressure against the diaphragm steadily increases toward the vertebral column. Question: What impact will anesthesia/paralysis have in a system with such an inhomogeneous distribution of forces across the diaphragm?

F.A.C.P., F.R.S.C. (then Professor of Medicine at McGill University in Montreal, Quebec, Canada, and Head of the Division of Respiratory Medicine at the Royal Victoria Hospital; now Professor Emeritus at the University of British Columbia, Vancouver, British Columbia, Canada). Later, Charlie’s move to the Toronto General Hospital to operate the hyperbaric chamber left him plenty of time to hang around the Respiratory Failure Unit with Alan Laws, B.Sc., M.B., B.S., F.R.C.P.C. (then Assistant Professor of Anesthesia at the University of Toronto; 1936–1995), who was busy investigating issues such as the decrease in functional residual capacity with anesthesia and paralysis and the effect of positive end-expiratory pressure on lung volume and gas exchange during mechanical ventilation of the sick lung. To that point, it was the lungs—not the container around them—on which people focused in the intensive care unit. About this time, a figure in the Handbook of Physiology grabbed Charlie’s attention.² Duomarco and Rimini² in 1947 had emphasized the impact of abdominal contents on the shape and position of the diaphragm. They modeled the abdomen as a fluid-filled container with two flexible walls, the ventral surface and the diaphragm (fig. 1A). Turn Rimini’s “container” into the horizontal position favored in anesthesia and intensive care, and the diaphragm’s response to paralysis looked extremely important to Charlie (fig. 1B).

I soon learned Charlie could throw out hypotheses faster than I could chase them. I also became increasingly aware that his notion of a feasible experiment far exceeded my comfort level. One day, he pulled out some chest radiographs of himself taken apparently at end-inspiration and end-expiration on some memorable previous occasion when he had been given succinylcholine to see what paralysis did to the diaphragm. I was told that “they lost his airway” and the experiment had been abruptly curtailed. The x-rays were not particularly convincing. The idea behind them was. It was clear that I was the one being appointed to pursue this burning question.

By that time, I had mastered the ground rules of working with Charlie. The most important rule was that if you had a lot of preparation to do, it was best to hide and hope Charlie would busy himself with something else long enough for you to work out the methodology. I hid, mainly in the radiology department. Fortuitously, the new head of the nuclear medicine department at Sick Kids was David Gilday, B.Eng.(EE), M.D., C.M., F.R.C.P.C. (then Assistant Professor of Radiology, University of Toronto; now Professor of Radiology in Toronto and still Head of Nuclear Medicine at the Hospital for Sick Children)—someone I knew from my medical school and internship days at the University of Manitoba in Winnipeg, Manitoba, Canada. David helped me to develop a protocol with a tolerable radiation dose using the “new technology” of the 1970s. Instead of multiple plain films, an image intensifier would be used to obtain cross-table videos of diaphragmatic movement, with a system of radiopaque markers to provide clear reference levels and magnification factors. Interestingly, almost identical methodology was used recently in a study of patients with chronic obstructive pulmonary disease.³

My one “nonnegotiable” was that no airways would be lost in the pursuit of this hypothesis, and so I started my series of awake blind nasotracheal intubations of supervisors and, later, my colleagues. Not surprisingly, I was afraid “volunteers” would pose a problem. However, Charlie, as usual, was determined to go first. Alan Laws, the anesthetist from the Toronto General Hospital who had pioneered the use of positive end-expiratory pressure in the intensive care unit, was a willing second, and Al Conn, M.D., B.Sc.(Med), F.R.C.P.C. (then Professor of Anesthesia, University of Toronto, and Director of the Intensive Care Unit, Hospital for Sick Children; now Professor Emeritus) became our third. A fourth keen volunteer had to be turned down because of previous thoracic surgery, and I was told by the ethics board that a research fellow could not volunteer for his or her own study because of the potential coercive effect of being desperate to get enough experimental subjects. That left our “n” at 3.

After several mock rehearsals with the radiology team, I was ready to go. The “protocol” was executed without a glitch. A preliminary review of the videos confirmed that we were getting the data we needed, and we proceeded with our other volunteers. Then, it was time to hide from Charlie again. Long hours followed, measuring diaphragmatic displacement frame by frame on transparency traced from the video screen. With obsessive attention to detail, every breath was analyzed for both supine and lateral decubitus positions (fig. 2).
Charlie was right (as usual). As expected from Duomarco and Rimini's model, the dependent diaphragm was displaced cephalad by anesthesia or paralysis. The nondependent diaphragm moved cephalad or caudad depending on body shape, size, posture, and the resulting pressure in that region of the abdomen. What was totally unpredictable was the reversal of regional diaphragm displacement we observed with paralysis. An actively contracting diaphragm displaced abdominal contents in dependent regions despite the increased load. A paralyzed diaphragm moved predominantly in nondependent areas where the abdominal pressures resisting displacement were least. The regional distribution of gravitational effects could not be ignored in horizontal postures.

I proceeded to write up our findings both as a manuscript and as an essay for the American Society of Anesthesiologists Annual Residents' Competition. A separate thorough literature review was a requirement for the latter. And that's when I hit bottom. As I reviewed the existing literature I came to the horrifying realization that all the clues needed to recognize the profound impact of paralysis on diaphragmatic mechanics were already out there. We could have deduced the concepts without ever doing the experiments. I went to Charlie in despair. We had been scooped by the library holdings! I have never forgotten Charlie's reassuring response. He explained that part of the job of a researcher is to package observations experimentally in ways that suddenly make sense of all the scattered unrelated clues that are out there. That is when the next stage of learning and application takes off. And our "n" of 3 had provided that package. That was enough to make it "original research."

Reassured, I sent off my submission. I was delighted to find I had won the first prize in the American Society of Anesthesiologists Resident Competition for 1973 and was invited to present my data at the Annual Meeting of the American Society of Anesthesiologists in San Francisco. The biggest problem seemed to be that nobody knew whether the prize could be awarded for work done in Canada. The compromise was to credit my project to both the Massachusetts General Hospital and the Hospital for Sick Children. Many interesting presentations and bull sessions with other researchers followed. Excitement grew as we identified further aspects of anesthesia and intensive care that were potentially impacted by our new understanding of diaphragm mechanics. And we waited, and waited, and waited for our response from ANESTHESIOLOGY. Finally, we contacted the Editor of ANESTHESIOLOGY and asked him to pursue the delay. A few weeks later, I received the reviews. One was a fairly long, thoughtful review that directed some useful revisions. The second was about a six-line commentary that basically said that our conclusions were so radical they had to be wrong. Fortunately, the Editor chose to go with the longer review, and the manuscript was finally accepted.

In the years since, that ANESTHESIOLOGY article has generated repeated requests for permission to reproduce one or more figures in a host of textbooks of anesthesia and respiratory physiology. Those requests continue to this day. Although the technology available became increasingly sophisticated such that three-dimensional reconstructions of the entire thorax could be produced by Kai Rehder and his group at the Mayo Clinic, the basic conclusions of that rather primitive "n of 3" study remain sound. Active contraction of the diaphragm definitely expands dependent lung regions better than a passive imposed breath of similar volume in supine and lateral decubitus positions. In fact, an actively "stiffened" diaphragm can make supine regional lung volumes and ventilation more homogeneous. Conversely, prolonged mechanical ventilation of the diseased lung in the supine position is characterized by loss of aeration in dependent regions.

The first impact of our data on us was the realization that the supine position was not good for lungs, especially when one was not breathing spontaneously. Charlie got the word out. This postulate quickly stimulated studies of ventilation in the prone position, studies that continue to the present (more than 50 articles on the topic in the past 4 yr). The prone position has never been proven to be a bad idea with respect to the lungs, just an idea difficult to implement and therefore generally resisted. During the past 20 yr, tremendous energy has been devoted to the manipulation of ventilator patterns and design in an attempt to preserve the supine

**Fig. 2.** Composite of some original records traced from the video screen under various conditions. ALA = awake, left lateral, breathing spontaneously unintubated; ASA = awake, supine, breathing spontaneously unintubated; SLPar = anesthetized, left lateral, paralyzed; SSPar = anesthetized, supine, paralyzed; SSS = anesthetized, supine, breathing spontaneously. Note: Not all frames are from the same subject.
position of patients in our intensive care units. Nonetheless, both recent expert-opinion articles about ventilator management of patients with acute respiratory distress syndrome recommend use of the prone position, albeit with differing criteria for its institution.\textsuperscript{11,12}

Our data also provided a mechanism to explain common phenomena such as the impairment of oxygenation during general anesthesia, plus patterns of redistribution of regional volume, ventilation, and positive end-expiratory pressure in a variety of postures and scenarios. As I have investigated the impact of ventilator patterns on lung injury in subsequent years, those 1974 concepts of diaphragm mechanics have provided sound guidance. Recent demonstrations of the value of maintaining some spontaneous ventilation during prolonged ventilatory support of supine patients with lung injury continue to prove that the spontaneously contracting diaphragm preserves dependent lung volume and ventilation better than any passively imposed pressure pattern yet devised\textsuperscript{13} while also minimizing disuse atrophy of that muscle.\textsuperscript{14} Gravity still can’t be ignored.

A. Charles Bryan, M.B., B.S., Ph.D., F.R.C.P.C., was an Associate Professor in the Department of Anesthesia, University of Toronto, Toronto, Ontario, Canada, and Director of Respiratory Research in the Research Institute of the Hospital for Sick Children in Toronto at the time of our study. In 1991, he became Professor Emeritus of Anesthesia and Pediatrics at the University of Toronto. Prior to the submission of this article, Charlie supplemented my memory of events relative to our experiments. Surprisingly, we agreed on most things despite the passage of 32 yr! Dr. Bryan died on August 1, 2005.

\textbf{References}

7. Froese AB: Anesthesia-paralysis and the diaphragm: In pursuit of an elusive muscle. \textit{Anesthesiology} 1989; 70:887–90