Plateau waves of intracranial pressure (ICP) were first described in 1950 by Janny,20 who named them “sudden rises in ICP;” however, the name and definition proposed in 1960 by Lundberg 23 became widely accepted within clinical terminology. Plateau waves can be observed in patients suffering from various cerebral pathological conditions such as subarachnoid hemorrhage,17,18 head injury,2,33,34 brain tumor,24 acute hydrocephalus,13,15,38 craniosynostosis,3,31 and benign intracranial hypertension.15

During the wave, ICP increases dramatically over a few minutes from a normal or moderately elevated ICP to gross intracranial hypertension (50–100 mm Hg). In his original definition, Lundberg23 described the duration of a plateau wave as never shorter than 15 to 20 minutes. Later studies15–17,24,31,34 did not specify the minimum duration of the wave so rigorously.

The dramatic increase in ICP was followed by a profound fall in CPP (by 45%). In contrast, flow velocity fell by only 20%. Autoregulation was documented to be intact both before and after plateau but was disturbed during the wave (p < 0.05). Pressure-volume compensatory reserve was always depleted before the wave. Cerebrovascular resistance decreased during the wave by 60% (p < 0.05) and TCD pulsatility increased (p < 0.05). Plateau waves did not increase the probability of an unfavorable outcome following injury.

Conclusions: The authors have confirmed that the plateau waves are a hemodynamic phenomenon associated with cerebrovascular vasodilation. They are observed in patients with preserved cerebral autoregulation but reduced pressure-volume compensatory reserve.

Key Words • head injury • plateau wave • intracranial pressure • ultrasound • autoregulation
ing structure, which suggested that the positive vasodilatory feedback loop could not be sustained by decreasing CPP alone in the absence of an active vasodilatory component.

In this report we describe the course of plateau waves by using our modern multimodal monitoring system, which includes ICP, ABP, and transcranial Doppler (TCD) ultrasonography, to study changes in cerebrovascular resistance, autoregulation, vascular reactivity, and pulsatile components of intracerebral waveforms and to correlate observations with clinical findings including outcome.

Clinical Material and Methods

Patient Population

Clinical data derived from the continuous or intermittent monitoring of 160 head-injured patients admitted to Addenbrooke’s Hospital (1992–1995) with a mean Glasgow Coma Scale (GCS) score of 6 (range of admission GCS Scores 3–13) was reviewed for this study. There were 40 females and 120 males ranging in age from 6 to 74 years (mean age 29 years). All patients were paralyzed, sedated, and ventilated to achieve mild hypocapnia (3.5–4 kPa). Alternating colloid and normal saline infusions, with addition of inotropic agents (infusion of dopamine at a rate of 2–15 µg/kg/minute), were used selectively to prevent arterial hypotension, leading to a reduction in CPP to below 60 mm Hg. Boluses of mannitol (200 ml of 20% solution administered over a period of ≥20 minutes) were given to manage episodes of ICP higher than 25 mm Hg and lasting longer than 15 minutes. Outcome was assessed 6 months or more after injury by using the Glasgow Outcome Scale.

Computerized tomography (CT) scans obtained at admission and during the acute phase of the illness were examined for symptoms of decreased volume-pressure compensatory reserve such as tight brain (effacement of sulci, basal cisterns, or ventricles), mass lesion (contusion, hematoma, or diffused axonal injury), midline shift, or hydrocephalus.

Patient Monitoring

All 160 patients underwent continuous monitoring of ICP, ABP, and other cerebral modalities with the results stored as trends of time-averaged values. The whole group was used to study the prevalence of plateau waves and its association with other clinical findings.

Ninety-six patients in whom waveforms of ABP, ICP, and middle cerebral artery (MCA) blood flow velocity (FV) were recorded intermittently (daily), were chosen for the time-dependent analysis of the hemodynamic effects of plateau waves, including changes in vascular reactivity and waveform envelopes.

Arterial blood pressure was monitored directly in the radial or dorsalis pedis artery. Intracranial pressure was monitored continuously by using a fiberoptic transducer; the catheter tip was inserted intraparenchymally into the right frontal region.

The MCA blood FV was measured using TCD ultrasonography daily for 20 minutes to 4 hours starting from the day of admission until discharge. Recordings were obtained during periods of stable respiration that were undisturbed by physiotherapy. Periods of tracheal suction or other nursing interventions were eliminated from recordings before further analysis.

Data Capture

Analog outputs from the ICP, ABP, and TCD ultrasonography units (the maximum frequency envelope was used) and bedside units used for other monitoring modalities were connected to a 386SX laptop computer fitted to an analog-to-digital converter. In all patients the digital
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monitoring system ICM, developed in house, was used for time-averaging (0.5–2 minutes, depending on the variability of input signals) to convert the monitored modalities into their time-dependent mean values.

In 96 patients in whom intermittent waveform recording was performed, signals from ABP, ICP, and TCD monitoring were sampled at a rate of 30 or 50 Hz and stored on the computer hard disk by using specific software for waveform recording.

Waveform Analysis

From more than 450 digital recordings obtained in 96 patients, only 16 were selected (belonging to eight patients) in which a whole course of plateau waves was included. Digital data were processed off line by using software developed in house. Time-averaged values of ICP, ABP, CPP (CPP = ABP – ICP), and MCA blood FV were calculated using waveform time-integration over 6-second intervals. Fast Fourier transform was used to calculate the fundamental harmonic components for pulse waveforms of ICP, ABP, and FV (denoted as ICPa, ABPa, and FVa, respectively). The pulsatility index (FVa) and cerebrovascular pressure resistance (mean CPP – mean FV) were calculated. Results of these calculations were used to compute the following secondary indices that described cerebral autoregulation and cerebrovascular pressure reactivity.

1) Index of Autoregulation. This index, also known as the Mx, is calculated as the moving correlation coefficient between 40 samples (width of time window was just > 3 minutes) of mean FV and CPP. A positive correlation signifies passive dependence of blood flow on CPP; that is, disturbed autoregulation. Zero or negative correlation characterizes good autoregulation that is the blood flow independent of changes in CPP?

2) Index of Pressure Reactivity. The pressure reactivity index (PRx) is calculated similarly to the Mx, but as a moving correlation coefficient between slow waves in ICP and ABP. Its positive association between these waves reveals a passive relationship between ABP, CBV, and ICP, indicating that the cerebral arterial bed is unreactive. A negative or zero PRx indicates a good cerebrovascular pressure–reactivity reserve.

3) Index of Cerebrospinal Compensatory Reserve. This index, known as the RAP, is calculated as a moving correlation coefficient between 40 values of mean ICP and ICPa. This index reflects the level of association between the pulse amplitude of ICP and the mean ICP. A positive correlation between pulse amplitude and mean ICP is disturbed (that is, RAP decreases) in states of maximum cerebrovascular vasodilation when ICP is very high and CPP is low, that is, the RAP index decreases to zero or negative values.

The mean values, pulse amplitudes, pulsatility indices, cerebrovascular resistance, and all secondary indices were time averaged over at least 3 to 5 minutes before the plateau wave, during the maximum phase of the plateau wave, and just after the plateau wave was recorded in eight patients (16 waves were recorded; the values for subsequent waves recorded repetitively in one patient

<table>
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<th>Table 1</th>
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<td>Mean values of pressure and hemodynamic parameters found in eight patients before, during, and after a plateau wave*</td>
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<tr>
<td>Factor</td>
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<tr>
<td>mean ICP (mm Hg)</td>
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<td>mean CPP (mm Hg)</td>
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<td>mean FV (cm/sec)</td>
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<td>CVR (mm Hg/ [cm/sec])</td>
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* Values are expressed as the mean ± SD. Significance levels of the differences between change in parameters before and during the wave are given (paired signed-rank test). Differences in values seen before and after the wave were all nonsignificant. Abbreviations: ABPa = pulse amplitude (first harmonic) of arterial pressure; AMP/p = slope of intracranial amplitude-pressure regression line; FV = blood FV in the MCA; Fva = pulse amplitude (first harmonic) of blood FV; HR = heart rate; NS = not significant; PI = pulsatility index.

were averaged). A paired signed-rank test was used to compare parameters calculated before, during, and after the plateau waves.

Sources of Supplies and Equipment

Arterial pressure was monitored using the System 8000 monitor available from S & W Vickers Ltd. (Sidcup, United Kingdom). Intracranial pressure was monitored using either the Camino Direct Pressure Monitor, obtained from Camino Laboratories (San Diego, CA) or the Codman Microsensors ICP Transducer, obtained from Codman & Shurtleff, Inc. (Randolph, MA). The MCA blood FV was measured using either the PCDop 842 Doppler Ultrasound Unit, purchased from Scimed (Bristol, United Kingdom) or the Neuroguard system, purchased from Medasonics (Fremont, CA). The laptop computer used for data capture was obtained from International Business Machines (AM-STRAD, Ltd., Kowloon, Hong Kong, PRC), and the DT 2814 analog-to-digital converter was obtained from Data Translation (Marlboro, MA).

Results

Observations of the Time Course of Plateau Waves

In eight of 31 patients with ICP plateau waves, 16 waves were recorded during the time period in which TCD investigations of MCA blood FV were made. Typical recordings of plateau waves are presented in Fig. 1. In most cases (11 of 16 waves) a short spontaneous decrease in ABP can be observed at the beginning of the plateau wave (Fig. 1A). In three cases, a decrease in ABP was not obvious. In those cases regular vasogenic waves of ICP were present, during which reduction in CPP may be the reason for the onset of the vasodilatory cascade (Fig. 1B).
The systolic–diastolic amplitude of blood FV always increased during the plateau phase of the wave. The average duration of the wave was 12.8 ± 4 minutes (mean ± standard deviation [SD]) and the rate of the pressure increase was 7.3 ± 2.5 mm Hg/minute. An increase in ABP, as a prelude to termination of the plateau wave, could be seen in only four waves. In seven other waves, a notch decrease in ABP followed by an increase (Fig. 1A) was recorded. In five waves the ABP was perfectly stable at the termination of the plateau wave in ICP.

Hemodynamic Profile of the Plateau Wave

The mean values and SDs of the calculated variables monitored before, during, and just after the plateau wave are given in Table 1. As expected, an increase in ICP corresponded to a significant drop in CPP (by 45%) with the ABP remaining unchanged. The mean FV decreased significantly but by only 20%. Calculated cerebrovascular resistance (CPP/FV) also decreased (p < 0.05).

The pulse amplitudes of both ICP and FV increased significantly (p < 0.05), whereas the amplitude of ABP remained unchanged. The TCD pulsatility index increased during the wave (p < 0.04).

Time-Dependent Changes in Autoregulation and Volume Compensation

Secondary indices, describing autoregulatory and compensatory reserves, all varied significantly (p < 0.05; Table 1). Before the wave, autoregulation and vascular reactivity were preserved (both PRx and Mx were nonsignificantly different from zero), whereas during the plateau wave, both indices increased significantly, indicating a temporary loss of the autoregulatory reserve (Fig. 2). Pressure-volume compensatory reserve was compromised before the wave (RAP index was high, close to +1). It decreased significantly during the wave, indicating a state of maximum vasodilation and derangement of the normal association between pulse amplitude of ICP and mean ICP.

Waveform Analysis

Waveforms of ICP and FV became more pulsatile during a plateau wave, with ICPa and FVa both increasing during the wave (Fig. 3 and Table 1). The ICP pulse wave became more of a triangular shape and the TCD waveform exhibited a much flatter diastolic profile (Fig. 4). A plot of the relationship between pulse amplitude of ICP and mean ICP or mean CPP (Fig. 5) showed the sections of positive and negative slopes, respectively, with nonlinear distortions at high ICPS and low CPPs (polynomial regression of order 5, goodness of fit better than r² = 0.75).
Both of these “breakpoints” correspond to a significant decrease in the RAP coefficient (see Table 1) and may be interpreted as entering the stage of maximum vasodilation (see Discussion).

Occurrence of Plateau Waves and Clinical Correlation

Plateau waves of ICP were seen in 31 (19%) of 160 patients with variable frequency ranging from two to three episodes during the whole monitoring period to once every hour. Occurrence of plateau waves was not related to the severity of injury (the median GCS score was 6 in patients with a plateau wave and 5 in patients without a wave) or to the patient’s age. Forty-eight percent of patients with a plateau wave had a favorable outcome compared with 46% of patients without a plateau wave, a difference that was not significant. The mortality rate for patients with plateau waves was 12% compared with 29% for all patients (that is, with and without a plateau wave; \(p < 0.04\)). Plateau waves were seen predominantly in patients in whom CT scans revealed any reason for possibly decreased pressure–volume compensatory reserve. Ninety-two percent of patients suffered from tight brain (classified as an effacement of ventricles or sulci or basal cisterns), 79% had hematomas, and 83% showed a significant region of brain contusion.

Which ICP Vasogenic Waves are Plateau Waves?

A variety of deep ICP waves of a magnitude within the range of plateau waves (\(\geq 40\) mm Hg) can be seen in continuous recordings of ICP in head-injured patients. However, not all of them can be classified as plateau waves according to the Rosner theory of vasodilatory cascade.\(^{33,34}\)

There are ICP waves with a sharply rising edge related to a sudden increase in ABP. They are seen in nonautoregulating patients and are characteristic of a passive transmission of pressure changes from the arterial to the intracranial compartment (Fig. 6a). Another kind of wave can be observed in autoregulating patients when ABP decreases temporarily and ICP increases inversely to the course of change in ABP (Fig. 6b). Contrary to the “true” plateau wave, this type of increase in ICP lasts only as long as the ABP remains reduced, and a self-sustaining “vasodilatory cascade” is never initiated. Finally, high vasogenic ICP waves may be seen during episodes of hyperemia (Fig. 6c). This type of wave cannot be distinguished from a genuine plateau wave without direct or indirect monitoring of cerebral blood flow or waveform analysis of ICP (the RAP coefficient does not decrease at the top of such waves).

Discussion

Plateau Waves and the Concept of a Vasodilatory Cascade

Our results seem to support the Rosner model of vasodilatory cascade.\(^{33,34}\) First, we demonstrated that plateau waves can be recorded only in patients in whom autoregulation was well preserved at the baseline. Second, we noted a consistent decrease in autoregulatory reserve at the top of each plateau wave. This finding, taken together with a decrease in cerebrovascular resistance seen during the wave, indicates that cerebral vasodilation is an important component of a plateau elevation in ICP.

However, is this mechanism always responsible for the onset of plateau waves? During the waves, a consistent
decrease in CPP can be observed. In autoregulating patients, a decrease in CPP always produces a decrease in cerebrovascular resistance and is followed by a disturbance in autoregulation and an increase in the TCD pulsatility index and the pulse amplitude of ICP. Thus, if spontaneous or stimulated vasodilation or an increase in venous blood volume provoked a plateau wave, all the hemodynamic changes we documented would appear to be the normal consequence of a decrease in CPP. There was no time delay between changes in the pulsatility of the recorded waveform, cerebrovascular resistance, and the onset of a plateau wave. This makes it difficult to elucidate what is the cause and what is the effect of the increase in CBV observed during the wave.

The second component, often referred to as a precondition for the vasodilatory cascade to develop, is a low cerebrospinal compliance. This precondition is consistent with the CT signs of tight brain or mass lesion in all patients with plateau waves. Also the RAP coefficient was elevated, which indicates a low pressure–volume compensatory reserve.

Change in Pulse Waveforms

The observed increase in the amplitude of blood FV can
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be attributed to cerebral vasodilation. It is known that
the compliance of large cerebral arteries increases in par-
allel with a decrease in cerebrovascular resistance. Therefore, blood flow encounters a lower hydrodynamic
impedance to the pulsatile component.

In spite of a decrease in cerebrovascular resistance, the
pulsatility index increases during plateau waves (as previ-
ously reported elsewhere). This is a good illustration of
the concept that the pulsatility index depends inversely on
the CPP, and is not a descriptor of cerebrovascular resis-
tance, as is commonly extrapolated from studies on pe-
ripheral circulation.

The relationship between pulse amplitude and mean
ICP has confirmed the earlier findings of Avezaat and van
Eijndhoven. The slope of the amplitude-pressure regression
line increases during the plateau wave (Table 1 and
Fig. 4a). This may be caused by a decrease in brain com-
pliance, an increase in cerebral pulsatile volume load (al-
so documented by TCD ultrasonography as an increase in
the FV pulse amplitude; Table 1), or an increase in com-
pliance of big cerebral arteries due to vasodilation. It is
likely that all these mechanisms act in parallel. The rela-
tionship between ICP pulse amplitude and CPP shows a
lower breakpoint at 30 to 40 mm Hg, probably indicating
the critical switching point below which maximally dilat-
ed arteries begin to collapse and the CPP-dependent
cerebral pressure–volume index starts to increase.

Plateau Waves and Outcome

Contrary to conventional wisdom, the incidence of pla-
teeu waves apparently did not reduce the rate of favorable
outcomes following head injury. Of course, all plateau
waves in our patients were treated by boluses of mannitol
(if the waves lasted > 15 minutes), and any conclusion
based on this limited group of 31 must be tentative.

The incidence of deaths was lower (p < 0.05) in pa-
tients in whom plateau waves were present. This paradox-
ical finding needs to be confirmed in a bigger study, but
merits discussion.

As we demonstrated (Table 1), the average decrease in
blood FV during a plateau wave, although significant, is
only 20% of the baseline. This finding is consistent with
other reports in which either CBF monitoring or TCD
ultrasonography was used. By comparison, the “safety
limit” applied in carotid artery surgery to avoid ischemia
is a decrease in FV of more than 60% (for a duration of
20–30 minutes). Therefore, during plateau waves, the
ischemic insult was rarely severe, at least as reflected by
MCA blood FV. Very deep waves, in which CPP decreases
to below 30 mm Hg and FV to 60% of baseline, are
rarely seen (Fig. 7). This concept is not entirely novel.

Finally, during plateau waves, the presence of the vaso-
dilatory cascade presumes that vascular reactivity is pre-
served. Preserved autoregulation and pressure-reactivity
indices have proved to be powerful determinants of favor-
able outcome following head trauma. The drop in CPP
during waves was always below the apparent threshold
(60 mm Hg) for secondary brain insults. Hence, the
method of counting CPP insults, without taking into ac-
count the state of autoregulation, may be misleading and
should be refined appropriately.

To Intervene or Not to Intervene?

It is obvious that plateau waves in head injury could not
be interpreted as a benign phenomenon. They are always
associated with a picture of reduced brain compliance on
CT scanning; however, one should remember that this is
usually a reason for and not a result of plateau waves.
Plateau waves can build on themselves, increasing the
baseline ICP level until there is terminal reduction in CPP
causing irreversible ischemia. However, our material does
not contain such a case, probably because of active treat-
ment of ICP when baseline pressure increased above 20
mm Hg. Long plateau waves, described by Lundberg as
lasting 1 to several hours, were not observed, again prob-
ably because of active treatment. However, there is no
question that plateau waves should be treated actively in
states after severe head injury. Two years ago we started
to hyperventilate patients manually with a Walters bag
whenever an onset of plateau wave was observed, a ma-
neuver that may be performed safely for 1 to 2 minutes,
decreasing end-tidal CO2 to 25 mm Hg. This can cause
vasoconstriction, reversing vasodilatory cascade (if the pa-
value 32 because they reveal preserved autoregulation but terminating intermittent manual hyperventilation. Plateau waves should be treated actively, for example, using is ischemic level. If such monitoring is not available, ICP blood flow encountered during a wave is reaching an sudden elevations of ICP to decide when the decrease in ever, hemodynamic monitoring is necessary during all decreased volume–pressure compensatory reserve. However, how- ever, hemodynamic monitoring is necessary during all sudden elevations of ICP to decide when the decrease in blood flow encountered during a wave is reaching an ischemic level. If such monitoring is not available, ICP plateau waves should be treated actively, for example, using intermittent manual hyperventilation.

Conclusions

We can confirm that plateau waves have diagnostic value because they reveal preserved autoregulation but decreased volume–pressure compensatory reserve. However, hemodynamic monitoring is necessary during all sudden elevations of ICP to decide when the decrease in blood flow encountered during a wave is reaching an ischemic level. If such monitoring is not available, ICP plateau waves should be treated actively, for example, using intermittent manual hyperventilation.

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