IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 14, Issue 11 Ver. VIII (Nov. 2015), PP 24-29
www.iosrjournals.org

Relative Efficacies of Nitroglycerine Infusion, Sublingual Nifedipine, and Intravenous Hydralazine in Attenuating the Hemodynamic Pressor Response to Direct Laryngoscopy and Tracheal Intubation in Severely Pre-eclamptic patients.

Shabir A. Shabir[1], G.A. Shah[2], Abdul Hakeem[3]

[1.] *Lecturer, Deptt; of Anesthesia & Critical Care. GMC, Srinagar (J&K) INDIA.*
[2.] *Associate Prof., Deptt; of Anesthesia & Critical Care. GMC, Srinagar (J&K) INDIA.*
[3.] *Asstt. Prof. Deptt; of Anesthesia & Critical Care. GMC, Srinagar (J&K) INDIA.*

Abstract
Background*: Pressor response to direct laryngoscopy and tracheal intubation is a very important issue in pre- eclamptic patients, so the study was taken in our Obstetric Anesthesia Department to compare the efficacies of continuous intravenous infusion of nitroglycerine, sublingual nifedipine and intravenous hydralazine in attenuating the pressor response to direct laryngoscopy and tracheal intubation in patients with severe pre-eclampsia undergoing cesarean section under general anesthesia.*
Patients and Methods*: In this prospective randomized observational study, 120 patients undergoing cesarean delivery with severe pre-eclampsia were randomly allocated into 3 groups (each of 40 patients). Each group received one of the following drugs before laryngoscopy and intubation.GROUP-1 received 5mcg/minute of Nitroglycerine infusion continuously. GROUP-2 received 10 mg capsule of Nifedipine deposited sublingually. GROUP-3 received 10 mgs of Hydralazine intravenously.*
Haemodynamic parameters viz; Heart Rate(HR), Systolic Arterial Pressure(SAP), Diastolic Arterial Pressure(DAP) and Mean Arterial Pressure(MAP) were simultaneously recorded in the patients at pre-induction, pre-intubation, and at 1, 3, 5 and 10 minutes post intubation.
Results*: In contrast to GROUP- 2 & GROUP- 3, patients of GROUP- 1 showed no significant rise in HR, SAP, DAP or MAP after intubation when compared to baseline parameters. In addition, the incidence of hypotension was significantly greater in GROUP-2[17(42.5%)] as compared to GROUP-3[10(20%)] or GROUP-1[5(12.5)]patients developed hypotension[p=0.025]*
Conclusions*: In patients with severe pre-eclampsia undergoing cesarean delivery, a continuous IV infusion of Nitroglycerine was able to attenuate the cardiovascular pressor response to a greater extent as compared to, with the use of sublingual Nifedipine or IV Hydralazine, without significant adverse effect on the newborn.*
Keywords*: Pre-eclampsia, Cesarean Section, Nitroglycerine, Nifedipine, Hydralazine.*

I. Introduction

The pressor response to laryngoscopy and tracheal intubation is a very challenging issue in severely pre-eclamptic patients, involving an increase in systemic and pulmonary arterial pressures and also pulmonary capillary wedge pressure[1-4].

In addition, the stress of laryngoscopy can be associated with an increase in intracranial pressure, with the risk of cerebral hemorrhage and cardiac failure with pulmonary edema and consequently increased maternal and neonatal mortality [5].

In order to reduce the occurrence and severity of these pressor response complications, many drugs such as Hydralazine [6] magnesium sulphate [7] labetalol [8] fentanyl [5] trimethaphan [2] sodium nitroprusside [9] lidocaine [10] nitroglycerine [11,12] and nifedipine[13] has been used with varying degree of success.

Nitroglycerine has been used for rapid peri-operative management of maternal hypertension [11]. Longmire et al[11] have shown that intravenous nitroglycerine infusion is effective in lowering maternal blood pressure(BP) and in blunting hemodynamic pressor response to laryngoscopy and endotracheal intubation in severely pre-eclamptic patients. Hydralazine has been the antihypertensive of choice for severe pre-eclampsia patients for many years [14,15]. Short acting sublingual nifedipine, a calcium channel blocker, is another effective antihypertensive agent that is sometimes used to control acute, severe hypertension in pre-eclamptic patients [16,17]. Kumar et al [13] have shown that nifedipine is effective attenuating the hypertensive to laryngoscopy and intubation in pregnancy-induced hypertension.

II. Patients And Methods

After obtaining approval from institutional ethical committee and informed consent for incorporating into the study from each patient, 120 patients with severe pre-eclampsia and gestational age of more than 20 weeks, who were either scheduled for elective or emergency cesarean section under general anesthesia were enrolled in this randomized, double-blind, observational study. All patients had BP ≥ 160/110mmHg and met the defining criteria of severe pre-eclampsia according to the American College of Obstetrics and Gynaecology[18]. Criteria for exclusion from the study were, a history of heart failure diagnosed by a cardiologist, difficult airway, or any contra-indication for treatment with the study drugs.

Specific indications for cesarean delivery in our study patients were fetal distress, abruption placentae, decline in renal functions, HELLP Syndrome, persistent severe headache or visual changes, or epigastric pain.

Laboratory assessment included serial measurements of complete blood count, coagulation profile liver function tests, and renal function tests.

Management of severe pre-eclampsia included bed rest; to prevent seizures, all patients initially received Magnesium Sulphate as a 4 gms Intravenous loading dose, followed by 1 gm/hr intravenously before delivery, intra-partum and for 24hrs post-partum.

As candidate patients were enrolled in the study, they were randomly allocated to one of the three groups according to the agent to be used for attenuation of the pressor response to laryngoscopy and intubation; Group-1 [Nitroglycerine infusion (n=40)]; Group-2[sublingual Nifedipine (n=40)]; Group-3 [Hydralazine IV (n=40)].

GROUP-1 patients received 5µgs/min of Nitroglycerine administered by continuous IV infusion. The same dose was administered every 5 minutes until the therapeutic goal was achieved, which was a decrease in systolic arterial pressure(SAP) to <140 mmHg but not <120 mmHg and a decrease in diastolic arterial pressure(DAP) to <100 mmHg but not <80 mmHg.

GROUP-2 patients received, 10 mg sublingual Nifedipine before induction of anesthesia until the therapeutic goal had been achieved.

GROUP-3 patients received 5-10 mg of Hydralazine intravenously, according to the protocol recommended by the American College of Obstetrics and Gynecology [18].

All stages of patient management before induction of anesthesia were performed under the supervision of an obstetrician.

The study solutions were prepared by an anesthesiologist, who took no further part in the study. The anesthesiologist, who evaluated the patients were unaware of their group allocation. All other individuals involved in the study, were also blinded to the group allocation.

Patients were pre-medicated with 10 mgs of Metoclopramide and 40 mgs of Pantoprazole intravenously,30-45 minutes before induction of anesthesia.

Monitoring of patients included pulse oximetry for oxygen saturation (spo_2), electrocardiogram (ECG) , end-tidal carbon dioxide ($EtCO_2$), non-invasive automatic blood pressure monitoring for systolic arterial pressure (SAP), diastolic arterial pressure (DAP), mean arterial pressure (MAP) and heart rate (HR).

After pre-oxygenation for 5 minutes, patients were given the study drugs. One hundred seconds after receiving the study drug, anesthesia was induced 2.5 mgs./kg of Propofol given over 15-20 seconds and an assistant applied cricoids pressure from the time of induction of anesthesia until airway was secured and sealed by cuffed endotracheal tube. Laryngoscopy was performed one minute after giving 1.5 mgs./kg of succinylcholine, thus ensuring that the time from administration of the study drug to tracheal intubation was almost three minutes. Anesthesia was maintained with 50% nitrous oxide in oxygen with 1% of Isoflurane. Atracurium besylate was used for muscle relaxation. $EtCO_2$ was maintained between 35-40 mmHg throughout the surgery.

After delivery of the baby, analgesia was provided by 0.1mg/kg morphine and 10 iu syntocinon IV in 500ml of Ringer′s Lactate was infused to initiate uterine contractions.

Hemodynamic parameters like HR, SpO_2, ECG and NIBP (SAP, DAP, MAP) were simultaneously recorded in the mother at pre-induction, pre-intubation (2 minutes after giving test drug) and at 1, 3, 5 and 10 minutes after intubation. During pre-intubation period, occurrence of any arrhythmia, or cardiovascular complications, such as hyper- or hypotension, was noted. After completion of surgery, the residual effects of neuromuscular blockade was reversed by neostigmine (0.05mg/kg) and glycopyrrolate (0.01mg/kg) combination.

Apgor Scores of the babies were evaluated at 1, 5 and 10 minutes of delivery.

Statistical analysis of data was performed using SPSS version 16.0. Statistical comparisons within and among groups were performed using both 1- way and 2- way analysis of variance (ANOVA) followed by unpaired t-test with Bonferroni′s correction. Continuous variables were presented as mean ±SD and ordinal variables were presented as numbers (%). The chi-squared test was applied to categorical data with Yate′s

corrections. Alternatively, when any expected number was less than 5, Fisher′s exact method was used. P<0.05 was considered the minimal level of statistical significance.

III. Results

Demographic Parameters: The demographic profile, parity, baseline hemodynamic parameters, duration of laryngoscopy and Cormack- Lehane grading of direct laryngoscopy were more or less similar among three groups and were statistically insignificant.

TABLE-1 Maternal Demographic Data, Parity, Baseline Hemodynamic and Cormack-Lehane Grading of Direct Laryngoscopy for patients of 3 groups. (Statistically insignificant parameters)

Demographic/Baseline Hemodynamic Parameters	Group-1(n=40)	Group-2(n=40)	Group-3(n=40)
Age (yrs.) Mean ±SD	28.3±4.8	28.8±4.9	28.1±4.1
Weight (kgs) Mean ±SD	73.6±5.2	72.8±4.7	75.1±5.1
Primipara/ Multipara	26/14	28/12	29/11
Emergency/ Elective	35/5	34/6	35/5
HR(Beats/min.)Mean ±SD	95.3±12.6	90.1±15.3	98.1±8.9
SAP(mmHg)Mean ±SD	165.2±10.3	168.3±7.8	164.2±11.1
DAP(mmHg)Mean ±SD	104.4±8.3	106.1±8.6	105.2±11.4
MAP(mmHg)Mean ±SD	128.3±9.4	129.3±8.5	126.7±7.8
C-L Grade of Direct Laryngoscopy(1/2/3)	14/26/0	17/23/0	20/20/0

Abbreviations: HR-Heart Rate, SAP-Systolic Arterial Pressure, DAP-Diastolic Arterial Pressure, MAP-Mean Arterial Pressure, CL-Cormack-Lehane.

Hemodynamic Parameters:

Heart Rate (HR): Heart rate was significantly lower just before laryngoscopy and intubation and at 1 , 3, 5 and10 minutes after endotracheal intubation in group-1 compared with group-2 (p<0.05). Heart rate (HR) was also significantly lower at 1, 3 and 5 minutes after endotracheal intubation in group-1 when compared with group-3 (p<0.05).The patients in group-1 did not show any significant rise in heart (HR) after laryngoscopy and intubation, while as patients in group-2 and group-3 showed a significant increase in heart rate (HR) just before laryngoscopy and at 1, 3, 5 and10 minutes after intubation, compared to baseline (p<0.05).

TABLE-2 Heart Rate (HR) changes at Different Intervals following Induction.

TIME	Group-1	Group-2	Group-3
Baseline Heart Rate (Beats/min) Mean ±SD (TO)	92.5±7.3	90.4±13.6	93.5±10.2
Heart Rate (Beats/min.) Mean ±SD Just Before Laryngoscopy(TBI)	94.2±6.5	97.4±19.5	108.1±20.1
Heart Rate (Beats/min.) Mean ±SD 1 Minute After Laryngoscopy(ET1)	96.4±6.2	117.2±20.1	119.3±13.3
Heart Rate (Beats/min.) Mean ±SD 3 Minute After Laryngoscopy(ET3)	95.1±4.6	112.6±9.3	116.4±10.1
Heart Rate (Beats/min.) Mean ±SD 5 Minute After Laryngoscopy(ET5)	94.8±8.6	99.1±10.3	111.4±13.1
Heart Rate (Beats/min.) Mean ±SD 10 Minute After Laryngoscopy(ET10)	95.1±7.2	92.3±12.1	107.2±15.3

Systolic Arterial Pressure(SAP): Systolic arterial pressure(SAP) was significantly lower at 1, 3 5 and 10 minutes after intubation in group-1 as compared to group-2 and group-3 (p<0.05). Patients in group-2 showed a significant rise in SAP just before laryngoscopy and at 1,3 and 5 minutes after intubation compared to baseline (p<0.05), while as patients in group-3 showed significant rise in SAP just before laryngoscopy and at 1,3 and5 minutes post intubation compared to baseline (p<0.05). Patients in group-1 showed no significant increase in SAP after laryngoscopy and intubation.

Diastolic Arterial Pressure(DAP): Diastolic arterial pressure(DAP) was significantly lower at 1,3,5 and 10 minutes post endotracheal intubation in group-1 compared to group-3 (p<0.05). While the patients in group-1 showed no significant rise in DAP post laryngoscopy and intubation, there was significant increase in DAP in group-2 and group-3, one minute post laryngoscopy and intubation compared to baseline (p<0.05).

Mean Arterial Pressure(MAP): It was significantly lower in group-1 at pre-intubation, at 1,3, 5 and10 minutes post-intubation as compared to group-2 and group-3 (p<0.05). Patients in group-1 showed no significant increase in MAP as compared to baseline, while as patients in group-2 and group-3 showed significant rise in MAP, just before laryngoscopy and intubation compared to baseline(p<0.05).

The incidence of hypotension was significantly higher in group-2 as compared to group -1 and group-3 [group-1; 12.5%, group-2; 42.5%, group-3;25%]($p<0.05$).There were 3 patients in group-3 and 2 patients in group-2, while no patient in group-1, who developed some kind of arrhythmia at induction ($p<0.05$).

TABLE-3 Systolic Arterial Pressure (SAP) Changes At Different Intervals Following Induction.

Time	Group-1	Group-2	Group-3
Baseline SAP(mmHg) Mean ±SD (TO)	162.3±7.8	164.2±6.3	163.7±8.1
SAP(mmHg)Mean ±SD Just Before Laryngoscopy(TBI)	159.8±4.5	158.2±4.9	157.3±6.1
SAP(mmHg)Mean ±SD 1 Minute Post-intubation(ET1)	161.4±8.6	172.7±12.3	169.9±9.4
SAP(mmHg)Mean ±SD 3 Minute Post-intubation(ET3)	160.1±9.5	174.2±15.5	168.2±10.3
SAP(mmHg)Mean ±SD 5 Minute Post-intubation(ET5)	160.8±3.5	173.4±11.2	165.9±12.0
SAP(mmHg)Mean ±SD 10 Minute Post-intubation(ET10)	162.5±8.9	169.3±12.3	166.2±13.5
SAP(mmHg)Mean ±SD 10 Minute Post-intubation(ET10)	162.5±8.9	169.3±12.3	166.2±13.5

IV. Discussion

Direct laryngoscopy and endotracheal intubation cause an increase in hemodynamic parameters viz; BP and HR[20]. This cardiovascular pressor response is presumed to be a sympathetic reflex response to mechanical stimulation of the larynx and trachea. There is significant rise in serum levels of epinephrine and nor-epinephrine subsequent to the laryngoscopy, with or without tracheal intubation [21, 22,23]. Pressor response involves an average rise in BP of 40-50% and a 20-30% increase in HR[24]. The pressor response to laryngoscopy and intubation increases myocardial oxygen demand and risk of cerebrovascular accidents, decrease uterine blood flow and may induce cardiac arrhythmias and pulmonary edema [1,2,5,25]. Many drugs have been used to attenuate these pressor responses including lidocaine, short-acting narcotics like fentanyl [26,27,28].

TABLE-4 Diastolic Arterial Pressure (DAP) Changes At Different Intervals Following Induction.

Time	Group-1	Group-2	Group-3
Baseline DAP(mmHg) Mean±SD (TO)	106.4±6.3	107.1±9.3	107.4±10.2
DAP(mmHg)Mean±SD Just Before Laryngoscopy(TBI)	104.3±5.8	105.2±12.3	106.3±11.8
DAP(mmHg)Mean±SD 1 Minute Post-intubation(ET1)	106.2±5.1	109.3±17.3	114.9±15.1
DAP(mmHg)Mean±SD 3 Minute Post-intubation(ET3)	105.5±2.2	106.8±16.5	112.7±14.3
DAP(mmHg)Mean±SD 5 Minute Post-intubation(ET5)	103.1±6.9	108.4±12.5	107.9±11.8
DAP(mmHg)Mean±SD 10 Minute Post-intubation(ET10)	104.2±6.9	111.3±14.6	107.4±10.5
SAP(mmHg)Mean±SD 10 Minute Post-intubation(ET10)	162.5±8.9	169.3±12.3	166.2±13.5

TABLE-5 Mean Arterial Pressure(MAP) Changes At Different Intervals Following Induction.

Time	Group-1	Group-2	Group-3
Baseline MAP(mmHg) Mean±SD (TO)	104.1±6.3	105.8±7.5	104.3±7.1
MAP(mmHg)Mean±SD Just Before Laryngoscopy(TBI)	102.8±4.2	101.9±6.9	103.3±8.2
MAP(mmHg)Mean±SD 1 Minute Post-intubation(ET1)	103.2.±4.4	105.2±8.1	109.1±7.9
MAP(mmHg)Mean±SD 3 Minute Post-intubation(ET3)	102.7±4.5	104.2±11.2	103.2±12.3
MAP(mmHg)Mean±SD 5 Minute Post-intubation(ET5)	100.2±7.1	104.1±12.2	102.9±11.8
MAP(mmHg)Mean±SD 10 Minute Post-intubation(ET10)	103.1±5.2	106.2±9.5	104.2±7.6

Nifedipine is a calcium channel blocker and is one of the several agents that has been used for the reduction of BP in severe pre-eclampsia[29]. Nifedipine was selected for comparison in our present study as it is one of the commonest antihypertensive agents used in pregnancy-induced hypertension[30]. It has been seen that

mode of administration of nifedipine that avoids first-pass metabolism in the gastro intestinal tract, such as use of sublingual perforated capsule, results in greater bioavailability and a faster accumulation and more stable concentration of nifedipine in serum than methods that do not avoid first-pass metabolism (oral administration of an aspirated or chewable capsule)[31].Since sublingual nifedipine causes a gradual drop in BP as compared to oral administration, so clinically sublingual administration is regarded as the most appropriate to achieve rapid onset and better control of the effects of nifedipine[31].

In our study, the group-2(nifedipine group) showed both greater maternal HR response and MAP variability than the group-1(nitroglycerine group) after laryngoscopy and intubation, which implies that under the present study conditions, sublingual nifedipine induces a greater variability in the arterial baroreflex system, through the mechanism that cannot be ascertained by the present study. However, in a study by Fenakel et al [32]showed that nifedipine has greater efficacy than hydralazine in achieving BP control in severe pre-eclampsia and same results were also shown by kwawukume and Ghosh[33].

In our study, the results showed that hydralazine was ineffective for attenuation of the pressor response caused by laryngoscopy and intubation during induction of general anesthesia for patients with severe pre-eclampsia. This may be due to slow onset and variable duration of action of hydralazine as well as compensatory tachycardia, which makes it difficult to titrate its action against the hypertensive response[33,34]. Van den Berg et al[35] in their study compared the effects of magnesium sulphate, esmolol, lidocaine and nitroglycerine on the attenuation of the pressor response to laryngoscopy and showed that nitroglycerine successfully blunted the pressor response, while as magnesium sulphate and lidocaine has the least effect on pressor response. Mikawek et al[36] also showed that nitroglycerine IV is a safe and effective method of attenuation of hypertensive pressor response following laryngoscopy and intubation. Similar results were also shown regarding blunting of pressor response following laryngoscopy and intubation in patients who received nitroglycerine IV before induction of anesthesia by various authors like Fassoulaki A. et al[37] and Mahajan RP et al[38].

Generally physicians aim at keeping SAP between 140 to 160 mmHg and DAP between 90 to 110 mmHg, because at these blood pressure levels, a reduction in either uteroplacental blood flow or cerebral perfusion pressure is unlikely. So, nitroglycerine appears to maintain blood pressure levels within desired range more successfully as compared to nifedipine or hydralazine.

Our results in present study are all in agreement with the results obtained by various workers mentioned above. In our present study, nifedipine and hydralazine were less effective in attenuating hypertensive pressor response as compared to nitroglycerine. It was seen in patients of group-1 (nitroglycerine group), not only the desired hypotensive effect was achieved earlier but the therapeutic goal was attained faster and with greater precision in this group(nitroglycerine group) as compared to group-2(nifedipine S/L) or group-3(hydralazine IV). In addition, the maternal tolerance to IV infusion of nitroglycerine was excellent as there were only five(5) episodes of hypotension in group-1 (nitroglycerine group) as compared to seventeen(17) episodes of hypotension in group-2(nifedipine group) and ten(10) episodes of hypotension in group-3(hydralazine group) in our present study

V. Conclusion

The results of the present study showed that in patients with severe pre-eclampsia who are managed with controlled extracellular volume expansion and magnesium sulphate loading and maintenance doses, continuous IV infusion of nitroglycerine was able to attenuate the pressor response to laryngoscopy and endotracheal intubation to a greater extent, faster and more precisely as compared to sublingual nifedipine or IV hydralazine without significant adverse effects on the fetus. Although more research must be done, we conclude that nitroglycerine continuous IV infusion may provide safe and effective prophylaxis for patients with severe pre-eclampsia undergoing cesarean delivery under general anesthesia, in attenuating hemodynamic pressor response to laryngoscopy and endotracheal intubation.

Reference

[1]. Hodgkinson R., Husain FJ.&Hayashi RH. Systemic and pulmonary blood pressure during cesarean section in parturients with gestational hypertension. Can. Anaesth Soc. J.1980;27(4)389-94
[2]. Connell H., Dalgleish JG., Downing JW. General anesthesia in mothers with severe preeclampsia. British J. Anesthesia.1987; 59 (11):1375-80.
[3]. Evans CS., Gooch I., Flotta D., et al. Cardiovascular system during the post-partum state in women with a history of preeclampsia. Hypertension.2011;58(1):57-62.
[4]. Kronborg CS., Gjedsted j.,Vittinghus E., et al. Longitudinal measurement of cytokines in preeclamptic and normotensive pregnancies. Acta Obstet Gynecol Scand. 2011;90(7): 791-796.
[5]. Lawes EG., Downing TW., Duncan PW. et al. Fentanyl-droperidol supplementation of rapid sequence induction in the presence of severe pregnancy-induced and pregnancy-aggravated hypertension. British J. Anesthesia.1987;59(11):1381-1391.
[6]. Magee LA., Abalos E., Dadelszen P et al. How to manage hypertension in pregnancy effectively. British J. Clin. Pharmacol. 2011;72(3):394-401.

[7]. Ashton WB., James MF., Janicki P. et al. Attenuation of pressor response to tracheal intubation by magnesium sulfate with or without alfentanil in hypertensive proteinuric patients undergoing cesarean section. British J. Anesthesia.1991;67(6):741-747.
[8]. Ramanathan J.,Sibai BM., Mabie WC.et al. The use of labetalol for attenuation of the hypertensive response to endotracheal intubation in preeclampsia. Am. J. Obstet Gynecol. 1988;159(3):650-654.
[9]. Strauss RG., Keeper JR., Burke T.et al. Haemodynamic monitoring of cardiogenic pulmonary edema complicating toxemia of pregnancy. Obstet Gynecol.1980;55(2):170-174.
[10]. Shinider SM., Levinson G. Anesthesia for obstetrics. Anaesthesiology.1987;67(4):615.
[11]. Longmire S., Leduc I., Jones MM. et al. The hemodynamic effects of intubation during nitroglycerine infusion in severe preeclampsia. Am. J. Obstet Gynaecol.1991;164(2): 551-556.
[12]. Trapani A., Jr,Gonclaves LF., Pires MM. Transdermal nitroglycerine in patients with severe per-eclampsia with placental insufficiency: Effects on Doppler velocimetry of the uterine,umlilical and middle cerebral arteries. Ultrasound Obstet Gynaecol. 2011:38(4):389-394.
[13]. Kumar N., Batra YK., Bala J.et al. Nifedipine attenuates the hypertensive response to tracheal intubation in pregnancy-induced hypertension. Can.J. Anesth.1993;40(4):329-333.
[14]. Lenfant C. Working group report on high blood pressure in pregnancy. J. Clin.Hypertens. (Greenwich Com.) 2003;3(2):75.
[15]. Rey E., Lelorier J.,Burgess E.et al. Report of the Canadian Hypertension Society Consensus Conference:3. Pharmacological treatment of hypertensive disorders in pregnancy. CMAJ. 1997;157(9):1245-1254.
[16]. Cutler JA. Calcium channel blockers for hypertension-uncertainty continues. N. Engl. J. Med. 1998;338(10):679-81.
[17]. Grossman E., Messerli FH., Grodzichi T. et al. Should a moratorium be placed on sublingual nifedipine capsules given for hypertensive emergencies and pseudoemergencies? JAMA. 1996;276(6):1328-1331.
[18]. Cunningham FC.,Williams JW., Leveno KJ. Et al. Williams Obstetrics. London: Prentice Hall International;1997.
[19]. Mabie WC., Gonzalez AR., Sibai BM. Et al. A comparative trail of labetalol and hydralazine in the acute management of severe hypertension complicating pregnancy. Obstet Gynaecol. 1987;70(3):328-33.
[20]. Stoelting RK. Circulatory changes during direct laryngoscopy and tracheal intubation: Influence of duration of laryngoscopy with or without prior lidocaine. Anaethesiology. 1997; 47(4):381-384.
[21]. Russel WJ., Morris RG.,Frewin DB.et al. Changes in plasma catecholamine concentrations during endotracheal intubation .British Journal of Anesthesia.1981;53(8):837-839.
[22]. Shribman AJ., Smith G., Achola KJ. Cardiovascular and catecholamine responses to laryngoscopy with and without tracheal intubation. British Journal of Anesthesia.1987;59(3)295-99.
[23]. Park BY., Jeong CW., Jang EA et al. Dose-related attenuation of cardiovascular responses to tracheal intubation by intravenous remifentanil bolus in severe pre- eclamptic patients undergoing cesarean delivery. British Journal of Anesthesia.2011;106(1):82-87.
[24]. Bruder N., Ortega D., Granthil C. [Consequences and prevention methods of hemodynamic changes during laryngoscopy and intratracheal intubation.] Ann. Fr. Anesth. Reanim. 1992;11(1):5771.
[25]. Miyake W., Oda Y., Ikeda Y. et al. Effects of remifentanil on cardiovascular and bispectral index responses following the induction of anesthesia with midazolam and subsequent tracheal intubation. J. Anesth. 2010;24(2):161-167.
[26]. Morison DH. Anesthesia and pre-eclampsia. Can. J. Anesth. 1987;34(4):514-422.
[27]. Talebi H., Nourozi A., Fateh S. et al. Effects of oral clonidine premedication on hemodynamic responses to laryngoscopy and tracheal intubation: A clinical trial. Pak.J. Biological Sciences 2010;13(23):1146-1150.
[28]. Rout CC., Rocke DA. Effects of alfentanil and fentanyl on induction of anesthesia in patients with severe pregnancy-induced hypertension. British J. Anesth. 1990;65(4):468-474.
[29]. Ferlinz J. Drugs five years later, nifedipine in myocardial ischaemia, systemic hypertension and other cardiovascular disorders. Ann. Intern. Med. 1986;105(5):714-29.
[30]. Jegasothy R., Paranthaman S. Sublingual nifedipine compared with intravenous hydralazine in the acute treatment of severe hypertension I pregnancy: Potential for use in rural practice. Journal. Obstet Gynaecol. Res. 1996;22(1):21-24.
[31]. Kubota R., KomiyamaT., Shimada H. Evaluation of the methods for nifedipine administration for a rapid onset of clinical effects: A clinical study in normal volunteers. Yakugaku Zasshi. 2001;121(5):355-364.
[32]. Fenakel K., Fenakel G., Appelmanz et al. Nifedipine in the treatment ofsevere pre-eclampsia. Obstet. Gynaecol. 1991;77(3):331-337.
[33]. Kwawukume EY., Ghosh TS. Oral nifedipine therapy in the management of severe pre-eclampsia. Internet J. Gynaecol. Obstet. 1995;49(3):265-269.
[34]. Ramanathan J. Pathophysiology and anesthetic implications in pre-eclampsia. Clin. Obstet. Gynaecol. 1992;35(2):414-425.
[35]. Ring G., Krames E., Shnider SM. et al. Comparison of nitroglycerine and hydralazine in hypertensive pregnant ewes. Obstet. Gynaecol. 1997;50(5):598-602.
[36]. Van den Berg AA., Savva D., Honjol NM. Attenuation of the haemodynamic responses to noxious stimuli in patients undergoing cataract surgery: A comparison of magnesium sulfate, esmolol, lignocaine, nitroglycerine, and placebo given IV with induction of anesthesia. Eur. Journal Anesthesiol. 1997;14(2):134-147.
[37]. Mikawa K., Hasgawa M., Suzuki T. et al. Attenuation of hypertensive response in tracheal intubation with nitroglycerine. J. Clin. Anesth. 1992;4(5):367-71.
[38]. Fassoulaki A., Kaniaris P. Intranasal administration of nitroglycerine attenuates the pressor response to laryngoscopy and intubation of the trachea. British Journal of Anesthesia. 1983;55(1):49-52.
[39]. Mahajan RP., Ramachandran R., Sexana N. Topical nitroglycerine prevents the pressor response to tracheal intubation and sternotomy in patients undergoing coronary artery bypass graft surgery. Anaesthesia.1993;48(4):297-300.