



# Systemic Arterial Pressure

# 2

Konstantin M. Lebedinskii and Yulia B. Mikhaleva

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The goal of this chapter is to discuss the clinical aspects of systemic arterial blood pressure measurement, monitoring, and management in the operating room, emergency, and intensive care settings.

K. M. Lebedinskii (✉)

Vladimir L. Vanevskii Department of Anesthesiology and Reanimatology, North-Western State Medical University named after Ilya I. Mechnikov, St. Petersburg, Russian Federation

Federal Research and Clinical Center of Intensive Care Medicine and Rehabilitology, Moscow, Russian Federation  
e-mail: [mail@lebedinski.com](mailto:mail@lebedinski.com)

Y. B. Mikhaleva

Vladimir L. Vanevskii Department of Anesthesiology and Reanimatology, North-Western State Medical University named after Ilya I. Mechnikov, St. Petersburg, Russian Federation

## 2.1 Physiological Considerations

A review of the general blood pressure physiology is described in detail in Chap. 1. Here, we would like to highlight few points, namely those specific to systemic arterial pressure and important from a practical point of view.

The value of arterial blood pressure depends upon the point of measurement, and for some extent systolic arterial pressure (SAP) in large systemic arteries increases along the distance from the left ventricle, whereas diastolic arterial pressure (DAP) decreases slightly (Figs. 1.1 and 1.2; Chap. 1). Certainly, as blood still flows unidirectionally, mean arterial pressure (MAP) in the peripheral arteries is lower than in the aorta. However, why is pulse pressure ( $PP = SAP - DAP$ ) in the *aa. radialis* or *tibialis* actually higher in

comparison with the aortic root? The best example of the explanation is probably the so-called tsunami wave: in the open ocean one can see (sometimes, but sometimes one even cannot) a very long wave of only 1 m in height, but when coming into shallow water and narrowness it becomes 30 m high. Blood flow in the arterial part of vascular bed is an oscillatory process; therefore, wave dynamics is completely applicable to the explanation [1].

It was shown that PP is proportional to stroke volume (SV). In turn, the so-called pulse wave transit time (PWTT) between the peak of the R wave on ECG and the beginning of the local pulse wave is linked with local PP by linear function:

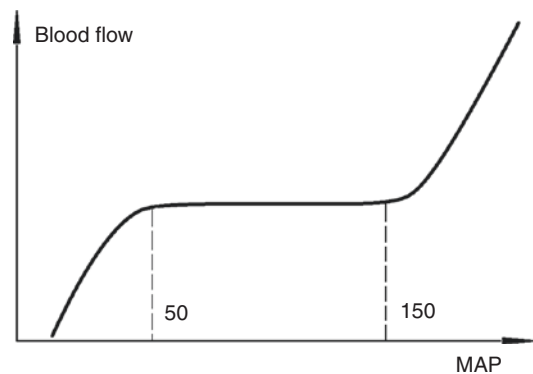
$$PP = SV / K = \alpha \times PWTT + \beta, \quad (2.1)$$

where  $K$  is the calibration coefficient for pulse wave contour analysis,  $\alpha$ —experimental constant and  $\beta$  is calculated based on PP and  $\alpha$  values [2]. Equation (2.1), giving the possibility of calculating SV from PP and PWTT, is a basis for the so-called estimated continuous cardiac output of non-invasive monitoring [3].

There is a common belief that DAP is determined mainly by systemic vascular resistance, whereas PP is an indicator of SV and, to a lesser extent, aortic compliance [4]. Although now we do have many reservations and exceptions (for example, abnormally low DAP in patients with sound aortic valve insufficiency, *etc.*), the above-mentioned simple rule may be specifically applied to the dynamic evaluation. Speaking rigorously, all four pressure values are individual for each cardiac cycle and are related primarily to the fluctuations of SV. Under the condition of stable cardiac rhythm, this pressure variability can be used for preload assessment in mechanically ventilated patients (see Chap. 15).

As for any other physiological variables, interpretation of the systemic arterial pressure values is based with certain limits on normal range, and extremes of very low and very high values, requiring immediate intervention. This physiological scale for arterial pressure seems to be associated with organ/tissue perfusion autoregulation ranges, which means an ability to maintain

stable blood flow (in milliliters per·100 g of tissue<sup>-1</sup>·min<sup>-1</sup>) despite changes in MAP level [5]. These autoregulation limits are known and can be applied not for single organ or tissue requirements but rather for most critical thresholds from the perfusion point of view. For the brain, normal autoregulation is preserved within the MAP range of approximately 50–150 mmHg whereas for the kidney it is between 75 and 170 mmHg [4]. However, in chronic hypertensive patients, these limits of flow pressure independence drift to the right (Fig. 2.1), and in a clinical setting MAP <65 mmHg persisting for 10 min is already associated with increased risk for ischemic stroke [6]. Therefore, the so-called “critical level” of systemic arterial pressure, which can be defined as MAP (or sometimes SAP) value, below which the perfusion of an organ (first of all, the kidney with its “structurally high” vascular resistance) becomes inadequate, seems to be individual and depends mainly upon the usual systemic arterial pressure level for an individual patient [7]. Whether such subcritical hypotension will lead to the ischemic organ damage or remain at the level of a critical incident depends primarily upon its extent and the time of exposure. In the retrospective study of 5127 patients, acute kidney injury (AKI) was associated with MAP <60 mmHg for 11–20 min, whereas 10 min of MAP <55 mmHg were sufficient for the same effect within 2 days of non-cardiac surgery [8]. Another study confirmed exactly the same for AKI risk thresholds for the population younger



**Fig. 2.1** Blood flow autoregulation phenomenon. MAP mean arterial pressure

than 60 years old [9]. A clear inverse relationship between hypotension depth and dangerous time exposure was also shown for ischemic stroke: in a cohort of 7457 patients who underwent cardiac surgery with cardiopulmonary bypass, a 10-min episode of MAP between 55 and 64 mmHg was associated with a 13% increased risk, whereas the same period of MAP below 55 mmHg led to a 16% increased risk of stroke [6].

Finally, systemic arterial pressure values are used for systemic vascular resistance (SVR) calculation as the main numerator constituent of the  $\Delta P/Q$  fraction. Although for monitoring purposes, vascular resistance is a calculated parameter derived from pressure and flow values, in circulation biomechanics, flow and pressure are, *vice versa*, determined by the interrelation between cardiac contractility and vascular resistance—both total and regional [10]. At the same time, from an informatics point of view, a calculated figure of SVR does not give us any additional independent data besides that given by the difference in driving pressure and the cardiac output. For example, if cardiac output is close to the upper reference limit whereas MAP is closer to the lower one, the SVR could appear to be below the lower reference limit, but... do we have any reason to consider it abnormal?

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## 2.2 Evolution of Measurement Methods

Although William Harvey (1578–1657), who discovered the circulation, used “tight and weak bandages” to compress arteries and veins separately, it was British priest, Rev. Stephen Hales (1677–1761), who measured blood pressure in the living creature for the first time: in 1727, he inserted a short brass cannula connected by a glass pipe into the carotid artery of a dying old mare. In 1876, Austro-Hungarian physician Samuel Siegfried Karl Ritter von Basch (1837–1905) invented a blood pressure gauge, based on gradual radial artery compression with a rubber bulb at the bottom of a mercury column barometer.

In 1896, Roman pediatrician Scipione Riva-Rocci used a common bike tire camera (of only 5 cm in width) with the same barometer to deter-

mine systolic pressure at the moment of pulse disappearance. In 1901, German physiologist Friedrich Daniel von Recklinghausen recognized significant overestimation of blood pressure because of too narrow cuffs and changed the standard width to 13 cm [11]. Nikolay S. Korotkoff (1874–1920), a surgeon from the military medical academy in St. Petersburg, while preparing his doctoral thesis, used a sphygmomanometer with Riva-Rocci’s cuff to stop the flow into arteriovenous aneurisms, which he confirmed by auscultation. The possibility of using turbulent flow noises for bloodless determination of both SAP and DAP values, which was suddenly discovered in 1905, was immediately developed by the famous internist Mikhail V. Yanovskii (1854–1927) and his school.

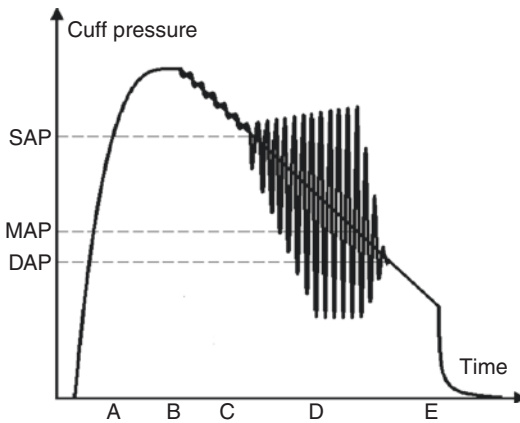
In 1931, F.D. von Recklinghausen invented the so-called oscillatory method based on cuff pressure oscillations amplitude changes during gradual pressure release; now, it is used most widely as an NIBP monitoring technique. Between 1947 and 1949, Lysle H. Peterson, Robert D. Dripps, Kenneth F. Eather, and George C. Risman from Philadelphia published several papers on direct arterial pressure measurement *via* a plastic intravascular catheter. Between 1967 and 1973, Czech physiologist Jan Peňáz proposed and patented the first principle of continuous non-invasive arterial pressure monitoring—the so-called vascular unloading (or volume clamp) technique.

As for blood pressure registration during surgery, it was the famous Harvey W. Cushing (1869–1939) who introduced in 1895 anesthesia records (“ether chart”) with temperature, heart, and respiratory rates; in 1901, on visiting the Ospedale di San Matteo (Padua), where Riva-Rocci’s sphygmomanometer had already become standard, he added blood pressure to the chart [12].

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## 2.3 Oscillatory Method

This technology, known as common NIBP (non-invasive blood pressure) monitoring, is most useful all over the world and has already been included in all the anesthesia safety standards for several decades. It is based on registration and analysis of pressure oscillations in the pneumatic



**Fig. 2.2** Principle of oscillatory method of arterial pressure measurement (see text for abbreviations and designations). *DAP* diastolic arterial pressure, *MAP* mean arterial pressure, *SAP* systolic arterial pressure

cuff placed around the extremity segment as the pressure in the cuff gradually decreases. The principle of measurement is illustrated in Fig. 2.2.

During the first phase A, a compressor (or pneumatic valve) increases the pressure in the cuff above the expected level of SAP. During phase B, pressure stabilizes, then the release valve opens and the cuff pressure begins to deflate at a rate of around  $2 \text{ mmHg}\cdot\text{s}^{-1}$  (phase C). A pressure sensor registers weak oscillations from the very beginning because until the cuff pressure exceeds SAP, the artery “pokes” into the cuff from above. When the cuff pressure drops to the level of SAP, the pulse wave penetrates under the cuff and thus the contact spot between the cuff and the artery rises rapidly, increasing the amplitude of pressure oscillations (phase D). Mean cuff pressure, which can be obtained by the damping of mechanical or digital oscillations, reflects SAP at the moment of the beginning of the amplitude rise. When the amplitude reaches its maximal value, the mean cuff pressure reflects MAP and, finally, the moment of oscillation disappearance gives a value of DAP. During the final phase E, the pressure drops to zero.

Despite their similarity at first sight, the physical difference between Korotkoff’s method and the oscillatory technique is related to the frequency range of audible sounds ( $>20 \text{ Hz}$ ) and the heart rate ( $\approx 1\text{--}2 \text{ Hz}$ ) respectively. Thus, although

providing the anesthesiologist with free time and hands, the oscillatory method is less protected from noise: in the case of any doubts or monitor faults we immediately return to “manual” (or, to be more precise, binaural) measurement. When the problem persists, DAP monitoring is indicated if possible.

Error sources and possible adverse events of “cuff technologies” are presented in Table 2.1. The most important technical rules to avoid errors and artifacts are (1) proper cuff size (its width should be 20–40% of the limb circumference), (2) proper cuff placement (the inlet of pneumatic tubing should be directly over the most palpable artery), and (3) proper pressure relief rate (around  $2 \text{ mmHg}$  per second) [4].

#### Practical Advice

To avoid inadvertent tourniquet ischemia, place the pulse oximeter sensor to the finger of the arm with the NIBP cuff. It also gives you the possibility of checking systolic arterial pressure value, at the same time looking at the current figures of decreasing cuff pressure and the plethysmography curve on the monitor screen.

## 2.4 Invasive Arterial Pressure Monitoring

Direct pressure measurement is the obvious “gold standard”, as its accuracy and precision depend only upon the properties of the measuring system, consisting of intra-arterial catheter, pressure sensor (transducer), digital monitor, pressurized flushing bag, stopcock, and connecting tubing.

As invasive pressure monitoring presumes arterial cannulation, its indications consist of two parts: (1) indications for direct pressure monitoring and (2) indications for frequent arterial blood sampling [13]. The first list includes existing or expected hemodynamic disturbances (shock, resistance to initial therapy, huge volume losses and their replacement, intracranial surgery, severe

**Table 2.1** Sources of errors and adverse events of non-invasive arterial pressure measurement and monitoring [17]

Cause of error, artifact or adverse event	Error direction, result	Preventive measures
1. Cuff is too narrow (<20% of the limb circumference)	Value overestimation	If a commonly used cuff is wrapped with tension or a large overlap, change the segment of the limb (forearm, thigh) or take a cuff of a different width
2. Cuff is too wide (>40% of the limb circumference)	Value underestimation	
3. Cuff pressure relief is too quick (>3 mmHg·s <sup>-1</sup> )	Value underestimation	Maintain the optimal cuff pressure release rate, not allowing it to be exceeded
4. Rigid tissue under the cuff (edema, shivering)	Value overestimation	Change the cuff placement, treat shivering, measure “manually”
5. Pressure is very low (SAP <60–70 mmHg)	Oscillatory method overestimates values	Find and treat the cause, turn to direct (invasive) monitoring, if possible
6. Pressure is very high (SAP >180–200 mmHg)	Oscillatory method underestimates values	
7. Shivering	Automatic measurement errors	Shivering prevention and treatment, “manual” pressure measurement (see point 4)
8. The inlet of the pneumatic tubing into the cuff is located far from the projection of the artery onto the skin	Oscillatory measurement impossibility	Place pneumatic tubing inlet exactly over the artery
9. The surgeon’s belly or the assistant’s buttocks lie on the cuff	Inability to measure or gross bidirectional errors	Think over the cuff placement in advance or... admonish colleagues throughout the surgery!
10. Compressor cycling in continuous mode due to measurement errors	Limb ischemia, compartment syndrome, thrombophlebitis, <i>n. ulnaris</i> paresthesia	Listen to the sounds of the compressor and manually interrupt measurements if necessary
11. Failure of the cuff pressure relief valve or accidental activation of static mode		Place the pulse oximeter sensor on the same limb as the cuff

SAP systolic arterial pressure

trauma, sound cardiovascular disorders or cardiac arrest, controlled hypotension or hypothermia), cardiac and/or major vascular surgery, and the inability to measure arterial pressure non-invasively (morbid obesity). The second group of indications includes clinical situations, when we need to monitor real-time blood gases, acid-base state, electrolytes or glucose levels, and blood coagulation.

As for the contraindications, all the absolute ones relate only to the site of catheterization—local skin and vascular lesions, collateral blood supply failure, *etc.*, while all the systemic states and diseases, making catheterization more risky, can be interpreted as only relative contraindications as the necessity for arterial cannulation is often absolute [14].

Choice of the artery for cannulation is usually based on technical convenience, as (1) the limb

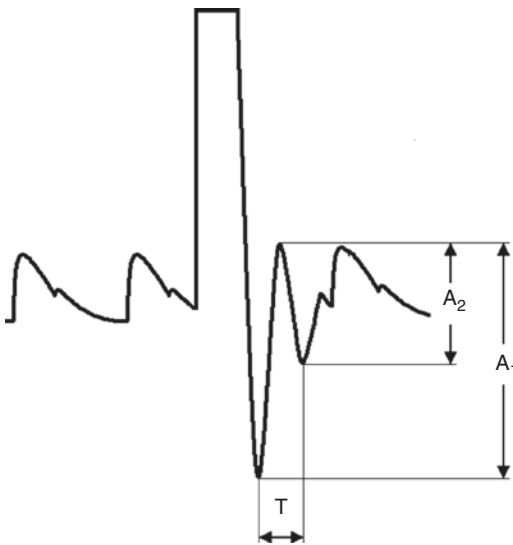
segment with the catheter should be easily immobilized and (2) all the pieces and especially connections of the hydraulic part of the monitoring system should be completely visible for the personnel at any moment [15]. Invisible system disconnection under the blanket for even 5 min could be fatal. Thus, the radial artery in the most popular choice, whereas the femoral artery is also often used, despite its proximity to the sources of infective media [16].

#### Practical Advice

To avoid unnoticed disconnection, all the parts and connections of the direct arterial line—from catheter entry under the skin to the pressurized flushing bag—should be permanently visible by the personnel.

Each exact monitoring system is characterized by its individual natural frequency  $f_c$  and damping coefficient  $\zeta$ . Because during monitoring the liquid column in the measuring system undergoes forced fluctuations under the influence of pressure fluctuations in the artery, when  $f_c$  is equal or multiple of heart rate, a resonance becomes possible with an increase in the amplitude of fluctuations and, accordingly, an overestimation of the systolic blood pressure and an underestimation of the diastolic blood pressure. As for the damping coefficient  $\zeta$ , its values range from 0 (the system does not dampen oscillations at all) to 1 (the system totally dampens oscillations and thus shows a horizontal line of MAP level). When  $\zeta$  is too low, the system can magnify the amplitude of oscillations as heart rate (HR) increases, whereas too high a  $\zeta$  value leads to a “rounded” curve with compressed amplitude. To avoid all these phenomena  $f_c$  should be not less than 10 Hz, optimally—between 10 and 20 Hz, whereas optimal  $\zeta$  seems to be around 0.4–0.6 [13].

Standard check-up procedure for pressure monitoring system is known as “pop-test” and illustrated in Fig. 2.3. To provide the test, based on a single high pressure impulse from the flushing bag, one should give a short high-pressure surge with quick closure of the flushing stopcock.



**Fig. 2.3** “Pop-test” of direct arterial pressure monitoring system.  $A_1$  and  $A_2$  adjacent curve shoulders

This leads to several damped free pressure oscillations in the measuring system, giving the possibility of determining its vibrational properties. Avoiding rather complex calculations, we may conclude that period  $T$  should be 0.1–0.05 s, whereas optimal damping between adjacent curve shoulders ( $A_1/A_2$ ) is 4–10 times. In an optimally damped system the number of free oscillations until arterial pressure curve indication restores is usually 2–3. In the real world, the vast majority of pressure monitoring systems are underdamped and have relatively low natural frequency (such as the system in Fig. 2.3); therefore the main goal is to avoid further natural frequency drop, leading to resonance as HR rises [17].

Catheters with proximal (*i.e.*, intra-arterial) pressure sensor at the tip (solid-state pressure catheters), although more expensive, are deprived of all the above-mentioned problems with natural frequency and damping intensity. The use of proximal pressure sensors, however, is mainly limited within experimental practice [18].

Possible causes of errors, artifacts, and adverse events while using a direct arterial line are summarized in Table 2.2.

#### Practical Advice

Do not remove the NIBP cuff when direct systemic arterial pressure monitoring is initiated.

It could help you to avoid too hasty activity, reflecting the highly volatile figures of “direct” arterial pressure [19].

## 2.5 Vascular Unloading (Volume Clamp) Technique

This principle provides the unique possibility of monitoring systemic arterial pressure, both continuously and non-invasively (Finapres, Finometer, Portapres, Cardiapres, and other similar devices for “continuous non-invasive arterial pressure”—CNAP). The main idea is to keep arterial wall permanently unloaded (*i.e.*, under zero transmural pressure) by means of a pneu-



**Table 2.2** Sources of errors, artifacts, and adverse events of invasive (direct) arterial pressure monitoring [17]

Cause of error, artifact or adverse event	Error direction, result	Preventive measures
1. Catheter is too thick	Artery wall injury	Correct choice of elements for assembling the measurement system; refusal to use “improvised means” for this purpose
2. Catheter is too thin	Low $f_c$ and high $\zeta$	
3. Connecting tubing is too long		
4. Connecting tubing is too compliant		
5. Connecting tubing is too short	Low $\zeta$	
6. Air bubbles in the system	Low $f_c$ and high $\zeta$	Regular efficient system flushing
7. Partial catheter occlusion with thrombus		
8. Complete catheter occlusion with thrombus	Display pressure curve interruption	Immobilization of the limb segment, catheter and the initial segment of the connecting tubing; effective sedation for severe motor restlessness
9. Catheter kinking or breaking	Low $f_c$ and high $\zeta$	
10. Plucking the catheter into the artery wall	Sudden blood pressure curve disappearance without patient’s condition changes	
11. Pressure sensor “zero drift”	Incorrect indication of blood pressure values while maintaining the proper shape of the pressure curve	Regular sensor zeroing at the level of the patient’s right atrium (in supine position—the level of the lower edge of the pectoralis major muscle in the armpit)
12. Pressure sensor dislocation in relation to the right atrium level		
13. Catheter tip whipping in the artery lumen, usually with a large difference in their calibers	Unusual shape of the curve—artifacts with the HR frequency; may resemble low $\zeta$	Slightly pulling the catheter out of the artery while evaluating the curve, and then re-fixing the catheter
14. System disconnection or huge leakage	Sudden blood pressure figures drop, curve shape distortion, massive blood loss	Careful coupling of all the system elements, exclusion of joints without threads. The whole system should be completely visible any moment
15. Shivering, chills	“Sawtooth curve”	Cause identification and treatment
16. Distal arterial approach (radial, femoral or even more distal)	Overestimation of blood pressure figures compared with those in the aorta	Know and correct or choose a more central approach (axillary or brachial)
17. Sharp vasodilation on rewarming after CPB	Underestimation of blood pressure value in the radial artery	Know and correct, impossible to prevent
18. Balloon work in intra-aortic counterpulsation	HR overestimation due to counting IABP peaks	Correction of the HR figure based on the IABP operation multiplicity
19. Tissue tension and compression of the subclavian artery on the side of the mammary artery harvesting for CABG	Falling figures and curve shape distortion in the arteries of ipsilateral arm	Contralateral arm artery choice or, for bilateral harvesting, femoral artery catheterization
20. Artery injury due to its puncture and catheterization	Thrombosis, bleeding, hematoma	Careful step-by-step execution of all the technique precautions
21. Artery injury as the system is used	Thrombosis, hematoma	See points 8, 9
22. Thromboemboli or air bubbles pumping from the catheter into the distal vascular bed	Limb ischemia	Careful hermetic assembly of the monitoring system, its regular effective flushing. If there are clear signs of a thrombosis, do not flush the system with high pressure, but immediately remove the catheter
23. Retrograde pumping of thromboemboli or air bubbles into the aorta	Embolism in the systemic circulation	

$f_c$  monitoring system natural frequency,  $\zeta$  monitoring system damping coefficient, *HR* heart rate, *CPB* cardiopulmonary bypass, *IABP* intra-aortic balloon pump, *CABG* coronary artery bypass grafting

matic cuff on a palm finger. Keeping stable zero transmural pressure is possible only when cuff pressure precisely follows arterial blood pressure, which, in turn, is provided by a digital tracing servocontroller, a driving cuff pressure control valve with closed loop feedback by the finger photoplethysmography curve. Therefore, the cuff pressure curve becomes an exact copy of the arterial blood pressure curve [20, 21].

Main limitations of the method are dependent upon baseline vascular tone (ambient temperature, emotions, exercise), cuff placement thoroughness, better measurement of MAP and DAP in comparison with SAP, *etc.* [22]. The devices based on this principle have shown acceptable accuracy both in adults [23] and in children [24] and are well-known in cardiology and sports medicine; however, their use in anesthesiology, emergency, and critical care is not common.

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## 2.6 Monitoring-Based Arterial Pressure Management

As has been well-known for many years, in contrast to alarm monitoring (for example, pulse oximetry), no method of numerical monitoring itself—without a clear uniform protocol of tactical decision making—could improve clinical outcomes [25].

What is the optimal blood pressure level? The answer evidently depends upon the patient, diagnosis, and multiple clinical features, including some special techniques, related to the surgery. There is no doubt, however, that an abnormal level of systemic arterial pressure is associated with unfavorable outcomes and, moreover, protocol-guided arterial pressure management improves outcomes [26]. Evidence- and common sense-based statements concerning systemic arterial pressure monitoring and management cited from the European Society of Intensive Care Medicine task force “Consensus on circulatory shock and hemodynamic monitoring” (2014) [7] and a brief fundamental review “Blood pressure targets in perioperative care” (2018, [26])

are summarized in Table 2.3. The main principle is personalization of optimal and acceptable systemic arterial pressure levels based on its individual baseline values.

When talking about induced controlled hypotension, especially desirable among ENT, orthopedic, and esthetic surgeons, it is very important to remember that all the considerations concerning acceptable arterial pressure limits are valid for these special settings also. Certainly, we do not now use the best traditional approaches such as cardiac output decrease with procainamide [27] or high spinal block [28], understanding that MAP but not cardiac output influences blood loss during surgery [29]. The situation has changed since the British confidential enquiry NCEPOD 1970–1982, when “controlled hypotension” was claimed to be the fourth main cause of anesthetic death and major neurological deficit [30]. However, the best modern definition of controlled hypotension, adjusted to the individual blood pressure level, is MAP decrease by 30% of the baseline values [31]—compare it with the limits marked in Table 2.3. In other words, controlled hypotension is a brilliant method, but only for patients who can survive it.

The authors of this chapter use a simple postural technique of induced hypotension, *i.e.*, head tilt for facial plastic surgery (the so-called SMAS lifting) under sevoflurane/dexmedetomidine anesthesia. It has the obvious advantages of fast and easy reversibility, the lack of an additional pharmacological burden, and thus never becomes uncontrolled [32, 33].

Almost the same words should be written about induced hypertension, which is used for some indications in neurological intensive care [34, 35] and for a final surgical hemostasis check-up. Although the latter technique is actually effective in diminishing postoperative blood loss and serious bleeding occurrence in thyroid surgery [36], its use should be approached extremely carefully and always requires both proper patient selection and reliable arterial pressure monitoring.



**Table 2.3** Statements concerning systemic arterial pressure in anesthesiology and intensive care

Statement/recommendation	GRADE level of recommendation; quality of evidence	Type of statement, [reference]
In noncardiac surgery patients, maintain systemic arterial pressure at 90–110% of baseline values	Level 1; QoE moderate (B)	Provisional consideration [26]
In noncardiac surgery patients, if baseline is low (SAP <90 mmHg, DAP <50 mmHg), maintain 100–120% of baseline values	Level 2; QoE moderate (B)	Provisional consideration [26]
In noncardiac surgery patients, if baseline is high ( $130 \leq$ SAP <160 mmHg, DAP $\geq$ 80 mmHg), maintain 80–110% of baseline values	Level 2; QoE low (C)	Provisional consideration [26]
In noncardiac surgery patients, if there is a high risk of organ ischemia or a high risk of pressure-related bleeding, maintain the upper or lower allowable ranges respectively (see above)	Level 2; QoE low (C)	Provisional consideration [26]
For cardiac surgery during CPB, maintain perfusion pressure (MAP equivalent) at 70–100 mmHg	Level 1; QoE moderate (B)	Provisional consideration [26]
For cardiac surgery during CPB, adjust perfusion pressure within the allowable range based on patient's baseline (see above)	Level 2; QoE low (C)	Provisional consideration [26]
We recommend frequent measurement of heart rate, blood pressure, body temperature, and physical examination variables (including signs of hypoperfusion, urine output and mental status) in patients with a history and clinical findings suggestive of shock	Ungraded	Best practice [7]
We recommend that the presence of arterial hypotension (defined as systolic pressure of <90 mmHg, or MAP of <65 mmHg, or decrease of $\geq$ 40 mmHg from baseline), although commonly present, should not be required to define shock	Level 1; QoE moderate (B)	Recommendation [7]
We recommend arterial and central venous catheter insertion in shock not responsive to initial therapy and/or requiring vasopressor infusion	Ungraded	Best practice [7]
We recommend individualizing the target blood pressure during shock resuscitation	Level 1; QoE moderate (B)	Recommendation [7]
We recommend initially targeting a MAP of $\geq$ 65 mmHg	Level 1; QoE low (C)	Recommendation [7]
We suggest tolerating a lower level of blood pressure in patients with uncontrolled bleeding ( <i>i.e.</i> , in patients with trauma) without severe head injury	Level 2; QoE low (C)	Recommendation [7]
We suggest a higher MAP in septic patients with a history of hypertension and in patients that show clinical improvement with higher blood pressure	Level 2; QoE moderate (B)	Recommendation [7]

CPB cardiopulmonary bypass, DAP diastolic arterial pressure, MAP mean arterial pressure; SAP systolic arterial pressure

## 2.7 Conclusion

Although from physical point of view, it is probably the simplest and oldest approach to hemodynamic monitoring, systemic arterial pressure measurement remains its absolutely essential component. Including the whole spectrum of methods from Korotkoff's sound auscultation to the most sophisticated vascular unloading technique, we cover all possible kinds of clinical situations where blood pressure has a well-proven association with out-

comes, while being rather volatile and easily controlled. Its effective and safe monitoring requires a good understanding of general blood pressure mechanics and physiology, knowledge of the principles of different techniques, details and pitfalls, and—for the invasive direct method—even certain manual skills. The current strategy of blood pressure management utilizes data of real-time monitoring and is focused on personalized choice of optimal and allowable ranges based on patient's individual baseline blood pressure values.

### Keynotes

- Systemic arterial blood pressure, after more than a century, remains one of the most important life signs and easily available circulation monitoring variables. Its significance is supported by both physiological and clinical considerations, including the blood flow autoregulation concept and a well-proven influence on outcomes.
- Despite its “technical” character and availability in comparison with heart performance, the systemic arterial pressure level itself could be a trigger for immediate intervention: so-called subcritical hypotension (usually mean arterial pressure below 65 mmHg) means that regardless of the level of cardiac output, organs with “structurally high” local vascular resistance could get time-dependent irreversible ischemic injury.
- Non-invasive systemic arterial pressure monitoring is now available in both discrete and continuous versions, which are almost equally accurate enough to be used in various clinical settings. However, in anesthesiology, emergency medicine, and critical care the need for continuous arterial pressure monitoring is usually met by an invasive direct approach.
- Direct pressure monitoring *via* intra-arterial catheter is a recognized gold standard for accuracy, precision, and practical convenience, providing the additional possibility of frequent arterial blood sampling. Indications for arterial cannulation are often imperative; therefore, absolute contraindications exist only for certain artery approaches but not for the procedure itself.
- The modern concept of systemic arterial pressure monitoring and management includes switching to invasive mode in case of any doubts and sound artifacts, and taking into consideration patient’s baseline arterial pressure level while targeting optimal and allowable figures of the parameter.

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### References

1. Avolio AP, Kuznetsova T, Heyndrickx GR, Kerkhof PLM, Li JK-J. Arterial flow, pulse pressure and pulse wave velocity in men and women at various ages. *Adv Exp Med Biol.* 2018;1065:153–68.
2. Sugo Y, Ukawa T, Takeda S, Ishihara H, Kazama T, Takeda Z. A novel continuous cardiac output monitor based on pulse wave transit time. *Conf Proc IEEE Eng Med Biol Soc.* 2010;2010:2853–6.
3. Suzuki T, Suzuki Y, Okuda J, Minoshima R, Misonoo Y, Ueda T, Kato J, Nagata H, Yamada T, Morisaki H. Cardiac output and stroke volume variation measured by the pulse wave transit time method: a comparison with an arterial pressure-based cardiac output system. *J Clin Monit Comput.* 2019;33:385–92.
4. Kam P, Power I. *Principles of physiology for the anaesthetist.* 3rd ed. CRC Press; 2015. 478 p.
5. Ackland GL, Brudney CS, Cecconi M, Ince C, Irwin MG, et al. Perioperative Quality Initiative consensus statement on the physiology of arterial blood pressure control in perioperative medicine. *Br J Anaesth.* 2019;122(5):542–51.
6. Sun LY, Chung AM, Farkouh ME, van Diepen S, Weinberger J, Bourke M, Ruel M. Defining an intraoperative hypotension threshold in association with stroke in cardiac surgery. *Anesthesiology.* 2018;129(3):440–7.
7. Cecconi M, De Backer D, Antonelli M, Beale R, Bakker J, Hofer C, Jaeschke R, Mebazaa A, Pinsky MR, Teboul JL, Vincent JL, Rhodes A. 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.
8. Sun LY, Wijeyesundera DN, Tait GA, Beattie WS. Association of intraoperative hypotension with acute kidney injury after elective noncardiac surgery. *Anesthesiology.* 2015;123(3):515–23.
9. Tang Y, Zhu C, Liu J, Wang A, Duan K, Li B, Yuan H, Zhang H, Yao M, Ouyang W. Association of intraoperative hypotension with acute kidney injury after noncardiac surgery in patients younger than 60 years old. *Kidney Blood Press Res.* 2019;44(2):211–21.
10. Hall JE. Guyton and Hall textbook of medical physiology. 12th ed. Philadelphia: Saunders Elsevier; 2011. 1091 p.
11. Albinali HHA. 4,500-year voyage: from pulse tension to hypertension. *Heart Views.* 2005;6(3):124–33.
12. Kim OJ. Experimental sciences in surgery: Harvey Cushing’s work at the turn of the twentieth century. *Korean J Med Hist.* 2006;15:49–76.
13. Gravlee GP, Martin DE, Bartels K, editors. Hensley’s practical approach to cardi thoracic anesthesia. 6th ed. Walters Kluwer; 2018. 848 p.

14. Bennett D. Arterial pressure: a personal view. In: Pinsky M, Payen D, editors. *Functional hemodynamic monitoring*. Berlin, Heidelberg, New York: Springer; 2005. p. 89–97.
15. Ragosta M. *Textbook of clinical hemodynamics*. Saunders Elsevier; 2008. 478 p.
16. Haddad F, Zeeni C, El Rassi I, Yazigi A, Madi-Jebara S, Hayeck G, Jebara V, Yazbeck P. Can femoral artery pressure monitoring be used routinely in cardiac surgery? *J Cardiothorac Vasc Anesth*. 2008;22(3):418–22.
17. Lebedinskii KM. Arterial pressure monitoring. In: Lebedinskii KM, editor. *Circulation and anesthesia*. 2nd ed. St.-Peterburg: Chelovek; 2015. p. 141–71. (In Russian).
18. Trevino RJ, Jones DL, Escobedo D, Porterfield J, Larson E, Chisholm GB, Barton A, Feldman MD. Validation of a new micro-manometer pressure sensor for cardiovascular measurements in mice. *Biomed Instrum Technol*. 2010;44(1):75–83.
19. Wax DB, Lin H-M, Leibowitz AB. Invasive and concomitant noninvasive intraoperative blood pressure monitoring: observed differences in measurements and associated therapeutic interventions. *Anesthesiology*. 2011;115:973–8.
20. Raggi EP, Sakai T. Update on finger-application-type noninvasive continuous hemodynamic monitors (CNAP and ccNexfin): physical principles, validation, and clinical use. *Semin Cardiothorac Vasc Anesth*. 2017;21(4):321–9.
21. Fortin J, Wellisch A, Maier K. CNAP—evolution of continuous non-invasive arterial blood pressure monitoring. *Biomed Tech (Berl)*. 2013;58(Suppl 1):4179.
22. Imholz BPM, Wieling W, van Montfrans GA, Wesseling KH. Fifteen years experience with finger arterial pressure monitoring: assessment of the technology. *Cardiovasc Res*. 1998;38:605–16.
23. Chin KY, Panerai RB. Comparative study of Finapres devices. *Blood Press Monit*. 2012;17(4):171–8.
24. Heeney ND, Habib F, Brar GK, Krahn G, Campbell DA, Sanatani S, Claydon VE. Validation of finger blood pressure monitoring in children. *Blood Press Monit*. 2019;24(3):137–45.
25. Lebedinskii KM, Kovalenko AN, Kurapeev IS, Karelov AE, Len'kin AI, Subbotin VV, Volkov PA, Martynov DV. Physical and physiological problems of medical monitoring. *Tech Phys*. 2020;65(9):1343–59.
26. Meng L, Yu W, Wang T, Zhang L, Heerdt PM, Gelb AW. Blood pressure targets in perioperative care. *Hypertension*. 2018;72(4):806–17.
27. Mason AA, Pelmore JF. Combined use of hexamethonium bromide and procaine amide in controlled hypotension: a preliminary report. *Br Med J*. 1953;1:250.
28. Griffith HWC, Gillies J. Thoraco-lumbar splanchnicectomy and sympathectomy, anaesthetic procedure. *Anaesthesia*. 1948;3:134–40.
29. Sivarajan M, Amory DW, Everett GB, et al. Blood pressure, not cardiac output, determines blood loss during induced hypotension. *Anesth Analg*. 1980;59:203–6.
30. Utting JE. Pitfalls in anaesthetic practice. *Br J Anaesth*. 1987;59:877–90.
31. Degoute CS. Controlled hypotension: a guide to drug choice. *Drugs*. 2007;67(7):1053–76.
32. Leigh JM. The history of controlled hypotension. *Br J Anaesth*. 1975;47(7):745–9.
33. Gillespie R, Shishani Y, Streit J, Wanner JP, McCrum C, Syed T, Haas A, Gobeze R. The safety of controlled hypotension for shoulder arthroscopy in the beach-chair position. *J Bone Joint Surg Am*. 2012;94(14):1284–90.
34. Lim TS, Hong JM, Lee JS, Shin DH, Choi JY, Huh K. Induced-hypertension in progressing lacunar infarction. *J Neurol Sci*. 2011;308(1–2):72–6.
35. Francoeur CL, Mayer SA. Management of delayed cerebral ischemia after subarachnoid hemorrhage. *Crit Care*. 2016;20(1):277.
36. Lebedinskii KM, Karelov AE, Lebedinskaia OV, Shevkulenko DA, Bestayev GG. Hemodynamic test for surgical hemostasis consistency. In: Lebedinskii KM, editor. *Circulation and anesthesia*. 2nd ed. St.-Peterburg: Chelovek; 2015. p. 551–6. (In Russian).