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B-waves revisited

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Abstract: Reduced intracranial compliance is a key manifestation common to a number of pathological conditions of the brain. It is encountered in, but not limited to, traumatic brain injury, cerebral edema, and hydrocephalus. There are no clinically accepted methods to measure intracranial compliance available to date. Intracranial pressure (ICP) waveform analysis is seeing a revival driven by advances in our understanding of cerebrospinal fluid and pressure dynamics. Its translation to widespread clinical use is dependent on the possibility to derive relevant metrics such as intracranial compliance reliably and non-invasively. The B-wave is one of the features of the ICP waveform, reflecting vasogenic activity of cerebral autoregulation. B-waves were originally defined to occupy the 0.5 to 2 cycles per minute frequency range. Recently renamed and redefined as slow waves with an extended range of 0.33 to 3 cycles per minute, specific changes in their pattern of occurrence are considered to be indicative of reduced intracranial compliance. With the still unmet need for a clinically acceptable method for acquiring intracranial compliance, and the revival of ICP waveform analysis, B-waves are moving back into the research focus. Herein we provide a concise review of the literature on B-waves, including a critical assessment of non-invasive methods for obtaining B-wave surrogates.

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Neuroanatomical Study

B-waves revisited

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ABSTRACT

Reduced intracranial compliance is a key manifestation common to a number of pathological conditions of the brain. It is encountered in, but not limited to, traumatic brain injury, cerebral edema, and hydrocephalus. There are no clinically accepted methods to measure intracranial compliance available to date.
Intracranial pressure (ICP) waveform analysis is seeing a revival driven by advances in our understanding of cerebrospinal fluid and pressure dynamics. Its translation to widespread clinical use is dependent on the possibility to derive relevant metrics such as intracranial compliance reliably and non-invasively.
The B-wave is one of the features of the ICP waveform, reflecting vasogenic activity of cerebral autoregulation. B-waves were originally defined to occupy the 0.5 to 2 cycles per minute frequency range. Recently renamed and redefined as slow waves with an extended range of 0.33 to 3 cycles per minute, specific changes in their pattern of occurrence are considered to be indicative of reduced intracranial compliance.
With the still unmet need for a clinically acceptable method for acquiring intracranial compliance, and the revival of ICP waveform analysis, B-waves are moving back into the research focus. Herein we provide a concise review of the literature on B-waves, including a critical assessment of non-invasive methods for obtaining B-wave surrogates.

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1. Introduction

The term *B-wave* was introduced by Nils Lundberg in 1959 [1], originally referring to repeating elements in the intracranial pressure (ICP) signal with frequencies of 0.5 to 2 cycles per minute. Lundberg viewed B-waves as a pathological phenomenon in patients with elevated ICP. Over the years, both the definition of these waves and their interpretation have changed, with frequency windows of 0.5 to 3 [2–4], 0.33 to 2 [5] and 1 to 2 cycles per minute [6,7] having been considered to contain B-waves. Czosnyka and co-workers introduced new terms, *slow waves* [8] and *slow vasogenic waves* [9–11], with a window of 0.33 to 3 cycles per minute. For the sake of simplicity, we will use herein solely the term *B-wave* with an associated frequency range of 0.33 to 3 cycles per minute.

While B-waves are, per definition, found in the ICP signal, other measurable quantities that may be influenced by the dynamics of intracranial pressure or that have a common source of influence with ICP can also show fluctuations in the same frequency range. We will refer to these as *B-wave surrogates*.

In this paper we provide an overview of the literature on B-waves and B-wave surrogates, starting with the characterization of proper B-waves in the upcoming section, followed by a treatise of their surrogates before analyzing the diagnostic value of both in Section 4.

2. What are B-waves?

2.1. Classification of B-waves

B-waves have been classified according to their waveform into subgroups of sinusoidal and ramp-shaped B-waves [3,4,12,6,13] using some slightly different nomenclatures describing the same phenomena. We will use herein the terms *sine-type* and *ramp-type B-waves*, where Fig. 1 shows an example of the former and Fig. 2 of the latter type. Furthermore, B-waves are referred to as *low B-waves* if their peak-to-peak amplitude is below 10 mm Hg, and *high B-waves* if it is above [12].

Sine-type B-waves are attributed to autoregulatory reactions to arterial blood pressure (ABP) changes, not associated with changes in respiratory rhythm or arterial CO_2, seen more during sleep, and also seen in ventilated patients. Ramp-type B-waves are described to be associated with changes in respiratory rhythm, rises in CO_2, rises in ICP-amplitude and seen in snoring [3,4,12,13].

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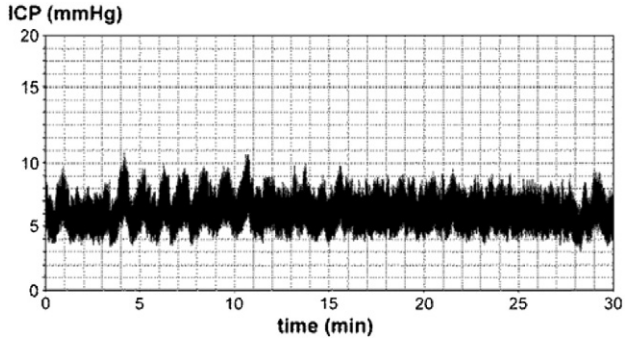

Fig. 1. Sine-type low B-waves in the ICP signal of a patient with severe Hakim triad and idiopathic normal pressure hydrocephalus (iNPH). Overnight ICP monitoring resulted in maximum ICP of 15 mm Hg and median value of 6 mm Hg. Lumbar infusion test was negative (cerebrospinal fluid [CSF] outflow resistance R_{out} of 4 mm Hg/ml/min). Lumbar drainage resulted in dramatic improvement of the patient. After shunt placement, the patient continued to recover substantially for 6 months, followed by decline to severe dementia and care dependence 24 months after surgery.

Raftopoulos defined *great symmetrical waves* and *intermediate waves* as subgroups of B-waves [14]. Further subgroups were defined by Diehl and Berlit [15]: A) High-amplitude uniform waves, discernible by visual inspection, found in normal subjects in 10% to 70% of the time monitored, and B) low-amplitude waves with changing period, recognizable only by frequency analysis.

2.2. B-wave surrogates

B-wave surrogates are oscillations of signals associated with, but different from ICP within the same frequency range as proper B-waves. Of particular interest are signals that can be measured non-invasively. B-wave surrogates have been found and researched in transcranial Doppler (TCD) and near infrared spectroscopy (NIRS) signals.

Fluctuations of arterial blood flow velocities measured with TCD have been reported to occupy the same frequency as and occur simultaneously with B-waves [16–21]. This TCD B-wave surrogate and the corresponding B-waves are described to be in phase: a rise in ICP coincides with a rise in blood flow velocity. Newell et al. [19] interpreted the measured fluctuation in TCD flow velocity as a corresponding change in blood flow rate by assuming that the diameter of the insonated artery remains constant. TCD B-wave surrogates do not only occur under pathologic conditions, but have also been observed in healthy subjects [18,19,16,17,22,4].

Näsi [23], Virtanen [24] and Weerakkody [10] investigated NIRS signals in relationship to sleep stage and ICP. They found that slow spontaneous hemodynamic activity including waves in the B-wave range

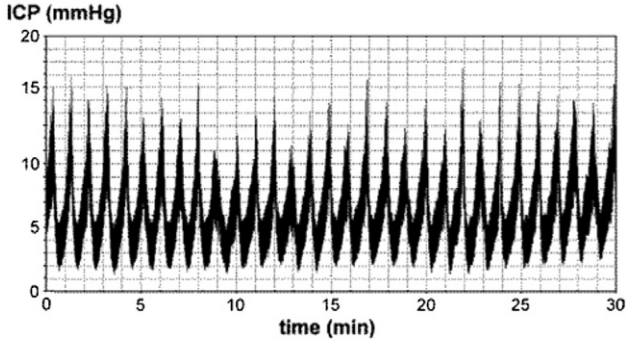

Fig. 2. Ramp-type high B-waves in the ICP signal of a patient with iNPH suffering from severe gait disturbance and presenting with complete Hakim triad. Overnight ICP monitoring revealed plateau ICP values of 20 mm Hg and median ICP of 11 mm Hg. Lumbar infusion test revealed R_{out} of 11 mm Hg/ml/min. The patient became symptom-free after shunt placement and has remained without symptoms since (follow-up of 4 years).

measured by NIRS was greatly reduced in Slow Wave Sleep (deep sleep, SWS) compared to light sleep and REM sleep, and it was found to occur, regardless of compliance, also in healthy individuals.

Non-invasive calculation of ICP from TCD blood flow velocity data was shown in [25–27]. We conclude that features of the ICP waveform, including B-waves, should theoretically also be derivable from calculated (rather than measured) ICP signals.

2.3. Origin of B-waves

ICP dynamics are largely governed by cardiovascular and respiratory action. Early research focused on the phase relationship between waves in ABP and ICP, aiming to determine whether waves in the ICP signal precede or follow the corresponding ABP waves [2,28–30] [20,31,29]. Later, the Pressure Reactivity Index (PRx) was introduced as a measure of this phase relationship. Czosnyka et al. [32] report that *a positive PRx indicates a positive gradient of the regression line between the slow components of ABP and ICP, which has been shown to be associated with passive behaviour of a non-reactive vascular bed. A negative value of PRx reflects normal reactive cerebral vessels, as ABP waves provoke inversely correlated waves in ICP due to vasoconstriction.*

While B-waves had originally been considered a pathologic phenomenon caused by rhythmic changes in arterial CO_2 concentration leading to oscillations in blood vessel volume [1], they were later also observed in healthy individuals [33] [16,17,22] as were B-wave surrogates [18,19,16,17,22,4]. Since sine-type B-waves are also seen in artificially ventilated patients with constant CO_2 [3], the hypothesis of rhythmic variation of arterial CO_2 concentration as the source of B-waves cannot be universally advocated. At the same time, it is known that ramp-type B-waves are associated with changes in arterial CO_2 [3,12]. This indicates that there is more than one physiological or pathophysiological mechanism underlying the occurrence of B-waves. Thus, differentiation between B-wave subgroups appears to be necessary for identification of possible mechanisms and for the use of B-waves for diagnostics purposes.

Rosner postulated in one of his fundamental papers on the vasodilatory cascade [31] that B-waves and A-waves are generated by the same mechanism. A-waves were first described as *large plateau-like waves, recurring at intervals of varying length* (*usual ranges*: *height 50–100 mm Hg, duration 5–20 min*) [1]. After initial observations in animals [34] followed by examinations in a mixed population of 22 patients (mechanical brain injury, neoplasm, metabolic encephalopathy and hydrocephalus), Rosner hypothesized that in the *vasodilatory cascade* a reduction of cerebral perfusion pressure (CPP) caused by a spontaneous drop of ABP is answered by vasodilation when autoregulation is intact. Vasodilation then results in an increase in cerebral blood volume (CBV) and a rise in ICP. The Cushing response, i.e. an increase in ABP in response to high ICP [35], brings the ABP back up and terminates the pressure wave. He stated in [31] that the "*B*" *waves and* "*plateau*" *waves were a function of unstable SABP* (systemic ABP). *There were no qualitative differences observed*: *quantitative differences accounted for all variation.* "*B*" *waves were associated with faster, usually greater SABP decrements* […]. *Plateau waves were associated with a delayed ischemic response and lower CPP.*

Gaab also suggested changes in CBV as the origin of both sine-type and ramp-type B-waves [3], with breathing-rhythm dependent variation of arterial CO_2 as the cause of the latter, and brain stem controlled changes in cerebral blood volume inducing the former. He described that in the absence of blood gas oscillations, B-waves occur without variation in ABP. This is in contrast to Brock who stated that sine-type B-waves are *by definition associated* with rhythmic respiratory variations [6]. Brock further stated that ramp-type B-waves are frequently observed in patients with *normotensive hydrocephalus*.

In a further contribution explaining the origin of B-waves, Auer and Sayama demonstrated in a cat model that B-waves can be provoked by artificially elevating ICP [36]. They showed, through simultaneous

recording of ICP and the diameter of small pial arteries, that B-waves coincide with pulsations of the arterial diameter that are synchronous with the B-waves. It had been established earlier that small arteries (smaller than 2.5 mm diameter) change their diameter by up to 25% under induced hypo- and hypercapnic conditions, whereas larger arteries maintain their size [37]. Sörensen et al. [7] observed that the occurrence of B-waves in normal pressure hydrocephalus (NPH) patients is inversely correlated to the sum of outflow conductance and craniospinal compliance, concluding that both low compliance and low outflow conductance are factors, but not the only factors, in the etiology of B-waves.

Venes [38] reported that according to his experience in children with Reye Johnson syndrome, *B-waves reflect changes in cerebral blood volume and/or flow, probably secondary to alterations in cerebrovascular resistance. Their occurrence independent of changes in pCO_2, blood pressure, respiration, central venous or airway pressure suggests an intrinsic neural mechanism.*

Contributing to the explanation of the generation of the different types of B-waves, Einhäupl et al. [13] suggested that sine-type B-waves and ramp-type B-waves, accompanied or not accompanied by Cheyne Stokes Respiration (periodic breathing, CSR), are *based on the same phenomenon*, namely *secondary phenomena to primary CNS-rhythms*. Hashimoto et al. [39] suggested that B-wave oscillations are derived from rhythmic cerebral vasoconstriction caused by the intrinsic brain stem rhythm, and Higashi [40] suggested a common generator of both B-waves and CSR.

A possible confirmation of a central nervous system (CNS)-rhythm as a contributor to the generation of B-waves was given by Lang et al. [2] who reported no reduction in TCD B-wave surrogate activity in groups of patients after traumatic brain injury (TBI), subarachnoid hemorrhage (SAH), and intracerebral hemorrhage (ICH). This was in contrast to a significant reduction of activity in the B-wave range of peripheral laser Doppler flowmetry (LDF) and in ABP vasomotion, indicating that the pathway for the control of cerebral vasculature is different from that for the control of the peripheral vasculature. They *concluded that this finding supports the theory of a distinct functional organization of brain stem mediated vascular control*. This may indicate *a different pathophysiological condition in TBI patients.* The fact that slow vasogenic waves were described to vanish under general anesthesia [11] may suggest an influence of the anesthetic on the mentioned brain stem rhythm.

Greitz [41] and Ågren-Wilsson [42] offered pathophysiological explanations for possible damage done by pressure waves and pressure peaks. Greitz suggested that *chronic hydrocephalus is due to decreased intracranial compliance, causing restricted arterial pulsations and increased capillary pulsations.* Furthermore: *Of outmost importance is the abnormal pressure and volume transmission into the brain capillaries, which increases ventricular pulse pressure, increases the pulsatile CSF flow in the aqueduct and dilates the ventricles.* Ågren-Wilsson stated that *theoretically, frequent ICP peaks over a long time could eventually cause axonal dysfunction and, eventually, persisting damage.*

In summary, B-waves can be considered a manifestation of the transmission of rhythmic changes in cerebral blood volume to ICP. The transmission factor or amplification is influenced by the pressure-volume relationship (compliance) in the cranial vault. A brain-stem rhythm, rhythmic changes of arterial CO_2 partial pressure, the time constant of autoregulation, the speed of ABP reduction, and the value of CPP have all been mentioned as possible factors influencing the generation, amplitude and frequency of B-waves.

2.4. B-wave detection and quantification

There is no general agreement on the quantitative description of B-waves beyond the recognition that they occupy a certain frequency spectrum of the ICP signal. B-wave quantification depends strongly on the way B-waves are detected. Traditionally, B-waves were identified and quantified by visual inspection.

First published reports on computerized methods for the detection and quantification of B-waves were given by Müller and co-workers [22,43] who mention the implementation of such an algorithm in a commercial device (Neurolab ICP Analyzer, production discontinued). According to Biomath GmbH, the current rights holder, the algorithm evaluates statistical models in real-time, recognizing artifacts and quantifying B-waves, respiratory waves and pulse waves by activity indices.

Eklund and co-workers [44] described two computerized methods to analyze B-waves. The first one identifies individual B-waves in the time domain based on waveform, while the second relies on the power calculated in the frequency spectrum of 10 minute segments of ICP signals. Both methods were found to give results similar to the traditional visual method, with the first one having a higher correlation.

Kasprowicz et al. [45] reported an algorithm that detects B-waves indirectly by recognizing therewith correlated features of the ICP signal through waveform analysis. The corresponding metrics are derived from the peaks of the percussion, tidal and dicrotic waves.

Maldonado [46] introduced two algorithms for B-wave detection, one based on frequency and amplitude, the other on ICP waveform analysis. He compared both methods to the one described in [45], with the method based on frequency and amplitude giving a closer match.

Further quantitative metrics that have been used are the time fraction of B-wave activity [1,47,44,8], and the *Coefficient of Variance* [2,48]. In the former, the time fraction of B-wave activity is determined by dividing the cumulative duration of B-waves by the total ICP monitoring time. B-waves are considered to occur when their amplitudes exceed a threshold pressure (e.g. 1 mm Hg as suggested in [44]). The latter, not to be confused with coefficient of variation, was defined by Diehl et al. [48] as the standard deviation of the bandpass-filtered ICP signal divided by the mean value of the ICP signal before filtering, with the bandpass filter having the limits of the B-wave range as its cut-off frequencies.

Finally, an index termed *SLOW* for the activity in the slow wave range is calculated by the widely used software ICM + (University of Cambridge Enterprise, Cambridge, UK). This index is determined using a function that estimates power spectral density based on a periodogram method within a user specified data window [49].

2.5. B-waves and sleep

The prevalence of B-waves is increased during sleep and correlates with different stages of sleep, with a maximum occurring during REM phases [16,50,32,51]. Nilsson et al. [52] and Stephenson et al. [53] offered a possible explanation by pointing out that increased cerebral blood flow during REM phases occurs on top of increased CSF production at night, which may result in reduced compliance. At the same time, there are fewer artifacts in the ICP-signal during sleep. Together, these aspects would result in higher prevalence and better detectability of B-waves. It should be noted that in light of recent findings about the origin and circulation of CSF [54], a re-evaluation of circadian changes in CSF production and absorption is warranted.

In conclusion, in studies and evaluations of B-waves, the subjects' sleep stages should be taken into account. Patients being observed overnight, for example, might show low B-wave activity because they may not be able to enter REM phase due to disturbed sleep.

3. Diagnostic value of B-waves and B-wave surrogates

B-waves may carry information on brain-stem function, pressure-volume compensation, and cerebral autoregulation. Potential diagnostic value should thus primarily be sought in conditions where changes in these are expected: Hydrocephalus and Traumatic Brain Injury.

3.1. Hydrocephalus

Based on the correlation of B-waves and compensatory parameters (i.e. compliance, outflow resistance, ICP-pulse amplitude, slope of amplitude vs. ICP), a correlation between shunt responsiveness and the occurrence of B-waves was shown in [14,55–62], with [57] specifically addressing…not *typical B-waves*, but *rampwaves*…, [14] addressing great symmetrical waves, and [62] taking into account B-waves, A-waves and baseline ICP. However, other studies showed poor or no correlation [63,53,64].

Next to proper B-waves, TCD B-wave surrogates have also been evaluated as predictors for shunt responsiveness [17,65]. However, due to the wide variability of the amplitude of TCD B-wave surrogates and due to the fact that these surrogates showed high prevalence in healthy individuals, Droste et al. [17] concluded that *the mere presence of B-wave activity over up to 73% of the time is probably not a good indicator for the diagnosis of NPH.*

3.2. Coma associated with traumatic brain injury, subarachnoid hemorrhage (SAH) and intracerebral hemorrhage (ICH)

Impaired autoregulation is a predictor of bad outcome in coma patients after TBI, SAH, and ICH. It has been shown that the occurrence of B-waves correlates with good outcome in these patient groups [66, 22]. Therefore, it is plausible that the vanishing of B-waves is caused by impaired autoregulation, which would be in agreement with Rosner's hypothesis of B-wave generation: if a B-wave is initiated by the autoregulatory response to a spontaneous drop in ABP (rising ICP) with Cushing's response (rising ABP) terminating the wave, this cycle would not be possible with impaired autoregulation.

4. Conclusions

There is agreement on B-waves being indicative of reduced intracranial compliance, on their dependence on a central rhythm, and on the presence of B-waves as an indicator for intact autoregulation. It is not clear whether these points apply to both sine-type and ramp-type B-waves.

There is no agreement on the value of B-waves for the diagnosis of hydrocephalus and as a predictor of shunt responsiveness. However, B-waves are used routinely at some institutions for these purposes.

Vanishing of B-waves in TBI patients as a sign of impaired autoregulation and thus as a predictor of bad outcome appears to be widely accepted, but not generally used. With PRx as an established parameter for autoregulation, there does not appear to be a need for another parameter with less well defined metrics.

The fact that slow spontaneous hemodynamic activity measured by NIRS is greatly reduced in SWS, regardless of compliance, also in healthy individuals, suggests that B-wave surrogates in the NIRS signal may not be useful for diagnostic purposes. In contrast, B-wave surrogates in the TCD signal bear, in our opinion, potential for diagnostic value and thus warrant further investigations.

Conflict of interest statement

We have no conflicts of interest associated with this work.

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