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Mean Arterial Pressure: Melatonin Versus Pregabalin in LarynGoscopy and Intubation in General Anaesthesia

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ABSTRACT
The degree of the response to external stimuli is determined by the severity, intensity and duration. There are several reflex reactions as well. The "stress response" or "alarm response" is a complicated interactionofsubstancesbetweenthehypothalamuspituitaryaxis,theclassical neuro-endocrinal hormone system and the autonomic nerve system that triggers these reflex responses to stimuli. A total of 50 patients planned for general anesthesia admitted in various surgical departments were included in the study after a written informed consent. Patients were taken to the operation the atre, pulse oximeter, noninvasive blood pressure, ECG and end-Tidal CO_2 monitors were connected. Baseline vital parameters and MAP were measured before induction. Comparison between mean arterial pressure at all time show statistically significant difference ($p<0.05$) between the two study groups.

INTRODUCTION

A direct laryngoscopy is done to visualize the larymx. It is used in general anaesthesia, resuscitation, and surgical operations involving the larynx. This is employed in operating room, critical care unit and emergency room. Endotracheal intubation is facilitated and airway is secured by visualising the larynx.

There are several steps to doing a successful direct laryngoscopy[1]. The patient must first be appropriately positioned. The "sniffing" position is characterised by atlanto-occipital extension and flexion of the lower cervical joints. Using the right hand, open the patient's mouth. The laryngoscope is next put into the mouth with the left hand, either in the middle or along the right side. The tongue should be pushed to the right side. It is inserted until it reaches the vallecula. The epiglottis is pushed higher by lifting the hyoepiglottic ligament. The vocal cords are visualized and an endotracheal tube is placed[2]. The human body responds to external stimuli in a variety of ways, ranging from slight to major injury, both locally and generally. The general response might manifest itself as a slew of endocrine, metabolic, and biochemical processes all across the body. The degree of the response to external stimuli is determined by the severity, intensity and duration. There are several reflex reactions as well.The "stress response"or"alarm response"is a complicated interaction of substances between the hypothal amuspituitary axis, the classical neuro-endocrinal hormone system, and the autonomic nerve system that triggers these reflex responses to stimuli[3-4]. The local response is for wound healing and infection protection. Chemical mediators, vascular endothelial cell products and even intracellular products of single cells play a role in it. The stress response causes the release of several hormones with anabolic and catabolic effects, causing hypermetabolism and the acceleration of most metabolic events. If the stress reaction is extended, it can cause weight loss, fatigue, decreased resistance, delayed ambulation and increased morbidity and mortality by depleting key biological components such as glucose, fat, protein and minerals[5-6]. The net effect of stress response "The Neuroendocrinal outflow"

MATERIALS AND METHODS

Study Design: Observational Analytical Study.

Study Population: A total of 50 patients planned for general anesthesia admitted in various surgical departments were included in the study after a written informed consent.

Inclusion Criteria:

- Patients between ages 20-45yrs undergoing elective surgery.
- ASA class 1 and 2.
- Both sexes.

Exclusion Criteria:

- Younger than 20yrs or older than 45yrs.
- ASA grade 3 and 4.
- Anaemia.
- Patients with compromised renal status, Cardiac disease, hypertension, COPD and asthma, diabetes, psychiatry illness, antiepileptic drugs, sedatives, anxiolytics, allergy to any anaesthetic medication, pregnant and lactating women.
- Anticipated difficulty in intubation (mallampati class 3 and 4) and those requiring more than three attempt or more than 20s for laryngoscopy.

Fifty normotensive patients of status ASA grade 1 and 2 between age 20-45 years old, 40-65kg body weight, undergoing elective surgeries under general anaesthesia were included in the study after ethical clearance. On the day of operation, the patient was evaluated preoperatively. After taking written and informed consent, indication for surgery, baseline heart rate and blood pressure were noted. Basic appropriate investigations like Hb, complete blood count, serum urea and creatinine, blood sugar, urine examination, chest X-ray and ECG were done. Patients were advised 6-8 hours fasting prior to surgery and are received tablet ranitidine 150mg the previous night.

- Group A (n=25) patients received oral 6mg melatonin tablets (two tablets of 3mg each) 120 mins before surgery.
- Group B (n=25) patients received 150mg of pregabalin tablet 90 mins before surgery.

Total of 50 patients who received these tablets before surgery were observed and studied.

Patients were taken to the operation theatre, pulse oximeter, noninvasive blood pressure, ECG and end-Tidal CO_2 monitors were connected. Baseline vital parameters-HR, SBP, DBP andMAP were measured before induction.

RESULTS AND DISCUSSIONS

The patients in this study were between the ages of 20 and 45. The average age of Melatonin patients was 38.2 years, whereas the average age of Pregabalin patientswas 34.68 years. There was significant difference in age among the two groups (p-0.09). (Figure 1)

There was statistically significant difference between the weight of participants in the Melatonin and Pregabalin group as shown in above graph (p-0.006)

Comparison between mean arterial pressure at all time show statistically significant difference ($p<0.05$)

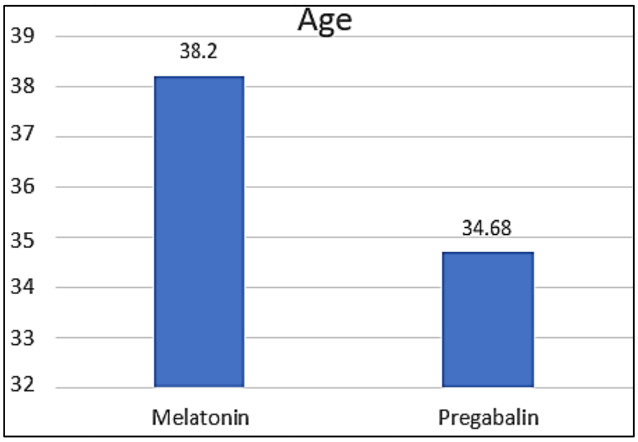

Fig 1: Distribution of age in 2 groups

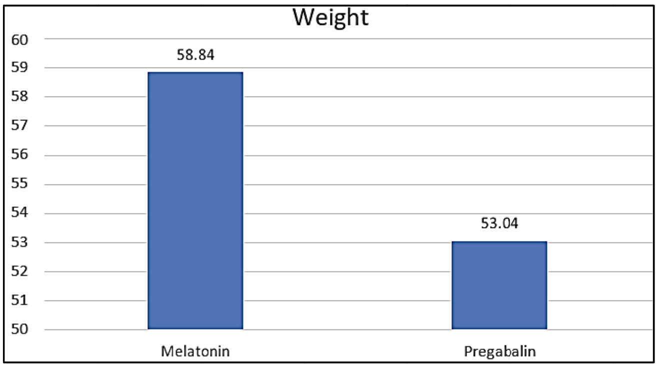

Fig 2: Distribution of weight in 2 groups

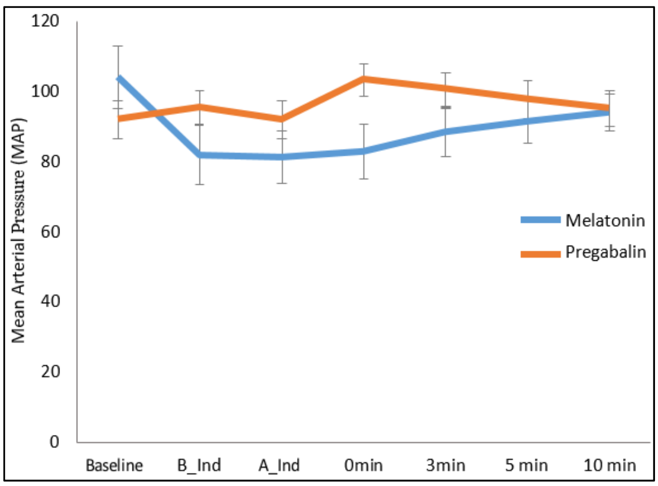

Fig 3:Comparison of MAP between 2 groups

Table 1: Comparison of map between 2 groups

	Group	N	Mean	Std. Deviation	t	p value
MAP-Baseline	1	25	104.16	9.049	-3.445	<0.001
	2	25	92.08	5.330		
MAP-B-Ind	1	25	81.84	8.410	-7.531	<0.001
	2	25	95.44	4.805		
MAP-A-Ind	1	25	81.28	7.525	-7.685	<0.001
	2	25	92.00	5.485		
MAP-0m	1	25	82.88	7.775	-11.562	<0.001
	2	25	103.36	4.627		
MAP-3m	1	25	88.48	6.881	-8.750	<0.001
	2	25	100.72	4.809		
MAP-5m	1	25	91.48	6.279	-5.520	<0.001
	2	25	97.76	5.403		
MAP-10m	1	25	94.00	5.252	-2.746	0.001
	2	25	95.24	5.174		

between the two study groups as seen in the above table and graph.

Melatonin is a natural chemical found in all major taxa of life and was originally referred to as a master hormone. It is generated primarily in the pineal gland of all mammals and vertebrates, with high secretion at night and reduced secretion during the day[7].Tryptophan is also used to make melatonin in a variety of different organs and tissues. Melatonin is a naturally occurring substance in diet. Rice, cucumber, cherries, tomatoes, human milk, bananas, yeast, wine and beer are all high in melatonin. Melatonin has a wide range of characteristics and functions in the body, many of which have therapeutic implications[8]. The pineal gland which is located in brain is referred as the 'Seat of the Soul. Melatonin (5-methoxy-N-acetyltryptamine), the main hormone produced by the internal clock, is often employed as a phase indicator. Melatonin synthesis at night offers a universal time cue for changing day length in animals whose seasonal functions are timed by day length. The profile of its secretion defines "biological night". Pregabalin was developed as an anticonvulsant in 1990. Richard Bruce Silverman, a scientist at Northwestern University in Evanston, Illinois, created it. In 2004, the European Union authorised the drug. In December 2004, the Food and Drug Administration (FDA) approved it for use in treating epilepsy, diabetic neuropathic pain and postherpetic neuralgia in the United States. Pregabalin was later introduced to the US market in the fall of 2005 under the trade name Lyrica[9]. Melatonin's influence on circulation has a complicated process. The particular binding of melatonin to melatonin receptors in the blood arteries, which interferes with the vascular response to catecholamines, may be responsible for the blood pressure reducing effect[6].It may cause a drop in adrenergic outflow and subsequent catecholamine levels by interfering with both the peripheral and central autonomic systems. Furthermore, by increasing the availability of nitric oxide, it may cause the smooth muscle of the artery wall to relax. It may also work through specialized melatonin type 1 or type 2 receptors found peripherally in blood vessels and centrally in the blood pressure-regulating region of the brain. It also has a free radical scavenging function, which causes blood vessel dilation and it may work through an epigenetic process in the area postrema of the brain. The sleepy effect of orally given melatonin might potentially be responsible for the blood pressure lowering effect. Binding to the GABA-A receptorand exerting its anaesthetic action is what causes the sedative effect. Its sedative and anxiolytic effects on the central nervous system may have a role in reducing haemodynamic reactions to laryngoscopy and intubation[10].

CONCLUSION

There was significant statistical difference in MAP between Melatonin and Pregabalin groups. Melatonin group showed significant decrease in MAP during study period compared to Pregabalin group.

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