Tracking Hypotension and Dynamic Changes in Arterial Blood Pressure with Brachial Cuff Measurements

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BACKGROUND: Arterial cannulation is strongly recommended during shock. Nevertheless, this procedure is associated with significant risks and may delay other emergent procedures. We assessed the discriminative power of brachial cuff oscillometric noninvasive blood pressure (NIBP) for identifying patients with an invasive mean arterial blood pressure (MAP) below 65 mm Hg or increasing their invasive MAP after cardiovascular interventions.

METHODS: This prospective study, conducted in three intensive care units, included adults in circulatory failure who underwent 45° passive leg raising, 300 mL fluid loading, and additional 200 mL fluid loading. The collected data were four invasive and noninvasive MAP measurements at each study phase.

RESULTS: Among 111 patients (50 septic, 15 cardiogenic, and 46 other source of shock), when averaging measurements of each study phase, NIBP measurements accurately predicted an invasive MAP lower than 65 mm Hg: area under the receiver operating characteristic curve 0.90 (95% CI: 0.71–1), positive and negative likelihood ratios 7.7 (95% CI: 5.4–11) and 0.31 (95% CI: 0.22–0.44) (cutoff 65 mm Hg).

For identifying patients increasing their invasive MAP by more than 10%, the area under the receiver operating characteristic curve was 0.95 (95% CI: 0.92–0.96); positive and negative likelihood ratios (cutoff 10%) were 25.7 (95% CI: 10.8–61.4) and 0.26 (95% CI: 0.2–0.34).

CONCLUSIONS: NIBP measurements have a good discriminative power for identifying hypotensive patients and performed even better in tracking MAP changes, provided that one averages four NIBP measurements.

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Aggressive therapy, started as early as possible and titrated to achieve predetermined goals of global and regional tissue perfusion, is now recognized as the standard of care for patients with circulatory failure.[1–4] In this setting, monitoring of arterial blood pressure (BP) is of utmost importance as it constitutes a primary index of global tissue perfusion.[1,2] Arterial cannulation is the "gold standard" for BP measurements and is recommended as soon as possible during shock.[1] Nevertheless, this procedure is associated with significant risks.[5–12] In case of technical difficulties, it may delay other emergent procedures, such as diagnostic imaging, surgery, or central venous line placement. These downsides probably explain, at least in part, why brachial cuff oscillometry is still so widely used,[13] despite the fact that important discrepancies in measured values have been reported compared with intraarterial BP measurements in critically ill patients.[14–17] In clinical practice, rather than providing very accurate absolute values, the purpose of BP monitoring during initial resuscitation is to assess the patient's response to therapy and to identify patients who have not achieved the recommended resuscitation end points, i.e., a mean arterial blood pressure (MAP) above 65 mm Hg.[3,18,19] Studies which evaluated only the absolute bias of noninvasive BP (NIBP) toward intraarterial measurements are therefore of little help in assessing the potential merits of NIBP monitoring in the critically ill. We hypothesized that NIBP measurements may be used during resuscitation to identify patients who require rapid therapy to increase BP and to assess their response to therapy. If true, even biased toward intraarterial measurements, NIBP measurements could allow time for other emergent procedures to be performed until an arterial line can be placed under optimal safety conditions. This could argue for the validity of using NIBP for initial monitoring of patients with acute circulatory failure, a common but poorly evaluated practice.[13]

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The aim of this study was to assess the ability of NIBP measurements to discriminate between 1) patients with or without a MAP below 65 mm Hg and 2) patients whose MAP does or does not increase by more than 10% after cardiovascular interventions often performed during the first day of resuscitation.

METHODS

Inclusion

The study protocol was approved by our regional ethics review board (Tours, France). Patients were included prospectively within 48 h of intensive care unit admission either after a written informed consent was obtained from a relative, or after emergency enrollment followed by delayed consent, as allowed by French law. Criteria for enrollment were patient undergoing sedation and mechanical ventilation, presenting with circulatory failure (defined as the presence of at least one of the following: systolic arterial BP below 90 mm Hg, MAP below 65 mm Hg, arterial lactate level above 2.5 mmol/L, vasopressor infusion) persistent after insertion of an arterial line. Patients were not included in case of diuretic treatment within the last hour, uncontrolled hemorrhage, leg amputation, phlebitis of the lower limbs, severe hypoxemia (Pao_2/fraction of inspired oxygen (Fio_2) <70 mm Hg), allergy to modified fluid gelatin, or Body Mass Index above 34 kg/m^2. When the NIBP device failed to display a BP value at baseline, the patient was excluded. Demographic, clinical (skin mottling, urine output, admission diagnosis), and laboratory data were collected at baseline.

Measurements

MAP was measured simultaneously with an automatic oscillometric measuring device (SC9000× monitor, Siemens AG, Munich, Germany or Intellivue M70 monitor, Philips Medical Systems, Best, The Netherlands), depending on the study center, using an adapted brachial cuff size (according to recent guidelines),[20] and with a pressure transducer (T100209A, Edwards Lifesciences, Irvine, CA or PiCCO®, Pulsion medical systems AG, Munich, Germany) connected to a radial (arm opposite to oscillometric measurement) or femoral intraarterial line. Measurements were performed four times at 30- to 60-s intervals at each protocol phase (Fig. 1): 1) at baseline, i.e., patient supine; 2) after 1 min of 45° manual passive leg raising, patient supine,[21] 3) after 300 mL modified fluid gelatin fluid administration, patient supine, legs horizontal, and 4) after an additional 200 mL fluid administration, patient supine, legs horizontal (total volume 500 mL). Cardiac output was also recorded at each time point.

Statistical Analysis

Hemodynamic changes induced by the cardiovascular interventions were assessed using analysis of variance between protocol phases and a *post hoc* Fisher procedure of least significant difference.

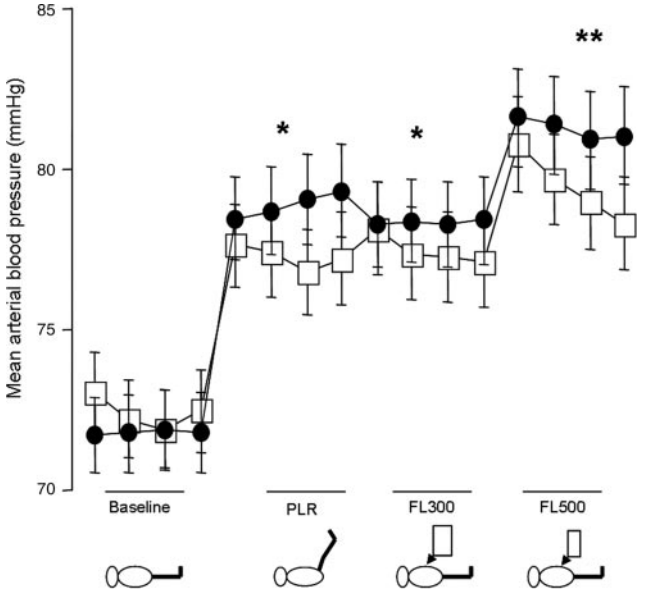

Figure 1. Protocol-induced mean arterial blood pressure changes. Circles represent intraarterial pressure measurements. Squares represent noninvasive oscillometric blood pressure measurements. Bars represent standard errors. PLR = passive leg raising; FL300 = measurements after 300 mL rapid fluid loading; FL500 = measurements after additional 200 mL rapid fluid loading (total volume 500 mL). *$P < 0.05$ for invasive and oscillometric measurements compared with baseline; **$P < 0.05$ for invasive and oscillometric measurements compared with all other study phases.

For both absolute MAP values and changes between phases, agreement between the two techniques was assessed using Bland and Altman analysis.[22] Limits of agreement and their 95% confidence intervals (95% CI) were calculated, considering repeated measurements within subjects as appropriate and the repeatability coefficient within each phase ($1.96 \times \sqrt{2} \times \sqrt{}$[intrapatient variance of measurements within one phase]) was calculated and expressed as a percentage of the mean of the four measurements.[23,24]

The discriminative power of NIBP for identifying patients with an invasive MAP below 65 mm Hg and patients increasing their invasive MAP by more than 10% was determined using receiver operating characteristic curve analysis and calculation of positive and negative likelihood ratios (LHR) for different cutoffs.[25] Analysis was performed on the whole dataset and in different subgroups to look for confounding factors (NIBP device, femoral or radial artery cannulation, arrhythmia, study phase, patients with low MAP, and/or low cardiac output). Before data analysis, the Gaussian distribution of all variables was ascertained. Data were processed using StatsDirect 2.5.0.7 (StatsDirect, Altrincham, UK) and reported as mean ± SD. An α risk below 5% was considered significant.

This study was previously presented as an abstract[26] and shares patients with another work.[27]

RESULTS

One hundred sixteen patients were included over an 18-mo period. Five patients were excluded because the NIBP device failed to display a BP figure at baseline; NIBP measurements could be performed in all other patients at all time points. All the excluded patients had either anuria, skin mottling, high lactate level (median 5.3 [range, 5.6–14.7]), low invasive MAP (median 57.5 [range, 53–64]), low cardiac index (median 1.65 [range, 0.3–2.1]), or were receiving IV catecholamines, and were monitored by a Siemens SC9000× monitor. All of them were hypotensive (invasive MAP < 65 mm Hg) and one was arrhythmic.

One hundred eleven patients were further analyzed (Table 1). Of these, 109 were studied within the first 24 h of admission, and 2 between 24 and 48 h. The cardiovascular interventions were successful in inducing significant BP changes throughout the protocol (Fig. 1). All included patients were undergoing IV sedation. The mean values of repeatability coefficients for NIBP measurements were 9.4% and 7.4% for the Siemens SC9000× and the Philips MP70 monitors, respectively. As we considered these values as clinically significant, we performed all subsequent calculations using the average of four measurements. The repeatability coefficient for intraarterial MAP was significantly lower, ranging from 3.1% to 4.8% according to study phases.

Table 1. Patients' main clinical characteristics at baseline

Variable	Value
Age (years)	58 ± 17.3
BMI (weight [Kg]/height^2 [m^2])	24 ± 4
SAPS II	58 ± 17
Systolic arterial blood pressure (mm Hg)	107 ± 20
Diastolic arterial blood pressure (mm Hg)	55 ± 11
MAP (mm Hg)	71.6 ± 12.2
MAP <65 mm Hg	32 (29%)
MAP increase ≥10% after 500 ml volume expansion	59 (54%)
Central venous pressure (mm Hg)	11 ± 4
Cardiac index ($L \cdot min^{-1} \cdot m^{-2}$)	3.5 ± 1.3
Cardiac index <2.0 $L \cdot min^{-1} \cdot m^{-2}$	12 (11%)
Arterial lactate concentration (mmol/L)	3.5 ± 4
Arterial lactate concentration >2.5 mmol/L/>4 mmol/L	42 (38%)/26 (23%)
Urine output during the last hour ($ml \cdot Kg^{-1} \cdot h^{-1}$)	0.8 ± 0.7
Urine output during the last hour < 0.5 $ml \cdot Kg^{-1} \cdot h^{-1}$	41 (37%)
Mottled skin	36 (32%)
Mottled skin and/or urine output < 0.5 $ml \cdot Kg^{-1} \cdot h^{-1}$	63 (57%)
Catecholamine infusion	104 (94%)
Norepinephrine (μg/Kg/min) [n = 87]	0.71 ± 0.84
Epinephrine (μg/Kg/min) [n = 27]	0.4 ± 0.42
Dobutamine (μg/Kg/min) [n =31]	9.6 ± 9.5
Sinus rhythm/arrhythmia (atrial fibrillation in all cases)	96 (86%)/15 (14%)
Radial/femoral artery cannulation	22 (20%)/89 (80%)
Siemens SC9000X/Philips Intellivue MP70 monitor	55 (50%)/56 (50%)
Main diagnosis at admission:	
Septic shock	50 (45%)
Acute respiratory failure	19 (17%)
Cardiogenic shock	15 (14%)

SAPS: simplified acute physiology score. BMI: body mass index. MAP: mean arterial blood pressure. Septic shock was defined as persistent MAP below 65 mmHg despite abundant fluid loading. Cardiogenic shock was diagnosed when a demonstrated cardiopathy was not associated with other cause of shock. "Respiratory failure" denotes MAP below 65 mmHg that was linked to high dosage of sedative drugs and high intra-thoracic pressure during mechanical ventilation. Quantitative variables are expressed as mean±SD, qualitative variables as n (%).

Agreement Between Invasive and Noninvasive Measurements

The bias between invasive and noninvasive MAP ranged from −4.3 to +5.3 mm Hg depending on the study phase, but mostly depending on the oscillometric device used, with limits of agreement constantly higher than ±20 mm Hg. Considering the whole dataset, Bland and Altman analysis revealed a normally distributed bias of −2.5 and +4.4 mm Hg, with limits of agreement of ±21 mm Hg (95% CI: 18–24) and ±19 (95% CI: 16–22) for the Siemens SC9000× and the Philips MP70 monitors, respectively (Fig. 2).

In subgroups of patients with an invasive MAP below 65 mm Hg (n = 32) or a cardiac index below 2.0 $L \cdot min^{-1} \cdot m^{-2}$ of body area at baseline (n = 12), or mean dosage of norepinephrine above 1 $\mu g \cdot kg^{-1} \cdot min^{-1}$ (n = 20), the agreements between invasive and noninvasive measurements were similar to the agreement in the whole population (Fig. 3).

For detecting changes (as expressed in percentage) in MAP in the whole dataset, the bias between the two techniques was −3.3% and 1.0%, with limits of agreement of ±28% (95% CI: 24–31) and ±24% (95% CI: 21–27) for the Siemens SC9000× and the Philips MP70 monitors, respectively (Fig. 2).

Discrimination of Patients with a MAP Below 65 mm Hg

When all study phases were analyzed together, the area under the receiver operating characteristic curve (AUC) for noninvasively discriminating patients with an invasive MAP below 65 mm Hg was 0.90 (95% CI: 0.71–1). With a cutoff at 65 mm Hg, the positive LHR was 7.7 (95% CI: 5.4–11.0) and the negative LHR was 0.31 (95% CI: 0.22–0.44). With a cutoff at 70 mm Hg, the positive LHR was 3.7 (95% CI: 3.0–4.6) and the negative LHR was 0.20 (95% CI: 0.12–0.33) (Fig. 4). At baseline, three hypotensive patients (invasive MAP below 65 mm Hg) had a NIBP measurement above 70 mm Hg. They all had oliguria (<0.5 $mL \cdot kg^{-1} \cdot h^{-1}$) and/or skin mottling.

Discrimination of Patients Increasing Their MAP by at Least 10%

When all study phases were analyzed together, the AUC for noninvasively discriminating patients who

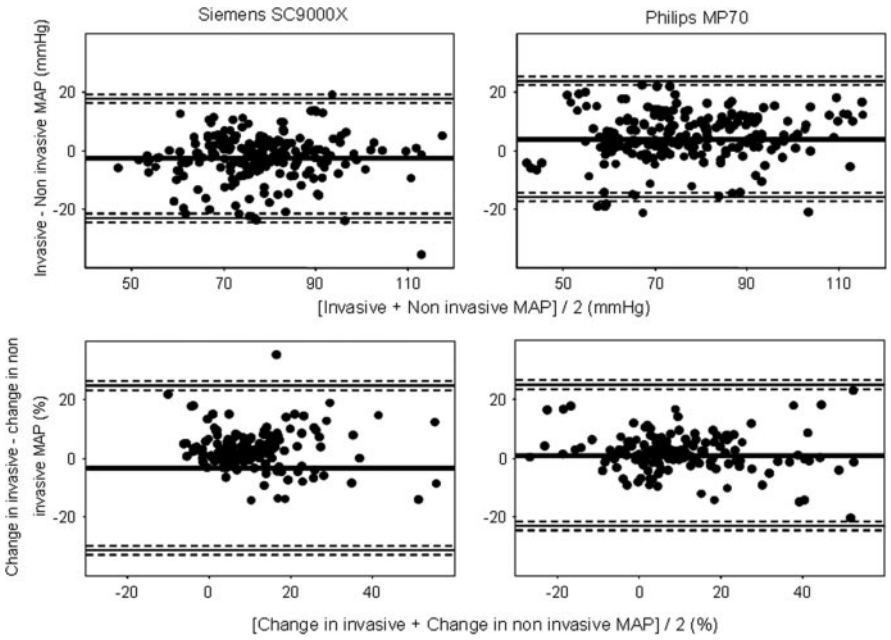

Figure 2. Agreement between invasive and noninvasive mean arterial blood pressure measurements. Bland and Altman diagrams for the Siemens SC9000× monitor (left) and Philips MP70 monitor (right) for absolute values (top) and changes (bottom) in mean arterial blood pressure (MAP). Thick horizontal lines represent the mean bias, thin continuous lines the limits of agreement (2 standard deviations), and the dotted lines their 95% confidence interval.

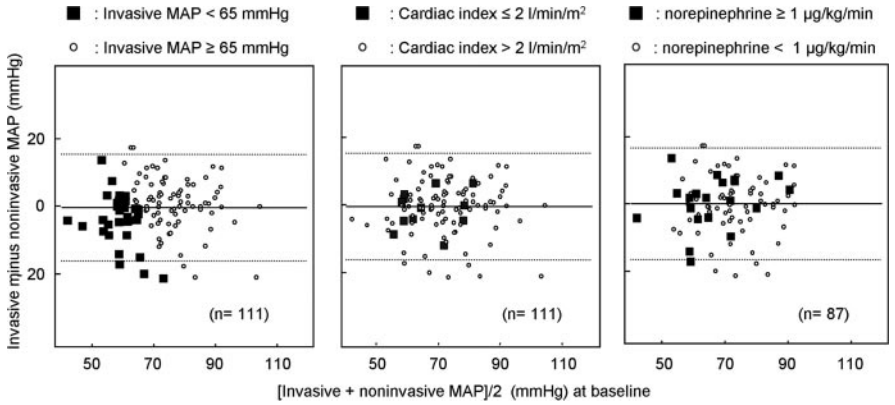

Figure 3. Agreement between invasive and noninvasive baseline mean arterial pressure (MAP) in different subgroups of patients. The bias and limits of agreement between invasive and noninvasive MAP at baseline were similar to the whole population values. Values are shown for the 32 patients with invasive MAP below 65 mm Hg (bias −0.4 mm Hg [15.4–16.2]) (left), for the 12 patients with cardiac index below or equal to 2 $L \cdot min^{-1} \cdot m^{-2}$ (bias −1.3 mm Hg [−12.5 to 9.9]) (middle), and for the 20 patients out of 87 receiving norepinephrine with a norepinephrine dosage above or equal to 1 $\mu g \cdot kg^{-1} \cdot min^{-1}$ (bias −0.4 mm Hg [−6.3 to 15.4]) (right).

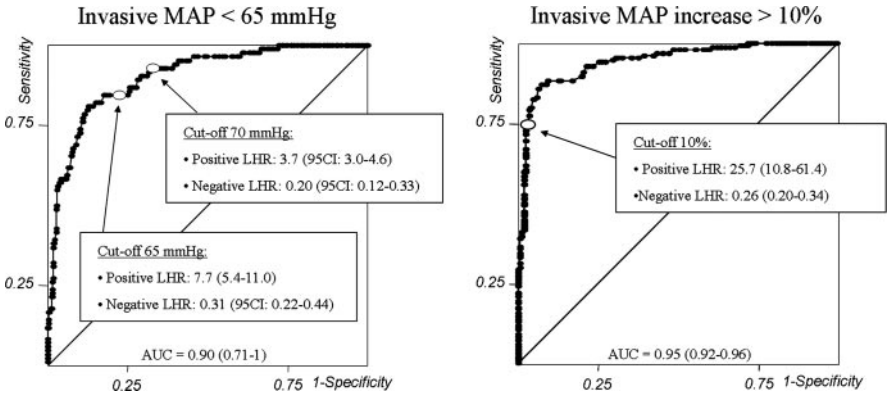

Figure 4. Receiver operating characteristic curves. Curves are plotted for the noninvasive prediction of an invasive mean arterial pressure below 65 mm Hg (left) or an increase of more than 10% between two study phases (right), using the mean of four measurements at all study phases. MAP = mean arterial pressure; LHR = likelihood ratio; AUC = area under the curve.

increased their invasive MAP by at least 10% between two phases was 0.95 (95% CI: 0.92–0.97). The positive LHR (cutoff 10%) was 25.7 (95% CI: 10.8–61.4) and the negative LHR was 0.26 (95% CI: 0.20–0.34) (Fig. 4).

When all study phases were analyzed together, NIBP performed similarly in the subgroup of 32 patients with an invasive MAP below 65 mm Hg at baseline (AUC was 0.93 [95% CI: 0.87–0.98], positive LHR was 12.9 [95% CI: 4.3–38.7], negative LHR was 0.13 [95% CI: 0.06–0.27], best cutoff 8%). When examining the performance of NIBP to track a 10% change in invasive MAP at each study phase in this subgroup,

we found similar results: AUC = 0.90 (95% CI: 0.74–0.98) during passive leg raising, AUC = 0.88 (95% CI: 0.72–0.99) after 300 mL fluid administration, AUC = 1 (95% CI: 0.89–1) after 500 mL fluid administration.

All results were similar when we considered specific study phases, specific NIBP monitor, presence of arrhythmia, or site of arterial cannulation (data not shown).

DISCUSSION

Principal Findings

As expected, our results show bias and limits of agreement between invasive MAP and NIBP of the same magnitude as reported by others.[14,16,17] Furthermore, we observed a high variability of NIBP measurements, which stresses the need for their averaging over time. Nevertheless, our results strongly suggest that, in patients with circulatory failure, NIBP measurements may be used to discriminate between patients who have or have not achieved resuscitation goals, and to very accurately assess dynamic cardiovascular changes induced by therapy. Therefore, our findings validate the wide use of NIBP measurements for these purposes.

Our study focused on MAP, as oscillometric MAP is a direct measurement and represents overall organ perfusion pressure, systolic and diastolic values being obtained indirectly through manufacturer-owned extrapolation algorithms.[20]

Sixty-five millimeters of mercury is the MAP target of initial resuscitation currently recommended[1,3] on the basis of studies showing little benefit of increasing this threshold[18,19] or demonstrating a mortality reduction, thanks to an algorithm including this MAP target.[4]

Strengths of This Study

Major strengths of the present study are that 1) we included a large number of patients with only four repeated measurements for each patient and 2) we recorded MAP after cardiovascular interventions, thereby allowing us to assess the accuracy of NIBP measurements in tracking individual changes in MAP over a wide pressure range.[28] In this regard, most studies evaluating agreement between NIBP and intraarterial BP in critically ill patients have reported impressively large numbers of measurements[15,16] in a limited number of patients or did not track MAP changes within the same patient,[17] which is the major purpose of continuous monitoring in the intensive care unit.

Weaknesses of This Study

We used two different devices for NIBP measurements in two distinct groups of patients and showed that the measurement device influences NIBP measurements' accuracy, as the two algorithms exhibited opposite bias. We noted, as already reported by others,[15] that the older algorithm used (Siemens SC9000× in our study) was not as reliable as the more recent one (Philips M70 in our study), as five failures to provide any BP value occurred in five patients with severe shock, whereas this never happened with the more recent algorithm. One can suppose that in the future, NIBP algorithms may further improve. Nevertheless, the failure to display any BP figure may be interpreted as an alert signal. Indeed, this situation always occurred in patients with severe shock exhibiting invasive MAP values below 65 mm Hg. Thus, including those five excluded patients in our whole population would have increased the number of hypotensive patients correctly diagnosed by NIBP measurements (better negative LHR [0.12 (0.04–0.36)] with a cutoff of 70 mm Hg). Specific evaluation of invasive MAP in a larger number of patients where NIBP fails to provide a MAP figure would be needed to draw conclusions in this situation. The number of cuff insufflation cycles which were necessary to obtain a MAP value was not recorded during the study, and in some cases the time needed to obtain four MAP measurements was relatively long. Again, this point should be addressed in a study assessing the duration of oscillometric measurements.

Because NIBP measurements were shown to be particularly inaccurate among critically ill patients who were significantly overweight,[14,16] those patients were excluded from our study. Thus, our results cannot be generalized to this patient population. This point is also true for patients in hemorrhagic shock.

A patient's cardiac rhythm can influence the accuracy of NIBP measurements,[29] and the 15 patients (14%) with arrhythmia we included could have attenuated the good discriminative power of NIBP we found. However, as we studied few arrhythmic patients, our results cannot be generalized to this specific population.

We studied a heterogeneous population of patients treated with various dosages of vasopressors, in whom BP was sometimes measured via radial (20%) and mostly via femoral cannulation (80%) because of the frequent use of the PiCCO® monitoring device. Vasoconstrictors can affect the difference between brachial and intraradial BP measurements[17] as well as raise the femoral-to-radial pressure difference.[30] However, the bias between NIBP and invasive MAP measurements in patients receiving large dosages of vasopressors was similar to its value in the entire population.

Due to the study design (need for arterial cannulation before inclusion), we mostly included patients already resuscitated and stabilized and thus presenting only moderate degrees of hypotension (Table 1). Furthermore, the inclusion of patients over an 18-mo period may have led to some selection bias. Therefore, our results may not fully apply to patients in the very early stage of circulatory failure in the emergency department or to severely hypotensive patients. This may be the major weakness of our study. Nevertheless, 29% of the patients we studied had MAP below

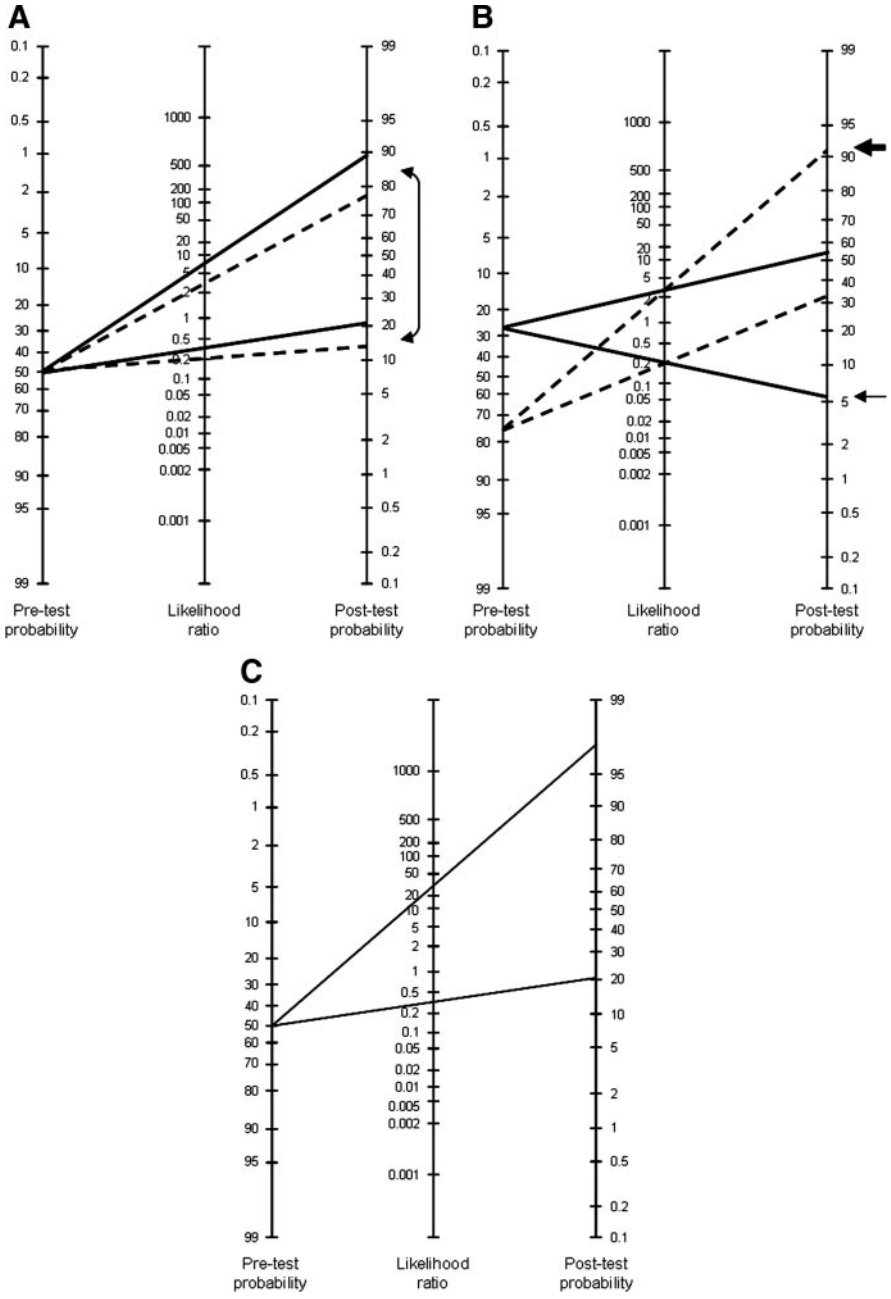

Figure 5. Baye's diagram showing posttest probabilities of hypotension (invasive mean arterial blood pressure [MAP] below 65 mm Hg) or changes in MAP (change of more than 10%) for different pretest probabilities and different noninvasive MAP cutoffs. Panel A: For a 50% pretest probability of hypotension (one does not have any clinical information to judge if the invasive MAP is higher or lower than 65 mm Hg), the solid line indicates the posttest probability of hypotension with a 65 mm Hg noninvasive MAP cutoff, the dotted line with a 70 mm Hg cutoff. In both cases the test result (noninvasive MAP above or below the cutoff) induces a change in absolute probability of about 60%–70% (arrow). Panel B: In a clinical situation the physician often has an idea of the probability of hypotension (suspicion of hypotension based on clinical signs like skin mottling, oliguria, etc.). The diagram presents the posttest probabilities of hypotension for a cutoff at 70 mm Hg with two distinct pretest probabilities of hypotension: low (25%) and high (75%), represented by the solid and the dotted lines, respectively. Posttest probability is then about 5% (thin arrow) if noninvasive MAP is higher than 70 mm Hg, and above 90% (thick arrow) if noninvasive MAP is below 70 mm Hg. Panel C: diagram for probabilities concerning the detection of changes in MAP. An increase in noninvasive MAP of more than 10% is associated with a posttest probability of increase in invasive MAP of more than 95%. The change in posttest absolute probability depending on the test result is higher than 75% (50% pretest probability).

65 mm Hg, 32% had mottled skin, and 38% had high arterial lactate. In addition, 59 patients (54%) increased their MAP by at least 10% after 500 mL fluid challenge in our population. As fluid responsiveness is characteristic of the early hypodynamic phase of septic shock (sepsis being the first cause of shock in our study population), we believe that a significant portion of the patients was still in an early phase of circulatory failure and that our results may be valid in emergency room patients without an arterial line.

Implications for Clinicians

In our study, for tracking changes in MAP, the positive LHR was 25.7. Therefore, as illustrated in Figure 5, NIBP measurement seems to be a very powerful tool to track significant changes in BP.[25] The use of NIBP measurements for identifying hypotensive patients (intraarterial MAP below 65 mm Hg) was also good, as a cutoff of noninvasive MAP set at 65 mm Hg was associated with a positive LHR of 7.7 (40% change in pretest probability) and a negative LHR of 0.31 (hypotension probability decrease from 50% to 24%). Therefore, as also illustrated in Figure 5, it seems that NIBP measurements are of good diagnostic value, because, depending on the test result, the probability of hypotension shifts from 90% to 24%.[25] Depending on the clinical context, some clinicians may prefer to choose a cutoff higher than 65 mm Hg that makes NIBP measurements more sensitive for detecting hypotensive patients. For example, a patient with a NIBP measurement above 70 mm Hg has only a 17% probability of being hypotensive (negative LHR

0.2) (Fig. 5). Indeed, with this cutoff chosen at 70 mm Hg, among our population, NIBP left only 3 (2.7%) true hypotensive patients undetected, all of them presenting other alerting clinical signs of circulatory failure (skin mottling and/or oliguria). With these points in mind, one may consider that a NIBP measurement above 70 mm Hg could be a reasonable resuscitation end point in the absence of an arterial line.

Concerning patients in whom an NIBP device fails to measure BP (patients with very low flow states), the clinical implication is straightforward, as they require immediate treatment.

One may argue that arterial cannulation takes little time and is associated with few complications, thus limiting the clinical value of NIBP measurements.[10] Unfortunately, when complications occur, they may have very severe consequences, often requiring surgical repair or amputation[12] and involving bloodstream infection (about 2–4 cases per 1000 catheter days, an incidence similar to that of central venous catheter infection).[31] Although the amount of time needed for arterial cannulation and its impact on complications have not been extensively studied, emergency cannulation in the rush of initial resuscitation may not be consistent with optimal safety. In particular, the time needed for skin antisepsis and maximal sterile barrier precautions cannot be reduced,[32] and longer than expected lengths of cannulation have been reported.[33]

NIBP measurements thus have broad clinical applications: 1) for monitoring during emergent procedures (e.g., effusion drainage, diagnostic imaging, initial resuscitation) which could be unduly delayed by difficult and potentially dangerous emergent arterial cannulation and 2) in patients in whom risks of arterial cannulation may exceed benefits, such as rapidly terminated circulatory failure after fluid administration in patients with severe sepsis.

Our results do not support the indiscriminate application of the current international guidelines that recommend the quick cannulation of an artery to guide therapy during shock.[1] Indeed, our study tends to validate the operational use of NIBP during the first day of circulatory failure. This less invasive attitude may avoid a significant number of hasty and unnecessary arterial cannulations, although not doing harm to the patients who absolutely require an arterial line after initial treatment. However, at this point, our results only show that NIBP allows detection of patients with a MAP below 65 mm Hg and the tracking of 10% changes in MAP in moderately hypotensive patients; determining whether NIBP can replace arterial cannulation will require further studies.

CONCLUSION

In our population of partly resuscitated patients, NIBP measurements allow an accurate diagnosis of hypotension and a highly reliable tracking of MAP changes, provided that one averages four NIBP measurements.

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