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CORRESPONDENCE

Intracranial pressure pulse morphology: the missing link?

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Hawryluk et al. made a comprehensive report on the intracranial pressure (ICP) knowledge historical evolution, and its physiological aspects and rationale for establishing the ranges of safety and thresholds for therapeutic interventions when intracranial hypertension overcomes (IHT) [1]. However, these authors stated that the ideal ICP value to trigger dedicated therapy is still not known with certainty. Accordingly, Wijdicks in another recent seminal publication stated: "the use of numeric thresholds does not tell the full picture of changes in autoregulation and cellular dysfunction. We have to come to realize that refractoriness of increased ICP is more important than ICP values" [2].

The key for patient individualization regarding ICP monitoring may be harbored by ICP pulse morphology. Recently, Nucci et al. observed the ICP waveform (ICPW) changes according to variations in intracranial volume [3] (Fig. 1). When the tidal peak (P2) acquires an amplitude higher than the upstroke peak (P1) it is known that the intracranial space has lost its compensatory reserve, with impairment in intracranial compliance (ICC) [3].

Contrary to what was stated by Hawryluk et al. citing a study from 1988, that the ICP pulse morphology is imprecise to reflect ICC, two recent studies have been published on the correlation of ICPW variations against ICP mean values. Brasil et al., assessing the slopes from 41 acute brain injured patients, mainly traumatic, found that, for patients with undamaged skull who underwent exclusively a procedure for installing an ICP monitor, P2 amplitudes 20% higher than P1 amplitudes were correlated with ICP > 20 mmHg [$r = 0.72$, area under curve (AUC) 0.9, $p < 0.001$] [4]. The authors observed a dissociation between ICP pulse morphology and mean values in the early days after neurosurgical procedures for IHT alleviation, especially decompressive craniectomy, since P2 amplitudes remained significantly elevated despite the drop in ICP mean values after surgery, and hypothesized that ICC remained impaired in this population. De Moraes et al. assessed 18 non-traumatic acute brain injured patients, mainly after subarachnoid hemorrhage (SAH) but with no neurosurgical manipulations [5]. In their study, sustained P2 amplitude higher than P1 was correlated with IHT ($r = 0.75$, AUC 0.78, $p < 0.001$), whereas patients with ICP < 20 mmHg presented with transitory elevations of P2. On the other hand, P2 amplitudes under P1 were always observed among patients without IHT.

The most important limitation considering ICP pulse morphology for practice standards is lacking of automated analysis, leading to rely on subjective interpretation of the slopes depicted on the screens of dedicated devices, often of poor resolution, precluding also the possibility for recognizing in which direction the peaks amplitudes are moving following the interventions applied.

In conclusion, parameters derived from the slopes of ICP pulse morphology may open avenues for therapy individualization and may be applied besides ICP mean values, potentially indicating for each particular patient the ICP threshold that has become plausibly noxious. Challenges remain to become this information widely accessible, whereas next studies can prove whether ICPW plus ICP values for the treatment of acute brain injured patients may be more valuable than periodically updating guidelines switching from one ICP number to another.

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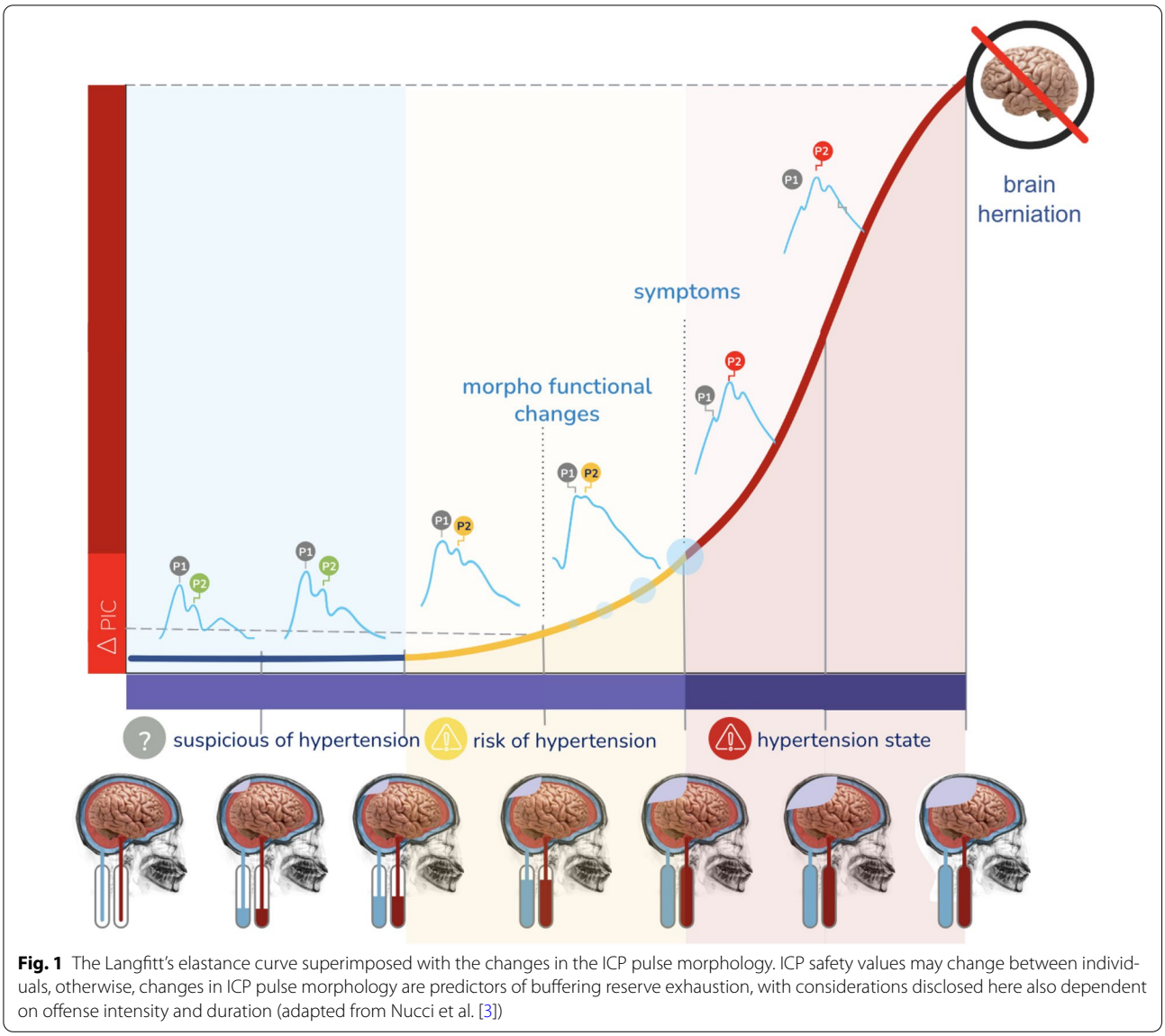

Fig. 1 The Langfitt's elastance curve superimposed with the changes in the ICP pulse morphology. ICP safety values may change between individuals, otherwise, changes in ICP pulse morphology are predictors of buffering reserve exhaustion, with considerations disclosed here also dependent on offense intensity and duration (adapted from Nucci et al. [3])

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SB is member of B4C scientific committee.

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