Cardiovascular Response during Induction, Laryngoscopy, Intubation and Surgery in Normotensive and Controlled Hypertensive Patients Undergoing Abdominal Surgeries under General Anaesthesia

Abrar Mohammad[1]

ABSTRACT

Introduction: Hypertension is very common condition among adult and elderly patients. Ii is one of the important risk factor of atherosclerosis of vessels. Anesthetist's more commonly tackle the management of treated and untreated hypertensive patients. The considerable concern of anaesthtist not only strong association with coronary artery disease but potential target organ damage. This study was conducted to assess cardiovascular response in different stages of general anesthesia in abdominal surgeries.

Material and methods: Sixty adult patients undergoing different elective abdominal operations under general anesthesia were included. Patients were divided into A and B group. A group consists of normotensive patients while B group consists of controlled hypertensive patients. Their blood pressure were controlled on single antihypertensive drug. B group further divided into B1 and B2. B1 group patients were on beta blockers (BB) while B2 group patients were on angiotensin converting enzyme inhibitors(ACEI).

Results: There was decrease in SAP, DP and HR in all groups after induction while there was increase in SAP, DP and HR in all groups after laryngoscopy and intubation. There was minimal rise in SAP, DP and HR in all groups during surgery. The rise of HR was comparatively less in BB group than ACEI group during laryngoscopy and intubation while rise in SAP was comparatively more in ACEI than BB group during surgery.

Conclusion: On the basis of the present observation it can be concluded that pressure response and cardiac response (cardiovascular response) to laryngoscopy and intubation appears to be unaffected with these antihypertensive drugs.

Keywords: Cardiovascular Response, General Anesthesia, Abdominal Surgery, Controlled Hypertension

INTRODUCTION

Introduction of general anesthesia is known to induce clinically relevant changes in hemodynamic variables probably generated by direct laryngoscopy and endotracheal intubation. Tracheal intubation causes a reflex increase in sympathetic activity that may result in hypertension, tachycardia and arrhythmia.[1-3] A change in plasma catecholamine concentration have been demonstrated to the part of stress response to tracheal intubation.[4]

Since the first description in 1940, hemodynamic response to laryngoscopy and intubation.[4] It is well recognized response to laryngoscopy and intubation. The rise in pulse and blood pressure are usually transitory and unpredictable. These changes are of no consequence and are well tolerated by healthy individuals. But in patients with hypertension, heart disease and coronary artery disease, these changes can result in increase in cardiac work load.[5]

The rise in blood pressure has also a significant importance in neurosurgical patient. sudden rise in blood pressure as seen during laryngoscopy and intubation can result in a sudden rise in intracranial pressure and consequently, acute cerebral edema and herniation of brain. The aim of the study was conducted to assess cardiovascular response in different stages of general anesthesia in abdominal surgeries.

MATERIAL AND METHODS

This was a prospective study conducted at CIMSH during 2016-2017 on Sixty adult patients undergoing different elective abdominal operations under general anesthesia were included. Patients were divided into A and B group. A group consists of normotensive patients while B group consists of controlled hypertensive patients. Their blood pressure was controlled by single antihypertensive drug. B group further divided into B1 and B2. B1 group patients were on beta blockers (BB) while B2 group patients were on angiotensin converting enzyme inhibitors(ACEI). Out of sixty patients,20 were from A group while 20 each were from B1 and B2 group. Preoperatively these patients underwent CBC, urine analysis, serum biochemistry (blood sugar, serum cholesterol serum creatinine, serum electrolytes), cardiac evaluation (ECG) and x-ray chest PA view where ever required. Administration of general anaesthesia (GA) was started with premedication with 10 mg diazepam orally night before surgery and 10 mg diazepam orally 2 hours before surgery. Glycopyrrolate (0.2mg) as anticholinergic was administered intramuscularly 45 minutes before induction. Prior to induction of anaesthesia, intravenous line was maintained with 20-22 gauze intravenous catheter and Pentazocine 15 mg plus promethazine hydrochloride 5 mg slowly intravenously were given. After pre-oxygenation for

[1]Assistant Professor, Department of Anesthesia, Prasad Institute of Medical Sciences, Lucknow, India

Corresponding author: Dr. Abrar Mohammad, Assistant Professor, Department of Anesthesia, Prasad Institute of Medical Sciences, India

How to cite this article: Abrar Mohammad. Cardiovascular response during induction, laryngoscopy, intubation and surgery in normotensive and controlled hypertensive patients undergoing abdominal surgeries under general anaesthesia. International Journal of Contemporary Medical Research 2019;6(8):H22-H25.

DOI: http://dx.doi.org/10.21276/ijcmr.2019.6.8.41

3 minutes' patients were induced with 2.5% sodium pentothal in a dose of 4-6mg/kg body weight till sleep dose were achieved, followed by Suxamethonium in the dose of 1-1.5 mg/ kg body weight was given. Following this intubation was done with appropriate size of cuffed endotracheal tube with minimal manipulation. Following above intubation anaesthesia was maintained with mixture of oxygen, nitrous oxide (40-60%}, All these patients do not have any history of coronary artery or other known complication of hypertension. Vitals like systolic arterial pressure (SAP) and diastolic pressure(DP), heart rate(HR) were recorded before induction, after induction, after laryngoscopy and intubation, during surgery were recorded. The data were collected, tabulated, analysed and following observation was made. halothane (0.5-1%) and Vancronium 60-100 microgram /kg body weight. Continuous vital recording was done during pre and post induction, laryngoscopy and intubation and surgery. Data were collected and analysed and following observations were made.

RESULTS

Table 1 showing SAP in different group of patients in various stages of anesthesia. After induction there is fall in SA in all group of patients. The maximum fall was observed in group B1. There is rise in SAP in all groups of patients during laryngoscopy and intubation and during surgery. But the maximum rise of 23.4% of SAP was observed in B1 group during laryngoscopy and intubation (Table 1).

Table 2 showing DP in different group of patients in various stages of anesthesia. After induction there is fall in DP in all group of patients. The maximum fall was observed in group B2. There is rise in DP in all groups of patients during laryngoscopy and intubation and during surgery. But the rise of 5.01% of DP was observed in B1 group during laryngoscopy and intubation (Table 2).

Table 3 showing HR in different group of patients in various stages of anesthesia. There was rise in HR in all the groups and in all the stages of anesthesia. The maximum rise of 21.3% was observed during laryngoscopy and intubation in group B2. The rise in HR was minimum after induction but more during laryngoscopy and intubation in B1 group (Table 3).

DISCUSSION

Chronic hypertension is a common preexisting problem associated with surgical patients. Nearly 1/3rd of adult patients presenting for non-cardiac procedures and 2/3rd of those undergoing coronary revascularization are suffering from chronic hypertension.[6] It is associated with long term consequences like coronary ischemic heart disease, heart failure, cerebrovascular disease, renal insufficiency which increases perioperative risk.[7,8]

Myocardial ischemia occurs often during anesthesia.[9-11] It is even more often in untreated or poorly controlled.[12-15] In the latter group of patients, laryngoscopy and intubation can result in significant rise in both heart rate and arterial pressure.[12,13] To attenuate these circulatory changes many methods like topical anesthesia, intravenous lignocaine, vasodilators, beta adrenoreceptor block, narcotics have been tried. Due to above measures, many of the study reported reduced incidence of ECG directed myocardial ischemia.[16,17] Ischemia was related to episodes of tachycardia.[16]

Pryas-Robinson concluded that untreated high arterial pressure constituted a serious risk to patients undergoing

Groups	Before induction	After induction		Laryngoscopy and intubation		During surgery	
	Mean ±SD	Mean ±SD	% fluctuation	Mean ±SD	% Fluctuation	Mean ±SD	% fluctuation
A	122.18±4.429	115.36±2.59	-4.6	132.84±2.64	9.12	128.11±2.32	4.6
B1	130.25±6.26	120.5±3.94	-8.33	161.02±6.95	23.8	134.28±4.87	3.07
B2	120.92±4.26	123.09±3.94	-4.06	135.9±4.42	5.4	130.85±7.98	1.56
A=Normotensive, B1=On BB,B2= on ACEI							

Table-1: Systolic arterial pressure in different group of patients in different stages of anaesthesia

Maximum Groups	Before induction	After induction		Laryngoscopy and intubation		During surgery	
	Mean ±SD	Mean ±SD	% fluctuation	Mean ±SD	% Fluctuation	Mean ±SD	% fluctuation
A	78.39±1.29	76.89±1.385	-1.5	81.89±0.039	3.5	79.99±1.38	1.6
B1	85.27±3.57	84.59±4.058	-0.68	90.82±3.21	5.01	86.99±3.21	1.72
B2	80.80±2.12	78.7±2.295	-2.1	82.82±4.059	2.02	81.34±4.29	0.54
A=Normotensive, B1=On BB, B2= on ACEI							

Table-2: Diastolic pressure in different group of patients and different stages of anaesthesia

Groups	Before induction	After induction		Laryngoscopy and intubation		During surgery	
	Mean ±SD	Mean ±SD	% fluctuation	Mean ±SD	% Fluctuation	Mean ±SD	% fluctuation
A	95.59±2.57	94.89±2.37	2.12	100.29±3.99	8.31	91.81±2.87	3.9
B1	72.92±3.77	74.09±3.03	1.16	82.92±4.098	13.7	77.32±3.22	6.03
B2	82.52±2.9	90.12±3.95	9.1	100.12±6.952	21.32	96.32±6.92	16.72
A=Normotensive, B1=On BB, B2= on ACEI							

Table-3: Heart rate in different group of patients in different stages of anesthesia

anaesthesia and surgery. This conclusion was based on the finding of high incidence of myocardial ischemia related to arterial hypertension during steady state of anesthesia in both treated and untreated patients.[12] Later on study have shown that hypertensive patients as much if not more at risk during induction of anaesthesia and during subsequent laryngoscopy and tracheal intubation as they are during steady state of anesthesia.[17]

In the present study we randomly chosen three end points to stress the potency of various treatment regimen. Systolic arterial pressure(SAP) after induction of anaesthesia of ≤90 mmHg; SAP after laryngoscopy and intubation rising the baseline pre-induction value of 20 mmHg or more and rise in heart rate(HR) more than 20% or more from baseline after laryngoscopy and intubation or HR >100 beat per minute. Some authors have chosen HR >100 or 110 beat per minute, rate pressure product >2000 or pressure –rate ratio >1 end point of myocardial ischemia.[19,22] It is very difficult to say which data is better in the paucity of comparative data. In one publication it was observed that in majority of perioperative ischemic ECG changes occurred without hemodynamic changes before onset of ischemia[18] but in one of the another study, author have noted tachycardia (HR>110 beat/ minute) was only hemodynamic abnormality related to intraoperative ischemia.[19] Both these studies were related to cardiac surgery and unrelated to our study and may not be comparable to hypertensive patient group. A pressure –rate ratio of >1 be in error.[22] High pressure and fast rate yield a normal ratio, yet the fast rate may be a major contributor to ischemia A low pressure and slow rate may also be disastrous.

Hemodynamic swing is more common and exaggerated in hypertensive patients as compared to normotensive patients. Rightward shift of autoregulation in hypertensive patients means that organ perfusion occurs at higher mean arterial pressure as compared to normotensive and thus intraoperative hypotension leads to hypo-perfusion and target organ damage during hypotension.[23] A retrospective study has found that intraoperative hypotension but not hypertension is associated with a higher mortality in hypertensive patients undergoing non-cardiac surgery.[23] Even short period of hypotension (mean arterial pressure < 55mmHg) have been associated with myocardial and renal injury after non cardiac surgery.[25] This does not imply that hypertension should not be treated. It has been observed in one of the study that hypertensive patient and diabetic patients who had cumulative 1hour decrease in mean arterial pressure >20 mmHg or <1hour decrease of >20 mmHg and 15-minute increase of 20 mmHg were at greatest risk of post operative myocardial ischemia.[26] It has been reported in previous studies that patients receiving ß –adreno-receptor blocking agents chronically or as a single premedicant dose, act to reduce tachycardia but do not blunt the pressure response to laryngoscopy and intubation.[26] Similar finding was also observed in the present study. Recently, it has been proposed that angiotensin II may be responsible hemodynamic regulation during stress.[27] So interest of the physician to control hypertension with ACE begun. There are reports of profound hypotension and bradycardia during anaesthesia in patients treated with ACE.[29] However other reports do not support above observation.[30-32] In a recent report it has been observed that cases treated with ACE, if induced with fentanyl and flunitrazepam leads to reduction in arterial pressure accompanying decrease in pulmonary capillary wedge pressure and cardiac index, necessitating treatment with fluid and phenylephrine.[33] The present study also does not support former observation.

CONCLUSION

On the basis of the present observation it can be concluded that pressure response and cardiac response to laryngoscopy and intubation appears to be unaffected with these antihypertensive drugs.

REFERENCES

1. Sharma N, Parikh H. A comparative study of hemodynamic response to intubation: fentanyl versus nalbuphine. GMJ 2014;69:48-53.
2. Stoelting RK. Blood pressure and heart rate changes during short duration laryngoscopy for tracheal intubation: influence of viscus or intravenous lignocaine anaesthesia Analgesia 1978; 57:197-199.
3. Robert PK, Greene LT, Meloche R. Studies of anesthesia in relation to hypertension-II, hemodynamic consequences of induction and endotracheal intubation. Br J Anaesth 1971; 43:541-547.
4. Barak M, Zeiser A, Greenburg A.Lischinsky S,Rosenberg B.hemodynamic and catecholamine response to treacheal intubation direct laryngoscopy compared with fibrotic intubation. J Clin Anaesth. 2003; 15:132-136
5. Reid LC, Brace DE. Irritation of the respiratory tract and its reflex effects upon heart. Surg gynec and Obst 1940;70:157-162.
6. Kaplan Nw. New issues in the treatment of isolated systolic hypertension. Circulation 2000; 102:1079
7. Fleisher LA, Fleischmann KE, Auerbach AD, et al.2014 ACC/AHA guideline on preoperative cardiovascular evaluation and management of patients undergoing non-cardiac surgery: a report of the American College of Cardiology /American Heart Association Task force on practice guideline. Jam Coll Cardiol 2014;64: e77
8. Kristensen SD, Knuti J, Saraste A, et al.2014 ESC/ ESA Guidelines on non-cardiac surgery: cardiovascular assessment and management: The Joint Task Force on non-cardiac surgery, cardiovascular assessment and management of the European Society of Cardiology (ESC) and the European Society of Anaesthesiology (ESA). Eur Heart J 2014;35:2383.
9. Raby KE, Goldman L, Creager MA, Cook EF, Weisberg MC, Whittemore AD, Selwyn AP. Correlation between preoperative ischemia and major cardiac evens after peripheral vascular surgery. New England Journal of Medicine 1989; 321:1296-1300.
10. Lutsch P, Heijke S, Rowbothom D. Preoperative ST abnormalities detected by the compass ambulatory ECG monitor in patients to undergo peripheral arterial surgery. Br J Anaesth 1990; 65:282P.
11. McHug P, Gill NP, Wyld R, Nimmo WS, Reilly CS. Continuous ambulatory ECG monitoring in the

perioperative period: Relationship of preoperative status and outcome. British Journal of Anaesthesia 1991;43: 122-137.
12. Prys-Roberts C, Greene L, Meloche R, Foex P. Studies of anesthesia in relation to hypertension. I. Hemodynamic consequences of induction and endotracheal intubation. Br J Anaesth 1971;122-137.
13. Prys-Roberts C, Greene L, Meloche R, Foex P. Studies of anesthesia in relation to hypertension. II. Hemodynamic consequences of induction and endotracheal intubation. Br J Anaesth 1971; 43:531-546.
14. Muir AD, Reeder MK, Foex P. Preoperative silent ischemia is related to hypertension. British Journal of Anaesthesia 1991; 67:373-377.
15. Muir AD, Reeder MK, Foex P. Preoperative silent ischemia is related to hypertension. British Journal of Anaesthesia 1992; 69:540P.
16. Stone JG, Foex P, Sear WJ, Johonson LL, Khambatta HJ, Triner L. Myocardial ischemia in untreated hypertensive patients: effect of single oral dose of a beta adrenergic blocking agent. Anaesthesiology 1988; 68:495-500.
17. Prys –Roberts C, Greene LT, Meloche R, Foex P. Studies of anaesthesia in relation to hypertension II< Hemodynamic consequences of induction and endotracheal intubation. Brit J Anaesth 1971; 4:531-547
18. Stone JG, Foex P, Sear WJ, Johnson LL, Khambatta HJ, Triner L. Risk of myocardial ischemia during anaesthesia in treated and untreated hypertensive patients. British Journal of Anaesthesia 1988; 61:579-581.
19. Knight AA, Hollenberg M, London MJ,e al. Perioperative myocardial ischemia: importance of the preoperative ischemic pattern. Anaesthesiology 1988; 68:681-688.
20. Slogoff S, Keats AS. Randoised trial of primary anaesthetic agents on outcome of coronary artery bypass operations. Anaesthesiology 1989; 70:179-188.
21. Nelson RR, Gobel FL, Jorgensen CR, et al. Hemodynamic predictor of myocardial oxygen consumption during static and dynamic exercises. Circulation 1974; 50:1179-1189.
22. Buffington CW. Hemodynamic determinant of ischemic myocardial dysfunction in the presence of coronary stenosis in dogs. Anesthesiology 1985; 63:651-662.
23. Lapage KG, Wouters PF. The patients with hypertension undergoing surgery. Curr Opin Anaesthesiol 216;29:397-402.
24. Monk TG, Bonsert MR, Henderson WG, e al. Association between intraoperative hypotension and hypertension and 30-day postoperative mortality in non-cardiac surgery. Anaesthesiology 2015; 123:307-319
25. Walsh M, Devereaux PJ, Garg AX, Kurtz A, Turan A,Rodseth RN, et al. Relaionship between intraoperative mean arterial pressure and clinical outcome after non-cardiac surgery. Towards an empirical definition of hypotension. Anaesthesiology 2013; 119:507-15.
26. LonajaretL, Lairezo,
27. Sear JW, Jewkes C, Sanders D, Verhoeff F, Foex P. B adrenoreceptor block affects only heart rate response to laryngoscopy and intubation in hypertensive patients. British Journal of Anaesthesia 1990; 65:284-285P
28. Miller ED, Longnecker DE,Peach MJ.The regulatory function of the renin- angiotensin system during general anaesthesia. Anaesthesiology 1978; 48:399-403.
29. Katja JHK, Kaukinen S,Viinamaki OVK, et al. Hemodynamic and hormonal changes in patients pretreated with captopril for surgery of the abdominal aorta. Journal ofcardiothoracic Anaesthesia 1989; 3:425-432.
30. Colson P, Grolleau D, Chaptal PA, et al.Effect of preoperative renin-angiotensin system blockade on hypertension following coronary surgery. Chest 1988; 93:1156-1158.
31. Colson P, Ribstein J, Mimran A, et al. Effect of angiotensin converting inhibition on blood pressure and renal function during open heart surgery. Anaesthesiology 1990; 72:23-27
32. Yate AP, Hunter DN. Anesthesia and angiotensin converting enzyme inhibitors. Anaesthesia 1988;43:935-938.
33. Colson P, Sussine M, Seguin JR, et al. Hemodynamic effect of anesthesia in patients chronically treated with angiotensin-converting enzyme inhibitors. Anesthesia and Analgesia 1992; 74:805-3 5808.

Source of Support: Nil; **Conflict of Interest:** None

Submitted: 17-07-2019; **Accepted:** 02-08-2019; **Published:** 28-08-2019