



THE STRESS INDEX

by John Marini MD

A primary objective in modern ventilator care is to avoid injuring the already damaged lung. Investigators generally agree that over distention of open, aerated tissues and tidal opening and closure of unstable ones should both be avoided, especially when high inflation pressures are used. For many years, clinicians have sought simple ways to determine the PEEP levels and tidal volumes that accomplish effective gas exchange without placing undue stresses (tensions, strains) on delicate airways and lung tissues. Because the compliance of the respiratory system changes as the lung opens up or over expands, the contours of the inflation pressure-volume curve have been considered to offer valuable - if imperfect - indicators of the safety and danger zones of operation. According to common wisdom, the inspiratory PV curve - volume on the vertical axis, pressure on the horizontal one - has a sigmoid shape. Concave at the beginning of inflation, the curve straightens out through much of its course and then becomes convex as over inflation predominates on the approach to total lung capacity (TLC).

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Although conceptually useful, this attractive but oversimplified interpretation is based more on single compartment (balloon-like) behavior than on the more realistic multi-compartment model of the damaged lung. Computed tomography has shown us that both recruitment and over distention can (and routinely do) occur at the same time in different sectors of the heterogeneous lung. The contours of the curve are the volume-averaged synthesis of contributions coming from lung regions with dramatically different mechanical characteristics. Here it should be pointed out that the slope of the PV curve is also influenced by the inflation properties of the chest wall, a confounding issue of some importance at low thoracic volumes. Ignoring the chest wall for the moment, the initial region of rapidly changing P-V slope could be said to represent the "recruitment zone" where the number of overstretched units is comparatively low in comparison to those that are coming "on-line". As the lung inflates further, curve shape reversal signals that progressively larger volumes of overstretched lung predominate over those that are undergoing recruitment. The linear portion of the inspiratory PV curve often has been interpreted to be the "safe zone". (But maybe not so "safe" for all lung regions--see later.)

Because time directly reflects volume when flow holds constant, advantage has been taken of this fact to construct "quasi-stat-

ic" P-V curves using very slowest rates of inspiratory flow (<5 L/min) in patients who are passively inflated. In that way, the pressure needed to overcome airflow resistance is kept negligible, allowing the airway pressure profile to reflect the alveolar component of pressure. The resulting pressure-time curve mimics the static inspiratory P-V profile quite well. This "slow flow PV curve" option is available on some machines we use clinically.

It is but a short step from the construction of such "quasi-static" curves inscribed during very low flow to the analysis of the pressure-time contours during passive tidal breathing at normal rates of "square wave" (constant) flow. Assuming flow and resistance are unchanging as the chest inflates, so too is the amount of pressure needed to overcome the flow-resistive component of the work of breathing. Under such conditions, the passive profile of tidal airway pressure is influenced primarily by the underlying elastic properties and therefore reflects the static P-V relationship. Today's microprocessor-driven ventilators precisely regulate inspiratory flow and have almost unlimited analytical capacity. Consequently, it is relatively easy for such machines to fit a smooth curve to the pressure data points in order to access the shaping information they contain.

When an algebraic expression of $y = mx^Z + b$ is fit to the inflation pressure tracing, the exponent "Z" becomes a numerical indicator of curvature. When this exponent is 1, airway pressure rises in a straight line over time; when $Z < 1$, the tracing is concave to the time (think 'volume') horizontal axis; and when $Z > 1$, the curve is convex to it. In other

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words, a $Z < 1$ indicates less pressure needed for each volume increment (implying tidal recruitment predominates over excessive stress), and $Z > 1$ indicates pressure rising progressively fast (implying the converse—over-distention predominates over that range). Theoretically, the objective should be to keep “Z” approximately 1.0 during tidal ventilation, with the PV relationship linear and breathing occurring in the “safe” zone. In fact, there are intriguing laboratory data to indicate that stress indexes that differ from 1.0 correlate with inflammatory cytokine release into lavage fluid and perhaps into the bloodstream as well. If so, this shaping parameter (Z), proposed as a “stress index”, may convey important information of value to the caregiver. Although this simple interpretation is conceptually logical, reality is a bit more complicated than that because of concerns regarding the chest wall contribution, the mechanical heterogeneity of the diseased lung, and a few (as yet) unsettled technical issues. Such topics are worth thinking about, since the stress index might offer some bedside help and is now being wired into some of the latest ventilators offered.

A better interpretation of $Z=1.0$ might be “not obviously un-safe”, as it is perfectly possible to have large areas of the lung being overstretched and other areas being recruited at the same time, with all data merged at the airway opening into a linear pressure-time tracing. What should a thoughtful clinician’s interpretation be if the stress index were 1.0 and the plateau pressure 45 cm H₂O? Knowing that CT studies have shown that over-distention may occur in some parts of the injured lung at pressures < 30 cmH₂O, it would not seem prudent to continue without

concern and a darn good explanation of the otherwise dangerous tidal ventilating pressure (e.g., very stiff chest wall). Moreover, because the stress index cannot separate chest wall properties from lung properties, values that deviate seriously from 1.0 do not always indicate lung tissue at risk for VILI. Without PEEP, the pressure-volume (compliance) properties the chest wall change radically at volumes near FRC, often accounting for the lower inflection zone of the static PV curve and, by inference, a $Z < 1.0$, without the need to invoke tidal recruitment. At the other end of the volume range (approaching total lung capacity), the normal chest wall is very compliant. Consequently, $Z > 1.0$ suggests that large portions of the lung are indeed being overstretched. On the other hand, a stiff chest wall may limit or prevent lung over-distention but produce an unnecessarily worrisome $Z > 1.0$ for the entire respiratory system as it approaches its ‘paper bag-like’ or ‘stop-length’ limit.

Apart from the interpretive problems presented by the chest wall and mechanical heterogeneity, a number of purely technical questions - if not actual issues - remain to be answered for the Stress Index. The assumption that the shape of the airway pressure curve primarily reflects the elastic properties of the chest seems quite defensible. Nonetheless, although I believe the stress index to be relatively robust to Δ 's of flow rate, it may not be entirely unaffected by them. For example, although a zone of constant flow can be assured by discarding the initial and terminal portions of the pressure tracing, how constant is airway resistance over the tidal range? (Secretions and cardiac pulsation may cause undulations and vibrations that have little to do with parenchyma elastance. Fortunately, appropriate mathematical smoothing of the curve helps to average out these disturbances.) However constant it might be, does the amplitude of flow rate influence the calculated Z value? Do tidal volume variations influence the calculated stress index? After all, not much shape change might be evident or present at very small tidal volumes. Exactly how much deviation from the ‘ideal’ 1.0 should worry us in the clinical setting? How do variations in body position affect it? Some of these questions simply have not yet been studied in depth but should have answers that are not “deal breakers” for the SI.

Astute therapists and physicians have always been attentive to the contours of the airway pressure profile and have made adjustments when clearly prompted by corroborating clinical indicators. Visual inspection of the airway pressure tracing, however, is imprecise and can be misleading if flow regulation is inexact or inconstant, if resistance varies, if tidal volumes are small, if the tracing time scale is too long to facilitate visual interpretation, and especially if the patient is not entirely passive. By automatically and numerically characterizing the shape change of the pressure tracing that occurs during passive ventilation with rigidly constant flow, the stress index is a step forward and a welcome addition. I feel relatively sure of this. Alone, however, the stress index is not a sufficient guide for keeping the patient safely ventilated. As with most monitoring tools, it needs to be integrated with other numerical data and, most importantly, with the observational skills and critical thinking ability of the conscientious bedside practitioner.

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