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Editorial Views

Airway Pressure—Neurosurgical Aspects

THE PIONEER NEUROSURGEONS had to learn the hard way about the effects of sequential increases in airway, intrathoracic, central venous and intracranial pressures (ICP) when operating on struggling patients using local anesthesia or primitive general anesthesia. Swelling and protrusion of the brain occurred in the craniotomy opening, making the surgical work difficult or impossible.[1] Modern anesthesiologic techniques have, of course, virtually eliminated these intraoperative hazards. The relationship between airway pressure and ICP remains, however, a major concern in the management of patients who have intracranial disorders, especially in connection with mechanical ventilation. In this issue, Aidinis, Lafferty and Shapiro present an experimental analysis of the intracranial effects of positive end-expiratory pressure (PEEP) ventilation in cats with supratentorial mass lesions. Their results are of both theoretical interest and practical importance. In brief, they show that the application of PEEP may result in elevations of central venous pressure and ICP and a decrease in arterial pressure, with consequent reduction of cerebral perfusion pressure. In this situation, signs of neurologic impairment could appear, with changes in pupillary dimensions and EEG abnormalities.

Actually, isolated short-term increases in intrathoracic and systemic venous pressures may be of little consequence to intracranial conditions so long as the system is closed. The central venous pressure is the terminal pressure in both cerebral vascular and cerebrospinal fluid (CSF) circuits. The spinal dural sac, the only distensible part of the system, is surrounded by the vertebral venous plexus. For these reasons changes in the central venous pressure result in additive changes in the CSF pressure, *i.e.*, the venous pressure may be regarded as a reference level for the CSF pressure.[2] In this situation the system remains balanced, the *effective* CSF pressure is not altered by a change in venous pressure, and provided cerebral perfusion pressure is not significantly reduced, there is no accompanying change in the relations between intracranial volume components or any subsequent volume displacements. The increase in the CSF pressure will be slightly less than that in the central veins, as there is a gradient of pressure along the vascular channels and an elevation of the venous pressure diminishes this gradient. With an increase of the intracranial venous pressure alone, however, as in compression of the neck veins, the system is unbalanced and localized volume expansion takes place, with resultant dislocation of CSF to the spinal compartment. Also, when the system is opened by craniotomy to ambient atmospheric pressure, changes in systemic venous pressure will produce changes in volume of the brain. The thoracic cage may be compared to a pair of bellows as the respiratory excursions force blood to and from the intracranial vascular bed, with resultant fluctuations of the brain in the operative field.[3] The lack of any effect in

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the closed system on volumetric relations is further evident from the fact that the craniospinal pressure–volume relationships are unaffected by an increase in the central venous pressure apart from a displacement of the CSF pressure–volume curve in parallel along the pressure axis in proportion to the increase in venous pressure.[4] The elastance of the system, *i.e.*, the slope dP/dV of the pressure–volume curve, is not appreciably affected. The term "elastance," which denotes a measure of the stiffness of the system, seems preferable to the inverse term, "compliance," as pressure usually is treated as the dependent variable in analyses of intracranial mechanics. In respiratory physiology "compliance" is a more suitable term, as the primary concern here is changes in volume. Thus, while an increase in systemic venous pressure may have little physiologic significance in these respects, it will act in the direction of reducing the cerebral perfusion pressure. In PEEP ventilation, however, the influence of the change in venous pressure on cerebral perfusion pressure seems of minor importance compared with the influence of the arterial hypotension that may develop concomitantly.

The key to understanding the behavior of the ICP is the distinction between steady and transient states. According to present concepts, the CSF compartment constitutes a simple flow system where the fluid pressure (P_{csf}) under steady conditions is determined by the flow (F), the resistance to the outflow of the fluid (R_o) and the pressure in the recipient system (P_{rec}), according to the formula:

$$P_{csf} = F \cdot R_o + P_{rec}$$

which is an application of the law of Poiseuille (Ohm) to the outflow of the fluid through the arachnoid villi. In a system of this type, volumes, *e.g.*, of expanding lesions or the vascular bed, do not directly affect the pressure in a steady state, as is often erroneously assumed. A volume change results only in a transient and reversible change in the pressure insofar that it does not modify the factors in the steady-state equation. The magnitude of the pressure response to a volume disturbance is determined by the value of the elastance at the prevailing pressure level. The pressure is returned to the initial value by a compensatory inflow or outflow of fluid, as the case may be, under the influence of the displaced pressure. Thus, a volume addition results in an exponential decay of the pressure at a rate that is a function of the time constant of the system, *i.e.*, the ratio between the outflow resistance and elastance (R_o/E). The process of spatial compensation takes place within a few minutes, but may be abnormally prolonged in expanding lesions or subarachnoid hemorrhage due to an increase in the outflow resistance, which is also probably the main cause of the elevated steady-state pressure under these conditions. The result is a persistent tendency to sustained high pressures in response to volume additions.

A reduction of the cerebral perfusion pressure, such as may be induced by an exposure to PEEP, results in decreases in the flow and pressure in the vascular bed. As a consequence, an autoregulatory vasodilator response is elicited, which tends to re-establish the cerebral blood flow. This response is associated with an increase in the cerebral blood volume, which in turn causes a transient pressure change as in any other type of volume increment. The change in pressure may be insignificant under normal conditions but considerable in the presence of a large space-occupying lesion due to an increased elastance. This increase in the elastance is caused by the elevated ICP, transferring the system to the steep part of the pressure–volume curve. It may be further augmented by a transtentorial or foraminal herniation that shuts off the volume-buffering action of the spinal compartment.[5] The subsequent return of the pressure to a steady-state value through the process of spatial compensation may be slower than normal, because of an increased time constant under these circumstances.

These basic mechanical events resulting in a pressure peak followed by a gradual decline are discernible in most of the pressure curves shown in the paper by Aidinis *et al*. At the highest PEEP, 15 cm H_2O, the ICP sometimes follows a more erratic course, which presumably indicates unstable hemodynamic conditions in this situation. When ICP is elevated it becomes increasingly sensitive to variations in the arterial pressure. This effect is not primarily related to associated changes in the vas-

cular volume, as is sometimes inferred. The intracranial fluid pressure and the subarachnoid venous pressure are approximately equal, *i.e.*, the ICP may be thought of as an expression of the subarachnoid venous pressure. According to hydrodynamic laws, in a rigid system a fractional change in the arterial pressure results in an appropriate change in flow and the same fractional change in the venous pressure. Thus, when the subarachnoid venous pressure and ICP are high, a given change in the arterial pressure has a greater effect on these pressures than when they are low. Of course, a passive distention of the vascular bed may also occur as the result of increased intravascular pressure, especially when restraints are removed by craniotomy. Autoregulatory responses that occur with a delay of some seconds counteract these changes in venous and CSF pressures and eventually restore the initial conditions.

The changes in ICP during PEEP ventilation, as measured relative to atmospheric pressure, are consequently the net effect of 1) increased systemic venous pressure which increases ICP, 2) reduced arterial pressure, which diminishes ICP, and 3) vasodilation, which increases ICP because of both the augmented blood volume and the increase in the venous pressure. The authors suggest that the ICP changes would have been greater if the arterial pressure had stayed higher. However, the vasodilator component of the ICP change is obviously dependent upon a significant reduction in the arterial pressure. It is clear, on the other hand, that the actual changes in ICP during PEEP ventilation, being the sum of opposing effects, may not reflect accurately the reactions taking place intracranially. This fact could explain the clinical experiences of neurologic complications occurring during PEEP treatments in spite of unchanged ICP.

The increase in ICP consequent to vasodilation is thus associated with and actually dependent upon a relative increase in cerebral blood flow. Evidently the decrease in the flow resistance caused by dilation of the resistance vessels is greater than the concomitant increase in the flow resistance due to compression of the venous outflow tract, produced by the volume addition. However, under certain extreme mechanical conditions, vasodilation theoretically may be accompanied by venous compression and increase in ICP to the extent that the outcome is a paradoxical decrease in the blood flow. A more frequent clinical problem is the possibility that vasodilation and increased ICP induced by PEEP ventilation produce a critical reduction in the blood flow in regions of the brain where marginal conditions for tissue perfusion exist. This may be the case in focal expanding lesions of a traumatic or ischemic nature, where a state of vasodilation may pre-exist with a reduced capacity for further compensatory dilation when the general fluid pressure is increased. The risks in this regard would be enhanced by a state of high ICP, high elastance, and sluggish spatial compensation. Obviously, arterial hypotension during PEEP treatment may also have adverse effects in nonexpansive cerebral disorders characterized by acidosis, vasoparalysis and edema.

Discontinuation of PEEP resulted in a rapid further gain in ICP related to the recovery and transitory overshoot of the arterial pressure. This pressure effect is due to the surge in cerebral blood flow that occurs when blood is forced under increasing pressure through the dilated vascular bed, augmenting the venous pressure. The arterial pressure declines and the resistance vessels constrict rapidly, however, reducing the high ICP. This event has the character of a transitory reactive hyperemia. The peak in ICP may presumably accentuate the mechanical stresses in the system related to expanding lesions and shifts of the brain.

In some respects, the effects of PEEP are similar to those of a Valsalva procedure.[2] During the period of thoracic strain, there is an increase in systemic venous pressure, often without a marked reduction in arterial pressure. The ensuing reductions in cerebral blood flow and blood volume may even be so severe that ICP drops below the systemic venous pressure. On resumption of normal breathing there is a brief period of overcorrection of the arterial pressure associated with a transient rise in ICP. The ICP response is amplified by a high initial pressure. Situations involving a Valsalva maneuver are notoriously dangerous in the presence of an intracranial mass lesion and transtentorial or foraminal herniation. As in the case of PEEP ventilation, there are

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theoretically two moments of danger, *i.e.*, the period of arterial hypotension and cerebral vasodilation, and the period of excessive ICP on release of the elevated pressure in the airways.

Mechanical ventilation with PEEP in cats with supratentorial space-occupying lesions could produce neurologic dysfunction consisting of ipsilateral dilation of the pupil and flattening of the EEG, *i.e.*, effects that are at least partially referable to a transtentorial herniation. Presumably a state of significant herniation existed in the experimental situation and the added effects of the hemodynamic disturbances were sufficient to precipitate a manifest herniation syndrome. But what is the precise mechanism of this result? The authors point to the two principal alternatives, *i.e.*, ischemia due to the reduced perfusion pressure in mechanically compromised regions of the brain, and/or an actual increase of the transtentorial displacement secondary to the stress of an increased brain vascular volume. There are some correlative evidences in favor of the latter explanation. In a similar experimental model with a supratentorial mass lesion the vasodilator action of halothane administration induced dilation of the pupil concomitantly with the development of a pressure differential between the supratentorial and infratentorial compartments.[6] In another study dilation of the pupil was produced by increasing arterial P_{CO_2} in spite of an increased cerebral blood flow, apparently discrediting the hypothesis of an ischemic cause.[7] The relative importances of generalized pressure and discrete herniations in producing cerebral dysfunction in compression of the brain remain, however, an unsettled problem in neurosurgical pathophysiology.

PEEP therapy may have detrimental side effects in the presence of intracranial mass lesions, and should be applied with close observation of hemodynamic and neurologic functions. The main indication for PEEP ventilation in neurosurgical patients is severe traumatic injury of the brain with associated respiratory insufficiency, *e.g.*, in neurogenic pulmonary edema or concomitant thoracic trauma.[8] Accessible expanding lesions should preferably be surgically removed prior to the institution of PEEP ventilation. Surgical decompression may not be feasible in cases of multiple hemorrhagic contusions from severe deceleration injuries, and PEEP treatment should probably be used with special caution in such cases. The adverse effects seem largely proportional to the level of PEEP. However, the transmission of the applied pressure is dependent upon the unknown factors of airway resistance and pulmonary tissue resistance and compliance, and the hemodynamic consequences are therefore somewhat unpredictable.

The investigation of Aidinis and co-workers clearly demonstrates the extreme sensitivity of the intracranial system under the conditions of expanding lesions to changes in hemodynamic modalities such as those induced by the application of PEEP, and presumably also other ventilation patterns that significantly elevate mean airway and intrathoracic pressures.

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