The effect of increased intracranial pressure on cerebrovascular hemodynamics

HARRY M. LOWELL, M.D., AND BYRON M. BLOOR, M.D.
Division of Neurosurgery, West Virginia University Medical Center, Morgantown, West Virginia

Both brain edema (increased water content) and enlargement of the vascular compartment have been implicated as being responsible for intracranial hypertension following trauma. In this study pertinent cerebrovascular hemodynamic parameters have been investigated in states of increased intracranial pressure (ICP) and graded trauma to determine whether cerebral edema or vascular factors are of major importance. Utilizing the monkey-epidural balloon experimental model, continuous measurements of the mean arterial pressure (MABP), jugular outflow pressure (MJVP), and sagittal sinus wedge pressure (SSWP) were obtained. Shulman's observations that the sagittal sinus wedge pressure accurately reflects the intracranial pressure have been confirmed. The total cerebral blood flow (CBF) and mean transit time (t) were determined and the total cerebral blood volume (CBV) computed. From these data the venous (Rv), arterial (Ra), and total resistances (Rt) were calculated. Analysis of these parameters during both the acute elevation of ICP and that following graded trauma has demonstrated: 1) a progressive decrease in the total cerebral blood flow and volume and a concomitant increase in the mean transit time; 2) a progressive increase in the total resistance with a shift from the arterial to the venous side; 3) a progressive decrease in the perfusion pressure (PP = MABP-SSWP); 4) impairment of CO2 reactivity pari passu with vasomotor activity and autoregulation of flow to pressure. The findings did not support the concept that increased intracranial pressure following trauma is the result of an increase in the size of the cerebrovascular compartment.

KEY WORDS cerebrovascular hemodynamics · trauma · edema · intracranial pressure · cerebrovascular resistance · cerebral blood flow · CO2 reactivity · perfusion pressure

To understand the pathophysiology of increased intracranial pressure (ICP) the Monro-Kellie doctrine has been a useful, albeit not an entirely accurate, concept. Monro22 (1783), on the basis of anatomical studies, concluded that because the intracranial contents are fixed in a bone encasement, the volume of blood within the brain must be constant at all times. He demonstrated this conclusion by exhibiting a glass water-filled ball from which no fluid escaped when inverted and concluded that there was little justification for jugular blood-letting in the treatment of intracranial disease. Later, Kellie13 (1824) suggested that a portion of the circulatory fluid could be withdrawn from within the cranium if its place was simultaneously occupied by some equivalent. Thus, the concept was expanded to include possible alterations in the brain blood volume. It is now considered that the intracranial contents consist of brain sub-
Vascular effects of increased intracranial pressure

stance, blood, and water including the cerebrospinal fluid (CSF), and reciprocal changes among these components may occur. As the cerebral blood volume and water content are the principal variable components, significant changes in either of these should lead to changes in intracranial pressure.

Both brain edema (increased water content) and enlargement of the vascular compartment have been named as major factors per se in the etiology of intracranial hypertension following trauma. Bakay and Lee in their recent review considered brain swelling and edema as separate processes, but they did not necessarily relate swelling to an increase in the size of the vascular compartment. On the other hand, since the early work of Wolff and Forbes demonstrating dilatation of pial vessels with increased intracranial pressure, Pilcher, Obrador and Pi-Sueir, Langfitt, et al., Ishii, and others have emphasized the role of vascular factors in producing brain swelling. They have postulated that brain injury causes vasomotor instability, vasoparalysis, and ultimately vasodilatation, all of which increase the size of the vascular compartment. Therefore, they suggest that intracranial blood volume is of greater importance than cerebral edema in producing acute brain swelling following trauma.

The intent of this study has been to measure total cerebral blood flow, total brain blood volume, and other pertinent cerebrovascular hemodynamic parameters not heretofore investigated in states of increased intracranial pressure and graded injury. A systematic analysis of these parameters should allow one to determine in individual cases whether cerebral edema or vascular factors are of major importance in the etiology of intracranial hypertension.

**Methods**

Using an epidural balloon, we studied the effect of incremental elevations in intracranial pressure on cerebrovascular hemodynamics in eight Rhesus monkeys. Each animal was lightly anesthetized with sodium thiopentol (Pentothal) and paralyzed with succinylcholine chloride (Anectine). Animals were artificially respirated with known CO₂ and O₂ concentrations. The end tidal CO₂ was monitored with an infrared CO₂ analyzer (Goddart Capnograph) and the PaCO₂ was maintained at 38 (± 5) mm Hg. Catheters were appropriately placed to obtain continuous measurement of the mean arterial (MABP), jugular outflow (MJVP), and sagittal sinus wedge (SSWP) pressures. The calvarium was sealed with dental acrylic (Hygon blue) to insure a "closed box" effect. In the initial experiments the intracranial pressure (ICP) was monitored by either an epidural balloon or small transducer and a cisternal catheter. In the former, values either inordinately high or relatively unresponsive to changes in ICP were frequently obtained. This inaccuracy of measurement was felt to be due to balloon tension and the inelasticity and anatomical partitioning by the interposed dura. The cisternal measurement was vitiated at higher pressures by tonsillar herniation. The most consistent and reproducible measurement was the sagittal sinus wedge pressure which, according to the work of Shulman, is increased proportionately with the cisternal spinal fluid pressure (slope = 0.68). Since his data included no CSF pressures exceeding 16.8 mm Hg (227 mm H₂O), 122 observations were made in four animals to confirm these findings and extend the range of observations. By manipulating the ICP with intracisternal saline infusion through a range of 2.7 mm Hg (37 mm H₂O) to 134 mm Hg (1822 mm H₂O), a

**List of Abbreviations Used in this Report**

| CBF     | cerebral blood flow |
| CBV     | cerebral blood volume |
| CSF     | cerebrospinal fluid |
| ICP     | increased intracranial pressure |
| MABP    | mean arterial pressure |
| MJVP    | mean jugular outflow pressure |
| PD<sub>1</sub> | post deflation measurement after 30 min stabilization period |
| PD<sub>2</sub> | second post deflation measurement after 30 min stabilization period |
| PD<sub>3</sub> | third post deflation measurement after 30 min stabilization period |
| PP      | perfusion pressure |
| R<sub>a</sub> | arterial resistance |
| R<sub>v</sub> | venous resistance |
| SSWP    | sagittal sinus wedge pressure |

J. Neurosurg. / Volume 34 / June, 1971 761
linear relationship was observed between the SSWP and ICP. A typical study may be seen in Fig. 1. Therefore, the SSWP accurately reflected changes in the intracranial pressure and was used to monitor the latter throughout these experiments. On the basis of a mean slope of 0.79 and an intercept of 10 mm Hg, the ICP was calculated from the SSWP measurements. This is in complete agreement with Shulman's observations in dogs and validates this relationship over the entire range of pressures encountered in these experiments.

Approximately 125 cerebral blood flow determinations were made on each animal by the dye-dilution technique. Following ligature of the external carotid artery, a known amount of dye was injected into the ipsilateral common carotid via a nonobstructive needle, and concentration curves were obtained by withdrawal of jugular blood through a densitometer at a constant rate of 3.9 ml/min and reinfused. The dynamic response in the total cerebral blood flow (CBF) and the mean transit time (t) were measured (Zeirler). From these data the total cerebral blood volume (CBV) was computed (CBV = CBF \times t). The cerebrovascular resistances were calculated by the following formulas:

\[ R_t = \frac{MABP - MJVP}{CBF} \]  

(1) Total resistance

\[ R_v = \frac{SSWP - MJVP}{CBF} \]  

(2) Venous resistance

\[ R_a = R_t - R_v \]  

(3) Arterial resistance (\( R_a = R_t - R_v \)).

It has previously been reported that in man the log of the cerebrovascular resistance (\( R_t \)) is a straight line function of the PaCO₂ and the slope of this line is proportional to the cerebrovascular reactivity. Unpublished data indicate that a similar reactivity slope is also true in the monkey and is calculated by the formula,

\[ \frac{\Delta \log R_t}{\Delta CO_2} \]

Reactivity at two levels of PaCO₂ and autoregulation of blood flow to spontaneous variations in blood pressure were determined. Stepwise elevations in intracranial pressure were produced by inflation of the epidural balloon in 0.25 or 0.50 ml increments at 3-min time intervals until decompression occurred; the point at which the EEG became isoelectric and/or pupillary changes were noted (average balloon volume, 3.5 ml). At this time the balloon was deflated and the animal allowed to stabilize for 30 min. Deflation data were obtained within 3 min of decompression and post-deflation after a 30-min period of stabilization. The latter were designated as PD₁, PD₂, and PD₃ respectively, depending on the number of previous compressions. This procedure was repeated until data could no longer be obtained. Necropsy was performed to confirm the correct placement of the sagittal sinus wedge catheter and that no intracranial bleeding had occurred. The data of all animals were averaged and the differences statistically tested. All parameters were plotted against increasing intracranial pressure, and third degree polynomial regression curves computed.

Results

Control

The mean values of 23 control flows and resistances plotted against the perfusion pressure (MABP - SSWP) over a range of 50 to 100 mm Hg indicated excellent autoregula-
Vascular effects of increased intracranial pressure

tion of cerebral blood flow to perfusion pressure changes with some over-compensation frequently seen in this pressure range (unpublished data). As the perfusion pressure increased, regulation of flow was accomplished by an increased arterial resistance (PP - Ra slope = .025). Since the venous component in the absence of increased intracranial pressure is negligible, the total resistance was only slightly higher and followed the arterial change (Fig. 2).

Concomitant observations of arterial resistance changes to 10 mm Hg alterations of PaCO₂ indicated normal CO₂ reactivity with a slope of -.04.

**Dynamics of Increasing Intracranial Pressure**

**Effect on Mean Arterial Pressure (MABP), Mean Jugular Venous Pressure (MJVP), Sagittal Sinus Wedge Pressure (SSWP), Perfusion Pressure (PP)**

As the intracranial pressure was elevated from 15 mm Hg (204 mm H₂O) to 160 mm Hg (2176 mm H₂O), the MABP increased from 82 mm Hg to a maximum of 154 mm Hg. Concurrently, the SSWP (subarachnoid venous pressure) mirrored the rising intracranial pressure; however, essentially no change in the jugular outflow pressure was noted. Therefore, in the face of increasing intracranial pressure, the diminishing perfusion gradient across the cerebrovascular bed could only be represented by the difference between the MABP and SSWP (Fig. 3).

**Effect on Cerebral Blood Flow (CBF) and Perfusion Pressure**

The effect of increasing ICP on CBF and PP (MABP - SSWP) is presented in Fig. 4. As the intracranial pressure was elevated from 15 to 40 mm Hg (204 to 544 mm H₂O), there was nearly a proportional rise in both the MABP and SSWP. As the result, there was little change in perfusion pressure and there was no significant decrease in the cerebral blood flow. Thereafter, the perfu-
tion pressure progressively decreased, and a passive flow-pressure relationship was observed. Decompensation occurred at a mean ICP of 152 mm Hg (2065 mm H₂O), with a range of 110 to 175 mm Hg. The concurrent PP and CBF were 17 mm Hg and 27 ml/min respectively. Beyond this point the CBF fell precipitously, and extrapolation indicated a zero flow at a residual perfusion pressure of 10 mm Hg (not shown).

Effect on Cerebrovascular Resistance

Between the intracranial pressure range from 200 to 2176 mm H₂O, the total resistance (Rₜ) rose progressively from 1.057 to 6.56 mm Hg/ml/min. The concurrent venous resistance change was from .05 to 5.1 mm Hg/ml/min. There was no significant change in the arterial resistance (Rₐ) until the ICP reached 40 mm Hg (544 mm H₂O) at which point the Rₐ began to decrease from 1.048 to .598 mm Hg/ml/min pari passu with the perfusion pressure. This decrease in Rₐ, while sufficient to offset the dramatic increase in Rₜ, indicated an active vasomotor system. The sudden increase in the Rₜ observed when the intracranial pressure was increased (ICP = 110 mm Hg, 1496 mm H₂O; PP = 27 mm Hg) probably indicates the approach of the critical closing pressure (Fig. 5).

Effect on Cerebral Blood Volume and Mean Transit Time

With increasing ICP, a decremental change in the cerebral blood volume from 11.4 to 5.4 ml was observed. Concurrent with this reduction in CBV was an increase in the mean transit time from 9.4 to 14.6 sec (Fig. 6).

Deflation Data

To compare the immediate effects of decompression, a comparison was made between average values of the three observations immediately before and following balloon deflation. The MABP and SSWP fell 29% and 72% respectively, however, the perfusion pressure (MABP—SSWP) increased 118%. A precipitous drop in total resistance occurred with decompression. The major change, as indicated by the decrease in the Rₜ/Rₐ ratio from 84% to 18%, was on the venous side. The arterial resistance decreased initially and rapidly returned toward normal. Accompanying these hemodynamic changes, there was an overshoot in the CBF of 127% with a concomitant decrease in the mean transit time of 50% and a minimal increase in the CBV of 12% (Table 1).

Post-Deflation Data

Post-deflation data (PD₁, PD₂, and PD₃) have been listed in Table 2. The effect of these repetitive insults was cumulative, producing a progressive elevation of the SSWP (ICP), and differing only quantitatively from the changes seen with acute elevations in ICP. Again, a progressive decrease in both
Vascular effects of increased intracranial pressure

TABLE 1

<table>
<thead>
<tr>
<th>Parameter</th>
<th>1 rain</th>
<th>2 rain</th>
<th>3 min</th>
<th>3 rain</th>
</tr>
</thead>
<tbody>
<tr>
<td>MABP</td>
<td>116</td>
<td>84</td>
<td>79</td>
<td>81</td>
</tr>
<tr>
<td>SSWP</td>
<td>99</td>
<td>28</td>
<td>27</td>
<td>29</td>
</tr>
<tr>
<td>PP</td>
<td>17</td>
<td>56</td>
<td>52</td>
<td>53</td>
</tr>
<tr>
<td>( R_t )</td>
<td>4.011</td>
<td>0.929</td>
<td>0.996</td>
<td>1.149</td>
</tr>
<tr>
<td>( R_v )</td>
<td>3.375</td>
<td>0.158</td>
<td>0.164</td>
<td>0.226</td>
</tr>
<tr>
<td>( R_a )</td>
<td>0.869</td>
<td>0.741</td>
<td>0.786</td>
<td>0.961</td>
</tr>
<tr>
<td>( R_v/R_t )</td>
<td>84%</td>
<td>17%</td>
<td>16%</td>
<td>20%</td>
</tr>
<tr>
<td>CBF</td>
<td>33</td>
<td>86</td>
<td>75</td>
<td>63</td>
</tr>
<tr>
<td>CBV</td>
<td>7.8</td>
<td>9.8</td>
<td>9.1</td>
<td>8.2</td>
</tr>
<tr>
<td>i</td>
<td>15.3</td>
<td>7.9</td>
<td>7.4</td>
<td>7.9</td>
</tr>
</tbody>
</table>

* The second column (\( \bar{x} \)) is the average of the three predeflation values. Columns to the right are recorded at 1-min intervals after decompression. In the last column a significance between mean values of < .01 is denoted by (S).

the perfusion pressure and blood flow occurred and was accompanied by an increase in the total resistance with a shift toward the venous side. The most striking of the hemodynamic changes observed was a sequential decrease in the total cerebral blood volume as the result of each successive cycle from 11.4 ml to 8.5, 6.5, and 5.4 ml, respectively. Concomitantly, there was a progressive decrease in the epidural balloon volume necessary to produce decompression. The mean values of post-deflation flows and resistances plotted against a perfusion pressure range similar to that in the control indicated impaired autoregulation by a passive pressure-flow relationship. Vasomotor activity was diminished as evidenced by a reduced PP--\( R_a \) slope of .005 (control PP--\( R_a \) slope = .025) (Fig. 7). Reactivity to \( CO_2 \) was lost with loss of autoregulation (\( CO_2 \) reactivity slope = -.0140, 34% of control).

**Discussion**

It should be noted that the sagittal sinus wedge pressure of 22 mm Hg and the mean transit time of 9.4 sec, obtained when the epidural balloon was not inflated, are higher than the mean of 18 mm Hg and 7.1 sec respectively in 1392 observations from monkeys in which epidural transducers had not been implanted (unpublished data). This difference can only be attributed to the fact that the implants were behaving as space-occupying lesions. In the same unpublished series a control CBF value of 82 ml/min/100 gm brain weight (wt) was obtained. This compares favorably with a CBF of 75 ml/min/100 gm brain wt observed in these experiments. It is recognized that these values are somewhat higher than those reported by others using different techniques. We are not aware of other published data utilizing dye-dilution and assume the discrepancy is best

TABLE 2

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Control</th>
<th>PD1</th>
<th>PD2</th>
<th>PD3</th>
</tr>
</thead>
<tbody>
<tr>
<td>SSWP</td>
<td>22</td>
<td>46(8)</td>
<td>49(8)</td>
<td>50(8)</td>
</tr>
<tr>
<td>PP</td>
<td>60</td>
<td>53(8)</td>
<td>51(8)</td>
<td>41(8)</td>
</tr>
<tr>
<td>( R_t )</td>
<td>1.057</td>
<td>1.404(8)</td>
<td>1.826(8)</td>
<td>2.046(8)</td>
</tr>
<tr>
<td>( R_v/R_t )</td>
<td>5%</td>
<td>42%</td>
<td>44%</td>
<td>48%</td>
</tr>
<tr>
<td>CBF</td>
<td>60</td>
<td>56</td>
<td>51(8)</td>
<td>37(8)</td>
</tr>
<tr>
<td>CBV</td>
<td>11.4</td>
<td>8.5(8)</td>
<td>6.5(8)</td>
<td>5.4(8)</td>
</tr>
<tr>
<td>Bal. vol. in ml at decompensation</td>
<td>3.5</td>
<td>2.7</td>
<td>1.9</td>
<td>1.0</td>
</tr>
<tr>
<td>( CO_2 ) reactivity slope</td>
<td>-.0407</td>
<td>-.0140</td>
<td>-.0140</td>
<td>-.0140</td>
</tr>
</tbody>
</table>

* (S) denotes a significant difference from the control of < .01.

*J. Neurosurg. / Volume 34 / June, 1971* 765
POST DEFLATION

Fig. 7. The mean values of post-deflation flows and resistances are plotted against the perfusion pressure. Impaired autoregulation and vasomotor activity is indicated by the passive pressure-flow relationship and the diminished PP - Ra slope of .005.

explained by a systematic error in the different methodologies.

Both brain edema (increased water content of the brain) and an increase in the vascular compartment have been implicated as the etiology of increased intracranial pressure following brain injury. To explain the latter of these mechanisms, it has been postulated that brain injury causes cerebrovascular instability, paralysis, and ultimately vasodilatation; thereby elevating intracranial pressure by increasing the size of the vascular compartment. If this postulate were correct, one could reasonably assume that cerebral blood volume and/or flow would be increased.

In direct opposition to this was our finding that the flow and volume were decreased in both acute intracranial hypertension and in the post-injury state. Data have been presented that demonstrate, as the result of sequential insults, a progressively smaller epidural balloon volume necessary to produce decompensation. This intolerance to balloon volume in the face of a diminished CBV must have been secondary to an increase in the nonvascular brain volume (edema). Assuming the intracranial volume was constant and equal to the sum of the brain, blood, CSF, and balloon volume, then by simple addition one can approximate this theoretical increase in the nonvascular compartment. Initially the cerebrovascular compartment represented 14% of the total content, and the addition of 4% volume by the epidural balloon produced decompensation. With repeated insults the vascular compartment progressively decreased to 7% of the total intracranial content, and the addition of only 1% volume by the balloon produced decompensation. As the vascular and balloon content were diminished by 10%, there must have been an equivalent increase in the nonvascular brain volume (Fig. 8). This estimate was most likely conservative as alterations in the CSF volume, not taken into account, can occur rather rapidly.

Neither the acute elevation of ICP produced by the epidural balloon nor the more chronic elevation of ICP associated with impaired vasomotor activity, produced by repeated cerebral insults, was accompanied by an increase in the cerebrovascular compartment. This was attested to by the fact that both the cerebral blood flow and volume, as measured by this technique, were significantly decreased, and the calculated nonvascular volume was increased. We may, therefore, conclude that a repeated cerebral compression injury that results in increased intracranial pressure and vasomotor paresis is

![CONTROL PD1 PD2 PD3](image)

Fig. 8. Note, as the result of successive insults, the progressive decrease in both the cerebrovascular compartment and the balloon volume necessary to produce decompensation. Total intracranial volume was derived from a x brain wt of 80 gm.
Vascular effects of increased intracranial pressure

not secondary to an increase in the size of the cerebrovascular compartment but, rather, is the result of an increase in the volume of the nonvascular compartment (edema).

The transitory hyperperfusion (overshoot) following the rapid decrease in elevated ICP is not precisely understood. The two most plausible explanations are: 1) extracellular acidosis secondary to cerebral ischemia as espoused by Zwetnow, et al., who described increased flows lasting more than 1 hour following a reduction in perfusion pressure to 40 mm Hg; and 2) the loss of autoregulation to pressure change (Bayliss effect). In our experiments an even lower perfusion pressure was reached for a comparable time; however, only a very evanescent overshoot was observed. Thus, acidosis as an explanation seems unlikely. As seen in Table 1, the CBF within 1 min following decompression was 86 ml/min (43% above the control) and returned to 63.4 ml/min (5% above the control) by 3 min. Neither was there any evidence for increased flow 30 rain post-deflation at which time the CBF averaged 7% below the control.

The arterial resistance was 26% below that in the control immediately following decompression and by 3 min was approximately normal. From these data it would appear that the sudden hemodynamic response is best explained by the instantaneous increase in the perfusion pressure and the reduced arterial resistance followed by partial recovery of the Bayliss effect. Consonant with this, Rapela and Green have reported a similar overshoot that subsided within approximately 90 sec following the release of temporary carotid occlusion.

Green, et al., have stated that there is little change in flow until the CSF pressure approaches the arterial pressure. On the other hand, deterioration of homeostasis in humans was indicated by the findings of Ferris who reported diminution in cerebral blood flow in two subjects when the intracranial pressure was raised above 25.7 mm Hg (350 mm H_2O). Likewise, Kety, et al., and Greenfield and Tindall have found reductions in the cerebral blood flow at intracranial pressures of 33 mm Hg (450 mm H_2O) and 28 mm Hg (380 mm H_2O) respectively.

Shulman and Verdier have reported the effects of cisternal CSF pressures up to 29 mm Hg in the dog. They observed that no change in the R_t or CBF occurred in spite of an increasing venous resistance. Our data have indicated little evidence for or against autoregulation during intracranial pressure ranges up to 40 mm Hg (500 mm H_2O). During the initial phase of rising ICP, homeostasis of flow was not accomplished by a significant decrease in the arterial resistance but, rather, the perfusion pressure was maintained by an elevation in the systemic MABP which was nearly equal to the rise in the SSWP. As the intracranial pressure was progressively increased above 40 mm Hg (500 mm H_2O), the perfusion pressure began to fall and the arterial resistance decreased, indicating arteriolar vasomotor activity in an attempt to maintain cerebral circulation. However, unlike the control situation, the venous resistance was no longer a negligible component of the total resistance and became the overriding factor. The sudden increase in the R_t observed when the ICP was elevated (ICP = 110 mm Hg, 1496 mm H_2O; PP = 27 mm Hg) probably indicates approach of the critical closing pressure and impending vascular collapse.

The observation of Shulman and Verdier of apparent autoregulation is not inconsistent with our data. The basic difference between the two investigations is the ICP range studied, i.e., 10 to 29 vs 15 to 160 mm Hg. From the data at hand it is probably fair to say that autoregulation, effected by an R_t decrease, is operant up to ICP levels of 30 to 40 mm Hg. Beyond this point, however, the increasing venous resistance cannot be compensated, the R_t inevitably increases, and the CBF becomes pressure dependent.

The observations of flow at levels of ICP exceeding the mean arterial blood pressure by about 6 mm Hg confirms the observation of Wright who noted that circulation did not cease until the ICP had almost reached the systolic arterial pressure.

We have presented data to demonstrate that the jugular outflow pressure does not reflect changes in intracranial pressure. On the other hand, consistent with the work of Shulman, the sagittal sinus wedge pressure (subarachnoid venous pressure) was linearly related to the intracranial pressure. There-
fore, in states of increased ICP, a valid perfusion gradient across the cerebrovascular bed is represented by the difference between the MABP and the SSWP. These data suggest the possible usefulness of continuous wedge pressure recording as an indication of an increasing ICP. Decreasing perfusion pressure would be of value in predicting impending decompensation.

Summary and Conclusions

In the monkey-epidural balloon experimental model, changes in the total cerebral blood flow, blood volume, and mean transit time were measured using the dye-dilution technique and the cerebrovascular resistances calculated. Shulman's observations indicating that the SSWP is a measure of the subarachnoid venous pressure which is in turn linearly related to the ICP have been confirmed and extended. These data have indicated that in the absence of intracranial hypertension, autoregulation was accomplished by pre-venous (arteriolar) resistance changes. This mechanism was ineffective in the presence of increased intracranial pressure because of the overriding increase in the venous resistance.

Observations made during acute elevations of ICP have demonstrated:

1. Failure of the MJVP to reflect changes in the ICP
2. Correlation between the perfusion pressure (MABP—SSWP) and changes in ICP and CBF
3. A progressive decrease in the CBF and CBV and a concomitant increase in the
4. A progressive increase in the total resistance with a shift from the arterial to the venous side
5. An apparent lack of "autoregulation" of flow despite retained vasomotor activity.

Following sequential cerebral insults, it was observed that:

1. The SSWP indicated a progressive increase in ICP.
2. A decrease in the vascular compartment was indicated by decreasing CBF and CBV values.
3. The calculated nonvascular brain volume (edema) increased from the first to the final insult.
4. Reactivity to PaCO₂ was essentially nil following the first insult.
5. Vasomotor activity and autoregulation was impaired.

Acknowledgments

The authors wish to express their gratitude and sincere appreciation to Mrs. J. Wenger (R.N.), Mrs. L. Orenstein (M.A.), Mr. R. Berry, Mr. F. Cole, Mr. P. Feorene, and Mr. D. Logue (B.S.) for their assistance.

References

Vascular effects of increased intracranial pressure

13. Kellie G: An account of the appearances observed in the dissection of two of three individuals presumed to have perished in the storm of the 3 D, and whose bodies were discovered in the vicinity of Leith on the morning of the 4th, November, 1821; with some reflections on the pathology of the brain. *Trans Med Chir Soc Edinburgh* 1:84-169, 1824


24. Pilcher C: Experimental cerebral trauma: the fluid content of the brain after trauma to the head. *Arch Surg* 35:512-527, 1937


Received for publication June 16, 1970.
This work was supported in part by USPHS Grants NB03343 and NB07733.

Address reprint requests to: Byron M. Bloor, M.D., Chairman, Division of Neurosurgery, Loyola University Medical Center, 2160 South 1st Street, Maywood, Illinois 60141.