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Clinical Article

Continuous cerebral compliance monitoring in severe head injury: its relationship with intracranial pressure and cerebral perfusion pressure

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Summary

Background. Cerebral compliance expresses the capability to buffer an intracranial volume increase while avoiding a rise in intracranial pressure (ICP). The autoregulatory response to Cerebral Perfusion Pressure (CPP) variation influences cerebral blood volume which is an important determinant of compliance. The direction of compliance change in relation to CPP variation is still under debate. The aim of the study was to investigate the relationship between CPP and compliance in traumatic brain injured (TBI) patients by a new method for continuous monitoring of intracranial compliance as used in neuro-intensive care (NICU).

Method. Three European NICU's standardised collection of CPP, compliance and ICP data to a joint database. Data were analyzed using an unpaired student t-test and a multi-level statistical model.

Results. For each variable 108,263 minutes of data were recorded from 21 TBI patients (19 patients GCS ≤ 8; 90% male; age 10–77 y). The average value for the following parameters were: ICP 15.1 ± 8.9 mmHg, mmHg, CPP 74.3 ± 14 mmHg and compliance 0.68 ± 0.3 ml/mmHg. ICP was $\geq$20 mmHg in 20% and CPP <60 mmHg for 10.7% of the time. Compliance was lower (0.51 ± 0.34 ml/mmHg) at ICP ≥ 20 than at ICP <20 mmHg (0.73 ± 0.37 ml/mmHg) ($p<0.0001$). Compliance was significantly lower at CPP <60 than at CPP ≥ 60 mmHg: 0.56 ± 0.36 and 0.70 ± 0.37 ml/mmHg respectively ($p<0.0001$). The CPP – compliance relationship was different when ICP was above 20 mmHg compared with below 20 mmHg. At ICP <20 mmHg compliance rose as CPP rose. At ICP ≥ 20 mmHg, the relation curve was convexly shaped. At low CPP, the compliance was between 0.20 and 0.30 ml/mmHg. As the CPP reach 80 mmHg average compliance was 0.55 ml/mmHg., but compliance fell to 0.40 ml/mmHg when CPP was 100 mmHg.

Conclusions. Low CPP levels are confirmed to be detrimental for intracranial compliance. Moreover, when ICP was pathological, indicating unstable intracranial equilibrium, a high CPP level was also associated with a low volume-buffering capacity.

Keywords: Traumatic brain injury; ICP; compliance; CPP; Spiegelberg.

Introduction

The ability of the intracranial compartment to compensate for added volume is an important factor in the development of raised intracranial pressure (ICP) particularly following severe traumatic brain injury (TBI) [16, 28, 29]. Intracranial compliance, or its inverse elastance, is considered an index of the volume buffering capability of the brain and reduced compliance eventually will lead to increased intracranial pressure (ICP) [13, 15]. The mechanisms involved in the reduction of compliance and increase in brain stiffness after TBI are not well understood. Cerebral vascular blood volume (CBV) is considered the most important determinant of intracranial compliance [14]. Variations in cerebral perfusion pressure (CPP = MAP – ICP) have a significant influence upon cerebral vascular resistances and on cerebral blood volume which regulate a constant cerebral blood flow [9]. The variation in CBV with CPP depends upon whether autoregulation is still functional or not [4]. Most studies so far have investigated the influence of systemic blood pressure (MAP) and cerebral perfusion pressure (CPP = MAP – ICP) on cerebral compliance

[5, 7, 8, 19, 21]. However these studies have shown contrasting results on both the extent and the direction of the CPP influence on compliance. There is also concern that the invasive nature of the methods used to measure compliance, may in themselves alter the normal autoregulatory steady state. Different methods have been developed to measure cerebral compliance in the clinical setting. The most well known are the Pressure Volume Index introduced by Marmarou (PVI) [11, 13, 15] and the Volume Pressure Response (VPR) of Miller [17, 18]. These methods are based upon the manual injection or withdrawal of known volumes of fluid into and from the CSF space of the patient while measuring the ICP before and after the volume change. However, these methods provide only in-frequent measures of compliance often with high variability between measurements. This is because it is difficult to manually inject consistent volumes of fluid rapidly at a constant rate of injection. Furthermore, with this method, the need for access to the CSF system to inject the volume can increase the risk of infection. As a consequence of these limitations the PVI or VPR tests are not routinely used in neurosurgical practice. Other methods, based upon waveform analysis of the ICP signal, have been proposed to measure compliance less invasively have not, as yet, been validated in clinical practice [1, 2, 27].

Recently, a new technology able to continuously monitor cerebral compliance (Spiegelberg Brain-Pressure monitor and compliance-monitor; GmbH & Co) [10] has been developed using an automated method to inject and withdraw small volumes (0.2 ml) based upon a pulse averaging method developed by Piper *et al.* [23]. Using this system we were able to monitor, continuously, intracranial compliance by a ventricular catheter used to measure ICP as part of the routine intensive care treatment in severely brain injured patients.

The aim of the study was to investigate the relationship between CPP and compliance by using a new method for continuous monitoring of intracranial compliance. This new methodological approach will, for the first time, make it feasible to study this relationship in detail during both therapeutically induced and inherent fluctuations in CPP and compliance.

Materials and methods

Data were collected from 3 European Neuro-Intensive Care Units (Dept. of Neurosurgery, University Hospital, Uppsala, Sweden; Dept. Neurosurgery, University of Heidelberg, Berlin, Germany, Intensive Care Dept., Ospedale San Gerardo, Monza, Italy). These centres are all members of the Brain IT Group (www.brainit.org) a multicentre internet based research group conducting a clinical assessment of a new ventricular catheter technology for continuously monitoring ICP and cerebral compliance in severe head injury patients (Spiegelberg Brain-Pressure monitor and compliance-monitor; GmbH & Co) [24]. Each member of the group contributed data to a joint database collected at the patient's bedside by use of a standardised data collection software tool. A standardised protocol was employed based on minute by minute averages of the simultaneously collected variables of ICP, compliance and CPP. Measurements were prospectively performed in twenty-one severely head injured patients from September 1998 to May 2000. The patients were selected based only on the absence of compressed ventricles to allow ventricular cannulation. Patients who needed bony decompression were excluded to avoid difficult interpretation of compliance values. Systemic arterial pressure was concomitantly monitored by an indwelling radial artery catheter with the pressure transducer zeroed at the level of the mid-ear. CPP was calculated as the difference between mean arterial pressure (MAP) and ICP. All patients were sedated, intubated and mechanically ventilated. Surgical and medical treatment, according to international protocols (Guidelines for the management of severe traumatic brain injury) [3], was applied with the intent to maintain $ICP < 20$ mmHg and $CPP \geq 60$ mmHg.

The Spiegelberg catheter is a double lumen ventricular catheter with an air pouch mounted on the tip. The Spiegelberg compliance monitor calculates intracranial compliance ($C = \Delta V/\Delta P$) from a moving average of small ICP disturbances (ΔP) resulting from a sequence of up to 200 pulses of added volume ($\Delta V = 0.1$ ml). Once a stable average has developed, the device produces a minute-by-minute measure of mean intracranial compliance [24].

ICP, ABP, compliance and derived CPP were continuously collected on a minute by minute basis at the bedside by a personal computer running the Edinburgh Browser Software [12] in 2 centres and CMA-ICU pilot in one centre. The data collected by each centre were centralized in a common database transferred via the Internet. All data were examined and obvious artefacts were removed based upon data exceeding known physiological threshold values or as indicated from text notes supplied with the data.

Statistical methods

Data were summarized as mean $\pm$ standard deviation (SD). The median is also reported for data with no normal distribution.

The data were analyzed using unpaired student t-test and a multi-level model, as described by Goldstein [9]. This is an appropriate modelling approach as there are multiple measurements across patients and across centres, so any model used must account for any random effects of patients and centres upon the dependent variable. In this approach, an overall model is described which is assumed to be true for all the patients in the study. The models are then arranged so that any multiple observations obtained from each patient are considered in the estimates of the p-values for each effect. This is equivalent to a random-effects model. The levels in the model were defined as the center in which the patient was treated and the patient within each center. The data were all transformed to have an overall mean of zero. The dependent variable was compliance. The predictor variables were cerebral perfusion pressure and CPP squared, whether the ICP was less than or equal to 20

or not, and the interaction of CPP and CPP squared with the ICP ≤ 20 or not variable. The model can be written in equation form as:

$$\begin{aligned}\text{Compliance} = {} & B1 * \text{CPP} + B2 * \text{CPP}^2 + B3 * \text{ICP} \leq 20 \\ & + B4 * \text{CPP by ICP} \leq 20 \text{ interaction} \\ & + B5 * \text{CPP}^2 \text{ by ICP} \leq 20 \text{ interaction} \\ & + B6 \text{ (intercept)}.\end{aligned}$$

The coefficients were estimated using the restricted iterated generalized least squares method.

Results

The three centres named A, B, C contributed to data collection with 8, 9 and 4 patients respectively, GCS on admission was ≤ 8 except in two patient with intracerebral hemorrhage (GCS = 10 and 9). Ninety percent were male and age ranged from 10–77 years. On discharge from ICU 12 patients were obeying, 6 were not obeying simple commands and 3 died from intractable intracranial hypertension. Demographic and injury details are summarised in Table 1.

Table 1. *Demographic characteristic of study population*

Patient	Age (years)	Sex	GCS at admission	Lesion (first CT)	Outcome at ICU discharge
A1	10	male	4	ICH	obeying
A2	51	male	3	ICH	obeying
A3	28	male	3	ICH	obeying
A4	56	male	6	ICH, SDH	not obeying
A5	77	male	3	ICH, SDH	not obeying
A6	32	male	3	EDH, ICH	obeying
A7	58	male	8	SDH, ICH	obeying
A8	62	male	3	EDH, ICH	not obeying
B1	76	male	10	ICH	obeying
B2	40	male	6	TSAH	obeying
B3	48	male	7	ICH, SDH, EDH	obeying
B4	69	male	7	ICH	not obeying
B5	63	male	7	SDH, ICH, EDH	obeying
B6	22	male	5	ICH	dead
B7	77	female	9	ICH	not obeying
B8	18	female	4	DAI	not obeying
B9	19	male	7	SDH	dead
C1	54	male	8	ICH, SDH	obeying
C2	56	male	7	ICH, SDH	dead
C3	64	male	8	EDH	obeying
C4	67	male	7	ICH, SDH	obeying

CT lesion: refers to the first CT scan; Outcome at the ICU discharge: is classified as obeying, not obeying and dead; *ICH* Intracerebral hemorrhage; *SDH* subdural hematoma; *EDH* Extradural hematoma; *tSAH* traumatic subarachnoid hemorrhage.

After removing obvious artefacts, 108, 263 minute by minute values were recorded for each variable (compliance, CPP and ICP) and available for analysis. These data corresponded to a total of 692 hours of valid data from Centre A: 73.2% of total monitoring time, percentage valid data ranged by patient between: 14.5%–90.5%). From centre B: 759 hours (80% total monitoring time, percentage valid data ranged by patient between: 60.5%–96.6%) and from centre C: 353 hours (36.2% total monitoring time, percentage valid data ranged by patient between: 18.3%–66.3%). The average of all recorded values were ICP 15.1 ± 8.9 mmHg, MAP 89.3 ± 12 mmHg, CPP 74.3 ± 14 mmHg and compliance 0.68 ± 0.3 ml/mmHg (median: 0.58 ml/mmHg). In spite of our efforts to avoid pathological secondary insults, there was an incidence of ICP ≥ 20 mmHg in 20% and CPP < 60 mmHg in 10.7% of the recorded time.

Low CPP was caused by a high ICP (>20 mmHg) in 53% of cases and a systemic secondary insult (low ABP) was responsible in the remaining 47% of cases.

When ICP was higher than 20 mmHg, the average compliance value was significantly lower (0.51 ± 0.34 mmHg) then values associated with an ICP < 20 mmHg (0.73 ± 0.37 mmHg) ($p < 0.0001$).

Similarly, compliance was significantly lower at CPP $<$ 60 mmHg than at CPP ≥ 60 mmHg: 0.56 ± 0.36 and 0.70 ± 0.37 ml/mmHg respectively ($p < 0.0001$) (Fig. 1).

The relationship between CPP and compliance was then analysed over a range of ICP below and above 20 mmHg considering this threshold as pathological

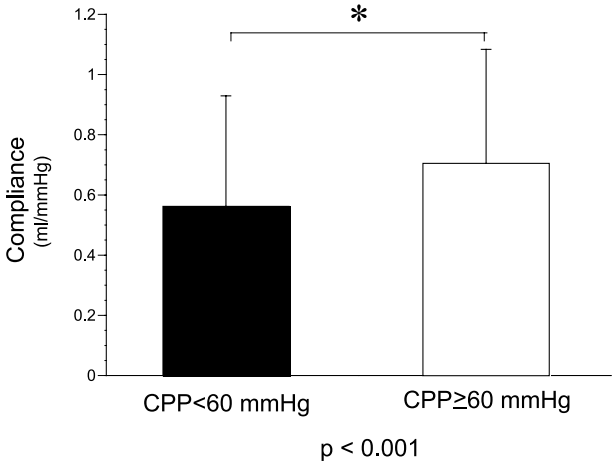

Fig. 1. Compliance (Mean ± Standard Deviation) at CPP < 60 mmHg and CPP ≥ 60 mmHg, $p < 0.001$

according to the Guidelines for the management of severe traumatic brain injury [3].

Results of the multilevel modelling

The model presented in the methods section was estimated using 108,263 data points and the full model equation shown below.

$$\begin{aligned}\text{Compliance} = {} & B1 * \text{CPP} + B2 * \text{CPP}^2 + B3 * \text{ICP} \leq 20 \\ & + B4 * \text{CPP by ICP} \leq 20 \text{ interaction} \\ & + B5 * \text{CPP}^2 \text{ by ICP} \leq 20 \text{ interaction} \\ & + B6 \text{ (intercept)}.\end{aligned}$$

The model results are best presented separately for ICP ≤ 20 and for ICP > 20 and the equations are:

$$\begin{aligned}\text{For ICP} \leq 20\text{: Compliance} = {} & 0.179089 * \text{CPP} \\ & + 0.011936 * \text{CPP}^2 + 0.083\end{aligned}$$

$$\begin{aligned}\text{For ICP} > 20\text{: Compliance} = {} & 0.110614 * \text{CPP} \\ & - 0.01592 * \text{CPP}^2 - 4.657\end{aligned}$$

The equations are very different with respect to the sign of the coefficient for the CPP squared coefficient. These equations are best illustrated with the two plots in Fig. 2.

With such a large sample size, all of the coefficients were significant at the $p < 0.001$ level. Any random variance due to centre and patient were accounted for in the multi-level model.

Discussion

Several authors have studied the correlation between CPP and the intracranial volume-pressure relationship. From these studies, autoregulation has been recognized as the key issue influencing this relationship [5, 7, 8, 19, 21]. Although it is still under debate as to which is the direction of compliance change in response to CPP variation. It is our belief that the opportunity to monitor compliance continuously, by this new device together with other cerebral hemodynamic parameters, could give novel insight into the pathophysiology of intracerebral volume balance related to CPP. Pilot experimental and clinical studies suggested a satisfactory agreement between the Spiegelberg device and the gold standard methods to measure ICP and compliance [6, 23, 31, 32]. In this study, it was possible to collect a satisfactory amount of valid data although the variation in percent of ''useful'' data between centres should be explained. One possible explanation is that, between centres, there is a different availability of researchers who can follow, day by day, the collection and correct or mark by text notes any possible artifact prior to data transfer. As a result of this finding, a new data validation procedure has been developed as part of the published BrainIT group operating procedures [25].

In this data set, the majority of ICP and CPP values were within a normal physiological range probably due to the start of treatment to avoid and eventually correct

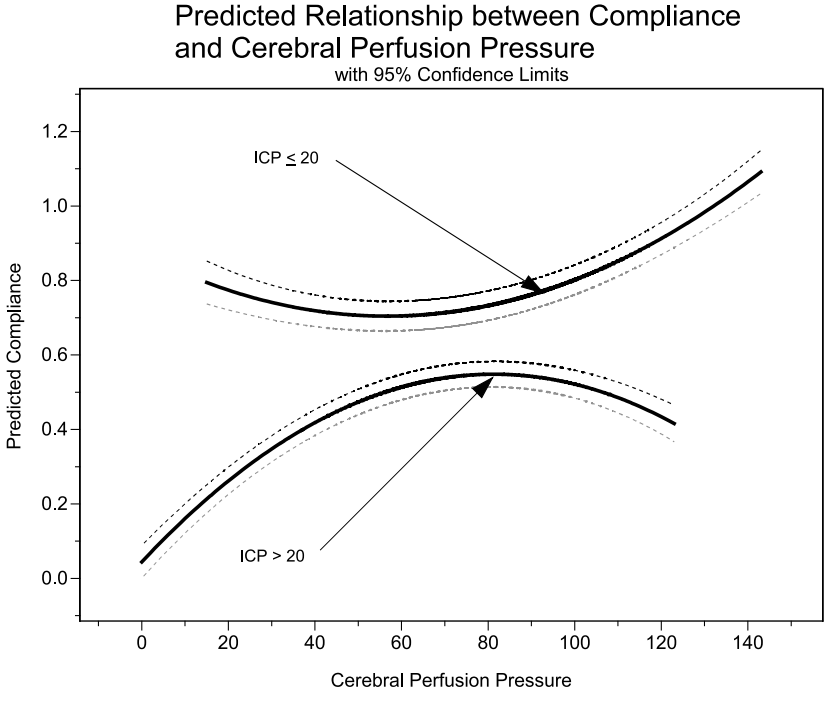

Fig. 2. Predicted relationship between compliance and cerebral perfusion pressure at different ICP levels

pathological events. Although intracranial and systemic secondary insults could not always be avoided with an ICP $\geq$ 20 mmHg and CPP $<$ 60 mmHg being documented in 20% and 10.7% of the recorded time respectively. Physiological values of cerebral compliance available from the literature are not measured but derived from normal PVI measurement using the calculation method of Marmarou and are reported to be in the range of 0.25–1.5 ml/mmHg [14]. However, to our knowledge, human studies to define a normal compliance range have still to be carried out. For this reason we can only study our measurements in the context of them being derived from a pathological population (TBI). In our data set cerebral compliance was significantly lower at CPP $<$ 60 mmHg which is believed to be below the lower autoregulatory limit where vessel dilatation is documented to occur [9]. Under these conditions, cerebral vascular engorgement occurs from impending vasoparalysis and causes an increase in CBV responsible for the lower intracranial volume-buffering capacity [4]. In this intensive care management study, we focused upon the relation between compliance and CPP when pathological ICP was unavoidable indicating a more unbalanced intracranial state possibly including impaired autoregulatory status. From this working hypothesis, we propose that we might discriminate different CPP/compliance behaviours depending upon whether ICP was higher or lower than 20 mmHg.

Relationship between CPP and compliance

There was a strong and statistically significant difference in the relationship between compliance and CPP depending upon whether the ICP was above or below 20 mmHg. When the ICP was below 20 mmHg, compliance dropped slightly as CPP rose from about 20–30, with a minimum estimated compliance of 0.70 ml/mmHg at a CPP of 60 mmHg. From then on, increasing CPP was associated with increasing compliance, so that at a CPP of 100 mmHg, the compliance was nearly 0.90 ml/mmHg (upper line in Fig. 2). However, when ICP was above 20 mmHg, the curve was convexly shaped and the overall compliance was lower compared with the ICP $<$ 20 relationship. At low CPP, the compliance was very low (0.20–0.30 ml/mmHg) and rose rapidly as the CPP rose to a maximum compliance of 0.55 ml/mmHg at a CPP of approximately 80 mmHg. Then as the CPP continued to rise, the compliance began to fall again to a value of approximately 0.40 ml/mmHg at CPP of 100 mmHg (lower line in Fig. 2). This relationship was not critically dependant on the choice of ICP threshold. When an analysis was conducted with an ICP threshold of 25 mmHg, the relationship we have reported between high and low ICP with compliance remained unchanged.

These results indicate that factors other then just the ICP level at which compliance was measured influences compliance. In this regard, it is likely that the status of autoregulation plays an important role. The positive association between CPP and compliance we found in the low ICP range, suggests preserved autoregulation consistent with an increased compensatory volume induced by CPP driven cerebral vasoconstriction. However, when ICP was pathological ($\geq$20 mmHg) compliance showed a tendency to decrease both at low CPP and at very high CPP levels. This is consistent with previous studies where it has been shown that at low CPP levels, autoregulation is often impaired with impending vasomotor paralysis [8]. Such an impaired autoregulatory status would influence CBV in a pressure passive manner and so determine intracranial volume imbalance and ICP rise [4]. Our data have also shown that lower compliance values were associated with very high CPP levels. Clinical studies on severe head injury patients revealed the same behaviour when autoregulation was defective [5, 19]. A limitation of our clinical study was our inability to continuously assess the status of autoregulation, however the complex relationship identified between CPP and compliance when ICP is high does support an impaired autoregulatory state. From our results it is possible that, at a pathological ICP, the positive association found between CPP and compliance at low CPP is due to CBV engorgement inducing raised ICP and thus lowering compliance. We plan further clinical studies in this area using measures of autoregulation to confirm this hypothesis.

Until now we have not considered blood pressure in this relationship, despite it being a well know determinant of craniospinal compliance. Our study design, was based upon previous literature in this area, which focused on the relationship between CPP and compliance. To focus on CPP is pertinent as CPP, as pointed out in Douglas Miller's classic monograph ''Concepts of Perfusion Pressure'' [20]'' is in effect the closest approximation we have to ''Transmural Pressure'' which will be the key to the myogenic drive underlying pressure autoregulation. Figure 3 shows the relationship between ICP, mean arterial pressure and compliance. From this it can be seen that in our data both MAP falls and ICP increases as CPP falls which shows the importance of not analysing just one factor but a combination.

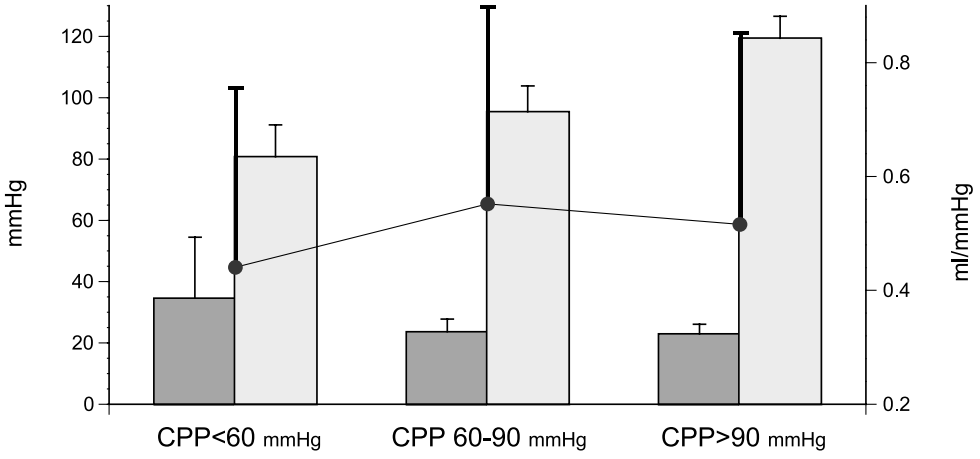

Fig. 3. Relationship between compliance ICP, MAP with falling compliance. —— Comp, ▓ ICP, □ MAP

We also took the decision to study only the relationship between compliance and not PVI with CPP. Compliance (dv/dp) is physically not the same as the PVI which is the inverse of the elastic coefficient E1 in the steady state equation $P = Poe^{E1V}$. Again it was Douglas Miller who pointed out that if there were just a single pressure-volume curve for each patient than measuring compliance would add no new predictive value over that of ICP. It is known though that the pressure volume curve can shift to the left or right and change it's slope. The value of compliance is that it informs one of where on the pressure volume curve the patient sits and PVI provides information on ''which curve'' the patient is currently on. The difficulty with calculating PVI from compliance using Marmarou's formula is that, as pointed out by Raabe [26] the calculated PVI is very dependant upon the opening pressure which is significantly affected by hydrostatic pressure gradients. In this multicentre study we could not control or measure the degree of head up tilt under which the patients were managed (causing variation in HPG), thus making it difficult to consider calculation of PVI in this study.

Conclusion

Our results corroborate that intravascular pressure and its effect on CBV will affect not only ICP but also the mechanical properties of the brain and volume buffering capacity of the intracranial space. Low CPP levels are confirmed to be detrimental for the intracranial volume balance. Moreover, when ICP was pathological, indicating unstable intracranial equilibrium, a high level of CPP also affected volume-buffering capacity. Under these conditions, when we suspect impaired autoregulation, the effort to ''over-correct'' CPP by increasing MAP does not always guarantee intracranial volume stability.

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Comments

The collection of ICU data from several ICU sites is a significant accomplishment. However, there are conceptual problems with the analysis, which clouds the issue of clearly understanding the importance of these findings regarding compliance, CPP and ICP. From the work of Douglas Miller and others cited by the authors it is well known that there is an exponential relationship between intracranial pressure and compliance. In a head injured patient as well as a normal volunteer, the compliance will decrease as ICP increases as a result of this exponential relationship. Therefore, when CPP is altered and since CPP contains the variable ICP by definition, it is difficult to determine exactly which phenomenon is being altered.

The PVI on the other hand does simplify this approach to some degree. If the PVI is altered as CPP is altered, then one can deduce that the shape of the exponential curve is becoming more or less steep. In summary, the fact that compliance is reduced or increased is dependent upon ICP, and since ICP is included in the parameter CPP, it is difficult for the reader to interpret the findings of this report. The authors must further expand on this issue in discussion and since PVI can be calculated from their data, it would seem reasonable to let the reader know if the curve becomes more or less steep as a result of the CPP change. In either case, PVI or compliance, the changes in blood pressure must be addressed.

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In this multicenter study, the authors collected a large volume of monitored data on ICP, CPP and brain compliance, aiming to determine the relationship between these parameters. The data were scrutinized for artifacts and entered into a centralized database through the internet. This paper proves that cooperation between centers at the level of data collection and analysis of complex and sophisticated clinical ICU monitoring parameters is indeed possible. The authors are to be commended for this accomplishment.

This article provides clinical data on the use of the Spiegelberg monitor which can serve as a reference for further studies. Nevertheless, I remain somewhat sceptical about the clinical usefulness of continuous cerebral compliance monitoring, which in my opinion is not proven by this study.

The data again confirm that the relationship between CPP and brain compliance is a very complex one which exphasizes the risks of pharmacological manipulations of blood pressure in head-injured patients without a thorough understanding of the underlying pathophysiology.

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