Is a rethink of our approach to hypertension necessary?

A Coetzee, A Levin
Department of Anaesthesiology and Critical Care, Faculty of Health Sciences, University of Stellenbosch, Tygerberg, Stellenbosch, South Africa

Introduction

The risks and management of hypertensive patients for elective anaesthesia are often debated. There is a perception that patients with untreated or ineffectively treated hypertension are at risk, in particular for cardiac and cerebrovascular events.

The change in internal to external diameter of the vasculature results in an altered vascular pressure-flow relationship in the hypertensive patient. This gives rise to the exaggerated hypertensive and hypotensive response seen. The hypotensive tendency is aggravated by the diastolic dysfunction often present. In addition, autoregulation is right shifted, impacting on the lower limit of autoregulation.

Treating patients for their hypertension requires months to years to restore the autoregulation towards normal, even though the blood pressure may have been be normalized.

Except for patients with significant end-organ dysfunction (such as cardiac failure), application of our knowledge of the pathophysiology of hypertension, combined with skill, should allow the anaesthetist to reconsider some of the current dogma in the management of the hypertensive patient.

Anaesthetists have differing approaches to the patient with hypertension scheduled for elective surgery. On many occasions, this leads to surgery being cancelled because the practitioner is of the opinion that the hypertension is uncontrolled. In this paper, the position projected by the authors, is that if the pathophysiology of hypertension is clearly understood, the majority of hypertensive patients can be accepted for anaesthesia, whether blood pressure control is deemed adequate or not. Nonetheless, it is also important that the anaesthetist appreciates the exceptions, and the reasons for these exceptions.

It must be made clear at the outset that this paper is written with the intent to provoke debate and discussion on this topic. Furthermore, the opinion stated in this article does not question existing wisdom that long-term advantages can be gained from the effective treatment of chronic hypertension. The discussion that follows therefore applies only to the perioperative period.

Correspondence:
Prof Andre Coetzee
e-mail: arc1@sun.ac.za

Anaesthetic problems associated with the hypertensive patient

The hypertensive patient presents the anesthetist with three main problems:

1. Episodes of hypertension;
2. Periods of hypotension;
3. Concerns regarding (pre-existing or consequent) end-organ damage, especially the brain, heart, vasculature and kidneys.

The first two are well known occurrences in patients with hypertension subject to anesthesia and surgery. In our view, understanding the pathophysiology should enable the anaesthetist to prevent these problems. We will also argue that concerns regarding end-organ damage are speculative and or can be effectively managed.

The essential pathophysiology of hypertension

Vascular pathophysiology and blood pressure lability
Changes occur both in:

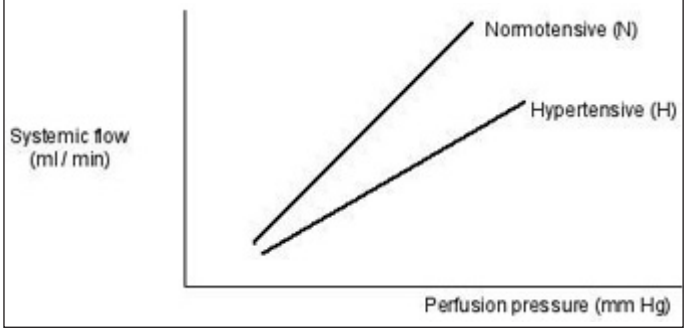
- The structure of the peripheral arteries ;
- Intravascular blood volume.

These pathophysiological changes are of practical significance as they are central to the understanding of the variations in blood pressure that anaesthetist has to deal with.

There are a number of well described changes that occur in the large, medium and small arteries and arterioles. Cumulatively this is referred to as medial hypertrophy.[1] Medial hypertrophy results in the normal vascular lumen to wall ratio of 1:5.2 decreasing to 1.3 to 4.7 in the hypertensive patient.[2]

The importance of this smaller than normal lumen to wall ratio was summarized in experimental work by

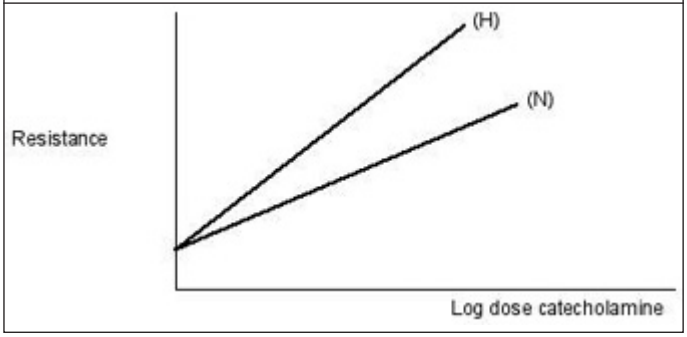
Fig 1: Flow and pressure relationship in normo (N) and hypertensive (H) rat vasculature. The resistance is higher in the H rat.



Folkow.[3,4] In his studies, controlled perfusion of the vasculature of normotensive and hypertensive rats resulted in the flow – pressure relationships depicted in Figure 1. From the flow – pressure quotient, which is the inverse of resistance, one can deduce from the graph that normotensive rats have a lower vascular resistance when compared to the hypertensive rat.

When Folkow subjected the vasculature to catecholamine stimulation, (Figure 2), the threshold for a response was the same in the normal and hypertensive rats. However, once a response was obtained, the slope of the dose-resistance relationship was steeper for the hypertensive rats compared with the normotensive ones.[3,5]

Fig 2: Response of the resistance to catecholamine stimulation. The threshold for the normotensive (N) and hypertensive (H) are the same but the response thereafter is clearly more in the H vasculature as compared to the N vasculature.

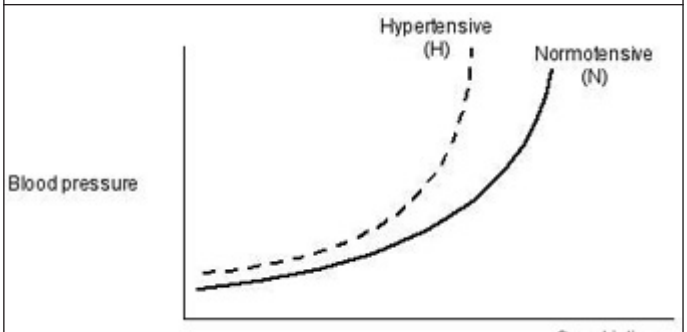


The following conclusions can be drawn from these experiments:

- The threshold for obtaining a pressure response appears to be similar for the hypertensive and normotensive rat. Therefore, the problem is not that the vasculature of hypertensive rats is inherently more sensitive to stimuli.
- However, the problem is that once a response is obtained, the extent of this response is more pronounced in the hypertensive compared with the normotensive rats.

The interpretation of these results is that there is no difference in autonomic control of the vasculature in hypertensive and normotensive subjects. However, the pathophysiological problem is that resistance to flow is higher in hypertensive subjects because of the smaller internal lumen of the vasculature. These conclusions apply equally to hypertensive human subjects.[5]

Fig. 3: Vessel diameter versus resistance (or blood pressure) in normotensive (N) and hypertensive (H) rats. For any degree of constriction the blood pressure in H will exceed the pressure in N.

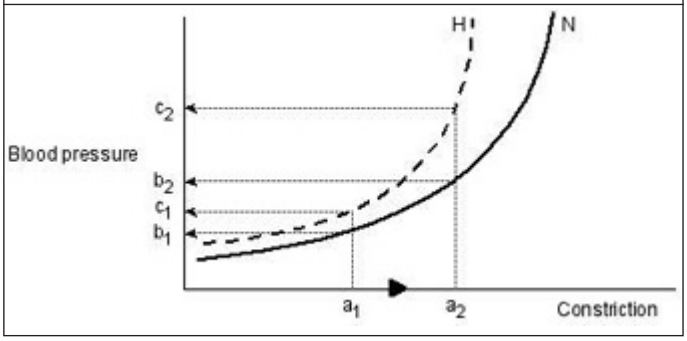


Blood pressure changes in lieu of the vascular changes

From the above results, Folkow and colleagues compiled the instructive vascular response curves for both normotensive and hypertensive subjects (Figure 3).[3,4] The authors used % shortening of the circumferential arterial muscles and resistance on the x and y-axes respectively. For the purpose of clinical applicability and clarity, the x and y-axes may be relabeled "vasoconstriction" and "blood pressure" respectively. These extrapolations are justified because of the relationship between the circumference of a vessel and its radius (circumference = $2\pi r$) and the relationship between pressure, flow and radius described in Pouseuille's equation.[6]

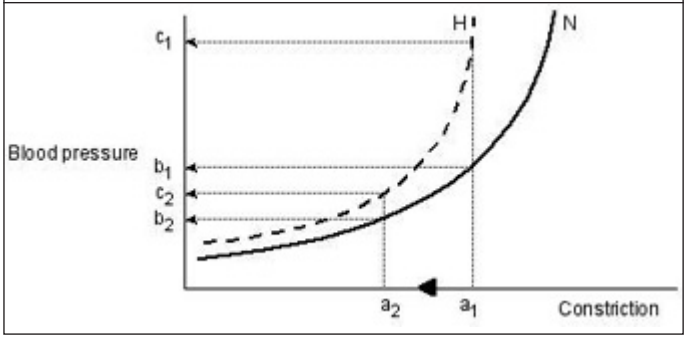
Consider, on the one hand, what would happen if vasoconstriction occurred and the vessel diameter in Fig 4 decreased from a1 to a2. The resulting increase in blood pressure would be significantly greater in the hypertensive patient, compared with the normotensive one.

Fig 4: For any particular increase in vasoconstriction (a1 to a2) , the resultant increase in blood pressure will be significantly more in the H (c1 to c2) compared to the normotensive (N) (form b1 to b2). This graph explains the excessive pressor response seen in the uncontrolled hypertensive patient.



On the other hand, if the stress is removed, autonomic mediated vasoconstriction will decrease and the internal radius of the vessel will increase. For similar decreases in vessel radius (from c1 to c2 on the y axis of Fig. 5), the resultant fall in blood pressure is represented by decreases from d1 to d2, and from e1 to e2, for the normotensive and hypertensive patients respectively. Figure 5 therefore illustrates that the extent of the fall in blood pressure, after similar percentage increases in

Fig 5: If vasodilation occurs (from a1 to a2), the extent of the fall in pressure will be from b1 to b2 for the normotensive (N) as compared to the greater fall (from c1 to c2) in the hypertensive.



vessel diameter, will be much greater in hypertensive than normotensive patients.

On studying Figures 4 and 5, it is easy to appreciate both the excessive hypertension following a vasoconstrictory stimulus (for example laryngoscopy and intubation) and also the exaggerated fall in pressure that follows even small amounts of vasodilation (for example when the appropriate anaesthetic level for surgery has been achieved).

On studying Figures 4 and 5, it is also noteworthy that, when the point of maximal vasodilatation is approached, the Folkow relationships for both hypertensive and normotensive patients approximate each other and are located on the less steep part of the vessel radius - pressure curve. Therefore, albeit the resting vessel resistance remains increased in hypertensive patients, the percent blood pressure change which occurs when the vessel diameter changes, is less when the patient is operating on the left side of the Folkow curve.

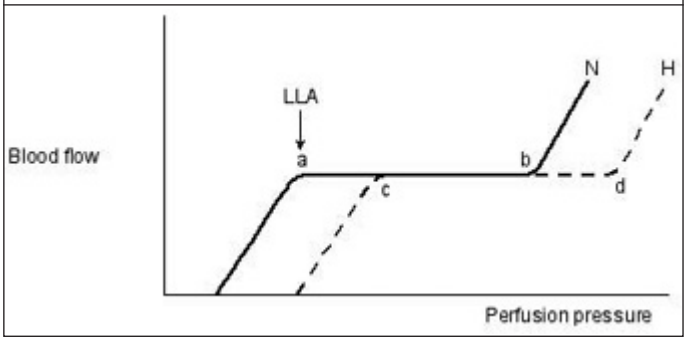
Another important pathophysiological principle is that uncontrolled hypertensive patients have a contracted intravascular blood volume.[7,8] The effect of this will be aggravated by the well documented diastolic dysfunction often seen in the hypertensive patient.[9] The single most important defense against a fall in blood pressure, when the stress stimulus is withdrawn, is the status of the intravascular volume.

Hence, the hypertensive patient is not only penalized by virtue of the vessel diameter dynamics, but preload, the main physiological buffer protecting against hypotension, is less efficient. Notwithstanding this problem, the opinion has often been expressed that, because a patient is hypertensive, fluid loading should be on the conservative side. Such an approach (partially) explains the subsequent hypotension the anaesthetist has to deal with. In contrast, the aforementioned pathophysiology suggests that a liberal fluid policy should be applied, while simultaneously considering the cardiac function of any particular patient. The vast majority of hypertensive patients have normal or near normal cardiac function and can cope with the required 10 – 15% expansion of the blood volume. To effectively expand the intravascular volume, consideration should be given to using a colloid that is retained within the intravascular space for a significant period of time.

Autoregulation

Autoregulation is defined as the "intrinsic tendency of an organ or tissue to maintain constant blood flow despite changes in arterial pressure".[10] The salient feature illustrated in a characteristic normal autoregulation curve is that between certain limits of blood pressure (marked by points a and b in Figure 6), organ blood flow is constant. If blood pressure decreases below point a, termed the lower limit for autoregulation (LLA), or above point b, the upper limit for autoregulation, flow becomes pressure dependant. Pressure dependant flow has particular physiological implications for the anaesthetist. To illustrate these implications, one can apply the autoregulation concept to the brain. Normal brain blood flow averages 50 ml/min/100g tissue. Below the lower limit of autoregulation, brain blood flow progressively decreases. When brain blood flow decreases to 20 - 25 ml/100g/min, electrical activity of the brain ceases.[11] Cessation or a decrease of organ function with reductions in their blood flow and nutrient supply is termed hibernation, a term more often used in cardiac pathophysiology. Hibernation represents a method whereby organs protect themselves against low oxygen supply scenarios and does not necessarily equate to tissue damage. Once nutrient flow is improved, function will be restored. Only when blood flow decreases to 6 - 15 ml/min/100g brain tissue, will membrane dysfunction and neuronal death occur.

Fig 6: Autoregulation in the normotensive (N) and hypertensive (H) patient. The H curve is right shifted and of significance is the lower limit for autoregulation (LLA) which increases from a to c. The upper limit also increases from b to d.



The important question that has to be addressed is what is the mean arterial pressure (MAP) at which organ blood flow decreases to potentially dangerous levels? Strangaard and colleagues[12] studied the effects of decreasing blood pressure on cerebral blood flow in awake patients. The 3 groups they studied comprised normotensive (mean arterial pressure [MAP] 98 ± 10 mm Hg), treated hypertensive (MAP 116 ±18 mm Hg) and untreated hypertensive subjects (MAP 145 ±17 mm Hg). The initial part of their study comprised lowering blood pressure and noting the point where flow becomes pressure dependant (LLA). Thereafter, they continued lowering blood pressure to the level where the patients demonstrated signs of cerebral ischaemia (ischaemic threshold, IT).

A particularly instructive aspect of the Strangaard study is that, if the LLA is described as a percentage of the pre-test MAP, the LLA occurred at approximately 75% of the baseline MAP. This study also demonstrated that a significant linear relationship between the MAP and LLA (and IT) existed. The

Strangaard study is valuable in that it provides the anaesthetist with a rough guide as to how much reduction of the awake preoperative blood pressure can be tolerated intraoperatively. The important guideline that is created is that a reduction of blood pressure in excess of 20% of baseline should not be tolerated.

Albeit the above guideline is useful, cognizance should be taken of the wide variation in LLA that occurs. Drummond[13], in a letter to the editor of Anesthesiology, pointed out that mean LLA values range between 73 to 91 mm Hg in awake normotensive patients; however LLA values for individuals varied between 41 to 113 mm Hg. This wide variation in LLA represents perhaps one of the limiting factors when dealing with the concept of autoregulation i.e. the anaesthetist does not know the exact limits for any particular patient.

In the hypertensive patient it has been confirmed that the positions of the coronary and cerebral autoregulation curve are shifted to the right.[12,14]

The problem that the anaesthetist is therefore presented with is that the position of the LLA and slope of the pressure dependant portion of the autoregulation is unknown for individual patients. In other words, it is not known at what blood pressure organ blood flow will decrease sufficiently to result in tissue damage. Hence it is prudent that anaesthetists elect not to approach the LLA too closely and remain on the flow independent part of the autoregulation curve.

Changes in pathophysiology with treatment

Rat studies indicate that antihypertensive drug therapy effectively reduces blood pressure. However, from the anaesthetist's perspective, the reduction of blood pressure is not the final aim of antihypertensive treatment. What is of importance to the anaesthetist is preservation of organ blood flow. It is therefore of some significance to note that only if this therapy is effective in reducing blood pressure and is administered for sufficient time, will remodeling of the vasculature occur. Chronic antihypertensive treatment (over an extensive period) represents a method of normalizing autoregulation and lessening the risk of organ underperfusion during anaesthesia.

Two studies are instructive in this regard. In the one study, Hoffman[15] subjected both spontaneous hypertensive (SHR) and normotensive rats to antihypertensive therapy. In the SHR group that was treated with antihypertensive drugs, blood pressure was effectively reduced to that of the control group after 2 weeks of therapy. At this point, they subjected both the treated and untreated animals to hypotensive episodes. There was no difference between cerebral blood flow and oxygen consumption in the treated and untreated SHR groups. The lesson learned from this study is that normalization of blood pressure is not synonymous with normalization of autoregulation.

It is also noteworthy that in the Hoffman study, autoregulation was not restored by 10 weeks of antihypertensive therapy. If it is considered that a single rat month approximates 37 human months, and if we are allowed the liberty of a direct extrapolation of these time periods, it implies that blood pressure must be effectively controlled for years in humans before the anesthetist can rely on a normal autoregulation curve.

Another study[16] demonstrated similar results to the Hoffman study. In this study, a renal artery clip hypertensive rat model was studied. After release of the clip, blood pressure decreased to normal values within 1 week. However, the exaggerated pressor response seen in hypertensives only normalized 19 weeks after removal of the clip.

This perspective presents anaesthesiologists with the following dilemma. Even if the patient is on treatment for hypertension, we cannot assume that autoregulation is normalized until months or years have passed after the therapy was initiated. Therefore, when we are presented with patients that have hypertension who have relatively recently started treatment, we do not know which intraoperative blood pressure should be regarded as "safe" in order to maintain organ blood flow?

Risk to organs

Long standing uncontrolled hypertension is a proven threat to the brain, heart, vasculature, kidneys and eyes. However, in the anaesthetist's perioperative management, the two organs that are paramount are the brain and heart. This does not imply that the other organs can be managed with a casual attitude, but that these two organs present a greater potential to cause acute life threatening crises.

Cardiac risk

The question arises whether hypertension results in an increased cardiovascular risk? It is interesting that the Goldman cardiac risk index[17,18], one of the earliest and best known scoring systems developed to determine perioperative cardiovascular risk, did not include hypertension as one of its parameters. Furthermore, a large study published in 1990 also did not consider hypertension as an independent risk factor in patient undergoing non-cardiac surgery.[19] However, Howell and colleagues[20] did report an association between the history of hypertension and perioperative cardiovascular death. A meta-analysis could not confirm a significant increase in the odds ratio for perioperative cardiac outcome and hypertensive disease (OR 1.35 (1.17 – 1.56)).[21]

At a minimum there is significant doubt whether a clear link has been established between hypertension and perioperative cardiovascular risk with the proviso that certain aspects of the cardiac risk be managed in an effective manner as is explained below.

The earliest studies from the Oxford Group, lead by Prys-Roberts[22,23], warned that the untreated or ineffectively treated hypertensive patient is at risk of both excessive pressor response and myocardial ischaemia. They also reported that, in these hypertensive patients, the perioperative use of β adrenergic receptor blockers reduced the incidence of myocardial ischaemia. In the non β-blocked patients, the incidence of myocardial ischaemia was 38% compared to 4% in those who received practolol. The Oxford Group's studies were conducted in a small group of patients, but were reconfirmed in a larger and more recent study.[24] Stone and colleagues[24] (comparing oxprenolol, acebutolol and labetolol with no treatment in hypertensives) showed that use of β adrenergic blockers in the perioperative period,

reduced the incidence of myocardial ischaemia to much the same figures as reported in the earlier Prys Roberts study. On closer inspection of the Stone data, it is evident that the peak blood pressures recorded during intubation and extubation were within reasonable clinical proximity of each other (except for a group who received labetolol). The untreated patients had mean blood pressures of 125±4 and 127±3 mmHg on intubation and extubation respectively. This compares favorably with the 118±4 and 119±3 mmHg recorded in the patients treated with oxprenolol. However, more consistent differences in heart rate were observed in the Stone study. Data demonstrated that the group who received β adrenergic blockade had lower heart rates during periods of stress compared with non-treated patients. It is reasonable to suggest that the differences in the incidence of myocardial ischaemia between the groups were most probably the result of the slower heart rate associated with β adrenergic blockade and not the insignificant differences in blood pressure per se.

Why is it that the myocardial ischaemia seen during or after laryngoscopy and intubation as well as other periods of stress, appears to be related to increases in heart rate rather than increases in blood pressure? The answers may lie, on the one hand, with the observation that coronary blood flow varies directly with the perfusion pressure in the presence of a critical coronary artery stenosis.[25] This means that small increases in blood pressure will increase or maintain coronary blood flow. On the other hand, the myocardial ischemia associated with periods of stress is most likely the result of an oxygen delivery problem associated with the decrease in diastolic time that accompanies an increase in heart rate.[26]

According to this interpretation of the data, the main cardiac risk for the hypertensive patient is an ischaemic insult. This risk is more closely related to diastolic coronary perfusion time and not the blood pressure. In the hypertensive patient, provided that the pressor response is not so great that it causes the left ventricle (LV) to fail, the logical conclusion is that the major acute perioperative cardiac risk (i.e. myocardial ischaemia) can be managed with β adrenergic blockers. Therefore, the perceived cardiac risks should not deter the anaesthetist from subjecting the hypertensive patient to anaesthesia and surgery, whether the hypertension is controlled or not.

In fact, the popular practice of using β blockers as an adjuvant to reduce cardiovascular stress, acknowledges the above logic. Vascular tone is the result of a balance between β_2 and α adrenergic stimuli. If the β_1 and β_2 receptors are blocked with an appropriate drug, the alpha-receptors are left, so to speak, relatively unopposed. This explains why in many patients there may well be a further small increase in blood pressure when α adrenergic blockers are initiated. Hence, treatment of the expected hypertensive pressor response with a β adrenergic blocker alone defies logic, but in most instances, serendipitously brings the ischemic risk under control by containing the heart rate.

There are 3 other caveats regarding the cardiovascular system and anaesthesia in hypertensive patients.

1. Assidao and Donegan[27] have shown that uncontrolled preoperative hypertension is associated with an increased risk for transient ischemic attacks (TIA) in patients undergoing carotid endarterectomy. This represents one of the few studies and scenarios in which the hypertensive patient has unequivocally been shown to have an increased perioperative risk.
2. It is important to appreciate that, if left ventricular (LV) afterload is high enough to prevent the LV ejecting effectively, the blood pressure requires effective treatment before embarking on anaesthesia. This caveat especially applies to patients with marginal or poor LV performance.
3. There is also the fear that the hypotensive episodes will result in trespassing into the region below the LLM. This hypotensive risk can be managed in the following ways:
   a. Careful fluid preloading, taking cognizance of the 15-200% intravascular fluid deficit,
   b. The use of the synergistic relationship that exists for example between opioids and anaesthesia agents. This synergistic relationship applies to both the reduction in the dose of induction agents needed to produce sleep, as well as the reduction in the MAC of inhalation agents.
   c. Frequent measurements of blood pressure.
   d. The availability of pressor agents to maintain blood pressure if required.

In summary, there is no final and or convincing proof that blood pressure per se represents the major risk in the hypertensive patients when viewed from a cardiac perspective. The real risk appears to be that of an increase in heart rate and myocardial ischaemia; we have drugs that can effectively manage this potential problem. Furthermore, the caveats mentioned emphasize that the approach to the hypertensive patient must be individualized and end-organ function examined before a decision can be made whether or not to accept the current blood pressure for the purpose of anaesthesia.

The Brain

Another organ that is of great concern to the anaesthetist when the topic of hypertension is discussed, is the brain. The concern is that because perioperative blood pressure can vary significantly in hypertensive patients, this may lead to cerebral injury. Because of the position of the autoregulation curve, hypotension would cause hypoperfusion of the brain, whereas high blood pressures would rupture the vessels and cause cerebral bleeding.[28]

Any brain injury (which was caused by excessive pressure and flow variation during anaesthesia) is likely to be evident shortly (hours rather than days) after surgery.

A review by Kam and Calcroft[29] estimates the incidence of perioperative stroke during general surgery at 0.2 – 0.7%. Nonetheless, this incidence of perioperative stroke is modified by several factors. Both Kam and Calcroft[29] and Wong[30] emphasize that the incidence of stroke varies with age. The risk of perioperative stroke in males is 0.38% for subjects over 50, 0.5% over 70, 3.54% over 80 and 3.2% over the age of 90. The stroke usually occurred well into the postoperative period, happening on average on the 7th postoperative day.

Moreover, a prior history of cerebrovascular pathology has

been shown to be associated with a 10 fold increase in the incidence of perioperative stroke.[31]

The association between hypertension and postoperative stroke has been examined by a limited number of studies. A study often referred to is that of Parikh and Cohen.[32] This retrospective study with matched controls studied 24641 patients and reported a perioperative stroke rate of 0.08%. On close inspection of the paper, issues are raised which belie the conclusions the authors made i.e. hypertension is a risk for perioperative stroke. Firstly, 6 of their 20 patients that suffered a stroke had atrial fibrillation (AF), a major risk factor in itself for perioperative stroke. Secondly, one patient had an episode of significant hypertension in the postoperative period. A subsequent CT scan revealed the patient had suffered a cerebral bleed. This begs the question as to whether the bleed or the hypertension occurred first. Lastly, if both the published stroke rate for the 67 year average age in this study and also the atrial fibrillation group is taken into account, it becomes clear that the conclusions of this study, that hypertension is a risk for perioperative stroke, were indeed speculative.

Data studying the association of perioperative stroke and hypertension suffers from a problem with the definition of what is meant by the term "perioperative"? It is the opinion of the authors that if blood pressure fluctuations, be it hyper- or hypotension, are indeed the cause for cerebral injury, it should be present immediately or very soon after the anaesthetic. We are critical of studies that employ, for instance, a 30-day postoperative evaluation period as a reflection of the effects of blood pressure fluctuations during anesthesia on brain tissue. While it may be acceptable to term this period "perioperative", it is probably not acceptable and speculative to attribute a stroke to blood pressure variation occurring during the procedure.

It is relevant to this discussion that the data from a study conducted by Hart and Hindman[33] be considered. This study showed that stroke was associated with hypotension that occurred in the postoperative recovery period. There was no association between stroke and intraoperative events and this study emphasizes the necessity for close control of blood pressure into the post operative period.

The role of AF is repeatedly stressed as one of the major causes of postoperative stroke. The data from Hart and Hindman[32] reveals that in 42% of their cases, stroke was cardiogenic in origin with AF being the most important single factor.

Larsen[34] found 9 postoperative strokes in 2463 surgical patients. However, on close examination of this data, the majority of the strokes occurred after the 5th postoperative day. In addition, 3 patients had AF and only 4 had hypertension. Of those 4 patients with hypertension, 2 had AF. Furthermore, only two patients did not have other known risk factors such as previous cerebral incidents and dementia. If the 2 patients with AF in the hypertensive group are discounted, only two patients who suffered a stroke postoperatively (from day 5 onwards) had hypertension. Also, it is reasonable to ask why the authors considered that stroke on or after the 5th postoperative day was related to anaesthesia. This interpretation gives a different perspective of the data as presented by the authors.

In summary, studies looking at the association between perioperative stroke and hypertension suffer from many limitations, and are difficult to interpret. While logic suggests that tight blood pressure control is to be advocated perioperatively, at least one study disputes the contention that intraoperative hemodynamic instability increases perioperative stroke. Furthermore, there is clear evidence that factors other than perioperative blood pressure fluctuations such as AF, increasing age, postoperative hypotension, uncontrolled hypertension in patients scheduled for carotid endarterectomy, and prior cerebrovascular incidents increase the risk of perioperative stroke.

A time for change?

Decisions to accept hypertensive patients for elective surgery are frequently non-scientific and based on "gut feeling" driven opinions. The available guidelines also reflect personal preference and the uncertainty.[35] In developing a new approach, the following should be considered:

1. The anaesthetist must understand the pathophysiology of the variation in blood pressure that is so often seen in these patients. The initiation of antihypertensive treatment will result in rapid reductions in blood pressure. However, the anaesthetist may be misled to accept the new lower pressure as a reference point. It must be remembered that vascular remodeling is still taking place and both the pressor response and autoregulation are still that of the hypertensive patient. It probably requires many months or even years of effective treatment before it is reasonable to accept that vascular remodeling has normalized. One must not be misled by the dangerous practice of colleagues who start treatment in their rooms and admit the patient one week later with a normal blood pressure.
2. Our (extrapolated) knowledge about the change in the autoregulation curve must be meticulously applied. This means that variations of more than 20% in the preoperative mean arterial pressure should not be allowed.
3. Contingency plans should be made to measure, prevent and treat hypotension by restoration of the intravascular volume and use of α adrenergic agonists. Hypertensive episodes can easily be managed firstly by ensuring a proper surgical level of anaesthesia and secondly, with the use of intravenous vasodilators.
4. It appears that the cardiac risk, is primarily that of myocardial ischaemia. To a large extent, heart rate control will contain this problem. However, it must also be understood that β adrenergic blockers will not contain the pressor response to stressful stimuli. As mentioned, administration of an intravenous vasodilator may be required to control pressure surges.
5. Literature seems to suggest an increased perioperative risk of stroke in hypertensive subjects. However, rigorous interrogation of the published data indicates that the real risk of stroke appears to be a very uncertain issue.
   a. Nonetheless, cognizance must be taken of the fact

that patients with prior cerebral incidents appear to be at a higher risk of sustaining postoperative cerebral events.

b. Furthermore, carotid artery surgery results in a higher incidence of cerebral events in patients who have elevated blood pressure preoperatively. In this subgroup of patients, the available data suggests that preoperative blood pressure control is indeed wise.

6. The blood pressure that the anesthetist should accept must be individualized. For instance, patients with a raised blood pressure who is either in congestive heart failure, or is scheduled for carotid endarterectomy, have specific and understood reasons why the blood pressure needs to be controlled before embarking on elective surgery.

In the light of better knowledge of the pathophysiology, appreciation of the above proviso's, combined with anaesthetic skills and the judicious use of the appropriate drugs, the often quoted views on hypertension in the perioperative period need to be reconsidered.

References

1. *Leitschuh M, Chobanian A. Vascular changes in hypertension. Med Clin North America 1987; 71: 827 – 839.*
2. *Van Citters RL. Occlusion of lumina in small arterioles during vasoconstriction. Circ Res 1966; 18: 199 – 204.*
3. *Folkow B. The hemodynamic consequences of adaptive structural changes of the resistance vessels in hypertension. Clinical Sciences 1971; 41: 1 – 12.*
4. *Folkow B, Grimby G, Thulesius O. Adaptive structural changes of the vascular walls in hypertension and their relation to the control of the peripheral resistance. Acta Physiologica Scandinavica 1956; 44: 255 – 272.*
5. *Sivertsson R, Olander R. Aspects of the nature of increased vascular resistance and increased "reactivity" to noradrenaline in hypertensive subjects. Life Sciences 1968; 7: 1291 – 1297*
6. *Giancoli D. In : Physics. Principles and Applications. Prentice Hall , 5th Ed, 1988; 295 and 532.*
7. *Bauer JH, Brooks CS. Body fluid composition in normal and hypertensive man. Clinical Science 1982; 62: 43 – 49.*
8. *Dagnino J, Prys Roberts C. Strategy for patients with hypertensive heart disease. Clin Anesth 1989; 3: 261.*
9. *Pagel P, Grossman W, Haering JM, Warltier DC. Left ventricular diastolic function in the normal and diseased heart. Anesthesiology 1993; 79: 836 - 854.*
10. *Dorland's Illustrated Medical Dictionary. 25th edition. WB Saunders, Philadelphia, London, Toronto. 11.*
11. *Drummond J, Sharpiro H. Cerebral physiology In: Anesthesia . Ed RD Miller 4th Ed, Churchill Livingstone, New York 1994; 711.*
12. *Strangaard S, Oleson J, Skinhoj E et al. Autoregulation of brain circulation in severe arterial hypertension. Br Med J 1973; 1: 507 - 509.*
13. *Drummond J. The lower limit of autoregulation. Time to revise our thinking ? Anesthesiology 1997; 86: 1431 – 1432.*
14. *Polese A, DeCesare N, Montorsi P et al Upward shift of the lower range of coronary flow autoregulation in hypertensive patients with hypertrophy of the left ventricle. Circulation 1991; 83: 845 - 853.*
15. *Hoffman WE, Miletich DJ, Albrecht RF. The influence of antihypertensive therapy on cerebral autoregulation in aged hypertensive rats. Stroke 1982; 13: 701 - 705.*
16. *Lundgren Y. Regression of cardiovascular changes after reversal of experimental hypertension in rats. Acta Physiol Scand 1974; 91: 275 - 283.*
17. *Goldman L, Caldera D, Nussbaum SR et al. Multifactorial index of cardiac risk in non-cardiac surgical procedures. N Eng J Med 1977; 297: 845 - 850.*
18. *Goldman L, Caldera D. Risk of general anesthesia and elective operation in the hypertensive patient. Anesthesiology 1979; 50: 285 - 292.*
19. *Mangano D, Browner W, Hollenberg M et al. Association of preoperative myocardial ischemia with cardiac morbidity and mortality in men undergoing non-cardiac surgery. N Eng J Med 1990; 323: 1781 - 1787.*
20. *Howell SJ, Hemming AE, Allman KG et al. Predictors of postoperative myocardial ischemia. The role of intercurrent arterial hypertension and other cardiovascular risk factors. Anaesthesia 1996; 57: 1781.*
21. *Howell SJ, Sear J, Foex P. Hypertension, the hypertensive heart disease and perioperative cardiac risk. Br J Anaesth 2004; 92: 570 - 583.*
22. *Prys Roberts C, Foex P, Biro G et al. Studies of anaesthesia in relation to hypertension. V Adrenergic beta receptor blockade. Br J Anaesth 1973; 45: 671 – 681.*
23. *Prys Roberts C, Meloche R, Foex P. Studies of anaesthesia in relation to hypertension. I Cardiovascular responses of treated and untreated patients. Br J Anaesth 1971;43: 122- 137.*
24. *Stone JG, Foex P, Sear J et al. Myocardial ischemia in untreated hypertensive patient: Effect of a single small oral dose of beta adrenergic blocking agent. Anesthesiology 1988; 68: 495 - 500.*
25. *Coetzee A, Foex P, Ryder A. Coronary blood flow during variation in blood pressure. S Afr Med J 1985; 68; 15.*
26. *Coetzee A, Coetzee G. Beta-adrenergic blockade during ischemia. J Cardiothorac Vasc Anesth 2002; 16: 670 – 679.*
27. *Assidao C, Donegan JH. Factors associated with perioperative complications during carotid endarterectomy. Anesth and Analg 1982; 61: 631.*
28. *Mayham WG, Faraci FM, Heistad D. Disruption of the blood brain barrier in the cerebrum and brain stem during acute hypertension. Am J Physiol 1986; 251: H 1171 – H 1175.*
29. *Kam PCA, Calcroft RM. Perioperative stroke in general surgical patients. Anaesthesia 1997; 52: 879 – 883.*
30. *Wong D. Perioperative stroke: general surgery, carotid disease and carotid end-arterectomy. Can J Anaesth 1991; 38: 347 – 373.*
31. *Landecasper J, Metz EJ, Coghill TH et al. Perioperative stroke risk in 173 consecutive patients with a past history of stroke. Arch Surg 1990; 125: 986 – 989.*
32. *Parikh S, Cohen JR. Perioperative stroke after general surgical procedures. NY State J Med 1993; 93; 162 – 165.*
33. *Hart R, Hindman B. Mechanisms of perioperative cerebral infarction. Stroke 1982; 13: 766 - 773.*
34. *Larsen SF, Zaric D, Boysen G. Postoperative cerebrovascular accidents in general surgery. Acta Anaesthesiol Scand 1988; 32: 698 -701.*
35. *Miller pp 937. In: Anesthesia . Ed RD Miller, 4th Ed, Churchill Livingstone, New York, 1994; 937.*