

Blood Pressure: The History and Development of Monitoring Modalities

Desiree M. Cihelka, PhD(c), MSN, RN, ARNP-C, ANVP-BC¹

Abstract

Background

Blood pressure (BP) is one of the most frequently measured and monitored physiologic vital signs by all stroke clinicians, yet data suggest that only 1 out of 5 clinicians applies evidence-based methods for BP monitoring.

Methods

An exhaustive review of the literature was conducted and assembled to provide a historical clinical account of BP monitoring modalities and related evidence-based clinical methods.

Results

Evidence-based clinical methods are described for use of manual sphygmomanometry, noninvasive oscillometric automatic BP (NIBP) monitors, and arterial lines. Implications for practice are discussed in relation to provision of acute and critical care of ischemic and hemorrhagic stroke patients.

Conclusion

Use of evidence-based BP monitoring methods ensures accurate management of highly vulnerable stroke patients. Knowledge of the history of BP monitoring, along with the benefits and limitations of different measurement methods enables accuracy in BP treatment, benefitting stroke patient outcomes.

Key words: Blood pressure; arterial pressure; mean arterial pressure; clinical monitoring; vital signs; acute stroke.

INTRODUCTION

Blood Pressure (BP) is one of the most important signs of human life. It reflects not just the cardiovascular health of an individual but, also is a vital sign of perfusion to organs and cells throughout the body.¹ Because the brain is the most energy-demanding organ in the body, it is the most important and “particular” organ to perfuse.² The healthy adult brain receives an estimated 15-20% of the cardiac output in resting conditions.² Autoregulation ensures that the brain maintains a delicate

balance of cerebral perfusion and neurovascular coupling for the high metabolic demands of oxygen and glucose.³ Neurons are unique because they do not regenerate nor survive without blood flow to deliver oxygen.⁴ Correct measurement of blood pressure is essential to the treatment and management of both chronic diseases and acute illness and can profoundly affect patient outcomes in patients with stroke.

Simply defined, BP is the force of blood pushing through and against arterial walls



with each heartbeat.⁵ Most commonly, the BP measurements used clinically are systole and diastole, followed by mean arterial pressure (MAP).⁶ Systolic blood pressure (SBP) is the pressure caused by the heart contracting and forcing outward blood flow, whereas diastolic blood pressure (DBP) is the pressure during heart relaxation while the left ventricle fills with blood. MAP is the average arterial pressure that pulses through the vasculature in one cardiac cycle.⁷

The importance of SBP/DBP goals in adults has been well established, and both are used for hypertension management in primary and secondary prevention of cardiovascular and cerebrovascular disease.^{5,6,8} In patients with systemic blood flow limitations, as in trauma or types of shock, MAP becomes an increasingly valuable parameter for assessing tissue and organ perfusion.⁹ MAP is influenced by cardiac output and systemic vascular resistance, making it ideal to reflect overall systemic organ perfusion.⁷ Maintaining a MAP that is too low may result in inadequate perfusion and subsequent organ failure, with catastrophic outcomes; conversely, a MAP that is too high can strain the heart muscle and rupture small perforating arterioles.^{10,9} The parameters for MAP in cardiac diseases are tailored to the vascular dynamics of the patient.^{10,11,12} However, goals for MAP remain understudied in neurovascular diseases. In fact, ischemic and hemorrhagic stroke guidelines are totally silent on MAP goals for both acute and post-acute care.

THE HISTORY OF BLOOD PRESSURE MEASUREMENT

There are several methods used in the clinical setting to measure BP. Non-invasive measurements utilizing manual sphygmomanometer as well as automated intermittent oscillometric devices are most common across the world and are used in

nearly all clinical settings. Invasive monitoring is used in select patient groups requiring critical care services for severe illness, injury, frequent blood draws, or in intraoperative monitoring.

While the first clinical BP measurement was reported in 1856 by the surgeon Faivre, who connected an artery to a mercury manometer to obtain direct readings during an operation, the earliest crude observations of pulse pressure in horses were published by Hales in the middle of the eighteenth century.¹³ Hales' methods resulted in the death of each horse, making them unsuitable for studying human BP. It was not until the nineteenth century, nearly 100 years after Hales' work, that the physician-physicist Poiseuille published his work on resistance in the cardiovascular system, pushing innovation forward. Poiseuille's 1882 doctoral dissertation was published on the use of a mercury manometer to measure arterial blood pressure.¹⁴ After some modifications to Poiseuille's equipment, the remaining two decades of the nineteenth century offered methods equipped with graphic recordings. In 1855, Vierordt's sphygmograph emerged which provided the first attempt at non-invasive methods of BP monitoring.¹⁴

NON-INVASIVE BP METHODS

Early non-invasive BP measurement methods required obliteration of the radial pulse by a cumbersome weight attached to a lever on a sphygmograph; this did not fare well in clinical practice. Finally, in 1881, Von Basch presented a more accurate, portable apparatus to investigate the first hemodynamic pathologies with a mercury bulb and rubber bag of water; later, an air-filled bladder replaced the water for arterial compression.¹⁴

Manual Sphygmomanometer

After gradual improvement in BP measurement devices, Riva-Rocci's

mercury sphygmomanometer method emerged in 1896 and is what present-day monitoring is based upon.¹⁵ His inflatable cuff method compressed the brachial artery from all sides equally and allowed for observation of oscillations associated with SBP. Interestingly, it was Harvey Cushing, the American neurosurgeon, who brought this method to the US in 1901 from Italy, marking the beginning of modern non-invasive BP measurement.¹⁶

Riva-Rocci's mercury sphygmomanometer method worked well for SBP, and ultimately, assumptions about DBP arose in relation to different compression pressures. However, Korotkoff's technique offered a crucial advancement in BP measurement by using a stethoscope to listen for the sounds of blood flowing through the artery, ultimately providing more valid and reliable measures of both SBP and DBP.¹⁷ The Korotkoff auscultatory method involves compression of the brachial artery and auscultation over the antecubital fossa to determine the SBP and DBP. Although true MAP is obtained using invasive monitoring, it can also be calculated based on a physiologically sound formula that uses SBP and DBP obtained during auscultation. The MAP calculation was developed from the knowledge that diastole persists for 2/3 and systole for 1/3 of each cardiac cycle; therefore the calculation uses a third of the pulse pressure added to the DBP.^{1,7} $MAP = (SBP - DBP)/3 + DBP$.^{1,7} This simple calculation is still used today to determine MAP from manually-derived SBP and DBP.

Korotkoff's technique has limitations, such as artifacts due to movement and challenging physiologic variations of Korotkoff sound patterns or poor signals that can be difficult to hear by the clinician.¹⁸ Korotkoff's sounds can also be hard to hear in morbidly obese patients or

those with rigid arteries due to systemic atherosclerotic disease or simply advanced age.¹ Additionally, auscultation is often difficult when patients have severe hypotension or organ failure, making Korotkoff sounds difficult to distinguish due to fluctuations in intra-arterial pressure.

The American Heart Association (AHA) Scientific Statement for Recommendations for Blood Pressure Measurement in Humans and Experimental Animals describes the proper cuff and sphygmomanometer technique.¹⁹ First, the examiner palpates the brachial artery in the antecubital fossa and places the bladder of the cuff midline on the patient's upper arm just above the brachial arterial pulsation, with the lower end of the cuff 2 to 3 cm above the antecubital fossa, leaving room for the stethoscope. Artifact can be generated by noise in the room, clinicians or the patient speaking, and if the stethoscope touches the cuff.⁶ The cuff is first inflated without auscultation until the pulse is no longer palpable; this indicates suspected systolic pressure.⁶ The cuff is further inflated to at least 30 mmHg above the point at which the radial pulse disappeared; auscultation occurs during slow deflation of the cuff.²⁰ The rate of deflation has a significant effect on blood pressure determination.²¹ Deflation rates > 2 mm per second can lead to a significant underestimation of SBP and an overestimation of DBP.¹⁹ Korotkoff sounds accompany the return of pulsatile blood flow as the cuff deflates, with phase 1 sounds indicating SBP and phase 5 sounds indicating DBP. A full 5 minutes should occur between re-inflation of the BP cuff to allow for arm revascularization.

Automatic Oscillometric Methods

Until the 1980s, the manual sphygmomanometer was the only means of obtaining a non-invasive BP. The evolution

of computers and the need for timely, non-invasive BP measurement led to the first automatic oscillometric BP (NIBP) device patent by Ramsey, III (Johnson & Johnson, assignee), with approval for use in 1982 (Device Non-invasive Measurement of Arterial Pressure [DINAMAP]).²² These early devices only displayed MAP, but this quickly evolved to devices that displayed algorithmically determined SBP and DBP, which gave way to later generations of the more sophisticated devices used today.^{1,22,23} NIBP devices have become the standard of care within American and most high and middle income healthcare settings, with many companies manufacturing bedside instruments and software packages.

Digital NIBP devices do not measure SBP and DBP. Instead, these devices use software to algorithmically assign an estimate of SBP and DBP from the measurement of MAP.^{23,24} The endpoint for DBP is indistinct since cuff pressure oscillations continue when cuff pressure falls beneath diastolic blood pressure.^{23,25} MAP is determined by measuring cuff pressure oscillations as the cuff pressure is reduced by discrete increments.²⁶ The heartbeat-induced pulse volume changes small oscillations of intra-cuff pressure, which are sensed by the cuff and measured by a pressure transducer, allowing incremental data to be tested for artifact and averaged rather than to allow for continuous artifact-rejection.²³ MAP is selected as the lowest cuff pressure at maximum oscillation amplitude.²⁴ MAP correlates directly with maximum oscillations and is accepted as the most accurate value derived from NIBP devices.²⁴ NIBP devices are not recommended for use in patients with arrhythmias that impact stroke volume, such as atrial fibrillation,^{23,24,27} because of an erratic MAP with stroke volume variation that results in inaccurate device

performance. Unfortunately, very few interprofessional clinicians are aware of this fact, leading to continued use of NIBP in patients with atrial fibrillation which leads to highly invalid and unreliable BP measurement. Many clinicians also fail to document MAP when documenting vital signs from an NIBP device despite the fact that MAP is the most accurate pressure measured by NIBP. Although the MAP formula could be applied, interestingly, there is no agreement between the MAP generated from an NIBP and the MAP calculated from NIBP-produced SBP and DBP; this fact clearly demonstrates the proprietary, unknown nature of the formulas used by these devices to produce SBP and DBP variables from the NIBP estimated MAP.

Each NIBP manufacturer uses different computer algorithms and software for testing and validation procedures to estimate MAP, SBP, and DBP data, and these vary significantly from one device to another.²⁸ These proprietary patented algorithms for SBP/DBP present a scientific dilemma as they cannot be easily validated by external authorities.²¹ To ensure the accuracy of data from machines and the validity of devices, in 1987, the American National Standards Institute (ANSI) and the United States Association for the Advancement of Medical Instrumentation (AAMI) published the first joint standards and recommended practices for automated non-invasive oscillometric devices.²⁸ Updates have been made in subsequent years from ANSI/AAMI and others from around the world through protocol publication by the International Organization of Standardization (ISO) for device validation.²⁸ The most recent update from ANSI/AAMI/ISO states that a device is considered acceptable if its estimated probability of a tolerable error (< 10 mm Hg) is at least 85%;^{29,28} this means that

there should be no more than a 10 mm Hg difference in BP readings in at least 85% of patients tested. While statistically, this tolerable error may be acceptable, clinically, a 10 mm Hg difference in BP readings can lead to potentially harmful therapeutic interventions or missed opportunities to implement critical treatments. For example, failure to lower BP in an intravenous thrombolysis patient because the reading was 172 mm Hg SBP, while the “true” reading was 182 mm Hg, may increase the risk for a symptomatic intracerebral hemorrhage (sICH).

The proper cuff size is paramount for any “cuff-based” non-invasive method of BP measurement, and inappropriate cuff sizing constitutes the most common form of measurement error. The “ideal” cuff should have a bladder length of 80% and a width of at least 40% of the arm circumference (a length-to-width ratio of 2:1).¹⁹ However, in patients with morbid obesity, the very large arm circumference combined with a short upper arm length often results in geometry that cannot be correctly cuffed.²¹ Difficulty with properly applying BP cuffs in obese patients often causes clinicians to resort to the use of a thigh cuff placed on the forearm, with Korotkoff sounds auscultated over the radial artery by the wrist; unfortunately, this method may overestimate SBP.^{30,19} Even when brachial cuffs are used, on average, NIBP tends to overestimate BP during hypotension and underestimate BP during hypertension, demonstrating significant measurement bias.³⁰ In comparison, invasive BP measurement with an arterial catheter can detect nearly twice as many episodes of hypotension as NIBP taken with a brachial cuff.³¹

INVASIVE BP MONITORING

Invasive arterial pressure monitoring by arterial lines, or “A-lines,” constitutes the

gold standard for BP measurement; however, it is used most commonly in critical care and intraoperative settings.^{7,32} Unlike discontinuous cuff measurements, the A-line offers a direct and continuous measurement method that gives more accurate and frequent detection of alterations in BP for the most critical patients at high risk of complications. Precise measurements in the most vulnerable patient populations can make a significant difference, as observed measurement differences are clinically significant because they trigger a change in treatment in as many as 20% of critical care patients.³³ Indications for A-lines are as follows:³⁴

- Patients who require timely adjustments for sudden changes in hemodynamics, where interval BP measurements may be unsafe.
- Patients being treated with vasoactive medications requiring safe and precise titration of medication to the desired blood pressure effect.
- Surgical patients at increased risk of morbidities or mortality, either because of preexisting comorbidities (cardiac, pulmonary, anemia, etc.) or because of more complicated procedures. These include but are not limited to neurosurgical and cardiopulmonary surgeries and procedures in which a large volume of blood loss is anticipated.
- Patients who require frequent lab draws. The A-line allows clinicians easy blood sampling access without repeat venipuncture, thereby decreasing discomfort and infection risks. Additionally, frequent arterial blood gas measurements can be obtained by A-line to guide adjustments in mechanical ventilation settings; hemoglobin, and hematocrit can also be monitored in relation to blood loss or transfusion, and both electrolyte imbalance/correction,

and patients' responsiveness to fluid resuscitation can be monitored.

Much like Faivre's original technique, modern arterial pressure monitoring is accomplished by cannulating an artery to measure real-time BP changes. Generally, a peripheral artery puncture site, such as the radial artery, is chosen for ease of access. A short catheter is placed inside the artery's inner wall and connected to a pressure transducer. The pressure transducer interface changes the mechanical pressure into SBP, DBP, and MAP measures for use in clinical practice.³⁵ The phlebostatic axis corresponds to the height of the patient's right atrium, which is the hydrostatic pressure reference level, and the transducer must be kept at this level to ensure accurate pressure measurements.³⁴ Most high income countries utilize transducers that are disposable and calibrated by the manufacturer; these are filled with a saline interface and zeroed at the phlebostatic axis. Accuracy of disposable transducers must be less than $\pm 3\%$ or ± 3 mm Hg in accordance with the published ISO/ANSI standards.^{36,37}

Transducers are prone to drift and must be re-zeroed at regular intervals.³³ To accurately measure BP using an A-line, the clinician must level and zero the transducer and check the quality of the resulting BP waveform.²⁶ The morphology of the BP waveform changes when the BP wave moves from the aorta to a more peripheral artery.³⁸ Correct leveling of the transducer is crucially important, as a height difference between the transducer level and the level of the cannulated vessel of only 10 cm results in a pressure difference of 7.5 mmHg because of the hydrostatic pressure gradient.³⁸ Zeroing is considered successful when the fluid-filled transducer interface shows a pressure line at 0 mmHg with the stopcock turned off to the patient's

artery; the stopcock is then returned to a position that opens the interface between the patient's artery and the transducer to resume monitoring. The optimal quality of the arterial waveform is fundamental to correct BP and measurements, as well as hemodynamic variable measurements.³⁹

There are risks of inaccuracy during leveling, zeroing, and transducing A-lines caused by human error. The natural frequency of the measurement must exceed the frequency range of the arterial pulse; this extends to 20-25 Hz for accurate measurement of maximum pressure during left ventricular isovolumetric contraction (dP/dtmax), which is reflected by the systolic upstroke on the arterial waveform.⁴⁰ The A-line system requires critical dampening to prevent overshoot of measurements. In theory, proper dampening ensures the amplitude is accurately measured within 2% in up to two-thirds of the natural frequency, with distortion rates no more than 6% of the natural frequency.³³ Over-dampening results in under-reading of SBP and dP/dtmax, while over-reading the DBP. Additionally, when under-dampening occurs, the SBP average can be overestimated.⁴⁰ Some studies have shown that SBP overestimation of 2.6 ± 1.9 (mean $\pm$ SD) mm Hg can occur even in adequately dampened systems.³³ Clinicians should understand the importance of the shape of arterial BP waveforms due to dampening and resonance phenomena. The BP waveform is a complex amalgamation of antegrade and retrograde pressure waves. It is affected by vascular compliance (e.g., atherosclerosis), distance of the transducer from the left ventricle, and the 3D structure of the vascular tree.⁴¹ MAP is considered the most accurate variable produced by A-line since it is less affected by dampening and resonance than SBP and DBP.³⁹

Mean arterial pressure is the product of cardiac output (CO), systemic vascular resistance (SVR), and venous volume (as reflected by central venous pressure [CVP]), where CVP is typically a value of 0 mm Hg. However, SVR is strictly a calculated variable that cannot be measured directly and requires a known MAP to calculate ($SVR = 80 \times [MAP - CVP] / CO$); therefore, the simplified MAP equation of one-third the pulse pressure added to the DBP is used in clinical practice.⁴² But the relationship between CO, SVR, and MAP is important to acknowledge as the bedrock for understanding systemic hemodynamics, in that a decrease in CO produces an increase in SVR in an attempt to ensure the stability of MAP. This inverse relationship between CO and SVR is essential to understand; in fact, under non-septic conditions, severe drops in CO that lead to a decrease in MAP are classically still accompanied by markedly elevated SVR. DBP can be used clinically to reflect changes in SVR since SVR cannot be measured directly; for example, abnormally low DBP reflects extremely low SVR, as seen in conditions such as sepsis, whereas abnormally high DBP reflects increases in SVR which may occur during severe hypovolemia, early cardiogenic shock, or in combination with elevation in SBP during states of acute downstream increased resistance to blood flow resulting in sympathomimetic activation (e.g. increased intracranial pressure, vascular stenoses or occlusions).

Although non-invasive continuous BP monitoring has fewer complications than arterial cannulation, it has not replaced A-line monitoring as the standard of care in high-risk patients. For example, patients with hemorrhagic stroke are at high risk for mortality and morbidity, making them more likely to require A-lines. According to the AHA/ASA guidelines, these patients

should be admitted to critical care or specialized stroke units and managed by specialty-trained vascular neurologists and neurocritical care clinicians.⁴³ While not all hemorrhages are severe nor require mechanical ventilation, hemorrhagic stroke patients very commonly require vasoactive medications that must be titrated. Of paramount significance, most current therapeutic interventions for intracerebral hemorrhage are based upon the assumption of accurate BP measurements, making A-lines the most optimal choice despite concerns for infection tied to invasive monitoring.

COMPARISON STUDIES BETWEEN BLOOD PRESSURE METHODS

Clinicians and researchers must ensure that valid and reliable measures of BP are obtained. Efforts have been made to determine factors associated with different BP measurement devices, most of which have been done on healthy individuals, making it difficult to determine consensus regarding chronically ill or acutely ill individuals. The AHA states there is little to no evidence regarding validation of BP measurements obtained in the acute care setting^{6,1} which is unsettling.

There are limited comparison studies for BP methods in specific disease processes and patient populations, surprisingly even in patients with hypertension diagnoses. Epidemiologists who study the prevalence of hypertension conducted comparison studies between NIBP and mercury sphygmomanometer and found that despite minor differences in mean values, the agreement and reliability were deemed not good enough to adopt in epidemiologic surveys of hypertension because the “true” prevalence of hypertension was determined to be significantly underestimated.⁴⁴

Comparison studies in critically ill patients can be especially challenging because of compromised vascular systems and less than favorable critical care unit conditions. Additionally, some studies have shown that NIBP measurements are inaccurate in critically ill patients.^{35,45} For example, a critical care study using the AAMI protocol compared 150 pairs of measurements using three different brands of NIBP devices, versus the average of 1 minute of intra-arterial reference pressures, found that only MAP was accurately measured by all, one device measured DBP accurately, and there were no devices that measured SBP accurately.⁴⁵ Another study with 852 patients utilized regression-based Bland-Altman technique to evaluate differences of 27,022 concurrently measured A-line/NIBP sample pairs, found that clinically significant discrepancies existed between the two, especially during hypotension and suggested that MAP rather than SBP is the preferred metric to guide ICU therapy.³⁵

Another critical care study looking at 3 different BP methods (oscillometric, auscultated, and palpated) compared to A-line that included 44 patients with common diagnoses known to have unstable hemodynamics requiring A-line placement (septic shock 47.7%; stroke 13.6%; increased intracranial pressure 13.6%).⁴⁶ The data from this study showed that palpated SBP values had a clinically significant difference compared to A-lines. There was no significant statistical difference between the oscillometric and auscultated SBP readings, but NIBPs were found to significantly under-estimate A-line measurements. Findings also suggested that auscultated and oscillometric BPs may provide similar measurements.⁴⁶

Recently, comparison work on the agreement between manual and NIBP measurements of SBP, DBP, and MAP in

ischemic stroke patients treated with intravenous thrombolysis with analysis using Bland-Altman technique and Lin Concordance correlation coefficient found that NIBP devices produce significantly different BP measures than manual sphygmomanometry auscultated BP.⁴⁷ The differences in SBP, DBP, and MAP between NIBP and sphygmomanometry failed to reach AAMI guideline recommendations.⁴⁷ These data are concerning since the BP thresholds used for BP lowering to reduce the risk for sICH after thrombolysis were derived from manual sphygmomanometry methods in the NINDS rt-PA Stroke Study, because of a concern that bruising would occur from NIBP cuffs after thrombolytic treatment. Currently, a similar study is underway in patients with hypertensive intracerebral hemorrhage. Collectively, these two studies may create a need to rethink the widespread use of NIBP, and/or the neurovascular community's use of SBP values instead of use of MAP when NIBP devices are utilized.

Current comparison research has led to no consensus as to which device is "best" to use in the acute care setting or with any cohort of critically ill patients such as patients with stroke. There is a vital need to better understand the agreement of BP methods and how this affects blood pressure management. Furthermore, it is not without consequences when there are discrepancies between devices.

IMPLICATIONS FOR PRACTICE

This article identifies many key implications for practice that are tied to the need for accurate BP monitoring. The physiology of cerebral blood flow (CBF) and BP are closely intertwined. Optimal perfusion to the brain is driven by

autoregulation, which, under normal conditions, allows for constant blood flow to the brain despite variations in arterial pressure. Intact autoregulatory processes illustrate the relationship between MAP and mean CBF⁴⁸; active regulation of brain arterial vasoconstriction and dilation occur to maintain a MAP between 60 to 150 mm Hg.³

Overall cerebral perfusion can be estimated by calculating cerebral perfusion pressure (CPP), which is the product of MAP minus intracranial pressure (ICP). However, this formula does not account for discrete CPP within the boundaries of local hemodynamically challenged vascular regions.^{49,50} In fact, within local regions deprived of sufficient arterial blood flow due to hematoma compression in ICH, vasospasm in aneurysmal subarachnoid hemorrhage (aSAH), or arterial occlusion in ischemic stroke, all autoregulatory mechanisms are lost since they are dependent on intracellular adenosine triphosphate reserves produced by oxygen and glucose. Poor perfusion with absence of autoregulatory processes results in passive vasomotor exhaustion of arterial vessels along with accumulation of lactic acidosis. Autoregulatory failure makes perfusion fully dependent on BP within ischemic regions to ensure some degree of forward arterial flow through affected vasculature.

Sympathomimetic activation^{51,52} from arterial occlusion, hematoma expansion, and/or development of increased ICP can be contributors to elevated BP in stroke patients.^{53,54} Large hematoma volume or large territory ischemic stroke in patients with less brain atrophy, or stroke occurring in specific territories that may compress the ventricular system or brainstem (e.g. cerebellum) all contribute to whether ICP will increase after a stroke. Clinicians must fully understand factors associated with BP

elevation to ensure appropriate patient management and optimal perfusion when increased ICP occurs. However, there are additional considerations in stroke patients that are not tied to ICP when considering optimal BP monitoring and management.

In ICH, the primary danger of precipitous BP elevation is worsening of secondary brain injury with the extension of bleeding into the ventricular and subarachnoid spaces, resulting in worsening neurologic disability.^{55,56} While it is clear that BP elevation and hematoma expansion are related, it still remains unclear whether the hematoma expansion causes BP elevation, or whether the BP elevation causes hematoma expansion; in fact, it may be that this differs on a patient to patient basis. While the current ICH guidelines recommend that BP should be controlled as quickly as possible (within two hours of ICH onset, with BP at goal within one hour),⁴³ it is also important to note that sharp spikes and large variability in BP should also be avoided. Secondary analyses of data suggest that smooth and sustained control that limits variability may reduce hematoma expansion and produce better functional outcomes,⁵² although at this time, this finding is hypothesis generating only. Despite this, the early effects of BP lowering should be evaluated in the first 24 hours by repeat non-contrast CT of the brain to assess hematoma growth, as well as frequent neurologic exams using the National Institutes of Health Stroke Scale (NIHSS)⁵⁷ to detect early neurologic deterioration.

Guidelines supporting BP management in ischemic stroke are supported by better evidence for BP lowering, despite the arbitrary selection of the BP values that originated with the NINDS rtPA Stroke Study.^{58,59} Elevated risk for sICH has been documented with excessive BP levels⁵⁹

ensuring widespread adoption of BP thresholds of 180/105 mm Hg. Similarly, guidelines supporting management of aneurysmal subarachnoid hemorrhage call for control of BP to less than 140/90 mm Hg in the period before aneurysm occlusion,⁶⁰ although use of permissive hypertension and induced hypertension may also support the management of patients with arterial vasospasm by augmenting brain perfusion following aneurysm occlusion.⁶⁰

Most compelling is the fact that guidelines are silent regarding goals for MAP, despite the knowledge that MAP significantly affects CBF,^{48,49,50,61} and despite the widespread use of NIBP monitoring which only directly measures MAP and algorithmically assigns systolic and diastolic pressures. CBF is known to decrease in the setting of both ischemic and

hemorrhagic stroke,^{62,63,64} yet few clinicians document or even consider MAP values in the management of acute stroke patients.

CONCLUSIONS

Although BP is among the most commonly monitored vital signs, the methods supporting accurate BP monitoring are often overlooked in everyday practice. Additionally, given the widespread use of NIBP devices and the importance of MAP values, further exploration is paramount to increase understanding of critical MAP thresholds that should support stroke patient management. Clinicians are encouraged to document and examine MAP values as they manage both ischemic and hemorrhagic stroke patients to further knowledge of this key parameter in relation to stroke patient outcomes.

Author Affiliations

PhD Candidate, University of Tennessee
Health Science Center, Memphis, TN.

Corresponding Author

Desiree M. Cihelka
Dmetzge1@uthsc.edu

Conflict of interest statement

The author reports no conflicts.

Funding

The author reports no grant support for this work.

REFERENCES

1. Muntner P, Shimbo D, Carey RM, et al. Measurement of Blood Pressure in Humans: A Scientific Statement From the American Heart Association. *Hypertension*. 2019;73(5):e35-e66. doi:10.1161/HYP.0000000000000087
2. Xing CY, Tarumi T, Liu J, et al. Distribution of cardiac output to the brain across the adult lifespan. *J Cereb Blood Flow Metab*. 2017;37(8):2848-2856. doi:10.1177/0271678X16676826
3. Paulson OB, Strandgaard S, Edvinsson L. Cerebral autoregulation. *Cerebrovasc Brain Metab Rev*. 1990;2(2):161-192.
4. Brain Basics: The Life and Death of a Neuron | National Institute of Neurological Disorders and Stroke. Accessed January 24, 2023. <https://www.ninds.nih.gov/health-information/public-education/brain-basics/brain-basics-life-and-death-neuron>



5. High Blood Pressure www.heart.org. Accessed January 24, 2023. <https://www.heart.org/en/health-topics/high-blood-pressure>
6. Whelton PK, Carey RM, Aronow WS, et al. 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *J Am Coll Cardiol*. 2018;71(19):e127-e248. doi:10.1016/j.jacc.2017.11.006
7. DeMers D, Wachs D. Physiology, Mean Arterial Pressure. In: *StatPearls*. StatPearls Publishing; 2022. Accessed January 24, 2023. <http://www.ncbi.nlm.nih.gov/books/NBK538226/>
8. Gaciong Z, Siński M, Lewandowski J. Blood Pressure Control and Primary Prevention of Stroke: Summary of the Recent Clinical Trial Data and Meta-Analyses. *Curr Hypertens Rep*. 2013;15(6):559-574. doi:10.1007/s11906-013-0401-0
9. Morrison CA, Carrick MM, Norman MA, et al. Hypotensive resuscitation strategy reduces transfusion requirements and severe postoperative coagulopathy in trauma patients with hemorrhagic shock: preliminary results of a randomized controlled trial. *J Trauma*. 2011;70(3):652-663. doi:10.1097/TA.0b013e31820e77ea
10. Association of Fatal and Nonfatal Cardiovascular Outcomes With 24-Hour Mean Arterial Pressure. doi:10.1161/HYPERTENSIONAHA.120.14929
11. Ono M, Brady K, Easley RB, et al. Duration and magnitude of blood pressure below cerebral autoregulation threshold during cardiopulmonary bypass is associated with major morbidity and operative mortality. *J Thorac Cardiovasc Surg*. 2014;147(1):483-489. doi:10.1016/j.jtcvs.2013.07.069
12. Kiabi FH, Soleimani A, Habibi MR. Neuroprotective Effect of Low Mean Arterial Pressure on Postoperative Cognitive Deficit Attenuated by Prolonged Coronary Artery Bypass Time: A Meta-Analysis. *Braz J Cardiovasc Surg*. 2019;34(6):739-748. doi:10.21470/1678-9741-2018-0263
13. Tsyrlin VA, Pliss MG, Kuzmenko NV. The history of blood pressure measurement: from Hales to our days. *Arter Gipertenz Arter Hypertens*. 2016;22(2):144-152. doi:10.18705/1607-419X-2016-22-2-144-152
14. Booth J. A Short History of Blood Pressure Measurement. Published online November 1977.
15. Roguin A. Scipione Riva-Rocci and the men behind the mercury sphygmomanometer: The History Behind the Mercury Sphygmomanometer. *Int J Clin Pract*. 2005;60(1):73-79. doi:10.1111/j.1742-1241.2005.00548.x
16. Doyle NM, Doyle JF, Walter EJ. The life and work of Harvey Cushing 1869–1939: A pioneer of neurosurgery. *J Intensive Care Soc*. 2017;18(2):157-158. doi:10.1177/1751143716673076
17. Estañol B, Delgado G, Borgstein J. Korotkoff Sounds - The Improbable also Occurs. *Arq Bras Cardiol*. 2013;101(5):e99-e100. doi:10.5935/abc.20130217
18. Bonnafox P. Auscultatory and oscillometric methods of ambulatory blood pressure monitoring, advantages and limits: a technical point of view. *Blood Press Monit*. 1996;1(3):181-185.
19. Pickering TG, Hall JE, Appel LJ, et al. Recommendations for Blood Pressure Measurement in Humans and Experimental Animals. *Hypertension*. 2005;45(1):142-161. doi:10.1161/01.HYP.0000150859.47929.8e

20. Kurtz TW, Griffin KA, Bidani AK, Davisson RL, Hall JE. Recommendations for Blood Pressure Measurement in Animals: Summary of an AHA Scientific Statement From the Council on High Blood Pressure Research, Professional and Public Education Subcommittee. *Arterioscler Thromb Vasc Biol.* 2005;25(3):478-479. doi:10.1161/01.ATV.0000153088.15433.8f
21. Grove M. *Mean Arterial Pressure in Acute Ischemic Stroke Study*. University of Tennessee Health Science Center; 2020. doi:10.21007/etd.cghs.2020.0511
22. Iii MR, Jr RWH, Medero R. Improved automated mean arterial blood pressure monitor with data enhancement. Published online January 14, 1987. Accessed March 9, 2023. <https://patents.google.com/patent/EP0208521A2/en>
23. Alpert BS, Quinn D, Gallick D. Oscillometric blood pressure: a review for clinicians. *J Am Soc Hypertens JASH.* 2014;8(12):930-938. doi:10.1016/j.jash.2014.08.014
24. Ramsey M. Noninvasive automatic determination of mean arterial pressure. *Med Biol Eng Comput.* 1979;17(1):11-18. doi:10.1007/BF02440948
25. Babbs CF. Oscillometric measurement of systolic and diastolic blood pressures validated in a physiologic mathematical model. *Biomed Eng OnLine.* 2012;11(1):56. doi:10.1186/1475-925X-11-56
26. Meidert AS, Saugel B. Techniques for Non-Invasive Monitoring of Arterial Blood Pressure. *Front Med.* 2018;4:231. doi:10.3389/fmed.2017.00231
27. Li Y, Wang B, Fan F, et al. Impact of atrial fibrillation on the accuracy of oscillometric blood pressure monitoring in ICU patients from a large real-world database. *J Hypertens.* 2023;41(5):838-844. doi:10.1097/HJH.0000000000003408
28. Stergiou GS, Alpert B, Mieke S, et al. A universal standard for the validation of blood pressure measuring devices: Association for the Advancement of Medical Instrumentation/European Society of Hypertension/International Organization for Standardization (AAMI/ESH/ISO) Collaboration Statement. *J Hypertens.* 2018;36(3):472-478. doi:10.1097/HJH.0000000000001634
29. Association for the Advancement of Medical instrumentation. Non-invasive sphygmomanometers- part 2: Clinical validation of automated measurement type ANSI/AAMI/ISO 81060-2:2019. Published online 2019.
30. Singer AJ, Kahn SR, Thode HC, Hollander JE. Comparison of forearm and upper arm blood pressures. *Prehosp Emerg Care.* 1999;3(2):123-126. doi:10.1080/10903129908958919
31. Naylor AJ, Sessler DI, Maheshwari K, et al. Arterial Catheters for Early Detection and Treatment of Hypotension During Major Noncardiac Surgery: A Randomized Trial. *Anesth Analg.* 2020;131(5):1540-1550. doi:10.1213/ANE.0000000000004370
32. Nguyen Y, Bora V. Arterial Pressure Monitoring. In: *StatPearls*. StatPearls Publishing; 2023. Accessed March 24, 2023. <http://www.ncbi.nlm.nih.gov/books/NBK556127/>
33. Lam S, Liu H, Jian Z, Settels J, Bohringer C. Intraoperative Invasive Blood Pressure Monitoring and the Potential Pitfalls of Invasively Measured Systolic Blood Pressure. *Cureus.* 13(8):e17610. doi:10.7759/cureus.17610
34. Weiner R, Ryan E, Yohannes-Tomicich J. Arterial Line Monitoring and Placement. In: Oropello JM, Pastores SM, Kvetan V, eds. *Critical Care*. McGraw-Hill Education; 1AD. Accessed March 24, 2023. accessanesthesiology.mhmedical.com/content.aspx?aid=1136419179

35. Lehman L, wei H, Saeed M, Talmor D, Mark R, Malhotra A. Methods of Blood Pressure Measurement in the ICU. *Crit Care Med*. 2013;41(1):34-40. doi:10.1097/CCM.0b013e318265ea46
36. McEvoy MD, Gupta R, Koepke EJ, et al. Perioperative Quality Initiative consensus statement on postoperative blood pressure, risk and outcomes for elective surgery. *Br J Anaesth*. 2019;122(5):575-586. doi:10.1016/j.bja.2019.01.019
37. Meng L, Yu W, Wang T, Zhang L, Heerdt PM, Gelb AW. Blood Pressure Targets in Perioperative Care: Provisional Considerations Based on a Comprehensive Literature Review. *Hypertension*. 2018;72(4):806-817. doi:10.1161/HYPERTENSIONAHA.118.11688
38. Saugel B, Kouz K, Meidert AS, Schulte-Uentrop L, Romagnoli S. How to measure blood pressure using an arterial catheter: a systematic 5-step approach. *Crit Care*. 2020;24(1):172. doi:10.1186/s13054-020-02859-w
39. Romagnoli S, Ricci Z, Quattrone D, et al. Accuracy of invasive arterial pressure monitoring in cardiovascular patients: an observational study. *Crit Care*. 2014;18(6):644. doi:10.1186/s13054-014-0644-4
40. Pede S, Lombardo M. [Cardiovascular risk stratification. Systolic, diastolic or pulse pressure?]. *Ital Heart J Suppl Off J Ital Fed Cardiol*. 2001;2(4):356-358.
41. Bartels K, Esper SA, Thiele RH. Blood Pressure Monitoring for the Anesthesiologist: A Practical Review. *Anesth Analg*. 2016;122(6):1866-1879. doi:10.1213/ANE.0000000000001340
42. Irwin RS, Rippe JM, eds. *Irwin and Rippe's Intensive Care Medicine*. 8. edition. Wolters Kluwer Health/Lippincott Williams & Wilkins; 2018.
43. Greenberg SM, Ziai WC, Cordonnier C, et al. 2022 Guideline for the Management of Patients With Spontaneous Intracerebral Hemorrhage: A Guideline From the American Heart Association/American Stroke Association. *Stroke*. Published online May 17, 2022;10.1161/STR.0000000000000407. doi:10.1161/STR.0000000000000407
44. Lim YH, Choi SY, Oh KW, et al. Comparison Between an Automated Device and a Manual Mercury Sphygmomanometer in an Epidemiological Survey of Hypertension Prevalence. *Am J Hypertens*. 2014;27(4):537-545. doi:10.1093/ajh/hpt100
45. Muecke S, Bersten A, Plummer J. The Mean Machine; Accurate Non-invasive Blood Pressure Measurement in the Critically Ill Patient. *J Clin Monit Comput*. 2009;23(5):283-297. doi:10.1007/s10877-009-9195-7
46. Rebesco MR, Pinkston MC, Smyrnios NA, Weisberg SN. A Comparison of Non-Invasive Blood Pressure Measurement Strategies with Intra-Arterial Measurement. *Prehospital Disaster Med*. 2020;35(5):516-523. doi:10.1017/S1049023X20000916
47. Grove MA, Paliwal M, Shearin A, et al. Manual and Oscillometric Blood Pressure in tPA-Treated Acute Ischemic Stroke: What Constitutes Agreement? *Stroke Vasc Interv Neurol*. 2023;3(4):e000711. doi:10.1161/SVIN.122.000711
48. Steiner LA, Andrews PJD. Monitoring the injured brain: ICP and CBF. *Br J Anaesth*. 2006;97(1):26-38. doi:10.1093/bja/ael110
49. Cerebral Perfusion Pressure - an overview | ScienceDirect Topics. Accessed March 7, 2023. <https://www.sciencedirect.com/topics/agricultural-and-biological-sciences/cerebral-perfusion-pressure>
50. Mount CA, M Das J. Cerebral Perfusion Pressure. In: *StatPearls*. StatPearls Publishing; 2022. Accessed January 24, 2023. <http://www.ncbi.nlm.nih.gov/books/NBK537271/>

51. Anderson CS, Huang Y, Arima H, et al. Effects of early intensive blood pressure-lowering treatment on the growth of hematoma and perihematoma edema in acute intracerebral hemorrhage: the Intensive Blood Pressure Reduction in Acute Cerebral Haemorrhage Trial (INTERACT). *Stroke* 00392499. 2010;41(2):307-312. doi:10.1161/STROKEAHA.109.561795
52. Wang X, Sandset EC, Moullaali TJ, et al. Determinants of the high admission blood pressure in mild-to-moderate acute intracerebral hemorrhage. *J Hypertens*. 2019;37(7):1463-1466. doi:10.1097/HJH.0000000000002056
53. Qureshi AI, Ezzeddine MA, Nasar A, et al. Prevalence of elevated blood pressure in 563704 adult patients with stroke presenting to the ED in the United States. *Am J Emerg Med*. 2007;25(1):32-38. doi:10.1016/j.ajem.2006.07.008
54. Kazui S, Minematsu K, Yamamoto H, Sawada T, Yamaguchi T. Predisposing Factors to Enlargement of Spontaneous Intracerebral Hematoma. *Stroke*. 1997;28(12):2370-2375. doi:10.1161/01.STR.28.12.2370
55. Davis SM, Broderick J, Hennerici M, et al. Hematoma growth is a determinant of mortality and poor outcome after intracerebral hemorrhage. *Neurology*. 2006;66(8):1175-1181. doi:10.1212/01.wnl.0000208408.98482.99
56. Hemphill JC, Farrant M, Neill TA. Prospective validation of the ICH Score for 12-month functional outcome. *Neurology*. 2009;73(14):1088-1094. doi:10.1212/WNL.0b013e3181b8b332
57. Dusenbury W, Tsivgoulis G, Chang J, et al. Validation of the National Institutes of Health Stroke Scale in Intracerebral Hemorrhage. *Stroke Vasc Interv Neurol*. 2023;3(4):e000834. doi:10.1161/SVIN.123.000834
58. Jauch EC, Saver JL, Adams HP, et al. Guidelines for the Early Management of Patients With Acute Ischemic Stroke: A Guideline for Healthcare Professionals From the American Heart Association/American Stroke Association. *Stroke*. 2013;44(3):870-947. doi:10.1161/STR.0b013e318284056a
59. Powers WJ, Rabinstein AA, Ackerson T, et al. Guidelines for the Early Management of Patients With Acute Ischemic Stroke: 2019 Update to the 2018 Guidelines for the Early Management of Acute Ischemic Stroke: A Guideline for Healthcare Professionals From the American Heart Association/American Stroke Association. *Stroke*. 2019;50(12):e344-e418. doi:10.1161/STR.0000000000000211
60. Hoh BL, Ko NU, Amin-Hanjani S, et al. 2023 Guideline for the Management of Patients With Aneurysmal Subarachnoid Hemorrhage: A Guideline From the American Heart Association/American Stroke Association. *Stroke*. 2023;54(7):e314-e370. doi:10.1161/STR.0000000000000436
61. Gobiet W, Grote W, Bock WJ. The relation between intracranial pressure, mean arterial pressure and cerebral blood flow in patients with severe head injury. *Acta Neurochir (Wien)*. 1975;32(1-2):13-24. doi:10.1007/BF01405899
62. Jovin TG, Demchuk AM, Gupta R. PATHOPHYSIOLOGY OF ACUTE ISCHEMIC STROKE: *Contin Lifelong Learn Neurol*. 2008;14:28-45. doi:10.1212/01.CON.0000275639.07451.e7
63. Lam AM. Emergency Neurological Life Support: Intracerebral Hemorrhage. :15.
64. Magid-Bernstein J, Girard R, Polster S, et al. Cerebral Hemorrhage: Pathophysiology, Treatment, and Future Directions. *Circ Res*. 2022;130(8):1204-1229. doi:10.1161/CIRCRESAHA.121.319949