Anesthesiology 77:403-404, 1992

## Intracranial Elastance *versus* Intracranial Compliance: Terminology Should Agree with That of Other Disciplines

To the Editor:—In clinical medicine and research, it often is beneficial to describe the relationship between the forces of pressure acting on a body—or, alternatively, the pressure within a space—and the volume of the body or space. In the respiratory system, changes in lung volume in response to changes in transpulmonary pressures are used to characterize lung mechanics. <sup>1,2</sup> Similarly, in neurology, neuroanesthesia, and neurosurgery, one often assesses the well-being of the intracranial contents by describing the changes in intracranial pressure in response to changes in intracranial volume. <sup>5,4</sup> For example, a large increase in pressure in response to a small increase in volume suggests that the compensatory reserves of the intracranial contents are reaching or have exceeded their limits, and suggests the presence of underlying pathology such as an intracranial mass lesion.

The standard terminology used to describe pressure-volume relationships, or alternatively, volume-pressure relationships, has existed in the literature for decades.<sup>5</sup> By convention, elastance is defined as  $\Delta P/\Delta V$  (where  $\Delta P=a$  change in pressure and  $\Delta V=a$  change in volume).<sup>5,6</sup> Compliance is defined as  $\Delta V/\Delta P^{5,6}$  and thus is the reciprocal of elastance.<sup>5,6</sup>

With this as background, it is thus surprising that among neuroanesthesiologists, neurosurgeons, and neurointensivists the terminology for pressure–volume relationships has so frequently been misused. Specifically, the relationship in figure 1A is typically described inappropriately as an intracranial compliance curve. For example, this terminology has previously been misused by one of us (WLL) in a report to this journal. In addition, in a recently published anesthesiology text, the authors of the chapters addressing cerebral physiology and intracranial hypertension initially (and correctly) state that the relationship  $\Delta P/\Delta V$  is that of elastance. Then, acknowledging the "well entrenched misuse of terminology," they revert to traditional neuroanesthesia terminology. In label a figure demonstrating  $\Delta P/\Delta V$  as one of "compliance". The confusion in terminology in neurologically impaired patients is further compounded if the relationship in figure 1A is referred to as a "cerebral compliance curve." The relationship is not between the volume and pressure of the cerebrum, but instead between the volume and pressure of the intracranial space.

Others who are perhaps aware of these controversies and do not wish to perpetuate the error have relied on yet other terminology. Neuroscientists have introduced indices calculated from pressure and volume measurements, e.g., the so-called "pressure-volume index."

This path of least resistance in terminology has been observed in other medical disciplines as well. In reference to the eye, authors have referred to the ocular  $\Delta P/\Delta V$  relationship as that of ocular "distensibility." Some cardiovascular researchers, perhaps wishing to avoid the controversy altogether, have used the term "pressure-volume relationship." Aside from these alternative approaches, it is of interest that, when used in reference to systems other than the intracranial space, there are few published examples of misuse of the elastance/compliance terminology. 12

To improve communication among scientists and practitioners of medicine, it is important that all parties use similar terminology to describe similar events. Thus, the term compliance should be used to describe the relationship  $\Delta V/\Delta P$ , and the term elastance should be used to describe the relationship  $\Delta P/\Delta V$ . Specifically, the term "intracranial elastance" should be used to describe the slope of the curve shown in figure 1A. (The reader will note that an alternative to changing the terminology is to retain the "compliance" terminology and change the shape of the curve to that in figure 1B. This is not recommended since pressure becomes the independent variable and volume becomes the dependent variable, rendering the display of the curve physiologically less valid.)

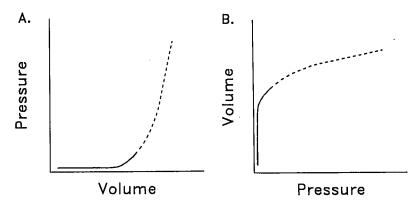
Clinicians will recognize that the dashed portion of the curve in figure 1A represents a large increase in pressure in response to a small increase in volume. This has been referred to previously as a state of reduced cerebral compliance or reduced intracranial compliance. In place of this incorrect use of terminology, we suggest that the state be more appropriately called "increased intracranial elastance," or if this is unpalatable, "abnormal intracranial elastance."

WILLIAM L. LANIER, M.D. Neuroanesthesia Research Laboratory

DAVID O. WARNER, M.D. Respiratory Physiology Laboratory

Department of Anesthesiology Mayo Clinic and Mayo Medical School Rochester, Minnesota 55905

FIG. 1. The relationship between volume and pressure of the intracranial space. A: Intracranial elastance, defined as a change in pressure in response to a change in volume. B: The same data have been rearranged to depict intracranial compliance, defined as a change in volume in response to a change in pressure. Clearly, the relationship depicted in A, the intracranial elastance curve, is the more clinically relevant and is the more often used of the two relationships. Once the compensatory reserves of the intracranial space are exceeded, there will be large increases in pressure in response to small increases in volume (A, dashed line). This state of "increased elastance" is easily remembered because the elastance increases as the slope of the line increases.



## REFERENCES

- Rohrer F: Der Zusammenhang der Atemkrufte und ihre Abhungigkeit vom Dehnungszustand der Atmungsorgane. Pflugers Archiv (Berlin) 165:419–444, 1916
- Wirz K: Das Verhalten des Druckes im Pleuraraum bei der Atmung und die Ursachen seiner Verunderlichkeit. Pflugers Archiv (Berlin) 199:1-56, 1923
- Lanier WL, Weglinski MR: Intracranial pressure, Clinical Neuroanesthesia. Edited by Cucchiara RF, Michenfelder JD. New York, Churchill Livingston, 1990, pp 77-115
- Shapiro HM, Drummond JC: Neurosurgical anesthesia and intracranial hypertension, Anesthesia. 3rd Edition. Edited by Miller RD. New York, Churchill Livingstone, 1990, pp 1737–1789
- Mead J, Milic-Emili J: Theory and methodology in respiratory mechanics with glossary of symbols, Handbook of Physiology: Respiration. Section 3, Volume 1. Edited by Fenn WO, Rahn H. Washington, DC, American Physiologic Society, 1964, pp 363-376
- Dorland's Illustrated Medical Dictionary, 27th Edition. Edited by Taylor EJ. Philadelphia, WB Saunders, 1988

- Lanier WL, Milde JH, Michenfelder JD: Cerebral stimulation following succinylcholine in dogs. ANESTHESIOLOGY 64:551–559, 1986
- Drummond JC, Shapiro HM: Cerebral physiology, Anesthesia.
   3rd Edition. Edited by Miller RD. New York, Churchill Livingstone, 1990, pp 621-658
- Maset AL, Marmarou A, Ward JD, Choi S, Lutz HA, Brooks D, Moulton RJ, DeSalles A, Muizelaar JP, Turner H, Young HF: Pressure-volume index in head injury. J Neurosurg 67:832–840, 1987
- Aboul-Eish E: Physiology of the eye pertinent to anesthesia. Int Ophthalmol Clin 13:1-20, 1973
- Smiseth OA, Frais MA, Junemann M, Kingma I, Refsum H, Smith ER, Lipton MJ, Tyberg JV: Left and right ventricular diastolic function during acute pericardial tamponade. Clin Physiol 11: 61-71, 1991
- George NJR: Technique and theory of cystometry, Diagnostic Techniques in Urology. Edited by O'Reilly PH, George NJR, Weiss RM. Philadelphia, WB Saunders, 1990, pp 299–307

(Accepted for publication May 19, 1992.)

Anesthesiology 77:404-405, 1992

## Intensive Analgesia Reduces Postoperative Myocardial Ischemia? I

To the Editor:—An article by Mangano et al. demonstrates that the incidence of severe ischemic episodes is decreased in patients who receive intravenous sufentanil for postoperative pain control. Overall outcome, however, was not improved. The incidence of myocardial infarction and left ventricular failure was not statistically different from one group to another.

To demonstrate clearly that any treatment would decrease the incidence of adverse outcome, a large study is needed. For example, patients undergoing peripheral vascular surgery have a 40% incidence of postoperative ischemia, and 30% of those develop a bad outcome (death, myocardial infarction, or death).<sup>2-4</sup> If a therapy is to reduce the incidence of complications from 30 to 20%, approximately 3,000 patients would be required to demonstrate that aggressive therapy is useful (power of 0.80 for a 33% reduction in complications). Thus, unless a multicenter study enrolling a significant number of patients is planned, definitive conclusions will be difficult to draw.

The incidence of postoperative ischemia may be closely related to the activation of the neuroendocrine response. In fact plasma norepinephrine and renin levels are higher during episodes of silent ischemia. In healthy humans, the coronary vessels dilate due to endothelium-derived relaxing factor and prostacyclin produced in response to acetylcholine and norepinephrine. When atherosclerosis is present, this stimuli will result in coronary vasoconstriction. Moreover, postoperative hypercoagulability is experienced by patients with atherosclerotic disease. This vasoocclusive phenomenon acting synergistically with a high adrenergic tone may be responsible for the high incidence of postoperative myocardial ischemia and bad outcome in patients with coronary artery disease.

We suggest that postoperative epidural analgesia using a local anesthetic and opioid in low doses may be associated with a better outcome in this high-risk population. <sup>8,9</sup> Moreover, a short-acting  $\beta$  blocker and/ or calcium-channel blocker, when indicated, may further reduce the incidence of ischemia associated with supply-demand or spasm. <sup>10,11</sup>

The use of intravenous opioids, on the other hand, not only provide inferior analgesia when compared with epidural opioids but are also unable to suppress hemodynamic and hormonal responses even when high doses are used.<sup>12</sup> Since no improvement in cardiac outcome could be derived from postoperative infusions of sufentanil, based on these data, one must evaluate the effects of these infusions (cost, intubation time, complications, *etc.*) with the potential benefits (improved clinical outcome).

OSCAR A. DE LEON-CASASOLA, M.D. Assistant Professor of Anesthesiology SUNY at Buffalo Director, Acute Pain Service Roswell Park Cancer Institute Elm and Carlton Streets Buffalo, New York 14263

MARK J. LEMA, PH.D., M.D.

Head, Department of Anesthesiology and
Critical Care Medicine
Roswell Park Cancer Institute
Director, Anesthesia Research
SUNY at Buffalo
Buffalo, New York 14214

## REFERENCES

- Mangano DT, Siliciano D, Hollenberg M, Leung JM, Browner WS, Goehner P, Merrick S, Verrier E, SPI Research Group: Postoperative myocardial ischemia: Therapeutic trials using intensive analgesia following surgery. ANESTHESIOLOGY 76:342–353, 1992
- McCann RL, Clements FM: Silent myocardial ischemia in patients undergoing peripheral vascular surgery: Incidence and association with perioperative cardiac morbidity and mortality. J Vasc Surg 9:583–587, 1989