

Intraoperative hypotension – update on pathophysiology and clinical implications

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SUMMARY

The incidence of intraoperative hypotension is high. An evidence-based definition of intraoperative hypotension, however, is still a matter of debate. Major risk factors include patient age, ASA physical status 3 or higher, blood loss, combination of regional and general anesthesia, duration of surgery, and emergency status. The hemodynamic significance of intraoperative hypotension is related to the fact, that cerebral, renal and myocardial blood flow and its autoregulation depend on perfusion pressure. Mean arterial pressure (MAP) seems more appropriate for intraoperative hemodynamic control than systolic arterial pressure (SAP), since pulse pressure and SAP are strongly dependent on stroke volume and arterial elastance. Because of physiological considerations and several observational studies a lower limit of 60 mm Hg is generally accepted in patients without risk factors. However, in patients with arterial hypertension the lower limit for hemodynamic intervention should be set at a relative decrease in MAP $> 30\%$. Similarly, impaired autoregulation, significant arterial stenosis and specific problems associated with intraoperative beach-chair position increase the lower limit of MAP which is necessary to ensure adequate organ blood flow. Several retrospective studies comprising large patient cohorts demonstrated that intraoperative hypotension is associated with increased 1-year mortality. A causal relationship, however, has not yet been verified. Treatment of intraoperative hypotension should not only rely on vasoactive agents to control decreased systemic vascular resistance, but should also focus on other reasons, which may include hypovolemia, redistribution of blood volume, and impaired myocardial performance.

Keywords: Hypotension, perfusion pressure, cerebral ischemia, myocardial ischemia

INTRODUCTION

For decades, arterial blood pressure (ABP) has been an indispensable basic parameter in the perioperative monitoring of the cardiovascular system. This is because ABP is an easily quantifiable parameter of the circulatory system (according to Riva-Rocci, measured by oscillometrically or by using an electromechanical pressure transducer).

However, ABP is only an incomplete indicator of an adequate oxygen supply (oxygen-delivery, DO_2) as mean arterial pressure (MAP) depends not only on cardiac output (CO) - as the most important determinant of DO_2 - but also on systemic vascular resistance (SVR).

Recently increased attention has been brought to flow- and volume-related parameters of the systemic circulation, particularly in the case of perioperative optimization of high-risk patients and in critical care medicine, which allow a more direct assessment of cardiac function and DO_2 . Nevertheless, ABP has a great physiological and clinical significance in various respects.

PHYSIOLOGICAL ASPECTS

Darcy's law

The relationship between perfusion pressure (PP), flow (Q) and resistance (R) is described by Darcy's law, representing an analogy to Ohm's law for liquids:

$$\text{PP} = \text{R} \cdot \text{Q}$$

For systemic perfusion pressure (MAP - CVP; CVP = central venous pressure), cardiac output (CO) and systemic vascular resistance (SVR) applies analogously

$$\text{MAP} - \text{CVP} = \text{SVR} \cdot \text{CO} / 80,$$

where the factor 80 results from the approximation of the dimensions (conversion of the pressure of mmHg in $\text{dyn} \cdot \text{cm}^{-2}$, the conversion of CO of $\text{L} \cdot \text{min}^{-1}$ in $\text{cm}^3 \cdot \text{sec}^{-1}$. Disregarding the CVP then results in:

$$\text{MAP} \sim \text{CO} \cdot \text{SVR}$$

$$\text{CO} \sim \text{MAP} / \text{SVR}$$

The MAP is thus directly proportional to cardiac output (CO) and SVR. Conversely, cardiac output (at a given CVP) is then directly proportional to MAP and inversely proportional

to SVR. This relationship also applies analogously to the perfusion of individual organs and the respective regional vascular resistance.

It has to be considered that flow, pressure and resistance are not independent variables but are linked through various physiological regulatory circuits.

Significance of arterial pressure for the organ perfusion

General

MAP is the most important determinant of perfusion pressure and, in this respect, essential for organ perfusion. Organ specific reactive changes in vascular resistance can only provide limited compensation for fluctuations in MAP in order to keep blood flow constant:

“If each local tissue is to control its own blood flow by dilating and constricting its blood vessels, then it is essential that the arterial pressure be maintained at a pressure high enough to supply the blood demanded by the tissue” [1].

Cerebral blood flow

Due to the high oxidative metabolic activity, the resulting high O₂ demand and low ischemia tolerance, brain function is particularly dependent on adequate perfusion pressure (CPP; cerebral perfusion pressure). Under physiological conditions, the blood flow to the brain shows an effective autoregulation – cerebral blood flow is held constant within a MAP range of approximately 60 – 160 mmHg due to cerebral vasodilation and vasoconstriction.

A further reduction of the MAP may result in a shortfall below the critical cerebral O₂ supply with consecutive loss of function and structural neuronal damage. It should be noted that even though a sufficiently high cardiac output is an important requirement for the global O₂ supply, it does not guarantee an adequate organ perfusion due to limited regional autoregulation. Thus, a critical decrease in cerebral blood flow may occur due to a critical reduction of MAP even when the cardiac output is kept constant or even increased due to a greatly reduced peripheral resistance [2]. This is difficult to verify under clinical conditions, since a drop in a patient's MAP - due to changes in afterload amongst other things - is often coupled with changes in cardiac output. However, investigations during extracorporeal circulation clearly show that cerebral blood flow under cardiopulmonary bypass declines in parallel to MAP, even at a constant and sufficiently high cardiac output once a critical perfusion pressure has been undercut.

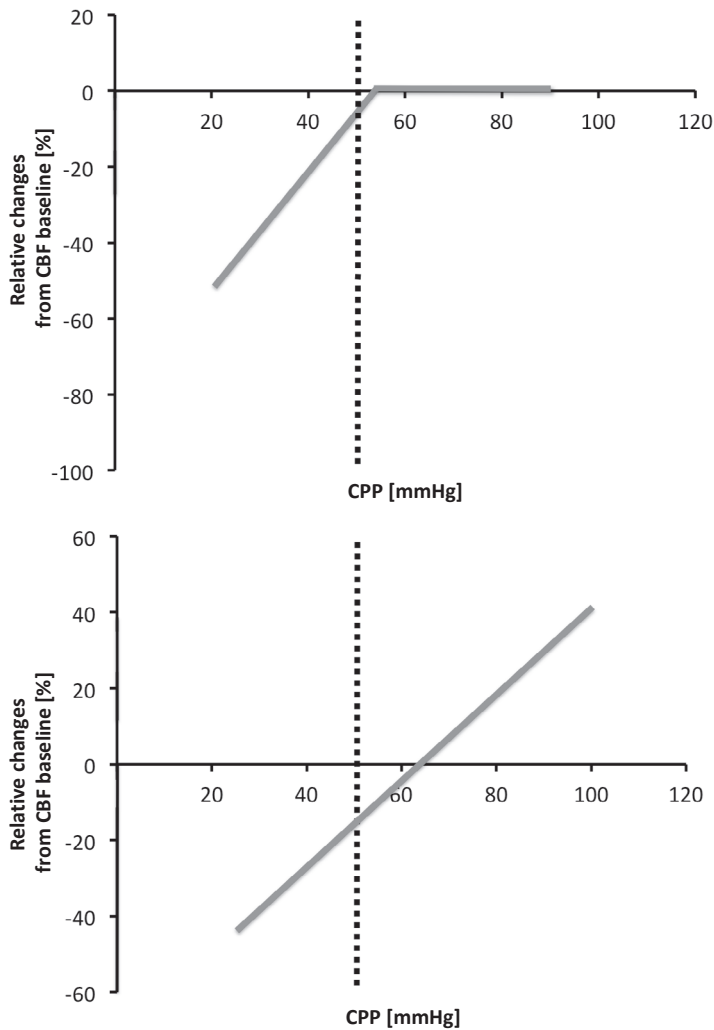


Figure 1: Relationship between cerebral blood flow (CBF) and cerebral perfusion pressure (CPP) with intact (above) and impaired autoregulation (below).

The dotted vertical lines mark the lower border of the autoregulation. Modified from Lewis et al. 2001 [2].

If MAP drops below 50 - 60 mmHg in healthy individuals, a steep drop in cerebral blood flow and a consecutive reduction in cerebral O₂ supply occurs, which can only be compensated within narrow limits by increased O₂ extraction.

Kidney perfusion

An autoregulatory mechanism also exists for the perfusion of the kidney. In a healthy patient kidney perfusion and glomerular filtration is constant if the MAP ranges from about

60 – 150 mmHg. Renal blood flow is approximately 20% of cardiac output. The largest share thereof supplies the renal cortex, which is essentially required for ultrafiltration; only 10% of blood flow is necessary for metabolic needs. Due to the kidney's particular functional anatomy, hypoperfusion quickly leads to ischemic tubular damage.

The kidney is the only organ with two capillary beds, the glomerular and peritubular capillaries, which are connected in series by intervening efferent arterioles. If there is a decrease in arterial pressure with depletion of autoregulation range, the Vasa efferentes contract significantly stronger than the Vasa afferentes, so that the peritubular vascular bed is particularly vulnerable to ischemia.

Coronary blood flow

Sufficient perfusion is also required for sufficient coronary blood flow. Coronary perfusion pressure is, however, mainly determined by mean diastolic pressure (DAP; diastolic arterial pressure) in the aorta. When perfusion pressure drops, an autoregulatory decrease in coronary vascular resistance occurs, so that the myocardial perfusion remains constant under normal circumstances. However, there is a critical coronary perfusion pressure, below which there is an exhaustion of the coronary reserve and decrease in coronary blood flow.

The critical perfusion pressure of the heart, unlike the autoregulation of cerebral and renal perfusion, will vary, depending on cardiac work and heart rate.

Importance of systolic arterial pressure

Systolic arterial pressure (SAP) is in contrast to MAP not only dependent on cardiac output and SVR, but largely on arterial elastance (EA) and stroke volume (SV). The arterial elastance in turn is determined through the interplay of impedance, compliance and resistance.

In older individuals, varying cardiac filling led to disproportionately greater changes in systolic arterial pressure as well as in changes of arterial pulse pressure (the difference between systolic and diastolic pressure) due to an increased vascular stiffening (EA) accompanied by increased ventricular stiffening (end systolic ventricular elastance, EES). These conditions should not divert attention from the importance of the MAP (or the mean DAP) for perfusion of the dependent organs [3].

The progressive decrease in the compliance of the vascular system, in particular, occurring with age leads to a progressive widening in pulse pressure (the difference between systolic and diastolic pressure).

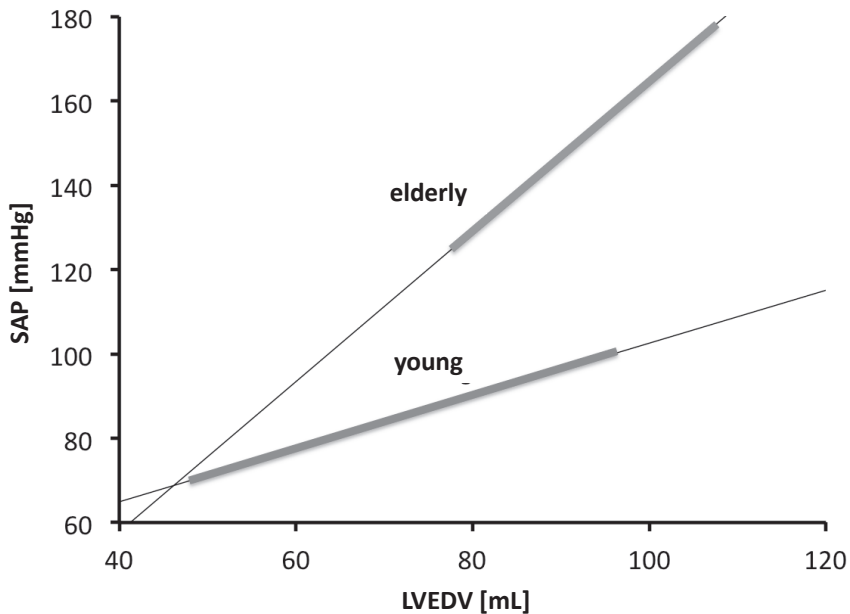


Figure 2: Example of changes in systolic arterial pressure (SAP) to alterations in preload expressed as left ventricular diastolic volume (LVEDV) in a young and in an elderly patient.

There is much greater sensitivity of systolic pressure to volume changes in the elderly patient, due to a reduced arterial compliance indicated by the steeper slope. Modified from Chen et al. 1998 [3].

PATHOPHYSIOLOGICAL ASPECTS

Impairment of autoregulatory mechanisms

The autoregulatory mechanism of the brain, in particular, can be affected by various acute and chronic disorders of homeostasis.

In general, strong cerebral vasodilators significantly disturb the brain's ability to compensate for arterial pressure fluctuations and thus may lead to a pressure-passive decrease or increase in cerebral blood flow.

Thus, a pronounced hypercapnia or hypoxia with consecutive cerebral vasodilation leads to a suppression of cerebral autoregulation [4]. In higher concentrations volatile anesthetics can also result in impaired autoregulation due to cerebral vasodilatation. Cerebral ischemia and severe traumatic brain injury are also accompanied by a disturbance of autoregulation, so that cerebral blood flow decreases passively with decreasing MAP relative to the perfusion pressure.

Chronic hypertension shifts the autoregulation thresholds on the pressure-flow diagram to the right. This leads to the assumption that arterial blood pressure drops in patients with arterial hypertension cause a decrease in cerebral blood flow at blood pressure values that would be well compensated by a healthy individual.

The kidney is also affected in patients with hypertension due to an analogous shift of the upper and lower limits of autoregulation.

Importance of vascular and heart valve stenosis

Stenoses of the afferent vessels lead to a fall in perfusion pressure for the subsequent circulation of the organs depending on the degree of stenosis. As a consequence, despite normal systemic pressures the effective arterial pressure before the arterioles is already critically reduced and thus no longer reaches autoregulatory thresholds.

As a result, even a small decrease of the MAP may lead to a critical reduction of organ perfusion and O₂ supply in affected patients. In the case of brain circulation, the extent of perfusion restriction depends greatly on the individual compensation by other brain supplying vessels due to the Circle of Willis.

In an experimental coronary stenosis, as perfusion pressure decreases, the subendocardial blood flow decreases earlier than the subepicardial poststenotically. The subendocardial autoregulation range is therefore smaller than the subepicardial.

Consequently, the subendocardial regions of the myocardium are mainly affected by a lack of O₂ supply at a low perfusion pressure. This is due to the fact that the intramyocardial pressures are higher there than in subepicardial areas and so the increased outflow pressure (see below) reduces the perfusion pressure additionally.

Patients with higher-grade aortic valve stenosis are particularly at risk in this context, as these patients are threatened in many ways by arterial hypotension.

In the presence of an aortic valve stenosis, the SV is largely fixed by the impaired ejection of the left ventricle. Thus, a drop in the SVR cannot be compensated by an increase in cardiac output.

The resulting decrease in arterial pressure leads to a critical reduction in coronary blood flow due to myocardial hypertrophy and the increased end-diastolic ventricular pressure - and by the consecutive impairment of ventricular function and further drop of the MAP to a vicious circle.

Meaning of “downstream pressure”

The perfusion pressure in the systemic and regional blood circulation is calculated - as stated above - from the difference between the upstream prevailing arterial pressure (regularly the MAP) and the downstream prevailing (postcapillary) pressure, which is determined by the (central) venous pressure (classical physiological model). Since the CVP is much smaller in relation to the MAP, and also subject to less variation, the downstream prevailing pressure is often ignored in the estimating assessment of the perfusion pressure.

This however is not true for the brain circulation due to the specific anatomy of the cranial cavity and the compressibility of cerebral bridging veins. The CPP is therefore the difference between the MAP and the intracranial pressure (ICP), which exceeds the central venous outflow pressure already under normal circumstances. In cases of intracranial hypertension ICP becomes highly important. The effective downstream pressure (EDP) may even exceed the venous pressure in other organs under certain physiological and pathophysiological conditions, since a critical closing pressure (CCP) exists, determined by the vascular tone and the tissue pressure. Especially under pathological conditions (e.g. compartment syndrome, increased intra-abdominal pressure) it is to be considered that perfusion pressure is determined by the difference between the MAP and the EDP and therefore results in perfusion pressure being significantly lower in the case of hypotension than assumed in the simplified calculation using MAP and CVP.

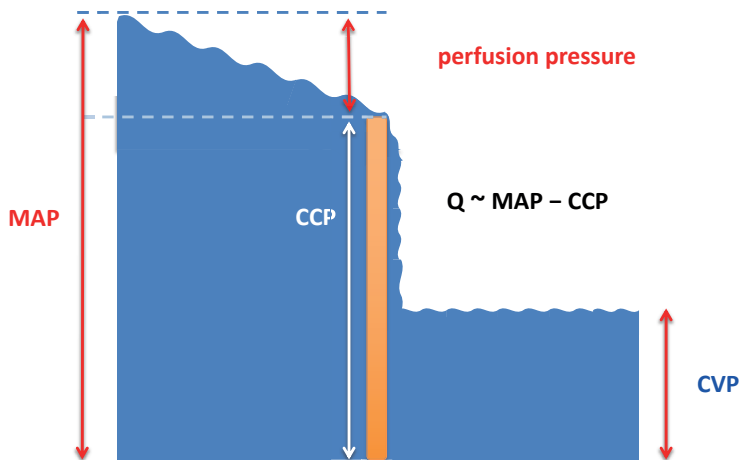


Figure 3: Waterfall of the systemic circulation.

MAP = mean arterial pressure; CCP = critical closing pressure; Q = blood flow; CVP = central venous pressure.

CLINICAL ASPECTS

Definition and incidence of intraoperative hypotension

Intraoperative hypotension may occur due to various reasons. These include the type of anesthesia, the pathology of the disease and its indication for a surgical intervention, underlying comorbidities, pharmacological effects, intraoperative blood loss, and potential logistic and human errors in the perioperative patient care.

The reported incidence in observational studies of intra- and perioperative hypotension varies greatly. This is not only due to different concepts of anesthesia and patient population, but also due to the very inconsistent and widely differing definitions of hypotension.

An analysis of 147,000 patients from the Swiss anesthesia database consisting of patients from 21 hospitals showed that despite predetermined definition (MAP reduction > 30% for > 10 min), a significant variability between the different hospitals in the incidence of hypotension exists, ranging between 0.6 and 5.2% [5]. Even greater was the influence of operational discipline, with a range from 0.3 to 12%, with the highest incidences in major thoracic-, vascular- and general surgery interventions.

A systematic literature search of Bijker et al. [6] analyzing 130 studies, showed the use of over 100 different, sometimes considerably varying definitions of hypotension. The most frequently used definitions entailed a systolic pressure <80 mm Hg, a relative decrease of the SAP by more than 20%, as well as a combination of SAP reduction <100mm Hg and a relative decline of the SAP by more than 30%. A study carried out following this literature review, applying these different definitions to the electronic anesthesia records of approximately 15,000 non-cardiac surgical patients, showed a variation in the incidence of 5 – 99% depending on the chosen definition. Very similar results were obtained in a systematic study of definitions of hypotension under spinal anesthesia. No definition of hypotension accepted by the majority could be identified in this study [7]. Even a survey conducted among members of the Society of Pediatric Anesthesia showed that no consensus on the definition of intraoperative hypotension in children could be reached [8].

The circadian fluctuations and the physiological decrease in arterial pressure during normal sleep complicate the definition of hypotension further [9]. Especially in elderly patients with atherosclerosis, a pronounced drop in habitual nightly MAP seems however to be associated with an increased cerebral ischemia risk [10].

Risk factors and predictors of hypotension under general anesthesia

In a large observational study of more than 147,000 patients the following risk factors for intraoperative hypotension were identified [5]:

- Age (odds ratio / OR 6.6 at over 75 years compared to 18 - 25 years)
- ASA classification (OR 2.6 at ASA 4 compared to ASA 1)
- Combination of regional and general anesthesia (OR 1.7, compared to general anesthesia)
- Duration of the operation (OR 10.6 at > 6 h in comparison to short procedures of 15 min)

Although a significant correlation between emergency interventions and the occurrence of hypotension was detected, this relationship was surprisingly much less pronounced (OR 1.14) than with the above-mentioned factors [5]. A similar observational study in children [8] using multivariate logistic regression analyses showed that the risk of hypotension in pediatric patients under general anesthesia in the preoperative phase was increased significantly by the following factors:

- Pre-existing hypotension,
- ASA Risk group ≥ 3 ,
- Propofol induction and
- Age

Antihypertensive premedication is also accompanied by an increase in the incidence of hypotension. However, on the morning of surgery the preoperative dose of diuretics and ACE (Angiotensin Converting Enzyme) inhibitors should be taken, if the medication is primarily for the treatment of heart insufficiency.

Overall impact on morbidity and mortality

Of crucial clinical importance for anesthetic care is the extent to which the incidence of hypotension is associated with an increase in perioperative complications and mortality. Monk et al. [11] demonstrated in a study of more than 1,000 non-cardiosurgical patients that cardiovascular comorbidity and especially the occurrence and duration of intraoperative hypotension are a significant (independent) predictors of one-year mortality after surgery under general anesthesia. Thus, a 10-minute hypotension (MAP <55 mm Hg, SAP <80 mm Hg) was already associated with an approximately 1.4-fold higher risk of mortality. Although this relationship between the duration of intraoperative hypotension and the one-year mortality could not be globally confirmed in a recent cohort study, a mortality increase in elderly patients in the event of prolonged hypotension was still observed [12]. The retrospective analysis of 147,000 patients from the Swiss anesthesia database was also able to demonstrate a strong correlation between the occurrence of intraoperative hypotension and perioperative mortality [5]. In particular, a combination

of intraoperative hypotension, low Bispectral Index (BIS) and a low volatile anesthesia concentration (presumably as an expression of a low anesthesia demand) seems to be associated with increased mortality [13].

All abovementioned observational studies have in common that a causal relationship between the occurrence of hypotension and the observed mortality increase ultimately cannot be proven. Despite correction of the data for other risk factors, it cannot be excluded that intraoperative hypotension is not the direct cause but merely an indicator of an increased perioperative risk, caused by various other factors.

Unsettling in this context, however, is that a large retrospective study of over 17,000 patients showed that early intervention with a vasopressor could reduce mortality risk almost entirely to the level of non-hypotensive patients. Thus suggesting, at least in the context of deep anesthesia and a low anesthetic concentration ("triple low"), a causal relationship [14].

In contrast, in a randomized prospective study on the effect of permissive hypotension during epidural anesthesia in 253 elderly patients no effect could be detected for either the postoperative complication rate, nor the mortality associated with the intraoperative hypotension (MAP 45 - 55 mmHg) [15]. To what extent the results of this study are specific to the performance of regional anesthesia, is - not least due to the limited number of patients - unclear. Especially, since some of the above-mentioned observational studies also included patients under regional anesthesia.

An indirect relationship between perioperative hypotension and the occurrence of cerebral ischemia can be postulated from the results of the POISE study [16] on the effect of high-dose perioperative beta-blockade. In the group of patients treated with metoprolol, increased mortality and increased incidence of stroke was associated with a significantly higher incidence of hypotensive episodes amongst other things. However, a direct causal relationship cannot be deduced because of the study design due to the complex pathophysiological processes in the context of perioperative myocardial and cerebral infarctions.

Influence of intraoperative positioning

Neurological risks

With regard to the incidence and consequences of intraoperative hypotension, elevated positioning of the upper body due to operative conditions becomes of particular importance in several respects.

There were reports on cases of severe cerebral ischemia, vision loss and cases of dissociated brain death after surgery in the sitting (beach chair) position during shoulder surgery [17, 18]. This is all the more alarming as severe neurological damage also occurred in younger patients without increased cerebrovascular risk.

Possible causes include a critical reduction in cerebral blood flow due to inadequate perfusion pressure or compression of brain-supplying vessels by hyperextension and / or rotation of the cervical spine. Recent studies on changes in regional cerebral O₂ saturation (rSO₂) using near-infrared spectroscopy (NIRS) could show that a semi-sitting position under general anesthesia goes along with a significant reduction of rSO₂ as a sign of impaired equilibrium between cerebral O₂ supply and O₂ requirements [19, 20]. In a prospective study of orthopedic patients in 80% of the cases, a relative decrease of rSO₂ by more than 20% occurred [21]. Analogously Doppler-sonographic examinations in the beach chair position showed, a significant reduction of cerebral blood flow velocity as a sign of decreased cerebral blood flow [21]. All these changes were accompanied by simultaneous impairments of MAP and imply a failure to reach cerebral autoregulation threshold.

Hemodynamic particularities

Various reasons are responsible for the often pronounced hypotension occurring in a sitting position. A major cause is the redistribution of blood volume from central to peripheral compartments with subsequent decrease of cardiac preload. This was evident when repositioning the patient into an upright position under general anesthesia, causing a decrease of intrathoracic blood volume by 15%, which resulted in a decrease of the cardiac index from 2.4 to 1.8 L/min/m² body surface area [22]. Furthermore, due to the orthostatic effects the influence of antihypertensive medication under general anesthesia is heightened [23]. During shoulder arthroscopic surgery, permissive hypotension is often specifically requested due to the improved overview [24].

The MAP in an upright / seated position is of central importance, since the influence of the hydrostatic pressure gradient between the cerebral flow path and the heart in terms of its impact on the CPP is not fully understood and is scientifically highly controversial. Essentially there are two competing hypotheses regarding the hemodynamic effects of the hydrostatic pressure difference:

- The “*siphon hypothesis*” is based on a continuous closed tube system. This leads to the fact that the effects of the hydrostatic pressure in the afferent (arterial) side are irrelevant, as they are acting in the same way on the efferent (venous) side and thus cancelling themselves out with respect to the resulting perfusion pressure. This model is comparable to a running infusion. The infusion line is raised in a loop above the level of the infusion bag, which is known to have no effect on the flow rate, as long as the outlet of the infusion line is well below the liquid level. In terms of the

practical impact, this hypothesis implies that the measured values of arterial pressure at heart level when sitting are also valid for the CPP without having to correct the non-invasively measured pressure or having to reposition the pressure sensor in invasive measurements at skull base level [25].

- The “*waterfall hypothesis*”, on the other hand, assumes a functional discontinuity in the arteriovenous vascular system. For example, as a result of Starling’s resistance and the related critical vascular closing pressure [26,27], through compression of the vessels from the outside or from serious stenoses [28]. The clinical consequences of this theory imply that the non-invasively measured arterial pressure must be corrected with respect to the hydrostatic effects or rather that the pressure transducer in invasive measurements of arterial pressure must be positioned at the height of the outer ear canal to determine a functionally relevant perfusion pressure. Although an assumed hydrostatic pressure difference of approximately 20 mmHg at normal MAP seems to be of little relevance, when measured at heart level a MAP of 55 mmHg can already be critically reduced to a CPP of 35 mmHg even when neglecting a relevant outflow pressure.

Regardless of the increasing evidence of the “waterfall” hypothesis [29], due to the existing case reports of severe neurological damage in cerebrovascular healthy patients and because of the hemodynamic consequences, the following procedure is to be recommended:

In a sitting / half-sitting position the measured MAP is to be corrected by the hydrostatic pressure difference between the atrial level and the skull base. In addition, it should be ensured that this corrected value does not fall below the lower threshold of cerebral autoregulation.

PRACTICAL CONSEQUENCES AND RECOMMENDATIONS

Based on the present retrospective and prospective studies there still does not exist an evidence-based recommendation when to intervene in the case of intraoperative hypotension, despite the immense clinical importance. This fact is the cause of the greatly differing definitions of hypotension in clinical trials. The definitions most commonly used - presumably for historical reasons and aspects of practicality - refer to the SAP. Nonetheless, it is generally recommended for physiological reasons and because of the strong dependency of pulse pressure on age to use the MAP as a target parameter instead.

For patients with no specific risk factors a MAP <60 mmHg is recommended instead of the commonly used SAP-limit <80 mm Hg as the lowest intervention limit. Especially for patients with hypertension, higher intervention limits apply, where a MAP drop > 30% applies as a relative threshold.

Apart from the question of causality, recent analyses of large patient populations showed that the statistical association between the occurrence of intraoperative hypotension and postoperative mortality significantly depend on the duration and extent of hypotension [12] (Figure 4).

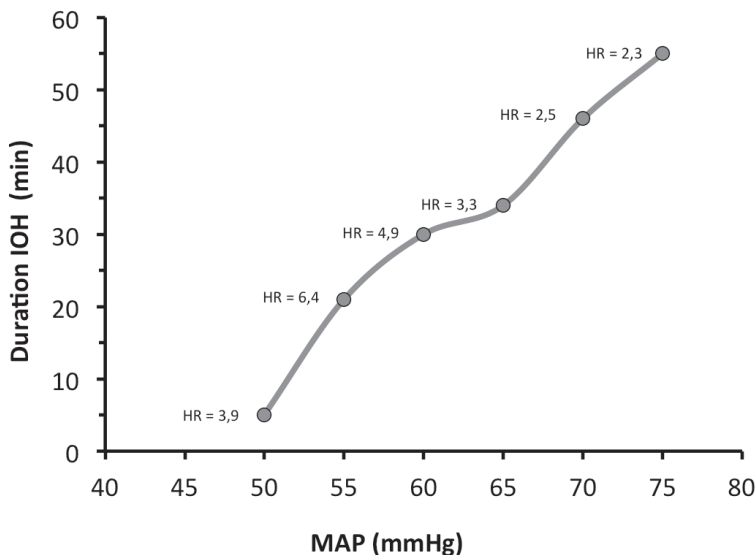


Figure 4: Relationship between arterial blood pressure and duration of an intraoperative hypotension.

Modified from Bijker et al. 2009 [12]. IOH = intraoperative Hypotension, HR = Hazard ratio, MAP = mean arterial blood pressure

If prolonged hypotension occurs, greater intervention limits for the MAP must be adhered to than when there is a momentary drop in blood pressure.

Since the perfusion pressure is calculated by the difference between MAP and the downstream pressure, the limits are only valid for a non-pathologically elevated ICP or for a normal venous outflow pressure. Further, in the case of hemodynamically significant stenoses, as well as in impaired cerebral or renal autoregulation, higher limits must also be applied.

All therapeutic interventions, in terms of treatment of intraoperative hypotension, should counteract the cause of arterial pressure drop as directly as possible.

Therefore one must primarily determine whether the hypotension is due to a predominant drop in SVR or due to a pathological decrease in cardiac output. In case of insufficient cardiac output it is important to distinguish to which extent the hemodynamic impairment is caused by hypovolemia (with subsequent decrease in pre-load) or by an impairment of contractility.

Regardless of specific pre-existing medical conditions (e.g. severely impaired heart pumping function) or procedure-related disorders in hemodynamics (e.g. blood loss, obstruction of venous return), general anesthesia has an effect on all relevant hemodynamic variables and to varying degrees simultaneously - the SVR, the preload and the pumping function of the heart are all affected.

In summary, it should be noted:

- The MAP is essentially determined by the product of cardiac output and SVR. The cardiac output in turn depends on heart rate, preload, afterload and inotropy. All of these variables can affect the MAP.
- The perfusion pressure of an organ is calculated from the difference between the upstream prevailing (arterial) and the downstream prevailing (mostly venous) pressure.
- There is a critical closing pressure of the circulation, which may determine the downstream pressure in place of CVP under certain conditions, depending on the ambient pressure (e.g. the ICP) and the vascular tone.
- Autoregulatory blood supply to the brain, kidneys and myocardium are dependent on perfusion pressure.
- Disturbances of autoregulation, pre-existing hypertension and relevant stenoses move the MAP threshold required for adequate organ perfusion upwards.
- There is no universally accepted definition of intraoperative hypotension. The MAP is more suitable than the SAP due to the highly age-dependent pulse pressure. A lower threshold of 60 mmHg is usually considered, due to physiological considerations and on the basis of observational studies, to be sufficient for patients without specific risk factors. But, especially in patients with an elevated arterial baseline, a relative decrease in MAP >30% is already determined as a limit.
- The incidence of intraoperative hypotension is high. The most important risk factors for intraoperative hypotension include the patient's age, the ASA status, the combination of general and regional anesthesia, the duration and the indication for an emergency surgery.

- Several retrospective studies on very large patient populations suggest a correlation between the occurrence of intraoperative hypotension and postoperative one-year mortality. However, direct causality cannot be established on the basis of the data.
- A sedentary or semi-sitting position (beach chair position) under general anesthesia requires special attention in many ways. Particularly due to recent case reports of severe and catastrophic neurological damage and due to prospective studies that point to a frequent impairment of cerebral blood flow. Due to the lack of conclusive evidence, and in light of the above described, it is currently recommended that the non-invasively measured MAP should be corrected by the hydrostatic pressure difference between the outer ear canal and atrial level or to position an arterial pressure transducer on skull base level. This allows the cerebral perfusion pressure to be set above the lower autoregulation limit with sufficient certainty.
- Treatment of intraoperative hypotension should not be done by means of indiscriminate use of vasoconstrictors. Interventions should be aimed primarily at the underlying causes, which may include hypervolemia and / or an impairment of cardiac pump function in addition to a decreased SVR.

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