Is Continuous Intraoperative Monitoring of Mean Arterial Pressure as Good as the Hypotension Prediction Index Algorithm?

To the Editor:

The Hypotension Prediction Index is a validated machine learning algorithm based on numerous characteristics of the arterial waveform and aimed at predicting hypotension up to 15 min in advance.[1] The recent special article by Enevoldsen and Vistisen focused on the data selection process for the development of this algorithm, highlighting the overrepresentation of the mean arterial pressure (MAP) within the Hypotension Prediction Index as substantiated with simulated data.[2] This led us to perform a detailed comparison between the two signals in clinical practice, and investigate the relationship between Hypotension Predicting Index alarms and different simultaneously assessed MAP alarms, to further clarify the presumed added value of the Hypotension Prediction Index over MAP.

In an observational pilot study, the Hypotension Prediction Index was used in addition to routine monitoring in the operating room on a convenience sample of 33 adult, high-risk noncardiac surgery patients requiring invasive blood pressure monitoring (local ethical committee waived approval [#K22-42], Medisch Spectrum Twente, Enschede, The Netherlands). Hemodynamic management was performed according to standard care, with nonblinded addition of the HemoSphere advanced monitor (version 2.1, Edwards Lifesciences, Inc., USA). The Hypotension Prediction Index and averaged MAP values were recorded every 20 s from start of incision until end of surgery, and no additional selection or filtering was applied.

A cross-correlation analysis was performed between the normalized Hypotension Prediction Index and MAP signals. This signal analysis technique quantifies the level of similarity between two time series as a function of the time displacement relative to each other. This primary analysis was performed on each individual patient, and the group-level descriptive statistics are reported here (mean ± SD, when normally distributed, or median [25th to 75th interquartile ranges] otherwise). Second, we focused on the association between Hypotension Prediction Index alarms and different concurrent MAP alarms.

The study population comprised 15 (45%) men, 18 (55%) American Society of Anesthesiologists (ASA; Schaumburg, Illinois) Physical Status II, and 15 (45%) ASA III patients. A total of 11 (33%) had previous cardiovascular disease, 18 (55%) hypertension, 6 (18%) diabetes, and 3 (9%) chronic obstructive pulmonary disease, with an age of 71 [53 to 76] yr. A total of 8,025.7 min of data were analyzed, with a surgery duration per patient of 244.2 ± 109.6 min. At least one Hypotension Prediction Index alarm (default threshold of 85 or greater) was detected in 28 (85%) patients, with a total alarm duration of 25.8 [8.1 to 41.3] min for this subgroup. Hypotension, defined as MAP less than 65 mmHg for at least 1 min, was recorded in 18 (55%) patients, with a total duration of 7.7 [4.2 to 17.8] min for this subgroup.

The MAP and Hypotension Prediction Index signals show an inverted trend, *i.e.*, when MAP decreases, the Hypotension Prediction Index value increases, predicting a low blood pressure (example in fig. 1). The maximum cross-correlation coefficient between MAP and Hypotension Prediction Index signals is –0.91 ± 0.04, with the relationship being negative due to the inverted trend mentioned (fig. 2). The maximum cross-correlation was detected at 0.0 ± 0.0 min time shift for all patients, indicating no time delay between the two signals.

The Hypotension Prediction Index alarm started 3.0 [1.0 to 8.9] min before hypotension occurred, with a concurrent MAP of 71 [70 to 73] mmHg. When a Hypotension Prediction Index alarm was present, the MAP was 75 mmHg or less 94% of the time. When a threshold of MAP 70 mmHg or less was chosen, 98% of the time a Hypotension Prediction Index alarm was also present.

The cross-correlation analysis shows that the Hypotension Prediction Index and MAP are highly correlated, while exhibiting no time delay. This is surprising, since one would expect the Hypotension Prediction Index to leverage multiple features of the preceding arterial waveforms to predict future MAP values. In this sense, our results substantiate the hypothesis by Enevoldsen and Vistisen that the predictive value of the Hypotension Prediction Index above the concurrent MAP may be limited.[1] From our data, it is tempting to hypothesize that setting an alarm at a MAP approximately 70 to 75 mmHg (fig. 1) might yield a prediction of intraoperative hypotension comparable to the Hypotension Prediction Index. In line with our hypothesis, another MAP-based predictive method has recently been reported, using linear extrapolation on current and previous MAP values to predict future MAP.[3]

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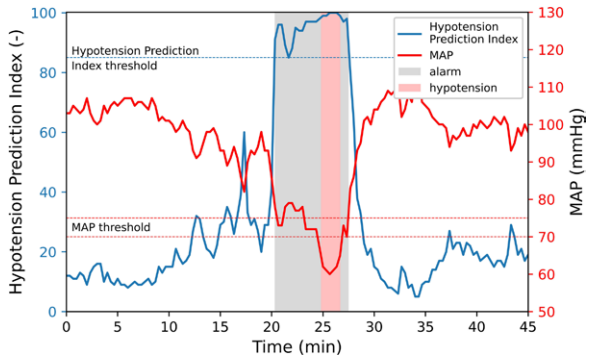

Fig. 1. Continuous intraoperative Hypotension Prediction Index and mean arterial pressure (MAP) monitoring. Representative time course of a patient's Hypotension Prediction Index (*blue*) and MAP (*red*) before, during, and after an alarm (*gray shaded*) and intraoperative hypotensive event (*red shaded*). Thresholds for Hypotension Prediction Index alarm (*blue dashed*) and the hypothesized MAP alarm range (*red dashed*) are given.

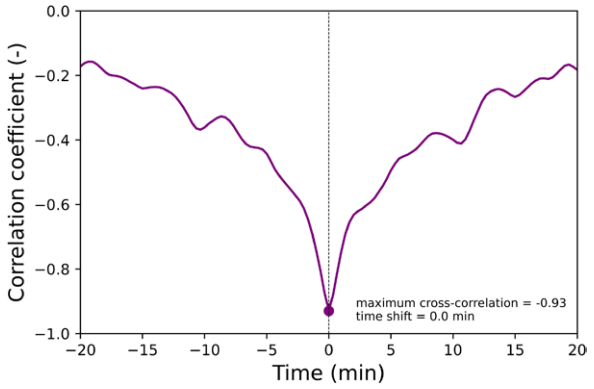

Fig. 2. Hypotension Prediction Index and mean arterial pressure (MAP) cross-correlation. Representative example of a patient's cross-correlation coefficient between the Hypotension Prediction Index and MAP signals. Maximum cross-correlation of –0.93 is detected at 0.0 min time shift.

We realize that the observational nature and limited sample size of this pilot study warrant caution when interpreting our results, including the inferred predictive validity of concurrent MAP alarms. Nevertheless, our results should instigate larger clinical studies to meticulously analyze the clinical performance of the Hypotension Prediction Index in comparison to intraoperative monitoring based solely on MAP.

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Competing Interests

M. P. Mulder, Dr. Donker, and Dr. Fresiello provide research consultancy to Maquet Critical Care AB (Solna, Sweden) but do not receive personal fees. The other authors declare no competing interests.

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