VOLUME 22
NUMBER 2
JULY 2009

Journal of the Bangladesh Society of Anaesthesiologists

JOURNAL OF THE BANGLADESH SOCIETY OF ANAESTHESIOLOGISTS

VOL 22, NO. 2, JULY 2009

EDITORIAL BOARD

This is the official Journal of The Bangladesh Society of Anaesthesiologists, published biannually in January and July by Scientific Committee of BSA.

Correspondence for publication of manuscripts : Dr. Md. Abdul Hye, (Executive Editor), Department of Anaesthesia, Analgesia and Intensive Care Medicine, Bangabandhu Sheikh Mujib Medical University, Shahbag, Dhaka - 1000. Requests for reprints by the author should be made to the Editorial Office after receiving the letter of acceptance. Price for reprints will be for every 25 copies.

This journal is officially sponsored by S. T. MARKETING CORPORATION LTD. & CITY ELECTRO-MEDICS CO. as part of their continuing educational support.

Printed at the Asian Colour Printing, 130, DIT Extension Road, Fakirerpool, Dhaka-1000, Phone : 9357726

BANGLADESH SOCIETY OF ANAESTHESIOLOGISTS

EXECUTIVE COMMITTEE
2009-2011

President	:	Dr. UH Shahera Khatun
Senior Vice-President	:	Dr. Rokeya Sultana
Vice President	:	Dr. M Manzoorul Hoq Laskar
Secretary General	:	Dr. Debabrata Banik
Treasurer	:	Dr. AKM Nur Nobi Choudhury
Joint-Secretary	:	Dr. Md. Mozaffer Hossain
Organising Secretary	:	Dr. Paresh Chandra Sarker
Scientific Secretary	:	Dr. Md. Abdul Hye

Members :

Dr. Abdul Mannan	Dr. Iqbal Hossain Chowdhury
Dr. Md. Khalilullah	Dr. MA Shukur
Dr. Md. Jahangir Kabir	Dr. Md. Rezaul Huda
Dr. Md. Abdul Karim	Dr. AKM Azizul Hoque
Dr. Md. Hasibul Hossain	Dr. Md. Abdul Kalam Azad Khan
Dr. AKM Akhtaruzzaman	Dr. Md. Muniruzzaman Siddiqui
Dr. Anwar Ahmed	Dr. Wahiuddin Mahmud
Dr. Moinul Hossain	Dr. Md. Noor-E-Alam Siddiquee

Ex-Officio Members
Dr. SN Samad Choudhury, Dr. ABM Muksudul Alam

SCIENTIFIC SUB COMMITTEE

Chairman : Prof. Monirul Islam

Member Secretary : Dr. Md. Abdul Hye

A. JOURNAL SUB-COMMITTEE

Members

Dr. Moinul Hossain
Dr. Gulshan Ara Chowdhury
Dr. Manzoorul Hoq Laskar
Dr. Debasish Banik
Dr. AK Qumrul Huda
Dr. Sabina Yeasmeen
Dr. Amirul Islam
Dr. Subrata Kumar Mondal
Dr. Kawser Sarder
Dr. SM Abdul Alim
Dr. Mohammad Abdul Karim Miah

B. SEMINAR SUB-COMMITTEE

Members

Prof. AKM Azizul Haque
Prof. A Khaleque Beg
Prof. Hasibul Hossain
Prof. Jalilur Rahman Barbhuya
Dr. Md. Rezaul Islam
Dr. Mahmudur Rahman Khandokar
Dr. Anwar Hossain Hawlader
Dr. Aminul Islam
Dr. Mazibar Rahman
Dr. Harun-Ur-Rashid
Dr. Sohel Ahmed
Dr. Montosh Kumar Mondal
Dr. Md. Abdul Karim
Dr. Nargis Fatima
Dr. Habibur Rahman
Dr. Motiur Rahman
Dr. Nihar Ranjan Kundu
Dr. Nigar Jahan
Dr. Mohsinuzzaman Chowdhury
Dr. Helaluddin Chowdhury
Dr. Masud Ahmed
Dr. Saiful Islam
Dr. Manzurul Alam
Prof. Ahsanul Habib
Dr. Rezaul Haque PK
Dr. Masumul Haque
Dr. ANM Badruddoza (Dipu)
Dr. Akhter Hossain Loban
Dr. Brig.Gen(Rtd) Dr. Razia Khanam
Dr. Rafiqul Islam
Col. (Rtd) Dr. M.A Hamid
Dr. Ragib Manzoor
Dr. Shafiqul Islam

GUIDE TO CONTRIBUTORS

For preparation of manuscripts, please follow the guidelines as described in **Uniform requirements for manuscripts submitted to Biomedical Journals** published in the *Ann lalern Medi 1997; 126 :36-37 (Reproduction: this issue).* For quick reference following checklist may be useful.

Articles

Articles submitted for printing in this journal must not be published in whole or in part in any other journal. They are subject to revision by the Editorial Board and the publishers own the copyright of the papers accepted for publications.

Articles on clinical investigation should abide by the ethical standards set out in Declaration of Helsinki. About animal studies, the author should convince the Board that the involved animals have not been subjected to pains or suffering not absolutely necessary for the sake of the finding.

Use of names, initials etc. should be avoided so that patients must be unrecognizable in photographs unless permission in writing is obtained from him/ her to that effect. Proposal for reproduction of an illustration of data published elsewhere should be accompanied by a written undertaking that the original author and publishers have granted permission for this.

Manuscript should be accompanied by a formal letter of request for publication and it should be signed by all the authors. Manuscript needs to be limited within 2000-3000 words and submitted in duplicate, with authors qualifications and full addresses. It should be typed with a 5 cm margin at the left hand side of the sheet and in double space on one side of A4 paper by the english language. The manuscripts are also to be submitted in electronic foam (on the disc). It is suggested that the authors retain a copy. If the manuscript is rejected, it may be returned if posted is covered. The editor reserves the right to style and if necessary to shortens the material accepted for publication. The editor also reserves the right to determine the priority and time of publication. Editor believes that the work is based on honest investigation and observation. It is not the duty of the editor to investigate scientific fraud paper.

The articles should be divided in general into the following parts:

Summary

Introduction

Materials & Methods

Results

Discussion

Acknowledgements (if any)

Tables and Illustrations

Title Page:

The title page should be in a separate page which will include apart from the title of the paper, the name (s), professional degree (s) and address (es) of the author (s). Addresses should be clearly indicated with respect to the relevant authors. If authors present addresses differ from those at which the study was carried out should be given as a footnote and appropriately referenced in the authors list. The title page should be paginated as page I of the article. A short running title having not more than 50 letters should also be suggested.

Introduction:

The introduction should not be headlined. Please start a new sheet. It should be in the form of a concise account of the background of the problem and the object of the study. Foregoing work should be quoted only if it has a direct bearing on the present problem.

Materials & Methods:

Methods should be described in detail sufficient to allow the work to be interpreted and repeated by the reader. Modifications of previously published methods should be explained and appropriately referenced. In case of commonly used methods, only mention of the original source should be suffice. Statistical analysis should be described in the materials and methods and are to be supported by references if possible.

Results:

Results of the experiments should be brief and in sequence. Avoid unnecessary repetition of data in the text, tables or illustrations. Significance should be given as 'P' values. Total number of tables, charts and figures should be limited, approximately one for each 500 words.

Drugs:
At the time of mentioning a drug for the first time, the generic or official name should be used.

Discussion:
The discussion should be used to interpret the results of the study in the background of the current knowledge. Repetition of the data for sake of mere recapitulations is unwanted. The discussion should also include on which the conclusion is drawn.

Reference:
It is required that each of the papers submitted for printing is accompanied by a list of references at the end. These references should be arranged according to the Vancouver system. If the reference is made; name of the journal or book in which published and abbreviated according to cumulative index medicos, year of publication; volume number in Arabic numerals; the number of the first appearance in the text and numbered accordingly. In the text, references should be numbered with Arabic numerals placed as superscript. Examples:

Paper Published in a journal

1. Yamashita M., Matsuki A., Oyamei T. Anaesthetic considerations in Von Recklinghusen's disease. Anesthetist 1977, 26;317-323.
2. Books
3. Article in books : Wise RP, Wylie WD. The thymus gland: Its implications in clinical anesthetic practice. In: Jennings TM., ed. Clinical Anesthesia: Anaes these for patients with endocrine disease. Philadelphia, Davis and Co. 1988; 178-181. text reference to personal communication or unpublished observations by the concerned person lies with the author.

Reference to papers submitted and accepted for publication may be included in the phrase "in press" replacing volume and page numbers. Authors are solely responsible for the verification of authenticity and content of the references.

Tables:
All tables should be on separate sheets with proper captions and be self explanatory. They should be numbered consecutively using Roman numbers. Units in which data are expressed should be given in brackets at the top of the relevant column. Ditto signs should not be sued. All units of measurements should be expressed in SI System if not otherwise required.

Illustration:
Graphs charts, drawings etc. Photographs should be unmounted glossy prints and protected adequately for mailing. Surfaces should not be marred by clips. pins or heavy writing on the back. Illustrations should be clearly numbered on the back, preferably with soft pencil, with reference to the text and using Arabic numbered on the back, preferably with soft pencil, with reference to the text and using Arabic numerals. They should be accompanied on a separate sheet with a suitable legend.

Lettering should be professional looking and uniform, large enough to be read clearly at a reduced size. Magnification in photomicrograph itself. The name of the author and title of the article should be written on the back with a soft pencil on the back of the illustrations.

Note
Please send your articles to the following address

Dr. Md. Abdul Hye
Associate Professor and Chairman
Department of Anaesthesia, Analgesia and Intensive Care Medicine, Bangabandhu Sheikh Mujib Medical University, Shahbag, Dhaka - 1000.

JOURNAL OF THE BANGLADESH SOCIETY OF ANAESTHESIOLOGISTS

VOLUME - 22 **NUMBER - 2** **JULY 2009**

CONTENTS

Editorial

Smokers' Anaesthesia

Smoking is a personal and social hazard worldwide that is frequently overlooked. In developed part of the globe the habit has shown a recent down trend, but unfortunately in developing nations it is yet on a rising trend. What is more appalling is that, raw and less refined tobacco is sold in these countries. Though there is govt rule firing for smoking in public place, seldom it is implemented.With limited govt. and NGO initiative at least the affluent part of society is aware of the harmful effect of smoking. Malignancy, hypertension, IHD, DM renal disease, airway infection, bronchial asthma all are worsened by smoking. Active smoking is responsible for 90% of lung cancer, smokers shows 20 fold rise of lung cancer[1]. Smoking related risk of squamous cell carcinoma and small cell carcinoma is more than adenocarcinoma[2].But people irrespective of strata are not aware of the surgico anaesthetic hazard of smoking. Much needed to be done by govt, NGO, mass media and BSA as well to make people conscious of the magnitude effect of smoking in this field. BSA in the light of ASA guideline of quitting smoking(available at www.smokefree.gov) may chalkout its own smoking abstinence program.

Poor surgical healing, delayed and poor anaesthetic recovery, high carboxy haemoglobin causing hypoxia refractory to oxygen therapy[3]. Perioperarive airway spasm, retention of sputum leading to focal to global pulmonary collapse all should be taken into account. People should be motivated that everyone is vulnerable to undergo anaesthesia at some stage of life. Patient scheduled for an upcoming surgery should be motivated to abstain for good. At least 3 weeks abstinence can correspond with half life of already circulating CO.A prospective study on 6026 patients undergoing GA comparing smokers(S) to non smokers(NS) showed significant high ICU transfer in former[4]. Delgado study demonstrated Increased ICU admission and more death in smokers in a sample of 2989 patients[5]. Schwilk study in 26961 surgical patients shows relative risk (RR) or respiratory events 2.3 in young smokers, 6.3 in overweight smokers, while RR of bronchospasm is 25.7 in young with COPD[6].Moller randomized study demonstrates cardiovascular complication 0%&10% while overall complication 15% & 52% in S versus NS[7]. In another series on surgical wound healing complication were 2% & 12% in nonsmoker and smoker group[8]. Necrosis of mastectomy scrap were smokers7%. Meta analysis of vascular by-pass in a randomized trial showed significant by pass failure in smokers[10].

Conclusion:

It is now fairly evident that added to non sugicoanaesthetic complication of malignancy, cardio-reno-pulmonary complication smokers has more valnurability to poor recovery, delayed healing, hypoxia. atelactasis, airway spasm etc. Surgery is therefore a good opportunity on the part of anaesthtist to motivate patient to abstain from smoking permanently. It is also high time that social conscience about perioperative health hazads is raised by launching combined effort from govt.ngo,media and health professionals.

Prof. Munirul Islam
Head of Anaesthesiology
Mymensingh Medical College, Mymensingh

(Journal of BSA, 2009; 22(2): 38-39)

References:

1. Alberg et al epidiomology of lung cancer.Chest 03;123:21-49.
2. Wyender et al: Smoking releted risk of lung cancer Cancer 1988;62:1223-30.
3. Moller et al:A study of impact of long term smoking on post operative intensive care admission;Anesthsia 2003Jan 50.
4. Delgado et al:A prospective study of tobacco smoking and prediction of complication following sutgery:In feet contr.hosp.epidiomo 12003:Jan 24.
5. Myles et al : Risk of respiratory complication in smokers following surgey:Anesthesiology 2002,97(4) 842-847.

6. Schwilk et a1:Perioperative respiratory complication in smokers versus on smokers undergoing GA: Acta anesthesiol stand 1997;41 (3) 348-58.

7. Moller et al: Effect op preoperative smoking on post operative complication.Lancet 2002 Jan12 114-17.

8. Sorensen et a1: Abstinence from smoking decreases incidence of wound infection, randomized control trial Acta surg. 2003 Jul 1-5.

9. Padubidrzi et al: Complication following breast reconstruction in smokers,non smokers and ex smokers; Plast. reconst. surg. 2001Feb342-48

10. Willigendal et al: Smoking and patency of lower extremity by pass graft; J vas.surg. 2005 Jul,42; 67-74.

Original Article

Clinical outcome of the off-pump coronary artery bypass surgery- a comparison between combined high thoracic epidural anaesthesia with GA and GA alone

Saiful Islam Azad[1*], Abdul Khaleque Beg[2], A. Y. F. Elahi Chowdhury[3], U. H. Shahera Khatun[4]

[1]Assistant Registrar, [2]Professor and Head, [3]Ex. Professor and Head, Dept. of Anaesthesiology,NICVD, Dhaka, [4]Professor and Head, Dept.of Anaesthesiology and ICU, DMCH, Dhaka, Bangladesh.

***Corresponding author:** E-mail: saifulazad@yahoo.com*

Abstract:

Background: The common challenges for the cardiac anaesthesiologist during off pump coronary artery surgery (OPCAB) include haemodynamic stability during the different stressful surgical events and multiple cardiac manipulations, providing adequate myocardial protection, and obtaining effective postoperative analgesia leading to early discharge from the intensive care unit.

Objective: This study has been undertaken with a view to find out whether a combined high thoracic epidural anaesthesia (HTEA) with general anaesthesia (GA) is safe and more efficient in providing overall cardiovascular stability as well as improving the parameters leading to a better outcome in terms of a shorter and more predictable road maps to recovery.

Methods: Sixty patients aged within 40-70 years, without having any coagulopathy disorder , any emergency surgery or left main disease scheduled for CABG on beating heart were enrolled in prospective, randomized observational comparative study. Patients were divided in two groups. In group A patients received GA alone and in group B patients received high thoracic epidural anaesthesia with GA. The parameters including heart rate , SPO2 , CVP , arterial blood pressure, ECG, and ABG analysis were recorded before induction, during induction , intubation and during different events of the surgery. Post operative pain score , sedation score , ventilator hour, duration in the ICU stay , rescue analgesic need and post operative complications was assessed and recorded.

Results: significant per-operative mean heart rate changes were observed all the events except at wound closure and during anastomosis with D1/D2 and the mean difference of mean arterial pressure at intubations, skin incision, sternotomy, pericardiotomy, during anastomosis of distal end of the graft with RCA, PDA, LCX and D1/D2 were observed statistically significant ($p<0.05$) . No incidence of different arrhythmia occurred in group B, premature ventricular complex (PVC) was statistically significant ($p<0.05$) between two groups. No significant change was found in per-operative pH of arterial blood, $PaCO_2$ and PaO_2 changes at different times. Post operative pain score (VAS 0-100) in different time interval was found significant ($p<0.05$) change between two groups in all follow-up times. Status of rescue analgesics were observed statistically significant ($p<0.05$). The mean ventilator hours were 7.4±1.09 hours in group A and 5.3±0.81 hours in group B. The mean ICU stay was 72.9±9.2 hours in group A and 57.1±12.0 hours in group B. No post-operative complication was observed in both groups. The data were compiled and analyzed by using statistical software SPSS (ver. 12.0) and significance test performed by unpaired t test and Chi square test. P value <0.05 was considered as statistically significant.

Conclusion: Both anaesthetic techniques are equally safe but better clinical outcome of the OPCAB surgery with the high thoracic epidural anaesthesia with GA.

***Keywords:** HTEA, GA, OPCAB*

(Journal of BSA, 2009; 22(2): 54-60)

Introduction:

General anaesthesia is the most commonly used anaesthetic technique and considered the "gold standard" for coronary artery by-pass grafting (CABG) performing either on pump or off pump[1].During general anaesthesia in cardiothoracic surgery neurohumoral response is an important problem for patients who is augmented

by various painful surgical interventions[2,3] and haemodynamic changes during endotracheal intubation may be detrimental in patients with coronary artery diseases due to concomitant increase in myocardial oxygen demand, decrease in oxygen supply, and the possibility of myocardial ischaemia [4-6]. In the postoperative period inadequate analgesia may increase morbidity by causing adverse haemodynamic, metabolic, immunologic and haemostatic attentions[7-10]. This is often difficult to achieve optimal pain relief.During beating-heart surgery, to obtain an adequate exposure of the anastomosis site with restrained cardiac motion; and to protect the myocardium from ischaemia during coronary artery flow interruption the anaesthetist must be prepared to handle severe haemodynamic alterations, transient deterioration of cardiac pump function, and acute intraoperative myocardial ischaemia. Within the past few years, high thoracic epidural anaesthesia (HTEA) as an adjunct to general anaesthesia (GA) has become more prevalent[1,11] and has been shown to be potentially beneficial in patients with coronary diseases. Thoracic epidural anaesthesia provides good protection from stress response[12,13], ensures hemodynamic stability[12,14], allows early extubation[15], improves distribution of coronary blood flow and reduce demand for oxygen[16]. Awake CABG is associated with a certain amount of undeniable psychological stress and therefore requires excellent patients' compliance. There may be significant hemodynamic compromise due to Trendelenburg position and luxation of the heart during revascularization of the circumflex artery so circumflex artery disease represents an exclusion criterion for this anaesthetic technique. In addition, because the anaesthetic level does not extend to the lower limbs, TEA alone is limited to complete arterial revascularization[1]. So combination of high thoracic epidural anaesthesia and general anaesthesia will provide better intraoperative and postoperative better outcome in off pump CABG surgery than general anaesthesia alone. In the previous studies in different countries, various benefits have been reported in combining thoracic epidural anaesthesia with general anaesthesia during cardiac surgery.The statistical risk of epidural haematoma following TEA as between 1: 150,000 and 1: 1500(95% confidence) and up to 1: 1000 (99% confidence)[17].In this study we have tried to evaluate the clinical outcome of the OPCAB surgery including the above mentioned limitations of the previous related studies with the anaesthetic technique combined high thoracic epidural anaesthesia with GA.

This study has been undertaken with a view to find out whether a combined HTEA with GA is safe and more efficient.

Materials And Methods:

Sixty patients aged between 40-70 years, ASA I, II, III & IV and NYHA – I, II, III & IV scheduled for OPCAB were included in this study and divided into two groups, thirty in each group and were randomly selected. Group A patients received GA alone and group B patients received high thoracic epidural anaesthesia with GA. Patients were excluded if they had left main disease, history of previous cardiac surgery, left ventricular ejection fraction <35% ,contraindications of regional anaesthesia and emergency surgery. After selection of the patients, grouping, entry of name of the patient in the case record form and the written informed consent was taken from all patients on the preoperative day. Pre-medication with oral midazolam 7.5mg was given in the night before surgery. Patients were fasted six hours before operation.After transferring the patients into the operation room, standard monitoring (five lead ECG, pulse oxymetry) was instituted. All patients were pre-oxygenated with 100% oxygen. With all aseptic precaution intravenous cannulation, radial arterial catheterization and central venous catheterization was established. Epidural catheter was inserted at the level of T1-2 or T2-3 interspaces in the patients of group B . Fifteen minutes before surgery , high thoracic epidural anaesthesia was given in group B patients with 0.25% bupivacaine 10 ml bolus followed by 8 ml/hr through continuous epidural infusion during operative period and general anaesthesia was induced in all patients of both group with fentanyl 10μg/kg IV , propofol 1mg/kg IV .Tracheal intubations were facilitated by pancuronium bromide 0.1 mg/kg and anaesthesia was maintained with propofol infusion 50-100μg/kg/min, fentanyl infusion 1-2 μg/kg/hr , oxygen 35% and air 65%. The parameters including heart rate , SPO2 , CVP , arterial blood pressure , , arrhythmia in ECG, and ABG analysis were recorded before induction, during induction , intubation and during different events

of the surgery (skin incision, sternotomy, pericardiotomy, coronary artery anastomosis with graft , sternum closure and wound closure). At the end of the surgery , all patients were transferred to the intensive care unit without extubation and ventilation was maintained by the ventilator. When there were accepted criteria for tracheal extubation ,patients were extubated and shifted to the post operative care unit after accepted criteria for discharge from intensive care unit. Post operative analgesia was performed by Ketorolac 30 mg IV every eight hourly for all patients but in group B , in addition to this , thoracic epidural infusion 0.25% bupivacaine 3-4 ml/hr up to 48 hours was given also. If need rescue analgesics was given. Post operative pain score , sedation score , postoperative ventilator hour, duration in the ICU stay , rescue analgesic need, and post operative complications (respiratory, neurological, and epidural haematoma) was assessed and recorded. The data were compiled and analyzed by using statistical software SPSS (ver. 12.0) and significance test performed by unpaired t test and Chi square test. P value <0.05 was considered as statistically significant, P<0.01 highly significant .

Results:

Table I

Anthrometric characteristic of the study patients
ns= Not significant

	Group A (n=30) Mean±SD	Group B (n=30) Mean±SD	p value
Age (years)	49.9 ±7.1	49.3±7.2	0.789 ns
Weight (kg)	62.3 ±7.4	62.1±9.9	0.934 ns
Height (cm)	151.4±5.5	148.9±15.7	0.490 ns
BSA (m^2)	1.6 ±0.11	1.7±0.15	0.410 ns

No significant mean age, weight, height and body surface area (BSA) differences were found between two groups (Table I).

Table II

Changes in heart rate during study period

	Group A (n=30) Mean ±SD	Group B (n=30) Mean ±SD	p value
Pre-op value	79.3 ±7.5	77.2±13.8	0.538 ns
Induction	83.2 ±14.1	72.2±11.1	0.007
Intubation	93.3 ±11.8	75.3±11.9	0.001
Skin incision	88.3 ±13.7	74.2±11.3	0.001
Sternotomy	92.3 ±11.7	76.2±12.4	0.001
Pericardiotomy	86.9 ±14.9	78.0±11.6	0.028
Sternum closure	96.9 ±13.5	85.6±13.4	0.007
Wound closure	94.3 ±13.7	86.9±13.5	0.075 ns
During anastomosis of distal end of the graft with			
LAD	92.5 ±14.5	81.3±10.4	0.004
RCA	103.6±17.7	90.6±7.4	0.018
PDA	104.8±18.7	86.4±9.1	0.016
LCX	110.3±11.0	67.0±6.5	0.001
D1/D2	98.6 ±13.5	93.6±17.8	0.229 ns
OM1/ OM2/ OM3	78.4 ±13.4	87.2±12.7	0.011
During anastomosis of proximal end of the graft with the aorta	98.7 ±18.7	87.4±13.9	0.023

The mean heart rate changes at wound closure and during anastomosis with diagonal (D1/D2) were not statistically significant (p>0.05) in unpaired t-test and others were statistically significant (p<0.05) (Table II).

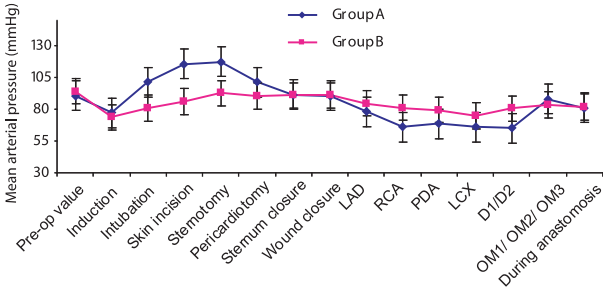

Fig.-1: *Line diagram showing mean arterial pressure (mmHg) changes.*

The mean of mean arterial pressure changes at intubation, skin incision, sternotomy, pericardiotomy, during anastomosis of distal end of the graft with RCA, PDA, LCX and D1/D2 were statistically significant and others were not statistically significant (Figure 1).

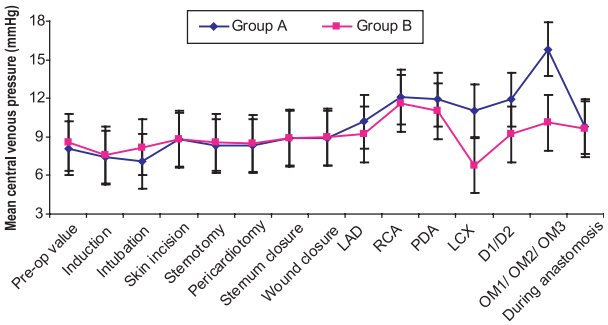

Fig.-2: *Line diagram showing central venous pressure changes .*

The mean central venous pressure changes during anastomosis of distal end of the graft with PDA, LCX, D1/D2 and OM1/OM2/OM3 were statistically significant and others were not statistically significant (Figure 2).

Table III

Per-operative different arrhythmia (n=60)

	Group A (n=30)		Group B (n=30)		p value
	n	%	n	%	
Ventricular tachycardia	3	10.0	0	0.0	0.118 NS
Ventricular fibrillation	1	3.3	0	0.0	0.500 NS
Premature Ventricular complex (PVC)	9	30.0	0	0.0	0.001
Atrial fibrillation	0	0.0	0	0.0	-
Supra ventricular tachycardia	3	10.0	0	0.0	0.118 NS

No incidence of different arrhythmia occurred in group B. Premature ventricular complex was statistically significant between two groups but other arrhythmia was not statistically significant (Table III).

Table IV

PH of arterial blood changes at different times. ns= Not significant

	Group A (n=30) Mean ±SD	Group B (n=30) Mean ±SD	p value
Pre-op value	7.41 ±0.02	7.42±0.05	0.238 ns
Intubation	7.46 ±0.04	7.44±0.02	0.555 ns
Sternotomy	7.39±0.06	7.43±0.04	0.190 ns
Pericardiotomy	7.42±0.03	7.43±0.04	0.280 ns
Sternum closure	7.39±0.04	7.42±0.02	0.107 ns
Wound closure	7.40±0.05	7.42±0.04	0.196 ns

PH of arterial blood changes between two groups within normal range in both groups (Table IV).

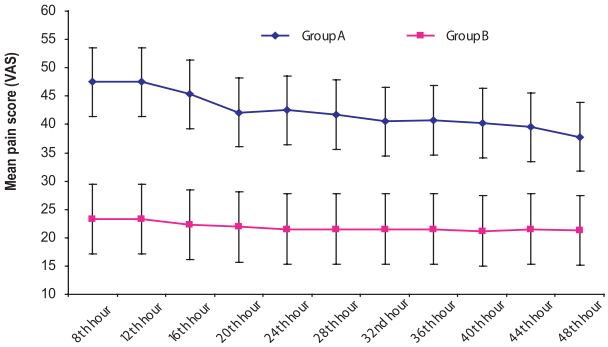

Fig.-3: *Line diagram showing Post operative pain score(VAS; 0-100) of the patients after extubation.*

The post operative pain score (VAS; 0-100) after extubation in different time interval found significant ($p<0.05$) change between two groups in all follow-up times (Fig.-3).

Table V

Mean distribution of post operative sedation score at first six hour

One hour interval	Group A (n=30) Mean±SD	Group B (n=30) Mean±SD	p value
1st hour	2.9±0.3	2.8±0.5	0.352 ns
2nd hour	2.8±0.5	2.1±0.6	0.001
3rd hour	2.4±0.7	1.4±0.5	0.001
4th hour	2.1±0.5	1.1±0.3	0.001
5th hour	1.4±0.5	1.0±0.0	-
6th hour	1.15±0.4	1.0±0.0	-

The mean distribution of post operative sedation score at first six hour were significant change between two groups except 1st hour, which was not significant (Table V).

Table VI

Status of rescue analgesics received by the patients

Rescue analgesics	Group A (n=30)		Group B (n=30)		p value
	n	%	n	%	
Received	16	53.3	6	20.0	0.015
Not received	14	46.7	24	80.0	

The status of rescue analgesics received was statistically significant in chi square test (Table VI).

Table VII

Mean ventilator hour and duration of ICU stay

	Group A (n=30) Mean±SD	Group B (n=30) Mean±SD	p value
Ventilator hour	7.4±1.09	5.3±0.81	0.001
ICU stay	72.9±9.2	57.1±12.0	0.001

The difference of the mean postoperative ventilator hour and duration of ICU stay were statistically significant (Table VII).

Discussion:

In the present study the mean age, weight, height and body surface area of the patients were similar in both groups but were higher in different studies than the present study. The higher range of the different studies may be due to increased life expectancy in their country.In the current study the male patients was predominant, which is consistent with Kessler et al.[1] , Salvi et al.[20] and Scott et al.[19] studies. In this study it was observed that per-operative mean heart rate changes at wound closure and during anastomosis of distal end of the graft with D1/D2 were not statistically significant ($p>0.05$), however rest of others times were significantly ($p<0.05$) less in group B, which is comparable with Kundu et al., Kessler et al., and Salvi et al. Studies[18,1,20]. It was found in this current study that per-operative mean of mean arterial pressure (MAP) changes at intubation, skin incision, sternotomy, pericardiotomy were significantly ($p<0.05$) higher in group A but during anastomosis of distal end of the graft with RCA, PDA, LCX and D1/D2 were ignificantly ($p<0.05$) higher in group B and others were almost similar between two groups. Kessler et al.[1] obtained consistent findings in their study in group B, which is similar with the present study, however in group A the present study found higher with the above study findings before anastomosis but during anastomosis of distal end of the graft with different coronary artery mean of MAP were decline below 70 mmHg. Similarly, Salvi et al.[20]reported identical finding in this regard, which is closely support the present study. In this study it was observed that per-operative mean CVP changes at during anastomosis of distal end of the graft with PDA, LCX, D1/D2 and OM1/OM2/OM3 were significantly ($p<0.05$) higher in group A and others were almost consistent between two groups. On the other hand Kessler et al. (2005)[1] and Salvi et al. (2004)[20] have observed identical mean CVP of their patients, which are closely resemble with the present study.

In this study it was found that per-operative arrhythmia ventricular tachycardia (VT) 10.0% in group A but none was found in group B which is similar with Kundu et al.[18] study. Arrhythmia ventricular fibrillation (VF) was 3.3% in group A and none was found in group B, which closely resembles with Bakhtiary et al. Study[21]. Arrhythmia premature ventricular contraction

(PVC) was 30.0% in group A and none was found in group B, which also support Bakhtiary et al. Study[21], where they observed 27.3% and 6.1% in group A and group B respectively and Kundu et al.[18]study observed 40.0% and 10.0%. Arrhythmia atrial fibrillation (AF) not occurred in both group in the present study, however Kundu et al.[18]observed in this study 10.0% in group A. Bakhtiary et al. [18] reported 27.3% in group A and 6.1% in group B, which differ with the present study, this may be due to left main disease was excluded and GA was given with TIVA in the presents study.

In the present study, per-operative pH of arterial blood was changed within normal limit in both groups and per-operative $PaCO_2$, PaO_2 changes at different times had no significant ($p>0.05$) change between two groups which support Kessler et al. study[1].Kessler et al.[1] have assessed the VAS (0-100mm) were subsequently higher in group A than group B at all times after surgery, always reaching significance level except at 48 hours. Similarly Salvi et al.[20]assessed the VAS (0-10 mm) for the first 24 hour period were 0.9 at rest and 1.7 during coughing in each patients the VAS score always less than <2, which indicating that the post-operative pain relief was excellent in their study patients (group B). In the present study it was found that post operative pain score VAS (0-100mm) was >40 in group A and <30 in group B after extubation which was significantly ($p<0.05$) higher in group A at all the different follow-up times. The results obtained in the present study are comparable with the above studies. In the present study post operative sedation score at first hour was almost similar between two group, however the remaining times sedation score were significantly ($p<0.05$) higher in group A. Kessler et al.[1] reported that sedation score were significantly higher at 6 hour post operatively in group A. In the current series it was observed that the status of rescues analgesics need 53.3% in group A and 20.0% group B and the difference was statistically significant ($p<0.05$). In this study no post-operative complication was found between two groups. In a study Scott [19] done a study on 202 patients in group A and 206 in group B and found the incidence of postoperative confusion was 5.5% and 1.5% in group A and group B respectively. The incidence of CVA was 3.0% in group A and 1.0% in group B. In this study it was found that the mean ventilation hours was significantly ($p<0.05$) higher in group A, which were 7.4±1.09 in group A and 5.3±0.81 hours in group B. Similarly, the mean ICU stay was also significantly ($p<0.05$) higher in group A, which were 72.9±9.2 hours in group A and 57.1±12.0 hours in group B. Kessler et al.[1]observed the higher mean ventilation hours in group A, which was 5.0±2.6 hour in group A and 3.7±2.4 hours in group B. The mean ICU stay was also higher in group A, which was 11.2±7.9 hours and 9.2±8.1 hours in group B. Salvi et al.[26] observed the mean ventilation hour was 4.6±2.9 in group B and the mean ICU stay was 36.0±19.2 hours.

Conclusion:

Based on the present study results, both anaesthetic techniques were equally safe. However, HTEA with GA appeared to be more safe and efficient.

References

1. P. Kessler ,T. Aybek , Gerd Neidhart, Selami Dogan , Volker Lischke, Dorothee H Bremerich, et al., Comparison of three anesthetic techniques for Off-pump coronary artery bypass grafting: general anaesthesia, Combined general and high thoracic epidural anaesthesia, or high thoracic epidural anaesthesia alone. *Journal of Cardiothoracic and Vascular Anaesthesia* 2005;19: 32-39.
2. Didem Dal ,Meral Kanbak ,Meltem Caglar ,Ulku Aypar . A background infusion of morphine does not enhance postoperative analgesia after cardiac surgery.*Can J Anesth* 2003; 50(5): 476-479.
3. Wolman RL. Patient controlled analgesia following thoracic surgery. In: Gravlee a Rauk (Eds). Pain management in cardiothoracic surgery. Philadelphia: Lippincort company ;1993; 59-87.
4. Stoelting RK. Circulatory changes during ditrect laryngoscopy and tracheal intubation. Influence of duration of laryngoscopy with or without prior lidocaine. *Anaesthesiology* 1977;47: 381-3.
5. Ebert JP, Pearson JD, Gelman s, Harris C, Bradley EL. Circulatory responses to laryngoscopy: the comparative effects of placebo, fentanyl and esmolol.*Can J. Anesth* 1989; 36: 301-6.
6. Stone DJ, Gal TJ. Airway management. In: Miller Rd (Ed.) Anaesthesia, 5th. New york;

Churchill Livingstone Inc; 2000;1444-6.

7. Mark A. Chaney, Intrathecal and Epidural Anaesthesia and Analgesia for Cardiac Surgery.*Anesth Analg* 2006;102:45-64.
8. Weissman C. The metabolic response to stress: an overview and update. *Anesthesiology* 1990;73:308-27.
9. Kehlet H. Surgical stress: the role of pain and analgesia. *Br J Anaesth* 1989;63:189-95.
10. Roizen MF. Should we all have a sympa-thectomy at birth or at least preoper-atively. *Anaesthesiology* 1988;68:482-4.
11. Goertz AW, Seeling W, Heinrich H, et al., Influence of high thorasic epidural anaesthesia on left ventricular contractility assessed using the end systolic pressure length relationship.*Acta Anaesthesiol Scand* 1993;37:38-133.
12. Blomberg S, Curelaru I, Emanuelsson H, et al., Thoracic epidural anaesthesia in patients with unstable angina pectoris.*Eur Heart J* 1989;10:437-44.
13. Reiz S, Nath S, Rais O, Effects of thoracic epidural block and prenalterolon coronary vascular resistance and myocardial metabolism in patients with coronary artery disease. *Acta Anaesthesiol Scand* 1980;24:11.135.
14. Blomberg S, Emanuelsson H, Ricksten SE, Thoracic epidural anaesthesia and central hemodynamics in patients with unstable angina pectoris.Anesthesiology 1990; 73: 840–7.
15. Kock M, Blomberg S, Emanuelsson H, et al. Thoracic epidural anaesthesia improves global and regional left ventricular function during stress induced myocardial ischemia in patients with coronary artery disease. *Anesth Analg* 1990;71:625-30.
16. RF Davis, LW DeBoer, PR Maroko, Thoracic epidural anaesthesia reduces myocarial infract size after coronary artery occlusion in dogs. *Anaesthesia and Analgesia* 1986; 965:7l 1-717.
11. Goldstein Dean, SJ Kim, et al. A survey of spinal and epidural techniques in adult cardiac surgery. *J Cardiothoracic Vase Anesth* 2001;15:158-168.
12. JF Olivier , N.Le, et al. Comparison of the three different epidural solutions in off-pump cardiac surgery: pilot study. *Br J Anaesth* 2005;95(5):685-691.
13. Ganapathy S, Murkin JM, Dobkowski W, Boyd D. Stress and inflammatory response after beating heart surgery versus conventional bypass surgery: the role of thoracic epidural anaesthesia. *Heart Surg Forum* 2001;4:323-7.
14. Stenseth R, Bjella L, Berg EM, Christensen O, Levang OW, Gisvold SE. Thoracic epidural analgesia in aortocoronary bypass surgery. I: haemodynamic effects. *Acta Anaesthesiol Scand* 1994; 38: 826–33.
15. Joachimsson PO, Nystrom SO, Tyden H. Early extubation after coronary artery surgery in efficiently rewarmed patients: a postoperative comparison of opioid anaesthesia versus inhalation anaesthesia and thoracic epidural analgesia.*J Cardiothorat Anesth* 1989;3:444-54.
16. Stenseth R, Berg EM, Bjella L, Christensen O, Levang OW, Gisvold SE. Effects of thoracic epidural analgesia on coronary hemodynamics and myocardial metabolism in coronary artery bypass surgery. *J Cardiothorac Vasc Anesth* 1995; 9: 503–9.
17. Ho AM, Chung DC, Joynt GM. Neuroaxial blockade and hematoma in cardiac surgery: estimating the risk of a rare event that has not (yet) occurred. *Chest* 2000;117:551-555.
18. RK Kundu, AK Beg, S Hossain, N Haque, Mahafuddoza, A Chowdhury. Comparative study of haemodynamic status and arrhythmia between combined epidural with general anaesthesia and general anaesthesia alone in Off-pump coronary artery bypass (OPCAB) surgery. *J Bangladesh Coll Phys Surg* 2008;26:116-120.
19. Scott MB, Turfrey DJ, Ray DA. Aprospective randomized study of the potential benefits of thoracic epidural anaesthesia and analgesia in patients undergoing coronary artery bypass grafting. *Anesth Analg* 2001;93(5):28-35.
20. Salvi L, Sisillo E, Brambillasca C, Juliano G, Salis S and Marino MR. High thoracic epidural anaesthesia for off-pump coronary artery by pass surgery. *J Cardiothorac Vasc Anesth* 2004;18(3):256-62.
21. Bakhtiary F, Therapidis P, Dzemali O, Ak K, Ackermann H, Meininger D, Kessler P, Kleine P, Moritz A, Aybek T, and Dogan S. Impact of high thoracic epidural anaesthesia on incidence of perioperative atrial fibrillation in off-pump coronary bypass grafting: A prospective randomized study. *Thorac Cardiovasc Surg* 2007;134:460-4.

Original Article

Use of labetalol and glyceryl trinitrate for induced hypotension in spine surgery- A comparative study

Sabina Yeasmeen[1*], Md. Rafayet Ullah Siddique[1], Amirul Islam[2], AKM Aktaruzzaman[3], Debabrata Banik[3], Abdul Hye[3].

[1]Assistant Professor, [2] Medical Officer, [3]Associate Professor, Department of Anaesthesia, Analgesis and Intensive Care Medicine, BSMMU, Dhaka

***Correspondence author**, E-mail: yeasmeens@yahoo.com

Abstract:

Background: *Induced hypotension is used to reduce blood loss especially in those operations where even a small amount of blood can obscure the operative field such as spine surgery.*

Objectives: *To compare the effect of labetalol with that of glyceryl trinitrate to reduce intraoperative blood loss by inducing elective hypotension without any tachycardia and to improve quality of surgical field during spine surgery.*

Method: *A total number of thirty patients (ASA grade I & II) were selected randomly into two groups, fifteen in each group. Group-I received glyceryl tinitrate (1000µg) and Group-II received labetalol (5mg) intravenously 3 minutes before induction of anaesthesia. Heart rate, mean arterial pressure was observed in two study groups 15 minutes interval in intra-operative period and quality of surgical field was detected by 4 points VRS (Verbal rating scale) after completion of surgery by asking the surgeon.*

Results: *Heart rate varied significantly in two study groups after induction of anaesthesia except baseline and pre induction ($p< 0.05$). Labetalol associated with improved quality of surgical field visualization than glyceryltrinitrate ($p = 0.034$).*

Conclusion: *Our study concluded that labetalol is effective than glyceryltrinitrate to reduce blood loss in spine surgery.*

Key words: *spine surgery, labetalol, glyceryltrinitrate, intraoperative period, quality of surgical field, mean arterial pressure, verbal rating scale.*

(Journal of BSA, 2009; 22(2): 48-53)

Introduction:

Anaesthesia may influence intraoperative bleeding in several ways both physiologically and pharmacologically. Elective hypotension is used to reduce operative blood loss especially in those operations like spine surgery[1], middle ear surgery, cerebro-vascular surgery where even a small quantity of blood can obscure the operative field and make difficulties for the surgeon for a good proper surgery and prolong duration of operation[1]. Laryngoscopic stimulation of oropharyngeal structure may be an important factor in the haemodynamic stress response associated with tracheal intubation.[2] Instrumentation of pharynx and tracheal intubation may result in tachycardia, hypertension and increased plasma catecholamine concentration that may evoke life threatening condition among susceptible individuals especially those with cardiovascular disease.[3] Hypotensive anaesthesia has long been established as safe as effective method for reducing blood loss upto 80%.[4]

Various types of pharmacological agents used to achieve hypotensive anaesthesia have been studied during spine surgery. They include ganglion blocking agents, volatile agent.[5] Calcium channel blocker[6], sodium nitropruside[7],

nitroglycerine[8], α & β adrenergic blocker[9]. Mean arterial pressure (MAP) is typically maintained at 60-70 mmHg. These drugs, however, are not entirely satisfactory, as tachyphylaxis and undesirable tachycardia often occur[7]. There is little evidence that any particular agent is superior but the avoidance of tachycardia is an essential part of a good anaesthetic technique.

Labaetalol is both α & β adrenergic receptor blocker which reduces blood pressure without significant altering either resting heart rate or cardiac output[9]. Glyceryl trinitrate alternative relaxes vascular smooth muscle with venous dilatation predominating over arterial dilatation reduces blood pressure by reducing preload but it causes reflex tachycardia[6].

In our present study we compare the effects of labetalol with that of glyceryltrinitrate for safe elective hypotensive anaesthesia to reduce the blood loss so as to produce a clear surgical field during intraoperative period of spine surgery.

Methods

Study population:

Total 30 patients of type of spinal pathology scheduled for spinal surgery was recruited for the study. University departmental ethics committe give consent before carrying out the study. Informed consent were taken from all patients before the procedure.

After recruitment, thirty patients of both sex, male and female aged 15-60 years , ASA grade I and II divided in two groups. Fifteen patients in each group. Patients were randomly divided into two groups. According to card number, patients grouped. Patients with history of bronchospasm, significant ventricular hypertrophy, Sinus bradycardia (< 45-50 beats/min), allergic rhinitis, 2nd and 3rd degree heart block, congestive heart failure, diabetes mellitus were excluded from this study.

Patients data were collected from pre anaesthetic assessment audit form. Preoperative parameters (pulse rate, systolic and diastolic blood pressure, mean arterial pressure, SpO_2) were recored. Intravenous canula inserted on left hand. After recording pulse, blood pressure, SpO_2, Group-I received glyceryl trinitrate (1000μg) and Group-II was received inj. Labetalol (5mg) intravenously 3 minutes before induction of anaesthesia.

Pre-oxygenation was done for 3-5 minutes with 100% oxygen, induction of anaesthesia was done with fentanyl (1μg/kg) and thiopentone sodium (5mg/kg) and endotracheal intubation was done by suxamethonium (1.5mg/kg). Maintenance of anaesthesia with N_2O 70%, O_2 30% and halothane (0.5% -1%) with non depolarizing neuromuscular blocking agent vecuronium bromide (0.1mg/kg) incremental dose of fentanyl (0.3 – 0.4μg/kg) was given as needed. Intraoperative fluid was maintained with Hartmann's solution or normal saline. Intraoperatively 15 minutes interval pulse rate, systolic and diastolic blood pressure and mean arterial pressure observed. Mean arterial pressure was maintained at 70-80 mmHg. Quality of surgical field was observed by points VRS (Verbal rating scale) (1. Not satisfactory 2. Moderate satisfactory, 3. Good, 4. Excellent) and detected by asking the surgeon after completion of surgery. In Group-I glyceryl trinitrate (1000μgm) given at 15 minutes interval and also given as required to maintain desired mean arterial pressure and in Group- II. labetalol (5mg) given at 30 minutes interval also given as required. Operation time was on average about two hours.

Statistical Analysis:

All the variables were expressed as mean ± SD. Student t-test and chi-square (χ^2) test were done as the tests of significance where applicable to compare the mean of different groups. The statistical analysis was done by using SPSS programme. P value < 0.05 was considered as significant.

Results:

Observation of the present study was analyzed in the light of comparison among each subject groups. Each group having n=15. All results were expressed as mean ±SE or in frequencies as applicable. The groups became statistically matched for age (p=0.366), weight (p=0.697), sex (p=.705). ASA grading (p=0.713). There was no significant difference among the study groups.

Table-I

Demographic data of two stud y group.

Group/variable		Group-I(n=15)	Group-II(n=15)	P value
Age (years)		41.27±2.809	38.07±2.046	0.366
Weight (kg)		60.20±3.854	62.07±2.763	0.697
Sex	Male	9 (60.0)	10 (66.7)	0.705
	Female	6 (40.0)	5 (33.3)	
ASA	I	8 (53.3)	9 (60.0)	0.713
	II	7 (46.7)	6 (40.0)	

Data was analyzed by unpaired students 't' test, Values are regarded significant P < 0.05.

Table-II

Changes of heart rate in two study group.

	Baseline	Pre-induction	15min after induction	30 min after induction	45 min after induction	60 min after induction	75 min after induction	90 min after induction	105 min after induction	120 min after induction
Group-I	81.80±	85.60±	90.93±	86.07±	84.40±	80.80±	78.93±	82.85±	74.40±	68.00±
(n=15)	2.219	2.276	2.074	2.804	2.348	2.487		2.6452.780	3.855	0.0
Group-II	82.13±	83.07±	82.73±	74.67±	74.07±	68.73±	69.73±	67.27±	64.33±	65.00±
(n=15)	1.633	2.525	2.817	2.937	3.586	2.504	2.381	1.832	2.042	2.408
t-value	.121	.745	2.344	2.808	2.411	3.419	2.585	4.679	2.308	.509
P-value	.905^{ns}	.462^{ns}	.027^{s}	.009^{s}	.024^{s}	.002^{s}	.015^{s}	.001^{s}	.058^{s}	.638^{ns}

Values are expressed as mean ±SD. Data are analyzed by student t test, values are regarded as significant P<0.05. ns = non significant, s =significant

Baseline mean heart rate in Group-I was 81.80±2.219 and in Group-II was 82.13±1.633, where, p= 0.905 (Table-II).

Pre-induction mean heart rate in Group-I was 85.60±2.276 and in Group-II 83.07±2.525, where p=0.462.

Heart rate of two studied groups are displayed, where baseline and pre-induction values were not significantly difference in two groups.

Heart rate varied significantly at 15min (p=0.027), 30 min (p= 0.009), 45 min (p= 0.024), 60 min (p= 0.002), 75 min (p= 0.015), 90 min (p= 0.001) after induction of anaesthesia.

Heart rate of two study groups displayed in Fig: I

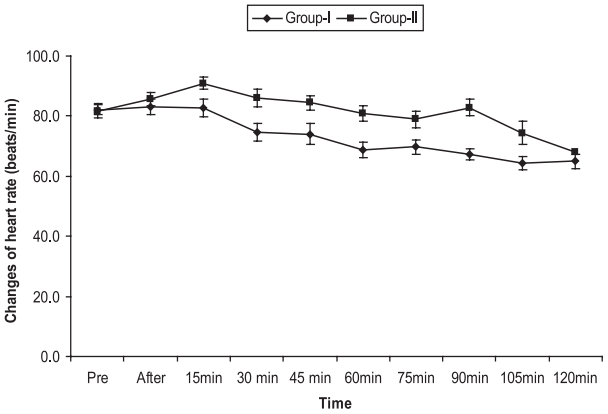

Fig. 1: *Changes of heart rate in two study groups*

Regarding heart rate there is slight increase in heart rate in group I. There was significant change in Fig.-1.

Table-III

Changes of mean arterial pressure in two studies group.

	Baseline	Pre-induction	15min after induction	30 min after induction	45 min after induction	60 min after induction	75 min after induction	90 min after induction	105 min after induction	120 min after induction
Group-I	101.00± 2.591	97.00± 2.908	97.07± 4.975	86.00± 2.878	78.93± 3.051	86.60± 4.591	84.80± 3.234	85.92± 2.690	83.33± 3.333	84.33± 3.333
Group-II	98.27± 3.724	93.40± 3.634	80.33± 1.985	77.93± 1.666	77.67± 2.352	75.93± 1.578	78.13± 2.204	80.83± 2.063	81.00± .577	82.00± .577
t-value	.602	.773	3.124	2.425	.329	2.197	1.704	1.501	.690	.590
P-value	.552^{ns}	.446^{ns}	.006 ^{s}	.024 ^{s}	.745 ^{ns}	.042 ^{s}	.101 ^{ns}	.147 ^{ns}	.558 ^{ns}	.548 ^{ns}

Values are expressed as mean ±SD. Data are analyzed by student t test, value are regarded as significant P<0.05.

Baseline mean arterial pressure in Group I was 101.00±2.951 and in Group II was 98.27± 3.591 where p =0.552 (Table-III).

Pre-induction mean arterial pressure in Group-I was 97.00±2.908 and in Group-II 93.40±3.634 where p = 0.446.

Mean arterial pressure of two studied groups are displayed, where baseline and pre-induction values were not significantly different in two groups, but varied significantly at 15 min (p = 0.006), 30 min (p = 0.024), 60 min (p = 0.042) after induction of anaesthesia.

Mean arterial pressure of study groups is displayed in Fig: II.

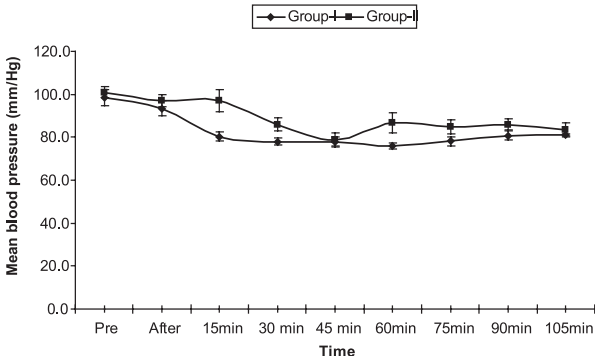

Fig.-2: Changes of mean arterial pressure in two study groups

Regarding mean arterial pressure there is slight change in two groups. The difference between two groups are not significant showed in Fig: 2.

Table-IV

Quality of surgical field by verbal rating scale (VRS) in two study groups.

	Group-I	Group-II	Total	χ^2-value	P-value
Not satisfactory	3(20.0%)	0 (.0%)	3(10.0%)		
Moderate satisfactory	8(53.3%)	4(26.7%)	12(40.0%)		
Good	426.7%	8(53.3%)	12(40.0%)	8.667	0.034^{s}
Excellent	0(.0%)	3(20.0%)	3(10.0%)		
Total	15(100.0%)	15(100.0%)	30(100.0%)		

Data analyzed by chi-square (χ^2) test, value are regarded as significant (P <0.05).

Improved quality of surgical field visualization was detected after completion of surgery by asking the surgeon by verbal rating scale (VRS). "Bleeding obscure surgical field" group I =3, group II= 0. Moderate

bleeding obscure surgical field group I =8, group II= 4, Mild bleeding but surgical field is clear group I =4, group II= 8, Excellent surgical field group I =0, group II= 3 (Table-IV).

Group-II associated with significant improved quality of surgical field (p=0.034).

Discussion:

Elective hypotension is a specific anaesthetic technique which goes beyond the ability of a good, safe, non-stress-inducing anaesthetic to reduce blood loss. Its use may be classified broadly as applicable to situations in which the particular operation would otherwise be impossible (for example cardiovascular or cerebrovascular surgery), Situations in which excessive blood loss might be detrimental(for example orthopaedics, spinal, and maxillo-facial operations) and situations in which blood loss interferes with surgical visibility or technique(such as middle ear surgery and spine surgery).

Bloodless surgical field helps the surgeon to operation in short time and without damage of any important nerve roots due to clear visible surgical field.

The benefits of induced hypotension during spine surgery include reduction in blood loss and also reduce need for blood transfusion, improved quality of surgical field and shorter duration of surgery. A number of hypotensive agents have been studied include ganglion blocking agents, volatile agents, calcium channel blocker, sodium nitropruside, nitroglycerine, â-blocker. These drugs however are not entirely satisfactory as tachyphylaxis or undesirable tachyardia often occur[6].

Labetalol has stereoisomers[9,10,14]. It is a racemic mixture of four isomers. Two of these isomers, the (S,S)- and (R,S)- forms are inactive. The third, the (S, R)-isomer, is a powerful α_1 blocker. The fourth isomer, the (R, R)-isomer, is a mixed nonselective β blocker and selective β_2 antagonist.

It works by blocking these adrenergic receptors, which decreases peripheral vascular resistance without significantly altering heart rate or cardiac output.[9] The α:β antagonism of Labetalol is approximately 3:1 [15,16]. Glyceryl causes venous dilatation predominating over arterial dilatation reduced blood pressure by reducing preload but it causes reflex tachycardia[6,13].

Kadam PP[12] studied hypotensive anaesthesia for spine surgery comparing nitroglycerine vs halothane. In this prospective study 30 patients (ASA I or II) requiring spine surgery under general anaesthesia were studied. Group-I received halothane 0.5-2.5% and group- II received intravenous nitroglycerine infusion 1-2 μg/kg/min. They studied blood pressure, blood loss, operating time and recovery score. In their study there was no significant between groups differences in patients demographic data. There was no significant difference in haemodynamic parameters. The blood loss with nitroglycerine was significantly less (202±114ml) than halothane groups (602±312 ml). All the patients were alert at the end of surgery in nitroglycerine groups (recovery score 9.8± 0.76) as against the halothane groups (7.98±0.9) (p< 0.01).

By comparing with this study, in our study we found there were no significant difference between groups in patients demographic data and less blood loss in Labetalol group. Kaplan et al[13] did not observe significant change in heart rate when nitroglycerine was used to control blood pressure during coronary artery surgery. They suggested that gradual reduction in blood pressure by nitroglycerine prevented an increase in heart rate. But in our study heart rate varied significantly at 15 min (p =0.027), 30 min (p =0.009), 45 min (p =0.024), 60 min (p =0.002), 75 min (p =0.015), 90 min (p =0.001) after induction of anaesthesia except base line and pre induction.

There was no significant difference in mean arterial pressure between groups except 15 min (p=0.006), 30 min (p =0.024), 60 min (p =0.042), after induction of anaesthesia.

Also in our study labetalol was associated with improved quality of surgical field than glyceryltrinitrate 10%, 0% respectively (p=0.034).

In our study, 1 patient in labetalol group, 2 patients in glyceryltrinitrate required blood transfusion.

So from above observation we found that heart rate remains stable in labetalol group than glyceryltrinitrate intraoperatively. Mean arterial pressure was maintained at 70-80 mm of Hg in labetalol group. Dose requirement and time interval decrease in labetalol group and also decrease the requirement of blood transfusion.

So our result showed that labetalol (5mg) administered intravenously 3 minutes before

induction of anaesthesia is effective than glyceryltrinitrate to reduce blood loss intraoperatively and improved quality of surgical field in spine surgery.

Conclusion:

Under the condition of present study, it was found that intravenous labetalol is a more safe hypotensive agent in comparison to glyceryltrinitrate in spine surgery.

References:

1. Grundy BL, Nash CL, Brown RH. Deliberate hypotension for spinal fusion. Prospective randomized study with evoked potential monitoring. Cand Anaesth J 1982; 29: 452-462
2. Forbes AM, Dally FG. Acute hypertension during induction and endotracheal intubation in normotensive man. Br J Anaesth 1970; 42: 18-628.
3. Prys-Roverst SG, Green LT, Neloche R, Foep. Study of anaesthesia in relation to hypertension hyperdynamic consequences of induction of endotracheal intubation. Br J Anaesth 1971; 43: 531-547. .
4. Malcolm- Smith NA, Mack Master MJ. The use of induced hypotension to control bleeding during posterior fusion for scoliosis. J Bone Joint Surg 1983; 65: 255-8.
5. Tobias JD. Sevoflurance for controlled hypotension during spine surgery: Preliminary experience in five adolescents Paediats Anaesth 1998; 2;167-70.
6. Her Sey SL, O' Dell NE, Dowe S, et al. Nicardipine Versus nitroprusside for controlled hypotension during spinal surgery in adolescents. Anas Analg 1997; 84: 1239-44.
7. Bale R, Powler A, Nyatt R. Intravenously glyceryltrinitrate haemodynamic effect clinical use in cardiac surgery. Br J Anaesth 1982; 54: 297-301.
8. Richards DA, Prichard, BNC, Boaker AJ, Tuckman JE, Knigh EJ. Pharmacological basis for antihypertensive effects of intravenous Labetalol, Br Heart J 1977; 39: 99-106.
9. Scott, DB, Budkleg, FOUND, Drummono GB, Little Wood, DGE Macrae WR et al. Cardiovascular effects of Labetalol during halothane Anaesthesia. Br J Clin Pharmac 1976; 3: 817-821.
10. Jones SER. Controlled hypotension using labetalol and halothane anaesthesia 1979; 34: 1052-1055.
11. Uirich PE Jr, Keene JS, Hogan KJ, Rocekor FB. Results of hypotensive anaesthesia in operative treatment thoracol umber fractures. J Spinal Disord 1990; 3: 329-33.
12. Kadam PP, Sakesena SG, Jagtap SR, Pantavaidya SM. Hypotensive anaesthesia for spine surgery. J Postgrade Med 1993; 39(1): 26-28.
13. Kalplan JA, Dunbar RW, Jones EL. Nitroglycerin as a hypotensive drug during general anaesthesia. Anaesthesiology 1978; 20: 17-20.
14. Riva E, Mennini T, Latini R .The alpha- and beta-adrenoceptor blocking activities of labetalol and its RR-SR (50:50) stereoisomers. Br. J. Pharmacol 1991; 104 (4): 823–8.
15. Katzung, Bertram G. Basic and clinical pharmacology. New York: McGraw-Hill Medical. 2006; p. 170.
16. D A Richards, J Tuckman, and B N Prichard. Assessment of alpha- and beta-adrenoceptor blocking actions of labetalol. Br J Clin Pharmacol. 1976; **3** (5): 849–855.

Case Report

Perioperative anaesthetic management of a child with cushing's syndrome for bilateral adrenalectomy

Sabina Yeasmeen[1*], Debasish Banik[2], Quamrul Huda[2], Sayeed Mahmud Ali Reza[1], Abdul Hye[3], Anwar Ahmed[4]

[1]Assistant Professor, Associate Professor, Professor, [3] Consultant, Department of Anaesthesia, Analgesis and Intensive Care Medicine, BSMMU, Intensive Care Medicine, BSMMU, Dhaka

**** Corresponding author:*** *e-mail: yeasmeens@yahoo.com*

Abstract:

Nearly twenty five percent of the cases of cushing's syndrome are due to adrenal hyperplasia without an ACTH secreting tumour. Twenty percent of patients with endogenous cushing have adrenocortical tumour about half of which are benign adenoma. Surgical intervention done due to failed medi cal therapy and in case of adrenal adenoma. A child aged 4½ years, weighting 29kg with features suggestive of cushing's syndrome was admitted under paediatric surgery unit in Bangabandhu Sheikh Mujib Medical University. On investigation serum cortisol levels were raised. blood pressure was controlled by ACE inhibitor, calcium channel blocker and beta-blocker. He was scheduled for resection of adrenal cortical tumour. Electrolyte imbalance was corrected, steroid replacement was done. Patient was haemodynamically stable preoperatively.Surgery was completed unevenetfuly. Postoperatively patient was kept in ICU, ventilation maintained by control mode (CMV). After 24 hours the patient was extubated. When the patient found haemodynamically stable he was sent to the recovery room.

Key words: *Cushing's syndrome, anaesthetic management, bilateral adrenalectomy.*

(Journal of BSA, 2009; 22(2): 84-87)

Introduction:

Cushing's syndrome is a clinical entity resulting from adrenocortical hyperfunction. The most common cause is iatrogenic resulting from administration of corticosteroids. Twenty five percent of the cases are due to adrenal hyperplasia without an ACTH secreting tumour.[1] Other causes of cushing's syndrome because bodies produce too much cortisol. Normally, the production of cortisol follows a precise chain of events. First, the hypothalamus, a part of the brain about the size of a small sugar cube, sends corticotrophin-releasing hormone (CRH) to the pituitary gland to secrete adrenocorticotropin hormone (ACTH), which stimulates the adrenal glands. When the adrenal gland receive the ACTH. They respond by releasing cortisol into blood stream.[2] Most causes of cushing's syndrome are not inherited. young adults develop small cortisol producing adrenals tumour with multiple endocrine neoplasm type I (MEN) hormone secreting tumours of the parathyroid glands. Pancreas and pituitary develops cushing's syndrome due to pituitary ectopic or adrenal tumour.[4,5] This case reports described the perioperative anaesthetic management of child who is undergoing bilateral adrenalectomy.

Case Report:

A 4½ years old male child presented with complaints of excessive weight gain for last 2½ years and increased appetite for same time was seen in picture 1.

On General Examination: Patient was obese, weighting 29kg, Body Mass Index (BMI) was-35.8kg/m^2, moon shaped face, trunchal obesity, buffalo hump, facial acene, hypertrichosis, pulse-140/min, blood pressure 170/110 mmHg, respiratory and cardiovascular system was normal, per-abdominal striae were visible. Haematological and Biochemical investigation were normal, X-ray chest normal and ECG shows sinus tachycardia. X-ray skull- Sella turcica was normal. 2D Echocardiography revealed left ventricular ejection fraction (LVEF) 60%, CT brain – Mild cerebral

atrophy, 24 hr urinary cortisol was – 427 µgm/ 24hrs, serum cortisol (after 1 mg Dexamethasone at mid night)- 39.2µg/dl. USG of adrenal gland – bilateral enlarged suprarenal gland.

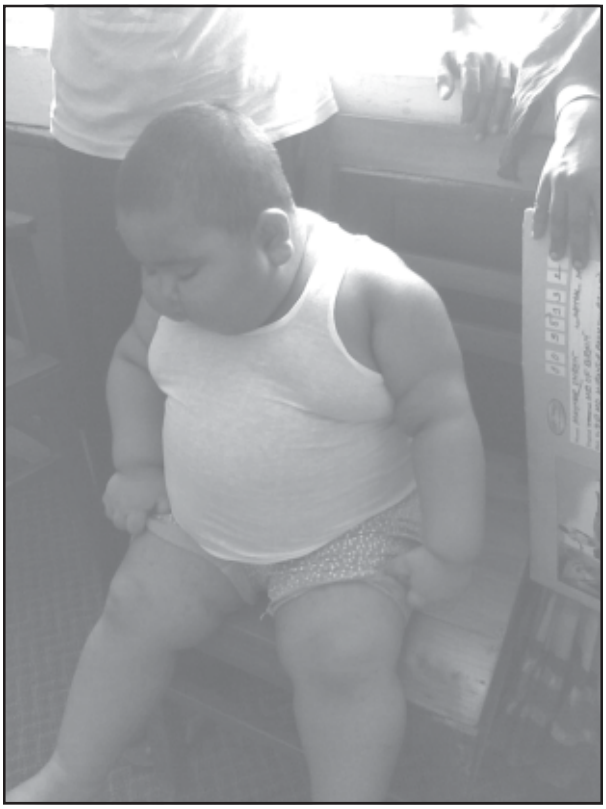

Fig. 1: *Moon shaped face and trunchal obesity are suggestive of cushing's syndrome*

Patient was scheduled for bilateral adrenalectomy as ASA grade III. Difficult intubation wasdue to limited mouth opening, Mallampati grade IV, short neck so adequate and difficult intubation cart was kept ready. Patient was optimized preoperatively, hypertension was controlled with tab Nifedipine (10mg) three times daily, tab. Captropril (25mg) 6.5mg at night. tab. Propranolol (20mg) two times daily. Patient received antibiotic prophylaxis. Preoperative sedative and premedication was avoided. Routine antihypertensive medication were given in the morning tab. Ranitidine (150mg) was given at night. Inj. Hydrocortisone 60mg intravenous bolus was given preoperatively andperoperatively Inj. Hydrocortisone infusion 100mg in 500ml normal saline was started at 3-5mg/hr. In operation theatre patients heart rate was 114/min, blood pressure was 150/78 mmHg, SpO_2–92%. Non invasive blood pressure, ECG and SpO_2, temperature was continuously monitored.

Intravenous canulation was done with 22 G canula. Preoxygenation was done with 100% O_2 for 5 minutes. Anaesthesia was induced by . fentanyl - 30µg,. thiopental sodium – 100mg, intubation was done by rocuronium 25mg intravenously. Intubated the patient with cuffed endotracheal tube 4.5mm size internal diameter. Anaesthesia was maintained with halothane 0.5%, 50% oxygen, 50% N_2O. Incremental doses of . fentanyl and atracurium were given.

Intraoperatively, due to meticulous surgical dissection and adequate depth of anaesthesia, there were no major blood pressure fluctuation during adrenal manipulation except slight decrease in blood pressure after resection of bilateral adrenal gland which was controlled by . 0.9% normal saline 500ml mixed with dopamine (400mg) I/V at. 10 microgram/ kg / min. Duration of surgery was 3 hours.

Though vasodilator like sodium nitroprusside, glycerintrinitrate were kept ready for blood pressure control, it were not required. Fluid supplementation was given as 500ml crystalloid solution. Blood loss was minimal. Urine output was maintained through out the procedure. At the end of surgery neuromuscular blockade was reversed with neostigmine 1.2mg and atropine 0.18mg .As patient could not maintained spontaneous respiration then patient was sent to the intensive care unit for mechanical ventilation. for next 24 hours.

Postoperatively arterial blood gas and blood sugar, serum electrolyte were monitored.The patient was kept in ICU and electively ventilated in controlled mode (tidal volume 300ml, respiratory rate-14/min). FiO_2 –0.6. Postoperative pain relief was done by. pethedine (1mg/kg) 6 hourly, introtropic support was given by dopamine (200mg) in 50 ml in N/S at 10. microgram/kg /min Hypokalemia was corrected by potassium containing syrup through nasogastric tube. Hyperglycaemia was controlled by 30unit insulin and 50ml normal saline at 1 dial/min (according to the blood sugar). Patient received steroid supplementation with. hydrocortisone succinate by infusion 100mg in 500ml normal saline Extubation was done 24 hours after surgery in ICU. When the patient vital signs were stable patient then send to recovery room.

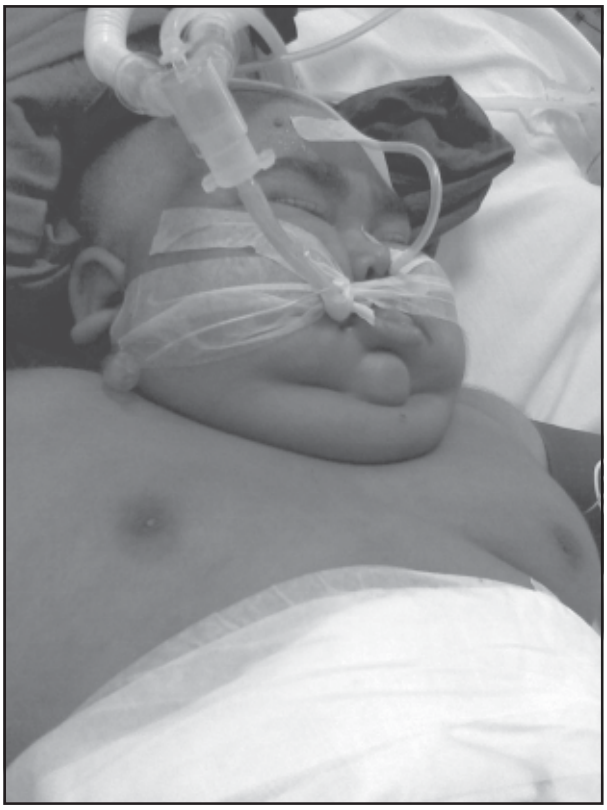

Fig.-2:: *Patient was in controlled mode ventilator in ICU.*

Discussion

Cushing's syndrome is clinical entity resulting from adrenocortical hyperfunction. The signs and symptoms of cushing's syndrome are related to excess glucocroticoids. Patients present with increased body weight, truncal obesity with buffalo hump, cutaneous striae, odema, glucose intolerance.[1,3] All these presentation make these patients a challenge to anaesthesists. The most common cause of cushings syndrome is iatrogenic administration of corticosteroids. Approximately 40% of endogenous causes are ACTH producing tumours and ACTH producing non pituitary tumour such as tumours of the lung, prostate, testis, parotid or pancreas. Nearly 25% of cases are due to adrenal hyperplasia without ACTH secreting tumours, 20% of patients have adrenocortical tumour, about half of which are benign adenomas. This patient was a case of bilateral adrenal hyperplasia. The clinical presentation with cushinoid feature.[2]In this case, hormonal levels showed gross elevation of cortisol, MRI did not show pituitary or adrenal neoplasm. While CT abdomen and brain yielded normal study, ruling out multiple endocrine neoplasm (MEN) which is frequently associated with higher incidence of bilateral pheochromocytoma in children (20%) than in adults.[6,7,8]

Hypertension due to adrenal hyperplasia in children tends to be more sustained along with salt retention and increased intravascular and interstitial volume.[7] Patients may present with paroxymal symptoms like palpitation, trembling, sweating due to dopamine and epinephrine secretion. This patient was on tab Nifedepine and tab Captopril two times daily preoperatively. Surgical intervention is indicated for failed medical therapy and in cases of adrenal adenoma.[9]

The key factor is to remember when preparing patient for surgery is to get medically stabilized. Hyperglycemia is best controlled with regular insulin if needed. In this patient preoperative sugars were controlled (FBS=5.5mmol). The patient scheduled for bilateral adrenalectomy should be treated as addisonian crisis intraoperatively and postoperatively because normal adrenal tissue is suppressed by high level of circulating corticosteroids.[10] Therefore, steroid therapy was instituted by giving . hydrocortisone 60 mg I/V half an hour prior to surgery, followed by 3-5mg/hr infusion peropertively and continued postoperatively. Goals of anaesthetic management were aimed at suppressing response to endotracheal intubation. Surgical stimulation, adrenal handling and devasculrization plus providing optimal surgical condition as in case of pheochromocytoma.[8,11] Histamine releasing sedative premedicants are avoided in view of difficult airway. The anaesthetic agents preferred are thiopental sodium, propofol or inhalational agents as sevoflurane. Tracheal intubation may be facilitated by succinylcholine but due to fasciculation cortisol secretion may increase. So intubation was done by rocuronium. Anaesthesia is maintained by midazolam, fentanyl , sevoflurane and N_2O. Adequate muscle relaxation is necessary for good exposure of surgical field. Any of nondepolarizing muscle relaxants may be employed. Trans-abdominal resection is recommended in children, sodium nitropruside, phentolamine can be used for BP fluctuation during adrenal handling or Metoprolol I/V bolus to control tachycardia. In this case, meticulous surgical handling did not cause much fluctuation. Steriod cover is mandatory for

patients undergoing bilateral adrenalectomy. Our patient was discharged on oral prednisolone and fludrocortisone. After one week of surgical intervention this patient body weight reduced and patient able to walk.

Conclusion

Diagnosis and management of cushing's syndrome in paediatric patients is as challenging as in adult. Early involvement of anaesthesiologist is essential with better understanding of pathophysiology of adrenal hyperplasia, necessary monitoring and diagnostic modalities, availability of rapid acting drugs which can alter BP. Sophisticated and skilled anaesthesia and surgery all of these have given success to management of bilateral adrenalectomy in a 4½ years old child with hypertension and cushinoid features.

References:

1. Wealtheril D, Spenece AA. Anaesthesia and disorders of the adrenal cortex, Br J Anaesth 1984; 56: 741-7.
2. Robert Hardin, William, Daniel W. Fortser, Henry M Kronanbug. William's textbook of endocrinology. Paul M, Stewart. The adrenal cortex. 10th edition, Saunders Company, New York, 2007; 4: 514.
3. Orth DN, Cushing's syndrome, N Engl J Med 1995; 332: 791-803.
4. Nicman LK, Ilias I. Evaluation and treatment of cushing's syndrome. Journal of American Medicine 2005; 118(12): 1340-1346.
5. Govindan R, Page N, Morgensztern D et al. Changing epidemiology of small cell lung cancer in the United States over the last 30 years analysis of the surveillance, epidemiologic, and end results database. Journal of clinical oncology 2006; 24: 4539-4544.
6. Bruce HK, Telender LR, Heerden JA et al. Pheochromocytoma in paediatric age group. Current status. Journal Paed Surg 1983; 18(6): 879-883.
7. Stringe G, Ein SH, Creighton R. Pheochromocytoma in children-An update. J Paed Surg 1980; 15: 496-500.
8. Gupta Poonam, Wason G.A.R. Anaesthetic management of bilateral pheochromocytoma ina child- A case report. Indian J. Anaeth 2005; 49(1): 54-55.
9. Kurt D, Alewman, Todd Pnsky. Diagnosis and management of endocrine tumours causing hypertension in children. Annals of New York Academy of Science 2007; 970: 155-158.
10. Stefanic Hahner, Bruno Allolio. Management of Adrenal insufficiency in different clinical settings. Expert opinion on pharmacotherapy 2005; 6(14): 2407-2417.
11. Brown R, Brunell Jr. Anaesthesia for pheochromocytoma. Roberts C Prys and Prown R. Brunell Jr. editors. International practice of anaesthesia. Butterwort Heinemarnn, New York, 1996; 83: 7.

Original Article

Organisms associated with ventilator associated pneumonia (VAP) in intensive care units (ICU)

Farhana Selina[1*], Khandaker Abu Talha[2], Amirul Islam[3], Zahidul Hasan[4], Masum Hyder[5], S. Selvapandian[6]

[1]Department of Anaesthesia, Square Hospital Ltd. [2] Associate Consultant, Neurosurgery, Square Hospital Ltd.[3]Department of Anaesthesia, Analgesia & Intensive Care Medicine, BSMMU.[4] Consultant, Microbiology, Square Hospital Ltd.[5] RMO, Neurosurgery, Square Hospital Ltd. [6]Consultant, Neurosurgery, Square Hospitals Ltd.

***Corresponding author**: E-mail :< katalha@yahoo.com>*

Abstract

Background: *Organisms associated with ventilator associated pneumonia (VAP) in intensive care units (ICU).*

***Aims and objectives**: To identify the organisms associated with ventilator associated pneumonia and to compare our study with other international studies.*

Methods: *This observational study on VAP was conducted in different ICU of Square Hospital Limited during the two years. Any ventilated patient who developed fever or respiratory distress was clinically assessed by attending physician. This includes time of developing fever after intubation, physical examination findings (auscultation finding, pattern of temperature) and X-ray findings. On the day of onset of fever or respiratory distress, tracheal aspiration was done and the aspirated fluid was sent to microbiology lab. for culture and sensitivity test. If patient was designated to be suffering from VAP all relevant information was documented in a structured questionnaire.*

Results: *In our study we found 15 organisms responsible for VAP. Among these common organisms were pseudomonus(35%), acinetobacter(29%), klebsiella(16%), MSSA (10%), MRSA(9).Total number of cases was 79. Among these 21 patients expired which was 26.5% of the whole series. Total 534 patients died in this hospital during this 2 years. Death for VAP was 4% of the total death of the hospital.*

Conclusion: *Pseudomonas was the most commonest among identified organisms with ventilator associated pneumonia and medicine department having maximum number of VAP patients. To compare our results with those of international studies we found similarity in organisms, primary diagnosis and mortality.*

Key words: *ventilator associated pneumonia, organism and intensive care unit.*

(Journal of BSA, 2009; 22(2): 72-77)

Introduction:

Ventilator-associated pneumonia (VAP) is the most common nosocomial infection in the ICU and contributes disproportionately to both poor outcomes and the high cost of care in critically ill patients[1]. Since the initial 1996 American Thoracic Society (ATS) guideline on nosocomial pneumonia, a number of new developments have appeared, mandating a new evidence-based guideline for hospital-acquired pneumonia (HAP) including healthcare-associated pneumonia (HCAP) and ventilator-associated pneumonia (VAP).

Ventilator-associated pneumonia is a common and highly morbid condition in critically ill patients[2]. Epidemiologic investigations have shown cumulative incidence rates of 10% to 25%[3], crude mortality rates of 10% to 40%[4] and attributable mortality rates of 5% to 27%[5]. Hospital length of stay and cost are both increased in patients who develop ventilator-associated pneumonia[6].

The predominant organisms responsible for infection are *Staphylococcus aureus Pseudomonas aeruginosa* and *Enterobacteriaceae* but etiologic agents widely differ according to the population of patients in an intensive care unit duration of hospital stay and prior antimicrobial therapy. Because appropriate antimicrobial treatment of patients with VAP significantly improves outcome more rapid

identification of infected patients and accurate selection of antimicrobial agents represent important clinical goals[7].

Organisms causing ventilator-associated pneumonia generally fall into two groups: those causing early-onset ventilator-associated pneumonia (<4 days of mechanical ventilation) and those causing late-onset ventilator-associated pneumonia (>4 days of mechanical ventilation)[3]. Early-onset organisms are typically antibiotic-susceptible community-acquired bacteria, while late-onset organisms are commonly antibiotic-resistant nosocomial organisms. Colonization of the oropharynx and the stomach with potentially pathogenic organisms precedes the development of ventilator-associated pneumonia in most patients. The pathogenesis of ventilator-associated pneumonia probably involves microaspiration of oropharyngeal or gastric secretions contaminated with these organisms[4].

The most widely studied preventive strategies have focused on the prevention of oropharyngeal or gastric colonization and the prevention of aspiration of contaminated oropharyngeal or gastric secretions[8]. This evidence-based systematic review aims to identify interventions for the prevention of ventilator-associated pneumonia, critically evaluate their efficacy and adverse effects and recommend an approach to their use.

The diagnosis of VAP is usually based on three components: systemic signs of infection, new or worsening infiltrates seen on the chest roentgenogram and bacteriologic evidence of pulmonary parenchymal infection[9]. The systemic signs of infection such as fever, tachycardia and leukocytosis are nonspecific findings and can be caused by any condition that releases cytokines[10]. In trauma and other surgical patients, fever and leukocytosis should prompt the physician to suspect infection but during the early post traumatic or postoperative period (i.e., during the first 72 hours) these findings usually are not conclusive. However, later, fever and leukocytosis are more likely to be caused by infection but even then other events associated with an inflammatory response (e.g., devascularized tissue, open wounds, pulmonary edema and/or infarction) can be responsible for these findings.

Aim of our study was to identify the organisms associated with ventilator associated pneumonia (VAP) and to compare our study with other international studies.

Method:

This observational study on ventilator associated pneumonia (VAP) was conducted at Square Hospitals Ltd. Dhaka during April 2007 to March 2009. The aim of the study was to determine the organisms associated with the disease.

Any ventilated patient who developed clinical pneumonia along with culture positive aspirated tracheal fluid constituted VAP. Clinical pneumonia was defined as inflammation of one or both lungs with consolidation which is frequently but not always due to infection. The infection may be bacterial, viral, fungal or parasitic. Symptoms may include fever, chills, cough with sputum production, chest pain, and shortness of breath.

Patients who developed other infections along with VAP and patients who underwent tracheostomy were excluded from the study.

Any ventilated patient who developed fever or respiratory distress was clinically assessed by attending physician. This includes time of developing fever after intubation, Physical examination findings (auscultation findings, pattern of temperature) and chest X-ray findings. On the day of onset of fever or respiratory distress tracheal aspiration was done and the aspirated fluid was sent to microbiology lab for culture and sensitivity test. If bacteria were isolated in culture, the patient was evaluated to asses whether the patient fell under case definition of VAP. If patient was designated to be suffering from VAP all relevant information was documented in a structured questionnaire.

At the end of the data collection period all the questionnaires were complied and a master sheet was made.

Results:

This is an observational analytical cross-sectional study of 'Organisms responsible for ventilator associated pneumonia (VAP) in different department of Hospital'. All the patients diagnosed as VAP was included in this study. Total number of cases was 79. Among these patients 21 patients died which was 26.5 % of the whole series. Study period was 2

years. Total 534 patients died in this hospital during this 2 years period. Death for VAP was 4 % of the total death of the hospital.

Among the 79 VAP patients 52 patients (66%) was male and 27 patients was female. Male : Female ratio was 1.9 : 1.

Table-I

Age distribution of VAP patients (n=79).

Age frequency (year)	No of cases	Percentage	Mean age (year)
<= 30	08	10.13 %	
31-40	05	06.33 %	
41-50	10	12.67 %	60.17
51-60	10	12.67 %	
61-70	18	22.78 %	
>70	28	35.44 %	

Shows the distribution of VAP patients according to their age frequency. Highest number of patients (35.44%) was in more than 70 year age group. Second highest was 61 to 70 years age group. Patients of 41-50 years and 51-60 years age group were equal in number (10 patients). We had 8 patients (10.13%) in below 30 years age group and only 5 patients (6.33%) in 31-40 years age group.

Table-II

Age distribution of expired VAP patients (n=21)

Age frequency (year)	No of cases	Percentage
<= 30	1	4.7 %
31-40	2	9.5 %
41-50	1	4.7 %
51-60	2	9.5 %
61-70	6	28.5 %
>70	9	43 %

This demonstrates the distribution of age frequency among the patients who died of VAP. Age of highest number of patients (43%) was more than 70 years. Six patients (28.5%) were in 61-70 year age group. Number of expired patients was 2 in 31-40 and 51-60 year age group. Where as that was only 1 in number (4.7%) in below 30 year and 41-50 year age group.

Table -III

Distribution of organisms among VAP cases

Sl.	Organism	Number of cases	Percentage
1.	Pseudomonas spp.	28	35 %
2.	Acinetobacter spp.	23	29 %
3.	Klebsiella spp.	13	16.3 %
4.	MSSA	08	10 %
5.	MRSA	07	9 %
6.	Group D non enterococci	05	6 %
7.	Candida	05	6 %
8.	CONS	05	6 %
9.	E. coli	04	5 %
10.	Sternotrophomonas	04	5 %
11.	N. meningitidis	03	4 %
12.	Sterptococcus pneumoniae	02	2.5 %
13.	Entero faecalis	01	1.3 %
14.	Proteus spp.	01	1.3 %
15.	Alpha hemolytic streptococci	01	1.3 %

In our study we found 15 organisms responsible for VAP. Single organism was isolated in 54 cases. Other 25 patients had more than 1 organism. Table-III has tabulated the list of isolated organisms in VAP cases. Pseudomonas spp. was responsible for more than one third cases (28 cases). Acinatobacter spp. was isolated in 23 cases (29%). We found Klebsiella spp. in 13 cases (16.3%). Methicillin Sensitive Staphylococcus Aureas (MSSA) and Methicillin Resistant Staphylococcus Aureas (MRSA) were found respectively in 08 and 07 cases. Group D non enterococci, Candida and Coagulase Negative Staphylococcus species (CONS) were present in 5 cases (6%). E.coli and Sternotrophomonas were isolated in 4 cases. Streptococcus pneumoniae was responsible for 2 cases. Other 3 organisms which were present in 1 case respectively were Entero faecalis, Proteus spp. and Alpha hemolytic streptococci.

Table – IV

Distribution of organisms among expired VAP cases

Sl	Name of the organism	No. of cases	Percentage
1	Pseudomonus spp.	10	48 %
2	Klebsiella	05	24 %
3	Acinetobacter spp.	04	19 %
4	Candida	03	14 %
5	E. coli	03	14 %
6	MRSA	02	9.5 %
7	MSSA	1	5 %
8	CONS	1	5 %
9	N. meningitidis	1	5 %
10	Sternotrophomonas	1	5 %

This shows the distribution of organisms among expired VAP cases. In near about half (48%) of the cases Pseudomonas spp. was responsible for VAP. Klebsiella was isolated in approximately quarter (24%) of the patients. Acinatobacter spp. was found in 19% (04 cases) of cases. Number of affected cases by Candida and E.coli was three (14%) respectively. Methicillin Resistant Staphylococcus Aureas (MRSA) was responsible for 2 cases. Number of patient affected by Methicillin Sensitive Staphylococcus Aureas (MSSA), Coagulase Negative Staphylococcus species (CONS), N. meningitidis and Sternotrophomonas was 1 for each organism.

This elaborates the statistics of VAP patients among all departments of hospital. Highest number of VAP patient was from medicine department (27 patients), followed by Neurosurgery (19 patients), Surgery (08 patients) and Cardiology (08 patients). Nephrology had 05 patients. Neurology and Oncology had 3 patients from each department. Number of VAP patients from CT surgery and Gastroenterology was respectively 2 in number. Orthopedics department had only 1 patient. Highest number of patients expired in medicine department (06 cases) followed by neurosurgery (3 cases) and cardiology (3 cases). Two patients expired respectively from the department of nephrology, CT surgery, oncology and gastroenterology. Only 1 patient expired from surgery department. Other departments like neurology and orthopedics didn't have any mortality. Time duration of developing VAP after intubation was 7 to 9.7 days for all cases. Mean was 8.3 days for all cases. Common organisms were acinatobacter, pseudomonus spp., staph aureas, klebsiella and MRSA.

Table-V

Statistics according to different departments.

Department	No. of cases	Mean age (year)	No of death	Percentage of death	Mean time of developing VAP after intubation (day)	Commom two organisms
Medicine	27	59	06	22 %	9.7	Acinetobacter Pseudomonus
Neurosurgery	19	55	03	16 %	7.0	Pseudomonus Staph aureas
Surgery	08	55	01	13 %	9.3	Pseudomonus Acinetobacter
Cardiology	08	74	03	38 %	8.5	Klebsiella Pseudomonus
Nephrology	05	79	02	40 %	8.6	MRSA Acinetobacter
Neurology	03	71	00	00 %	7	Acinetobacter MRSA
CT surgery	02	47	02	100 %	9	Pseudomonus Acinetobacter
Oncology	03	62	02	67 %	8	Pseudomonus Candida
Gastroenterology	02	68	02	100 %	8	Acinetobacter Pseudomonus
Orthopedics	01	30	00	00 %	7	Pseudomonus Klebsiella
Clinical Hematology	01	32	00	00 %	9	Acinetobacter
					Mean 8.3	

Discussion:

We have performed our study on "Organisms associated with ventilator associated pneumonia (VAP) in intensive care unit (ICU)". This was an observational analytic cross section study. Total number of patients was 79. Main objective of our study was to identify the organisms associated with VAP (Ventilator Associated Pneumonia) in intensive care unit (ICU). All patients were diagnosed on the basis of their clinical features, radiological findings and tracheal swab culture sensitivity. Age, sex, time of onset of VAP after intubation, primary diagnosis of patient, isolated organisms and clinical status at the time of discharge was documented as variable in data collection sheet. After collection of data we have tabulated the result and compared with that of international studies.

According to the study of Kollef et al 15.5 % of their ICU patient had developed VAP[11]. Common primary diagnosis of VAP was cardiothoracic (21.6%) and medical disease (9.3%). Mortality rate was 37.2% in their series. In our study we found primary diagnosis of cardiac origin was in 12.6% cases and that of medicine origin was in 34% cases. Mortality was 26.5% in our series.

Kimberly et al. found mortality of VAP in between 20% - 50% in their study[12]. In our study mortality rate was 26.5%.

Marin et al. had their study on VAP. According to their study primary diagnosis of VAP was post surgery (15.6%), neurologic disease (13.3%), sepsis (13.1%) and cardiac (10.8%) [13]. In our study primary diagnosis was surgical in 10%, neurological in 28% and cardiac in 12.6%. According to their study duration of developing VAP after intubation was 7.3 days. In our study mean was 8.3 days. Marin et al. found common responsible organisms MRSA (14.8%), *Staphylococcus aureas* (14.8%), *Pseudomonas aeruginosa* (14.3%) and other *Staphylococcus* species (8.8%) in their study. In our study we found *Pseudomonas spp.* commonest (35%) followed by A*cinatobacter spp.* (29%) and *Klebsiella* (16.3%). We found only 9% cases caused disease by MRSA. Overall mortality was 25.1% in their series which was 26.5% in our series.

According to the study of Chastre J. et al. mortality was 24% to 50% for VAP in their series. Isolated organisms mostly were *Staphylococcus aureas*, *Pseudomonas aeruginosa* and *Enterobacteriaceae*[14]. We found mortality 26.5% in our study. *Staphylococcus aureas* (10%) and *Pseudomonas aeruginosa* (35%) were also common in our series.

Donald E et al. performed their epidemiological study on VAP. According to their study rate of VAP increases 6 to 21 fold for intubated patients in ICU[15]. They found the rate of the occurrence of VAP was higher in surgical patients then the medical patients. In our study percentage of medical cases (34%) was more than that of surgical cases (10%).

According to the study of Bonten et al. primary cause of VAP was COPD, ARDS, head injury and trauma in most of the patients[16]. In our study we found primary diagnosis was neurosurgical, medical and surgical in most of the patients. Bonten et al found that maximum number of their patient developed VAP after 5 days of intubation. In our study mean time was 8.3 days to develop VAP.

Shaw et al. found *Staphylococcus aureas* as commonest organism in VAP cases of their series[17]. Other common organisms were *Pseudomonas aeruginosa* and *Acinatobacter baumanii*. In our study commonest organism was *Pseudomonas aeruginosa* (35%) followed by A*cinetobacter* (29%) and *Klebsiella* (16.3%).

Mortality of VAP patients was 37% in the study of Rakshit et al. In their study male was 56.9% and female was 43.1%. We found mortality 26.5% in our study[18]. Male was 55% in our study where as female was 45%. Rakshit et al. found commonest organism *Pseudomonas aeruginosa* in their study followed by *Klebsiella pneumonae*. We also found *Pseudomonas spp.* commonest organism in our series.

Conclusion:

Pseudomonas spp was the commonest among identified organisms with ventilator associated pneumonia and medicine department having maximum number of VAP patients and compared our results with those of international studies we found similarity in organisms, primary diagnosis and mortality.

References:

1. Andrew F, Shorr MD. MPH. FCCP and Marin H. Kollef, MD Ventilator-associated pneumonia insights from recent clinical trials . Chest, 2005;128:583 – 591.

2. Ibrahim EH, Tracy L, Hill C, Fraser VJ, Kollef MH. The occurrence of ventilator-associated pneumonia in a community hospital: risk factors and clinical outcomes Chest. 2001;120:555-61.

3. George DL, Falk PS, Wunderink RG, Leeper KV Jr, Meduri GU, Steere EL, et al. Epidemiology of ventilator-acquired pneumonia based on protected bronchoscopic sampling Am J Respir Crit Care Med. 1998;158:1839-47.

4. Craven DE, Steger KA. Nosocomial pneumonia in mechanically ventilated adult patients: epidemiology and prevention Semin Respir Infect. 1996;11:32-53.

5. Fagon JY, Chastre J, Hance AJ, Montravers P, Novara A, Gibert C. Nosocomial pneumonia in ventilated patients: a cohort study evaluating attributable mortality and hospital stay Am J Med. 1993;94:281-8.

6. Heyland DK, Cook DJ, Griffith L, Keenan SP, Brun-Buisson C. The attributable morbidity and mortality of ventilator-associated pneumonia in the critically ill patient. The Canadian Critical Trials Group Am J Respir Crit Care Med. 1999;159:1249-56.

7. Jean Chastre and Jean-Yves Fagon. Ventilator-associated pneumonia : Am. J. Respir. Crit. Care Med.. 2002; 165(7) :867-903.

8. Livingston DH. Prevention of ventilator-associated pneumonia Am J Surg. 2000;179:12-17.

9. Andrews CP, Coalson JJ, Smith JD, Johanson WG. Diagnosis of nosocomial bacterial pneumonia in acute, diffuse lung injury. *Chest* 1981; 80: 254-258

10. Ayala A, Perrin MM, Meldrum DR, Ertel W, Chaudry IH. Hemorrhage induces an increase in serum TNF which is not associated with elevated levels of endotoxin. *Cytokine* 1990; 2: 170-174

11. Kollef MH . Ventilator-associated pneumonia. A multivariate analysis. JAMA. 1993; 270(16):1965-70

12. Kimberly A. Davis, MD, FACS. Ventilator-Associated Pneumonia: A Review. Journal of Intensive Care Medicine, 2006;21(4): 211-226.

13. Marin H. Kollef, MD, FCCP; Lee E. Morrow, MD, FCCP; Michael S. Niederman, MD, FCCP; Kenneth V. Leeper, MD, FCCP; Antonio Anzueto, MD; Lisa Benz-Scott, BS and Frank J. Rodino, MS. Clinical Characteristics and Treatment Patterns Among Patients With Ventilator-Associated Pneumonia.. *Chest*. 2006;129:1210-1218.

14. Chastre J, Fagon JY. Ventilator-associated pneumonia. Am J Respir Crit Care Med. 2002 ;165(7):867-903.

15. Donald E. Craven, MD, FCCP. Epidemiology of Ventilator-Associated Pneumonia. *Chest*. 2000; 117: 186-187.

16. Bonten M,Kollef MH, Hall JB.Risk Factors for Ventilator-Associated Pneumonia:From Epidemiology to Patient Management.Clinical Infectious Diseases.2004;38:1141–9.

17. Shaw, M. J. Ventilator-associated pneumonia. Current Opinion in Pulmonary Medicine, 2005; 11(3); 236-241.

18. Rakshit Panwar, Nagar Vidya S, Deshpande Alaka K. Incidence, clinical outcome, and risk stratification of ventilator-associated pneumonia-a prospective cohort study. Indian Journal of Critical Care Medicine. 2005;9(4): 211-216.

Original Article

Effects of hypertonic saline preloading in sub arachnoid blockade for transurethral resection of prostate - A comparative study

Amirul Islam[1*], Md. Rafayet Ullah Siddique[2], Md. Mustafa Kamal[2], Debabrata Banik[3], AKM Akhtaruzzaman[3], Md. Abdul Hye[3]

[1] Medical Officer, Department of Anaesthesia, Intensive Care &Pain Medicine BSMMU,Shahbag, Dhaka-1000. [2]Assistant Professor, Department of Anaesthesia, Intensive Care &Pain Medicine BSMMU,Shahbag, Dhaka-1000. [3]Assoc. Professor, Department of Anaesthesia, Intensive Care &Pain Medicine BSMMU,Shahbag, Dhaka-1000.

***Corresponding author:** *Email*: <dramirul68@yahoo.com>

Abstract:

Background*: Hypertonic solution is used to combat hypotension in sub-arachnoid block during trans urethral resection of prostate.*

Aims and objectives*: To compare the effect of 3% sodium chloride solution with that of 0.9% sodium chloride solution, to combat sub-arachnoid block induced hypotension in trans urethral resection of prostate.*

Methods*: A total number of sixty patients ASA grade I & II were selected randomly in two groups , thirty in each group. Group A received 15ml/kg of 0.9% NaCl solution and group B 4ml/kg of 3% NaCl solution as a preload. Sub arachnoid block performed at the $L_{3/4}$ interspace in the sitting position. Heart rate, mean arterial pressure, amount of ephedrine, amount of used additional I/V normal saline, serum electrolytes and level of sensory block were observed.*

Results*: Mean arterial pressure was differed significantly at late hours ie, 50min, 60min(P<0.001). Incidence of hypotension was 43% in group A, 16% in group B and was significant(p<0.05). Less additional I/V fluid was required in groupB and difference was significant(P<0.05). Low doses of ephedrine was needed in groupB and was highly significant(P<0.001).*

Conclusion*: Preloading of hypertonic solution is superior to isotonic solution in trans urethral resection of prostate under sub arachnoid block.*

Key Words*: sub arachnoid block, hypertonic saline, trans urethral resection of prostate.*

(Journal of BSA, 2009; 22(2): 66-71)

Introduction:

Now a day's most of the urological operations including transurethral resection of prostate (TURP) are performed under sub-arachnoid block (SAB). SAB is gaining increase popularity and replacing general anaesthesia gradually. Advantages are virtual avoidance of risks of general anaesthesia, for example, gastric aspiration and difficult tracheal intubation and also TURP syndrome can not be diagnosed early, whereas SAB is very useful to diagnose early and potentially prevent the above mentioned problems. However, hypotension after SAB results from functional sympathetic devervation, not only of the arterial and arteriolar circulation but also of the large veins, venules. Venodilation can increase significantly venous capacitance with a consequent decrease in venous return and cardiac output. The rational of prehydration is to expand the plasma volume. However isotonic crystalloid solutions, in volume expansion commonly used, may not be effective, as about 70% of the solution diffusion occurs extra vascularly into the interstitial space. In contrast hypertonic saline is more effective as it can remain in the intravascular space, induce instantaneous mobilization of endogenous fluid also the osmotic gradient from the intracellular to the extracellular space[1].

Circulatory preloading is one of the mainstays in the preventing of the hypotension and a variety of crystalloid and colloid solutions have been used for this purpose[2]. Prophylaxis colloid fluid is not popular because it may produce anaphylactic reaction and interferes with blood grouping and cross matching and also interferes of blood coagulation[3].Despite this, a significant incidence of hypotension which necessitates the use of a vasopressor is still reported[4].

The usual practice is to use crystalloid solutions, these are mainly isotonic but its half life is short. So to require three to four times more than hypertonic crystalloid solutions, only 25% of fluid remains intravascularly.

Hypertonic saline (3% NaCl) has an osmolality of 1026mOsmol/L that about three times more of plasma[5]. The fluid shift and osmolar changes that occur with its infusion can be predicted. The hypertonic solution will also draw water out of the cells increasing extracellular fluid volume. But it has some warning that rapid correction of chronic hyponatremia and hypotension can be fatal or cause severe neurological injury[6]. So patients selection with serum elctrolytes will be within normal ranges and no chronic hypotension. Hypertonic solution(3%NaCl) is better in some points of views-less incidence of hypotension, it prevents hyponatremia during TURP because hyponatremia contributes TURP syndrome, it prevents water intoxication[7].

Method:

60 patients of hypertrophy of prostate undergoing TURP were taken in this study. Every patient included in the study, allowed choosing a card. According to card number, patients grouped. Each group received either 0.9% NaCl solution or 3% NaCl solution for preloading through 18G intravenous cannula placed in the forearm.

Subarachnoid block performed at the level of L_{3-4} inter-space with the patient in the sitting position. All subjects received 10mg of 0.5% hyperbaric bupivacaine delivered through a 25 gauge Quincke needle to achieve uniform performance. All patients were getting O_2 through nasal prong at the rate of 2L/min throughout procedure. Blood pressure was measured with an automated blood pressure device and S_PO_2 continuously displayed on Datex-Ohmeda machine by using S_PO_2 probe and reading recorded in time to time.

Hypotension is defined as a systolic blood pressure less than 20% of the base line blood pressure. Base line blood pressure was determined by calculating the mean of three blood pressure measurement in the pre operative period before fluid loading commenced. Hypotension treated with a bolus of 5mg ephedrine intravenously and rapid infusion of running fluid to both groups. Variables were recorded including the maximum height of block as assessment by pin prick 15 minutes following subarachnoid block, other drugs used and their doses, and the amount of intravenous fluid was given throughout the procedure.

Study parameters:

Mean arterial Pressure, mean heart rate, amount of ephedrine used, Amount of used additional intravenous (I/V) fluid, Serum electrolytes were taken during procedure.

Data collected in a specially designed 'Data sheet'. It was collated and analyzed for statistical significance by student's t-test and chi square (χ^2) as appropriate. Values regarded as significant if $P<0.05$, (CL95%).

Results:

Patient's demographics were similar and comparable in both groups and differences were statistically not significant.

Table-I

Demographic data

Variable	Group A n=30	Group B n=30	p-value
Age (year)	65.94±1.90	66.74±1.76	0.759
Weight (kg)	57.41±1.08	59.27±1.73	0.362
Height (cm)	166.24±0.94	167.67±1.18	0.347

Data are presented as mean ± SEM. Unpaired t-test was performed. The studied groups became statistically matched for age (p=0.759), weight (p=0.362), and height (p=0.347). Values are regarded as significant if $p<0.05$.

Table II

Changes of heart rate

Groups	Before Preload	Before SAB	After SAB	5min	10mins	15mins	30mins	40mins	50mins	60mins
GroupA	78.27± 1.64	81.37± 1.82	77.84± 3.15	76.77± 2.23	76.66± 2.34	74± 2.58	72.77± 2.85	72.11± 2.51	71.14± 3.34	73.41± 2.43
GroupB	81.24± 1.8	82.51± 1.49	80.81± 1.8	79.14± 1.93	75.07± 2.1	69.37± 2.02	67.51± 1.94	68.74± 1.95	68.21± 2.14	69.67± 2
P value	0.234	0.952	0.415	0.424	0.614	0.161	0.130	0.283	0.466	0.241

Data are presented as mean±SEM . Unpaired t-test was performed. There was no significant difference between the two groups.

Table -III

Changes of mean arterial pressure.

Groups	Base line	After pre-load	Just after block	3mins	10mins	20mins	30mins	40mins	50mins	60mins
Group-A	101± 1.87	02.4± 11.93	100± 1.80	92.17± 1.37	85.24± 2.07	81.17± 2.38	76.67± 3.15	77± 2.91	75.24± 2.59	74.74± 2.61
Group-B	101.7± 2.02	104.1± 1.99	97.67± 2.27	92.3± 2.37	88 ± 2.25	86.24± 2.60	81.77± 2.63	83.87± 2.58	86.24± 2.32	86.61± 2.29
P value	0.86	0.559	0.36	0.81	0.368	0.111	0.218	0.081	0.0006	0.0009

Data are presented as mean±SEM. Unpaired t-test was performed. Values are regarded as insignificant that was up to 40 minutes from onset of sub arachnoid block. But at 50 mins and 60 mins after SAB mean arterial pressure difference were highly significant ie, p =0.0006 and p=0.0009 respectively.

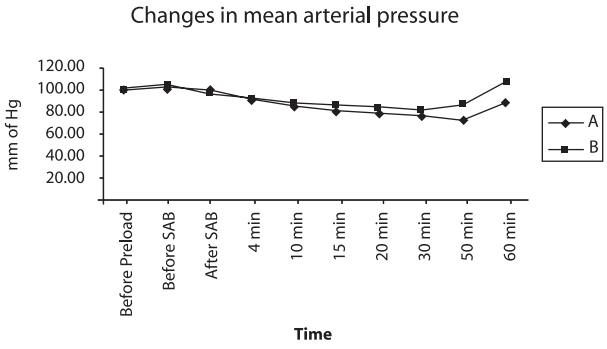

Fig.-1: *Changes in mean arterial pressure.*

Table-IV

Incidence of hypotension.

Groups	Hypotension	P value
Group A (n=30)	13 (43.33%)	0.042
Group B (n=30)	5 (16.66%)	

Data are presented as mean±SEM. Unpaired t-test was performed. Overall incidence of hypotension in group A was 13 (43.33%) and in group B was 5 (16.66%) after SAB. The incidence of hypotension in two groups were found significant.

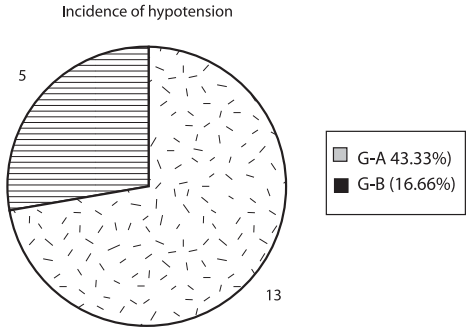

Fig.-2: *Incidence of hypotension .*

Table -V
Treatment of hypotension

Group	Additional I/V fluid (ml)	Ephedrine	
		Number of pt.	mean dose (mg)
Group A	213.33±	10	2.34±
(n=30)	46.92	(33.34%)	0.37
Group B	93.34±	3	0.833±
(n=30)	35.54	(10%)	0.49
P value	0.02		0.001

Data are presented as mean±SEM. Unpaired t-test was performed. Treatment of hypotension with I/V normal saline and ephedrine. It was found that use of additional I/V normal saline significantly differed between two groups and also the use of ephedrine was significant. Mean dose of ephedrine used in group B was significantly less then group A.

Table- VI
Maximum level of analgesia

Groups	T_4	T_6	T_8	T_{10}	P-value
Group-A n=30	1	3	14	12	0.467
Group-B-n=30	0	3	15	12	

Data are presented in number, sensory block achieved. Chi square test was performed.

Maximum level of sensory analgesia included in this range from $T_4 - T_{10}$. Desired level of sensory block is the T8 in both groups.One patient had sensory level at T_4, six patients had sensory level at T_6, twenty nine patients had sensory level at T_8 and twenty four patients had sensory level at T_{10}. This is not significant difference in achieving sensory blocked in both groups.

Table- VII
Serum electrolytes

	Groups	Preoperativem Eq/L	Postoperativem Eq/L	Significant level
Na+	A	139.33± 0.474	138.3± 0.58	0.062
	B	139.39± 4.00	141.19± 0.39	0.001
K+	A	4.31± 0.099	4.53± 0.08	0.104
	B	4.46± 0.087	4.64± 0.08	0.241
Cl-	A	103.04± 2.15	102.06± 3.16	0.067
	B	104.62± 3.11	105.15± 2.67	0.042

Data are presented as mean ± SEM; Analysis was done by student's t-test. Serum electrolytes of two groups were done preoperative and postoperative respectively. All compare were insignificant except sodium ion in group B (p 0.001)& chloride ion in group B (p 0.042)which were significant.

Discussion:

Arterial hypotension after sub aracnhoid block (SAB) is more or less common. Now a day's most of the urological operations including trans urethral resection of prostate (TURP) are performed under sub arachnoid block(SAB). SAB is also useful in trans urethral resection of prostate (TURP) because TURP syndrome can be early diagnosed by this method[5] . Spinal anaesthesia induced hypotension results from functional sympathetic denervation not only of the arterial and arteriolar circulation, but also of the large veins & venules. Venodilation can increase significantly venous capacitance with a consequent decrease in venous return and cardiac output. The rationale use of pre-hydration is to expand the plasma volume. However, isotonic crystalloid solution is the fluid commonly used to expand the plasma volume, may not be effective as about 75% of the solution diffuses extravascularly into the interstitial space within short time. In contrast, hypertonic saline is more effective as it can remain in the vascular system more time than crystalloid solution, it can induce instantaneous mobilization of endogenous fluid along the osmotic gradient from the intracellular to the extracellular space. In addition, hypertonic saline (3%NaCl) may result in direct myocardial stimulation and venoconstriction[1].

Small volume resuscitation (3-5ml/kg) of hypertonic saline is effective for hemorrhagic shock[5]. Resuscitation using hypertonic saline (3-7.5% NaCl) association of rapid improvement in organ perfusion in anaesthetized person subjected to burn injury. In comparison to isotonic saline (0.9% NaCl) greater increase in blood flows to the heart, kidney, liver and testis observed with hypertonic saline. The results suggested that significant improvement in blood flow distribution can achieved hypertonic saline by using one fourth volume in that of normal saline. The apparent improvement of the left ventricular systolic function in response to hypertonic saline is caused mainly by the combined effect of increased left ventricular preload and reduced left ventricular after load[10].

Although hemodynamic effects induced by the spinal anaesthesia are usually tolerated well in healthy young patient, prevention of hypotension is commonly achieved using fluid preloading. Anaphylactic reaction may occur after the administration of colloids, this risk is rare, but may not be acceptable for prophylaxis. Ringer's lactate solution is the solution, used most commonly for fluid preloading even though its Na+ concentration (130mmol/l) is less than that in 0.9% NaCl solution (155mmol/l)[8] .

Hypertonic saline (3%NaCl) has an osmolality of 1026mosmol/L that about three times of plasma.The Na^+ content of this fluid limits the distribution of the fluid to the extra cellular fluid. The hypertonic saline will also draw water out of the cells decreasing intracellular fluid volume. Glycine solution is used randomly as an irrigation fluid. It has an osmolarity of 200mosmol/l. Glycine is metabolized in the liver to ammonia may lead to visual impairment. High ammonia levels may result in neurological distrubance[9]. There is a more chance of water intoxication in TURP because to use of isotonic solution in excess amount to prevent hypotension and also absorption of irrigation solution due to prolong use. Water intoxication (restlessness, frothing, retching, tremor and twitching of muscles) was described by another auther[7]. Patient undergoing TURP may develop this syndrome from dilutional hyponatremia secondary to systemic absorption of the irrigation fluid, the degree of bleeding type, volume and pressure of the irigation fluid and also prolong duration of operation . So, to prevent TURP syndrome, some strategies should be maintained-i.e.-hydrostatic bag pressure will be less than 60cm of H_2O, Short operating time i.e., less than 90 min, Minimize intra vesicle pressure by frequent emptying, Adequate hemostasis, Limit the position of the irrigarion bag to maximum 60cm above the surgical field, Maintain adequate blood pressure and therefore normal periprostatic venous pressure in order to avoid increased absorption through open venous sinuses. Necessary to observe neurological status (possible in patient with regional anaesthesia), temperature and laboratory measurement frequently.

Hypo-osmolality and hyponatremia appear to be the principal culprit's contributing to the neurological changes seen in TURP syndrome[9]. In our study, we have not found such type of emergency crisis, it may not be due to such preventive measures that was against contributing factors.

Kien et.al.[1] also suggested that hypertonic saline required fifth times less than isotonic saline for the initial treatment of burn shock.

We found that 4ml/kg of 3%NaCl solution was as effective as 15ml/kg of NaCl solution in the prophylaxis of hemodynamic changes in ASA II patient in TURP under spinal anaesthesia.

Mouren S.et al[11], found that hypertonic saline increase myocardial contraction and vasoconstriction. This effect of hypertonic solution probably explains improved cardiac output. Because of plasma osmolarity is the driving force for volume distribution. Our observation found that 10% ephedrine needed in group B and 33% in group A, to keep Mean Arterial Pressure (MAP) greater than the acceptable limit. Ueyama H[12] found that initial fluid administration might provide a protection against undesired cardiovascular side effects but not all, our studies had shown this the augmentation of blood volume with initial fluid administration, regardless of the fluid used, must be large enough to result in a significant increase in cardiac output for effective prevention of hypotension.

In our observation, mean arterial blood pressure changes was insignificant in two groups except 50 min and 60 min after SAB(Table-III) which was very significant. This supports the study by Arndt J.O[13], where he found that administration of preloading fluids reduces the incidence of early cardiac events, but a vasopressor was needed for prevention of the late events. This is probably due to shifting of I/V fluids to the extra-vascular spaces.

Hypertonic saline administration causes a rapid increase in serum sodium concentration and osmolarity related to the sodium dose. This has been associated with central pontine myelinolysis in chronically debilitated patients with a prolonged period of hyper osmolarity or hypernatremia[6]. There for it is very important to keep in mind while using 3% NaCl saline.

In our study, preoperative and post operative serum electrolytes were within normal limits in both

groups.. But in case of group B, post operative serum sodium and chloride concentration slightly increased and statistically significant($p<0.001$) & ($p<0.05$) respectively.

Conclusion:

The preloading of hypertonic solution is superior to isotonic solution in trans urethral resection of prostate under sub arachnoid blockade in patients of benign hypertrophy of prostate.

Reference:

1. Kien, Nguyen D, Antognini, Joseph F, Reilly, et al:Resuscitation using small volume of hypertonic saline is effective for haemorrhagic shock.Br J Anaesth 1996; 83:137-40.
2. Crawford JS, Davis P, Lewis M.Some aspect of epidural block provided for elective caesarean section. Anaesthesia 1986; 41:1039-46.
3. Ring J, Messmer K. Incidence and severity of anaphylactoid reactions to colloid volume substitutes. Lancet 1977; 1:466-70.
4. Mc Care AF & Wildsmith JAW. Prevention and treatment of hypotension during central neural block. Br J Anaesth 1993;70:672-74.
5. Nguyen D, Antognini, Kien .Resuscitation using small volume of hypertonic saline is effective for haemorrhagic shock. Br J Anaesth 1996; 83:4.
6. Mc Kee AC, Winkelman MD, Banker BQ. Control pontine myelinolysis in severely bumed patient's relationship to serum hyperosmolarity. Neurology 1988; 38:1211-7.
7. Wier . The TURP syndrome. Canadian Journal of Anaesthesia 1922; 38:90-97
8. Veroli P, Benhamou D. Comparison of hypertonic saline isotonic saline and Ringer's lactate solution for fluid preloading before lumber extradural anaesthesia. Br J Anaesth1992; 69:461-64.
9. Imiak S, Weavind I, Dabney T, Wenker O.Interactive case report in Anaesthesia and Critical Care. The Internet Journal of Anaesthesiology 1999; 3: 1.
10. Goertz, Axel W, MD.Mehl, Tobies, MD Linder, Karl H.MD. A positive ionotropic effect of hypertonic saline has been demonstrated in isolated cardiac tissues.Br J Anaesh 1995; 82:1389-95.
11. Mouren S, Delayance S, Mion G. Mechanism of Myocardial contractility with hypertonic saline in isolated blood-perfusied rabit hearts, Anesth Analg 1995; 81:77-82.
12. Ueyama H, Heyanling, Tanigami H. Effect of crystalloid and colloid preload on blood volume in the parturient undergoing spinal anaesthesia for Elective cesarean section. Anesthesiology 1999; 91:1571-76.
13. Arndt J.O, Walter B, Joachim K. Incidence and time course of cardiovascular side effects during spinal anaesthesia after prophylactic use of intravenous fluid or vasoconstriction, Anaesth Analg 1998; 84:347-54.
14. Deutsch, S-As a refresher course in Anaesthetics-1992. New Orleans surgical 4 anesthetic consideration in Turp-Hatch, PD-A/C,15: 203-11.
15. Wang BW, Chiou YH, Chen WB. Intravenous pretreatment of hypertonic saline can prevent systemic hypotension induced by spinal anaesthesia. Acta Anaesthesiol Sin 1997;35:85-90.
16. Stern RH . Severe hypernatremia: the case for conservative management. Critical Care Med 1992; 20: 534-9.

Original Article

Comparative study of quality of subarachnoid blocks for caeasarean section by using bupivacaine alone & bupivacaine - fentanyl combination

Manisha Paul[1]*, Satyajit Dhar[2], DebasishBanik[3], AK Quamrul Huda[3], Moinul Hossain[3], Gulshan Ara Choudhury[4], AKM Shamsul Alam[5], A.Y.F Elahi Chowdhury[6]

[1]Dept. of Anaesthesiology, Square Hospital, Dhaka. [2]Dept of Anaesthesiology and Intensive care, Chittagong Medical College Hospital. [3]Associate Professor, Dept of Anaesthesia, Analgesia and Intensive Care Medicine, Bangabandhu Sheikh Mujib Medical University, Shahbag, Dhaka. [4]Associate Professor, Dept of Anaesthesiology and Intensive care, Chittagong Medical College Hospital. [5]Professor and Head, Dept of Anesthesiology and Intensive care, Chittagong Medical College Hospital. [6]Ex-professor National Institute of Cardiovascular Diseases, Dhaka

***Corresponding Author:** E-mail: manishachittagong@yahoo.com

Abstract

Background: *The popularity of subarachnoid block (SAB) in cesarean section in recent times is due to better understanding of the physiological changes associated with it and proper appreciation of its advantages & complications. Hyperbaric bupivacaine in adequate dose (12mg or more) for SAB often causes complications like hypotension, shivering, nausea, vomiting, chest pain & epigastric pain.*

Objectives: *The aim of study is to reduce these complications by using bupivacaine-fentanyl combination.*

Method: 150 patients of ASA grade I & II waiting for cesarean section under SAB were randomly allocated into three equal groups.

Group A : Received 0.5% hyperbaric bupivacaine-10 mg (2ml)
Group B : Received 0.5% hyperbaric bupivacaine-10 mg (2ml) & fentanyl- 15 µg [0.3ml]
Group C : Received 0.5% hyperbaric bupivacaine - 8 mg (1.6ml) & fentanyl- 15 µg. [0.3ml]

Parametric data like pulse, blood pressure, among the groups were analyzed by ANOVA test & nonparametric data like chest discomfort, epigastric pain, nausea, vomiting were analyzed by chi-square test.

Results: *In this study we found better analgesia & quality of block in bupivacaine-fentanyl group than bupivacaine alone group (p<.001).*

Conclusion: *By adding fentanyl we can reduce the dose of bupivacaine & also improve the quality of block.*

Key words: subarachnoid block, bupivacaine-fentanyl combination, quality of block.

(Journal of BSA, 2009; 22(2): 61-65)

Introduction

Subarachnoid block (SAB) is popular for caesarean section, being simple to perform, economical & it avoids the complications of general anaesthesia. Successful spinal anesthesia for cesarean section requires upto T4 level block which can be achieved with 10-15mg intrathecal dose of 0.5% hyperbaric bupivacaine[1]. But intraoperative common complications of SAB like hypotension, shivering, nausea, vomiting, chest pain and epigastric pain are common when using bupivacaine alone in this dose. The addition of intrtathecal fentanyl10-20µg may reduce the dose of bupivacaine & thereby reducing the intraoperative complications[1]. Bogra et al showed that fentanyl as adjunct of local anesthetics in SAB in various doses has effects on following parameters - visceral pain, hemodynamic stability, intraoperative sedation, intraoperative & postoperative shivering & postoperative pain. They also showed that, fentanyl is able to reduce the dose of bupivacaine & therefore its harmful effects[2]. In view of these points discussed above, the current study is intended to make a comparison of quality of block with different small doses of bupivacaine along with fentanyl and various complications occurring intraoperatively during cesarean section under SAB.

Methods

After taking informed written consent 150 healthy women with term pregnancy, aged between 20-35 years, height between 150-157.5cm, ASA I & II undergoing elective cesarean section under SAB were randomly allocated into three equal groups by simple lottery method. None of the patients had any contradiction for spinal anaesthesia. Complicated pregnancies such as multiple pregnancies, pregnancy induced hypertension and placenta previa were excluded. Foetal and maternal distress patients are also excluded from the study.

Group A : Received 0.5% hyperbaric bupivacaine 10mg(2ml)

Group B : Received 0.5% hyperbaric bupivacaine-10mg(2ml) & fentanyl- 15 μg. (0.3ml)

Group C : Received 0.5% hyperbaric bupivacaine-8mg(1.6ml) & fentanyl- 15 μg (0.3ml).

No premedication was given. SAB was done in lateral decubitus position at L3-L4 interspace using 25G Quincke spinal needle in each patient. Drugs was injected as per allocated schedule. Then the patient were turned supine position with a wedge under right buttock & 4L / min O_2 by face mask was started. Heart rate, blood pressure & oxyhaemoglobin saturation were measured just after positioning the patient & was recorded continuously at 2 minutes interval from induction to 20 mins. & then at 5 min intervals upto the end of operation or 60 min whichever was longer. After assuming the supine position, the upper level of block was evaluated. Sensory block was evaluated by using pinprick & chlorhexidin soaked swab by wiping it up to the abdomen, starting from inguinal region up to 4th intercostal space in midclavicular line[4]. Hypotension was defined as a decrease in systolic arterial pressure below 90 mm Hg or 25% decrease from the base line and was treated with IV isotonic fluid & ephedrine 5mg incremental dose as required[5]. intraoperative sedation was used.

Shivering, nausea, vomiting, chest pain, epigastric pain, pruritus were observed during that period. Each patient was asked a complete standard questionnaire regarding nausea, vomiting, chest pain, epigastric pain, pruritus. Shivering was treated by wrapping the patient with warm blanket & warmed IV fluid.

Results

There was no significant difference between groups in ages, parity & gravida. Regarding sensory block 74% in group A, 70% in group B & 92% in group C was at the level of T_7. The level of sensory block was significantly ($p<0.05$) higher in group C.

Table-I

Distribution of level of sensory block

Level of Sensory Block	Group A (n = 50)		Group B (n = 50)		Group C (n = 50)		P Value
	n	%	n	%	n	%	
T6	10	20.0	14	28.0	0	0.0	0.35
T7	37	74.0	35	70.0	46	92.0	0.02*
T8	3	6.0	1	2.0	4	8.0	-
Total	50	100.0	50	100.0	50	100.0	

* Data are analysed by Chisquare test. Values are regared as significant P< .05

The mean difference of pulse at different times was not statistically significant.

The mean differences of systolic & diastolic blood pressure, SpO2, respiratory rate were not statistically significant.

Chest pain & epigastric pain of the patients were very highly significant($p<0.001$) higher in group A with compared to group B & C.

Table-II

Distribution of chest pain & epigastric pain.

Pain	Group A (n=50)		Group B (n=50)		Group C (n=50)		P value
	n	%	n	%	n	%	
Chest pain	23	46.0	0	0.0	1	2.0	0.001 ***
Epigastric pain	9	18.0	0	0.0	0	0.0	0.001 ***

Data are analysed by chisqare test.Values are regared as significant p<.05

Table-III

Distribution of patient's satisfaction level.

Satisfaction of	Group A (n=50)		Group B (n=50)		Group C (n=50)		P value
patient	n	%	n	%	n	%	
Excellent	17	34.0	48	96.0	49	98.0	0.010 **
Good	33	66.0	2	4.0	1	2.0	-
Total	50	100	50	100	50	100	

Data are analysed by Chisqare test.Values are regared as regared as significant P <.05

The incidence of nausea & vomiting was significantly (p<0.05) higher in group A with compared to group B& C . Mild nausea & vomiting was found 2(4.0%) in group A & none was observed in group B & C.

There was no significant difference between 3 groups in other complications like shivering, chest heaviness, pruritus. But the level of satisfaction of patients was significantly (p<0.05) higher in group B & C with compared group A.

Discussion

Recent trends of obstetrics anesthesia show increased popularity of regional anesthesia amongst obstetric anesthetists. The increasing use of low dose technique of local anesthetics & opioids in recent years become popular for elective cesarean sections. Hyperbaric bupivacaine at 10mg or less has been shown to carry a risk of inadequate block. For this reason most of the anesthesiologists have favoured the use of higher doses (12mg or more) to overcome the incomplete blocks during cesarean section.[6] But higher dose itself has some complications. Bogra et al in 2005 showed that fentanyl as adjunct of local anesthetics in SAB in various doses has effects on following parameters - visceral pain, hemodynamic stability, intraoperative sedation, intraoperative & postoperative shivering & postoperative pain. They also showed that fentanyl is able to reduce the dose of bupivacaine & its harmful effects[6].

This randomized prospective study was carried out with an objective to compare the incidence of intraoperative complications in cesarean section under spinal anesthesia with bupivacaine[2,6] alone & the addition of fentanyl with various doses of bupivacaine. Regarding the quality of block addition of fentanyl to hyperbaric bupivacaine significantly improved of intraoperative surgical anesthesia for caesarean section[7]. In this study we have found complete analgesia & quality of block were better in bupivacaine - fentanyl group than bupivacaine only group.

In our study the level of sensory block was up to T_7 in majority such as 74% in group A, 70% in group B & 92% in group C. The level of sensory block T_7 was significantly (p<0.05) higher in group C with compared to group A & group B, whereas T_6 level was not significant between group A and group B. Although ideally T_4 level of block is required for caesarean section[1], but in our study the level of analgesia achieved is T_6 which is sufficient for caesarean section.

In this study, there were no significant difference among three groups regarding haemodynamic parameters, SpO2 and respiratory rate.

Comparing of equipotent doses of bupivacaine alone & bupivacaine fentanyl combination, we found no significant change after four, six, eight & ten minutes. Bogra et al. also have found that intraoperative hypotension increases with increasing the doses of bupivacaine along with fentanyl[2].

In our study we use smaller dose of fentanyl in subarachnoid space thereby producing no effect on APGAR score of newborn babies in group B&C. Dahlgren et al has also showed that, use of the smallest effective opioid dose minimize potentially adverse maternal & neonatal risks[7].

Chest pain was very highly significant ($p<0.001$) in group A (46%) compared to group B (2.0%) & C (none). Palmer CM et al. proved that 47.3% patients developed symptoms of chest pain during cesarean delivery under regional anesthesia & electrocardiographic changes occurred[8]. The changes were suggestive of myocardial ischemia. No patient without electrocardiographic change developed symptoms of chest pain. Incidence of chest pain was similar to our study. But we did not monitor ECG changes of the patients. Moran C et al speculate that the myocardial ischemia is a likely cause of both the ECG changes & of the symptoms of chest pain that they sometimes experience. Significant ST changes were recorded in 42% of patients preoperatively who felt chest pain requiring opioid analgesia[9].

In our study those patients who received intrathecal fentanyl did not complain any chest pain. Sivam Ramanthan et al[10] showed that the chest pain may be due to hyperkinetic myocardial contractility state which causes ST segment depression & it was heralded by tachycardia occurring within 15 mins after delivery. The time of onset of chest pain is similar to our study.

In same way, epigastric pain was found 18% in group A & none was found in group B & C. Cesarean section required traction of peritoneum & handling of intraperitoneal organs resulting in intraoperative visceral pain[2]. In our study time appearance of epigastric pain during operation was correlated with the time of peritoneal closing. Most probably this visceral pain might be expressed by patients as epigastric pain.

In this study, nausea & vomiting was significantly ($p<0.05$) higher in group A with compared to group B & C. Jaishri et al[2] also reported that incidence of vomiting was more in bupivacaine alone group than fentanyl combination group. Nausea & vomiting have multiple etiologies, which include hypotension, vagal hyperactivity, visceral pain, I/V opioid supplementation, uterotonic agents & increased gut motility[11]. In our study all 3 groups were heamodynamically stable. Despite achieving an adequate sensory level nausea during manipulation of the uterus & at the time of peritoneal closure was sometimes a problem in the present study group A. Christer Hulstrand et al. have shown beneficial effects of adding various opioids to the local anesthetic solution administered intrathecally[7]. Subarachnoid opioids successfully decrease the incidence of intraoperative visceral pain[12]. In our study incidence of nausea & vomiting was negligible in group B&C. Chest heaviness was highly significant ($p<0.001$) in group A compared to group B&C which were 66.0% in group A, 2.0% in group B & none was found in group C. Gunner Dahlgren et al. have showed that co-administration of small doses of opioids & bupivacaine for spinal anesthesia reduces intraoperative feeling of discomfort in the chest.

Bruce et al. showed in their study that pruritus was common in the patients receiving intrathecal fentanyl, although in most cases, it is mild as do not require treatment. They found that the use of 20 μg dose of fentanyl lead to more pronounced pruritus that although well tolerated. In our study we found that pruritus after a 15 μg dose of fentanyl was less prominent which did not cause any ill effect to the patient. Christer Hulstrand et al. also observed that intraoperative pruritus was almost exclusively related to the use of sufentanyl[11].

Incidence of intraoperative complications (like chest pain, epigastric pain, nausea - vomiting) during elective caesarean section under SAB in mentioned and in our study are same. We can minimize the intraoperative complication by reducing the dose of bupivacaine and adding small dose of fentanyl (15μg)

Conclusion

We can routinely use fentanyl in combination with bupivacaine intrathecally to improve the quality of subarachnoid block.

References

1. Farragher Rachel A and Kodali Bhavani Shankar. anesthesia, In: Thomas E J Healy, Paul R Knight, Wylie Churchill-Davidson's A practice of anesthesia; 7th ed. London, Arnold; 2003: 930.
2. Bogra Jaishri, Arora Nomita, Srivasta Pramita, Synergistic effect of intrathecal fentanyl and bupivacaine in spinal anesthesia for cesarean section, BMC Anesthesiology 2005,5:5.
3. Barua PC, Rahman AKM, Rahman A. Reports on awareness workshop on research methods for the specialists and program managers. Institute of Child and Maternity Health. 2001; 80.
4. Nagata E et. al., Comparison of 8 mg and 10 mg hyperbaric bupivacaine during spinal anesthesia for cesarean section in Japanese parturient, Masui. 2004; 53(2):131-6.
5. Gupta S. Controversies in obstetric anesthesia, Indian J. Anesthesia, 2005; 49(3):180-189.
6. Choi DH, Ahn Hj, Kim MH, Bupivacaine-sparing effect of fentanyl in spinal anesthesia for cesarean delivary, Reg Anes Pain Med. 2000; 25(3): 240-5.
7. Gunnar Dahlgren, Hulsrnd christer, Jakobson JPN et al. Intrathecal sufentanyl, fentanyl or placebo added to bupivacaine for caesarean section, Anaesth Analg 1997; 85(6): 288-93.
8. Palmer CM, Norris MC, Giudici MC, Leighton BL, DeSimone CA. Incidence of electrocardiographic changes during cesarean delivery under regional anesthesia, Anesth Analg.1990; 71(1):36-43.
9. Moran C.I, Bhuinneain NI, Cunningham MI et al. Myocardial ischemia in normal patients undergoing elective Cesarean section : a peripartum assessment, Anesthesia 56, 11; 2001: 1051-1058(8).
10. Roy Louise, Ramanthan Sivam, ST-segment depression and myocardial contractility during Cesarean section under spinal anesthesia, Can J Anesth 1999; 46 (1): 52-56.
11. Balki M, Carvalho JC intraoperative nausea and vomiting during cesarean section under regional anesthesia. Int J Obstetrics Anesth.2005, Jul; 14(3):230-41.
12. B.H. Minzter, Pain Relief and Anesthesia in Obstetrics, 1st edition, Andre van Zundert; 257.

Article for Special Interest

Sacroiliac joint arthropathy and low back pain

K. Sardar[1*], G Das[2], V Kapoor[3], P Mahta[4]-, Sabina Yeasmeen[5] , Raihan Uddin[6]

[1]Assistant professor, Department of Anaesthesiology, Ibrahim Medical College and BIRDEM, Dhaka, Bangladesh, [2]Chairman, Indian chapter of World Institute of Pain, [3]K K Health Care Centre, Gurgaon, Haryana, India, [4]HCG Medisurge Hospitals, Navrangpura, Gujrat, India[5]Assistant Professor, Department of Anaesthesia Analgesia and Intensive Care Medicine, BSMMU, [6]Junior Consultant, Department of Anaesthesiology, Ibrahim Medical College and BIRDEM, Dhaka, Bangladesh

*** Corresponding author:** Email: kawsardr@yahoo.com

(Journal of BSA, 2009; 22(2): 78-83)

Key words: SI joint, low back pain, intervention pain management

Introduction

Sacroiliac (S-I) joint dysfunction is understood by clinicians as one of many causes of the general category of low back pain. S-I joint dysfunction may wholly be responsible for the low back pain syndrome and/or may be contributory to low back pain in concern with other pathology of the lumbar spine. It is often an overlooked and underappreciated diagnosis. Sacroiliac (SI) joint pain is a challenging condition affecting 15% to 25% of patients with axial low back pain, for which there is no standard long-term treatment. Recent studies have demonstrated that historical and physical examination findings and radiological imaging are insufficient to diagnose SI joint pain. The most commonly used method to diagnose the SI joint as a pain generator is with small-volume local anesthetic blocks, although the validity of this practice remains unproven.

Brief Anatomy

The S-I joint can be thought of as the bottom joints of the spine relating to the hip bones, The sacrum (bottom of the spine) relates on each side to the ilia (hip bones) to form the sacroiliac joints. The sacroiliac (SI) joint is the largest axial joint in the body, with an average surface area of 17.5 cm^2.[1] There is wide variability in the adult SI joint, encompassing size, shape, and surface contour. Large disparities may even exist within the same individual. The SI joint is most often characterized as a large, auricular-shaped, diarthrodial synovial joint. In reality, only the anterior third of the interface between the sacrum and ilium is a true synovial joint; the rest of the junction is comprised of an intricate set of ligamentous connections. Because of an absent or rudimentary posterior capsule, the SI ligamentous structure is more extensive dorsally, functioning as a connecting band between the sacrum and ilia[2]. The main function of this ligamentous system is to limit motion in all planes of movement. In women the ligaments are weaker, allowing the mobility necessary for parturition.

Nerve supply

The innervation of the SI joint remains a subject of much debate. The lateral branches of the L4-S3 dorsal rami are cited by some experts as composing the major innervation to the posterior SI joint[1]. Other investigators claim that L3 and S4 contribute to the posterior nerve supply[3]. The innervation of the anterior joint is similarly ambiguous. Early 20th century German literature asserts the anterior SI joint is supplied by the obturator nerve, superior gluteal nerve and the lumbosacral trunk[4]. More recent literature suggests the anterior joint is innervated by L2-S2[1], L4-S2[5], and the L5-S2 ventral rami[6]. Some authors have even suggested that the anterior SI joint is devoid of nervous tissue[7]. In a study testing the ability of L5 dorsal ramus and S1-4 lateral branch blocks to protect the SI joint from an experimental stimulus, 6 of 10 subjects retained the ability to perceive ligamentous probing [8].

Biomechanics and function

There have been numerous attempts to discern the biomechanics of the SI joint. These motion studies can be summarized as follows: the SI joint rotates about all 3 axis, although the movements are very small and difficult to measure[9]. Miller et al.[10] studied the load-displacement behavior of single and paired SI joints in 8 elderly cadavers. The authors found that with 1 leg immobile, movements in all planes ranged from between 2 to 7.8 times more than that measured with both legs fixed. In a series of cadaveric studies, Vleeming et al.[11] found that

the total range of motion during flexion and extension at the SI joint rarely exceeded 2 degrees, with 4 degrees being the upper limit during sagittal rotation.

Prevalence

Studies are further compromised by the fact that most have used either physical examination findings and/or radiological imaging techniques to make the diagnosis of SI joint pain. Prevalence of SI joint arthropathy has not been well studied. The largest of these is a retrospective study by Bernard and Kirkaldy-Willis[12], who found a 22.5% prevalence rate in 1293 adult patients presenting with LBP. Diagnoses in this series were based predominantly on physical examination.

Schwarzer et al. [13] and Maigne et al. [14] conducted a prevalence study in 54 patients with unilateral LBP using a series of blocks done with different LA based on International Spinal Injection Society guidelines. Nineteen patients had a positive response (75% pain relief) to the lidocaine screening block. Among these patients, 10 (18.5%) responded with >2 h pain relief after the confirmatory block with bupivacaine and were considered to have true SI joint pain (95% CI, 9%–29%). Based on these studies, the prevalence of SI joint pain in carefully screened LBP patients appears to be in the 15%–25% range.

Mechanism of injury

The mechanism of SI joint injury has previously been described as a combination of axial loading and abrupt rotation. On an anatomic level, pathologic changes affecting many different SI joint structures can lead to nociception. These include capsular or synovial disruption, capsular and ligamentous tension, hypomobility or hypermobility, extraneous compression or shearing forces, abnormal joint mechanics, microfractures or macrofractures, chondromalacia, soft tissue injury, and inflammation. Mechanistically, there are numerous reported etiologies for SI joint pain. To simplify matters, these causes can be divided into intraarticular and extra-articular sources. Arthritis and infection are two examples of intraarticular causes of SI joint pain. Extra-articular sources are the more common of the two and include enthesopathy, fractures, ligamentous injury, and myofascial pain. Clinical studies have demonstrated significant pain relief after both intraarticular and periarticular SI joint injections[15, 16,17].

Diagnosis

History and Physical Examination

Many involve distraction of the SI joints, with 2 of the most common ones being Patrick's test and Gaenslen's test. Despite the plethora of diagnostic tests, clinical studies have for the most part demonstrated that neither medical history nor physical examination findings are consistently capable of identifying dysfunctional SI joints as pain generators. In addition, Dreyfuss et al.[18] found 20% of asymptomatic adults had positive findings on 3 commonly performed SI joint provocation tests. Some of these studies have found moderate to high inter-examiner reliability[19], most have not[20].

Radiological Studies

Radiologic findings in patients with SI joint pain have been similarly disappointing. In studies by Maigne et al. [21] and Slipman et al. [22], the investigators found sensitivities of 46% and 13%, respectively, for the use of radionuclide bone scanning in the identification of SI joint pain. In a retrospective analysis by Elgafy et al.[23], CT imaging was found to be 57.5% sensitive and 69% specific in diagnosing SI joint pain.

Pain Referral Patterns

Fortin et al.[24] performed provocative SI joint injections using contrast and lidocaine. Sensory changes were localized to the ipsilateral medial buttock inferior to the posterior superior iliac spine in 6 of the 10 subjects. In a follow-up study, independent examiners selected 16 individuals among 54 with chronic LBP whose pain diagrams most closely resembled the pain referral patterns obtained in the first study[25]. These 16 patients proceeded to undergo provocative SI joint injections with contrast and LA. All 16 experienced concordant pain during the injection, with 14 obtaining pain relief after deposition of LA. Slipman et al.[26] conducted a retrospective study to determine the pain referral patterns in 50 patients with injection-confirmed SI joint pain. In contrast to the findings by Fortin et al.[25] the authors found the most common referral patterns for SI joint pain to be radiation into the buttock (94%).

Diagnostic Blocks

Extravasation of LA to surrounding pain-generating structures such as muscles, ligaments, and lumbosacral nerve roots can lead to false-positive blocks. Conversely, failure to obtain adequate LA spread to the anterior and cephalad portions of the SI joint can result in false-negative blocks. In a classic study by North et al.[27] examining the specificity and sensitivity of a battery of lumbosacral LA blocks in 33 patients with a chief complaint of sciatica, the authors found the specificity of all blocks to be exceedingly low. SI joint blocks were not performed in this study.

In a pilot study by Fortin et al.[25] mapping SI joint referral patterns in asymptomatic volunteers, extravasation of contrast (mean 1.6 mL injected) occurred in 9 of 10 subjects during SI joint injection, with half having at least moderate spread outside the joint. After the injection of LA, 40% of subjects noted lower extremity numbness, indicating inadvertent anesthetization of the lumbosacral nerve roots. In the Maigne et al. [14] study, 3 of the initial 67 patients were excluded because of "sciatic palsy" after the screening block and another 7 were excluded because penetration of the SI joint was impossible. Regardless of the imaging modality used to confirm intraarticular injection, SI joint injections should never be performed blindly. Rosenberg et al.[28] performed a double-blind study in 37 patients (39 joints) to determine the accuracy of clinically guided SI joint injections using CT imaging as the standard. The authors found that intraarticular injection was accomplished in only 22% of patients, whereas sacral foraminal spread occurred 44% of the time. In 3 patients, no contrast was seen on CT scanning, indicating probable vascular uptake. In 24% of injections, contrast extended into the epidural space. Maigne et al.[14] sought to determine the prevalence of SI joint pain using a series of blocks with 2 different LA. In the 54 patients who completed the study, 19 obtained 75% pain relief with the lidocaine screening block.

Treatment

The treatment of SI joint pain is widely acknowledged to be one of the most challenging problems confronting pain physicians. Evidence supporting this statement can be seen by the plethora of different therapies that have been advocated for this disorder. Generally, these treatments can be divided into 2 categories: those directed at correcting the underlining pathology and those aimed at alleviating symptoms. For both of these categories, the evidence supporting any one therapy is limited by the lack of controlled outcome studies.

Psychosocial Issues

Recent studies have provided incontrovertible evidence that psychopathology and other psychosocial factors can influence both the development of chronic pain conditions and the response to treatment. In a study by Polatin et al.[29] conducted in 200 chronic LBP patients, the authors found that 77% met lifetime criteria and 59% demonstrated current symptoms for at least one psychiatric diagnosis, with the most common being depression, substance abuse, and anxiety disorders. Notably, more than 50% of those with depression and more than 90% of patients with substance abuse or an anxiety disorder experienced symptoms before the onset of LBP. Most, but not all, studies have shown untreated psychopathology to negatively affect LBP treatment outcomes.

Conservative Management

Nonsurgical stabilization programs have been advocated for SI joint pain. These range from the application of pelvic belts that reduce the sagittal rotation of incompetent SI joints in pregnant women to exercise-induced pelvic stabilization programs. In a study by Mooney et al.[30], the authors found that 5 women with injection-confirmed SI joint pain had electromyographic-documented hyperactivity of the ipsilateral gluteus muscles and contralateral latissimus muscle compared with 15 asymptomatic control patients. After a 2-1/2 month exercise program, all 5 patients achieved a significant reduction in pain and a return of myoelectric activity to normal patterns.

Intraarticular Injections

Intraarticular injections with steroid and LA often serve the dual function of being therapeutic and aiding in diagnosis. To summarize these studies, most but not all investigators have found radiologically guided SI joint injections to provide good to excellent pain relief lasting from 6 months to 1 year. Along with a multitude of studies demonstrating prolonged pain relief after intraarticular SI joint steroid injections, double-blind studies have shown a beneficial effect for periarticular corticosteroid treatment as well [16].

Radiofrequency Denervation Procedures

Several investigators have performed radiofrequency (RF) denervation procedures in an attempt to provide prolonged pain relief to patients suffering SI joint pain. The techniques used have ranged from denervating the nerves supplying the SI joint[31] to creating lesions in the joint itself [32], with one study using a combination of the two[33]. The success rates of studies targeting the nerve supply are higher than those focusing on the joint itself, with approximately two thirds of patients reporting significant pain relief. The major drawback to percutaneous RF denervation procedures is that they should not be expected to alleviate pain emanating from the ventral SI joint. In the study by Schwarzer et al.[14], ventral capsular pathology was shown to account for 69% of all CT pathology in the 13 patients with a positive response to diagnostic SI joint blocks. Complicating matters further are that the nerves lesioned during RF procedures innervate other pain-generating structures besides the SI joint, and the SI joint is likely innervated by other nerves inaccessible for denervation.

Surgical and Other Invasive Interventions

In 1999, Srejic et al.[34] reported 12–16 months of significant pain relief in 4 patients with SI joint pain who received a series of 3 intraarticular injections with hyaluronic acid. Three of these patients had postsurgical SI joint pain and one suffered from severe osteoarthritis of the spine. The rationale for this treatment stems from studies demonstrating long-term pain relief with hyaluronic acid injections in degenerative joint disease of the knee. Ongley et al.[35] found that LBP patients who received 6 wks of proliferant therapy had lower pain scores and disability indices at their 6 months follow-up than "control" patients who received saline injections. Despite these findings, the lack of specific diagnoses, the numerous other treatment differences between groups, and the targeting of pain generators outside the SI joints limit the relevance of this study. Neuroaugmentation of the third sacral nerve root has also been reported to provide adequate pain relief in 2 patients with severe SI joint pain unresponsive to conventional therapy[36].

Conclusion

The SI joint is a real yet underappreciated pain generator in an estimated 15% to 25% of patients with axial LBP. Whereas historical and physical examination findings have been previously advocated as useful tools in identifying patients with SI joint pain, more recent studies have demonstrated they have limited diagnostic value. Presently, small-volume diagnostic blocks remain the most commonly used method for diagnosing this disorder. Owing to the complexity of the joint, the mechanisms of SI pain are numerous and ill-defined. When a pathological condition such as leg length discrepancy or altered gait mechanics is present, correcting the underlying defect is the safest and most reliable treatment option. Intraarticular and periarticular corticosteroid injections have been shown in most, but not all, studies to provide good to excellent pain relief lasting up to 10 month in patients with and without spondylarthropathy. One promising area in the treatment of SI joint pain is RF denervation, although the conclusions that can be drawn are limited by the heterogeneous methods used and the lack of controlled studies.

References:

1. Bernard TN, Cassidy JD. The sacroiliac syndrome. Pathophysiology, diagnosis and management. In: Frymoyer JW, ed. The adult spine: principles and practice. New York: Raven, 1991;2107–30.
2. Bowen V, Cassidy JD. Macroscopic and microscopic anatomy of the sacroiliac joint from embryonic life until the eighth decade. Spine 1981;6:620–8.
3. Murata Y, Takahashi K, Yamagata M, et al. Sensory innervation of the sacroiliac joint in rats. Spine 2000;16:2015–9.
4. Pitkin HC, Pheasant HC. Sacrarthrogenic telalgia I: a study of referred pain. J Bone Joint Surg 1936;18:111–33.
5. Solonen KA. The sacroiliac joint in the light of anatomical, roentgenological and clinical studies. Acta Orthop Scand 1957;27:1–27.
6. Ikeda R. Innervation of the sacroiliac joint: macroscopic and histological studies. J Nippon Med Sch 1991;58:587–96.
7. Fortin JD, Kissling RO, O'Connor BL, Vilensky JA. Sacroiliac joint innervation and pain. Am J Orthop 1999;28:68–90.
8. Dreyfuss P, Park K, Bogduk N. Do L5 dorsal ramus and S1–4 lateral branch blocks protect

the sacroiliac joint from an experimental pain stimulus? A randomized, double-blinded controlled trial. Presented at the International Spinal Injection Society 8th Annual Scientific Meeting, San Francisco, CA, September, 2000;8–10.

9. White AA, Panjabi MM. Clinical biomechanics of the spine. 2nd ed. Philadelphia: JB Lippincott, 1990.
10. Miller JA, Schultz AB, Andersson GB. Load-displacement behavior of the sacroiliac joints. J Orthop Res 1987;5:92–101.
11. Vleeming A, van Wingerden JP, Snijders CJ, et al. Load application to the sacrotuberous ligament: influence on sacroiliac joint mechanics. Clin Biomech 1989;4:204–9.
12. Bernard TN, Kirkaldy-Willis WH. Recognizing specific characteristics of nonspecific low back pain. Clin Orthop 1987;217:266–80.
13. Schwarzer AC, Aprill CN, Bogduk N. The sacroiliac joint in chronic low back pain. Spine 1995;20:31–7.
14. Maigne JY, Aivaliklis A, Pfefer F. Results of sacroiliac joint double block and value of sacroiliac pain provocation tests in 54 patients with low back pain. Spine 1996;21:1889–92.
15. Luukkainen R, Wennerstrand PV, Kautiainen HH, et al. Efficacy of periarticular corticosteroid treatment of the sacroiliac joint in non-spondyloarthropathic patients with chronic low back pain in the region of the sacroiliac joint. Clin Exp Rheumatol 2002;20:52–4.
16. Frymoyer JW, Howe J, Kuhlmann D. The long-term effects of spinal fusion on the sacroiliac joint and ilium. Clin Orthop 1978;134:196–201.
17. Cush JJ, Lipsky PE. Reiter's syndrome and reactive arthritis. In: Koopman WJ, ed. Arthritis and allied conditions: a textbook of rheumatology, Vol. 1, 14th ed. Philadelphia: Lippincott Williams& Wilkins, 2001;1:1324–44.
18. Dreyfuss P, Dreyer S, Griffin J, et al. Positive sacroiliac screening tests in asymptomatic adults. Spine 1994;19:1138–43.
19. Laslett M, Williams M. The reliability of selected pain provocation tests for sacroiliac joint pathology. Spine 1994;19:1243–9.
20. Meijne W, van Neerbos K, Aufdemkampe G, Van der Wurff P. Intraexaminer and interexaminer reliability of the Gillet test. J Manipulative Physiol Ther 1999;22:4–9.
21. Maigne JY, Boulahdour H, Chatellier G. Value of quantitative radionuclide bone scanning in the diagnosis of sacroiliac joint syndrome in 32 patients with low back pain. Eur Spine J 1998;7:328–31.
22. Slipman CW, Sterenfeld EB, Chou LH, et al. The value of radionuclide imaging in the diagnosis of sacroiliac joint syndrome. Spine 1996;21:2251–4.
23. Elgafy H, Semaan HB, Ebraheim NA, Coombs RJ. Computed tomography findings in patients with sacroiliac pain. Clin Orthop 2001;382:112–8.
24. Fortin JD, Dwyer AP, West S, Pier J. Sacroiliac joint: pain referral maps upon applying a new injection/arthrography technique. Part I: asymptomatic volunteers. Spine 1994;19:1475–82.
25. Fortin JD, Aprill CN, Ponthieux B, Pier J. Sacroiliac joint: pain referral maps upon applying a new injection/arthrography technique. Part II: clinical evaluation. Spine 1994;19:1483–9.
26. Slipman CW, Jackson HB, Lipetz JS, et al. Sacroiliac joint pain referral zones. Arch Phys Med Rehabil 2000;81:334–8.
27. North RB, Kidd DH, Zahurak M, Piantadosi S. Specificity of diagnostic nerve blocks: a prospective, randomized study of sciatica due to lumbosacral disease. Pain 1996;65:77–85.
28. Rosenberg JM, Quint DJ, de Rosayro AM. Computerized tomographic localization of clinically-guided sacroiliac joint injections. Clin J Pain 2000;16:18–21.
29. Polatin PB, Kinney RK, Gatchel RJ, et al. Psychiatric illness and chronic low-back pain. The mind and spine: which goes first? Spine 1993;18:66–71.
30. Mooney V, Pozos R, Vleeming A, et al. Exercise treatment for sacroiliac pain. Orthopedics 2001;24:29–32.
31. Yin W, Willard F, Carreiro J, Dreyfuss P. Sensory stimulation-guided sacroiliac joint

radiofrequency neurotomy: technique based on neuroanatomy of the dorsal sacral plexus. Spine 2003;28:2419–25.

32. Ferrante FM, King LF, Roche EA, et al. Radiofrequency sacroiliac joint denervation for sacroiliac syndrome. Reg Anesth Pain Med 2001;26:137–42.
33. Gevargez A, Groenemeyer D, Schirp S, Braun M. CT-guided percutaneous radiofrequency denervation of the sacroiliac joint. Eur Radiol 2002;12:1360–5.
34. Srejic U, Calvillo O, Kabakibou K. Viscosupplementation: a new concept in the treatment of sacroiliac joint syndrome: a preliminary report of four cases. Reg Anesth Pain Med 1999;24:84–8.
35. Ongley MJ, Klein RG, Dorman TA, et al. A new approach to the treatment of chronic low back pain. Lancet 1987;2:143–6.
36. Calvillo O, Esses SI, Ponder C, et al. Neuroaugmentation in the management of sacroiliac joint pain: report of two cases. Spine 1998;23:1069–72.

Original Article

Effect of small dose intermittent IV pethidine in combination with diclofenac for post operative pain relief

Debabrata Banik[1*], Md. Abdul Hye[1], Akhtaruzzaman AKM[1], Lutful Aziz[2], Sabina Yeasmeen[3], K M Iqbal[4]

[1]Associate Professor, Dept. of Anaesthesia, Analgesia and Intensive Care Medicine, Bangabandhu Sheikh Mujib Medical University, Shahbag, Dhaka. [2] Consultant, Dept. of Anaesthesiology, Apollo Hospital, Dhaka. [3]Assistant Professor, Dept. of Anaesthesia, Analgesia and Intensive Care Medicine, Bangabandhu Sheikh Mujib Medical University, Shahbag, Dhaka. [4]Chief Consultant, Dept. of Anaesthesiology, Apollo Hospital, Dhaka

***Corresponding Author:** dbanik85@gmail.com

Abstract:

Background: Post operative pain is an acute type of pain. There is convincing evidence that unrelieved post operative pain may result in harmful physiological and psychological effects with significant morbidity and even mortality. Complete and effective pain management is an essential for the patient who recovers from surgery. For this reason we used easy available drugs opioid, NSAID and its combination for better post operative analgesia with the aim of avoiding expensive patient controlled analgesia technique, infusion or intramuscular painful roout of administration.

Methods: 120 patients ASA grade-I and II were selected for post operative pain releive in upper and lower abdominal surgery. All patients divided into three groups and each group are equal in number and same type of surgery. Among the three groups, two groups received small intermittent intravenous pethidine and diclofenac pre and post operatively and other groups received intra muscular pethidine with diclofenac pre and post operatively (controlled group). All vital parameter and pain score both VAS and VRS were recorded in perioperative period up to 24 hours postoperatively.

Result: Good level of anaesthesia was achieved in all groups (VAS>30mm) of upper and lower abdominal surgery except early post operative period. In early post operative period significant difference of VAS in group II and III (P<.01) was found with group I. But after half an hour and one hour significant difference of VAS in group II and III (P<0.02, P<0.001) was found with group I. Over all excellent analgesia was possible in group II VAS, VRS always more than 30mm and all vital sign were stable.

Conclusion: Small intravenous intermittent dose of pethidine in combination with diclofenac sodium effectively controlled post-operative pain.

Key Word: Post operative analgesia, small intra venous dose of pethidine, diclofenac.

(Journal of BSA, 2009; 22(2): 40-47)

Introduction:

Pain is one of the most common fears for patients coming into hospital for surgery. Unfortunately the patients worries about and the fears of pain are how on the priority list of the medical staff[1]. One of the reasons frequently mentioned for inadequate post operative pain relieve in the literature is the nurse and doctor fear to administer opioid analgesics because of their side effects[1]. Prevention of post operative complication[2], evidence of shortness hospital stay, increased patients satisfaction with effective relief of post operative type of acute pain[3].

Pain management is not too easy because the variable response to pain. The response to pain can be highly variable between individual as well as in the same individual in different times[4]. It is likely that all form of acute type of post operative pain is poorly managed. A wide range of pharmacological and psychological treatment for post operative pain

is now available inadequate or improper application of knowledge and therapies currently available is certainly one of the most important factor indequate relief of pain[5,6].

The post operative pain relief immediately after major surgery can not be achieved with opioids alone in all patients without respiratory depression or other significant side effects[7]. So it is not always applicable or without side effect and require skill manpower. Now a days another combined analgesic method, opioid with NSAIDs is used to manage postoperative pain[9] with the aim of reduction of opioid doses.[8]

Combined analgesic techniques are also popular for postoperative pain management in this country without definitive effective method of drugs administration. In our setup intramuscular opioids and NSAIDs are popular in combined analgesic techniques. Intramuscular injection is painful and absorption is unreliable, as a result analgesic effect is unpredictable. For this reason we may use easy available drugs and formulation for better post operative analgesia to avoid expensive PCA, infusion or intramuscular route of administration.

In our setting among the NSAIDs diclofenac sodium is commonly used, easily available and very cheap and equianlgesic with ketoprofen, ketorolac and piroxicam[9,10]. Among the opioids pethidine is one which is supplied to the Government hospital though not easily procurable from the open market. So, in this combined study diclofenac sodium and pethidine are included.

This investigation was conducted for better control of postoperative pain and in addition it was done with the intention of reducing the incidence of side effects and to search for better method of pethidine administration while improving the quality of pain relief.

Methods and materials:

A double blind, randomized controlled study of 120 cases of different surgical plans carried out in IPGMR (BSMMU). Sixty adult patients age ranging from 20-50 years, undergoing upper abdominal surgery like open cholecystectomy by subcostal incision and sixty adult of same age range under going lower abdominal surgery like abdominal hysterectomy of not more than 90 minutes duration under general anaesthesia were enrolled in the study. All the patients were in ASA group I & II and weighing between 50 to 70kg. Patients were included in the study after obtaining informed consent. Each surgical plan patients were placed in three different groups. Any patient with known history of sensivity of NSAID, peptic ulcer, bronchial asthma, hepatic or renal disease, any bleeding history or anti coagulant therapy were excluded from study.

Group-I (Control group) This group of patients received diclofenac sodium as rectal suppository 1 mg/kg body weight (bw) half an hour before commencing surgery in the ward / anaesthetic preparation room and 50mg 8 hourly in postoperative period. Time maintained from first dose administered by a nurse who was not aware of the study. This group of patients also received intramaucular pethidine 1.5mg/kg in post-operative period upto 24-hours, when VAS is more than 30mm, not repeated with 2 hours.

Group-II This group of patients received diclofenac sodium as rectal suppository 1mg/kgbw half an hour before commencing surgery in the ward / anaesthetic preparation room and 50mg 8 hourly in post-operative period. Time maintained from first dose administered by a nurse who was not aware of the study. This group also received 1st dose pethidine 0.5mg/kg bw intravenously in postoperative period when VAS more than 30mm. Then 10mg Pethidine was given hourly by intravenously up to 24 hours when VAS is more than 30mm, not repeated within 10 minute.

Group-III: this group of patients received rectal suppository of diclofenac sodium 1mg/kgbw at the end of surgery and 50mg 8 hourly in post-operative period. Time maintained with first dose administered by a nurse who was not aware of the study. This group also received 1st dose pethidine 0.5mg/kg intravenously in post-operative period when VAS more than 30mm. Then 10mg pethidine was given hourly intravenously up to 24 hours when VAS is more than 30mm but not repeated within 10 minute.

Pre operatively all patients were examined properly and record sheet was filled for each patient. 100mm (10 cm) visual analogue scale slide roller and verbal rating rating score were used to assess the level of postoperative pain. The VAS was explained to the patient that one extreme of the scale indicate no pain and other end worst pain possible. The verbal rating score also explained to the patient (No pain,

mild pain, moderate pain and severe pain). All patients were premedicated with midazolam 7.5mg and Ranitidine 150mg orally at night before operation.

In all general anaesthesia was started with pre oxygenation induced with thiopental sodium 5mg/kg and suxamethonium (1.5 mg/kg) body weight and fentanyl 1 µg/kgbw was injected to facilitate intubation. Anaesthesia was maintain with nitrous oxide and oxygen (60:40) and halothane (0.50%). atracurium 0.5mg/kgbw was given as a bolus dose and repeated dose of 0.2mg/kg bw given when necessary to continue relaxation. Fentanyl (0.25µg/kg bw) was given at 30 minute interval for maintaining analgesia and amount of fentanyl given during surgery was recorded. Pulse, Systolic blood pressure, diastolic blood pressure, arterial oxygen saturation and $ETCO_2$ were recorded in the pre, per and postoperative period. Patient was recovered from anaesthesia to maintain by standard procedure. Pain was assessed by VAS and VRS on arrival, 30 minutes, 1 hour, 2 hours, 4 hours, 8 hours, 12 hours, 18 hours and 24 hours after arrival in the post-operative ward. Sedation score were recorded with pain assessment in same interval. (awake and alert 1, awake and drowsy 2, asleep and ready arousal 3 and asleep[4]) and also recorded any side effect.

For use of intravenous preparation of pethidine, one ampoule (100mg) pethidine dissolved in 18 ml distilled water in 20ml syringe. So 1ml solution contains 5mg pethidine. Time of first pethidine requirement and total amount of pethedine and diclofenac sodium were recorded postoperatively. Data were analyzed by student's "t", ANOVA test, X^2-test and SPSS version 12 for window were used for comparative analysis. A P value of <0.05 was considered as significant.

Observation and Result:

Observations were made on haemodynamic parameter, analgesic requirement and level of analgesia in perioperative period. Pain (VRS/VAS), level of sedation, haemodynamic parameter, side effects were recorded 24 hours of post operative periods. Demographic data and different parameter were described as mean with standard error in table and graph.

Demographic characteristics in all groups in respect to age, weight, gender and educational status were similar (Table - I). Pre-operative and peroperative parameter like pulse rate, respiratory rate, SBP, DBP, SPO_2 EtCO2 and resting VAS, VRS between the groups were not statistically significant (Table – II & III). There was no significant difference of amount of fentanyl used in intra operative period and duration of surgery (Table-IV) and recovery score.

Pain score (VAS and VRS) at each time of measurement after surgery were mostly in acceptable limit but early post operative period (0 - 1 hours) were significantly different in group-I and group II with group III at arrival ($p < .05$) and at 30 minute and one hour. After two hours VAS and VRS gradually reduced and achieved acceptable level but score was significantly higher in group I and III ($p<0.05 – 0.01$).

Table-I

Demographic data of different study group

	Upper abdominal Surgery			Lower abdominal Surgery		
	Group I (N=20) Mean±SE	Group-2 (N=20) Mean±SE	Group 3 (N=20) Mean ±SE	Group I (N=20) Mean+SE	Group-2 (N=20) Mean+SE	Group 3 (N=20) Mean ±SE
Age (yr)	39. 6± 1.39	39.2±1.99	38.7±1.8	40.30 ± 2.38	42.05±1.46	41.30±1.22
Sex	F 16M4	F 15M5	F 16M4	F 17 M3	F 16M4	F 16M4
Wt (kg)	58.15±0.86	58.00±0.96	59.60±0.8	58.25±0.80	58.50±0.81	59.0±0.75
Edu	UNG 12 G 8	UNG 10 G 10	UNG 11 G 09	UNG 13 G 7	UNG 11 G 9	UNG 12 G 8

Data was analyzed by ANOVA test, Values regarded significant, there in different study group $p<0.05$. no significant difference between the groups, F = Female, M = Male, UNG = Undergraduate, G= Graduate

Table-II

Preoperative parameter of the patients

	Upper abdominal Surgery			Lower abdominal Surgery		
	Group I (N=20) Mean±SE	Group-2 (N=20) Mean±SE	Group 3 (N=20) Mean ±SE	Group I (N=20) Mean+SE	Group-2 (N=20) Mean+SE	Group 3 (N=20) Mean ±SE
ASA	ASA 1 17 ASA 2 03	ASA 1 16 ASA 2 04	ASA 1 16 ASA 2 04	ASA 1 14 ASA 2 6	ASA 1 15 ASA 2 05	ASA 1 14 ASA 2 6
Pulse	79.10±3.37	81.7±1.46	81.15±2.22	82.40±1.29	83.65±1.24	83.40±1.67
Resp	16.50±0.41	16.85±0.37	16.55±0.32	16.70±0.35	17.05±0.35	16.75±0.33
SBP	128.25±2.09	127.25±2.26	125.75±2.18	128.75±2.23	128.50±2.35	126.50±2.26
DBP	79.7±1.05	78.70±1.68	77.95±1.29	79.70±1.10	79.20±1.60	78.05±1.44
Hb	12.75±0.24	12.75±0.26	12.82±0.24	12.87±0.23	12.72±0.24	12.77±0.23
VAS	35.25±2.25	36.45±2.15	33.80±2.20	37.75±2.35	38.23±2.85	36.23±2.75

Data was analyzed by ANOVA test Values regarded as significant value $p<0.05$.

Table: III

Intra operative parameter of different study groups

	Upper abdominal Surgery			Lower abdominal Surgery		
	Group I (N=20) Mean±SE	Group-2 (N=20) Mean±SE	Group 3 (N=20) Mean ±SE	Group I (N=20) Mean+SE	Group-2 (N=20) Mean+SE	Group 3 (N=20) Mean ±SE
Pulse	93.90±1.54	94.30±1.70	93.30±1.85	94.90±1.49	93.65±1.67	92.85±1.56
SBP	129.0±2.26	128.75±1.70	126.0±1.97	127.65±2.23	126.75±1.93	123.05±1.59
DBP	81.5±1.09	81.75±1.01	79.25±1.37	82.45±1.04	82.30±1.14	80.85±1.25
SpO2	98.25±0.16	98.40±0.16	98.45±0.15	98.55±0.15	98.75±0.14	98.60±0.19
ET CO2	40.05±0.23	40.05±0.16	39.95±0.16	38.85±0.34	39.35±0.13	39.45±0.30

No significant different between the three groups

Table-IV

Intra operative amount of fentanyl used and duration of surgery.

	Upper abdominal Surgery			Lower abdominal Surgery		
	Group I (N=20) Mean±SE	Group-2 (N=20) Mean±SE	Group 3 (N=20) Mean ±SE	Group I (N=20) Mean+SE	Group-2 (N=20) Mean+SE	Group 3 (N=20) Mean ±SE
Amount of Fentanyl used (mg)	119.75±3.77	118.50±2.99	119.50±2.98	120.0±3.20	122.0±3.27	120.75±3.16
Duration of surgery (min)	72.5±2.25	73.5±1.70	73.75±1.88	71.75±1.96	75.0±1.76	74.75±1.86

No significant difference between the three groups

Table-V

Post operative VAS of different study groups

	Upper abdominal Surgery			Lower abdominal Surgery		
	Group I (N=20) Mean±SE	Group-2 (N=20) Mean±SE	Group 3 (N=20) Mean ±SE	Group I (N=20) Mean+SE	Group-2 (N=20) Mean+SE	Group 3 (N=20) Mean ±SE
At arrival	34.50±2.88†	36.25±3.07‡	46.0±2.62	34.75±2.95†	33.50±3.14‡	43.45±2.78
30 minute	51.75±3.46	25.0±2.87***‡	35.25±2.00‡‡	45.25±3.56	24.0±2.85 ***‡	33.0±2.52‡‡
One hour	37.0±2.92	28.0±3.59*	28.0±4.38†	32.50±3.23	22.0±3.16*	24.50±3.84†
two hour	26.50±3.42*	17.0±3.06	19.0±2.87†	22.0±3.37	17.0±3.06*	18.75±3.03
Four hour	20.50±3.58	15.25±3.15*	19.50±3.10	20.50±3.58	12.75±3.19*	17.0±2.88
Eight hour	30.50±4.25	20.50±3.93*‡	26.50±3.64	28.50±3.92	18.0±3.44*	19.0±0.62†
Twelve hour	24.50±3.93	17.50±3.54*‡	20.0±3.69†	22.0±0.69	15.00±2.20*	17.50±3.23†
Eighteen hour	21.0±4.03	15.50±3.51**	18.50±3.58	19.50±3.93	11.0±2.89*‡	17.00±3.70
Twenty four hour	19.0±4.03	15.50±3.51*‡	19.0±4.28	16.50±3.99	12.50±2.89*‡	16.0±3.93

Between the Groups
‡/†/*/• P <.05 **p< .01
* group! vs. group2
†p group! vs. group3
‡p group2 vs. group3

Between the Surgical plan
UAS vs. LAS
• Gr. 1vs Gr. I
• Gr. 2 vs. Gr. 2
• Gr. 3 vs. Gr. 3

Table VI

Time of 1st pethidine requirement and total amount of pethidine requirement in different group study.

	Upper abdominal Surgery			Lower abdominal Surgery		
	Group I (N=20) Mean±SE	Group-2 (N=20) Mean±SE	Group 3 (N=20) Mean ±SE	Group I (N=20) Mean+SE	Group-2 (N=20) Mean+SE	Group 3 (N=20) Mean ±SE
Time (min)	29.75±2.54	28.25±2.74	29.60±2.50	30.85±2.63	27.25±2.90	31.25±2.26
Amount (mg)	186.50±6.66	112.75±1.24**‡	122.45±0.96	171.65±4.39	109.45±1.50**‡	120.0±1.50

Between the Groups
‡/†/*/• P <.05 **p< .01
* group! vs. group2
†p group! vs. group3
‡p group2 vs. group3

Between the Surgical plan
UAS vs. LAS
• Gr. 1vs Gr. I
• Gr. 2 vs. Gr. 2
• Gr. 3 vs. Gr. 3

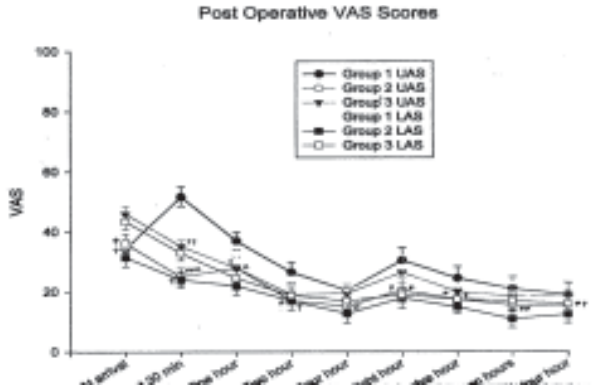

Fig.-1: *Line diagram showing the Post Operative VAS Score in different time.*

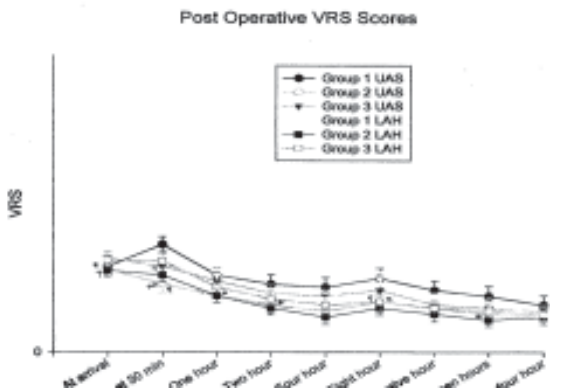

Fig.-2. *Line diagram showing the Post Operative VRS scores in different time.*

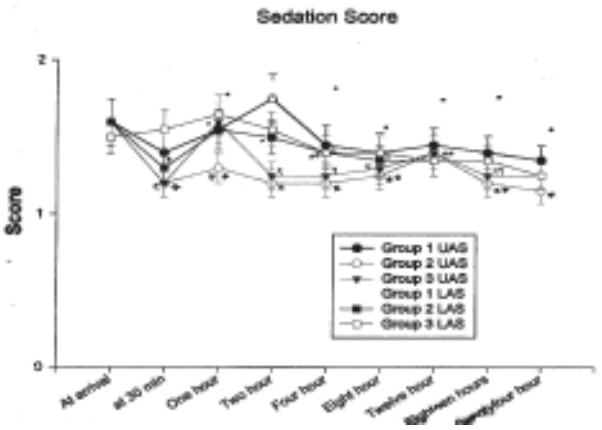

Fig.-3. *Line diagram showing the Post Operative sedation Scores in different time.*

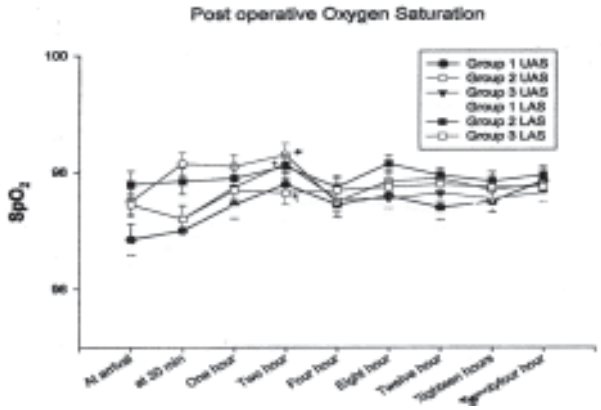

Fig.-4. *Line diagram showing the Post Operative Oxygen Saturation in different time.*

Discussion:

In this study it was found that good level of post operative analgesia (VAS within 30mm) was achieved within one in all groups having both upper and lower abdominal surgery.But excellent quality of analgesia was achieved in group II within half an hour (VAS 25 ±2.87) and continued to maintain with significant lower VAS score than other two groups. Although VAS score appeared to be higher at arrival in the post operative ward in group III but analgesia was achieved within half an hour. The similar analgesic effect was also obtained using other combination such as morphine with indomethacine, morphine with ketoralac and fentanyl with diclofenac etc. however, in those combined studies, routes of drugs administration were different most of such studies opioids were administered by PCA and NSAIDs by intra-muscularly or intra-venously or a suppository forms [7,11,12] .Results of present study was showing better analgesic effect than combination of diclofenac and pethidine or morphine and ketorolac when used by demand basis intramuscularly[14,15,16]. Lancker P et al [17] in 1996 showed that post operative pain score had decreased during the first 2 hour and had reached a lower level by 4 hours with their combined analgesic study with PCA opioid administration. But they used alfentanil as an opioid and piroxicam as an NSAIDs and administrated post operatively. The different NSAIDs and different opioids and different modes of administration used for the comparison and the different setting in which the analgesia was tested makes direct comparison difficult.[18]

Small intermittent dose of intravenous pethidine groups showed effective and safe analgesia in this study which was compared with intravenous PCA based combined analgesic studies using morphine, fentanyl, indomethacine and diclofenac.[7,11,17] Early analgesia (within 30 min) was achieved in this study with small does of intravenous pethidine which was compared with PCA based single opioid therapy[19,20,21,22] . PCA therapy is considered to be a safe but high doses of opioid was required. For this high doses or instrumental error, there was a serious adverse outcome exists[23,24,25]. Life threatening respiratory events associated with the use of PCA have reported [24,26,27,28]. These events were almost always associated with human error, usually related to pump programming major factor limiting the use of PCA other than side effects was patient factors and cost effectiveness [20.22,29,30]. There was different survey of post operative analgesic service with PCA. Semple P, Jackson IJB[31] did survey in 1991 of post-operative analgesia practice in anaesthetic departments in England and wales, PCA was considered by 58% of respondents to be the ideal method of analgesia where there was no limitation in staffing or equipment. However only 18% rated it as the safest technique on normally staffed wards. In contrast 63% of the departments felt on demand I/M analgesia was the safest form of analgesia. PCA technique are offered to fewer than 30% of patients either in North America or in Europe[32]. Present combined technique with small intermittent intravenous dose of pethidine provides excellent level of post-operative analgesia without respiratory depression or severe hypoxeamia. So this pain relief technique was safe and cost effective

compared to PCA based combined analgesia. PCA therapy provides improved analgesia compared with 'as needed intramuscular' opioid administration in patients undergoing a variety of surgical procedures [19,33,34,35]. Present study showed that small intermittent intravenous dose of pethidine provides improved and better analgesia compared with intramuscular pethidine administration (controlled group) in-patient undergoing upper and lower abdominal surgery. Mean VAS of intramuscular pethidine group varied from 19.0±4.03 to 51.75±3.46 (SE) (UAS) and 16.50±3.99 to 45.25±3.56 (SE) (LAS). mean value of intravenous pethidine group varied from 15.25±3.15 to 36.25±3.07 (SE) (UAS) and 11.0±2.89 to 33.52±3.14 (SE) (LAS).

Good analgesia was achieved after one hour in intramuscular pethidine based combined group in this study (VAS 26.50±3.42 & 22.0±3.37 at 2 hour) Iqbal KM et al 1986 [16] demonstrated that analgesia started after one hour and good analgesia was achieved after three hour of postoperative period by using combined intramuscular pethidine and declofenac study (VAS 20.5±1.9 SD). Onset of action and level of analgesia of these study was differed from my study because they used both drugs intramuscularly and post operatively.

In finding of the present study in combined analgesic method showed that the marked reduction of the pethidine dose specially in small intermittent intravenous group can maintain good postoperative analgesia with maintaining good post-operative sedation and respiratory pattern.

Incidence of nausea and vomiting in all groups was similar and it was varied from 40% to 50% and 35% to 45% respectively but frequency of vomiting was more in intrramuscular pethidine group. This finding was due to every patient gets metronidazol post-operatively. Anti-emetic requirement was more in-group I. This finding similar or less than the other same study.

Conclusion:

The combination of pre and post operative application of rectal suppository of diclofenac sodium and post-operative small intravenous intermittent dose of pethidine is an efficient method of treating post-operative pain. It is also effective and better alternative method of intramuscular or PCA based pethidine administration.

References:

1. Mamie C, Kloptenstein CE et al. Comparison of three standard analgesic regimens on postoperative pain and satisfaction. Br. J. Anaesthesia 1996; 70; 130.
2. Atkinson RS, Rushman GB, Davies NJH. Acute pain. Lee's Synopsis of Anaesthesia. 11th edn. Butterworth – Heinemann 1993; 750.
3. Yeager MP, Glass DD et al. Epidural anaesthesia and analgesia in high risk surgical patients. Anaesthesiology 1987; 66: 729-736.
4. Morgan G E, Mikhail MS. pain Management. Clinical Anaesthesiology 2nd ed. Stamford, Appleton and Lange, 1996; 274 - 316.
5. Bonica JJ. Pain research and therapy: past and current status and future needs. In: Ng LKY, Bonica JJ eds. Pain, discomfort and humanitarian care. Amsterdam: Elsevier / North Holland 1980: 1-46.
6. Bonica JJ, Loeser JD. History of Pain conceptes and Therapies. In: Loeser J D eds. Bonica's Management of pain 3rd ed. Lippincott William and Wilkins; 2001; 3-16.
7. Laitinen JL, Nuutinen L. Intravenous diclofenac coupled with PCA fentanyl for pain relief after total hip replacement. Anaesthesiology 1992; 76: 194-198.
8. Acute pain management operative or medical procedures and trauma: Clinical practice guidelines AHCPR pub No. Agency for health Care policy and Research, Public – health Service, US Department of health and Human Services, Rockville, MD. 1992;92;32
9. Rorarius MGF, Suominen P et al. Diclofenac and ketoprofen for pain treatment after elective cesarean section. Br. J. Anaesthesia. 1993; 70: 293 – 297.
10. O'Hanlon J, Miligan K et al. A comparison f the effect of intramuscular diclofenac, ketorolac or piroxicam on postoperative pain following arthroscopy. Br. J. Anaesth. 1996; 70: 132.
11. Sims C, Johnson CM et al. Rectal indomethacin for analgesia after Appendicectomy in Children. Anaesthesia Intensive Care 1994; 22: 272-275.
12. Murphy DF, Medley C. preoperative indomethacin for pain relief after thoracotomy. Comparison with postoperative indomethacin. Br. J. Aanaesthesia 1993; 70: 298-300.

13. Van P, Lancker J et al. Systemic piroxicum as an adjunct to patient controlled analgesia with altentanil for postoperative pain relief. Anaesthesia 1996; 51: 658-662.

14. Cataldo PA, Senagore AJ, Kilbride MJ. Ketorolac and patient controlled analgesia in the treatment of post-operative pain. Surg Gyenocol Obstet 1993; 176: 435-438.

15. Kenny GN McArdle CS, Aitken HH. Parenteral Ketorolac: opiate sparing effect and lack of cardiorespiratory depression in the perioperative patient. Pharmacotherapy 1990; 10: 127 – 131.

16. Iqbal KM, Gour KB et al. Assessment of post operative analgesia: A comparative study of Pethidine and Diclofenac sodium. Journal of Bangladesh College of Physicians and Surgeons. 1986; 4 (1): 1 – 7.

17. Lancker PV et al. Systemic piroxicam as an adjunct to patient controlled analgesia with alfentanil for postoperative pain relief. Anaesthesia 1996; 51: 658 – 662.

18. Turner GA, Anson N, Williamson R. Comparison of intramascular Ketorolac with Indomethacin suppositories in the treatment of pain after oral surgery. Anaesth Intens Care 1996; 24: 665 – 668.

19. Bolish SJ, Collins CL et al. Efficacy of patients controlled versus conventional analgesia for post-operative pain. Clin. Pharm. 1985; 4: 48 – 52.

20. Ferrante FM, Orav EJ et al. A statistical model for pain in patient controlled analgesia and conventional intramuscular opioid regimens. Anasth Analg 1988; 67: 457 – 461.

21. White PF. patient controlled analgesia: a new approach to the management of post operative pain. Semin Anaesth 1985; I.V: 255 – 266.

22. Jamison R, Taft K et al. Psychological and pharmacologic predictors of satisfaction with intravenous patient controlled analgesia. Anaesth Analg 1988; 67: 121-125.

23. Kreitzer JM, Kirschenbaum LP Eisenkraft JB. Safety of PCA devices. Anaesthesiology 1989, 70: 881.

24. Thomas DW, Owen H. patient-controlled analgesia: the need for caution. A case report and review of adverse incidents. Anaesthesia 1988; 43: 770 – 772.

25. White PF, parker RK. Is the risk of using a "basal" infusion with patient controlled analgesia therapy justified (letter). Anaesthesiology 1992; 76:489.

26. Ngan Kee, Warwick D et al. Respiratory depression associated with patient controlled analgesia. Canadian Journal of Anaesthesia 1995; 42 (10): 953.

27. Etches RC. Respiratory depression associated with patient-controlled analgesia: a review of eight cases. CJA 1994; 41(2): 125-132.

28. White PF. Mishaps with patient-controlled analgesia. Anaesthesiology 1987; 66: 81-83.

29. Thomas T, Robinson C et al. Prediction and assessment of the severity of postoperative pain and of satisfaction with management. pain 1998; 75: 177-185.

30. Magnani B, Johnson LR, Ferrante M. Modifiers of patient-controlled analgesia efficacy II. Chronic pain. Pain 1989; 39:23-29.

31. Semple P, Jackson IJB. Postoperative pain control. A survey of current practice. Anaesthesia 1991; 46: 1074-1076.

32. Rawal N eds. Regional opioids in anaesthesiology and pain management. Boston: Little, Brown and Company, 1986; 1-15.

33. Eisenach JC, Grice SC, Dewan DM. Patient controlled analgesia following cesarean section: a comparison with epidural and intramuscular narcotics. Anaesthesiology 1988; 64: 444-448.

34. McIntosh D, Rayburn W. Patient-controlled analgesia in obstetrics andgynecology. Obstet Gynaecol 1991; 78: 1129-1135.

35. Webb, Sterigios D, Rodgers B. Patient-controlled analgesia as postoperative pain treatment for children. J Pediatr nurs 1898; 4: 162-171.

36. Doyle E, Byers G, McNicol LR, Morton NS. Prevention of postoperative nausea and vomiting with transdermal hyoscine in children using patient-controlled analgesia. Br. J. Anaesth. 1994; 72: 72-76.

Event in 2010 Anaesthesia Pain and ICU

MONTREAL 2010 13TH WORLD CONGRESS ON PAIN
August 29 – September 2, 2010
International Association for the Study of Pain
WWW.iasp-pain.org/montreal

7TH ANNUAL CRITICAL CARE SYMPOSIUM
Ramada Piceadilly Hotel, Manchester, UK,
United Kingdom
April 22-23, 2010
Critcaresymposium.co.uk

3RD INTERNATINAL CONGRESS ON NEUROPATHIC PAIN (NEUPSIG 2010)
Megaron Athens International Conference Center
MALCC, Athens, Greece
May 27-30, 2010

14TH WORLD SOCIETY OF PAIN CLINICIANS CONGRESS (WSPC 2010)
Beijing, China
October 28-31, 2010
E-mail: wspc@kenes.com;
"Web site: http://www.kenes.com/WSPC

INTERNATIONAL CRITICAL CARE SYMPOSIUM
Chennai Trade Centre, Chennai, India,
October23-30, 2010
Eimail: ct.veerappan@gmail.com
Website : http://www.critcaresymposiumco.uk