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*Harold L. Rekate*

Section of Pediatric Neurosurgery,
Division of Neurological Surgery,
Barrow Neurological Institute,
Phoenix, Ariz., USA

# Brain Turgor (Kb): Intrinsic Property of the Brain to Resist Distortion

In previous reports related to a computer simulation of a mathematical model of ventricular volume regulation, we introduced a mathematical term called Kb [9, 10, 12]. This term was a mathematical modifier or 'fudge factor' that described an intrinsic property of the living brain related to the energy required to distort the brain itself. This term was given a value from zero to one. A Kb of zero indicated a complete absence of resistance to distortion. In other words, any additional volume of cerebrospinal fluid (CSF) or other growing mass led to a diminution of cerebral volume with no increase in intracranial pressure (ICP). In engineering terms, this situation is analogous to a totally viscous material that has no propensity to resume its previous shape after distortion.

In our model, a Kb of one indicates that with any attempt to increase the intracranial volume, there is a rapid rise in ICP with no decrease in cerebral volume. The incorporation of this term in our model has allowed us to describe the pathophysiology of such enigmatic conditions as normal pressure hydrocephalus (NPH) and pseudotumor cerebri (PC) [9]. The purpose of the present work is threefold: (1) to describe how Kb (brain turgor) is useful in understanding the pathophysiology of hydrocephalus, describing what is known of the biologic substrate that goes into Kb; (2) to link Kb with other descriptors of ICP dynamics, such as compliance, elastance, and pressure volume index (PVI); and (3) to describe how Kb can lead to innovative approaches to the management of confusing problems of CSF dynamics.

## The Biologic Substrate of Kb

The Monro-Kellie hypothesis describes the intracranial compartment as an interacting system of three fluid compartments contained within a fixed intracranial volume. Whenever volume of any sort is added to the intracranial compartment, whether in the form of an enlargement of the three intracranial compartments or the addition of an unwelcome mass such as hematoma, tumor, or abscess, room for this new addition must be made by displacing fluid from one or several of the other compartments. The three fluid compartments include the blood in venous sinuses and large cortical veins, the CSF, and the brain parenchymal fluid. The latter has often been assumed to represent brain extracellular fluid (ECF), but for the purposes of this discussion, it will be assumed to include not only ECF but also the blood volume present within the brain parenchyma (fig. 1).

Water, and by analogy, CSF and blood are incompressible. Studies of brain viscoelasticity as removed, nonliving tissue, have also documented the incompressible nature of brain tissue [4]. Changes in the total volume of brain tissue can therefore occur as a result of two processes that may interact. The first process is the loss of brain tissue or parenchymal elements. The second process is the displacement of fluid (water or blood) from the brain substances. Brain tissue may be lost in the case of longstanding hydrocephalus or if the hydrocephalus is refractory or not shunt responsive [7]. However, in most instances, the placement of a shunt or its repair leads to such a rapid expansion of cortical mantle that changes in the fluid

Harold L. Rekate, MD
Barrow Neurological Institute
350 West Thomas Road
Phoenix, AZ 85013 (USA)


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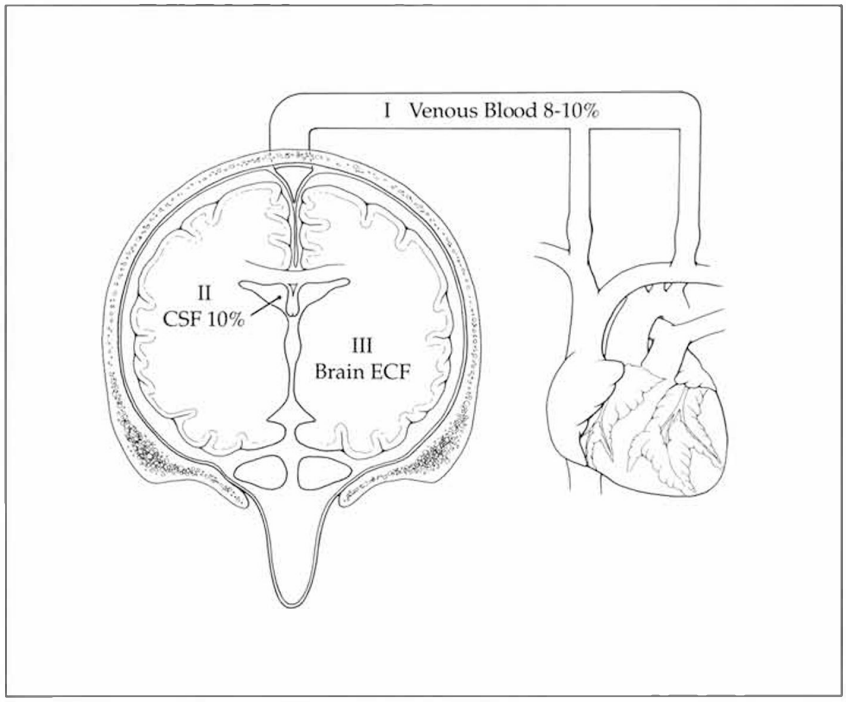


**Fig. 1.** Schematic diagram of the interrelationships among the three intracranial compartments as described in the Monro-Kellie hypothesis.

content of the cerebral tissue become the most likely explanation for these volumetric changes.

Based on this discussion, is seems likely that the Kb (i.e., the brain turgor term) is a descriptor of several interdependent elements: the density of brain parenchymal elements, which is more or less fixed; intra- and extracellular water; and the intraparenchymal blood volume. This blood volume depends on autoregulation and resistance to venous outflow. The brain parenchymal elements include neurons with their axonal arborizations and glial and vascular tissues. These elements are essentially measured by wet weight/dry weight determination. The higher the percentage of tissue to water or fluid, the greater the change that will be created in the system by an incremental change in the fluid space.

The second element contributing to Kb is cerebral blood volume within the interstices of the brain especially related to the resistance to removal of this element from the tissue itself. The presence of a large volume of blood within the capillaries or venules of the brain usually indicates a very high Kb. However, if this blood can easily be displaced into the cortical veins or sinuses, the Kb can be low despite high cerebral blood volume. The presence of a high cerebral blood volume also results from an increase in cerebral blood flow with dilatation of the precapillary arterioles. In each of these cases, the turgor (Kb) of the brain is increased.

The final element in making up Kb is brain water and the resistance of flow of this fluid from the brain substance. This fluid can exist within the cells themselves as in cytotoxic edema. When the fluid is intracellular, it is trapped there and must await cell death or repair to be removed. When the fluid is extracellular, such as normal brain ECF or vasogenic edema, clearance of the water is via bulk flow into the ventricle and absorption with the remainder of CSF [5, 14]. It is therefore reasonable to assume that the same volumetric increase in water will lead to a greater increase in Kb when that increase is intracellular than when it is extracellular.

### Relation of Kb to Measures of Compliance

Clearly, Kb is related to cerebral compliance and its inverse, cerebral elastance. In fact, it represents that portion of cerebral elastance contributed by the brain parenchyma. However, calling it elastance or compliance confuses the issue since these terms deal with the entire

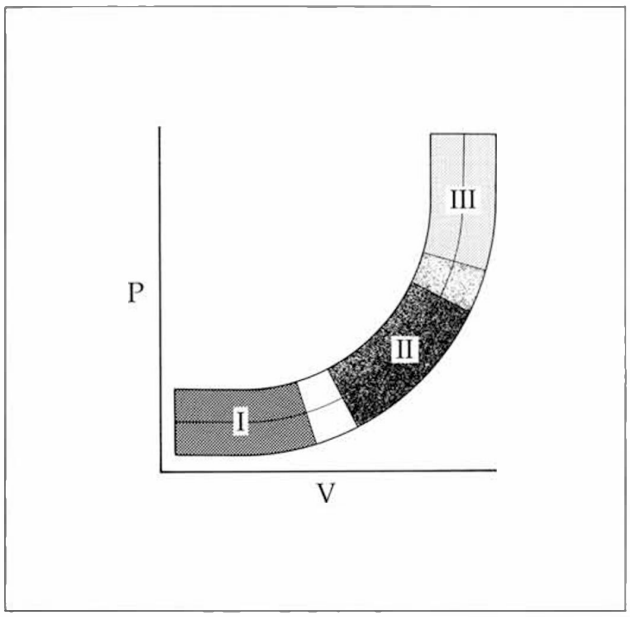


**Fig. 2.** Theoretical graph of ICP versus volume demonstrating the three fluid compartments and their overlapping provinces [from 18, with permission].

intracranial compartment as well as the spinal subarachnoid space (SSAS). Figure 2 is a schematic of the relationship of pressure to volume of the intracranial compartment as it relates to the fluid components of the Monro-Kellie hypothesis. The first compartment of this curve is primarily the province of venous blood in the major venous sinuses and cortical veins. Additions of volume to the intracranial compartment result in the displacement of venous blood under little, if any, resistance to outflow. This venous blood represents about 15% of intracranial volume. As the volume of this compartment becomes depleted, CSF begins to be displaced from the intracranial compartment with some overlap of these elements as shown in figure 2. CSF exits by two mechanisms. Absorption of CSF is increased as ICP increases [3]. Also, as volume is added to the intracranial compartment, CSF is displaced into the SSAS. In the lumbar region, the volume of the SSAS can increase by as much as 30% with distention of the lumbar dura at the expense of the lumbar epidural veins.

Several mathematical formulations have been devised to describe the pressure-volume relationships within the CSF compartment. Avezaat et al. [1, 2] derived equations to describe the intracranial compartment based on the observation that with experimental increases in the volume of the system, the pressure rose in a semilogarithmic fashion (fig. 2). Alternatively stated, the graph of the logarithm of pressure versus the linear plot of volume change led to a straight line. When these investigators analyzed the curve of pressure versus volume, the instantaneous slope of the curve at any point was $\Delta P/\Delta V$, or cerebral elastance. In the case of this particular curve, dividing this equation by the pressure at that point was the same at all points of the curve and therefore defined the curve. $(\Delta P/P)/\Delta V$ was called $E_I$, the elastance coefficient, and just as the slope defines a straight line, this term defines the pressure-volume curve. The authors emphasized the importance of the pulse wave amplitude as a reflection of intracranial compliance [1, 2].

Under physiological conditions, $E_I$ is affected little by Kb since Kb is only related to the stiffness or turgor of the brain. As discussed above, the lower portions of the pressure-volume curve are the province of the freely displaceable venous blood and CSF. Theoretically, Kb could have two effects on calculations related to $E_I$. If the cause of the increase in Kb were due to an increased concentration of cerebral water, either in the form of blood, which is resistant to leaving the cerebral parenchyma, or in the form of increased white matter water as in cytotoxic or vasogenic edema, the effect would be to increase cerebral elastance markedly. In the face of a high Kb from this cause at the onset of the measurement, the effect would be to increase $E_I$, leading to a shift to the left of the pressure-volume curve. Increase in Kb by infusion of artificial CSF into the cerebral white matter would simply increase elastance without affecting $E_I$ (i.e., change the position on the curve).

If the cause of the increased Kb is an increased stiffness or hardness of brain parenchymal elements and not a change in brain water concentration, the early portions of the pressure-volume curve would be unchanged. But at the point that the brain parenchyma becomes involved, there would be a sudden increase in the slope of the curve and the mathematics on which the $E_I$ calculation are based would be negated.

Because of the totally theoretical nature of Kb as our knowledge of this parameter now stands, it is difficult, if not impossible, to predict the effect that the rate of change of ICP volume relations would have on Kb and vice versa. For a variety of logical reasons, Spertell [17] modeled the brain as a form of viscoelastic substance, termed a Maxwell solid. The mathematics of the properties of this particular class of substances postulate that a rapidly

applied distorting force will be resisted more effectively and with a higher concomitant change in pressure than a force that is applied more slowly. Since $E_I$ is primarily a description of the effect of rapid transient increases in intracranial dynamics, the effect of rate dependence on these interdependent parameters can probably be presumed to be negligible.

The mathematical description of the intracranial compartment proposed by Marmarou et al. [6] interrelates *both* rapid transient changes in ICP and more chronic changes that would be reflected by steady-state infusions into the CSF compartment. The concept of PVI revolves around the total volume of buffering capacity of the central nervous system (CNS) compartment. A small PVI indicates that the volume-buffering capacity of the total CNS system is small [6, 15, 16]. This can reflect a variety of factors, including total volume of the compartment when emptied of contents (adults are larger than children), free flow of fluid contents from one compartment to another as in compartmentalization of the CSF contents, and displacement of buffering fluids by growing mass lesions or edema. An increase in Kb decreases the ability of brain to diminish in volume when a distorting force is applied. Consequently, an increase in Kb will be reflected in a decrease in PVI.

## A Clinical Example of the Utility of Kb

In the original description of Kb, the concept was found useful to explain, at least theoretically, such enigmatic conditions as normal pressure hydrocephalus and pseudotumor cerebri [8–10, 12]. Subsequently, the concept has proved helpful in understanding why some patients continue to require CSF diversion despite total removal of brain tumors that led to the hydrocephalus in the first place [11]. The infrequent finding of symptomatic shunt malfunction without concomitant ventricular dilatation can be explained by analogy with pseudotumor cerebri of a high Kb and nonacute increase in resistance to outflow of CSF [8, 13].

Using the logic implicit in the above description of Kb, enigmatic conditions related to abnormal presentations of hydrocephalus, shunt malfunctions, or response to shunting may be explained and occasionally innovative methods of treatment may be suggested. The following clinical example will emphasize this point.

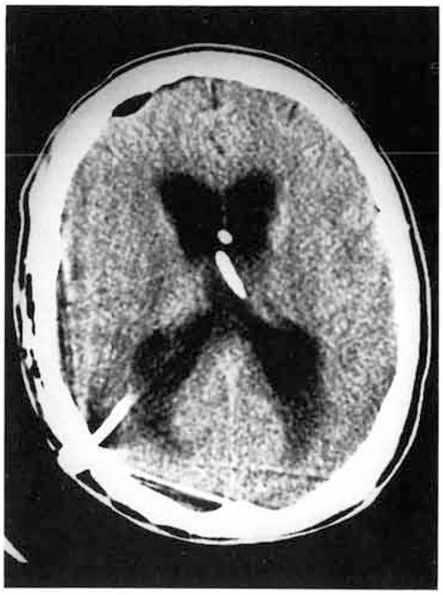

**Fig. 3.** CT scan of patient described in text showing marked ventricular enlargement despite low measured ICP.

## Case Report

A 24-year-old Caucasian female was referred for treatment of symptomatic 'slit ventricle syndrome'. At the age of 7 years, she had undergone the total removal of a cerebellar hemangioblastoma. After a stormy postoperative course, she underwent a ventriculoperitoneal shunt and during the next 14 years underwent 24 shunt revisions, primarily for proximal obstruction but also for low pressure syndromes and valve failures. At the initial attempt at shunt revision with valve change, the ventricular catheter was dislodged from the burr hole reservoir valve mechanism, leading to an avulsion of the choroid plexus and an intraventricular hemorrhage. This incident led to multiple valve failures and a period of external ventricular drainage (EVD). During the period of EVD management, it was noted that maintaining the drainage bag at head level resulted in marked ventriculomegaly and a somnolent patient. Improvement occurred only after maintaining the drainage bag below the level of the head.

Eventually, the CSF cleared. Attempts to use valve systems that impeded flow of CSF were not tolerated. Shunt systems that permitted siphoning led to severe headaches from slit ventricle syndrome. Finally, a medium pressure adult Hakim® valve (Cordis Corp., Miami, Fla.) was placed in series with a siphon control device (P.S. Medical, Santa Barbara, Calif.). The patient developed marked ventriculomegaly and impaired level of consciousness (fig. 3). At this point a shunt tap revealed an opening pressure of 8 cm/$H_2O$ in the recumbent position that dropped to negative ICP (sucking of CSF) in the erect position. It was thought that the shunt was working optimally and that the problem lay not in the shunt but in the lack of turgor within the

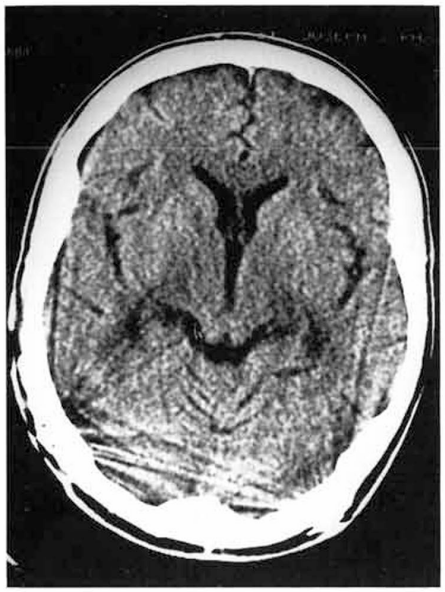

**Fig. 4.** CT scan of the same patient 24 h later after neck wrapping showing normal sized ventricles in an asymptomatic patient.

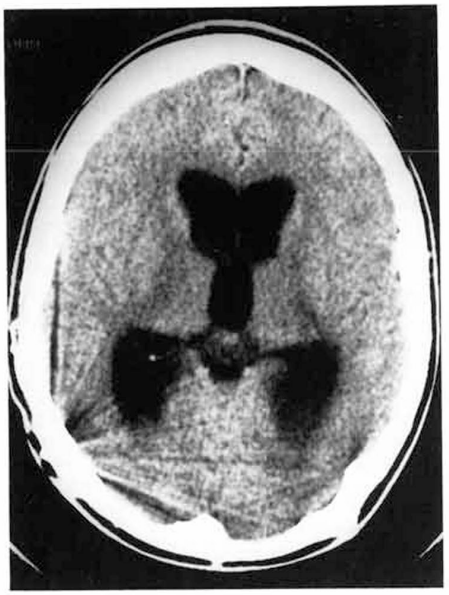

**Fig. 5.** After the removal of the neck wrap, the ventricles again became dramatically larger.

brain itself. Why this should occur is unknown. One way to increase Kb is to increase the water or blood content of the brain by impeding venous drainage from the intracranial compartment. After informed consent was obtained, the patient's neck was wrapped with an ace bandage as a venous tourniquet tight enough to impede venous return but loose enough to be reasonably comfortable. The following morning after careful observation in the intensive care unit, she was bright, awake, and without headache for the first time since her initial assessment. A computerized tomography (CT) study showed that the ventricles were normal in size (fig. 4).

After 72 h of continuous neck wrapping, the ace bandage was removed. Within 24 h, the ventricles had again increased dramatically and the patient was again somnolent (fig. 5). Shunt tap revealed a negative opening pressure but free flow of CSF from the ventricle and rapid run down of a loaded manometer indicating that the shunt was functioning. The siphon control device was removed. The ventricles again became slit-like and the patient's severe low pressure headaches returned. She remains in this condition 8 months later.

## Discussion

Neck wrapping impedes venous return and increases cerebral blood volume. In this manner, brain turgor or Kb is increased. Increasing brain stiffness or turgor allows an increase in brain volume at the expense of the ventricular system. A working shunt is in place that prevents siphoning; therefore, the increase in brain volume displaces CSF leading to a decrease in the size of the ventricles. ICP will not rise until sufficient CSF has been displaced to allow recording of brain parenchymal pressure. The effect of neck wrapping in this instance decreased ventricular volume only as long as the wrap was in place. The return of the abnormally low Kb led to ventricular distention because the sucking effect of siphoning was precluded by the siphon control device.

An alternative explanation to this discussion has been proposed [Sanford, pers. commun., 1991]. The neck wrapping may somehow lead to compression of the shunt tubing distal to the point that the siphon control device was inactivated (higher downstream pressure). It is true that removing the siphon control device led to ventricular decompression and a return to her slit ventricle syndrome. However, the inactivation of the siphon control feature by raising downstream pressure would still not allow the extreme low pressure states that would be present in the absence of a siphon control device. Change in Kb remains the most likely explanation for the observed phenomena.

## Conclusion

Brain turgor or Kb is a term that is part of brain elastance or PVI, but it represents only that portion contributed by the brain. Changes in Kb occur in many settings, not all of which have been defined. Kb is increased when venous drainage is impaired and in cases of vasogenic and cytotoxic edema. It is decreased from cranial irradiation and probably also from the normal process of senescence. This parameter is important to consider when analyzing the pathophysiology of abnormalities of CSF physiology and when deciding among treatment options.

## References


1 Avezaat CJJ, Van Eindhoven JHM, Wyper DJ: Effects of hypercapnia and arterial hypotension and hypertension on cerebrospinal fluid pulse pressure and intracranial volume-pressure relationships. J Neurol Neurosurg Psychiatry 1980;43:222–234.

2 Avezaat CJJ, Van Eindhoven JHM, Wyper DJ: Cerebrospinal fluid pulse pressure and intracranial volume-pressure relationships. J Neurol Neurosurg Psychiatry 1979;42:687–700.

3 Cutler RWP, Page L, Galicich J, Watters GV: Formation and absorption of cerebrospinal fluid in man. Brain 1968;91:707–720.

4 Galford JE, McElhaney JH: A viscoelastic study of scalp, brain and dura. J Biomech 1970;3:211–221.

5 Klatzo I: Pathophysiologic effects of brain edema. Acta Neuropathol (Berl) 1987;72:236.

6 Marmarou A, Shulman K, Rosende RM: A nonlinear analysis of the cerebrospinal fluid system and intracranial pressure dynamics. J Neurosurg 1978;48:332–344.

7 Mori K: Hyrodcephalus – revision of its definition and classification with special reference to 'intractable infantile hydrocephalus'. Childs Nerv Syst 1990;6:198–204.

8 Olivero WC, Rekate HL, Chizeck HJ, Ko W, McCormick JM: Relationship between intracranial and sagittal sinus pressure in normal and hydrocephalic dogs. Pediatr Neurosci 1988;14:196–201.

9 Rekate HL, Brodkey JA, Chizeck HJ, El Sakka W, Ko WH: Ventricular volume regulation: A mathematical model and computer simulation. Pediatr Neurosci 1988;14:77–84.

10 Rekate HL, McCormick J, Ko W: Failure to demonstrate a brain transmissibility factor. Concepts Pediatr Neurosurg. Basel, Karger, 1990, vol 10, pp 235–242.

11 Rekate HL, McCormick J, Yamada K: An analysis of the need for shunting after brain tumor surgery. Concepts Pediatr Neurosurg. Basel, Karger, 1990, vol 11, pp 39–46.

12 Rekate HL, Williams F, Chizeck HJ, Elsakka W, Ko W: The application of mathematical modeling to hydrocephalus research. Concepts Pediatr Neurosurg. Basel, Karger, 1988, vol 8, pp 1–14.

13 Rekate HL, Williams FC Jr, Brodkey JA, McCormick JM, Chizeck HJ, Ko W: Resistance of the foramen of Monro. Pediatr Neurosci 1988;14:85–89.

14 Reulen HJ, Suyumie T, Toch A, et al: Clearance of edema fluid into cerebrospinal fluid. J Neurosurg 1978;48:754.

15 Shapiro K, Fried A, Takei F, Kohn I: Effect of the skull and dura on neural axis pressure-volume relationships and CSF hydrodynamics. J Neurosurg 1985;63:76–81.

16 Shapiro K, Marmarou A, Shulman K: Characterization of clinical CSF dynamics and neural axis compliance using the pressure-volume index. I. The normal pressure-volume index. Ann Neurol 1980;7:508–514.

17 Spertell RB: The response of brain to transient elevations in intraventricular pressure. J Neurol Sci 1980;48:343–352.

18 Rekate HL: Increased intracranial pressure; in Blumer JL (ed): A Practical Guide to Pediatric Intensive Care, ed 3. St. Louis, Mosby Year Book, 1990, pp 234–246.