



## Comparison of VAS score in patients administered with the general and regional anesthesia diagnosed with the hypertension undergoing surgeries

Dr. Bhuvneshwar Kumar<sup>1</sup>, Dr. Satyeshwar Jha<sup>2</sup>, Dr. Kiran Bharati<sup>3</sup>

<sup>1</sup> Assistant Professor, Department of Anaesthesia, Darbhanga Medical College & Hospital, Laheriasarai, Darbhanga, Bihar, India

<sup>2</sup> Anesthesia, Associate Professor & HOD, Department of Anaesthesia, Darbhanga Medical College & hospital, Laheriasarai, Darbhanga, Bihar, India

<sup>3</sup> Department of Obstetrics and Gynaecology, Govt. medical College, Bettiah, W. Champaran, Bihar, India

\* Corresponding Author: Dr. Satyeshwar Jha

### Abstract

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.[10] In India, the prevalence of hypertension is 28%–32% in the urban population and 27.6% in the rural population.[11] 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%. Hence based on above findings the present study was planned for Comparison of VAS Score in Patients Administered with the General and Regional Anesthesia Diagnosed with the Hypertension Undergoing Surgeries.

The present study was planned in Department of Anaesthesia, Darbhanga Medical College and Hospital, Laheriasarai, Darbhanga, Bihar, India. Total 30 patients diagnosed with the hypertension undergoing surgeries under general and regional anesthesia were enrolled in the present study. General anesthesia was initiated with Fentanyl 2µg kg-1 and propofol 2.5 mg kg-1. Anesthesia was kept up with 0.7-1.5% end-tidal sevoflurane after orotracheal intubation encouraged by atracurium 0.5 mg kg-1. Regional anaesthesia was administered using spinal block with a 25 Gauge spinal needle between the L3–L4 intervertebral spaces. Regional anaesthesia was achieved by 3.2ml-3.6ml accordingly hyperbaric bupivacaine injection. Hypertension is a modifiable risk factor for cardiovascular diseases and outcomes, and the same is true of the patient presenting for surgery. Adequate blood pressure control must be maintained in all three perioperative (pre, intra and postoperative) settings, as its instability is associated with multiple adverse events. There is increased intra-operative fluctuations and reduced post-operative pain among the patients in the regional anaesthesia group in comparison to those under general anaesthesia group.

**Keywords:** general anaesthesia, hypertension, regional anaesthesia, visual analog scale (VAS) pain, etc

### Introduction

High blood pressure (BP), or hypertension, is defined by two levels by 2017 American College of Cardiology/American Heart Association (ACC/AHA) guidelines<sup>[1, 2]</sup>: (1) elevated BP, with a systolic pressure (SBP) between 120 and 129 mm Hg and diastolic pressure (DBP) less than 80 mm Hg, and (2) stage 1 hypertension, with an SBP of 130 to 139 mm Hg or a DBP of 80 to 89 mm Hg. Hypertension is the most common primary diagnosis in the United States [3]. It affects approximately 86 million adults (≥20 years) in the United States<sup>[4]</sup> and is a major risk factor for stroke, myocardial infarction, vascular disease, and chronic kidney disease.

Compelling indications for specific agents include comorbidities such as heart failure, ischemic heart disease, chronic kidney disease, and diabetes. Drug intolerance or contraindications may also be factors<sup>[5]</sup>.

The AHA/ASA recommends a diet that is low in sodium, is high in potassium, and promotes the consumption of fruits, vegetables, and low-fat dairy products for reducing BP and lowering the risk of stroke. Other recommendations include increasing physical activity (30 minutes or more of

moderate intensity activity on a daily basis) and losing weight (for overweight and obese persons).

The 2018 European Society of Cardiology (ESC) and the European Society of Hypertension (ESH) guidelines recommend a low-sodium diet (limited to 2 g per day) as well as reducing body-mass index (BMI) to 20-25 kg/m<sup>2</sup> and waist circumference (to < 94 cm in men and < 80 cm in women).

If lifestyle modifications are insufficient to achieve the goal BP, there are several drug options for treating and managing hypertension. Thiazide diuretics, an angiotensin-converting enzyme inhibitor (ACEI) /angiotensin receptor blocker (ARB), or calcium channel blocker (CCB) are the preferred agents in nonblack populations, whereas CCBs or thiazide diuretics are favored in black hypertensive populations. These recommendations do not exclude the use of ACE inhibitors or ARBs in treatment of black patients, or CCBs or diuretics in non-black persons. Often, patients require several antihypertensive agents to achieve adequate BP control.

High blood pressure, or hypertension, is the most common primary diagnosis in the United States<sup>[3]</sup>, and it is one of the

most common worldwide diseases afflicting humans and is a major risk factor for stroke, myocardial infarction, vascular disease, and chronic kidney disease. Despite extensive research over the past several decades, the etiology of most cases of adult hypertension is still unknown, and control of blood pressure is suboptimal in the general population. Due to the associated morbidity and mortality and cost to society, preventing and treating hypertension is an important public health challenge. Fortunately, recent advances and trials in hypertension research are leading to an increased understanding of the pathophysiology of hypertension and the promise for novel pharmacologic and interventional treatments for this widespread disease.

According to the American Heart Association (AHA), approximately 86 million adults (34%) in the United States are affected by hypertension, which is defined as a systolic blood pressure (SBP) of 140 mm Hg or more or a diastolic blood pressure (DBP) of 90 mm Hg or more, taking antihypertensive medication, or having been told by clinicians on at least 2 occasions as having hypertension<sup>[4]</sup>. Substantial improvements have been made with regard to enhancing awareness and treatment of hypertension. However, a National Health Examination Survey (NHANES) spanning 2011-2014 revealed that 34% of US adults aged 20 years and older are hypertensive and NHANES 2013-2014 data showed that 15.9% of these hypertensive adults are unaware they are hypertensive; these data have increased from NHANES 2005-2006 data that showed 29% of US adults aged 18 years and older were hypertensive and that 7% of these hypertensive adults had never been told that they had hypertension<sup>[4]</sup>.

Furthermore, of those with high blood pressure (BP), 78% were aware they were hypertensive, 68% were being treated with antihypertensive agents, and only 64% of treated individuals had controlled hypertension<sup>[4]</sup>. In addition, previous data from NHANES estimated that 52.6% (NHANES 2009-2010) to 55.8% (NHANES 1999-2000) of adults aged 20 years and older have prehypertension, defined as an untreated SBP of 120-139 mm Hg or untreated DBP of 80-89 mmHg<sup>[4]</sup>.

Data from the Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC 7), which was released in 2003, were relatively similar to the NHANES data. The JNC 7 noted that approximately 30% of adults were unaware of their hypertension; up to 40% of people with hypertension were not receiving treatment; and, of those treated, up to 67% did not have their BP controlled to less than 140/90 mm Hg<sup>[5]</sup>.

Hypertension is the most important modifiable risk factor for coronary heart disease (the leading cause of death in North America), stroke (the third leading cause), congestive heart failure, end-stage renal disease, and peripheral vascular disease. Therefore, health care professionals must not only identify and treat patients with hypertension but also promote a healthy lifestyle and preventive strategies to decrease the prevalence of hypertension in the general population.

Defining abnormally high blood pressure (BP) is extremely difficult and arbitrary. Furthermore, the relationship between systemic arterial pressure and morbidity appears to be quantitative rather than qualitative. A level for high BP must be agreed upon in clinical practice for screening

patients with hypertension and for instituting diagnostic evaluation and initiating therapy. Because the risk to an individual patient may correlate with the severity of hypertension, a classification system is essential for making decisions about aggressiveness of treatment or therapeutic interventions.

The classification above is based on the average of 2 or more readings taken at each of 2 or more visits after initial screening<sup>[5, 7]</sup>. Normal BP with respect to cardiovascular risk is less than 120/80 mm Hg. However, unusually low readings should be evaluated for clinical significance.

Prehypertension, a new category designated in the JNC 7 report, emphasizes that patients with prehypertension are at risk for progression to hypertension and that lifestyle modifications are important preventive strategies.

However, the 2017 ACC/AHA guidelines eliminate the classification of prehypertension and divides it into two levels<sup>[1, 2]</sup>: (1) elevated BP, with a systolic pressure (SBP) between 120 and 129 mm Hg and diastolic pressure (DBP) less than 80 mm Hg, and (2) stage 1 hypertension, with an SBP of 130 to 139 mm Hg or a DBP of 80 to 89 mm Hg.

From another perspective, hypertension may be categorized as either essential or secondary. Primary (essential) hypertension is diagnosed in the absence of an identifiable secondary cause. Approximately 90-95% of adults with hypertension have primary hypertension, whereas secondary hypertension accounts for around 5-10% of the cases. However, secondary forms of hypertension, such as primary hyperaldosteronism, account for 20% of resistant hypertension (hypertension in which BP is >140/90 mm Hg despite the use of medications from 3 or more drug classes, 1 of which is a thiazide diuretic).

Especially severe cases of hypertension, or hypertensive crises, are defined as a BP of more than 180/120 mm Hg and may be further categorized as hypertensive emergencies or urgencies. Hypertensive emergencies are characterized by evidence of impending or progressive target organ dysfunction, whereas hypertensive urgencies are those situations without progressive target organ dysfunction. In hypertensive emergencies, the BP should be aggressively lowered within minutes to an hour by no more than 25%, and then lowered to 160/100-110 mm Hg within the next 2-6 hours<sup>[5]</sup>.

The pathogenesis of essential hypertension is multifactorial and complex. Multiple factors modulate the blood pressure (BP) including humoral mediators, vascular reactivity, circulating blood volume, vascular caliber, blood viscosity, cardiac output, blood vessel elasticity, and neural stimulation. A possible pathogenesis of essential hypertension has been proposed in which multiple factors, including genetic predisposition, excess dietary salt intake, and adrenergic tone, may interact to produce hypertension. Although genetics appears to contribute, the exact mechanisms underlying essential hypertension have not been established.

Investigations into the pathophysiology of hypertension, both in animals and humans, have revealed that hypertension may have an immunological basis. Studies have revealed that hypertension is associated with renal infiltration of immune cells and that pharmacologic immunosuppression (such as with the drug mycophenolate mofetil) or pathologic immunosuppression (such as occurs with HIV) results in reduced blood pressure in animals and humans. Evidence suggests that T lymphocytes and T-cell

derived cytokines (eg, interleukin 17, tumor necrosis factor alpha) play an important role in hypertension [8].

One hypothesis is that prehypertension results in oxidation of lipids such as arachidonic acid that leads to the formation of isoketals or isolevuglandins, which function as neoantigens, which are then presented to T cells, leading to T-cell activation and infiltration of critical organs (eg, kidney, vasculature). This results in persistent or severe hypertension and end organ damage. Sympathetic nervous system activation and noradrenergic stimuli have also been shown to promote T-lymphocyte activation and infiltration and contribute to the pathophysiology of hypertension.

The natural history of essential hypertension evolves from occasional to established hypertension. After a long invariable asymptomatic period, persistent hypertension develops into complicated hypertension, in which end-organ damage to the aorta and small arteries, heart, kidneys, retina, and central nervous system is evident.

As evident from the above, younger individuals may present with hypertension associated with an elevated cardiac output (high-output hypertension). High-output hypertension results from volume and sodium retention by the kidney, leading to increased stroke volume and, often, with cardiac stimulation by adrenergic hyperactivity. Systemic vascular resistance is generally not increased at such earlier stages of hypertension. As hypertension is sustained, however, vascular adaptations including remodeling, vasoconstriction, and vascular rarefaction occur, leading to increased systemic vascular resistance. In this situation, cardiac output is generally normal or slightly reduced, and circulating blood volume is normal.

Cortisol reactivity, an index of hypothalamic-pituitary-adrenal function, may be another mechanism by which psychosocial stress is associated with future hypertension. In a prospective sub-study of the Whitehall II cohort, with 3 years follow-up of an occupational cohort in previously healthy patients, investigators reported 15.9% of the patient sample developed hypertension in response to laboratory-induced mental stressors and found an association between cortisol stress reactivity and incident hypertension [9].

Hypertension may be primary, which may develop as a result of environmental or genetic causes, or secondary, which has multiple etiologies, including renal, vascular, and endocrine causes. Primary or essential hypertension accounts for 90-95% of adult cases, and a small percentage of patients (2-10%) have a secondary cause. Hypertensive emergencies are most often precipitated by inadequate medication or poor compliance.

Hypertension develops secondary to environmental factors, as well as multiple genes, whose inheritance appears to be complex [14, 23]. Furthermore, obesity, diabetes, and heart disease also have genetic components and contribute to hypertension. Epidemiologic studies using twin data and data from Framingham Heart Study families reveal that BP has a substantial heritable component, ranging from 33-57%.

In an attempt to elucidate the genetic components of hypertension, multiple genome wide association studies (GWAS) have been conducted, revealing multiple gene loci in known pathways of hypertension as well as some novel genes with no known link to hypertension as of yet. Further research into these novel genes, some of which are immune-related, will likely increase the understanding of hypertension's pathophysiology, allowing for increased risk

stratification and individualized treatment.

Epigenetic phenomena, such as DNA methylation and histone modification, have also been implicated in the pathogenesis of hypertension. For example, a high-salt diet appears to unmask nephron development caused by methylation. Maternal water deprivation and protein restriction during pregnancy increase renin-angiotensin expression in the fetus. Mental stress induces a DNA methylase, which enhances autonomic responsiveness. The pattern of serine protease inhibitor gene methylation predicts preeclampsia in pregnant women.

Despite these genetic findings, targeted genetic therapy seems to have little impact on hypertension. In the general population, not only does it appear that individual and joint genetic mutations have very small effects on BP levels, but it has not been shown that any of these genetic abnormalities are responsible for any applicable percentage of cases of hypertension in the general population.

Secondary causes of hypertension related to single genes are very rare. They include Liddle syndrome, glucocorticoid-remediable hyperaldosteronism, 11 beta-hydroxylase and 17 alpha-hydroxylase deficiencies, the syndrome of apparent mineralocorticoid excess, and pseudohypoaldosteronism type II [5].

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 [10]. In India, the prevalence of hypertension is 28%–32% in the urban population and 27.6% in the rural population [11]. 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% [12]. Hence based on above findings the present study was planned for Comparison of VAS Score in Patients Administered with the General and Regional Anesthesia Diagnosed with the Hypertension Undergoing Surgeries.

### Aim & Objective

To compare VAS score in patients with Hypertension undergoing under General and Regional anesthesia.

### Methodology

The present study was planned in Department of Anaesthesia, Darbhanga Medical College and Hospital, Laheriasarai, Darbhanga, Bihar, India. Total 30 patients diagnosed with the hypertension undergoing surgeries under general and regional anesthesia were enrolled in the present study. General anesthesia was initiated with Fentanyl 2µg kg-1 and propofol 2.5 mg kg-1. Anesthesia was kept up with 0.7-1.5% end-tidal sevoflurane after orotracheal intubation encouraged by atracurium 0.5 mg kg-1. Regional anaesthesia was administered using spinal block with a 25 Gauge spinal needle between the L3–L4 intervertebral spaces. Regional anaesthesia was achieved by 3.2ml-3.6ml accordingly hyperbaric bupivacaine injection.

All the patients were informed consents. The aim and the objective of the present study were conveyed to them. Approval of the institutional ethical committee was taken prior to conduct of this study.

**Results & Discussion**

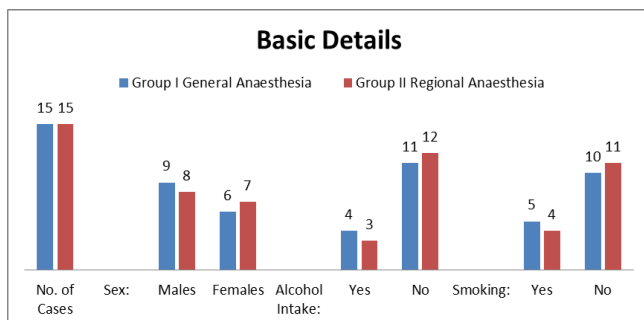
For the anaesthesiologist, there are two main concerns: Should the diagnosis or detection of hypertension lead to further testing and/or postponement of the planned surgery and if surgery does proceed, what would be the expected outcome of the patient's perioperative journey? Although long-standing hypertension 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 [13]; and yet, uncontrolled hypertension constitutes a major reason for cancellation of elective surgeries [14]. 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.

Perioperative hypertension presents at different times around the surgery: induction of anesthesia (especially if no opioid analgesia is used), intraoperatively (associated with acute pain-induced sympathetic stimulation that leads to vasoconstriction), or in the early post anesthesia period (associated with pain-induced sympathetic stimulation, hypothermia, hypoxia, or as a result of intravascular volume overload from excessive intraoperative intravenous fluid therapy). Post anesthesia hypertension associated with volume overload can remain for 24–48 hours until adequate mobilization of fluid from the extravascular space occurs. As stated previously, elevation of blood pressure is also associated to preoperative discontinuation of antihypertensive medications [15].

The increased activity of the sympathetic nervous system is the main driver of intraoperative hypertension, and this is associated with tachycardia and arrhythmia. And this might be a result of inadequate analgesia or anesthesia, surgical stimulation, or due to airway manipulation from the laryngoscopy and extubation. Other causes of hypertension during anesthesia are related to the hypoxemia and hypercapnia, or related to overdose of the drugs being used intraoperatively such as vasoconstrictors and inotropes. However, all instances of intraoperative hypertension must prompt exclusion of awareness and malignant hyperthermia as the cause [16].

**Table 1:** Basic Characteristics

Groups	Group I	Group II
Administration of	General Anaesthesia	Regional Anaesthesia
No. of Cases	15	15
Age (years)	40 – 58	38 – 62
Weight (kg)	68 – 85	72 – 84
BMI kg/cm2	22.1 – 25.3	21.9 – 26.1



**Fig 1:** Basic Details

**Table 2:** Mean VAS Score

Groups	Group I	Group II
Administration of	General Anaesthesia	Regional Anaesthesia
No. of Cases	25	25
Time		
0 hr	1	1
1 hr	3	1
2 hr	4	1
3 hr	5	1
4 hr	3	2
5 hr	2	3
6 hr	1	4
8 hr	1	2
12 hr	1	1

Howell *et al.* [17] 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.

Intraoperative haemodynamic abnormalities are associated with peri- and postoperative cardiovascular events (cardiac death, myocardial infarction or stroke). Indeed, bradycardia, tachycardia, hypotension and hypertension (including pulmonary hypertension) have been correlated significantly with cardiovascular complications of coronary bypass surgery. As patients with uncontrolled hypertension are very likely to develop major haemodynamic abnormalities, these data suggest indirectly that preoperative treatment of hypertension should be beneficial.

Sympathetic activation during the induction of anesthesia can cause the blood pressure to rise by 20 to 30 mmHg and the heart rate to increase by 15 to 20 beats per minute in normotensive individuals [18]. These responses may be more pronounced in patients with untreated hypertension in whom the systolic blood pressure can increase by 90 mmHg and the heart rate by 40 beats per minute.

The mean arterial pressure tends to fall as the period of anesthesia progresses due to a variety of factors, including direct effects of the anesthetic, inhibition of the sympathetic nervous system, and loss of the baroreceptor reflex control of arterial pressure. These changes can result in episodes of intraoperative hypotension. Patients with preexisting hypertension are more likely to experience intraoperative blood pressure lability (either hypotension or hypertension) [19], which may lead to myocardial ischemia [20].

Blood pressure and heart rate slowly increase as patients recover from the effects of anesthesia during the immediate postoperative period. Hypertensive individuals, in particular, may experience significant increases in these parameters [21].

**Conclusion**

Hypertension is a modifiable risk factor for cardiovascular

diseases and outcomes, and the same is true of the patient presenting for surgery. Adequate blood pressure control must be maintained in all three perioperative (pre, intra and postoperative) settings, as its instability is associated with multiple adverse events. There is increased intra-operative fluctuations and reduced post-operative pain among the patients in the regional anaesthesia group in comparison to those under general anaesthesia group.

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