

VOLUME 31
NUMBER 2
JULY 2018

ISSN 2220-8992

Journal of the Bangladesh Society of Anaesthesiologists



Journal of The Bangladesh Society of Anaesthesiologists

Volume 31, No. 2, July 2018

OFFICIAL JOURNAL OF BANGLADESH SOCIETY OF ANAESTHESIOLOGISTS

EDITORIAL BOARD (2017 - 2019)

Editor-in-Chief

Prof. Md. Mozaffer Hossain

Executive Editor

Dr. Subrata Kumar Mondal

Associate Editors

Prof. Md. Abdul Hye
Prof. AKM Aktaruzzaman
Prof. Md. Abdul Khaleque Beg
Prof. Shamsul Alam
Prof. Monirul Islam
Prof. Md Shah Alam Bhuiyan
Prof. Kawsar Sardar
Dr. AKM Faizul Hoque

Advisory Board Members

Prof. KM Iqbal
Prof. M Khalilur Rahman
Prof. AKM Shafiqur Rahman
Prof. AYF Elahi Chowdhury
Prof. SM Jahangir
Prof. UH Shahera Khatun
Prof. Shahidul Islam
Prof. Sayed Golam Molla
Prof. Kamal Ibrahim
Prof. Brig. Gen (Retd) Razia Khanam
Prof. Nezamuddin Ahmed
Prof. AKM Azizul Hoque
Prof. ABM Muksudul Alam
Prof. AKM Nur Nobi Chowdhury
Prof. Wahiuddin Mahmud
Prof. Md. Abdur Rahman
Prof. Debabrata Banik
Prof. Latifur Rahman
Prof. Paresch Chandra Sarkar
Prof. Moinul Hossain

International

Graham J Arthurs
Prof Sayeeda Haider
Prof Anjan Datta

PUBLISHED BY

Dr. AKM Faizul Hoque on behalf of the
Bangladesh Society of Anaesthesiologists

ADDRESS OF CORRESPONDENCE

Dr. Subrata Kumar Mondal

Associate Professor, Dept. of Anaesthesiology
Dhaka Medical College and Hospital
Cell : +88-01715-437240,
E-mail : subrata326@yahoo.com,
Web : www.bsabd.com

EXECUTIVE COMMITTEE (2017 - 2019)

President

Dr. A.B.M Muksudul Alam

Senior Vice President

Dr. Paresch Chandra Sarkar

Vice President

Dr. Md. Mozaffer Hossain

Secretary General

Dr. Debabrata Banik

Joint Secretary

Dr. Kawsar Sardar

Organizing Secretary

Dr. AKM Faizul Hoque

Treasurer

Dr. Md. Abdul Hye

Scientific Secretary

Dr. Subrata Kumar Mondal

Members

Dr. M. Khalilur Rahman
Dr. A.K.M. Nur Nobi Chowdhury
Dr. Md. Abdul Karim
Dr. Muhammad Manzoarul Hoq Laskar
Dr. U.H. Shahera Khatun
Dr. Golam Ambia
Dr. Md. Abdur Rahman
Dr. Rabeya Begum
Dr. Md. Shah Alam Bhuiyan
Dr. Md. Mustafa Kamal
Dr. Atiqul Islam
Dr. Amir Hossain Rahat
Dr. Md Tanveer Alam
Dr. AJM Shafiul Alam Shah
Dr. ABM Sarwar Jahan
Dr. Mohammed Sharif Uddin Siddique

Branch members

Chittangong Branch

Dr. Gulshan Ara Chowdhury

Mymensingh Branch

Dr. A.N.M. Fazlul Hoq Pathan

Comilla Branch

Dr. Md. Mohsinuzzaman Chowdhury

Khulna Branch

Dr. Sheikh Farid Uddin Ahmed

Shylet Branch

Dr. Sabbysachi Roy

Rajshahi Branch

Dr. Md. Intakhab Alam

Rangpur Branch

Dr. Md. Sayedur Rahman

Bogra Branch

Dr. Md. Abdul Khaleque

Jessore Branch

Dr. AHM Ahsan Habib

SCIENTIFIC SUB COMMITTEE

Secretary Members

Dr. Subrata Kumar Mondal

Members

Prof. Mostafizur Rahman
Prof. Md. Mozaffer Hossain
Prof. Rubina Yasmin
Prof. Gulshan Ara Chowdhury
Prof. Debasish Banik
Dr. A.K. Qumrul Huda
Dr. Rabeya Begum
Dr. Sabina Yesmeen
Dr. AKM Faizul Hoque
Dr. Monstosh Kumar Mondal
Dr. Manash Kumar Basu
Dr. Hasina Begum
Dr. Muslima Begum
Dr. Atiqul Islam
Dr. Md. Nurul Islam
Dr. Md. Tanveer Alam

This is the **official journal** of the Bangladesh Society of Anaesthesiologists. JBSA starts its publication from 1987. It is published biannually in January and July each year and recognized by Bangladesh Medical and Dental Council (BMDC).

The Journal publishes original work in all branches of anaesthesia, intensive/ critical care, palliative care and pain medicine, including the application of basic sciences, clinical practice, equipment and training. In addition, the journal publishes review articles, case reports and special articles of general interest.

Printed at

Asian Colour Printing

130 DIT Extension Road, Fakirerpool, Dhaka
Tel: +8-02-49357726, 58313186
E-mail: asianclr@gmail.com
Web : acp1980.com

This journal is officially sponsored by **City Electro-Medics Co.** as part of their continuing educational support.

INFORMATION FOR THE AUTHORS

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 Authors are requested to visit the Web site www.icmje.org for this purpose.

Condition for publication of the articles:

The BSA Journal is intended to promote prompt publication of concise, scientific article based on the study in all fields of medical and health sciences.

- Manuscript submitted for printing in this journal must not be published in whole or in part in any other journal
- All submitted articles will undergo double blind peer review as per recommendations by subject specific experts selected by editors
- Reviewed manuscripts will be sent to the corresponding author for appropriate response if it is indicated
- Acceptance is based on significance, originality, clarity and fulfillment of the criteria of the publication policy of this journal.
- The Editor-in-Chief will take all final decisions regarding acceptance
- The editors reserves the right to style, if necessary to shorten the manuscript accepted for publication
- The editors also reserves the right to determine the priority and intended for publication of the reviewed and accepted manuscripts in a particular issue.
- Rejected manuscript will be returned if accompanied by stamped or self-addressed envelope
- Upon acceptance for publication the copy right of the paper automatically transfers to the BSA and will not be published elsewhere either in part or whole without written permission of the copy right holder
- Review article should be written by a subject expert
- It is not the duty of the editor to investigate scientific fraud paper.

Ethical aspects:

- Ethical aspect of the study will be very carefully considered at the time of assessment of the manuscript

- Articles on clinical investigation should abide by the ethical standards laid down in the 1964 Declaration of Helsinki revised 2000
- Manuscript must contain a statement in the method section that ail human subjects involved studies have been approved by appropriate ethical committee
- Use of names, initials etc. should be avoided so that patients must be unrecognizable
- Permission of the patients or their families to reproduce photographs of the patients where identity is not disguised
- Author should obtain written permission to reproduce any table, illustration from any other source
- About animal studies, the author should convince that the involved animals have not been subjected to pains or suffering not absolutely necessary for the sake of the finding.

Preparation of manuscript:

a. *Manuscript Organization:*

- Manuscript should be written in English
- Margin should be 5 cm for the header and 2.5 cm for the remainder
- Font type is Time New Roman with size 12
- Printed in double space on one side good quality A4 80 gm paper
- Manuscript should have uniform style, correct journal format, carefully proofread for grammar, spelling and punctuation.

b. *Manuscript format:*

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

- Title page
- Abstract and keywords
- Text
- Acknowledgement
- Table with titles
- References
- Attentively graph with title and illustration with legends
- Each of the sections is to start on a separate page. Pages should be numbered consecutively beginning from the title page.

Manuscript submission:

The authors are requested to strictly follow the guide lines below for submission of manuscript to JBSA for publication. The following documents with manuscripts are to be submitted for publication.

- A covering letter addressed to the Editor-in-Chief of the journal (Sample given at the end)
- A Title page (Sample given at the end)
- Abstract and key words in the first page followed by the text, prepared in the format of IMRAD
- Authors must submit 2 hard copies of all documents and one copy in electronic form preferably written in a CD-RW with adequate labeling
- In special case, submission through E-mail with file attachment of all documents is welcome

Covering letter:

- All authors must sign after seeing the manuscript with the statement that they are the only authors
- The corresponding author should mention the contribution of each author to the work
- It should contain a declaration that this manuscript has not been submitted elsewhere or not under consideration in any journal
- It should clearly indicate the publication type (Original/Review/Case report/Letter etc.)
- Should also mention the expected benefit of the medical science from publishing of this article

Title Page:

- The title page should be in a separate sheet which will include apart from the title of the article (concise but informative and self-explanatory), the name of all authors with their designation and institutional affiliation
- Name of the institute and department where the study was carried out
- Name of the corresponding author with contact address, telephone number, Email address
- Name and address of the authors to whom request for reprint should be addressed
- Number of tables given

- Number of figures given
- Word count of abstract
- Disclosure of sources of funding or sponsor
- A short running head having not more than 50 letters should also be suggested

Abstract and Keywords: The abstract should state the purposes of the study main findings and the principal conclusion.

- Structured with headings- Background, Objectives, Methods with statistical analysis, Result and Conclusion
- Preferably within about 250 words
- Avoid abbreviations except standard
- A non structured abstract is suggested for review article and case report
- Authors name should not be given
- Below the abstract provide and identify as such upto maximum 7 key words or phrases that will assist indexers in cross indexing. Use terms from the medical subject headings list of Index Medicus.

Text:

- The text of observational and experimental articles is usually but not necessarily should be arranged into following sections with the four main heading; Introduction, Methods, Results and Discussions.
- Other type of articles such as case reports, reviews and editorials are likely to need other formats.

Introduction:

- Provide a context or background for the study that is the nature of the problem and its significance
- Review of the literature directly related to the problem with pertinent reference
- Nature/rationale/objectives/hypothesis/benefits of the study expected stated in 1-2 paragraph.

Methods:

- This section should include only information that was available at the time the plan or protocol for the study was being written.
- Methods, subjects, grouping and variables 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.

- Approval of the study by ethical review committee
- Mention study type, place, time, selection and exclusion criteria
- Description of procedure, methods, apparatus, drugs or chemicals as applicable

Statistics:

- Statistical analysis should be described with enough detail to enable a knowledgeable reader with access to the original data to verify the reported results
- When possible, quantify finding and present them with appropriate indicators of measurement error
- Specify the computer software used

Results:

- All information obtained during the study should be described here and it should be in the text, table and illustration with the main or most important finding first
- Described without comment
- Results of the experiments should be brief and in logical sequence
- Total number of tables, charts and figures should be limited need to support assessment of paper
- Avoid unnecessary repetition of data, table and figure. Emphasize or summarize only the important observations
- Use graph as an alternative to tables with many entries

Table:

During preparation of tables following principle should be followed:

- All tables should be on separate sheets with proper captions and be simple, self explanatory and supplement, not duplicate the text
- They should be numbered consecutively using Roman numbers in order of text
- Units in which data are expressed should be given in brackets at the top of the relevant column
- Ditto signs, internal horizontal and vertical rules should not be used
- Uses of many tables are not encouraged

Illustrations:

Graph, chart, drawing, photographs etc. surfaces should not be marred by clips, pins or heavy writing on the back

- All illustrations must be clearly numbered consecutively in Arabic numerals as they appear in the text
- Submit print photograph of each illustration along with its electronic file
- Original drawing, graphs, charts and lettering should be prepared on an illustration board or high-grade white drawing paper by an experienced medical illustrator

Figures and photographs:

- Should be used only where data cannot be expressed in any other form
- Should be unmounted glossy print in sharp focus, 12.7 X17.3 cms in size
- Figure number, title of manuscript, name of the corresponding author and arrow indicating top should be written on a sticky label affixed on the back of each or on the back preferably with soft pencil

Discussion:

The discussion section should reflect:

- The authors comment on the results, important aspects of the study and the conclusion
- Explore possible mechanism or explanation for the findings
- The discussion should not merely recapitulate the results but compare and contrast the results with other relevant studies
- State the limitations of the study
- Explore the implication of the finding for future research and for clinical practice

Conclusion:

- Conclusion follows from the new and important aspects of the study in the context of the totality of the best available evidence
- Link the conclusion with the goals of the study but avoid unqualified statements and conclusions not adequately supported by the data

Legend:

- Must be typed in a separate sheet of paper
- Photomicrograph should indicate the magnification, internal scale and the method of staining.

- All drugs should be mentioned in their generic form. The commercial name may be used in parenthesis

Units:

- All scientific units should be expressed in System International (SI) units

Abbreviations and symbols:

- Use only standard abbreviations; use of non-standard abbreviations can be confusing to readers

Acknowledgement:

- Institution, organization, sponsor, individuals or any bodies can be acknowledged in the article for financial, material, technical or any form of assistance or contribution to the work with their consent.
- Acknowledgement should be placed at the end of the text before the references list.

References:

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 which is based on an **American National Standards Institute (ANSI)** style for its database adapted by the **National Library of Medicine (NLM)** and used in Index Medicus.
- References should be numbered consecutively in the order in which they are first mentioned in the text

- Identify references in the text, tables and legends by Arabic numerals in superscript
- All citations to electronic references should be presented in numbered references following the text
- The titles of the journals should be abbreviated according to the style used in Index Medicus
- Write names of 6 authors followed by et al. if authors' number is more than six.
- The reference list is not usually checked by the editorial staff or reviewer. It is the total responsibility of author to provide accurate information.

Examples:

- **Standard journal article:**
Coetzee GJ. Flow through disposable alternatives to the laryngeal mask. *Anaesthesia* 2003; 58:280-281
- **Standard book with initials for authors:**
Bowman WC. Pharmacology of Neuromuscular function. 2nd ed; London: Wright, 1990: 196-202.
- **Contributed chapter of a book:**
Carter EP, Wang Y, Campbell AR. Resolution of alveolar edema. In: Malthy MA, Ingbar DH (eds). Pulmonary edema. New York: Marcel Dekker, 1998
- **Standard journal article on the internet:**
Kaul S, Diamond GA. Good enough: a primer on the analysis and interpretation of noninferiority trials. *Ann Intern Med* [Internet]. 2006 Jul4 [cited 2007 Jan4]; 145(1):662-9
Available from : <http://www.annals.org/cgi/reprint/145/1/62.pdf>.

COVERING LETTER FOR SUBMISSION TO J B S A

Date.....

To

The Editor-in-Chief

Prof.....

Journal of the Bangladesh Society of Anaesthesiologists.

Sub: Submission of manuscript

Dear Sir,

We are submitting our manuscript entitled by 1.....2.....3.....for publication in your journal. This article **has not been published or submitted for publication elsewhere.**

We believe that this article may be of value to medical professionals engaged in Anaesthesiology/surgery. We are submitting 2 copies of manuscript along with an electronic version (CD).

We therefore, hope that you would be kind enough to consider our manuscript for publication in your journal as **Original/Review article/Case Report.**

Thanks and best regards

A (X)

Professor, Department of
BSMMU, Dhaka.

B (X)

Dr., Department of
BSMMU, Dhaka.

C (X)

Associate Professor, Department of.....
Chittagong Medical College, Chittagong.

D (X)

Assistant Prof., Department of.....
Dhaka Medical College, Dhaka.

TITLE PAGE OF ARTICLE

The title of the article.....

.....

1. Authors' names and institutional affiliations:

A (X) Professor, Department ofBSMMU, Dhaka.

B (X) Dr., Department ofBSMMU, Dhaka.

C (X) Associate Professor, Department of.....Chittagong Medical College, Chittagong.

D (X) Assistant Professor, Department of..... Dhaka Medical College, Dhaka.

2. The name of the department(s) and institution(S) to which the work should be attributed: Department of Rangpur Medical College, Rangpur. Bangladesh.

3. Disclaimers any: Yes, subject to nonprofit purpose

4. Corresponding author:

The name:.....

Mailing address:.....

Telephone:.....

E-mail address:

5. The name and address of the author to whom requests for reprint should be addressed:.....

Mailing address:.....

6. Source(s) of support in the form of grants, equipment, drugs or all of these- [Self finance]

7. A Short running head:.....

8. Word counts of abstract: []

9. The number of tables: []

10. The number of figures: []

Teamwork Practice In Operation Theatre for Patient Safety – Bangladesh perspective

Teamwork in operation theatre is the collaborative effort of a health professional to achieve a common goal or to complete a surgical task in most effective and efficient way (Safe Surgery). Safe surgery saves lives is a WHO program designed to avoid complication and death by improving peri-operative care. Peri-operative deaths solely due to anaesthesia or anaesthetic error are extremely uncommon (0.5-0.8:100,000), but the intra-operative death of anaesthetized patient is relatively more (1-30:100,000). There are also risk of development of complications in surgical patient at postoperative period. There are different action plan was taken for safe surgery and patient safety. Every action plan was developed by a group of individual responsibility but all action plan did not provide guarantee for safe surgery. In last 15 years surgical thinking about errors and patient safety has changed dramatically. With this background idea of teamwork was developed in worldwide. That effective teamwork, not only within the operating theatre but also across the entire peri-operative pathway, is acknowledged as a critical component of safe and effective surgical care, resulting in good outcomes and quick recovery of the surgical patient. So effective teamwork in the operating theatre is a necessity, not a luxury - better teams have better outcomes. Researchers have also made the distinction between task work and teamwork. Teamwork is defined as a non technical skill. This suggests that teamwork is not a task but a generic behavior. So components of teamwork are Communication, Performance and Facility support. Communication is supporting to get the necessary information to the right people so decisions can be made and achieve an interaction among members of the surgical team. Communication has two part- briefing and debriefing. WHO recognized and customized safe surgical checklist is a main communication tools for safe surgical teamwork. There are systematic review of 16 studies of surgical safety checklist

implementation in hospitals worldwide at 2014 noted that surgical checklists have been shown to significantly improve patient's outcome subsequent to surgery, and therefore their use is being widely encouraged and accepted. The evidence is also strong for briefings and debriefings. Patients whose surgical teams exhibited fewer teamwork behaviors were at higher risk for death or complications. Civil and Shuker noted that briefings and debriefings in the theatre environment have reduced communication failures by two-thirds, reduced non-routine events by one-fourth, effectively surfaced potential surgical safety hazards, reduced staff perception of risk and increased their sense of team collaboration and that article was published in the Australian and New Zealand Journal of Surgery, 2015. So it is obvious that there is a powerful link between routinely undertaking briefings and the safety culture within the operating theatre. With all these reasons recognition of the importance of teamwork for surgical patient safety is increasing. So significant efforts are taking place globally to improve team functioning through team training but it is still a common expectation of healthcare professionals of most of the developing countries are to maintain effective team functioning without significant effort in team training.

Present status in Bangladesh: It is a new increasing strategy in Bangladesh blame someone among the team or outside of team if go anything out of expectation specially in surgical task. This blaming an individual does not change the factors and the same error is likely recurring. To overcome this problem there is a demand for maintaining proper documentation by using safe surgical check list and developing well communication skill which are essential for teamwork approach.

Teamwork for surgical patient safety is a new concept among the surgical team in our country.

This idea has been introduced when surgical safety checklist practice are demanded. Most of the operation theatre staffs are doing their activities according to the usual instruction of surgeon and anesthetist. All of the operation theatre manpower in present condition do not act as a team rather than act individually and independently. Still safe surgical checklist is not properly followed in most of the hospital setup. For this reason quality cell of health ministry is very much concern about proper practice of safe surgical checklist for safety of surgical patient and avoidance of unwanted problem. This safe surgical checklist practice needs good communication and teamwork approach. Practice of communication before starting of any operation between team members and patient is very poor in different hospitals of Bangladesh. In the SAFE obstetric anesthesia course both anesthesiologist and obstetrician are being trained for better outcome of surgical obstetric patient including proper training regarding use of safe surgical checklist and communication skill. After completion of 6th SAFE obstetric anesthesia course outcome of this training was evaluated by a obstetric anaesthesia fellow of BSA in collaboration with Lederal group, WFSA. Most of the participants were involved in evaluation did not properly follow the safe surgical checklist and initial communication which are the essential components for teamwork concept.

How to practice teamwork concept in Bangladesh? In worldwide there are significant efforts have been made to understand how teams work within surgical care pathways and how to improve teamwork consequently. Availability of regular team training remains limited to a relatively small number of hospitals. Sometime this training driven by enthusiastic and committed individuals and often seen in response to an adverse event. But in Bangladesh 1st priority is to change the attitude of surgeon and anesthetist with the other worker in the operation theatre for practicing teamwork approach. All experts work together does not ensure that they will merge into an expert team. Essential component of teamwork are cooperation, communication, coordination, leadership and monitoring. So the importance of training of theatre team to work effectively together is gaining international acceptance as a

key strategy to maximize surgical safety. The development of team training interventions by the health ministry for health care provider of Bangladesh is very much essential. Aim of this training interventions are to provide theatre teams with the knowledge, skills, and attitudes (KSAs) that underpin effective team performance. After that it will become an increasingly prominent feature in the surgical, anaesthetic, and nursing literatures in Bangladesh. Team training may be embedded into the early stages of residency curriculum and giving emphasis on the advocacy efforts to aware the patient and the society regarding their role in safe surgery.

Conclusion:

Anaesthesia has been described as a leading medical specialty in addressing issues surrounding adverse events related to anesthesia. This discipline is first to discover and embrace CPR. They are giving training about teamwork in patient resuscitation and safe anesthesia for surgical patient for more than 20 years. Now Anesthesia department will develop awareness and lead the Teamwork training to the all theatre staffs for surgical patient safety.

(JBSA 2018; 31(2): 50-52)

Prof. Dr. Debabrata Banik

Dept. of Anaesthesia, Analgesia & Intensive Care Medicine, Bangabondhu Sheikh Mujib Medical University, Dhaka

References

1. Makary MA, Mukherjee A, Sexton JB, et al. Operating room briefings and wrong-site surgery. *Journal of the American College of Surgeons*. 2007; 204(2):236-43.
2. Sevdalis N, Hull L, Birnbach D. Improving patient safety in the operating theatre and perioperative care: obstacles, interventions, and priorities for accelerating progress. *Br J Anaesth*. 2012;109 Suppl. 1:i3-16
3. Amaya Arias AC, et al. Efectividad de un programa para mejorar el trabajo en equipo en salas de cirugía. *Rev Colomb Anestesiología*. 2014.
4. Salas E, Guthrie JW, Wilson-Donnelly KA, Priest HA, Burke CS. Modeling team

- performance: the basic ingredients and research needs. In: Rouse WB, Boff KR, editors. *Organizational simulation*. New York: John Wiley & Sons, Ltd; 2005. p. 185-216.
5. Salas E, Cooke NJ, Rosen M. On teams, teamwork, and team performance: discoveries and developments. *Hum Factors*. 2008;50:540-7.
 6. West MA. *Effective teamwork-practical lessons from organizational research*. West Sussex: British Psychological Society and John Wiley & Sons, Ltd; 2012.
 7. Vincent C. *Patient safety*. West Sussex: Wiley-Blackwell; 2010.
 8. Hull L, Arora S, Symons NR, Jalil R, Darzi A, Vincent C, Sevdalis N. Training faculty in nontechnical skill assessment: national guidelines on program requirements. *Ann Surg*. 2013;258:370-5.
 9. Reason J. Human error: models and management. *BMJ* 2000;320:768-770.
 10. Healey AN, Undre S, Vincent CA. Developing observational measures of performance in surgical teams. *Qual Saf Health Care* 2004;13(Suppl 1):i33-i40.
 11. Calland J, Guerlain S, Adams R. *et al* A systems approach to surgical safety. *Surg Endosc* 2002;16:1005-1014.
 12. Vincent C, Moorthy K, Sarker SK. *et al* Systems approach to surgical quality and safety: from concepts to measurements. *Ann Surg* 2004;239:475-482.
 13. Baker DP, Gustafson S, Beaubien J. *et al* Medical teamwork and patient safety: the evidence based relation. Literature review. AHRQ Publication No. 05 0053. Rockville, MD: Agency for Healthcare Research and Quality (AHRQ), 2005.
 14. Xiao Y, Seagull FJ, Mackenzie CF. *et al* Adaptation of team structure of trauma resuscitation teams. *Proceedings of the Human Factors and Ergonomics Society 46th Annual Meeting 2002*. 569-573.
 15. Gladstein D. Groups in context: a model of group task effectiveness. *Admin Sci Q* 1984;29:499-517.
 16. Leonard M, Graham S, Bonacum D. The human factor: the critical importance of effective teamwork and communication in providing safe care. *Qual Saf Health Care* 2004;13(Suppl 1):i85-i90.
 17. Lingard L, Espin S, Whyte S. *et al* Communication failures in the operating room: an observational classification of recurrent types and effects. *Qual Saf Health Care* 2004;13:330-334.
 18. Undre S, Sevdalis N, Healey AN. *et al* Teamwork in the operating room: cohesion or confusion? *J Eval Clin Pract* 2006;12:182-189.
 19. Militello LG, Kyne MM, Klein G. *et al* A synthesized model of team performance. *Int J Cognit Ergon* 1999;13:1131-1158.
 20. Fletcher G, Flin R, McGeorge P. *et al* Anaesthetists' non technical skills (ANTS): evaluation of a behavioural marker system. *Br J Anaesth* 2003;90:580-588.
 21. Lingard L, Reznick R, Espin S. *et al* Team communications in the operating room: talk patterns, sites of tension, and implications for novices. *Acad Med* 2002;77:232-237.

Ultrasound Guided Nerve Stimulation and Nerve Stimulation Alone for Supraclavicular Brachial Plexus Block – A Randomized Comparative Study

Mehdi Hassan^{1*}, AKM Akhtaruzzaman², AKM Faizul Hoque³, Rezwanur Rahman⁴, Sabina Yeasmeen⁵, Debabrata Banik⁶

¹Medical Officer, ²Professor, ³Associate Professor, ⁴Medical Officer, ⁵Associate Professor, ⁶Professor and Chairman, Department of Anaesthesia, Analgesia & ICU, Bangabandhu Sheikh Mujib Medical University, Shahbag, Dhaka.

Corresponding Author: e-mail: mehdi hassanmmmc@yahoo.com

Abstract:

Background: The safety of regional anaesthesia become more pronounced by the use of ultrasound and nerve stimulator. Supraclavicular nerve blocks known as ‘spinal of the arm’ are the most attractive upper extremity blocks to perform in our practice. In this study less experienced hands try to found the best approach for upper extremity block.

Objective: To compare the success rate when Ultrasound added with Peripheral nerve stimulator in supraclavicular brachial plexus block.

Methods: After IRB approval and written consents from patients, total 66 patients divided into two groups, Group USNS had supraclavicular block guided by both ultrasound and Nerve stimulator. On the other hand Group BNS had this block by only Nerve stimulator. All the equipments kept ready and maintaining sterility a mixture of 0.5% Bupivacaine and 2% plain Lignocaine were prepared. The amount injected according to the body weight without crossing the toxic dose (2mg/Kg 0.5% Bupivacaine, 5mg/Kg 2% Lignocaine). Total volumes were 25-30ml for every patient. The sensory block was assessed by observers who unaware of the technique for every 2 minutes till the onset of block and every 10 minutes thereafter for 30 minutes. Any failure in establishing the block was converted to GA. The sensory dermatomes were assessed by alcohol swab. The motor blocks were evaluated by the same observer in each joint for every 2 minutes till onset than 10, 20 & at the end of 30 minutes. Successful block was considered if no supplementation or conversion to general anaesthesia required.

Results: In all demographic variables and ASA Class, there was no differences in between the USNS group and BNS group. In group USNS block execution time was significantly higher ($P < 0.05$). The time required for both sensory and motor block was statistically significantly less in Group USNS compared to Group BNS (P value < 0.05). Regarding quality of motor block, at wrist joint statistical significance present between two groups (p value < 0.05). The duration of analgesia is significantly lower in Group BNS than Group USNS (P value is 0.012). In Group USNS, only one (3.03%) patient needed supplementation. But in Group BNS 7 (21.21%) patients needed supplementation. According to the definition, these cases were regarded as failed case. The success rate is significantly higher in Group USNS (P value is 0.024).

Conclusion: Combined use of ultrasound and peripheral nerve stimulator increases success rate than peripheral nerve stimulator alone in supraclavicular brachial plexus block. This combined method also reduces block execution time, early onset of both sensory and motor block, improve quality of sensory and motor block and less incidence of complications.

Keywords: Supraclavicular Block, Ultrasound guide, Peripheral nerve stimulator.

Introduction:

Regional anaesthesia is a well accepted modality to achieve both economic and clinical benefits during the peri-operative period. The key to successful regional anaesthesia is to deposit the local anaesthetics as near as possible to the nerve structures. To achieve this, electrical stimulation by nerve stimulators or paresthesia (Blind technique) are being used, both of which relied on surface landmark identification. However, landmark techniques have limitations that includes variations in anatomy ¹ and nerve physiology ² as well as equipment accuracy have had an effect on success rates and complications.

Supraclavicular nerve blocks known as 'spinal of the arm' are the most attractive upper extremity blocks to perform in our practice. According to Kulenkampff and Persy, (1928) ³ in the early 20th century, the supraclavicular approach to the brachial plexus provides more effective and consistent regional anaesthesia to the upper extremity than other approaches to brachial plexus blockade. This block is ideal in providing a rapid onset, dense and efficient anaesthesia and analgesia for procedures from mid humerus proximally to those performed on the hand distally however the potential risk for pneumothorax and injury to surrounding structures had decrease its popularity. To locate peripheral nerves during the initiation of nerve blocks using peripheral nerve stimulator, with a low-intensity electrical current has become common practice in regional anaesthesia. The ability of a peripheral nerve stimulator to produce a motor response depends on the distance of the stimulus from the nerve (*i.e.*, the needle-to-nerve distance), as well as the intensity and duration of the current that has been set on the device ⁴. Every device have a defined sensitivity and specificity. The peripheral nerve stimulation device arises potential false negative response when needle is in the correct perineural position but there is no corresponding motor response. This failure rate will result an inappropriate needle repositioning and the potential for unnecessary nerve injury and discomfort for the patient. It is found in a previous literature that 13.5% of the time, nerve stimulation failed to elicit a motor response despite the ultrasound confirmation of correct needle location during the performance of a supraclavicular block

⁵. The needle tip may be located intra-neurally, intravascularly, or on the other side of the fascia ⁶. Moreover, any part of the axon may be depolarized and may propagate an action potential. So it is not possible to decide with certainty where the local anaesthetics are being injected. Besides, upon a visually confirmed needle-nerve contact, paresthesia is felt by only 38% of the patients and an electrical stimulation of 0.5mA elicits a visible muscle twitch only in 75% of them ⁷. Therefore, a visual control of needle advancement in real time could improve needle placement and outcome precisely.

Ultrasound guided regional anaesthesia offers several potential advantages and this supported by many literature. For example, direct visualization of nerve trunks under ultrasound helps accurate localization. Likewise, it enables direct visualization of anatomical structures that is vessels, muscles, bones, fascias, tendons. This may help to assess individual variations in anatomy and facilitate identification of nerves correctly. Also, real-time control of needle advancement may reduce repeated needle penetration, block performance time and other potential complications *e.g.*, vascular puncture, pneumothorax or neuropraxia. Assessment of local anaesthetics spread around the nerves can be done and immediate supplementary injections in case of insufficient spread can be possible. This may improve block effectiveness, shorten latency, prolong duration, allow local anaesthetics dose reduction and lower the risk of overdose ⁸. Ultrasound frees the operator from using the anatomical landmarks. Nerves can be targeted at any point along their course where they can be seen. 'Blind techniques' rely on clicks, pops and twitches needing multiple trial and errors. Drawbacks like needle passes with lack of accuracy and reliability, longer placement times, patient discomfort and injury, can be avoided with imaging help. The aim of this study was to compare different parameters between ultrasound and peripheral nerve stimulator guided supraclavicular block with peripheral nerve stimulator guided block alone.

Methods:

This randomized study has been conducted in Anaesthesiology department of Bangabandhu

Sheikh Mujib Medical University after approved by the ethical review board of this hospital and also written informed consent obtained from all patients. This study was done on 66 adult patients, age above 18 yrs, male or female belonging to ASA I or II. Randomization was done by computer generated randomization technique with internet based software. (<http://www.randomizer.org/form.htm>). Total patient divided into two groups- Group USNS for Ultrasound guided nerve stimulation and Group BNS for only nerve stimulation. After entering into the Block execution place all basic monitoring were attached and with 18G cannulation port Inj. Prochlorperazine (0.25mg/Kg) then Inj. PethidineHydrochloride (0.5mg/kg) were injected. No other sedative or analgesics were used till evaluation of block up to 30 minutes. If even after 30 minutes block was not adequate for surgery, were supplemented. All these blocks were executed by, Residents who were in training phase for Ultrasound and Nerve stimulator guided regional anaesthesia, supervised by consultant anaesthesiologist.

Group USNS:

In this group patients were in supine position with 45° head up and tilted to opposite side. A pillow has placed below the shoulder & head in a way that operator could have sufficient space for USG probe movement. For all the cases SonositeMicromax HFL linear 38 probe (6-13 MHz) was used. Sterility of the probe maintained by a sterile plastic cover and Povidone Iodine was used as coupling agent. The probe placed in a coronal oblique plane to the supraclavicular fossa and tried to visualized subclavian artery and Brachial plexus in relation to the artery. Other spatial structures had been scanned to avoid injury to important structures. Next skin of the selected site was anaesthetized with 1-2ml of 1% Lignocaine. Then Nerve Stimulator attached with 20G 50mm stimulating needle and inserted from lateral to medial direction through in plane view. When needle entered between lower part of the plexus, stimulation been given at 0.5mA and observe the response. After negative aspiration 2/3 of total volume of drugs injected then repositions the needle at the upper part of the plexus to inject remaining 1/3 of the volume and spread of the drugs observed. When necessary needle reposition done to achieve adequate spread.

Group BNS:

In this group the positive electrode of the nerve stimulator was attached to an ECG led and

stacked with a suitable site. Patient position was supine and head tilted to the opposite side and operator stand at the head end of the patient. Then identify the lateral boarder of the sternocleidomastoid muscle by raising patient's head. After that rolled the index finger to identify Interscalene groove and in that groove go inferiorly to pulpatessubclavian artery and mark a point of needle entry. With 2ml of 1% lignocaine anaesthetized that point of needle entry. A 20G 50mm insulated needle attached with the negative electrode of the stimulator and inserted through the marked point to anteroposterior direction towards ipsilateral nipple. Nerve stimulator was initially set at 1.5mA, 0.1ms with SENSE output and tried to elicit distal motor response. To obtain this end motor response needle repositioning had to be done. After end motor response, the current reduced till the presence of muscle twitch with 0.5mA and no twitch with a current of 0.2mA. The drugs than injected intermittently after negative aspiration.

Block execution time recorded for both groups. The sensory and motor block was assessed by observers who unaware of the technique for every 2 minutes till the onset of block and every 10 minutes thereafter for 30 minutes. Any failure in establishing the block was converted to GA. The sensory dermatomes were assessed by alcohol swab. The motor blocks were evaluated by the same observer in each joint⁹ for every 2 minutes till onset than 10, 20 & at the end of 30 minutes.

Statistical analyses was carried out by using the Statistical Package for Social Sciences version 20.0 for Windows (SPSS Inc., Chicago, Illinois, USA). A descriptive analysis was performed for all data. The mean values were calculated for continuous variables. The quantitative observations were indicated by frequencies and percentages. The parametric data was analyzed by Student "t" test and the nonparametric data was analyzed by Chi-square test. *P* value < 0.05 consider as significant.

Result:

Observations of this study were analyzed in the light of comparison among the subjects (Group USNS and Group BNS, each group having sample size of 33). All results were expressed as mean±SD or in frequencies or percentages as applicable. Statistical significance was considered if *p* value is <0.05. The studied groups became statistically matched for age, weight, sex, ASA class.

Table-I shows mean age distribution of the sample cases in Group USNS and Group BNS were 39.21 ± 15.33 and 38.88 ± 17.65 respectively. The mean weight of Group USNS was 61.67 ± 10.19 kg and of Group BNS was 63.82 ± 12.26 kg. The mean height of the patients in Group USNS was 1.64 ± 0.09 meters and in Group BNS was 1.60 ± 0.11 meter. In Group USNS, male female ratio was 1:0.57 and in Group BNS it was 1:0.5. In the Group USNS, 24 (72.73%) patients were in ASA I and 9 (27.27%) were in ASA II. Again, in Group BNS, 22 (66.67%) were in ASA I and 11 (33.33%) in ASA II status. In all demographic variables and ASA Class, there were no differences in between the USNS group and BNS group.

Table-I Demographic characteristics and ASA class of the cases.

Variables (N=66)		Groups		P value
		Group USNS n=33	Group BNS n=33	
Age (in years)		39.21 ± 15.33	38.88 ± 17.65	0.94
Weight (in kg)		61.67 ± 10.19	63.82 ± 12.26	0.44
Height (in meter)		1.64 ± 0.09	1.60 ± 0.11	0.15
BMI (in kg/m^2)		22.89 ± 3.27	24.88 ± 4.57	0.05
Sex	Male	21 (63.66%)	22 (66.66%)	1.00
	Female	12 (36.36%)	11 (33.33%)	
ASA Class	I	24 (72.73%)	22 (66.67%)	0.79
	II	9 (27.27%)	11 (33.33%)	

Values are expressed in numbers (percentage) and mean $\pm$ standard deviation. In case of Age, weight, height and BMI, P values are calculated by independent sample t test method. In case of sex and ASA class, P values are calculated by Chi square test.

Figure 1 shows block execution time in Group USNS was 403.09 ± 66.63 seconds and in Group BNS was 468.12 ± 83.35 seconds. This is statistically significant. The calculated P value is 0.001.

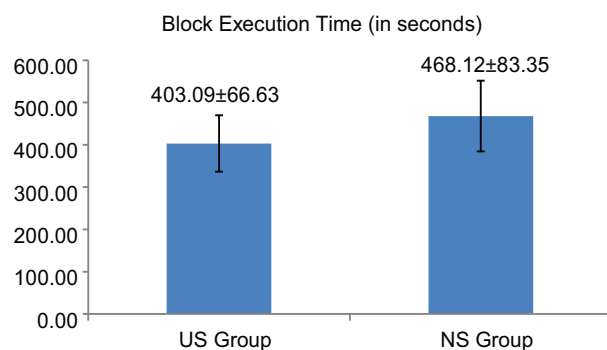


Fig 1: Block Execution Time

Table-II represents distribution of cases according to onset of Sensory block expressed as mean $\pm$ SD, and p value less than 0.05 are considered statistically significant. The time required for onset of sensory block in C5 dermatome of Group USNS was 3.64 ± 1.29 min, in Group BNS it was 4.92 ± 1.77 min and p value is 0.003 which imparted statistical significance. Likewise, in C6 dermatome of Group USNS was 3.81 ± 1.28 min, in Group BNS it was 5.83 ± 2.50 min and p value is 0.001 which imparted statistical significance. In C7, C8 and T1 dermatomes of Group USNS sensory onset time were 4.56 ± 1.70 min, 5.19 ± 1.82 min and 5.38 ± 1.72 min and in Group BNS 6.17 ± 2.50 min, 6.83 ± 0.35 min and 7.08 ± 2.28 min respectively. In each of the mentioned dermatomes, onset of sensory block is significantly lower in Group USNS than Group BNS.

Table-II Onset of Sensory Block in different dermatomes between two groups

Dermatome	Group USNS (n=33)	Group BNS (n=33)	P Value
	(in min)	(in min)	
C5	3.64 ± 1.29	4.92 ± 1.77	0.003
C6	3.81 ± 1.28	5.83 ± 2.50	0.001
C7	4.56 ± 1.70	6.17 ± 2.50	0.006
C8	5.19 ± 1.82	6.83 ± 0.35	0.005
T1	5.38 ± 1.72	7.08 ± 2.28	0.002

Values are expressed in mean $\pm$ SD. P values are calculated by Independent sample t test

Table III shows number of cases whether blocks have been achieved or not along each dermatome. In Group USNS, only one (3%) patient showed patchy block in C8 dermatome and one (3%) patient showed no block in T1 dermatome. All other dermatome showed no deviations after 30 minutes of onset of sensory block. But in Group BNS, 3 (9.1%) patients had no block in all of the dermatomes, one (3%) patient showed patchy sensation in C5, C6, C7 and C8 dermatome and six (18.2%) patient showed patchy sensation along the T1 dermatome after 30 minutes of onset of sensory block which was significantly higher than Group USNS (P value is 0.013).

Table IV represents distribution of cases according to onset of Motor Block expressed as mean $\pm$ SD. In Group USNS, the onset of motor block was found 3.82 $\pm$ 1.45 minutes in shoulder joint, 4.36 $\pm$ 1.62 minutes in elbow joint and 4.97 $\pm$ 1.94 minutes in wrist joint. In Group BNS, the onset of motor block was found 5.93 $\pm$ 1.96 minutes in shoulder joint, 6.69 $\pm$ 2.09 minutes in elbow joint and 7.86 $\pm$ 2.56 minutes in wrist joint. In every joint, the time required for block was statistically significantly less in Group USNS compared to Group BNS (P value < 0.05).

Table-III Distribution of cases according to Quality of Sensory Block.

	Group USNS (n=33)			Group BNS (n=33)			P value
	Blocked	Patchy	No Block	Blocked	Patchy	No Block	
C5	33	0	0	29	1	3	0.114
C6	33	0	0	29	1	3	0.114
C7	33	0	0	29	1	3	0.114
C8	32	1	0	29	1	3	0.355
T1	32	0	1	24	6	3	0.013

Values are expressed in number. P values are calculated by Chi square test

Table-IV Onset of Motor Block between two groups at different joints.

Joints to evaluate	Groups		P value
onset of motor block	Group USNS (n=33) (in minutes)	Group BNS (n=33) (in minutes)	
Shoulder	3.82 $\pm$ 1.45	5.93 $\pm$ 1.96	0.015
Elbow	4.36 $\pm$ 1.62	6.69 $\pm$ 2.09	0.023
Wrist	4.97 $\pm$ 1.94	7.86 $\pm$ 2.56	0.014

Values are expressed in mean $\pm$ SD. P values are calculated by Independent sample t test

Table V represents distribution of patients according to quality of Motor block. In Group USNS, only one (3.03%) patient showed patchy block over the elbow joint and another one (3.03%) had no block over the wrist joint. But in Group BNS, it was found that 3 (9.09%) patient had no block over all three joints, one (3.03%) had patchy block over shoulder joint, 2 (6.06%) over elbow joint and 3 (12.12%) had patchy block over wrist joint. In shoulder and elbow joint there is no statistical significance present but at wrist joint statistical significance present between two groups (p value < 0.05).

Table-V Quality of Motor Block between two groups at different joints.

	Group USNS (n=33)			Group BNS (n=33)			P value
	Blocked	Patchy	No Block	Blocked	Patchy	No Block	
Shoulder	33	0	0	29	1	3	0.114
Elbow	32	1	0	28	2	3	0.197
Wrist	32	0	1	26	4	3	0.054

Values are expressed in number. P values are calculated by Chi square test

Figure 2 shows that in Group USNS, the mean duration of analgesia was 236.36 ± 20.89 min and in Group BNS it was 201.82 ± 73.59 . The duration of analgesia is significantly lower in Group BNS than Group USNS (P value is 0.012).

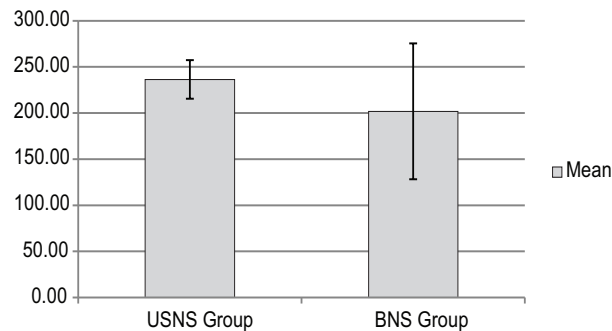


Figure 2 Distribution of patients according to duration of analgesia

Table VI represents distribution of patients according to incidence of complication. In Group USNS, there was no complication. But in Group BNS, in 4 (12.12%) patient complication (all were vascular injury) were present. Although this data is not statistically significant (0.114).

Table VI Complications between Group USNS and Group BNS.

	Group		Total	P value
	USNS (n=33)	BNS (n=33)		
No Complication	33 (100%)	29 (87.88%)	62 (93.93%)	0.114
Complication	0 (0%)	4 (12.12%)	4 (6.06%)	

Values are expressed in numbers and percentage over column total. P value is achieved by Chi Square test

Table VII shows distribution of patient according to success rate. In Group USNS, only one (3.03%) patient needed supplementation. But in Group BNS 7 (21.21%) patients needed supplementation. According to the definition, these cases were regarded as failed case. The success rate is significantly higher in Group USNS (P value is 0.024).

Table VII Success rate (according to operational definition) between two groups.

	Group		Total	P value
	USNS (n=33)	BNS (n=33)		
Failed Block	1 (3.03%)	7 (21.21%)	8 (12.12%)	0.024
Successful Block	32 (96.97%)	26 (78.79%)	58 (87.88%)	

Values are expressed in numbers and percentage. P value was calculated by Chi Square test

Discussion:

For the last decade, the use of real time ultrasonography guided peripheral nerve block has been revitalized as there has been rapid improvement in transducer device, lessening of cost, availability and advancement of portable ultrasonogram device. The rapid evolution of the ultrasonogram device enables its more elaborate use in the field of regional anaesthesia resulting in escalation of use of previously unpopular techniques like supraclavicular brachial plexus block due to visualization of plexus and its relationship with surrounding vessels, first rib and pleura.

The demographic variables in this study have imparted no statistical significance in between the two groups. The block execution time, expressed as mean $\pm$ SD, for cases in Group USNS was 403.09 ± 66.63 seconds and in Group BNS was 468.12 ± 83.35 seconds. This was statistically highly significant as the calculated P value is 0.001. A review from Liu et al. (2009)¹⁰ comparing US guided versus NS guided techniques, observed similar results in expert hand. The faster performance in ultrasound guidance blockade can be explained logically. Ultrasonography enables the performer to visualize the location, the spatial anatomy and ascertain the size and position of the plexus. Also it visualizes the needle, enabling its positioning and repositioning under direct real time vision. However, PNS guided technique is a blind technique needing speculation rather direct visualization of the surrounding structures and needle position.

In this study, the onset of sensory blockade was examined in each dermatome and found significantly lower in every dermatome as

compared to PNS guided group. In the series of Williams et al. (2003)¹¹ it was showed that significant decrease in the onset of blockade time in the USG group. Also in Lo et al. (2008)¹² reported that reduction of onset time when compared to PNS guided axillary blockade. The early onset of sensory blockade can be explained by the fact that, under direct vision of ultrasound, local anaesthetics can be placed very near to the nerve plexus.

In Group USNS, only one (3%) patient showed patchy block in C8 dermatome and one (3%) patient showed no block in T1 dermatome. All other dermatome showed no deviations after 30 minutes of onset of sensory block. But in Group BNS, 3 (9.1%) patients had no block in all of the dermatomes, one (3%) patient showed patchy sensation in C5, C6, C7 and C8 dermatome and six (18.2%) patient showed patchy sensation along the T1 dermatome after 30 minutes of onset of sensory block. In the series of Duncun et al. (2013)⁹ it was reported that complete anaesthesia at 30 minutes was achieved more reliably and rapidly in the C6, C7, C8 and T1 dermatomes compared to C5 dermatomes. They had found no statistical significance in the degree of sparing of the C5 dermatome between the Group US and Group NS. Interestingly, in this study, sparing of T1 occurred more in Group BNS with statistical significance than that of Group USNS, probably due to confirmation of drugs distribution around lower trunks (corner pocket) in Group USNS.

In Group USNS, the onset of motor block was found 3.82 ± 1.45 minutes in shoulder joint, 4.36 ± 1.62 minutes in elbow joint and 4.97 ± 1.94 minutes in wrist joint. In Group BNS, the onset of motor block was found 5.93 ± 1.96 minutes in shoulder joint, 6.69 ± 2.09 minutes in elbow joint and 7.86 ± 2.56 minutes in wrist joint. In every joint, the time required for block was statistically significantly less in Group USNS compared to Group BNS (P values < 0.0001). This result was comparable to the study of Chan et al. (2003)¹³ where the onset of motor blockade in ultrasound guided group was 5.40 ± 1.80 minutes. Though in present study all blocks executed by a trainee but had faster motor onset, may be because of adequate drug distribution around the plexus which could not be confirmed by blind neurostimulation technique.

This study also examined the quality of motor block at the shoulder, elbow and wrist in each

group. In Group USNS, only one (3.03%) patient showed patchy block over the elbow joint and another one (3.03%) over the wrist had no block. But in Group BNS, it was found that 3 (9.09%) patient had no block over all three joints, one (3.03%) had patchy over shoulder joint, 2 (6.06%) over elbow joint and 3 (12.12%) had patchy block over wrist joint. After statistical analysis it is found that at wrist joint statistical significance present which may be due to equal distribution of drugs in Group USNS.

In Group USNS, the mean duration of analgesia was 236.36 ± 20.89 minutes and in Group BNS it was 201.82 ± 73.59 minutes. The duration of analgesia is significantly lower in Group BNS than Group USNS (P value is 0.012). In the series of Thomas et al. (2011)¹⁴ the duration of analgesia is indifferent in both groups. But in the series of Duncan, et al. (2013)⁹ the duration of analgesia was much higher in both groups compared to this study which was 429.5 ± 90.79 minutes in Group US and 401.1 ± 105.65 minutes in Group NS. Importantly, their series indicated a shorter duration of analgesia in Group NS which was comparable to this study. Their longer duration can be explained by using different drugs combination during anaesthesia.

Reasonably, the Group USNS has fewer complication rates in this study. In Group USNS, there was no complication. But in Group BNS, in 4 (12.12%) patients complication (all were vascular injury) were present. This was in line with the series of Thomas et al. (2011)¹⁴, Rupera et al. (2013)¹⁵ and with Liu et al. (2013)¹⁶. In every above mentioned series, the complication rates were fewer or nil in the Group US compared to Group NS. Marhofer et al. (1998)¹⁷ also reported improved safety profile in USG guided three in one block than PNS guided block. This is because of real time visualization of vessels by ultrasound device.

Success rate in this study results was 96.97% in Group USNS as compared to Group BNS which was 78.79%. This result was comparable to the study of Williams et al. (2003)¹¹, who reported success rate of 85% in Group USNS and 78% in Group BNS. In the series of Rupera, et al. (2013)¹⁵ success rate in Group USNS was 96.67% and in Group BNS was 80%. In this present study success rate is significantly higher in Group USNS because

all procedure performed by trainee and for a trainee it may take less experience and less time to learn ultrasonoguided plus PNS stimulated brachial plexus block than landmark guided PNS stimulated brachial plexus block.

Under the condition of present study, it can be concluded that combined use of ultrasound and peripheral nerve stimulator increases success rate than peripheral nerve stimulator alone in supraclavicular brachial plexus block. This combined method also reduces block execution time, early onset of both sensory and motor block, improve quality of sensory and motor block and less incidence of complications.

References:

1. Retzl G, Kapral S, Greher M, Mauritz W. Ultrasonographic findings of the axillary part of the brachial plexus. *Anesthesia & Analgesia*. 2001 May 1;92(5):1271-5.
2. Perlas A, Niazi A, McCartney C, Chan V, Xu D, Abbas S. The sensitivity of motor response to nerve stimulation and paresthesia for nerve localization as evaluated by ultrasound. *Regional anesthesia and pain medicine*. 2006 Sep 1;31(5):445-50.
3. Kulenkampff D. Brachial plexus anaesthesia: its indications, technique, and dangers. *Annals of surgery*. 1928 Jun;87(6):883.
4. Pither CE, Raj PP, Ford DJ. The Use of Peripheral Nerve Stimulators for Regional Anesthesia A Review of Experimental Characteristics, Technique, and Clinical Applications. *Regional Anesthesia and Pain Medicine*. 1985 Apr 1;10(2):49-58.
5. Beach ML, Sites BD, Gallagher JD. Use of a nerve stimulator does not improve the efficacy of ultrasound-guided supraclavicular nerve blocks. *Journal of clinical anesthesia*. 2006 Dec 1;18(8):580-4.
6. Thompson GE. The Labat Lecture 1997 From Pauchet to Today The French Connection. *Regional anesthesia and pain medicine*. 1998 Mar 1;23(2):126-33.
7. Selander D, Edshage S, Wolff T. Paresthesiae or no paresthesiae? Nerve lesions after axillary blocks. *Acta Anaesthesiologica Scandinavica*. 1979 Feb 1;23(1):27-33.
8. Marhofer P, Greher M, Kapral S. Ultrasound guidance in regional anaesthesia. *British journal of anaesthesia*. 2004 Jul 26;94(1):7-17.
9. Duncan M, Shetti AN, Tripathy DK, Roshansingh D, Krishnaveni N. A comparative study of nerve stimulator versus ultrasound-guided supraclavicular brachial plexus block. *Anesthesia, essays and researches*. 2013 Sep;7(3):359.
10. Liu SS, Ngeow JE, YaDeau JT. Ultrasound-guided regional anesthesia and analgesia: a qualitative systematic review. *Regional anesthesia and pain medicine*. 2009 Jan 1;34(1):47-59.
11. Williams SR, Chouinard P, Arcand G, Harris P, Ruel M, Boudreault D, Girard F. Ultrasound guidance speeds execution and improves the quality of supraclavicular block. *Anesthesia & Analgesia*. 2003 Nov 1;97(5):1518-23.
12. Lo N, Brull R, Perlas A, Chan VW, McCartney CJ, Sacco R, El-Beheiry H. Evolution of ultrasound guided axillary brachial plexus blockade: retrospective analysis of 662 blocks. *Canadian Journal of Anesthesia*. 2008 Jul 1;55(7):408-13.
13. Chan VW, Perlas A, Rawson R, Odukoya O. Ultrasound-guided supraclavicular brachial plexus block. *Anesthesia & Analgesia*. 2003 Nov 1;97(5):1514-7.
14. Thomas LC, Graham SK, Osteen KD, Porter HS, Nossaman BD. Comparison of ultrasound and nerve stimulation techniques for interscalene brachial plexus block for shoulder surgery in a residency training environment: a randomized, controlled, observer-blinded trial. *The Ochsner Journal*. 2011 Sep;11(3):246-52.
15. Rupera KB, Khara BN, Shah VR, Parikh BK. Supra-Clavicular Brachial Plexus Block: Ultra-Sonography Guided Technique Offer Advantage Over Peripheral Nerve Stimulator Guided Technique. *Natl J Med Res*. 2013;3(3):241-4.
16. Liu SS, Ngeow JE, YaDeau JT. Ultrasound-guided regional anesthesia and analgesia: a qualitative systematic review. *Regional anesthesia and pain medicine*. 2009 Jan 1;34(1):47-59.
17. Marhofer P, Schrögender K, Wallner T, Koinig H, Mayer N, Kapral S. Ultrasonographic guidance reduces the amount of local anesthetic for 3-in-1 blocks. *Regional anesthesia and pain medicine*. 1998 Nov 1;23(6):584-8.

Effectiveness of plethysmographic variability index for prediction of subarachnoid block induced hypotension in caesarean section

Md. Abdul Alim¹, AKM Habibullah¹, Ranjit Kumar Ghosh², Ranjit Ghosh³, Debabrata Banik⁴, Akm Akhtaruzzaman⁵

¹Consultant, Department of Anaesthesia, Analgesia and Intensive care, Bangabandhu Sheikh Mujib Medical University, Dhaka, ²Medical Officer, Department of Internal Medicine, Bangabandhu Sheikh Mujib Medical University, Dhaka, ⁴Assistant Professor, Department of orthodontics, Bangabandhu Sheikh Mujib Medical University, Dhaka, ⁴Professor, ⁵Professor and chairman, Department of Anaesthesia, Analgesia and Intensive care, Bangabandhu Sheikh Mujib Medical University, Dhaka

Corresponding Author: Dr. Md. Abdul Alim, Consultant, Department of Anaesthesia, Analgesia and Intensive care, Bangabandhu Sheikh Mujib Medical University, Dhaka

Abstract

Background: Hypotension is frequently observed after spinal anaesthesia for cesarean section and can be detrimental to both mother and baby. The pleth variability index (PVI) is a new algorithm used for automatic estimation of respiratory variations in pulse oximeter waveform amplitude, which might predict fluid responsiveness. Because anaesthesia-induced hypotension may be partly related to patient volume status. The pleth variability index (PVI) was developed as a noninvasive bedside measurement of this variation in the pulse oximetry waveform.

Objective: To observe the hypotension predictive capacity of PVI and to find out association & correlation of PVI with sphygmomanometric blood pressure measurement.

Methods: This observational study was carried out in the department of anaesthesia, Analgesia and Intensive Care Medicine Bangabandhu Sheikh Mujib Medical University, Dhaka between July 2015 to Dec 2015. A total 100 elective caesarean section patients under subarachnoid block were selected by the inclusion and exclusion criteria. Patients who fulfill the ASA physical status i, ii. and full term singleton pregnancy height from 152cm to 160cm. were included and patients suffering from obesity (body weight > 115 kg), hypertension, COPD, bronchial asthma, haemoglobinopathies, severe anaemia, arrhythmia, heart failure, any congenital heart disease, pre-eclampsia, total placenta praevia or patient who took anti hypertensive medications were excluded from the study. Patients were divided in two groups, PVI ≥ 22.0 in group-A and PVI < 22.0 in group-B.

Dehydration was corrected 10 min before sub arachnoid block (sab). Pre-anesthetic Hemoglobin% SPO₂, Heart rate, PVI & blood pressure was recorded at baseline after 5 minutes of rest by one anesthesiologist. Subarachnoid block performed with 0.5% hyperbaric bupivacaine (12.5 mg) at the L₃-L₄ intervertebral space on sitting position. After spinal block patient was returned to supine position with a wedge under buttock to facilitate left uterine displacement. Oxygen 4 lit/min was administered via face mask. Immediately after sub arachnoid block SpO₂, heart rate, SBP and DBP was recorded by another anesthesiologist at 2 minutes interval in first 10 minute. Surgical incision was allowed when a block level at least T₆ dermatome was obtained with cold & pin prick.

All data was recorded by two anesthesiologist who were not involved in the study. The study ended with delivery of the baby. Chi-Square test was used to analyze the categorical variables, shown with cross tabulation. Student t-test was used for continuous variables. p value < 0.05 was considered as statistically significantly.

Result: In baseline, majority (58.0%) patients was found PVI ≥ 22 (group A) and 42(42.0%) was PVI < 22 (group B). Mean age of the patients was 27.5 $\pm$ 4.5 years, Mean heart rate was found 93.2 $\pm$ 5.8 beats/min in group A and 89.7 $\pm$ 12.7 beats/min in group B. The mean systolic BP was found 132.1 $\pm$ 7.7 mmHg in group A and 128.7 $\pm$ 8.5 mmHg in group B. The mean diastolic BP was found 80.9 $\pm$ 3.8 mmHg in group A and 79.1 $\pm$ 5.1 mmHg in group B. The mean MAP was found 98.0 $\pm$ 5.6 mmHg in group A and 95.6 $\pm$ 6.8 mmHg in group B. The mean SPO₂ was found 97.8 $\pm$ 1.4 in group A and 97.4 $\pm$ 1.5 in group B. The mean perfusion index was found 5.0 $\pm$ 2.6 in group A and 5.4 $\pm$ 3.5 in group B. The mean pleth variability index

was found 22.5 ± 2.3 in group A and 15.1 ± 3.1 in group B. The mean pleth variability index was statistically significant ($p < 0.05$) between two groups.

Conclusion: Higher baseline pleth variability index can associated with hypotension after spinal anaesthesia for cesarean section may be a clinically useful predictor.

Key words: Plethysmographic variability index(PVI), subarachnoid block, hypotension

(JBSA 2018; 31(2): 62-66)

Introduction:

Sub arachnoid block is one of the central neuroaxial blocking technique of involving injection of a local anaesthetic within the subarachnoid space at the level of lower lumbar vertebrae. The sympathectomy produced by subarachnoid block induces hemodynamic changes. Arterial and venodilation both occur in SAB and combine to produce hypotension. Hypotension is frequently observed after sub arachnoid block(SAB) in caesarean section and can be detrimental to mother as well as fetus. Severe or sustained hypotension may lead to cardiac arrest & irreversible brain damage. However no easy and convenient indexes to predict hypotension before subarachnoid block have been reported. Masimo Pulse CO-Oximetry is a Motion and Low Perfusion pulse oximeter. pleth variability index (PVI) is non-invasive and convenient dynamic indicator of fluid responsiveness which can be used to detect risk for mean arterial pressure (MAP) decreases and consequently hypotension during sub arachnoid block(SAB).¹

Plethysmograph variability index or PVI, is a new introduction of noninvasive measurement that quantifies changes in the plethysmographic waveform. It is a graphical display of the changes in blood volume caused by arterial pulsation over the respiration cycle. So, PVI may be useful in monitoring surgical patients, both intraoperatively and postoperatively, for appropriate hydration states & blood pressure. For example, a rising PVI may indicate developing hypotension. PVI is appropriate to use to predict fluid responsiveness in most ICU and surgical patients. In general, PVI provides an accurate prediction of fluid responsiveness in mechanically ventilated adults under general anesthesia with a normal sinus rhythm. PVI is less accurate and therefore not recommended for patients with cardiac arrhythmia for the same reasons.

Methods: This observational study was carried out in the department of anaesthesia, Analgesia and Intensive Care Medicine Bangabandhu Sheikh Mujib Medical University, Dhaka between July 2015 to Dec 2015 after the ethical clearance from institutional review board of BSMMU. Informed written consent was obtained from each patient before enrolling in this study. A total 100 elective caesarean section patients under subarachnoid block were selected by the inclusion and exclusion criteria. Patients who fulfill the ASA physical status *i*, *ii*. and full term singleton pregnancy height from 152cm to 160cm. were included and patients suffering from obesity (body weight > 115 kg), hypertension, COPD, bronchial asthma, haemoglobinopathies, severe anaemia, arrhythmia, heart failure, any congenital heart disease, pre-eclampsia, total placenta praevia or patient who took anti hypertensive medications were excluded from the study. Patients were divided in two groups, PVI ≥ 22.0 in group-A and PVI < 22.0 in group-B.

Dehydration was corrected 10 min before sub arachnoid block (sab). Pre-anesthetic Hemoglobin% SPO₂, Heart rate, PVI & blood pressure was recorded at baseline after 5 minutes of rest by one anesthesiologist. Subarachnoid block performed with 0.5% hyperbaric bupivacaine (12.5 mg) at the L₃-L₄ intervertebral space on sitting position. After spinal block patient was returned to supine position with a wedge under buttock to facilitate left uterine displacement. Oxygen 4 lit/min was administered via face mask. Immediately after sub arachnoid block SpO₂, heart rate, SBP and DBP was recorded by another anesthesiologist at 2 minutes interval in first 10 minute. Surgical incision was allowed when a block level at least T₆ dermatome was obtained with cold & pin prick.

All data was recorded by two anesthesiologist who were not involved in the study. The study ended with delivery of the baby. Statistical analyses of the results were obtained by using window based

computer software devised with Statistical Packages for Social Sciences (SPSS-16). Chi-Square test was used to analyze the categorical variables, shown with cross tabulation. Student t-test was used for continuous variables. p value <0.05 was considered as statistically significantly.

Results:

Table I Distribution of the study patients by baseline parameters (n=100)

Baseline parameters	Mean±SD
Age (in years)	27.5±4.5
Heart rate (beats/min)	90.4±9.3
SpO ₂	97.7±1.9
Hb%	11.7±0.9
BMI (kg/m ²)	24.5±6.5
Perfusion index	5.2±3.0
Pleth variability index	19.7±4.5
Systolic blood pressure (mmHg)	129.3±8.4
Diastolic blood pressure (mmHg)	80.2±3.7
Mean arterial pressure (mmHg)	96.6±4.6

Mean age was found 27.5±4.5 years, mean heart rate was found 90.4±9.3 beats/min, mean SpO₂ was found 97.7±1.9, mean Hb% was found 11.7±0.9 gm/dl, mean BMI was found 24.5±6.5 kg/m², mean perfusion index was found 5.2±3.0, mean pleth variability index was found 19.7±4.5, mean systolic blood pressure was found 129.3±8.4 mmHg, mean diastolic blood pressure was found 80.2±3.7 mmHg and mean MAP was found 96.6±4.6 mmHg.

Table II Classification of patients according to PVI (n=100)

Baseline PVI parameters of	No. of patients	Percentage
≥22 (group A)	58	58.0
<22 (group B)	42	42.0

In baseline, majority (58.0%) patients was found PVI ≥22 (group A) and 42(42.0%) was PVI <22 (group B).

Table III Preoperative patients status (n=100)

Baseline parameters	Group A (n=58)	Group B (n=42)	P value
Heart rate (beats/min)	Mean±SD 93.2±5.8	Mean±SD 89.7±12.7	0.074 ^{ns}
Systolic blood pressure (mmHg)	132.1±7.7	128.7±8.5	0.052 ^{ns}
Diastolic blood pressure (mmHg)	80.9±3.8	79.1±5.1	0.057 ^{ns}
Mean arterial pressure (mmHg)	98.0±5.6	95.6±6.8	0.056 ^{ns}
SPO ₂	97.8±1.4	97.4±1.5	0.174 ^{ns}
Perfusion index	5.0±2.6	5.4±3.5	0.533 ^{ns}
Pleth variability index	22.5±2.3	15.1±3.1	0.001 ^s

s= significant, ns= not significant

P value reached from unpaired t-test

Mean heart rate was found 93.2±5.8 beats/min in group A and 89.7±12.7 beats/min in group B. The mean systolic BP was found 132.1±7.7 mmHg in group A and 128.7±8.5 mmHg in group B. The mean diastolic BP was found 80.9±3.8 mmHg in group A and 79.1±5.1 mmHg in group B. The mean MAP was found 98.0±5.6 mmHg in group A and 95.6±6.8 mmHg in group B. The mean SPO₂ was found 97.8±1.4 in group A and 97.4±1.5 in group B. The mean perfusion index was found 5.0±2.6 in group A and 5.4±3.5 in group B. The mean pleth variability index was found 22.5±2.3 in group A and 15.1±3.1 in group B. The mean pleth variability index was statistically significant (p<0.05) between two groups.

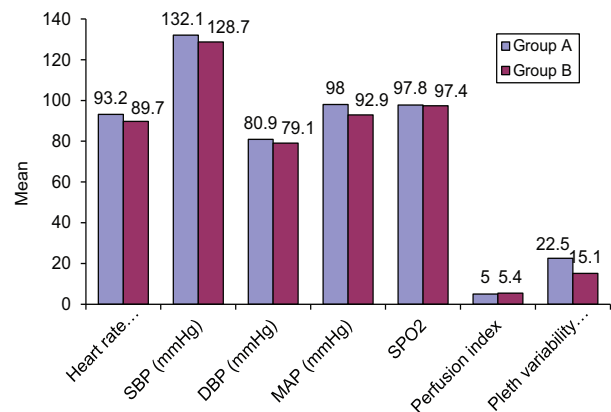


Fig 1 Bar diagram showing comparison of baseline parameters between group A and group B.

The mean pleth variability index was statistically significant (p<0.05) between two groups.

Table-4 Systolic blood pressure dropped >20% and critical dropped >30% of baseline (n=100)

	Group A(n=58)		Group B(n=42)		P value
	n	%	n	%	
Hypotension (SBP dropped >20% of baseline)	47	81.0	13	31.0	0.001 ^s
Hypotension (SBP critically dropped >30% of baseline)	39	67.2	11	26.2	0.001 ^s

s= significant

P value reached from chi square test

Table-5 Positive and negative predictive value of baseline PVI for hypotension after spinal anesthesia

Baseline PVI	Hypotension after induction	No hypotension after induction	Total
Positive (≥ 22.0)	47(True positive)	11(False positive)	58
Negative (< 22.0)	13(False negative)	29(True negative)	42
Total	60	40	100

In baseline, majority (81.1%) patients was found systolic blood pressure dropped >20% mmHg in group A and 13(31.0%) in group B. More than two third (67.2%) patients was found systolic blood pressure critically dropped >30% in group A and 11(26.2%) in group B. The difference were statistically significant ($p < 0.05$) between two groups.

Baseline PVI, true positive 47 cases, false positive 11 cases, false negative 13 cases and true negative 29 cases in identification by hypotension after spinal anesthesia.

Discussion:

In this present study it was observed that mean age was found 27.5 ± 4.5 years, mean heart rate was found 90.4 ± 9.3 beats/min, mean SpO_2 was found 97.7 ± 1.9 , mean Hb% was found 11.7 ± 0.9 gm/dl, mean BMI was found 24.5 ± 6.5 kg/m², mean perfusion index was found 5.2 ± 3.0 , mean pleth variability index was found 19.7 ± 4.5 , mean systolic blood pressure was found 129.3 ± 8.4 mmHg, mean diastolic blood pressure was found 80.2 ± 3.7 mmHg and mean MAP was found 96.6 ± 4.6 mmHg. Similarly, Sun & Huang (2014) found mean age was 29.3 ± 6.1 years, weight 74.3 ± 9.3 kg, height 162 ± 6.1 cm, BMI 29.2 ± 7.6 kg/m², perfusion index 5.6 ± 0.6 and pleth variability index was 20.6 ± 5.9 . In another study Tsuchiya et al. (2010) found Pre-anesthesia PVI varied from 7 to 28, with a mean value of 16 ± 5.5 .

In this current study it was observed that in baseline, majority (58.0%) patients was found PVI ≥ 22 (group A) and 42(42.0%) was PVI < 22 (group B). The mean hemoglobin was found 11.8 ± 1.2 gm/dl in group A and 11.4 ± 0.9 gm/dl in group B. The mean hemoglobin was almost alike between two groups.

In this study it was observed that mean heart rate was found 93.2 ± 5.8 beats/min in group A and 89.7 ± 12.7 beats/min in group B. The mean systolic BP was found 132.1 ± 7.7 mmHg in group A and 128.7 ± 8.5 mmHg in group B. The mean diastolic BP was found 80.9 ± 3.8 mmHg in group A and 79.1 ± 5.1 mmHg in group B. The mean MAP was found 98.0 ± 5.6 mmHg in group A and 95.6 ± 6.8 mmHg in group B. The mean SPO_2 was found 97.8 ± 1.4 in group A and 97.4 ± 1.5 in group B. The mean perfusion index was found 5.0 ± 2.6 in group A and 5.4 ± 3.5 in group B. The mean pleth variability index was found 22.5 ± 2.3 in group A and 15.1 ± 3.1 in group B. The mean pleth variability index was statistically significant ($p < 0.05$) between two groups. Similarly, Wrench et al. (2015) found that the PI was higher during spinal anaesthesia in women who became hypotensive compared with those who did not ($P < 0.05$). Sun & Huang (2014) observed that there was no significant difference in baseline hemodynamic parameters between patients who developed hypotension after spinal anesthesia compared with patients who did not. Baseline PVI in patients who developed hypotension was

significantly greater than PVI in patients who did not develop hypotension ($P = 0.017$) but there was no difference in the baseline PI. In another study Toyama et al. (2013) found that baseline PI ranged from 0.7 to 8.6, with a mean value of 4.0 (2.3). Twenty-one parturients (60%) developed hypotension; the maximum decrease in SAP from baseline ranged from 9.1–55.1%, with a mean value of 28.7%. Yokose et al. (2013) observed that pre-anesthetic PVI of patients who developed hypotension and who did not were $20.3 \pm 6.3\%$, and $16.8 \pm 5.3\%$, respectively ($P < 0.05$), which are comparable with the current study.

Yokose et al. (2015) found the mean perfusion index was found 6.1 ± 3.3 in group A and 5.9 ± 2.2 in group B. The mean pleth variability index was found 18.4 ± 6.6 in group A and 17.8 ± 4.9 in group B. The mean heart rate was found 84.0 ± 10 bpm in group A and 77 ± 13 bpm in group B. The difference were not statistically significant ($p > 0.05$) between two groups, which is comparable with the current study.

Baseline PVI was significantly related to the incidence of hypotension, whether the covariates were adjusted or not. Neither baseline PVI nor baseline PI were significantly related to the magnitude of the decrease in SBP (Sun & Huang 2014). In this present study it was observed that in baseline, majority (81.1%) patients was found systolic blood pressure dropped $>20\%$ mmHg in group A and 13(31.0%) in group B. More than two third (67.2%) patients was found systolic blood pressure critically dropped $>30\%$ in group A and 11(26.2%) in group B. The difference were statistically significant ($p < 0.05$) between two groups. Similarly, George et al. (2010), demonstrated that a 100 or 120 mcg bolus was usually successful in reversing a 20% drop in systolic blood pressure (SBP) or $SBP < 90$ mmHg within 1 minute. No hypertensive episodes were observed in their study population although bradycardia was noted at 140 mcg.

References:

1. Yoshioka M, Yamada T, Miyake W, Tanaka K, Nishikawa K 2011, "The effectiveness of

pleth variability index to predict hypotension induced by spinal anesthesia for cesarean delivery", Annual Meeting of the American Society of Anesthesiologists. Chicago, A960.

2. Partridge B, 1987, "Use of pulse oximetry as a noninvasive indicator of intravascular volume status", *Journal of Clinical Monitoring*, vol-3 page-263-8.
3. Shamir M, Eidelman LA, Floman Y, Kaplan L, Pizov R, 1999, "Pulse oximetry plethysmographic waveform during changes in blood volume", *Br J Anaesth*, vol-82 page-178-81.
4. Natalini G, Rosano A, Bernardini A, Taranto M, Faggian B, Vittorielli E, 2006, "Arterial versus plethysmographic dynamic indices to test responsiveness for testing fluid administration in hypotensive patients", a clinical trial *anaesth analg*, vol- 103 pages-1478-84.
5. Wyffels PA, Durnez PJ, Helderweirt J, Stockman WM, De Kegel D, 2007, "Ventilation-induced plethysmographic variations predict fluid responsiveness in ventilated postoperative cardiac surgery patients", *Anesth & Analgesia*, vol-105 pages-448-52.
6. Cannesson M, Desebbe O, Rosamel P, et al, 2008, "Pleth variability index to monitor the respiratory variations in the pulse oximeter plethysmographic waveform amplitude and predict fluid responsiveness in the operating theatre" *Br J Anaesth*, vol-101 pages-200-6.
7. Tsuchiya M., Yamada T, Asada A, 2010, "Pleth Variability Index Predicts Hypotension During Anesthesia Induction", *Acta Anaesthesiologica Scandinavica*, vol. 54, pages 596-602.
8. Yoshioka M, Yamada T, Miyake W, Tanaka K, Nishikawa K, 2011, "The effectiveness of pleth variability index to predict hypotension induced by spinal anesthesia for cesarean delivery", *American Society of Anesthesiologists*, pages-960.

Controlled Hypotension for Functional Endoscopic Sinus Surgery: A Comparative study between Dexmedetomidine versus Esmolol

Md. Shafiul Alam Shaheen¹, Kawsar Sardar², A.K.M.Nurnobi Chowdhury³, Subrata Kumar Mondal⁴, Raju Ahmed⁵, S.M. Shafiqul Alam⁶

¹Assistant Professor, ^{2,3} Professor, Department of Anesthesia & Surgical ICU, Ibrahim Medical College & BIRDEM General Hospital ⁴Associate Professor, Department of Anesthesia, Analgesia, Palliative and Intensive Care Medicine, Dhaka Medical College Hospital ⁵Assistant Professor, Department of Anaesthesia & Surgical ICU, Ibrahim Medical College & BIRDEM General Hospital, ⁶Senior Consultant, Department of Anaesthesia, Analgesia, Palliative & Intensive Care Medicine, Dhaka Medical College Hospital

Corresponding Author: E-mail: drshafiul27@yahoo.com, Mobile: +88 01912078807

Abstract

Background: Functional endoscopic sinus surgery (FESS) requires effective control of bleeding for better visibility of the operating field and reduced risk of injury to the optic nerve or the internal carotid artery. Controlled hypotension is a technique used to limit intraoperative blood loss to provide the best possible field for surgery.

Objectives: Our study is undertaken to evaluate the efficacy of dexmedetomidine as a hypotensive agent in comparison to esmolol in Functional Endoscopic Sinus Surgery (FESS).

Method: Sixty (60) patients 20 – 50 years of age, ASA I/II scheduled for FESS were equally randomly assigned to two equal groups of 30 patients each. Patients of group D received dexmedetomidine 1µg/kg over 10 min before induction of anesthesia followed by 0.4 – 0.8 µg/kg/hr infusion during maintenance and group E received esmolol loading dose 1mg/kg was infused over one min followed by 0.4 – 0.8 mg/kg/hr infusion during maintenance to maintain mean arterial blood pressure (MAP) between (55 – 65 mmHg). The surgical field was assessed using Average Category Scale and average blood loss was calculated. Hemodynamic variables (MAP, HR); intraoperative fentanyl consumption and total recovery from anesthesia (Aldrete's score ≥ 9) were recorded. Sedation score was determined at 10, 20, 30, 40 & 60 min after tracheal extubation and time to first analgesic demand was also recorded.

Results: In both group D and group E reached the desired MAP (55 – 65 mmHg) with no intergroup difference in MAP or HR. Mean intraoperative fentanyl consumption was significantly lower in group D than group E. Recovery time to achieved Aldrete's score ≥ 9 were significantly lower in group E compared with group D. The sedation score were significantly lower in group E compared with group D at 10 minutes, 20 minutes and 30 minutes postoperatively. Time to first analgesic demand was significantly longer in group D.

Conclusion: The result of this study showed that both dexmedetomidine and esmolol can be used as agents for controlled hypotension and are effective in providing ideal surgical field during FESS. But dexmedetomidine offers the advantage of inherent analgesic, sedative and anesthetic sparing effect.

Keywords: Controlled hypotension, dexmedetomidine, esmolol, functional endoscopic sinus surgery (FESS).

(JBSA 2018; 31(2): 67-74)

Introduction:

Functional endoscopic sinus surgery (FESS) is the

treatment of choice for acute and chronic sinus pathologies and nasal polyp. This surgical intervention restores the drainage pathways and aeration of the paranasal sinus. There are many

benefits of a well-performed endoscopic sinus surgery with appropriate indications, but major complications of orbital hematoma, injury to the optic nerve, cerebrospinal fluid fistula, and intracranial injuries could occur as bleeding reduces the visibility of the operative field. To minimize these complications, effective control of bleeding at the surgical site is required.

Various techniques to minimize bleeding during sinus surgery are head elevation of 30° (reverse Trendelenburg), infiltration or topical application of epinephrine, and electively controlled hypotension. Controlled hypotension is applied widely in several surgical interventions using different techniques.¹ Benefits for controlled hypotension for FESS include reduction in blood loss with improved quality of surgical field. Various agents e.g. magnesium sulfate², Vasodilators (sodium nitroprusside)³, nitroglycerine⁴, high dose of potent inhaled anesthetics⁵, and beta adrenergic antagonist⁶ have been used to achieve controlled hypotension. Although these pharmacological agents effectively lower the blood pressure, they are associated with delayed recovery from inhaled anesthetics, resistance to vasodilator or tachyphylaxis, and cyanide toxicity from nitroprusside. Esmolol and nitroglycerine precisely control the blood pressure because of their rapid onset and short duration of action, but unambiguous hemodynamic monitoring is required. An infusion of 10 – 20 mg/kg/hr remifentanyl is also useful, but is associated with the side effect of hyperalgesia.^{7, 8} Therefore; the choice of an ideal agent is still controversial.

Esmolol is an ultrashort acting selective α_1 adrenergic antagonist that reduces heart rate and blood pressure. It has rapid onset of action of bolus IV injection and infusion. Upon termination of infusion gradual recovery of arterial blood pressure to the pre-infusion level occurred without development of rebound hypertension.

Dexmedetomidine is a potent highly selective α_2 agonist, is used as an adjuvant to general anesthesia for sedation, analgesia, and hemodynamic stability with no postoperative respiratory depression. It is valuable because of its analgesic and anesthetic-sparing effects.⁹

Our study was designed to compare the efficacy and safety of dexmedetomidine or esmolol as a hypotensive agent in FESS with attention on the

amount of blood loss, quality of the surgical field, recovery profile, and tolerability in adult patients.

Methods:

This prospective randomized single-blind study was conducted from January 2016 to December 2016 at the department of Anesthesia & Surgical ICU of BIRDEM General Hospital, Shahbagh, Dhaka, Bangladesh. After approval from hospital ethics committee and getting informed written consent to participate in the study, 60 patients aged 20-50 years, ASA physical status I & II scheduled for elective FESS were recruited. Patients with recurrent sinus surgery, hypertension, coronary artery diseases and renal, hepatic or cerebral insufficiency and patients with coagulopathies or receiving drugs influencing blood coagulation were excluded from the study. All patients had bilateral nasal polyposis with opacity of all paranasal sinuses and they were assessed clinically in addition to ECG, chest X-ray and basal laboratory tests. The patients were divided into two groups randomly by envelop method where Group D received dexmedetomidine and group E received esmolol.

In the operating room, two cannulae were inserted, one for infusion of dexmedetomidine or esmolol and the other for administration of fluids and other drugs. In group D, patients received loading dose of 1 µg/kg dexmedetomidine diluted in 10ml 0.9% normal saline infused over 10 minutes before induction of anaesthesia, followed by continuous infusion of 0.4 – 0.8 µg/kg/hr. in group E, patients received esmolol as a loading dose 1mg/kg was infused over one minute followed by continuous infusion of 0.4 – 0.8 mg/kg/hr. in both groups infusion rate was titrated to maintain MAP within 55 – 65 mmHg. All patients were received general anaesthesia with induction dose of inj. Fentanyl 2 microgram/kg, inj. Propofol 1 – 2 mg/kg until loss of verbal response and muscle relaxant inj. Atracurium 0.5mg/kg. The required induction doses of Propofol were recorded. After induction, general anaesthesia maintained by 60% N₂O and 40% O₂ and continuous infusion of Propofol @ 5mg/kg/hr. Incremental muscle relaxant was given every 20 minutes interval 1/4th of the initial dose. In both groups, signs of inadequate anesthesia as increase in the blood pressure, heart rate or somatic responses as movement, tearing, or

sweating were treated with additional dose of fentanyl. Respiratory rate and tidal volume were adjusted according to body weight to maintain normocapnia. Nitroglycerine was infused if these target limits could not be achieved with upper most doses. The drug infusion dose was decreased when targeted MAP was achieved. Patients were placed head elevation of 30^0 (reverse Trendelenburg) to improve venous drainage. In both groups cottonoids soaked with epinephrine in a concentration of 1: 80,000 was inserted into the nasal cavity to minimize the blood loss. Oropharyngeal pack was used in all patients.

During the procedure, the quality of the surgical field was assessed by the surgeon every 10 minute interval. The same surgeon performed all operations to ensure consistency in the estimation of the surgical field. The surgeon was blinded to the hypotensive agent used. When MAP reached the desired range 55 – 65 mmHg and was maintained for at least 10 minutes, the surgeon estimated the quality of the surgical field using a predefined category scale adopted from that of Fromme et al.¹⁰.

Average category scale for assessment of intraoperative surgical field:

0	–	No bleeding
1	–	Slight bleeding: no suctioning of blood required
2	–	Slight bleeding: occasional suctioning required. Surgical field not threatened
3	–	Slight bleeding: frequent suctioning required. Bleeding threatens surgical field a few seconds after suction is removed
4	–	Moderate bleeding: frequent suctioning required. Bleeding threatens surgical field directly after suction is removed
5	–	Severe bleeding: constant suctioning required. Bleeding appears faster than can be removed by suction. Surgical field severely threatened and surgery not possible.

The ideal category scale values for surgical conditions were predetermined to be two and three. The total blood loss was measured from the suction apparatus. Infusion of the study drugs was

stopped five minutes before the anticipated end of the surgery, and Propofol was stopped at the end of the surgery and residual neuromuscular blocked was antagonized with neostigmine (0.05mg/kg) and atropine(0.01mg/kg).

Monitoring included the heart rate, non-invasive blood pressure, continues ECG monitoring, ETCO₂ concentration, SPO₂ were recorded preoperatively (base line), post induction (after administration of hypotensive and anaesthetic agent), Intraoperatively (10, 20, 30, 40 & 60 minutes), 5 minutes and 10 minutes after stoppage of hypotensive agents and lastly after recovery. Intraoperative fentanyl consumption and requirements for additional hypotensive agent (nitroglycerine) were recorded. After extubation and full recovery, patients were transferred to the postoperative ward to be observed where time to first analgesic demand was recorded. Post operative recovery was evaluated using a modified Aldrete's Score (0 – 10)¹¹, and time needed to achieve e"9 was recorded. Sedation score¹² was measured using the following scale at 10, 20, 30, 40 and 60 minutes after tracheal extubation. Sedation score: 1 – anxious, agitated, or restless; 2 – cooperative, oriented, and tranquil; 3 – responsive to commands; 4 – asleep, but with brisk response to light, glabellar tap, or loud auditory stimulus; 5 – asleep, sluggish response to glabellar tap, or auditory stimulus; and 6 – asleep, no response. Patients were also asked about recalling intraoperative events or any sign of awareness.

Data processing and analysis:

Statistical analysis was done using software SPSS (Statistical Package for Social Science), version 15. Demographic & haemodynamic data were analysed using unpaired student t-test or chi-square(χ^2). Statistically significance was set at p-value < 0.05.

Results:

Sixty patients who underwent functional endoscopic sinus surgery (FESS) were enrolled in the study. Demographic data of age, sex, weight, ASA physical status and duration of surgery were comparable between the groups (Table – I). The induction dose of Propofol was significantly lower in group D (1.42 ± 0.38 mg/kg) than group E (2.38 ± 0.42 mg/kg) ($P < 0.001$).

Table I Demographic variables

Variable	Group-D Dexmedetomidine (n=30)	Group-E Esmolol (n=30)	P value
Age (years)	34.40±11.12	36.20±12.55	0.56 ^{ns}
Sex (male/Female)	11/19	12/18	0.78 ^{ns}
Weight (kgs)	53.67±8.13	52.30±9.44	0.55 ^{ns}
ASA(I/II)	21/9	20/10	0.78 ^{ns}
Duration of surgery (min)	90.03±9.44	91.07±8.13	0.63 ^{ns}
Estimated blood loss (ml)	121.0±6.95	123.03±7.85	0.74 ^{ns}

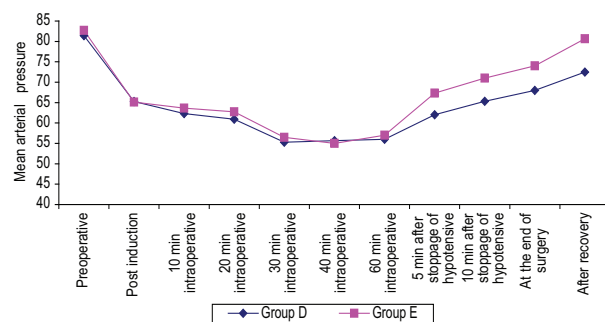
All values were presented as mean±SD or in frequencies; ASA, American society of Anesthesiologists; Data were analysed using unpaired student t-test. Statistically significance was set at p-value < 0.05. (NS=not significant).

The baseline values of MAP and HR were comparable in both groups. In group D and group E, there was a significant reduction of MAP in both groups compared to baseline value intraoperatively. In both groups the desired MAP (55 – 65 mmHg) was achieved with no significant differences after induction or during hypotensive period. There was no need to use additional hypotensive agent nitroglycerine in both groups. At 5 minutes and 10 minutes after stoppage of hypotensive agents, at the end of surgery and after recovery, MAP was significantly lower in group D than group E (Figure 1). Heart rate decreased significantly relative to baseline after administration of loading dose in both groups. There were no significant differences in HR in between the groups after induction or during the hypotensive period. Heart rate showed significant increased in group E at 5 & 10 minutes after stoppage of hypotensive agent,

at the end of surgery and after recovery compared to group D (Figure 2).

Mean intraoperative fentanyl consumption in group D (30.0±2µg) was significantly less than group E (65.0±3.5µg).

The average category scale(ACS) for quality of surgical field was comparable in both groups in the range of MAP(55 – 65 mmHg).scores for a bloodless surgical fields were low in both groups; there was no significant difference in between the groups. The median range of score was 2 (1 – 3) in both groups. The ACS score were ≤2 throughout the hypotensive period (Table II). There was no significant difference in between the groups regarding the amount of blood loss intraoperatively. The time needed to achieve ≥9 of modified Aldrete's score were significantly longer in group

**Fig 1** Line diagram showing intraoperative mean arterial blood pressure (MAP) in two groups**Table II** Average category scale (0 -5) during hypotensive anesthesia periods

Time during hypotensive Period	Group-D Dexmedetomidine (n=30)	Group-E Esmolol (n=30)
10 min	2(1-3)	2(2-3)
20 min	2(2-3)	2(1-2)
30 min	2(1-2)	2(1-3)
40 min	2(1-3)	2(1-2)
60 min	2(1-2)	2 (1-2)

All values were presented as mean±SD or in frequencies; Data were analysed using unpaired student t-test. Statistically significance was set at p-value < 0.05. (NS=not significant, S= significant).

Table III Recovery characteristics, sedation scores and first analgesic demand

	Group-D Dexmedetomidine (n=30)	Group-E Esmolol (n=30)	P value
Aldrete's score e"9 (min)	10.4±2.5	8.5±2.3	Â0.01 ^S
Sedation score 10 min after surgery	4.0±0.6	2.5±0.4	Â0.01 ^S
Sedation score 20 min after surgery	3.8±0.4	2.3±0.2	Â0.01 ^S
Sedation score 30 min after surgery	3.6±0.5	2.1±0.3	Â0.01 ^S
Sedation score 40 min after surgery	2.7±0.6	2.1±0.5	0.34 ^{NS}
Sedation score 60 min after surgery	2.5±0.4	2.0±0.5	0.22 ^{NS}
1 st analgesic demand (min)	58.65±8.22	31.25±5.15	<0.01 ^S

All values were presented as mean±SD or in frequencies; Data were analysed using unpaired student t-test. Statistically significance was set at p-value <0.05. (NS=not significant, S= significant).

D (10.4±2.6 minutes) than group E (8.5±2.4 minutes) (P<0.01).

The mean postoperative sedation scores were significantly lower in group E than group D at 10 min, 20min and 30 min. No significant difference was observed in sedation score at 40 min and 60 min in both groups (Table III). No patients complain any sign of awareness in both groups. First analgesic requirement was recorded and there was significantly earlier in analgesic requirement in group E than group D (Table 3). There was no postoperative nausea or vomiting observed in both groups.

The mean arterial blood pressure at different time in between two groups which showed statistical significant at 5 minutes and 10 minutes after stoppage of hypotensive agents, at the end of

surgery and after recovery and MAP was significantly lower in group D than group E (p < 0.05)

The mean heart rate at different time in between two groups which showed significant increase in group E at 5 & 10 minutes after stoppage of hypotensive agent, at the end of surgery and after recovery compared to group D (p < 0.05)

Discussion

The development of a nasal endoscope has facilitated the surgical treatment (FESS) of acute and chronic sinus pathologies when conservative treatment fails. The procedure perpetuates the mucociliary clearance mechanism and conserves the normal nonobstructing anatomic structures. However, major or minor complications could occur as bleeding reduces the visibility of the operative field and hampers the surgical intervention.

There are a lot of efforts have been done to optimize the surgical conditions for FESS. Induced hypotension has been widely used to control bleeding during FESS to improve the quality of surgical field.^{13, 14} Our study of dexmedetomidine or esmolol we planned to provide this optimal surgical field. Both drugs were effective providing MAP of 55 – 65mmHg, and lowering the heart rate ensured good surgical condition and providing dry surgical field during FESS.

The patients who were treated with

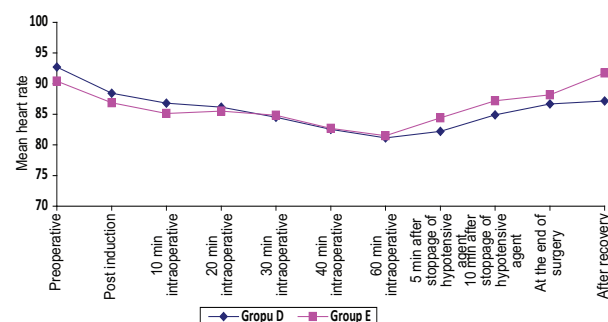


Fig 2 Line diagram showing intraoperative heart rate in two groups

dexmedetomidine 10 minute before induction of anesthesia had significant decrease in MAP and HR after administration of loading dose. Dexmedetomidine is a potent highly selective α_2 -adrenergic receptor agonist. It has sedative, analgesic and anesthetic sparing effect, and sympatholytic properties.¹⁵ The use of β_2 -adrenergic agonist cause decrease in sympathetic tone that causes decrease in heart rate, blood pressure and hemodynamic response to surgery.¹⁶ The analgesic and hypnotic effects of dexmedetomidine and other β_2 -agonists is due to its action at locus coeruleus in the upper brain stem.¹⁷ The β_2 – receptors are also involved in regulating the autonomic and cardiovascular systems. These α_2 receptors are located on blood vessels, where they mediate vasoconstriction, and on sympathetic terminal, where they inhibit, nor-epinephrine release.¹⁸

Basar et al.¹⁹ provided the effect of single dose of dexmedetomidine 0.5 μ g/kg administration 10 minute before induction of anesthesia and reported significant reduction in MAP and HR. The efficacy of dexmedetomidine in providing better surgical and less blood loss during controlled hypotension was previously reported during tympanoplasty, septoplasty and maxillofacial surgery.^{20, 21} In the present study, the induction dose of propofol was significantly lower in group D than in group E. This effect coinciding with the result of Peden et al.²², who reported that dexmedetomidine caused a reduction in the overall dose of Propofol required to produce loss of consciousness. Guven et al.²³ and Goksu et al.²⁴ found that better hemodynamic stability, visual analogue scale for pain and clear surgical field with less side effects in dexmedetomidine group than placebo group when FESS done under either conscious sedation or local anesthesia respectively.

Esmolol lowers arterial blood pressure through a decrease in cardiac output secondary to negative chronotropic and inotropic effects of α -adrenergic antagonism. It provides a stable course of controlled hypotension and produces beneficial effects in the surgical fields and in blood conservation.^{25, 26} Esmolol has been used effectively to provide controlled hypotension intraoperatively in many studies.²⁷ Lim et al.²⁸

used esmolol for controlled hypotension in patients undergoing spinal surgery. They reported that esmolol was an appropriate agent for controlled hypotension in acute normovolemic hemodilution from the prevention of blood loss in patients except those who do not have cardiovascular problems. Esmolol provided a stable course of controlled hypotension and produces beneficial effects in the surgical field and in blood conservation. The optimal anesthetic technique seems to be relative bradycardia with associated hypotension.²⁹

In our study intraoperative fentanyl consumption was significantly less in group D compared with group E. Several studies have found that perioperative use of dexmedetomidine was associated with a significant decrease in the consumption of inhalational agent, fentanyl, and analgesic in dose dependent manner.^{30, 31}

Our study also showed that postoperative analgesia requirement was prolonged in group D than group E. This is accordance with Gurbet et al.³² who stated that intraoperative infusion of dexmedetomidine reduces perioperative analgesic requirements. The analgesic effects of dexmedetomidine had been appreciated in various setting and various populations.^{33, 34} Dexmedetomidine was associated with significant longer recovery time from anesthesia.

Conclusion

In conclusion, our study was the first study conducted in Bangladesh population and this study demonstrated that dexmedetomidine or esmolol both were safe agents for controlled hypotension and both were effective in providing ideal surgical field during FESS. But compared with esmolol dexmedetomidine offers the advantage of inherent analgesic, sedative and anesthetic sparing effect.

References

1. Shams T, EI Bahnasawe NS, Abu-Samra M, EI-Masry R. Induced hypotension for functional endoscopic sinus surgery: a comparative study of dexmedetomidine versus esmolol. Saudi J Anaesth 2013 Apr; 7(2): 175-180.
2. Elsharnouby NM, El Sharnouby MM. Magnesium sulphate as a technique of hypotensive anesthesia. Br J Anaesth 2006;

- 96: 727-31.
3. Degoute CS, Ray MJ, Manchon M, Dubreuil C, Banssillon V. Remifentanyl and controlled hypotension; comparison with nitropruside or esmolol during tympanoplasty. *Can J Anaesth* 2001; 48: 20 – 7
 4. Degoute CS, Dubreuil C, Ray MJ, Guitton J, Manchon M, Banssillon V. Effect of posture, hypotension and locally applied vasoconstriction on the middle ear microcirculation in anaesthetized humans. *Eur J Appl Physiol Occup Physiol* 1994; 69: 414 – 20
 5. Pavlin JD, Colley PS, Weymuller EA Jr, Van Norman G, Gunn HC. Propofol versus isoflurane for endoscopic sinus surgery. *Am J Otolaryngol* 1999; 20: 96 – 101
 6. Degoute CS. Controlled hypotension: Guide to drug choice. *Drugs* 2007; 67: 1053 – 76
 7. Abdullah AO, Yaman O, Ayten S, Hakan E, Hiisnti S, Gülten A, et al. Dexmedetomidine versus remifentanyl for controlled hypotensive anesthesia in functional endoscopic sinus surgery. *Turk J Anaesth Reanim* 2012; 40: 257 – 261
 8. Lee J, Kim Y, Park C, Jeon Y, Kim D, Joo J, Kang H. Comparison between dexmedetomidine and remifentanyl for controlled hypotension and recovery in endoscopic sinus surgery. *Ann Otol Rhinol Laryngol* 2013; 122: 421 – 426
 9. Sudhesh K Hassoor SS. Dexmedetomidine in anesthesia practice: a wonder drug? *Indian J Anaesth* 2011; 55: 323 - 324
 10. Fromme GA, MacKenzie RA, Gould AB Jr, Lund BA, Offord KP. Controlled hypotension for orthognatic surgery. *Anesth Analg* 1986; 65: 683 - 6
 11. Alderete JA. The post-anesthesia recovery score revisited. *J Clin Anesth* 1995; 7: 89
 12. Ramsay M, Savege T, Simpson BR, Good R. Controlled sedation with alphaxolone-alphadolone. *Br Med J* 1974; 2: 656 - 9
 13. Eberhart LH, Folz BJ, Wulf H, Geldner G. Intravenous anesthesia provides optimal surgical conditions during microscopic and endoscopic sinus surgery. *Laryngoscope* 2003; 113: 1369 – 73.
 14. Cincikas D, Ivaskevicius S. Application of controlled arterial hypotension in endoscopic rhino-surgery. *Medicina (Kaunas)* 2003; 39: 852 – 9
 15. Bloor BC, Ward DS, Belleville JP, Maze M. Effect of intravenous dexmedetomidine in humans. Hemodynamic changes. *Anesthesiology*. 1992; 77: 1134 – 42
 16. Muzi M, Goff DR, Kampine JP, Roerig DL, Ebert TJ. Clonidine reduces sympathetic activity but maintains baroreflex responses in normotensive humans. *Anesthesiology*. 1992; 77: 864 – 71
 17. Mariappan R, Narayana Prabhu AH, Kuppuswamy B. Comparing the effects of oral clonidine premedication with intraoperative dexmedetomidine infusion on anesthetic requirement and recovery from anesthesia in patients undergoing major spine surgery. *J Neurosurg Anesthesiol*. 2013 Jul 24
 18. Langer SZ. Presynaptic regulation of the release of catecholamines. *Pharmacol Rev* 1980; 32: 337 – 62
 19. Basar H, Akpınar S, Doganci N, Buyukkocak U, Kaymak C, Sert O,. The effect of preanaesthetic single dose of dexmedetomidine on induction, hemodynamic and cardiovascular parameters. *J Clin Anesth* 2008; 20: 431 - 6
 20. Durmus M, But AK, Dogan Z, Yucel A, Miman MC, Ersoy MO. Effect of dexmedetomidine on bleeding during tympanoplasty or septoplasty. *Eur J Anaesthesiol* 2007; 24: 447 – 53
 21. Richa F, Yazigi A, Hage C. Dexmedetomidine an agent for controlled hypotension in maxilla fascial surgery. *Eur J Anaesthesiol* 2004; 21: A-242
 22. Peden CJ, Cloote AH, Stratford N, Prys-Roberts C. The effect of intravenous dexmedetomidine premedication on the dose of requirements of propofol to induce loss of consciousness in patients receiving alfentanil.

- Anaesthesia 2001; 56: 408 – 13
23. Guven DG, Demiraran Y, Sezen G, Kepek O, Iskender A. Evaluation of outcomes in patients given dexmedetomidine in functional endoscopic sinus surgery. *Ann Otol Rhinol Laryngol* 2011; 120: 586 – 92
 24. Goksu S, Arik H, Demiryurek S, Mumbuc S, Oner U, Demiryurek AT. Effects of dexmedetomidine infusion in patients undergoing functional endoscopic sinus surgery under local anaesthesia. *Eur J Anaesthesiol* 2008; 25: 22 – 8
 25. Degoute CS, Ray MJ, Manchon M, Dubreuil C, Banssillon V. Remifentanyl and controlled hypotension; comparison with nitropruside or esmolol during tympanoplasty. *Can J Anesth.* 2001; 48: 20 – 7
 26. Boezaart AP, Merwe JV, Coetzee A. Comparison of sodium nitropruside and esmolol-induced controlled hypotension for functional endoscopic sinus surgery. *Can J Anesth.* 1995; 42: 373 – 6
 27. Ibraheim OA, Abdulmonem A, Baaj J, Zahrani TA, Arlet V. Esmolol versus dexmedetomidine in scoliosis surgery: study on intraoperative blood loss and hemodynamic changes. *Middle East J Anesthesiol.* 2013 Feb; 22(1): 27 - 33
 28. Lim YZ, Kim CS, Bahk JH, Ham BM, Do SH. Clinical trial of esmolol induced controlled hypotension with or without acute normovolemic hemodilution in spinal surgery. *Acta Anesthesiol Scand.* 2003; 47: 74 - 8
 29. Timperley D, Sacks R, Parkinson RJ, Harvey RJ. Perioperative and intraoperative maneuvers to optimize surgical outcomes in skull base surgery. *Otolaryngol Clin North Am* 2010; 43: 699 – 730.
 30. Aantaa R, Jaakola ML, Kallio A, Kanto J. Reduction of the minimum alveolar concentration of isoflurane by dexmedetomidine. *Anesthesiology* 1997; 86: 1055 – 60
 31. Scheinin H, Jaakola ML, Sjoval S, Ali-Melkkila T, Kaukinen S, Turunen J. Intramuscular dexmedetomidine as premedication for general anaesthesia. A comparative multicenter study. *Anesthesiology* 1993; 78: 1065 - 75
 32. Gurbet A, Basagan-Mogol E, Turker G, Ugun F, Kaya FN, Ozcan B. Intraoperative infusion of dexmedetomidine reduces perioperative analgesic requirements. *Can J Anesth* 2006; 53: 646 – 52
 33. Huncke TK, Adelman M, Jacobowitz, G, Maldonado T, Bekker A. A prospective randomized, placebo- controlled study evaluating the efficacy of dexmedetomidine for sedation during vascular procedures. *Vasc Endovasc Surg* 2010; 44: 257 – 61
 34. Karaaslan K, Yilmaz F, Gulcu N, Colak C, Sereflican M, Kocoglu H. Comparison of dexmedetomidine and midazolam for monitored anaesthesia care combined with tramadol via patient – controlled analgesia in endoscopic nasal surgery: A prospective randomized, double-blind, clinical study. *Curr Ther Res Clin Exp* 2007; 68: 69 - 81

Procedural Sedation & Analgesia by Ketamine-Propofol (Ketofol) And Ketamine-Diazepam Combination In Day Case Surgery - A Comparative Study

Farzana Kalam¹, Pervez Altaf Hussain², Masud Ahmed³, Abdullah Masum⁴

^{1,3}Classified Specialist in Anaesthesiology, Department of Anaesthesia, CMH, Dhaka, ^{2,4}Classified Specialist in Anaesthesiology, Department of Anaesthesia, CMH, Comilla

Corresponding Author: Lt. Col. Farzana Kalam, Classified Specialist in Anaesthesiology, Department of Anaesthesia, CMH, Dhaka

Abstract

Introduction: Traditionally Procedural Sedation and Analgesia (PSA) is provided by various drugs and its combinations with mixed effect regarding safety and efficacy.

Objective: The aim of the study was to compare the combination of Ketamine & Propofol (Ketofol) and Ketamine-Diazepam for PSA in day case surgery.

Materials & Methods: This prospective study was carried out in CMH Dhaka on sixty patients equally divided in two groups between the period of May 2015 to September 2015. Group I patients received Injection Ketamine 1mg/kg & Injection Propofol 1mg/kg combination while group II patients received Injection Ketamine 2mg/kg & Injection Diazepam 0.2mg/kg combination. Clinical parameters like pulse rate, non invasive blood pressure (NIBP), percentage saturation of oxygen (SpO₂) were monitored, recorded and analyzed. Recovery was assessed by Aldred Recovery Score (ARS) and compared between groups.

Results: Rise of heart rate, systolic blood pressure (SBP), diastolic blood pressure (DBP) were statistically significant in group II patients when compared to group I both during the procedure & recovery but rise of mean arterial pressure (MAP) was significant only during procedure. The recovery time in group II patients (24.7 ± 3.6 min) was significantly higher (29.7 ± 4.1 min) than group I patients. Side effects like excessive bronchial secretion and postoperative agitation were also significantly lesser in Ketofol group.

Conclusion: Ketofol provides safer and more effective sedation and analgesia than Ketamine and Diazepam combination.

Keywords: Ketofol, Procedural sedation and analgesia, Day case surgery.

(JBSA 2018; 31(2): 75-82)

Introduction

Procedural sedation is a technique of administering sedatives or dissociative agents with or without analgesics to induce a state that allows the patient to tolerate unpleasant procedures while maintaining cardiorespiratory function. Procedural Sedation and Analgesia (PSA) is intended to result in a depressed level of consciousness that allows the patient to maintain oxygenation and airway control independently^{1,2}. Various drugs are available and used to provide procedural sedation like Benzodiazepines, Opioids, Ketamine, Propofol, Etomidate etc. either alone or in combinations. Minimal stress, maximum comfort, early recovery & early ambulation are the principle of selecting drugs for

PSA or day case surgery. But every drug or combination of drugs has got its own merits and demerits. The objective of the study is to compare the effectiveness and safety of traditional combination of Ketamine and Propofol (Ketofol) for PSA in day case surgery.

Materials and Methods

It was a prospective, double blinded, comparative study carried in Combined Military Hospital (CMH) Dhaka, between the period of May 2015 to September 2015 after getting approval from hospital ethical committee.

Sixty patients scheduled for elective day case procedure were randomly selected for the study. Adult patients of age group 18-45 years from both

sexes of ASA status I & II, undergoing procedures of less than one hour duration were induced in the study. Patients with history of cerebrovascular disease (CVD), ischemic heart disease (IHD), raised intra cranial pressure (ICP), hypertension, seizure disorder, pregnancy or known hypersensitivity reaction to Ketamine, Propofol or Diazepam were excluded from the study. After taking informed written consent from all patients they were divided into two equal groups of thirty by lottery method. All patients were pre medicated with injection Ondansetron 8 mg IV. Group I patients received injection Ketamine 1 mg/kg & injection Propofol 1mg/kg mixed in a single syringe and diluted with same amount with distilled water as a bolus whereas group II patients received injection Ketamine 2mg/kg & injection Diazepam 0.2 mg/kg combined as a bolus and diluted with distilled water and intra lipid (for double blinding) making 10 ml solution. All patients were oxygenated in requirement basis at a rate of 4 L/minute by face mask. Heart rate, noninvasive blood pressure (NIBP), respiratory rate,

Electrocardiogram (ECG), percentage saturation of oxygen (SpO_2) were monitored during procedure & during recovery at every 5 minutes interval. Ramsay Sedation Scale (RSS)⁽³⁾ was monitored throughout the procedure by incremental doses of (25% of initial dose) as per requirement. Quality of analgesia was assessed by haemodynamics, sweating and respiratory rate. Aldrede recovery score⁽⁴⁾ (≥ 8) was used to ascertain recovery. Peroperative & postoperative (up to 6 hours) all complications were also recorded. All data were compiled in a preformed data sheet and analyzed by students't test, two proportion test & chi square test. P value < 0.05 was considered significant.

Results

There was no significant difference regarding patient's characteristics like age, sex, weight and ASA grading (Table I).

There were predominance of orthopaedic and gynaecological procedures in both groups such as, close reduction of fractured bones or dislocated joints, dilatation & curettage, incision & drainage

Table I Patients' characteristics

Characteristics	Group I (n=30) Mean $\pm$ SD	Group II (n=30) Mean $\pm$ SD	p value	Result
Age (years)	34.73 $\pm$ 9.04	32 $\pm$ 7.1	0.2	NS (Student 'T' test, unpaired)
Weight (Kg)	60.8 $\pm$ 9.75	58.26 $\pm$ 7.82	0.4	NS (Student 'T' test, unpaired)
Sex- Male :Female	17:13	16:14	0.8	NS(Chi square test)
ASA Grading I/ II	20(66.66%)/ 10(33.33%)	22(73.33%)/ 8(26.66%)	0.7 0.6	NS(Chi square test) NS(Chi square test)

Table II Type of procedure

Surgical Procedure	Group I		Group II	
	Number	%	Number	%
Amputation of little finger	1	3.3%	1	3.3%
Cervical polypectomy	1	3.3%	1	3.3%
Close reduction of joints	7	23.3%	6	20.0%
Dilatation & Curettage (D&C)	6	20.0%	5	16.7%
Incision and Drainage of Abscess	7	23.3%	4	13.3%
Repair of tendon	2	6.7%	3	10.0%
Surgical toileting & repair	2	6.7%	2	6.7%
Wound debridement and dressing	2	6.7%	2	6.7%
Excision Biopsy	2	6.7%	6	20.0%
Total	30		30	

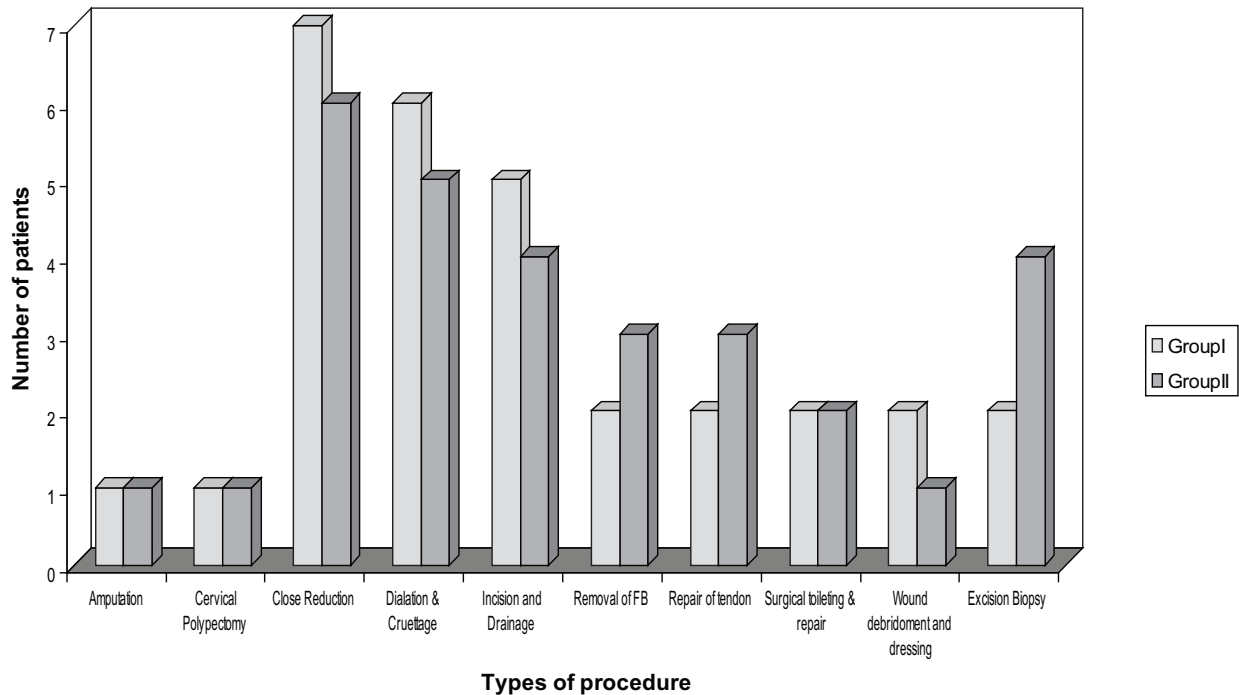


Figure I Types of procedure undertaken

Changes in heart rate are shown in table III. The mean of the value of during procedure and during recovery was first calculated and considered as mean $\pm$ SD of the values. As expected heart rate increased in patients of both groups during the procedure, during recovery but when compared between two groups it was significantly higher in group II patients as shown in table-III.

Table-III Heart rate in two groups

Time	Group I (n=30)	Group II (n=30)	p value	Result
Pre induction	77.9 $\pm$ 9.84	78.1 $\pm$ 11.43	0.86	NS (Student's 'T' test, unpaired)
After induction	82.35 $\pm$ 3.65	88.45 $\pm$ 4.84	0.90	NS (Student's 'T' test, unpaired)
5 minutes	84.2 $\pm$ 3.75	110 $\pm$ 10.4	0.04	S (Student's 'T' test, unpaired)
10 minutes	89.80 $\pm$ 5.05	116.60 $\pm$ 5.90	0.03	S (Student's 'T' test, unpaired)
15 minutes	89.9 $\pm$ 9.97	117.6 $\pm$ 7.05	0.03	S (Student's 'T' test, unpaired)
20 minutes	87.88 $\pm$ 6.70	112.15 $\pm$ 5.65	0.04	S (Student's 'T' test, unpaired)
25 minutes	84.44 $\pm$ 6.4	100.94 $\pm$ 6.55	0.05	S (Student's 'T' test, unpaired)
30 minutes	77.1 $\pm$ 3.94	88.63 $\pm$ 9.62	0.08	NS (Student's 'T' test, unpaired)

Values are presented as mean $\pm$ SD. Analysis was done by Student's 't' test. NS: Not significance $p > 0.05$ (among two groups). Sig: Significant $p < 0.05$ (among two groups).

Although the base line Blood Pressure (systolic, diastolic & mean) were quite similar in two groups but they were raised during the procedure and remained higher during recovery. In group II patients the rise of systolic & diastolic blood pressure were significantly higher during the procedure and during recovery ($P < 0.05$), when compared to group I patients (Table IV, V).

Table IV *Systolic blood pressure in two groups*

Time	Group I (n=30)	Group II (n=30)	p value	Result
Pre induction	117.9 ± 9.84	118.1 ± 11.43	0.86	NS (Student's 'T' test, unpaired)
After induction	118.35 ± 3.65	126.45 ± 4.84	0.20	NS (Student's 'T' test, unpaired)
5 minutes	117.2 ± 3.75	138 ± 10.4	0.04	S (Student's 'T' test, unpaired)
10 minutes	119.80 ± 5.05	146.60 ± 5.90	0.03	S (Student's 'T' test, unpaired)
15 minutes	117.9 ± 9.97	137.6 ± 7.05	0.03	S (Student's 'T' test, unpaired)
20 minutes	115.88 ± 6.70	132.15 ± 5.65	0.04	S (Student's 'T' test, unpaired)
25 minutes	114.44 ± 6.4	127.94 ± 6.55	0.05	S (Student's 'T' test, unpaired)
30 minutes	117.1 ± 3.94	121.63 ± 9.62	0.08	NS (Student's 'T' test, unpaired)

Values are presented as mean ± SD. Analysis was done by Student's 't' test. NS: Not significance $p > 0.05$ (among two groups). S : Significant $p < 0.05$ (among two groups).

Table V *Diastolic blood pressure in two groups*

Time	Group I (n=30)	Group II (n=30)	p value	Result
Pre induction	77.9 ± 9.84	78.1 ± 11.43	0.86	NS (Student's 'T' test, unpaired)
After induction	78.35 ± 3.65	79.45 ± 4.84	0.90	NS (Student's 'T' test, unpaired)
5 minutes	77.2 ± 3.75	88 ± 10.4	0.04	S (Student's 'T' test, unpaired)
10 minutes	74.80 ± 5.05	86.60 ± 5.90	0.03	S (Student's 'T' test, unpaired)
15 minutes	77.9 ± 9.97	87.6 ± 7.05	0.03	S (Student's 'T' test, unpaired)
20 minutes	75.88 ± 6.70	85.15 ± 5.65	0.04	S (Student's 'T' test, unpaired)
25 minutes	74.44 ± 6.4	83.94 ± 6.55	0.05	S (Student's 'T' test, unpaired)
30 minutes	76.1 ± 3.94	81.63 ± 9.62	0.08	NS (Student's 'T' test, unpaired)

Values are presented as mean ± SD. Analysis was done by Student's 't' test. NS: Not significance $p > 0.05$ (among two groups). S: Significant $p < 0.05$ (among two groups).

But the mean blood pressure was significantly higher only during the procedure in group II patients ($P < 0.05$) when compared to group I patients (Table: VI).

Table VI *Mean blood pressure in two groups*

Time	Group I (n=30)	Group II (n=30)	p value	Result
Pre induction	87.9 ± 9.84	88.1 ± 11.43	0.86	NS (Student's 'T' test, unpaired)
After induction	88.35 ± 3.65	89.45 ± 4.84	0.90	NS (Student's 'T' test, unpaired)
5 minutes	87.2 ± 3.75	100 ± 10.4	0.04	S (Student's 'T' test, unpaired)
10 minutes	84.80 ± 5.05	106.60 ± 5.90	0.03	S (Student's 'T' test, unpaired)
15 minutes	87.9 ± 9.97	107.6 ± 7.05	0.03	S (Student's 'T' test, unpaired)
20 minutes	85.88 ± 6.70	105.15 ± 5.65	0.04	S (Student's 'T' test, unpaired)
25 minutes	84.44 ± 6.4	103.94 ± 6.55	0.05	S (Student's 'T' test, unpaired)
30 minutes	86.1 ± 3.94	101.63 ± 9.62	0.05	S (Student's 'T' test, unpaired)

Values are presented as mean ± SD. Analysis was done by Student's 't' test. NS: Not significance $p > 0.05$ (among two groups). S: Significant $p < 0.05$ (among two groups).

There was no significant difference in respiratory rate between two groups although increased respiration observed in both groups during induction which subsequently came down to baseline level during recovery (Table: VII).

Table VII *Respiratory rate in two groups*

Time	Group I (n=30)	Group II (n=30)	p value	Result
Pre induction	16.9 ± 9.84	15.1 ± 1.43	0.86	NS (Student's 'T' test, unpaired)
After induction	15 ± 3.65	16.45 ± 1.84	0.90	NS (Student's 'T' test, unpaired)
5 minutes	14.2 ± 3.75	14 ± 10.4	0.94	NS (Student's 'T' test, unpaired)
10 minutes	14.80 ± 5.05	16.60 ± 5.90	0.30	NS (Student's 'T' test, unpaired)
15 minutes	15.9 ± 9.97	17.6 ± 7.05	0.35	NS (Student's 'T' test, unpaired)
20 minutes	15.88 ± 6.70	15.15 ± 5.65	0.40	NS (Student's 'T' test, unpaired)
25 minutes	14.44 ± 6.4	13.94 ± 6.55	0.50	NS (Student's 'T' test, unpaired)
30 minutes	16.1 ± 3.94	16.63 ± 9.62	0.45	NS (Student's 'T' test, unpaired)

Values are presented as mean ± SD. Analysis was done by Student's 't' test. NS: Not significance $p > 0.05$ (among two groups). S: Significant $p < 0.05$ (among two groups).

The mean time required to complete the procedure was (20.2 ± 8.9) minute in group I and (24.7 ± 9.71) minute in group II & the difference was not statistically significant (Table: VIII).

Table VIII *Procedure time in two groups*

Time	Group I (n=30)	Group II (n=30)	p value	Result
Procedure time	20.2 ± 8.9	19.83 ± 9.71	0.75	NS (Student's 'T' test, unpaired)

Values are presented as mean ± SD. Analysis was done by Student's 't' test. NS: Not significant $p > 0.05$ (among two groups). Sig: Significant $p < 0.05$ (among two groups).

The recovery time in group I patients was (24.7 ± 3.6) min and (29.7 ± 4.1) min in group II patients and the difference was statistically significant ($P < 0.05$) (Table: IX).

Table IX *Recovery time in two groups*

Time	Group I (n=30)	Group II (n=30)	p value	Result
Recovery time	24.7 ± 3.6	29.7 ± 4.1	0.02 S	(Student's 'T' test, unpaired)

Values are presented as mean ± SD. Analysis was done by Student's 't' test. NS: Not significance $p > 0.05$ (among two groups). Sig: Significant $p < 0.05$ (among two groups).

Immediately after recovery the differences of recovery score found statistically not significant between groups but was significant later (Table: X)

Table X *Average recovery score in two groups*

Time	Group I (n=30)	Group II (n=30)	p value	Result
After 10 minutes	6.93 ± 0.78	6.87 ± 0.73	1.00	NS (Student's 'T' test, unpaired)
After 20 minutes	8.93 ± 0.78	7.87 ± 0.73	0.01	S (Student's 'T' test, unpaired)
After 30 minutes	9.96 ± 0.21	8.28 ± 0.48	0.02	S (Student's 'T' test, unpaired)

Values are presented as mean ± SD. Analysis was done by Student's 't' test.

NS: Not significance $p > 0.05$ (among two groups). Sig: Significant $p < 0.05$ (among two groups).

There were few drug related complications in both groups during procedure and recovery. Twenty four patients (80%) had raised BP and twenty three of them also developed tachycardia in group II while these incidences were negligible in group I patients (Table: XI). Although none of them required any extra medication to combat these

haemodynamic changes. Eight patients of group II had profuse bronchial secretion compared to three of group I who were treated with injection Atropine 0.02mg/kg body weight. Frequency of desaturation ($\text{Spo}_2 < 90\%$) was more on group I. Difference between the groups regarding complication was statistically significant (Table: XI).

Table XI *Complications during procedure in two groups*

	Group I		Group II		P value	Result
	Count	%	Count	%		
High blood pressure	7	23.3%	24	80.0	0.02	S(Chi square) test (Student's "T" test, unpaired)
Tachycardia	6	20.0%	23	76.7		
Profuse secretion	3	10.0	8	26.7		
Desaturation	4	13.3%	2	6.7		

During recovery 11 patients (36%) developed recovery agitation in group II but 3 patients (10%) in group I. All of them were further sedated with Injection Midazolam. Mean BP remained higher than the baseline in 4 patients (3.3%) in group I. The differences of recovery agitation and raised mean blood pressure (MBP) between two groups were statistically significant (Table: XII). Few patients of both groups experienced post-operative nausea & vomiting but the incidences were not statistically significant.

Table XII *Complications during recovery in two groups*

	Group I		Group II		p value	Result
	Count	%	Count	%		
Recovery agitation	3	10.0%	11	36.7%	0.042	S(Chi square)
Profuse secretion	1	3.3%	3	10.0%		
Vomiting	3	10.0%	2	6.7%		
High blood pressure	1	3.3%	4	13.3%		
Nausea	2	6.7%	2	6.7%		

Average cost required for Ketofol group (Taka 76.60 ± 17.50) was significantly higher than that of group II (Ketamine + Diazepam) patients (Taka 29.43 ± 6.23) (Table XIII).

Table XIII *Average cost of sedation in two groups*

Time	Group I (n=30)	Group II (n=30)	p value	Result
Cost of sedation	Tk 76.60 ± 17.52	TK 29.43 ± 6.23	0.05	S(Chi square test)

Discussion

This study shows that ketofol is an effective and apparently safe drug for PSA in day case surgery. ketamine and propofol are physiologically compatible when administered together, the mixture of ketamine and propofol in a single syringe in 1:1 ratio offers a simple practical approach to medication in preparation and use⁵. Ketofol can be used in patients of all ages and with a broad range of acute and chronic co-morbid conditions with a wide safety limit and high level of patient satisfaction which highlights its versatility in the short minimally invasive procedure in emergency department or day case surgery⁶. In this study ketamine is mixed with propofol in sub anaesthetic dose which complements the effect of each drug. Mean dose of ketofol required to produce induction is 0.75 mg/kg of ketamine and propofol each, subsequent top up dose (0.25 mg/kg) given as and when required depending on length of the procedure. However, two patients of ketofol group (group 1) required larger than the mean dose of ketofol which may be due to the extent of tissue trauma, type of procedure or individual patient variation which simulates the other studies^{6,7}.

The sign of pain in group 1 patients as evaluated by tachycardia, tachypnoea, hypertension, sweating, movements were absent or insignificant unlike in group II patients as found in the study. The results are consistent with Furdy et al⁸ & Hui et al⁽⁹⁾ who suggested that minimal change observed in arterial pressure may be dose related and opposite action of ketamine and propofol on hemodynamic system.

Aboeldahab H et al¹⁰ studied on sixty patients and compared the hemodynamic status between propofol ketamine and their combination as induction agent and found that MAP decreased in propofol group, increased in ketamine group and remains comparable to baseline in ketofol group and the differences were statistically significant. Akon A et al¹¹ published a trail of sixty patients between one month and thirteen years of age undergoing cardiac catheterization received sedation with propofol and or ketofol (at a ratio of 3:1) significant decrease in MAP in eleven patients in propofol group and three patients in ketofol group.

In this study all patients of both groups had increased depth of respiration initially without any change in rate although four patients developed subtle hypoxia in group I and two in group II may be due to over sedation or increased salivation subsided with simple head extension. Persson J et al¹² reported that ketamine induced sympathoadrenal activation may account for improved ventilation.

Mean recovery time in group I patient was significantly better than group II patients in this study which simulate with a study done by Saeed E et al¹³ who evaluated three ratios of ketofol infusion (ketamine: propofol 1:1, 1:2, 1:3) for close reduction of distal arm fractures & recommended ketofol 1:2 as an appropriate procedural sedation modalities providing early recovery and shorter hospital stay with minor haemodynamic changes and postoperative side effects.

Adverse events as found in this study were very few and there was no case of hypertension, bradycardia vomiting or laryngospasm in any group. During procedure 80% patients developed insignificant rise of BP from baseline in Group II in comparison to 23.3% patients of Group I. about 33.3% patients of Group II had agitation during recovery period in comparison to 10% as Group I. Higher rate of emergence reaction, postoperative vomiting, compromised airway were found in ketamine monotherapy specially when used (> 2.5 mg/kg) as found by Green SM et al⁽¹⁴⁾ & Strayer et al⁽¹⁵⁾. The suggested explanation is ketamine and propofol complements each other to be more effective clinically while counter acting each other's adverse effects.

Midazolam could be a better alternative than diazepam as it has shorter elimination half- life (2-3 hrs) compared to diazepam (20-50 hrs) which would allow a shorter recovery time in Group II if selected. However, the study was done giving more emphasis on easily available, cheaper and popular drug. It would be more scientific if we could analyze blood gases of all patients and also duration of analgesia. Feedback of patients also not accomplished as most of the procedure was done on outdoor basis.

Conclusion

Combination of ketamine and propofol in bolus (1:1) form provides safer and more effective sedation

and analgesia in PSA than commonly used ketamine and Diazepam combination. Ketofol ensures better haemodynamic stability, procedural success, smooth and early recovery with negligible complications. Ketofol can be a handy option in the armoury of anaesthesiologist for short duration procedures specially in the area of low resources.

References

1. ACEP, Clinical policy: Procedural Sedation and Analgesia in the Emergency Department. *Annals of Emergency Medicine* 2005 February; 42(2):177-196
2. Aouad M, Moussa A, Dagher C. Addition of Ketamine to propofol for Initiation of Procedural Anaesthesia in Children Reduces propofol consumption and Preserves Haemodynamic Stability. *Acta Anaesthesiologica Scandinavica* 2008; 52(4):561-565
3. Ramsay MA, Savage TM, Simpson BR, Goodwin R. Controlled sedation with alphaxolone-alphadolone
4. Aldrete JA. The post anaesthesia recovery score-revisited. *J Clin Anaesth* 1995; 7:89
5. Trissel LA, Gilbert DL, Martinez JF. Compatibility of Propofol Injectable Emulsion with Selected Drugs During Simulated Y-Sight Administration. *Am J Hosp Pharm* 1997; 54:1281-92.
6. Williams EV, Andolfetto G. A Prospective Evaluation of "Ketofol" (Ketamine/ Propofol combination) for Procedural Sedation and Analgesia in the Emergency Department. *Annals of Emergency Medicine* 2007 January; 49(1):23-30.
7. Andolfatto G, Williams E. A prospective case series of single syringe Ketamine – propofol (ketofol) for emergency department procedural sedation and analgesia in adults. *Academic Emergency Medicine*, March 2011;18(3)
8. Furuya A, Matsukawa T, Czaki M, Nishiyama T, Kume M, Kumazawa T. Intravenous Ketamine attenuates arterial pressure changes during induction of anaesthesia with Propofol. *Eur J Anaesthesiology* 2001; 18: 88-92.
9. Hui TW, Short TG, Hong W, Suen T, Gin T, Plummer J. Additive interactions between Propofol and Ketamine when used for anaesthesia induction in female patients. *Eur J Anaesthesiology* 1995; 82: 641-648.
10. Aboeldahab H, Samir R, Hosny H, Omar A. Comparative study between Propofol Ketamine and their combination (ketofol) as an induction agent. *Egyptian Journal of Anaesthesia* 2011;27:145-150.
11. Akin A, Esmaoglu A, Guler G, Demercioglu R, Narin N, Boyaci A. Propofol and Propofol Ketamine in paediatric patient undergoing cardiac catheterization. *Pediatr Cardiol* 2005; 26(5): 553-557.
12. Frey K, Sukhani R, Pawlowski J, et al. Propofol versus Propofol-Ketamine sedation for retrobulbar nerve block: comparison of sedation quantity, intraocular pressure changes and recovery profiles. *Anaesth Analg* 1999; 89: 317-321.
13. Saeed E. Ketofol infusion as a procedural sedation and analgesia for minor orthopaedic surgeries: evaluation of dose- outcome relation. *Ain Shams Journal of Anaesthesiology* 2011; 4(1): 63-74.
14. Green SM, Krauss B. Clinical practice guideline for emergency department Ketamine dissociative sedation in children. *Annals of Emergency Medicine* 2004; 44: 460-471.
15. Strayer RJ, Nelson LS. Adverse events associated with Ketamine for procedural sedation in adults. *American Journal of Emergency Medicine* 2008; 26: 985-1028.

Efficacy of Magnesium Sulphate to Prevent Arterial Pressure Increases During Laparoscopic Cholecystectomy

Naimul Hoq¹, Abdullah Al Maruf²

¹Classified Specialist in Anaesthesiology, Department of Anaesthesia, CMH, Dhaka, ²Classified Specialist in Anaesthesiology, Department of Anaesthesia, BGB Hospital, Pilkhana, Dhaka

Corresponding author: Email: naim838@gmail.com

Abstract:

Background: Carbon dioxide pneumoperitoneum (PP) for laparoscopic surgery increases arterial pressure and systemic vascular resistance, these vasopressor responses are likely to be due to increased release of catecholamine. Magnesium is well known to inhibit catecholamine release and attenuate vasopressin stimulated vasoconstriction.

Objectives: This study has been undertaken with a view to find out the efficacy of magnesium sulphate to prevent arterial pressure increases associated with carbon dioxide pneumoperitoneum (PP) in patients undergoing laparoscopic cholecystectomy.

Methods: Sixty patients, of either sex, 18-60 years of age, undergoing elective laparoscopic cholecystectomy were randomly allocated in one of the two groups containing 30 patients each. Magnesium Sulphate Group "M" received magnesium sulphate 30 mg/kg intravenously as a bolus before carbon dioxide pneumoperitoneum (PP). A control Group "C" received same volume of normal saline before carbon dioxide pneumoperitoneum (PP).

Results: Mean arterial pressure was significantly less throughout the period of pneumoperitoneum in patients of Group M. Intravenous labetalol was required in 46.66% (14 out of 30) of the patients in group C to control intraoperative hypertension and it was clinically significant in comparison to group M.

Conclusion: Our study concluded that intravenous Magnesium Sulphate administered before carbon dioxide pneumoperitoneum (PP) attenuates arterial pressure increases during laparoscopic cholecystectomy effectively and provides better hemodynamic stability.

Key words: Cholecystectomy, Pneumoperitoneum, Mean Arterial Pressure, Magnesium Sulphate.

(JBSA 2018; 31(2): 82-87)

Introduction:

Laparoscopic cholecystectomy is very popular now a days, carbon dioxide is commonly used to create pneumoperitoneum (PP) in laparoscopic cholecystectomy.^{1,2} Both carbon dioxide and pneumoperitoneum affects homeostasis and leads to alterations in cardiovascular physiology, pulmonary physiology and stress responses.^{1,2,3,4,5} Cardiovascular changes include increase in mean arterial pressure (MAP) with no significant change in heart rate,^{6,7,8} decrease in cardiac output and

increase in systemic vascular resistance (SVR). These vasopressor stress responses are consequent to hypercarbia induced release of catecholamine,^{9,10,11} vasopressin, or both.^{6,12,13} These circulatory responses to PP is usually attenuated by opioids,¹⁴ vasodilators,¹⁵ beta blocking agents¹⁶ and alpha 2 adrenergic agonists.¹⁷ Magnesium has the ability to block the release of catecholamine from both the adrenal gland and the adrenergic nerve terminals.¹⁸

Magnesium also produces vasodilation by acting directly on blood vessels¹⁹ and in high doses magnesium attenuates vasopressin mediated vasoconstriction.²⁰

In this study, we investigated the efficacy of magnesium sulphate to prevent arterial pressure increases in patients undergoing laparoscopic cholecystectomy.

Materials and methods:

This prospective clinical study was conducted at department of Anaesthesia, CMH Dhaka from January 2017 to June 2017. After approval from departmental review board and obtaining patient's written informed consent, 60 patients of American Society of Anesthesiologists (ASA) physical status I & II aged 18-65 years undergoing elective laparoscopic cholecystectomy with CO₂ pneumoperitoneum, were enrolled in this study. Patients with a known allergy to magnesium sulphate, any degree of heart block, hypertension, diabetes mellitus, cardiovascular or kidney disease, acute cholecystitis were excluded from the study. Patients are assigned to one of the two groups, each containing 30 patients, Group M (Magnesium Sulphate Group) and Group C (Control Group). All the patients received tab. diazepam 10 mg orally on the night before surgery. On arrival to operation theatre ECG, pulse oximetry, non-invasive blood pressure (NIBP) monitoring were started and baseline vital parameters like heart rate, mean arterial pressure (MAP), and arterial oxygen saturation (SPO₂) were recorded. An intravenous line was started. Patients were induced with pethidine 1.5 mg/kg and thiopentone sodium 5 mg/kg. Endotracheal intubation was facilitated by muscle relaxant vecuronium 0.1 mg/kg. Group M patients received magnesium sulphate 30 mg/kg intravenously as a bolus and group C received same volume of normal saline as a bolus intravenously immediately before pneumoperitoneum. Anaesthesia was maintained with 33% O₂ in N₂O, 0.5% halothane and vecuronium. CO₂ was insufflated into the peritoneal cavity to create PP. Intra abdominal pressure (IAP) was maintained to 14 mmHg throughout the laparoscopic procedure. All the patients were positioned in a

head up tilt for about 15°. During hemodynamic fluctuations, the following medical interventions were taken: for hypotension (MAP < 60 mmHg) increased rate of infusion of i.v fluid and/or i.v bolus dose of inj. Ephedrine 5 mg and for hypertension (MAP > 110 mmHg) i.v bolus dose of inj. Labetalol 5 mg. At the end of the operation, ondansetron 4 mg i.v was administered for prophylaxis against nausea and vomiting. Residual neuromuscular block was reversed by appropriate dose of neostigmine and atropine and tracheal extubation was performed. Heart rate and MAP were recorded at the following points of time: (1) Baseline (2) before PP (3) 15 min, 30 min, 45 min and 60 min after PP (4) after release of PP and (5) after extubation. Patients were observed for any adverse events like bradycardia, hypotension, and hypertension during postoperative period.

All the variables were expressed as mean $\pm$ SD; student t-test and chi-square test were done as the test of significance. The statistical analysis was done by using Graph pad software. P-value < 0.05 was considered as statistically significant.

Results:

Patient's demographics data were shown in Table-I. Both the groups were comparable with respect to age, sex, body weight and duration of surgery. No statistical significant differences were found between the two study groups.

Table- I Patient's characteristics and duration of surgery

	Group M (n=30)	Group C (n=30)	P value
Age (years)	42.5 $\pm$ 9.77	44.66 $\pm$ 7.88	0.3498
Sex (M/F)	12/18	11/19	
Weight (kg)	60.55 $\pm$ 9.7	63.30 $\pm$ 5.4	0.1801
Duration of surgery (min)	70.7 $\pm$ 4.8	72.4 $\pm$ 5.6	0.2118

Values were expressed as Mean $\pm$ SD, values are regarded significant if P-value < 0.05. There was no significant changes between the groups.

Changes in heart rate were shown in Table-II. No statistical significant differences were found between the two study groups.

Table-II *Changes in heart rate*

	Group M (n=30)	Group C (n=30)	P value
Preoperative	73.4±7.3	75.3±5.2	0.2504
Before PP	78.8±9.5	79.2±7.9	0.8599
15 min after PP	82.4±9.5	84.88±12.7	0.3953
30 min after PP	81.6±9.7	86.4±14.3	0.1336
45min after PP	82.3±7.7	85.7±11.9	0.1941
60min after PP	81.9±12.3	84.8±12.7	0.3727
After release of PP	79.7±13.7	82.4±13.9	0.4517
After extubation	90.5±8.7	92.5±8.6	0.3742

Values were expressed as Mean ± SD, values are regarded significant if P-value < 0.05

There was no significant change in heart rate between the groups.

Changes in mean arterial pressure (MAP) were shown in Table-III. There was no significant difference in the preoperative mean arterial pressure (MAP) values and before pneumoperitoneum (PP) values between two study groups. MAP values in Group-M were significantly lower ($P < 0.05$) throughout the PP, after the release of PP and after extubation compared to Group-C.

Table-III
Changes in mean arterial pressure

	Group M (n=30)	Group C (n=30)	P value
Preoperative	91.5±7.4	92.3±8.7	0.7026
Before PP	95.6±8.5	97.6±7.9	0.3491
15 min after PP	97.8±9.2	107.7±11.9	0.0006
30 min after PP	98.4±8.5	108.9±12.4	0.0003
45 min after PP	96.5±9.9	106.6±8.87	0.0001
60 min after PP	97.2±5.5	107.5±7.45	0.0001
After release of PP	93.2±6.8	102.4±11.7	0.0004
After extubation	95.5±8.7	105.8±9.8	0.0001

Values were expressed as Mean ± SD, values are regarded significant if P-value < 0.05. MAP vary significantly from 15 min after PP to extubation between the groups.

No patient suffered from bradycardia or hypotension in our study. Hypertension occurred in 14 patients of group C, whereas no patient of group M suffered from hypertension. (Table-IV) The difference was statistically significant ($P < 0.05$).

Table-IV
Distribution of patients according to adverse effects

Adverse effects	Group M (n=30)	Group C (n=30)	Percentage %	P value
Bradycardia	0	0	0.0	-
Hypotension	2	0	6.67	0.1502
Hypertension	0	14	46.67	0.0001

Values are regarded as significant if P-value < 0.05.

Discussion:

In this study, we studied the effects of magnesium sulphate on hemodynamic in patients undergoing laparoscopic cholecystectomy. During laparoscopic surgery, CO₂ is routinely used to create PP. Elevated IAP induced by PP and CO₂ itself produce some adverse effects on the cardiovascular and respiratory system.^{1,2,3,4,5} Immediately after PP, plasma level of catecholamine's and vasopressin is increased.^{9,10,12,13} Increased catecholamine level activates the renin angiotensin aldosterone system (RAAS) leading to some characteristic hemodynamic alterations^{3,4,9,10} which include decreased cardiac output, elevated arterial pressure, and increased systemic pulmonary vascular resistance. Vasopressin also contributes to elevation of arterial pressure and systemic vascular resistance.^{12,13} As already mentioned in introduction, various pharmacological agents have been used to attenuate these adverse hemodynamic effects of PP.^{14,15,16,17} Magnesium is effective in blocking the release of catecholamine's from both adrenergic nerve terminals and the adrenal gland.¹⁸ Besides, magnesium produces vasodilatation by acting directly on blood vessels.¹⁹ In addition to catecholamine's, vasopressin is a major contributor to the hemodynamic changes induced by PP.^{12,13} Magnesium attenuates vasopressin stimulated vasoconstriction.²⁰ Laparoscopic cholecystectomy is performed in reverse

trendelenburg position, this particular position causes diminished venous return which ultimately leads to further decrease in cardiac output.²¹

In a study, James *et al.*²² observed that i.v magnesium sulphate was able to attenuate the adverse hemodynamic response of endotracheal intubation. Because of the ability of magnesium sulphate to attenuate adverse hemodynamic response, we have administered 30 mg/kg magnesium sulphate as a bolus before PP and observed its effect on hemodynamic response to PP. Telci and *et al.*²³ used i.v Magnesium sulphate in a dose of 30 mg/kg bolus before induction and 10 mg/kg/h continuous i.v infusion in a study to observe the efficacy of magnesium sulphate to decrease anaesthetic requirement.²³ We did not administer any infusion of magnesium intraoperatively. In our study we observed that i.v magnesium sulphate in a bolus dose of 30 mg/kg before PP was able to attenuate the adverse hemodynamic response. Diamant *et al.*²⁴ reported 35% decrease in cardiac output in dog with a raised IAP of 40 mmHg. Ishizaki *et al.*²⁵ tried to evaluate the safe IAP during laparoscopic surgery. They observed significant fall in cardiac output at 16 mmHg of IAP and hemodynamic alterations were much less at 14 mm Hg of IAP. So in our study, we kept IAP 14 mm Hg. In spite of maintaining normocapnia and keeping IAP 14mm Hg, there was significant rise of MAP in patients of group C. However, in group M, hemodynamic responses to PP were effectively blunted and MAP remained at a significantly lower level compared to group C. Thus i.v magnesium sulphate effectively attenuated adverse hemodynamic response. Regarding the incidence of adverse effects, no patient of either group suffered from bradycardia in our study. Hypertension occurred in 14 patients of group C for which they had to be treated with Inj. labetalol whereas no patient of group M suffered from hypertension.

To conclude, administration of magnesium sulphate before commencement of Carbon dioxide pneumoperitoneum effectively prevent arterial pressure increases during laparoscopic cholecystectomy and thereby provides perioperative hemodynamic stability.

References

1. Hodgson G, McClelland RM, Newton JR. Some effects of the peritoneal insufflation of carbon dioxide at laparoscopy. *Anaesthesia* 1970; 25:382-90
2. Blobner M, Felber AR, Göglér S, Weigl EM, Jelen ES. Carbon dioxide uptake from the pneumoperitoneum during laparoscopic cholecystectomy. *Anesthesiology* 1992; 77: A37-40
3. Richardson JD, Trinkl JK. Haemodynamic and respiratory alterations with Increased intra-abdominal pressure. *J Surg Res* 1976; 20:401-4
4. Lenz RJ, Thomas TA, Wilkins DG. Cardiovascular changes during laparoscopy. Studies of stroke volume and cardiac output using impedance cardiography. *Anaesthesia* 1976; 31:4-12
5. McLaughlin JG, Scheeres D, Dean R, Bonnell BW. The adverse haemodynamic effects of laparoscopic cholecystectomy. *Surg Endosc* 1995; 9:121-4
6. Joris JL, Chiche JD, Canivet JL, Jacquet NJ, Legros JJ, Lamy ML. Haemodynamic changes induced by laparoscopy and their endocrine correlates: Effects of clonidine. *J Am Coll Cardiol* 1998; 32:1389-96
7. Wabha RW, Beique F, Kleiman SJ. Cardiopulmonary function and laparoscopic cholecystectomy. *Can J Anaesth* 1995; 42:51-63
8. Sharma KC, Brandstetter RD, Brensilver JM, Jung LD. Cardiopulmonary physiology and pathophysiology as a consequence of laparoscopic surgery. *Chest* 1996; 110:810-5
9. Mikami O, Kawakita S, Fujise K, Shingu K, Takahashi H, Matsuda T. Catecholamine release caused by carbon dioxide insufflation during laparoscopic surgery. *J Urol* 1996; 155:1368-71
10. Myre K, Rostrup M, Buanes T, Stokland O. Plasma catecholamines and haemodynamic changes during pneumoperitoneum. *Acta Anaesthesiol Scand* 1998; 42:343-7
11. Koivusalo AM, Kellokumpu I, Scheinin M, Tikkanen I, Halme L, Lindgren L.

- Randomized comparison of the neuro-endocrine response to laparoscopic cholecystectomy using either conventional or abdominal wall lift techniques. *Br J Surg* 1996; 83:1532-6
12. Walder AD, Aitkenhead AR. Role of vasopressin in the haemodynamic response to laparoscopic cholecystectomy. *Br J Anaesth* 1997; 78:264-6
 13. Mann C, Boccara G, Pouzeratte Y, Eliet J, Serradel-Le Gal C, Vergnes C, *et al.* The relationship among carbon dioxide pneumoperitoneum, vasopressin release, and haemodynamic changes. *AnesthAnalg* 1999; 89:278-83
 14. Lentschener C, Axler O, Fernandez H, Megarbane B, Billard V, Fouqueray B, *et al.* Haemodynamic changes and vasopressin release are not consistently associated with carbon dioxide pneumoperitoneum in humans. *Acta Anaesthesiol Scand* 2001; 45:527-35.
 15. Joris JL, Hamoir EE, Hartstein GM, Meurisse MR, Hubert BM, Charlier CJ, *et al.* Haemodynamic changes and catecholamine release during laparoscopic adrenalectomy for pheochromocytoma. *AnesthAnalg* 1999; 88:16-21.
 16. Koivusalo AM, Scheinin M, Tikkanen I, Yli Suomu T, Ristkari S, Laakso J, *et al.* Effects of esmolol on haemodynamic response to CO₂ Pneumoperitoneum for laparoscopic surgery. *Acta Anaesthesiol Scand* 1998; 42:510-7.
 17. Joris JL, Chiche JD, Canivet JL, Jacquet NJ, Legros JJ, Lamy ML. Hemodynamic Changes induced by laparoscopy and their endocrine correlates. *J Am Coll Cardiol* 1998; 32:1389-96.
 18. Lishajko F. Releasing effect of calcium and phosphate on catecholamine's, ATP, and protein from chromaffin cell granules. *Acta Physiol Scand* 1970; 79:575-84.
 19. Altura BM, Altura BT. Magnesium and vascular tone and reactivity. *Blood Vessels* 1978; 15:5-16.
 20. Laurant P, Touyz RM, Schiffrin EL. Effect of magnesium on vascular tone and reactivity in pressurized mesenteric resistance arteries from spontaneously hypertensive rats. *Can J Physiol Pharmacol* 1997; 75:293-300.
 21. Wilcox S, Vandar LD. Trendelenburg and his position! A critique of its uses and effectiveness. *AnesthAnalg* 1988; 67:574-8.
 22. James MF, Beer RE, Esser JD. Intravenous magnesium sulphate inhibits catecholamine release associated with tracheal intubation. *Anesth Analg* 1989; 68:772-6.
 23. Telci L, Esen F, Ackora D, Erden T, Canbolat T, Akpir K. Evaluation of effects of magnesium sulphate in reducing intraoperative anaesthetic requirements. *Br J Anaesth* 2002; 89:594-8.
 24. Diamant M, Benumof JL, Saidman LJ. Haemodynamic of increased intra abdominal pressure: Interaction with hypovolemia and halothane anaesthesia. *Anaesthesiology* 1978; 48:23-7.
 25. Ishizaki Y, Bandai Y, Shimomura K, Abe H, Ohtomo Y, Idezuki Y. Safe intraabdominal pressure of carbon dioxide pneumoperitoneum during laparoscopic surgery. *Surgery* 1993; 114:549-54.

Role of Ephedrine for Management of Hypotension During Spinal Anaesthesia for Caesarean Delivery

Esrat Zahan¹, Md. Zakir Hossain², Abdur Rahman³, Waheeda Nargirs⁴

¹Medical Officer, Department of Anaesthesiology, Uttara Adhunik Medical College & Hospital, ²Senior Consultant & Admin, Head Dept. of Anaesthesiology, Uttara Adhunik Medical College & Hospital, ³Professor and Head of Intensive Care Unit, Bangladesh Medical College & Hospital, ⁴Associate Professor, Department of Biochemistry, Uttara Adhunik Medical College & Hospital

Corresponding Author: Dr. Esrat Zahan, Medical Officer, Dept. of Anaesthesiology, Uttara Adhunik Medical College & Hospital

Abstract

Background: Hypotension during spinal anaesthesia for caesarean section remains a common scenario in our clinical practice. Certain risk factors play a role in altering the incidence of hypotension. Ephedrine has been the drug of choice for more than 30 years in the treatment of spinal anesthesia induced maternal hypotension. It has a good safety record, ready availability, and familiarity to most anesthesiologists.

Aims: To determine the efficacy and safety of prophylactic bolus dose of 0.5 mg/kg intravenous ephedrine for the prevention of hypotension during spinal anesthesia for cesarean delivery.

Methods: It was designed a randomized, double-blinded study. Patients were randomly allocated into two groups: ephedrine group (n=30) and control group (n=30). Intravenous preload of 15 mL/kg lactated Ringer's solution was given. Shortly after the spinal injection, ep-hedrine 0.5 mg/kg or saline was injected intravenous for 60 sec.

Results: The mean of high-est and lowest heart rate in the ephedrine group was higher than those of control group ($p < 0.05$). There were significant lower incidences of hypotension and nau-sea and vomiting in the ephedrine group compared with the control group 11(36.7%) vs. 24(80.0%); 6(20.0%) vs. 17 (56.7%), respectively) ($p < 0.05$). The first rescue ephedrine time in the ephedrine group was significantly longer (14.9 ± 7.1 min vs. 7.9 ± 5.4 min) than that of the control group ($p < 0.05$). Neonatal outcome were similar between the study groups.

Conclusion: The above findings suggest, the prophylactic bolus dose of 0.5 mg/kg intravenous ephedrine given at the time of intrathecal block after a crys-talloid fluid preload, plus rescue boluses reduce the incidence of hypotension.

Key Words: Anesthesia, Spinal; Cesarean Section; Ephedrine; Hypotension

(JBSA 2018; 31(2): 88-94)

Introduction

Spinal anesthesia, recently, has been known as an acceptable anesthesia technique, especially for cesarean section, due to advantageous on epidural anesthesia, such as rapid onset, intensity, symmetric sensory and motor block^{1,2}. However, hypotension triggered by spinal anesthesia during cesarean delivery has been known as a common complication that might endanger the lives of both mother and fetus.

Spinal anesthesia provides a fast, profound, and symmetrical sensory and motor block of high quality in patients undergo-ing cesarean delivery^{1,2}. The most common serious adverse effect of spinal anesthesia for cesarean delivery is hypotension, with a reported incidence greater than 80%³.

A number of strategies for preventing hypotension have been investigated, because it may have detrimental mater-nal and neonatal effects. The

use of lateral uterine displacement is routine procedure to prevent hypotension⁴. Other strategies have included the use of intravenous fluid preload, gravity (Trendelenburg or leg rising), compression devices on the legs, and prophylactic vasopressors¹. However, no methods have proved satisfactory. Ephedrine is the most commonly used drug among the vasopressors.

The prophylactic administration of ephedrine by the intra-muscular route is very controversial because its systemic absorption and peak effect are difficult to predict, thus, possibly resulting in rebound hypertension⁵. The intravenous route may be more effective and controllable, although large doses are used; the incidence of hypotension was still high in some studies^{6,7}.

Intravenous ephedrine given immediately after the induction of spinal anesthesia has been described^{7,8}. Doses of 10-20-30 mg or 0.25 mg/kg were not effective in eliminating hypotension completely⁷⁻¹⁰. Therefore, we designed a case controlled study to determine efficacy and safety of 0.5 mg/kg intravenous ephedrine for preventing hypotension during spinal anesthesia for cesarean delivery.

Methods

It was designed a randomized, double-blinded study. During the study period, 60 consecutive patients were identified suitable for the study. They were women, ASA status I or II, undergoing elective cesarean section under spinal anesthesia and included in the study. Written informed consent was obtained from each subject, and the study protocol was approved by the Ethical Committee of Uttara Adhunik Medical College, Uttara, Dhaka. Patients with pre-existing or pregnancy-induced hypertension, known cardio-vascular or cerebrovascular disease, abnormal cardiotocography (CTG) tracing, or contraindications to spinal anesthesia were excluded. Randomization was based on a computer-generated code that was prepared at a remote site and sealed in opaque, sequentially numbered envelopes. The patients were randomly divided into 2 groups: ephedrine group (n=30) and control group (n=30) after spinal anesthesia.

None of patients was premeditated. On arrival in the operation room, baseline measurements of systolic arterial pressure (SAP) and heart rate (HR) were calculated with a Criticare System 1100

monitor as the mean of three successive measurements, 1 min apart and in the modified supine position with at least 15° of left lateral tilt. 18-gauge intravenous cannula was sited in the non-dominant hand and intravenous preload of 15 mL/kg lactated Ringer's solution was given, within 15 min, after which the intravenous infusion was slowed to the minimum rate required to maintain vein patency.

Spinal anesthesia was administered with the patient in the right lateral position. After skin infiltration with lidocaine, a 25-gauge Whitacre needle was inserted at the L2-3 or L3-4 vertebral interspace and hyperbaric 5% bupivacaine 2 mL with fentanyl 10 mg was deposited intrathecally. The patient was then immediately turned supine with left lateral tilt. Oxygen 4 L/min was given by nasal cannula until delivery.

Shortly after the spinal injection, ephedrine 0.5 mg/kg in the ephedrine group or saline in the control group was injected intravenously slowly. Study medication and management of the patient in the preoperative period was done by the author which the data were collected by a second anesthesiologist who was unaware of the study. The study period started at the time of randomized group up to end of surgery. The blood pressure and heart rate were recorded at 2-min intervals. The baseline SAP and HR, lowest and highest SAP and HR, nausea, vomiting, dizziness, and chest symptoms were recorded up to clamping of the cord and then at 10 min interval till end of surgery. Any hypotension in the intraoperative period were treated with fluid bolus and further IV ephedrine 5 mg bolus. Upper sensory level of anesthesia was achieved a level of 6th thoracic vertebra and the level was ascertained T6 assessing by loss of cold sensation. After ascertained block of T6 surgery was allowed to start.

Hypotension was defined as 20% decrease in SAP from baseline. Hypertension was defined as 20% increase in SAP from baseline. Maternal bradycardia was defined as heart rate <60 beats/min and treated immediately by using intravenous atropine 0.5 mg. Tachycardia was defined as heart rate >120 beats/min. Hypotension was treated immediately by using rescue intravenous ephedrine 5 mg IV bolus until SAP returned to values (>80 of baseline value).²⁹

After delivery, Apgar scores were assessed at 1 and 5 min by the attending pediatrician. Arterial blood

samples were taken from umbilical cord for blood-gas analysis within 2 min. All patients received oxytocin 20 units/L in crystalloid after delivery.

Data were presented as mean±standard deviation, median (range), or percentage, as appropriate. Statistical analyses were performed by SPSS version 22. Demographic parameters, delivery time, first rescue ephedrine time, total ephedrine requirement, umbilical arterial pH, SAP, and HR were compared with t-test. Changes over time in SAP and HR between and within the study groups, comparing values at each time point, were analyzed with repeated measures ANOVA followed by a post hoc Bonferroni test to identify significant differences. Total doses rescue requirement of ephedrine. Hypotension, hypertension, tachycardia, bradycardia and nausea and vomiting of the study groups were compared with Fisher's exact test, as appropriate. A P value of <0.05 was considered significant.

Results

Of 60 patients randomized, two in the both group (n=30). In each study group, 30 patients completed the study protocol. There was no difference between the study groups with regard to the age, weight, height, and delivery time ($p>0.05$) (Table 1). All patients had adequate surgical anesthesia. The median upper sensory level 10 min after the intrathecal injection was T4 (T3-T5) for all the study groups.

Table 1 Comparison of Patient characteristics (n=60)

Variables	Ephedrine group(n=30) Mean±SD	Control group(n=30) Mean±SD	p-value
Age (yr)	25.6±4.1	27.9±6.4	0.103
Height (cm)	156.2±5.2	155.6±4.9	0.647
Weight (kg)	61.9±7.7	61.8±6.9	0.957
Spinal to delivery time (min)	21.0±2.5	20.6±2.6	0.546

Values are expressed as mean±SD, p value reached from Unpaired t-test.

There was no significant difference in the SAP and HR values at baseline between the study groups ($p>0.05$). The mean highest and lowest HR in the ephedrine group was higher than those of control group ($p<0.05$). There were significant differences in mean lowest SAP between the study groups ($p<0.05$). The mean highest SAP in the ephedrine

group was higher than that of control group, but this difference was not significant (Table-2).

Table-II Comparison of systolic arterial pressure and heart rate between case and control group.

Variables	Ephedrine group (n=30) Mean±SD	Control group (n=30) Mean±SD	p-value
Systolic arterial pressure (mmHg)			
Baseline	124.4±2.31	126.2±2.31	0.185 ^{ns}
Lowest	104.5±2.3	92.12±1.83	0.001*
Highest	133.1±3.12	131.8±3.02	0.107 ^{ns}
Heart rate (mmHg)			
Baseline	100.2±3.68	98.9±2.93	0.136 ^{ns}
Lowest	94.6±2.1	85.6±1.75	0.001*
Highest	126.3±2.39	112.3±2.17	0.001*

Values are expressed as mean±SD, p value reached from Unpaired t-test, *significant, ns= not significant

From 2 to 8 min, the mean SAPs in the control group were significantly lower than those of the ephedrine group ($p<0.05$) (Fig. 1). From 6 to 12 min, significant decreases of the mean SAP in the control group were observed as compared with baseline ($p<0.05$) (Fig. 1). From 4 to 8 min, the mean HR in the control group was significantly lower than those of the ephedrine group ($p<0.05$) (Fig. 2).

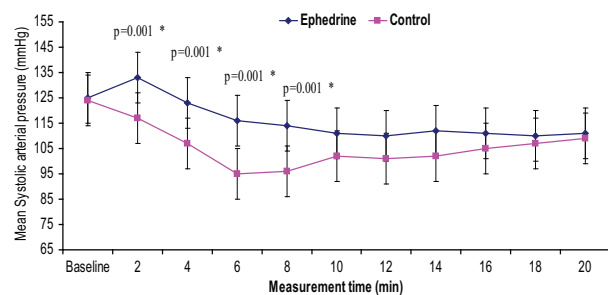


Fig 1 Systolic arterial pressure of the ephedrine and control groups, *significant

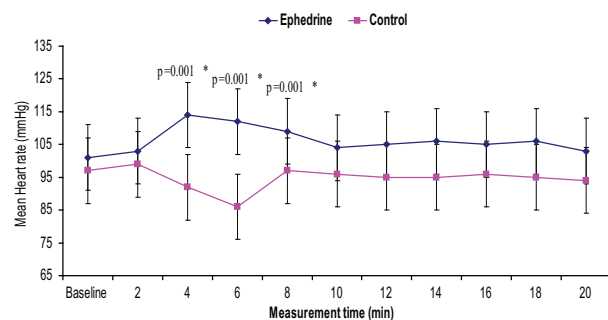


Fig 2 Heart rate of the ephedrine and control groups, *significant

The occurrence of hypotension, hypertension, tachycardia, bradycardia, nausea or vomiting, the total doses rescue ephedrine, and the first rescue ephedrine time are summarized in Table 3. There was significant lower incidences of hypotension in the ephedrine group compared with the control group 11(36.7%) vs. 24(80.0%) ($p<0.05$). There were significant lower incidences of nausea and vomiting in the ephedrine group compared with the control group 6(20.0%) vs. 17(56.7%) ($p<0.05$). There was no difference in the ratio of hypertension between the study groups ($p>0.05$). The ratio of bradycardia in the control group was significantly higher than that of the ephedrine group (13.3% vs. 0%; $p<0.05$). There were significant decrease total doses of rescue ephedrine required in the ephedrine group ($p<0.05$). Total doses of used ephedrine in the ephedrine group were significant higher than that of control group. The first rescue ephedrine time in the ephedrine group was significantly longer (14.9 ± 7.1 min vs. 7.9 ± 5.4 min) than that of the control group ($P<0.05$) (Table III).

Table III Comparison of hemodynamic data between two groups (n=60)

Variables	Ephedrine group (n=30) Mean $\pm$ SD	Control group (n=30) Mean $\pm$ SD	p-value
Hypotension	11 (36.7%)	24 (80.0%)	<0.001*
Hypertension	8 (26.7%)	6 (20.0%)	0.542
Tachycardia	16 (53.3%)	14 (46.7%)	0.605
Bradycardia	1 (0.0%)	5 (13.3%)	0.038*
Nausea or vomiting	6 (20.0%)	17(56.7%)	0.003*
Total ephedrine requirement (mg)	18.6 $\pm$ 11.2	39.6 $\pm$ 8.6	<0.001*

The first rescue ephedrine time (min) 14.9 ± 7.1 vs. 7.9 ± 5.4 <0.001*

Values are expressed as frequency (%) and mean $\pm$ SD, p value reached from Chi-square test for qualitative variables and Unpaired t-test for quantitative variables, *significant, ns= not significant

Analysis of neonatal data showed no differences between the study groups. No Apgar scores were below 7 at 1 min or 5 min. Umbilical arterial pH were similar between the study groups ($p>0.05$). There was no pH <7.2 in the both groups (Table IV).

Table IV Comparison of APGAR score and umbilical arterial pH between two group (n=60)

Variable	Ephedrine group (n=30) Mean $\pm$ SD	Control group (n=30) Mean $\pm$ SD	P-value
APGAR score 1 min	7.5 $\pm$ 0.50	7.4 $\pm$ 0.51	0.800 ^{ns}
APGAR score 5 min	8.6 $\pm$ 0.52	8.7 $\pm$ 0.53	0.463 ^{ns}
Umbilical arterial pH	7.34 $\pm$ 0.05	7.32 $\pm$ 0.03	0.093 ^{ns}

Values are expressed as mean $\pm$ SD, p value reached from Unpaired t-test, *significant, ns= not significant

Discussion

This case randomized, double-blinded study conducted in the Department of Anaesthesia, Uttara Adhunik Medical College, Uttara, Dhaka. This is the first report to our knowledge to investigate the effect of intravenous ephedrine given according to maternal weight dose of 0.5 mg/kg after the induction of spinal anesthesia for cesarean section to prevent hypotension related to spinal anesthesia. Our findings demonstrated that prophylactic intravenous ephedrine during spinal anesthesia for cesarean section can prevent hypotension without significant maternal tachycardia or hypertension, and also it increases the first rescue ephedrine time and decreases the ratio of nausea and vomiting. Umbilical arterial pH and Apgar scores were not influenced by hypotension or ephedrine medication.

The incidence of hypotension during spinal anesthesia for cesarean section is reported to be as high as 80%, despite fluid preload, lateral uterine displacement and use of vasopressor agents¹¹. In the anesthesia practice, prevention and management of hypotension related to spinal anesthesia remains a difficult problem and there was no consensus on its optimal management.

Phenylephrine, α_1 -adrenergic agonist whose action would be expected to counteract the decrease in systemic vascular resistance induced by spinal anesthesia¹². Phenylephrine can be used for the prevention and treatment of maternal hypotension¹³⁻¹⁵ but a reduction of fetal oxygenation due to uterine vasoconstriction has been observed in animals¹⁶. It may cause maternal bradycardia^{14,17}. Loughrey et al.¹⁸ compared intravenous bolus of ephedrine and phenyle-

phrine combination with ephedrine alone. They found the combination of ephedrine and phenylephrine given as an in-travenous bolus was not superior regarding to the incidence of hypotension, maternal side effects, or umbilical blood gases when administered as a prophylactic bolus followed by rescue boluses and compared to ephedrine alone.

Ephedrine, an indirectly acting sympathomimetic amine, is probably the vasopressor of choice in obstetric anesthesia. Although ephedrine has mixed α - and β -adrenoreceptor activity, it maintains arterial pressure mainly by increases in cardiac output (CO) and heart rate as a result of its predominant activity on β_1 -adrenoreceptors¹⁹. Variable intravenous infusions of ephedrine appear to be successful^{14,20-22}. Kee et al.¹⁰ investigated the efficacy and optimum dose of intravenous ephedrine for prevention of hypotension during spinal anesthesia for cesarean delivery. They compared the effect of ephedrine 10, 20, or 30 mg intravenous for the prevention of hypotension. They found that a bolus dose of 30 mg intravenous ephedrine was required to reduce the incidence of hypotension during spinal anesthesia for cesarean delivery. They concluded that although the incidence of hypotension was reduced to 35% in the patients who received ephedrine 30 mg compared with the control rate of 95%, this was at the expense of an increased incidence of hypertension, which occurred in 45% of the patients. They suggested that 30-mg intravenous ephedrine may not be suitable in some patients such as those with cardiovascular or cerebrovascular disease. Compared with the study of Kee et al.¹⁰, the incidence of reactive hypertension is lower in our study (45% vs. 28.6%). Duration of ephedrine administration in the study of Kee et al. was 30 sec, however, in our study; it was 60 sec.

Decreased ratio of reactive hypertension in the ephedrine group in our study may result from the longer duration of ephedrine administration. Particularly if sympathetic block level is low,

reactive hypertension may be a problem. In the ephedrine and control groups, upper sensory level was T4 (T3-T5), however, it was T4 (C2-T7) in the study of Kee et al.¹⁰, and the range of sensorial

block was wide compared to our study. Increased sympathetic activity might be related to compensatory stimulation of thoracic sympathetic nerves, including the fibers supplying the heart (T1-T4) in the patients undergoing spinal anesthesia²³. Such event also was reported in low spinal anesthesia and epidural blocks in which sympathetic block does not reach the T4 level²⁴. The ratio of reactive hypertension was similar the patients given intravenous ephedrine and saline (28.6% vs. 19%). In the control group, cause of reactive hypertension may result from the administration of higher doses of rescue ephedrine.

Lee et al.⁹ reviewed available studies to determine the dose-response characteristics of prophylactic intravenous ephedrine for the prevention of hypotension during spinal anesthesia for cesarean delivery. They reported that, significant dose-response relationships were found for hypotension, hypertension and umbilical arterial pH. They suggested that, the use of larger doses of ephedrine (>14 mg) does not completely eliminate hypotension but causes reactive hypertension and a minor decrease in umbilical arterial pH. They found no evidence of a dose-response relationship for nausea or vomiting, fetal acidosis, or Apgar scores. Both ratio of hypotension and nausea and vomiting decreased with ephedrine dose used in this study.

Some studies found significantly higher umbilical arterial pH when using prophylactic ephedrine⁷. Thus, it seems that ephedrine must be used during cesarean section to avoid spinal hypotension, which remains a major determinant of fetal acidemia^{10,25}. Ephedrine has been shown to cross the placenta and to affect the fetal and neonatal heart rate²⁶ due to β -adrenoreceptor activity. A greater proportion of low umbilical artery pH has been observed with ephedrine than phenylephrine¹². Previous studies have shown that the use of ephedrine to prevent or treat hypotension associated with spinal and epidural anesthesia for cesarean delivery may not correct fetal acidosis and may even increase it, especially if hypotension still occurs^{5,22,27}. Kee et al.¹⁰ found that umbilical blood pH values were lower in patients who had hypotension compared with patients who did not, whereas hypertension was not associated with adverse effects. Although they

did not measure uteroplacental flow, their results suggest that, within the range of doses used in their study (10, 20, or 30 mg), the potential vasoconstrictive effects of ephedrine may have a less detrimental effect on uteroplacental blood flow than the effects of hypotension. Eisler et al.²⁸ demonstrated that fetal catecholamine stimulation before delivery might be beneficial. They suggested that when a Q-adrenergic agonist was administered before elective cesarean section, lower respiratory morbidity, and better lung function and reduced risk of hypoglycaemia in the newborn infant were found. In our study, lowest SAP was maintained better in patients who received intravenous ephedrine compared with the control groups. We found no significant difference in neither Apgar scores nor umbilical arterial blood gases data between the study groups, despite a difference in the incidence of hypotension, probably reflecting the early recognition and restoration of hypotension with rescue ephedrine.

Although mean highest HR in the ephedrine group was higher, we found no difference in ratio of tachycardia between the study groups. This could be explained by both the effect of “rescue” ephedrine and baroreceptor-mediated reflex increases in heart rate in patients who became hypotensive. In addition, atropine was applied for bradycardia in the control group.

Conclusion

The above findings suggest, the prophylactic bolus dose of 0.5 mg/kg intravenous ephedrine given at the time of intrathecal block after a crystalloid fluid preload, plus rescue boluses reduce the incidence of hypotension. It has not been shown to eliminate the need to treat maternal hypotension during spinal anesthesia for elective cesarean delivery compared to intravenous rescue boluses alone.

References

1. Ueyama H, He YL, Tanigami H, Mashimo T, Yoshiya I. Effects of crystalloid and colloid preload on blood volume in the parturient undergoing spinal anesthesia for elective Cesarean section. *The Journal of the American Society of Anesthesiologists*. 1999 Dec 1;91(6):1571-78.
2. Kafle SK. Intrathecal meperidine for elective caesarean section: a comparison with lidocaine. *Canadian journal of anaesthesia*. 1993 Aug 1;40(8):718.
3. Rout CC, Rocke DA, Levin J, Gouws E, Reddy D. A reevaluation of the role of crystalloid preload in the prevention of hypotension associated with spinal anesthesia for elective cesarean section. *Anesthesiology* 1993; 79: 262-9.
4. Clark SL, Cotton DB, Pivarnik JM, Lee W, Hankins GD, Benedetti TJ, Phelan JP. Position change and central hemodynamic profile during normal third-trimester pregnancy and post partum. *Am J Obstet Gynecol* 1991; 164: 883-7.
5. Webb AA, Shipton EA. Re-evaluation of i.m. ephedrine as prophylaxis against hypotension associated with spinal anaesthesia for Caesarean section. *Can J Anaesth* 1998; 45: 367-9.
6. Husaini SW, Russell IF. Volume preload: lack of effect in the prevention of spinal-induced hypotension at caesarean section. *Int J Obstet Anesth* 1998; 7: 76-81.
7. Chan WS, Irwin MG, Tong WN, Lam YH. Prevention of hypotension during spinal anaesthesia for caesarean section: ephedrine infusion versus fluid preload. *Anaesthesia* 1997; 52: 908-13.
8. King SW, Rosen MA. Prophylactic ephedrine and hypotension associated with spinal anesthesia for cesarean delivery. *Int J Obstet Anesth* 1998; 7: 18-22.
9. Lee A, Ngan Kee WD, Gin T. A dose-response meta-analysis of prophylactic intravenous ephedrine for the prevention of hypotension during spinal anesthesia for elective cesarean delivery. *Anesth Analg* 2004; 98: 483-90.
10. Ngan Kee WD, Khaw KS, Lee BB, Lau TK, Gin T. A dose-response study of prophylactic intravenous ephedrine for the prevention of hypotension during spinal anesthesia for cesarean delivery. *Anesth Analg* 2000; 90: 1390-5.
11. Rout CC, Rocke DA. Prevention of hypotension following spinal anesthesia for cesarean section. *Int Anesthesiol Clin* 1994; 32: 117-35.

12. Thomas DG, Robson SC, Redfern N, Hughes D, Boys RJ. Random-ized trial of bolus phenylephrine or ephedrine for maintenance of arterial pressure infusion during spinal anaesthesia for Caesarean section. *Br J Anaesth* 1996; 76: 61-5.
13. LaPorta RF, Arthur GR, Datta S. Phenylephrine in treating maternal hypotension due to spinal anaesthesia for caesarean delivery: effects on neonatal catecholamine concentrations, acid base status and Apgar scores. *Acta Anaesthesiol Scand* 1995; 39: 901-5.
14. Hall PA, Bennett A, Wilkes MP, Lewis M. Spinal anaesthesia for caesarean section: comparison of infusions of phenylephrine and ephedrine. *Br J Anaesth* 1994; 73: 471-4.
15. Desalu I, Kushimo OT. Is ephedrine infusion more effective at pre-venting hypotension than traditional prehydration during spinal an-aesthesia for caesarean section in African parturients? *Int J Obstet Anesth* 2005; 14: 294-9.
16. Greiss FC, Crandell DL. Therapy for hypotension induced by spinal anesthesia during pregnancy: observations on gravid ewes. *JAMA* 1965; 191: 793-6.
17. Lee A, Ngan Kee WD, Gin T. A quantitative, systematic review of randomized controlled trials of ephedrine versus phenylephrine for the management of hypotension during spinal anesthesia for cesare-an delivery. *Anesth Analg* 2002; 94: 920-6.
18. Loughrey JP, Yao N, Datta S, Segal S, Pian-Smith M, Tsen LC. He-modynamic effects of spinal anesthesia and simultaneous intravenous bolus of combined phenylephrine and ephedrine versus ephedrine for cesarean delivery. *Int J Obstet Anesth* 2005; 14: 43-7.
19. Critchley LA, Stuart JC, Conway F, Short TG. Hypotension during subarachnoid anaesthesia: haemodynamic effects of ephedrine. *Br J Anaesth* 1995; 74: 373-8.
20. Jackson R, Reid JA, Thorburn J. Volume preloading is not essential to prevent spinal-induced hypotension at caesarean section. *Br J An-aesth* 1995; 75: 262-5.
21. Kang YG, Abouleish E, Caritis S. Prophylactic intravenous ephedrine infusion during spinal anesthesia for cesarean section. *Anesth Analg* 1982; 61: 839-42.
22. Lee A, Ngan Kee WD, Gin T. Prophylactic ephedrine prevents hy-potension during spinal anesthesia for cesarean delivery but does not improve neonatal outcome: a quantitative systematic review. *Can J Anaesth* 2002; 49: 588-99.
23. Owczuk R, Sawicka W, Wujtewicz MA, Kawecka A, Lasek J, Wu-jtewicz M. Influence of spinal anesthesia on corrected QT interval. *Reg Anesth Pain Med* 2005; 30: 548-52.
24. Veering BT, Cousins MJ. Cardiovascular and pulmonary effects of epidural anaesthesia. *Anaesth Intensive Care* 2000; 28: 620-35.
25. Simon L, Provenchere S, de Saint Blanquat L, Boulay G, Hamza J. Dose of prophylactic intravenous ephedrine during spinal anesthe-sia for cesarean section. *J Clin Anesth* 2001; 13: 366-9.
26. Wright RG, Shnider SM, Levinson G, Rolbin SH, Parer JT. The effect of maternal administration of ephedrine on fetal heart rate and variability. *Obstet Gynecol* 1981; 57: 734-8.
27. Shearer VE, Ramin SM, Wallace DH, Dax JS, Gilstrap LC 3rd. Fetal effects of prophylactic ephedrine and maternal hypotension during regional anesthesia for cesarean section. *J Matern Fetal Med* 1996; 5: 79-84.
28. Eisler G, Hjertberg R, Lagercrantz H. Randomised controlled trial of effect of terbutaline before elective caesarean section on postnatal respiration and glucose homeostasis. *Arch Dis Child Fetal Neonatal Ed* 1999; 80: F88-92.
29. Kinsella SM, Carvalho B, Dyer RA, Fernando R, McDonnell N, Mercier FJ, Palanisamy A, Sia AT, Van de Velde M, Vercueil A, Consensus Statement Collaborators. International consensus statement on the management of hypotension with vasopressors during caesarean section under spinal anaesthesia. *Anaesthesia*. 2018 Jan;73(1):71-92.

Anesthetic Management of a Patient with Severe Dilated Cardiomyopathy: Case Report

Md. Shafiul Alam Shaheen¹, A.K.M.Nurnobi Chowdhury², Md. Mushfiqur Rahman³, Md. Mahabubul Hasan⁴, Raju Ahmed⁵, Jashim Uddin⁶

¹Assistant Professor, ²Professor, ³Junior Consultant, ⁴Associate Professor, Department of Anesthesia & Surgical ICU, Ibrahim Medical College & BIRDEM General Hospital. ⁵Assistant Professor, ⁶Registrar, Department of Anesthesiology, Ibrahim Cardiac Hospital & Research Institute (ICHRI)

Corresponding Author: E-mail: drshafiul27@yahoo.com

Abstract

Anesthetic management of patients with dilated cardiomyopathy (DCM) is a challenge to the anesthesiologist, due to poor left systolic function, ventricular enlargement, risk of malignant arrhythmias and sudden cardiac death. Therefore, preoperative assessment and appropriate anesthetic management are important in patients with DCM. Five to eight people per 100,000 develop this disorder each year. Malignant arrhythmias are the most common cause of death in DCM.¹ Around 50% of cases of nonischemic dilated cardiomyopathy is idiopathic. Other causes are familial, infectious, infiltrative and connective tissue diseases. This is a report of successful anesthetic management of a patient with severe DCM undergoing laparoscopic cholecystectomy using general anesthesia (GA).

Keywords: Anaesthetic management, dilated cardiomyopathy, laparoscopic cholecystectomy.

(JBSA 2018; 31(2): 95-98)

Introduction:

Dilated Cardiomyopathy (DCM) is characterized by dilatation and impaired systolic function of one or both ventricles. Cardiomyopathies are diseases of the heart muscle and may present with cardiac dysfunction. DCM is defined by the presence of: (a) Fractional myocardial shortening <25% and/or left ventricular ejection fraction (LVEF) <45%; and (b) LV end diastolic diameter >117% excluding any known cause of myocardial disease.² DCM is the most common type of non-ischemic cardiomyopathy, the third most common cause of heart failure, and the most common indication for cardiac transplantation. World Health Organization reclassification in 1995 was expanded to include all known causes and is based upon anatomical and physiological features. Within this classification, the three main identified types of cardiomyopathy are dilated, hypertrophic, and restrictive. The cause of DCM is unknown, although it may be associated with myocarditis,

neuromuscular disorders, familial disease, idiopathic causes and other possible diseases. Previously, it was thought that the largest proportion of DCM was idiopathic (66%).³ Increasing evidence has shown that DCM has a familial basis.⁴ Over 30 genes have been confirmed to be related to DCM⁵, and sudden cardiac death in DCM was found to be associated with the long arm of chromosome 10. Approximately, 50% of cases of non-ischemic DCM are idiopathic. Here, we report a case of DCM with low EF posted for laparoscopic cholecystectomy surgery under general anesthesia

Case report

A 32 years old female, weighting 52kg having Cholelithiasis is scheduled for laparoscopic cholecystectomy. She was a known case of idiopathic dilated Cardiomyopathy. On preanesthetic examination her heart rate was 68/min and regular. The systolic and diastolic blood pressures were 112mmHg and 60mmHg respectively.

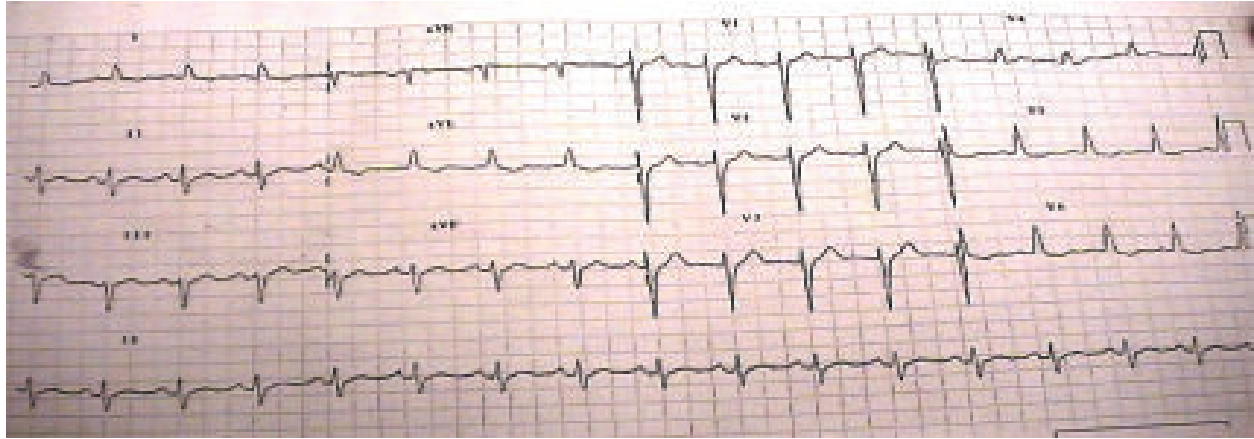


Fig 1 ECG showing left bundle branch block and poor progression of R wave in lead V1 – V5

The respiratory rate was 16/min. There was no ronchi or rales on auscultation and heart sounds were normal. Jugular venous pressure was not raised and there was no hepatomegaly. Preoperative 12 lead electrocardiograph (ECG) [Fig-1] showed Left Bundle Branch Block (LBBB) and poor progression of R wave in leads V1-V5. X-Ray chest [Fig-2] revealed cardiomegaly.



Fig 2 Chest X-ray Showing dilated cardiomyopathy

The lung fields were clear. Echocardiography reports demonstrated global hypokinesia of left ventricle, poor systolic function, ejection fraction of 20%, mitral regurgitation and left ventricular end diastolic dilatation. She was on Losartan 25 mg OD, Spirinolactone 50 mg OD and Digoxin 0.125 mg OD for the last 1 year. All investigations including serum sodium, potassium, calcium and magnesium were within normal limits. A high risk consent was obtained and general anaesthesia was planned. No premedication was advised. On the day of surgery, patient Blood Pressure (BP) was

120/80 mm Hg, Heart Rate (HR) was 72/min and oxygen saturation (SaO₂) was 97% on room air.

Right internal jugular central catheter was placed under local anaesthesia prior to the induction of anaesthesia. Other parameters were monitored with continuous ECG, NIBP, end tidal carbon dioxide and oxygen saturation. Anaesthesia was induced slowly with iv fentanyl 100µgm, iv propofol 50 mg (stop when eyelash reflex diminished) and iv Atracurium 30mg. Lignocaine 50mg was given to blunt the hemodynamic response to intubation. The patient was intubated with cuffed endotracheal tube 7.0 mm ID (Internal Diameter). There was minimal response to intubation, her preintubation BP was 120/80 mmHg and HR was 72/min while post intubation BP was 126/82 mm Hg and HR was 76/min. Anaesthesia was maintained with N₂O : O₂ 50:50 and continuous infusion of propofol @ 15 – 20 ml/hour. Pneumoperitoneum was made by CO₂ insufflation. Intra abdominal pressure (IAP) was maintained 8 – 10 mm Hg. Surgery was completed in 40 minutes. Intraoperatively her mean arterial pressure was 66 -78 mm Hg, heart rate 60-70 / min, SPO₂ was 99-100%, CVP was 8-10 cm H₂O, end tidal carbon dioxide was 32-36 mm Hg and airway pressure was 15-20 cm H₂O but ECG tracing showed sinus rhythm with infrequent PVC (< 4/min) without ischemic changes. Injection paracetamol 1 gm intraoperatively was given for postoperative pain management. At the end of surgery, patient was extubated smoothly and shifted to surgical ICU for better monitoring & management. On the 1st postoperative day patient

was discharged to surgical ward with stable hemodynamics.

Discussion:

Dilated cardiomyopathy is characterized by progressive cardiac dilatation and results in impaired ventricular function. It has a prevalence of 36 per 100, 000 population.⁶ A large number of cases are idiopathic but within these there is a familial association. Clinical picture of dilated cardiomyopathy may vary from only cardiomegaly to severe CHF.⁷ Apart from CHF, dysarrhythmias and embolism are (systemic or pulmonary) also common.⁸ Recent management include medical therapy with drugs for example vasodilator, diuretics or beta blockers and atrio-ventricular pacemakers for patients with incoordinate movements of heart chambers.⁹ It is difficult to decide the optimal time for surgery but the medical control of heart failure for >1 week is desirable.

Anaesthetic management of patients with Cardiomyopathy with reduced systolic function is challenging and may be associated with high mortality.¹⁰ The goals of anaesthetic

management are: (i) avoiding myocardial depression by carefully titrating the anaesthetic drugs; (ii) maintaining normovolaemia; (iii) avoiding overdose of drugs during induction as the circulation time is slow; (iv) avoiding increase in ventricular afterload and (v) avoiding sudden hypotension where regional anaesthesia is the choice. Two key factors exist in the management of patients with Cardiomyopathies. These include: (i) to improve systolic function; and (ii) to prevent sudden death due to ventricular arrhythmias. To improve systolic function, patients should initially be managed medically with administration of diuretics, beta-blockers, angiotensin converting enzyme inhibitors or angiotensin receptor blockers. Our patient was being managed with these drugs. The preoperative preparation of these patients must be meticulous as they have minimal or no cardiac reserve. Preoperatively patients tend to be dehydrated as they would have been on diuretics leading to hypotension during anaesthesia. However, excessive preoperative hydration is not desirable as it may lead to congestive heart failure. As patients may develop ventricular arrhythmias in the perioperative period, antiarrhythmic medications should be

continued. Arrhythmias occur commonly when potassium or magnesium levels are low. These electrolytes should be measured preoperatively and corrected as necessary.

Pneumoperitoneum should be started with reduced gas flow and intra-abdominal pressures should be kept as low as possible to minimize hemodynamic changes. Upto 10 mmHg of IAP, metabolic and haemodynamic parameters do not change significantly. With standard IAP of 12-15 mmHg, there is compression of capacitance vessels as opposed to collapse leading to increased venous return. The pressure threshold during Pneumoperitoneum, associated with minimal changes in the hemodynamics, is 12 mmHg.¹¹

The acceptable limit of decrease in blood pressure and heart rate for a patient depends upon underlying medical condition. It is recommended that fluid therapy and pharmacological management be guided by the use of pulmonary artery catheterization and the determination of cardiac filling pressure.¹² Continuous monitoring of preload by Transesophageal Echocardiography (TEE) and of myocardial performance by cardiac output measurement (CCO) is also useful but this was not available in our hospital so, we relied on central venous pressure.

The optimum anesthetic technique for patients undergoing appendicectomy with dilated or other forms of congestive cardiomyopathy is controversial and both general anesthesia^{13,14} and regional anesthesia have been described.

Brown et al. described the use of general anesthesia because they feared catastrophic effects of reduction in systemic vascular resistance caused by epidural blockade.¹³

Regional anaesthesia used alone or in combination with general anaesthesia has the advantage of reducing after load which can improve cardiac output. However, hypotension must be prevented to avoid myocardial hypoperfusion. Treatment of arterial pressure changes should be considered if a 10% decrease in systolic pressures occurs.

For patients undergoing higher risk procedures under general anaesthesia or those in whom heart failure management is not optimized, invasive direct arterial pressure monitoring is indicated. Central venous pressure monitoring offers some

additional information on right ventricular preload but does not provide information on left heart pressures. Information from a pulmonary artery catheter can be useful, although may not improve outcome. Intraoperative transoesophageal echocardiography may also be useful for examining dynamic changes in cardiac performance and the response to inotropes and fluid loading. Where such technology is unavailable, then the use of oesophageal Doppler monitoring of aortic root velocities can provide information on cardiac performance. Inotropic support if required during and after surgery can be provided by the use of a variety of agents, including dobutamine, dopamine, phosphodiesterase inhibitors, and levosimendan. It may be necessary to counteract some of the peripheral vasodilator effects of general anaesthetic agents to assist coronary perfusion. This is achieved with the cautious use of norepinephrine which increases systemic vascular resistance and maintains mean arterial pressure.

Postoperative regional anaesthesia can be beneficial and good quality pain management avoids increases in systemic vascular resistance and heart rate. Cautious fluid management is important, and is best undertaken in an intensive care area.

Fluid management in patients with DCM is very critical. In our case intra-operative 500 ml ofringer lactate was given to prevent fluid overload. Over hydration may not be advisable as it may lead to CHF. Drop in BP was corrected with injection of ephedrine, a vasopressor which can neutralize the vasodilating effect of the anaesthetics rather infusing intravenous fluids.

Conclusion

In conclusion, a patient of DCM poses many risks for anesthesiologist. Our patient was managed successfully under general anesthesia without any complications by a thorough preoperative assessment, optimized cardiac status, formulating good anesthetic plan & postoperative care.

References

1. Dec GW, Fuster V. Idiopathic Dilated Cardiomyopathy. *N Engl J Med*, 1994; 331: 1564-1575.
2. Wood WL, Kuczkowski KM, Beal BR. Anesthetic considerations for cesarean section in the parturient with familial cardio-

- myopathy. *Acta Anaesthesiol Belg* 2008; 59:87-9.
3. HSU DT, CANTER CE. Dilated cardiomyopathy and heart failure in children. *Heart Fail Clin* 2010; 6:415-432.
4. HERSHBERGER RE, MORALES A, SIEGFRIED JD. Clinical and genetic issues indilated cardiomyopathy: a review for genetics professionals. *Genet Med* 2010; 12: 655-667.
5. TESTER DJ, ACKERMAN MJ. The role of molecular autopsy in unexplained sudden cardiac death. *Curr Opin Cardiol* 2006; 21: 166-172.
6. Dec GW, Fuster V. Idiopathic dilated Cardiomyopathy. *N Engl J Med* 1994; 331: 1564-75.
7. Stevenson LW, Perloff JK .The Dilated cardiomyopathy: clinical aspects. *Cardiol Clin*. 1988; 6:187-218.
8. Stoelting RK, Dierdorf SF. Cardiomyopathy. In: Stoelting RK (Ed). *Anaesthesia and Coexisting Disease* (3rd ed). New York: Churchill Livingstone 1993; 97-102.
9. Molhoek SG, Bax JJ, Erven RV. Effectiveness of resynchronization therapy in patients with end stage heart failure. *Am J Cardiol*. 2002;90:379-83.
10. Tabib A, Chalabreysse L, Barel C, Loire R, Malicier D, Miras A. Sudden death during anaesthesia: human error, drug related or cardiac death? *Therapie* 2001;56:735-8.
11. Koivusalo AM, Pere P, Valjus M, Scheinin T. Laparoscopic cholecystectomy with carbon dioxide pneumoperitoneum is safe even for high risk patients. *Surg Endosc*. 2008;22:61-7
12. Christopher M Bernards: Epidural and Spinal Anaesthesia. In: *Clinical Anaesthesia*, Lippincot Raven. 3rd Edition 1996; 665.
13. Brown G, O'Leary M, Douglas I, Herkes R. Perioperative management of a case of severe peripartum cardiomyopathy. *Anaesth Intensive Care* 1992;20:80-3.
14. Lavies NG, Turner DA. Peripartum cardiomyopathy. A rare cause of pulmonary oedema in late pregnancy. *Anaesthesia* 1989;44:770-2.