IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861. Volume 9, Issue 6 (Sep.- Oct. 2013), PP 01-05
www.iosrjournals.org

A Study of Heart Rate Recovery Following Exercise in Subjects with Normal Pulmonary Function Test

Dr.Jagnyaseni Panda[1], Dr.SnigdhaPrava Mishra[2], Dr.BipinBihari Pradhan[3]
[1]*Tutor, Department of physiology, M.K.C.G Medical College, Berhampur, Odisha.*
[2]*Associate Professor, Department of physiology, M.K.C.G Medical college, Berhampur, Odisha.*
[3]*Professor and Head of the department, Department of physiology, M.K.C.G Medical College,Berhampur, Odisha.*

Abstract:*Exercise is a common physiological stress which has positive chronotropic effect on heart rate and on cessation heart rate returns to pre-exercise level. A delay in heart rate recovery (HRR) (≤12beats in first minute) is considered abnormal and reflects autonomic dysfunction.Pulmonary function tests (PFT) are good indicator of autonomic function.Few studies are available on abnormal HRR (heart rate recovery) in COPDCases. Hence the present study was taken up to find out the presence of abnormal HRR in subjects with normal PFT and to establish HRR as an independent autonomic marker. For the study 150 healthy young adults (both male and female) with normal PFT were subjected to exercise by Bicycle ergometer till targeted Heart Rate (85%Maximum Heart Rate(MHR)) was achieved. HRR at the end of 1 minute followingcessation of exercise were tabulated. In our study 29 subjects (19.33%) with normal PFT show Abnormal HRR indicating HRR could be an independent autonomic marker.keyword-PFT,HRR,MHR.*

I. Introduction:

Exercise is a common physiological stress used to elicit cardiovascular abnormalities not present at rest and to determine the adequacy of cardiac function[1]. Physiologically it has positive chronotropic effect on heart rate due to sympathetic stimulation and parasympathetic (Vagal) withdrawal (Kluessetal 2000)[2]. On cessation of exercise parasympathetic reactivation and withdrawal of sympathetic activity ultimately leads to return of heart rate towards pre exercise level (Arai etal 1989)[3].Heart Rate Recovery (HRR) is defined as the change in heart rate from peak exercise to 1minute following cessation of exercise. Fall in heart rate ≤12beats after first minute following peak exercise is considered abnormal (cole.etal)[4].A delay in HRR reflects autonomic dysfunction. Role of Autonomic nervous system in regulation of cardio respiratory function is well documented. Pulmonary function tests which are good indicators of autonomic function are abnormal in patients with obstructive lung diseases[5].

There are few information regarding study on association of HRR and PFT. More ever the studies available are on subjects with lung pathology like COPD. Previousauthor have found out that patients with COPD demonstrate abnormal HRR (La Rovere MT) [6] 5,7,8,9,10,11). So the present study was taken up with the objective to find out the presence of abnormal HRR following exercise in healthy young adults with normal PFT and in such cases whether HRR by itself can be an independent autonomic marker.

II. Materials and Methods

This prospective study was conducted in the P.G Research Laboratory of Department of physiology, M.K.C.G Medical College Berhampur during the period from 2009-11 after due approval from the institutional ethics committee.

For the study healthy young adults between 17-24 years both male and female were included. From those, we selected 150 volunteers who confirmed to baseline spirometry with normal PFT. Cardiac monitor was connected to each subject and Blood pressure (B.P), Heartrate (H.R) and Oxygen saturation at rest were recorded. They were subjected to exercise by Bicycle Ergometer with digital display till targeted Heart Rate (85%MHR) was achieved or appearance of limiting symptoms like(chest discomfort,shortness of breath, dizziness) whichever was earlier. During exercise continuous recording of heart rate, Blood pressure and oxygen saturation were done by cardiac monitor.

At cessation of exercise therecording of HR, B.P and oxygen saturation were noted. HRR at the end of 1minute after cessation of exercise were tabulated and analyzed.

III. Observation and Analysis

Basal spirometry was done and subjects with normal pulmonary function test (PFT) were included in our study. The data of both dynamic lung function and flow rates are given in table 1 & 2 for male and female.

TABLE 1

OBSERVED AND PREDICTED VALUE OF DYNAMIC LUNG FUNCTION PARAMETERS MALE AND FEMALE.

Parameters	Male(n=106)		Female(n=44)	
	Observed Value (Mean ± SD)	Predicted Value (Mean ± SD)	Observed Value (Mean ± SD)	Predicted Value (Mean ±SD)
FVCex(L)	3.96±0.59	4.41±0.53	2.99±0.43	3.24±0.47
FEVI(L)	3.54±0.49	3.77±0.44	2.77±0.38	2.81±0.41
FEVI/FVC(%)	89.58±6.06	83.31±0.82	93.00±4.73	84±0.92

TABLE 2

OBSERVED AND PREDICTED VALUE OF EXPIRATORY FLOW REATES MALE & FEMALE

Parameters (L/sec)	Male(n=106)		Female(n=44)	
	Observed Value (Mean ±S.D)	Predicted Value (Mean ± S.D)	Observed Value (Mean± S.D)	Predicted Value (Mean ±S.D)
MEF_{25}	2.32±1.02	2.23±0.88	2.18±0.98	2.01±0.81
MEF_{50}	5.30±1.21	4.97±0.50	4.47±1.21	4.11±0.52
MEF_{75}	7.77±1.45	7.5±0.90	5.88±1.26	5.75±0.78
MEF_{25-75}	4.56±1.08	4.64±0.49	3.96±1.02	3.77±0.54
PEF	8.44±1.37	8.81±1.12	6.21±1.27	6.48±0.95

TABLE 3

ANTHROPOMETRIC PARAMETERS OF THE STUDY GROUP

Parameters	Male(n=106) Male (Mean ±S.D)	Female(n=44) Female (Mean ±S.D)
Age(yrs)	19.00±1.24	21.00±10.78
Height (mtr)	1.66±0.07	1.55±0.066
Weight(Kg)	62.6±12.22	56.00±12.14
B.M.I(kg/m^2)	22.75±4.04	23.50±5.35

Study includes subjects with normal B.M.I both male and female.

TABLE 4

PRE EXERCISE PARAMETERS

Parameters	Male(n=106) Male (Mean ±S.D)	Female(n=44) Female (Mean±S.D)
Heart Rate (beats/min)	77±3.86	78±5.79
Blood Pressure(mmHg)		
Systolic	119±6.34	114±6.23
Diastolic	81±3.86	79±4.35
Oxygen Saturation (%)	98±1.25	98±1.03

Mean heart rate, Blood Pressure both systolic, diastolic &oxygen saturation are within normal range in both male and female.

TABLE 5

EXERCISE PARAMETERS (Male)n=106

Parameters	Pre-Exercise (Mean±S.D)	End of Exercise (Mean ±S.D)	1 min after cessation of exercise (Mean ±S.D)
Heart Rate (Beats/Min)	77±6.34	163±9.13	140±13.58
Blood Pressure(mmHg)			
Systolic	119±6.34	142±10.54	136±8.63
Diastolic	81±3.86	89±6.32	87±5.15
Oxygen Saturation (%)	98±1.25	98±1.14	99±0.81

After exercise mean heart rate increased to 163 ± 9.13 and came down to 140 ± 13.58, 1 minute following cessation of exercise.

TABLE 6

EXERCISE PARAMETERS OF SUBJECTS WITH ABNORMAL HEART RATE RECOVERY (Male) n=23

Parameters	Pre-Exercise (Mean ±S.D)	End of Exercise (Mean ± S.D)	1 min after cessation of exercise(Mean ± S.D)
Heart Rate (Beats/Min)	78±5.75	164±6.46	155±5.79
Blood Pressure(mmHg)			
Systolic	119±8.15	144±10.45	136±9.43
Diastolic	82±4.05	92±4.80	89±3.11
Oxygen Saturation (%)	98±1.56	98±0.79	98±0.65

23 male subjects showed abnormal HRR following 1 minute after cessation of exercise, the mean heart rate came down to 155±5.79 following 1 minute after cessation of exercise from 164±6.46 at end of exercise.

TABLE 7

EXERCISE PARAMETERS (Female) n=44

Parameters	Pre-Exercise (Mean ±S.D)	End of Exercise (Mean ± S.D)	1 min after cessation of exercise(Mean ± S.D)
Heart Rate (Beats/Min)	76±5.79	161±8.19	137±13.25
Blood Pressure(mmHg)			
Systolic	114±6.23	136±7.20	130±7.53
Diastolic	79±4.35	89±5.49	87±6.51
Oxygen Saturation (%)	98±1.03	98±1.01	99±0.58

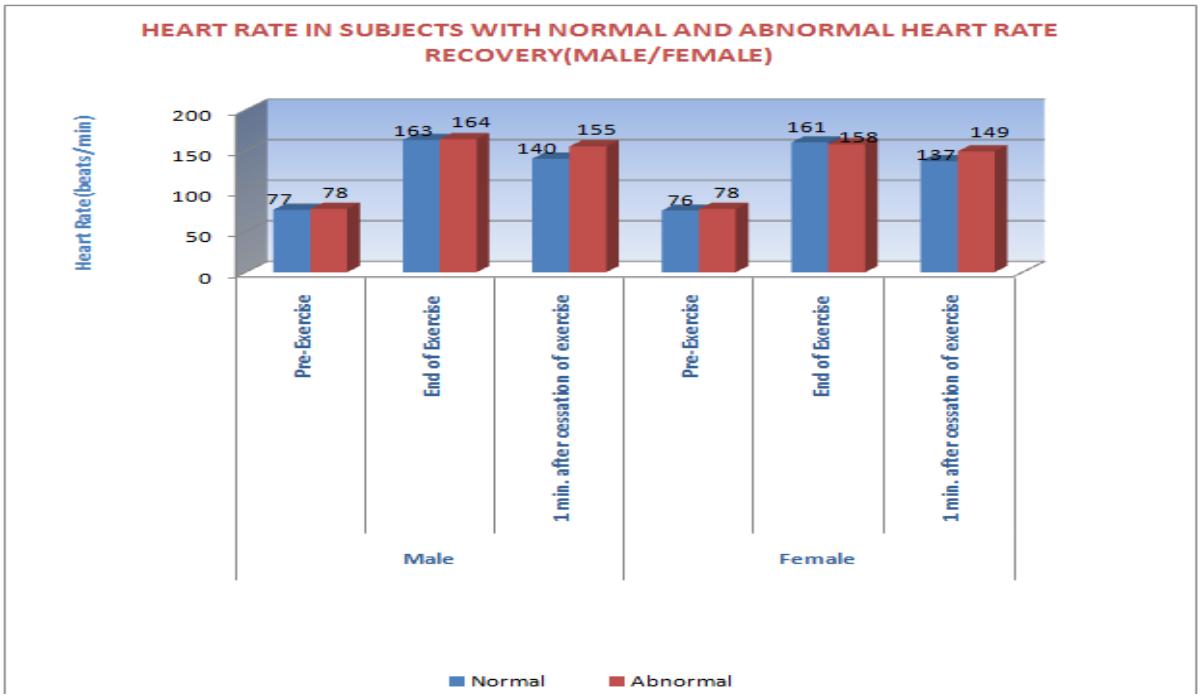
The mean heart rate in female was 76±5.79 in pre-exercise state and increased to 161±8.19 following exercise. The HRR was normal with 137±13.25 following 1 min after cessation of exercise.

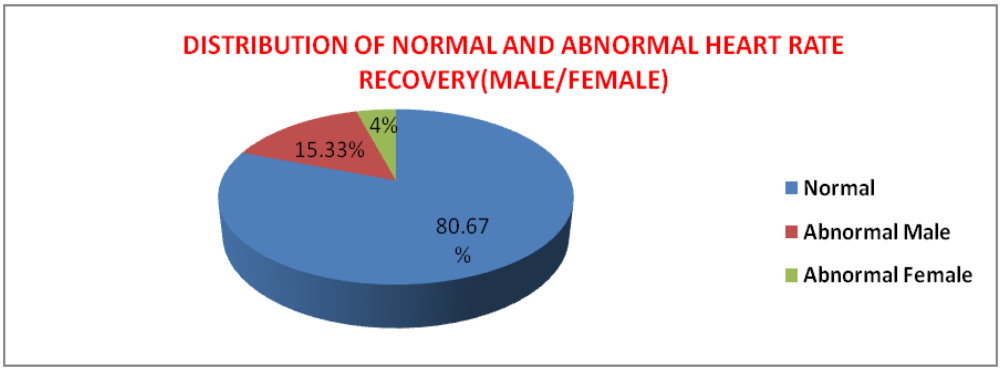
TABLE8

EXERCISE PARAMETERS OF SUBJECTS WITH ABNORMAL HEART RATE RECOVERY (Female) n=6

Parameters	Pre-Exercise (Mean ± S.D)	End of Exercise (Mean ± S.D)	1 min after cessation of exercise (Mean ± S.D)
Heart Rate (Beats/Min)	78±4.50	158±12.66	149±11.29
Blood Pressure(mmHg)			
Systolic	118±5.66	146±6.05	141±8.38
Diastolic	79±6.20	93±4.43	89±0.98
Oxygen Saturation (%)	98±1.89	99±0.49	99±0.49

Out of 44 females only 6 showed abnormal HRR with heart rate 149±11.29 following 1 min after cessation of exercise from 158±12.66 at the end of exercise.





IV. Discussion

Out of the total 150 healthy subjects of our study, only 29 showed abnormal HRR[4] .All of themhad normal PFT. Heart Rate (HR) during dynamic exercise is regulated by a combination of neural, hormonal and intrinsic mechanism. At the onset of exercise the rise in heart rate is thought to be mediated by withdrawal of inhibitory vagal tone. Central command from higher brain centre and input from mechanoreceptors in muscle contribute to this early response.[12, 13] At higher heart rate, increase sympathetic out flow to the heart, increased level of circulatory catecholamine and temperature of pacemaker tissue also play a role [14, 15, 16]. Immediately after exercise acceleratory influence from higher brain centre and peripheral nerve reflexes diminish and heart rate is thought to be primarily regulated by restoration of vagal inhibitory tone[17-20]

HRR following exercise correlates with vagal tone and a decrease of HRR in the first minute of the exercise is associated with increased mortality[7-11]. Our study therefore is suggestive of possibility that the HRR following exercise is highly sensitive enough to reflect the autonomic dysfunction due to decreased vagal tone. So, it can be an independent autonomic marker, which is easy to obtain in any clinical set up. Few studies have found out that delay in HRR following exercise, which may be a influence of decrease vagal activity is a powerful predictor of overall mortality, independent of work load, the presence or absence of myocardial perfusion defects [21]. This marker is simple to calculate from data that are already contained in the results of standard exercise tests and may be valuable for the assessment of risk in routine clinical practice.

V. Summary and Conclusion:

Our study consisting 150 healthy individuals of either sex, within age group 17-24 years, was carried out to assess the presence of abnormal HRR following dynamic exercise in subjects with pulmonary functions. Also we aimed at using HRR as an independent autonomic marker. From our study we found out that, 19.33% of total subjects with normal lung function showed abnormal HRR. Therefore we can conclude that although both pulmonary functions and HRR reflect automatic dysfunction, HRR could be an independent variable as a prognostic marker.

References:

[1]. Hurst's The Heart ValentinFuster, Richard A Walshs , Robert ,A.Harrington ,Thirteenth Edition, McGraw Hill Medical, Vol.1, P.371.
[2]. KlussH.A.Wood.R.H. andWelsch.M.A(2000) Vagal modulation of the heart and central hemodynamics during handgrip exercise.American journal of physiology 279,H1648-H1652.
[3]. Arai.Y.Saul.J.P.Albrecht,P.Hartley,L.H,Lilly,L.s,Cohen, R.J. and Colucci,W.S(1989). Modulation of Cardic autonomic activity during and immediately after exercise. American journal of physiology256,H132-H141.
[4]. Cole CR, Blackstone EH, PashkowFJ,et al. Heart-rate recovery immediately after exercise as a predictor of mortality .N Engl J Med 1999,341:1351-1357.
[5]. NiranjanSeshadri,ThomasR,Gildea,Kevin McCarthy, RCPT, Claire Pothier, Mani S .Kavure and Michael S.Lauer.Association of an abnormal exercise Heart Rate Recovery with Pulmonary Function Abnormalites. Chest/125/4/April,2004.
[6]. La RovereMT,BiggerJT,MarcusFI,et al. Baroreflex sensitivity and Heart Rate variability in prediction of total cardiac mortality after myocardial infraction . Laneet 1998;351:478-484.
[7]. Stewart AG,Waterhosue JC, HowordP.TheQTc interval, autonomic neuropathyand mortality in hypoxemic COPD,Respir Med,1995;89:79-84.
[8]. Stewart AG,Marsh F, Waterhosue JC et al. Autonomic nerve dysfunction in COPD as assed by the acetylcholine sweat spot test .EurRespir J ,1994;7:1090-1095.
[9]. Stewart AG,Waterhosue JC, HowordP.Cardiovascular autonomic nerve function in patients with hypoxemic chronic obstructive pulmonary disease. EurRespir J,1991;4:1207-1214.
[10]. SteinPK,NelsonP,Rottman JN et al.Heart rate variability reflects severity of COPD in Piz Alpha-antitryspindeficiency.Chest 1998;113,327-333.
[11]. Volterrani M,ScalvineS,Mazzuero G, et al. Deceased heart rate variability in patients with chronic obstructive pulmonary disease,Chest,1994;106:1432-1437.
[12]. MacielBC,L.Gallo,M.Neto,EC Lima Filho,and LB Martin .Autonomic nervous control of the heart rate during dynamic exercise in normal man. Clin.Sc.71:457:460,1986.

[13]. Mitchell JH, WC Redon and DI McCloskey. Reflex effect on circulation and respiration for contracting skeletal muscle.Am.J.Physiol,233(Heart Circ.Physiol 2):H374-H378,1977.
[14]. Lamb DL. The physiology of aerobic endurance.In: Physilogy of exrcise(2nd e3d),New York,Macmillan,1984,P.137-143.
[15]. Robinson BF,SE Epstin,GDBeiser and E.Braunwald. Control of Heart rate by the autonomic nerves system .Circ.Res.19:400-411,1966.
[16]. VonEuler,US.Sympatho-adrenal activity in physical exercise.Med.Sci.Sports,6:165-173,1973.
[17]. Blomqvist CG and B. Saltin. Cardiovascular adaptations to physicicaltraining.Annu Rev.Physiol.45:169-189,1983.
[18]. Savin WM,DM Davidson and Wl Haskell. Autonomic contribution to heart rate recovery from exercise in humans.J.Appl.Physiol,53:1572-1576,1982.
[19]. Savin WM,WM Haskell, JS Schroeder and EB Stinson. Cardiorespiratory responses of cardiac transplanting patients to graded ,symptom limited exercise.Circulation 64:55-60,1980.
[20]. Scheuer J. and CM Tipton. Cardiovascular adaptations to physical Training.Annu Rev Physiol,39:221-251,1976.
[21]. Christopher R,ColeMD,Eugene H, Black Stone MD,FredricJ,Pshkow MD, Claire E,Snader MA and Michael Laucher MD. Heart rate recovery immediately after exercise as a predictor of mortality,Vol.341:1351-1357,October,28,1999.

IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861. Volume 9, Issue 6 (Sep.- Oct. 2013), PP 06-07
www.iosrjournals.org

Morphometry of the Nasolabial Complex in Adult Indian Population

Ankit Srivastav, B.V. Murlimanju, Latha V. Prabhu, K.U. Prashanth, K.M. Sukritha

Department of Anatomy, Manipal University, Centre for Basic Sciences, Kasturba Medical College, Bejai Campus, Mangalore - 575004, India.

I. Introduction

Anthropometry of lip-nose complex has been extensively studied for European population.[1-6] However, corresponding studies in case of Indian population are very scarce.[3-9] Hence anthropometric study of lip-nose complex was undertaken. This study involves comparison with available data from literature. The objective was to study the morphometry of the nasolabial complex in Indian population.

II. Methods

The procedure of measuring the lip-nose complex was explained to each individual resulting in the accurate measurements due to lack of anxiety on the part of individual. The morphometry was done on 100 adults (age range: 18-45 yrs). Among them 44 were males and 56 were females. The parameters like vertical lip height, cupids bow width, total width of the mouth, columellar height, columellar width, nasal width and dome height were measured. The measurements were repeatedly taken for each individual to ensure the accuracy. The points of lip-nose complex were marked using marking pen (Fig. 1) and the linear measurements were taken using digital vernier caliper. The vertical height of the lip was from the columellar base to cupids bow peak. This parameter was chosen against base of columella to tubercle since it has significance in cleft lip surgery. The cupids bow width was measured from philtrum peak to peak. The total width of mouth was the commissure to commissure distance.

III. Results

The measurements were statistically analysed (arithmetic mean and standard deviation were calculated) and tabulated (Table 1). The results were compared with available data for Caucasions, blacks and Chinese (Table 2).

IV. Discussion

The present study establishes the basal values for various parameters of lip-nose complex of the Indian population. Since standard deviations for Caucasions and blacks being not available only mean values are compared. The present study reveals that vertical lip height, cupids bow width, total width of the mouth and columellar height were more amongst males compared to females. The same pattern is seen with Caucasions and blacks. Columellar height is more in male as well as in female adult Caucasions compared to Indians and blacks. Columellar height is lowest amongst Chinese compared to other races.

The Indian males as well as females have lowest columellar width compared to Caucasions and blacks, which again indicates a change in growth pattern. There is a resemblance in columellar width between Chinese and Indians. Nasal width is more amongst Indian males compared to Indian females. Nasal width is maximum amongst blacks compared to Indians and Caucasions. Indian males have larger values for columellar height and columellar width. Vertical height of the lip is more amongst Indian males compared to females. Adult Caucasions have more vertical height of the lip compared to Indians and blacks and Indian males and females have the lowest vertical height of the lip.

Cupid's bow width is same amongst adult InIndian males have wide oral commissure compared to females at all age groups. Oral commissure is smallest amongst Indian males and females at all age groups compared to Caucasions and blacks. Adult male and female blacks have wide oral commissure compared to Caucasions and blacks. Dome height is more in Indian adult males compared to females. The data was not available for Caucasions and blacks for comparison. Indian males and females differ significantly in certain parameters from that of Caucasions and blacks but shows resemblance to Chinese. For years together the anthropometric measurements for surgical reconstructions are based on basic values for western population

resulting in the time of surgical repair being based on western growth patterns, which actually differs for Indians. This has not been given a serious thought. Our study has been conducted to generate Indian data.

References

[1]. Farkas LG, Lindsay, Cleft Craft, 1976;1:36-8.
[2]. Farkas LG, Cheung GCK. Nostril asymmetry: Microform of cleft lip palate? An anthropometrical study of healthy North American Caucasians. Cleft Palate J 1979;16 :351.
[3]. Shah M, Verma IC, Mahadevan S, Puri RK. Facial anthropometry in Newborns in Pondicherry. Indian J Paediatrics 1991;25:259-63.
[4]. Farkas LG, Posnick JC, Hreczko, T.M., and Pron, G.E. Growth patterns of the nasolabial region : A morphophetric study. Cleft Palate Craniofac J 1992;29:318.
[5]. Farkas LG, Hajnis K, Posnick JC. Anthropometric and anthroposcopic findings of the nasal and facial region in cleft patients before and after primary lip and palate repair. Cleft Palate Craniofac J 1993;30:1.
[6]. Farkas LG. Anthropometry of the Head and Face, 2nd Ed. New York : Raven Press 1994.
[7]. Farkas LG, Tompson B, Philips J, Katic M, Cornfoot L. Comparison of Anthropometric and Cephalometic Measurements of the adult Face. Cleft Palate Craniofac 1999;5:10-8.
[8]. Mulliken JB, Burvin R, Leslie G, Farkas LG, Repair of bilateral complete cleft lip: Intraoperative Nasolabial Anthropometry, Plastic and Reconstructive Surgery 2001;107:307-14.
[9]. Prasad NN, Reddy D. Anthropometry: Lip-Nose Complex. Indian Journal of Plastic Surgery 2001;34:3-8.

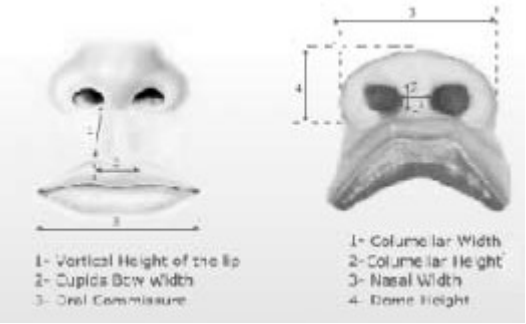


Fig. 1 Diagram showing the details of measurements of the present study

Table 1 Comparison of nasolabial morphometry among males (n=44) and females (n=56)

values in cm	male	female
vertical lip height*	12.9 ± 2.2	10.5 ± 2.2
cupids bow width*	11.5 ± 2.1	10 ± 1.5
total width of the mouth*	52 ± 7.1	48.7 ± 6.9
columellar height*	10.9 ± 2.5	8.8 ± 2.4
columellar width	8.1 ± 1	8.1 ± 1.1
nasal width	32.1 ± 3.1	31.6 ± 3.7
dome height	18 ± 6.8	18.2 ± 3

values are mean ± SD, statistical significance (independent t-test) *p<0.05

Table 2 Showing the racial variability of the parameters among various populations

	Present study (Indian)		West Indian		Chinese		Caucasions		Blacks	
values in cm	M	F	M	F	M	F	M	F	M	F
vertical lip height*	12.9	10.5	16.2	14.2	16	13	22	15	21	21
cupids bow width*	11.5	10	11.7	11.8	12	11	15	13	12	13
total width of the mouth*	52	48.7	53.5	47	56	53	63	57	72	57
columellar height*	10.9	8.8	9.8	8.6	6	5	13	11	10	9
columellar width	8.1	8.1	5.7	5.7	6	7	8	6	9	7
nasal width	32.1	31.6	32.3	30.5	43	39	36	33	44	45
dome height	18	18.2	20.4	16.9	-	-	-	-	-	-

IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861. Volume 9, Issue 6 (Sep.- Oct. 2013), PP 08-12
www.iosrjournals.org

Introduction of Problem Based learning as an innovative T-L method in Physiology at Dr Shankarrao Chavan Govt Medical College, Nanded

Dr.Mungal Shreechakradhar U.[1], Dr. Santoshkumar A. Dope[2], Dr. Sushil P. Dube[3], Dr. N. D. Somwanshi[4], Dr. S.S.Karadkhedkar[5], Dr. M. B. Kulkarni[6].

[1] *Assistant Professor,* [3] *Assistant Professor ,* [4] *Professor,* [5] *Assistant Professor,* [6] *Assistant Professor, Department of Physiology, Dr.Shankarrao Chavan Govt. Medical College, Nanded. Maharashtra, India.*

[2] *Assistant Professor, Department of Anatomy, Government Medical College, Latur. Maharashtra, India.*

Abstract : Many *organizations across the world have adopted Problem Based learning (PBL) as a small group teaching – learning (T-L) tool. PBL inculcates self directed learning in students. If PBL is included in the curriculum of basic sciences like Physiology, it can help students to apply knowledge of Physiology in clinical practice. So this study is undertaken as a pilot project to introduce PBL as an innovative T-L method in curriculum of Physiology at Dr. Shankarrao Chavan Government Medical College, Nanded.*

In this study one PBL session was conducted on cardiovascular Physiology in Dept of Physiology at Dr. S.C. Govt. Medical College Nanded, India. An MCQ test was conducted before and after PBL session. Students' perspectives on PBL were studied by taking feedback from the students.

Result: *Students commented that PBL made the teaching-learning more interesting. Students opined that PBL enabled them for better understanding of the subject and motivated them for self directed learning and to read more. Students also commented that PBL enabled them to remember the subject better and helped to integrate their knowledge. Most of the students expressed a desire and a need to continue PBL sessions in future also.*

Keywords: *PBL – Problem based learning, Teaching – Learning (T-L) method, Physiology curriculum*

I. INTRODUCTION

Teaching learning is a continuously changing process. Over the years, various studies convinced that traditional discipline based curriculum in medical education is dehumanizing and demotivating. It is also convinced that students learn better when actively involved in their learning tasks and basic science would be better understood, remembered and subsequently applied if learned in a clinically relevant format [1]. Nurturing these needs, in recent years, Problem based Learning is being widely used in Medical Education all over the world [2, 3].

With the observation of a gap between the qualitative and quantitative advancement in medical education and achievements in the field of health care prompted the Medical Council of India to adopt a need based curriculum for undergraduate medical education in India. "Regulations on Graduate Medical Education, 1997" recommend a teaching approach characterized by maximal efforts to encourage integrated teaching between traditional subject areas using a problem based learning approach and de-emphasize compartmentalization of disciplines so as to achieve both horizontal and vertical integration in different phases[4].

Many organizations across the world have adopted Problem Based learning (PBL) as a small group teaching – learning (T-L) tool. PBL inculcates self directed learning in students .Also it develops problem solving attitude and analytical skills in students. If PBL is included in the curriculum of basic sciences like Physiology, it can help students to apply knowledge of Physiology in clinical practice. Also students will get early orientation of clinical education.

So this study is undertaken as a pilot project to introduce PBL as an innovative T-L method in curriculum of Physiology at Dr. Shankarrao Chavan Government Medical College, Nanded.

II. AIMS AND OBJECTIVES

2.1 Aims

The aim of this study is to develop self directed learning, problem solving attitude and analytical skills in the students and also to increase competency of forthcoming health professionals.

2.2 Objectives

2.1.1 By the end of six months, students should develop self directed learning.

2.1. 2 By the end of six months, faculties should get sensitized and develop interest to use PBL as

teaching learning method.

2.1.3 To study perspectives of students and faculties regarding PBL as teaching learning method.

III. Material And Method

Present study was carried over a period of four months in the Department of Physiology, Dr. Shankarrao Chavan Government medical College, Nanded. Participants for this study were Ist MBBS students of 2012 batch of Dr. S. C. Govt. Medical College, Nanded, India. First approval of ethical committee was taken. Students were randomly distributed in 10 groups.

Faculties and students were sensitized for procedure of PBL by an interactive seminar on pattern of PBL. A Problem (Annexure I) based on applied aspect of cardiovascular Physiology was constructed by consensus of all faculties. Specific learning objectives were also set by consensus of all faculties. A MCQ test (Annexure II) was taken to assess students' knowledge on the topic.

A Problem (Annexure I) based on applied aspect of cardiovascular Physiology was posed to groups. In all groups there was one leader, one time keeper, one recorder, one reporter and a teacher as a facilitator. Groups discussed and extracted learning areas for study from the problem. Then student went home and studied different learning areas, they have identified during discussion. After 3-4 days the groups of students sat together and discussed facts they studied about the problem. They analyzed if there was any learning area which still required more study. Again they went home and read about learning areas newly identified and again discussed after 3-4 days. They repeated the procedure till they satisfied about solution of the problem.

The same MCQ test, which was taken before PBL session, was repeated after PBL session to assess the knowledge gained. Feedback was taken from the students by providing pretested and validated questionnaires (Annexure III). Feedback included constructed response with open ended questions and selected responses with Likert scale and overall Global Rating Scale.

IV. Results

48 students participated and gave the feedback.

Data obtained was analyzed using SPSS 21.00 Version

Paired T Test was applied for comparing pre PBL and post PBL test scores of students

Table 1 Comparison between Pre PBL score and Post PBL score

Paired t test analysis for comparison between Pre PBL score and Post PBL score					
	Mean	N	Std. Deviation	Std. Error Mean	P value
Pre PBL test score	5.1667	48	0.8337	0.1203	< 0.0001
Post PBL test score	8.1875	48	0.5322	0.07682	

(P value < 0.001 – highly significant)

There is significant difference in scores of students before and after PBL session.

Table 2 Students perspectives on different aspects of PBL

	Item	No. of students said Don't agree	No. of students said Somewhat agree	No. of students said agree	No. of students said Strongly agree	No. of students said Very strongly agree
1	In understanding a particular topic PBL was very useful	5 (10.41%)	3 (6.25%)	26 (54.17%)	12 (25%)	2 (4.17%)
2	In PBL sessions, a valuable exchange of ideas took place in group discussions	2 (4.17%)	2 (4.17%)	20 (41.67%)	21 (43.75%)	3 (6.25%)
3	By virtue of PBL sessions, clinical conditions could be better related to basic mechanisms	4 (8.33%)	3 (6.25%)	14 (29.17%)	23 (47.92%)	4 (8.33%)
4	PBL sessions will help you in preparing you for the final university examination	6 (12.5%)	5 (10.42%)	21 (43.75%)	14 (29.17%)	2 (4.17%)
5	It is better to replace tutorial classes with PBL	2 (4.17%)	2(4.17%)	27 (56.25%)	13 (27.08%)	4 (8.33%)
6	PBL helps to develop self directed learning	9 (18.75%)	4 (8.33%)	16 (33.33%)	13 (27.08%)	6 (12.5%)
7	PBL helps to develop analytical skills in students	8 (16.67%)	5 (10.42%)	18 (37.5%)	14 (29.17%)	3 (6.25%)
8	All didactic lectures should be replaced by PBL	21 (43.75%)	10 (20.83%)	12(25%)	3 (6.25%)	2 (4.16%)
9	PBL increases your involvement in teaching learning process	2 (4.16%)	2 (4.16%)	32 (66.67%)	9 (18.75%)	3 (6.25%)

Eighty three percent participants opined that they better understood the topic with PBL. Ninety two percent of participant opined a valuable exchange of ideas took place in group discussions. Eighty five percent participants opined clinical conditions could be better related to basic mechanisms. Seventy seven percent of participants reported that PBL sessions will help them in preparing you for the final university examination. Huge agreement i.e. 92% was reported by participants for replacing tutorial classes with PBL. Seventy three percent of participants reported that PBL developed self directed learning and analytical skills in them. Ninety two percent of the participants reported that PBL increased their involvement in teaching learning process. Surprisingly there was disagreement on replacing all lectures by PBL, only thirty five percent of participant thought it is better to replace all lectures by PBL. Response of the participants on Global Rating Scale showed maximum score from 7-9 towards high side. Participants reported no disadvantage of the new method.

In the comments received from students they reported that in PBL students got fair chance to discuss about their each and every queries with peers as well with faculties which made their understanding of the topic better. Some of the students also reported that while explaining a concept to others they found a flaw in their own understanding and it was corrected there and then only. Some of the students opined that PBL can not replace lectures as all the basic mechanism in Physiology can't be covered in PBL. Instead they suggested there should be a short lecture either at the start of the PBL session or as concluding component of PBL session. Some students also pointed out that there is a need of formal training of facilitators for PBL as some of the facilitators were excellent in their job but some facilitators were job dominating group discussions instead of facilitation.

V. Discussion

There has been always a question in whether a problem based form of teaching could be conducted in basic science departments. Faculty members have expressed doubts about the wisdom, effectiveness, and educational efficacy of such a format to teaching the sciences basic to medicine [5]

Experiments by medical teachers have shown that it is possible to introduce a problem-based form of learning into a new course in parallel with more traditional modes of teaching, making it successful to some extent for students who are used to a didactic form of curriculum [6]. Our study revealed that posing problems of clinical situation to students make the inquiry driven learning more interesting. Students commented in their feedback that PBL enhanced their understanding of Physiology and motivated them to read more. Students also commented that PBL enabled them to remember the subject better and helped to integrate their knowledge. All these outcomes are may be due to active involvement of students and peer to peer teaching and motivation in teaching learning process in PBL. Most of the students expressed a desire and a need to continue PBL sessions in future also. On contrary they don't want to replace all the lectures by PBL as they think all the basic mechanisms cannot be covered in PBL.

Apparent benefits of PBL include development of self directed learning, problem solving attitude, and analytical skills. PBL forms a crucial part in the initiation of students into medicine.

VI. Conclusion

In view of excellent rating from students for PBL, it should be continued in the curriculum of Physiology incorporating students' suggestions. An objective analysis of effectiveness of PBL can be made by comparing students' performances with and without PBL.

References

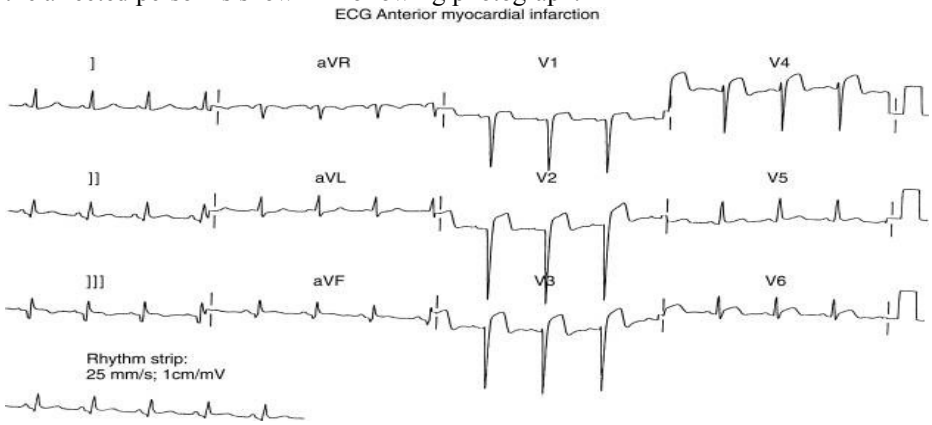
[1]. Laeora Berkson. Problem based learning: Have the expectations been met? Academic Medicine, Volume 68, Number 10, October Supplement 1993, 579-588.
[2]. Albanese, M. A., and S. Mitchell. Problem-based learning: a review of literature on its outcomes and mplementation issues. Acad. Med. , 68(1)), January 1993, 52–81,.
[3]. Barrows, H. S. Problem-based learning in medicine and beyond: a brief overview. New Dir. Teach. Learn, 68, 1996, 3–12.
[4]. Medical Council of India: Salient features of Regulations on Graduate Medical Education 1997 [http://www.mciindia.org/know/rules/rules_mbbs.htm]. accessed Oct 08, 2007
[5]. Schmidt HG. Problem based learning: rationale and description. Med Educ, 17, 1983, 11–16.
[6]. 6. Morrison JM, Murray TS. An experiment in problem based learning. Med Educ, 28, 1994 139–145.

ANNEXURE 1

A 57 year old man presented to the Accident and Emergency Unit of his local district general hospital complaining of 2 h of central 'crushing' chest pain radiating to the neck and shoulders. This had begun shortly after dinner and was more severe than any pain he had previously experienced. His wife explained that for the last 4 months he had been having episodes of 'indigestion' at work which were only partially relieved by self-administered antacid preparations. They had first rung their General Practitioner but he had advised them to call for an ambulance urgently. The past medical history included a diagnosis of hypertension 8 years earlier. At the

time of admission his only relevant drug history was nifedipine 20 mg twice daily. The family history included the sudden death of his father aged 55 years (cause unknown). It was also known that his mother was hypertensive, his elder brother had angina .He was married with two sons and worked as a bus driver. He was a regular smoker (20 per day) and consumed approximately 16 units of alcohol per week. On examination he looked grey and unwell, and was sweating. The blood pressure was 138/94 mmHg, pulse 96 regular and the jugular venous pressure was not elevated. The apex beat was undisplaced but thrusting in character and the heart sounds were normal. The peripheries were cold to the touch but there was no ankle oedema. The foot pulses on the left were absent and there was a femoral bruit on the left. He was mildly tachypnoeic but the chest was clear on auscultation. Discuss the use of ECG for the diagnosis of underlying disease:

ECG of the affected person is shown in following photograph:



ANNESURE II

1- In the fundamental rules of the ECG all the following are right EXCEPT:

a) It is a biphasic record of myocardial action potential potential fluctuations.
b) Deflection record occurs only during complete depolarization or repolarization.
c) +ve wave occurs when depolarizing current approaches the +ve terminal electrode of the voltmeter.
d) -ve wave happens when repolarizing current approaches the +ve terminal electrode.

2. Regarding the causes of the ECG waves all the following are correct EXCEPT:

a) P wave is caused by atrial depolariazation.
b) QRS complex is caused by ventricular depolarization.
c) T wave is caused by ventricular repolarization.
d) U wave by papillary muscle depolarization.

3. The causes of the ECG "intervals":

a) PR by AV nodal conduction.
b) ST by atrial repolarization.
c) QT by ventricular depolarization and ventricular repolarization.
d) All of the above.

4. In the ECG:

a) ST segment is part of depolarization & coincides with plateau.
b) T wave has the same voltage of QRS.
c) QRS & T wave are in the same direction.
d) QRS & T wave are in opposite directions.

5. The voltage of the ECG:

a) Is normal summation of QRS complexes in standard limb leads.
b) Is normally >1.5 mV.
c) Is low in individual having body mass index (BMI) 35.
d) All of the above.

6. Regarding the flow of electrical currents around the heart all the followings are correct EXCEPT:

a) From left to right in the mid portion of the ventricular septum.
b) From the base to the apex during almost depolarization.
c) From the epicardium to the subendocardium during repolarization.
d) From the epicardium to the subendocardium during depolarization.

7. Axis of the heart is:

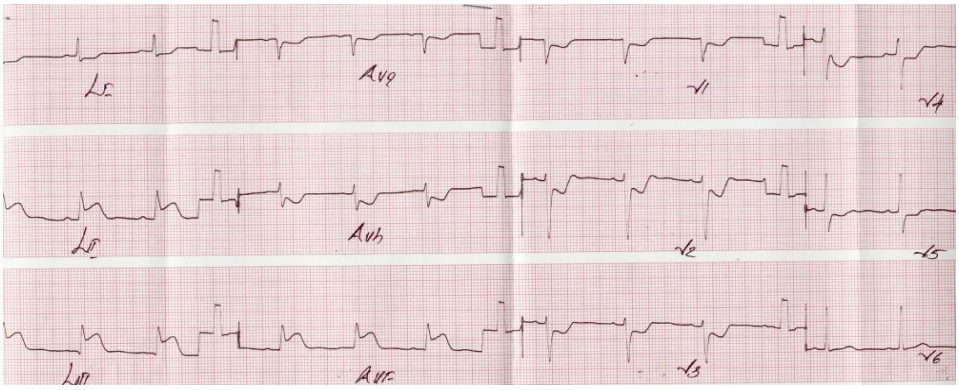
a) Mean instantaneous vector of QRS complexes in vertical plane.

b) Normally from-30 to +60.
c) Deviated to left side in full term pregnant woman.
d) Deviated to right side in long slender person (e.g., +80)

8. The 'J' point on ECG is

a) End of Q wave and beginning of R wave
b) End of P wave and beginning of P–R interval
c) End of S wave and beginning of ST segment
d) End of PR interval and beginning of R wave

9.



The ECG in above figure is suggestive of:

a) Acute anterior wall myocardial infarction
b) Old inferior wall infarction
c) Hyperacute inferior wall infarction
d) Posterior wall infarction

10 . Right axis deviation is seen in

a) Right bundle branch block
b) Left bundle branch block
c) AV nodal block
d) Block in inter atrial pathway

ANNEXURE III

	Item	Don't agree	Somewhat agree	agree	Strongly agree	Very strongly agree
1	In understanding a particular topic PBLwas very useful					
2	In PBL sessions, a valuable exchange of ideas took place in group discussions					
3	By virtue of PBL sessions, clinical conditions could be better related to basic mechanisms					
4	PBL sessions will help you in preparing you for the final university examination					
5	It is better to replace tutorial classes with PBL					
6	PBL helps to develop self directed learning					
7	PBL helps to develop analytical skills in students					
8	All didactic lectures should be replaced by PBL					
9	PBL increases your involvement in teaching learning process					

0=don't agree, 1= Somewhat agree, 2=Agree, 3=strongly agree, Very strongly agree

Q.1 What aspects of PBL contributed most to your learning?
Q.2 What aspects of PBL should be changed to make it better for you?
Q. 3 Have the skills learned during PBL made a difference in your other academic or social situations ? If yes How?
Q.4 General comment on PBL session.

IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861. Volume 9, Issue 6 (Sep.- Oct. 2013), PP 13-16
www.iosrjournals.org

Survival of Dynamic Hip Screw in Unstable Intertrochanteric Fractures

ThudukuchiRamanathanAshok[1], GanesanGanesanRam[2],
Balasugumar Thamodharan[3], Suresh Perumal[4]
[1,2,3,4]*(Department of Orthopaedics,Sri RamachandraUniversity,Chennai-116,Tamilnadu,INDIA)*

Abstract: *51 patients with unstable intertrochanteric fracture treated with sliding hip screw alone were selected in our retrospective and prospective study for a period of 28 months with an average follow up of minimum 4 months.Lateral femoral wall integrity was assessed in all patients radiologically prior and after surgery and tip apex distance was calculated following DHS fixation as described by Baumgartner et al. 5 out of 51 patients had screw cutout within six months of surgery. In our study, unacceptable TAD combined with loss of lateral femoral wall integrity is a definite indicator of DHS implant cutout. Lateral femoral wall fracture resulted in six times higher risk of a reoperation due to technical failure when gold standard method of sliding hip screw was used. Tip Apex Distance alone was not a reliable indicator for screw cut out.The simple treatment guideline should be if the lateral femoral wall or greater trochanter is fractured, the use of DHS implant must be guarded.*

Keywords:*Intertrochanteric fracture, lateralfemoralwallintegrity(LFW), zones in femoral head,tipapexdistance(TAD)***.**

I. Introduction :

Intertrochanteric fracture is one of the most common factures of the hip especially in the elderly with porotic bones, usually due to low-energy trauma like simple falls.Problems of these fractures are (1) association with substantial morbidity and mortality (2) malunion (3) implant failure, cutout of screw head, and penetration into hip. (4) great financial burden to the family and (5) associated medical problem like diabetes, hypertension.
It is universally agreed that the treatment of intertrochanteric fractures is stable internal fixation at the earliest opportunity. Stable fixation is the keystone for successful union of trochanteric fractures. Factors beyond the control of surgeon for successful treatment are: (i) fracture geometry and stability, (ii) bone quality, (iii) comminution. Factors under the control of surgeon are: (i) good reduction, (ii) proper choice of implant, (iii) proper surgical technique, and (iv) availability of modern operation rooms, entire set of implants, instrumentation and image intensifier.The factors most significant for instability and fixation failure are: (i) loss of posteromedial support, (ii) severe comminution, (iii) subtrochanteric extension of the fracture, (iv) reverse oblique fracture. (v) shattered lateral wall (vi) extension into femoral neck area and (vii) poor bone quality. Osteoporosis is particularly important in the fixation of proximal femoral fractures.The mechanism of failure has been the collapse of the neck-shaft angle into varus leading to cut out of the screw from the femoral head. There are various factors which results in the screw cut out, such as age of the patient, quality of the bone, pattern of the fracture, stability of reduction, angle of the implant and position of the lag screw. But there has been no clear consensus to the interrelationships or the relative importance of each factor. Most of the authors have recognized the importance of accurate placement of screw in the femoral head. There have been various methods to evaluate the position of the screw. We have used the method formulated by Baumgartner et al (JBJS Am,1995,77:1058-1064)- Tip apex distance and Lateral Femoral Wall Integrity by Palm et al JBJS (Am) 2007; 89 : 470-475.

II. AIM :

To analyze lateral femoral wall integrity and tip apex distance in unstable intertrochanteric fracture with DHS fixation an important predictor of screw cut out for reoperation.

III. Materials And Methods :

Our study was conducted at Sri Ramachandra Medical College and Hospital from June 2010 to October 2012,it was prospective and retrospective study.We included all unstable intertrochanteric fractures fixed with DHS alone and excluded stable intertrochanteric fracture and intertrochanteric fractures treated with all other modalities. The major factors contributing to the fracture in our study group was person age, landing on the hip, inadequate reflexes and osteoporosis. AO/OTA classification was used to classify fracture pattern in all selected patient. 51 patients were selected totally among them male34(66.67%),female17(33.33%) and nature of injury

was trivial fall-50(98%) and RTA1(2%). Majority of our patients are in the age group of 45-83yrs, with an average age of 64.67 yrs.According to AO/OTA classification fracture pattern of A2.1-22(43.13%),A2.2-18(35.29%)A2.3-5(9.8%),A3.1-5(9.8%),A3.2-1(1.96%).Tip apex distance of Good limits<25mm-1(1.96%), acceptable limits (26-30mm)-11(21.56%), poor limit (31-35mm)-19(37.25%) and unacceptable limits>35mm-20(39.21%). In our study screws were most frequently placed in center-center zone (49%) and least frequently posterior-inferior (1.96%).Lateral femoral wall integrity preoperatively present-45(88.23%),lost-6(11.76%) and postoperatively present-22(43.13%),lost-29(56.86%).

FIGURES AND TABLES :

Table 1.Age distribution

Age (In years)	No. of patients
21-30	-
31-40	-
41-50	3 (5.88%)
51-60	19 (37.25%)
61-70	17 (33.33%)
71-80	8 (15.68%)
81-90	4 (7.84%)

Table 2.Gender distribution

Male	Female
34 (66.77%)	17 (33.33%)

Table3.Mode of injury

Trivial Fall	RTA
50(98%)	1(2%)

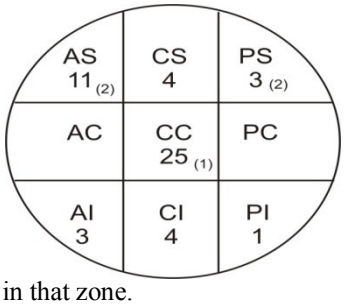
Table 4.AO/OTA classification for intertrochanteric fracture

AO/OTA- 31	NOS.
A 2.1	22 (43.13%)
A 2.2	18 (35.29%)
A 2.3	5 (9.8%)
A 3.1	5 (9.8%)
A 3.2	1 (1.96%)
A 3.3	-

Table 5.Tip apex distance (TAD)

TAD	Nos.
Good (<25mm)	1 (1.96%)
Acceptable(26-30mm)	11 (21.56%)
Poor (31-35mm)	19 (37.25%)
Unacceptable(>35mm)	20 (39.21%)

Fig 1.Zones of Screw placement in femoral head



(No) – Numbers indicate screw cut out in that zone.

Fig 2.Lateral femoral wall integrity (LFW)

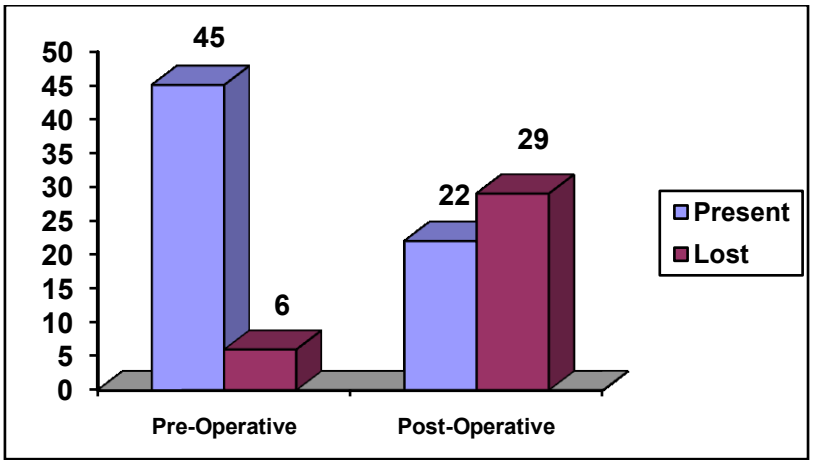


Table 6.Post opeative– Lateral femoral wall (LFW) integrity loss

AO/OTA	Nos.
A 2.1	9 (40.90%)
A 2.2	10 (45.45%)
A 2.3	4 (18.18%)

IV. Results :

1)In our study, 5 out 51 patients had screw cutout which occurred within six months after the surgery.2) In the study,sliding screw were most frequently placed in center-center zone(49%) and least frequently in posterior-inferior(1.96%) zone. 3)The highest rate of screw cutout occurred in anterior-superior(two of five screws) and posterior-superior(two of five screws),the rate of cutout in these two peripheral zones was significantly higher than rate in other zones. However, the placement of screws in any of the other zones had no predictive significance with respect to cut out.4) In five patients who had screw cutout TAD averaged 35.04mm (range 30.4 to 38.8mm) compared with 34.91mm (range,24 to 46.5mm) in those who had no screw cutout.5) In our study lateral femoral wall was lost preoperatively (fracture pattern) in 6 patients (11.76%) and intact in 45 patients (88.23%), postoperatively loss of lateral femoral wall integrity was seen in 22 patients (48.88%) we found that there was seven times higher risk of losing lateral wall integrity due to a technical failure when the gold standard method of sliding compression hip-screw fixation was used.

V. Discussion :

In our study we had used the AO/OTA classification and noticed that forty five fractures (88.23%) of the 51 were type 31-A2 and six (12.77%) were of type A31-3. In our study lateral femoral wall integrity (LFW) was lost preoperatively (fracture pattern) in 6 patients (11.76%) and intact in 45 patients (88.23%), postoperatively loss of lateral femoral wall integrity was seen in 22 patients (48.88%).we found that there was seven times higher risk of losing lateral wall integrity due to a technical failure when the gold standard method of sliding compression hip-screw fixation was used. Twenty three patients out of twenty nine fractures (79.31%) that were identified postoperatively, known to have occurred during the surgery itself had been classified as type A2.1 were 9 fractures (39.1%), type A2.2 were 10 fractures (43.42%) and type A2.3 were 4 fractures (17.39%). In our study the tip apex distance (TAD) averaged 34.92mm (range, 24 to 46.50) for all 51 fractures. The screw cut out through the femoral head was noticed in 5 of the 51 patients. All the screw cut out occurred within 6 months after the surgery.In the 5 patients who had screw cut out TAD averaged 35.04mm (range, 30.4 to 38.8mm) compared with 34.91mm (range, 24 to 46.5mm) in those from which the screw had not cut-out. Forty five patients out of fifty one had sustained type A2.1, A2.2, A2.3 fracture and 80% (4 of the 5) of type A2.3 had an intraoperative fracture of the lateral wall compared with 40% (9 of the 22) of type A2.1. In our study screws were most frequently placed in center-center zone (49%) and least frequently posterior-inferior (1.96%). The highest rate of cutout occurred in the anterior –superior (two of five screw) and posterior- superior (two of five screw zones). The rate of cut out in these two peripheral zones was significantly higher than the rate in the center-centerzone.However, the placement of screws in any of the other four zones – that is, placement of 23.52% of all screws – had no predictive significance with respect to cut out.In our study one patient with type 31- A2.1 fracture and TAD 30.4mm with lag screw in the center-center zone had screw cut out as the lateral femoral wall integrity was lost postoperatively. The other three patients in whom the screw had cutout were type 31A2.1,A2.2 fractures, but the lateral femoral wall integrity was lost postoperatively and the TAD was in the range of poor and unacceptable limits (33.2-38.6mm).

VI. Conclusion:

1)In our study, unacceptable TAD combined with loss of lateral femoral wall integrity is a definite indicator of DHS implant cutout.
2)Lateral femoral wall fracture resulted in six times higher risk of a reoperation due to technical failure when gold standard method of sliding hip screw was used.
3)DHS has to be better avoided in AO/OTA 31A2.2 and 31A2.3 intertrochantric fractures as the incidence of post operative loss of femoral wall integrity was statistically significant.
4)Tip Apex Distance alone was not a reliable indicator for screw cut out.
5)The simple treatment guideline should be if the lateral femoral wall or greater trochanter is fractured, the use of sliding hip screw must be guarded.

References :

[1]. Integrity of the Lateral Femoral Wall in Intertrochanteric Hip Fractures : An Important Predictor of a Reoperation. Henrik Palm. JBJS Am. 2007; 89 : 470-475.
[2]. The value of the tip-apex distance in predicting failure of fixation of peritrochanteric fractures of the hip. MR Baumgaertner. JBJS Am. 1995; 77 : 1058-1064.
[3]. Prediction of Fixation failure after sliding hip screw fixation. Pervez et al. Vowler S. Injury 2004 Oct.; 35(10) : 994-998.
[4]. Bannister, G.C., Gibson, A.G., Ackroyd C.E., Newman, J.H., The fixation and prognosis of trochanteric fractures. A randomized prospective controlled trial.Clin.Orthop. 254:242-246, 1990.
[5]. Bridle, S.H., Patel, A.D.; Bircher M., and Calvert P.T.: Fixation of intertrochanteric fractures of femur. A randomized prospective comparison of the Gamma nail and dynamic hip screw. J.B.J.S. 73-B(2):330-334,1991.
[6]. Clawson, D.K., Trochanteric fractures treated by sliding screw plate fixation method. J. Trauma, 4:737-752, 1964.
[7]. Davis, T.R.C.; Sher, J.L.,; Horsman, A.; Simpson, M., Porter, B.B.; and Checketts, R.G.: Intertrochanteric femoral fractures. Mechanical failure after internal fixation J.B.J.S., 72-B(1) : 26-31, 1989.
[8]. Doherty, J.H., and Lyden, J.P., Intertrochanteric fractures of hip treated with the hip compression screw. Analysis of problems.ClinOrthop., 141:184-187,1979.
[9]. Kaufer, H. : Mechanics of treatment of hip injuries. Clin.Orthop.146 : 53-61, 1980.
[10]. Kyle R.F.; Gustilo, R.B., and Premer R.F. : Analysis of six hundred and twenty two intertrochanteric fractures. A retrospective and prospective study. J.B.J.S. 61-A:216-221, March 1979.
[11]. Laros, G.S. and Moore, J.F., Complication of fixation in intertrochanteric fractures. Clin.Orthop, 101:110-119,1974.
[12]. Larsson, S. Friberg S; and Hanson L : Trochanteric fractures. Influence of reduction and implant position on impaction and complication. Clin.Orthop.259 :130-139,1990.
[13]. Mainds, C.C. and Newman R.J. : Implant failures in patients with fractures of proximal femur and treated with sliding screw device. Injury, 20 : 98-109, 1989.
[14]. Mulholland R.C. and Gunn, D.R. Sliding Screw plate fixation of intertrochanteric femoral fractures. J. Trauma, 12 : 581-591, 1972.
[15]. Parker, M.J. : Cutting out of dynamic hip screw related to its position. J.B.J.S., 74 – B(4) : 625, 1992.
[16]. Schumpelick W.; and Jantzen P.M. : A new principle in the treatment of trochanteric fractures of femur. J.B.J.s. 37-A : 693-698, July 1955.
[17]. Thomas, A.P., : Dynamic hips screws that fail, Injury, 22 : 45-46, 1991.
[18]. Whitelaw, G.P.; Segal, D; Sanzone C.F; Ober, N.S. and Hadley N : Unstable intertrochanteric / subtrochanteric fractures of the femur. Clin.Orthop.252 :238-245, 1990.
[19]. Wolfang, G.L., Bryant, M.H., and O'Neil J.P. : Treatment of intertrochanteric fractures of femur using sliding screw plate fixation. Clin.Orthop.163 : 148-158,1992.
[20]. Dimon, J.H., Hughston J.C., Unstable trochanteric fractures of the hip. J.B.J.S. 49A, 400-450, 1967.
[21]. Sarmiento A. : The unstable intertrochanteric : Treatment with a valgus osteotomy & 1 Nail plate. J.B.J.S. 52A (10) : 1309-1318,1970.
[22]. Current concept review – Intertrochantric fractures.GS Kulkarni, Rajiv limaye, Miliandkulkarni, Sunil kulkarni. Indian journal of Orthopaedics, January 2006;Vol 40: No.1: p 16 – 23.

IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861. Volume 9, Issue 6 (Sep.- Oct. 2013), PP 17-21
www.iosrjournals.org

An Experimental Study of Biochemical and Histopathological Study on Gentamycin Induced Renal Failure in Albino Rat And The Effectiveness Of Punarnava (BOERHAEVIA Diffusa) On Reversal Of Renal Damage

M.Pramila Padmini[1], J. Vijay Kumar[2]
[1] *(Assistant Professor of Anatomy, MIMS Medical College Vizianagaram, Nellimarla, India)* [2]*Professor of Anatomy, Saveetha Medical College, Chennai, India)*

Abstract: *Drug-induced nephrotoxicity is an important cause of renal failure. Aminoglycosides throughout the endocytic pathway are taken up into the epithelial cells of the renal proximal tubules and stay there for* a long time, which leads to nephrotoxicity. Wistar- albino male rats weighing 125–150gms, are utilized for *the present study.Blood samples were collected with cardiac puncture for biochemical investigations like blood urea, uric acid, creatinine, serum Na, K, Ca, determination.By using one way ANOVA the results are significant at .001. Hyaline cast formation is observed in PCT with atrophic glomeruli effecting half of the cortical region when rats treated with 80mg/kg b.w. administration of Punarnava 400mg and 800mg/kg.bw rejuvenated necrotic cells of kidney .Gentamicin must be given in the lowest effective therapeutic doses in patients with normal kidney function along with punarnava.*

Key words: *gentamycin, glomeruli, lymphatic infiltration, proximal convoluted tubules, punarnava*

I. Introduction

Drug-induced nephrotoxicity is an important cause of renal failure. Aminoglycosides throughout the endocytic pathway are taken up into the epithelial cells of the renal proximal tubules and stay there for a long time, which leads to nephrotoxicity. Hydroxyl radicals play a role in the pathogenesis of gentamicin nephrotoxicity, gentamicin can induce suppression of Na(+)-K(+)-ATPase activity and DNA synthesis in rats proximal tubules leading to renal injury; this injury may be relevant to reactive oxygen metabolites generated by gentamicin. Renal cortical mitochondria is the source of reactive oxygen metabolites, which induces renal injury (Nephrol Dial Transplant. 1994;9 Suppl 4:135-40[1]).

Pharmacological studies have demonstrated that *B. diffusa* exhibits a wide range of properties such as diuretic [Gaitonde BB, et al 1974[2]; nephrotic syndrome [Singh RH et al 1972[3]; antiurolithiatic [Pareta SK et al 2011[4]; antioxidant and antidiabetic activity . Due to the combination of diuretic, antioxidants and anti-inflammatory activities B. diffusa is regarded as therapeutically highly efficacious for the treatment of inflammatory renal diseases and common clinical problems such as nephrotic syndrome, oedema, and ascites. Boerhavia Diffusa has been reported to be useful in the treatment of elephantiasis, night blindness, corneal ulcers and nephritic syndrome(Mishra J, et al 1980[5] , Singh RH, et al 1972[3]).

The protection offered by the B.diffusa aqueous extract could have been due to the presence of any of the active principles contained in it. Literature has shown B. diffusa contains a large number of compounds such as flavonoids, alkaloids, steroids, triterpenoids, lipids, lignins, carbohydrates, proteins, and glycoproteins . Flavonoids and other antioxidant constituent of medicinal plants have been reported to inhibit xenobiotic induced nephrotoxicity in experimental animal models due to their potent anti-oxidant effects [Devipriya S et al 1999[6]. Very few studies of histopathology were reported in literature with the effectiveness of punarnava (Boerhaevia diffusa) on gentamicin induced renal failure in Albino rats and to estimate the damage and revival of renal tissue. So, the present study is taken up to record the anti-nephrotoxic effects of punarnava.

II. Materials And Methods

Wistar - albino male rats weighing 125–150 g, are utilized for the present study. Experiments were performed with the permission of the institutional ethics committee.

In the present study, male albino rats were used and are grouped as follows:

*group I- 6 albino rats with normal saline for 10 days and are sacrificed on the 11th day.

*(group-II- 6 albino rats with 400mg/kg. b.wt of punarnava(pure extract obtained in capsules from himalaya product-each capsules containing 250mg of extract of pure punarnava powder). for 10 days and are sacrificed on the 11th day.

*group-III-10 albinorats with 80mg/kg. b.wt of gentamycin for 10 days. On 11th day 6 animals are sacrificed and remaining 4 are left without giving any treatment to see whether there is self regeneration.
*group-IV-10 albino rats with 80mg/kg. b.wt of gentamycin for 10 days and from 11th day treated with punarnava 400mg/kg. b.wt for 3weeks by the end of third wk two rats were sacrificed to see the regenerative changes. By the end of six weeks another two rats were sacrificed to see the changes.by the end of 9 wks all the 6 rats were sacrificed to see the changes.
*group-V- 10 albino rats with 80mg/kg. b.wt of gentamycin for 10 days and from 11th day treated with with punarnava 800mg/kg. b.wt for 3weeks by the end of 3rd wk two rats were sacrificed to see the regenerative changes. by the end of 6wks another two rats were sacrificed to see the changes. By the end of 9wks all the 6 rats were sacrificed to see the changes.

- All animals were fed standard rat chow and were provided tap water to drink *ad libitum*. All animals were weighed before the injections. The animals were anaesthetized with ether inhalation.
- Blood samples were collected from retro-orbital plexus for biochemical investigations like blood urea, uric acid, creatinine, serum Na, K, Ca, determination. Bilateral periumbilical vertical incisions were made. Right and left kidneys were removed quickly and weighed and preserved in 10% formalin.
- **Histopathological examination:**

Anterior half of Kidneys from all three groups were fixed in 10% neutral buffered formalin and processed to paraffin wax. 5 microns Sections are stained with Haematoxylin and Eosin , Massons trichrome, and Periodic Acid Schiff and are examined under light microscope at 100 and 400 magnification.

III. Results

Table (1): Comparative Nephrotoxic effects of gentamicin and nephroprotective effects of different doses of Boerhavia diffusa (Punarnava) on some biochemical parameters in rats.

	Group-I CONTROL-SALINE	Group-II 400mg B.diff/kg.b.w	Group-III 80mg gen./kg.b.w	Group-IV 80mg gen./kg.b.w for 10days and 400mg/kg.b.wt punarnava from 11th day for 9 weeks	Group-V 80mg gen./kg.b.w for 10days and 800mg/kg.b.wt punarnava from 11th day for 9 weeks
PARAMETERS	MEAN+SD	MEAN+SD	MEAN+SD	MEAN+SD	MEAN+SD
žUrea (mg/dl)	16.8+0.43	16.76+0.45	74.3+1.46	38.23+4.45	22.16+2.15
žCreatinine (mg/dl)	0.46+0.02	0.46+0.29	2.14+0.21	1.21+0.23	0.67+0.07
žUric acid (mg/dl)	0.36+0.05	0.37+0.04	1.05+0.11	0.68+0.07	0.51+0.03
žNa (meq/L)	125.53+1.44	124.62+2.26	117.97+1.93	119.6+1.56	121.9+1.72
žK (meq/L)	4.06+0.08	4.11+0.11	5.58+0.43	5.06+0.21	4.5+0.22
žTotal calcium (mg/dl)	9.21+0.45	9+0.25	7.7+0.50	8.07+0.26	8.66+0.46

Mean , Standard deviation and one way ANOVA was done to know the significance. All The Values Are Significant Between And Within Groups At .001

- Values are expressed as means ± S.D. By using one way ANOVA the results are significant .

Group-III –1 rat out of 4 rats which are not treated after inducing gentamycin toxicity for 10 days , died on the fourth day and remaining 3 rats were sacrificed on the eight day. Mean and S.D are similar to that of group-III and are excluded from the table.

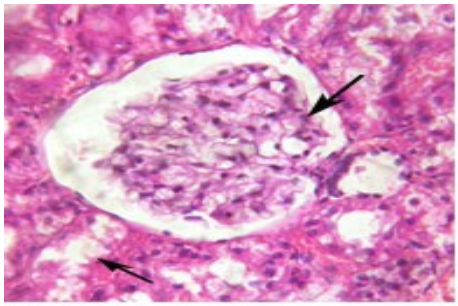
3.1 Histopathological Observations

3.1.1 Group-I –6 rats treated with normal saline(0.9% Nacl)

Male albino rats with intake of normal saline showed normal architecture of renal glomeruli with intact bowmans capsule. Brush bordered cuboidal epithelium lining the proximal convoluted tubules. Simple cuboidal epithelium lining the distal convoluted tubules .Macula densa is very prominent(fig.1).

3.1.2 Group-II- rats treated with punarnava 400mg/kg b.w

The cytoarchitecture of kidneys showed normal structure of glomeruli, distinct proximal and distal convoluted tubules(fig.2).



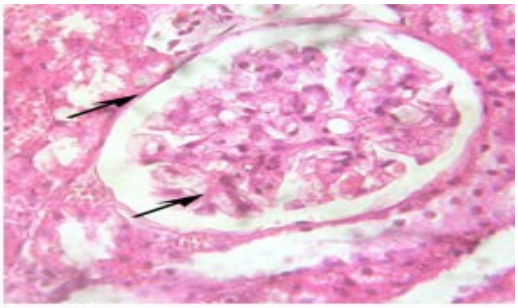


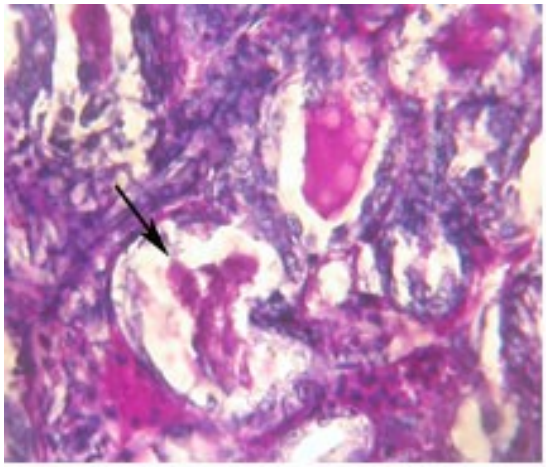
fig.1 **fig.2**

fig.1 showing normal glomeruli and PCT with brush borded cuboidal epthelium in G-I rats

fig.2 showing normal glomeruli and continuous Boowman's capsule in G-II rats

3.1.3 Group-III –rats induced with gentamycin 80mg/kg b.w

The use of the periodic acid-Schiff reaction confirmed that these apoptotic cells were almost exclusively found in proximal tubules causing obstruction of PCT'S. Tubular basement membrane is interrupted. Glomerular congestion, disruption of glomerular capillaries,vacuolar degeneration of tubular epithelial cells is observed with hyaline cast formation is observed in PCT. Atrophic glomeruli are present effecting half of the cortical region(fig.3). Lymphocytic infiltration has increased(fig.4) . Rats induced with gentamycin 80mg/kg b.w for 10 days and not treated showed atrophic glomeruli and hyaline casts in PCT'S. There is no self regeneration.



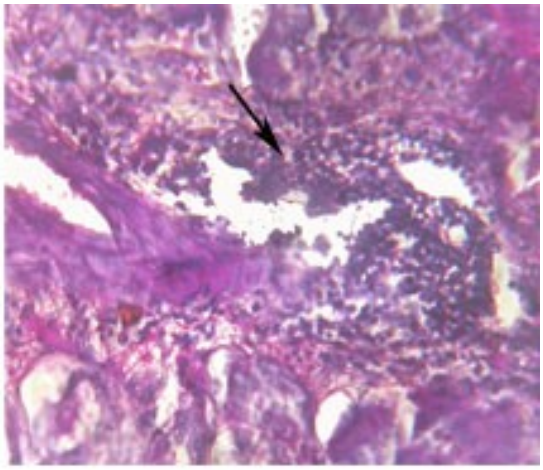


fig.3 **fig.4**

fig.3 showing atrophic glomeruli effecting half of the cortical region in G-III rats, 10x40, PAS.

fig.4 showing peritubular lymphocytic infiltration in G-III rats in G-III rats, 10x40, PAS.

3.1.4 Group-IV- rats induced with 80mg/kg.bw gentamycin and treated with 400mg/kg.bw of punarnava

After 3wks of treatment , the renal microscopic details did not show any regeneration in kidneys of two rats. By the end of 6 wks , the PCT'S are lined with cuboidal epithelium and are clear without any hyaline cast(fig.5) and regeneration of luminal epithelium. By the end of 9wks , the renal architecture showed 50% improvement in regeneration of glomerular tuft and distinct epithelium formation of proximal convoluted tubules(fig.6).

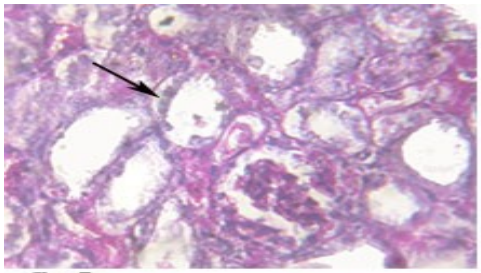
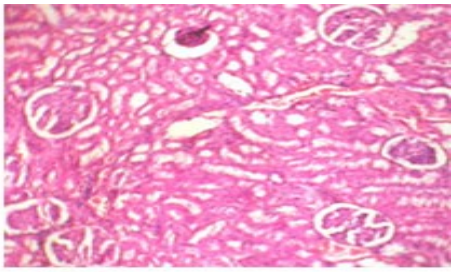



fig.5 **fig.6**

fig.5 showing PCT'S without any hyaline cast, 10x40, PAS.
fig.6 showing 50% improvement in regeneration of glomerular tuft , 10X10, H&E

3.1.5 Group-V- rats induced with 80mg/kg.bw gentamycin and treated with 800mg/kg.bw of punarnava

By the end of 3wks the lymphatic infiltration in the proximal convoluted tubules disappeared indicating anti-nephrotoxic effect of punarnava. By the end of 6wks , hyaline cast disappeared and by the end of 9wks, 80% of glomeruli have regained their normal structureand enclosed ny continuous bowman's capsule(fig.7) showing continuity in the Bowman's membrane(fig.8).

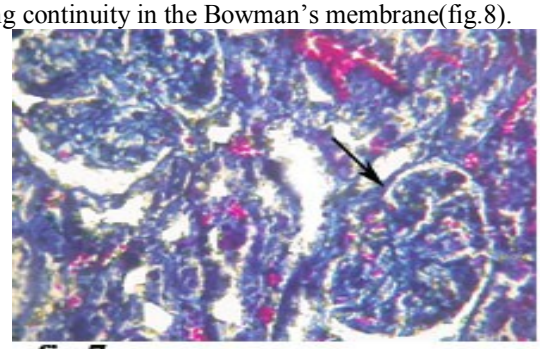
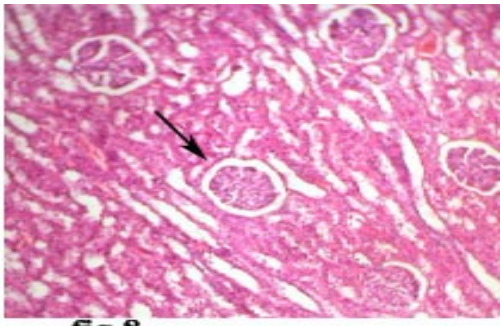



fig.7 **fig.8**

fig.7 showing glomeruli having normal structure, 10x40, Massons Trichrome
fig.8 showing continuity in the Bowman's membrane, 10x10, H&E.

IV. Discussion

Acute renal failure is characterized by disorders in some biochemical parameters in gentamicin treated rats .Rats treated with 80mg/kg b.w. Gentamicin produced increase in the concentration of serum urea, creatinine and uric acid.These results confirmed that gentamicin produced nephrotoxicity as previously reported by Ali *et al., 2003[7], Goto, 2004[8] and Heibashy et al.,*2009[9]. More than half of proximal tubules showing desquamation of necrosis but involved tubules easily found, complete or almost complete tubular necrosis. Serum electrolytes were disturbed in GM treated rats as compared with control animals. Lower value of serum sodium indicated inability of kidney to conserve sodium and chloride(Heibashy & Abdel Moneim (1999)[10] and Heibashy *et al. (2009[9]).* Gentamicin treated rats show tubular eptithelial damage with intense granular degeneration involving > 50% of renal cortex. Some of the tubular epithelium contains tubular casts as observed by K.VIJAY KUMAR et al 2000[11]) . Gentamicin renal cell damage as induced by tubular necrosis ie, marked congestion of the glomeruli with glomerular atrophy, degeneration of tubular epithelial cells with casts in the tubular lumen and infiltration of inflammatory cells in the interstitium was confirmed on histopathological examination by Shirwaikar A et al 2003[12]. In the present study shrunken glomeruli and glomerular atrophy is observed by gentamycin induced renal damage (80mg/k.g. b.wt for 10 days) are in harmony with the above authors.

Surendra K 2011[13] investigated the effects of pre-treatment of aqueous extract of *B. diffusa* root (200 – 400 mg/kg/day) in repeated dose acetaminophen nephrotoxic rats for 14 days. pre-treatment with *B. diffusa* extract protected against degenerative changes renal cortical architecture in the experimental rats. Antioxidant enzymes such as Glutathioneperoxidase, Reduced glutathione, Vitamin C and Catalase were elevated in animals treated with Boerhaavia *diffusa* aqueous extract which showed that the aqueous extract of *Boerhaavia diffusa* leaves significantly reduced the nephrotoxicity induced by mercuric chloride(T.Indhumathi et al 2011[14]). The herb is a diuretic that acts on the glomeruli of the kidney and also protects the kidney from being damaged (Rawat *et al*, 1997[15]).

Post -treatment with *B. diffusa pure* extract (400mg/kg.b.wt and 800mg/kg.bwt for 9wks exhibited anti-nephrotoxic effects(curative) against degenerative changes of renal cortical architecture and also significant normal values of biochemical parameters.

V. Conclusion

- Daily intraperitonial injection of rats with 80 mg gentamicin /kg b.w for 10 days caused a serious harmful effects on renal function tests.
- Treatment with *B. diffusa pure* extract (400mg/kg.b.wt and 800mg/kg.bwt for 9wks exhibited anti-nephrotoxic effects(curative). Histopathological findings indicate rejuvination of necrotic cell in kidney
- Thus, it could be suggested that gentamicin must be given in the lowest effective therapeutic doses in patients with normal kidney function.
- Also, gentamicin therapy should be accompanied with administration of punarnava which will nullify the nephrotoxic effect of gentamycin.
- Boerhavia diffusa (Punarnava) even though given in larger doses will not have any side effects .

Acknowledgements

I thank Dr.B.Narasinga Rao, Professor and HOD of Anatomy , Dr.T.A.V.Narayana Raju Dean, Dr. Ramalakshmi, Professor and HOD of pathology and Dr.Mamatha bandopadhyay, Professor and HOD of Pharmacology of Maharajahs Institute Of Medical Sciences and Dr. Saraswathi Professor and HOD of Anatomy , Saveetha Medical College for their encouragement and support. I Thank Dr. Gopi , Saveetha Medical College, Chennai for rendering his help in statistical work,.

Note: This study is a partial work of my Ph.D research , titled “An experimental anatomical study on the effectiveness of punarnava (Boerhaevia diffusa) on gentamycin induced renal failure in albino rat and to estimate the drug induced damage and revival of renal tissue” . Part of this work has been published in iosrjdms journals with gentamycin nephrotoxicity.

References

[1] Nephrol Dial Transplant. 1994;9 Suppl 4:135-40 Singh RH, Udupa KN. Studies on the Indianindigenous drug Punarnava (*Boerhaavia diffusa* L.) Part I, Identification and pharmacological studies. *J Res Ind Med* 1972; 7: 1-12. 25

[2] Gaitonde BB, H. Kulkarn, Nabar SD. Diuretic activity of *punarnava* (*Boerhaavia diffusa*). Bull. Haffkine Inst. 1974; 2: 24-25.

[3] Singh RH, Udupa KN. Studies on the Indianindigenous drug Punarnava (*Boerhaavia diffusa* L.) Part I, Identification and pharmacological studies. *J Res Ind Med* 1972; 7: 1-12. 25.

[4] Pareta SK, Patra KC, Mazumder PM, Sasmal D. *Boerhaavia diffusa* linn aqueous extract as curative agent in ethylene glycol induced urolithiasis. Pharmacologyonline 2011; 3: 112-120.)

[5] Mishra J, Singh R. The effect of indigenous drug *Boerhaavia diffusa* on kidney regeneration. *IndJ Pharmacol* 1980; 12: 59-64. 24.

[6] Dvipriya S, Shyamaladevim CS. Protective effect of quercetin in cisplatin induced cell injury in the rat kidney. Indian J. Pharmacol. 1999; 31: 422-423.)

[7] Ali,B.H.; Al-Qarawi, A.A.; Haroun,E.M. and Mousa, H.M.(2003): The effect of treatment with gum arabic on gentamicin nephrotoxicity in rats Ren Fail., 25(1):15-20.

[8] Goto, A.M. (2004): "The role of lipid coronary heart disease" Kalamazoo, M. I. Upjhion Company

[9] Heibashy, M.I.A.; El-Nahla, A.M.; Ibrahim, A.I. and Saleh, Sh.Y.A. (2009): Comparative study between dimethyl sulfoxide (DMSO),allopurinol and urate oxidase administration in nephrotoxic rats induced with gentamicin. 43rd Annual Veterinary Medical Symposium, College of Veterinary Medicine Nursing and Allied Health, Tuskegee University, Alabama, USA

[10] Heibashy, M. I. A. and Abdel Moneim, A. E. (1999): Kidney and liver function tests after late Dimethyl sulfoxide (DMSO) administration in rats with gentamicin induced acute renal failure. J. Egypt. Ger. Soc. Zool., 30(A): 35-48.

[11] K.Vijay Kumar, M.U.R. Naidu, Anwar A. Shifow, *K.S. Ratnakar Probucol protects against gentamicin-induced nephrotoxicity in ratsindian journal of pharmacology 2000; 32: 108-113

[12] Shirwaikar A, Malini S, Kumari SCProtective effect of Pongamia pinnata flowers against cisplatin and gentamicin induced nephrotoxicity in ratsIndian J Exp Biol. 2003 Jan;41(1):58-62.

[13] Surendra K. Pareta, Kartik C.Patra, Ranjeet Harwansh, Manoj Kumar, Kedar Prasad Protective Effects of Boerhaavia Diffusa Against Acetaminophen-Induced "Nephrotoxicity in Rats Pharmacologyonline 2: 698-706 (2011)

[14] Indhumathi T, K.Shilpa, S.Mohandass Evaluation of Nephroprotective role of *Boerhaavia diffusa* leaves against mercuric chloride induced toxicity in experimental rats.Received on: 11-02-2011; Revised on: 16-03-2011; Accepted on:21-04-2011*T.Indhumathi et al. / Journal of Pharmacy Research 2011,4(6),1848-1850)*

[15] Rawat, A.K.S., Mehrotra, S., Tripathi, S.K., and Shama, U. (1997). Hepatoprotective activityin *punarnava* – a popular Indian ethnomedicine. *Journal of Ethnopharmacology* 56(1):61–68.

IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861. Volume 9, Issue 6 (Sep.- Oct. 2013), PP 22-42
www.iosrjournals.org

Maternal and Fetal Outcome of Grandmultipara in Comparison to Multiparous Woman in Two hospital in Khartoum State

Dr Isamaldin Alamin Mohmed Ahmed *MBBS.MD.DOWH*
Obstetrics and Gynaecology, Sudan Medical Specialization Board, Sudan

Abstract:
Aims and Objectives: *To compare the incidence of feto-maternal complications of pregnancy and labor between grandmultiparas and multiparous women.*
Material and Methods: *This prospective study was carried out in the Departments of Obstetrics and Gynaecology in two hospitals in Khartoum state (Omdurman Maternity Hospital, and Khartoum North Teaching Hospital), in the period from January 2010 to June 2010. A total of 450 deliveries in the two hospital were divided in two groups (150 grandmultiparas and 300 multiparas) in whom the maternal and fetal outcomes were analyzed and compared.*
Results: *It was found that in the GMPs there were increase in age more than 35 years 38% vs 9% ($p < 0.05$), lower socioeconomic class 48% vs 28.7%, ($p < 0.05$), incidence of hypertensive disorder of pregnancy 30.0% vs 13.7% ($p<0.05$), diabetes mellitus 8% vs 3% ($p<0.05$). anemia 28.7% vs 12.7% ($p<0.05$), multiple pregnancy 8.0% vs 3.3% ($p<0.05$), breech presentation 9.4% vs 5.5%, ($p<0.05$), transverse lie 3.6% vs. 0.3% ($p>0.05$), preterm labour 16.0 vs. 12.7% ($p>0.05$), overall APH 8.7% vs 5.7% ($p<0.05$), and Macrosomic babies 18.8% vs 5.8% ($p>0.05$), and SVD 76.7% vs 60.3% ($p<0.05$).*
there was a decreased incidence in induction of labour 2% vs 11.0% ($p<0.05$), instrumental delivery 6% vs 12%, ($p<0.05$), Em C/S 17.3% vs 27.7%,($p<0.05$), overall PPH 8.0% vs 9.0%, ($p>0.05$), LBW 7.2% vs 11.0%, the ($p<0.05$) and maternal mortality 0.7% vs 1% ($p>0.05$
Other than birth weight, there was no significant differences in fetal outcomes, we found that, still birth 10.1% vs 10.0% and babies distressed 3.2% vs 4.2%.
Keyword: *grandmultipara, multiparous, maternal outcome, fetal outcome, placenta previa, abrubtio placentae,caesarean section.*

I. Introduction

1.1 Definition

The International Federation of Gynaecology and obstetrics 1993 defined grandmultiparity as the delivery of fifth to ninth, whereas women who are undergoing their tenth or more deliveries considered to be great-grandmultiparaes.[8].

King et al, and Toohey et al, considered grandmultiparae to be women who gave birth to five or more previous deliveries[2,7]. (definition which we used in this study).

For a pregnancy to count as a "birth", it must go to at least 24 weeks gestation or yield an infant that weighs at least 500 grams, irrespective of whether the infant is live born or not.{26}.

In his 1934 article[1], entitled 'The dangerous multiparae', Bethel Solomons wrote: "my main object is to remove if possible once and for all, the idea that a primigravida means a difficult labour but a multiparae means an easy one, It is altogether a mistake to suppose that in childbearing practice makes perfect".

Solomons' concern for the multiparae was prompted by a study at the time from the Department of Health for Scotland which showed the maternal mortality rate associated with multiparity increasing "steadily and speedily". from the fifth pregnancy until women bearing their tenth child or more; had a mortality rate five times as high as all women bearing children.

Since that time a number of studies have been reported from various parts of the world investigating the role of high parity on perinatal outcome. [1,4.36,19,18,42].

Pregnancy after fifth delivery is viewed with anxiety, especially by obstetricians in developing countries working under difficult conditions and with limited facilities. The complications that occur to these patients during pregnancy, at the time of delivery, and in the perperium warrants them special respect, first of all the ordinary complications of pregnancy and child birth are increased in this group of women, secondly, since such complications tend to be more common in the lower socioeconomic groups, their ill-effects have a far reaching impact on the patients affected due to poverty, lack of facilities and the usual accompanying delayed presentation to medical personnel. Moreover, these patients usually lack medical education, they are undernourished and almost always overworked, with usually poor antenatal care.[4,5,6,42].

increasing cognizance has been taken of grandmultiparity as a clinical entity in its own right. These patients are liable to a series of dramatic complications, all the more dangerous because they are often unsuspected.

Since most highly parous women are above the age of 35, these women are more prone to develop complications such as diabetes, hypertension, anemia, and chronic renal disease which complicate their ongoing pregnancies even more as opposed to the younger patients who are usually free of such complications.

Intrapartum complications that are classically associated with grand multiparas include fetal malpresentations, dysfunctional labour, hypertensive disease, anemia, placenta previa, retained placenta, uterine rupture, abruptio placentae, post partum haemorrhage and macrosomic babies.

Other general conditions which are part of the normal process of ageing are liable likewise to intrude themselves upon the clinical picture, obesity, which is often gross in these cases, increases the dangers of child bearing, as is well known, and not the least reason for this is the difficulty of making an accurate examination.

Sociological factors play a very important part, for, the majority of these patients are poor, overworked and tired, many of them have never fully regained a good blood picture, and anemia may dog them from one pregnancy to the next without respite, they tend to feed their numerous children at the expense of their own nutrition, so that they are consequently often very short of vitamins and first class proteins, they are too busy to attend to their health, and due to the rapid succession of pregnancies and periods of lactation they are likely to become depleted from calcium..[6,36,37,19,18].

Although grandmultiparity has long been considered an obstetric complication for both mother and fetus recent studies indicated that, with proper perinatal care, women with high-parity rates are no longer at high risk.

in March 2002 the British Journal of Obstetrics and Gynaecology published a new study of grandmultiparae in the UK. The authors concluded that: in a developed country with satisfactory health care conditions, grandmultiparity should not be considered dangerous, and risk assessment should be based on past and present history and not simply on the basis of parity, so the controversy concerning the risk of grandmultiparity can be somewhat resolved in countries where obstetrics is uniformly practiced at modern level of excellence where almost all pregnancies are followed in antenatal outpatient clinics, and deliveries are carried out in well equipped medical centers.[9].

Nordin N.M. in his study Is grandmultiparity a significant risk factor in this new millennium, With adequate care, the maternal fetal outcome of grandmutiparous women is good and comparable to the multiparous women.[(45)].

Shah P.S. conclude that grand multiparity and great grand multiparity were not associated with increased risk of pregnancy outcomes[20].

II. Literature Review

2.1RISK FACTORS

2.1.1 Age

Advanced maternal age is an important factor that contributes to adverse maternal outcome and can be associated with high parity.

Hecht-Resnick R, Harel found that the age of the grandmultiparas was significantly higher compared with the control groups, which may explain the higher incidence among them of antenatal medical disorders, such as diabetes mellitus and hypertensive disease[11].

Maymon E, Ghezzi F assess the importance of birth order and advanced maternal age on peripartum complications he found that Higher birth order remained an independent risk factor for peripartum complications after adjustment for maternal age[13].

2.1.2 SOCIO ECONOMIC STATUS

Since high parities tend to be more common in the lower socioeconomic groups who have low income this leads to affect nutritional and health status of these mothers and with no facilities to contact health centre and family planning services.

Nassar A.H.found that Grandmultiparity was associated with a low socioeconomic status[43].

Mor-Yosef et al in his survey which covers 22,815 deliveries in a 3 month period, the 1542 grandmultiparous women were divided into two groups: low socioeconomic group (947) and high socioeconomic group (595). Perinatal mortality and low birth weight were found to be in correlation with low socioeconomic status but not with grand multiparity. Maternal diseases complicating pregnancy were found to be significantly more common for grand multipara for both socioeconomic groups [18].

Both Toohey et al[7] and King et al.[21] reported highly favorable outcomes in a group of women with low socio-economic status so the effect of the socioeconomic status on perinatal outcome need to be evaluated.

2.2 Antepartum and Intrapartum Complications

2.2.1 Hypertensive disorders

Hypertensive disorders of pregnancy are common complication it include:

Pregnancy induced hypertension which occurs in around 16-24% of first pregnancies and 12-15 %of subsequent pregnancies, pre-eclampsia [PE] which complicates 3-5% of first pregnancies and 1% of subsequent pregnancies with around 5-10% of cases being sever, chronic hypertension [CHT] is present in 2-4%of pregnant women, over 90% of cases are due to essential hypertension, and these women tend to be older and heavier with a family history of hypertension[25].

In a series of 1567 deliveries over 10 years in the same hospital in Oulu, Finland, Vehaskari A et al, reported the special obstetric characteristics of grand multiparas (GMs) and the extent to which the parturient age affected the incidence of complications, differences were noted between the obstetric behavior of GMs as compared with other parturients. Hypertensive disease was distinctly more frequent among GMs than among the other parturients [12].

2.2.2 Diabetes mellitus

The WHO has defined diabetes mellitus as either a raised fasting blood glucose level of >7.8 mmol/l or a level of 11.1 mmol/l 1-2hours following a 75 grams glucose load. The importance of good glycaemic control during pregnancy is reinforced by the direct relationship between blood glucose levels and the incidence of fetal and maternal complications.[23].

Nassar A.H.found that Grandmultiparas had ~2-fold increased risk of gestational diabetes.[43].

Goldman GF et al in his case control study found that, the age of grand multipara was significantly higher which may explain the higher incidence among them of antenatal medical disorders such as diabetes mellitus and hypertensive disorders [11].

2.2.3 Anaemia

The WHO recommends that the HB concentration should not fall below 11g/dl at any time during pregnancy, but many clinicians use the figure of 10.5 g/dl as recommended by the centres for Disease Control of North America[26].

Anaemia is one of the most frequently observed nutritional deficiencies in the world today. It is especially prevalent in women of reproductive age particularly during pregnancy, it is often a contributory cause of maternal morbidity and mortality. GMP and short birth interval is well documented as a risk factor of anaemia. This is because there is not enough interval between pregnancies for the women to replenish their iron stores .However, it is generally accepted that a women requires an interval of two to three years between births to recover physiologically from the effect of previous delivery [23].

A matched cohort study in an inner city university maternity hospital in the United Kingdom, three hundred and ninety-seven grandmultiparous women were compared with three hundred and ninety-seven age-matched multiparous women. Grandmultiparous women were significantly more likely to have a haemoglobin <10g/dL antenatally than ordinary multiparous women [9].

2.2.4 Antepartum Haemorrhage (APH)

APH: Bleeding from the birth canal after viability until the onset of labour. The major causes of APH are placenta praevia and placenta abruption.

In developing countries, today, wide spread pre-existing anaemia, difficulties with transportation and restricted medical facilities ensure that APH continues to be responsible for many maternal deaths, fetal loss is much more common than maternal death, 15% of perinatal deaths can be attributed to APH, notably from abruption'[25].

In a series of 1567 deliveries over 10 years in the same hospital in Oulu, Finland, Vehaskari report the special obstetric characteristics of grand multiparas (GMPs) and the extent to which the parturient age affected the incidence of complications. Differences were noted between the obstetric behaviour of GMPs as compared with other parturient, found the frequency of abruptio placentae, placenta praevia, and retained placenta was significantly higher in GMPs. Although the difference was not statistically clear in this study, Abruptio placentae is affected both by high parity and age, while placenta praevia is independent of age and predisposed by high parity[12].

Nassar A.H.found that Grandmultiparity was associated with a 3-fold increased risk of abruption; [43].

The incidence of antenatal and intrapartum complications and neonatal outcomes was compared G. J. Bugg et al found that there was no significant difference observed in the incidence of placental abruption, dysfunctional labour, malpresentation, or postpartum haemorrhage'[9].

A Babinski et al, studied One hundred thirty-three great-grand multiparas, 314 grand multiparas, and 2195 multiparas who were delivered of their infants between 1988 and 1998 for the study, and he found the

incidence of postpartum hemorrhage, , placenta previa, was significantly higher in grand multiparas than in multiparas [8].

F A Aziz et al, during the period 1975-1979 in the three major hospitals in Khartoum, Sudan, the study population consisted of 8858 patients who delivered in those hospitals; 3130 of them had five or more children. The obstetric complications and the fetal outcome were investigated, and comparisons were made between groups according to parity, there was a high rate of antenatal complications, such as anaemia, antepartum haemorrhage and postpartum haemorrhage, among the grand multiparous patients. The stillbirth rate and neonatal mortality were high [32].

2.2.5 Pattern of labour in GMPs

Gurewitsch et al, in his retrospective cohorts study of spontaneously labouring, vertex-presenting, term, grand multiparous women (parity >5) from two medical centres over 5.5 years were matched randomly to nulliparous women and lower parity multiparous women controlled for age, hospital, and year of delivery. Descent curves for labour were modelled and the results were, GMP women maintain a higher station for a longer time before delivery compared with nulliparous women or P1-4 women but transition rapidly to delivery once full dilatation is reached. Higher station likely contributes to the slower progress of first-stage labour among GM women compared with P1-4 women. More important is the slower initial progress of either dilation or descent should not be considered abnormal for the GMP woman, and although high station is associated with both primary dysfunctional labour and secondary arrest of dilation in nulliparous women, this is not the case for multiparous women, as evidenced by the differences in operative delivery rates between these groups yet similar results in terms of perinatal outcome[21].

S.AruIkmara et al carried out study to see the effect of increased parity in uterine activity during labour, they studied 400 multipara including GMP of Chinese origin. The result was multiparus women with previous vaginal delivery seems more likely to have an easy labour due to easily progression in cervical dilatation, more efficient uterine contraction and reduced pelvic and cervical tissue resistance than those with low parity and prirnigravida'[24].

2.2.6 Postpartum haemorrhage (PPH)

Postpartum haemorrhage is defined as excessive bleeding from the genital tract following the delivery of the baby. The WHO defines primary PPH as bleeding in excess of 500 ml in the first 24h following delivery ,the main causes of primary PPH are failure of the uterus to contract effectively [atonic uterus] .retention of placenta and membranes in the uterus and trauma to the genital tract[25].

A case control study to determine the risk factors for primary PPH at Obafemi Awolowo University Hospital. The study consisted of 101 women who developed PPH after normal vaginal delivery and 107 women with normal unassisted vaginal delivery without PPH. The results showed significant relation ship with prolonged second and third stages of labour and non use of oxytocics after vaginal delivery. Previously hypothesized risk factors for PPH such as grand multiparity, primigravidity and previous episodes of PPH were not significantly associated with PPH [27].

In Nigeria 204 cases of primary PPH were studied compared with the same number of normally delivered cases, the result was; primary PPH occurs more frequent in PG and GMPs than multipara, with more occurrence in GMPs. The commonest cause was uterine atonia which attributed to mismanagement of third stage of labour also they add the mood of delivery and anaemia as contributing factors'[28]

2.2.7 Uterine Ruptures

Rupture of the gravid uterus is one of the most serious obstetrical emergencies, carrying a high maternal morbidity and mortality with high rate of perinatal loss, associated with loss of future fertility due to performance of hysterectomy or repair with tubal ligation in some patient[29].

Vadat A et al analyzed 150 cases of uterine ruptures in late pregnancy in 8 years duration, he found that; the common etiological factors were GMP, malpresentation and oxytocin hyperstimulation in labour in unscarred uterus, hysterectomy or repair with tubal ligation were performed to all cases with increase maternal morbidity and mortality[29].

Matched cohort study was done in an inner city university maternity hospital in the United Kingdom. Three hundred and ninety-seven grandmultiparous women were compared with three hundred and ninety-seven age-matched multiparous women .The incidence of antenatal and intrapartum complications and neonatal outcome

G. J. Bugg found that overall, grandmultiparous women had an intrapartum complication incidence of 18%, which was not significantly different from the 18% rate observed in the mtiltiparous group. There were no uterine ruptures or maternal deaths in either group [9].

In a series of 1567 deliveries over 10 years in the same hospital in Oulu, Finland, Vehaskari A et al reported the special obstetric characteristics of grand multiparas. The incidence of uterine rupture has been reported to be higher in GMPs than in other parturient,[12],

2.2.8 Other Complications

G. J. Bugg et al found that Overall, grandmultiparous women had an intrapartum complication incidence of 16%, which was not significantly different from the 18% rate observed in the multiparous group. There were no uterine ruptures or maternal deaths in either group. there was no significant difference in the incidence of emergency caesarean section (7.0% vs. 8.3%) between the two groups[191].

The case-notes and records of grandmultiparous patients delivered at the Lagos University Teaching Hospital between 1st January, 1994 and 31st December, 1996 were analyzed. The incidence of intrapartum complications, cephalopelvic disproportion, obstructed labour and Caesarean section, were found to be higher in the unbooked grandmultiparous patients'[10].

Hecht-Resnick et al examines the outcome of delivery in 1700 women in their fifth or more delivery, as compared with two control groups: 622 primiparas and 735 multiparas (two to three previous deliveries). No significant differences were found among the three groups for preterm or post-term births, small-for-gestational-age infants, polyhydramnios, oligohydramnios, perinatal death, fetal distress, multiple births, placenta previa, abruptio placentae or cord prolapse [11}

In a series of 1567 deliveries over 10 years in the same hospital in Oulu, Finland, differences were noted between the obstetric behaviour of GMPs as compared with other parturient study showed that breech presentation to be less frequent among GMs. Vehaskari A et al, found the incidence of operative deliveries was roughly similar in both groups. Caesarean section, including repeat sections, was distinctly lower among GMPs; no difference appeared neither in the incidence of multiple pregnancies, nor in the incidence of prematurity [12].

Babinski found the incidence of malpresentation at the time of delivery, anemia, preterm delivery, and meconium-stained amniotic fluid increased with higher parity, whereas the rate of caesarean delivery decreased The incidence of pre eclampsia, , macrosomia, postdate pregnancy, and low apgar scores was significantly higher in grand multiparas than in multiparas, whereas the proportion of induction, forceps delivery, and total labour complications was significantly lower than in the multiparous group'[8].

Al-sibai et al, reviewed 1130 patients who had 7 or more viable pregnancies at the University Teaching Hospital in Al-Khobar, Saudi Arabia. Delivery complications were higher for the study group, however, than for total deliveries: breech deliveries, 7% vs. 2.7%; premature labour, 7.5% vs. 2.7; caesarean section, 11.4% vs. 8.9%; and postpartum haemorrhage. 6.5% vs. 3.1 % [14].

Rizk DE et al, reviewed the records of 418 grand multiparous women (study group), defined as having had given birth at least 5 times after completed 22 weeks gestational age, and 418 women of parity 2-4 (control group). Diabetes mellitus (both overt and gestational) was significantly more common in the study group) but there was no significant increase in the incidence of other- obstetric complications or in perinatal mortality rate. Babies of grand multiparous mothers required significantly more admissions to special care unit because of maternal diabetes mellitus)[16].

Sipila P. et al found that the grand multipara had a higher incidence of essential hypertension than women of lower parity. The grand multipara had fewer caesarean sections (7.5% vs. 14.1%) and vacuum extractions (0.5% vs. 5.1%o) but more inductions of labour (33.1% vs. 23.5%) than mothers of lower parity [19].

Karl Fuchs et al study is based on 5785 cases of GM which were compared to the general obstetrical population in terms of pregnancy and delivery complications. Face and breech presentations as well as transverse lie were twice, brow presentations were three times as frequent in the GM group. Postpartum haemorrhage (P.P.H.) was four times and premature separation of the placenta twice as frequent. Rupture of the uterus was about 20 times more frequent. Forceps delivery and caesarean section rate were twice, while the vacuum extraction 5-fold more frequent. Though there was no maternal mortality, and perinatal mortality was not higher than in the general population.[22].

A total of 7785 mothers was studied, 889 (11.5%) by Eideiman of whom were grandmultiparas. Comparison of grandmuftiparous mothers with all others revealed no increase in the incidence of hypertension, diabetes, uterine atonia, antenatal or postnatal haemorrhage, caesarean sections, stillbirth rate, or congenital malformations. The grandmultipara had significantly lower neonatal mortality and low birth weight rates and a significantly higher incidence of multiple births and trisomy 21[30].

A retrospective analysis of 646 Arab grandmultiparas who booked for hospital confinement between 1983 and 1985 was carried out. The results were compared with that of non-grandmultiparas during the same period. In the grandmultiparas, the incidences of gestational diabetes, hypertension, rheumatic heart disease, antepartum, postpartum haemorrhage and macrosomic infants were increased. However, contrary to some previous reports the incidences of anemia, caesarean sections, induced labour, dysmaturity and perinatal deaths

were decreased. This is thought to be due to the provision of modern specialist perinatal care and improved socioeconomic standards[31].

2.2.9 Maternal Mortality rate (MMR)

Every minute of every day some where in the world, a woman dies as a result of complications arising during pregnancy and childbirth the majority of these deaths are avoidable. Maternal death is a tragedy for individual women, for families ,and for their communities The medical causes of maternal deaths are similar throughout the world.

Maternal mortality is the death of a women while pregnant or within 42 days of termination of pregnancy, regardless of the site or duration of pregnancy, from any cause related to or aggravated by pregnancy or its mismanagement direct or indirect [WHO].

A report of the special obstetric characteristics of grand multiparas (GMPs) and the extent to which the parturient age affect the incidence of complications was done in a series of 1567 deliveries over 10 years in the same hospital in Oulu, Finland, differences were noted between the obstetric behaviour of GMPs as compared with other parturients, the maternal mortality rate of GMPs in the series was significantly higher than that of the other parturient (12%). The primary causes of death were abruptio placentae, rupture of the uterus and eclampsia a state of shock was a feature common to all the fatalities[12].

Ogedengbe OK, found that The maternal mortality ratio was 44.4/1000 amongst the grandmultipara which was not statistically more significant than in the general obstetric population'[10].

2.3 Fetal Outcome

G. J. Bugg et al found that there was one stillbirth and two neonatal deaths in the grandmultiparous group and two stillbirths and three neonatal deaths in the multiparous group. Mean birth weights were 3329g (720) in the grandmultiparous group and 3307g (695) in the multiparous group. No significant differences in neonatal outcomes were found between the two groups[9].

Hecht-Resnick et al, found that Macrosomia was markedly higher in the grandmultiparas and multiparas than in nulliparas'[11].

A Babinski, T Kerenyi found that low apgar scores and macrosomia, were significantly higher in grand multiparas than in multiparas although perinatal mortality remains low in these patients[8].

From 1971 to 1988, out of 22001 deliveries (multiple pregnancies excluded) Newborn infants in the GMPs group were severely asphyxiated at birth more frequently than those in the control group. The overall perinatal death rate in the GMPs and control group was 2.83% and 1.81%, respectively'[36].

A total of 382 grand multiparous women (para > or = 5) were compared with 382 age-matched control subjects (para 2 to 4), all delivering between July 1989 and September 1991, Grand multiparity was associated with an increased incidence of macrosomia (16% vs 11%), Macrosomia increased the incidence of intrapartum complications from 31% to 46% ($p< 0.03$) in the grand multiparous patients, and a trend was observed in the multiparous patients, from 26% to 37%. However, when properly controlled, this was noted to be a confounding variable and was not related to parity'[7].

III. Justification of the study

The Grand Multipara (GMP) has almost disappeared in the western countries (3-4% of all birth)[36]. due to the improvement of economic status, advancement of family planning and elective termination of pregnancy, In our country Sudan- having heterogeneous population- the problem of grandmultiparity still exsit. For that reason and because we have a good improvement in the setup of our medical services in the last few years, we focus on a relevant clinical questions:

Grand Multiparous woman in our population, is she at increased risk of complications? and multiparous lady, is she really in the safe group, called safe parity?.

IV. Objectives

4.1 General:

To compare the incidence of maternal and fetal complications of pregnancy and labor between grandmultiparae and multiparous women.

4.2 Specific:

4.2.1 Maternal:

To compare the two groups in the incidence of

1-antepartum complications (hypertension, diabetes mellitus and anaemia).

2-Intrapartum complications (multiple ,pregnancy, presentation, and APH).

3-Post partum complications (mode of delivery, PPH, and maternal mortality).

4.2.2 Fetal:

to compare the incidence of respiratory distress, admission to the nursery, fetal weight, and stillbirth between two groups.

V. Methodology

5.1 Study design:

This is a prospective case control, hospital based study.

5.2 Study area:

This study was done in Omdurman Maternity Hospital(OMH), and Khartoum North Teaching Hospital(KNTH), they are two major hospital in Khartoum, the capital of Sudan (population more than 5.2 millions).[38].

Omdurman Maternity Hospital(OMH), is the first specialized hospital in Sudan that has been established in 1957. The hospital cover the area of Omdurman, (population about 2.3 millions), receives patients from different areas of Khartoum, and some patients come from other parts of Sudan, the hospital contains labor wards, antenatal wards, and postnatal wards. there is a blood bank laboratory , two theatres (elective and emergency).department of laparoscopic surgery, well equipped neonatal unit (SCBU). Pharmacy, ICU. The labour ward contains 25 beds, ultrasound and CTG. machines The total number of beds are 136, the services are covered by consultants ,registrars, house officers, sisters and midwifes, distributed in five units, the average number of deliveries are about 70 to 100 per day.

Khartoum North Teaching hospital (KNTH) is central hospital that provides services to most of the inhabitants of Khartoum north and east Nile, population about (1450000), KNTH services cover the relevant branches of medicine, surgery, pediatrics, pediatrics surgery, dermatology, orthopedics, E.N.T, Obstetrics and Gynaecology, blood bank, laboratory, pharmacy, Services in the obstetrics and Gynaecology unit are covered by consultants, registrars, house officers and midwives divided into five units. The department is separated and composed of labor room, theatre, neonatal unit, antenatal wards postnatal wards, septic and gynecology wards and eclampsia room, there are US and CTG unit in the hospital, the average number of deliveries are about 40 to 50 per day. In the two hospital about 15% GMP and 60%multiparous woman.

5.3 Study population:

All pregnant women who delivered in Omdurman maternity and Khartoum North Teaching Hospitals who had been fulfilling the inclusion criteria in the period of the study.

5.4 Inclusion criteria:

A case is defined as a woman who delivered 5 or more after 24 weeks gestation (group A).
Control is defined as woman who delivered 1 to 4 birth after 24 weeks gestation (group B).
Willingness to participate in the study.
Exclusion criteria:
primigravidas.
Women with previous caesarian section.
Refusal to participate in the study.

5.5 Data collection:

a detailed questionnaire, was designed, and filled by direct questioning, examination and follow up of the patients since admission till discharge from hospital, I observed and followed up the patients during their stay in the hospital and filled the questionnaire in a proper way, and, I trained some doctors who were working in the labour room about how to fill the questionnaire correctly because they did that during my rest time and when there were more than one patient laboring at the same time.

During our data collection we did not interfere with the hospital protocol of management of patients.

The questionnaire consists of:

- Personal history
- Parity.
- Social class which was classified to low, moderate or high. According family income.
- gestational age in weeks and days
- haemoglobin measurement .
- Urine analysis was done for Albumin.
- diabetes mellitus ,hypertentions, and a anemia.
- Duration of the first Stage: the time from active labor until the cervix becomes fully dilated(2 to 6 hour) [29]

- o Number of fetuses: singletone twins, triplet.
- o presentation: cephalic, breech, others.
- o Indication of Em c/s.
- o Complications during pregnancy .
- o Intrapartum complications.
- o Causes of PPH.
- o Fetal outcome: alive, stillbirth.
- o apgar score.
- o Birth weight:

Data Analysis:
Data was entered into SPSS (statistical package for social science) computerized program for analysis, chi-square and P value less than 0.05 was considered significant.
Data Presented in tables and graphs.
Ethics:
1-informed consent was taken verbally from patient and we stress the fact that participation of this study was voluntary.
2- Approval to do this study was taken from the general director of Omdurman Maternity Hospital.

VI. Results

A total of 450 patients, 150 were cases (GMPs) and 300 were control group Multiparas (Para1-4) were studied. Data was collected and analyzed using statistical package of social science SPSS17.
The results of the study were presented in tables & figures.
Table (1):
Shows the distribution of the cases and control groups by their ages 0.7% of the cases were less than 20 years old, compared to 3.7% of the control group.
On the other hand 38.0% of the cases were above 35 years old, while only 9.0% of the control group.
This difference is statistically highly significant. (P=0.00).
Figure(1):
Shows the correlation between the parity and socioeconomic status. 48.0% of the cases were found to be of low socioeconomic status compared with 28.7% of the control group.
42.7% of the cases were found to be of moderate socioeconomic status compared with 46.3% of control group.
9.3% of the cases were found to be of high socioeconomic status compared with 25% of the control group.
This difference is statistically significant. $P < 0.05$.

Figure (2):
Shows the relation between parity, and gestational ages of the two groups.
16.0% of cases had gestation ages less than 37 weeks (preterm labour), while 12.7% the control group had.
62.7% of cases had gestation ages 37 to 40 weeks (term), while 60.3% the control group had.
21.3% of cases had gestation ages more than 40 weeks (post-term), while 27,0% the control group had.
This difference is statistically not significant. $P > 0.05$.
Figure (3):
Shows the correlation between parity and hypertensive disorders of pregnancy.
Of the grandmultipara 30.0% had hypertension disorders of pregnancy while 13.7% of the multipara had.
This difference is statistically significant. $P < 0.05$.
Figure (4):
Shows the distribution of cases and control group by presence of DM or gestational DM.
8.0% of the cases were found to have the disorder while, 3.0% of the control group had.
This difference is statistically significant. $P > 0.05$.
Figure (5):
Shows the distribution of cases and control group by the presence of anaemia or not.
28.7% of the grandmultipra had anaemia while 12.7% of the control group had.
The difference is statistically significant. $P < 0.05$.
Figure (6):
Shows the relation between parity and multiple pregnancy. 8.0% of cases had multiple pregnancy while 3.3% of control group had.
This difference is statistically significant. $P < 0.05$
Figure (7):
Shows the correlation between parity and presentation.

9.4% of the grand multipra had breech presentation while 5.5%of the control group had.
3.6% of cases had transverse lie compared to 0.3% of the control group had.
This difference is statistically significant. $P < 0.05$.
Table(2):
Shows transverse lie, and face presentation.
In transverse lie there were 80% in cases and 20%in the controls group.
In face presentation all the cases were in control group .
This difference is statistically not significant. $P > 0.05$.
Figure (8):
Shows the distribution of the cases and control group according to antipartum hemorrhage (APH).
2.7% of the cases had bleeding due to placenta previa while 4.0% of the control had.
6.0% of the grand multipara had bleeding due to abruptio placentae compared to 1.7% of the multipara
This difference is statistically significant. $P < 0.05$.

Table(3):
Shows the relation between parity and onset of labour. 2.0% of the GMPs had induction of labour, while 11.0% of the Multipara had.
This difference is statistically highly significant. (P=001).
Table (4):
Shows the relation between parity and the duration of the first stage of labour.18.0% of GMP had first stage of labour less than 2 hours while 7.7% control had.
21.3% of GMP had first stage of labour more than 6 hours while 47.7% control had.
This difference is statistically highly significant. (P=0.00).
Figure (9):
Shows the distribution of cases and control groups by the mode of delivery.
76.7% of cases had SVD compared with 60.3% in control group.
Assisted VD was 6.0% in cases compared with 12.0% in control group.
Ems C/S was 17.3% in cases compared to 27.7% of control group.
This difference is statistically significant. $P < 0.05$.
Table (5):
Compare the indications of emergency caesarean sections in the two groups.
Failure to progress: 9.1% in GMPs while 90.9% were in the control group.
Fetal distress: 27.3% in GMPs while 72.7% were in the control group.
Obstructed labour: 14.3% in GMPs while 85.7% were in the control group.
Abnormal lie: 83.3% in GMPs while 16.7% were in the control group.
APH: 32.0% in GMPs while 68.0% were in the control group.
PE: 16.7% in GMPs while 83.3% were in the control group.
Others causes: 40.0% in GMPs while 60.0% were in the control group.
This difference is statistically significant P =003.
Figure (11):
Shows the distribution of the cases and control group by presence of PPH.
8.7% of cases had PPH compared to
9.7% of control group.
This difference is statistically not significant. $P > 0.05$.
Table (6):
Compare the causes of PPH in the two groups.
Uterine atony: 75.0% in GMPs, while 25.0% in the control group.
Genital tract injury : 19.0% in GMPs, while 81.0% in the control group.
Retained products : 14.3% in GMPs, while 85.7% in the control group.
Blood coagulpathy : 33.3% in GMPs, while 66.7% in the control group.
This difference is statistically significant. $P < 0.05$.
Table (7):
Shows the types of genital tract injuries in the two groups.
Ruptured uterus : 20.0% in the GMPs, while 80.0% in the control group. 25.0% of perineal tear: 10.0% in the GMPs, while 90.0% in the control group.
Extended episiotomy: 25.0% in the GMPs, while 75.0% in the control group.
Other causes: 50.0% in the GMPs, while 50.0% in the control group.
This difference is statistically not significant. $P > 0.05$.
Figure (12):

Shows the relation between parity and maternal death.
0.7% (1-woman) of the cases died compared to 1.0% (3 women) died from the control group.
This difference is statistically not significant.P>0.05.
Table (8):
Shows the causes of maternal death between the two groups:
PPH: 33.3% in GMPs, while 66.7% in the control group.
pulmonary embolism : all the cases were in the control group.
This difference is statistically not significant. P > 0.05.
Figure (13):
Shows the relation between parity and fetal outcome.
89.9% of the GMPs had alive babies, while 90.0% of the control had.
10.1% of the GMPs had still birth babies, while 10.0% of the control had.
This difference is statistically not significant. P > 0.05.
Table (8):
Shows the babies`s apgar score in the two groups.
1.6% of the GMP babies had apgar score 4 to 6 while multiparas had 3.4%.
1.6% of the GMP babies had apgar score less than 3, while multiparas had 0.8%.
This difference is statistically not significant. P > 0.05.
Table (9):
Compare the condition of the babies after birth between two groups
3.2% of the babies of the GMP were distressed, while 4.2% of the multiparas`s babies were.
This difference is statistically not significant. P > 0.05.
Table (11):
Shows the fate of the distressed babies`after delivery in the two groups.
resuscitated and beside their mothers: 12.5% in the GMP, while 87.5% were in the control group.
admitted to SCBU: 42.9% in the GMP, while 57.1% were in the control group.
This difference is statistically not significant. P > 0.05.

Table (12):
Compare the fresh and macerated still birth between two groups.
Fresh still birth: 25.0% were babies of GMP, while 75.0% of control group.
macerated still birth: 40.9% were babies of GMP, while 59.1% of control group.
This difference is statistically not significant. P > 0.05.
Figure (15):
Shows the relation between parity and birth weight.
7.2% of cases delivered baby weight less than 2500 gm compared to 11.0% of control group.
73.9% of cases delivered babies with weight between 2500gms and 4000gms, compared to 83.1% of control group.
18.8% of cases delivered babies with weight more than4000 gm, compared to 5.9% of control group.
This difference is statistically significant. P < 0.05

Table (1):Showing the distribution of the cases and control groups by their ages

			Age group			
			<20	20-35	>35	Total
Study population	Cases	Count	1	92	57	150
		%	.7%	61.3%	38.0%	100.0%
	Controls	Count	11	262	27	300
		%	3.7%	87.3%	9.0%	100.0%
	Total	Count	12	354	84	450
		%	2.7%	78.7%	18.7%	100.0%

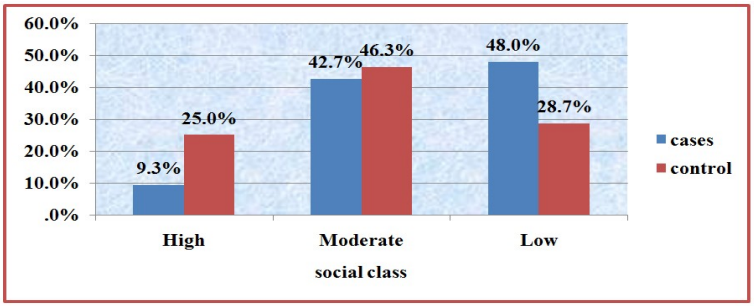


Fig (1) showing distribution according to socioeconomic class

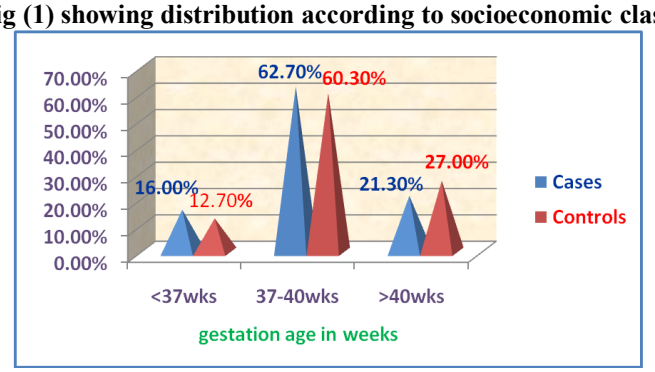
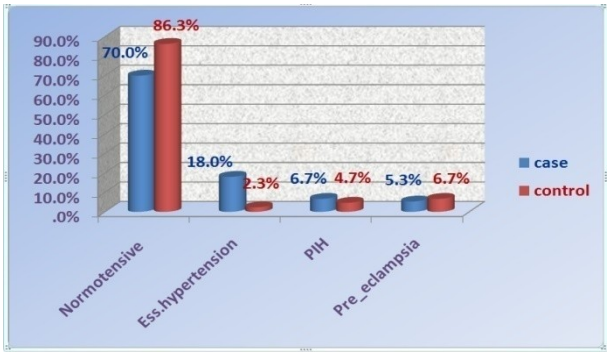
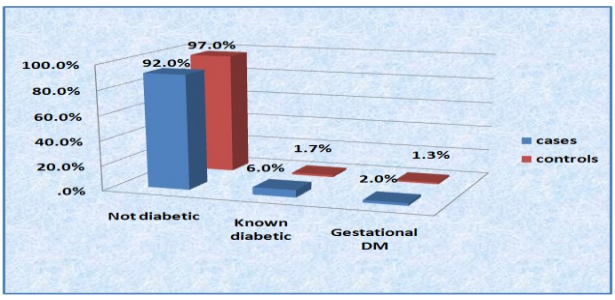


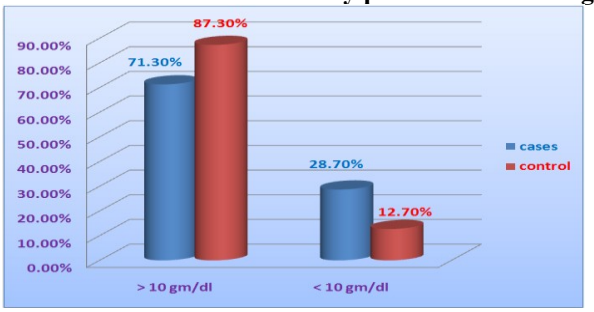
Fig (2) showing the relation between parity and gestational age



Figure(3) Showing the correlation between parity and hypertensive disorders of pregnancy.



Figure(4) Showing the distribution of cases and control by presence of DM or gestational DM.



Figure(5) Showing the distribution of cases and control by the presence of anaemia or not.

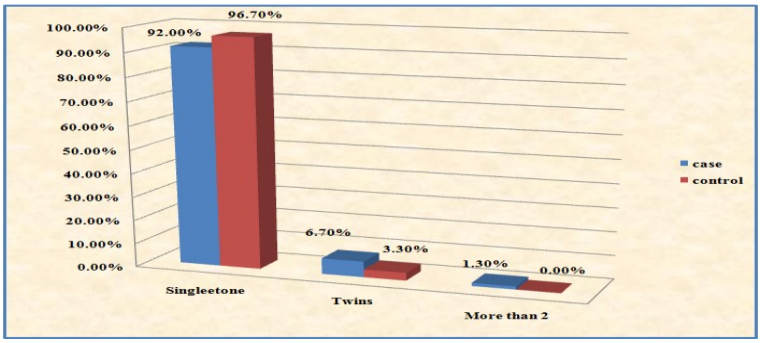


Figure (6): Shows the relation between parity and multiple pregnancy.

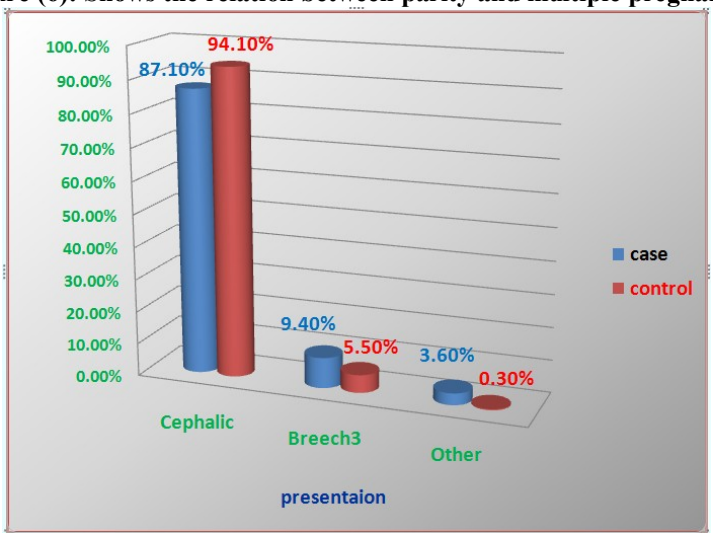


Figure (7) Showing the correlation between parity and presentation.

Table(2): showing transverse lie and face presentation

.		Cases	Controls	Total
Transvese lie	Count	4	1	5
	%	80.0%	20.0%	100.0%
face	Count	1	0	1
	%	100.0%	.0%	100.0%
Total	Count	5	1	6
	%	83.3%	16.7%	100.0%

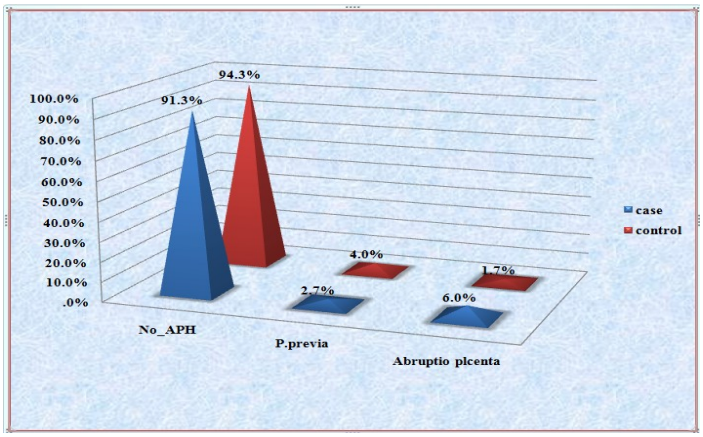


Figure (8) Shows the distribution of the cases and control group according to antipartum hemorrhage (APH).

Table(3): Showing the relation between parity and onset of labour

			Onest_of_labor		
			Spontaneous	Induced	Total
Study population	Cases	Count	147	3	150
		%	98.0%	2.0%	100.0%
	Controls	Count	267	33	300
		%	89.0%	11.0%	100.0%
	Total	Count	414	36	450
		%	92.0%	8.0%	100.0%

Table(4) showing the duration of the first stage of labour

			Duration_of_first_stage			
			<2hours	2-6 hours	>6 hours	Total
Study population	Cases	Count	27	91	32	150
		%	18.0%	60.7%	21.3%	100.0%
	Controls	Count	23	134	143	300
		%	7.7%	44.7%	47.7%	100.0%
	Total	Count	50	225	175	450
		%	11.1%	50.0%	38.9%	100.0%

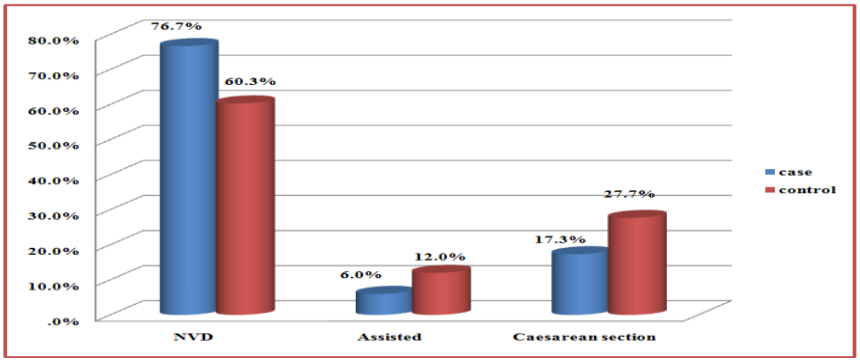


Figure (9) Showing the distribution of cases and control groups by the mode of delivery.

Table (5): compare the the indications of emergency C\S in the two groups

		Cases	Controls	Total
Failure to progress	Count	4	40	44
	%	9.1%	90.9%	100.0%
Fetal distress	Count	3	8	11
	%	27.3%	72.7%	100.0%
Obstructed labour	Count	1	6	7
	%	14.3%	85.7%	100.0%
Abnormal lie	Count	5	1	6
	%	83.3%	16.7%	100.0%
APH	Count	8	17	25
	%	32.0%	68.0%	100.0%
PE	Count	1	5	6
	%	16.7%	83.3%	100.0%
0thers	Count	4	6	10
	%	40.0%	60.0%	100.0%
Total	Count	26	83	109

		Cases	Controls	Total
Failure to progress	Count	4	40	44
	%	9.1%	90.9%	100.0%
Fetal distress	Count	3	8	11
	%	27.3%	72.7%	100.0%
Obstructed labour	Count	1	6	7
	%	14.3%	85.7%	100.0%
Abnormal lie	Count	5	1	6
	%	83.3%	16.7%	100.0%
APH	Count	8	17	25
	%	32.0%	68.0%	100.0%
PE	Count	1	5	6
	%	16.7%	83.3%	100.0%
0thers	Count	4	6	10
	%	40.0%	60.0%	100.0%
Total	Count	26	83	109
	%	23.9%	76.1%	100.0%

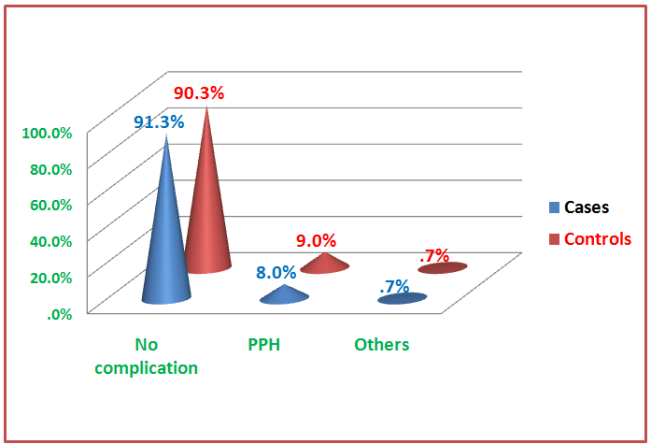


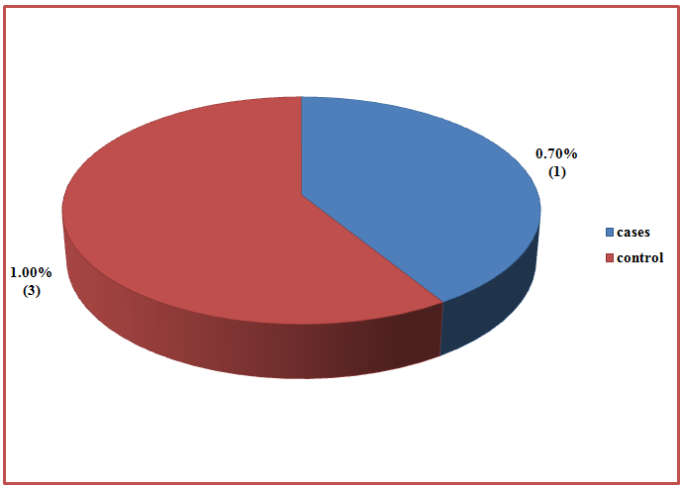
Figure (10) Showing the distribution of the cases and control group by presence of PPH.

Table(6): Compare the causes of PPH in the two groups

		Cases	Controls	Total
Uterine atony	Count	6	2	8
	%	75.0%	25.0%	100.0%
Gentital tract injury	Count	4	17	21
	%	19.0%	81.0%	100.0%
Retained product	Count	1	6	7
	%	14.3%	85.7%	100.0%
Blood coagulpathy	Count	1	2	3
	%	33.3%	66.7%	100.0%
Total	Count	12	27	39
	%	30.8%	69.2%	100.0%

Table(7): showing types of genital tracts injuries

			Cases	Controls	Total
Types of genital tract	Ruptured uterus	Count	1	4	5
		%	20.0%	80.0%	100.0%
	Perineal tear	Count	1	9	10
		%	10.0%	90.0%	100.0%
	Extended episiotomy	Count	1	3	4
		%	25.0%	75.0%	100.0%
	Others	Count	1	1	2
		%	50.0%	50.0%	100.0%
	Total	Count	4	17	21
		%	19.0%	81.0%	100.0%



Figure(11) Showing the relation between parity and maternal death.

Table(8) showing the causes of maternal death

			Cases	Controls	Total
Causes of maternal deaths	PPH	Count	1	2	3
		%	33.3%	66.7%	100.0%
	P- embolism	Count	0	1	1
		%	.0%	100.0%	100.0%
	Total	Count	1	3	4
		%	25.0%	75.0%	100.0%

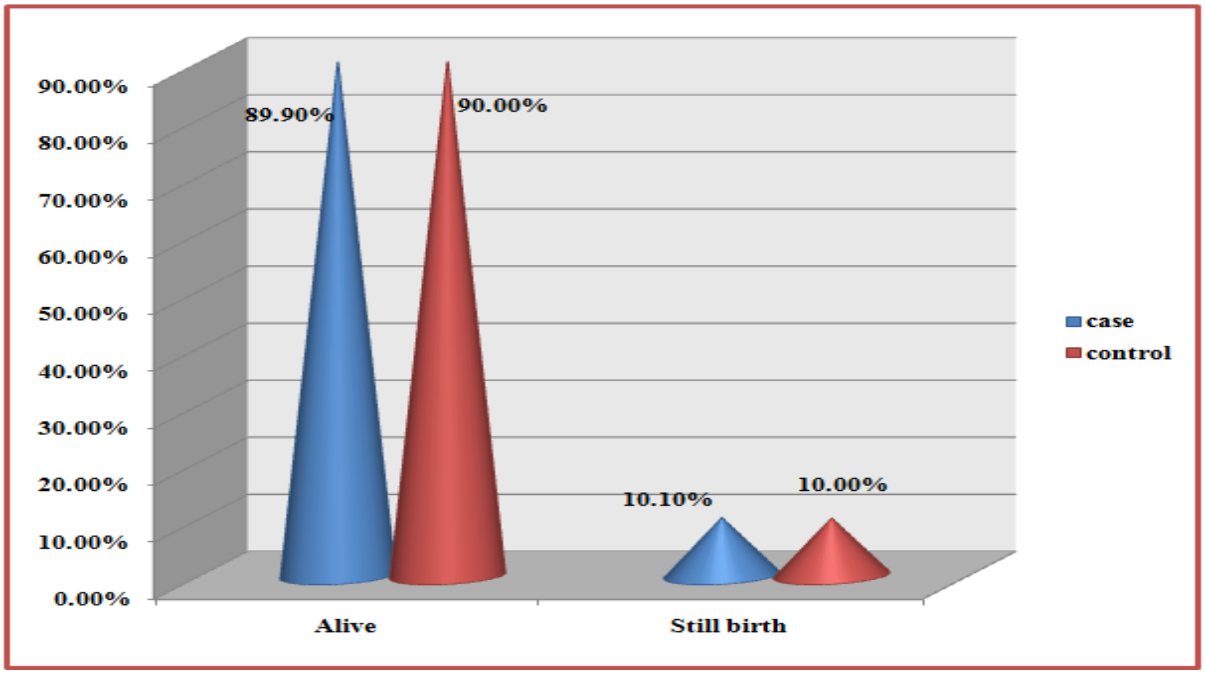


Figure (12) Showing the relation between parity and fetal outcome.

Table (9) Showing the babies' apgar score in the two groups

			Apgar score			
			>=7	4to6	<=3	Total
Study population	Cases	Count	120	2	2	124
		%	96.8%	1.6%	1.6%	100.0%
	Controls	Count	250	9	2	261
		%	95.8%	3.4%	.8%	100.0%
	Total	Count	370	11	4	385
		%	96.1%	2.9%	1.0%	100.0%

Table(10) Compare the condition of the babies after birth between two groups

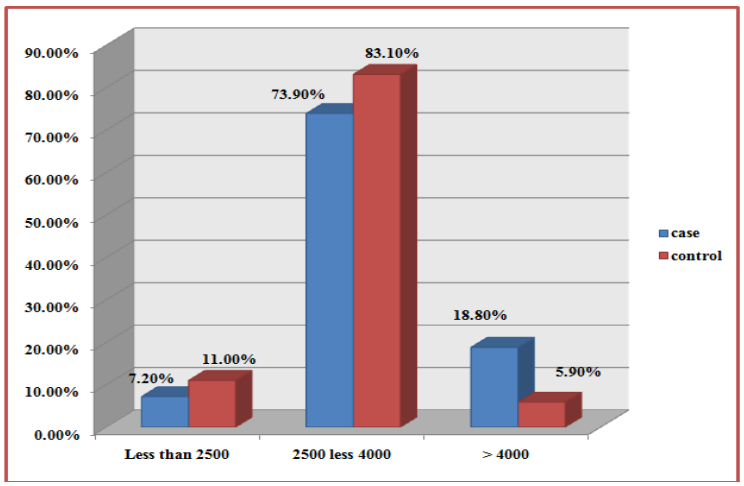
			Not distressed	Baby distressed	Total
Study population	Cases	Count	120	4	124
		%	96.8%	3.2%	100.0%
	Controls	Count	250	11	261
		%	95.8%	4.2%	100.0%
	Total	Count	370	15	385
		%	96.1%	3.9%	100.0%

Table (11) Showing the fate of the distressed babies`after delivery in the two groups

		Cases	Controls	Total
Resuscitated and beside his mother	Count	1	7	8
	%	12.5%	87.5%	100.0%
Admitted to SCBU	Count	3	4	7
	%	42.9%	57.1%	100.0%
Total	Count	4	11	15
	%	26.7%	73.3%	100.0%

Table (12) Compare the fresh and macerated still birth between two groups

			Cases	Controls	Total
Still births	Fresh still birth	Count	5	15	20
		%	25.0%	75.0%	100.0%
	Macerated still birth	Count	9	13	22
		%	40.9%	59.1%	100.0%
	Total	Count	14	28	42
		%	33.3%	66.7%	100.0%



Figure(13) Showing the relation between parity and birth weight.

VII. Discussion

Our aim in this study is to identify the common obstetrical problems of the grandmultipara (GMP), comparing it with multiparous woman.

In the age distribution of the cases, there were 38% above 35 years, while only 9.0% in the control highly significant (p=0.00). Advanced maternal age is associated with increase incidence of maternal complication like hypertension, diabetes mellitus, and high incidence of prenatal complication. This goes with Hench et al [(11)] who found that the age of GMPs were significantly higher compared with control group which may explain the higher incidence of antenatal medical complication, but Maymon et al[(13)] state that higher birth order remained an independent risk factor for peripartum complication after adjustment for maternal age.

Regarding socioeconomic status 48% of the cases were of low social class compared with 28.7% of the control which is statistically highly significant (p=0.00); this explain the increase in the incidence of anaemia among cases. Mor-Yosef et al[(18)] found that the prenatal mortality and low birth weight were in correlation with low socioeconomic status but not with grandmultiparity. Another study by Bugg et al [(9)] concludes that high socioeconomic backgrounds are not prerequisite for favorable result.

Hypertensive disorder of pregnancy are found to be 30.0% in GMP compared to 13.7% in control group which is statistically highly significant (p=0.00), the essential hypertension was the commonest form of hypertensive disorder in the cases. This is explained by increased age of this group; the same finding as Vehaskari et al [(12),] Maymon et a![(13)] ,and Al-Sibia, et al[(14)].

Regarding DM and gestational DM 8.0% of the cases were found to have it , while 3.0% of the control have. It is statistically significant (p=0.03), the higher incidence in GMP may be related to the age of the cases and this may explain the higher incidence of macrocosmic babies in this group, This agrees with Mwambingu FT retrospective studies [(42)] in which there were higher incidence of DM, gestational DM and macrosomic babies in GMP. But Eidelman et al [(4)] found no increase in the incidence of DM in GMP.

Anaemia was found to be 28.7% in GMPs group while 12.7% in the control which is statistically highly significant (p=0.00). The increased incidence reflect the low social class of GMP as well as short pregnancy interval this finding agree with most of the studies like Ogedengbe et al[(10)], Bugg et al[(9)] and Al-Sibai et al[(14)].

The incidence of multiple pregnancy was 8.0% in GMPs.group compared to 3.3% in the multiplarous group which is statistically significant (p=0.03). Vehaskari et al[(12)] found that no significant difference in the rate of multiple birth in GMPs.

The incidence of breech presentation was found to be 9.4% in the GMPs compared to 5.5% in the control which statistically, is highly significant (p=0.00), while transverse lie (3.6%) in cases vs. 0.3% in the control group. This may be explained by the laxity of the abdominal muscle of the GMPs. This finding agrees

with studies done by Babiniski et al[8], Sibai et al[14], Karl et al[22] while Vehaskari et al[12] found no significant increase of the incidence of breech presentation.

Preterm labour is 16.0% in cases and 12.7% in control groups which statistically is not significant. Resnick et al[11] found no significant difference in the incidence of preterm labour among study groups while Sabai et al[14] found increase incidence of preterm labour in GMPs group.

Regarding the relation between GMPs & APH, the incidence of 39bruption placentae was 6.0% in GMPs , vs. 1.7% in control group. It is statistically significant ($p<0.05$). This may be explained by increased incidence of hypertensive disorder and increased age in the GMPs group also it may be explained by increased incidence of anaemia especially due to folic acid deficiency. While the incidence of placenta previa is 2.7% in GMPs compared to 4.0% in the control group, Which is statistically significant ($p<0.05$), this may be explained by the occurrence gynaecological problems like abortions and evacuations , past history of placenta previa, also the GMPs had higher incidence of multiple pregnancy, abnormal lie and presentations . Verhaskari et al[12] found the frequency of 39bruption placentae, placenta previa and retained placenta were significantly higher in GMPs, 39bruption placentae is affected by both high parity and age, placenta previa is independent of age. Another study in Sudan done by Aziz FA found that there is higher rate of APH in GMPs group[32] Abu-heja et al[17] found in his study that there were no difference in the incidence of placental 39bruption and placenta previa among study group.

Regarding induction of labour there is a decreased incidence in GMPs 2.0% vs 11.0% it is highly significant ($p=0.00$). This may be explained by most of our protocols in hospital takes GMPs as relative contraindication for induction of labour especially (Prostaglandin).

Comparing the mode of deliveries between two groups we found that SVD is mode of delivery of most GMP 76.7% vs. 60.3% which statistically is highly significant ($p=0.02$).There was significant decrease in the incidence of assisted vaginal delivery 6.0% vs. 12.0% in control group. Em C/S was found to be reduced in GMP 17.3 vs. 22.3% in the control group which statistically is highly significant ($p=0.02$),

in indications of emergency caesarean section we found that:

C|S due to failure to progress: 9.1% in GMPs while 90.9% were in the control group.
C|S due to fetal distress: 27.3% in GMPs while 72.7% were in the control group.
C|S due to obstructed labour: 14.3% in GMPs while 85.7% were in the control group.
C|S due to abnormal lie: 83.3% in GMPs while 16.7% were in the control group.
C|S due to APH: 32.0% in GMPs while 68.0% were in the control group.
C|S due to PE: 16.7% in GMPs while 83.3% were in the control group.
C|S due to others causes: 40.0% in GMPs while 60.0% were in the control group.
This difference is statistically significant P =003.

All this may be explained by tight perineum, anaemia in GMPs, sizable babies abnormal lie.

Vehaskari et al [12] found that the incidence of operative delivery was roughly similar in GMPs and control group while C/S is lower among GMP. Another study Sipila P[19] stated that GMPs had fewer C/S and vacuum extractor but more induction of labour than mother with low parity, also Karl et al[22] found increase of rate of forceps delivery and C/S rate among GMP. In Lagos study was done and found increase incidence of obstructed labor in GMPs[10].

The overall incidence of PPH was slightly less in GMP 8.7% vs 9.7% in the control group, but according to the causes, it was found that:

Uterine atony: 75.0% in GMPs, while 25.0% in the control group.
Genital tract injury : 19.0% in GMPs, while 81.0% in the control group.
Retained products : 14.3% in GMPs, while 85.7% in the control group.
Blood coagulpathy : 33.3% in GMPs, while 66.7% in the control group.
This difference is statistically significant. $P < 0.05$.

The main cause of PPH in GMPs was uterine atonia, On the other hand, the most common cause in multiparas were genital tract injuries, this may be explained by tight perineum, so the overall incidence of PPH is higher in control group. This agree with Babinzki et al(8). And the study done in Nigeria[28]. But another study done at a Wallowa university hospital showed no significant association between GMP and PPH but related to prolong 2nd and 3rd stage of labor and nonuse of oxytocics after vaginal delivery[30].Therefore more studies needs to be done after adjusting these variables.

If we look at the causes of Genital tract injures in, we found that:

Ruptured uterus : 20.0% in the GMPs, while 80.0% in the control group. 25.0% of perineal tear: 10.0% in the GMPs, while 90.0% in the control group.

Extended episiotomy: 25.0% in the GMPs, while 75.0% in the control group.
Other causes: 50.0% in the GMPs, while 50.0% in the control group.
This difference is statistically not significant. $P > 0.05$.

Comparison of maternal mortality between GMPS and multiparous women (safe parity) we found that: 1 maternal death in GMPs and 3inmultiparas (0.7% vs1.0%).Total maternal death rate 888.9\100000, while GMPs death rate 666.7\100000, and Multiparas death rate 1000\100000.MMR was higher in the control group, this reflects the increase incidence in the so called safe parity. Most of these deaths occurred during the delivery. This may be explained by increase in labour ward complication like obstructed labour, assisted vaginal delivery, Genital tract injures, Em C/S and bleeding from placenta previa in the control group. Also due to decreased attention by doctors in this group and decreased anticipation of complication, unlike in GMPs which considered as dangerous. This finding agrees with Ogedengbe et al[10] who found maternal mortality rate 44,4/1000 amongst GMPs which is not significantly difference from general obstetrical population but two studies Vehaskari in Finland and the other Mikulondra F in Tunis found GMP had higher incidence of maternal mortality[12,35].

In maternal deaths due to PPH, we found that 33.3% in GMP, and 66.7% in multiparas.
In maternal deaths due to Pulmonary embolism, we found that no cases in GMP, and all cases were occur in multiparas.

Regarding fetal outcome, condition of the babies after birth, and the fate of the distressed babies we found that: total still birth rate 98.1\1000, GMP still birth rate 101.4\1000, and multiparas still birth rate 96.6\1000, (10.1% vs 10.0%). This difference is statistically not significant. 1.6% of the GMP babies had apgar score 4 to 6 while multiparas had 3.3%. 1.6% of the GMP babies had apgar score less than 3, while multiparas had 0.8%. This difference is statistically not significant. 3.2% of the babies of the GMP were distressed, while 4.2% of the multiparas`s babies were.This difference is statistically not significant.
In the distressed babies we found that :
those resuscitated and beside their mothers: 12.5% were babies of the GMP, while 87.5% of the multiparas.
Those admitted to SCBU: 42.9% were babies of the GMP, while 57.1% of the multiparas.
This difference is statistically not significant.
Regarding the stillbirth we found that:
Fresh still birth babies, 25.0% of them from babies of the GMP, while 75.0% from control grup.
Macerated still birth, 40.9% of them from babies of the GMP, while 59.1% from control group.
This difference is statistically not significant.

Macerated still birth is common in GMPs this may be explained by being poor, anaemic, hypertensive, no antenatal follow up, and delayed at home hoping for home delivery. While fresh still birth is common in multiparas because of prolonged labour, increased obstructed labour, assisted, and operative delivery, also the believe that this is the safe parity so the team become relaxed, and this may reflect poor fetal monitoring in the labour ward, and the resuscitation team and facilities are not optimal.
Eidelman et al[4] found that there is no difference between GMP and less parous ladies in still birth rate. This agree with G. J. Bugg et al. who found No significant differences in neonatal outcomes between the two groups[9]. MikulondraF, found the babies of GMPs group were severely asphyxiated at birth more frequently than those in the control group.'[36] A Babinski, T Kerenyi found that low apgar scores significantly higher in grand multiparas than in multiparas although perinatal mortality remains low in these patients[8].

Fetal macrosomia is another risk factor which is frequently found in GMPs, regarding the relationship between birth weight and parity, Macrosomic babies are significantly higher in GMPs groups (18.8% vs 5.9% in the control group). This may be explained by obesity, increased, maternal age and high incidence of DM in GMP group. This agrees with studies of Babiniski et al[8] and Heija et al[17],
on the other hand, the incidence of LBW is higher among the control group. (7.2%in cases while 11.0% in the control group). The difference is statistically significant. This agreed with Kaplan B et al, and Eidelman AI et al who found lower rates of LBW infants among GMPs[11] [4]. This may be explained by that the women in this group –control group- are younger ,less obese, and low rates of diabetes mellitus among them.

Nordin N.M. in his study Is grandmultiparity a significant risk factor in this new millennium, there was no significant difference in diabetes and glucose intolerance, ante partum and post partum hemorrhage. There was a significantly lower risk of first and second-degree perineal tear, and prolonged first stage of labor. There was a significant increased in induction of labor but there was no uterine rupture and no increased in Cesarean Section. There was an increased in meconium stain liquor but there was no increased risk of fetal distress. The fetal outcome was good and there was no tendency to macrosomic infants or shoulder dystocia. With adequate care, the maternal fetal outcome of grandmutiparous women is good and comparable to the multiparous women. Anemia is still common and patient education is important to overcome this problem[45].

VIII. Recommendations

Most GMPs were of older age and poor socio-economic status so improvement in social class, health education, use of contraception and good antenatal and intrapartum monitoring are needed.

Careful monitoring and anticipation of PPH in GMP with active Management of 3rd stage of labour, and this should be the routine practice.
Efficient care for obstetric complications and effective referral system.
Resuscitation of babies should be done by well trained doctors with adequate facilities, and in suitable time.

Most of the maternal deaths occur during the delivery, so labour and delivery room should be covered by consultant on duty not on call only, and by well trained doctors, sisters and midwifes.
Excellent maternal and perinatal outcome is possible in grand multiparas with improvement of health care system. by doing so GMP can no longer be considered high risk.
Safe parity group should have more attention by doctors and labour ward staff, through increase anticipation of complications, so as to improve the outcome in this group.

IX. Conclusion

Grand Multiparous women still had some complication mainly antenatal; they had significantly higher incidence of hypertension anaemia, DM, multiple pregnancy, malpresentation, and APH.
Most of GMP delivered normally with less use of instrumental delivery, Em C/S and overall less complicated labour.

The GMP delivered significantly macrosomic babies with significantly higher incidence of MSB.
Maternal deaths, Emergency caesarean section, PPH, and instrumental deliveries, genital tract injuries are increased in multiparas group, which was so called safe parity.

Acknowledgement

I would like to express my sincere gratitude to my supervisor:
Dr: *Bashier G. M. ElImam* .
Consultant Obstetrician and Gynecologist Soba University Hospital
Vice dean for Academic Affair Faculty of Medicine University of Khartoum
For his advices, valuable suggestions, and help.
I also thank Dr: *A. A. Shiddo* and Prof: *Taha Umbali* for their valuable advices.
I extend my thanks to Dr: *Isamaldin M. Abdulla* (community board), and Dr: *Babikier M. Ali* (MD community) M.O.H for their supports.
My best regards to my colleagues doctors, midwifes and nurses who helped me a lot during collecting the data, and finally thanks a lot to my patients who accepted to participate in this study .

References

[1]. B Solomons, the dangerous multgipara. Lancet 2 (1934), pp 8-11.
[2]. PA King, SJ Duthie and HK Ma, Grandmultiparity: a reappraisal of risks. Int. J Gynaecol Obstet 36 (1994), pp 13-16.
[3]. DS Seidman, Y Armor, D Roff, DK Stevenson and R Gale , Grand multiparity: an obstetric or neonatal risk factor?. *Am J Obstet Gynecol* 158 (1988), pp. 1034–1039. View Record in Scopus | Cited By in Scopus (25).
[4]. A Eidelman, R Kamar, MS Schimmel and B Eichanan , The grandmultipara: is she still at risk?. *Am J Obstet Gynecol* 158 (1988), pp. 389–392. View Record in Scopus | Cited By in Scopus (29).
[5]. K Fuchs, BA Peretz, R Marcovic and I Timor-Tritsch , The grandmultipara: is it a problem? A review of 5,785 cases. *Int J Gynaecol Obstet* 23 (1985), pp. 321–325. View Record in Scopus | Cited By in Scopus (40).
[6]. A Samueloff, S Mor-Yosef, D Seidman, R Rabinowitz, A Simon and J Schenker , Grand multiparity: a nationwide survey. *Isr J Med Sci* 25 (1989), pp. 625–629. View Record in Scopus | Cited By in Scopus (18).
[7]. Toohey JS, Keegan KA, Morgan MA, Francis J, Task S, deVeciana M. The "dangerous multipara": fact or fiction? Am J Obstet Gynecol 1995;172:683–6. Buy Now [Context Link].
[8]. Babinszki A, Kerenyi T, Torok O, Grazi V, Lapinski RH, Berkowitz RL. Perinatal outcome in grand and great-grand multiparity: effects of parity on obstetric risk factors. Am J Obstet Gynecol 1999;181:669–74. Buy Now [Context Link].
[9]. Bugg GJ, Atwal GS, Maresh M. Grandmultiparae in a modern setting. Br J Obstet Gynaecol 2002;109:249–53. [Context Link]
[10]. Ogedengbe OK, Ogunmokun AA Grandmultiparity in Lagos, Nigeria. Niger Postgrad Med J. 2003 Dec; 10(4):216-9.
[11]. Goldman GA, Kaplan B, Neri A, Hecht-Resnick R, Harel L, Ovadia J. The grand multipara. Eur J Obstet Gynecol Reprod Biol 1995;61:105–9. [Context Link].
[12]. Vehaskari A, Lahtinen J, Terho J Hazards of grand multiparity,: Ann Chir Gynaecol Fenn. 1968;57(4):476-84.
[13]. Maymon E, Ghezzi F, Shoham-Vardi I, Hershkowitz R, Franchi M, KatzM, Mazor M. Peripartum complications in grand multiparous women: para 6-9 versus para > or=10.: Eur J Obstet Gynecol Reprod Biol. 1998 Oct;81(l):21-5.
[14]. Al-sibai MH, Rahman MS, Rahman J. Obstetric problems in the grand multipara: a clinical study of 1330 cases. Obstetric problems in the grand multipara: a clinical study of 1330 cases. J Obstet Gynaecol (Lahore). 1987;8(2):135-8.
[15]. Ujah~IA,Uqru-VA The trend of MMRin NigeriaEast Africa Med j. Aug:76[8],436-9.
[16]. Rizk DE, Khalfan M, Ezimokhai M. Obstetric outcome in grand multipara in the United Arab Emirates: a case control study. Arch Gynecol Obstet 2001;264:194–8. [Context Link].
[17]. Abu-Heija AT, Chalabi HE. Great grand multiparity: is it a risk? Int J Gynaecol Obstet 1997;59:213–6. [Context Link]
[18]. Mor-Yosef S, Seidman DS, Samueloff A, Schenker JG. The effects of the socioeconomic status on the perinatal outcome of grand multipara. Eur J Obstet Gynecol Reprod Biol 1990;36:117–23. [Context Link]
[19]. Sipila P, von Wendt L, Hartikainen-Sorri AL.
[20]. The grand multipara—still an obstetrical challenge? Arch Gynecol Obstet. 1990:247(4); 187-95.

[21]. Shah P.S. Parity and low birth weight and preterm birth: A systematic review and meta-analyses (2010) *Acta Obstetricia et Gynecologica Scandinavica*, 89 (7), pp. 862-875.
[22]. Edith D. Gurewitsch MD, Elizabeth Johnson MS, Robert H. Allen PhD, Paul Diament PhD, Jill Fong MD, Daniel Weinstein MD, PhD and Frank A. Chervenak MD The descent curve of the grand multiparous woman.
[23]. Karl Fuchs , Bezalel-Alik Peretz , Rami Marcovici, Eitan Paldi and llan Timor-Tritsh The "Grand Multipara" — Is it a problem? Int J Gynaecol Obstet. 1985 Sep;23(4):321-6.
[24]. obstetric ten teachers .edited by Geoffrey V P Chamberlain .sixteen edition . P247.
[25]. AkuIkumaran The effect of parity on uterine activity in labour. British journal of Obstet and Gyne September J984-vol.91p.843-848.
[26]. Dewhurst textbook of Obstetrics and Gynaecology, for postgraduates. edited by D. Keith Edmonds. Sixth edition 1999. P166-134-330.
[27]. an evidence -based text for MRCOG .edited by David M . Luesley ,Philip N - Baker 2004. P131.
[28]. Selo-Ojeme DO ; Okonofua FE the risk factors for primary PPH A case control study Arch Gyecol obstet 1997;259[4] : 179-87.
[29]. Adetero. pregnancy and PPH in Nigerian women . West Africa J. Med .1992 jul -sep; U [3]; 172-8.
[30]. Vedat -A ,Hasan-B, Ismail-A. Rupture of uterus in labour . 1st J.M.Sci 1993-oct ;29[I0];634-43.
[31]. Selo-Ojeme DO ; Okonofua FE the risk factors for primary P.PH A case control study Arch Gyecol obstet 1997;259[4] : 179-87.
[32]. Mwambingu FT; AI Meshari AA, Akiel A. : The problem of grandmultiparity in current obstetric practice. Int J Gynaecol Obstet 1988 Jun;26(3):355-9.
[33]. Aziz FA. Pregnancy and labor of grand mult iparous Sudanese women. Int J Gynaecol Obstet. 1980 Sep-Oct;18(2):144-6.
[34]. 33- Simonsen SM, Lyon JL, Alder SC, Varner MW Effect of grand multipariry on intrapartum and newborn complications in young women.Obstet Gynecol. 2005 Sep;106(3):444-5.
[35]. A. Bianco, J. Stone, L. Lynch, R. Lapinski, G. Berkowitz and R.L. Berkowitz, Pregnancy outcome at age 40 and older. Obstet Gynecol 87 (1996), pp. 917-922.
[36]. MikuJandra F; Perisa M; Merlak 1; Kimer M; Sikic D, Jerkovic J Pregnancy, labor and the neonate in pluriparas and multiparas] Jugosl Ginekol Perinatol 1990 May-Aug;30(3-4):83-6 52.
[37]. Mikulondra-F ,Persia-M ,Mertak-I GMP-an obstetric problem. Zentrabl-gynecol 1992,114[10]:491-6.
[38]. Tsu VD Postpartum haemorrhage in Zimbabwe : a risk factors analysis Br J Obstet Gynaecol 1993 Apr; I00[4] :327-33.
[39]. Sudan census 2008.
[40]. Tanbo TG, Bungum L. The grand multipara: maternal and neonatal complications. Acta Obstet Gynecol Scand 1987;66:53–6. [Context Link].
[41]. Bai J, Wong FW, Bauman A, Mohsin M. Parity and pregnancy outcomes. Am J Obstet Gynecol 2002;186:274–8. Buy Now [Context Link].
[42]. Roopnarinesingh S, Ramsewak S, Reddy S Complications of grand multiparity. West Indian Med J. 1988 Dec;37(4);222-5.
[43]. Pregnancy complications of gradmultiparity at a rural setting of South Africa Hoque, M., Hoque, E., Kader, S.B. *Iranian Journal of Reproductive Medicine* 6 (1), pp. 25-31-2008.
[44]. Grandmultiparas in modern obstetrics (2006) *American Journal of Perinatology*, 23 (6), pp. 345-349.
[45]. Iftikhar S., Shams-Un-Nisa, Sheikh M.A., Malik A.M. Fetomaternal outcome in women with grandmultiparity and multiparity (2010) *Medical Forum Monthly*, 21 (4), pp. 50-54.
[46]. Nordin N.M., Fen C.K., Isa S., Symonds E.M. Is grandmultiparity a significant risk factor in this new millennium? (2006) *Malaysian Journal of Medical Sciences*, 13 (2), pp. 52-60.

IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861. Volume 9, Issue 6 (Sep.- Oct. 2013), PP 43-47
www.iosrjournals.org

Pure Honey a potent fertility booster: *Activities of Honey on sperm parameters in young adult rats.*

[1,2]Igbokwe V.U., [1]Gege-Adebayo G.I., and Ogbadu Samuel
Department of Physiology, College of Medicine, Bingham University, Karu, Nasarawa State
Department of Physiology, College of Medicine, Usmanu Danfodiyo University Sokoto, Sokoto State

Abstract: *Honey is a natural product of bees containing numerous nutrients that have significant benefits to human race. These benefits stretch across from its antiseptic, antibacterial, and wound healing properties to its possible sex boosting properties. Due to environmental, nutritional, behavioral and physiological factors, male infertility has become an ubiquitous issue over the world.*

This study therefore investigates the effect of honey on some sperm parameters in rats. Eighteen adult male rats weighing 130g-165g were used for this study. They were randomly divided into three groups with group A serving as the control; group B served as the standard group treated with a standard drug (622mg/kg); and group C was treated with honey (1ml of honey per 100g of body weight). They were treated for 65 days thereafter, the sperm count, motility, and morphology were evaluated.

The mean value of the sperm count of group C rats (130.5±7.50 x10^6/ml) was significantly higher than the group B rats (104.3±3.48 x10^6/ml) and the group A rats (93.5± x10^6/ml) at $p<0.05$. The sperm motility of group C animals, 85 ± 5.0%, was significantly higher ($p<0.05$) when compared to group B animals, 33.3 ± 3.33%, and group A animals, 75 ± 5.0%. The percentage of abnormal sperm was reduced in group C rats (5%) as compared to group B (8.3%) and group A rats (10%). This study showed that honey increases the sperm count, the sperm motility and improves the sperm morphology. Thus it serves as a potential fertility booster in experimental animals.

Keywords*: Fertility, Honey, Sperm morphology, Sperm motility, Sperm count,*

I. Introduction

Reproduction and childbirth are part of the central themes of life, occurring when planned and wished in many but not all cases (Johansson *et al.,* 2011). The prevalence of infertility in men varies between countries mainly due to variations in lifestyle factors, the time interval between the age where sexuality is initiated and the age where first pregnancy is desired, and of course, the prevalence of sexually transmitted diseases (STDs) (Nygren and Zegers-Hochschild, 2008). Boivin *et al., (*2007) following their study of world population reported that 72.4 million people were infertile and of these 40.5 million people were seeking infertility medical care. Female factors were responsible for 35% of infertile cases, while the male factors accounted for 30% of infertile cases. Both the male and female factors were responsible for 20% of infertile cases and unexplained causes of infertility accounts for 15% of cases (Rubenstein and Brannigan, 2011). Over the years, curative measures have been applied to curb infertility, these measures include the use of herbs (e.g. Alfafa, Kelp, or Mandrakes used by Rachel in the Bible), vitamin supplements, drugs (e.g Metformin, Spermomax, Manix) and medical procedures {e.g. in vitro fertilization and embryo transfer (IVF-ET) and gamete intrafallopian transfer (GIFT)} **(**Carangelo, 2002).

Honey is the natural product of bees (Honey bees) formed from the nectar collected from flowering vegetation (Mahaneem *et al.,* 2010). It is an alkaline forming food and contains ingredients similar to those found in fruits, which become alkaline in the digestive system (Bradley, 2010). From ancient times, honey has been used as both a natural sweetener and a healing agent (National Honey Board, 2012).

Honey is a high nutrient source. It contains sugars such as glucose and fructose, as well as minerals like magnesium, potassium, calcium, sodium chloride, sulphur, iron, zinc, phosphates and vitamins B_1, B_2, C, B_6, B_5 and B_3 (Estevinho *et al.,* 2008; Syazana *et al.,* 2011). In recent years, scientific support is beginning to emerge confirming the beneficial effects of honey on certain medical and surgical conditions. Honey has been shown to have biological properties such as antiseptic, antibacterial and antifungal (Tan *et al.,* 2009), anti-inflammatory and antioxidant properties (Viuda-Martos *et al.,* 2008) and immunomodulatory effects (Mandal and Mandal, 2011). It has also wound healing properties (Green, 1988).

Male reproductive performance, particularly sperm count, can be affected by environmental (Sikka and Wang, 2008), genetic (Skakkebaek *et al.,* 1994), behavioural and physiological factors. Honey, also known as an aphrodisiac, has been reported to increase sperm count, testosterone and libido level (Austin, 2011). It contains glucose and fructose, which may be used by the body to obtain energy, thereby improving sexual virility (Bradley, 2010). According to the British Broadcasting Corporation (BBC), Nigerian honey is raw, un-

pasteurized, and is almost crunchy with sugar crystals. Although large amounts of honey are produced in Nigeria, packaging and distribution are disorganized and the Nigerian honey has not well been researched into. Often the place to buy the best honey is on the side of the road.

Previous work done by Syazana *et al.,* (2011) on the effect of gelam honey on sperm quality and testis of rats show no significant differences in the weight, width and length of testes between the groups, but showed significant increase in sperm count and improvement of sperm motility in the honey treated group than in the control group. Also, work done in Palestine by Abdul-Ghani *et al.,* (2008) on the effect of Palestinian honey on spermatogenesis in rat showed that there was increase in weight of the epididymis and epididymal sperm count by 37% after administration of honey.

The effect of honey on sperm parameters such as sperm count, morphology and motility are further investigated in this study with the Nigerian honey. It is hoped that honey might be a booster of low sperm count and motility and thus of use in treatment of male infertility

II. Materials And Method

a. REAGENTS

Honey was purchased from Wiloff Global Venture, Lagos, Nigeria. Manix herbal capsule from Workhardt Limited, India was also purchased.

b. PREPARATION OF DRUGS

Ten capsules of Manix drug was dissolved in 100ml of distilled water to give a concentrated solution of 6220mg/100ml of drug solution. This was administered to the rats according to their body weights, i.e 1ml/100g bodyweight.

c. METHOD

A total of eighteen adult male wistar rats, (4-6 weeks old) were obtained from the National Institute for Veterinary Research, Plateau State, Nigeria. The rats were housed in cages and kept at the Bingham University Animal house, Nasarawa State, Nigeria. They were fed with standard rat pellet and allowed to acclimatize for a period of two weeks. The duration of the study was 65 days.

d. TREATMENT REGIME

The rats were divided randomly into three groups. These were the control group, the standard group and the honey treated group.

Group 1: This was the control group, it had six rats, and they received a daily dose of 1.0ml/100g body weight of normal saline (0.9%) orally for a period of 65 days according to Syazana *et al.,* (2011). Group 2: The standard group had six rats, and they were given 622 mg/kg body weight of manix for 65 days. Group 3 was the honey treated group. It also contained six rats that received a daily administration of 1.0ml/100g body weight of honey orally for 65 days (Syazana *et al.,* 2011). The body weights were monitored weekly.

e. SPERM COUNT DETERMINATION

Methods of Selmanoğlu *et al.,* (2009) and Mahaneem *et al.,* (2010) were used to analyze sperm count. The cauda epididymis was dissected and minced in 1ml of normal saline (0.9%), then filtered using a nylon mesh. The suspension was then fixed in normal saline and spermatozoa were counted using the Neubauer hematocytometer chamber.

f. SPERM MOTILITY

The method of Biswas *et al.,* (2002) was used with a minor modification. The content of the vas deferens was collected with the aid of a syringe and needle, and a drop was placed on a clean pre-warmed slide (37°c) and covered with a cover slip. The motility was determined by eye estimation of the proportion of spermatozoa moving forward (motile) and those that did not move were considered non-motile.

g. SPERM MORPHOLOGY

A drop of stained sperm suspension was prepared for sperm count. This was done by a smear on a glass slide, air-dried for a maximum of 5 minutes and then visualized microscopically at a magnification of 400X. For each rat, the sperm was screened and the percentage of total abnormalities of heads (such as microcephalus, detached head, flattened head, doubled head and bent neck) and/or tails (such as coiled tail, bent tail and doubled tail) was determined (Narayana *et al.,* 2005; Mahaneem *et al.,* 2010).

III. Statistical Analysis

The student T - test was used to analyze the differences between the three groups. A value of $p<0.05$ was considered statistically significant. Data are presented as mean ± standard error of mean in the result.

IV. Results

The initial mean body weights of groups A, B and C rats were 135.6 ± 5.77g, 138 ± 10.62g and 135.3 ± 8.68g respectively. The final mean body weight (i.e at the end of the experiment) of group C animals was significantly higher (230.2 ± 10.04g) than Group B (228.3 ± 10.54g)^c ($p = 0.14$) and Group A animals (215.7± 3.25g)^c ($p = 0.45$) as shown in table 1.

The sperm count of Group C rats was significantly higher (130.5 ± 7.50 x 10^6/ml) compared to Group A (93.5 ± 2.50 x 10^6/ml) ($p = 0.02$)^a and group B (104.3 ± 3.48 x 10^6/ml) ($p = 0.01$)^a animals. Fig 1

Following 65 days of treatments, the sperm motility of honey treated Group C animals was significantly higher (85 ± 5.00%), compared to manix treated, Group B (33.3 ± 3.33%) ($p = 0.001$)^a and saline treated, Group A (75 ± 5.00%) ($p=0.14$)^c animals. Fig.2. During the analysis it was observed that sperm motility of manix treated rats, Group B, showed significant numbers of pus cells indicating a possible seminal infection by the drug administered to this group. This could have affected the motility.

Also, the sperm morphology (% abnormality) of Group C rats was significantly better (5%) than Group B (8.3%) ($p = 0.11$)^c and Group A (10%) ($p = \infty$) rats. Fig.3

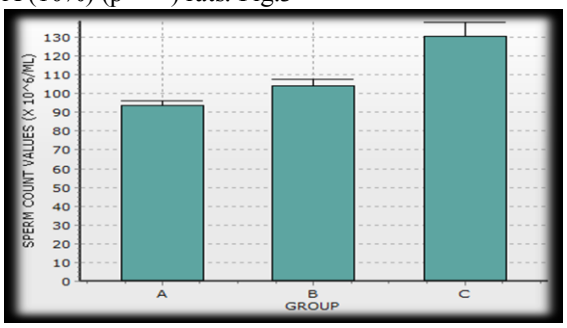


Figure 1: Effect of honey on sperm count. All data are presented as mean ± standard error of mean, p<0.05 considered statistically significant. A= control, B= standard, C= honey-treated.

.

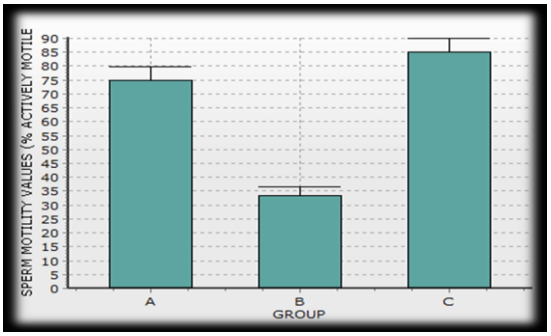


Figure 2: Effect of honey on sperm motility. All data are presented as mean ± standard error of mean, p<0.05 considered statistically significant. A= control, B= standard, C= honey-treated.

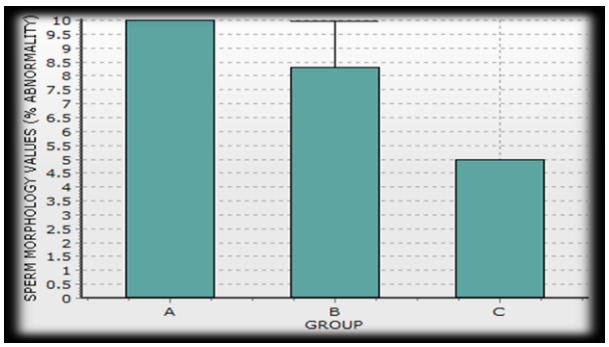


Figure 3: Effect of honey on sperm morphology. All data are presented as mean ± standard error of mean, p<0.05 considered statistically significant. A= control, B= standard, C= honey-treated.

V. Discussion

In this study, it was found that the administration of honey at 1ml per 100g of body weight increases the mean body weight of rats. This increase in weight gain might be due to the fact that honey contains carbohydrates and also has simple sugars (Ajibola *et al.,*2012), instead of complex carbohydrates that break down slowly.

Honey administered at a dose of 1ml per 100g of body weight also caused increases in sperm count, sperm motility and sperm morphology in the experimental rats. Previous works done by Syanza *et al.,* (2011), Mahaneem *et al.,* (2010), and Abdul-Ghahi *et al.,* (2008) on the effect of honey on sperm quality in experimental rats all concur with the results of this study in showing that honey improves the semen quality in experimental rats.

Spermatogenesis is the process of spermatozoa production and occurs within the seminiferous tubules of the testes (Johnson *et al.,* 2000). It is regulated by paracrine and endocrine mechanisms (De Krester *et al.,* 1998). Since spermatogenesis occurs as a result of the stimulation of testosterone (Tesarik *et al.,* 1998), it is probable that the honey has interacted positively with luteinizing hormone, which stimulates the leydig cells to secrete more testosterone, which also explains its effect on libido. Also, honey could have interacted positively with the sertoli cells directly or indirectly through the follicle stimulating hormone to stimulate conversion of spermatids to mature spermatozoa (Syazana *et al.,* 2011).

Abdul-Ghani *et al.,* (2008) reported that honey increased the activities of testicular marker enzymes for spermatogenesis such as sorbitol dehydrogenase by 31% and reduced the activity of lactate dehydrogenase by 48%. Lactate dehydrogenase has been indicated to have increased activity in infertility (Eliasson and Virji, 1985). Sorbitol dehydrogenase is an enzyme in carbohydrate metabolism converting sorbitol, the sugar alcohol form of glucose, into fructose (El-Kabbani *et al.,* 2004). Since fructose is a component of honey and is an important marker in the seminal fluid, honey enhances sperm quality in that fructose provides energy and nutrients for the sperm and the perfect alkaline medium for the sperms to maintain their viability, thus enhancing their motility.

Honey, also known as a novel antioxidant, contains pinocembrin, pinostrobin, vitamins, glucose oxidase, diastase, (Erejuwa *et al.,* 2012). It reduces lipid peroxidation and oxidative stress on the sperm cells by reactive oxygen species like super oxide, hydrogen peroxide, and the likes (Syazana *et al.,* 2011).

Manix is a drug that has been invented to help boost fertility in men. The administration of this drug to the male rats showed that it might be associated with the formation of pus cells and reduced motility following prolonged use. Honey however did not exhibit such trait in its sperm boosting properties following sixty – five days of administration.

VI. Conclusion

This study showed that administration of honey throughout the period of spermatogenesis enhanced sperm quality through improving the sperm parameters of: sperm count, sperm motility and sperm morphology. Thus honey aside from its other numerous effects, also has positive reproductive effects on the male reproductive system**.**

VII. Recommendation

It is suggested that honey could be used in the place of synthetic fertility drugs in boosting fertility and treating infertility cases in men, as it poses no significant side effects. It is also recommended that more studies should be carried out on the precise mechanisms in which honey influences the gonadotropins in improving male gamete production and fertility. Studies should also be carried out on its possible effects on the female reproductive system.

.

Parameters	Control Group	Standard Group	Honey Treated Group	P value
Initial mean body weight (g)	135.6±5.77	138.5±10.62^{c}	135.3±8.68^{c}	<0.05
Final mean body weight (g)	215.7±3.25	228.3±10.54^{c}	230.2±10.04^{c}	<0.05
Weight gain (g)	80.1±2.70	89.8±5.29^{c}	94.9±4.69^{c}	

Table 1. effect of honey on body weight in experimental rats after 65 days. All Data are represented as mean ± standard error of mean, p<0.05 considered statistically significant.^{c} signifies that p>0.05 when compared to control group, meaning there's no significant difference.

Parameter / Group	Sperm count (x 10^{6}/ml)	Sperm motility (% actively motile)	Sperm morphology (% abnormality)	p value
Control	93.5± 2.50	75± 5.00	10±0.00	<0.05
Standard	104.3± 3.48^{a}	33.3± 3.33^{a}	8.3± 1.67^{c}	<0.05
Honey treated	130.5± 7.50^{a}	85± 5.00^{c}	5±0.00^{d}	<0.05

Table 2: effect of honey on sperm count, sperm motility and sperm morphology in experimental rats. All Data are represented as mean ± standard error of mean, p<0.05 considered statistically significant.

Acknowledgement

We acknowledge the Laboratory technologists especally Mr.Joseph Egene for his efforts during the course of the project,Mr.Friday of the animal house,Bingham University.

References

[1] Johansson M, Hellström A, Berg M :Severe male infertility after failed ICSI Treatment-a phenomenological study of men's experiences *Reproductive Health,* :2011., vol 8 iss 4 pg 1

[2] Nygren K G and Zegers-Huchschud F : Documentation of infertility prevalence, treatment access and treatment outcomes in developing countries. *Oxford Journals: ESHRE monographs.* 2008, issue 1, pg 5-7.

[3] Boivin J, Bunting L, Collins J A. and Nygren K G.: International estimates of infertility prevalence and treatment-seeking: potential need and demand for infertility medical care. *Human Reproduction* 2007;Vol.22, No.6 pp. 1506– 1512

[4] Rubeinstein J; Brannigan R.E Male infertility. *Medscape reference* copyright 1994-2012. All rights reserved.

[5] Mahaneem M, Sulaiman S A, Jaafar H 3, Sirajudeen K N S, Ismail Z I M and Islam N M Effect of Honey on Testicular Functions in Rats Exposed to Cigarette Smoke. *Journal of ApiProduct and ApiMedical Science* 2010;vol 3 iss 1: pg 12 - 17

[6] Carangelo L, . Infertility cures (instead of adoption). *Americans for open records.* ©2001, 2002 ISBN# 0-942605-23-3. All rights reserved

[7] Bradley R. Raw Honey and Cinnamon, 2010. © *One 80 Turn* LLC.

[8] National Honey Board (2002) Honey-health and therapeutic qualities. Copyright © 2009 National Honey Board, All Rights Reserved

[9] Estevinho, L.; Pereira, A.; Moreira, L.; Dias, L.; Pereira, E Antioxidant and antimicrobial effects of phenolic compounds extracts of Northeast Portugalhoney. *Food and Chemical Toxicology ;* . 2008:.46: 3774-3779

[10] Syazana N.S., Hashida N.H. , Majid A.M., Durriyyah H.A. Sharifah , Kamaruddin M.Y Effects of Gelam Honey on Sperm Quality and Testis of Rat *Sains Malaysiana.* ;2011: vol 40 iss 11 pg 1243–1246

[11] Tan, H.T; Halim, A.S; Hassan, S.A; Suleiman, S.A; Rahman, R.A.; Gan, S.H; Kirpin-Kaur, B.S.) antibacterial properties of Malaysian tualang honey against wound and enteric microorganisms in comparism to manuka honey.*BMC complementary and Alternative Medicine.* 2009, Vol 9, iss 34.

[12] Viuda-Martos, M; Navajas Y.R; Fernandez-Lopez, J; Perez-Alvarez J.A):Functional properties of honey, propolis and royal jelly. *Journal of Food Science.* 2005 Vol. 73, iss 9.R117-R124

[13] Manley W.: United States Standards for Grades of Extracted Honey. *Agricultural marketing service.* T ,1985,USDA copyrights. Iss 5 (50 FR 15861).

[14] Sikka, S C; Wang, R: Endocrine disruptors and estrogenic effects on male reproductive axis. *Asian Journal of Andrology*,2005.,10**,** 134-1S45.

[15] Skakkebaek, N.E; Giwercman, A; De-krester, D :Pathogenesis and management of male infertility. *The Lancet* 1994,*v343, 8911.*

[16]. Austin, S :Sexual performance natural supplements for boost sexual libido. *Sooper articles:sexual health.* Copyright © 2012 All Rights Reserved.

[17] Abdul-Ghani AS, Dabdoub N, Muhammad R, Abdul-Ghani R, Qazzaz M. (2008) Effect of Palestinian honey on spermatogenesis in rats. *Journal of Medicinal Food* vol 11 iss 4 pg 799-802

[18] Ajibola .A.,Chamunorwa.J.D.,and Erlwanger.K.H.: Nutraceeutical values of natural honey and its contribution to human health and wealth.Nutriton and metabolism,2012,9:6

[19] Selmanoğlu G, Hayretdağ S, Kolankaya D, Tuylu A O and Sorkun K (2009) The Effect of Pollen on Some Reproductive Parameters of Male Rats. *Pestic. Phytomed*. 24 Pg 59-63

[20] Biswas D; Bari, F.Y; Shamsuddin M; Rahmann M.M : Determination of glycerol percentages for preserving the black Bengal block (Capra hircus) Spermatozoa for long time. *Pakistan Journal of Biological Sciences* 2002,vol 5, issue;6 pg 715-718

[21] De Krester, D M; Loveland K L; Meinhardt, A; Simorangkir D and Wreford, N :Spermatogenesis. *Human Reproduction Volume 13 Supplement 1 1998 pg 1*

[22] Tesarik, J; Guido, M; Mendoza C; Greco, E. (1998) Human spermatogenesis in vitro: Respective effects in Follicle stimulating Hormone and testosterone Meiosis, Spermiogenesis and Sertoli cell apoptosis. *Journal of clinical endocrinology and metabolism.* Vol 83, iss 12, pg 4467.

[23] Eliasson R, Virji N (1985) LDH-C4 in human seminal plasma and its relationship to testicular function II. Clinical aspects. *International Journal of Andrology. 8(3) 201-214.*

[24] El- kabbani O, Darmanin C, Chung R.P.T (2004) Sorbitol dehydrogenase: structure, function and ligand design. *Current medicinal chemistry.* Vol 11, iss 4. Pg 465-476.

[25] Erejuwa O.O, Sulaiman S.A, Wahab M.S.A.B (2012) Honey: a novel antioxidant. *Molecules* 17, pg 4400-4423.

IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861. Volume 9, Issue 6 (Sep.- Oct. 2013), PP 48-52
www.iosrjournals.org

Different Non – Surgical Treatment Modalities for Class III Malocclusion

Dr. Handa Amit Satish[1], Dr. Suchita Daokar[2], Dr. Mandira Gulati[3].

Class III malocclusions are usually growth-related discrepancies & are associated with deviation in the sagittal relationship of the maxilla and the mandible, characterized by a deficiency and/or a backward position of the maxilla, or by prognathism and/or forward position of the mandible or both[1,2,3].

A class III malocclusion is defined by the presence of a class III molar and incisor relationship, which may range from a reduced overjet or edge-to-edge incisor relationship to a frank reversed overjet, the severity typically reflecting the underlying skeletal pattern[1,4].

Treatment planning in class III cases is notoriously difficult and primarily influenced by the likelihood of future growth , skeletal discrepancy, size of the reverse overjet, extent of crowding, and degree of existing dento-alveolar compensation.

An old orthodontic maxim states **"the best time to treat a crossbite is the first time it is seen."**[5] Thus, Class III malocclusion should be intercepted and treated at an early stage so as to prevent an orthodontic problem from progressing into severe dento-facial anomaly.

So, early treatment of Class III malocclusion has been advocated to reduce the need of treatment in the permanent dentition, when camouflage orthodontic treatment or surgery become the only options.[1]

The clinician should determine whether the crossbite is skeletal or dental in origin from the profile analysis, cephalometric readings & intra oral findings[6].

In this paper, the non-surgical orthodontic treatment of three patients with a Class III malocclusion is discussed and the use of compensation mechanics is illustrated.

Though a series of treatment approaches can be found in the literature regarding treatment in Class III malocclusion. However, as with other types of malocclusion, there are really three main approaches to the correction of a class III malocclusion, i.e. growth modification, camouflage orthodontic treatment and surgical approach.

A) **Growth modification**: A class III malocclusions usually present in the mixed dentition. A decision is required at this stage as to whether correction of the incisor relationship and the underlying skeletal discrepancy should be attempted with interceptive treatment. This treatment can be aimed at modifying growth, either with reverse-pull headgear (with or without maxillary expansion) or a functional appliance. Success of this treatment will depend on establishing a positive overjet and overbite, and the nature and direction of future growth.

Case 1:-A 12 year old female reported with the chief complain of backwardly placed upper front teeth. On examination the patient was diagnosed as skeletal class III malocclusion because of retrognathic maxilla. Patient`s parents also reported a familial tendency for class III malocclusion. Reverse overjet, deep bite, highly positioned maxillary canines, class III molar relationship was seen (Figure 1).

Cephalometric values:-

Sr. no	Cephalometric variable	Pre treatment values	After facemask values	Post treatment values
1	Facial angle (FH to N-Pog)	89^0	85^0	87^0
2	Y-growth axis	54^0	57^0	57^0
3	Angle SNA	78^0	80^0	81^0
4	Angle SNB	82^0	78.5^0	80^0
5	Mandibular plane angle	25^0	26^0	27^0
6	Wits appraisal	BO ahead of AO by 1.5mm	AO ahead of BO by 0.5mm	AO ahead of BO by 1mm
7	Inter Incisal angle	132^0	145^0	129^0
8	Upper incisor to NA	33^0 / 2.5mm	34^0 / 2mm	32^0 / 4mm
9	Lower incisor to NB	19^0 /2mm	13^0 / 1.5mm	22^0 / 3mm

The treatment was initiated with a rapid maxillary expansion screw which was bonded intra-orally & activated with 1 turn / day for 8 days. The intention with this expansion screw was to open up the circum maxillary sutures. This was followed by a reverse pull headgear & extra-oral elastics to protract the retrognathic maxilla. To start with light forces were applied around 200-300 grams, which were later increased to around 500 grams on each side (Figure 2). Within a period of 6-7 months the maxilla got protracted and positive overbite & overjet was achieved (Figure 3). Fixed orthodontic treatment was initiated to eliminate the deep bite, rotations, co-ordination of arches and refining the occlusion & the case was debonded and upper and lower retainers delivered (figure 4).

(Figure 1):- Case 1 pre treatment photographs.

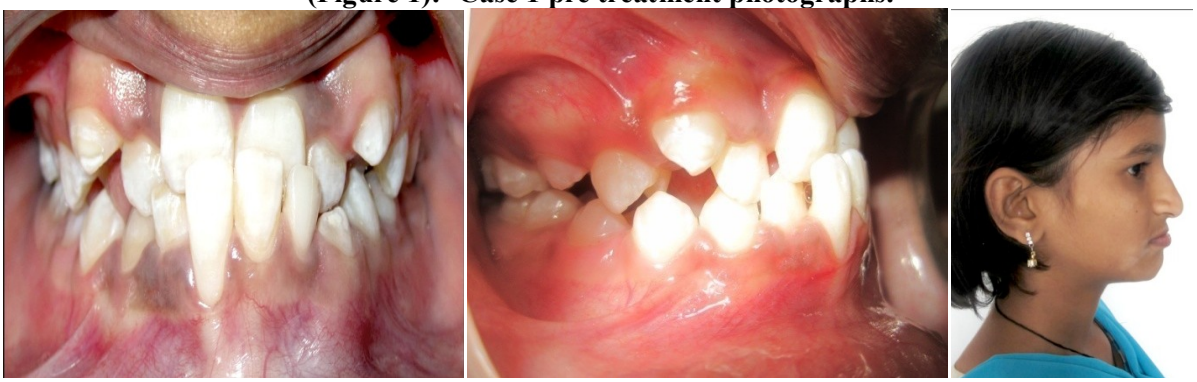


Figure 2:-With face mask and extra-oral elastics and Hyrax screw bonded:-

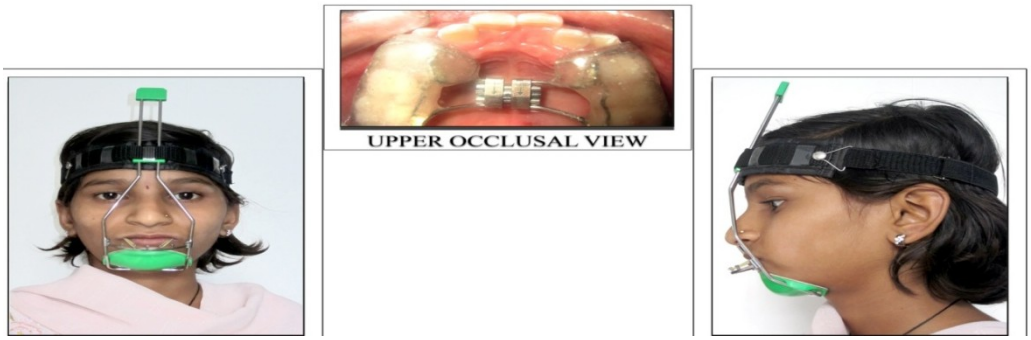


Figure 3:- After FACEMASK:-

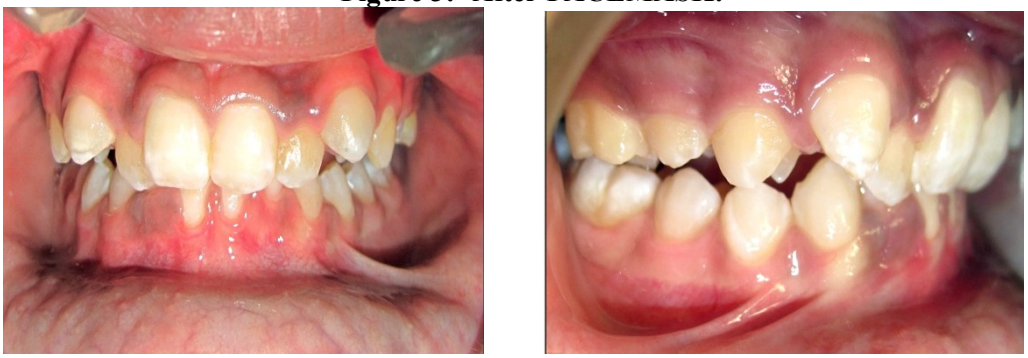
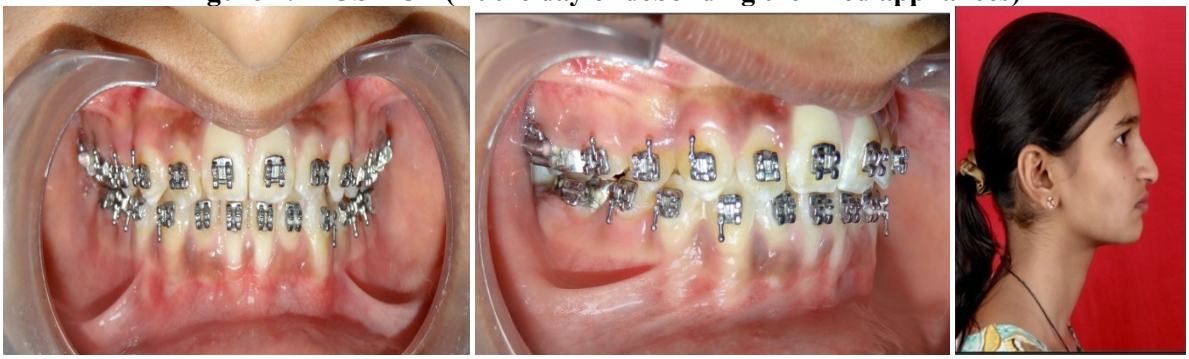


Figure 4:- POST OP (At the day of debonding the fixed appliances)



B) **Orthodontic camouflage** : Definitive treatment can be carried out in the permanent dentition if the skeletal discrepancy is mild and facility for dento-alveolar compensation still exists. Comprehensive correction in the permanent dentition typically involves the use of fixed appliances with class III inter-arch elastic traction. Extractions are often required in the upper arch of class III cases because of crowding; however, when attempting camouflage, lower arch extractions are also commonly required to create space for retraction of the lower labial segment. Mid-arch extractions are usually undertaken in both arches, although in adult patients a single lower incisor extraction can be considered. The prolonged success of treatment depends on establishing a positive overjet and overbite, and the pattern and magnitude of further growth.

Case 2:-A 19 year female reported with chief complain of irregular placed upper front teeth and abnormal relationship between upper and lower front teeth. On examination the patient was diagnosed as skeletal class III malocclusion and anterior crossbite. Class III molar relationship existed on both sides & positive overbite only with right central incisor (Figure 5).

Sr. no	Cephalometric variable	Pre treatment values	Post treatment values
1	Facial angle (FH to N-Pog)	88^0	87^0
2	Y-growth axis	57^0	58^0
3	Angle SNA	80^0	81^0
4	Angle SNB	81^0	80^0
5	Mandibular plane angle	26^0	27^0
6	Wits appraisal	AO & BO coincides	AO ahead of BO by 0.5mm
7	Inter Incisal angle	135^0	133^0
8	Upper incisor to NA	30^0/ 5mm	32^0 / 6mm
9	Lower incisor to NB	24^0 / 3mm	22^0 / 2mm

Orthodontic treatment was initiated in this case and after completion of leveling & alignment phase intra-oral class III elastics i.e. from maxillary molars to mandibular canines were given. The anterior cross-bite got corrected because of the class III mechanics with the help of intra-oral elastic traction. Thus a pleasing facial profile with mild to moderate class III malocclusion can be treated orthodontically (Figure 6).

Figure 5:- Case 2 treated orthodontically with the help of Class III elastic traction.

Pre-op photographs:-

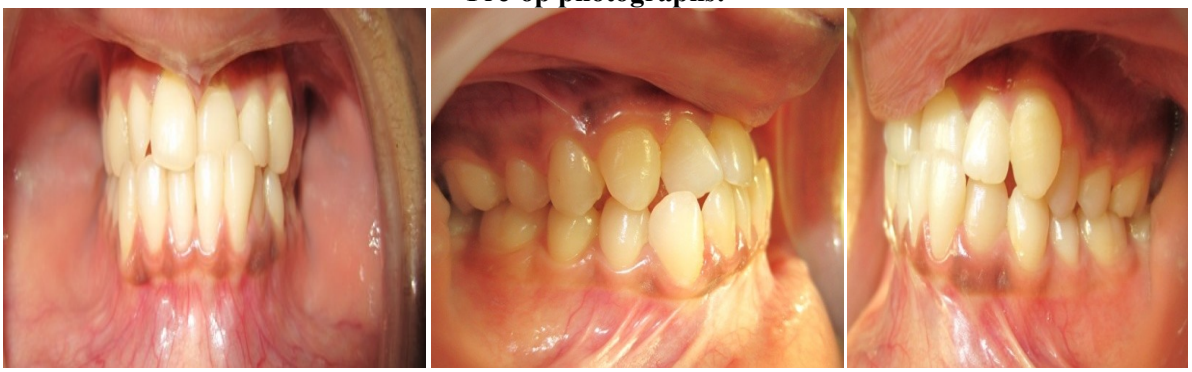
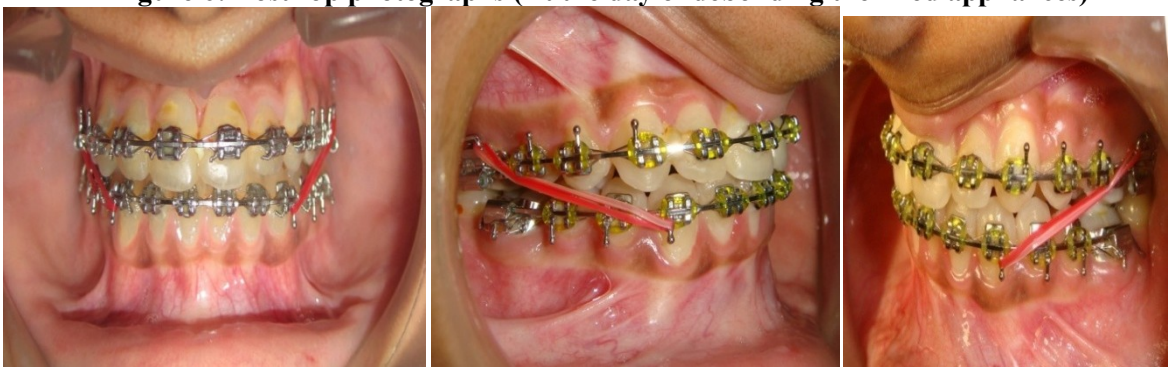


Figure 6:-Post--op photographs (At the day of debonding the fixed appliances)



Case 3:- A 27 year old male reported with chief complain of backwardly placed upper front teeth. On examination the patient was diagnosed as skeletal class III malocclusion with anterior crossbite. A concave profile, anterior crossbite with both central incisors & reverse overjet, class I molar relation on both sides existed (figure 7).

Sr. no	Cephalometric variable	Pre treatment values	Post treatment values
1	Facial angle (FH to N-Pog)	88^0	87^0
2	Y-growth axis	66^0	67^0
3	Angle SNA	80^0	81^0
4	Angle SNB	82^0	81^0
5	Mandibular plane angle	32^0	33^0
6	Wits appraisal	BO ahead of AO by 1.5mm	BO ahead of AO by 0.5mm
7	Inter Incisal angle	136^0	128^0
8	Upper incisor to NA	25^0/ 5mm	26^0 / 6mm
9	Lower incisor to NB	27^0 / 5mm	21^0 / 3mm

Orthodontic treatment was initiated and upper and lower arches were bonded. Initial leveling and alignment was initiated and mandibular first pre-molars were extracted on both the sides. Mandibular anteriors were retracted and upper anteriors were aligned to achieve positive overbite and overjet. Thus a moderate skeletal class III malocclusion was orthodontically treated by camouflage (Figure 8).

Figure 7:- A skeletal class III Case treated orthodontically with camouflage treatment.
Pre-op photographs:--

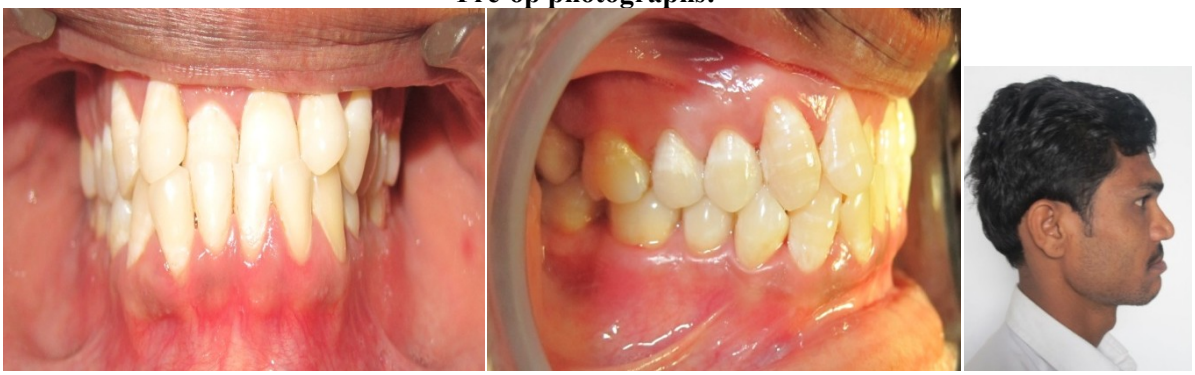
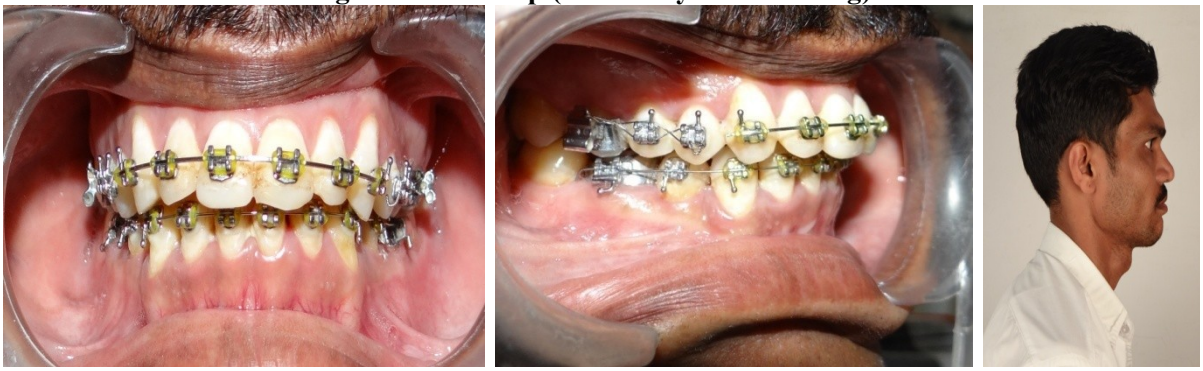


Figure 8:- Post-Op (At the day of debonding):-



Conclusion:-

The non-surgical treatment options to treat Class III malocclusions were been discussed and case reports illustrated. Treatment was undertaken using a combination of orthopedics treatment options , compensation mechanics and fixed orthodontic appliance treatment only and suggests that in some, carefully selected cases, this approach can be a viable treatment option.

References:-

[1]. Proffit WR. Contemporary Orthodontics. 4th ed. St Louis, Mo: Mosby; 2007:689–707.
[2]. Guyer EC, Ellis EE, McNamara JA Jr, Behrents RG. Components of Class III malocclusion in juveniles and adolescents. *Angle Orthod* 1986; **56**: 7–30.[Medline]
[3]. Williams S, Andersen CE. The morphology of the potential Class III skeletal pattern in the growing child. *Am J Orthod Dentofac Orthop* 1986; **89**: 302–11.
[4]. Moyers, R.E. Handbook of Orthodontics, 3rd Ed. Year Book Medical Publishers, Inc; Chicago, 1983, 574-77.
[5]. Bhalajhi S.I, Orthodontics- The Art and Science. 3rd Ed., Anja (Med) Publishing House , chap20 , pg. 233 .
[6]. Pinkham J.R , Pediatric Dentistry- Infancy through Adolescence , 4th Edition, Elseveir , a division of Reed Elsevier India Pvt. Ltd. 2005 , chapter 35, pg. 642-644.
[7]. Baccetti T, McGill JS, Franchi L, McNamara JA Jr, Tollaro I. Skeletal effects of early treatment of Class III malocclusion with maxillary expansion and face-mask therapy. *Am J Orthod Dentofacial Orthop.* 1998;113:333–343.
[8]. Kajiyama K, Murakami T, Suzuki A. Evaluation of the modified maxillary protractor applied to Class III malocclusion with retruded maxilla in early mixed dentition. *Am J Orthod Dentofac Orthop.* 2000;118:549–559.
[9]. Yuksel S, Ucem TT, Keykubat A. Early and late facemasktherapy. *Eur J Orthod.* 2001;23:559–568.
[10]. Popp TW, Gooris CGM, Schur AJ. Nonsurgical treatment for a Class III dental relationship: a case report *Am J Orthod Dentofac Orthop* 1993; **103**: 203–11.[Medline]

Author's Information:-

1) Dr. Handa Amit Satish,
M.D.S., Orthodontics And Dentofacial Orthopaedics, Aurangabad, Maharashtra, India.

2) Dr. Suchita S. Daokar,
Professor and P.G. Guide, Department of Orthodontics, Aurangabad, Mah., India.

3) Dr. Mandira Gulati,
B.D.S.,
Practicing As Dental Surgeon, Delhi, India.

IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861. Volume 9, Issue 6 (Sep.- Oct. 2013), PP 53-56
www.iosrjournals.org

Effects of analgesic & Anti-inflammatory drugs on orthodontic tooth movement- A biochemical & Histological study in guinea pigs

Dr.Suchita Tarvade/ Daokar1, Dr.Sadashiv Daokar[2] , Dr. P.V. Hazarey.[3]
1-Professor & P.G.Guide, Dept.of Ortho, C.S.M.S.S. Dental College, Aurangabad.
2- Professor & P.G.Guide, Dept.of conservative dentistry, C.S.M.S.S. Dental College, Aurangabad.
3- Professor, H.O.D.,& P.G.Guide, Dept.of Ortho, S.P.D.C. Dental College,Nagpur.

I. Introduction :-

The essence of orthodontic treatment is the movement of teeth throughbone to obtain a more perfect dental occlusion. Mechanical forces exerted ontooth and transmitted to the surrounding tissues of periodontal ligament initiates the remodeling activity and facilitates the movement of teeth through bone.

Cells of the nervous , immune and endocrine systems become involved in the activation and response of periodontal ligament and alveolar bone cells during tooth movement.Orthodontic tooth movement has an inflammatory response and so evokes painwhich is the most unpleasant symptom which drives the patient to seek medicaments. Analgesic drugs are commonly prescribed in day to day orthodontic practice to control pain evoked by orthodontic forces. These drugs are also available as OTC (over-the –counter) drugs and can be bought from medical shops without prescription.

T he present investigation is done to study the effects of three different analgesic and anti-inflammatory drugs, acetaminophen, ibuprofen and nimesulide, on the rate of orthodontic tooth movement in guinea pigs.

Aims and Objectives:-

1. Compare the effects of three commonly used analgesics acetaminophen. Ibuprofen and nimesulide on the rate of orthodontic tooth movement in male guinea pigs for 72 hours.
2. Compare the effects of three commonly used analgesics acetaminophen. Ibuprofen and nimesulide on the acid phosphatase level in serum during orthodontic tooth movement.
3. To study and compare the histological changes in periodontal ligament during orthodontic tooth movement with the administration of acetaminophen, ibuprofen and nimesulide.
4. To correlate the histological changes with biochemical changes during orthodontic tooth movement when these analgesic drugs are administered.

Review of literature :-

In 1880, Norman Kingsley, discussed about alveolar bone and its response to forces . He explained orthodontic movement of teeth as a result of elasticity of the alveolar bone.

In 1888, Farrar described the orthodontic movement of teeth as a result of the resorption and apposition of bone and bending of the alveolar process.

Herzberg in 1932 was the first to move a human tooth with an orthodontic appliance and study its surrounding tissues.

In 1953, Storey recognized 4 zones of activity around a tooth which was being moved with light orthodontic forces. On the pressure side resorption and then deposition, on the tension side deposition and then resorption.

D.L. Buck and D. H. Church in 1972 studied histological changes during human tooth movement.

Klein and Raisz in 1970 reported for the first time that prostaglandin stimulate bone resorption by acting on osteoclasts in tissue cultures.

Harell et al in 1977 suggested for the first time that prostaglandins may be important mediators of mechanical stress.

Storey in 1978 showed that inflammatory reactions are associated with the application of orthodontic forces.

Davidovitch and Shanfeld in 1980 also reported the involvement of PGE2 in bone remodeling of orthodontically treated cats showing the rise of PGE2 levels in alveolar bone.

Yamasaki in 1984 studied the effect of locally administered PGE upon tooth movement and showed increase rate in tooth movement.

II. Materials and Methods:-

A total number of 28 healthy male guinea pigs were used in this study. The animals were divided into two main groups

1. Group I- 24 animals were used in this group for biochemical study.
2. Group II- 4 animals were used in this group for histological study.

Group I and Group II were further divided into 4 subgroups each

Subgroup I and II (a)- Animals in these subgroups were used as control . orthodontic treatment was given but no drugs were administered throughout the study to these animals.

Subgroup I & II (b)- animals in these subgroups were given orthodontic treatment and acetaminophen suspension was administered 12 hourly for 3 days.

Subgroup I & II (c) – animals in these subgroups were given ibuprofen suspension 12 hourly for 3 days along with orthodontic treatment.

Subgroup I & II (d) – animals included in these subgroups were administered nimesulide suspension 12 hourly for 3 days along with orthodontic treatment.

Orthodontic appliance :-

After sedating the guinea pigs with 2 mg/kg of ketamine intramuscularly bands with vertically welded eyelet were cemented on both mandibular central incisor.

A spring was prepared of 0.014" (A. J. Wilcock's wire) with two vertical loops mesial arm slightly shorter then the distal arm. 2 helical coils of 1.5 mm inner diameter and one and a half turn were incorporated in these vertical loops in between the mesial and distal arm.

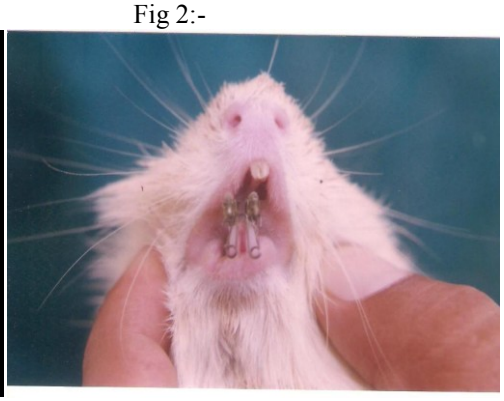
The spring was engaged in the two eyelets.

The guinea pigs were then isolated .They were given green leafy vegetables and were administered drugs .

The tooth separation measurements were done between the mesial margins of the incisal edges of the two mandibular incisors using vernier caliper. The recording were done prior to placement of the appliance, 24 hrs, 48 hours and 72 hours after orthodontic appliance placement. At each seating, measurement was repeated for 3 times, of each animaland a mean of 3 measurements was taken.

Fig 1:-

Fig 2:-



Blood was collected after 72 hours of appliance placement.

For histological examination , tissue was collected by administering a lethal dose of ketamine to all the animals of group II.

III. Results:-

Table no1:- the results of tooth separation in mm among the sub-groups were:

At	Subgroup I (a)		Subgroup I (b)		Subgroup I (c)		Subgroup I (d)	
	Mean	S.D	Mean	S.D	Mean	S.D	Mean	S.D
24 hrs	1.56	+/0.12	1.41	+/-0.14	0.91	+/-0.11	0.7	+/-0.104
48hrs	2.66	+/-0.12	2.53	+/-0.18	1.4	+/- 0.08	1.25	+/-0.164
72 hrs	3.70	+/-0.08	3.6	+/-0.17	2	+/- 0.08	1.75	+/- 0.148

Table no 2:- the results of Acid Phosphates level in the serum among the sub-groups were:-

At	Subgroup I (a)		Subgroup I (b)		Subgroup I (c)		Subgroup I (d)	
	Mean	S.D	Mean	S.D	Mean	S.D	Mean	S.D
24 hrs	2.05	+/-2.06	1.81	+/-0.15	0.81	+/-0.26	0.86	+/-0.12
72 hrs	2.8	+/-2.06	2.65	+/-0.15	1.7	+/- 0.08	1.36	+/-0.20

The student 't' test and ANOVA test were applied to all the observations .
Tooth separation and acid phosphatase level were tested for all the

Fig 3:-

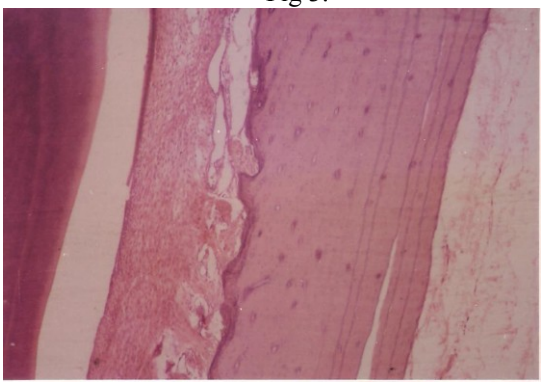


Fig 4:-

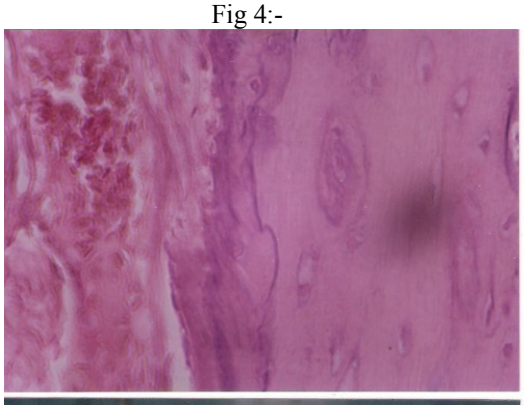


Fig 5:-

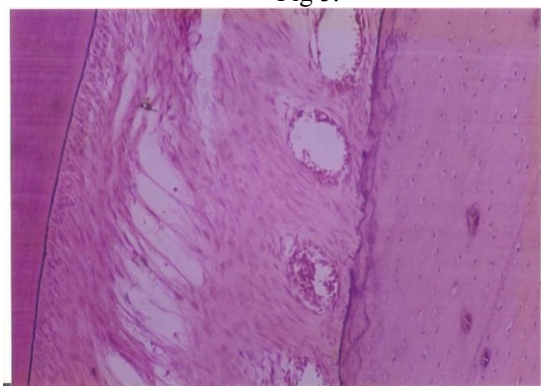
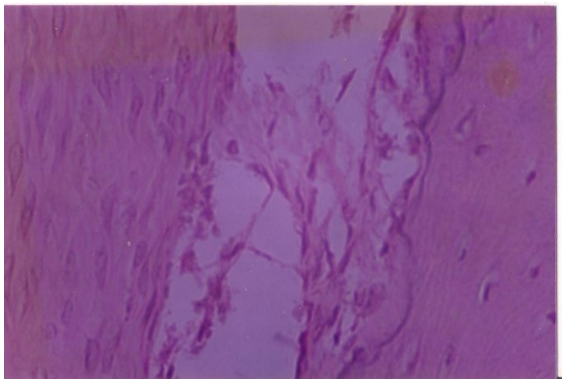


Fig 6:-



IV. Discussion :-

Student 't' test and ANOVA test was performed to comoare the significance of difference between the tooth separation and acid phosphatase level in serum amongst the subgroups.
The above parameters were compared in between control group i.e; subgroup I(a) and

1. Subgroup I (b)- Acetaminophen
2. Subgroup I (c)- Ibuprofen
3. Subgroup I (d)- Nimesulide

When the parameters were compared between the 4 subgroups the acid phosphatase levels were found to be highest in control group followed by the acetaminophen subgroup. The acid phosphatase levels were found to be significantly lower in ibuprofen and nimesulide subgroup when compared to control group . When levels of acid phosphatase were compared within the subgroups again it was found that there was statistically significant.
The histological finding showed serrated border of the bone and large no. of howship's lacunae and osteoclasts in the distal side of the tooth in control and acetaminophen subgroups, where as the ibuprofen and nimesulide subgroups showed fewer howship's lacunae and osteoclasts .

Thus the biochemical and histological findings were in correlation with each other.These findings were similar to the findings reported by Stephen Keeling et al. they investigated histochemically and biochemically acid and alkaline phosphatase level in serum and alveolar bone during orthodontic tooth movement in 288 male Sprague Dawley strain rats.

V. Conclusion :-

In summary the mean tooth separation was found to be highest in control group (subgroup I a). This was followed almost the same amount of tooth separation in acetaminophen (subgroup I b). this suggest that acetaminophen can be administered to patients for pain control during orthodontic treatment.Minimal tooth separation was found in ibuprofen (subgroup I c) and nimesulide (subgroup Id). This suggest that ibuprofen and nimesulide decrease the rate of tooth movement and so should be avoided during orthodontic therapy.
Similar findings were found in acid phosphatase level in serum betweenthe subgroups suggesting that acid phosphatase level in serum reflect the alveolar bone turnover during orthodontic tooth movement.
Further these findings were also confirmed by light microscopic histological study.

The conclusions drawn from the study were :-

1. The effect of acetaminophen administration on rate of tooth movement, acid phosphatase level in serum and bone resorption was not significantly different as compared to control group.
2. The administratin of ibuprofen significantly decreased the rate of tooth movement and acid phosphatase level in serum as compared to control group and acetaminophen group.
3. The administration of nimesulide also significantly decreased the rate of tooth movement and acid phosphatase level in serum as compared to control group and acetaminophen group.
4. The effect of ibuprofen and nimesulide administration on the rate of tooth movement and acid phosphatase level in serum was not significantly different when compared with each other.
5. The effect of acetaminophen administration on the rate of bone resorption was not significantly different as compared to control group.
6. The administration of ibuprofen and nimesulide significantly decreased the rate of bone resorption and appearance of osteoclasts as compared to control group and acetaminophen group, but was not significantly different when compared with each other.
7. When the histological findings and biochemical findings were correlated with other a high correlation was found in between them.
   Thus it can be concluded that acid phosphatase level in serum reflects the alveolar bone turn-over during orthodontic tooth movement.
   This study suggest that acetaminophen should be the drug of choice for pain control in orthodontic practice as it has minimal adverse effect on orthodontic tooth movement.

References:-

[1]. Angle Edward H : Treatment of malocclusion of teeth. Ed. 7 Philadelphia 1907 chap 6.
[2]. Baumrind Sheldon : A reconsideration of the property of the pressure and tension hypothesis. Am. J. Orthod & Dentofac. Orthop .55; 12-21: 1969
[3]. Binderman I : The transduction of mechanical force into biochemical event in bone cells may involve activation of phospholipase A2.Calcif Tissue Int. 42; 261-6:1988
[4]. Breitnar C : Bone changes resulting from experimental orthodontics treatment. Am.J. Orthod & Dentofac. Orthop. 26:521 : 1940
[5]. Buck D.L. & Church D. H. : a histologic study of human tooth movement .Am>J. Orthod. & Dentofac. Orthop. 62;507-516;1972
[6]. Burstone M. S : Histochemical demonstrationof acid phosphatase activity in osteoclasts. J. Histochem Cytochem. 7,39-41,1959
[7]. Chumbley A.B. & Tuncay O.C. : the effects of indomethacin (an aspirin like drug) on the rate of orthodontic tooth movement . Am.J. Orthod & Dentofac. Orthop. 89;312-314;1986
[8]. Davidovitch Z & Shanfeld J.L. : Biochemical mediators of the effects of mechanical forces & electric currents on mineralized tissues. Calcif Tissue Int. 36;586-597 : 1984
[9]. Davidovitch & Shanfeld : Cyclic AMP level in alveolar bone of orthodontically treated cats.
[10]. Feldman R. S. et al : Inhibition of alveolarbone loss in humans by aspirin . J. Dent. Resc.59 ; Special issue A; 1980.
[11]. Golamman M & Gianelly A : Histology of tooth movement. DCNA; 439-448 : 1972
[12]. Keeling Stephen et al : Serum & alveolar bone phosphatase changes reflect bone turnover during orthodontic tooth movement . Am.J. Orthod & Dentofac. Orthop. 103;320-326; 1993.
[13]. Kehoe M. J. : The effect of acetaminophen, ibuprofen & misoprostol on prostaglandin E2 synthesis & the degree & rate of orthodontic tooth movement. Angle Orthod. 66 ; 339-350: 1996.
[14]. Kharbanda O.P & Mohaptra P: A review of Arachidonic Acid metabolite & Orthodontic tooth movement .JIDA: 66 ; 287-292 : 1995
[15]. Klein D.C. & Raisz L.G : Prostaglandin – Stimulatin of bone resorption in tissue culture . Endocrinology : 86: 1436- 40 : 1970
[16]. Samuelson et al : Pathways of arachidonic acid metabolism its advances in inflammation research . New York : 1975 , Raven Press Vol I, P 405
[17]. Takimoto , Kazuo Deguchi & Mori : Histochemical detection of acid & alkaline phosphatase in periodontal tissue after experimental tooth movement. J.Dent Res. 47; 340 ; 1968.

IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861. Volume 9, Issue 6 (Sep.- Oct. 2013), PP 57-63
www.iosrjournals.org

Efficacy and Optimal Time of Oropharyngeal Topical 10% Lignocaine Spray Before Induction of Anaesthesia in Attenuating the Pressor Response to Direct Laryngoscopy and Endotracheal Intubation.

Dr. Faisal Mushfeen Qureshi[1], Dr. Sampathila Padmanabha[2],
Dr. Habib Rahaman[3], Dr. Sheikh Imran[4], Dr. Pratibha Ram Mohan[5]

[1]*Department of Anaesthesia and Critical care,Yenepoya Medical College, Yenepoya University, Mangalore, Karnataka,India.*
[2]*Professor & Head, of Anaesthesia and Critical care,Yenepoya Medical College,Yenepoya University, Mangalore, Karnataka,India.*
[3]*Associate Professor, of Anaesthesia and Critical care,Yenepoya Medical College,Yenepoya University, Mangalore, Karnataka,India.*
[4,5]*Post graduate final year, of Anaesthesia and Critical care, Yenepoya Medical College,Yenepoya University, Mangalore, Karnataka,India.*

Abstract: *The study aimed to ascertain the efficacy of oropharyngeal topical 10% lignocaine spray applied prior to induction of anaesthesia and also to determine the optimal time for spraying to attenuate pressor response to laryngoscopy and endotracheal intubation. 80 patients of either sex, in the age group of 20-60 years (ASA I & II) undergoing different elective surgical procedures under general anaesthesia were taken up for the study. 10 puffs of Lignocaine 10% sprayed 1 min before induction in Group I, 3 min before induction in Group 2, 5 min before induction in Group 3 and normal Saline in Group 4 as control. Heart rate(HR), systolic (SBP), diastolic (DBP) and mean arterial pressure (MAP) were measured at baseline, after intubation, after 1min, 3 min and 5 min following intubation. There was statistically significant increase in HR, systolic, diastolic and MAP in control group when compared to baseline as well as to study groups. Following laryngoscopy and intubation attenuation of pressor response did not show a significant difference at 1min, 3min and 5min of 10% lignocaine spray within study groups. Lignocaine 10% when sprayed to the oropharynx prior to induction of anaesthesia attenuated the pressor response to laryngoscopy and intubation irrespective of timing of the spray.*

Keywords: *Endotracheal Intubation, Lignocaine spray (10%), Laryngoscopy, Pressor response, Systolic Blood pressure (SBP), Diastolic blood pressure (DBP), Mean arterial pressure (MAP).*

I. Introduction

In practice of anaesthesia, laryngoscopy and tracheal intubation forms the basis of controlling the patient's airway during general anaesthesia or for artificial ventilation. Laryngoscopy and endotracheal intubation has been a challenging procedure due to associated cardiovascular changes called *pressor response.* This response includes widespread release of norepinephrine from adrenergic nerve terminals and secretion of epinephrine from the adrenal medulla[1]. In adults and adolescents, the more common response to this airway manipulation is hypertension and tachycardia, mediated by the cardio-accelerator nerves and sympathetic chain ganglia.

In addition to activation of the autonomic nervous system, laryngoscopy and endotracheal intubation result in stimulation of the central nervous system (CNS), as evidenced by increases in electroencephalographic activity, cerebral metabolic rate, and cerebral blood flow (CBF)[2-5].

Pressor response has deleterious effects on patients with preexisting hypertension, ischaemic heart disease which may lead to tachycardia, arrhythmia, cerebrovascular accidents etc.

Several drugs like lignocaine[6], clonidine[7], fentanyl[8] and betablockers[9], has been tried to control the pressor response to laryngoscopy and intubationbut it is not possible to completely abolish the response.

The present study was undertaken to study the efficacy of oropharyngeal topical 10% lignocaine spray in attenuating the pressor response as well as to find the optimal timing of application of lignocaine spray.

II. Materials And Methods:

We conducted a prospective, randomized, controlled study with 80 patients of ASA grade I & II, of either sex, aged between 20 and 60 years, undergoing routine elective surgical procedures under general anaesthesia. Study was conducted after getting institutional ethical committee approval and informed consent from all the patients.We excluded patients with uncontrolled hypertension, significant hepatic or renal disease, predicted difficult intubation, history of hypersensitivity to amide local anaesthetics, seizure disorder and patients taking any systemic medications.After pre-operative evaluation and necessary routine investigations, premedicated with tab Ranitidine 150mg, and tab Lorazepam 1mg 2 hour before induction. Glycopyrollate 0.2mg was administered intramuscularly 30 min before induction. Routine monitoring was done using

noninvasive blood pressure (NIBP), electrocardiogram (ECG), pulse oximeter (SPO2). Baseline values of heart rate(HR), systolic blood pressure(SBP), diastolic blood pressure(DBP) and mean arterial pressure(MAP) were measured before spraying with lignocaine(10%).

Patients were randomly allotted into one of the four groups using lottery method; each group consisted of 20 patients. In group 1, patients were subjected to lignocaine 10% spray 1 min before induction, in group 2 lignocaine 10% spray was administered 3 min before induction, similarly in group 3 lignocaine 10% spray was administered at 5min before induction, patients in group 4 were administered normal saline spray who served as control.Lignocaine 10% oral spray was administered in the sitting position by asking the patient to fully open their mouth and using a disposable spray canula into the mouth without using laryngoscope, a total of 10 puffs (2 puffs to soft palate, 3 puffs to posterior oropharyngeal wall, 2 puffs to palatopharyngeal arch and 3 puffs to posterior 3rd of tongue, each puff delivering 10mg, resulting in a total dose of 100mg and not exceeding the toxic dose of 3-4mg/kg. All patients were induced with propofol 2mg/kg followed by rocuronium 0.9mg/kg to facilitate endotracheal intubation. Using Macintosh blade laryngoscopy and intubation was done by anesthesiologist having 5 years of experience. Patients with difficult intubation which require laryngoscopy more than 20 seconds and who require more than one attempt were excluded from the study. Fentanyl 2µgs/kg was given as an analgesic. A second recording of HR, SBP, DBP and MAP were noted immediately after laryngoscopy and intubation, and then 1min, 2min, 3min and 5min intervals after laryngoscopy and intubation.

The results were subjected to statistical analysis using Student's unpaired**'t'**test and ANOVA was used for overall differences. Any p value less than 0.05 were taken as statistically significant. SPSS version 17 was used for analysis.

III. Results:

There was no statistically significant difference in the demographic characteristics among the 4 groups (Table 1)

There was significant rise of heart rate in the control groupwhen compared to the baseline heart rate.There was also a statistically very highly significant ($p< 0.001$) difference compared to study groups 1, 2, 3. However,among the study groups,change in the heart rate was not statistically significant (Tables 2&3). SBP, DBP and MAP showed statistically highly significant change ($p< 0.001$) in control group(4) when compared to study groups and baseline. Moreover, there was statistically insignificant difference in SBP, DBP and MAP ($p= 0.321$, $p= 0.772$, $p= 0.520$) respectively between groups 1, 2, 3 (Tables 4-9).

IV. Discussion:

Laryngoscopy and intubation is associated with a sympathoadrenal response leading to increase in arterial blood pressure, heart rate and may also result in arrhythmias.This bears a particular significance in patients of cardiac condition like hypertension, patients with acute myocardial infarction[1-5].

The laryngoscopy and endotracheal intubation(ET) intubation is considered to be a stronger stimulus than a surgical incision**.** The amount of pressor response is related to the force applied to the base of tongue, the duration of laryngoscopy.There have been many different ways tried to decrease the pressor response ranging from non-pharmacological methods like using LMA[10], various types of laryngoscope blades like McCoy[11] for doing laryngoscopy, to pharmacological methods like lignocaine, alpha-2 agonists, opioids, nitroglycerine, beta-blockers among others[6-9,12.]

Lignocaine has been tried as intravenous administration prior to laryngoscopy, mouth gargles in various concentration, sprays. The mechanism of action of lignocaine in blunting pressor response differs according to the method of administration. Local administration like gargles and sprays may be effective due to its local anaesthetic property at the base of tongue and pharyngeal walls preventing the receptor stimulation. The intravenous administration mainly acts by decreasing the cerebral blood flow and depression of myocardial contractility[13]. Abou-Madi *etal*[14] have discussed the possible mechanisms to account for these observations with IV lignocaine. These include a direct myocardial depressant effect, a peripheral vasodilation effect and finally an effect on synaptic transmission.

In our study we used oropharyngeal topical 10% lignocaine sprayprior to inductionto determine the efficacy in attenuating the pressor response and optimal timing of spraying. Weused 10 puffs in total, each puffdelivering 10mg, so total dose given was 100 mg. This dose is well below the toxic dose of lignocaine. We used glycopyrollate[15] 0.2 mg as a premedication which has been proved to be enhancing absorption, prolongs the analgesic action of lignocaine and has anti-sialagogue property. We found that the lignocaine spray was effective in attenuating pressor response (in terms of heart rate, systolic blood pressure, diastolic blood pressure and mean arterial pressure) in all the three groups receiving the drug in comparison to the group 4 which received normal saline and to the baseline values.

We performed a single laryngoscopy prior to endotracheal intubation whereas laryngoscopy was performed twice by Mostafa *etal*[16]because they administered the spray after induction, thus results are not

comparable. Hamill *etal*[17] compared topical lignocaine with I.V. lignocaine. In the topical group, after induction of anesthesia they performed laryngoscopy to spray the oropharynx, two minutes later, they performed another laryngoscopy for endotracheal intubation. While in other group they performed only one laryngoscopy and hence noxious stimuli were given twice in the topical group.Mostafa[16] and Hamill[17] in their respective studies had concluded that topical lignocaine is ineffective in preventing the pressor response. Takita *etal*[18] suggested that differing intervals between tracheal lignocaine and endotracheal intubation probably caused the inconsistent conclusion reported in other investigations and showed that endotracheal intubation performed two minutes after tracheal lignocaine attenuated the cardiovascular responses to endotracheal intubation M. Jain *et al*[19] performed a study on lignocaine 10 percent spray to study its efficacy in attenuating the pressor response and found it to be effective in attenuating and not abolishing the response, we had similar findings in lignocaine being able to attenuate the pressor response, but they didn't study the optimal timing of the spray and they didn't use glycopyrrolate as a premedication.

V. Conclusion

We conclude that spraying oropharynx with 10 puffs (100mg) of topical 10% lignocaine is effective in blunting the pressor response to laryngoscopy and intubation when it is applied before induction of general anaesthesia irrespective of timing of the spray.

Table 1: Demographic characteristics (mean ± SD) of four groups

Demographic characteristics	Group 1 (n=20)	Group 2 (n=20)	Group 3 (n=20)	Group 4 (n=20)	Statistical significance
Age	40.76± 5.8	41.10 ± 7.0	41.06 ± 7.2	40.82 ± 8.1	P = 0.986
Sex (Male/Female)	11/9	10/10	11/9	8/12	X^2 = 0.973 P = 0.823

n = number of subjects

Table 2: Table showing Mean ± SD changes in Heart rate in groups 1, 2, 3 (Study) and group 4 (Control)

HEART RATE	Groups 1,2,3 STUDY Mean ± S.D	Group 4 CONTROL Mean ± S.D
Baseline	86.5 ±12.1	87.0±11.3[NS]
After L & I*	103.0±8.1	118.0±14.2[a]
After 1 MIN	100.2±7.8	113.6±14.6[a]
After 2 MIN	96.0±6.2	106.7±11.5[a]
After 3 MIN	92.2±5.3	99.3±9.8[a]
After 5 MIN	88.9±4.2	95.2±7.8[a]

*Laryngoscopy and Intubation

[a] = Highly significant (p<0.001)

[NS] = Not significant (p>0.05)

Table 3 Table showing Mean ± SD changes in Heart rate in groups 1, 2, 3 (ANOVA)

HEART RATE	Group 1 Mean	Group 2 Mean	Group 3 Mean	P Value[#]
Baseline	86.8	85.9	88.0	0.839
After L & I*	103.1	103.2	102.8	0.989
After 1 MIN	100.4	100.2	100,0	0.989
After 2 MIN	96.1	95.4	96.4	0.867
After 3 MIN	92.0	91.3	93.3	0.411
After 5 MIN	88.8	88.1	89,9	0.338

[*] Laryngoscopy and Intubation

[#] P values >0.05 are insignificant

Table 4 Table showing Mean ± SD changes in Systolic blood pressure in groups 1, 2, 3 (Study) and group 4 (Control)

SYSTOLIC BLOOD PRESSURE	Groups 1,2,3 STUDY Mean ± S.D	Group 4 CONTROL Mean ± S.D
Baseline	127.1 ±8.5	128.2±7.1[NS]
After L & I[*]	141.6±7.0	161.2±5.7[a]
After 1 MIN	138.6±6.6	155.4±5.6[a]
After 2 MIN	135.8±6.0	149.6±5.6[a]
After 3 MIN	132.9±5.6	144.9±5.1[a]
After 5 MIN	131.8±5.7	140.8±6.4[a]

[*]**Laryngoscopy and Intubation**
[a] **= Highly significant (p<0.001)**
[NS] **= Not significant (p>0.05)**

Table 5 Table showing Mean ± SD changes in Systolic blood pressure in groups 1, 2, 3 (ANOVA)

SYSTOLIC BLOOD PRESSURE	Group 1 Mean	Group 2 Mean	Group 3 Mean	P Value[#]
Baseline	125.8	126.9	128.7	0.578
After L & I[*]	140.2	141.7	143.0	<0.001
After 1 MIN	137.3	138.6	139.8	<0.001
After 2 MIN	134.6	135.8	136.8	<0.001
After 3 MIN	132.0	132.7	133.9	<0.001
After 5 MIN	130.6	131.2	133.4	<0.001

[*]**Laryngoscopy and Intubation**
[#]**P values >0.05 are insignificant**

Table 6 Table showing Mean ± SD changes in Diastolic Blood Pressure in groups 1, 2, 3 (Study) and group 4 (Control)

DIASTOLIC BLOOD PRESSURE	Groups 1,2,3 STUDY Mean ± S.D	Group 4 CONTROL Mean ± S.D
Baseline	81.3±7.5	81.8±6.7[NS]
After L & I[*]	93.4±7.4	105.3±6.1[a]
After 1 MIN	90.8±7.0	100.6±4.7[a]
After 2 MIN	87.6±6.9	96.3±4.9[a]
After 3 MIN	85.3±6.5	91.9±5.0[a]
After 5 MIN	82.9±6.4	87.8±4.9[a]

[*]**Laryngoscopy and Intubation**
[a] **= Highly significant (p<0.001)**
[NS] **= Not significant (p>0.05)**

Table 7 Table showing Mean ± SD changes in Diastolic Blood Pressure in groups 1, 2, 3 (ANOVA)

DIASTOLIC BLOOD PRESSURE	Group 1 Mean	Group 2 Mean	Group 3 Mean	P Value[#]
Baseline	80.8	80.7	82.5	0.807
After L & I[*]	93.0	92.9	94.3	<0.001
After 1 MIN	90.4	90.5	91.5	<0.001
After 2 MIN	87.7	87.0	88.0	<0.001
After 3 MIN	85.4	84.7	85.8	<0.001
After 5 MIN	83.0	82.3	83.6	<0.001

[*]**Laryngoscopy and Intubation**
[#]**P values >0.05 are insignificant**

Table 8 Table showing Mean ± SD changes in Mean Arterial Pressure in groups 1, 2, 3 (Study) and group 4 (Control)

Mean arterial pressure	Groups 1,2,3 STUDY Mean ± S.D	Group 4 CONTROL Mean ± S.D
Baseline	97.6 ±6.8	97.2±5.6[NS]
After L & I[*]	109.5±6.2	123.9±5.3[a]
After 1 MIN	106.7±5.8	118.9±4.0[a]
After 2 MIN	103.6±5.7	114.O±4.2[a]
After 3 MIN	101.1±5.2	109.6±4.0[a]
After 5 MIN	99.2±5.1	105.5±4.0[a]

[*]**Laryngoscopy and Intubation**
[a] **= Highly significant (p<0.001)**
[NS] **= Not significant (p>0.05)**

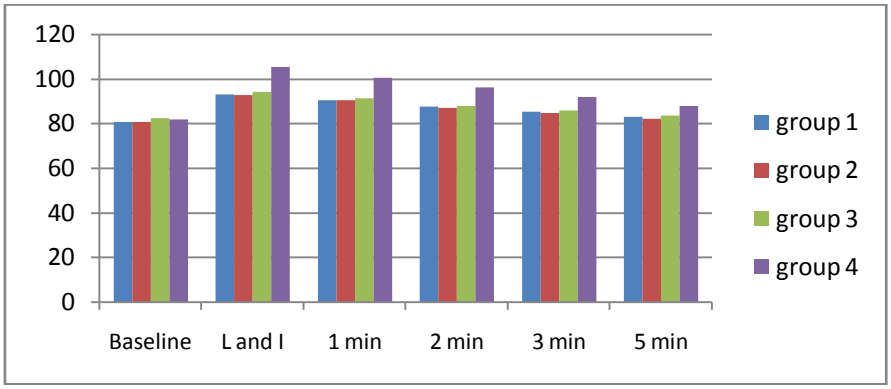
Table 9 Table showing Mean ± SD changes in Mean Arterial Pressure in groups 1, 2, 3 (ANOVA)

Mean arterial pressure	Group 1 Mean	Group 2 Mean	Group 3 Mean	P Value[#]
Baseline	95.8	96.1	97.9	0.496
After L & I[*]	108.7	109.2	110.6	0.553
After 1 MIN	106.1	106.5	107.6	0.641
After 2 MIN	103.3	103.3	104.3	0.780
After 3 MIN	100.9	100.7	101.8	0.713
After 5 MIN	98.9	98.6	100.2	0.501

[*]**Laryngoscopy and Intubation**
[#]**P values >0.05 are insignificant**

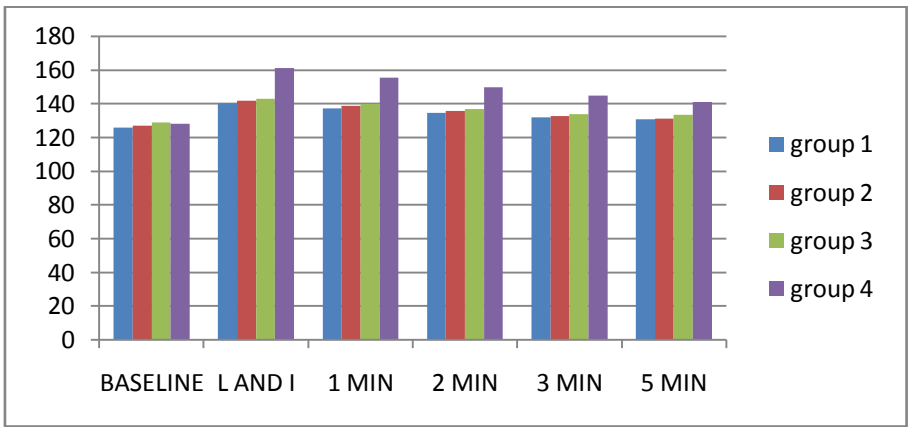
Mean ± SD changes in Heart rate in groups 1, 2, 3 (Study) and group 4 (Control)

Heart rate



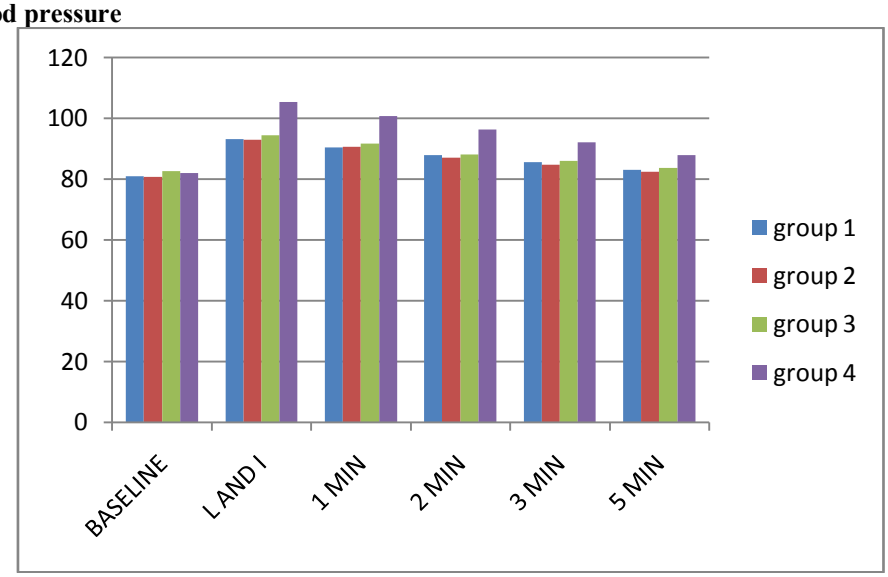
Mean ± SD changes in Systolic blood pressure in groups 1, 2, 3 (Study) and group 4 (Control)

Systolic BP

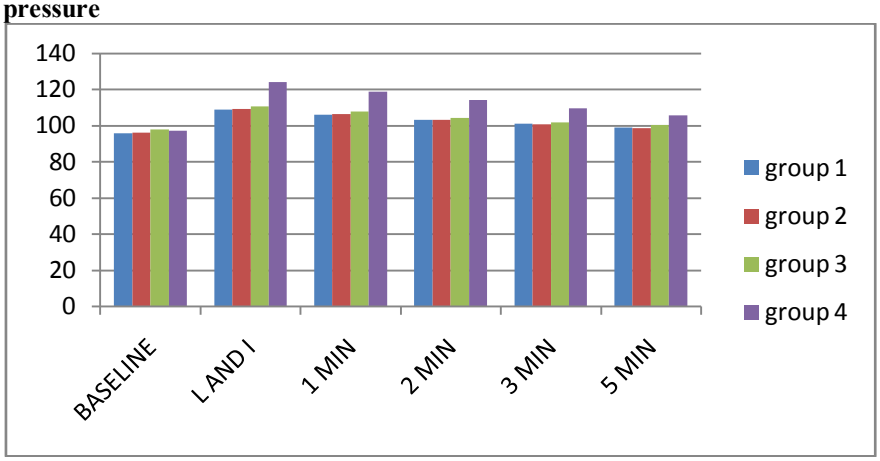


Mean ± SD changes in Diastolic Blood Pressure in groups 1, 2, 3 (Study) and group 4 (Control)

Diastolic blood pressure



Mean ± SD changes in Mean Arterial Pressure in groups 1, 2, 3 (Study) and group 4 (Control)



References

[1]. Hassan, H. G., El-Sharkawy, T. Y., Renck, H., Mansour, G. and Fouda, A. (1991), Hemodynamic and catecholamine responses to laryngoscopy with vs. without endotracheal intubation. *Acta Anaesthesiologica Scandinavica*, 35: 442–447. doi: 10.1111/j.1399-6576.1991.tb03325.x.

[2]. Mi WD, Sakai T, Takahashi S, Matsuki A,Haemodynamic and electroencephalograph responses to intubation during induction with propofol or propofol/fentanyl. *Can J Anaesth* 1998; 45:19.

[3]. Wilson IG, Meiklejohn BH, Smith G: Intravenous lignocaine and sympathoadrenal responses to laryngoscopy and intubation. The effect of varying time of injection. *Anaesthesia*. 1991 Mar;46(3):177-180.

[4]. Shribman AJ, Smith G, Achola KJ: Cardiovascular and catecholamine responses to laryngoscopy with and without tracheal intubation. *Br J Anaesth* 1987; 59:295.

[5]. King BD, Harris LC, Greifenstein FE, Elder JD, Dripps RD. Reflex circulatory responses to direct laryngoscopy and tracheal intubation performed during general anesthesia. *Anesthesiology* 1951; 12:556-566.

[6]. Stoelting RK. Circulatory changes during direct laryngosocpy and tracheal intubation - influence of duration of laryngoscopy with or without prior lidocaine. *Anesthesiology* 1977; 47(4): 381-384.

[7]. RavalDipak L., Mehta Malini K. Oral clonidine pre medication for attenuation of haemodynamic response to laryngoscopy and intubation.*Indian J. Anaesth*. 2002; 46 (2) : 124-124.

[8]. Carabine UA, Allen RW, Moore J. Partial attenuation of the pressor response to endotracheal intubation. A comparison of the effects of intravenous clonidine and fentanyl. *Eur J Anaesthesiol*. 1992 Jul;9(4):325-329.

[9]. Vucevic M, Purdy GM, Ellis FR. Esmolol hydrochloride for management of the cardiovascular stress responses to laryngoscopy and tracheal intubation. *Br J Anaesth1992*; 68: 529-530.

[10]. Braude N, Clements EAF, Hodges UM, Andrews BP. The pressor response and laryngeal mask insertion - a comparison with tracheal intubation Anaesthesia, 1989; 44: 551-554.

[11]. S. Singhal, .Neha: Haemodynamic Response To Laryngoscopy And Intubation: Comparison Of McCoy And Macintosh Laryngoscope. *The Internet Journal of Anesthesiology*. 2008 Volume 17 Number 1. DOI: 10.5580/107d

[12]. Fassoulaki A, KaniarisP.Intranasal administration of nitroglycerine attenuates the pressor response to laryngoscopy and intubation of the trachea.*Br J Anaesth*. 1983 Jan;55(1):49-52.

[13]. Lev R, Rosen P. Prophylactic lidocaine use preintubation: a review. *J Emerg Med*. Jul-Aug 1994;12(4):499-506

[14]. Abou-Madi MN, Keszler H, Yacoub JM. Cardiovascular reactions to laryngoscopy and tracheal intubation following small and large intravenous doses of lidocaine. *Can AnaesthSoc J* 1977;24: 12-19.

[15]. Watanabe H, Lindgren L, Rosenberg P, Randell T. Glycopyrronium prolongs topical anaesthesia of oral mucosa and enhances absorption of lignocaine. *British Journal of Anaesthesia* 1993; **70**: 94–5.

[16]. Mostafa SM, Murthy BVS, Barrett PJ, McHugh P. Comparison of the effects of topical Lignocaine spray applied before or after induction of anaesthesia on pressor response to direct laryngoscopy and intubation *Eur J Anaesthesiol*.1999;16:7-10.

[17]. Hamill JF,BedfordRF, Weaver DC. Lidocaine before endotracheal intubation: Intravenous or Laryngotracheal. *Anaesthesiology* 1981; 55:578-581.

[18]. Takita K, Morimoto Y,Kemmotsu O. Tracheal Lidocaine attenuates the cardiovascular response to endotracheal intubation. CJA 2001:48(8):732-736.

[19]. M. Jain, S.A. Gurcoo, A.N. Shora, M.S. Qazi, B.A. Dar, V.N. Buchh, S.I. Ahmad. Efficacy Of Topical Lignocaine Spray (10%) Applied Before The Induction Of Anaesthesia In Attenuating The Pressor Response To Direct Laryngoscopy And Endotracheal Intubation In Controlled Hypertensive Patients. *The Internet Journal of Anesthesiology*. 2009, 20(2) DOI: 10.5580/25bc

IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861. Volume 9, Issue 6 (Sep.- Oct. 2013), PP 64-67
www.iosrjournals.org

Impact of Periodontal Disease on Low Birth Weight and Preterm Birth

Dr. K. Malathi, MDS, PGDHA[1,] Dr. N. S. Nalni[2]
[1]*Head of the department of periodontics, Tamil Nadu Govt. Dental College & Hospital, Chennai.*
[2] *Post Graduate Student, Department of Periodontics, Tamil Nadu Govt. Dental College & Hospital, Chennai.*

Abstract: *Preterm birth represents a major problem in the world because of its increasing frequency and accompanying socioeconomic impact. Globally prematurity is the leading cause of newborn deaths and now the second leading cause of death after pneumonia under age of five. Periodontitis and adverse pregnancy outcomes may be linked through a chronic, systemic inflammatory challenge to the mother and fetus in response to pathogens. Several studies in the past have demonstrated an association between infection and preterm birth. However several other risk factors need to be considered. The principle reasons cited for the continued high rate of preterm and low birth weight is poor understanding of the risk factors associated. This article explains the association between preterm birth and periodontitis by stating various complications of preterm birth, pathophisiology of the associated risk factors like bacterial infection, viral infection, gene polymorphism, adaptive immune responses, preeclampsia. The early diagnostic predictors in the form of biomarkers and the effect of periodontal therapy in preventing the preterm birth are discussed. As Peridontitis is an important risk factor for preterm birth there is a need to expand preventive measures during pregnancy to avoid this adverse effect.*

Keywords: *adversepregnancyoutcome, lowbirthweight, periodontitis, pretermbirth.*

I. Introduction

Periodontal disease is a chronic destructive disease presenting as a low grade infection dominated by gram negative microaerophilic organism resulting in local and systemic inflammatory and immune response. Periodontal disease is the second most prevalent oral disease. In 19th and early 20th century the theory of focal infection was reported, which stated that the foci of sepsis were responsible for the initiation and progression of a variety of systemic diseases. Periopathogenic organisms and their products may have wide range of effects most likely mediated through stimulation of host cytokine production in target tissue.

Galloway(1931) reported that periodontal disease of pregnant women is associated with adverse pregnancy outcome . Preterm low birth weight babies is most commonly associated with periodontitis. Offenbacher et al(1996) reported an association between periodontitis and Preterm low birth weight(PTLW), stating that maternal periodontal disease could lead to 7 fold increase in risk of preterm delivery. Preterm birth(PTB) (WHO 1992) is defined as delivery before the end of 37wks of gestation(<259d). Less than 32wks is termed as very preterm and less than 28wks is termed as extremely preterm. Low birth weight(LBW) is defined as infants weighing <2500g at birth. Very low birth weight is <1500g and extremely low is <1000g.

PTB represents as a major problem for modern obstetrics because of its increasing frequency and accompanying socioeconomic impact. [(1)]This article mainly focuses on periodontal disease as a risk factor for PTB and LBW by discussing on various mechanisms leading to PTB. The influence of periodontal therapy on PTB, possible means of predicting through biomarkers in pregnant women and a few suggestions in preventing this adverse outcome are also discussed.

II. Health sequences to the Newborn with PTB/LBW[2]

2.1Short term sequences

- Respiratory distress syndrome
- Intraventricular haemorrhage
- Periventricular haemorrhagic infarction
- Necrotising enterocolitis
- Bronchopulmonary dysplasia
- Sepsis
- Patent Ductus Arteriosus

2.2 Long Term Consequenses

- Cerebral Palsy

- Attention deficit disorder
- Retinopathy of prematurity
- Mental retardation
- Cardiovascular malformation

2.3 Effect on oral growth and development.[3]

Generalized enamel hypoplasia, localized enamel hypoplasia, Crown dilacerations, Palatal distortions which are usually associated with traumatic laryngoscopy and prolonged intubation and low rate of dental development particularly before 6yrs of age are reported.

III. Risk Factors of Preterm birth.[4]

Denise M Main(1985) summarized various risk factors which were found to be associated with PTB

3.1 Demographic risk: Maternal age (<19yrs/>40yrs), education, Race (Black are 2 times more prone), Low socioeconamis status, Height of mother(<150cm), weight(<20 pounds).

3.2 Behavioural risk: Cigarette smoking, cocaine, alcohol, coffee, tea, poor nutrition, extensive physical activity.

3.3 Health care risk: Poor Obstetric history of previous PTB was reported to be associated with recurrence risk of 17-40%.

3.4 Selected medical and surgical disease: Sickle cell trait, uterine malformation, heart disease, diabetes mellitus, hypothyroidism, hyperthyroidism, nephritis, urinary tract infection.

3.5 Current pregnancy complications: Multiple gestation, placental pathology, abnormalities in amniotic fluid volume, fetal abnormalities.

3.6 Infections: Genitourinary infection[5], bacterial vaginosis, Periodontal disease as an important and independent risk factor[6].

IV. Pathophysiological mechanisms and factors associated with periodontal disease leading to PTB/LBW

4.1 Pathogenic Microflora contributing to PTB/LBW : It is suggested that oral infection may contribute upto 50% of PTB's. This may include systemic infections (Pneumania, Genitourinary tract infections, bacterial vaginosis, Chlamydia trachomatis, Syphilis)[7] and oral inflammatory diseases like Mucositis, Gingivitis and Periodontal infection[8].

4.1.1 Specific Bacterial Colonization

Some of the suggested organisms in studies favouring PTB are P.Gingivalis, T.Forsythia, T.Denticola, F.Nucleatum[9]. Chronic periodontitis is strongly associated with preterm labor, PTB and LBW. The presence of periodontal pockets, clinical attachment loss and gingival bleeding are shown to be important factors[10] .P.Gingivalis is a gram negative anaerobic bacteria and has bio active components including lipopolysaccharide capsule and fimbriae on cell surface.

Delivery is started in the late stage of gestation by events including changes of various hormones and stimulation of biomechanical molecules by fully grown fetus. The initial signals enhance the production of proinflammatory molecules including IL-6, IL-8, IL-1b. These factors lead to uterine contration, cervical ripening, directly or indirectly which leads to parturition.

Two possible mechanisms linking periodontal disease and PTLBW are

- The bacteria in periodontal lesions migrate to the materno-fetal unit via the blood and directly cause adverse pregnancy outcomes[11,7].-Lopez etal(1987), Romero etal(1982), Gibbs RS(1992).
- Proinflammatory molecules produced by the periodontal organisms before the later stages of gestation cause parturition.

Cytokines which are capable of eliciting the acute phase response, are a component of this inflammatory response and are classified as

1.Proinflammatory molecules, which initiate or enhance a cascade of events. Example TNFα(Tumour necrosis factor-α) and IL-1(interlukin).

2.Those which propagate many of the systemic manifestation of acute phase response.Example IL-6,IL-11.
3.Anti inflammatory which down regulate the acute phase response. Example IL-10,TGF-β(transforming growth factor-β)[12].

P.Gingivalis lipopolysaccharide induces IL-6, 8 production via both TLR-2 and TLR-4 in chorion derived cell. [13].Increased systemic levels of pathogenic microorganisms , their endotoxin or directly via inflammatory mediators especially Prostaglandin E2($PGE_{2)}$ contribute to PTB/LBW.[14] The actions of prostaglandin are effected through specific receptors. PGE2 induces myometrial contractions by binding to EP-1 and EP-3 receptors, which mediate contractions through mechanisms that lead to increased calcium mobilization and reduced levels of production of inhibition of intracellular cAMP. Prostaglandins also enhance the production of matrix metalloproteinases (MMP) in the cervix and decidua to promote cervical ripening and decidual and fetal membrane activation. PGF2α binds to FP receptors to induce myometrial contractions. In contrast, in the lower uterine segment PGE2 induces myometrial relaxation by binding to EP-2 and EP-4 receptors that increase the level of cAMP formation.

Prostaglandins are formed from arachidonic acid by PGHS(prostaglandin H2 synthetase). In turn, prostaglandins are metabolized to inactive forms by the actions of PGDH. Cortisol, CRH(corticotropic releasing hormone), and estrogens stimulate PGHS activity and cortisone and CRH also inhibit PGDH expression. Thus, increases in fetal steroid hormone production following fetal HPA(Hypothalamic pituitary axis) activation leads to a net increase in prostaglandin levels. Similarly, proinflammatory cytokines such as IL-1 and
tumor necrosis factor alpha (TNF-α) up-regulate PGHS expression and down-regulate PGDH expression leading to prostaglandin synthesis associated with preterm delivery in the setting of infection[15]

Systemic antibody Responses to selected periodontal Bacteria[16].

Oral bacteria trigger local and systemic adaptive immune responses in healthy adults and childre.PTB is associated with low levels of IgG antibody to periodontal pathogens in women with periodontitis.Madionos et al, found that elevated IgG antibody to certain oral bacteria in mothers serum was related to decreased rate of PTB and increased birth weight .Decreased levels of antibody to P.Gingivalis are reported in women during second trimester, who delivered at preterm. Increased levels of serum antibody to F.Nucleatum were elevated in women who suffered fetal loss.

4.I.2 Human Immuno deficiency virus-a factor for PTB[17]

There is a positive risk of having adverse neonatal outcomes in HIV infected women who had moderate periodontitis .In the of (HAART) Highly active anti retroviral therapy, a combination of anti retroviral medications administered during preconception period increased of PTB/LBW.

II. Gene polymorphism as a factor to cause PTB[18]

FCγR11b(CD32B) is a human type 2 low infinity receptor of IgG .This receptor is encoded by three highly homologous genes-FCRY11a,FCYR11b,FCYR11c, that are clustered on chromosome Iq23.One of the polymorphism,FCγR11b-1232T, in patient with periodontitis was associated with increase in serum specific I_gG_2 levels against the outer membrane protein from P.Gingivalis.Thus FCYR11b gene polymorphism may mediate the relationship between periodontitis and PTB. FCγR11b-nt645+25AA carriers are more likely to experience preterm birth than FCγR11b-nt645+25AG and GG carriers. Also women with FCγR11b-nt645+25AG exhibited a greater tendency to have periodontitis than those with FCγR11b-nt645+25A.

4.3 Pre-eclampsia poses a risk factor of PTB[19]

Pre-eclampsia is usually defined as maternal systolic pressure>/= 140mmHg or diastolic pressure>/=90mmHg with proteinuria. The syndrome is characterized by inappropriate inflammatory and abnormal vascular response to placentation which causes endothelial dysfunction resulting in maternal hypertention during pregnancy(Sibbas et al).The hypothetical mechanism is that inflamed periodontal tissue release elevated levels of C-Reactive protein and other inflammatory mediators(PGE_2) which enter the systemic circulation and induces inflammation that damages the placenta and causes pre-eclampsia

V. Early Prediction and Possible Prevention of PTB

Periodontal disease is a source of persistent infection and raises the plasma levels of CRP(C reactive protien) by 14wks of gestation(Pitiphat et al)[20].Depending on the source of the sample(maternal serum,amniotic fluid, vaginal swabs) many diagnostic markers can predict the PTB before 21wks of gestation.Ex: wbc count, IL-1b, IL-6, IL-8 , alkaline phosphatase in serum [21] and fibronectin in cervical /vaginal secretions indicates the border between the chorion and deciduos has been destructed.

BANA Test[22] :P.Gingivalis, T.Forsythia, T.Denticola, posses a trypsin like enzyme that can hydrolyse the synthetic trypsin substrate- Benzoyl-DL arginine-Naphthylamide(BANA).The presence of these organisms

in the sub gingival plaque hydrolyse BANA using a 5minute chais side assay. The third trimester bacterial status of subgingival plaque may be an important predictor of PTBs.

VI. Non-Surgical Periodontal therapy in pregnant mothers.

Non-Surgical Periodontal Therapy which includes Scaling, root planning, plaque control decreases the levels of inflammatory cytokines[23].This serves as a protective factor promoting the birth of children with normal weight.

VII. Conclusion

PTB / LBW and thus the prematurity is responsible for 70% of perinatal deaths(Goldenberg et al).It occurs in about 10% of pregnancies and is responsible for 50% of neurological disorders in the new born(McCornick1985). Periodontal disease is an independent risk factor for adverse outcome of pregnancy.Non-Surgical periodontal therapy significantly reduces the rate of PTLBW and improved the general health of women with periodontal disease.The various biomarkers suggested may be used as tools to predict the risk associated and early intervention may be done to prevent the adverse outcome. In HIV associated periodontitis the pathophysiological mechanism that relates the viral load and preterm birth requires further conformational studies. The earliest biomarker of PTB associated with periodontitis and periodontal therapy to completely avoid this adverse outcome may challenge the scope for further research.

References

[1] WHO-International statistical of diseases and related health problems.*Tenth vision*. Geneva: WHO 1992.

[2] Bryan S.Michalowtz and Robert Dwand,Maternal periodontal disease and spontaneous preterm birth*, Periodontology 2000,44*, 2007,103-112.

[3] W.Kim Seow MDSc, DDSc, Phd, FRACDS, Effect of preterm birth on oral growth and development, *Australian dental journal*, *42(2)* ,1997,85-91.

[4] AN Guimaraes, AS Mato, LO Miranda Cota, FM Siqueira and FO corta, Maternal periodontal disease and preterm or extreme preterm birth: An ordinal logistic regression analysis, *Journal of Periodontology, 81(3*), 2010, 350-358.

[5] LA.Schieve, A.Handler, R.Hershow, V.Persky, F.Davis, Urinary tract infection during pregnancy, its association with maternal morbidity and perinatal outcome, *American journal of publichealth, 84(3*), 1994, 405-410.

[6] J.K.Baskaradoss, A.Geevarghese, V.R.Kutty,Maternal Periodontal status and preterm delivery a hospital based case control study, *Journal of periodontal research, 46(5*), 2011, 542-549.

[7] Lorief. Cram, Maria-Isabel Zapata, Eugene C. Toy, and Benton Baker *Am Fam Physician*. 2002 Jan 15;65(2):241-249.,.

[8] Auro Heimonen, Sok-ja-Janket, Risto Kajaa, Leland K Ackerson, Preetika Muthukrishnan, Jukka H.mausman, Oral inflammatory burden and preterm birth, *Journal of Periodontology, 80(6),*2009, 884-891.

[9] Hui-chen chan, Chen-trai Wu, Kathleen B.Welch, and Watter J.Loeshe, Periodontal disease activity measured by the Benzoyl-DL-Arginine Naphthilamide test is associated with preterm births, *Journal of Periodontology, 81(7),* 2010, 982-991.

[10] SA.Santos Pareira, Gealdo.PC, Eduardo Saba, RLG Amaral, SS Morais, AM Gonlalalics, Chronic Periodontitis and preterm labour on Brazilian pregnant women : an association to be analyzed. *Journal of clinical Periodontology, 34(3*), 2007, 208-213.

[11] Bryan.S.Michalowicz, M.John Novak, James s. Hodger, Antony Di Angels, William Buchanan, Panos N.Papanou etal, Serum inflammatory mediators in pregnancy, changes after periodontal treatment and association with pregnancy outcomes, Journal of *Periodontology, 80(11*), 2009, 1731-1741.

[12] K.Hasegawa Nakamura, F.Jateishi, T.Nakamura, Y.Nakajima, k.kawamata , T.Douchi etal, The possible mechanism of preterm associated with periodontopathic P.Gingivalis*, Journal of periodontal research,46(4*), 2011, 497-504.

[13] Romero R, HObbins JC, Mitchell MD, Endotoxin stimulates PGE_2 production by human amnion, *Journal of Obstetrics Gynynecology,71*,1989,211-218.

[14] Offenbacher S, Jared HL, Reilly PG etal, Potential Pathogenic mechanism of periodontal associated pregnancy complications, *Ann Periodontology,3(1),* 1998, 233-250.

[15] Behrman RE, Butler AS, Institute of medicine(us) Committee on understanding premature birth and assessing healthy outcomes (Ed), Biological Pathways leading to Preterm birth, (*Washington DC National Academis Press(US):2007*).

[16] Jeffrey L.Eberson, M.John Novah, Bryan SM, James S. Hodger, Mitchelle J.Steffen, James E Fergusson etal,Systemic Immune Responses in pregnancy and Periodontitis.Relationship to pregnancy outcomes in obstetrics and periodontal therapy(OPT) study,*Journal of Periodontology, 80(6*), 2009,

[17] Pakkaporn Pattrapornan, Timothy A. Deroven, Yupin Songpaissan, Increased risks of preterm birth and a low birth weight baby in Thai Human immunodeficiency Virus-Positive pregnant women with periodontitis, *Journal of Periodontology,83(11),* 2012,1372-1381.

[18] R.Iwanga, N.Sugita, E.hirano, J.Sasahara, A.Kikuchi, K.Tanaka, etal, FCγR11B ppolymorphisms, periodontitis and preterm birth in Japanese pregnant women, *Journal of periodontal research, 46(3*), 2011,292-302.

[19] C.Nabet, N.Lelong, ML Colombrie, M Sixon, AM Goffinet, F Kamineh M etal, Maternal periodontitis and cause of preterm birth, case control epipap study, *Journal of Periodontology,37(1*), 2010, 37-45.

[20] Waranuch Pitiphat, Mathew W .Gilman, Kaumudi J.Joshipura etal, Plasma C-reactive protein in early pregnancy and preterm delivery, *American Journal of Epidemiology, 162(11*), 2005, 1108-1113.

[21] Stephen W. Looney, Treatment of periodontal disease and risk of preterm birth, *Journal of Periodontology, 79(1*), 2008, 1-2.

[22] Francois Goffinet, Primay predictors of preterm labour, *International journal of Obstetrics and gynecology,112(1*), 2005, 38-47.

[23] Fouzia Tarannum and Mohammed Faizudin, Effect of periodontal therapy on pregnancy outcomes in women affected by periodontitis, *Journal of periodontology,78(11*), 2007.

IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861. Volume 9, Issue 6 (Sep.- Oct. 2013), PP 68-72
www.iosrjournals.org

Obstructive Sleep Apnea -An Orthodontic review

Dr. Sathish Kumar N, Dr. Divya.K, Dr. Appasaheb Naragond,
Dr. Smitha Naragond, Dr. K. Rajasigamani, Dr. V. Baskar
(Assistant Professor & HOD Department of Dentistry, SVMCH & RC, Ariyur,Puducherry, India)
(Assistant Professor & Department of Emergency Medicine, SVMCH & RC,Ariyur, Puducherry, India)
Reader (Department of Orthodotic &Dentofacial orthopedics Vasantdada Patil dental College and Hospital Kavalapur -Sangli) India
Senior Lecturer Dept.Of Conservative and Endodontics Vasantdada Patil dental College and Hospital ,Kavalapur -Sangli) India
Hod,Prof Department of Orthodontics and Dentofacial Orthopedics, RajahMuthiah Dental College and Hospital, Chidambaram, Tamil Nadu, India
Profesor Department of Orthodontics and Dentofacial Orthopedics, RajahMuthiah Dental College and Hospital, Chidambaram, Tamil Nadu, India

Abstract: *Obstructive Sleep Apnea is caused by an interplay between a variety of factors, including sleep related loss of muscle tone in the tissues supplied by the glossopharyngeal nerve,, anatomical obstruction of the nasal passages, large tonsils, large tongue, a retrognathic Mandible, obesity, alcohol, sedative medication, allergies. The orthodontic approach is intended to provide patients with immediate relief from OSA, as well as changes to the airway that may address an underlying cause. It can be treated using surgery, continuous positive airway pressure and oral appliances therapy. This article review some of the basic aspects of this sleep-related disorder, its diagnosis and treatment modalities.*

Keywords*: Orthodontics, Obstructive Sleep Apnea, mandibular advancement, Oral appliance, CPAP*

I. Introduction:

Snoring sound is produced by the vibration of the soft palate or other oropharyngeal tissues, it can became a medical concern because it is key symptom of OSA. Disease is characterized by upper airway obstruction, associated with repeated gaps in breathing and interrupted sleep. Cessations of breathing for 10 seconds or longer are termed apneas (from greek- without breath), When 30 or more apneic episodes occur in the course of seven hours of sleep, resulting in excessive sleepiness during the working hours, a person is described as having sleep apnea syndrome. This may begin at any age, but incidence increases with age. Anatomic factors that can contribute to OSA are maxillary or mandibular retrognathism, increased lower facial height, large tongue elongated soft palate, inferiorly positioned hyoid bone. This points out how critical the role of orthodontist can be in diagnosing and treating OSA.

The first description of this disorder in the medical literature was in 1965. effective treatment that modify these health risks have emerged. Although continuous positive airway pressure (CPAP) is the most efficacious treatment. It requires the used of a mask interface, sealed tubing , and a device connected to a power source. This complexity limits its acceptance by patients and leads to suboptimal treatment adherence. Oral appliances are a simpler alternative to CPAP for the treatment of Obstructive sleep
Apnea (OSA). They are often considered by patients to be a more acceptable treatment modality compared to CPAP. As they are quiet, portable, and do not require a power source,. While the role of oral appliances for the treatment of OSA was unclear in the past, this has changed dramatically. There are served modalities of treatment for obstructive sleep apnea. The use of the oral appliances for management of airway obstruction started in 1900 when the French stomatologist Pierre Robin used oral appliances to manage upper airway obstruction of neonates born with Pierre Robin syndromes.

II. Pathophysiology

It has been hypothesized genioglossal that individuals with OSA have impaired genioglossal function, allowing the prolapse of the tongue against the posterior pharyngeal wall with inspiratory effort during sleep.

The situation now appears to be more complicated – evidence suggests that an invagination of the pharyngeal walls and a general hypotonia of the dilating muscles of the upper airway can also be involved in allowing airway occlusion during sleep.. The nasal airway can also play an important role in total airway occlustion. Nasal obstruction increases resistance to air flow, which in turn results in increased inspiratory effort and greater negative pressure in the pharyngeat airway. This suction increases the likelihood of collapse of the pharyngeal airway. Various factors which predispose to obstructive sleep apnea, the most important

being obesity structural abnormalities in the face, skull, or airways that cause some obstruction or collapse in the upper airways and reduce air pressure can produce sleep apnea syndrome. People with micorgnathia, adenoids, retrognathia, enlarged tonsils, tongue enlargement, acromegaly and longer anterior facial height are especially predisposed to obstructive sleep apnea.

III. Symptoms;

Snoring (OSA is unlikely in the absence of habitual snoring)
Apneic pauses (choking, gasping, snoring during the night)
Restless leg syndrome (RLS) restless sleep and increased body movements
Bruxism (nocturnal tooth grinding)
Nocturnal and daytime enuresis
Sleep position (side and stomach sleepers) or neck hyper extended
Growth failure restriction
Sleep walking or sleep terrors
Obesity
Daytime symptoms can include:
Mouth breathing, due to adenoidal hypertrophy, and dry mouth
Chronic nasal congestion ,rhinorrhea
Adenotonsillar hypertrophy
Hyponasal speech ,Fatigue
Excessive daytime sleepiness: difficulty waking or falling asleep at school
Mood changes; ireitability, low frustration tolerance, impatience, depression anxiety, and social withdrawal, a negative sense of well being
Acting-out beaviors including aggression and hyperactivity
Cognitive impairment and poor school performance
Inattention, poor concentration, and distractibility
ADHD-like symptoms
Infraorbital venous congestion

IV. Diagnosis;

A patient is suspected of having OSA , the diagnosis is confirmed by an overnight polysomnography (PSG), commonly referred to as a sleep study. Communicate this recommendation to the patient's physician. If the patient refuses a sleep study, he recommends having the patient sign a waiver prior to providing an oral appliance.

Once a sleep study is undertaken, the resulting data is the Respiratory Disturbance Index (RDI). This is a somewhat complex index that measures the number of apnic (total cessation of breathing) and hypopnic (shallow breathing) events per hour of REM and non REM sleep. For example, someone who has an RDI of 30 has 30 apnic or hypopnic events an hour. The range of RDIs is as follows. RDI < 5 Normal RDI 5-15 Mild; RDI 15-30 Moderate: RDI> 30 Severe.

V. Treatment

1. ORAL APPLIANCE THERAPHY:

Orthodontic appliances are made in such a manner that it can be worn permanently or removeably depending upon the condition.. Appliance are designed to bring the mandible and tongue forward, opening up the lower pharynx to allow unrestricted breathing..

2. INDICATIONS OF ORAL APPLIANCE THERAPY;

Oral appliance are indicated for use in patients with primary snoring or mild OSA who do not respond or are not appropriate Candidates for treatment with behavioral measures such as weigh loss or sleep position change; Patients with moderate to servere OSA should have an initial trial of nasal CPAP because greater effectiveness has been shown with this intervention that with the use of oral appliances; Oral appliances are indicated for patients with moderate to severe OSA who are intolerant of, or refuse treatment with, nasal CPAP, oral appliances are also indicated for patients who refuse or who are not candidates for tonsillectomy and adenoidectomy , cranial facial operations or tracheostomy.

3. ORAL APPLIANCES;

Dental devices include tongue retaining devices (TRD) and mandibular advancement appliances (MAA). Tongue retaining device is a splint that holds the tongue in place to keep the airway as open as possible. Mandibular advancement device (MAA)(Fig.1) is by farther most common type of dental appliance in

use today. It protrudes the mandible forward, thus preventing or minimizing upper airway collapse during sleep.Mechanism of action- oral appliances are worn only during sleep and they help to maintain an open and unobstructed airway by repositioning or stabilizing the lower jaw, tongue, soft palate or uvula.Mandibular advancement devices – as mentioned earlier the first use of mandibular advancement devices was suggested by Pierre Robin in 1903. it protrudes the mandible forward, thus preventing or minimizing upper airway collapse during sleep.(Fig.2)

Currently available appliances:

First category: one piece appliance with no ability to advance the mandible incrementally.

Second category: Appliance are principally tow piece in design and offer the potential for incremental advancement.

Third category : They permit incremental advancement and lateral movement of mandible.

Tongue retaining devices: Tongue retaining device is a splint that holds the tongue in place to keep the airway as open as possible. They are excellent devices for patients with Temperomandibular joint sensitivity. There are several advantages of Tongue retaining devices, they do not require retention from dentition, minimal adjustments are required and cause minimal sensitivity to teeth and temperomandibular joint.

Advantage of Oral appliances:

Significant reduction in apneas for those with mild-to-moderate apnea, they may also improve airflow for some patient with severe apnea, improvement and reduction in the frequency of snoring and loudness of snoring in most patients and higher compliance rates than with CPAP.

Disadvantages of Oral appliances:

Mandibular advancement splints generate reciprocal forces on the teeth and jaw that can result in acute symptoms, as well as long-term dental and skeletal changes. While mandibular advancement splints are primarily attached to the dental arches, most extend beyond these and thus apply pressure to the gums and oral mucosa. The incidence of reported side effects and complications vary significantly between studies. This is probably due to difference in the type of oral appliance used, the design of the oral appliance, the degree of mandibular advancement, as well as the frequency and duration of follow-up.

During the acclimatization period, it is common for adverse effects to develop, which are usually minor and self-limiting. These include execessive salivation, mouth dryness, tooth pain, gum irritation, headaches, and temporomandibular joint discomfort. Patients should have regular visits with a health professional to check the devices and make adjustments:.

4. SKELETAL SURGERY:

Surgical correction of the jaw position is the most effective treatment for OSA, with good long-term stability. Skeletal surgery can involve maxillary and mandibular expansion and/or maxillary and mandibular advancement. Orthognathic surgery primarily involves advancements usually have to be over 10mm to be effective in treating OSA, so, typically maxillay advancement is also necessary.

5. SOFT TISSUE SURGERY:

Another procedure that has been used is the uvulopalatopharyngoplasty (UPPP) the reconstruction of the throat by resecting te posterior margins of the soft palate and redundant mucsosa on the lateral pharyngeal walls. Unfortunately, the success rate for this approach to sleep apnea is only 40% because obstructions at other sites are not affected.

6. CPAP:

A continuous positive airway pressure machine (CPAP) is a new device with a mask that fits snugly over the sleeper's nose. It sends a continuous stream of air under positive pressure that is adjusted for each person using it to hold the throat open through the night.

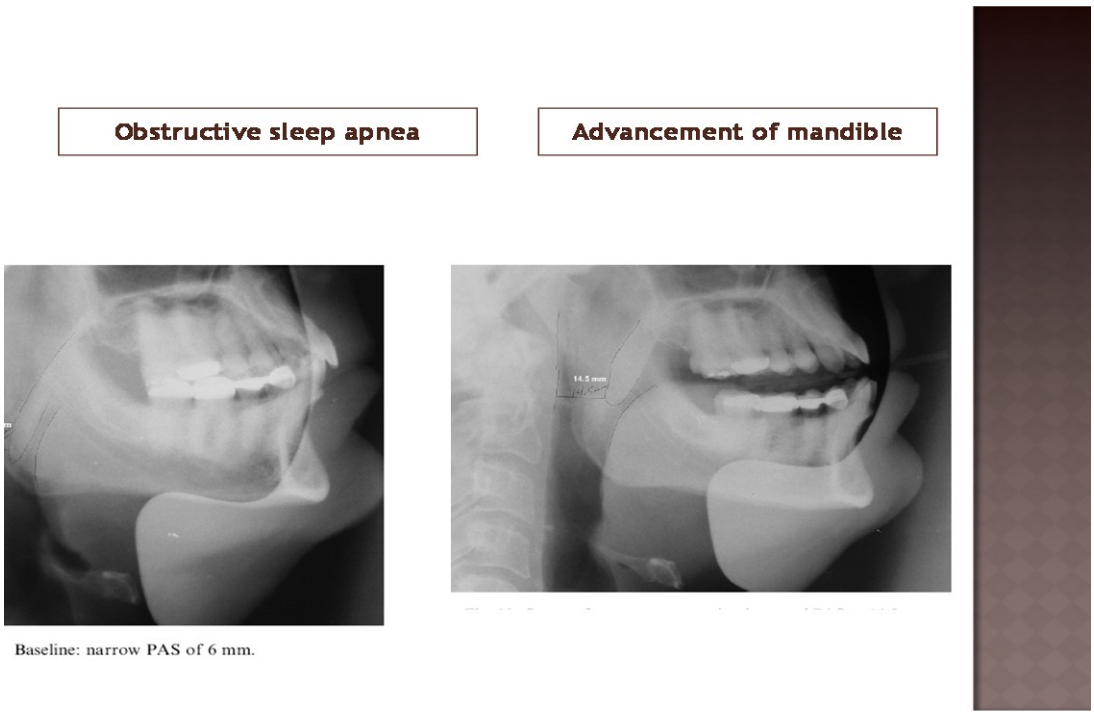


Figure.01 PAS increased after advancement

Figure.02

Oral Appliance for OSA

VI. Summary And Conclusions

OSA is characterized by breathing and sometimes pronounced body and arm movements. The victim may wake up suddenly with choking sensations, gasping for air, or in a sweat, other symptoms may be frequent napping during the day, especially in inappropriate places (like meetings, or while driving), memory problems, lack of concentration, high blood pressure….. Many of these symptoms can also be caused by many other conditions, so diagnosis may be difficult. Orthodontic diagnosis may discover anatomic conditions that could cause this condition. Enlarged tonsils or adenoids in a lateral cephalometric radiagraph, or maxillary width

deficiency and narrow nasal cavity in a P.A. radiograph, are indications for questioning the patient about other symptoms. If obstructive sleep apnea is suspected, a medical consultation is in order. It is important to distinguish between central and mixed sleep apnea. CENTRAL sleep apnea involves a cessation of respiratory effort, as well as reduced air flow. This condition is relatively rare. MIXED sleep apnea might involve two hundred obstructive and twenty kinds of central apneas, so some A.C. P's group them together as mixed apneas. The belief is that the central apneas in the [category are a secondary effect of the reduced air flow caused by the obstruction rather than a primary etiologic factor.

OSA is simply rendered ineffective by the obstruction. It is in this condition that the orthodontist may best participate in relieving or curing the symptoms. Developments have been reflected in the updated practice parameters of the American Academy of Sleep Medicine, which now recommend the use of oral oppliances for mild-to-moderate OSA, or for patients with servere OSA who are unable to tolerate CPAP or refuse treatment with CPAP. With this review article we research should focus on determining the influence of the design of oral appliances on clinical outcome, the development of a clinically reliable method for identifying those patients who are most likely to achieve a favorable treatment response, and the characterization of facotors predisposing to long-term adverse effects of oral appliance treatment.

Bibliography

[1]. Guilleminault, Christian, 1985, "Obstructive sleep apnea", Med Clin, N.AM 69-1187-1203. .
[2]. Guilleminault, Cand Dement, W.C. 1978 "sleep apnea Syndromes," Kroc Foundation Series, Vol 11,Newyork Alan R.Liss.
[3]. Kales, A, Bider, E.O. Cadieux, R.J. etal/1984, Sleep apnea in a hypertensive population.Lancet.2.1005-1008
[4]. Cistulli PA, Sullivan CE "Pathophysiology of sleepapnea" In :Saunders NA, Sullivan CE, eds. Sleep and breathing, Newyor, NY: Marcel Dekker, 1994:405-448.
[5]. Bilwise, D.I, and Pascualy, R.A 1984, "Sleep –related respiratory disturbance in elderly persons",Compr Ther, 10(7):8-14.
[6]. Cortese S, Konofal E, "Sleep and alertness in children with attention – deficit/hyper activity disorder :a systemic review of literature." Sleep 2006:29:504-511.
[7]. Peltinaju T. "The effect of mode of breathing on crantiofacial growth-revisited", Eur J Orthod 2007;29-426-429.
[8]. Alexandar, J.K. Guthrie, A.E. , 1959 "Lung volume changes with extreme obesity".Clin. Res 7;171-176..
[9]. Schwab RZ, Gupta KB. "Upper airway soft tissue anatomy in normal and patients with sleep disordered breathing: Significance of the lateral pharyngeal walls " Am J Respir; criti care med 1995; 152;1673-89.
[10]. Alexander J.K Amad, H, "Observations on some clinical features of extreme obsecity, with particular reference to cardiorespiratory effects", Am J. Med.32:512-517.
[11]. Moran, W.B and Orr, W.C 1985 "Diagnosis and management of obstructive sleep apnea", Part-II Arch Otolaryngol. 111(10): 650-658.
[12]. Bornstein SK, "Respiration during sleep polysomnography.' Addision – Wesley; 1982 p.183.
[13]. Dillon JE, Blunden S "Diagnosis and obstructive sleep apnea in children before and one year After adeno tonsillectomy". J Am Acad child Adolesc Psychiatry 2007;46;1425-36.
[14]. Gastaut H, Tassinari CA, Duron B, "Polygraphic study ofdiurnal and nocturnal (hypnic and respiratory) episodal manifestations of Pickwick syndrome" . Rev Neurol (paris) 1965; 112:568-579
[15]. Cistulli PA, Grunstein RR. "Medical devices for the diagnosis and treatment of obstructive Sleep Apnea". Expert Rev Medical Dev 2005; 2;749-763.
[16]. Mimi Yow, "Overview of oral appliances and managing the airway in obstructive sleep apnea".Semin orthod. 2009;15: 88-93.
[17]. Cistulli PA,Gotsopoulos H. "Treatment of snoring and obstructive sleep apnea with mandibular repositioning appliances" Sleep med rev 2004;8;443-57.
[18]. Ferguson KA, Rogers, "Oral appliance for snoring and obstructive sleep apnea: review" Sleep 2006;29;244-262.
[19]. Ng ATm ,Gotsopoulos H "Effect of oral appliance theraphy on upper airway collapsibility in obstructive sleep apnea" Am J Respir Crit Care med 2003;168;238-241.
[20]. Lowe AA "Oral appliance for the treatment of snoring and obstructive sleep apnea" J Can Dent Assoc 1999;65:571-574.
[21]. Oktay H,Ulukaya E , "Maxillary protraction appliances effect on the size of the upper airway passage" Angle orthod 2008;78;209-214.
[22]. Usumez S, Orhan M "Effect of rapid maxillary expansion on nocturnal enuresis" Angle orthod 2003;73;532-538.
[23]. Kurol J, Modin H "Orthodontic maxillary expansion and effect on nocturnal enuresis" Angle orthod 1998;68;225-232.
[24]. Wei JL, Smith HJ. "Improved behavior and sleep after adenotonsillectomy in children with sleep disordered breathing" Arch Oto laryngol Head Neck Surg 2007;133;974-979.
[25]. Thawley, Stanley E 1985 "Surgical treatment of obstructive sleep apnea" Med. Clin. Of N.Am 69;1337-58.
[26]. Hamada T, Ono T "Mandibular distraction Osteogenesis in skeletal class II patient withobstructive sleep apnea" Am J Orthod Dento facial Orthop 2007;131;415-425.
[27]. Barnes M, MeEvoy RD, "Efficacy of positive airway pressure and oral appliance in mild to moderate obstructive sleep apnea" Am j Respir Crit Care Med 2004;170;656-664.
[28]. Weaver TE, Chasens ER "Continuous positive airway pressure treatment for sleep apnea in older adults", Sleep Med Rev 2007;11;99-111.
[29]. Mc Ardle N, Devereux G "Long term use of CPAP therophy for sleep apnea/hypopnea syndrome" Am J Respir Crit Care Med 1999;159;1108-1114
[30]. Robertson CJ, "Dental and skeletal changes associated with long term mandibular advancement", sleep 2001;24;531-537.
[31]. Marklund M. "Predictors of long-term orthodontic side effects from mandibular advancement devices in patients with snoring and obstructive sleep apnea," Am J Orthod Dentofac Orthop 2006; 129;214-221.

IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861. Volume 9, Issue 6 (Sep.- Oct. 2013), PP 73-79
www.iosrjournals.org

Evaulation of Surgical Treatment of Fracture Capitullum (Review Report).

Dr. Sunil V Patil, Dr. P.B. Magdum, Dr. Vishwas Phadake,
MS , Mch.(Ortho),MBA (HR). Assoc Prof Bharati Vidyapeeth University Medical College & Hospital, Sangli.
Prof Bharati Vidyapeeth University Medical College & Hospital, Sangli.
Asst. Prof Bharati Vidyapeeth University Medical College & Hospital, Sangli

I. Introduction.-

Fractures of the capitellum are rare. The complete capitellar fracture pattern was first described in the 19th century (1853) by doctors Hahn and Steinthal; the eponym for this fracture pattern includes their names. Later, doctors Kocher and Lorenz described an additional variation of this fracture pattern; a classification system includes their names.

Because of the rarity of capitellar fractures, controversies exist regarding the most appropriate treatment. The fracture fragment is intra-articular and requires treatment and reduction to reestablish normal elbow motion. Difficulty arises from the varying sizes of the fracture fragment and from the amount of suitable subchondral bone that is present to achieve stable fixation and to allow early elbow motion. Failure of adequate intervention may result in an incongruous joint, as well as in stiffness, instability, and chronic pain.[1]

Treatment modalities vary from conservative, in the form of closed reduction[6,15] and immobilization, fragment excision to open reduction and internal fixation[12] with K wires and 4 mm partially threaded cancellous screws or Herbert screws.[9,18]

Open reduction and stable internal fixation helps in early mobilization[17], preventing stiffness of the elbow, and subsequent degenerative arthritis, as the articular congruity is maintained by anatomical reduction. Extension of the capitellar fracture well medially into the trochlea is reported and currently classified as the fourth type.

This is also described as the coronal shear fracture. The fragment is intra-articular, usually, without any soft tissue attachments. If not properly treated it results in malunion interfering with flexion of the elbow. The fragment has to be anatomically reduced and properly stabilized to prevent articular incongruity and late onset arthritis[2]. A proper classification helps in pre-operative planning and execution of the surgical stabilization.

As early as 1935, Mazel described capitellum fracture as a layer of bone with a portion of trochlea attached to it. Capitellar fractures are classified into three types.

Type 1 (Hahn-Steinthal fracture) which consists of a large fragment of cancellous bone of the articular surface of capitellum and may include a portion of the trochlea, typically the lateral third;

Type 2 fracture (Kocher-Lorenz fracture) which is cartilaginous articular fracture of the capitellum and may include a small fragment of sub-chondral bone typically described as "uncapping" of the capitellum;

Type 3 (Broberg and Morrey), a comminuted capitellar fracture.

If the fracture extends to more than the lateral half of the trochlea it is considered a separate entity. Another fracture, described only in children, the "sleeve fracture," has also been reported.

As per AO classification, these will be classified as B3.1 (capitellar fractures), B3.2(trochlear) and B3.3 (capitellar and trochlear fractures).

- Bryan and Morrey classification[7]
  - Type I: (Hahn-Steinthal fracture)[24]
    - Complete fracture of capitellum
  - Type II: (Kocher-Lorenz fracture)
    - Superficial osteochondral fracture fragment
  - Type III
    - Comminuted
- McKee modification[8,20]
  - Type IV
    - Coronal shear fracture including capitellum and trochlea[25]

Frequency

Capitellar fractures account for 0.5-1% of all elbow fractures and 6% of all distal humeral fractures.[4] Capitellar fractures are seen with greater frequency in females than in males; this is thought to be secondary to a greater carrying angle and an increased possibility of osteoporosis in females. In 20% of patients with capitellar

fractures, radial head fractures also are found.[5]Capitellar fractures do not occur in children younger than 10 years[14]. Because of the cartilaginous composition of the capitellum in children, a similar injury in a child would be a supracondylar or lateral condylar fracture.[13]

Fractures of the capitellum occur in the coronal plane. Separating the capitellum from the lateral column, capitellar factures are the result of shear forces from a fall onto the outstretched hand or of a fall directly onto the elbow. The capitellum is susceptible to shear forces because its center of rotation is 12-15 mm anterior to the humeral shaft. Capitellar fractures may be associated with radial head fractures and posterior dislocations of the elbow.[5,21]

II. Material & Methods:-

Between 2007 and 2010 fourteen patients with 10 right sided type IV capetullar fracture & 4 Left Sided fractures were treated in Bharati Hospital ,Sangli. There were Eight males aged between 15 to 25 and Six women age gr from 25 to 38 years. A double arc sign in the lateral views of the X-rays of the elbow was seen in all the cases. Almost all cases operated with in 3-5 days of Trauma. When plain Xrays were misleading only those cases were subjected to CT scan examination.

Under tourniquet, using extended lateral (Kocher's) approach, The extensor origin was elevated in all cases subperiosteally including the origin of the extensor carpi radialis longus. The origin of the lateral collateral ligamentous complex from the lateral epicondyle was not disturbed. The exposure is extended distally between the anconeus and the extensor carpi ulnaris. Keeping the forearm pronated the extensor carpi ulnaris is elevated anteriorly. This allows the surgeon to reflect the soft tissues to keep the bone levers over the medial column. The extensive exposure aided in keeping a bone lever over the medial aspect of the distal humerus, thus helping in visualization of the entire articular surface of the distal humerus.

The fracture was reduced by checking the anterior articular surface, and held reduced with smooth K wires open reduction and internal fixation was done using 4mm partially threaded AO cancellous screws (n=06) and 2.7 mm AO screws (n=8)(Herbert Screws), under vision from posterior to anterior direction from the posterior aspect of lateral condyle of humerus avoiding articular penetration according to the fracture anatomy & ease of Fixation.

Plaster of Paris (POP) slab was given in all cases with elbow at 90 degrees of flexion and the forearm in neutral rotation. The patients were mobilized out of posterior slab after three weeks. Range of motion exercise was started under supervision of physiotherapist after six weeks. Clinical and radiological follow up was done at six weeks, three months, six months and one year. The elbows were tested for range of movements, and instability.

III. Results:

All the fractures united uneventfully. At the end of one year follow-up, twelve cases had excellent elbow function; implants were removed and there were no signs of AVN or arthritis. The other two cases had good elbow ROM at 11 months without AVN. The results were analysed by the Mayo Elbow Score.

Mayo Elbow Performance Score

Section 1 - Pain Intensity
None
Mild
Moderate
Severe

Section 2 - Motion
Arc of motion greater than 100 degrees
Arc of motion between 50 and 100 degrees
Arc of motion less than 50 degrees

Section 3 - Stability
Stable
Moderate instability
Grossly Unstable

Section 4 - Function (Tick as many as able)
Can comb hair
Can eat
Can perform hygiene
Can put on shirt
Can wear shoe
Interpreting the Mayo Elbow Performance Score

Interpreting the Mayo Elbow Performance Score

Score greater than 90 Excellent **Score 75-89** Good **Score 60-74** Fair **Score below 60** Poor

Sr No	Age/Sex	MOInjury	Xray	CTscan	Followup	Type Of Fixation	Functional Results.
1	27/ F	RTA	+++	-----	10 mon	4mm CC Screws	Excellent
2	17/M	Playing	+++	-----	12 mon	2.7 Hbt Screw	Good.
3	22/M	Fall	+++	----	8 mon	4mm CC Screws & TBW	Good.
4	35/F	RTA	+++	---	12 mon	2.7 Hbt Screw	Good
5	15/M	Direct Trauma	+++	---	14 mon	2.7 Hbt Screw	Excellent
6	22/M	Fall	+++	+++	10 mon	4mm CC Screws	Fair
7	28/M	Direct Trauma	+++	----	09 mon	2.7 Hbt Screw	Good.
8	33/F	RTA	+++	+++	15 mon	2.7 Hbt Screw	Excellent
9	20/M	Fall	+++	-----	12 mon	4mm CC Screws & TBW	Good.
10	16/M	Direct Trauma	+++	----	08 mon	2.7 Hbt Screw	Excellent
11	34/F	Sword trauma	+++	----	10 mon	4mm CC Screws	Fair
12	28/F	Fall	+++	----	12 mon	2.7 Hbt Screw	Good.
13	20/F	Direct Trauma	+++	----	14 mon	2.7 Hbt Screw	Good.
14	24/M	RTA	+++	+++	09 mon	4mm CC Screws & TBW	Fair

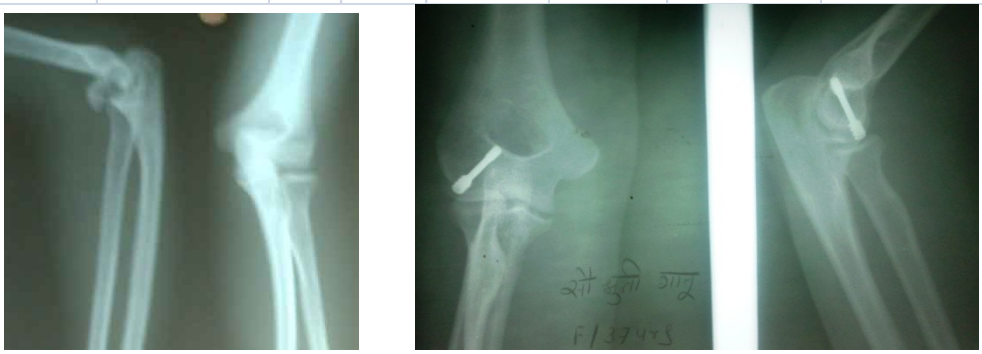


Fig Showing Fracture Capitullum Mckee Type3 fixed by 1 Herbert Screw.

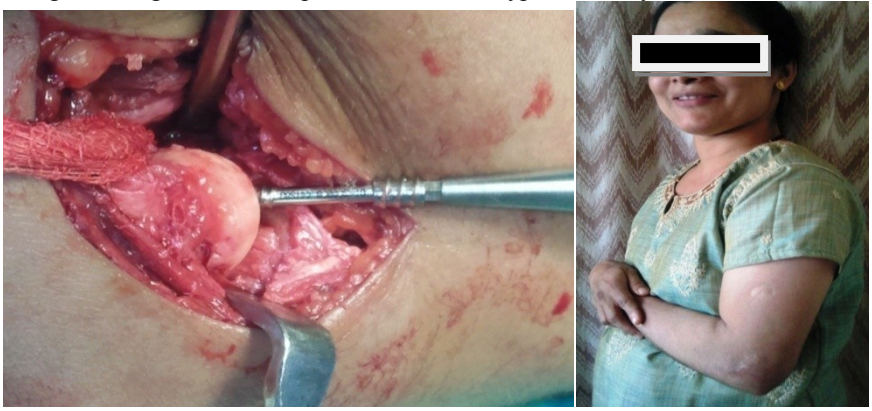
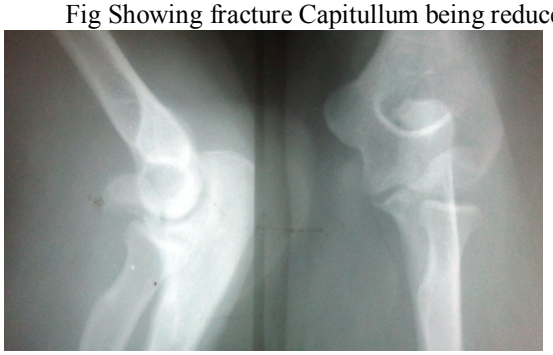


Fig Showing fracture Capitullum being reduced & Fixed by AP Herbert Screw.& Excellent Results.



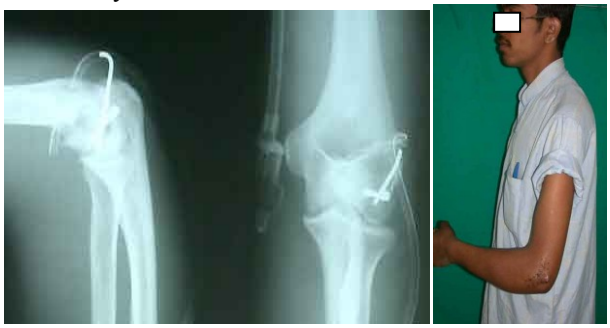
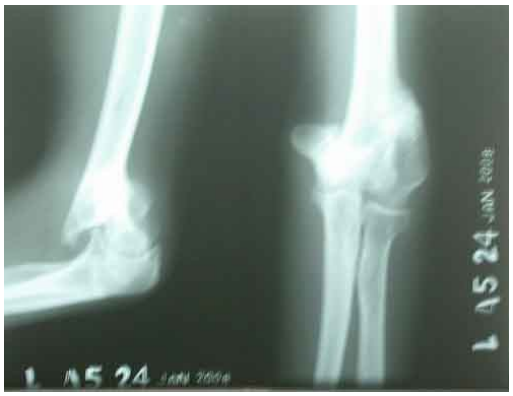
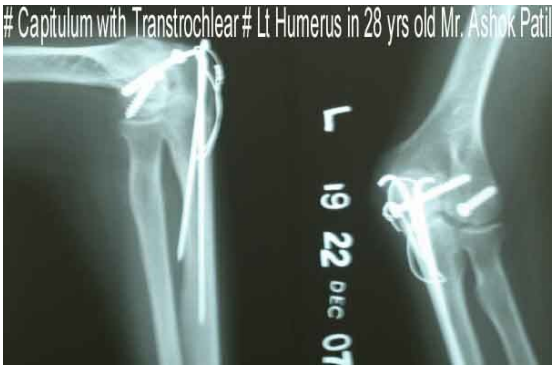


Fig Showing Fracture Capitullum treated with 1 Herbert Screw & Threaded K-wire which was removed after 4 weeks.





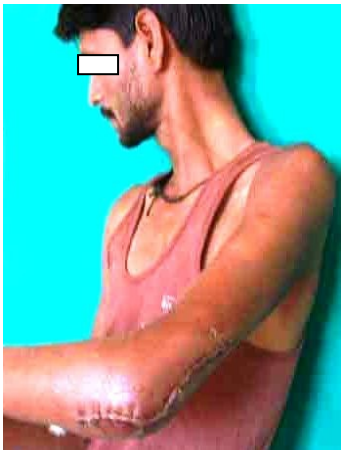
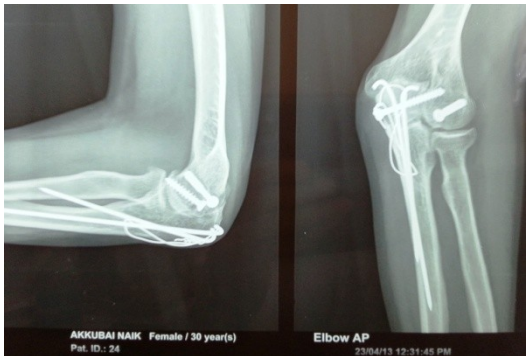
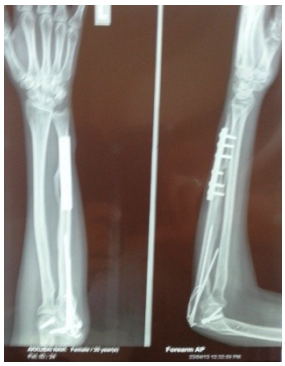


Fig showing T-Y elbow Fracture variant across the Capitullum.Treated by TBW for Olecranon 2 Herbert screws for the Fracture Capitullum.





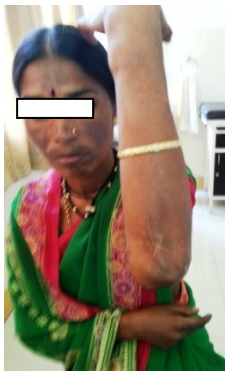
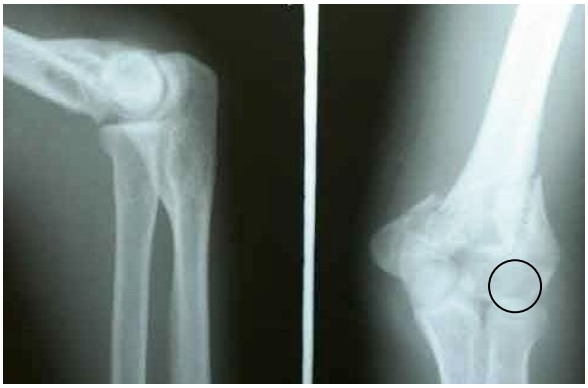
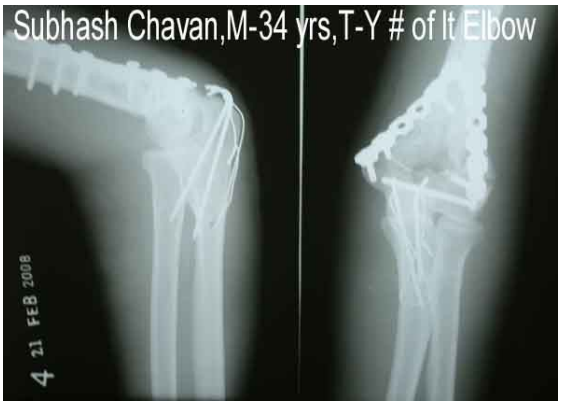
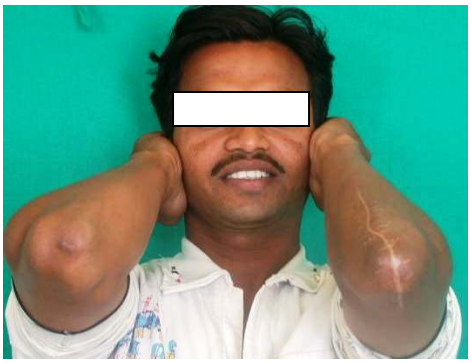


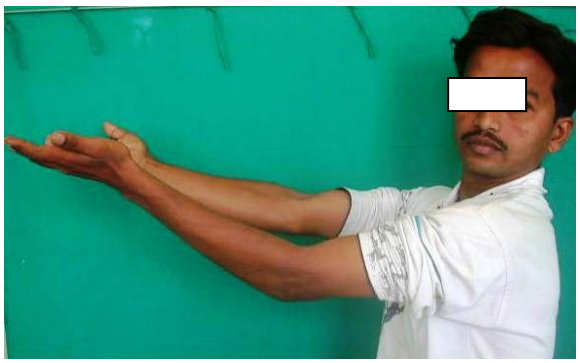
Fig Showing Post Traumatic (Sword Injury) across the Capitullum & Olecranon & Ulnar Fracture Distal Third.





T-Y elbow Fracture variant across the Capitullum & Trochlea. Treated by Trans Olecranon with 1 Cancellous screw for the Fracture Capitullum.& 2 lateral Pillar Plates for I/A Fracture of distal Humerus. With Good Results. But With loss of terminal 10^0 Extension.





IV. Discussion:-

Capitellum fractures are rare injuries that occur in adolescents over the age of 12 years. Though, reportedly, more common in females, with a male to female ratio of 1:4, 8 of the 14 cases in this series were males. Mechanism of injury is usually a fall on the out-stretched hand, the radius imparting a shearing force on the capitellum. Maximum force transmission through the radial head[21] to the capitellum occurs at zero to thirty degrees of elbow flexion.[20]

Proper visualization of the capitellar fragment is sometimes not possible in the routine views of the elbow and a radial head- capitellum view may help in better delineation of the fracture personality. Properly positioned lateral view is essential for diagnosis, with the fracture easily missed if the projection is slightly oblique as per Fowles and Kassab.[15]

A comparative view of the opposite elbow or CT scan will help in diagnosis. A properly taken lateral view usually shows anterior and superior migration of the capitellar fragment. Characteristic finding in the lateral X-ray is the "double-arc sign" because of the sub-chondral bone of the capitellum and lateral part of trochlea. The sub-chondral bone of the trochlea creates the double arc and when this sign is present it signifies that a part of the trochlea is also involved.[3]

Radiological diagnosis is difficult in a child because the capitellum is not fully ossified and fused before the age of 9-10 years. Other authors have suggested an oblique radiograph to detect this injury. In case of difficulty, in interpreting the radiographs.CT is advocated.

Fractures are often missed in the emergency room setting as the outline of the distal end of the humerus is intact. A CT scan delineates the fracture extent more clearly and helps the surgeon plan the approach, since, if the fragment is displaced on the medial side, another medial approach may be needed for reduction.

Treatment of type 2 and 3 capitullum fractures can be either conservative or excision of the fragments. Ochner reported, in 1996, successful outcome of closed reduction of coronal fractures of the capitellum in nine cases with long term follow-up.

In none of our cases closed reduction was attempted even before open reduction. Closed reduction of the fracture can lead to early arthritis, loss of motion of the elbow or instability of the elbow as it is usually a non anatomical reduction.[10]

Excision of the fragment can lead to instability of the elbow. Excision to prevent avascular necrosis is suggested by few authors. Fragment excision due to fear of avascular necrosis or redisplacement can lead to

radio-humeral osteoarthritis and instability of the elbow. Alvarez[11] advocated excision of the fragment in 10 out of 14 cases.

Approaches described include lateral approach (Modified Kocher approach), posterior approach with olecranon osteotomy[19]. Sano advocates olecranon osteotomy approach for proper visualization of the trochlea, but in the present series by retracting the medial structures with a bone lever the entire medial aspect of the trochlea could be visualized. The authors found the olecranon osteotomy approach useful if the trochlea also need to be fixed. Screws inserted from posterior to the anterior (PA) direction have more bio-mechanical stability than antero-posterior screws and this prevents damage to the articular cartilage.

Moreover, purchase of screw threads in the sub-chondral bone is more in PA directed screws, and splintering of the sub-chondral bone due to countersinking is less. Lateral collateral ligament has to be preserved during the procedure.

Various internal fixation methods have been described, including K wires, 4 mm cancellous screws, Herbert screws and absorbable polyglycide pins. There are also reports of plate fixation of the fracture. Kirschner wires do not provide enough stability for mobilization before fracture healing and also damage the articular cartilage. The better functional outcome of operative fixation has been documented.

Headless screws can have problems if the patients develop AVN or chondrolysis, because erosion of the radial head is a possibility due to exposed implants. This problem is avoided by the 4 mm partially threaded screws, which could be easily removed through stab incisions. Reports of avascular necrosis of the capitellum are very rare.

Grantham reported an elbow assessment based on stability, pain and range of movements, which is easy to follow.

Excellent - normal stability, no pain and full range of movements,
Good - less than 10 degree of instability, mild pain and less than 40 degree restriction of range of movements,
Fair -10-15 degree of instability, moderate pain or 40-60 degree of loss of range of motion,
Poor - 15 degree or greater instability, troublesome pain, or 60 degree or more of loss of range of motion.

Articular damage is thought to be the reason for **residual extensor lag** in spite of anatomical reduction and early mobilization.

V. Conclusion:-

Type 4 Isolated capitellar fractures[23] are less due to rarity of the injury. The importance of noting double arc sign in lateral view X-rays of the elbow and CT scan evaluation preoperatively is emphasized. The results of fixation with cannulated AO screws through extended lateral Kocher's approach has given good results. Good Anatomical Reduction, Rigid Internal Fixation & Early mobilization gives excellent results. This report is presented though there is no long term follow-up & sample size is also small to document post-traumatic arthritis & AVN

References

[1]. Ashwood N, Verma M, Hamlet M, Garlapati A, Fogg Q. Transarticular shear fractures of the distal humerus. J Shoulder Elbow Surg. Jan-Feb 2010;19(1):46-52. [Medline].
[2]. Ruchelsman DE, Tejwani NC, Kwon YW, Egol KA. Open reduction and internal fixation of capitellar fractures with headless screws. Surgical technique. J Bone Joint Surg Am. Mar 1 2009;91 Suppl 2 Pt 1:38-49. [Medline].
[3]. Guitton TG, Doornberg JN, Raaymakers EL, Ring D, Kloen P. Fractures of the capitellum and trochlea. J Bone Joint Surg Am. Feb 2009;91(2):390-7. [Medline].
[4]. Ruchelsman DE, Tejwani NC, Kwon YW, Egol KA. Coronal plane partial articular fractures of the distal humerus: current concepts in management. J Am Acad Orthop Surg. Dec 2008;16(12):716-28. [Medline].
[5]. Nalbantoglu U, Gereli A, Kocaoglu B, Aktas S, Turkmen M. Capitellar cartilage injuries concomitant with radial head fractures. J Hand Surg Am. Nov 2008;33(9):1602-7. [Medline].
[6]. Ochner RS, Bloom H, Palumbo RC, et al. Closed reduction of coronal fractures of the capitellum. *J Trauma*. Feb 1996;40(2):199-203. [Medline].
[7]. Bryan RS, Morrey BF. Fractures of the distal humerus. In: Morrey BF, ed. The Elbow and Its Disorders. Philadelphia, Pa: WB Saunders; 1985:302-39.
[8]. McKee MD, Jupiter JB, Bamberger HB. Coronal shear fractures of the distal end of the humerus. J Bone Joint Surg Am. Jan 1996;78(1):49-54. [Medline].
[9]. Ruchelsman DE, Tejwani NC, Kwon YW, Egol KA. Open reduction and internal fixation of capitellar fractures with headless screws. J Bone Joint Surg Am. Jun 2008;90(6):1321-9. [Medline].
[10]. .Brouwer KM, Jupiter JB, Ring D. Nonunion of operatively treated capitellum and trochlear fractures. J Hand
[11]. Alvarez E, Patel MR, Nimberg G, Pearlman HS. Fractures of the capitulum humeri. J Bone Joint Surg Am. 1975;57:1093–6.[PubMed]
[12]. Elkowitz SJ, Polatsch DB, Egol KA, Kummer FJ, Koval KJ. Capitellum fractures: A biomechanical evaluation of three fixation methods. J Orthop Trauma. 2002;16:503–6.[PubMed]
[13]. De Boeck H, Pouliart N. Fractures of the capitellum humeri in adolescents. Int Orthop 2000;24:246-8
[14]. Pradhan BB, Bhasin D, Krom W. Capitellar fracture in a child: The value of an oblique radiograph: A case report. J Bone Joint Surg Am. 2005;87:635–8.[PubMed]
[15]. Ochner RS, Bloom H, Palumbo RC, Coyle MP. Closed reduction of coronal fractures of the capitellum. J Trauma. 1996;40:199–203.[PubMed]

[16]. Fowles JV, Kassab MT. Fracture of the capitulum humeri: Treatment by excision. J Bone Joint Surg Am. 1974;56:794–8.[PubMed]
[17]. Dubberley JH, Faber KJ, Macdermid JC, Patterson SD, King GJ. Outcome after open reduction and internal fixation of capitellar and trochlear fractures. J Bone Joint Surg Am. 2006;88:46–54.[PubMed]
[18]. Lambert SM, Pike J, Railton GT. Fractures of the humeral capitellum: Herbert screw fixation. J R Coll Surg Edinb. 1994;39:321–3.[PubMed]
[19]. Ring D, Jupiter JB, Gulotta L. Articular fractures of the distal part of the humerus. J Bone Joint Surg Am. 2003;85:232–8.[PubMed]
[20]. McKee MD, Jupiter JB, Bamberger HB. Coronal shear fractures of the distal end of the humerus. J Bone Joint Surg Am. 1996;78:49–54.[PubMed]
[21]. Morrey BF, An KN, Stormont TJ. Force transmission through the radial head. J Bone Joint Surg Am. 1988;70:250–6.[PubMed]
[22]. Greenspan A, Norman A, Rosen H. Radial head- Capitellum view in elbow trauma: Clinical application and radiographic-anatomic correlation. AJR Am J Roentgenol. 1984;143:355– 9 [PubMed]
[23]. Grantham SA, Norris TR, Bush DC. Isolated fracture of the humeral capitellum. Clin Orthop Relat Res. 1981;161:262–9.[PubMed]
[24]. Pogliacomi F, Concari G, Vaienti E. Hahn-Steinthal fracture: Report of two cases. Acta Biomed. 2005;76:178–84.[PubMed]
[25]. Mehdian H, McKee MD. Fractures of the capitellum and trochlea. Orthop Clin North Am. 2000;31:115–27.[PubMed]