

The relationship of blood flow velocity fluctuations to intracranial pressure B waves

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✓ Intracranial pressure (ICP) and continuous transcranial Doppler ultrasound signals were monitored in 20 head-injured patients and simultaneous synchronous fluctuations of middle cerebral artery (MCA) velocity and B waves of the ICP were observed. Continuous simultaneous monitoring of MCA velocity, ICP, arterial blood pressure, and expired CO₂ revealed that both velocity waves and B waves occurred despite a constant CO₂ concentration in ventilated patients and were usually not accompanied by fluctuations in the arterial blood pressure. Additional recordings from the extracranial carotid artery during the ICP B waves revealed similar synchronous fluctuations in the velocity of this artery, strongly supporting the hypothesis that blood flow fluctuations produce the velocity waves. The ratio between ICP wave amplitude and velocity wave amplitude was highly correlated to the ICP ($r = 0.81$, $p < 0.001$). Velocity waves of similar characteristics and frequency, but usually of shorter duration, were observed in seven of 10 normal subjects in whom MCA velocity was recorded for 1 hour. The findings in this report strongly suggest that B waves in the ICP are a secondary effect of vasomotor waves, producing cerebral blood flow fluctuations that become amplified in the ICP tracing, in states of reduced intracranial compliance.

KEY WORDS • intracranial pressure • B wave • transcranial Doppler ultrasound • intracranial pressure monitoring • head injury

LUNDBERG¹³ was the first to observe B waves in the intracranial pressure (ICP); these waves are characterized as repetitive alterations in the ICP at frequencies of 0.5 to 2 waves per minute. The etiology and mechanism responsible for this phenomenon are unclear; however, the alterations have been attributed to cerebral blood flow (CBF) fluctuations, thought to be secondary to respiratory variations during abnormal breathing patterns.¹³ Previous observations using standard transcranial Doppler ultrasound (TCD) monitoring have demonstrated velocity fluctuations at frequencies similar to those of B waves in head-injured patients and also in normal subjects.^{12,17,18} Simultaneous recording of TCD and ICP signals has demonstrated synchronous variations in both parameters.¹²

Recent advances in TCD and digital signal processing allow continuous recording of velocity signals from the basal intracranial arteries simultaneously with other physiological parameters. Moreover, the relationship of these parameters can be compared using computer analysis. These techniques allow new insights into the pathophysiology of intracranial hemodynamics in head-injured patients. The present study investigates the hy-

pothesis that B waves are primarily due to vasomotor waves of the regulating vessels in the cerebral circulation, which are independent of respiration. It is further hypothesized that these vasomotor waves in turn produce fluctuations in CBF and cerebral blood volume which are then reflected in the ICP tracing, more prominently when the intracranial compliance is low.

Clinical Material and Methods

Simultaneous monitoring of arterial blood pressure, middle cerebral artery (MCA) velocity, ICP, and end-tidal CO₂ was performed on 20 patients admitted with a diagnosis of closed head injury to the intensive care unit at Inselspital, in Bern, Switzerland. A total of 150 continuous recordings were obtained for intervals ranging from 15 minutes to 2 hours. All patients were intubated and artificially ventilated; agitated patients were mildly sedated using midazolam or paralyzed using muscle relaxants. Patient ages ranged between 17 and 59 years (average 35 years); there were 17 males and three females. Admission Glasgow Coma Scale (GCS) scores ranged between 3 and 10, with an average score of 6. Patients were treated using a standard ap-

proach, with early surgical evacuation of mass lesions. Postoperative ICP was monitored in many patients, which accounted for the inclusion of some with a higher GCS score. Moderate hyperventilation and intravenous mannitol administration were used to control elevated ICP.

Blood pressure monitoring was accomplished with standard intra-arterial catheters and pressure transducers. Intracranial pressure was monitored using an epidural pressure recording device,* which was inserted through a burr hole. Continuous end-tidal CO₂ signals were obtained using side-port sampling with an infrared CO₂ detector. A 2-MHz pulsed range-gated ultrasound transducer, fixed with a headband, was used for TCD monitoring. Signals were recorded from the MCA trunk at a depth between 50 and 60 mm, located using previously accepted criteria.² The spectral display was continuously observed to insure signal consistency. The spectral outline that corresponds to the maximum velocity (V_{max}) of blood flow at the center of the artery was then taken as an analog signal and processed with three other analog signals (arterial blood pressure, ICP, and end-tidal CO₂) through an analog-to-digital converter. These waveforms were sampled at a 50-Hz frequency and stored digitally for analysis by an IBM-AT-compatible 386 personal computer.

In five head-injured patients, the extracranial internal carotid artery velocity was monitored simultaneously with arterial blood pressure, ICP, and end-tidal CO₂ during ICP wave recording. This was accomplished using a hand-held 2-MHz probe, set at a depth of 50 mm and aimed from the angle of the mandible toward the foramen lacerum. All head-injured patients had velocity recordings obtained from both extracranial internal carotid arteries as part of their initial examination, and both arteries were patent in all.

Recordings were also made of MCA velocity and end-tidal CO₂ in a group of 10 healthy volunteers with no history of cerebrovascular disease. There were five men and five women, with ages ranging from 20 to 43 years (average 31 years). With the subjects on a stretcher, continuous recording was performed from the left MCA for 1 hour. Continuous simultaneous CO₂ monitoring was conducted on expired air through a tight-fitting mask with an infrared CO₂ detector.

Data Analysis

Data were analyzed using an IBM-AT-compatible 386 personal computer with a numerical coprocessor. Custom software, written by one of the authors (R.A.), was used to review the stored digital tracings from all patients. Those with continuous data recorded for at least 1 hour (between 1 and 2 hours) were selected for further analysis; the recordings in this group (69 recordings from 17 patients) were analyzed for the presence of repeating fluctuations of the MCA velocity occur-

ring at a frequency of 0.5 to 2 cycles per minute and were accompanied by similar fluctuations in the ICP. Fourier analysis was performed in selected cases in order to determine the amplitude-frequency relationships between the velocity waves and the ICP waves and to determine the relationship between these waves and the arterial blood pressure and CO₂.

The effect of ICP on the velocity wave amplitude was examined using a linear regression analysis of the two parameters during a typical wave episode in each tracing of more than 1 hour. The effect of ICP on the ICP wave amplitude was also examined using an exponential regression analysis of values obtained in this interval. Finally, the relationship of the degree of ICP fluctuation to a given degree of velocity fluctuation was analyzed; this was done by calculating the ICP wave amplitude during a typical occurrence of waves in each tracing of more than 1 hour that demonstrated waves. The ICP amplitude was divided by the amplitude of the MCA velocity, which was expressed as a percentage of the average mean velocity during the same wave. An exponential regression analysis was then performed comparing this ratio to the average ICP values when they were obtained.

Results

Recordings in Head-Injured Patients

Continuously monitored tracings lasted at least 1 hour in 69 of the 150 recordings. In 38 (55%) of the 69 recordings, repeat fluctuations in the MCA velocity were observed at a frequency of 0.5 to 2 waves per minute, each lasting at least 2 minutes. These velocity waves were completely synchronous with waves in the ICP tracing, and continued for quite variable periods, ranging from 2 minutes to the entire recording period in some patients (Fig. 1). The velocity waves often began and subsided with no identifiable cause; at times they were irregular in frequency and at other times they occurred quite regularly. They were always in phase with the ICP waves. Rapid changes in blood pressure did not appear to influence the waves. Hyperventilation did not abolish the waves but usually reduced the velocity and ICP wave amplitudes.

Fourier analysis was performed on all four simultaneously recorded parameters (arterial blood pressure, MCA velocity, ICP, and end-tidal CO₂) during selected intervals of waves to determine their relationship to each other. It was found that, during typical waves, the MCA velocity and ICP showed the same frequency-amplitude relationship; the arterial blood pressure and end-tidal CO₂ did not typically share this same frequency-amplitude relationship. Figure 1 illustrates a Fourier analysis showing the ventilator frequency affecting all four parameters and the velocity wave frequency affecting ICP, but no changes in arterial blood pressure or end-tidal CO₂ are seen. Occasionally, small blood pressure perturbations were found synchronous with the peaks of the waves, but there were many

* Epidural pressure recording device manufactured by Galtec, Ltd., Dunvegan, Scotland.

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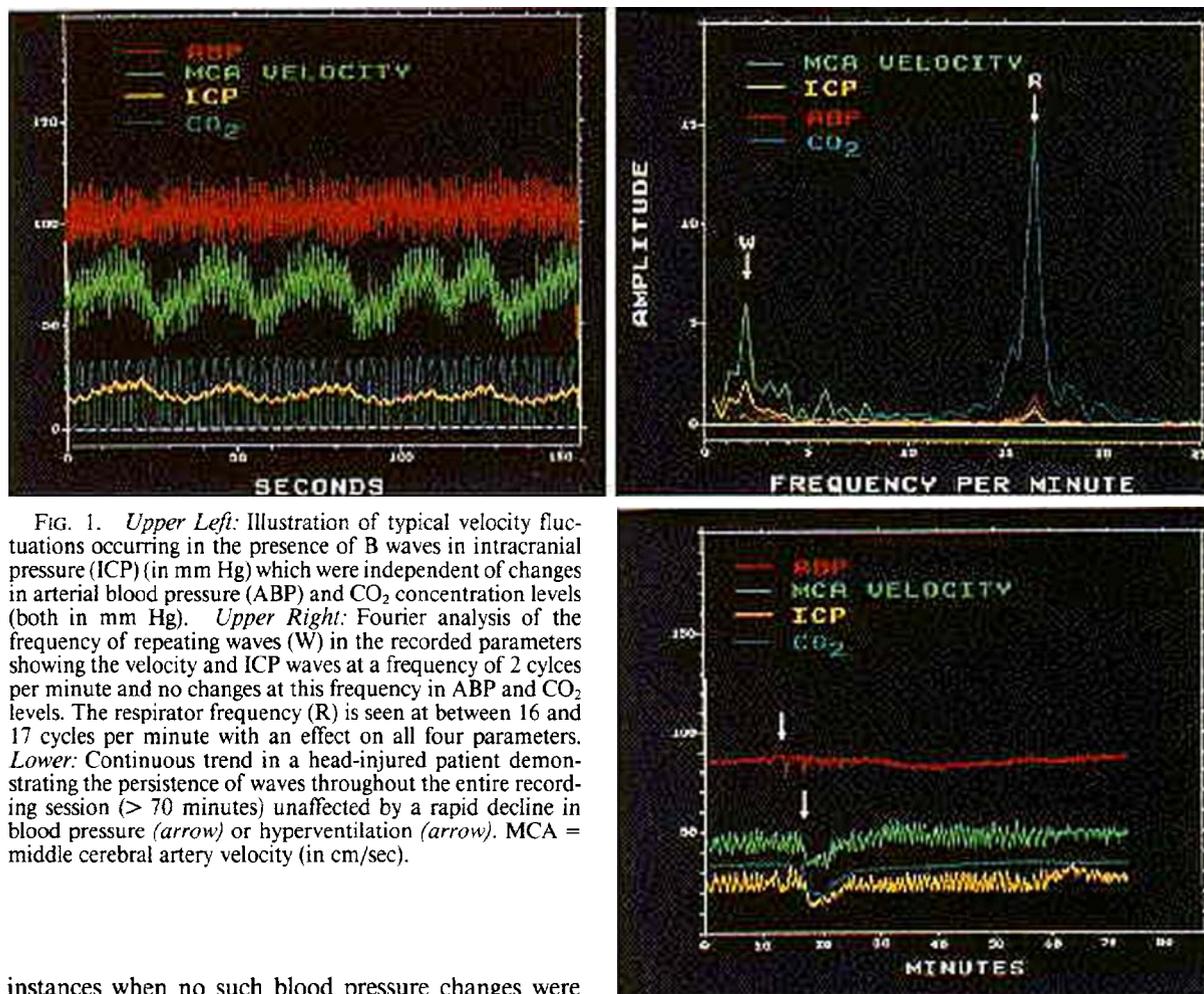


FIG. 1. *Upper Left:* Illustration of typical velocity fluctuations occurring in the presence of B waves in intracranial pressure (ICP) (in mm Hg) which were independent of changes in arterial blood pressure (ABP) and CO₂ concentration levels (both in mm Hg). *Upper Right:* Fourier analysis of the frequency of repeating waves (W) in the recorded parameters showing the velocity and ICP waves at a frequency of 2 cycles per minute and no changes at this frequency in ABP and CO₂ levels. The respirator frequency (R) is seen at between 16 and 17 cycles per minute with an effect on all four parameters. *Lower:* Continuous trend in a head-injured patient demonstrating the persistence of waves throughout the entire recording session (> 70 minutes) unaffected by a rapid decline in blood pressure (arrow) or hyperventilation (arrow). MCA = middle cerebral artery velocity (in cm/sec).

instances when no such blood pressure changes were seen.

The amplitude of the velocity waves varied between patients and also during continuous tracings in the same patient. Since relative changes in the MCA velocity have been shown to correlate with relative changes in volume flow in certain circumstances,¹¹ the amplitude of the waves was calculated as a percentage of the average mean velocity. If there was no significant change in the MCA diameter, variations from this baseline should represent percentage changes in blood flow. The maximum velocity wave amplitude observed in one head-injured patient was 49% of the average mean velocity during the fluctuations. The average velocity wave amplitude for the group analyzed during the maximum wave activity was $23.1\% \pm 8.05\%$ (\pm standard deviation) of the average mean velocity.

The amplitude of the ICP fluctuation in response to a given velocity wave amplitude varied between different monitoring sessions and also during different intervals of the same session, and appeared to be dependent on the ICP. First, to determine whether the amplitude of the velocity waves was affected by the ICP, a regression analysis was performed between these two parameters. This regression analysis revealed an inverse relationship between ICP and velocity wave amplitude

($r = -0.39$, $p < 0.05$) (Fig. 2 left). Second, an exponential regression analysis was performed between the average ICP and the amplitude of the ICP waves, revealing a strong correlation ($r = 0.69$, $p < 0.001$) (Fig. 2 center). The best correlation, however, was found by exponential regression analysis between the ICP and the ratio of amplitudes of the ICP wave to the same velocity wave ($r = 0.81$, $p < 0.001$), as shown in Fig. 2 right. This ratio corrects for the varying strength of the underlying "wave generator," and essentially expresses how the amplitude relationship between classic B waves in the ICP and the velocity waves seen on the TCD recordings is influenced by changing levels of ICP.

One frequently seen characteristic of the velocity waves was that the rate of increase in velocity was less than the rate of decrease (Fig. 3). This characteristic was sometimes but not always reflected in the ICP waves.

A recording was recently obtained that permitted simultaneous velocity recording from both MCA's. This recording was made using a two-channel TCD system from a head-injured patient not included in the origi-

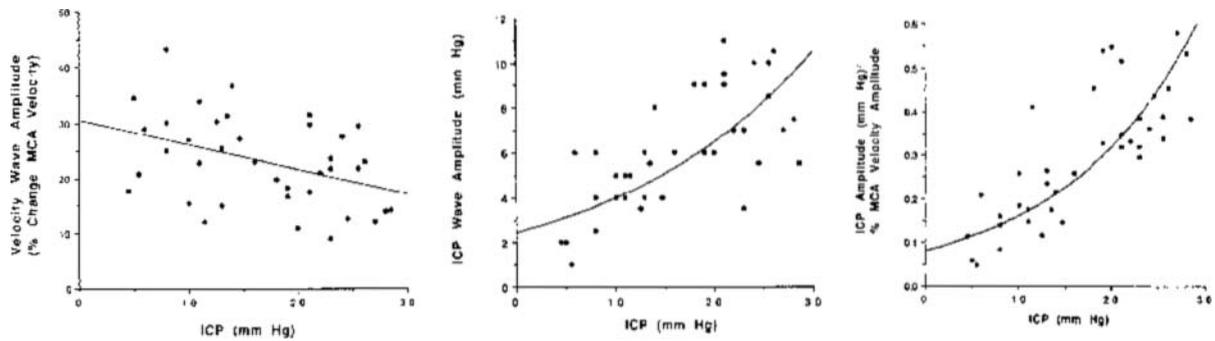


FIG. 2. *Left:* Linear regression analysis between velocity wave amplitude and intracranial pressure (ICP) indicating a slight inverse relationship between the two variables ($r = -0.39$, $p < 0.05$). MCA = middle cerebral artery. *Center:* Exponential regression analysis between ICP wave amplitude and ICP in the presence of B waves, indicating that B waves are more prominent at higher ICP levels ($r = 0.69$, $p < 0.001$). *Right:* Exponential regression analysis between the ICP and the ratio between ICP and velocity wave amplitudes ($r = 0.81$, $p < 0.001$). This graph illustrates that increasing ICP induces a sharp increase in the ICP B wave amplitude relative to the velocity amplitude of the same wave.

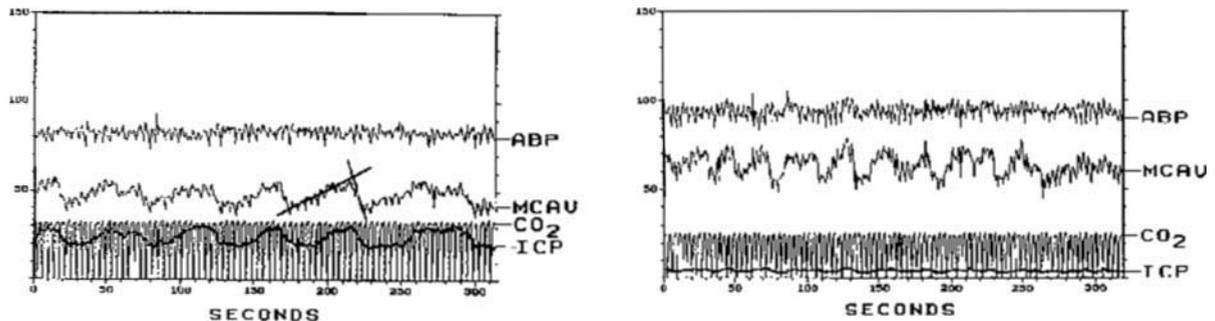


FIG. 3. Comparison of the effect of velocity fluctuations on intracranial pressure (ICP, in mm Hg) fluctuations at different ICP levels. ABP = arterial blood pressure (in mm Hg); CO_2 = CO_2 concentration (in mm Hg); MCAV = middle cerebral artery velocity (in cm/sec). *Left:* Illustration of marked ICP fluctuations at higher ICP levels (mean ICP 24 mm Hg). Lines illustrate the slower rate of increase in the velocity waves. *Right:* Illustration showing minimal ICP fluctuations in response to similar velocity fluctuations at a lower ICP (mean ICP 5 mm Hg).

nal analysis. Velocity fluctuations in both MCA's were completely synchronous with one another and in phase with the ICP B waves (Fig. 4).

Velocity recordings from the extracranial internal carotid artery in five patients during ICP B waves revealed simultaneous velocity fluctuations that were synchronous with the ICP fluctuations (Fig. 5).

Recordings in Normal Volunteers

Review of the monitored tracings of MCA velocity and end-tidal CO_2 revealed brief episodes of MCA velocity fluctuations lasting at least 2 minutes in seven (70%) of the 10 normal volunteers. These waves were similar in character, frequency, and amplitude to those seen in the head-injured patients. However, they tended to occur for shorter durations (Fig. 6). The maximum duration of the waves observed in the control group was 15 minutes, whereas some of the head-injured

patients had waves for the entire recording period. The maximum amplitude in this group was 41% of the average mean velocity versus a maximum of 49% in one head-injured patient. The average velocity wave amplitude for the control group during the maximum wave activity was $29.0\% \pm 7.73\%$ of the average mean velocity. This value was slightly higher than that in the head-injury group but did not reach statistical significance by the Student t-test ($0.05 < p < 0.01$). Small changes in respiratory rate were also noted to occur in some subjects at a frequency similar to that of the waves. As a result, end-tidal CO_2 changed slightly; this change, however, did not appear to be of a sufficient magnitude or duration to be responsible for the change in MCA velocity. For example, in the volunteer with the maximum amplitude velocity changes, the end-tidal CO_2 fluctuated briefly by 5 mm Hg during the wave and only for a period of 10 to 12 seconds. During the

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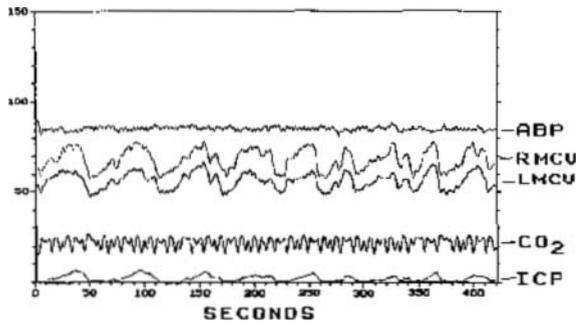


FIG. 4. Illustration of simultaneous velocity recording from both middle cerebral arteries in the presence of B waves using a two-channel Doppler ultrasound device. The velocity fluctuations are synchronous with each other and in phase with the intracranial pressure (ICP) B waves. ABP = arterial blood pressure; RMCV = right middle cerebral artery velocity; LMCV = left middle cerebral artery velocity.

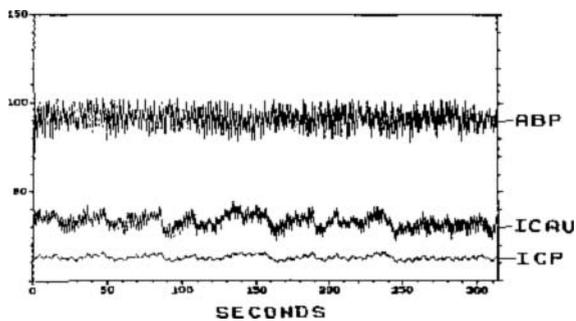


FIG. 5. Illustration of simultaneous recording from the neck of arterial blood pressure (ABP, in mm Hg), intracranial pressure (ICP, in mm Hg), and extracranial internal carotid artery velocity (ICAV, in cm/sec) in the presence of B waves, strongly indicating that velocity fluctuations occur due to volume flow fluctuations.

same wave, which lasted 40 seconds, the MCA velocity changed by 41%; this represents an 8% change in MCA velocity per mm of CO₂ change.

Discussion

Theoretical Aspects of TCD Monitoring

Monitoring of the blood flow velocity of the MCA, both intraoperatively and in the intensive care unit, has been reported previously.^{8,12} For continuous recording, the TCD velocity signal can be taken from the outline of the velocity spectrum, which is normally calculated by the TCD instrument. This tracing corresponds to the V_{max} of blood flow at the center of the MCA.¹ During laminar flow, which is the normal condition in the basal arteries, the flow velocity at the center of the MCA will be directly proportional to the average cross-sectional velocity. Since blood flow is a product of the average velocity and the cross-sectional area of the vessel, changes in velocity directly reflect changes in blood flow through this conducting artery, provided that the diameter does not change significantly³ and

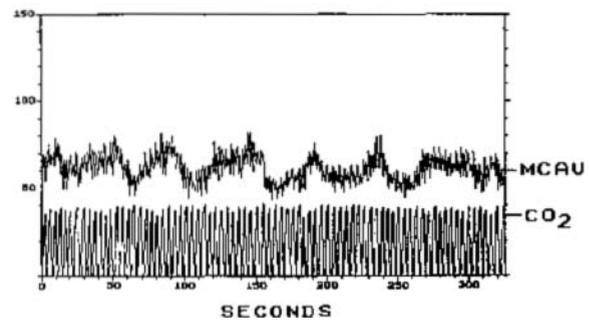
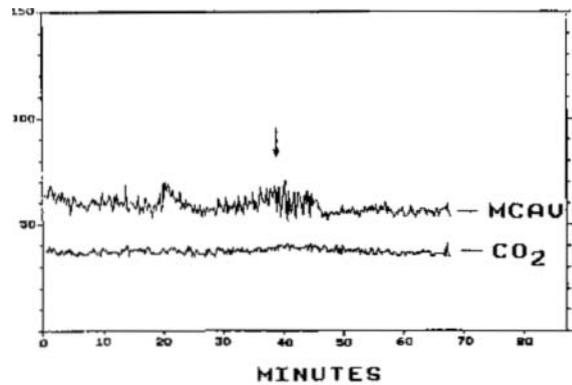


FIG. 6. *Upper:* Continuous recording of middle cerebral artery velocity (MCAV, in cm/sec) and CO₂ (in mm Hg) in a normal subject demonstrating a brief episode of velocity fluctuations (*arrow*) similar in frequency and character to those seen in the presence of B waves in the intracranial pressure of head-injured patients. *Lower:* Enlarged detail of this interval illustrating small fluctuations in end-tidal CO₂ during the waves; however, these fluctuations appeared to be insufficient to be causative.

that the probe angle does not change. By employing a monitoring probe fixed with a headband, variations in probe angle are eliminated. Previous experiments have demonstrated a linear correlation between V_{max} and volume flow variations in the internal carotid artery, measured by an electromagnetic flowmeter during carotid endarterectomy.¹¹ This finding indicates that the MCA diameter did not change significantly and, therefore, changes in V_{max} directly reflected relative changes in blood flow. Additional studies confirm a strong correlation between V_{max} and relative blood flow changes during blood pressure fluctuations³ and changes in CO₂ concentration.⁵

Theoretically, fluctuations in MCA velocity such as those seen in this study could either be due to phasic MCA diameter changes or to phasic blood flow variations. Two findings in this study support the concept that the V_{max} fluctuations are due primarily to blood flow variations and not to phasic vessel diameter changes; they are: 1) that velocity fluctuations similar to those in the MCA were found in the extracranial internal carotid artery and occurred simultaneously with ICP fluctuations; and 2) that the ICP fluctuations were in phase with the velocity fluctuations. If increases

in velocity were due to rhythmic contractions of the MCA, then a decrease would be expected in the ICP, due to vasoconstriction at the peak velocity rather than the observed increases in ICP.

Current Findings

The finding of periodic fluctuations in the MCA velocity can be explained most easily by fluctuations in the CBF; these are caused by a phasic dilation and contraction of the small regulating arteries (vasomotor waves). These vasomotor waves produce fluctuations in cerebral blood volume, which are eventually reflected in the ICP. This interpretation is supported by experiments reported by Auer and Sayama⁴ who, with the use of a pial window preparation in cats, observed pial vessels oscillating in synchrony with ICP variations using a pial window preparation in cats. They clearly demonstrated that increases in ICP occurred when the pial arteries dilated. These fluctuations were noted at a frequency between 0.5 and 2 cycles per minute and thus correspond to the present findings and previous descriptions of B waves in the ICP.¹³

In his original description of B waves in ICP tracings, Lundberg¹³ frequently observed respiratory variations synchronous with the wave frequency. He therefore suggested that the B waves could be caused by fluctuations in the CO₂ concentration, causing variations in CBF. However, B waves were observed in some artificially ventilated patients. Lundberg noted that "These observations permit no definite conclusion as to the cause of the B waves."¹³ The concentration of CO₂ was not monitored during the recordings. Other investigators have recorded respiratory rate simultaneously with ICP in nonventilated neurosurgical patients and found a relationship between respiratory variations and B waves.⁹

Our data demonstrate conclusively that velocity waves and B waves of the ICP can occur in ventilated patients with a constant CO₂ concentration. Fourier analysis in selected cases shows that the MCA velocity oscillations are clearly in synchrony with the ICP oscillations and are not necessarily accompanied by oscillations in arterial blood pressure or CO₂. Some cases, however, demonstrated minor fluctuations in arterial blood pressure that occurred regularly at the wave frequency and may be a secondary effect or an accompanying phenomenon. During the peak of the velocity waves in the normal subjects, there was often minor slowing of the respiratory rate, slightly effecting the end-tidal CO₂. However, the change in CO₂ concentration was not sufficient to cause the change seen in the velocity.¹⁵ Therefore, it is unlikely that changes in CO₂ concentration are a primary cause of the velocity changes; it appears more likely that respiratory changes may be an accompanying phenomenon.

Etiology of Blood Flow Fluctuations and B Waves

The exact cause of the fluctuations in CBF resulting in B waves is unknown. Some investigators have sug-

gested that a mechanism intrinsic to the arterial smooth-muscle cells in the regulating arteries is responsible.⁴ Others have implicated an intrinsic brain-stem pacemaker that alters other physiological parameters as well as CBF at a regular frequency.^{10,14} The finding of synchronous fluctuations of blood flow in both MCA's suggests the presence of a central control mechanism or a synchronizing mechanism. It was commonly observed (Figs. 3 and 4) that the velocity wave would begin before the change in ICP took place and thus was independent of any change in transmural or cerebral perfusion pressure.

The inverse relationship found between ICP and velocity wave amplitude was initially unexpected, but may be explained by the very large amplitudes of the ICP waves at higher ICP's. The cerebral perfusion pressure is much lower at the peak of the ICP wave than in the valley. If autoregulation is poor (or slow), the flow (and velocity) amplitude will then necessarily be attenuated at the peak, compared to a situation where perfusion pressure does not fluctuate. Many of our patients had poor autoregulation (unpublished data).

The finding of a variable response in the ICP for a given degree of fluctuation in CBF velocity can be most easily explained by variations in intracranial compliance at different ICP levels. Although intracranial compliance was not measured in these patients, it is generally lower in patients with elevated ICP.¹⁶ Thus, a given amount of cerebral blood volume fluctuation would produce a higher amplitude of the ICP wave with reduced intracranial compliance. Our results illustrated by Fig. 2 *lower* support this interpretation. Dirnagl, *et al.*,⁷ demonstrated a clear inverse relationship between intracranial compliance measured by the pressure volume index and the ICP amplitude of B waves in neurosurgical patients. There was, however, no correlation between intracranial compliance and the duration of B wave activity. Other studies have also confirmed that B waves in the ICP are prominent in conditions associated with reduced intracranial compliance.^{6,19}

The present data, supported by previous observations,⁴ suggest that phasic variations in CBF can occur at a frequency of 0.5 to 2 cycles per minute under normal conditions as well as in pathological states. The relationship between various pathological states and the magnitude and duration of these blood flow fluctuations is unclear. If ICP is monitored during these fluctuations, the magnitude of ICP variations will be influenced by the degree of CBF fluctuation and by the intracranial compliance during monitoring. It is likely that B waves of the ICP have been recognized as an amplification of a physiological phenomenon under conditions of reduced intracranial compliance.

Implications for TCD Examination

One implication of the current findings concerns the effect of MCA fluctuations on values recorded during TCD diagnostic examination. The finding of significant fluctuations of velocity in normal subjects indicates

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that this phenomenon could be a source of variability in the velocity values and should be kept in mind when performing examinations. To record the most accurate mean velocity during appearance of these waves, the timed average mean velocity value between the extremes of the fluctuations should be noted.

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