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NARRATIVE REVIEW

Importance of basic hemodynamic monitoring: a narrative review

Victor Galvani Vianna Amarilla[1], Isabel Mieko Miamoto[1], Daiane Dyba[1],
João Manoel Silva Junior[2], Brenno Cardoso Gomes[1]*

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ABSTRACT

Basic hemodynamic monitoring in patient assessment and treatment is established on fundamental principles of medicine. In practice, basic monitoring consists of relatively simple actions that effectively reflect the patient's clinical condition. Bedside evaluation includes history, focused physical examination, accurate blood pressure measurement, urine output assessment, vital sign monitoring, and non-invasive evaluation of peripheral perfusion. These fundamental approaches provide crucial information for a personalized patient management strategy. Intermediate and advanced monitoring techniques, involving various technological instruments and devices, play a critical role in identifying the pathophysiological state of critical illness. However, their high cost and the invasive nature of some methods require a balanced approach. Complementary basic monitoring strategies should not be overlooked, as they can enhance or even substitute more advanced and potentially invasive monitoring techniques. Despite the advantages of advanced technology, it is essential to balance its use with fundamental bedside assessment principles. Basic hemodynamic monitoring remains pivotal in-patient care, particularly in resource-limited healthcare systems. This narrative review aims to describe key aspects of basic hemodynamic monitoring in the context of critical illness through a literature review based on electronic searches in PubMed®.

KEYWORDS

Hemodynamic monitoring; hemodynamics; monitoring, physiologic; patient care.

INTRODUCTION

In the first half of the 20th century, bedside assessment primarily consisted of clinical history and physical examination. These actions were the primary diagnostic methods in most clinical scenarios at the time. The renowned physician Sir William Osler emphasized the significance of observation, stating that it was one of the "principles of medical practice" and that "the whole art of medicine is in observation." This continuous process of observation and serial physical examination laid the foundation for what is now referred to as basic hemodynamic monitoring – continuous and integrated monitoring of physiological functions by the clinician. In the context of Intensive Care Medicine, bedside monitoring of vital functions plays a crucial role in providing essential information about the patient's cardiopulmonary function. This process not only complements but also enhances the management of critical illness[1].

More recently, with technological advancements and digitalization, there has been an increasing reliance on sophisticated imaging and laboratory tests at every

[1] Universidade Federal do Paraná, Departamento de Medicina Integrada, Curitiba, Paraná, Brasil
[2] Universidade de São Paulo, Divisão de Anestesiologia, São Paulo, São Paulo, Brasil

step of disease management. This shift has led to a gradual distancing of the clinical team from fundamental bedside actions. This trend also impacts critically ill patients, resulting in a significant decline in physical examination practices and the use of basic hemodynamic monitoring. There appears to be a current tendency among clinicians to prioritize numerical data over direct physical assessment. The decline in bedside evaluation skills raises concerns about the accuracy of clinical reasoning and the holistic understanding of patients with diverse medical conditions. Striking a balance between technological advancements and the fundamental principles of bedside assessment is essential for efficient, comprehensive, and precise patient monitoring[2,3].

The lack of healthcare resources in Brazil and the significant disparities in their distribution are widely recognized in medical and non-medical literature. These issues highlight various vulnerabilities of the country's public health system. Brazil's large population and vast geographical area contribute to resource scarcity. This compromises the healthcare system's ability to provide adequate and equitable services, including medical supplies, imaging and laboratory tests, and technological support, to the entire population[4]. Therefore, in our country, strengthening medical education in fundamental aspects of vital sign monitoring and simple bedside physiological assessment is essential. It is imperative to reclaim the core principles of medicine – the process of patient monitoring through close physician-patient interaction. It is important to remember that examining and re-examining a patient is a valuable and cost-effective resource, particularly in regions where it may be the only available tool.

To achieve this, we must understand the key aspects of basic hemodynamic monitoring in critically ill patients.

METHODS

This was a narrative review on the description and importance of basic hemodynamic monitoring. The methodology followed the standard narrative review model found in the literature[5,6]. An electronic literature search was conducted in PubMed®. A clear and specific research question guided this review: "What are the most commonly used basic hemodynamic monitoring methods in critically ill patients in intensive care units?".

The following MeSH terms were used to refine the search: "Hemodynamics", "Monitoring, Physiological", "Critical Illness", and "Intensive Care Units". These descriptors were combined with relevant keywords: "basic hemodynamic monitoring", "invasive monitoring", and "non-invasive monitoring". A PubMed filter was applied to refine the search by article type, including narrative reviews, book chapters, and original studies published in English and Portuguese between 1998 and 2022. Meta-analyses and systematic reviews were not included. This process was conducted by two physicians: an intensive care specialist and a critical care medicine resident.

DISCUSSION

Basic hemodynamic monitoring

Hemodynamic monitoring can be defined as the observation of pathophysiological parameters aimed at analyzing the quality of tissue perfusion in critically ill patients and guiding treatment to restore hemodynamic stability. Basic monitoring is typically sufficient for less complex cases, whereas advanced monitoring is reserved for more complex situations, taking into account the necessity and cost of the techniques[7,8].

The recent COVID-19 pandemic fundamentally altered the way physicians interact with patients. Due to concerns about personal safety during the pandemic, some physicians were reluctant to spend time at the patient's bedside, contributing to the decline of bedside skills and available resources[4,9]. Could this deliberate distancing from the bedside have caused harm to the monitoring of physiological aspects necessary for better patient outcomes? This question may have crossed the minds of many physicians who, during the pandemic, risked their safety to care for critically ill patients infected with the virus.

Currently, there is no consensus distinguishing what is considered basic monitoring from what is considered advanced monitoring. In general, basic methods are those with low risk because they are less invasive, are easily learned, and use simpler techniques. On the other hand, advanced methods involve longer learning curves, are not as direct and intuitive in their application, and carry higher costs and risks. Thus, basic monitoring is the initial step in any case where hemodynamic status requires assessment. It is simpler and quicker to employ, requiring less expertise or practice from the operator. Its cost is affordable, and the risks are low, making it widely applicable and less restrictive[10].

Hemodynamic monitoring plays a crucial role in the assessment and management of the patient. The auditory, visual, and tactile assessment performed by the clinician constitutes the most basic and non invasive level of this monitoring. In many cases, a targeted history, clinical examination, and basic bedside monitoring are sufficient to determine the appropriate approach for a critically ill patient. Although these elements are subjective and their precision and accuracy may vary compared to definitive diagnostic tools, they are essential for guiding the selection of appropriate hemodynamic monitoring techniques and devices. This initial approach provides a solid foundation for the continuous monitoring and management of patients, ensuring adequate and personalized care[3]. Therefore, with simple and accessible tools, frequent basic physical examination provides crucial information on the hemodynamic status of critically ill patients[11].

The understanding of the pathophysiological mechanism is the main determinant in the choice between basic and advanced monitoring. Increased awareness of the underlying etiology correlates with a more predictable clinical progression. In this way, we can resort to simpler, less invasive and more cost-effective methods for therapy management[7].

Components of Basic Monitoring: Methods considered basic are[10,12]:

a) Clinical Assessment: Medical History and General Physical Examination;

b) Systemic Blood Pressure;

c) Peripheral Perfusion;

d) Vital Signs: Pulse, temperature, and respiration;

e) Oximetry;

f) Urine Output;

g) Arterial Blood Lactate Concentration;

h) Level of Consciousness.

Medical history and general physical examination

To assess the possibility or presence of hemodynamic and clinical instability, it is crucial to pay close attention to the information that can be extracted from the patient's medical history[8]. Thus, therapeutic decisions and diagnostic misinterpretations often result from an inadequate medical history or failure to understand its components.

Access to the patient's medical history allows us, for example, to understand that borderline blood pressure may be less concerning if these values are typical for the individual, regardless of who performs the measurement. In this context, blood pressure may remain at a borderline value for hours without necessarily being associated with other signs of tissue hypoperfusion[8].

Moreover, although conducting a general physical examination may provide highly nonspecific elements common in various conditions, it reveals significant information when performed systematically and analyzed meticulously. This is especially true when the examination is repeated and interpreted in conjunction with therapeutic interventions. This is evident in daily practice when a general physical examination, viewed in isolation, seemingly does not indicate anything significant. However, when interpreted alongside the suspected etiological mechanism and medical history, it presents several possibilities, leading to differential diagnoses and/or suggesting improved therapeutic options[8].

Systemic blood pressure

Systemic blood pressure can be measured in various ways, with invasive arterial catheterization considered the gold standard. Comparatively, invasive techniques, such as measuring pressure in the brachial or axillary artery, are more reliable than measurement in the radial or ulnar arteries. Non-invasive blood pressure measurement, using auscultatory or oscillometric methods, is an interesting option for patients with less complex conditions[13].

In this context, understanding pulse pressure, defined as the difference between systolic and diastolic pressure, is relevant. Generally, in biological systems, blood perfusion is considered more favorable in the presence of pulsatile circulation. Thus, greater tissue oxygenation is achieved with higher perfusion pressure. In a hypothetical scenario involving two cases with the same mean systemic blood pressure but different pulse pressures, the patient with the higher pulse pressure would likely exhibit superior tissue perfusion[8].

Patients with hypotension typically exhibit a compensatory mechanism of increased heart rate to ensure blood perfusion in this state of imbalance. In this compensatory state, the relationship between heart rate (HR) and blood pressure (BP) is often maintained within a certain range. The Shock Index (HR (bpm) / systolic BP (mmHg)), which physiologically ranges from 0.5 to 0.7 in adults, was developed based on this concept. Early values greater than 0.7 may indicate conditions such as hypovolemic, cardiogenic, and septic shock[14].

Additionally, there is the Modified Shock Index, defined as the ratio between heart rate (HR) (bpm) and mean arterial pressure (MAP) (mmHg). In this case, values above 0.9 may detect various shock conditions early[15].

For the basic identification of hemodynamic instability, the fraction represented by shock indices is preferred over the isolated measurement of blood pressure or heart rate, as it has superior diagnostic value. This is because, under physiological conditions, the compensatory increase in heart rate often occurs in response to a decrease in blood pressure, aiming to maintain cardiac output.

It is important to emphasize that during a simple adrenergic discharge, a common occurrence in critically ill patients, both heart rate and blood pressure typically rise concurrently. In such instances, the shock index may remain relatively unchanged as long as functional reserve is maintained. It is only when organ failure occurs that the index tends to rise. Additionally, bradyarrhythmias of cardiac or secondary origin can affect the accuracy of this method[8].

Finally, it is important to emphasize that the determination of the ideal blood pressure designation and the diagnosis of a patient in a state of significant hypotension are outlined individually through a comprehensive assessment focusing on medical history and general physical examination. Medical history provides crucial clues to the etiological mechanism of the condition. Additionally, the general physical examination highlights potential organ dysfunctions and the body's responses to the specific aggressor. The physician can use this information to propose immediate interventions when symptoms or signs of organ dysfunction are present, such as global blood hypoperfusion (characterizing shock syndrome). Another critical situation arises when symptoms or signs indicate decompensation, such as the loss of physiological compensation due to exhaustion or development of pathophysiological mechanisms that override them. Thus, confirmation of organ dysfunction should raise suspicion of circulatory failure/shock. Additionally, cases of extreme or persistent tachycardia deserve attention, suggesting a compensatory response or, potentially, a pathological mechanism that impairs its compensatory action. Interventions should be immediate, and monitoring should be intensified under any of the circumstances mentioned.

Peripheral perfusion

Understanding the relationship between cutaneous perfusion (clinically evaluated) and peripheral perfusion (the fraction of blood flow effectively delivering oxygen to tissues) offers a basic method to assess organ perfusion. Frequently, simple monitoring signs are overlooked, notwithstanding their potential impact on the interpretation of hemodynamic status and the recognition of situations requiring rapid interventions[12,16].

Hemodynamic control, regardless of the tissue, results from the action of catecholamines, global metabolic control, and internal autoregulatory mechanisms. Among these determinants, the skin is significantly related to catecholamine levels, while other organs are largely influenced by the systemic metabolic rate and mediators associated with the regulation of local circulation[17,18]. Consequently, cold sweat is an indicative sign of hypoperfusion/shock, a common and early manifestation resulting from the characteristic release of catecholamines in hemodynamically unstable conditions[18].

The circulatory changes expected from variations in metabolism and the release of local mediators are less evident in the skin. Therefore, the onset of cold sweat is highly likely, as the mechanisms opposing it are attenuated in the skin. In this scenario, more accurate measurement of sweat is considered advantageous due to its early manifestation and high sensitivity for identifying severe hemodynamic changes, although its specificity is low[19].

Due to the difficulty in grading sweat, capillary refill time and the score of cutaneous livedo can provide monitoring of the temporal evolution of peripheral perfusion, helping to identify improvement or deterioration in the patient's hemodynamic status[17]. Typically, the technique for measuring capillary refill time involves observing the time it takes for the skin to return to its original color after a 4- to 6-second compression[18]. The reference value is up to 2 seconds, and the observed area is typically the fingertip or the nailbed. Mechanical or functional obstructions that interfere with the arterial vascular bed, as well as room temperature, can influence this variable. Therefore, the area being analyzed should be free of obstructions and maintained at an appropriate temperature[19].

A test revealing a capillary refill time longer than 2 seconds has high sensitivity in diagnosing significant hemodynamic alterations in hemorrhagic shock, pulmonary embolism, and sepsis. Furthermore, a prolonged capillary refill time after resuscitation in sepsis is associated with increased in-hospital mortality[20]. The manifestation is early and highly sensitive; however, its specificity is low due to interfering factors that can result in extended time without systemic hypoperfusion[21].

Regarding cutaneous livedo, as hypoperfusion worsens, the marbled and purplish appearance of the skin progresses into a reticular pattern. To assess the intensity of hypoperfusion, the boundaries of the area where livedo is visible are marked, with grading varying according to the extent of limb involvement[17].

Pulse, temperature, and respiration

The recommendations regarding the comparative and simultaneous analysis of pulse, temperature, and respiration are valuable. First, temperature, reflecting the systemic metabolic rate, correlates with pulse and respiration[22]. For example, an increase in metabolism leads to elevated temperature, respiratory rate, and pulse rate. In adults, the literature suggests that a 1 °C increase in temperature results in an increase of 8 to 10 bpm in pulse rate and 3 to 5 rpm in respiratory rate, regardless of the patient's hemodynamic status[16,18]. Disproportionate variations may indicate the involvement of hemodynamic mechanisms as a determining factor. On the other hand, a decrease in temperature/metabolism does not show a linear correlation among the variables. In hypothermia/hypometabolism, pulse and respiration often remain within the normal range until the core temperature drops significantly, leading to a marked reduction in frequency[17,22].

Due to the stringent control conditions, this monitoring is typically confined to the perioperative period. One way to overcome this limitation is by measuring the gradient between skin temperature and room temperature. Additionally, the gradient between proximal and distal temperatures in the upper limb can be measured. Some studies with small case series indicate that smaller gradients are associated with better patient outcomes. Despite advancements in understanding the importance of temperature measurement, challenges remain in its widespread application as a fundamental tool in basic hemodynamic monitoring[23].

Pulse oximetry

When reading oxygen saturation from pulse oximetry, it is essential to understand that the obtained value reflects only the arterial oxygen content. Therefore, drawing conclusions about hemodynamic determinants based solely on oxygen saturation above 90% is unreliable. This indicates that the inspiratory oxygen supply and the integrity of functional gas exchange are within an appropriate range. Only extreme cases of circulatory failure impair the hemoglobin saturation measured by pulse oximetry[6]. In other cases, when hypoxemia is observed, it is typically related to primary ventilatory issues or ventilation/perfusion mismatch[8].

Diuresis

Oliguria can be a predictable response to the etiological mechanism, often protecting the patient more than representing a risk. In hypovolemia or hypotension, oliguria is the expected response, resulting from the release of antidiuretic hormones secondary to the stimulation of pressure or flow sensors along the circulatory system[24]. To correct hypotension and hypoperfusion, the body employs a compensatory mechanism that, by reducing diuresis, allows an increase in blood volume. Once blood pressure and circulatory flow are restored, diuresis normalizes[25,26].

The maintenance of adequate urinary flow depends on central hemodynamics and renal functionality, primarily the glomerular filtration rate ensured by the rhythm of peripheral perfusion. However, the main current cause of renal failure is not purely hemodynamic but rather due to toxic mechanisms associated with the systemic inflammatory response and sepsis[27,28]. Consequently, there are cases of acute renal failure and oliguria without previously identified hypotension or hypoperfusion[29]. Even with complete hemodynamic restoration, renal function may not recover, and oliguria may persist. Additionally, due to its low oxygen extraction rate, the kidney can capture sufficient oxygen even in the presence of tissue hypoperfusion. The kidney's ability to slightly increase oxygen extraction can help prevent organ failure, even in the setting of tissue hypoperfusion[30].

On the other hand, although oliguria is a defense mechanism in a hypovolemic patient with negative consequences for the body, it is incorrect to assume that polyuria or sustained urine flow would benefit the critically ill patient. The administration of diuretics significantly differs from ensuring diuresis through the global and regional hemodynamic restoration of the kidney. Thus, even though diuretics may be used in established acute renal failure to facilitate fluid balance management, they do not improve the prognosis of renal failure or mortality[29,30].

Lactate

The presence of hyperlactatemia is associated with increased morbidity and mortality in patients, regardless of whether the etiology is aerobic or anaerobic. Elevated lactate levels, particularly at concentrations above 0.75 mmol/L, have been correlated with higher hospital mortality, suggesting that lactate measurement may aid in the early identification of critically at-risk patients[31]. In general, the aerobic mechanism may be related to the overproduction of lactate, increased cellular release, or decreased clearance due to liver/renal failure. On the other hand, the anaerobic pathway is primarily caused by hypoperfusion due to shock. In this context, the presence of hyperlactatemia alongside acidosis strongly suggests that the anaerobic mechanism is a significant contributing factor in these patients[7,32,33].

Lactate can be included in basic hemodynamic monitoring due to its easy assessment through blood gas equipment and its significance in this context. In this sense, the presence of hyperlactatemia in scenarios with difficult or questionable interpretation of hemodynamic data raises a strong suspicion of cryptic shock[32]. Additionally, a shorter time to lactate clearance may be a valuable prognostic marker, as evidenced by studies that used serial lactate measurements to guide therapy[8,34].

Level of consciousness

Changes in the level of consciousness have distinct classification levels, such as drowsiness, lethargy, obtundation, stupor, and coma. It is important to recognize and distinguish them because an altered mental status constitutes one of the diagnostic criteria for shock[35]. Furthermore, more severe presentations require immediate medical intervention, and in these cases, the clinical approach should be precise and goal-oriented.[8]

CONCLUSION

Despite the availability of more advanced hemodynamic monitoring methods, these complex techniques are not a reality in many underdeveloped countries. During the COVID-19 pandemic, several deficiencies in the care of critically ill patients in these settings were observed, primarily related to the availability of beds and lack of trained human resources and equipment[36,37]. Therefore, basic hemodynamic monitoring still holds significant importance, especially in such settings. Thus, understanding these fundamental techniques can aid in the management of critically ill patients in general wards who may require intensive care or are already admitted to these units. Additionally, hemodynamic monitoring is applicable in the perioperative period, when the anesthesiologist requires access to and mastery of the patient's hemodynamic data for the smooth progression of the surgical procedure[38].

Basic hemodynamic monitoring plays a vital role in the assessment and treatment of patients, complementing clinical evaluation. Although the use of advanced tests has reduced the emphasis on direct bedside assessment, it is important to balance technological advancements with the fundamental principles of direct evaluation. In settings with limited healthcare resources in Brazil, basic hemodynamic monitoring becomes a valuable tool. The initial bedside approach is crucial for appropriate and personalized care, providing a foundation for comprehensive and accurate hemodynamic assessment.

REFERENCES

1. Ogedegbe AO. The bedside evaluation. Ann Intern Med. 2012;156(2):170. PMid:22250158. http://doi.org/10.7326/0003-4819-156-2-201201170-00026.
2. Garibaldi BT, Russell SW. Strategies to improve bedside clinical skills teaching. Chest. 2021;160(6):2187-95. PMid:34242633. http://doi.org/10.1016/j.chest.2021.06.055.
3. Laher AE, Watermeyer MJ, Buchanan SK, Dippenaar N, Simo NCT, Motara F, et al. A review of hemodynamic monitoring techniques, methods and devices for the emergency physician. Am J Emerg Med. 2017;35(9):1335-47. PMid:28366285. http://doi.org/10.1016/j.ajem.2017.03.036.
4. Cerda AA, García LY, Rivera-Arroyo J, Riquelme A, Teixeira JP, Jakovljevic M. Comparison of the healthcare system of Chile and Brazil: strengths, inefficiencies, and expenditures. Cost Eff Resour Alloc. 2022;20(1):71. PMid:36527052. http://doi.org/10.1186/s12962-022-00405-9.
5. Green BN, Johnson CD, Adams A. Writing narrative literature reviews for peer-reviewed journals: secrets of the trade. J Chiropr Med. 2006;5(3):101-17. PMid:19674681. http://doi.org/10.1016/S0899-3467(07)60142-6.
6. Baethge C, Goldbeck-Wood S, Mertens S. SANRA: a scale for the quality assessment of narrative review articles. Res Integr Peer Rev. 2019;4(1):5. PMid:30962953. http://doi.org/10.1186/s41073-019-0064-8.
7. Cecconi M, De Backer D, Antonelli M, Beale R, Bakker J, Hofer C, et al. Consensus on circulatory shock and hemodynamic monitoring. Task force of the European Society of Intensive Care Medicine. Intensive Care Med. 2014;40(12):1795-815. PMid:25392034. http://doi.org/10.1007/s00134-014-3525-z.
8. Marik PE. Evidence-based critical care. 3rd ed. Cham: Springer International Publishing; 2015. 835 p. http://doi.org/10.1007/978-3-319-11020-2.
9. Hall AK, Nousiainen MT, Campisi P, Dagnone JD, Frank JR, Kroeker KI, et al. Training disrupted: practical tips for supporting competency-based medical education during the COVID-19 pandemic. Med Teach. 2020;42(7):756-61. PMid:32450049. http://doi.org/10.1080/0142159X.2020.1766669.
10. Suess EM, Pinsky MR. Hemodynamic monitoring for the evaluation and treatment of shock: what is the current state of the art? Semin Respir Crit Care Med. 2015;36(6):890-8. PMid:26595049. http://doi.org/10.1055/s-0035-1564874.
11. Sivakorn C, Schultz MJ, Dondorp AM. How to monitor cardiovascular function in critical illness in resource-limited settings. Curr Opin Crit Care. 2021;27(3):274-81. PMid:33899817. http://doi.org/10.1097/MCC.0000000000000830.
12. Pinto Lima AA, Silva E. Monitoring tissue perfusion in shock: from physiology to the bedside. Cham: Springer International Publishing; 2018. 206 p.
13. Meidert AS, Saugel B. Techniques for non-invasive monitoring of arterial blood pressure. Front Med. 2018;4:231. PMid:29359130. http://doi.org/10.3389/fmed.2017.00231.
14. King RW, Plewa MC, Buderer NMF, Knotts FB. Shock index as a marker for significant injury in trauma patients. Acad Emerg Med. 1996;3(11):1041-5. PMid:8922013. http://doi.org/10.1111/j.1553-2712.1996.tb03351.x.
15. Koch E, Lovett S, Nghiem T, Riggs RA, Rech MA. Shock index in the emergency department: utility and limitations. Open Access Emerg Med. 2019;11:179-99. PMid:31616192. http://doi.org/10.2147/OAEM.S178358.
16. Ait-Oufella H, Bourcier S, Alves M, Galbois A, Baudel J-L, Margetis D, et al. Alteration of skin perfusion in mottling area during septic shock. Ann Intensive Care. 2013;3(1):31. PMid:24040941. http://doi.org/10.1186/2110-5820-3-31.
17. Ferraris A, Bouisse C, Mottard N, Thiollière F, Anselin S, Piriou V, et al. Mottling score and skin temperature in septic shock: relation and impact on prognosis in ICU. PLoS One. 2018;13(8):e0202329. PMid:30114284. http://doi.org/10.1371/journal.pone.0202329.
18. Lima A, Bakker J. Clinical assessment of peripheral circulation. Curr Opin Crit Care. 2015;21(3):226-31. PMid:25827585. http://doi.org/10.1097/MCC.0000000000000194.
19. Hernández G, Ospina-Tascón GA, Damiani LP, Estenssoro E, Dubin A, Hurtado J, et al. effect of a resuscitation strategy targeting peripheral perfusion status vs serum lactate levels on 28-day mortality among patients with septic shock: The ANDROMEDA-SHOCK randomized clinical trial. JAMA. 2019;321(7):654-64. PMid:30772908. http://doi.org/10.1001/jama.2019.0071.

20. Ait-Oufella H, Bige N, Boelle PY, Pichereau C, Alves M, Bertinchamp R, et al. Capillary refill time exploration during septic shock. Intensive Care Med. 2014;40(7):958-64. PMid:24811942. http://doi.org/10.1007/s00134-014-3326-4.
21. Lima A, Jansen TC, van Bommel J, Ince C, Bakker J. The prognostic value of the subjective assessment of peripheral perfusion in critically ill patients. Crit Care Med. 2009;37(3):934-8. PMid:19237899. http://doi.org/10.1097/CCM.0b013e31819869db.
22. Rady MY, Rivers EP, Nowak RM. Resuscitation of the critically ill in the ED: responses of blood pressure, heart rate, shock index, central venous oxygen saturation, and lactate. Am J Emerg Med. 1996;14(2):218-25. PMid:8924150. http://doi.org/10.1016/S0735-6757(96)90136-9.
23. Matika R, Ibrahim M, Patwardhan A. The importance of body temperature: an anesthesiologist's perspective. Temperature (Austin). 2016;4(1):9-12. PMid:28349090. http://doi.org/10.1080/23328940.2016.1243509.
24. Ostermann M, Joannidis M. Acute kidney injury 2016: diagnosis and diagnostic workup. Crit Care. 2016;20(1):299. PMid:27670788. http://doi.org/10.1186/s13054-016-1478-z.
25. Bellomo R, Ronco C, Kellum JA, Mehta RL, Palevsky P. Acute renal failure - definition, outcome measures, animal models, fluid therapy and information technology needs: the Second International Consensus Conference of the Acute Dialysis Quality Initiative (ADQI) Group. Crit Care. 2004;8(4):R204. PMid:15312219. http://doi.org/10.1186/cc2872.
26. Bellomo R, Kellum JA, Ronco C. Acute kidney injury. Lancet. 2012;380(9843):756-66. PMid:22617274. http://doi.org/10.1016/S0140-6736(11)61454-2.
27. Angus DC, van der Poll T. Severe sepsis and septic shock. N Engl J Med. 2013;369(9):840-51. PMid:23984731. http://doi.org/10.1056/NEJMra1208623.
28. Rhodes A, Evans LE, Alhazzani W, Levy MM, Antonelli M, Ferrer R, et al. Surviving sepsis campaign: international guidelines for management of sepsis and septic shock: 2016. Intensive Care Med. 2017;43(3):304-77. PMid:28101605. http://doi.org/10.1007/s00134-017-4683-6.
29. Waxman KS, Holmes G. Renal management in the critically ill patient. Surg Clin North Am. 2012;92(6):1503-18. PMid:23153882. http://doi.org/10.1016/j.suc.2012.08.012.
30. Pakula AM, Skinner RA. Acute kidney injury in the critically Ill patient: a current review of the literature. J Intensive Care Med. 2016;31(5):319-24. PMid:25752308. http://doi.org/10.1177/0885066615575699.
31. Nichol AD, Egi M, Pettila V, Bellomo R, French C, Hart G, et al. Relative hyperlactatemia and hospital mortality in critically Ill patients: a retrospective multi-centre study. Crit Care. 2010;14(1):R25. PMid:20181242. http://doi.org/10.1186/cc8888.
32. Vincent JL, Silva AQ, Couto L Jr, Taccone FS. The value of blood lactate kinetics in critically ill patients: a systematic review. Crit Care. 2016;20(1):257. PMid:27520452. http://doi.org/10.1186/s13054-016-1403-5.
33. Boldt J. Clinical review: hemodynamic monitoring in the intensive care unit. Crit Care. 2002;6(1):52-9. PMid:11940266. http://doi.org/10.1186/cc1453.
34. Nguyen HB, Rivers EP, Knoblich BP, Bernhard P, Jacobsen G, Muzzin A, et al. Early lactate clearance is associated with improved outcome in severe sepsis and septic shock. Crit Care Med. 2004;32(8):1637-42. PMid:15286537. http://doi.org/10.1097/01.CCM.0000132904.35713.A7.
35. Vincent JL, De Backer D. Circulatory shock. N Engl J Med. 2013;369(18):1726-34. PMid:24171518. http://doi.org/10.1056/NEJMra1208943.
36. Lobo SM, Creutzfeldt CJ, Maia IS, Town JA, Amorim E, Kross EK, et al. Perceptions of critical care shortages, resource use, and provider well-being during the COVID-19 pandemic: a survey of 1,985 health care providers in Brazil. Chest. 2022;161(6):1526-42. PMid:35150658. http://doi.org/10.1016/j.chest.2022.01.057.
37. Fonseca MCM, Araújo GTB, Scorza FA, Silva PSL, Andrade TRM, Farah D, et al. Who is going to turn on the ventilators? einstein. 2021;19:eAO6211. https://doi.org/10.31744/einstein_journal/2021AO6211.
38. Bein B, Renner J. Best practice & research clinical anaesthesiology: advances in haemodynamic monitoring for the perioperative patient: perioperative cardiac output monitoring. Best Pract Res Clin Anaesthesiol. 2019;33(2):139-53. PMid:31582094. http://doi.org/10.1016/j.bpa.2019.05.008.

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Correspondence
Brenno Cardoso Gomes
Universidade Federal do Paraná, Departamento de Medicina Integrada
Rua Padre Camargo, 280, CEP 80.060-240, Curitiba, Paraná, Brasil
Fone: +55 (41) 3360-7228
brennogomes@ufpr.br