

COMPARISON OF SALINE FLUSH TEST (SFT) AND INTERNAL JUGULAR VEIN OCCLUSION TEST (IJV OT) FOR DETECTION OF MISPLACED SUBCLAVIAN VEIN (SV) CATHETER INTO IPSILATERAL INTERNAL JUGULAR VEIN (IJV)

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ABSTRACT

Central Venous Cannulation is a useful tool for long term vascular access in a variety of clinical situation.¹ Subclavian Vein approach is frequently used in the placement of catheters into the great intra-thoracic veins and it is the principal method of inserting CVCs in our institute. During procedure misplacement of SV catheter into the ipsilateral IJV is very common^{2,3}. A malpositioned catheter can result in faulty CVP reading or lead to thrombosis of the vein^{1,2,4,5}. Chest radiograph is confirmatory but it requires time, man power, special equipment and has a disadvantage of radiation exposure which has to be repeated after any repositioning². Therefore we compared the efficacy of saline flush test (SFT) and internal jugular vein occlusion test (IJV OT) for the misplaced SV catheter into ipsilateral internal jugular vein.

AIM

Aim of the study was to note the incidence of misplacement of SV catheter into ipsilateral IJV, to compare efficacy of SFT and IJV OT and to compare their sensitivity and specificity.

METHOD

After approval by the scientific committee of the institute 200 cancer patients of either sex scheduled to undergo CVC were planned for SV

catheterization via infraclavicular approach with 100 patients in each group. Informed written and verbal consent was obtained from all the patients before procedure. Procedure was done using seldinger's technique in day stay unit under strict aseptic and antiseptic precautions. At the end of procedure length of the catheter to be inserted was calculated from distance between the puncture point and sternal angle and sternal angle to second rib. Free flow of saline and free blood aspiration was confirmed and the catheter was fixed to anterior chest wall.

Now in **SFT group** of patients the flush test was performed by pushing ten ml of normal saline in the distal port of the catheter while anterior angle of ipsilateral neck was palpated by an independent observer. A thrill of fluid elicited on the palm of hand (Positive test) was suggestive of catheter misplacement into ipsilateral IJV.

In **IJV OT group** catheter was connected to the pressure transducer. A firm external pressure was applied over ipsilateral neck in the supraclavicular area for five to ten seconds and increase in CVP value of three mm of Hg or more above the baseline value and flattened tracing suggest misplacement into the ipsilateral IJV. The test was repeated if the rise in CVP was two mm of Hg. The test was considered negative if there was no change in CVP value or if the change was less than two mm of Hg.

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At the end of procedure chest radiograph was performed in all the patients to detect any complications and to confirm the catheter tip position. Sensitivity and Specificity of both the tests were calculated taking chest x-ray as the gold standard. All the misplaced catheters were withdrawn and redirected by occluding IJV during guide wire insertion

Successful cannulation was achieved in 96 patients in SFT group and in 98 patients in IJV OT group. Median age and weight of the patients were 38 (05-70) years and 46 (15-96) kg in SFT group and 42 (07-74) years and 50 (18-102) kg in IJV OT group respectively. M:F ratio in SFT group was 58:42 and in IJV OT it was 64:36. There were total 19 (9.8%) misplacements as detected by post procedural chest radiograph. (Table 1 – Patient profile)

RESULTS

Table - 1: Patient Profile

	Group SFT (N = 100)	Group IJV OT (N = 100)
Age (Yrs) (Median/Range)	38 (05-70)	42 (07-74)
Weight (Kg)	46 (15-96)	50 (18-102)
Gender(M:F)	58:42	64:36.
Successful cannulation of SV	96	98

In **Group SFT** there were nine misplacements as detected by chest radiograph. In six patients catheter tip was located in the ipsilateral IJV and in two patients catheter tip was in contralateral SV and in one patient it was in right brachiocephalic vein. SFT was positive in all the six patients who had misplaced SV catheter into the ipsilateral IJV and the test was negative in rest of the patients (in patients who had normally placed catheters or misplaced catheter other than into ipsilateral IJV.) In **Group IJV OT** there were ten misplacements as detected by chest radiograph. In seven patients catheter tip was located in the ipsilateral

IJV and in three patients it was in contralateral SV. IJV OT was positive in six patients with misplaced catheter into ipsilateral IJV with increase in CVP of around 3-6 mm of Hg associated with flattening of its waveform. In one patient with misplaced catheter in ipsilateral IJV, CVP reading was two mm of Hg. So the test was repeated which again showed rise in CVP of two mm of Hg. Therefore we excluded this patient. IJV OT was negative in rest of the patients (in patients who had normally placed catheters or misplaced catheter other than into ipsilateral IJV.) (Table 2 – Incidence of misplaced catheter)

TABLE - 2: Incidence of misplaced catheter

	Group SFT (N = 100)	Group IJV OT (N = 100)
Total no. of catheter misplacement (X-ray)	9 (9.4%)	10 (10.2%)
Misplacement in ipsilateral IJV (X-ray)	6 (6.2%)	7 (7.1%)
Misplacement in ipsilateral IJV (SFT and IJV OT)	6 (6.2%)	6 (6.1%)

TABLE – 3: Sensitivity and Specificity

	Group SFT	Group IJV OT
Sensitivity	66.6%	100%
Specificity	60%	85.7%

DISCUSSION

Vascular access is a major problem in cancer patients requiring diagnostic and therapeutic regimens for a longer period of time. Introduction of CVC has greatly simplified these problems and facilitated the management of such patients with poor peripheral venous access. Of the many approaches for CVC, subclavian route is considered a classic procedure^{1,6} and it is the principal method of inserting CVCs in our institute.

One of the most common misplacements of the SV catheter is into the ipsilateral IJV. There are several techniques to assess position of the catheter tip placed through SV. But most of them require additional monitoring instruments which are not available all the times.^{1,2} Also there are bedside clinical maneuvers to prevent the placement of SV catheter into IJV like turning the head of the patient towards same side,³ occlusion of ipsilateral IJV at supraclavicular fossa⁴, turning the bevel of the needle and 'J' tip of the guide wire inferiorly⁵. These methods may not be always successful. In one randomized, double-blind clinical study the incidence of catheter misplacement into the ipsilateral internal jugular vein was 2% in a group in which 'J' tip of the guidewire directed caudad and 40% in group in which 'J' tip was directed cephalad ($P < 0.01$).⁵ It is near impossible to maintain the catheter tip in any particular orientation when doing this procedure without any monitoring gadgets. Influence of shoulder position on CVC tip position is also important. Lowered shoulder position to reduce the angle between the SCV and brachiocephalic

vein may increase the risk of directing SCV catheter into the ipsilateral IJV.⁶ Clinical tests like SFT and IJV OT detect malpositioned catheter into ipsilateral IJV without any expensive instruments.

Misplacement of the catheter tip into IJV increases the risk of chemical and bacterial thrombophlebitis, clot formation, impaired CVP measurement, arrhythmias, vascular erosion with local infection.^{1,2,13} In a study of 57 oncology patients with short and long term CVC, different degree of incomplete thrombosis was found in 25 patients (45.5%) and complete thrombosis, clinically silent in six patients (10.5%).⁷ Administration of chemotherapeutic agents in high risk cancer patients increases the risk of thrombosis when the catheter tip is in IJV and application of clinical methods to prevent such malpositioning is useful. Risk factors for SVC related venous thrombosis were intracranial hypertension, massive transfusion and tip position in the internal jugular or innominate vein. Early diagnosis is important and chest x-ray is confirmatory but because of the risk of repeated exposure after catheter insertion and repositioning we compared SFT and IJV OT to detect such misplacements. The incidence of misplacement of CVP catheters through infraclavicular approach was found to be 5.5%⁸ in one study and 6.2%¹ in another study and 5.2% of these malpositioned catheters were located in ipsilateral IJV.¹ Dietel and McIntyre found higher (24%) incidence of misplaced CVC through the same route.⁹ In our study incidence of misplaced

SV catheter inserted through right infraclavicular approach was 9.5% which was higher compared to previous studies because of insertion by learners, greater length of guide wire and catheter insertion, steep head low position and lowered shoulder position.

There is relationship between the length of the guide wire and catheter malposition and implicate excessive guide wire length as a cause for this complication and recommended a guide wire length of 18 cm which should be considered as the upper limit. A steep head down position to fill the central veins is associated with increased venous pressure which preferentially directs the floating guide wire to the neck veins.¹⁰ The insertion of a greater length may place the malpositioned catheter tip in the jugular bulbs so the length of the catheter inserted should be adequate but relatively shorter.¹¹ A study of 202 SV catheterization reported that persons who had performed 50 or more catheterizations had 0% complication rate and those who had performed less than 50 catheterizations had 8.1% complication rate.¹²

G P Rath and P K Bithal (2009)² performed SV catheterization in 570 patients. SFT was positive in 19 patients (3.3%) who has misplaced catheter into the ipsilateral IJV demonstrating 100% sensitivity and specificity of SFT. In our study in group SFT there were nine (9.4%) misplacements as detected by chest x-ray and the test was positive in all the six (6.2%) patients with misplacements of SV catheter into ipsilateral IJV demonstrating 66.6% sensitivity and 100% specificity of SFT to detect misplacement of SV catheter into ipsilateral IJV

Ambesh SP and Dubey PK (2001)¹ performed SV cannulation in 96 patients. IJV OT was positive in five (5.2%) patients and negative in rest of the patients showing 83.3% sensitivity and 100% specificity of this test to detect the misplacement

of SV catheter into ipsilateral IJV. In our study in group IJV OT there were ten (10.2%) misplacements as detected by chest x-ray. The incidence of misplaced SV catheter into ipsilateral IJV was seven (7.1%). IJV OT was positive in six (6.1%) patients showing 60% sensitivity and 85.7% specificity of this test in detecting this malposition.

Following this we made a protocol to be followed before inserting SV Catheter:

- Each student will assist minimum 25 SCV catheterization before independent insertion
- Guide wire length → 16 to 18 cm
- Head low position → 15° to 20°
- Tip of the bevel and 'j' tip of guide wire directed downwards
- Position of the shoulder should be neutral
- Length of catheter insertion:
 - Female : 10 to 12 cm
 - Male : 11 to 14 cm
 - Paediatric : length calculated from distance between the puncture point and sternal angle and sternal angle to second rib
- SFT to be done in all patients before fixation of the catheter and submission to x-ray.

CONCLUSION

Both the tests successfully detect the misplacement of SV catheter into ipsilateral IJV and allow avoidance of repeated exposure of x-ray after catheter insertion and repositioning. However they do not detect any other misplacement. SFT is simpler, quicker and inexpensive bedside technique as compared to IJV OT. Any person performing this procedure should have undergone training and experience.

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COMPARISON OF ORAL GABAPENTIN AND CLONIDINE FOR ATTENUATION OF PRESSURE RESPONSE TO LARYNGOSCOPY AND INTUBATION

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SUMMARY

Aim and Objectives : Laryngoscopy and tracheal intubation increases blood pressure and heart rate. We studied the effect of 900 mg gabapentin and 200mcg clonidine given orally 90 min before induction of anaesthesia on pressor response to laryngoscopy and tracheal intubation.

Methods: Seventy five patients of ASA grade I and II were selected and randomly divided into three groups. Group C received tab clonidine 200mcg, group G received tab gabapentin 900mg and group P received placebo tablet 90 min before induction of anaesthesia. Heart rate, Systolic, Diastolic and mean arterial blood pressure were recorded before and after induction and at 0,1,3,5,7 and 10 min after laryngoscopy and tracheal intubation.

Results: Mean systolic, diastolic and mean arterial blood pressure (SBP, DBP and MAP) raised in all groups after laryngoscopy and intubation which was insignificant in group G (p 0.0002, 0.0063, 0.0026) whereas in group C it was significant for SBP(p 0.0043) but not significant for DBP and MAP(p 0.1053, 0.0266) In intragroup comparison, mean SBP, DBP, and MAP were lower in group C as compared to placebo group at 0,1,3,5,7 and 10 min (p0.0001, 0.0001, 0.0001). While comparing group G and

group P, mean SBP and MAP were lower in group G at 0,1,3,5,7 min (p 0.0001, 0.0003) at 10 min (p 0.0076, 0.0536). While change in mean DBP was not statistically significant at 0,1 min (p 0.086, 0.159), but was significant at 3,7 min(p 0.04, 0.03).

While comparing group C and group G, the variables were lower in group C at 0 min(p 0.006,0.0001, 0.0001), 1 min(p 0.0236, 0.0001, 0.0005) and 5 min (p 0.013, 0.0001,0.0012). After five min it was not statistically significant for SBP (p0.05), but DBP and MAP were lower in group C (p 0.0034, 0.0093).

Heart rates were raised highest in placebo group. There was no statistical significance between group P and group G at all time intervals at 0,1,3,5,7 and 10 min(p 0.028, 0.090, 0.080, 0.088, 0.143). Comparing group G and group C, HR were significantly lower in group C at all time intervals(p 0.0001).

Conclusion: Clonidine premedication attenuates pressure response and rise in heart rate to laryngoscopy and intubation while gabapentin premedication attenuates only pressure response and not rise in heart rate.

Key words: Clonidine, gabapentin, laryngoscopy, pressure response, tracheal intubation, premedication

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INTRODUCTION

Endotracheal intubation is commonly performed as a part of general anaesthesia. Manipulation of the respiratory tract such as direct laryngoscopy and intubation can produce marked haemodynamic and circulatory effects which is characterized by sudden increase in heart rate and blood pressure which can lead to serious consequences like myocardial ischemia, dysarrhythmia, increase intracranial pressure, pulmonary oedema.¹²

To attenuate these harmful effect various methods are in practice including deeper plane of anaesthesia and topical anaesthesia of the upper respiratory tract before laryngoscopy with lignocaine.¹³ Vasodilators like nitroglycerine, beta blocker, calcium channel blocker, opioids such as fentanyl, alfentanyl are used to control haemodynamic response to laryngoscopy.⁴⁻⁷ No technique is free of side effects and thus no single method has achieved universal acceptance. Clonidine is a α 2 adrenoreceptor agonist mainly used as an antihypertensive agent having sedative, analgesic, anxiolytic, antisialogogue properties which make it an ideal premedication. It has beneficial effects on the hyperdynamic response to laryngoscopy and tracheal intubation.^{8,9}

Gabapentin is a structural analogue of γ – amino butyric acid mainly used as anticonvulsant drug and to control neuropathic pain.¹⁰ It is a multimodal perioperative drug with favourable side effect profile and has less interaction with other drug. It has shown to prevent postoperative nausea and vomiting, reduction of postoperative delirium, preoperative anxiety, and attenuation of haemodynamic responses to laryngoscopy and intubation.² It acts by decreasing synthesis of neurotransmitter glutamate and by binding to α 2 delta subunit of voltage unit calcium channel. This action is similar to calcium channel blocker

may be responsible for blunting haemodynamic responses to laryngoscopy and intubation.^{11,12}

Memis and colleagues¹¹ showed that oral gabapentin 800mg given one hour before induction to anaesthesia can attenuate pressure reponse to the laryngoscopy and tracheal intubation. Nishikawa et al¹³ showed that clonidine 4mcg/kg could attenuate pressure response following laryngoscopy and tracheal intubation. We planned to compare the effect of oral clonidine 4mcg/kg and oral gabapentin 900mg to attenuate haemodynamic responses following laryngoscopy and tracheal intubation.

METHODS

After obtaining approval from the hospital ethics committee and informed consent from the patient the randomized double blind study was conducted on 75 patients of either sex aged between 20-60 years of ASA grade I and II undergoing elective surgery under general anaesthesia. Patients with a history of hypertension, heart block, ischemic heart disease, on calcium channel blocker, beta blocker, antacid, sedative, hypnotic and antidepressant drug, having pregnancy, lactating mother, anticipating difficult intubation were excluded from the study.

All the patients were randomly allocated to three groups of 25 each according to the drug given as premedication 90 minutes before induction of anaesthesia. Patients of group C received oral clonidine 4mcg/kg(maximum 200mcg), group G received oral gabapentin 900mg and group P patient received placebo tablet same time. In the preanaesthetic room before and 90 minutes after administration of the oral drug heart rate, systolic, diastolic and mean arterial blood pressure(SBP,DBP,MAP), status of tongue, degree of sedation and anxiety, respiratory rate and any undesirable effect were noted. The degree of sedation and anxiolysis was graded as follows

Sedation score⁸

0 – Patient awake and talkative

1 – Patient awake but calm

2 – Patient drowsy, quiet and easily arousable

3 – Patient asleep

Anxiety score⁸

0 – Patient quite and comfortable

1 – Patient uneasy

2 – Patient worried or anxious

3 – Patient very worried or very upset

4 – Patient frightened or terrified

On arrival in the operation theatre an intravenous line was set up and 5ml/kg ringer's lactate infusion started. Monitoring for noninvasive blood pressure (NIBP), heart rate, electrocardiogram, peripheral arterial oxygen saturation was applied. A uniform technique of anaesthesia was used in all three groups. Following preoxygenation for 5 minutes all patients were given inj glycopyrrolate 0.2 mg, inj fentanyl citrate 1mcg/kg, inj thiopentone sodium 5mg/kg and inj vecuronium bromide 0.1 mg/kg. After ventilation with 100% oxygen for three minutes patients were intubated by single experienced anaesthesiologist using Macintosh blade size 3 with 7.5-8 size cuffed portex endotracheal tube. Patient requiring second attempt and laryngoscopy longer than 30 seconds were excluded from the analysis. Isoflurane in O₂ and N₂O (50%) was started after 10 minutes at 0.5-1 MAC. Bolus doses of vecuronium was given as required. All patients were reversed with inj glycopyrrolate (0.01mg/kg) and inj neostigmine (0.05mg/kg) at the end of surgery and were extubated.

Heart rate, SBP, DBP and MAP were recorded before and after induction, before intubation, immediately after intubation (0 min), and at 1,3,5,7,10 minutes after intubation using multipara monitor. Decrease in MAP greater than 30% below the baseline value was treated by intermittent doses of inj mephenteramine 6mg IV. Decrease in the heart rate below 45 beats/min

was treated with incremental doses of inj atropine 0.3mg IV.

Statistics

Data are expressed as mean \pm se or number of patient. Haemodynamic responses to laryngoscopy and intubation were analysed using repeated measurement ANOVA (Timex haemodynamic variables)two way followed by pair t test for comparison within the group and on independent sample t test was used to compare two groups at baseline and each time of the study. We used SPSS (SPSS Inc, Chicago, Illinois, USA) version 10 for statistical analysis. P value < 0.05 was considered significant.

RESULTS

No patients were excluded from study. The demographic data were comparable between the groups. There was no statistical difference regarding age, sex weight and vital data of the patients between the groups (table 1). After 90 minutes of premedication there was significant difference in sedation score between group C and group P (p 0.0001) and group G and group P (p 0.0001) where as no significant difference was there between group C and group G (table-2). There was significant difference in anxiety score as shown in(table-2) while comparing study group with control group but no significant difference was there while comparing study groups. Status of tongue was dry in 21 patients in group C (84%) and in only 4 patients in Group G (16%).

Ninety minutes after premedication heart rate decreased significantly in group C from premedication value (p 0.0001) as shown in table 3. Decrease in group G and group P was not significant (p 0.05, p 0.05). There was no significant rise in heart rate immediately after laryngoscopy and intubation at 0 min in group C (p 0.1), less significant rise in group G (p 0.0869) and highly significant rise in group P (p 0.0001) as

compared to basal value. Heart rate decreased to basal value within 3 min (62.4± 1.4) in group C which took 10 minutes in group G (77.5± 3.5) and more than 10 minutes in group P (88± 1.4)

SBP, DBP and MAP was significantly lower in group C (p value 0.02, 0.001, 0.004) after 90 minutes of premedication as compare to group G (p value 0.61, 0.55, 0.61) and group P(p 0.46, 0.19, 0.46). As shown in table 4,5,6 there was a rise in SBP, DBP and MAP immediately after laryngoscopy and intubation in group G (p

0.0002) and group P(p 0.0001). There was less significant rise in group C (P 0.004) as compared to basal value. In between group comparison, rise was maximum in group P compared to group C and group G. P values were as shown in table 4,5,6. SBP, DBP and MAP reached at basal value within 5 minutes in group C, at 10 minutes in group G and did not return to basal value even after 10 minutes in group P. Statistically highly significant difference was observed between groups at 0,1,3,5,7 and 10 minutes.

Table - 1 - Patients characteristics

	Group C	Group G	Group P
No of patients	25	25	25
Age (years)	48.6±1.8	49.3±2.3	46.9±3.4
Weight (kg)	55.3±3.1	54.2±2.8	52.7±4.1
Sex (M/F)	11:14	12:13	14:11
Pulse rate/min	82.1±1.2	84.2±1.6	82.6±1.4
SBP mm of Hg	126.4±2.1	124.6±2.7	123.5±2.9
DBP mm of Hg	78.1±1.8	76.4±2.0	78.6±2.1
MAP mm of Hg	94.2±1.9	92.4±2.2	93.5±2.3

Data are presented as Mean ± SD

Table - 2 - Sedation Score And Anxiety Score

	Group C	Group G	Group P	P & C	P & G	C & G
	Mean ±SD	Mean ±SD	Mean ±SD	P Value	P Value	P Value
Sedation score	1.12±0.73	0.96±0.68	0.12±0.33	0.0001 *	0.0001 *	0.0220 #
Anxiety Score	0.20±0.4	0.08±0.28	2.42±0.8	0.0001 *	0.0001 *	0.020 #

Data shows as **Mean ±SD** * Statistically significant # Statistically non-significant

Table 3 shows changes in heart rate/min at different time interval

	Group C		Group G		Group P		C&G	P&G	P&C
	Inter-group		Inter-group		Inter-group		Intra-group		
	Mean ±SD	p value	Mean ±SD	p value	Mean ±SD	p value	p value	p value	p value
Before induction	62.2±1.3	0.0001	84.6±3.6	0.05	88±1.4	0.05	0.001	0.3831	0.0001
after induction	64.2±1.3		88.3±3		90.2±4.1		0.0001	0.7101	0.0001
0 min	65.1±1.6	0.1	94.4±4.3	0.0869	107.4±3.8	0.0001	0.0001	0.028	0.0001
1 min	64.3±1.8	0.3	94.1±4.6	0.1104	104.2±3.6	0.0001	0.0001	0.0902	0.0001
3 min	64.2±1.5	0.3	91.6±4.2	0.2118	101.6±3.7	0.0012	0.0001	0.0803	0.0001
5 min	63.2±1.1	0.53	90.6±4.2	0.2835	101.7±4.8	0.0086	0.0001	0.0882	0.0001
7 min	62.4±1.4	0.88	90.7±4.3	0.2821	100.3±4.2	0.0078	0.0001	0.1168	0.0001
10 min	61.8±1.2	0.82	88.5±4.2	0.4842	96±2.8	0.0138	0.0001	0.1439	0.0001

Data shows as **Mean ±SD**

Table 4 shows systolic blood pressure in mm of Hg at different time interval

	Group C		Group G		Group P		C&G	P&G	P&C
	Inter-group		Inter-group		Inter-group		Intra-group		
	mean±se	p value	mean±se	p value	mean±se	p value	p value	p value	p value
Before premed	126.4±2.1		124.6±2.7		123.5±2.9		0.6011	0.7825	0.422
90min after premed	119.6±2.1	0.0265	122.8±2.3	0.6141	126.3±2.5	0.4682	0.3094	0.308	0.0456
after induction	102.6±1.8		119.4±3.2		124.2±4.6		0.0001	0.3959	0.0001
0 min	128.1±1.9	0.0043	138.3±3.0	0.0002	172.3±3.3	0.0001	0.006	0.0001	0.0001
1 min	126.3±1.9	0.0221	134.6±3.0	0.003	168.4±3.6	0.0001	0.0236	0.0001	0.0001
3 min	124.3±1.7	0.0883	128.8±2.2	0.1043	164.3±3.8	0.0001	0.1759	0.0001	0.0001
5 min	121.4±1.6	0.4986	128.2±2.1	0.0894	150.4±3.6	0.0001	0.0131	0.0001	0.0001
7 min	119.8±1.8	0.9427	126.6±2.6	0.2791	148.6±3.4	0.0001	0.0366	0.0001	0.0001
10 min	116.8±1.6	0.2942	123.8±3.2	0.8008	138.1±4.1	0.0177	0.0562	0.0076	0.0001

Data shows as means ± SE

Table 5 shows diastolic blood pressure in mm of Hg at different time interval

	Group C		Group G		Group P		C&G	P&G	P&C
	Inter-group		Inter-group		Inter-group		Intra-group		
	mean±se	p value	mean±se	p value	mean±se	p value	p value	p value	p value
Before premed	78.1±1.8		76.4±2.0		78.6±2.1		0.5304	0.4518	0.8573
90min after premed	70.1±1.6	0.0017	79.3±4.5	0.5587	84.6±4.1	0.199	0.06	0.3883	0.0019
after induction	69.1±1.7		76.4±3.9		80.9±3.9		0.0926	0.4186	0.0079
0 min	74.2±1.9	0.1053	92.6±1.2	0.0063	99.9±4	0.0103	0.0001	0.0869	0.0001
1 min	72.1±1.8	0.4104	88.9±3.6	0.1023	96.8±4.2	0.043	0.0001	0.1597	0.0001
3 min	70.6±1.9	0.8413	85.6±2.6	0.2314	95.4±3.9	0.0623	0.0001	0.0419	0.0001
5 min	69.1±2.2	0.7148	84.1±2.9	0.3744	93.6±4	0.1227	0.0001	0.0604	0.0001
7 min	67.6±1.8	0.3044	80.4±3.2	0.8429	91.8±4.2	0.2259	0.0011	0.0359	0.0001
10 min	67.2±2.0	0.2631	80.7±3.9	0.8151	88.6±3.8	0.4777	0.0034	0.1533	0.0001

Data shows as means ± SE

Table 6 shows mean blood pressure in mm of Hg at different time interval

	Group C		Group G		Group P		C&G	P&G	P&C
	Inter-group		Inter-group		Inter-group		Intra-group		
	mean±se	p value	mean±se	p value	mean±se	p value	p value	p value	p value
Before premed	94.2±1.9		92.4±2.2		93.5±2.3		0.5387	0.7311	0.8155
90min after premed	86.6±1.7	0.0045	93.8±3.7	0.7464	98.5±3.5	0.2384	0.0834	0.3607	0.0036
after induction	80.2±1.7		90.7±3.6		95.3±4.1		0.0112	0.4034	0.0014
0 min	92.1±1.9	0.0266	107.3±2.1	0.0026	124.3±3.7	0.0001	0.0001	0.0003	0.0001
1 min	90.1±1.8	0.1639	104.1±3.3	0.0431	120.6±4.0	0.0001	0.0005	0.0026	0.0001
3 min	88.5±1.8	0.4466	99.3±2.7	0.2357	118.3±3.8	0.0004	0.0017	0.0002	0.0001
5 min	87.5±2	0.7332	98.8±2.6	0.2744	112.5±3.8	0.0093	0.0012	0.0046	0.0001
7 min	85.±1.8	0.5212	95.5±3.0	0.7227	110.7±3.9	0.0242	0.0043	0.0033	0.0001
10 min	83.7±1.8	0.2473	94.6±3.6	0.8775	105.1±3.9	0.2139	0.0093	0.0536	0.0001

Data shows as mean ± se

DISCUSSION

The present study compared the effect of oral clonidine 200mcg and gabapentin 900 mg administered 90 minutes prior to surgery to attenuate the stress response to laryngoscopy and intubation. The result of our study showed significant decrease in heart rate, SBP, DBP and MAP in clonidine group after 90 minutes of premedication whereas in gabapentin group fall in SBP, DBP and MAP was significant but was not significant in HR. Similarly Raval and Mehta⁸ found decrease in SBP, DBP, MAP and pulse rate 90 minutes after premedication in clonidine group was significant.

Immediately after intubation we found statistically significant rise in all parameters in group P whereas it was less significant (or not significant) in group C and group G. H. Talebi and colleague¹⁴ studied 200mcg clonidine as premedication on haemodynamic response to laryngoscopy and intubation and found intergroup significant difference but no significant difference while comparing intragroup. Kamran et al¹⁵ studied 0.3mg oral clonidine and 800mg gabapentin 90 minutes prior to surgery and found less significant rise in HR, DBP, SBP and MAP immediately after endotracheal intubation in group C and group G compare to placebo group. Which significantly decreased at 5,10,15 minutes after intubation compare to before laryngoscopy. It is in concordance with other studies which showed no change in systolic, diastolic and MAP in 200mcg oral clonidine group after laryngoscopy and intubation.⁸ Previous investigations showed that a 300mcg oral clonidine decreases sympathetic activity.⁹ We preferred a dose of 200mcg clonidine because previous study showed that administration of 4 mcg/kg clonidine blunted catecholamine release during intubation and surgery and larger doses were not more effective.¹⁵ This may be because larger dose might cause peripheral α stimulation which could

result in increase in blood pressure. The Matot et al studied oral clonidine 4-4.5mcg/kg in laryngoscopy or bronchoscopic procedure and found effective in attenuating haemodynamic response.¹⁶ In other study by Kalka J with different doses of clonidine and found the effect of clonidine is dose related on haemodynamic variables and sedation and increasing the dose more than 4 mcg/kg does not further enhance efficacy.¹⁷ In most researches 4 mcg/kg clonidine was applied without signs of peripheral α stimulation.^{8,9,13-17}

Clonidine activates alpha-2 adrenoreceptors which causes reduction in peripheral sympathetic tone towards the heart and increases vagally induced reflex bradycardia. This alpha-2 adrenoreceptor agonist activity interacts with the catecholaminergic neuronal system which modulates tonic and phasic (reflex) blood pressure control and reduces release of norepinephrine from the nerve endings towards the vasculature and reduction in peripheral sympathetic tone causing reduction in arterial pressure. Peripherally it stimulates presynaptic alpha adrenoreceptors which again decreases release of noradrenaline.¹⁵

To attenuate the pressure response to laryngoscopy and intubation, studies were done on gabapentin at various doses. Most of these studies showed predominantly blood pressure attenuating effect of gabapentin.^{2,11,18,19} The results of our study suggest that there was a generalised trend towards less haemodynamic response in both clonidine and gabapentin group as compared to the placebo group. We observed gabapentin attenuated the pressure response to tracheal intubation as SBP and DBP which was not raised significantly after tracheal intubation, whereas HR and MAP raised significantly after laryngoscopy and intubation but came to basal value at 3mins. While intergroup comparison with placebo there was no significant difference in

increase of heart rate after laryngoscopy and intubation, but significant difference was there in increase in SBP, DBP and MAP.

Similarly in a study by Tahira Iftikhar and et al²⁰ using 800 mg gabapentin 60 mins before surgery found mean SBP, mean DBP and mean MAP significantly lower but HR was significantly lower only at 10 and 15 minutes after intubation. Other study using 800 mg gabapentin by Kiran and Deepak Verma²¹ observed that heart rate is not significantly attenuated as compared to baseline value although mean increase in heart rate occurs less with gabapentin as compared to placebo. Mean SBP and DBP were significantly lower in gabapentin group at 0,1,3,5 min as compared to control group.

Memis et al¹¹ studied the effect of gabapentin on MAP and HR at induction of anaesthesia and tracheal intubation. Patients receiving placebo and 400 mg gabapentin showed significant increase in BP and HR associated with tracheal intubation compared to baseline levels and patients receiving 800 mg gabapentin showed that 400 mg dose of gabapentin was not adequate to blunt pressure response. The results of our study were similar to the group receiving 800 mg gabapentin in this study.

Usha Bafna and colleagues¹⁹ studied different doses of gabapentin 600 mg and 1000 mg 60 minutes before surgery and found MAP and HR significantly increased in patients receiving placebo and 600 mg gabapentin after laryngoscopy and intubation as compared to patients receiving 1000 mg gabapentin. Fassoulaki and colleagues² showed SBP and DBP significantly lower in gabapentin group than in the control group immediately and 1, 3, 5, 10 minutes after laryngoscopy but heart rate did not differ between two groups at any time. Kayan and colleagues demonstrated attenuation of gabapentin on MAP in the first 10 minutes

following endotracheal intubation.¹⁸ They reported that gabapentin blocked the increase in the MAP but not the HR associated with tracheal intubation. While doing intergroup comparison we found that there was highly significant difference between group C and group G with regard to HR and SBP, DBP and MAP at 90 minutes after premedication (p 0.0001) and immediately after intubation (p 0.0001) and that remains significant up to 10 minutes. The increase in SBP, DBP and MAP in placebo group was statistically highly significant as compared to group C and group G. In accordance with a study by S. Shermak, R Angral²² who found no statistically significant difference between clonidine 300 mcg and gabapentin 800 mg with regard to HR at 0,1 minute after laryngoscopy and intubation (p >0.05). But the mean HR was lesser in group B (clonidine) as compared to group A (gabapentin) at 3,5 and 10 minutes after intubation. Statistically higher arterial blood pressure was noted in group B as compared to group A. Other study by Marashi et al²³ who studied hemodynamic responses after laryngoscopy and tracheal intubation in patients premedicated with 200mcg clonidine and 900mg gabapentin and found the highest rates of HR, SBP, DBP, MAP were in the placebo group at 1 min after laryngoscopy and the lowest SBP, DBP and MAP were in the gabapentin group at 10 min after laryngoscopy and the lowest HR was in clonidine group at 10 min after laryngoscopy.

In contrast to this in our studies we found lowest HR, SBP, DBP, MAP in clonidine group at all time and highest rates were in placebo group. Clonidine and gabapentin both have other beneficial side effects as sedation, anxiolysis and postoperative analgesia and used as anaesthetic adjuvant. We found 21 patients in clonidine group and 22 patients in gabapentin group having sedation score 1 or 0 (awake and calm) and 20 patients in group C and 22 patients in group G having anxiety score 0 (patient quiet and comfortable). Similar to our results Raval and

Mehta found 4mcg/kg clonidine produced both sedation and anxiolysis.⁸ Meningenx C et al²⁴ studied gabapentin to allay anxiety and reported that it produces significantly lower preoperative VAS anxiety scores. We did not assess for postoperative pain. Postoperative analgesia may be an extra advantage of the drug. We observed dryness of mouth in 21 patients in clonidine group may be because of the effect of drug on presynaptic alpha adrenoreceptors in the brainstem as well as on parasympathetic nerves which supply the salivary glands. We did not encounter hypotension, bradycardia or respiratory depression in any of our patients in any group. The most frequently reported side effects are somnolence (20%), dizziness (8%), ataxia (13%) and fatigue (11%). Other side effects are nystagmus, tremor, diplopia and nausea each less than 10%.²¹ However none were observed in our patients.

CONCLUSION

Premedication with oral gabapentin 900 mg or clonidine 200 mcg attenuated cardiovascular responses to the laryngoscopy and tracheal intubation. Gabapentin attenuates the pressure response associated with laryngoscopy and tracheal intubation, but tachycardia response is not completely attenuated. Whereas clonidine provides better attenuation and hemodynamic stability after laryngoscopy and intubation.

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STUDY TO EVALUATE THE EFFICACY OF SINGLE DOSE DEXAMETHASONE INJECTION AS PROPHYLAXIS AGAINST POST DURAL PUNCTURE HEADACHE FOLLOWING SPINAL ANAESTHESIA FOR CAESAREAN SECTION.

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Summary

Objectives: To study the efficacy of a single dose of dexamethasone administered immediately after spinal anaesthesia for CS with 25 G Quincke spinal needle in reducing the incidence and/or duration of post-dural puncture headache (PDPH).

Design: Parallel, randomized, double blind, placebo-controlled prospective study.

Setting: Administration of spinal anaesthesia to Caesarean section patients in three obstetric operation theatre suites in Khoula Hospital, Muscat over a three months period.

Participants: 132 patients undergoing Caesarean section under spinal anaesthesia.

Interventions: Dexamethasone (8 mg) administered immediately after spinal anaesthesia in Study group and compared with saline in the Control group at same time interval.

Main outcome measures: Incidence and duration of PDPH.

Results: Incidence of headache (10.6%) was identical in the two groups. Mean onset time of PDPH was marginally longer and duration slightly

shorter in patients receiving dexamethasone but neither reached the level of statistical significance ($p > 0.05$).

Conclusions: Single dose of dexamethasone administered after spinal anaesthesia in patients undergoing Caesarean section is ineffective in preventing PDPH but prolongs its onset and shortens duration marginally.

Key words: Dexamethasone, spinal anaesthesia, post-dural puncture headache, Caesarean section.

Post dural puncture headache (PDPH) following Caesarean Section (CS) under spinal anaesthesia remains a major problem to both the obstetrician and the obstetric anaesthesiologist. Shaikh et al.¹ and Shah et al.² reported the incidence of PDPH in full term pregnant patients undergoing spinal anaesthesia with 25-gauge Quincke spinal needles as 8.3% and 20% respectively. Steroids are one of the several modalities used to treat established PDPH. Consyntropin 1.0 mg has been shown to decrease the incidence of PDPH following accidental dural puncture and to decrease the need for extradural blood patch.³ More recently, others have demonstrated the efficacy of hydrocortisone in

relieving established PDPH.^{4,5} However, there is great paucity of literature on the prophylactic use of pharmacological agents against the development of PDPH rather than managing an established PDPH. Till date, only one study by Douroudian et al.⁶ have looked at the efficacy of a single dose dexamethasone in providing prophylaxis against PDPH. In their study, a wide bore 22 G Quincke needle was used in a small sample size of 60 patients undergoing orthopedic surgery and failed to note any significant advantage. In the present study, we hypothesized that a single dose of dexamethasone administered immediately after the administration of spinal anaesthesia for CS with a finer 25 G Quincke spinal needle would help to reduce the incidence and/or duration of PDPH.

MATERIAL & METHODS

Following approval by the Hospital Ethical Issues Committee, 132 patients undergoing CS were administered spinal anaesthesia after written consent in this parallel, randomized, double blind, placebo-controlled prospective study. Patients with history of off and on headache, neurological illness, or any contraindication to spinal anaesthesia were excluded from the study. Patients were randomly divided using Chit – in – Box Technique⁷ into two groups: Control Group (n=66)- patients received 2.0 ml of 0.5% bupivacaine intrathecally + 2 ml saline IV. Study Group (n= 66)- patients received 2.0 ml of 0.5% bupivacaine intrathecally + 8 mg dexamethasone in 2 ml IV immediately after spinal anaesthesia.

All patients were premedicated with ranitidine 50 mg and metoclopramide 10 mg intravenously approximately 30-45 min prior to spinal anaesthesia.

Well-experienced anaesthesiologists posted in the obstetrical operation theatre suite performed spinal anaesthesia. Patients of either group

received a preload of 500 ml normal saline prior to the procedure. Intrathecal injection of 2 ml 0.5 bupivacaine was administered in sitting or lateral position through midline approach with bevel facing lateral using 25-gauge Quincke needle with or without 8 mg dexamethasone IV as per group allotment.

All patients received optimal hydration and 1g paracetamol intravenously every 6 hours for the first 24 hours. For any breakthrough pain of surgical site, pethidine 1.5 mg/kg was administered intramuscularly. No restriction of movement was advocated in any of the patient.

Patients were unaware of the group they belonged. A blinded observer visited the patients at 12, 24, 48 and 72 hr after the surgery and interviewed the patient for any evidence of PDPH. If PDPH was confirmed clinically, it was treated as per standard protocol of the hospital that included bed confinement, adequate hydration, simple analgesics and if needed epidural blood patch. Patients were hospitalized and followed up till PDPH had resolved.

STATISTICS

Chi square test has been used to analyze the data. $p < 0.05$ has been considered as significant in this study.

RESULTS

Patients in either group were comparable ($p > 0.05$) in respect of age (Table I).

Table II shows that the mean number of spinal attempts (1.3 in each group) and incidence of headache (10.6% in each group) were identical in the two groups. Mean onset time of PDPH was longer in patients receiving dexamethasone but it did not reach the level of statistical significance ($p = 0.9$). Mean duration of PDPH was shorter in patients of dexamethasone group as compared to

Table I showing mean age of the patient in the two groups.

Group	No. of patient	Mean age in yr ± SD	p-value
Control Group	66	30.7 ± 5.6	0.06
Study group	66	28.8 ± 4.6	

No. = number, yr= year, SD= standard deviation

Table II showing the incidence, mean onset time and duration of PDPH.

Group	Mean Spinal Attempts	Incidence of PDPH No. of pt (%)	Mean onset time hr ± SD	Mean duration in hr ± SD
Control group (n= 66)	1.3 ± 0.6	7 (10.6)	36.0 ± 12.8	58.2 ± 23.8
Study group (n= 66)	1.3 ± 0.7	7 (10.6)	39.4 ± 15.0	54.9 ± 18.9
p-value	0.95	1.0	0.90	0.80

PDPH= post dural puncture headache, n= number, No.= number, pt= patient, hr= hour, SD= standard deviation

Table III showing onset and duration of PDPH in different ranges (hr)

Group	Onset of PDPH (Range in hr)		Duration of PDPH (Range in hr)		
	12-24 hr	24-48 hr	24-48 hr	48-72 hr	>72 hr
	No. of pt (%)	No. of pt (%)	No. of pt (%)	No. of pt (%)	No. of pt (%)
Control Group n= 66	3 (42.8)	4 (57.1)	4 (57.1)	2 (28.6)	1 (14.3)
Study Group n= 66	2 (28.6)	5 (71.4)	3 (42.8)	4 (57.1)	0

PDPH= post dural puncture headache, n= number, No.= number, pt= patient, hr= hour

control group but overall this difference in duration was also insignificant ($p = 0.8$). None of these patients with PDPH needed epidural blood patch to resolve the headache. All responded to conservative line of management as advocated by the hospital.

PDPH started in all patients between 12-48 hours after spinal anaesthesia. With the institution of treatment, PDPH resolved in all patients between

24-72 hours except one patient in the control group in whom mild headache continued for up to 96 hours (Table III).

DISCUSSION

Our hypothesis that prophylactic use of dexamethasone would reduce the incidence of PDPH in patients undergoing CS was based on the fact that we selected a much finer bore spinal needle (25 G instead of 22 G) than the earlier

workers addressing similar problem.⁶This study showed that prophylactic administration of 8 mg dexamethasone does not reduce the incidence of PDPH in patients undergoing CS. However it showed some promise to prolong the onset and reduce the duration of PDPH though without any statistical significance. Doroudian et al.⁶ had investigated the use of prophylactic administration of single dose dexamethasone to reduce the incidence of PDPH in patients undergoing orthopedic surgery. They too did not note any significant difference in the incidence of PDPH ($p= 0.284$) but observed a significant reduction in the intensity of headache ($p= 0.046$). The preventive effect of dexamethasone against PDPH intensity as shown by Doroudian et al.⁶ or marginally delaying the onset and shortening its duration (as shown by the present study) may be due to its anti-inflammatory effect on the inflammatory process initiated at puncture site. Dexamethasone like hydrocortisone is likely to suppress the synthesis of inflammatory mediators in immune cells. This reduction in the release of inflammatory mediators into the CSF would result in lesser stimulation of pain receptors in the central nervous system and hence PDPH.⁵ Steroids are also known to exert their clinical effects by favoring the reabsorption of CSF from the extradural space and thus increase CSF volume.⁸ However, steroids have no effect on CSF production.⁹

Unlike our hypothesis expectation, these mechanisms did not play any significant role in clinically reducing the incidence or duration of PDPH after a single dose of dexamethasone. It is quite possible that the result may have been different if dexamethasone administration was done as multiple dosing over first 24-48 hours instead of a single dose regimen. This option may be explored in future studies.

In conclusion, single dose dexamethasone offers minimal protection against onset, duration or

overall incidence of PDPH when administered to patients undergoing CS under spinal anaesthesia.

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