Review Article

Systemic hypertension and non-cardiac surgery

Address for correspondence:
Dr. Satyajeet Misra, Department of Anaesthesiology, All India Institute of Medical Sciences, Bhubaneswar, Odisha, India.
E-mail: misrasatyajeet@gmail.com

Access this article online

Website: www.ijaweb.org

DOI: 10.4103/ija.IJA_377_17

Quick response code

Satyajeet Misra
Department of Anaesthesiology, All India Institute of Medical Sciences, Bhubaneswar, Odisha, India

ABSTRACT

Primary systemic hypertension affects 10%–25% of individuals presenting for surgery and anaesthesia and constitutes an important cause of cancellation of elective surgeries. Much of the fear stems from the fact that hypertension may lead to adverse perioperative outcomes. Although long-standing hypertension increases the risk of stroke, renal dysfunction or major adverse cardiovascular events, the same is usually not seen in the perioperative period if blood pressure is <180/110 mmHg and this has been the overriding theme in the recent guidelines on perioperative blood pressure management. Newer concepts include isolated systolic hypertension and pulse pressure hypertension that are increasingly used to stratify risk. The aim of this review is to focus on the adult patient with chronic primary systemic hypertension posted for elective non-cardiac surgery and outline the perioperative concerns.

Key words: Anaesthesia, complications, hypertensive emergencies, hypertensive heart disease, systemic hypertension

INTRODUCTION

Primary systemic hypertension is defined as persistent (average of 2 or more readings on 2 or more occasions) systolic blood pressure (SBP) >140 mmHg and/or diastolic blood pressure (DBP) >90 mmHg in adults, in the absence of any known precipitating cause.[1] With changing lifestyles and an increasing older population, anaesthesiologists are likely to encounter more patients with comorbid illnesses presenting for elective surgery. In the US, hypertension accounts for >30% of individuals >20 years, with increasing prevalence in older individuals (50% of individuals aged > 65 years) and a slight male:female preponderance.[2] In India, the prevalence of hypertension is 28%–32% in the urban population and 27.6% in the rural population.[3] Hypertension being mostly asymptomatic, there is an increased probability of diagnosing it during a routine pre-operative assessment. Data from western countries reveal that the incidence of hypertension in pre-operative patients ranges from 10% to 25%.[4] Secondary hypertension is when the elevations in blood pressure can be attributed to a known cause, such as pregnancy-induced hypertension, coarctation of aorta, renal artery stenosis or phaeochromocytoma, and is outside the scope of this review.

Articles used as references in this review were searched using a combination of the following terms in the MEDLINE: systemic hypertension and non-cardiac surgery; systemic hypertension and perioperative complications OR outcomes; systemic hypertension and anaesthesia OR anesthesia; hypertensive emergencies AND anaesthesia OR anesthesia. Searches were limited to articles in English.

CLASSIFICATION OF HYPERTENSION

Various stages/grades of blood pressure have been defined in several guidelines for risk stratification and management [Table 1].[1,5,6] Recent guidelines do not however consider the universal definition of blood pressure >140/90 mmHg in all adults to determine the initiation of antihypertensive therapy.[6] The 8th report of the Joint National Committee on Prevention, Detection, Evaluation and Treatment of high blood pressure suggests that antihypertensive treatment should be initiated in older individuals >60 years if the blood pressure is >150/90 mmHg with a goal

How to cite this article: Misra S. Systemic hypertension and non-cardiac surgery. Indian J Anaesth 2017;61:697-704.

Table 1: Definitions and classification of office blood pressure			
Category	**Systolic (mmHg)**		**Diastolic (mmHg)**
Optimal	<120	and	<80
Normal	120-129	and/or	80-84
High normal	130-139	and/or	85-89
Grade 1 hypertension	140-159	and/or	90-99
Grade 2 hypertension	160-179	and/or	100-109
Grade 3 hypertension	>180	and/or	>110
Isolated systolic hypertension	>140	and	<90

Blood pressure is defined by the highest level, whether systolic or diastolic. Isolated systolic hypertension should be Graded 1, 2 or 3 according to systolic values in the ranges indicated. Reproduced with permission from reference 1

of reduction to <150/90 mmHg; whereas in younger individuals aged 18–59 years, antihypertensive treatment should be initiated if the blood pressure is >140/90 mmHg with a goal to reduce the pressure below this level.[6] These recommendations have not been universally accepted by other societies such as the American Heart Association which are due to come out with their own guidelines very soon. However, most of these guidelines are for ambulatory blood pressure management in the community with very few devoted specifically to perioperative high blood pressure management.

PERIOPERATIVE CONCERNS

For the anaesthesiologist, there are two main concerns: Should the diagnosis or detection of hypertension lead to further testing and/or postponement of the planned surgery and if surgery does proceed, what would be the expected outcome of the patient's perioperative journey? Although long-standing hypertension is a major risk factor for stroke, myocardial infarction, congestive heart failure, renal and peripheral vascular disease, it is less clear whether elevated blood pressure constitutes an increased perioperative risk;[7] and yet, uncontrolled hypertension constitutes a major reason for cancellation of elective surgeries.[8] A thorough understanding of the disease process, its influence on perioperative outcomes and knowledge of best evidence and practice is imperative to facilitate decision-making and optimise the management of such patients presenting for elective surgeries.

IMPACT OF CHRONIC HYPERTENSION ON ORGAN FUNCTION

Cardiovascular system

Chronic hypertension leads to loss of arterial elasticity and compliance, and both smaller arterioles and larger conduit arteries are affected. In addition, the arterial stiffening leads to widening of the pulse pressure i.e., increased difference between the SBP and DBP, due to systolic pressure summation and loss of diastolic augmentation.[9] The augmented systolic pressures impose an increased afterload on the heart which results in compensatory hypertrophy of the myocardium to minimise the wall stress (Laplace's law). Loss of diastolic augmentation leads to decreased coronary perfusion since coronary perfusion pressure is equal to the difference of the DBP and the left ventricular end-diastolic pressure. Therefore, not only the myocardial oxygen demand is increased due to myocardial hypertrophy, but the supply is also reduced due to decreased coronary perfusion, especially the sub-endocardial perfusion which chiefly takes place during diastole.[9]

Untreated chronic hypertension may lead to myocardial ischaemia and/or infarction.[9,10] Ultimately, both diastolic and systolic performance of the left ventricle decline over time and may result in congestive heart failure.[9,10] A subset of patients with diastolic dysfunction may progress to isolated diastolic heart failure with preserved left ventricular ejection fraction. Even asymptomatic diastolic dysfunction is associated with a greater risk for adverse cardiovascular events following high-risk surgery.[9] Hypertension is also associated with diabetes, dyslipidaemia and obesity which are known risk factors for the development of coronary artery disease.[10]

Cerebrovascular system

Hypertension is a risk factor for ischaemic and haemorrhagic brain injury.[2] Abrupt increases in the blood pressure can lead to stroke due to intracerebral bleed. There may be luminal narrowing of carotid arteries due to atherosclerosis which may lead to flow insufficiency. In addition, because the auto-regulation shifts to the right in hypertensives, any degree of hypotension would reduce the cerebral blood flow leading to worsening of cerebral ischaemia. Studies have shown a history of stroke to be a predictor of adverse perioperative cardiovascular events.[11]

Renal system

Chronic renal insufficiency is a common sequelae of hypertension and leads to a decrease in the performance of kidneys. In the revised cardiac risk index, a pre-operative serum creatinine >2.0 mg/dl is identified as an independent factor that predicts increased cardiovascular risk.[12]

DOES HYPERTENSION INCREASE THE PERIOPERATIVE CARDIOVASCULAR RISK IN A PATIENT UNDERGOING SURGERY?

Given the fact that there is a linear association between hypertension and cardiovascular risk in the community, the same association should be expected to naturally extrapolate to the perioperative scenario, more so, since there are profound haemodynamic perturbations due to the stresses of anaesthesia and surgery. Surprisingly, however, there is no clear evidence that hypertension, in itself, constitutes a major risk factor for adverse perioperative cardiac events or stroke in the adult population undergoing elective non-cardiac surgery.

Howell *et al*.[7] performed a meta-analysis to evaluate the effect of hypertension on composite 30-day perioperative adverse cardiovascular events following surgery. Although the odds ratio for an adverse cardiovascular event in the analysis was 1.31 which was statistically significant, more importantly, this was not deemed to be a clinically significant finding. The findings were further tempered by the fact that there was much heterogeneity of the included studies. Thus, the authors concluded that there is very little evidence of admission blood pressures <180/110 mmHg causing any adverse perioperative complications. In other words, there is little benefit to be obtained by deferring or cancelling elective surgeries if the blood pressure is <180/110 mmHg.

There appears to be a small increase in the incidence of perioperative major cardiovascular adverse events in the presence of hypertension with organ damage, or with blood pressure >180/110 mmHg, but it is not clear whether postponing surgery to reduce blood pressure reduces the rate of this complication.[7] Weksler *et al*.[13] studied 989 treated chronic hypertensives scheduled for elective non-cardiac surgery, with DBP of 110–130 mmHg on day of surgery, but without any evidence of target organ damage or disease such as previous myocardial infarction or history of coronary revascularisation, unstable or severe angina pectoris, left ventricular hypertrophy, renal failure, aortic stenosis, pregnancy-induced hypertension, any active cardiac conditions or stroke.

Patients were randomised into control and treatment arms; the control group had their surgery postponed and remained in hospital to optimise blood pressures before surgery whereas the treatment group received 10 mg of intranasal nifedipine and proceeded to surgery. The frequency of perioperative hypotension, hypertension, brady- and tachy-arrhythmias was similar between the two groups, and there was no cardiovascular or neurological complication in either group.[13] Although the study drew several criticisms including absence of blinding, prolonged study duration (>9 years) and no evaluation of systolic hypertension, it implies that patients without evidence of significant co-existing cardiovascular disease can be taken up for surgery even with elevated blood pressures on the day of surgery.

The American College of Cardiology and the American Heart Association list 'uncontrolled systemic hypertension' as a minor predictor that has not been shown to independently increase perioperative risk.[14] Similarly, recent guidelines from the Association of Anaesthetists of Great Britain and Ireland and the British Hypertension Society state that in the absence of organ damage, blood pressure <180/110 mmHg does not warrant cancellation or deferment of elective cases in an attempt to optimise the blood pressure.[15] The recommendations of the guidelines are as follows:[15]

a. If the documented blood pressure in primary care is <160/100 mmHg with or without optimal antihypertensive treatment in the last 1 year, then further measurements and assessments need not be performed in the pre-anaesthetic clinic (PAC)
b. In case no documented blood pressure readings are available, then blood pressure can be measured in the PAC. Any measured blood pressure <180/110 mmHg without evidence of organ damage can be cleared for surgery without the need for further assessment. Evidence of organ damage includes electrocardiography (ECG) changes, a history of transient ischaemic attacks and/or stroke or raised serum creatinine. Additional testing is rarely required (e.g., echocardiography) unless the patient is undergoing a high-risk surgery such as vascular surgery
c. The higher allowable blood pressure measured in PAC is because many patients can develop 'white coat' hypertension in stressful surroundings. In addition, it takes years and decades of blood pressure control to reduce target organ damage and there is no evidence that acute perioperative reduction of blood pressure confers any advantage in reduction of adverse cardiovascular events beyond 1 month in primary care.

The guidelines are less clear as to what to do with patients with blood pressures of >180/110 mmHg or those with evidence of organ damage, obviously because there is a paucity of studies exploring such high blood pressures and perioperative outcomes. The guidelines suggest that due to limited evidence, the decision to proceed with surgery in this subset of patients should look at other factors such as associated comorbidities, functional class of the patient and urgency of the surgery.[15]

Much of the thinking that hypertension is associated with adverse perioperative outcomes comes from a study by Prys-Roberts *et al*.,[16] wherein they demonstrated increased cardiovascular lability and greater risk of perioperative myocardial ischaemia in patients with poorly controlled hypertension. It is important to understand that most of the patients in their study had blood pressure consistent with Stage 3 hypertension. In fact, hypertension during that period was classified by virtue of DBP i.e., DBP >95 mmHg. Normotensive patients in the Prys-Roberts study,[16] would currently fit into Stage 1 and 2 hypertension. Studies in Stage 1 and 2 hypertensive patients show that there is no association between admission blood pressure and perioperative cardiac risk,[17,18] and this is the spectrum of patients that anaesthesiologists are mostly going to encounter in their practice.

An important sub-type of primary hypertension is isolated systolic hypertension. Isolated systolic hypertension is defined as SBP >140 mmHg with DBP <90 mmHg, in adults, in the absence of any other factors and is the most common sub-type of elevated blood pressure in >2/3rd of individuals >50 years.[1,6] DBP plateaus off by the 5th decade, and elevated SBP or a wide pulse pressure >80 mmHg may be more important risk factors for the development of adverse cardiovascular outcomes, including congestive cardiac failure, higher incidence of fatal and non-fatal strokes, coronary artery disease and renal dysfunction.[7,9,19]

Primary hypertension must be differentiated from 'white coat hypertension', which is a physician measured blood pressure of >140/90 mmHg when in fact the individual is normotensive (average blood pressures <135/85 mmHg) and is due to environmental stress.[20] An increased incidence of silent myocardial ischaemia has been seen in patients with 'white coat hypertension' and is thought to justify treatment with antihypertensive medications,[21,22] though other studies have shown that the long-term rate of complications in patients with 'white coat hypertension' is no different than that of normotensives.[23]

PRE-OPERATIVE EVALUATION

Given the fact that hypertension may be mostly silent and first detected in the PAC, a blood pressure of <180/110 mmHg in a patient scheduled for non-cardiac surgery and without evidence of organ damage should not constitute a ground for referral, postponement or cancellation of same-day surgery. If investigations such as ECG or serum creatinine are not available, it may be prudent to consider obtaining and evaluating the same before proceeding for surgery.

If the pre-anaesthetic check-up occurs a few days before planned surgery, the primary detection of raised blood pressure (>140/90 mmHg) represents a unique opportunity to initiate antihypertensive treatment in this subset of patients, though it is not clear whether such short-term treatment confers any advantage in reducing perioperative adverse cardiovascular events. The need for advanced cardiac testing would probably be considered in the presence of poor or unknown functional class, risk posed by surgery or presence of associated comorbidities (diabetes mellitus, renal dysfunction), which increase the risk for coronary artery disease.

The mainstay of treatment is antihypertensive drugs, which apart from reducing blood pressure also induce reverse remodelling of the left ventricle, such as regression of left ventricular hypertrophy, and confer long-term survival benefits.[24] The main class of drugs include angiotensin-converting enzyme (ACE) inhibitors, angiotensin receptor blockers (ARBs), calcium channel blockers (CCBs), diuretics and β-blockers.

Guidelines exist on how to initiate and maintain antihypertensive therapy, and interested readers may refer these guidelines for a more detailed description.[24] Briefly, patients <55 years should be started on ACE inhibitors or a low-cost ARB (if ACE inhibitor is not tolerated or contraindicated).[24] Combination of these two agents should be avoided. The much-feared risks of intraoperative hypotension may be more prevalent in patients with heart failure and those receiving combination therapies with other antihypertensives,[25] and current literature suggests continuing ACE inhibitors even perioperatively, especially if started for hypertension.[25-27]

Patients >55 years should be offered a calcium channel blockers (CCB) or a thiazide diuretic if CCBs are not tolerated (e.g., oedema, heart failure or at risk for heart failure). CCBs can be safely continued up to the morning of surgery without significant risk of hypotension.[24] Caution should be exercised, however, in patients who have heart failure or are hypovolaemic.

Diuretics may also be continued into the perioperative period, especially in patients who are diuretic dependent (e.g., heart failure).[24] Patients on diuretics may have hypokalaemia. Correction of potassium should be attempted over 24–48 h as rapid correction can precipitate arrhythmias due to increased transmembrane potassium gradients.[28]

β-blockers are no more the preferred initial therapy for hypertension and are mainly reserved for younger patients in whom there is intolerance or contraindication to ACE inhibitors or ARBs, in women of child-bearing potential, or in those patients with evidence of increased sympathetic drive.[24] There is increased recognition that much of the evidence for cardioprotection with β-blockers may have been overstated since it was mainly seen in high-risk patients undergoing vascular surgery[29] and also possibly due to data fabrication.[30] Results from the POISE trial showed that though myocardial protection was better with β-blockers, there was a greater incidence of all-cause mortality and stroke if this was started acutely perioperatively.[31] This may be due to the fact that the hypotensive effect of untitrated doses might be worsened due to anaesthesia, blood loss and profound inflammation that occur during surgery. Furthermore, different β-blockers behave differently as was demonstrated in the COMET trial where mortality was reduced with carvedilol compared with metoprolol in patients with chronic heart failure.[32]

ANAESTHETIC MANAGEMENT OF THE HYPERTENSIVE PATIENT

Haemodynamic swings are more common and exaggerated in hypertensive patients as compared to normotensives. Rightward shift of auto-regulation in hypertensive patients means that organ perfusion occurs at higher mean arterial pressures as compared to normotensives, and thus, intraoperative hypotension leads to hypoperfusion and target organ damage during hypotension.[9] A retrospective study has found that intraoperative hypotension, but not hypertension, is associated with a higher mortality in hypertensive patients undergoing non-cardiac surgery.[33] Even short periods of hypotension (mean arterial pressure <55 mmHg) have been associated with myocardial and renal injury after non-cardiac surgery.[34] This is not to imply that hypertensive surges should not be treated as Charlson *et al.*,[35] have shown that hypertensive patients and diabetic patients who had a cumulative 1 hour decrease in mean arterial pressure >20 mmHg or <1 hour decrease of >20 mmHg and >15 minutes increase of >20 mmHg were at greatest risk for post-operative adverse events. Common causes of perioperative hypo and hypertension are listed [Table 2].[36]

No anaesthetic is superior to another though sevoflurane has been reported to confer cardioprotection and better haemodynamic stability as compared to propofol. It is important to maintain an adequate depth of anaesthesia with monitoring such as bispectral index or end-tidal minimum anaesthetic concentration that allows appropriate titration of anaesthetic agents. Similarly, for spinal/epidural anaesthesia, one must avoid precipitous and sudden fall of blood pressure. In general, since the lower limit of safe blood pressure in hypertensive patients is unknown, it is advisable to keep the blood pressure within 20%–30% of baseline values.[37]

Table 2: Common causes of perioperative hypo and hypertension

Haemodynamic changes	Causes
Hypotension	Systemic vasodilatation (general anaesthesia)
	Sympathetic blockade (spinal/epidural anaesthesia)
	Hypovolaemia
	Blood loss
	Mechanical ventilation
	Drugs (Angiotensin receptor blockers)
	Arrhythmias
	Acute coronary events
	Pulmonary thromboembolism (high risk surgery for pulmonary thromboembolism and/or patient predisposing factors)
Hypertension	Laryngoscopy and intubation
	Surgical stimulus
	Inadequate plane of anaesthesia and/or analgesia
	Hypothermia
	Hypervolaemia
	Reversal and recovery
	Hypoxia (postoperative)
	Indaquate analgesia (postoperative)
	Full bladder (postoperative)

Although not specifically evaluated in hypertensive patients, where possible, supraglottic airway devices such as laryngeal mask airway and its prototypes may be used because of less haemodynamic perturbations compared to laryngoscopy and intubation. Hypertensive surges more >20% from baseline are associated with adverse outcomes[15] and should be urgently treated with the goal of blood pressure reduction. Initial management should be to increase the depth of anaesthesia and give additional analgesics; however, if this fails to resolve the hypertensive surges, then drugs that modify the vascular system may be used to reduce the blood pressure [Table 3],[36,38] with the goal being not >20%–25% reduction within the

Table 3: Drugs used for management of hypertensive surges/emergencies

Drug	Class	Intravenous dose	Onset of action	Duration of action	Adverse effects	Contraindications
Esmolol	Cardio-selective β_1-receptor blocker	250-500 µg/kg bolus dose over one min followed by 50-100 µg/kg/min for 4 min; repeat boluses for further crises and increase maximum infusion dose to 300 µg/kg/min	2-10 min	10-30 min	Unopposed β-blockade may lead to α-storm	Higher degrees of heart block Use with caution in asthmatics/COPD
Labetalol	Combined α_1 and non-selective β-receptor blocker; blood pressure reductions are achieved primarily by β-blockade since the α:β activity is 1:7	Loading dose 20 mg; if crises not controlled then 20-80 mg bolus (every 10 min) or alternatively 2 mg/min infusion	5-10 min	Single bolus 2-4 min; repeated bolus or infusion 2-6 h	Nausea Angioedema (rare)	Asthmatics COPD Higher degrees of heart block
Clevidipine	Dihydropyridine type of CCB; highly selective for vascular smooth muscles	1-2 mg/h Remaining solution should be discarded after 24 h as it promotes bacterial growth; maximum infusion rate is 32 mg/h	2-4 min	5-15 min	Non-specific	Allergic to soya or egg products Disorders of lipid metabolism such as pancreatitis, lipoid nephrosis
Nicardipine	Dihydropyridine type of CCB; highly selective for vascular smooth muscles	5 mg/h; increase by 2.5 mg/h every 5 min to maximum of 30 mg/h	5-15 min	4-6 h	Non-specific	Severe aortic stenosis
Hydralazine	Peripheral vasodilator	Initial dose is 10 mg slow intravenous bolus, every 4-6 h as required; bolus doses should not exceed 20 mg	10-30 min	2-6 h	Vascular collapse Peripheral neuropathy Thrombocytopaenia Volume overload	CAD Rheumatic MS SLE
Sodium nitroprusside	Nitric oxide donor; acts on both arterial and venous smooth muscles	0.3-0.5 µg/kg/min; avoid doses >2 µg/kg/min	Immediate	2-3 min	Cyanide toxicity Chromaturia (red urine) Erythema	Raised ICP MI
Nitroglycerin	Venodilator	5 µg/min up to maximum of 20 µg/min	2-5 min	5-10 min	Headache Methaemoglobinaemia	-
Enalaprilat	Intravenous form of enalapril	0.625-1.25 mg initial dose; repeat doses can be increased up to 5 mg maximum	15-30 min	6-12 h	Headache (awake patients) Cough (awake patients) Hyperkalaemia	Should be used in caution with Hypertrophic cardio-myopathy Severe aortic stenosis Unstented renal artery stenosis Contraindicated in pregnancy

Contd...

Table 3: Contd...

Drug	Class	Intravenous dose	Onset of action	Duration of action	Adverse effects	Contraindications
Fenoldopam	Synthetic benzazepine derivative which is a partial D_1 receptor agonist	0.01-0.3 μg/kg/min as a continuous infusion	1-3 min	15-30 min	Cardiac dysrhythmias Dose-dependent tachycardia	Contains sodium meta-bisulphite which can cause anaphylactic reaction May cause increase in intraocular pressure in patients with glaucoma

COPD – Chronic obstructive pulmonary disease; CAD – Coronary artery disease; MS – Mitral stenosis; SLE – Systemic lupus erythematosus; ICP – Intracranial pressure; MI – Myocardial infarction; CCB – Calcium channel blocker

first 30–60 minutes and gradual return to baseline over the next 24–48 hours. Metoprolol has been used in hypertension due to acute coronary syndromes, but use in other settings is not well documented. Nifedipine can cause uncontrolled hypotension and reflex tachycardia and is not indicated in most hypertensive crises.

Intra- and post-operative monitoring are for the detection and treatment of haemodynamic swings in hypertensive patients and to maintain the cardiac output. Invasive arterial blood pressure is usually not required unless the patient is undergoing a high-risk surgery or has a hypertensive crisis which requires treatment with vasoactive drugs. Similarly, central venous monitoring is usually reserved for cases with expected significant fluid shifts or if inotropic support is anticipated and/or required. Pulse contour algorithms-based cardiac output monitoring may not be reliable in hypertensive patients as these algorithms are based on a normal arterial compliance. Dynamic indices of preload such as stroke volume and pulse pressure variation have not been validated in hypertensive individuals.[9]

Transesophageal echocardiography is an excellent tool to differentiate the cause of intraoperative hypotension, but routine use is not indicated presently in patients, including hypertensive patients undergoing non-cardiac surgery. Modalities which seek to evaluate oxygen consumption and hence oxygen utilisation such as near-infrared spectroscopy (NIRS) or mixed venous oximetry have largely not been evaluated in this subset of patients.[9] It would be interesting to see whether NIRS can be used to define limits of auto-regulation in organs at risk such as brain and kidneys in hypertensive individuals.

CONCLUSION

Hypertension is a modifiable risk factor for cardiovascular diseases and outcomes, and the same is true of the patient presenting for surgery. Greater understanding of the disease process and the fact that anaesthesiologists have better drugs and monitors at their disposal to treat and evaluate the effects of such treatment has pushed the envelope continuously as to what constitutes a blood pressure which warrants cancellation of elective non-cardiac cases. It is not merely the elevated blood pressure values, but what these values do to organs is what constitutes risk. A diabetic patient may be at a greater risk of myocardial dysfunction with intraoperative blood pressures of 150/90 mmHg than an individual without any risk factors. Therefore, it is important to be careful but not necessarily fearful, as care improves patient outcomes whereas fear results in unnecessary delays and case cancellations.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Mancia G, Fagard R, Narkiewicz K, Redon J, Zanchetti A, Böhm M, *et al.* 2013 ESH/ESC guidelines for the management of arterial hypertension: The Task Force for the Management of Arterial Hypertension of the European Society of Hypertension (ESH) and of the European Society of Cardiology (ESC). Eur Heart J 2013;34:2159-219.
2. Fleisher LA. Preoperative evaluation of the patient with hypertension. JAMA 2002;287:2043-6.
3. Anchala R, Kannuri NK, Pant H, Khan H, Franco OH, Di Angelantonio E, *et al.* Hypertension in India: A systematic review and meta-analysis of prevalence, awareness, and control of hypertension. J Hypertens 2014;32:1170-7.
4. Varon J, Marik PE. Perioperative hypertension management. Vasc Health Risk Manang 2008;4:615-27.
5. Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL Jr., *et al.* The seventh report of the joint national committee on prevention, detection, evaluation, and treatment of high blood pressure: The JNC 7 report. JAMA 2003;289:2560-72.
6. James PA, Oparil S, Carter BL, Cushman WC, Dennison-Himmelfarb C, Handler J, *et al.* 2014 evidence-based

guideline for the management of high blood pressure in adults: Report from the panel members appointed to the Eighth Joint National Committee (JNC 8). JAMA 2014;311:507-20.
7. Howell SJ, Sear JW, Foëx P. Hypertension, hypertensive heart disease and perioperative cardiac risk. Br J Anaesth 2004;92:570-83.
8. Hanada S, Kawakami H, Goto T, Morita S. Hypertension and anesthesia. Curr Opin Anaesthesiol 2006;19:315-9.
9. Lapage KG, Wouters PF. The patient with hypertension undergoing surgery. Curr Opin Anaesthesiol 2016;29:397-402.
10. Spahn DR, Priebe HJ. Editorial II: Preoperative hypertension: Remain wary? "Yes" – Cancel surgery? "No". Br J Anaesth 2004;92:461-4.
11. Boersma E, Poldermans D, Bax JJ, Steyerberg EW, Thomson IR, Banga JD, *et al.* Predictors of cardiac events after major vascular surgery: Role of clinical characteristics, dobutamine echocardiography, and beta-blocker therapy. JAMA 2001;285:1865-73.
12. Lee TH, Marcantonio ER, Mangione CM, Thomas EJ, Polanczyk CA, Cook EF, *et al.* Derivation and prospective validation of a simple index for prediction of cardiac risk of major noncardiac surgery. Circulation 1999;100:1043-9.
13. Weksler N, Klein M, Szendro G, Rozentsveig V, Schily M, Brill S, *et al.* The dilemma of immediate preoperative hypertension: To treat and operate, or to postpone surgery? J Clin Anesth 2003;15:179-83.
14. Fleisher LA, Beckman JA, Brown KA, Calkins H, Chaikof E, Fleischmann KE, *et al.* ACC/AHA 2007 guidelines on perioperative cardiovascular evaluation and care for noncardiac surgery: Executive summary: A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing committee to revise the 2002 guidelines on perioperative cardiovascular evaluation for noncardiac surgery): Developed in Collaboration with the American Society of Echocardiography, American Society of Nuclear Cardiology, Heart Rhythm Society, Society of Cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, Society for Vascular Medicine and Biology, and Society for Vascular Surgery. Circulation 2007;116:1971-96.
15. Hartle A, McCormack T, Carlisle J, Anderson S, Pichel A, Beckett N, *et al.* The measurement of adult blood pressure and management of hypertension before elective surgery: Joint Guidelines from the Association of Anaesthetists of Great Britain and Ireland and the British Hypertension Society. Anaesthesia 2016;71:326-37.
16. Prys-Roberts C, Meloche R, Foëx P. Studies of anaesthesia in relation to hypertension. I. Cardiovascular responses of treated and untreated patients. Br J Anaesth 1971;43:122-37.
17. Howell SJ, Sear YM, Yeates D, Goldacre M, Sear JW, Foëx P, *et al.* Risk factors for cardiovascular death after elective surgery under general anaesthesia. Br J Anaesth 1998;80:14-9.
18. Goldman L, Caldera DL. Risks of general anesthesia and elective operation in the hypertensive patient. Anesthesiology 1979;50:285-92.
19. Aronson S, Boisvert D, Lapp W. Isolated systolic hypertension is associated with adverse outcomes from coronary artery bypass grafting surgery. Anesth Analg 2002;94:1079-84.
20. Muscholl MW, Hense HW, Bröckel U, Döring A, Riegger GA, Schunkert H. Changes in left ventricular structure and function in patients with white coat hypertension: Cross sectional survey. BMJ 1998;317:565-70.
21. Nalbantgil I, Onder R, Nalbantgil S, Yilmaz H, Boydak B. The prevalence of silent myocardial ischaemia in patients with white-coat hypertension. J Hum Hypertens 1998;12:337-41.
22. Sear JW. Perioperative control of hypertension: When will it adversely affect perioperative outcome? Curr Hypertens Rep 2008;10:480-7.
23. Celis H, Staessen JA, Thijs L, Buntinx F, De Buyzere M, Den Hond E, *et al.* Cardiovascular risk in white-coat and sustained hypertensive patients. Blood Press 2002;11:352-6.
24. National Institute for Health and Care Excellence. Hypertension in Adults: Diagnosis and Management. NICE Clinical Guideline CG 127. Available from: http://www.nice.org.uk/guidance/cg127. [Last accessed on 2017 May 11].
25. Reich DL, Hossain S, Krol M, Baez B, Patel P, Bernstein A, *et al.* Predictors of hypotension after induction of general anesthesia. Anesth Analg 2005;101:622-8.
26. Schulte E, Ziegler D, Philippi-Höhne C, Kaczmarczyk G, Boemke W. Angiotensin-converting enzyme inhibition and blood pressure response during total intravenous anaesthesia for minor surgery. Acta Anaesthesiol Scand 2011;55:435-43.
27. Höhne C, Meier L, Boemke W, Kaczmarczyk G. ACE inhibition does not exaggerate the blood pressure decrease in the early phase of spinal anaesthesia. Acta Anaesthesiol Scand 2003;47:891-6.
28. Wong KC, Schafer PG, Schultz JR. Hypokalemia and anesthetic implications. Anesth Analg 1993;77:1238-60.
29. Wijesundera DN, Duncan D, Nkonde-Price C, Virani SS, Washam JB, Fleischmann KE, *et al.* Perioperative beta blockade in noncardiac surgery: A systematic review for the 2014 ACC/AHA guideline on perioperative cardiovascular evaluation and management of patients undergoing noncardiac surgery: A Report of the American College of Cardiology/American Heart Association Task Force on practice guidelines. J Am Coll Cardiol 2014;64:2406-25.
30. Erasmus MC Follow-up Investigation Committee. Report on the 2012 Follow-up Investigation of Possible Breaches of Academic Integrity; 2012. Available from: https://www.cardiobrief.files.wordpress.com/2012/10/integrity-report-2012-10-english-translation.pdf. [Last accessed on 2017 Jul 12].
31. POISE Study Group, Devereaux PJ, Yang H, Yusuf S, Guyatt G, Leslie K, *et al.* Effects of extended-release metoprolol succinate in patients undergoing non-cardiac surgery (POISE trial): A randomised controlled trial. Lancet 2008;371:1839-47.
32. Poole-Wilson PA, Swedberg K, Cleland JG, Di Lenarda A, Hanrath P, Komajda M, *et al.* Comparison of carvedilol and metoprolol on clinical outcomes in patients with chronic heart failure in the Carvedilol or Metoprolol European Trial (COMET): Randomised controlled trial. Lancet 2003;362:7-13.
33. Monk TG, Bronsert MR, Henderson WG, Mangione MP, Sum-Ping ST, Bentt DR, *et al.* Association between intraoperative hypotension and hypertension and 30-day postoperative mortality in noncardiac surgery. Anesthesiology 2015;123:307-19.
34. Walsh M, Devereaux PJ, Garg AX, Kurz A, Turan A, Rodseth RN, *et al.* Relationship between intraoperative mean arterial pressure and clinical outcomes after non-cardiac surgery: Toward an empirical definition of hypotension. Anesthesiology 2013;119:507-15.
35. Charlson ME, MacKenzie CR, Gold JP, Ales KL, Topkins M, Shires GT, *et al.* Intraoperative blood pressure. What patterns identify patients at risk for postoperative complications? Ann Surg 1990;212:567-80.
36. Lonjaret L, Lairez O, Minville V, Geeraerts T. Optimal perioperative management of arterial blood pressure. Integr Blood Press Control 2014;7:49-59.
37. Kristensen SD, Knuuti J, Saraste A, Anker S, Bøtker HE, De Hert S, *et al.* 2014 ESC/ESA Guidelines on non-cardiac surgery: Cardiovascular assessment and management. The Joint Task Force on non-cardiac surgery: Cardiovascular assessment and management of the European Society of Cardiology (ESC) and the European Society of Anaesthesiology (ESA). Eur J Anaesthesiol 2014;31:517-73.
38. Vaughan CJ, Delanty N. Hypertensive emergencies. Lancet 2000;356:411-7.