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Clinical Article

Intracranial hypertension: what additional information can be derived from ICP waveform after head injury?

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Summary

Objective. Although intracranial hypertension is one of the important prognostic factors after head injury, increased intracranial pressure (ICP) may also be observed in patients with favourable outcome. We have studied whether the value of ICP monitoring can be augmented by indices describing cerebrovascular pressure-reactivity and pressure-volume compensatory reserve derived from ICP and arterial blood pressure (ABP) waveforms.

Method. 96 patients with intracranial hypertension were studied retrospectively: 57 with fatal outcome and 39 with favourable outcome. ABP and ICP waveforms were recorded. Indices of cerebrovascular reactivity (PRx) and cerebrospinal compensatory reserve (RAP) were calculated as moving correlation coefficients between slow waves of ABP and ICP, and between slow waves of ICP pulse amplitude and mean ICP, respectively. The magnitude of 'slow waves' was derived using ICP low-pass spectral filtration.

Results. The most significant difference was found in the magnitude of slow waves that was persistently higher in patients with a favourable outcome ($p < 0.00004$). In patients who died ICP was significantly higher ($p < 0.0001$) and cerebrovascular pressure-reactivity (described by PRx) was compromised ($p < 0.024$). In the same patients, pressure-volume compensatory reserve showed a gradual deterioration over time with a sudden drop of RAP when ICP started to rise, suggesting an overlapping disruption of the vasomotor response.

Conclusion. Indices derived from ICP waveform analysis can be helpful for the interpretation of progressive intracranial hypertension in patients after brain trauma.

Keywords: Brain injury; intracranial hypertension; intracranial pressure; waveform analysis; outcome.

Introduction

The prognostic power of clinical variables such as age, admission GCS, pupillary abnormalities, results of computed tomography (CT) scans, episodes of hypoxia and hypotension have been extensively studied in order to define a reliable predictive model of outcome in patients with severe brain injury [3, 6, 7, 15, 24]. High intracranial pressure (ICP) and systemic hypotension are detrimental to outcome [4, 5, 29, 36]. Improved knowledge about the physiological mechanisms regulating cerebral homeostasis and the availability of new monitoring techniques have added new variables to management algorithms in neuro intensive care. However, ICP measurements still provide crucial information about the time-course of events in post-traumatic intensive care. Methods like spectral ICP waveform transmission [26], high frequency centroid of ICP pulse wave [8] or evaluation of brain compliance [39] has been introduced as 'promising' in the past but their usefulness still requires clear evidence. Cerebral autoregulation and cerebrovascular pressure-reactivity have been shown to correlate with outcome after severe brain injury [12, 14, 20, 26, 31]. The spectral components of the ICP waveform, such as pulse amplitude, slow waves (e.g. Lundberg's B waves), plateau waves, etc., have been commonly documented in the past [21, 23, 28, 30], but not always with convincing proof about their value in management of head injury.

We have studied retrospectively the patients with sustained intracranial hypertension to determine whether there is a pattern of selected indices derived from the ICP waveform analysis specific to the recordings acquired from those patients who died, compared to

those achieving favourable outcome. Dichotomy of outcome groups has been chosen intentionally to achieve the better sensitivity to pick up these differences in morphology of ICP waveform, which may be helpful in future prospective studies. We have also attempted to describe the time-profile of these variables in the different outcome groups.

Patients and methods

We studied retrospectively data from 96 head injuried patients. Patients were selected from the larger group of 418 head injured patients (age 32 ± 16 yrs; median GCS on admission 6, range 3 to 15), monitored using our computerised multi-modality-monitoring system between January 1992 and December 2001. Criteria for selection were determined by the main aim of this study: they all presented with a period of intracranial hypertension defined as mean ICP > 25 mmHg for at least 4 hours continuously. Only patients with favourable outcome (GOS 4–5; 39 patients) and patients who died (GOS 1; 57 patients) were considered in our analysis in order to determine whether and which components of ICP waveform may distinguish between these two terminally different outcomes under condition of sustained intracranial hypertension.

Patients were treated according to in-house guidelines for brain trauma [18]: focal cerebral contusions or brain oedema were confirmed on CT scan and any lesion causing a significant mass effect was surgically evacuated. ICP and arterial blood pressure (ABP) were monitored invasively. Systemic hypotension was controlled with fluids and infusion of norepinephrine or dopamine as appropriate in order to maintain a cerebral perfusion pressure (CPP) above 70 mmHg. Patients were mechanically ventilated to moderate hypocapnia ($PaCO_2 > 4.5$ kPa). Sedation and paralysis were the first line therapy in cases of intracranial hypertension. Mannitol, thiopentone and moderate hypothermia (34 °C) were administered in cases of severe intracranial hypertension. An extraventricular drainage was used if enlarged ventricles were present on the CT scan.

Monitoring

Monitoring included invasive ABP from the radial or dorsalis pedis artery. ICP was monitored either through an intraventricular drain hydraulically coupled to an external pressure transducer (Baxter), or using an intraparenchymal probe (Camino ICP transducer with V202 monitor or Codman ICP MicroSensors). CPP was calculated as the difference between mean ABP and mean ICP. The heart rate (HR)

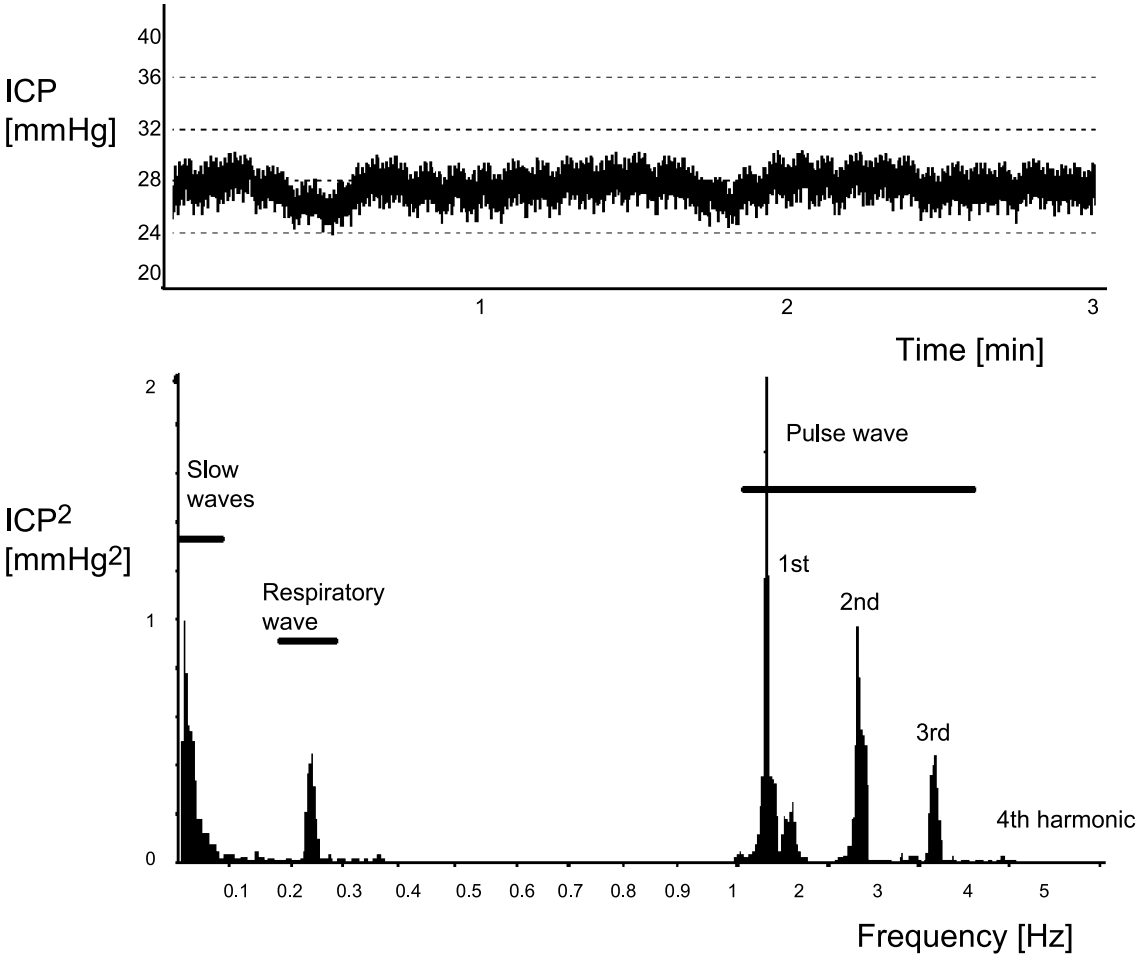


Fig. 1. Example of ICP recording showing pulse, respiratory and 'slow waves' overlapped in time-domain (upper panel) and separated in frequency domain (lower panel)

was derived from the ABP waveform via spectral analysis. Digitized waveforms (11 bits over the range from 0 to 500 mmHg for ABP and 0–120 mmHg for ICP) were continuously sampled with a frequency of 40 Hz (Data Translation DT2814, 12 bit analog-to-digital converter was used), and analysed using Fast Fourier Transformation over 6.4 s time-window. The code for this form of analysis was prepared in-house [9, 10]. For further analysis one-minute averages were calculated and stored on a portable computer.

Data analysis

Slow waves of ICP

The ICP waveform consists of three components, which overlap in the time domain, but can be separated in the frequency domain (see Fig. 1). The pulse waveform has several harmonic components, of these the fundamental component has a frequency equal to a heart rate. The amplitude of this component (AMP) was analysed using frequency analysis and stored for further processing. The respiratory waveform is related to the frequency of the respiratory cycle (8–20 cycles per minute).

'Slow waves' were not as precisely defined as in the original Lundberg study 'B waves' (1/2 to two per minute; [23]). All components having spectral representation within the frequency limits of 0.05 to 0.0055 Hz (20 sec to 3 minutes period) were classified as slow waves. The magnitude of these waves was calculated as the square root of the power of the signal of the pass band of the equivalent frequency range at the output of the digital filter.

Reduction of the noise to signal ratio has been achieved through selective filtration of the desired components (slow and fundamental of heart rate) using spectral analysis.

Pressure-volume compensatory reserve index (RAP)

The RAP index (correlation coefficient [R] between AMP amplitude [A] and mean pressure [P]) was derived by linear correlation between 40 consecutive, time-averaged data points of AMP and ICP acquired over 6.4 s. This index indicates the degree of correlation between AMP and mean ICP over short periods of time (~4 minutes). Its clinical significance has been discussed before [10]. Theoretically, the RAP coefficient indicates the relationship between ICP and changes in volume of the intracerebral space, known as the 'pressure-volume curve' [2, 21, 32]. An RAP coefficient close to 0 indicates lack of synchronisation between changes in AMP and mean ICP. This denotes a good pressure-volume compensatory reserve at low ICP (see Fig. 2). When RAP rises to +1, AMP varies directly with ICP and this indicates that the 'working point' of the intracranial space shifts to the right towards the steep part of the pressure-volume curve. Here compensatory reserve is low, therefore any further rise in volume may produce a rapid increase in ICP. Following head injury and subsequent brain swelling RAP is usually close to +1. With further increase in ICP, AMP decreases and RAP values fall below zero. This occurs when the cerebral autoregulatory capacity is exhausted and the pressure-volume curve bends to the right as the capacity of cerebral arterioles to dilate in response to a CPP decrement is exhausted, and they tend to passively collapse. This

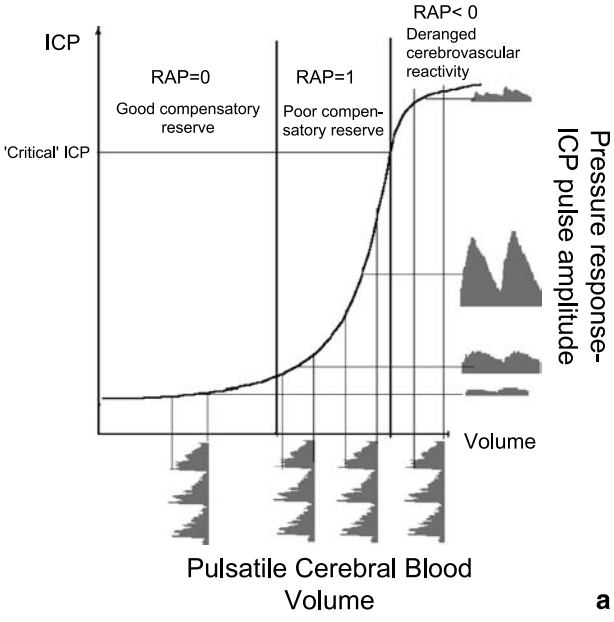
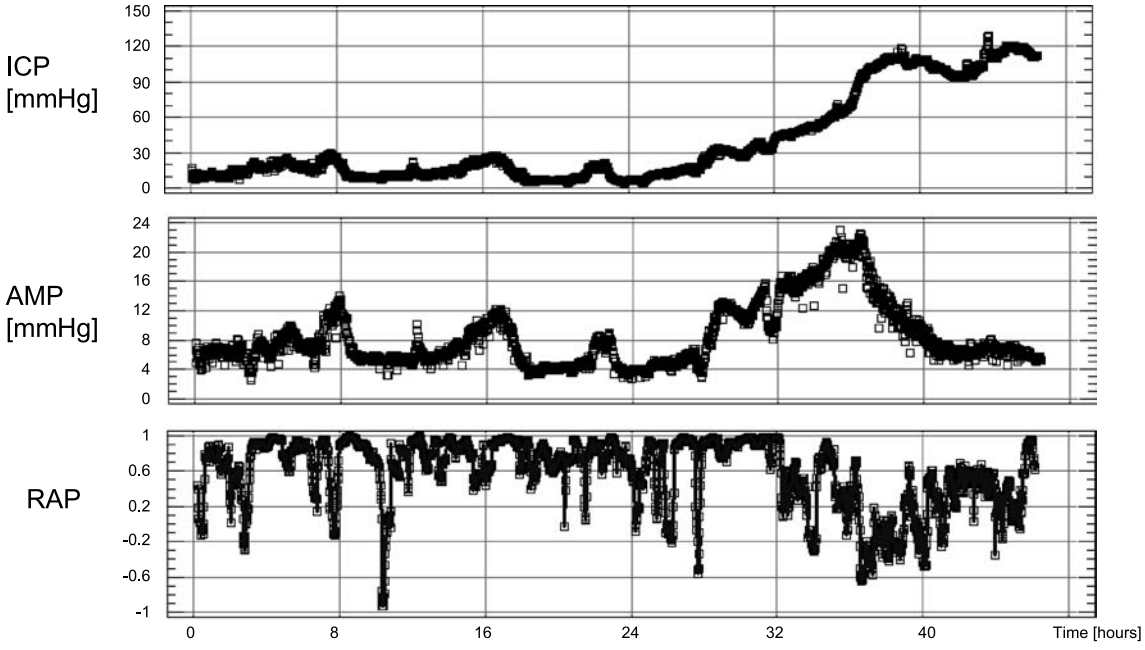


Fig. 2. (a) Example of the relationship between pulse wave amplitude (*AMP*) and mean intracranial pressure (*ICP*) recorded during a 46 hour period, during which terminal intracranial hypertension developed. Pulse amplitude increased first proportionally to the change in ICP but started to decrease when ICP increased above 80 mmHg. The regression plot between AMP and ICP (bottom panel) indicated a biphasic relationship of positive and negative slopes. The correlation coefficient between AMP and ICP (*RAP*) was positive before 32 hours but negative after that, indicating terminal cerebrovascular deterioration. (b) In a simple model, pulse amplitude of ICP (expressed along the y-axis on the right side of the panel) results from pulsatile changes in cerebral blood volume (expressed along the x-axis) transformed by the pressure-volume curve. This curve has three zones: a flat zone, expressing good compensatory reserve, an exponential zone, depicting poor compensatory reserve and a flat zone again, seen at very high ICP (above the 'critical' ICP) depicting derangement of normal cerebrovascular responses. The pulse amplitude of ICP is low and does not depend on mean ICP in the first zone, resulting in values of RAP close to 0. The pulse amplitude increases linearly with mean ICP in the zone of poor compensatory reserve, resulting in RAP close to +1. In the third zone, the pulse amplitude starts to decrease with rising ICP making RAP theoretically negative. Adapted from [2, 21]



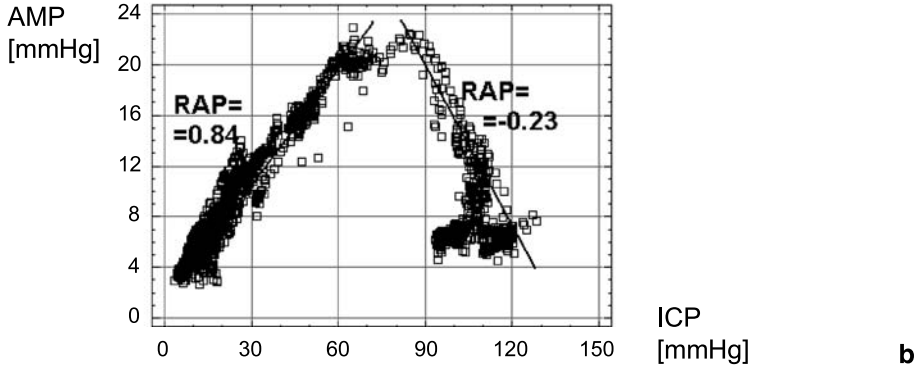


Fig. 2 (*continued*)

indicates terminal cerebrovascular derangement with a decrease in pulse pressure transmission from the arterial bed to the intracranial compartment.

Cerebrovascular pressure-reactivity index (PRx)

A second ICP-derived index is the pressure-reactivity index (PRx), which incorporates the philosophy of assessing cerebrovascular pressure-reactivity by observing the response of ICP to spontaneous changes in ABP [11]. Using computational methods similar to calculation of the RAP index, PRx was determined by calculating the correlation coefficient between 40 consecutive, time-averaged data points of ICP and ABP. A positive PRx signifies a positive gradient of the regression line between the slow components of ABP and ICP, which we hypothesize to be associated with a passive behaviour of a non-reactive vascular bed. A negative value of PRx reflects a normally reactive vascular bed, as ABP waves provoke inversely correlated waves in ICP. This index correlates well with indices of autoregulation based on transcranial Doppler ultrasonography [12, 19]. Furthermore, abnormal values of both PRx and RAP, respectively indicative of poor autoregulation or deranged cerebrospinal compensatory reserve, have been demonstrated to be predictive of a poor outcome following head injury [10, 11, 34].

All parameters, RAP, PRx and magnitude of slow waves were calculated prospectively.

Statistical analysis

Physiological parameters from every patient were summarized as mean values and compared between the two outcome groups. In order to describe the 'time-profile' of the variables, the recording time was standardized for every patient: time 0% was considered the beginning of the recording and time 100% was the end of it.

Values between the groups of 'dead' and 'favourable' outcome patients were compared using the Kruskall-Wallis test (non-parametric test). Analysis of variance was applied to reveal significance of standardized time trends (i.e. values versus percent of time of monitoring with 10% – long time bins). Kolmogorov-Smirnoff test (paired, non-parametric) was used to compare values of parameters at the beginning and end of monitoring time.

Results

Clinical profiles

Patients who died, had obviously worse admission Glasgow Coma Score. Distributions of nature of injury do not present remarkable differences between two groups. Patients who died were on average older than those who achieved good outcome.

Analysis of mean values of the monitored variables

All variables, with exception of CPP and ICP had normal distribution (ICP and CPP had log-normal distribution). The results of the analysis of each variable over the entire monitoring period in the two outcome groups are presented in Table 1. There were no differences in mean values of ABP and heart rate between the two groups, whereas a significantly greater ICP ($p < 0.0003$) was observed in patients who died. CPP was lower in the fatal outcome patients, but the difference was only just significant ($p < 0.02$). In patients who died PRx index was higher ($p < 0.024$) with a lower value of RAP ($p < 0.003$), probably representing exhaustion of the cerebrovascular pressure-reactivity overlapping with poor pressure-volume compensatory reserve. The most striking difference between the two groups was the magnitude of slow waves, which was much lower ($p < 0.000036$) in patients who died.

Using multivariate analysis independent determinants of outcome are mean ICP and PRx ($R_2 = 0.37$; $p < 0.0001$). Neither initial injury (GCS), slow waves nor RAP can be taken as independent predictors of outcome. However, the magnitude of slow waves cannot be regarded as simply dependant on intracranial hypertension or initial injury as both parameters did not correlate with it. On the other hand, RAP showed significant negative correlation with ICP ($R = -0.23$; $p < 0.006$) but only in patients who died.

The time-profile of the monitored variables

The averaged time-profiles of the measured variables were analysed (Fig. 3). No differences were found in ABP and HR time-trends whereas specific patterns were defined for ICP, PRx and RAP in the two groups.

Table 1. *Mean values and standard deviation of physiological variables and ICP-derived indices in the two outcome groups. For ICP and CPP medians, 25% and 75% percentiles were additionally provided*

	Favourable outcome	Fatal outcome	Level of significance
Age (range)	27 (17–61)	33 (16–87)	$p < 0.02$
GCS (median/range)	8 (5–15)	5 (3–13)	$p < 10^{-7}$
ABP (mmHg)	100 (±19)	102 (±15)	Not significant
ICP (mmHg) Median, 25%,75% percentiles	30 (±3.3) 21, 19, 24	40 (±16) 28, 23, 45	$p < 0.00029$
CPP (mmHg) Median, 25% and 75% percentiles	72 (±15.3) 75, 65, 81	61 (±8.4) 68, 54, 76	$p < 0.019$
HR (c/s)	88 (±17)	85 (±16)	Not significant
AMP (mmHg)	5.09 (±2.9)	6.95 (±4.21)	$p < 0.021$
AMP/ABPa	0.12 (±0.07)	0.16 (±0.09)	$p < 0.016$
PRx	0.09 (±0.21)	0.301 (±0.26)	$p < 0.024$
RAP	0.569 (±0.23)	0.41 (±0.25)	$p < 0.0028$
Slow (mmHg)	1.21 (±0.58)	0.65 (±0.45)	$p < 0.000036$
Slow/ICP	1% (±1.8%)	2% (±1.6%)	$p < 0.0001$

ABP Mean arterial pressure; *ICP* intracranial pressure; *CPP* cerebral perfusion pressure; *HR* heart rate; *AMP* fundamental harmonic component of pulse amplitude of ICP waveform; *ABPa* fundamental harmonic component of arterial pressure pulse waveform; *PRx* pressure-reactivity index; *RAP* index of pressure-volume compensatory reserve; *Slow* magnitude of slow waves in ICP; *Slow/ICP* ratio of slow waves of ICP to mean value of ICP.

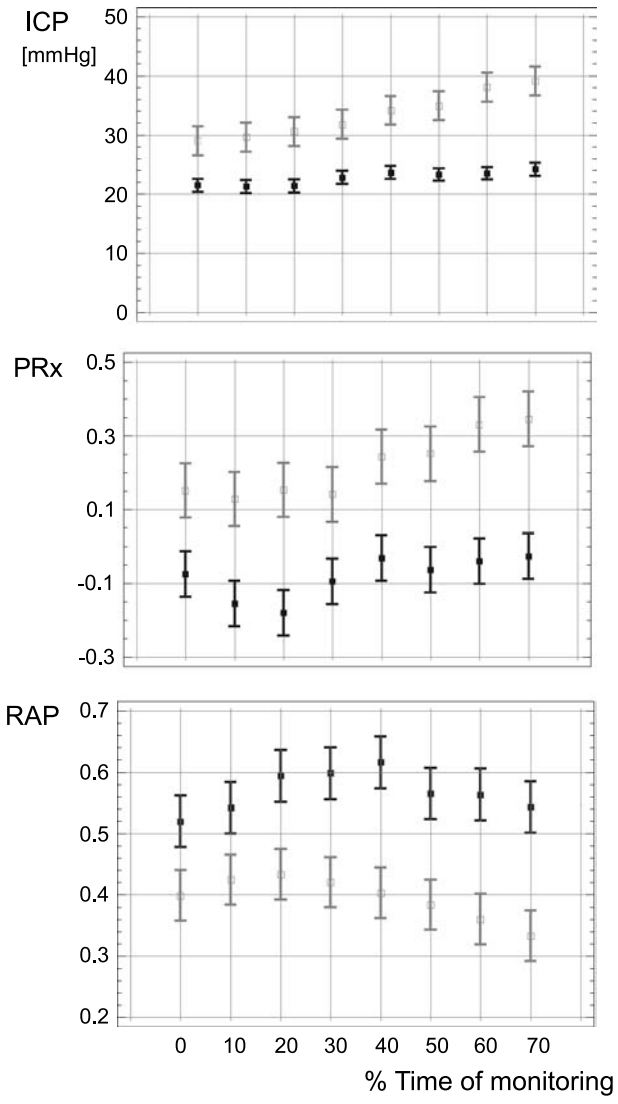


Fig. 3. Average values (squares: open-patients who died; closed-patients with favourable outcome) and 95% confidence limits for mean values of the monitored parameters versus of time of monitoring (Time was expressed in % of total time from zero to 70%; the last 30% was omitted as it was commonly disturbed by weaning from ventilator or contain data from monitoring during the period with formally unconfirmed brain death criteria). ICP is higher in patients who died (a) with an increasing time-trend. Autoregulation, which appears to be compromised from the beginning as confirmed by higher values of PRx in patients who died, deteriorated throughout the monitoring period when the ICP climbs (b). RAP was consistently lower in patients who died showing a typical pattern in response to intracranial hypertension (c)

Averaged ICP was stable and evenly distributed in time (between 20 and 25 mmHg) in patients with a favourable outcome (Fig. 3a). On the contrary, in patients who died, an increasing trend of ICP was noticed (Kruskall-Wallis: $p < 0.0001$). In patients with favourable outcome PRx was constantly below 0.1 confirming satisfactory cerebrovascular pressure-reactivity and preserved autoregulation (Fig. 3b). On the contrary, in patients who died, PRx was above this threshold (0.1) and mean values increased significantly from 0.139 ± 0.04 to 0.25 ± 0.35 ($p < 0.01$) throughout the monitoring period (ANOVA; $p < 0.04$). This time-profile of PRx, suggesting impaired cerebrovascular pressure reactivity, seemed to agree with a trend of increasing intracranial hypertension. RAP was persistently higher in patients with good recovery than in patients who died. RAP tended to increase over time (up to 30% of monitoring time) and then decrease when ICP started to climb (regression versus time: $R = -0.332$; $p < 0.035$) but only in the patients who died (Fig. 3c). The time-distribution of the slow waves was uniform in both groups with the magnitude of slow waves being persistently lower in patients who died in comparison to those who survived.

Monitored variables in individual cases

Apart from statistical analysis, clinical advantage the monitored parameters can be appreciated using examples of recordings made in individual cases:

A typical intracranial hypertension in a patient who died is presented in Fig. 4a. In this case the progressive increase in ICP caused a severe fall in CPP (time = 48 hours), although normal values of ABP were maintained. PRx, was at first within the normal range, then started to climb in the second part of the monitoring period (time > 40 hours) when the progressive increase in ICP was associated with CPP reduction. Similarly RAP, initially above 0.6, started to fall suggesting a derangement of normal vascular responses, overlapping with poor pressure-volume compensation (time > 40 hours). The magnitude of slow waves was low at the beginning, increasing with time (time between 14 and 36 hours) to decrease almost down to 0 mmHg later (time > 36 hours).

However, such a typical pattern may not always be found in clinical practice. An atypical case of intracranial hypertension with ABP well maintained in a patient who died is presented in Fig. 4b. Cerebrovascular pressure-reactivity was maintained in the normal range as confirmed by PRx values below 0.1 and RAP is constantly above 0.6 suggesting a reduction of pressure-volume compensatory reserve, but without loss of vasomotor responses. Also atypical was the behaviour of the slow waves, which presented with normal amplitude all through the monitoring period and did not suggest any characteristic relationship with the increased ICP values and bad outcome.

A specific time-profile is usually observed in patients with a favourable outcome (Fig. 4c). An increase in ICP

was associated with a reduction in CPP, but PRx values were within the normal range throughout the monitoring time, suggesting preserved cerebral autoregulation. RAP confirmed an intact vasomotor reserve with reduced pressure-volume compensation even during phases of elevated ICP (time between 25 and 30 hours). Similarly, the magnitude of slow wave amplitude was above 1 mmHg, except for few hours after a peak of ICP > 30 mmHg.

Discussion

Intracranial pressure: mean values and time-trends

The role of ICP monitoring in patients with severe head injury has been extensively analysed and it is well recognised that persistently raised ICP correlates with higher risk of mortality [11, 20, 34], however not all patients with intracranial hypertension have poor outcome. Resnick and Marion [29] selectively studied a group of 37 patients with persistent intracranial hypertension (more than 96 hours) and found that 38% of these patients had good outcome, but they did not describe any specific pattern for ICP and CPP in different outcome groups. Unterberg *et al*. [38] described four specific ICP time-profiles in a retrospective-prospective study of 53 head injured patients. They extensively analysed a subgroup of patients characterized by secondary intracranial hypertension whose outcome was particularly poor, but they did not discuss the other ICP-related variables in relation to outcome. In our study we have focused attention on patients with ICP continuously above 25 mmHg for more then 4 hours to define specific patterns for physiological variables and derived indices

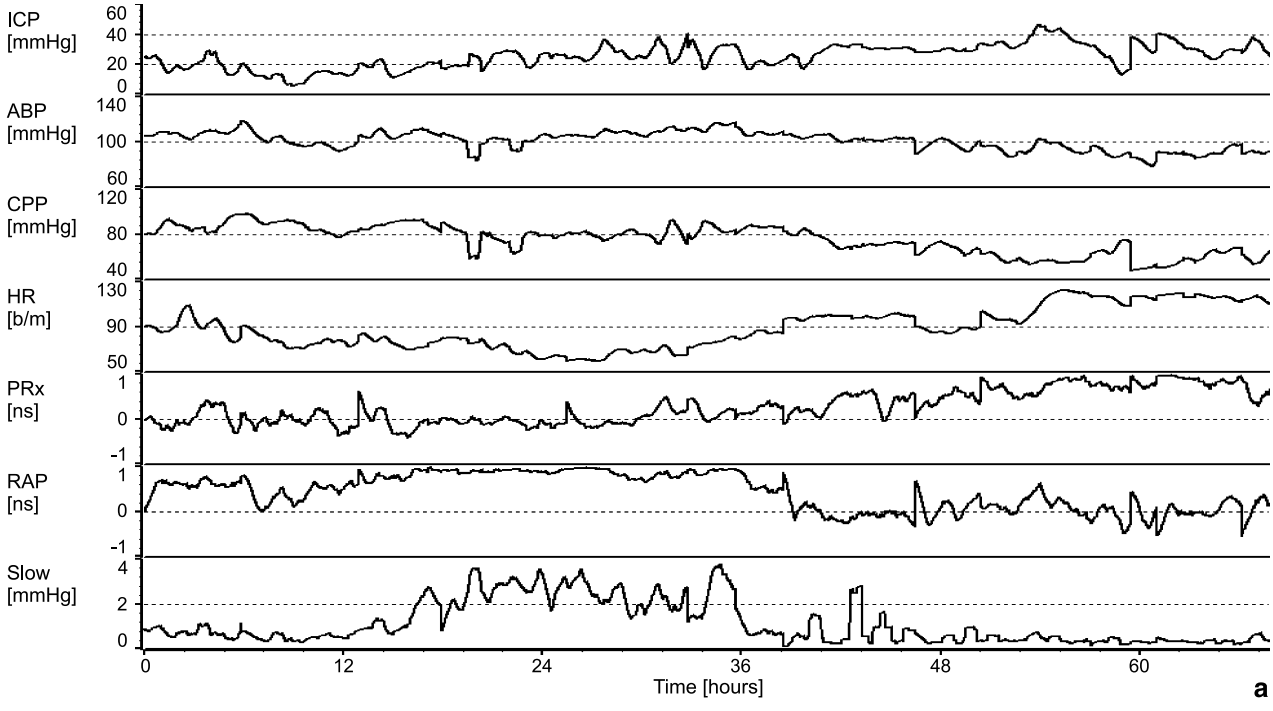
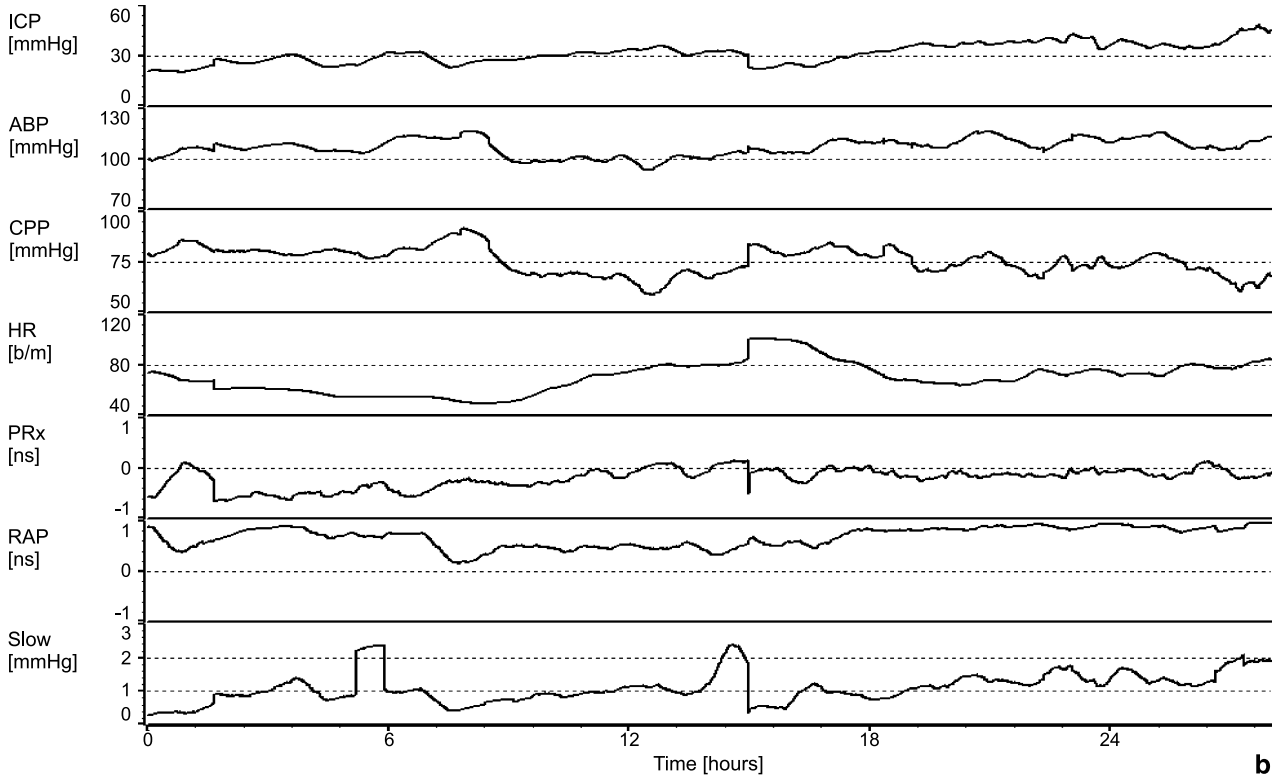


Fig. 4. (a) A typical example of intracranial hypertension in a patient who died. In this case the progressive increase in ICP caused a severe fall in CPP (time = 48 hours), although normal values of ABP were maintained. PRx, was at first slightly above the normal range, started to climb in the second part of the monitoring (time > 40 hours) when the progressive increase in ICP was associated with CPP reduction; similarly RAP, initially above 0.6, started to fall suggesting a loss in vasomotor reserve (time > 40 hours). Slow waves were low at the beginning, increasing with time (time between 14 and 36 hours) to decrease almost down to 0 mmHg later (time > 36 hours). (b) However, such a typical pattern as previously described may not always be found in clinical practice. A case of intracranial hypertension with ABP well maintained above the ischaemic threshold in a patient who died is presented here. Cerebral autoregulation was maintained in the normal range as confirmed by PRx values below 0.2 and RAP is constantly above 0.6 suggesting a reduction of compensatory reserve, but without loss of vasomotor responses. Also atypical is the slow waves behaviour whose normal amplitude during the monitoring period doesn't suggest any characteristic relationship with increased ICP values and bad outcome. (c) A different profile is observed in patients with favourable outcome. The ICP increase is associated with a reduction in CPP, but PRx values are within the normal range throughout the monitoring time suggesting a preserved cerebral autoregulation. RAP confirms an intact vasomotor reserve also during phases of elevated ICP. Similarly slow waves amplitude is above 1 mmHg, except for few hours after a peak of ICP above 30 mmHg



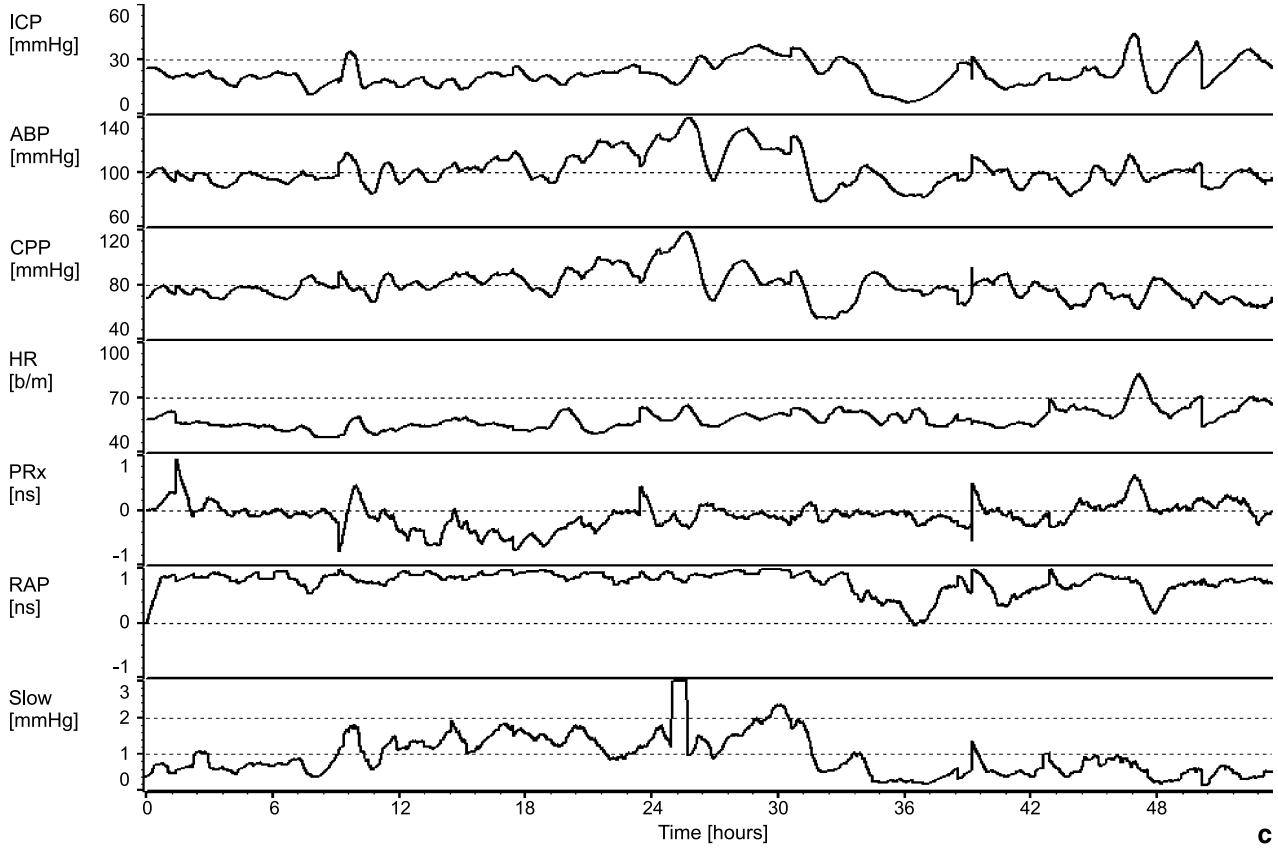


Fig. 4 (*continued*)

in patients whose intracranial hypertension was generally recognized as requiring aggressive treatment.

Although systemic hypotension is one of the major factors leading to secondary ischaemic damage and consequently poor outcome, we could not find a significant difference in mean values and time-trends of ABP between the two outcome groups, confirming good general medical treatment and adoption of 'CPP-oriented' therapy.

Indices of vasomotor and compensatory reserve: PRx and RAP

The index of cerebrovascular pressure reactivity PRx [11] was significantly higher ($PRx > 0.2$) in patients who died. This relationship was observed regardless of whether PRx was expressed as mean value for the entire monitoring period or as time-profile, with an increasing trend in time as ICP rises. This is in agreement with other reports that describe the importance of autoregulation for outcome, since many methods have been described for its assessment [1, 10, 13, 16, 26, 33, 35]. However, impaired cerebral autoregulation may be either an indicator of severe injury or a determinant of progressive decompensation. Indeed, patients who died were elder and admission GCS was significantly worse. Unfortunately, we were unable to derive full data about pre-admission hypoxic/hypotensive events. These factors may also contribute both to poor outcome and disturb normal control of CBF. However, these would exceed the scope of our study: Large number of new and more sophisticated implications ranging from overall intensity of intensive treatment, to genetics [37] has been highlighted recently.

The increased mean value of RAP in patients with favourable outcome is indicative of poor pressure-volume compensatory reserve with preserved cerebrovascular pressure-reactivity in the presence of intracranial hypertension. In contrast, the time-profile of RAP in patients who died stressed that RAP increased with time then rapidly decreased as ICP started to rise. This pattern is characteristic of a vascular system that is close to exhaustion of the compensatory reserve either in term of volume-compensation or vasomotor reserve.

Slow waves

Of all ICP-derived indices, the mean magnitude of slow waves provided the best discrimination between favourable and dead outcome groups. Slow waves have been studied less frequently in head injured patients [21] than in other brain disorders such as hydrocephalus or craniosynostosis. Slow waves, also known as Lundberg's 'B' waves, are probably a manifestation of variations of cerebral blood volume by mechanisms that remain unclear [21]. Their disappearance in dying patients coincides with failure of autoregulation (indicated by higher PRx) and exhaustion of vasomotor reserve (fall of RAP) at high ICP level.

To combine the value of 'slow waves' and ICP we have calculated the slow wave ratio (SWR) defined as the ratio between 'magnitude of slow wave' and mean ICP. This index could be a helpful predictor of the fatal outcome since slow waves were so significantly lower and mean ICP was greater in patients who died. Indeed, the significance level of the difference between the mean values of SWR in the two groups of patients is the highest we found in our data ($p < 0.00001$). However its value should be confirmed by a prospective study.

It is impossible to state definitely whether monitored ICP-derived indices can anticipate rises of ICP. Their time profiles are highly individual. Time-to-event analysis is difficult as the uncertainty of definition 'dangerous ICP rise' is substantial. Based on our data we can only state that they rather signify than anticipate increases in ICP which may contribute to fatal outcome following head trauma.

Limitations

In our analysis we looked at a selected group of patients. It is not clear that the same finding would hold if the analyses were applied to a more general population. But this should be a subject of prospective study.

We also have not analyzed CT scans. It would be interesting whether indices of ICP waveform have anything in common with characteristic future of brain images. Any value of the analyzed parameters in predicting outcome after head injury should be confirmed in a prospective study.

Comparison between the described parameters and other method of ICP waveform analysis also awaits investigation.

Only patients with good and fatal outcomes were compared. We designed the study in this way as these two groups of patients are probably best-separated by the selected variables. We have not included patients with severe disability to trace the differences in ICP waveform in patients having radically different outcome. Again, prospective study will be conducted to cover whole population of head injured patients. Our previous studies [10, 11] indicate that PRx and RAP coefficient

differentiate patients who died from those who survived. Differentiation between severe disability and favourable outcome was rather difficult. There is no published data about slow waves, so far.

Conclusion

We described the differences in the cerebrovascular pathophysiology in patients with sustained intracranial hypertension, in an attempt to determine the variables that best discriminate between death and a favourable outcome following brain trauma. Slow waves were found to have decreased magnitude in patients who died.

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Comments

The manuscript entitled ''Intracranial hypertension: What additional information can be derived from ICP waveform after head injury'' is an interesting analysis of several parameters that have been used to describe changes in the ICP waveform, including the pressure reactivity index and the index of pressure-volume compensatory reserve.

Because the idea was to determine what additional information (besides the JCP value) could be obtained with the waveform analysis, the investigators analysed a very select group of patients with sustained intracranial hypertension. Furthermore, they compared these parameters in patients with a good recovery (GOS-GR or MD) and in patients who died. They found significant differences in all of the waveform parameters that were measured, but they also had differences in age, GCS, and the actual average level of ICP and CPP.

The paper is well-written. Although the parameters that were measured are complex, the concepts are clearly explained. And the topic is of interest now since computerized data collection systems in the ICU are widespread, and there is a desire to be able to anticipate early those patients who will do poorly.

The major weakness in the paper is the simplicity of the statistical analysis. The question posed by the authors in the title really requires a multivariate technique to demonstrate that the ICP waveform parameters are not simply related to the higher ICP or the more severe injury seen in the patients who died. Secondly, the investigators only looked at a select population. It is not clear that the same findings would hold if the analyses were applied to a more general population.

C. S. Robertson

The authors present a detailed retrospective analysis of ICP waveform derived indices in a group of 96 patients with persistent intracranial hypertension and try to identify patterns of change that distinguish groups that have favourable and unfavourable outcomes. They were able to demonstrate differences in their derived indices between patients with good outcome (GOS 4 & 5) and patients that died although there were other differences between these groups in terms of injury severity, age and absolute levels of ICP inter alia. The topic is of interest and relevance given the increasing availability of computerised multimodality bedside monitoring. The population studied is highly selected and the results may not be applicable to the more general head injury ITU population. To be of clinical use it will be necessary to demonstrate that waveform analysis has real-time independent predictive value additional to currently known predictors of outcome. This would require further prospective studies, as would comment on the potential interaction between therapeutic interventions and the described waveform indices.

L. T. Dunn

Despite the lack of a significant evidence base to support the use of intracranial pressure monitoring in patients with a severe head injury, many neurosurgical units would have difficulty in not being able to identify changes in the intracranial dynamics. The development of new transducer technologies has made the use of such monitoring much easier, however this has not been followed by any successful methodology that can provide ''added value'' to the simple numerical information.

This is a relatively small study *f* and the findings that there are significant differences between the independent and poor outcome groups in the waveform parameters that were measured need to be extended to a larger series. If the results can be replicated in a larger group where differences in age and GCS can be accounted for then this technique would show promise as a tool to help identify those patients who are likely to make a poor outcome at an early stage.

Advances in monitoring systems now mean that there are sophisticated computers already at the bedside with considerable computational resources that are not fully exploited. If additional information that resides within the analysis of ICP recordings can be used reliable within the clinical rather than the research environment then it should be possible to include such a facility within the monitoring systems. This would be essential to rollout the technique to routine clinical practice.

I. R. Chambers

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