Perioperative Control Of Hypertension: When Does It Adversely Affect Perioperative Outcome?

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In the United States, approximately 72 million people suffer from hypertension (that equates to about 30% of the population aged over 20 years-old). It is one of the most common chronic medical conditions worldwide (US National Center for Health Statistics, 2005), and occurs almost twice as often in the African-American population as those of Caucasian origin. The incidence of hypertension increases with age,[1] and affects men with a slightly greater incidence than women. In the USA, hypertension affects about 255 of all adults over the age of 40 years. More importantly, the prevalence of undiagnosed hypertension is about 1 in 15 (ie. about 15 million patients). In the UK, there are about 7.5 million patients suffering from raised blood pressure; but importantly 80-85% of these patients are either not treated or are poorly treated. Based on these two reported prevalences, the worldwide incidences of patients suffering from hypertension is about 600 million people, and hence the likelihood of a hypertensive patient undergoing elective non-cardiac surgery is high. Is there any evidence that hypertension affects perioperative outcome?

The following questions summarize some of the present controversies in the management of these patients:

1. Are all therapies equally effective at controlling the exaggerated hemodynamic responses; should drugs be continued until surgery; are there any important interactions with anesthesia?
2. Does drug therapy affect peri-operative outcome?
3. Does hypertension contribute to post-operative adverse cardiovascular events in surgical patients?
4. What should the clinician do for the surgical patient with isolated systolic hypertension or 'white-coat' hypertension?
5. Which patients (if any) should the anesthetist consider cancelling?

Blood pressure can be classified into 4 categories, as described in the JNC VII Report (2003):[2]

Normotension	<120 and <80 mmHg
Pre-hypertension	120-139 or 80-89
Stage I	140-159 or 90-99
Stage II	>160 or > 100

In primary care practice, both the WHO and British Hypertension Society guidelines target a blood pressure of < 140/85 in non-diabetic patients and < 140/80 in hypertensive diabetics.

1. DRUG THERAPIES, ANESTHESIA AND HYPERTENSION

For much of the last two decades, β-blockers have been the mainstay of the treatment of arterial hypertension in the United Kingdom and many other countries. However they are no longer the initial therapy for hypertension in many patients. Why is this? The change in treatment modality relates to the adverse and side-effects of the drugs. All β-blockers have a pre-diabetic potential. Is this new form of 'type 2 diabetes' significant for patients? Based on the VALUE trial, Aksnes et al suggest that the cardiac risk profile with β-blockers is about 50% that seen in patients of established diabetes mellitus.[3] If the results of the ASCOT study (Anglo-Scandinavian Outcome Trial) are included in any meta-analysis, nearly all outcomes are more favourably influenced by a regimen based on a calcium entry blocking drug when compared with atenolol.[4]

Treatment with β-blockers results in a decrease in aortic pressure that is less than that seen with calcium entry blocking drugs.[5] There is a lack of data for the capacity of β-blockade to achieve adequate regression of target organ damage such as left ventricular hypertrophy or endothelial dysfunction.[6] The cardiac protective effects of β-blockers are often over-stated; there is only one study investigating blood pressure management in patients with both hypertension and coronary artery disease (INVEST).[7] Hence, treatment with β-blockers is the least cost effective of all the standard therapies with regard to hospitalization; clinical events and therapy of new diabetes. Although they are no longer the first line drug for primary care treatment of hypertension, patients with the combination hypertension and coronary artery disease should continue to receive these agents. Interactions with both general and regional anesthetic techniques show them to be well tolerated, and to confer hemodynamic stability.[8]

Do the changes in drug therapies for management of hypertension in primary care have implications for the preoperative surgical patient with hypertension? Are all therapies equally effective at controlling exaggerated hemodynamic responses? Should all drugs be continued up until surgery? Are there any important interactions with anaesthesia?

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Anesthesia and hypertensive therapies

Diuretic-treated patients can present with hypokalemia, raising the issue of preoperative potassium supplementation. However the studies of Wong et al showed that rapid normalization of the plasma potassium concentration may worsen the trans-membrane potassium gradient, thereby increasing the risk of arrhythmias.[9] The electro-physiological indicators of hypokalemia therefore make slow replacement of potassium over 24-48 hours the optimum approach. Current policy is that anti-hypertensive therapies are continued up to the morning of surgery, with the possible exceptions of ACEIs and ARAs. Our studies found no significant differences in blood pressure and heart rate responses between agents, with no excessive hypotension on induction of anesthesia in patients receiving monotherapy of ACE inhibitors, β-blockers, calcium channel entry blockers or diuretics. Only -adrenoceptor blockers protect against the noxious pressor and chronotropic responses to laryngoscopy and intubation, thereby reducing the risk of myocardial ischemia.[10,11]

Interactions between anesthesia and angiotensin converting enzyme inhibitors (ACEIs) and angiotension II receptor antagonists (ARAs) are controversial. High doses of ACEIs and ARAs may accentuate the hypotension caused by anesthesia, and patients on ARA are less responsive to ephedrine and phenylephrine. Although some authors suggest that these drugs are stopped 24 hours before surgery,[12-14] this may increase the need for active postoperative management of hypertensive episodes.[15] The doses of ACEIs and ARAs prescribed in the UK and many other countries tend to be lower than in the studies of Coriat and Brabant;[12,13] and hence maintaining these drugs (as for all other therapies) up to and including the morning of surgery may be practised. To date (January 2010), there are no data on the interaction between anesthesia and surgery for a new class of drugs (direct renin inhibitors - eg. aliskiren).

α_2 agonists achieve hemodynamic stability by reducing sympathetic activity. Clonidine also causes anxiolysis and sedation; so decreasing the requirements for volatile and intravenous anesthetic agents, and may also reduce the risk of postoperative adverse cardiac events in both cardiac and noncardiac surgical patients)![16] The meta-analysis by Wijeysundera et al[17] reported a reduction in morbidity (myocardial infarction) and mortality in vascular surgical patients receiving an α_2 agonist, and a reduction in myocardial ischemia in cardiac surgical patients. However the heterogeneity of the studies in this analysis stresses the need for further large randomized clinical trials. With β-blockers, the numbers-needed-to-treat (NNT) to prevent cardiovascular complications range between 2.5-3.8 compared with NNTs of 19-38 for α_2 agonists. A meta-analysis of studies with dexmedetomidine (DMD) including 840 patients from 20 trials has reported TRENDS towards improved cardiac outcomes (myocardial infarction, myocardial ischemia) but an increased incidence of hypotension and bradycardia.[18]

All anti-hypertensive therapies show no major effect on cardiovascular disease risk in hypertensive patients that is independent of their effect on blood pressure. The effect of lowering on risk of blood pressure is independent of the pre-treatment blood pressure; different drugs differ little in their efficacy; and there is a halving of the risk of coronary heart disease events and strokes for each 10 mmHg reduction in diastolic blood pressure.[19]

Recent data suggest that aspirin may antagonise the hypotensive effects of spironolactone, ACEI's and ARA's. Although statins have been shown to be beneficial in cardiovascular at-risk patients, this benefit is lost by acute pre-operative withdrawal. Another important issue in the hypertensive patient is the change in organ autoregulation with disease and treatment. Does anesthesia affect these autoregulatory mechanisms? Kadoi et al have demonstrated there is better preservation of cerebral blood flow control in the presence of changes in paCO2 during isoflurane anesthesia when compared with sevoflurane anesthesia.[20] If similar results are demonstrated by other research groups, this may influence our choice of anesthetic technique in the hypertensive patient.

2. β-BLOCKERS, MYOCARDIAL ISCHEMIA AND ADVERSE CARDIAC OUTCOMES

Besides their effects on heart rate and blood pressure, β-blockers have other useful perioperative properties including reduction of myocardial ischemia[19,21] and adverse cardiac events.[22,23] The studies of Mangano et al[22] and Poldermans et al,[23] in which more than 60% of the patients were described as being 'hypertensive', both show improvements in 2 and 1 year outcomes respectively in patients with or at high-risk for coronary artery disease undergoing non-cardiac surgery and who were treated with perioperative β-blockers. In the study of Mangano et al, atenolol started preoperatively and continued for 1 week after surgery increased event-free survival from 81% to 91%.[22] Despite some limitations of the study design (analysis did not include cardiac events occurring during hospitalization; and β-blockers were withdrawn preoperatively in some patients prior to randomization), the American College of Medicine proposed in 1997 that atenolol be given before operation to all patients with coronary artery disease or with associated risk factors. This approach was supported by the data available at that time relating to the efficacy of β-blockers in the treatment of myocardial infarction, hypertension, and later in the management of patients with cardiac failure.

In 1999, Poldermans and colleagues reported a randomized control trial (RCT) in patients with reversible cardiac ischemia presenting for major elective vascular surgery.[23] The results showed a

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100% reduction in the incidence of myocardial infarction and an 80% reduction in cardiac deaths in those patients treated with bisoprolol before and for 28 days post-surgery. Again the study presents difficulties for the clinician - as it was an unblinded study, and was stopped at the first interim analysis. However, it appeared to strengthen the case for β-blockade in patients with coronary artery disease or with significant risk factors (including hypertension), such that the American College of Cardiology/ American Heart Association (ACC/AHA) 2002 guidelines state 'that current studies suggest that appropriately administered β-blockers reduce perioperative ischemia and MAY (emphasis added) reduce the risk of myocardial infarction and death in high-risk cases'. These guidelines categorized the use of β-blockers in patients undergoing vascular surgery with ischemia detected during preoperative testing as a class I recommendation, and the use of preoperative β-blockers in patients with preoperative untreated hypertension, known coronary artery disease, or major risk factors for coronary artery disease as class IIa recommendations.

Subsequent studies, meta-analyses and systematic reviews have questioned these data, such that the 2006 and 2007 ACC/AHA guidelines stated that 'current studies SUGGEST (emphasis added) that β-blockers reduce perioperative ischemia and MAY (emphasis added) reduce the risk of myocardial infarction and death in patients with known coronary disease'.[24]

In summary, our knowledge about the efficacy of β-blockers in medical settings has changed with regard to hypertension (due to the findings of the ASCOT and ALLHAT studies); in the management of acute myocardial infarction (with publication of the COMMIT trial); and because of doubts over their efficacy of β-blockade in the perioperative period. Although the studies of Mangano et[22] and Poldermans et al[23] support the advantages of perioperative β-blockade, subsequent RCTs (including DIPOM; MaVS; POBBLE; BBSA) fail to show similar results.[25-28] Because of this uncertainty,there was need for a large multinational RCT of β-blockers versus placebo in patients at risk of perioperative cardiac events. This has recently reported as the PeriOperative Ischemia Study Evaluation [POISE], a multicenter study recruiting 8351 patients. POISE compared the study drug (metoprolol succinate extended-release) and placebo.[29] Treatments were started 2-4 hours before surgery and continued for 30 days. The primary 30 day outcome was a combined one of cardiovascular death, non-fatal myocardial infarction, and non-fatal cardiac arrest. Secondary outcomes included total all-cause mortality, stroke, myocardial infarction, need for coronary revascularization, new atrial fibrillation, congestive cardiac failure, hypotension and bradycardia. 63% of the 8351 patients were classified as 'hypertensive'. Thirty day results show a significant reduction in ALL myocardial infarction [4.2% in the metoprolol group vs. 5.7% in the placebo arm]; a reduced need for coronary revascularization; and a reduction in the number of patients developing atrial fibrillation. However there were significant increases in total mortality [3.1% vs. 2.3%]; stroke [1% vs. 0.5%]; and clinically significant hypotension and bradycardia in the metoprolol group.

As a result of all these studies, the utility of β-blockade in the medical and perioperative management of the hypertensive patient is presently debated, and best practice remains to be defined.

3. HYPERTENSION AND PERIOPERATIVE OUTCOMES

Does hypertension contribute to post-operative adverse cardiovascular events in surgical patients? Until recently, there were few data on the influence of hypertension on postoperative outcomes, although Sprague in 1929 reported a 32% incidence of perioperative cardiac death in patients with hypertensive heart disease.[30] But what is the effect of hypertension on cardiovascular morbidity? In 1973-75, Prys-Roberts and colleagues examined the influence of anesthesia and hypertension, and showed that induction of anesthesia, laryngoscopy and intubation were associated with development of hypotension, ventricular arrhythmias and cardiac ischemia in untreated or poorly treated hypertensive patients, but that the effects can be obtunded by pre-operative β-blockade.[8,31,32] However Goldman and Caldera found no relationship between preoperative blood pressure and development of cardiac arrhythmias, ischemia, failure or postoperative renal failure in patients with mild hypertension.[33]

Results from examination of the influence of hypertension as a determinant of cardiovascular complications give a confused picture. The study of Forrest et al[34] including more than 17000 patients showed no difference in incidence of complications between the whole group and a sub-group of hypertensive patients (3.5% vs. 7.0%)). More recently, Davenport et al reported on 183069 patients from 128 VA and 14 academic medical centers who underwent surgery during the years 2002-2004.[35] The main outcome measures were the incidence of serious cardiac adverse events (namely acute myocardial infarction and cardiac arrest needing resuscitation within 30 days of surgery). The authors reported that 2362 patients (1.29%) suffered one or other of these adverse events, with 59.44% dying. This compared with a death rate of 1.85% in patients not undergoing a serious cardiac adverse event. Using univariate modeling, they identified 31 preoperative risk factors, and 10 preoperative tests as significant markers. These data were then subjected to forward stepwise logistic regression analysis. After adjusting for confounding variables, the authors showed that hypertension was not a significant risk factor.

Because the incidence of perioperative complications in noncardiac surgical patients is low,

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surrogate end-points that occur more frequently are often used to assess perioperative outcome (these include hemodynamic instability; cardiac ischemia; major cardiac complications as well as cardiac death).

Cardiac ischemia and biomarkers

We have previously reported a greater incidence of preoperative silent myocardial ischemia (SMI) in hypertensive compared with normotensive patients; and in untreated and poorly treated hypertensive patients compared with normotensive patients and treated hypertensive patients with blood pressure <160/90.[36,37] Different intercurrent treatments appear to have no effect on the occurrence of preoperative SMI in these at-risk patients. Conflicting evidence exists for an association between hypertension, postoperative SMI and outcome.[38-40] Howell et al found univariate associations between diagnosed and treated arterial hypertension, admission systolic and diastolic blood pressures and post-operative SMI. Only hypertension per se and systolic blood pressure remained following multivariate analysis with the odds ratio for post-SMI being 1.2 per 10 mmHg increase in systolic pressure.[40] This finding agreed with the work of Stamler et al who showed systolic hypertension to be the more potent risk factor for the complications of hypertensive disease.[41] Most studies addressing the question of hypertension in surgical patients have examined the association between hypertension and outcome without paying any attention to either the level of blood pressure, or the presence of absence of treatment. We found no association between hypertension and perioperative myocardial ischemia when the data are adjusted for the presence of confounders (β-blockade, calcium channel entry blockade and vascular surgery).[42]

Although hypertension is associated with increased perioperative SMI, which may in turn be associated with an increased incidence of adverse cardiovascular complications including death, we still need firm data against which to use these results for cancellation of elective surgical patients.

A recent study from Switzerland has reported the effects of anaesthesia and surgery on a composite cardiovascular endpoint (hypo- or hypertension [< or > 30% MAP for > 5 minutes; occurrence of new arrhythmias; angina pectoris or ECG changes compatible with ischemia; or related death] in 124939 patients undergoing elective surgery under either general or regional anesthesia.[43] 27881 patients were hypertensive on treatment or had a preoperative blood pressure > 160/100. In 7549 patients, at least one cardiac adverse event was observed in the 24 hours following surgery (6% [CI 5.9-6.2]). The incidence in hypertensive patients was 11.2% compared with 4.6% in normotensive patients [crude relative risk (RR) ratio 2.64; adjusted RR 1.38 (1.27-1.49)]. In the patient with hypertension AND OTHER CARDIOVASCULAR DISEASE, the RR ratio was 1.62.

Another surrogate marker is myocardial necrosis measured either using monoclonal antimyosin antibodies or serum troponins. Cardiac ischemia causes release of troponins. Pons-Llado et al showed a greater degree of underlying myocardial damage in symptomatic hypertensive patients compared with both asymptomatic hypertensive and normotensive patients;[44] whereas Reddy et al found elevated serum troponin T concentrations in hypertensive medical patients compared with controls.[45] In an unpublished meta-analysis of 22 studies, Biccard and Sear have found a relative risk ratio of 1.36 [1.21-1.53] for the relationship between hypertension and elevated postoperative cardiac troponins. These data indicate that hypertension may be associated with an increased risk of myocardial damage in the perioperative period.

4. MAJOR CARDIAC COMPLICATIONS AND HYPERTENSION

In 2004, Howell et al published a meta-analysis of 30 studies examining hypertensive disease as a univariate marker for adverse cardiac outcome. There was a significant association with cardiac death and cardiac complications (odds ratio 1.25 [1.07-1.47]);[46] however this analysis did not take account of any confounding factors, or heterogeneity between the different studies included in the paper. Nevertheless again the results suggest a small but significant influence of a preoperative diagnosis of arterial hypertension on cardiac outcome.

Does the admission blood pressure influence perioperative outcome? Goldman and Caldera found no association between admission blood pressure and perioperative cardiac death;[33] and the data of Howell et al were insufficient to allow comment on an association between level of blood pressure elevation and outcome.[40,46] However two other recent studies suggest there may be an association between admission blood pressure and adverse outcomes.[47,48]

Further support for hypertension as a predictor of cardiac adverse events has been found in a prospective observational study of 7740 (general, vascular and urologic surgery) operations.[49] 83 patients experienced a perioperative cardiac adverse event (cardiac arrest; non-STEMI; Q wave myocardial infarction; or new cardiac arrhythmia) within 30 days of surgery. Using univariate analysis, nine independent predictors of adverse outcome were identified (age > 68 years; BMI > 30; emergency surgery; previous PCI or cardiac surgery; active CCF; cerebrovascular disease; operation lasting >3.8 hours; blood requirement of >1 unit intraoperatively; and hypertension). When logistic regression was used to include both pre- and intra-operative variables, there was a significant adjusted hazared ratio associated with pre-existing hypertension [HR: 1.7 (1.0-2.9)] –

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the magnitude of the increased risk being similar to that reported by Howell et al.[46]

Another recent study examined the predictors of acute renal failure after noncardiac surgery in patients with pre-existing normal renal function,[50] again based on a prospective observational study of 65043 patients. 15102 of these patients fulfilled inclusion criteria. Outcome measures were acute renal failure in the first seven postoperative days; and 30-, 60- and 365-day all-cause mortality. Hypertension was a co-morbidity factor in 30% of all patients; and in 40% of those developing postoperative renal dysfunction. However, logistic regression modeling did not show hypertension to be an independent predictor of adverse outcome; although both coronary artery disease and peripheral vascular disease (both of which may be associated with hypertension) were predictive markers.

5. CARDIAC DEATH AND HYPERTENSION

There are fewer data examining the association between hypertensive disease and postoperative mortality.[51,52] We have shown both univariate and multivariate associations between 30-day postoperative cardiac mortality and a history of hypertension in elective surgery using a case-control analysis of data from the Oxford Record Linkage Study. An analysis involving over 22000 patients shows a relative risk ratio for cardiac death in the perioperative period of 1.40 [1.11-1.75] in hypertensive patients.

6. ISOLATED SYSTOLIC HYPERTENSION AND ELECTIVE SURGERY

Isolated systolic hypertension (ISH: SBP >140mmHg; DBP <90mmHg in the absence of any other secondary disease input in patients aged > 18-years) is the most common subtype of raised blood pressure. Diagnosis is based on the average of 2 or more seated blood pressure readings on 2 or more occasions. It affects 2/3 of all patients aged >50 years, and is more prevalent than diastolic hypertension. ISH is associated with greater risk of patients developing fatal and non-fatal strokes, and coronary heart disease, congestive cardiac failure, renal insufficiency and cardiac death. ISH occurs because of increased conduit vessel stiffness and decreased distensibility of the aorta and large arteries. The heart responds to the increase in wall tension by LV hypertrophy and an increased myocardial contraction time, and in turn secondary diastolic dysfunction. There is also an impairment of endothelial function. Effective treatments in non-surgical patients (thiazides, calcium channel blockade or β-adrenoceptor antagonists with vasodilating properties eg. dilevalol) decrease overall mortality, as well as decreasing the incidence of CVA, myocardial infarction and congestive cardiac failure. Many ISH patients also have pulse pressure hypertension (PP > 80 mmHg).[53]

To date, there are few observations on anesthetic interactions in these patients.[54] In the presence of uncontrolled systolic hypertension, induction of anesthesia causes decreases in blood pressure and stroke volume. This is difficult to prevent. Useful approaches may include head-down tilt to increase venous return and increments of metaraminol (with its predominant veno-constrictor properties) titrated to response. Use of vagotonic drugs (especially the combination propofol and opioids) is best avoided, and pre-treatment with glycopyrrolate may be advantageous. These patients also suffer marked vasopressor responses to noxious stimulation. Tamborini and colleagues have also shown there to be a reduction in coronary flow during induction of anesthesia.[55]

Hence it is relevant to ask whether we need to do anything special for the patient with isolated systolic hypertension who is scheduled for surgery?

Other than a possible association with postoperative silent myocardial ischemia (SMI), there are few data suggesting that ISH is a risk factor per se in relation to anesthesia. There is, however, evidence that patients with ISH may show a greater 'white-coat' effect than those with systo-diastolic hypertension, with the blood pressure settling with time;[56] but the relevance of this to the surgical patient is unclear. The data of Howell et al[40] showed that only systolic arterial pressure (not diastolic pressure) was a risk factor for the development of postoperative SMI - with an odds ratio of 1.20 for increasing the risk for each 10 mmHg increase in systolic pressure. There are few other outcome data for patients with ISH. One such set of data showing an association between ISH and adverse cardiovascular outcome is that of Aronson et al using a prospective analysis of 2417 patients undergoing coronary artery bypass surgery.[57] The unadjusted odds ratio for the association between ISH and adverse outcome was 1.4; and after adjusting for confounders, this was still an increased OR of 30% over controls. Other data from Benjo et alhave confirmed that pulse pressure is an age-independent predictor of stroke development after cardiac surgery.[58]

Whether cancellation of the surgical patient with ISH in order to initiate treatment is only justified if this can be shown to improve outcome; these data are awaited.

7. WHITE-COAT HYPERTENSION AND ANESTHESIA

White-coat hypertension (WCHT) is defined as a nurse-taken blood pressure of <140/90 when compared with a physician-taken value of >160/95. It is thought that the blood pressure increase is associated with stress.[59] An increased incidence of SMI is seen in patients with white-coat hypertension.[60] Because of this association, treatment may be justified. Diagnosis depends on use of 4 hourly BP chart for 12 hours - does it settle?

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8. HYPERTENSION AND PATIENT CANCELLATION

Which groups of patients (if any) should the anesthetist consider cancelling? The 2007 ACC/AHA guidelines offer few substantive recommendations as to which hypertensive patients should be cancelled to allow treatment prior to surgery, or how long such treatment should be continued before surgery. Indeed the ACC/ AHA Guidelines list 'uncontrolled systemic hypertension' as a low-risk factor for cardiac complications.

Observational data agree that stage 1 and 2 hypertension is not an independent risk factor for peri-operative cardiovascular complications, and hence there is no scientific evidence to support postponing these patients IN THE ABSENCE of target organ damage. However, the case for stage 3 (SAP >180 and/or DAP >110 mmHg) hypertension is less clear; the ACC/AHA recommend control of blood pressure before surgery, but this is not supported by a large body of data relating exclusively to patients with these levels of blood pressure.

Our recommendation and practice is only to cancel and treat in those with documented target-organ damage. Blood pressure control should be optimized pre-surgery in patients in whom hypertension is associated with accompanying significant risk factors such as diabetes mellitus, coronary artery disease, peripheral vascular disease, impaired renal function, smoking or hyper-cholesterolemia.[61] In patients with ISH, there is a clear association with an increased prevalence of SMI; but the influence of ISH on perioperative outcomes has not been studied. In patients with 'white-coat' hypertension, as many repeat blood pressures as possible should be obtained to inform clinical decisions. Starting a normally normotensive patient with white-coat hypertension on inappropriate therapy is dangerous. If surgery is to be deferred to allow white-coat hypertension to be treated, it is unclear how long treatment be given before the patient is subjected to surgery.

CONCLUSIONS:

Patients with hypertension are frequently encountered in noncardiac surgical practice. They require careful assessment by the anesthetist with rewgard to adequacy of treatment and identification of accompanying target-organ damage. In managing preoperative hypertension, cosmetic control of blood pressure is not recommended because both vascular and cerebrovascular autoregulation remain abnormal for several weeks, whereas it may take 2-18 months for treatment of hypertension to influence diastolic dysfunction. There is no doubt that severe perioperative hypertension is a major threat to hypertensive patients especially in the presence of increases in blood pressure in excess of about 20% of the preoperative value. The consequences of these pressure surges include bleeding from vascular suture lines, cerebrovascular haemorrhage and myocardial ischemia/ infarction. The mortality from such events may be as high as 50%. Such perioperative hypertensive crises are generally sympathetically medicated, and are associated with increases in peripheral vascular resistance.

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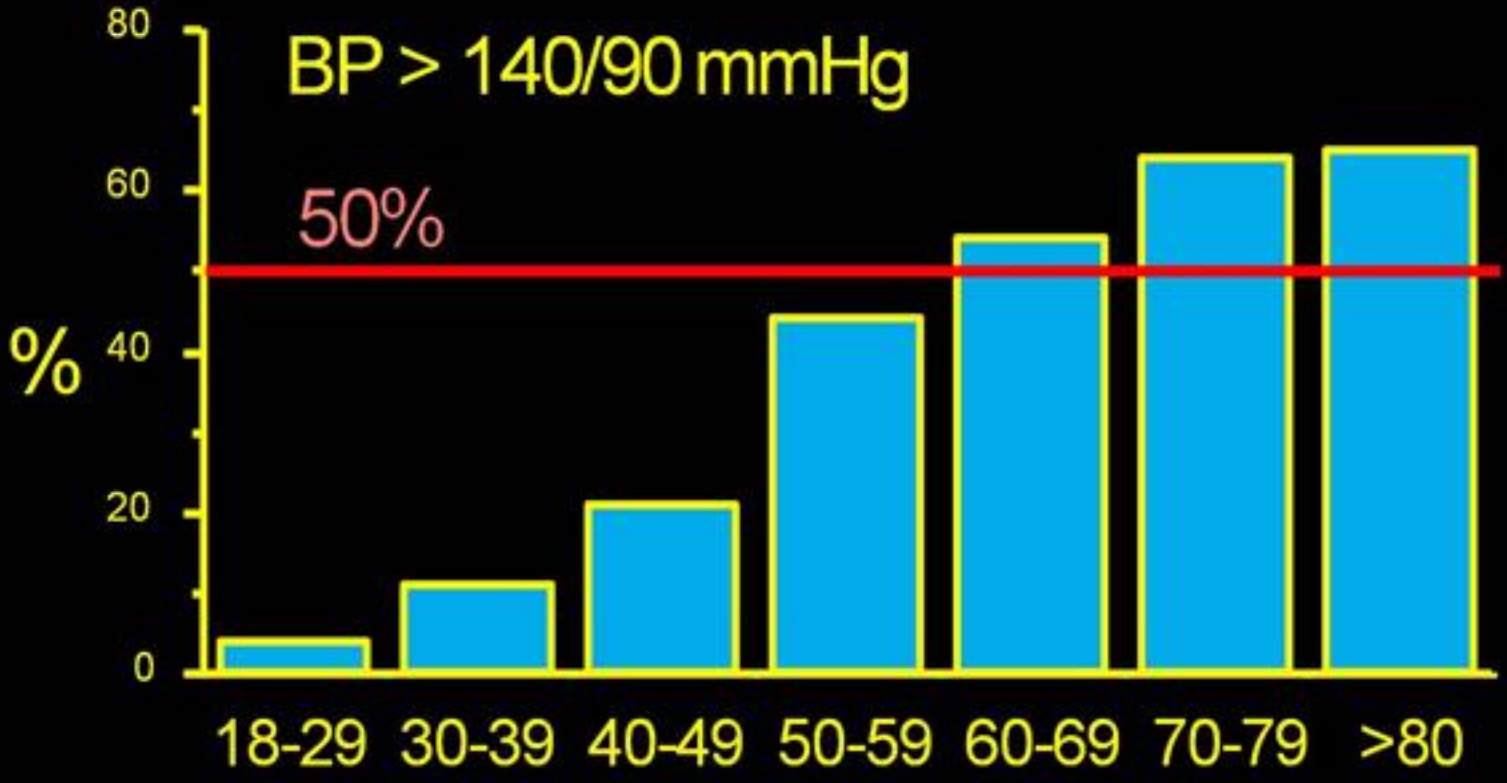
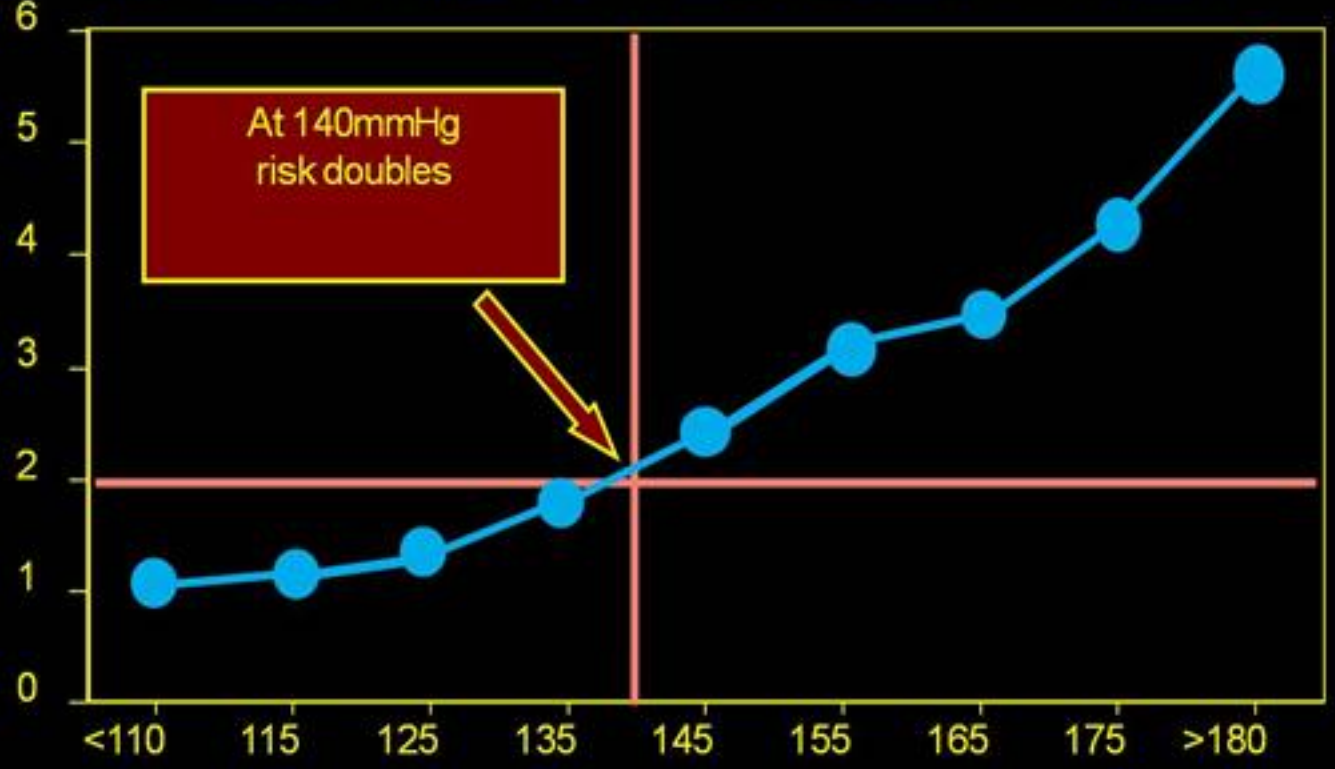
Prevalence of hypertension: role of age



National Center for Health Statistics, 1988-1991

Relative risk of death from coronary disease

(US National High Blood Pressure Education Program)



Stamler et al. Arch Intern Med 1993; 153: 598-615

New classification of blood pressure

Joint National Committee VII report 2003

Hypertension 2003; 42: 1206-1252

Optimum	<120	<80 mmHg
Normal	120-129	80-84 mmHg
High normal	130-139	85-89 mmHg
Hypertension (stage 1)	140-159	90-99 mmHg
Hypertension (stage 2)	>160	>100 mmHg

Hypertension - NICE guideline (UK) June 2006

Offer drug therapy to

- Patients with BP 160/100 mmHg or more
- Patients with BP 140/90 mmHg or more and raised cardiovascular risk

> 55 yrs calcium channel blocker or thiazide diuretic

< 55 yrs ACE inhibitor or angiotensin receptor antagonist

Beta-blockers: if coronary heart disease

Beta-blockers in the treatment of hypertension

1. Meta-analysis of RCTs 94,492 patients
2. Meta-analysis of RCTs 30,842 patients

Beta-blockers appear to increase

a. the risk of new onset diabetes

b. the risk of stroke compared with other agents

Beta-blockers do not appear to decrease the risk of death or myocardial infarction

Beta-blockers should not be used as first line treatment

1. Bangalore et al. Am J Cardiol 2007; 100: 1254-1262
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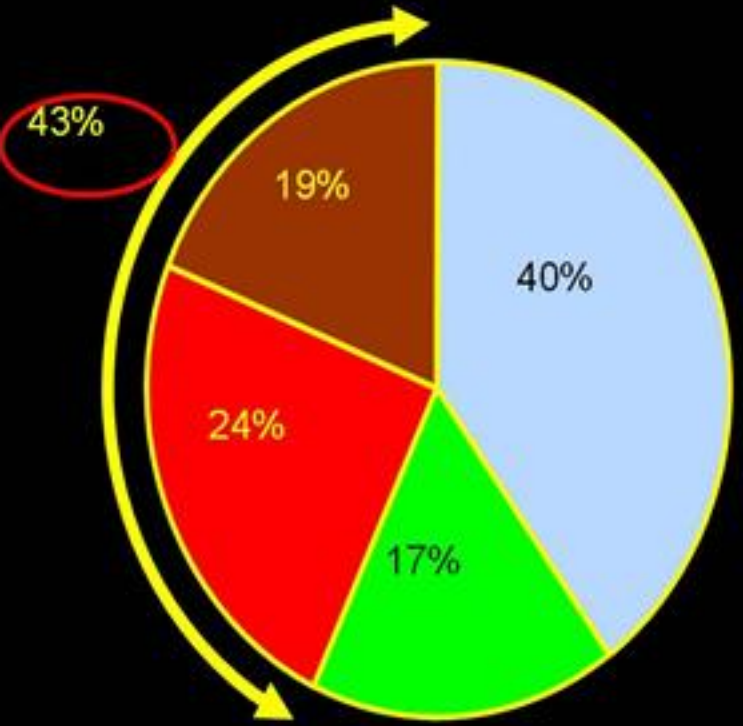
Prevalence of hypertension (>160/90mmHg) in a USA preoperative assessment clinic



2953 patients

- Normotensive
- Well controlled HT
- Poorly controlled HT
- Untreated HT

Definition: > 160/90 mmHg

O'Reilly et al. Anesthesiology 2001; 95: A1

Perioperative silent myocardial ischaemia

- most frequent perioperative complication
- associated with cardiac events
- high incidence of cardiac morbitity in patients with prolonged (> 2 hrs) ischaemia (odds ratio 21.7)

Mangano et al. N Engl J Med 1990; 323: 1781
Landesberg et al. Lancet 1993; 341: 715

ACC/AHA guideline

For stage 1 and 2 hypertension (<179 / <109 mmHg); there is no scientific evidence for postponement of surgery to control the BP in order to improve outcome in the absence of target organ damage

Eagle et al, 2002. J Am Coll Cardiol; 39: 542-553

ACC/AHA guideline

Stage 3 hypertension (>180 / >110 mmHg) should be controlled before surgery (days to weeks) if there is evidence of TOD. What if not?

Emergency: use rapidly acting agents

Eagle et al, 2002. J Am Coll Cardiol; 39: 542-553

Isolated systolic hypertension and increased pulse pressure

Associated with increased risk of congestive cardiac failure, coronary artery disease and stroke

Each 10 mmHg increase in pulse pressure associated with 20% increased risk of cardiovascular mortality in hypertensive patients.