

VOLUME 23
NUMBER 1
JANUARY 2010

Journal
of the
Bangladesh
Society of
Anaesthesiologists



JOURNAL OF THE BANGLADESH SOCIETY OF ANAESTHESIOLOGISTS

VOL 23, NO. 1, JANUARY 2010

EDITORIAL BOARD

Editor - in - Chief	: Prof. KM Iqbal
Executive Editor	: Dr. Md Abdul Hye
Assistant Editor	: Dr. Paresh Chandra Sarker
Members	: Prof. SN Samad Choudhury Prof. M Khalilur Rahman Prof. AKM Shafiqur Rahman Prof. SM Jahangir Prof. AYF Elahi Chowdhury Prof. UH Shahera Khatun Prof. Shamsul Alam Prof. Munirul Islam Prof. Latifur Rahman Prof. Shahidul Islam Prof. Syed Golam Moula Prof. Kamal Ibrahim Prof. ABM Muksudul Alam Prof. AKM Nur Nobil Chowdhury

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 - 23

NUMBER - 1

JANUARY 2010

CONTENTS

Editorial

- Beware of randomly used opioid ! 1
Prof. Debabrata Banik

Original Articles

- Evaluation of intubation condition following administration of rocuronium in comparison to atracurium in paediatric patients 3
Abdul Kuddus Khan, MM Abdul Wadud, Azizul Gafur, Rafayetullah Siddique, Debabrata Banik
- Caudal bupivacaine - midazolam for post operative analgesia in children 8
Idris Ali, Amirul Islam, Golam Morshed, Nurul Islam, Ashia Ali, UH Shahera Khatun
- Epidural triamcinolone for management of low back pain with radiation- a comparative study of two dose regime 14
Md. Mustafa Kamal, Abdullah Al Maruf, Md. Shahadat Hossain, Md Abdus Samad Md. Hasanuzzaman, Akhtaruzzaman AKM
- Monitored anaesthesia care in elderly patient - a prospective descriptive study 19
Abdullah Al Maruf, Iqbal Hossain Chowdhury, Kazi Ashkar Latif, Md. Mustafa Kamal
- Effect of preemptive intravenous ketorolac and bupivacaine infiltration on post operative pethidine consumption - a comparative study 25
Jalal Uddin Ahmed, Md Shahidul Islam, Md Shafiqul Islam, Mantosh Kumar Mandal, Mohammad Sirajul Islam, Md. Shamsuzzoha, Akhtaruzzaman AKM

Review Article

- Nutritional Support to Critically Ill Patient 30
Iqbal Hossain Chowdhury

Case Reports

- Rarest of rare bombay blood group in bangladesh: a case report 34
Tahmina Banu, S M Ali
- Unintentional epidural catheter migration to subarachnoid space followed by continuous spinal anaesthesia: a case report 37
K Sardar, AKMN Chowdhury, MK Rahman

Beware of randomly used opioid !

Opioid is an ancient pain killer. The Greek philosopher Theophrastus was first used an opioid as a pain killer in the third century. Till now it is randomly used in acute, chronic and cancer pain management. There are various opioids (eg- fentanyl, ramifentanyl etc.) continue to be manufactured those share the same basic properties as morphine and pethidine, but at much greater cost. Because of prolong use of opioid , some adverse situation inevitably developed, among them tolerance and hyperalgesia are really a problematic. Opioid induced hyperalgesia (OIH) is a clinical challenge now. OIH is a paradoxical response to an opioid agonist, whereby instead of an analgesic or antinociceptive effect occurring, there is an increased in pain perception. This may occur in the area of the pain being treated or may be a more generalized increased in pain, often with features associated with neuroapthic pain such as hyperalgesia or allodynia. Though, clinically OIH and tolerance are overlapped with each other but in case of OIH, pain will increase with doses paradoxically and in case of tolerance is not.

Basic science studies are beginning to clarify some of the contributory mechanisms, many of which are similar to those that underlie the development of tolerance.¹ From laboratory models of OIH, it is clear that as with many chronic pain states, there are both peripheral and central changes in nociceptive processing. Alteration in the spinal cord are important, with some form of central sensitization occurring. This is likely to involve the ionotropic glutamate receptor, the *N*-methyl-D-aspartate (NMDA) receptor, known to play a key role in central sensitization . C-fibre potentiation has been demonstrated similar to that seen with central sensitization in chronic opioid administration. This presented with NMDA receptor block and a range of studies have demonstrated the efficacy of NMDA receptor antagonist in preventing OIH.^{2, 3} Spinal removes in culture show increased NMDA receptor activity after chronic morphine administration, also seen acutely with remifentanyl or a dymorphin agonist.⁴ Further evidence for the involvement of glutamate

comes from work using gabapentin , which has a presynaptic effect on glutamate release and dose dependently decrease OIH from repeat fentanyl in rats.⁵

Modulation of spinal input by descending pathways from the brainstem is also implicated in the development of OIH, with a shift in the balance between descending inhibitory control towards pronociceptive system. These pronociceptive system may be more active in certain chronic pain states and also seems to play a role in OIH, acting via 5-HT₃ and possibly 5-HT₂ receptors. Ondansetron a widely used 5-HT₃ antagonist blocks signs of OIH.

Peripheral receptors also play a role in OIH , with evidence that the transient receptor potential (TRP)-V1 is important in the development of hyperalgesia. A TRPV1 antagonist was found to reverse OIH, with an associated increased in TRPV1 in the dorsal root ganglia and an increased response to capsaicin. TRPV1 knockout mice did not develop either tactile or thermal hypersensitivity to chronic morphine administration.¹⁰ Alteration in cytokine levels has also been detected in the periphery in mice with OIH, where higher levels of IL-1beta, IL-6, G-CSF, KC, and TNF-alpha were found along with increased mechanical sensitivity.⁶

Intracellular mechanism share some similarities with opioid tolerance. In that blocking L-type calcium channels or using PKC antagonist prevent or reduce OIH. Nitric oxide synthase (NOS) knockout mice show much reduced development of OIH and NOS inhibitors preventing development of OIH.³

Genetic factors are also likely to play a role in susceptibility to OIH. A clinical study of 43 healthy volunteers using a painful thermal stimulus found that individuals homozygous for the met (158) polymorphism of catechol O-methyl transferase gene had greater pain sensitivity after a potent parenteral opioid.⁷

Reviewing studies of the clinical syndrome of OIH has highlighted the lack of good quality clinical research in this area, despite the fairly extensive basic science evidence.

Further research is needed to define the clinical problem, in order to develop clinical strategies to reduce OIH. Likely targets would include agents that act on glutaminergic system, such as NMDA receptors antagonists or gabapentin, and also using agents to target peripheral effects, such as non steroidal anti-inflammatory drugs, or more novel agents, such as TRPV1 antagonists. Given the complex nature of the problem and the multiple factors likely to be involved, including genetic influences, dose, duration, type and route of administration of opioid, along with the effect of the type of pain being treated, clinical research will need to be appropriately targeted to produce meaningful results.

A small number of studies have looked at the clinical characteristics of OIH patients with chronic pain on strong opioids. Both opioid dose and duration of treatment seem to be important factors affecting descending inhibitory control and also pain and unpleasantness to a defined noxious stimulus particularly in females.^{9,10}

In summary, it is simply defined that a paradoxical increase in pain as a result of opioid administration in OIH but in practiced the situation is much more complex either clinically or neurobiologically. Though there are many evidences in basic science studies in favour of OIH but in clinical studies still to small. Besides, studies need to be designed to differentiate between acute tolerance and OIH. Now a days, we are anxious due to misuse and abuse of opioid in our country. As an anaesthesiologist we faced some problem in perioperative management of patient specially in anaesthetic requirement.

(Journal of BSA, 2010; 23(1): 1-2)

Prof. Debabrata Banik

Dept. of Anaesthesia, Analgesia and Intensive Care Medicine
BSMMU, Shahbag, Dhaka, Bangladesh

References:

1. Dietis N, Guerrini R, Calo G, Salvadori S, Rowbotham DJ, Lambert DG. Simultaneous targeting of multiple opioid receptors a strategy to improve side-effect profile. *Br J Anaes* 2009; 103: 38-49.
2. Haugan F, Rygh LJ, Tjolsen A. Ketamine blocks enhancement of spinal long-term potentiation in chronic opioid treated rats. *Acta Anaesthsiol Scand* 2008; 52: 681-7.
3. Li X, Angst MS, Clark JD. A murine model of opioid-induced hyperalgesia. *Brain Res Mol Brain Res* 2001; 86: 56-62.
4. Zhao M, Joo DT. Enhancement of spinal N-methyl-D-aspartate receptor function by remifentanyl action at delta-opioid receptors as a mechanism for acute opioid-induced hyperalgesia or tolerance. *Anesthesiology* 2008;109: 308-17.
5. Van Elstraete AC, Sitbon P, Mazoit JX, Benhamou D. Gabapentin prevents delayed and long-lasting hyperalgesia induced by fentanyl in rats. *Anesthesiology* 2008;108: 484-94.
6. Waxman AR, Arout C, Caldwell M, Dahan A, Kest B. Acute and chronic fentanyl administration causes hyperalgesia independently of opioid receptor active in mice. *Neuroscience Lett* 2009; 462: 68-72.
7. Wang HY, Friedman E, Olmstead MC, Burns LH. Ultra-low-dose naloxone suppresses opioid tolerance, dependence and associated changes in mu opioid receptor-G protein coupling and Gbetagamma signaling. *Neuroscience* 2005;135:247-61.
8. Bannister K, Bee LA, Dickenson AH. Preclinical and early clinical investigations related to monoaminergic pain modulation. *Neurotherapeutics* 2009; 6: 703-12.
9. Vardanyan A, Wang R, Vanderah TW, et al. TRPV1 receptor in expression of opioid-induced hyperalgesia. *J Pain* 2009;10: 243-52.
10. Lambert DG. Capsaicin receptor antagonists: a promising new addition to the pain clinic. *Br J Anaes* 2009;102: 153-5.

Evaluation of intubation condition following administration of rocuronium in comparison to atracurium in paediatric patients

Abdul Kuddus Khan^{1*}, MM Abdul Wadud², Azizul Gafur³, Rafayetullah Siddique⁴, Debabrata Banik⁵

¹Assistant Professor, Department of Anaesthesiology, NICVD, Dhaka; ²Assistant Registrar, Department of Anaesthesiology, NICVD, Dhaka; ³Assistant Professor, Department of Anaesthesiology, Jahurul Islam Medical College & Hospital, Bajitpur, Kishoregonj, ⁴Assistant Professor, Dept. of Anaesthesia, Analgesia and Intensive Care Medicine, Bangabandhu Sheikh Mujib Medical University, Shahbag, Dhaka; ⁵Professor, Department of Anaesthesia, Analgesia and Intensive Care Medicine, Bangabandhu Sheikh Mujib Medical University, Shahbag, Dhaka.

*Corresponding author: drak_khannicvd@yahoo.com

Abstract

Background. Optimum intubation condition is paramount important for early and easy passage of endotracheal tube through the glottis and that results from adequate muscle relaxation. Rocuronium and atracurium are muscle relaxants used for short and intermediate duration of surgical processes.

Objective: This study, compares the intubation condition and haemodynamic changes in paediatric patients following administration of rocuronium and atracurium to get rid of the side effects of succinylcholine.

Method. The study was carried out in 60 patients aged 1¹/₂ to 10 years, ASA I-II, under general anaesthesia. The patients were divided into two groups: rocuronium Gr R and atracurium Gr A. Induction was done with halothane 3.5-4% and for intubation rocuronium 0.6 mg/kg and atracurium 0.46 mg/kg were given to patients of Gr R and Gr A respectively. The intubation condition was assessed and graded at 60 seconds after neuromuscular blocking agents. At the same time TOF ratios were recorded.

Results. Rocuronium produced good to excellent intubation condition (score 3.80 ± 0.07) in all patients at 60 seconds whereas that of atracurium poor intubation condition (score 2 ± 0.00). TOF ratios showed more relaxation of adductor pollicis muscle in Gr R than Gr A (Gr R = 60.43 ± 0.87 , Gr A = 78.90 ± 0.72). ($p < 0.001$). Cardiovascular stability was not significantly difference in both the groups before induction and intubation.

Conclusion. Rocuronium produced better intubation condition in comparison to atracurium.

Keywords: Paediatric patients, intubation, rocuronium, atracurium

(Journal of BSA, 2010; 23(1): 3-7)

Introduction

In anaesthetic practice muscle relaxation is used to serve two prime purposes; one, to facilitate endotracheal intubation and the other to provide surgical relaxation¹. Adequate muscle relaxation plays an important role in the concept of balanced anaesthesia² and it demands for intubations to maintain artificial ventilation. Rapid sequence intubation is very important for paediatric patients because of their high metabolic rate, less functional residual capacity and more O₂ requirement, during which there is a chance of

hypoxia³. Children are more susceptible than adult to cardiac arrhythmias, hyperkalaemia, rhabdomyolysis, myoglobinemia, masseter spasm and malignant hyperthermia after succinylcholine administration. Unlike in adult patients, profound bradycardia and sinus node arrest develop in paediatric patients following the first dose of succinylcholine without atropine pretreatment⁴.

Atracurium was developed in an attempt to obtain a non-depolarizing agent which had a more rapid onset, was shorter acting and had less

cardiovascular effects than did the older agents but may release histamine and may be accompanied by a slight fall in arterial pressure⁵. Good to excellent intubating conditions after rocuronium 0.6mg/kg obtained within 60 seconds when compared with vecuronium and atracurium in young children with intravenous anaesthetic agents⁶. Aleksandra J. Mazurek et al.1998 found that rocuronium (1.2 mg/kg) can be substituted for succinylcholine during rapid sequence intubation in paediatric patients with intravenous anaesthetic agents⁷.The remarkable advantage of rocuronium is that one drug is used for intubation and maintenance of anaesthesia. In spite of these advantages, this drug is not used in our daily practice. So, this study was done to determine and compare the intubation condition following administration of rocuronium and atracurium in paediatric patients combined with halothane induction with the aim of good to excellent intubation condition and reduction of intubation time.

Subjects and methods

This randomized prospective clinical study was carried out on sixty patients of both sex, aged between 1½ to 10 years for operations of an average duration of ½ to 2 hours.

This study was approved by the ethical committee of the University and written informed consent was obtained from patients undergoing variety of surgical procedures requiring general anaesthesia. Patients were excluded if overweight, if they or their family had a history of neuromuscular diseases and if they received antibiotics before anaesthesia. Induction was done with halothane 3.5-4% and at the same time venous access was secured. Fentanyl was used 1.5 µgm/kg in both the groups. Rocuronium 0.6 mg/kg and atracurium 0.46 mg/kg were given to patients of Gr R and Gr A respectively. Laryngoscopy was performed and intubation conditions were assessed and graded at 60 seconds after neuromuscular blocking agents using the 4 step scale proposed by Goldberg and his colleagues scoring system⁸ (4= excellent [easy passage of the tracheal tube without coughing, vocal cord relaxed], 3= good [slight coughing, vocal cord relaxed], 2 = poor [passage of the tracheal tube with moderate coughing, some movement of the vocal cord], and 1= impossible), and at the same

time TOF ratios were recorded. TOF ratio indicates the degree of neuromuscular block. In the event of unsuccessful intubation ventilation was continued for another 30- 45 sec until the next attempt was made. Pulse, mean arterial pressure, oxygen saturation were recorded just before induction, before intubation and after intubation. The results were compiled and analyzed statistically using unpaired Student's 't' test. P < 0.05 was considered as significant (by using SPSS version-12 software).

Observation and Results

There were no significant differences in the patients characteristics including age, weight and sex (Table I). Rocuronium produced 20% good and 80% excellent intubation condition. But atracurium produced poor intubation condition in all patients (Table II).

The mean ± SEM values of intubation scores at 60 seconds were 3.80 ± 0.07 and 2 ± 0.00 in Gr R and Gr A respectively. They showed highly significant difference between the two groups (Table II). ($p < 0.001$).

The mean ± SEM values of train of four ratio at 60 seconds were 60.43 ± 0.87 and 78.90 ± 0.72 in Gr R and Gr A respectively. They showed highly significant difference between the two groups (Table II). ($p < 0.001$).

The mean ± SEM values of heart rate, mean arterial pressures and oxygen saturation before induction were 103.33 ± 3.29 , 78.03 ± 0.64 and 99.33 ± 0.09 in Gr R and 97.13 ± 0.96 , 78.53 ± 0.59 and 99.37 ± 0.09 in Gr A respectively. They showed no significant difference between the two groups (Fig.1,2,3 and Table-III).

The mean ± SEM values of heart rate, mean arterial pressures and oxygen saturation before intubation (when eye-lash reflex was lost), were 88.83 ± 1.00 , 74.03 ± 0.60 and 99.40 ± 0.09 in Gr R and 86.80 ± 1.06 , 72.90 ± 0.56 and 99.37 ± 0.09 in Gr A respectively. They showed no significant difference between the two groups (Fig.1,2,3 and Table-III).

The mean ± SEM values of heart rate and mean arterial pressures after intubation were 103.33 ± 1.37 and 78.47 ± 0.65 in Gr R and 115.80 ± 1.53 and 74.93 ± 0.54 in Gr A respectively. They showed

highly significant difference between the two groups (Fig.1,2 and Table-III). ($p < 0.001$).

The mean \pm SEM values of oxygen saturation after intubation were 99.47 ± 0.09 and 99.40 ± 0.09 in Gr R and Gr A respectively. They showed no

significant difference between the two groups (Figure-3 Table-III). After discreet scrutiny it is revealed that before intubation oxygen saturation was a little bit less than after intubation in Gr A and it was due to poor intubation condition.

Table-I
Demographic characteristics of patients.

	Age (yrs)	Weight (kg)	Sex	
			Male	Female
Gr R (n=30)	5.20 ± 0.48	16.13 ± 1.03	19 (63.3%)	11 (36.7%)
Gr A (n=30)	5.55 ± 0.41	16.17 ± 0.83	18 (60.0%)	12 (40.0%)
t-value	0.551	0.025	37 (61.7%)	23 (38.3%)
p-value	0.584	0.980		

Values were expressed as Mean \pm SEM, values are regarded significant if < 0.05 .

Table II
Intubation Scores, Train of four ratios and intubation condition recorded at 60 seconds

	Intubation scores	Train of four ratios (T4/T1)	Intubation condition		
			Poor	Good	Excellent
Gr R (n=30)	3.80 ± 0.07	60.43 ± 0.87		20% = 6 patients	80% = 24 patients
Gr A (n=30)	2 ± 0.00	78.90 ± 0.72	100% = 30 patients		
t-value	24.233	16.408			
p-value	0.001	0.001			

Values were expressed as Mean \pm SEM, $P < 0.001$ was considered as highly significant

Table III
Heart Rate, MAP (mm of Hg) and SpO₂ %

	Before induction			Before intubation			After intubation		
	Heart rate	MAP	SpO ₂	Heart rate	MAP	SpO ₂	Heart rate	MAP	SpO ₂
(Gr R) (n=30)	103.3 ± 3.29	78.03 ± 0.64	99.33 ± 0.09	88.83 ± 1.00	74.03 ± 0.60	99.40 ± 0.09	103.33 ± 1.37	78.47 ± 0.65	99.47 ± 0.09
(Gr A) (n=30)	97.13 ± 0.96	78.53 ± 0.59	99.37 ± 0.09	86.80 ± 1.06	72.90 ± 0.56	99.37 ± 0.09	115.80 ± 1.53	74.93 ± 0.54	99.40 ± 0.09
t-value	1.811	0.574	0.266	1.398	1.386	0.261	6.065	4.174	0.513
p-value	0.075	0.568	0.791	0.167	0.171	0.795	0.001	0.001	0.610

Values were expressed as Mean \pm SEM, $P < 0.001$ was considered as highly significant.

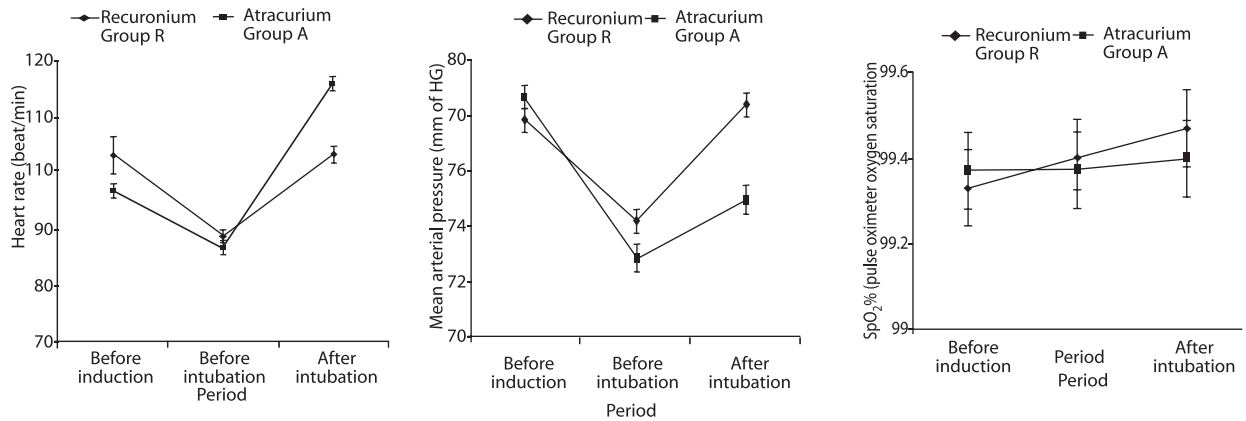


Fig.-1 : Changes of heart rate

Fig.-2 : Changes in mean arterial pressure

Fig.-3 : Changes in oxygen saturation

Discussion

Early intubation is a very essential part of general anaesthesia particularly in paediatric patients. Rocuronium is popular in adult patients but clinical experience is less in our country in paediatric patients.

Intubation was done at 60 seconds following administration of rocuronium 0.6 mg/kg and atracurium 0.46 mg/kg, with 3.5-4% halothane as an induction agent in 50% nitrous oxide and oxygen.

Rocuronium had shown 20% good and 80% excellent intubation conditions at 60 seconds whereas that of atracurium poor intubation condition in all patients.

Our study is supported by M. Bock et al. 2000⁹, Zhou et al. 2000¹⁰, Fuschs-Buder T and Tassonyi E, 1996¹¹ and M. Eikermann et al. 2000¹².

The mean \pm SEM values of train of four ratio at 60 seconds were 60.43 ± 0.87 and 78.90 ± 0.72 in Gr R and Gr A respectively. They showed highly significant difference between the two groups ($p < 0.001$).

This result indicates that in our study the neuromuscular blockade at 60 seconds at the adductor pollicis muscle after rocuronium was greater than that of atracurium. TOF ratios are also comparable to intubation grading system. So, TOF ratios quantitatively supported the intubation condition in this study.

Mogorian et al. 1993 and De Mey JC 1994, found in adults that the onset of neuromuscular blockade at the adductor pollicis muscle after rocuronium is more rapid compared with atracurium¹³. This finding is also comparable to present study.

Before induction and before intubation there was no significant difference between Gr R and Gr A in terms of heart rate, mean arterial pressure and oxygen saturation. The mean \pm SEM values of heart rate and mean arterial pressures after intubation were 103.33 ± 1.37 and 78.47 ± 0.65 in Gr R and 115.80 ± 1.53 and 74.93 ± 0.54 in Gr A respectively. They showed highly significant difference between the two groups ($p < 0.001$). The above findings are of rise of heart rate and fall of mean arterial pressure in the atracurium group and slight rise of heart rate, almost no change in mean arterial pressure in rocuronium group just after intubation. In case of atracurium increases of heart rate may be due to poor intubation condition¹⁴ and fall of blood pressure may be due to histamine release¹⁵.

It is also supported by Samia Elbaradie, 2004, and reported that following administration of 0.6mg/kg atracurium resulted with the decrease in mean arterial pressure and increase in heart rate¹⁶.

Conclusion

Our findings suggest that rocuronium is very much effective and produced good to excellent intubation condition at 60 seconds in paediatric patients.

References

- Misra MN, Agarwal M, Pandey RP, Gupata A. A Comparative Study of Rocuronium, Vecuronium and Succinyl Choline for rapid sequence induction of anaesthesia. Indian J. Anaesth 2005; 49 (6): 469-473.
- Foldes FF, Nagashima H, Nguyen HD, et al. The neuromuscular effects of ORG 9426 in patients receiving balanced anaesthesia. Anesthesiology 1991; 75: 191-6.

3. Nunn JF. Nunn's applied respiratory physiology. Oxford: Butterworth-Heinemann Ltd. 1993. P
4. Morgan GE, Mikhail MS, Murray MJ Paediatric anaesthesia. Clinical Anesthesiology 3rd edition. New York: The Mc Graw-Hill, 2002; 849-858.
5. Aitkenhead AR, Rowbotham DJ, Smith G. 'Textbook of Anaesthesia'. Fourth Edition, New York: Churchill Livingstone; 2001.
6. Gerd S, Flavio CR, Albert, et al. Intubating conditions and onset of action after rocuronium, vecuronium, and atracurium in young children anesth analg 1996; 83: 320-4.
7. Mazurek AJ, Rae B, Hann S, Kim JI, et al. Rocuronium versus succinylcholine are they equally effective during rapid sequence induction of anaesthesia? Anesth Analg 1998; 87:1259- 62.
8. Goldberg ME, Larijani GE, Azad SS, et al. Comparison of tracheal intubating conditions and neuromuscular blocking profiles after intubating doses of mivacurium chloride or succinylcholine in surgical patients. Anesth Analg 1989; 69: 93-9.
9. Bock M, Klippel K, Nitsche B, et al. Rocuronium potency and recovery characteristics during steady-state desflurane, sevoflurane isoflurane or propofol anaesthesia. Br J Anaesth 2000; 84 (1): 43-7.
10. Zhou TJ, White PF, Chiu JW, et al. Onset/offset characteristics and intubating conditions of rapacuronium: a comparison with clinical investigation'. Br J Anaesth 2000; 85: 246-250.
11. Fuchs-Buder T, Tassonyi E. Intubating conditions and time course of action of rocuronium-induced neuromuscular block in children. Br J Anaesth 1996; 77: 335-8.
12. Eikermann M, Hunkemoller I, Peine L et al. Optimal rocuronium dose for intubation during inhalation induction with sevoflurane in children. Br J Anaesth 2002; 89 (2): 277-81.
13. Magorian T, Flannery KB, Miller RD. Comparison of rocuronium, succinylcholine and vecuronium for rapid- sequence intubation of anaesthesia in adult patients. Anesthesiology 1993; 79: 913-8.
14. Naguib M, Samarkandi AH, Bakhamees HS, et al. Histamine-release haemodynamic changes produced by rocuronium, vecuronium, mivacurium, atracurium and tubocurarine. Br J Anaesthesia 1995; 75: 588-92.
15. Savarese JJ, Ali HH, Basta SJ, et al. The cardiovascular effects of mivacurium chloride (BW B1090U) in patients receiving nitrous oxide-opiate-barbiturate anaesthesia. Anesthesiology 1989; 70: 386-394.
16. Elbaradie S. Neuromuscular efficacy and histamine-release hemodynamic changes produced by rocuronium versus atracurium: A comparative study. Journal of the Egyptian Nat. Cancer Inst., 2004; 16: 107-113.

Epidural triamcinolone for management of low back pain with radiation- a comparative study of two dose regime

**Md. Mustafa Kamal^{1*}, Abdullah Al Maruf², Md. Shahadat Hossain³, Md Abdus Samad⁴,
Md. Hasanuzzaman³, Akhtaruzzaman AKM⁵**

^{1*}Assistant Professor, Department of Anesthesia, Analgesia and Intensive Care Medicine, Bangabandhu Sheikh Mujib Medical University, Dhaka, ¹Classified Anaesthesiologist, Combined Military Hospital, Dhaka, ³Consultant, Department of Anaesthesia, Analgesia and Intensive Care Medicine, Bangabandhu Sheikh Mujib Medical University, Dhaka, ⁴Department of Anaesthesiology, BNS Patanga Naval Base, Haliahahar, Chittagong, ⁵Professor of Neuroanaesthesia, Bangabandhu Sheikh Mujib Medical University, Shahbag, Dhaka-1000, Bangladesh

**Corresponding author:* Md. Mustafa Kamal, E-mail: mili@bol-online.com

Abstract:

Background: *Low back pain is a very common specially in elderly people. Relief of pain is associated with decrease in morbidity, shorten hospital stay and increase patients' satisfaction. Nerve block by epidural steroid and local anaesthetic is an acceptable method to reduce low back pain.*

Aim and objective: *The present study was performed to compare low dose (40 mg) and high dose (80 mg) Triamcinolone to see their effectiveness and side effects in controlling low back pain.*

Method: *This study was a randomized controlled trial. 60 patients with low back pain with radiation, and with positive CT/MRI support has been randomly selected by blind envelop method and divided into two groups, 30 patients in each. Group-A, patients received inj. Triamcinolone (40 mg) and Group-B patients received inj. Triamcinolone (80 mg) epidurally.*

Results: *Both dosage produced effective analgesia in low back pain as assessed by visual analogue scale (VAS) and verbal rating scale (VRS) for the period up to 30 days after administration. Haemodynamic changes showed no significant difference between the two drugs after 30 days. Both drugs produced significant rise of fasting plasma glucose and leucocytosis for the period up to 30 days after administration.*

Conclusion: *Low back pain can be effectively reduced by epidural low dose Triamcinolone (40 mg).*

Key Words: *Low back pain, triamcinolone, epidural*

(Journal of BSA, 2010; 23(1): 14-18)

Introduction

Back pain is an extremely common complain and a major cause of work disability worldwide.¹ Common cause of low back pain are paravertebral muscle and lumbo sacral sprain / strain, intervertebral disc disease or herniated disc, facet syndrome, congenital abnormalities, tumour, infection, arthritis². Approximately 80-90% of low back pain is due to sprain/strain associated with lifting heavy objects, falls, or sudden abnormal movement of the spine. Another important cause of low back pain is intervertebral disc disease. Intervertebral disc bear at least one third of the

weight of the spinal column. Their central portion, which is called the nucleus pulposus, is composed of gelatinous material early in life. Disc pain may be due to (1) protrusion or extrusion of the nucleus pulposus posteriorly or (2) loss of disc height resulting in the reactive formation of bony spurs from the rims of the vertebral bodies above and below the disc. Intervertebral disc disease most commonly affects the lumbar spine because it is subjected to the greatest motion and the posterior longitudinal ligaments is thinner at lumbar.²⁻⁵

Over the past two decades, the biochemical contributions to low back pain have been the focus

of much attention. In the late 1970's the nuclear material of the vertebral disc was found to be antigenic and capable of producing an in vitro autoimmune reaction. It was hypothesized that a chemical radiculitis might explain radicular pain in the absence of a more mechanical stressor. Phospholipase A2 (PLA₂), a potent inflammatory mediator, has demonstrated to be released by discs following injury. The anti-inflammatory and immunosuppressive effects of glucocorticoids are largely secondary to their inhibition of the immune responses of lymphocytes, macrophages, and fibroblasts. NSAIDs principally inhibit prostaglandin synthesis, corticosteroids interfere earlier in the inflammatory cascade by inhibiting PLA₂ actions and thereby curtailing both the leukotriene and prostaglandin mediated inflammatory response.⁴

Low back pain management may involve the following after full pain evaluation. Simple measures e.g. rest, exercise, heat and cold therapy, systemic drug therapy, nerve blocks, electrical stimulation and psychotherapy. Epidural steroid injections are most effective for symptomatic relief of pain associated with nerve root compression. Epidural steroid injection is clearly superior to local anaesthetics alone⁴. Methyl prednisolone and recently triamcinolone are most commonly used in low back pain through epidural route.

An epidural steroid injection delivers steroids directly into the epidural space in the spine. Sometimes additional fluid (local anesthetic and/or a normal saline solution) is used to help 'flush out' inflammatory mediators from around the area that may be a source of pain.⁵

Considering the effectiveness of steroids and lack of any comparative study between 40mg and 80mg triamcinolone, present study was performed to compare these two dosing, and to see their effectiveness in decreasing low back pain and associated complications.

Methods

The present study comprised of 60 patients of low back pain with radiation and a positive CT/MRI support. The purpose of the study was clearly explained to each subject and recruited only after they had given written consent. Patients with known allergy to study drugs, with haemorrhagic diathesis, diabetes, and preexisting neurological, local skin infection were excluded. After being recruited for the study, all patients were randomly divided into two groups (30 patients each).

Group A: The patient received Inj. Triamcinolone 40 mg epidurally

Group B: The patient received Inj. Triamcinolone 80 mg epidurally

Protocol outline for epidural steroid injection [ESI]

The patients were placed in the lateral decubitus position with the involved side down. The loss of resistance technique was applied to identify the epidural space at the level of suspected or proven nerve root involvement. The patient will be warned that the injection might cause transient radicular pain.

The experimental drug was injected along with 1% lignocaine 5ml. The needle was clear of steroid with a small volume of lignocaine to avoid a fistulous track. The decubitus position was maintained for 10 minutes. Test for improvement was checked by straight leg raising test (SLR), 10 minutes after being moving the patient supine; this provides immediate feedback to the patient about the potential benefit for ESI.

Patients were told about the interval during which the local anesthetic effect wears off (30 to 90 minutes) and the steroid effect materializes (24 to 48 hours).

Haemodynamic, Blood sugar and leukocyte count were observed during and after ESI. Steroid increase blood sugar level and it has also anti-inflammatory action. So blood sugar and leukocyte count were monitored. Pain assessment was measured using visual analogue scale (VAS) and verbal rating scale (VRS).

Data were expressed as mean \pm standard error of mean (SEM). Data were managed and analysed using computer program Statistical Package for Social Science (SPSS) for Windows, version 12.0. A p value less than 0.05 were considered significant.

Results

The mean (\pm SEM) of age, sex, height and weight of the subjects (are shown in Table 1). in Group A and Group B were similar. The VAS and VRS scores show that the pain reduces significantly in each group A from the base line value up to 30 days. But when compared between the groups, the VAS scores shows a lower value in Group A at all points up to 30 days but the difference is not significant (Table-II). But in VRS scoring system Group B shows a significant reduction of pain with low VRS scores up to 30 minutes after epidural injection (Table-III).

Table-I
Characteristics of the patients in two groups

Variables	Group A	Group B	Significance level
Age	52.70±1.67	533.5±1.95	NS
Sex (M:F)	35:15	39:11	NS
Height	164.63±1.08	164.64±0.96	NS
Weight	64.56±1.12	65.70±1.05	NS

Statistical analysis was done by student's 't' test.
Legend: NS - non-significant

No difference was seen in the heart rate of patients at different duration of the same groups. And in between groups there were no significant differences. The Systolic and diastolic blood pressure showed gradual increase after epidural administration from baseline in each group. But no difference were observed between groups A and B. In Group A, the mean (\pm SEM) fasting plasma glucose of the subjects measured at baseline, after 10 min, 30 min, 1 hour, 24 hour, 3 days, 7 days and 30 days of epidural administration of triamcinolone showed significant rise over time. In Group B, the mean (\pm SEM) fasting plasma glucose of the subjects measured at baseline, after 10 min, 30min, 1 hour, 24 hour, 3 days, 7 days and 30 days of epidural administration of triamcinolone also showed significant rise (Table-IV). But when compared between groups, the values were a little increased in group B than in group A, but not significant. Blood leukocyte count also showed a similar result like blood glucose.

Table-II
Visual Analogue scale pain score in two groups

Time	Visual Analogue scale pain score		P value
	Group A	Group B	
Baseline	7.50±0.11	7.10±0.12	0.0158
10 minutes	6.10±0.15*	5.80±0.16*	0.1745
30 minutes	4.48±0.13*	4.40±0.14*	0.6763
1 hour	3.82±0.14*	3.87±0.13*	0.7941
24 hours	3.90±0.15*	3.80±0.12*	0.6038
3 days	4.10±0.14*	3.92±0.13*	0.2977
7 days	4.50±0.19*	4.35±0.18*	0.2545
30 days	4.93±0.19	4.72±0.15	0.3878

Statistical analysis was done by student's 't' test.

Table-III
Verbal rating scale pain score

Time	Verbal rating scale pain score		P Value
	Group A	Group B	
Baseline	2.18±0.05	2.15±0.06	0.7017
10 minutes	1.65±0.08*	1.40±0.09*	0.0405
30 minutes	1.10±0.08*	0.88±0.06*	0.0302
1 hour	0.83±0.05*	0.68±0.06*	0.0577
24 hours	0.80±0.05*	0.75±0.08*	0.5973
3 days	0.84±0.04*	0.79±0.07*	0.5366
7 days	0.93±0.09*	0.95±0.09*	0.8755
30 days	1.13±0.10*	1.10±0.09*	0.8240

Statistical analysis was done by student's 't' test.

Table-IV
Biochemical changes in two studied groups

Time	Plasma Glucose			Leukocyte		
	Group A	Group B	pvalue	Group A	Group B	Pvalue
Baseline	5.60.12	5.700.18	0.6449	80166157.13	8136.66141.29	0.5712
10 minutes	5.80.11	5.90±0.19	0.6498	8043±155.11	8130.00±139.75	0.6778
30 minutes	5.90.13	6.38±0.18	0.0331	8305.33±137.47	8413.33±139.10	0.1386
1 hour	6.12±0.14	6.70±0.12	0.0022	8353.331138.02	7930.00142.06	0.0351
24 hours	6.17±0.12	6.850.14	0.0004	8405.33±138.02	8020.00±142.06	0.0546
3 days	6.10±0.11	6.30±0.12	0.2222	8353.33}138.02	8106.66±144.94	0.2207
7 days	6.09±0.15	6.150.19	0.8048	8200.30146.22	8112.66144.94	0.6713
30 days	5.96±0.15	5970.19	0.9671	8126.66±166.91	8156.66±137.77	0.8900

Discussion

Pain is a common phenomenon that almost everyone experiences in life. Acute pain is generally a short-term illness for which specific therapy over a limited period of time can result in total elimination of the symptoms and the cause. Appropriate therapy can be provided only after the patient has been evaluated and a diagnosis determined. When pain lasts longer than 6 weeks, the physical aspects of the persisting symptoms precipitate further, physical and nonphysical consequences that became progressively devastating. Whereas in acute pain the symptoms disappear with the use of specific therapy, patients with chronic pain require perpetual coordination of all modes of therapy. Even then the practitioner must realize that just eliminating pain is not the answer to all the patient's problems. The patient must participate actively in the treatment planning and all aspects of the therapeutic program. Even with maximal therapy, some chronic pain will not disappear: The best treatment then is to help the patient understand the condition and provide coping strategies. All those involved in the care of patients with chronic low back and lower extremity pain must be sensitive to the many factors that contribute to complaints of pain. They must also be willing to step outside the boundaries of their special training and appreciate the holistic attitude that is necessary to evaluate and treat these patients.⁶

Epidural steroid injections produce significant pain relief lasting 1-6 months in patient with chronic back pain. It also produces an improvement in functional activity and a general sense of well being. It is an effective alternative in the management of chronic non malignant back pain. Their use was predicated on the reality that something other than physical compression of a nerve root by a herniated disc or entophyte was contributing to complaints of radicular pain⁶. In the present study all the patients had nerve root irritation as suggested by the MRI reports. Low dose triamcinolone found to be effective as high dose triamcinolone, as seen by VAS and VRS, but the side effects of the high dose were more.

In our study, there was no significant difference in the effectiveness of the two dosing of triamcinolone in controlling pain as seen by VAS,

but in VRS, triamcinolone showed significantly less pain for the initial 30 minutes. Corticosteroids are well known for their anti-inflammatory properties⁷, and also stabilize neural membranes, suppress ectopic neural discharges⁸, and may have direct anesthetic effect on small unmyelinated nociceptive C-fibers^{9, 10}. Painful lumbar intervertebral discs are innervated by substance-P containing nerve fibers¹¹, unmyelinated C-fibers, and thinly myelinated A_α fibers¹² that provide a substrate on which corticosteroids and local anesthetics exert therapeutic benefit.

Changes (heart rate, systolic blood pressure and diastolic blood pressure) of low dose and high dose triamcinolone administration showed no significant difference.

In the present study low dose and high dose triamcinolone both produced significant rise of plasma glucose for the period up to 30 days after administration and then returned to normal levels. The dose of depo-steroids used is not inert in the body.¹³ In some patients the depo steroids have an unexpectedly long half life and can lead to Addison's disease or Cushing's syndrome. Kay¹⁴ showed that after repeated steroid injections the endogenous steroid production takes 1-3 months to return to normal.

Both low dose and high dose triamcinolone produced leucocytosis for the period up to 7 days after administration. This may be due to the local Anaesthetic effect but it reduced after 7 days showing the anti inflammatory effect of the steroids. The changes in blood leukocyte count after administration of low dose and high dose triamcinolone showed no significant difference.

Sample size of the study could not be matched to a statistically calculated one due to limited number of cases available during the period of study. But both dosing showed its efficacy in reducing the radicular low back pain which was comparable to the largest meta-analysis study.

Conclusion:

Low back pain can be effectively reduced low dosing technique, predictable side effects on glycaemic status.

References

1. Yentis SM, Hirsch NP, Smith GB. Anesthesia and Intensive care A-Z; 2nd ed., Oxford: Butterworth; Heinemann 2000: 418.
2. Raj P. Prithvi. Low back pain and pain of the lower extremity, Practical management of pain. 2nd ed. London, Mosby Year Book. 1992; 296-311.
3. Jensen MC, Brant-Zawadzki MN, Obuchowski N, et al. Magnetic resonance imaging of the lumbar spine in people without back pain. *NEJM* 1994; 331:69-73
4. Malanga Gerard. Corticosteroids in the Treatment of Acute Low Back Pain. Available at: <http://www.spineuniverse.com/treatments/pain-management/steroids-cortisone-corticosteroids>. Accessed on 2010
5. Staehler Richard. Spine Health. Available at: <http://www.spine-health.com/treatment/injections>. on 28.03.2010.
6. Kelloy M: Pain due to pressure on nerves spinal tumors and the intervertebral disc. *Neurology* 1956; 6:32
7. Delaney TJ, Rowlingson JC, Canon H. Epidural steroid effects on nerves and meninges. *Anaesth Analg* 1980; 59: 610-614
8. Devor M, Govrin-Lippmann R, Raber P. Corticosteroids suppress ectopic neural discharge originating in experimental neuromas. *Pain* 1985;22:127-37.
9. Johansson A, Hao J, Sjolund B. Local corticosteroid application blocks transmission in normal nociceptive C-fibres. *Acta Anaesthesiol Scand* 1990;34:335-8.
10. Woodward JL, Weinstein SM. Epidural injections for the diagnosis and management of axial and radicular pain syndromes. *Phys Med Rehabil Clin N Am* 1995;6:691-714.
11. Freemont AJ, Peacock TE, Goupille P et al. Nerve ingrowth into diseased intervertebral disc in chronic back pain. *Lancet* 1997; 350: 178-81.
12. Brown MF, Hukkanen MV, McCarthy ID, et al. Sensory and sympathetic innervation of the vertebral endplate in patients with degenerative disc disease. *J Bone Joint Surg Br* 1997; 79: 147-53.
13. Spaccarilli KC. Lumbar and Caudal epidural in pain research and therapy, New York. *Mayo Clin Proc* 1996; 71: 169.
14. Kay J, Findling JW, Raff H. Epidural Triamcinolone suppresses the pituitary adrenal axis in human subjects. *Anesth Analg* 1978;78: 501
15. Walts RW, Selagy CA: A meta-analysis on the efficacy of epidural corticosteroids in the treatment of sciatica. *Anaesth Inten Care* 1995; 23: 564-9

Monitored anaesthesia care in elderly patient - a prospective descriptive study

Abdullah Al Maruf^{1*}, Kazi Ashkar Latif¹, Iqbal Hossain Chowdhury², Md. Mustafa Kamal³

*1.Graded Specialist in Anaesthesiology, Department of Anaesthesia and Intensive care, CMH, Dhaka, 2.Associate Professor, Department of Anaesthesia, Analgesia and Intensive care unit, BSMMU Dhaka 3. Assistant Professor, Department of Anaesthesia, Analgesia and Intensive care unit, BSMMU Dhaka

* *Corresponding author:* Dr. Abdullah Al Maruf, E-mail: iqbal-hossain-56@gmail.com

Abstract

Background: *In elderly patients for some diagnostic, therapeutic and surgical procedure, Monitored anaesthesia care (MAC) may be an anaesthetic option for them.*

Aim and objective: *This prospective study was designed to assess the efficacy, safety, and tolerability of MAC in elderly patients.*

Method: This study was performed on elderly patients of both sex, age from 50 years and above, scheduled to undergo different therapeutic, diagnostic and surgical procedures. The patient's characteristics, pre-anaesthetic problems, anaesthetic techniques, anaesthetic agents, anesthetic time, MAC procedure and complications were assessed.

Result: *They involved mainly hypertension (26.49%) and diabetes mellitus (21.78%). Almost all procedure was done under sedation (70.38%), local anaesthesia (22.71%) and under only monitoring without sedation or local anaesthesia (6.90%). There were no serious adverse events reported in any patients during MAC. The common complications were arrhythmia (9.60%), vomiting (7.36%) and desaturation (4.56%), hypertension (5.64%). Complications were minor, transient and promptly managed and corrected. MAC duration was ranged from 15 to 180 minutes. The mean procedure time was 35.1 ± 12.5 minutes. Majority of cases were completed within 30 minutes (51.21%).*

Conclusion: *In properly selected elderly patients, MAC is a safe and effective method of providing intra-operative care for some common diagnostic, therapeutic, and operative procedures.*

Keywords: *Monitored anaesthesia care (MAC), elderly, sedation, monitoring.*

(Journal of BSA, 2010; 23(1): 19-24)

Introduction

Monitored Anaesthesia Care (MAC) is a specific anaesthesia service in which an anaesthesiologist has been requested to participate in the care of a patient undergoing a diagnostic or therapeutic procedure. Indication for monitored anaesthesia care includes the nature of the procedure, the patient's clinical condition and the potential need to convert to a general or regional anaesthesia.¹ MAC includes all aspects of anaesthesia care; a pre-procedure visit, intra-procedure care and post procedure anaesthesia management. During MAC, the anaesthesiologist directs a number of specific services, including but not limited to; (a) diagnosis

and treatment of clinical problems those occur during the procedure, (b) support of vital functions, (c) administration of sedatives, analgesics, hypnotics, anaesthetic agents or other medications as necessary for patients safety, (d) psychological support and physical comfort, and (e) provision of other medical services as needed to complete the procedure safely. MAC may include varying levels of sedation, analgesia and anxiolysis as necessary. The provider of MAC must be prepared and qualified to convert general anaesthesia when necessary. If the patient loses consciousness and the ability to respond purposefully, the anaesthesia care is a general anaesthetic, irrespective of whether airway instrumentation is required or not.

As the number of elderly patients undergoing surgery continues to rise, it is important to consider anaesthetic options that minimize physiological stress in patients. MAC, sedation and monitoring are an attractive option for certain common procedures.² However, those anaesthesiologists administering MAC must consider the normal functional reserve in elderly patients.³ These include (a) loss of normal compensation for the stress of hypovolaemia, (b) decreased peripheral vascular resistance, (c) altered mental status and, (e) reduced response to hypoxia and hypercarbia associated with peri-procedure and sedated state in this population. Because of changes in body composition, as well as renal and hepatic function, the time to onset and offset of even short acting sedatives will be prolonged.⁴ There is also extreme variability in the response to sedative among these patients, cautions must be exercised through full monitoring of intra-operative and peri-operative mental status, oxygenation, and perfusion status. Elderly patients, scheduled for MAC, vigilance is necessary to identify co-morbid states, which increase incidence with age and often present, atypically.

This prospective descriptive study was designed to assess the efficacy, safety, and tolerability of MAC in elderly patients undergoing diagnostic, therapeutic, and surgical purposes under sedation, local anaesthesia or with only monitoring without any sedation or local anaesthesia.

Materials and Methods

We performed a prospective descriptive study on elderly patients of both sex, age from 50 years and above, scheduled to undergo different therapeutic, diagnostic and surgical procedures at CMH, Dhaka, Gastro Liver Clinic, Dhaka, and IBN SINA Medical Imaging Centre, Dhaka in one calendar year from July 2005 to June 2006. Patients with anatomic airway abnormalities, severe cardiovascular and respiratory disease and severe psychological problems were excluded from the study. During pre-procedural assessment, every patient underwent thorough physical examination with ASA classifications. Total MAC procedure was explained to every patient and informed consent was taken. A baseline pulse, blood pressure, respiratory rate, ECG and SpO₂ were recorded.

Anaesthesiologist was constantly available throughout the diagnostic, therapeutic or surgical procedure to provide MAC. Patient's level of consciousness, heart rate, blood pressure, respiratory rate, SpO₂ and electrocardiography were monitored throughout the whole procedure. A full set of resuscitation equipments including suction apparatus, oxygen, a bag valve mask, age appropriate airway, resuscitation drugs and defibrillator were available throughout sedation and recovery to combat any adverse event. Any serious adverse events as well as side effects during MAC like desaturation (SpO₂ less than 93 %) hypertension (systolic BP more than 30% of baseline record), hypotension (systolic BP less than 90 mm of Hg), arrhythmia, vomiting, agitation and nightmares were observed, recorded and managed.

Data from anaesthetic, procedure records and history charts of patients were recorded. The general data included age, sex, height, body weight, and ASA physical status. The anaesthetic data encompassed pre-anaesthetic problems, different procedures, choice of anaesthesia, variety of drug usage, duration of procedure and complications evolved during procedure. Results were expressed as mean \pm standard deviation (SD) or percentage (%) where appropriate.

Results

Characteristics of patients were shown in table I. There were 2144 procedures performed during the study period, majority patients were male (53.07%), mean age was 64.56 \pm 6.78 years. Age groups between 50 to 70 years were 85.95%, ASA physical status between I and II were 90.8%. Pre-anaesthetic problems were shown in table II. There were 1331 patients in less than ASA-II classification but some had multiple diseases and total 1428(66.60%) pre-anaesthetic problems found in 2144 patients. They involved mainly hypertension (26.49%) and diabetes mellitus (21.78%). Other problems were heart disease(7.78%); coronary artery disease, valvular heart disease, liver disease(2.09%); cirrhosis, respiratory disease(3.21%); chronic obstructive pulmonary disease, asthma, renal disease(2.65%); chronic renal failure, haematologic disease(1.95%); anaemia, and others(0.60%); electrolyte imbalance. Details of different diagnostic and therapeutic Procedures done under MAC were shown in table III. Almost all procedure was done under sedation

(70.38%), local anaesthesia (22.71%) and under only monitoring without sedation or local anaesthesia (6.90%). The details of sedative agents, narcotic drugs and local anaesthetics were shown in table IV. There were no serious adverse events reported in any patients during MAC. Complications during procedure and recovery were observed, recorded, and shown in table V. The common complications were arrhythmia (9.60%), vomiting (7.36%) and desaturation (4.56%), hypertension (5.64%). Other complications were hypotension (0.97%) and

agitation and nightmares (1.77%), and local anaesthetic toxicity (0.13%). Complications were minor, transient and promptly managed and corrected. Details of duration of MAC were shown in table VI. The MAC duration was ranged from 15 to 180 minutes. The mean procedure time was 35.1 ± 12.5 minutes. Majority of cases were completed within 30 minutes (51.21%), and between 30 to 60 minutes (40.48%). Other procedures completed between 60 – 89 minutes (7.78%) and more than 90 minutes required in 0.51% cases.

Table I*Demographic data of different study group*

Characteristics	Number	Percentage
Sex		
Male	1138	53.07%
Female	1006	46.92%
Age(years)		
50 - 59	1007	46.96%
60 - 69	834	38.99%
70 - 79	235	10.96%
80 - 89	57	2.65%
> 90	11	0.51%
Mean age±SD(years)	64.56±6.78	
ASA physical status		
I	813	37.91%
II	1134	52.89%
III	128	5.97%
IV	69	3.21%

There were 2,144 cases, but age and ASA physical status were not the same.

Table II*Preanaesthetic problem*

Preanaesthetic problem	Number	Percentage
Hypertension	568	26.49%
Diabetes Mellitus	467	21.78%
Heart disease	167	7.78%
Respiratory disease	69	3.21%
Renal disease	57	2.65%
Hepatic disease	45	2.09%
Haematologic disease	42	1.95%
Others	13	0.60%
Total	1428	66.60%

Total number of cases 2144 and there were 1331 patients in less than ASA-II classification but some had multiple diseases and total 1428 pre-anaesthetic problem found in them.

Table III*Procedures done under MAC*

Procedure	Number	Percentage
Under sedation		
ERCP	237	11.05%
Upper GI endoscopy	356	16.60%
Lower GI endoscopy	307	14.31%
MRI	238	11.10%
CT scan	124	5.78%
Radiotherapy	67	3.12%
Chemotherapy	34	1.58%
Wound dressing	146	6.80%
Under local anaesthesia		
Cataract surgery	352	16.41%
Excision of cyst/lipoma	103	4.80%
Wound repair/dressing	32	1.49%
Without sedation or local anaesthesia		
Upper GI endoscopy	34	1.58%
Lower GI endoscopy	23	1.07%
MRI	27	1.25%
CT scan	17	0.79%
Radiotherapy	09	0.41%
Chemotherapy	07	0.32%
Wound dressing	31	1.44%

Total number of cases 2144

Table-IV
Anaesthesia related data

Data	Number	Percentage
Sedative agents		
Diazepam	245	11.42%
Midazolam	234	10.91%
Propofol	108	5.03%
Ketamine	370	17.25%
Narcotics		
Pethidine	189	8.81%
Morphine	121	5.64%
Fentanyl	145	6.76%
Nalbuphine	97	4.52%
Local anaesthetics		
2%Lignocaine(plain)	152	7.08%
2%Lignocaine with adrenaline(1:200000)	31	1.44%
0.5%Bupivacaine(plain)	175	8.16%
0.5%Bupivacaine mixed with 2% lignocaine	121	5.64%

Total number of cases 2144

Table V
MAC related complications

Complications	Number	Percentage
Desaturation	97	4.56%
Hypertension	121	5.64%
Hypotension	21	0.97%
Arrhythmia	206	9.60%
Tachycardia	145	6.76%
Bradycardia	47	2.19%
Other arrhythmias	14	0.65%
Vomiting	158	7.36%
Agitation and nightmares	38	1.77%
Local anaesthetic toxicity	3	0.13%
Total	850	39.64%

Total number of cases 2144

Table VI
Duration of MAC

Duration of time (minutes)	Number	Percentage
<30	1098	51.21%
30 - 59	868	40.48%
60 - 89	167	7.78%
>90	11	0.51%
Mean procedure time±SD (minutes)	35.1 ± 12.5	

Total number of cases 2144

Discussion

The Monitored Anesthesia Care (MAC) phrase refers to instances in which an anesthesiologist has been called upon to provide specific anesthesia services to a particular patient undergoing a planned procedure, in connection with which a patient receives sedation, local anesthesia or in some cases no anesthesia at all. In such a case, the anesthesiologist is providing specific services to the patient and is in control of the patient's non-surgical or medical care, including the responsibility of monitoring of the patient's vital signs, and is available to administer anesthetics or provide other medical care as appropriate.

Purpose of MAC is to provide the patient with anxiety relief, amnesia, pain relief, comfort and safety during any therapeutic, diagnostic, and surgical procedure. MAC can be requested for patients undergoing uncomfortable procedures and minor surgeries, which do not require general anaesthesia.⁵ At one time, the term local standby described the role of anaesthesiologists in these cases. Monitored Anaesthesia Care has now replaced this term, as the anaesthesiologist should be continually monitoring the patient during surgery or procedure not just standby.⁶ There are few anaesthetic techniques those are used in younger patient cannot be used to anaesthetize the elderly patient. For elderly patients for some diagnostic, therapeutic including some surgical procedures MAC may be a better option for them.² In general, elderly patients are frailer, with greater likelihood of peri-operative complications. However even in the absence of a specific organ-based disease process, anaesthesia for the elderly may require an alteration in technique to the effects

that the aging process, the disease process, and any residual effects of previous illness have hand on the elderly patient and tailor the anaesthetic technique accordingly.⁷ The appearance and activity of the patient are thought to be more relevant than the actual age e.g. 90 years old may present less risk than frail 70 years old.⁸ Cardiopulmonary and other disease that are more frequent in older patients have been regarded as the major risk factors for the complications associated with sedation and procedures.^{9,10,11} In this study we performed MAC for surgeries and different diagnostic and therapeutic procedures with or without sedation or under local anaesthesia in elderly patients. There were several studies on surgeries and diagnostic and therapeutic procedures under MAC. They prove MAC can be successfully achieved during surgeries like cataract surgery, skin surgery and diagnostic procedure like endoscopies and imaging procedures.^{12,13,14} MAC combines intravenous sedation with local anaesthesia, nerve blocks or with endoscopies.^{15,16} There were no special anaesthetic techniques needed for MAC. Different sedation regimens are available. In this study, we used benzodiazepines, opioids, propofol and ketamine in titrated low doses. The primary disadvantages of sedation are the lack of airway control and the threat of aspiration or airway obstruction. To minimize the risks, the anaesthesiologist must titrate medications carefully to maintain spontaneous respirations while maintaining an anaesthetic depth, allowing the patient to remain comfortable. Careful selection and administration of medications is essential in producing the desired and optimal intra-procedure anaesthetic effects and post procedure outcomes. Sedatives and opioids can also make patient drowsy and may cause desaturation.

Duration of procedure should not take prolong time as related with patient's discomfort and complications.¹⁷ In this study, most of the procedure completed within 60 minutes duration. Complications may arise during MAC in elderly patients. Causes are co-existing diseases in elderly, sedation, local anaesthetic and the procedure itself. Hypoxia or desaturation are common with sedation with benzodiazepine and opioids.¹⁸ Arrhythmias are usually observed with coexisting cardiac diseases and with upper GIT endoscopies and

cataract surgeries.^{19,20} Patient undergo MAC are never mend to be without recall.²¹ Whether or not a patient remembers the procedure depends on the type of medications uses, dosage, patients physiology, and other factors. Many patients undergoing MAC do not remember the experiences. In this study, no serious complications were observed. Incidence of complications associated with sedation, procedure and local anaesthetics found less and easily manageable with available resources.

Conclusion:

Concluded in properly selected elderly patients, MAC is a safe and effective method of providing intraoperative care for some common diagnostic, therapeutic, and surgical procedures. However, few complications may arise during procedure but they were minor, transient and promptly managed and corrected.

References:

1. ASA position on Monitored Anesthesia Care. Approved by the House Delegates September 2008.
2. Margaret Ekstein, Doron Gavish, Tiberia Ezri, Avi Weinbroum. Monitored Anesthesia Care in the elderly: Guidelines and Recommendations. *Drugs Aging* 2008; 25(6):477-500.
3. Raymond R. Anesthetic management of the elderly patient. 53rd ASA Annual Meeting Refresher Course Lectures no. 2002; 321: 1-7.
4. Muravichik S. Pharmacological changes of aging. 53rd ASA Annual Meeting Course Lectures 2002; 1-7.
5. Rego M, White PF. Monitored Anesthesia Care. RD Miller(ed). *Miller's Anesthesia* 5th ed. Philadelphia. Churchill Livingstone, 2000:1452-1467.
6. G. Edward Morgan, Jr. Maged S. Mikhali, Michael J. Murray. *Clinical Anesthesiology*. 4th ed. USA. McGraw-Hill, 2006:831.
7. David Murray, Chris Dodds. Perioperative care of the elderly. *Anaesthesia, Critical Care & Pain* 2004;4(6):193-196.
8. Steven MY, Nicholas PH, Gary BS. *Anaesthesia and Intensive care A-Z*. 3rd ed. UK Butterworth Heinemann, 2004:175-176.

9. Nagengast EM. Sedation and monitoring in gastrointestinal endoscopy. *Scand J Gastroenterol* 1993;200:28-32.
10. Bell GD, McCloy RF, Campell D, et al. Recommendations for standards of sedation and patient monitoring in gastrointestinal endoscopy. *Gut* 1991; 32:823-827.
11. Bell GD. Premedication and intravenous sedation for upper gastrointestinal endoscopy. *Aliment Pharmacol Ther* 1990; 4: 103-122.
12. Bing J, McAuliffe MS, Lupton JR. Regional anesthesia with monitored anesthesia care for dermatologic laser surgery. *Dermatol Clin* 2002;20(1):123-124.
13. Rosenfeld SI, Litinsky SM, Snyder DA, Plosker H, Astrove AW, Schiffman J. Effectiveness of Monitored Anesthesia Care in Cataract surgery. *Ophthalmology* 1999; 106:1256-1261.
14. Malhotra S, Dutta A, Gupta A. Monitored anaesthesia in elderly ophthalmic patients. *The Lancet* 2003; 359: 532.
15. Badrinath S, et al. The use of ketamine - propofol combination during Monitored Anesthesia Care. *Anesth. Analg* 2000; 90:858-862.
16. Michael Mercandetti. Anesthesia, local with sedation. *Medicine*. Update on March 7, 2008.
17. Warner D, Warner M. Anesthetic risk and the elderly. *Syllabus on Geriatric Anesthesiology*. ASA. 2002:1-4.
18. Anh - thuy Nguyen. Monitored Anesthesia Care. *The Internet Journal of Health* 2000; 1.
19. Lee JF, Leung JWC Cotton PB. Acute cardiovascular complications of endoscopy: prevalence and clinical characteristics. *Dig Dis* 1995; 13(2): 130-135.
20. Lieberman DA, Wuerker CK, Katon RM. Cardiopulmonary risk of endoscopic diameter and systematic sedation. *Gastroenterology* 1985; 88: 468-472.
21. Lekpraser Varinee. "Preanaesthetic Assessment of Patient Who Reports Previous Intraoperative Awareness." *Anaesthesiology News* 2008:35-38.

Caudal bupivacaine - midazolam for post operative analgesia in children

Idris Ali^{1*}, Amirul Islam², Golam Morshed³, Nurul Islam⁴, Ashia Ali⁵, UH Shahera Khatun⁶

¹Consultant Anaesthesiologist, Dhaka Dental College Hospital, Dhaka. ²Anaesthesiologist, Department of Anaesthesia, Analgesia & Intensive Care Medicine, BSMMU, Shahbag, Dhaka. ³Consultant Anaesthesiologist, Chuadanga Health Complex, Kushtia. ⁴Anaesthesiologist, Department of Anaesthesiology and ICU, DMCH, Dhaka. ⁵Associate Professor, Cardiac Anaesthesia unit, Department of Anaesthesia, Analgesia & Intensive Care Medicine, BSMMU, Shahbag, Dhaka. ⁶Professor and Head, Department of Anaesthesiology and ICU, DMCH, Dhaka.

*Corresponding author: Email: <idrisali74@yahoo.com>

Abstract

Background: Adjuvant used with local anaesthetic agent in caudal is more effective for post operative analgesia in children.

Aim and objective: To find out the duration and quality of caudal analgesia in children undergoing genitourinary surgery by combination of bupivacaine and midazolam.

Methods: A total number of sixty patients ASA grade I&II were selected randomly as per inclusion & exclusion criteria in two groups. Thirty in each group. In group A, caudal block was given by bupivacaine-midazolam mixture and in group B, caudal block was given by bupivacaine in lateral decubitus position, just after completion of surgery before reversed from GA. In post operative period arterial blood pressure, heart rate, and duration of analgesia were recorded.

Results: There was no significant difference between the groups of blood pressure, heart rate, and pain score up to 30 min but after one hour of post operative period pain scores were significant ($p < 0.05$).

Conclusion: Midazolam improves the duration and quality of analgesic effect of bupivacaine.

Key words: bupivacaine, midazolam, caudal, genitourinary, analgesia.

(Journal of BSA, 2010; 23(1): 8-13)

Introduction

Caudal epidural is one of the most common regional techniques used for post-operative pain management in pediatric patients¹. It is commonly used for procedures like urogenital, rectal, inguinal and lower extremity surgery².

Drugs which used commonly in caudal analgesia, are bupivacaine & lignocaine. Opioids may also be used as adjunct, although they are not recommended for day case surgery because of the risk of delayed respiratory depression. We want to use bupivacaine –midazolam combination to know the duration and quality of caudal analgesia. Antinociceptive effect, effective analgesic properties and safety of intrathecal-administered midazolam are well established in animals and human beings³. In-vitro autoradiography has shown that there is a high density of benzodiazepine (GABA-A) receptors in lamina-II of

the dorsal horn in the human spinal cord, suggesting their possible role in pain modulation. The delta selective opioid antagonist naltrindol suppresses the antinociceptive effect of intrathecal midazolam, suggesting that intrathecal midazolam involved in the release of endogenous opioid acting at spinal delta receptors^{4,5}. In 1987, Goodchild and Serrao reported that benzodiazepines might have analgesic effects at spinal cord level in animal^{4,5}. Recently it has been demonstrated the analgesic efficacy of intrathecal midazolam in human^{4,5}.

There have been some reports on the spinal application of midazolam in humans, which show no neurotoxic effect^{4,5}. A single intrathecal injection of 2 mg midazolam did not cause any clinical neurological deficits and produce significant analgesia for 2 months in patients with chronic low back pain⁶. Intrathecal midazolam was also effective after leg surgery without any side effects⁷.

So we can think that there is no neurotoxicity of caudal midazolam administration.

In addition to the effectiveness of intrathecal midazolam against somatic pain, an antinociceptive effect against visceral pain has been demonstrated in rabbits subjected to intestinal distention and in humans after caesarean section⁸.

Intrathecal midazolam has been used in a continuous infusion for a long-term period with refractory neurogenic and musculoskeletal pain. In-vitro studies have suggested that clinically useful doses of intrathecal midazolam are unlikely to cause neurotoxic⁹.

Intrathecal midazolam does not have the unwanted effects like pruritis, nausea and vomiting and respiratory depression as caused by intrathecal opioids⁹.

The use of conventional dose of bupivacaine is associated with a high incidence of hypotension, prolonged motor recovery, nausea and vomiting and discharge time. It may be possible to minimize these unwanted outcomes by using small dose of bupivacaine combined with midazolam.

On the other hand low dose of bupivacaine may cause inadequate analgesia leading to discomfort during traction of peritoneum. This may be overcome by addition of midazolam to bupivacaine, which may prolong the duration of analgesia because of its antinociceptive action.

Method

After approval of Ethical Committee, 60 patients were selected randomly as per inclusion and exclusion criteria. Before taking the patient into the operation theatre, legal guardian of the patient was consulted about post-operative analgesia and consent for caudal analgesia was taken. Patients were allocated randomly into two groups, 30 patients in each group.

The same conventional GA was given to all patients. After completion of the surgery in group A patient caudal block was given by bupivacaine-midazolam mixture (0.125% bupivacaine 0.8 ml/kg, midazolam 0.08 mg/kg) and in group B patient caudal block was given by bupivacaine (0.125% bupivacaine 0.8 ml/kg) only. Caudal block was done in lateral decubitus position. Then patient was reversed in supine position.

Following parameters were recorded: - Time of caudal block, heart rate, blood pressure, duration of analgesia by first demand of analgesia of the patient.

Data was collected on pre-designed data collection sheet and was analyzed for statistical significance by Chi-square or student's t-test as appropriate. P value less than 0.05 was considered statistically significant.

Results

Observation of the present study was analyzed in the light of comparison among the subject groups, each group having n=30. All results are expressed as mean \pm standard error of mean (SEM). The studied groups became statistically matched for age (p=0.871), weight (p=0.796), duration of surgery (p=0.016), pulse rate (p=0.096), systolic blood pressure (p=0.932), diastolic blood pressure (p=0.503) and SpO₂ (p=0.237), and pain score (p<0.05).

Table-I
Demographic data

Groups	Age (year)	Weight (Kg)	Duration of surgery(Min.)
	Mean \pm SEM	Mean \pm SEM	Mean \pm SEM
Group A	4.209 \pm 0.206	12.90 \pm 0.354	48.45 \pm 1.199
Group B	4.200 \pm 0.221	12.800 \pm 0.414	45.50 \pm 0.874
P	0.871	0.796	0.016

Values were expressed as mean \pm SEM. Analysis was done by student's t- test. There was no significant changes.

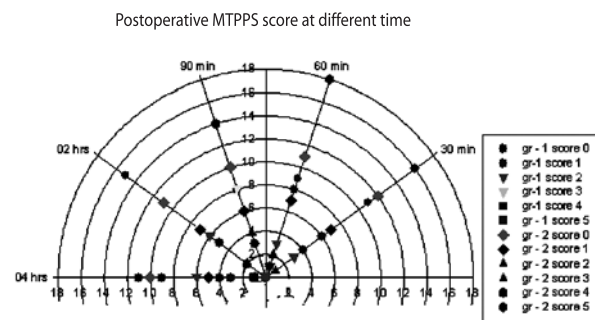


Fig.-1. Modified TPPPS score in different time period.

Table-II
Changes of heart rate.

Groups	Pre - operative	After Induction	After Caudal	End of Surgery	½ hr POP	1hr POP	1 ½ hr POP	2 hr POP	4 hr POP	6 hr POP	8 hr POP	12 hr POP
A	103.00 ± 1.76	140.15 ±2.24	140.65 ±1.80	103.60 ±1.75	104.95 ±1.95	102.40 ±4.90	102.50 ±5.14	108.25 ±2.14	111.60 ±2.62	104.00 ±5.37	111.44 ±1.87	109.85 ±1.97
B	102.35 ±2.28	102.50 ±2.24	103.40 ±2.27	98.15 ±5.10	103.45 ±1.74	103.00 ±2.00	102.90 ±1.68	99.60 ±4.6	104.00 ±1.66	98.60 ±4.64	104.20 ±1.85	100.00 ±5.15
P	0.945	0.665	0.377	0.467	0.611	0.947	0.994	0.158	0.019	0.539	0.022	0.137

Values were expressed as mean ± SEM. Analysis was done by student's t-test. There was no significant change in heart rate between groups.

Table-III
Changes in systolic blood pressure

Groups	Pre- operative	After Induction	After Caudal	End of Surgery	½ hr POP	1hr POP	1 ½ hr POP	2 hr POP	4 hr POP	6 hr POP	8 hr POP	12 hr POP
A	29.00 ±10.95	79.75 ±11.59	79.85 ±13.03	80.00 11.08	78.70 ±11.62	77.30 ±11.53	79.30 ±11.55	81.90 ±10.59	81.90 ±11.96	80.90 ±10.40	81.10 ±10.90	79.20 ±10.55
B	77.60 ±13.17	77.60 ±13.18	77.60 ±13.18	77.65 13.18	77.85 ±12.44	77.85 ±12.44	77.85 ±12.44	79.65 ±12.35	79.35 ±11.92	79.30 ±11.89	79.15 ±12.30	79.60 ±12.62
P	0.932	0.845	0.718	0.717	0.958	0.927	0.899	0.630	0.804	0.825	0.872	0.985

Values were expressed as mean ± SEM. Analysis was done by student's t- test. There was no significant change in systolic blood pressure between groups. Mean systolic blood pressure (SBP) in group A was 79.00 ± 10.95 and in group B was 77.60 ± 13.17, (P= 0.932). Mean values of systolic BP varied from 77.30 ± 11.53 to 81.90 ± 10.59 in-group A and 77.60 ± 13.17 to 79.63 ± 12.35 in-group B. Systolic blood pressure did not vary significantly in different time period between groups . (Table-III)

Table-IV
Changes of diastolic blood pressure.

Groups	Pre - operative	After Induction	After Caudal	End of Surgery	½ hr POP	1hr POP	1 ½ hr POP	2 hr POP	4 hr POP	6 hr POP	8 hr POP	12 hr POP
A	42.25 ±9.66	42.15 ±10.01	42.33 ±9.49	42.95 ±8.127	40.60 ±5.78	39.25 ±6.98	40.60 ±6.16	40.10 ±6.86	40.65 ±5.05	41.65 ±4.55	42.10 ±5.05	42.25 ±4.20
B	46.50 ±30.96	46.50 ±30.96	45.70 ±14.20	44.75 14.45	44.90 ±15.05	44.85 ±15.09	45.25 ±14.78	46.20 ±13.96	46.80 ±13.62	46.95 ±13.73	48.10 ±14.36	47.60 ±14.76
P	0.503	0.428	0.665	0.928	0.518	0.363	0.442	0.397	0.202	0.301	0.228	0.290

Values were expressed as mean ± SEM. Analysis was done by student's t -test. There was no significant change in diastolic blood pressure between groups. Mean diastolic blood pressure was 42.25± 9.66 in-group A and 46.50 ± 13.96 in-group B (P = 0.503). Diastolic blood pressure in-group A varied from 39.25 ± 6.98 to 42.75 ± 8.12 and from 44.90 ± 15.05 to 47.70 ± 14.67 in group B. Diastolic blood pressure did not vary significantly between groups in different time period.(Table-IV)

Table-V
Postoperative MTPPS score at different time.

Group	Score	At 30 min		At 1 hr		At 90 min		At 2 hr		At 4 hr	
		No of Pt		No of Pt		No of Pt		No of Pt		No of Pt	
A	0	20		18		16		15		10	
	1	10		10		12		13		15	
	2	0		2		2		2		3	
	3	0		0		0		0		2	
	4	0		0		0		0		0	
	5	0	P=0.249	0	P=0.023	0	P=0.008	0	P=0.000	0	P=0.006
B	0	18	X ² =5.40	17	X ² =11.38	15	X ² =17.28	15	X ² =30.97	10	X ² =24.76
	1	12		10		12		11		12	
	2	0		3		3		4		5	
	3	0		0		0		0		3	
	4	0		0		0		0		0	
	5	0		0		0		0		0	

Intensity of pain in different time period was measured using modified TPPPS score. Shown in (Figure-1, Table-V). At 30 min pain score was insignificant (p=0.249) but at one hour to four hours 30 min interval there were significant difference between the groups ie, (p=0.023), (p=0.008), (p=0.000), (p=0.006) respectively.

Table VI

Rescue pethidine required first time in minute

Group	Number of Pt.	Mean ± SEM	P
A	30	210.00 ± 30.00	<0.05
B	30	150.00 ± 30.00	

Rescue pethidine required first time was 210.00 ± 30.00 in group A and 150.00 ± 30.00 in group B. (Table-VI)

Discussion

Prolongation of analgesic effect of bupivacaine by caudal route is a desired goal for anaesthetologist. In this study we used caudal midazolam along with 0.125% plain bupivacaine to achieve that goal.

Bupivacaine is the local anaesthetic with longest duration of action currently available. When we used for analgesia in children in a dose of 2.0-2.5 mg/kg, it lasts for 2-3 hrs., most of the children undergoing sub umbilical operation require further analgesia during postoperative period¹⁰ which influenced many authors to search for means to prolong the duration of caudal analgesia.

Many drugs including epinephrine, morphine¹¹, clonidine¹¹, ketamine¹¹, and tramadol^{12, 13} have

been co-administered with caudal bupivacaine to maximize and extend the duration of analgesia. Morphine extends the duration of analgesia but frequent association of delayed respiratory depression, itching, vomiting, and postoperative retention of urine has limited its use¹⁴. Behavioral side effects are reported with use of ketamine and increased incidence of vomiting was observed with use of tramadol^{12, 13}.

Midazolam is being investigated for use with bupivacaine caudally for postoperative pain control. Antinociceptive effect, effective analgesic properties and safety of intrathecal- midazolam are well established in animals and human beings³.

There have been some reports on the spinal application of midazolam in humans, which show no neurotoxic effect^{4,5}. A single intrathecal injection of 2 mg midazolam did not cause any clinical neurological deficits and produce significant analgesia for 2 months in patients with chronic low back pain⁶. Intrathecal midazolam was also effective after leg surgery without any side effects⁷.

So we can think that there is no neurotoxicity of caudal midazolam administration.

In addition to the effectiveness of intrathecal midazolam against somatic pain, an antinociceptive effect against visceral pain has been demonstrated in rabbits subjected to intestinal distention and in humans after caesarean section⁸.

Intrathecal midazolam has been used in a continuous infusion for a long-term period with refractory neurogenic and musculoskeletal pain. In vitro studies have suggested that clinically useful dosages of intrathecal midazolam are unlikely to be neurotoxic⁹.

Intrathecal midazolam does not have the unwanted effects like pruritis, nausea and vomiting and respiratory depression as caused by intrathecal opioids⁹.

The use of conventional dose of bupivacaine is associated with a high incidence of hypotension, prolonged motor recovery, nausea and vomiting and discharge time. It may be possible to minimize these unwanted outcomes by using small dose of bupivacaine combined with midazolam.

On the other hand low dose of bupivacaine may cause inadequate analgesia leading to discomfort during traction of peritoneum. This may be overcome by addition of midazolam to bupivacaine, which may prolong the duration of analgesia because of its antinociceptive action.

In our study we used Midazolam along with Bupivacaine at the end of the surgery through caudal route in childrens undergone genitourinary surgery.

Our observation showed that there were no significant changes in heart rate, blood pressure and oxygen saturation in both groups. We also observed no untoward event in either of the groups.

Our result shows that caudal midazolam along with bupivacaine prolongs the postoperative analgesic action.

In group A, duration of analgesia is 210 ± 30 minutes and in group B, duration of analgesia is 150 ± 30 minutes.

So, Conclusion is that midazolam improves the duration and quality of analgesic effects of bupivacaine without any side effects.

References

1. Markakis DA, Regional anaesthesia in pediatrics. *Anesthesiol Clin North America* 2000; 18:355-81.
2. Murat I, Dalens B, pharmacology, In: Dalens B eds. *Regional Anaesthesia in infants, children and adolescents*. Waverly Europe. London 1995; 67-99.
3. Bhattacharya D, Biswas B, Banerjee A. Intrathecal midazolam with bupivacaine increases the analgesic effects of spinal blockade after lower abdominal surgery. *Journal of Anaesthesiology clinical pharmacology* 2002; 18(2): 183-6.
4. Shah FR, Halbe AR, Panchal ID et al. Improvement in postoperative pain relief by the addition of midazolam to an intrathecal injection of buprenorphine and bupivacaine. *Eur. J. Anaesthesiol* 2003; 20(11): 904-10.
5. Batra YK, Jam K, Chari P et al. Addition of intrathecal midazolam to bupivacaine produces better post-operative analgesic without prolonging recovery. *Mt. J Clinical Pharmacol Ther* 1999; 37(10): 519-23.
6. Bharti N, Madan R, Mohanty PR et al. Intrathecal midazolam added to bupivacaine improves the duration and quality of spinal anaesthesia. *Acta Anaesthesiol Scand* 2003; 47(9): 1101-5.
7. Valentine JM, Lyons G, Bellamy MC . The effect of intrathecal midazolam on post-operative pain. *Eur J Anaesthesiol* 1996; 13(6): 589-93.
8. Kim MH, Lee YM . Intrathecal midazolam increases the analgesic effects of spinal blockade with bupivacaine in patients undergoing haemorrhoidectomy. *Br J Anaesth* 2001; 86(1): 74.
9. Tomokiki , Kazuo. Midazolam can potentiate the analgesic effects of intrathecal bupivacaine on thermal- or Inflammatory induced pain. *Abstracts: Nishiyama and Hanaoko* 96(5): 1386.
10. Wolf AR, H ghes D, Wade A, et al. Postoperative analgesia after paediatric orchidopexy; evaluation of a bupivacaine – morphine mixture. *Br J Anaesth* 1990; 64:430-5.
11. Cook B, Grubb DJ, Albridge LA, Doyle E. Comparison of the effects of adrenaline, clonidine and ketamine on the duration of caudal analgesia produced by bupivacaine – in children. *Br J Anaesth* 1995; 75:698-701.

12. Gunduz M, Ozcengiz D, Ozbek H, Isik G. A comparison of single dose caudal tramadol, tramadol plus bupivacaine and bupivacaine administration for post operative analgesia in children. *Paediatr Anaesth* 2001;1:323-6.
13. Batra YK, Prasad MK, Arya VK, et al. Comparison of caudal tramadol vs bupivacaine for post operative analgesia in children undergoing hypospadias surgery. *Int J Clin Pharmacol Ther* 1999;37:238-42.
14. Ness TJ, Gebhart GF. Visceral pain :a review of experimental studies. *Pain* 1990;41:167-234.
15. Goodchild CS, Gua Z, Musgreave A, Gent JP. Antinociception by intrathecal midazolam involves endogenous neurotransmitters acting at spinal cord delta opioid receptors. *Br J Anaesth* 1996; 77:758-763.
16. Tardell SE, Cohan IT, Marsh JL. The modified toddler preschooler post operative pain scale. An observational scale for measuring postoperative pain children age 1-5, Preliminary report. *Pain*; 50 :273-80.

Nutritional Support to Critically Ill Patient

Iqbal Hossain Chowdhury*

Associate Professor (Intensive Care Unit), Anaesthesia, Department of Analgesia, & Intensive Care Medicine, BSMMU, Dhaka
* Corresponding author: iqbalhossain56@gmail.com

(Journal of BSA, 2010; 23(1): 30-33)

Introduction

Nutrition is defined as science of food and relationship to health. It is not a single science but a cluster of sciences related to the production and utilization of food¹. Critical illness evoke a constellation of metabolic changes in the host including a transitory “ebb” phase followed by a hyper metabolic “flow” phase. Magnitude of the change is proportional to the extent of insult or illness. Those changes require an extra amount of energy in addition to basic metabolic requirement to maintain the nutritional status. If the basic and extra amount of energy cannot be provided, the patient may show diverse systemic functional impairments. Nutrients are needed for protein synthesis, for organ function and to sustain life.² Critical illness is usually accompanied by anorexia or inability to eat because of impaired consciousness, sedation or intubation through upper airway. Patients are also metabolically stressed by the severity of the illness. Therefore, without nutritional support there is rapid loss of body weight and muscle mass. American Society for Parenteral and Enteral nutrition has included the following,^{2,3} (a) detection and correction of pre-existing malnutrition, (b) prevention of progressive protein energy malnutrition, (c) optimizing patients metabolic state, and (d) reduction of morbidity and time of convalescence. Nutritional support to critically ill patient in Intensive Care Unit (ICU) must follow the same rules as any other form of treatment with careful appraisal in each patient of likely benefit or harm to be expected from it.⁴

Effects of Malnutrition

The accelerated catabolism associated with acute illness or injury may further exacerbate tissue loss superimposed upon weight loss. Weight loss more than 8% results increasing impairment of function, handicap recovery from disease and multiply its complications.⁴ Malnourished persons suffer from

muscle weakness and muscle fibers as well as respiratory muscles including diaphragm, impairing respiratory drive, ability to cough and clearing secretion.^{4,5} Malnutrition impaired immune function and increase rate of infection.^{6,7,8} Acute illness and malnutrition also impair the digestive and barrier function of gut and may be protected by enteral feeding. Cardiovascular reflexes, vasoconstrictor responses to cold, heat conservation also affected by malnutrition.⁹ Malnutrition also contributes to increased surgical risk, poorer wound healing and slower recovery from surgery.⁴ Starvation and the responses to injury and immobility contribute to excess salt and water retention and negative nitrogen balance.

Clinical Decision to Treat

Nutritional support has been shown to be effective in improvement in nitrogen balance, wound healing, restore immune competence, facilitated weaning from ventilator and reduce mortality and morbidity in critically ill patients.³⁻⁴

From a consideration of the evidence outlined so far, the following indications for nutritional support are suggested.⁴

- a. Weight loss greater than 10 percent and continuing.
- b. Continuing inadequate oral intake.
- c. The presence of the diseases whose known natural history is associated with accelerated catabolism and poor food intake for 10 days or more.

Nutritional Assessment of Patient

A normal nutritional status is a key element in the ability of a patient to overcome a critical illness. All the traditional markers of malnutrition lose their specificity in the sick adults as a number of non-nutritional factors may affect each. Nutritional

assessment can be done by obtaining dietary history, clinical examination, anthropometry and laboratory investigations.

- a. **Dietary history:** History includes dietary habits, nutrients intake, quality and quantity of food and omission of any major item may lead to malnutrition. Important questions include recent unintentional weight loss (>10%), recent surgical stress, nausea, vomiting, diarrhoea, and the presence of co-morbid illness.
- b. **Clinical examination:** Signs of nutritional deficiency, such as weakness, muscle wasting, loss of subcutaneous fat, skin rashes, hair thinning, pallor, oedema, ascitis, fingernail abnormalities and many other clinical parameters come under practically feasible. Particular signs of specific nutrient deficiencies must be noticed.
- c. **Anthropometry:** Anthropometric measurements are sufficient to define the nutritional status in healthy individuals but may be affected by non-nutritional factors. Therefore, anthropometric measurements must be interpreted with care. Measurement includes body weight, mid-arm circumference, mid-leg circumference, and triceps skin fold thickness etc.
- d. **Laboratory investigations:** Investigations consists those indicate protein status and biochemical tests for micro nutrient deficiencies.
 - i. Haemoglobin estimation, ii. Serum total protein. iii. Serum albumin, iv. Lymphocyte count.

Routes of Administration of Feed

In deciding upon the rule of administration of feed, the rules are simple. If the gut works, try to use it. If the patient can swallow, try oral supplements or failing this, some form of enteral feeding by a fine-bore naso-gastric tube. Alternative enteral routes to oral feeding can be shown separately as under.

- a. Naso-duodenal tubes,
- b. Gastrostomy.
- c. Jejunostomy tube or catheter-either feeding or percutaneous.

Enteral route of administration may be a good choice among the enteral and parenteral feeding in consideration of the complications of parenteral feeding and advantages of enteral feeding.

Advantage of Enteral Feeding over Parenteral

If the gastrointestinal tract is functional, the tube feeding is easier, safe and less costly than parenteral nutrition. It is possible that enteral feeds may also permit better utilization of nutrients, maintain mucosal integrity, and decrease the incidence of stress related haemorrhagic gastritis.¹¹ It stimulates intestinal blood flow. Recent works also suggest that enteral nutrition may lead to reduction on mortality in patients ventilated for prolonged periods.³ Consequently, when spontaneous oral feeding is inadequate then feeding can be given to all patients except non-functioning gut cases. Enteral feeding has been shown to be gut protective and reduce the associated rise in hepatic enzyme in haemorrhagic shock or endotoxic shocked patient.² Patients with blunt and penetrating trauma enteral feeding is better tolerated and associated with a lower frequency of infection within 24 hours.² Enteral delivery of nutrients compared to total parenteral nutrition (TPN) may reduce some complication in severely injured trauma patients and has been associated with a decrease in GIT mucosal permeability.¹² Avoidance of immune suppression and the complications of central venous canula insertion required for parenteral feeding give additional advantage to enteral feeding.

Energy Expenditure and Calculation of Energy Requirement

The key decision to provide nutritional support to critically ill patient involves the provision of adequate but not excessive amount of energy. Basal Metabolic Rate (BMR) can be estimated by heat loss using direct calorimetry, which is only possible in laboratory setting. In practice, caloric requirement is estimated by indirect calorimetry, which measures oxygen consumption, and energy expenditure is calculated. Formula like Harris-Benedict equation or other simpler more practical formula can be used to predict the basic energy expenditure.¹³ Another 500-1000 kcal should be added for the hyper catabolic state of critically ill patients.

Feed Composition

There has been considerable effort to determine what constitute adequate and optional nutritional support to critically ill patient. Enteral nutritional supplement should be composed of optimal combination of protein, carbohydrate and fat. The volume, water content, ionic composition and addition of trace elements and vitamins are of great importance. They can be prepared from fresh foods or commercially prepared diets. Many prepared feeds in liquid or powder form are commercially available. These vary in their protein, carbohydrate and fat source, electrolyte, mineral, vitamin content, osmolality and contents of specific nutrients including fiber, branched chain-1 acid (BCAA), essential amino acid, glutamine, arginine, nucleotides and other nutrients. Lactose-free, isotonic liquid feed providing approximately two-third of non-protein energy as carbohydrate meets the need of most patients. When necessary, such a feed can be modified by the addition of individual carbohydrate, protein or fat sources to meet specific need.

Complications Associated with Enteral Feeding

- a. Complications associated with feeding tube.
 - i. Trauma and bleeding.
 - ii. Gastric or bowel perforation.
 - iii. Tube obstruction.
 - iv. Tube displacement.
 - v. Patient discomfort.
- b. Complications related to enteral feeding,
 - i. Nosocomial infection from bacterial contamination.
 - ii. Nausea, abdominal distension and discomfort.
 - iii. Regurgitation or vomiting
 - iv. Pulmonary aspiration of feed.
 - v. Diarrhoea.
 - vi. Intestinal pseudo-obstruction.
- c. Complications related to feed content.
 - i. Hyper and hypoglycaemia.
 - ii. Glucose intolerance.
 - iii. Azotaemia.

- iv. Hypercarbia.
- v. Electrolyte abnormalities.
- vi. Specific deficiency disorder with long term use.

Monitoring of Patients Receiving Enteral Feeding

- a. Clinical
 - i. Examine the abdomen for distention and bowel sounds.
 - ii. Record the frequency and consistency of stool, and their colour, odour and estimated weight or volume.
 - iii. Note patient's complaints of fullness, nausea, vomiting, abdominal pain or tenderness.
- b. Blood
 - i. Measure blood glucose, blood urea nitrogen and serum electrolyte levels at least twice a week or more frequently if they are abnormal.
 - ii. Measure SGOT, SGPT, LDH, serum albumin, bilirubin, calcium, magnesium and phosphate levels once week.
 - iii. Measure serum triglyceride and cholesterol levels at least once a week in patients receiving fat in their diets.
- c. Urine

Test for glucose for 6 hours and cover with crystalline insulin as follows: 5U for 0.23g/dl, 15u for 1g/dl and 20U for 2g/dl.

Repeat the test hourly for 2g/dl and cover with 20U for 2g/dl every 2 hours. Inform physician if glycosuria lasts for 4 consecutive hours. Then resume feeding with a lesser rate or with a formula containing less carbohydrate.

Present Status of Nutritional Support to Critically ill Patient in Bangladesh

The method of nutritional support to critically ill patient in different hospitals of Bangladesh is in primitive state. Because we are running shortage of specialist dietetics, lack of knowledge, and other resources etc. There is no organized way of providing nutritional support; moreover, it is difficult to specify a food composition without adequate knowledge about nutrition. As a result present trend is that either the clinician prescribe

a branded preparation or patients party prepare a homemade preparation at there own without proper calculation about the patients energy requirement. Some times doctors become delayed to start the nutritional support to patient even after several days of admission in hospital. So there are many scopes remain to work in this area for better improvement.

Conclusion

Patient undergoing treatment in ICU remains in critical state of their health, where provision of nutritional support can ensure a good organ function or cat not prevent their functional impairment. Method of nutritional support may vary from individual to individual considering nature of disease process. A team of personnel, which should include clinician, nutritionist, dietetics and cools, should do it. Problem may arise due to lack of coordination between the members of treating committee or absence of such committee. As nutritional support to critically ill patient is an obvious life support; it should be ensured with proper importance and dedication.

References

1. Tod Hunter EN. Historical landmark in Nutrition. 5th ed, Washington DC Nutrition foundation 1984.
2. Andrew RW, Marc JS, Mervyn S, Peter MS. Oxford Text Book of Critical Care. 1st ed. Oxford: Oxford University Press, 1999: 394.
3. TE Oh. Intensive Care Manual. 4th ed. Oxford: Butterworth- Heinemann; 1998; 716-26.
4. Th E J Healy, Peter J Cohen. A practice of Anaesthesia. 6th ed. London: Wylie and hurchill-Davidson; 1995; 886-896.
5. Doekel Jr RC, Zwillich CW, Scroggin CH. Clinical semi-starvation: depression of hypoxic ventilatory response. New England Journal of Medicine 1976;295:358-361.
6. Shizgal HM. Nutrition and immune function. Surgery Annals 1981; 12:15-29.
7. Chandra RK. Immunity and infection. In: Kinney JM, Jeejeebhoy KN, Hill GL, Owen OE, eds. Nutrition and metabolism in patient care. Philadelphia; WB Saunders, 1988; 598-604.
8. Windsor .IA, Hill GL. Risk factors for postoperative pneumonia: the importance of protein depletion. Annals of Surgery 1988; 208:209-14.
9. Macdonald IA, Bennett T, Sainsbury R. The effect of a 48 hour fast on the thermoregulatory responses to garded cooling in man. Clinical Science 1985; 67: 445-52.
10. Al Maliki T, Langer JC, Thompson V, et al. A prospective evaluation of the button gastrostomy in children. Can Surg 1991; 10: 18-22.
11. Frisancho AR: New norms of upper limb fat and muscle areas for assessment of nutritional status. Am J Clin Nutr 1981; 34: 2540.
12. Jesse BH, Gregory AS, Lawrence DH. Wood, Principles of Critical Care. 2nd ed. New York: Mc Graw-Hill. 1998; 203.
13. Park K. Park's Text Book of Preventive and Social Medicine. 19th ed, India. M/S Banarsidas Bhanot Publishers 2007; 501-507.

Effect of preemptive intravenous ketorolac and bupivacaine infiltration on post operative pethidine consumption - a comparative study

Jalal Uddin Ahmed¹, Md Shahidul Islam², Md Shafiqul Islam³, Mantosh Kumar Mandal⁴,
Mohammad Sirajul Islam⁵, Md. Shamsuzzoha⁶, Akhtaruzzaman AKM⁷

¹Associate Professor, Department of Anaesthesiology, Dhaka Dental College, ²Consultant, Department of Anaesthesiology, Dhaka Medical College & Hospital, ³Assistant Professor, Shaheed Suhrawardy Medical College & Hospital, ⁴Assistant Professor, Department of Anaesthesiology, National Cancer Hospital and Research Institute, Mohakhali Dhaka, ⁵Consultant Anaesthesiologist, Sarail Up-Zilla Health Complex, B Baria, ⁶Assistant Professor, Dept. of Anaesthesia, Mansur Ali Medical College, Uttara, Dhaka, ⁷Professor of Neuroanaesthesia, Bangabandhu Sheikh Mujib Medical University, Shahbag, Dhaka-1000

Corresponding Author: Dr. Jalal Uddin Ahmed, Associate Professor, Department of Anaesthesiology, Dhaka Dental College, email: <jalal@gmail.com>

Abstract

Background: Preemptive analgesia significantly reduces postoperative analgesic requirement. Multimodal approach for preemption has greater range of benefit and its use in treating postoperative pain has gaining popularity.

Objectives: To observe the effect of preemptive bupivacaine infiltration and intravenous ketorolac on postoperative pethidine consumption in single and multiple preemptive techniques.

Methods: One hundred and twenty patients of ASA physical status I&II requiring lower abdominal surgery under general anaesthesia were randomly allocated in four groups. Gr- A received bupivacaine infiltration and ketorolac injection, Gr-B received intravenous Ketorolac injection, Gr-C bupivacaine infiltration and Gr- D received nothing regarded as Control group. Data were analyzed by student's t test, ANOVA and chi-square test as appropriate.

Results: Patients characteristics, duration of surgery, haemodynamic status were comparable in four groups. Pain intensity in VAS was significantly reduced in Gr-A ($p < 0.00$) than other three groups. Total pethidine consumption was also reduced significantly in Gr-A ($p < 0.05$).

Conclusion: Preemptive analgesics using in multiple modes gives better postoperative analgesia and patient comfort and less postoperative complications.

Key words- multimodal approach, postoperative pain, analgesia, ketorolac, infiltration, bupivacaine

(Journal of BSA, 2010; 23(1): 25-29)

Introduction:

Pain is an extra ordinary complex sensation which is difficult to define and equally difficult to measure in an accurate objective manner. The International Association for the Study of Pain (IASP) defines pain as an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage.

Peripheral tissue injury provokes modification in the responsiveness of the nervous system. Peripheral sensitization, a reduction in the threshold of nociceptive afferent peripheral

terminal and central sensitization, an activity dependent increase in the excitability of spinal neurons; contributes together to post injury pain hypersensitivity state¹. Damaged tissue produces two phases of sensory input. First they are associated directly with tissue damaging stimulus i.e. during surgery. Secondly results from inflammatory reaction to damaged tissue. These causes release of wide range of chemicals that activate chemo sensitive afferent directly, sensitizes high threshold nociceptors. The phase of sensory input occurs post operatively during period of wound healing.

Post operative pain is a form of acute pain caused by noxious stimuli typically associated with neuro-endocrine stress response that is proportional to pain intensity². Moderate to severe acute pain regardless of site, affect nearly all organ function and may adversely influence postoperative pain management.

Postoperative pain can be reduced by pre-emptive analgesia. Pre-emptive analgesia implies analgesia directly as a result of reducing peripheral and or central sensitization. This type of management pharmacologically induces an effective analgesic state prior to surgical trauma by infiltration of the wound with local anaesthetics, central neural blockade or administration of opioids, NSAIDs or NMDA receptor agonist ketamine³.

Local anaesthetic act by binding to Na⁺ channel, it slows the rate of depolarization, so don't alter resting membrane potential or threshold level and ketorolac act by inhibiting cyclooxygenase, enzyme responsible for biosynthesis of prostaglandin, prostacyclin and thromboxane⁴. NSAIDs also attenuate hyperalgesic state caused by sensitization of afferent nerve fibres by prostaglandin⁵.

Materials and Methods:

A total number of one hundred and twenty female patients requiring lower abdominal surgery were studied. The purpose of the study were clearly explained to each of the subject and recruited only after they had given their written consent. Selected patients were female, age ranging from 20 to 55 years, weight ranging from 30 to 80 kgs, ASA grade I & II. Those who were with hypersensitivity to study drugs, peptic ulcer disease, gastritis, bleeding disorder, on anticoagulant therapy, respiratory, cardiac, hepatic, renal disease and patient not being motivated were excluded. They were divided into four groups, thirty in each group. Gr-A: bupivacaine infiltration and intravenous ketorolac after induction, Gr-B: intravenous ketorolac alone, Gr-C: bupivacaine infiltration after induction of anaesthesia and Gr- D: control group, no pre-emptive therapy.

All patients were examined pre-operatively and pre operative base line haemodynamic status was recorded. A 10cm visual analogue scale (VAS) slide roller and verbal rating scale were used to assess level of postoperative pain. VAS and VRS scale was explained to the patient that one extreme of scale indicates no pain and the other is worst possible

pain. PCA function was explained and pushing of PCA buttons practiced.

After pre-oxygenation for 3 minutes with 100% O₂, general anaesthesia was induced with thiopental sodium 5mg/kg intravenously and suxamethonium 2mg/kg to aid tracheal intubation. Anaesthesia was maintained with N₂O (66%), O₂ (33%) & Halothane (0.5%). No opioid was used during the operation. Competitive muscle relaxant vecuronium 0.1 mg/kg initially then 0.05mg/kg incremental doses was used for skeletal muscle relaxation. Residual effect of competitive muscle relaxant was antagonized with neostigmine 40µg/kg and atropine 20µg/kg at the end of skin closer. In the postoperative period intravenous patient controlled analgesia (PCA) machine was attached with IV line by three way extension tube. A loading dose of pethidine 30mg given intravenously and PCA dose adjusted to 10 mg with lock out interval of 30 minutes. Patient was assessed at immediate post operative period, 4 hour afterward, 12 hour afterward and 24 hour after starting of operation (time of incision considered as zero hour).

Post operative complications e.g. nausea, vomiting, pruritus, allergic rash, hypotension, bronchospasm and respiratory depression etc was also studied among different groups. Patient satisfactions e.g. excellent, good, fair and poor among the groups and within the groups were also studied.

All results were expressed as mean \pm SEM or as percentage as applicable. Mean \pm SEM was calculated for each of the observation in 30 patients in each group. The data yielded from this study were compiled and analyzed with the help of students' t test, Chi-square and ANOVA test as appropriate. A p value of <0.05 were considered as statistically significant.

Results:

One hundred and twenty female patients undergoing elective gynaecological surgery were included in this study. They were randomly allocated into four groups, 30 in each (Gr-A, Gr-B, Gr-C and Gr-D). They were matched for age (p=0.65), body weight (p=0.89) and ASA physical status (p=0.801). The mean age in Gr-A was 41.20 \pm 1.18 yrs, in Gr-B was 39.87 \pm 1.48, in Gr-C was 41.57 \pm 1.19 and in Gr-D 42.80 \pm 1.35. Body weight of in Gr-A was 56.57 \pm 1.79, in Gr-B 57.20 \pm 1.77, in Gr-C 55.17 \pm 1.95 and in Gr-D 56.80 \pm 2.36.

Table-1
Characteristics of the study population

Groups / variable	Age (years)	Body weight (kg)	Duration of Surgery (minutes)
Gr-A	41.20 ± 1.18	57 ± 1.79	103.43 ± 5.9
Gr-B	39.87 ± 1.48	57.20 ± 1.77	101.33 ± 5.5
Gr-C	41.57 ± 1.19	55.17 ± 1.95	99.10 ± 4.9
Gr-D	42.80 ± 1.35	56.80 ± 2.36	99.33 ± 4.

Values are expressed as mean ± SEM

Duration of surgery was recorded in minutes and it starts from skin incision to complete closure and strapping of the wound. In Gr-A total operation time was 103.43 ± 5.9, in Gr-B it was 101.33 ± 5.5, in Gr-C 99.10 ± 4.9 and in Gr-D 99.33 ± 4.0 and no statistical significance difference were found (p= 0.44).

Haemodynamic parameters

Heart rate (HR), mean blood pressure and arterial oxygen saturation (SpO₂) were recorded from the immediate postoperative period (recorded as zero time) to the next twenty four hours in the post-operative ward.

Heart rates:

Changes in the heart rate in the immediate post operative period, 4 hours afterward, 12 hour after operation and 24 hour after operation in the next morning were recorded and it were highly significant (P<0.05) in Gr-A than other three groups in all recoded period.

Mean Blood Pressure

Changes of mean blood pressure in all the four group scheduled intervals were not significant (P>0.05).

Arterial oxygen saturation was recorded also in same interval and there were no significant change in all four observation points (P>0.05).

Measurement of pain intensity:

Pain intensity was recorded by the help of a 10cm long visual analogue scale (VAS) in the predetermined time period. VAS score showed significantly reduced in Gr-A at all observation period (p<0.05) than other three groups.

First analgesic demand:

First analgesic demand was recorded after the operation in minutes. In Gr-A it was 407 ± 9.5, in Gr-B 147 ± 6.2, in Gr-C 298 ± 7.7 and in Gr-D 106 ± 8.4 minutes. It was highly significant in Gr-A than other three groups (P<0.00).

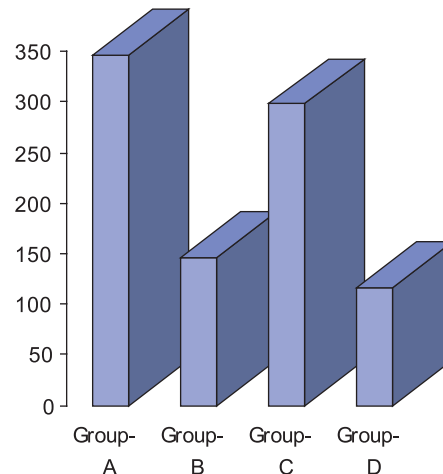


Fig-1: Time of first demand of analgesics in minutes

In the post operative period in 24 hours of post operative ward the total amount of pethidine consumed in the different groups were shown Fig.-2. They were analyzed by ANOVA test and showed a significantly reduced amount of pethidine consumed (p<0.05) in Gr-A than other three groups.

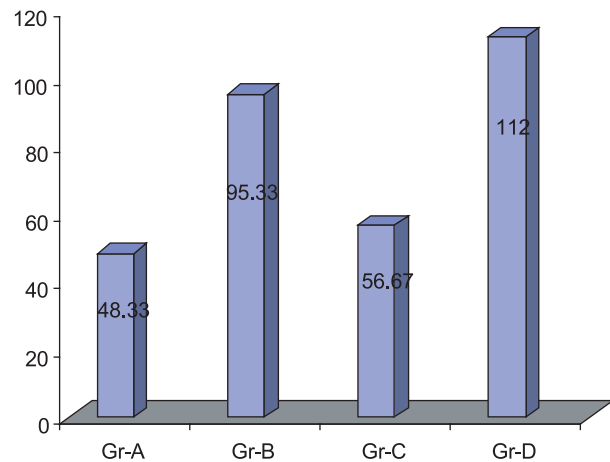


Fig-2: Total pethidine consumption in twenty four hour

Patient satisfaction- Degree of patient satisfaction among the groups and within the groups was recorded by verbal rating scale (VRS) as- excellent, good, fair and poor. The results were found as in Gr- A: Excellent 40%, Good 36.7%, Fair 23.3%, Poor-0%. In Gr-B: no patient graded as excellent, good- 6.75%, Fair- 36.7%, Poor- 56.70%. Gr. C- Exc. 6.7%, Good-60%, Fair- 30%, Poor- 3.3%. In Gr- D- Exc. 0%, Good-6.7%, Fair- 20%, Poor- 73.3%. They were analysed by ANOVA and showed

highly significant value ($p < 0.05$) in Gr-A than other three groups.

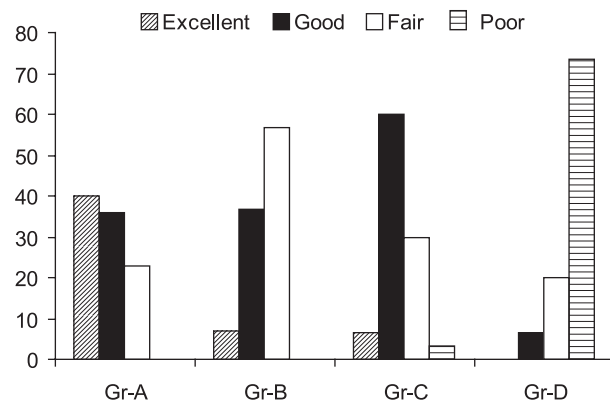


Fig-3: Patients Satisfaction in four studied groups

Postoperative complications:

Postoperative complications like nausea, vomiting, pruritus, hypotension, bronchospasm and respiratory depression in four groups were recorded in predetermined time interval. The complications were less in Gr-A, than other three groups and it was highly significant ($p < 0.05$).

Table-II

Distribution of different types of complications in different time of four studied groups

Groups n=30	Gr-A n=30	Gr-B n=30	Gr-C n=30	Gr-D n=30
Nausea and /or vomiting	10 (33%)	16 (53%)	14 (46%)	22 (73%)
Pruritus	-	2 (6%)	3 (10%)	4 (13%)
Bronchospasm	-	-	-	1 (3%)
Hypotension	-	1(3%)	2 (6%)	-
Respiratory depression	-	-	-	1(3%)
No complications	20 (67%)	11 (36%)	11 (36%)	2 (6%)

Data were expressed as number; within parenthesis are percentages over column total

Discussion:

Pre-emptive analgesia is an anti-nociceptive treatment that prevents establishment of altered processing of afferent input that amplifies pain. Concept of pre-emptive analgesia was formulated

on basis of clinical observation⁶. Later on series of studies on animal has been performed⁷⁻¹⁰. Pre-emptive treatments are directed in the periphery at inputs along sensory axis and at central nervous system by using NSAIDs, local anaesthetics and opioids, either alone or in combination. With these underlying principles, therapeutic intervention is made¹.

Many studies that favour and promotes the idea of pre-emption and others against led us to review the use of pre-emptive drugs prior to similar type of major surgical procedures in near about identical types of patients and to see the difference between single and multiple pre-emptions. The postoperative response was reviewed by pain scoring, first urge for analgesic, total analgesic consumption and together with vital parameters like heart rate, blood pressure, respiratory rate and SpO₂ up to the period of twenty four hours. We studied 120 patients randomly divided in 4 groups of 30 patients each. All cases performed lower abdominal surgery. Duration of operation was around 100 min. Four different groups received pre-emptive analgesic as per protocol. Time of first demand for analgesic was much later than other groups. Again total postoperative pethidine consumption was minimum in case of Gr- A in multiple pre-emptive groups than those receiving single pre-emptive. Those receiving no pre-emptive needed maximum postoperative opioids (Gr. D). Pethidine needed by bupivacaine only group (Gr. C) was also significantly less than group receiving ketorolac only (Gr. B). In our study, we have seen patients' maximum satisfaction with multiple pre-emptions than single pre-emption and pre-emption with bupivacaine infiltration gave better patient comfort than ketorolac pre-emption. Postoperative complication was seen more in patients receiving no pre-emption (Gr. D). Patients receiving pre-emption resulted in less postoperative pethidine consumption and less postoperative complication. Thus nausea, vomiting and respiratory depression were seen more in Gr. D.

Conclusions:

Under the condition of the present study, it can be concluded that pre-emptive application of analgesics using in multiple modes gives better post operative analgesia and patient comfort, less post operative complications than when used single analgesic technique.

References:

1. Clifford J Woolf, Mun Seng Chong. Pre-emptive analgesia-treating post operative pain by preventing the establishment of central sensitization. *Anaesth Analg* 1993; 77: 362-79.
2. Cousins MJ. Acute pain and the injury response; immediate and prolong effects. *Regional Anaesthesia* 1989; 14:162-178.
3. Morgan GE, Mikhail MS, Murray MJ. Pain management. *Clinical Anaesthesiology*. 3rd ed. New York, McGraw-Hill, 2002, 319.
4. Brooks PM, Day RO. Nonsteroidal anti-inflammatory drugs- differences and similarities. *N Engl J Med* 1991; 13:324: 1716-25.
5. Vickers M D, Morgan M, Spencer P S J. Systemic Analgesics, *Drugs in Anaesthetic and Intensive Care Practice*, 8th ed., Butterworth-Heinemann, 1999;
6. Crile GW. The kinetic theory of shock and its prevention through a nociception. *Lancet* 1913; 185: 7-16.
7. Woolf CJ, King AE. Physiology and morphology of multi-receptive neurons with C-afferent fibre inputs in the deep dorsal horn of rat lumbar spinal cord. *J Neurophysiol* 1987; 58: 460-79.
8. Woolf CJ, Thompson SWN, The induction and maintenance of central sensitization is dependent on NMDA receptor activation: implications for the treatment of post injury pain hypersensitivity states. *Anesthesiology* 1991; 44:293-9.
9. Woolf CJ. Recent advances in the pathophysiology of acute pain. *Br J Anaesth* 1983; 63:139-46.
10. Woolf CJ. Evidence for a central component of post injury pain and hypersensitivity. *The Lancet* 1983; 308:686-8.

Case Report

Rarest of rare bombay blood group in bangladesh: a case report

Tahmina Banu¹, S M Ali²

¹Associate Consultant, Department of Anaesthesia, Square Hospital Limited, ²Consultant, Department of Anaesthesia, Square Hospital Limited.

Corresponding author: E-mail: tbanu1960@yahoo.com

Summary:

A 35 years old lady was admitted to Square Hospital for termination of pregnancy on a medico-legal background. She was a diagnosed case of carcinoma of pancreas with Whipple's operation performed six months back and was on chemotherapy. During pre-operative check-up we surprisingly noticed that she has 'Bombay Blood Group'. It is a very rare type of blood group and on routine blood grouping behaves as "O" unless reverse grouping or serum grouping has been done and can receive transfusion from only peoples having Bombay group. As the patients general condition was poor and had recently received chemotherapy and also had a extremely rare blood group (I in 250000) we decided to keep ready one unit of blood. We decided to provide autologous blood transfusion, as there was no known person in family or in our blood banks record with Bombay Blood Group. The procedure was uneventful and needed no transfusion. As the patient was anaemic and weak, we decided to transfuse the blood in the post-operative period and the patient was discharged from the hospital next day morning.

Key words: *Bombay blood group, autologous blood transfusion.*

(Journal of BSA, 2010; 23(1): 34-36)

Introduction:

Bombay blood group comprises an immunologically distinct¹, genetically determined group of human erythrocytes characterized by lack of A, B and H antigens but having antibodies in the serum against all the above three antigens. It is called Bombay Blood Group; it was first discovered in Bombay, now known as Mumbai, by Dr. Y M Bhende in 1952¹. This group is a rare exception to the commonly accepted ABO blood types. It is observed to occur 1 out of every 250,000 people in the world except in parts of India (Orissa) where the incidence has been observed to be as much as 1 in 7600². People who have Bombay blood group can not donate blood to any member of the ABO group, they can not receive blood from any member of the ABO blood group. They can only receive blood other people who have Bombay phenotype. This group is commonly mistaken as O group and many times not identified at all because of lack of necessary technology in blood banks. Bombay blood

group differs from O group by lacking H antigen on RBC and could be Rh positive or negative³. If a Bombay blood group recipient is transfused with the blood other than Bombay, it can lead to a severe haemolytic transfusion reaction, which can be fatal and even lead to death⁴. Given that this condition is very rare, any person with this blood group who needs an urgent blood transfusion will probably be unable to get it, as no blood bank would have any in stock. Those anticipating the need for blood transfusion (e.g. in scheduled surgery) may stock their blood for their own use (i.e. an autologous blood transfusion)¹.

Case report:

A 32 years old lady was admitted at hospital for termination of 7wks of pregnancy on medicolegal ground. The patient had developed periampullary carcinoma of the pancreas and she went to Christian Medical College (CMC) at Vellore, where whipple's pancrea-ticoduodenectomy was done

followed by chemotherapy. She had taken six cycle of chemotherapy at a regular interval and the last dose was taken 4 weeks before her pregnancy. She has three children. During routine pre-anaesthetic check up, to our surprise we found that she is a patient with Bombay Blood Group. This was our first case with this rare blood group in Square hospital and a few more cases have been identified in Bangladesh. At CMC Vellore the patient was first detected as Bombay Blood Group. So two units of whole blood were collected from her at two weeks interval which were given during the whipple's operation. After finishing the last cycle of chemotherapy the patient became pregnant within one month. So the patient was advised for termination of pregnancy. Due to the rarity of the blood group and post chronic illness poor general condition we decided to keep one unit of blood ready and took it from the patient for autologous blood transfusion.

During preoperative visit her vital sign and investigation were, BP-110\65mm of Hg., pulse-96 beats \min Hb-11.6%, TC- 7.5-K/micro lit, platelet count -237 lacks. X-ray chest- NAD, ECG -NAD Test for liver function was normal. Patient was very weak and her general condition was poor.

Anaesthesia was induced with propofol and fentanyl and maintained with nitrous oxide, oxygen and isoflurane. The procedure was uneventful and blood loss was minimal. As the patient was very weak the whole blood was re-transfused during postoperative period. The patient was discharged on the next day morning with good vital sign.

Discussion:

There are four blood groups in the ABO system - A, B, AB, and O, and classification is based on the presence or absence of antigenic substance that appear on the surface of red blood cells. Both parents contribute to a child's blood type and the alleles that contribute to this are O, A and B⁵.

However, there are rare instances when a couple produces a type O child even if they don't possess any allele. If this situation occurs, the child possibly carries Bombay Blood Group in which absence of H antigen on the red blood cell surfaces. The H antigen is located on the surface of red blood cells and is the precursor of A and B antigen. The A allele is needed to produce a transferase enzyme to

modify the H antigen into A antigen. Likewise, the B allele is needed to make the transferase enzyme that would transform the H antigen into B antigen. For type O individuals, the H antigen cannot be transformed further because no H glycosyl transferase (FUT1) is produced to modify the antigen.

A person of the Bombay Blood Group inherited the recessive form of the allele for the H antigen from each of his parents. He carries the homozygous recessive (hh) genotype instead of the homozygous dominant (HH) or heterozygous (Hh) genotypes of the ABO blood group. As a result the H antigen is not expressed in the red blood cell surface; consequently; the A and B antigens are not formed. The h allele is a result of the mutation of the H gene (FUT1) that would express the H antigen in the red blood cells of ABO blood group^{6,7}.

People of the Bombay Blood Group produce antibodies against H, A, and B antigens to protect themselves. Since they have antibodies against H, A and B antigens, they can only receive blood donations from other people with Bombay Blood Group. Receiving blood transfusions from the ABO blood group can be fatal. The antibodies of the Bombay Blood group react with the red blood cells of the donor causing cell destruction. In the past many patients who were classified as type O by the ABO test died because doctors failed to test them for the Bombay blood type.

Conclusion:

Bombay phenotype is a rarest of rare blood group in the world but its frequency is relatively high in India especially on its eastern part (Orissa) and tribal areas where lot of marriages do occur amongst near relatives.

Generally there is no hindrance to normal living with Bombay blood group except when they need blood transfusion. For elective cases autologous transfusion is a good proposition, as in such cases we can plan ahead and collect blood from the patient herself well ahead of the operation. But in case of emergency it is a difficult proposition altogether. It will be very difficult to find Bombay blood group at ready stock in any blood bank. Blood bank can maintain a rare blood type donor file and develop exchange programs in times of need amongst themselves. Facilities for cryo-preservation can also be beneficial for rare blood groups.

If proper blood grouping or testing practices are not followed it can lead to people with Bombay blood group not being detected. This group would be categorized as the “O” group because it would not show any reaction to anti-A and anti-B antibodies just like a normal “O” group. When cross matching with “O” group is done, then it would show cross reactivity or incompatibility. Therefore reverse grouping or serum grouping has to be performed to detect this group and thereby avoid fatal transfusion reaction.

References:

1. Bhende YM , Deshpande CK , Bhatia HM , Sanger R , Race RR ,et al. 1952. A new blood group character related to the ABO system. *Lancet*; 1952; 1: 903-4.
2. Balgir. Detection of a rare “Bombay (OH) Phenotype” among the Kutia kundh primitive tribe of Orissa, India. *Int J Hum Genetics* 2005.
3. Mansoor QK, Bombay Blood group: A case report. *The Pacific Journal of Science and Technology* 2003; 1(1): 333-337.
4. Uady Foundation. Rare blood groups: Bombay blood group, Bombay blood type. http://www.rarebloodtypes.org/Bombay_blood_group.php, accessed on/2009.
5. Daniels G. *Human Blood Groups*, Second ed. Blackwell Science. 2002
6. Kelly RJ, Ernst LK, Larsen RD, Bryant JG, Robinson JS, Lowe JB. Molecular basis for H blood group deficiency in Bombay (Oh) and para-Bombay individuals. *Proc Natl Acad Sci USA* 1994.; 91 : 5843–7.
7. Kaneko M, Nishi. Wide variety of point mutations in the H gene of Bombay and para-Bombay individuals that inactivate H enzyme. *Blood* 1997; 90: 839–49.

Case Report

Unintentional epidural catheter migration to subarachnoid space followed by continuous spinal anaesthesia: a case report

K Sardar¹, AKMN Chowdhury², MK Rahman³

¹Assistant Professor, ²Professor and Head, ³Honourary senior consultant, Department of Anaesthesiology, BIRDEM, Dhaka

Corresponding author: email: kawsardr@yahoo.com

Abstract:

Among the complications of epidural anaesthesia catheter migration is a very rare one. A 45 years old lady was scheduled for repairing of post caesarean incisional hernia. We prefer the hanging drop technique for epidural space identification, and 3 ml air injection to reconfirm the epidural space. After a test dose of 2% lignocaine 2 ml with 10 microgram adrenaline, the catheter was secured with at 3 cm of its length within the epidural space. Immediately after test dose, she complained of lower limb motor lost. On monitor, bradycardia and severe hypotension was shown. Hemodynamic instability was corrected promptly. After proper resuscitation, we aspirate through epidural catheter. CSF was coming freely. We decided to continue with continuous spinal anaesthesia. We assembled a syringe pump. Continuous spinal anaesthesia was maintained with 0.125% bupivacaine @ 3ml/hour.

Key words: Epidural, catheter migration

(Journal of BSA, 2010; 23(1): 37-39)

Introduction:

Epidural anaesthesia is a central neuraxial block technique with many applications. The epidural space was first described by Corning in 1901, and Fidel Pages first used epidural anaesthesia in humans in 1921. In 1945 Tuohy introduced the needle which is still most commonly used for epidural anaesthesia. Both single injection and catheter techniques can be used. The advantages of epidural anaesthesia include a reduced perioperative stress response, lower blood loss, less postoperative analgesia, lower cost, early ambulation and oral intake, making it a good alternative to general anaesthesia¹. Among the complications of epidural anaesthesia hypotension, inadvertent high epidural block, local anaesthetic toxicity, total spinal, accidental dural puncture, epidural haematoma, infection, failure of block and catheter migration are remarkable².

Case report

A 45 years old lady was scheduled for repairing of post caesarean incisional hernia. Her

anthropometric measurement was- height 157 cm, weight 66 kg. She was coexisted with diabetes mellitus and hypertension. Her DM was controlled with insulin and there were no visible micro or macrovascular complications. Her HbA_{1c} and 24 hours blood sugar profile was within normal limit. Hypertension was controlled with single dose atenolol 50 mg. Cardiovascular renal and liver function was within normal limit. Lung function test shows mild restrictive disorder.

Premedication was done with 3mg bromazepam at night before surgery. She was fasted for 8 hours prior to surgery and morning dose of insulin was omitted but antihypertensive agent was given. On arrival at preoperative room EMLA was applied over the relevant site of skin (both for peripheral cannulation and epidural needle insertion). After 40 minutes an 18G intravenous cannula was inserted into the left cephalic vein near to wrist for administration of fluid and other medications. The patients were next brought to the operating

room where she underwent epidural anesthesia and surgery.

After preloading with 300 ml Ringer's lactate, the patient was made to sit on operation table with her neck flexed anteriorly and arms hanging by the side of the body and holding a pillow with abdomen. After skin disinfection, the T₇ spinous process was identified by the help of lower end of scapula. By counting caudally T₉-T₁₀ interspace was found and lignocaine 1% was infiltrated locally in this interspace. An 18-gauge Tuohy's needle was then introduced into the interspace in the mid-sagittal plane, and a drop of anesthetic solution was placed in its hub. Entry into the epidural space was heralded by feeling of "giving way" and immediate inward movement of the fluid drop from needle hub as a result of negative pressure in the epidural space. Localization of the epidural space was further confirmed by free movement of air (3 ml) injected through a resistance-free syringe.

We prefer the hanging drop technique for epidural space identification, and 3 ml air injection to reconfirm the epidural space, over the use of normal saline or 1% lignocaine. The bevel of the needle was directed cephalad in the epidural space, and an 18-gauge epidural catheter was placed overcoming the resistance with force during insertion. After a test dose of 2% lignocaine 2 ml with 10 microgram adrenaline, the catheter was secured with at least 3 cm of its length within the epidural space. Immediately after test dose, she complained of lower limb motor. On monitor, bradycardia and severe hypotension was shown. Hemodynamic instability was corrected promptly with atropine, ephedrine, 15° head down position and fluid load. After proper resuscitation, we aspirate through epidural catheter. CSF was coming freely. Surgeons refused to do the surgery. After counseling, surgeons agree to do the surgery. We decided to continue with continuous spinal anaesthesia. We assembled a syringe pump. Continuous spinal anaesthesia was maintained with 0.125% bupivacaine @ 3ml/hour. Twenty five mg of pethidine was given intravenously for mild sedation and to prevent shivering. Surgery was completed within hour without any pain experienced by the patient.

After surgery catheter was removed without prophylactic epidural blood patch. Analgesia was

maintained with intramuscular ketorolac 30mg combination with pethidine 100mg 8 hourly. Patient was followed up upto 7 days. She developed no remarkable complications.

Discussion

Catheter misplacement into the subarachnoid space is a rare complication having the incidence less than 1 in 1000³. Catheter is accidentally misplaced into the subarachnoid space either due to accidental dural puncture by epidural needle or catheter tip itself may penetrate dura. Total spinal is a rare complication occurring when the epidural needle, or epidural catheter, is advanced into the subarachnoid space without the operator's knowledge, and an "epidural dose" e.g. 10-20 ml of local anaesthetic is injected directly into the CSF. The result is profound hypotension, apnoea, unconsciousness and dilated pupils as a result of the action of local anaesthetic on the brainstem. The use of a test dose can prevent most cases of total spinal. In our case where the epidural initially appeared to be correctly sited, but subsequent test dose of 2ml 1% lignocaine caused the symptoms of motor block in both lower limbs. This has been ascribed to migration of the epidural catheter into the subarachnoid space, although the precise mechanism is uncertain.

In our case, it was due to catheter penetration through dura because after reaching the tip of Touhy needle in epidural space there was no CSF in the hub of needle. Partial tearing of dura by the tip of Touhy needle followed by catheter penetration is another possibility. Normally cerebrospinal fluid can be freely aspirated from the catheter. If, however, this is not recognized, large doses of anaesthetic may be delivered directly into the cerebrospinal fluid. This may result in a high block, or, more rarely, a total spinal, where anaesthetic is delivered directly to the brainstem, causing unconsciousness, seizures and sometimes even death.

The incidence of accidental dural puncture is about 1-3 in 100 insertions^{4,5}. The epidural space in the adult lower thoracic spine is only 3-4mm deep, which means it is comparatively easy to cross it and accidentally puncture the dura (and arachnoid) with the needle. It is more common in inexperienced hands. This may cause cerebrospinal fluid (CSF) to leak out into the epidural space, which

may in turn cause a post dural puncture headache (PDPH). This can be severe and last several days, and in some rare cases weeks or months. It is caused by a reduction in CSF pressure and is characterized by postural exacerbation when the patient raises their head above the lying position. When the headache is severe or unresponsive to conservative measures, an epidural blood patch may be used to treat the headache. This procedure is effective in treating approximately 90% of post dural puncture headaches. If unsuccessful, the blood patch may be repeated, and the success rate increases to 96% on the second attempt. The blood injected into the epidural space is thought to seal the hole in the dura. In our case, she developed no symptom of PDPH in postoperative follow up.

Catheter misplacement into a vein is very uncommon and incidence is less than 1 in 300⁶. Occasionally the catheter may be misplaced into an epidural vein, which results in all the anaesthetic being injected intravenously, where it can cause seizures or cardiac arrest in large dose⁷.

Conclusion

Test dose should be given routinely before giving full dose of drugs to prevent life threatening complications. Catheter migration into subarachnoid space does not need to convert to general anaesthesia to continue operation. But need more monitoring and attention of anaesthetist.

References

1. Kchict H. Stress response to surgery: release mechanisms and the modifying effect of pain relief. *Acta Chir Scand Suppl* 1988; 550: 22.
2. Bonnet F, Derosier JP, Pluskwa F, *et al.* Cervical epidural anaesthesia for carotid artery surgery. *Can J Anaesth* 1990;37:353–358.
3. Norris MC, Leighton BL, DeSimone CA. “Needle bevel direction and headache after inadvertent dural puncture”. *Anesthesiology* 1989; **70** (5): 729–31.
4. Sprigge JS, Harper SJ. “Accidental dural puncture and post dural puncture headache in obstetric anaesthesia: presentation and management: a 23-year survey in a district general hospital”. *Anaesthesia* 1980; **63** (1): 36–43.
5. Wilson IH, Allman KG. *Oxford handbook of anaesthesia*. Oxford: Oxford University Press. 2006. p. 20.
6. Clarkson CW, Hondeghem LM (April 1985). “Mechanism for bupivacaine depression of cardiac conduction: fast block of sodium channels during the action potential with slow recovery from block during diastole”. *Anesthesiology* 1985; **62** (4): 396–405.
7. Groban L, Deal DD, Vernon JC, James RL, Butterworth J. “Cardiac resuscitation after incremental overdosage with lidocaine, bupivacaine, levobupivacaine, and ropivacaine in anesthetized dogs”. *Anesth Analg*. 2001; **92** (1): 37–43.

Obituary



Dr. Shafiqul Islam was born in the month of September 22, 1959 at Bhaluka, Mymensingh. He got himself admitted in the Mymensingh Medical College in the year 1979 and passed MBBS from Mymensingh Medical College in the year 1986.

He belongs to the batch M-16 of Mymensingh Medical College. He obtained postgraduate Diploma in Anaesthesiology from Mymensingh Medical College in the year 2004.

He was promoted and posted as consultant Anaesthesiologist in Bhaluka in the year 2005. Later on he joined department of Anaesthesiology, Mymensingh Medical College as Assistant professor. In the department he was given the important task of further developing the anaesthesia department thereby improve the anaesthetic service.

He was an active member of Bangladesh Society of Anaesthesiologist and took active part in different activities of the society.

Dr. Saiful Islam, a good soul left this world on January 18, 2010 with massive cardiac arrest.